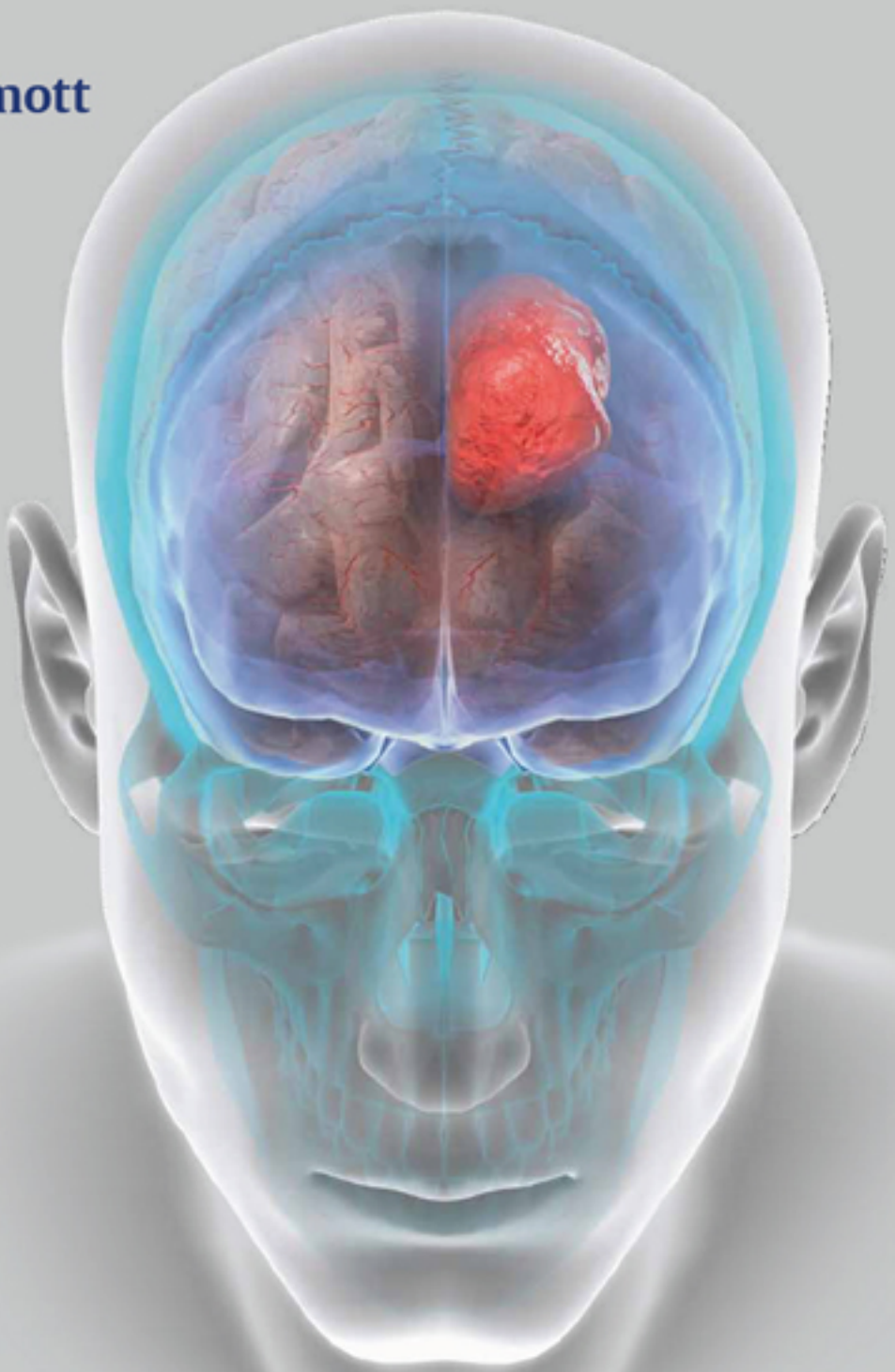
Al-Mefty's Meningiomas

Franco DeMonte
Michael W. McDermott
Ossama Al-Mefty

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Dedication

To our families and patients, who have allowed us to dedicate considerable effort to further the understanding and treatment of meningiomas.

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Foreword

Meningioma management has become a very complex, multifaceted process in the daily lives of all neurosurgeons. Meningiomas are extraordinarily complex lesions that require a different set of surgical skills and judgment than what we use for other tumors such as gliomas. To this end, this definitive textbook logically addresses all of the vital components necessary to make the appropriate decisions for patients with meningiomas. The contributors are a "who's who" of masterful neurosurgeons, neuroradiologists, and neuroanesthesiologists who have come together to create a tremendous resource that belongs in every neurosurgeon's personal library.

I greatly appreciate the way this textbook is organized, as it allows us to access critical data with ease. The opening contributions, outlining the latest in pathological classification and epidemiology of meningiomas, are welcome components. It is also important to recognize that the molecular mechanisms associated with tumorigenesis are slowly becoming known and merit further consideration as depicted in this text. I particularly enjoyed the section on the natural history of untreated meningiomas and how these tumors affect the elderly. These are important considerations that come up almost on a daily basis in clinic. I believe that the information provided within the text will help us make rational decisions with regard to the natural history of these lesions and how we should approach these tumors in asymptomatic individuals. The sections on perioperative management and diagnostic imaging, as well as preoperative embolization, are beautifully done and clearly present the state of the art in these areas. The book evolves into an encyclopedia of knowledge, with each meningioma per location being thoroughly depicted in terms of how that lesion should be approached and what issues to consider. The photos and illustrations throughout the book are superb!

The latter components in the book deal with important issues such as special operative considerations. These chapters describe in detail the use of navigation, intraoperative MRI, endoscopy, and how surgeons should treat tumors that invade venous sinuses. A section on spinal meningiomas completes this comprehensive textbook.

Finally, no textbook would be complete without an evaluation of outcomes and quality of life associated with resecting or treating meningiomas in various locations. We have come to realize that these complex operations should be done by surgeons who have repetitive experience and a significant volume under their belt in order to minimize the morbidity and mortality associated with removing these often difficult tumors.

All in all, *Al-Mefty's Meningiomas* is a phenomenal addition to the neurosurgical armamentarium. This second edition will be the definitive text on meningioma management and surgery for our specialty for years to come. It is nothing short of a tremendous accomplishment by one of the great masters in neurosurgery today.

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Preface

Twenty years have passed since Sam Al-Mefty edited the first publication of *Meningiomas*. To say much has changed over the ensuing two decades would be a gross understatement. One need only contemplate the incorporation of image guidance into routine surgical practice and the rise of radiosurgery as a treatment option for meningiomas as prime examples. Yet many questions still remain unanswered, both with respect to the biologic nature of these tumors and to the optimal choices for patient management.

This book was composed with the goal of updating students, residents, and practicing neurosurgeons and neurologists on the advancements made over the past twenty years in the field of meningioma biology, diagnosis, and management.

Following a powerful opening chapter by Dr. Al-Mefty, and an enlightening and entertaining overview of meningioma surgery in the twentieth century by Dr. Fred Barker, the book's contents correspond to four general thematic areas. The first set of chapters presents advancements in our understanding of the anatomy, pathology, molecular biology, and natural history of meningiomas. Special notice should be taken of the many new associations being made between meningiomas and various oncogenes, growth factors, and genetic polymorphisms, as well as the sometimes indolent or even stagnant growth patterns that form part of the natural history of these tumors.

The second selection of chapters discusses the medical, anesthetic, and diagnostic evaluation of both the adult and pediatric meningioma patient. These chapters update and modernize the standards of perioperative care for patients undergoing meningioma surgery.

The third grouping of chapters presents the reader with a site-specific comprehensive review of the advancements made in meningioma surgery. Of particular note are new chapters on image-guided surgery, intraoperative MRI, and endoscopic resection. These chapters represent the main corpus of the book and present detailed information on the surgical management of meningiomas from the olfactory groove to the cervicomedullary junction and spinal column.

The concluding chapters of the book highlight advances made in the radiosurgical, radiotherapeutic, and chemotherapeutic management of meningiomas and presents critical new information on patient outcomes and quality of life.

We hope that this new edition of *Meningiomas* proves a valuable contribution to the literature on the fascinating aspects of meningioma biology, diagnosis, and management. We hope it stimulates further investigative interest in the field of meningioma research, so that the questions left unanswered by this work will ultimately be answered. We wish to express our thanks to the contributors for their willingness to participate, their comprehensive review of their subjects, and their timeliness. We stand indebted to them for their contributions.

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Introduction

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Chapter 1

Meningiomas: A Personal Perspective

Ossama Al-Mefty and Rami O. Al-Mefty

A meningioma is, in many ways, the soul of neurosurgery. The progress in meningioma treatment mirrors advances in neurosurgery, while advancements in neurosurgery are put to maximum use to improve the treatment of meningiomas.[1]

The last 20 years confirmed the statement above. Many advances have taken place, in both the treatment and the biological understanding of meningiomas. I (OA) commend Dr. DeMonte and Dr. McDermott for their outstanding effort embodying these new advances in this current edition of *Meningiomas*.

Over the past two decades and especially in the last several years, the biology and natural history of meningiomas have become better understood. The reader is referred to Chapters 2 through 9 for a detailed summary of these advances. Unlike the decade before, however, the last decade has been overwhelmed with reports on radiosurgery's role in the treatment of meningiomas, reports declaring a "change in paradigm" in the treatment of meningiomas and implying the need to alter time-tested surgical goals and attitudes. Some authors have called for the primary treatment of meningiomas by radiosurgery,[2–5] whereas others have evolved their surgical role into a decompressive, partial removal to be followed by radiosurgery.[6–10] These changes have taken place without data on the long-term outcome of such approaches or recognition of the profound effect of failure on both longevity and quality of life.

Durante's success in the total resection of an olfactory groove meningioma in 1885 began paving the way for treatment of meningiomas.[11] The goal of treatment cannot be more eloquently stated than the words of Cushing: "There is to-day nothing in the whole realm of surgery more gratifying than the successful removal of a meningioma with subsequent perfect functional recovery."[12] Meningiomas, as a benign tumor, are amenable to surgical *cure*. This should be the aim of their management. Complete removal of a meningioma, Simpson grade I, offers a cure in more than 90% of cases.[13,14] That extent of surgical removal directly influences the recurrence rate has been repeatedly confirmed. A subtotal removal, Simpson grade IV is doomed to recur when the patient is followed longer than 15 years.[15] Sixty percent of patients who experience recurrence after subtotal removal die from the tumor, with the majority of deaths occurring within 10 years.[16]

Radiation therapy administered to treat residual meningioma has failed in its purported goal of long-term control; 75% of patients thus treated experience recurrence, and 56% of these patients endure neurological complications related to radiation.[14] Recent long-term reports on radiosurgery not only raise the concern of increasing failure but also address the aggressive tumor growth that occurs after the failure of radiosurgery.[17] The 15-year patient actuarial survival rate after gamma knife surgery for meningioma is only 53%, and 68% of the deaths in these patients are caused by the tumor.[18] We are seeing increasing numbers of patients who have been treated in various centers around the world with subtotal removal and radiosurgery who are now desperate; they have undergone multiple surgeries and radiosurgery, only for the tumor to grow faster and transform to a higher pathological grade. The result is a long course of accumulated morbidity, agony, and expense for palliative management, while the chance for a cure is lost because of the failure to attempt or to achieve total surgical removal the first time. One must admit that not every meningioma can be removed totally, and the surgeon is forced in some cases to accept residual tumor, but it should be the surgeon's goal to attempt a total removal with zeal while preserving or improving the patient's neurological function. We believe cranial base approaches facilitate the achievement of these goals. If these approaches are associated with higher morbidity in an early experience, then the technique should be refined, but not abandoned. Today's neurosurgeons are fortunate to be armed with microneurosurgical techniques and skull base approaches in their quest for cure of meningiomas.

In pursuing safe and total removal of meningiomas, which not only provides elimination of the tumor but also contributes to the preservation and improvement of neurological function and the high quality of life that

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accompanies it, I (OA) have found the following points particularly important.

Bony Invasion by Meningioma

Meningiomas frequently invade adjacent bone, manifested by the radiological appearance of hyperostosis. We have documented pathologically the almost universal presence of meningioma in the Haversian canals of this hyperostotic bone[19] (**Fig. 1.1**). The floor of the middle fossa is an area frequently involved. We believe that removal of the hyperostotic bone provides a more complete resection and leads to a lower rate of recurrence (**Fig. 1.2**). Conservative resection of the bone is typically the cause of the high rate of delayed recurrence after presumed total tumor removal, such as is seen in olfactory groove meningiomas.[20] This hyperostotic bony invasion is particularly well known in parasagittal and sphenoid wing meningiomas, which are two sites historically associated with a higher rate of recurrence. We believe their treatment requires the complete removal of the hyperostotic bone.[21]

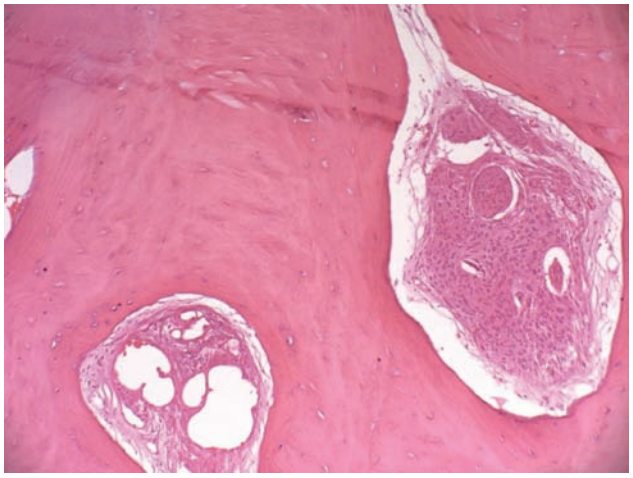


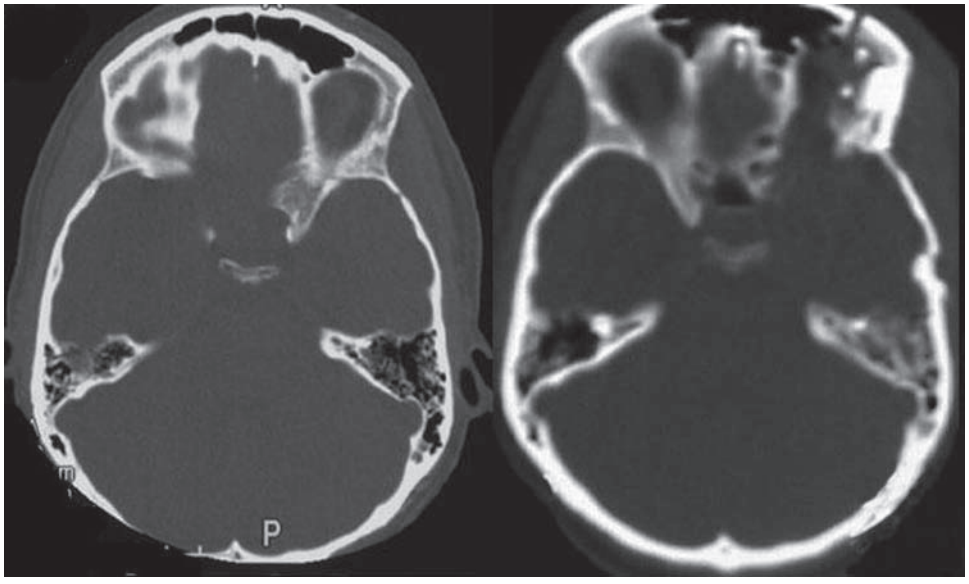
Fig. 1.1 Hematoxylin and eosin stain of hyperostotic bone demonstrating meningioma invasion into the Haversian canal.

Grade 0 Removal

Excising the involved dura is a component of achieving a Simpson grade I removal. To further minimize the recurrence of convexity meningiomas, we excise an additional dural margin of about 2 cm around the tumor.[22] This "grade 0" removal is based on the findings of Borovich and Doron. Their study showed that the dura mater of patients with meningiomas harbors meningotheliomatous cell aggregates around the attached globoid tumor, bringing attention to the role of regional multicentricity in the recurrence of meningioma.[23] The achievement of a grade 0 removal may not be feasible in skull base meningiomas. We do, however, pursue the excision of the dural tail (**Fig. 1.3**) as far as is visible intraoperatively or as depicted by intraoperative neuronavigation.

Pathological Anatomy and Intraarachnoidal Dissection

We believe that the site of origin must be the basis for classification of meningiomas, as established by Cushing[24] and supported by others.[1,25,26] It is critical to the understanding of the pathological anatomy of tumoral displacement, encasement, and adherence to the neurovascular structures; thus, it has a profound influence on the surgical intervention and its outcome. The presence of multiple arachnoid layers facilitates the safe dissection of neurovascular structures despite their encasement by tumor. In other words, surgery in basal meningiomas is intraarachnoidal surgery.[27] The success



A B

Fig. 1.2 Computed tomographic (CT) scan demonstrating hyperostosis indicative of bony invasion that requires surgical resection. **(A)** Preoperative CT scan, bone window. **(B)** Postoperative CT scan demonstrating removal of the hyperostotic bone.

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comes in the presence of a double arachnoid plane that facilitates the resection of the tumor while preserving the neurovascular structures and their finest details, including perforating arteries, veins, and nerve rootlets. Clinoidal meningiomas present striking evidence supporting this concept. We have distinguished three categories of these tumors, groups I, II, and III, each with a marked influence on the surgical difficulties, ability to achieve total removal, and outcome. These groups relate to the presence of an interfacing arachnoid membrane between the tumor and the cerebral vessels. The presence or absence of this arachnoid membrane depends on the origin of the tumor and its relation to the short intradural carotid artery segment that lies outside the carotid cistern[28] (**Figs. 1.4, 1.5, 1.6,** and **1.7**). This same principle not only applies in the classification of tumors of the petroclival area but also has a profound effect on the degree of removal, the rate of complications, and the surgical approach for these tumors (**Figs. 1.8, 1.9,** and **1.10**).

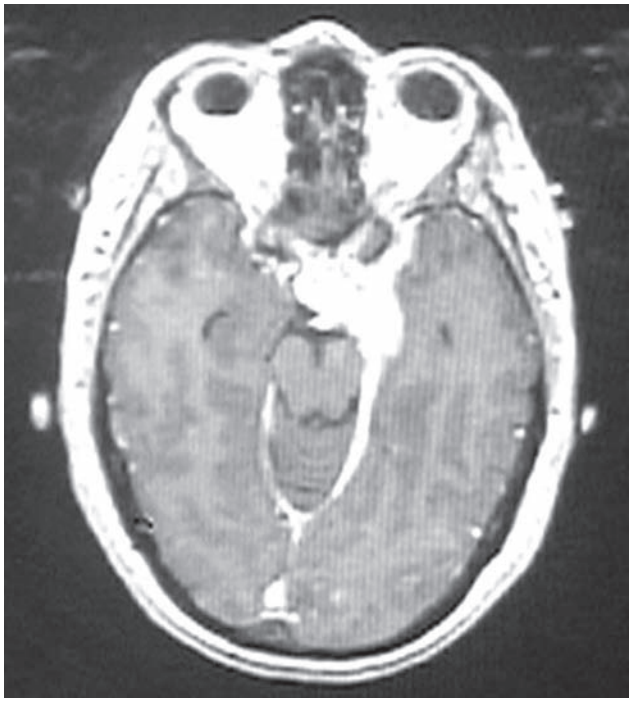


Fig. 1.3 Magnetic resonance imaging demonstrating the tentorial involvement depicted as a long dural tail.

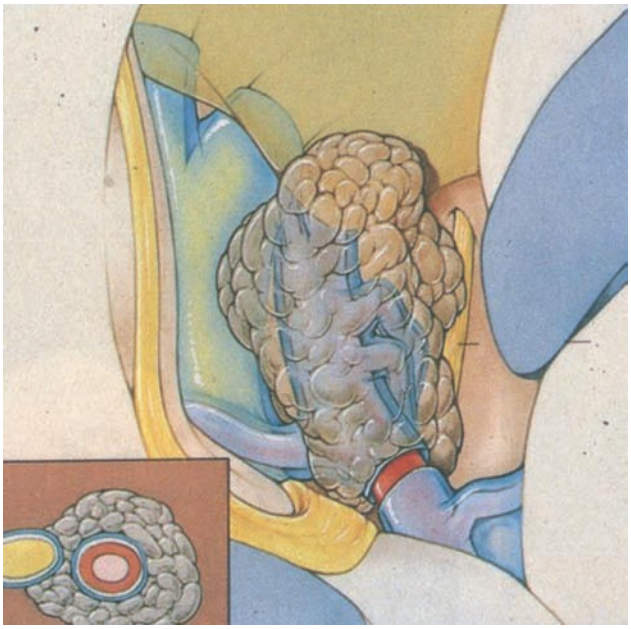
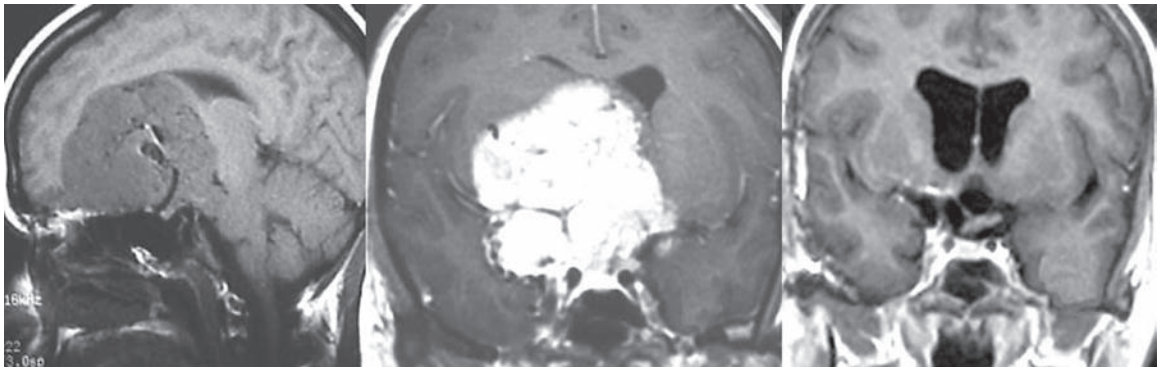


Fig. 1.4 Artist's illustration of a group II clinoidal meningioma. Although the tumor engulfs the carotid artery and its branches, it can be totally removed because of the intervening arachnoidal plane. Used with permission from Al-Mefty O. Clinoidal meningiomas. *J Neurosurg* 1990;73(6):842.



A **B** **C**

Fig. 1.5 Imaging demonstration of a group II meningioma that is amenable to total removal despite vascular encasement. **(A)** Preoperative T1 sagittal magnetic resonance imaging (MRI). **(B)** Preoperative coronal enhanced MRI. **(C)** Postoperative coronal enhanced MRI showing total removal.

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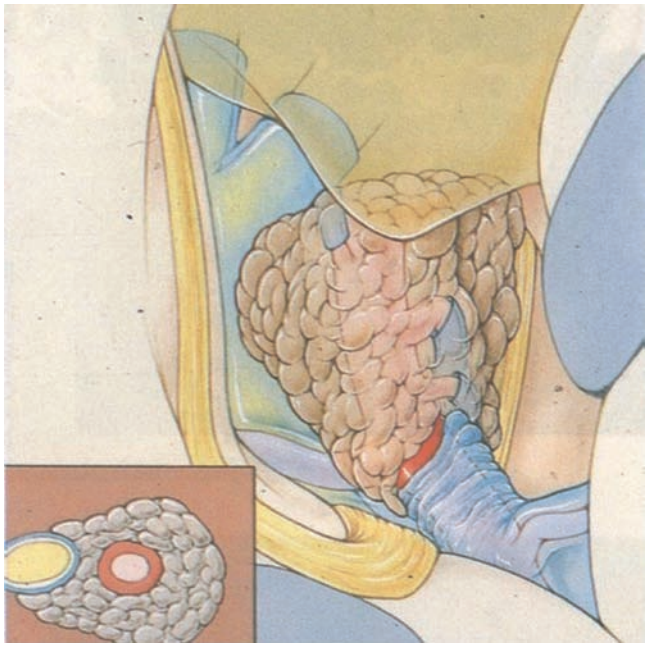


Fig. 1.6 Artist's illustration of a group I anterior clinoidal meningioma, which arises proximal to the carotid cistern. The tumor adheres directly to the arterial adventitia. Used with permission from Al-Mefty O. Clinoidal meningiomas. *J Neurosurg* 1990;73(6):841.

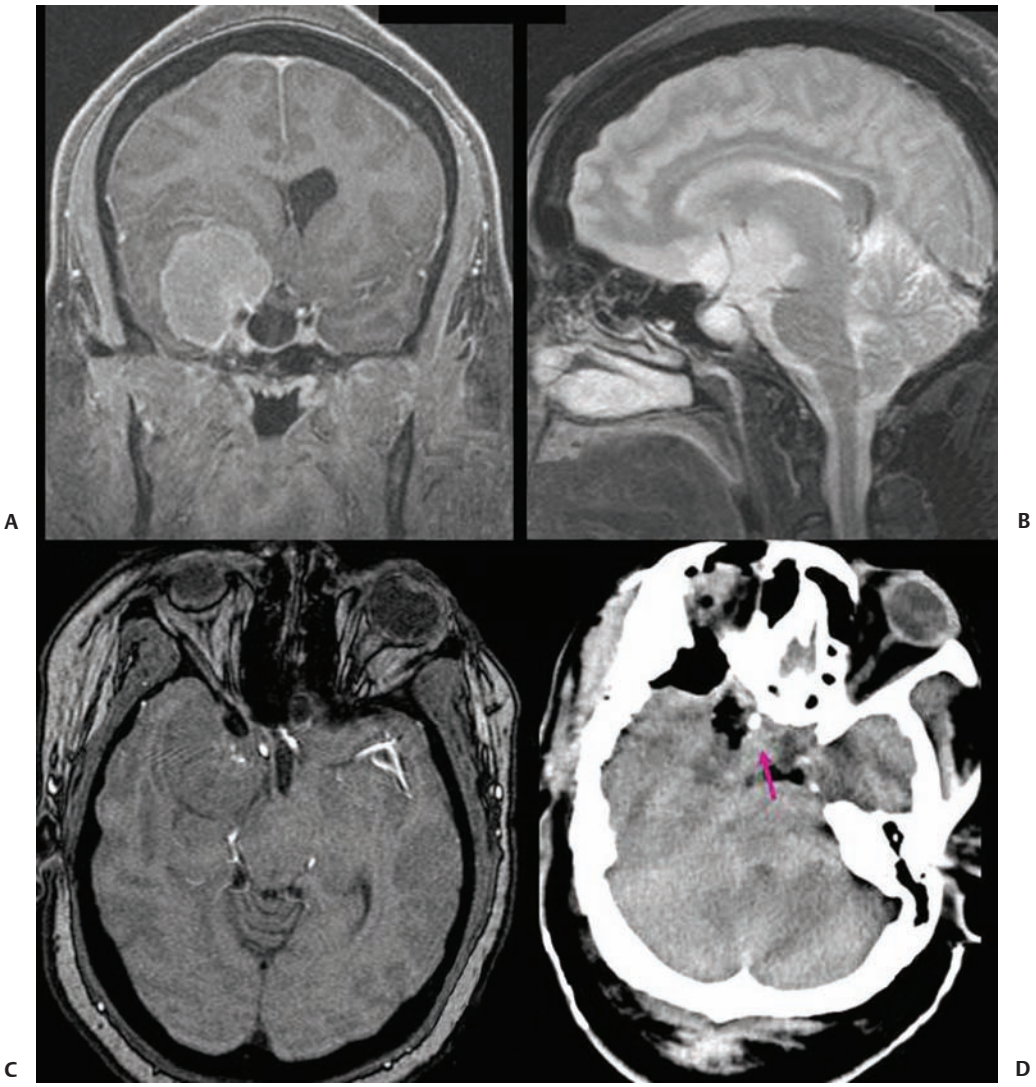


Fig. 1.7 Imaging studies of group I clinoidal meningioma that was not amenable to total removal because of its origin from the anterior surface of the clinoid. **(A)** Preoperative coronal enhanced magnetic resonance imaging (MRI). **(B)** Preoperative sagittal T2 MRI. **(C)** Preoperative axial enhanced MRI. **(D)** Immediate postoperative MRI demonstrating residual tumor.

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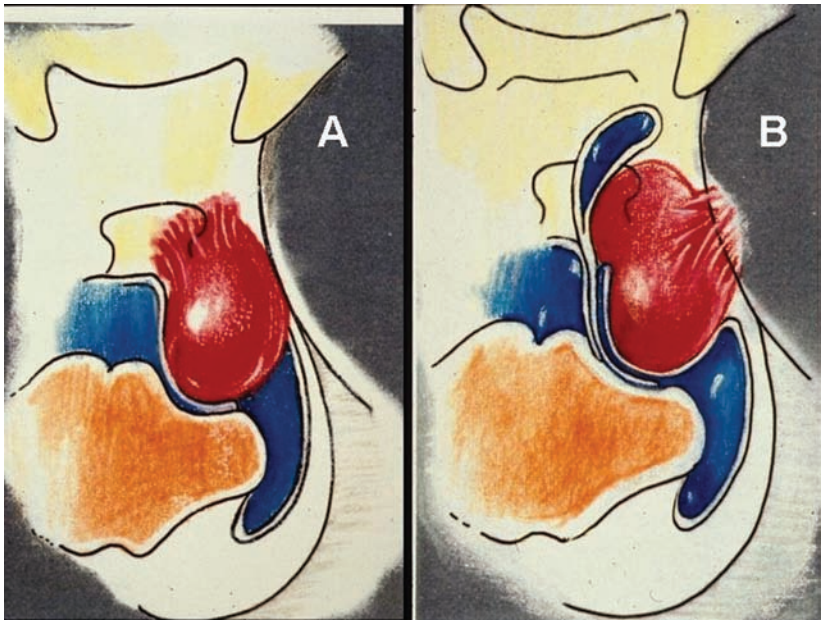


Fig. 1.8 Artist's illustration of two petroclival meningiomas separated in origin by a few millimeters. **(A)** Medial origin of the tumor, with only a single arachnoid plane of the prepontine cistern between the tumor and the brain stem, contributing to adhesion and difficulty in resection. **(B)** Slightly more lateral origin allowing a multilayer arachnoid from the prepontine, ambient, and crural cisterns, facilitating intraarachnoidal dissection.

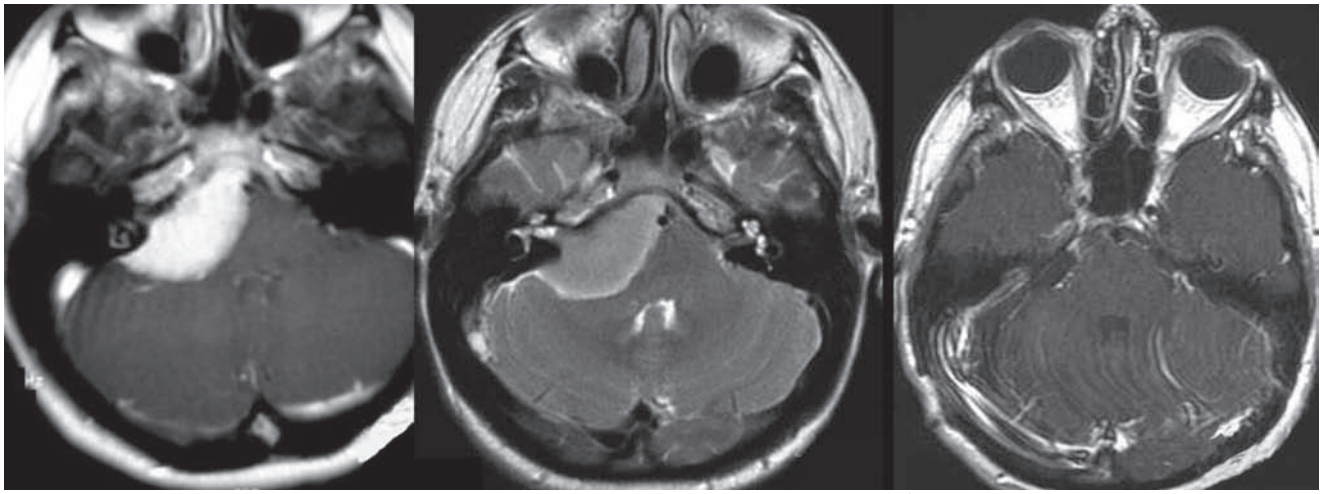


Fig. 1.9 Magnetic resonance imaging (MRI) of a petroclival meningioma that maintains a multiarachnoidal layer, which facilitates intraarachnoidal dissection. **(A)** Preoperative axial enhanced MRI. **(B)** Preoperative T2 axial MRI. **(C)** Postoperative axial MRI demonstrating total removal.

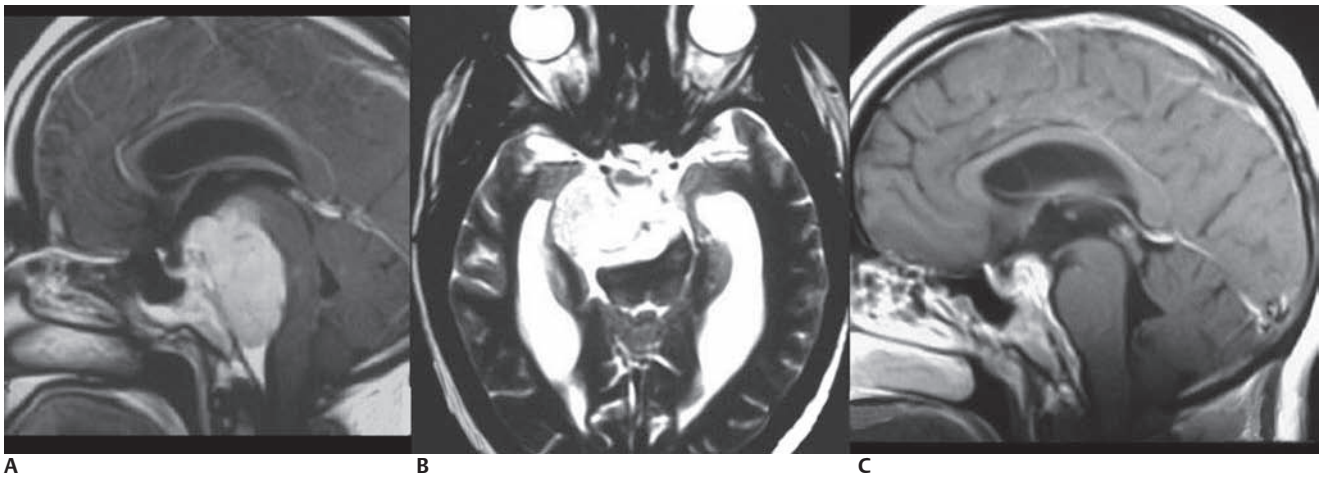


Fig. 1.10 Magnetic resonance imaging (MRI) of a clival meningioma that originated medially and was not able to be removed totally because of the absence of multiple arachnoid layers against the upper brain stem. **(A)** Preoperative enhanced sagittal MRI. **(B)** Preoperative axial T2 MRI. **(C)** Postoperative sagittal enhanced MRI demonstrating a small residual at the dorsum sellae.

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The Role of Skull Base Approaches

Skull base meningiomas continue to pose a formidable challenge to neurosurgeons in their quest for total removal. The historically dismal outcome of these interventions has been largely overcome by the introduction of microsurgery. However, meningiomas like petroclival meningiomas, sphenocavernous meningiomas, and foramen magnum meningiomas continue to be characterized by high morbidity, high mortality, and high failure of total removal despite the advent of microsurgical technique, advanced imaging, and modern anesthesia. To better their treatment, improve their outcome, and achieve a higher cure rate, the development of skull base approaches and their utilization were well founded in principle and proven beneficial in practice. I (OA) speak with avid enthusiasm and vast experience to the advantage and instrumental role of skull base approaches in the surgical removal of skull base meningiomas. The wide incorporation into neurosurgical practice of the various skull base approaches is a fulfillment of Cushing's hope: "It is possible of course that a method may some day be evolved whereby a Gasserian neurinoma or meningioma, even after it has crossed the ridge, may be safely approached and removed. Should this come to pass, it will be another conquest for neurosurgery" **(Fig. 1.11)**.

Skull base approaches are particularly advantageous for posterior fossa basal meningiomas. The utilization of the traditional approach for a meningioma in a location such as the petroclival region or the ventral foramen magnum is associated with a lower rate of removal or reluctance in attempting total removal.[8,9,29–32] The posterior petrosal approach, for example, offers a superb advantage in the management of large petroclival meningiomas. The posterior petrosal approach, as we described it, is centered on the petrous bone, allowing exposure of the tumor from the middle fossa to the foramen magnum (**Fig. 1.12**). We prefer this approach for several reasons: (1) there is minimal retraction of the temporal lobe and cerebellum; (2) the operative distance to the clivus is shortened when compared with the suboccipital approach; (3) the surgeon has a direct line of sight to both the lesion and the anterior and lateral aspects of the brain stem; (4) the cochlea, labyrinth, and facial nerve are preserved; (5) the transverse and sigmoid sinuses, as well as the vein of Labbé and the basal and occipital veins, are preserved; (6) the tumor's vascular supply is encountered early in the procedure; (7) multiple axes for dissection are provided; (8) tumor invading the petrous bone can be safely removed; and (9) it is easily extended by combining it with other approaches, such as anterior petrosal (**Fig. 1.13)**, total petrosectomy, and transcondylar approaches.[33]

The transcondylar approach is extremely advantageous in dealing with ventral foramen magnum meningiomas. The partial drilling of the condyle provides a superb exposure of the lesion, control of the vertebral artery, minimal cerebellar retraction, and ease of dissection of the vital neurovascular structures[34] (**Figs. 1.14** and **1.15**). These advantages lessen morbidity, lower mortality, and provide a high surgical cure rate.

I (OA) not only recommend the utilization of these approaches, but also believe that they are crucial in achieving safe and total removal. Hence they are the vehicle for increasing success and lowering mortality and morbidity, thus subsequently improving the patient's quality of life.

Predictors of Aggressive Meningiomas

By and large, meningiomas are benign tumors. World Health Organization (WHO) grade II meningiomas include atypical, chondroid, and clear cell types, and WHO grade III tumors include anaplastic, papillary, and rhabdoid types. All other variants of meningioma are WHO grade I and benign. Therefore, it is essential to identify markers or parameters, other than histopathology, to determine the tendency of a meningioma to recur, grow rapidly, or behave aggressively. This is particularly vital given that not all meningiomas can be totally removed surgically, and subsequent decisions must be made regarding the management of residual disease. It is our practice to use these parameters, and it is our expectation that they will be widely used. The determination of proliferative indices has gained wide usage due to the ease of MIB-1 and its utilization in a fixed specimen. MIB-1 is a commonly used monoclonal antibody that detects the K_i-67 antigen. It is used in clinical applications to determine the *K_i-67 labeling index*. We also found an impressive correlation with sex hormone receptors. Progesterone receptor (PR)-positive meningiomas were associated with a lower recurrence rate, less aggressive pathology, longer survival, lower proliferative indices, and a better prognosis. The expression of the PR alone in meningiomas signals a favorable clinical and biological outcome. A lack of receptors or the presence of estrogen receptors correlates with an accumulation of chromosomal abnormalities and an increasing potential for aggressive clinical behavior, progression, and recurrence.[35] We urge that sex hormone receptor status be studied routinely for its prognostic value, especially in female patients, and it should be taken into account in tumor grading. The initial receptor status of a tumor may change after progression or recurrence.

Today, however, it appears that cytogenetic analysis is becoming the most definitive predictor of an aggressive meningioma. A meningioma with a normal karyotype is definitely associated with a lower recurrence rate and slower growth. Monosomy of chromosome 22 is a frequent finding in meningiomas and may still be associated with a benign meningioma. The deletion of chromosome 1P or 14Q, however, has been definitively associated with a higher-grade meningioma and more aggressive biology.[36] We consider the presence of a chromosomal anomaly other than chromosome 22 a strong indicator of an aggressive meningioma that must be followed closely (**Fig. 1.16**).

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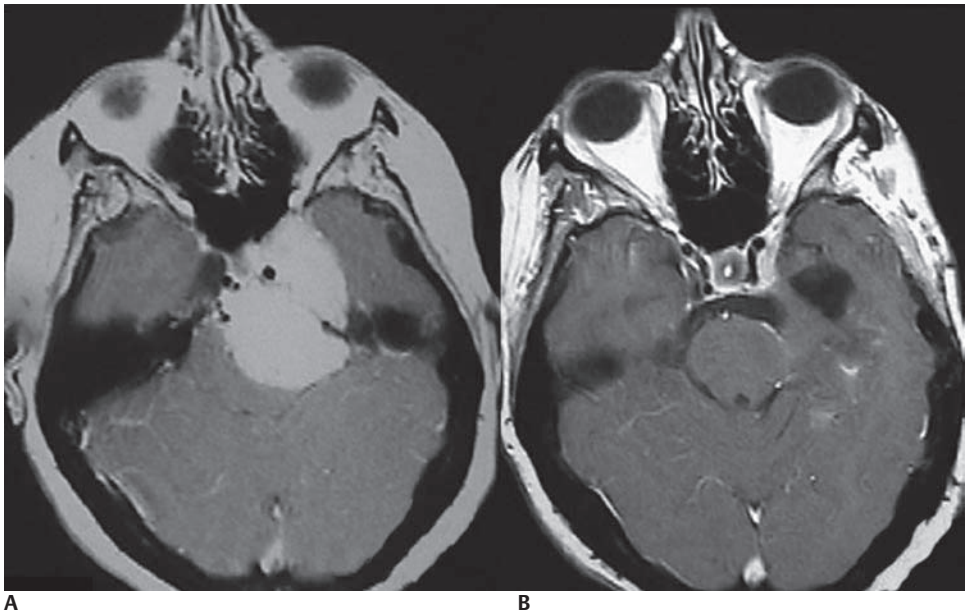


Fig. 1.11 A sphenopetroclival meningioma with invasion of the cavernous sinus. A skull base approach facilitated total removal. **(A)** Preoperative enhanced axial MRI. **(B)** Postoperative axial magnetic resonance imaging.

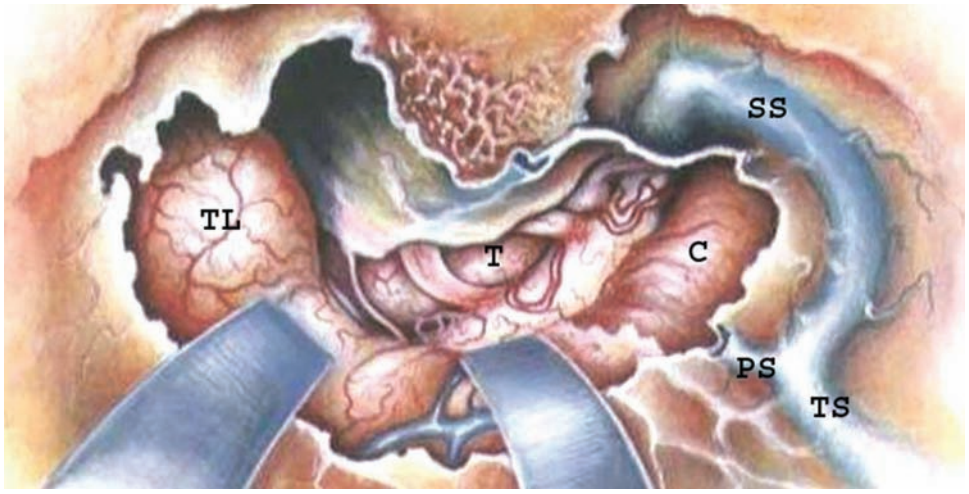


Fig. 1.12 Artist's illustration demonstrating the superb exposure obtained by mobilizing the sigmoid sinus through the posterior petrosal approach. Used with permission from Al-Mefty O. *Operative Atlas of Meningiomas*. Philadelphia, PA: Lippincott-Raven; 1998:304. TL, temporal lobe; T, tumor; C, clivus; SS, sigmoid sinus; PS, petrosal sinus; TS, transverse sinus.

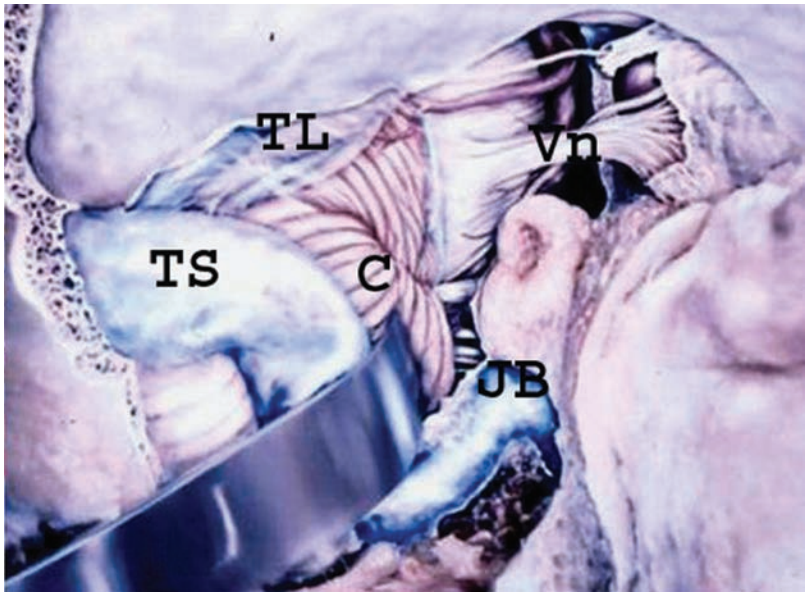


Fig. 1.13 Artist's illustration of double petrosal approach, anterior and posterior, which enables the total removal of clivus meningiomas while preserving hearing. Used with permission from Cho CW, Al-Mefty O. Combined petrosal approach to petroclival meningiomas. *Neurosurgery* 2002;51(3):712. TS, transverse sinus; TL, temporal lob; C, clivus; JB, jugular bulb.

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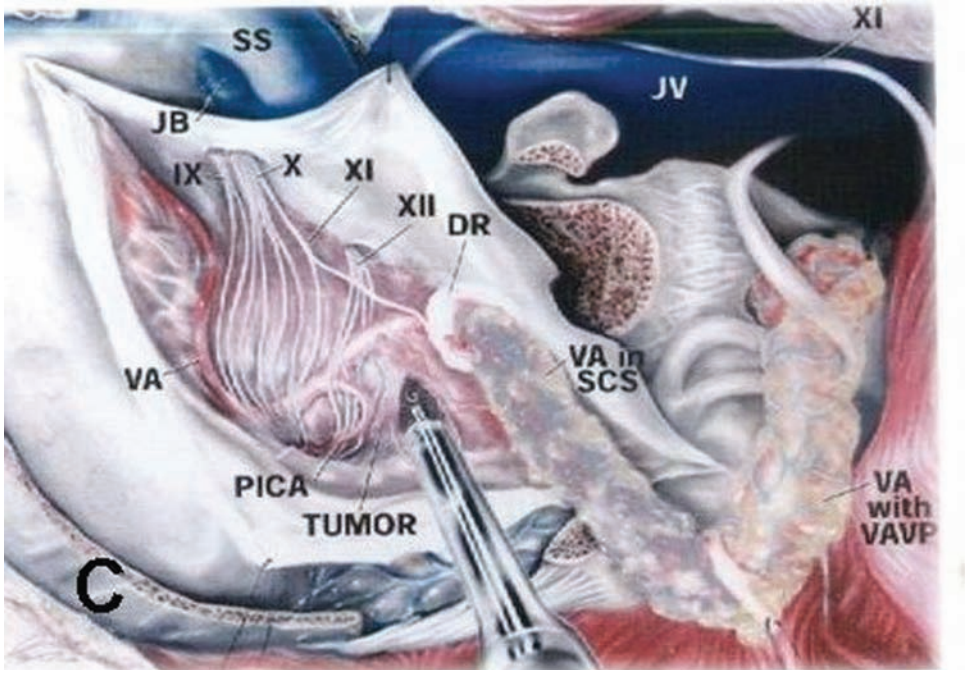
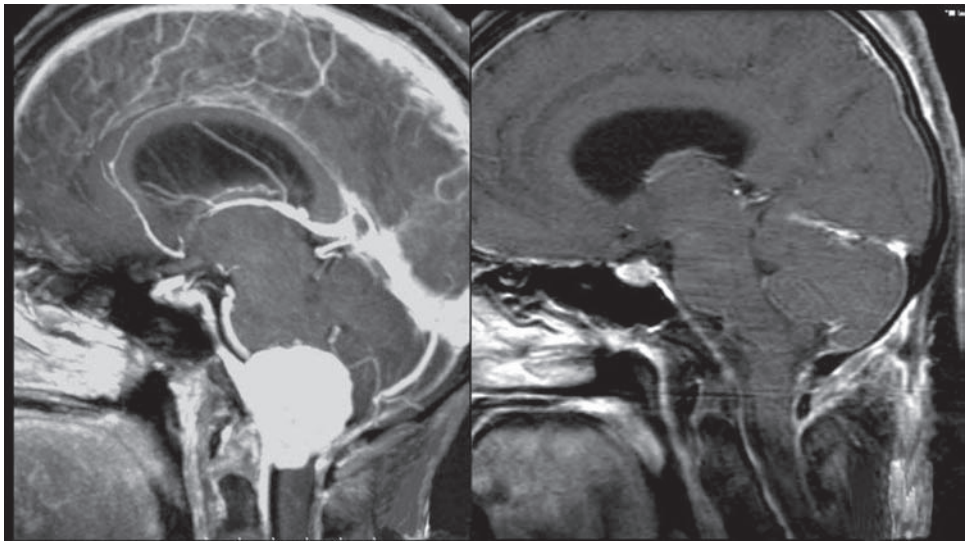


Fig. 1.14 Artist's illustration of the fine exposure obtained through a transcondylar approach to a ventrally located foramen magnum meningioma, facilitating its total removal. Used with permission from Arnautovic KI, Al-Mefty O, Husain M. Ventral foramen magnum meningiomas. *J Neurosurg* 2000;92(1 Suppl):72. VA, vertebral artery; PICA, post inferior cerebellar artery; JB, jugular bulb; SS, sigmoid sinus; SCS, suboccipital sinus; JV, jugular vein; DR, dural ring; VAVP, vertebral artery venous plexus.



A B

Fig. 1.15 Magnetic resonance imaging (MRI) of a large ventral foramen magnum meningioma totally removed through a transcondylar approach. **(A)** Preoperative sagittal enhanced MRI. **(B)** Postoperative sagittal MRI demonstrating complete removal.



A B

Fig. 1.16 Cytogenetic analysis is the most reliable predictor of meningioma behavior. **(A)** Meningioma with a normal karyotype indicative of a benign course. **(B)** Meningioma with monosomy 22 and additional cytogenetic abnormalities indicative of aggressive behavior.

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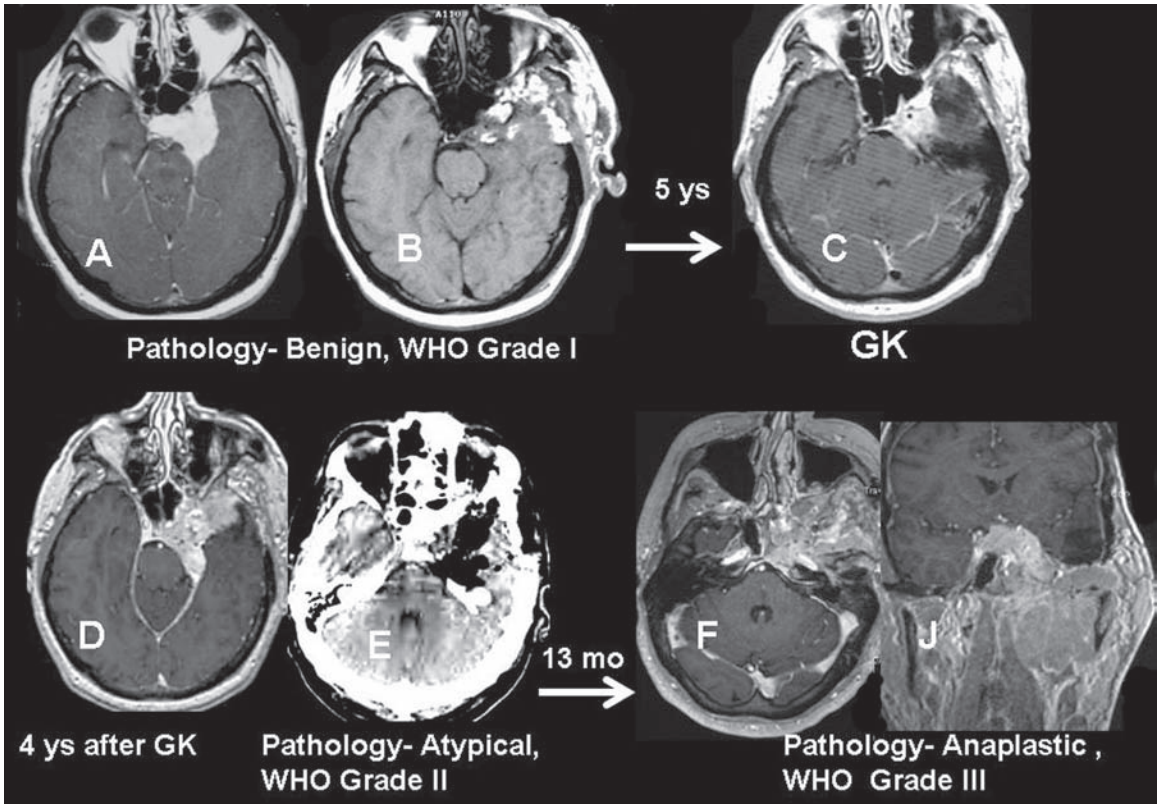


Fig. 1.17 A case of meningioma progression to malignancy with a history of radiation administered to residual tumor. **(A)** Magnetic resonance imaging (MRI) of the original tumor, which was a grade I benign tumor. **(B)** Postoperative residual tumor remaining around the cavernous carotid. **(C)** MRI at the time of treatment with gamma knife (GK) for residual tumor. **(D)** Recurrent tumor after radiosurgery. **(E)** Postoperative MRI of surgical resection of a now aggressive grade II meningioma. **(F)** Axial and coronal MRI of a massive recurrence with now grade III anaplastic meningioma.

◆ Malignant Progression

Nowell established the cytogenetic evolution theory that is the basis of tumor progression to malignancy.[37] Cells from the original clone can acquire additional genetic alterations, which permit a stepwise selection of variant subclones with a greater growth advantage. This phenomenon of tumor progression has been a great interest of mine (OA) spurred by an increasing number of cases that have undergone previous attempts at treatment, often repeatedly, with various combinations of surgery, radiation, radiosurgery, chemotherapy, and medical treatment. As these patients reach the end of hope, after a long struggle, their meningioma shows the classic form of tumor progression. Their pathology transforms from a benign WHO grade I to a frankly malignant WHO grade III, and their cytogenetic studies show accumulation of acquired changes. Our experience is consistent with Jääskeläinen et al's finding that up to 28.5% of recurrent meningiomas transform to a malignant variety.[38,39] The period of time that elapses between the appearance of a tumor and its progression to malignancy is variable. This progression is more common and of a shorter time frame in the conversion from atypical tumors to anaplastic ones.[40] What is even more striking is that meningiomas with malignant transformation fare worse in outcome than de novo malignant and atypical meningiomas.[41] In many cases of tumor progression associated with accumulated cytogenetic aberrations, radiation therapy has been previously administered (**Fig. 1.17**). The role of radiation in this transformation requires additional scrutiny. Notwithstanding that some meningiomas that progress contain cytogenetic aberrations while histopathologically benign,[39] one cannot escape the thought that the administration of radiation to these benign tumors might promote malignant transformation. Such has been reported, as rare as it might be, for other tumors.[18] Radiation's effect is known to be through damage to cellular DNA and is proven to induce meningiomas. These radiation-induced meningiomas occur after high- or low-dose radiation; are very aggressive; occur at a younger age; are more frequently associated with atypical or malignant histopathology and multiple cytogenetic aberrations; recur 100% of the time; and fully exemplify the process of tumor progression.[39]

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Chapter 2

Meningioma Surgery During the Twentieth Century

Anna R. Terry and Fred G. Barker II

There is today nothing in the whole realm of surgery more gratifying than the successful removal of a meningioma with subsequent perfect functional recovery. . . . The difficulties are admittedly great, sometimes insurmountable, and though the disappointments are many, another generation of neurological surgeons will unquestionably see them largely overcome. —Harvey Cushing (1922)[1]

Before the Twentieth Century

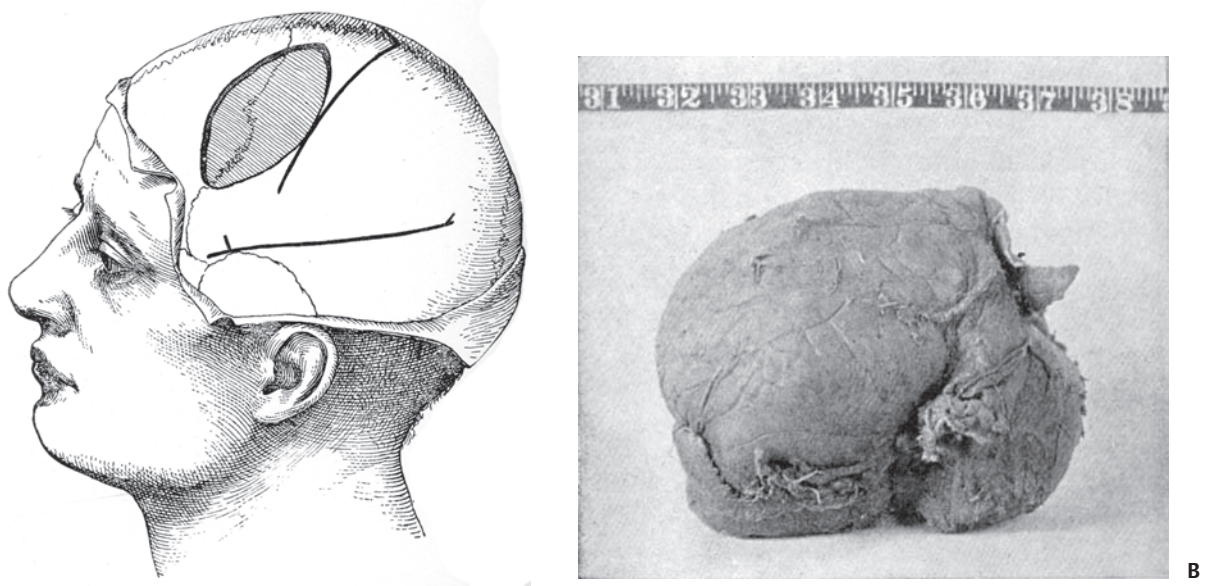
The intracranial tumors we now call *meningiomas* existed before the start of recorded history, which we know because of the unique changes they sometimes induce in the adjacent skull. The oldest paleopathological evidence of a hyperostosing meningioma is a skull excavated in southwestern Germany estimated to be 365,000 years old,[2] and many additional specimens have been preserved from North and South America, Europe, and Africa.[3] There is no written record of a tumor that can be recognized as a meningioma until the seventeenth century, when a patient who died with progressive dementia was found at autopsy to have a hard, rounded extraaxial mass adjacent to the falx that had not infiltrated the brain.[4,5] However, before the advent of modern imaging, the presentation of meningiomas, as with other intracranial lesions, was an impenetrable mystery unless the tumor caused some external sign such as a hyperostotic mass. Clinical signs such as headaches, vomiting, visual changes, hemiparesis, and seizures certainly could have been observed but would not have been well enough understood to support a specific diagnosis. Indeed, many meningiomas are clinically silent, and only symptomatic cases would have come to medical attention before modern imaging.

The first documented attempts at surgical removal of extraaxial convexity lesions, some of which were probably meningiomas, took place in the eighteenth century. One French series of some 20 patients having hyperostotic skull lesions likely included osteomyelitis and skull metastases as well as meningiomas.[6] Early attempts at resection were severely hindered by the lack of anesthesia, antisepsis, and appropriate surgical tools. Some surgeons merely exposed the tumor surface and applied a caustic solution to it, allowing the wound to heal by granulation—if it healed at all. Other surgeons performed a craniotomy around the mass by drilling holes and connecting them with a saw, then cauterizing or ligating the tumor. The French master surgeon Antoine Louis used this method to perform a complete resection of a parietal meningioma in at least one patient, treating the open tumor with "Malmsey wine mixed with herbs and rose honey" until the underlying tissue became necrotic and was able to be removed in several stages.[7] The patient apparently survived.

Obviously, these treatments were fraught with danger and only the most courageous or desperate patients would have submitted to such an ordeal. Several documented operations in the eighteenth and nineteenth centuries ended in the patient's death within minutes or hours.[6,8] Inadequate exposure (**Fig. 2.1**) was common because the externally apparent, hyperostotic portion of the tumor might not reflect the full extent of the underlying intracranial mass. Lack of modern histological examination makes it impossible to be sure what types of tumors these early surgeons actually took in hand, and many of these masses were probably syphilitic gummas, skull metastases, plasmacytomas, and other types of pathology rather than meningiomas as we know them today.

Advances in pathology and physical diagnosis during the eighteenth and nineteenth centuries, especially in France, made it possible to correlate histories, symptoms, and physical examination findings during life with autopsy findings. Jean Cruveilhier's *Anatomie pathologique du corps humain* (published 1829–1842) included detailed illustrations of meningiomas, accurately describing their typical locations as the convexities, petrous bone, olfactory groove, middle fossa, tentorium, and falx, and correlating them with symptoms and physical findings.[9] At least in theory, these advances laid the groundwork for diagnosing such a tumor by neurological examination alone. It would be several decades before this was actually achieved, but during the nineteenth century several successful operations for dural-based tumors with over-

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A B

Fig. 2.1 Inadequate exposure in early meningioma surgery. Operation by Robert McBurney for convexity meningioma, 1891. **(A)** Diagram indicating location and size of craniotomy. **(B)** The operative specimen. A 6 cm tumor was removed en bloc with finger dissection although the tumor "extended in every direction beyond the opening." The patient died 8 hours after surgery. From Starr MA, 1893.[16]

lying hyperostoses or palpable masses were performed, by Pecchioli in 1835,[10] by Macewen in 1881 (perhaps a syphilitic gumma),[11] and most famously in 1885 by Francesco Durante, a professor of surgery in Rome who successfully diagnosed and removed a left olfactory groove meningioma in a 35-year-old woman with anosmia, a left third nerve palsy, downward displacement of the left eye by the tumor, and cognitive and mood changes.[12,13] This operation, which lasted 1 hour, resulted in the complete resection of a 70 g tumor. The surgical cavity was drained through the left nasal cavity via an opening in the ethmoid sinus, which had been eroded by the tumor. The patient made a good recovery, even after undergoing a second operation for recurrence 11 years later, and was still alive and in good health 20 years after her initial operation.[12] The case gained renown for its satisfying surgical result and favorable neurological outcome, and was published in the *Lancet*[13] and presented at the International Medical Congress in Washington, D.C., in 1887. At least another two dozen operations on hyperostotic tumors that were probably meningiomas were recorded in the last decades of the nineteenth century.[6]

The Early Twentieth Century: Before Cushing

By the beginning of the twentieth century, the three necessary ingredients for minimally successful brain surgery were firmly in hand. Anesthesia had been available since the late 1840s, Lister's antiseptic methods since the 1860s, and Ferrier's advances in cerebral localization since 1876. But not until ~1910 would the next important technical advances in brain surgery become available—Cushing's hemostatic methods and his understanding and early control of intracranial pressure. During this interval it became increasingly possible to localize many enlarging intracranial mass lesions, particularly those located near the primary motor cortex, by means of Jacksonian seizures, localized weakness, and the ophthalmoscopic diagnosis of elevated intracranial pressure. Surgeons were willing to operate despite the dangers, because of the known dismal natural history under medical treatment alone. But early mortality rates were very high. Of 38 brain tumor cases collected in an 1894 review, 20 (53%) had ended in early postoperative deaths.[14] In reaction to this situation some neurologists and surgeons attempted to specify "requisites for operability" for brain tumors, and then to use autopsy data to define the percentage of brain tumor patients who might conceivably benefit from operation. Ernst von Bergmann specified that a truly operable tumor must be not too large, not metastatic, not multiple, encapsulated, and exposed on the surface of the convexity.[15] Using similar criteria, Starr estimated in 1893 that ~7% of brain tumors were operable.[16] In 1905 Walton and Paul classified only 3.3% of 424 tumor cases as definitely operable—"primary, accessible, well defined tumors which may be removed without cutting into brain tissue . . . [with] distinctive symptoms."

From our present perspective, these criteria essentially limit the field of "operable tumors" to convexity meningiomas, but this diagnostic entity had not yet been defined. Still, in retrospect, we can identify two operations undertaken in the United States in the late-nineteenth century for meningiomas based on cerebral localization, rather than using an external bony swelling as a landmark.[17] Robert Weir's patient presented in 1887 with homonymous hemianopsia and papilledema, and an occipital tumor was diagnosed. Through an occipital craniotomy, a large fleshy mass (~9 by 7 cm) that presented on the

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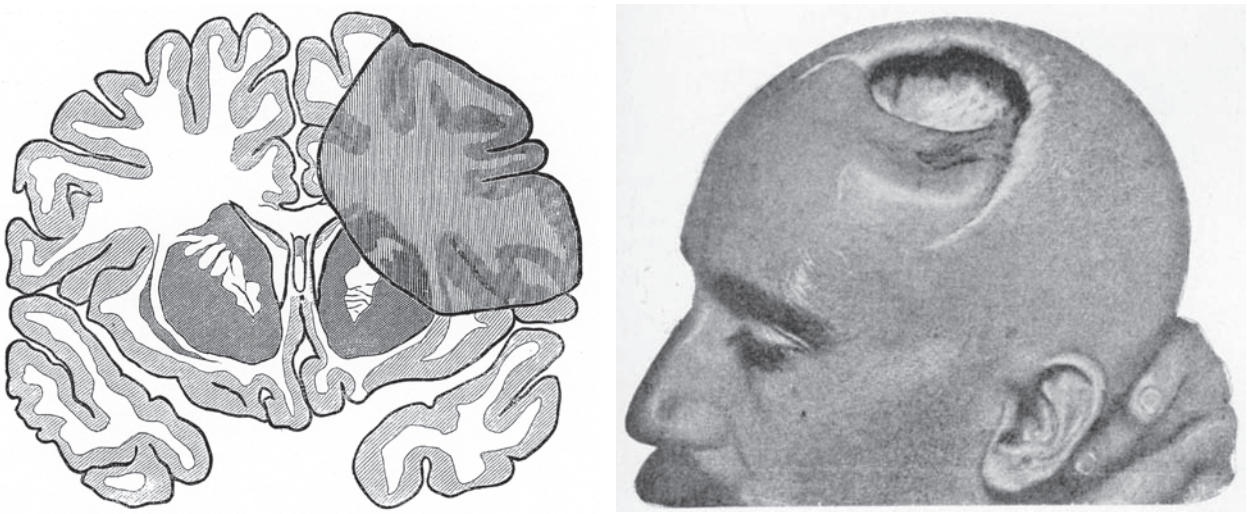


Fig. 2.2 Keen's successful operation for convexity meningioma (1887). **(A)** In a two-stage procedure, the tumor (outlined) was removed through **(B)** the craniotomy. The patient survived for more than 30 years. From Keen, 1888[19] and Keen et al, 1903.[99]

convexity was enucleated with a spoon, and the shell was fractured off its attachment to the falx using finger dissection. The patient died that night from postoperative hemorrhage.[18] W.W. Keen's patient, also in 1887, presented with headaches, papilledema, progressive aphasia, and right hemiplegia beginning in the arm; Keen diagnosed a left frontal tumor. In a 2-hour operation, he completely removed an 88 g left frontal convexity meningioma by dissecting around it with his little finger. After a lengthy convalescence, marked by poor wound healing, the patient recovered and lived for 30 years without recurrence (**Fig. 2.2**).[19,20] For Keen, at age 50 one of America's leading surgeons, it had been his "very first modern brain operation . . . my heart 'sank down into my boots.' But dangerous as the procedure might be I had to go ahead." Harvey Cushing, at the end of his own career, wrote that Keen's "vivid account" should be read "by every beginner who may . . . feel trepidation at the task ahead of him."[21]

◆ Cushing's Contributions

Today, successful surgical treatment of most meningiomas is a standard expectation of medical care, but a century ago, the favorability of these tumors to surgical management was at best a theoretical extrapolation from what was then known about their pathology, etiology, and location. At that time, only a handful of surgical cases had been described in the literature, most with daunting mortality rates (46.6% in Victor Horsley's series of 1913).[22] It was known, principally from Horsley's relatively few survivors, that the long-term recurrence rate of completely excised meningiomas (generally called "meningeal endotheliomas" or "sarcomas" during this period) was very low.[22] By 1906, Horsley considered that "all tumors which, growing from the meninges, penetrate the brain, or which are encapsulated . . . can all be excised with a good permanent result."[23] Cushing, before having a single personal case of meningioma, wrote in 1908 that they "offer[ed] the most favorable type of tumor for operation."[24] The challenge was clearly framed: successful complete excision might represent a cure, if the patient could only survive the surgery.

The cumulative contributions of one neurosurgeon, Harvey Cushing, over his career stand alone in their significance for the safe and successful surgical management of meningiomas. Cushing's 1938 monograph *Meningiomas: Their Classification, Regional Behavior, Life History, and Surgical End Results* describes in great detail 313 patients who underwent surgical treatment of their meningiomas between 1903 and 1932.[21]

Just as successful diagnosis and appropriate surgical exposure were crucial obstacles to successful surgical resection of meningiomas before the twentieth century, the next great hurdle to be overcome was achieving adequate hemostasis. Due to the possibility of severe and often fatal blood loss, as well as the inadequacy of anesthesia, most cranial operations before the twentieth century were shockingly brief by today's standards—less than an hour in many cases. Cushing demonstrated an acute recognition of the importance of hemostasis and was a careful, meticulous, even slow surgeon by the standards of the time.[25,26] In his words, "More operative fatalities have been due to exsanguination and its consequences than all other causes combined." This was especially true of meningiomas, due to their frequent vascularity and proximity to the dural venous sinuses. Dandy, a fearless surgeon who was technically without peer, felt that meningiomas "offer[ed] the most difficult battles in cranial surgery."[27]

Cushing observed his first meningioma resection as a medical student. The procedure was performed by John Elliot at the Massachusetts General Hospital on a 31-year-old man with a growing vertex skull lesion and vision and gait problems. The operation was conduced in two stages. During the first stage, a portion of the bony mass was removed, but blood loss was so significant that the sec-

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ond stage had to be delayed by 3 weeks. Cushing served as the anesthetist for the second stage, using chloroform as recommended by Horsley. The operation, which lasted 90 minutes, involved removal of the residual tumor with "hammer, chisel, scissors, and fingers."[28] The patient died half an hour later, presumably from massive blood loss and shock. Autopsy confirmed tumor infiltration of the sagittal sinus.[8]

Perhaps impelled by this early experience, Cushing helped popularize the use of intraoperative cardiorespiratory monitoring and fluid resuscitation, frequently arranging for a typed blood donor to be available in case a transfusion was needed, and experimenting with autotransfusion of blood recovered from suction in the operating room.[28] Many operations were conducted under local anesthesia to avoid the hypotensive effects of general anesthesia. Early in his career, Cushing discarded chloroform as a general anesthetic in favor of ether, which did not have as severe a hypotensive effect.

Technical innovations specifically addressed hemostasis, as with his use of silver clips to ligate cortical vessels from 1910 onward.[29] Raw muscle (from another operating room—usually pectoralis muscle from a radical mastectomy for cancer—or from the patient's own leg)[30] was used as a topical hemostatic agent due to its high concentration of fibrin. The introduction of Bovie's electrocautery in 1926 made possible the surgical management of tumors previously considered unresectable, enabling the control of excessive bleeding and the removal of tumor core with wire loops.[26,31,32] Cushing felt the device was such an important advance that he brought back several patients whose first attempted resections had been limited by uncontrollable bleeding, attempting to complete resections he had previously considered impossible. The difficult cases increased his operative mortality for a period while he learned the limits of the new technique.[28]

Cushing initially attempted en bloc resections of meningiomas by preference, sometimes also removing adjacent involved bone, leaving significant dural defects, which he closed with fascial grafts. However, he eventually adopted a strategy of debulking the center of the tumor, often with electrocautery, before removing its capsule, a technique still commonly used today.[21,33]

Many of these innovations are illustrated in Cushing's final meningioma case, from 1938, which involved a very large (9 cm) tumor in a 45-year-old woman. The procedure was performed under local anesthesia. Although systolic blood pressure at one point dropped to 40 mm Hg, the tumor's arterial supply was successfully defined and severed, and a complete resection was achieved by debulking the core and stripping away the capsule. The patient was kept on the operating table for several hours for close blood pressure monitoring, and thereafter made a complete recovery, returning to her job as a decorator. Routine skull x-rays showed no signs of recurrence up to 5 years after surgery.[28]

Cushing's contributions are notable not only for innovations in surgical technique but also for exhaustive attention to detail, particularly with respect to the clinical examination. Like his mentors William Osler and Henry Thomas, he ascribed great importance to accurate and complete description of the neurological examination. He was thus able to define coherent clinical syndromes relatable to meningiomas of the convexities, suprasellar region, olfactory groove, sphenoid wing, falx, and parasagittal region as well as the cerebellopontine angle, thus aiding in informed, intelligent surgical exposure of these tumors. In collaboration with his assistant Louise Eisenhardt, he conducted detailed follow-up on his patients, slowly amassing invaluable data on the long-term outcomes of his cases. Eisenhardt began collecting survival data prospectively on all of Cushing's tumor patients beginning in 1922, with enough accuracy to enable retrospective calculation of Kaplan-Meier curves.[28] In anticipation of the contemporary standards of brain tumor treatment, these data included information on quality of life as measured by a return to "useful work" or the patient's previous occupation: "the mere lengthening of a patient's months or years without making them more livable is . . . no justification whatsoever of an operative procedure."[34]

This attention to long-term follow-up is apparent in the story of Cushing's most famous meningioma patient, General Leonard Wood. Wood was a military surgeon, originally one of Theodore Roosevelt's Rough Riders and renowned for his courage in battle. He had a long history of a right frontoparietal skull-based mass, for which he underwent an extracranial procedure with a diagnosis of "fibrosarcoma." Intractable left-sided motor seizures led him to seek a second opinion from Cushing in 1910, thereby making him the third patient in Cushing's series. In two operations, performed 4 days apart, Cushing successfully removed a right parasagittal meningioma.[21,35] Wood was discharged from the hospital a month later and went on to make a full recovery, later becoming governor of Cuba, serving as President Taft's chief of staff, and running for president in 1920 as the original Republican favorite to succeed Woodrow Wilson. Eventually, he experienced renewed left-sided seizures and underwent a second resection of his tumor in 1927. Unfortunately, he experienced an intraventricular hemorrhage and died a few hours after the operation, a tragedy that greatly distressed Cushing.[25] Wood's brain is preserved at the Yale University School of Medicine.

Mid- to Late-Twentieth Century

In its general principles, surgical removal of meningiomas has changed less in the decades since Cushing than it did during his career. Meningioma surgery still involves devascularization of the tumor mass, internal debulking, and maximal safe removal, including the dural attachment where possible. Many incremental technical innovations, such as the pneumatic drill,[36,37] bipolar cautery,[38] and the operating microscope[39] and endoscope,[40] have made procedures progressively easier for the surgeon and safer for the patient. Few of these innovations are used exclusively for meningioma surgery, and most are described more extensively in works on general neurosurgical history.[41–44] We will summarize some twentieth-century technical advances that are important in meningioma surgery.

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Hemostasis and Devascularization of the Tumor

Cushing's specific hemostatic contributions, such as "silver clips"[29] and electrocautery,[31–33] added to Horsley's bone wax for bone hemostasis,[45,46] made much modern meningioma surgery possible. Cushing also adopted Horsley's use of muscle as a local hemostatic application.[47] Various substances, such as oxidized cellulose,[48] fibrin,[49] and gelatin sponge,[49] have since been added to the neurosurgical armamentarium for local application. Bipolar electrocoagulation has replaced Bovie's monopolar instrument for much modern intracranial surgery.[38,50]

Other techniques involve interruption of the tumor's blood supply more proximally. Direct surgical attack on the vascular supply was used by Cushing[21] and Olivecrona,[51] both of whom ligated the external carotid artery for selected cases; Cushing considered ligation of the *internal* carotid in the neck advisable before resecting meningiomas "anchored" by the intracranial carotid artery.[21] Ligation of the middle meningeal artery for tumors in the middle fossa base became common practice by the 1950s. Surgical interruption of smaller arteries that feed basal tumors (e.g., ethmoidal arteries) has also been described.[52,53] More commonly, endovascular techniques are used to interrupt arterial feeders or to embolize smaller arteries within the tumor using particulate materials. Early descriptions of meningioma embolization began in 1973 with the work of Djindjian and colleagues using superselective catheterization.[54,55]

Central Debulking

Some early surgeons removed convexity meningiomas en bloc,[19] even removing a layer of normal brain around the tumor in some cases, whereas others morcellated the masses or even removed just the central portion, leaving the tumor capsule behind.[43] Cushing's early meningioma resections were typically conducted en bloc to reduce the risk of recurrence.[21,28] Perhaps Halsted's radical mastectomy for breast carcinoma influenced his thinking; he also had documented cases of postoperative meningioma recurrence as subcutaneous tumor nodules growing in the operative wound.[30] In contrast to earlier surgeons who used digital dissection, a teaspoon, or blunt curved scissors to separate the tumor capsule from surrounding brain,[16,56] Cushing favored a technique in which the cortex was "brushed away" from the tumor with wet cotton[30]; bridging vessels were then clipped and divided individually so that the tumor could at last be "tilted out" of its cortical "nest."[21] Other surgeons adopted similar methods.[57]

Tumors located at the cranial base could not generally be removed en bloc without damaging surrounding structures, and by the late 1920s Cushing was using electrocautery (the Bovie loop) to debulk the tumor centrally before collapsing the tumor capsule inward and dissecting it from surrounding structures (**Fig. 2.3**).[32,33] The technique was based on his earlier use of central debulking developed for subtotal resection of acoustic neuromas[58] but with the addition of resection of the tumor capsule.[59] Although most surgeons adopted these methods, Walter Dandy favored resection of one frontal lobe to expose olfactory groove and tuberculum sellae tumors; operative descriptions and photographs of the specimens suggest an en bloc removal was then done.[27,60] He did sometimes perform intracapsular "enucleation" for deeper tumors, but only rarely for convexity lesions.[27]

As with hemostasis, subsequent years have brought better technical methods of debulking tumor masses. Ultrasonic aspiration[61,62] and various types of laser[63,64] have been most commonly used.

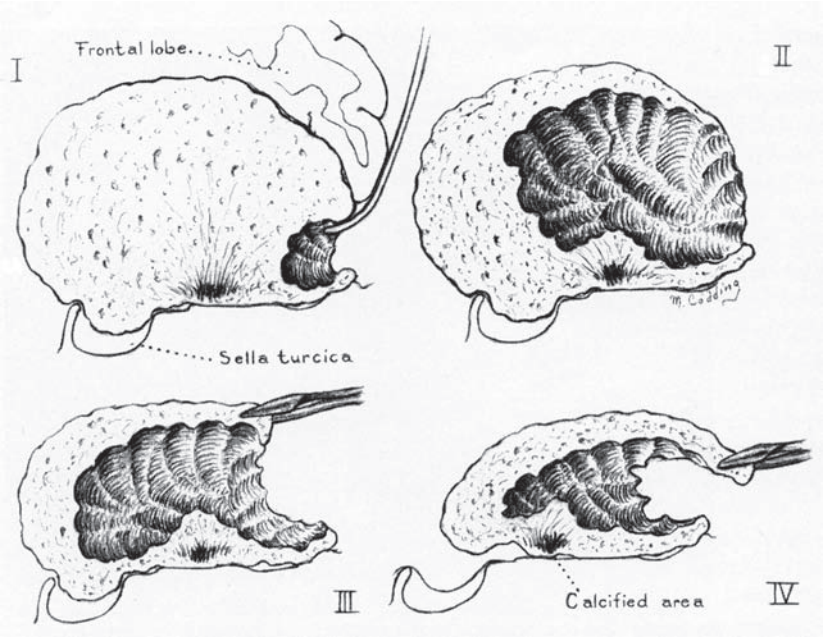


Fig. 2.3 Cushing's method of central debulking of an olfactory groove meningioma using electrosurgery (Bovie). From Cushing, 1928.[32]

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Involvement of Adjoining Structures: Bone and Blood Vessels

One of the earliest-noted unique characteristics of meningiomas, as already noted, is the tendency to induce hyperplastic changes in adjacent bone. Cushing was aware that the hyperostotic bone contained tumor cells in the Haversian canals, as were others before him (**Fig. 2.4**).[65,66] He sometimes achieved a procedure in which the bone flap was allowed to remain attached to the affected dura, and the tumor, dura, and bone were removed as a single specimen.[21] Complete removal of the affected bone was felt to be necessary to eliminate a recurrence risk. This was problematic when the bony changes involved basal structures such as the sphenoid wing, but surgeons became increasingly aggressive about removing the infiltrated bone, first with osteotomes and later with high-speed drills.[67] Another approach, first reported in 1923 by Phemister, was to sterilize the involved flap by immersion in boiling water before replacement.[68]

The other structure abutting many meningiomas that has received much special attention is the superior sagittal sinus, often called the longitudinal sinus in older literature. As a medical student in 1895, Cushing saw a meningioma that involved the sinus resected after the sinus was ligated on either side of the tumor; the patient died soon after surgery.[28] By 1910 he appreciated the occasional involvement of the sinus by meningiomas and 5 years later he had devised a means of both resecting and repairing a corner of the sinus.[21] When the sinus was completely occluded by tumor he would open it, remove the tumor from the lumen, and replace it with muscle plugs at either end.[43] His willingness to resect the involved sinus in its anterior third, with the opinions of his contemporaries on the subject, has recently been reviewed.[69] Olivecrona, who had extensive experience with parasagittal meningiomas, felt that an extensively occluded sinus should not be resected to avoid interrupting collateral venous drainage through the adjacent falx.[51] Much more recently, the ability of cavernous sinus meningiomas to invade the wall of the cavernous carotid artery has been demonstrated and adduced as a reason to resect that artery in the pursuit of complete tumor removals.[70]



Fig. 2.4 Figure illustrating the various types of hyperostosis due to meningiomas. From Cushing, 1922.[1]

Radiological Diagnosis of Meningiomas

The twentieth century saw immense advances in imaging methods of brain tumor diagnosis, many of which are applicable to meningioma diagnosis.[71] Meningiomas were often the earliest tumors detected with new radiographic methods because of their effects on neighboring bone and their intrinsic propensity to calcify, so many are visible on x-ray studies even without contrast. Charles Mills reported the first radiographic diagnosis of a meningioma in 1902, less than a decade after Roentgen first reported the new rays.[72] Real experience with x-ray diagnosis in larger numbers of meningiomas was reported in 1925 from Cushing's clinic by Sosman and Putnam, who found that about half of 25 meningiomas studied showed some characteristic radiographic change, such as bone erosion or enlarged vascular channels, osteomatous changes, spicule formation, or calcification within the tumor.[73] Basal growths were sought using oblique and stereoscopic views.[74] By the mid-1920s Cushing had begun to use routine skull radiographs to monitor his postoperative meningioma patients, interpreting any shift over time in the location of the silver clips left after the resection as evidence of a recurring mass.[21]

Because of the extraaxial location of most meningiomas, the air or hyperdense contrast methods such as ventriculography and pneumoencephalography introduced during the 1920s by Dandy were less helpful in meningioma cases than in deep-seated gliomas, except in tumors arising from the falx or anterior skull base.[27] Cerebral angiography for meningiomas was more commonly helpful and rapidly gained widespread use after its introduction in 1927 by Moniz and its popularization in North America by List around 1945.[75] Features of meningiomas on angiography were first reported by List and Hodges in 1947 (20 cases).[76] Cross-sectional imaging of meningiomas was reported soon after the initial clinical use of computed tomography (CT) in the early 1970s, with a small series described in 1975.[77] The initial impression was that angiography would continue to be useful for most patients, but the rapid advances in CT quality followed by the advent of magnetic resonance imaging (MRI) have made angiography superfluous for most meningioma patients today, except in the context of embolization treatment.

Radiation Treatment and Radiosurgery

Cushing and Dandy seem not to have used radiation treatment on meningioma patients; Dyke and Davidoff (1942), Simpson (1957), and Northfield (1973) considered radiation treatment of meningiomas as being of little apparent value.[78–80] By the mid-1970s radiation treatment was coming into wider use in some clinics, with favorable reports documenting decreased recurrence rates being published in the 1980s and later.[81–83] Early experience with radiosurgery was reported in 1989,[84] and this treatment has also come into widespread use. Brachytherapy[85] has not been widely used for meningioma treatment.

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Meningioma Themes at the Turn of the Twenty-first Century

As historical reviews work their way closer to the present, the clarity and the impartial detachment of hindsight are no longer available to the historian, but perhaps some recent general themes in meningioma progress can be loosely sketched. In the last 2 decades of the twentieth century, two strong opposing forces driving meningioma treatment emerged. As a consequence of more secure data showing that extent of resection was the most important predictor of disease-free long-term survival, surgeons pushed harder for greater extent of resection in previously difficult locations. At the same time, an increasing consciousness of the paramount importance of quality of life led some surgeons to decrease surgical aggressiveness while searching for effective salvage strategies, such as radiosurgery. The tension between these poles is still relevant for the chapters in this book on current treatment.

Aiding the forces opposing maximal resection is the awareness that many incidentally discovered meningiomas in modern practice need no treatment at all, only watchful waiting. This management tactic is largely a product of the recent widespread use of cross-sectional imaging such as CT and MRI for patients without specific neurological findings—those with headache, dizziness, and minor head trauma. Before this imaging era, essentially every meningioma discovered would be sufficiently large or critically located as to cause symptoms that might justify treatment. Now, increasing numbers of patients have incidentally discovered small convexity lesions that should be followed over time but not necessarily treated. The natural history of untreated meningiomas began to be studied seriously in the 1980s.[86] With the constant clinical reminder that small meningiomas can be observed instead of requiring immediate treatment came a sense that small remainders after subtotal resections of benign meningiomas could be safely watched,[87] as well as (perhaps) microscopic disease after gross total resections of atypical lesions.[88] Additional arguments against aggressive surgery would come from safe and effective adjuvant treatments for growing postoperative meningioma remnants, but aside from radiation, at the close of the twentieth century none existed.[89]

For a more nuanced picture of important themes in meningioma research we can turn to citation analysis (ISI Web of Science, Thomson Reuters, New York, NY). As of April 2010, the most-cited single work on meningiomas was Cushing and Eisenhardt's classic 1938 text *Meningiomas*, with more than 1200 citations. The most-cited single journal article (almost 700 citations) was Simpson's 1957 correlation of extent of meningioma resection with subsequent recurrence risk,[79] and two other articles in the ten most cited also treated the risk of postoperative recurrence in relation to extent of resection and other factors such as location, age, and grade.[90,91] Three of the ten most-cited articles addressed cytogenetic or molecular genetic alterations in meningiomas.[92–94] One article each on growth factors in meningiomas, quality of life after meningioma surgery, and radiation for meningiomas, and a technical article on skull base approaches completed the list of ten most-cited articles.[81,95–97]

The results of the intensive study on meningioma treatment deployed during the twentieth century are impressive. From almost 50% operative mortality in Victor Horsley's series at the end of the nineteenth century,[22] mortality rates decreased progressively during Cushing's career[21] and subsequently. By 2000, the in-hospital mortality after meningioma resections in U.S. hospitals had fallen to 1.8%.[98] Important challenges for the future include improving results for atypical and malignant meningiomas, finding effective medical treatments for patients with neurofibromatosis type 2 and other multiple and unresectable meningiomas, and improving quality of life for patients requiring surgical resection of these challenging tumors.

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II

Anatomy and Pathology

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Chapter 3

Anatomy and Biology of the Leptomeninges

Michael C. Huang and Harry R. van Loveren

Why Is the Anatomy of the Meninges Critical to a Book on Meningiomas?

Meningiomas are the most common benign intracranial neoplasms, accounting for 13 to 26% of all intracranial tumors.[1] Because they originate from meningothelial cells found in the arachnoid layer of the meninges, the successful management of meningiomas demands comprehensive understanding of the anatomy of the meninges. Knowledge of meningeal architecture and its osseous and neurovascular relationships provides the basis for understanding the sites of origin and the routes of spread of meningiomas. Successful operative strategies must account for both the tumor itself and its dural attachments. Only by eliminating the affected dura and bone can the risk of recurrence be minimized.[2]

Familiarity with meningeal anatomy allows for the design of safe surgical approaches to complex regions of the cranial base while minimizing neurovascular injuries. Intraoperatively, the arachnoid layer provides an avascular plane of dissection, separating tumor from vital neurovascular structures. When meningiomas violate the arachnoid–pia barrier, as can be predicted by abnormal signal changes in brain parenchyma on magnetic resonance imaging, gross total resection becomes less likely. Advancements in our knowledge and appreciation of the anatomy of the meninges have driven the evolution of the surgical treatment of meningioma, including the development of advanced skull base techniques.

The term *meninges* is attributed to Erasistratus (304–250 BC), the Greek anatomist and royal physician to Seleucus I Nicator of Syria. Along with fellow physician Herophilus, Erasistratus performed human dissections in their school of anatomy in Alexandria and brought objectivity through direct observation to the study of anatomy. Their studies confirmed the findings of Aristotle (384–322 BC), who noted a dual-layered membrane surrounding animal brains, one layer opposing the skull and the other following the contours of the brain. The Greeks, however, were not the first to describe the coverings of the brain. In the Egyptian trauma surgery text of the Edwin Smith Papyrus (ca. 2200 BC), the presentation of a head trauma patient included descriptions of membranes enveloping the brain that when torn would release the "fluid of the interior of the head."[3]

Although the presence of the dura and pia mater were well known in antiquity, the weblike arachnoid was not recognized until it was first reported by the Dutch anatomist Gerardu Blasius in 1664.[4,5] The discovery of the arachnoid further advanced our concepts of cerebrospinal fluid (CSF) production, circulation, and absorption. Today, the meninges are divided into the pachymeninges, which include the dura mater, and the leptomeninges, which consist of the arachnoid and the pia mater.

Embryology

The development of the meninges starts early in gestation and reaches the basic adult forms by the end of the first trimester. Meningeal precursors are derived from both neural crest and mesodermal cells. As the neural tube fuses at 22 to 24 days of gestation, a single layer of cells, with some attachments to the neural crest, surrounds the developing neural axis. A thicker, looser collection of mesenchymal cells further covers the neural tube starting around day 24 to 28 and completely envelops the developing spinal cord and brain by day 33 to 41. This mesodermal-derived cellular network, along with the neural crest–derived monocellular layer will differentiate into the meninx primitiva (primary meninx).[4,6,7]

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As the pluripotent meninx primitiva develops, it subdivides into two distinct layers between days 34 and 48. The outer portion, the ectomeninx, is dense and compact, whereas the inner layer, the endomeninx, is more loosely arranged. The ectomeninx is the precursor to the dura and the bones of the neurocranium, thus the close apposition of dura and skull stems from their shared embryological ancestry. The inner portion of the endomeninx, containing the neural crest–derived cells covering the neural tube, begins to form the pia during the gestation interval of 45 to 55 days.[4,6,7] Meanwhile, as cerebrospinal fluid invades the endomeninx, cavitations (future cisterns) begin to appear in the outer portion of the endomeninx and become obvious by 55 days of gestation.[8] Although the dura and pia are distinguishably formed structures by this point of development, a distinct arachnoid layer is not obvious and may not appear until much later during fetal development.

The Fine Structures of the Meninges

Dura Mater

The dura mater, from Latin for "hard mother," is the most superficial layer of the meninges. It is also known as the pachymeningeal, or thick layer, with its dense connective tissue. The tenacity of the dura mater stems from its composition of elongated fibroblasts and extensive amounts of extracellular collagen (**Fig. 3.1**). The varying orientations of fibers create a matrix of intertwining collagen that provides significant strength. The outer periosteal layer of dura is generally thicker, with more abundant collagen, than the inner meningeal layer.

Located at the dura–arachnoid junction, a distinct cell population termed the dural border cells has been described. Unlike the collagen-rich superficial dural layers, the dural border cell layer consists of flattened fibroblasts devoid of any extracellular collagen.[6,7] Instead, the extracellular spaces are filled with irregular cellular processes and an amorphous, nonfilamentous granular material. Cellular connections with the superficial dural fibroblasts are absent. Only sparse, but morphologically distinct, cell-to-cell connections exist with the underlying arachnoid layer. The dural border cell layer, therefore, is continuous with the arachnoid layer.[7,9]

Arachnoid

The arachnoid and the pia mater form the leptomeningeal, or thin layer of meninges. Immediately deep to the dural border cell layer sits the arachnoid barrier cell layer. This layer consists of tightly packed large fibroblasts with minimal extracellular space and absence of collagen[9] (**Table 3.1**). There is a unique abundance of cell junctions. Tight junctions among cells strengthen the arachnoid barrier cell layer and render it impermeable to fluids, large molecular weight substance, and even some ions.[9–11] In addition, a continuous basement membrane lines the inner surface of the arachnoid, abutting the subarachnoid CSF space.[7]

Arachnoid trabecular cells are specialized fibroblasts with long processes and attachment to the arachnoid barrier layer. They bridge the subarachnoid space with their long, flattened, irregular processes and may form cellular attachments with pial cells. Collagen may be found within the trabecular matrix created by the processes in the subarachnoid space.[7,9]

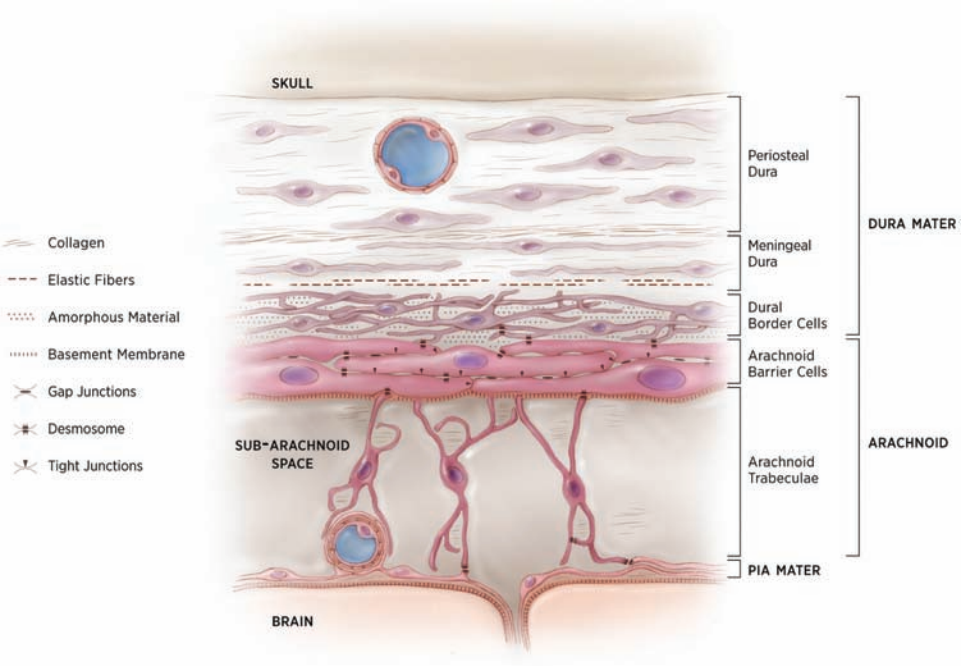


Fig. 3.1 Schematic illustration of the fine structure of the meninges. Used with permission of the Department of Neurosurgery, M.D. Anderson Cancer Center.

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Table 3.1 Summary of the Fine Structural Characteristics of the Meninges

	Layer	Cell Characteristics	Cell Organelles	Cell Junctions
Dura mater	Periosteal dura	Large, elongated, somewhat flattened fibroblasts in varying orientations with extensive amounts of intertwining extracellular collagen	Large amounts of granular endoplasmic reticulum, chromatin-containing nuclei, a Golgi apparatus, ribosomes, mitochondria, fat droplets, filaments	Infrequent
	Meningeal dura	Increased amounts of elongated, flattened fibroblasts with long processes and proportionately less collagen	Condensed cytoplasm with elongated nuclei	Infrequent
	Dural border cells	Modified, elongated, flattened fibroblasts, with amorphous, nonfilamentous material and no collagen in enlarged extracellular compartment	Dense, dark cytoplasm with elongated nuclei	Few cell junctions; occasional desmosomes and gap or intermediate junctions with arachnoid layer
Arachnoid	Arachnoid barrier cells	Closely packed plump cells without any significant amount of extracellular space	Translucent cytoplasm with prominent Golgi apparatus, numerous mitochondria, large oval-shaped nuclei	Numerous cell junctions, especially desmosomes and tight and gap junctions
	Arachnoid trabeculae	Loosely organized fibroblasts with long, flattened, irregular processes	Translucent cytoplasm with prominent Golgi	Cell junctions join trabeculae processes to each other and to overlying arachnoid barrier layer
Pia mater		Flattened fibroblasts	Few organelles with translucent cytoplasm	Few cell junctions, underlying basement membrane between pia and brain

Adapted from Barshes et al[4] and Haines and Frederickson.[7]

Historically, the existence of a subdural space has been advocated as a naturally occurring space, or potential space similar to those found in serous cavities such as pleural, pericardial, or peritoneal cavities. Embryological and histological studies, however, do not support such a concept. The dura and arachnoid share common precursor cells and remain attached throughout development.[6] Instead, the characterization of the dural border cell layer identifies a structurally weak layer at the dura–arachnoid junction. Sandwiched between the dural layer dense in collagen and the arachnoid layer reinforced with cellular junctions, the dural border cell layer represents the plane of least resistance. The creation of a space at the dura–arachnoid junction with hematoma or fluid accumulation secondary to trauma or other pathological processes is the result of tissue damage through the dural border cell layer, and not the opening of a preexisting space.[6,9,12,13]

Pia Mater

The pia mater is a thin sheet of flattened fibroblast cells that separates the subarachnoid space from the subpial and cortical perivascular spaces. The basement membrane of the outer glial layer of the brain and spinal cord, termed glia limitans, separates the pia from the underlying neural tissue to create the subpial space.[4,14] These modified fibroblasts of pia form junctional complexes at their margins, rendering the pia impermeable to particulate matter, such as blood. The pia is reflected from the surface of the brain to surround vessels traveling in the subarachnoid space but does not accompany the vessels into parenchyma. This arrangement seals the subarachnoid space from the subpial and perivascular spaces, such that subarachnoid blood does not enter the subpial space.[4,9,14]

Arachnoid Villi/Granulations

Arachnoid villi are specialized segments of the meninges that project into the sinuses and major venous structures (**Fig. 3.2**). They are essential in the absorption of CSF through both passive and active mechanisms.[15] Whereas arachnoid villi are microscopic, arachnoid granulations are visible to the naked eye, and Pacchionian bodies are especially large, elaborate complexes.[16] A fibrous capsule reflected from the surrounding dura covers arachnoid villi except at the apices, where the underlying arachnoid cell layer and specialized arachnoid cap cells are exposed to the venous blood of the sinus.[7,15] These cells are highly metabolically active and are involved in the resorption

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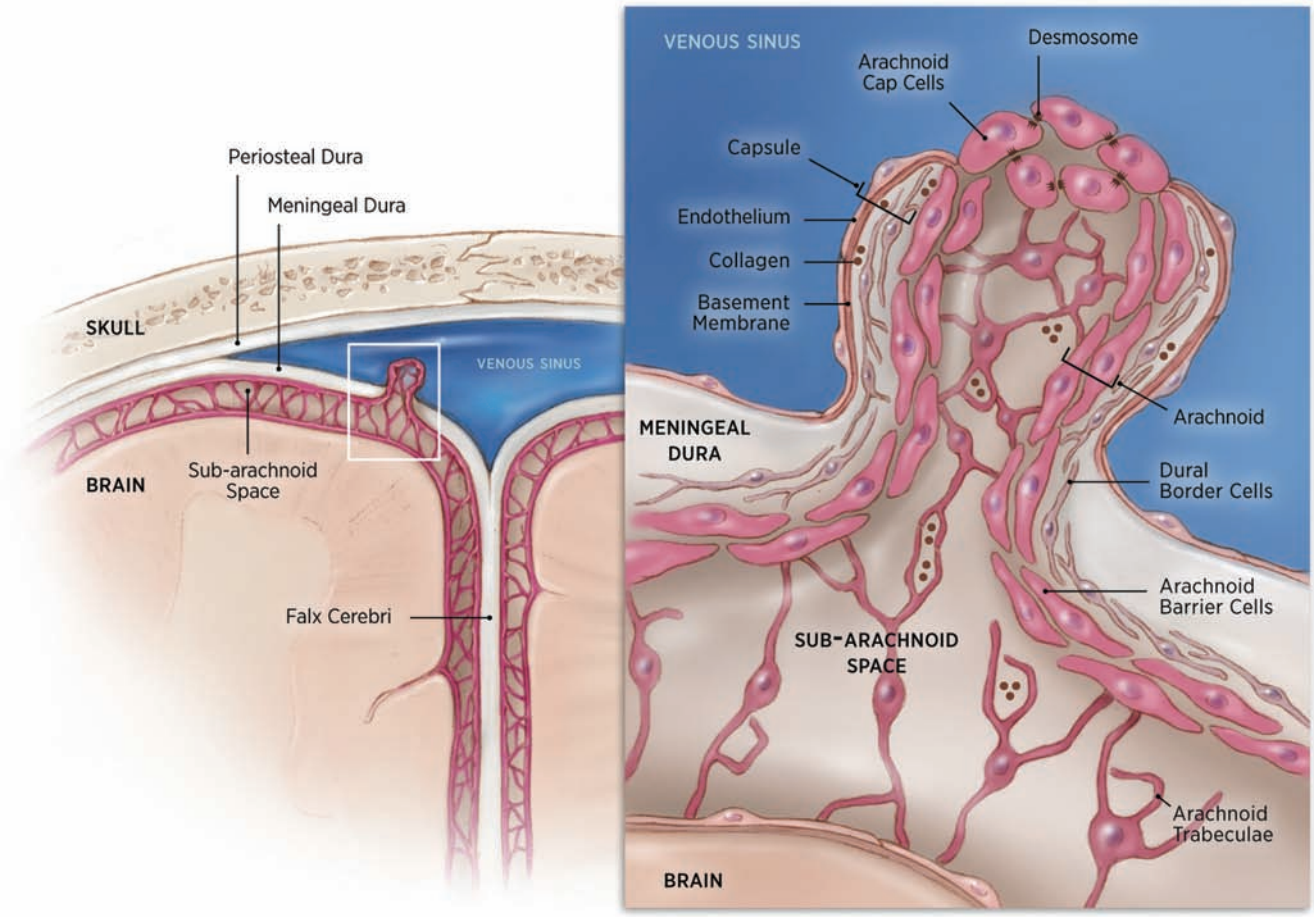


Fig. 3.2 Illustration of the fine structure of an arachnoid granulation and its relation to the superior sagittal sinus. Note the continuity of the layers and spaces of the meninges that surround the brain into the granulation. Used with permission of the Department of Neurosurgery, M.D. Anderson Cancer Center.

of CSF.[1] Arachnoid cap cells are derived from the outer portions of the endomeninx and are considered the cells of origin of meningiomas. Although they can be located throughout the central nervous system, including within the ventricles, the sylvian fissure, and the pineal region, they are found in greatest concentration adjacent to the major sinuses, large cerebral veins, and basilar plexus, and around the crista galli, over the cribriform plate, and at the exit foramina of cranial nerves II through VII and IX through XII.[7]

Dural Anatomy

The dura mater is composed of two distinct layers that remain fused throughout the majority of the cranium.[8,17] The outer periosteal (endosteal) layer is highly vascularized and intimately adherent to the overlying cranium, and it functions as its periosteum.[9,18] The periosteal dura is continuous with the periosteum covering the external cranium at suture lines and neural foramina. It merges with the periorbital membrane through the optic canal and the superior orbital fissure.[17,19] As the cranial nerves exit through their respective cranial foramina, the inner meningeal layer surrounds the nerves as tubular sheaths that will fuse with the epineurium. The meningeal layer reflects away from the periosteal layer at multiple locations to form the venous sinuses. It folds inward to form dural septa that partition and maintain the positions of the intracranial neural structures. These dural folds include the falx cerebri, the tentorium cerebelli, the diaphragma sellae, and the falx cerebelli.

The largest of the dural reflections, the falx cerebri, lies in the deep fissure between the two hemispheres of the cerebrum. It extends posteriorly from its anterior attachment at the crista galli of the cribriform plate to insert onto the superior surface of the tentorium. Superiorly it attaches to the midline of the frontal, parietal, and occipital bones, whereas inferiorly it remains unattached. The sickle-shaped falx is shorter in its anterior portion. Although posteriorly the inferior free edge closely opposes the corpus callosum, anteriorly there is a wide space between the two structures where the cerebral hemisphere can herniate beneath the free edge.[20]

The tentorium cerebelli divides the cranium into the supratentorial and infratentorial compartments. Except for

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its free edge that comprises the incisura, the tentorium is firmly anchored to the inner surface of the temporal, parietal, and occipital bones at its margins. Anteriorly, the free edges of the incisura pass over the trigeminal ganglion to insert onto the petrous apex and the anterior and posterior clinoid processes. These insertions form three dural folds: the anterior and posterior petroclinoid folds and the interclinoid fold. Together, these dural folds form the oculomotor trigone, through which the oculomotor nerve enters the cavernous sinus. The medial extension of the dura covering the oculomotor trigone is the diaphragma sellae. Dura extending anteriorly from the free edge will form the lateral wall of the cavernous sinus and cover the middle cranial fossa.[21]

In addition to the formation of dural septa and sinuses, the layers of the dura also separate around the sella and parasellar regions and the Meckel cave. Situated lateral to the sella, resting on the sphenoid and temporal bones, the cavernous sinus is a paired venous structure enclosed by five dural walls.[22] It faces the temporal lobe laterally and the sphenoid bone, sella turcica, and pituitary gland medially. Anteriorly, the cavernous sinus fills the posterior margin of the superior orbital fissure below the anterior clinoid process, and it extends posteriorly to the petrous apex. Its posterior wall stretches from the lateral edge of the dorsum sellae to the medial margin of the Meckel cave. Finally, the roof of the cavernous sinus is composed of the clinoidal and oculomotor triangles and faces the basal cisterns.[23] These dural folds that envelop the cavernous sinus and its contents serve as consistent landmarks and provide routes for surgical access to the cavernous sinus.

Since Parkinson's initial description of surgical exposure of the cavernous carotid artery through the lateral wall of the cavernous sinus in 1965,[24] there have been numerous investigations of the anatomy and approaches to the cavernous sinus.[22,25–31] The dura of the middle fossa is composed of an outer periosteal layer that is adherent to the inner surface of the cranium, and an inner meningeal layer that faces the brain. At the lateral edge of the cavernous sinus, the middle fossa dural lining separates. The meningeal layer turns upward as the temporal lobe dura and forms the superficial layer of the lateral wall of the cavernous sinus, whereas the periosteal layer continues medially along the skull base to become the medial wall.[22,29] Dissection of the lateral wall of the cavernous sinus reveals a two-layered construct. The superficial layer that is the continuation of the middle fossa meningeal dura can be easily separated from a thinner deep layer. The dural sheaths that accompany cranial nerves III, IV, V1, and V2 as they penetrate into the sinus form this semitransparent deep layer.[22,30]

The lateral and medial walls join anteriorly at the superior orbital fissure (SOF), and the cranial nerves exiting the apex of the cavernous sinus are wrapped in a common meningeal sheath.[29] The middle fossa periosteal dura continues through the SOF as the periosteal layer of the periorbita, creating the orbitotemporal periosteal fold.[32] Sectioning of this periosteal bridge is the initial step in the extradural elevation of the superficial layer of the lateral wall and a useful step to enhance extradural exposure of the anterior clinoid process for anterior clinoidectomy (**Fig. 3.3**).[29,32] Posterolateral to the SOF at the site of middle cerebral vein drainage, venous channels are covered by meningeal dura only. This absence of a deep inner membrane creates a weak point in the cavernous sinus wall and a potential route of invasion into the cavernous sinus apex from medial sphenoid wing meningiomas.[29]

Unlike the dual-layered lateral wall, the medial wall of the cavernous sinus is composed of a single thin layer of dura that cannot be separated. This single-layered construction of the medial wall may explain the tendency for pituitary adenomas to invade the cavernous sinus. Although a single layer of dura constitutes the medial wall, it can be divided into two parts, each with a different dural origin. It has a sellar portion that faces the sella

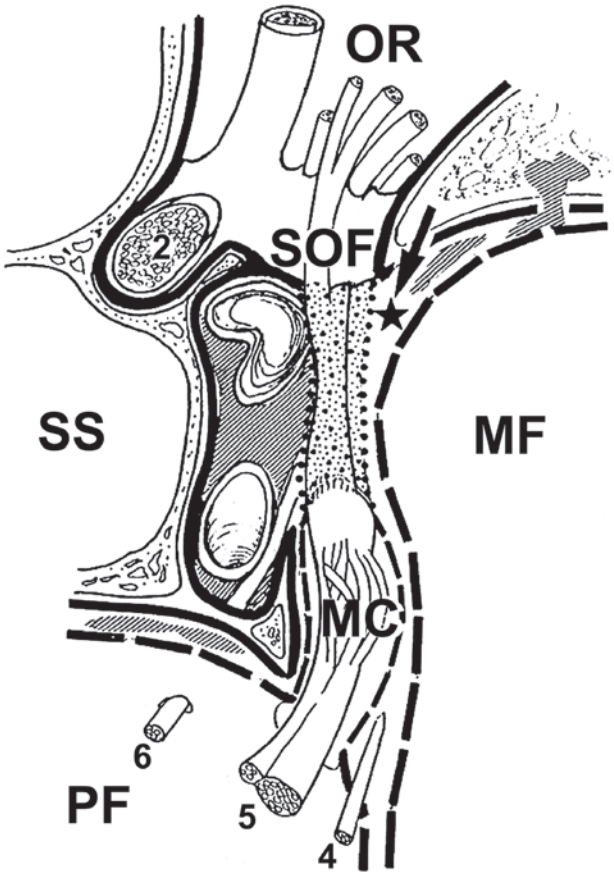


Fig. 3.3 Schematic illustration of the meningeal architecture of the cavernous sinus. Broad line, periosteal dura lining the sphenoid bone; broken line, meningeal dura; dotted line, deep layer; shaded areas, venous channels. The star indicates a cleaving plane that is accessible between the superficial and deep layers by incision of the orbitotemporal periosteal bridge (*arrow*). OR, orbit; MC, Meckel cave; SS, sphenoid sinus; MF, middle cranial fossa; SOF, superior orbital fissure; PF, posterior fossa. Reprinted with permission from Kawase T, van Loveren HR, Keller JT, et al. Meningeal architecture of the cavernous sinus: clinical and surgical implications. *Neurosurgery* 1996;39:527–536.

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turcica and the pituitary gland, and a sphenoidal portion that sits upon the carotid sulcus on the lateral aspect of the sphenoid bone. The sellar portion of the medial wall is a continuation of the meningeal layer of dura that extends downward from the free edge of the diaphragma sellae. It separates the pituitary gland from the venous space of the cavernous sinus and can be easily dissected away from the pituitary capsule. The sphenoidal portion, on the other hand, is the medial extension of the periosteal dura lining the middle fossa floor. At the level of the sellar floor, the two dural components of the medial cavernous sinus wall join together and continue together medially to line the inferior aspect of the pituitary gland and the sellar floor.[33]

The roof of the cavernous sinus extends posteriorly from the superior junction of the optic strut with the body of sphenoid bone to the posterior clinoid process. The optic strut is the posterior root of the lesser wing and is the inferiomedial anchor of the anterior clinoid process to the lesser wing. Several dural attachments, including the anteromedial part of the tentorium, the anterior petroclinoid and interclinoid dural folds, insert on the anterior clinoid process. Together with the posterior petroclinoid dural fold, they form the oculomotor triangle that marks the roof over the posterior cavernous sinus.[22]

Two layers of dura envelop the anterior clinoid process and form the dural roof of the anterior cavernous sinus. The superficial meningeal layer covers the superior surface of the clinoid process and extends medially to fuse with the adventitia of the internal carotid artery (ICA) to form the distal dural ring (**Fig. 3.4**). This dural ring encircles the carotid completely and tightly, marking its entrance into the intradural compartment. Further medial, it forms the falciform ligament, the dural sheath of the optic nerve, and continues as a dural covering of the tuberculum sellae and planum sphenoidale. Laterally, the distal dural ring merges with the anterior petroclinoid dural fold, which stretches from the petrous apex to the anterior clinoid process.

Below the distal dural ring, an inner dural layer termed the caroticooculomotor membrane lines the inferior surface of the anterior clinoid process. It separates the clinoid process from the oculomotor nerve and surrounds the carotid artery medially as the proximal dural ring. As it sweeps upward along the ICA, it forms a carotid collar between the proximal and distal rings and joins the distal ring at the posterior tip of the anterior clinoid process. Posterolaterally, this thin inner membrane continues as the dural sheath of the oculomotor nerve and contributes to the inner layer of the lateral wall of the cavernous sinus. The proximal and distal dural rings serve as the boundaries for the clinoidal segment of the ICA, which can be surgically exposed with the removal of the anterior clinoid process.[22,34]

The posterior wall of the cavernous sinus is delineated by an area between the posterior clinoid process, the dural entrance of the abducens nerve, and the medial border of the porus trigeminus. The posterior petroclinoid dural fold marks the superior margin while the petrosphenoid ligament (Gruber ligament) defines the inferior limit of the posterior wall. A large venous confluence located lateral to the dorsum sellae opens into the basilar, superior, and inferior sinuses in the posterior portion of the cavernous sinus. The basilar sinus is the largest connection between the two sinuses and sits on the posterior surface of the dorsum sellae.[22]

At its posterolateral border, the lateral wall of the cavernous sinus extends downward and fuses with the dura of the middle fossa and Meckel cave.[23] Situated in the trigeminal depression at the petrous apex and sandwiched between the two layers of the middle fossa dura, the Meckel cave is a cleftlike dural pouch containing the

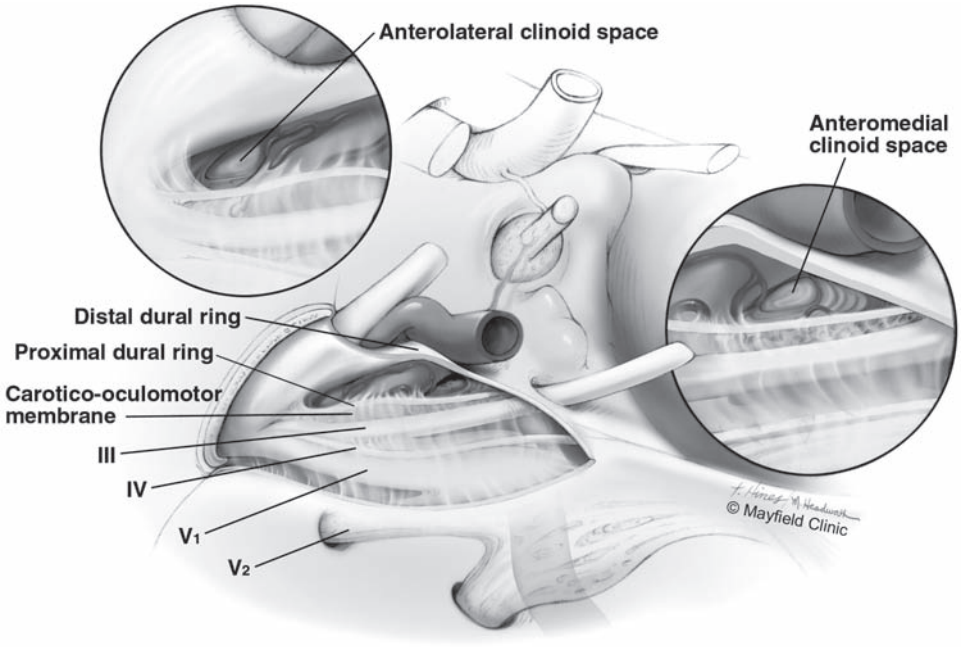


Fig. 3.4 Illustration of the microanatomy of the left cavernous sinus after an anterior clinoidectomy. The inner dural layer of the roof of the cavernous sinus reflects from the oculomotor nerve to form the proximal dural ring. This caroticooculomotor membrane travels with the clinoidal segment of the internal carotid artery (ICA) to fuse with the distal dural ring. Printed with permission from the Mayfield Clinic.

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sensory and motor roots of the trigeminal nerve, and the gasserian ganglion. The entrance to the Meckel cave, the porus trigeminus, sits midway between the dorsum sellae and the internal auditory canal just below the tentorium and the superior petrosal sinus.[25,35]

As the trigeminal nerve enters the porus trigeminus from the posterior fossa, it carries with it posterior fossa meningeal dura and underlying arachnoid, which continues anterolaterally to envelop the gasserian ganglion and to become the Meckel cave (**Fig. 3.5**). The dual layers of the temporal dura split around this pocket of the posterior fossa dura, with the meningeal layer blanketing its lateral surface and the periosteal layer underlying its medial side. Therefore, two layers of meningeal dura cover the trigeminal roots and gasserian ganglion on their dorsolateral aspects. Inside the Meckel cave, CSF sits in the subarachnoid space of the trigeminal cistern. Beyond the gasserian ganglion, the inner components of the posterior fossa dura and arachnoid continue as the epineurium and the perineurium of the divisions of the trigeminal nerve, respectively.[8,19] A cleavage plane exists between the two layers of the meningeal dura that can be exploited surgically to expose the Meckel cave in an extradural fashion.[25]

The sella turcica sits in the middle of the cranium flanked by the cavernous sinus on either side. With the exception of its lateral walls, which are the single-layered medial walls of the cavernous sinus, two layers of dura engulf the sella.[33] Meningeal and periosteal layers of the lateral wall

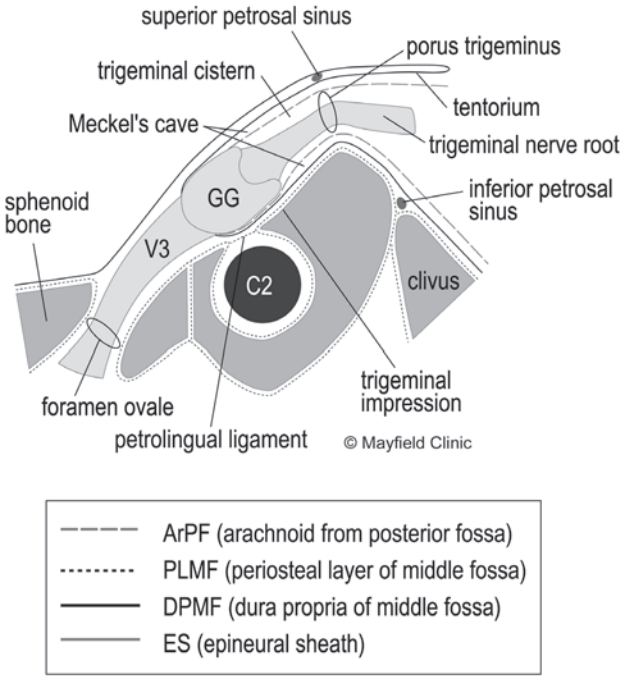


Fig. 3.5 Schematic illustration of coronal view of the petrous apex. The Meckel cave is a dural recess formed by the meningeal dura of the posterior fossa traveling with the trigeminal nerve. It is situated on the trigeminal impression of the petrous ridge, sandwiched between the two layers of the middle fossa dura. Printed with permission from the Mayfield Clinic.

and roof of the cavernous sinus converge medially to form the diaphragma sellae and extend anteriorly to line the anterior skull base and posteriorly to cover the dorsum sellae and clivus. The diaphragma sellae forms the roof of the sella with a conduit for the passage of the infundibulum, connecting the pituitary gland with the hypothalamus.[36] The diaphragm opening demonstrates significant variability among individuals. The size of the diaphragm opening and the degree of its incompetence may affect the direction of tumor growth either into or out of the sella.[37]

Subarachnoid Cisterns

CSF flows throughout the central nervous system between the arachnoid and the pia mater in the subarachnoid space and drains into the venous system through the arachnoid villi and granulations. In locations where the natural contours of the brain pull away from the underlying cranium and in spaces between neural structures the arachnoid and pia can separate widely to form cisterns. These enlargements of subarachnoid spaces are partitioned by fenestrated condensation of leptomeningeal tissue, and they take their shapes and names according to their relation to neighboring neural elements. Arteries, veins, nerves, and other brain structures traverse or protrude into subarachnoid cisterns (**Table 3.2**). Strategic navigation through the cisterns and judicious release of CSF from the cisterns allow surgeons to minimize retraction and trauma to neural structures.[7,38]

Blood Supply to the Dura

Meningeal arteries originate from the internal and external carotid and vertebral arteries to supply the dura and the adjacent cranium (**Table 3.3**). These branches provide overlapping patterns of vascularization that are more complex at the skull base than over the cerebral convexity. In general, the internal carotid system supplies the midline dura of the anterior and middle fossae via the anterior and posterior ethmoidal arteries, the recurrent ophthalmic arteries, and the lacrimal artery. The external carotid system supplies the lateral segment of the three cranial fossae through the middle meningeal artery, the accessory meningeal artery, and the ascending pharyngeal artery. The vertebrobasilar system provides blood to the midline structures of the posterior fossa and the foramen magnum through the anterior and posterior meningeal arteries and the subarcuate artery. Finally, the convexity dura receives the majority of its blood supply from the middle meningeal artery.[7,17]

The dural reflections of falx cerebri, falx cerebelli, and tentorium represent anastomotic sites between dural and parenchymal arteries because they receive supply from meningeal arteries and cerebral arteries. The falx cerebri receives the majority of its blood supply from its insertion along the cranial vault. It receives augmented blood flow from tentorial branches of the cavernous carotid and posterior cerebral artery. The tentorium receives blood

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Table 3.2 Summary of Major Cisterns and Their Contents

Cistern	Artery	Vein	Cranial Nerve	Structure
Carotid	ICA, origins of AChorA, PComm			
Chiasmatic	ACA	Anterior communicating vein	II and chiasm	Hypophyseal stalk
Lamina terminalis	ACA, AComm, Heubner, hypothalamic and frontoorbital arteries	Lamina terminalis venous system		
Sylvian	MCA	Sylvian, frontoorbital veins		Insular gyri
Ambient	P2 of PCA, SCA	Basal vein (of Rosenthal)	IV	Lateral crus cerebri
Crural	AChorA, MedPostChorA	Basal vein (of Rosenthal)		
Quadrigeminal	Posterior pericallosal, P3 of PCA, SCA	Vein of Galen	IV	Pineal, superior and inferior colliculi
Interpeduncular	BA bifurcation, P1 of PCA, proximal SCA, PComm	Basal vein (of Rosenthal)	III	Mammillary bodies, medial crus cerebri
Prepontine	BA, origins of AICA and SCA	Anterior and anteromedial pontine veins	VI	
Superior cerebellopontine	AICA, labyrinthine	Petrosal and lateral pontine veins	V, VII, VIII	
Inferior cerebellopontine	VA, origin of PICA	Lateral medullary and postolivary veins	IX, X, spinal XI, XII	Pyramid, choroid plexus, inferior olivary eminence
Cisterna magna	Distal PICA	Tonsillar vein		C1, C2 root

Abbreviations: ACA, anterior cerebral artery; AChorA, anterior choroidal artery, AComm, anterior communicating artery; AICA, anterior inferior cerebellar artery; BA, basilar artery; ICA, internal carotid artery; MCA, middle cerebral artery; MedPostChorA, medial posterior choroidal artery; PCA, posterior cerebral artery; PComm, posterior communicating artery; PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery; VA, vertebral artery.

Adapted from[38] with modifications based upon.[7]

Table 3.3 Summary of Meningeal Branches and Area of Supply

Source	Parent Vessel	Branch	Area of Supply
ECA	Ascending pharyngeal artery	Carotid branch	Dura of foramen lacerum and carotid canal
		Jugular branch	Dura of jugular foramen, walls of inferior petrosal sinus, jugular bulb and inferior sigmoid sinus, dura of posterior petrosal surface
		Hypoglossal branch	Dura of foramen magnum, inferolateral cerebellar fossa
	Occipital artery	Jugular branch	Dura of jugular foramen, walls of inferior petrosal sinus, jugular bulb and inferior sigmoid sinus, dura of posterior petrosal surface
		Hypoglossal branch	Dura of foramen magnum, inferolateral cerebellar fossa
		Mastoid branch	Dura over posterior petrosal surface, lateral and paramedial cerebellar fossa
		Parietal emissary branch	Dura over posterior parietal convexity
	Middle meningeal artery	Petrosal branch	Lateral wall of cavernous sinus, dura over posteromedial floor of middle fossa, medial tentorium insertion along petrous ridge
		Anterior division	Dura over frontal and anterior parietal convexity, walls of superior sagittal sinus and falx, dura over lateral portion of anterior and middle fossa
		Posterior division	Dura over posterolateral floor of middle fossa, lateral tentorium insertion along petrous ridge; dura around confluence between superior petrosal, transverse, and sigmoid sinus and dura of lateral cerebellar fossa; dura of temporosquamous and parietooccipital convexity
	Accessory meningeal artery		Middle fossa dura, lateral wall of cavernous sinus

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Table 3.3 *(Continued)*

ICA	Cavernous carotid	Recurrent artery of foramen lacerum	Dura of foramen lacerum and carotid canal
		Medial tentorial artery (artery of Bernasconi and Cassinari)	Roof of cavernous sinus, medial third of tentorium, and posterior attachment of falx cerebri
		Lateral tentorial artery	Lateral third of tentorium
		Dorsal meningeal artery	Dura over dorsum sellae and clivus
		Inferior hypophyseal artery	Dura over posterior sellar floor, posterior clinoid, and medial wall of cavernous sinus
		Medial clival artery	Dura over posterior clinoid, dorsum sellae, and medial wall of cavernous sinus
		Inferolateral trunk	Inferolateral wall of cavernous sinus and adjacent middle fossa
		Capsular arteries	Dura of floor and anterior margin of the roof of sella
	Ophthalmic artery	Anterior ethmoidal artery	Dura over anterior convexity, medial third of floor of the anterior fossa
		Posterior ethmoidal artery	Dura over medial third of floor of anterior fossa
		Deep recurrent ophthalmic artery	Dura of the walls of cavernous sinus around SOF
		Superficial recurrent ophthalmic artery	Dura over the anterior clinoid process and lesser sphenoid wings
		Lacrimal artery	Dura over the lateral part of SOF, sphenoid wings, and ridge
	Anterior cerebral artery	Olfactory branches	Dura over the medial third of the floor of the anterior fossa
		Pericallosal branches	Free edge of falx cerebri
VBA	Vertebral artery	Anterior meningeal artery	Dura over odontoid process, atlantooccipital space, and anterolateral border of foramen magnum, including dura over occipital condyles
		Posterior meningeal artery	Dura over posterior atlantooccipital space, falx cerebelli, medial and paramedial cerebellar fossa, and occipital convexity; walls of transverse sinus and torcula
	Anteroinferior cerebellar artery	Subarcuate artery	Dura of the posterior surface of petrous bone
	Posterior cerebral artery	Tentorial branch	Posterior third of the falx cerebri and adjacent tentorium

Abbreviations: ECA, external carotid artery; ICA, internal carotid artery; SOF, superior orbital fissure; VBA, vertebrobasilar arteries.
Adapted from Martins et al.[17]

Table 3.4 Summary of Dural Innervation

Trigeminal nerve	Ophthalmic nerve	Innervation of dura over cribriform plate, medial orbital roof, crista galli, diaphragma sellae, tentorium, falx cerebri, inferior and superior sagittal, transverse and straight sinuses
	Maxillary nerve	Innervation of dura of the anterior floor of the middle fossa
	Mandibular nerve	Innervation of dura over the lateral floor of the middle cranial fossa and most of the convexity of the cranium
Upper three cervical roots	Ascending meningeal rami	Innervation of dura lining anterior floor of posterior fossa, clivus, and ventral craniospinal junction
	Recurrent meningeal branch of vagus	Innervation of walls of sigmoid sinus, occipital sinus, falx cerebelli, dura over the petrous surface of temporal bone, and suboccipital cerebellar surface
	Hypoglossal nerve	Innervation of dura of posterior fossa up to inferior petrosal sinus

Adapted from Larrier and Lee.[39]

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flow from both supratentorial and infratentorial sources. The supratentorial components include branches from the cavernous carotid and middle meningeal arteries, whereas branches from the ascending pharyngeal artery, occipital artery, and posterior meningeal artery constitute the infratentorial contribution.[17]

Dural Innervation

The cranial dura receives dense innervation from multiple ganglia, including trigeminal, glossopharyngeal, vagal, and the first through third cervical spinal ganglia (**Table 3.4**).[7,39,40] The trigeminal nerve, in particular the ophthalmic division, provides the only source of sensory innervation to the supratentorial dura, venous sinuses, and meningeal arteries. Somatic afferent fibers from the superior vagal ganglion and the upper cervical ganglia travel via the recurrent meningeal branch of the vagus through the jugular foramen to innervate posterior fossa dura and vasculature. The upper two cervical ganglia also send nerve fibers with the hypoglossal nerve through the hypoglossal canal to reach the posterior fossa. These fibers are predominantly unmyelinated and terminate as free nerve terminals along meningeal vessels and sinuses and in the connective tissue.[39,40] They are responsible for the pain response in the numerous pathological conditions affecting the meninges and manifesting with headaches.

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Chapter 4

Epidemiology of Meningiomas

Elizabeth B. Claus and Alan L. Morrison

Introduction

Meningiomas accounted for 33.8% of all primary brain and central nervous system (CNS) tumors reported in the United States between 2002 and 2006 and thus represent the most frequently diagnosed primary brain tumor.[1] With this in mind, researchers are examining the role of both genetic and environmental risk factors for this tumor. Family history studies suggest a role for inherited genes for meningioma in addition to the neurofibromatosis type 2 (NF2) gene, and genetic variants in genes involved in the DNA repair pathway, some of which appear common to several tumor types, have been implicated.[2] Of note, the environmental risk factor most strongly associated with a diagnosis of meningioma remains exposure to ionizing radiation.[3] A role for hormones (both endogenous and exogenous) and meningioma risk is hypothesized but less clearly defined.[4] The increased emphasis on research dedicated to the study of brain tumors coupled with the advent of new tools in genetic and molecular epidemiology make the current era an ideal time to advance knowledge for intracranial meningioma. This review highlights current knowledge of meningioma epidemiology and new directions for research efforts in this field.

Population Statistics

Meningiomas account for ~20% of all intracranial tumors in males and 38% in females, yet little is known regarding the risk factors associated with these lesions.[3] The prevalence of meningioma is estimated to be ~97.5/100,000 in the United States, with more than 160,000 individuals currently diagnosed with this tumor. Data from the Central Brain Tumor Registry of the United States (CBTRUS)[1] reveal an age-adjusted incidence rate (per 100,000 person-years) of 8.36 and 3.61 for females and males, respectively. Reported rates for black non-Hispanics are higher (6.67 per 100,000 person years) than for white non-Hispanic and Hispanics (5.90 and 5.94 per 100,000 person-years, respectively).[1] Age-specific incidence rates (**Table 4.1**) reveal an increasing risk with age.[1] Data from CBTRUS indicate that incidence rates for meningioma have increased over time,[1] likely in part due to increased imaging and passage of legislation requiring the registration of benign tumors into state cancer registries. In 2002 legislation was passed in the United States [the Benign Brain Tumor Cancer Registries Amendment Act (H.R. 5204)] mandating registration of benign brain tumors such as meningiomas. This legislation went into effect January 1, 2004, and will allow for improved reporting of both incidence rates and survival times for patients with meningiomas. To date, meningioma mortality rate estimates have been hampered by incomplete reporting, potential selection biases with respect to the individuals who are included in the databases as well as limited follow-up information. Analyses based on information from the National Cancer Database (NCDB), which includes data from more than 1000 hospitals that participate in the American College of Surgeons tumor registry program, report unadjusted 2- and 5-year survival rates for patients with meningioma of 81% and 69%.[5]

Table 4.1 Age-Specific Incidence Rates for Meningioma in the United States (2002–2006)[1]

Age	0–19	20–34	35–44	45–54	55–64	65–74	75–84	85+
Rate	0.11	1.05	3.72	7.62	12.39	20.58	29.40	34.94

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Risk Factors

Ionizing Radiation

At present, the primary environmental risk factor identified for meningioma is exposure to ionizing radiation (IR), with risks from 6- to 10-fold reported.[6–10] At high dose levels, data exist for atomic bomb survivors[9] and show a greatly increased risk for meningioma, with increasing risk correlating with the proximity to the epicenter of the explosion.[29,30] Evidence also exists for lower dose levels. In one of the most well-known studies to date of IR and meningioma risk, children who were given radiation therapy for scalp ringworm in Israel between 1948 and 1960 (the Tinea Capitis Cohort) were observed to have a relative risk of almost 10 for meningioma.[7] Several studies have linked the number of full-mouth dental radiographs to risk of meningioma, although the sample sizes are limited, and most later studies (also small in size) did not confirm these findings. The most recent case-control study of 200 meningioma patients reported that the full-mouth series was associated with a significantly increased risk of meningioma [odds ratio (OR): 2.06, 95% confidence interval (CI): 1.03 to 4.17], although evidence for a dose response relation was lacking (p for trend = 0.33).[10] Radiation therapy for intracranial tumors has also been linked to meningioma risk,[7] and animal studies support the contention that IR can induce intracranial tumors, including meningiomas, by damaging DNA, with resultant single-strand or double-strand breaks. No recent large-scale studies of meningioma risk relative to IR exist, when x-ray doses for dental and other procedures have decreased but during which time new radiographic procedures have been introduced, including computed tomography (CT).

Hormones

An association between hormones and meningioma risk has been suggested by several findings, including the increased incidence of the disease in women versus men (2:1); the presence of estrogen, progesterone, and androgen receptors on some meningiomas; an association between breast cancer and meningiomas; indications that meningiomas change in size during the luteal phase of the menstrual cycle and pregnancy; and in vitro proliferation of meningioma-cell lines in culture after exposure to estrogens has been observed.[3,4] A pilot study of 31 meningioma samples reported that gene expression appeared more strongly associated with progesterone receptor (PR) status than with estrogen receptor (ER) status.[11] Genes on the long arm of chromosome 22 and near the *NF2* gene (22q12) were most frequently noted to have expression variation, with significant upregulation in PR+ versus PR– lesions suggesting a higher rate of 22q loss in PR– lesions.

Researchers have only begun to address the question of whether the use of exogenous hormones such as oral contraceptives and/or hormone replacement therapy is associated with an increased risk of meningioma.[4,12–16] Data from two cohort studies and several case-control studies exist. In a case-control study nested within the Nurse's Health Study (including 125 cases of meningioma), the relative risk of meningioma associated with hormone use for premenopausal women was 2.48 (95% CI: 1.29 to 4.77) when compared with postmenopausal women who had never used hormones.[14] For postmenopausal women who were hormone users the relative risk was 1.86 (95% CI: 1.07 to 3.24). No excess risk was associated with past hormone use. No association was found for past or current use of oral contraceptives. Recently published data from a cohort study of 1.3 million women with a mean age of 55.9 and recruited from 1996 to 2001 (The Million Women Study) did not find an association between oral contraceptive use within the past 5 years and meningioma risk (OR:1.06, 95% CI:0.8 to 1.38), but did not report results for hormone replacement therapy use.[12] In the largest and most recent case-control study to date, the Interphone Group reported an increased relative risk of meningioma among postmenopausal women for every use of hormone replacement therapy (OR:1.7, 95% CI: 1.0 to 2.8).[16] Women who had used long-acting hormonal contraceptives also had an increased risk of meningioma; the odds ratio for at least 10 years of use was 2.7 (95% CI: 0.9 to 7.5). A retrospective cohort study using the Mayo Clinic Jacksonville patient database between 1993 and 2003 confirms the positive Nurse's Health Study findings (OR: 2.2, 95% CI: 1.9 to 2.6) of an association between hormone replacement therapy use and meningioma risk,[13] whereas a case-control study including 219 meningioma cases identified from three Chicago area hospitals between 1987 and 1992 reports a protective effect for oral contraceptive use (OR: 0.2, 95% CI: 0.0 to 0.8) and a nonstatistically significant protective effect associated with hormone replacement therapy use.[15] Hence, at present, there is limited statistical evidence of an increased risk of meningioma among users of oral contraceptives. Although not definitive, available data suggest an association between the use of hormone replacement therapy and increased meningioma risk. Further evaluation of exogenous hormone use in women with meningioma in a large sample is needed, with particular attention to stratification by hormone (i.e., estrogen and/or progesterone) composition, duration of and age at use, and tumor receptor subtype.

Researchers have also reported conflicting results when examining meningioma risk across categories of pregnancy, menstrual, and anthropometric variables.[4,12–17] In the Nurse's Health Study,[14] when examining age at first menstrual period, investigators observed a relative risk of 1.29 (95% CI: 0.86 to 1.92) for meningioma in women with age at menarche between 12 to 14 years, and 1.97 (95% CI: 1.06 to 3.66) in women with age at menarche after 14 years when compared with those women with menarche before the age of 12 years. The group also observed a tendency for increased risk of meningioma for parous compared with nonparous women [relative risk (RR): 2.39, 95% CI: 0.76 to 7.53), although this value is not statistically significant. In a second nested case-control study, Lambe et al[17] examined 1088 patients with meningioma

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within the Swedish Cancer Registry and matched to data from the Swedish Fertility Registry. This group found no association between either parity or age at first birth and meningioma risk; however, they were not able to adjust their analyses for other possible meningioma risk factors such as use of exogenous hormones or radiation history. Data from the Interphone Study suggest that meningioma risk among women aged < 50 years is increased with increasing number of live births (OR: 1.8; 95% CI: 1.1 to 2.8 for three vs no live births) but found no association with menopausal status.[16] The Million Women Cohort reported an increasing risk of meningioma with increasing body mass index (OR: 1.46, 95% CI: 1.11 to 1.91) but no association with number of pregnancies or age at first birth.[12] An additional case-control study,[15] which included 219 women, found a protective effect for pregnancy, which increased with the number of pregnancies and age at first pregnancy. Neither age at menarche or menopause was reported to show any effect in unadjusted analyses, although menopause showed an increased risk (OR: 2.0, 95% CI: 1.0 to 4.0) in adjusted analyses. As this review makes evident, the associations between pregnancy and menstrual risk factors and meningioma risk are not consistent and deserve a more formal examination.

Head Trauma

Since the time of Harvey Cushing, head trauma has been suggested as a risk factor for meningioma, although the results across studies are not consistent. Although several small case-control studies from the early 1980s report an increased risk of meningioma associated with head trauma for both males and females, other studies report no such association. A cohort study of 228,055 Danish residents hospitalized for concussion, skull fracture or other head injury between 1977 and 1992 did not find any significant increase in the incidence of meningioma, although the mean follow-up was only 8 years. The standardized incidence ratio (SIR) for meningioma after the first year was 1.2 (95% CI: 0.8 to 1.7).[18]

Cell Phone Use

The question of whether cell phone use is related to meningioma risk remains of great interest to the general public. At least 10 studies have examined the association between cell phone use and tumors of the brain. At present, little evidence exists for an association between the two, although sample sizes specific to meningiomas are relatively small, the follow-up time since commencement of cell phone use relatively short, and, in some instances, the measurement of cell phone use somewhat crude.[19–21]

Association with Breast Cancer

An association between breast cancer and meningioma has been examined in several studies.[2,4,22] Several explanations have been proposed for this association, including the presence of common risk factors, such as endogenous and exogenous hormones as well as shared genetic predisposition, including variants in DNA repair polymorphisms.[2] Custer et al provide a review of the literature and an analysis of the association between breast cancer and meningioma using the western Washington State cancer registry data.[23] The relative risks observed across existing studies range between 1.5 and 2.0, with the majority statistically significant. Most of these studies have been conducted with tumor registry data and have relatively small sample sizes, and none has been able to examine the association while controlling for risk factors that are likely to be shared by the two tumors, such as pregnancy and menstrual variables and exogenous hormone use. Intriguing new data gathered from an analysis of five of the case-control series from the Interphone Study report suggest that variation in the breast cancer susceptibility gene 1 (*BRCA1*)-interacting protein 1 (*BRIP1*) is associated with meningioma risk.[2]

Industry/Occupation/Diet/Allergy

Attempts to link specific chemicals with meningiomas in occupationally or industrially exposed groups have proved inconclusive.[3] A recent international case-control study found no association between diet and meningioma (n = 332).[23] Although several studies that examine the relationship between glial brain tumors and allergic disease such as asthma and eczema have found evidence for an association, little evidence has been found for such an association for meningioma.[24]

Family History of Meningioma

Few studies have examined the relationship between meningioma risk and a family history of meningioma. Malmer et al[25] examined cancer risk in spouses and first-degree relatives of brain tumor patients in Sweden and reported that a meningioma diagnosis conferred a twofold increase in meningioma risk to first-degree relatives (SIR: 2.2, 95% CI: 1.4 to 3.1) but not to spouses of affected individuals. An inverse association between risk and age at onset was appreciated, with an SIR of 2.5 (95% CI: 1.5 to 4.0) for probands under 50 years of age versus 1.3, (95% CI: 0.6 to 2.6) for probands older than 50 years of age. Similar analyses by Hemminki et al[26] using data from the Swedish and Norwegian Registry Databases reveal an increased risk with increasing numbers of affected first-degree relatives with persons having one or two first-degree family members with meningioma (SIR: 1.6, 95% CI: 1.3 to 42.0 and SIR: 5.0, 95% CI: 0.9 to 14.8, respectively). Despite the fact that up to 1% of the adult population may harbor a meningioma,[27] the total number of families with multiple members diagnosed with meningioma is relatively small (indicating, in part, a wide spectrum of phenotypic expression with respect to clinical import and hence screening undertaken), and most such families are currently attributed to inherited *NF2* mutations. At present no family-based linkage or segregation analyses studies of meningioma have been reported.

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Genetic Polymorphisms

In the most recent and largest study[2] to date of genetic polymorphisms and meningioma risk, Interphone Study investigators reported a statistically significant association with meningioma for 12 single-nucleotide polymorphisms (SNPs) drawn from DNA repair genes. These investigators examined 1127 tagged SNPs selected to capture most of the common variation in 136 DNA repair genes as well as an additional 388 putative functional SNPs (including 69 nonsynonymous coding SNPs that may identify the function of expressed proteins) in 631 cases and 637 controls drawn from five case-control series from the Interphone Study. The Interphone Study is a case-control project initially designed to examine the relationship between cell phone use and the risk of brain tumors, including meningioma. Study subjects are primarily Caucasian of Western European background. The group reported a novel and biologically intriguing association between meningioma risk and three variants in the gene that encodes breast cancer susceptibility gene 1-interacting protein 1 (*BRIP1*) (17q22). The most significant was SNP rs4968451, which maps to intron 4 of the gene. An odds ratio of 1.61 (95% CI: 1.26 to 2.06) was found for heterozygotes, and 2.33 (95% CI: 1.25 to 4.34) for homozygotes. The *BRIP1* gene is involved in the repair of DNA double-strand breaks by homologous recombination in a manner that depends on its association with *BRCA1*. Defects in *BRIP1* are linked to breast cancer susceptibility (as well as Fanconi anemia), leading researchers to speculate that the reported association between breast cancer and meningioma risk may be due to similar defects in DNA repair genes rather than, or in addition to, the previously assumed shared hormonal risk factors (such as hormone replacement therapy). This group also reported a statistically significant association between four variants in the *ATM* gene, a member of the phosphatidylinositol 3 kinase family known to be involved in homologous and nonhomologous DNA break repair and meningioma risk. Previous groups have also noted significant associations between *ATM* variants for meningioma as well as breast cancer.[8] These findings are again of interest in light of the associations between ionizing radiation and meningioma risk as well as between breast cancer and meningioma risk.

Directions for Future Studies

The study of risk factors for intracranial meningioma remains a challenging and largely unexplored field. The known risk factors of genetic predisposition and high-dose radiation exposures account for a small proportion of cases. Although a role for hormones is likely given the gender distribution of meningiomas, few specific or consistent data exist on hormonal risk factors. Tools from epidemiology may be used to collect and define appropriate subject data from well-defined source populations to define risk factors for the overall group of meningioma patients as well as for specific subgroups. High-quality follow-up data of sufficient time period must be collected on meningioma patients to obtain representative estimates of sex- and age-specific rates for recurrence, quality of life, and overall survival. In addition to the collection of data on environmental risk factors such as hormone use, new projects will need to consider the inclusion of information on genetic variants. In addition to exploring environmental and genetic factors for meningioma risk separately, the interaction between the two must be examined. For example, the integration of environmental risk factors such as oral contraceptive use or radiation exposure with information on genetic polymorphisms in steroid hormone or DNA repair genes may help researchers understand the complex relationship between genetic susceptibility and environmental exposures in the development of meningioma. Given the large numbers of subjects needed to study such gene–environment interactions, collaborative, multicenter efforts between a variety of researchers will be needed, including experts from such fields as neurosurgery, epidemiology, genetics, statistics, and neuropathology.

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Chapter 5

Pathology of Meningiomas

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Introduction

Meningiomas are slowly growing neoplasms thought to arise from meningothelial cells found within arachnoid granulations. Concentrated in the walls of the major venous sinuses, these structures, which contain "arachnoid cap cells," account for the dural localization of most meningiomas within the cranium and spinal cord.[1] Since the original classification of meningiomas in 1931,[2,3] there has been a gradual yet significant change in our views regarding the pathology of these tumors. The collective experience from the literature has shown that the vast majority of meningiomas do not behave in a biologically aggressive fashion, with tumors that exhibit aggressive behavior representing only ~10% of cases. Intriguingly, not all studies have demonstrated a correlation of histology with survival. Although metastases are uncommon and usually restricted to anaplastic examples, even tumors judged benign are capable of metastasis. Detailed correlative clinicopathological studies by Perry and others have greatly improved consistency in predicting clinical outcome and contributed to our understanding of the biology of meningiomas.[2–7] The recently revised World Health Organization (WHO) classification has incorporated these advances into a more precise and objective classification system.[8] Nevertheless, challenges remain in identifying reliable biomarkers that will lead to the refinement of prognosis and define treatment responsiveness.

Localization

The majority of meningiomas are supratentorial, with a large number located along the convexities. Approximately 17 to 25% occur in a frontobasal location; however, only about 10% occur in the posterior fossa. Within the frontobasal region, the olfactory grooves, tuberculum sellae and parasellar region, and the petrous bone are preferred sites. Approximately 5% occur along the cerebellar convexity, 2 to 4% at the tentorium cerebelli, and 2 to 4% within the cerebellopontine angle.[9]

Uncommonly, meningiomas are found within the ventricular system or arise within the optic nerve sheath, where they produce diffuse circumferential thickening of the nerve sheath rather than a focal mass (**Fig. 5.1**). Notably, meningiomas are the most common trigonal intraventricular mass in adults.[10] Atypical locations, including those within the posterior fossa, brain parenchyma, and ventricles, are more frequent in the pediatric population.[11]

Second only to nerve sheath tumors in this location, spinal meningiomas account for ~10% of meningiomas. They are most frequently found anterolaterally or dorsolaterally in the thoracic spine and show greater proclivity for psammomatous change. Extracranial meningiomas are very rare tumors and have been described in ectopic locations such as intraosseous, scalp, paranasal sinuses, parotid, parapharyngeal space, mediastinum, lung, or ad-

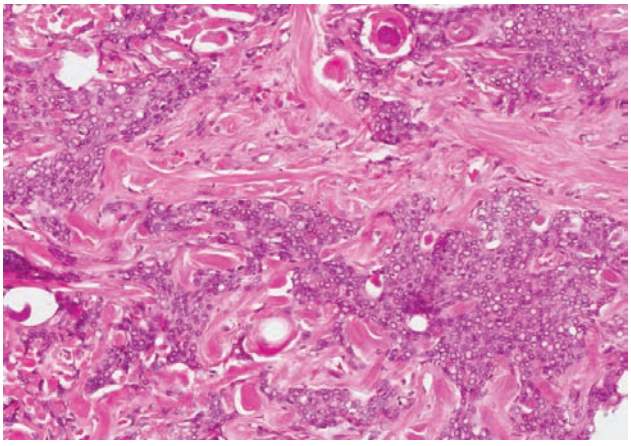


Fig. 5.1 Meningioma infiltrating the dura mater of an optic nerve.

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renal gland. Histologically, these lesions are indistinguishable from ordinary intracranial meningiomas, with most cases diagnosed as benign variants. Multicentric meningiomas account for 1 to 10% of meningiomas and tend to present at a younger age, with most examples confined to a single hemicranium. Metastases are uncommon and may be seen with benign or malignant meningiomas.

Gross Features

Grossly, meningiomas manifest as tan, well-demarcated solitary masses with a firm, broad-based dural attachment and smooth or bosselated surfaces. Falcine or tentorial meningiomas may have a bilobed, dumbbell shape. Except for aggressive examples, meningiomas are readily separated from underlying brain or spinal cord. Similarly, hemorrhage and necrosis are usually restricted to malignant tumors. Rare meningiomas cover the dura in a flat, carpetlike growth and are referred to as meningioma en plaque. This variant often elicits hyperostosis and is more common along the sphenoid ridge or within the cavernous sinus, where they become enmeshed with sinus contents. Most meningiomas compress and protrude into the underlying brain without true invasion. True invasion implies penetration of the pial membrane.

Specimens vary from soft and gelatinous to firm and calcified. On sectioning, mineralization due to the development of psammoma bodies imparts a gritty consistency. The cut surface is translucent and pale gray or homogeneously reddish brown, particularly in tumors with increased vascularity or hemorrhage. Tumors with a high lipid content (xanthomatous change) show yellow streaks, and, exceptionally, bone or cartilage may be detected. Cysts, either within or surrounding the tumor, are reported in only 1.6 to 10% of meningiomas and may be visible grossly.

Histopathology

As a group, meningiomas probably display the greatest variety of patterns of any primary brain tumor (**Table 5.1**), which sometimes poses diagnostic challenges. The three most common histologic subtypes are the meningothelial (syncytial), transitional, and fibroblastic meningiomas; however, meningiomas may display more than one histologic pattern in a tumor. According to WHO criteria, meningiomas are designated as benign (grade I), atypical (grade II), or anaplastic/malignant (grade III). The following sections briefly describe the morphological subtypes associated with each grade.

WHO Grade I

Meningothelial

This variant of meningioma exhibits a syncytial growth pattern and is composed of polygonal cells with abundant eosinophilic cytoplasm and indistinct cell borders

Table 5.1 2007 World Health Organization Meningioma Classification

Meningothelial meningioma	WHO grade I
Fibrous (fibroblastic) meningioma	WHO grade I
Transitional (mixed) meningioma	WHO grade I
Psammomatous meningioma	WHO grade I
Angiomatous meningioma	WHO grade I
Microcystic meningioma	WHO grade I
Secretory meningioma	WHO grade I
Lymphoplasmacyte-rich meningioma	WHO grade I
Metaplastic meningioma	WHO grade I
Chordoid meningioma	WHO grade II
Clear cell meningioma	WHO grade II
Atypical meningioma	WHO grade II
Papillary meningioma	WHO grade III
Rhabdoid meningioma	WHO grade III
Anaplastic (malignant) meningioma	WHO grade III

(**Fig. 5.2**). The sheetlike growth of the cells is interrupted by vascularized connective tissue trabeculae that divide the tumor into lobules. The nuclei are round to oval with pale chromatin. A diagnostically useful cytologic feature is the presence of nuclear pseudoinclusions or clear nuclear vacuoles (**Fig. 5.3**). These nuclear features are more commonly seen in this subtype and arise from invaginations of cytoplasm into the nucleus.

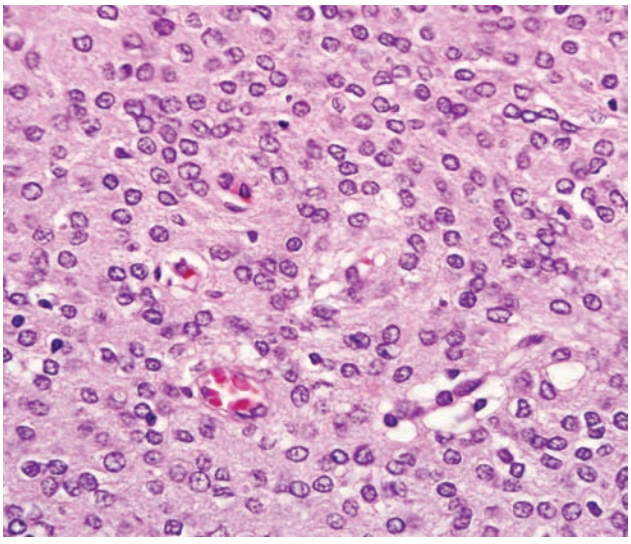


Fig. 5.2 Syncytial growth pattern in meningothelial meningioma.

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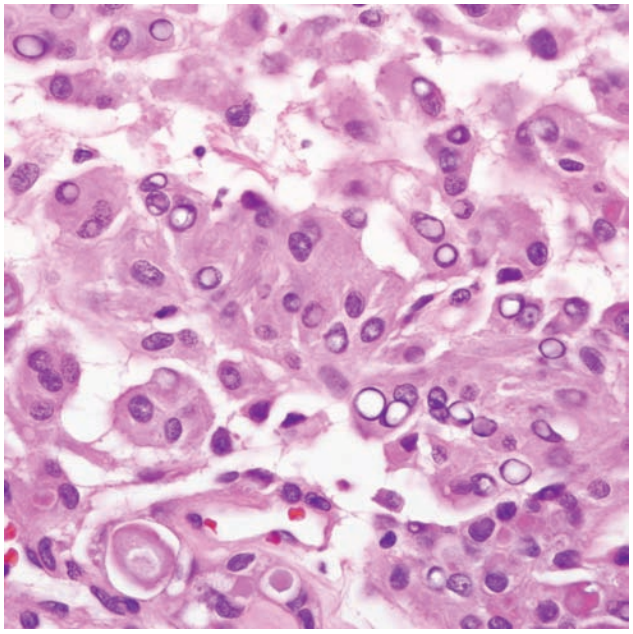


Fig. 5.3 Meningothelial meningioma with intranuclear inclusions.

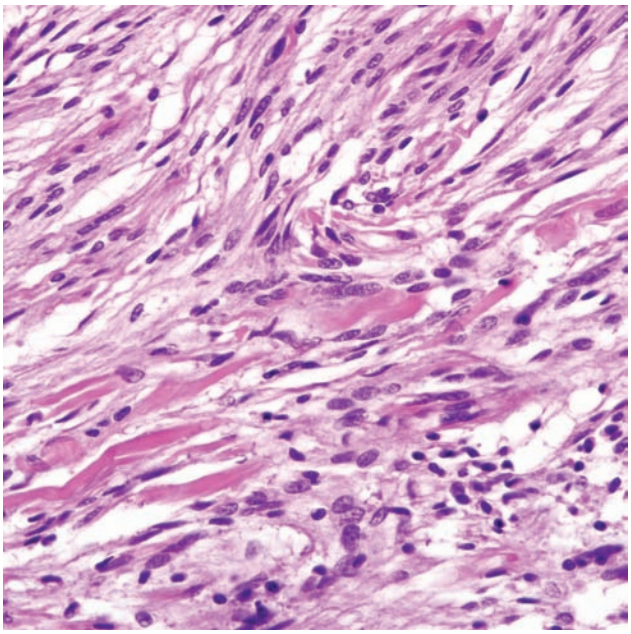


Fig. 5.4 Fibrous meningioma with fascicles of spindled cells.

Fibrous

The predominant cell shape is spindled, with the tumor composed of fascicles of interlacing bundles of slender elongated cellular elements (**Fig. 5.4**). Although most meningiomas are reticulin-poor, a rich reticulin and collagen network is found between cells in this variant. The spindle cell appearance of fibrous meningioma may simulate schwannoma, which should prompt a cautious approach to definitive intraoperative diagnosis.

Transitional

Transitional meningiomas are a mixture of the meningothelial and fibrous types. Of note, this variant contains meningothelial cells arranged in whorls, sometimes around a central blood vessel, and is occasionally punctuated by concentrically laminated mineralized bodies known as psammoma bodies.

Microcystic

Microcystic meningiomas are composed of loosely arranged clusters of cells with long, thin processes containing variably sized intercellular vacuoles that either appear empty or contain pale mucinous material, xanthomatous cells, and hyalinized blood vessels (**Fig. 5.5**). Pleomorphic-appearing cells may be noted in this variant but are not of prognostic significance. Whorls, lobules, and psammoma bodies are not common in this variant.[12–14]

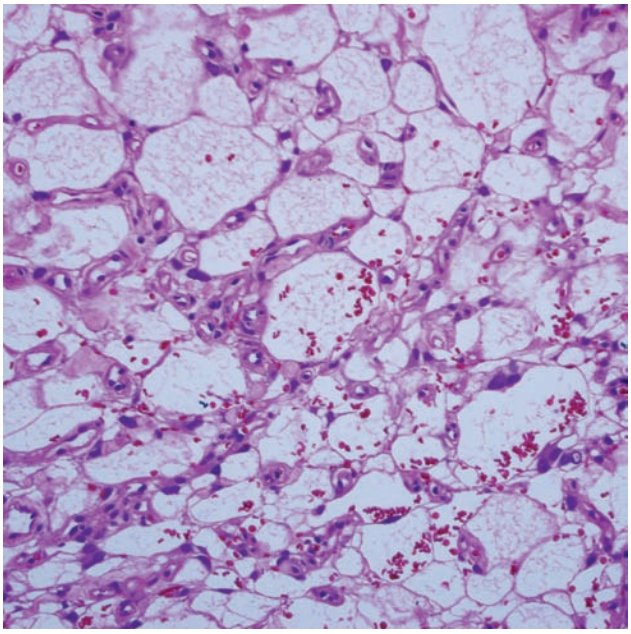


Fig. 5.5 Microcystic meningioma with intercellular spaces and scattered pleomorphic nuclei.

Psammomatous

Psammomatous meningiomas contain many psammoma bodies to the extent that in extreme examples only sparse remnants of tumor cells are present in highly calcified examples (**Fig. 5.6**). Although psammoma bodies are found in a variety of meningiomas, most densely calcified tumors are transitional. Bone-related proteins, including osteopontin produced by CD68-positive macrophages, are thought to play a role in psammoma body formation.[15]

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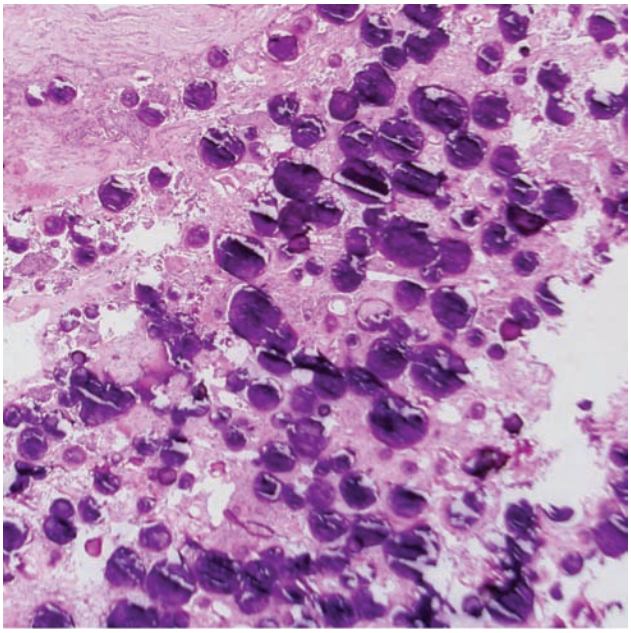


Fig. 5.6 Numerous psammoma bodies in a psammomatous meningioma.

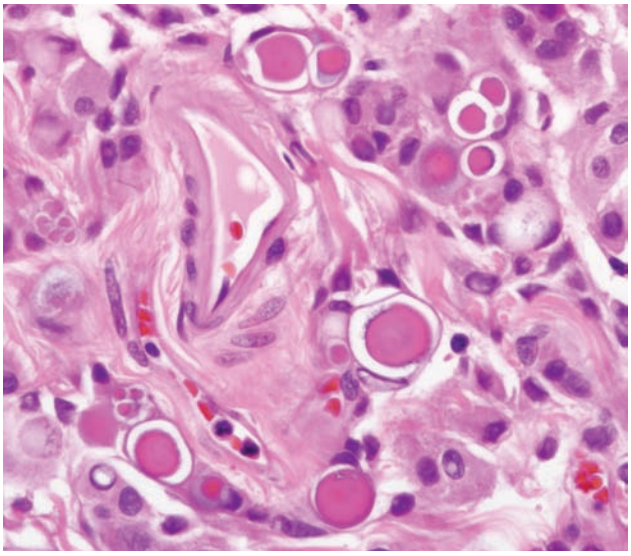


Fig. 5.7 Secretory meningioma with eosinophilic pseudopsammoma bodies.

Secretory

Secretory meningiomas are typically meningothelial or transitional lesions in which epithelial differentiation has produced intracellular lumina. This variant is distinguished by eosinophilic globular hyaline inclusions that are intensely periodic acid–Schiff (PAS) positive and diastase resistant, which have been termed pseudopsammoma bodies[16,17] (**Fig. 5.7**). The tumor cells are carcinoembryonic antigen (CEA) and cytokeratin positive while the luminal secretions are CEA positive (**Fig. 5.8**). Blood CEA levels can be monitored to follow tumor recurrence.

Angiomatous

Angiomatous meningiomas feature a predominance of blood vessels in comparison with meningothelial cells. The abundant blood vessels are small to medium in size with hyalinized vascular walls (**Fig. 5.9**), sometimes mimicking the appearance of a vascular malformation. Degenerative nuclear atypia can be noted in this variant.

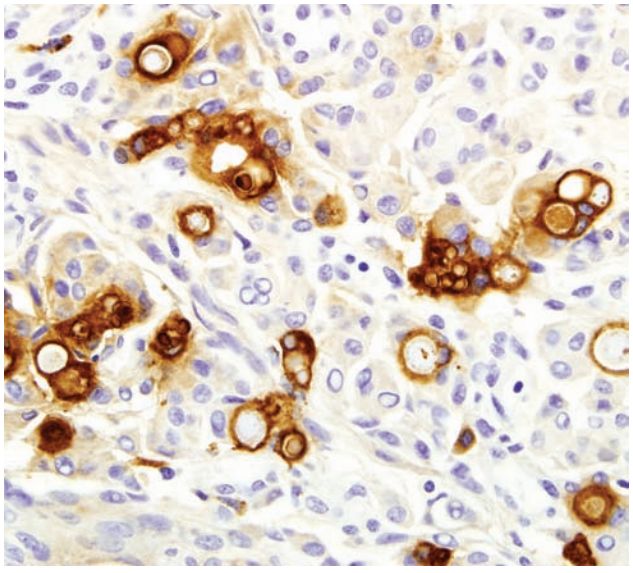


Fig. 5.8 Secretory meningioma with carcinoembryonic antigen–positive pseudopsammoma bodies and surrounding meningothelial cells.

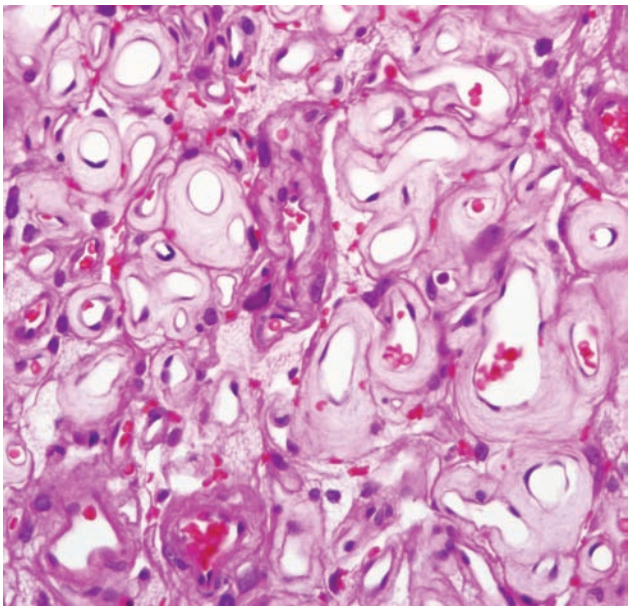


Fig. 5.9 Angiomatous meningioma with hyalinized blood vessels.

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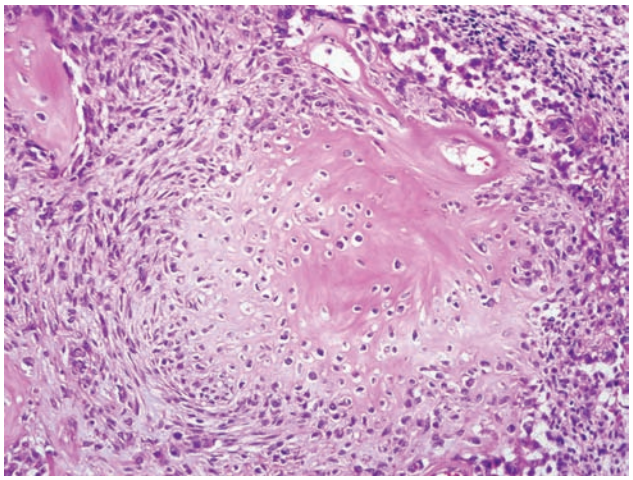


Fig. 5.10 Metaplastic meningioma with osseous metaplasia.

Metaplastic

Metaplastic meningiomas are meningothelial, fibrous, or transitional with metaplastic changes that may be cartilaginous, osseous, xanthomatous, myxoid, or lipomatous (**Fig. 5.10**).

Lymphoplasmacyte-Rich

The rare lymphoplasmacyte-rich variant is typically a meningothelial, fibrous, or transitional meningioma accompanied by a variable chronic inflammatory infiltrate. Extensive lymphoplasmacytic infiltration, which sometimes includes lymphoid follicle formation, may obscure the meningothelial components of the tumor, requiring immunohistochemistry to rule out a lymphoproliferative process.[18] This tumor may be associated with hyperglobulinemia and iron-refractory anemia.

◆ WHO Grade II

Atypical Meningioma

On microscopic examination, atypical meningiomas deviate from their benign counterparts by the presence of increased mitotic activity [four or more mitoses per 10 high power fields (HPFs)], or three or more of the following changes: increased cellularity, small cell formation, prominent nucleoli, sheetlike growth, and areas of spontaneous necrosis (**Figs. 5.11** and **5.12**). It should be noted that preoperative embolization can cause necrosis in benign meningiomas and may lead to the mistaken diagnosis of higher grade features.

Brain invasion is generally noted as irregular groups of tumor cells infiltrating the brain without an intervening pial layer (**Fig. 5.13**). Meningioma invasion may elicit a gliotic reaction. Immunohistochemistry with glial fibrillary acidic protein (GFAP) can be used to help highlight the intervening brain parenchyma between the invading tongues of tumor cells. Extension of tumor into the Virchow-Robin space is not brain invasion. The presence of brain invasion increases the risk of recurrence and is considered another feature for grade II (atypical meningioma).

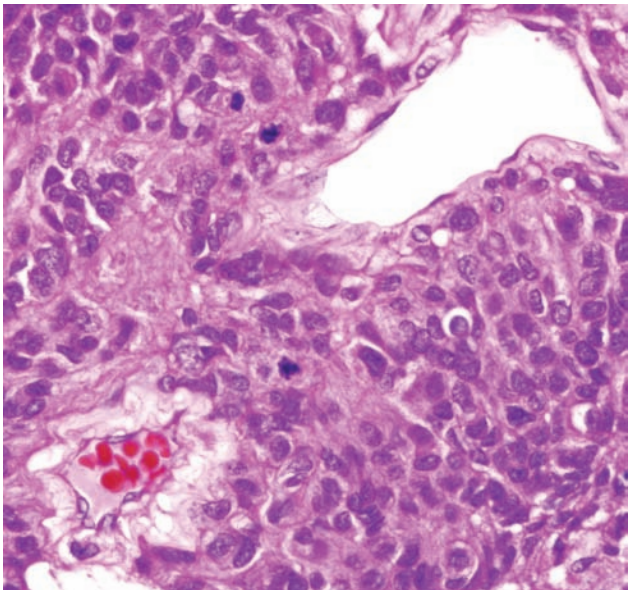


Fig. 5.11 Atypical meningioma with increased mitotic activity.

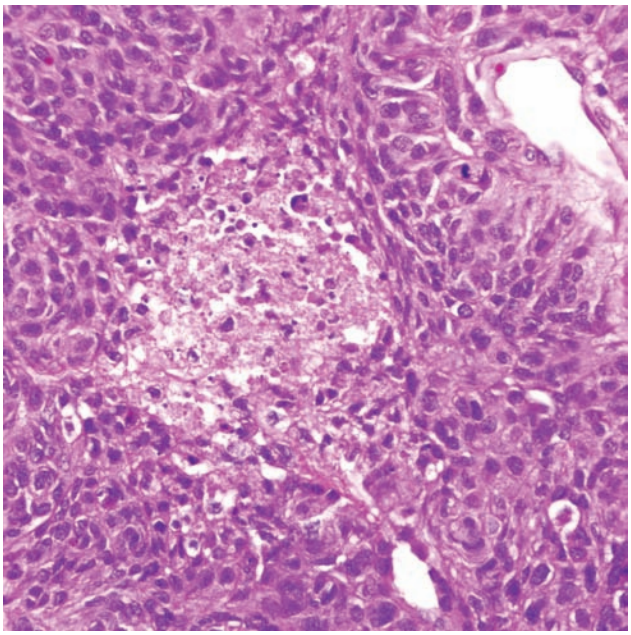


Fig. 5.12 Atypical meningioma with focal necrosis.

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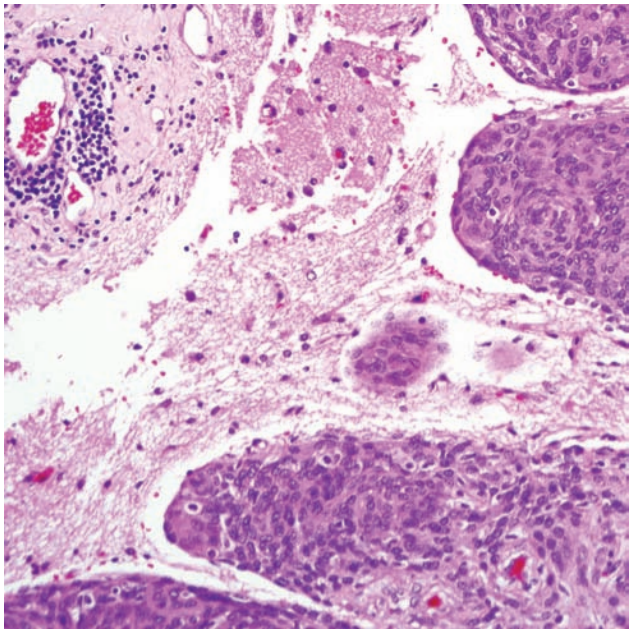


Fig. 5.13 Tongues of meningioma invading brain parenchyma.

Clear Cell

Distinction from other subtypes is warranted because of the less favorable clinical course of clear cell meningioma.[19] This tumor simulates other clear cell tumors and is more commonly found in the cerebellopontine angle or cauda equina. Immunostaining is similar to that of other meningiomas. Clear cell meningiomas contain sheets of polygonal cells with clear cytoplasm and round nuclei (**Fig. 5.14**). Characteristically and diagnostically helpful, the cytoplasm stains positive for periodic acid-Schiff because of the presence of glycogen. Whorl formation is not well defined in this variant and psammoma bodies are not seen. Hyalinized blood vessels may be prominent in clear cell meningiomas. This tumor usually affects younger patients more often than other meningiomas.

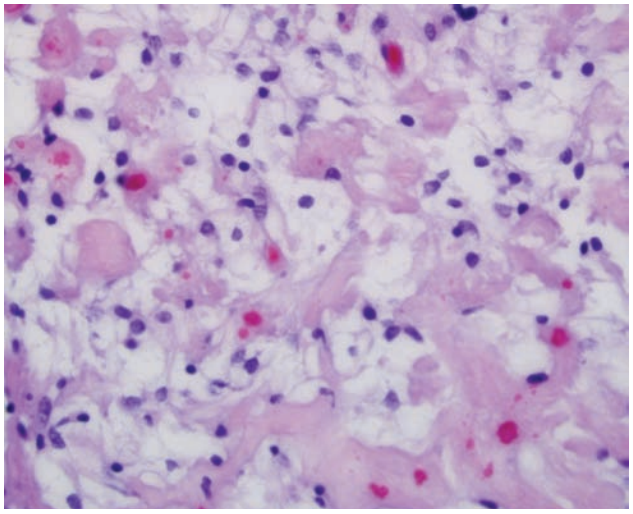


Fig. 5.14 Clear cell meningioma.

Chordoid

First described by Kepes in 1988, chordoid meningiomas are characterized by chords of eosinophilic, epithelial, or spindle-shaped cells within a myxoid stroma, resembling chordoma[20,21] (**Fig. 5.15**). Variable numbers of lymphocytes and plasma cells characterize these tumors.[22,23] Sporadic reports of chordoid meningiomas associated with Castleman disease have been cited in the literature.[20] Similar to rhabdoid meningiomas, chordoid features may become more prominent as a meningioma becomes more aggressive or recurs.

◆ WHO Grade III

Anaplastic

This malignant variant can be recognized by its greater cellularity, malignant cytology, and increased mitotic activity, usually more than 20 mitotic figures per 10 HPF. Necrosis is common in atypical and malignant forms of meningioma. Fortunately, their incidence is relatively low, ranging from 0.9 to 10.6% in different series, with an overall mean representation of 2.8% of meningiomas.

Rhabdoid

Rhabdoid meningiomas are uncommon. Recurrent variants of other meningioma types can acquire rhabdoid features.[24,25] Rhabdoid cells are recognized by their round eccentric nuclei, vesicular chromatin, prominent nucleoli, and abundant eosinophilic cytoplasm containing whorls of intermediate filaments (**Fig. 5.16**).

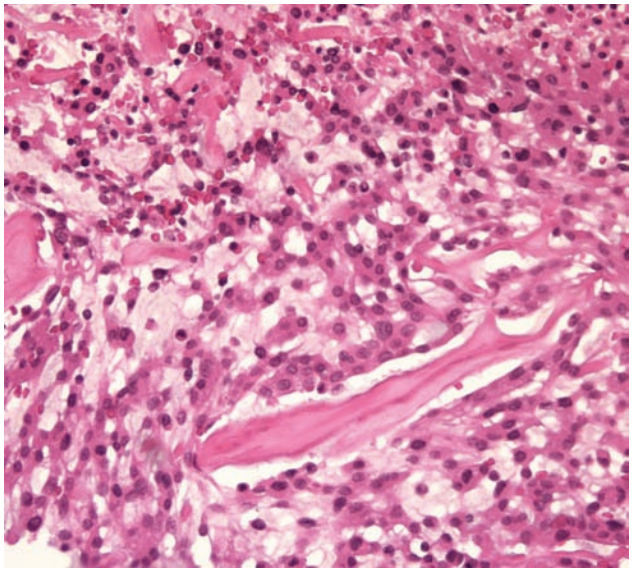


Fig. 5.15 Chordoid meningioma with eosinophilic cells in a mucinous background.

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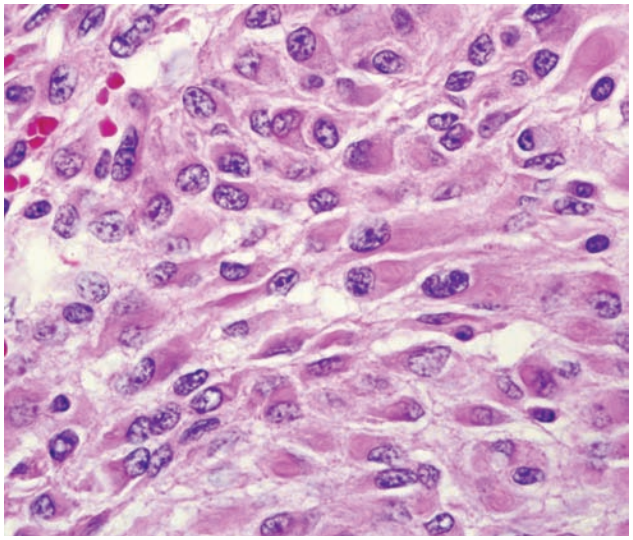


Fig. 5.16 Rhabdoid meningioma with eccentric nuclei, prominent nucleoli, and eosinophilic cytoplasmic inclusions.

Papillary

This aggressive variant of meningioma is defined by the presence of a perivascular or pseudopapillary pattern of tumor cell growth, either entirely or more commonly in combination with other common histological components of meningiomas (**Figs. 5.17** and **5.18**). These tumors frequently invade brain and bone and may exhibit extracranial metastases.[26] Like clear cell meningiomas, these tumors tend to occur in young patients.

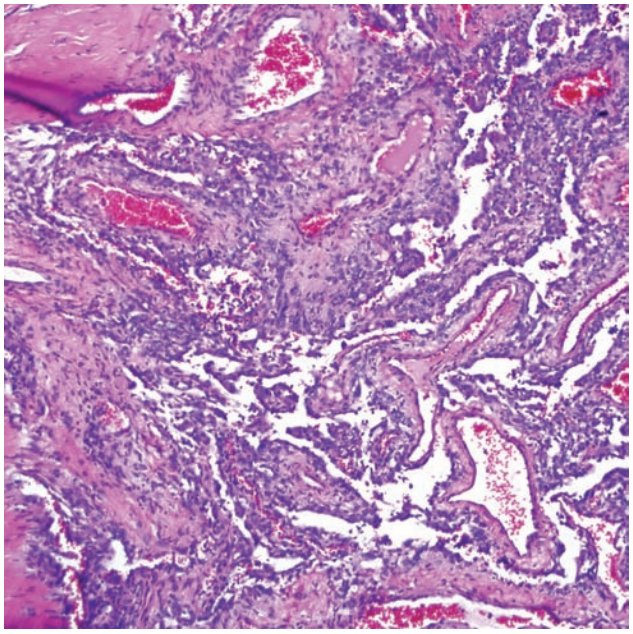


Fig. 5.17 Papillary meningioma with perivascular rosettes and papillary formation.

Immunohistochemistry

The role of immunohistochemistry in the diagnosis of the majority of meningiomas has traditionally been an ancillary one, primarily serving as a means of excluding other tumors that may bear a resemblance. There is no single immunohistochemical marker that is infallible in confirming the diagnosis. Most meningiomas have immunoreactivity for epithelial membrane antigen (EMA), making this a useful marker in separating meningiomas from other tumors (**Fig. 5.19**).[27] With the exception of fibrous meningiomas, only weak and focal immunoreactivity for S-100 protein is seen. Slightly more than half of meningiomas express progesterone receptors, whereas the estrogen receptor status is usually negative. Keratins are known to be expressed in meningiomas, but unlike in carcinoma, staining is focal or patchy in meningiomas.[28] More recently, claudin-1, a novel immunostain, has been shown to be useful in distinguishing meningiomas from histologic mimics. Although the specificity of this marker appears to be high, the sensitivity is low, which suggests that its utility is greater when used in a panel of immunostains.[29] It has been argued that the presence of hormone receptors, especially progesterone, may partially explain the greater incidence of meningiomas in females.

Cytology

Although meningiomas can have a diverse histologic presentation, many meningiomas have similar cytologic findings. Intracellular adhesion makes it difficult to smear the cells on a slide and is demonstrated by broad

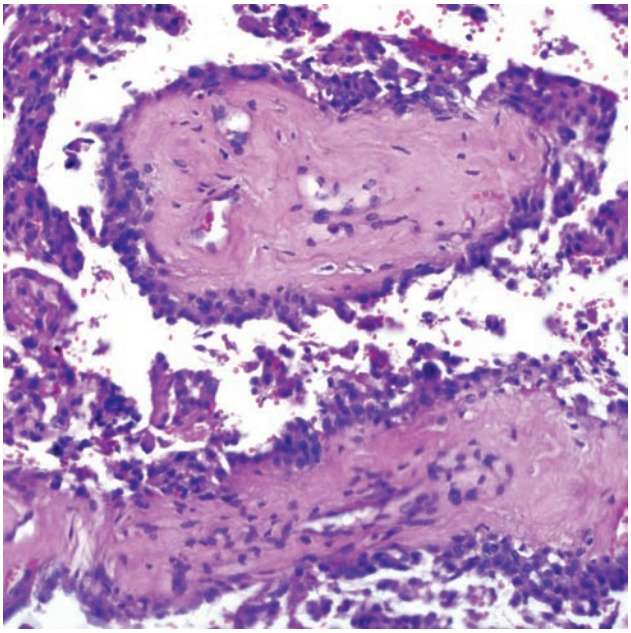


Fig. 5.18 Papillary meningioma with collagenous cores.

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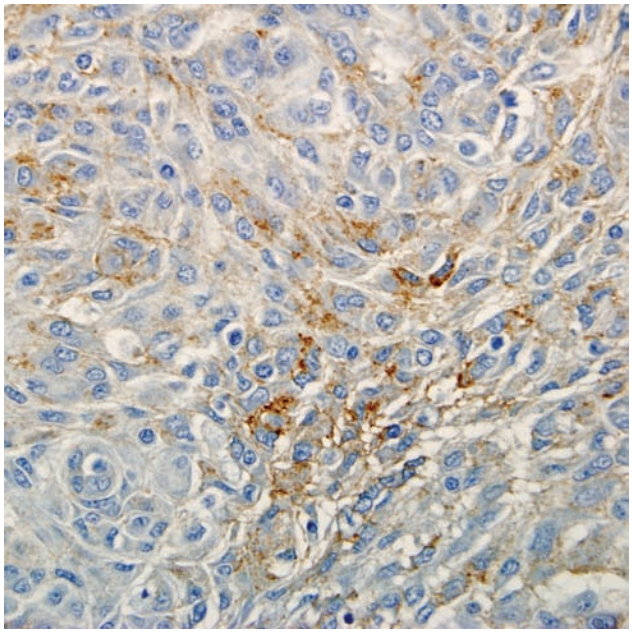


Fig. 5.19 Epithelial membrane antigen–positive cells in rhabdoid meningioma.

processes between the cell bodies. Low-grade meningiomas have round to oval nuclei with delicate pale chromatin and small nucleoli. Nuclear pseudoinclusions, whorls, and psammoma bodies may be present. With increased atypia, nucleolar prominence and hyperchromatism can be noted.

Frozen Section

History, radiological localization, and cytologic features assist in the frozen section interpretation of meningiomas. Whorls, intranuclear inclusions, and psammoma bodies help with the diagnosis, especially when the pathologist is told that the lesion is dural based. These features may not always be prominent, which can lead to other considerations in the differential diagnosis. Gliomas usually have fine cellular processes and are not dural based. Typically, carcinomas exhibit more cytologic atypia, including nuclear pleomorphism and hyperchromatism. Schwannomas in general have a biphasic appearance, some nuclear pleomorphism, and hyalinized blood vessels. Another consideration, hemangiopericytoma, is more cellular, has disordered cells, exhibits a branching (staghorn) vascular pattern, and lacks the nuclear features of meningioma.

Differential Diagnosis

Problems in diagnosis are unlikely for anyone familiar with the characteristic picture of the more common variants of this tumor. Yet problems may arise when the pathologist is confronted with small specimens that may not be representative of the overall appearance of the tumor or with rare morphological variants that mimic other entities.

Within WHO grades I and II, as noted earlier, fibrous meningiomas bear a close resemblance to peripheral nerve sheath tumors. Distinction of these two entities is more of an academic point because they share similar prognoses. Otherwise, a panel of immunohistochemical stains is helpful in separating these tumors. Angiomatous meningiomas may mimic the appearance of a vascular malformation or hemangioblastoma. In addition to being highly vascular, hemangioblastomas are lipid-rich tumors, which stain with fat stains such as Oil Red O or Sudan black. These stains can be performed rapidly during intraoperative consultation and may facilitate the exclusion of this consideration. Occasionally, idiopathic hypertrophic pachymeningitis, plasma cell granuloma, or Rosai-Dorfman disease may masquerade as lymphoplasmacyte-rich meningioma. Once again, appropriate immunostains are helpful in interpreting these difficult cases.

According to the WHO definition, anaplastic meningiomas (grade III) "resemble carcinoma, sarcoma, or melanoma," which implies that these entities should be considered in the differential diagnosis. The diagnosis of meningioma is supported by the following immunohistochemistry profile: at least focal membranous positivity for EMA, positivity for vimentin, negativity for cytokeratin, and weak or negative staining for S-100 protein. Most carcinomas will stain positive for cytokeratin and negative for vimentin. Most sarcomas will be positive for vimentin only or also show positivity for mesenchymal markers such as smooth muscle actin. Melanoma is usually positive for one or more melanoma markers such as HMB-45, S-100 (often strong and diffuse staining), and melan-A. In truly difficult cases, such as in tumors that are convincingly positive only for vimentin, ultrastructural examination may be helpful. Features typical of meningioma include interdigitating processes and intercellular junctions. However, in very poorly differentiated tumors, even ultrastructural evidence of meningothelial differentiation may be unconvincing, and the presumptive diagnosis must be made on clinical grounds.

The tumor that most commonly has to be differentiated from an anaplastic meningioma is probably hemangiopericytoma. In contrast to anaplastic meningiomas (which only occasionally metastasize but have a median survival of under 2 years), 25 to 60% of hemangiopericytomas metastasize outside the central nervous system, and median survivals range from 5 to 12 years, depending on the histopathological grade.

Sometimes the classic "staghorn" vascular pattern of hemangiopericytoma is absent. In this case, its patternless high cellularity and numerous mitotic cells can lead to confusion with anaplastic meningioma. Generally, the diagnosis can be made on the basis of negativity for EMA and abundant intercellular reticulin in the hemangiopericytoma. Ultrastructural analysis may confirm the diagnosis by showing basal lamina–like material that is

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present in hemangiopericytoma (but not meningioma) and the lack of meningothelial characteristics.

For papillary meningiomas, the histopathologic differential diagnosis includes metastatic papillary adenocarcinoma, which is EMA and cytokeratin positive but vimentin negative; papillary ependymoma, which is glial fibrillary acidic protein (GFAP) positive; astroblastoma which is characterized by a typical palisaded pattern of astrocytic cells with broad and nontapering strongly GFAP-positive processes radiating toward the central blood vessels. Other tumors like choroid plexus papilloma may sometimes cause diagnostic confusion with a papillary meningioma.

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III

Molecular Biology and Laboratory Techniques

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Chapter 6

Molecular Biology of Meningiomas: Tumorigenesis and Growth

Brian T. Ragel and Randy L. Jensen

Introduction

Meningiomas are generally characterized as slow-growing tumors derived from arachnoid cap cells (**Fig. 6.1**). Meningiomas are graded as benign (~92% of meningiomas), atypical (6%), or anaplastic/malignant (4%), based on histological characteristics.[1] More recently, molecular factors have been found to correlate with tumor grade, and they are the focus of this chapter (**Fig. 6.2**). Genetic abnormalities are especially important in meningioma tumorigenesis, particularly loss of heterozygosity (LOH) of chromosome 22 and neurofibromatosis 2 (*NF2*) gene mutations. The role of radiation in meningioma development is also discussed. Furthermore, tumor growth may result from oncogene- or growth factor–mediated growth dysregulation. Studies have implicated various growth factors, including the major mediator of tumor angiogenesis, vascular endothelial growth factor (VEGF). The section on the role of sex hormones in meningioma tumorigenesis highlights hormones associated with tumor growth as well as the association between tumor progression and progesterone receptor loss. Finally, the possible involvement of the inflammatory cascade mediated through cyclooxygenase-2 (COX-2) is explored. Overall, this chapter provides an overview of the current knowledge of meningioma molecular biology with an emphasis on the history, clinical observations, and laboratory models that have made this possible.

Chromosomal Alterations in Meningiomas

Meningiomas were among the first tumors analyzed for genetic abnormalities. This was first performed by using Giemsa staining, but over time more sophisticated methods, including fluorescence in situ hybridization (FISH), comparative genomic hybridization, and spectral karyotyping, have confirmed and characterized these genetic changes. Chromosome 22 abnormalities, usually in the form of LOH or partial deletion of 22q, are the most frequent abnormalities in all meningioma types (**Fig. 6.3**).[1,2] Meningiomas occurring in the setting of NF2 always exhibit chromosome 22q abnormalities, whereas roughly 50% of meningiomas occurring sporadically exhibit 22q abnormalities. Chromosome 1 abnormalities have been associated with aggressive tumor phenotype.[1,3] In general, karyotypic aberrations increase with meningioma tumor grade (**Fig. 6.2**).[3] In addition to 1q loss, chromosome abnormalities associated with higher-grade meningiomas include 6q, 10p, 10q, 14q, and 18q (**Fig. 6.2**).[1,3]

Chromosome 22q: *NF2* Gene and the Gene Protein Product, Schwannomin/merlin

The link between chromosome 22 abnormalities and meningiomas was first suspected in patients with NF2. The hallmark of this disease is bilateral acoustic schwannomas. Interestingly, meningiomas occur in ~50% of patients with *NF2*. The *NF2* tumor suppressor gene, located on chromosome 22q12.1, and its protein product, schwannomin or merlin (**Figs. 6.3** and **6.4**), have been identified in cytogenetic and molecular studies.[1,2] The *NF2* gene codes for the schwannomin/merlin tumor suppressor (TuS) protein (moesin-, ezrin-, radixin-like protein), which is a part of the band 4.1 families of cytoskeleton-associated proteins (**Fig. 6.4**; **Table 6.1**).[1,2] Possible functions of schwannomin/merlin include roles in cytoskeletal functions (e.g., contact inhibition) and secondary signaling pathways (e.g., Ras) (**Fig. 6.4**).[2,4] Insertions or deletions of this gene produce a nonfunctional merlin protein, resulting in decreased cell adhesion and

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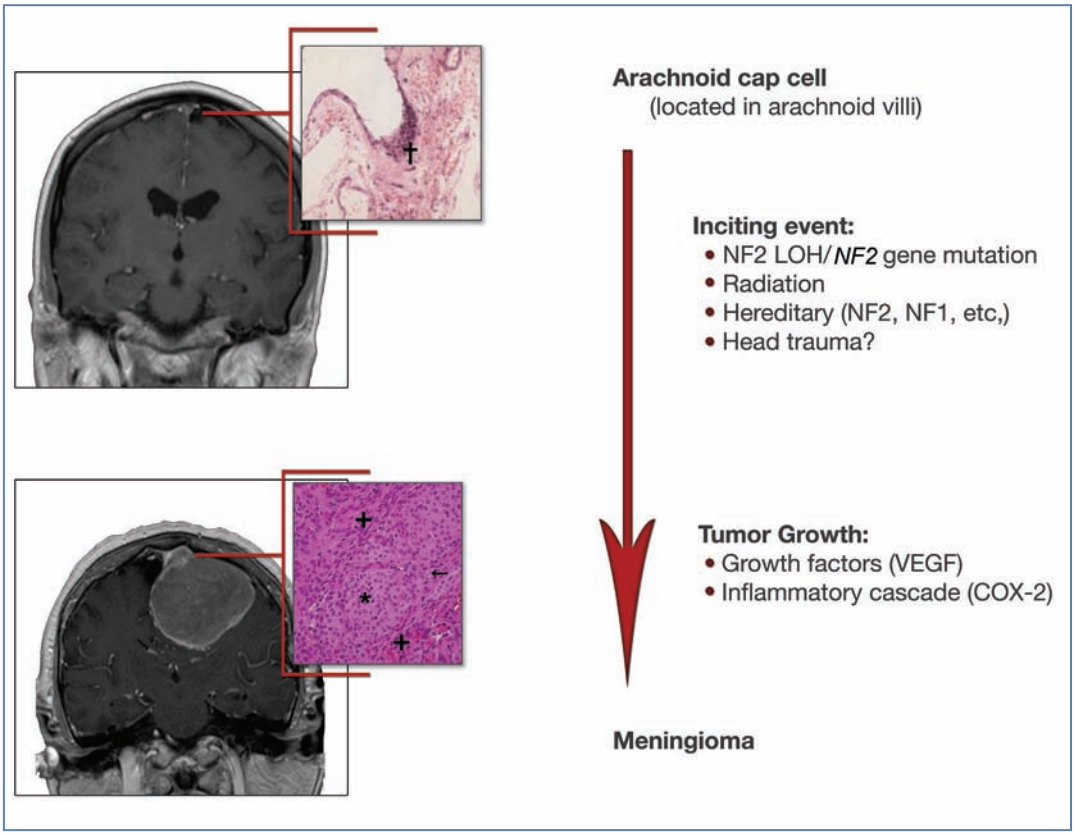


Fig. 6.1 Factors involved with meningioma tumorigenesis. Arachnoid cap cells (upper inset, *cross*) are the cell of origin. Known meningioma inciting events and factors associated with tumor growth are listed. (Lower left) Coronal T1-weighted magnetic resonance image with contrast showing parasagittal meningioma with histologic features consistent with meningothelial subtype (lower inset, World Health Organization grade I) with lobules (*asterisk*) surrounded by thin fibrous septa (*plus*) and intranuclear halos (*arrow*). COX-2, cyclooxygenase-2; NF, neurofibromatosis; LOH, loss of heterozygosity; VEGF, vascular endothelial growth factor. Figure modified, with permission from Ragel BT, Jensen RL, Couldwell WT. Inflammatory response and meningioma tumorigenesis and the effect of cyclooxygenase-2 inhibitors. Neurosurg Focus 2007;23(4):E7.

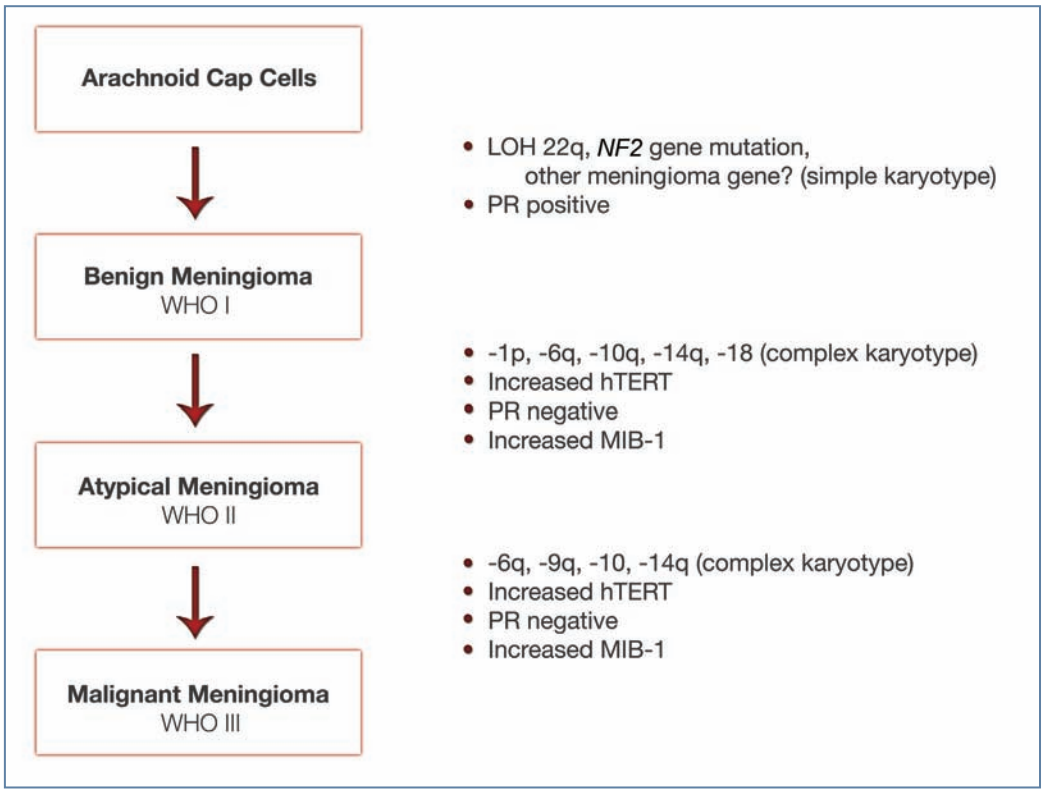


Fig. 6.2 Factors involved with meningioma tumor progression. Loss of chromosome 1p is a decisive step. Immunohistochemically, higher-grade tumors are associated with decreased progesterone receptor (PR) staining and increased MIB-1 nuclear staining. Human telomerase reverse transcriptase (hTERT) activity is also increased in higher-grade meningiomas. LOH, loss of heterozygosity; NF2, neurofibromatosis 2; WHO, World Health Organization.

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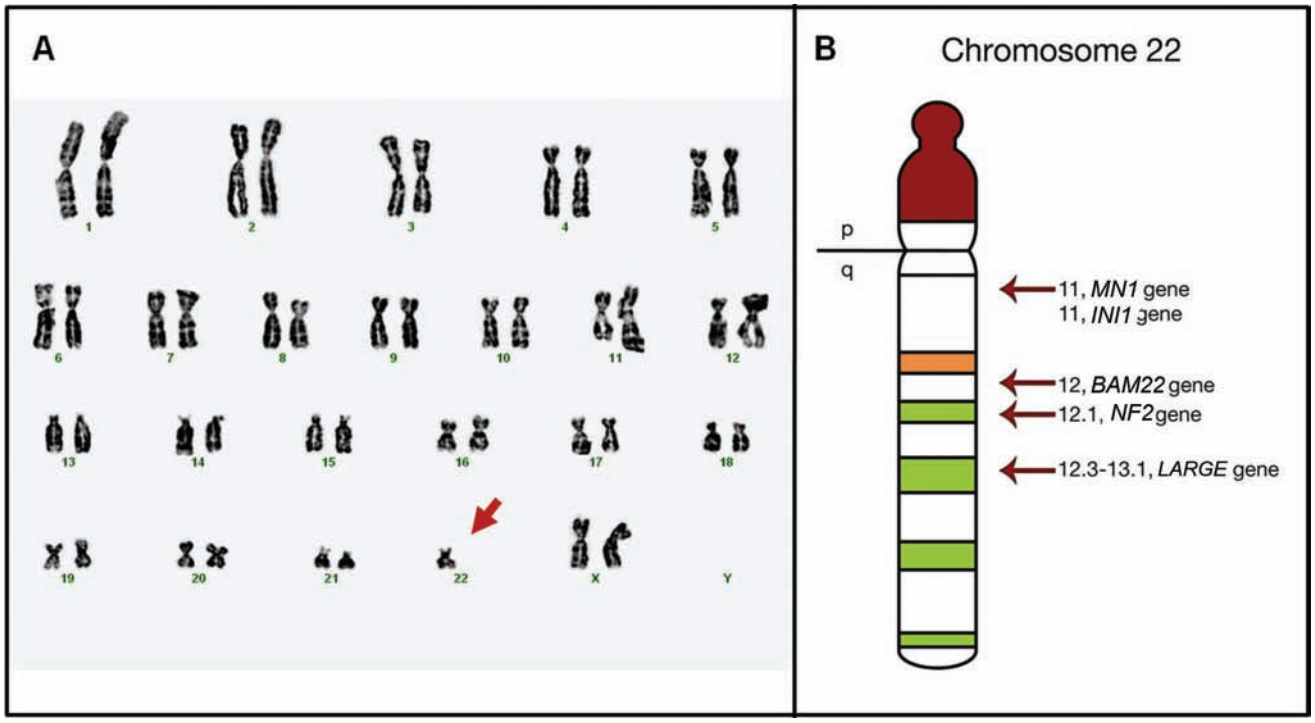


Fig. 6.3 **(A)** Representative G-band karyotype of a benign meningioma exhibiting the classic finding of monosomy 22 (i.e., loss of heterozygosity) (*arrow*). **(B)** Ideogram of the short (*p*) and long (*q*) arms of chromosome 22. Arrows indicate locations of proposed meningioma tumor suppressor genes. Figure reprinted with permission from Glick et al, 1989.[23]

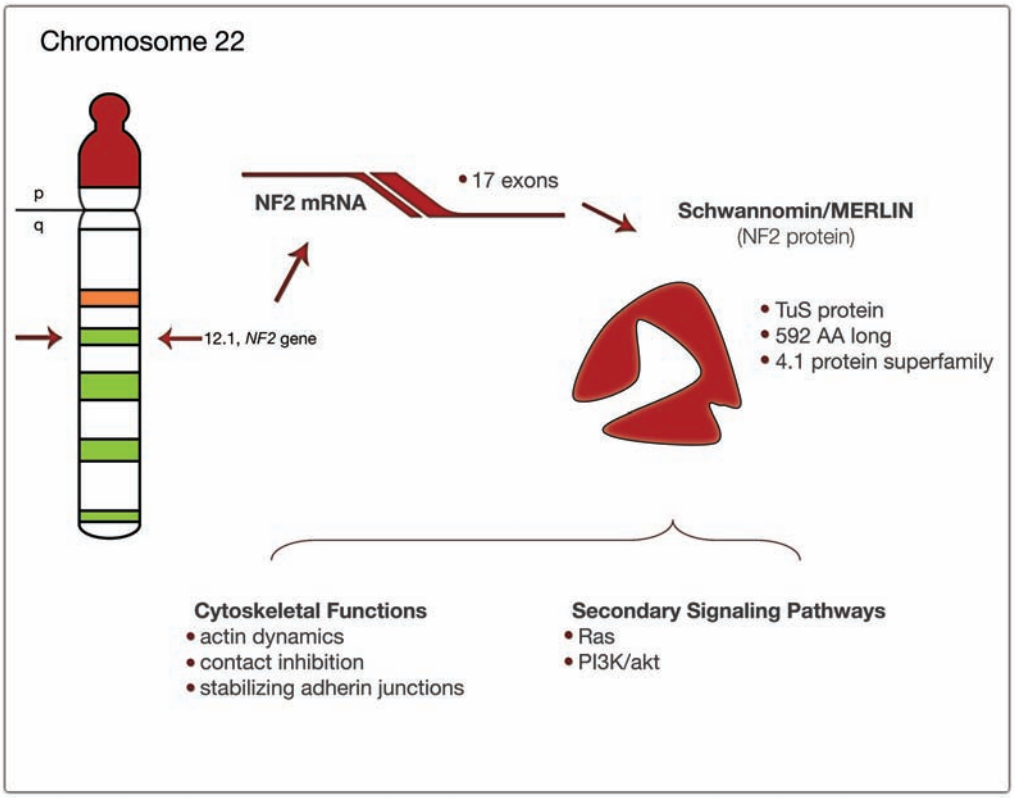


Fig. 6.4 Schematic of chromosome 22 containing the neurofibromatosis 2 (*NF2*) gene, which encodes for the tumor suppressor (TuS) protein schwannomin/merlin. Possible functions of the schwannomin/merlin TuS protein include roles in cytoskeletal functions and secondary signaling pathways.

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Table 6.1 Meningioma Chromosomal Abnormalities and Their Corresponding Genes, Proteins, and Protein Function

Chr	Gene(s)	Protein(s)	Function(s)	Note(s)
22q12.1	*NF2*	Merlin/ schwannomin	Regulation of cell growth and motility	Merlin protein is structurally similar to the protein 4.1 (DAL-1) superfamily
22q12	β-*adaptin*(*BAM22*)	BAM22 protein	Intracellular transport of receptor–ligand complexes	Beta-Adaptin-Meningioma-chromosome 22
22q12.3-q13.1	*LARGE*	LARGE protein	Synthesis of glycoprotein and glycosphingolipid sugar chains	Human-like acetylglucosaminlyltransferase
22q11	*MN1*	MN1 protein	Transcriptional regulation	
22q	*INI1* (SMARCB1/ hSNF5)	INI1 protein	Transcriptional regulation	
1p	der(1)(1qter→ 1p11::22q12→ 22pter)	Unknown	Unknown	1p13 implicated in radiation-induced meningiomas
9p21	CDKN2A ($p16^{INK4\alpha}$) $P14^{ARF}$ CDKN2B ($p15^{INK4b}$)	CDKN2A ($p16^{INK4\alpha}$) protein $P14^{ARF}$ protein CDKN2B ($p15^{INK4b}$) protein	Cell-cycle checkpoint proteins	
17q22	*BRIT1*	DEAH-box DNA helicase	BRCA1-dependent DNA repair and checkpoint functions	BRCA interacting protein 1, breast cancer susceptibility gene 1
18p11.3	*DAL-1*		Cytoskeletal protein	Member of 4.1 protein superfamily

Modified from Ragel and Jensen.[2]

tumorigenesis.[4] Reduced schwannomin/merlin expression has been demonstrated in sporadic meningiomas.[5]

Interestingly, the frequency of *NF2* gene mutation varies between meningioma subtypes. Among the three most common meningioma World Health Organization (WHO) grade I subtypes, 70 to 80% of fibroblastic and transitional meningiomas show *NF2* gene mutations, whereas the meningothelial subtype shows *NF2* mutations only 25% of the time, suggesting that cytogenetic differences in the tumorigenesis of meningioma subtypes may exist.[6] In both atypical and anaplastic meningiomas, the frequency of *NF2* gene mutations is ~70%.[3] Therefore, *NF2* gene mutations are probably involved with tumorigenesis but not tumor progression. In vivo mice experiments have provided further support for this theory but suggest that merlin loss alone is not sufficient for meningioma development.[7]

A search for a second tumor suppressor gene on 22q stems from the discrepancy between the frequency of chromosome 22 LOH, which exceeds that of *NF2* gene abnormalities.[6] Deletions of chromosome 22 are found in all NF2-associated meningiomas and in 54 to 78% of sporadic meningiomas. Further analysis of the *NF2* gene in sporadic meningiomas reveals that roughly one third to one half of these tumors have an inactivating mutation, often accompanied by loss of the other allele. These studies have resulted in other possible gene candidates (**Fig. 6.3**; **Table 6.1**).[2]

Chromosome 1

Deletions of the short arm of chromosome 1 are the second most frequent alteration detected by cytogenetic analysis of meningiomas (**Table 6.1**).[2,3] FISH studies showing monosomy 1p in 70% of atypical and almost 100% of anaplastic meningiomas indicate a correlation between loss of chromosome 1p and meningioma progression. Loss of 1p also correlates with tumor recurrence; the rate of recurrence is 30% with loss of 1p but only 4.3% when 1p is retained.[8] Which gene on the 1p arm results in the clinical effects on tumor is unknown, but research has suggested alkaline phosphatase as a possible tumor suppressor whose location on chromosome 1p (1p34→1p36.1) and loss of function are correlated with higher-grade meningiomas (**Table 6.1**).[3,8]

Other Meningioma Chromosomal Abnormalities

Many cytogenetic abnormalities are associated with meningioma progression and typical or anaplastic histology. These chromosomal aberrations include the presence of dicentric or ring chromosomes, losses of chromosome arms 1p, 6q, 7, 9p, 10, 14q, 18q, 19, or 20, and gains/amplifications of 1q, 9q, 12q, 15q, 17q, or 20q (**Fig. 6.2**).[3,8,9] It is unknown how these chromosome changes lead to tumor progression, although several chromosomes and

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genes appear to have specific associations with benign, atypical, and anaplastic meningioma grades. For example, benign meningiomas are more liable to have 14q deletions.[3] Roughly two thirds of anaplastic meningiomas exhibit altered cell-cycle checkpoint tumor suppressor genes located on chromosome 9p (**Table 6.1**). Further evidence implicating these deletions in meningioma pathogenesis comes from the significantly shorter survival times of patients. Other rare molecular abnormalities include phosphatase and tensin homologue gene deletion, cyclin-dependent kinase inhibitor 2c gene deletion, and ribosomal protein S6 kinase gene amplification.[1,2]

Cytogenetic alterations can also include changes in chromosome number. Sixty percent of meningiomas have been found to be hypodiploid, 33% diploid, 4.5% hyperdiploid, and 2.5% hypotriploid.[10] Complex karyotypes with hypodiploidy, structural rearrangements such as ring chromosomes, dicentrics, double minutes, and association between satellites seem to be associated with aggressive tumor characteristics.[3] Identification of a microsatellite instability phenotype in meningiomas has also been described. Finally, studies have identified what appear to be sporadic, familial, radiation-induced, and pediatric meningiomas.[2,11] Mounting evidence suggests that these tumors are genetically different, which may explain the more aggressive nature of the familial, radiation-induced, and pediatric cases.[2,11]

Radiation-Induced Meningiomas

The definition of a radiation-induced meningioma is one that occurs within a previously irradiated field, is histologically different from the original tumor, shows an elapsed time between irradiation and presentation (usually at least 5 years, average latency period of 11 to 43 years after exposure), does not have family history of phakomatosis, and was not present before irradiation.[11,12] Most patients with postradiation meningiomas were exposed during childhood to cranial radiation for the treatment of tinea capitis, leukemia, lymphoma, or craniopharyngioma.[11,12] Compared with patients with sporadic meningiomas, those with radiation-induced meningiomas have a younger age of presentation, a higher incidence of multiple tumors, a higher malignancy rate, an equal division between male and female (instead of the usual 3:1 ratio of female to male), and a higher recurrence rate after treatment with either surgery or radiation.[11] Estimated relative risk for development of a meningioma after low-dose childhood radiation is 10-fold that of those not exposed.[13]

Radiation-induced meningiomas usually exhibit a complex karyotype at presentation (i.e., the presence of multiple chromosomal arrangements). Unlike sporadic meningiomas, radiation-induced meningiomas have less frequent *NF2* gene inactivation and loss of chromosome 22, with chromosome 22 LOH occurring in 29 to 56% of cases, compared with 43 to 80% of tumors displaying a loss or translocation of 22q.[11] The most frequent cytogenetic abnormalities are found on chromosomes 1p (57 to 89%), 6q (67%), and 22 (29 to 58%).[11] The region of 1p13 seems to be especially vulnerable to radiation injury, implicating a role for an unknown gene in this region in the pathogenesis of radiation-induced meningiomas.[11,12] This may explain the link between aggressive behavior of both meningiomas with chromosome 1 loss and those thought to be radiation-induced.

Oncogene- and Growth Factor–Mediated Meningioma Angiogenesis and Tumorigenesis

Oncogenes

Human meningiomas are marked by enhanced expression of the *c-sis* and *c-myc* oncogenes.[14] Similarly, the rare *Ha-ras* and *c-mos* oncogenes have a higher activation in individuals with intracranial tumors, including meningiomas.[1,2] It has been proposed that the nuclear transcription-regulating genes *c-myc* and *c-fos* are normally under the control of tumor suppression genes, which are lost in meningiomas, resulting in >70% occurrence of proto-oncogene mRNA expression for *c-myc* and *c-fos*.[1,2] Tumor suppressor gene *TP53* mutation is a marker for meningioma malignant transformation and the *bcl-2* proto-oncogene is also correlated with higher grades.[1,2] Furthermore, expression of the *ROS1* oncogene for tyrosine receptor kinase is common in meningiomas, indicating a possible role in the origin of these tumors. Telomerase activity is rare in benign tumors and present in roughly half of atypical and most malignant meningiomas. The human telomerase reverse transcriptase (*hTERT*) gene encodes for the catalytic component of the telomerase complex, and increasing meningioma grade correlates with this gene's upregulation (**Fig. 6.2**).[15] Normal cells stop dividing when telomeric DNA becomes shortened through continued mitotic division. By continually adding telomeres to DNA, cancer cells can avoid terminal differentiation and senescence. Although no single oncogene has been directly implicated in meningioma development, it is possible that either one or multiple oncogenes contribute.

Epidermal Growth Factor/Transforming Growth Factor-α

Functional epidermal growth factor receptors (EGFR) are widely expressed in human meningiomas (**Table 6.2**). No correlation has been demonstrated between histopathological subtype or tumor grade and the presence of EGFR in meningiomas.[16] Normal adult human and rat meninges do not exhibit detectable EGFR, but neonatal rat meninges do, suggesting a role in development for EGF.[17] These receptors have also been demonstrated on cultured meningioma cells.[16] Transforming growth factor-α is a potent agonist of the EGFR, and this protein has been demonstrated in meningiomas taken from hu-

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Table 6.2 Expression of Growth Factors and Receptors in Meningiomas

Growth Factor	Positive	Total	Percent
EGF	404	542	75
PDGF	178	180	99
FGF	16	16	100
FGFr	41	43	95
IGF I	30	39	77
IGF II	11	16	69
VEGF	180	233	77
VEGFr	2	3	67

EGF, epidermal growth factor; PDGF, platelet-derived growth factor; FGF, fibroblast growth factor; FGFr, FGF receptor; IGF I, insulin-like growth factor receptor type I; IGF II, insulin-like growth factor receptor type II; VEGF, vascular endothelial growth factor; VEGFr, VEGF receptor. Data adapted from Rangel and Jensen.[2]

man patients.[18] Recurrent tumors have been shown to have increased TGF-α expression over time, even when the tumors remained histologically benign.[14]

EGF exposure activates signal transduction pathways stimulating cell proliferation and DNA synthesis in human meningioma cultures.[16] We have shown that this EGF-mediated signal transduction pathway and subsequent growth are inhibited by calcium channel antagonist treatment both in vitro and in vivo.[19]

Platelet-Derived Growth Factor

Most meningiomas studied express platelet-derived growth factor receptors (PDGFr) (**Table 6.2**). PDGF stimulates proliferation and DNA synthesis in human meningioma cultures through a mechanism involving the oncogene *c-fos*.[20] PDGF is produced by meningiomas, and exposure to the protein stimulates DNA synthesis[21]; Northern blot analysis has demonstrated *c-sis*/*PDGF-2* proto-oncogene and the *PDGFR* gene in meningiomas taken from humans; whereas only the *PDGFR* gene, but not the proto-oncogene, was found in control meninges.[16] Studies have shown that PDGF was a component of "conditioned media" produced from meningiomas in culture, which could stimulate growth in both meningioma and neuroblastoma cells in serum-free cell culture. This autocrine effect could be blocked by an antibody to PDGF, especially antibodies directed toward PDGF-BB.[22]

One might postulate that overexpression of a given growth factor would result in growth dysregulation produced by autocrine loops, resulting in increased proliferation of tumor cells. This area of meningioma research is especially appealing because of the ability to inhibit various growth factor receptors with systemic therapies.

Fibroblast Growth Factor

Fibroblast growth factor (FGF) receptors as well as FGF protein have been found in all meningiomas studied (**Table 6.2**). Like the other growth factors mentioned in this chapter, FGF has been reported to stimulate cell proliferation and DNA synthesis in human meningioma cultures.[18]

Insulin-Like Growth Factor and Somatostatin

Patients with acromegaly have a higher incidence of meningiomas than the general population. Insulin-like growth factor (IGF) I and II receptors have been found on meningiomas.[23] Seventy-seven percent of meningiomas studied have been positive for IGF-I (30/39) and 11/16 (69%) positive for IGF-II (**Table 6.2**). Glick et al[23] first demonstrated that meningiomas in serum-free media showed increased growth in the presence of insulin.

Somatostatin receptors are present on meningiomas in high density, and the addition of somatostatin in vitro inhibits meningioma cell proliferation.[18,24] Schulz et al[24] showed that 73% (29/40) of meningiomas were positive for the sst2A subtype somatostatin receptor. In contrast, all other somatostatin receptors were noted to stain weakly and sporadically. García-Luna et al[18] reported on the clinical use of octreotide, a long-acting somatostatin agonist, in three patients with unresectable meningiomas. Findings included the subjective improvement of symptoms but no change in meningioma size based on computed tomographic (CT) measurements. This report suggests the safety of octreotide but is too small to draw any meaningful conclusions.

Vascular Endothelial Growth Factor

VEGF is secreted by meningiomas, and two of the major receptors for VEGF have been located on the intratumoral vasculature of these tumors.[16] Many meningiomas are positive for VEGF protein or for VEGFr (**Table 6.2**).[16] VEGF has been implicated in meningioma peritumoral edema and angiogenesis. Several studies have correlated meningioma peritumoral edema with VEGF expression by mRNA transcription and immunohistochemical detection of protein within the tumor. The relationship of meningioma grade and VEGF expression is less clear; some reports show a positive correlation and others reported no correlation.[16] Furthermore, studies that have shown a correlation between VEGF and meningioma grade did not show increased microvascular density or invasiveness. Others have shown a high correlation between VEGF expression and neovascularization in meningioma. VEGF is principally regulated by the transcription factor hypoxia inducible factor-1 (HIF-1). We have shown that HIF-1 and VEGF are elevated in embolized meningiomas but not under normal circumstances.[25] Taken together, these data suggest that VEGF may be serving functions other than angiogenesis in meningiomas, such as stimulating tumor growth in an autocrine fashion.

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Sex Hormones and Meningiomas

In his description of bitemporal visual field defects and optic atrophy associated with sellar meningiomas, Cushing noted that one of the patients had worsening visual symptoms during pregnancy, with improvement after delivery. The association between pregnancy or menstruation and worsening neurological symptoms (usually visual) has been reaffirmed by other investigators and is further supported by the fact that the majority (two thirds) of patients with meningioma are women.[16] Other investigators have raised the question of the hormonal dependency of meningiomas. In addition, a statistical association between obesity and meningiomas has been found. Conversion of androgens to estrogens in peripheral fat has been hypothesized to make obese men and women more likely to develop hormone-related tumors.[16]

Since sex steroid receptors were discovered in meningiomas, further studies have revealed little estrogen receptor binding but a high percentage of meningioma cells that have progesterone and androgen receptors.[16] Progesterone receptors have been identified from the cytosols of human arachnoid granulations (from which meningiomas presumably originate). **Table 6.3** contains a summary of all measurements of estrogen, progesterone, and androgen receptors in meningiomas by four methods, which reveals that 532/2384 (22%) of meningiomas tested were positive for estrogen receptors, 1537/2039 (75%) were positive for progesterone receptors, and 152/243 (63%) were positive for androgen receptors (**Table 6.3**).[16] Interestingly, the expression of the progesterone receptor alone appears to signal a more favorable clinical outcome than lack of progesterone receptor or the presence of estrogen receptors in meningiomas, which correlate with a higher number of karyotype abnormalities, a higher proportional involvement of chromosomes 14 and 22 in de novo tumors, and an increasing potential for aggressive clinical behavior, progression, and recurrence of these lesions (**Figs. 6.1** and **6.2**).[16]

The Inflammatory Cascade and Cyclooxygenase-2

Clinical evidence supports a link between previous head trauma and meningioma formation.[26] In vivo and in vitro studies show that the proinflammatory enzyme COX-2 is upregulated after head trauma as well as ubiquitously expressed in meningioma operative specimens.[26] The ability to inhibit this enzyme with COX-2 inhibitors (e.g., celecoxib) has proven to decrease meningioma growth in vitro and in vivo.[26]

Meningioma Clonality

X-chromosome inactivation studies and single *NF2* gene mutations indicate that most solitary meningiomas are monoclonal in origin, although polymerase chain reaction evidence suggests that a small number may be polyclonal.[2,27] Interestingly, some patients with multiple meningiomas show X-chromosomal analysis and mutational *NF2* gene evidence suggesting that these multiple tumors are monoclonal in origin, supporting the concept of dural dissemination of multiple meningiomas via the subarachnoid space.[27] Approximately 50% of multiple meningiomas exhibit different *NF2* gene mutations, however, indicating independent tumorigenesis origins.[2]

Models to Study Meningiomas

Human meningiomas have been grown in cell culture for research and pathological diagnosis since Dr. Cushing and Dr. Eisenhart did so in the 1920s. This has allowed ample opportunity for development of meningioma models. The morphology of meningioma cells in culture is bipolar cells with relatively large, rounded central nuclei and generous cytoplasm.[28] At confluence, cells cease to be bipolar and become homogeneously flat and polygonal with early senescence and prolonged contact inhibition. Meningiomas and normal meninges are immunohistochemically reactive for epithelial membrane antigen and vimentin, a marker of mesenchymal tissues. Electron microscopy studies of cultured meningiomas reveal interdigitation of cell membranes and intercellular junctions such as gap junctions and desmosomes.[28] Most researchers believe that meningiomas lose their primary characteristics in culture and become largely populated by cells different from the original tumor after several passages, but that in vitro growth studies can be undertaken in the early stages. Because of in vitro senescence of meningiomas grown from operative specimens, the immortal cell lines IOMM-Lee and CH-157 have been used for laboratory work.[28] However, these aggressive cell lines behave like malignant meningiomas, which might make

Table 6.3 Estrogen, Progesterone, and Androgen Receptors in Meningiomas

Assay	Estrogen (%)	Progesterone (%)	Androgen (%)
Dextran-coated charcoal, radioligand assay	381/1424 (27)	987/1263 (78)	65/109 (60)
Enzyme immunoassay	119/843 (14)	501/705 (71)	33/53 (62)
Northern blot analysis	35/117 (30)	49/71 (69)	54/81 (66)
Total	**535/2384 (22)**	**1537/2039 (75)**	**152/243 (63)**

Data adapted from Rangel and Jensen.[2]

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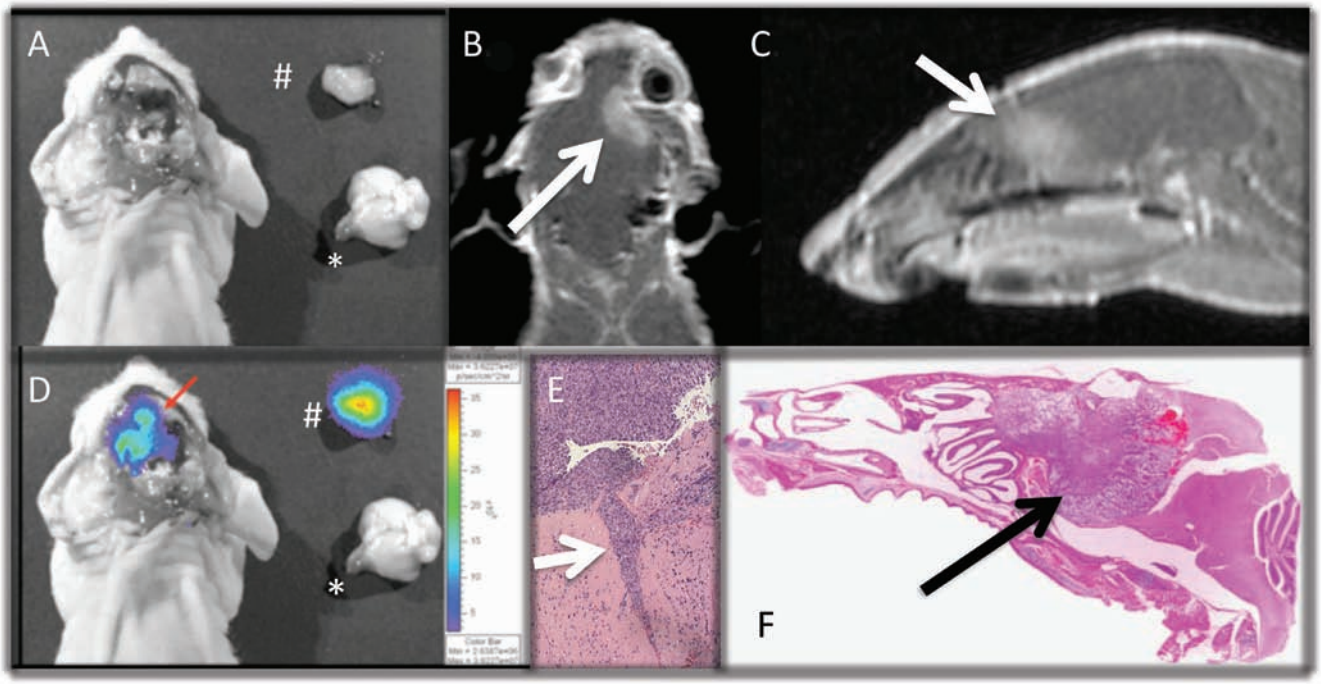


Fig. 6.5 **(A)** Mouse with brain (*) and skull base meningioma (#) removed from skull, before bioluminescent imaging. **(B)** Axial T1-weighted brain magnetic resonance imaging (MRI) with gadolinium enhancement of skull base meningioma (*arrow*). **(C)** Midsagittal MRI with gadolinium enhancement of skull base meningioma (*arrow*). **(D)** Same mouse image as in **(A)** during bioluminescence imaging showing radiance reading from mouse xenograft meningioma (*) and lesser signal from skull base (*arrow*). **(E)** 20× Hematoxylin and eosin (H&E)-stained meningioma tumor section demonstrating invasion of meningioma along Virchow-Robin spaces (*arrow*). **(F)** Parasagittal 2× H&E stained mouse brain and skull demonstrative of invasive skull base meningioma (*arrow*).

conclusions made in the laboratory with these cell lines not applicable to their benign counterparts. Recently, a benign meningioma cell line, Ben-Men-1, has been immortalized by retroviral transfection of the *hTERT* gene, thus bypassing cellular senescence.[15,29] Even though this is an immortalized cell line, it may be a closer model to benign meningiomas.

Although in vitro models offer the opportunity to understand some aspects of meningiomas, an in vivo model of meningioma cell growth is necessary for a better understanding of meningioma tumor biology and for testing the efficacy of new therapeutic measures before attempting clinical human trials.[28,30] Studies of meningioma cells in culture have the disadvantage of the inability to study in vivo characteristics of these tumors such as morphology, neovascularization, or growth characteristics.[28,30] Numerous xenograft animal transplantation models have been developed: guinea pig eyes; the chorioallantoic membrane of the duck and chick; and the subcutis, subrenal capsule, flank, brain, and skull base of athymic mice. Most researchers have used the mouse meningioma flank model because of the ease with which tumors can be continually assessed during treatments and the relatively low cost of using mice as an animal model.[28] Subcutaneous xenografts allow serial tumor volume measurement but have been criticized for low tumor "take," measurement inaccuracies, and the fact that this site differs from the intracranial space in physiologic and immunologic parameters.[28,31] The low tumor implantation rate is overcome using cells initially mixed in Matrigel (a mixture of basement membrane proteins, tissue plasminogen activators, and growth factors that polymerizes at body temperature), but the model still lacks advantages of a natural intracranial model.[21] Intracranial models, although ideal, require serial imaging with magnetic resonance imaging, which is costly, time consuming, and logistically difficult. Recently, an intracranial bioluminescence meningioma mouse model has been developed to take advantage of this noninvasive, inexpensive technique for measuring serial tumor growth (**Fig. 6.5**).[32] This has the potential to allow for repeated measures of tumor growth over time, enabling for preclinical studies of therapeutic modalities for the treatment of meningiomas.[32]

Summary

Meningioma tumorigenesis has been linked to an initial inciting event of radiation, head trauma, and/or a genetic predisposition. LOH of chromosome 22 and *NF2* gene mutation are implicated in ~50% of sporadic and 100% of NF2-associated meningiomas. The *NF2* gene encodes for the cytoskeletal protein schwannomin/merlin, which probably functions in cell contact inhibition. Tumor progression may also result from oncogene expression, unregulated growth factor–mediated or sex hormone-

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mediated signal transduction, and/or inflammatory cascade dysregulation. These dysregulated systems ultimately lead to tumor angiogenesis and cellular proliferation. Ongoing in vitro and in vivo work with immortal meningioma cell lines and xenograft models provides a means for further study of meningioma tumorigenesis. With a better understanding of the molecular biology of meningioma development and growth, treatment modalities may be developed to treat these tumors when current modalities of surgery and radiation fail.

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IV

Clinical Considerations

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Chapter 7

Natural Course of Untreated Meningiomas

Shigetoshi Yano and Jun-ichi Kuratsu

The advances in diagnostic techniques and instrumentation have led to an increase in the identification of incidental meningiomas in the growing elderly population. In 1989, Kuratsu et al[1] established the Kumamoto University Brain Tumor Data Bank, and between 1989 and 2008, 1784 new cases of meningiomas were diagnosed in the Kumamoto Prefecture. Of those, 714 (40%) were asymptomatic. The rate of incidence of asymptomatic meningiomas has increased in the past decade, and asymptomatic meningiomas account for almost half of the meningiomas diagnosed (**Fig. 7.1**).

The information on untreated meningiomas that has been accumulated in recent years has contributed to clarifying their natural course of progression. In this section, the natural course of untreated meningiomas is reviewed with a focus on the features of growth rate, predictable factors for growth, rate of symptomatic change, and therapeutic strategy. The untreated meningiomas described in this section are limited to incidental meningiomas that occur without related symptoms and do not include radiation-induced, neurofibromatosis type 2–associated meningiomas, or multiple meningiomas. The untreated meningiomas were diagnosed on the basis of the presence of an extraaxial mass, with broad-based attachment along the dura or attachment to the choroid plexus in the ventricles, that was homogeneously and markedly enhanced by contrast medium.

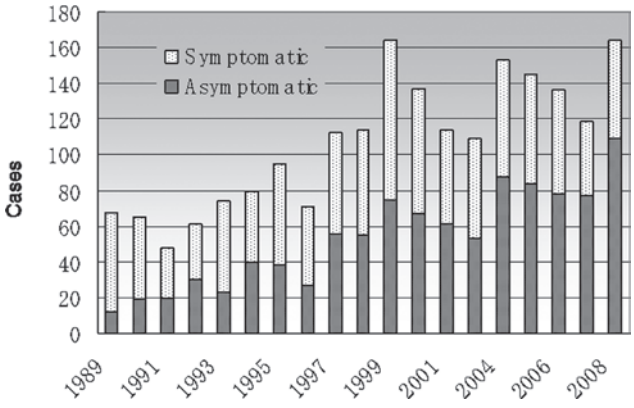


Fig. 7.1 Rate of asymptomatic meningiomas in Kumamoto-Brain Tumor Data Bank.

Pattern and Rate of Tumor Growth

The reported growth rates of asymptomatic meningiomas are summarized in **Table 7.1**. Olivero et al[2] reported that 78% of 45 meningiomas followed up over a 2.5-year period did not grow, and the remaining 22% showed an increase in the mean maximum tumor diameter, with a growth rate of 0.24 cm/year. Herscovici et al[3] reported that only one third of the meningiomas in their study grew during the follow-up period, and the mean annual growth was 3.2 mm. Yano et al[4] found that 25 of 67 (37.3%) asymptomatic meningiomas continued to grow during the 5-year follow-up period; their growth rate, calculated by the maximum tumor diameter, was 1.9 mm/year, and this finding was consistent with those of previous studies. In these studies, tumor diameter was used to quantify tumor size. Although this method is the easiest and most practical for measuring tumor growth and may be useful for determining treatment response, this method is not accurate.[5,6] Because the shape of meningiomas, especially at the cranial bases, is complex, tumor growth in any direction that is not in line with the measuring axis can go undetected.[7]

Growth rate can be more accurately estimated by volumetric analysis. Nakamura et al,[7] who measured tumor volume on computed tomography and magnetic reso-

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Table 7.1 Reported Rates of Tumor Growth in Untreated Meningiomas

Authors	Followed Cases	Method	Growth Cases (%)	Average Follow-up Year (range)	Tumor Growth Rate per Year
Firsching et al (1990)[15]	17	Volume	Not defined	1.8 (0.2–7.4)	3.60% (0.5–21.0%)
Olivero et al (1995)[2]	45	Maximum diameter	10 (22.2)	2.7 (0.5–15)	0.24 cm
Braunstein and Vick (1997)[8]	5	Three diameters	1 (20.0)	7.9 (3.3–11.5)	2.43 cm^3
Go et al (1998)[12]	32	Maximum diameter	4 (14.0)	5.1 (0.4–15.2)	12% (1.2–25.6%)
Kuratsu et al (2000)[1]	63	Volume	20 (31.7)	2.3 (1.0–8.0)	ND
Niiro et al (2000)[11]	40*	Maximum diameter	14 (40.0)	3.2 (0.5–8.1)	0.08 cm
Yoneoka et al (2000)[10]	37	Volume	9 (24.3)**	4.2 ± 0.7 (0.5–17)	5.3 ± 2.1 cm^3
Nakamura et al (2003)[7]	41	Volume	ND***	3.6 (0.5–8.8)	0.796 cm^3 (0.03–2.62)
Herscovici et al (2004)[3]	44	Maximum diameter	16 (36.4)	5.6	3.9 ± 3 mm
Nakasu et al (2005)[6]	5	Volume	2 (40.0)**	5.9 (4.2–8.7)	0.31 cm^3 (0.22–0.40)
Yano et al (2006)[4]	67	Maximum diameter	25 (37.3)	7.8 (5.0–13.6)	1.9 mm (0.42–11.47)
Hashiba et al (2009)[5]	70	Volume	44 (62.9)	3.3 (1.0–10.3)	15–25%

* Patients were older than 70 years old.
** Growth was defined if the tumor volume increased more than 1 cm^3/year.
*** Described as 66% of growth rates were less than 1 cm^3/year.
Abbreviation: ND, not described.

nance imaging (MRI) and calculated the tumor-doubling time in 41 patients, found that the mean growth rate was 0.796 cm^3/year (range 0.03 to 2.62 cm^3/year) resulting in a 14.6% volume increase per year. At that rate, the mean tumor doubling time was 21.6 years. After conducting studies with 20 patients, including 13 symptomatic patients, Nakasu et al[6] reported three meningioma growth patterns: Atypical meningiomas grew exponentially, whereas benign meningiomas grew exponentially, linearly, or not at all. More recently, Hashiba et al[5] performed serial volumetric assessments throughout the follow-up period in patients with incidentally discovered meningiomas and investigated the growth patterns of these lesions by regression analysis. Results showed that 16 of the tumors grew exponentially and 15 grew linearly. The authors emphasized that in lesions that followed linear growth patterns, the yearly growth rates varied over time.

Braunstein and Vick[8] hypothesized that meningiomas have variable growth rates, and that the growth rates reduce or even cease after the tumors attain a certain size. Another group also indicated that tumor doubling time and relative growth rates change during the life of the tumor.[7] On the other hand, growth patterns of incidentally diagnosed meningiomas may fit the Gompertzian model, which describes exponential growth in the early stage, linear growth in the middle stage, and a plateau in size as tumor size increases.[5,9]

Although average tumor growth rates cannot be compared directly among the different studies because they employed different measurement methods, most asymptomatic meningiomas exhibited minimal growth.

Factors That Predict Tumor Growth

Several authors have identified various factors as predictors of tumor growth (**Table 7.2**). Olivero et al[2] showed that tumors in patients younger than 60 years of age did not grow more frequently or more rapidly than those found in patients older than 60 years of age, although the average follow-up time in this study was short, ~2.5 years. Nakamura et al[7] also showed that in patients aged < 60 years, the absolute and relative tumor growth rates were higher and the tumor doubling time was shorter than in older patients. Herscovici et al[3] indicated that older age was significantly associated with lower incidence of tumor growth and suggested that this relationship may involve aging-induced endocrine changes.

Yoneoka et al[10] reported that tumor growth rates increase as tumor volume increases, and they suggested that patients with relatively large tumors should be observed carefully because the risk of tumor growth is high for these patients. Similar findings were reported in Niiro et al.[11] On the other hand, Nakamura et al[7] reported that

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Table 7.2 Reported Initial Tumor Size, Rate of Symptomatic Change, and Predictive Factors for Tumor Growth in Untreated Meningiomas

Authors	Followed Cases	Initial Tumor Size (range)	Patients Become Symptomatic (%)	Significant Factors Related to Tumor Growth
Firsching et al (1990)[15]	17	4.7 cm^3	ND	-
Olivero et al (1995)[2]	45	2.15 cm (0.5–5 cm)	0 (0.0)	-
Braunstein and Vick (1997)[8]	5	ND	1 (20.0)	
Go et al (1998)[12]	32	2.06 cm (1–7 cm)	1 (3.1)	Calcification
Kuratsu et al (2000)[1]	63	9.75 cm^3	ND	Calcification
Niiro et al (2000)[11]	40	2.60 cm	5 (12.5)	Tumor size, T2 signal, calcification
Yoneoka et al (2000)[10]	37	ND	2 (5.4)	Age, volume of tumor
Nakamura et al (2003)[7]	41	9.0 cm^3	-	Age, calcification, T2 signal
Herscovici et al (2004)[3]	44	17 ± 8 mm (3–45 mm)	ND	Age
Nakasu et al (2005)[6]	5	6.56 cm^3 (0.27–17.4)	ND	Calcification
Yano et al (2006)[4]	67	2.40 cm (0.5–6.6)	11 (16.4%)	Calcification
Hashiba et al (2009)[5]	70	10.4 cm^3 (0.63–69.2)	0.0	Calcification

Abbreviation: ND, not described.

initial tumor size cannot be considered as a predictive factor because tumor size shows only a moderate positive correlation with absolute annual growth rate and shows no correlation with tumor doubling time.

It has also been demonstrated that MRI hypointensity on T2-weighted images is associated with slowed tumor growth,[1] and that MRI hyperintensity on T2-weighted images is associated significantly with faster tumor growth.[7,11]

There are no standard criteria for evaluating meningioma growth by radiology. The definition of tumor growth varies in the literature. For instance, in volume measurement studies, an annual growth rate of > 1 cm^3/year[10,6] or volume increases > 15%[5] were considered tumor growth. In diameter measurement studies, growth has been defined as a change in tumor size of at least 2 mm,[3] 5 mm,[12] or any measurable change.[4] From these variations of definition, each study may yield very different predictive factors. However, even if these differences are taken into consideration, calcification may be the most significant predictable factor of slow tumor growth. Many authors have pointed out that meningiomas without calcification on imaging are more likely to progress than are calcified meningiomas,[1,4–7,11,12] and no negative correlation between calcification and slow growth has been reported.

No significant differences in location have been reported between growing and nongrowing tumors. Whether a tumor was located at the skull base, or not, and whether it was located at the falx or parasagittal location did not appear to affect tumor growth (**Table 7.2**).

Hashiba et al[13] suggested that the proliferative potential of symptomatic meningiomas can be predicted using a noninvasive preoperative examination that considers the presence of peritumoral edema, ambiguous brain-tumor borders, and irregular tumor shape. However, the applicability of this method to predict the growth of asymptomatic meningiomas should be investigated.

◆ Symptomatic Change

Few reports have described the symptomatic changes during follow-up in patients with incidentally diagnosed meningiomas (**Table 7.2**). In three reports, one or two patients became symptomatic during follow-up.[8,10,12] Although rates of symptomatic change varied between studies, depending on how many cases were followed, incidental meningiomas appear to rarely become symptomatic within a follow-up period of 2 years or less.

On the other hand, Yano et al[4] reported that 11 of 67 patients (16.4%) developed symptoms over a mean follow-up period of 3.9 years. All of these patients had an increase in tumor size during the same period (**Table 7.3**). Niiro et al[11] reported that 5 of 40 (12.5%) elderly patients showed symptoms during follow-up, and the mean time for symptom occurrence was 3.2 years. These studies showed similar results and demonstrated that asymptomatic meningiomas become symptomatic only rarely, and only several years into follow-up.

We compared the maximum size of asymptomatic meningiomas registered in the Kumamoto Brain Tumor Data Bank (1989–2008) with the maximum size of asymptomatic meningiomas operated on at Kumamoto University

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Table 7.3 Characteristics of 10 Patients Who Became Symptomatic during Follow-Up

Age	Sex	Location	Follow-up Period (yrs.)	Tumor Size (cm)		Symptoms	Treatment
				Initial	Latest		
56	F	Cerebellar	1.2	4.0	5.0	Cerebellar sign	Operation
66	F	Convexity	2.0	3.0	3.5	Seizure	Follow-up
67	F	Parasagittal	1.6	2.4	4.2	Seizure	Operation
67	F	Tentorial	2.8	3.5	4.0	Ophthalmoplegia	Operation
69	M	Convexity	1.0	4.0	4.5	Hemiplegia	Operation
74	F	Convexity	3.5	3.0	3.8	Seizure	Follow-up
79	F	Temporal base	7.4	3.0	5.0	Hemiplegia	Follow-up
81	F	Falx	5.3	4.8	5.5	Dementia	Follow-up
81	F	Sphenoid ridge	9.4	3.4	6.0	Gait disturbance	Follow-up
82	F	Frontal base	4.6	6.0	6.0	Disorientation	Follow-up
		Mean	**3.9**	**3.7**	**4.6**		

With permission from Yano S, Kuratsu J-i; Kumamoto Brain Tumor Research Group. Indications for surgery in patients with asymptomatic meningiomas based on an extensive experience. J Neurosurg 2006;105:538–543.

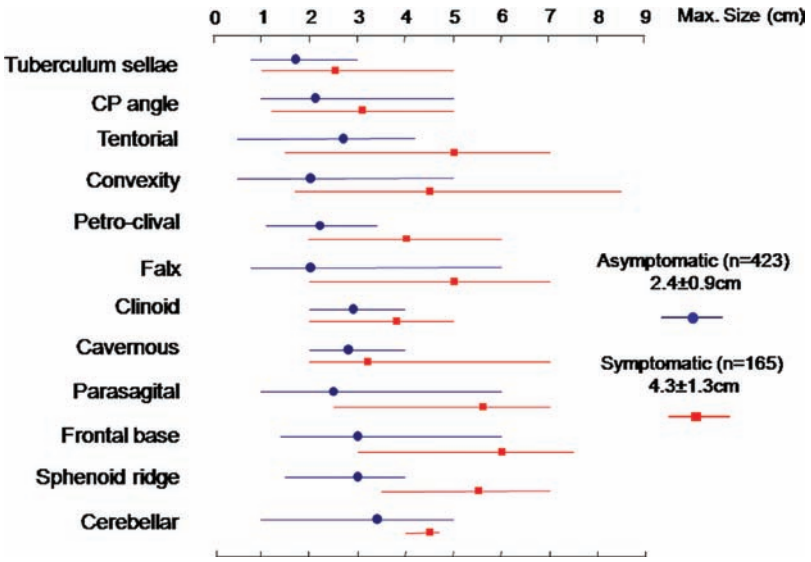


Fig. 7.2 Comparison of the maximum tumor diameter at the time of diagnosis in asymptomatic and symptomatic meningiomas in Kumamoto University. The tumor location and the mean tumor diameter at the time of diagnosis for 423 asymptomatic and 165 symptomatic meningiomas in each location were shown. The mean size of asymptomatic meningiomas was 2.4 ± 0.9 cm, whereas symptomatic meningiomas were 4.3 ± 1.3 cm. Symbols with burr indicate the median tumor size and range. CP, cerebellopontine.

during the same period (**Fig. 7.2**). Irrespective of their location in the brain, the mean diameter of asymptomatic meningiomas at the time of diagnosis (2.4 ± 0.9 cm, n = 423) was smaller than that of symptomatic tumors at the time of diagnosis (4.3 ± 1.3 cm, n = 165). However, in the tuberculum sellae, CP-angle, tentorial, convexity, and petroclival regions, there were some symptomatic tumors smaller than 2 cm, and in the sphenoid ridge and cerebellar regions there were symptomatic meningiomas of larger sizes. Yano et al[4] suggest that at follow-up understanding the tumor size and location is important.

Bindal et al[14] reported the natural course for 40 untreated skull base meningiomas involving the cavernous, anterior clinoid, and petroclival regions. Eleven of the 40 patients (27.5%) experienced neurological progression during a mean follow-up of 7 years (range 10 to 312 months). However, this clinical progression was mild and in most instances did not change the patients' ability to function. The authors stated that understanding the indolent natural course of these tumors is critical for rational therapeutic decision making and for evaluating the effects of treatment.

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Therapeutic Strategy

The management of incidental meningioma is controversial and includes observation, surgery, radiosurgery, and fractionated radiotherapy. The majority of incidental meningiomas appear to show minimal growth. However, it should be borne in mind that some patients, albeit a small number, exhibit rapid increases in tumor volume. Yoneoka et al[10] recommend that surgery should be considered for patients whose tumor growth rate is more than 1 cm^3/year on repeated examinations, and the patient's age and medical condition should be taken into account. Nakamura et al[7] recommend that surgical excision should be considered only if repeated tumor growth is observed and if the tumor significantly compresses brain parenchyma or surrounding neurovascular structures.

Surgery and radiosurgery are associated with morbidity and mortality, particularly in the elderly population. Kuratsu et al[1] reported that the perioperative morbidity in patients younger than 70 years who have asymptomatic meningiomas is only 3.5%, whereas the perioperative morbidity in patients older than 70 years who have asymptomatic meningiomas is 20%. Therefore, surgical intervention for elderly patients should be considered carefully.[4]

Conservative treatment should be recommended for patients over 60 and patients under 60 with high-surgical-risk tumors, such as skull base tumors.[3,14] Surgery should be recommended for patients under 60 who have low-surgical-risk tumors, such as convexity tumors. These patients have a higher incidence of tumor growth, longer follow-up, and lower surgical morbidity than do other meningioma patients.[13] Surgery should also be recommended for tumors that become symptomatic, grow, or are large or irregularly shaped. Brain edema and mass effects are also indications for resection.[3]

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Chapter 8

Meningiomas in Children

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Meningiomas are not common in the pediatric population. When they do occur, they are less likely to appear and behave like their counterparts in the adult population. Their pathological characteristics can also be quite variable, both at presentation and over time, and this can play an influential role in the ultimate clinical outcome. As a result, a diagnosis of meningioma in a child carries with it unique challenges and treatment concerns.

Epidemiology

Pediatric meningiomas represent ~0.7 to 4.2% of all intracranial tumors in children.[1–8] A retrospective review by Drake and colleagues at the Hospital for Sick Children revealed only 13 patients with meningiomas out of 1283 cases of pediatric intracranial tumors (~1%).[9] Perhaps the largest single series of 2620 pediatric intracranial tumors, reported by Mendiratta et al in 1967, estimated an incidence of 1.5%.[6] Meningiomas have been reported throughout childhood, with a median age of ~13 to 15 years.[5,10,11] The appearance of meningioma in children is bimodal, with one peak in infancy[12] and one in the second decade of life.[1,3,9,13]

The gender distribution of pediatric meningiomas trends toward males, which is in contrast to the female predominance in the adult population. Ferrante et al report a male to female ratio of 1.3:1 in children.[14] There may be a particularly higher prevalence in males in the infant age range, as demonstrated by Sakaki et al.[12] In that review of 24 total infant cases, 17 children (71% of cases) were male. As children increase in age and as their hormonal profiles shift, so does the incidence of meningiomas in females.[15] The absence of sex hormones and their subsequent interaction with corticosteroid receptors on meningeal cells suggests that the development of pediatric meningiomas occurs as a result of unique pathogenetic processes in children versus adults.[16–19]

Clinical Presentation

Intracranial meningiomas in children most frequently come to the attention of parents, caregivers, and physicians as a result of increasing intracranial pressure (ICP) (**Table 8.1**).[1,10,12,20–23] The expansile mass and likely subsequent peritumoral edema result in headache (**Fig. 8.1**), nausea, and vomiting in approximately half of patients. Given the prevalence of intraventricular tumors in this age group,[3] rapidly progressive hydrocephalus can also lead to increased ICP. Liu and colleagues demonstrated the presence of papilledema in as many as 55% of patients.[24] The mass effect and direct irritation of the Rolandic cortex by tumor can cause seizures and motor deficits in ~12% and 13% of patients, respectively (**Table 8.1**). Visual deficits and cranial nerve palsies represent the next tier of symptoms, occurring in ~10% and 8%, respectively (**Table 8.1**). As will be discussed, pediatric meningiomas occur in a myriad of locations in the brain, distinct from the adult. As a result, the neurological symptoms and functional deficits related to these masses have considerable variability.

Meningiomas are associated with a variety of inherited syndromes, the most notable of which is neurofibromatosis type 2 (NF2). Meningiomas appear in ~25 to 40% of NF2 patients.[1,3,4,13,16,22,25] This intimate relationship should raise the suspicion of NF2 in a child with meningioma, especially in the setting of multiple meningiomas. Perry and colleagues documented a 100% incidence of NF2 in children presenting with multiple meningiomas, 36% of which were not known to have NF2 at the time of meningioma surgery.[13,25] The same group also described less in-

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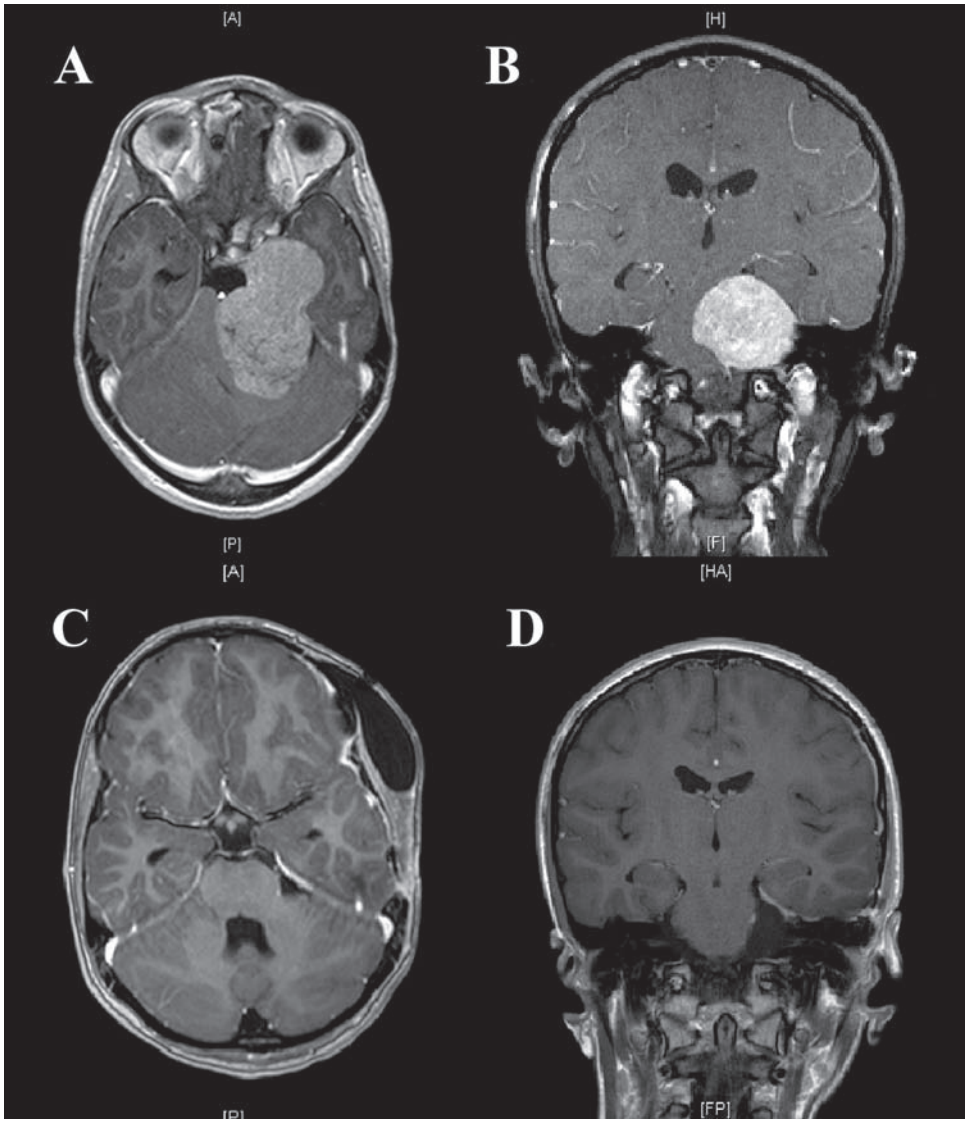


Fig. 8.1 Pre- and postoperative magnetic resonance images of a cerebellopontine angle meningioma in an 11-year-old girl. The patient presented with intermittent headache and progressive left-sided dyscoordination. **(A)** A large, homogeneously contrast-enhancing, dumbbell-shaped lesion in the left cerebellopontine angle with extension into the middle fossa was seen on T1-weighted axial and **(B)** coronal views. **(C)** The tumor was completely resected through an orbitozygomatic craniotomy as shown on postoperative axial and **(D)** coronal T1-weighted, contrast-enhanced scans. Pathology specimens revealed clear cell meningioma. Her postoperative examination was notable for transient left cranial four and six palsies, which steadily improved. Her preoperative dyscoordination also resolved completely.

vasiveness in NF2-associated meningiomas but no other histopathologic differences.

Children with a prior history of radiation exposure are also at a higher risk of developing meningiomas later in life. Widely accepted treatment for tinea capitis in the 1950s, radiation therapy, resulted in a fourfold increase in the risk of developing meningiomas.[26] The relative risk of developing meningiomas after ionizing radiation increases by approximately 10-fold when compared with controls.[14,25,27] Although the latency period for meningioma growth ranges from 11 to 43 years,[25] higher doses may cause these tumors to appear sooner.[28] Radiation-induced meningiomas are thought to present at earlier ages, are more likely to be multifocal, and occur in the field of radiation.[27,29] It remains to be determined, however, that meningiomas secondary to radiation carry higher pathological grades or exhibit more malignant behavior.[25]

Tumor Location

Pediatric meningiomas occur most frequently in the frontal and parietal convexities (**Table 8.2**), and the vast majority are supratentorial.[16,30] These tumors are more likely to occupy unusual locations in children when compared with older age groups. The ventricle is another common location for pediatric meningiomas, representing 9[4] to 15% (**Table 8.2**) of childhood cases but only 5% of adult cases.[31] Intraventricular meningiomas are thought to arise from choroid plexus or tela choroidea of the lateral ventricle.[32] Sylvian fissure meningiomas reported in children likely arise from arachnoid cap cells trapped within pia-arachnoid membranes deep within the cleft.[32–34] The posterior fossa has a reported involvement of up to 19% in children,[35] though the informal survey conducted in this chapter finds childhood incidence

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Table 8.1 Presenting Signs and Symptoms of Intracranial Meningioma in Children*

Symptom/Exam Finding	Number	Percentage
ICP, HA, N/V, lethargy	116	43.9
Seizure	34	12.9
Motor deficit	31	11.7
Decreased vision	25	9.5
Cranial nerve deficit	21	8.0
Ataxia	9	3.4
Exophthalmos/proptosis	8	3.0
Palpable mass	5	1.9
Diplopia	3	1.1
Behavior change	3	1.1
Ptosis	3	1.1
Head circumference	3	1.1
Vertigo	1	0.4
Dysphasia	1	0.4
Stillbirth	1	0.4

Abbreviations: ICP, intracranial pressure; HA, headache; N/V, nausea/vomiting.
*The accompanying data are tabulated from comparable case series detailing presenting symptoms and physical exam findings in a total of 264 pediatric patients with intracranial meningioma.[1,10,12,20–23]

Table 8.2 Localization of Intracranial Meningiomas in Children*

Location	Number	Percentage
Frontal/parietal convexity	180	29.4
Intraventricular	94	15.4
Parasagittal/parafalcine	55	9.0
Posterior fossa	44	7.2
Sphenoid	36	5.9
Sellar region	32	5.2
Frontal/middle fossa base	31	5.1
Multiple sites	30	4.9
Orbital	26	4.2
Tentorial	23	3.8
Petrous	18	2.9
Intraparenchymal	17	2.8
Foramen magnum	12	2.0
Epidural	3	0.5
Occipital/torcular	3	0.5
Sylvian	2	0.3
Sinus: sphenoid/ethmoid	2	0.3
Skull/intraosseus	2	0.3
Corpus callosum	1	0.3
Fontanelle	1	0.3

*The accompanying data are a representative summary of 612 cases compiled from series in which anatomic location is clearly specified.[1,3,5,7–10,12,14,16,20,21,23,24,29,35,37,45,56,63–65]

(~7%, **Table 8.2**) to be much closer to the adult incidence of 10%.[4,31]

Although reports in patients less than 1 year of age are scarce, the few that exist suggest that meningioma localization in infants may differ slightly from the remainder of the pediatric population. These tumors occur within the brain parenchyma, in an *intra*axial location, with greater frequency in this subgroup when compared with older children (~33% vs ~5%, respectively). Intraventricular meningiomas, which occur in up to 17% of presenting pediatric cases overall,[14] have been reported in ~3% in infant-specific surveys.[12,36,37] Posterior fossa meningiomas occur in ~19% of all pediatric cases[35] but rarely, if ever, in infants.[12]

Imaging Characteristics

Before the advent of modern computed tomography and magnetic resonance techniques, the neurosurgeon relied on skull radiographs as first-line imaging for pediatric patients harboring meningiomas. Approximately 70 to 75% of patients will demonstrate radiographic abnormalities such as tumor calcification, hyperostosis, bone destruction, pressure-induced skull thinning, and evidence of increased vascularity.[3,38] For historical purposes, the outdated techniques of pneumoencephalography and screening angiography were invaluable before computed tomography (CT) and magnetic resonance imaging (MRI) became commonplace in the workup of suspected meningioma.[38] Pneumoencephalograms showed abnormalities such as ventricular displacement and dilatation in up to 75% of pediatric patients. Angiograms in children showed such abnormalities as prolonged tumor blush, tumor vascular supply, and/or displaced vascular anatomy in up to 80% of patients.

The current standard for the radiographic evaluation of pediatric meningiomas involves a preliminary CT scan followed by a gadolinium-enhanced MRI.[39] Though not always present, the "classic" features of meningioma on CT are hyperdensity, diffuse enhancement, and intratumoral calcifications. With respect to MRI, meningiomas are characterized by hypointense signal on T1-weighted images, isointense signal on T2-weighted images, and homogeneous contrast enhancement on gadolinium-infused studies (**Fig. 8.2**). A margin of the meningioma may be associated with an area of thickened dura on CT or MRI, resembling an aptly named dural tail, though this finding is not exclusive to meningiomas and is shared with many other pathological processes.[40]

Pediatric meningiomas distinguish themselves radiologically from adult meningiomas in multiple ways. As mentioned previously, the distribution of tumor location is much more variable in children (**Table 8.2**). The dural tail has been well described for adult tumors, but the complete absence of a dural attachment occurs in as many as a third of pediatric cases.[9,12] Meningeal tumors in this age group are often cystic in greater than 12% of cases[32,41] compared with 2 to 4% of adult cases.[42,43] Cyst formation appears unrelated to histological type of pediatric meningioma.[41] Finally, meningiomas have a tendency to be larger in children at presentation.[3,32] This is thought to be a result of the resilient pediatric brain's tolerance

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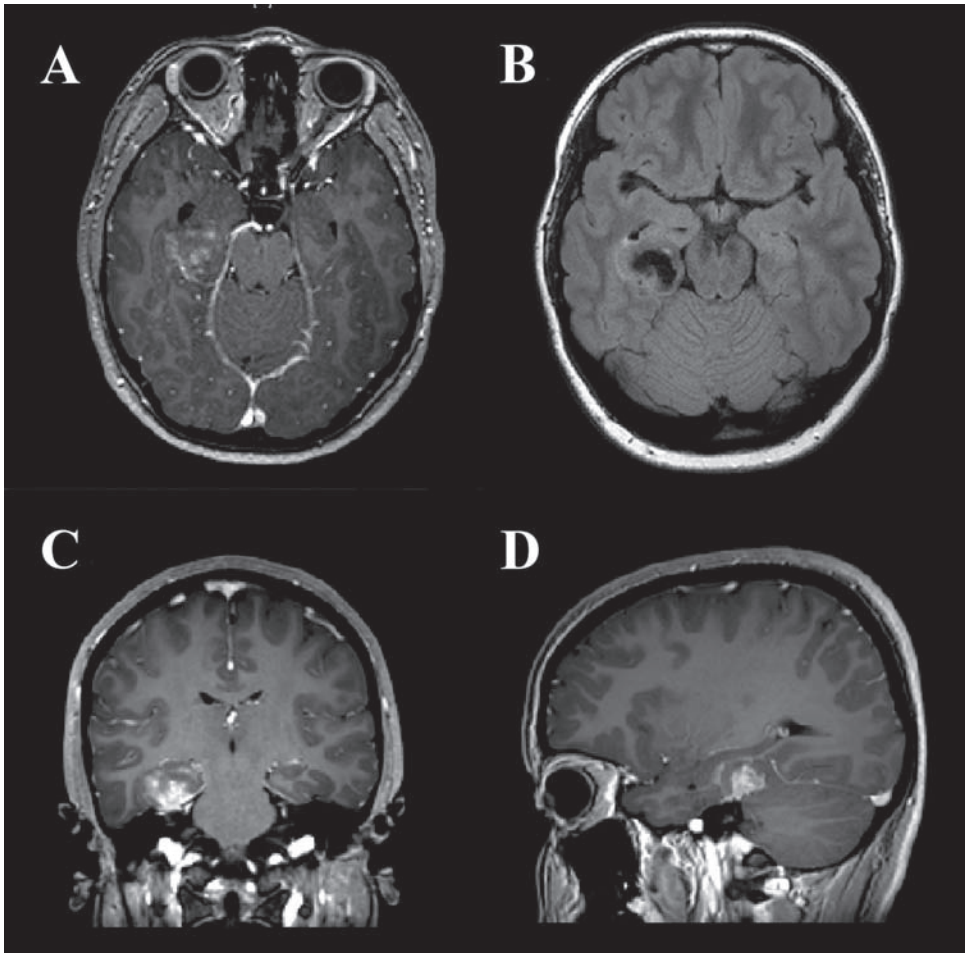


Fig. 8.2 Pre- and postoperative magnetic resonance images of a right mesial temporal meningioma in a 17-year-old girl. The patient experienced a first-time seizure at 14 years of age. **(A)** Her workup included imaging, which showed a right mesial temporal lesion with heterogeneous enhancement that appeared to be intraaxial on axial gadolinium-enhanced T1 and **(B)** fluid-attenuated inversion recovery (FLAIR) images as well as **(C)** gadolinium-enhanced coronal and **(D)** sagittal views. The lesion was resected through a right temporal craniotomy, and no residual tumor appeared on postoperative images. The posterior margins of the tumor appeared to blend with neighboring gyri, and no clear dural attachment was encountered. Pathology specimens were consistent with clear cell meningioma. The patient was neurologically intact on postoperative exam.

Table 8.3 Pathological Subclassification of Meningiomas in Children*

Meningioma Subtype	Number	Percentage
Meningotheliomatous/syncytial	178	31.0
Transitional/mixed	127	22.1
Fibrous/fibroblastic	118	20.5
Angiomatous/angioblastic	35	6.1
Psammomatous	32	5.6
Sarcomatous	27	4.7
Atypical	26	4.5
Papillary	12	2.1
Unidentified	10	1.7
Clear cell	3	0.5
Anaplastic	2	0.3
Malignant—unspecified	2	0.3
Lipoblastic	2	0.3
Chondroblastic	1	0.2

*The data were compiled from comparable pediatric meningioma series that specified tumor histopathology in 575 patients.[1,4,7–10,14,16,20–22,24,45,56,62,65]

of slow-growing lesions and the malleable, expandable skulls of younger children whose sutures have yet to fuse.

Pathology

The differential diagnosis for meningioma is broad (**Table 8.4**). Though virtually every meningioma subtype can occur in children, the most common type is meningotheliomatous (~31%), followed by transitional (~22%) and fibroblastic (~21%) (**Table 8.3**). Pediatric meningiomas are typically negative for glial fibrillary acidic protein and positive for vimentin on histologic analysis.[4] Early pathological analyses of pediatric meningiomas concluded that these tumors were more often malignant when compared with adult meningiomas. Many of these studies included meningeal sarcomas and other malignant tumors of meningeal origin, which are more common in children when compared with adults.[30,36,44–46] Papillary meningiomas, a subtype characterized by frequent mitotic figures and local invasion, are also seen more commonly in children and represent 10% of cases.[16,47] Inclusion of these

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Table 8.4 Differential Diagnosis for Meningioma of the Pediatric Brain and Spine

Tumor Class	Tumor Type
Meningioma	Benign meningioma variants (WHO grade I) • I: Meningothelial, fibrous, transitional, psammomatous, angiomatous, microcystic, secretory, lymphoplasmacyte-rich, metaplastic Aggressive variants (WHO grade II and III) • II: Clear cell, chordoid, atypical • III: Papillary, rhabdoid, anaplastic Meningiomatosis
Sarcomas/ "small blue cell tumors"	Hemangiopericytoma Fibrosarcoma/MFH Ewing sarcoma/pPNET Mesenchymal chondrosarcoma Rhabdomyosarcoma Postradiation sarcoma Leiomyosarcoma (EBV-associated) Medulloblastoma/cPNET Melanocytic neuroectodermal tumor of infancy
Benign mesenchymal	Chondroma Hemangioma Lipoma Solitary fibrous tumor
Hematopoietic/ histiocytic	Chloroma/granulocytic sarcoma (leukemia) Lymphoma Juvenile xanthogranuloma Langerhans cell histiocytosis Rosai-Dorfman disease Xanthoma/fibroxanthoma Histiocytic lymphoma/malignant histiocytosis Hemophagocytic lymphohistiocytosis Extramedullary hematopoiesis/myeloid metaplasia
Melanocytic	Meningeal (neurocutaneous) melanosis Melanoma Melanocytoma
Infectious/ inflammatory	Tuberculous meningitis/tuberculoma Inflammatory pseudotumor Inflammatory myofibroblastic tumor
Spread of CNS tumor	Medulloblastoma/cPNET Atypical teratoid/rhabdoid tumor (AT/RT) Ependymoma Diffuse astrocytoma/gliosarcoma Pilocytic astrocytoma Ganglioglioma/DIG Pleomorphic xanthoastrocytoma

Abbreviations: CNS, central nervous system; cPNET, central primitive neuroectodermal tumor; DIG, desmoplastic infantile ganglioglioma; EBV, Epstein-Barr virus; MFH, malignant fibrous histiocytoma; pPNET, peripheral primitive neuroectodermal tumor; WHO, World Health Organization.

Adapted from Perry et al, 2003.[25]

more malignant tumor types in pediatric meningioma series would portray this disease as more frequently higher grade. In a more recent clinicopathological analysis using revised World Health Organization (WHO) criteria to classify 87 pediatric meningiomas, Rushing and colleagues found that 71% of patients possessed a WHO grade I tumor, 24% a grade II, and 5% a grade III.[11] Current thinking is that pediatric meningiomas are not as malignant as once believed.

Treatment

The standard of care for pediatric meningiomas continues to be surgery with a goal of gross total resection.[48] This approach is based fundamentally on the principles set by meningioma treatment in the adult population.[49] Not surprisingly, degree of resection is often dictated by tumor location and adherence to neighboring neural and vascular structures.[3,7,16] Although the stereotypical dural tail may be absent, removal of the dural base and attachment is essential given the higher likelihood of recurrence should they remain.[50–52] As with all pediatric surgery, every effort should be made to minimize blood loss and length of surgery. Trained pediatric neuroanesthesiologists and pediatric neurointensivists are also invaluable assets in the perioperative period.

Radiation treatment for meningiomas has met with limited success. Its use has been restricted to partially resected, progressive meningiomas[29] and highly malignant tumors.[53] Radiation has been employed to lower tumor recurrence for partially resected pediatric meningiomas.[4,45] However, given that this form of treatment carries with it significant risks in children,[54] its use for benign lesions is difficult to justify.[3] In the end, surgery remains the preferred treatment strategy over radiotherapy for primary or recurrent disease.[3,29]

To date, chemotherapy has not proven useful for meningiomas.[48] Seven patients received chemotherapy in four of the larger published pediatric meningioma series.[3,11,23,29] No promising results emerged from these cases because most patients succumbed to complications from their disease, had limited postchemotherapy course descriptions, or were lost to follow-up.

Outcome

The earliest studies of pediatric meningiomas created a general consensus that these tumors behave more aggressively in children than in adults.[55,56] The explanation for this early opinion may be due to many factors.[57] First, before comprehensive CT and MRI became commonplace, diagnoses were likely delayed, allowing many meningiomas to reach considerable size.[15] Furthermore, earlier studies report surgical resection percentages that may reflect less advanced microsurgical and anesthesiological techniques. Many patients included in pediatric studies also carried a diagnosis of NF2, which is known to be a poor prognostic factor for progression of syndrome-related tumors.[58] Finally, earlier pediatric studies may have had more aggressive histologic variants included, such as papillary and anaplastic subtypes, without the benefit of current radiological and histological criteria to assist in pathological differentiation.

As expected, some of the earliest pediatric meningioma series described particularly grim results. Perhaps the earliest series reported by Bodian and Lawson in

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1953 described five children with 100% mortality within 10 years without surgery, indicating that intervention, in some form, would be warranted for these tumors.[36] In 1956, Odom and colleagues reported 3 children with meningeal tumors who received surgery, 2 of whom died within 3 years for a 66% mortality.[46] Twenty years later, Leibel and colleagues reported a mortality rate of 69% in a series of 13 children with meningiomas.[45] A long-term follow-up study conducted in Denmark spanning nearly 50 years showed an overall mortality of 65%, with a mean survival of 10 years.[15] Operative deaths and recurrence rates were high for these early series, no doubt a reflection of the limitations of neurosurgery and neuroanesthesia at the time. In a recent comprehensive survey of 440 cases reported in the literature, Caroli and colleagues highlighted diminishing reported operative mortality from 27% before 1970 to 0.3% since 1990.[57]

The issue of recurrence is a pertinent one in pediatric meningioma patients with respect to prognosis. Recurrence occurs in ~11% of cases,[14] with a mean time to recurrence of 7 years.[16] The importance of extent of meningioma resection in determining the likelihood of recurrence was clearly illustrated by Dr. Simpson in his seminal paper of 1957.[49] Accordingly, degree of resection and histopathological grading appear to be crucial determinants in predicting outcome for children with meningiomas.[3,8,14,16,53,56] Gross total removal for these tumors has been reported in the range of 54 to 86%.[3,4,14] Resection and thus survival are heavily influenced by tumor location as demonstrated by Deen et al.[16] Convexity meningiomas in this series had a recurrence rate of 32% and 20-year survival of 75%, whereas technically challenging skull base tumors recurred 77% of the time and had a 20-year survival of 32%. The association between WHO grade of meningioma and recurrence rate is not as strong in children when compared with adults, and the behavior of these pediatric tumors appears more difficult to predict over time.[25]

Tumor classification and pathological grading play a pivotal role in survival and outcome for children with meningiomas. When meningiomas are considered as a separate entity, apart from meningeal sarcomas and other malignant tumors of meningeal origin, outcomes are dramatically better. Drake and colleagues report good or excellent outcomes in 85% of children at an average of 6 years follow-up and 63% at 15 years.[9] In a study of 87 patients with childhood meningiomas, Rushing and colleagues concluded that recurrence-free survival is significantly related to WHO grade and that overall survival was not influenced by such factors as age, sex, extent of resection, history of radiotherapy, or presence of other comorbid conditions.[11] When looking strictly at WHO grade I and II meningiomas, the authors documented a 5-year survival rate of 98% and 100%, respectively. At 10 years, these numbers were 72% and 100%, respectively.

Spinal Meningiomas

Meningiomas of the spine are truly rare in children, representing just 2 to 8% of all pediatric meningiomas.[11,35,56] Unusual locations, en plaque formation, and multiplicity have all been reported for these tumors.[59–61] As many as 15% of spinal meningiomas may actually occupy the *extra*dural space, with the root exit zone arachnoid villi serving as the likely tumor source.[16] They often present first with pain, then with focal neurological deficits.[29] Clear cell meningioma, a rare but more aggressive meningioma subtype, may have a predilection for the pediatric spine. In a report of 18 clear cell meningiomas appearing in children, 11 cases (61%) occurred in the spine, and all but one of them involved the lumbar spine.[62] Total resection was achieved in all 11 cases, but recurrence occurred in eight cases (73%), highlighting the need for frequent follow-up imaging for this aggressive tumor type.

Spinal meningiomas are best treated with surgery. This is attributed to the relatively small tumor size at presentation and locations that are favorable for surgical resection. Lund-Johansen et al report one recurrence out of six spinal meningiomas that had undergone gross total resection (17%).[63] Deen et al report lower recurrence rates for meningiomas of the spine than for those of the intracranial space—20% versus 39%, respectively.[16] Overall, the prognosis for isolated spinal meningiomas that are surgically resected is therefore quite good.

Conclusion

Though rare, the pediatric meningioma is a unique clinical entity. From a surgical standpoint, these tumors are often found in predictable locations, are easily approachable, and carry a favorable prognosis in adults. This is not always the case in children. Pediatric tumors are often relatively larger, more cystic, and in unusual locations. Their pathological classification and resultant prognosis are somewhat controversial. In reality, they may have decidedly better outcomes than previously thought. Regardless of the debates that remain, the neurosurgeon's role and purpose remain clear: Complete resection and careful surveillance for recurrence of this potentially curable disease.

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Chapter 9

Meningiomas in the Elderly

M. Necmettin Pamir and Koray Özduman

Introduction

In the last few decades, a change in patient populations has occurred. With advancing health care technology and improvements in public health, human societies are aging. It is estimated that in 2020 a fourth of the European population will be in the elderly age range. In the United States one of five persons will be 65 years of age or older by the year 2050, and this population can expect a mean of 17 additional years of life once the age of 65 years is reached.[1]

The incidence of meningiomas increases with age. Combined with a greater ease of diagnosis, with widely and readily available computed tomography (CT) and magnetic resonance imaging (MRI), this aging patient population is having incidental meningiomas identified more than at any time before. With improvements in neuroanesthesia, neurosurgical technique, intensive care, radiosurgery, and modern radiotherapy planning, treatment of these meningiomas is safer than ever and much more easily tolerated today. All these changes have led neurosurgeons to reconsider the questions of when to treat, whom to treat, how to treat, and what to expect when dealing with a meningioma in an elderly patient.

Several studies have been published on meningiomas in the elderly, and several thousand cases have been reported so far in the literature. However, there are still no clear clinical guidelines for everyday practice.

Initial publications reported dismal outcomes for elderly patients after meningioma surgery.[2,3] However, a review of the recent literature indicates that this risk is not as pronounced as it was (**Table 9.1**). More careful attention is being paid to subgroup analysis among elderly meningioma patients to identify those patients likely to benefit most from surgical intervention and conversely those at highest surgical risk.

Definition of the Elderly Population

Although age greater than or equal to 65 years is widely accepted as the standard definition of the elderly population, there is no specific definition of an elderly meningioma patient. Studies have taken 60, 65, 70, 80, or 90 years as their point of dichotomization, which complicates a simple comparison among them (**Table 9.1**). One of the earliest studies on the subject was published by Papo.[3] The authors grouped their patients into those younger than 60 years, 61 to 65 years, 66 to 70 years, and older than 70 years. Their analysis indicated that the chance of a good outcome decreased from 75% in those patients less than 60 years of age to 71%, 41%, and 13% in the subgroups of incrementally increasing age. The percentage of meningioma patients over 60 years of age rose in that study from 26% to 43% and that of patients over 70 years from 5% to 12% after CT became available.

The Incidence of Meningiomas in the Elderly Population

Along with malignant gliomas, meningiomas are the most common intracranial tumor encountered in the elderly population.[4] Meningiomas in the elderly may be symptomatic or they may be found incidentally. Hospital-based studies indicate that the peak of meningioma incidence is in the sixth to seventh decades.[5] This may be biased by the fact that meningiomas may more commonly remain asymptomatic in the elderly. Both the incidence and the prevalence of meningiomas steadily increase with age to reach a peak at the eighth decade.[6–11] Population-based studies give a clearer picture on the incidence of intracranial tumors when compared with reports form surgical centers.

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Table 9.1 Major Studies on Elderly Patients with Meningioma

Study	*n*	Female Gender (%)	Convexity versus Complex Location (%)	ASA I–II–III–IV	Age	Gross-Total Resection Rate (%)	30-Day Mortality Rate (%)	3-Month Mortality Rate (%)	Morbidity (%)	Satisfactory Outcome (%)	Factors Influencing Outcome
MacCarty and Taylor, 1979[2]	51	N/A	N/A	N/A	70–79	N/A	11.8	N/A	N/A	N/A	—
Papo, 1983[3]	54	N/A	N/A	N/A	>60	N/A	39	N/A	N/A	48	Poor results in patients over 65 years of age
Djindjian et al, 1988[41]	30	N/A	43–57	N/A	>70	N/A	23	37	N/A	N/A	Poor results in patients with poor KPS No difference in outcome between the seventh and eighth decades
Awad et al, 1989[40]	75	72	37–63	N/A	>60	N/A	8	15	52	44	Poor results in skull base tumors and patients with severe preoperative neurological deficits
Arienta et al, 1990[25]	34	67	27–73	N/A	>70	74	12	20	40	77	Poor results in patients with poor preoperative clinical status, in those with marked peritumoral edema, in those with diabetes mellitus, and after long durations of surgery
Cornu et al, 1990[27]	96	62.5	35–65	21–48–27	>65	N/A	16	23	43	63	Poor results in skull base tumors and patients with poor general health status, and patients with severe preoperative neurological deficits
Umansky et al, 1992[35]	37	54	27–73	N/A	>70	76	N/A	5.4	37	N/A	—
Maurice-Williams and Kitchen, 1992[28]	46	N/A	N/A	N/A	>65	N/A	2.7	N/A	30	89	No difference of morbidity between middle aged and the elderly
McGrail and Ojemann, 1994[30]	56	N/A	N/A	N/A	>70	70	3.6	N/A	11.3	95	Higher risk in posterior fossa meningiomas
Nishizaki et al, 1994[34]	78	N/A	N/A	N/A	>70	73	13	N/A	N/A	95	Poor results in patients with severe neurological deficits,* recurrent cases,* and cases of histologically proven malignancy*
Mastronardi et al, 1995[42]	17	77	59–41	2–11–4-0	>80	77	29	29	11.8	N/A	Poor results in patients with large tumors,* marked peritumoral edema, and low preoperative Karnofsky performance score

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Proust et al,1997[43]	39	N/A	N/A	N/A	>70	N/A	N/A	N/A	N/A	N/A	Poor results if ASA III, KPS < 70, tumor > 5 cm diameter
Lieu et al, 1998[33]	36	75	36–64	N/A	>65	N/A	N/A	N/A	N/A	63.9	Poor results if ASA III and with marked preoperative neurological deficits
Black et al, 1998[29]	57	72	N/A	21–23–8-5	>65	N/A	1.8	1.8	7	86	—
Buhl et al, 2000[26]	66	65	24–76	N/A	>70	N/A	7.6	-	18.2	74.2	Poor results in recurrent tumors, after long operation durations
Tucha et al, 2001[36]	33	64	36–64	N/A	>70	N/A	N/A	N/A	N/A	N/A	—
Bateman et al, 2005[12]	2304	67	N/A	N/A	>70	N/A	4	N/A	N/A	46.8	Poor results in patients with advanced age, nonroutine admission, and high comorbidity index score, and in those operated in a low patient volume hospital
D'Andrea et al, 2005[32]	37	78	62–38	11–19–7-0	>80	81.1	N/A	13.5	N/A	86.5	Poor results in patients with high preoperative ASA score, low KPS index, in patients with tumors located at the skull base and posterior fossa
Caroli et al, 2005[31]	90	67	44–56	84.4	>70	73.3	6.7	7.8	N/A	84.4	Worse prognosis in the female gender and in patients with concomitant disease and marked peritumoral edema
Sacko et al, 2007[44]	74	64	58–42	0–22–44–8	>80	82.4	0	1.4	9.4	N/A	Higher mortality in men in the first year, in those with higher ASA grades, lower KPS scores, critical location, those with marked peritumoral edema; higher morbidity with radical removal
Patil et al, 2009[37]	258	5.3	N/A	0–9.3–68.2–22.1–0.4	>70*	N/A	12	N/A	29.8	N/A	—
Cohen-Inbar et al, 2010[45]	250	61	45–55	N/A	>65	49.9	8.4	N/A	N/A	N/A	—
Total	**3818**										

* Statistically significant
** Multicenter, Veterans Administration (VA) hospital population

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Kuratsu et al[9] reported 504 meningioma cases diagnosed in the Kumamoto prefecture in Japan; 38.9% of the cases were incidental meningiomas and 31.7% of all meningiomas were found in patients older than 70 years. Elderly patients made up 26.2% of all symptomatic individuals and 40.3% of individuals with incidental meningiomas. Bateman et al[12] analyzed 8861 patients from the U.S. Nationwide Inpatient Sample Database who underwent surgery for intracranial meningioma and found that patients older than 70 years constituted 26% of this population.

The incidence of asymptomatic, incidental meningiomas in the general population ranges from 1 to 1.4% in noninvasive imaging studies in the general population[11,13] and in autopsy series.[13–15] Rengachary and Suskind[16] report an incidence of 4.6% for intracranial meningiomas incidentally found at autopsy in subjects older than 80 years. Similarly, the incidence of meningiomas that come to clinical attention is reported to be 3.5 times higher in patients over the age of 70.[5] The prevalence in cases over 60 years of age is 3%.[14] Vernooij et al[11] have performed MRI on 2000 subjects in a population-based study and found that the incidence of meningiomas at 45 to 59 years was 0.5%, at 60 to 74 years 1%, and at 75 to 97 years 1.6%. Longer life expectancy and increased availability of diagnostic imaging have certainly contributed to a more common diagnosis of meningioma in the elderly.

Clinical Behavior of Meningiomas in the Elderly

The treatment decision in an asymptomatic elderly patient with a meningioma is tougher than in a young patient. The likelihood of an incidental meningioma remaining asymptomatic increases with advancing age.[7] Most meningiomas remain asymptomatic throughout life; in fact, half of all meningiomas are discovered at autopsy.[15] Studies that have analyzed the clinical behavior in asymptomatic meningiomas have reported growth rates ranging from 0 to 37.3%.[17–23] This indicates that, in the short term, at least two thirds of meningiomas do not grow. There are very few studies on the natural history of incidental meningiomas in the elderly. Among 504 meningiomas reported by Kuratsu et al,[9] the percentage of asymptomatic cases was higher in the elderly population (49% vs 34%). A study by Niiro et al[20] showed that 35% of incidental meningiomas grew on follow-up at a mean of 32.1 months. This suggests that the incidence of growth is similar to that of the general population; however, the patient sample is rather small. The authors also indicated that calcified and small tumors were significantly less likely to grow. There are, however, no long-term studies that have evaluated the incidence of growth in asymptomatic meningiomas in the elderly.

Outcome of Meningioma Surgery in the Elderly

Maintaining life span and quality of life, while providing long-term relief/prevention from intracranial tumor growth–associated problems, is the fundamental aim of meningioma surgery in elderly people.[24] Very little is known about the effects of neurosurgery on the elderly; however, studies reported so far indicate a higher incidence of surgical morbidity in this age group. Most commonly reported surgical complications were postoperative hematoma, cerebrospinal fluid (CSF) fistulas, infection, seizures, motor deficits, and hydrocephalus. Several studies have reported fairly high incidences of postoperative hematomas, ranging from 15 to 25%.[25–28] Whether this is a coincidence or related to an underlying hemorrhagic tendency in the elderly population is not known. The incidence of medical comorbidities is higher in the elderly population, and the most commonly reported postoperative complications were atrial fibrillation, acute myocardial infarction, pneumonia, deep vein thrombosis (DVT), and pulmonary embolism.[29]

Some four thousand cases of meningioma surgery in the elderly have been reported in the literature to date (**Table 9.1**). Some studies have indicated that the outcome of meningioma surgery in the elderly is no different than that in the younger population.[28–30] Other studies have concluded that the outcome of meningioma surgery in the elderly is less satisfactory and the complication rate is higher than in the younger patients. However, no studies have indicated that advanced age is a contraindication for surgery. A satisfactory result with good neurological recovery or stabilization has been reported in 63.9 to 95%.[26,29–34] Umansky et al[35] reported that the mean Karnofsky Performance Score improved from 59 to 80 after meningioma surgery in the elderly. Tucha et al[36] also showed that cognition (including attention functions, memory, and concept formation) was improved in elderly meningioma patients after surgery. The authors showed that the cognitive functions of elderly patients reached the level of healthy, age-adjusted controls except for verbal and figural working memory, which were also disturbed preoperatively in meningioma patients.[36]

Several studies analyzing cohorts containing both old and young patients have provided outcome data on meningioma surgery in the elderly in comparison with the younger population. Yano et al[22] reported their experience with a large cohort of meningioma patients consisting of 603 asymptomatic and 831 symptomatic meningiomas. When 213 surgically treated asymptomatic meningiomas were considered, a perioperative morbidity of 4.4% was observed in patients younger than 70 years and 9.1% in patients older than 70 years. Bateman et al[12] have analyzed the surgical outcome after intracranial meningioma surgery in 8861 patients from the U.S. Nationwide Inpatient Sample Database. Twenty-six percent of these patients were 70 years and older. The authors indicated an almost fourfold higher in-house mortality (1.1% vs 4%) in patients older than 70 years when compared with the younger population. The authors have also shown that this difference in mortality became more pronounced with advancing age. The in-house mortality figures for patients in their fifth, sixth, and seventh decades and for those older than 80 years were 1.4%, 1.2%, 3%, and 7%, respectively. Similarly, this study showed that the incidence

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of an adverse outcome (death or discharge to a facility other than home) was 16.6% for patients younger than 70 years; 53.2% for those older than 70 years, and 67.9% for those older than 80 years. All of these differences in morbidity and mortality were statistically significant, indicating that the risk of surgery in the elderly is higher and that it increases with increasing age. A more recent study analyzed the outcome of meningioma surgery in 258 patients operated in 123 Veterans Administration (VA) hospitals over a 9-year period.[37] The study concluded that the 30-day mortality was significantly higher (12% vs 4.6%) in meningioma patients over 70 years. Similarly, the study showed that the surgical morbidity was also higher (29.8% vs 13.1%) in patients over 79 years.

Factors Influencing the Outcome

No study so far has clearly indicated a single causative factor for the poorer outcome after meningioma surgery in the elderly. When individual risk factors are analyzed, most studies concluded that increasing age, poor preoperative general health status, severe neurological deficits, large tumor size, critical location, and marked peritumoral edema were indicators of poor outcome.

Advanced age is not a contraindication for neurosurgery.[38] In a recent study on 289 elderly patients (> 70 years) with brain tumors (astrocytoma, meningioma, metastasis, and pituitary adenoma), Rogne et al[39] found that more than 85% of the patients were alive after 6 months and had stable or improved performance scores. The authors found that gender, age, and American Society of Anesthesiologists (ASA) score were not related to the survival. Most studies on meningiomas, however, have come to the conclusion that the mortality and morbidity of meningioma surgery increase with advancing age.[12,40] Analyzing the outcome in a fairly large nationwide patient database, Bateman et al[12] compared the surgical outcome data after meningioma surgery in the elderly to other surgical procedures such as sigmoidectomy, hemicolectomy, and open cholecystectomy in the same age group and concluded that a worsening outcome with age is not specific to the underlying pathology but rather relates to the increased risk of any surgery in this patient population.

It is not known how much the surgical outcome is affected by the process of aging in the central nervous system. Atherosclerosis, amyloid angiopathy, and leukoaraiosis as well as an impairment of cerebrovascular autoregulation may all contribute to difficulty during surgery and adverse outcome thereafter.

Studies of elderly meningioma patients also note that ASA classes of 3 and 4 and Karnofsyky Performance Score (KPS) of 60 or less were associated with higher mortality and morbidity. In patients with good functional status and no associated systemic morbidity, surgery is a safe and effective treatment. In their multivariate analysis of 8861 intracranial meningioma patients, Bateman et al found that a higher case load of the hospital correlated with significantly lower incidence of adverse outcome.[12] Female sex was also associated with poor outcome in some studies.[31]

Summary

Roughly one third of all meningiomas are diagnosed in the elderly. The prevalence of meningiomas increases with age to reach its peak in the ninth decade. The likelihood of an incidental meningioma remaining asymptomatic increases with age. Advanced age is not a contraindication for meningioma surgery, and patients benefit from successful surgery. Surgical risks, however, are higher, and the outcome is worse in elderly meningioma patients when compared with the younger population. Advanced age, preoperative health status, and the presence of neurological deficits are the most commonly reported parameters affecting surgical outcome. The treatment decision is harder to make for an elderly patient with an incidental meningioma, when compared to a younger individual with a symptomatic meningioma. Treatment must be tailored for each patient and the balance between the risks and benefits of surgery evaluated in light of the tumor's biology, degree of mass effect, symptom profile, and patient preference. It must be kept in mind that the surgical outcome will be worse when an untreated meningioma presents later in life with more significant symptoms.

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Chapter 10

Radiation-Induced and Multiple Meningiomas

Ian F. Dunn and Ossama Al-Mefty

Introduction

Meningiomas are largely benign sporadic tumors occurring as solitary lesions whose management complexity is usually provided by the troublesome locations of their arachnoidal origins throughout the cranial vault and by their capacity to recur if a Simpson grade I resection is not achieved. More rarely, meningiomas may arise as multiple distinct or contiguous tumors. In some cases, multiple tumors arise in the context of prior cranial irradiation, begetting "radiation-induced meningiomas" (RIMs). In other cases, multiple tumors may arise in the setting of neurofibromatosis 2 (NF2) or, as with their solitary counterparts, as sporadic cases of multiple tumors with no clear inheritance pattern. Multiplicity of tumors is a shared feature of patients with radiation-induced tumors and nonirradiated patients harboring multiple meningiomas as part of, or distinct from, NF2.

Radiation-Induced Meningiomas

Accumulating evidence over the last 50 years has convincingly linked radiation exposure to the development of cancer in humans. Indeed, radiation-induced meningiomas are the most common form of such neoplasms reported in the literature,[1] and specific criteria have been developed to appropriately define them (**Table 10.1**).[2] Large cohorts of patients treated with cranial irradiation or who survived the catastrophic atomic bombings of the last world war have, in particular, provided devastating evidence of the role of radiation in meningioma tumorigenesis. In contemporary neurosurgery, the expanding role of therapeutic stereotactic radiation, while greatly reducing the exposure of nontarget tissues to radiation, provides an appropriate context to review the concept of meningiomas wrought by cranial irradiation.

Biology and Pathology

Tumors arise from the accumulation of mutations in genes that program the neoplastic phenotype. Indeed, nearly all tumors show evidence of activation of mutant versions of protooncogenes and the inactivation of tumor suppressor genes. Ionizing radiation is capable of inciting the diverse genetic events that occur in tumor formation through direct mutagenesis of participating oncogenes and tumor suppressors; widespread genomic instability, which is perpetuated in surviving dividing cells; and the direct compromise of DNA repair mechanisms.[3] Recent genetic studies have illuminated the karyotypic chaos found in radiation-induced tumors, in stark contrast to the majority of nonradiation-induced meningiomas, whose hallmark is loss of part or all of chromosome 22 (harboring the *NF2* gene). These studies have reported several genetic alterations, including loss of genetic material on chromosome 1, 6, and 22, among others[2,4,5]; selected data from our center are shown in **Table 10.2**.[2] In particular, structural abnormalities of chromosomes 1 and 6 may portend more aggressive meningioma behavior.[6] Interestingly, only 56% of cases in our series showed loss or deletion of chromosome 22, an abnormality characteristic of nonradiation-induced meningiomas.

Although the specific genetic events underpinning the genesis of RIMs have not been fully clarified, it is likely that this markedly aberrant genetic landscape governs the abnormally aggressive behavior of these tumors when compared with their nonradiation-induced counterparts: they tend to possess atypical histological features (**Fig. 10.1**), display more rapid growth, and show higher rates of multiplicity and recurrence.[2]

Dose Effect

The risk of developing solid cancers varies directly with lifetime exposure to radiation. Historically, RIMs have

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Table 10.1 Criteria for Inclusion as Radiation-Induced Meningioma[2]

1. Tumor must arise in the irradiated field
2. Histological features must differ from those of any previous neoplasm
3. A sufficient latency or induction period following radiation must elapse before meningioma is diagnosed (usually > 5 years)
4. No family history of phakomatosis
5. Tumor must not be recurrent or metastatic
6. Tumor must not be present before radiation therapy

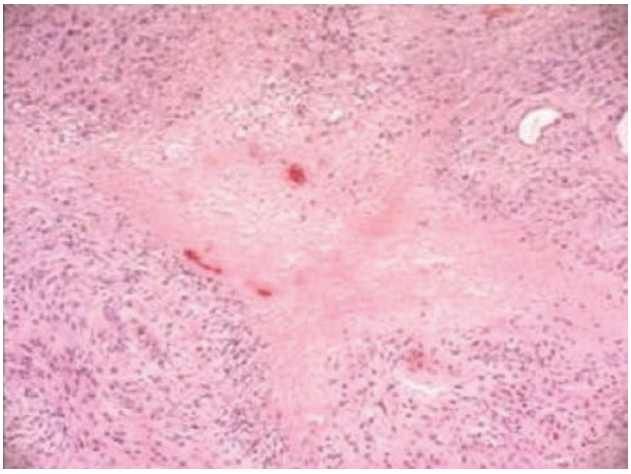


Fig. 10.1 Radiation-induced meningioma, low-power field, displaying necrosis, vascular proliferation, and nuclear atypia.

Table 10.2 Cytogenetic Alterations in Selected Cases of Radiation-Induced Meningiomas[2]

Case No.		Pathology	Chromosomal Analysis			
			Chromosome 1	Chromosome 6	Chromosome 22	Other
1		Atypical	der(1)inv(1)(p21;p36)	der(6)t(6;7)(p23;q23)		7
2		Meningothelial	der(1)t(1;3)(p11;p21)			3;4;10;19;21
5	First operation	Meningothelial	der(1)t(1:14)(p32;q11)	der(6)t(4:6)(q12;q13)	del(22)(q12), -22	4;5;10;11;12;14
	Second operation	Meningothelial	add(1)(p22)	der(6)t(1:6)(p21;q11)		13;14;16
	Third operation	Meningothelial	der(1)t(1:14)(p32;q11)	der(6)t(4:6)(q12;q13); del(6)(q23)	del(22)(q12)	5;9;10;11;12;14;19
10		Transitional				5;11;14;18
11		Meningothelial	-1	-6	-22	7;8
12		Transitional	der(1)t(1:20)(p21;q11)		-22	7
13		Meningothelial	der(1)t(1:12)(p13;p11)	del(6)(q23)	-22	2;13

Abbreviations: del, deletion; der, derivative.

been categorized by the level of prior radiation given to patients in whom these tumors later arise.[7] Low dose is defined as doses of less than 10 Gy, intermediate refers to doses between 10 and 20 Gy, and high dose is characterized by doses of greater than 20 Gy. Overall, tumor formation appears to be accelerated by radiation exposure early in life and higher dosages of radiation, and dose of radiation appears to correlate with earlier development of tumors in patients. Because meningiomas may be incited by even low doses of radiation, several authors have presaged an inevitable increase in the incidence of RIMs induced at any dosage in the setting of expanding use of radiotherapy and diagnostic radiological studies.[1,8–11]

High Dose

The overwhelming majority of patients who develop high-dose RIMs have undergone cranial irradiation early in life, with reported doses ranging from 22 to 87 Gy.[12] The average latency from radiotherapy to meningioma diagnosis is ~19 years,[8,9] which is consistent with our series in which the latency period in high-dose RIMs was 24.6 years.[2] More recently, a multicenter case-control study as part of the Childhood Cancer Survivor Study investigated the development of new primary brain tumors in more than 14,000 survivors of cancer in childhood.[13] Meningiomas were diagnosed with a median latency of

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17 years after radiation, exposure to which was the most important risk factor for meningioma development in these patients (odds ratio [OR] = 9.94; 95% confidence interval [CI]: 2.17 to 45.6).

Low and Intermediate Dose

Meningiomas also occur in patients exposed to doses of radiation less than 20 Gy. The development of meningiomas in patients who had undergone scalp irradiation for tinea capitis in the first half of the twentieth century provided the first large-scale evidence of radiation-induced meningioma. The treatment paradigm, known as the Adamson-Kienbock method, involved radiating the entire scalp, with the surface receiving 5 to 8 Gy and the skull base ~0.7 Gy. In their landmark paper, Modan et al reported an increased risk of meningioma in more than 10,000 adults who had undergone scalp irradiation for tinea capitis as children.[14] More recent follow-up studies have reported that a mean of 1.5 Gy conferred a 10-fold increased risk of meningioma development,[15] with risk correlated with dose and rising to 18.8-fold when exposure was greater than 2.6 Gy.[16] Multiple meningiomas were also more common in patients who had undergone prior irradation.[17]

Compelling evidence demonstrating the tumor-inducing effects of low-dose radiation has also been provided by large studies of survivors of the atomic bombings of Hiroshima and Nagasaki. Beginning in 1994, an increasing number of reports have documented an increased incidence of meningiomas among survivors from both catastrophic events, with incidence correlated with distance from bomb epicenter and age at time of exposure.[18–20] A more recent analysis of a cohort of more than 85,000 survivors over the two atomic bombings in the Life Span Study[21] reports that RIMs in this cohort more closely resemble the epidemiological characteristics of "spontaneous" meningiomas.

Diagnostic irradiation has historically been linked to meningioma development, with Preston-Martin and White reporting a higher incidence of meningioma in patients who had undergone full-mouth dental x-rays.[22] Current applicability of these data are unclear because dosages administered today are drastically lower.[23] The use of computed tomographic (CT) scanning has escalated dramatically in the last decade, and, although the radiation dose per scan is low, their common use has caused significant concern for their potential roles in delayed carcinogenesis.[24]

Other contemporary uses of radiation in the low- and intermediate-dose range include stereotactic radiosurgery. Given the long latency periods between cranial irradiation and meningioma formation as already outlined, studies reporting no increased risk of tumor formation after radiosurgery with mean follow-up periods of 6 years[25] may be ill equipped to address the phenomenon of radiosurgery-induced meningioma. Isolated reports describing the development of meningiomas in patients after radiosurgery exist, but a true incidence of this clinical entity will only be ascertained through careful long-term radiologic follow-up.[26]

We have observed an average latency period of 30 years from time of irradiation to tumor detection, which is largely consistent with other published reports of latency after low- to moderate-dose radiation.[2]

Cellular Phones

The explosive worldwide rise in cellular phone use has focused interest on the tumorigenic properties of the range of electromagnetic radiation in which mobile phones operate. Although the associated radiation dose is considered "nonionizing" and hence safe, studies with long-term follow-up (> 10 years) have noted a trend to increasing risk of meningioma development on the ipsilateral side of phone use[27] as encapsulated in a recent meta-analysis (OR = 1.3, 95% CI: 0.9 to 1.8),[28] although other studies have not supported these data.[29] Given the latency periods observed in cases of ionizing radiation–induced meningiomas, it is clear that much longer follow-up is required to accurately assess the effects of mobile use on meningiomagenesis.

Unique Clinical Features of Radiation-Induced Meningiomas

By definition, RIMs occur in an irradiated field, and they appear as multiple lesions with greater frequency than sporadic or nonradiation-induced meningiomas. In our series, we documented a 31% incidence of multiple lesions—35.7% in patients who received high-dose radiation—compared with reported rates of multiplicity of 0.58 to 2.8% in patients with no prior history of cranial radiation.[2]

As might be inferred from the higher rate of anaplasia and malignancy, radiation-induced meningiomas are more aggressive tumors than their nonirradiated counterparts. Patients harboring radiation-induced tumors are younger, and their tumors bear markedly higher rates of atypical and anaplastic grades.[2,12]

Scalp atrophy and alopecia are hallmark clinical feature of patients who have undergone cranial irradiation and are always a significant consideration during the surgical management of RIMs.

Management of Radiation-Induced Meningioma

Surgical resection remains the treatment of choice in most cases of meningioma, including radiation-induced lesions. However, their inherently aggressive nature and the frequently tenuous soft tissue in irradiated patients renders surgical resection of these tumors particularly formidable.

Given the greater propensity of radiation-induced meningioma to recur, an exceptionally wide bone and dural margin should be resected where feasible.[7] In skull base tumors in which wide margins cannot be obtained, one must unfortunately accept a greater recurrence risk (**Fig. 10.2**).

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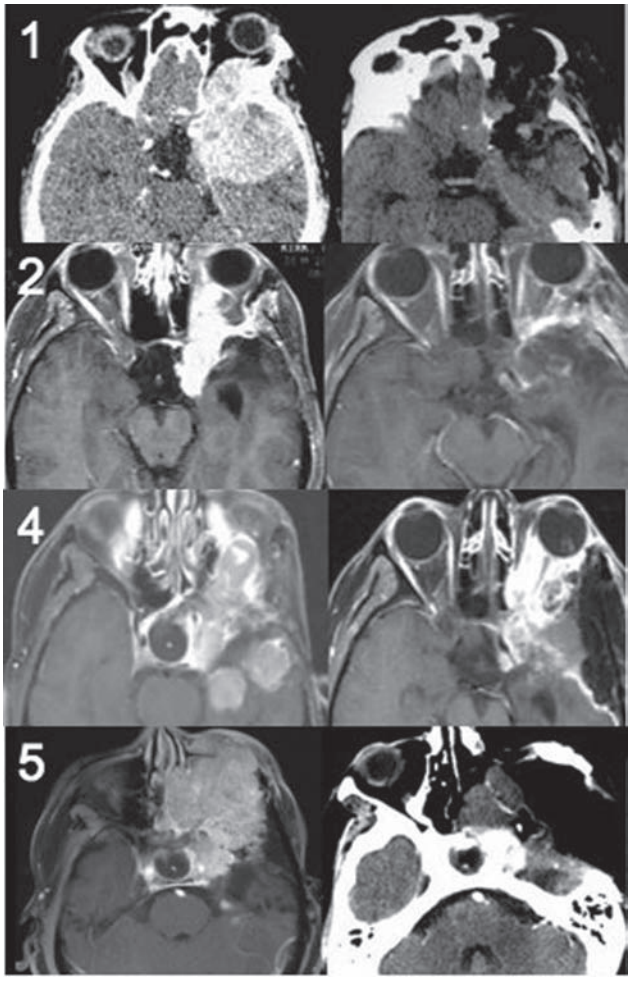


Fig. 10.2 Radiation-induced meningioma in a patient who underwent five resections, illustrating the aggressive clinical behavior of these tumors. Numerals denote side-by-side pre- and postoperative magnetic resonance imaging (MRI) associated with four of the five resections. Of note, the patient underwent gamma knife and interferon treatment before the fourth resection.

The scalp and underlying soft tissue must be handled with extreme care in patients who have received prior irradiation; wound infection and cerebrospinal fluid (CSF) leaks are of great concern and mandate extreme vigilance during closure. One should limit the use of monopolar or bipolar coagulation or skin clamps given the often fragile nature of the scalp in these patients. In select cases of anticipated poor wound healing or scalp atrophy, a plastic surgeon should assist in wound management.

In the medically infirm patient or in other situations in which surgical treatment is contraindicated, fractionated irradiation or radiosurgery may be considered,[30] although the maximum tolerable dose of radiation may already have been reached in some patients with radiation-induced meningioma. There is no viable chemotherapeutic option with proven efficacy for patients harboring radiation-induced meningiomas.

Follow-Up

Radiation-induced tumors recur more frequently, and vigilant serial imaging of these patients is mandated. Tumor recurrence rates of up to 25.6% have been reported,[31] as compared with recurrence rates of up to 11.4% in nonradiation-induced meningioma controls. The rate of recurrence is also more rapid, with RIMs recurring ~4 years earlier than in nonirradiated patients with recurrent meningiomas.[32] In our series of mainly skull base tumors, there were second recurrences in 62% of patients, and a third recurrence in 17% of patients.[2]

Multiple Meningiomas: Familial and Sporadic

As Cushing and Eisenhardt observed, some patients harbor "more than one meningioma and less than a diffusion" of them.[33] Nearly a century after Cushing and Eisenhardt's observation, their oft-quoted description still captures the continued ambiguity surrounding cases of multiple meningioma not associated with NF2.

Historically, 1 to 5% of patients with no history of cranial irradiation present with more than one meningioma, though this figure has risen to 10% and 20% with increased use of CT and magnetic resonance imaging (MRI), respectively.[34,35] This condition is most commonly associated with the autosomal dominantly inherited phakomatosis NF2; however, patients without NF2 may also develop multiple meningiomas (**Fig. 10.3**). This may occur sporadically or in an inherited fashion.

Whether or not multiplicity of meningiomas outside the context of NF2—in either a sporadic or an inherited fashion—represents a particular clinical syndrome is unclear. The alternative is that multiple meningiomas represent a *forme fruste* of NF2, although preliminary genetic investigations suggest otherwise.

Also to be elucidated is where a contiguous, or "diffuse," meningioma pattern lies in the spectrum of multiple meningiomas. At hand is whether a carpeting of the arachnoid with tumor, referred to by some as meningiomatosis, represents a coalescence of multiple tumors or is a unique pathological entity supported by some authors' contention that multiple meningiomas represent a subarachnoid dissemination of meningioma cells.[36,37] Although *meningiomatosis* is sometimes used to encompass the clinical condition of multiple meningiomas, it should perhaps be used more discriminately because it more commonly describes the meningeal involvement by sarcoma or other diffuse conditions where no dominant mass is identified.[38]

The following sections discuss the inherited and sporadic contexts in which multiple meningiomas arise.

Inherited Forms of Multiple Meningioma

Neurofibromatosis 2

Neurofibromatosis 2 is an autosomal dominant disorder caused by mutation of the *NF2* gene and has an incidence

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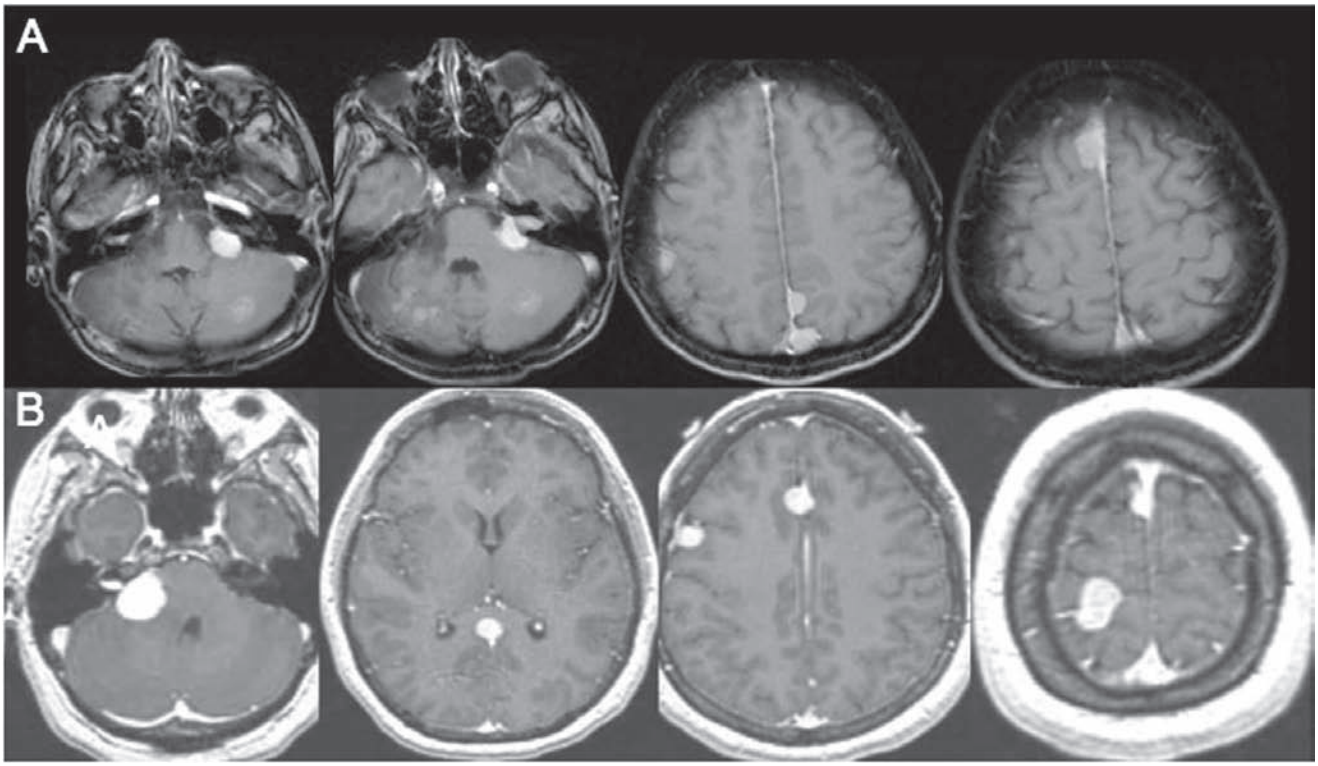


Fig. 10.3 Multiple meningiomas in a patient **(A)** with and another **(B)** without NF2.

of ~1/25,000. It is characterized by mutation of the *NF2* gene on the long arm of chromosome 22, which encodes merlin, a member of the 4.1 protein family which acts as a tumor suppressor by inhibiting mitogenic signaling at the plasma membrane.[39] Merlin is widely expressed in meningeal cells, Schwann cells, peripheral nerves, and the lens. Its inactivation in NF2 classically yields bilateral vestibular schwannomas, but is also associated with meningiomas, ependymomas, astrocytomas, peripheral neuropathies, cutaneous tumors (schwannomas), and ocular abnormalities[40] (**Table 10.3**[41]).

Affected individuals may inherit a mutant allele of *NF2* or acquire a de novo somatic mutation, after which an additional "hit" in the remaining allele is required to confer the disease phenotype. Interestingly, the type of mutation affects disease severity; nonsense and frameshift mutations cause severe disease, whereas milder forms are associated with missense mutations, in-frame deletions, and large deletions.[42] The *NF2* gene is also mutated or lost in ~60% of sporadic, or non-NF2, meningiomas, rendering it the most common genetic lesion in these tumors.[43]

About 50% of patients with NF2 eventually develop intracranial meningiomas. When compared with patients with sporadic nonfamilial meningiomas, these tumors occur earlier in life and have a greater tendency to appear in multiples,[44–46] with some series reporting up to a 38% rate of multiplicity in NF2 patients with meningioma.[45] NF2-associated meningiomas differ histologically from their sporadic counterparts. Tumors in NF2 patients feature higher rates of atypical and anaplastic grades, higher proliferative indices, more mitotic figures, and more frequent brain invasion.[47]

The marked propensity of patients with NF2 to develop multiple schwannomas and meningiomas has mandated close screening in children of parents affected with NF2, with some recommending that this begin at 10 years of age.[48]

Familial Multiple Meningioma (Not Associated with NF2)

Although multiple meningiomas frequently occur in the context of NF2, a genetically distinct form of multiple meningioma not associated with *NF2* mutations has also been described.[49–51] Similar to NF2, familial multiple meningiomas show autosomal dominant inheritance. However, tumors in this familial form do not show linkage to the *NF2* locus[52]; additional studies have shown that *NF2* is neither mutated nor lost in familial multiple meningiomas.[53,54] Tumors from at least one reported kindred retain immunopositivity for merlin.[55]

More recently, an additional member of the 4.1 protein family, *DAL-1* (differentially expressed in adenocarcinoma of the lung), has been implicated in meningioma. Located on chromosome 18p11.3, early reports suggested that *DAL-1* is lost in up to 60% of sporadic meningiomas.[56] Studies employing more contemporary genomic techniques, however, have suggested that the *DAL-1* locus may be lost in only 20% of sporadic tumors, and that *DAL-1* loss is nearly always associated with *NF2* loss.[57] That the merlin and DAL-1 proteins are both 4.1 protein family members linked to the cytoskeleton suggests a co-

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Table 10.3 Diagnostic Criteria for Neurofibromatosis 2[41]

Definite diagnosis of NF2

1. Bilateral vestibular schwannomas

   or

2. First-degree relative with NF2 and either:

   a. Unilateral vestibular schwannoma with onset < 30 years

      or

   b. Any two of the following: meningioma, glioma, schwannoma, juvenile posterior subcapsular cataract

Presumed diagnosis of NF2

1. Unilateral vestibular schwannoma with onset < 30 years and at least one of: meningioma, schwannoma, glioma, juvenile posterior subcapsular cataract

   or

2. Multiple meningiomas (at least two) and:

   a. Unilateral vestibular schwannoma with onset < 30 years

      or

   b. One of schwannoma, glioma, juvenile posterior subcapsular cataract

ordinate molecular pathway in meningioma tumorigenesis, but the specific molecular events await clarification. Although such circuitry may participate in sporadic meningioma, loss of *DAL-1* does not appear to be associated with familial multiple meningioma not associated with NF2,[53] as had been suspected.

The retention of chromosome 22 and absence of *NF2* mutation and apparent lack of involvement of *DAL-1* in some series of familial multiple meningiomas not only mark this syndrome as genetically distinct from NF2 but may provide a fertile context in which to uncover additional tumor suppressors and/or oncogenes responsible for meningioma initiation.

Sporadic Multiple Meningioma

Patients with no family history may also develop multiple meningiomas. Data from limited reports to date show that a majority of these tumors harbor *NF2* mutations, similar to single sporadic meningiomas.[35,53] In a large population-based analysis of meningioma that included solitary and multiple meningioma cases, the gender ratio of nonfamilial multiple meningioma cases was similar to that observed in solitary cases, as were the histologies[58]; smaller case series have reported a stronger female preponderance.[49,59,60] Multiple meningiomas may be found at the convexity, skull base, and posterior fossa and in either hemicranial space.

Several theories have been posited to explain the pathogenesis of sporadic multiple tumors. That some patients harbor multiple tumors with different histology and that karyotypes differ between tumors in the same patient had suggested that each tumor may arise independently. Clonality studies, however, have suggested a monoclonal origin of sporadic multiple meningiomas. A high percentage of patients with multiple tumors harbor the same *NF2* mutation[35] and feature loss of the same copy of chromosome 22, identical *NF2* mutations, and identical methylation status of select genes.[61] These data have supported the notion that multiple tumors may arise from a parent tumor through dissemination of tumor cells to distant rests in any cranial compartment.[36,37]

Management

The management of patients with multiple meningiomas is similar to the approach taken for solitary tumors. Symptomatic or progressively enlarging tumors are candidates for treatment, the gold standard of which is complete surgical resection. In the symptomatic patient with multiple tumors, thorough physical examination and radiological interpretation are required to definitively establish which tumor is causing symptoms. Asymptomatic lesions adjacent to the intended surgical target are often removed if easily accessible through the fashioned craniotomy.

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V

Preoperative Considerations

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Chapter 11

Perioperative Medical Management of Meningioma Patients

Matthew B. Potts, Hugo Q. Cheng, Lewis S. Blevins Jr., Michael W. McDermott, and Susan M. Chang

Introduction

The safe and effective surgical management of meningiomas is dependent upon maximizing medical strategies to reduce the risk of perioperative complications. The surgical management of meningiomas necessarily begins with a preoperative medical evaluation to determine associated risks and help the patient and surgeon make an informed decision about whether to proceed with surgery. This chapter highlights some of the major components of a preoperative evaluation, including assessment of cardiac and pulmonary risks and an appropriate laboratory workup. We also discuss several pre- and postoperative medical strategies to reduce the risk of perioperative complications associated with meningioma resection.

Preoperative Evaluation

There are three main goals to a preoperative medical evaluation: (1) identify whether patients are at increased risk for perioperative complications, (2) determine whether further preoperative workup is needed, and (3) implement strategies to reduce perioperative complications.[1] Such an evaluation is typically done in conjunction with a patient's surgeon, primary care provider, and anesthesiologist, and may involve other specialists as needed. A proper preoperative evaluation should include a complete history and physical exam, with special attention to a patient's medical comorbidities, medications, and surgical history. Much research has been done related to the preoperative evaluation, especially with regard to cardiac and pulmonary evaluations. In a series of 348 meningioma patients, ~1% suffered a postoperative cardiac or respiratory complication.[2]

Cardiac Evaluation

The specific incidence and risk factors for major cardiac complications (cardiovascular death and nonfatal myocardial infarction) after meningioma resection are uncertain. The widely utilized Revised Cardiac Risk Index identifies ischemic disease, congestive heart failure, diabetes, cerebrovascular disease, and chronic kidney disease as risk factors for cardiac complications.[3] Poor functional capacity, as evidenced by the inability to walk at least two blocks, also predicts a higher rate of cardiac complications. Recently revised guidelines for perioperative cardiac evaluations in noncardiac operations recommend delaying elective surgery in patients with unstable coronary artery disease, decompensated heart failure, severe arrhythmias, or severe valvular disease until their cardiac condition has been evaluated and treated.[4] These guidelines recommend noninvasive stress testing for patients with active cardiac conditions, but there is no evidence that stress testing is required for patients undergoing intermediate risk procedures, such as craniotomy. Similarly, there is no evidence to support prophylactic coronary revascularization in surgical patients. In fact, patients with recently placed coronary artery stents are at very high risk of cardiac complications if their antiplatelet agents are stopped prematurely.

Pulmonary Evaluation

The American College of Physicians (ACP) has published guidelines and systematic reviews regarding the assessment and prevention of perioperative pulmonary complications.[5–7] The major predictors of pulmonary complications are the type of procedure and the patient's age. Neurosurgical procedures have been shown to in-

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crease the risk of postoperative pulmonary complications, such as mortality, prolonged intubation or reintubation, and pneumonia, with an odds ratio of 2.53.[6] Of note, prolonged surgery time, defined as >2.5 to 4 hours, also increases the risk of postoperative pulmonary complications with an odds ratio of 2.26. Complicated meningioma resection cases commonly last beyond that time frame. Other risk factors related to pulmonary complications include history of chronic obstructive pulmonary disease or congestive heart failure, patients who are functionally dependent, and patients whose American Society of Anesthesiologists classification is ≥2.6. Surprisingly, obesity and well-controlled asthma have not been shown to be significant risk factors. In addition to history, abnormal findings on lung auscultation predict postoperative pulmonary complications.[8] On the other hand, preoperative pulmonary studies such as chest radiography and pulmonary function testing are generally not useful.[8] The ACP only recommends preoperative chest radiographs in patients with cardiopulmonary disease, age >50 years, or those undergoing abdominal or thoracic surgery.

Useful interventions to reduce pulmonary complications include preoperative respiratory training and postoperative lung expansion procedures, including incentive spirometry and positive pressure therapies.[7] In addition, preoperative smoking cessation has been demonstrated to reduce wound-related complications in orthopedic surgery patients when undertaken at least 6 to 8 weeks before surgery.[7]

Laboratory Workup

The use of routine laboratory studies, including complete blood count, coagulation studies, and electrolytes, as a preoperative evaluation tool is controversial. They are costly to the health care system, and there is evidence that their use may not reduce postoperative complications.[1] The American Society of Anesthesiologists issued a practice advisory in 2002 that does not recommend routine preoperative laboratory studies but instead recommends their selective use based on individual patients and the planned procedure.[9] Intracranial procedures such as meningioma resections have unique risks, including bleeding, that likely warrant the use of routine laboratory studies.

Endocrine Workup

Meningiomas of the suprasellar or hypothalamic region also warrant an endocrinology evaluation to assess for hormone abnormalities related to pituitary stalk effect or hypothalamic compression. Workup should include evaluation of thyroid function (thyroid-stimulating hormone [TSH] and free T4), growth hormone function (somatomedin-c), serum prolactin, random or morning cortisol level, and possibly testosterone function if clinically indicated. Identification of hypothalamic-pituitary-axis abnormalities can help determine whether hormonal replacement therapies are needed (including perioperative stress-dose steroids) and provide documentation if abnormalities exist postoperatively. In addition, an ophthalmology assessment, including formal visual field testing, should be considered for suprasellar lesions. Although confrontation visual field examinations will detect gross visual field defects, alterations in light threshold responsiveness can only be done with modern ophthalmologic equipment. A computerized Humphrey visual field evaluation is recommended as a baseline before surgery for meningiomas in locations around the visual pathways, including the medial third sphenoid wing, clinoid, tuberculum, diaphragma sella, tentorium, and falx. This evaluation should be repeated 3 months postoperatively.

Management of Preoperative Medications

Preoperative Beta-Blockade

Much controversy exists regarding the benefit of perioperative β-blockade. Large randomized trials have shown that prophylactic use of β-blockers can prevent postoperative cardiac complications in higher-risk patients. However, overly aggressive use of these medications increases the risk of stroke and all-cause mortality.[10,11] With regard to most neurosurgical operations, guidelines recommend continuing β-blockers in patients already taking them to treat cardiac disease such as angina, hypertension, or arrhythmia.[4] They may be cautiously started in patients with more than one Revised Cardiac Risk Index predictor.

Anticoagulation

Many patients will be on chronic anticoagulation for thromboembolic prophylaxis in the setting of arterial stents, mechanical heart valves, atrial fibrillation, or a history of arterial or venous thromboembolic disease. Unfortunately, there are few data to provide guidance regarding the perioperative management of anticoagulation in patients undergoing intracranial or intraspinal procedures, such as meningioma resections. A 2008 guideline recommends stopping warfarin 5 days and antiplatelet therapies (aspirin or clopidogrel) 7 to 10 days before a surgical procedure.[12] They also suggest bridging during the perioperative period with low molecular weight heparin or ultrafractionated heparin. These should be stopped 24 hours before the scheduled operation. For high bleeding risk procedures, they recommend restarting bridging therapy 48 to 72 hours postoperatively or when hemostasis has been obtained, or withholding bridging therapy altogether.[12] The use of both pre- and postoperative anticoagulation has not been shown to affect postoperative intracranial hemorrhage in meningioma patients.[13,14] Patients at very high thromboembolic risk, such as those with mechanical mitral valves or recent thromboembolism, may benefit from bridging with parenteral anticoagulants. The relative risk of thrombo-

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embolism and hemorrhage should be discussed with the patient's medical specialists to determine whether a strategy of bridging is justified.

Preoperative Seizure Prophylaxis

Antiepileptic medication should naturally be continued during the perioperative period in patients with a known history of seizures. For meningioma patients with no history of seizures, however, a meta-analysis of five randomized controlled trials in patients with primary brain neoplasms found no effect of prophylactic antiepileptic medication on seizure control.[15] The American Academy of Neurology does not recommend the use of antiepileptic medications in brain tumor patients with no history of seizures.[16]

Other Preoperative Medication Considerations

Although beyond the scope of this review, there are other comorbidities and medications that must be considered before surgical resection of a meningioma. Poorly controlled diabetic patients are at risk of infectious complications and higher mortality rates. In addition, use of oral hypoglycemics can lead to several adverse effects in the perioperative period, including hypoglycemia, and should be stopped preoperatively. Glycemic control should be managed with insulin intraoperatively and during the immediate postoperative period. Patients on chronic steroid therapy should also be assessed and the use of stress-dose steroids considered perioperatively.

Preoperative Autologous and Designated Blood Donation

Given the high vascularity of many meningiomas, blood loss is a definite risk of surgical resection. Many states require a discussion with patients regarding the risks and benefits of blood transfusions as well as the options of preoperative autologous or designated blood donation. Although the risk of contracting various infections through a blood transfusion is low (hepatitis B: 1/200,000; hepatitis C/HIV: 1/2 million),[17] autologous blood transfusions remain the safest option. There is no evidence that directed donor blood is any safer than receiving blood from a random volunteer. Autologous blood donations can typically be stored for more than 1 month and can be made up until just a few days before the planned operation.

Preoperative Imaging and Embolization

A necessary component of the preoperative workup of meningiomas includes imaging. Some imaging modality has usually been obtained to diagnose the meningioma, but additional modalities often provide valuable information for operative planning. Magnetic resonance imaging (MRI) with contrast is the gold standard for diagnosis and evaluation of a meningioma and can also detail involvement of adjacent sinuses. Fat-suppressed T1-weighted sequences are good for parasellar and skull base tumors because they eliminate the high signal of bone marrow and allow true contrast enhancement to be appreciated. Operative planning for parasagittal, falcine, and tentorial meningiomas can be assisted by magnetic resonance venography, which can also be displayed as three-dimensional models on current image guidance systems in the operating room. Diffusion tensor imaging can be employed for meningiomas in the atria of the lateral ventricles to identify adjacent visual fiber white matter tracts for operative planning. For hyperostosing meningiomas, computed tomographic (CT) scans can still provide valuable information about the extent of bony involvement that is not well appreciated on MR. Preoperative angiography is used to evaluate the arterial supply and can be combined with embolization to facilitate subsequent surgical resection.[18] The venous phase of the angiogram is the definitive method for determining venous sinus patency. Finally, in any case where significant rotation of the neck is needed for positioning, or in patients with neurofibromatosis type 2, a preoperative MRI scan of the cervical spine should be obtained to rule out cervical stenosis or the presence of other lesions.

Postoperative Medical Management and Prophylaxis

Postoperative Fluids

Fluid management is important after any surgical procedure, especially prolonged surgeries where extensive fluid shifts can be expected. With respect to meningioma resections, appropriate postoperative fluid management can also be used to minimize complications, including cerebral edema or venous infarction in patients with meningiomas adjacent to venous sinuses. In contrast to patients with intraaxial tumors, it is customary at our institution to maintain meningioma patients on fluids for several days postoperatively to avoid dehydration and prevent venous infarction. Serum electrolytes, especially sodium, should be closely monitored postoperatively due to risk of syndrome of inappropriate antidiuretic hormone secretion (SIADH), cerebral salt wasting, or diabetes insipidus.

Postoperative Pain

Adequate pain control is often not achieved after intracranial operations due to concern over the neurological side effects of common analgesics.[19] Short-acting opiates are the mainstay of postoperative pain management immediately after an intracranial or intraspinal tumor

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resection. The use of patient-controlled analgesia with intravenous fentanyl has even been shown to be more effective than standard as-needed analgesia administration, with no increase in complications.[20] Several other techniques can be used to safely minimize postoperative pain, including administration of local anesthetic to the incision site at the conclusion of an operation and use of adjunctive medications such as anticonvulsants and glucocorticoids.[19]

Postoperative Anemia

Blood loss is a major risk factor of intracranial and spinal surgery, especially with highly vascular meningiomas. Both pre- and postoperative anemia have been associated with pneumonia and increased mortality.[21] There is also mounting experimental evidence that anemia may adversely affect neurological outcome,[22] although it is not known how anemia specifically affects outcomes in neurosurgical patients. Blood transfusion is associated with its own risks, including infection, immune reactions, and even increased mortality.[23] Comparisons of liberal (hemoglobin < 10 g/dL) or restrictive (hemoglobin < 7 g/dL) transfusion strategies in critically ill patients have shown no difference in 30-day mortality except in patients with myocardial infarction or pulmonary edema, where a restrictive threshold improved outcomes.[21] The decision to transfuse a postoperative meningioma patient should therefore be based on several factors, including the patient's comorbidities and hemodynamic status. In addition, other therapies, such as iron supplementation and erythropoiesis-stimulating agents, can be considered.[21]

Postoperative Deep Vein Thrombosis Prophylaxis

Patients with primary brain tumors are at increased risk for venous thromboembolism, especially during the postoperative period and if any hemiplegia is present.[24] Heparin or low-molecular-weight heparin is commonly used to prevent postoperative venous thromboembolism, but its use is controversial in neurosurgical patients, where the consequences of hemorrhage can be devastating. Several studies have demonstrated an overall benefit of postoperative anticoagulation with low-molecular-weight heparin in neurosurgical patients.[25,26] A recent study of low-molecular-weight heparin in postoperative meningioma patients similarly demonstrated a trend toward improved venous thromboembolism rates with no increase in hemorrhagic complications.[14] Intermittent pneumatic compression devices should be used if anticoagulants are deemed to be too risky. In our practice, we start uncomplicated meningioma patients on deep vein thrombosis (DVT) prophylaxis beginning 48 hours postoperatively.

Postoperative Steroids

Dexamethasone is the standard therapy for peritumoral and postoperative cerebral edema.[24] There is no evidence to guide the dosage or duration of postoperative steroid use in meningioma patients, so the use of dexamethasone is typically based on a surgeon's personal experience and clinical intuition. It is important to be aware of the many adverse effects associated with prolonged glucocorticoid use.[24]

Postoperative Seizure Prophylaxis

Meningioma resection improves seizure control in patients with preoperative epilepsy, but ~5 to 20% of patients may develop new postoperative seizures.[27,28] The use of postoperative antiepileptic medications such as phenytoin after intracranial neurosurgical procedures has been shown to reduce early postoperative seizures by ~40 to 50%.[29] Recent studies comparing levitiracetam to phenytoin have also shown no difference in postoperative seizure rates in glioma patients.[30,31] The American Academy of Neurology currently recommends discontinuing postoperative seizure prophylaxis after 1 week in patients with no history of seizures.[16]

Postoperative Antibiotics

Prophylactic antibiotics should be administered immediately before skin incision,[32] but there is little evidence supporting their prolonged postoperative use in routine patients.[33] Extended antibiotic prophylaxis is recommended for endonasal transsphenoidal cases where the dura is opened, in which case antibiotics are generally continued until any nasal packing is removed.

Postoperative Cerebrospinal Fluid Drainage

Little is written on the management of cerebrospinal fluid (CSF) diversion and drainage in the postoperative period in meningioma patients. At our institution, where more than 1200 meningioma patients have been surgically managed since 1992, we have adopted some standard practices. In patients where a lumbar subarachnoid drain (LSAD) was placed intraoperatively to drain CSF and reduce intracranial pressure, the drain is kept in place and clamped after surgery until the next day when the patient can be fully evaluated. This avoids the risk of overdrainage and the potentiation of postoperative hematomas should a CSF leak persist after drain removal. If the patient remains stable, the drain is removed around 24 hours postoperatively and the patient is kept lying flat with the head of bed < 10 degrees for 2 hours before mobilization. If symptoms of intracranial hypotension appear, a CT-guided blood patch is performed by interventional radiology and the patient is kept flat for anoth-

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er 2 hours after that. For patients whose LSAD was left in place postoperatively to drain CSF and assist with dural healing, the drain is once again kept clamped for 24 hours after surgery. Thereafter, the drain is opened to drain 10 to 15 mL per hour. We typically continue drainage for 3 days for posterior fossa cases and 5 days for anterior skull base cases. Low-molecular-weight heparin is contraindicated while the LSAD is in place. Anticoagulation is not started until 24 hours after drain removal.

Postoperative Delirium

Delirium is a common postoperative complication, especially in elderly patients, and is associated with longer lengths of stay, need for discharge to a long-term care facility, and higher mortality.[34] Known factors associated with postoperative delirium include age > 70 years, history of alcohol abuse, poor cognitive or functional status, and abnormal preoperative serum sodium, potassium, and glucose levels.[34] Strategies to prevent delirium include maintenance of adequate oxygenation, correction of electrolyte abnormalities, close attention to pain management, elimination of unnecessary psychoactive medications (i.e., anticholinergics and benzodiazepines), promotion of normal bowel and bladder function, attention to nutritional status, early mobilization, prevention and management of postoperative complications, and use of environmental stimuli.[35] A directed medical workup for postoperative delirium should include limited laboratory studies (complete blood count, electrolytes, urinalysis), electrocardiogram, and review of medications. Neuroleptics such as haloperidol are the preferred agents to treat severe, agitated delirium.

Management of Postoperative Medical Complications

Deep Vein Thromboses and Pulmonary Emboli

The acute management of DVTs and pulmonary emboli typically involves anticoagulation with intravenous unfractionated heparin or subcutaneous low-molecular-weight heparin,[36] but the hemorrhagic risk of such treatments in the postoperative period must be carefully weighed. Intravenous unfractionated heparin has a shorter half-life and can be reversed with protamine. Thus its anticoagulation effects can be more rapidly stopped if a hemorrhagic complication occurs. The dosage of intravenous unfractionated heparin is titrated to a patient's activated partial thromboplastin time (aPTT), although this value is often laboratory specific, and most hospitals therefore have their own protocol for administering intravenous unfractionated heparin. Standard heparin infusion protocols often start with a bolus dose. At our institution, however, we omit the bolus dose to minimize risk of hemorrhage. Once stable on parenteral anticoagulants, patients should be transitioned to long-term anticoagulation with warfarin [goal international normalized ratio (INR) 2.0 to 3.0].[36] If hemorrhagic risk is deemed too high to permit anticoagulation, an inferior vena cava filter can be placed to prevent embolization of clot from the lower extremities to the lungs. In our practice, we wait until 2 weeks to begin full anticoagulation in uncomplicated postoperative meningioma patients.

Postoperative Seizures

Seizures persist postoperatively in ~11 to 37% of meningioma patients with a history of preoperative seizures, whereas new seizures occur postoperatively in 5 to 20% of surgically treated meningioma patients.[27,28] Seizures in the immediate postoperative period, especially if they progress to status epilepticus, carry the risk of inducing other complications, including intracranial hemorrhage or anoxic brain injury. Management of seizures in this setting should include rapid treatment with antiepileptic medications as well as a workup of the etiology. Typical first-line therapy for postoperative seizures includes a loading dose of phenytoin or fosphenytoin (15 to 20 mg/kg IV). Fosphenytoin has an advantage over phenytoin in that it can be infused much more rapidly if necessary (150 mg phenytoin-equivalents/min for fosphenytoin compared with 50 mg/min for phenytoin). In the setting of status epilepticus, a benzodiazepine such as lorazepam (0.1 to 0.15 mg/kg) should be used in conjunction with phenytoin/fosphenytoin. A second dose of lorazepam may be given if seizures persist. For refractory status, second-line therapies include additional phenytoin/fosphenytoin, phenobarbital, propofol, pentobarbital, and additional antiepileptic medications such as valproate, levetiracetam, and topiramate.[37,38] A workup to identify correctable causes should also be started and should include a noncontrast head CT scan to rule out hemorrhage and standard laboratories (electrolytes and complete blood count) to rule out metabolic causes such as hyponatremia. Electroencephalography may be used to identify subclinical seizures in a patient with altered mental status but no outward signs of seizure activity.

Sodium Dysregulation

Two common disorders of sodium regulation after neurosurgical procedures are SIADH[39] and diabetes insipidus (DI).[40] SIADH occurs when excess antidiuretic hormone (arginine vasopressin) is released for a given plasma tonicity, leading to decreased renal free water clearance and dilutional hyponatremia.[41] Standard therapies include fluid restriction and sodium supplementation with oral sodium chloride tablets or 3% intravenous sodium chloride. A new class of arginine vasopressin antagonists known as vaptans has also recently been shown to be effective in the treatment of SIADH in neurosurgical patients.[42] In contrast, DI is caused by inadequate arginine

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vasopressin activity and manifests as polyuria and polydipsia in the setting of dilute urine (urine specific gravity ≤ 1.005). DI can occur after any operation involving the pituitary, stalk, or hypothalamus. Patients may be able to maintain a normal serum sodium if they can match their urine output with oral free water input. If not, however, patients may become hypernatremic. Standard therapy for DI is to allow patients to drink to thirst and, if they become hypernatremic, to use desmopressin (DDAVP, 1 µg IV or 10 µg intranasal) to restrict urine output to less than 3L/day in an average-sized person. Postoperative DI is typically transient, but patients can be discharged home with intranasal DDAVP on an as needed basis if DI persists.

Postoperative Imaging, Rehabilitation, and Follow-Up

A postoperative scan (either CT or MRI) is typically obtained within the first few postoperative days to serve as a baseline for future imaging studies. It is also important to evaluate patients early in their recovery for any physical, occupational, or speech therapy needs. Such therapies can be initiated in the inpatient setting and continued at a rehabilitation facility or on an outpatient basis. Postoperative care must also include monitoring of the surgical incision, typically done within 1 to 2 weeks after the operation and at subsequent follow-up appointments. The patient should also be evaluated for any possible adjuvant therapies if residual tumor exists and should continue to undergo long-term follow-up with serial imaging to monitor for recurrence.

Conclusion

Overall, the proper perioperative management of meningioma patients requires a thoughtful multidisciplinary approach to maximize the safety and efficacy of the operative experience for the patient. A preoperative evaluation should be done with cooperation with anesthesia and other medical disciplines as the patient's comorbidities dictate. The surgeon's attention to detail in the pre- and postoperative period will provide the best possible outcomes.

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Chapter 12

Risk Evaluation and Anesthesia for Intracranial and Spinal Meningiomas

W. Scott Jellish

The evaluation of a patient's fitness to undergo the stress of an anesthetic and surgical procedure constitutes the anesthesiologist's assessment of risk. This assessment is based on the knowledge of prevalence rates for unwanted consequences in population groups sharing the same characteristics. The anesthesiologist must identify the risks of the surgery and either avoid the conditions that would predispose the patient to that risk or develop a means to alter the consequences of the surgical intervention that would lead to the risk. The risks for meningioma resection consist of those related to the operative site and those related to the anesthetic. The operative site is influenced by the position of the tumor, its characteristics, and its size. Depending on the location, these tumors may engulf nerves and blood vessels, invade large vascular structures such as the cavernous sinus, and extend into multiple cranial fossa and foramina. As a result, these surgeries may be time consuming, with the potential for large blood loss and fluid shifts.

Risk factors such as age, poor preoperative clinical condition, and tumor characteristics have been associated with increased morbidity and mortality of meningioma surgery.[1] Tumor characteristics that should be known to the anesthesiologist for assessment of risk include size of the vessels supplying and draining the meningioma and whether the tumor has been embolized. Blood dyscrasias secondary to medication or chronic alcohol use could increase the propensity for bleeding. The role of aspirin and other nonsteroidal antiinflammatory agents cannot be discounted as important risk factors for bleeding, especially in patients with chronic pain due to headache, rheumatoid arthritis, osteoarthritis, gout, and ankylosing spondylitis.

Hypertension, cardiomyopathies, diabetes, pneumopathies, and peripheral vascular disease may all affect the patient's surgical risk. The preoperative risk assessment should be based on a thorough and efficient evaluation of the patient that addresses the following key points:

1. Does the physical status of the patient increase the risk of mortality and morbidity during the perioperative period?
2. What current disease processes and medications influence the intraoperative and postoperative course?
3. What immediate medical action would be most beneficial to the patient to increase the chances for a successful outcome? The assessment may also include decisions for noninvasive testing to better estimate risk.

The anesthesiologist's risk assessment is uniquely suited to the patient and includes an adequate and specific history with physical exam that will lead to confirmatory laboratory tests. This history is crucial to the discovery of cardiac and other symptomatic diseases that could place the patient in a higher risk category.

The history has been demonstrated to give primary information concerning a patient's physical state in ~60% of the cases.[2] The history should identify serious cardiac conditions, signs of congestive heart failure, arrhythmias, or severe valvular disease. The first key aspect of the history is the assessment of exercise tolerance. Usually patients are asked if they can climb two sets of stairs, which is equivalent to 4 metabolic equivalent tasks (METs) of activity (**Table 12.1**). The inability to perform this task should arouse suspicion of possible congestive heart failure or coronary artery disease. Other basic questions determining vitality, mobility, and fitness are asked, along with a review of systems to determine evidence of chronic disease, pulmonary disorders, and recent upper airway or genitourinary infection. Histories of hospitalization and surgeries, family history, and social history,

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Table 12.1 Estimated Energy Requirements for Various Activities

1 MET
Can you:
Take care of yourself?
Eat, dress, or use the toilet?
Walk indoors around the house?
Walk a block or two on level ground at 2 to 3 mph (3.2 to 4.8 kph)?
Do light work around the house like dusting or washing dishes?
4 METs
Can you:
Climb a flight of stairs or walk up a hill?
Walk on level ground at 4 mph (6.4 kph)?
Run a short distance?
Do heavy work around the house like scrubbing floors or lifting or moving heavy furniture?
Participate in moderate recreational activities like golf, bowling, dancing, doubles tennis, or throwing a baseball or football?
Greater than 10 METs
Can you:
Participate in strenuous sports like swimming, singles tennis, football, basketball, or skiing?

Abbreviations: kph, kilometers per hour; MET, metabolic equivalent task; mph, miles per hour.
Adapted from Fleisher LA, Beckman JA, Brown KA, et al,[9] with permission

including alcohol intake and use of drugs, are important considerations.

Age must always be considered in patients undergoing meningioma resection.[3] This factor can contribute to increased morbidity and mortality associated with the meningioma resection. Although much of the increased morbidity related to age is appropriately attributed to comorbidity, a patient > 80 years of age will have reduced physiological reserve. Numerous studies have found a significant increase in mortality related to surgery beginning at the age of 70. The elderly patient has diminished cardiac reserve and a higher incidence of atrial fibrillation secondary to degenerative changes in the conduction system.[4] Depression, dementia, and other neurological disorders may also be associated with advanced age, and neurological impairment, visual loss, or changes in vision should be assessed. Vital capacity and forced expiratory volume at 1 second decrease roughly 1% per year.[5] Preoperative questions concerning cough, sputum production, hemoptysis, pneumonias, shortness of breath, chest pain, and reduced physical activity may point to changes in either cardiac or respiratory reserve, which may need further evaluation before surgery.

Age also affects renal function and liver synthetic activity. Glomerular filtration rates decline linearly from 80 mL/min/m^2 at age 30 to 58 mL/min/m^2 by age 80.[6] Blood flow to the kidneys will be half that of young adults by age 65. This may result in a decreased ability to concentrate urine and an inability to regulate electrolytes. Liver function and blood flow may be reduced in elderly patients, and the nutritional status may be questionable. Drug clearance and metabolism are reduced, and serum albumin may be low, producing an increase in the free fraction of highly protein bound drugs. The history obtained by the anesthesiologist should focus on eating habits, bowel and bladder function, and overall general nutrition.

The next major anesthetic assessment to determine risk is the ability to intubate and ventilate the patient for the surgical procedure. Closed claims analysis reveals that 85% of airway incidents involved brain damage or death due to the inability to ventilate and intubate the patient.[7] This is only exacerbated in an individual with increased intracranial pressure secondary to a large brain mass. The identification of a patient with a difficult airway is vital in planning the anesthetic management and assessment of risk the patient will assume. Malformation of the face, acromegaly, cervical spondylosis, occipitoatlantoaxial disease, tumors of the airway, and long-term diabetes producing stiff joint syndrome carry added risk. Head movements and the ability to hyperextend the neck with a thyromental distance of greater than 6.5 cm and the ability of the patient to prognath the jaw and an interincisor gap of > 5 cm would provide evidence of a large mouth opening and easy laryngoscopy. Dental pathology, especially loose teeth, should be identified as they could become dislodged and be aspirated during laryngoscopy, which could add considerable morbidity to the procedure (**Table 12.2**). The Mallampati Classification consists of the criteria most used for assessing difficulty with intubation and adjusting patient risk for obtaining a patent airway.[8] Class I–IV designations of anatomical appearance predict ease of intubation, with class (IV) the most difficult to intubate. Dyspnea related to airway compression, dysphagia, and sleep apnea along with clinical signs (obesity, limited mouth opening, or large tongue) should be treated as portending a difficult airway until proven otherwise.

The next major assessment of perioperative risk involves the cardiovascular system and the patient's fitness to undergo the procedure without major morbidity or mortality. The cardiovascular examination should include an assessment of vital signs, carotid pulse, evidence of jugular venous distention, auscultation of the lungs, precordial palpation and auscultation, abdominal palpation, and examination of the extremities for edema and vascular integrity. The patient who presents for major noncardiac procedures poses the biggest problem for assessment of fitness to undergo a surgical procedure. The general appearance of the patient provides evidence of overall physical health. Cyanosis, pallor, dyspnea during conversation or with minimal activity, Cheyne-Stokes respiration, obesity, skeletal deformities, tremor, and

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Table 12.2 Components of the Preoperative Airway Physical Examination

Airway Examination Component	Nonreassuring Findings
Length of upper incisors	Relatively long
Relationship of the maxillary and mandibular incisors during normal jaw closure	Prominent overbite (maxillary incisors anterior to the mandibular incisors)
Relationship of the maxillary and mandibular incisors during voluntary protrusion of the mandible	Patient cannot bring the mandibular incisors anterior to (in front of) the maxillary incisors
Interincisor distance	Less than 3 cm
Visibility of the uvula	Not visible when the tongue is protruded with the patient in a sitting position (Mallampati class greater than II)
Shape of the palate	Highly arched or very narrow
Compliance of the mandibular space	Stiff, indurated, occupied by a mass, or nonresilient
Thyromental distance	Less than three fingerbreadths
Length of the neck	Short
Thickness of the neck	Thick
Range of motion of the head and neck	Patient cannot touch the tip of the chin to the chest or cannot extend the neck

Adapted from Stackhouse RA, Infosino A. Airway management. In: Stoelting RA, Miller RD, eds. Basics of Anesthesia. Philadelphia, PA: Churchill Livingstone Elsevier; 2007:211.

anxiety are a few clues of underlying disease that can be recognized by a skilled physician.

High-risk cardiac patients, as assessed from the history and physical exam, need further investigation to determine functional status and to help in developing the plan for intraoperative monitoring (**Table 12.3**). An electrocardiogram (ECG) sometimes uncovers occult disease in older adults, but it rarely shows clinically important abnormalities in younger asymptomatic patients. Preoperative resting ECGs are recommended for patients undergoing large meningioma resection if they have evidence of coronary artery disease, peripheral vascular disease, high blood pressure, diabetes, history of congestive heart failure (CHF), shortness of breath, and cigarette smoking.[9] ECG abnormalities that have the potential to alter management include atrial flutter or fibrillation; first-, second-, or third-degree atrioventrivular (AV) block; ST segment changes suggestive of ischemia, premature ventricular and atrial contractions; left ventricular (LV) or right ventricular (RV) hypertrophy; short PR interval; Wolff-Parkinson-White syndrome; prolonged QT interval; peaked T waves and small voltages indicative of cardiomyopathy.

Further cardiac studies to stratify risk may be beneficial for patients who by history or physical exam are considered to be at intermediate risk of cardiac complications. The ECG exercise treadmill test is useful in patients who can exercise but is rarely applicable to patients with ischemic lower extremities. A positive exercise test only slightly increases the likelihood of coronary artery disease, and a negative test correlates poorly with the absence of heart disease.[9]

Pharmacological stress testing should be considered for patients with an abnormal ECG (including left and possibly right bundle branch block) or a history of myocardial infarction. It should also be considered for those taking digoxin and in those who cannot exercise to acceptable levels. Studies of prospective cardiac risk using dipyridamole thallium scans (DTSs) suggest that patients with normal studies have a low risk for cardiac complications, but the prognostic implication of an abnormal scan is less well established. Studies have shown that reversible perfusion defects, which reflect jeopardized viable myocardium, carry the greatest risk of cardiac death or myocardial infarction (MI).[10] In more recent publications, the positive predictive value of myocardial perfusion imaging has decreased significantly. This fact has reduced this procedure's usefulness for evaluating assessment of surgical risk.

The predictive value of 24- or 48-hour ambulatory ECG monitoring for determining the perioperative risk of MI in patients undergoing high-risk noncardiac surgery is not widely used because differences in study protocols and ambulatory versus in-hospital monitoring may account for variability in the predictive value of the test.

Radionuclide ventriculography (RNVG) for assessment of LV function and ejection fraction (EF) can predict perioperative cardiac morbidity in patients undergoing high-risk procedures. The scan shows ventricular wall motion abnormalities and systolic/diastolic dysfunction. Pasternak et al[11] demonstrated that a calculated EF of < 35% was associated with a perioperative MI rate of 20%. The combined relative risk with the stipulated EF was 3.7, delineating a positive result. Measurement of EF using this

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Table 12.3 Cardiac Risk Stratification for Noncardiac Surgical Procedures*

Stratification
High (reported cardiac risk often > 5%)
Emergent major operations, particularly in the elderly
Aortic and other major vascular
Peripheral vascular
Anticipated prolonged surgical procedures associated with large fluid shifts and/or blood loss
Intermediate (reported cardiac risk generally < 5%)
Carotid endarterectomy
Head and neck
Intraperitoneal and intrathoracic
Orthopedic
Prostate
Low ** **(reported cardiac risk generally < 1%)**
Endoscopic procedures
Superficial procedure
Cataract
Breast

* Cardiac risk signifies combined incidence of cardiac death and nonfatal myocardial infarction.
** Does not generally require further preoperative testing
Reprinted from Belzberg H, Rirkind AI. Preoperative cardiac preparation. Chest 1999;115:86S, with permission.

technique is one of the strongest predictors of overall and late survival, especially after vascular surgery.

Dobutamine stress echo (DSE) was developed as a tool for assessing the presence of coronary artery disease. It has become the method of choice for pharmacological stress testing. The test assesses the effect of incremental infusions of supratherapeutic doses of dobutamine, which increases myocardial contractility and heart rate. Significant coronary disease can be identified by induction of LV ischemic regional wall motion abnormalities.[12] DSE is recommended in patients with intermediate clinical predictors (prior MI, compensated CHF, diabetes, and mild angina). Integration of these clinical risk factors with ischemic wall motion abnormalities enhances the value of DSE in predicting perioperative nonfatal MI or death with high-risk procedures.

Coronary angiography is not recommended for risk assessment in patients having noncardiac surgery unless there is clinical evidence of coronary artery disease and the patient is undergoing a moderate- to high-risk procedure. If coronary artery disease or cardiac dysfunction is severe enough to indicate coronary angiography, the anesthesiologist presumes the risk to be so high that the patient will undergo a cardiac event. The anesthesiologist also presumes a corrective procedure will be performed if an appropriate lesion is found. The presence of significant coronary stenosis does not always indicate that an MI is unavoidable and that an invasive procedure is needed before surgery because the involved artery may supply scar tissue and not viable myocardium.

The potential complexity of perioperative cardiac risk evaluation makes the need for a simple algorithm apparent (**Table 12.4**). Eagle et al used multivariate predictors of age > 70, angina, Q waves on ECG, ventricular ectopic activity, and diabetes to divide patients by these clinical variables.[13] Patients with no risk factors had a 3.1% incidence of a myocardial event, whereas those that had three or more factors had a 50% incidence. Patients with one or two variables who were subjected to DTS with negative results had a 3.2% incidence of cardiac events, whereas those with a positive DTS test had a 29.2% incidence of cardiac complications. Thus the incorporation of invasive tests along with known cardiac risk factors improved the positive predictive value of the known risk factors.

The evaluation of pulmonary function is important in assessing patient risk for surgery. Pulmonary complications are common with abdominal and thoracic procedures, with less morbidity associated with intracranial procedures. In addition to pneumonia, postoperative pulmonary complications may include massive lobar collapse due to mucous plugging, pneumonitis, atelectasis, or a combination of one or more of these problems, which are exacerbated by prolonged intubation and intensive care unit (ICU) stays. The high incidence of these complications and the associated costs make it imperative that patients at risk be identified and pulmonary function optimized before the surgical procedure.

Table 12.4 Association between Clinical Markers of Risk and Rate of Perioperative Death or Myocardial Infarction

Clinical Markers	Clinical Risk Groups Based on Number of Markers	Perioperative Myocardial Infarction or Death (%)
Age > 70 years	Low risk (no clinical markers)	3
Diabetes	Intermediate risk (1 or 2 markers)	8
Prior angina	High risk (3 markers)	18
MI by history or electrocardiogram (Q waves)		
Congestive heart failure		

Data from L'Italien GJ, Paul SD, Hendel RC, et al. Development and validation of a Bayesian model for perioperative cardiac risk assessment in a cohort of 1,081 vascular surgical candidates. *J AM Coll Cardiol* 1996;27(4):779–786.

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A variety of metabolic diseases could accompany the presentation of the meningioma. Diabetes predisposes the patient to specific risks, and its presence should heighten the suspicion of coronary artery disease because older patients with diabetes are more likely to develop heart failure after surgery than those without diabetes. Management of blood glucose levels may be difficult in the perioperative period, especially in patients receiving glucocorticoids to reduce cerebral edema and increased intracranial pressure associated with some large meningiomas. The risk assessment by the anesthesiologist must take into account the problems associated with both hyper- and hypoglycemia and the increased risk of postoperative infection and metabolic abnormalities. Azotemia is commonly associated with cardiac disease, and renal function should also be assessed in patients undergoing meningioma resection. Maintenance of adequate intravascular volume for renal perfusion during diuresis of a patient with an intracranial mass and associated renal insufficiency may be challenging.

Anemia in the surgical patient will produce additional stresses during and after surgery, which could place the patient at risk of perioperative morbidity.[14] Cardiac output and tissue perfusion can increase fourfold with anemia in patients with normal heart function. If heart function is not compromised, normal blood flow can be maintained at hemoglobin levels as low as 5 gm/dL. In a patient with coronary artery disease, however, hemoglobin levels less than 10 gm/dL may be detrimental to ventricular function. Few studies have considered the effect of preoperative anemia on mortality. However, an inverse relationship exists between the perioperative hemoglobin level and the incidence of mortality in Jehovah's Witness patients. The morbidity associated with transfusion, especially that of infection or human error associated with administration of the wrong blood, must be factored into the risk of undergoing surgery.

The numerous factors that are considered in the perioperative assessment of risk are difficult to convey; thus a generalized scoring system that stratifies patients according to the severity of their illness is imperative to better estimate morbidity and mortality. The scoring system most often used by anesthesiologists to assess risk is the American Society of Anesthesiologists (ASA) Physical Status Classification.[15] Patients are allocated to one of five categories (1 best, 5 worst) based on the medical history and physical exam (**Table 12.5**). A variant of the ASA scoring system has been developed (by Klotz et al[16]) in which patients are assigned to one of three risk groups on the basis of a score obtained from a combination of four variables: (1) severity of the operation, (2) ASA grade, (3) presence of malignancy, (4) symptoms of respiratory disease. This scoring system provides a more accurate assessment of risk but is not widely used.

Once operative risk has been identified, the anesthesiologist should make specific recommendations for strategies to reduce risk in preparation for surgery. The preoperative "tune up" can be done on an outpatient basis if time and the nature of the surgery permit. Patients with pulmonary disease should have their bronchodilator therapy maximized and any evidence of infection cleared. Incentive spirometry done before surgery helps recruit lung units and reduces the incidence of perioperative hypoxia and atelectasis. Cardiac patients should be allowed to continue their medications in the preoperative period. Antiarrhythmics can be temporarily switched to intravenous forms, and sublingual medications like nifedipine can be switched to IV formulation for the surgery.

Table 12.5 American Society of Anesthesiologists Classification

1. Normal healthy patient
2. Mild systemic disease
3. Severe systemic disease that limits activity
4. Incapacitating systemic disease that is a constant threat to life
5. Moribund patient not expected to survive > 24 hours, with or without operation

Data from American Society of Anesthesiologists. New classification of physical status. Anesthesiology 1963:24:111.

Airway concerns with spinal meningiomas are usually minimal unless the tumor is located in the cervical portion of the cord and could compress structures with neck movement, especially hyperextension, during laryngoscopy. An awake fiberoptic intubation may be the appropriate option to secure the airway with tumor involvement of the cervical cord. Intracranial and skull base meningiomas, depending on size and location, may have associated increased intracranial pressure (ICP). If the patient presents with evidence of increased ICP, the anesthetic induction must blunt any increase in blood pressure associated with laryngoscopy but not lower blood pressure, which could jeopardize cerebral perfusion. Selection of the anesthetic agent for induction is less important than careful titration of the drug to control hemodynamics. The ideal intravenous induction agent in patients with increased ICP should maintain cerebral perfusion pressure, prevent changes in mean arterial pressure, and preferably decrease ICP. Thiopental has a successful history of use during induction of anesthesia in these patients. ICP is lowered by cerebral vasoconstriction, but myocardial depression and peripheral vasodilatation may be profound. A reduced dose of this drug coupled with the administration of narcotics often reduces the myocardial depression observed. Etomidate tends to maintain cardiovascular stability on induction with a dose-dependent decrease in cerebral blood flow. Use of this drug is not widespread because associated myoclonus resembles seizure activity, and its administration is associated with adrenal suppression. Propofol can also be used to induce general anesthesia. Systemic vasodilation may reduce mean arterial pressure to such a degree that it could compromise cerebral perfusion. A common induction sequence for a patient with increased ICP would be 3 to 5 mg/kg of thiopental, 3 to 5 µg/kg fentanyl with 100 mg of lidocaine with small doses of esmolol, if needed, to blunt the cardiovascular response to laryngoscopy.

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Use of muscle relaxants can be controversial. Pancuronium is a sympathomimetic and may increase mean arterial pressure. Vercuronium and rocuronium produce stable hemodynamics, with rocuronium used to replace succinylcholine if a rapid sequence induction is needed. Succinylcholine use in patients with high ICP is controversial because fasciculations and an increase in muscle spindle activity have been shown to increase ICP. Volatile anesthetics have been used for many years in patients undergoing both spinal and intracranial meningioma resection. Isoflurane produces moderate vasodilatation, which can be attenuated by hyperventilation. Newer volatile agents, such as desflurane and sevoflurane, behave in a similar fashion, producing mild cerebral vasodilatation coupled with a decrease in cerebral metabolic rate. Responsiveness to CO_2 is maintained so the effect of increased cerebral blood flow is attenuated with hyperventilation. Narcotic use in these long and sometimes stimulating cases is the foundation for maintenance anesthesia. These drugs provide hemodynamic stability without deleterious effects on ICP and allow for rapid emergence at the conclusion of the surgery. Remifentanil, with its rapid clearance by esterase metabolism, may provide rapid emergence from general anesthesia, enabling a quick neurological assessment. Meningiomas have large vascular footprints, and care must be taken to control blood pressure with emergence from remifentanil anesthesia. Hypertension after meningioma resection could produce bleeding at the site of the tumor bed, which could increase the incidence of a subdural hematoma after surgery.

The anatomical position of the tumor will also dictate the operative positioning of the patient. Certain skull base meningiomas dictate that the patient be placed in a semisitting, lateral decubitus, or park bench orientation. Abnormal flexion or extension of the extremities is avoided, and care is taken to avoid abnormal neck and arm position, which could produce brachial plexus injury.

Positioning can also dictate the type of invasive lines that should be placed for the surgical procedure. The surgical field above the heart (sitting or park bench) could offer better anatomical exposure with less blood loss. However, these positions produce a high prevalence of venous air entrainment. Convexity meningiomas, especially if there is bony involvement, should also be considered for venous air embolus (VAE) monitoring.[17] The bone has been noted to be a source of VAE in 43% of all sitting craniotomies. If the procedure warrants VAE monitoring, a central multiorifice catheter should be placed and positioned in the right atrium 1 to 2 cm above the tricuspid orifice. Monitoring for VAE has been extensively described. We prefer the use of the precordial Doppler (Versitone Model D8, Medasonics, Inc., Fremont, CA). Although many feel that the transesophageal echo is more sensitive in detecting venous air entrainment, the increased sensitivity produces a high false-positive rate, which reduces its specificity as a monitor to detect air emboli.

Blood loss during meningioma resection can be quite large. The position of the tumor, close to vascular structures, could produce a great deal of blood loss during resection. Many of these tumors are highly vascular and have been shown to produce a tissue type plasminogen activator that leads to significant fibrinolysis, producing increased blood loss during surgery.[18] Some investigators have noted a disseminated intravascular coagulopathy during primary brain tumor resections. This could develop from tumor-specific antigens or destruction of the blood–brain barrier leading to liberation of factors activating hemolysis. Large-bore IV access should be initiated in these patients before surgery. If anticipated blood loss is large, central venous access should be obtained, with fluid and blood administration managed with the use of a Swan-Ganz catheter.

Intraoperative neurophysiological monitoring for resection of spinal or cranial meningiomas is an important consideration when developing the anesthetic plan for the procedure. Cranial nerve monitoring may be employed if the tumor is located in the skull base and surrounds nerve structures. Electromyography of the facial, vagus, or trigeminal nerve may be used during surgical resection to identify the nerve and preserve its integrity. The use of muscle relaxants, in conjunction with electromyographic (EMG) monitoring, is problematic, with the best conditions realized by complete avoidance of muscle relaxants.[19] Maintenance of anesthesia with low-dose desflurane administration in a 50:50 air/O_2 mixture with a baseline infusion of fentanyl 2 to 3 μg/kg/hr or remifentanil 0.25 to 0.35 μg/kg/min will produce the intraoperative conditions necessary to monitor cranial nerves and provide a stable surgical field without the use of muscle relaxants.

Other neurophysiological monitors may also be utilized for skull base or spinal cord meningioma resection. Brain stem auditory evoked responses or electrocochleography can be used for skull base meningiomas, especially if there is a possibility of vascular compromise or brain stem manipulation. Both techniques are not markedly affected by inhalational anesthesia, with concentrations as high as 1 minimum alveolar concentration (MAC) used with minimal effects on the response.[20] Somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs) could be used to assess the integrity of the brain stem and spinal cord during meningioma resection. Because SSEPs and MEPs have cortical components to their measurement, volatile anesthetics produce a dose-dependent increase in latency due to an increase in central conduction time and a decrease in amplitude.[21] Satisfactory monitoring of both SSEPs and MEPs is possible with 0.5 to 0.75 MAC of isoflurane, desflurane, or sevoflurane. The effect of volatile agents on SSEPs and MEPs is compounded by N_2O, which is usually not used as an anesthetic for these procedures. The newer volatile agents, desflurane and sevoflurane, affect these monitoring techniques in a fashion similar to isoflurane but can be used at slightly higher concentrations. A continuous infusion of low-dose propofol with opioids coupled with low concentrations of background inhalation anesthetics is ideal and recommended for SSEP and MEP monitoring during resection of spinal cord and infratentorial meningiomas.

After completion of the surgical procedure, the decision to extubate is predicated on the patient's ability to

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follow commands, ventilate, and maintain oxygenation. Hemodynamic stability, blood loss, and fluid replacement also play a role in the decision to extubate. Recovery from anesthesia is expedited by the use of short-acting, low-solubility inhalational agents and infusions of short- or intermediate-acting opioids. Nausea, vomiting, and pain are important problems that must be treated in the immediate postoperative period. The incidence and magnitude of pain are not well characterized after craniotomy but are different depending on approach to the tumor and the anatomical structures disrupted. Pain after spinal procedures may be severe, depending on trauma to muscle and the number of laminectomies performed. There is evidence that neurosurgical patients receive inadequate analgesia from currently practiced regimens.[22] Although no ideal analgesic exists, many practitioners have begun the use of patient-controlled analgesia with potent opioids. This produces a method to titrate analgesia, allows patients to control their pain, and may alleviate some of the physiological stress associated with pain.

Nausea and vomiting could be severe, depending on opioid administration or the area of surgery. Evidence suggests that a higher incidence of vomiting may be associated with infratentorial and skull base approaches rather than supratentorial or spinal cord resection of meningiomas.[23] Different antiemetic strategies may be employed to provide relief from emesis after these procedures. Droperidol, a dopaminergic antagonist, has been used with success, but the possibility of extrapyramidal side effects and its synergism with opioids to increase sedation may limit its use. Ondansetron and other serotonin receptor antagonists, along with dexamethasone, have been used with some success to reduce nausea and vomiting.[24] A multimodal approach to the treatment of nausea and vomiting after these surgeries may be more effective than single-therapy regimens.

In conclusion, the perioperative management of patients undergoing intracranial or spinal cord meningioma resection must account for the physical status of the patient and their ability to undergo the surgical procedure. The risks associated with anesthesia, blood loss, and surgical trauma are weighed against the patient's estimated physical ability to successfully survive the surgery with minimal associated morbidity. Once the estimate of surgical risk is determined, the anesthesiologist must develop the anesthetic technique that will provide optimum conditions for intraoperative monitoring, a stable surgical field, and a hemodynamically stable patient. The anesthesiologist can improve postoperative outcomes and reduce morbidity by knowing information concerning tumor size, characteristics, surgical approach, and involved neurological structures. Successful perioperative evaluation and management of surgical patients undergoing meningioma resection require teamwork and communication between the surgeon, anesthesiologist, primary caregiver, and consultants to produce an optimal outcome with low morbidity. The preoperative assessment and estimate of operative risk and the patient's acceptance of that risk play a vital role in the informed consent process and are key to a successful outcome.

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Chapter 13

Modern Imaging Techniques for Meningiomas

William P. Dillon and Alina Uzelac

Introduction

Meningioma, a central nervous system tumor characterized by indolent growth, is the most common extraaxial lesion encountered in neuroimaging. The high frequency of incidental diagnosis during neuroimaging for other indications reflects the fact that up to 2% of autopsies reveal a meningioma.[1]

Historically, meningiomas were characterized by plain roentgenograms and conventional diagnostic angiography, but the introduction of computed tomography (CT) and magnetic resonance imaging (MRI) dramatically improved detection and accuracy of diagnosis. Continuous refinements in neuroimaging technology have led to the exquisitely detailed contemporary images with which we are familiar today.

Recent years have witnessed the development of advanced imaging techniques, such as MR spectroscopy, MR perfusion, indium-111-octreotide scintigraphy, and positron emission tomography (PET), which at times aid in the diagnosis of meningiomas.

Although these studies are becoming more standardized and their usefulness in certain clinical scenarios better defined, these newer techniques remain peripheral at present and serve only as adjuncts to CT and routine MRI sequences.

Computed Tomography

Although MRI is the imaging study of choice for evaluation of suspected meningioma or in the context of known or highly suspected pathology, CT is highly accessible and indicated for rapid screening in urgent settings. As such, many meningiomas are first encountered on CT scans obtained for different reasons. CT has a place in the diagnosis of meningioma because it is superior in demonstrating the effects of this neoplasm on adjacent bone, specifically osseous destruction or hyperostosis, and is more sensitive in detecting psammomatous calcifications in the tumor (seen grossly in ~25% of meningiomas).

Benign meningiomas typically appear as rounded or elongated extraaxial masses that demonstrate a broad attachment to the dura. On CT, they are usually isodense but can occasionally be hyperdense or slightly hypodense compared with cerebrum.

Their extraaxial nature is suggested by a sharp interface with displaced brain parenchyma, the presence of a cerebrospinal fluid attenuation cleft (**Fig. 13.1A**), and intense enhancement. Meningiomas exhibit homogeneous attenuation before and after administration of contrast material but can show some heterogeneity depending on the consistency of tumor (i.e., the presence of calcium, fat, tumor necrosis).

Hyperostosis of adjacent skull is highly suggestive of benign meningioma and is best demonstrated by CT, windowed on bone algorithm, as cortical thickening and hyperdensity (**Fig. 13.1B**). Hyperostosis typically indicates infiltration of bone by meningioma. In one study, meningioma infiltration of radiographically shown hyperostotic bone was identified histologically in 25 of 26 patients.[2] Hyperostosis is usually more difficult, but not impossible, to ascertain by MRI.

The World Health Organization (WHO) classifies meningiomas into grade I (benign), atypical (grade II), and malignant (grade III and IV) stages. The latter often, but not always, have an aggressive behavior, invade bone and brain, and tend not to cause hyperostosis but, rather, frank bone destruction. Malignant meningiomas can metastasize as well as spread along the leptomeninges.

Dural metastases or other malignancies (i.e., plasmacytomas, leiomyomas) may have an appearance similar to meningioma, but associated lytic calvarial or skull base lesions may help differentiate these lesions from meningioma.

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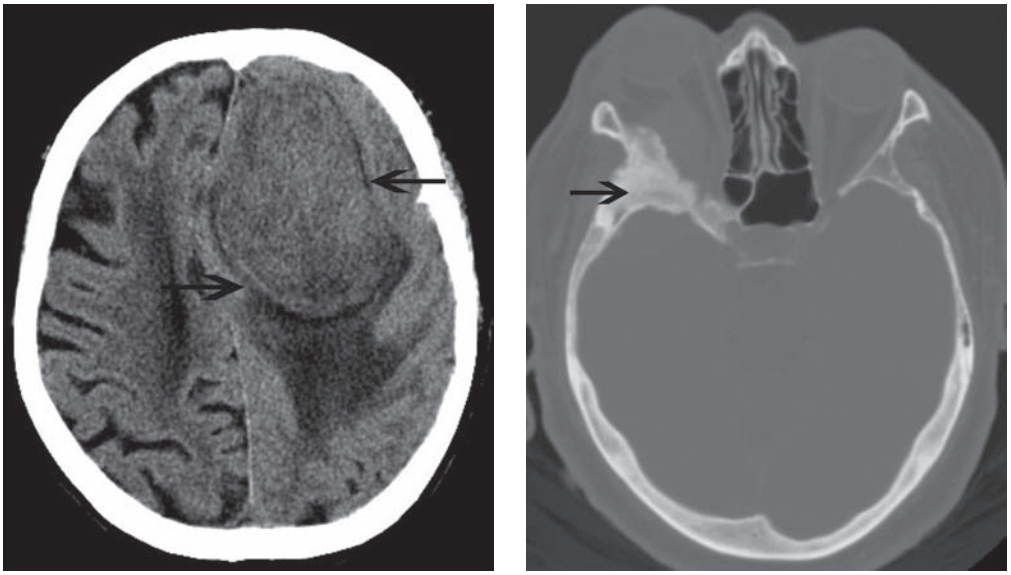


Fig. 13.1 **(A)** A thin rim of low attenuation represents cerebrospinal fluid (CSF) trapped around a large left frontal meningioma. This is a feature indicating the extraaxial nature of the tumor. Note the vasogenic edema in adjacent brain parenchyma (low density) outside the CSF cleft (*arrows*). **(B)** Meningioma centered in the right sphenoid wing with extension into the right lateral orbit. There is resultant deviation of the optic nerve and muscles with proptosis of the globe. There is associated hyperostosis of the sphenoid wing, lateral orbital wall, and squamosal temporal bone.

Calcifications are also typical of meningioma but not pathognomonic because other extraaxial neoplasms, such as chondromas, may also contain calcifications.[3] The morphology and volume of calcifications are varied. Parasellar thrombosed aneurysms and schwannomas arising from the cavernous sinus can masquerade as meningioma (**Fig. 13.2**), so extraaxial skull base masses should be carefully evaluated before assigning a diagnosis of meningioma.

◆ Magnetic Resonance Imaging

Common Imaging Features of Meningiomas on Magnetic Resonance Imaging

Approximately 85 to 90% of meningiomas have typical features, including an extraaxial mass with signal intensity isointense to cortex on T1 and T2 MRI sequences, avid homogeneous enhancement following administration of gadolinium contrast, and an enhancing "dural tail" (**Fig. 13.3A**), which reflects neoplastic dural infiltration or reactive vascularity (or both) draining into the adjacent dura. An eccentric core of lower signal intensity is often seen and indicates the vascular pedicle entering at the original nidus of the meningioma. Low signal intensity within the tumor may often be due to calcification or to vascular flow voids, a distinction sometimes difficult to make, unless the morphology is clearly that of branching vessels.

Meningiomas can be spherical or elongated (en plaque), multiple, and often take origin from a dural sinus, a feature important for surgical planning. These tumors also tend not to respect the dural boundary and may extend on both sides of the falx and the tentorium, which is a distinctive feature not typical of other neoplasms (**Fig. 13.3B**).

Hyperostosis of bone can also be appreciated on MRI, particularly when florid, as seen with en plaque meningiomas (**Fig. 13.4A**). Hyperostosis appears as an area of thicker cortical bone with low signal intensity on T1- and T2-weighted images. The hyperostosis (also **Fig. 13.4B**) usually reflects osseous neoplastic infiltration and is best removed at the time of surgery, if possible.

Narrowing of engulfed arteries, so-called encasement, is also a common feature, especially of parasellar meningiomas, which often encase and narrow the supraclinoid internal carotid artery (**Fig. 13.5**). The encasement, on occasion, can lead to cerebral ischemia.

Although most benign meningiomas are innocuous from the standpoint of metastatic potential, they may result in serious complications secondary to dural sinus invasion (**Fig. 13.6**) (with or without thrombosis), narrowing and thrombosis of significant arterial structures, and compression of cranial nerves and other important neural structures, such as the brain stem. The use of MR vascular imaging or conventional angiography for preoperative detailing of the vascular structures adjacent to these tumors before surgical removal is often helpful.[4]

Edema associated with meningioma is thought to be vasogenic in origin and probably related to tumor secretion of vascular endothelial growth factor (VEGF), rather than a result of direct mass effect on adjacent brain or venous invasion causing vascular congestion.[5] The presence of intraaxial edema is said to predict an increased potential for recurrence.[6,7] **Figure 13.7** demonstrates a meningioma

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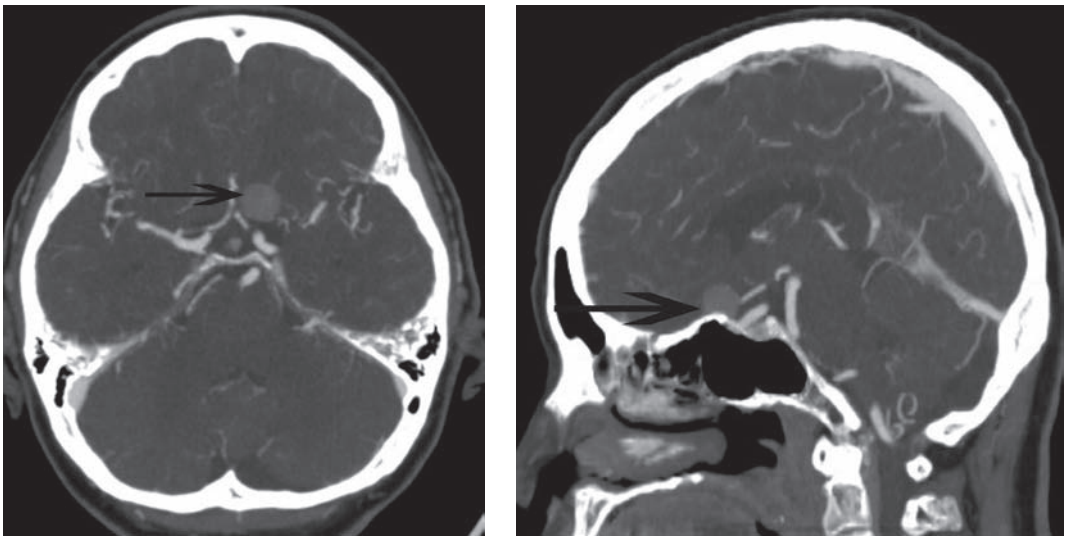


Fig. 13.2 **(A)** Suprasellar mass lesion adjacent to the left anterior clinoid process that on axial computed tomographic angiography (CTA) could be mistaken for an aneurysm. **(B)** Reformatted sagittal image demonstrates no true connection with artery. Diagnosis was meningioma.

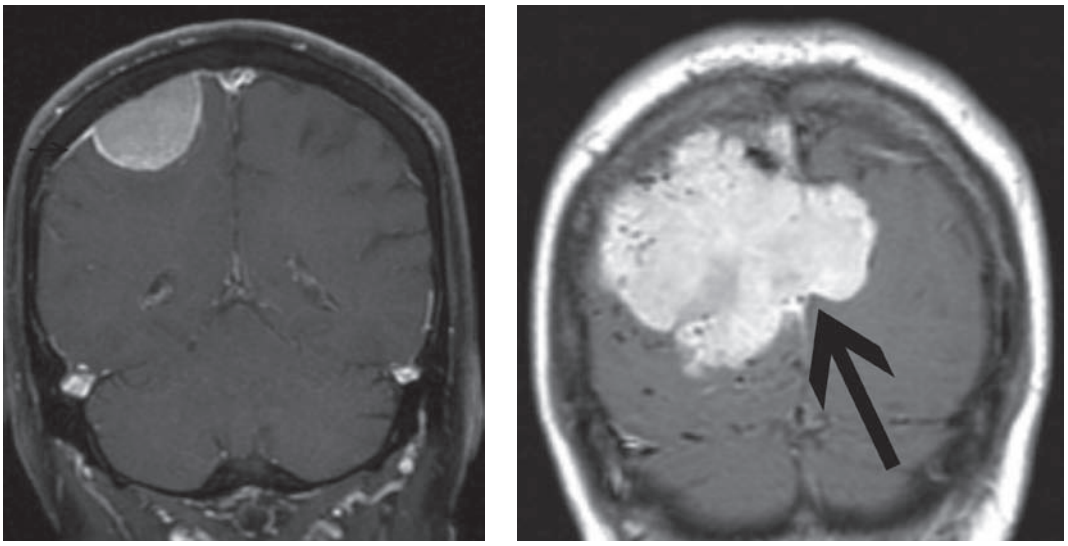
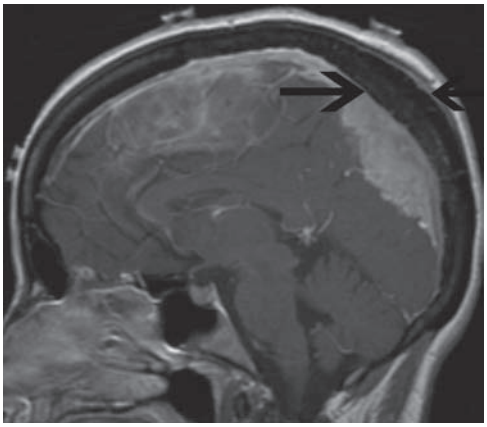
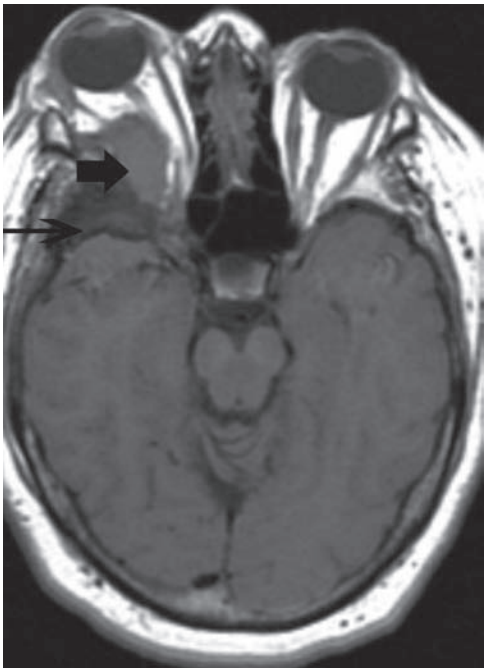


Fig. 13.3 Typical appearance of meningioma on magnetic resonance imaging (MRI). **(A)** Coronal postcontrast T1-weighted MRI demonstrate avid homogeneous enhancement and a dural tail of a convexity meningioma arising from the dura overlying the right parietal lobe. Note that the sagittal sinus is separated from the meningioma by normal brain. **(B)** A large falcine meningioma does not respect the dural boundary and extends on both sides of the falx.

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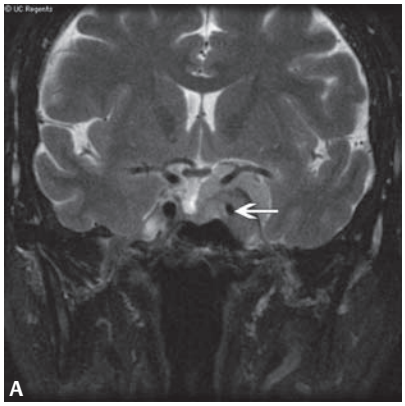


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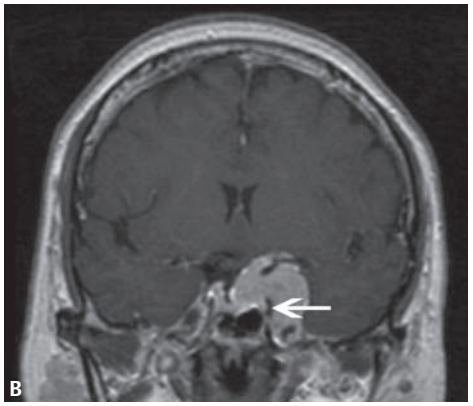


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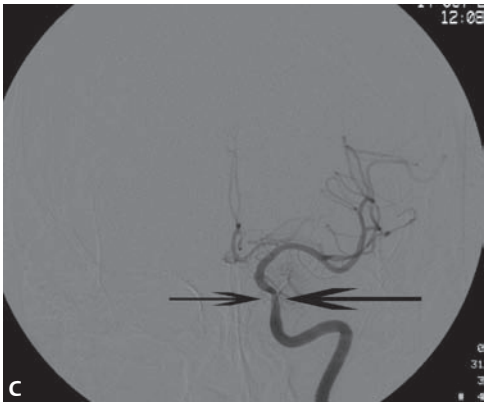
Fig. 13.4 **(A)** Gadolinium-enhanced sagittal T1-weighted magnetic resonance image obtained for surgical navigation (note fiducials on scalp) demonstrates a posterior parietal convexity en plaque parasagittal meningioma causing extensive calvarial hyperostosis. **(B)** Right sphenoid wing hyperostosis (*thin arrow*) of a sphenoid meningioma with intraorbital extension (*thick arrow*).



A

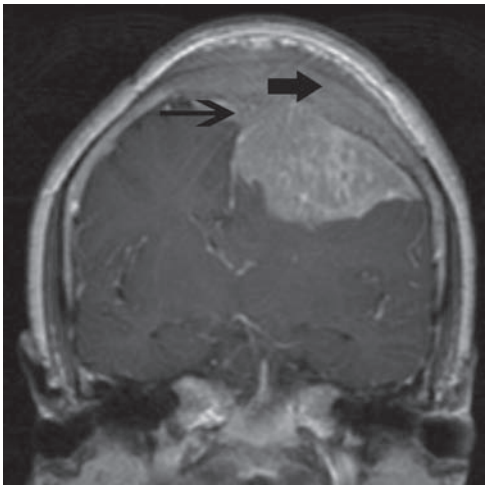


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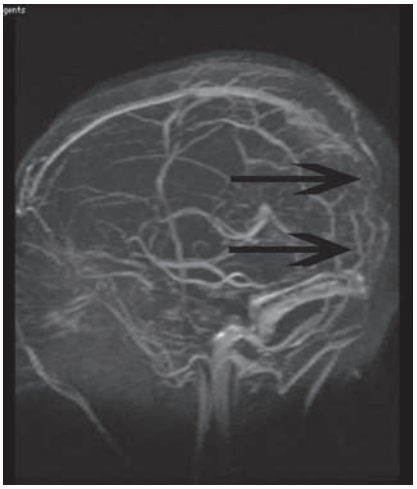


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Fig. 13.5 **(A)** Coronal T2, **(B)** T1 postgadolinium, and **(C)** conventional angiogram demonstrate a left WHO grade 1 parasellar cavernous sinus meningioma that focally narrows the cavernous and supraclinoid segments of the left internal carotid artery.



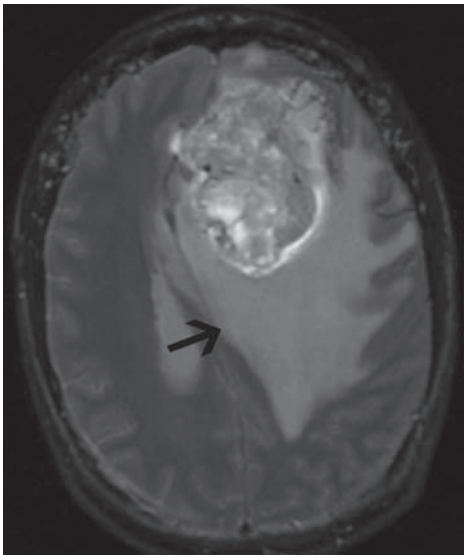
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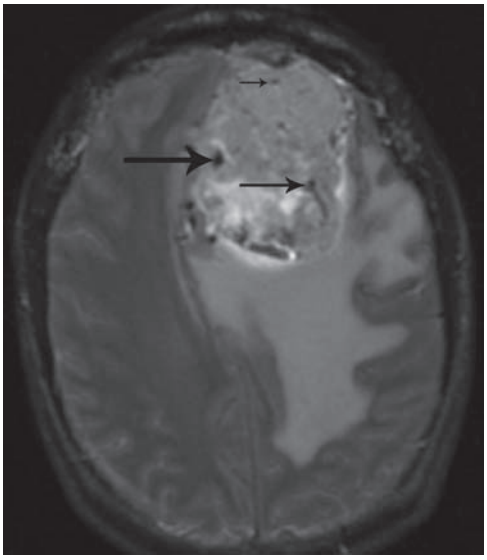
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Fig. 13.6 **(A)** Coronal postgadolinium magnetic resonance image demonstrates an atypical parasagittal meningioma with sagittal sinus invasion. This tumor crosses the midline and infiltrates the calvarium. **(B)** Magnetic resonance venogram demonstrates absent/decreased flow in the sagittal sinus (*arrows*).

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A



B

Fig. 13.7 Twenty-six-year-old male with frontal meningioma. Axial T2-weighted images demonstrate a hypervascular left frontal meningioma with **(A)** surrounding vasogenic edema (*arrow*) and necrosis. **(B)** Vascular flow voids are noted within the tumor (*arrows*). Although this degree of edema, necrosis, and hypervascularity might suggest an aggressive or malignant meningioma, the pathological diagnosis was WHO grade I meningioma.

associated with extensive vasogenic edema and low T2 signal serpentine structures within/high vascularity.

"Cystic" meningiomas, named for focal areas of cerebrospinal fluid signal intensity on MRI (or attenuation on CT), are not truly cystic in their entirety (**Fig. 13.8**). These collections may result from adjacent trapped CSF collections or intratumoral cystic collections. Lipomatous changes within meningiomas are also uncommon and result from accumulation of lipid inside meningothelial cells rather than metaplasia.[8]

Intraventricular meningiomas arise from arachnoid cell nests within the choroid plexus (**Fig. 13.9**). These rare tumors represent only ~1 to 2% of all meningiomas[9] but are the most common atrial intraventricular neoplasms in adults.[10] These lesions can become quite large; yet, despite their size, they produce less mass effect and little edema because they reside within the ventricle.

Atypical and Malignant Meningiomas

Atypical (World Health Organization [WHO] grade II) and malignant (WHO grade III) meningiomas represent ~6% and 2%, respectively, of all meningiomas.[1] They are typically characterized by their aggressive and invasive nature and higher rates of recurrence. These tumors tend to penetrate the brain parenchyma through the perivascular subarachnoid spaces (**Fig. 13.10**) and may erode through the dura into skull and scalp (**Fig. 13.11**). Irregularity of the margin with brain, so-called mushrooming, has been suggested as a feature of malignant meningiomas, but in the authors' experience this is an unreliable marker.

Malignant meningiomas' aggressiveness and tendency to recur are linked and directly proportional to the degree of adjacent skull osteolysis, extent of brain parenchymal invasion, and volume of pial-cortical supply (as opposed to dural-meningeal supply).[11]

The recurrence rate of grossly resected atypical/malignant meningiomas is reported as high as 48% at 10 years.[12] Malignant meningiomas also have a higher tendency than their benign counterparts to disseminate through the cerebrospinal fluid or hematogenously (to lungs and other organs).[13]

Advanced Imaging

Diffusion Magnetic Resonance Imaging

With diffusion-weighted imaging (DWI), each image voxel (three dimensional) has an image intensity that reflects a single best measurement of the rate of microscopic water motion at that location. Reduced water diffusivity (**Fig. 13.12A**) has been correlated with more aggressive tumor behavior and is seen with atypical/malignant meningiomas, high cellular density, and recurrence.[14]

The apparent diffusion coefficient (ADC) map, a calculated image from the DWI image, shows the average diffusion that water molecules have in each voxel. This parameter is calculated from all the diffusion-weighted images. A decrease in ADC values (**Fig. 13.12B**) at follow-up of a benign meningioma should raise suspicion for dedifferentiation to higher tumor grade.

Perfusion

Perfusion MRI can provide useful information on the vascular supply of meningiomas, which is only implied from conventional MRI. Perfusion curves provide additional prognostic information by helping distinguish between benign and atypical/malignant meningiomas.

Perfusion imaging curves are obtained by using echo-planar contrast-enhanced T2-weighted sequences rapidly performed before, during, and after the bolus infusion of gadolinium contrast material. The curves reflect the

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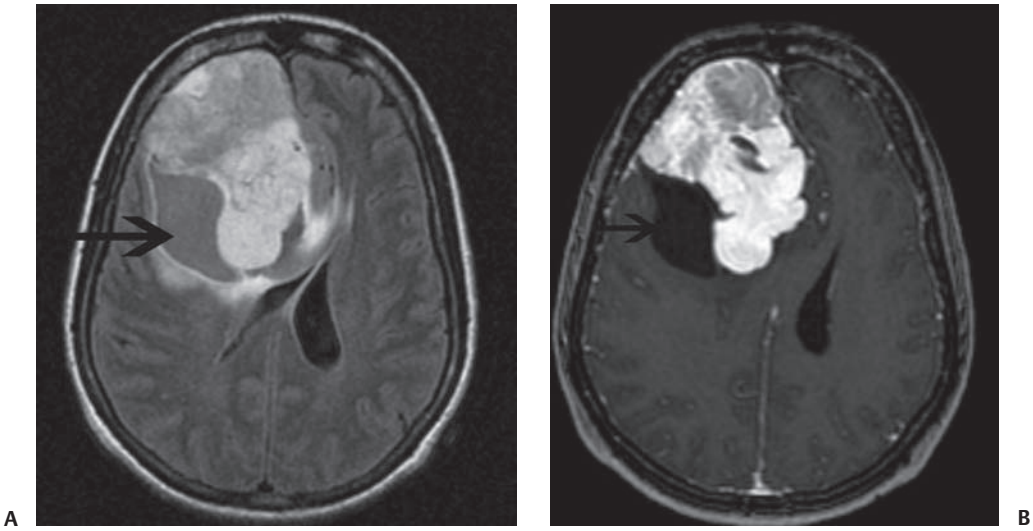


Fig. 13.8 **(A)** Axial fluid-attenuated inversion recovery and **(B)** postgadolinium T1-weighted images. Preoperative magnetic resonance imaging demonstrates a complex right frontal lobe extraaxial mass with intense enhancement, typical of meningioma. A peripheral nonenhancing cerebrospinal fluid intensity cyst is also present (*arrows*). The lack of peripheral enhancement suggests that the cyst is of arachnoidal origin, which can occur adjacent to any extraaxial tumor, including esthesioneuroblastoma and vestibular schwannoma, as well as with meningiomas. Peritumoral arachnoidal cysts can sometimes be larger than the meningioma itself and be the primary cause of mass effect and symptoms.

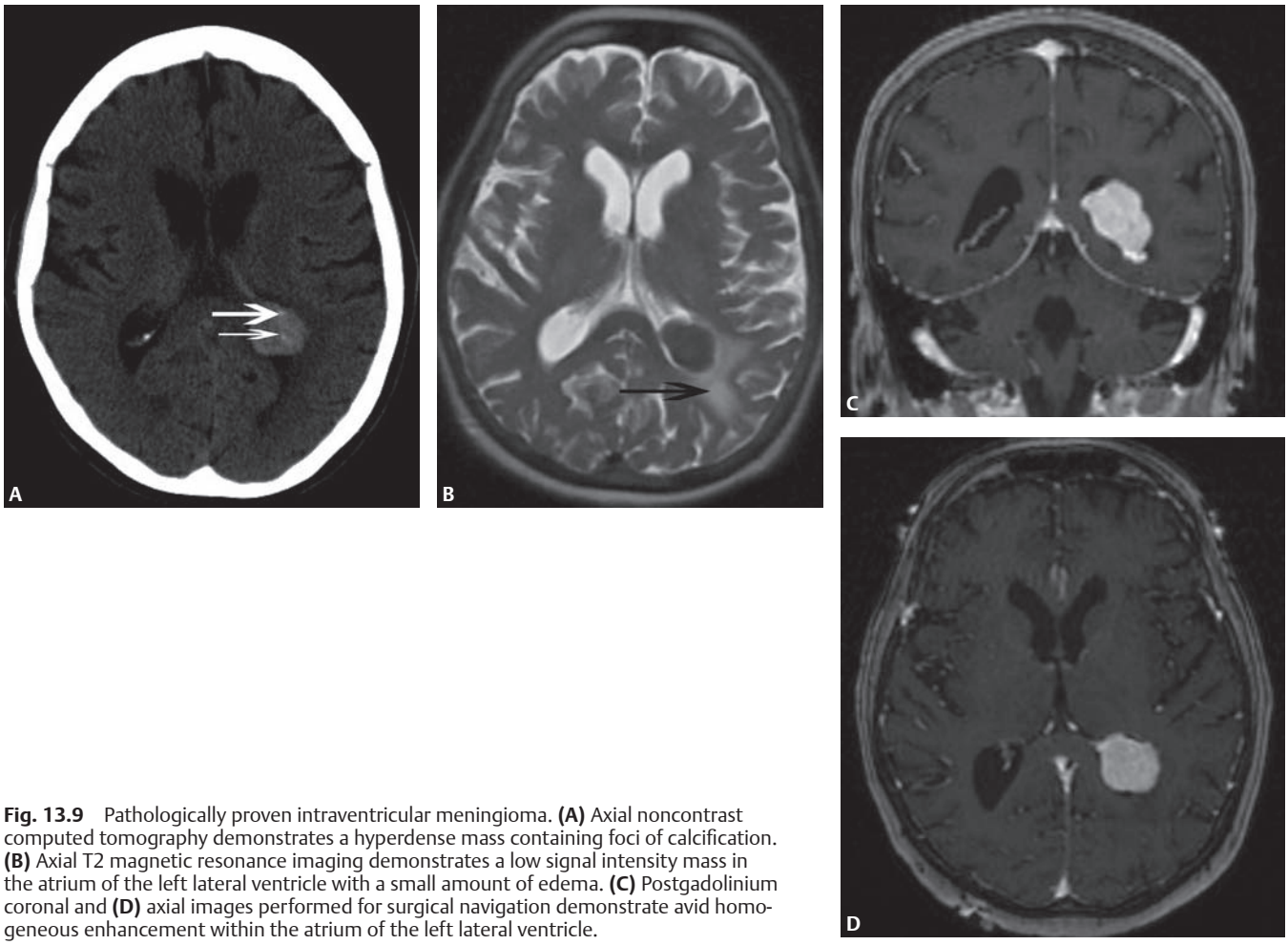


Fig. 13.9 Pathologically proven intraventricular meningioma. **(A)** Axial noncontrast computed tomography demonstrates a hyperdense mass containing foci of calcification. **(B)** Axial T2 magnetic resonance imaging demonstrates a low signal intensity mass in the atrium of the left lateral ventricle with a small amount of edema. **(C)** Postgadolinium coronal and **(D)** axial images performed for surgical navigation demonstrate avid homogeneous enhancement within the atrium of the left lateral ventricle.

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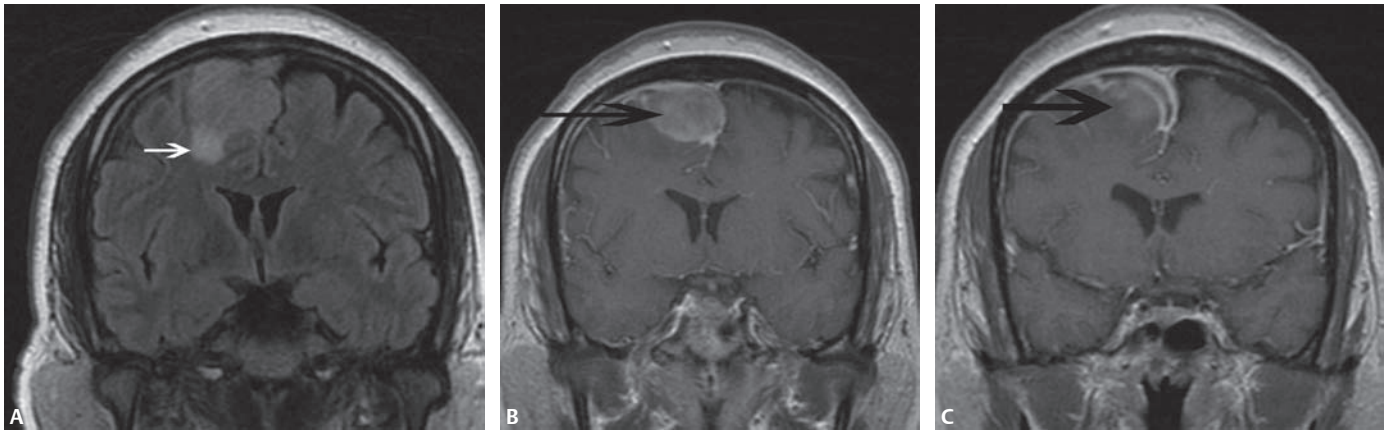


Fig. 13.10 Malignant meningioma. **(A)** Coronal T2 fluid-attenuated inversion recovery and **(B,C)** coronal T1 postgadolinium demonstrate an irregular parasagittal mass with a focal area of increased T2 intensity and postcontrast enhancement within the adjacent brain parenchyma and leptomeninges, consistent with brain invasion.

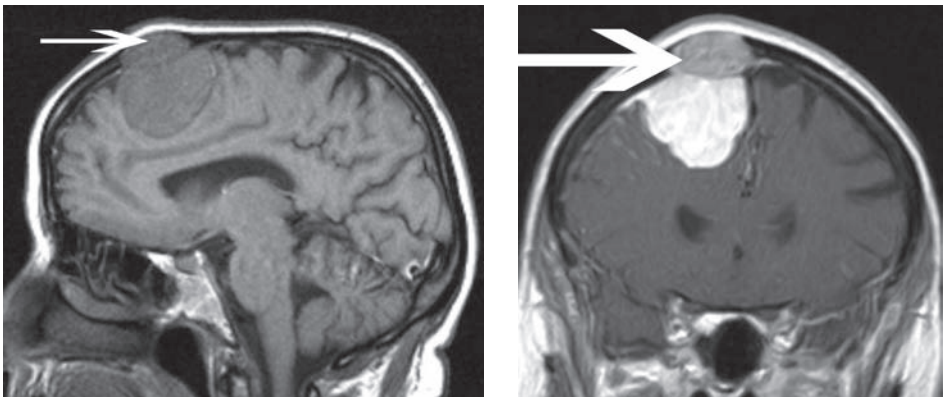


Fig. 13.11 **(A)** Sagittal T1 noncontrast and **(B)** coronal T1 magnetic resonance image after gadolinium administration demonstrate invasion of the calvarium by this atypical (World Health Organization grade II) meningioma.

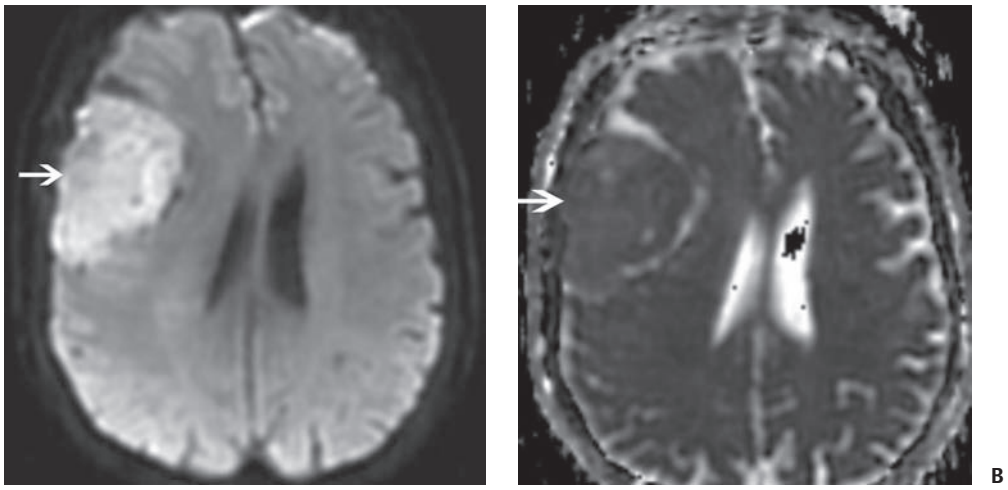


Fig. 13.12 Reduced diffusion is seen within this right frontal convexity atypical meningioma. **(A)** High signal on the diffusion-weighted image, with **(B) a** corresponding low signal on the apparent diffusion coefficient maps.

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permeability between intravascular and extravascular compartments, as well as cerebral blood volume. The relative cerebral blood volume (rCBV) is measured within a tumor compared with the contralateral normal white matter and is displayed on color maps.[15]

Benign meningiomas typically derive their blood supply from the external carotid via dural branches. These vessels do not contain a blood–brain barrier and are thus quite permeable to gadolinium, which is reflected by a curve with little or no return to baseline following infusion (**Fig. 13.13B**). As the meningioma enlarges, it may parasitize pial branches from the brain parenchyma, which do contain a blood–brain barrier. The perfusion scans will show an elevated cerebral blood volume with intensities that return to baseline signal levels, reflecting an intact blood–brain barrier of the internal carotid artery supply, as seen in **Fig. 13.13D**.

Spectroscopy

MR spectroscopy (MRS) has limited utility in the diagnosis of meningiomas, given that the majority of meningiomas do not represent a dilemma for the radiologist. MRS has been reported to aid in the diagnosis of meningioma and in the detection of brain parenchymal invasion by demonstrating a decreased N-acetyl aspartate (NAA) peak, which is a marker of neuronal damage, and an elevated choline peak usually seen with cellular division of neoplasms.[16] That said, little if any use is derived from MRS in the case of meningioma diagnosis or follow-up after therapy.

Nuclear Scintigraphy

Limited and occasional benefit has been obtained from the use of nuclear scintigraphy (positron emission tomography and indium-111 octreotide), but these tests lack specificity and have value only as adjuncts to CT and routine MRI. Meningiomas have high somatostatin receptor density allowing for the use of octreotide brain scintigraphy to assess extent of disease and to help distinguish between postsurgical changes and residual disease.[17]

Conventional Angiography and Endovascular Embolization

Conventional angiography is indicated in the diagnostic workup of meningiomas only when the diagnosis is in question and most often in association with preoperative endovascular embolization. Angiography, designed to minimize blood loss intraoperatively, is sometimes performed for concomitant evaluation of venous sinus patency, venous collateral supply, and arterial supply. With the increased use of preoperative embolization, the subsequent MRI changes and treatment complications[18] (i.e., hemorrhage [**Fig. 13.14**] and necrosis [**Fig. 13.15**]) sometimes present a confusing imaging picture for a radiologist who is unaware of the prior embolization procedure.

MRI changes that occur after embolization of meningiomas usually include a decrease in gadolinium contrast enhancement (**Fig. 13.15B**), reduced diffusion of the devascularized segment of the tumor (**Fig. 13.15C,D**), and a decrease in precontrast T1 and T2 signal. Focal areas of precontrast T1 and T2 shortening can be seen due to retained iodinated contrast used during embolization.

◆ Miscellaneous

Radiation-Induced Meningioma

Meningioma is the most common tumor induced by ionizing radiation. Prior radiation to the brain for other tumors or for skin conditions can give rise to meningiomas 5 to 10 years following exposure. Tumors may be single or multiple and because of the latency period may not be easily recognized as associated with prior radiation treatment (**Fig. 13.16**). The history of prior radiation suggests the diagnosis. Signs of vasculopathy or other radiation-related changes involving adjacent brain also strengthen suspicion, such as postradiation cavernous malformations, moyamoya disease, port-related small vessel changes, aneurysms, or arteriopathy-related microhemorrhages.[19,20]

Neurofibromatosis Type 2

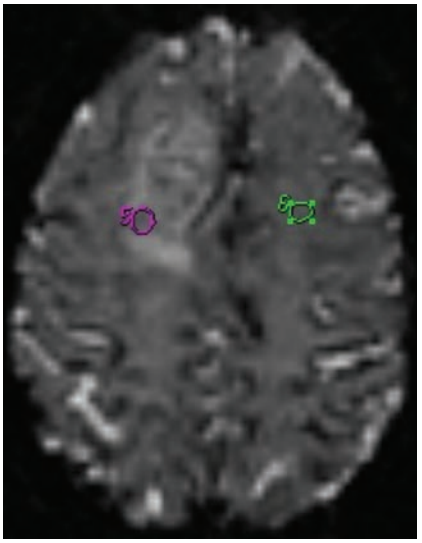
Approximately 1% of meningioma patients have neurofibromatosis 2 (NF2). These patients are at risk for meningiomas and the more frequent schwannomas. Of those with NF2 and meningioma, a high percentage (more than 50%) will have multifocal meningiomas (**Fig. 13.17**). Patients with early childhood NF2 presentation tend to be diagnosed as a result of a symptomatic meningioma, which may occur before the appearance of vestibular schwannomas.[21] In these patients, a more aggressive meningioma histology has been reported,[22] and close imaging follow-up is recommended.

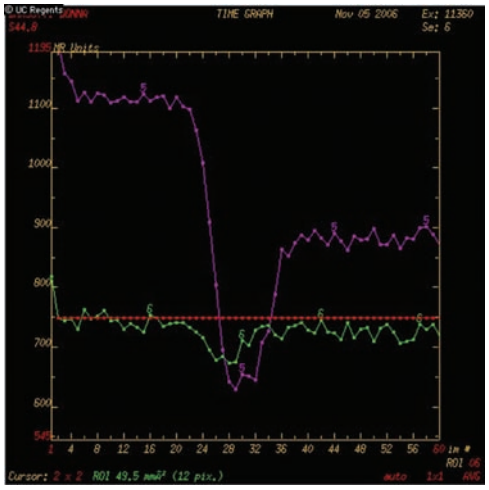
Optic Nerve Sheath Meningioma

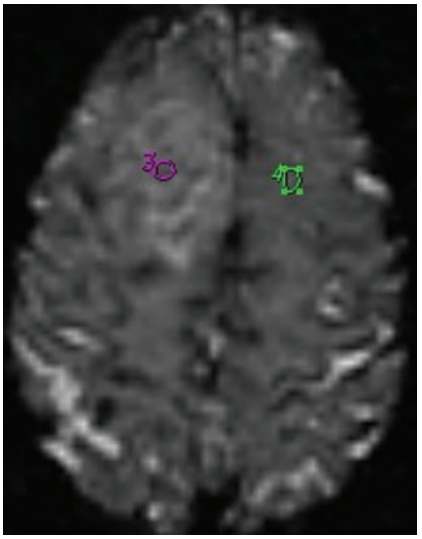
Although representing only ~1 to 2% of meningiomas, optic nerve sheath meningiomas deserve mention due to their significant potential for visual loss[23] and their characteristic imaging appearance. Primary optic nerve meningiomas most frequently arise from the intraorbital nerve sheath surrounding the optic nerve.

Avid circumferential contrast enhancement results in the typical “tram-tracking” appearance on axial T1 gadolinium-enhanced MRI scans (**Fig. 13.18**). Care must be taken to exclude optic canal meningioma in patients with visual loss because these can easily be overlooked. Indeed, many such patients have a delay in diagnosis or misdiagnosis of optic neuritis, which can lead to delay in treatment and permanent visual loss.[24]

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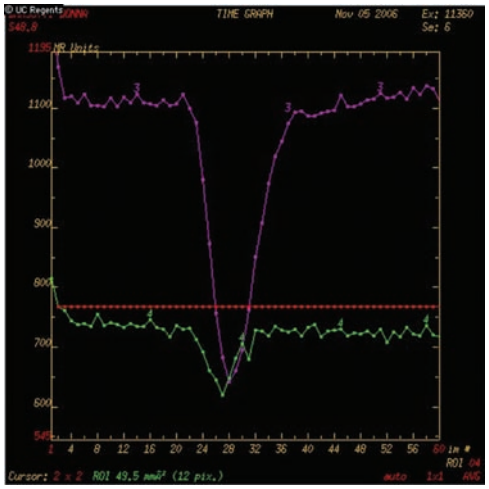
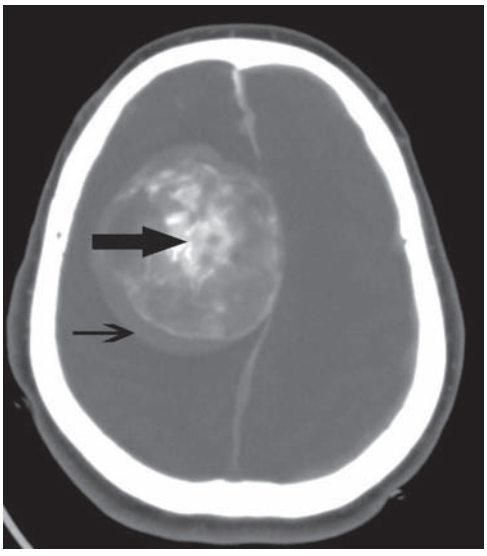


Fig. 13.13 Magnetic resonance perfusion of a right parafalcine meningioma demonstrates significantly elevated relative cerebral blood volume (area under the curve is large) (*purple curve* in **[B]**), compared with contralateral normal centrum semiovale (*green curve*). **(A)** The first region of interest demonstrates less than 50% return to **(B)** baseline typical of the lack of blood–brain barrier of the external carotid artery, whereas **(C)** a different region of interest shows a curve demonstrating complete return to baseline more typical of **(D)** internal carotid artery supply.



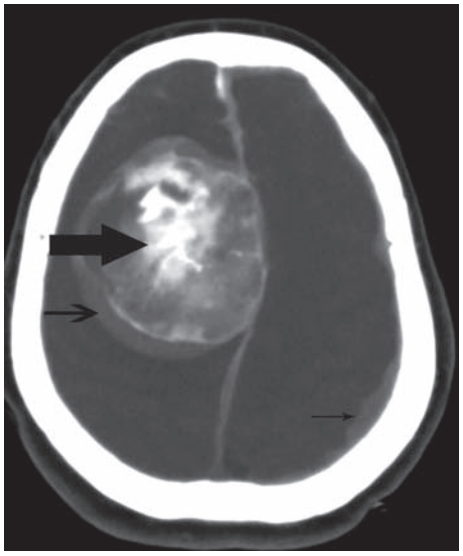


Fig. 13.14 **(A)** Noncontrast computed tomography of a right frontal lobe meningioma status postembolization demonstrates high central density (*large arrow*) radiating to the periphery, compatible with contrast material retained in slow-flowing vessels. Uniform high attenuation surrounding the tumor (*small arrow*) is compatible with hemorrhage and extravasated contrast. **(B)** Additional left frontal, falcine, and left parietal subdural (*smallest arrow*) and bifrontal subarachnoid hemorrhage is seen.

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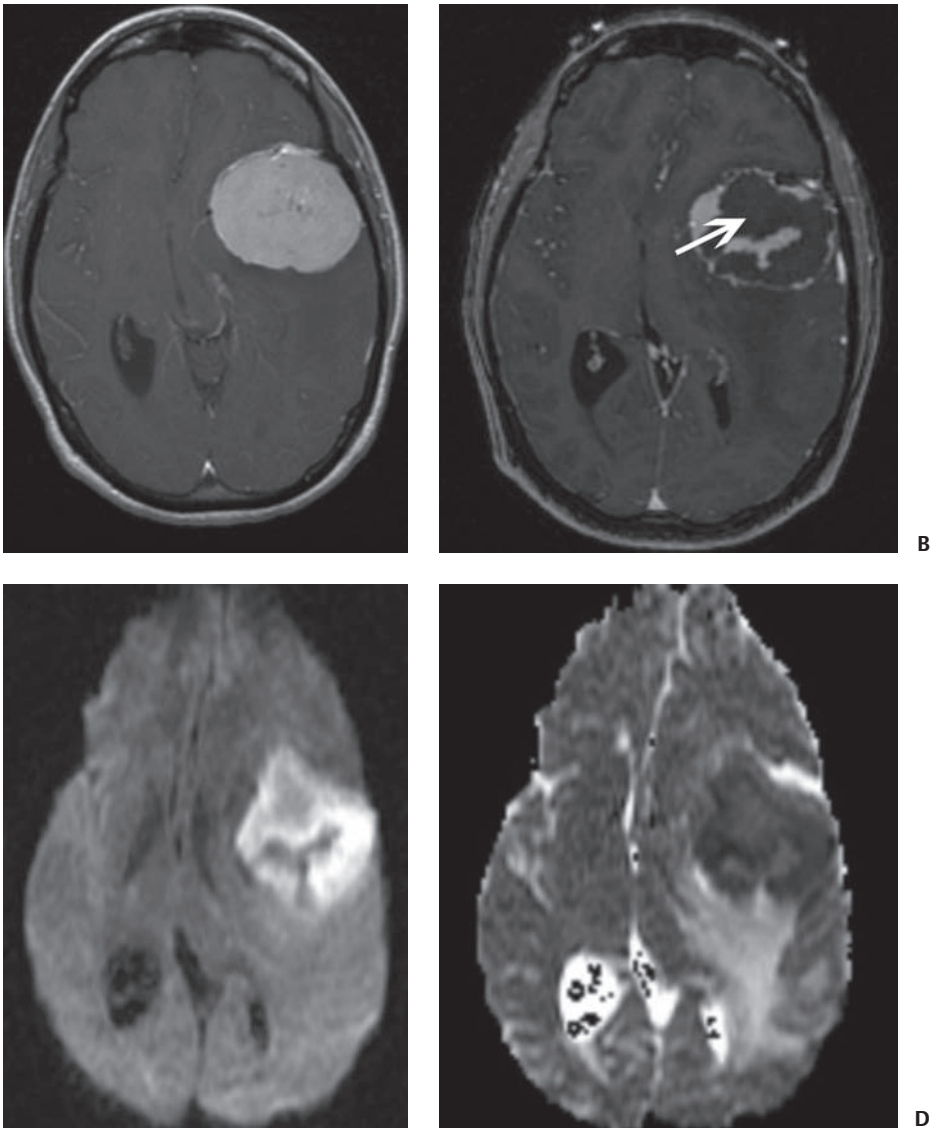


Fig. 13.15 Pre- and postembolization appearance of meningioma. **(A)** Postgadolinium axial T1-weighted magnetic resonance imaging (MRI) demonstrates a homogeneously enhancing left frontal meningioma. **(B)** Postcontrast T1-weighted MRI obtained following embolization reveals areas of reduced contrast enhancement, which correspond to **(C)** decreased diffusion (high signal on diffusion-weighted imaging) and **(D)** low apparent diffusion coefficient (ADC) map signal, suggesting necrosis. The latter are common features accompanying embolization but can be misleading for radiologists or surgeons if they are not provided the history of the embolization procedure.

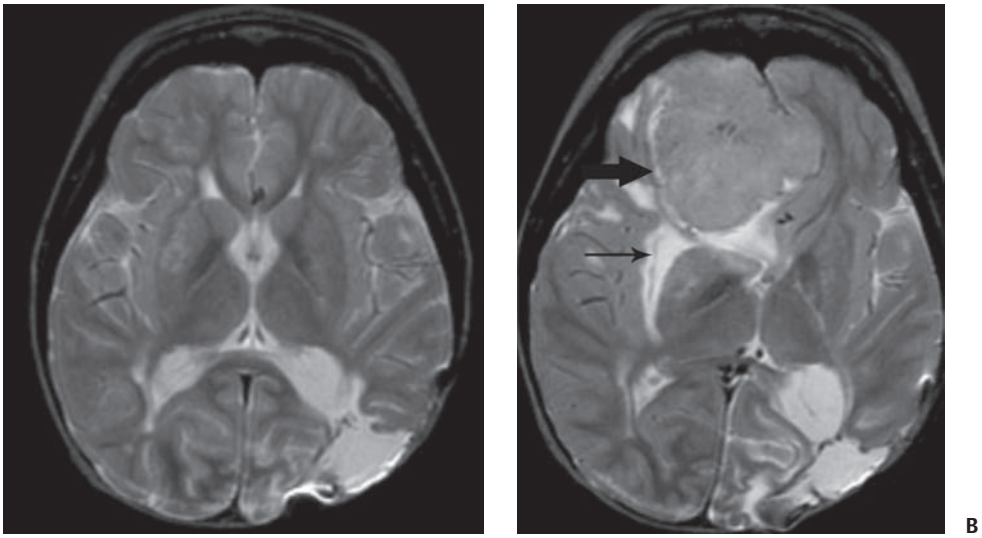
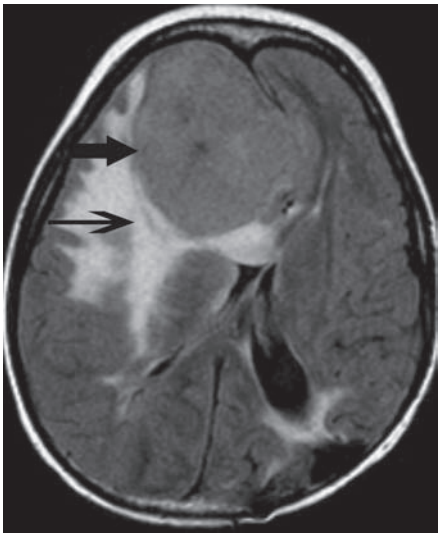
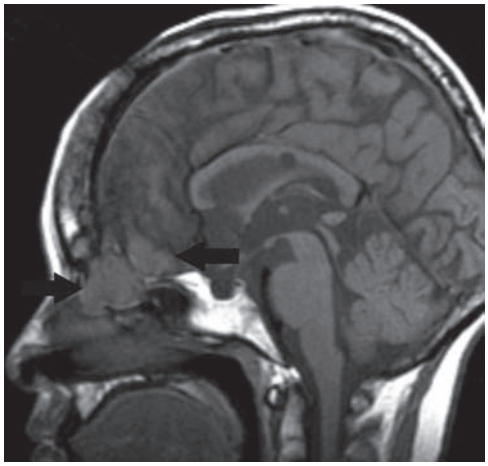


Fig. 13.16 Radiation-induced atypical meningioma from prior treatment of a primitive neuroectodermal tumor (PNET) tumor 8 years prior. **(A)** Baseline scan following prior resection of a PNET in the left parietal lobe. **(B,C)** Right frontal dural-based mass with prominent central vascular pedicle and adjacent vasogenic edema (*small arrow*) has developed over 15 months since the baseline scan in **(A)**. *(continued)*

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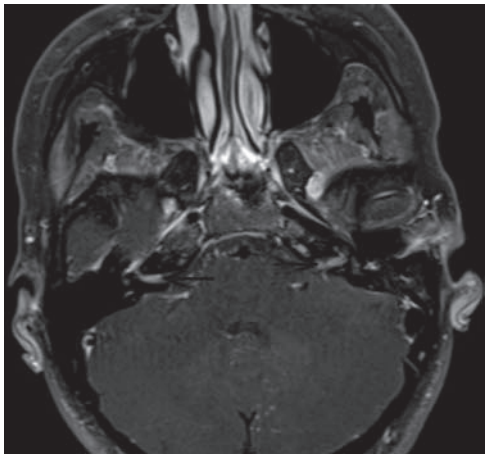


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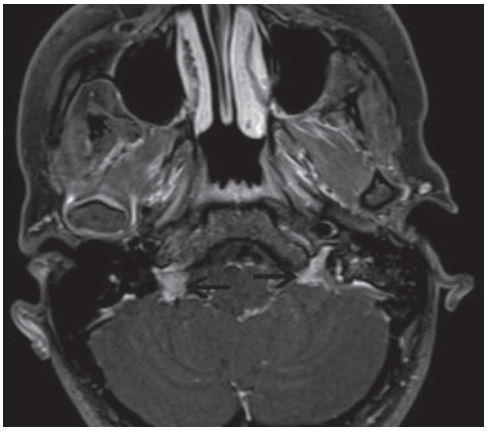
Fig. 13.16 *(continued)* **(D)** A sagittal noncontrast T1 scan performed another 13 months later demonstrates development of another meningioma in the ethmoid sinus growing through the cribriform plate (between *arrows*).



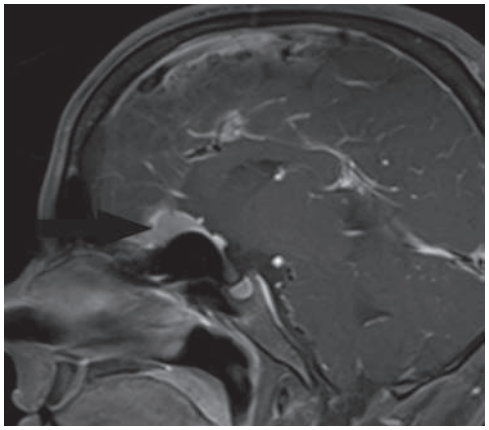
A



B



C



D

Fig. 13.17 A 20-year-old patient with neurofibromatosis 2 (NF2) with multiple meningiomas and schwannomas. **(A)** Right intraorbital third nerve schwannoma (*large arrow*) and bilateral tentorial meningiomas (*small arrows*), **(B)** bilateral vestibular schwannomas (*arrows*), **(C)** bilateral jugular foramen meningiomas (*arrows*), and **(D)** planum sphenoidale meningioma.

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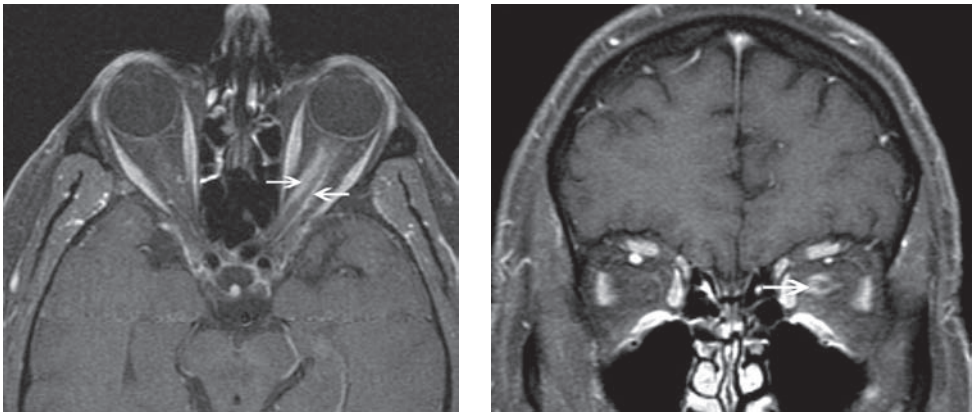


Fig. 13.18 **(A)** Axial and **(B)** coronal T1 gadolinium-enhanced magnetic resonance imaging scans demonstrate the classic appearance of a meningioma surrounding the left optic nerve with circumferential enhancement.

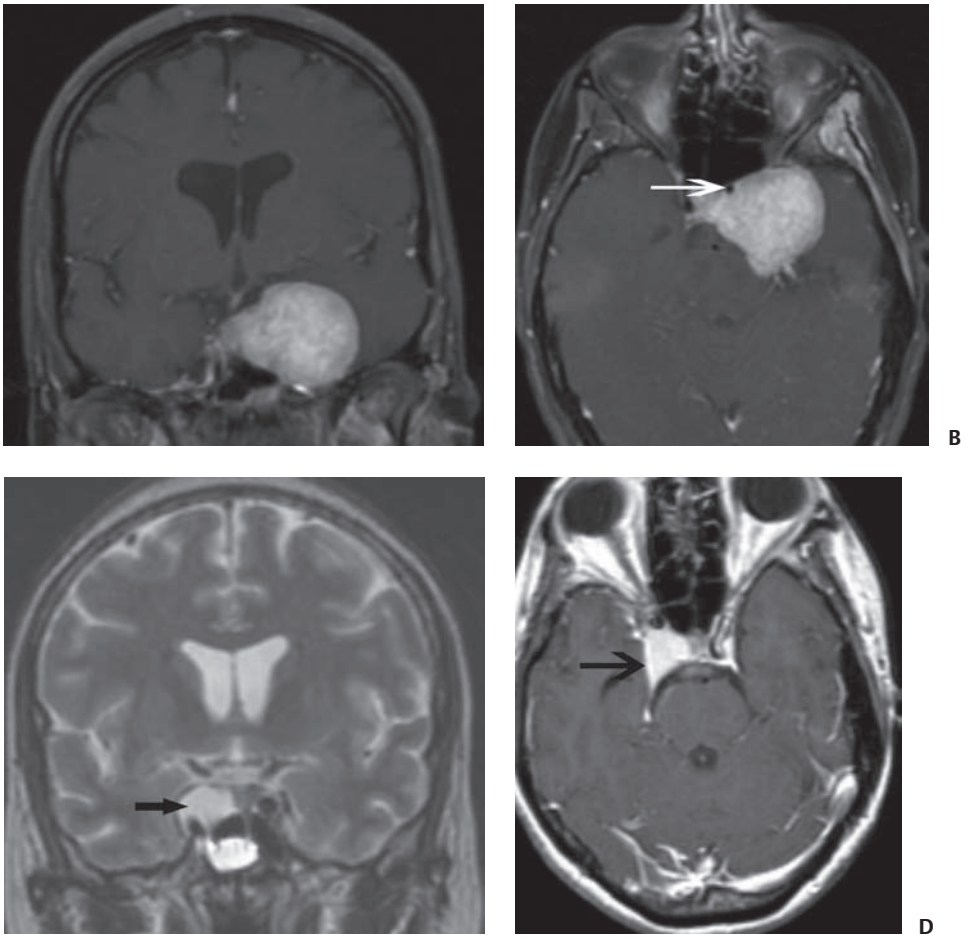


Fig. 13.19 Meningioma mimics. **(A)** Coronal T1 and **(B)** axial T1 postcontrast views demonstrate a left parasellar and sellar intensely enhancing mass proven to be a hemangiopericytoma. Note anterior displacement of the left internal carotid artery (*arrow* in **[B]**). **(C,D)** A different patient, with a mass within the right cavernous sinus demonstrating **(C)** high signal intensity on T2-weighted sequences and **(D)** marked and homogeneous contrast enhancement. This was a proven cavernous sinus hemangioma.

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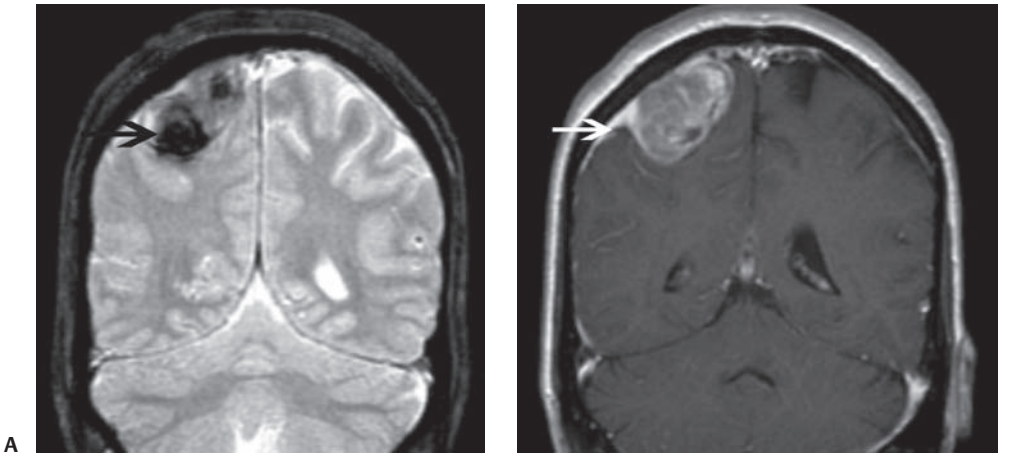


Fig. 13.20 Dural leiomyosarcoma in an acquired immunodeficiency syndrome (AIDS) patient, associated with Epstein-Barr virus. **(A)** Low signal intensity/blooming artifact on coronal gradient echo images is due to calcifications. **(B)** Contrast-enhanced coronal T1 demonstrates heterogeneously enhancing extraaxial lesion with a dural tail (*arrow*), identical to the appearance of meningioma. Thus this diagnosis should be entertained in any patient with AIDS presenting with a meningioma-like extraaxial enhancing mass.

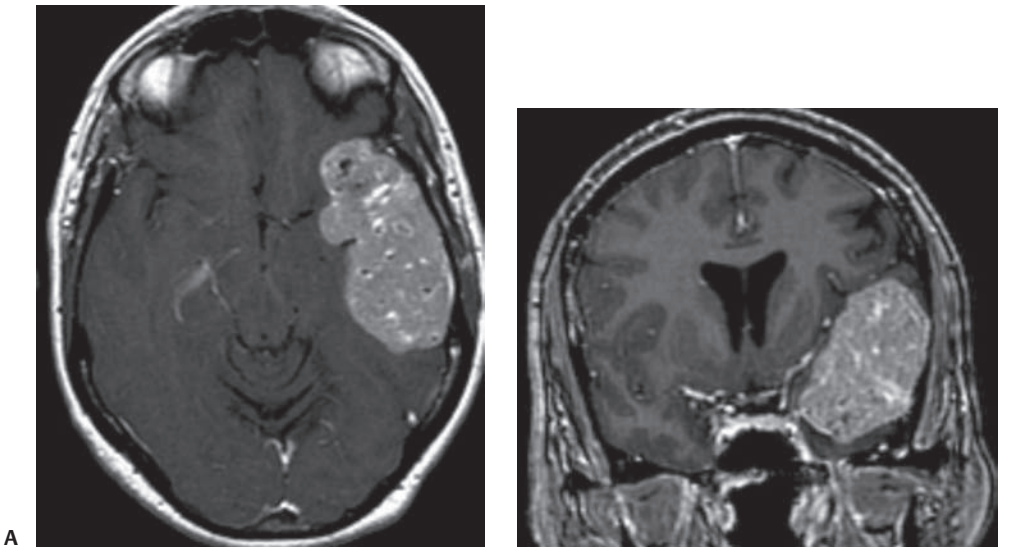


Fig. 13.21 **(A)** Gadolinium-enhanced axial T1 and **(B)** coronal T1 3-D spoiled gradient echo (SPGR) demonstrate a left middle cranial fossa extraaxial neoplasm containing flow voids, similar in appearance to meningioma; however, note that the underlying dura does not appear to be involved. A metastasis from lung adenocarcinoma was found at operation.

Meningioma Masqueraders

Numerous disease processes may mimic meningioma, the most frequent neoplasms being hemangiopericytoma (**Fig. 13.19A,B**), cavernous hemangioma (**Fig. 13.19 C,D**), dural-based metastatic disease, and lymphoma.

A rare type of neoplasm with appearance similar to meningioma is the primary intracranial neoplasm arising from pleuripotent mesenchymal stem cells in the dura. An increased incidence of dural leiomyoma and leiomyosarcoma (**Fig. 13.20**), which look similar to meningiomas, has been observed in immunocompromised and acquired immunodeficiency syndrome patients. These lesions are related to Epstein-Barr virus infection.[25,26]

Dural-based metastasis is also occasionally mistaken for meningioma. This is most frequent with prostate, breast, and occasionally lung cancer (**Fig. 13.21**).

Underlying bone destruction is almost always present with dural-based metastases. Finally, fibrous dysplasia of bone may appear similar to meningioma. Typically fibrous dysplasia appears on MRI as an enhancing intradiploic mass. The key to differentiating these lesions from meningioma, however, is noting that they expand bone, have no dural base or dural enhancement, and are very low in signal intensity on T1- and particularly on T2-weighted MRI scans. CT scanning is very helpful in confirming the "ground-glass" bone appearance typical of fibrous dysplasia.

◆ Conclusion

Meningiomas have a typical but sometimes variable appearance on MRI and CT scans. Angiography is rarely indicated for diagnosis but is performed most commonly in association with preoperative embolization. Modern imaging tools can usually suggest the histological diagnosis but not the grade of tumor. Calcification is almost always indicative of benign meningiomas. Perfusion and diffusion imaging has been useful in diagnosis and can suggest alternative histologies and predict aggressive histological features.

Once a meningioma has been identified, it is incumbent on the radiologist to look carefully for others because multiple meningiomas are not uncommon. One needs always to assess the patency of the dural sinuses, adjacent cranial nerves, and arterial structures when evaluating meningiomas.

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Chapter 14

Diagnostic Evaluation and Embolization of Meningiomas

William J. Mack and Fernando Vinuela

Since Manelfe et al first described preoperative embolization of intracranial meningiomas in 1973,[1] the technique has been utilized to reduce intraoperative blood loss and facilitate microsurgical tumor excision. In the ensuing decades, rapid technological advances in the development of microcatheters and embolic materials have resulted in greater procedural efficacy and increased safety. Meningiomas arise from the dura. Thus meningeal branches, usually of the external carotid artery, are the initial source of blood supply to the vast majority of these tumors. However, meningiomas may recruit substantial supply from pial vessels. Because these lesions are often quite vascular, preoperative embolization can ease complete resection by diminishing surgical time and intraoperative blood loss.[2–5] Many cranial base meningiomas are characterized by vascular pedicles that are medially located with respect to the operative approach.[6] In such cases, the surgical corridor is often narrow and deep, which amplifies the difficulty of operative resection. In addition to improving operative safety and visualization, preoperative embolization is believed to reduce transmitted forces to the adjacent neural tissues during surgical resection by causing ischemic necrosis and a resultant softening of the tumor mass.[7]

◆ Diagnostic Cerebral Angiogram

It is critical to perform a thorough diagnostic cerebral angiographic evaluation before meningioma embolization. Interpreted in conjunction with findings evident on noninvasive imaging, this will enable a detailed appraisal of the dural, pial, and transosseous arterial supply to the tumor and assessment of the patency and drainage pattern of the venous sinuses. Superselective angiograms afford an opportunity to delineate the nature of dangerous anastomoses involving the internal carotid and vertebral arteries and identify small branches that supply the cranial nerves. Anatomical variants, especially those of the middle meningeal and ophthalmic arteries, must be noted.

The classic angiographic appearance of a meningioma is that of a uniform hypervascular tumor blush that appears in the early arterial phase and persists through the late venous phase. A dense network of small vascular branches supplying the tumor is arranged in a "sunburst pattern," centered on larger feeding vessels[8] (**Figs. 14.1** and **14.2**). The vascularity of meningiomas is variable, however, with some lesions demonstrating relatively little angiographic evidence of contrast staining. The predominant supply to intracranial meningiomas occurs via the dural vasculature. This includes branches of both the external carotid artery and the internal carotid artery/vertebrobasilar system. Secondary supply commonly derives from small, pial branches.

The middle meningeal artery, which supplies the dura of the anterior and middle cranial fossa, is the vessel most commonly associated with meningiomas. There is often bilateral middle meningeal arterial supply to tumors in the parasagittal region. Not infrequently, transosseous supply from the superficial temporal artery is also noted (**Fig. 14.3**). Olfactory groove and planum sphenoidale meningiomas often receive additional blood supply from the ethmoidal divisions and anterior falcine branch of the ophthalmic artery. Meningeal branches of the cavernous internal carotid artery contribute significantly to the blood supply of tentorial, sphenoid wing, and petroclival meningiomas.[9] Posterior fossa meningiomas residing at the cerebellopontine angle or located along the cerebellar convexities are often fed by the ascending pharyngeal artery and transosseous branches of the occipital artery, in addition to the petrosquamosal branch of the middle meningeal artery. Midline posterior fossa and foramen magnum lesions are more often supplied by posterior meningeal branches of the vertebral artery (**Fig. 14.4**).

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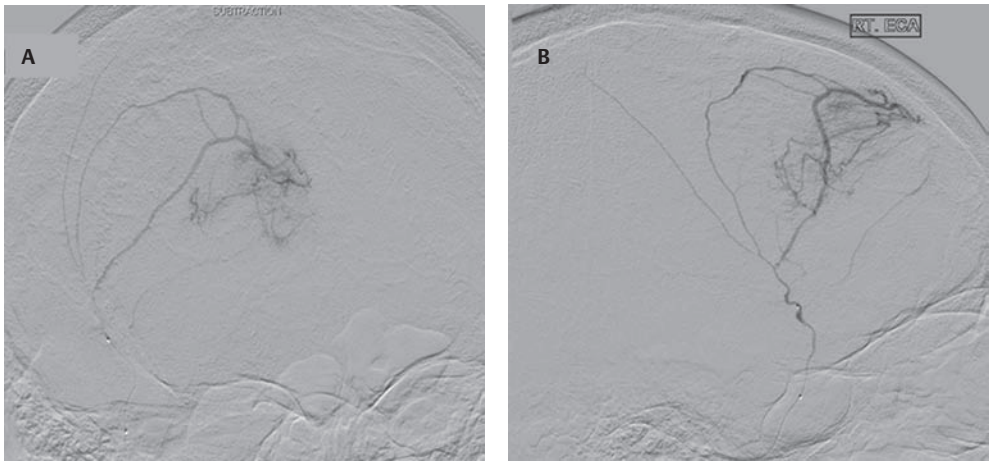


Fig. 14.1 **(A)** Oblique and **(B)** lateral views of a superselective right middle meningeal artery angiogram demonstrating the hypervascular tumor blush associated with meningiomas.

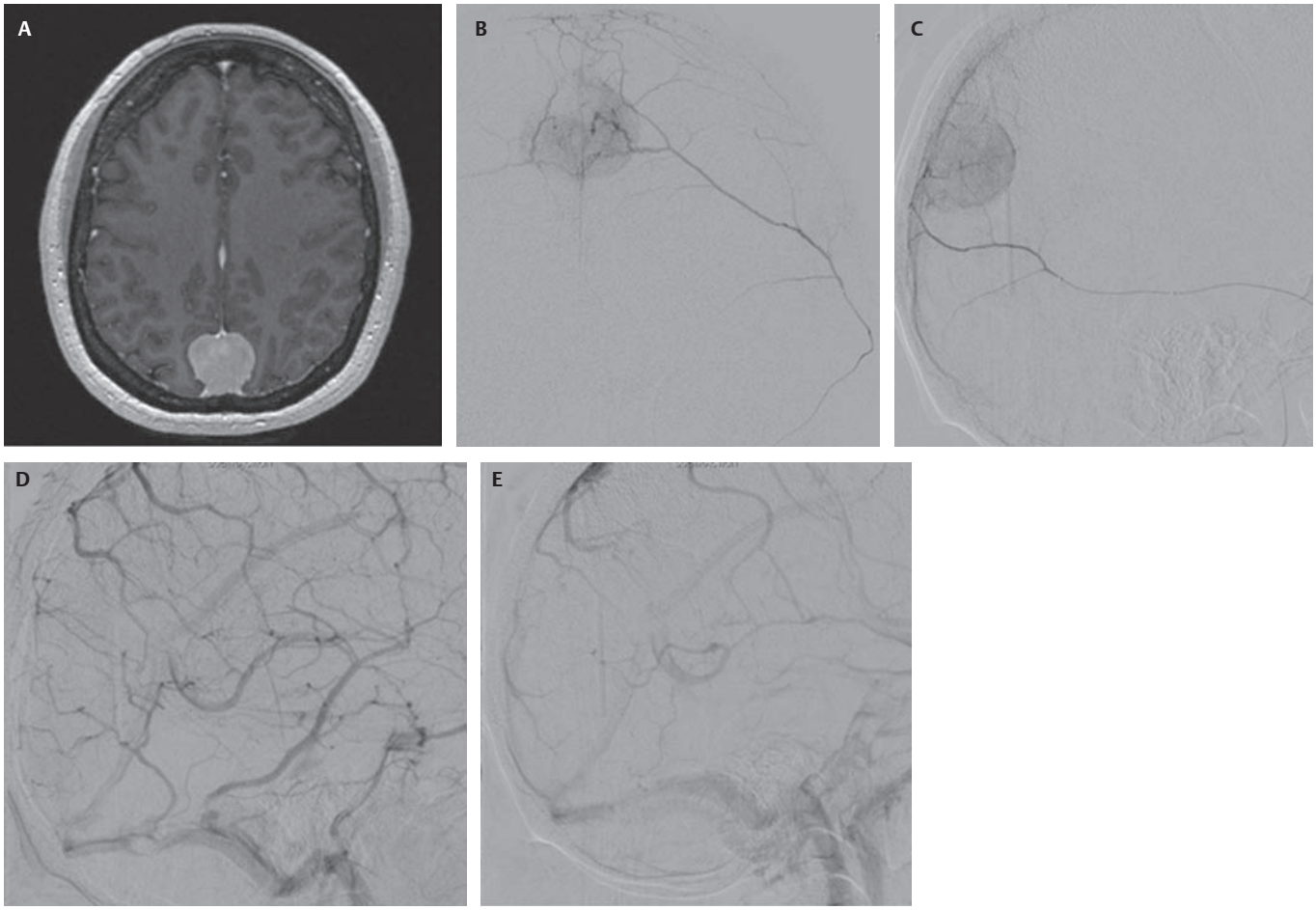


Fig. 14.2 A midline parietal convexity meningioma involving the posterior aspect of the superior sagittal sinus. **(A)** Axial magnetic resonance imaging (MRI) with contrast demonstrating the typical homogeneous enhancement characteristic of meningiomas. **(B)** Anteroposterior and **(C)** lateral superselective angiograms of the left middle meningeal artery. **(D)** Early and **(E)** late venous phase internal carotid artery angiograms demonstrating no patency of the posterior aspect of the superior sagittal sinus due to tumor invasion/thrombosis.

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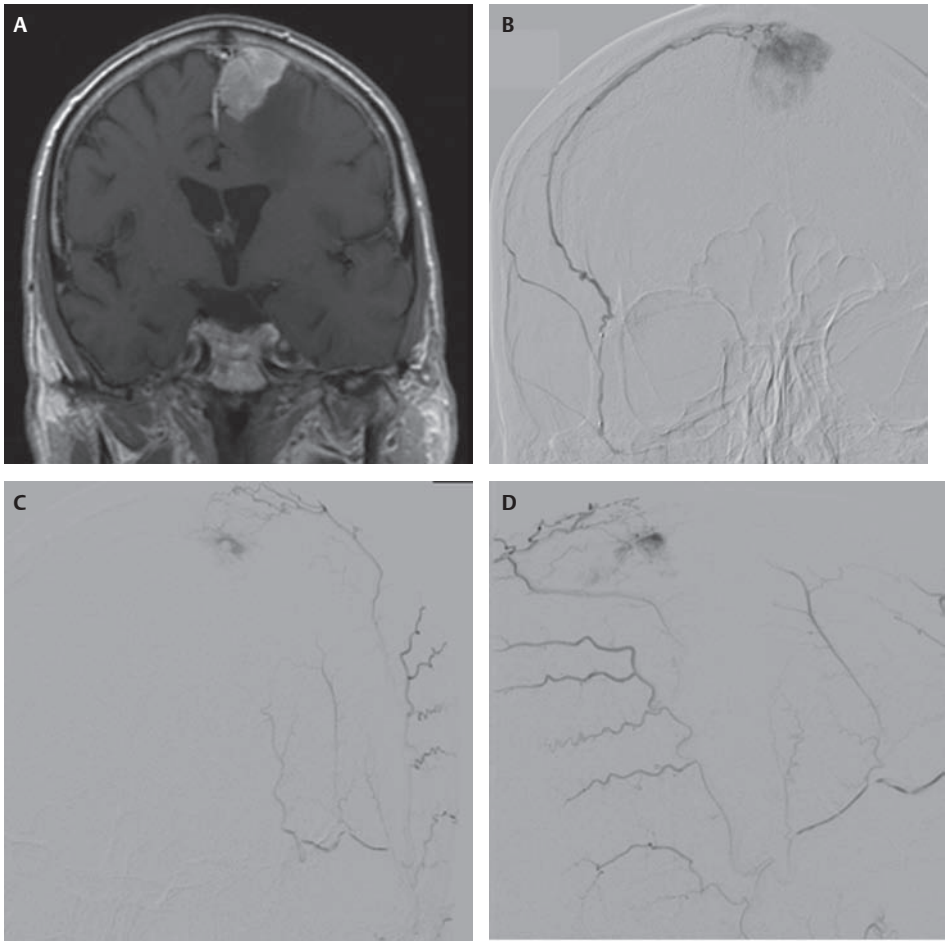


Fig. 14.3 Parasagittal convexity meningioma. **(A)** Coronal magnetic resonance imaging (MRI) with contrast. **(B)** Superselective angiogram in the anteroposterior (AP) projection of the right middle meningeal artery illustrating contralateral vascular supply that crosses midline. **(C)** AP and **(D)** lateral superselective angiograms of the left superficial temporal artery demonstrating transosseous vascular supply to the tumor.

Embolization

After a diagnostic cerebral angiogram is performed to identify blood supply and assess for feasibility and safety of embolization, a microcatheter is advanced over a microguidewire through the larger diagnostic or guide catheter. Under real-time digital subtraction fluoroscopy, the microsystem is advanced into the vascular pedicle supplying the tumor. Superselective angiography is performed through the microcatheter to confirm proper position and to identify any distal branches supplying normal tissue and to assess for the presence of dangerous anastomoses. Provocative testing with intraarterial sodium amytal or lidocaine may aid in the detection of unsafe collaterals or cranial nerve supply distal to the microcatheter tip.[3,7] After proper positioning of the microcatheter, injection of embolic material is performed under constant real-time digital subtraction fluoroscopy. Distal tumoral penetration prevents flow to the mass via collateral vessels and results in devascularization and subsequent tumor necrosis.

Several agents have been utilized to embolize meningiomas, including particles,[10,11] n-butyl cyanoacrylate,[12] Onyx (ev3 Neurovascular, Irvine, CA),[13] platinum coils,[14] ethanol,[15] fibrin glue,[16] and Gelfoam pledgets (Pfizer, Inc., NY).[5,17] Particulate agents are favored over liquid embolic materials due to their ease of use and relative safety. Because the precise penetration capacity of particles is dependent on their size, appropriate selection precludes infiltration into the small arterioles supplying cranial nerves. Because the vasa nervorum are small, the use of particles larger than 150 µm in diameter has been suggested to lessen the risk of ischemic cranial nerve injury.[18,19] Polyvinyl alcohol (PVA) particles are believed to remain occlusive in vessels for time periods in the order of weeks, after which vascular recanalization may occur.[20] This property is suitable for preoperative tumor embolization. Given that the tumor mass is excised during definitive surgical resection, a long-term durable embolization is not necessary. Trisacryl gelatin microspheres are deformable, homogeneous, and uniformly shaped. They are believed to result in less catheter obstruction and recanalization than do irregularly shaped and sized PVA particles.[21] Although they may penetrate deeper than similar-sized PVA particles, they are also more likely to inadvertently enter and remain in small blood vessels. After particle embolization is performed, a platinum coil or Gelfoam pledget may be placed more proximally, in the treated vessel, to help

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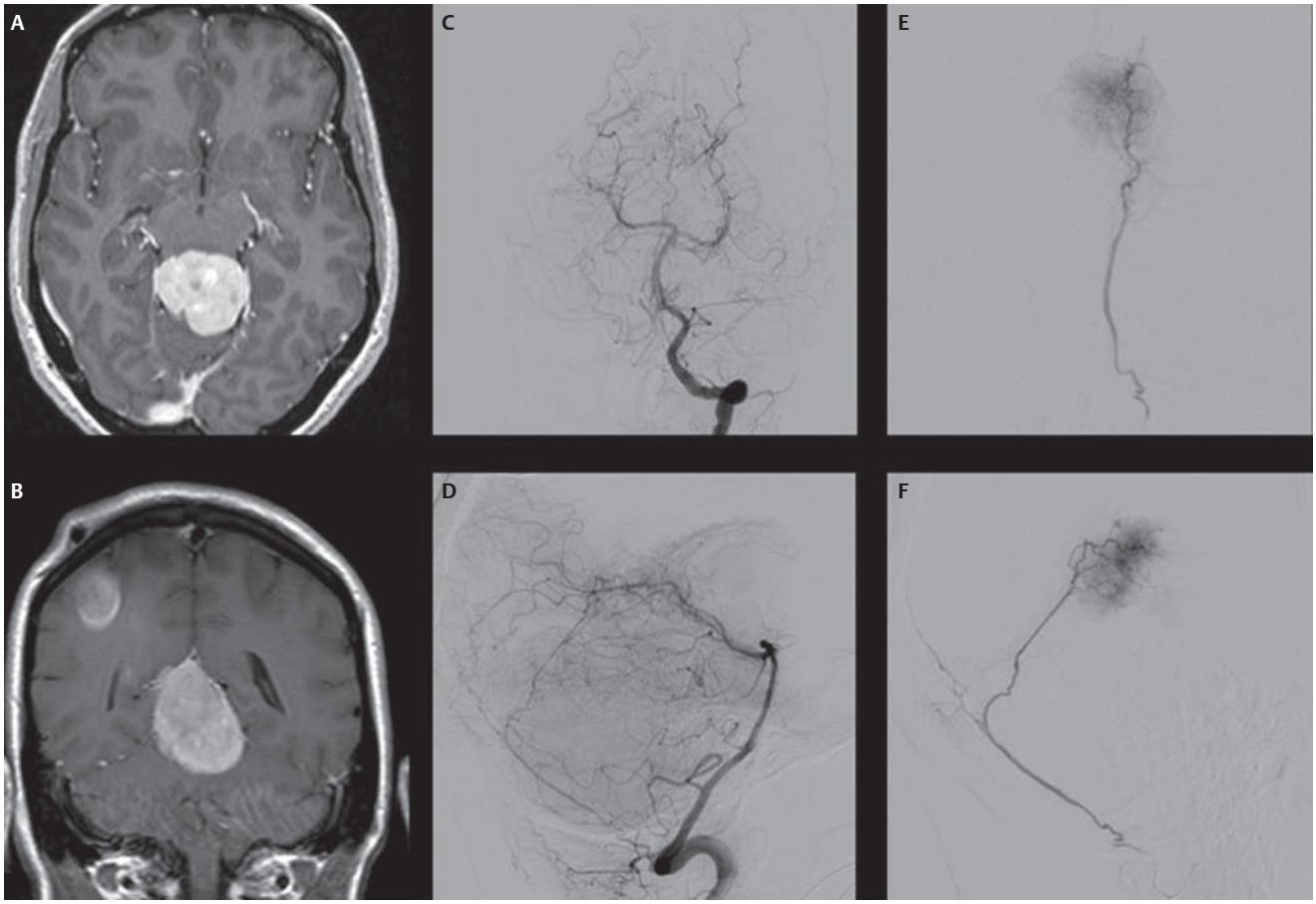


Fig. 14.4 Midline tentorial meningioma. **(A)** Axial and **(B)** coronal magnetic resonance images. Artifact is seen in the right parietal lobe on the coronal image secondary to the presence of a ventriculoperitoneal shunt. **(C)** Anteroposterior (AP) and **(D)** lateral left vertebral artery angiograms demonstrating a tumor blush in the midline tentorial region being supplied by the posterior meningeal branch of the extracranial vertebral artery. **(E)** AP and **(F)** lateral superselective angiograms of the posterior meningeal branch of the left vertebral artery demonstrating the dural vascular supply to this deep midline tumor.

prevent recanalization and aid in surgical resection[3] (**Fig. 14.5**). Care is taken to avoid proximal reflux of any embolic material into eloquent vascular branches.

In contrast to particles, liquid embolic materials such as glue (n-butyl cyanoacrylate) and Onyx can more easily enter anastomotic channels that supply cranial nerves or cortical vessels not evident on the initial diagnostic angiogram. The operator must be acutely aware of this possibility when utilizing such agents for tumor embolization. Additionally, microcatheter retention can result in vessel rupture or ischemic sequelae. The use of particles, rather than liquid embolic agents, eliminates the prospect of this complication. As such, meningiomas located distant from the skull base foramina and exiting cranial nerves are most suitable for embolization with liquid agents. (**Fig. 14.6**).

Determining which vessels to embolize is not always straightforward. Most agree that large, external carotid artery vessels that directly supply the tumor mass and are distant from dangerous anastomoses may be treated. However, meningiomas supplied by both the external and internal carotid artery are reported to exhibit increase pial supply subsequent to embolization of external branches.[5] Augmented blood flow and hypertrophy of the small, deep intracranial vessels may increase the difficulty of the surgical resection because this vascular supply is often not encountered until the end of the operation.

Preoperative embolization of pial blood supply is generally not performed because the benefit of embolization is usually outweighed by the potential risk of ischemia and stroke (**Fig. 14.7**). However, some authors advocate embolization of hypervascular tumors purely, or predominantly, supplied by internal carotid artery or pial branches.[7,22] This technique requires the ability to advance a microcatheter just proximal to the tumor bed, excluding any en passage vessels from inadvertent embolization. Furthermore, there must be no adjacent normal brain staining evident on superselective angiography. Preexisting irreversible neurological deficits may decrease the subsequent risks inherent to embolization of pial vessels before surgical resection.[7]

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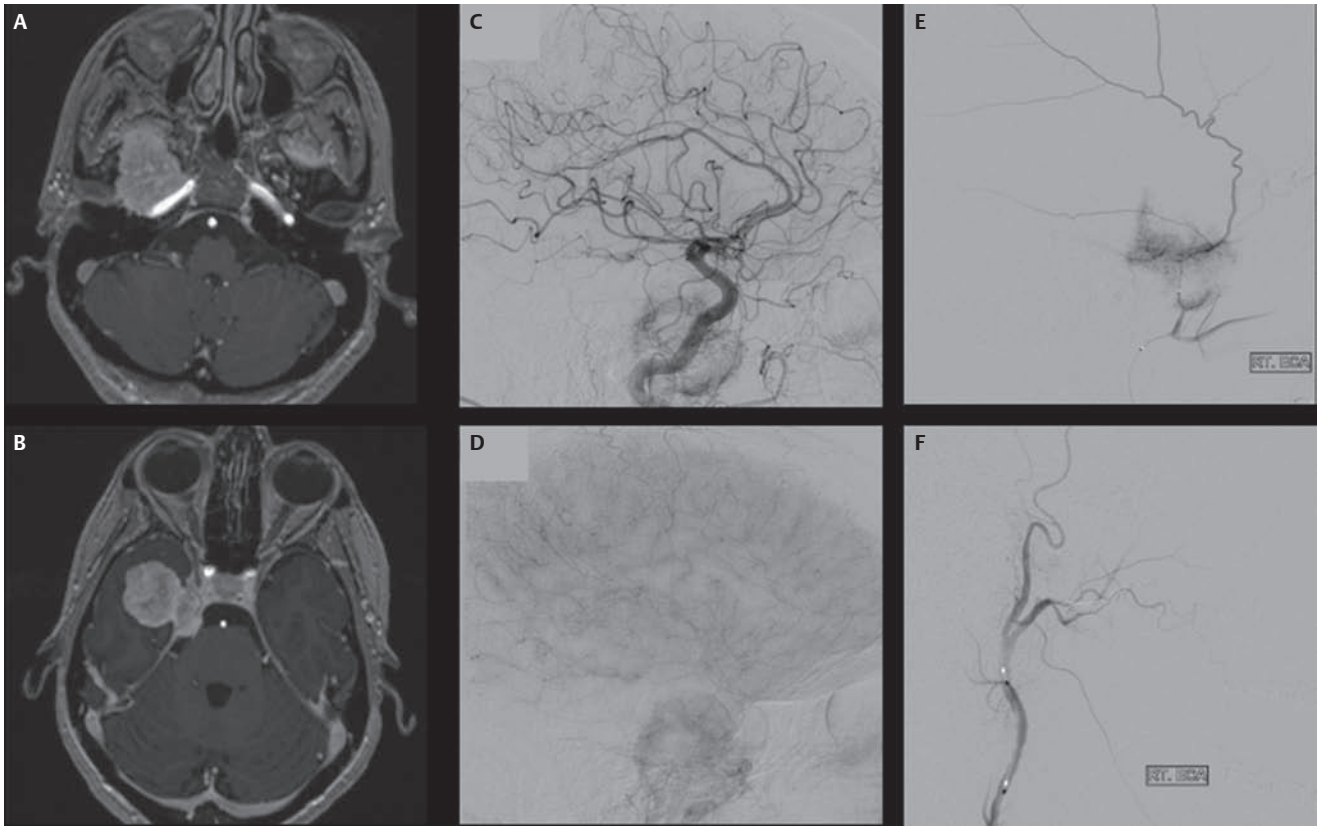


Fig. 14.5 Right inferior temporal meningioma. **(A,B)** Axial magnetic resonance images. **(C)** Arterial and **(D)** venous phase right common carotid artery angiograms demonstrating a large round tumor blush centered in the right inferior temporal region. **(E)** Superselective lateral right internal maxillary artery angiogram demonstrating significant vascular supply from the middle meningeal and accessory meningeal arteries. **(F)** Postintervention angiogram of the right external carotid artery after embolization with polyvinyl alcohol particles followed by platinum coils. Note the absence of appreciable residual tumor blush.

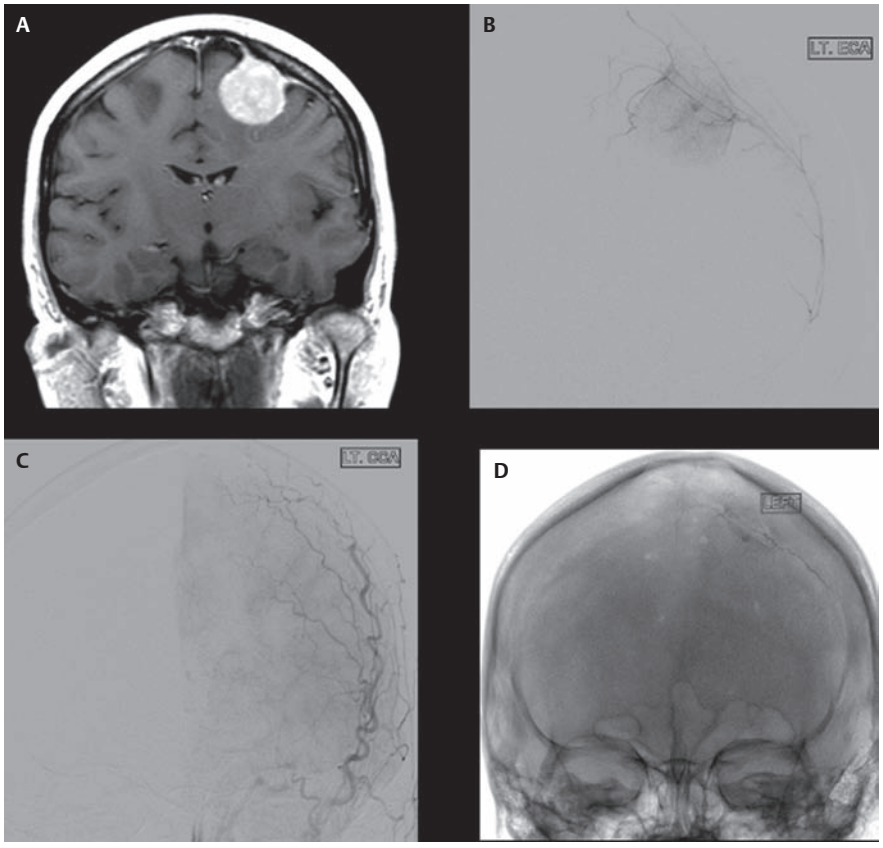


Fig. 14.6 Left convexity meningioma. **(A)** Coronal magnetic resonance image. **(B)** Superselective left middle meningeal artery angiogram demonstrating the hypervascular mass in the late arterial phase. **(C)** Anteroposterior (AP) left external carotid artery angiogram demonstrating the absence of tumor blush following embolization with n-butyl cyanoacrylate (nBCA). **(D)** AP fluoroscopic image demonstrating the glue cast in the distal left middle meningeal artery. nBCA was utilized to embolize this tumor due to its distal location, far from the skull base foramina and exiting cranial nerves.

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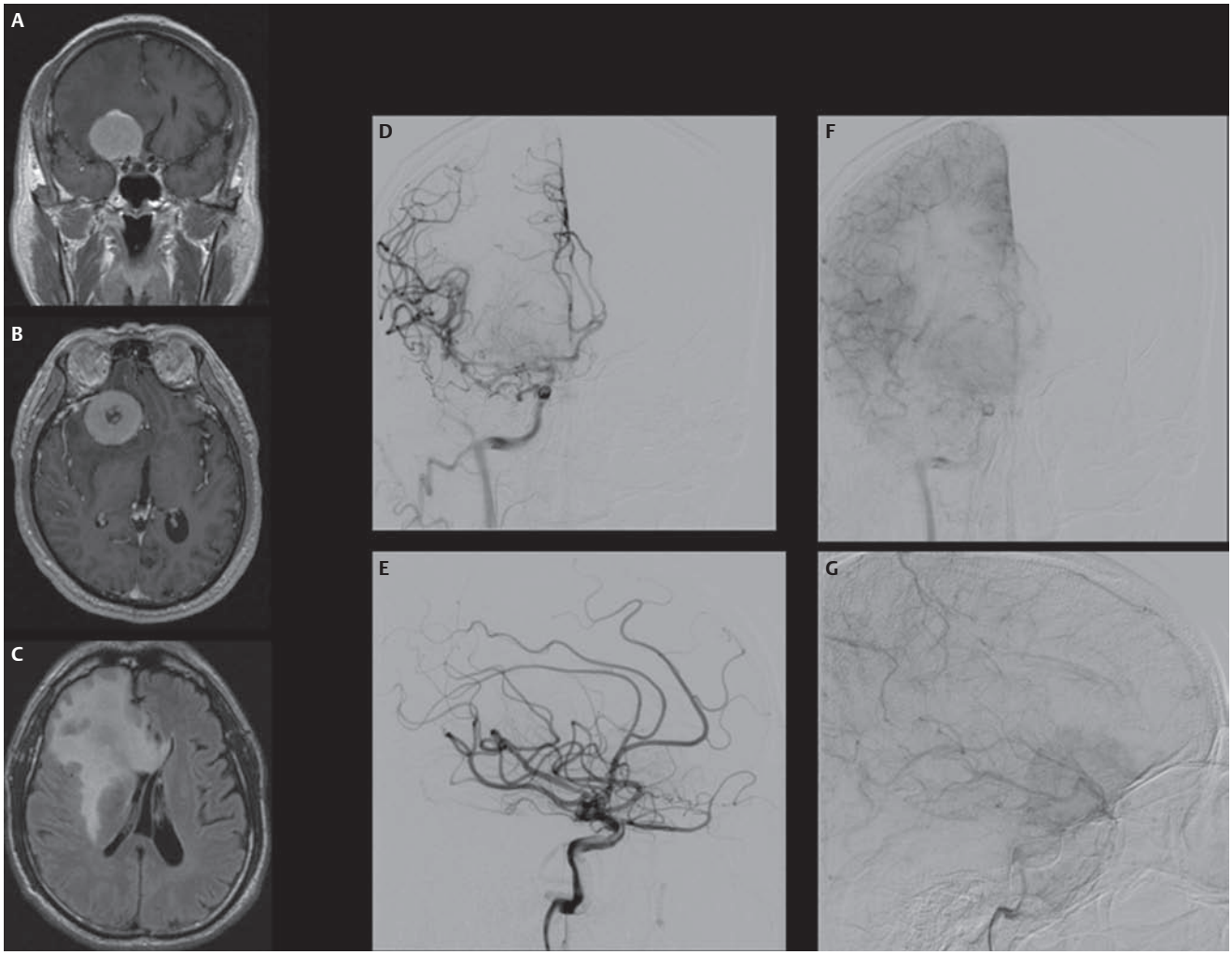


Fig. 14.7 Right anterior paraclinoid meningioma. **(A)** Coronal and **(B)** axial magnetic resonance imaging (MRI) demonstrating a homogeneously enhancing mass with central necrosis. **(C)** Axial fluid-attenuated inversion recovery MRI demonstrating a vast amount of edema, often evident with pial vascular supply. **(D,F)** AP and **(E,G)** lateral, **(D,E)** arterial and **(F,G)** venous phase left internal carotid artery angiograms demonstrating extensive ophthalmic and anterior cerebral artery supply to the hypervascular tumor blush centered in the anterior cranial fossa. Significant mass effect is noted on the anterior cerebral arteries below the level of the falx cerebri. Due to the extensive pial vascular supply to this tumor, preoperative embolization was not undertaken.

◆ Dangerous Anastomoses

Embryologically and phylogenetically, the external carotid artery and the cervical muscular branches are closely associated with the intracranial vasculature. It is critically important to understand and respect the small anastomotic channels connecting the systems when performing tumor embolization procedures. Inadvertent penetration of embolic material through these channels can result in stroke or cranial nerve palsies.[23] Most of the channels enter the cranial vault through the skull base foramina. Often not visualized on routine angiography, these anastomoses demonstrate patency with increased intraarterial pressure during microcatheter injections for embolization procedures.

Orbital Region

The important risk to consider when embolizing meningiomas centered in the orbital region is occlusion of the central retinal artery, which results in blindness.

Rarely, the ophthalmic artery can have a middle meningeal origin, called the meningoophthalmic artery, located at the level of the foramen spinosum. In this variation, the remnant of the primitive stapedial artery assumes the entire orbital supply from the embryological ophthalmic artery. Even proximal middle meningeal artery occlusion is of high risk in this setting.[23–25] More commonly, a spectrum of incomplete variants exist, in which portions of the orbital supply are annexed by the middle meningeal artery. Inadvertent embolization of any branches that

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anastomose with the central retinal artery can lead to loss of vision. The presence of a choroidal blush on external carotid artery injection or nonvisualization of the ophthalmic artery on internal carotid angiograms suggests the existence of these anatomical variants. In the normal anatomical configuration, the superficial recurrent meningeal branch of the middle meningeal artery anastomoses with the lacrimal or ethmoidal branches of the ophthalmic artery through the superior orbital fissure,[26] and the anterior branch of the middle meningeal artery anastomoses with the anterior falcian branch of the ophthalmic artery[23] (**Fig. 14.8**).

The internal maxillary artery also provides a rich network of anastomotic channels to the ophthalmic artery. The anterior deep temporal arteries (transmalar) and infraorbital branches (inferior orbital fissure) provide collateral supply to the lateral and medial muscular branches of the ophthalmic artery, respectively. The sphenopalatine and septal arteries anastomose with the ethmoidal branches of the ophthalmic artery.[23,24,26]

The frontal branch of the superficial temporal artery anastomoses with the supraorbital branch of the ophthalmic artery. The angular branch of the facial artery travels through the inferior orbital fissure to connect with the ethmoidal or lacrimal branches of the ophthalmic artery.[23]

Parasellar Region

This is the region of interest when performing embolization on cavernous, sellar, and sphenoid wing meningiomas. Small branches of the external carotid artery anastomose with the C4 and C5 segments of the internal carotid artery siphon through the inferolateral and meningohypophyseal trunks. Embolization risks include injury to the cranial nerves in the cavernous region and stroke. Enlargement of the meningohypophyseal trunk sometimes allows for safe, selective distal microcatheterization and direct embolization of large parasellar and clival meningiomas (**Fig. 14.9**).

The cavernous rami of the middle meningeal and accessory meningeal branches of the internal maxillary artery anastomose with the internal carotid artery via the meningohypophyseal and inferolateral trunk through the foramen spinosum and the foramen ovale/vesalius, respectively.[12,23,27] The artery of the foramen rotundum is a small tortuous branch of the distal internal maxillary artery that travels through its named foramen and connects with the inferolateral trunk.[27,28] Inadvertent injection of particulate or liquid embolic agents through these small channels can result in an internal carotid artery territory stroke or injury to cranial nerves III through VI.

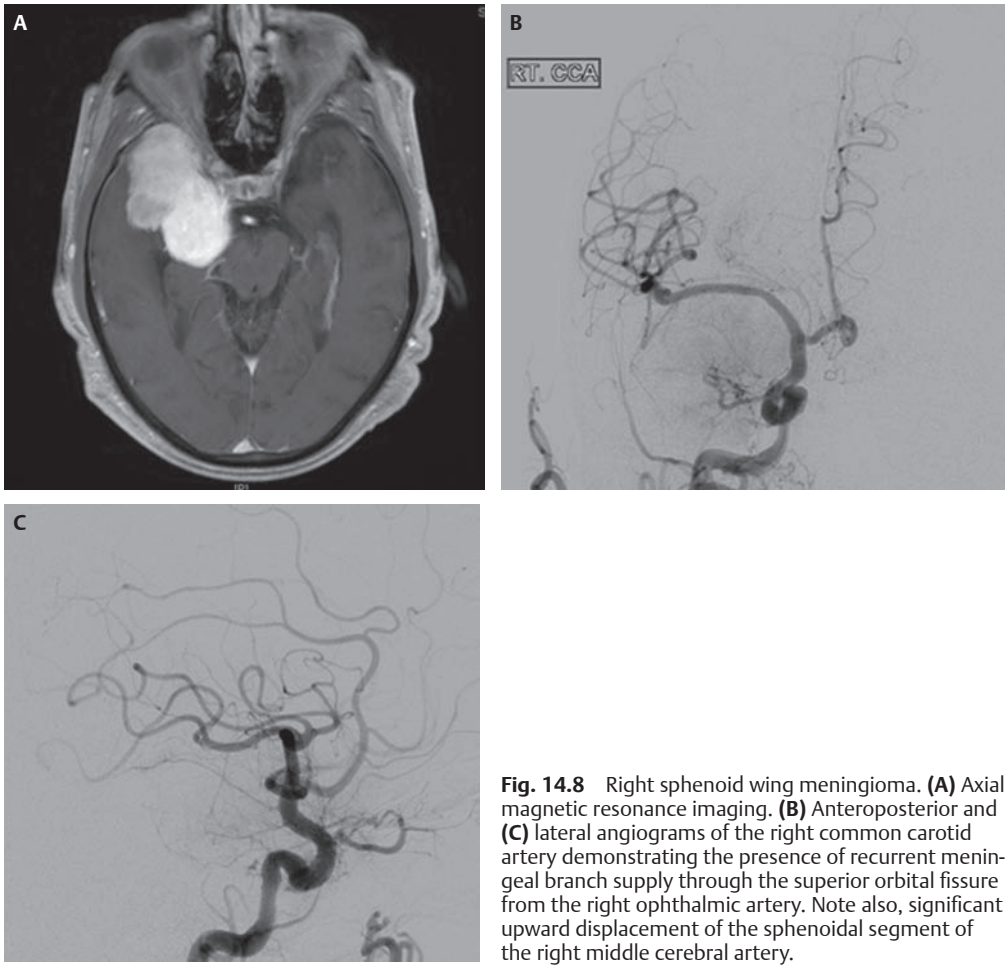


Fig. 14.8 Right sphenoid wing meningioma. **(A)** Axial magnetic resonance imaging. **(B)** Anteroposterior and **(C)** lateral angiograms of the right common carotid artery demonstrating the presence of recurrent meningeal branch supply through the superior orbital fissure from the right ophthalmic artery. Note also, significant upward displacement of the sphenoidal segment of the right middle cerebral artery.

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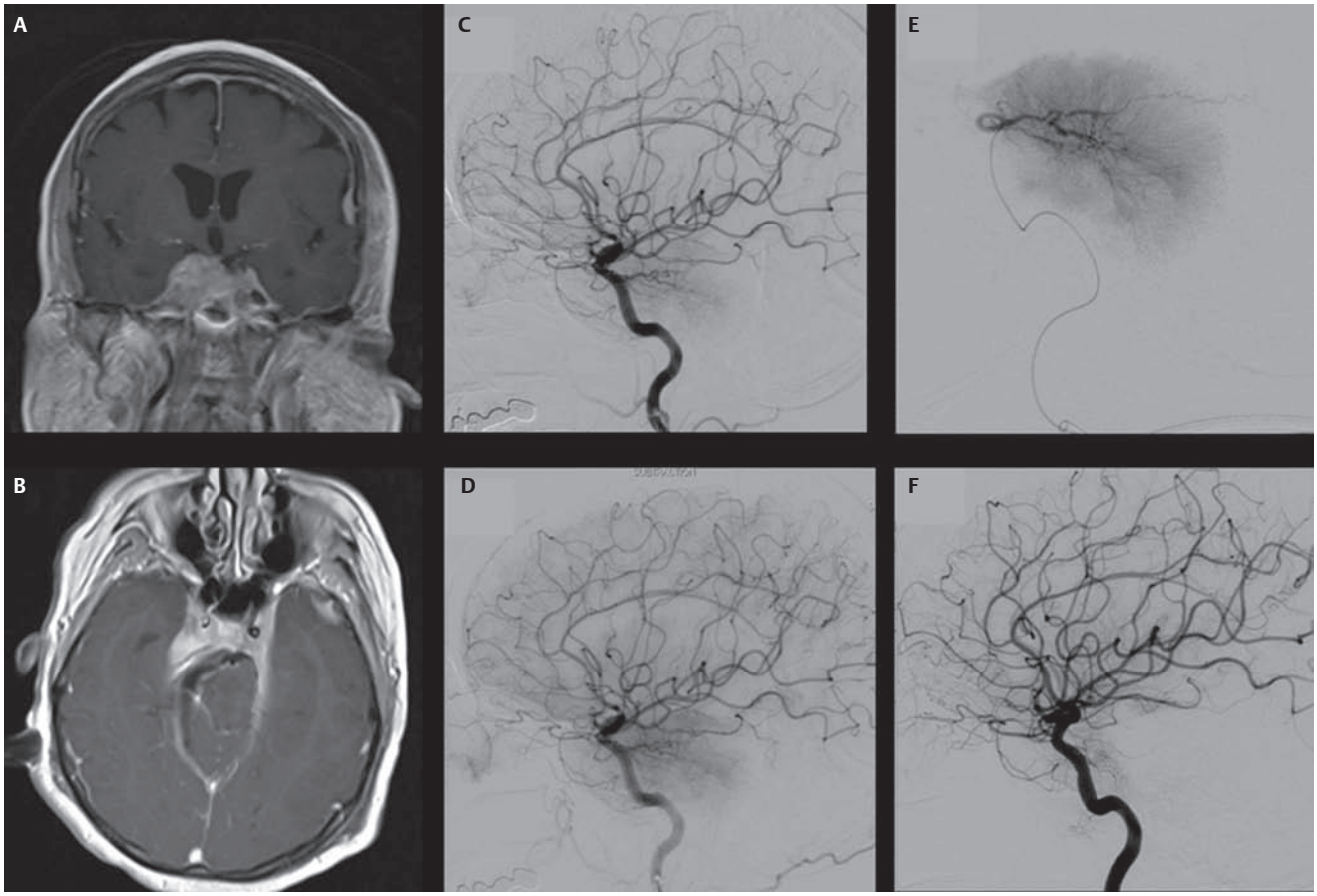


Fig. 14.9 Suprasellar meningioma. **(A)** Coronal and **(B)** axial magnetic resonance imaging demonstrating a homogeneously enhancing mass that encases the bilateral internal carotid arteries. Lateral right internal carotid artery angiograms in the **(C)** arterial and **(D)** capillary phases demonstrating a suprasellar tumor blush supplied by enlarged dural branches of the meningohypophyseal trunk. **(E)** Superselective angiogram of the meningohypophyseal trunk. **(F)** A significant decrease in the size and intensity of the tumor blush following polyvinyl alcohol and coil embolization of the meningohypophyseal branch supplying the tumor.

Petroclival Region

When embolizing meningiomas in the petroclival and cerebellopontine angle regions, care must be taken to document the presence of anastomotic channels that, if embolized, can result in devastating lower cranial nerve deficits or internal carotid/vertebral artery territory stroke.

The pterygovidian branch of the distal internal maxillary artery travels through the vidian canal to the foramen lacerum and anastomoses with the mandibulovidian branch of the petrous internal carotid artery.[29] Additionally, the petrosquamous branch of the middle meningeal artery can anastomose with the marginal tentorial artery, arising from the meningohypophyseal trunk (or inferolateral trunk/ophthalmic artery) of the cavernous carotid artery.[23,25]

The ascending pharyngeal artery anastomoses with the internal carotid artery via both of its branches, the pharyngeal and the neuromeningeal trunks. The superior pharyngeal branch of the ascending pharyngeal artery travels through the foramen lacerum and anastomoses with the recurrent artery of the foramen lacerum and the inferolateral or meningohypophyseal trunk of the cavernous carotid artery.[25] The neuromeningeal trunk has both jugular and hypoglossal branches, which anastomose with clival branches of the meningohypophyseal trunk through the jugular and hypoglossal foramina, respectively.[30] These clival branches often provide supply to meningiomas located in the petroclival region (**Fig. 14.10**). The inferior tympanic branch of the ascending pharyngeal artery enters the skull base through the Jacobson canal and anastomoses with the caroticotympanic branch of the petrous internal carotid artery. These vessels also anastomose with the superior tympanic branch of the petrous middle meningeal artery, the anterior tympanic branch of the internal maxillary artery, and the stylomastoid branch of the occipital/posterior auricular artery. Vascular supply to the seventh and eighth cranial nerves is derived from arteriolar branches of these external carotid artery vessels and anastomoses through the facial arcade.[23] These connections must be addressed when performing preoperative embolization of cerebellopontine region meningiomas.

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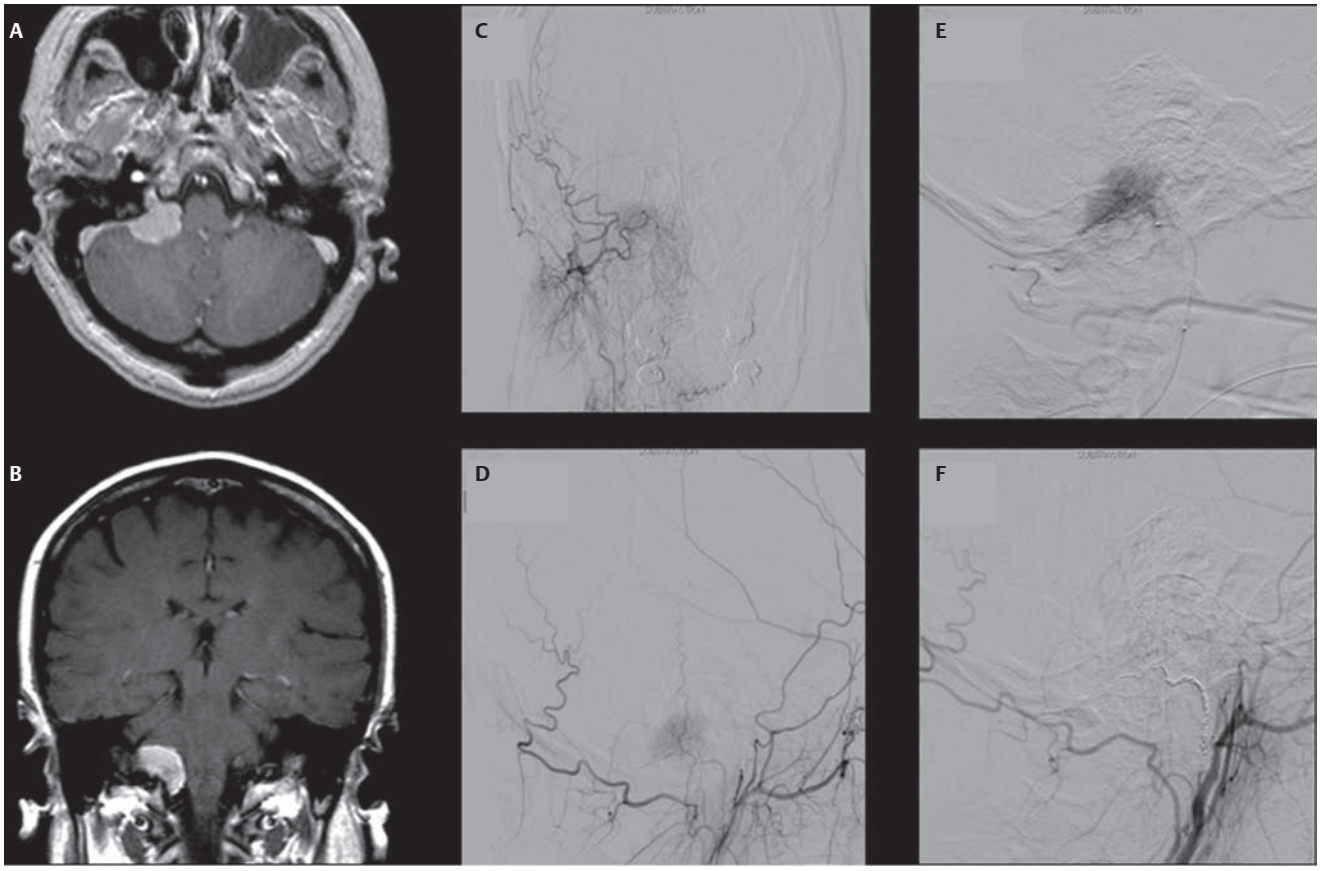


Fig. 14.10 Right ventral cerebellar meningioma. **(A)** Axial and **(B)** coronal magnetic resonance imaging. **(C)** Anteroposterior and **(D)** lateral superselective angiograms of the left external carotid artery demonstrating a tumor blush supplied by neuromeningeal branches of the ascending pharyngeal artery. **(E)** Superselective angiogram of the ascending pharyngeal artery in the lateral projection demonstrates robust vascular supply through the neuromeningeal trunk. **(F)** Lateral external carotid artery angiogram following coil embolization demonstrates a significant decrease in the size and intensity of the tumor mass. Platinum coils were chosen in this case due to safety resulting from proximal placement. Embolization with polyvinyl alcohol or liquid embolic agent may risk ischemia to the cranial nerves exiting through the jugular and hypoglossal foramina.

Cervicomedullary Region

Meningiomas located at the foramen magnum and cervicomedullary level often derive vascular supply from a network of branches from the ascending pharyngeal artery, the occipital artery, the thyrocervical and costocervical subclavian trunks, and the vertebral artery. Particular care must be paid to the numerous anastomotic channels in this highly vascular region because risks of inadvertent embolization include ischemic injury to the lower cranial nerves and posterior circulation or spinal cord stroke.

The occipital artery maintains connection to the vertebrobasilar system through posterior muscular anastomotic branches at the C1 and C2 cervical interspaces. The ascending pharyngeal artery anastomoses with the vertebral artery through the musculospinal branch at the third cervical interspace and the direct lateral branch at the fourth cervical space.[23] The external carotid artery trunk also maintains connections with the vertebral artery at the fourth cervical space. Additionally, the hypoglossal branch of the ascending pharyngeal artery descends through the hypoglossal canal to supply the odontoid arch arcade. This rich vascular network anastomoses with the vertebral artery at the third cervical space.[23,31]

The ascending cervical branch of the thyrocervical trunk and the posterior deep cervical branch of the costocervical trunk both arise from the subclavian artery. They harbor multiple anastomotic channels at the high cervical spaces with the vertebral artery and the pharyngeal-occipital system through the suboccipital carrefour. Angiographic localization of the anterior spinal artery and assessment of dangerous connections to the vertebrobasilar system must be performed before embolization of meningiomas in the foramen magnum or high cervical region.

◆ Outcome

The utility of preoperative meningioma embolization has been debated because quantitative measurement of success has proven difficult. Several large case series

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document angiographic, histologic, and surgical benefit.[2,4,5,7,10,32] Some reports question the benefit, citing no differences in operative time, blood loss, or clinical outcome.[11] Other groups reserve preoperative embolization for difficult skull base meningiomas, referencing complication rates deemed unacceptable for straightforward surgical resections.[6]

In a nonrandomized cohort of 52 patients who underwent surgical resection of meningiomas, Macpherson reported less intraoperative blood loss, decreased incidence of surgical complications, and superior clinical outcomes in patients whose tumors were embolized preoperatively. The results were encouraging; however, the study lacked rigorous statistical analysis and was subject to an inherent selection bias. Patients who did not undergo embolization were those in whom little external carotid artery supply was noted or embolization was not possible for technical reasons.[4]

Dean et al reported a retrospective paired analysis of 36 patients harboring meningiomas that were matched with respect to size, anatomical location, and histologic subtype of tumor. Only the estimated blood loss and number of blood transfusions differed significantly, and favored preoperative embolization.[32] In a series of 60 consecutive patients, Bendszus et al demonstrated a beneficial effect of embolization with respect to intraoperative blood loss during surgery only in the cohort of patients with complete tumor devascularization following preoperative embolization.[11] Other parameters failed to reach clinical significance.

The timing of neurosurgical resection following embolization has been debated. Some groups contend that timing of surgery does not affect outcome, whereas others have advocated delayed resection to allow for interval tumor necrosis. A comparative, retrospective analysis of 50 patients who underwent preoperative embolization for meningioma resection indicated that delaying resection more than 24 hours after embolization can decrease intraoperative blood loss.[33] However, other reports suggest that histological changes demonstrate no obvious relationship with the interval between embolization and surgical resection.[34] A significantly long interval between particulate embolization and surgical resection, however, may allow for recanalization of blood vessels supplying the tumor.

Complications

Significant neurological deficits have been reported following preoperative meningioma embolization. Causes include distal vessel occlusion, reflux of embolic agents, and tumor swelling or hemorrhage following devascularization.[18,19,35,36] Therefore, potential benefits of embolization must be carefully weighed against the risk of adverse outcome. The majority of complications occur during the perioperative period.[18] In several small studies, complications are reported at rates between 12 and 16%.[17,32] In a large series of skull base meningiomas, neurological deficits were documented in as many as 21.6% of patients, with permanent sequelae reported in 9%.[6] Such tumors are the most challenging to embolize due to the proximity of the skull base foramina and the exiting cranial nerves. Bendszus et al recently described an overall complication rate of 6.4% in a series of 185 patients.[19] Hemorrhagic and ischemic complications were equally frequent. Carli et al reported a large retrospective series of 198 patients harboring intracranial meningiomas that underwent preoperative particle embolization and report a 5.6% complication rate. The vast majority of adverse events occurred as a result of hemorrhage.[18]

It has been postulated that necrosis, as a result of deep particle penetration and resultant devascularization, renders embolized tumor tissue susceptible to hemorrhage.[19,35,36] Venous outflow compromise secondary to particle occlusion may increase the risk of hemorrhagic complications. Some authors suggest that malignant or aggressive meningiomas are inherently more inclined to hemorrhage. They site fragile, thin-walled arterioles proximal to the site of microparticle embolization and an increased susceptibility to blood pressure elevation and subsequent rupture.[36] A review of eight cases of reported postembolization hemorrhage[35,36] suggested an increased incidence in large tumors that were aggressively embolized. Hemorrhage was unrelated to tumor location, feeding vessels, or type or size of particle utilized. The authors noted a striking preponderance of cystic or necrotic tumors in this group of patients, disproportionate to the incidence of such features among all meningiomas. Preoperative T2-weighted gradient echo magnetic resonance imaging can be useful to detect previous microhemorrhage and identify at-risk patients.[19] Whereas small particles might be more effective in devascularizing meningiomas and may lead to facilitated surgical tumor resection, they have also been cited as an independent risk factor for both ischemic and hemorrhagic complications.[10,18,19,36]

Intracranial mass effect can worsen when a very large meningioma is embolized. Dissolution of particles and resolution of vasospasm may result in early recanalization and reperfusion of incompetent vascular beds, contributing to edema and swelling.[13] Administration of intravenous steroids often ameliorates tumor swelling following embolization. Availability of neurosurgical support is important if immediate operative resection is necessary.[6]

Asymptomatic pulmonary migration of embolic material likely occurs more frequently than is appreciated. Cases of pulmonary embolus have been reported following cerebral embolization procedures.[37] Although a theoretical risk occurs when treating tumors, this sequela is more likely to occur when embolizing high-flow lesions, such as arteriovenous malformations, with cyanoacrylate embolic material.

Although preoperative meningioma embolization is generally a safe procedure, the quoted complication rates are not negligible. Patients harboring large, hypervascular tumors supplied primarily by external carotid artery branches that are located in anatomical regions difficult to access surgically, likely derive the greatest benefit from

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preoperative embolization. However, when the internal carotid artery supply to the tumor is substantial and the benefits of embolization appear to be low, even small procedural risks are unacceptable. Because embolization typically serves as an adjunct to definitive surgical resection, and is almost never curative, one must be judicious in the use of aggressive embolic agents and targeting of pial vessels.

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VII

Surgical Treatment of Intracranial Meningiomas by Site

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Chapter 15

Convexity Meningiomas

Shaan M. Raza, Alfredo Quiñones-Hinojosa, and Alessandro Olivi

Background

Accounting for 15% of all meningiomas, convexity meningiomas are the most common tumors of the cranial vault.[1] The term *convexity* refers to those meningiomas that do not extend into the dura mater of the skull base and do not involve the dural venous sinuses. As opposed to tumors in other locations, these lesions are often readily accessible. Advancements in diagnostic imaging (leading to earlier diagnosis) and surgical technology have cumulatively resulted in improved surgical outcomes, with modern studies indicating lower morbidity and mortality rates (complication rate 1.7 to 9.4% and 30-day mortality of 0%) in comparison with other anatomical locations.[2,3] The difficulties often lie in deciding when to operate and how to manage recurrent or residual disease. The emergence of stereotactic radiosurgery as a viable treatment option for smaller meningiomas not only has altered the patient population undergoing microsurgical resection but also has altered the goals of resection, the management of recurrent disease, and the expectations of the patient. Unfortunately and surprisingly, there are relatively few modern studies analyzing long-term outcomes of patients with convexity meningiomas that can aid in this decision-making process. Data are often extrapolated from studies assessing various other anatomical sites. This chapter discusses the epidemiology, location, presentation, radiographic findings, indications for treatment, and operative management of these lesions.

Epidemiology and Location

Convexity meningiomas account for nearly 15% of all meningiomas (second-highest incidence after parasagittal meningiomas).

Originally, convexity meningiomas were classified by site: frontal, paracentral, parietal, occipital, and temporal. In 1938, in *Meningiomas: Their Classification, Regional Behavior, Life History and Surgical End Results*, Cushing and Eisenhardt incorporated the following stratification: precoronal, coronal, postcoronal, paracentral, parietal, occipital, and temporal. This subclassification is advantageous in that it recognizes the eloquent (or noneloquent) nature of the underlying cortex. To this categorization, one can also add pterional or lateral sphenoid wing meningiomas that grow solely or mainly outward toward the frontal and temporal lobes.

Convexity meningiomas can further be classified on radiographic appearance: globose or en plaque. Globose refers to the classic spherical, lobulated mass, whereas en plaque meningiomas are those lesions that have a flatter, carpetlike appearance infiltrating the dura.

Presentation

Considering that meningiomas are generally slow-growing tumors and that the underlying cortex can be noneloquent, patients with convexity meningiomas typically have a protracted clinical course and larger-than-expected tumors at the time of diagnosis (if they are diagnosed due to the onset of symptomatology). However, due to the increased accessibility and safety of obtaining imaging (diminished risk of radiation with magnetic resonance imaging as opposed to computed topography), an increasing number of asymptomatic lesions are being discovered (14 to 20%).[2,3] In these situations, the reasons for imaging can range from history of carcinoma or lymphoma to sinusitis.[2] The increased incidence of detection of a meningioma in an asymptomatic patient has placed a renewed focus both on the natural history of menin-

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giomas and on the importance of identifying firm indications to offer treatment (see the following discussion).

In the "symptomatic" patient population, most patients present with signs and symptoms attributable to mass effect and the tumor site. Headache, by far, is the most common symptom, occurring in anywhere from 39 to 48% of this patient population.[2,3] A history of seizures (20 to 34%) remains common in modern-day studies and is most often seen with temporal meningiomas.[2,3] Other site-specific symptoms can include confusion, memory loss, depression/personality alteration (frontal); motor or sensory deficit (perirolandic lesions); visual field deficits, including field cuts, disturbances in color perception and tracking objects (occipital); neglect, alexia, and difficulty with calculation (parietal); aphasias (dominant-sided pterional or posterior temporal lesions).

Radiographic Findings/Diagnostic Aids

Tremendous improvements in modern radiographic techniques have resulted in increasing the amount of information that can be gleaned preoperatively to help with surgical planning. Magnetic resonance imaging is the mainstay not only of diagnosis but also of surgical planning. On T1-weighted magnetic resonance, ~60% of meningiomas are isointense, whereas 30% are hypointense compared with gray matter. As with computed tomography (CT), meningiomas display strong enhancement with contrast administration; in addition, enhancement of the surrounding dural margin may be noted ("dural tail"). The significance of this dural tail has been controversial in the surgical management of these tumors. Many believe this to represent neoplastic infiltration of surrounding dura (and hence requiring resection), whereas others feel this represents more of a reactive inflammatory response. Correlative radiographic-histopathologic studies have demonstrated that in 65% of patients this dural tail represents tumor invasion, whereas in the remainder of patients only nonneoplastic meningothelial proliferation, hypervascularity, and vascular dilatation were seen.[4]

On T2-weighted magnetic resonance imaging (MRI), several important radiographic findings can be appreciated preoperatively. Information regarding the extent of demarcation between tumor and surrounding brain can be noted by carefully assessing for a "cerebrospinal fluid (CSF) cleft" around the lesion. T2-weighted imaging also allows an easier determination of the possibility of adjacent brain invasion, which can be evidenced by T2 hyperintensity (edema), and of the local vasculature, identified by vascular flow-voids. T2-weighted imaging can often foreshadow the nature of the tumor's consistency that may be encountered intraoperatively. Hyperintensity on T2-weighted imaging suggests a higher water content indicating a softer tumor, whereas hypointensity can indicate a fibrous or calcified tumor requiring different surgical considerations.

The extent of peritumoral edema can also be analyzed on T2-weighted imaging. The extent of edema is often an indication for treatment intervention. In addition, the presence of peritumoral edema can be associated with particular radiological and histological features affecting surgical treatment.[5] Most often, meningothelial, anaplastic, microcystic, and angiomatous subtypes display higher edema indices than other types. However, more importantly, studies have demonstrated two significant radiographic factors to be associated with peritumoral edema: vascular supply from pial-cortical arteries and cortical invasion (absence of a well demarcated T2 plane on MRI).

Although we do not typically obtain magnetic resonance angiography or conventional angiography for most convexity meningiomas, it can serve as an adjunct in the preoperative assessment of some tumors. It enables the surgeon to assess the extent and pattern of vascularity, the extent of tumor encroachment on vascular structures, and the feasibility of embolization. Occasional irregular patterns of vascularity can be discovered, such as vascular supply from the contralateral middle meningeal artery (**Fig. 15.1**).

Indications for Treatment

The decision to treat convexity meningiomas can be complex and must take into account several factors. The options of observation, surgery, or radiation are all relevant. The natural history or potential growth rate must be considered.[6,7] A recent article by Yano and Kuratsu demonstrated that 37% of meningiomas showed growth on imaging during observation for 3.9 years.[7] Furthermore, additional consideration is necessary based on the fact that the growth rate of meningiomas might be linear and not logarithmic (as seen with malignant astrocytomas).[6,7] However, in our experience, the growth of observed lesions can often be erratic (or intermittent) where lesions that have been stable on serial imaging can demonstrate relatively rapid growth within a short time period. The last issue to consider in deciding upon a period of observation is that higher-grade meningiomas can be missed, with subsequent delay in surgery resulting in potentially higher operative risk and possibly affecting the long-term outcome.

Although there have been several attempts in the literature to develop a decision-making algorithm for meningiomas (i.e., the "ABC" system and "CLASS" algorithm), these systems apply to cranial base meningiomas.[8] Our decision to treat is primarily based on the patient's age/medical status, tumor size, symptom complex, and associated edema. We have adopted an increasingly aggressive treatment philosophy considering the favorable long-term outcomes from convexity meningioma surgery—0% surgical mortality, ~3 to 5% morbidity (neurological and nonneurological), and 0% recurrence with grade zero resection and World Health Organization (WHO) grade I meningiomas.[2,3] We will typically observe lesions in asymptomatic patients who are over the age of 70 or have poor medical status and do not have any associated peritumoral edema. In younger patients who

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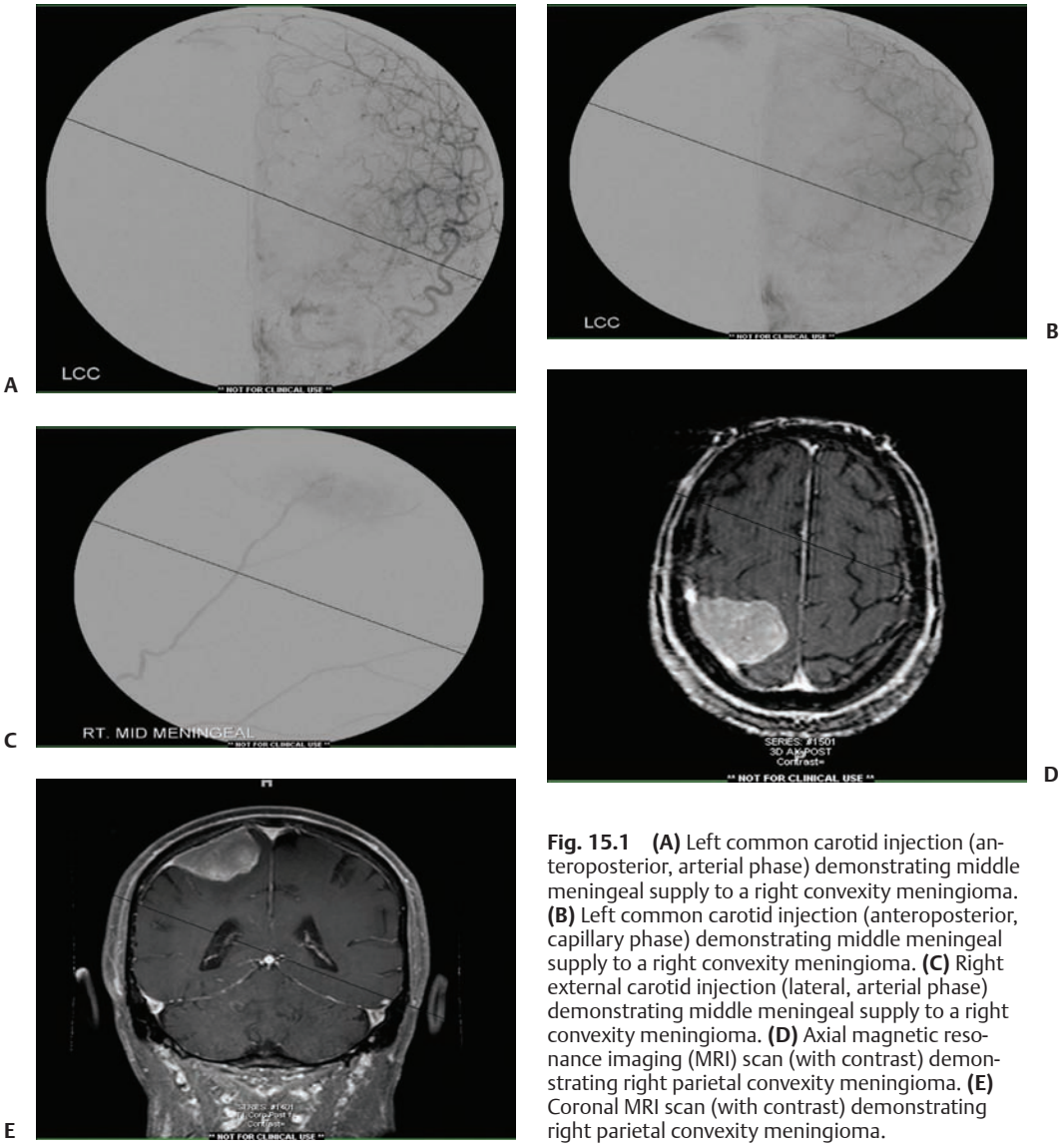


Fig. 15.1 **(A)** Left common carotid injection (anteroposterior, arterial phase) demonstrating middle meningeal supply to a right convexity meningioma. **(B)** Left common carotid injection (anteroposterior, capillary phase) demonstrating middle meningeal supply to a right convexity meningioma. **(C)** Right external carotid injection (lateral, arterial phase) demonstrating middle meningeal supply to a right convexity meningioma. **(D)** Axial magnetic resonance imaging (MRI) scan (with contrast) demonstrating right parietal convexity meningioma. **(E)** Coronal MRI scan (with contrast) demonstrating right parietal convexity meningioma.

are asymptomatic, with tumors smaller than 3 cm, and who do not have associated edema, we discuss the surgical and the conservative options and let the individual patient decide after presenting a detailed outline of the risks and benefits of each plan. We do have several patients who have been followed vigilantly, and only a small fraction of them have required subsequent surgery (without negative repercussions related to a "delayed" surgical treatment).

Otherwise, in most of the other patients we will recommend surgical intervention for diagnosis and complete resection. We are reluctant to recommend radiosurgery as the initial treatment for any tumor where histological grade is not known for fear of improperly treating WHO grade II and III meningiomas, where a combined surgical and radiation therapy approach is preferable.

Operative Considerations

The goal of operative intervention in patients with convexity meningioma should be a grade zero resection (tumor resection plus excision of a surrounding 2 cm dural margin).[9]

Preoperative Management

In patients with preoperative imaging demonstrating peritumoral edema, dexamethasone is started at least 2 weeks preoperatively in preparation for surgery. Anticonvulsants are started preoperatively only in those patients presenting with seizures, otherwise a loading dose of an anticonvulsant (i.e., levetiracetam) is given intraoperatively at the start of surgery.

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Operative Technique

Patient Positioning, Incision, and Craniotomy

The ease with which complete resection is obtained is primarily determined by the surgical exposure. Critical to obtaining this exposure is patient positioning and creation of an optimal craniotomy flap. The patient is positioned in such a way as to allow the surgeon to operate in a plane parallel to the floor. This allows the surgeon to work in an ergonomically comfortable position, in addition to ensuring that any blood pooling occurs at the bottom of the resection cavity—ensuring good visualization. Furthermore, the incision should be planned, using neuronavigation to ensure a large enough craniotomy can be created to encompass the entire tumor and surrounding dural margin. The scalp flap should be planned to avoid cosmetic deformity and to avoid division of vascular pedicles supplying the scalp. We typically perform a two-layer dissection during the exposure, harvesting a large pericranial graft that can be used for dural closure.

For frontal pole tumors, the patient is positioned supine with the head turned contralaterally (if necessary) to bring the surgical plane parallel to the floor. A unilateral craniotomy is performed via a bicoronal incision placed behind the hairline. For lateral sphenoid wing or frontotemporal tumors, the head is turned contralaterally ~45 degrees with the patient positioned supine. For such lesions, a standard pterional incision is performed, with the scalp and temporalis dissected separately. For tumors located in the posterior temporal, lateral parietal region, the patient is positioned supine with an ipsilateral shoulder roll; we prefer a large linear incision over a horseshoe incision to perform the craniotomy. For medial parietal or occipital lobe tumors, the patient can be positioned straight prone or park-bench with the head appropriately rotated. Depending on the size of the lesion, a linear incision or a mitar flap can be created to perform an appropriately sized craniotomy.

After scalp dissection with preservation of a pericranial graft, a craniotomy large enough to completely expose tumor and surrounding dura is performed. After creation of the flap, the underlying dura is carefully dissected free with a Penfield dissector. The bone flap is lifted gently to gain more space for dural dissection in an effort not to manipulate the underlying meningioma, which could inadvertently tear the dura and pia–capsule connections between the tumor capsule and surrounding cortex. Bleeding should be controlled with bipolar coagulation, hemostatic agents, or rapid dural incision/early devascularization. In this process, copious bleeding from the dural base of the meningioma can be encountered. Special consideration should be given the tumors with calvarial involvement. Attempting to dissect the tumor–calvarial interface with a Penfield dissection as with standard craniotomies can be difficult and even harmful to underlying brain parenchyma. For these tumors, neuronavigation (with CT and MRI) can be used to delineate the margins of the tumor–calvarial interface. Once this is delineated, then a series of burr holes ~2 mm apart surrounding the invaded bone are created, with the intervening bone removed by rongeur or drilling. Bleeding from the bone edges is controlled with bone wax. Subsequently, if necessary, a larger craniotomy flap can then be created to expose the remaining intradural component of the tumor. In addition, with tumors invading the calvarium, the overlying pericranium may also be infiltrated with tumor; hence this segment of the harvested pericranial graft must be excised and cannot be used in reconstruction.

Dural Incision and Early Devascularization

A dural incision is made circumferentially around the tumor with approximately a 2 cm margin from the contrast-enhancing component of the lesion based on MR neuronavigation. Meningiomas can be quite vascular, with vascular supply for convexity meningiomas coming from hypertrophied and tortuous branches of the meningeal arteries. Due to the possibility for significant blood loss, early devascularization is critical and facilitates a meticulous and bloodless extracapsular dissection as opposed to a hurried and bloody dissection. With convexity meningiomas, this step is easily performed at the time of dural incision where the tumor's vascular pedicles are encountered and sacrificed.

Internal Debulking and Capsular Dissection

After the dural incision, the majority of the operation is focused on tumor resection. Whereas small tumors can often be removed en bloc, medium-sized and larger tumors require an initial internal debulking before the extracapsular dissection to minimize brain retraction. For most debulking, the Cavitron Ultrasonic Surgical Aspirator (Integra Life Sciences, Plainsboro, NJ) remains the major tool (**Fig. 15.2**). However, its effectiveness is limited with fibrous or calcified tumors, and its use can be tedious with vascular tumors. In vascular tumors, the yttrium-aluminum-garnet laser can prove quite useful because it slices tumor tissue with direct contact while providing hemostasis. The process of debulking is performed until a thin rim of capsule remains.

The surgical microscope is critical to the extracapsular dissection stage where the goal is to dissect the capsule free from any attached pia while preserving all adjacent neurovascular structures (**Fig. 15.3**). This dissection commences with the identification of a preserved layer of arachnoid at the brain–tumor interface. As the dissection proceeds circumferentially around and toward the apex of the tumor, the capsule (as opposed to brain) is manipulated to any cortical attachments or adherent vascular structures. Any small pial–tumor adhesions are bipolared and sharply divided. As a general rule, during extracapsular dissection no artery is sacrificed unless the vessel is confirmed to be a tumor feeder. A majority of arterial structures encountered are often en passant vessels or loops of vessels that have become encased by tumor or adherent to the capsule. Lateral sphenoid wing

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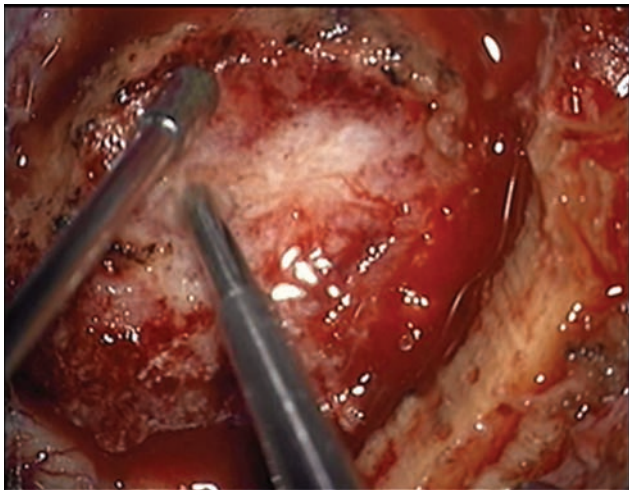


Fig. 15.2 Use of the Cavitron Ultrasonic Surgical Aspirator (Integra Life Sciences, Plainsboro, NJ) to debulk a right parietal convexity meningioma before extracapsular dissection.

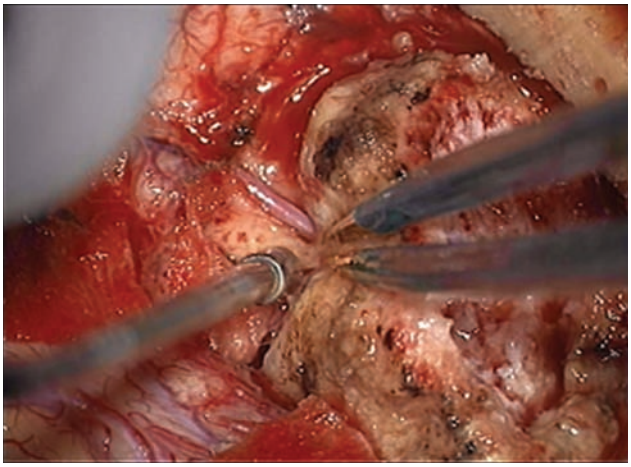


Fig. 15.3 Extracapsular dissection of a right parietal convexity meningioma. Meticulous dissection is necessary to preserve en passant vessels (i.e., the angular branch of the middle cerebral artery in this patient).

meningiomas can often dive into the sylvian fissure, engulfing middle cerebral artery (MCA) branches; convexity meningiomas rarely have feeders directly coming from large cerebral arteries; hence these branches must be preserved. Aside from arterial structures, sizeable cortical veins may be encountered and also must be carefully dissected to prevent local and distant venous infarction.

As this extracapsular dissection proceeds, the tumor–arachnoid plane must be maintained such that the arachnoid is dissected free from the tumor and not from the brain. In this process, cottonoid patties are sequentially and circumferentially placed to preserve already dissected brain-arachnoid and any major vascular structures (**Fig. 15.4**). Difficulty can be encountered with aggressive meningiomas with brain invasion. The decision to chase tumor into underlying parenchyma is based on whether the invaded cortex is eloquent or noneloquent. In this decision making, diffusion tensor imaging and cortical stimulation can serve as useful adjuncts to anatomically and functionally identify critical white fiber tracts. After complete dissection, the tumor is completely removed, exposing the tumor bed and patties.

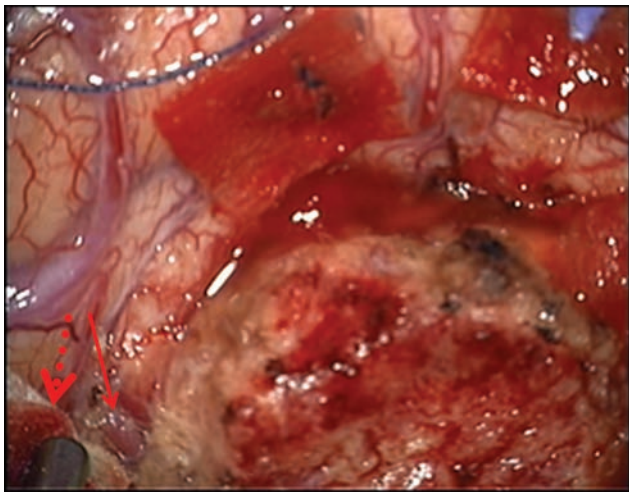


Fig. 15.4 Careful extracapsular dissection should proceed in an organized fashion using cottonoids (*dotted arrow*) to aid in skeletonizing and identifying vasculature (*single arrow*) before deciding whether they should be sacrificed.

Dural Reconstruction and Closure

After tumor resection, the surrounding dura is carefully inspected for any residual tumor. A watertight dural closure is obtained utilizing a harvested pericranial graft (preferable) or an artificial dural substitute. The dural suture line is then reinforced with glue (i.e., fibrin glue). For convexity meningiomas with calvarial invasion and resection, a recessed titanium cranioplasty is performed (**Fig. 15.5**).

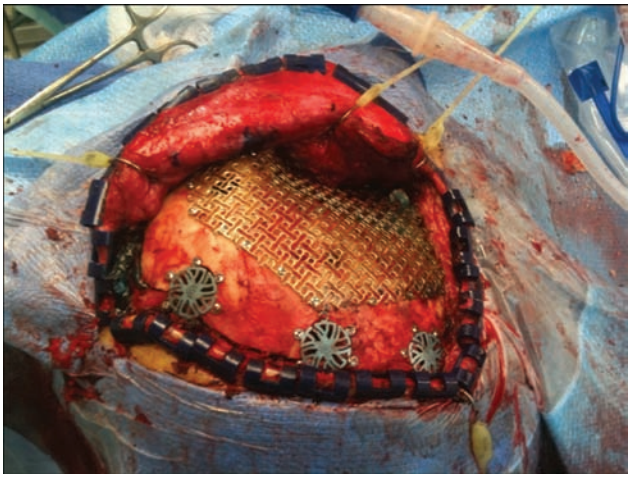


Fig. 15.5 A titanium cranioplasty is necessary for good cosmetic reconstruction after resection of a meningioma with calvarial invasion.

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Postoperative Management

Patients typically spend 1 night in the intensive care unit. The onset of a new postoperative neurological deficit or seizure must be investigated with vigilance, including a set of basic laboratory tests and imaging to rule out a hemorrhage or infarction secondary to intraoperative sacrifice of a critical arterial or venous structure. Otherwise, an enhanced MRI scan is obtained postoperatively to document the extent of resection.

Seizure prophylaxis is typically continued for at least 3 months postoperatively, at which point an electroencephalogram is obtained to rule out any epileptic focus. Meanwhile, in symptomatic patients or those with peritumoral edema, steroids are slowly weaned postoperatively over the course of days. Deep vein thrombosis prophylaxis with subcutaneous heparin is begun with the knowledge that patients with meningiomas have among the highest rates of thromboembolic complications in the neurosurgical population.

Surgical Outcomes: Morbidity and Mortality

Due to their anatomical location and ease of access, WHO grade I convexity meningiomas are considered a surgically curable entity. Modern-era neurosurgical studies demonstrate an overall recurrence rate of ~3 to 4%. Recurrence is primarily seen with atypical and anaplastic tumors or subtotally resected lesions.[10–14] Concurrently, survival analysis has demonstrated a 5-year survival of 90%.[2]

As expected, the mortality from convexity meningioma surgery is minimal, with most recently published studies reporting a 0% 30-day mortality rate. The incidence of surgical morbidity is ~1.7 to 3%. These complications include new-onset neurological deficit/seizure, surgical site hematoma, CSF leaks, and wound infection.

Management of Residual and Recurrent Disease

The management of residual tumor is primarily an issue with grade II and III tumors with or without brain invasion; otherwise, for the small group of subtotally resected grade I meningiomas, we perform regular follow-up imaging to assess for growth. Our approach is to utilize irradiation in the form of focused stereotactic radiation for these meningiomas. Controversy exists around the issue of postoperative radiation therapy. Modha and Gutin have suggested that grade II and III tumors that are either subtotally excised, invade the brain, or have a mindbomb homog-1 (MIB-1) index greater than 4.2% should be treated with radiation.[15]

The mean time to recurrence has been reported to be anywhere from 11 to 14 months—highlighting the importance of close imaging follow-up.[2,4] Repeat microsurgical resection, when needed, is undertaken in young patients (where the long-term effects of radiation are best avoided), symptomatic patients with associated edema, or WHO grade II and III meningiomas that have demonstrated growth despite postoperative radiation. Otherwise, stereotactic radiation can be used for the remainder of patients with demonstrated recurrence. Although a majority of the radiosurgical literature has been focused on meningiomas in general, several studies assessing the utility of focused radiation in convexity meningiomas have demonstrated actuarial tumor control rates of nearly 71% at 5-year follow-up.[16]

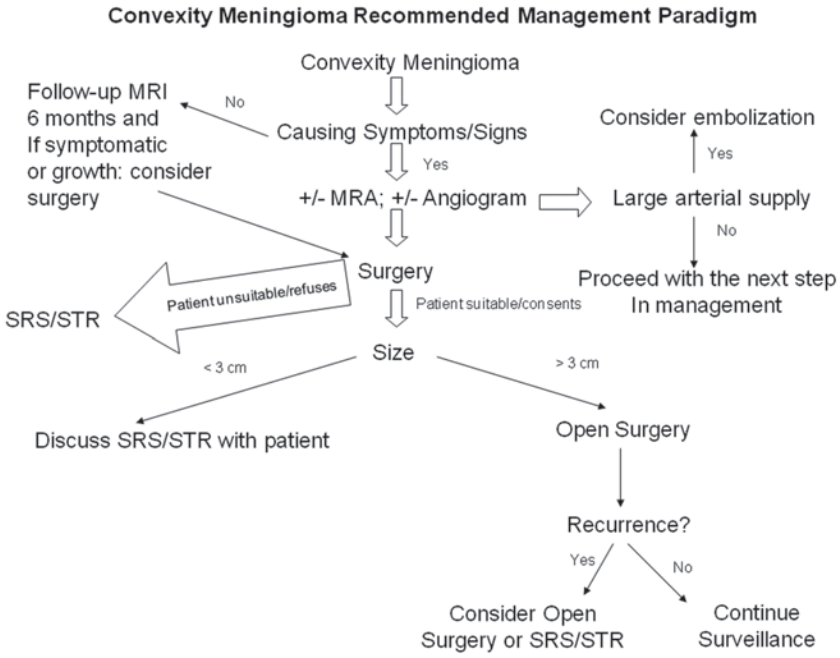


Fig. 15.6 Convexity meningioma recommended management paradigm. MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; SRS/STR, stereotactic radiosurgery.

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Conclusion

Convexity meningiomas represent an anatomical subset of meningiomas that lend themselves to surgical cure. The decision to treat these lesions is more of an art than a science, with this process based on the patient's medical status, symptom complex, radiographic findings, and concern for the possibility of a higher-grade lesion. The goal of surgical intervention is grade zero resection with removal of the tumor with a surrounding dural margin. Critical to the success of surgical intervention and minimization of morbidity is good exposure and meticulous extracapsular dissection with preservation of arterial and venous structures. The surgical expectations are for no recurrence with minimal surgical morbidity. Postoperative management primarily consists of close follow-up to confirm lack of recurrence in totally resected tumors and when to treat residual and recurrent disease.

Based on currently available data and our institutional surgical experience, our overall treatment paradigm for convexity meningiomas is summarized in **Fig. 15.6**. For all asymptomatic lesions, we prefer to observe for signs of growth or the development of edema or symptoms. The management of symptomatic lesions and the decision to treat with initial surgical resection versus radiosurgical treatment are dependent upon a myriad of factors, including size, patient suitability for surgery (e.g., medical comorbidities, age), and patient preference.

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Chapter 16

Parasagittal Meningiomas

Gustavo Pradilla, Carlo L. Solero, and Francesco DiMeco

Introduction

Parasagittal meningiomas are complex lesions with a wide spectrum of clinical and surgical nuances. Retrospective single-center clinical series and technical case reports make up the majority of the literature to date, and lack of class I evidence has hindered consensus on clinical management, surgical treatment, and adjuvant therapeutics for these lesions. In 1938 Cushing and Eisenhardt[1] first defined parasagittal meningiomas as those that fill the parasagittal angle, without brain tissue between the tumor and the superior sagittal sinus (SSS). These tumors may involve one, two, or all the SSS walls and may or may not completely occlude flow. Since that description, advances in diagnostic neuroradiology, larger clinical series, and novel surgical techniques have increased our ability to diagnose these lesions at earlier stages and have facilitated our ability to achieve more radical resections with preservation or restoration of vascular flow. Furthermore, development of interventional neuroradiology techniques has increased our understanding of cerebral venous hemodynamics, and stereotactic radiosurgery continues to improve our ability to treat residual or recurrent disease. The following chapter reviews our current understanding of the diagnostics and natural history of these challenging tumors and summarizes modern surgical and adjuvant treatment algorithms.

Epidemiology

Parasagittal meningiomas represent 21 to 31% of intracranial meningiomas. Cushing and Eisenhardt[1] and Olivecrona[2] proposed the first classification of these tumors according to their location along the SSS. The reported incidence of tumors located in the anterior third (between the crista galli and the coronal suture) has ranged from 14.8 to 33.9%, tumors located in the middle third (from the coronal to the lambdoid suture) range from 44.8 to 70.4%, and those located in the posterior third (from the lambdoid suture to the torcula) range from 9.2 to 29.6%.[3] In a clinical series by Black and colleagues, tumors involved the anterior third of the SSS in 12.8% of the cases, the middle third in 69.2%, and the posterior third in 17.9%. Tumors in this series were preferentially located on the right side (59% vs 33.3%), and bilateral tumors presented in only 7.7% of cases.[4]

Pathological Findings

Parasagittal meningiomas tend to occur where arachnoid granulations are denser, with ~15% of tumors presenting with invasion of the SSS.[5] A higher incidence of atypical and malignant meningiomas has been reported in the parasagittal region when compared with meningiomas in other locations.[6] In some series, the percentage of malignant and atypical lesions is 3.7% and 14.8%, respectively.[7] In a surgical series of 108 patients previously presented by the authors, benign, mostly transitional meningiomas (World Health Organization [WHO] grade I) were identified in 86 patients (79.6%); "atypical" meningiomas (WHO grade II) were diagnosed in 16 patients (14.8%), and malignant meningiomas (WHO grade III) were diagnosed in four patients (3.7%). Whereas grade I lesions were significantly more frequent among women, grade II and III lesions as well as hemangiopericytomas prevailed among men. Since publication of this series, 76 more patients have been acquired (total of 184). Of these, 151 patients (82%) were WHO grade I, 26 patients (14.1%) were WHO grade II, and five patients (2.7%) were WHO grade III. **Table 16.1** illustrates the pathological characterization of tumors in this series.[8]

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Table 16.1 Histopathological Types of Parasagittal Meningiomas

Histopathological Findings	
Histological Type	**No. of Cases**
Transitional (mixed)	87
Fibrous (fibroblastic)	28
Atypical	26
Meningothelial	21
Psammomatous	6
Malignant	5
Secretory	4
Microcystic	2
Hemangiopericytoma	2
Angioblastic	2
Chordoid	1
Total	184

From DiMeco et al.[8]

Natural History

In 1957 Simpson[9] described infiltration of the SSS as a major factor related to recurrence of parasagittal meningiomas. Since then it is known that the extent of surgical resection and pathological grade correlates with the rates of recurrence, although residual parasagittal tumors may remain stable over time, and effective predictive factors are yet to be established.[5] Among the subtypes of WHO grade I tumors, psammomatous tumors with a high density of calcification rarely recur,[10] and angioblastic meningiomas appear to have markedly higher rates of recurrence.[11–13]

Clinical Presentation

Symptoms

Presenting symptomatology is largely related to the proximity of the lesion to the Rolandic fissure. As illustrated by Cushing's description of General Leonard Wood's case,[1,14] these patients typically present with sensory or motor seizures involving the contralateral lower extremity. After seizures, contralateral hemiparesis constitutes the second most common presenting symptom, followed by paresthesias, papilledema, and dementia (**Table 16.2**). Tumors arising from either the anterior or posterior third, however, can remain undetected for long periods of time until mass effect triggers noticeable symptoms. Lesions in the anterior third occasionally present with a long-standing history of headaches or a frontal lobe syndrome, whereas posterior third–based lesions may present with homonymous hemianopsia. The mass effect exerted by tumors arising in the middle third on the precentral and postcentral gyri, and on the paracentral lobule, triggers earlier symptomatology and facilitates faster diagnosis, resulting in smaller lesions upon presentation.[3] Before the advent of magnetic resonance imaging (MRI), midline calvarial bossing was considered to be a cardinal sign of a large parasagittal meningioma (**Fig. 16.1**). In a series of 154 parasagittal meningiomas, anterior third tumors most often presented with headaches (36%) or mental status changes (36%), whereas posterior third tumors presented with headaches (36%), visual symptoms (21%), focal seizures (21%), or mental status abnormalities (21%).[15]

In a recent series of combined convexity and parasagittal meningiomas by Black et al, most patients who received surgical treatment were symptomatic at the time of surgery, with headache constituting the most prevalent symptom (35.9%), followed by motor findings (38.4%), and seizures (30.1%). The number of asymptomatic patients constituted 28.2% in that surgical series.[4]

Anatomical Considerations

Cross-sectional analysis of the SSS reveals a basic triangular shape that gradually increases in size as it extends distally toward the posterior third.[16] The SSS communicates laterally with irregular interdural venous lacunae, which lie on either side of the dura mater and are occasionally accompanied by arachnoid granulations. These structures often appear as a filling defect or an intrasinusal mass on MRI. Eight to 12 external medial cortical veins and a similar number of internal cortical veins carry out most cortical venous drainage on each hemisphere.[3]

Angiographic analysis of 100 patients by Apuzzo et al showed that most parasagittal veins (70%) join the SSS in a segment of the sinus located between the coronal suture and 2 cm behind it.[17] Furthermore, 53 of 100 SSS studied angiographically by Yamamoto and colleagues[18] had venous tributaries that entered the sinus within 2 cm of the coronal suture, with 76% of these entering within 2 cm behind the coronal suture.

The anterior half of the SSS is narrower and has fewer associated venous lacunae, fewer pacchionian bodies, and smaller numbers of adjoining cortical veins entering the sinus than the posterior half, which facilitates surgical exploration.

Collateral venous blood flow was studied in 242 cases of parasagittal meningiomas by Tigliev et al.[19] This study showed that, whereas in anterior third tumors 52.1% of cases had collateral blood flow through cortical veins, in middle or posterior third tumors, collateral drainage occurred through cortical veins in 67% of cases. Collateral blood flow through the extracerebral veins occurred in 56% of patients regardless of the location along the sinus.

Furthermore, in a study by Oka et al, collateral blood flow was also identified through meningeal veins and

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Table 16.2 Common Presenting Symptoms in Parasagittal Meningiomas

	Location along the Superior Sagittal Sinus			
	Anterior Third (50 cases)	**Middle Third (89 cases)**	**Posterior Third (14 cases)**	**Total (153 cases)**
Focal seizures	1 (2%)	29 (33%)	3 (21%)	33 (22%)
Generalized seizures	13 (26%)	10 (11%)	-	23 (15%)
Headache	18 (36%)	9 (10%)	5 (36%)	32 (21%)
Confusion/cognitive decline	18 (36%)	6 (7%)	3 (21%)	27 (18%)
Monoparesis (leg)	2 (4%)	23 (26%)	-	25 (16%)
Hemiparesis	-	6 (7%)	2 (14%)	8 (5%)
Monoparesis (arm)	1 (2%)	1 (1%)	-	2 (1%)
Visual symptoms	1 (2%)	4 (5%)	3 (21%)	8 (5%)
Calvarial deformity	6 (12%)	2 (2%)	-	8 (5%)
Dysphasia	-	4 (4%)	-	4 (3%)
Stroke	-	2 (2%)	-	2 (1%)
Vertigo	-	1 (1%)	-	1 (1%)
Other symptoms	7 (14%)	11 (12%)	3 (21%)	21 (14%)

Adapted from Gauthier-Smith.[15]

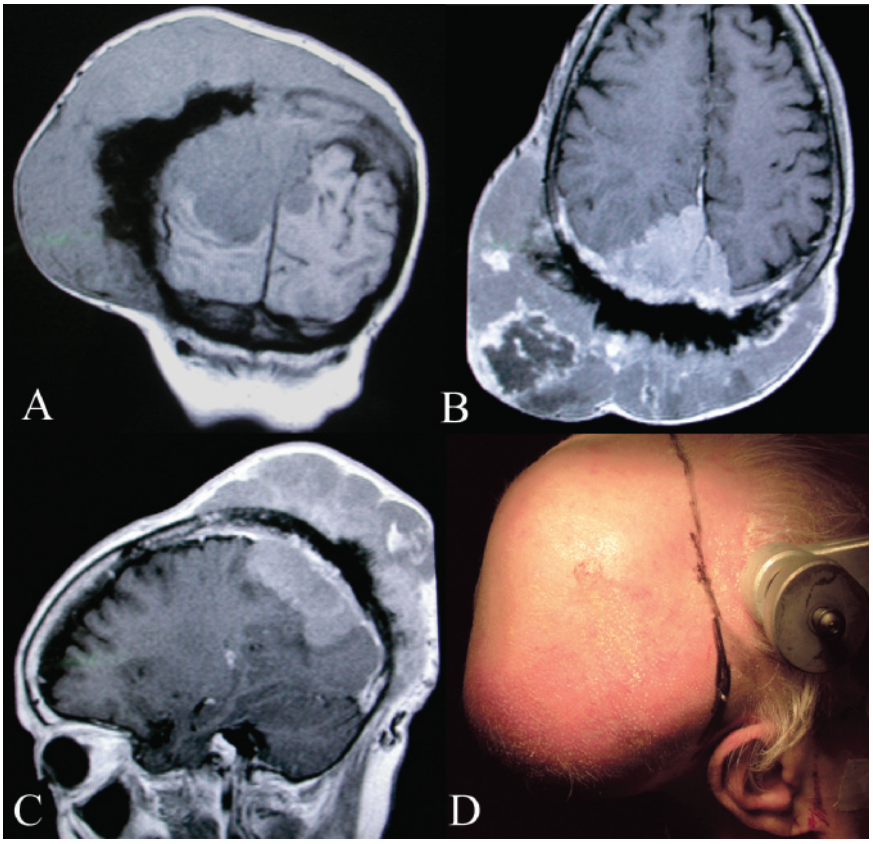


Fig. 16.1 **(A–C)** Coronal, axial, and sagittal magnetic resonance imaging views showing a large, extraaxial contrast enhancing mass involving the posterior third of the sagittal sinus and the skull of a 72-year-old female who presented with a 7-month history of progressive midline parietal calvarial bossing, slightly more pronounced on the right side. **(D)** Photograph taken during the semi-sitting positioning of the patient showing a large calvarial bossing fashioning a sort of "chapeau de clown" head deformity.

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end-to-end anastomoses of superficial cerebral veins.[20] These superficial cerebral veins are classified into four groups depending on the cortical area drained by them: with a superior sagittal group that drains the superior part of the medial and lateral surfaces of the frontal, parietal, and occipital lobes and the anterior part of the basal surface of the frontal lobe and empties into the SSS; a sphenoidal group that drains into the sphenoparietal and cavernous sinuses; a tentorial group that converges on the tentorial sinuses; and a falcine group that empties into the inferior sagittal or straight sinus.[20] Andrews et al also presented an anatomical classification based on cadaveric analysis of 10 human brains,[21] in which the parasagittal veins were divided into anterior frontal, posterior frontal, parietal, and occipital. In that study the anterior frontal parasagittal region had an average of 6.5 veins on each hemisphere, the posterior frontal region had three veins, the parietal region had four veins, and the occipital region had one vein.

The results of these studies suggest that resection of the anterior third of the sinus has limited impact on the overall hemispheric venous drainage in most cases, and that sacrifice of the middle third of the SSS significantly affects venous return. Disruption of venous structures in the posterior third results in less restrictive venous drainage than sacrifice of middle third structures, but the true effect of these changes is directly related to the caliber of the sacrificed vessel and the cortical region draining through it.[16,22,23]

Classifications

Bonnal and Brotchi first provided a surgical classification of parasagittal meningiomas in 1978 that included eight tumor subtypes.[24] Since then this classification has been modified and now describes five tumor subtypes developed to facilitate resection strategies.[5] In this classification system type I lesions are those that only attach to the outer surface of the sinus wall. Type II lesions enter the lateral recess of the SSS. Type III lesions are those that invade one wall of the SSS. Type IV tumors have already invaded two sinus walls but have not compromised patency, and type V tumors are those that spread over the midline and invade all the walls of the sinus, resulting in complete occlusion (**Table 16.3**).

Table 16.3 Brotchi Classification

Type I	Tumor is attached to the outer surface of the sinus
Type II	Tumor enters the lateral recess of the SSS
Type III	Tumor invades one wall of the SSS
Type IV	Tumor invades two walls of a still patent sinus
Type V	Tumor spreads over the midline, invades the three walls, and occludes the SSS

Modified from Hancq et al.[5]

Table 16.4 Sindou and Alvernia Classification

Type I	Tumor attaches to the outer surface of the sinus wall
Type II	Tumor fragment inside the lateral recess
Type III	Tumor invades the ipsilateral wall
Type IV	Tumor invades the lateral wall and roof
Type V	Complete sinus occlusion with one free wall
Type VI	Complete sinus occlusion without any free walls

From Sindou and Alvernia.[7]

Sindou and Alvernia have proposed a similar classification that attempts to guide surgical decision making and preoperative planning based on six categories. Type I lesions present with attachment to the outer surface of the sinus wall but without disruption of the wall or intrasinusal invasion. Type II lesions are those with tumor invading the lateral recess but without invading the lateral wall. Type III lesions show invasion of the ipsilateral sinus wall. Type IV lesions are those with invasion of both the lateral wall and the roof of the SSS. Types V and VI reflect a complete sinus occlusion, with or without one free wall, respectively[3,7] (**Table 16.4**).

Differential Diagnosis

A myriad of lesions can affect the parasagittal region, ranging from meningiomas of all histopathological grades, to hemangiopericytomas, lymphomas, metastatic disease, and extramedullary hematopoiesis, among others.[13] The consistency of findings on imaging studies, however, greatly facilitates diagnosis. Similarly, intraoperative appearance of these lesions and frozen-section analysis are characteristic, can quickly confirm the diagnosis and contribute to intraoperative decision making.

Diagnostic Evaluation

Computed Tomography

Computed tomography (CT) is particularly beneficial in cases in which either hyperostotic or lytic calvarial changes are expected because it can aid in planning of the craniotomy flap. In addition, in cases in which bone flap replacement is hindered by marked tumor invasion, CT data can be used to fashion precise prosthetic implants for cranioplasty.[5]

MRI/MRA/MRV

MRI with and without contrast remains the imaging study of choice for evaluation of parasagittal meningiomas. MRI provides specific information on the size and

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consistency of the lesion and on its relationship with the falx, the meninges, the surrounding cerebral cortex, and the vascular structures involved (**Fig. 16.2**). Magnetic resonance angiography (MRA) in combination with contrast-enhanced MRI is now considered the gold standard in most centers.[5] MRA provides accurate, noninvasive visualization of the arterial and venous anatomy, which facilitates analysis of sinus patency and invasion, and visualization of collateral venous drainage patterns that develop following sinus occlusion. MRA exhibits some advantages over DSA due to its noninvasive nature and its ability to detect multidirectional flow. Although a comprehensive overview of the major dural sinuses and their patency can be obtained with two-dimensional (2-D) phase contrast imaging, more specific details on cortical venous anatomy can be seen with 3-D phase contrast sequences.[5] Magnetic resonance venography (MRV) can provide preoperative insight on venous infiltration and visualize collateral venous anastomoses; in one study, up to 87% of the collateral venous anastomoses secondary to parasagittal meningiomas were visualized.[25]

Furthermore, through additional image reconstruction from 2-D and 3-D sequences, information regarding direction of flow in a given vascular structure can also be obtained. The MRA data obtained is in some cases, however, limited when compared with digital subtraction angiography (DSA), particularly in visualization of the arterial supply to the tumor and in confirming sinus patency in cases with high degrees of obstruction where sinus flow is scarce and slow. Therefore, both techniques are used in a complementary fashion in cases requiring clarification, and in those in which embolization is considered.[5]

The use of functional MRI and fiber tractography has been reported and could be beneficial in patients with parasagittal tumors that invade beyond the pial surface.[4]

Digital Subtraction Angiography

Before surgery, visualization of venous anatomy is fundamental in operative planning. Details on patency of the sinus, arterial tributaries to the tumor and its relationship with cortical structures, and location of cortical draining veins and their point and angle of entry into the SSS are carefully considered.

Depression of the internal cerebral veins is frequently seen in parasagittal tumors, and the shape of the vessel was used to guide angiographically assisted localization before CT and MRI imaging. Marc and Schechter described multiple angiographic findings related to venous flow rerouting secondary to partial or complete SSS occlusion, including nonvisualization of the occluded segment, failure of cortical veins to reach the SSS, delayed emptying of veins at the site of obstruction, and reversal of normal venous flow with collateral venous channels connecting the SSS with other venous structures, such as the lateral sinus and the middle cerebral vein.[26] The thalamostriate vein can also be depressed in the more anteriorly located lesions; this finding was often referred to as "closing of the venous angle."

For large meningiomas (> 5 cm), determination of arterial feeding branches to the tumor, which may arise from the anterior or middle cerebral arteries, can facilitate preemptive intraoperative devascularization by selective

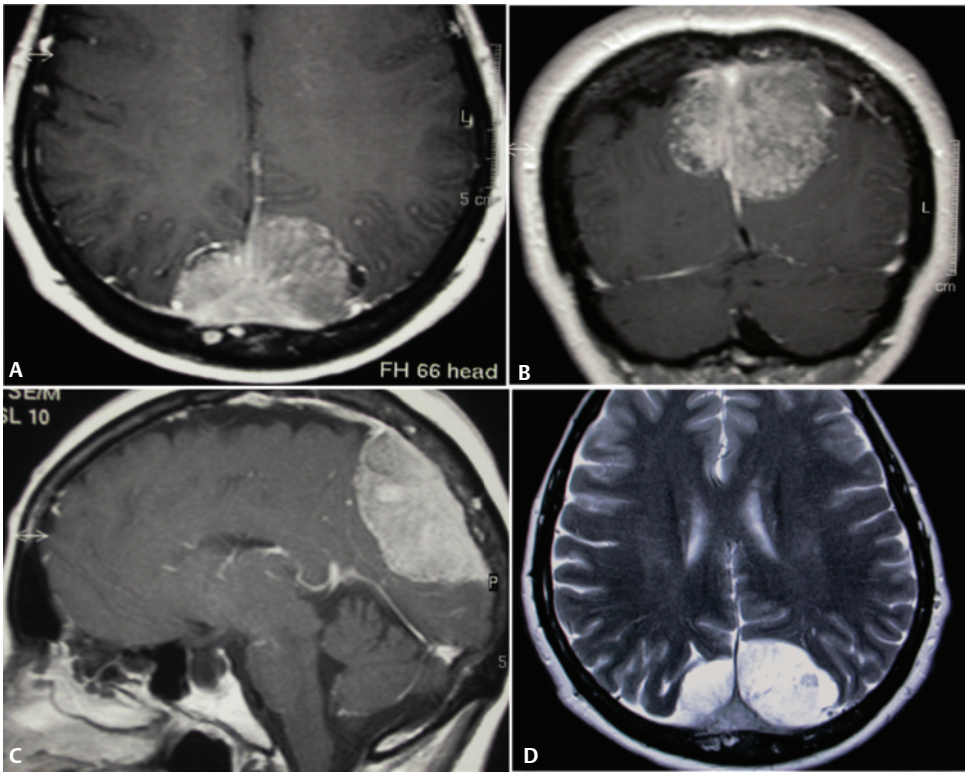


Fig. 16.2 **(A–C)** T1 sagittal, axial, and coronal views showing a bihemispheric extraaxial enhancing mass in close relationship with the middle third of the superior sagittal sinus in a 52-year-old woman who presented with a history of headaches. **(D)** T2 sequence demonstrates complete obliteration of the sinus.

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embolization[4] (**Fig. 16.3**). DSA is an invasive diagnostic procedure with variable morbidity risks that can vary significantly among operators and between centers; for this reason some authors do not advocate its use in a routine manner unless preoperative embolization is being considered.[5]

Surgical Indications

The slow-growing nature of most parasagittal meningiomas, combined with our ability to conduct close radiological follow-up with serial MRI/MRA imaging and to treat residual or recurrent disease with radiosurgery,[27] has significantly changed the surgical indications for these lesions and modified the goals of surgery.[5] Tumors involving critical cortical veins or partially patent sinuses can be subtotally resected, and the remnants can be followed radiographically or treated with adjuvant radiosurgery. If the lesion recurs, a conservative approach to allow for proper collateral circulation to be established and for progressive sinus thrombosis to occur can significantly decrease morbidity.[5,20] The long-term recurrence seen by some high-volume centers despite gross total resection (GTR) of sinus-invading tumors followed by graft repairs has also raised questions about the benefits of radical resection and grafting, and the frequency of sinus reconstruction procedures appears to be decreasing. Black and colleagues recommend yearly MRI follow-up for patients older than age 65 who are asymptomatic or have tumors less than 3 cm in diameter.[4]

Hancq and colleagues now propose conservative extrasinusal tumor resection of lesions invading a partially patent sinus, leaving residual intrasinusal tumor and conducting yearly MRI/MRA follow-up with adjuvant radiosurgery if tumor progression is observed.[5] They reserve intrasinusal tumor removal for lesions with completely obliterated sinuses, in which robust collateral flow has reestablished venous drainage over time. This treatment algorithm is shared by the authors and continues to be followed by several centers.[28,29]

Preoperative Considerations

Endovascular Interventions

Embolization

Preoperative embolization has been used as an adjuvant therapy to reduce intraoperative blood loss and decrease surgical time in meningioma surgery.[30–33] The role of palliative embolization, however, remains controversial. Tumor edema or hemorrhage has been reported after embolization of meningiomas with polyvinyl alcohol (PVA) particles, and the safety and efficacy of stand-alone embolization of meningiomas have been questioned.[34] The potential complications of embolization for these lesions, however, can be devastating.[35,36]

After a diagnostic arteriogram is obtained, blood supply to the tumor is identified, and the safety and feasibility of embolization are determined. Using real-time

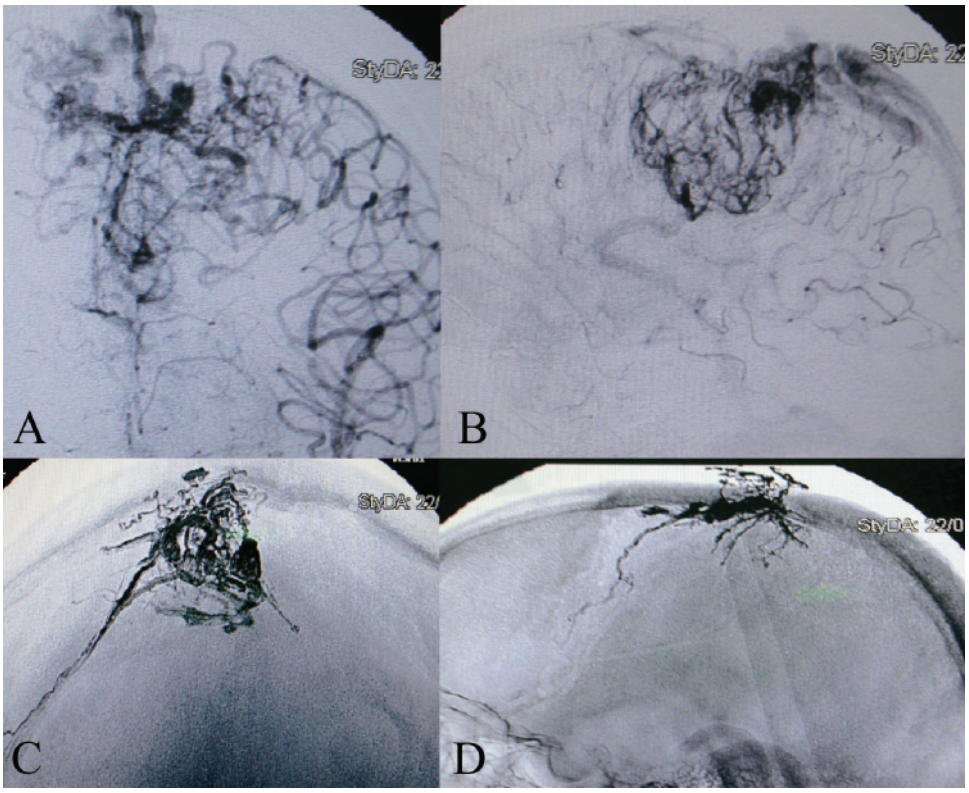


Fig. 16.3 Left middle third parasagittal meningiomas partially invading the superior sagittal sinus of a 32-year-old male with a history of recent onset of right leg motor impairment. **(A,B)** Preoperative angiogram demonstrating arterial feeders and dural arteriovenous fistulas. **(C,D)** Embolization with Onyx (ev3 Neurovascular, Irvine, CA) of the fistula and neoplastic mass.

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digital-subtraction fluoroscopy or "road-mapping" technology, a microcatheter is navigated through the larger diagnostic angiography catheter and directed over a microguidewire to the artery supplying the meningioma. Superselective angiography is performed through the microcatheter to verify the location and identify any normal branches, which would preclude safe embolization.[37,38] The embolic material is injected under continuous real-time digital-subtraction fluoroscopy to allow penetration into the tumor bed, which will result in devascularization and subsequent necrosis.

The use of new embolization materials such Onyx (ev3 Neurovascular, Irvine, CA), in combination with superselective catheterization, has increased our ability to perform these procedures safely.[34] Nonetheless, larger series and randomized trials are needed to determine specific safety and efficacy for parasagittal meningiomas.

Stenting

In most cases, venous collateralization typically prevents the onset of intracranial hypertension as a result of sinus occlusion from invading tumor. If intracranial hypertension presents, surgical decompression and tumor resection are attempted. In some cases, additional comorbidities, poor functional status, or advanced age may prevent surgical treatment. Similarly, partially resected lesions can also exhibit significant recurrence and associated intracranial hypertension. An alternative palliative method for intracranial hypertension therapy is ventriculoperitoneal shunt (VPS) placement, which can decrease intracranial pressure until further venous collateralization develops or additional aggressive surgical treatment is performed.[39] If intracranial hypertension were to occur despite VPS placement, venous outflow obstruction can be relieved by endoluminal stent placement.[39,40] Higgins et al[39] reported the case of a patient with a recurrent falcine meningioma that developed persistent intracranial hypertension from occlusion of the straight sinus and of the sinus confluence point in the posterior third of the SSS. This patient was treated with transvenous stenting of the SSS into the right transverse sinus via a left jugular puncture. The patient required full anticoagulation for 2 months thereafter and remained on low-dose aspirin after that. Intracranial hypertension symptoms had significantly improved at 3 months, and the patient continued to do well on the 9-month follow-up evaluation. Ganesan et al[40] also described their experience with a posterior third parasagittal meningioma with sinus invasion and intracranial hypertension. The patient responded to transvenous stenting with an Omnilink balloon-mounted stent (Guidant Corp., Indianapolis, IN), with significant improvement at 3 months postprocedure. In this case the patient was able to receive stereotactic radiosurgery after stenting without the risk of additional postradiation edema. Despite the inherent risk of poststenting thrombosis and the need for anticoagulation followed by antiplatelet therapy, venous sinus stenting constitutes a viable salvage therapy for flow restoration.

Operative Approach

Patient Position

The location of the lesion along the SSS dictates the position. Patients with tumors located in the anterior third are preferentially positioned supine with the head slightly flexed.[4] Those with lesions in the middle third can be positioned supine or lateral with the area overlying the tumor positioned as the highest point in the vertical plane. Patients with tumors in the posterior third can be positioned in the semisitting, lateral, three-quarter prone, or prone position with the tumor below the horizontal plane. This takes advantage of gravity-mediated retraction of the ipsilateral cerebral hemisphere, which facilitates dissection, particularly in lesions with significant falcine attachments.[5,41,42] In the authors' experience the semisitting position has been successfully used for lesions located on the middle and posterior third of the sinus. This position maintains the midline in a vertical plane and facilitates anatomical orientation.

Intraoperative Neuronavigation and Neurophysiological Monitoring

Frameless stereotactic image guidance has significantly impacted our approaches to parasagittal meningiomas. It allows for smaller incisions, tailored craniotomies, and anatomical localization of eloquent cortex, which translates into decreased morbidity, faster healing times, and improved cosmesis.[4] This technology is particularly useful in tumors that violate the pial surface and complements preoperative planning studies.

Surgical Incision

Patients are secured in a three-point fixation head holder. A modified bicoronal skin incision is made for tumors in the anterior third of the SSS. A trapdoor or horseshoe-shaped incision is made for tumors on the middle or posterior third of the SSS (**Fig. 16.4**). The incision extends at least 2 cm across the midline and is placed with careful dissection of the subjacent pericranium, which can later be used for dural reconstruction.[5] When planning the incision and scalp flap elevation, the location of prominent scalp and diploic veins formed through collateralization must be observed because brisk bleeding can be encountered, and early obliteration of this collateral flow can result in significant cerebral edema.[3]

Craniotomy

The technique and location of the parasagittal craniotomy are of paramount importance in these lesions. Most authors recommend multiple burr holes, but consensus regarding the specific placement of the parasagittal burr holes is lacking. Some surgeons prefer to place burr

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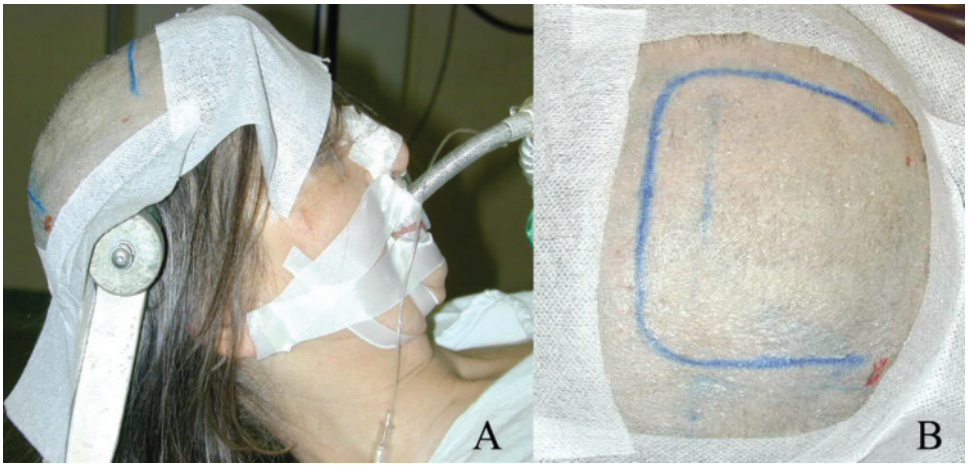


Fig. 16.4 A 45-year-old female with a history of headaches and recent onset of mild left hemiparesis. Magnetic resonance imaging showed a right extraaxial parasagittal contrast-enhancing mass partially invading the middle third of the SSS. **(A)** The patient is positioned in a semisitting fashion **(B)** Right frontoparietal horseshoe skin incision extending 1.5/2 cm across the midline.

holes right over the SSS by using controlled drilling with small drill bits and conduct the dural release from the inner table in a medial to lateral direction. Others prefer to place burr holes on both sides of the sinus and perform the dural dissection and the release of the sinus wall from lateral to medial until both parasagittal burr holes are communicated epidurally. Black and colleagues advocate for elevation of the bone flap in two stages, with the first flap located on the side of the tumor ~1 cm lateral to the SSS. Following dissection of the dura overlying the sinus from the bone, the second flap is elevated crossing the midline to the contralateral hemisphere.[4]

The senior authors have previously reported their experience with parasagittal craniotomies using an oscillating saw (TPS Micro Oscillating Saw, Stryker Instruments, Kalamazoo, MI, or GB128 Oscillating Saw, Aesculap Co., Tuttlingen, Germany), without burr hole placement.[43] The oscillating saw can be particularly useful when extending the craniotomy across the venous sinuses (**Fig. 16.5**) because dissection of the sinus wall from the inner table can be performed under direct visualization.[29,43] Use of the Control Depth Attachment (CDA) available for the Anspach drill (Anspach Effort, Inc., Palm Beach Gardens, FL) has further refined this technique. The length of this attachment can vary from 1 to 5 mm, and it allows the surgeon to cut through the outer table and the cancellous bone while sparing the inner table. The inner table is then fractured with an osteotome, which is used along the perimeter of the craniotomy. Great care is taken when the osteotome is used over the sinus (this segment can be avoided by continuing the osteotomy to the opposite side).

Regardless of the technique used for cranial perforation and osteotomy, the parasagittal bone flap must be centered over the tumor utilizing frameless stereotactic guidance and surface landmarks. It should expose an area 2 to 3 cm anterior and 2 to 3 cm posterior to the tumor margins. It must cross the midline and should expose the contralateral hemisphere at least 2 cm beyond the edge of the sinus or the lateral extent of the tumor, whichever is more lateral. Direct visualization of the sinus wall during dissection remains as the primary principle of the craniotomy. Special attention is given to the contralateral dura and adjacent veins, and osteotomies over the SSS are performed last.[5]

Elevation of the bone flap can be complicated by engorged diploic anastomosis and by frequently encountered invasion of the dura and bone by the tumor. In cases of severe tumor-induced hyperostosis, the craniotomy flap can be planned around the tumor, and a central portion of bone can be left attached to the tumor until better SSS control can be obtained later in the dissection. Following elevation of the bone flap, hemostatic agents such as Gelfoam (Pfizer, Inc., NY) and Surgicel (Ethicon, Inc., Somerville, NJ) can be applied over the sinus and stabilized with cotton strips and gentle pressure. Careful monitoring of the precordial Doppler, pCO_2, oxygenation, and hemodynamic parameters is particularly needed at this point because most air emboli will present during or immediately after flap elevation. Placing tacking sutures along the edge of the craniotomy can control intraoperative and postoperative epidural bleeding. If precordial Doppler or pCO_2 anomalies indicate air embolism, a Valsalva maneuver enacted by the anesthesiologist can reveal the site of air penetration.

Dural Opening

A semilunar dural flap based along the SSS is elevated under direct microscopic visualization of the pial surface to avoid injury to cortical draining veins. The contralateral dura is opened in the same fashion but only when the tumor significantly involves the contralateral hemisphere.

Tumor Resection

Dissection is initiated by visualization and preservation of the tumor capsule on the lateral margins. If the lesion is extrapial, a surgical plane can be obtained and the space between the pia and the capsule can be developed with cottonoid strips, which are placed circumferentially

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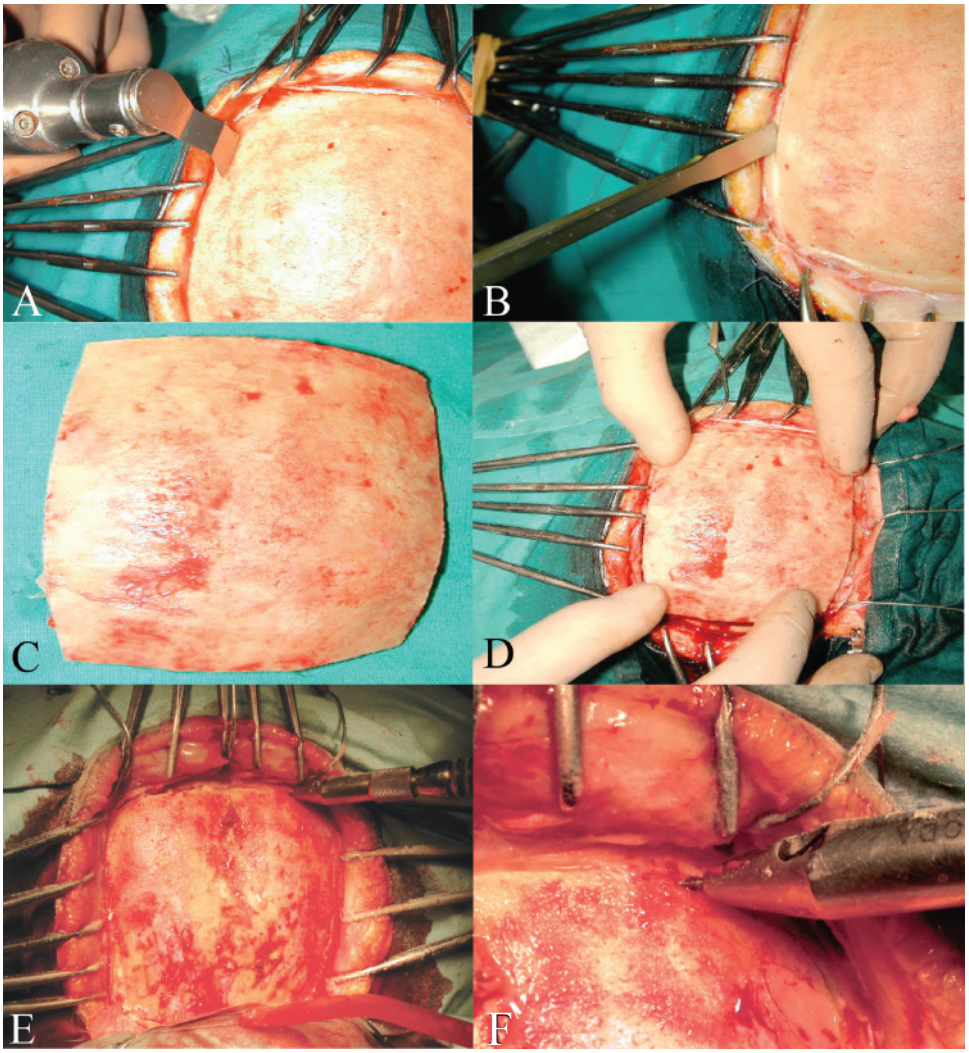


Fig. 16.5 **(A)** A craniotomy performed using an oscillating saw **(B)** A chisel is used to gently crack the inner layer of the bone. **(C)** Bone flap. **(D)** Reconstruction of the skull showing the absence of burr holes or irregularities/deformities. **(E,F)** Craniotomies performed using the Anspach Control Depth Attachment (Anspach Effort, Inc., Palm Beach Gardens, FL).

from the periphery to the midline and mobilized toward the deeper planes as dissection progresses[5] (**Fig. 16.6**). If the tumor has violated the pial surface, gentle dissection with selective bipolar coagulation is used to separate the capsule. Tenting sutures can be placed on the lateral edges of the dural flap. These sutures are tagged to either bulldog or Kocher clamps rotated medially toward the contralateral hemisphere. The weight of the clamps on the dural flap serves to distract the tumor, which is usually well anchored to the dura, from the surrounding parenchyma. This helps find a dissection plane between tumor and brain.

For tumors greater than 3 cm in diameter, which constitute a large portion of the surgical lesions, internal debulking with ultrasonic aspiration or monopolar cautery must be performed to facilitate dissection of the tumor capsule. Following internal debulking, dissection can proceed in a circular fashion, alternating from the lateral margin up to the anterior or posterior pole and down to the opposite pole until the deep aspect of the lesion is reached.

All vascular structures must be identified and followed to determine their contribution to the tumor vasculature before coagulation because pericallosal and callosomarginal branches are frequently parasitized by these tumors and supply the inferomedial aspect of the lesions. Small lateral feeding branches are coagulated and divided. Meticulous preservation of all bridging veins is crucial; when a vein is invaded by tumor not easily removed with gentle dissection, most authors recommend leaving residual disease rather than sacrificing the vessel. This is particularly true at the anterior and posterior margins of the tumor (anterior and posterior pole), in which hypertrophic veins reaching the sinus are frequently encountered. Injury to critical venous structures (i.e., rolandic veins) can occur during dissection, and the surgeon must be prepared for a venous reconstruction to avoid a devastating venous hemorrhagic stroke in eloquent cortex.[5] In cases that require SSS wall reconstruction, an end-to-end venous graft to the stump of the bridging vein can be performed. If the SSS is not involved, cutting the dura parallel to the SSS after all parasagittal veins have been released completes the resection.

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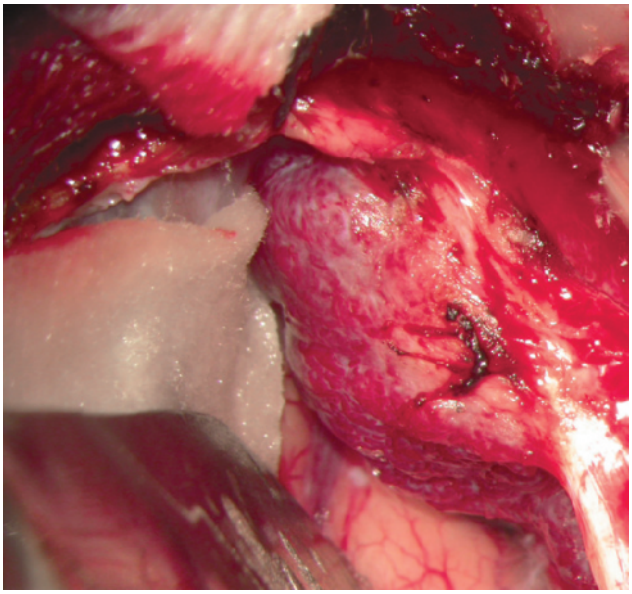


Fig. 16.6 A 33-year-old female with a recent history of transient, short episodes of right body paresthesias and concomitant aphasia. Magnetic resonance imaging showed a left parasagittal contrast-enhancing mass completely obstructing the superior sagittal sinus. An intraoperative photograph shows the exposure of the left aspect of the mass and its dissection from the brain.

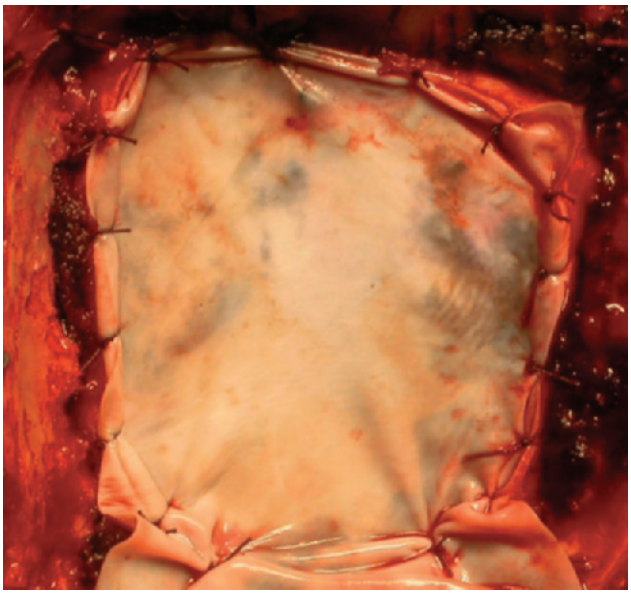


Fig. 16.7 Same patient as shown in **Fig. 16.6**. Intraoperative photograph showing dural reconstruction using the dural graft Tutopatch (Tutogen Medical, Gainesville, FL).

Dural Closure

Dural closure can be performed with either a pericranial graft harvested during the opening, fascia temporalis if accessible through the opening incision,[7] or a dural substitute such as allogenic human skin (AlloDerm, LifeCell Corp., Palo Alto, CA),[44] bovine pericardium (Dura-Guard, Synovis, St. Paul, MN; Tutopatch, Tutogen Medical, Gainesville, FL),[45] or bovine dermis (Durepair, Medtronic Sofamor Danek, Inc., Memphis, TN)[46] (**Fig. 16.7**). Watertight closure is important, especially in patients with residual disease or higher-grade lesions in whom adjuvant radiosurgical therapy will be pursued. If tumor-induced hyperostosis is significant, the bone flap should be replaced by a cranioplasty, which can be performed with methylmethacrylate, titanium mesh, or other reconstructive materials.[4]

Sinus Management

Conservative Resection with Residual Intrasinusal Tumor

With the evolution of high-resolution imaging and stereotactic radiosurgery, aggressive resection of intrasinusal components of parasagittal meningiomas is less frequently pursued.[5] For lesions that invade the SSS wall but do not compromise patency, some authors advocate for resection of the extrasinusal component, with periodic MRI/MRA follow-up of the residual intrasinusal tumor. If tumor growth is detected during follow-up, radiosurgery may be recommended before pursuing a second surgical resection. In tumors that completely obliterate the SSS, collateral anastomotic networks have usually developed over time and can prevent symptomatic venous hypertension.[16,20,47–49] In this situation, resection of the obliterated sinus is performed after detailed assessment of these venous collaterals, which are preserved at the time of surgery. The encased portion of the sinus is first ligated at both the proximal and the distal ends using heavy silk sutures and then cut using dural scissors. The falx is then cut from one end to the opposite along the edge of the tumor (**Fig. 16.8**).

Sinus Exploration

Exploration and reconstruction of the sagittal and parasagittal venous system have been described in the literature for the past three decades, and several different techniques for sinus repair have been reported with variable results.[24,50–55]

Primary Repair and Grafting

For lesions that invade only the lateral recess, a "marginal resection" of the sinus is performed, which consists of resecting the corner of the lateral sinus invaded by the tumor. This is followed by placement of 3–0 Vicryl sutures at 5 mm intervals along the length of the sinus resection. The ends of these sutures are preserved; the tails are left long and tagged with bulldog or Kocher clamps. At the end of the operation these sutures are used to secure a dural graft to the sinus to complete the dural closure.

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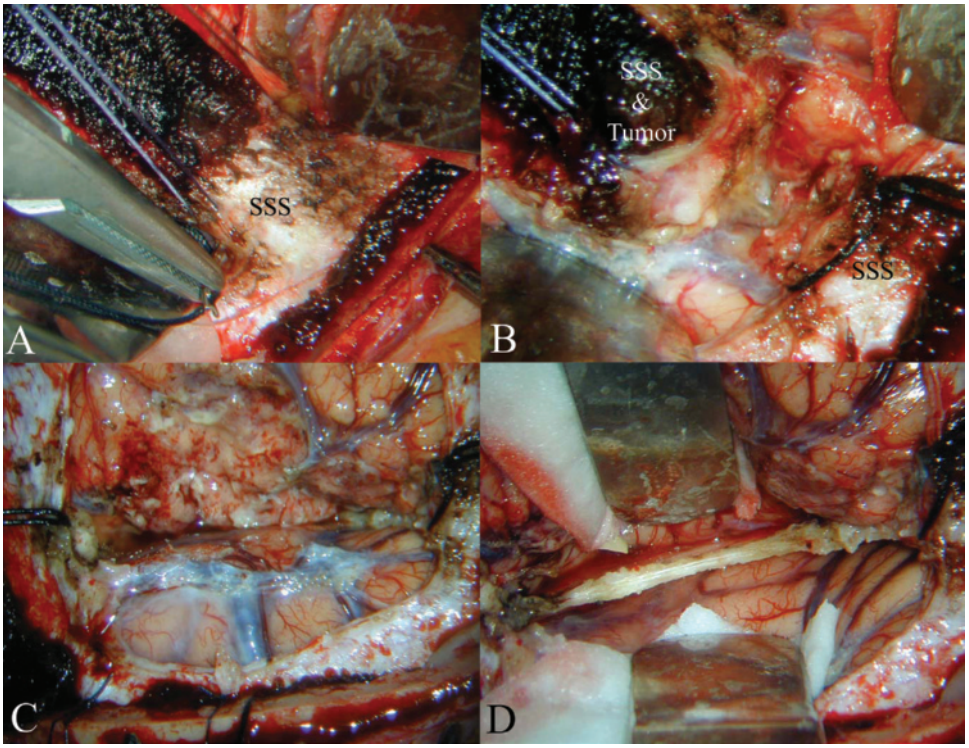


Fig. 16.8 A 63-year-old woman with a history of recent onset of intracranial hypertension and left-sided hyposthenia. Magnetic resonance imaging showed a right-sided parasagittal meningioma with complete occlusion of the superior sagittal sinus (SSS). **(A)** A silk suture is placed across the falx just distal to the proximal end of the tumor-encased portion of the SSS. **(B)** The sinus has been ligated and cut. **(C)** The sinus has been ligated and cut at the proximal and distal ends of its involvement within the tumor. **(D)** A self-retracting spatula helps to visualize the resected margin of the falx and the two ends of the resected sinus.

For lesions with partial SSS wall involvement in which GTR is pursued, reconstruction of one or two walls of the sinus can be performed with an autologous vein graft,[5] which is usually harvested from the saphenous venous complex. For small single sinus wall defects, grafting with a free fragment of saphenous vein under loose tension can be achieved if the remaining sinus walls are stable enough to maintain patency of the SSS. Alternatively, a flap from the falx may be rotated upward, and sutured anteriorly, posteriorly, and superiorly to reconstruct the lateral wall of the sinus.[9,56]

For lesions in which two sinus walls are compromised without complete occlusion of the SSS, grafting can be performed after the invaded sinus walls are resected with careful preservation of the remaining uninvolved sinus wall, thereby preserving rolandic venous inflow from the contralateral hemisphere.[24,57] The venous graft, usually harvested from the internal saphenous vein, must be procured and marked to maintain its original cephalocaudal orientation because the internal venous valves determine the direction of blood flow.[5] Interrupted, loose circumferential sutures with a nonabsorbable material are placed at regular intervals, and patency is verified before completing the closure of the graft by selective compression of the proximal and distal ends. If the opening of a cortical draining vein joins the opening of the uninvolved sinus wall, partial occlusion with light digital compression by the assistant is performed until grafting is completed. When cortical draining veins are absent, selective shunting during grafting can be performed as described by Hakuba.[23]

Bypass

Sindou and colleagues in Lyon, France, have extensively reported on their experience with aggressive resection of parasagittal meningiomas and have presented interesting results in terms of recurrence rates and perioperative morbidity following venous flow restoration surgery.[7,16,48,58] This group particularly questions the safety of aggressive resections of totally occluded portions of a sinus, and cautions on the potential complications that can occur related to congestive cerebral edema, venous infarction, and pseudomeningoceles, even in patients with vigorous collateral circulation. In their series, the morbidity related to venous injury or flow interruption in patients with a completely obliterated sinus who did not undergo a venous flow reconstruction procedure was estimated at 8% with a 3% mortality (from cerebral edema likely related to the surgical procedure).[3,7] Similarly, this group also argues that in a series of 21 cases reported by Bonnal and Brotchi, in which one patient (4.7%) died after an en bloc tumor resection, death could have been prevented by venous restoration.[24] Based on these data, flow restoration has been routinely pursed by this group.

In the senior author's initially reported experience of 108 patients, cerebral edema occurred in 8.9% of

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patients, one third of whom underwent an en bloc resection without venous reconstruction.[29] Since that report, this series now includes 52 en bloc resections of tumors encasing the sinus that have been performed without sinus restoration, without significant morbidity. Of these patients, three developed transient cerebral edema, which resolved with good long-term recovery; one patient developed a bone flap infection, and one patient presented with a cerebrospinal fluid (CSF) subgaleal collection that resolved with temporary CSF spinal drainage (unpublished data). No perioperative deaths occurred.

Common discrepancies regarding the amount of intrasinusal tumor are frequently seen between preoperative images and intraoperative findings. For this reason, some authors recommend routine exploration of the sinus in all cases of sinus-invading tumors through a short incision on the sinus wall.[3] Insertion of small pledgets of hemostatic material (Surgicel, Ethicon) within the SSS lumen and inside the ostia of adjoining veins helps control venous bleeding during the dissection[16,58–61] (**Fig. 16.9**). Intrasinusal septations, particularly along the middle third, can significantly obstruct the passage of inflatable balloons for selective shunting and can also injure sinusal endothelium. The crushing effect of vascular clamps and aneurysm clips on the sinus walls also precludes their use. Venous reconstruction is performed with either patches or bypasses.[23,51–54,56,62–65] Small nonabsorbable sutures (e.g., Prolene 8–0) are recommended to secure the graft or patch. The patch can be fashioned from autologous sources such as pericranium, fascia lata, or fascia temporalis. Autologous vein harvesting can be used for patching, but the procurement procedure would add unnecessary time and potential complications; therefore, it is only recommended for bypass grafting. Sinuses with complete venous occlusion may be suitable for bypass grafting. When longer segments of autologous venous graft are needed (over 6 cm), the internal saphenous vein is a suitable donor. If a shorter graft is required, it can be procured from the external jugular vein. Postoperative management of patients undergoing bypass procedures increases the clinical complexity because blood pressure parameters, volume status, and blood viscosity need to be carefully titrated to prevent thrombosis of the graft or the sinus. Anticoagulation is always required in these patients, beginning 24 hours after surgery, with heparin, for a total of 3 weeks, and concomitant transition to Coumadin (Bristol-Myers Squibb, New York, NY), which is continued for a period of 3 months until sinus reendothelization is achieved.[3,7] In Brotchi's series, 13 of the 15 sinus repairs (86.6%) performed with either dura or fascia had angiographically confirmed patency. In their experience, fascia temporalis was the most suitable material for sinus patching, and their experience with synthetic Gore-Tex (W. L. Gore and Associates, Inc., Newark, DE) grafts did not result in patent sinuses despite prompt and sustained anticoagulation therapy.

Algorithms for decision making on the need to bypass following an occluded sinus segment resection have not been formulated, and the benefit of measuring intrasinusal venous pressures intraoperatively with an occlusion test to formulate a decision is not supported by published data. Sindou and colleagues represent a single-center experience of a high-volume surgeon. This group strongly recommends advanced preparation and training before attempting any flow restoration procedures, and the results of these techniques in other hands have not been systematically analyzed.[7]

Potential Complications

Advances in neuroanesthesia, preoperative imaging, intraoperative image guidance, microsurgical techniques, neurophysiological monitoring, surgical devices, postoperative monitoring, and neurocritical care continue to decrease the incidence and severity of complications. Initially, the reported complications were considerable, as exemplified by the series of Hoessly and Olivecrona.[66] In this series of 196 parasagittal meningiomas, all of which were treated without sinus reconstruction, the morbidity rate was 12.3% (with half of the complications resulting from venous injuries), and the mortality was 10%, with 12.8% of these occurring in patients with tumors of the middle third, 9% in patients with posterior third tumors, and 5.3% in patients with tumors in the anterior third. Complications included one postoperative hematoma, severe blood loss in six cases, two carotid thromboses, one patient who developed cardiac insufficiency, two patients with pulmonary embolisms, three surgical site infections, and four patients with pneumonia.

The risk of air embolism varies between published series and is influenced by the patient position. In Alvernia and Sindou's series of 100 cases,[3] just one air embolism case was diagnosed (1%) despite the use of the semisitting position for most cases. The incidence of postoperative hematoma was 3%, and postoperative infections occurred in 6%, with three bone flap infections (two cerebral abscesses, and one an extradural empyema). In the senior author's series of 108 patients,[29] five patients (4.7%) developed wound infections, two developed a postoperative hematoma, one of which was fatal, while the second one had a poor outcome after evacuation. This series now comprises 184 patients, and morbidity was related to cerebral edema in 12 cases (6.5%) and postoperative hematoma in four patients (2.2%). Complications and outcome data from other published series to date were insufficient.[4,24,67]

Contemporary Surgical Outcomes Series

DiMeco and Colleagues[29]

This is a retrospective series of 108 patients with parasagittal meningiomas with SSS invasion who underwent

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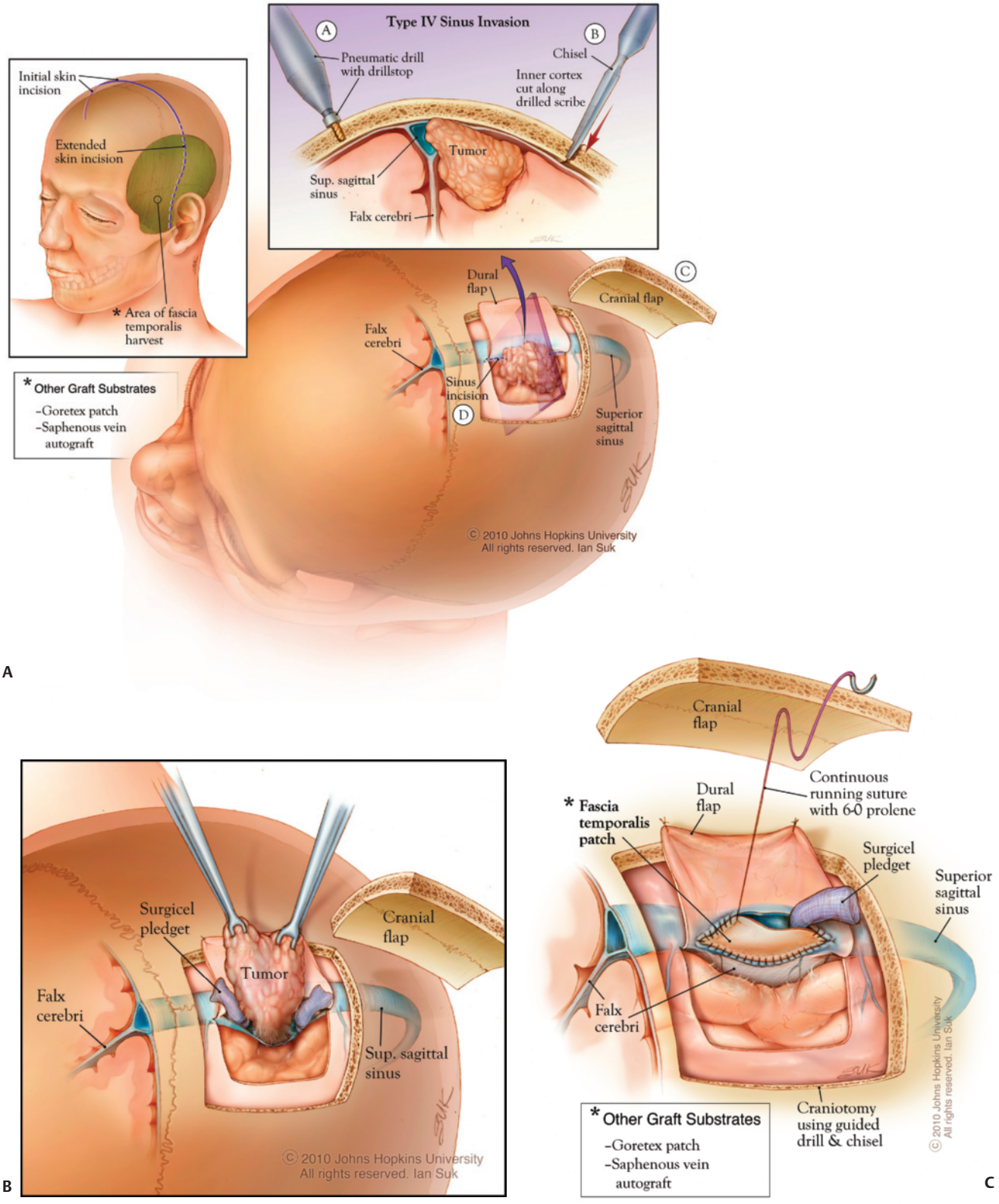


Fig. 16.9 **(A)** Artist's illustration showing the skin incision with harvesting of the fascia temporalis (top left), craniotomy for a left parasagittal meningioma using (A) the pneumatic drill with drill-stop and (B) chisel. (C) The elevated bone flap and (D) the dural opening with the dural flap based on the sinus wall, and the incision in the lateral sinus wall for removal of intrasinusal tumor. **(B)** Artist's illustration showing removal of the intrasinusal portion of a parasagittal meningioma of the middle third, with temporary placement of Surgicel pledgets (Ethicon, Inc., Somerville, NJ). **(C)** Artist's illustration of the technique for repair of the lateral superior sagittal sinus wall with a fascia temporalis graft following resection of the tumor from the intraluminal space.

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Table 16.5 Contemporary Surgical Outcome Series

Surgical Series of Parasagittal Meningiomas (1955 to 2010)					
Authors	**Year**	**No. of Cases**	**Recurrence Rate (%)**	**Median Follow-up (years)**	**Overall Mortality (%)**
Hoessly and Olivecrona[66]	1955	196	6	5	12.3
Simpson[67]	1957	107	19	5	–
Logue[95]	1975	91	11	–	4.4
Bonnal and Brotchi[24]	1978	21	14	–	4.7
Kropp et al[73]	1978	96	16.6	7	–
Yamashita et al[96]	1980	80	14.6	5	–
Chan and Thompson[97]	1984	16	13	–	–
Giombini et al[72]	1984	243	17.7	5	–
Mirimanoff et al[70]	1985	38	24	10	–
Jääskeläinen et al[98]	1986	136	8	–	–
Philippon et al[74]	1986	153	14.4	10	–
Baird and Gallagher[11]	1989	46	23.9	–	–
DiMeco et al[43]	2004	108	13.9	13	2
Sindou and Alvernia[7]	2006	100	4	8	3
Colli et al[10]	2006	53	32.7	7.8	1.9
Raza et al[68]	2010	61	11	4.4	1.5
DiMeco et al[8]	2010	184	12.5	–	1

–, not available
Modified from Sindou and Alvernia.[7]

marginal or GTR at the senior author's institution **(Table 16.5)**. Grade I resections were achieved in 37% of patients with partial sinus invasion (72%), grade II in 31%, and grade IV in four patients. In patients with complete sinus obliteration (n = 30), grade I removal was achieved in 23, grade II in three, and grade IV in four patients. Eight patients received postoperative radiation (four malignant meningiomas, one hemangiopericytoma, and three atypical meningiomas). Perioperative mortality was 1.85% (one secondary to a postoperative hematoma and one due to pulmonary embolism). Overall morbidity was 29%, and the most serious complications included cerebral edema (8.3%) and postoperative hematoma (1.9%). Recurrence was noted in 13.9%, median time to recurrence (MTR) was 156 months for men (MTR not reached for women). MTR for malignant meningiomas, hemangiopericytomas, and atypical meningiomas were 16, 15, and 97 months, respectively (not reached for benign meningiomas). Whereas recurrence free survival (RFS) rates for benign meningiomas were 98% at 5 years and 93% at 10 years, RFS rates for atypical meningiomas were 77% at 5 years and 46% at 10 years.

Sindou and Alvernia[7]

This is a retrospective series of 100 consecutive patients who underwent surgery for SSS meningiomas (n = 92), transverse sinus meningiomas (n = 5), and meningiomas at the confluence of sinuses (n = 3). The classification scheme proposed in the study was described earlier (**Table 16.2**). Type I lesions were treated with dissection of the tumor capsule from the sinus wall. Types II to VI lesions were treated with either a nonreconstructive (coagulation of the residual fragment or GTR), or a reconstructive approach (primary repair, patch, or bypass). GTR was achieved in 93%. Sinus reconstruction was attempted in 65% of the cases with wall and lumen invasion. Overall recurrence rate was 4%, with mean follow-up of 8 years (range 3 to 23 years). Mortal-

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ity rate was 3% (consisting of type VI meningiomas who developed cerebral edema after en bloc resection of the tumor-invaded sinus without venous restoration). The authors concluded that their low recurrence favored aggressive resections with removal of intrasinusal tumor. Even though patients without venous reconstruction had worse neurological outcomes than those patients undergoing flow restoration, the authors cautioned on the technical difficulty of these procedures and the additional morbidity of pursuing them without proper experience.

Colli and Colleagues[10]

In this series, 53 parasagittal meningioma patients were treated at a single center. Anterior third lesions (n = 9) and those occluding the SSS (n = 5) had GTR with removal of the infiltrated sinus; tumors abutting but not invading the SSS (n = 20) were resected and their dural attachments coagulated; tumors invading one sinus wall (n = 10) were partially excised with sinus wall reconstruction; and tumors invading more than one sinus wall and located in the posterior two thirds of the SSS (n = 7) had subtotal resections. Sinus resection or reconstruction was not attempted in the posterior two thirds of the SSS. Total resection was achieved in 85%, with 13% subtotal resections. Overall recurrence was 33% (18% for grade I tumors, 75% for grade II, and 100% for grade III). At 5 and 10 years, grade I had 81% and 68% RFS rates respectively, grade II had 71% and 18% RFS rates, and grade III had 0% RFS rates. Operative mortality (first 30 days) was 1.9%, surgery-related mortality was 5.7%, and overall mortality rate was 26.4%. On follow-up, eight unrelated deaths occurred, and three patients with grade III meningiomas died from disease progression. Neurological deterioration occurred in 7.8% (all with middle third tumors). Recurrence was more predominant in males, grade II/III tumors, subtotal resections, and lesions with greater invasion of the SSS.

Black and Colleagues[4]

This series included 46 consecutive patients who underwent parasagittal meningioma resections at the senior author's institution. All extrasinusal tumor and dura involved were resected, and residual intrasinusal tumors were followed radiographically. When tumor progression was detected, it was treated with radiosurgery. GTR was achieved in 63%, with no recurrence on follow-up MRIs. Residual tumor on postoperative MRI was detected in 14 patients (37%), and of these, 13% progressed with mean and median times to progression of 8 years. RFS was 95% at 5 years.[4] Of patients that presented with a preoperative neurological deficit, 56% had complete resolution after surgery, and 5.5% had a new deficit. One elderly patient died within 1 month of surgery of a pulmonary embolus. Multivariate analysis confirmed that histological type, tumor size, and Simpson grade were significant as independent prognostic factors for recurrence.

Raza and Colleagues[68]

This retrospective series reported 110 patients with parasagittal meningiomas who underwent surgery at Johns Hopkins Hospital. GTR was pursued for all lesions invading the sinus, without flow restoration. Residual or recurrent lesions were observed and managed with radiosurgery if progression was detected. Patients with a minimum of 24 months of follow-up (n = 61) were further analyzed. Mean follow-up was 41 months. All patients underwent surgical resection, and those exhibiting residual/recurrent disease were treated with radiosurgery if indicated (19.6%). Pathological examination showed 80% grade I meningiomas, 13% grade II, and 7% grade III. Simpson grade I/II resections were achieved in 81% of the patients. Major complications consisted of venous thrombosis/infarction (7%), intraoperative air embolism (1.5%), and death (1.5%). The recurrence rate was 11%, and the rate of improvement on Karnofsky Performance Status (KPS) was 85%.

Tumor Recurrence

Despite the benign and slow-growing nature of meningiomas, recurrence can affect all histopathological grade tumors, and prior studies have shown that parasagittal meningiomas tend to recur more frequently than meningiomas at other locations.[11,69,70] Recurrence appears to be multifactorial, with little consensus on the impact of specific factors, such as histological type, tumor size, and extent of resection. With regard to age and sex, some authors have found increased risk and shorter RFS in younger patients, whereas no difference in the recurrence rates between sexes has been detected regardless of pathological grade.[29,71]

Following Simpson's study in 1957 on meningioma recurrence,[9] the relationship between extent of resection and recurrence rates continues to be validated. In the original study, complete tumor resection with excision of the dural attachment was associated with a 6% recurrence rate, complete resection with coagulation of the dural attachment resulted in a 16% rate of recurrence, and GTR without treatment of the dural attachment resulted in a 29% recurrence rate. Although multiple series published since had shown recurrence rates between 14 and 24%, the latest surgical series have significantly decreased these numbers.

Sindou and Alvernia[7] reported GTR in 93% of patients. Of 69 patients with wall and lumen invasion, 65% underwent sinus reconstruction. The recurrence rate for patients with en bloc GTR was only 4% after an 8-year follow-up period. Permanent neurological worsening from venous infarction occurred in 8% of patients. In this se-

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ries the clinical outcomes of patients with tumors invading the sinus wall were more favorable when autologous venous graft reconstructions were performed. Using this approach, patients underwent higher complexity surgeries with higher perioperative risk because nearly one third of them underwent a two-stage procedure, and several required postoperative anticoagulation or antiplatelet therapy to maintain graft patencies and prevent sagittal sinus thrombosis.

In the senior author's series of 108 patients, Simpson grades I and II resections were achieved in 100 patients. The recurrence rate was 13.9%, with most recurrences seen in patients with higher tumor grades. Recurrence-free survival rates for patients with benign meningiomas were 98 and 93% at 5 and 10 years, respectively. Previous published series with documented 5- to 10-year follow-ups showed recurrence rates between 8 and 23.9%.[24,70,72–75] The recurrence rate for grade I meningiomas was 3.5% and 13.9% for all tumors.

In the series by Black and colleagues, a less aggressive surgical approach was used,[4] with extrasinusal tumor resection up to the sinus wall without sinus exploration, close follow-up of residual lesions, and radiosurgery treatment after detected recurrence. Of the patients treated with radiosurgery, one patient required a second operation due to persistent tumor growth. With this approach, although 36.8% of patients had postoperative residual tumors, less than one third showed progression on follow-up, with 7.7% of grade I lesions showing progression.

Adjuvant Therapies

Stereotactic Radiosurgery

Radiosurgical treatment of parasagittal meningiomas can be administered as fractionated stereotactic radiation delivered by a linear accelerator (LINAC)[76,77] without or with robotic assistance (Cyberknife, Accuray, Sunnyvale, CA),[78–80] intensity-modulated radiotherapy (IMRT),[79,81] tomotherapy,[82,83] or single-dose gamma therapy via gamma-knife radiosurgery.[84–87] Its use has been popularized to treat residual or recurrent disease.[88–91] Black and colleagues, using a conservative surgical approach, reported adjuvant radiation surgery for residual or recurrent disease in 6% of their surgical patients within 5 years of their first surgery.[4] Some authors have also proposed stereotactic radiosurgery as a first line of treatment in symptomatic patients with high preoperative risks (advanced age, multiple comorbidities, poor performance status). A study by Kondziolka et al[27] consisting of 203 patients with parasagittal meningiomas that were treated with stereotactic radiosurgery showed a tumor control rate of 93 ± 4% with radiosurgery as a first-line therapy, but a control rate of 60 ± 10% for patients who had undergone surgery before the radiation treatment. The rate of tumor control for the radiosurgery-treated volume was 85%, and they concluded that radiosurgery was an effective treatment for tumors less than 3 cm in diameter.

Parasagittal meningiomas treated with radiosurgery alone, however, have been reported to have a higher incidence of symptomatic post-treatment peritumoral edema,[92,93] which appears to be more prevalent with larger tumors and when higher prescription doses are given.[21,93] In a recent study by Girvigian and colleagues of 32 patients with convexity and parasagittal meningiomas, post-treatment edema appears to be higher with single-fraction regimens than with fractionated stereotactic radiotherapy, despite larger tumor volumes treated with the latter. This effect was decreased when single fraction therapy was applied to smaller tumors and with a dose < 14 Gy.[21]

Pathological grade can be used to guide the decision for radiotherapy. Consensus on radiotherapy for residual or recurrent disease in patients with grade III lesions is uniform across the neurosurgical and radiation-oncology literature. On the contrary, radiation for grade II lesions remains controversial. In these cases, the authors recommend closer postoperative follow-up with serial MRI at more frequent intervals, and when recurrence or progression of residual disease is detected, reoperation or radiosurgery will be recommended on a case-by-case basis.

Chemotherapy and Other Adjuvant Treatments

Treatment of recurrent or high-grade meningiomas remains a challenge.[94] A specific study of chemotherapeutic agents for treatment of recurrent or high-grade parasagittal meningiomas has not been performed, but patients with parasagittal meningiomas have been moderately represented in previous trials of recurrent meningiomas gathering multiple locations. Adjuvant chemotherapeutic agents are discussed in detail in another section of this book.

Conclusion

Since the first edition of this book, the treatment of parasagittal meningiomas has significantly evolved from aggressive GTR and flow restoration with bypass techniques to a multimodality technology-enhanced approach involving microsurgery, endovascular assistance, and radiosurgery. Although this evolution has decreased surgical morbidity and improved outcomes, much work remains to be done to assess the long-term effects of new adjuvant therapies, and more rigorously designed randomized studies should follow. As global life expectancies continue to increase, more patients with complex comorbidities will be diagnosed with these lesions, and more asymptomatic patients will be diagnosed at earlier stages before complete sinus occlusions.

The goals of treatment are now to achieve as complete a tumor resection as possible without affecting neuro-

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logical function and quality of life. Our ability to conduct periodic follow-up of these patients with high-definition imaging technologies has also changed the criteria for clinical observation, timing of surgery, and decision to treat residual or recurrent disease. Although venous flow restoration techniques are available, their safety and efficacy have not been validated in multicentric randomized studies, and they expose patients to significantly added morbidity when performed by inexperienced centers. Based on our and other's experience, resection of completely occluded sinuses should be considered a generally safe strategy, although judicious study of the collateral venous flow and careful planning of the approach to avoid interference of such collateralization are warranted. The continuously evolving field of stereotactic radiosurgery allows for treatment of residual or recurrent disease, and other adjuvant technologies will continue to contribute to the care of patients afflicted with these challenging lesions.

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Chapter 17

Falx Meningiomas

Paulo Henrique Pires de Aguiar, Adriana Tahara, Marcos Vinicius Calfatt Maldaun, and Celso Agner

Introduction

A primary falx meningioma, as defined by Cushing and Eisenhardt, is a meningioma arising from the falx cerebri that is completely concealed by the overlying cortex.[1] Falcine meningiomas tend to grow predominantly into one cerebral hemisphere but are often bilateral, and in some patients extend to the inferior edge of the sagittal sinus.

Falcine meningiomas and parasagittal meningiomas with falcine extension may arise at any point along the anterior to posterior midline and have similar clinical presentations. Although similar clinically, technical considerations differ between these tumor locations. Parasagittal meningiomas are discussed in Chapter 15.

Whether considering their clinical presentation or surgical treatment, it is useful and customary to categorize falx meningiomas based on their point of origin along the falx cerebri (**Fig. 17.1**). Thus, anterior falx meningiomas arise between the crista galli and the coronal suture, those of the middle third between the coronal and lambdoidal sutures, and those of the posterior third between the lambdoidal suture and the torcula.

Epidemiology

Falcine meningiomas account for 8.5% of intracranial meningiomas. The transitional variant is the most common histological subtype encountered.[2] The ratio of male to female incidence (1:2.1) is similar to that for meningiomas overall. The average age at presentation is 55 years.[2]

Clinical Presentation

Falcine meningiomas arising anterior to the coronal suture compromise relatively silent areas of the brain, and patients typically have an insidious onset of mental decline. These tumors may grow very large before being discovered (**Fig. 17.2A,B**). Symptoms of increased intracranial pressure, including headache and blurred vision secondary to papilledema, are not uncommon. Seizures, usually generalized but occasionally associated with speech arrest, although infrequent, may be seen.[3] Multiple cognitive impairments, including amnesia,[4,5] have been described in patients harboring falx meningiomas.

The middle third of the falx is the most common site of origin of falx meningiomas (**Fig. 17.3**). This region borders the supplementary motor area and the primary sensorimotor cortex for the foot and leg.[6] Focal motor or sensory seizures are often the first symptom of these tumors. Large tumors of the middle third can present with progressive hemiparesis as well as with mental decline.[7] In addition, with lesions on the dominant side, the seizures may be heralded by speech arrest, a syndrome that derives from compromise of the dominant supplementary motor area.

Finally, falcine meningiomas arising in the posterior third generally present with headaches and signs and symptoms of increased intracranial pressure (ICP). Homonymous hemianopsic visual field defects of varying degrees of completeness, with or without macular sparing, indicate compromise of the visual cortex. Visual hallucinations may be present. If located sufficiently far posteriorly, these tumors may involve the junction of the falx with the tentorium (**Fig. 17.4**) and may reach the tentorial incisura. Falcotentorial meningiomas are specifically addressed in Chapter 19.

Rarely, falcine meningiomas may present with apoplexy due to intraparenchymal hematomas, subdural hematomas, and subarachnoid hemorrhage.[8–10] Spontaneous hemorrhage in a previously asymptomatic falcine meningioma has been described after the use of low-dose aspirin over a prolonged period.[9]

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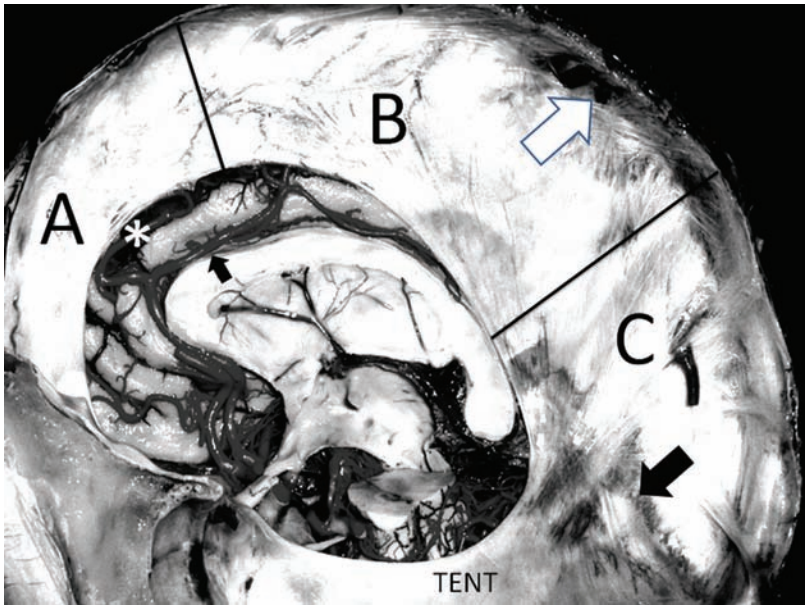


Fig. 17.1 Photograph of anatomical specimen identifying the anterior (A), middle (B), and posterior (C) portions of the falx. Visible are the superior sagittal sinus (*white arrow*) and the straight sinus (*black arrow*). The tentorium (*TENT*), pericallosal artery (*small arrow*), and callosomarginal artery (*asterisk*) are also seen.

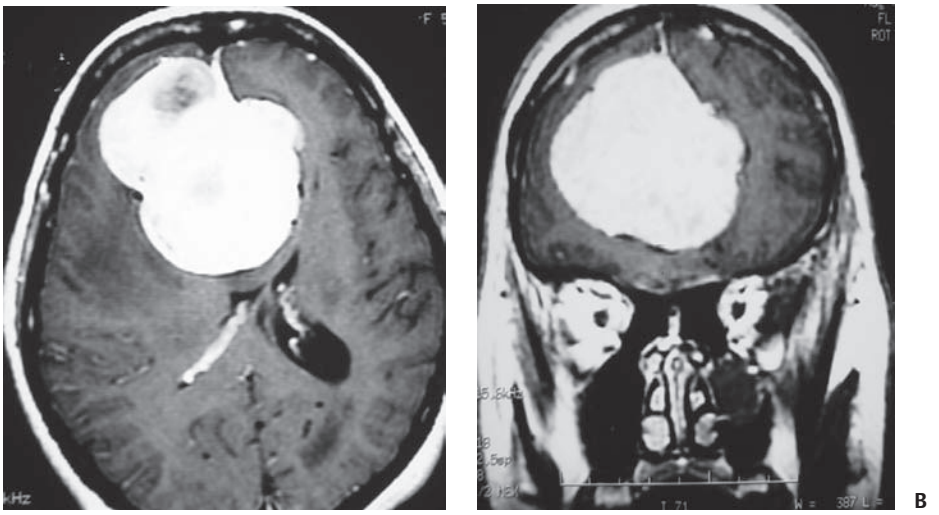


Fig. 17.2 **(A)** Axial postcontrast T1-weighted magnetic resonance imaging (MRI). Huge anterior third falx meningioma. The vascular displacement of the pericallosal and callosomarginal arteries, identified as flow-voids at the edge of the tumor, are well seen. **(B)** Coronal post-contrast T1-weighted MRI. Note that the tumor is completely hidden by overlying cortex.

Differential Diagnosis

There are several tumors that may mimic a falcine meningioma. Osteochondromas, chondrosarcomas, solitary fibrous tumor of the meninges, epidermoid tumors, and metastasis are the most frequently reported.[11–14]

Current Treatment

General Comments

Perioperative planning and operative removal of falcine meningiomas have been significantly enhanced by developments in neurodiagnostic methods, neurosurgical techniques, and the understanding of multiple neuroanatomical nuances in the arterial supply of these tumors. Paramagnetic enhanced magnetic resonance imaging (MRI),[15] superselective diagnostic and therapeutic cerebral angiography (digital subtraction angiography [DSA]),[16] ultrasonography and neuronavigation for intraoperative localization, ultrasonic surgical aspiration, new hemostatic agents, and surgical laser are some of the technological advances of the last few decades that further improved our ability to totally resect falcine meningiomas with minimal morbidity. Continuous monitoring of neural function in patients under general anesthesia using somatosensory evoked potentials has augmented our ability to assess the health of "at risk" brain adjacent to the tumor. Lastly, radiosurgery has become an option for primary or adjuvant treatment, especially in elderly patients, patients with severe comorbidities, instances of residual and recurrent atypical meningiomas, and patients who refused surgery.

Preoperative Diagnosis and Management

MRI with and without gadolinium helps to delineate the tumor's relationship with the venous sinuses, the tumor

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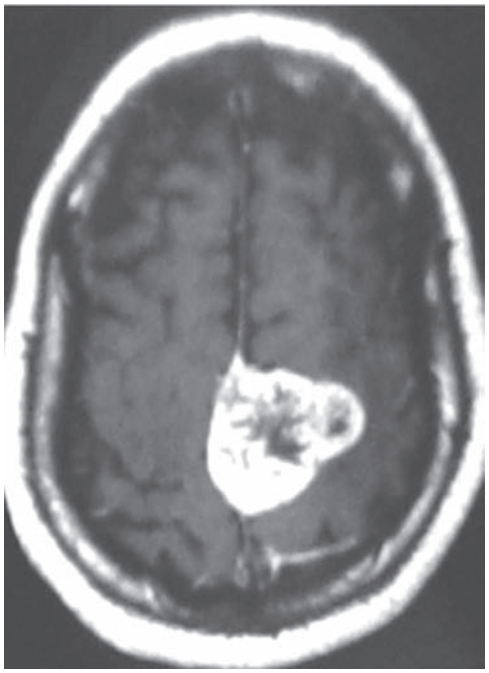


Fig. 17.3 Axial postcontrast T1-weighted magnetic resonance imaging. This partially calcified middle third falx meningioma is markedly invaginated into the paracentral lobule on the left.

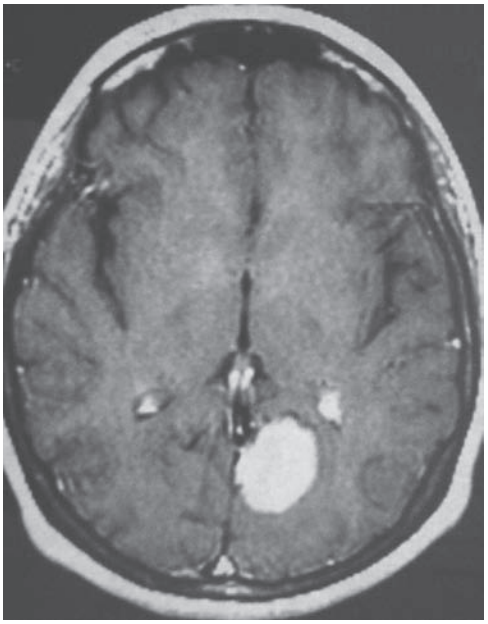


Fig. 17.4 Axial postcontrast T1-weighted magnetic resonance imaging. Small posterior third falx meningioma just above the junction with the tentorium.

interface with the cerebral cortex, the presence of significant blood supply, and the presence of atypical imaging features or cerebral edema, which might predict increased tumor aggressivity or an increased incidence of neurological deficits postoperatively. These imaging data can be integrated into neuronavigation protocols to be utilized in the operating room. Gadolinium-enhanced MRI allows demonstration of tumoral or adjacent dural enhancement. The radiological appearance affords a valid predictor of the degree of dural involvement in the region of the sinus and adjacent falx.

◆ Operative Procedure

Gross total resection of tumor is the single most important predictor of an improved surgical outcome.[2]

Surgery of a falx meningioma is composed of four essential consecutive steps; devascularization, detachment, debulking, and dissection.[17] In large tumors, attempts at en bloc resection may lead to cerebral damage, and debulking will be necessary to decrease the tumor volume and allow access to the falx for devascularization.

Positioning the Patient

The key to positioning the patient who has a falcine meningioma is to place the tumor uppermost in the operative exposure, with the midline of the skull positioned in the true vertical plane. For those tumors in the anterior and middle falx, we prefer to position the patient in the slouch or semisitting position. This position requires the use of a central venous catheter and Doppler monitoring so that any air embolus can be recognized and effectively managed. Some authors prefer the lateral position, usually with the tumor side down, for meningiomas of the middle third. For those tumors located in the posterior portion, we prefer the prone position, whereas others prefer the three-quarter prone or park bench position.

Craniotomy

The skin incision for those tumors of the anterior third is a classic coronal incision, and for those tumors further posteriorly, a horseshoe incision is appropriate. (**Fig. 17.5**). It is always important to be certain that the skin incision is carried well across the midline so that the superior sagittal sinus may be exposed over the length of the bone flap, not only on the side of the tumor but also 2 to 3 cm to the opposite side, to provide access to the contralateral dura as well as the entire superior sagittal sinus. The bone flap must be designed to afford access not only to the tumor but also to adjacent cortical surfaces to provide sufficient exposure for an elective cortical resection should it be necessary (**Fig. 17.6**). For tumors in those areas anterior to the sensorimotor region, therefore, the bone flap should extend somewhat further anteriorly. Conversely, for those tumors posterior to the sensorimotor area, the bone flap should expose an adequate cortical surface posterior to the tumor.

Once the bone flap has been elevated and any bleeding from the epidural space and superior sagittal sinus

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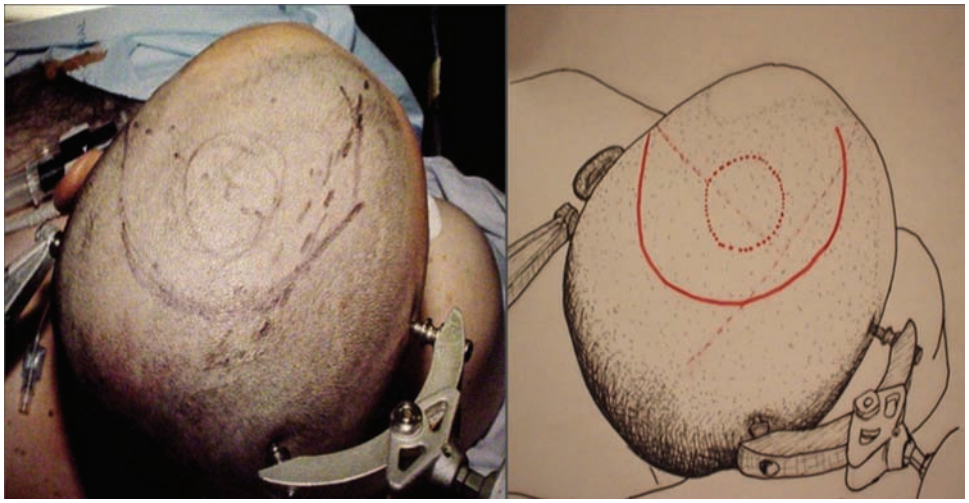


Fig. 17.5 Intraoperative photograph and accompanying line drawing depict a horseshoe-shaped scalp flap being used to expose a middle third falx meningioma. Note the extension across the midline.

is controlled, dural tack-up sutures are placed. Intraoperative ultrasonography is used to identify the anterior, posterior, and lateral margins of the tumor below the cortical surface. Frameless stereotactic navigation may be used for the same purpose but lacks real-time feedback. The dura is opened, beginning from the anterolateral corner and then extending from lateral to medial, the initial exposure being restricted to the exposure of only a small amount of adjacent brain tissue in all directions. The dura should be opened up to the margin of the superior sagittal sinus and retracted medially over the sinus with stay sutures. When the tumor is large or located near the superior sagittal sinus, the interhemispheric fissure may be splayed and the tumor immediately visible. It is helpful, regardless, to use ultrasonography once again to be certain that adequate exposure of the tumor has been achieved.

Removal of Meningioma

During initial dissection, it is important to use the surgical microscope to gain an appreciation of the depth and presence or absence of an arachnoid plane between the meningioma and adjacent brain tissue and its attachment to the falx (**Fig. 17.7A**). Tumor removal begins with the coagulation and sectioning of the tumor's attachment to the falx. Gentle sharp dissection of the arachnoidal plane identifies the tumor's pseudocapsule (**Fig. 17.7B**). Careful attention must be given to the bridging veins, especially the vein of Trollard in middle third falx meningiomas. Note that most of the retraction is against the falx to minimize retraction of already compromised brain. The surface of the tumor capsule is coagulated before its incision, and intratumoral debulking with the use of the ultrasonic aspirator is begun (**Fig. 17.7C**). Care is taken

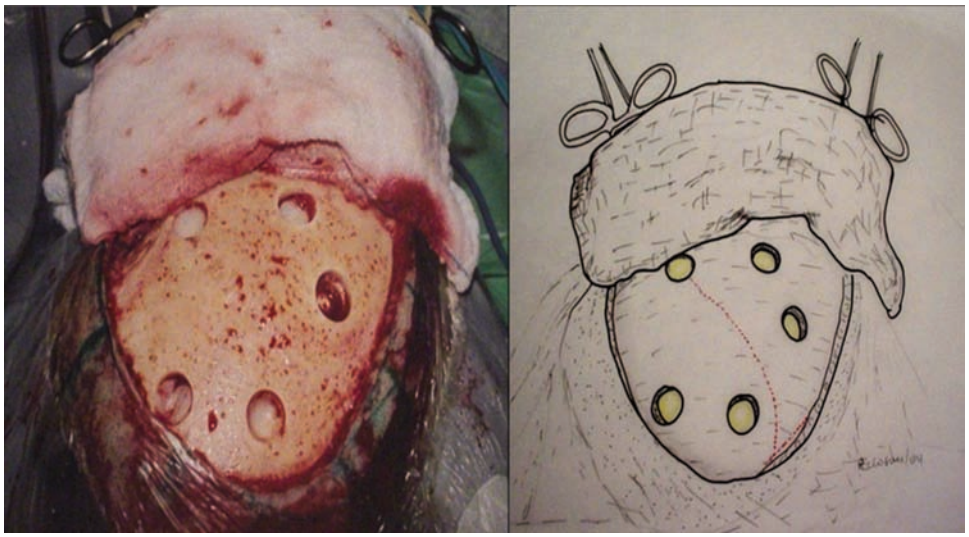


Fig. 17.6 Intraoperative photograph and accompanying line drawing show the planned craniotomy. Care is taken elevating the bone flap off of the superior sagittal sinus.

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not to violate the capsule. The arachnoid plane is gently developed with bipolar forceps and microdissection, and cottonoid pledgets are inserted into the interface between tumor and brain tissue. The deepest midline aspect of the tumor is frequently supplied by branches of the ipsilateral pericallosal artery (**Fig. 17.7D**). Extra care should be exercised during tumor resection in this area. Tumor extending to the contralateral medial hemisphere is removed though the falx.

When the tumor has been significantly reduced in bulk, the surgeon approaches the midline and identifies the medial aspect of the tumor in its relation to the normal falx anterior and posterior to the tumor. If the tumor's attachment to the falx is fairly broad at the margin of the superior sagittal sinus, it is often wise to consider amputating the tumor at a point ~1 cm from the falx and then to confront resection of the tumor's midline attachment to the superior sagittal sinus after the bulk of the tumor has been removed. This maneuver prevents significant bleeding from the superior sagittal sinus while a major portion of the tumor still remains within the cerebral substance.

The infiltrated falx may be coagulated and the falx, and the sinus if occluded, can be removed en bloc and the proximal and distal limbs of the sinus closed with Prolene 2–0 suture (Ethicon, Inc., Somerville, NJ). If the sinus is partially infiltrated, we remove the falx just below the sinus, avoiding resection of tumor in the sinus wall. The falx should be resected beyond the site of tumor attachment. The inferior sagittal sinus can be safely divided provided that the superior sagittal sinus is patent. In young patients, with tumors of the anterior and middle third of the sinus we may remove the infiltrated falx and infiltrated wall of the sinus, with reconstruction of the sinus wall with a dural flap, bovine pericardium, pericranium, or synthetic dural replacement. Vascular clips are applied to isolate the compromised wall, and the suture is done with Prolene (Ethicon) as quickly and carefully as possible to avoid venous thrombosis.

In falcine meningiomas localized to the posterior third of the skull, the vein of Galen and the straight sinus should be preserved at all costs. Tributaries of the internal cerebral veins and the basal vein of Rosenthal are responsible for drainage of the midbrain structures. Inadvertent compromise of these structures may lead to severe compromise of neurological function and death. Subtotal resection should be achieved to preserve neurological function.

Following a watertight dural closure, the bone flap is replaced and held firmly in position with miniplates. In cases where bony resection is needed, either autologous bone grafts or premanufactured prostheses could be utilized.

In the treatment of tumors anterior to the coronal suture, the surgeon can be relatively aggressive with excision of the lateral walls of the superior sagittal sinus and tumor-involved falx because the whole dural venous sinus can be ligated in this area, with minimal neurological complications, even if it is patent. If the sinus is occluded and the falx is infiltrated, then the involved sinus and subjacent infiltrated falx are excised in one piece. In treating lesions posterior to the coronal suture, the patent superior sagittal sinus cannot be removed safely, and excision of lateral wall infiltrated with falcine parasagittal meningioma is difficult and complex. In the past, the

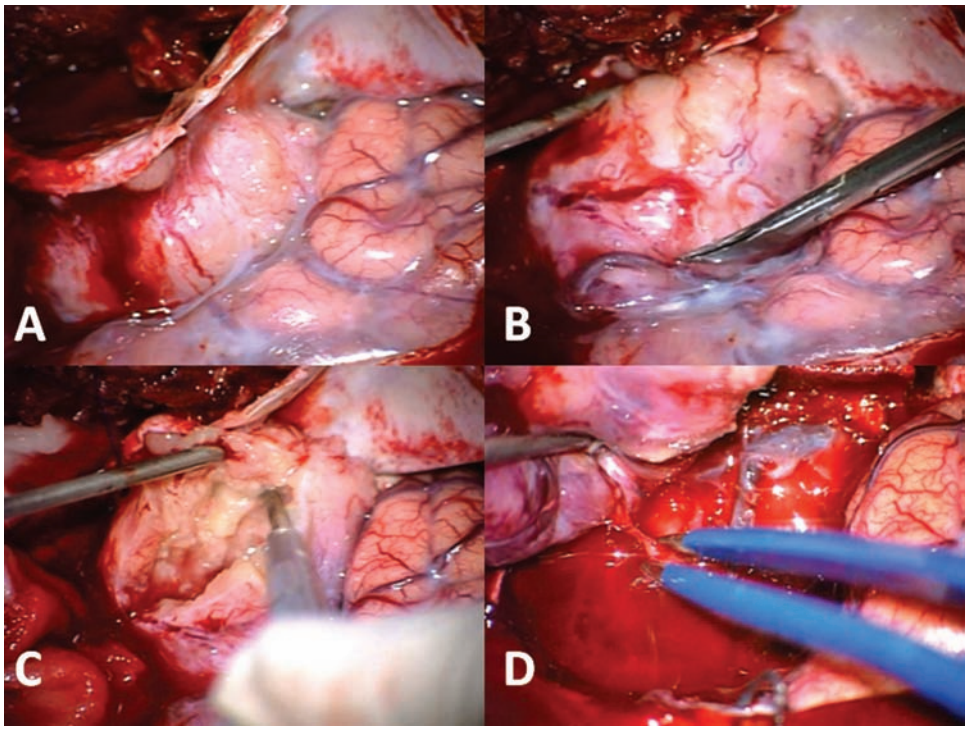


Fig. 17.7 **(A)** Initial exposure of a falx meningioma in the interhemispheric fissure. **(B)** Sharp arachnoidal dissection exposes the tumor pseudocapsule. The cortical veins are carefully preserved. **(C)** Intratumoral debulking is performed with the ultrasonic aspirator. **(D)** Careful dissection identifies vessels feeding the tumor allowing their coagulation and division.

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lateral sinus wall was sutured successively as the tumor was excised.

According to Barajas et al, large falcine meningiomas may be successfully removed using a contralateral interhemispheric approach. This provides an excellent way of directly dealing with large, deep interhemispheric feeding vessels unsuitable for embolization.[18–23]

For tumors placed posteriorly and in the inferior portion of the falx, the occipital interhemispheric approach should be tailored to the dural origin and extent of the tumor as depicted from preoperative MRI. Preservation of the straight sinus and galenic venous system is always recommended.[24] In those cases, additional resection of the falx and/or incision of the tentorium may be performed to allow complete resection (Simpson grade 1 and 2) in almost 85% of patients.[19–24]

Trigeminocardiac Reflex

Mechanical stimulation of the falx may induce a trigeminocardiac reflex (TGR), which could lead to bradycardia and even cardiac asystole.[20–25] Attentive monitoring of the patient's blood pressure and heart rate and clear communication with the surgeon are necessary.

Surgical Outcome and Prognosis

The rate of recurrence of falx meningiomas significantly increases in cases of subtotal resection of tumor. Aggressive surgical treatment, although of increased risk at times, significantly reduces the risk of recurrence.[26]

Cushing and Eisenhardt reported a 57% mortality in their series of seven patients.[1]

Chung et al[2] identified 22 men and 46 women who had surgical removal of a falx meningioma between 1990 and 2004 by members of the Department of Neurosurgery, Seoul National University College of Medicine. There were 22 meningiomas that arose from the anterior third of the falx, 20 from the middle third, and 15 from the posterior third. Total removal (Simpson grade I or II) was achieved in 58 of the 68 patients (85%). Six of the 10 patients with subtotal resection had their residual tumor treated with radiosurgery and had no evidence of recurrences. Reasons for subtotal removal were typically related to the need for venous preservation. Two patients with a total removal and four patients with a subtotal resection had evidence of recurrence/progression. All underwent reoperation. A good outcome was seen in 59 of 68 patients. Six patients had temporary neurological deficits and two had new deficits (one contralateral leg weakness, one with severe visual loss). One patient (1.4%) who was herniating on arrival had emergent surgery but unfortunately died.

Nowak and Marchel studied a series of 87 consecutive patients surgically treated for parasagittal and falcine meningiomas[26] and concluded that there were no tumor recurrences following radical resection of the tumor and invaded part of the sinus, but two postoperative deaths due to hemodynamic complications were observed. In the other 12 patients, meningiomas were removed but sinus infiltration was left in place; the postoperative period was uneventful but the rate of clinically important regrowth in this group of patients was 25% in long-term follow-up.[21–25]

Close to 13% of meningiomas are huge at the time of diagnosis. Extent of resection, recurrence rate, postoperative outcome, operative morbidity and mortality rates, and survival time are all negatively affected.[22]

Radiosurgery is an effective treatment option, mainly for malignant meningiomas or recurrent meningiomas.

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Chapter 18

Tentorial Meningiomas

Hischam Bassiouni and Siamak Asgari

Meningiomas of the posterior cranial fossa account for ~9% of all intracranial meningiomas.[1] Approximately 3 to 6% of all intracranial meningiomas and ~30% of posterior fossa meningiomas originate from the tentorium cerebelli.[2] The first account of a tentorial meningioma was given by Andral in 1833 as an incidental finding.[3] Surgical morbidity and mortality after resection of tentorial meningiomas have steadily declined in recent years due to advances in neuroimaging, refinements in microsurgical techniques, and improved intra- and postoperative neuroanesthetic care of the patient. Despite these continuing advances, surgical treatment of tentorial meningiomas still presents a major challenge, particularly in tumors that have intimately involved critical neurovascular structures.

Surgical Anatomy

The tentorium cerebelli is a dural duplication that covers the upper surface of the cerebellum and supports the occipital lobes, thus separating the supra- from the infratentorial intracranial compartment. It is attached posteriorly to the transverse ridges on the inner surfaces of the occipital bone and encloses the transverse sinuses at these sites. Anterolaterally it is attached to the superior ridge of the petrous portion of the temporal bone, enclosing the superior petrous sinus on both sides. It continues anteriorly to be attached to the posterior and anterior clinoid processes. Its medial free concave border bounds an oval opening (incisura tentorii) for transmission of the midbrain. The falx cerebelli is a small triangular dural process that arises in the midline from underneath the tentorium. It indents between the two cerebellar hemispheres. The upper surface of the tentorium connects to the posterior end of the falx cerebri in the midline, enclosing the straight sinus along the line of junction. The straight sinus receives the vein of Galen and the inferior sagittal sinus at the tentorial apex and runs posteriorly to meet the transverse sinuses from both sides and the superior sagittal sinus from above at the torcular Herophili. The tentorial incisura is divided into an anterior incisural space located in front of the brain stem, a middle incisural space situated lateral to the brain stem, and a posterior incisural space located behind the brain stem.[4] Anatomical structures located in relation to the tentorial incisura may be compromised by tentorial meningiomas and may be at risk during surgery. Preservation of these structures is usually facilitated at first surgery by arachnoid dissection layers forming the lateral walls of the chiasmatic, crural, ambient, and interpeduncular cisterns and is important to obtain a good surgical result. At the anterior incisural space, the most important neurovascular structures in close proximity include the oculomotor nerve, the basal vein, the posterior communicating artery, the anterior choroidal artery, the P1 and proximal P2 segments of the posterior cerebral artery, and the superior cerebellar artery. Less often, the optic nerve and the optic chiasm may be involved. The middle incisural space is bounded medially by the cerebral peduncle and upper pons. This narrow space contains the crural cistern, located between the uncus and cerebral peduncle anteriorly, and the ambient cistern situated between the midbrain and parahippocampal gyrus posteriorly. The trochlear nerve, the anterior choroidal artery, the P2 segment of the posterior cerebral artery, the superior cerebellar artery, and the basilar vein constitute the neurovascular contents of the middle incisural space. Particular attention should be paid to preserve the tiny trochlear nerve located just beneath the tentorium because it may be damaged in the subtemporal or petrosal approach. The ambient cistern continues posteriorly into the quadrigeminal cistern, which is the main cistern of the posterior incisural space. This space forms the pineal

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region and is related to anterior falcotentorial meningiomas. It contains the trunks and branches of the posterior cerebral and superior cerebellar arteries, and the vein of Galen, which receives the paired internal cerebral and basal veins. The trochlear nerve exits from below the inferior colliculi, curves around the dorsal midbrain, and enters the ambient cistern in the middle incisural space.

The tentorium receives its arterial supply from the basal tentorial artery (artery of Bernasconi-Cassinari) originating from the meningohypophyseal trunk, the marginal tentorial artery arising from the inferolateral trunk of the intracavernous carotid, and tentorial branches originating from the superior cerebellar and posterior cerebral arteries.[4] A peculiarity of the tentorium is the presence of interdural venous sinuses, which may become particularly prominent when major venous channels are occluded by tumor. Brisk bleeding from these venous lakes may be anticipated during incision of the tentorium in the occipital transtentorial approach. This bleeding can be controlled by bipolar coagulation. In the authors' experience, no neurological sequelae have been caused by their occlusion. The surgeon should study the venous drainage pattern of the temporal lobe and the point of termination of the subtemporal veins into the venous sinuses when planning for a subtemporal or petrosal approach.[5]

Classification

Classification of tentorial meningiomas, as any classification for meningiomas, has inherent limitations due to their inconsistent pattern and extent of dural attachment, direction of growth, and potential to transgress or obviously invade different anatomical structures (e.g., dura, bone, brain, vascular channels, and cranial nerves). This concern was already expressed by Cushing and Eisenhardt in their discussion of meningiomas of the cerebellar chamber.[6] The surgical approach should be tailored for each individual patient with these limitations in mind. Nonetheless, classification of these tumors is useful to systematize the most appropriate surgical approaches and to discuss problems and potential complications inherent to each tumor subgroup. Tentorial meningiomas may be confined to the infra- or supratentorial space or may extend both infra- and supratentorially (**Fig. 18.1**). Several classification schemes have been proposed; however, the one introduced by Yaşargil[7] is the most accurate in regard to surgical anatomy: (1) meningiomas arising from the free tentorial notch (i.e., inner ring meningiomas, anterior T1, middle T2, and posterior T3), (2) meningiomas originating from the intermediate tentorial surface (T4), (3) meningiomas involving the torcular Herophili (T5), (4) meningiomas arising from the lateral outer tentorial ring (posterior T6, anterior T7), and (5) falcotentorial meningiomas (T8). In our retrospective study we have modified this classification because often we could not accurately differentiate between T1 and T2 and T6 and T7 tumors from preoperative neuroimaging.[8] Furthermore, we have considered T3 and T8 tumors as one group of falcotentorial meningiomas. For practical purposes we therefore prefer a modified Yaşargil classification scheme comprising the following five tumor subgroups (**Fig. 18.2**):

1. **T1–T2** (medial "incisural" meningioma)
2. **T4** (paramedian "intermediate" meningioma)
3. **T5** (peritorcular "torcular" meningioma)
4. **T6–T7** (lateral tentorial meningioma)
5. **T3–T8** (falcotentorial meningioma)

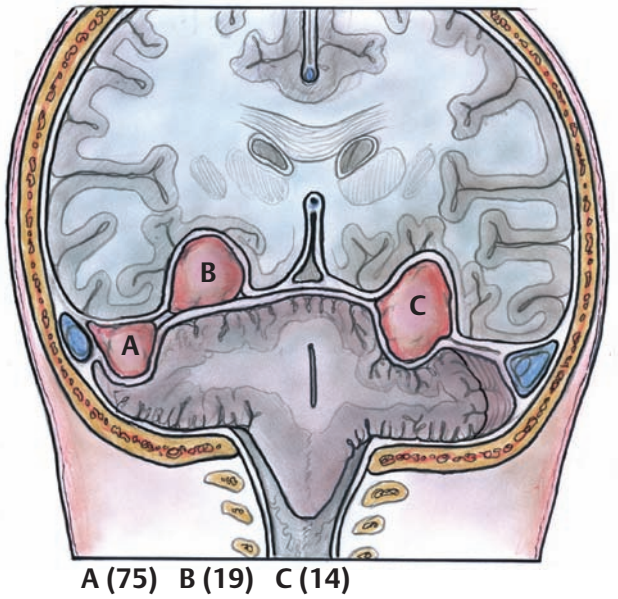


Fig. 18.1 Coronal sketch showing infratentorial **(A)**, supratentorial **(B)**, and suprainfratentorial **(C)** extension of tentorial meningiomas. Number of patients operated on in parentheses.

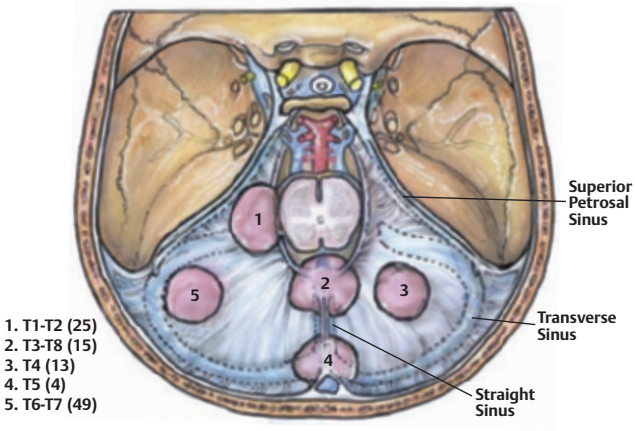


Fig. 18.2 Scheme showing location and subtypes in 106 tentorial meningiomas. Number of patients operated on in parentheses. Two falx cerebelli meningiomas treated surgically not shown.

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Table 18.1 Symptoms and Neurological Signs in 108 Patients Treated for Tentorial Meningioma

Symptoms	No. of Patients	% Patients
Headache	76	70.4
Dizziness	50	46.3
Gait disturbance	46	42.6
Mental changes	13	12.0
Visual disturbance	13	12.0
Hearing impairment	12	11.1
Seizures	11	10.2
Hemiparesis	8	7.4
Trigeminal neuralgia	7	6.5
Hemihypesthesia	5	4.6
Tinnitus	4	3.7
Dysphagia	2	1.9
Neurological Signs	**No. of Patients**	**% Patients**
Gait ataxia	54	50.0
Cranial nerve deficits	30	27.8
2nd nerve	5	4.6
3rd nerve	4	3.7
5th nerve	6	5.6
8th nerve*	14	13.0
9th nerve	2	1.9
Mental deficits	14	13.0
Hemiparesis	8	7.4
Homonymous hemianopsia	6	5.6
Hemihypesthesia	5	4.6
Aphasia	3	2.8
Symptoms—no deficits	15	13.9
Incidental finding	4	3.7
Arterial hypertension†	2	1.9

* Complete recovery in six patients and partial recovery in five patients after surgery.
† Significant improvement after surgery in both patients with infratentorial T6–T7 meningiomas and brain stem compression.

Clinical Presentation

Clinical symptoms and signs depend on the location and size of the tumor (**Table 18.1**). Tumor-related complaints are often present for long periods, usually months or even years, although rarely patients can present with acute symptoms of increased intracranial pressure caused by obstructive hydrocephalus. Patients harboring a T6–T7 infratentorial meningioma, the most often encountered subtype, commonly present with headache, dizziness, and gait unsteadiness[8,9] (**Fig. 18.3 A–E**). Clinical examination usually reveals a gait ataxia and occasionally impairment of the vestibulocochlear nerve. Hearing loss may be caused by direct involvement of the eighth cranial nerve (CN), or it may be the result of distortion of the central auditory pathways, such as the lateral lemniscus or the inferior colliculi.[10,11] Marked improvement of hypacusis or even restoration of normal hearing after tumor removal has been observed by several investigators.[8,10] It may be difficult to differentiate between infratentorial T6–T7 and posterior petrosal meningiomas on clinical and radiological grounds; indeed, lateral tentorial meningiomas may intraoperatively prove to have an additional origin from the suprameatal posterior petrosal surface.[8,12] Supratentorial meningiomas, particularly those closely related to the medial temporal lobe, may present with seizures. T1–T2 meningiomas may intimately involve the brain stem and the fifth CN. Accordingly, patients may present with hemiparesis, trigeminal neuralgia, and facial numbness.[13–15] Patients with T3–T8 meningiomas often present with headache (**Fig. 18.4 A–D**). Mental changes are reported in up to 46% of patients and a gait ataxia in 43 to 62% of cases. A homonymous hemianopsia is present in 20 to 46% of these patients.[16–18]

Diagnostic Workup and Preoperative Considerations

A small tentorial meningioma may be discovered incidentally on magnetic resonance imaging (MRI) in an elderly patient who is asymptomatic or presents with minor unspecific symptoms, such as headache and dizziness. In these cases a wait-and-see policy may be adopted and follow-up clinical and radiological examinations scheduled. The tumor may remain quiescent for many years, and the initial unspecific symptoms may resolve spontaneously. If follow-up examinations demonstrate progressive tumor growth or a deterioration of symptoms, surgery may be indicated. Radiosurgery may alternatively be considered in selected patients who are not good surgical candidates due to advanced age or significant comorbidity. Triplanar contrast-enhanced T1-weighted MRI gives the most accurate information for planning the surgical approach. The dural attachment zone and extent of the tumor, displacement of the brain stem, displacement or engulfment of vertebrobasilar arteries, invasion of the cavernous sinus, and patency of the straight, transverse, and sigmoid sinuses can be suf-

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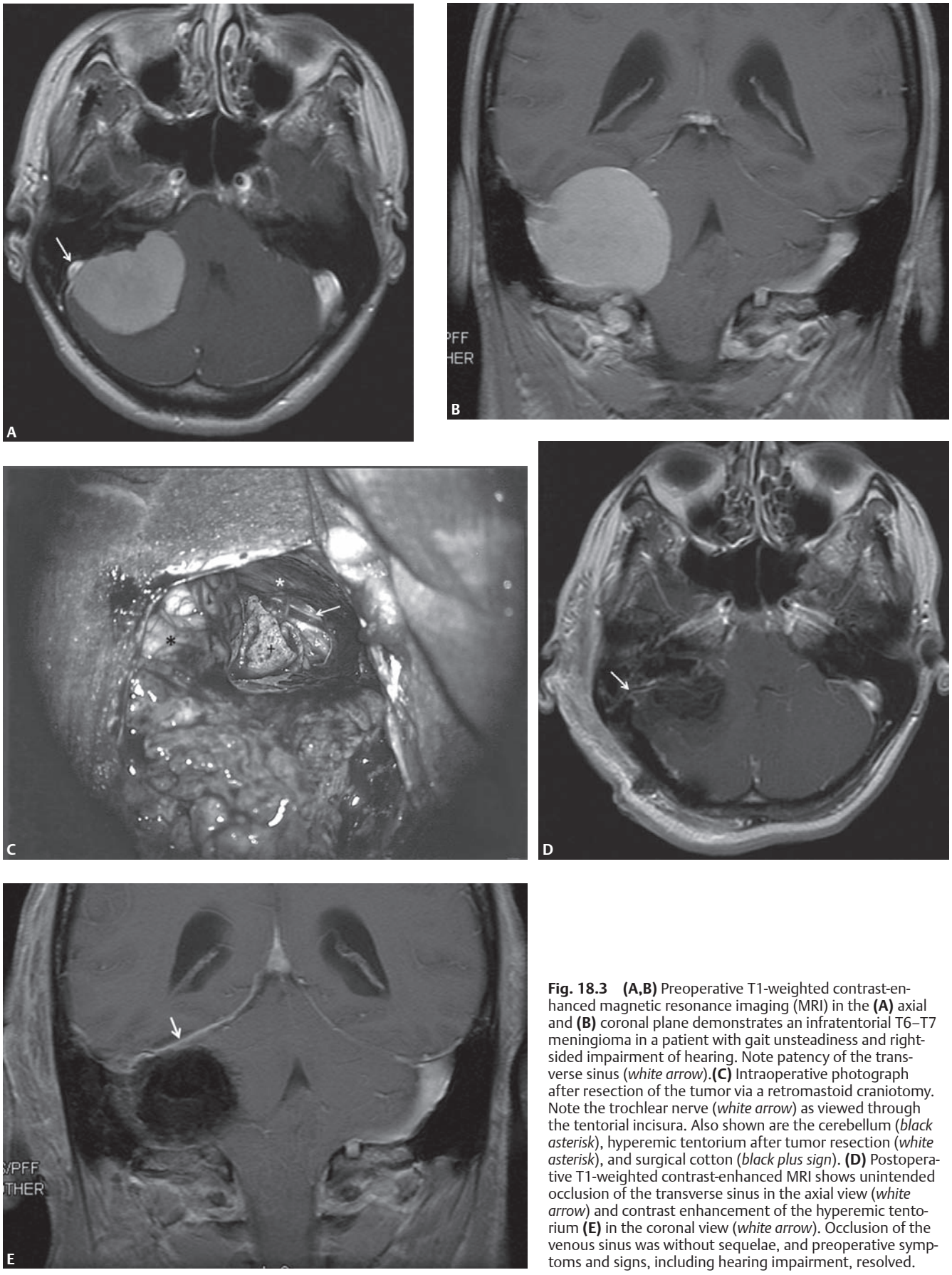


Fig. 18.3 **(A,B)** Preoperative T1-weighted contrast-enhanced magnetic resonance imaging (MRI) in the **(A)** axial and **(B)** coronal plane demonstrates an infratentorial T6–T7 meningioma in a patient with gait unsteadiness and right-sided impairment of hearing. Note patency of the transverse sinus (*white arrow*).**(C)** Intraoperative photograph after resection of the tumor via a retromastoid craniotomy. Note the trochlear nerve (*white arrow*) as viewed through the tentorial incisura. Also shown are the cerebellum (*black asterisk*), hyperemic tentorium after tumor resection (*white asterisk*), and surgical cotton (*black plus sign*). **(D)** Postoperative T1-weighted contrast-enhanced MRI shows unintended occlusion of the transverse sinus in the axial view (*white arrow*) and contrast enhancement of the hyperemic tentorium **(E)** in the coronal view (*white arrow*). Occlusion of the venous sinus was without sequelae, and preoperative symptoms and signs, including hearing impairment, resolved.

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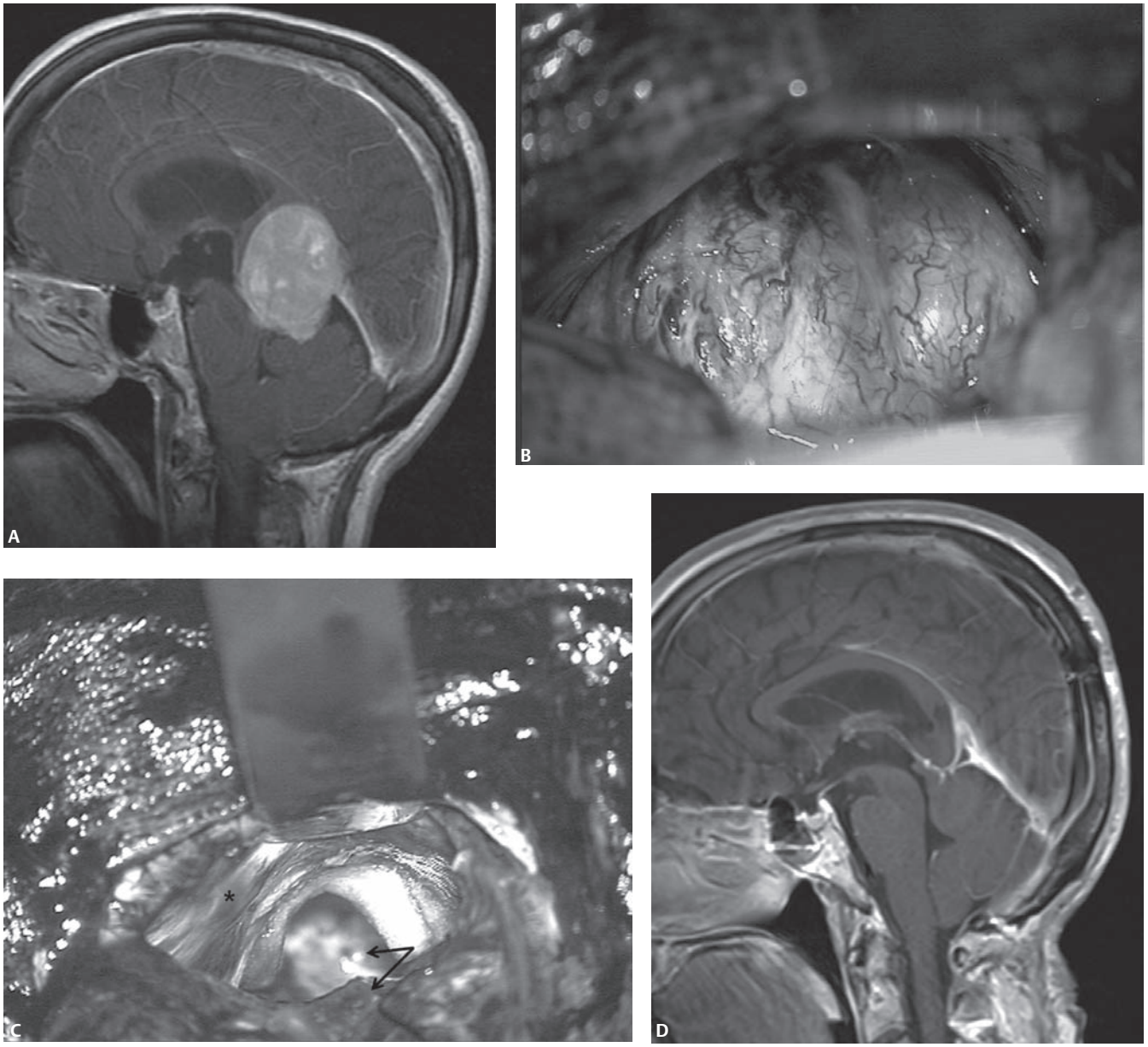


Fig. 18.4 **(A)** T3–T8 meningioma displayed in preoperative sagittal contrast-enhanced T1-weighted magnetic resonance imaging (MRI) in a patient presenting with gait ataxia and slight mental deficits. Note the patency of the straight sinus and accompanying obstructive hydrocephalus due to occlusion of the aqueduct. **(B)** An intraoperative photograph shows the tumor as viewed via the supracerebellar infratentorial approach. **(C)** A second intraoperative photograph reveals the pineal region above the cerebellum (*black arrows*) and below the tentorium (*black asterisk*) after resection of the meningioma. **(D)** Three months postoperative sagittal contrast-enhanced T1-weighted MRI demonstrates removal of tumor. Note patency of the aqueduct, straight sinus, inferior sagittal sinus, vein of Galen, and internal cerebral veins. Obstructive hydrocephalus and preoperative neurological symptoms resolved.

ficiently studied. The interface between the tumor and the brain stem is well depicted on T2-weighted MRI. A hyperintensive signal within the brain parenchyma indicates disruption of the blood–brain barrier and difficulties in resecting the tumor from the brain stem may be anticipated. It is often safer to leave a tumor remnant in these situations. The drainage pattern of the subtemporal venous complex and patency of venous sinuses are demonstrated with good-quality magnetic resonance venography (MRV). This investigation has largely replaced invasive catheter angiography. Computed tomographic (CT) scanning is less important for the preoperative workup of tentorial meningiomas due to its inaccuracy in demonstrating posterior fossa pathologies and due to the fact that involvement of bone is not a prominent feature in these tumors. The rare presence of tumor calcification can give some clue to tumor consistency. Even the most sophisticated radiological investigations available today have important shortcomings that should be recognized. A nonvisible venous sinus on MRV or catheter angiog-

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raphy may prove to be patent during surgery. Tentorial sinuses, which may be a source of brisk bleeding, are usually not visualized preoperatively.[19] Reliable information on functional significance of venous sinuses and major veins is usually lacking. Finally, the exact relationship of the tumor to neighboring cranial nerves (displacement, infiltration) can only be fully appreciated during surgery.

Surgical Approach and Technique

Treatment of recurrent tentorial meningiomas carries a high risk of surgical morbidity and mortality due to the absence of safe arachnoid dissection planes that help to protect important neurovascular structures. Resection of recurrent tumor is often incomplete. Therefore, the primary goal of treatment is complete resection of the tumor, including its dural origin and any involved bone at first operation.[8,20] The other nonnegotiable goal is preservation of the integrity of neurovascular structures and restoration of normal function or at least preservation of preexisting neurological function. The surgical approach should be performed after careful analysis of preoperative radiological investigations (**Table 18.2**). The lateral suboccipital retrosigmoid approach is suitable for most infratentorial T1–T2 and T6–T7 tumors. The supracerebellar infratentorial route may be more appropriate in more medial infratentorial T1–T2 tumors and in infratentorial T3–T8 tumors, and it is the approach of choice in infratentorial T4 tumors. Supratentorial tumor extensions can be resected transtentorially. It is usually possible to resect the tumor matrix along with the outer dural layer to achieve a complete resection.[8] The same principle applies to tumors attached to the wall of venous sinuses. The inner dural lining can often be kept intact. Sometimes, however, both dural layers are intermingled with tumor. In these cases we prefer preservation of a patent venous sinus and merely devitalize the tumor attachment area by low-current bipolar coagulation. A radiologically completely occluded venous sinus may be resected along with the tumor; however, there remains some uncertainty in regard to patency of the venous sinus. Atresia or septation in the region of the sinus confluence may lead to misinterpretation with regard to dominance of the venous sinus.

The usual avenue for resection of T3–T8 meningiomas is the occipital transtentorial approach. Transection of the tentorium should be individualized and not routinely performed because there is a risk of significant blood loss due to opening a tentorial sinus. The occipital approach should be considered for tumors that have displaced the galenic venous system inferiorly. This information can be obtained from preoperative coronal or sagittal contrast-enhanced MRI. Contrary to our earlier practice, bioccipital approaches are usually avoided. Contralateral tumor portions can be resected transfalcially, which includes interruption of the inferior sagittal sinus. If the galenic venous system is displaced superiorly, a supracerebellar infratentorial approach may be more appropriate for tumor resection. Further considerations in choosing the most suitable route for resection of T3–T8 tumors include steepness of the tentorium and location of the main tumor bulk in relation to a line drawn along the straight sinus on T1-weighted sagittal MRI scans. Complete removal of the rare T5 meningioma is often impossible because the venous sinus wall is usually heavily infiltrated, and no attempt should be made to resect it at this site. Removal of the supra- and infratentorial tumor bulk, usually via a "four quadrant" (bioccipital/bisuboccipital) craniotomy is followed by cautious bipolar coagulation along its sinus wall attachment.

Supratentorial T1–T2 and T6–T7 tumors can usually be resected via a subtemporal route. Positioning of the patient's head to take advantage of gravity and preoperative insertion of lumbar cerebrospinal fluid (CSF) drainage may facilitate temporal lobe retraction. The craniotomy should be flush with the base of the middle cranial fossa; in some

Table 18.2 Location of Tentorial Meningioma and Surgical Approach (***n*** = 108)

Tumor Location	No. of Patients	Surgical Approach
T1/2 (incisural)		
Infratentorial	11	Supracerebellar infratentorial Suboccipital retrosigmoid
Supratentorial	9	Subtemporal
Suprainfratentorial	5	Infrasupratentorial presigmoid
T3/8 (falcotentorial)		
Supratentorial	5	Bioccipital interhemispheric
Suprainfratentorial	6	Occipital transtentorial Bioccipital/suboccipital
Infratentorial	4	Supracerebellar infratentorial
T4 (paramedian)		
Infratentorial	13	Supracerebellar infratentorial
T5 (peritorcular)		
Suprainfratentorial	2	Bioccipital/suboccipital
Infratentorial	2	Supracerebellar infratentorial
T6/7 (lateral)		
Infratentorial	43	Suboccipital retrosigmoid Supracerebellar infratentorial
Suprainfratentorial	1	Retrosigmoid-subtemporal
Supratentorial	5	Subtemporal
Falx cerebelli		
Infratentorial	2	Supracerebellar infratentorial

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cases it is advantageous to add a zygomatic osteotomy to provide a flatter angle of view to the lesion.[21] The relation of the zygomatic arch to the base of the middle cranial fossa can be appreciated on preoperative reformatted coronal bone window CT scan. The craniotomy should be large enough to facilitate preservation of the subtemporal veins.[22] In supratentorial meningiomas that have not extended to the surface of the petrous bone, a paramedian supracerebellar transtentorial approach has been suggested for T1–T2 meningiomas to avoid the veins of the temporal lobe.[23] The suprainfratentorial presigmoid approach was rarely applied in our series and was reserved for large suprainfratentorial T1–T2 meningiomas.[24]

Patients harboring an infratentorial T1–T2 and T6–T7 tumor as well as those with T4, T5, and T3–T8 meningiomas were operated on in the semisitting position after employing standard anesthetic monitoring to detect and treat air embolism. Although we have not encountered significant adverse complications using this patient position, alternative positions such as the three-quarter prone, park bench, or Concorde position may be used to reduce the risk of air embolism. In patients with significant obstructive hydrocephalus, insertion of a ventricular CSF drain at the time of tumor resection to relieve elevated brain pressure is advisable.

Complications and Outcome

Recent microsurgical series report a mortality rate of 0 to 3.7% and a surgical morbidity rate ranging between 14 and 55%.[8,9,13,14,25] Complications are often transient and resolve on follow-up.[8,13,25] Postoperative morbidity is related to the tumor site and to the approach selected (**Table 18.3**). CN III, IV, and V are endangered during resection of T1–T2 tumors and in subtemporal and petrous transtentorial approaches, whereas CN VII and VIII are jeopardized in infratentorial T6–T7 tumors and the retromastoid approach.[8] One patient in our series experienced a persistent cortical blindness after undergoing a bilateral occipital transtentorial approach for resection of a large T3–T8 tumor. Prolonged and pointed spatula retraction of the medial occipital lobe (calcarine area) should be avoided to prevent ischemia with resulting postoperative visual compromise. To reduce the incidence of postoperative complications, tumor resection should strictly follow arachnoid cleavage planes. The arachnoid may be disrupted in large and recurrent tumors. It is our policy to leave a tuft of tumor if it is adherent to critical structures, namely the brain stem, cranial nerves, and blood vessels, to prevent serious neurological sequelae or even surgical fatalities. Functional outcome in patients harboring tentorial meningiomas is expected to be favorable if microsurgical principles are applied. In recent microsurgical series, 75 to 86% of the patients resumed a normal life after surgery with no or minimal symptoms (Karnofsky Performance Scale [KPS] score 80 to 100).[8,13,19,25,26] Serious complications requiring permanent postoperative assistance of the patient (KPS score ≤ 50) are reported in less than 5% of the cases.[8,19,25]

Resection Rate and Tumor Recurrence

Total tumor resection rate depends on the site, pattern of growth, and extent of the tumor. A Simpson grade I and II removal is reported in 77 to 91% of the patients in microsurgical series.[8,9,13,14,19,25,27] A complete tumor resection is usually achieved in T4 meningiomas, whereas a total removal is often not feasible in the face of en plaque growth and in the T1–T2 and T5 subgroups. T6–T7 tumors can usually be completely resected via a retromastoid craniotomy, but infiltration of the adjacent venous sinuses may prevent complete resection. Current data in the literature do not support aggressive resection of an infiltrated patent venous sinus.[8,9,15,19] Furthermore, it has been demonstrated that subtotal removal of meningioma can be associated with a long progression-free period and high quality of life.[28] We therefore recommend preservation of an infiltrated but patent dural venous sinus. Tumor recurrence or progression after subtotal removal has been noted in 0 to 26% of the patients in larger series with a long follow-up.[8,9,13–15,19,25] Stereotactic radiosurgery may be a useful treatment option in these patients. A recent small series using stereotactic radiosurgery in recurrent or incidental tentorial meningiomas reported an overall tumor control rate of 98% after a (short) mean follow-up period of 3 years.[29]

Results

From January 1989 to July 2008, 108 patients harboring a tentorial meningioma were treated microsurgically in the University Clinic of Essen and the Academic Hospitals of Kaiserslautern and Ingolstadt, Germany. Tentorial meningiomas constituted 7.7% of all intracranial meningiomas treated in our facilities during this time period. The main presenting symptoms in the 92 women and 16 men, mean age 55 years (range 18 to 72 years) were headache (70%), dizziness (46%), and gait unsteadiness (43%). Clinical examination revealed a gait ataxia in 50%, a CN deficit in 28%, and mental changes in 13% of the patients (**Table 18.1**). Infiltration of a dural venous sinus, predominantly the transverse sinus, was present in 34 patients (31%); complete occlusion of the transverse sinus was confirmed intraoperatively in only four patients, in whom a radical resection of the tumor, including the venous sinus, was performed. A Simpson grade I and II was achieved in 98 patients (91%). A gross tumor remnant (Simpson grade IV) was left in seven patients (7%) due to en plaque growth or tight adherence of the tumor to critical neurovascular structures (**Table 18.4**). Permanent surgical morbidity and mortality rates were 18% and 2%, respectively. Twelve (11%) tumor recurrences were observed after a mean follow-up period of 7.4 years (1 to 18 years) with clinical and MRI examination. Of these, seven patients underwent a second surgery. One additional patient with a malignant falcotentorial meningioma died on follow-up due to intracranial tumor dissemination. Ninety-five patients (88%) resumed full daily activity with either no or minor symptoms (KPS score 80 to 100). Six patients (6%) require permanent daily assistance due to severe neurological deficits (KPS score ≤ 50).

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Table 18.3 Intra- and Postoperative Complications in 108 Patients with Tentorial Meningioma

Tumor Site	Complication	No. of Patients	Tumor Site	Complication	No. of Patients
T1/2	Hemiparesis	3 (permanent in two)	**T5**	Gait ataxia	1 (improved)
	Hemianopia	1 (permanent)		Hemiparesis	1 (walking but permanent)
	CN III deficit	2 (permanent in one)			
	CN IV deficit	2 (permanent in one)	**T6/7**	Gait ataxia	1 (postoperative deterioration)
	CN V deficit	1 (permanent)		Hemiparesis	1 (resolved)
	Gait ataxia	1 (improved)		Cerebellar swelling	1 (decompression)
	Mental disturbance	1 (resolved)		Cerebellar abscess	1 (surgical revision)
	Epidural hematoma	1 (surgical revision)		Air embolism	4 (no sequelae)
	CSF leak	2 (treated with lumbar drainage)		CSF leak	4 (lumbar drainage in three, shunting in one)
	Meningitis	1 (treated with antibiotics)		CN VII	2 (permanent in one)
	Death	1		Tinnitus	1 (permanent)
				Meningitis	1 (resolved with antibiotics)
T3/8	Hemiparesis	2 (permanent in one)		Transverse sinus occlusion	3 (no sequelae)
	Cortical blindness	1 (bilateral, permanent)		Wound infection	2 (surgical revision in 1, antibiotics)
	Gait ataxia	1 (resolved)			
	Phlebitis	1 (no sequelae)			
T4	CN V deficit	1 (partially resolved)			
	Hemorrhage	1 (surgical revision)			
	Gait ataxia	1 (permanent)			
	Air embolism	1 (no sequelae)			
	Death	1			
Total	47 complications (44%), permanent in 19 (17.9%) 2 patients died (mortality 1.9%)				

Abbreviations: CN, cranial nerve; CSF, cerebrospinal fluid.

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Table 18.4 Extent of Tumor Resection According to Simpson's Grading and Recurrences (***n*** = 108)

Simpson's Grade	Tumor Site*					Falx Cerebelli
	T1/2	T3/8	T5	T4	T6/7	
I	6	4	0	11	18 (1)	2
II	15 (2)	11 (2**)	2	2 (1)	27 (3)	0
III	0	0	0	0	1‡	0
IV†	4 (1±)	0	2 (1)	0	1 (1±)	0
Unknown	0	0	0	0	2	0
Total	**25**	**15**	**4**	**13**	**49**	**2**

* Recurrences/regrowth in parentheses.
** One chondroid (World Health Organization grade II) and one malignant (World Health Organization grade III) subtype.
† Incomplete removal usually due to tight adherence to brainstem, cranial nerves or vascular structures.
‡ Infiltration of petrous bone.
± En plaque growth.

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Chapter 19

Peritorcular Meningiomas

Griffith R. Harsh IV

Whether in relation to the torcular they start in the NE, NW, SW or SE corners, [peritorcular meningiomas] may come in time to box the entire regional compass; and when they have done so, there are few more formidable lesions surgically to encounter. —H. Cushing, 1938.[1]

Introduction

Torcular meningiomas arise from, invade, or are attached to a wall of the torcular Herophili, the site of confluence of the superior sagittal, straight, occipital, and both transverse sinuses (**Fig. 19.1**). Dura forming the torcular Herophili comprises part of their primary dural base. Peritorcular meningiomas, in contrast, arise primarily from the dura of the posterior falx cerebri, posteromedial tentorium cerebelli, superoposterior falx cerebelli, or adjacent occipital or suboccipital convexity; although they may alter the venous flow through the torcular, they do not involve the torcular wall itself. Torcular and peritorcular meningiomas, hereafter referred to jointly as peritorcular meningiomas, although rare, have a special place in neurosurgical history. As noted by Cushing and Eisenhardt, the tendency of these tumors to produce visual field defects facilitated their preoperative localization, and such precise localization encouraged neurosurgical pioneers to attempt to remove them.[1] Birdsall and Weir in 1887 reported the first effort at removal of a peritorcular meningioma; their case was one of the earliest intracranial operations for tumor, and it ended in fatal postoperative hemorrhage.[2] One of the first successful operations for brain tumor was the removal of a parasagittal and peritorcular meningioma, as reported by Oppenheim and Krause in 1906.[3] The first case in Dandy's initial description of ventriculography was a peritorcular meningioma.[4]

The imprecision with which the term *peritorcular meningioma* has been used and the rarity of meningiomas in the torcular region make estimation of their true incidence difficult. Cushing and Eisenhardt's 12 peritorcular meningiomas constituted 16% of their 77 parasagittal tumors.[1] Additionally, three series of posterior fossa meningiomas included four (5%) of 82 peritorcular meningiomas,[5–7] and three series of tentorial meningiomas contained 14 (13%) of 109 peritorcular meningiomas.[8–10] If the relative incidences of parasagittal, posterior fossa, and tentorial tumors are 10%, 10%, and 5%, respectively, of all intracranial meningiomas, then peritorcular tumors represent ~1% of intracranial meningiomas.[11]

Pathology

Peritorcular meningiomas arise from arachnoidal cap cells in the region of the torcular Herophili. All subtypes of meningioma may develop. Cushing and Eisenhardt noted a predominance of angioblastic meningiomas in their group of peritorcular meningiomas; of the 12 tumors, six were angioblastic, five of which showed malignant histologic and clinical features. Three of 12 tumors were of the fibroblastic "whorl" type, one was psammomatous, one was fibrotic, and one was mesothelial. Such a skewed distribution of histologic subtypes has not been noted in the peritorcular tumors of other series of meningiomas. Interesting variants of peritorcular meningiomas include those associated with extensive hyperostosis in the region of the internal occipital protuberance (e.g., Cushing's case 8, in which there was hyperostosis from the lambda to the foramen magnum) and those with en plaque extension of tumor along the venous sinuses (e.g., also Cushing's case 8, in which tumor extended along the transverse sinus to involve three of the four peritorcular quadrants).[1] Although meningiomas of benign histology may extend through dural barriers, this extension occurs with greater frequency among tumors with malignant histologic characteristics.

Although arachnoid cap cells giving rise to peritorcular meningiomas exist in each quadrant of occipital and suboccipital dura about the intersection of the superior sagittal, occipital, and each transverse sinus, they are relatively abundant along the posterior margin of the tentorium.[8]

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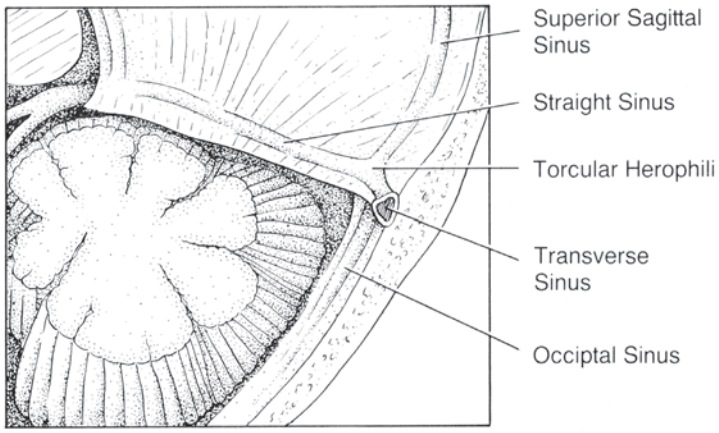


Fig. 19.1 Peritorcular venous anatomy. The interrelations of the five venous sinuses in the region vary from individual to individual. The sinuses as well as the torcular itself may be septate. Often a true confluence may not exist.

The dura is a poor barrier to tumor extension; tumors frequently penetrate the distal falx or posterior tentorium to reach an adjacent torcular quadrant. By compressing or invading the superior sagittal, straight, transverse, and/or occipital sinuses, peritorcular meningiomas threaten dural venous sinus flow at the torcular Herophili. The anatomical relationship of peritorcular meningiomas to the posterior dural venous sinuses is paramount because of its profound implications for the clinical presentation, diagnosis, treatment, and outcome of patients with these tumors.

◆ Clinical Presentation

Because of their low incidence, peritorcular tumors have not been distinguished from other meningiomas in reported series other than that of Cushing and Eisenhardt.[1]

In that series, seven patients were male and five were female.[1] The average age at presentation was 35.5 years. These patients uniformly had large tumors causing severe neurological deficits. The median interval between symptom onset and diagnosis was 1 year; the mean interval was 1.5 years. Predictably, the presenting symptoms and signs reflected either occipital or cerebellar compression or intracranial hypertension secondary to venous outflow obstruction (**Table 19.1**). All patients had papilledema; at least half also had homonymous field cuts suggestive of an occipital contribution to their visual loss and the supratentorial presence of tumor. Headache was global in half of the patients who noted it; this most likely reflects increased intracranial pressure. Occipital and suboccipital pain, probably resulting from deformation of surrounding dura, was described by the other half. Neck pain and stiffness may have indicated incipient tonsillar herniation. Cerebellar signs, indicative of infratentorial tumor extension, included nystagmus, dysmetria, hypotonia, and ataxia. Notably, there was no case of acute neurological deterioration that might result from sudden thrombosis of a partially occluded dominant sinus. Presumably, the slow growth of these tumors, except in cases of malignant histology, permits the development of sufficient collateral flow to preclude such a catastrophe.

Table 19.1 Clinical Presentation of Patients with Peritorcular Meningiomas (***n*** = 12)*

Symptoms		Signs	
Visual loss	11	Papilledema	12
Headache	10	Homonymous field cut	7
Neck pain/stiffness	4	Cerebellar deficits	5
Gait difficulty	3	Scotoma/atrophic blindness	3
Memory problems	2	Cortical sensorimotor loss	3

* Reported by Cushing and Eisenhardt.[5]

Modern imaging has permitted documentation of progressive tumor expansion that displaces the adjacent occipital lobe or cerebellar hemisphere and compresses peritorcular sinuses. Dysfunction of the occipital lobe (visual field loss and seizures) or cerebellum (ataxia and weakness) may result from direct compression by tumor or from regional venous congestion. Venous congestion may be widespread and result in symptoms of generalized intracranial hypertension (headache, vomiting, and seizures). The development of these clinical symptoms is usually insidious, and the tumor may become very large before symptoms become clinically evident. An exception to this tendency is the tumor that arises in the wall of a dural sinus and extends inwardly rather than outwardly; in such a case, florid clinical symptoms can be produced by a small tumor obliterating a venous sinus lumen (**Fig. 19.2**). Rational selection of cases for surgery requires a detailed understanding of the anatomy of the lesion, consideration of the indications and contraindications of surgery for each individual patient, and awareness of the alternatives to complete surgical resection.

◆ Diagnostic Imaging

The most valuable neurodiagnostic tools for assessing the anatomy of peritorcular meningiomas are gadolinium-

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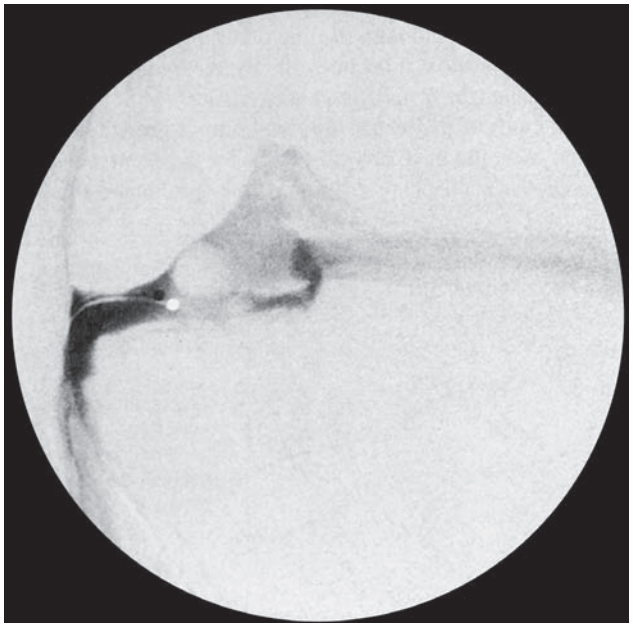


Fig. 19.2 Intraluminal tumor growth. A selective retrograde venogram of the peritorcular venous sinuses depicts a spherical meningioma within the right transverse sinus obstructing venous outflow from the torcular.

enhanced magnetic resonance imaging (MRI), magnetic resonance angiography/venography (MRA/MRV), and multiprojection subtraction angiography.

Most meningiomas are isointense with brain on T1-weighted images. There is some increase in relative intensity of tumor to brain on T2-weighted images. Large parenchymal or tumor vessels are apparent as cylindrical areas of signal void. Intratumoral calcification appears as irregularly shaped signal void on T1- and T2-weighted MRI scans. Peritumoral edema shifts from hypointense on T1-weighted images to hyperintense with T2 weighting. Gadolinium enhances the contrast between tumor and brain (**Figs. 19.3** and **19.4**). The distinctiveness of the margin between tumor capsule and cortex may correlate with the ease of maintenance of a plane of surgical dissection and with benign rather than invasive malignant histology.

Multiplanar MRI offers anatomical detail of the tumor and its relation to adjacent venous sinuses and the tentorium; this information is extremely valuable in preoperative planning. The coronal view (**Fig. 19.3A**) clearly shows the relation of the tumor to the tentorium, and the midsagittal view shows the relation of the tumor to the falx cerebri and falx cerebelli (**Fig. 19.3B**). Supratentorial and infratentorial portions of the tumor are readily distinguished. Tumor extension along the falces and the tentorium is evidenced by thickening of dural leaves, which appears more intense with administration of gadolinium.

Gadolinium-enhanced MRI not only identifies sinus walls involved with the tumor but also helps to predict the status of the sinus lumina. A patent sinus has a signal void characteristic of flowing blood: this is especially evident on T2-weighted scans, which maximize contrast with the

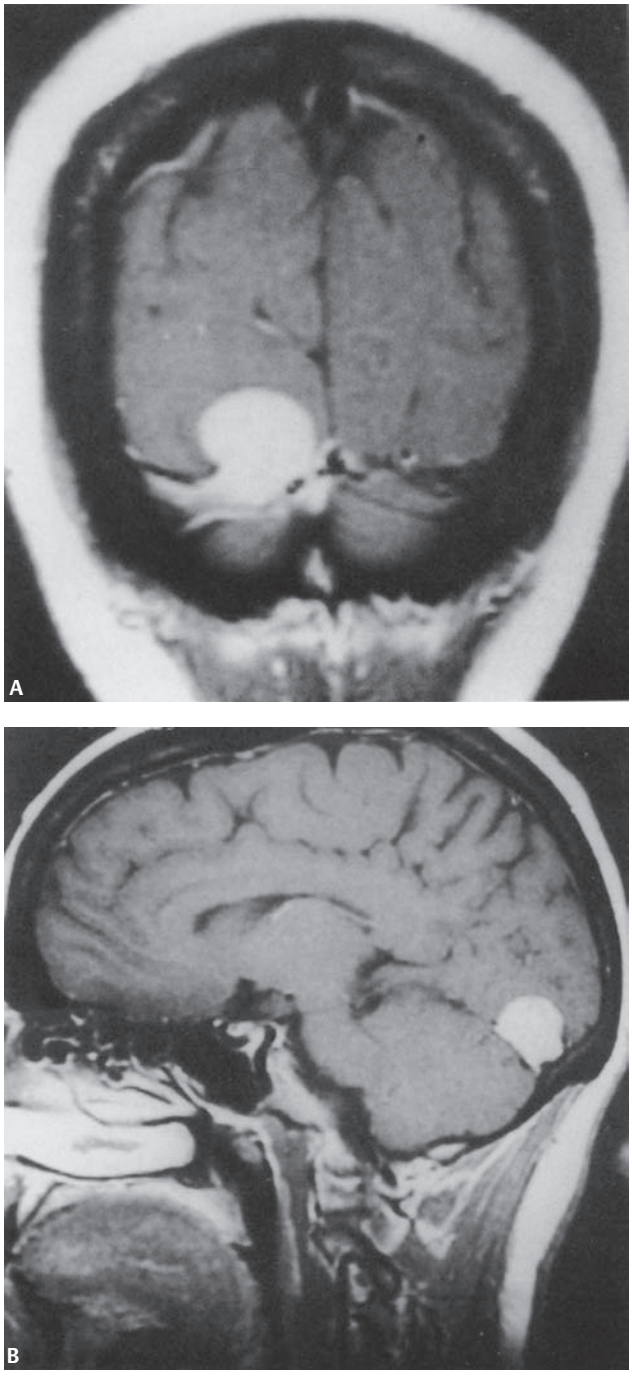


Fig. 19.3 TI-weighted gadolinium-enhanced magnetic resonance image in **(A)** coronal and **(B)** parasagittal plane. Multiplanar imaging of peritorcular meningiomas depicts the tumor's relation to the torcular, greatly facilitating the planning of the surgical approach. This peritorcular meningioma envelopes the right transverse sinus at the torcular.

higher intensity of cerebrospinal fluid (CSF). A partially occluded sinus is heterogeneous in intensity; regions of signal void corresponding to normal flow are interrupted by regions of increased intensity corresponding to stasis. A uniformly intense sinus suggests complete occlusion: a sinus with very slow flow, however, occasionally appears

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bright and resembles a completely occluded sinus. MRA/MRV demonstrates the full course of all phases of vascular supply and can identify points of compromise of venous flow without the invasiveness of standard angiography.

The relation of normal arteries and veins to the tumor and the dural location of the blood supply to the tumor are best seen on multiplanar angiography. The feeding arteries (usually the middle meningeal and occipital branches from the external carotid artery, the meningeal branches of the vertebral artery, and the tentorial branches of the cavernous internal carotid artery) can be identified and possibly embolized, as can vessels within the tumor itself. Large feeding arteries can be filled with embolic material and occluded more proximally by a detachable balloon after embolization is completed.

Angiography also allows delineation of the peritorcular venous anatomy and of the pattern of flow in each sinus. The specific configuration of the torcular Herophili in an individual patient is often critical not only to a tumor's clinical pathophysiology but also to its resectability (**Fig. 19.4**). Rarely do the sinuses intersect symmetrically at a central point beneath the internal occipital protuberance.[12] Rather, peritorcular venous channels are usually asymmetrical and septate. The right transverse sinus frequently carries most of the superior sagittal sinus outflow and may be larger than the left transverse sinus, which frequently carries most of the straight sinus flow. The distal superior sagittal sinus has a double lumen in many cases. Occasionally, one transverse sinus is congenitally atretic or even absent, or distal outflow is obstructed because tympanic disease has occluded the sigmoid sinus or jugular vein.

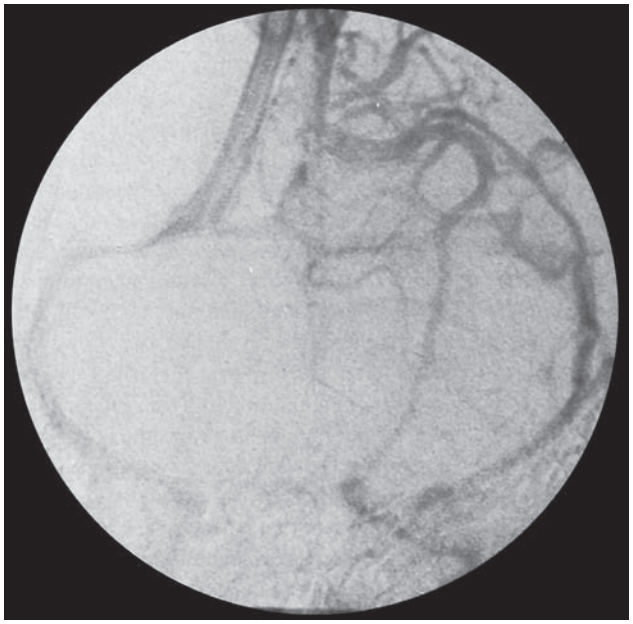


Fig. 19.4 Anterior-posterior projection of the venous phase of an internal carotid angiogram. Incomplete filling of the distal superior sagittal sinus, torcular, and right transverse sinuses suggests tumor compression of the proximal right transverse sinus. The left transverse sinus is congenitally atretic. Inadequacy of venous outflow resulted in elevated intracranial pressure.

Sinus occlusion manifests as absence of sinus filling and flow of blood from the torcular into dural, cerebral, or cerebellar collaterals. The details of the site and cause of venous sinus obstruction are best seen on retrograde dural sinus venography. Direct endovascular cannulation of the transverse sinus also allows assessment of tolerance of sinus obliteration; neurological function and intraluminal pressure proximal to the blockage should be measured during periods of balloon inflation. The extent of communication of the superior sagittal flow with each transverse sinus and of one transverse sinus with the other determines the safety of sinus occlusion and thus the surgical resectability of a peritorcular meningioma.

Selection of Treatment

Relief of neurological manifestations and the prevention of further tumor growth are the goals of treatment of patients with peritorcular meningiomas. These are best accomplished through total surgical resection of the tumor. Efforts at total removal, however, must be tempered by the patient's age, medical condition, and type of neurological deficit, by the probability that a catastrophic outcome will follow occlusion of a sinus on which the brain relies, and by the favorable long-term results achievable via subtotal resection followed by radiation therapy or, in cases of small tumors, stereotactic radiosurgery.[13,14] Clearly, a more aggressive surgical stance is warranted for an otherwise healthy young patient with progressive focal neurological deficits caused by a large tumor than for an elderly patient whose tumor, incidentally discovered, appears to be growing slowly.

When a patient's medical condition or the tumor's anatomical geometry precludes tumor resection by craniotomy, other surgical measures, such as optic nerve sheath fenestration to relieve visual deficits arising from elevated intracranial venous pressure or ventriculoperitoneal shunting of obstructive hydrocephalus, may be indicated. Such procedures, if they relieve neurological deficits, may also permit delay of an operation on a tumor that incompletely occludes a major sinus; if further tumor growth completely occludes the sinus, the sinus can be divided and resected along with the tumor. Ventriculoperitoneal shunting is not advisable if removal of the peritorcular meningioma is anticipated; intraoperative drainage of an expanded ventricle facilitates occipital-lobe retraction in supratentorial approaches to peritorcular tumors, and compression of the aqueduct and the fourth ventricle responsible for the hydrocephalus may be relieved by tumor removal.

Surgery

Surgical Planning

The choice of operative approach depends on the size and location of the tumor, the nature of sinus involvement, and the goals of surgery. The extent of peritorcular

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exposure necessary depends on the number of peritorcular quadrants containing tumor and on the tumor's geometry. A unilateral occipital craniotomy or a unilateral suboccipital craniectomy may suffice for cases in which the tumor occupies only a single quadrant and sacrifice of a major torcular component is not planned. Exclusively supratentorial tumors that penetrate the falx cerebri but not the tentorium may be exposed by a bilateral occipital craniotomy. In the case of large bilateral supratentorial extensions, the risk of inducing cortical blindness from bilateral calcarine injury may be reduced by monitoring visual evoked potentials or by staging sequential unilateral procedures. Similarly, exclusively infratentorial tumors that perforate the falx cerebelli can be removed through a bilateral suboccipital craniectomy. For unilateral tumors extending through the tentorium, a combined supratentorial and infratentorial approach is often justified. In many cases, however, the portion of tumor extending across the tentorium can be removed through a unilateral occipital craniotomy or suboccipital craniectomy alone. The supratentorial approach has the advantage of extensive exposure gained by wide lateral retraction of the occipital lobe; the few cortical veins that drain medially from the occipital lobe can be sacrificed without risk of neurological deficit. The infratentorial approach, however, diminishes the risk of retraction-induced injury to the visual cortex. In either case, wide transtentorial exposure can be obtained by dividing the tentorium from the incisura to the anterior margin of the transverse sinus. For tumors arising directly from the torcular wall, exposure of all four quadrants of the peritorcular region is advisable because it permits control of all venous flow in and out of the region.

Perioperative Management and Anesthetic Technique

Preoperative administration of prophylactic corticosteroids and antibiotics is indicated; anticonvulsants should be given if there is a history of seizure. General anesthesia using a combination of inhalation agents and intravenous narcotics is required. Moderate hyperventilation and osmotic diuresis reduce brain volume and facilitate exposure. The use of intermittent-compression boots reduces the risk of deep vein thrombosis. Two large-gauge intravenous lines are placed in the event that rapid volume replacement is needed. An arterial line facilitates monitoring of blood pressure and respiratory function. Signs of venous air embolism are sought with an esophageal cardiac Doppler monitor and a gauge for end-tidal P_{CO_2}.

Position

The position of the patient is a matter of the surgeon's preference. The 45-degree prone oblique, Concorde, and sitting positions all afford adequate access in unilateral approaches; for bilateral exposures, either the prone, the Concorde, or the sitting position is acceptable. The sitting position has a higher risk of venous air embolism.

Incision

An inverted U-shaped incision with its apex at the lambda and its base between the mastoid process is excellent for bilateral combined exposures. The incision may be narrowed, shortened, or even replaced by a linear incision if less exposure is required. The incision is carried through all layers of the scalp and pericranium, which are then elevated and held retracted over a rolled sponge.

Craniotomy/Craniectomy

Bone is ordinarily removed through a free-flap occipital craniotomy or a suboccipital craniectomy (**Fig. 19.5**). When traversing the superior sagittal sinus, transverse sinuses, torcular Herophili, thinning the bone with a drill and then removing the eggshell remnant with a fine bone punch will reduce the risk of sinus injury. Dural adhesion to bone resulting from osseous tumor invasion, hyperostosis, or patient age greatly increases the risk of injury to the dura, the tumor's feeding arteries, or a venous sinus. Subdural tracking of the craniotome footplate can be avoided by performing a strip craniectomy at the margins of the bone flap, exposing the marginal dura under direct vision, and interrupting dural arteries before removal of the bone flap. If the roof of a venous sinus is incorporated in the bone to be removed, its laceration can be avoided by performing a unilateral free-flap craniotomy to the edge of the superior sagittal sinus and torcular Herophili on one side: then, under direct vision, the superficial surface of the sinuses can be freed from overlying bone before the contralateral craniotomy plate is removed (**Fig. 19.5**). Infiltrated or hyperostotic bone should be removed and discarded. When dural adhesion to bone is unlikely because the patient is young, the tumor is small, and hyperostosis is absent, all four quadrants of the peritorcular region can be exposed by a single craniotomy flap. Peripheral epidural tacking sutures are placed.

Durotomy

The dura should be incised 1 to 2 cm from the tumor's margin (**Fig. 19.6A**). Large afferent tumor arteries within the dura should be well coagulated or clipped or both before being cut. The dura should be hinged to maximize the view of a peritorcular tumor (**Fig. 19.6B**). Relaxation of protruding tumor and/or brain is facilitated by draining CSF either supratentorially by ventriculostomy of the lateral ventricle or infratentorially by opening the cisterna magna. Additional attachments of the tumor to convexity dura should be coagulated and divided.

Tumor Resection

Tumor removal is best accomplished in four stages: (1) coagulation and division of the tumor at its attachments to accessible dural leaves and sinus walls; this

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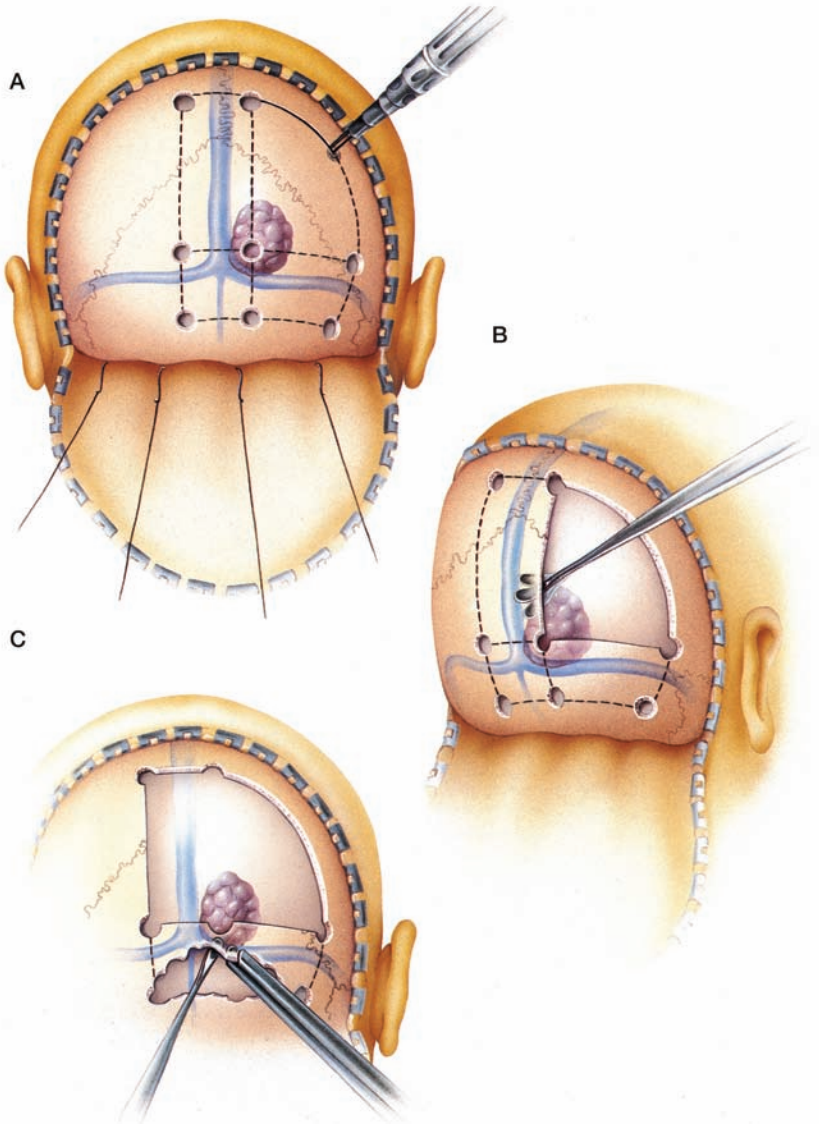


Fig. 19.5 Craniotomy. The extent of bone removal needed depends on the location and geometry of the peritorcular meningioma. Safe removal of bone over major venous sinuses can be accomplished by performing a unilateral free-flap craniotomy to the edge of the superior sagittal sinus and torcular Herophili on one side **(A)** and, under direct vision, freeing the superficial surface of the sinus from overlying bone before removing the contralateral craniotomy plate **(B)**. Piecemeal removal of bone thinned by a drill is the safest method of dural exposure when traversing the superior sagittal sinus, transverse sinuses, or torcular **(C)**.

devascularizes the main tumor mass of all but its parenchymal inflow; (2) internal debulking of the main tumor mass; (3) microdissection of the tumor capsule from the surrounding cortex; and (4) removal of tumor remnants along or within the major sinuses. Exposure of the dorsal surface of the tumor is increased by gentle superior retraction of the overlying occipital pole and inferior mobilization of the cerebellar hemisphere. As exposure is deepened, the remaining dural arterial inflow can be divided as access is gained (**Fig. 19.6C**). Removal of most tumors is accomplished by a repetitive series of internal debulking using morcellization by coagulation and suction or an ultrasonic aspirator (**Fig. 19.6D**) and microdissection of the thinned capsule away from occipital or cerebellar cortex. Meticulous extraarachnoidal microdissection that preserves the pia of the occipital lobe is critical to avoiding visual loss.

Management of Sinuses

The likelihood of achieving total resection of a peritorcular meningioma depends on the nature of sinus involvement by the tumor: only rarely is tumor resection precluded by tumor attachment to eloquent cortex or critical arteries. The portion of a venous sinus completely occluded by tumor can be resected safely. When removing an occluded superior sagittal sinus, special care must be taken to preserve the anterior and lateral collaterals carrying the hemispheric flow. An involved transverse sinus can be divided and resected if preoperative venography has shown that (1) the sinus is occluded by tumor or is congenitally atretic, (2) the medial portion of the sinus communicates completely with a patent contralateral transverse sinus of adequate caliber, or (3) the superior sagittal sinus and straight sinus are fully confluent with a patent contralateral transverse sinus of adequate

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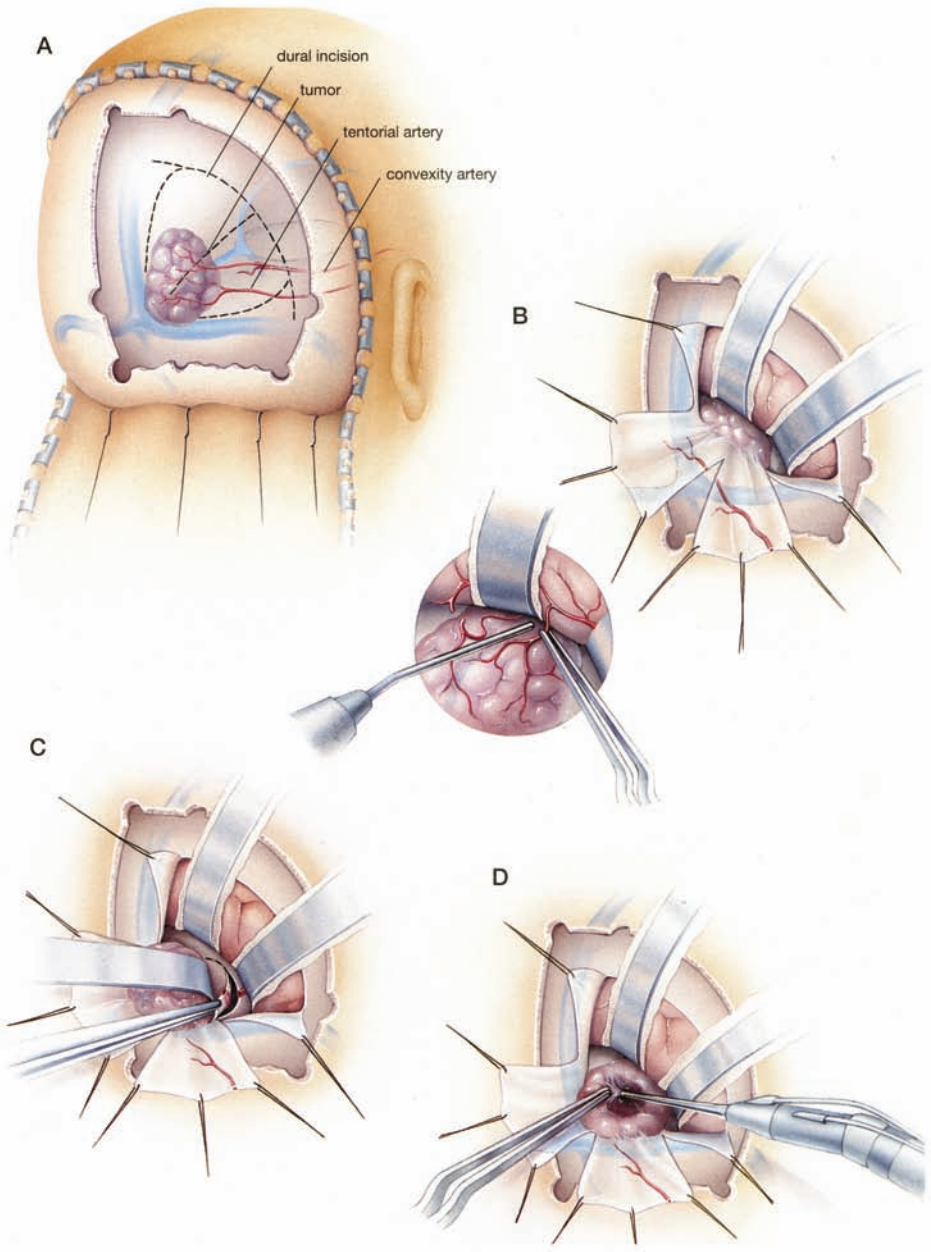


Fig. 19.6 Durotomy and tumor removal. **(A)** The dura is incised 1 to 2 cm from the tumor's margin and hinged to maximize exposure of **(B)** the peritorcular tumor. **(C)** As exposure is deepened, the remaining dural arterial inflow can be divided as access is gained. **(D)** Iterative internal tumor debulking (using morcellization by coagulation and suction or an ultrasonic aspirator) thins the annular capsule. **(Center Inset)** As the thinned capsule is infolded into the space created by debulking the tumor, meticulous microdissection attempting to preserve the arachnoidal plane and careful coagulation and sharp division of fine bridging vessels are essential to preserving the pia of the occipital lobe so as to avoid visual loss.

caliber. Interruption of a transverse sinus should be medial (posterior) to the junction of the vein of Labbé and the transverse sinus. If preoperative venography has not unequivocally demonstrated the adequacy of the contralateral sinus, trial occlusion of the involved sinus by temporary intraoperative clamping may help assess the compensatory capacity of the contralateral sinus. The occipital sinus is of little importance unless, as a hypertrophied collateral of an obstructed transverse sinus, it carries substantial superior sagittal or straight sinus outflow to the jugular bulb.

When the venous anatomy demands preservation of the flow through a sinus involved with tumor, the possibility of removing the tumor from the sinus depends on the extent of sinus involvement. If a tumor merely abuts or is attached to the sinus by arachnoidal adhesions, it can be peeled from the sinus wall. The external sinus surface should then be coagulated with bipolar cautery. When tumor invasion is limited to one wall or a corner of a critical sinus, the tumor external to the sinus should be truncated (**Fig. 19.7A,B**). The invaded wall or corner and intrasinus extension of tumor can often be removed, and the resultant defect closed with direct suture or patch graft. This is best done by sequentially opening and closing small segments of the sinus as the tumor is progressively removed from within the sinus. The walls of the partially opened sinus are grasped with vascular forceps or small clamps such that they can be released to permit removal of tumor or approximated to allow closure (**Fig. 19.7 C,D,E**). Transient opening of the sinus in such a con-

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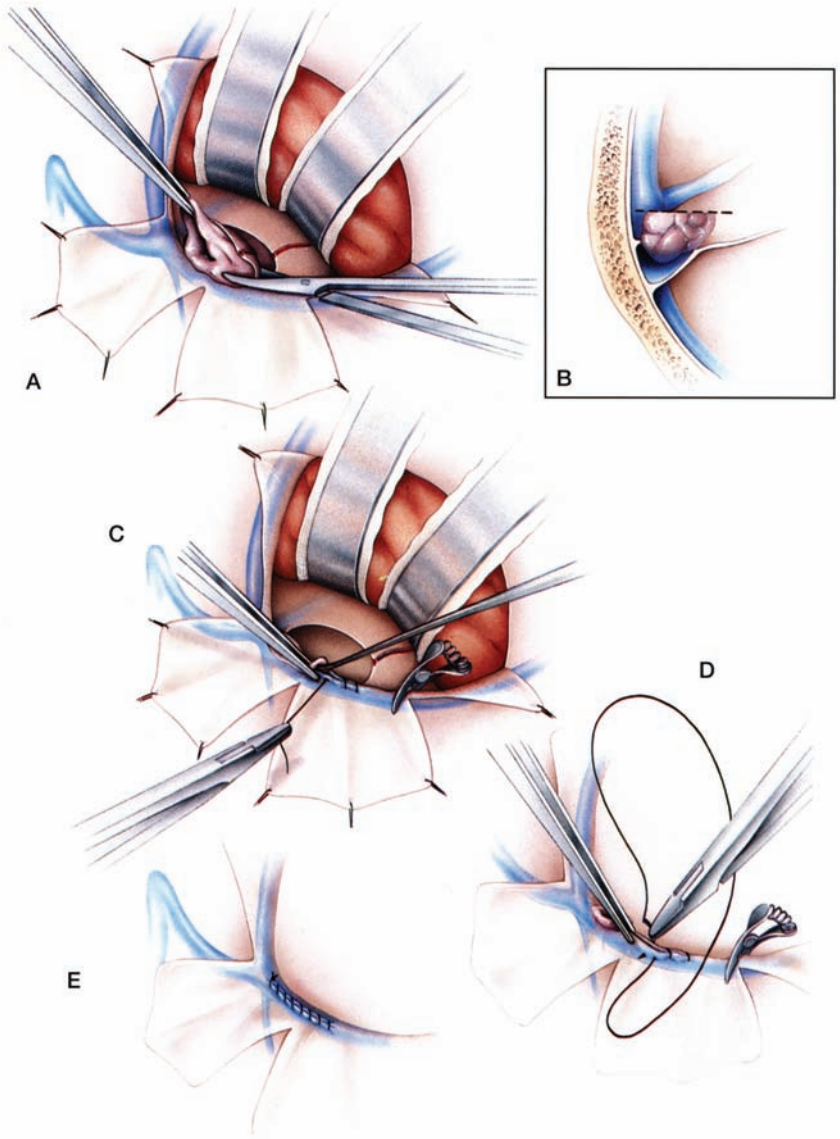


Fig. 19.7 Removal of sinus tumor and sinus repair. Removal of the remaining tumor outside the sinus provides unobstructed surgical exposure for the dural sinus work **(A)**. When tumor invasion is limited to one wall or a corner of a critical sinus or the involved sinus is occluded (as occurred with this peritorcular meningioma extending from within an occluded right transverse sinus into the torcular, **B Inset**), or the involved sinus is occluded, the tumor can be completely removed. The right transverse sinus, compromised by tumor has been clipped distally **(C)**. Proximally, the dural leaves of the torcular–transverse sinus junction are alternately opened, closed, and sutured as tumor extending into the torcular is progressively removed **(C,D)**. This restores full flow within the sinus and torcular **(E)**. If the sinus has been completely occluded by tumor that does not involve torcular walls, proximal and distal ligation and excision of involved transverse will also clear the torcular-transverse sinus junction of tumor.

trolled fashion permits removal of an intrasinus tumor with relatively little blood loss.

Removing a tumor involving more than one wall or corner of the sinus is much more hazardous because sinus flow must be interrupted during repair of the sinus. A blood-diverting shunt has been used in tumor removal and patch graft repair of the more proximal superior sagittal sinus.[15] An analogous approach using a shunt from the superior sagittal sinus to the transverse sinus during isolation and repair of a tumor-invaded torcular Herophili is appealing in theory but would likely be quite hazardous. Patch grafts that replace more than one sinus wall are often unsatisfactory because wall collapse and luminal obstruction can occur when sinus venous pressure is low. Removing a tumor involving the torcular itself might necessitate shunting flow from the superior sagittal, straight, and occipital sinuses and replacement of the torcular by a four-limbed prosthesis. In such cases, the risks concomitant with interruption or diversion of venous sinus flow and reconstruction of the torcular Herophili in an attempt to achieve complete resection of a peritorcular tumor are likely unwarranted.

Closure

Closure is begun only after the integrity of all sinus suture lines is confirmed during a Valsalva maneuver. Occipital and cerebellar cortex is checked for injury as retraction is released. Defects in convexity dura are closed with grafts of pericranium harvested from the inner surface of the scalp flap. The craniectomy plate is fixed with titanium miniplates. The craniectomy defect is bridged with titanium wire mesh. The wound is closed in two layers and dressed in routine fashion.

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Surgical Outcome

Postoperative Complications

Catastrophic outcomes can result from mismanagement of the dural sinuses. Failure to repair an opened sinus can result in life-threatening intracranial hemorrhage. Compromise of a previously patent sinus can lead to cerebral venous congestion, infarction, and intraparenchymal hemorrhage. Visual loss can result from this, as well as from indelicate microdissection of the tumor capsule or heavy-handed retraction of the occipital lobe. Excessive retraction is best avoided by extensive internal debulking of a tumor that permits inward collapse of an attenuated capsule. Infection is best avoided by attention to operative technique, copious irrigation of the operative site, and prophylactic antibiotics. The risk of seizures is diminished by careful manipulation of the occipital cortex, preservation of cortical venous drainage, and prophylactic anticonvulsants.

Long-Term Prognosis

The risk of tumor growth varies inversely with the extent of resection[16]; complete resection is often precluded by involvement either of the walls of the torcular or of more than one wall of an adjacent sinus. For such cases, the risks of attempts at resecting all involved dura are unwarranted. Small residua of tumors should be followed by periodic neurological examination and gadolinium-enhanced MRI (**Fig. 19.8**). Recurrent tumor growth can be treated by reoperation, stereotactic radiosurgery, or, if dural spread is extensive, fractionated radiation therapy (50 to 60 Gy).[13,14,17]

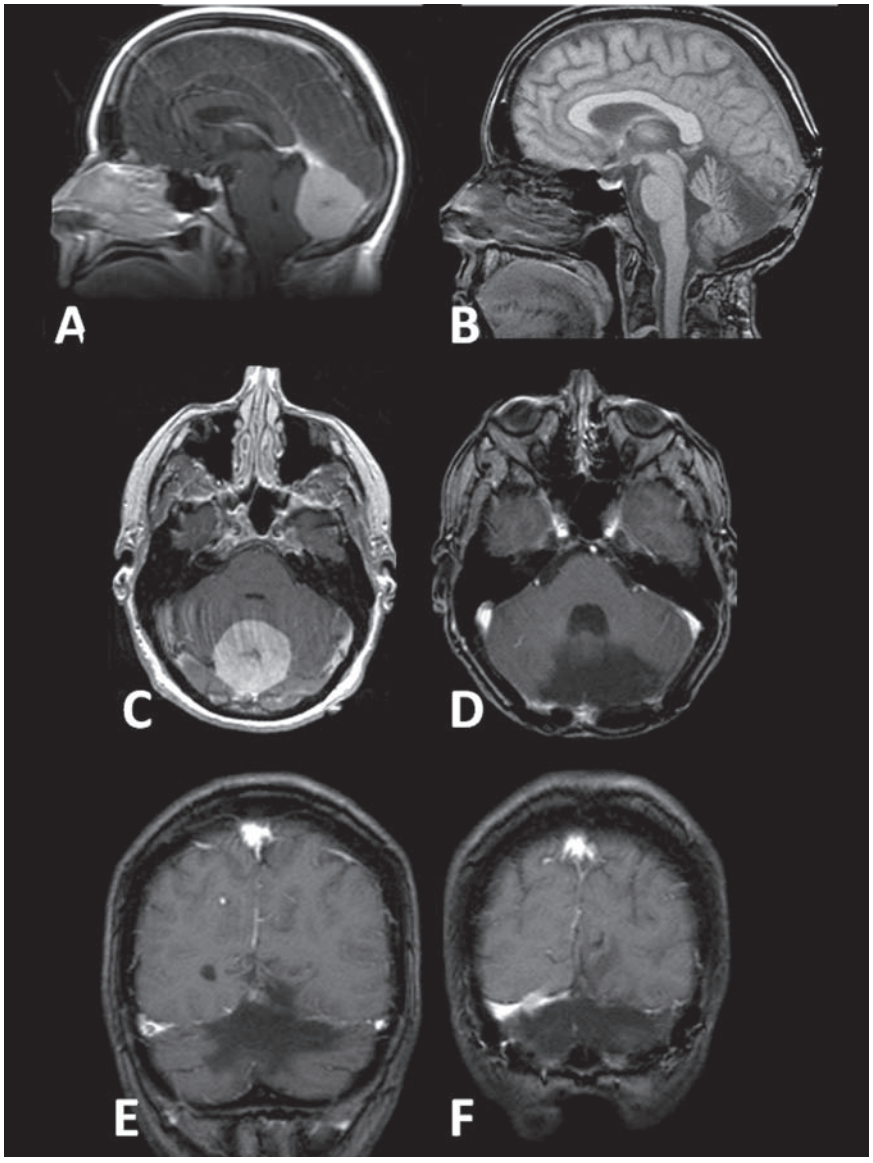


Fig. 19.8 Torcular-tentorial meningioma. A 56-year-old woman presented with headache and ataxia. This large meningioma arising from the torcular and tentorium along the straight sinus [T1-weighted magnetic resonance imaging (MRI) with gadolinium in **(A)** sagittal and **(C)** axial planes] was completely removed with the exception of a small plaque along the junction of the torcular and right transverse sinus **(E)**. Follow-up over 9 years has shown no recurrence—T1-weighted sagittal without gadolinium **(B)**, and axial **(D)**, and coronal **(E,F)** MRI, with gadolinium.

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Chapter 20

Falcotentorial Meningiomas

Alfredo Quiñones-Hinojosa and Michael W. McDermott

Introduction

Falcotentorial meningiomas are rare; ~60 cases have been reported.[1–25] These tumors occur at the junction of the dural folds of the tentorium and falx cerebri, either anteriorly at the junction of the vein of Galen with the straight sinus, or all along the length of the junction of the falx cerebri/cerebelli and tentorium in which the straight sinus, torcular, and arachnoid granulations are found. Large lesions involving the falcotentorial junction (FTJ) can be challenging to remove, leaving patients with transient or permanent neurological deficits, such as cortical blindness. Complications may arise during the ligation or manipulation of the sagittal sinus, bridging veins, vein of Galen, and torcula.

Symptoms and Signs

Most patients present with headaches (with or without nausea and vomiting) and visual disturbance.[19] Incontinence, personality changes, gait ataxia, dizziness, and mild cognitive impairments have also been reported.[3,13,16,26] Some of these complaints may be secondary to associated obstructive hydrocephalus.

The most common finding on physical exam has been papilledema, a sign of increased intracranial pressure.[3,13,16,26] Patients may also have cranial nerve dysfunction (such as facial droop or homonymous hemianopsia). Some series have reported that patients report progressively worsening symptoms for an average of 29 months (ranging from 6 months to 4 years) before seeking medical attention.[19]

Preoperative Planning

We recommend that patients be evaluated using magnetic resonance imaging (MRI) and cerebral angiography. For MRI, patients undergo T1-weighted imaging, T2-weighted imaging, and contrast enhancement with gadolinium. Magnetic resonance venograms can also be obtained and represented as a three-dimensional (3-D) object at surgery on image-guided systems. Cerebral angiography is usually performed in all cases to evaluate the arterial supply of the tumor, and the venous phase can be used as the gold standard to assess the patency of the straight sinus, transverse sinuses, and torcula. We also recommend that patients have a preoperative Humphrey visual field examination and funduscopic and acuity examination documented by a neuroophthalmologist. Patients and their families are counseled that postoperatively there will be a few days of cortical blindness with a bilateral occipital transtentorial approach and a hemianopsia with a unilateral occipital transtentorial approach. In both situations the blindness and field defects have spontaneously recovered in all the patients most recently reported in our series.[19]

Placement of a lumbar subarachnoid drain for drainage of cerebrospinal fluid during the case may be considered in large lesions. Postoperatively the drain is left in place, clamped off for the first 1 to 2 days so as not to potentiate a postoperative hematoma and to prevent the occurrence of an occult cerebrospinal fluid (CSF) leak through the durotomy made by the needle used to insert the drain. Intraoperatively, adjuvant treatments include administration of steroids and mannitol as well as hyperventilation, all of which reduce postoperative edema and intraoperative intracranial pressures.

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Radiographic Evaluation: Magnetic Resonance Imaging and Angiography

Most tumors in this region are found to have marked homogeneous enhancement upon administration of gadolinium. On T1-weighted images the main characteristic is isointensity to the brain parenchyma. On T2-weighted images, these lesions can be slightly hyperintense or hypointense, the latter predicting a fibrous or partly calcified nature of the tumor. Hydrocephalus can be present, and it has been reported.[19]

Preoperative cerebral angiography reveals that falcotentorial meningiomas usually derive their vascular supply from the internal carotid artery, external carotid artery, or the abnormal branches of the posterior cerebral artery. Supply from the internal carotid artery includes meningohypophyseal branches, branches off the inferolateral trunk, and the anterior choroidal artery. Posterior cerebral artery supply is derived from the medial and lateral posterior choroidal arteries. The external carotid supply for many of these tumors comes mainly from branches of the middle meningeal artery and falcine artery via the ethmoidal and ophthalmic arteries. If the predominant supply to the tumor is from a muscular branch of the left vertebral artery in the atlantal segment, this can be ligated with exposure of the region of the foramen magnum. In a majority of cases reported in the literature, either the vein of Galen or the straight sinus was occluded.[3] The transverse sinus can be partially occluded depending on the location of the tumor, usually in its proximal portions to the lateral edge of the tentorial component of the tumor.

Embolization is thought to be unsuccessful in most cases because the feeding vessels are either too small or inaccessible. Despite this, the results of the angiography can be key to determining preoperatively the status of the venous sinuses and therefore how they could be safely managed during tumor resection. Knowledge about the blood supply is useful in planning the surgery and understanding the vascular supply and anatomy of the tumor. This allows the surgeon to direct initial attempts at identifying and interrupting the arterial supply before tumor debulking.

Surgical Approach

In our experience, when the tumor is smaller (< 3 cm) and involves predominantly one side of the falx or tentorium, a unilateral occipital craniotomy with the retracted right occipital lobe in a gravity-dependent position can be used[3,16] (**Fig. 20.1**). In most cases a left hemianopsia seems to be tolerated better than a right hemianopsia, even transiently, due the fact that we scan from left to right for reading. For patients in the prone position, in addition to routine mannitol and steroids, we would consider placing a lumbar subarachnoid drain for smaller tumors and an external ventricular drain for those with hydrocephalus or some degree of posterior fossa mass effect. Drainage of CSF will reduce intracranial pressures and make the large dural openings on the convexity less troublesome.

Patient Positioning

Patient positioning (i.e., prone versus semisitting) depends on the surgeon's preference and the patient's body habitus. We recommend the prone or three-quarter-prone position because in our experience it is safe and has a lower risk of air embolism. In this position, the patient's neck is extended on the chest and the head flexed on the neck. We elongate the neck as much as we can by placing the neck in extension when the patient is prone and then flexing the head on the neck so that a finger passes between the chin and the chest to avoid rubbing between the lower portion of the jaw and the sternum.[20] In obese patients, the semisitting position is a viable alternative because the prone position makes it difficult to adequately ventilate the patient without causing extreme airway pressures leading to elevated transmitted venous pressures. Obese patients or patients in the semisitting position should be preoperatively evaluated for a patent foramen ovale to potentially avoid intraoperative air emboli. A thorough discussion with the anesthesia team will most times lead to the utilization of an armored endotracheal tube to avoid kinking or obstruction associated with positioning or prolonged operative time. We also recommend that an external ventricular drain be routinely placed via the parietooccipital trajectory with the aid of the surgical navigation system.

Craniotomy: Unilateral versus Bilateral Occipital Transtentorial/Transfalcine Approach

There have been several reports describing different surgical approaches for falcotentorial meningiomas. These include the infratentorial supracerebellar,[10,27,28] the biparietooccipital craniotomy in the sea lion position,[21] and a combined supra-/infratentorial transsinus approach as described by Sekhar et al and Ziyal et al.[29,30] Furthermore, Okami and colleagues have described the occipital transtentorial and the combined midline occipital and suboccipital approach.[16] The majority of these reports include patients without falcotentorial meningiomas and include patients with pineal meningiomas, teratomas, and pineal cysts, among others.[10,27,28,29,30] Isolated case reports from the past make it difficult to assess the efficacy of a particular type of approach.[16,29] In studies with more than two patients, gross total resection (GTR) was achieved in 25 to 50% regardless of surgical approach.[10,16,28,30] Complications, including vision loss and cranial nerve injuries, ranged from 20 to 50% in series that had more than two patients.[16,27,30]

For large tumors, a large U-shaped, inferiorly based incision is marked out to allow for a supra- and infratentorial craniotomy (**Fig. 20.2**). When the tumors are large

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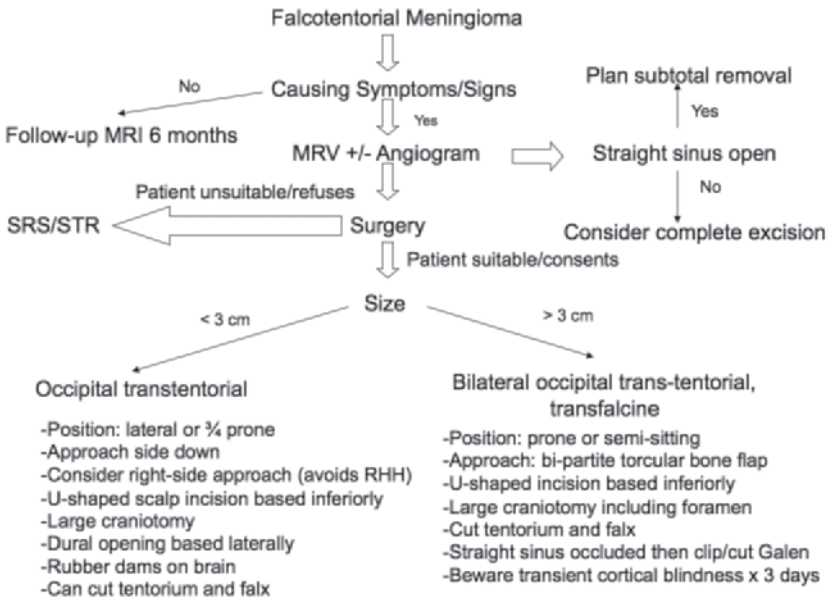


Fig. 20.1 Potential recommended management strategy for falcotentorial meningioma**s** depending on size and location.

and involve both sides of the tentorium, as well as the supra- and infratentorial spaces, a bilateral occipital transtentorial/transfalcine (BOTT) approach is recommended (8/9 cases in our most recent series).[19] We prefer this combined bilateral BOTT craniotomy technique, exposing the supra- and infratentorial compartments, for larger tumors, which have most often occluded the straight sinus.

In tumors with a significant infratentorial component, we prefer opening of the foramen magnum to reduce the resistance against inferior retraction of the cerebellar hemispheres. The bone flap over the torcular and transverse sinuses is removed in two components: the supratentorial bone flap is removed first and then the infratentorial bone flap is removed only after the dura over the torcular and transverse sinuses is dissected under direct vision (**Fig. 20.2**).

The advantages of the occipital transtentorial approach are that (1) it allows for good visualization of the internal cerebral veins as well as the posterior and lateral midbrain, (2) it has a low risk of air embolism, and (3) it provides a relatively wide exposure of the lesion. The wide exposure achieved with a torcular craniotomy that extends far out laterally reduces compression of the occipital lobes against the dural openings, especially in long cases. The availability of exposure above and below the tentorium also provides the surgeon with more options intraoperatively and allows one to alter the plan of attack when troublesome bleeding is encountered.

Dural Opening and Torcular/Transverse Sinus Management

The dura, both above and below the tentorium, is opened beyond the lateral margins of the tumor (**Fig. 20.3**). If the tumor involves the torcular, the tentorium can be incised on one side from immediately in front of the transverse sinus to its free edge anteriorly at the hiatus (**Fig. 20.4B** illustrates the potential cuts for a tumor involving the torcular—the cuts can be made similarly for a smaller tumor). The same maneuver can be done on the other side of the tentorium (**Fig. 20.4C**). This will eliminate the tentorial arterial supply.

Sinus ligation depends on the patency of the sinuses and the torcular structures. We recommend assessing the status of the torcular and transverse sinuses with preoperative angiography and venography and not from magnetic resonance venography. If the torcular and one or both of the transverse sinuses are patent, the tentorium is incised from lateral to medial in front of the torcular. However, if these sinuses are occluded, the sinuses are suture ligated at the lateral margin of the tumor, and the dura of the posterior fossa is opened. To determine the transition point between patent and occluded venous sinuses, we recommend the use of an intraoperative Doppler probe. The subdural space must be checked for adjacent draining veins that might be important in the collateral circulation that may have developed—we recommend their preservation. Ligation of the straight sinus can be done with a 2–0 braided nylon suture on a tapered needle with the needle passing through the tenorium, falx cerebri, tentorium, and falx cerebelli.

Tumor Debulking and Resection

For large tumors involving the FTJ, the falx cerebri over the top of the tumor is incised as far as practical. It is likely that the final incision through the anterior free edge can be done only after tumor debulking, which allows for better visualization of deep structures (**Fig. 20.4A**). After all the circumferential dural incisions around the tumor are made and a very thorough tumor debulking has taken place, then the tumor can be removed (**Fig. 20.5**).

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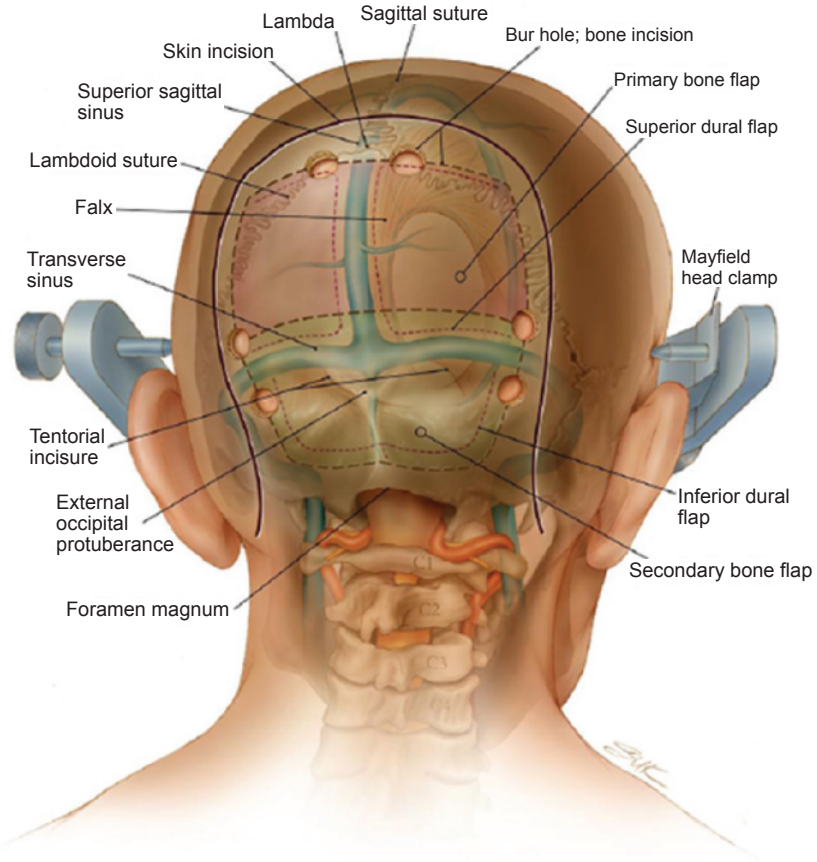


Fig. 20.2 Skin incision for a bilateral occipital transtentorial/transfalcine approach. A large U-shaped, inferiorly based incision. The foramen magnum may be opened if the tumor has a significant infratentorial component. We recommend removal of the bone flap over the torcular in two components, and a dural dissection over the sinus is done under direct visualization after the supratentorial bone piece is removed. With permission from Quinones-Hinojosa A, Chang EF, Chaichana KL, McDermott MW. Surgical considerations in the management of falcotentorial meningiomas: advantages of the bilateral occipital transtentorial/transfalcine craniotomy for large tumors. Neurosurgery 2009;64:260–268; discussion 268.

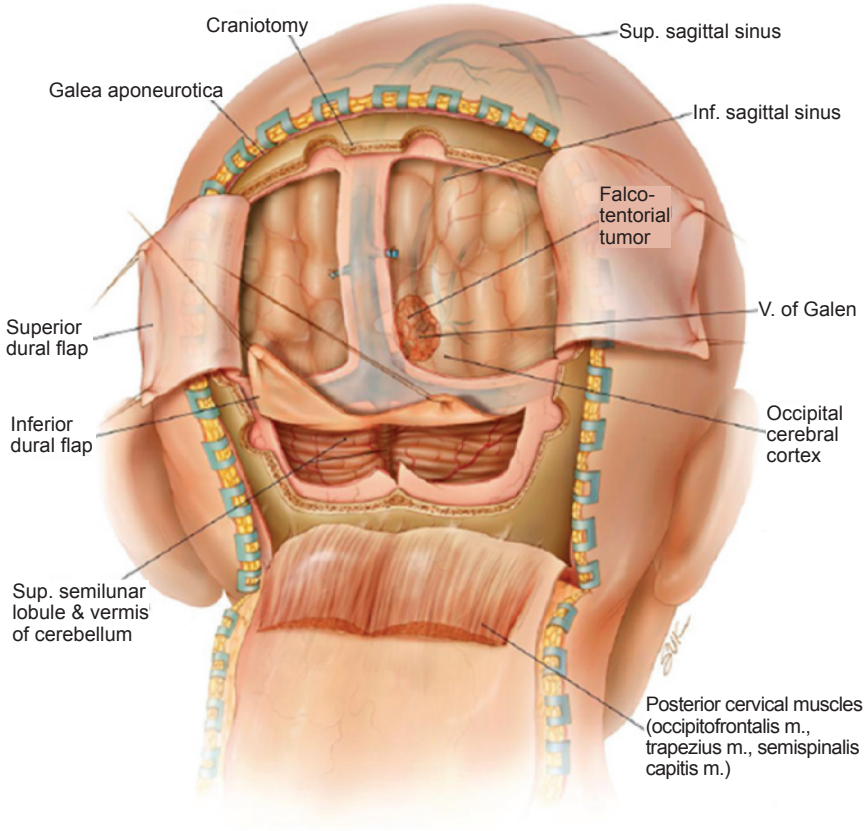


Fig. 20.3 Dural opening. Depending on the size of the tumor and involvement of the torcular, the opening can be done in one side or both sides of the superior sagittal sinus and above and below the tentorium/torcular/transverse sinuses. An opening beyond the lateral margins of the tumor allows for a more complete resection. With permission from Quinones-Hinojosa A, Chang EF, Chaichana KL, McDermott MW. Surgical considerations in the management of falcotentorial meningiomas: advantages of the bilateral occipital transtentorial/transfalcine craniotomy for large tumors. Neurosurgery 2009;64:260–268; discussion 268.

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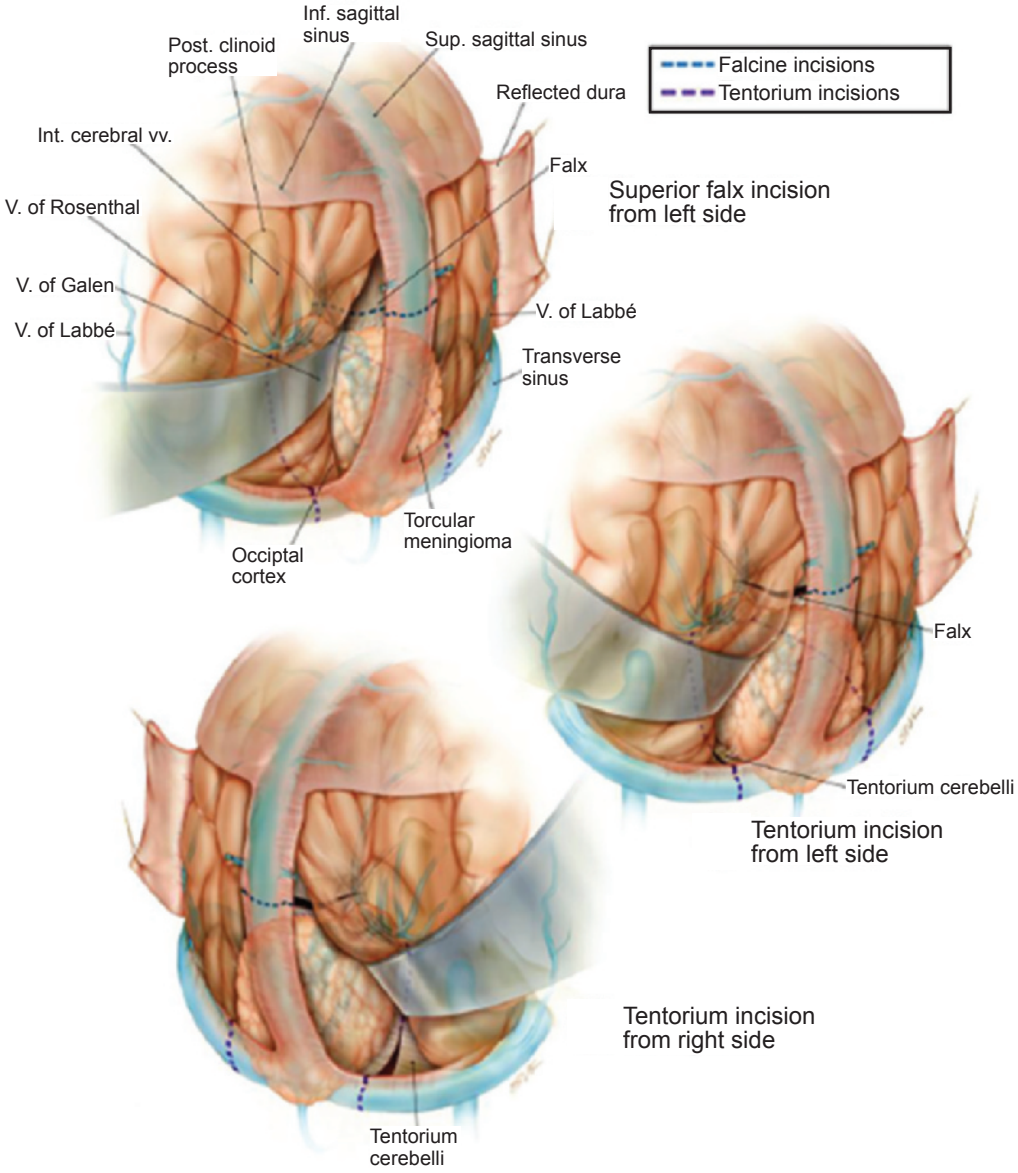


Fig. 20.4 Torcular involvement by tumor and tumor resection. Cuts are made in the falx and the tentorium to remove the arterial supply to the tumor from these structures. We recommend careful bipolar coagulation and meticulous cutting with microscissors With permission from Quinones-Hinojosa A, Chang EF, Chaichana KL, McDermott MW. Surgical considerations in the management of falcotentorial meningiomas: advantages of the bilateral occipital transtentorial/transfalcine craniotomy for large tumors. Neurosurgery 2009;64:260–268; discussion 268.

In our experience, we have found that, supratentorially, the meningioma has grown within the folds of the dura so that only the expanded smooth surfaces of the tentorium and falx are seen. We have incised the dura, and then the tumor is debulked within the leaves of the dura. The infratentorial portion of the meningioma, on the other hand, is more commonly the exophytic part of the tumor seeming to arise from the undersurface of the dural folds. It is separated from the cerebellum by an arachnoid plane and can be dissected from either a supra- or infratentorial route. When approaching the last remaining tumor near the vein of Galen and the deep venous complex, it is helpful to dissect the tumor from both routes. In cases where the straight sinus has been confirmed to be occluded by angiography, the vein of Galen can be clipped using a permanent aneurysm clip at its junction within the straight sinus in front of the falcotentorial junction, reducing the chance of tearing the vein (**Fig. 20.5**). In the unusual case of a large tumor with a patent straight sinus, a subtotal resection preserving flow is considered. We have not noted problems associated with the medial occipital veins draining into the falx but we tailor the dural opening and tumor resection to keep the larger parasagittal veins in the posterior third of the superior sagittal sinus patent (**Fig. 20.6**).

The occipital lobes can be retracted for hours intermittently as the resection proceeds; therefore, covering the pial surface with rubber dams is recommended to avoid the adherence of cottonoids or telfa. In cases of significant cerebral swelling at the conclusion of the operation, the bone flap can be left off and the patient positioned safely until the bone can be replaced. We place our patients in the semilateral position with a hollow circular foam or gel head rest to provide the appropriate cushion and to minimize the pressure that may be present in the completely supine position.

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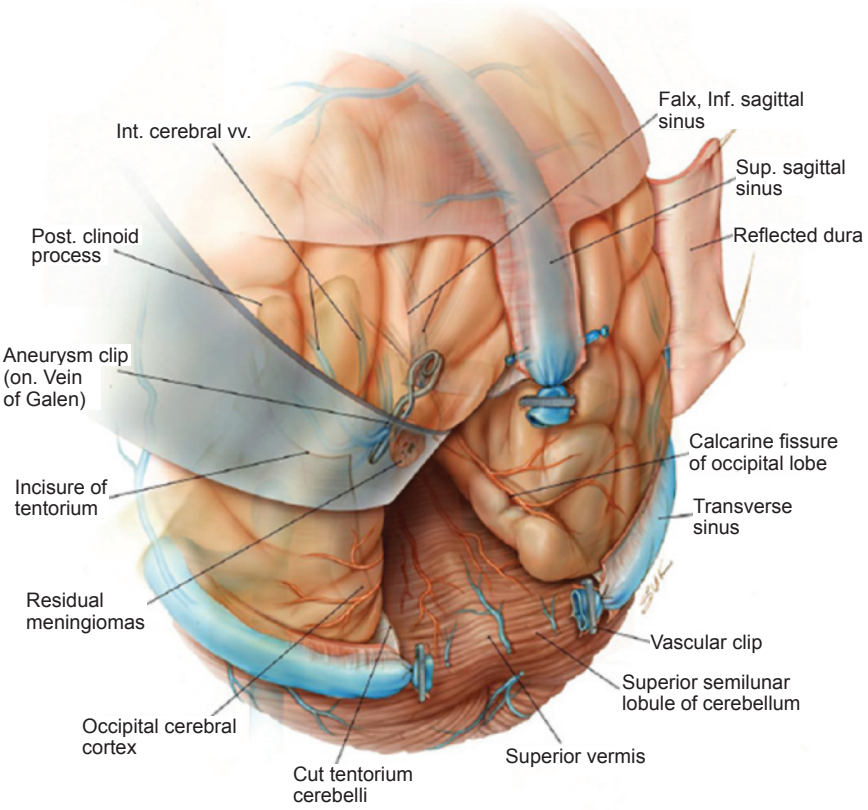


Fig. 20.5 Complete tumor removal and ligation/clipping of the major sinuses. With permission from Quinones-Hinojosa A, Chang EF, Chaichana KL, McDermott MW Surgical considerations in the management of falcotentorial meningiomas: advantages of the bilateral occipital transtentorial/transfalcine craniotomy for large tumors. Neurosurgery 2009;64:260–268; discussion 268.

Adjuvant Treatments of Recurrent and/or Residual Disease

Adjuvant treatment with focal radiation should be considered depending on age, clinical presentation, location, stage and/or size of the tumor, and diagnosis (**Fig. 20.1**). In our most recent series[19] recurrences were observed in patients who either had a subtotal resection (i.e., Simpson score > I) or had histopathology consistent with atypical or malignant meningioma. All four patients with Simpson IV resection received Gamma Knife© for the residual tumors (cases 2, 3, 4, and 7). Two patients with grade II and III tumors (cases 5 and 6), had local recurrences, which required Gamma Knife© radiosurgery despite Simpson I resections (**Table 20.1**). No patients with benign grade I histopathology and Simpson I resection had any recurrence at last follow-up.

Recovery of Function

All patients in our series demonstrated markedly improved vision by discharge, and they continued to improve over the following several months. All patients recovered to their preoperative baseline.[19] Preoperatively, two patients had visual field deficits. In case 2, a right hemianopsia resulted from a left occipital infarct during preoperative embolization. In case 7, the patient had a stable left hemianopsia after first operation at another hospital. Significant visual field impairments were observed in all patients immediately postoperatively: cortical blindness was observed in eight patients, and a unilateral hemianopsia was found in one. However, in all cases the visual impairment recovered dramatically after the first 3 days and continued to do so over the weeks and months after surgery. In one of the more remarkable cases that took a team of two surgeons 27 hours, and after which the patient had transient cortical blindness, the patient recovered full vision and currently drives without adjuvant treatment or recurrence 9 years postoperatively (**Fig. 20.7**).

Conclusion

Meningiomas arising from the falcotentorial junction can be safely managed with an understanding of the status of the patency of surrounding venous sinuses and that transient visual disturbances will occur and recover. The aid of detailed neuroimaging, preoperative angiography, a multidisciplinary team, and a wide exposure with a combined supra-/infratentorial torcular craniotomy technique is associated with acceptable risks and good outcomes. For larger tumors, a two-surgeon team is recommended for an efficient tumor removal. A common, but reversible, complication is transient postoperative cortical blindness, which should be discussed with the patients preoperatively.

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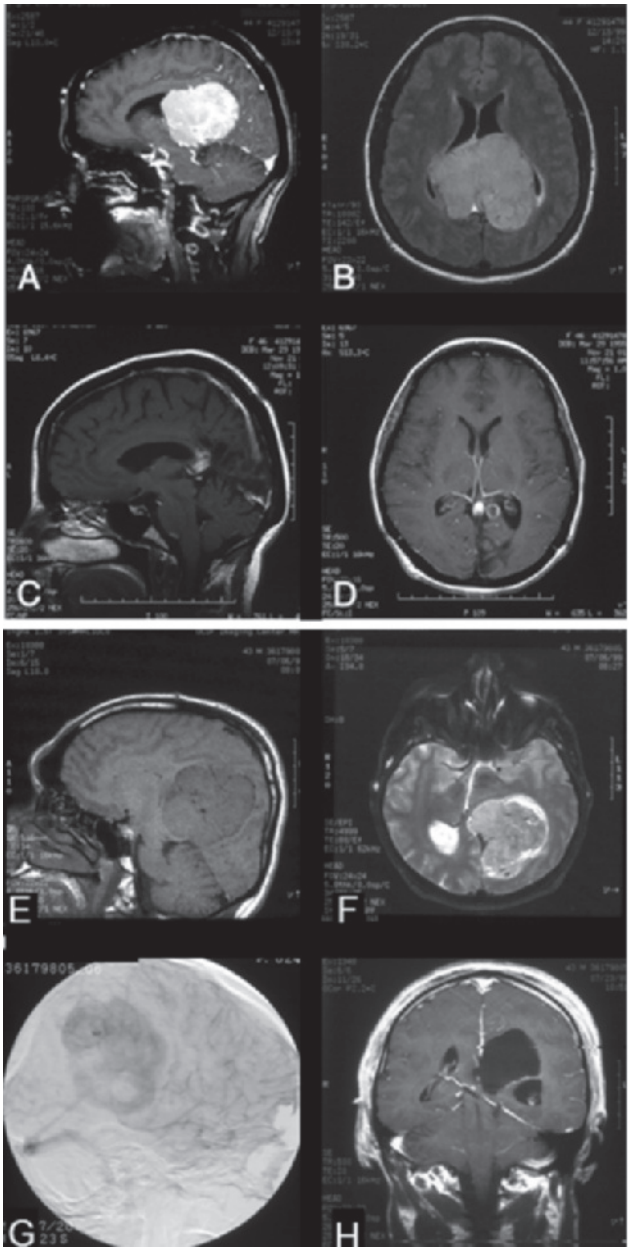


Fig. 20.6 **(A–D)** A 44-year-old woman presented with progressive cognitive impairments and personality changes over a 3-year period, with recent episodes of visual hallucinations and urinary incontinence. **(A)** Preoperative sagittal T1-weighted magnetic resonance imaging (MRI) with contrast illustrates a large homogeneous enhancement (7.0 × 4.9 × 3.8 cm) mass arising from the falcotentorial junction. **(B)** Preoperative axial T1-weighted MRI without contrast shows slight hypointensity. **(C)** Postoperative sagittal and **(D)** axial T1-weighted MRI scans with contrast show a near total resection, with small residual enhancement at the vein of Galen. This patient underwent subsequent radiotherapy for the residual tumor. **(E–H)** A 43-year-old man presented with recent onset of mild cognitive impairment, papilledema, and a right homonymous hemianopsia. **(E)** Preoperative sagittal T1-weighted MRI shows a large 6.9 × 6.4 × 5.9 cm mass, attached to the falx anteriorly and the region of the falcotentorial junction. **(F)** Preoperative axial T2-weighted MRI reveals slight hyperintensity. **(G)** Preoperative angiography (here shown after injection of the left vertebral artery) shows evidence of hypervascularity, with predominant vascular supply from medial and lateral choroidal arteries off the left posterior cerebral artery. Given that the vast majority of blood supply was via pial arteries and not from the external carotid artery, embolization was not attempted. **(H)** Postoperative coronal T1-weighted image with contrast demonstrates a gross total resection. Modified from Quinones-Hinojosa et al 2003.

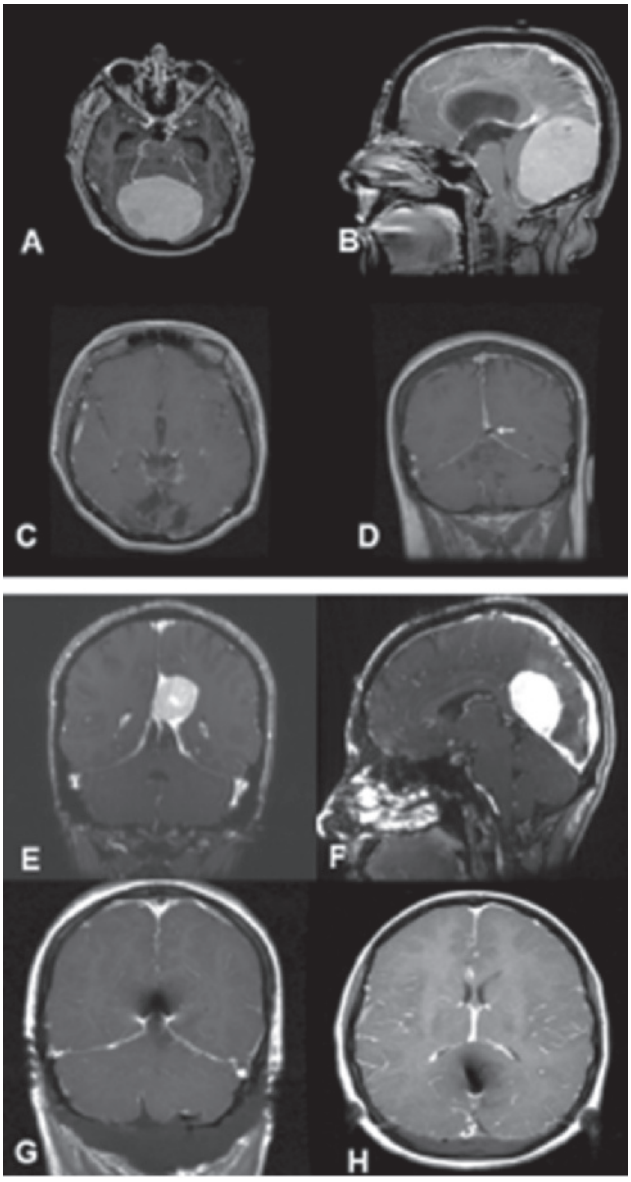


Fig. 20.7 **(A–D)** Representative magnetic resonance imaging (MRI) demonstrating a patient with a large falcotentorial meningioma. **(A)** Preoperative axial and **(B)** sagittal T1-weighted MRI with contrast, illustrating homogeneous enhancement. **(C)** Postoperative axial and **(D)** coronal T1-weighted MRI with contrast showing gross total resection (Simpson grade I). An aneurysm clip was left in place (*arrow*). **(E–H)** A 32-year-old woman presented with headaches and visual symptoms and a visual field defect. The lesion was approached via bilateral occipital transtentorial approach. The patient had cortical blindness (bilateral transient hemianopsia) for a few days but recovered her vision completely and most recent exam reveals that she has 20/20 vision with only very small, incongruous field defects left < right. She is reading, driving, and back to work full time. **(E)** Preoperative coronal and **(F)** sagittal T1-weighted MRI with contrast, illustrating homogeneous enhancement. **(G)** Postoperative coronal and **(H)** axial T1-weighted MRI with contrast showing gross total resection (Simpson grade I). Also there is an artifact from an aneurysmal clip associated with the vein of Galen venous anatomy of this falcotentorial meningioma. A–D are modified from Quinones-Hinojosa A, Chang EF, Chaichana KL, McDermott MW. Surgical considerations in the management of falcotentorial meningiomas: advantages of the bilateral occipital transtentorial/transfalcine craniotomy for large tumors. Neurosurgery 2009;64:260–268; discussion 268.

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Table 20.1 Patient Characteristics: Preoperative and Postoperative Characteristics

Case No.	Age at Surgery/ Sex	Tumor Size (cm)	Vascular Supply	Complications	Visual Exam at Discharge	Visual Fields at 1 Year
1	38/F	6.6 × 6.6 × 7.0	Meningohypophyseal trunks (bilateral), medial and lateral choroidal arteries	None	OS—finger counting OD—finger counting Mild diplopia	normal
2	44/F	7.0 × 4.9 × 3.8	Left posterior cerebral artery (P2/P3 segments)	None	OS—20/800 OD—20/800	R hemianopsia (same as preop)
3	42/F	5.5 × 4.0 × 2.0	Not able to be visualized	None	OS—20/200 OD—20/400	Normal
4	40/F	1.9 × 1.7 × 2.3	Left meningohypophyseal trunk	None	OS—20/20 OD—20/20	Normal
5	48/M	3.3 × 3.6 × 4.8	Right internal carotid artery (inferolateral trunk), branch of left and right occipital arteries, left middle meningeal artery	None	OS—20/30 OD—20/30	Normal
6	43/M	6.9 × 6.4 × 5.9	Pial branches from left anterior choroidal artery, distal parietal occipital artery, lateral posterior choroidal artery	None	OS—20/20 OD—20/20	Normal
7	73/M	3.4 × 3.0 × 2.4	No preoperative angiography	Cerebrospinal fluid leak	OS—hand movement OD—hand movement	L hemianopsia (same as preop)
8	47/F	7.3 × 6.2 × 6.4	Right occipital artery, bilateral meningeal arteries, left posterior cerebral artery	None	OS—20/40 OD—20/40	Normal
9	33/F	4.0 × 3.1 × 3.1	Bilateral superior cerebellar artery, left occipital artery, left collateral anterior cerebral artery	None	OS—finger counting OD—finger counting	Normal

Abbreviations: OD, oculus dexter; OS, oculus sinister.
Modified from Quinones-Hinojosa A, Chang EF, Chaichana KL, McDermott MW: Surgical considerations in the management of falcotentorial meningiomas: advantages of the bilateral occipital transtentorial/transfalcine craniotomy for large tumors. Neurosurgery 2009;64:260–268.

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Chapter 21

Olfactory Groove Meningiomas

Stephen J. Hentschel and Franco DeMonte

Introduction

Olfactory groove meningiomas (OGMs) account for ~9 to 18% of all meningiomas.[1–4] These tumors are typically World Health Organization (WHO) grade I meningiomas, with the meningothelial and fibrous varieties being particularly common.[4–7] Although Cushing may have been the first to describe his experience with OGMs in great detail, it is probable that Durante published the first case of an OGM resection in 1885.[8]

Anatomy

Meningiomas of the olfactory groove arise from the region of the frontosphenoid suture and may involve any part of the area from the crista galli to the planum sphenoidale (**Fig. 21.1**).[2,9,10] Anterior, middle, and posterior OGMs have been described, but subclassification into these subtypes is rarely useful, and the site of origin may be uncertain when they are large and extend back to the pituitary fossa (**Fig. 21.2**). Although this area of origin can be quite close to that of tuberculum sellae meningiomas (TSMs), the behavior of these two lesions is very different. The differentiation between the two is made by the location of the optic chiasm; OGMs depress the chiasm inferiorly, whereas TSMs elevate the chiasm (**Table 21.1**). Although generally symmetric due to their midline origin, OGMs may grow to one side and cause predominantly unilateral symptoms.

These tumors commonly cause hyperostosis of the floor of the anterior cranial fossa. They may also erode the bone and grow into the ethmoid sinus in ~15 to 20% of cases (**Fig. 21.3**).[4,11–14] The olfactory nerves are rarely found to be completely encased by tumor and can usually be identified displaced laterally by the tumor over the orbital roofs.

The anterior cerebral arteries (ACAs) become separated around the tumor as far as the anterior communicating complex will allow as the tumor enlarges.[2] The ACAs are thus found posterosuperior and lateral to OGMs. The medial orbitofrontal and frontopolar branches may become incorporated into the tumor capsule (**Fig. 21.4**). The tumor itself receives its blood supply from the anterior and posterior ethmoidal arteries as well as from sphenoidal branches of the middle meningeal artery. Occasionally a pial supply from the anterior cerebral and anterior communicating arteries will be present when the tumors are quite large.

Clinical Features

Due to their slow growth and subfrontal location, OGMs present insidiously (**Table 21.2**). One of the most common symptoms is that of a change in personality, judgment, or motivation noted by family members or close contacts, but not usually by the patient. These lesions may grow to a very large size before diagnosis, with headaches and visual disturbances occurring only late in the course of the disease. Patients rarely complain of altered smell or taste, despite the olfactory tracts becoming distorted early. Although the Foster-Kennedy syndrome of unilateral optic atrophy and contralateral papilledema was initially described in a midline frontal meningioma and in 24% of Cushing's cases, it is only rarely seen in modern series.[2]

Because of the growth pattern of OGMs and their effect on the regional anatomy, these tumors may extend posteriorly and inferiorly to compress the optic nerves and cause an inferior visual field defect, but this may be difficult to detect in patients with large tumors who are apathetic and who may have papilledema. In contrast, the more posteriorly situated TSMs produce a bitemporal visual field defect due to elevation and splaying of the optic chiasm.[2]

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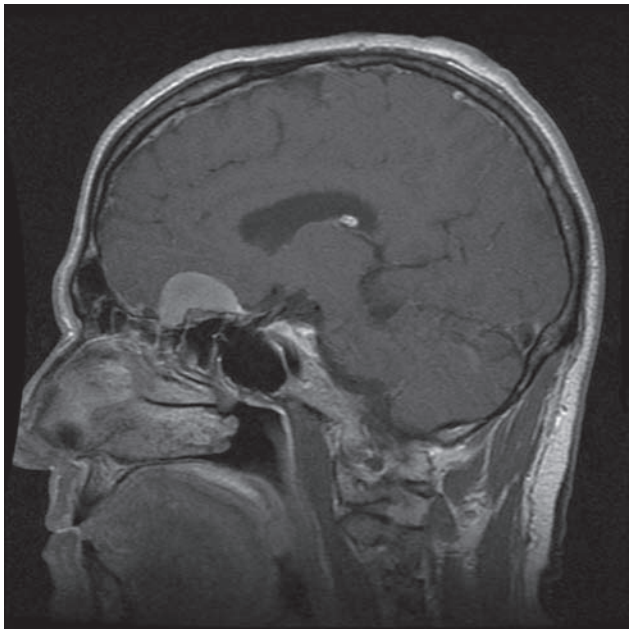


Fig. 21.1 Sagittal postcontrast T1-weighted magnetic resonance images of a small olfactory groove meningioma. Property of the Division of Neurosurgery, University of British Columbia. Used with permission.

Table 21.1 A Comparison of Olfactory Groove and Tuberculum Sellae Meningiomas

	Olfactory Groove Meningioma	Tuberculum Sellae Meningioma
Site of origin	Frontosphenoid suture	Tuberculum sella, limbus sphenoidale, chiasmatic sulcus, diaphragma
Main blood supply	Anterior and postethmoidal	Postethmoidal
Olfactory nerves	Lateral	Inferior
Optic apparatus	Inferolateral	Superolateral
Visual field deficit	Inferior	Bitemporal
Anterior cerebral arteries	Posterosuperior	Posterosuperior

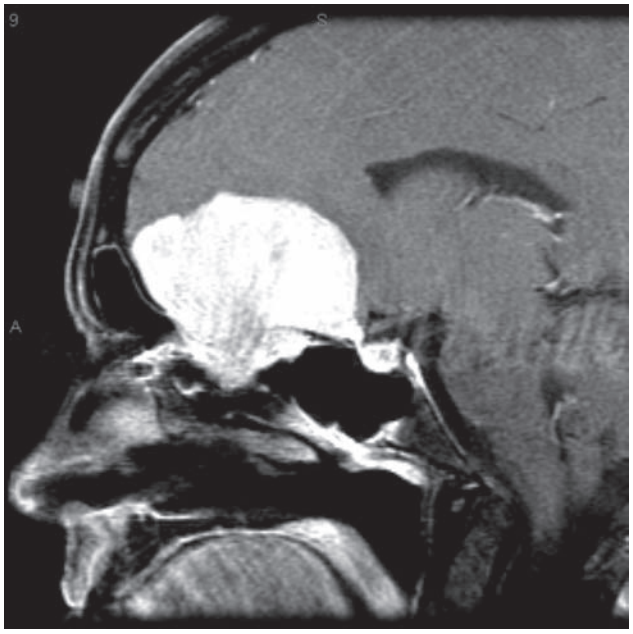


Fig. 21.2 Sagittal postcontrast T1-weighted magnetic resonance images of a large olfactory groove meningioma extending from the back wall of the frontal sinus to the sella. Note the ethmoid sinus extension. Property of the Division of Neurosurgery, University of British Columbia. Used with permission.

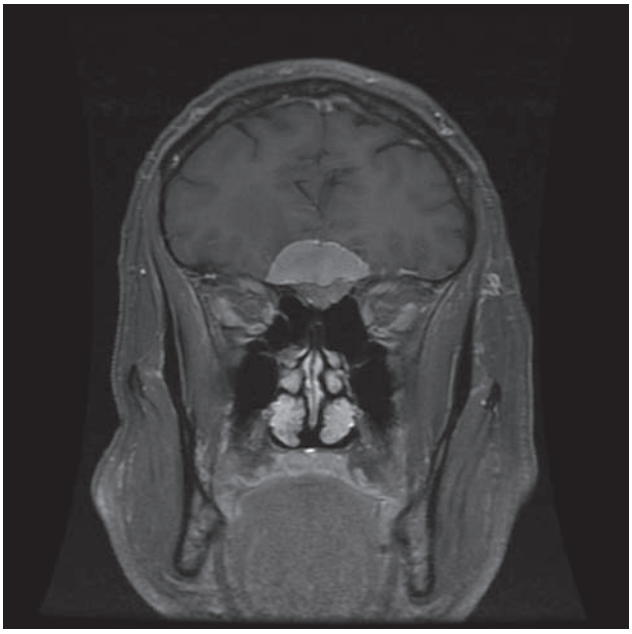


Fig. 21.3 Coronal postcontrast T1-weighted magnetic resonance imaging identifies tumor extension into the ethmoidal sinuses. Property of the Division of Neurosurgery, University of British Columbia. Used with permission.

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Table 21.2 Clinical Features of Olfactory Groove Meningiomas Reported in the Literature

Series and First Author	Year	No.	Mean Age (range)	Sex M:F	H/A (%)	Mental Change (%)	Visual Loss (%)	Hyposmia/ Anosmia (%)
Cushing[2]	1938	29	43 (29–62)	NA	12 (41)	6 (21)	11 (38)	3 (10)
Solero[4]	1983	98	(18–72)	36:62	47 (48)	33 (34)	53 (54)	39 (40)
Bakay[9]	1984	36	55 (NA)	15:29	18 (50)	16 (44)	17 (47)	26 72)
Mayfrank[30]	1996	18	NA (45–75)	5:13	NA	10 (56)	4 (22)	11 (61)
Tsikoudas[7]	1999	13	NA (34–74)	3:10	8 (62)	8 (62)	4 (31)	7 (54)
Turazzi[20]	1999	37	57 (32–64)	15:22	NA	27 (73)	16 (43)	27 (73)
Spektor[14]	2005	80	55 (16–85)	21:59	41 (51)	21 (26)	22 (28)	39 (49)
Ojemann[25]	2006	17	NA (24–73)	5:14	3 (18)	9 (53)	7 (41)	1 (6)
Bassiouni[5]	2007	56	51 30–74	15:41	11 (20)	22 (39)	9 (16)	8 (14)
Nakamura[28]	2007	82	58 (33–91)	19:63	26 (32)	59 (72)	20 (24)	48 (59)
Gazzeri[6]	2008	36	56 (28–78)	12:24	18 (50)	25 (69)	20 (56)	28 (78)
Romani[29]	2009	66	57 (38–85)	31:35	11 (17)	33 (50)	22 (33)	38 (58)
Aguiar[42]	2009	21	50 (21–76)	6:15	12 (57)	4 (19)	4 (19)	21 (100)

Abbreviations: H/A, headache. NA, not available.

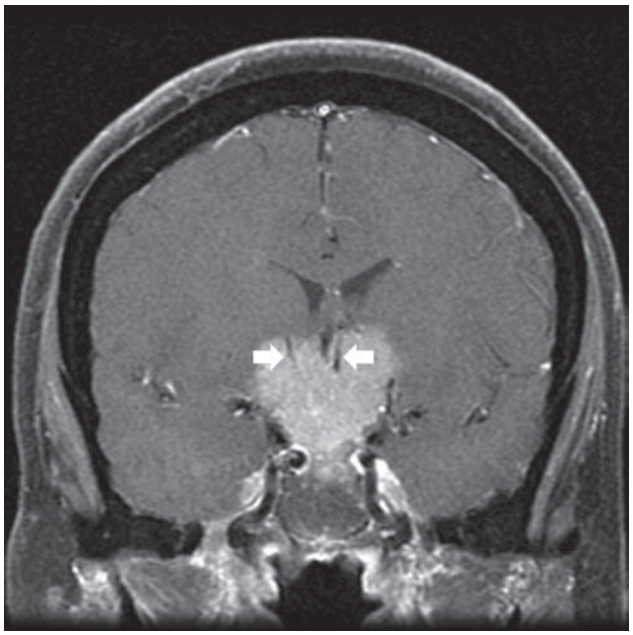


Fig. 21.4 Coronal postcontrast T1-weighted magnetic resonance imaging identifies encasement of the A2 segments of the anterior cerebral arteries (*arrows*). Property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center. Used with permission.

Imaging Features

Computed tomography (CT) and magnetic resonance imaging (MRI) have replaced all other imaging modalities in the diagnosis and evaluation of OGMs. CT, particularly with coronal views and bone algorithms, is useful to determine the extent of hyperostosis (**Fig. 21.5**). MRI is the modality of choice for definition of the pathological anatomy and allows determination of paranasal sinus involvement by tumor, the location of the optic chiasm and usually the cerebral vessels, and any tumor extending posteriorly to involve the sella (**Fig. 21.6**). MRI with T2 sequences demonstrates the amount of frontal lobe edema, which may correlate with the extent of pial blood supply.[15] T2-weighted images are also best for identifying the vascular flow voids of blood vessels in proximity to the tumor. Noninvasive computed tomographic and magnetic resonance angiography (CTA, MRA) can be useful to confirm the vascular relationships, but generally do not provide any new information. Conventional catheter angiography is rarely required because the major feeding vessels can be surgically divided early in the operative procedure, and embolization is rarely required or technically possible.[16]

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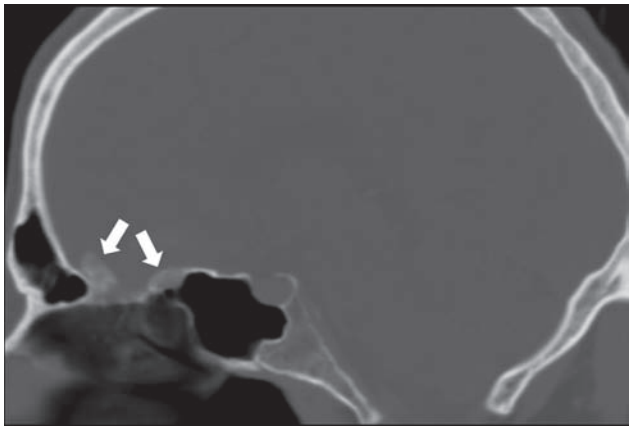


Fig. 21.5 Sagittal CT at bone window settings identifies hyperostosis of the anterior skull base (*arrows*). Property of the Division of Neurosurgery, University of British Columbia. Used with permission.

Management

Incidentally identified, asymptomatic tumors less than 2.5 cm can almost always be managed, at least initially, by serial clinical, neuropsychological, ophthalmological, and magnetic resonance (MR) observation. Should evidence of tumor growth or clinical, neuropsychological, or ophthalmological deterioration become evident, then surgical excision is the management of choice for those medically fit. Primary radiation should be reserved for patient refusal of surgery or for medically unfit patients.

Surgical Considerations

Approaches

Many different surgical approaches have been described for the resection of OGMs. The common approaches include pterional[17–20] and subfrontal approaches,[15,16,21–25] but endoscopic,[26,27] lateral-orbital,[26,33] and interhemispheric[22] approaches have also been described. A comparison of the advantages and disadvantages of the two classical approaches to OGMs is shown in **Table 21.3**. We favor the bifrontal approach for larger tumors because of the excellent access to the anterior skull base, the opportunity to devascularize the tumor early in the procedure, and the ability to create a robust vascularized reconstruction. Whatever the approach, certain key maneuvers must be possible (**Table 21.4**). Early interruption of the blood supply is critical in minimizing blood loss during tumor resection. The frontal lobes should be readily dissected free of the tumor without the need for frontal lobe resection. Excellent visualization of the optic apparatus, cerebral vessels, and floor of the anterior cranial fossa is required to permit preservation of critical structures and repair of surgical defects. Access to the anterior skull base is critical in those tumors causing hyperostosis and particularly those invading the ethmoid sinuses; this access may be lacking in the frontolateral-type of approaches.[29]

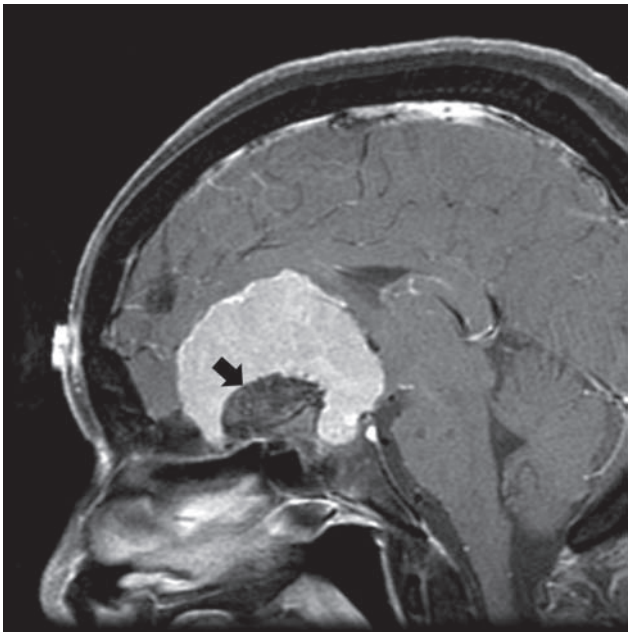


Fig. 21.6 Sagittal postcontrast T1-weighted magnetic resonance imaging reveals a large tumor extending back into the sella turcica. Extensive hyperostosis is present (*arrow*). Property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center. Used with permission.

The goals of surgery are to completely resect the tumor, including involved bone and any tumor extending into the ethmoid sinus, with preservation of neurological function. For tumors less than 3 cm we prefer a unilateral subfrontal approach with orbital osteotomy (lateral supraorbital); for larger tumors we utilize a bifrontal craniotomy with bilateral orbital osteotomies. This latter approach will be described here in detail.[16]

Technique

The patient is positioned supine with the head midline but slightly extended to bring the brows to the highest point. A bicoronal skin incision is then made extending from zygomatic root to zygomatic root. Dissection continues in the immediate subgaleal plane so as to preserve a thick and well-vascularized pericranial flap. Once the superficial fat pad over the temporalis muscle is identified, the fascia is incised sharply and a subfascial dissection of the temporalis muscle performed as the scalp flap is turned anteriorly.[31] We favor a subfascial dissection because it is easier and provides more tissue coverage over the facial nerve branches than does the interfascial dissection. In this fashion, the zygomatic arch can be exposed while protecting the facial nerve. The scalp flap is also undermined posteriorly so a large flap of pericranium will be available for reconstruction at the end of the procedure. The pericranium is then elevated separately from the skin flap, and the supraorbital neurovascular bundles are freed from their foramen using a fine drill bit or a quarter-inch osteotome (**Fig. 21.7**).

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Table 21.3 Advantages and Disadvantages of the Pterional and Subfrontal (with orbital osteotomies) Approaches to Olfactory Groove Meningiomas

Approach	Advantage	Disadvantage
Pterional	Optic apparatus and ACA seen early Early cistern opening drains cerebrospinal fluid Avoids frontal sinus entry Shorter operative time Shorter distance to ipsilateral tumor	Narrow working space Upper portion of tumor may be hidden Greater distance to contralateral tumor Orbital roof height obscures base
Subfrontal	Wide frontal exposure Direct access to cranial base Early division of main blood supply Easier to repair cranial base	Frontal sinus opened SSS divided ACA and optic apparatus seen late

Abbreviations: ACA, anterior cerebral arteries; SSS, superior sagittal sinus.

Table 21.4 Key Procedural Steps in the Resection of Olfactory Groove Meningiomas

Key Maneuver
Early interruption of blood supply
Visualization of origin along cranial base
Dissection, not resection, of frontal lobes
Tumor debulking and continued devascularization
Identification and preservation of optic apparatus, olfactory nerve, cerebral vessels
Resection of involved bone and dura in the cranial base
Repair of surgical defects



Fig. 21.7 Subperiosteal dissection elevates the pericranial flap from the calvarium. The supraorbital neurovascular bundle is preserved (*arrow*). Property of the Division of Neurosurgery, University of British Columbia. Used with permission.

Burr holes are placed at both keyholes with exposure of the superolateral periorbital membrane as well as the frontal dura. An additional burr hole is placed at the posterior aspect of the craniotomy straddling the superior sagittal sinus. The bifrontal craniotomy is then elevated and the frontal sinus demucosalized and cranialized. Bilateral orbital osteotomies are performed with cuts at both frontozygomatic sutures and in the midline at the frontonasal suture (**Fig. 21.8**). A smaller craniotomy with more limited medial orbital osteotomies is an option for smaller tumors (**Fig. 21.9**).

The crista galli is identified and resected. A subperiosteal dissection is performed within the orbits, and the periorbita is dissected off the bone. The anterior and posterior ethmoidal vessels are coagulated and divided within the orbits, thereby devascularizing the tumor (**Fig. 21.8**).[32] The dura is then opened over both frontal poles and the superior sagittal sinus is suture-ligated and divided along with the falx just above the crista galli. We have encountered no adverse consequences associated with sinus ligation at this very anterior point.

Small feeders along the skull base continue to supply the tumor; these can be divided as the dissection proceeds along the cranial base from anterior to posterior. Large tumors have usually displaced the frontal lobes superiorly and posteriorly and brain retraction should be avoided as much as possible to prevent postoperative cerebral edema, which has been reported to be associated with increased morbidity and mortality in some series.[28] Internal tumor debulking with the ultrasonic aspirator alternates with dissection of the tumor off of the frontal lobes. The olfactory nerves should be identified laterally to the tumor and can anatomically be preserved in a surprising number of cases, if only unilaterally (**Fig. 21.10**). As the tumor is debulked further, its superior, posterior, and inferior margins can be dissected in sequence with identification of the ACA and optic apparatus. The posterior arachnoidal membranes are intact in nearly all cases of primary operation and permit a relatively straightforward dissection from the optic nerves, pituitary stalk, and blood vessels of the Circle of Willis. Feeding vessels from the anterior communicating artery complex and from the A2 segments of the ACA can be divided, but care must be taken not to mistake these vessels for perforating arteries that supply the optic chiasm and hypothalamus. The anterior cerebral vessels can become incorporated into very large tumors, and careful sharp dissection is required to free them from the tumor. If tumor is adherent to perforating or other vessels that cannot be divided, it is best to leave a small amount of tumor behind rather than risk vascular injury and a possible neurological deficit. This small amount of residual tumor can be followed with serial imaging or treated with stereotactic radiosurgery depending upon the clinical circumstances.

After the tumor has been resected, attention should be turned toward the floor of the anterior cranial fossa. Any hyperostotic bone should be removed by drill curettage and the dura of origin removed to reduce the risk of recurrence.[24,33–35] Tumor extension into the ethmoid sinuses, nasal cavity, or orbits should be completely resected. The

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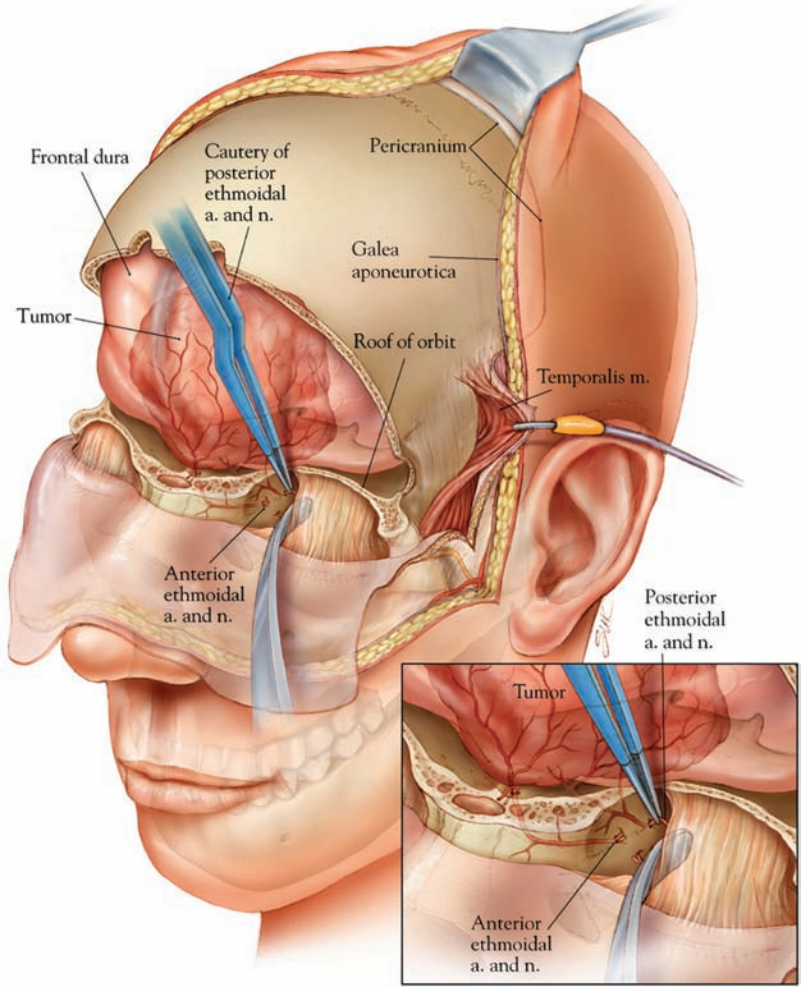


Fig. 21.8 Artist's illustration of the surgical approach to a large olfactory groove meningioma (OGM) and the technique of transorbital occlusion of the ethmoidal arteries to devascularize OGMs. A bifrontal craniotomy with biorbital osteotomies has been performed. The anterior and posterior ethmoidal arteries are divided bilaterally. Property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center. Used with permission.

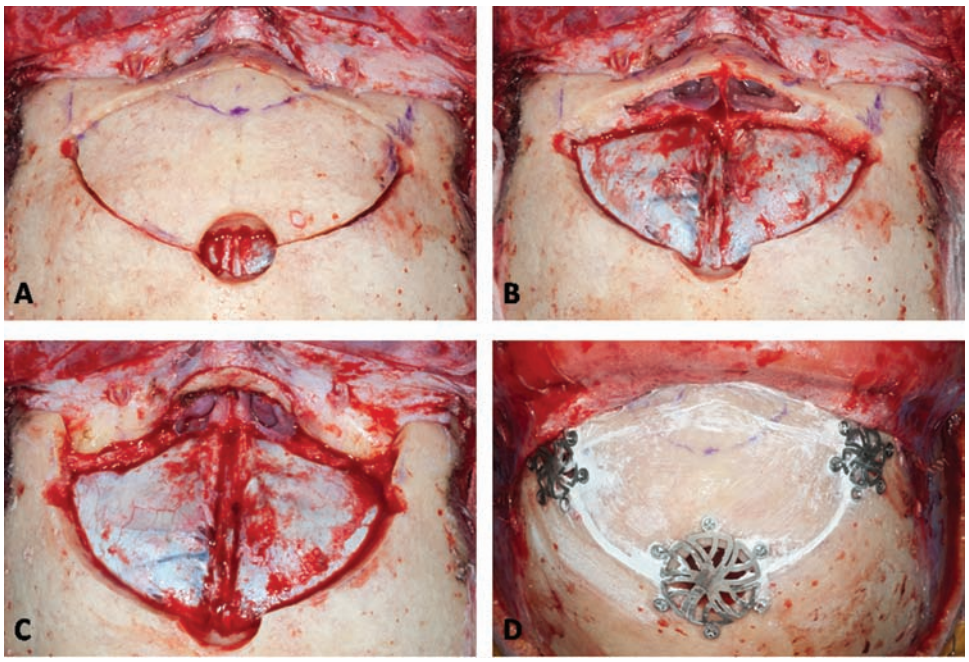


Fig. 21.9 Intraoperative photographs of a limited orbitocranial approach to a 3.5 cm meningioma. **(A)** The midline entry hole straddling the superior sagittal sinus and the bilateral osteotomies to the floor of the anterior fossa are illustrated. **(B)** The bone flap has been removed following osteotomies of the anterior table of the frontal sinus. **(C)** Biorbital osteotomies have been performed. **(D)** Reconstruction with the pericranial graft rotated beneath the rigidly fixated orbital and cranial osteotomies. Bone cement is used to fill in the gaps created by the osteotome. Property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center. Used with permission.

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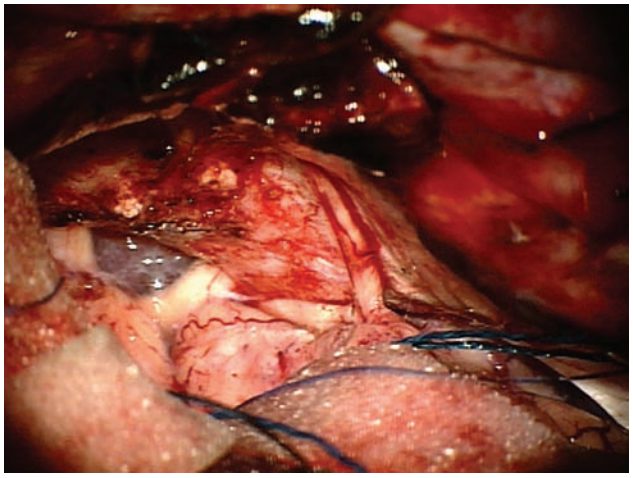


Fig. 21.10 Intraoperative view following resection of a 3.5 cm olfactory groove meningioma. The optic nerves are undisturbed and the right olfactory tract has been preserved anatomically. Property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center. Used with permission.

dura is then closed primarily or with the aid of a dural substitute. The large piece of vascularized pericranium that was preserved in the initial portion of the procedure is then brought down over the floor of the anterior cranial fossa and secured in place using holes drilled in the planum sphenoidale. The orbital osteotomy and bifrontal bone flap are then plated back into place without compressing the pericranial flap to avoid graft swelling or infarction.[36]

Reconstruction

For recurrent tumors where the pericranium has been previously utilized or disrupted, the galea can be harvested as a vascularized pedicled graft. Other methods of repair include use of fascia lata, dural substitutes, autologous fat, or fibrin glue. Rarely, in a multiply operated patient with no viable local options for reconstruction, free tissue transfer may be required.

Outcome

Oncological

Despite the large size that these lesions may attain, OGMs are able to be completely resected in a high percentage of cases[19,20,24,30,37] (**Table 21.5**). In our experience, nearly all nonrecurrent tumors can be completely resected, including their dural attachment and all involved bone (**Fig. 21.11**). Two patients in the M. D. Anderson series of 18 OGMs underwent incomplete resections: one with a recurrent tumor adherent to the ACA, and another with a very small fragment of adherent tumor on the olfactory nerve, which was left in an unsuccessful attempt to preserve olfaction. Both of these patients had stable minimal residual disease over 2.5 and 4.6 years of follow-up, respectively.

Cognitive

The majority of patients with preoperative cognitive impairment improve following surgical removal of OGMs. In one of the few studies to systematically examine cognitive improvement postoperatively, Gazzeri et al found moderate or severe cognitive impairment utilizing the Mini-Mental State Examination (MMSE) in 22 of 36 patients preoperatively. Postoperatively, only eight patients were in the moderate category (MMSE 10 through 19) and none were in the severe category (MMSE 1 through 9), with most of the improvements seen in the first month following surgery (35 bifrontal approaches).[6] Other authors have reported cognitive improvement in 50 to 90% of patients.[5,9] Bassiouni et al correlated the cognitive recovery in their patients with improvement of frontal lobe edema on follow-up MRI.[5] In our series, only one patient with mild preoperative cognitive impairment failed to improve postoperatively (**Table 21.5**).

Visual/Olfactory

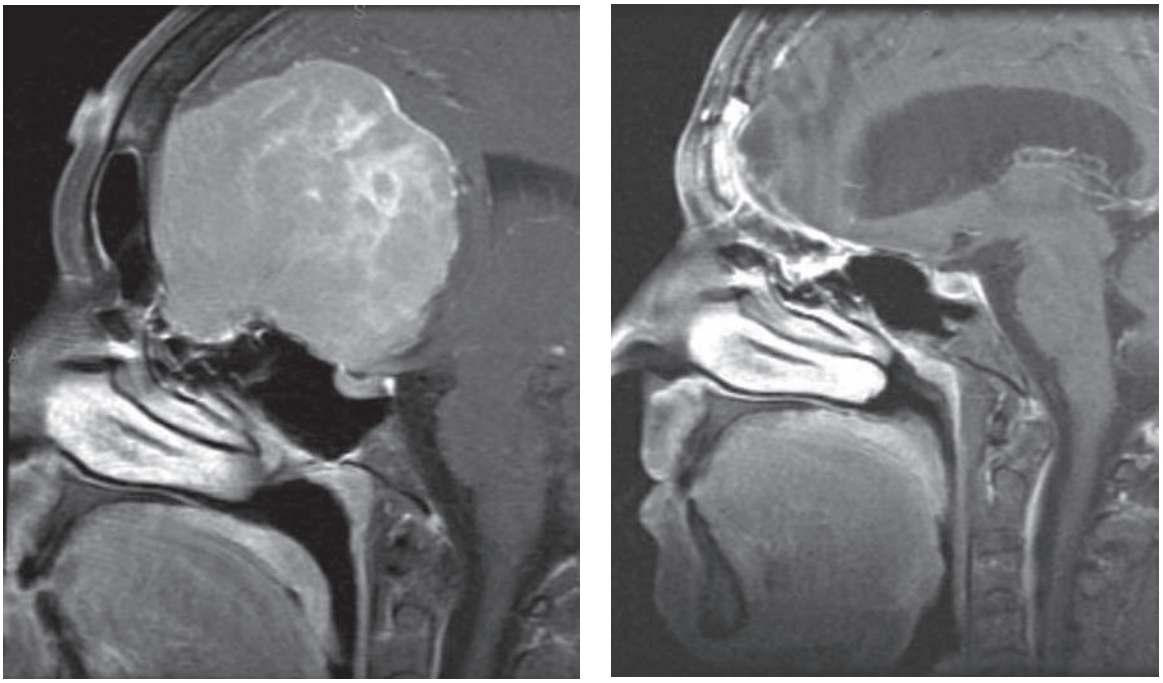
Visual acuity has been shown to improve in 26 to 83% of patients, whereas deficits of visual field have improved in 29 to 100%.[5,6] Five of our six patients with preoperative visual dysfunction improved significantly following resection of the tumor (**Fig. 21.12**). The sixth patient's visual function remained stable.

Preservation of olfaction is disappointing despite anatomical preservation of at least one olfactory tract. Welge-Luessen et al reported preservation of at least one olfactory tract in two (of 12) OGM cases.[38] With rigorous olfactory testing these authors found no evidence of olfactory function on that side in either patient. Bassiouni found that all patients with preoperative anosmia remained so postoperatively despite anatomical preservation of at least one olfactory tract in four patients. In addition, those authors found 15 (37%) patients with worsened olfaction postoperatively, the majority of whom underwent bifrontal resections.[5] It is more likely for olfaction to be preserved in small- to medium-sized tumors that are more to one side. Anatomical preservation of the olfactory tracts may not be enough to preserve olfaction, as ischemia secondary to bipolar coagulation may play a significant role as well. Excessive frontal lobe retraction and anterior skull base resections are also potential factors affecting olfactory outcome. In our series, no patient with preoperative anosmia regained olfactory function and two patients with some olfaction preoperatively lost their sense of smell, despite anatomical preservation of the olfactory tracts in both cases.

Complications

In the era of modern microsurgery, the surgical mortality associated with the resection of olfactory groove meningiomas has ranged from 0 to 17%, with most recent reports listing no deaths.[14,23,24,30] The complications of cerebrospinal fluid (CSF) leak, meningitis, hematoma, seizure, and visual worsening

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A B

Fig. 21.11 **(A)** Preoperative and **(B)** postoperative sagittal postcontrast T1-weighted magnetic resonance images depict complete resection of a huge olfactory groove meningioma. Property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center. Used with permission.

Table 21.5 M. D. Anderson Series of Olfactory Groove Meningiomas 1996–2009

	Preop	Postop
Number	18	
Size (mean) Range	5 cm 1.9–8 cm	
Female:male	14:4	
De novo:recurrent	17:1	
Symptoms:		
Mental status change	10 (56%)	1 (nine improved, one unchanged)
Visual dysfunction	6 (33%)	1 (five improved, one unchanged)
Anosmia	11 (61%)	13 (two new)
Gross total resection:subtotal resection	16:2	
Follow-up (median) range		4.0 years 0.3–12 years
Recurrences		0

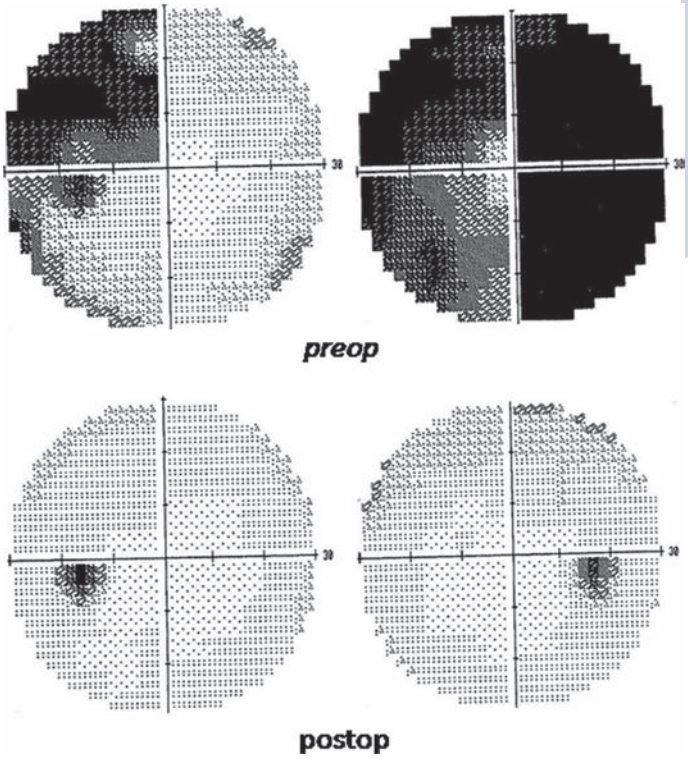


Fig. 21.12 Preoperative (top) and postoperative visual fields in a patient whose magnetic resonance imaging scan is shown in **Fig. 21. 6**. A gratifying recovery of field has occurred. Property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center. Used with permission.

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are the most common procedure-specific complications, but occur infrequently in most series (**Table 21.6**). Some authors have found the bifrontal approach to be associated with more serious complications than other approaches, leading to their use of laterally based approaches.[28,29] We could find only one study reporting significant cerebral edema following bifrontal craniotomy. The authors recommended avoidance of the bifrontal approach and of sectioning of the superior sagittal sinus.[28] We had no such complications in our series of patients. Gazzeri found no increased cognitive deficits postoperatively in 35 bifrontal approaches for OGMs; in fact, many patients had improvement in their cognitive function.[6]

Recurrence

The recurrence rates for OGMs of 30% at 5 years and 41% at 10 years are often quoted in the literature.[25] This is probably due to the reluctance of some surgeons to aggressively resect involved tissues in the cranial base for fear of increased complications and difficulty in repair.[11,13,39] Al-Mefty and others have stated that failure to resect all involved tissues, including the bone and dura of the anterior cranial base, is the most significant factor that determines recurrence.[10–12,14,24,39,41] We also advocate resection of the dural attachment, removal of hyperostotic tumor-involved bone, and resection of any tumor entering the sinonasal and orbital cavities.[16] Utilizing such an approach, recurrence rates less than 10% can be achieved.[5,6,28,29] In our series of 18 OGMs, no tumor recurrence was identified after a median follow-up of 4.0 years (range 1 to 12 years).

In their series of 15 patients with OGMs, including six recurrent tumors, Obeid and Al-Mefty noted a median of 8 years for tumor recurrence, with the most common site of recurrence being along the cranial base.[24] In that series, which also included nine de novo cases, an increased incidence of postoperative complications was not identified, even in the group of recurrent tumors, when compared with previous series reported in the literature. Aggressive and complete resection was performed in all cases.

Conclusion

Patient outcomes following resection of these frequently large tumors are excellent, even when symptoms have been prolonged. Microsurgical techniques and attention to detail, rather than a specific approach, minimize morbidity and maximize the completeness of resection. Removal of hyperostotic tumor-involved bone and dura at the cranial base minimizes the risk of recurrence. A robust vascularized reconstruction minimizes the risks of cerebrospinal fluid leakage.

Table 21.6 Complications of Surgery for Olfactory Groove Meningiomas

Series and First Author	Approach	*n*	Visual Loss (%)	Cerebrospinal Fluid Leak (%)	Infection (%)	Seizure (%)	Hematoma (%)	Mortality (%)
Hassler[17]	Pt	11	0	0	0	0	0	1 (9)
Solero[4]		98	0	2 (2)	3 (3)	6 (6)	1 (1)	17 (17)
Mayfrank[30]	IH	15	0	0	1 (7)	0	1 (7)	0
Paterniti[19]	Pt	20	0	0	0	0	0	2 (10)
Tsikoudas[7]	BF	13	1 (8)	3 (23)	1 (8)	1 (8)		2 (15)
Turazzi[20]	Pt	37	0	0	0	0	0	1 (3)
El Gindi[23]	BF	25	3 (12)	1 (4)	1 (4)	12 (48)	2 (8)	0
Obeid[24]	BF/SF	15	1 (7)	3 (20)	1 (7)	1 (7)	0	0
Spektor[14]	BF/Pt/SF	80	0	10 (13)	4 (5)	3 (4)	4 (5)	0
Bassiouni[5]	BF/Pt	56	1 (2)	3 (5)	0	1 (2)	1 (2)	3 (5)
Nakamura[28]	FL BF	34 46	0 1 (2)	2 (6) 2 (4)	1 (3) 0	4 (12) 2 (4)	1 (3) 5 (11)	0 4 (9)
Gazzeri[6]	BF	36	1 (3)	2 (6)	0	1 (3)	0	1 (3)
Romani[29]	LSO	66	5 (8)	6 (9)	4 (6)	0	1 (2)	0
Aguiar[42]	Pt/BF	21	0	4 (19)	1 (5)	0	1 (5)	1 (5)
MDACC	BFO/LSO	18	0	0	0	0	0	0
Total		**591**	**13 (2)**	**38 (6)**	**17 (3)**	**31 (5)**	**17 (3)**	**32 (5)**

Abbreviations: BF, bifrontal; BFO, bifrontoorbital; FL, frontolateral; IH, interhemispheric; LSO, lateral supraorbital; Pt, pterional; SF, subfrontal; MDACC, M.D. Anderson Cancer Center.

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Chapter 22

Tuberculum Sellae Meningiomas

Michael E. Sughrue, Nader Sanai, and Michael W. McDermott

Introduction

Although meningiomas represent the most common primary tumor histology after the age of 35 in adults, most of them grow slowly and take many years to produce clinical symptoms.[1,2] Tuberculum sellae meningiomas (TSMs) represent a small percentage of surgical case series and arise from the region of the chiasmatic sulcus and tuberculum, usually with a point of origin at the junction of the optic canal and lateral aspect of the chiasmatic sulcus.

Decision making is usually rather straightforward for TSMs. Patients generally present with symptoms related to compression of the optic apparatus. As a result, observation is usually an undesirable option, and tumoristatic therapies, such as stereotactic radiosurgery, three-dimensional (3-D)-conformal radiotherapy, and intensity-modulated radiotherapy, do not decompress the optic apparatus and are similarly unsuitable in most situations.[2–4] Thus, the current thinking regarding the management of TSMs generally focuses on which surgical approach best achieves optic decompression with the best morbidity and success profile.

In this chapter, we review the clinical presentation of TSMs and discuss the available surgical approaches utilized in contemporary practice. We provide our insights and experiences regarding the bifrontal–extended frontal approach to TSMs, which is our approach of choice. We review existing modern series regarding surgical outcomes for TSM.

Clinical Features

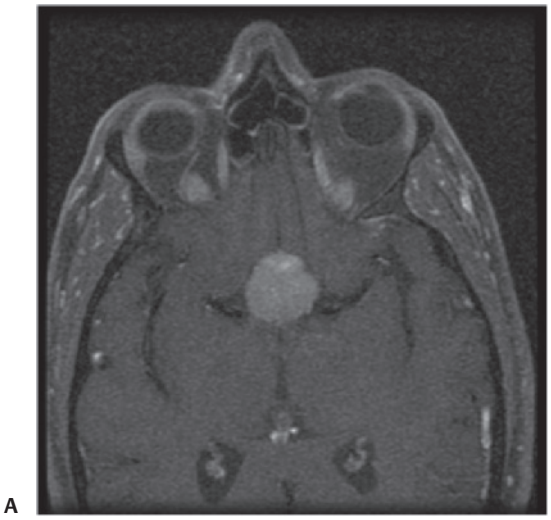
The intimate relationship between TSMs and the optic apparatus characterizes the clinical picture, surgical decision making, and risk–benefit profile of patients with TSM. Most patients with TSM present with visual loss.[5–7] This may be unilateral or more commonly bilateral with a chiasmatic syndrome (bitemporal visual field loss). The optic nerves are usually displaced laterally and superiorly and the optic chiasm superiorly or posteriorly. Frequently there is extension down the medial aspect of one or both optic canals, best seen on fat-suppressed coronal and axial thin-sliced magnetic resonance imaging (MRI).

With very large tumors, a frontal lobe syndrome may become evident, with mental status changes, including changes in personality or behavior, loss of motivation, depressed mood, apathy, and changes in short-term memory.[5] As with other meningiomas, TSMs are more frequent in women than in men, typically presenting in the fifth or sixth decades of life. Physical findings include those consistent with chronic compression of the optic apparatus with reduced visual acuity, an enlarged blind spot, impaired visual fields, and optic atrophy in chronic cases. These findings may be asymmetric.

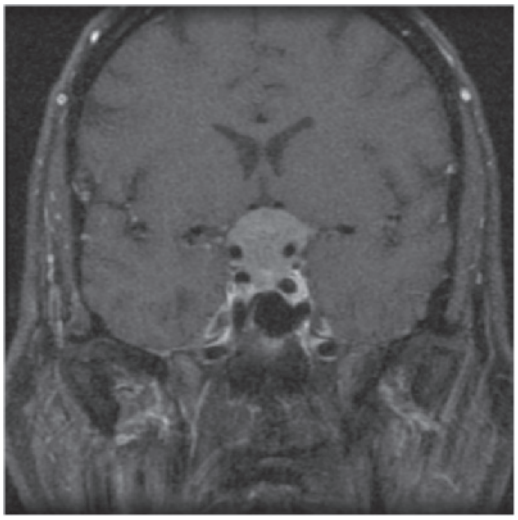
Imaging Characteristics

Most TSMs are assessed by MRI[5,8] (**Fig. 22.1**). Classically, these tumors are distinguished from other midline anterior fossa meningiomas by their general trajectory of growth, which is frequently superior and posterior toward the optic apparatus (**Fig. 22.2**).[8] Because of their location adjacent to the skull base and orbits, fat suppression techniques should be used to identify tumor extension down the medial aspect of the optic canal, as already mentioned. Thin slices, with little or no interslice spacing, are necessary when postcontrast MR images are obtained. Sade and Lee analyzed the incidence of optic canal involvement on a series of 29 patients[9] and found that radiographic evidence of canal involvement was present in 23 of 29 patients (79%) and was lateralized in 19 of 23 (83%).

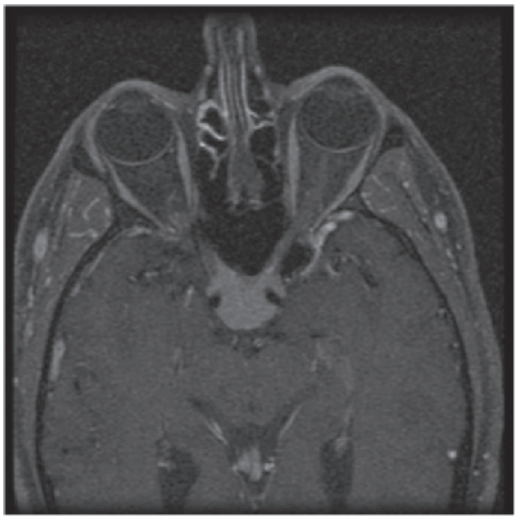
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A

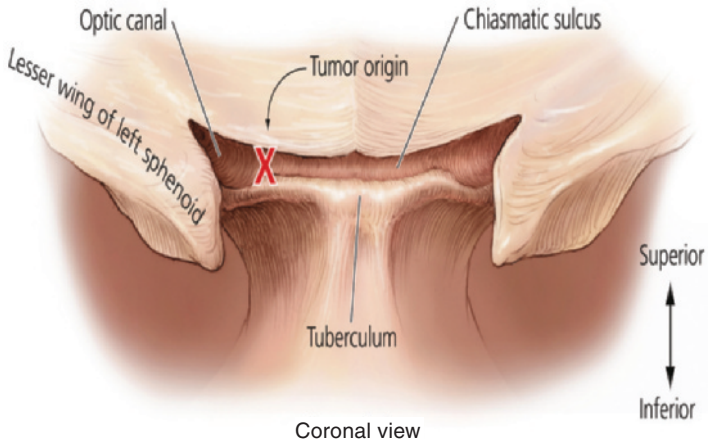


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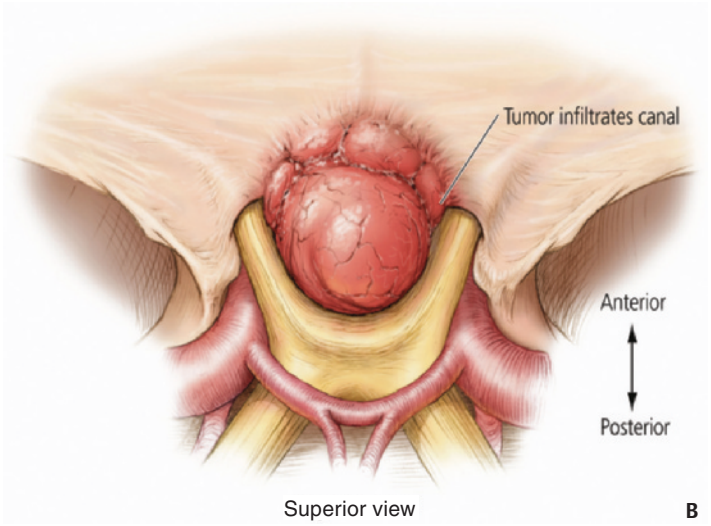


C

Fig. 22.1 Fat-saturated T1-postcontrast **(A,B)** axial, and **(C)** coronal images depicting the imaging appearance of a tuberculum sellae meningioma.



A



B

Fig. 22.2 Schematic diagram demonstrating the **(A)** site of origin and **(B)** anatomical characteristics of a tuberculum sellae meningioma.

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Cerebral angiography is usually unnecessary, except for the largest of tumors, where a roadmap for the course of the displaced vessels is desirable. Given that the typical blood supply of these tumors arises from pial, posterior ethmoid, and unnamed meningeal vessels arising from the cavernous segment of the carotid artery in the largest tumors, significant preoperative embolization is rarely possible.[10] Magnetic resonance angiography can also demonstrate the course of displaced vessels. Magnetic resonance venography has little role in the assessment of these tumors preoperatively or in the choice of surgical approach.

Approach Selection

As already stated, surgical resection is the only reasonable treatment option for most patients with TSM because visual loss is usually present, making any therapy that does not relieve this mass effect on the optic apparatus an undesirable option in most cases. Further, the proximity of these tumors to the optic apparatus usually eliminates radiosurgery as a treatment option for these tumors.[4]

Thus, clinical decision making in TSM usually involves which approach to use. A brief summary of approach options for large and small TSMs is provided in **Table 22.1**. In large part, given the lack of definitive comparative data, this decision stems from an individual surgeon's comfort with these approaches, especially with the endonasal approaches. However, with larger tumors, it is important to systematically study the preoperative images because specific pathoanatomical characteristics can make a specific approach more or less favorable for a given tumor's anatomy.

1. **Optic canal invasion:**
   - TSMs usually invade the optic canal in its medial aspect. Although the medial optic canal can be opened endonasally to address this tumor through the sphenoid sinus, opening the medial optic canal in its length and successfully closing this defect is challenging; thus the presence of medial optic canal invasion is a contraindication to the endonasal approach in all but experienced hands. The presence of tumor lateral to the optic nerve is presently a contraindication to the endonasal approach in all hands.
   - Bioptic canal involvement is usually easier to address via the bifrontal approach (**Fig. 22.3**), however the anterolateral approaches (pterional/orbitozygomatic) are also able to deal with this situation in most cases.
2. **Optic chiasm location:**
   - The chiasm is typically displaced superiorly or posteriorly by these tumors. The location of the tumor in relation to the optic nerve can significantly change the relative simplicity of the transcranial or transsphenoidal approaches. For example, a superiorly displaced, somewhat prefixed chiasm can have a relatively small intraoptic triangle to work through transcranially, especially toward the end of the resection, but might be a straightforward surgery approached transsphenoidally. Alternately, the presence of a significant portion of tumor superior to the chiasm may be better approached transcranially.
3. **Vascular encasement**:
   - Significant involvement of the internal carotid, anterior cerebral, or anterior communicating arteries largely contraindicates the endonasal approaches.

Outcomes

Over the past decade, several surgeons have published their results with the use of the microsurgical transcranial and the extended endonasal approaches for removal of these lesions.[1,5–7,9,11–32] These studies are summarized in **Table 22.2**. In addition, we provide a summary of the published literature regarding extent of resection, and visual outcomes for the transcranial and transsphenoidal approaches, for studies published since 2000. The 95% confidence intervals (95% CI) in this table were calculated using a random effects iterative least squares meta-analysis method with weighting performed using the inverse variance method as published by DerSimonian and Laird.[33–36] In short, this method provides a pooled portion that analyzes between center heterogeneity and generates a pooled proportion (in this case, the rate of gross total resection [GTR], visual worsening, or improvement) for these approaches that considers the effect of between-center differences in surgical technique, or personal philosophies when generating an estimate of pooled rates.

As demonstrated in **Table 22.2**, GTR was achieved in 88 to 92% (95% CI) of cases operated via the transcranial approaches. Visual worsening was reported in 12 to 17% of these cases, whereas visual improvement was noted in 57 to 63%. Significantly fewer data are available for TSMs operated via the extended endonasal approaches; however, we estimate a pooled rate of 55 to 78% (95% CI) of GTR with these surgeries, with stable or improved vision in 91 to 100% of cases. Taken together, these data imply a more conservative approach by surgeons performing the endonasal approach, with marginally better visual outcomes traded for fewer complete resections. However, more data are clearly needed to further elaborate this issue, especially given the limited experience with the endonasal approach to treat TSMs.

In the following discussion, we focus on a few studies that address issues outside the simple rates of success represented in a pooled rate of the literature.

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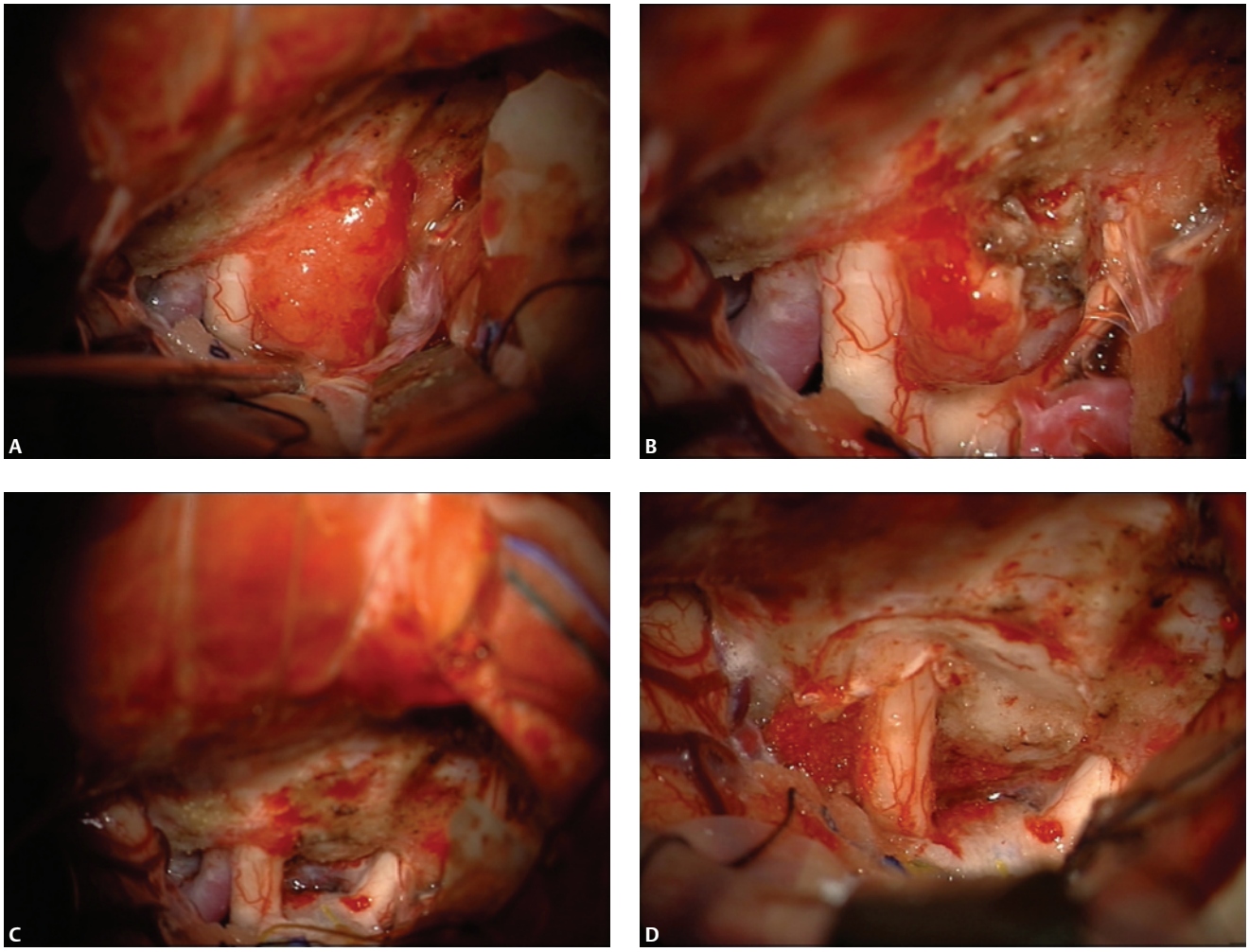


Fig. 22.3 Intraoperative photos demonstrating the microsurgical appearance of a tuberculum sellae meningioma **(A,B)** before and **(C,D)** after tumor resection.

Table 22.1 A Summary of Approach Options for Tuberculum Sellae Meningiomas of Various Sizes

Tumors < 2.5 cm	Tumors > 2.5 cm
Approach options:	Approach options:
Pterional	Pterional
Orbitozygomatic	Orbitozygomatic
Lateral subfrontal	Lateral subfrontal
Endonasal, endoscopic Transsphenoidal	Bifrontal
Endonasal, microscopic Transsphenoidal	Bifrontal–extended frontal

Which Transcranial Approach Is Best?

More than a mere semantic argument, the bifrontal and frontolateral approaches provide different intraoperative trajectories, and as a result, different views of the optic apparatus and tumor. There are no definitive data supporting the superiority of one approach over another, though we discuss what is known regarding this topic in the following pages.

Using a standard pterional craniotomy, Fahlbusch and Schott reported on their early experience between 1983 and 1998.[18] There were 47 cases and a GTR was accomplished in 98%. Mortality was 0%, morbidity 15%, and the cerebrospinal fluid (CSF) leak rate 6%. Nakamura et al reported on a series of 72 patients using three different transcranial approaches: frontolateral (n = 27); pterional (n = 16), or bifrontal (n = 13).[26] GTR was accomplished in 92% of the patients overall, with mortality of 2.8%, morbidity 9.5%, and CSF leak rate of 4.1%. The postoperative visual impairment score seemed to be better with either the frontolateral or pterional approach, as opposed to the bifrontal approach.

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Table 22.2 Summary of Transcranial and Transsphenoidal Surgical Series Published in the Last 10 Years, Including Our Own Institutional Experience

	Transcranial					Transsphenoidal				
Study and First Author	***n***	**% GTR**	**% Worse**	**% Same**	**% Better**	***n***	**% GTR**	**% Worse**	**% Same**	**% Better**
Bassiouni	62	90	17	30	53	0	NA	NA	NA	NA
Chicani	18	78	33	56	11	0	NA	NA	NA	NA
Couldwell	0	NA	NA	NA	NA	11	64	9	91	
de Divitiis	44	86	13	26	61	7	83	0	29	71
Dusick	0	NA	NA	NA	NA	7	57	0	15	85
Fahlbusch	47	98	19	7	74	0	NA	NA	NA	NA
Fatemi	0	NA	NA	NA	NA	14	NA	14	86	
Goel	70	84	11	35	54	0	NA	NA	NA	NA
Jallo	23	87	19	26	55	0	NA	NA	NA	NA
Kim	27	74	19	31	51	0	NA	NA	NA	NA
Kitano	12	NA	42	16	42	16	NA	19	19	62
Laufer	0	NA	NA	NA	NA	10	90	0	0	100
Li	43	74	19	34	47	0	NA	NA	NA	NA
Margalit	20	80	14	58	28	0	NA	NA	NA	NA
Mathiesen	29	90	0	7	93	0	NA	NA	NA	NA
Nakamura	72	92	35	65		0	NA	NA	NA	NA
Nozaki	22	72	23	32	45	0	NA	NA	NA	NA
Otani	32	88	13	9	78	0	NA	NA	NA	NA
Pamir	42	81	14	28	58	0	NA	NA	NA	NA
Park	30	77	30	70		0	NA	NA	NA	NA
Sade	31	84	3	17	80	0	NA	NA	NA	NA
Schick	53	91	13	49	38	0	NA	NA	NA	NA
UCSF	69	78	11	42	47	0	NA	NA	NA	NA
Wang	0	NA	NA	NA	NA	7	14	0	14	86
Zevgaridis	62	92	17	18	65	0	NA	NA	NA	NA
95% CI		88–92	12–17	20–26	57–63		55–78	0–9	91–100	

Abbreviation: NA, not applicable.
Rates of gross total resection (GTR), as well as rates of visual worsening, stability, and improvement are listed for patients undergoing transcranial and transsphenoidal approaches for surgical resection of TSM. At the bottom of each column, a calculated pooled 95% confidence interval (CI) is listed for each category based on the results of our random effects meta-analytic modeling.

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We utilize the bifrontal extended frontal approach for larger meningiomas (i.e., > 2.5 cm). The decision to remove the orbital bar in these cases is based on the philosophy that removal of bone reduces risk of secondary brain injury because the tumors typically do not provide much space in these cases. We use a single medium-blade retractor, which requires movement many times during the case to maintain exposure and thus reduces the likelihood of prolonged brain compression and subsequent ischemia. Advantages of this approach include good visualization of the medial sides of both optic nerves at their entrance into the canal, the most common site of TSM origin; ease of access to the regions lateral to both carotids; good visualization of the suprasellar cistern; and good access for drilling down the planum/tuberculum for excision of dura along the anterior face of the sella. Disadvantages of this approach include longer operating times for the surgical approach and reconstruction; requirement for dissection of the olfactory apparatus, which risks anosmia; and opening of both frontal sinuses and the risk of supraorbital bar mucoceles. Unilateral approaches for large tumors may be hampered by the inability to see past the ipsilateral optic nerve. In Chi and McDermott's study of patients between 1997 and 2005 operated via this extended frontal approach,[5] FLAIR T2 signal changes related to surgical retraction were characterized through four grades: (grade A) no edema, (grade B) gyrus rectus edema, (grade C) edema beyond the gyrus rectus, and (grade D) extensive bifrontal edema. There were 45 patients, 54% with tumors greater than 4 cm operated using this technique. Ninety-one percent of the patients were classified as group A or B in the immediate postoperative period, indicating that less than 10% had evidence of new retraction-related injuries. There were no infections and two CSF leaks in this series.

Is It Beneficial to Unroof the Optic Canal?

TSMs frequently invade the optic canal, which raises the question whether it is a good idea to remove the bone of the optic canal to address this. Although the potential benefits of decompressing the optic nerve and removing tumor from this tight space seem evident, the process of unroofing the nerve has some risk of heat or physical injury to the optic nerve. There presently are insufficient data for, or against, this practice. One study by Nozaki et al in 2008 reported on 20 patients and the effect of early optic canal and roofing on the outcome of visual function.[27] Nine patients had early opening of the optic canal, seven late opening, and six no opening. Timing of optic canal unroofing was a significant predictor of improved visual outcome in multivariate analysis controlling for patient characteristics such as age, sex, preoperative visual status, and duration of preoperative symptoms, all of which were not significant factors. Early optic canal opening was significantly associated with improved visual outcome. The authors recommended early optic canal unroofing in the surgery for TSMs.

What Are the Risks of the Endonasal Approach?

One of the earliest reports on the extended transsphenoidal microsurgical approach to these tumors was included in the report by Couldwell et al[12] on a series of 105 patients between 1982 and 2003. There were 11 TSMs or diaphragma sellae meningiomas. Sixty-four percent of the patients had GTR, with a mortality of 0%, morbidity 14%, CSF leak rate of 6%, and visual worsening rate of 10%. More recently, de Divitiis et al[13] reported on the fully endoscopic extended transsphenoidal approach for TSMs in six patients. GTR was accomplished in five of six, with CSF leaks in 33%, mortality in one of six, and visual improvement in five of six patients. Fatemi et al, in 2009, compared and contrasted the endonasal with the supraorbital approach for a series of patients with craniopharyngiomas or TSMs.[19] In this series of 14 patients undergoing endoscopic endonasal surgery for TSM, one had a new extraocular palsy, one had new postoperative hypopituitarism, and four (29%) had CSF leaks. In the supraorbital craniotomy group of nine TSMs, one had carotid or other vascular injury, one had postoperative visual worsening, and none had hypopituitarism or CSF leak.

Our Own Results

A recent evaluation of 69 patients with TSMs at the University of California, San Francisco, revealed a mean age of 54 years and that visual loss was the presenting symptom in 87%.[5] Humphrey visual fields were performed preoperatively in all patients, and bilateral visual field impairment was more common than unilateral. The mean duration of symptoms was 18 months. The median maximum tumor dimension was 2.35 cm (1.8 to 2.9 cm) and median tumor volume was 7.68 cc (2.25 to 18 cc). Surgical pathology was World Health Organization (WHO) grade I in 71% and WHO grade II in 29%. Extent of resection was evaluated as Simpson grade I to III in 78%, and grade IV or V in 21%. The bifrontal extended frontal approach was used in 58% of the cases, unilateral subfrontal in 38%, and transsphenoidal in 4%. The bifrontal extended frontal approach was similar to that previously reported by Chi and McDermott.[5] In that report there was a very low incidence of new secondary brain injury related to retraction. Mean operative time in that series of patients was 10.2 hours and mean blood loss 552 mL. In terms of visual outcomes, 47% were improved, 42% were stable, and 11% were worse. In the four patients who developed postoperative unilateral blindness, a delay of 1 to 2 days postoperatively was nearly universal. There was one case of bilateral blindness in a patient where both optic canals were opened. There were no new endocrine deficits, and two patients (3%) had CSF leaks. There was no mortality, and combined medical/surgical morbidity was 11%. Median clinical follow-up was 20 months, radiographic follow-up 19 months. The venous infarction rate was 4% and new seizure rate was 4%.

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Complication Avoidance

Visual Loss

Avoidance of visual decline is not possible in all cases, in part due to our limited understanding of the etiology of visual loss following surgery for TSMs.[6,12,13,15,16] The onset of visual loss can be immediate, and in these cases is probably a result of direct operative trauma.[6] Alternatively, in some cases, vision loss can be delayed, without evidence of postoperative hematoma, brain swelling, or other neurological change.

It has been suggested that early opening of the optic canal may reduce the risk of visual impairment by reducing tension and sheer stresses on the optic nerve during early tumor manipulation and debulking.[27] Intraoperative monitoring via direct retinal stimulation is generally not possible given the incompatibility of the surgical approaches and the equipment necessary for generating the light stimulus for visual evoked potentials. Until the ideal technology for safely unroofing the optic nerves becomes available, we recommend gentle drilling with a coarse diamond bit drill with continuous irrigation.

We have found that preservation of the vascularized arachnoidal layer overlying the anterosuperior surface of the optic apparatus is almost always possible with careful sharp microsurgical dissection, and we think this is an essential technique for reducing the risk of vision loss. We believe that it is important to maintain the patient's blood pressure in the high normal range for 48 hours postoperatively and to maintain adequate hydration because we speculate that many delayed cases of vision loss come from tenuous perfusion exacerbated by postoperative swelling in this region.

Poor Cosmetic Results

Cosmetic results with the bifrontal extended frontal approach can be improved by countersinking any titanium plates used along the midline, or on the posterior edge of the craniotomy, by thinning out a small area of the outer table for the plate to rest in, then using hydroxyapatite to fill gaps between the craniotomy cuts. Removing the frontal bone in two pieces, the so-called bipartite flap, makes dissection of the midline venous sinus under direct vision easy, and a reasonable cosmetic result can be obtained with the two-piece flap if the foregoing reconstructive procedures are used.

The supraorbital approach raises the most significant cosmetic concerns, in large part due to the use of an eyebrow incision. Cosmesis should be planned into the opening and can be improved by beveling the inner edge of the bone cut so that the lower supraorbital bone flap cut will sit flush, providing a more cosmetically appealing surface.

Cerebrospinal Fluid Leakage

CSF leakage is an issue in both transcranial and endonasal approaches; however, it is unquestionably a greater concern with endonasal surgery. It is challenging to close the area of the tuberculum sellae from below,[12,14] given the difficulty of safely placing absorbable plates or bone under the edges of the bony defect, which typically contain the optic nerves. Fat placed in this space should be compacted using suture before placing it in the tubercular defect[37] to reduce the risk of optic compression. Further, the use of a pedicled vascularized nasoseptal flap is very helpful in these cases, given the difficulty obtaining a safe multilayered conventional closure in this region[38] and the widely violated arachnoidal layer after endonasal TSM resection.

Frontal Sinus Mucocele

Mucoceles may be a consequence of the transcranial approaches when the frontal sinus is inadvertently or intentionally entered, and it is insufficiently addressed at the time of surgery. This problem is exacerbated in situations when the frontal sinus outflow is obstructed.[5] We routinely harvest pericranium as a vascularized flap in all transcranial cases and utilize this flap to exclude the frontal sinus from the intracranial space, with the flap placed below the orbital osteotomy piece, if present. We strip the mucosa from the orbital unit in the bifrontal–extended frontal approach and the orbitozygomatic approach (if the sinus is entered); however, we do not exenterate the frontal mucosa or obliterate the nasofrontal duct because the pericranial patch is usually adequate to prevent mucocele formation if the nasofrontal duct remains patent.

Conclusion

The treatment for TSMs is surgery in almost all cases, and the remaining questions involve the best way to perform vision-preservation surgery in these patients. Although visual recovery or stabilization is achievable in most cases, in most series there is at least a 10% risk of visual worsening with surgery for these tumors, suggesting that there is room for improvement in current techniques. One major limitation in our ability to improve visual outcomes in TSM patients is an inadequate understanding of all the mechanisms of postsurgical visual loss in these patients. Although sharp dissection preserving the arachnoidal vascular supply to the chiasm and possibly unroofing the optic canal probably reduce the risk of vision loss in these cases, it still happens for reasons we do not entirely understand, and it serves as one future area of focus for the treatment of TSMs.

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Chapter 23

Lateral and Middle Sphenoid Wing Meningiomas

Matthias Simon and Johannes Schramm

Introduction

A sizable proportion of meningiomas arise in the sphenoid region. In large surgical series, sphenoid wing meningiomas account for 11 to 18% of cases.[1–3] There are no pathological and molecular genetic features specific to sphenoid wing meningiomas. However, malignant and atypical meningiomas are less frequently encountered in the skull base than at the convexity.[4] Ionizing radiation can cause sphenoid meningiomas.[5,6] Meningiomas are more common in females. This is particularly true for meningiomas en plaque of the sphenoid wing. Between 77 and 94% of these growths are diagnosed in women.[7]

Surgery for sphenoid wing meningiomas poses a variety of problems reflecting the complex anatomy of the sphenoid region. Tumors of the outer third of the sphenoid ridge and many small middle wing tumors are essentially convexity meningiomas with regard to clinical presentation and surgical treatment (**Figs. 23.1** and **23.2**). More medially located sphenoid wing meningiomas may compress and encase the carotid artery and its branches, the optic apparatus, and the pituitary stalk (**Fig. 23.3**). Sphenoid wing meningiomas may show prominent dural involvement (en plaque growth, **Figs. 23.4, 23.5,** and **23.6**). Tumors may grow in the dural layer overlying the cavernous sinus and in the cavernous sinus itself (**Fig. 23.7**). Some sphenoid ridge meningiomas ("sphenoorbital meningiomas") are characterized by prominent bone invasion and hyperostosis involving the lateral orbital wall, the orbital roof, and eventually the orbit itself, resulting in cosmetic problems and exophthalmos, but also in compression of the optic nerve with subsequent loss of visual function. Bone invasion and hyperostosis are usually associated with meningioma growth along the underlying dura. Tumor growth may extend into the facial and subcranial regions (**Figs. 23.4** and **23.7**). In some patients, bilateral or multifocal tumors are encountered (**Figs. 23.6** and **23.7**).

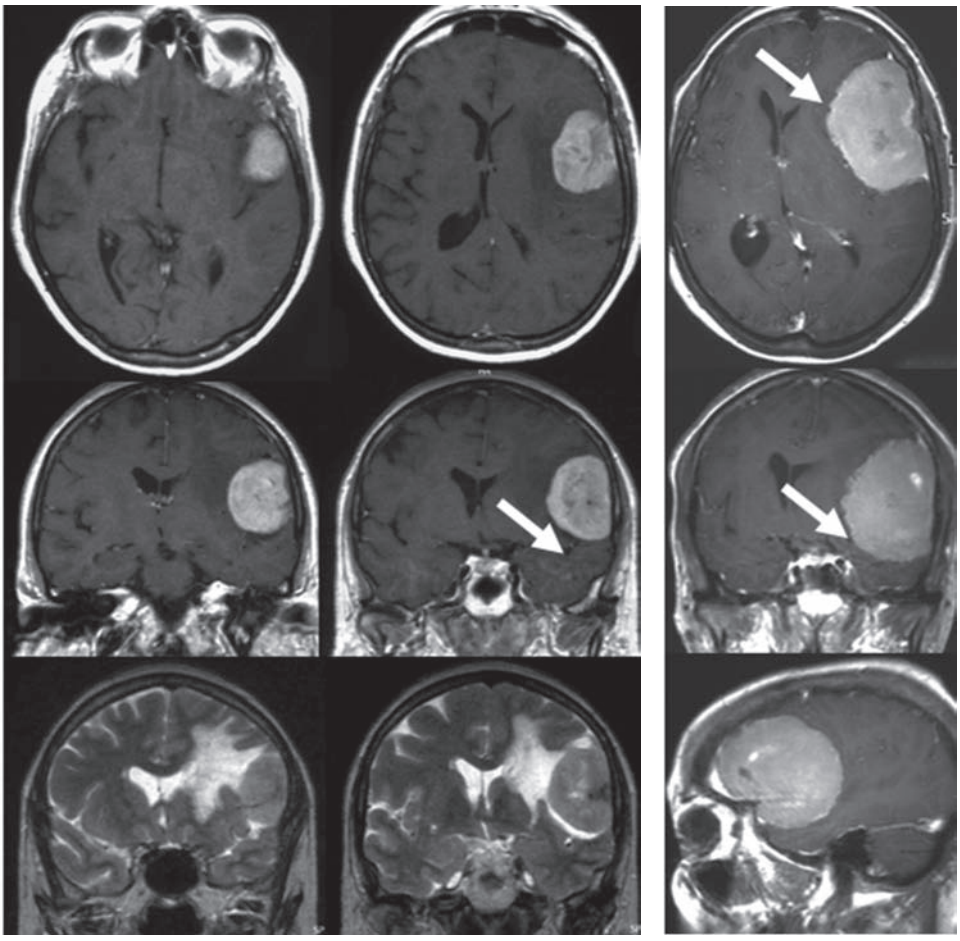
Surgical objectives vary considerably with tumor site and growth pattern. Intradural tumor with significant mass effect usually warrants surgery. Ideally, one should aim for a complete resection. Intracranial tumor close to the optic apparatus should be resected aggressively because even a small regrowth can cause loss of vision and visual field defects, sometimes before an unequivocal mass can be visualized by magnetic resonance imaging (MRI). On the other hand, visual symptoms and exophthalmos due to orbital involvement are often adequately treated by orbital decompression and limited intraorbital surgery. Osseous involvement is often primarily a cosmetic problem.

Most neurosurgeons broadly distinguish between (globular) tumors of the lateral and middle third of the sphenoid ridge, medial wing tumors, and hyperostosing/en plaque (sphenoorbital) meningiomas. This chapter focuses on tumors of the outer and middle sphenoid ridge. However, many sphenoid tumors do not fit very well into these categories (**Figs. 23.4, 23.5, 23.6,** and **23.7**). The concepts of surgery for medial sphenoid wing and sphenoorbital meningiomas often have to be applied to operations for middle wing tumors as well.

Classification of Sphenoid Meningiomas

The first classification for sphenoid wing meningiomas was proposed by Cushing and Eisenhardt. Cushing and Eisenhardt distinguished between globular meningiomas of the deep or clinoidal third, those of the middle ("alar"), and those of the outer third of the sphenoid ridge ("pterional meningiomas"), and *en plaque* pterional tumors.[8] Bonnal et al and Brotchi and Pirotte describe five categories of sphenoidal meningiomas.[9,10] Clinoidal tumors are

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A B

Fig. 23.1 Two lateral sphenoid wing (pterional) meningiomas. **(A)** Medium-sized meningioma originating from the most lateral aspect of the left greater sphenoid wing (T1-weighted contrast-enhanced axial and coronal, and T2-weighted coronal scans). Surgery for this tumor was no different from surgery for a convexity meningioma. The middle cerebral artery (*arrow*) was protected by a thin layer of brain tissue, and identification and preservation of its distal branches were not difficult. **(B)** A much larger tumor in a similar location diagnosed in a 67-year-old female patient who presented with flattened affect, cognitive decline, a possible seizure, and a mild hemiparesis (triplanar T1-weighted contrast-enhanced scans). The middle cerebral artery (*arrow*) proved relatively easy to dissect; however, several M3 branches were engulfed by the tumor. Copyright M. Simon and J. Schramm, reproduced with permission

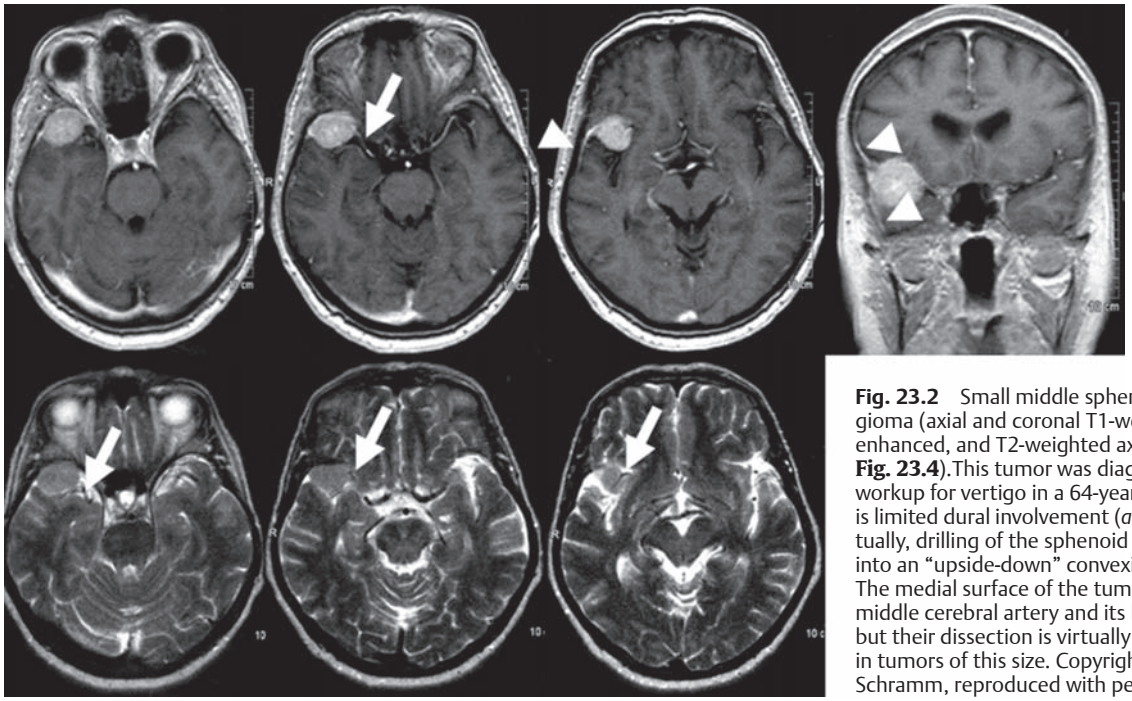
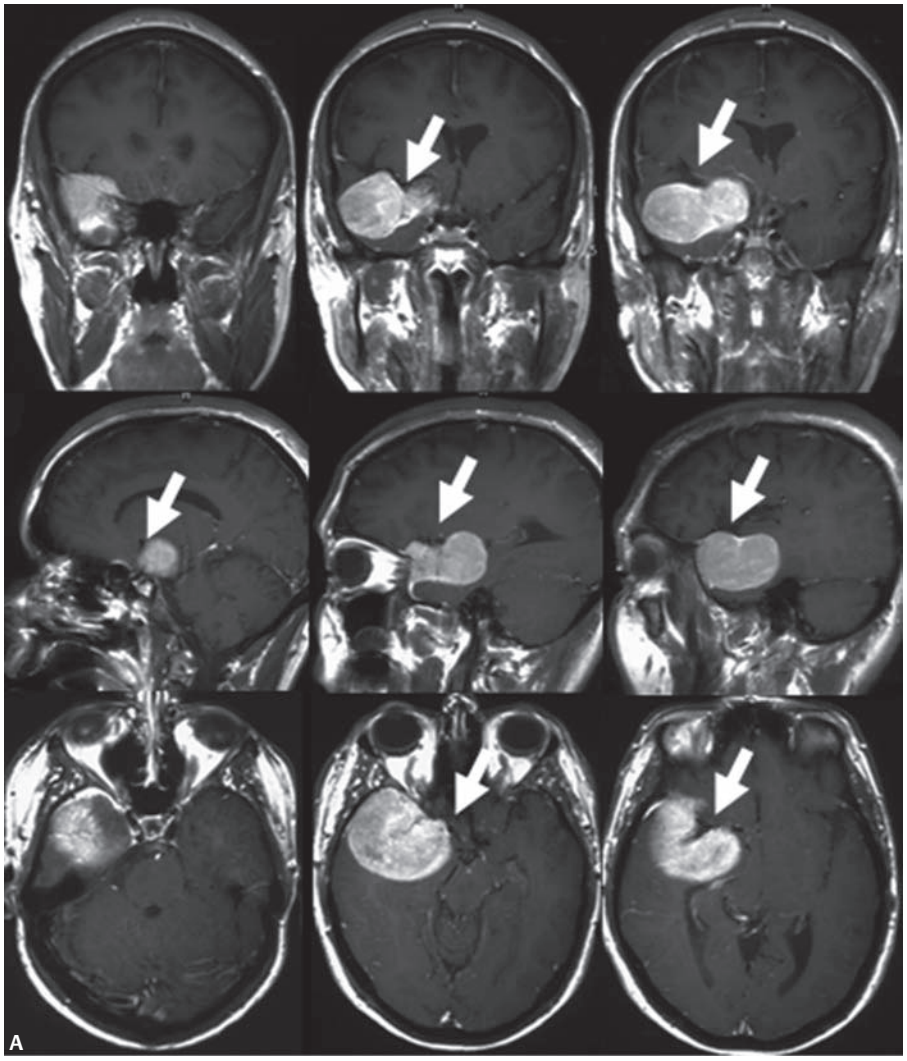


Fig. 23.2 Small middle sphenoid wing meningioma (axial and coronal T1-weighted contrast-enhanced, and T2-weighted axial scans; compare **Fig. 23.4**).This tumor was diagnosed during the workup for vertigo in a 64-year-old female. There is limited dural involvement (*arrowheads*). Conceptually, drilling of the sphenoid can turn this tumor into an "upside-down" convexity meningioma. The medial surface of the tumor is related to the middle cerebral artery and its branches (*arrows*), but their dissection is virtually never a problem in tumors of this size. Copyright M. Simon and J. Schramm, reproduced with permission.

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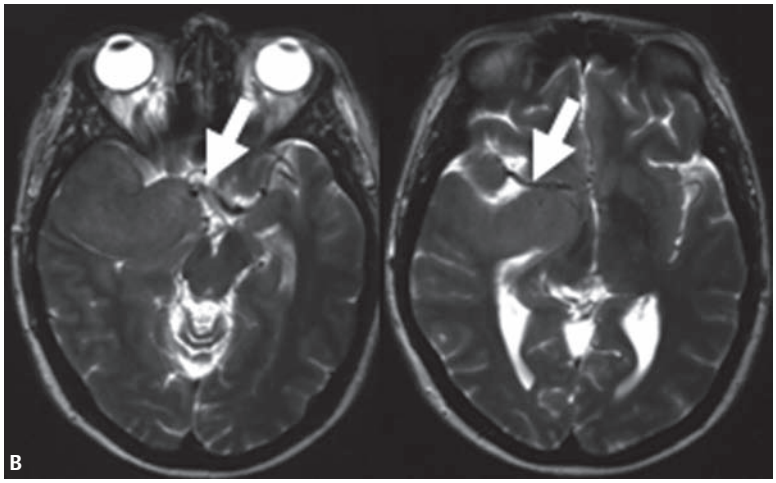




Fig. 23.3 Large middle sphenoid wing meningioma. The patient was a 41-year-old woman who presented with a generalized seizure. **(A)** The size and location of the tumor are well delineated by triplanar T1-weighted magnetic resonance imaging. There is only minimal dural involvement. The course of the carotid and the middle cerebral artery and its branches are visible (*arrows*). Tumors of this size may encroach upon and compress the optic apparatus (for comparison see **Figs. 23.6** and **23.9**: optic nerve compression from dural and intraosseous growth). **(B)** The vascular relations (*arrows*) are depicted even more clearly on T2-weighted images. **(C)** At surgery the tumor was found to originate from the middle sphenoid ridge (i.e., this tumor is not a medial wing [clinoidal] meningioma with a large lateral component). Pamir et al have pointed out that clinoidal meningiomas usually infiltrate the bone of the anterior clinoid process.[16] There was no bone infiltration in this case. Copyright M. Simon and J. Schramm, reproduced with permission.

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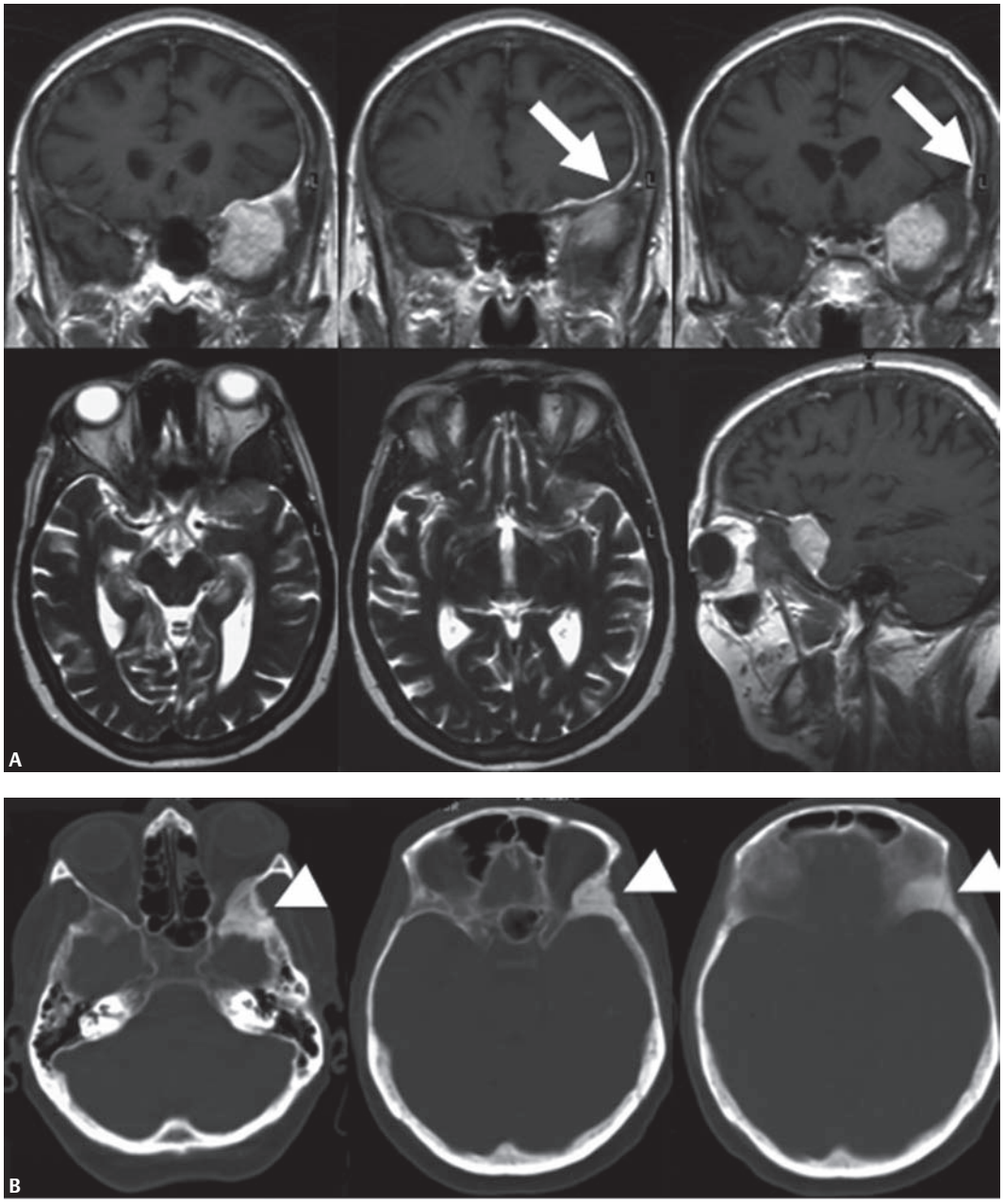


Fig. 23.4 Another small middle sphenoid ridge meningioma (coronal and sagittal T1-weighted contrast-enhanced and axial T2-weighted images; compare **Fig. 23.2**). **(A)** In contrast to the tumor shown in **Fig. 23.2,** there is more widespread dural growth (*arrow*). **(B)** A computed tomographic scan shows tumorous infiltration of the sphenoid bone (*arrowheads*). Thickening of the lateral orbital wall has already resulted in mild exophthalmos. The bony and dural involvement render this lesion more challenging than the case presented in **Fig. 23.2**. One could argue that this tumor should be classified more precisely as a sphenoorbital meningioma (compare **Fig. 23.7**). Copyright M. Simon and J. Schramm, reproduced with permission.

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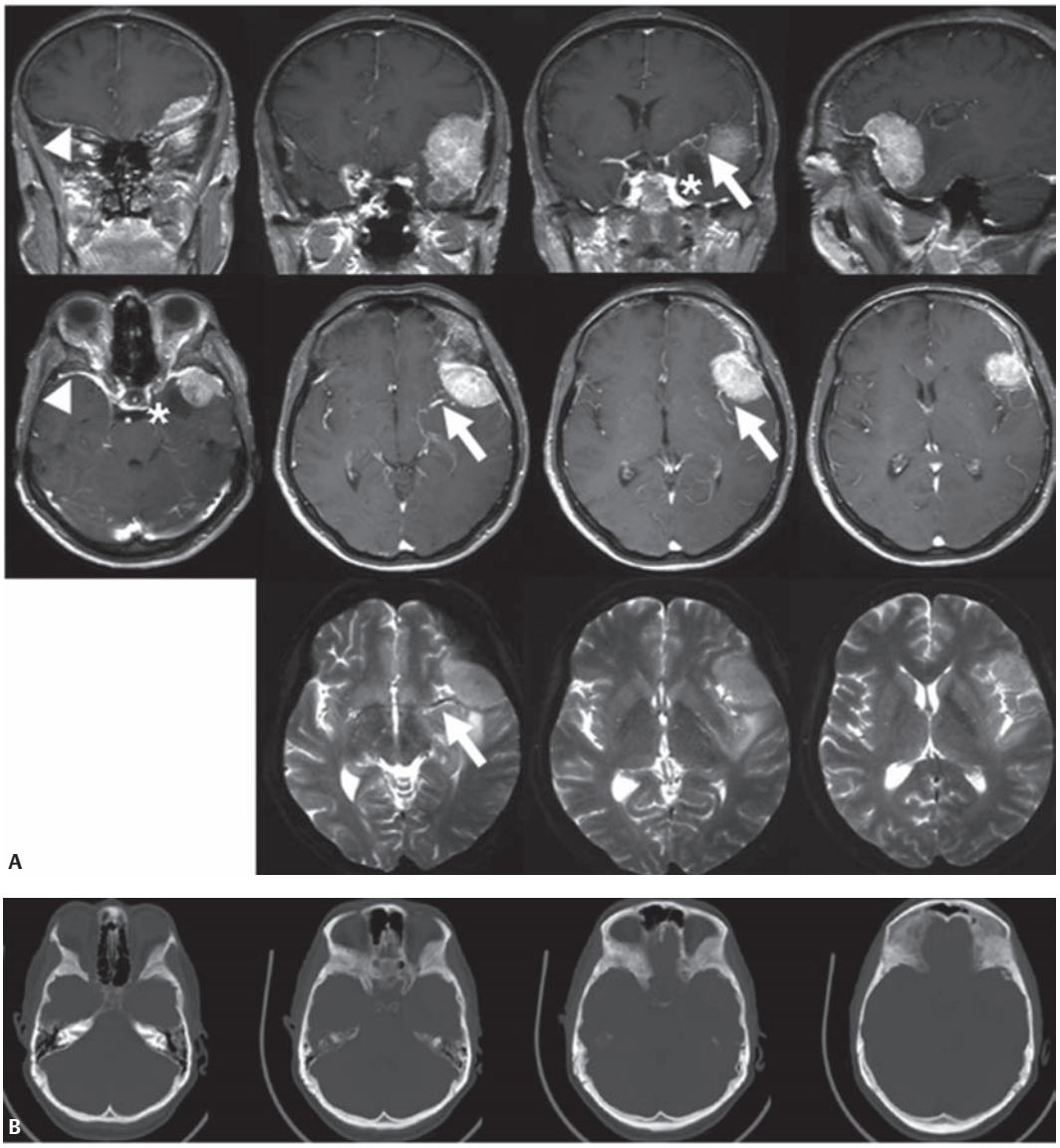


Fig. 23.5 A bilateral sphenoid wing meningioma with a left-sided, medium-sized globular pterional (lateral wing) component. The tumor became symptomatic with seizures. See **Fig. 23.8** for the corresponding intraoperative findings. **(A)** T1-weighted and axial T2-weighted images depicting a tumor mass centered on the left lateral sphenoid ridge, and tumorous infiltration of the ipsilateral as well as the contralateral dura overlying the right sphenoid ridge (*arrowhead*). The dorsomedial border of the tumor mass appears related to the middle cerebral artery and its branches (*arrows*). There is also a (nonneoplastic) cyst located immediately adjacent to the medial surface of the tumor (*). **(B)** Computed tomographic scans show bilateral and extensive involvement of the sphenoid bone largely sparing the left anterior clinoid process. Copyright M. Simon and J. Schramm, reproduced with permission.

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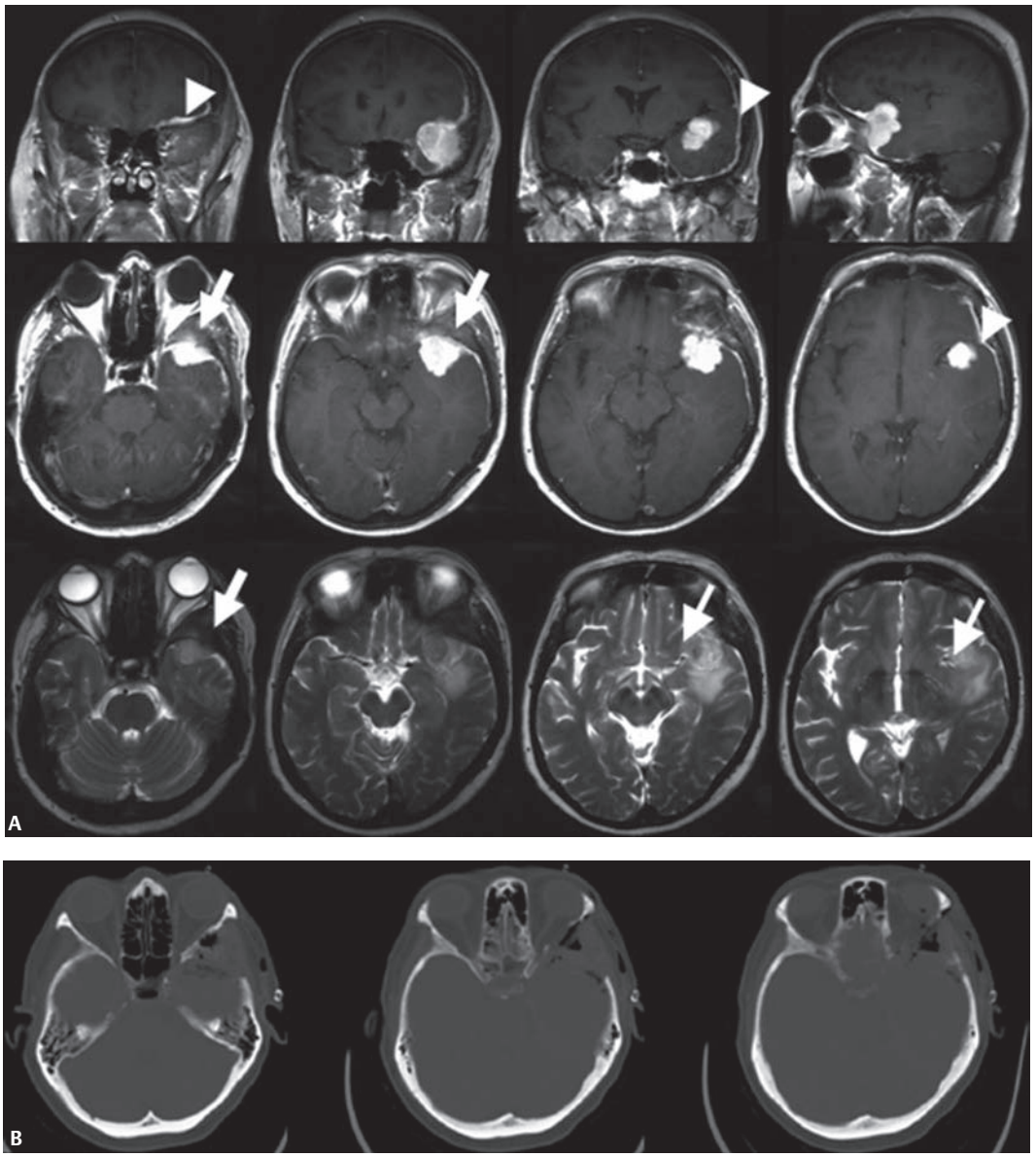


Fig. 23.6 A medium-sized meningioma of the left middle sphenoid wing with extensive dural involvement and intraosseous growth. The tumor was diagnosed in a 73-year-old woman following several seizures. The ophthalmological exam was normal. **(A)** T1-weighted contrast-enhanced magnetic resonance imaging (MRI) scans showing the origin and size of the tumor (upper and middle row). There is extensive carpetlike involvement of the frontotemporal dura (*arrowheads*). The course of the middle cerebral artery and the M2 branches (*thin arrows*) are nicely depicted on T2-weighted images (lower row). At surgery, the vessels could be easily identified and preserved (see **Fig. 23.9**). Intraosseous growth is already apparent on the MRI scans (*arrows*). Involvement of the lateral orbital wall has resulted in mild exophthalmos. Dural and intraosseous tumor spread will sooner or later lead to optic nerve compression even before MRI will show a definite tumor mass (for comparison see **Fig. 23.3**: tumor mass encroaching upon the optic apparatus). **(B)** Postoperative computed tomographic scans depicting the extent of the bony resection. Copyright M. Simon and J. Schramm, reproduced with permission.

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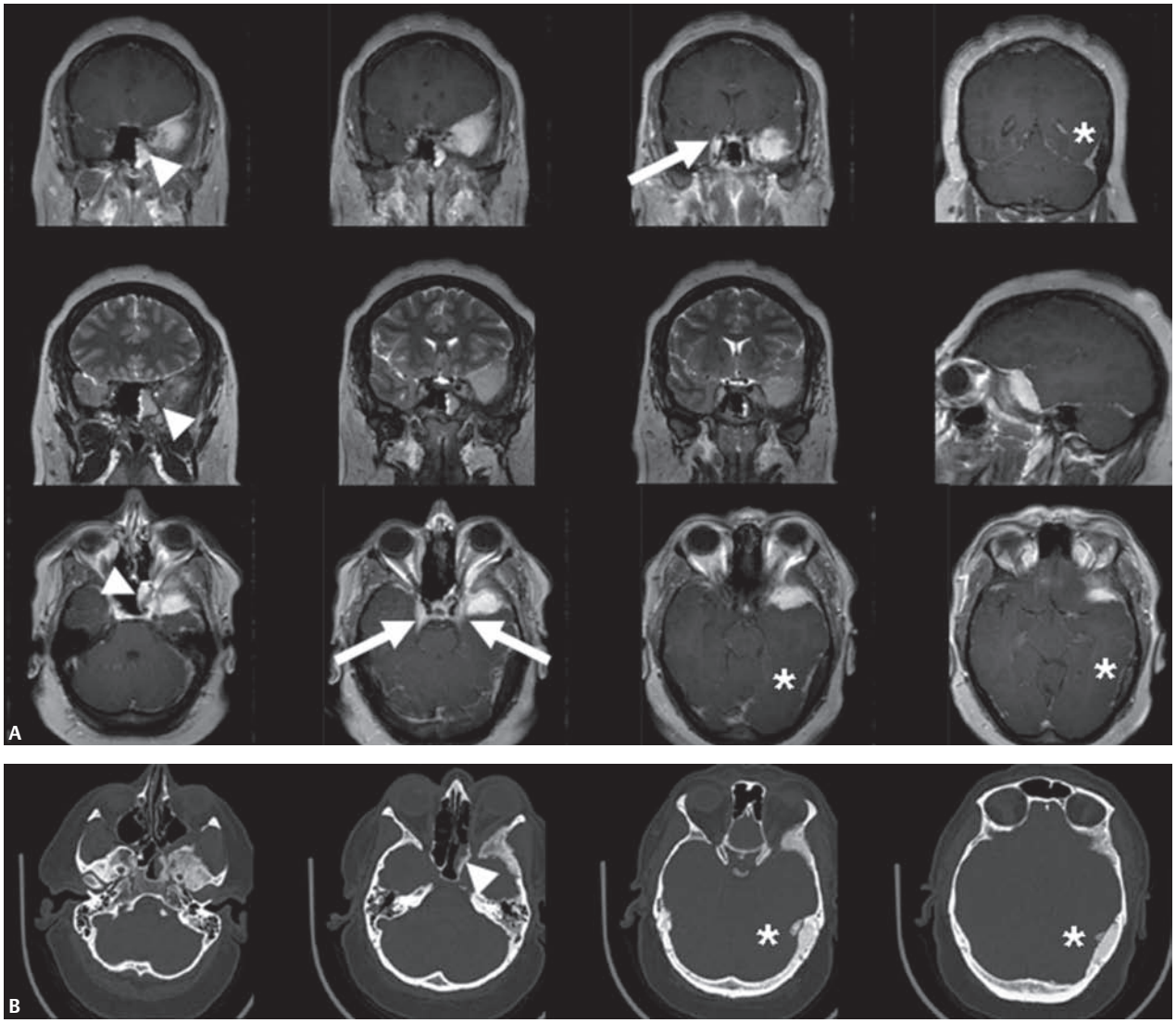


Fig. 23.7 **(A,B)** A sphenoorbital meningioma (compare **Fig. 23.4**) with a sizable globular tumor component centered on the middle sphenoid wing, bilateral infiltration of the cavernous sinus (*arrows*) and growth in the paranasal sinuses (*arrowheads*). Note that there is a second en plaque tumor in the left temporodorsal/occipital region (*). This growth pattern precludes a surgical cure. Surgical objectives in this case include removal of the intradural globular mass, and decompression of the orbit and the optic canal. **(A)** Triplanar T1 weighted contrast enhanced and coronal T2 weighted images delineating the globular and the en-plaque component of the tumor. **(B)** CT scans depict the rather extensive bony involvement. Copyright M. Simon and J. Schramm, reproduced with permission.

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referred to as group A tumors. Invasion of the cavernous sinus is listed as one characteristic of these growths. Globular tumors of the middle and lateral sphenoid ridge are termed group D and E meningiomas, respectively. Meningiomas en plaque with hyperostosis of the sphenoid bone (i.e., sphenoorbital meningiomas) are designated group B tumors. It is recognized that the designation *pterional* chosen by Cushing for these growths may not be fully appropriate. Large invasive tumors of the sphenoid ridge ("invasion en masse") are assigned to a separate new category (group C tumors). These tumors are thought to combine features of both group A and B tumors (i.e., globular and invasive growth en plaque). Roser et al further subdivided hyperostosing/en plaque (sphenoorbital) tumors into meningiomas en plaque with and without cavernous sinus infiltration, and purely intraosseous tumors.[11]

Tumors of the medial sphenoid wing are often subclassified based on the presence of invasion of the cavernous sinus.[12–15] Some authors use the term *clinoidal meningiomas* to exclusively refer to meningiomas originating from the anterior clinoid process rather than as a more general designation for middle sphenoid wing meningiomas.[16,17] Al-Mefty has proposed to distinguish between tumors originating proximally to the end of the carotid cistern, which directly enwrap the carotid artery (group I), those with a point of origin at the superolateral aspect of the clinoid process and an arachnoidal membrane interposed between the tumor and the carotid artery (group II), and finally tumors originating medially in the region of the optic foramen (group III).[17] This classification has been adhered to by some but not all authors.[12,13,15,18] Group I tumors are probably rare, and the lack of an arachnoidal dissection plane between the tumor and the carotid artery may be related to other factors such as repeat surgery. Group III tumors may be more appropriately referred to as optic foramen, optic sheath, or optic canal meningiomas.[12,13,18]

Tumor classifications should ideally help to choose the surgical strategy and predict the clinical outcome, including the risk of recurrence. To this end it is probably sufficient to broadly distinguish between (globular) lateral and medial sphenoid wing meningiomas, and hyperostosing en plaque/sphenoorbital meningiomas, and to note any cavernous sinus involvement. For obvious reasons, size matters and should be well recognized.[16] Importantly, one will quite often encounter tumors that do not seem to fit ideally into any of the categories outlined (**Figs. 23.4, 23.5, 23.6,** and **23.7**). The surgical strategy in a specific case will be influenced by the location of the tumor along the ridge (and in particular the corresponding arterial relations), by the extent of bone infiltration (e.g., with or without involvement of the orbital roof or the base of the middle fossa), by the extent of dural involvement, and by invasion of the cavernous sinus beyond the mere infiltration of its outer wall. These factors relate directly to different types of surgical risks (and complications) and to the chances for complete removal.

Clinical Presentation

Patients with sphenoid wing meningiomas may present with loss of vision, visual field cuts and optic atrophy. Visual symptoms result from direct compression or encasement of the optic apparatus but may also reflect tumor growth into and around the optic foramen and canal. Visual symptoms due to optic atrophy and intracranial hypertension may rarely be encountered in very large tumors. Foster-Kennedy syndrome (ipsilateral optic atrophy and contralateral papilledema) has been traditionally associated with large meningiomas of the frontotemporal skull base.

Diplopia is not rare. Tumor growth in the cavernous sinus or superior orbital fissure may lead to ocular palsies. Intraorbital tumor growth characteristically causes diplopia due to restriction of ocular movements rather than oculomotor nerve paresis. Involvement of the cavernous sinus will also sometimes result in sensory loss in the distribution of the ophthalmic (and eventually other) division(s) of the trigeminal nerve. Some patients present with prominent exophthalmos. Exophthalmos reflects the presence of intraorbital tumor, but also intraosseous growth in the lateral orbital wall and orbital roof (**Figs. 23.4, 23.6,** and **23.7**). Exophthalmos may also result from venous stasis. Extracranial tumor growth and hyperostosis can cause cosmetic disturbances, such as frontal and temporal bulging.

Hemiparesis (and aphasia, if the dominant hemisphere is involved) can be encountered in patients with large tumors. Large growths may also present with cognitive and memory deficits, a flattened affect, and personality changes (**Fig. 23.1**). In general, compression and encasement of the major arteries of the anterior circulation are clinically silent. Seizures are relatively frequent. Uncinate fits, and gustatory and olfactory hallucinations pointing to the temporal lobe as their origin are observed, as well as complex-partial and generalized tonic-clonic seizures (**Figs. 23.1, 23.3, 23.5,** and **23.6**). Psychiatric symptoms may be encountered in some patients.

Clinical signs and symptoms reflect tumor location and growth pattern. Tumors of the lateral sphenoid wing will often become relatively large before the development of a focal deficit such as hemiparesis or aphasia will result in the diagnosis. Neuropsychological disturbances will often only become apparent in retrospect. Tumors of the medial sphenoid ridge commonly cause earlier and more specific symptoms due to the proximity of the optic apparatus and cavernous sinus. Patients with sphenoorbital meningiomas typically present with exophthalmos, deterioration of visual acuity and field cuts, and diplopia. Finally, the number of patients with more or less asymptomatic meningiomas diagnosed during the course of a workup (e.g., for headache or vertigo) is increasing (**Fig. 23.2**).

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Diagnostic Workup

Diagnosing a sphenoid wing meningioma on a computed tomographic (CT) or MRI scan is often easy if one is familiar with the neuroimaging characteristics of meningiomas, and, importantly, with the typical growth patterns of these tumors as already outlined. Plain x-rays may show a thickening of the ala minor and the pterion ("smoking pterion") and are said to allow for the diagnosis of sphenoid meningiomas in up to 90% of cases. However, this is now of historic interest only, given the widespread availability of CT and MRI.

Surgery for sphenoid meningiomas requires the depiction of the tumor and its meningeal extensions in relation to the surrounding structures. A triplanar MRI including contrast-enhanced T1-weighted sequences will usually delineate the soft tissue component of the tumor. However, it is impossible to reliably differentiate invasion of dural layers at the margin of the solid portion of the tumor from adjoining neovascularization. CT scans (bone window) are necessary to show the extent of bone invasion (**Figs. 23.1–23.7).** CT scans are therefore of particular importance for surgical planning in cases of en plaque and intraosseous meningiomas. Thin CT slices (e.g., 1 mm) allow for a superb visualization of the bony tumor. T2-weighted images (2 mm) will often delineate the course of the optic apparatus and the major vessels (i.e., the middle cerebral artery in cases of a meningioma of the lateral sphenoid wing, or the carotid artery and its major branches in patients with tumors of the medial ridge). This will obviate the need for formal digital subtraction angiography in most cases. More MRI studies (e.g., of the orbit) may provide additional anatomical information. MRI or CT angiography may also help to delineate the course of major cerebral vessels.

Some neurosurgeons feel more comfortable assessing the vascular relations of sphenoid meningiomas on digital subtraction angiograms. Angiography will also allow for a test occlusion of the carotid artery if injury or sacrifice of this vessel is a possibility. Preoperative embolization with polyvinyl alcohol particles or acrylic microspheres is used as an adjunct in some centers to reduce intraoperative blood loss,[19,20] although in a comparative study no advantage of preoperative embolization could be demonstrated.[19] Meningiomas of the medial sphenoid ridge are usually fed by direct (sometimes intracavernous) branches of the internal carotid, or the ascending pharyngeal artery, and sometimes a recurrent branch of the ophthalmic artery passing through the superior orbital fissure. Lateral tumors derive much of their blood supply from the superficial temporal and middle meningeal arteries. Additional feeding vessels may include the anterior meningeal and other branches of the ethmoidal arteries.

Ophthalmology consults are obtained when patients present with exophthalmos, loss of vision or field cuts, or oculomotor palsies and double vision, or whenever there is a risk that such deficits may be incurred during the surgery.

Surgical Technique

Lateral Sphenoid Ridge Meningiomas

Surgery for lateral wing tumors aims at the complete removal of the tumor and excision of its dural origin, including a safety margin. This will be possible in most cases and will conceptually cure the patient. Large tumor may encroach upon the superior orbital fissure or the cavernous sinus, which will preclude a truly radical resection. We utilize a generous and carefully planned frontotemporal craniotomy exposing the tumor and also any part of the dura invaded by the meningioma. The pterion is drilled as required. A carefully executed craniotomy provides early control of the tumor's main blood supply through dural and pterional branches of the external carotid meningeal artery. The dura is incised in a circumferential manner starting laterally. This results in further devascularization. It may be helpful not to excise the mediobasal dura at this stage of the operation, even if it is involved by the tumor, to prevent undue epidural oozing into the surgical field. Removal of the tumor will often start with internal decompression. Using a cavitronic ultrasonic surgical aspirator (CUSA) may be helpful in some (but not all) cases. Small to medium-sized tumors may not require initial debulking (**Fig. 23.8A–C**).

The borders of the tumor are dissected in a systematic way (**Fig. 23.8A–C**). The main issue at this step is the identification and preservation of the distal branches of the middle cerebral artery. Major branches of the middle cerebral artery and sometimes the main trunk itself may be engulfed by the tumor, but true invasion of the wall of the middle cerebral artery is very rare.[17] We try to identify the distal branches of the middle cerebral artery and follow them around or through the tumor. This has proven much easier than attempting to dissect the proximal middle cerebral artery when there is still a substantial bulky tumor nodule left in the surgical field. Dissecting the middle cerebral artery and its major branches from the capsule of the tumor, and identifying, coagulating, and dividing tumor-feeding vessels can be easy in patients with small tumors and preservation of an arachnoid plane (**Fig. 23.8**). However, if the tumor has encased the middle cerebral artery or its branches, the preservation of these vascular structures may become very difficult. A sharp scalpel can be very useful at this stage of the operation. One has to keep in mind that it is possible to tear the walls of major vessels, including the middle cerebral and the internal carotid artery, with the ultrasonic aspirator.

Finally, any remaining tumor nodules and tumor-infiltrated dura are removed. If a particular part of the dura cannot be safely excised or is intentionally left behind (e.g., in cases with very large tumors extending medially and infiltrating the cavernous sinus) it is coagulated (**Fig. 23.8E–F**). We use free pericranial grafts to obtain a reliable watertight dural closure. It is important to suture the graft onto the basal dura rather than its cut edges, allowing for a generous overlap of dura and graft. In addition, the suture can be sealed with fibrin glue. The bone flap

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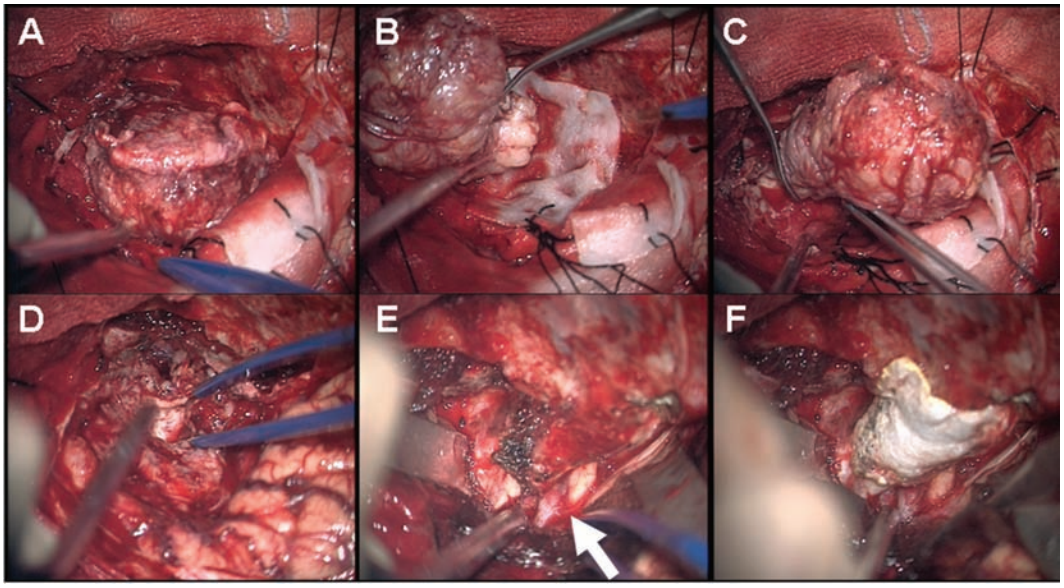


Fig. 23.8 Surgery for a lateral sphenoid wing tumor mass in a patient with bilateral sphenoid bone and dural involvement (see **Fig. 23.5** for neuroimaging findings and clinical presentation). The tumor was removed through a pterional craniotomy followed by drilling of the tumor-infiltrated orbital roof and lateral orbital wall (not shown). **(A–C)** After circumferential incision of the dura the plane between tumor and brain tissue is systematically defined using cottonoids. Internal decompression was deemed unnecessary in view of the limited size of the mass. The M3 branches of the middle cerebral artery were all protected by a thin layer of brain tissue. **(D)** A small nontumorous cyst is held open with the bipolar forceps and inspected in order not to overlook any residual tumor (compare **Fig. 23.5A**). **(E,F)** The anterior clinoid process is not involved by the tumor (see **Fig. 23.5B**), hence only the overlying dura is removed and reconstructed using fibrin glue and Gelfoam (Pfizer, Inc., New York, NY) (*arrow*, optic nerve and carotid artery). Copyright M. Simon and J. Schramm, reproduced with permission.

is reimplanted after all tumorous ingrowths have been removed with a drill. Methylmethacrylate can be used to cover small bone defects, and the wound is closed using standard techniques.

Middle Sphenoid Ridge Meningiomas

Many of the concepts of surgery for lateral wing tumors have to be applied to operations for middle sphenoid ridge meningiomas as well (**Fig. 23.9**). Removal of any bone overlying the tumor effectively turns many tumors of the middle ridge into "upside-down" convexity meningiomas (**Fig. 23.2**). However, large tumors may extend far enough medially to involve the medial sphenoid wing, and surgery for such lesions may not be very much different from operations for medial sphenoid wing meningiomas (**Fig. 23.3**). As with lateral wing meningiomas, the surgeon should aim at a complete removal of the tumor, including any tumor-infiltrated bone and the tumor's dural origin. Involvement of the cavernous sinus or extensive bony involvement and orbital growth ("sphenoorbital meningiomas") may render this impossible (**Fig. 23.7**). In such cases, the surgeon will have to settle for the lesser aim of long-term control of any globular intracranial component, relief and prevention of future compromise of the optic apparatus, and sometimes good cosmesis.

We generally operate meningiomas of the middle sphenoid wing through a pterional craniotomy providing access to the orbital roof and the base of the middle fossa as necessary. The craniotomy has to be large enough to allow for the excision of any dural tumor carpet (en plaque growth; **Figs. 23.4, 23.6,** and **23.9**). A bicoronal incision may be helpful in selected cases if one anticipates the need for very large periosteal flaps, bilateral temporalis muscle fascia, or split calvarial grafts. Any extracranial tumor extensions are excised, and the tumor-infiltrated bone is drilled extradurally. The lesser wing of the sphenoid is removed up to the level of the anterior clinoid process if needed. The base of the middle cranial fossa can be resected up to the level of the foramen ovale, foramen rotundum, and foramen spinosum, providing access to the infratemporal fossa if needed. Entry into the paranasal sinuses should be avoided (**Fig. 23.7**). Several variations of a combined pterional/frontotemporal and orbitozygomatic approach for large middle wing and in particular for sphenoorbital tumors have been described. Orbital and zygomatic osteotomies may be added to a conventional pterional approach, allowing for a more direct exposure of the lateral orbital wall and the middle fossa.[10,14,21] Skull base approaches, however, carry their own approach-related morbidity, and several large series report good neurological, resection, and cosmetic outcomes after surgery for sphenoorbital meningiomas without the routine use of skull base techniques.[22–24]

Some middle ridge tumors may involve the anterior clinoid process and the optic canal. Extradural clinoidectomy and unroofing of the optic canal have been advocated for such cases to prevent additional damage to an already compromised optic apparatus during the intradural dissection.[18] We and others feel that extensive extradural drilling

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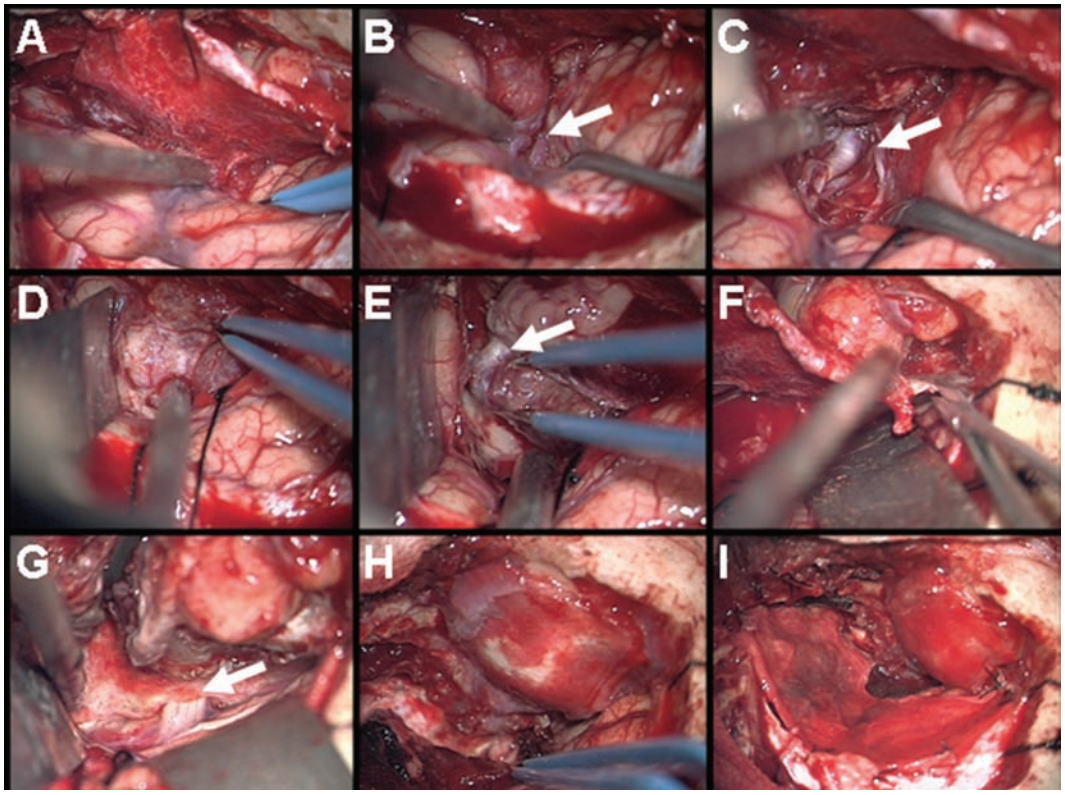


Fig. 23.9 Surgery for a medium-sized meningioma of the left middle sphenoid wing with extensive dural involvement and intraosseous growth (see **Fig. 23.6** for imaging findings and clinical presentation). A pterional craniotomy has been performed with extensive drilling of the sphenoid ridge, and the orbital roof and lateral orbital wall (see **Fig. 23.6B**). **(A)** Carpetlike dural tumor growth is seen after reflection of the dura. The size of the tumor does not require internal debulking, and the borders of the tumor can be dissected in a systematic manner. The large sylvian vessels are identified laterally (*arrows*). **(B–E)** The arachnoid is still largely intact, allowing for a relatively easy dissection. **(F)** Tumor-infiltrated dura and the globular part of tumor are finally removed en bloc. **(G)** This is followed by further resection of the dura and opening of the optic canal with a drill (*arrow*, optic nerve). **(H)** Minimal orbital reconstruction is performed with Gelfoam and fibrin glue. **(I)** A free pericranial graft is used for dural closure. Copyright M. Simon and J. Schramm, reproduced with permission.

may even increase the risk for optic nerve damage due to the retraction necessary for these maneuvers.[11–13,15,16]

The large arteries of the anterior circulation may be engulfed by large tumors, involving the medial sphenoid wing.[12,17] In such cases early visualization of the internal carotid artery is sometimes impossible. As with lateral wing tumors, we start with the vascular dissection laterally (i.e., we identify distal branches of the middle cerebral artery in the sylvian fissure) and follow them medially until one reaches the main trunk of the middle cerebral (and eventually the internal carotid) artery. After the large tumor bulk has been removed, it is often possible to expose the hidden proximal carotid artery from a subfrontal route and to dissect the remaining tumor away from the vessel, alternating between a proximal and a distal/lateral approach (**Fig. 23.9B–E**).[12]

Large tumors may encroach upon the optic apparatus. Developing proper (arachnoidal) dissection planes is very important. Inappropriate retraction, tearing of feeding vessels, and even bipolar coagulation in the vicinity of an already impaired optic nerve or chiasm can result in further deterioration. In general, surgery for recurrent tumors is much more demanding (and carries increased complication rates) due to the absence of arachnoidal planes. This is an important argument for aggressive initial surgery. Of note, the optic apparatus is a radiosensitive structure, and even if the patient will undergo radiosurgery anyway, one of the crucial goals of surgery is to gain as large a distance as possible between any residual tumor and the optic nerve and optic chiasm.

Infiltration of the cavernous sinus is seen in a sizable proportion of large or en plaque middle sphenoid wing meningiomas. The outer layer of the wall of the cavernous sinus can be resected. Intracavernous tumor is nowadays frequently, and many say probably best, left behind. Surgery for cavernous sinus meningiomas has met with limited success. Sindou et al have reported a series of 100 cases with cavernous sinus meningiomas, including 40 patients who had surgery within the cavernous sinus itself. Persistent palsies of cranial nerves III, IV, and VI were seen in 26 to 33%, and trigeminal nerve dysfunction in 31% in this latter subgroup. Only 12 patients had a gross total resection of their tumor.[25] We and others feel that intracavernous tumor should be treated with radiosurgery rather than microsurgery in all but a few patients with ophthalmoplegia.[11–13,15]

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Tumor in the superior orbital fissure, the annulus of Zinn, and the apex of the orbit in general should not be resected aggressively. The periorbita can be stripped if necessary. Outright tumor-infiltrated periorbita can be resected. Opening the periorbita is only necessary in the rare case of nodular intraorbital tumor growth. Tumor growth will usually be restricted to the superior or lateral parts of the orbita. Any intraorbital tumor is removed under meticulous avoidance of the intraorbital nerves and extraocular muscles. It is important to keep the extraocular muscles grossly intact, even if that entails leaving some tumor behind.

The need for orbital reconstruction continues to be debated. Orbital reconstruction is thought to prevent enophthalmos, a pulsating eye, and oculomotor muscle fibrosis. Most would agree that the supraorbital rim margin needs to be preserved (or reconstructed, if violated). Reconstruction of the orbit has been advocated if more than one of its walls has been resected.[14] We and others have reported good clinical results after only minimal cranioorbital reconstruction (e.g., with methylmethacrylate, Gelfoam (Pfizer, Inc., NY), and fibrin glue) (**Fig. 23.9H,I**).[22,23] Of note, all reconstructive procedures may interfere with postoperative surveillance imaging.

Surgical Outcomes

Variable recurrence and complication rates after surgery for sphenoid ridge meningiomas have been reported. This reflects the different growth patterns and degrees of resectability of the various types of sphenoid meningioma, but also different treatment philosophies. As already pointed out, one can argue that surgery for many tumors with extensive bony involvement and cavernous sinus infiltration aims at symptom control and improvement rather than cure. Finally, assessing the degree of resection is not a trivial matter. Current neuroimaging cannot distinguish reliably between Simpson resection grades I, II, and III. Residual dural tumor cannot be reliably delineated by MRI scanning. The extent of the bony resection is best appreciated on postoperative CT scans.

Virtually all globular lateral wing meningiomas, the great majority of globular tumors of the middle sphenoid ridge, and many small to medium-sized medial wing tumors show only limited bone infiltration and no cavernous sinus involvement. Such tumors can be resected completely, including their dural origin, conceptually curing the patient. Recurrence rates should not exceed the figures seen in convexity meningiomas, that is, 5 to 10%.[26] Recurrence rates after operations for medial wing tumors and for hyperostosing/en plaque meningiomas will be higher. Recurrence rates of 9 to 23% have been quoted in the more recent large series of medial wing tumors,[12,13,15,16] and 8 to 31% for sphenoorbital meningiomas.[11,22,23,27]

Surprisingly significant mortality and morbidity rates after meningioma surgery in general have been reported in unselected series. Two recent American studies including 15,028 and 1281 patients, respectively, detail mortality rates of 2.3% and 6%, respectively, and complication rates ranging from 4.5% to 16.5%.[28,29] Complication rates after operations for intracranial meningiomas vary considerably with the location and the size of the tumor and patient-dependent factors such as age and comorbidities.[29] Increasing tumor size correlates with peritumoral edema, pial vascularization of the tumor, and ultimately a failure to maintain an extrapial dissection plane, resulting in cortical damage and sometimes vascular injuries.[30]

Operations for globular tumors of the middle and outer third of the sphenoid wing probably carry complication rates very similar to those for surgery for convexity tumors. Morokoff et al recently reported a surgical series of 163 cases of convexity meningiomas with 0% mortality, new neurological deficits in 1.7%, and an overall complication rate of 9.4%.[31] Complication rates after surgery for medial sphenoid wing meningiomas are higher. Visual deterioration, aphasia, and hemiparesis may occur in up to 5 to 10% of patients. On the other hand, visual dysfunction may improve after surgery in 40 to 85%.[12,13,15,16] Similarly, there is a > 30% chance of visual improvement following operations for sphenoorbital meningiomas. Visual deterioration, new diplopia, and significant trigeminal symptoms may be seen in > 5 to 10% of patients.[7,11,22,23,27] Cosmesis after surgery for sphenoorbital tumors, despite their often extensive bony involvement, is usually quite acceptable. Skull base approaches may carry a higher risk for postoperative temporomandibular joint dysfunction, trigeminal neuralgia/hyperpathia, and adverse cosmetic outcomes.[7]

Radiotherapy

Radiotherapy is increasingly used to control residual tumor after meningioma surgery and to treat unresectable progressive disease. There is no evidence indicating that early radiotherapy is better than deferring radiotherapy until tumor progression. We prefer to follow patients with residual tumor smaller than the critical 30 to 35 mm limit for radiosurgery with serial imaging. Radiosurgery is initiated if the tumor remnant has demonstrably enlarged. In patients with residual tumor > 30 to 35 mm, we start with radiosurgery after the 3-month follow-up MRI scan has been obtained.

Progression-free survival after fractionated radiotherapy is ~90% at 5 years and 80 to 90% at 10 years for benign meningiomas. The corresponding figures for stereotactic radiotherapy and radiosurgery are even slightly better.[32] These numbers reflect both the efficacy of meningioma radiotherapy and the natural history of the disease. The 5-year progression-free survival rate was 60% in a series of 38 patients with petroclival meningiomas who had a subtotal resection and no postoperative radiotherapy.[33]

Tumor size and the vicinity of the optic apparatus often limit radiotherapeutic options for sphenoid wing meningiomas. Only 69% of the patients with sphenoid wing meningiomas reported by Nutting and coworkers

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had no progression at 10 years following mostly subtotal surgery and fractionated radiotherapy. Results for other skull base locations were better, and the authors felt that these differences were most likely due to the on average larger size of the sphenoid meningiomas in their study.[34] Surgical decompression of the optic chiasm and optic nerves may be a prerequisite for successful radiotherapy of sphenocavernous meningiomas. Cavernous sinus disease in general is a good indication for radiosurgery or stereotactic radiotherapy.[32,35] Data exist that suggest that radiotherapy may help to control residual intraorbital meningioma.[27]

Radiosurgery and stereotactic radiotherapy are important additions to the neurosurgical armamentarium. However, aggressive surgery is still appropriate in many cases. A conservative operation followed by radiotherapy for a challenging tumor may result in a patient 10 years older with a progressive and unresectable tumor, and few remaining therapeutic options. The risk of the formation of radiation-induced cancers may be an underappreciated concern, especially in younger patients.

◆ Summary and Outlook

The surgical management of many meningiomas of the lateral and middle sphenoid wing is not very different from that of convexity tumors. Surgery will often cure the patient. However, in other cases, extensive intraosseous growth and extension into the orbit and cavernous sinus will preclude a truly complete resection. Surgical objectives in such cases include relief of significant mass effect, decompression of the optic apparatus, and correcting a cosmetic deformity or exophthalmos.

Radiotherapy and radiosurgery play an increasing role in controlling residual or progressive disease. Even though virtually all patients with sphenoid meningiomas will still have to undergo surgery, the ability to follow asymptomatic residual tumor with serial neuroimaging, and the availability of efficient radiotherapeutic treatment have shifted the focus from curing sphenoid meningiomas toward symptom and growth control.

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Chapter 24

Clinoidal Meningiomas

Ali F. Krisht

Introduction

Clinoidal meningiomas are best described as meningiomas arising in the vicinity of the anterior clinoid process. The typical clinoidal meningioma seen on magnetic resonance imaging (MRI) looks as if it were mushrooming out from the anterior clinoid process (**Fig. 24.1**). Some, however, may grow into the region of the mesial sphenoid wing and be confused with medial sphenoid wing meningiomas (**Fig. 24.2**). When clinoidal meningiomas grow to a very large size they can involve the parasellar region in its anteroposterior and mediolateral extensions. In this case the tumor may extend laterally into the cavernous sinus or posteriorly into the region of the posterior clinoid and the petroclival region (**Fig. 24.3**).

Anatomical Considerations

The anterior clinoid process is a complex anatomical entity that has several neurovascular structures passing in its immediate vicinity.[1] The oculomotor nerve course is along the superolateral aspect of the anterior clinoid process. The internal carotid artery crosses the inferior aspect of the anterior clinoid process, and the optic nerve passes along its superomedial aspect (**Fig. 24.4**). Based on surgical findings and observations regarding the site of origin (**Fig. 24.5**) and the adhesiveness of the tumor to the internal carotid artery and its branches, Al-Mefty suggested that clinoidal meningiomas be classified into three different types.[2] Type I clinoidal meningiomas are thought to arise from the subclinoidal dura at the most proximal point of intradural entry of the internal carotid artery, just before the carotid enters into the arachnoidal cisternal space. As a result, Al-Mefty suggested that these tumors are extraarachnoidal and because of this tend to become more adherent to the internal carotid artery and much more difficult to remove surgically. These are the tumors that have a higher rate of subtotal resection and recurrence.

Type II clinoidal meningiomas are thought to originate from the superolateral aspect of the anterior clinoid process. When these tumors grow, they are invested by the arachnoid layers around the carotid cistern. As a result, the tumor is separated from the internal carotid wall by arachnoidal layers that prevent the significant adherence of the tumor to the adventitia of the internal carotid artery wall. This makes the tumors easier to dissect off the wall of the internal carotid artery; as a result, a more complete resection is achievable. Al-Mefty also makes the observation that the growth of type I and II clinoidal meningiomas starts at a distance from the optic nerve; as a result, the arachnoid membranes of the chiasmatic cistern invest the optic nerve and help protect it from immediate invasion by the tumor.

Type III clinoidal meningiomas originate from the region of the optic foramen and extend into the optic canal. Because of the pattern of their growth within the region of the optic canal, these tumors become symptomatic at an early stage and are diagnosed earlier before they achieve a large size, unlike types I and II.

Clinical Presentation

The most common clinical presentation of clinoidal meningiomas consists of visual disturbances and headaches.[2–14] Other clinical symptoms usually correlate with the size of the tumor and its extension. Tumors that achieve a large size with more lateral extension result in symptoms due to compression of the parasellar structures. Extension of the tumor into the cavernous sinus region may result in associated cranial neuropathies.[15–20] Occasionally some of the tumors extend to the middle temporal fossa and may

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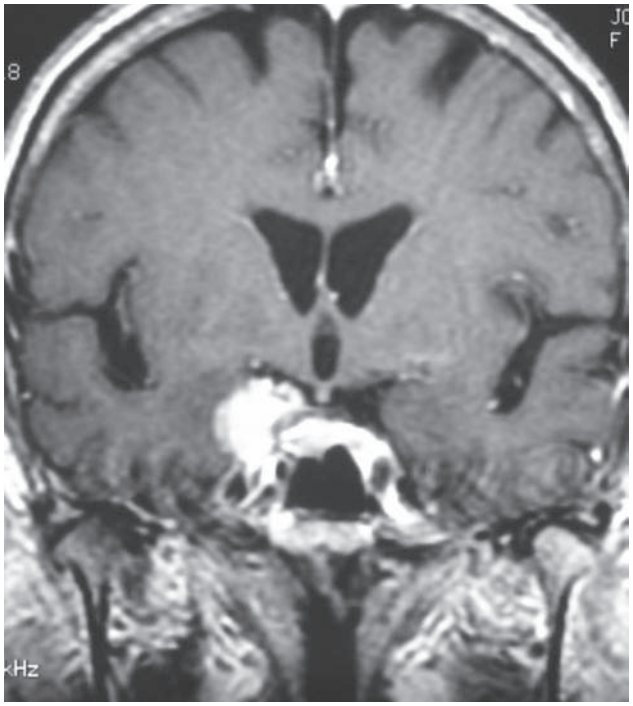
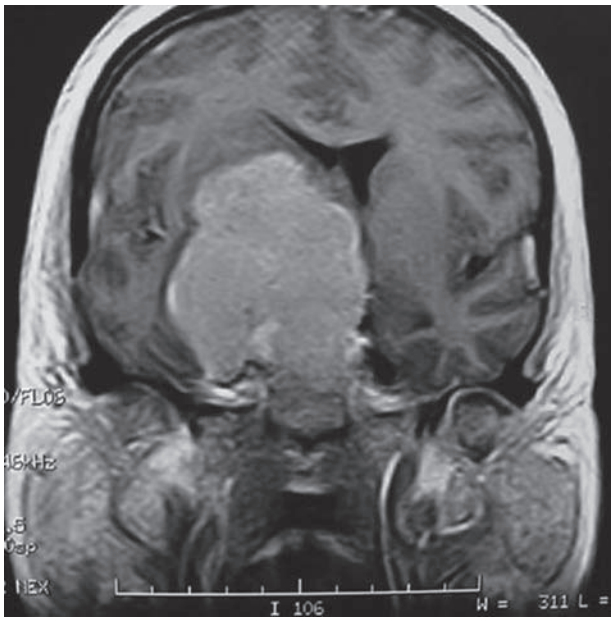


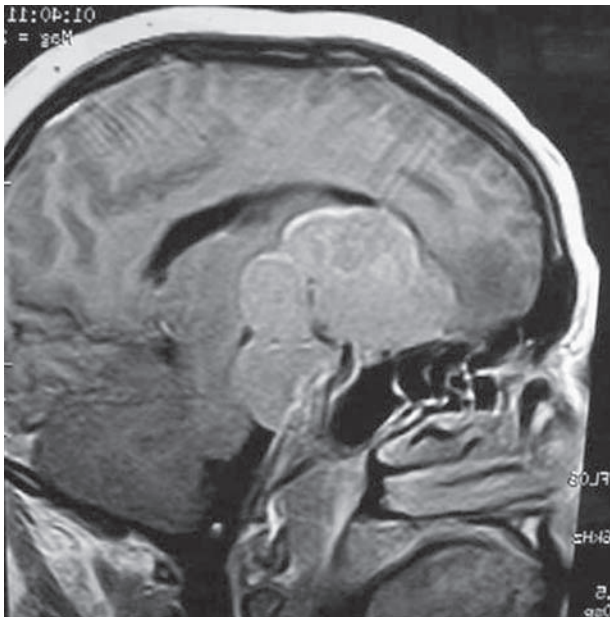
Fig. 24.1 T1-weighted coronal magnetic resonance imaging with contrast showing a typical clinoidal meningioma mushrooming out from the right anterior clinoid process.



Fig. 24.2 T1-weighted coronal magnetic resonance imaging showing a left clinoidal meningioma with parasellar extension to the medial sphenoid wing region.



A



B

Fig. 24.3 **(A)** Coronal and **(B)** sagittal T1-weighted magnetic resonance imaging of a giant type I clinoidal meningioma engulfing the right internal carotid artery and its branches. *(continued)*

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C

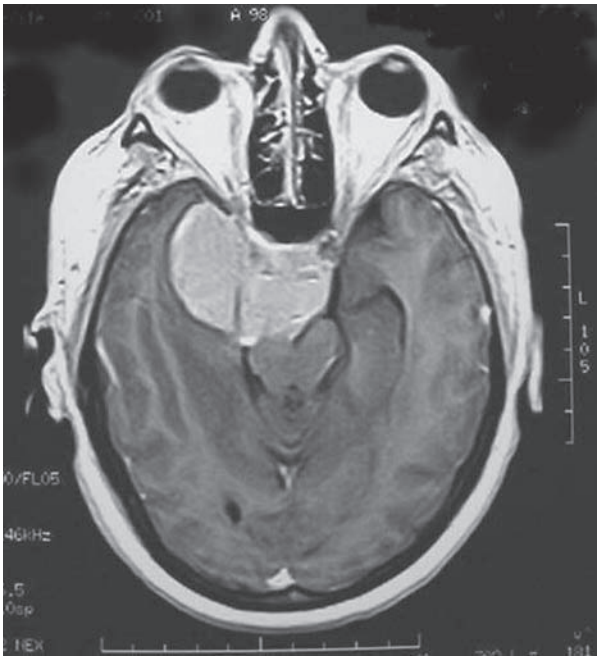


Fig. 24.3 *(continued)* **(C)** Axial T1-weighted magnetic resonance imaging of a giant type I clinoidal meningioma engulfing the right internal carotid artery and its branches.

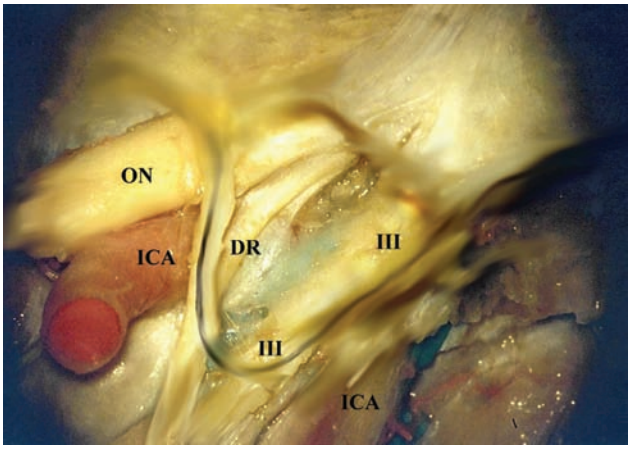


Fig. 24.4 Anatomical dissection of the anterior clinoid process and the different neurovascular entities within the wall of the anterior clinoid process. ON, optic nerve; DR, dural ring; ICA, internal carotid artery; III, oculomotor nerve.

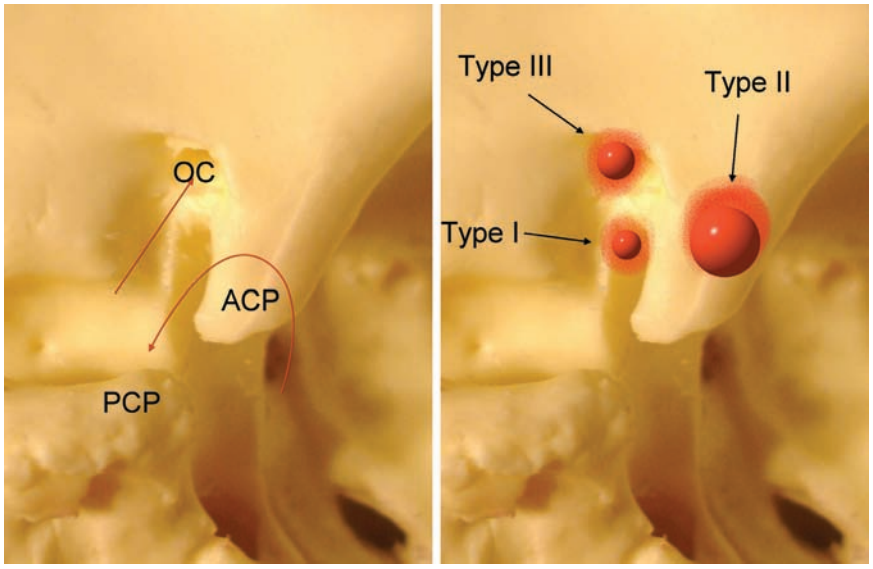


Fig. 24.5 Bony anatomy of the anterior clinoid process region. (Left) The course of the optic nerve (*straight arrow*) and the course of the internal carotid artery (*curved arrow*). OC, optic canal; ACP, anterior clinoid process; PCP, posterior clinoid process. (Right) The origin of the different types of clinoidal meningiomas.

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enlarge posteriorly to compress the temporal lobe, resulting in seizures, or may even compress the brain stem, resulting in hemiparesis. Some patients with associated significant hyperostosis of the sphenoid wing may present with proptosis and orbital pain.

As with all meningiomas, clinoidal meningiomas are more common in females. However, the ratio of females to males is larger in clinoidal meningiomas compared with other meningiomas.

Radiological Evaluation

Traditionally, a computed tomographic (CT) scan of the head with evidence of hyperostosis involving the anterior clinoid process was strongly suggestive of a clinoidal meningioma (**Fig. 24.6**). Nowadays clinoidal meningiomas are best evaluated with an MRI scan of the brain (**Fig. 24.7**). Thin-cut MRI with fat suppression through the optic canal can help identify the presence of tumor extension into the orbit. Four-vessel cerebral angiography was also typically part of the workup of patients with clinoidal meningiomas (**Fig. 24.8**). Today, magnetic resonance angiography (MRA), or in some cases CT angiography, is as informative and provides the needed information about the relationship of the tumor to the supraclinoid internal carotid artery and its branches. However, in recurrent cases or in patients who have had previous radiation, four-vessel cerebral angiography with balloon test occlusion may be necessary to have a plan in place in case of injury to the carotid artery.

Treatment

Surgery remains the most effective treatment modality for clinoidal meningiomas. This is true even for small lesions that may be considered for radiosurgery in other locations, because of their proximity to the optic apparatus and the higher possibility of radiation injury.

Surgery on clinoidal meninigomas demands a full understanding of the bony as well as the neurovascular anatomy of the sellar and parasellar region. A full understanding of the three-dimensional aspect of the anterior clinoid process and the adjacent neurovascular structures is of utmost importance. In addition, the knowledge of the vascular anatomy and potential normal anatomical variations is essential for a safe resection of these tumors.

Surgical Steps

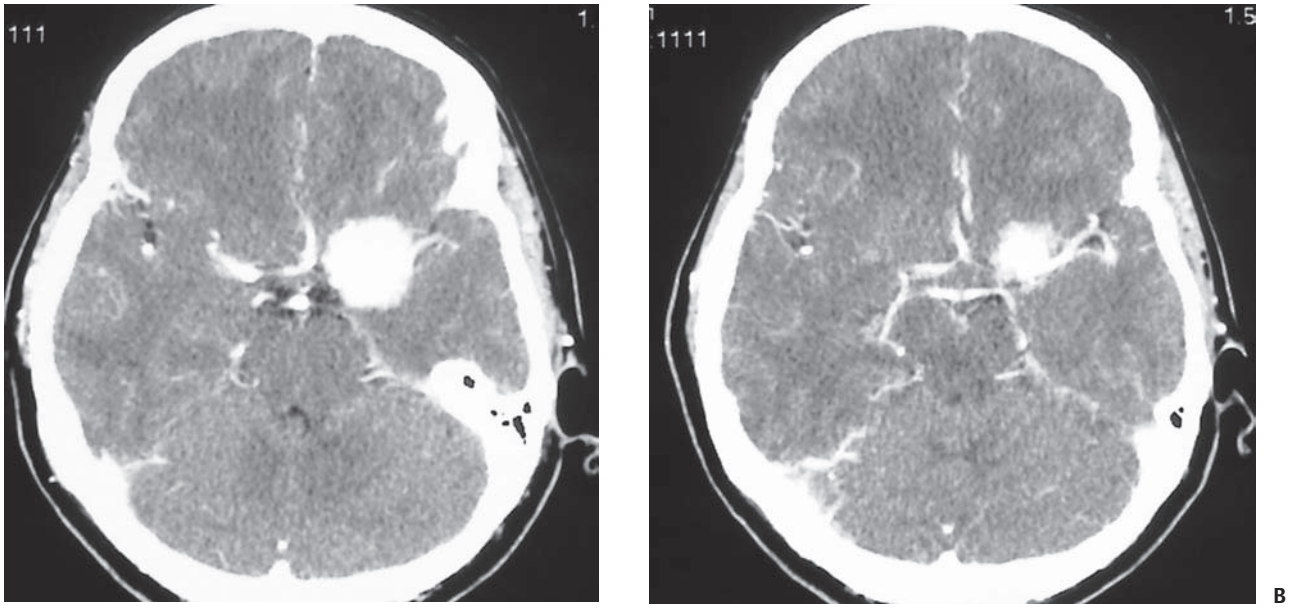
Removal of clinoidal meningioma is best achieved with a wide frontoorbital craniotomy. Other names used for this craniotomy are extended pterional approach or extended pretemporal approach. In addition the craniotomy should include a very low threshold to include the orbital roof in the craniotomy flap (cranioorbitozygomatic approach) to be able to access the suprasellar extension of these tumors without the need of excessive frontal lobe retraction. In the overall majority of our cases, we are able to avoid or minimize the use of self-retaining retractors.

Once the craniotomy is established, we proceed with the extradural work that leads to the removal of the anterior clinoid process. The steps include dissection of the pretemporal and subfrontal dura away from the sphenoid wing, which will be removed with the posterior roof of the orbit. Then the dural fold at the level of the meningioorbital artery is cut to allow disconnection of the dura propria of the temporal lobe from its attachment to the frontal dura across the sphenoid wing over anterior clinoid process. This will bring the anterior clinoid process to a more superficial point. This process of exposing the anterior clinoid process in and of itself leads to significant devascularization of the tumor, which gets its blood supply from anterior dural branches of the middle meningeal artery as well as the meningioorbital artery. It also gets blood supply from posterior ethmoidal branches, which normally provide blood supply to the clinoidal and frontal fossa dura. In addition to tumor devascularization, this extradural work will help expose the clinoidal internal carotid artery segment. The exposure of the clinoidal internal carotid artery and later exposure of the sylvian fissure arterial branches will provide the two normal ends of the vascular tree, which can then be dissected proximally and distally as the tumor is being resected. This provides a good road map for the course of the internal carotid artery as it merges with the middle cerebral artery and anterior cerebral artery at the level of the carotid bifurcation.

Once the extradural work is done, the intradural exposure is performed by opening the dura and visualizing the suprasellar and posterior borders of the tumor. The sylvian fissure is routinely opened from the level of the limen insulae toward its proximal point to help visualize the whole course of the middle cerebral artery. Once this is achieved, debulking of the tumor is started with intermittent dissection of the tumor away from the adjacent brain and blood vessels. Once adequate debulking is achieved and the course of the different arterial branches is visualized, final tumor removal is performed. The dura at the base of the skull and over the clinoid region is totally removed. The underlying bone, which has partially been removed during the extradural removal of the anterior clinoid process, is further drilled and resected with a high-speed diamond drill and copious irrigation aiming at as complete a resection as possible and at the same time achieving bone hemostasis.

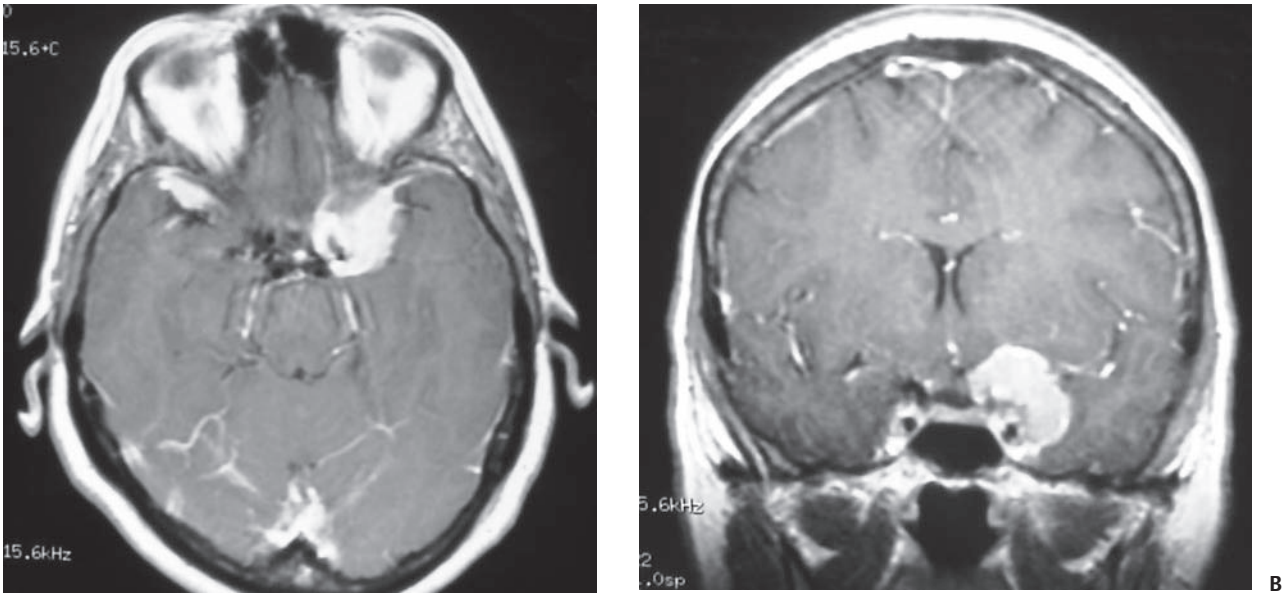
As part of the process of removing the anterior clinoid, the optic canal is decompressed by removing the optic strut and the optic roof. The dural layer over the optic canal is opened along the optic nerve. This allows access to tumor extending into the optic canal. Even though this is more common in type III tumors, we perform this step in all tumor types. The tumor is followed along its extension into the orbit. Extra caution is taken while dissecting the optic nerve to preserve the very small blood vessels providing blood supply to the optic canal from the superior hypophyseal branches. It is also important to pay attention to the fact that occasionally direct arterial supply from the ophthalmic artery close to its origin may inad-

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A B

Fig. 24.6 **(A,B)** Axial computed tomographic scans of a typical clinoidal meningioma.



A B

Fig. 24.7 **(A)** Axial and **(B)** coronal gadolinium T1-weighted magnetic resonance imaging of the clinoidal meningioma shown in **Fig. 24.6**, showing the epicenter of the tumor at the level of the left anterior clinoid process.

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Fig. 24.8 An internal carotid artery angiogram showing the arterial blush of the clinoidal meningioma shown in **Fig. 24.3**. Notice the extensive recruitment of several branches of the proximal internal carotid artery to supply the tumor. There are also ethmoidal branches arising from the ophthalmic artery.

vertently be injured. This can best be avoided by careful dissection with magnification and with the anatomical knowledge of those vessels.

In tumors that extend further laterally, the dissection of the temporal dura away from the lateral wall of the cavernous sinus may be extended to a more posterior point to remove tumor extending into the region of the cavernous sinus. In tumors with extensions toward the suprasellar and subchiasmatic regions, it is important that the dural ring around the internal carotid artery be dissected to widen the space between the internal carotid artery and the optic nerve to gain access to the tumor segments extending into the subchiasmatic and possibly retrochiasmatic region. Occasionally these tumors extend more posteriorly along the tentorial edge and compress the third nerve inferiorly. The third nerve can be identified extradurally at the level of the superior orbital fissure and followed from its extradural to its intradural segments under full visualization to mobilize and avoid its injury during tumor resection.

Gross total resection should always be the goal of surgery (**Fig. 24.9**). However, in type I tumors where there is no arachnoid layer to separate the tumor from the wall of the internal carotid and its branches, good judgment is exercised to decide the extent of tumor resection to avoid injury to adherent vessels and to avoid additional morbidity (**Fig. 24.10**).

Once the appropriate degree of resection is achieved, the reconstruction of the skull base is carefully attended to. The dural defect is reconstructed with dural replacement materials. In our experience, the best tissue to be used is either fascia lata or pericranium. This is reinforced with subcutaneous fat harvested from an abdominal incision to be used for obliteration of the clinoidal space, especially if there is communication with the sphenoid sinus. The closure is reinforced with fibrin glue. We avoid the use of lumbar drains because adequate closure rarely leads to a spinal fluid leakage.

Postoperative Care

In the postoperative period patients spend at least a night in the intensive care unit for close observation. They are usually started on a moderate amount of fluids running at around 80 mL an hour of half normal saline. The next day they are started on po diet and gradually increased as tolerated. They are given Dilantin (Pfizer, Inc., New York, NY) for seizure prophylaxis and 100 mg of hydrocortisone every 8 hours to be tapered by the time of discharge. We routinely obtain a postoperative MRI scan within the first 24 hours. Patients in a stable condition are transferred to a regular bed floor and their activities are gradually increased. Most

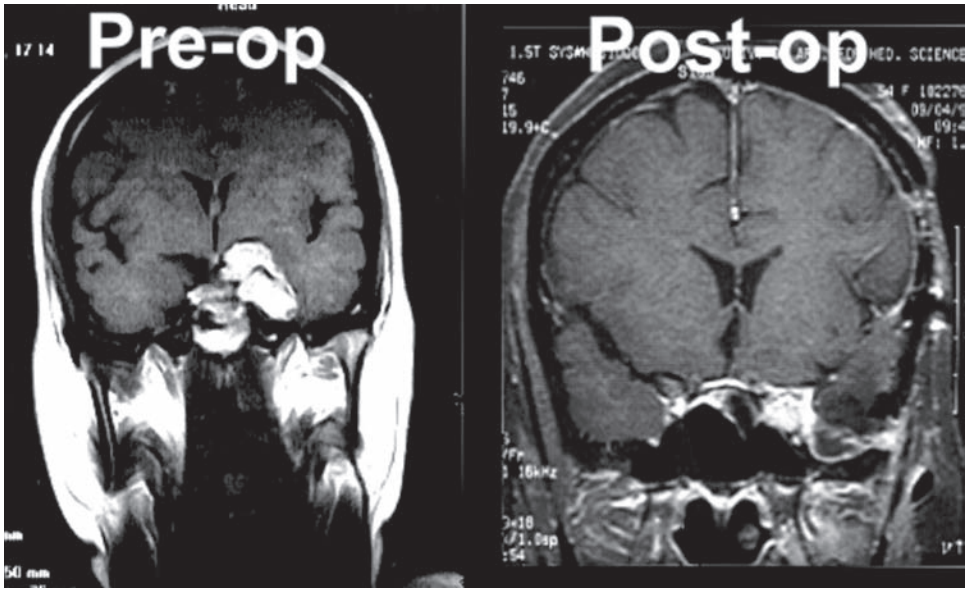


Fig. 24.9 Preop and postop coronal magnetic resonance imaging of a completely resected clinoidal meningioma.

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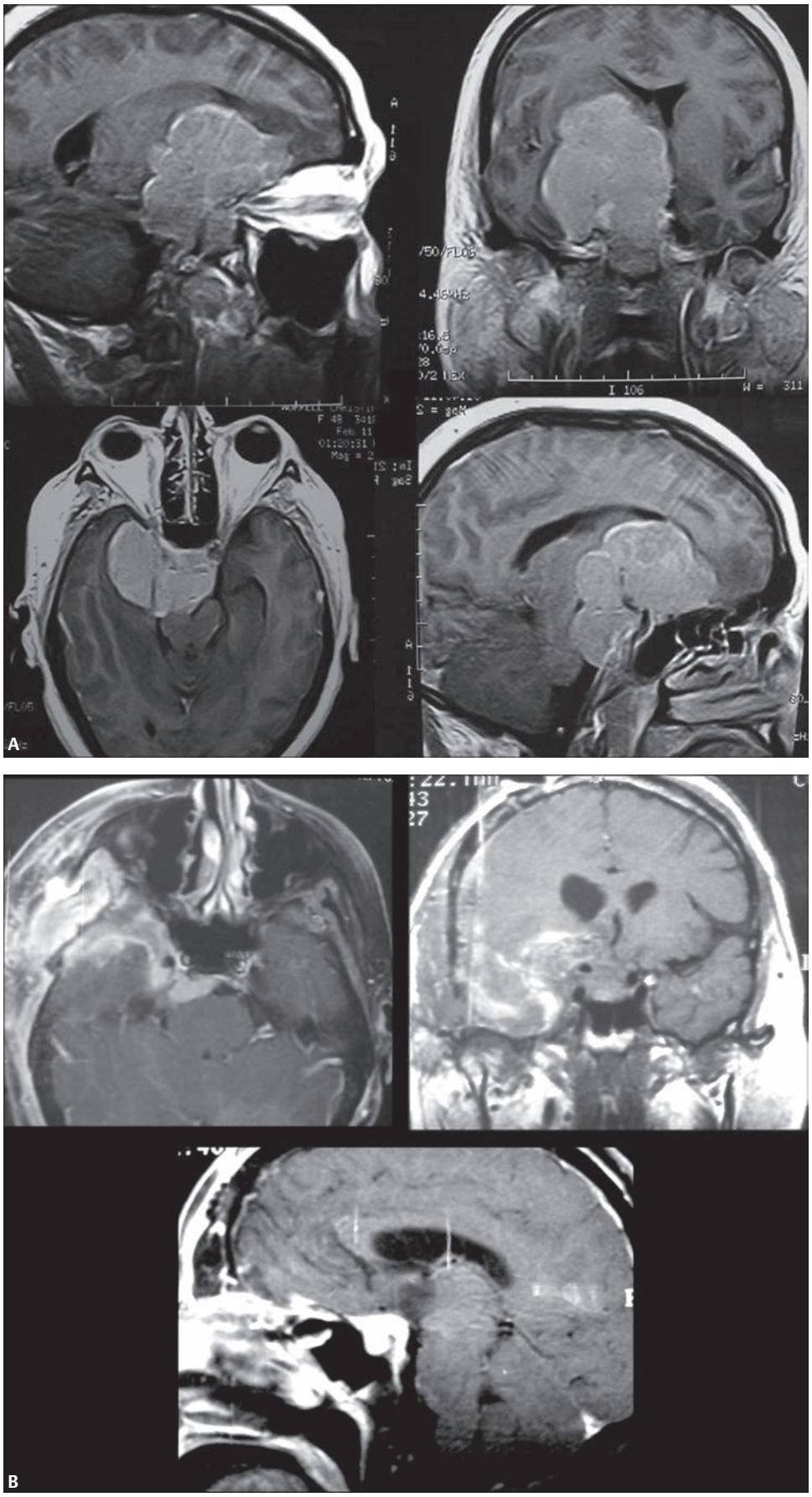


Fig. 24.10 **(A)** Preop and **(B)** postop magnetic resonance imaging (MRI) of a giant clinoidal type I meningioma after radical subtotal resection. Pieces of the tumor adherent to the lenticulostriate arteries in the region of the anterior perforated substance were left (coronal MRI). A small piece was also left in the region of the entry site of the abducens nerve (axial cut).

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Table 24.1 Anterior Clinoidal Meningioma Resection: The Peer-Reviewed Literature

Series	No. of Patients	Mortality	% CS Involvement	% Improvement of Vision	Extent of Resection—%	% Estimated Major Morbidity (gleaned from article)	Mean Follow-Up	Recurrence/ Progression		
								% Over all	% From total	% Rest
Al-Mefty 1990[2]	24	8%	38	8	Gross total—83	8	57 months	4		
Puzzilli et al, 1999[12]	33	15%	39.4		Gross total— 54.5	12	53.7 months	26	0	42
Day, 2000[7]	6	0	100		Gross total—66.6	0	3 months	0		
Goel et al, 2000[8]	60	5%		23	Gross total—70	12	26 months	2		
Lee at al, 2001[9]	14	0	14	75	Gross total—87	0	37.2 months	0		
Tobias et al, 2003[14]	26 *	0	23	71	Gross total—77	11.5	42.3 months	0		
Nakamura et al, 2006[19]	108	0	64		Gross total—43	7.4	6.59 years	23		
Pamir et al, 2008[11]	43	0	2	85	Gross total—91	18.6	39 months	9.3		
Russell et al, 2008[13]	35	0	31	63	Gross total—69	18	12.8 years	9		
Bassiouni et al, 2009[6]	106	1.9%	29	40	Simpson grade I–II—59	17	6.9 years		10	38

* 25 meningiomas, one hemangiopericytoma.

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patients are discharged within 2 to 3 days. Deep vein thrombosis prophylaxis using ted hose, sequential devices, and subcutaneous anticoagulants are essential in all patients with meningiomas due to their predisposition to deep vein thrombosis and its potential for pulmonary embolism.

Outcomes

The extent of resection of clinoidal meningiomas varies in different series. The rates of total resection vary between 43 and 91% (**Table 24.1**).[2,6–14] This is most likely related to differences in patient mix as well as type and size of the tumor. This is also true for the reported rates of mortality and morbidity. **Table 24.1** shows the different reported series of anterior clinoidal meningiomas. It is noticeable that the mortality is higher in Al-Mefty's and Puzzilli et al's series (**Table 24.1**).[2,6–14] However, their patient mix had a higher rate of involvement of the cavernous sinus. Also, Al-Mefty's referral pattern has a higher percentage of recurrent or previously radiated tumors.

Among the most common morbidities after surgery of clinoidal meningiomas are those related to visual deterioration. The most common etiology is ischemic injury due to compromise of the blood supply to the optic apparatus. These complications are generally avoidable with a good knowledge of the normal as well as the pathological anatomy of these tumors. Ischemic complications resulting in major morbidities should be avoidable and usually occur at a very low rate after surgery of clinoidal meningiomas. A judicial approach to these tumors with a clear understanding of the pathological anatomy should help avoid such complications. For example, in patients with type I tumors where significant adhesion between the tumor and the vasculature is encountered, the decision to leave some tumor behind to preserve function is a more appropriate decision than attempting gross radical resection and increasing the risk of arterial injury and ischemia. Tumor recurrence or progression, similar to all other meningiomas, is related to the extent of tumor resection during the first surgery.[21–25] **Table 24.1** shows the reported tumor recurrence and progression rates. There is clear dependence on the extent of tumor resection as well as the length of follow-up. For this reason it is important to follow all patients with meningiomas for no less than a 10-year period after tumor resection before the patient is permanently discharged. There are some studies that suggest even continuing the follow-up up to 20 to 25 years after tumor resection.

Postoperative spinal fluid leakage is rare in our experience; however, it is not an uncommon complication and needs to be addressed by a good skull base reconstruction as mentioned earlier. In the presence of a satisfactory reconstructive repair recurrence of spinal fluid leakage can be addressed with lumbar drainage for 3 to 4 days. If the cerebrospinal fluid leakage persists and is from the wound then reexploration is recommended. If the leak is through the air sinus and resulting in rhinorrhea then the repair is decided based on the surgeon's judgment to either be done by reexploring the wound or through an endonasal–transsphenoidal approach.

In general, anterior clinoidal meningiomas are best treated with surgical resection, and, in the presence of advances in microsurgical technique, the majority of patients should enjoy a favorable long-term outcome.

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Chapter 25

Cavernous Sinus Meningiomas

Ian F. Dunn and Ossama Al-Mefty

Introduction

Cavernous sinus meningiomas have an estimated incidence of only 0.5 per 100,000.[1] Despite this relative rarity, their management has incited a disproportionate degree of controversy, with the precise role for surgical resection often debated, initially due to some degree of unfamiliarity with intracavernous surgery and its possibilities.

Indeed, the development, refinement, and careful application of skull base approaches have antiquated the notion of the cavernous sinus as a surgical "no-man's land." Initial seminal reports described novel surgical routes to vascular lesions, such as cavernous-carotid fistulae and intracavernous aneurysms.[2,3] These were followed shortly thereafter by descriptions of surgical approaches to cavernous sinus tumors.[3–5]

Since then, accumulating experience with meningiomas of this region has established surgery as a technically feasible and clinically durable method of managing these tumors in patients in whom tumor control by total removal is desired. This chapter reviews the clinical presentation, radiographic features, and treatment principles of cavernous sinus meningiomas.

Clinical Presentation and Physical Examination

Meningiomas may involve the cavernous sinus either primarily or secondarily. Those originating from within the cavernous sinus proper may extend to the Meckel cave, medially to the sella, and to the anterior, middle, or infratemporal fossae. Clinoidal, medial sphenoid wing, and petroclival meningiomas may extend to the cavernous sinus secondarily.

Patients with tumors in the cavernous sinus may present with symptoms referable to compression or congestion of anatomical structures in or near the cavernous sinus. Proptosis, headache, facial pain or numbness, and disturbances of ocular function or motility (diplopia, ptosis, anisocoria, complete ophthalmoplegia) are common. Tumors can compress the optic nerve, with resultant visual field deficits. Cavernous carotid artery compression may result in ischemic deficits. Less commonly, patients may present with pituitary dysfunction.

Physical examination should include a thorough neurological exam, with particular attention paid to the function of cranial nerves II through VI, including a formal visual field assessment. Examination of coordination and motor, sensory, and cerebellar functions assists with assessment of any tumor extension into the posterior fossa with brain stem compression.

In addition to a standard history and physical exam, it is critical to clarify the nature of any prior surgery or radiation in this region given the enormous implications of these factors for future surgical intervention. Scarring from prior surgery or cranial radiation may compromise the integrity of the carotid arterial wall and increase the risk of carotid injury during surgery. Ascertaining any possible systemic disease is important for surgical decision making because other cancers, such as lymphoma, breast carcinoma, and paranasal sinus cancers, may metastasize to the dura and mimic meningioma.[6,7]

Imaging

Magnetic resonance imaging (MRI) is the imaging modality of choice in the evaluation of cavernous sinus tumors and surrounding structures. Meningiomas are typically dural-based masses that are isointense on T1, variably intense on T2, and avidly enhancing (**Fig. 25.1A,B**). The consistency of

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the tumor may be signaled by the intensity of the lesion on T2, with greater T2 signal suggesting a higher water content and, hence, a softer tumor. More recently, some groups have loosely correlated T2 hyperintensity with rate of growth.[8–11] Calcifications may be seen as hypointense regions within the tumor and may portend a more indolent tumor. Computed tomography (CT) is also important because it displays the presence of any associated hyperostosis (**Fig. 25.1C**). High-quality studies are essential to reveal important additional anatomical relationships that inform treatment choice. These include proximity to the optic nerve, extension into the optic canal, and involvement of the anterior clinoid by hyperostosis.

Magnetic resonance angiography (MRA) generally reveals the intracranial circulation without the risks of conventional angiography (**Fig. 25.1D**) and is important in delineating the caliber of the carotid artery. Conventional angiography with cross-compression should, however, be considered if there is any question about the integrity of the ipsilateral carotid artery or the intracranial circulation in general, or in a patient who has received prior irradiation to the cavernous sinus. Thorough study is recommended to identify patients in whom the carotid artery is at excess risk. In these selected cases, a balloon test occlusion (BTO) with single-photon emission computed tomography and acetazolamide challenge may be considered to assess the collateral circulation. Onset of symptoms during BTO, suggesting the inability of the posterior circulation or contralateral carotid to compensate, may lead the surgeon to consider a less aggressive resection or, conversely, to consider carotid bypass as part of the surgical strategy.

◆ Treatment Options and Indications

Natural History

The outcome of any treatment for cavernous sinus meningiomas must be weighed against the natural history of these tumors. In most series, the natural behavior of cavernous sinus meningiomas is inferred from the observation of meningiomas in other locations because few natural history series include substantial numbers of cavernous sinus tumors. In a retrospective review of 40 patients with skull base meningiomas observed for a mean period of 7 years, 27% of patients worsened clinically,[12] similar to a separate series of 24 patients followed for a mean of 57 months in which 25% worsened neurologically.[13] Growth rates and radiographic progression are variably reported. Among seven patients with cavernous sinus meningiomas followed from 6 months to 8 years, predicted radiographic doubling times ranged from 1.7 to 49.6 years with a mean of 7.6 years in 6/7 patients.[9] Other representative series report annual growth rates of 3.6%[14] and radiographic enlargement in 20% (over 57 months) and 32% of patients over 38 months.[8] Taken together, reported growth rates, estimated radiographic doubling time, and clinical and radiographic progression, although reported with variable consistency in the literature, suggest that over 60% of patients may harbor quiescent tumors. Other factors drawn from these studies portending a more benign course include tumor calcification, older age, and T2 hypointensity. Asymptomatic patients or minimally symptomatic patients with cranial nerve involvement may thus be managed conservatively, but the reported rates of clinical worsening and radiographic progression in some series mandate close clinical follow-up at a minimum of yearly intervals.

Treatment Options

Intervention is considered in patients whose tumors demonstrate growth on serial imaging or who become progressively more symptomatic. Once this decision is made, one must clarify the goals of treatment because these differ widely among treatment options. Current interventional options include surgery, with complete tumor resection as the primary goal; radiotherapy, with tumor growth control rather than cytoreduction the principal aim; and emerging paradigms wherein intentionally incomplete surgical resection or decompression is coupled with adjuvant radiosurgery, with the stated aim of safe cytoreduction and postoperative growth control of residual tumor. Not all patients, however, may be treated equally; cavernous sinus tumors causing visual loss are best treated with surgical resection. The goal of treatment in our center when achievable is complete tumor resection, accomplished by the application of microsurgical skull base approaches.

Surgery

Surgery: General Principles Technical advances in skull base surgery have established the cavernous sinus as an approachable space whose contents may be navigated successfully during meningioma surgery. Core principles of these approaches include maximal bone removal for exposure; the avoidance of brain retraction; and control of the carotid artery in its petrous, cavernous, and clinoid segments. During tumor resection, arachnoid planes facilitate tumor removal, but these planes become scarred and obliterated after prior resection or irradiation, emphasizing the importance of extensive resection during the initial operation. If necessary, when no arachnoidal plane is encountered, small remnants of adherent tumor are left on the carotid artery. The same philosophy is true regarding the cranial nerves. Attempts should be made to remove all affected dura, and any affected dura that cannot be resected should be electrocoagulated, if possible. All involved or hyperostotic underlying bone should be drilled away. Any submucosal tumor spread within the paranasal sinuses should be removed. Sphenoid sinus defects must be repaired with extreme care during the reconstruction.

Surgical Approaches Two primary surgical approaches to the cavernous sinus are used by the authors: the cranioorbitozygomatic (COZ) approach, and the zygomatic

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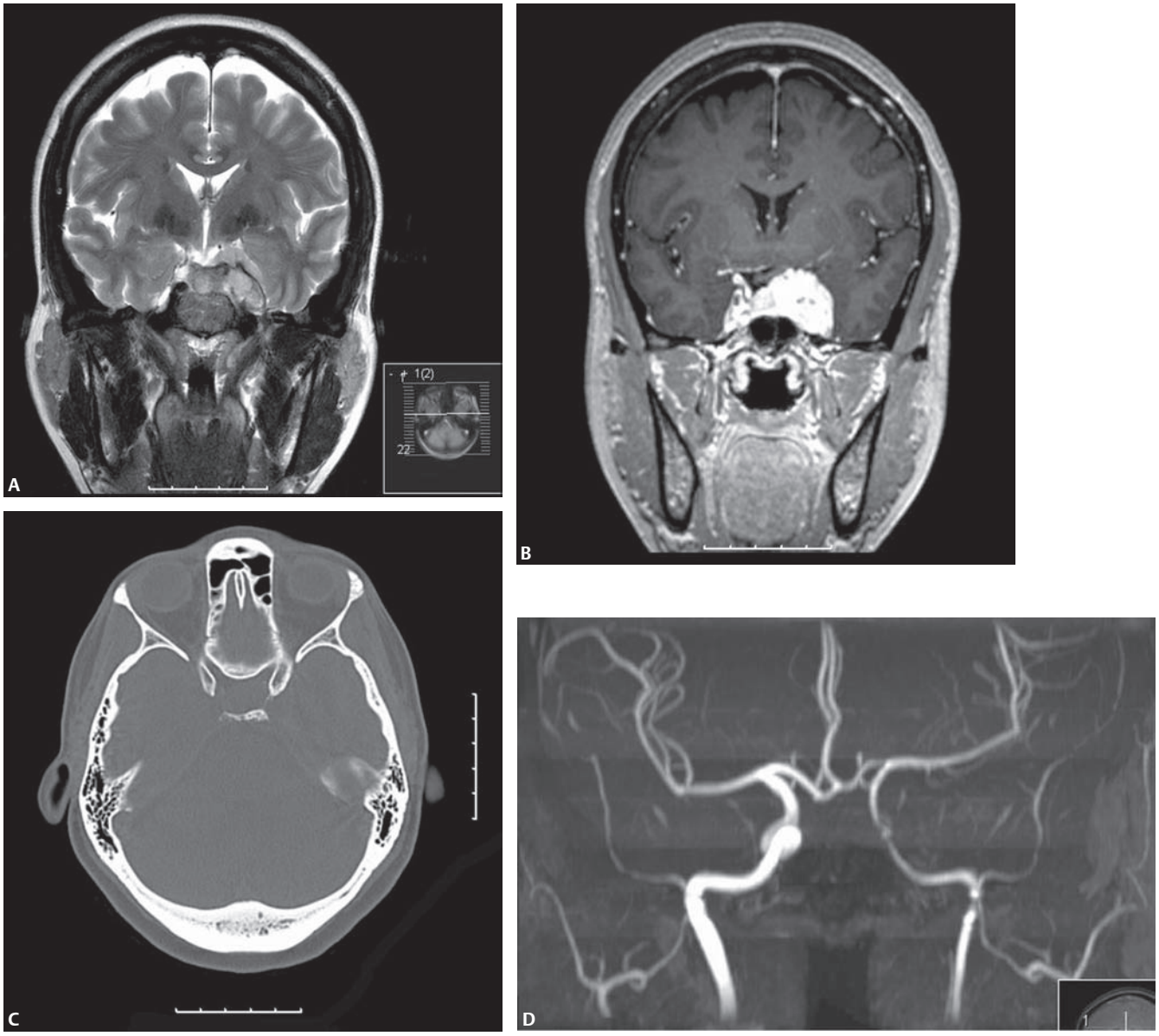


Fig. 25.1 Patient with tumor originating in the cavernous sinus. **(A)** T2 coronal magnetic resonance imaging (MRI) showing a hyperintense mass in the left cavernous sinus pushing the lateral dural margin of the cavernous sinus outward (*dark band*). **(B)** T1 coronal postcontrast MRI showing enhancing mass in left cavernous sinus. **(C)** Axial computed tomography showing hyperostosis of the left anterior clinoid process. **(D)** Magnetic resonance angiography showing narrowing of the left internal carotid artery.

approach.[15] The COZ approach affords wide exposure of the entire cavernous sinus, including the proximal and distal carotid artery, with minimal cerebral retraction. The zygomatic approach is used for tumors in the posterior cavernous sinus and petrous apex. This approach is more limited, does not offer readily obtainable distal carotid control, and does not expose the medial or superior cavernous sinus as easily as does the COZ approach.

Cranioorbitozygomatic Approach A lumbar drain is placed for cerebrospinal fluid (CSF) drainage, and the patient is positioned supine with the upper body slightly elevated and the head rotated 30 degrees to the contralateral side. Leads to monitor somatosensory evoked potentials, brain stem auditory evoked potentials, and cranial nerves V and VII are placed at this time, with those to monitor III, IV, and VI placed subsequently (to be discussed). The ipsilateral neck is prepared should more proximal carotid control be required, and the abdomen is prepared for fat graft harvest.

The skin incision for the COZ starts at the zygomatic root and is carried behind the hairline toward the contralateral superior temporal line (**Fig. 25.2**). The superficial temporal artery is identified and carefully protected, and a large pericranial flap is raised by undermining the scalp posterior to the incision and dissecting sharply against the scalp flap anteriorly. A subfascial dissection of the temporalis fascia is performed to preserve the frontal

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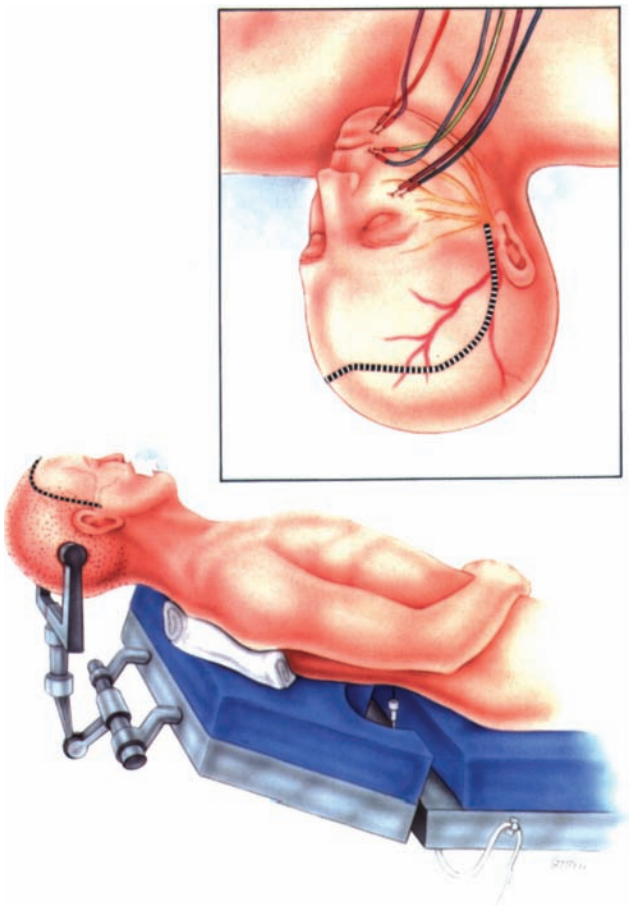


Fig. 25.2 Operative positioning for the cranioorbitozygomatic approach. Main illustration shows head and body positioning, Mayfield pin placement, and lumbar drainage. Inset shows proposed scalp incision and monitoring electrode placement. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

branches of the facial nerve (**Fig. 25.3**). The zygomatic arch and superior and lateral orbital margins are exposed by subperiosteal dissection, after which the zygoma is divided at either end and displaced inferiorly on its masseteric pedicle (**Fig. 25.4**). The temporalis muscle is then elevated in subperiosteal fashion, beginning low on the temporal squama and proceeding superiorly to detach the muscle at the superior temporal line (**Fig. 25.4**). The entire temporalis muscle is then reflected inferiorly with the freed zygoma.

The superior and lateral orbital rims are dissected free from the periorbita, with the supraorbital nerve and vessels preserved (**Fig. 25.5**). A burr hole is placed in the keyhole to gain simultaneous entrance into the cranium and orbit. Burr holes are then placed anteriorly and posteriorly, adjacent to the temporal floor. A cut is made from the medial aspect of the lateral orbital wall to its lateral aspect and is continued to the keyhole. The keyhole is then connected to the posterior burr hole by cutting through the temporal fossa. A cut starting at this burr hole is then brought superiorly to the frontal bone, then anteriorly through the supraorbital rim, taking care to protect orbital contents during any cuts involving the bony orbit. Care must be taken to ensure that the posterior wall of the frontal sinus is cut, if the sinus has been entered. A cut is made from the first burr hole through the orbit, again taking care to protect the orbital contents. A notched osteotome is used to incise the orbital roof from the second burr hole toward the nasion, while protecting the orbital contents during this cut. The bone flap is now elevated. Remaining portions of the orbital roof, lateral orbital wall, and sphenoid wing can be removed with the craniotome for later reconstruction (**Fig. 25.6**). With the orbit now exposed, electromyographic electrodes may be directly placed into the superior oblique, superior rectus, and lateral rectus muscles to monitor cranial nerves III, IV, and VI.

Proximal control of the carotid artery is the next objective (**Fig. 25.7**). The middle fossa dura is elevated in a posterior-to-anterior direction. The greater superficial petrosal nerve (GSPN) emerges from the facial hiatus and should be dissected free of the dura. Traction of the GSPN is avoided to alleviate transmission to the geniculate ganglion, which can lead to facial palsy. The middle meningeal artery is identified, thoroughly electrocoagulated, and divided. Continued dural elevation reveals V3 and foramen ovale. The apices of the Glasscock triangle are now exposed: the facial hiatus, the anterior aspect of the foramen ovale, and the intersection of the GSPN and the

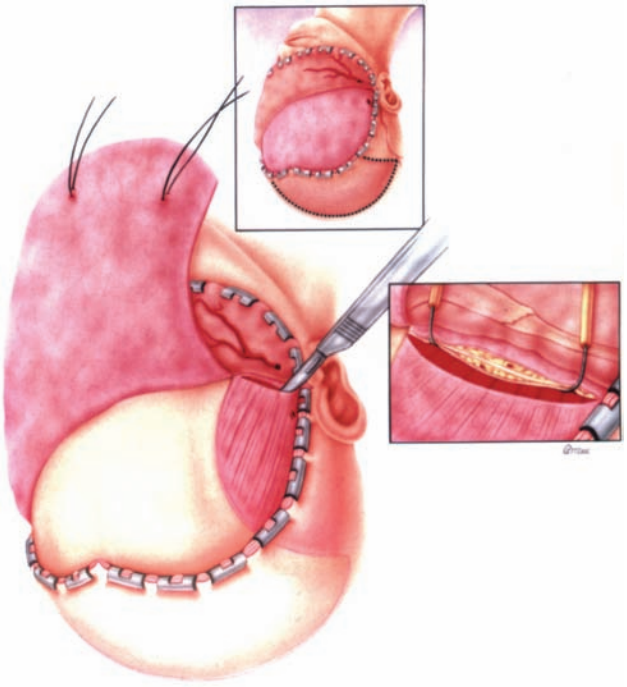


Fig. 25.3 Superficial soft tissue dissection. Upper inset: Dissection of the anterior and posterior scalp flaps while leaving the pericranial flap adherent to the calvarium. The main illustration shows elevation of the pericranial flap and the temporal fascia incision. Right inset: Subfascial dissection to preserve the facial nerve branches to the frontalis muscle. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

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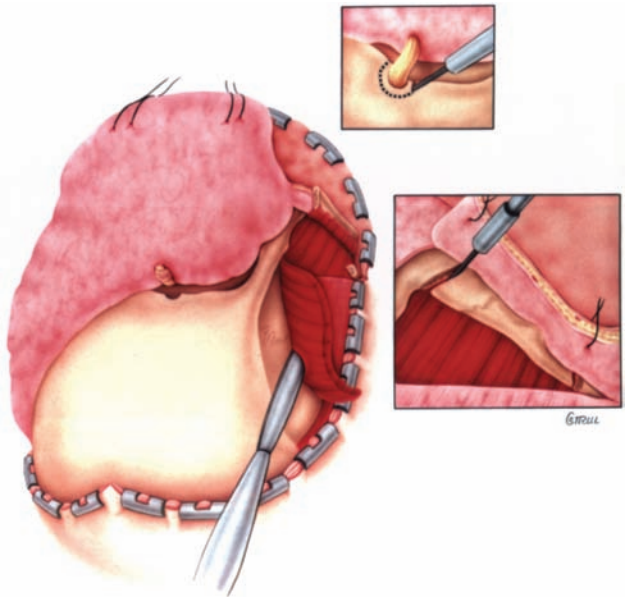


Fig. 25.4 Management of the temporalis muscle and zygomatic arch. Upper inset: Use of the air drill to preserve the supraorbital nerve. Right inset: Osteotomies at either end of the zygoma, which allows the temporalis muscle, shown in the main illustration, to be elevated off the calvarium and retracted inferiorly. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

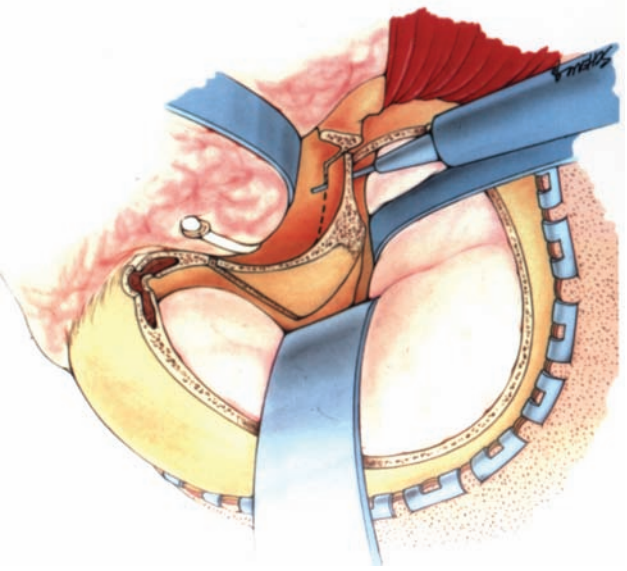


Fig. 25.6 Remaining portions of the orbital roof, sphenoid wing, and lateral orbital wall can be removed and used during reconstruction. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

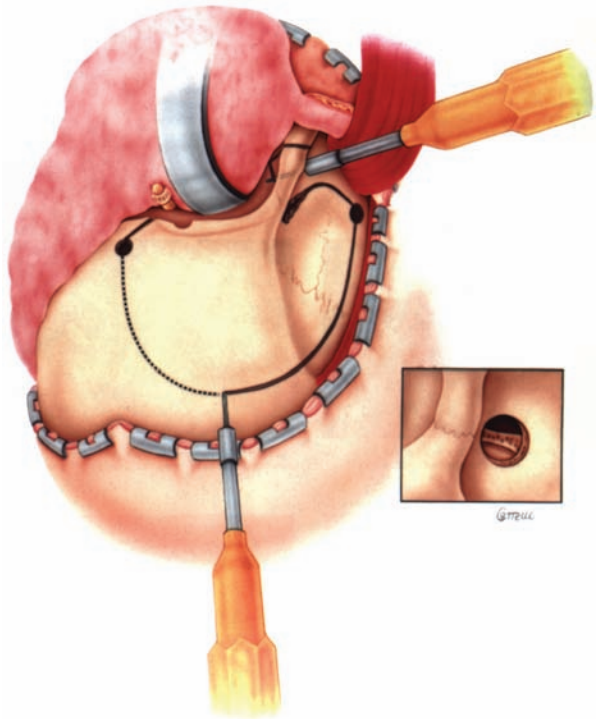


Fig. 25.5 Creating the cranioorbital flap. Burr holes are placed in the keyhole and posteroinferiorly in the temporal squama. A cut is made across the lateral orbital wall, then brought up to the keyhole. The keyhole is connected posteriorly to the posterior burr hole. The cut is then continued up to the frontal bone and through the supraorbital bar. If the frontal sinus is entered, the posterior wall of the sinus must be cut as well. Orbital contents must be protected when drilling the bony orbit. A V-chisel is then used to cut the orbital roof from the keyhole (right inset) to the medial cut behind the supraorbital bar. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

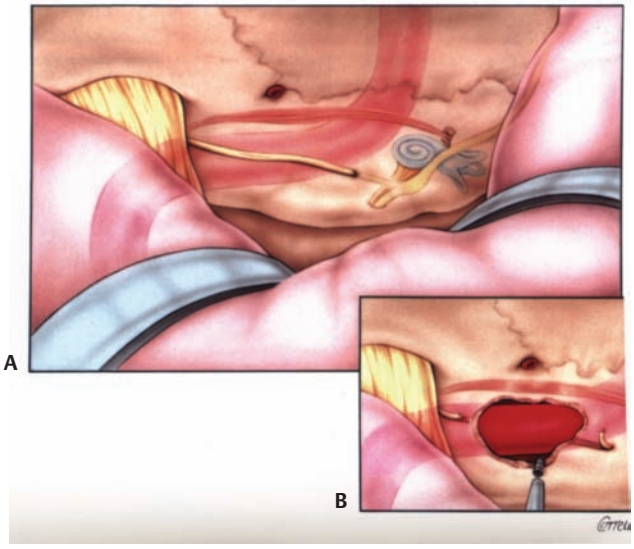


Fig. 25.7 **(A)** Middle fossa dissection. The main illustration shows anatomy of the floor of the middle fossa. Lower inset: Drilling lateral to V3 and posterior to the greater superficial petrosal nerve dissection reveals the petrous carotid artery, thereby providing proximal control. **(B)** Drilling to obtain medial cavernous sinus exposure. The main illustration (upper image) shows opening of the optic canal and drilling of the anterior clinoid process. Inset: Appearance of the anterior clinoid process and optic strut after resection. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

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lateral aspect of the V3. This triangle overlies the carotid artery, and drilling here with a diamond bit and constant irrigation exposes the carotid artery. This may be sufficient for proximal control of the artery or to allow drilling posterolaterally from the known location of the artery. Proximal control may be obtained by sufficient exposure for placement of a temporary clip on the petrous carotid artery, if necessary. Alternatively, a Fogarty catheter may be inserted into the carotid canal. Should vascular control be required, the catheter balloon can be inflated to occlude the carotid artery in the carotid canal.[16]

Medial exposure of the cavernous sinus and exposure of the paraclinoid carotid artery are obtained by drilling out the remainder of the orbital roof, the superior orbital fissure, the anterior clinoid process, and the optic strut (**Fig. 25.8**). Drilling adjacent to the orbital apex and optic canal mandates a diamond burr and copious irrigation to dissipate the heat of drilling. The anterior clinoid process is cored out with the drill and then disarticulated by drilling out the optic strut. The clinoid is subperiosteally dissected and resected. The optic canal is opened first. The superior orbital fissure is opened by drilling along the lesser sphenoid wing. This procedure exposes the subclinoid portion of the carotid artery, which is both extradural and extracavernous, and provides distal control of the carotid artery.

Entrance into the cavernous sinus has been described in relationship to the intervals between the neurovascular structures of the cavernous sinus. These intervals have been annotated as 10 triangles distributed among the parasellar, middle fossa, and paraclival locations.[17] The actual approach taken depends on the anatomy of the lesion in relationship to the cavernous sinus structures and must be individualized for each patient. In general, there are two approaches for entry into the cavernous sinus: a superior and a lateral approach. The superior approach is particularly suited to those lesions adjacent to the anterior loop of the carotid artery, and those that are superior and/or medial to the cavernous carotid artery. The lateral approach lends itself well to exposing those lesions lateral and/or inferior to the carotid artery and those that are posteriorly located within the cavernous sinus. Frequently, these approaches are combined for lesions widely involving the sinus.

*Superior entry (***Fig. 25.9***).* After exposing the superior surface of the cavernous sinus, the dura overlying the optic nerve is divided over the length of the optic canal to free the optic nerve. The distal carotid ring is now divided. The dura is then incised toward the oculomotor nerve, providing initial entry into the cavernous sinus. Exposure can be increased by dissecting along the length of the carotid artery. Further exposure can be obtained by subperiosteal dissection of the posterior clinoid process and drilling off the process, the dorsum sellae, and the superior clivus. These maneuvers allow increased exposure of the posterior fossa.

For tumors with medial extension, the planum sphenoidale can be drilled away. This allows exposure of the sphenoid sinus. Dissection and incision of the diaphragma sellae allows visualization of the pituitary gland.

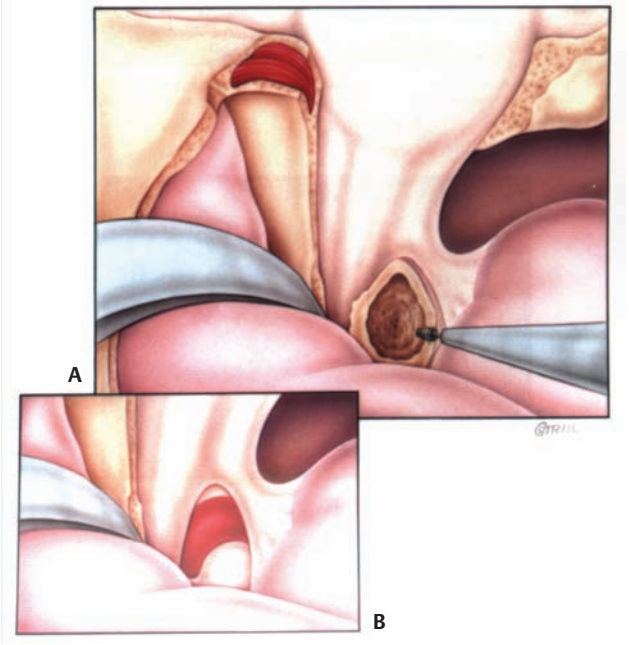


Fig. 25.8 **(A)** Superior entry into the cavernous sinus. Inset: Incising the dura over the optic nerve and the medial cavernous sinus. Main illustration shows the superior exposure obtained. **(B)** Intradural lateral entry into the cavernous sinus through the Parkinson triangle. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

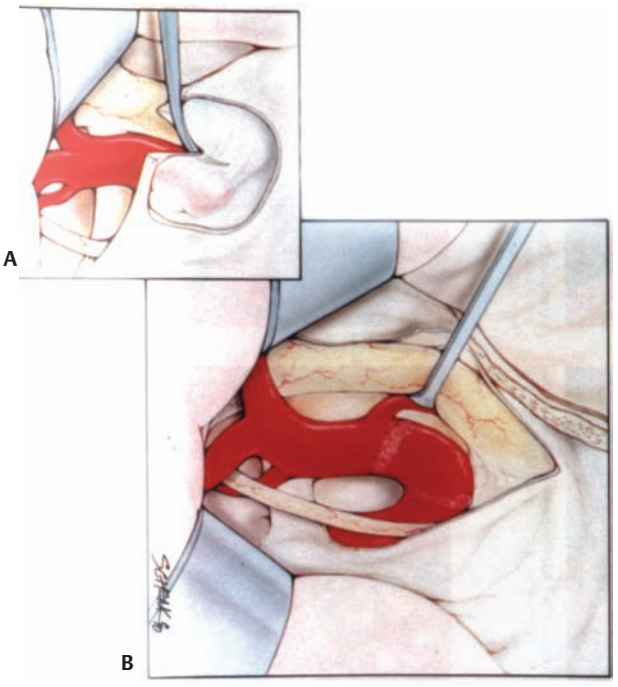


Fig. 25.9 **(A)** Illustration demonstrating extradural and intradural from both the superior and lateral entry of the cavernous sinus. The carotid artery is identified behind the gasserian ganglion proximally and in the anterior superior cavernous sinus distally. **(B)** Illustration demonstrating preservation of the cavernous sinus neurovascular structures after the removal of meningioma. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

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Great care must be taken during closure to obliterate any communication between the cavernous sinus and sphenoid sinus to prevent CSF leakage.

Lateral entry. Lateral entry into the cavernous sinus can be intradural or extradural. Extradural entry begins by incising the dura propria overlying V3. The dura propria is peeled away from the trigeminal branches and ganglion with superiorly directed traction. This will initially expose the third division and lateral ganglion followed by the second division and the majority of the remainder of the ganglion. Anterior extension from the region of the Meckel cave can be addressed by drilling away bone around the foramina rotundum and ovale to allow exposure of the sphenoid sinus and infratemporal fossa. Drilling bone here will also free the trigeminal branches, which will, in turn, allow greater mobility of these branches and the ganglion. A mass beginning to enter the posterior fossa can be further exposed by drilling the petrous apex. This drilling also allows greater exposure around and under the trigeminal ganglion.

For lesions requiring intradural exposure, intradural entry into the cavernous sinus is achieved through the Parkinson triangle[18] (**Fig. 25.10**). Cranial nerves III and IV are identified over the tentorial edge. An incision beneath the anticipated position of the fourth nerve is fashioned and extended ~8 mm anteriorly and 8 mm inferiorly. The external dural layer is peeled away from the thin inner dural layer in which nerves III, IV, and V are found. The dural flap can be further dissected from the trigeminal ganglion to expose the Meckel cave. Exposure can be increased posteriorly and into the posterior fossa by drilling the petrous apex. The trigeminal nerve can be mobilized by drilling the foramina rotundum and ovale.

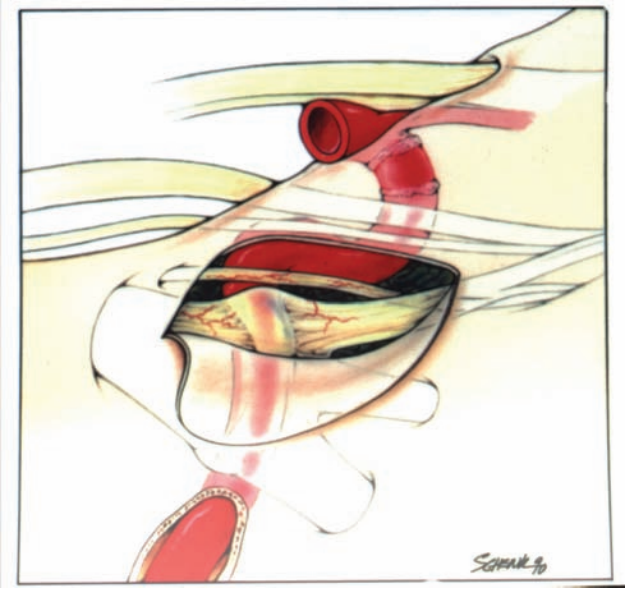


Fig. 25.10 Illustration demonstrating the operative anatomy of a lateral approach to the cavernous sinus with the lateral wall opened. Reprinted with permission from Al-Mefty O. Operative Atlas of Meningiomas. Philadelphia, PA: Lippincott Williams and Wilkins; 1998.

The inner dural layer between the fourth nerve and the ophthalmic division can be incised to expose the lateral space of the cavernous sinus, the posterior bend and the horizontal segment of the intracavernous carotid artery, and the lateral cavernous and meningohypophyseal arteries. The abducens nerve is the only cranial nerve coursing inside the cavernous sinus proper, often appearing in fascicles of two to five nerves, and should be carefully located and protected.[19,20] Frequently, meningiomas necessitate the combination of extra- and intradural cavernous sinus dissection with a combination of superior and lateral entry.

Reconstruction after the COZ approach begins by directing attention toward preventing CSF leaks by searching for and obliterating any feature of the dissection that may result in a CSF leak. Any entrance into the paranasal sinuses or the eustachian tube should be obliterated with fat and fascia. Any tenuous or incomplete dural closures should be reinforced with tissue—preferably autologous—such as fascia, muscle, or fat. Fibrin glue can be used for further reinforcement. The thick pericranial flap is now brought down under the frontal lobe, over the orbit, and over any sinus entries in the middle fossa or petrous apex. The orbital roof is reconstructed to prevent late enophthalmos. Dural tack-up sutures are placed circumferentially, including the subtemporal region, to obliterate dead space and prevent postoperative development of epidural hematomas. If the frontal sinus has been entered, the mucosa should be exenterated and the cavity packed with fat or tissue to prevent mucocele formation and CSF leakage. The cranioorbital flap is secured in place with titanium miniplates. Bony defects can be obliterated with titanium plates or mesh, or any of several cranioplastic materials, such as hydroxyapatite cement. The temporalis muscle is sutured to the superior temporal line. The zygoma is plated into position with titanium miniplates. The scalp is closed in layers and a craniotomy headwrap is applied to decrease postoperative fluid collection under the flap.

Zygomatic Approach Lumbar drain placement facilitates brain relaxation and temporal lobe elevation. Scalp incision, temporalis dissection, and zygomatic osteotomies are performed as previously described (**Figs. 25.3, 25.4,** and **25.5**). Four burr holes are placed (**Fig. 25.11**): the first in the keyhole, the second low and anterior in the middle fossa, the third low and posterior in the middle fossa, and the last along the superior temporal line. These four burr holes are connected using a craniotome. Residual bone along the sphenoid wing or temporal squama can be removed with the craniotome as well. Dissection along the middle fossa floor (dural elevation, middle meningeal artery division, GSPN dissection, and acquisition of proximal carotid artery control) is performed as previously described (**Fig. 25.7**). Further dural elevation exposes the confines of the petrous apex: the lateral aspect of V3, the GSPN, and the facial hiatus. If exposure of the petroclival area is required, then the petrous apex is drilled down. Lateral entrance is similar to the extradural lateral cavernous sinus entrance previously described.

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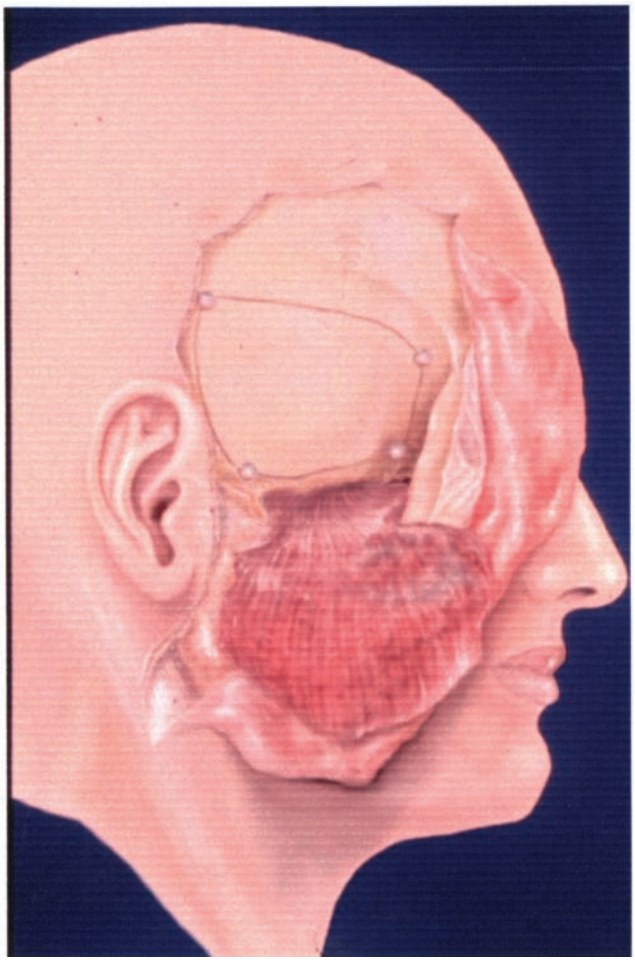


Fig. 25.11 Four burr holes are placed using the zygomatic approach.

The dura propria overlying V3 is incised and peeled away from the trigeminal branches and ganglion. Further dissection of the dura propria reveals V1 and the fourth cranial nerve. Anterior extension from the Meckel cave is exposed by drilling bone around the foramina rotundum and ovale to allow exposure of the sphenoid sinus and infratemporal fossa. If intradural extension of the tumor is discovered, such as with invasion into the temporal lobe and cisterns anterior to the brain stem, then intradural exposure can be easily obtained using this approach (**Fig. 25.10**). Lateral intradural entrance as already described can also be performed using this approach, although distal control of the carotid artery cannot be obtained.

Closure of the zygomatic approach requires meticulous attention to prevent CSF leaks, as described. A strip of posterior temporalis muscle is placed along the petrous apex to prevent leakage. The remainder of the closure is similar to that of the COZ closure.

Dissection within the Cavernous Sinus Dissection within the cavernous sinus is performed with microdissectors and microscissors. Sharp dissection is performed, whenever possible, to prevent traction of intracavernous cranial nerves and arteries. Coagulation, where necessary, should be bipolar. As mentioned, once the cavernous sinus is entered, the abducens nerve should be located and preserved. Venous bleeding can be controlled using head elevation and judicious packing with hemostatic agents. Any cranial nerve that is frankly severed should be directly repaired. If a tension-free direct repair is not possible, an interposition graft should be performed. Injury to the carotid artery can be addressed in several ways. Temporary clipping and direct repair of tears can be performed with 8–0 suture; more severe carotid injury can be treated by vein graft repair.

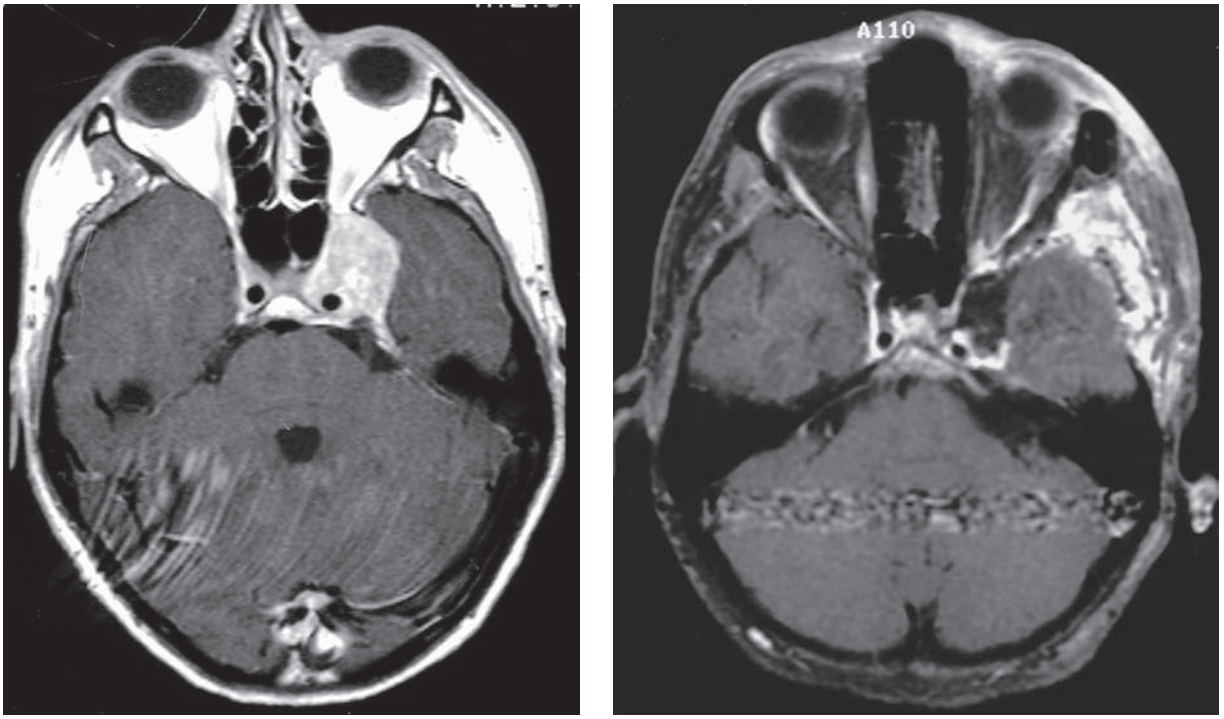
Surgical Outcomes

Resection Rates and Progression Surgical resection of tumors within the cavernous sinus is not the prohibitive procedure it once was (**Fig. 25.12**). Published series have established the technical feasibility of achieving gross total resection (GTR), and moreover have correlated extent of resection with rate of recurrence (**Table 25.1**). Reports vary widely, as do outcome metrics and surgical goals. In the senior author's (OAM) experience, GTR in a series of 41 patients was achieved in 76% of patients, with an 11% recurrence rate in cases of GTR. Twenty percent of subtotally resected tumor recurred.[38] A more recent review of 163 cases operated on by the senior author (OAM) has shown a GTR rate of 44%, with 7% of these patients experiencing recurrence.[1] Of subtotally resected cases, 57% showed radiographic progression.[21] Other published series report GTR rates of 12 to 92%[1,22–27] (**Table 25.1**). De Jesús and colleagues reported 87% progression-free survival at 3 years and 62% at 5 years.[23] Two recent series described results with varying degrees of surgical resection.[26,27] GTR in one series was 92% when aggressive surgery was pursued, with a recurrence rate of 12.5% compared with 26% in subtotally resected cases.[27] In a similar report, no patients had recurrent tumors over a 15-year mean follow-up period when aggressive resection with a 12% GTR rate was pursued compared with an 18% recurrence rate in subtotally resected patients.[26]

Vascular Complications Stroke is a significant concern during cavernous sinus meningioma surgery, and fortunately reported rates of ischemic complications are low.[21–23,26,27] In the senior author's (OAM's) series, seven of 188 patients (3.7%) experienced ischemic stroke. Cusimano et al[22] reported four disabling strokes in 89 patients, and De Jesús et al[23] reported six strokes (5%); however, this group's operative philosophy includes bypass and resection of an invaded carotid artery. More recent series have reported a 4 to 5% rate of ischemic complications.[26,27]

Cranial Nerves Cranial nerve morbidity is a central issue in cavernous sinus meningioma surgery. The preponderance of evidence shows that existing preoperative cranial nerve deficits infrequently improve, and that few new permanent cranial nerve deficits appear postoperatively. Most series of aggressive surgical resection have reported that the majority of preoperative cranial neuropathies remain the same. DeMonte and Al-Mefty reported improvements in 14% of preoperatively affected cranial nerves, whereas 80% remained stable.[1] They re-

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Fig. 25.12 Magnetic resonance imaging of a patient with a left cavernous sinus meningioma. **(A)** Preoperative image. **(B)** Image showing complete resection.

Table 25.1 Results of Surgical Resection of Cavernous Sinus Meningiomas

Series	No. Patients	Mean Follow-Up (Range)	Gross Total Resection (%)	% Tumor Recurrence	Progression-Free Survival	New Cranial Nerve Deficits	Mortality
Knosp et al[24]	29	(3 mos–7 yrs)	5 (17)			CN III—14% CN IV—14% CN VI—3.4% CN V1—3.4% CN V2—10% CN V3—17%	3 (10%)
DeMonte et al[38]	41	45 mos (2 mos–10 yrs)	31 (82)	13		10 in 7 (18%) patients	6 (16%)
De Jesús et al[23]	119	33.8 mos	73 (61)	10	87% @ 3 years 62% @ 5 years		
O'Sullivan et al[25]	39	24 mos (6 mos–5.3 yrs)	8 (21)	5		23 (12% of 192 normal cranial nerves at risk)	
Cusimano et al[22]	89	28 mos	59 (66)	7			5 (6%)
Sekhar et al[39]	114	3.9 yrs median	78 (68)				0.8%
Heth and Al-Mefty[21]	163		44 (63)	9			
Sindou et al[27]	100	8.3 yrs (3–20 yrs)	12 (12)	11		CN III—29% CN IV—15% CN VI—17% CN V—24% (CN worsening)	5 (5%)
Pichierri et al[26]	24*	9.7 yrs	22 (92)	12.5			

* "Open" cavernous sinus surgery.

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ported 10 new neuropathies in seven (18%) patients. In a recent reevaluation of the cases operated on by one author (OAM), 21 of 163 patients (12.8%) experienced new cranial nerve deficits, 14 of 163 operated patients (8.5%) experienced worsening of a preoperative deficit, and 11 of 163 patients (6.7%) experienced both a new cranial nerve deficit and a deterioration of existing deficits. The majority of neuropathies remained stable. O'Sullivan et al[25] reported four of 17 patients (24%) with preoperative optic nerve deficits improved, whereas one of 17 patients (6%) with normal preoperative vision suffered a new visual field deficit. They reported an overall 12% risk of new cranial nerve morbidity. Knosp et al[24] report that 43% of oculomotor deficits improved, 50% of sixth nerve deficits improved, and 33% and 28% of V2 and V3 deficits improved, respectively. Preoperative deficits in nerves IV and V1 improved rarely (0% and 7%, respectively). In their experience, no patient who had intact cranial nerves experienced any postoperative deficits. There were no new third nerve palsies, one new sixth nerve palsy, one new V1 nerve palsy, two new fourth nerve palsies, three new V2 palsies, and five new V3 palsies.

In a more recent series of intracavernous tumor resections, 33% patients developed new CN deficits or worsening of preoperative deficits, whereas 67% remained stable. Aggressive resection was associated with an increasing incidence of CN III dysfunction from 50 to 79%, but with a reduction in CN VI palsies, stability of CN IV deficits, and dramatic improvement in CN V deficits.[26]

Among 100 patients with varying degrees of resection, preoperative neuropathies of II through VI were unchanged at 1 year in 60 to 74% of cases.[27] When stratified by degree of resection, cranial nerve deficits were more prevalent in patients who had undergone a more aggressive resection (33%) compared with subtotally resected intracavernous tumors (19%) at 1-year follow-up. Longer follow-up, though not detailed, suggested a reduction in the difference in cranial nerve deficits between the to groups.

Radiosurgery and Radiotherapy

A vast literature has emerged in the past 15 years on the utility of radiation therapy in the treatment of cavernous sinus and other skull base meningiomas (reviewed in Minniti et al[28] and in Chapters 39 and 40 of this text). Although the aim of aggressive surgical resection is complete tumor removal, the goal of treatment with radiotherapy in any form is tumor growth control. Proponents of this treatment modality commonly cite the difficulty and attendant morbidity of complete microsurgical resection of cavernous sinus tumors.

Early reports described the use of conventional fractionated external beam radiotherapy. More recently, groups have amassed considerable experience in the use of stereotactic radiosurgery (SRS) (e.g., gamma knife, LINAC, and Cyberknife [Accuray, Sunnyvale, CA]), fractionated stereotactic radiotherapy (FSRT), intensity-modulated radiotherapy (IMRT), and, less commonly, proton beam radiotherapy, in the up-front or adjuvant treatment of cavernous sinus meningiomas. The largest reported series of SRS in cavernous sinus meningioma, in which half were treated with surgery and half with gamma knife alone, described 306 patients followed for a median of 4 years. Tumor control, though not defined, is reported as 97%, whereas 6.3% of patients experienced new cranial nerve deficits.[29]

There are several considerations for the surgeon considering the use of radiotherapy as an up-front treatment modality other than the absence of obtaining a pathological diagnosis. These tumors often abut the optic apparatus, whose dose limitation is usually considered to be 8 to 10 Gy; at least 2 to 3 mm should separate the field from the optic apparatus. Dosage, however, correlates with tumor control; in one particular study, treatment with 10 to 12 Gy was associated with a 20% progression rate over a mean follow-up of 42 months, whereas a minimum of 14 Gy conferred a 0% tumor progression rate.[30] A more recent report describes ipsilateral intracavernous carotid occlusion following a dose of 22.3 Gy.[31] Radiotherapy complications can include, among others, imaging changes on MRI (25% in some series, with just under half of these cases symptomatic) and varying rates of seizure and cognitive decline depending on dose administered.[32,33]

A notable caveat to the increasing use of radiotherapy as an up-front or adjuvant treatment strategy is the specter of aggressive late regrowth potential. One report of such a group of 13 patients describes a particularly aggressive pattern of growth in cases of failed tumor control after radiation therapy occurring as late as 14 years after treatment.[34] Long-term follow-up of irradiated patients is mandatory given the prevalence of radiotherapy use as a therapeutic strategy.

Currently, we favor postoperative radiosurgery in patients with biologically aggressive residual tumor or in small to moderate recurrence after a period of documented growth.

Subradical Surgery and Radiation

Concerns over the morbidity of complete microsurgical resection and the limitations of radiation dosing conferred by proximity to the optic apparatus have led to an emerging therapeutic paradigm of planned subtotal resection and postoperative radiotherapy. The stated aim of such an approach is to debulk as much tumor as is safe—and in so doing remove tumor from around the optic apparatus, freeing residual tumor to receive higher doses of postoperative radiation. Long-term results after such approaches will be informative.[35–37]

◆ Conclusion

Cavernous sinus meningiomas are challenging lesions whose management continues to inspire considerable debate. A thorough understanding of the patient's history, physical examination, radiographic findings, and

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other evaluations is crucial to their evaluation. It is also critical that patients with cavernous sinus meningiomas understand the natural history, options, and risks associated with every treatment.

Skull base techniques have rendered the resection of cavernous sinus meningiomas a viable treatment option, and currently the only treatment option if complete tumor removal is the goal of therapy. The main problem encountered in the pursuit of total resection is cranial neuropathy; most neuropathies encountered in these patients are evident at presentation, whereas a minority occur postoperatively and are permanent. Optic nerve deficits are infrequently encountered. Microsurgery continues to be the sole means of achieving complete tumor resection; we await long-term evaluations of the success and side effects of alternative or complementary approaches.

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Chapter 26

Sphenoorbital Meningiomas

Mustafa Aziz Hatiboglu and Franco DeMonte

Cushing and Eisenhardt[1] first described meningioma en plaque as "the flat spreading tumors (meningiomas en plaque) which provoke hyperostosis chiefly of the greater wing of the sphenoid." Cushing described that the infiltration of the bone by meningioma cells stimulates osteoblastic activity resulting in hyperostosis.[2] This hyperostosis is typical of the sphenoid bone, and is less frequent at other cranial sites.[3] These tumors have been referred to in the literature as sphenoid wing meningioma *en plaque*, pterional meningioma en plaque, hyperostosing meningioma of the sphenoid ridge, invading meningioma of the sphenoid ridge, or more generically as sphenoorbital meningiomas (SOMs).[4]

SOMs constitute up to 9% of all intracranial meningiomas.[5] They originate from the dura of the sphenoid wing, and they can extend into the cavernous sinus, superior orbital fissure (SOF), orbital apex, and convexity dura. Soft tissue growth can spread to extracranial compartments, including the orbit, the infratemporal fossa, and the temporal fossa. Bone involvement may extend to include the anterior clinoid process and lesser sphenoid wing, the orbital roof, the lateral orbital wall, and the middle fossa base. Tumor may also extend to the optic canal and the paranasal sinuses.[4,6–8]

Most patients with SOM are middle-aged women whose symptoms are primarily attributable to the hyperostosis.[9] Earlier reports concluded that SOMs were not usually resectable because of their extensive bone and soft tissue involvement, and surgical treatment was discouraged.[10–12] Seminal to our understanding of these tumors and to the development of the surgical techniques for their resection was the work of Guiot, Derome, and Tessier.[3,13,14] With further development of microsurgical techniques, skull base approaches, new neuroimaging techniques, and the use of image-guided surgery, more complete resection of SOMs has become technically feasible and safe. Recent studies report that successful results can be achieved in the majority of patients.[3,4,6–9,15–19] Even so, with efforts to preserve neurological function, residual tumor involving the cavernous sinus, orbital apex, and SOF remains a problematic surgical limitation. These areas are major sites for residual tumor, recurrence, and continued neurological deterioration. Adjuvant radiotherapy for residual tumor or recurrence commonly forms part of modern management plans.

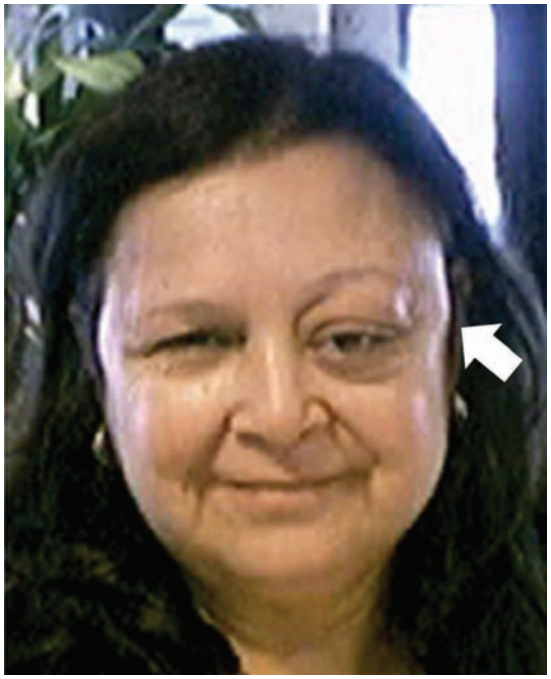
Clinical Presentation

The most common symptom is unilateral, nonpulsating, progressive proptosis (80 to 90%) (**Fig. 26.1A,B**). Optic neuropathy, evidenced by decreased visual acuity, loss of color vision, and a constricted visual field with an enlarged scotoma has been identified in 27 to 80% of patients in published series.[5] Other cranial nerve (CN) deficits (CN III, IV, V, VI, and VIII) are seen in 20 to 25% of patients. Even though the oculomotor nerve is the most commonly affected CN besides the optic nerve,[4,6–8,15,20–23] diplopia is usually on the basis of extraocular muscle restriction due to intraorbital tumor rather than secondary to neuropathy of the ocular motor nerves.

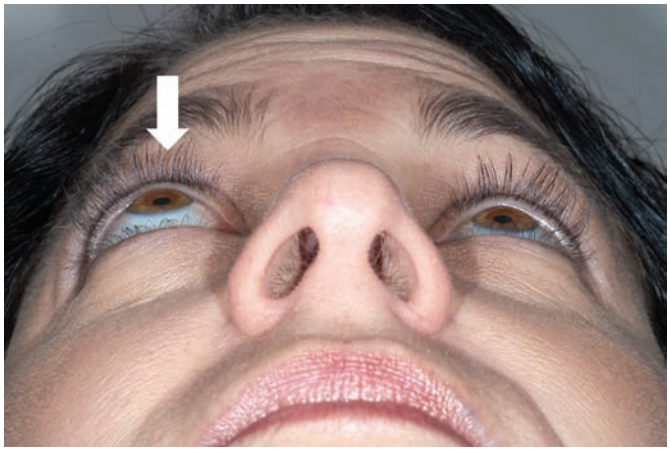
Radiological Evaluation

Although plain roentgenograms routinely demonstrate the hyperostosis, both computed tomography (CT) and magnetic resonance imaging (MRI) are critical for tumor diagnosis and management planning. CT scans show the

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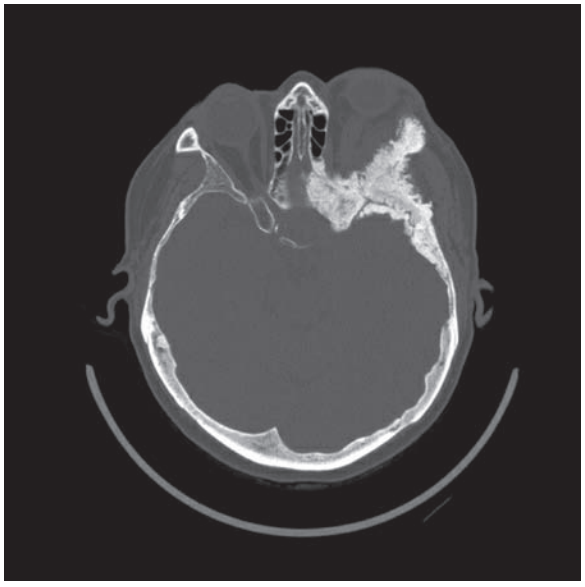


A



B

Fig. 26.1 **(A,B)** Two examples of patients with sphenoorbital meningioma (SOM) and proptosis. **(A)** This patient also has left temporal swelling indicative of tumor extension to the temporalis fossa. **(B)** Lesser degrees of proptosis can be best appreciated by observing the patient with the head tipped back. All figures are property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center and are used with permission.



A



B

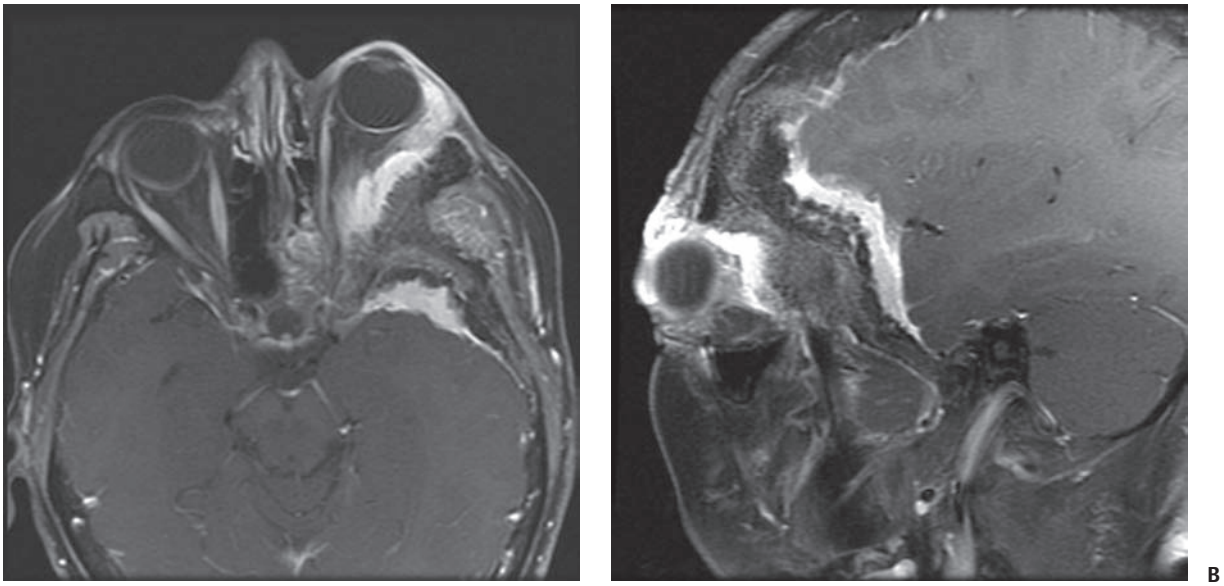
Fig. 26.2 **(A)** Axial and **(B)** coronal computed tomographic scans show typical hyperostosis including both lesser and greater sphenoid wings, anterior clinoid process, ethmoidal sinus, temporal squama, and frontal bone. Note the marked volume reduction of the orbit. All figures are property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center and are used with permission.

characteristic periosteal pattern of hyperostosis, surface irregularity of the hyperostotic bone, and remodeling of the orbital roof and sphenoid wing[7,20] (**Fig. 26.2A,B**). MRI demonstrates tumor extension to the soft tissues and the dura much better than contrast-enhanced CT (**Fig. 26.3A,B**). MRI, especially with postcontrast fat-suppression techniques, best identifies soft tissue involvement of the orbital contents, the infratemporal fossa, and the temporalis muscle.[4,7,20,24]

Histopathology

Most reports on the surgical treatment of SOMs describe these meningiomas as typically being of low grade (grade I) and most commonly of the meningothelial variant.[3,4,6,7,9,20,25] Atypical and anaplastic meningiomas have been reported.[7,24] Pathological analysis of the hyperostotic bone typically identifies direct tumor involvement and meningothelial cells within Haversian canals.[27]

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A B

Fig. 26.3 **(A)** Axial and **(B)** sagittal contrast-enhanced T1-weighted magnetic resonance imaging shows the widespread dural involvement and the intraorbital, frontal, and temporal fossa extensions of tumor. All figures are property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center and are used with permission.

Differential Diagnosis

Fibrous dysplasia, osteoma, osteoblastoma, Paget disease, hyperostosis frontalis interna, osteoblastic metastases, erythroid hyperplasia, and sarcoid may mimic hyperostosing meningiomas and should be considered in the differential diagnosis.[9,20,28–30]

Management

The management of a patient with SOM should be individualized based on considerations of the patient's age, symptom complex, presence or absence of neurological findings, presence of comorbidities, and desired outcomes of intervention. Observation, radiation therapy or surgical resection can be options for management of SOMs.

Observation

Almost all patients without evidence of optic neuropathy can be managed nonoperatively, at least initially. Should observational management be chosen, close clinical follow-up with detailed cranial nerve testing and ophthalmological examination of visual acuity, visual field, and degree of proptosis (Hertel exophthalmometry) are necessary in addition to follow-up MRI. Tumor growth, progressive proptosis, and the onset of neuropathy are all indications for intervention.

Surgical Resection

Preoperative Evaluation

A comprehensive ophthalmological and neurological examination should be completed for all patients with the diagnosis of SOM. A detailed visual evaluation, with visual field testing, assessment of visual acuity, fundus examination, and quantification of the degree of exophthalmos using Hertel measurements, is crucial. Radiological assessment including high-resolution CT scans to assess degree and extent of hyperostosis and contrast-enhanced MRI are performed. These are complementary studies and together optimally identify the full extent of the tumor.

The surgical approach should be tailored according to the extent of tumoral involvement of the orbit and adjacent anatomical compartments. Different surgical approaches, including pterional, frontotemporal, transzygomatic, frontotemporal orbitozygomatic, and frontotemporal-orbital, have been described for SOM resection. The approach selected must allow access to the orbit and the middle fossa base for resection of the tumor-infiltrated bone and soft tissue. Decompression of the SOF, the optic canal, and the cavernous sinus should be possible, and brain retraction should be minimal.

Surgical Technique

The patient is positioned supine with the head rotated to the contralateral side. A large frontotemporal or bicoro-

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nal skin incision is recommended to provide access to enough pericranial tissue to repair the dural defect and the cranial base.[31] The extent of the craniectomy and the size of the craniotomy are "right-sized" intraoperatively based on feedback from CT and MRI coregistered stereotactic navigational data. These systems are very helpful in the delineation of the extent of hyperostotic bone and dural involvement. The temporalis fascia is incised 2 cm above the "keyhole" region, from the superior temporal line to the root of the zygoma. The periosteum and the superficial and deep layers of the temporalis fascia are elevated off of the frontal bone, orbital rim, and zygoma as one, thus protecting the frontalis branch of the facial nerve (**Fig. 26.4A**) Subperiosteal dissection is utilized to elevate the temporalis muscle laterally and inferiorly, carefully preserving the blood supply and innervation. When infratemporal fossa exposure is required, a zygomatic osteotomy is performed, leaving the zygomatic arch attached to the masseter muscle (**Fig. 26.4B,C**). Tumor-infiltrated temporalis muscle is excised. The hyperostotic bone of the lateral sphenoid wing is usually encountered during elevation of the temporalis muscle (**Fig. 26.4D**). This tumor-infiltrated bone is removed with a high-speed cutting burr and rongeurs. This allows entry into the orbit and intracranial spaces to allow for removal of tumor extensions to these spaces (**Fig. 26.5A**). If more extensive dural or orbital exposure is necessary, a frontotemporal craniotomy can be elevated. If necessary, osteotomies of the orbital rims can be cut, and the supralateral orbital rim elevated, either separately or in continuity with the frontotemporal bone flap (**Fig. 26.6**).[31] All hyperostotic bone of the lesser sphenoid wing, middle fossa floor, lateral orbital wall, orbital roof, and anterior clinoid process is removed extradurally using a high-speed drill under magnification and constant irrigation. As the lesser sphenoid wing and anterior clinoid are removed, the optic canal and upper part of the SOF are opened. The foramen ovale and rotundum are opened as the floor of the hyperostotic middle fossa is removed (**Fig. 26.5B**). Intradural tumor involvement is removed with sharp dissection and microscopic techniques (**Fig. 26.5C**). Dural resection is extended to include the medial temporal dura of the lateral cavernous sinus wall if necessary. Meningioma extension within the cavernous sinus and SOF can directly invade the cranial nerves and the connective tissue planes between the cranial nerves and is typically left in place to avoid neurological complications. Once the intracranial portion of the tumor has been excised, attention is turned to the intraorbital involvement. The dura is repaired with pericranium, temporalis fascia, or allograft material before the commencement of intraorbital tumor resection.

Often, tumor extension into the orbit is extraconal and extraperiorbital. This extent is removed when the bone of the lateral orbit is removed. When involved, the periorbita is resected, and intraorbital tumor invasion is removed. The lateral rectus can be tagged with a suture transconjunctivally at the beginning of the case to help identify the muscle once tumor resection has started. All intraorbital, extraconal tumor can be removed. Invasion into the orbital apex, annulus of Zinn, or SOF is left in place due to the high risk of injury to the cranial nerves.

If the sphenoid or ethmoid sinuses are entered, these are obturated with autologous fat graft to prevent cerebrospinal fluid (CSF) leakage. If a periorbital defect is present, it is closed with locally harvested temporalis fas-

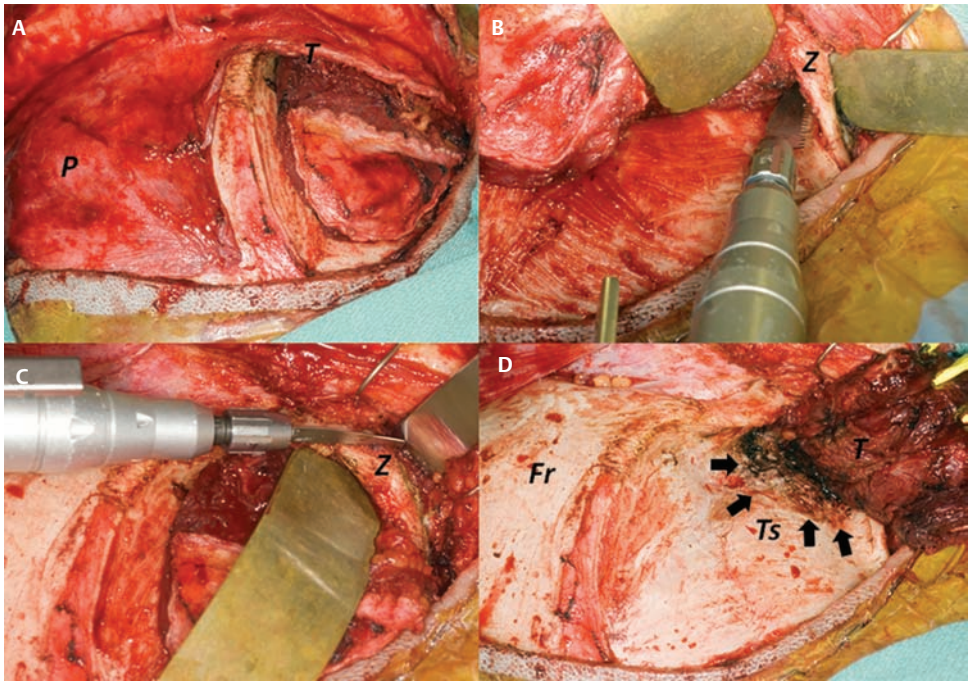


Fig. 26.4 **(A)** The temporalis fascia (T) has been incised through both superficial and deep layers to allow for a subfascial dissection of the zygomatic bone. The temporalis muscle has been elevated and the pericranial graft (P) prepared. **(B)** Posterior and **(C)** anterior osteotomies in the zygomatic arch (Z) allow its inferior translocation and access to the infratemporal fossa when needed. **(D)** With elevation of the temporalis muscle, the hyperostotic bone of the greater sphenoid wing and squamous portion of the temporal bone (Ts) becomes evident (*arrows*). Fr, frontal bone. All figures are property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center and are used with permission.

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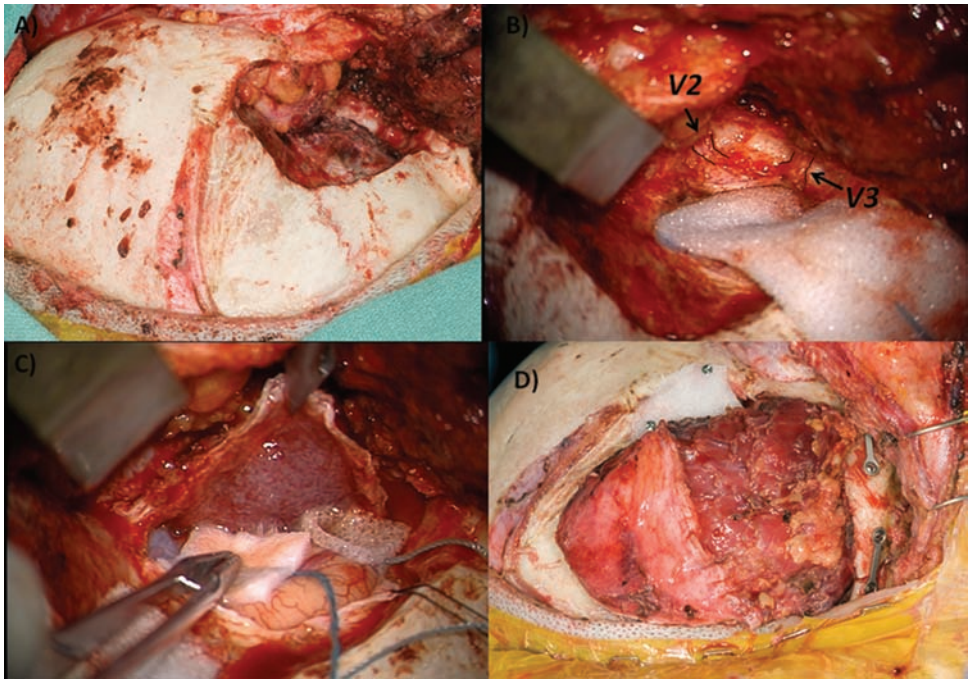


Fig. 26.5 **(A)** All hyperostotic bone of the greater sphenoid wing and squamous portion of the temporal bone has been removed allowing entry into the orbit and temporal fossa. **(B)** Hyperostotic bone of the floor of the middle cranial fossa has been removed and the maxillary (V2) and mandibular (V3) divisions of the trigeminal nerve completely decompressed (*arrows*). **(C)** The dural and intradural component of the tumor is excised microsurgically. **(D)** Reconstruction includes a cranioplasty of the pterion and anatomical replacement of the soft tissues and zygomatic arch. All figures are property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center and are used with permission.

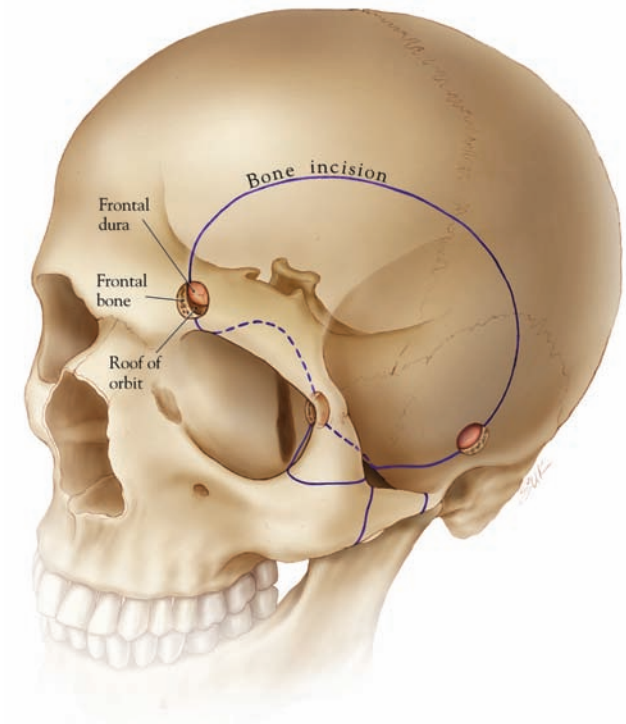


Fig. 26.6 The placement of entry holes and osteotomies for the orbitocranial zygomatic approach to the anterolateral skull base is illustrated. The extent of the bone opening is individually tailored by adding or omitting various portions of this approach. All figures are property of the Department of Neurosurgery, The University of Texas M.D. Anderson Cancer Center and are used with permission.

cia. The pericranial graft is then rotated over the orbit. This helps to compartmentalize the orbit from the intracranial space and to avoid adhesion of the orbital tissues to the dura.

Reconstruction

Options for cranioplasty include polymethylmethacrylate, various commercially available bone cements, or commercially available prostheses. Bony reconstruction of the orbital walls and sphenoid ridge is not necessary for achieving a good cosmetic result if the periorbita is intact or repaired.[32] If the superior or lateral orbital rim is removed, it can be reconstructed with split calvarial bone grafts or commercially available orbital prostheses. The orbital and zygomatic osteotomies are fixated with low-profile cranial plating. Remaining dead space from hyperostotic bone removal can be filled with autologous fat graft. If a temporal fossa defect exists, a cranioplasty is performed (**Fig. 26.5D**).

◆ Outcome

In modern case series, perioperative mortality rates vary from 0 to 4%. Since 2001, two perioperative deaths have been reported, one from a pulmonary embolus[19] and one from carotid laceration.[4] Temporary and permanent ptosis, diplopia (neuropathic and restrictive), visual loss, facial palsy, hemiplegia, aphasia, and diabetes insipidus

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have been reported as neurological complications of surgery.[4,6–9,20,23] The most commonly reported postoperative cranial neuropathies are of the oculomotor, trochlear, and trigeminal nerves. Temporal hollowing, orbital dystopia, chemosis, CSF leakage, meningitis, osteomyelitis, retroorbital hematoma, epidural hematoma, and brain edema have also been described.[4,6–9,20,33]

Oncological Outcome

Complete resection, usually corresponding to a Simpson grade II resection, has been reported in 33 to 83% of patients described in the literature.[4,6–8,34,35] In their series of 30 patients with SOM, Scarone et al, using the strictest criterion of a Simpson grade I resection, reported a zero rate of complete resection.[36] This highlights both the diffuse infiltrative nature of these tumors and the generally held philosophy of accepting a subtotal resection in the orbital apex, SOF, and cavernous sinus to preserve neural function.

Maroon et al, based on their experience with recurrent SOM, hypothesized reasons for recurrence as (1) failure of early diagnosis of the tumor; (2) inadequate surgical resection due to the complicated neurovascular anatomy of the orbit, cavernous sinus, and SOF; (3) the failure to appreciate that hyperostotic bone is indeed part of the neoplastic process; (4) the biological behavioral tendency of these tumors to insinuate themselves into the foramina, fissures, crevices, and interstices of the orbit, basal skull, and dura; and (5) the apprehension of surgeons that they will produce iatrogenic morbidity or mortality associated with a too-radical tumor resection.[20] Recurrence rates of 5 to 60% have been reported.[4,6–9,34–37,] Although some authors have found no difference in tumor recurrence between patients who underwent complete resection and those that underwent incomplete resection,[6,7,36] the majority of reports describe a greater incidence of tumor recurrence/progression in subtotally removed tumors.[4,9,19,34,35,37] Mirone et al reported a 5% recurrence rate in the 83% of patients that had a complete resection and a 25% progression rate in the 12 patients left with residual disease.[35] The most common clinical sign in patients with recurrent SOM is progressive proptosis. Optic neuropathy is the most common cranial nerve deficit in patients with recurrence.

Proptosis

Improvement of proptosis is common and was evident in 77 to 100% of patients.[3,4,7,34–37,] Methods used to evaluate the degree of proptosis varied from patient questionnaires, patient perception, clinical examinations, MRI measurements, and Hertel measurements. In a study by Heufelder et al[38] evaluating ophthalmological outcomes following surgical resection of SOM, a marked reduction of globe protrusion after surgery was seen in 85% of the patients. Proptosis was reduced to an average value of 2.7 mm by reduction of globe protrusion of up to 11 mm. No protrusion was noted in postoperative follow-up of all patients without tumor recurrence. Maroon et al[20] reported good cosmetic result without orbital reconstruction. However, no objective measurements or strict criteria were utilized in the evaluation of globe position. Subsequent reports that utilized objective measures emphasize that reconstruction of the orbit is necessary if more than one orbital wall is removed during surgery.[9,26] DeMonte et al, however, demonstrated that as long as the periorbita is intact, or has been primarily repaired, isolated bone defects of the medial or lateral orbital walls do not need to be reconstructed.[32] Similarly, isolated defects of the orbital roof, or combined defects of the orbital roof and lateral wall, do not need reconstruction as long as the periorbita is intact or repaired. Large defects of the orbital floor always need reconstruction, regardless of whether the periorbita is intact or repaired, to prevent hypoglobus or enophthalmos.[32]

Visual Outcome

Surgery generally provides a good visual outcome, with either stable or improved function in most patients. Differences in visual outcome can usually be attributed to the severity and duration of preoperative optic neuropathy. Once vision has deteriorated to light perception only, it is unlikely that acuity will improve. Mirone et al noted a 73% incidence of improvement of visual acuity in their patients with preoperative deficits. Eight patients (19.5%) remained stable, although two were blind preoperatively. Three patients with preoperatively severely depressed visual acuity worsened (7.3%). A single patient with normal preoperative vision experienced transient visual impairment postoperatively. Complete optic nerve decompression is key to improve and stabilize vision.[39] In their study evaluating the long-term follow-up of patients with SOM, Shrivastava et al[7] showed that visual acuity remained stable in 18 of 25 patients (72%) and improved in seven patients (28%). No patient had worsening of vision. Furthermore, the improvement in visual acuity and visual field continued in the long term, with an additional eight patients (32%) having improved visual function on their last neurological examination. In the study by Pompili et al, visual acuity returned to normal or improved in eight of 17 patients (48%) who had impaired visual acuity preoperatively.[3]

Preoperative diplopia has been reported to be improved in 50 to 68% of patients following tumor resection.[3,7,36] Ocular motility is often disturbed secondary to either extraocular muscle restriction or cranial nerve dysfunction. Either can cause diplopia as a presenting symptom. When diplopia is secondary to extraocular muscle restriction, symptoms typically resolve after orbital decompression and reconstruction. In Shrivastava's series,[7] five patients presented with double vision, thought to be secondary to extraocular muscle restriction. All five patients eventually had significant improve-

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ment after surgery. Similar to their data on visual acuity, Shrivastava et al noted a gradual improvement of ocular motor function over time; complete improvement was seen in 30% of the patients in 3 months and in 90% of the patients in 9 months.[7] Sandalcioglu et al[6] reported ptosis and CN III palsy in seven patients postoperatively. One patient had a preexisting CN III palsy, and one patient had a new permanent CN III palsy following surgery. Ringel et al[4] reported transient deficits of CN III in eight, CN VI in one, and CN IV in two patients after surgery. In that series, there were an additional eight patients with permanent palsy of CN III, one with palsy of CN IV, and two with palsy of CN VI. It is unclear what number of CN deficits may have been preexisting.

In summary, double vision secondary to restricted movement of the extraocular muscles improves with orbital decompression. When patients present with true CN III, IV, or VI deficits, these are less likely to improve after decompression. True cranial nerve palsies are usually due to direct tumor invasion of the cavernous sinus, SOF, or orbital apex and annulus of Zinn.

Radiation Therapy

The role of radiation therapy for subtotally resected meningioma is still controversial. Most reports advocate postoperative radiation therapy to improve progression-free survival time.[3,20,40,41] However, recurrence-free rates of 40 to 95% argue more for a policy of careful clinical and radiographic observation, with judicious use of radiation therapy for proven recurrence or progression.[4,6–9,19,34–37] Soyuer et al[42] demonstrated that the 5-year progression-free survival is significantly improved in patients who have had adjuvant radiotherapy for subtotally resected meningiomas when compared with those patients who have had subtotal resections without additional treatment. There was not, however, an overall survival advantage for those receiving immediate postoperative radiation. This significant finding indicates that delaying adjuvant radiation therapy does not compromise overall patient survival and may have the added benefit of delaying treatment-related toxicities. These tumors represent challenging targets for standard radiotherapy due to the vicinity of normal critical organs such as the eyes, optic nerves and chiasm, lacrimal gland, and pituitary gland.[41]

Although there is no specific study assessing the use of stereotactic radiosurgery (SRS) for SOMs, there are many published series evaluating the efficacy of SRS for skull base meningiomas.[43–49] Radiosurgical doses between 12 Gy and 18 Gy have been used to control skull base meningiomas.[50] Raw local tumor control rates between 80 and 100% have been reported in these studies. The University of Pittsburgh experience with 972 patients reported tumor control rates of 93% at 5 years and 87% at 15 years using a median dose to the tumor margin of 13 Gy.[51] A study assessing SRS for skull base meningiomas by Igaki et al[52] demonstrated that tumor volume ≤ 4 cm^3 was associated with better local control and that the local control was better in patients treated by SRS for postoperative residual tumor compared with those treated by SRS as primary treatment or for recurrent disease.

Summary

SOMs are typically slow-growing tumors that present with hyperostosis of the skull base and widespread, carpetlike dural involvement, visual impairment, and proptosis. Quantitative examinations of visual acuity, visual field, ocular motility, and degree of proptosis are important to evaluate the patient and the outcomes of surgery. Detailed CT and MRI studies are required for accurate diagnosis and planning of treatment. Options for management include careful clinical and radiographic observation, and surgical resection. Radiation therapy is typically reserved for progressive disease or recurrence following surgery.

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Chapter 27

Cerebellar Convexity Meningiomas

Michael E. Sughrue and Andrew T. Parsa

Introduction

The lateral and posterolateral dura of the posterior fossa are relatively uncommon sites of origin for meningiomas, representing ~10% of meningiomas of the posterior cranial fossa. These meningiomas are diverse, and depending on how extensive the lesion is, the complexity of surgical removal can range from a straightforward suboccipital craniectomy and dissection of the tumor's arachnoidal plane away from the cerebellar convexity, to a complex and extensive removal of the transverse and sigmoid sinuses and adjacent bone. We feel that preoperative planning is especially critical for successful and safe removal of these tumors. In this chapter, we describe our strategy for systematically preparing a surgical plan for addressing these lesions and provide some insight into methods of removing these meningiomas effectively.

Definition

We define cerebellar convexity meningiomas as tumors that possess a dural base lying completely or mostly under the lower occipital and suboccipital bone that overlies the convexity of the lateral or posterior hemisphere. Superiorly, the tumors are bounded by the transverse sinus, laterally by the sigmoid sinus, medially by the occipital sinus on the midline, and inferiorly by the circular sinus and foramen magnum. It is important that the location of the tumor's dural base is delineated in its entirety, so that the tumor's dural and bony attachments can be removed and hyperostotic bone drilled, if possible. Other related meningiomas that are similar in appearance to the untrained eye are the posterior petrous meningioma, the tentorium meningioma, and the foramen magnum meningioma. These tumors are compared in **Fig. 27.1**. Differentiating these lesions is important because the best approach for removing them differs between the locations. Often, more extensive tumors can extend into these areas from the cerebellar convexity dura, and in these cases, identifying the likely site of origin preoperatively can greatly facilitate devascularizing tumors early and achieving complete tumor removal.

Important Issues to Determine Preoperatively

Where Is the Tumor Primarily Based?

The exact site of origin on the cerebellar convexity affects the positioning, the skin incision, the ideal placement of the bone and dural flaps, and the reconstruction. For simplicity, these tumors can be divided into three basic types (**Fig. 27.2**), medial, lateral, and multicompartmental.

Medial

Medial meningiomas occupy the region of the posterior cerebellar convexity dura from the midline to the midway point between the occipital and sigmoid sinuses and are best approached through a conventional suboccipital craniectomy, with the patient positioned prone. The major concern with these tumors is involvement of the torcula because some of these tumors represent inferior extension of posteriorly positioned falcotentorial or peritorcular meningiomas. If questionable, the sinus patency should be determined with magnetic resonance venography or catheter venography. In most cases, the bony reconstruction of these lesions is straightforward because the large nuchal muscle mass makes this area largely cosmetically invisible.

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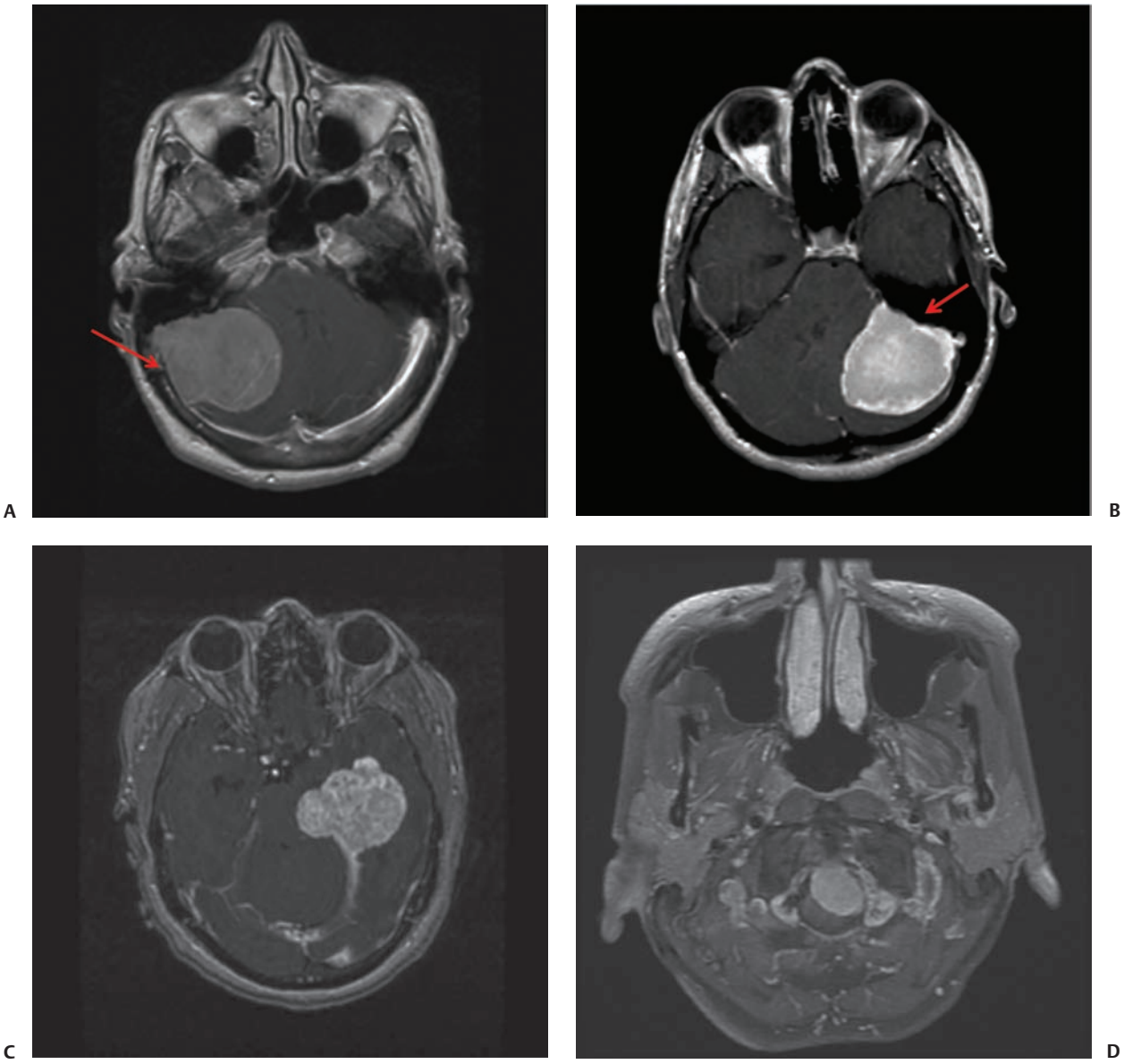


Fig. 27.1 A comparison of the radiographic appearance of **(A)** cerebellar convexity meningiomas with **(B)** meningiomas of the posterior petrous face, **(C)** the tentorium, and **(D)** the foramen magnum.

Lateral

Lateral lesions overlie the cerebellar hemisphere from the midpoint of the cerebellar convexity to the sigmoid sinus laterally, and in many cases involve or even occlude the transverse or sigmoid sinuses. These tumors are best approached with the patient in the full lateral or three-quarter prone position and through some form of a modified retrosigmoid approach, depending on the posterior extent of the tumor and the patency of the sinus. Reconstruction is more important than with the medial tumors because the removal of hyperostotic retromastoid and mastoid bone is cosmetically deforming if not appropriately replaced.

Multicompartmental

Multicompartmental lesions extend into other regions, namely over the posterior petrous face, into the tentorium, across the transverse sinus into the supratentorial space, or even invading cranial nerve foramina. They are included based on their primary point of origin on the cerebellar convexity dura and not primarily by the regions they extend into. These complex cases can vary significantly, and preoperative planning is critical. Reconstruction can be complex, especially when surgical removal of hyperostotic occipital or temporal bone is necessary.

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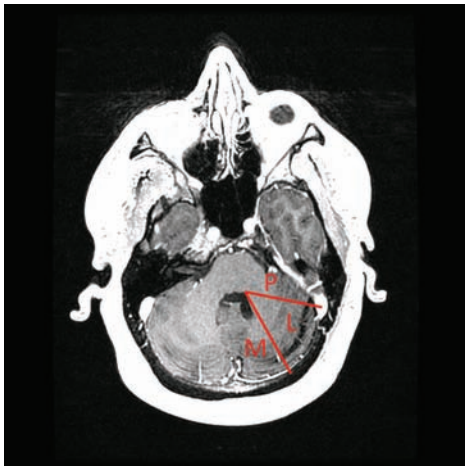


Fig. 27.2 T1-weighted magnetic resonance imaging of a normal cerebellum, which schematically demonstrates the approximate anatomical boundary between medial (M), lateral (L), and petrous (P) meningiomas.

Are the Sinuses Involved, and If So Are They Patent?

The patency of the sinus (or its occlusion) should be known if the tumor is at all near the sinuses (**Fig. 27.3**). A lack of sinus patency makes sacrifice of the involved occluded segment of sinus a possibility; however, complete occlusion should confirmed with catheter venography before sacrificing the sinus.

Where Does the Vein of Labbé Insert on the Sinuses?

The vein of Labbé can drain into the transverse–sigmoid system on the transverse sinus, at the transverse–sigmoid junction, or on the superior petrosal sinus. Knowing the exact location of the insertion of this vein is critical if the transverse or sigmoid sinus is going to be sacrificed in cases of multicompartmental tumors, if an extension of bone removal beyond the suboccipital convexity is going to be used, or if any tumor extension into the supratentorial space is going to be addressed at the same surgery.

Where Is the PICA Relative to the Tumor?

Arterial encasement is uncommon in these tumors; however, encasement of the distal hemispheric segments of the posterior inferior cerebral artery (PICA) should be known preoperatively so that internal debulking of the tumor can proceed safely. Usually with lower-grade tumors, there is an arachnoidal plane internally surrounding the artery, which can be exploited to peel the tumor off the artery; however, this is not constant, and should not be expected with higher-grade tumors.

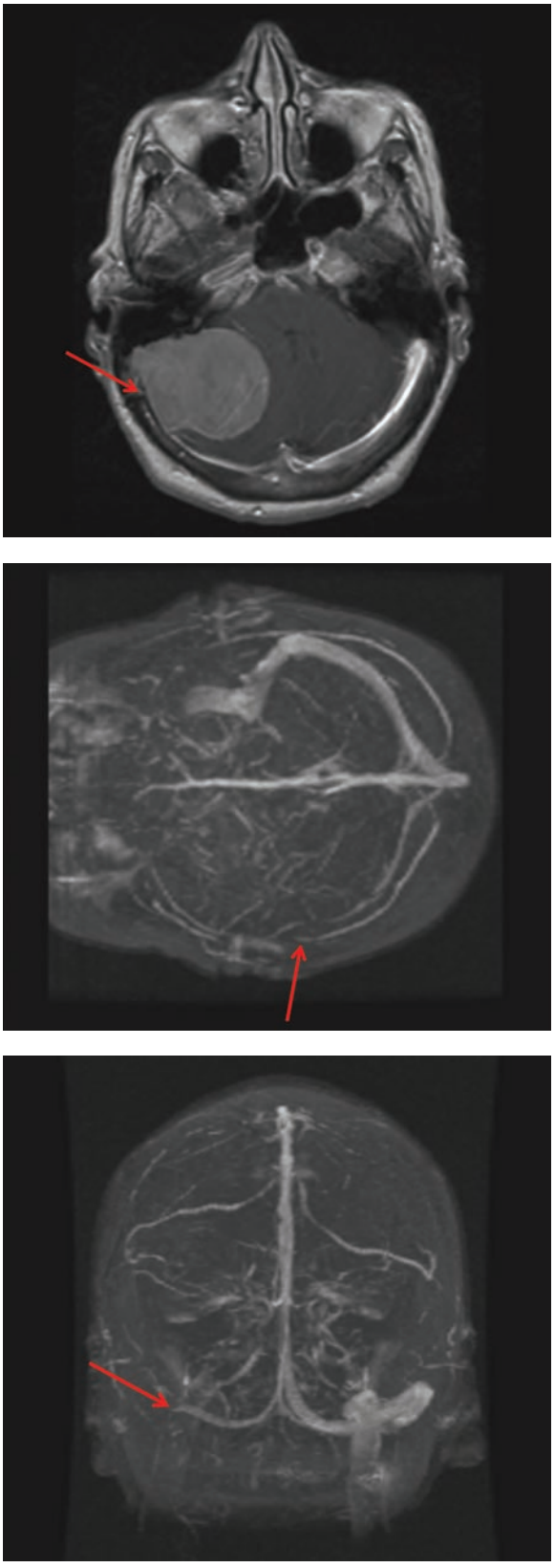


Fig. 27.3 Magnetic resonance imaging of a lateral cerebellar convexity meningioma and magnetic resonance venography from a basal and lateral view demonstrating occlusion of the sigmoid sinus.

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Is There Significant Intrinsic Cerebellar or Brain Stem T2 Signal?

This finding suggests intraaxial edema and increases the risk to the cerebellum beyond the margins of the tumor during dural opening and during dissection of the tumor–cerebellar interface. In these cases, it is probably wise to open the dura over a cerebrospinal fluid (CSF) cistern and drain CSF early in the case and to use the operating microscope for dissection of the tumor–cerebellar interface. The cistern magna is a good choice of a CSF space to be opened early for decompression of the posterior fossa in many of these cases.

What Is the Tumor's T2 Signal Appearance?

Darker T2 appearance on magnetic resonance imaging (MRI) implies more fibrous, less easily suctioned tumors, which may be riskier and more difficult to remove, especially when the tumor extends into the vicinity of cranial nerves (such as the posterior petrous face extension).

Is There Hydrocephalus?

With significant hydrocephalus, a temporary or permanent CSF diversion procedure may be necessary before definitively addressing the meningioma. This treats the secondary effects of elevated intracranial pressure (papilledema, reduced intracranial compliance) and helps prevent postoperative CSF leakage.

Decision Making

Observation should be considered in cases where the tumors are less than 2 cm in maximum diameter and are discovered incidentally without documented growth. For smaller tumors where there has been documented growth and the patient has no clinical symptoms referable to the tumor mass, radiosurgery can be considered as a treatment option. In our experience, this is relatively uncommon, given that these tumors frequently grow to an impressive size before causing specific symptoms prompting an imaging study. Further, these modalities do not provide a tissue diagnosis, and given the ~10 to 15% of meningiomas that are higher-grade tumors (World Health Organization [WHO] grade II or III), and the possibility that the tumors might represent malignant pathologies mimicking meningioma, nonsurgical treatment should be undertaken only with close follow-up. Masses that grow on serial imaging studies should undergo surgical excision, especially if growth continues after radiosurgical treatment.

Preoperative catheter angiography should be performed in nearly all of the cases where the tumor is large (> 3.5 cm). Not only can significant dural, and sometimes pial, feeding arteries be embolized, greatly simplifying surgical debulking and resection, but also the patency of the nearby venous sinuses can be definitively assessed. The latter feature is essential if sacrifice of the sinus is contemplated, given that magnetic resonance venography may not be definitive or correct.

Surgical Technique

General Preoperative Issues

Image guidance is invaluable in these cases to plan the position and extent of craniectomy/craniotomy performed. Even if one is using a system that provides for anterior skin surface–based registration, it is typically necessary to perform the imaging study with convexity, parietal eminence, and mastoid region fiducials, given the often inaccessible location of the face in these positions. It is important to avoid overflexing the head or positioning the head below the neck in these cases, especially when one or more venous sinuses are occluded. When there is significant intraaxial T2 signal, it is wise to give a dose of mannitol (1 g/kg) at the time of skin incision to reduce intracranial blood volume/pressure and thereby assist with safe dural opening. The relevant accuracy of image guidance should not be affected by this maneuver.

Surgery for Medial Cerebellar Convexity Meningiomas

- Patient position
  - Prone, head flexed on the neck, no rotation, neck extended on chest
- Option
  - Semisitting, head flexed on the neck, no rotation
- Craniotomy
  - Small tumors < 3 cm, suboccipital craniotomy/craniectomy
  - Large tumors > 3 cm, suboccipital craniotomy/craniectomy + supratentorial craniotomy/craniectomy

In most cases, medial tumors are best removed through a conventional suboccipital craniectomy. A linear incision is best; however, one should consider the lateral extent of the tumor and prep and drape for the possible need to extend the incision in the rostrocaudal plane to facilitate lateral soft tissue dissection or to use a U-shaped scalp incision. Care should be taken incising the skin to prevent injury to the occipital pericranium, which should be harvested as a large pericranial patch.

It is usually wise to continue the craniectomy down through the foramen magnum so that CSF can be drained from the cistern magna early in the dural opening. If the tumor remains entirely above the posterior foramen magnum, then C1 laminectomy is usually unnecessary. It is usually not possible to perform the craniectomy as a single bone flap, due to the position of these tumors.

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Further, the need to remove the hyperosteotic bone usually makes any preserved bone flap of minimal use. In most cases when there is hyperostosis, we delineate the expected borders of the tumor–dural interface with image guidance and use a cutting burr to drill circumferentially around the tumor down to the inner table of bone. The trough is then completed with a Kerrison punch. If possible, the bone is removed from the dura using sharp dissection with a small periosteal elevator or the cupped end of the No. 1 Penfield.

As already stated, obtaining brain relaxation by draining CSF from the cistern magna is wise at this point. The circular sinus is divided using Weck clips, and the dural incision is continued around the circumference of the dural base. Usually the occipital sinus is occluded in these cases, but if not this should also be sacrificed methodically using Weck clips. After the dural base is incised, the tumor frequently delivers itself enough for one to enter the tumor capsule from the side, leaving the dural base attached or along the posterior midline of the tumor if the convexity dura is excised separately, and one can begin internally debulking the tumor. As soon as the tumor has been debulked enough to fold the surfaces inward, the tumor–brain interface should be inspected to identify the PICA bilaterally with larger tumors, and the vessel can be freed from the tumor using sharp dissection. Sharp dissection can be used to remove the tumor, usually in a piecemeal fashion, until the last remnant is removed en bloc. After repairing the dural defect with pericranium, cranial reconstruction can be performed using mesh and methylmethacrylate for regions above and below the nuchal line.

Surgery for Lateral Cerebellar Convexity Meningiomas

- ◆Patient position
  - Supine, head turned to opposite side
  - Full lateral, head turned toward floor, shoulder taped down
  - Three-quarter prone, head turned toward floor, shoulder taped down
- ◆Craniotomy
  - Retrosigmoid
  - Extended retrosigmoid
  - Retrolabyrinthine plus retrosigmoid
  - Modified far lateral, retrosigmoid

Many of the small to medium-sized tumors can be approached through an extended retrosigmoid craniectomy, with the extent of bone resection determined using image guidance. Usually a full lateral position is sufficient; however, the three-quarter prone position may be needed for more posteriorly located lateral tumors or tumors > 5 cm. A C-shaped incision, with the apex centered ~1 cm behind the asterion, is superior to a linear incision because a larger pericranial flap can be harvested with the former incision. A modified far lateral approach may be used for the largest lateral tumors, allowing access to the cervical subarachnoid space for release of CSF.

The exact strategy for craniectomy in these cases varies significantly depending on the presence or absence of venous sinus involvement and the patency of the sinus. Lateral cerebellar meningiomas without sinus involvement can be resected in a manner similar to medial meningiomas, namely circumferential bone removal around the tumor base, dural incision, internal debulking, and sharp arachnoidal dissection. It is usually wise to skeletonize and to identify the transverse and sigmoid sinuses, which are boundaries of the posterior fossa dura. Cases of nonocclusive sinus involvement make it difficult to achieve gross total resection, and we generally leave tumor in the sinus, follow the tumors with imaging, and perform radiosurgery if interval growth is documented on serial imaging.

In cases of transverse sinus occlusions, it is usually necessary to first perform a small occipital craniotomy just superior to the transverse sinus, which is replaced at the end of the case. After opening of the supratentorial dura, the sinus can be surrounded and suture ligated by placing two 2–0 silk sutures through the tentorium and convexity dura on each side of the affected portion of the sinus, then tying them over the patent part of the sinus to occlude it. The sinus is then cut between the ties and removed, along with the affected portion of the tentorium, the tumor, and its involved posterior fossa dura. It is critical that the exact status and insertion of the vein of Labbé be clearly established before sacrificing the lateral portion of the transverse sinus. The supratentorial craniotomy allows for visual confirmation of the entry point of the vein into the tentorium just medial to the transverse sigmoid junction. Intraoperative Doppler ultrasonography can be used as an adjunct to confirm the position if either of the sinuses is occluded.

In cases where the sigmoid sinus is occluded, we generally find the addition of limited retrolabyrinthine drilling to be helpful to skeletonize the sinus and expose the presigmoid dura anterior to the affected portion of the sinus. For high sigmoid lesions, usually complete exposure of the labyrinths and facial nerve is unnecessary; thus, this procedure can be performed without the assistance of a neurootologist; however, mid- and lower-sigmoid lesions probably require a formal retrolabyrinthine approach. Again, the vein of Labbé should be characterized before sinus sacrifice.

Proper reconstruction of the resulting defect is cosmetically important and might prevent occipital headaches by preventing muscular adherence to the dura. It is usually best to first use a paper cut-out of the defect to form a template, which is used to mold the mesh and methylmethacrylate to the surface cranial defect. Further, it is important to vigorously wax the mastoid air cells or cover the opening with a fat graft to obliterate dead space caused by a retrolabyrinthine approach. If either the distal transverse sinus or any portion of the sigmoid sinus is excised, it is virtually impossible to get a watertight dural closure. Cases with preoperative hydrocephalus might benefit from temporary CSF diversion with a ventriculostomy to help the pericranial graft heal.

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Surgery for Multicompartmental Cerebellar Meningiomas

The potential variations of these lesions are numerous, and discussing all variations in detail is beyond the scope of this chapter. It is important to completely catalog every portion of the tumor, including involvement of the tentorium and supratentorial or middle fossa extension, involvement of the cranial nerves or invasion of cranial nerve foramina, and involvement of the torcular and superior sagittal sinus. These lesions require careful preoperative planning and the selection of an approach that addresses all portions of the tumor. In some cases, subtotal resection with observation or radiosurgery, or both, for the remnant may be necessary. Cases with extensive bony involvement can require complex reconstructive efforts to achieve good cosmetic results.

Conclusion

Meningiomas of the cerebellum convexity vary in complexity, from straightforward tumors that are readily amenable to gross total resection, to those that require more complex approaches and for which subtotal resection and radiosurgery are probably the best option. Proper planning is an essential part of successful and safe surgery for these tumors. This chapter provides a framework for surgical decision making and planning for these tumors, many of which probably require the assistance of a neurootologist to properly treat. Importantly, in our opinion, it is wise to identify cases when total tumor removal is not best, and to prepare the patient for the need for planned subtotal resection and adjuvant treatment.

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Chapter 28

Cerebellopontine Angle Meningiomas

Madjid Samii and Venelin M. Gerganov

Introduction

The first successful complete removal of a cerebellopontine (CP) angle tumor was performed on November 19, 1894, by Sir Charles Balance in London. At the first stage, a right posterior fossa craniectomy was performed, and 1 week later the tumor was removed with the finger inserted in an unsterile fashion between the pons and the tumor. The tumor was found to be firm and well encapsulated, and it had a wide attachment to the dura of the petrous bone. According to Cushing, this tumor was most probably a meningioma.[1] In 1928, Cushing and Eisenhardt[2] described their experience with the surgical management of seven patients with meningiomas "simulating acoustic neuromas." During the following decades, surgery of CP angle tumors fascinated and attracted many of the most outstanding neurosurgeons. New operative approaches and management concepts were introduced that allowed dramatic reductions in mortality and morbidity rates. Gradually, the focus changed from performing "life-saving" to "function-preserving" surgeries. Nowadays, surgery of tumor in the CP angle is safe, major morbidity is exceptional, and preservation of the function of the facial and cochlear nerves is the rule.

Classification

Meningiomas arising from the dura of the posterior surface of the pyramid lateral to the trigeminal nerve are defined as "CP angle meningiomas."[3] We consider those meningiomas that arise from adjacent areas but have their main bulk in the CP angle also as CP angle meningiomas. Meningiomas are the second most frequent tumor of the CP angle after vestibular schwannomas and comprise 6 to 15% of all CP angle tumors.[4–6]

The variable attachment sites and direction of growth determine the high heterogeneity of CP angle meningiomas in terms of clinical presentation and operative challenge. The two main groups are the premeatal and retromeatal meningiomas, determined according to their relation to the internal acoustic meatus (IAM).[3] This classification has operative and prognostic significance—the more medially located tumors are related to greater surgical complexity and to poorer outcome. We subdivide CP meningiomas further into premeatal, postmeatal, suprameatal, inframeatal, and centered at the IAM.[7] As presented in a previously published evaluation of the senior author's experience with CP angle meningiomas,[7] 33% of them originated at the petrous ridge anterior to the IAM (mean tumor diameter 3.1 cm), 20% originated superior to the IAM (mean tumor diameter 3.4 cm), 12% inferior to the IAM (mean tumor diameter 4 cm), 13% posterior to the IAM (mean tumor diameter 4.1 cm), and 22% with involvement of the IAM (mean tumor diameter 3.4 cm). A specific pattern of dislocation of the seventh and eighth cranial nerves is observed in each of the subgroups.

According to their extension pattern, CP meningiomas can be classified as follows:

- Postmeatal meningiomas
  - Without extension into the IAM
  - With extension into the IAM
- Premeatal meningiomas
  - Medial and superior extension (with or without extension into the Meckel cave, supratentorially, or into the IAM)
  - Medial and inferior extension (with or without extension into the jugular foramen or into the IAM; extending to the level of the foramen magnum or not)
  - Combination thereof
- Large meningiomas with pre- and postmeatal extension

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Neuroimaging

Tumor features and extension are best appreciated on contrast-enhanced magnetic resonance imaging (MRI). On T1-weighted MRI studies, meningiomas are mostly isointense to slightly hypointense relative to brain parenchyma.[4] On T2-weighted MRI studies, they show higher intensity than that of vestibular schwannomas. Contrast enhancement is usually homogeneous in both meningiomas and vestibular schwannomas. The radiological differential diagnosis between CP angle meningioma and vestibular schwannomas is based on several criteria.[8,9] Usually, meningiomas are centered away from the IAM and have broad contact with the petrous bone or the tentorium. The angle between the tumor and the pyramid is obtuse and the IAM is not widened. Although secondary invasion of the IAM might be observed in 10 to 22%, primary IAM meningiomas are very rare.[10,11] A tail of enhancement along the dura (the dural tail sign), although not pathognomonic, is visible in 60 to 72% of meningiomas. In contrast, vestibular schwannomas are centered at the widened IAM, form an acute angle with the posterior surface of the petrous bone, and almost always extend into the IAM. Thin-slice computed tomography (CT) with bone window settings is essential for the approach planning and provides information on the degree of bone involvement, whether there is erosion or hyperostosis, and the presence or absence of tumor calcification. Magnetic resonance angiography (MRA) and venography (MRV) demonstrate the degree of tumor vascularity. Digital subtraction angiography as a diagnostic and/or planning tool is required in exceptional cases, such as giant tumors with wide extension and involvement of main vessels. Preoperative embolization of tumor feeding arteries may be very helpful in highly vascular meningiomas.

Clinical Presentation

Meningiomas cannot be distinguished from other CP angle tumor types based on their clinical presentation alone. Common findings are hearing loss, vertigo, headache, and trigeminal and cerebellar symptoms.[6,10,12–14] Symptoms and signs secondary to brain stem and cerebellar compression with obstructive hydrocephalus occur late.

Premeatal and retromeatal meningiomas have distinctive clinical development and symptomatology.[3,5,7,15] Premeatal tumors are diagnosed earlier and consequently have smaller size. Their clinical presentation is with trigeminal symptoms (numbness and/or trigeminal neuralgia) and facial and cochlear nerve signs. Typical for the larger retromeatal meningiomas are cerebellar signs and symptoms. Patients with large meningiomas that extend anterior and posterior to the IAM can present with a combination of these symptoms.

Management

Treatment options for CP angle meningiomas are observation, surgery, radiotherapy/radiosurgery, or a combination thereof. In each case the decision should be taken individually, considering the patient's biological age, expectations, activities, and general and neurological status, as well as the tumor's size and extension. The wide availability of MRI allows earlier meningioma detection during the "preclinical" stage and is a reliable method for control of tumor growth. Initial follow-up and surgery are frequently recommended only when the tumor shows growth or new symptoms appear. On the other hand, earlier active management is justified because the outcome of surgery is related to tumor size and is optimal in neurologically intact patients. We recommend observation as the initial treatment option in elderly or medically unstable patients with small tumors and mild stable symptoms or in those unwilling to undergo surgery.

Complete operative tumor removal is the optimal treatment of CP meningiomas but should not be achieved at the expense of new neurological dysfunction or deterioration of the patient's quality of life. Different surgical approaches are used for resection of CP angle meningiomas: retrosigmoid, extended retrosigmoid, translabyrinthine, transcochlear, transpetrosal, and modified far lateral, as well as approaches through the middle cranial fossa.[4,7,12,14,16] However, the general concepts that are important for the successful removal of most meningiomas are similar: adequate exposure, interruption of the blood supply along the dural attachment, internal decompression, and cautious dissection of the tumor capsule from the brain stem and cranial nerves in the arachnoid plane.

In the early ages of the skull base surgery, an extensive bony exposure allowing complete visualization of the meningioma and all adjacent neural and vascular structures was considered a major prerequisite for complete and safe tumor removal. Approaches providing such exposure, however, have higher risks of complications, including a higher rate of facial nerve palsy and venous-related complications.

Another important issue consists of surgeons' attempts to preserve hearing. Some authors suggest that hearing preservation should be attempted only in retromeatal meningiomas.[15] Hearing-destructive approaches through the pyramid have been proposed for patients with severe hearing deficits. In CP angle meningiomas, contrary to vestibular schwannomas, hearing loss is usually due to compression of the cochlear nerve. Hearing improvement is often observed after tumor removal and has been reported even in cases of large meningiomas.[7,17,18]

Having extensive experience with all skull base techniques, the senior author believes that simpler and safer approaches are the prerequisite to improving outcome and reducing morbidity. The retrosigmoid suboccipital approach (RSA) offers an excellent panoramic visualization of the whole CP angle, wide exposure of the tumor (whatever its size), and increased safety when working in the vicinity of the brain stem. It has a very low procedure-related morbidity rate and allows hearing preservation. For CP meningiomas, the RSA is our favorite technique. In CP meningiomas with medial extension toward the petroclival area or the Meckel cave, we utilize the retrosigmoid suprameatal approach (Samiis technique), introduced by the senior author in 1982.

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Surgical Approaches

Retrosigmoid Approach

Although surgeries are performed successfully with various patient positions on the operating table, we prefer the semi-sitting position. It has the important advantage of allowing bimanual manipulations because there is no need for constant suction. Furthermore, the continuous irrigation of the operative field performed by the assistant obviates the need for frequent coagulation during tumor removal.

The head is flexed and rotated ~30 degrees to the involved side, avoiding occlusion of venous jugular outflow or hyperflexion of the cervical spine. A drawback of this patient position is the risk of venous air embolism, paradoxic air embolism, tension pneumocephalus, or circulatory instability. However, in experienced hands these are not related to any lasting morbidity.[19,20] Continuous neurophysiological monitoring—somatosensory evoked potentials, facial nerve electromyography (EMG), and auditory brain stem response (ABR)—should be performed throughout the surgery, from the positioning of the patient to the skin closure.[21–23] Facial nerve motor evoked potential monitoring is a recently introduced, very promising technique.[24] The abducent and caudal cranial nerves are monitored as needed according to the particular tumor extension and clinical presentation.

A slightly curved skin incision 2.5 to 3.5 cm medial to the mastoid process should allow sufficient access to the mastoid tip. Considering the fact that the asterion is not a reliable anatomical landmark, we place the burr hole 2 to 2.5 cm below the superior nuchal line, two thirds behind and one third in front of the occipitomastoid suture.

The craniectomy is performed most safely using bone rongeurs or a high-speed cutting burr, and the edges of the sigmoid and transverse sinuses are exposed. More extensive exposure of these sinuses is unnecessary and might lead to their laceration or desiccation, with the risk of subsequent thrombosis. The bone opening should extend caudally to the floor of the posterior fossa. Special care should be directed toward preservation of the mastoid emissary vein. Therefore, the vein should be skeletonized with a diamond drill until it is free of any bony encasement and can be safely coagulated.

The dura is incised in a curvilinear manner just 1.5 to 2 mm medial to the sigmoid and inferior to the transverse sinus. This allows for a primary watertight dural closure and avoids the need for a dural substitute in almost all cases. The lateral cerebellomedullary cistern is then opened, and sufficient cerebrospinal fluid is drained. Thus the cerebellum relaxes away from the petrous bone, and the self-retaining retractor supports and protects the cerebellar hemisphere instead of compressing it. With this technique, any retraction-related injury of the cerebellar hemisphere can be avoided.

CP meningiomas may exhibit varying extension into the CP angle, engulfing or dislocating the neurovascular structures. Individual tumor characteristics, such as size and extension, determine the subsequent steps.

Postmeatal meningiomas (meningiomas located between the sigmoid sinus and IAM) can be safely removed because they usually displace the facial and cochlear nerves anteriorly (63%) and inferiorly (25%).[7] Initial internal decompression or debulking is performed with a Cavitron Ultrasonic Surgical Aspirator (Integra Neurosciences, Plainsboro, NJ), suction, or platelet knife. Thereafter, the tumor capsule can be relatively easily dissected from the neural structures, which are generally protected by an arachnoid sheath.

In *premeatal meningiomas* the facial and cochlear nerves are most commonly displaced posteriorly and inferiorly (45% and 43%, respectively). The surgeon must approach the tumor between the cranial nerves via one of the CP angle levels, as viewed from a retrosigmoid perspective: the upper level between the tentorium and the trigeminal nerve, the second level between the trigeminal and the facial and cochlear nerves, the third level between the facial and cochlear nerves and the caudal nerves, and the lowest level, defined by the caudal nerves and foramen magnum. Although anatomically the space between the cranial nerves may be relatively narrow, the meningioma usually widens it and provides an adequate working corridor. Tumor removal should start initially at the most expanded level. The dissection of the tumor capsule is performed only after sufficient internal decompression has been achieved. Then the surgeon moves to the next level. Dissection is performed usually from the lateral to medial direction: from the bone toward the brain stem. The cranial nerves are thus identified earlier near their entrance or exit in bone or dura. Their dissection is performed by carefully stripping off the arachnoid from the tumor with one hand. Simultaneously, a slight traction is done with the second hand. Bipolar coagulation is used only to control major bleeding vessels and is avoided in the vicinity of the cranial nerves. In very rare cases, the meningioma infiltrates the facial nerve. Such patients have preoperative facial nerve palsy and hearing loss. In cases of profound facial weakness, to achieve complete tumor removal, the nerve can be sacrificed and reconstructed primarily in the CP angle with an interposition graft.

Some premeatal meningiomas expand more cranially toward the petroclival junction and might extend into the Meckel cave or supratentorially (see the next section, Retrosigmoid Suprameatal Approach). Other meningiomas extend caudally toward the foramen magnum or occasionally into the jugular foramen and are approached via one of the lower CP angle levels (**Fig. 28.1**). Dissection from the caudal cranial nerve should be performed with utmost care. With this approach the abducent nerve can be identified early at its brain stem exit zone and followed during tumor removal up to the Dorello canal, thus reducing the risk of its injury. From a lateral perspective, such as the one provided by the Kawase approach, the nerve is hidden by the tumor and can be identified at a later stage. The chances of its preservation are respectively lower. Meningiomas extending to the foramen magnum level can be removed via a similar retrosigmoid craniectomy and a C1 hemilaminectomy/laminectomy.[23]

Both pre- and retromeatal meningiomas might extend into the IAM (**Fig. 28.2**). Exposure of the intrameatal

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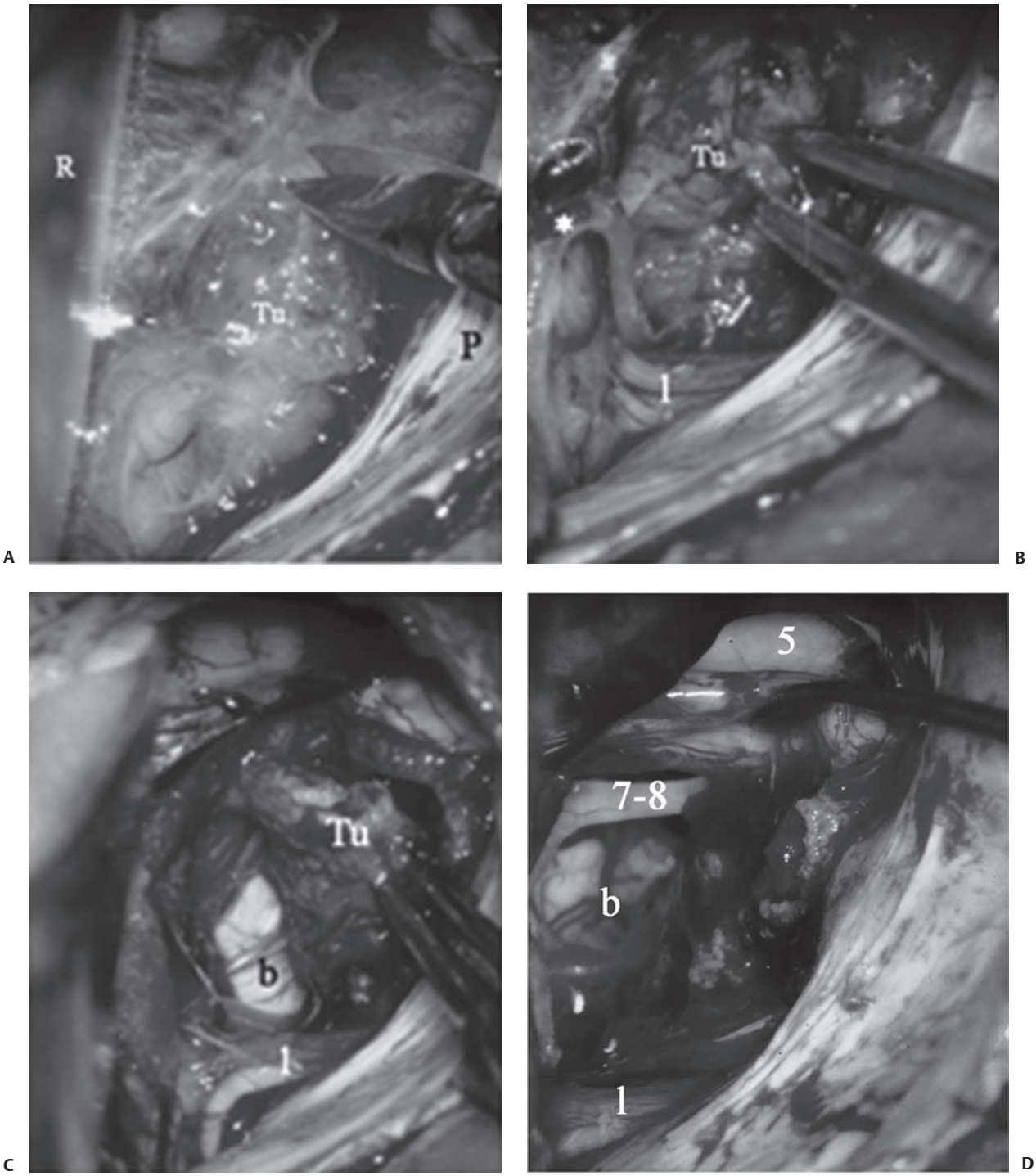


Fig. 28.1 Cerebellopontine angle meningioma arising inframeatally with posterior extension. The CN VII–VIII complex is pushed anteriorly, while the lower cranial nerves are pushed caudally. The tumor is removed through the space between the CN VII–VIII complex and the lower cranial nerves. **(A)** Initial view. All cranial nerves are hidden by the tumor. **(B)** After internal decompression and partial tumor removal, the lower cranial nerves (l) and the posterior inferior cerebral artery (PICA) (*) are visualized. **(C)** Further tumor removal, the lower cranial nerves are free. **(D)** Complete tumor removal, preserved CN V, VII, and VIII and lower cranial nerves. Tu, tumor; b, brain stem; R, retractor; P, pyramid; 5, trigeminal nerve.

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part requires opening the posterior wall of the IAM. The amount of bone drilling is defined by the extension of the tumor.[11] In such meningiomas the facial and vestibulocochlear nerves are either surrounded by the tumor or displaced superiorly or inferiorly. A combination of the aforementioned techniques has to be used in case of large CP meningiomas that extend both pre- and retromeatally.

The wide variability of the venous system in the CP angle does not allow predicting the consequences of an interruption of the petrous or other main draining veins. Therefore, every attempt should be made to preserve their integrity.[25] Peritumoral edema, visible as a low-intensity signal on T1-weighted MRI and as a high-intensity signal on T2-weighted MRI, frequently reflects tumor invasion of the pia or of the brain. In such tumors, great care must be exercised during dissection to avoid injury to the brain stem, and the most adherent part of the capsule *should not* be removed. The dural origin of the tumor should be excised or, if excision is not possible, coagulated meticulously. A high-speed drill is used to remove any bone hyperostosis, which may contain viable tumor cells.

Retrosigmoid Suprameatal Approach

The retrosigmoid suprameatal approach (RSMA) avoids the risks related to alternative approaches to the petrous apex, such as extensive petrous bone resection or retraction of the temporal lobe with the associated risks of damage to neural and vascular structures. It comprises a simple retrosigmoid craniotomy and intradural resection of the petrous apex. Following removal of tumor bulk in the CP angle, the bone located above and anterior to the IAM—the suprameatal tubercle—is drilled off, providing access to the petroclival area and Meckel cave. The amount of bone resection depends on the individual anatomical characteristics and the tumor extension. The exposure provided by the classical retrosigmoid approach (RSA) can be thereby extended as far as 13.0 mm anterior.[26] Opening the Meckel cave allows for mobilization of the trigeminal nerve, which further increases the working space.[27] Great care to avoid inadvertent damage to the internal carotid artery anterolaterally, the petrosal sinus superiorly, and the superior and posterior semicir-

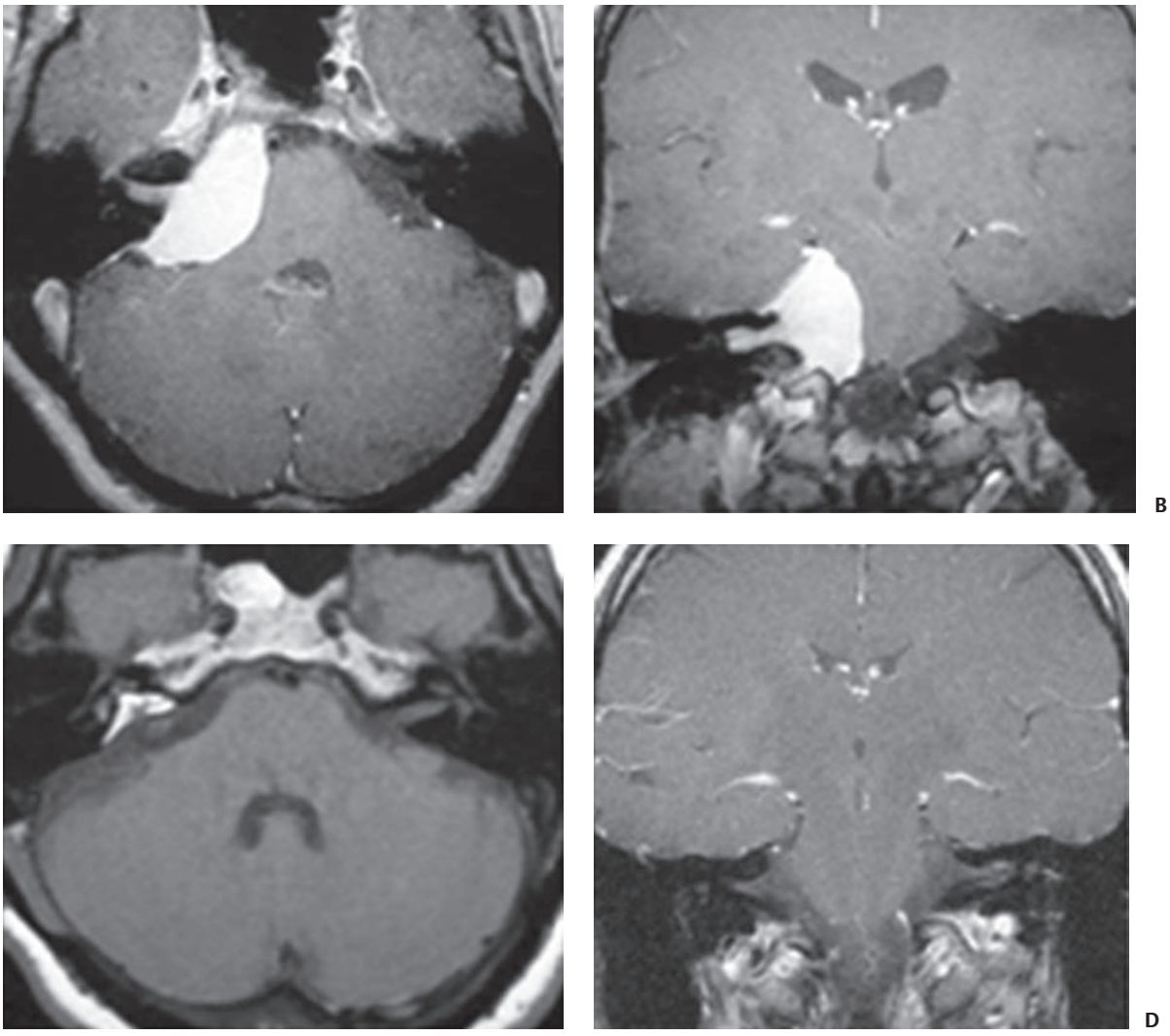


Fig. 28.2 **(A,B)** Preoperative and **(C,D)** postoperative T1-weighted magnetic resonance imaging of a meningioma with extension into the internal acoustic meatus. The fat tissue used to seal the opened IAM is seen as a hyperintense signal.

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cular canals laterally.[28] The exposure of the rostral tumor portion can be further enlarged by resection of the tentorium. Thus the RSMA provides access to the Meckel cave, petroclival area, and middle fossa, including the posterior cavernous sinus (**Fig. 28.3**). In the case of large meningiomas, the brain stem is displaced to provide access to the contralateral side or supratentorially.

Following tumor removal and thorough hemostasis, the drilled area of the pyramid is sealed with multiple pieces of fat tissue that are fixed with fibrin glue. Any opened mastoid air cells are occluded in a similar way. Bone wax is avoided, except for hemostasis if there is significant bleeding from the bone edges. If necessary, a piece of fat is applied to the watertight sutured dura. In cases of incomplete resection, follow-up MRI studies are performed 3 to 6 months after surgery and radiosurgery/radiotherapy or follow-up of the residual tumor is recommended accordingly.

Outcome

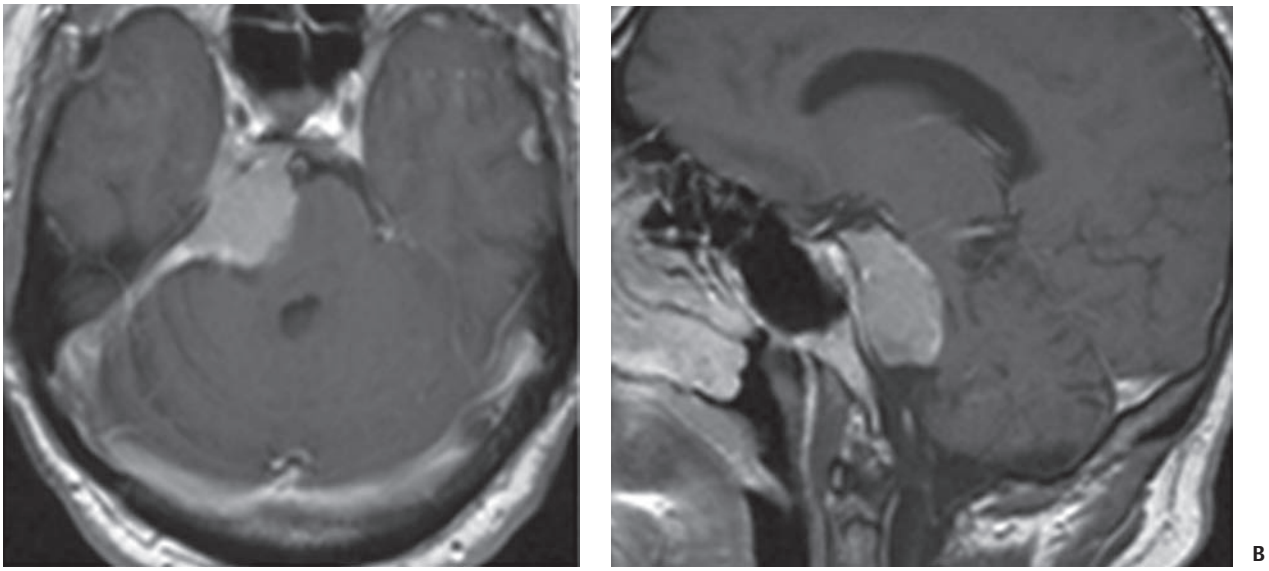
The outcome of surgery depends mainly upon the location and consistency of the tumor, and to a lesser extent upon its size. In premeatal meningiomas, the preservation of facial and auditory function is less likely. Two further important factors are the extent of skull base involvement and the adherence of the tumor to the brain stem, cranial nerves, and vessels.[14] In large recent series, the rate of total removal is above 80% and the mortality is below 5%.[3,4,6,10,13,14,29] Nakamura et al[7] evaluated 421 patients with CP meningiomas operated by the senior author. They found that the "radicality" of tumor removal as well as the functional and general outcome correlated with the topographic location of the tumors. The overall rate of total removal (Simpson grades I and II) was 86%. Considering the tumor subtypes, the highest rate was achieved in the suprameatal (90%), and retromeatal meningiomas (89%), and in those involving the IAM (88%). The rate was lowest in premeatal (83%) and inframeatal tumors (78%).

Currently, facial nerve function can be preserved in the majority of patients.[6,7,10,29] In our series, good facial nerve function (House-Brackmann grades 1 or 2) was observed in 89% of the patients. Normal facial nerve function preservation rate was highest in retromeatal meningiomas (90%) and lowest in the premeatal meningiomas (76%). The rate of preservation of functional hearing increased from 88% (earlier experience[13]) to 91% in the recent series.[7] In 2%, even recovery from preoperative deafness was observed.[7] CP angle meningiomas have a higher tendency to recur as compared with vestibular schwannomas—between 0 and 9,5%.[6,10,14] Therefore, neuroimaging follow-up should be performed regularly. If there is documented recurrence, a second surgery should still be considered an option for treatment.

Radiotherapy or radiosurgery has been proposed as a primary treatment option for small meningiomas and as an adjunctive therapy after incomplete removal.[30] In less than total removal, postoperative radiation therapy is known to prolong the interval to recurrence and to improve survival.

Specific Considerations

Patients with neurofibromatosis type 2–associated meningiomas or with meningiomatosis pose specific management problems. The major issue to be considered is the lifelong propensity for development of new neoplasms and the respective impossibility for definitive cure. Treatment is focused therefore on life prolongation,



A B

Fig. 28.3 **(A,B)** Petrous apex meningioma with extension into the Meckel cave. *(continued)*

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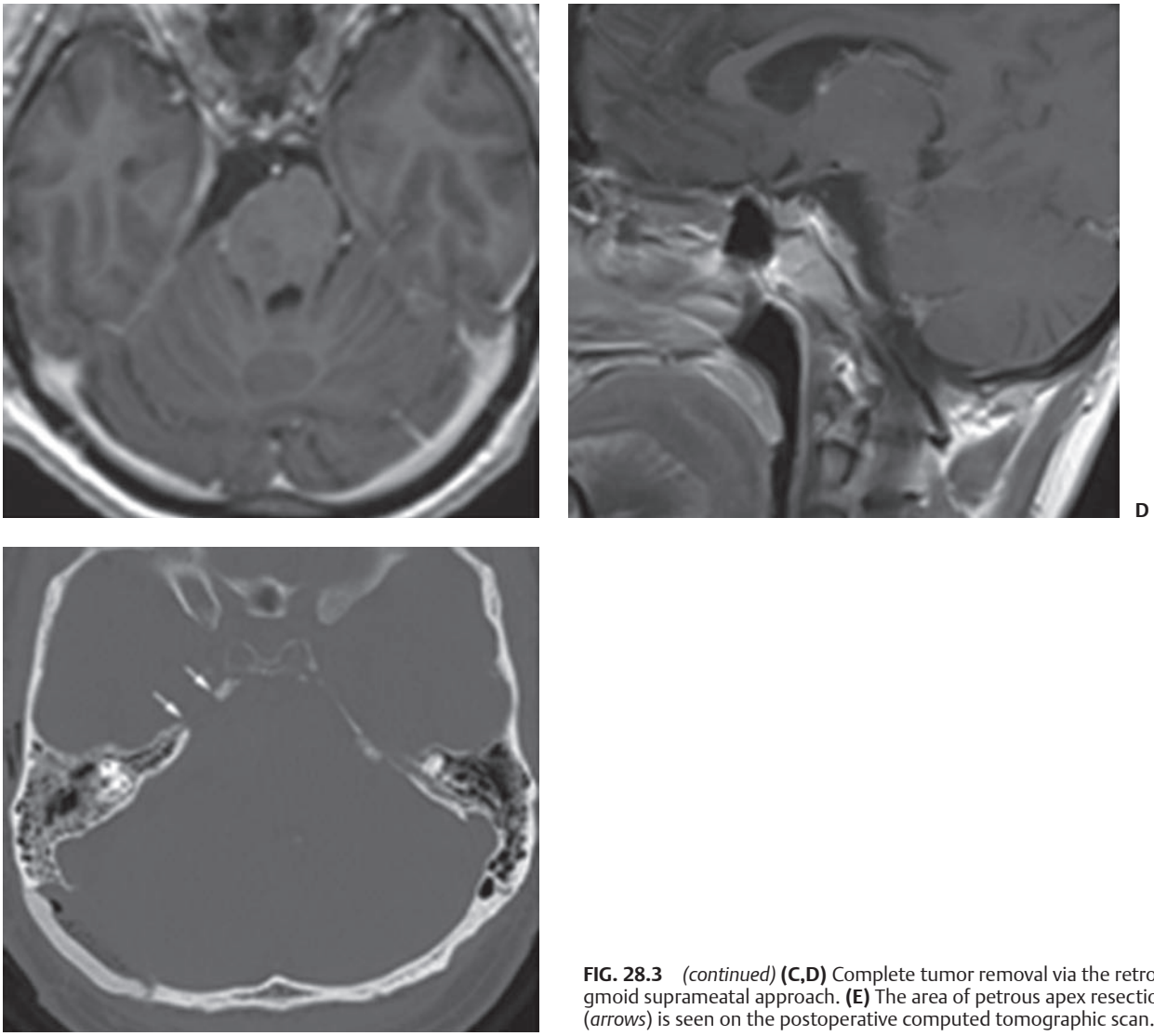


FIG. 28.3 *(continued)* **(C,D)** Complete tumor removal via the retrosigmoid suprameatal approach. **(E)** The area of petrous apex resection (*arrows*) is seen on the postoperative computed tomographic scan.

preservation of cranial nerve function, and maintenance of quality of life. Symptomatic meningiomas, as well as those with a progressive growth tendency, have to be treated surgically. The goal is to achieve decompression of essential neural structures and—if possible—to remove the tumor completely. Preservation of neural function is a priority: in cases where the tumor infiltrates or is very adherent to a cranial nerve, no attempt should be made to remove it completely.

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Chapter 29

Clival and Petroclival Meningiomas

Jeroen R. Coppens and William T. Couldwell

Introduction

Clival and petroclival meningiomas represent ~2% of all intracranial meningiomas.[1] Olivecrona[2] is credited with being the first to attempt surgical resection of these tumors in 1927, but he later deemed them to be inoperable. Early surgical experience with petroclival meningiomas remained dismal, with mortality rates exceeding 50%,[2–4] until the series published in the microsurgical era by Yasargil et al[5] in 1980.

Classification schemes for these posterior fossa meningiomas have evolved since the initial description by Castellano and Ruggiero.[6] Clival meningiomas have a dural attachment close to the midline along the upper two thirds of the clivus and displace the brain stem posteriorly as they enlarge.[7] Petroclival meningiomas also arise from the upper two thirds of the clivus but their dural attachment is centered on the petroclival junction.[7] Their location is medial to the internal auditory meatus and posterior to the gasserian ganglion. As they enlarge, petroclival meningiomas displace the brain stem and basilar artery posteriorly and to the contralateral side. Petroclival meningiomas usually present as large tumors when they become symptomatic, and associated invasion of the posterior aspect of the cavernous sinus, parasellar region, tentorium, or foramen magnum is not uncommon.

Many surgical approaches have been used for the treatment of clival and petroclival meningiomas. The rationale for the use of each approach balances the need to limit brain retraction and allow for good visualization of the neural and vascular structures involved, while limiting any morbidity due to the approach. Early approaches described to remove these tumors were pterional, subtemporal, or suboccipital.[8–10] Skull base approaches have been developed to improve the access to these tumors by removing varying portions of the petrous bone. Anterior petrosectomy as well as various posterior petrosectomy approaches have been defined, some of which offer a presigmoid corridor to the tumor. Depending on the extent of the inferior extension of the tumor, lateral transcondylar approaches may also be necessary. Petroclival meningiomas are difficult tumors to resect, primarily because the cranial nerves are interposed between the surgeon and the tumor and because of the intimate relationship between the brain stem vascular supply and the tumor. Extended skull base approaches may be used to remove the tumor in one surgery, or separate approaches may be staged.

This chapter reviews the presenting symptoms, radiological characteristics, and surgical results of clival and petroclival meningiomas. Results and approaches pertaining to clival meningiomas are included in the description of petroclival meningiomas because they present the same surgical challenges, and current published series have consistently grouped them together.

Symptoms and Signs

Petroclival meningiomas predominantly affect middle-aged and older women, as is true of meningiomas in other locations. Exceptional cases have occurred in the pediatric population (**Table 29.1**). The onset of these tumors is insidious, with patients developing headaches and gait problems. The onset of cranial nerve palsies is usually the first clinical sign, followed by cerebellar and brain stem compression signs. A slow onset of obstructive hydrocephalus may also be possible. Diagnosis is commonly made after the development of a cranial nerve palsy, at which point medical care is sought. Tumor size varies greatly at the time of diagnosis (**Table 29.1**). The majority of patients in the largest studies have presented at the time of diagnosis with tumor diameters of 2 to 4 cm.[11–13] The second most common group are tumors ex-

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Table 29.1 Presenting Signs and Symptoms of Patients with Clival and Petroclival Meningiomas

Study	*N*	Age Range in Years (mean)	Female: Male Ratio	Size Range in cm (mean)	Cranial Nerve Palsies at Time of Diagnosis (%)										Duration of symptoms
					CN III	CN IV	CN V	CN VI	CN VII	CN VIII	CN IX–X–XI	CN XII	Cerebellar	Somato-sensory	
Abdel Aziz et al, 2000[18]	35	NA	NA	NA	6 (17)	3 (9)	19 (54)	9 (26)	4 (12)	12 (35)	9 (25)	2 (6)	16 (46)	8 (23)	NA
Bricolo et al, 1992[8]	33	27–68 (52)	21:12	2–>6	3 (9)	0	22 (67)	7 (21)	10 (30)	13 (39)	15 (45)	3 (9)	20 (60)	10 (30)	7–204 months
Couldwell et al, 1996[11]	109	25–75 (51)	69:40	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
VanHavenbergh et al, 2003[15]	21	36–78	16:5	NA	1 (5)	0	2 (10)	5 (24)	2 (10)	6 (28)	1 (5)	0	5 (24)	0	2 months–10 years
Goel et al, 2004[9]	28	18–68 (38)	15:13	1.8–6.8 (4)	0	0	14 (50)	4 (14)	8 (28)	7 (25)	4 (14)		17 (61)	4 (14)	1 months–2 years
Erkman et al, 2005[17]	97	NA (50)	78:19	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Little et al, 2005[12]	137	4–81 (53)	99:38	NA	29 (21)		42 (31)	*	13 (9)	18 (13)	7 (5.5)	< 1%	19 (14)	16 (12)	NA
Park et al, 2006[26]	49	15–74 (46)	4:1	2.1–7.6 (4.1)	9 (18)		32 (65)	*	12 (24)	27 (55)	14 (29)	0	NA	NA	NA
Wei et al, 2006	25	28–67 (47)	21:4	2–7 (4.5)	3 (12)	0	15 (60)	5 (20)	3 (12)	6 (24)	5 (20)		6 (24)	6 (24)	1–244 months
Mathiesen et al, 2007[19]	29	18–71 (52)	22:7	3–7 (4.4)	4 (14)	3 (10)	10 (34)	3 (10)	6 (21)	9 (31)	5 (17)	0	NA	NA	NA
Natarajan et al, 2007[13]	150	18–76 (51)	121:29	0.79–8.38 (3.44)	36 (24)	21 (14)	64 (43)	43 (29)	16 (11)	46 (31)	10 (7)	4 (3)	27 (18)	16 (11)	NA
Goel 1999[25]	24	18–58 (39)	15:9	2.8–6	0	0	5 (21)	3 (12)	5 (21)	5 (21)	3 (12)	0	NA	NA	15 d–3 years

Abbreviations: CN, cranial nerve; NA, not available.
*Included in CN III–CN IV combined value.

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ceeding a diameter of 4 cm, whereas the discovery of small tumors remains the least common scenario.[11–13]

Cranial neuropathies are commonly present at time of diagnosis. Trigeminal nerve dysfunction is the most common and can present in the forms of trigeminal neuralgia, facial pain, or varying degrees of facial hypesthesia or frank anesthesia (**Table 29.1**). Diplopia, hearing loss, and vertigo are also very common, followed in frequency by dysphonia, facial weakness, and dysphagia (**Table 29.1**). Cranial neuropathies may not be isolated to the posterior fossa, and visual loss may occur with tumors that have significant cavernous sinus involvement through the Meckel cave.

Patients may be symptomatic for prolonged periods of time before the patient is imaged and the diagnosis is made (**Table 29.1**). A low threshold should exist for imaging patients exhibiting compatible symptoms.

Natural History of Petroclival Meningiomas

Initial observational studies of clival and petroclival meningiomas suggested these tumors have insidious continuous growth with ultimately fatal outcomes, based on clinical and postmortem observations.[1,6,14] More recent data derived from studying residual tumor growth after subtotal resection have suggested deaths are caused by tumor growth only in a minority of patients.[8,13] Overall tumor recurrence or progression has been observed in as few as 13% of patients at 6 years in a large series of patients who were surgically treated with the use of adjuvant radiation upon indication of progression.[11]

The authors of some recent observational studies of untreated or subtotally resected petroclival meningiomas have observed growth rates ranging from 0.81 mm/year[15] to 0.37 cm/year.[16] The significance of these differences is unclear but may reflect the small number of cases involved. A great variability in growth patterns exists between different tumors, and growth patterns for individual tumors are not linear.[15,16] An increase in tumor growth rate often precedes a clinical deterioration. An observational study of untreated petroclival meningiomas in 21 patients recorded a radiological tumor growth in 76% of patients at 4 years.[15] This correlated with a functional decline in the majority of patients.[15]

An observational study of subtotally resected petroclival meningiomas demonstrated 42% of tumors showed radiological progression over 4 years with a faster growth pattern.[16] The tumors' mean doubling time was 8 years.[16] Older patients and those who have gone through menopause seemed to have slower-growing tumors.[16]

Tumor growth of petroclival meningiomas remains unpredictable but is not universal. An absence of radiological tumor growth was noticed in 24% of patients over a 4-year observation period in untreated patients.[15] After subtotal microsurgical resection, 58% of tumors did not grow over a period of 4 years.[16] Thus conservative observation may be justified in asymptomatic elderly patients when close radiological follow-up can be performed.

Preoperative Evaluation

Magnetic resonance imaging (MRI) of the brain with and without contrast enhancement remains the examination of choice for diagnosis of clival and petroclival meningiomas. The radiological characteristics of these tumors are similar to those of meningiomas in other locations. They typically present as isointense extraaxial lesions on T1-weighted sequences and are of variable intensity on T2-weighted sequences. Upon gadolinium administration, clival and petroclival meningiomas enhance homogeneously, and a dural tail may be observed (**Fig. 29.1A,B**). Fat suppression imaging techniques may be useful in differentiating tumor involvement of bone from normal marrow in selected cases.

The presence of brain stem edema should be noted and raise concerns for pial invasion, which is reported in about one third of cases (**Table 29.2**). The relationships of the basilar artery, jugular tubercle, cavernous sinus, parasellar region, and internal auditory meatus should be noted. Extension of the tumor into the cavernous sinus seems to be very common (**Table 29.2**). The tumor may also extend past the tentorium cerebelli (**Fig. 29.2**), whereas the involvement of the internal acoustic meatus has not been reported with as much frequency (**Table 29.2**). Extension of the tumor to the midline of the clivus probably occurs in ~20% of cases and makes surgical resection more difficult. The basilar artery appears to be encased in the tumor in many cases, based on MRI criteria, which does not preclude the existence of intact arachnoid planes at time of surgery.

A fine-cut computed tomographic (CT) scan of the skull base can be used to identify extension of the tumor into the internal auditory meatus or Meckel cave as well as any potential bony involvement, the incidence of which has not been consistently reported (**Table 29.2**). An assessment of the exposure provided by various posterior petrosal approaches is possible.

A four-vessel cerebral angiogram may help in the surgical planning to identify venous sinus patency, anatomy of temporal draining veins, and tumor blood supply. Embolization of the tumor necessitates access to direct feeders from the posterior circulation as well as access to the meningohypophyseal trunk of the internal carotid artery. Given the logistic difficulty and associated risks with embolization of meningiomas in this location, the senior author rarely performs this before surgical resection, regardless of the size of the tumor. An angiogram with a balloon test occlusion of the carotid artery may be necessary in cases of young patients with symptomatic cavernous sinus involvement in which a high-flow bypass is contemplated before a radical resection.

Surgical Approaches

As already indicated, the surgical resection of clival and petroclival meningiomas was associated with dismal results until the microneurosurgical era. The central location of the tumors medial to the cranial nerve foramina

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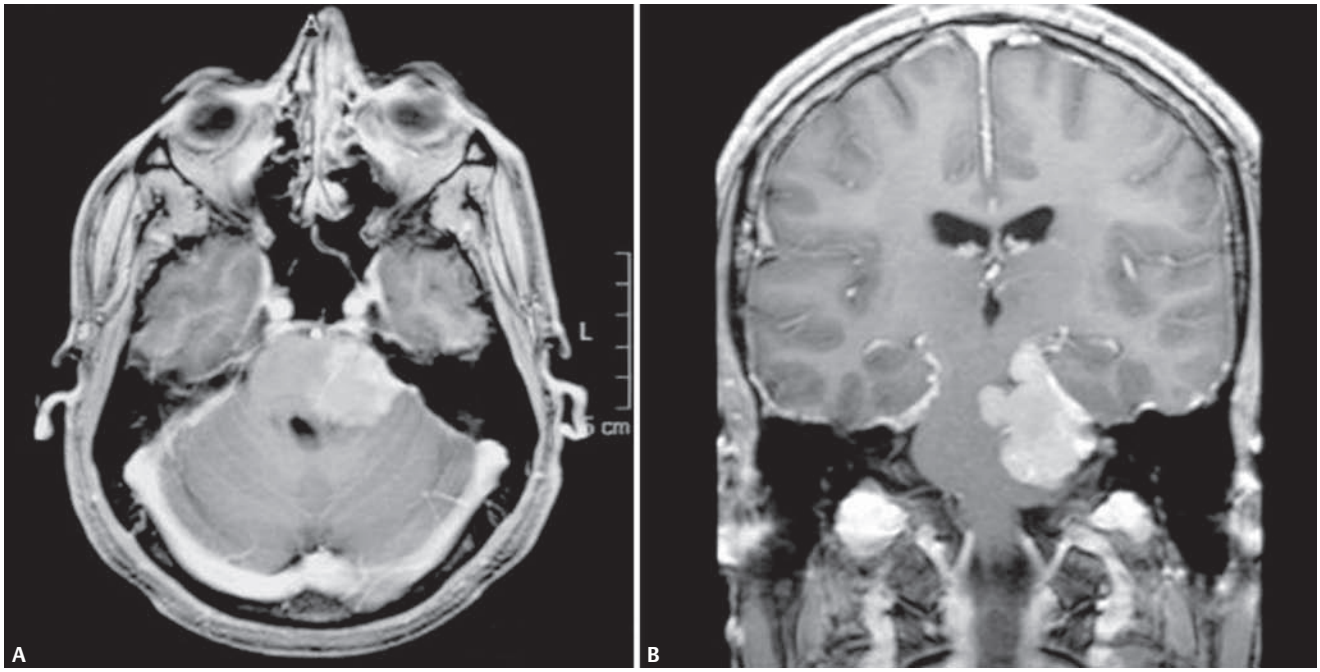


Fig. 29.1 **(A)** Axial and **(B)** coronal T1-weighted magnetic resonance imaging after gadolinium injection demonstrating a homogeneously enhancing petroclival and tentorial meningioma.



Fig. 29.2 Axial T1-weighted magnetic resonance imaging after gadolinium injection demonstrating a large petroclival meningioma with brain stem compression and displacement of the basilar artery as well as supratentorial extension into the middle fossa. Reproduced with permission from Liu JK, Couldwell WT. Petrosal approach for resection of petroclival meningiomas. In: Badie B, ed. Neurosurgical Operative Atlas 2E: Neuro-oncology. New York, NY: Thieme; 2007.[32]

and the intimate relationship to the brain stem and its vasculature explain the surgical difficulties encountered (**Fig. 29.3**).

The first surgical attempts at resection were performed through the pterional and suboccipital approaches. These approaches provide access to the tumor with a narrow central corridor, and dissection of the tumor in its periphery under direct visualization is limited. Skull base approaches provide a more direct route to the tumor and may extend the exposure with a better anterior view of the brain stem (**Fig. 29.4**).

Electrophysiological monitoring in the surgical resection of petroclival meningiomas includes somatosensory evoked potentials, electroencephalography, motor evoked potentials, facial nerve monitoring, and brain stem auditory evoked responses. In cases in which lower cranial nerve involvement is suspected, an electromyographic endotracheal tube can be used for cranial nerve (CN) X monitoring, and electrodes placed into the sternocleidomastoid muscle can provide monitoring for CN XI.

An anterior petrosectomy may be combined with a pterional or subtemporal approach (extended middle fossa approach) to provide better access to the tumor in its infratentorial compartment. An extradural approach should be used up to the outer layer (dura propria) of the lateral wall of the cavernous sinus, which is elevated extradurally to the gasserian ganglion and the inferior aspect of V3 to the foramen ovale. Bony removal is limited to the Kawase triangle, limited anteriorly by V3, laterally by the greater superficial petrosal nerve and internal carotid artery, posteriorly by the internal acoustic canal, and medially by the petrous edge. The dural opening

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Table 29.2 Radiological Characteristics of Petroclival Meningiomas at Time of Diagnosis

		Number with Extension of Petroclival Meningiomas beyond Structure at Time of Diagnosis							
Study	***N***	**Clivus Midline (%)**	**Tentorial Notch (%)**	**Cavernous Sinus (%)**	**Meckel Cave (%)**	**Internal Acoustic Meatus (%)**	**Bone Abnormal (%)**	**Vascular Encase-ment (%)**	**Stem Edema (%)**
Bricolo et al, 1992[8]	33	8 (21)	6 (18)	NA	NA	NA	16 (48)	11 (33)	NA
Cho et al, 2002	7	6 (86)	5 (71)	7 (100)	7 (100)	NA	4 (57)	7 (100)	NA
Goel 1999[25]	24	6 (25)	NA	7 (29)		16 (66)	NA	6 (17)	NA
Natarajan et al, 2007[13]	150	NA	7 (5)	75 (50)	5 (3)	NA	NA	72 (48)	NA
Roche et al, 2003[28]	32	NA	5 (16)	3 (9)	10 (31)	NA	NA	2 (6)	NA
Van Havenbergh et al, 2003[15]	21	NA	12 (57)	NA	NA	NA	NA	NA	NA
Zhu et al, 2007	7	3 (43)	5 (71)	6 (86)	7 (100)	NA	1 (14)	7 (100)	NA
Wei et al, 2006	25	NA	NA	7 (28)	NA	NA	NA	NA	NA
Little et al, 2001	139	NA	NA	42 (31)	NA	NA	NA	69 (50)	NA
Jung et al, 2000[16]	67	NA	NA	NA	NA	NA	NA	NA	12 (32)
Goel et al, 2004[9]	28	5 (18)	5 (18)	5 (18)	NA	NA	NA	9 (32)	NA
Carvalho et al, 2000[24]	70	NA	10 (15)	29 (42)	NA	NA	19 (27)	NA	23 (33)

Abbreviation: NA, not available.

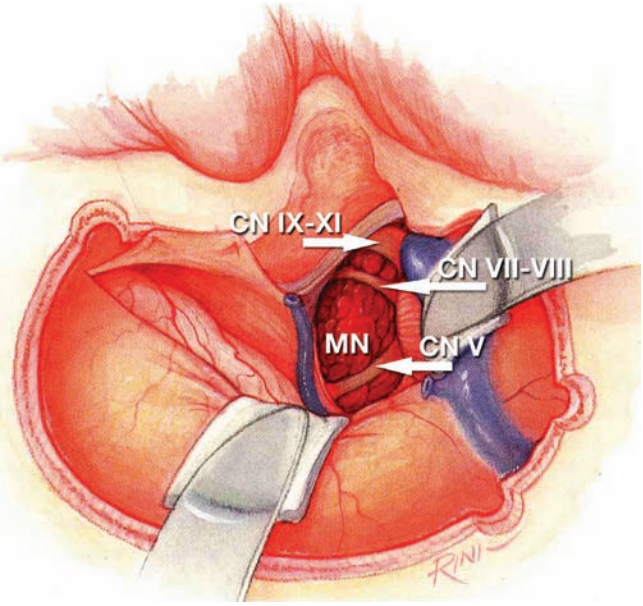


Fig. 29.3 An operative representation of the tumor location before resection through a posterior petrosal approach. CN, cranial nerve. Reproduced with permission from Liu JK, Couldwell WT. Petrosal approach for resection of petroclival meningiomas. In: Badie B, ed. Neurosurgical Operative Atlas 2E: Neuro-oncology. New York, NY: Thieme; 2007.[32]

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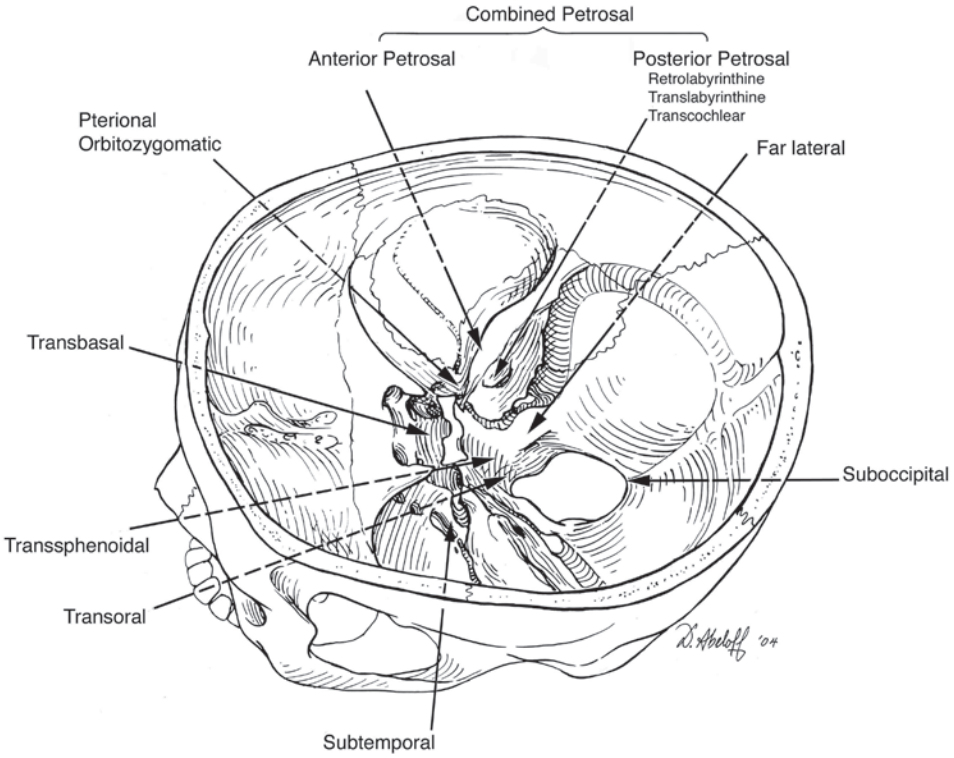


Fig. 29.4 Surgical approaches and trajectories available in the surgical treatment of clival and petroclival meningiomas. Reproduced with permission from Liu JK, Couldwell WT. © 2004.

should then be parallel to the base of the temporal lobe followed by ligation of the superior petrosal sinus anterior to the drainage of the vein of Labbé. The tentorium is divided, offering a combined supra- and infratentorial access. The infratentorial component of the tumor can be removed above the internal acoustic meatus, and the approach provides a good view of the anterior surface of the brain stem.[17] An anterior petrosectomy is useful in cases of tumors crossing the midline or when the central clival depression needs to be accessed.[17,18] An increased risk of injury to the trigeminal nerve is possible because of its central location in the surgical corridor. The potential morbidity of the approach consists of cerebrospinal fluid (CSF) leaks, hearing loss, decreased tearing, and facial nerve palsy, as well as injury to the temporal lobe secondary to the use of brain retraction or sacrifice of the superior petrosal sinus.

The posterior petrosal approaches expose the middle fossa as well as the posterior fossa in a presigmoid fashion and address the tumor laterally and inferior to the internal acoustic meatus (**Figs. 29.3, 29.5,** and **29.6**). Varying degrees of temporal bone can be removed (retrolabyrinthine, translabyrinthine, or transcochlear approaches) based on the patient's preoperative hearing status (**Fig. 29.4**). A retrolabyrinthine approach preserves hearing by leaving the otologic structures intact. A translabyrinthine approach involves drilling of the semicircular canals for a more anterior trajectory to the tumor, with sacrifice of hearing. A transcochlear approach involves drilling of all otologic structures as well as transposing the facial nerve posteriorly to maximize the surgical view. The translabyrinthine and transcochlear approaches are reserved for patients who do not have serviceable hearing (speech discrimination score inferior to 50% or hearing loss greater than 50 dB on formal audiogram testing). The superior petrosal sinus is sacrificed in all posterior petrosal approaches (**Fig. 29.6**), but care should be taken to preserve the drainage of the vein of Labbé. The tentorium should only be divided anterior to the drainage of the vein of Labbé, which may drain directly into the sigmoid sinus or the superior petrosal sinus. Potential complications of the approach include CSF leaks, hearing loss, or facial nerve paresis. Posterior petrosal approaches offer a more direct trajectory to the tumor with minimal retraction on the temporal lobe and cerebellar hemispheres. Earlier interruption of the tumor's blood supply is also feasible due to a more anterior surgical trajectory.

A combined petrosal approach offers the supratentorial access with an anterior petrosectomy and the infratentorial retrolabyrinthine presigmoid access. A complete petrosectomy combines the anterior and posterior petrosectomy with a labyrinthectomy as well as drilling of the cochlea and ossicles of the middle ear.

The choice of approach depends on the extent of the tumor based on preoperative imaging as well as the status of the patient's cranial nerves, especially hearing. The use of extensive skull base approaches increases the morbidity of the approach in petroclival meningioma surgery. Specific morbidity due to the posterior petrosal approaches consists of a risk of CSF leakage, hearing loss, and facial nerve palsy. CSF leaks are more prominent in the posterior petrosal approaches than in anterior ap-

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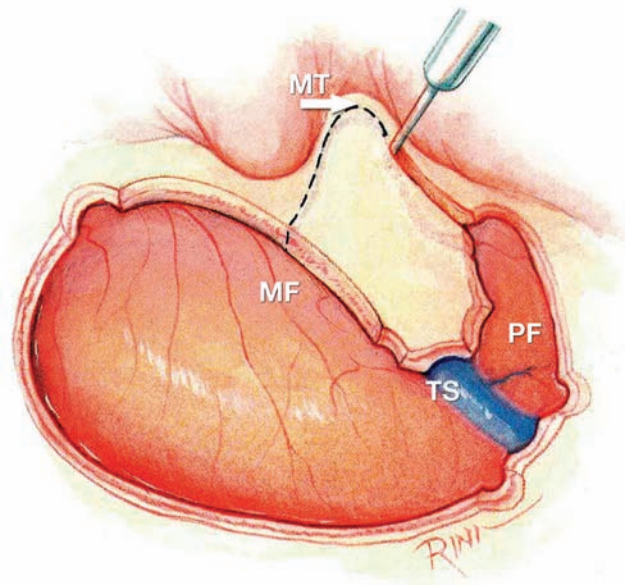


Fig. 29.5 Mastoidectomy with preservation of the outer cortex for later reconstruction after a supra- and infratentorial craniotomy. MT, mastoid tip; TS, transverse sinus; MF, middle fossa; PF, posterior fossa. Reproduced with permission from Liu JK, Couldwell WT. Petrosal approach for resection of petroclival meningiomas. In: Badie B, ed. Neurosurgical Operative Atlas 2E: Neuro-oncology. New York, NY: Thieme; 2007.[32]

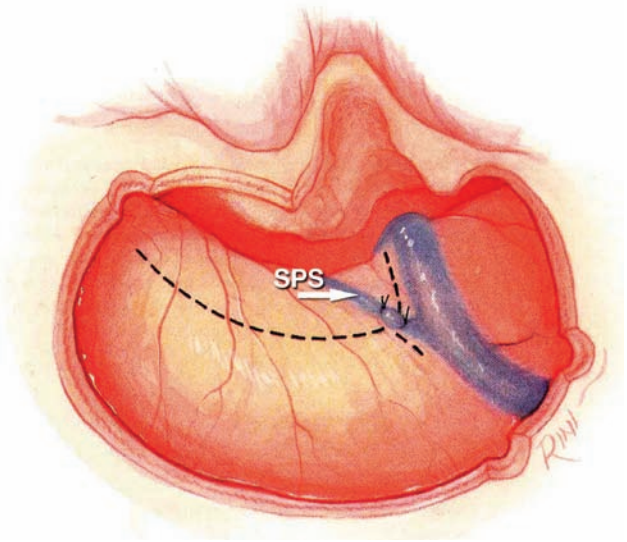


Fig. 29.6 Ligation of the superior petrosal sinus and the limits of dural opening. SPS, superior petrosal sinus. Reproduced with permission from Liu JK, Couldwell WT. Petrosal approach for resection of petroclival meningiomas. In: Badie B, ed. Neurosurgical Operative Atlas 2E: Neuro-oncology. New York, NY: Thieme; 2007.[32]

proaches but can be treated with temporary drainage in most cases.[17] At the time of closure, the dura should be closed, and a fascia lata graft may be necessary. Packing the defect created by the mastoidectomy with fat is often necessary, and care should be taken not to create mass effect on the dura. Hearing preservation has been reported in up to 92% of patients with petroclival meningiomas treated with a posterior retrolabyrinthine petrosal approach.[17,19] Partial resection of the posterior and superior semicircular canals may still preserve hearing[20–22] and improve midline access,[22] obviating the need to perform a full translabyrinthine approach. A transcochlear approach improves midline exposure at a higher risk of permanent facial palsy secondary to ischemia of the nerve if the nerve is translocated.[18,23] Because of the high incidence of facial nerve palsies in these approaches and the opportunity to access the midline structures through an anterior petrosectomy, the usefulness of the translabyrinthine and transcochlear approach in the context of petroclival meningiomas has been questioned.[17,22]

Venous anatomy may alter the effectiveness of a posterior petrosal approach. A high jugular bulb may limit the size of the presigmoid corridor, severely limiting the benefit of the approach.[17] Variations in the draining of the vein of Labbé also alter the posterior extent of the cut through the tentorium. Preoperative evaluation of the venous structures with the help of magnetic resonance venography (MRV) and computed tomographic angiography (CTA) can more accurately predict the usefulness of posterior petrosal approaches in specific cases.[17]

Regardless of the approach used, tumor removal is performed in a piecemeal fashion. Inspection of the inferior part of the tumor should permit visualization of the vertebral artery, posterior inferior cerebellar artery, and cranial nerves IX through XI (**Fig. 29.7**). The fourth and fifth cranial nerves are usually displaced toward the superior end of the tumor (**Fig. 29.8**). The sixth cranial nerve is usually displaced medially to the tumor and is encountered in the later stages of the dissection. The seventh and eighth cranial nerve complex may be displaced laterally to the tumor or can be embedded in the tumor (**Fig. 29.9**). Careful piecemeal resection of the tumor can be accomplished with microscissors, and cranial nerves and vascular branches to the brain stem should be preserved. Coagulation should be avoided in the close vicinity of the brain stem, and copious irrigation may be preferable to accomplish hemostasis.[8] A complete resection of the tumor against the brain stem may be dangerous in cases where the tumor is adherent or has parasitized the blood supply to the brain stem.

Several authors, including the senior author, still advocate the use of a suboccipital retrosigmoid craniotomy for petroclival meningiomas without supratentorial extent because they believe there to be typically less morbidity due to the approach.[8,9] A difference in postoperative morbidity has not been demonstrated in published series with the use of any of these approaches.

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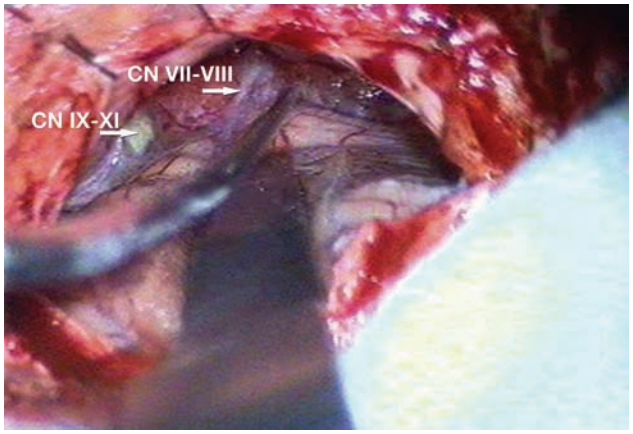


Fig. 29.7 Intraoperative photomicrograph of a left retrosigmoid approach for a petroclival meningioma. CN, cranial nerve.

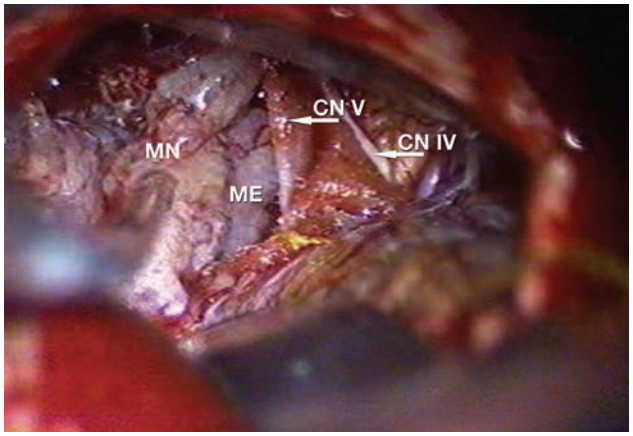


Fig. 29.8 Intraoperative high-magnification photomicrograph of resection of a petroclival meningioma through a left retrosigmoid approach. CN, cranial nerve; ME, mesencephalon; MN, meningioma.

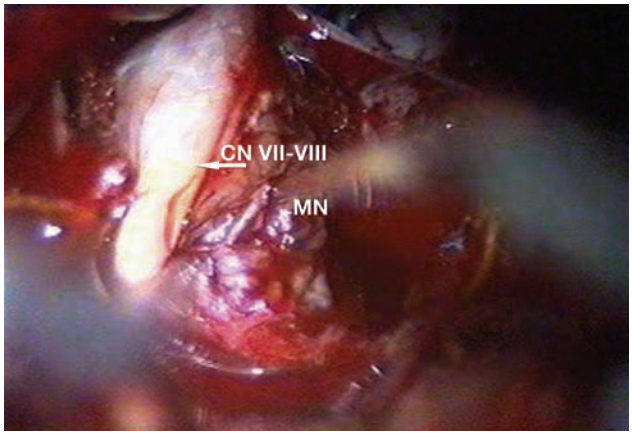


Fig. 29.9 Intraoperative high-magnification photomicrograph of resection of a petroclival meningioma through a left retrosigmoid approach with dissection of the meningioma off the cranial nerve VII and VIII complex. CN, cranial nerve; MN, meningioma.

Results

Surgical results in the microsurgical era have continued to improve since Yasargil et al[5] reported their results. Gross total resection rates of petroclival meningiomas vary greatly and have been reported to range from 20 to 79% of cases (**Table 29.3**). The ability to achieve a gross total resection is influenced by the tumor's involvement of the cavernous sinus as well as the relationship of the tumor with vascular structures and cranial nerves at their cranial foramina.[8] The most important goal at surgery remains brain stem decompression.[18] Long-term results, at least 3 months after surgery, show a most favorable response in preoperative cerebellar dysfunction (**Table 29.3**). Somatosensory symptoms usually improve long term, but the evolution of cranial neuropathies is less predictable because of the small numbers of patients in each series (**Table 29.3**).

The reported functional outcomes are also difficult to compare because significant neurological morbidity in the form of cranial nerve palsies is not always reflected in a change of the Karnofsky score. A subset of patients clearly more at risk of significant postoperative morbidity are those with prior resection, preoperative findings of cranial nerve palsies, and the presence of fibrous or adherent tumors.[12] Some centers have opted for a less aggressive surgical intervention, with decreased postoperative morbidity.[12] The presence of peritumoral edema and extension of the tumor to the lower cranial nerves has also been noted to be an increased risk factor for surgical morbidity.[24]

The surgical results of different series are difficult to compare because of the heterogeneity of these tumors in their invasion of adjacent structures. The benefits associated with the use of specific surgical approaches have not clearly been demonstrated because of the rarity of these tumors and the fact that a variety of approaches are used in most reported series. The surgeon's selection of an approach should optimize tumor exposure with adequate visualization of the dissection planes while limiting the need for brain retraction and the possibility of injury to cranial nerves and vascular structures, with the emphasis on decompressing the brain stem.

Morbidity and Mortality

Morbidity rates have improved in the treatment of petroclival meningiomas but remain significant. The most common complication after treatment of petroclival meningiomas is the development of cranial nerve palsies. New cranial nerve palsies or worsening of preexisting palsies has been reported to occur in up to 76% of patients (**Table 29.4**).[3] Trochlear nerve palsies are the most common, followed by palsies in CN III through VIII (**Table 29.4**).[24] The cranial nerve palsies may be transient in nature, and long-term outcomes for patients with cranial nerve palsies appear to return to at least preoperative status,[8] with improvement seen in some series.[13] The rate of

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Table 29.3 Surgical Results of Large Petroclival Meningioma Series since 1990

Study	*N*	Number with Gross Total Resection (%)	Preoperative and Long-Term (> 3 months) Postoperative Prevalence (and %) of Neurological Dysfunction											Recurrence/ Progression Total (%)	Mean length of follow-up
			CN II	CN III	CN IV	CN V	CN VI	CN VII	CN VIII	CN IX–XI	CN XII	Cerebellar	Somato-sensory		
Natarajan et al, 2007[13]	150	48 (32)	3 (2)/ 1 (1)	36 (24)/ 6 (4)	21 (14)/ 7 (5)	64 (43)/ 7 (5)	43 (29)/ 9 (6)	16 (11)/ 1 (1)	46 (31)/ 1 (1)	10 (7)/ 2 (1)	4 (3)/ 1 (1)	27 (18)/ 9 (6)	16 (11)/ 11 (7)	7 (5)	101 months
Abdel Aziz et al, 2000[18]	35	13 (37)	2 (6)/ 1 (3)	6 (17)/ 5 (14)	3 (9)/ 4 (11)	19 (54)/ 20 (57)	9 (26)/ 6 (17)	4 (11)/ 6 (17)	12 (34)/ 13 (37)	9 (26)/ 7 (20)	2 (6)/ 1 (3)	14 (40)/ 4 (11)	8 (23)/ 5 (14)	1 (3)	6 months–8 years (4 years)
Couldwell et al, 1996[11]	109	75 (69)												14 (13)	6.1 years (2–14 years)
Bricolo et al, 1992[8]	33	26 (79)												7 (26)	4.3 years
Goel 1999[25]	24	16 (67)												0	14 months (5–40 months)
Goel et al, 2004[9]	28	21 (75)												1 (3.5)	48 months
Jung et al, 2000[16]	67	26 (39)												16 (42)	47 months (6–141 months)
Little et al, 2005[12]	137	55 (40)												15 (18)	8.3 months (1–88 months)
Park et al, 2005	49	10 (20)												11 (22)	86 months (48–210 months)
Kawase et al, 1994	42	32 (76)												3 (7)	54 months

Abbreviation: CN, cranial nerve.

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Table 29.4 Mortality and Morbidity of Surgical Series of Petroclival Meningiomas since 1990

		Number of Patients with Stated Morbidity (%)																	
Study	***N***	**Cerebrospinal Fluid Leak**	**Stroke**	**CN II**	**CN III**	**CN IV**	**CN V**	**CN VI**	**CN VII**	**CN VIII**	**CN IX–XI**	**CN XII**	**HCP**	**New CN Palsy**	**Cerebellar**	**Somatosensory**	**Tracheostomy**	**PEG**	**Number of Deaths (%)**
Natarajan et al, 2007[13]	150	3 (2)	6 (4)	1 (1)	5 (3)	6 (4)	6 (4)	2 (1)	2 (1)	5 (3)	4 (3)	0	3 (2)	31 (20)	5 (3)	9 (6)	NA	NA	0
Couldwell et al, 1996	35	0	16 (46)	0	6 (17)	7 (20)	6 (17)	5 (14)	5 (14)	5 (14)	2 (6)	0	NA	36 (33)	7 (20)	9 (26)	NA	NA	4 (3.7)
Bricolo et al, 1992	33	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	25 (76)	NA	3 (9)	6 (18)	5 (15)	3 (1)
Goel 1999[25]	24	1 (4)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	17 (71)	NA	NA	NA	NA	0
Goel et al, 2004[9]	28	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	12 (43)	NA	NA	NA	NA	2 (7)
Little et al, 2005[12]	137	10 (7)	NA	0	10 (7)	9 (7)	15 (11)	12 (9)	8 (6)	3 (2)	11 (8)	1 (1)	2 (1)	31 (23)	6 (4)	10 (7)	4 (3)	NA	1 (1)
Park et al, 2006[26]	49	NA	NA	0	8 (16)		3 (6)	*	13 (26)	6 (12)	4 (8)	0	NA	14 (29)	NA	NA	NA	NA	1 (2)
Abdel Aziz et al, 2000[18]	35	1 (3)	NA	0	9 (26)	5 (14)	7 (20)	5 (14)	6 (17)	1 (3)	5 (14)	0	NA	11 (31)	0	4 (11)	NA	NA	0

Abbreviations: HCP, hydrocephalus; CN, cranial nerve; PEG, percutaneous endoscopic gastrostomy; NA, not available.
*Included in CN III–CN IV combined value.

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morbidity to cranial nerves is also related to the aggressiveness at surgery in view of preoperative imaging characteristics (e.g., presence of peritumoral edema,[24] prior surgery,[12] preoperative cranial nerve palsies[12]) or intraoperative findings (e.g., fibrous tumor[12]). Injury to lower cranial nerves is less commonly observed (**Table 29.4**) but results in a significantly decreased functional state, which may require the use of a tracheostomy or gastrostomy.[8,12] Injury to the brain stem at the time of dissection of the tumor off its pial surface or vascular supply may have devastating consequences resulting in serious morbidity or death. A fibrous and adherent tumor,[12] whose characteristics may be suspected preoperatively based on the presence of brain stem edema,[24] should prompt the surgeon to limit aggressiveness.[8,9,11–13]

Perioperative mortality rates vary from 0 to 7% in recently published series (**Table 29.4**).[8,9,11–13,18,25,26] Mortality is most commonly the consequence of a poor neurological outcome secondary to a brain stem stroke.

Recurrence

Recurrence and progression rates after the surgical resection of petroclival meningiomas have been reported to vary from 0 to 42% (**Table 29.3**).[8,9,11–13,16,18,25–27] Some studies vary in their definition of recurrence or progression based on clinical or radiological criteria. Tumor recurrence seems more likely with a less extensive surgical resection,[12,16,19] malignant histopathological results,[11,12,19] and cavernous sinus involvement.[11,12,19] Length of follow-up in most studies remains short, and the incidence with which radiosurgery is used postoperatively also varies among studies. The best long-term studies that have used surgical resection followed by radiosurgery for any residual tumor with evidence of progression demonstrate recurrence or progression rates of 4.5 to 22%, with follow-up intervals from 6 to 8 years.[11,13,26]

Role of Radiosurgery

Radiosurgery has had a prominent role in controlling progression of residual tumor. Failure of radiosurgery after microsurgery in controlling residual disease has been reported to be as low as 0% at 4-year follow-up[28] and as high as 13% at 3 years.[29] This clearly compares favorably with progression rates of residual tumor of 42% at 4 years.[16] The efficacy of radiosurgery has prompted surgeons to be less aggressive in resecting tumor extension into the cavernous sinus or to accept a subtotal resection in cases of high risk of postoperative morbidity.[8,11,13,25]

Radiosurgery can be accomplished with low morbidity for the treatment of petroclival meningiomas. Transient new cranial nerve palsies have been reported in up to 6% of cases,[28] and permanent new cranial nerve palsies in up to 5% of cases.[29] No mortality has been reported related to the use of radiosurgery (**Table 29.6**). Transient worsening of neurological function due to brain stem injury has been observed and can be permanent in up to 6% of cases.[28] In cases in which the volume of residual tumor is too large for radiosurgery, conventionally fractionated radiation therapy using three-dimensional conformal (3DCRT) or intensity-modulated techniques (IMRT) provides equally good long-term tumor control.[30,31]

The role of radiosurgery as a primary treatment modality has also been investigated. Recent series have all reported a 100% control rate of petroclival meningiomas by MRI criteria, with mean follow-up periods of 3 to 4 years.[26,28,29] Clinical outcomes have also been favorable in these series, with improvement of neurological function in 96 to 100% of cases[26,29] and improvement in cranial nerve function in up to 50% of cases (**Table 29.5**).[28] These series are difficult to compare with microsurgical series because they are still limited in number and have inadequate follow-up for these benign tumors. Furthermore, a higher proportion of cases in which the patient has undergone radiosurgery as a primary treatment have been asymptomatic in these studies compared with surgical series.[28]

Treatment Algorithm for Petroclival Meningiomas

Petroclival meningiomas are difficult lesions to treat because of their intimate relationship to the brain stem, cranial nerves, and vascular structures. Results of microsurgical resection have greatly improved over the last 2 decades, and the efficacy of radiosurgery has further augmented treatment options. Radiosurgery can be used as the primary treatment method in elderly patients, asymptomatic patients, or patients without significant brain stem compression. Radiosurgery is most effective at treating tumors whose diameter is less than 3 cm. Once tumor progression has been documented, delay of treatment is not warranted because it may precede a neurological decline.

Microsurgical resection should be contemplated in young patients with rapidly growing tumors, tumors with brain stem compression, or cases where the diagnosis of a benign lesion might be in doubt. The primary goal of microsurgical resection should be to address brain stem compression, and gross total resection should only be attempted if it can be done safely. Resection of tumor in the cavernous sinus or parasellar region is not warranted at the time of first resection, and radiosurgery or radiotherapy should be used as adjuvant therapy in cases of residual tumor progression (**Table 29.7**).

Conclusion

Petroclival meningiomas are defined by their dural attachment posterior to the Meckel cave and medial to the internal auditory meatus at the petroclival junction. Meningiomas with a more medial attachment that involve the upper two thirds of the clivus are classified as clival meningiomas. These are infrequent tumors in the posterior fossa, but their treatment is challenging. Their pro-

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Table 29.5 Results of Radiosurgery for the Treatment of Petroclival Meningioma

Study	*N*	Technique	Mean Age (years)	Mean Volume Treated (mL)	Mean Length of Clinical Follow-Up (mo) (range)	Number of Patients Clinically Stable/ Improved (%)	Number of Patients with CN Improve-ment	Mean Length of Radiological Follow-Up (mo)	Number of Patients Radiologi-cally Stable/ Improved (%)
Effects of Radiosurgery as Primary Treatment									
Subach et al, 1998[29]	23	Gamma knife	66.4	13.3	38.8	22 (96)	NA	34	23 (100)
Park et al, 2006[26]	12	Gamma knife	44 (range 18–64)	5.2	52 (range 48–71)	12 (100)	NA	Same	12 (100)
Effects of Radiosurgery after Microsurgery									
Subach et al, 1998[29]	39	Gamma knife	49.8	14	43.5	32 (82)	NA	36	34 (87)
Radiosurgery as Primary Treatment or Postmicrosurgery									
Roche et al, 2003[28]	32	Gamma knife	53 (range 34–77)	2.2	48 (range 24–118)	NA	11/22 (50)	48 (24–118)	24 (100)

Abbreviations: CN, cranial nerve; NA, not available.

Table 29.6 Morbidity and Mortality of Radiosurgery

Study	*N*	Number (%) of Cases of Hydrocephalus	Number (%) of Cases of Transient CN Palsy	Number (%) of Cases of Permanent CN Palsy	Number (%) of Cases of Procedural Mortality	Number (%) of Cases of Transient Brain Stem Dysfunction	Number (%) of Cases of Permanent Brain Stem Dysfunction
Subach et al, 1998[29]	62	1 (16)	2 (3)	3 (5)	0	0	0
Roche et al, 2003[28]	32	NA	2 (6)	0	0	1 (3)	2 (6)
Park et al, 2006[26]	12	NA	NA	NA	NA	NA	NA

Abbreviations: CN, cranial nerve; NA, not available.

Table 29.7 Treatment Algorithm for Petroclival Meningiomas

	Tumor Size	
Patient Age	**Small (< 3 cm)**	**Large (> 3 cm)**
Young	MS GTR	No radiographic evidence of cavernous sinus involvement: MS GTR Radiographic cavernous sinus involvement: - CN: STR and observation of residual or radiosurgery of residual + CN: MS GTR
Old	Symptomatic MS GTR vs radiosurgery Asymptomatic Observation vs radiosurgery	STR of symptomatic mass followed by: Observation if MRI is stable Radiosurgery if radiological progression

Abbreviations: CN, cranial nerve palsy; GTR, gross total resection; MRI, magnetic resonance imaging; MS, microsurgery; STR, subtotal resection.

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gression is insidious until patients present with cerebellar dysfunction, cranial nerve palsies, or symptoms of brain stem compression. Most tumors are diagnosed once their diameter exceeds 2 cm. Observation, radiosurgery, and microsurgery can be offered based on the patient's age, tumor size, and type of symptoms. Gross total resection is not feasible in many cases because of invasion of the cavernous sinus or the fibrous or adherent nature of the tumor. Surgical results have continued to improve, and any of several surgical approaches can be used. Radiosurgery and radiotherapy continue to have an increasing role in controlling tumor recurrence or progression and may be used in conjunction with microsurgery.

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Chapter 30

Meningiomas of the Temporal Bone

Paul W. Gidley

Introduction

Meningiomas of the temporal bone are rare. Chang et al found case reports of only 77 patients from 1886 to 1998.[1] However, the temporal bone is an important anatomical pathway for approaching meningiomas. This chapter concentrates on tumors that arise primarily within the confines of the temporal bone, including the internal auditory canal (IAC) and jugular foramen. Discussions of tumor within the cerebellopontine angle and petroclival region are found in Chapters 28 and 29.

Anatomy

The disarticulated temporal bone is a roughly pyramidal shape. In the lateral direction, its broad base is formed by the squamosal, tympanic, and mastoid portions of the temporal bone. Medially, the bone tapers into the petrous apex, including the region of the IAC. The superior surface of the temporal bone is the middle cranial fossa, containing the greater superficial petrosal nerve and arcuate eminence. This bony plate is the tegmen of the mastoid and middle ear cavities. The posterior surface is the cerebellar plate, and its most important structure is the sigmoid sinus. Anteriorly, the temporal bone articulates with the condyle of the mandible and is in continuity with the sphenoid bone. Inferiorly, the temporal bone gives off the jugular vein, the facial nerve, and the lower cranial nerves (IX, X, and XI) and receives the internal carotid artery. A thin plate of bone, the carotid crest, separates the carotid artery from the jugular bulb. Anatomical studies have shown that the anterior, or pars nervosa, portion of the foramen contains the inferior petrosal sinus and the glossopharyngeal nerve.[2–4] An intimate understanding of these relations is required to comprehend the surgical approaches in the temporal bone.

Sites of Involvement

Meningiomas have been described involving all portions of the temporal bone. The middle ear, eustachian tube, IAC, and jugular foramen are the four most common locations of meningiomas in the temporal bone.[5] Thompson et al described a series of 36 tumors from a pathology database.[15] Their series included a majority of middle ear only tumors (25), followed by ear canal only (four), temporal bone only (two), or a combination of sites (five). Their series did not include jugular foramen tumors. Rarely, meningiomas have been described occurring in the geniculate ganglion[6,7] and along the intratemporal facial nerve.[8]

Nager has divided temporal bone meningiomas into tumors that are an extension of an intracranial tumor (type 1) and those that are not (type 2),[9] but this terminology can be confusing. Therefore, these tumors have also been characterized as primary, meaning site of origin within the temporal bone, or secondary, meaning site of origin outside the temporal bone, but with extension into it. The secondary tumors are much more common.

Primary temporal bone tumors have their origin from a nest of ectopic, extracranial arachnoid villi cells.[1] Secondary tumors can extend into the middle ear through one of four pathways: the tegmen tympani, the posterior fossa plate, the IAC, or the jugular foramen.

Meningiomas of the jugular foramen can occur either as a primary tumor or as secondary extension from an intracranial location (cerebellopontine angle or petroclivus).[10] Primary jugular foramen meningiomas can extend superiorly into the middle and inner ear, medially into the cerebellopontine angle, or inferiorly into the neck.

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Incidence and Frequency

Meningiomas are the second largest group of brain tumors after gliomas, accounting for 15 to 18% of all intracranial tumors.[11] Only ~2% of meningiomas are found within the temporal bone.[10,12,13] In a series of posterior fossa meningiomas, Roberti et al reported that of 161 consecutive cases in 6 years only seven (4.3%) involved the jugular foramen.[13] Several case series, usually no more than 40 cases, have been reported in the literature since 1990 (**Table 30.1**). Of tumors limited to the temporal bone, the jugular foramen is the most common site. IAC meningiomas are exceedingly rare, with only 14 tumors reported in the literature.[14]

The majority of patients are women. The mean age at diagnosis is roughly 45 to 50 years.[15] The exception to this rule is meningiomas at the geniculate ganglion, which seem to present in younger patients and perhaps come to earlier diagnosis because of progressive facial paralysis.

Pathological Features

Meningiomas arise from arachnoid villi, which can be found in the jugular foramen, middle fossa, or IAC.[2] Arachnoid villi are also found throughout the temporal bone.[16] Nager has described arachnoid extensions along the nerve sheaths of the greater superficial petrosal nerve and cranial nerves VII through XII.[9] Arachnoid villi are numerous around dural sinuses, explaining the occurrence around the jugular bulb.

Psammoma bodies are spherical, laminar, calcified structures that are frequently associated with meningiomas.[17] However, psammoma bodies are also found in normal temporal bones, without an associated meningioma. The occurrence of psammoma bodies within the IAC, along the facial nerve, geniculate ganglion, and posterior ampullary nerve may be a normal consequence of aging.[17]

Pathologically, temporal bone meningiomas are frequently of the syncytial (meningothelial) variety.[5,10,15] A few secretory meningiomas have been found in the middle ear and produce symptoms similar to chronic otitis media.[18–20] Immunohistochemically, most temporal bone meningiomas are positive for vimentin and epithelial membrane antigen (EMA).[15] A minority of these tumors are positive for S-100 or cytokeratin or both.[5]

Although these tumors are classified as benign, the characteristics of local recurrence, invasion of bone and nerves, and radioresistance make treatment treacherous.

Mitotic activity, brain invasion, micronecrosis, nucleolar pleomorphism, nucleolar prominence, and increased cellularity are all linked to a diagnosis of atypical or malignant meningioma.[21]

Symptoms and Signs

The symptoms and signs of temporal bone meningiomas are nonspecific. The symptoms are common for any otic process: hearing loss, middle ear effusion or mass, external ear swelling or mass, otalgia, or otorrhea (**Fig. 30.1A**).[15,22] In a relatively large series of 56 patients, Arriaga et al reported hearing loss in 85%, dizziness in 61%, tinnitus in 75%, headaches in 32%, and cerebellar findings in 23%.[11] A middle ear mass was found in four of 13 (30.7%) patients with jugular foramen meningiomas.[10] Primary temporal bone meningiomas have also been described as presenting with signs of chronic otitis media with perforation[19,23] or serous otitis media[24] (**Fig. 30.2A**). Because these symptoms are largely nonspecific to a pathological diagnosis, biopsy might be necessary for confirmation. External ear canal masses are readily biopsied in the outpatient clinic, most without needing local anesthetic.

Facial weakness or paralysis and lower cranial nerve dysfunction are seen in patients with temporal bone meningiomas. Facial nerve dysfunction can be subtle and might be manifest only by a delayed blink or twitching in an isolated branch. Careful, directed inspection of facial function is required to detect this mild dysfunction (**Fig. 30.3**).

Tumors that involve the jugular bulb have the potential to involve the lower cranial nerves. The hallmarks of dysfunction in these nerves are a weak, breathy, or wet-sounding voice and dysphagia or aspiration. Careful evaluation of these nerves with fiberoptic nasopharyngoscopy is required. When vocal fold dysfunction or dysphagia is found, the patient should be referred to a speech pathologist for evaluation utilizing laryngeal videostroboscopy and modified barium swallow.

Auditory and Vestibular Evaluation

Patients with complaints of hearing loss are always evaluated by routine audiometry, including pure-tone testing, speech audiometry, and middle ear immittance (tympanometry). This test clearly defines the level and type of hearing loss: conductive (**Fig. 30.2B**), sensorineural (**Fig. 30.4A**) or mixed (**Fig. 30.5A**). This test is an essential first step in evaluating temporal bone meningiomas. The nature and degree of hearing loss at presentation have important implications for the approach taken for disease removal. The level of speech understanding (also called speech discrimination score [SDS]) and the pure tone threshold identify patients with useful or serviceable hearing versus those with measurable or nonserviceable hearing. A consistent rule for serviceable hearing is an SDS greater than 50% and a pure tone threshold less than 50 dB. Conductive hearing loss occurs with tumors that have a middle ear component. Tumors that erode into the middle and inner ear produce a mixed hearing loss. Hearing loss may be either sudden or progressive.[16,25] Pensak et al documented hearing loss in 6/15 patients (40%).[26] The implication is that perhaps 60% of patients will have normal hearing (**Fig. 30.6A**).

Auditory brain stem response (ABR) is an important test to measure the function of the auditory nerve and brain stem pathways. This test has important implications for tumors that are in the cerebellopontine angle, but it might not provide clinically useful information for tumors that are within the temporal bone. Arriaga et al reported normal ABR in 37% of their patients.[11] Pensak et al reported abnormal ABR in 5/14 patients (35.7%).[26]

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Table 30.1 Compilation of Contemporary Temporal Bone Meningioma Reports

Lead Author, Year	*N*	Locations	Age Mean, Range (yrs)	Postop Facial Function	Postop Hearing Loss	New postop Lower Cranial Nerve (IX,X,XI) Deficits	Treatment	Recurrence Rate/Outcomes
Sanna, 2007	13	Jugular foramen	41 years, (21–60 years)	HB I –II = 46.1% HB III = 53.9%		61.5%	Surgery—13 (GTR 84.6%; STR 15.4%)	No recurrence (mean 46.7 months)
Ramina, 2006[49]	10	Jugular foramen	38 years (12 to 62 years)	NS	NS	50%	Surgery—10/10 (GTR 5/10; STR 5/10) XRT—4/10	4/10 alive (mean 6.5 years) 6/10 DOD (mean 35 months)*
Gilbert, 2004[33]	6	Jugular foramen	52 years (22–65 years)	HB I –II = 50% HB III–IV = 33%	83.3%	60%	Surgery only	1/6 recurred within 2.5 years 2 pts required VP shunts
Oghalai, 2004[51]	10	Jugular foramen	42.4 years (7–69 years)	HB I–II = 92%	59%	57%	Surgery	NS
Thompson, 2003[15]	36	ME = 25 EAC = 4 TB = 2 Mixed = 5	49.6 years (10–80 years)	NS	NS	NS	Surgery only	10/30—Recurrent (5–24 months) 25/30–Alive (mean 19.0 years) 5/30–Died other causes
Arnautović, 2002[52]	8	Jugular foramen					Surgery	
Luetje, 1997[7]	6	Geniculate ganglion	19.5 years (5–38 years)	HB III–V	NS	NS	Surgery	NS
Arriaga, 1992[11]	56	Petroclival, IAC, jugular foramen, etc.	NS	HB I–II = 64.2% HB III–VI = 9.5%	NS	10–67%	Surgery only	NS
Molony, 1992[2]	8	Jugular foramen	40 years (19–52)		7/8 5 mixed 2 SNHL	3/8	Surgery only	2/8 in 5 years

Abbreviations: NS, not stated; DOD, died/dead of disease; EAC, external auditory canal; GTR, gross total resection; HB, House-Brackmann Score; IAC, internal auditory canal; ME, middle ear; SNHL, sensorineural hearing loss; STR, subtotal resection; TB, temporal bone; VP, ventricular-peritoneal; XRT, radiation therapy.
* Malignant histology.

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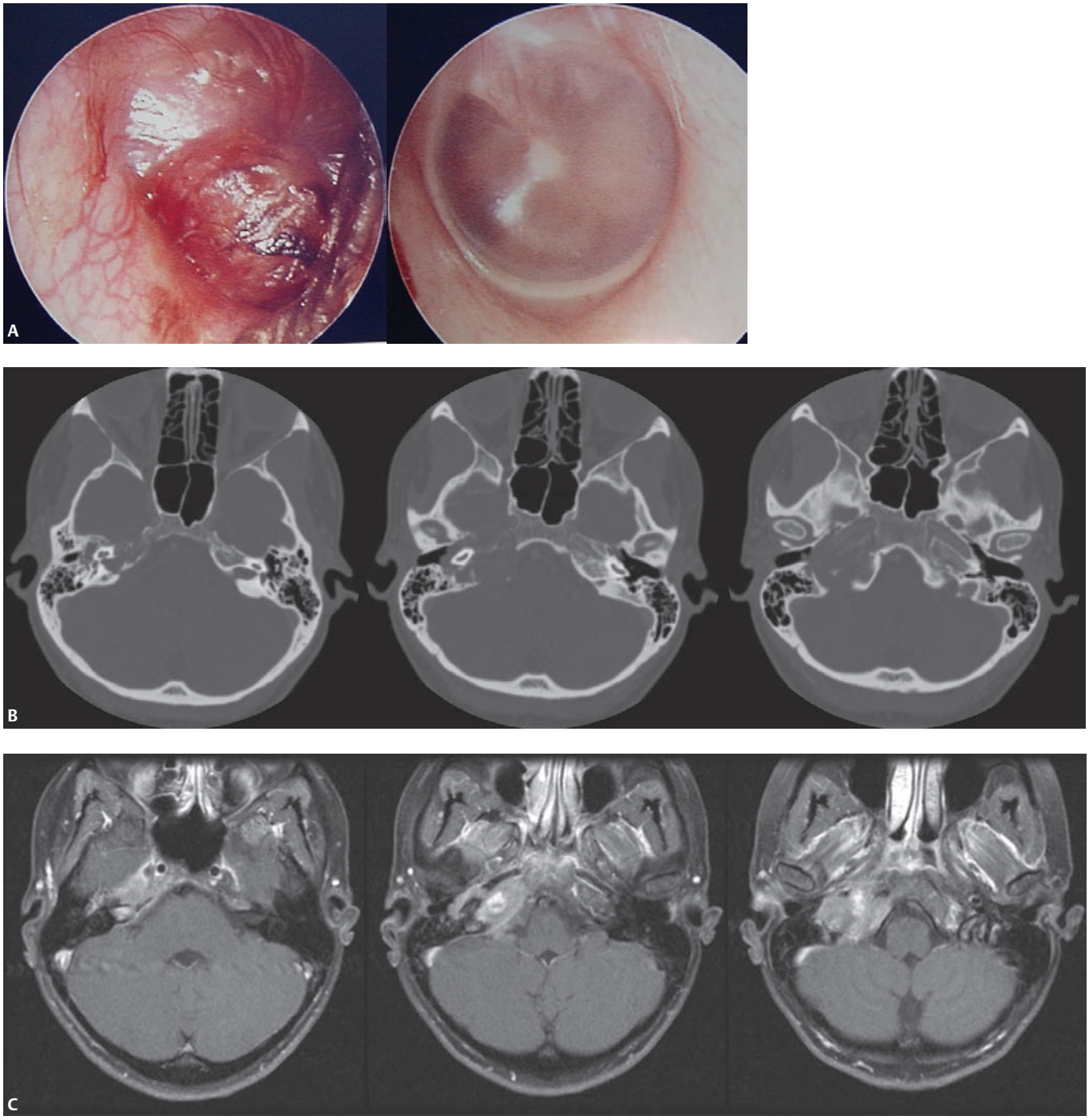
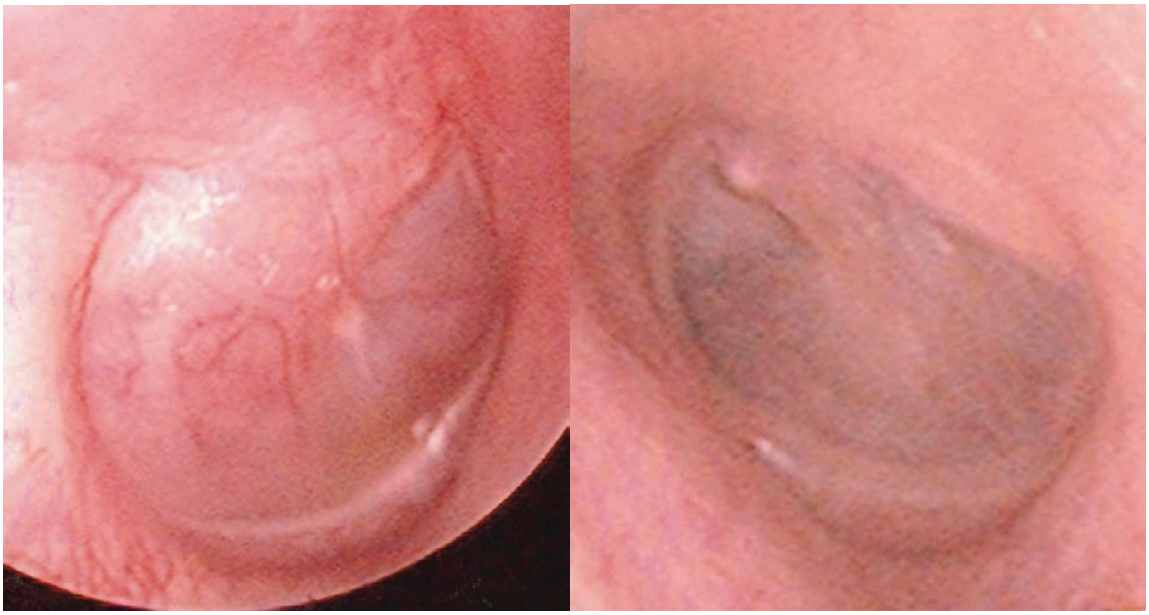


Fig. 30.1 Temporal bone meningioma presenting as severe right ear pain and facial paralysis. **(A)** Otoendoscopy of tumor breaking through the right eardrum and normal left eardrum. **(B)** Axial computed tomographic (CT) images showing tumor permeating and destroying the right temporal bone. The first frame shows tumor eroding into the internal auditory canal. The second frame shows tumor engulfing the basal turn of the cochlea. The third frame shows tumor surrounding the vertical segment of the carotid artery. **(C)** Postcontrast T1 magnetic resonance imaging at levels similar to the CT images.

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A



B

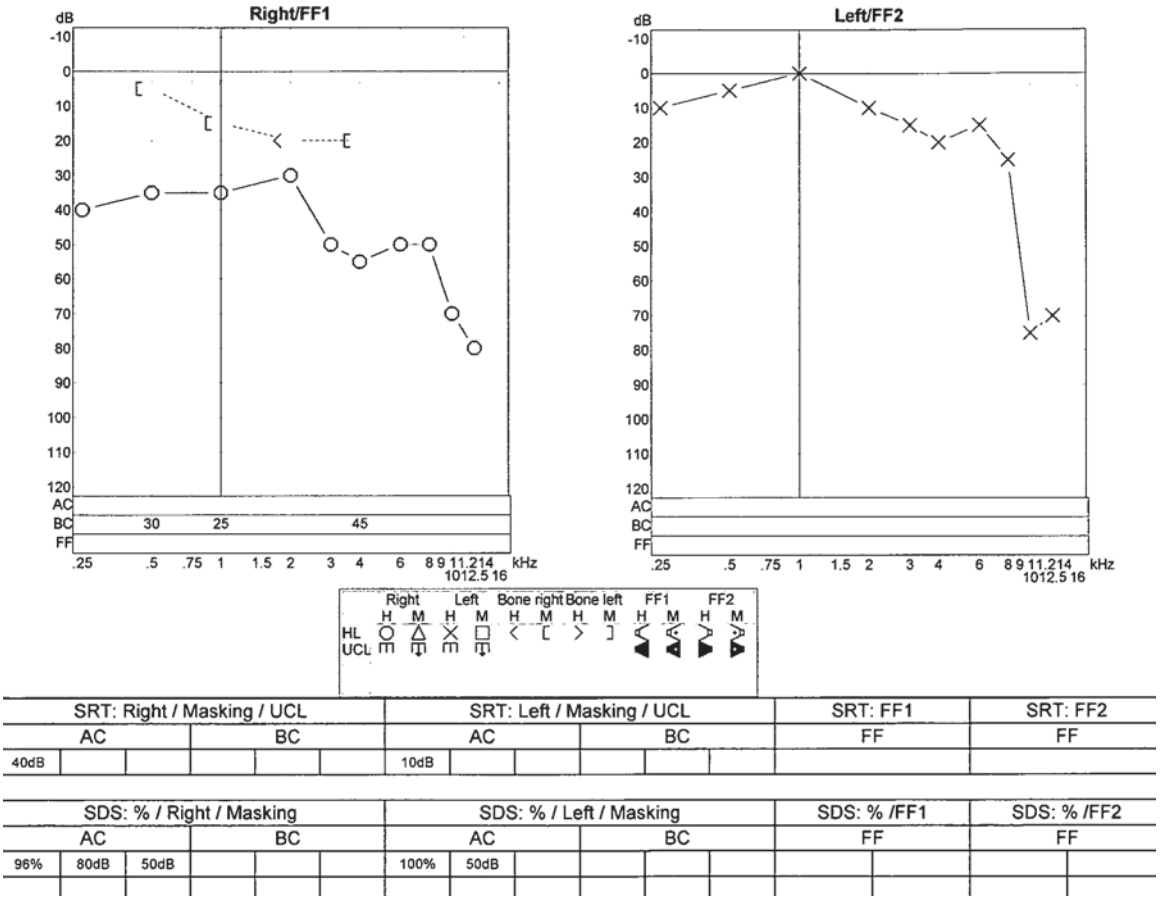
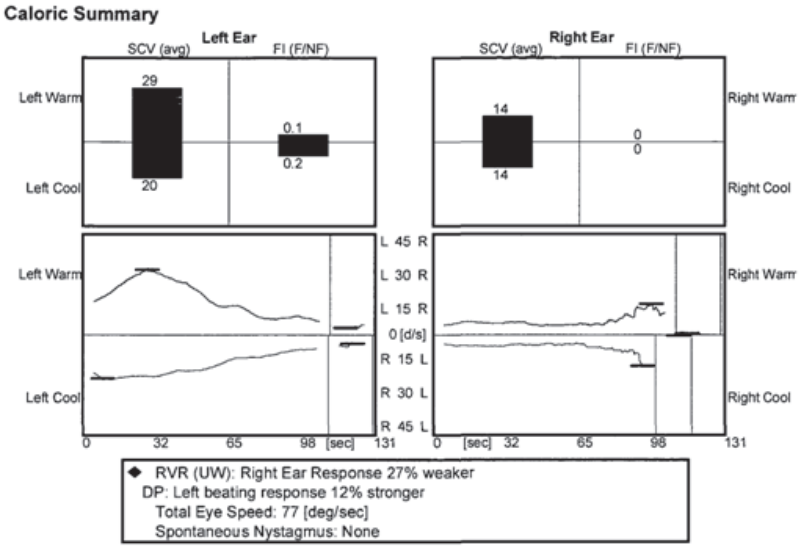
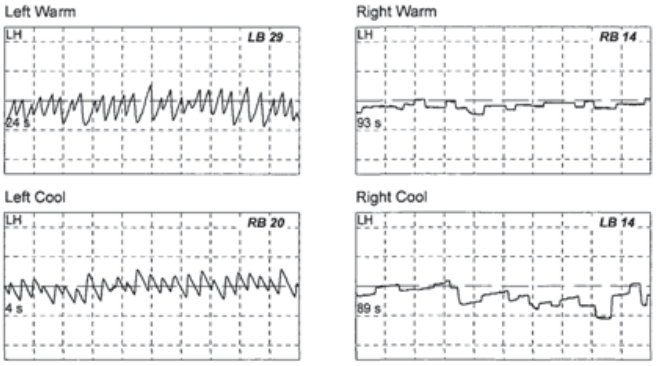


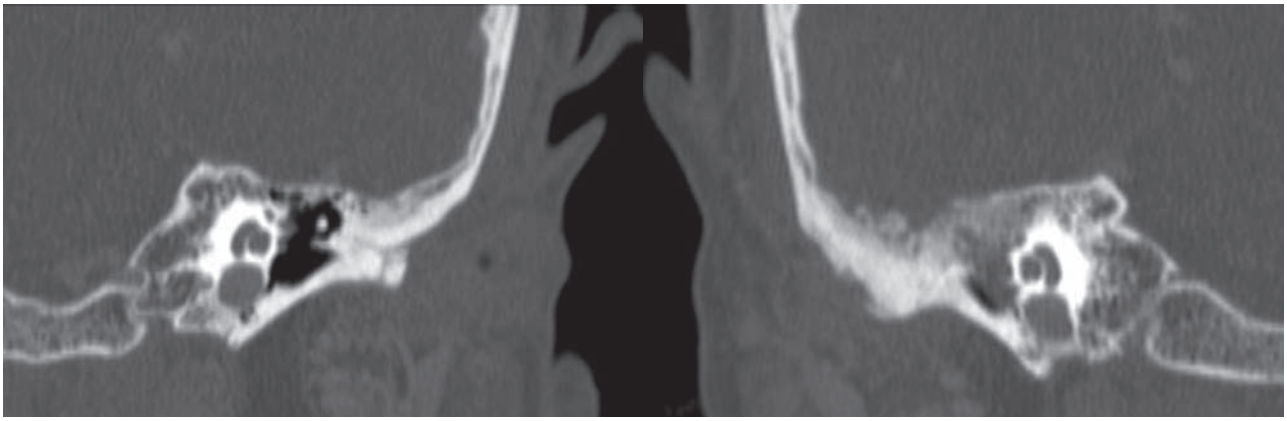
Fig. 30.2 Middle fossa meningioma presenting with headache, tinnitus, and hearing loss. **(A)** Endoscopic view of the ear canal showing reddish, bulging right tympanic membrane and a rim of serous fluid anteriorly compared with the normal left ear. **(B)** Audiogram showing a conductive hearing loss in the right ear. *(continued)*

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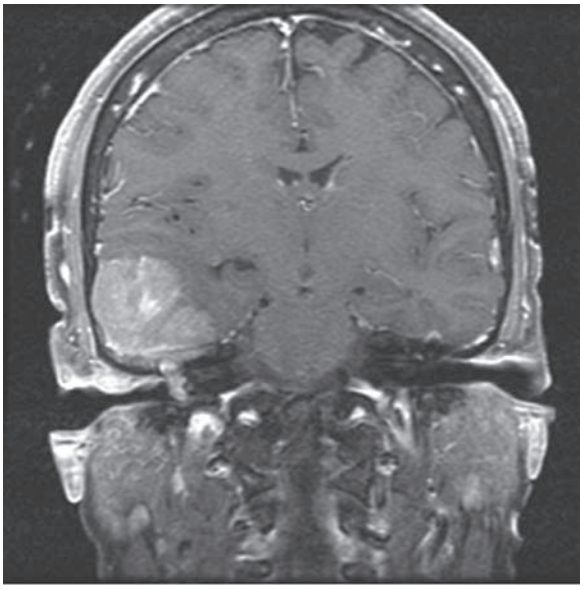
C



D

Fig. 30.2 *(continued)* Middle fossa meningioma presenting with headache, tinnitus, and hearing loss. **(C)** Videonystagmography showing a 27% reduced right caloric response. **(D)** Coronal noncontrast computed tomography of the temporal bone through the cochlea. The right side shows hyperostosis along the tegmen not seen on the left side. *(continued)*

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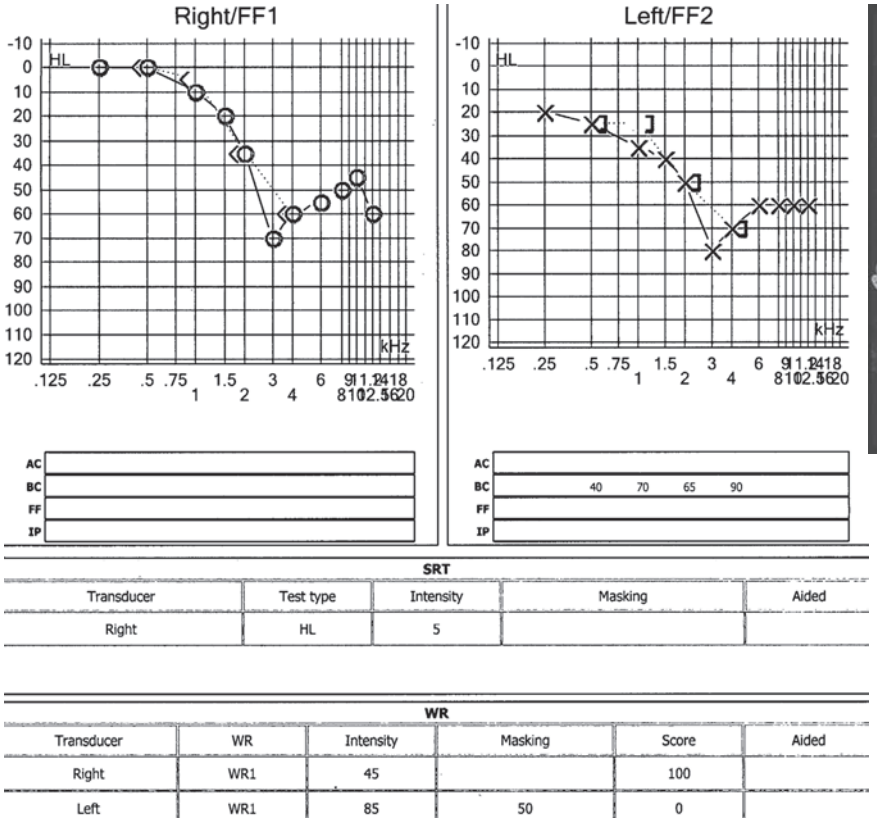


E

Fig. 30.2 *(continued)* Middle fossa meningioma presenting with headache, tinnitus, and hearing loss. **(E)** Coronal postcontrast T1 magnetic resonance imaging of a large middle fossa meningioma extending into the middle ear.



Fig. 30.3 Subtle left midface weakness due to meningioma.



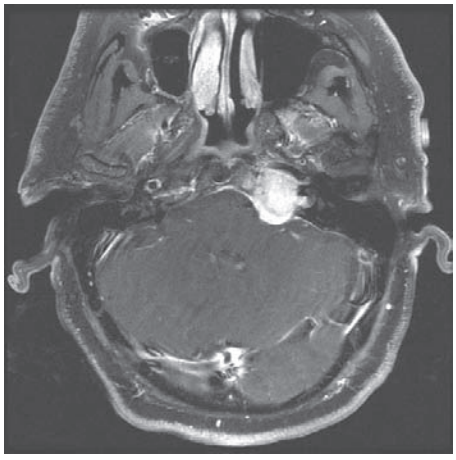
SRT

Transducer	Test type	Intensity	Masking	Aided
Right	HL	5		

WR

Transducer	WR	Intensity	Masking	Score	Aided
Right	WR1	45		100	
Left	WR1	85	50	0	

A

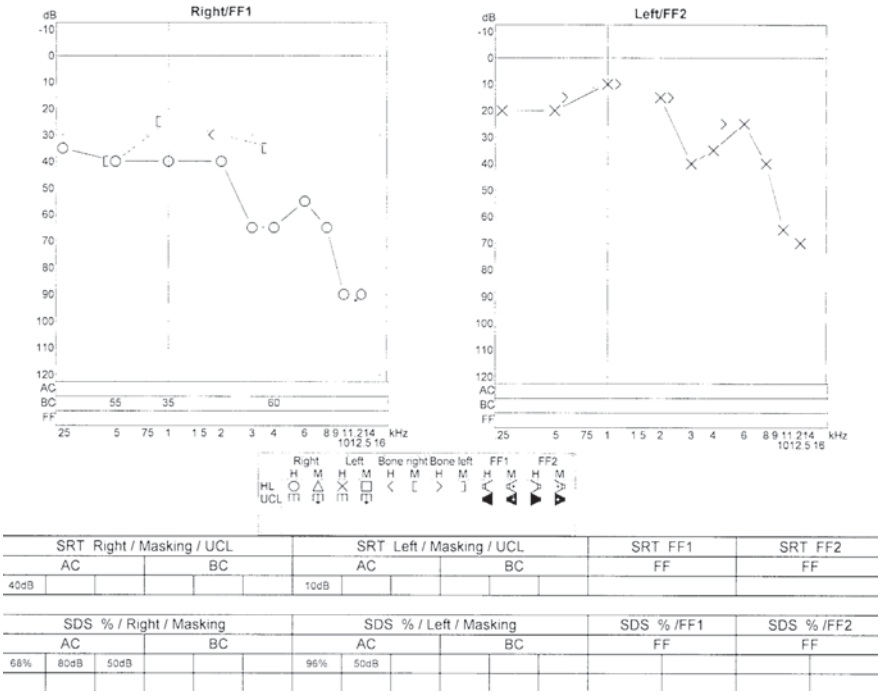


B

Fig. 30.4 Meningioma presenting with imbalance and hearing loss. **(A)** Pure tone testing reveals only a 10 to 20 dB difference between the two ears; however, speech discrimination is 0% on the affected left side. **(B)** Axial postcontrast T1 magnetic resonance imaging of an anterior petrous meningioma. Note that the tumor has displaced the internal carotid artery anteriorly and laterally.

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A



B

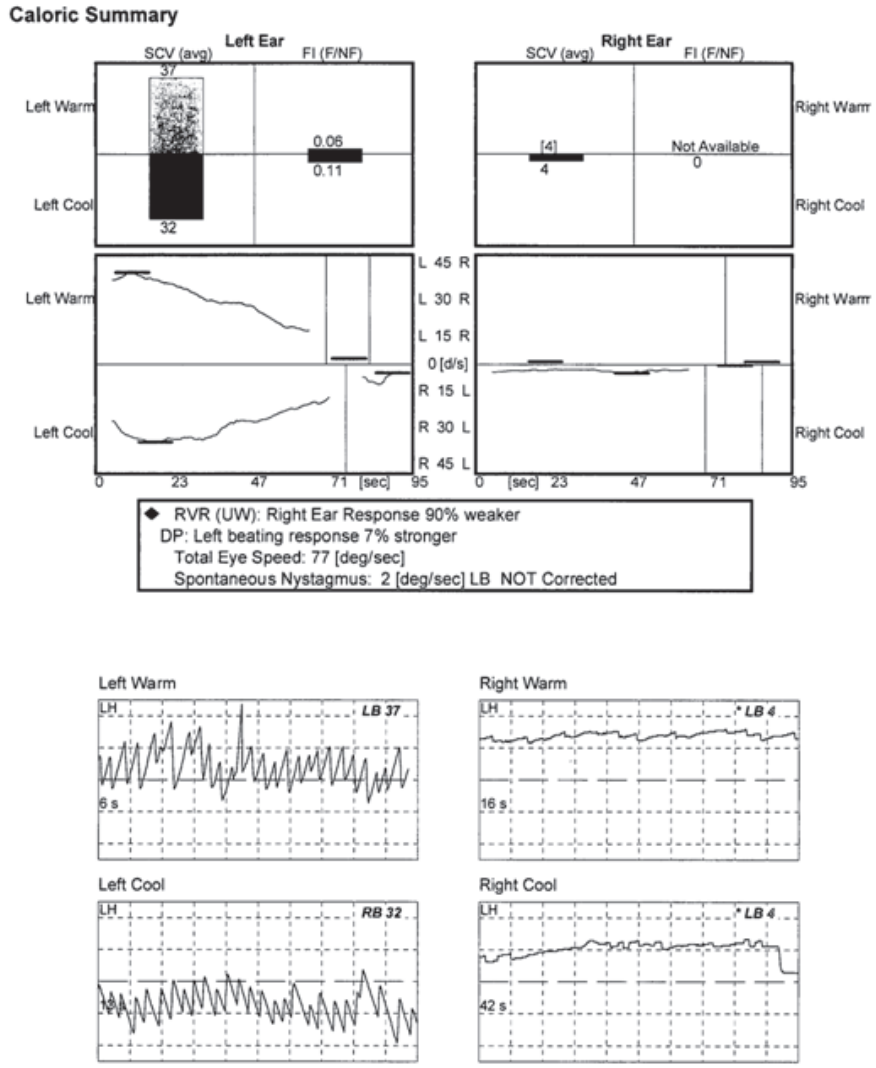
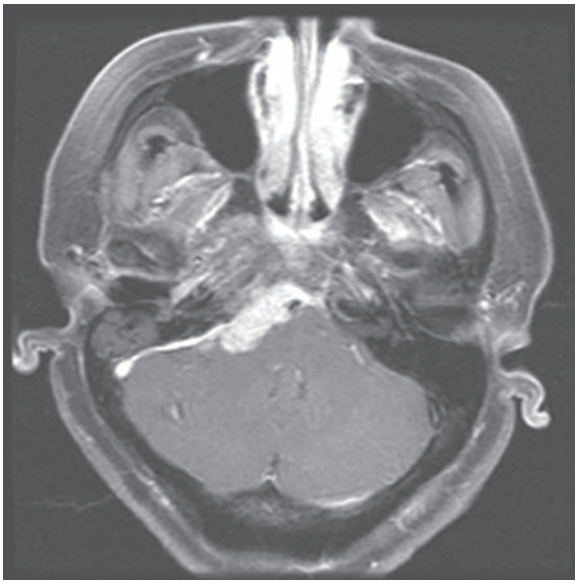
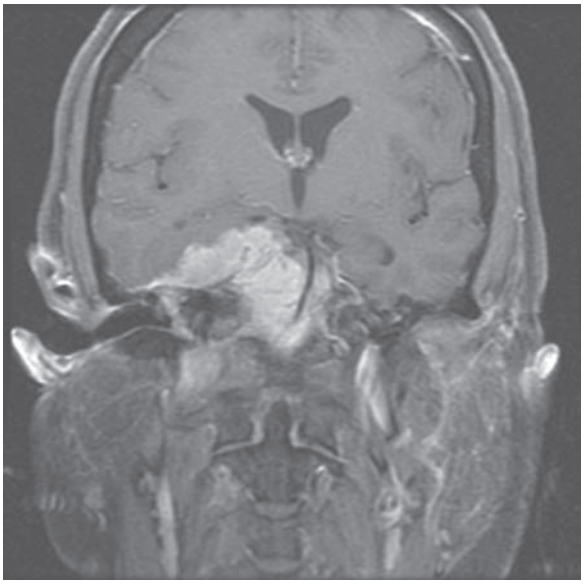


Fig. 30.5 Massive right petroclival meningioma presenting with a fluid sensation and hearing loss in the right ear. This massive tumor extends from the optic chiasm to the jugular foramen. **(A)** Audiogram showing mixed hearing loss. **(B)** Videonystagmography showing 90% reduced caloric response on the right side. *(continued)*

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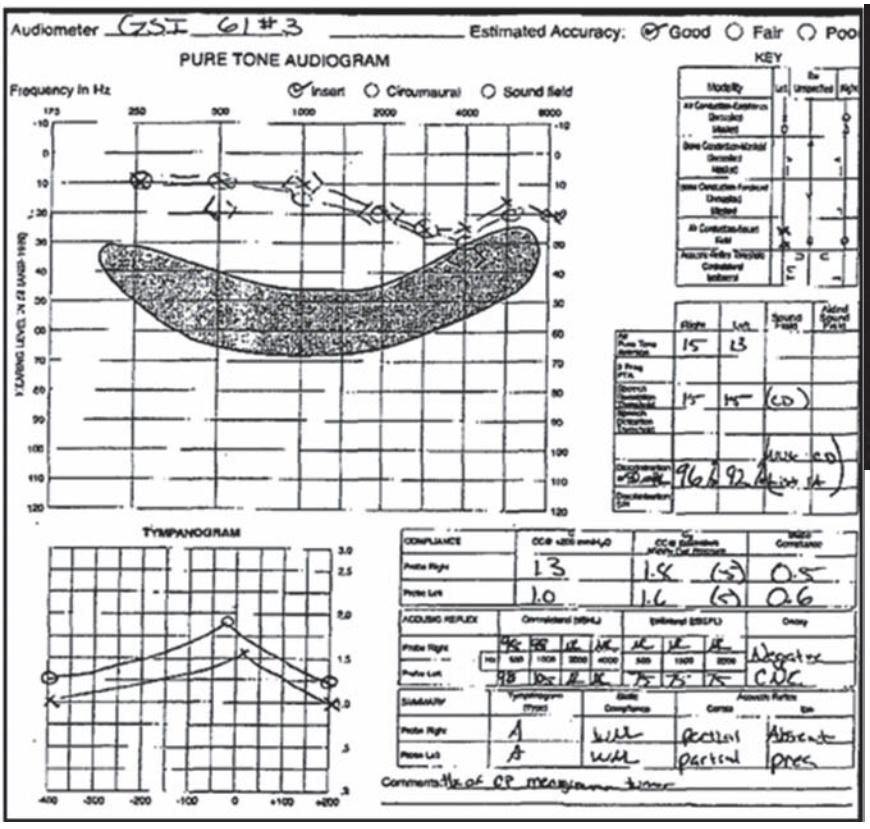


C

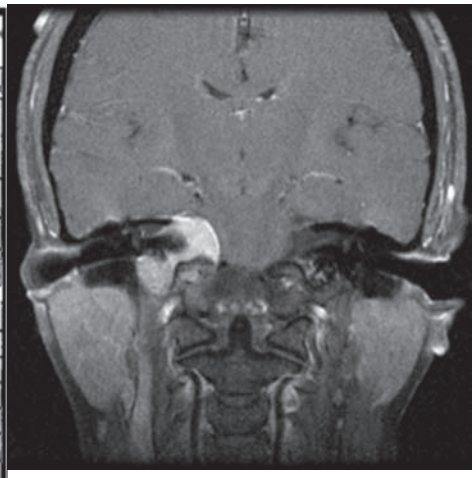


D

Fig. 30.5 *(continued)* Massive right petroclival meningioma presenting with a fluid sensation and hearing loss in the right ear. This massive tumor extends from the optic chiasm to the jugular foramen. **(C)** Axial magnetic resonance imaging (MRI) showing obstructive serous otitis media due to disease within the middle ear. **(D)** Coronal MRI showing displacement-encasement of the basilar artery. The tumor encircles the entire bony labyrinth.



A



B

Fig. 30.6 Meningioma presenting with hemifacial spasm. **(A)** Audiogram showing very mild sensorineural hearing loss at 3 and 4 kHz in both ears. The speech discrimination scores are 96% for the right ear and 92% for the left ear. **(B)** Coronal postcontrast T1 magnetic resonance imaging through the internal auditory canal (IAC) showing meningioma involving the right IAC and jugular foramen.

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Vestibular testing, such as electronystagmography (ENG), vestibular evoked myogenic potentials (VEMP), rotary chair test, and computerized dynamic posturography (CDP), can provide information on the function of the vestibular organs. These tests should be performed in patients who present with vertigo or symptoms of vestibular dysfunction. Arriaga et al reported that ENG was abnormal in 87% of their patients (**Figs. 30.2C** and **30.5B**).[11] However, these tests have not been used on a consistent and widespread basis and thus are not considered essential elements of the evaluation.

Speech and Swallowing

Equally important in the preoperative evaluation is attention to speech and swallowing. Flexible fiberoptic evaluation of laryngeal function is a necessary first step. Evaluation by a speech pathologist is useful to identify subtle changes in swallowing. Preoperative counseling regarding expected changes in speech and swallowing is beneficial to the patient.

Radiological Evaluation

Computed tomography (CT) and magnetic resonance imaging (MRI) are essential in managing temporal bone meningiomas (**Fig. 30.1B,C**). Excellent reviews of the anatomy and radiographic appearance of these tumors can be found in this textbook and throughout the literature.[3]

Features of meningiomas that differentiate them from acoustic neuroma are a broad base of attachment with a dural tail and areas of calcification with the lesions.[14] IAC meningiomas appear hyperintense on T2-weighted images, whereas acoustic neuromas tend to be isointense.

Molony et al have noted that meningiomas of the jugular foramen might not be conspicuous on postcontrast CT due to enhancement that is similar to the jugular bulb itself.[2] They have noted that bone destruction around the jugular bulb is less than that found for glomus tumors. The adjacent bone can be slightly sclerotic and mildly hyperostotic (**Fig. 30.2D**). Additional imaging is provided to correlate with otoscopic, audiometric, and vestibular testing (**Figs. 30.2E, 30.4B, 30.5C,D,** and **30.6B**).

Macdonald and colleagues have reviewed primary jugular foramen tumors and characterized several important clinical features of these tumors.[27] These tumors show (1) extensive skull base infiltration, (2) a centrifugal pattern of spread, (3) a permeative-sclerotic appearance of the bone of the jugular foramen, (4) the presence of dural tails, and (5) the absence of flow voids.[27]

Some authors report on the use of angiography to study the venous circulation and blood flow through the sigmoid sinus.[10,13] It is imperative to know that there is adequate contralateral flow in case the sigmoid sinus must be ligated, as in cases for jugular foramen tumors. However, even in their series of 13 patients, 92% of the ipsilateral jugular bulbs were already obstructed by the tumor at the time of diagnosis.

Differential Diagnosis

The differential diagnosis of primary temporal bone lesions is varied (**Table 30.2**). The clinical picture and imaging characteristics usually narrow the diagnosis. A vascular-appearing mass associated with pulsatile tinnitus and conductive hearing loss is most likely a glomus jugulare tumor. However, a small percentage of patients with such a clinical picture might have a meningioma in the jugular bulb.[2,10] Sanna et al reported that 30% of their patients with jugular foramen meningiomas had a middle ear mass and 30% had pulsatile tinnitus.

Glomus tumors are nearly 10 times more common than meningiomas in the jugular foramen region. The diagnosis of meningioma versus glomus tumor is important because meningiomas tend to invade surrounding bone and nerves and may have a higher recurrence rate than glomus tumors.[2]

Tumors that have an ear canal component can be readily biopsied in the outpatient clinic. Occasionally, biopsy of a middle ear mass is warranted as a separate procedure from tumor removal, especially when the diagnosis is in doubt.

Surgical Management

The goal of surgery is to remove the tumor completely and to preserve normal neural function. This goal is difficult to achieve even in experienced temporal bone surgeons' and neurosurgeons' hands.

For IAC tumors, meningiomas can invade into the cochlea and vestibule. The meningioma might displace the facial nerve posteriorly, whereas an acoustic neuroma usually displaces the nerve anteriorly. Furthermore, the meningioma might invade the facial nerve, making its preservation difficult.[14]

The difficulty in removing jugular foramen tumors is protean: (1) adequate exposure may require visualization in the intradural, intrapetrous, and extracranial spaces; (2) the facial nerve may overlie the jugular bulb, requiring transpositioning; and (3) many patients have normal hearing, which should be preserved if possible.[10] Patients presenting with cranial nerve deficits are unlikely to recover any function.[10] Facial paralysis, dysphagia requiring a gastrostomy tube, hoarseness, and hearing loss are all known morbidities for temporal bone meningiomas. New cranial nerve deficits might occur in up to 60% of patients (see **Table 30.1**). For this reason, patients must be adequately counseled about the surgical morbidity and long-term outcome for tumors in this location.[11]

Intraoperative monitoring of facial nerve function is imperative. Lower cranial nerve monitoring, via monitored endotracheal tube or needle electrodes in the palate or tongue, is important when these structures are at risk. Intraoperative ABR can be used for hearing monitoring, though hearing preservation is unlikely when the eighth nerve is enveloped by tumor.

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Table 30.2 Differential Diagnosis of Temporal Bone Tumors

Benign	Malignant
Cholesteatoma	Carcinomas
Paraganglioma	Squamous cell carcinoma
Meningioma	Adenoid cystic carcinoma
Neurofibroma	Adenocarcinoma
Lipoma	Endolymphatic sac tumor
Hemangioma	Basal cell carcinoma
Schwannoma	Sarcomas
Cholesterol cyst	Chondrosarcoma
Histiocytosis	Osteosarcoma
Meningocele	Neurofibrosarcoma
	Rhabdomyosarcoma
	Hemangiopericytoma
	Plasmacytoma
	Metastatic lesion
	Breast, lung, prostate, renal, skin

Key Factors for Decision Making in Temporal Bone Meningioma Surgery

- Location of tumor
- Size of tumor
- Evidence of growth
- Carotid artery involvement
- Hearing status
- Facial nerve status
- Lower cranial nerve function
- Extension of tumor to external, middle, inner ear
- Exposure with wide access to the tumor and minimal brain manipulation
- Sigmoid sinus sacrifice and status of the contralateral venous output system
- One- versus two-staged procedure
- Soft tissue reconstruction to prevent cerebrospinal fluid (CSF) leakage
- Health of the patient

Several different approaches have been used for temporal bone meningiomas. Middle fossa,[11] extended middle fossa,[28–30] transtympanic,[19] translabyrinthine,[11] retrolabyrinthine, transcochlear,[31–33] retrosigmoid,[13,34] combined middle fossa and retrolabyrinthine approach,[26,35,36] combined transpetrosal–transtentorial,[26,37,38] and infratemporal fossa[39] approach have all been described. Choice of approach depends on the neuroradiographic location and size of the tumor, the patient's health and presenting neurological deficits, the goal of total versus subtotal resection, and the surgeon's bias and level of experience.[26]

Intraoperative monitoring of the facial nerve is paramount for these procedures. Arriaga et al report anatomical facial nerve preservation in 96% of cases with monitoring, and 64% House-Brackmann I to II facial nerve function at 1 year.[11] Long-term facial function was worse for patients who underwent facial nerve transposition (i.e., transcochlear and infratemporal fossa approaches). Monitoring for the lower cranial nerves might help in intraoperative decision making regarding nerve sacrifice.

Hearing preservation can be attempted for meningioma surgery. Arriaga et al found that only 16 of 56 patients (29%) of their cases were amenable to hearing preservation.[11] Further, in a separate study, they found that eight of 12 patients with postoperative audiograms had good hearing postoperatively.[40]

For tumors around the IAC, surgical approach is dictated by tumor location. The extended middle fossa approach can be used for tumors that are anterior to the canal.[11] In this approach, the superior semicircular canal (SSCC) is blue-lined as a landmark for the inner ear. Bone can be removed safely anterior and medially to the SSCC to identify the IAC. Bone removal continues anteriorly and medially to the carotid artery to open the Kawase triangle. The advantage of this approach is the potential for hearing preservation and avoidance of transposition of the facial nerve. The disadvantage to this procedure is the narrow exposure to structures in the midline and lack of exposure to the posterior fossa.

A translabyrinthine or transcochlear approach might be required for tumors that are anterior, posterior, or inferior to the IAC. In this approach, hearing is sacrificed when the labyrinth and cochlea are removed. The ear canal is overseen and the facial nerve is transposed posteriorly. This facial nerve transposition usually results in permanent facial weakness. Most series report return of facial function to House-Brackmann III/VI function at 1 year.[32] However, Leonetti et al have described leaving the facial nerve within its bony canal as an intact "bridge" and avoiding facial nerve transposition.[38] The surgeon must work anteriorly and posteriorly to the nerve, but the results show normal or near-normal facial function in 20/26 patients treated with large (> 3 cm) cerebellopontine angle meningiomas.[38]

At the jugular foramen, surgical management is guided by preexisting cranial nerve deficits. All approaches will require a limited neck exposure, ligation of the jugular vein, and possibly control of the carotid artery.

An infratemporal fossa approach, as described by Fisch et al, allows total removal in a single-stage operation.[39,41] This approach allows exposure of the midline structures anterior and inferior to the carotid artery and usually requires transposition of the facial nerve distal to the geniculate ganglion.[11] As classically described, the bony external canal is removed and the external auditory meatus is oversewn. Because the labyrinth is removed, there is no attempt made to preserve the external canal or middle ear structures.

The transcochlear approach requires oversewing the external canal and performing a wide retrosigmoid decompression, labyrinthectomy, and exenteration of the cochlea. Decompression of the sigmoid permits medial displacement of the sinus and its potential ligation. This exposure allows decompression of bone for 180 degrees around the IAC. The tympanic bone is removed and the carotid artery can be decompressed. The facial nerve is skeletonized from the second genu to the stylomastoid

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foramen. If the tumor is located at the jugular foramen, the facial nerve can be displaced laterally for more exposure. If the tumor is located more anteriorly, toward the vertical segment of the carotid, then the facial nerve is displaced posteriorly by dividing the greater superficial petrosal nerve from the geniculate ganglion.

The transpetrosal–transtemporal approach combines a retro- and infralabyrinthine approach with an extended middle fossa approach.[26] The advantages of this approach include the potential for hearing preservation, the lack of facial nerve transpositioning, and the minimal requirement for brain retraction. This approach is ideal for tumors in the petroclival region with components in both the middle and posterior fossae.

Sanna et al have described a petrooccipital transsigmoid (POTS) approach that combines a retrolabyrinthine petrosectomy with a retrosigmoid craniotomy and occlusion of the sigmoid sinus and jugular vein.[10] The advantage of this approach is preservation of hearing and facial nerve function. This approach can be combined with other approaches (i.e., translabyrinthine or transotic) depending on the extent of disease. Sacrifice of the sigmoid sinus is not to be taken lightly. The surgeon must ensure that a dominant sigmoid sinus is not being sacrificed.[35] The potential complications that come from sacrificing a nondominant sigmoid sinus include: intracranial hypertension, retrograde transverse sinus thrombosis, temporal lobe infarction, temporary aphasia, and vein of Labbé injury.[35] This approach is rarely used today.

To achieve a lower recurrence rate, Molony et al recommend resection of all cranial nerves that are involved with the tumor and removal of bone until uninvolved bone is encountered.[2] This view is also held by Sanna et al.[10]

Total versus Subtotal Resection

The goal is gross total resection of the tumor; however, this goal cannot always be achieved. Subtotal resection is performed when a cleavage plane between tumor and brain stem is lacking, when vital neurovascular structures are encased, and when intact cranial nerves are encased in elderly patients.[10]

Closure and Soft Tissue Repair

Preventing CSF leakage is a primary requisite in any skull base procedure. The resection of jugular foramen meningiomas presents a particular predicament because a large dural opening is created along with a wide neck exposure. The occurrence of these two situations makes CSF leaks more likely than for other lateral or posterior skull base procedures, in one paper up to 33% of patients.[33] The typical mastoid dressing cannot give adequate pressure over the neck to overcome the intrinsic intracranial pressure. To avoid this complication, authors have used prophylactic lumbar drains, myoaponeurotic flaps, microvascular free flaps, titanium mesh, or staged procedures.

Postoperative Management

All patients are monitored closely in the intensive care unit. In addition to the neurosurgical issues, postoperative management will require close attention to swallowing and to prevention of aspiration. Most patients can be managed temporarily with a nasogastric feeding tube. Some patients with preoperative CN IX and X deficits can resume oral intake within the first postoperative week. Because many patients (perhaps as many as 60%) will experience a new lower cranial nerve deficit postoperatively, placement of a gastrostomy tube might be required for long-term alimentation.

Bedside evaluation by a speech pathologist can discover whether aspiration is occurring. Patients who demonstrate significant aspiration will probably need a tracheostomy. Modified barium swallow studies are used periodically in the postoperative period to assess the patient's recovery.

Vocal fold paralysis can be managed by vocal fold medialization, using either injectable material or an implant through a thyroplasty procedure. Occasionally, arytenoid adduction is required for these patients.

Patients who develop a CN XI deficit benefit from physical therapy to prevent frozen shoulder syndrome.

Perioperative mortality can be high in patients with jugular foramen meningiomas. In one series, the perioperative mortality was 14.3%.[13] New cranial nerve deficits are the most common morbidity associated with temporal bone meningiomas. New facial nerve palsy can occur in 10 to 30% of patients.

Radiotherapy

Radiotherapy has three roles in management of temporal bone meningiomas: (1) as primary treatment for patients unable to undergo surgery, (2) to control residual disease when growth continues postoperatively, or (3) as an adjuvant for malignant or atypical tumors. Radiotherapy has not been widely studied as a primary treatment for temporal bone meningiomas.

Stereotactic radiotherapy has been reported to control growth of partially resected tumors.[42–44] Subach et al have reported tumor control rates of 92% with gamma knife.[45]

Malignant meningiomas have longer overall survival with postoperative radiotherapy than without it.[46–48] Extrapolating these data to jugular foramen tumors, Ramina et al recommended postoperative radiotherapy for their jugular foramen tumor patients.[49]

Chemotherapy has been explored for patients with refractory tumors. Hydroxyurea has been a standard therapy, but targeted therapies are being developed.[50]

Recurrence and Survival Rates

Recurrence rates vary from 0 to 30%. In the pathology series reported by Thompson et al, the recurrence rate was 28% and is similar to the expected recurrence rate for intracranial meningiomas.[15] In this study, the 5-year

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disease-free survival was 82.1% and the 10-year disease-free survival was 78.6%. In their series, none of the patients received radiotherapy. However, their series does not comment on margin status, nor are the inclusion criteria specified.

Conclusion

Temporal bone meningiomas are rare tumors of the temporal bone. These tumors can present with nonspecific ear symptoms or progressive facial paralysis or jugular foramen symptoms. Audiometric testing and speech and swallowing evaluation are important to understand the degree of neurological deficit. CT and MRI are complementary; occasionally, angiography is needed to identify carotid stenosis or to define flow through the contralateral sigmoid sinus system.

Several surgical approaches have been used to address these tumors, with the goal of gross total resection and preservation of normal neural function. The talents of neurosurgeon and neurotologist are combined to create the optimum exposure and to resect the tumor as completely as possible.

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Chapter 31

Foramen Magnum Meningiomas

Michael D. Cusimano, Ahmed Faress, Youjin Chang, and Wilson Luong

Introduction

Although meningiomas account for three quarters of benign tumors of the foramen magnum (FM), as a group they account for only 1.8 to 3.2% of all meningiomas.[1,2] Like other meningiomas, they occur much more frequently in females and they rarely occur in childhood. First reported by Hallopeau in 1874,[3] their indolent development at the craniospinal junction makes clinical diagnosis complex and often leads to a long interval between onset of symptoms and diagnosis. The sensitivity of this region to surgical manipulation has sparked debate as to the most advantageous surgical approach. This chapter provides an overview of the relevant surgical anatomy, clinical features, and nuances of the management of FM meningiomas.

Foramen Magnum Anatomy

Several excellent reviews of FM anatomy have been published.[4–13] By definition, FM meningiomas arise from the arachnoid at the craniospinal junction. The borders of this zone, as defined by George[6] and George and colleagues[9] range anteriorly from the lower third of the clivus, to the upper margin of the body of C2, laterally from the jugular tubercle to the upper margin of the C2 laminae, and posteriorly from the anterior edge of the squamous occipital bone to the C2 spinous process.

The FM contains several critical neuroanatomical and vascular structures of which the surgeon must be aware (**Fig. 31.1**). The neural structures include the cerebellar tonsils, inferior vermis, fourth ventricle, caudal aspect of the medulla, lower cranial nerves (IX through XII), rostral aspect of the spinal cord, and upper cervical nerves (C1 and C2). The ninth through 11th cranial nerves arise as a series of rootlets along the anterior medulla, with the spinal component of the 11th cranial nerve arising midway between the anterior and posterior spinal rootlets of the spinal cord. The spinal accessory rootlets coalesce and ascend rostral to join the ninth, 10th, and cranial portion of the 11th nerve. Together, these nerves exit the skull through the jugular foramen. The 12th cranial nerve exits the medulla more anteriorly than the other lower cranial nerves and passes posterior to the ipsilateral vertebral artery (VA) on its course to the hypoglossal canal, located within the superior and anteriormost portion of the occipital condyle.

Major arterial structures located within the FM include the VAs, posterior inferior cerebellar arteries (PICAs), anterior and posterior spinal arteries, and meningeal branches of the vertebral, external, and internal carotid arteries. The suboccipital segment of the VA (also called V3) that courses from C2 to the dura consists of a vertical portion between C2 and C1 foramina transversaria (FT), a horizontal portion from the FT of C1 to the sulcus arteriosus (SA) of C1 and an oblique portion from the SA to the dura. The third portion of the V3 segment lies within the floor of the suboccipital triangle, curves above the lateral aspect of the posterior arch of C1, and proceeds rostral to pierce the dura mater just inferior to the lateral edge of the FM adjacent to the occipital condyle.

Understanding the anatomy of the suboccipital triangle is a key to a safe approach to V3 (**Fig. 31.2**). The V3 segment is fixed at the FT and at the entry into the dura but can be mobilized by freeing the periosteal sheath of the FT or the dural entry point. There are several other important anatomical considerations of V3: (1) in the neutral position the vertical and horizontal portions are perpendicular, but with head rotation they can run parallel, only separated by C1 lamina; (2) V3 can end in the PICA or the occipital artery; and (3) the proatlantal congenital anastomosis between the internal carotid and the VA is associated with an atretic VA and an extradural origin of the

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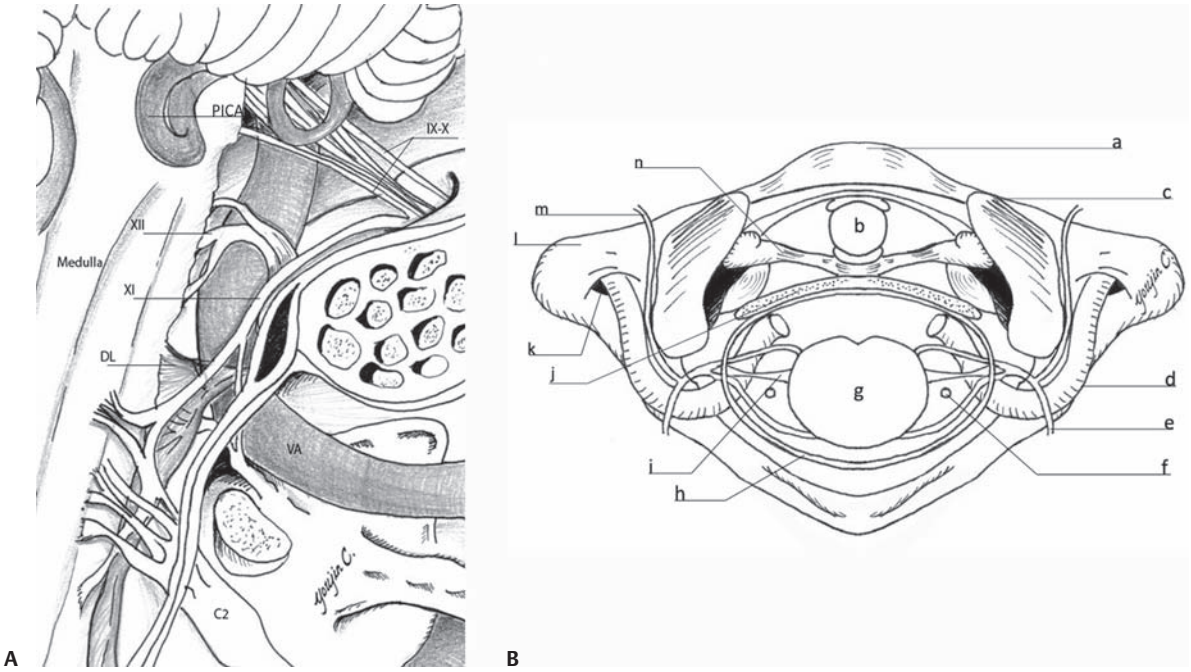


Fig. 31.1 **(A)** Posterolateral view of the foramen magnum. **(B)** Transverse view of the foramen magnum. a, anterior tubercle; b, dens; c, occipital condyle; d, vertebral artery (VA); e, posterior ramus of C1; f, nerve XI; g, medulla; h, dura; i, dentate ligament (DL); j, membrane tectoria; k, foramina transversaria; l, transverse process; m, anterior ramus of C1; n, transverse band of cruciform ligament.

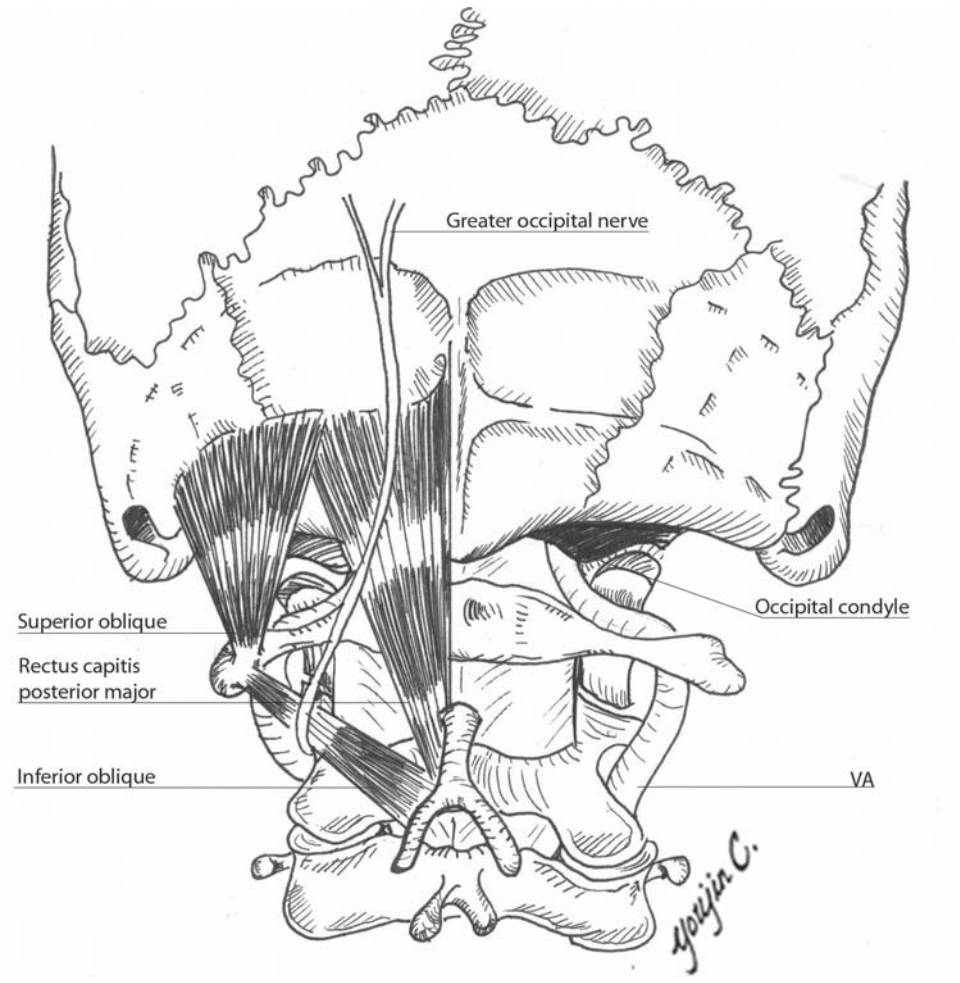


Fig. 31.2 Suboccipital triangle. The third segment of the vertebral artery (VA) courses from the foramina transversaria (FT) of C1, then along the lateral lamina of C1 to sit in the fat of the floor of the suboccipital triangle. It reaches the sulcus arteriosus (SA) of C1 and then courses in a superior and medial direction to pierce the dura medial to the occipital condyle. The rectus capitis posterior major sits deep to the rectus capitis posterior minor, which arises from the C1 and inserts along the midline and inferior to its major counterpart.

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PICA.[14] Branches of the VA that arise in this V3 segment can supply neural parenchyma (PICA has an extradural origin in 5 to 20% of cases) or provide supply to the tumor so a close review of preoperative vascular-based imaging is required to avoid vascular complications.[15] The V4 or intradural segment of the VA typically gives rise to the posterior spinal artery and PICA and then traverses anterior to the dentate ligament (DL) and anterior to the lower cranial nerves to join the contralateral VA beyond the hypoglossal canal at the vertebrobasilar (VB) junction.

The meningeal reflections around the FM are important to the surgical management of these lesions. The DL forms from a lateral projection of the pia into a flange that crosses the subarachnoid space. It connects the spinal cord to the dura at the FM via a series of "teeth," which are attached to the spaces between issuing nerve roots, and are situated posterior to the VA.[16] The uppermost dentate attachment is usually at the same level as the entrance of the VA intradurally.[13] The cervical contribution to nerve XI lies on the posterior surface of the DL, and the rootlets of C1 and C2 lie dorsal to XI. The VA moves from its dural entrance just anterior to the DL to move anterior to nerve XII and the nerves that enter the jugular foramen. From the surgeon's perspective via a posterior approach, XII is deep to the DL and sitting atop the VA, and the cervical part of XI and the high cervical rootlets sit on top of the DL. The arachnoid of the cisterna magna extends laterally to encompass the VA at its entrance from the dura. Maintaining this arachnoid plane allows the surgeon to dissect an apparently tumor-encased VA without hazard. If the tumor originates extradurally, tumor can extend between the adventitia of the VA and the arachnoid, thereby stenosing the VA. In these cases, dissection of the VA may place the adventitia at potential risk. It is sometimes difficult to identify the origin of a "transdural" tumor. At times, what looks like a "totally encased" VA may, in fact, be easily differentiable if this arachnoid plane is pressed around the VA. The surgeon should always determine whether the tumor is adherent to the VA; if it is not, it is likely that the arachnoid plane is present and the tumor will thus often be dissectible. If the VA is stentic, the arachnoid plane is often absent.

Classification of Foramen Magnum Meningiomas

FM meningiomas originate primarily from within the confines of the FM, or secondarily they invade the region after originating elsewhere. They most often arise intradurally but can also extend transdurally or rarely occur solely extradurally. We also classify the primary tumors according to their anteroposterior and lateromedial orientations. At surgery, the spinal DL delineates the anterior and posterior compartments. Of the intradural lesions, most (68 to 98%)[2] arise anterolaterally; a posterolateral origin is the second most frequent, purely posterior lesions are the third, and the least common are entirely anterior.

In thinking about the surgical management of the lesions, it is also advantageous to classify them according to their effects on critical structures, such as the VA, cervicomedullary parenchyma, and cranial nerves.

These lesions can also be classified in relation to the VA. Those that begin in the spine, so called "spinocranial" meningiomas, displace the VA superiorly. Those that begin at the entrance of the VA will encase the VA and displace the VA away from the petrous bone and the medulla away from the VA. Those that begin cranially, anterolaterally, or anterior to the entrance of the VA displace the VA *and* the medulla away from the petrous bone. In these cases, the VA and the medulla are in close apposition if the origin was along the lower clivus at or above the VB junction (**Fig. 31.3**). If the origin is lower on the clivus than the VB junction, the tumor may displace apart the relation between the medulla and the VA.

We also classify the site of origin of these lesions in relation to the neural foramina and cranial nerves. The *spinocranial* lesions will always originate below the FM and thereby displace the cranial nerves (and the VA) to the superior pole of the tumor. The surgeon can work from below to entirely decompress the neural structures in these cases to ultimately reach them at the end of the tumor resection. The purely *anterior* cranial lesions originating at the anterior lip of the FM will originate medial to the hypoglossal and jugular foramen and so displace the cranial nerves posterolaterally, and the surgeon will encounter nerves IX, X, and XII anterior or ventral to the dentate before the tumor's origin. If the origin is between the jugular and hypoglossal foramina, XII will be found medial and IX, X, and XI laterally. Most tumors originate inferolateral to the jugular foramen at the FM and so are similar to spinocranial meningiomas in their effect on the nerves.

Finally, we also recommend classifying these lesions based on their size relative to that of the FM (small: one third the transverse dimension of the foramen magnum; medium: one third to one half its dimension; large: more than one half the dimension of the FM). As a natural corollary of this size classification, we also classify the lesions according to the "surgical corridor" that they have created.[13] The surgical corridor is defined as "the space for surgical access to a lesion." It describes the space that the surgeon will work through to access the lesion. The surgical corridor can be enlarged naturally by a tumor displacing normal structures like the medulla oblongata in a confined space such as the FM. As tumors enlarge (**Fig. 31.4**), the corridor also enlarges, allowing access to the origin of the lesion more easily without retraction of the medulla or upper cervical spinal cord. The goal of the transcondylar approach is to enlarge the surgical corridor. By combining these classifications, we may describe a lesion such as that in **Fig. 31.2C** as intradural, spinocranial, with an adequate corridor, with encasement of the VA.

Clinical Presentation

The clinical presentation of FM meningiomas is protean, and the mean length of symptoms before diagnosis is 30.8 months, even in the era of magnetic resonance imaging (MRI).[16] The clinical differential diagnosis includes

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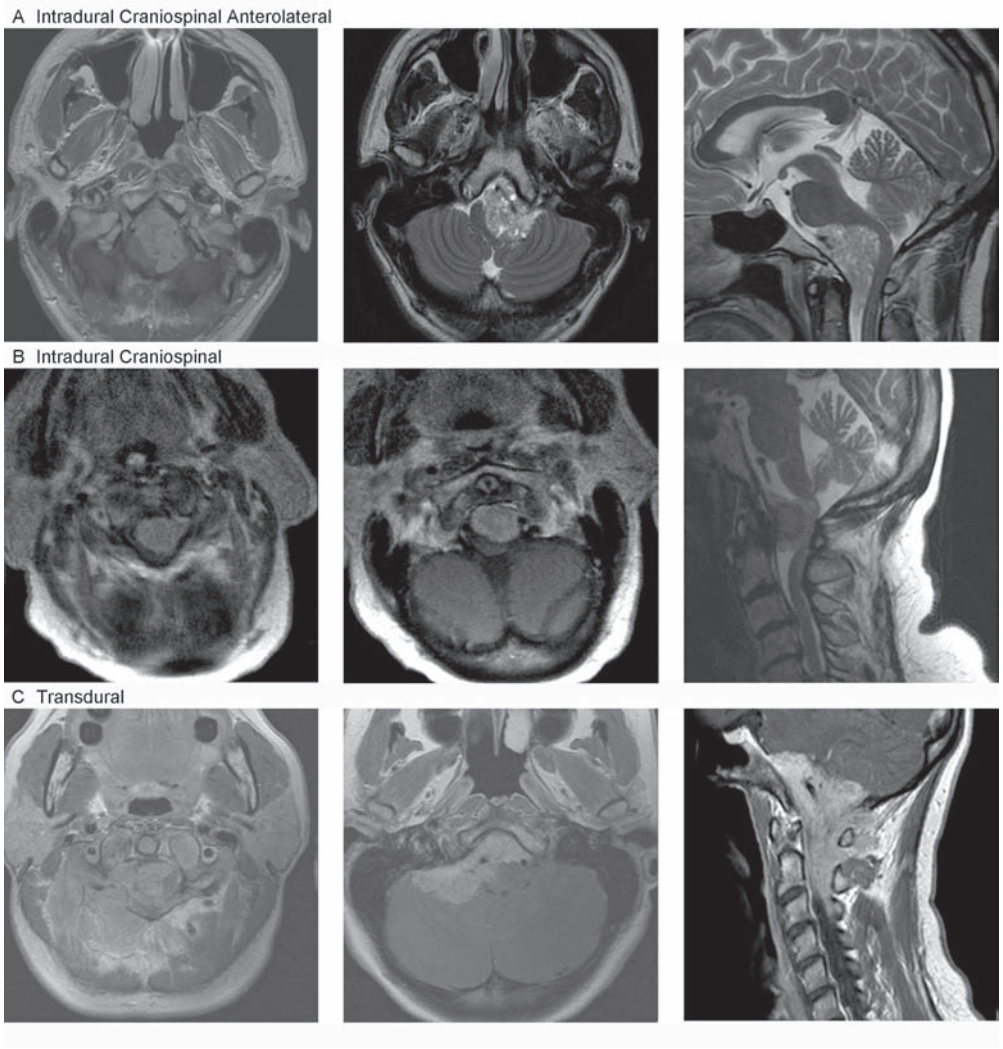


Fig. 31.3 Classification of foramen magnum (FM) meningiomas on magnetic resonance imaging and computed tomographic scans. **(A)** An intradural, craniospinal, anterolateral tumor displaces the medulla posteriorly and contralaterally, opening the surgical corridor, encasing the vertebral artery (VA). Note that on the T2 images (central and right) there is no T2 hypersignal. This meningioma was easily dissected from the brain stem and VA. **(B)** An intradural craniospinal tumor that is primarily anterior but also displaces the upper cord and medulla posterolaterally thus opening the access corridor for surgical resection. The VA is pushed up against the brain stem. **(C)** A transdural foramen magnum meningioma. This tumor transgressed the dura and the adventitial sheath of the VA. The patient presented with multiple cranial neuropathies, cerebellar deficits, and long tract findings. The tumor had an extensive intracranial base as well and was extremely firm and fibrous in the extradural component. The intradural component was dissectible from the VA and softer in consistency. Its adherence and firm texture with invasion of the extradural adventitial sheath led to a radical but incomplete resection. Decompression of the brain stem, upper cord, and cerebellum allowed a rapid clinical improvement.

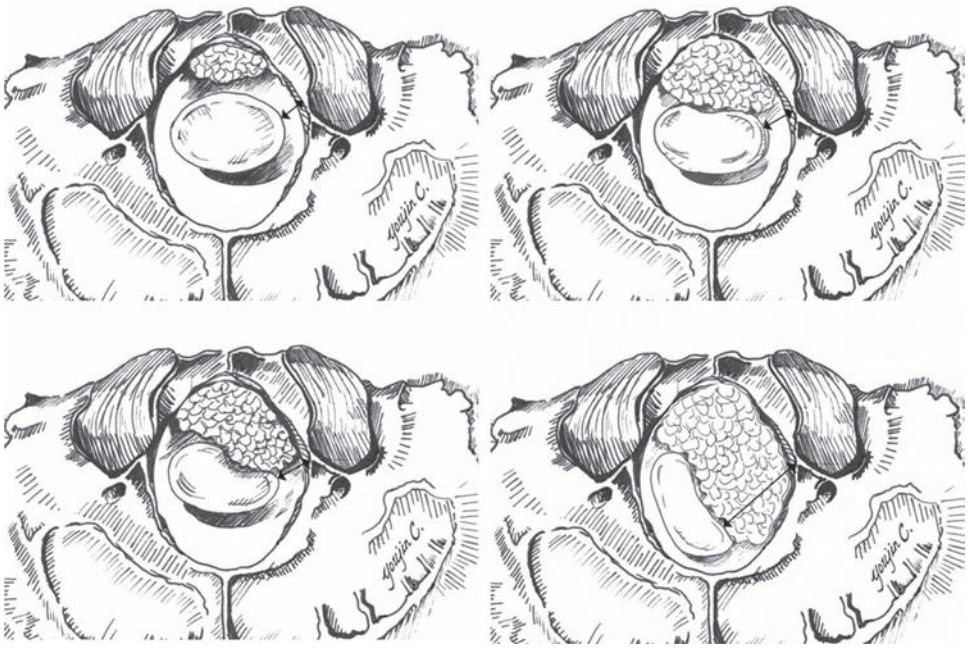


Fig. 31.4 Schematic showing growth of anterior and anterolateral intradural foramen magnum (FM) meningiomas and the development of the surgical corridor. The top left diagram shows the narrow space between the medulla and the bony wall of the FM (*double-headed arrow*) in a purely anterior FM meningioma. As the tumor enlarges (bottom left), the brain stem is displaced and the corridor widens. Often the patient begins to become symptomatic at this point but is rarely diagnosed until the corridor is widely established (bottom right).

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multiple sclerosis, amyotrophic lateral sclerosis, syringomyelia, and cervical spondylosis.[17–19] In a cursory examination the physician may miss subtle findings early in the stage of tumor progression, but later symptoms are often advanced, undeniable, and lead to significant and often permanent neurological deficit. Early features of FM meningiomas include occipital headache and upper cervical pain, which is often exacerbated by neck flexion or Valsalva maneuvers. Classic FM syndrome is defined by development of unilateral arm sensory and motor deficits, which progress to the ipsilateral leg, then the contralateral leg, and finally the contralateral upper extremity. Long tract findings characteristic of upper motor lesions are found paradoxically in the presence of atrophy in the intrinsic muscles of the hands. Motor loss is usually more pronounced ipsilateral to the lesion. Later findings include spastic quadriparesis and lower cranial nerve palsies. Patients may complain of cold or burning in one or both lower or upper limbs before other features arise. Slowly progressive lesions like these allow the development of accessory muscles to replace trapezius and sternocleidomastoid function. We therefore highly recommend that the patient be undressed and the sternocleidomastoid and trapezius muscles be closely inspected for atrophy. Likewise, the tongue should be inspected at rest for atrophy and fasciculation. Close attention to sensory testing of the C2 dermatome will help establish the diagnosis. Patients attest to initial sensory disturbances such as cold or burning dysesthesias, astereognosis, and anesthesia but often do not seek medical attention until intractable pain, motor deficits, or ataxia ensue. Terminal progression includes quadriplegia, an inability to maintain airway protection with secondary pneumonitis, and ultimately respiratory arrest.

Imaging Features

The role of neuroimaging is to confirm the clinical diagnosis and to allow the planning of a surgical approach. MRI is the modality of choice for defining tumors of the FM because it provides high-resolution images of soft tissue anatomy that is not susceptible to degradation by the surrounding skull base, a pitfall of computed tomographic (CT) scanning. Although plain T1-weighted MRI scans demonstrate excellent anatomical detail, they provide little discrimination between tumor and brain stem because the former may appear isointense, mildly hypointense, or hyperintense to surrounding brain. On T2-weighted images, meningiomas appear as isointense to slightly hyperintense compared with brain. The T2-weighted images should be carefully inspected for the presence of an arachnoid plane between the tumor, brain stem, and spinal cord. Edema depicted within the neuroparenchyma on T2-weighted sequences suggests that the pial membrane has been invaded; this should prompt an attempt at function preservation in which a near-total resection leaves a small thin plating of tumor intact.[20] The use of T1-weighted gadolinium-enhanced contrast imaging is particularly helpful in defining the dural attachment site of the tumor; additionally, it provides ready discrimination between tumor and brain stem, with often dramatic demonstration of brain stem distortion. Contrast-enhanced magnetic resonance or computed tomographic angiography should also be performed,[21] if available, to help demonstrate vascular anatomy, collateral vessels, and the effect of the tumor on the VAs. A VA that is encased and narrowed suggests that the adventitia of the artery has been invaded, and the surgeon needs to assess whether residual tumor will be left in the adventitia or whether reconstruction is necessary. In our experience, subtotal resection is the preferred approach in this instance.

Although MRI provides clearly superior soft tissue assessment, CT scanning with osseous algorithms remains the tool of choice for identifying calcification, hyperostosis, and osseous anatomy. Axial CT scanning allows planning of the extent of bone resection required to resect tumor safely because of the sharp contrast between bone and soft tissues. It is sometimes difficult to outline bone margins on MRI scans, and this technique may overestimate the size of the surgical corridor available for extirpation. It is clearly evident that optimal surgical planning requires both CT and MRI to assess appropriately bone and soft tissues, respectively.

An additional imaging modality that may assist surgery is CT angiography or conventional angiography with optional embolization of vessels that supply tumor exclusively. The dural blood supply typically arises as posterior and anterior meningeal branches from the VAs with the support of meningeal branches via ascending pharyngeal and occipital arteries. The tumor may derive its vascular supply from a dominant vessel, which, when subjected to contrast injection, is visualized as a "blush." If the vessel is accessible to endovascular catheterization, one might opt for preoperative embolization to diminish intraoperative bleeding during tumor debulking.[22] As well, angiography may help to define in the rare circumstance whether sacrifice of the VA is possible by defining collateral flow.

Preoperative Assessment

As is true with all oncology, neurosurgeons must remember that they are treating people and not just resecting tumors. Sometimes this requires consideration of subtotal resection or monitoring. In initial discussions about surgery, one must not assume that patients wish to undergo surgery because they are present at a neurosurgeon's office or that imaging has demonstrated a lesion. Often patients seek consultation to gather information about their situation, and depending on their age, ethnic background, and personal values, their decision-making process may not coincide with that of a neurosurgeon. It is therefore vital to reach an understanding of the patient's expectations of surgery as well as the individual's philosophy regarding quality of life issues. The possibility of residual tumor and subsequent treatment must also be discussed in relation to the risks of surgery.

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A careful and detailed history will often demonstrate that symptoms appeared long before the chief complaint. As the lesion progresses in size, the clinical course may seem to accelerate because compensatory mechanisms are exhausted and the neural compromise reaches critical levels. A history in which symptoms are rapidly progressive without a longer prodrome should raise clinical suspicion that lesions such as carcinoma or infectious/inflammatory entities may be present.

Potential surgery-related risks are not insignificant, and a careful assessment will give the astute clinician a better understanding of them in a particular patient. Care should be directed to lower cranial nerve examination. Deficits of any magnitude suggest neural compression and potential vasa nervosum involvement, thereby making the nerves more vulnerable to surgical manipulation. The ninth and 10th cranial nerves represent the afferent and efferent arms of the gag reflex, respectively, and play a pivotal role in protecting from aspiration pneumonia. Patients with unilateral preoperative gag deficits are often able to adapt because of the deficit's slow growth pattern and its chronic nature, which allow time for compensatory mechanisms to develop. An acute disruption of the gag reflex, however, can be lethal due to aspiration pneumonia. Thus preoperative and immediate postoperative endoscopic inspection of the pharynx and vocal cords should be performed to assess laryngeal function. New postoperative dysfunction should be treated using aggressive support measures, primarily by placing a gastric or jejunal feeding tube, and early tracheostomy if necessary. We routinely order an early otolaryngology and speech pathology consultation to test patients for vocal cord and swallowing functions preoperatively and postoperatively. Acute 11th cranial nerve deficits are sure to be problematic for the patient, especially in terms of shoulder abduction, but often the only symptom is shoulder pain. Patients with FM meningiomas rarely present preoperatively with an acute disturbance of this nerve, and its presence should lead one to consider alternate pathologies. Rapid onset with acute symptomatology related to XI often suggests an alternate pathology, such as carcinoma. Unilateral paralysis of the 12th cranial nerve may also be overlooked if the tongue is not inspected at rest within the mouth. Ipsilateral tongue deviation and furrowing are late and often irreversible signs of 12th cranial nerve palsy.

Planning for resection of FM meningiomas relies heavily on imaging findings and the clinical scenario.[23–25] Because the two posterior approaches to anterior FM meningiomas require dissection of skin and muscles in anatomically distinct regions, one must decide preoperatively which approach is most suitable for the given tumor. Selection is based on the basic skull base surgery principle of removing bone to provide a corridor of access to the tumor to allow total tumor resection and to preclude retraction of neurological structures. Evaluation of MRI data allows one to determine the relationship of tumor to the brain stem and its possible site of dural attachment. CT scans provide data regarding the osseous anatomy in relation to tumor. Most importantly, an assessment of the surgical corridor is made at this stage. Because the majority of FM meningiomas are anterolaterally situated, their growth tends to displace the brain stem in a posterior and contralateral direction.

In our opinion, this in situ retraction made by the tumor creates an adequate surgical corridor for resection of most of these lesions. In our experience, no drilling of the occipital condyle has been necessary to achieve resection, and in the cases in which residual tumor remained, resection of the condyle would not have affected the degree of resection.

Imaging also clearly displays the relationship of tumor to vascular structures. Encasement of the VA is not uncommon and should not be surprising intraoperatively during tumor debulking. Provided that encasement of the VA exists, proximal control of the vessel is prudent and may require mobilization of the VA at the C1 transverse foramen or, rarely, below, particularly if a transcondylar approach is needed. Assessment of the PICA is also of importance in some cases, particularly those in which one encounters an encased VA. Contrast-enhanced magnetic resonance angiography or CT angiography provides the best degree of noninvasive resolution and should be used to assess for encasement, contralateral VA, and focal narrowing that could indicate adventitial invasion. If it is hypoplastic, the surgeon may decide to leave residual tumor on the vessel if necessary or perform a bypass, rather than simply resect the affected segment. Conventional angiography with greater resolution is rarely necessary but can be revealing. An additional advantage of preoperative imaging is to determine whether the PICA originates above, at, or below the level of the FM.[13] Careful identification of PICA during routine posterior neck dissection is indicated if the PICA originates extradurally.

◆ Surgical Management

Intraoperative management requires a careful assessment of how the lesion has affected the normal anatomy. Understanding the relations of nerves, vessels, the medulla, the spinal cord, the DL, arachnoid, and bone are critical to performing optimal surgery (**Fig. 31.5**). Surgical dissection in which the cranial nerves and vascular structures are preserved is integral to FM tumor management. Every attempt should be made to keep the arachnoid with the patient and covering these structures. Perform dissection on the tumor's side of the nerves and vessels during surgery. Even in cases involving encasement of the VA, if the dissection is deliberate and selective to identify and preserve the arachnoid, successful complete dissection is frequently possible in nonstenosed VAs. It may be tempting to cauterize small vessels overlying tumor capsule, in light of the concept that the tumor is parasitizing blood supply from the meninges. If possible, however, these vessels should be left intact in the arachnoid because they may actually mislead the surgeon into moving outside the ideal plane where coagulation could potentially produce brain stem perforator ischemia. The cranial nerves, vessels, and neural parenchyma are generally on the patient-side of the arachnoid and not on the tumor side of the

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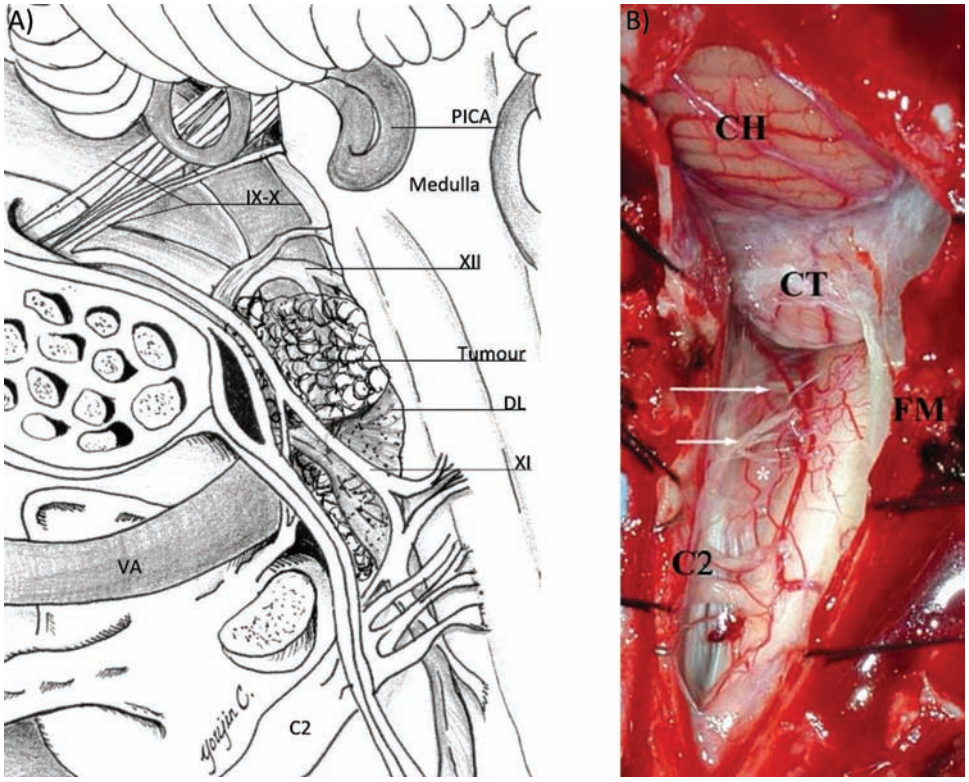


Fig. 31.5 **(A)** Artist's depiction of the surgeon's intraoperative view of the anterolateral foramen magnum meningioma. Note that most of the tumor is anterior to the dentate ligament and by definition is classified as anterolateral. The spinal component of nerve XI is on the surgeon's side of the tumor, and care should be taken not to injure it or mistake it for a leaf of the dentate ligament. **(B)** Intraoperative photograph demonstrating pathological displacement of anatomical structures of an anterolateral foramen magnum meningioma. A standard suboccipital craniectomy was performed without resection of the condyle. Tumor (*) is noted through intact arachnoid to the left of the rostral spinal cord and is draped with various nerve rootlets (*arrows*) and blood vessels. CH, cerebellar hemisphere; CT, cerebellar tonsil; FM, rim of foramen magnum; C2, C2 segmental nerve root; DT, dentate ligament; PICA, post inferior cerebral artery; VA, vertebral artery. Used with permission from Boulton MR, Cusimano MD. Foramen magnum meningiomas: concepts, classifications, and nuances. Neurosurg Focus 2003;14(6):e10.

arachnoid. Our general principle is to "leave the arachnoid with the patient" and "take the tumor from the patient."

Intraoperative monitoring is intended to aid the neurosurgeon in preserving neurological function. Somatosensory evoked potentials provide a measure of ascending pathways within the surgical field, whereas electromyographic recordings in the sternocleidomastoid muscle and tongue reflect 11th and 12th cranial nerve activity, respectively. If either of these modalities demonstrates a change, then the surgeon is alerted to a potentially threatening maneuver and may pursue a different manner of dissection. Although we have found electromyographic monitoring of the 11th cranial nerve useful, stimulation of the 12 cranial nerve occasionally will cause protrusion of the tongue, which, if not returned to position by the anesthesia staff, can lead to postoperative tongue swelling. There is insufficient evidence to support the use of routine evoked potential monitoring in this location. Changes, if noted, are always noted after the event has occurred, and if retraction is minimized, they rarely change intraoperative management. Currently, these modalities have not gained absolute clinical acceptance; their use instead is based on surgeon preference.

Surgical Approaches to Foramen Magnum Meningiomas

The FM can be approached via anterior, lateral, and posterior approaches. Each approach serves an important function and each was developed to deal with specific problems. The anterior transoral approach to the FM is rarely conducted to reach intradural lesions, such as meningiomas, because of problems with dural repair, risk of CSF leakage, and meningitis. Debate about FM meningioma resection primarily involves the posterior suboccipital craniectomy and posterolateral approaches, which necessitate drilling of the occipital condyle (**Fig. 31.6**). We limit our discussion to these approaches.

To simplify understanding of approaches to this region, we use the terms *suboccipital craniotomy* and *transcondylar approach*. Both require laminectomy, although the transcondylar is more commonly associated with mobilization of the VA from its lateral attachments to widen the surgical corridor. Terms such as *far lateral* and *extreme lateral* have only conjured up confusion and in our opinion should be avoided.

Suboccipital Craniotomy

Patient position: prone, head flexed on neck, neck kept neutral

Lateral decubitus, head turned 20 to 30 degrees toward floor

Craniotomy: suboccipital

With or without C1 laminectomy

Suboccipital craniotomy, or craniectomy, with or without cervical laminectomy, represents the classic approach to the FM meningiomas and is familiar to most neurosurgeons. For posteriorly situated lesions, we place the patient prone. The

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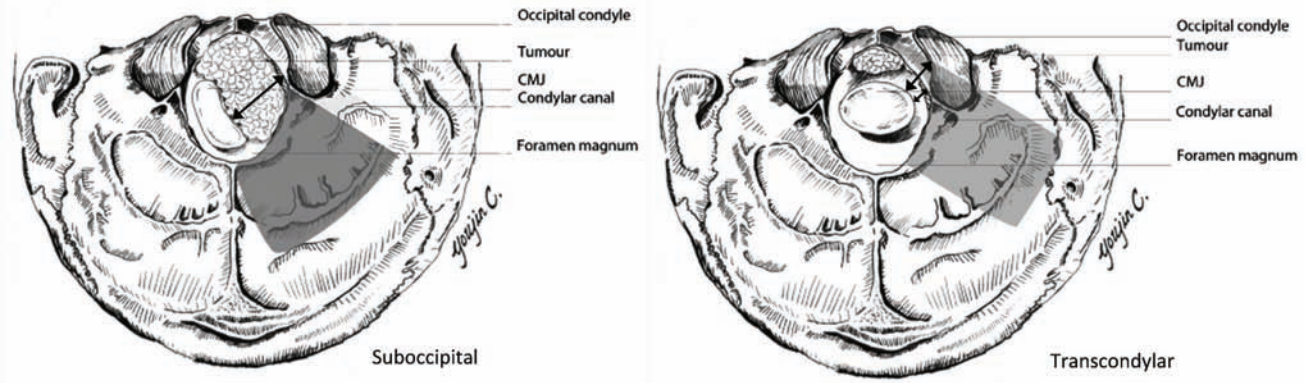


Fig. 31.6 Drilling of the occipital condyle is not necessary for the suboccipital approach. In the transcondylar approach, drilling of the occipital condyle enlarges the surgical corridor. CMJ, craniomandibular joint.

anethetist should always place a padding between the teeth to avoid clenching down on the endotracheal tube during flexion of the neck. For lateral or anterolateral lesions, the patient is placed in the lateral decubitus position with the vertex of the head displaced slightly downward to open the space between the occiput and the cervical spine. We also turn the head ~20 to 30 degrees toward the floor, depending on the extent to which the tumor is laterally situated. Because we use a diploscope mount to our surgical microscope, this allows both surgeon and assistant to access the surgical space and effectively use four hands to remove the tumor rather than only the surgeon's two hands. For the lateral position, we place the patient's contralateral shoulder overhanging the end of the operating table and allow the dependent arm to hang down from the bed, resting in a well-supported sling or armrest. Regardless of the approach, we use preoperative prophylactic antibiotics.

The surgical corridor defined on preoperative imaging must be easily within reach of the surgeon. The corridor should not be hidden under a large bulk of paracervical muscles deflected laterally. Sufficient soft tissue dissection to create access to the corridor is essential. Routine use of computerized neuronavigation helps to demonstrate subtle variations of anatomical distortions caused by these sessile meningiomas.

For midline posterior lesions, we make a midline incision. For posterolateral lesions that require exposure up to the condyle, we make a "hockey-stick" or inverted L–shaped extension laterally at the superior end of our incision just beneath the superior nuchal line. An S-shaped incision placed laterally can also be utilized. In addition, especially if a mastoidectomy is planned, a large C-shaped incision of the skin with the base toward the ear and a downward deflection of the suboccipital muscles can be performed. The bony exposure should include the superior and inferior extent of the lesion so that, at a minimum, the C1 lamina and superior part of the C2 are routinely exposed. Whichever the incision, cutting of the C2 nerve branches and the 11th cranial nerve distally in the neck should be avoided.

The VA is easily identifiable as it curves above the arch of the atlas, in the depth of the suboccipital triangle, providing proximal vascular control if required. We use neuronavigation to help determine the extent of the craniotomy needed. Although some authors prefer to conduct a craniectomy, we prefer a craniotomy because the incidence of postoperative occipital pain, we believe, is limited by replacing a firm protective covering over the dura, even if it covers only a fraction of the exposed dura.[26] If the surgical corridor to the tumor cannot be safely accessed as determined by neuronavigation before dura opening, more bone can be removed laterally toward the condyle. The craniotomy almost always has to be combined with a laminectomy to the inferior aspect of the tumor. At C1 the laminectomy should encompass at least the SA (i.e., the VA groove) in the lateral aspect of the C1 lamina. Care should be taken not to injure the thin-walled vertebral plexus of veins that surround the thick-walled VA. Of help in this procedure is bipolar coagulation with constant saline irrigation to avoid sticking of the instrument tips.

The advantage of suboccipital craniotomy includes visualization of the VA, brain stem, cranial nerves, and tumor in a safe, simple, and rapid manner. Criticisms of this approach primarily relate to the interposition of brain stem, cranial nerves, and vessels between an anterior tumor and the surgeon. The main problem we have seen with the simpler suboccipital craniotomy approach is the inability to mobilize the muscle mass of suboccipital muscles sufficiently laterally to get adequate lateral exposure. This can be overcome by extending the L-shaped incision into more of an inverted U and carrying the inferior limbs of the incision further inferiorly. Failure to do this results in the surgeon unduly retracting the neural structures to access anterolateral tumor or leaving residual. The purely anterior midline tumor without an adequate surgical corridor is completely obscured by these structures. The limitation of the unmodified suboccipital craniotomy approach is that it may necessitate undue retraction of critical neurological structures in cases in which the lesion is purely anterior with a narrow corridor. Fortunately, these purely ventrally located tumors are the rarest and can be dealt with using a suboccipital craniotomy combined with a partial condyle resection—what we call the transcondylar approach.

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Transcondylar Approach

Patient Position: lateral decubitus

Three-quarter prone, head turned 20 to 30 degrees toward floor

Craniotomy: suboccipital, C1 laminectomy

In an attempt to offer effective and safer resections, particularly in cases of more anteriorly situated lesions, the transcondylar approach was developed.[27] Several different names exist for the variations in this approach, and this leads to significant confusion regarding nomenclature.[21,28–30] In the literature on FM meningiomas, two major variations have evolved: (1) the suboccipital, which some have called the far-lateral approach, which necessitates removal of the FM rim toward the condyle and excision of the ipsilateral atlantal arch, and (2) the transcondylar approach, in which resection of some or all of the occipital condyle is required. The first of these is ultimately a suboccipital approach involving an appropriate soft tissue dissection to allow access to the surgical corridor. This is why we advise having only two named approaches to FM meningioma: the suboccipital and the transcondylar.

The transcondylar approach requires an inverted U–shaped incision with one limb of the U in the midline and the other along the anterior border of the sternocleidomastoid muscle. The sternocleidomastoid muscle is detached from the mastoid process and reflected as laterally as possible to avoid hindering access to the skull base. The superficial splenius capitis, semispinalis capitis, and longissimus capitis muscles are reflected downward to expose the underlying suboccipital triangle (**Fig. 31.2**). The suboccipital triangle is bordered by the superior and inferior oblique muscles and the rectus capitis posterior muscles; the VA courses in the fat of the suboccipital triangle below the occipital condyle. All three muscles are released and reflected away from the VA.

The craniotomy should extend from the medial to the sigmoid sinus, to the most medial aspect of the lesion, and to just above the rim of the FM. The residual bone over the sigmoid and FM is removed using rongeurs or a high-speed drill. The C1 laminectomy extends out to the foramen transversarium, which is unroofed separately. The posteromedial aspect of the occipital condyle and, if required, the C1 lateral mass are removed by drilling. If necessitated by the anatomy of the lesion, the foramen transversarium and C2 lamina are also decompressed. The VA is freed from collagenous tissue at the C1 foramen transversarium and adjacent to the condyle by using fine micro-dissection under surgical magnification. A fine Prolene stitch (Ethicon, Inc., Somerville, NJ) can be used to secure the VA in a medial position. Guided by preoperative planning of the surgical corridor and supplemented intraoperatively, if necessary, by computerized neuronavigation, the condyle is progressively removed in a mediolateral direction. Anterior condylar resection can include liberating the hypoglossal nerve from its canal if necessary to create an adequate surgical corridor. Because bone removal is extended much more laterally than in the suboccipital craniotomy, the ipsilateral VA is situated in the center of, or medially in, the "surgical corridor" before dural opening. The dura is opened by making an incision that parallels the lateral margins of the craniotomy, with the base of the flap located medially. A ring of dura can be left attached to the VA where it is pierced. This maneuver allows the artery to be retracted away from the surgical corridor, thereby providing a clear view of the anterior portion of the brain stem and rostral cord. Occipitocervical fusion is recommended in condylar resections of 50% or greater.[31]

◆ Personal Experience

Between 1992 and the end of 2009, 20 (3.4%) of 588 patients with meningiomas underwent resection of the lesion from the FM at our institution. Eleven of these arose from the anterior or anterolateral rim, five had a lateral origin, and four were predominantly posterior. Eighteen of the 20 patients were women, and the mean age was 52 years (range 34 to 79 years) in the entire series. The most common complaint was occipital pain with progression to hand paresthesias. Typically the mean duration of these symptoms was 11 months (range 2 to 34 months).

All patients underwent preoperative CT, MRI, or both. In cases of predominantly anteriorly located lesions, a suboccipital craniotomy and hemilaminectomy were undertaken. An adequate surgical corridor was obtained in all patients, and retraction of neural elements or resection of the occipital condyle was deemed unnecessary in all cases. Total excision of the tumor was achieved in 15 patients, and there was no recurrence as of the last follow-up examination. In one patient with MRI-documented residual tumor growth, gamma knife surgery (GKS) was performed 7 years after resection. One patient with a recurrent tumor originally operated at another institution had a densely calcified but growing lesion that we treated with fractionated radiotherapy after a second subtotal resection. This patient has remained stable for 4 years. Subtotal resection was not necessitated by the poor quality of the surgical corridor but rather by the lesion's adherence of the VA and the perceived risk of damage to the vessel if total excision were attempted.

Cerebrospinal fluid (CSF) leakage and transient worsening of preoperative symptoms were the only surgery-related complications. In three patients, CSF leakage developed, with two also having hydrocephalus. Both of these patients required a ventriculoperitoneal shunt. One of the patients with hydrocephalus also developed meningitis, which was treated with antibiotic therapy. Duraplasty was conducted to treat the uncomplicated CSF leak. Pseudomeningocele was associated with incomplete dural closure and we now recommend tight dural closure or duraplasty in all cases. There was no surgery-induced death; however, one patient suffered lateral medullary ischemia and one patient sustained a mild Brown-Séquard syndrome. At a mean follow-up pe-

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riod of 33 months (range 1 to 104 months, median 29 months) improved function was evident in 15 patients, there was relatively unchanged status in two, and three (two ischemic deficits, one meningitis) were more impaired functionally postoperatively. Of the two with unchanged status, one continues to experience unilateral leg weakness that was present preoperatively, and the other patient remains stable. The one patient with residual enlarging tumor who underwent GKS continues to experience hand ataxia and subtle neurocognitive difficulty 13 years after surgery.

◆ Nonsurgical Management

We have tended to recommend resection to patients with reasonably sized tumors in the FM (even with minimal symptoms in younger patients) because of the lack of space for future tumor growth or swelling during other treatments such as radiotherapy. The ideal treatment of meningiomas is a safe and complete resection. If contraindications to surgery exist or if the patient elects not to undergo surgical resection, then radiotherapy should be considered. Because of the critical anatomy within the FM and the size of most tumors (less than 14 cm^3 in volume), we usually recommend focused GKS rather than standard conformal radiotherapy if the lesion is accessible.[32] The Perfexion unit (Elekta) can be used for inferiorly situated lesions, excluding lower than 15 cm from the vertex of the cranium. Patients with small residual tumors undergo monitoring to assess growth rate, and in the event that the residual lesion grows, we recommend GKS.[24,33,34] In patients with multiple recurrent tumors or in whom the aforementioned contraindications exist, we search for alternatives to administer some form of biological therapy or chemotherapy, such as hydroxyurea therapy, with or without cyclooxygenase-2 (COX-2) inhibitor management.[35] There is a critical need for more effective treatments of these difficult to manage recurrent lesions; this is covered more fully in Chapter 41.

◆ Clinical Outcomes

Early reports of resection in cases of FM meningiomas were associated with a surgery-related mortality rate of 5 to 13%[36,37] and a morbidity rate of 36%.[38] Postoperative complications include intracranial hematomas, CSF leakage, meningitis, lower cranial nerve palsies, hemiparesis, quadriparesis, and aspiration pneumonia.[39] In the past decade, greater than 75% gross total resection has been reported in series, and morbidity rates have been lower than previously documented (**Table 31.1**). Anteriorly placed tumors, small tumors, tumor invasion, extradural tumors, and recurrent lesions naturally have worse outcomes. Outcome has been similar in series involving condylar drilling and those in which it has not been an aspect of approaching the lesion. No randomized, controlled trial of the two principal approaches has been conducted nor is one likely to be performed. Surgeons will have to continue managing patients as individuals by undertaking careful assessments of critically relevant issues such as the surgical corridor and the status of the VA. When the transcondylar approach is considered necessary to create an appropriate surgical corridor, it should be performed. Otherwise, the suboccipital craniotomy is likely sufficient.

Unfortunately, no authors have reported functional outcomes in terms of multidimensional quality of life measures. It behooves all neurosurgeons treating these patients to consider quality of life issues foremost in decision making and in discussion of preoperative planning with patients and their families.[40]

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Table 31.1 Summary of Recent Series Addressing the Management of Foramen Magnum Meningiomas

Study and First Author	Year	No. of Patients	Foramen Magnum Meningioma Location			Recurrence (%)	Vertebral Artery Encasement (%)	Approach	Vertebral Artery Transposition*	Resection Jugular Tubercle*	Partial Mastoidectomy*
			Anterior	Lateral	Posterior						
Cusimano†	2010	20	55	25	20	10	50	EL, PM, SO	Y	Y	Y
Kandenwein[41]	2009	16	81.3	12.5	6.3	0		SO			
Wu[42]	2009	114	70.2	21.1	8.8	18	40.4	PM, FL, EFL			
Borba[43]	2009	15	53.3	46.7				Lat	Y	Y	Y
Shin[44]‡	2006	16						EL			
Bassiouni[45]	2006	25	32	57	11	4	43	FL			
Margalit[46]	2005	18	100					Lat			
Pamir[47]	2004	22	91		9		40	FL			
Boulton[13]	2003	10	60	10	30						
Parlato[48]	2003	7							Y		
Marin Sanabria[49]	2002	7	72.5		28.5			TO, SO, TC			
Nanda[50]	2002	6	100					FL			
Roberti[12]	2001	21						EL, TC			
Arnautović[2]	2000	18	100			11.1		TC	Y		
Salas[22]	1999	24	100					TC/ELTJ	Y		Y
Sharma[27]	1999	10	50		50			PM, FL			
Pirotte[51]	1998	6	100						Y		
George[8]	1997	40	45	52.5	2.5		38				
Goel[52]	1997	17	100				59	SO			
Samii[53]	1996	38	95		5	5	40	PM, LSO			
Bertalanffy[54]	1996	19	100					FL, SO, TC		Y	
Akalan[1]	1994	8	12.5	87.5				PM			
Babu[28]	1994	9	100					EL	Y	Y	Y
Kratimenos[55]	1993	8	100			12.5		FL	Y		
Crockard[56]	1991	3	100			33	33	TO	No		
Sen[57]	1990	5	80	20		80		EL	Y	Y	Y
Guidetti[58]	1988	17	82.4								
Gilsbach[59]	1987	5	100					FL		Y	
Meyer[38]§	1984	78						PM			
Guidetti[60]	1980	18	83.3		16.7						
Yasuoka[61]¶	1978	37	59.6	21.1	19.3			PM			

Abbreviations: EFL, extended far-lateral; EL, extreme-lateral; FL, far lateral/posterolateral; Lat, lateral; SO, suboccipital, TC, transcondylar; TJ, tranjugular; TO, transoral.
* Y indicates that the procedure was performed when necessary.
† Data include 10 patients from Boulton's study (2003) and 10 additional patients.
‡ Data derived from 46 patients with 16 foramen magnum meningiomas, 17 chordomas, 1 chondrosarcoma, 2 Schwann cell tumors, 2 glomus tumors, and 8 other type tumors.
§ Data derived from 102 patients with 78 meningiomas, 23 neurofibromas, and 1 teratoma.
ÿ Data derived from 18 patients with 7 neurinomas and 11 meningiomas.
¶ Data derived from 57 patients with 37 meningiomas, 19 neurinomas, and 1 teratoma.

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Number Condyle Resection (%)	Extent Condyle Resection	Instability (%)	Outcome (%)			Resection (%)		Transient Morbidity (%)	Permanent Morbidity (%)	Mortality (%)	Follow-Up (months)	Recurrence (%)
			Improved	Unchanged	Worsened	Total	Subtotal					
0	0	0	75	10	15	75.0	25.0	40	10	0.0	33.1	0.0
18.75	1/3	0	50	31.2	12.5	87.5	12.5			6.3	43.5	
0.88	1/3–1/2					86.0	14.0			1.8	90.3	0.9
53.33	1/3– 2/3					80.0	13.3	6.7	6.7	0.0	23.6	0.0
43.75	< 1/3									0.0	66.1	
0	0					96.0	4.0	40	8	4.0	73.2	0.0
50	Partial (9/18)											
95	1/3	0				95.5	4.5	27	4.5	0.0	40.0	0.0
0	0		70	20	10	90.0	10.0	40	10	0.0		
	< 1/2	0				86.0	14.0			0.0	24.0	0.0
29	1/3–1/2	0	80		20	100.0	0.0	72.5	5	14.0		
0	0		100			100.0	0.0		0	0.0	43.0	0.0
						76.0	24.0		21.5	9.5		
100	1/2–1/3	0	89	11		75.0	12.5	55	11.1	16.6	40.0	5.5
100	1/3	0				66.0	33.0			0.0	14.8	
0	0	Yes				100.0				15.0		
100	1/2–1/3	0				100.0	0.0		17	17.0		
100	Partial	0	90	2.5	7.5	87.5	10.0		0	7.5	57.6	0.0
11.80	1/3–1/4	0	100			82.0	18.0		6	0.0		
17.50	1/3	0				63.0	30.0	37	5	6.0	21.0	5.0
100	1/3	0				100.0	0.0		0	0.0		
0	0		88	12		100.0			0	0.0		
100	1/3–1/2	0				88.8	11.2	78	56	11.1	9.4	0.0
	1/3	0				87.5	12.5		0	25.0		
0	0	33			100	0.0	66.0	100	100	66.0	20.6	33.0
100	1/3–1/2	0	20	20	60	60.0	40.0		60	20.0		
						100.0			12	11.0		
	1/3					100.0	0.0		20	0.0		
			75		25					5.0		
			88.9		11.1					11.1		0.0
			73.9	13.0	13.0	100.0	0.0			3.5	122.4	0.0

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Chapter 32

Meningiomas of the Lateral and Fourth Ventricles

Engelbert Knosp and Alexander Bertalanffy

Introduction

Meningiomas arising from the choroid plexus of either ventricle are rare but challenging lesions and represent ~1 to 2% of all meningiomas.[1–4] In a recent review of 400 intraventricular meningiomas by Criscuolo and Symon,[1] the majority of the tumors arose within the lateral ventricles (80%), followed by those arising from the third (15%) and fourth (5%) ventricle, respectively. This stratification correlates well with the amount of choroidal plexus tissue within these ventricles. Among meningiomas within the lateral ventricles, the trigone is by far the most common location, with more than 80% of tumors arising at this site.[1–4] In contrast, intraventricular meningiomas were almost never found within the anterior horn, and those tumors arising from the temporal horn were relatively uncommon lesions. Interestingly, analysis of the culled literature on intraventricular meningiomas[1] disclosed a distinct preponderance of left-sided lesions (59%). Whereas in the adult population intraventricular meningiomas represent 1 to 2% of all meningiomas, in the pediatric population as many as 9.4% of all meningiomas are located within the ventricular system.[5,6]

Intraventricular meningiomas can grow to considerable sizes before becoming clinically symptomatic. In patients reaching medical attention, symptoms may be intermittent and initially often nondescript and nonlocalizing. Patients may also present with symptoms of acute intracranial hypertension.

Since the first successful resection of an intraventricular meningioma by Cushing in 1916, surgical treatment has been particularly challenging due to the deep location of the tumor. Transcallosal or transcortical routes are mandatory for sufficient resection.[7] Despite the fact that meningiomas in general respond well to radiosurgical treatment,[8,9] in view of that modality's size limits, its radiotoxicity, and higher recurrence rates,[10] the treatment of choice for intraventricular meningiomas still remains surgery.

History

The first description of an intraventricular meningioma was given by Shaw in 1854 in an autopsy case. Cushing was the first to successfully remove an intraventricular meningioma, in 1916. In the classical monograph by Cushing and Eisenhardt,[7] the three intraventricular meningiomas reported represented 1% of their total experience. Walter Dandy, another great neurosurgical pioneer, also published three intraventricular meningiomas in his monograph.[11]

In the following decades, several case reports and case series have been published.[1–3,6,12–14] Delandsheer was the first to summarize the entity of intraventricular meningiomas by publishing 175 cases.[2] Given that even today in high-volume centers the frequency of intraventricular meningiomas rarely exceeds one case per year, institutional series rarely report more than 20 cases. The largest recent series have been published by Guidetti et al in 1991,[3] Criscuolo and Symon in 1986,[1] Fornari in 1982,[15] McDermott in 2003,[6] Nakamura in 2002,[4] Bertalanffy et al in 2006,[16] and Liu 2009.[3]

At the Neurosurgical Department of the Medical University of Vienna, a total of 20 meningiomas arising from the lateral and fourth ventricles were described between 1980 and 2006[16] (i.e., during a period with sufficient computed tomography [CT] and magnetic resonance imaging [MRI]). Another three cases, managed before the availability of CT imaging, have been described earlier at our institution.[15] All larger series of intraventricular meningiomas, including ours, suffer from the relatively small patient number treated over a long time period, making robust assessments of surgical outcomes difficult and thus preventing intergroup comparisons of surgical strategies and approaches. A common problem, however, remains since Cushing´s time: how to avoid damage to the visual pathway while preserving the interconnectivity of the dominant temporoparietal cortex in intraventricular meningiomas.

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Clinical Presentation

According to recent reports, the majority of intraventricular meningiomas are larger than 2.5 cm upon admission.[4,16] In the recent Hannover series,[4] 81% of all intraventricular meningiomas considered were larger than 3 cm in diameter. In our series, the average tumor size was 5 cm, with the largest lesions reaching 8 cm; only two tumors with a mean size of 2.5 cm were found incidentally. To date, only Kim et al[10] have published on the radiosurgical treatment of small intraventricular meningiomas. With the widespread use of CT and MRI, however, it is foreseeable that the number of incidentally discovered small intraventricular meningiomas will increase and that the management of small asymptomatic lesions by either surgery or radiosurgery will become an important issue in the near future. Tumor size is—together with tumor location and the presence of hydrocephalus—an important determinant of the patient's clinical presentation.

In general, the clinical presentation of patients with symptomatic intraventricular meningiomas does not differ significantly from those harboring other intraventricular tumors. Usually the most common symptom is headache. Two thirds of our patients had a history of headache, frequently intermittent in nature, and reaching back several years in some. Ultimately, headache was present in nearly all of our patients upon admission. The most common symptoms of patients presenting with intraventricular meningiomas are summarized in **Table 32.1**.

Focal neurological deficits, including hemiparesis or visual pathway defects, were present in 15 to 78% of the cases reported.[1–4,12,16–18] In larger tumors and lesions at strategic sites within the ventricular system, blockage of the cerebrospinal fluid (CSF) pathways can lead to signs of intracranial hypertension. Initially, these symptoms may be intermittent. However, in one third of cases, symptoms of permanently elevated intracranial pressure, including nausea and vomiting, were the leading signs bringing patients to medical attention. In most severe cases, patients may arrive comatose and require acute external ventricular drainage as a life-saving measure.

Gait disturbance is common in most series. Together with cognitive deterioration, urinary incontinence, and visual disturbance, these symptoms are usually due to chronic hydrocephalus and are not considered local neurological signs. Occlusive hydrocephalus or partial obstruction of the temporal horn may be present in all cases.[4]

Visual symptoms are more commonly observed in larger intraventricular meningiomas and were present in one third of all patients.[3] The mechanism underlying visual disturbances can be either intracranial hypertension or damage of the optic radiation. In cases of acute occlusive hydrocephalus, intracranial hypertension is not necessarily associated with papilledema. Either or both visual field defects and papilledema were found in 85% of Guidetti et al's cases, with bilateral papilledema being present in 60% of cases.[3] Visual field defects were detectable in 15 to 67% of all cases upon admission (**Table 32.1**).

The incidence of seizures is surprisingly high, ranging between 7 and 30% in the literature. According to Guidetti et al,[3] seizures were most frequently generalized in nature, although focal epilepsy and complex partial seizures of temporal origin were also reported (**Table 32.1**).

Only large tumors arising from the lateral ventricle of the dominant hemisphere will result in symptoms of aphasia, alexia, or acalculia.[4,16]

In contradistinction to tumors arising from the third ventricle, endocrinological disturbances are not typical signs of lesions within the lateral ventricles.

Neuropsychological abnormalities are commonly encountered but only infrequently reported. Modern diagnostic evaluation should include psychometric tests to reveal impairment of verbal and intellectual functions. The mechanism underlying impairment of memory functions is either direct compression of eloquent parenchyma in rare cases of temporal tumor location or, more frequently, blockage of CSF pathways with subsequent entrapment of the temporal horn.

Diagnostic Procedures

Plain skull radiograms can demonstrate calcifications in both normal choroid plexus and calcified intraventricular meningiomas.[18,19] Nevertheless, the first correct diagnosis of an intraventricular meningioma was made by Dandy in 1918, according to Criscuolo and Symon,[1,11] after ventriculography. Pneumencephalography, one of the first diagnostic procedures in neurosurgery, is mentioned only for the purpose of historical completeness.

Ten years later, angiography using direct carotid artery puncture allowed the direct visualization of contrast-enhancing intracranial tumors. Meningiomas were usually easily detectable because of their characteristic shape and staining. Intraventricular meningiomas were typically identified as global masses with sharp margins projecting to the lateral ventricle. Their arterial supply is derived exclusively from the choroidal arteries. Tumor blood supply can be drawn from either the anterior or posterior choroidal arteries exclusively, or from both arteries in conjunction[3,4,12,20,21] (**Fig. 32.1A**). The angiographic tumor blush indicates the extent of tumor blood supply and gives sufficient information about the vascularity of the tumor. Moreover, detailed angiographic assessment can disclose the exact site where the feeding arteries enter the tumor, which is usually at the anterior or anteroinferior tumor margin. This information is crucial for the planning of surgical strategies, especially in highly vascularized tumors. In selected cases, the reason to perform diagnostic angiography preoperatively is to derive this crucial anatomical information and to decide the most suitable surgical approach in view of these data. Additionally, the venous phase provides information about the possible enlargement and displacement of internal cerebral veins and shows the drainage pattern to the vein of Galen. Although the hemodynamic information derived from angiography is precise and helpful, noninvasive imaging technologies have obviated the need for catheter angiography in the majority of these cases.

Theoretically speaking, preoperative embolization would be helpful in these highly vascularized tumors

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Table 32.1 Symptoms of Intraventricular Meningiomas

	Knosp, 2010	Nakamura, 2003	Guidetti, 1991	Criscuolo, 1986	Fornari, 1981	Mani, 1978	Kobayashi, 1971	Delandsheer, 1965
% Headache	86	46	80	90	–	40	70	–
% Increased intracranial pressure (vomiting/nausea)	78	–	40	30	–	5	40	30
% Hemiparesis	43	38	25	30	78	20	60	15
% Hemisensory	39	–	15	–	22	–	–	10
% Visual field defects	36	30	20	30	67	15	50	40
% Cognitive	29	53	20	–	56	–	–	40
% Seizure	7	–	35	35	–	10	20	20
No. cases	20	16***	22	10**	18	22	0	175*

* According to the literature published by Delandsheer, 1965.[12]
** According to the literature published by Criscuolo and Symon, 1986.[8]
*** According to the literature published by Nakamura, 2003.[37]

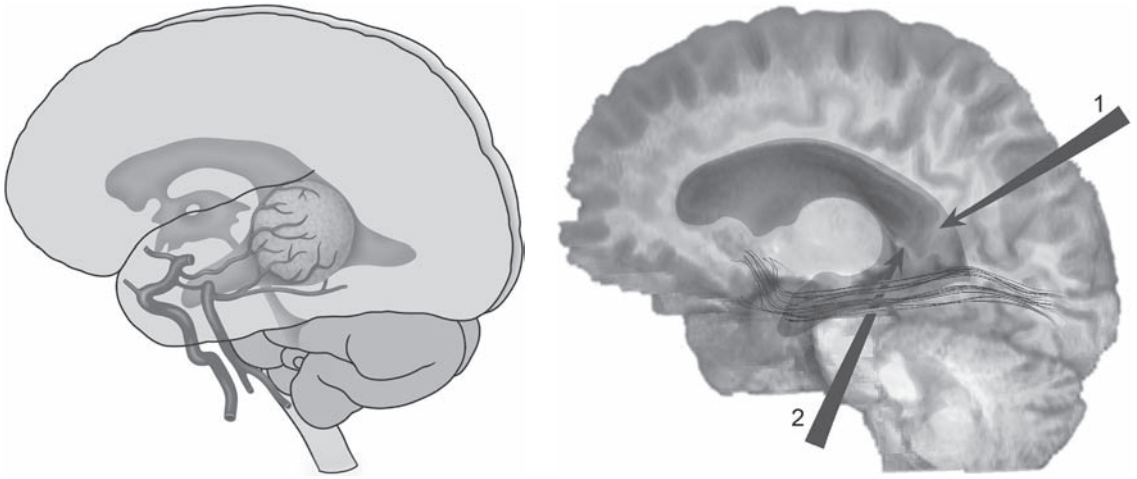


Fig. 32.1 **(A)** The vascular supply of the meningiomas within the trigone of the lateral ventricle is provided by the anterior and posterior choroidal arteries. **(B)** Fiber tracts of the optic radiation toward the calcarine sulcus: (1) transcortical parietal approach using the interparietal sulcus, (2) a low temporal approach to the trigone.

with their significantly enlarged choroidal arteries. The advantage of preoperative embolization would be most pronounced in parietal approaches because feeding artery control is obtained later than in other (i.e., temporal) approaches (to be discussed). Nevertheless, embolization was never performed in our series. Whereas superselective endovascular approaches to choroidal arteriovenous malformations for the purpose of glue embolization are usually possible using flow-directed microcatheter systems, this technique is not applicable in preoperative tumor embolization. In the latter cases, microcatheters of considerably larger diameter, allowing superselective polyvinyl alcohol (PVA) particle embolization, are required. These microcatheter approaches are usually more traumatic and may result in mechanical vasospasm, intimal damage, and infarction in this delicate vascular area. On the other hand, embolization followed by gamma knife radiosurgery has been reported as a possible alternative treatment for intraventricular meningiomas.[22]

Electroencephalography (EEG) does not usually have a role in the preoperative assessment of asymptomatic intraventricular meningiomas. In cases of documented seizure activity, EEG is the routine procedure to assess the efficacy of anticonvulsive treatment. In general, the rate of intraventricular meningiomas presenting with preoperative seizures ranges between 7 and 35% in the current literature (**Table 32.1**). In marked contrast, the rate of postoperative epilepsy after transcortical approaches ranges from 29 to 70% and in transcallosal approaches 0 to 10%.[1–3,12,16–18,23]

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CT gives the correct diagnosis of an intraventricular meningioma in the majority of cases. Additionally, the presence of hydrocephalus, possible intratumoral calcifications and hemorrhages, as well as the extent of peritumoral edema, are precisely outlined. The CT-derived information is usually accurate enough for emergency treatment (e.g., external ventricular drainage or third ventriculostomy).[24]

On noncontrast CT scans, the majority of intraventricular meningiomas usually appear as hypodense or isodense lesions. Because tumoral calcifications are encountered in roughly 50 to 90% of cases, CT is appropriate for their detection.[4,16,18]

After contrast administration, meningiomas usually show homogeneous and strong enhancement. Whereas choroid plexus papillomas often have lobulated surfaces, the shape of intraventricular meningiomas is rather smooth and globular. However, in three of the 16 cases reported by Nakamura et al,[4] the tumor shape was also described as lobulated.

Ventricular enlargement, either generalized or partial in nature, is a common finding on CT. The mechanism underlying partial ventricular enlargement is usually entrapment of the temporal and posterior horns. In the series of Nakamura et al,[4] all ventricular meningiomas located in the trigone presented with enlargement of temporal and posterior horns. Ventricular hemorrhage is a rare but potentially fatal complication of intraventricular meningiomas.[25]

CT angiography and MR angiography are standard diagnostic procedures for intraventricular meningiomas and provide crucial information about the arterial tumor supply, the vascularity of the tumor itself, the displacement of larger vessels, and the venous drainage pattern. When compared with standard catheter angiography, CT and MR angiography are noninvasive procedures that can be easily exported to, and processed in, modern neuronavigation systems.

There is no doubt that, in patients harboring intraventricular meningiomas, MRI is by far the most important imaging tool for the detection, surgical planning, and postoperative surveillance (**Table 32.2**). T1-weighted images with and without contrast as well as thin-slice T2 images are part of the basic workup used for standard neuronavigation. As in all cases of deep-seated tumors, a surface rendering of the convexity of the brain is created to plan the craniotomy, to exactly localize the site of corticotomy, and to define the trajectory to the target.

A more sophisticated MRI-based technique, specifically DTI tractography, has become an integral part of the radiological workup and is a prerequisite for accurate planning of the surgical approach to intraventricular meningiomas.[26–28] The main focus in these studies is on the course of the optic radiation and its relationship to the tumor. This preoperative morphological assessment is of paramount importance for the choice of the most suitable, atraumatic surgical approach (**Fig. 32.1B**). There is abundant literature describing the course of the optic radiation anatomically,[29,30] surgically,[26,30–33] and radiologically.[33–36] Still, one of the most impressive descriptions of fiber dissections was provided by Klingler in 1956.[37]

Additionally, functional MRI, MRI spectroscopy, and MR angiography are among the modern MRI-based technologies used for treatment planning. Functional MRI with motor and sensory tasks provides additional information to direct the appropriate surgical approach and to avoid parenchymal damage. MR spectroscopy[38] may be helpful in the differential diagnosis of intraventricular meningiomas and allows the differentiation between meningiomas and other intraventricular lesions (e.g., gliomas and neurocytomas). MR angiography is also able to detect the exact location of large bridging veins potentially obstructing the surgical approach and can demonstrate the displacement of important intraventricular veins.

In the majority of neurosurgical centers, neuronavigational devices are used in patients with deep-seated tumors. Some publications have outlined the usefulness of this procedure in the safe removal of intraventricular meningiomas.[39] We have integrated structural MRI (T1 postcontrast, T2), DTI imaging of important white matter tracts, and functional data into a three-dimensional (3-D) model. This information is of paramount importance for planning the exact surgical approach. Unfortunately, the accuracy of all these neuronavigational systems decreases over time with ongoing surgery and with the amount of CSF aspiration, which is a significant issue in all ventricular tumors.

Intraoperative MRI allows the surgeon to update the navigational information and can show the completeness of tumor resection if required.[27,40,41] Especially in cases of intraventricular meningiomas, intraoperative MRI has the potential to delineate the course of the fiber tracts of the optic radiation intraoperatively and repetitively.

Differential Diagnosis of Intraventricular Tumors

The differential diagnosis of intraventricular tumors includes meningiomas, choroid plexus papilloma, glial tumors, ependymomas, neurocytomas, and rare pathologies (e.g., epidermoid, plexus cysts, or tuberous sclerosis) (**Table 32.3**[14,42]). Most pathologies are easily diagnosed on grounds of their distinct location or characteristic appearance on CT and MRI. However, it is very difficult and sometimes impossible to distinguish intraventricular meningiomas from choroid plexus papillomas. Both pathologies are globoid lesions arising from the choroidal plexus and both exhibit a strong and homogeneous contrast enhancement. When compared with intraventricular meningiomas, choroid plexus papillomas are reported to have a lobulated rather than a smooth surface and exhibit an even more robust contrast enhancement. As previously mentioned, these distinctions are sometimes arbitrary in nature, and Nakamura et al[4] recently reported on three of 16 intraventricular meningiomas in their series presenting with a markedly lobulated surface reminiscent of plexus papilloma. When compared with choroid plexus papillomas, which occur more frequently in the pediatric population and arise more frequently

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Table 32.2 Radiological Signs of 20 Intraventricular Meningiomas Operated at the Vienna Medical University 1980 to 2009

Neuroradiological Features	Number	Percentage
CT/MRI: clear demarcation*	20	100
Homogeneous CE	20	100
Hydrocephalus	14	70
Peritumoral edema	15	75
Calcification	9	45
Cysts/formation of tumor	1	5
Angiography: AChA supply	3/4	75
PChA supply	4/4	100

Abbreviations: AChA, anterior choroidal artery; CE, contrast enhancement; CT, computed tomography; MRI, magnetic resonance imaging; PChA, posterior choroidal artery.
* CT has been performed in 20/20 cases, MRI in 14/20 and angiography in 4/20 cases comparison of applied approaches, morbidity and mortality and histology.

Table 32.3 Differential Diagnosis of Tumors of the Lateral Ventricle*

	Yaşargil, 1988	Pendl et al, 1992
Meningioma	9	8
Plexus papilloma	5	3
Neurocytoma	9	6
Ependymoma (II/III)	29	5
Subependymoma	9	1
Glioblastoma	10	5
Astrocytoma (I and II)	10	12
Tuberous sclerosis	8	–
Miscellaneous	19	15
Total	108	55

* According to Yaşargil 1988[14] and Pendl et al 1992.[42]

within the third and fourth ventricles, intraventricular meningiomas have a preponderance for the trigone of the lateral ventricle.[1,14,42–44]

Surgery

Increasing information about multidirectional white matter interconnectivity in the left temporoparietal lobe enhanced ongoing efforts to look for more sophisticated routes to enter the trigone of the ventricular system of the dominant hemisphere. During approaches to the ventricular trigone in the past, consideration was given to the preservation of the optic radiation, whereas preservation of parietal lobe interconnectivity seemed to be of less importance.

Neuronavigation is essential for the management of deep-seated lesions, with ventricular lesions representing one of the ultimate challenges for this technology. It should be pointed out, however, that neuronavigation has led to the (mis)conception of trying to look for the shortest and most direct surgical approach to a given lesion. Direct temporal transcortical approaches instead of transsylvian approaches to lesions of the mesial temporal lobe represent just one example for these misconceptions. In fact, the shortest route may not be the best. As a consequence, we have to keep our transcortical routes, not necessarily the craniotomy, as small as possible and as minimally invasive as possible to keep the inevitable damage to a minimum.[45–47]

Eighty percent of the meningiomas of the lateral ventricles arise from the choroid plexus in the trigonal area and grow toward the central part of the ventricle, the posterior horn, or, in rare cases, the temporal horn. Only exceptionally, meningiomas arise within the anterior horn.[4,23] In view of this distribution of intraventricular meningiomas, the discussion in this chapter focuses on approaches to the trigone of the lateral ventricle.

An acceptable approach to the trigone must not injure the optic radiation, the sensory and motor fiber tracts, or temporoparietal lobe function in the dominant hemisphere (**Fig. 32.2A, B**).

Because intraventricular meningiomas may reach considerable size (> 5 cm in diameter),[4,16] an adequate approach has to provide the possibility for removal of large tumors. In our experience, the smallest intraventricular meningioma encountered was 2.5 cm and the largest 8 cm in diameter, with a mean size of 5 cm. These findings are in line with the findings in previous reports.[4,23] In all cases, a piecemeal resection, usually with ultrasonic aspirators, is mandatory. In a recent publication, Fornari dramatically pointed out that en bloc resection of these lesions may result in fatalities.

Given that intraventricular meningiomas are usually highly vascularized, control and occlusion of the main feeding arteries is desirable during the early stages of surgery. This goal is more difficult to achieve in parietal or parietooccipital approaches than in temporal approaches. It is of particular importance to remove the tumor mass before one turns the tumor gently to the side to identify the choroidal arteries and to coagulate them exactly. Any traction of the tumor carries a risk of bleeding into the choroid fissure, which may be difficult to manage in the early stage of the operation.

After sufficient intracapsular tumor resection, the dissection plane along the tumor's surface has to be developed with maximal care to preserve the eloquent parenchyma of the optic radiation. If the surface is lobulated and the tumor is embedded into the white matter, resection can be extremely difficult and carries the high risk of neurologi-

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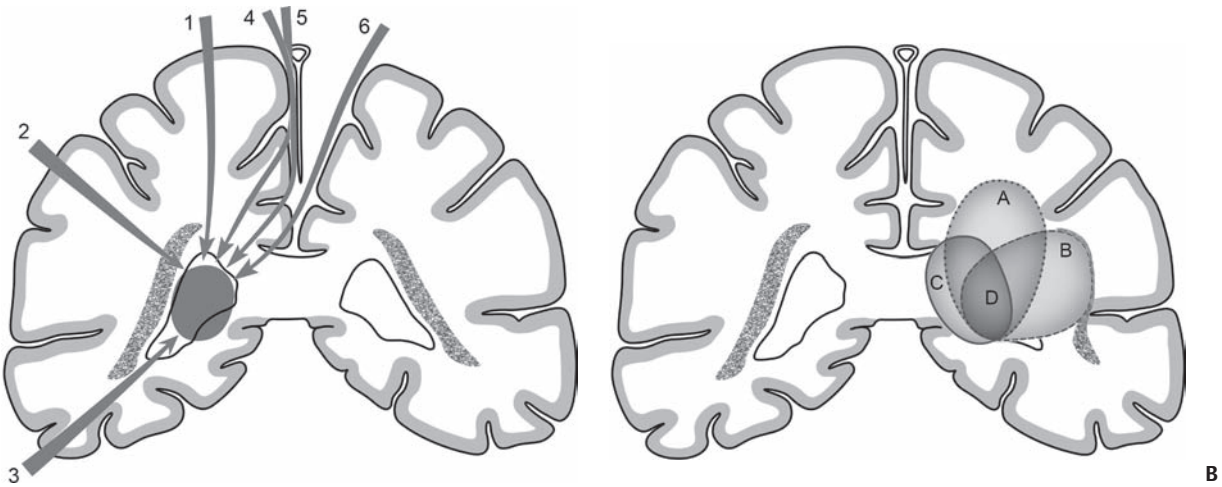


Fig. 32.2 **(A)** Different approaches to meningiomas of the lateral ventricle. Transcortical approaches: (1) parietal or parietooccipital approach, (2) temporal approach (middle temporal gyrus), (3) low temporal approach (T3 gyrus). Transcallosal approaches: (4) ipsilateral transcallosal approach, (5) ipsilateral precuneus approach, (6) contralateral transcallosal approach. **(B)** Approaches according to different types of meningiomas of the lateral ventricle. We can distinguish different tumor types favoring certain approaches: Parietal type (A), which facilitates a parietal or parietooccipital or interhemispheric precuneus approach and for small tumors the transcallosal approach. Temporal type (B) within the trigone offers a short direct approach on the nondominant hemisphere with preexisting visual field defects. In tumors within the inferior horn, a pterional or a low temporal is advantageous. Medial type (C) with tumor growth toward the midline favors a transcallosal or an interhemispheric precuneus approach, or a contralateral transcallosal approach. Central location within the trigone (D) offers many possibilities for resecting the tumor.[1,3–6]

cal deterioration. Because meningiomas tend to be harder than the surrounding tissue, the tumor sometimes has to be fixed by an instrument to minimize its movement during resection. After transsection of feeding arteries and draining veins, the tumor can be dissected safely along the fornix and the remaining part of the choroid plexus.

The approaches to resect intraventricular meningiomas have been divided into transcortical and interhemispheric transcallosal approaches. The transcortical approaches are either parietal, temporal, or frontal, and the interhemispheric transcallosal approaches are parietal approaches.

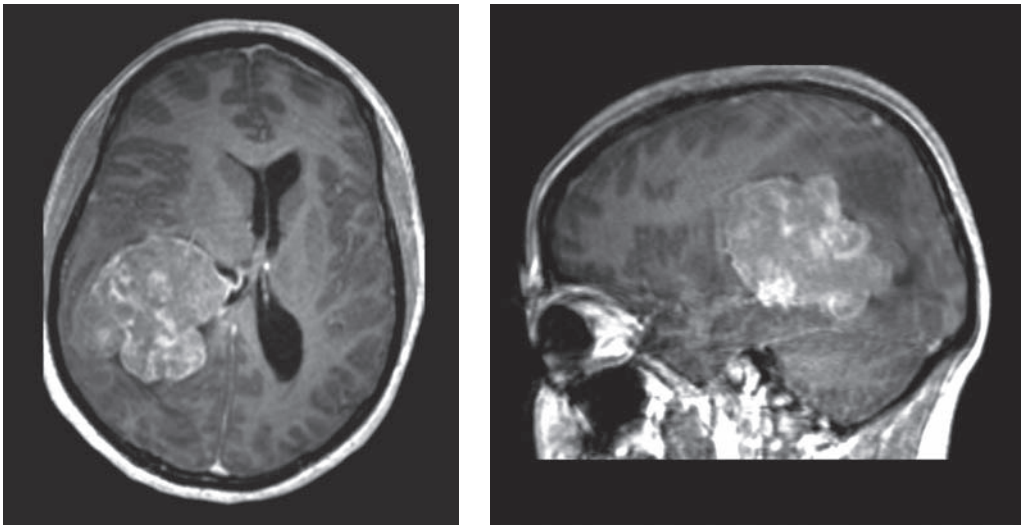
Parietooccipital Transcortical Approach

Parietal and parietooccipital transcortical approaches have been widely used to resect intraventricular meningiomas of the trigone. A parietal or a parietooccipital approach can be done with the patient in supine,[6] lateral, or semisitting positions.[4,33] The preferred positions for Guidetti et al[3] were the prone or supine position, with the corticotomy 2 cm lateral to the midline. Guidetti et al advocated performing the corticotomy on the superior parietal gyrus with further subcortical dissection targeted to the trigone. To avoid injuring sensory fiber tracts, a corticotomy should be at least 1 cm posterior to the postcentral gyrus.[17] Although the corticotomy can be made closer to the parietooccipital junction, this results in the disadvantage of a longer corridor to the trigone and a higher risk of entering the ventricle close to the posterior horn.[48] Parietal approaches with dissection of the deep interparietal sulcus and placement of the corticotomy at the bottom of the sulcus shorten the subcortical dissection considerably but require a slack brain or cortical atrophy, or both (**Fig. 32.3A,B**). Nevertheless, there is a higher risk of injury to the optic radiation with this approach than with the approach through the superior parietal gyrus and a higher risk of causing severe parietal lobe dysfunctions in case of left-sided lesions. Corticotomy lateral to the interparietal sulcus, however, is not recommended. Modern imaging provides exact information on the course of the fiber tracts and their displacement by the tumor. In large meningiomas with displacement of the central gyrus, additional electrophysiological monitoring is helpful. The main disadvantages of the parietal and parietooccipital approaches are the long trajectory through the white matter and the relatively late surgical control of the choroidal arteries, as already discussed. In tumors affecting the nondominant hemisphere, parietal or parietooccipital approaches are good choices for the resection of intraventricular meningiomas, whereas (posterior) temporal approaches are recommended only in cases with preexisting visual field deficits.

Temporal Approach to the Trigone

A temporal approach has been utilized for many years to resect intraventricular meningiomas.[49] The corticotomy is performed high, close to the parietal lobe, to preserve the optic radiation. Further dissection is directed toward the dilated temporal horn and to the lower tumor margin to identify the tumor blood supply. The temporal approach is the most direct and shortest route to the trigone, also facilitating further dissection along the choroid plexus and the fornix.[6] The high risk of postoperative

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A B

Fig. 32.3 (A,B) Intraventricular meningioma (type B) on the nondominant side. The 42-year-old female patient complained of having had headaches for years; she had no signs of increased intracranial pressure, except her papilledema and hemianopia on the left side. Disorientation and cognitive deterioration were present, but not recognized by her relatives. A parietal approach was chosen. During surgery, the most lateral part of the tumor was difficult to dissect from the tapetum, which showed marked perifocal edema. Postoperatively, improvement of cognitive functions was noted, but hemianopia persisted. The tumor could be removed completely and histology showed transitional meningioma without signs of malignancy.

hemianopia and severe speech disturbances following surgery of lesions in the dominant hemisphere are the reasons why many surgeons prefer either posterior parietal approaches or approaches from the midline.

Inferior Temporal Gyrus Approach

There is an alternative temporal approach to reach the lower aspect of the atrium that spares the optic radiation. Recently Nayar et al published two cases of intraventricular meningiomas completely resected using an inferior temporal gyrus approach and preserving visual function.[50] The craniotomy was performed directly at the bottom of the temporal fossa above the ear with a corticotomy at the inferior temporal gyrus immediately above the temporal floor. It is important that the subsequent subcortical dissection remain below the inferior temporal sulcus to reach the ventricle. During this dissection one has to stay below the optic radiation and elevation of the roof of the ventricle is restricted to a minimum. With these limitations in mind, this inferior temporal approach is a good option for tumors originating at the lower part of the trigone. The subcortical corridor is short, and the vascularization is detected early on at the choroid fissure.

Pterional Approach

In the rare situation of an intraventricular meningioma within the temporal horn, a frontotemporal approach is the best strategy. A typical pterional approach, as advocated by Yaşargil et al,[14,33] with extensive splitting of the sylvian fissure, provides enough space for dissecting the tumor. It also allows early identification of the blood supply along the choroidal fissure. The anterior choroidal artery, the optic tract, and the vein of Rosenthal can be safely dissected during tumor removal. In our series, we had only one intraventricular meningioma arising from the tip of the temporal horn. With a pterional approach it was possible to remove the tumor uneventfully without postoperative neurological deficits (**Fig. 32.4A,B**).

Transfrontal Approach

The anterior portion of the lateral ventricle is the least frequent site of intraventricular meningiomas. These meningiomas occupy the lateral ventricle close to the foramen of Monro and can occupy the anterior horn completely.[4] With large tumors in this area, a classical frontal transcortical approach is the approach of choice. In smaller tumors with occlusion of the foramen of Monro, one can consider a transcallosal approach as well.

Interhemispheric—Transcallosal Approach

The transcallosal approach to intraventricular meningiomas was initially described by Kempe and Blaylock in 1976 with the intention of avoiding the disadvantages of a transcortical route.[32] The transsection of commissural fibers in the posterior part of the corpus callosum seems to be better tolerated than temporal or parietal transcortical approaches. In this approach, it is important to preserve the fibers of the splenium to avoid a disconnection syndrome, as has

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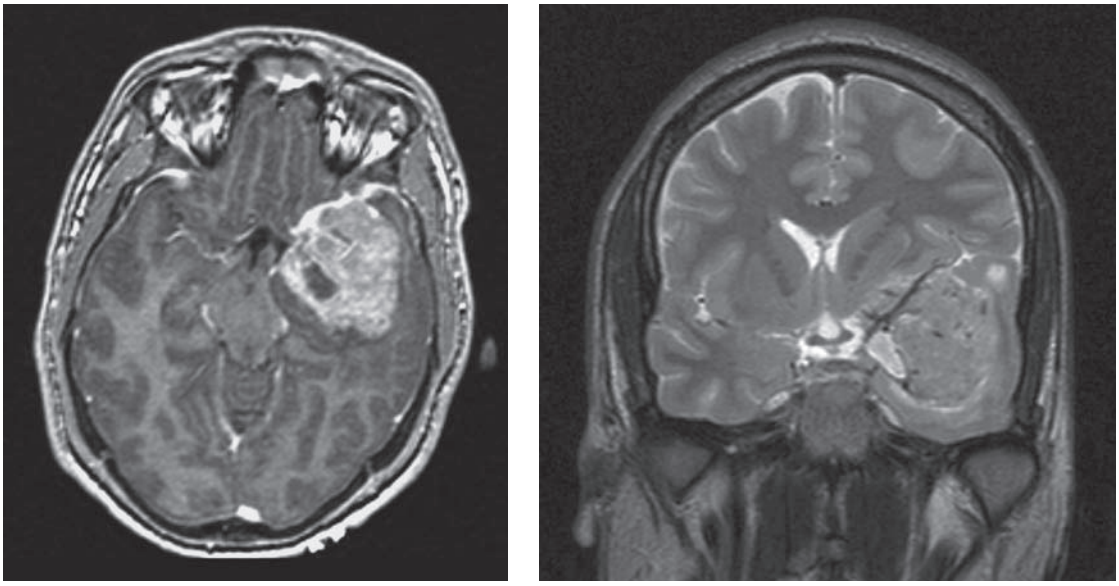


Fig. 32.4 **(A,B)** The 28-year-old male patient had a single epileptic seizure that led to the diagnosis. Magnetic resonance imaging was suspicious for ganglioglioma or tentorial meningioma. During a left pterional approach, there was no contact of the tumor to the dura. The tumor removal was uneventful, and the histology showed a fibrous type of meningioma. Postoperatively no neurological defects were found.

been reported by Levin and Rose and others.[17,32,33,51] Similarly, Rhoton, after extensive anatomical investigations, has described an interhemispheric approach to the trigone.[30] He advocates the transection of the posterior cingulum together with the most posterior part of the corpus callosum to enter the trigone of the ventricle medially. Tumor growth toward the corpus callosum facilitates all interhemispheric approaches (**Fig. 32.5**). To reach tumors of the posterior part of the third ventricle and the trigone, Yaşargil et al described another variant of an interhemispheric approach: the parietooccipital interhemispheric approach.[14,33] In this approach, the cortical incision is performed at the precuneus to reach the medial aspect of the ventricular tumor. Yaşargil pointed out that the parietooccipital vein has to be handled with caution, not to risk venous infarction.[52] This approach, like all interhemispheric approaches, does not compromise the temporoparietal association cortex and has the least risk of injury to the optic radiation.

In interhemispheric approaches, contralateral approaches may be of significant advantage. For (small) tumors, McDermott described a very elegant contralateral transcallosal approach from the nondominant side to resect the tumor in the dominant side.[6] However, it may be necessary to transect the lower part of the falx to have an optimal trajectory in these cases. The distance to the tumor is longer than in ipsilateral approaches, but the angle to reach the trigone is advantageous. The size of the tumor is a critical factor in considering this variant of interhemispheric approaches. Patients may be in a lateral position with the tumor side up, thereby reducing the need for brain retraction as gravity assists; sometimes retractors are not necessary at all.[6,53] For this unorthodox positioning, like in any surgical approach that is less frequently used, neuronavigation is often very helpful.

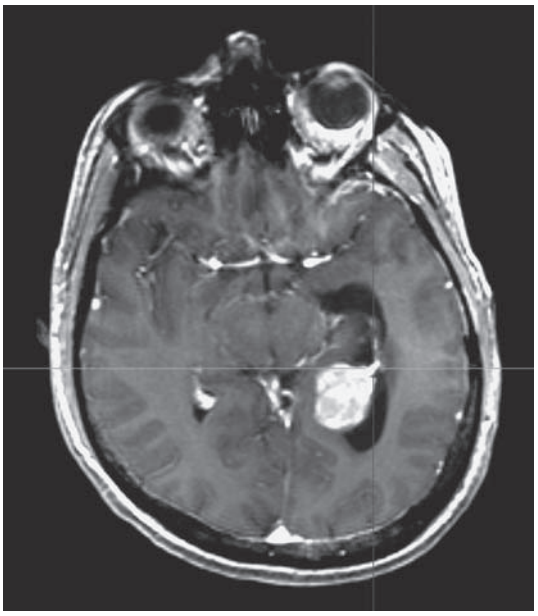


Fig. 32.5 Magnetic resonance imaging shows an incidental finding in a 37-year-old male harboring the smallest tumor in our series (2 cm). There is enlargement of the left temporal horn. This more medially localized tumor (type C) was resected uneventfully by an interhemispheric transcallosal approach (see also **Fig. 32.2A,B**).

Radiosurgery

Radiosurgery has proven to be effective in meningiomas, with a high rate of tumor control and a low morbidity in areas along the skull base. Long-term follow-up has reported excellent results.[8,9,54] Radiosurgery theoretically offers an ideal approach for intraventricular meningio-

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Table 32.4 Results of Intraventricular Meningiomas Compared in the Literature

	Knosp 2010	Liu 2006	Nakamura 2003	Guidetti 1991	Criscuolo 1986	Fornari 1981	Kim* 2009
Cases	20	25	16	20	10	18	9
Temporal approach	1	–	–	2	+		*
Parietal/parietooccipital	15	20	11	18	+	16	*
Transcallosal	1	2	3	No	No		*
Other approaches	2	2					
Posterior fossa	1	1					
Completeness	18	21	15		8	18	
Morbidity	2 HO, 1 HP	2 HO	2 HO		2	–	No
Mortality	1 (5%)		0%	0%	0%	4 (22%)	0%
Benign histology	15		15	20	9	18	8
Atypical/malignant	3	1	1	0	1	–	1
Recurrence	No	2	–	–	1	No	3

Abbreviations: HP, hemiparesis; HO, hemianopia.
* Radiosurgical study.
\+ No numbers given.

mas of the lateral ventricle because transcerebral dissection is unnecessary. The drawbacks of radiosurgery are the radiotoxicity and the size limit, which is a considerable problem, as most intraventricular meningiomas are larger than 30 mm in diameter.

Recently Kim et al[10] published their results with nine intraventricular meningiomas treated by gamma knife radiosurgery. The median volume of these meningiomas was 5.2 cc and the median margin dose 16 Gy. A third of the tumors recurred within the mean follow-up of 5 years (**Table 32.4**).

Small residuals or recurrences of intraventricular meningiomas may represent an indication for radiosurgery. In incidental meningiomas, however, use of radiosurgery should be cautioned.

Histology

There is no difference in the histology of intraventricular meningiomas when compared with those arising from dural attachments. The vast majority of intraventricular meningiomas are benign, although atypical and malignant meningiomas have been reported. In cases of malignant meningiomas, hematogenous metastases as well as seeding into the CSF compartment have been encountered.[55,56]

The tumors operated at our institution[16] were histologically classified as follows: meningothelial meningioma (8/16), transitional (mixed) meningioma (4/16), and lymphoplasmacyte-rich meningioma (1/16) according to the World Health Organization (WHO) criteria. The remaining three meningiomas (19%) were classified as atypical. We have not yet encountered malignant meningiomas in our limited series.

Reports describing growth rate measurements of intraventricular meningiomas are rare.[1,16,17] In all meningiomas operated at our institution, the growth rates were measured using the MIB-1 antigen, ranging from 1 to 11% in WHO grade I meningiomas. In the atypical meningiomas, the labeling indices were significantly higher, 13%, 24%, and 40%. Our findings are in accordance with the current literature on meningiomas in general, although the majority of institutional series do not report atypical or malignant intraventricular meningiomas.

Results

Mortality as well as morbidity after resection of intraventricular meningiomas have been dramatically decreased over the last decades. Before the microscopic era, the mortality rates had been reported to be 0 to 42%,[17] whereas recent publications have very low or no mortality at all.[4,13,16]

This improvement has been possible due to advances in diagnosis with CT and MRI, surgical improvement due to microscopic technique, and, in particular, the use of ultrasonic aspirators, which allow piecemeal resection. Minimally invasive techniques together with neuronavi-

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gation are the most recent development that helped to reduce morbidity (**Fig. 32.2A,B**).

The reduction of surgical mortality associated with the treatment of intraventricular tumors may reflect technological improvement, whereas decreased morbidity may be related to the selection of the approach. Over the years, the temporal approaches, although more direct and of shorter distance to the atrium, have been abandoned in favor of parietal or parietooccipital approaches. These approaches have been favored and used in 75 to 90% of all reported resections of intraventricular meningiomas during the last years, whereas transcallosal approaches have been used in 5 to 18% only (**Table 32.4**). As a consequence of more precise surgical planning abilities, it is likely that interhemispheric, transcallosal, and even contralateral approaches will be applied more frequently in the future.

For patients with intraventricular meningiomas today, neurosurgery can offer complete resection in almost all cases, with very low morbidity and no mortality. In all cases with intracranial hypertension, the symptoms can be cured and cognitive disorders reversed in a high percentage of cases. There is also a high rate (> 80%) of improvement of motor deficits or gait problems, but a 5% rate of new motor function deficits remains.

Visual field defects have been reported to be present in 15 to 67% of patients preoperatively (**Table 32.1**). Improvement of these symptoms is found in 4 to 57% of patients following surgery. Improvement is possible if the optic radiation was compressed but not disrupted. In cases in which the tumor was difficult to detach from the ependyma and the tapetum, preservation of vision is unlikely and may even be worsened. New visual field deficits are reported in 5 to 20%,[4,6,13,16] and these new deficits rarely improve at follow-up.

Hemiparesis is improved in 62 to 100% of all cases, and new deficits after tumor resection occur in 5 to 10%,[3,4,6,13,16] but in contrast to the optic radiation, the surgical compromise of the pyramidal tracts seems to be better tolerated, or rehabilitation is more effective, or both.

The rate of epileptic seizures as a symptom of intraventricular meningiomas is reported to be in the range of 7 to 29% (**Table 32.1**). The incidence of new epileptic seizures after surgery is significantly lower in transcallosal approaches (10%) compared with 20 to 70% in transcortical approaches.[23]

In the Neurosurgical Department of the Medical University of Vienna, we have operated on 20 patients with intraventricular meningiomas between 1980 and 2009 and have published these previously.[16] The median size of the tumors was 50 mm, ranging from 25 to 80 mm. Only the last two cases, which were incidental findings, were of smaller size (20 mm). The preoperative symptoms are shown in **Table 32.1**.

Our results are in keeping with recent published reports and show that the vast majority of our patients improved neurologically and neuropsychologically. The improvement of motor deficits was achieved in 83%, improvement of visual field deficits in 60%. The rate of epilepsy was 7% preoperatively and did not change postoperatively. New neurological deficits for corticospinal tracts were found in one patient (5%), and for optic radiation in two patients (10%). Parietal lobe symptoms, such as dysphasia, dyslexia, dysgraphia, and dyscalculia, were improved in all our cases. The single mortality occurred in the 1980s due to a massive postoperative hemorrhage.

Residual tumor or recurrence is rare, although in most cases the tumors are large and resection is difficult and challenging. Total removal of intraventricular meningiomas can be achieved in 80 to 100% of all tumors. Reoperation for residual disease is rarely reported,[1,6] and regrowth occurs mainly in atypical or malignant meningiomas.

Treatment of tumor residual or recurrence will depend on the size, the location, and the growth of the tumor, as well as on symptoms and the age of the patient. Treatment options include reoperation, fractionated radiotherapy, and radiosurgery. The results of radiosurgery in intraventricular meningiomas, however, are not convincing,[6,10] and radiation toxicity and tumor regrowth are reported after radiosurgery and radiotherapy treatment. We agree with McDermott that radiosurgery for intraventricular meningiomas should be reserved only for patents who are not candidates for surgery because of medical comorbidity.

A review of the literature and our own data[1,3,4,10,13,16,17] shows that the majority of intraventricular meningiomas arise within the left ventricle (62%), and we expect that future investigation will focus more on refined neuropsychological testing than has previously been done. These results will help guide our quest to find the best and the least traumatic approach to the atrium of the lateral ventricle.

Meningiomas of the Fourth Ventricle

Meningiomas of the fourth ventricle are one of the rarest pathologies within the posterior fossa, and they represent only 5% of all ventricular meningiomas. The differential diagnosis of intraventricular meningiomas of the fourth ventricle mainly includes metastasis and choroid plexus papillomas.

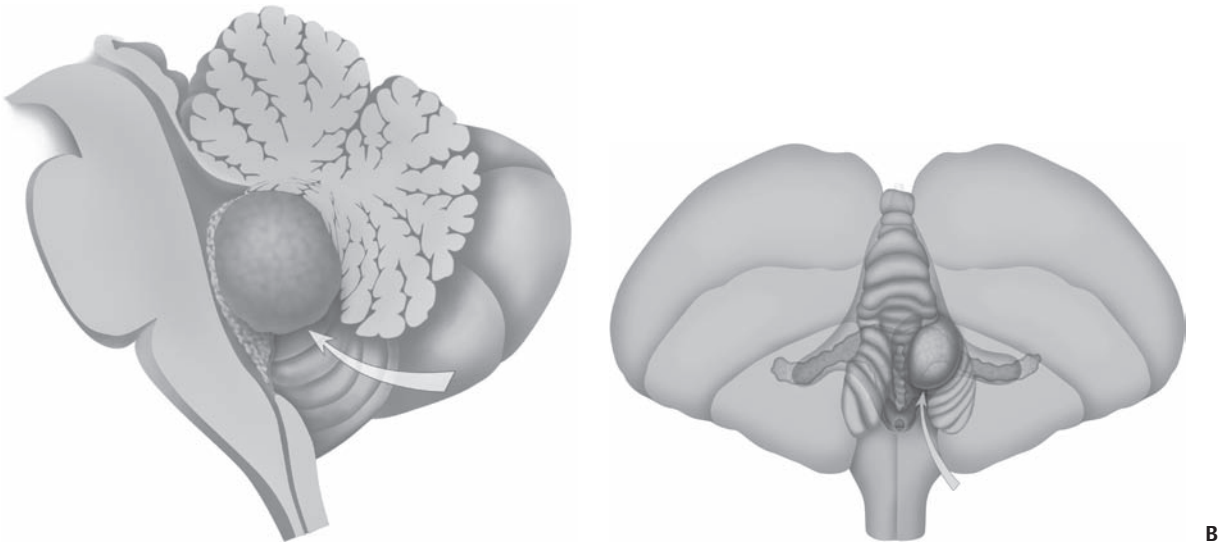
Symptoms of extraaxial pathologies of the fourth ventricle often begin with raised intracranial pressure due to CSF occlusion and hydrocephalus. Only in large tumors are symptoms like vertigo, double vision, or long tract signs present.

The diagnosis of a contrast-enhancing tumor within the fourth ventricle can be established by CT, although MRI is considered the best diagnostic tool for pathologies in the posterior fossa.

The plexus forms the posterior part of the fastigium of the fourth ventricle and reaches through the lateral recesses through the Luschka foramen (**Fig. 32.6A,B**). Meningiomas can arise from all parts of the plexus and the tela choroidea, but the majority of tumors develop close to the midline within the ventricle. Paramedian localization of intraventricular meningiomas is reported exceptionally.[48] The floor of the fourth ventricle is compressed by large meningiomas, but brain stem edema is rare.

Vertebral angiograms show that the choroidal arteries, originating at the choroidal point[33] of the posterior infe-

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A B

Fig. 32.6 (A,B) Intraventricular meningioma of the fourth ventricle. A tonsillouveal approach offers an excellent approach for meningiomas within the fourth ventricle without splitting the vermis.

rior cerebellar artery (PICA), provide the blood supply of these meningiomas. Digital angiography, however, is not performed routinely because MRI provides enough information about vascularity in these tumors.

Surgery

In cases with acute hydrocephalus, initial restoration of CSF pathways with an external ventricular drain or endoscopic third ventriculostomy is necessary before further diagnostic investigations or surgery of the posterior fossa are performed.

Tumors of the fourth ventricle are approached by a limited median suboccipital approach either in a sitting or in a prone position. A tailored craniotomy according to the size and localization includes the opening of the foramen magnum, whereas resection of the lamina of C1 is not necessary. A Y-shaped dural opening enables wide dissection of the Magendie foramen. Usually the vermis covers the tumor and is displaced and compressed. The tumor may be visible within the cistern between the tonsils. In midline tumors, the dissection starts within the Magendie foramen beneath the tonsils to identify the PICA on both sides. Although it is tempting to split the vermis to gain space for tumor removal, further dissection should be performed along the telouveal fissure on one side. Yaşargil[33] and Rhoton[57,58] advocate this tonsillouveal approach, which is performed within the fissure between the tonsil and the PICA on the lateral side and the vermis on the medial side. This tonsillouveal approach avoids splitting the vermis and reduces the risk of cerebellar mutism.[59]

Further dissection leads directly to the tela choroidea and the inferior medullary velum, which have to be transsected. After coagulation and transsection of the choroidal arteries, the whole fourth ventricle is widely open. Dissection along a cleavage plane between the tumor, the tela choroidea, and the cerebellum allows stepwise resection of the tumor. As in surgery of meningiomas elsewhere, the key is early devascularization and piecemeal resection. Sometimes the lateral vein of the fourth ventricle, which disappears through the Luschka foramen, is difficult to manage.

The floor of the fourth ventricle may be compressed by the tumor and needs meticulous dissection without compression, but in small or moderate-sized meningiomas, the dissection is feasible.

Conclusion

The management of intraventricular meningiomas is challenging in many ways. The tumors can reach considerable size before becoming symptomatic and usually grow in delicate areas. Treatment is complex even in small and asymptomatic intraventricular meningiomas (**Fig. 32.2A,B**). Despite the extraordinary tumor sizes described in recent studies, the majority of cases could be resected completely. Reports on recurrences of WHO grade I intraventricular meningiomas are extremely rare. One of the problems with intraventricular meningiomas is their low incidence. Most institutional series including more than 10 cases span more than a decade or longer. This makes it very difficult to compare results even within the same series. We were able to collect 20 cases operated between 1980 and 2009, covering the CT and MRI era. Diagnostic techniques as well as surgical abilities changed considerably within this time, and the results presented should therefore be viewed within their time

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frame. A growing body of knowledge, including data by Delandsheer, Criscuolo and Symon, Guidetti and Delfini, and Nakamura, offers sufficient evidence that intraventricular meningiomas behave similarly to meningiomas elsewhere in the central nervous system. The difficulty with intraventricular meningiomas arises from the rarity of these lesions, the very delicate neurological area surrounding the lesions, and the very complicated anatomical space within which one needs to work.

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Chapter 33

Meningiomas of the Third Ventricle and Pineal Region

Jason A. Ellis, Gaetan Moise, and Jeffrey N. Bruce

Introduction

Meningiomas of the third ventricle represent a distinct and rare anatomical subtype of meningiomas. They are not dural based and are thought to arise from arachnoid cap cells within the velum interpositum in the roof of the third ventricle. Third ventricle meningiomas may be further segregated by location into anterior and posterior third ventricular, with pineal region meningiomas included in the posterior group. Posterior third ventricle meningiomas were called posterior tumors of the velum by Cushing and Eisenhardt in their classic text.[1] This anatomical segregation is useful both as a way to understand the divergent clinical presentations of these two categories of tumors and also in choosing among the various surgical approaches for access to the lesion. It is worth noting that meningiomas arising from the base of the third ventricle have been described.[2] However, they are extremely rare and probably do not arise from the velum interpositum.

Before the advent of more sophisticated neuroimaging technology, several authors grouped falcotentorial meningiomas of the pineal region along with posterior third ventricle and pineal region meningiomas. However, it is now generally appreciated that these dural-based falcotentorial tumors are not true meningiomas of the third ventricle because they do not arise from the velum interpositum. Following the suggestion of Stein,[3] we prefer to limit our definition of posterior third ventricle and pineal region meningiomas to only those that arise from the velum interpositum.

Third ventricle meningiomas present a variety of diagnostic and therapeutic challenges for the treating neurosurgeon. Acute presentations secondary to obstruction within the ventricular system, atypical imaging features, and technical hurdles that make stereotactic biopsy of targets in the third ventricle and pineal region challenging render third ventricle meningiomas among the most difficult to manage. The development of modern neuroimaging and microneurosurgical techniques facilitates definitive surgical treatment, with a goal of gross total resection and low attendant morbidity. This chapter reviews the epidemiology, structural etiology, surgical anatomy, clinical and imaging features, and surgical approaches for resection of third ventricle meningiomas.

Epidemiology

Meningiomas of the third ventricle represent 0.15 to 0.18% of intracranial tumors and 6 to 15% of intraventricular meningiomas.[4–6] Although the exact number remains in question, very few cases of third ventricular meningiomas have been reported.[2, 7–10] Indeed, it is likely that only a subset of reported cases arise from the velum interpositum and are thus, by definition, of third ventricular origin. A literature review by Renfro et al[9] revealed 47 cases of third ventricular meningiomas since 1913. Of these, only 29 cases were truly thought to originate within the third ventricle based on their anatomical isolation to this region. Lozier and Bruce[2] found 27 histologically verified velum interpositum meningiomas of the posterior third ventricle and pineal region, only 17 of which had sufficient clinical information for analysis. Although extraventricular meningiomas primarily present in adults, with a higher incidence in women, meningiomas of the third ventricle may not strictly follow this trend. Third ventricle meningiomas may occur slightly more frequently in men and are seen relatively frequently in children. Renfro et al[9] found 12 reports of pediatric anterior third ventricle meningiomas among 29 cases reviewed from the literature. Within this pediatric cohort there was a slight male preponderance, with eight reports in boys and five in girls. This is in comparison with an ear-

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lier study by Cabezudo et al[4] that did not reveal a sex predilection. Similarly Lozier and Bruce[2] found four cases of pediatric velum interpositum meningiomas among their 17 reviewed cases. They too observed an increased incidence in males (11:6).

Developmental Considerations

The neural elements forming the structural framework for the roof of the third ventricle (see section on surgical anatomy) are lined by a double layer of tela choroidea called the velum interpositum. It can be thought of as a soft tissue partition between the diencephalon and the telencephalon.[11] Embryologically, the third ventricle roof is formed by a single layer of tela choroidea.[12] Formation of the velum interpositum begins as the fornix and corpus callosum extend posteriorly, causing the attached tela choroidea to reflect upon itself as it is pulled along. Anteriorly, these two layers become closely apposed, forming a potential space. Posteriorly, the layers separate with the inferior (ventral) layer coursing along the posterior wall of the third ventricle while the superior (dorsal) layer becomes adherent to the fornices and splenium of the corpus callosum. The potential space between this double layer may separate to form a velum interpositum cistern or cavum velum interpositum[13] that directly communicates with the quadrigeminal cistern.

Surgical Anatomy

Understanding the anatomy of the third ventricle and its relation to surrounding structures allows for an appreciation of the structural basis for third ventricle meningioma development, which helps guide the planning and implementation of appropriate surgical approaches. The midline, slitlike, ependyma-lined third ventricle has a superior and an inferior part delineated by the hypothalamic sulcus. Meningiomas of the third ventricle are generally limited to the superior part between the paired thalami. The anatomy of the third ventricle is commonly described in terms of the structures forming its roof, anterior wall, floor, posterior wall, and lateral walls. The structural relations of the third ventricle are likewise delineated within this schema. A detailed description of the surgical anatomy of the third ventricle can be found elsewhere[14]; however, a concise review is presented here.

Roof

The roof of the third ventricle is formed by the ventral surfaces of the body and crura of the fornix as well as that of the rostral hippocampal commissure. It extends from the interventricular foramen of Monro anteriorly to the suprapineal recess posteriorly. The neural structures forming the roof are lined by the velum interpositum. An intervening vascular complex composed of the paired posterior medial choroidal arteries, internal cerebral veins, and their respective branches/tributaries resides between the ventral and dorsal layers of the velum interpositum. The lateral margin of the roof is delineated by the choroidal fissure, the narrow cleft along the junction of the fornix, and the thalamus, through which the double-layered tela choroidea burrows to form choroid plexus within the lateral ventricles.

Anterior Wall

The anterior wall of the third ventricle extends from the interventricular foramen of Monro rostrally toward the optic chiasm. A small superior portion of the anterior wall is formed by the anterior commissure, whereas the majority of this wall is formed by the lamina terminalis, the remnant of anterior neuropore closure during development. A small inferior portion of the anterior wall is formed by the dorsal aspect of the optic chiasm.

Floor

The floor of the third ventricle extends from the optic chiasm posteriorly toward the cerebral aqueduct of Sylvius. The anterior portion from the optic chiasm to the mammillary bodies is composed of the tuber cinereum, with the posterior portion formed by the cerebral peduncles and midbrain tegmentum.

Posterior Wall

The posterior wall of the third ventricle extends from the suprapineal recess superiorly toward the cerebral aqueduct of Sylvius inferiorly. It is composed, from superior to inferior, of the suprapineal recess, the habenular commissure, the pineal body and recess, and the posterior commissure to its junction with the tectum. The deep cerebral venous system is intimately related to the posterior wall within the quadrigeminal cistern. The great cerebral vein of Galen lies posterior and dorsal to the pineal body. It is formed from the internal cerebral veins as they emerge from the roof of the third ventricle between the two layers of tela choroidea medially and from the basal veins of Rosenthal laterally. The great cerebral vein of Galen traverses thick arachnoid septa within the quadrigeminal cistern just inferior to the splenium of the corpus callosum, eventually joining the inferior sagittal sinus to create the straight sinus.

Lateral Walls

The lateral walls of the third ventricle are formed by the medial walls of the thalami anterosuperiorly, the habenula posterosuperiorly, and the medial walls of the hypothalamus inferiorly. The presence of an interthalamic adhesion commonly forms the massa intermedia within the superior third ventricle.

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Structural Considerations

Meningiomas of the third ventricle arise from arachnoid cap cells within the velum interpositum and originate from either the ventral or dorsal layer of the tela choroidea. This distinction has great significance for posterior third ventricle and pineal region meningiomas. The anatomical constraints provided by the structures forming the third ventricle roof make this distinction less important for anterior third ventricle meningiomas. Meningiomas arising posteriorly from the ventral tela choroidea displace the internal cerebral veins dorsally and grow caudally toward the floor of the third ventricle. Those arising from the dorsal layer of the tela choroidea displace the internal cerebral veins ventrally and bow into the roof of the third ventricle. Additionally, these meningiomas may lie predominantly in the pineal region (quadrigeminal cistern) and are thought to arise from either the posterior velum along its course on the inferior surface of the splenium of the corpus callosum or along the posterior tenia fornicis.[2,12]

Clinical Presentation

The clinical presentation of third ventricle meningiomas is primarily due to a combination of cerebrospinal fluid (CSF) obstruction and compression of surrounding structures (**Table 33.1**). Symptoms attributable to intracranial hypertension due to obstructive hydrocephalus are very common. Headache, cognitive disturbance, impaired gait, and visual symptoms are frequently seen at presentation. Transient symptoms presumably due to intermittent obstruction of the foramen of Monro have been reported for anterior lesions.[4] Memory deficits and endocrine abnormalities are also more frequent, with anterior lesions likely secondary to forniceal and hypothalamic involvement, respectively.[9] Posterior third ventricle and pineal region masses can present with extraocular movement disturbance and pupillary abnormalities from cranial nerve dysfunction and the Parinaud syndrome as a result of dorsal midbrain compression.

Imaging Characteristics

Meningiomas of the third ventricle may or may not demonstrate the typical features of dural-based meningiomas. Computed tomographic (CT) scans generally demonstrate a well-circumscribed iso- or hyperdense lesion with homogeneous contrast enhancement. As with dural-based meningiomas, calcifications may be evident. On magnetic resonance imaging (MRI), third ventricle meningiomas typically appear hypo- to isointense on T1 sequences, hyperintense on T2 sequences, with strong homogeneous postgadolinium enhancement (**Fig. 33.1**). Because CSF also demonstrates strong T2 signal characteristics, fluid-attenuated inversion recovery (FLAIR) imaging is often useful for delineating ventricular masses. Tekkök et al[15] reported a case of a third ventricle meningioma with minimal signal on T1 sequences and no contrast enhancement, but with easily detectable hyperintensity on FLAIR imaging. Weak, heterogeneous postgadolinium enhancement has also been reported.[2]

Hydrocephalus with dilation isolated to the lateral ventricles is commonly seen with anterior third ventricle meningiomas due to foramen of Monro obstruction. Tectal plate compression causing aqueductal stenosis and triventricular dilation is seen with posterior third ventricle and pineal region meningiomas (**Fig. 33.2**).

Contrast MRI sequences with gadolinium and magnetic resonance venography (MRV) may be useful in delineating the relationship of the tumor to the deep venous system, an important consideration for surgical planning. Whereas meningiomas arising from the dorsal layer of the tela choroidea displace the internal cerebral veins ventrally, those arising from the ventral layer of the tela choroidea displace the internal cerebral veins dorsally. Additionally, MRV supplies information regarding patency of the deep venous system, thus aiding in assessment of the risk of inadvertent damage to these veins.[2]

Cerebral angiography may be useful for further delineating the vascular supply of third ventricle meningiomas. Variable degrees of tumor blush may be observed. Additionally, angiography will aid in the identification of meningiomas suitable for preoperative embolization. However, in most cases embolization will not be possible because velum interpositum meningiomas are generally supplied by the medial posterior choroidal arteries (PChAs), which are not routinely embolized with impunity. On the other hand, Sagoh et al demonstrated the potential utility of preoperative embolization of velum interpositum meningiomas that have feeders from external carotid artery branches.[16]

Opposite displacement of the plexal segment of the medial PChAs and the internal cerebral veins are thought to be characteristic of tumors originating within the velum interpositum.[12] Although this finding is useful if present, it is important to note that meningiomas of the third ventricle may displace both vessels in the same direction.

The differential diagnosis for third ventricular lesions can be segregated anatomically into anterior third ventricle, posterior third ventricle, and pineal region lesions. Meningioma, astrocytoma, choroid glioma, craniopharyngioma, choroid plexus papilloma/carcinoma, and metastasis are the major entities in the anterior third ventricle that can share imaging features. In addition to these lesions, pineal parenchymal tumors and germ cell tumors must be considered in the differential for lesions in the posterior third ventricle and pineal region.

Surgical Management

Meningiomas of the third ventricle and pineal region are definitively managed by microsurgical resection. Medical management with chemotherapy is ineffective and radiotherapy is largely limited to recurrent tumors or those with malignant pathological features. Surgery within the third ventricle and pineal region was historically associated with a relatively high risk of morbidity and mortality; however, the recent refinement of microneurosurgical techniques has reduced this risk significantly. Gross total resection is curative and should be the goal for almost all meningiomas of the third ventricle and pineal region.

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Table 33.1 Cases of Velum Interpositum Meningiomas

Case	Sex	Age (years)	Presenting Symptoms	Duration of Symptoms (months)	Operative Approach	Resection	Postoperative Outcome
1	M	8	Visual disturbance	3	Parietal craniotomy	GTR	Residual visual disturbance
2	F	14	Headache, strabismus	129	Occipital craniotomy	GTR	Left cranial nerve III palsy
3	F	39	Headache, visual disturbance, cranial nerve VI palsy	3	Suboccipital craniotomy	None	Death
4	M	38	Headache, dementia, nausea, vomiting, left hemiparesis	3	Right frontal craniotomy	GTR	Death
5	M	24	Headache, visual disturbance, ataxia, Parinaud syndrome	8	Infratentorial-supracerebellar	GTR	Persistent Parinaud syndrome
6	M	28	Meningismus	NA	Infratentorial-supracerebellar	GTR	No deficits
7	F	40	Headache, visual disturbance, ataxia, seizure	NA	Right parietooccipital	NA	NA
8	F	48	Headache, ataxia	NA	Bifrontal transcallosal	NA	NA
9	M	40	Headache, dementia, left hemiparesis	NA	Right parietooccipital	GTR	Left visual field cut
10	F	59	Headache, ataxia, Parinaud syndrome, left hemiparesis	12	Infratentorial-supracerebellar	STR	No deficits
11	M	58	Headache, ataxia, dementia, left hemiparesis	12	Right occipital craniotomy	GTR	Left visual field cut and hemiparesis
12	M	61	Headache, ataxia, dementia	2	Right occipital-transtentorial	GTR	Dependent
13	F	9	Headache, altered mental status, nausea, vomiting	<1	Transcallosal	GTR	No deficits
14	M	6	Headache, ataxia, nausea, vomiting	12	Bifrontal transcallosal	GTR	NA
15	M	32	Headache, visual disturbance	18	Right occipital-transtentorial	GTR	No change
16	M	30	Headache, ataxia	7	Bilateral occipital-transtentorial	GTR	Left visual field cut
17	M	25	Incidental	0	Infratentorial-supracerebellar	GTR	No deficits
18	F	5	Vomiting, right hemiparesis	12	Right occipital-transtentorial	GTR	No deficits
19	M	1	Incidental	0	Right occipital-transtentorial	GTR	No deficits

Abbreviations: GTR, gross total resection; NA, not available; STR, subtotal resection.
Updated from Lozier AP, Bruce JN Meningiomas of the velum interpositum: surgical considerations. Neurosurg Focus 2003;15:E11.

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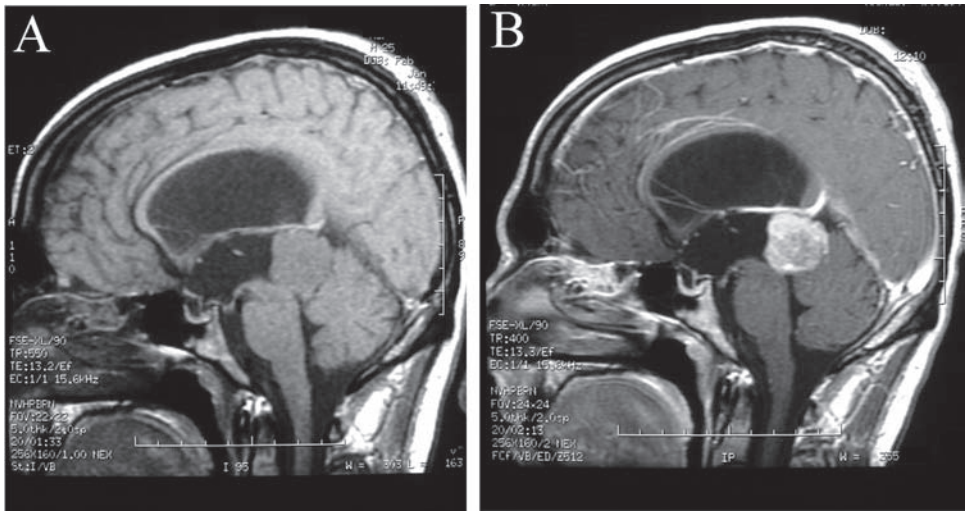


Fig. 33.1 Magnetic resonance imaging (MRI) features of velum interpositum meningiomas. **(A)** Sagittal T1 MRI demonstrates an extraaxial posterior third ventricle/pineal region tumor. There is local mass effect, with midbrain compression and upward displacement of the deep cerebral venous system. **(B)** Strong enhancement postadministration of gadolinium is apparent.



Fig. 33.2 Computed tomographic (CT) demonstration of triventricular dilation from a velum interpositum meningioma. In this precontrast CT it is evident that midbrain compression and the resultant aqueductal stenosis impair cerebrospinal fluid (CSF) outflow from the third ventricle. Areas of calcification seen posteriorly in this image are often present in velum interpositum meningiomas.

Management of Hydrocephalus

Hydrocephalus is frequently encountered in patients with third ventricle meningiomas. Symptomatic hydrocephalus should be addressed emergently, whereas mild asymptomatic hydrocephalus may be managed without preoperative CSF diversion if it is expected that tumor resection will alleviate the obstruction. Placement of an external ventricular drain (EVD) has the dual advantage of palliating acute symptomatic hydrocephalus and providing the surgeon with a means to increase brain relaxation if needed intraoperatively. Endoscopic third ventriculostomy is preferred for symptomatic hydrocephalus secondary to pineal region meningiomas. The long-term complications of ventriculoperitoneal shunting make this a less desirable option for CSF diversion.

Overview of Approaches

The particular approach for meningioma resection is determined both by its size and location as well as by the attendant distortions of normal anatomy by the tumor. In many cases multiple approaches will be equally appropriate, and the approach used should be dictated by the surgeon's preference (**Fig. 33.3**). Excellent reviews detailing surgical approaches to the anterior and posterior third ventricle have been published.[17,18] For anterior third ventricle meningiomas, the anterior transcallosal route is generally preferred.[4,5] Transcortical and subfrontal– or pterional–translamina terminalis approaches also give access to the third ventricle but are less desirable. The extent of brain retraction needed for both the transcortical and the subfrontal approaches poses an increased risk of neural injury. Additionally, the subfrontal approach may violate the frontal sinus and cause damage to the olfactory apparatus. Similarly, the pterional approach is less desirable because of the limited exposure it provides, especially for large meningiomas that extend posteriorly. Access to posterior third ventricle and pineal region meningiomas may be gained by infratentorial–supracerebellar, occipital transtentorial, or posterior transcallosal routes.[2]

Anterior Transcallosal

For those rare instances in which a third ventricular meningioma can only be reached by anterior approaches, a transcallosal route is favored, especially when the lateral ventricles are not enlarged. The patient is positioned supine with the head slightly elevated. A craniotomy over

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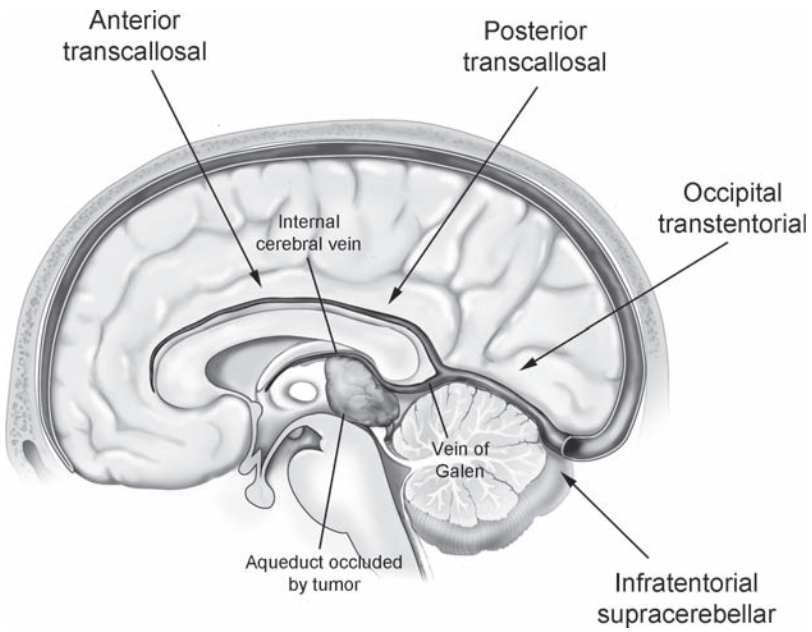


Fig. 33.3 Surgical approaches to the third ventricle. A posterior third ventricular meningioma that creates significant mass effect on the cerebral aqueduct is illustrated. An infratentorial-supracerebellar approach is often used in such cases where the internal cerebral veins course above the tumor. Alternative approaches for resection of third ventricular meningiomas are also shown.

the nondominant hemisphere extending to the midline and centered on the coronal suture is made. The ipsilateral frontal lobe is retracted from the falx with care to protect bridging veins so as to minimize the risk of venous infarction. A 2 to 3 cm midline (interforniceal approach) or paramedian (transchoroidal and subchoroidal approach) incision of the corpus callosum is made. Following the callosotomy, an interforniceal, transchoroidal, or subchoroidal route may be taken to access the third ventricle.[18–20]

Infratentorial-Supracerebellar

The infratentorial-supracerebellar approach to the posterior third ventricle and pineal region is appropriate when the meningioma arises from the ventral layer of the velum interpositum and displaces the internal cerebral veins dorsally. The patient is placed in the sitting position with the neck flexed so as to bring the tentorium horizontal (**Fig. 33.4**). A bilateral suboccipital craniotomy centered just inferior to the torcula is performed. A transverse dural incision through the falx cerebelli with division and ligation of the occipital sinus is made. Gentle retraction of the dural flap and tentorium upward with sacrifice of bridging veins to the superior cerebellar surface is then performed. The precentral cerebellar vein is cauterized and divided, revealing a corridor to the quadrigeminal cistern. Incision of the arachnoid septa within the cistern reveals the tumor capsule. Further details concerning this approach may be found elsewhere.[2,17,21]

Occipital Transtentorial

The occipital transtentorial approach is favored for meningiomas centered in the quadrigeminal cistern that displace the deep venous system inferiorly.[2] A sitting or three-quarter prone position may be used. A right occipital craniotomy is made, exposing the edge of the transverse and sagittal sinuses. The dura is opened and the occipital pole is retracted superolaterally. A paramedian incision of the tentorium is made extending to the free edge, giving exposure to the ambient and quadrigeminal cistern.

Posterior Transcallosal

The posterior transcallosal approach is best suited for meningiomas arising from the dorsal layer of the velum interpositum that displace the internal cerebral veins inferiorly and have significant upward extension toward the corpus callosum. The patient may be positioned in the lateral decubitus or three-quarter prone position. A right parietooccipital craniotomy is made extending just medial to the edge of the sagittal sinus and posterior to the rolandic vein. The dura is incised and reflected medially and the right hemisphere is retracted laterally from the falx. The corpus callosum is identified and a 2 cm midline callosotomy is made, giving access to the posterior third ventricle and pineal region.

Tumor Removal

Third ventricular meningiomas are usually well encapsulated and soft (**Fig. 33.5A**). Tumor removal should proceed in a stepwise fashion, starting with capsule incision, biopsy for frozen section analysis, and internal debulking. Bipolar cautery, cupped forceps, suction, and ultrasonic aspiration are extensively utilized at this stage. Adequate internal decompression is then followed by meticulous dissection of the tumor capsule away from

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Fig. 33.4 Sitting position for the infratentorial-supracerebellar approach to the third ventricle. The neck is slightly flexed and stabilized with three-point cranial fixation anteriorly.

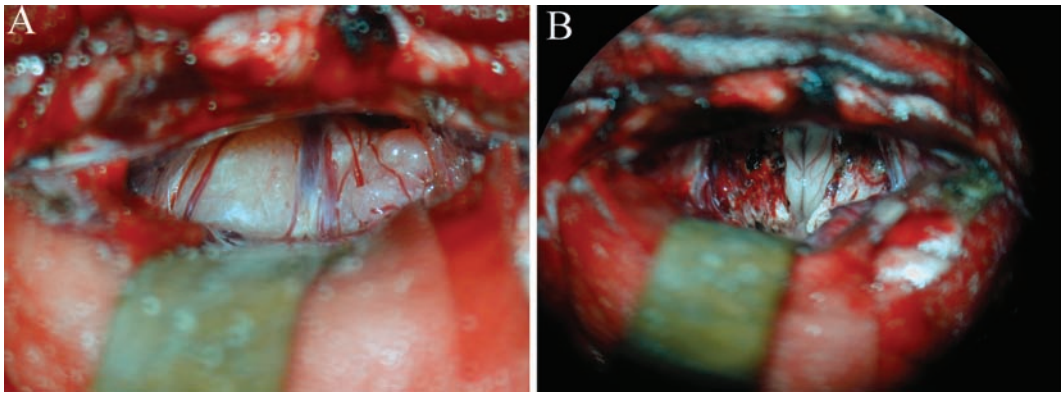


Fig. 33.5 Intraoperative view of a velum interpositum meningioma from an infratentorial-supracerebellar corridor. **(A)** Using gentle downward retraction to pull the cerebellum away from the tentorium, a direct view of tumor within the pineal region is achieved. **(B)** With gross total resection, the quadrigeminal cistern is opened and the posterior third ventricular structures can be seen.

the surrounding brain structures. Capsular wall vessels of choroidal origin may be safely cauterized and divided. Tributaries to the internal cerebral veins, including the thalamostriate, caudate, and septal veins, should be preserved whenever possible to minimize edema, hemorrhage, and infarction risk. Meticulous hemostasis should be maintained with bipolar cautery, rather than extensive use of hemostatic agents with particulate material that may cause aqueductal obstruction or chemical meningitis. Before closure, the resection bed is inspected (**Fig. 33.5B**) for residual tumor, and the patency of the cerebral aqueduct is confirmed with the aid of a flexible mirror. Dura should be reapproximated in a watertight fashion. Craniotomy flaps are replaced in standard fashion with the use of titanium miniplates.

Surgical Outcomes and Complications

Outcomes for third ventricular meningioma surgery have greatly improved due to the advent and refinement of microneurosurgical techniques. An early review of the literature on anterior third ventricle meningiomas by Cabezudo et al[4] revealed favorable surgical outcome in only 29% (4/14) of cases between 1913 and 1980. In this series, permanent morbidity or death was the outcome in 71% (10/14) of treated patients. A more contemporaneous review of velum interpositum meningiomas by Lozier et al[2] found that gross total resection could be achieved in 71% (12/17) of patients, with good outcomes in 59% (10/17). Permanent morbidity and mortality were relatively infrequent, each seen in 12% (2/17) of patients.

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Although the surgical outcomes for third ventricular meningiomas are quite favorable, several postoperative deficits may be encountered, depending on the location of the tumor and the approach used for resection. Brain retraction injuries causing contralateral sensory, motor, or visual field deficits may be seen with interhemispheric or trancortical approaches. Transcallosal approaches have the attendant risk of disconnection syndromes and forniceal injury, which may cause memory deficits. Transient extraocular movement difficulty, including upgaze paralysis and impaired convergence, is frequently encountered following pineal region exploration. Altered mentation postoperatively should alert the clinician to the possibility of cerebral edema, infarction, or hemorrhage that can be seen secondary to the sacrifice of superficial bridging veins or draining veins to the deep venous system. Vigilant postoperative patient checks are imperative for the early recognition and management of these rare but potentially serious perioperative complications.

Treatment of Recurrences

For most third ventricle meningiomas, surgery will be the only necessary treatment modality. When lesions are completely resected, tumor recurrences are rare. Because these tumors have mostly indolent growth curves, even a small residual tumor may have a benign natural history. For those rare tumors that recur and progress, reasonable treatment options include reoperation or radiosurgery.

Summary

Third ventricle and pineal region meningiomas present many technical challenges to surgical treatment due to their location deep within the brain. Modern techniques in imaging and microneurosurgery coupled with a thorough understanding of the region's surgical anatomy generally allow for safe and complete removal of these meningiomas in most cases.

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Chapter 34

Meningiomas of the Middle Fossa Floor

Michael E. Sughrue and Michael W. McDermott

Introduction

The middle cranial fossa is a common location of meningiomas of the cranial base. A large fraction of these meningiomas arise from the dural surfaces bordering the middle fossa, namely the cavernous sinus, sphenoid wing, tentorium, or convexity, and extend into the middle fossa, filling the concavity of the middle fossa floor secondarily. Meningiomas can, however, arise directly from the floor of the middle fossa, with minimal or no connection to the aforementioned border sites.

These tumors have been less well studied, in large part due to a lack of a firm radiographic definition of "middle fossa" meningioma, and lack of awareness of the fact that tumors do occasionally arise from the dura of the middle fossa floor. These tumors likely have been previously classified as sphenoid wing, cavernous sinus, or other meningiomas. We think that a lack of recognition of the middle fossa floor as a potential site of origin predisposes the surgeon to missing an opportunity to remove the tumor at its origin. To date, there are only two small case series specifically addressing this entity, including our own report, which is summarized here, and a 1994 report from Graziani et al.[1]

Definition

We define a middle fossa floor meningioma as a histologically confirmed meningioma with greater than 75% of its radiographic attachment on the floor of the middle fossa, with less than 25% attachment on either the sphenoid wing, cavernous sinus, petrous ridge/tentorium, or lateral convexity dura, which form the four anatomical boundaries of the middle fossa concavity as determined by magnetic resonance imaging (MRI) (**Fig. 34.1**). We further subclassify tumors that radiographically had no attachments to boundaries of the middle fossa (class 1), and those that had between 0 and 25% attachment to the sphenoid wing (class 2), cavernous sinus (class 3), dura over the petrous ridge and tentorium (class 4), or convexity dura (class 5) as shown in **Table 34.1**.

Note that it is possible in this definition for a tumor to be a middle fossa floor meningioma and still have some cavernous sinus invasion if the principal site of origin is the middle fossa floor. Radiographic examples of these meningiomas can be found in **Fig. 34.2**.

Incidence

The exact incidence of middle fossa floor meningiomas is not known, in large part due to a paucity of literature on the topic. Using our own experience as an estimate, between 1991 and 2006, 1213 patients were seen by neurosurgeons at the University of California–San Francisco (UCSF) for meningiomas, of which 1034 patients underwent treatment of their lesion with either open surgery or radiosurgery. A total of 17 patients in this series met our criteria for having a middle fossa floor meningioma. Two of these patients had had previous surgery and were excluded because it was unclear where the initial site of their tumor was located, and it is possible that the middle fossa component could have been missed during the initial resection of a tumor that predominantly arose from the sphenoid wing. Thus we estimate that these lesions represent 1.4% of all known or presumed intracranial meningiomas and 6% of all meningiomas in the middle fossa.

Clinical Presentation

These tumors are often rather large at diagnosis and can present with a wide variety of nonspecific or confusing

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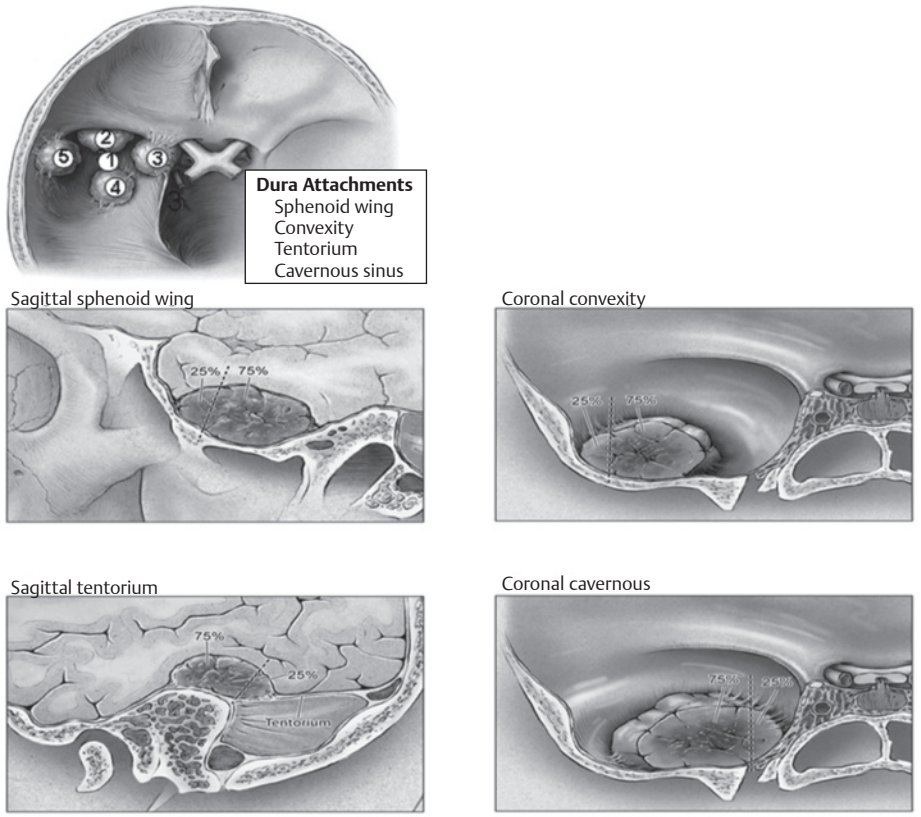


Fig. 34.1 Schematic diagram demonstrating anatomical definition of middle fossa meningiomas. The numbers 1–5 depict the classification scheme for these tumors. Reprinted with permission.

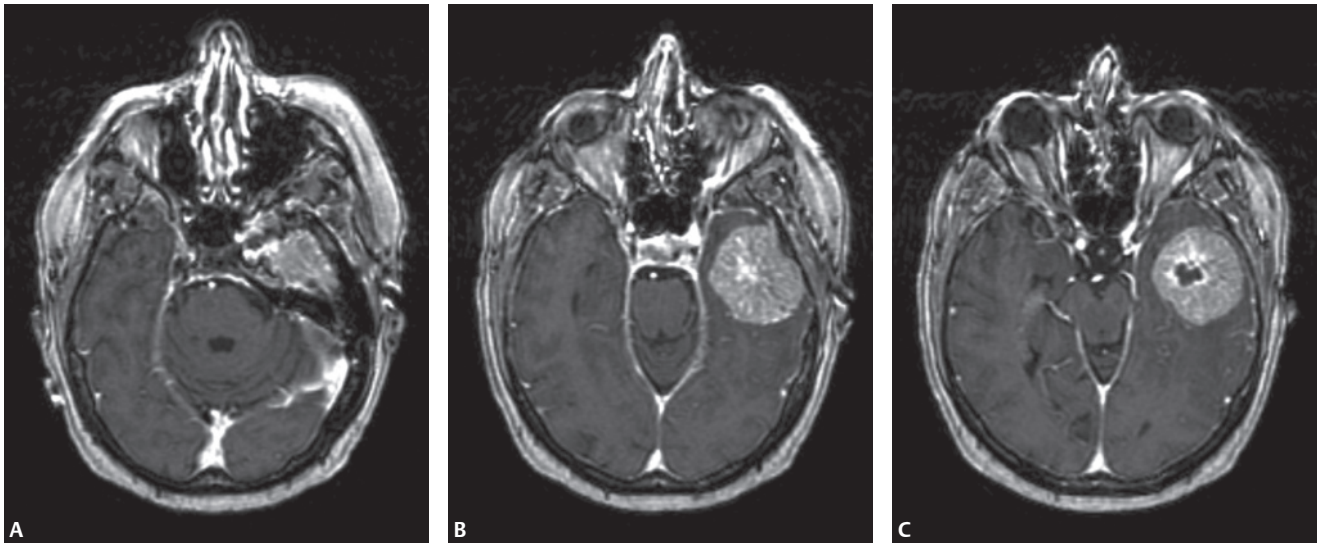


Fig. 34.2 **(A–C)** Axial T1 postgadolium image depicting a class 1 middle fossa floor meningioma.

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Table 34.1 Definition of Different Classes of Middle Fossa Floor Meningiomas

Class	Attachment
1	~100% on middle fossa floor, 0% on adjacent surfaces
2	> 75% on middle fossa floor, 0–24% on sphenoid wing
3	> 75% on middle fossa floor, 0–24% on cavernous sinus
4	> 75% on middle fossa floor, 0–24% on petrous ridge, tentorium
5	> 75% on middle fossa floor, 0–24% on convexity dura

symptoms. The median patient age at time of surgery was 57 years, and the male to female ratio was 6:9. The median volume of these tumors was 21 cc; however, we have seen volumes in excess of 70 cc, with a maximum tumor diameter of 5.5 cm.

Headache was the most common complaint in this series, being a presenting complaint in 60% of these patients. Six of 15 (40%) patients presented with seizures. Not unexpectedly, trigeminal nerve dysfunction (numbness, palsy, or neuralgia) was also common, present in 33% of these patients. Also common were gait disturbance (three patients) and cognitive decline (four patients). Only five patients had no objective neurological deficit at presentation. These symptoms are presented in **Table 34.2**.

Interestingly, hearing loss was a common symptom; five patients demonstrated audiographic evidence of hearing loss on presentation. Other objective symptoms are presented in **Table 34.2**.

Nearly identical to our experience, Graziani et al[1] noted headache in 55% of their patients and cognitive changes in 36%. Their incidence of auditory complaints was also similar to that in our series. They did not report any preoperative trigeminal nerve dysfunction in their series, which differs from our experience, but taken together, trigeminal dysfunction appears to be less common than the anatomical location of these tumors would predict.

Anatomical Tumor Considerations

In our series, eight patients had class 1 tumors, meaning that they arose solely from the floor of the middle cranial fossa. Two patients had class 2 tumors, meaning they had a minor attachment to the sphenoid wing; three patients had a minor degree of cavernous sinus invasion (class 3); and four patients had minor tentorial attachment (class 4). Interestingly, hearing loss was not noted in any of the posteriorly situated tumors. In all 15 cases, intraoperative findings confirmed the radiographic impression that these tumors arose predominantly from the dura of the middle fossa floor, with lesser attachments to surrounding ridges. Two patients had tumors with extension into the infratemporal fossa, and one of these patients additionally had extension into the pterygopalatine fossa.

Ten patients underwent preoperative endovascular embolization. In all cases, the predominant blood supply to these tumors came from the internal maxillary artery, usually via the middle meningeal artery. In one case with infratemporal fossa extension, there was no middle meningeal artery noted on angiography, and the tumor was supplied directly off the internal maxillary artery. One patient's tumor had a small supply from the ascending pharyngeal artery, whereas a minor supply from the anterior choroidal artery was seen in a second.

Operative Technique

We have found that lumbar cerebrospinal fluid (CSF) drainage is usually not necessary when an adequate craniectomy of the squamous temporal bone brings the approach angle flush with the middle fossa floor. Three patients (20%) had a lumbar drain placed at the time of surgery.

Most of these tumors are approached via a standard frontotemporal (pterional) craniotomy or temporal craniotomy. When deemed necessary, a zygomatic arch osteotomy or orbitozygomatic (OZ) osteotomy is added. In general, zygomatic arch osteotomy was used for more posteriorly positioned large tumors (class 5), whereas the OZ osteotomy was used for anteriorly and medially positioned tumors (classes 2 and 3). Suggestions for the surgical approach based on the middle fossa meningioma classifications are summarized in **Table 34.3**. In some cases where the superior pole of the tumor was quite high (> 5 cm from the middle fossa floor) a limited inferior and middle temporal gyrus corticectomy is used to facilitate efficient removal and avoid extensive retraction of the lateral temporal lobe. When possible, the base of the tumor is first detached from middle fossa floor attachments, devascularizing the tumor in the process. Internal debulking was then performed, followed by peripheral dissection. Care should be taken to avoid more than 60 degrees of head rotation to avoid restriction of venous outflow. Classification of the extent of resection was done using the Simpson classification.

Table 34.2 Presenting Symptoms and Preoperative Neurological Deficits of the 15 Patients in This Series

Symptom	Number of Patients	% of Patients
Headache	9/15	60
Seizures	6/15	40
Face pain	5/15	33
Cognitive decline	4/15	27
Gait disturbance	3/15	20
Hearing loss	5/15	33
Diplopia	1/15	7

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Table 34.3 Suggested Approach for Middle Fossa Meningiomas Based on Classification and Size

Class	Size	Position	Approach
1	< 3 cm	Semilateral/ lateral	Orbitozygomatic
	> 3 cm	Semilateral/ lateral	Temporal craniotomy + zygomatic osteotomy
2	< 3 cm	Supine	Pterional
	> 3 cm	Supine/ semilateral	Orbitozygomatic
3	< 3 cm	Supine/ semilateral	Pterional/orbitozygomatic
	> 3 cm	Semilateral	Orbitozygomatic
4	< 3 cm	Semilateral/ lateral	Temporal craniotomy + orbitozygomatic
	> 3 cm	Semilateral/ lateral	Temporal craniotomy + zygomatic osteotomy
5	< 3 cm	Supine	Pterional
	> 3 cm	Supine/ semilateral	Temporal craniotomy + zygomatic osteotomy

Surgical Outcome

Median length of stay for these patients was 6 days (range 3 to 15 days). We were able to achieve a Simpson grade I or II resection in 10/15 patients (67%). Four out of 15 patients (26%) had tumors demonstrating World Health Organization (WHO) grade II histology. Two of these patients had subtotal (Simpson grade IV) resections due to firm adherence to cranial nerves or cavernous sinus invasion. Three of five patients with a Simpson grade III or IV resection had tumors that involved the cavernous sinus. Four of these five patients underwent subsequent radiotherapy.

Despite the general lack of proximity to cranial nerves, these tumors should be viewed with relative caution because the rate of at least one complication in our experience was 33%. We hypothesize that this may be due to the rather large size these tumors reach before diagnosis. There was no early postoperative mortality in our patients. The operative morbidity in this series was clustered in five patients, as 10/15 patients (67%) experienced no operative morbidity (**Table 34.4**). Two patients suffered new neurological deficits postoperatively, and three patients experienced worsening of preexisting neurological deficits. In three cases, these deficits were transient and had resolved by the 6-month follow-up. Significant neurosurgical complications occurred in two of the 15 patients, including CSF leakage, wound infection, and entrapment of an extraocular muscle. This latter complication not surprisingly occurred in one of the five patients who underwent orbitozygomatic osteotomy. One patient suffered a significant medical complication (urosepsis resolving with antibiotics) postoperatively.

To date, we have followed these patients for a median of 5 years (range 1 to 15 years). There have been a total of four known clinical recurrences in this group. Three recurrences were treated with stereotactic radiosurgery, and one patient with a WHO grade II tumor received repeat surgery and external beam radiotherapy. Not surprisingly, all of the patients with recurrence had either higher-grade tumors (two patients), or had a Simpson grade III or higher resection (**Table 34.5**). In contrast, Graziani et al reported no recurrences over a period of 12 to 132 months.[1]

Table 34.4 Clinical Outcome for the 15 Patients in This Series

Pathology	Number of patients
WHO I	11
WHO II	4
WHO III	0
Imaging Results	
GTR	11/15
STR	4/15
Surgical Results	
GTR	
Simpson I	5/15
Simpson II	5/15
Simpson III	1/15
STR	
Simpson IV	4/15
Simpson V	0/15

Abbreviations: GTR, gross total resection; STR, subtotal resection; WHO, World Health Organization.

Table 34.5 Summary of Complication Rates in This Series

Symptom	Number of Patients	% of patients
New neurological deficit	3/15	20
Worse neurological deficit	3/15	20
Medical complications	1/15	7
Subdural hematoma	1/15	7
Cerebrospinal fluid leakage	1/15	7
Wound infection	1/15	7
Ocular muscle entrapment	1/15	7
None	10/15	67

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Conclusion

Meningiomas of the middle fossa floor are a recently recognized entity for which the natural history and outcomes are as yet not well understood. We propose that they can be classified by their degree of tumor attachment to the middle fossa floor and surrounding dura. Although meningiomas of the middle fossa floor are relatively uncommon compared with other meningiomas, it would be a mistake for a surgeon to mentally classify a tumor as a "sphenoid wing" meningioma, when it is truly a class 2 middle fossa meningioma, and to approach the case with the plan of primarily addressing the attachments of the tumor at the sphenoid wing, when greater than 50% of the tumor attachment lies on the floor of the middle fossa. Additionally, given their large size at diagnosis, it is not surprising that the morbidity of resecting these tumors is not trivial, with one third of patients suffering at least one notable postoperative complication. Clearly, investigations are warranted to further understand the clinical behavior of these tumors.

REFERENCES

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VIII

Special Operative Considerations for Intracranial Meningiomas

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Chapter 35

Image-Guided Surgical Techniques for Meningiomas

Robert E. Elliott and John G. Golfinos

The skin opening must be bigger than the bone opening.
The bone opening must be bigger than the dural opening.
The dural opening must be bigger than the tumor.
—Patrick J. Kelly, MD

Introduction

Meningiomas are one of the more common brain tumors. Given their typically benign histopathology, optimal treatment remains complete resection when it can be accomplished with no or minimal morbidity. For difficult tumors with higher-risk profiles, other treatment options include radiosurgery and radiation therapy—with or without surgical debulking.[1–5] Nevertheless, even completely resected benign meningiomas can recur in nearly 10 to 30% of cases with long-term follow-up, and progression of incompletely resected tumors is quite high[6–10] (**Table 35.1**). Incomplete resections predispose patients not only to a high probability of recurrence but also to a decreased chance of successful resection at subsequent operations[7] and worse overall survival.[11] These findings underscore the need for attempted complete removal at the initial operation—including removal of the bulky tumor in addition to the dural tail, sinus invasion, and bony involvement when possible. To better achieve this end safely and in a manner as minimally invasive as possible, image guidance surgery (IGS) systems have become an increasingly important and utilized tool.

Stereotactic neurosurgery was originally conceived for the treatment of intraparenchymal lesions and utilized a headframe-based platform. With engineering advances in imaging, optical, and infrared technologies, "frameless" stereotaxy systems (neuronavigation) were developed[12] and, given their ease of application, have grown in prominence for the treatment of all types of intracranial lesions—including meningiomas.[13] Many centers now routinely use IGS for resection of meningiomas to minimize skin incision and craniotomy size,[8] but few centers have reported on their experience.[14–19] This chapter discusses the techniques of IGS for intracranial meningiomas and their utility beyond minimally invasive neurosurgery.

Rationale for Use of Image Guidance Surgery Systems in Meningioma Resection

IGS systems use the information obtained in a two-dimensional (2-D) plane to re-create and display the patient's anatomy in a three-dimensional (3-D) space. This reconstructed 3-D volume can then be analyzed and manipulated, allowing visualization of the target lesion in multiple planes, planning of the optimal surgical trajectory, and accurate localization of critical nearby structures. All of this can be accomplished before the patient enters the operative suite and can be updated with real-time positional feedback with tracked instruments as the surgery progresses.

To date, however, no prospective, randomized studies have demonstrated improved outcomes using IGS for meningioma resection. Paleologos et al[17] retrospectively reported on 270 patients who underwent meningioma resection (100 with IGS, 170 with standard surgery). Matching 100 patients with similar baseline characteristics (50 in each group), they noted shorter operative times, fewer major complications, shorter hospital stays, and lower costs in the patients in whom IGS was used. In agreement with other centers,[13,15,17,20–23] we believe neuronavigation not only enhances surgeon confidence but can result in improved patient outcomes and satisfaction, with smaller scalp flaps and craniotomy sizes, shorter operative times, more complete resections, less trauma to the surrounding structures, lower surgical morbidity, and shorter hospital stays.

Types and Overview of Image Guidance Surgery Systems

Table 35.2 summarizes the major IGS platforms and the commercially available products. No objective, pro-

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Table 35.1 Simpson Grading Scale for Removal of Meningiomas and Risk of Recurrence or Progression

		Rates of Recurrence or Progression		
Grade	**Degree of Removal[a]**	**Simpson[b]**	**Miraminoff et al[c]**	**Adegbite et al[d]**
I	Macroscopically complete tumor removal with excision of dural attachment and abnormal bone; including sinus resection when involved	9%	32%	14%
II	Macroscopically complete tumor removal with coagulation (Bovie electrocautery or laser) of dural attachment	16%	NA	18%
III	Macroscopically complete tumor removal without resection or coagulation of dural attachment or of its extradural extensions (e.g., hyperostotic bone)	29%	NA	100%
IV	Partial tumor removal	39%	91%	48%
V	Simple decompression (with or without biopsy)	100%	NA	NA

Abbreviation: NA, not available.
[a] Grading scale and definitions adapted from Simpson D. The recurrence of intracranial meningiomas after surgical treatment. J Neurol Neurosurg Psychiatry 1957;20(1):22–39.[14]
[b] Median survival times are not reported.
[c] Outcome data from 225 patients derived from reported actuarial 15-year progression-free survival values.
[d] Outcome data from 114 patients with rates derived from reported 5-year progression-free survival values. The 100% recurrence rate for grade III resection is based on $n = 3$.

Table 35.2 Types of Image Guidance Surgery Systems and Commercially Available Products

Type of Image Guidance Surgery	Description	Products
Articulated arms	Consist of movable arm with multiple position sensors that provide correlation of pointer location with imaging Require movement into and out of operative field for use	ISG Wand (ISG Technologies, Inc., Mississauga, Ontario, Canada/Elekta, Atlanta, GA) Radionics Operating Arm (Radionics, Burlington, MA)
Light-emitting diode systems	Pointing probe and array attached to skull or head holder have light-emitting diodes that emit pulses of infrared light Cameras receive the infrared light and determine the location of the pointer relative to the head array Allow stereotactic microscope integration	iNtellect Cranial Navigation System (Stryker, Kalamazoo, MI) EasyGuide Neuro (Phillips, Shelton, CT) SMN-Zeiss (Carl Zeiss, Inc., Thornwood, NY)
Passive infrared systems	Consist of cameras that emit pulses of infrared light that are returned by reflective spheres attached to the pointer probe, head array, and surgical instruments Allow stereotactic microscope integration	Brainlab VectorVision (Brainlab USA, Redwood City, CA) StealthStation TREON (Medtronic Navigation, Louisville, CO)
Electromagnetic systems	Create a small magnetic field that tracks a magnetically active pointer	Cygnus Stereotactic System (Compass International, Inc., Rochester, MN) StealthStation AxiEM (Medtronic Navigation, Louisville, CO)

spective studies have determined the benefits of using IGS systems compared with not using neuronavigation for meningioma resection nor the superiority of one system over another. Ultimately, the choice of IGS device depends on surgeon preference and institutional availability.

Articulated Arms

Original frameless IGS devices involved articulated arms with multiple electropotentiometer position-sensors within the joints that allowed simple patient-to-image registration. They can be cumbersome to

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move into and out of the operative field—especially during use of the microscope for skull base tumors. Their bulk and frequent collisions with the microscope led to their demise. Nevertheless, accuracy on the order of 2 mm had been reported by multiple authors using these systems—adequate for most meningioma surgeries[20] and rivaling the accuracy of later systems.

Light-Emitting Diode Systems

Light-emitting diode (LED) neuronavigation systems are optically based systems that use an infrared camera array to monitor instrument and head position. The camera tracks the location of LEDs mounted to surgical instruments and to the reference array attached to the head holder or directly onto the skull. Rigid fixation is required with LED systems to maintain the 3-D coordinate space created during registration. These systems require maintenance of line of sight between the camera, head holder array, and probe but allow stereotactic integration with the operating microscope (using the focal point as the "pointer position").

Passive Infrared Sensors

Systems utilizing passive infrared sensors use cameras that flash pulses of infrared light reflected from specially coated spheres attached to a pointing probe or from an array of spheres attached to surgical instruments. Surgical instruments (e.g., endoscopes, ventricular catheters, biopsy probes, etc.) can be registered and calibrated using both device length and diameter to be used during surgery. Importantly, these systems allow a wide array of instruments to be registered and require no cable attachments for ease of use. Similar to LED systems, however, rigid fixation is required, line of sight must be maintained, and the spheres must be cleaned periodically to maintain their reflective properties. Infrared systems also allow stereotactic integration with the operating microscope (using the focal point as the "pointer position").

Electromagnetic Neuronavigation

Electromagnetic IGS systems involve the creation of a small magnetic field enveloping the cranial space and allow tracking of a magnetically active pointing device in the registered field. Magnetic systems avoid line-of-site issues but are prone to magnetic field distortion and disruption of navigation if instruments composed of ferromagnetic materials are brought into the field. Both the Cygnus Stereotactic System (Compass International, Inc., Rochester, MN) used at our center for years and the StealthStation AxiEM (Medtronic Navigation, Louisville, CO) can be used with or without a head holder attachment. The latter has been used successfully in small children where pinning is not advisable.

Intraoperative Magnetic Resonance Imaging

Given the discrete and obvious borders of convexity meningiomas, the utility of intraoperative magnetic resonance imaging (MRI) is unclear.[24] A possible exception may be malignant meningiomas, which can be friable and multifocal, and the completeness of meningioma resection may not be obvious and may require confirmation by MRI. Susceptibility artifact from the bones of the skull base may limit the ability of intraoperative MRI to detect tumor residual in patients with skull base meningiomas. The use of intraoperative MRI is discussed further in Chapter 36.

Ultrasonography

Some authors have used 3-D intraoperative ultrasonography with meningiomas to identify the tumor margins or to aid intracapsular resection of giant meningiomas.[25] However, given the accuracy of current neuronavigation systems, this incremental benefit may be superfluous.

Application of Imaging Guidance Surgery Technologies

Preoperative Imaging Acquisition and Registration

The critical step in the success of IGS is the correlation of high-resolution preoperative imaging data to patient anatomy. Fiducial markers provide reference points on the patient's scalp and obvious on images. These markers usually consist of adhesive stickers filled with a substance that is readily seen on either computed tomographic (CT) or MRI images or both. Newer technologies can use reflective lasers aimed at numerous surface points on the patient's scalp or face and "map" them to the imaging-acquired dataset, obviating the need for fiducial markers (z-touch, Brainlab, Inc., Westchester, IL).

Ideally, fiducial markers should be placed overlying bony or semi-immobile anatomical landmarks like the forehead, mastoid, or parietal boss. Placement in areas of the scalp that are mobile or with a significant amount of soft tissue overlying the bone can lead to inaccurate registration due to fiducial movement during image acquisition and/or registration. For cases involving the parietal, occipital, and suboccipital regions, fiducials should not be placed posterior to the parietal boss. Two sources of spatial inaccuracy are introduced: head flexion during prone positioning (or its variants) and depression of fiducials into skin while the patient is supine on the MRI or CT table. All fiducials should be placed directly over or anterior to the parietal boss without evident loss of accuracy.

The most detailed and accurate imaging modality for meningiomas is MRI. Thin-cut CT (with or without contrast) can be invaluable if hyperostosis or bony involvement is suspected. Image fusion technologies allow for

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both MRI and CT images to be incorporated into the same IGS system and obviate the need for separate fiducial registrations. Modern systems also provide software algorithms to incorporate functional imaging like fMRI and tractography to help identify eloquent cortices (e.g., motor, sensory, language) or functional fibers (e.g., pyramidal tracts) in relation to the tumor.

Patient Positioning and Registration

Once inside the operating room, the next steps are patient positioning and head fixation. Head fixation is performed in the usual fashion when using IGS, with one caveat: placing a skull fixation pin in close proximity to fiducial markers can cause a shift in the position. This should be avoided or those markers should be disregarded during registration. Otherwise, IGS follows the same positioning algorithm as other tumors (placing tumor toward the top of the operative field, etc.). Finally, LED and infrared systems require maintenance of line of sight between the camera, head array, and probe (or instrument). This cannot be overemphasized because the most frequent source of frustration with infrared systems is loss of the sight line to the camera. The IGS goes on holiday while the surgeon becomes increasingly irritated. However, the camera may be moved in relation to the patient both during and after registration to facilitate camera recognition of devices and probes. Registration is then performed using a pointing probe and fiducials or with a laser pointer using facial features.

Intraoperative Planning and Localization

The boundaries of the tumor are then localized on the scalp using axial, sagittal, and coronal images (**Figs. 35.1, 35.2, 35.3, 35.4, 35.5,** and **35.6**). We prefer to excise a minimum 10 mm margin of "normal" dura along the convexity and still allow at least 5 mm of dura from the craniotomy edge to allow for suturing of dural margins at closure. We prefer periosteal autograft, harvested during the opening of the scalp, if at all possible.

Thus craniotomies should ideally be made with a circumferential margin around the tumor of 15 to 20 mm—limited only by boundary structures like the major venous sinuses or to limit the exposure of nearby eloquent tissue. In fact, it could be argued that an unforeseen risk of IGS when used for convexity meningiomas is making the craniotomy too small. If the surgeon is seduced by the technical wizardry and succumbs to the temptation of a tailored craniotomy more suitable for an intraaxial tumor, the unfortunate result can be the incomplete resection of surrounding, diseased dura and an increased risk of recurrence. For skull base tumors, resection of the dural tail is more difficult, and neuronavigation is primarily used to identify the ideal trajectory to tumor during planning and identification of neurovascular structures intraoperatively.[15,22] That said, the skull base surgeon's mastery of anatomy limits the utility of IGS in planning surgical approaches.

In contrast to the resection of intraparenchymal lesions, IGS for meningioma is much less prone to the effects of brain shift as the operation proceeds. For intraaxial tumors, application inaccuracy can arise from registration error as well as tissue displacement (brain shift) due to release of cerebrospinal fluid (CSF) or following removal of tumor tissue.[26] Given their attachments to semifixed structures like the falx or tentorium and rigid structures like the skull base, meningiomas are far less susceptible to inaccuracy introduced by brain shift.[21]

Utility of Image Guidance Surgery for Meningiomas in Various Locations

Convexity and Parasagittal Meningiomas

Although complete resection rates of 90 to 95% are reported in most large series of convexity meningiomas, recurrence occurs in 10 to 25% of patients.[6–8] Borovich and Doron[27] analyzed linear strips of dura oriented radially from the margin of resected meningiomas of 14 patients. They noted macroscopic nodules or microscopic meningothelial cell aggregates as far as 3 cm from the "margin" in all cases. No such rests were found in specimens from 10 patients without meningiomas used as controls. The authors believe these rests of cells are responsible for the not insignificant rate of recurrence after total removal. They proposed a Simpson grade 0 for convexity meningiomas resected with a wide margin of "normal" dura.[28] Kinjo et al[29] reported no recurrences in a series of 45 patients who had what they called a grade 0 resection (tumor removal plus a 2 cm margin of "normal" dura). Their results provide support for the hypothesis that microscopic tumor cells lie within and beyond the imaging-defined dural tail and cause meningioma recurrence. However, only 19 patients had follow-up greater than 5 years, and the recurrence rate of meningiomas has been shown to increase directly with follow-up duration.[6,7]

Neuronavigation provides further benefits beyond tailoring the size and shape of the craniotomy flap to allow for complete resection with "margins." **Table 35.3** summarizes the advantages IGS systems provide in conjunction with convexity and parasagittal meningiomas. The most important ancillary benefits of IGS systems for these tumors are precise identification of eloquent cortices, major sinuses, and draining veins—potentially decreasing the risk of neurological injury.

Skull Base Meningiomas

Skull base meningiomas remain some of the most challenging lesions in neurosurgery, with lower rates of complete resection and higher neurological morbidity compared with convexity tumors.[3,30] However, advances in "skull base" approaches—with an emphasis on bony removal to shorten the working distance to the pathology and to minimize

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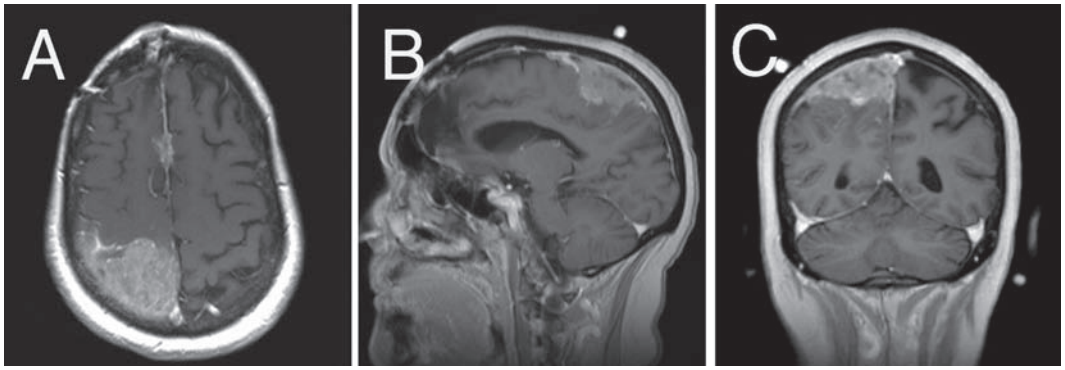


Fig. 35.1 This 62-year-old female diagnosed 18 months before admission with multiple anaplastic meningiomas—one of which was resected and six were treated with radiation therapy. She returned with left-sided arm and leg weakness and intractable simple partial seizures. **(A)** Postoperative axial, **(B)** sagittal, and **(C)** coronal magnetic resonance imaging revealed a new 6 × 6 x 4 cm right parietal meningioma abutting the central sulcus, with invasion of the brain and superior sagittal sinus.

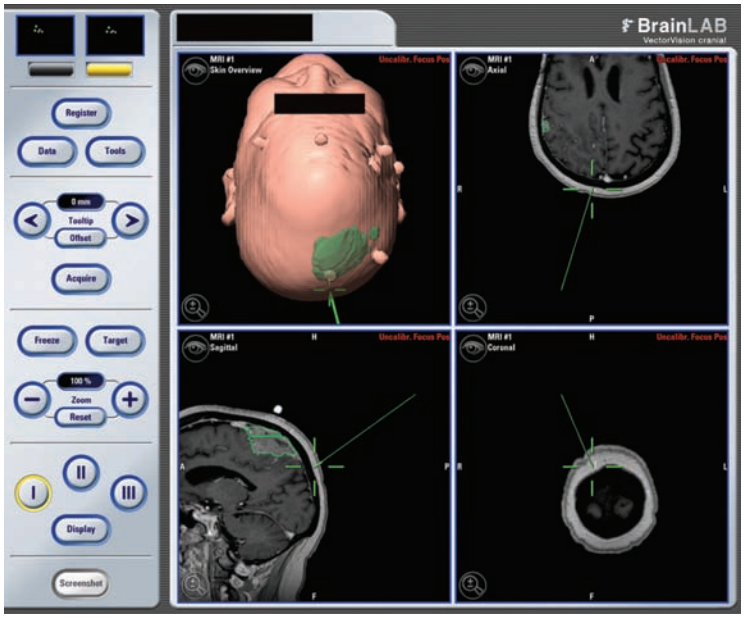


Fig. 35.2 Image of Brainlab screen used to determine the position of the sagittal sinus (coronal view) for planning the medial extent of the scalp incision as well as most anterior limit of this anaplastic meningioma (sagittal view). Image courtesy of Brainlab AG.

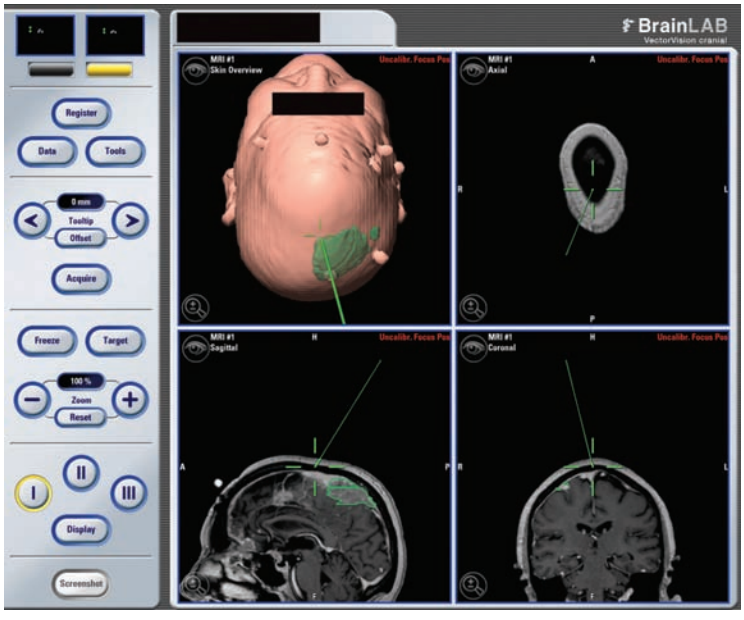


Fig. 35.3 Image of Brainlab screen used to determine the posterior extent of this anaplastic meningioma to determine the posterior extent of scalp incision (sagittal view). Image courtesy of Brainlab AG.

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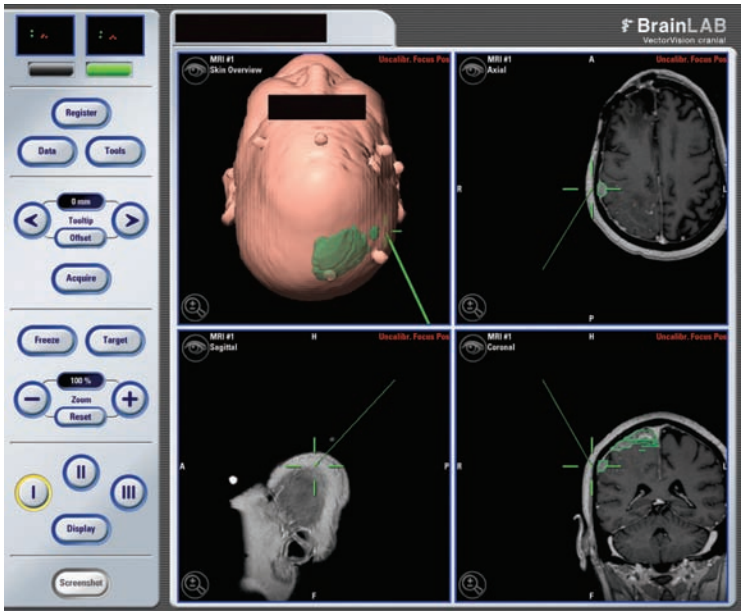


Fig. 35.4 Image of Brainlab screen used to identify the lateral extent of the dominant meningioma and to include the lateral satellite lesion in the flap (coronal and axial views). Image courtesy of Brainlab AG.

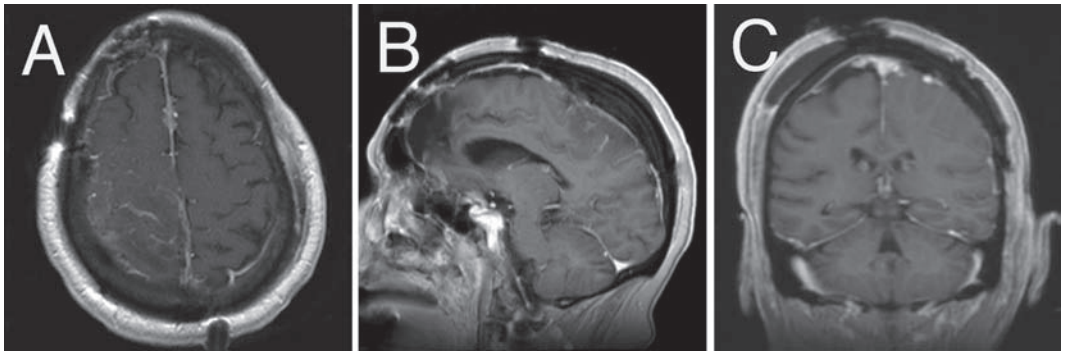


Fig. 35.5 **(A)** Postoperative axial, **(B)** sagittal, and **(C)** coronal magnetic resonance imaging show Simpson grade III resection with a small amount of residual tumor filling and partially occluding the superior sagittal sinus.

Table 35.3 Utility of Image Guidance Surgery Systems for Meningiomas of the Convexity and Parasagittal Regions

Advantage	Benefit to Surgeon/Patient
Identify the margins of the tumor and the dural tail	Ensure maximal extent of resection Decrease risk of recurrence, need for further treatment Create dural opening that limits the amount of uninvolved, normal brain
Plan scalp incision	Minimize the extent of tissue trauma without sacrificing completeness of tumor removal Decrease operative time
Plan size and shape of craniotomy	Minimize the extent of tissue trauma without sacrificing completeness of tumor removal Decrease operative time
Identify sagittal and lateral sinuses for burr hole placement	Increase safety by limiting trauma to major venous sinuses
Identify large draining veins entering major sinuses	Tailor dural opening to preserve veins Increase safety by limiting trauma to veins
Identify eloquent cortices in close proximity to tumor	Limit neurological injury Shorter hospital stays
Identification of the boundaries of the frontal sinus	Decrease risk of meningitis or mucocele
Determine the remaining margin of tumor during intracapsular debulking of large tumors	Limit neurovascular injury to deep structures beyond deep margin of tumor

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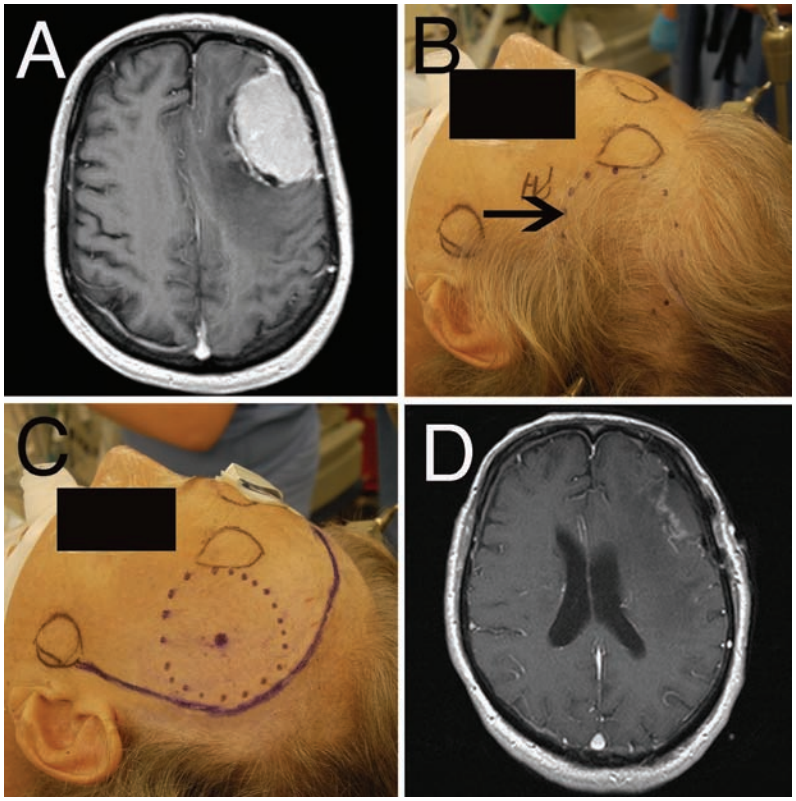


Fig. 35.6 **(A)** This 58-year-old female presented with headaches and a 4 cm left frontal convexity meningioma. **(B)** Following patient registration, the boundaries of this left frontal convexity meningioma were marked (*arrow*) to plan the **(C)** scalp and craniotomy flaps. **(D)** The patient underwent an uncomplicated gross total resection of the tumor with a 1 cm dural margin.

brain retraction—and neuronavigation have resulted in a paradigm shift in the management of tumors of the cranial base.[22] This shift has improved rates of total resection and decreased neurovascular injuries and lesions previously considered inoperable have now become resectable with a chance for cure. Moreover, the introduction of endoscopic minimally invasive techniques would not have been possible without the assistance of IGS.

The main benefit of IGS systems in skull base meningioma surgery lies in the ability to determine the optimal surgical trajectory and to identify neurovascular structures en route to and in close proximity to the tumor.[16,22] IGS can be useful intraoperatively to identify the venous sinuses before the bone opening and to assist in burr hole placement around the sinuses. During the resection, IGS can help in estimating the degree of interior decompression of a large tumor before mobilizing the tumor borders. Ultimately, though, slavish reliance on IGS will often lead to grossly subtotal resections. The only reliable method to gauge extent of resection is to identify all of the tumor borders visually, at which point all that remains is to sever the tumor from its attachment. Further advantages afforded by neuronavigation in the resection of skull base meningiomas are summarized in **Table 35.4**.

Critical to both open and endoscopic skull base meningioma removal is assessment of the normal and potentially hyperostotic bony anatomy via thin-cut CT, in addition to the excellent soft tissue resolution of gadolinium-enhanced MRI. With experience of over 1000 endoscopic endonasal procedures, Snyderman et al[23] note that CT and MRI fusion is essential to navigate the "black box" of the skull base endoscopically from below. For both open and endoscopic approaches, we prefer to obtain both imaging modalities for skull base tumors and fuse them into a single image set for registration and intraoperative navigation. This enhances the ability to use the device to recognize critical bony landmarks en route to the pathology and to discern any abnormal bony that should be removed if tumor infiltration is suspected.

Conclusion

Stereotactic neuronavigation systems can both increase extent of resection and improve the safety profile during intracranial meningioma resection. In contrast to some centers reporting minimization of craniotomy size as one of the most important benefits, we stress optimization of craniotomy size to ensure adequate resection of convexity tumors. Although most systems entail a small increase in operative setup time, the benefits of increased surgeon confidence and identification of critical structures during the operation will most likely translate into shorter overall operative times and less neurovascular injury. Increased extent of resection directly translates into less tumor recurrence, and the potential for improved neurological outcomes can shorten hospital stays—offsetting the increased cost of image guidance. With continued advancement in endoscopic instrumentation and techniques, IGS systems will assume an even greater importance in the resection of skull base meningiomas. It may be our predigital upbringing, but we doubt that any system will ultimately prove superior to the accomplished tumor surgeon's anatomical knowledge and "x-ray vision" as Dr. Albert L. Rhoton has always called

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Table 35.4 Utility of Image Guidance Surgery Systems for Meningiomas of the Skull Base

Advantage	Benefit to Surgeon/Patient
Identify the margins of the tumor and the dural tail	Ensure maximal extent of resection Decrease risk of recurrence, need for further treatment
Plan ideal trajectory and angle of attack	Determine shortest, safest route to tumor (optimal "skull base" approach)
Use probe's eye (trajectory) view to understand normal and pathological anatomy	Better understand regional anatomy Increase surgeon confidence Visualize structures not visible to surgeon along line of sight (arteries, veins, and cranial nerves) Limit neurovascular injury Invaluable teaching tool for residents and experienced surgeons
Determine the remaining margin of tumor during intracapsular debulking of large tumors	Limit neurovascular injury to deep structures beyond deep margin of tumor
Identify critical landmarks during endoscopic procedures; opticocarotid recess, internal carotid artery, foramen rotundum, vidian canal, posterior clival border	Limit neurovascular injury Limit operative time by early identification of critical neurovascular structures to facilitate bony removal
Identify abnormal bony involvement by tumor, especially if landmarks are obscured by pathology	Ensure maximal extent of resection Decrease risk of recurrence, need for further treatment

it. Prospective, randomized studies are ultimately needed to determine if the purported benefits of IGS prove superior to cases performed without neuronavigation in terms of tumor control, morbidity, and overall cost.

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Chapter 36

Intraoperative Magnetic Resonance Imaging–Guided Resection of Meningiomas

Amitabh David Singh and Garnette Roy Sutherland

To improve is to change; to be perfect is to change often. —Sir Winston Churchill

The progress of neurosurgery has, to a large extent, depended on advances in technology, in particular, those related to lesion localization. Adapting these technologies to the operating room (OR) environment refined both lesion localization and resection control.[1] In the surgical resection of meningioma, intraoperative imaging provides an update to diagnostic images, optimizes craniotomy placement, and provides intraoperative evidence that surgical goals have been accomplished.

History

The first intraoperative magnetic resonance imaging (iMRI) system was installed at Brigham and Women's Hospital, Boston, Massachusetts, in 1994.[2] The system was based on a vertical biplanar (double donut) magnet, allowing the surgeon to operate within the 56 cm aperture, and thus integrated real-time imaging with the surgical procedure. Because surgery was conducted within the magnetic field, all surgical instrumentation and equipment, including the OR table, microscope, anesthesia equipment, and patient monitoring devices were required to be manufactured from MR-compatible material.[3–5] Although the value of this technology was evident, it was not widely adopted, due, in part, to the restricted surgical space, the need for all instrumentation to be MR compatible, and the cost associated with installation and maintenance. General Electric (Milwaukee, WI), the manufacturer of this iMRI configuration, no longer markets the system.

Other low-field iMRI systems, based on magnets of variable field strengths ranging from 0.12T to 0.3T, were built or adapted for surgical use.[6–8] These iMRI systems are of open configuration and have the magnet adjacent to or below the OR table. For example, the PoleStar iMRI system (Odin Medical Technologies, Yokneam, Israel) is based on a portable 0.12T magnet positioned below the OR table and surgical field.[6] The very low field allows for use of standard neurosurgical instrumentation because magnetic pull does not affect tool manipulation. For imaging, the magnet is moved up into the surgical field. Although the system has many attractive features, the low magnetic field precludes advanced imaging sequences or images of diagnostic quality. Furthermore, magnet design makes imaging of the posterior fossa and upper cervical spine problematic.[9]

To obtain image resolution comparable to that of diagnostic MRI scanners, higher field 1.5T iMRI systems were developed.[1,10,11] For example, the University of Calgary, in collaboration with the National Research Council of Canada, developed an iMRI system based on a movable, ceiling-mounted 1.5T magnet.[1,11] The system included the design and manufacture of an MR-compatible OR table, radiofrequency (RF) coils specifically designed for OR use, and a custom-made gradient insert that increased the available working aperture. Together, these provided a patient-focused surgical environment with freedom to optimize patient positioning and improved signal-to-noise ratio.

The movement of the MR system obviated the need to transport the patient, thus enhancing patient safety, sterility, and efficiency within the OR. In addition, the configuration permitted the use of standard neurosurgical instruments and monitoring devices. The configuration, as adopted by other neurosurgical units, allowed sharing of technology between surgery and other disciplines. As a result, costs related to obtaining and maintaining the MR technology could be offset.

Recently, iMRI systems based on 3.0T magnets have been integrated into the OR.[12–15] The 3.0T system at the University of Calgary is based on the same moving magnet configuration as that developed for 1.5T. The trend toward a higher magnetic field is based on the linear relationship between signal to noise and magnetic field

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strength.[16] In general, improved signal to noise relates to improved image resolution and decreased acquisition time, and high performance gradients enhance diffusion tensor imaging,[16] functional imaging,[17] and multi-nuclear MR spectroscopy.[18]

The Magnetic Environment

Static Magnetic Field

The static magnetic field is the primary magnetic field of an MRI scanner. It is highest at the isocenter of the magnet and decays as distance from the isocenter increases in a nonlinear relationship.[4] The area around the MRI scanner that is considered safe is outside the 5 gauss (5G) limit.[3,4] A 3T magnet will have a larger restricted area (higher than 5G) compared with a 1.5T magnet. For 1.5T and 3T magnets, this restricted area has been considerably decreased through active magnetic shielding. Ferromagnetic objects, including instruments and equipment, if brought within the 50G magnetic field, may become projectiles, accelerating toward the magnet.[19] Based on this principle and to increase the margin of safety, ferromagnetic instruments or equipment need to remain outside the 5G limit. In the case of the movable iMRI system, when the magnet is moved toward the patient to obtain images, noncompatible equipment and instruments are moved out of the new 5G limit that forms around the patient.

Gradient Magnetic Field

In addition to the static magnetic field, a variable gradient (according to a prescribed sequence) is applied in three directions (x,y,z). The frequency of variable gradient is close to that of audible acoustic frequency, which is responsible for the noise during image acquisition. The variable gradient also has the potential to induce voltage or current in tissues, creating heat and neural or muscular activation. It can also cause voltage changes in pacemakers, inadvertent instrument movement, and image artifact.[3] To minimize the effect of the gradient magnetic field, U.S. Food and Drug Administration (FDA) guidelines for magnetic resonance equipment safety require all scanners to be programmed to function at a defined field.

Radiofrequency Electromagnetic Field

RF coils produce an electromagnetic field needed to excite hydrogen molecules within tissues, and the same coils, or different coils, are used to collect MR signal from the excited molecules as they relax. As relaxation rates differ between tissue types, soft tissue contrast is achieved.

In extreme cases, heat produced by an RF field can cause burns, interfere with electrical instruments (such as anesthetic equipment), and induce currents in looped conductors (such as electrocautery wires). Negative effects can be mitigated by avoiding looped wires and individually screening equipment for compatibility within the RF field.[3]

Anesthesia Considerations

The MR environment poses challenges for anesthetists as well as for the surgeons, making it important for them to be trained in device selection and limitations. These involve considerations related to the anesthesia machine, monitoring and infusion equipment, anesthetic agents, and patient positioning and exposure. Electric noise generated by pulsating electric and magnetic energy on conductors distorts the physiological waveforms on the monitors,[20] in particular the electrocardiogram ST segment. Likewise, temperature control, blood pressure monitoring, tissue oxygenation, and ventilation can be affected in the presence of RF waves produced by an MR scanner. Accepted guidelines for patient safety, monitoring, and equipment have been published.[3,20]

The iMRI System at the University of Calgary

Central to the patient-focused iMRI environment at the University of Calgary was the design and manufacture of an integrated MR-compatible OR table (**Fig. 36.1A**). The table design, head fixation, and hydraulic table movement allow the patient to be positioned supine, lateral, and prone. The table is as wide as conventional OR tables, with a slim profile so as to optimize head placement within the magnet isocenter. Both three- and four-pin head fixation have been developed for use with the table and magnet. The provision of hydraulic actuators allows maximum positioning capability without backlash and the ability to smoothly reposition during microdissection. Thus the table becomes an important adjunct to microsurgery.

The iMRI system is based on a ceiling-mounted movable 3.0T magnet (IMRIS, Winnipeg, Canada), (**Fig. 36.1B**) capable of transfer into and out of the OR. The magnet measures 1.73 m in length and has an internal diameter of 90 cm. The working aperture is 70 cm with shielded gradients in place. The 5G restricted area measures 4 m × 3.5 m. The system provides a large homogeneous field of view measuring 50 × 50 × 50 cm at the magnet's isocenter, and it includes high-performance 45 mT/m gradients.

RF coils allowing transmission and receipt of signal were developed and integrated into the head fixation apparatus. The RF-coil design (**Fig. 36.1C**) includes a transmit body coil within the bore of the magnet and several receiving coils. These receiving coils (IMRIS, Winnipeg, Canada) are able to be separated into two components with four channels each, such that the bottom coil is left in place during surgery. Increased number of signal channels, improved software design, and parallel imaging techniques permit high-resolution imaging with decreased total acquisition time.

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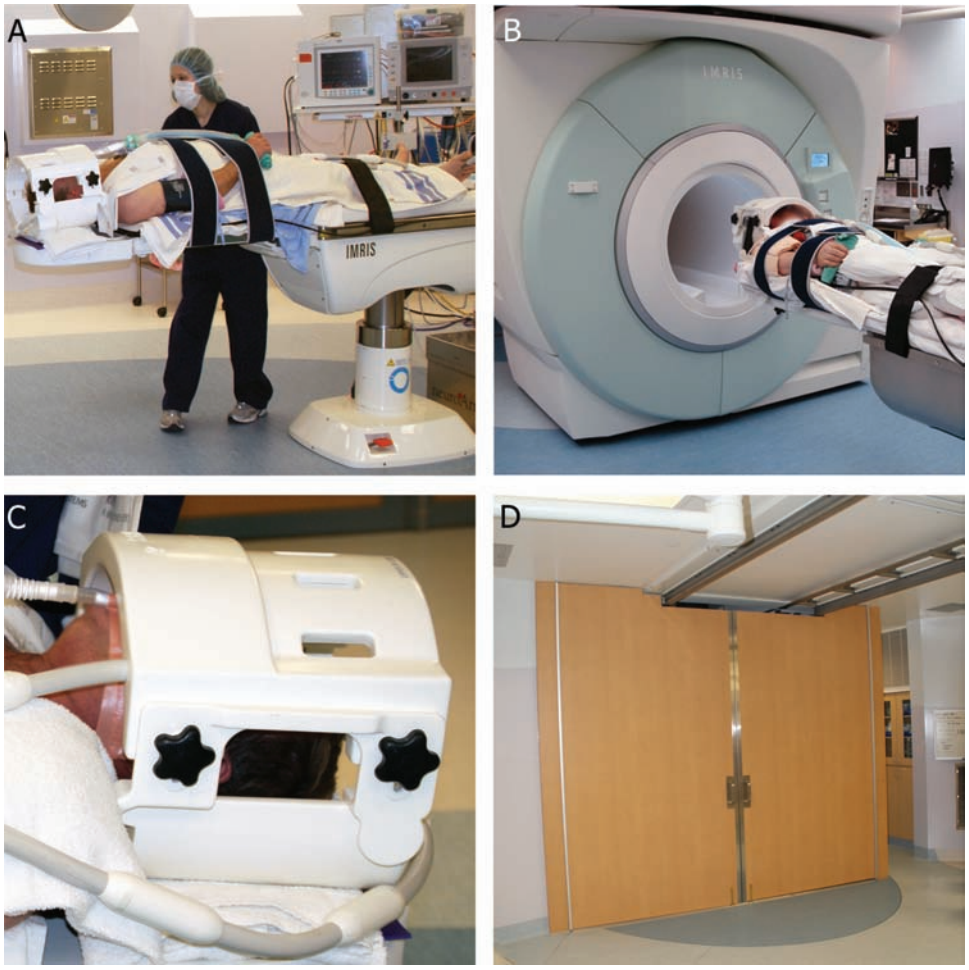


Fig. 36.1 Components of the intraoperative magnetic resonance imaging system at the University of Calgary. **(A)** A slim-profile, hydraulically controlled operating room table is manufactured from stainless steel and plastic composites and is shown with a patient positioned supine. **(B)** The large-bore 3.0T magnet is moved over a patient positioned for imaging. **(C)** A unique eight-channel radiofrequency (RF) coil is positioned for surgical planning imaging. During surgery (not shown), the top half of the RF coil can be removed and sterilely draped, while the bottom half can be incorporated into three-pin head fixation. **(D)** When the magnet is not needed for surgery, it is moved to an adjacent room behind wooden doors. Field lines corresponding to 50 gauss (*dark blue)* and 5 gauss (*light blue*) are marked on the floor for imaging and storage positions.

The OR is outfitted with whole-room copper shielding (Gaven Industries, Saxonburg, PA), making use of wave guides, filters, and the conversion of electrical signals to fiber optics to accommodate necessary electrical devices. The iMRI workstation is located adjacent to the operating room, separated by a transparent copper mesh-impregnated glass panel. Wooden doors (**Fig. 36.1D**) separate the magnet alcove from the operating room for safety.

Imaging sequences are provided by Siemens 3.0T software (Syngo MR B15V, Siemens, Erlangen, Germany; optimized for iMRI application by IMRIS, Winnipeg, Canada). Intraoperative imaging utilizes standard and advanced imaging sequences, including T1-weighted, T2-weighted, fluid-attenuated inversion recovery (FLAIR), time-of-flight MR angiography, diffusion-weighted images, and diffusion tensor images (DTI). Postprocessing software permits the rapid generation of three-dimensional tractography (**Fig. 36.2**). Gadolinium can be used to define unsuspected residual tumor at various stages of surgical dissection. However, if administered during the planning stage before surgery, it may diffuse into normal tissue, making the identification of residual tumor difficult in intradissection scans.

Application of iMRI in Meningioma Surgery

Indications

Although the authors recognize that meningiomas can be safely excised without iMRI, intraoperative imaging improves the surgical performance for all neurosurgeons and trainees with varying degrees of expertise. Furthermore, because meningioma surgery is already standardized, it is an ideal opportunity to train personnel and staff to work within an MR environment. With this in mind, there are several important aspects of meningioma resection that are benefited by iMRI.

Surgical Planning Images

Surgical planning images are obtained after induction of anesthesia and patient positioning. Intraoperative imaging provides guidance for optimal craniotomy placement, which is particularly important for convexity meningioma (**Fig. 36.3**). The three-dimensional localization of

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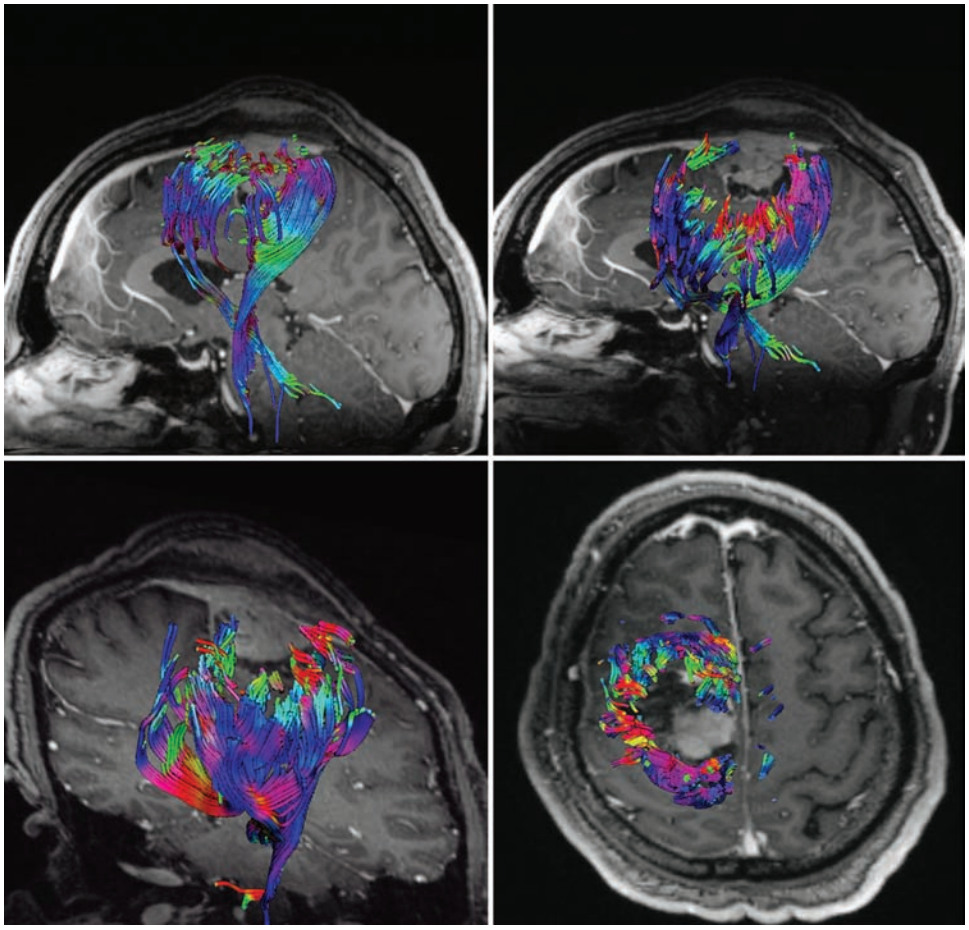


Fig. 36.2 Three-dimensional tractography. Surgical planning three-dimensional tractography obtained from a patient with a midsagittal atypical (grade II) meningioma. Visualized white-matter tracts surround the lesion, emphasizing the importance of dissecting the tumor–arachnoid interface throughout the circumference of the lesion.

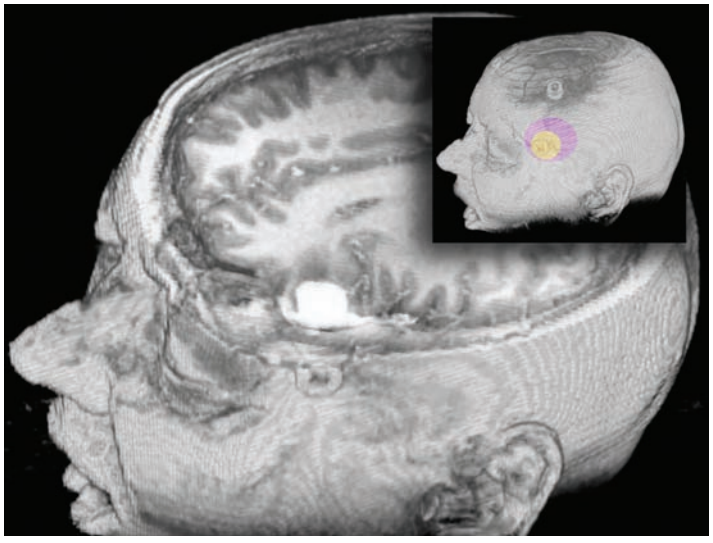


Fig. 36.3 Preoperative craniotomy placement. The figure shows a three-dimensional reconstruction from surgical planning T1-weighted, gadolinium-enhanced images. The relationship of the meningioma and its dural tail to a surface fiducial is demonstrated. (Inset) A limited craniotomy (*yellow)* and an extended craniotomy, to include the dural tail, are demonstrated on a surface reconstruction.

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the meningioma can be projected onto the scalp. Thus the localization of the scalp incision, the extent of bone flap removal or craniotomy size, and the dural opening are tailored to include the meningioma and its dural involvement. While one is operating on skull base or other deeply placed meningioma (such as intraventricular meningioma), surgical planning images often aid in determining and verifying the location of the tumor and its relationship to the planned surgical corridor.

Frameless neuronavigation is often integrated with iMRI to enhance craniotomy placement. The advantage of this combined approach is that it allows the registration of the navigation with immediate preoperative images, rather than with diagnostic imaging, thus decreasing the dependence on updated diagnostic imaging for surgical planning. Moreover, the risk of fiducial displacement is virtually eliminated because with iMRI, the fiducials are placed following anesthesia and patient positioning.

The immediate preoperative radiological assessment by iMRI provides knowledge of any potential change in the meningioma or adjacent brain. It is well known that meningiomas diagnosed during pregnancy may significantly decrease in size postpartum.[21] Atypical or malignant meningiomas may significantly increase in size[22] and, on very rare occasion, hemorrhage may occur within or adjacent to the meningioma.[23] Because brain edema associated with meningioma is dynamic and multifactorial, its extent may have changed considerably from diagnostic imaging. This knowledge assists in efficient and effective OR planning.

Tensor-weighted images obtained during surgical planning provide visualization of the white matter tracts. This provides information about the proximity of important white matter tracts to the tumor. Thus the surgeon knows in advance to proceed with caution while dissecting the tumor from adjacent white matter. It is important, however, for the surgeon to recognize that the tracts may shift during dissection; therefore updated intradissection images may be needed.

Intradissection Images

Intradissection images are obtained during surgical dissection to reestablish spatial orientation, to evaluate surgical goals, and to detect any residual tumor.

Resection of large meningiomas (**Fig. 36.4**) with brain shift can result in a loss of spatial orientation with respect to the tumor and adjacent brain. This is particularly so for tumors located at the skull base, tentorium cerebelli, and adjacent to the superior sagittal sinus. These updated images help to prevent inadvertent injury to the surrounding neurovascular structures. The learning curve for less experienced surgeons while operating in regions demanding more surgical finesse and proficiency may be reduced.

For some meningiomas, such as those invading the dural sinuses or encasing vessels, subtotal resection may be preferable. Intradissection MR images display residual tumor volume and its relationship to major vessels, thus enabling the surgeon to determine whether further dissection is needed. It is also important for residual tumor volume to be small enough to be compatible with a radiosurgery option. For most convexity meningiomas, it is unlikely that unsuspected residual tumor will be found, particularly if the surgical goal involved excision of the adjacent dura. In the series of patients operated on at the University of Calgary, intradissection images showed evidence of unsuspected residual tumor in two patients. One patient had residual tumor in the orbital roof and cavernous sinus,[24] whereas the other, with a recurrent falcine meningioma, had a satellite nodule beneath the thickened pia-arachnoid on an adjacent gyrus.[25]

Magnetic resonance angiography (MRA) and venography (MRV) provide images of the arterial supply and venous drainage of both the meningioma and adjacent brain. Intradissection MRA may show remaining vascular supply to the tumor, whereas MRV can be used to demonstrate patency of the dural venous sinuses. In rare cases, in which total resection is planned for meningioma that invades dural sinuses, intradissection imaging that shows failure to reestablish flow through the sinus could expedite endovascular and other postoperative treatment strategies.

Intradissection images also serve as useful media to examine the effect of surgery on the brain and white matter tracts. In addition, one can achieve proficiency in recognizing subtle postoperative artifacts, such as blood or oxidized regenerated cellulose (Surgicel, Ethicon, Somerville, NJ) in the resection cavity.

Quality Assurance Images

Quality assurance images provide confirmation that surgical goals have been achieved and whether any acute surgical complication is present. Postoperative hemorrhage, within the resection cavity or adjacent brain, is defined before reversal of anesthesia, and depending on size, may be evacuated. Similarly, the quality assurance images, depending on selected imaging sequences, will define the extent of brain edema or ischemia.

This knowledge is important in optimizing and expediting postoperative management. The known presence or absence of acute surgical complication determines length of recovery room time and the potential need for intensive care unit (ICU) care (**Fig. 36.5A,B**). Furthermore, quality assurance images replace the need for immediate postoperative images to be gathered by diagnostic imaging.

University of Calgary Data

From 1997 to 2009, a total of 109 patients with meningioma underwent surgery using iMRI guidance. There were 66 (61%) females and 43 (39%) males. The mean age of the patients was 55 ± 17 years (range 0 to 84 years). In this series, cerebral convexity was the commonest location of the meningioma, followed by the parasagittal/falcine and

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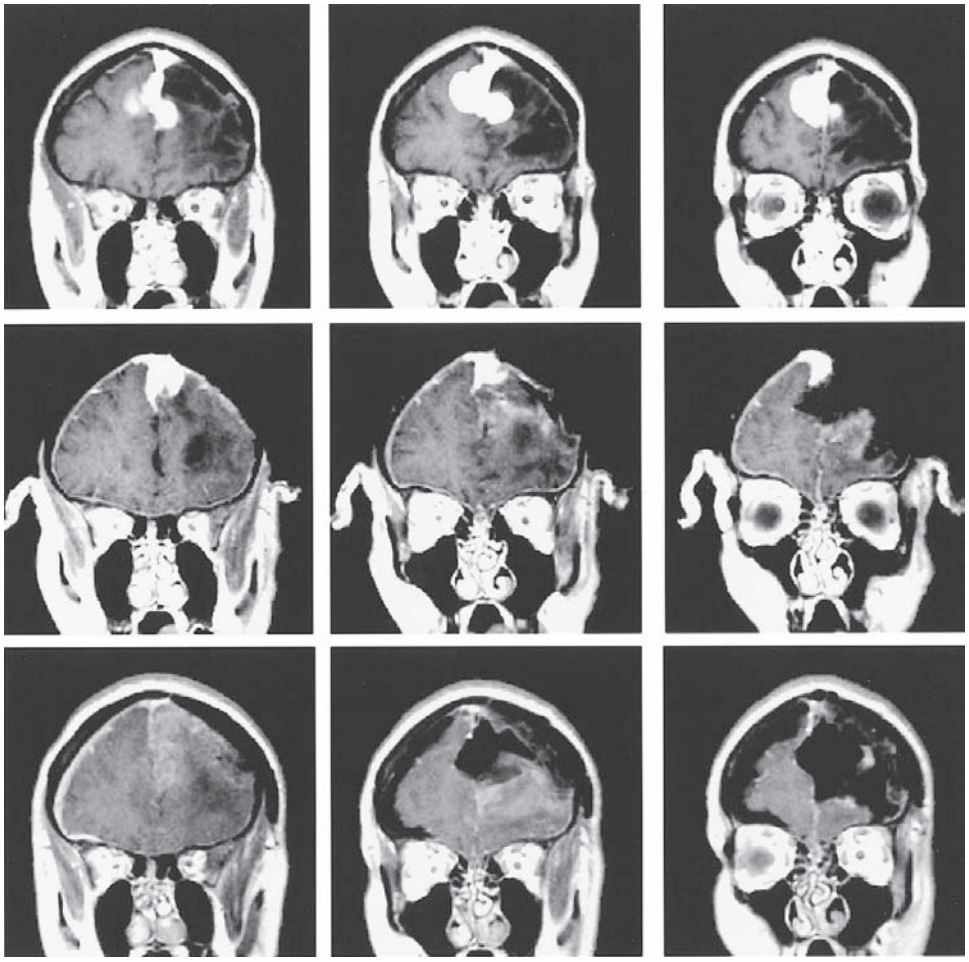


Fig. 36.4 Previously published T1-Gd images obtained from a patient with recurrent anterior parafalcine meningioma. These images were acquired in 1998, using the prototype 1.5T intraoperative magnetic resonance imaging system. (Upper panel) Surgical planning images showing the parafalcine meningioma. (Middle panel) Intradissection images acquired following presumed total resection of the tumor, demonstrating unsuspected residual neoplasm. (Lower panel) Quality assurance images acquired following wound closure, but before reversal of anesthesia. Used with permission of Kaibara T, Saunders JK, Sutherland GR. Utility of a moveable 1.5 tesla intraoperative MR imaging system. Can J Neurol Sci 1999;26,4:315.

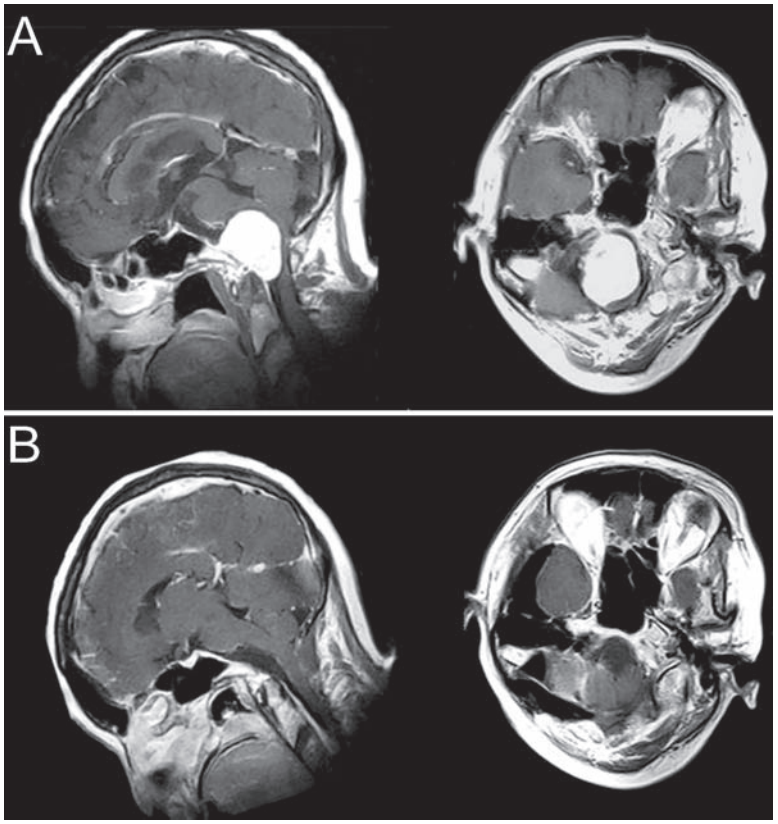


Fig. 36.5 Impact of quality assurance imaging. **(A)** Surgical planning and **(B)** quality assurance images from a patient with a large foramen magnum meningioma. The quality assurance images show excellent brain stem decompression and the absence of acute hemorrhage.

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sphenoid wing/ anterior clinoid process. The least common location was the foramen magnum (**Table 36.1**).

The 1.5T iMRI was used from 1997 to 2008 and then updated to the 3.0T iMRI. For 91 meningiomas, the prototype 1.5T iMRI system was used as a surgical adjunct, whereas the commercially available 3.0T iMRI system was used in 18. Unsuspected residual tumor was identified on intradissection images in two patients, both among the 1.5T iMRI cohort. In one case, the unsuspected residual tumor was resected, whereas in the other the decision was made not to excise the lesion. There was one postoperative infection. (**Table 36.2**).

Table 36.1 Meningioma Cases by Sex, Age, and Location

Meningioma surgeries (*n* = 109)	**Number**	**%**
Males	43	39
Females	66	61
Age groups		
0 to 20 years (mean ± SD)	4 (13 ± 9)	4
21 to 84 years (mean ± SD)	105 (55 ± 17)	96
Location		
Cerebral convexity	46	42.2
Parasagittal/falcine	14	12.8
Intraventricular	2	1.8
Subfrontal/olfactory groove	11	10.1
Suprasellar	5	4.6
Sphenoid wing/anterior clinoid process	14	12.8
Middle cranial fossa	4	3.7
Cerebellopontine angle	3	2.8
Petroclival	3	2.8
Posterior fossa convexity	2	1.8
Tentorium cerebelli	4	3.7
Foramen magnum	1	0.9

Abbreviation: SD, standard deviation.

Table 36.2 Meningioma Cases by Magnetic Resonance System, Infection, and Unsuspected Residual Tumor

	1.5T iMRI	**3.0T iMRI**
Number of cases	91	18
Unsuspected residual tumor on iMRI	2	0
Postoperative infection	1	0

Abbreviation: iMRI, intraoperative magnetic resonance imaging.

Integrating Robotics with 3T iMRI

The promise of robotics is to provide increased precision and accuracy and, when coupled with the unique capability of the human brain, to provide unparalleled opportunity to improve neurosurgical outcome.[26] NeuroArm, an image-guided robotic system for neurosurgery, was developed in collaboration between the University of Calgary and MacDonald, Dettweiler, and Associates, Ltd. (Brampton, ON) to exploit these advantages.[27,28]

The robot consists of two MR-compatible anthropomorphic manipulators, a digitizing arm for registration to intraoperatively acquired images, and a field camera to monitor the surgical field. These components are mounted on a counterweighted, adjustable mobile base, which can be easily transported into and out of the OR (**Fig. 36.6**).

The human–machine interface is the point of contact and control between the surgeon and robotic system. It consists of two force-feedback hand controllers to maneuver the robot manipulators, a monitor displaying the MR images in three-dimensional (3-D) with robot tool overlay, and a second monitor that presents the robotic manipulators in virtual space, including their relationship to the RF coil and magnet. Additional monitors show the surgical field and a two-dimensional (2-D) display of the operative site. A 3-D display of the operative site is provided by a binocular stereoscopic display unit. Sound is projected by two speakers, and the surgeon is able to communicate with all OR personnel via a microphone linked to a headset communication system. The human–machine interface therefore merges human decision making with the precision and accuracy of robotic movement by providing the surgeon with sensory input and by providing the robot with motor feedback. Touch is provided by two PHANTOM haptic hand controllers (SensAble Technologies Inc., Woburn, MA), each with six degrees of freedom. Force sensors have the ability to provide the surgeon with real-time physical quantification of the deformation of normal and pathological tissue. Based on this feedback, future surgeons may be able to set force limits, improving the safety of surgery (**Fig. 36.7**).

Complex neural circuits in the brain are responsible for the processes of making choices based on sparse datasets.[29] This result is a nonlinear computational system capable of evaluating multiple choices simultaneously as well as continuous processing and modification after the action has been initiated, incorporating past experience. A surgeon can anticipate unintended motion faster than a computer owing to this nonlinear method of evaluating choices.

On the other hand, computer circuitry is much faster than synaptic transmissions in the brain. This gives rise to a linear system of computation that involves step-by-step evaluation of all possible choices before selecting the most feasible option. Therefore, machines are capable of producing only a single solution to generate a given movement. Conversely, surgeons can intuitively alter speed, force, and hand position efficiently and effectively to complete a task in a variety of approaches.

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Fig. 36.6 The neuroArm surgical robotic platform system, composed of two manipulator arms mounted on a mobile base, along with a field camera and digitizing arm.



Fig. 36.7 The neuroArm in position for removal of an olfactory groove meningioma (upper). Lower image shows the surgeon controlling the manipulator arms while seated at a sensory-rich workstation adjacent to the operating room.

At any given point in surgery, an infinite number of actions are possible within the surgical field, which, based on past experience, are organized in ascending levels of importance by the brain. True automation of surgery would therefore require a fundamental change in computer design and capability. The surgeon, when presented with infinite sensory data, can rapidly combine multiple incomplete datasets to execute the correct action.

This master–slave organization therefore effectively integrates the distinctive strengths of humans (executive function) and machines (precision) for the conduct of neurosurgical procedures.

Science may set limits to knowledge, but should not set limits to imagination
—Bertrand Russell

Conclusion

In the world around us, evolving concepts and technology transform our lives to a higher degree of efficiency. The introduction of MR technology and, more recently, of a surgical robot into the neurosurgical arena is an example of this dynamic process that defines human existence. This chapter portrays how the integration of man and machine improves precision within the operating room, resulting in superior quality of surgery and patient outcome. Yet another step toward perfection, which has an indefinable limit!

The benefit that iMRI technology offers over traditional surgical methods has been demonstrated often. However, due to the limited number of iMRIs installed, no class I evidence exists to prove the merit of this innovation. Because the cost associated with acquisition and maintenance of iMRI is either decreased or offset by various

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strategies, more institutions will find it feasible to adopt this technology. Thereafter, it becomes imperative to establish the benefit of iMRI in the setting of a prospective, randomized, controlled trial.

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Chapter 37

Surgical Management of the Cerebral Venous Sinuses

Marc P. Sindou and Jorge E. Alvernia

Introduction

Surgery of meningiomas involving major dural sinuses challenges the surgeon with a dilemma: leave the invasive fragment and have a higher rate of recurrence, or attempt a total removal and have the venous circulation at risk. The current tendency is resection of the tumor mass outside the sinus wall(s) with coagulation of the remnant. For meningiomas with complete sinus occlusion, most authors advocate en bloc removal of the invaded portion based on the assumption that collateral venous pathways have developed in this scenario. Conventional wisdom states that complete removal of the invaded sinus is of little danger, and venous flow restoration is not needed. This, however, may not always be true. Other authors, although few, favor attempts at gross total removal with venous reconstruction. This latter attitude is our preference. Whatever approach is pursued, a clear understanding of the collateral venous anatomy and the degree of sinus invasion is warranted when surgery on the sinus is considered.

The outcomes, in terms of recurrence rate, morbidity, and mortality, of complete tumor removal in 100 of our patients (January 1980 to January 2001), including removal of the invaded portion of the dural venous sinus, with or without the restoration of venous circulation, were published in 2006.[1] Meningiomas originated at the superior sagittal sinus in 92 of the cases (28 in the anterior, 48 in the middle, and 16 in the posterior third), the transverse sinus in five, and the confluence of sinus in three.

A simplified classification scheme based on the degree of dural sinus involvement was applied: type I: lesion attachment to the outer layer of the sinus wall; type II: tumor fragment inside the lateral recess; type III: invasion of the ipsilateral wall; type IV: invasion of the lateral wall and roof; types V and VI: complete sinus occlusion, with or without the contralateral wall free, respectively (**Fig. 37.1**).

Lesions with type I invasion were treated by peeling the outer layer of the sinus wall. In cases of tumor sinus invasion types II to VI, two strategies were employed: a "nonreconstructive" (coagulation of the residual fragment or global resection) and a "reconstructive" one (suture, patch, or bypass).

Gross tumor removal was achieved in 93% of the cases and reconstruction of the sinus was attempted in 45 (65%) of the 69 cases with wall and lumen invasion. The overall recurrence rate in the study was 4%, with a follow-up ranging from 3 to 23 years (mean 8 years). The mortality rate was 3%, all cases due to brain swelling after en bloc resection of a type VI meningioma without venous restoration. Eight patients who harbored a lesion in the middle third portion of the superior sagittal sinus had permanent neurological worsening, likely due to local venous infarction. Six of these patients had not undergone a venous repair procedure. Venous reconstruction did not increase the morbidity and mortality rates in our series. From this study we concluded that (1) the relatively low recurrence rate of 4% favors attempts at complete removal, including the portion invading the sinus; (2) because the subgroup of patients without venous reconstruction displayed statistically significant clinical deterioration after surgery compared with the other subgroups (p = 0.02), venous flow restoration seems justified when not too risky (**Fig. 37.2**).

Preoperative Workup

Magnetic resonance imaging (MRI) scans with and without contrast medium are the key. MRI (T1-weighted sequences with and without gadolinium injection and T2 sequences) is more effective in delineating the tumor and differentiating it from surrounding structures. Gadolinium enhancement of the invaded dura allows the site

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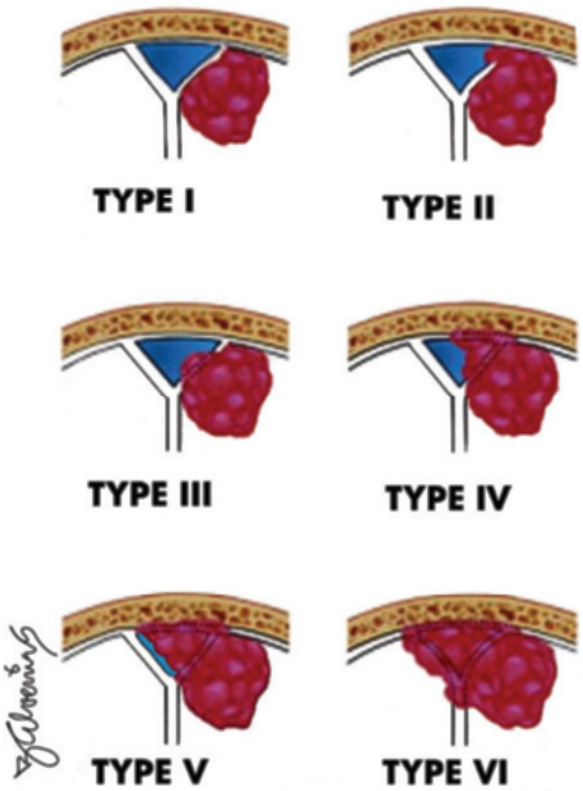


Fig. 37.1 Classification for meningiomas according to sinus invasion. Type I, meningioma attached to the outer layer of the sinus wall; type II, lateral recess invaded; type III: ipsilateral wall invaded; type IV, both ipsilateral wall and roof invaded; type V, sinus totally occluded, but contralateral wall free of invasion; type VI: sinus totally invaded, including all three walls. With permission from Sindou M, Alvernia JE. Results of attempted radical tumor removal and venous repair in 100 consecutive meningiomas involving the major dural sinuses. J Neurosurg 2006;105(4):514–525.

of exposure to be predicted. However, it must be kept in mind that enhancement of the dura adjacent to the main tumor mass may indicate actual tumor invasion or may simply indicate hyperemia. Venous magnetic resonance offers additional useful information on venous system involvement. Magnetic resonance angiography is not adequate in providing a reliable depiction of the tumor vascularization and local hemodynamics. Therefore, angiography through the transfemoral route is of value before establishing the detailed surgical strategy.

Selective bilateral internal and external carotid subtraction angiography as well as vertebral angiography serve to determine the dural and cortical–pial supply ipsilateral or contralateral to the tumor. The arterial phase is useful to predict the difficulty of dissection of the capsule from the cortex. As we have shown in prior publications, dissection entails neurological risk when a pial vascular supply is identified.[2,3] When meningeal supply is important, preoperative embolization may be of some value in producing tumor necrosis and dropping the operative risks to the patient by diminishing the blood loss that accompanies resection of these tumors.

The late venous phases with bilateral filling of the sagittal sinus are required for the full evaluation of sinus patency and collateral venous pathways. Oblique views can depict the superior sagittal sinus (SSS) throughout its entire course. Various degrees of sinus occlusion can be observed, from simple compression with narrowing of the sinus lumen to intraluminal defect to total occlusion. Complete occlusion may be assumed when segments of the sinus are not visible and collateral venous channels are present. The pattern of venous drainage and venous collateral channels must be established preoperatively to determine the surgical approach (**Fig. 37.2**).

◆ Surgical Technique

General Principles

Positioning

The patient is placed in the semisitting (lounging) position to allow a good venous return without increased intracranial pressure. Air embolism, although possible, is not a frequent risk because of the relatively high level of the intracranial venous pressure in these patients. The problem can be avoided or safely controlled in experienced surgical hands.

Exposure

The operative exposure should be as extensive as necessary. The skin flap and craniotomy should extend across the midline to permit visualization of both sides of the sinus and ~3 cm outside the margins of the occluded sinus. However, such a large access should be reconsidered in the presence of scalp, pericranial, or diploic collateral venous pathways, which may be impaired during the approach.

Arterial feeders within the dura, especially those coming from middle meningeal arteries, should be coagulated or clipped before being cut.

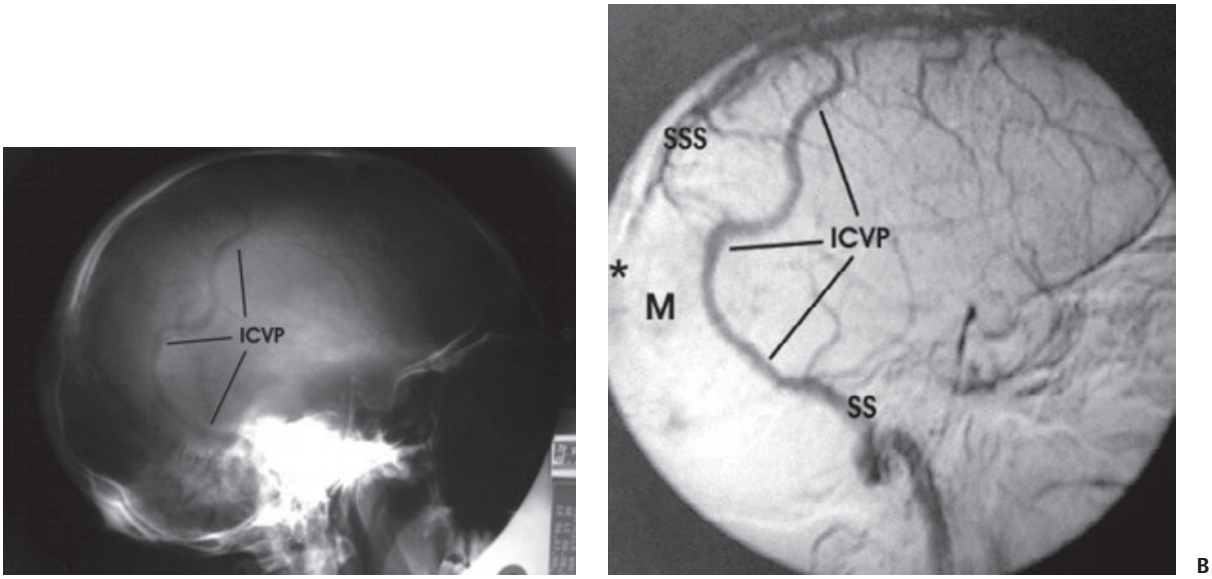
The dura is incised in a circumferential manner around the margin of the tumor on the convexity and along the border of the corresponding portion of the superior sagittal sinus, taking care not to compromise the adjacent veins (bridging veins) to the sinus.

Tumor Surgery

The microscope shows the division of the meningioma's attachment to the lateral wall of the sinus and to the neighboring falx. The tumor is detached from these structures by using the cutting mode of the bipolar coagulation forceps, thus cutting off the tumor's dural blood supply.

Intracapsular debulking is performed so that the remaining capsule of the tumor can be easily mobilized from the underlying cortex. Under the microscope, an extraarachnoidal plane of dissection must be carefully searched for and identified when possible. When absent, the plane of dissection becomes subpial due to pial incorporation into the tumor capsule[3].

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A B

Fig. 37.2 Grade VI parasagittal meningioma located at the posterior third. **(A)** Plain x-ray; lateral view showing a major intraosseous collateral venous pathway (ICVP). **(B)** Digital subtraction angiography, venous phase; lateral view showing the venous collateral from midthird of the superior sagittal sinus (SSS) to the sigmoid sinus (SS). Note a complete posterior third SSS occlusion (*). With permission from Sindou M, Alvernia JE. Results of attempted radical tumor removal and venous repair in 100 consecutive meningiomas involving the major dural sinuses. J Neurosurg 2006;105(4):514–525.

Sinus Surgery Procedure

Because there are frequent discrepancies between images and anatomical findings, the sinus should be explored through a short incision (~5 mm linear opening) to disclose any intrasinus fragment.

Temporary control of venous bleeding from the sinus and afferent veins is easily obtained by packing small pledgets of hemostatic material (Surgicel, Ethicon, Inc., Somerville, NJ) in the lumen and at the ostia of the afferent veins (**Fig. 37.3** and **37.4**). Balloons should not be used because they do not pass easily through the sinus septa and may injure the sinus endothelium. Vascular clamps and aneurysm clips should be avoided as much as possible because they may injure the sinus walls and afferent veins.

Bridging veins, especially in the rolandic outflow area, should be preserved by dissecting them free from adjacent brain and tumor.

Venous reconstruction is performed using patches (**Fig. 37.3**) or bypasses (**Figs. 37.5** and **37.6**) with two hemirunning sutures (Prolene 8–0, Laboratoire ETHNOR, Neuilly/Seine, France). Although autologous vein would appear as the most suitable material for use as a patch, vein harvesting seems excessive for patching purposes only. The locally situated dura mater, fascia temporalis, and pericranium can be used; however, we favor the use of fascia temporalis when possible because it is strong, thin, and smooth and has a more rigid structure. When a bypass is performed, the graft should not be compressed by any surrounding structure, and measures to decrease the intracranial pressure should be taken to prevent it from kinking or stenosis.[4]

Postoperative Care

To facilitate bypass patency after surgery, blood pressure, volume, and viscosity must be carefully monitored. Heparin therapy (two times normal value) is recommended for at least 21 days to prevent the reconstructed sinus from becoming clotted, and 3 months of antiplatelet therapy is recommended to allow sinus wall endothelialization.

Surgery According to the Degree of Sinus Invasion (Fig. 37.7)

Type I: Excision of outer layer, leaving a clean and glistening dural surface, and coagulation of dural attachment

Type II: Removal of intraluminal fragment through the recess, then repair of the dural defect by resuturing the access or by closing it with a patch, or, provided it is not stenosing, sealing the opening with an aneurysm clip

Type III: Resection of the sinus wall and repair with a patch

Type IV: Resection of both invaded walls and reconstruction of the two resected walls by a patch

Type V: The opposite wall to the tumor side is free of tumor. Thus we think that is preferable to reconstruct with a patch the two invaded walls after their resection, rather than to perform a bypass. This type can be distinguished from type VI only by direct surgical exploration of the sinus lumen.

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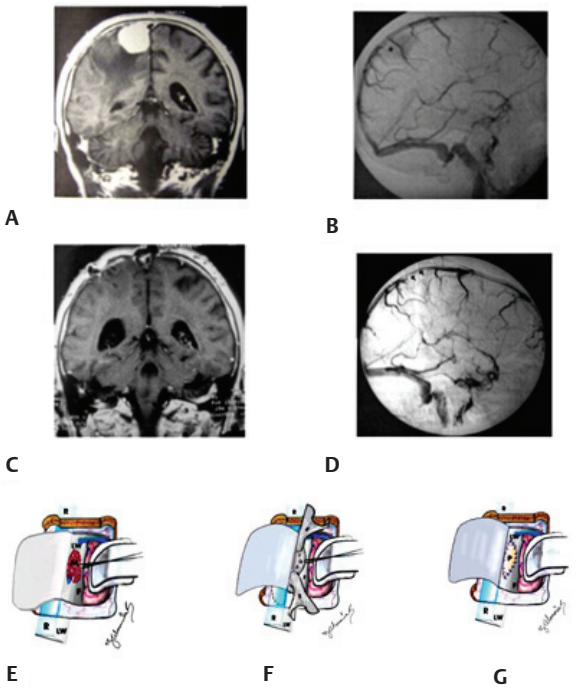


Fig. 37.3 Upper part: type III parasagittal meningioma in midthird of superior sagittal sinus (SSS). **(A)** Preoperative coronal gadolinium-enhanced T1-weighted magnetic resonance imaging (MRI) showing a right middle third parasagittal meningioma with type III invasion of the lateral wall of the sagittal sinus. **(B)** Preoperative venous-phase angiogram, lateral view, demonstrating a patent sinus and tumor blush (*). **(C)** Postoperative coronal gadolinium-enhanced T1-weighted MRI exhibiting no evidence of tumor remnant. The lateral sinus wall was resected and patched. **(D)** Postoperative venous-phase angiogram, lateral view, showing sinus patency (*arrows*). Lower part: Steps of the patching technique. **(E)** Lateral view of the sagittal sinus (right side) after removal of the tumor outside the sinus; the invaded lateral mass is visible (type III). **(F)** The invaded portion of the wall has been resected; venous bleeding is controlled by pledgets of Surgicel inserted in the sinus lumen (proximal and distal). **(G)** Venous reconstruction performed using an autologous patch (fascia temporalis in this case). F, falx cerebri; LW, lateral wall of SSS; R, roof; P, patch; S, Surgicel; O, venous ostia in the contralateral wall.

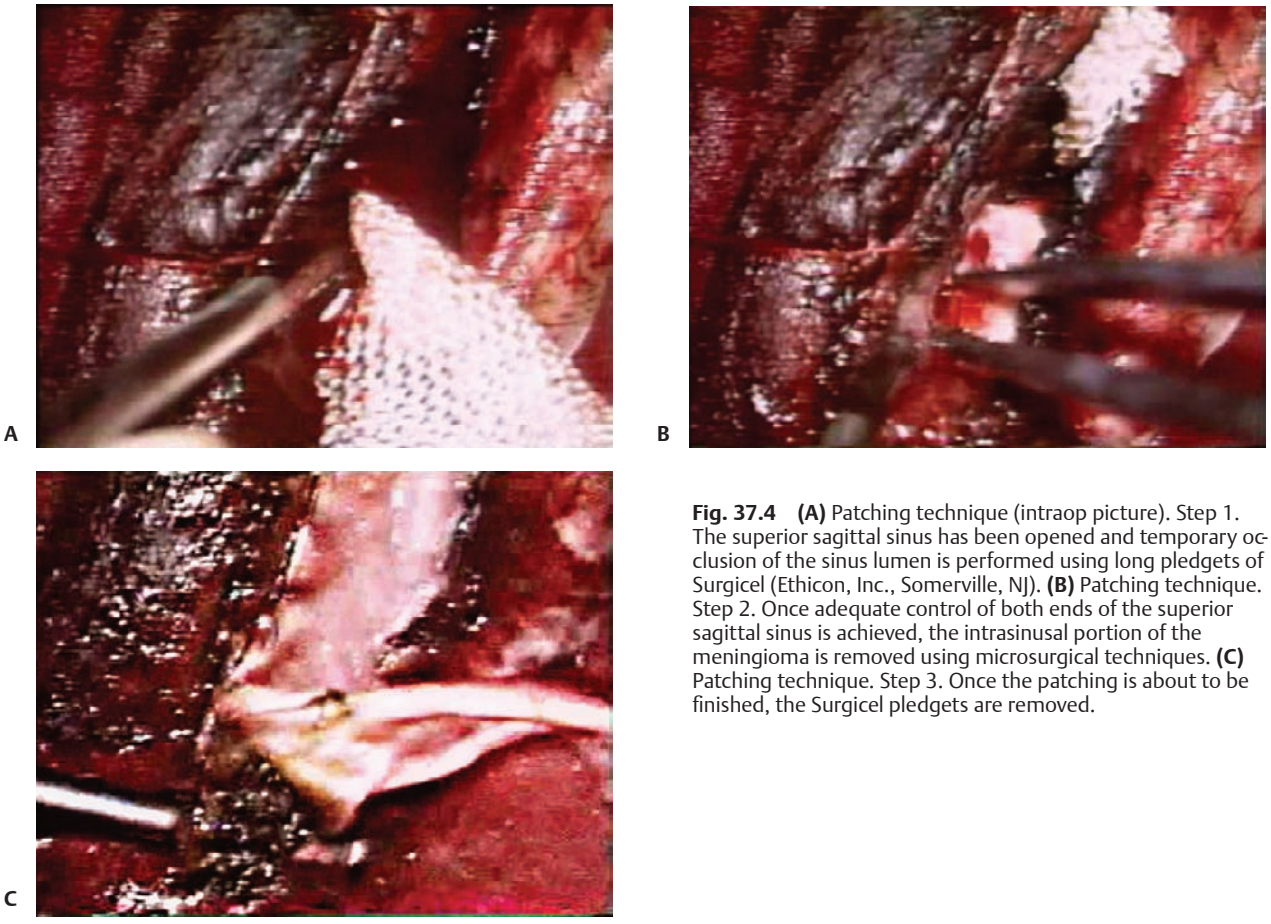


Fig. 37.4 **(A)** Patching technique (intraop picture). Step 1. The superior sagittal sinus has been opened and temporary occlusion of the sinus lumen is performed using long pledgets of Surgicel (Ethicon, Inc., Somerville, NJ). **(B)** Patching technique. Step 2. Once adequate control of both ends of the superior sagittal sinus is achieved, the intrasinusal portion of the meningioma is removed using microsurgical techniques. **(C)** Patching technique. Step 3. Once the patching is about to be finished, the Surgicel pledgets are removed.

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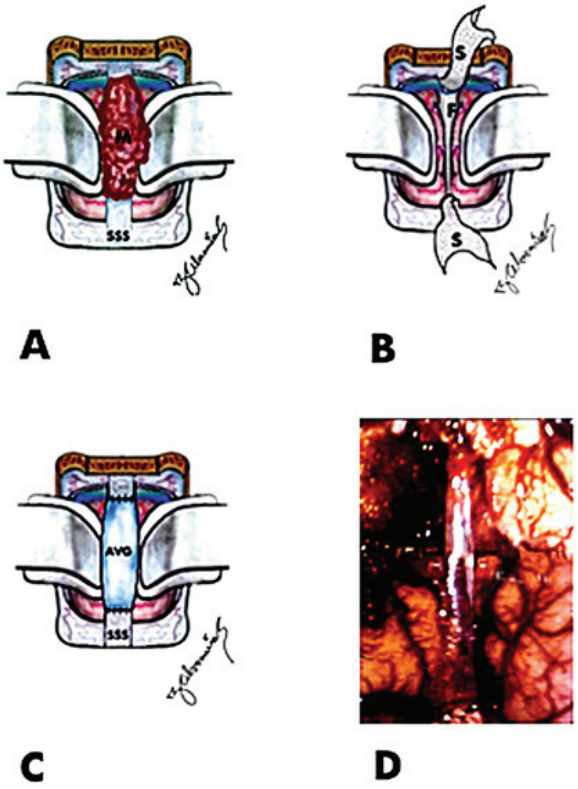
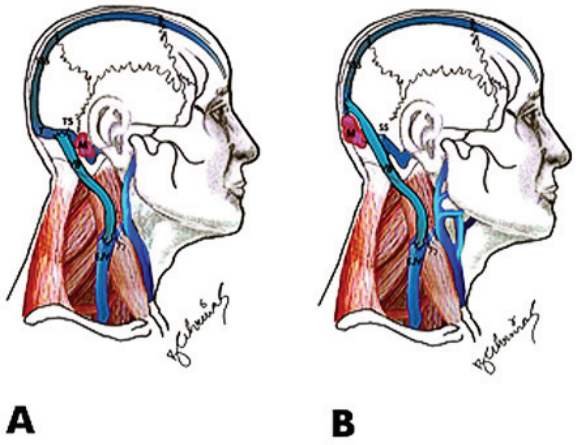


Fig. 37.5 Type VI parasagittal meningiomas totally invading and occluding the anterior half of the posterior third of the superior sagittal sinus. **(A)** Drawing of the tumor. **(B)** Total removal of the meningioma together with the totally invaded portion of the sinus and proximal and distal temporary sinus lumen occlusion with Surgicel pledgets (Ethicon, Inc., Somerville, NJ). **(C)** Venous circulation restored with a venous autologous graft harvested from the (external) jugular vein and mounted as a bypass of end to end type at both anastomoses. **(D)** Operative microsurgical view (taken from the intraoperative videotape) after completion of the venous bypass. Bypass is patent and circulating (*arrow flow*).



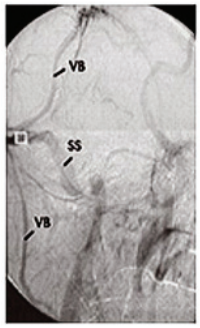


Fig. 37.6 Sinojugular bypass: schematic drawing. **(A)** A transverse-jugular bypass between right transversal sinus and external jugular vein, exposed superficially to sternocleidomastoid and trapezius muscles (for a bilaterally occluded sigmoid sinus). **(B)** A sagittojugular bypass between the sagittal sinus and the external jugular vein (for an occluded sagittal sinus–torcular at its posterior third by meningioma). **(C)** Postoperative control (at 2 weeks) by digital subtraction angiography of a patent sino-(sagittal) jugular (external) bypass performed with a greater internal saphenous vein graft (*arrowheads*), right side, in a patient with a totally occluded (type VI) sagittal sinus (posterior third), by a meningioma, and suffering severe intracranial pressure syndrome. J, external jugular vein; VB, venous bypass patent. With permission from Sindou M, Alvernia JE. Results of attempted radical tumor removal and venous repair in 100 consecutive meningiomas involving the major dural sinuses. J Neurosurg 2006;105(4):514–525.

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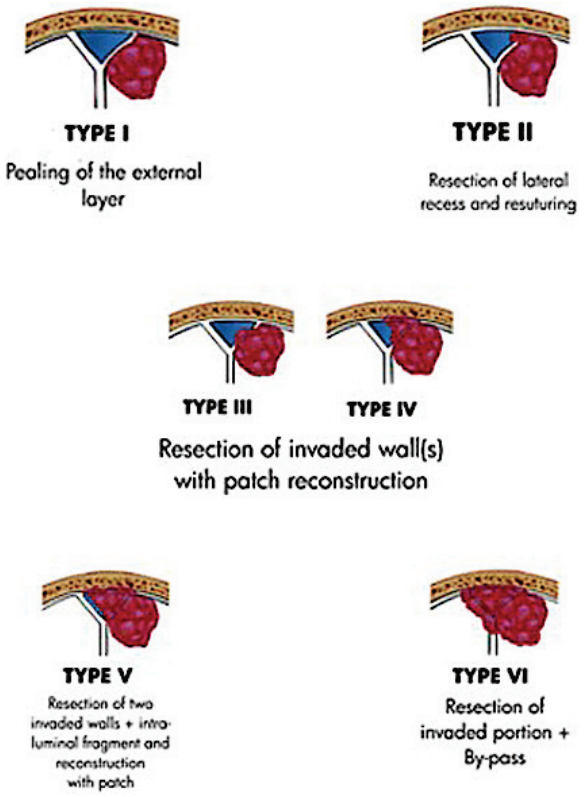


Fig. 37.7 Surgical strategies according to the degree of sinus invasion by the meningioma. With permission from Sindou M, Alvernia JE. Results of attempted radical tumor removal and venous repair in 100 consecutive meningiomas involving the major dural sinuses. J Neurosurg 2006;105(4):514–525.

Type VI: Removal of the involved portion of the sinus and restoration by venous bypass. The site of the bypass is on the sagittal sinus for meningiomas involving the sagittal sinus (SS), between the SS and the external jugular vein for meningiomas totally occluding the posterior third and the torcular, between the transverse sinus and the external jugular vein for meningiomas involving the transverse sinus.

Discussion

Surgical Venous Injury

All authors dealing with meningiomas invading dural sinuses agree on the importance of preserving the afferent (bridging) veins to the sinus, especially the ones of the central group in the middle third portion of the sagittal sinus, as well as those located at the transverse sinus and corresponding to the inferior cerebral vein of Labbé.

Avoiding the interruption of a partially occluded sagittal sinus is a matter of strong consensus. The safety of resecting a totally occluded portion of a sinus, although classically accepted, is disputable. Brain swelling, venous infarction, and cerebrospinal fluid collection may occur when venous collateral circulation is impaired. In the study by Hoessly and Olivecrona, which included 196 parasagittal meningiomas treated without venous reconstruction, morbidity amounted to 12.3%; half of the cases involved the sequelae of venous injury. Their 10% mortality corresponded to 14 (12,8%) of the 109 tumors located in the middle, three (9.7%) of the 31 lesions in the posterior third, and three (5.3%) of the 56 lesions in the anterior third.[5] In Bonnal and Brotchi's study including 21 cases, one patient (4.8%) died after an en bloc resection of the tumor mass without venous restoration.[6] In a recent article documenting the cooperative experience of eight surgeons and the grouping of 108 cases, DiMeco and colleagues mentioned the occurrence of severe brain swelling in three (10%) of the cases treated with en bloc resection without venous reconstruction. Also, persistent subgaleal fluid collection likely corresponding to a default in cerebrospinal fluid (CSF) reabsorption or persistent postoperative high intracranial pressure from venous origin was observed in 11 of their patients (10%).[7] In our series, morbidity and mortality related to venous damage without venous flow reconstruction were estimated at 8% and 3%, respectively.[1] Perhaps these patients would have had a better outcome if they had undergone venous repair. Perhaps shaving of the tumor outside the sinus with secondary stereotactic radiation would have been harmless. We really do not know

Dural Sinus Reconstruction

Reconstruction of the venous system is not new. Various techniques of dural sinus repair were reported long ago.[8–30]

First Bonnal and Brotchi[10,11] and then Hakuba and collegues,[13–16] Bederson and Eisenberg,[9] Sekhar and associates,[21] Steiger and collegues,[30] and Schmid-Elsaesser et al[20] reported satisfactory experiences patching the venous sinuses with pieces of dura or autologous venous graft. In our series, 13 of the 15 repairs performed with dura or fascia and confirmed angiographically were patent.[1] Harvesting autologous veins, theoretically the best material for patching the venous system, actually seemed unnecessary simply for patching. According to our experience, the most appropriate material is the thin and glossy fascia temporalis. For performing bypasses, the internal saphenous vein for a long graft (more than 10 cm) and the external jugular vein for a short graft (less than 10 cm) were used in 12 patients. Eight (72,7%) of the 11 angiographically assessed bypasses were patent. When a synthetic graft (namely, a Gore-Tex tube) was used (six cases), none remained patent, despite the use of anticoagulation, and thus we do not recommend synthetic grafts of the cerebral venous system.

When temporary occlusion of the sinus is required, the use of small pledgets of Surgicel within the lumen and at the ostia of the tributary afferent veins makes the process simple. This technique has been found much more preferable than using aneurysms clips or balloons. In fact, an-

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eurysm clips or even temporary clamps are too aggressive for sinus walls, and balloons, along with silicone tubes used as shunts, are not often useful due to the presence of septa within the sinus lumen, especially in the middle portion of the sagittal sinus. Moreover, all entail the risk of damaging or even avulsing the endothelium, which is known to be susceptible to secondary thromboses.

Whether a bypass is justified every time a total resection is attempted or whether it should be used only in selected cases with proven high venous pressure remains a debatable issue. The decision can be made based on intraoperative measurement of the intrasinusal pressure as recommended by Schmid-Elsaesser et al[20] and Bederson and Eisenberg.[9] We tend to favor the routine use of venous bypass in these situations, especially given that performing a bypass in the extradural space involves little additional risk.

The fact that all of the patients who had a graft that thrombosed (with the exception of one with a Gore-Tex tube) remained asymptomatic does not mean that venous reconstruction was useless. One can postulate that the bypass gave time for compensatory venous pathways to develop until occlusion occurred. Importantly, all three patients who died of brain swelling had meningiomas with type VI sinus invasion that had been totally resected without venous flow restoration.

Conclusion

The low recurrence rate in our series supports resecting not only the tumor portion outside the sinus but also the fragment invading the sinus. Ultimately, a decision must be made after weighing the benefits and risks. If an attempt at complete removal is decided upon, venous reconstruction is mandatory when the sinus is incompletely occluded. We find it potentially useful to restore the flow that might be compromised by impaired compensatory channels, even in cases of complete sinus occlusion. Achieving complete removal requires opening the sinus, exploring its lumen, and (temporarily) interrupting its circulation; this process can be easily performed with pledgets of Surgicel. Resected walls should be repaired with patching; a graft harvested from adjacent dura, fascia lata, or preferably the thin fascia temporalis appears adequate. For performing bypasses, only autologous grafts should be used: the external jugular vein for short grafts and the internal saphenous vein for longer ones. Postoperative anticoagulation is mandatory for at least 3 months until reendothelialization occurs; this strategy has not increased hemorrhagic complications in our experience.

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Chapter 38

Application of Endoscopy in the Management of Meningiomas

Charles Teo and Lawrence S. J. Choi

Introduction

Meningioma surgery often places a great emphasis upon achieving a complete resection due to the potential curability or long-term survival advantage associated with the degree of resection. In deep-seated lesions within the cranial base, posterior fossa, or ventricles, the balance between degree of resection and risk of surgical access to such areas often means that traditional microscopic skull base approaches do not always achieve a complete or near-complete degree of resection. This may result from a calculated risk assessment of potential damage to neurovascular structures but may also be due to lack of visual access in terms of restricted line of sight and lack of illumination of areas where brain retraction is not an option.

The advantage that endoscopy brings to neurosurgery, and in particular meningioma resection, is that it allows a surgeon to perform minimally invasive neurosurgery in areas that are risky and difficult to access, such as the ventricles and cranial base, without necessarily compromising the degree of resection. The natural advantages that endoscopy brings to surgical approach to a deeply situated meningioma include the ability to angulate the field of view within a narrow line of approach, improved illumination, and superior up-close visualization of surrounding neurovascular structures.

There are certain limitations to the use of the endoscope. The lack of a true three-dimensional image with endoscopy and the absence of any visualization of structures "behind" the endoscope lens make navigation in tight spaces and passing of instruments difficult. Furthermore, using an endoscope sometimes limits the surgeon in utilizing both hands for operating, thus requiring either reliance on an assistant or employment of specialized instrumentation.

A steady growth of interest in neuroendoscopy is currently producing an exhaustive series of operative atlases, and new courses are available worldwide to teach the operative art of neuroendoscopy. Industrial support is also producing innovative new equipment to overcome the natural disadvantages of endoscopy. Therefore, despite the steep learning curve and relative infancy of this technique, all neurosurgical trainees are strongly encouraged to train in the optimization of surgical resection of meningiomas by employment of all available technological aids.

Instrumentation in Neuroendoscopy

The surgeon's intention to perform minimally invasive surgery, as a general rule, will benefit from the availability of frameless stereotactic technology by allowing a surgeon to plan the least traumatic approach. Apart from planning the ideal trajectory to a lesion, it further allows a surgeon to identify and preserve vital structures without having to increase surgical exposure.

Figure 38.1A,B shows two variants of scopes commonly used within neuroendoscopy. Larger rigid scopes measuring between 2 and 6 mm in diameter are typically employed for either endoscopically assisted or endoscopically controlled surgery. In endoscopically assisted surgery, the surgeon utilizes traditional microsurgical techniques but uses the endoscope to assist in the operation. In endoscopically controlled surgery, the endoscope is the sole means of visualization but the surgeon places the instruments down the surgical corridor, not down a sheath. Pure endoscopic surgery refers to burr hole access with a sheath passed into the cranium through which the surgeon passes both the scope and the instruments. These scopes are smaller, between 0.9 and 2 mm in diameter. They are more fragile, and care

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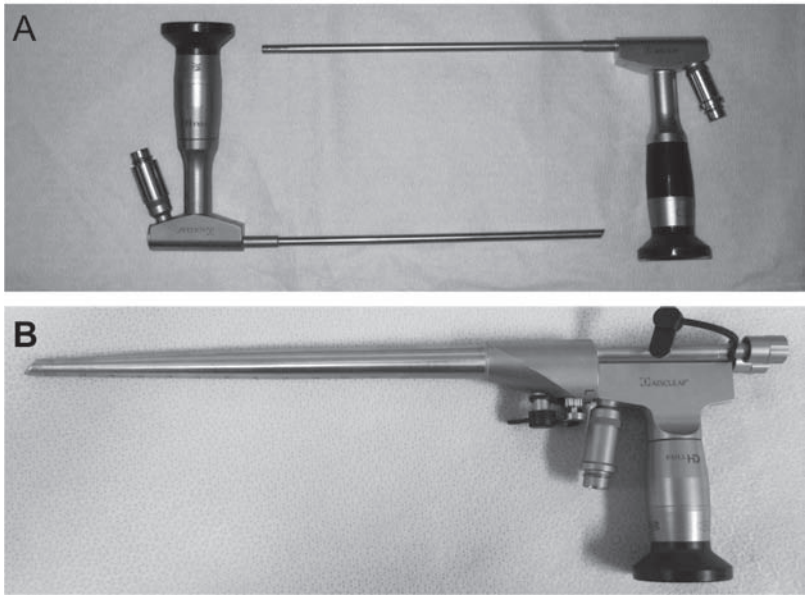


Fig. 38.1 Examples of endoscopes used in neurosurgery. **(A)** A 4 mm endoscope, used for all endoscopically controlled and endoscopically assisted techniques, 0 and 30 degree scopes featured. **(B)** A 2 mm endoscope within a ventricular sheath, used for pure neuroendoscopic techniques.

must be taken by operating staff, surgical attendants, and sterilizing units to prevent fracturing the internal glass construction. Instruments vary by the technique employed. For pure endoscopy, the instruments need to fit down narrow working channels and therefore are quite restricted in their size and versatility. This technique suffers from an inability of the surgeon to operate using two instruments at angles to each other because the channels in most sheaths run parallel. Endoscopically assisted surgery requires instruments that are angled at the ends to fully utilize the scope's ability to look around corners. It is pointless to be able to see behind an immovable structure unless one can also operate around that structure. Many instrument companies have identified this impediment and are now producing angled and flexible tools. The most common example of endoscopically controlled surgery is the endonasal approach to the skull base. With the popularity of this technique has come a plethora of instrument sets designed specifically for this application. The general principles behind these instruments are that the tools should not be bayoneted and that hybrid tools should be encouraged. The bayonet handle on microsurgical instruments was designed to keep the surgeon's hand out of the line of sight. This is not a problem when the surgeon is viewing the operation on a monitor. Furthermore, rotation of an instrument with a bayonet handle results in clashing of the tool against the endoscope when placed down the nostril. During many portions of the endonasal approach, the surgeon is operating with the scope in one hand and a tool in the other. If the tool can have more than one function, then it compensates for the negative impact of being a one-handed surgeon. Such "hybrid" instruments that are currently available include suction/bipolar, suction/monopolar, suction/curettes, and suction/endoscopes. We believe that the development of the videoscope (miniaturized cameras) will inspire a tremendous evolution of instrumentation.

Techniques

Pure Neuroendoscopy

The use of an endoscope through a burr hole opening as the sole means for resection of a meningioma would be extremely rare. There would be very few applications for this technique apart from small and symptomatic intraventricular tumors. Intraventricular tumors that are situated away from the cerebral aqueduct and foramen of Monro are often large and are fed by choroidal vessels that are medial and consequently "hidden" by the tumor mass. Due to the typically high vascularity of a meningioma, it is essential that the vessels of the stalk are readily and initially accessible. However, a meningioma of any significant size would likely restrict or preclude access to this pedicle, and render hemostasis impossible. Therefore we do not recommend removing these large meningiomas using pure endoscopic techniques.

If the tumor is small and blocking the cerebrospinal fluid (CSF) pathways at the level of the foramen of Monro, resulting in ventriculomegaly, then the technique by which one would remove such a lesion is very similar to removal of a colloid cyst. The burr hole is placed according to the desired trajectory. If the tumor is within the lateral ventricle, then its blood supply is from the choroid plexus, and for early devascularization it should be approached posterosuperiorly. In this case the burr hole is made ~13 cm behind the nasion and 3 to 5 cm from the midline. If the tumor is arising from the third ventricle, then its blood supply is coming from the roof and the trajectory is therefore more anterolateral. In this case the burr hole is made ~8 cm behind the nasion and 7 cm from the midline through the nondominant hemisphere. Once the tumor is devascularized, it can be removed either in piecemeal fashion or, if small enough, en bloc.

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Endoscopically Assisted

The nature of meningiomas to grow at the skull base and their relative biological inertia allow them to attain considerable size. This also causes vessels and nerves to be intimately involved in their capsules. It also means that many occupy more than one intracranial compartment. All these features make total extirpation of these tumors difficult through a standard craniotomy. Hence complex skull base techniques evolved in the 1980s to allow the surgeon to remove skull base meningiomas totally and with minimal collateral damage and morbidity. However, these techniques require fellowship training, are time consuming, and may create morbidity from the approach itself, and, in reality, most nonuniversity-based neurosurgeons perform these cases infrequently. Endoscopically assisted surgery allows most cranial surgeons to optimize removal of many skull base meningiomas without the need to perform extensive bony removal or dual approaches. It does so by helping the surgeon to see important neurovascular structures before, during, and after tumor removal. It also allows visualization through the tentorial incisura for petroclival tumors or over the sphenoid wing for all tumors of the anterior cranial base that extend into the middle cranial fossa. Tumors that extend into foramina, such as those in the cerebellopontine angle that may grow into the internal auditory meatus and those of the anterior cranial fossa that grow into the optic canal, may be more completely and safely removed using the endoscope to look around corners (**Figs. 38.2A–C** and **38.3A–C**). Of course, there will be tumors that cannot be removed through standard or minicraniotomies, even with the use of endoscopes. Some of these patients will need more extensive bony removal for complete resection. When the scope is used to assist in the removal of a meningioma, the majority of the mass is removed using a microsurgical technique, and the scope is placed into the cavity at any stage during the operation when enhanced visualization is considered necessary. Although scopes come in 30, 45, and 70 degree angles, in reality, operating at angles greater than 30 degrees is awkward and dangerous.

Endoscopically Controlled

The use of endoscopy to control visualization exclusively is epitomized by endonasal approaches to the skull base. There are very few other applications where the endoscope may replace the microscope entirely. That is not to say that the endoscope cannot be used in conjunction with the microscope, but the following paragraphs will make clear that the endoscope is uniquely applicable to this indication. Traditional skull base approaches reduce the need to retract brain, improve the line of sight to the tumor, aid in achieving proximal vessel control, and expose flaps for later repair of dural defects. Endoscopically controlled surgery performed through the nostril(s) or rarely the mouth has all of these benefits and more. There is no need for a skin incision, illumination is better, magnification is enhanced, access is more rapid, paramedian structures are better seen, and angled endoscopes offer views that are impossible with the microscope.

To date, most studies on endoscopic endonasal approaches to midline skull base lesions, in particular pituitary adenomas, report comparable or even favorable surgical outcomes to conventional microsurgical approaches.[1–3] However, complications such as CSF fluid leakage and central nervous system (CNS) infections have been reported to be higher. Some experienced centers have reported a 40% rate of postoperative CSF leaks[4] after complex endonasal surgery. We would echo this early experience when our CSF leakage rate was as high as 60% and infection was 15%. Most experienced endonasal centers, including our own, are claiming improvement in these rates, but until there are more peer-reviewed studies published, we should remain concerned about this serious complication. However, a series of 90 patients from Brown and colleagues demonstrates the significantly low risk of postoperative CNS infections from endoscopic endonasal skull base surgeries with the use of a single perioperative antibiotic (cefazolin with the use of intranasal gentamicin spray).[5]

Currently, there are limited data comparing the effectiveness of endoscopic endonasal resections of meningiomas versus microsurgical techniques. Gardner and colleagues describe a mixed endoscopic and microscopic series of 35 anterior cranial fossa meningiomas, whereas Divitiis and colleagues reported a series of 51 tuberculum sella meningiomas resected via transcranial (n = 43) and endoscopic endonasal approaches (n = 7) with a further report from the same group reporting an additional four olfactory groove meningiomas.[4,5,7] Both groups agree that the endoscopic endonasal route has an acceptable complication risk compared with that of a traditional open craniotomy and provides a minimally invasive option to resection. Importantly, both groups agree that the degree of resection is not influenced by approach, but rather by patient and tumor factors.

Review of our own series of skull base meningiomas, removed via an endonasal approach, is revealing. The number of incomplete resections (28%) appeared significantly higher than a similar cohort of patients who underwent transcranial approaches (15%). However, when those patients with petroclival meningiomas were singled out, the rate of neurological complications in those patients who had an endonasal approach (0%) was clearly better than that in those who had a traditional transcranial approach (75%). The long-term consequences of subtotal removal of meningiomas have been underscored by many authors, and consequently, the role of the endonasal approach to meningiomas will need honest evaluation in future publications.

Basic Tenets of the Endonasal Approach to Skull Base Meningiomas

Close collaboration with an ear, nose, and throat (ENT) surgeon is important for extradural surgical exposure of the cranial base. A wide corridor for passage of en-

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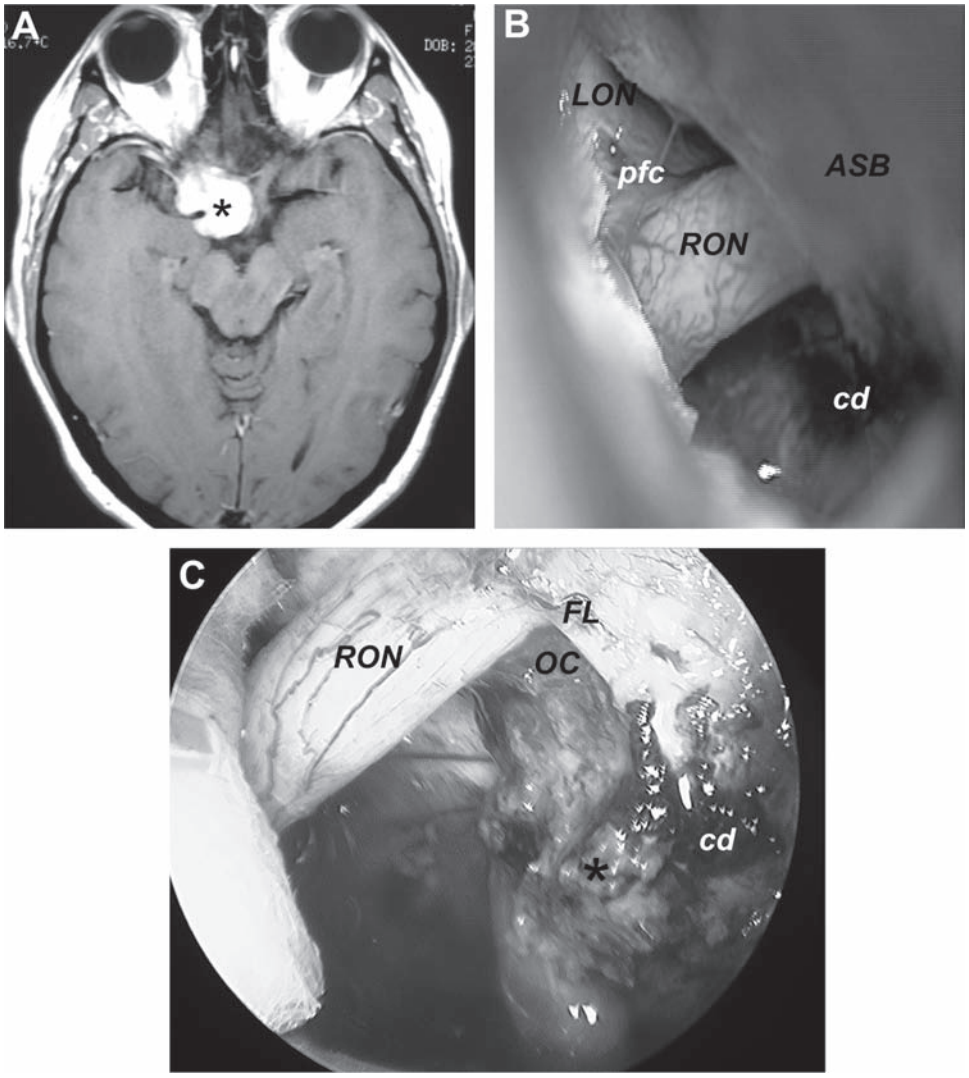


Fig. 38.2 Endoscopic assisted resection of an anterior clinoid meningioma. **(A)** Magnetic resonance imaging T1 with gadolinium injection. **(B)** Surgical microscopic view postdebulking via eyebrow craniotomy, no visible tumor. **(C)** 30 degree endoscopic view of the optic canal, residual tumor seen within the optic canal. *, tumor; ASB, anterior skull base; cd, cauterized dura; FL, falciform ligament; LON, left optic nerve; OC, optic canal; pfc, prefixed chiasm; RON, right optic nerve.

doscopic instrumentation typically requires a complete opening of the face of the sphenoid sinus with or without middle turbinectomy. Before this is done, consideration should be given to the potential need for a vascularized nasoseptal mucosal flap to repair the dural defect. If considered necessary, it should be harvested before the sphenoidotomy. With very anterior tumors, such as olfactory groove meningiomas, it may not be necessary to expose the sphenoid sinus. Almost all other skull base tumors will require this initial step. Once into the sinus it is imperative to identify certain landmarks. The midline is usually identified by the vomer. The intersinus septa are unreliable. Other important landmarks are seen in **Fig. 38.4**; the optic and carotid impressions, the medial and lateral opticocarotid recesses, the pituitary fossa, the clival recess, and the planum sphenoidale.

Exposure of any or all of these structures is dictated by the location of the tumor. Following are some general principles of endoscopic skull base drilling:

1. Copious venous bleeding may be quite disconcerting. It should be expected and preparation with hemostatic agents will reduce blood loss. More recent hemostatic matrices, such as Floseal (Baxter Healthcare Corp. Deerfield, IL) are extremely effective.
2. Section of the superior intercavernous sinus should be controlled with access gained above and below before transection.
3. Beware of dehiscence of bone over the carotid artery, especially the paraclinoid part of the siphon and the distal paraclival (vertical) carotid.
4. Ensure that the drill and other instruments are not hitting more superficial bony protuberances.
5. Use a two-handed technique whenever possible by having your assistant hold the endoscope. Although we dislike the use of scope holders, they are necessary if good assistance is unavailable.

Reconstruction after endonasal removal of an intradural tumor is an essential aspect of this operation. Al-

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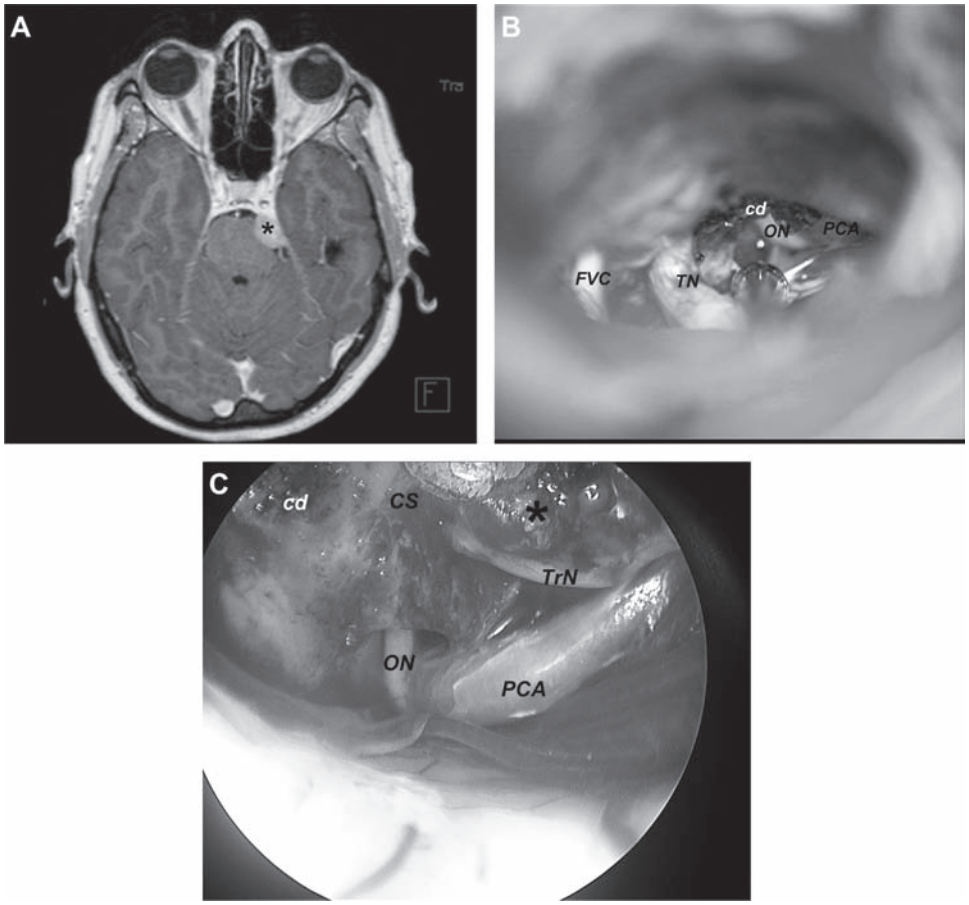


Fig. 38.3 Endoscopic assisted resection of a posterior fossa meningioma. **(A)** Magnetic resonance imaging T1 with gadolinium contrast, lesion abutting the trigeminal nerve extending to the tentorium. **(B)** Microscope view after debulking of tumor via retrosigmoid approach, view limited in structures above the oculomotor nerve (i.e., the tentorium) due to restriction to line of sight. **(C)** Endoscopic view within the posterior fossa reveals additional tumor within the tentorium closely associated with the trochlear nerve. *, tumor; cd, cauterized dura; CS, cavernous sinus; FVC, facial-vestibular complex; PCA, posterior cerebral artery; TN, trigeminal nerve; TrN, trochlear nerve.

though many effective techniques have been described, there are several principles that are common to all:

1. Multilayered closure is more effective than a single layer.
2. Vascularized tissue is better than nonvascularized.
3. Autologous graft is better than synthetic.
4. Counterpressure, either temporarily or permanently, is essential. An example of temporary pressure is a balloon catheter inflated with normal saline and positioned in the nasopharynx to apply pressure to the construct. An example of permanent pressure is a titanium plate placed against the construct and held in place inside the edges of the bony defect.
5. Glue or sealant products may be helpful.
6. Lumbar CSF drainage with a catheter is discouraged.

To underscore some of the important technical nuances of endoscopically controlled removal of meningiomas of the skull base, the following descriptions are provided. The authors also refer readers to the article produced by Gardner and colleagues[4] in detailing the extended endoscopic endonasal approach to anterior cranial fossa meningiomas.

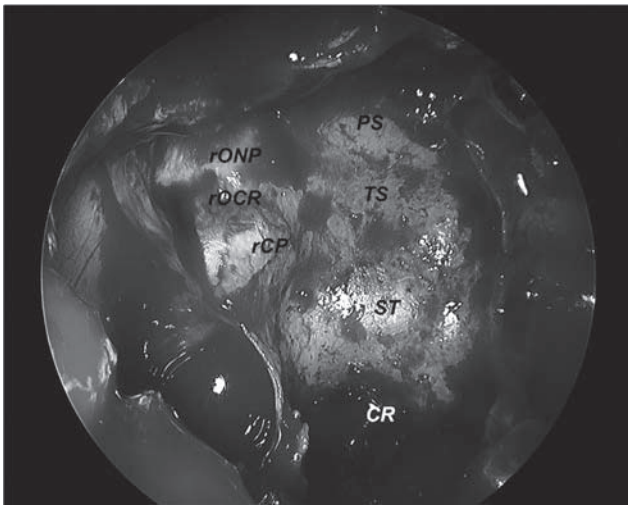


Fig. 38.4 Anatomy of a standard endonasal approach to the pituitary fossa. Anatomy to the left of midline is obscured by blood. CR, clival recess; PS, planum sphenoidale; rCP, right carotid protuberance; rOCR, right opticocarotid recess; rONP, right optic nerve protuberance; ST, sella turcica; TS, tuberculum sellae.

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Olfactory Groove Meningiomas

Olfactory groove meningiomas are typically very amenable to endonasal approaches (**Fig. 38.5A–E**). Located above the cribriform plate, a rostral extension of the basic endonasal approach involves removal of the ethmoidal air sinuses. The cribriform plate and crista galli are then removed to expose the dura of the midline anterior cranial fossa floor (**Fig. 38.5B**). This provides a wide bilateral working space extending from the frontal sinus anteriorly to the planum sphenoidale posteriorly and laterally to the superomedial wall of the orbits.

This approach allows the anterior and posterior ethmoidal arteries to be coagulated before durotomy and debulking, which in turn assists in devascularizing the tumor. The basic tenets of microsurgical removal of tumors apply to endoscopy. After initial internal debulking, the edges of the tumor are defined (**Fig. 38.5C–D**). A plane between tumor capsule and normal brain is then identified, developed, and maintained as far as possible (**Fig. 38.5D**). With large tumors that extend posteriorly to the planum and beyond, extreme care needs to be taken when dissecting the dome and posterior wall of the tumor because branches of the anterior cerebral artery may be intimately involved.

At no time should the capsule of the tumor be pulled without direct visual confirmation that surrounding neurovascular structures are no longer attached. This is mostly achieved using 0 degree scopes but may be enhanced with the use of 30 degree scopes.

The major advantage of such an approach lies in the avoidance of retraction of the juxtaposed brain. There are perceived neuropsychological benefits to avoidance of brain retraction in resection of olfactory groove meningiomas. However, this has yet to be validated in detailed neuropsychological testing. The authors believe the main advantages to this approach are the rapid and comprehensive access, the avoidance of brain retraction, the painless and rapid recovery, and the obvious cosmetic outcome.

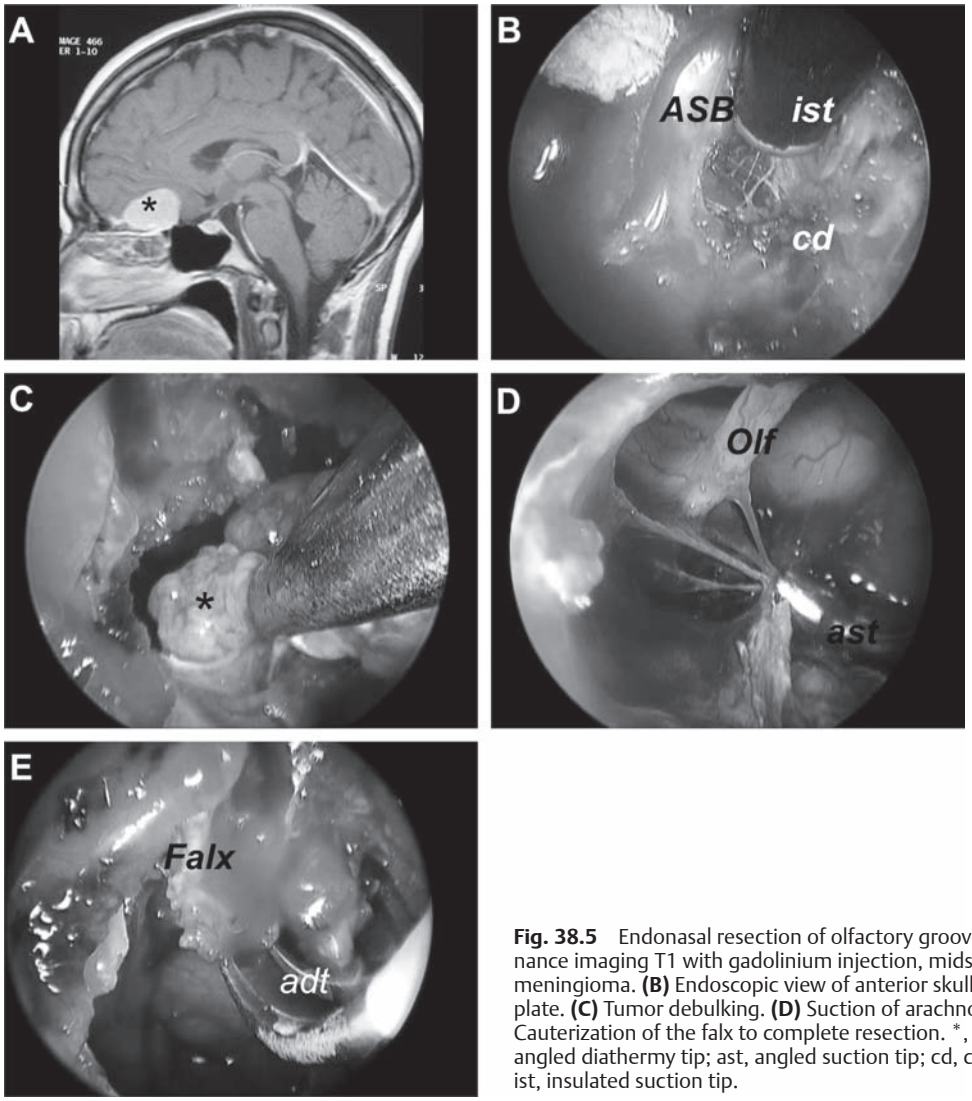


Fig. 38.5 Endonasal resection of olfactory groove meningioma. **(A)** Magnetic resonance imaging T1 with gadolinium injection, midsagittal plane reveals olfactory groove meningioma. **(B)** Endoscopic view of anterior skull base postremoval of cribriform plate. **(C)** Tumor debulking. **(D)** Suction of arachnoid adjacent to neural structures. **(E)** Cauterization of the falx to complete resection. *, tumor; ASB, anterior skull base; adt, angled diathermy tip; ast, angled suction tip; cd, cauterized dura; olf, olfactory nerve; ist, insulated suction tip.

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Tuberculum Sellae Meningiomas

Tuberculum sellae meningiomas are much more difficult to manage due to their frequent involvement with the optic nerves and anterior cerebral artery (ACA) complex (**Fig. 38.6A–D**). Despite the level of endoscopic experience of our team and the recent literature describing safe resection of tuberculum sellae meningiomas,[4,5,7] the authors prefer to resect these difficult lesions using an endoscopically assisted keyhole subfrontal craniotomy through an eyebrow incision. This allows for a more controlled and safer dissection of the A1–2 complex and the optic nerves that are not uncommonly circumferentially surrounded by densely adherent tumor (**Fig. 38.6D**).

In certain cases, where the tumor may have expanded inferiorly into the sphenoid sinus or within the inferomedial portion of the optic canal, a staged endonasal operation targeted toward these structures is recommended due to a more direct line of approach (**Fig. 38.6E**).

Petroclival Meningiomas

Petroclival meningiomas are treacherous lesions whether approached from above or below. The general principle of approaching a tumor from the side that avoids retraction of nerves and gives early control of major vessels does not readily apply to these tumors. By their location, often multiple cranial nerves, the cavernous sinus, paraclival and cavernous carotid, basilar and posterior cerebral arteries, and many small but vital perforating vessels are all involved (**Fig. 38.7D).** Total extirpation therefore is difficult and has only been achieved in 50% of those performed by the senior author. All of these tumors were small with very little cavernous sinus involvement.

The clivus can be accessed via an inferiorly extended transsphenoidal approach (**Fig. 38.7A–F**). The exposure and identification of the carotid prominence serves as a superior and lateral anatomical limit of

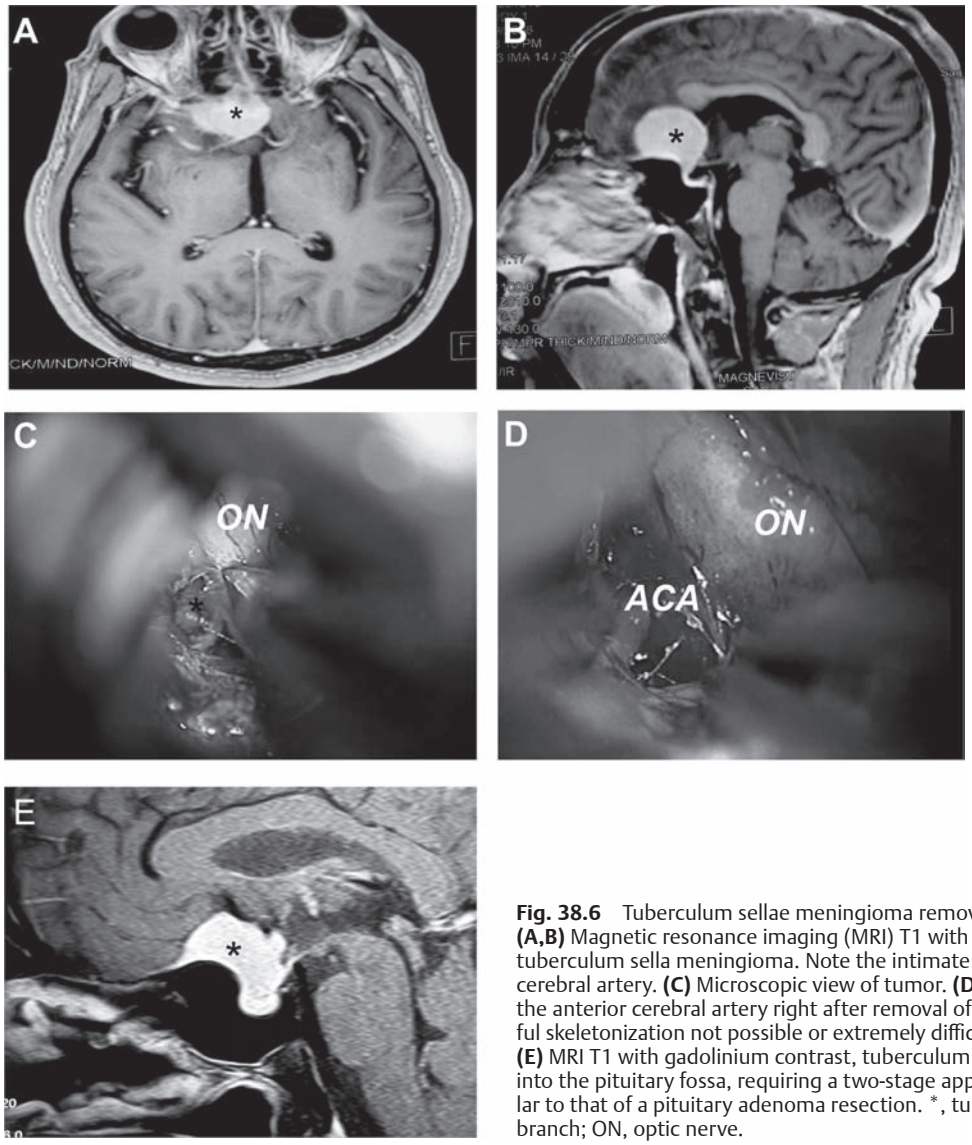


Fig. 38.6 Tuberculum sellae meningioma removed via eyebrow craniotomy. **(A,B)** Magnetic resonance imaging (MRI) T1 with gadolinium contrast revealing tuberculum sella meningioma. Note the intimate relation of the tumor to the anterior cerebral artery. **(C)** Microscopic view of tumor. **(D)** Intimate relation of the tumor to the anterior cerebral artery right after removal of tumor shown in **(C)**, requiring careful skeletonization not possible or extremely difficult via an endonasal approach. **(E)** MRI T1 with gadolinium contrast, tuberculum sellae meningioma with extension into the pituitary fossa, requiring a two-stage approach, the endonasal approach similar to that of a pituitary adenoma resection. *, tumor; ACA, anterior cerebral artery branch; ON, optic nerve.

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the craniotomy. The clivus over the lesion can then be drilled superior to inferior to expose the dura. Cautery of the dura is performed before a linear durotomy to reveal the bulk of the tumor, which should be gently suctioned. Care is observed during suction to prevent traction and subsequent damage to the underlying basilar artery and cranial nerves (**Fig. 38.7E**). The neurological sequelae of carefully chosen cases in endoscopic resection of clival lesions are rewarding, with Carrabba and colleagues describing a 79% improvement rate of patients with neurological symptoms.[8]

Conclusion

The application of an endoscope to the optimal resection of meningiomas cannot be overemphasized. Under specific circumstances it provides the sole means of visualization, and in other cases it may assist in safer dissection and more complete removal. The result is improved neurological outcomes and, in those circumstances where complete removal is achieved, longer survival. The role of endoscopically controlled/transnasal surgery for skull base meningiomas needs closer and more stringent evaluation. The better immediate neurological outcomes, if real, need to be assessed against recurrence and survival rates. The applications and results should improve with advances in technology.

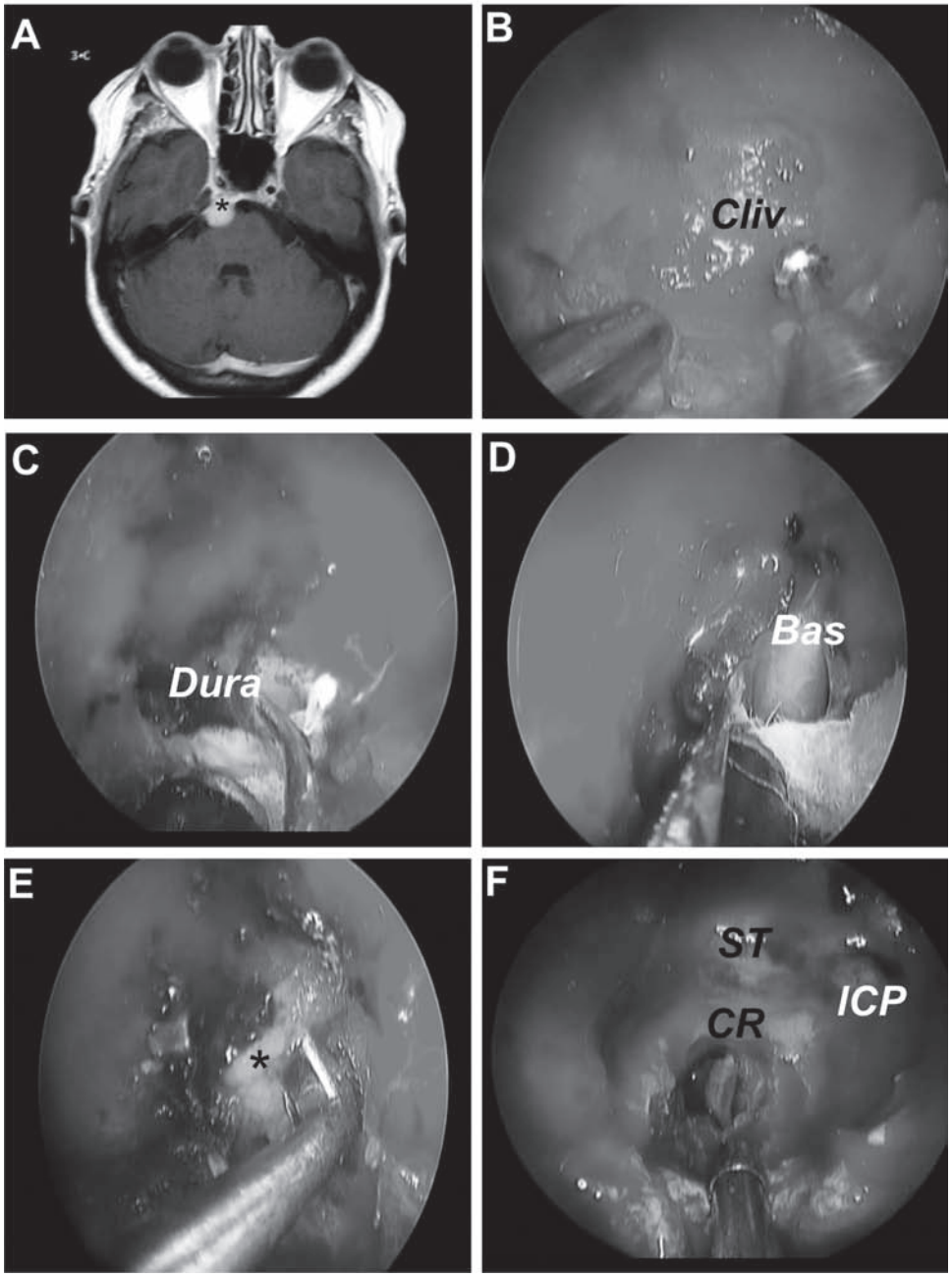


Fig. 38.7 Endonasal approach to clival meningioma. **(A)** Magnetic resonance imaging T1 with gadolinium contrast revealing a small clival lesion just right of the midline. **(B)** Clivus approach via endoscopic endonasal approach. **(C)** After drilling the clivus and linear durotomy, the dura is reflected with a blunt dissecting hook. **(D)** Basilar artery revealed underneath dura, highlighting the danger of this approach. **(E)** Tumor bulk gently removed via suction. **(F)** Anatomy of the clivus, seen under the sella turcica, the carotid protuberance forming its superior-lateral boundary. *, tumor; Bas, basilar artery; CR, clival recess; ST, sella turcica; lCP, left carotid protuberance.

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IX

Surgical Treatment of Spinal Meningiomas

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Chapter 39

Current Surgical Techniques in the Treatment of Spinal Meningiomas

Raqeeb Haque, Christopher P. Kellner, and Paul C. McCormick

Introduction

Spinal meningiomas are slow-growing, benign lesions that occur in the intradural, extramedullary space. They are predominantly treated with surgical resection. Despite their slow rate of growth, they can cause permanent neurological symptoms through progressive compression of the spinal cord or cauda equina, and therefore necessitate careful debulking and resection. Contemporary surgical techniques provide definitive treatment, with low recurrence rates, limited morbidity, and preservation or return of neurological function in the vast majority of patients. Surgical challenges lie mainly in access and vary depending on the location of the lesion throughout the spinal canal. For most spinal meningiomas, a posterior approach is appropriate. Ventrolateral and ventral lesions, however, may necessitate a more complicated and nuanced anterior approach, which will vary depending on the region of the spinal canal in which the tumor is found.

This chapter presents current knowledge of the epidemiology and histopathology of spinal canal meningiomas in general, and then describes the surgical approaches to spinal meningiomas that are found within different regions of the spinal canal.

Epidemiology

Epidemiological studies have reported that intradural tumors of the spine occur at an incidence of three to 10 per 100,000 people, of which two thirds are extramedullary. Meningiomas make up 13 to 19% of intracranial tumors, and spinal meningiomas account for 12% of all meningiomas.[1] Within the spinal canal, these lesions show a preference for the thoracic portion of the canal. They occur predominantly in the cervical (28%) and thoracic cord (64%), and only rarely in the lumbar spine (8%). Meningiomas are dural-based lesions that can arise from any aspect of the inner dural layer. Anterior meningiomas have been observed to be more common in the cervical spine (up to 48% of cervical meningiomas), whereas lateral meningiomas are the most common in the thoracic spine, and posterior lesions are the most common in the lumbar region. In one study, extradural tumor extension was identified in 6.3% of patients.[2]

The peak incidence for spinal meningiomas is between the sixth and eighth decades of life.[1,3,4] Younger patients (< 50 years) have a higher rate of tumors located in the cervical spine (39%), and a greater number related to a predisposing factor, such as neurofibromatosis 2 (NF2), radiation, or trauma. Young patients are also more likely to require reoperation for residual tumor (22% vs 5% in one particular case series).[5] As with intracranial meningiomas, there is a far higher incidence of spinal meningiomas in women. The rate has been recently quoted as 3– to 4.2:1.[1] This rate is reportedly even higher in the thoracic spine, with women presenting with significantly more spinal meningiomas than their male counterparts.

Clinical Features and Treatment Considerations

The clinical features of spinal meningiomas are variable and depend on numerous factors, such as tumor size, location, and biology. Due to the sensitivity of contemporary imaging, as well as its widespread access, incidental or asymptomatic intradural spinal tumors are now commonly identified on magnetic resonance imaging (MRI). In the absence of any "red flags," surgical treatment is usually not routinely recommended for incidental spinal meningiomas unless there is significant spinal cord com-

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pression. Small tumors can be followed over time with periodic imaging to document any expansion. Individual treatment decisions, however, are largely a matter of clinical judgment and patient preference.

Pain is the most frequent initial symptom of intradural tumors. Back pain is more typical in patients with meningiomas, but radicular pain, particularly thoracic, is also common. Night or recumbent pain is also common, although neither universal nor pathognomonic for intradural tumors, especially for larger tumors. Ultimately, features of myelopathy or cauda equina syndrome commence, the pattern and progression of which depend on tumor location. Significant deficits by the time of diagnosis have become less common, but relatively rapid acceleration of previously subtle or minimally functional or progressive neural dysfunction occurs with some frequency, presumably as the spinal cord reaches its tolerance for external compression.

Radiological Features

The imaging features and characteristics of intradural spinal meningiomas are well established.[6,7] MRI is obviously the imaging procedure for the diagnosis and evaluation of virtually all intradural pathology. Computed tomographic (CT) scanning may provide important complementary information, especially for mineralized or calcified tumors. Myelography is rarely needed, although the spatial resolution of myelo/CT can be more sensitive than MRI for identification of intradural extension of dumbbell tumors.

Spinal meningiomas are usually isointense to the spinal cord on both T1 and T2 sequences and demonstrate uniform enhancement following gadolinium administration. A dural attachment is also usually identified, although a dural tail is less common. Significant intratumoral mineralization or calcification will obviously alter MRI characteristics. Differential diagnosis includes mainly nerve sheath tumors, which more commonly show cystic regions or heterogeneous enhancement, or ependymomas.

Histopathology

Spinal meningiomas are generally benign low-grade lesions that demonstrate slow growth with limited risk of recurrence following resection.[2] Most are World Health Organization (WHO) grade I or II meningothelial and psammomatous types. Malignant examples, either de novo or transformation, account for only ~2% of reported cases. Calcification has been noted in approximately one in five spinal meningiomas, potentially complicating resection in that subset.[1,3,4,8] Despite their homogeneous histopathology, spinal meningiomas do exhibit some degree of biological heterogeneity with respect to growth rate. This underscores the importance of close periodic surveillance for newly diagnosed incidental lesions.

Surgical Approaches

The appropriate approach varies depending on the level of the tumor, the location of the tumor in relation to the spinal cord, and surgeon preference (**Table 39.1**). Tumor vascularity and consistency can also influence the choice of operative exposure, particularly for ventrally located tumors, but these characteristics are difficult to determine preoperatively. Anterior or anterolateral tumors may require a more complicated anterior approach, which could be problematic in a medically compromised patient. Approaches described in the literature for the cervical spinal canal include posterior, posterolateral, anterolateral, and anterior corpectomy.[9–11] Approaches described for the thoracic region are more varied and may carry a higher risk of morbidity due to the associated difficulty of accessing the anterior spinal cord in this region. Posterior approaches for this area include posterior, posterolateral, and the posterolateral endoscopic approach.[12–16] Other approaches should be divided by upper thoracic and lower thoracic lesions. Upper thoracic lateral and anterior approaches include the trapdoor approach and the parascapular extrapleural approach.[17–20] The lower thoracic spine is accessible through the lateral extracavitary approach and the retropleural approaches.[19–23] The lumbar spine is reached predominantly through the posterior, posterolateral, and lateral extracavitary approaches.[15,16,22,23]

Table 39.1 Surgical Approaches to Spinal Meningiomas

Region	Approach
Cervical	Posterior
	Posterolateral
	ACDF
Upper thoracic	Posterior
	Costotransversectomy
	Anterior trapdoor
	Transthoracic
Lower thoracic	Posterior
	Costotransversectomy
	Retropleural
	Transthoracic
Lumbar	Posterior
	Posterolateral

Abbreviation: ACDF, anterior cervical diskectomy and fusion.

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Posterior Approach with Laminectomy

The majority of spinal meningiomas can be adequately accessed and safely removed through a standard posterior approach with laminectomy. Minimally invasive exposures have also been described.[13,24,25] Following routine endotracheal intubation and placement of the appropriate vascular access and monitoring lines, the patient is placed in a prone position. We generally don't find awake fiberoptic intubation necessary for compressive cervical intradural pathology. Care is maintained to pad all bony prominences and subcutaneously coursing nerve trunks. A Mayfield head clamp is used for lesions of the upper thoracic (above T4) and cervical spine. The arms are tucked at the side for tumors above the T6 level. Perioperative intravenous antibiotics and steroids are administered. Fluoroscopy is utilized to localize the tumor level. Spinal level identification from fluoroscopy may need to be reconciled from the level noted on preoperative imaging, usually MRI, to ensure concordance. Specifically, issues related to number of lumbar vertebrae, transitional levels, and size and level of last rib should be addressed. A routine midline skin incision and subperiosteal dissection of the paraspinal muscles are performed. The amount of bone removed may be tailored to the individual situation. It is rare that spinal stability is compromised during posterior exposure of intradural pathology in the adult population. Meticulous hemostasis of the muscle, bone, and epidural space should be secured before dural opening because the intradural operative field is located in the most dependent part of the exposure. In general, we prefer to remove enough of the lamina so that the dural opening may extend beyond the rostral and caudal tumor poles. Unilateral laminectomy may be appropriate for laterally or ventrolaterally located tumors. Significant facet resection is usually not needed. A longitudinal midline dural opening is accomplished. The incision may be paramedian for eccentric tumors or for tumors with a midline posterior dural attachment. The dura is tented laterally with suture to the paraspinal muscles to maximized intradural visualization and tamponade the epidural venous plexus. Nearly all meningiomas arise from, and are attached to, a dural base. Unlike intracranial meningiomas, bony involvement or invasion almost never occurs because of a well-defined spinal epidural space. The arachnoid is opened over the surface of the tumor, and the rostral and caudal tumor poles are identified. A small cottonoid sponge can be placed at either pole. Depending on the size and location of the tumor, internal decompression may be performed with an ultrasonic aspirator, laser, or suction/cautery method. A surgical bone tool (Sonopet Ultrasonic Aspirator, Stryker, Kalamazoo, MI) can be extremely useful for highly mineralized or densely calcified tumors. In general these tumors have a very friable and moderately vascular surface. We will often leave the covering arachnoid layer intact on the tumor surface to facilitate much of the dissection. Dorsal and dorsolateral tumors typically present no significant problems with visualization and safe delivery off the spinal cord. Tumors located ventral to the dentate ligament, however, can be more challenging. Fortunately, in most cases, the tumor is either completely lateral to the spinal cord or eccentrically located in the ventral canal, producing some degree of lateral cord displacement and rotation that provides an adequate surgical corridor from the dorsal approach. This exposure can be further optimized by section and light suture retraction of the dentate ligaments (**Fig. 39.1**). Additional care with this maneuver must be taken with upper cervical lesions to avoid injury to the spinal accessory nerve. This nerve arises from a series of branches from the first six cervical levels that combine to form a trunk of ~1 mm in diameter that ascends to the craniocervical junction on the dorsal surface of the dentate ligament. Injury to this nerve causes a cosmetic and functional shoulder dysfunction, potentially of considerable morbidity. At other levels, either dorsal or ventral nerve roots may be reflected onto the tumor surface. In most cases these fascicles can be preserved, but in some instances it may not be possible to salvage the root. This is particularly true with tumors that originate from the lateral dura at the root exit zone. In cases of ventral tumor extension, the tumor is first debulked through a central trough. Tumors that are particularly vascular may need early devascularization of their dural origin with bipolar cautery. Ideally, the medial tumor component is completely disconnected from the dural attachment. The medial tumor can then be safely delivered away from the spinal cord. It may not be possible to directly visualize the ventral tumor–spinal cord interface. Fortunately, however, a well-developed arachnoid plane allows safe and gentle development of this plane to be achieved by tactile feel alone.

Management of the dural tumor origin varies according to surgeon preference and tumor location. Excision of the dural attachment often facilitates removal of dorsal and dorsolateral tumors, and competent dural patch grafting can be readily accomplished. Such is not the case with ventral and lateral attachments. We prefer to scrape the inner dural layers at the tumor origin and leave the outer layers intact (**Fig. 39.2**). There does not appear to be a difference in tumor recurrence rates related to whether the dural tumor origin is excised or preserved.

The dura is reapproximated with a running locked 4–0 silk or Prolene suture (Ethicon, Inc., Somerville, NJ). Valsalva to 35 mm Hg is performed to test the adequacy of the dural repair. A small muscle patch can be used for small dural defects. DuraGen (Integra LifeSciences Corp., Plainsboro, NJ) and/or Duraseal (Baxter Healthcare Corp., Deerfield, IL) may be used to augment the repair. A careful, layered closure of the muscle, fascia, and skin is performed. A subfascial drain can be placed. The patient is maintained flat in bed until postoperative day 2 and then progressively mobilized.

Ventral Approaches

As already noted, most lateral and ventrolateral meningiomas can be safely removed through a standard posterior exposure. Alternative posterolateral expo-

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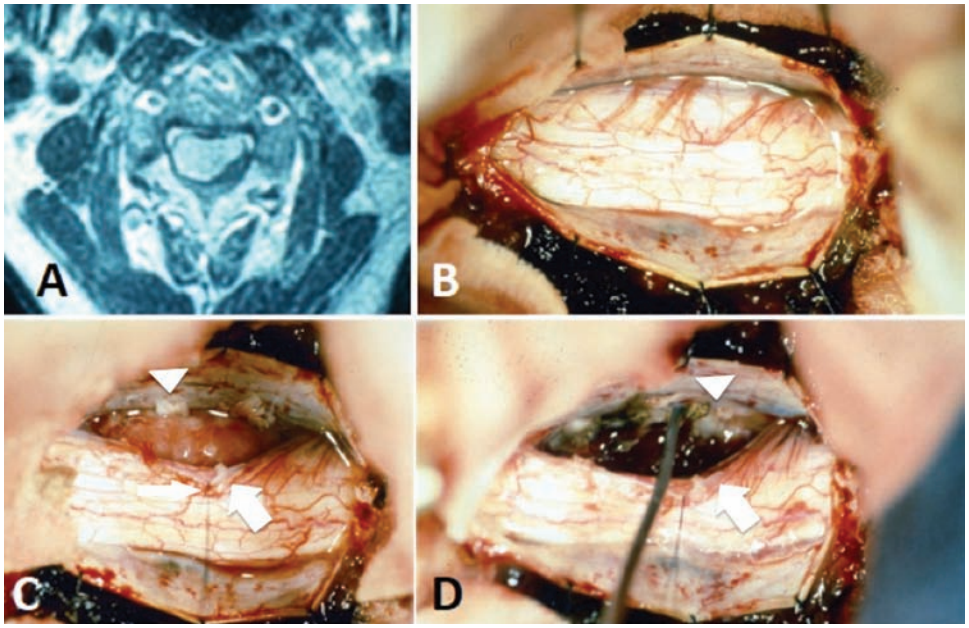


Fig. 39.1 **(A)** T1 axial magnetic resonance imaging (MRI) with contrast demonstrates a ventrolateral meningioma in the cervical spinal canal. **(B)** The spinal cord is seen through a posterior approach oriented with the rostral end to the left and caudal to the right. **(C)** A cervical dorsal root (*arrowhead*) and the dentate ligament (*thick arrow*) were cut to permit full access to the tumor. The spinal cord is gently rotated on a small suture placed in the dentate ligament (*thin arrow*). **(D)** Postexcision, the cut dorsal root (*arrowhead*) and dentate ligament (*arrow*) can be seen at the margin of the tumor bed.

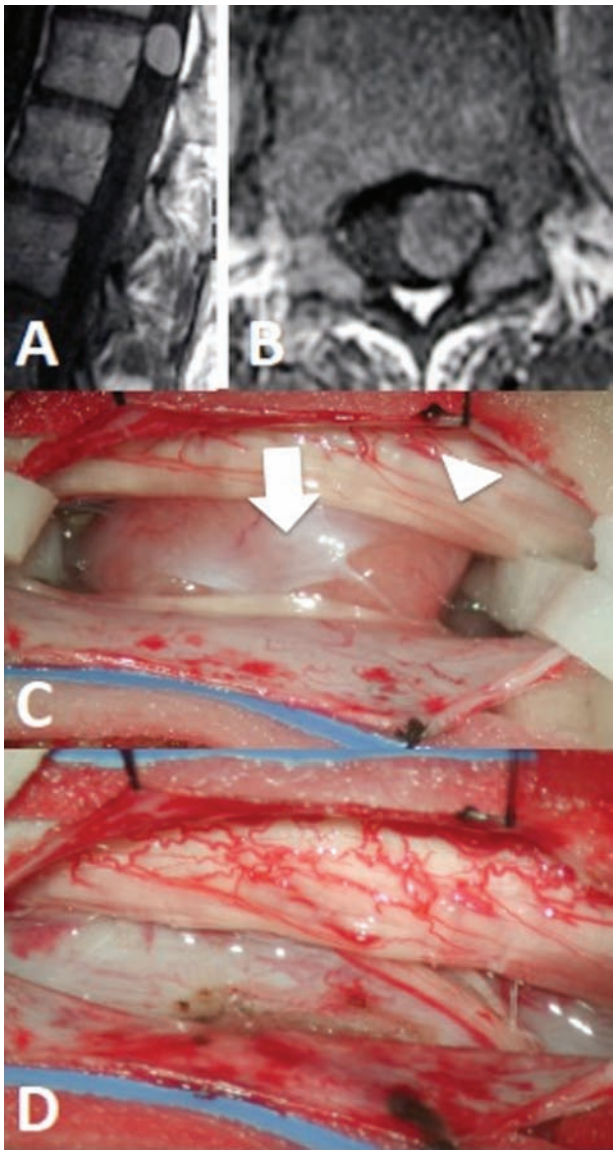


Fig. 39.2 **(A)** T1 sagittal and **(B)** T1 axial magnetic resonance imaging scans with contrast demonstrate a ventrolateral spinal meningioma at the level of L1. **(C)** At initial exposure, the dentate ligament is shown draped over the tumor (*arrow*), and the cauda equina (*arrowhead*) is observed displaced to the lateral aspect of the canal. **(D)** The tumor was fully resected with preservation of the dura to facilitate dural closure and minimize the chance of cerebrospinal fluid leakage.

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sures of the ventral intradural compartment have been described. Most describe fairly minor modifications to the standard posterior approach with respect to patient positioning, more lateral skin incision, and partial facet resection to improve ventral access. In some cases, however, such modifications may be inadequate to accomplish safe removal of tumors that occupy the ventral spinal canal. For example, meningiomas arising in a purely ventral location at cervical and thoracic levels with severe cord compression and significant bilateral ventral tumor extension but without spinal cord rotation or lateral displacement can be difficult to safely remove from a posterior or modified posterolateral exposure. Heavily calcified or highly vascular tumors further confound safe removal from a posterior route. For these tumors, a direct ventral or ventrolateral approach should be considered. Fortunately, improved techniques and equipment, increasing facility and experience with ventral spinal exposures, and secure reconstruction systems have allowed these exposures to be more routinely considered for the management of even intradural spinal lesions.[11]

Foramen Magnum and Upper Cervical

Foramen magnum and upper cervical (C1-2) meningiomas often occupy a ventral location. Direct ventral exposure of this region can be problematic (**Fig. 39.3**). Transoral exposure of the intradural compartment has largely been abandoned due to attendant risks, and extraoral exposures provide limited access and working space in the ventral intradural compartment. Fortunately, the spinal canal is quite capacious at these levels, allowing adequate ventral exposure to be achieved through a posterolateral trajectory. The ventral location of the occiput-C1 and C1-2 facet joints allows more lateral bone removal to further facilitate ventral exposure without compromising spinal stability. A far lateral approach with the patient in a lateral position can be utilized for patients with ventral meningiomas that extend beyond the midline or above the foramen magnum.

Cervical (C3-7)

More direct access to the ventral cervical canal (C3-7) can be accomplished through ventrolateral or ventral exposures.[11,26,27] Both exposures are performed with the patient in the supine position. Ventrolateral exposures require mobilization of the vertebral artery and provide ventral spinal canal entry through the intervertebral foramen. Varying degrees of vertebral body resection can be tailored according to the individual circumstance. Ventral exposures utilize the standard anterior exposures for anterior cervical diskectomy and corpectomy. Usually a one- or two-level corpectomy provides adequate exposure to remove a ventral tumor. Depending on the level of the thoracic inlet, ventral access may extend down to the upper thoracic levels (T1 or T2) through a standard supraclavicular cervical exposure. Following tumor removal, the dura should be closed in a watertight fashion. A patch graft or dural substitute may be required if a portion of the dura has been resected along with the tumor. DuraGen (Stryker) or Duraseal (Baxter) or both may be used to augment the dural repair. The corpectomy defect is reconstructed in standard fashion. Because the prevertebral dead space cannot be obliterated, a spinal drain is usually placed postoperatively for 3 to 5 days. Lesions with ventrolateral extension can be accessed through a posterior approach as well, with gentle spinal cord rotation to increase exposure.

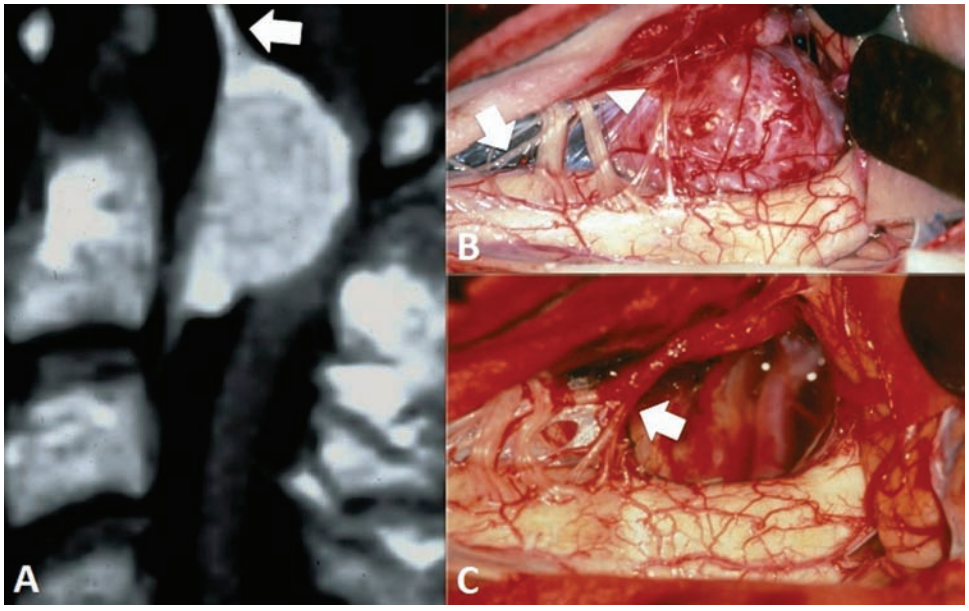


Fig. 39.3 **(A)** T1 sagittal magnetic resonance imaging with contrast demonstrates a ventral spinal meningioma at the level of C1/C2 with a dural tail extending rostrally to the foramen magnum (*arrow*). **(B)** At initial exposure, the dentate ligament (*arrowhead*) and spinal accessory nerve (*arrow*) are seen in contact with the tumor. **(C)** The postexcision tumor bed is shown with the spinal accessory nerve clearly preserved (*arrow*).

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Upper Thoracic (T1-3)

As already noted, standard supraclavicular anterior cervical approaches can be utilized to access the ventral intradural space at upper thoracic levels, depending on the level of the thoracic inlet. Such exposure may be further enhanced by partial resection of the clavicular head and upper manubrium. Preoperative imaging to localize the thoracic inlet, as determined by the upper margin of the manubrium, can assist in evaluating the appropriateness of this approach in individual cases. It is important to appreciate, however, that it is not simply enough to achieve adequate caudal exposure for tumor resection; additional caudal exposure will be required for spinal reconstruction. If upper thoracic ventral lesions cannot be adequately accessed through an extended anterior cervical exposure, then there are several potential anterior approaches that may be utilized for removal of a ventral upper thoracic meningioma. These approaches include the transthoracic, transsternal, and trapdoor approach. The major advantage to these anterior approaches in the upper thoracic region is that they facilitate full access to the ventral tumor and dural attachment for complete resection and an adequate dural closure. Major disadvantages include an increased risk of spinal instability from corpectomy; morbidity from the exposure of mediastinal structures; potential damage to anterior nervous elements, such as the recurrent laryngeal nerve, vagus, and phrenic nerves; and postoperative use of a chest tube.

The anterior trapdoor approach is often performed in conjunction with a thoracic surgery team for anterior spinal exposure. An incision is made from the angle of the mandible to the sternal notch and extended inferiorly to the fourth rib space, then continued along the inframammary crease. The sternum is split to the level of the fourth costal interspace. The right chest is then entered through the fourth intercostal interspace, and the neck dissection is carried down through the latissimus dorsi, taking the sternocleidomastoid muscle laterally. The "trapdoor" portion of the procedure is defined here, as the reflection of the sternocleidomastoid muscle laterally creates a trapdoor that allows exposure of the right side of the superior mediastinum. The anterior spine is then exposed; the great vessels identified; and the phrenic, vagus, and recurrent laryngeal nerves are identified, dissected, and mobilized. A plane is then developed between the right carotid, right subclavian, and trachea, which permits access to the anterior thoracic spine.

Here the vertebral bodies (T1-4) are dissected and the appropriate vertebral disks are curetted out. A high-speed drill is used to perform the appropriate corpectomy, which is then completed with a micro-Kerrison. The operating microscope is then brought into the field to better visualize and incise the posterior longitudinal ligament to tent it laterally as a separate layer. The dura is opened along the midline and also stitched to the side to visualize the tumor. The tumor is first decompressed using an ultrasonic aspirator, bone aspirating tool, laser, or suction/cautery. The ventral portion of the spinal cord should be identified and protected with a cottonoid. There is typically a well-defined arachnoid layer interposed between the tumor and the ventral surface of the spinal cord. The tumor should be debulked carefully. Depending on the extent of dural resection, a dural graft may be necessary at this point to reconstruct the dural closure. If the posterior longitudinal ligament has been preserved and opened separately, it may be closed with suture to provide an additional layer to prevent a postoperative cerebrospinal fluid (CSF) leak. An allograft or cage is then used to reconstruct the vertebral bodies, and a cervical fixation plate is placed.

Neurosurgical concerns during this procedure include avoiding a CSF leak through meticulous division of the posterior longitudinal ligament and performing a careful durotomy that preserves as much as possible for postresection closure. Identification of the recurrent laryngeal nerve is also important during the approach to avoid swallowing dysfunction, which can lead to major complications, such as respiratory stridor and insufficiency, and aspiration pneumonia, potentially requiring prolonged tracheostomy and enteral tube feeding postoperatively.

Lower Thoracic and Thoracolumbar (T4-L1)

Although the vast majority of intradural spinal meningiomas can be adequately exposed for safe removal through standard posterior or posterolateral exposures, large, purely ventral tumors with significant cord compression and bilateral extension, without cord rotation or lateral displacement, may require a more formalized anterior or anterolateral approach with thoracotomy (**Fig. 39.4**). Because entry into the subatmospheric intrapleural space can create problematic postoperative CSF fistulas following removal of intradural tumors, an extrapleural or retropleural exposure may be preferred. One such approach is the retropleural thoracotomy, which provides direct visualization and access to the ventral intradural space.[28,29] This procedure is performed with the patient in the lateral position. An 8 to 12 cm incision is made extending from the lateral aspect of the paraspinal muscles to the posterior axillary line over the rib at the tumor level (**Fig. 39.5**). The exposed 6 to 8 cm rib segment is resected and the endothoracic fascia is incised. The parietal pleura is then bluntly dissected off the inner thoracic wall to expose the rib head and adjacent vertebral bodies. The rib head is removed and a standard subtotal corpectomy is performed. The dura is opened and the tumor is resected utilizing standard techniques. Following tumor removal the dura is closed in a watertight fashion. If a portion of the dural tumor origin has been resected, then a patch graft may be necessary. The closure may be augmented with DuraGen (Stryker) or Duraseal (Baxter) or both. A suction drain is placed in the retropleural space and brought out through a separate stab incision. The patient is maintained at bed rest postoperatively until postoperative day 2 and then progressively mobilized. If a watertight dural repair cannot be accomplished, then placement of a spinal drain for 3 to 5 days may be considered.

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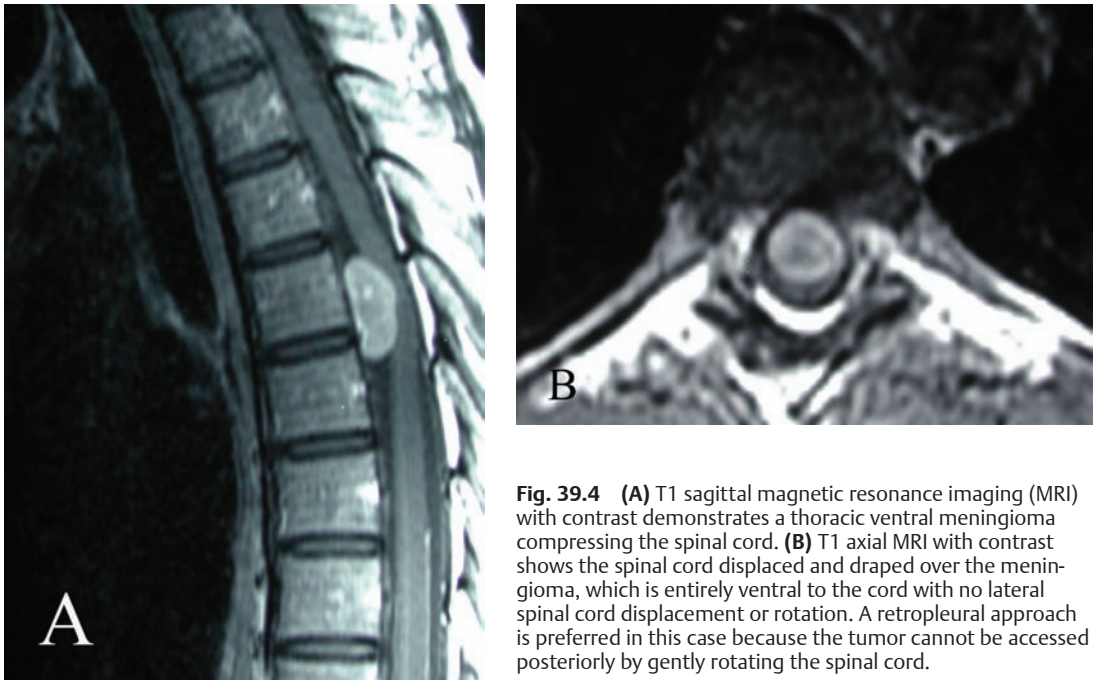


Fig. 39.4 **(A)** T1 sagittal magnetic resonance imaging (MRI) with contrast demonstrates a thoracic ventral meningioma compressing the spinal cord. **(B)** T1 axial MRI with contrast shows the spinal cord displaced and draped over the meningioma, which is entirely ventral to the cord with no lateral spinal cord displacement or rotation. A retropleural approach is preferred in this case because the tumor cannot be accessed posteriorly by gently rotating the spinal cord.

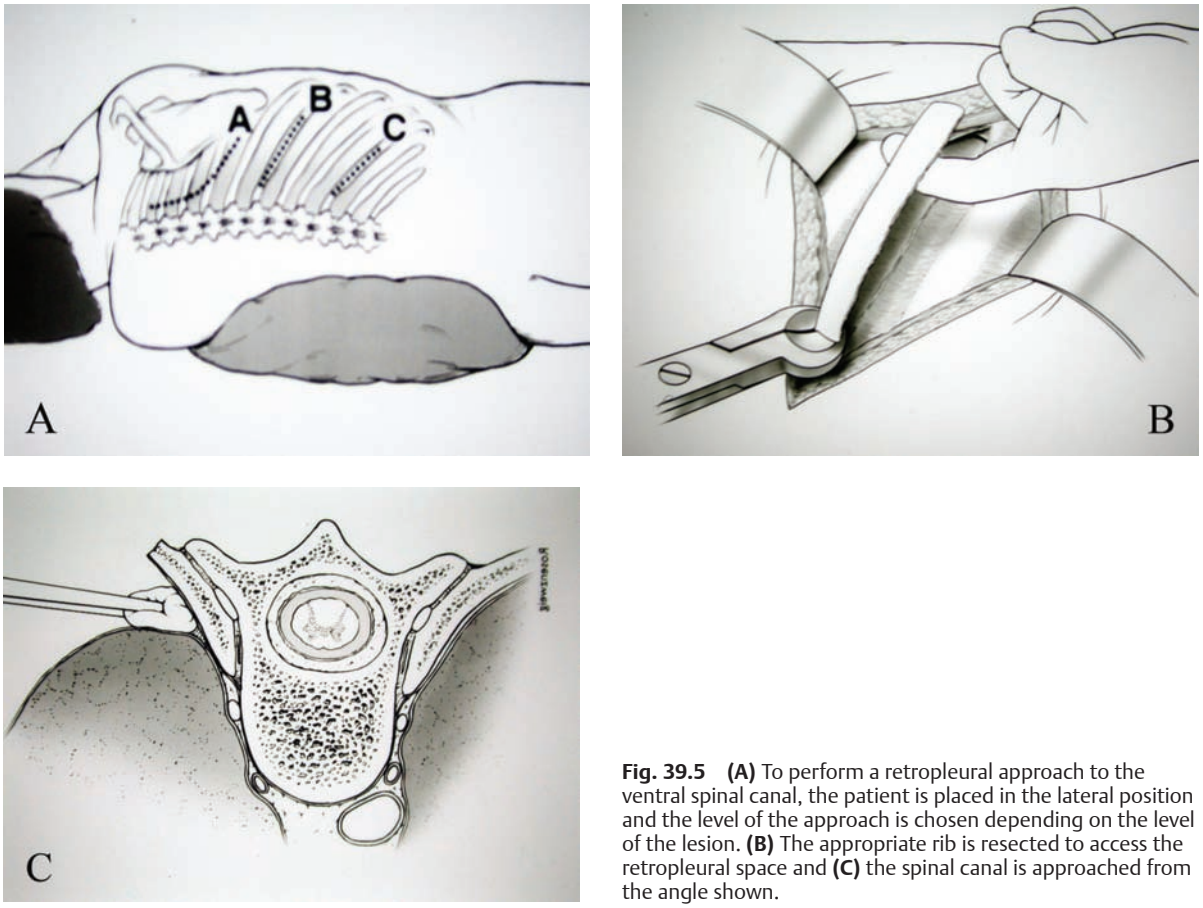


Fig. 39.5 **(A)** To perform a retropleural approach to the ventral spinal canal, the patient is placed in the lateral position and the level of the approach is chosen depending on the level of the lesion. **(B)** The appropriate rib is resected to access the retropleural space and **(C)** the spinal canal is approached from the angle shown.

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Lumbosacral (L2-S3)

Meningiomas in the lumbar and sacral spinal canal are rare, accounting for well under 10% of spinal meningiomas. Because only the filum terminale and cauda equina are present at these levels, a posterior spinal exposure through laminectomy provides adequate exposure for safe resection in nearly all cases. Even ventrally located tumors that displace the cauda equina dorsally can be safely accessed and removed following gentle mobilization and displacement of these nerves off the tumor surface. A more posterolateral approach may be preferred in some cases by some surgeons.

For large tumors, more than one corridor between these nerves may be necessary. Mobilization of these fragile nerves must be performed with great care because they lack any soft tissue–supporting matrix other than a thin fenestrated arachnoid layer.

Conclusion

Spinal meningiomas are a common intradural tumor that causes neurological dysfunction through slow growth and spinal cord compression. The major concerns regarding spinal meningiomas revolve around access to permit full resection while minimizing the chance of a CSF leak, neurological deficits, or systemic morbidity. Rarely, ventral or ventrolateral calcified tumors merit an anterior approach for adequate resection or dural reconstruction. These approaches can be challenging and should be performed by experienced surgeons in conjunction with other surgical specialists, if necessary.

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Chapter 40

Conformal Radiation Techniques for Meningiomas

Penny K. Sneed and Igor J. Barani

Radiation therapy plays an important role in the management of meningiomas that are not amenable to complete surgical resection or radiosurgery. In general, radiation therapy given 5 days weekly over ~6 weeks yields a high probability of preventing further tumor growth with a relatively low risk of significant complications. This chapter discusses the indications for conformal radiotherapy, limitations and expectations, techniques, results, and complications.

Indications for Radiotherapy

Surgical resection is the mainstay of treatment of meningiomas, but there are a variety of cases for which surgery alone may not be adequate treatment:

- Meningiomas not amenable to curative resection
- Recurrent or multiply recurrent meningiomas after surgery, which are at high risk for further recurrence at shorter intervals[1]
- Malignant meningiomas, and perhaps also atypical meningiomas, which have a high risk of recurrence even after total resection[2–5]

Furthermore, subtotal resection with later postoperative radiotherapy may be preferable over complete resection in some cases:

- Meningiomas for which risks of complete resection outweigh benefits (e.g., optic nerve sheath meningiomas and many cavernous sinus meningiomas)

There are also instances in which surgery may not even be indicated for tumors with typical appearance of benign meningioma:

- In patients who are not surgical candidates due to medical inoperability or advanced age
- In patients with asymptomatic or minimally symptomatic tumor in whom surgical debulking is not necessary for symptom relief and in whom curative surgical resection would not be possible or in whom risks of surgery may outweigh benefits.

In such cases, radiosurgery or radiation therapy may be indicated, either as an alternative to surgical resection, postoperatively, or at the time of progression or recurrence after surgery.

As shown in **Table 40.1**, gross total (Simpson grade I, II, or III)[6] resection alone results in a high likelihood of durable tumor control, 96% as a crude percentage[7] with actuarial freedom from progression probabilities of up to 98% at 5 years[4] and 75 to 80% at 10 years.[1,3,5] However, subtotal (Simpson grade IV) resection alone yields crude tumor freedom-from-progression rates of only 40 to 52%[7,8] and freedom-from-progression or progression-free survival probabilities of only 38 to 62% at 5 to 10 years.[1,3–5,9,10] The addition of postoperative radiotherapy to subtotal resection increases the progression-free survival probability to 78 to 91% at 5 to 10 years, comparable to the results of gross total resection alone.[3,9,10]

As clinicians have become more comfortable with radiotherapy in the treatment of meningiomas, it has become more commonplace to manage selected patients with radiotherapy alone as an alternative to surgery alone or surgery with postoperative radiotherapy, with equivalent results.[11–15] Thus radiotherapy alone without surgical resection yields progression-free survival equivalent to that of subtotal resection plus radiotherapy.

The choice of radiosurgery versus radiotherapy is governed primarily by risk to surrounding normal tissues. The tumor dose used for radiosurgery exceeds normal tissue tolerance but is acceptable for small targets because of the very precise targeting and very steep dose fall-off outside of the target. The larger the target, the less

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Table 40.1 Results of Surgery ± Radiotherapy for Meningiomas

First Author	Year	n	Median or Mean Follow-Up (years)	Freedom from Progression or Progression-Free Survival Probability (No. of Patients)			Time Point
				GTR Alone	STR Alone	Radiotherapy ± STR	
Mirimanoff[1]	1985	225	~6	80% (145)	45% (80)	NA	10 years
Barbaro[7]	1987	135	6.5	96% (51)	40% (30)	68% (54)	Crude
Miralbell[9]	1992	115	6.0	–	48% (79)	78–88% (36)	8 years
Mahmood[4]	1994	246	5.2	98% (181)	62% (65)	–	5 years
Peele[8]	1996	86	~4	–	52% (44)	100% (42)	Crude
Condra[3]	1997	246	8.2	80% (174)	40% (55)	87% (17)	10 years
Stafford[5]	1998	581	9	75% (463)	39% (115)	–	10 years
Soyuer[10]	2004	92	7.7	77% (48)	38% (32)	91% (12)	5 years

Abbreviations: GTR, gross total (Simpson grade I–III) resection; STR, subtotal (Simpson grade IV–V) resection.

steep the dose fall-off and the greater the volume of the shell of surrounding normal tissue exposed to a potentially damaging radiation dose level. Thus radiosurgery may be an excellent treatment option for small tumors less than ~3 to 4 cm in diameter and at least 2 to 3 mm away from the optic nerve and chiasm. For larger tumors or those adjacent to or compressing optic nerve or chiasm or with extensive brain stem compression, radiotherapy given over 5 to 6 weeks is preferred and is generally well tolerated by adjacent normal tissue.

Limitations and Expectations of Radiotherapy

Although radiotherapy is generally successful at preventing further growth of benign meningiomas, it tends to result in little or no tumor shrinkage over time owing to underlying radiobiological principles. The major mode of cell death after exposure to ionizing radiation is via double-strand breaks in DNA, and for most kinds of tumor cells, cell death occurs after one or more cell divisions, because DNA mistakes accumulate in daughter cells. Rapidly dividing cells tend to die quickly after radiotherapy, resulting in marked tumor shrinkage or even complete disappearance of malignant tumors over weeks or months. In contrast, very slowly dividing cells may survive for many years after radiotherapy, resulting in little or no tumor shrinkage. Thus bulky benign tumors causing significant symptoms from mass effect may require surgical debulking, given that radiation therapy cannot be counted upon to reduce significant tumor mass effect in a timely manner. Over years after radiotherapy, ~20 to 30% of meningiomas shrink at least 2 mm in diameter or 25% by volume (**Table 40.2**). In one series, 14% of meningiomas had partial response (at least 50% shrinkage) after radiotherapy,[16] but complete disappearance is rare. Cases of rapid, marked tumor shrinkage in the absence of histological verification should raise one's suspicion of a diagnosis of dural metastasis, hemangiopericytoma, or leiomyosarcoma, which typically shrink dramatically after radiosurgery or radiotherapy.

On the other hand, visible tumor shrinkage is not necessary to see improvement in cranial nerve deficits; symptomatic improvement of cranial nerve deficits is relatively common after radiotherapy, despite the fact that there may be little or no visible tumor shrinkage. Papers containing detailed reports of symptomatology before and after radiotherapy describe improvement in 50 to 81% of symptoms, including cranial neuropathies,[12,17] and clinical improvement in 43 to 71% of patients (**Table 40.2**).[11,14,18,19] This is particularly relevant for cavernous sinus meningiomas and optic nerve sheath meningiomas. In the case of optic nerve sheath meningiomas, surgery often carries a high risk of visual deterioration, whereas primary radiotherapy has a low risk of visual deterioration and may improve vision in about one third to one half of the patients with useful vision before radiotherapy.[20,21]

Evaluation of the success of radiotherapy for meningiomas is complicated by the facts that untreated meningiomas may grow very slowly and treated meningiomas may shrink very slowly, if at all. Because meningiomas are expected to remain stable or shrink very slowly over time, results of radiotherapy for meningiomas are usually described in terms of freedom from tumor progression or progression-free survival (sometimes called relapse-free survival), and at least 5 to 10 years of follow-up are desirable to assess treatment results.

Another important major limitation of radiotherapy is that it can generally only be given once to the same area without incurring major risks of brain necrosis or other serious complications, unless the volume of normal tis-

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Table 40.2 Results of Radiotherapy for Meningiomas, With or Without Prior Subtotal Resection

First Author	Year	Total No. of Patients	Benign/ Atypical/ Malignant/ No Tissue	Radiation Technique	Median or Mean Dose (Gy)	Median or Mean Follow-Up (years)	FFP/PFS Probability %	Time-Point (years)	Incidence of		
									Clinical Improvement %	Tumor Shrinkage %	Grade 3–5 Late Toxicity %
Goldsmith[22]	1994	117	117/0/0/0	EBRT	54.0	3.3	77	10	–	–	3.6
Nutting[25]	1999	82	82/0/0/0	EBRT	55–60	9	–/83	10	–	–	13
Vendrely[15]	1999	156	65/5/5/81	EBRT	50	3.3	79*/89.5	5	59	29	8
Wenkel[30]	2000	46	46/0/0/0	Protons+RT	59.0	4.4	–/88	10	–	–	20
Pourel[26]	2001	45	26/2/5/12	EBRT	56	2.5	–/76	5	–	–	2.2
Dufour[18]	2001	31	20/0/0/11	EBRT	52	6.1	–/93	10	59§/71	29	3.2
Jalali[23]	2002	41	33/0/0/8	FSRT	55	1.8	100/91	3	27	22	12.2
Uy[28]	2002	40	25/0/0/15	IMRT	50.4	2.5	93/88	5	–	23	5
Pirzkall[19]	2003	20	16/0/0/4	IMRT	52	3	100/100	–	60	25	0
Selch[27]	2004	45	29/0/0/16	FSRT	50.4	3	97/97	3	20	18	0
Weber[29]	2004	16	11/2/0/3	Protons	56	2.8	92/92	3	13	19	12.5
Milker-Zabel[14]	2005	317	153/26/0/138	FSRT	57.6	5.7	–/89†	10	43	23	0
Noël[17]	2005	51	44/0/0/7	Protons+RT	60.6	2.1	98/98	4	69§	20	3.9
Henzel[11]	2006	224	113/10/6/95	mostly FSRT	55.8	3	97/–	5	43	46	0
Milker-Zabel[24]	2007	94	51/9/4/30	IMRT	57.6	4.4	94*/95	5	40	20	4
Liter[12]	2009	100	26/0/0/74	FSRT	45	2.8	94/–	5	50–81§	9	0
Metellus[13]	2010	53	22/3/0/28	FSRT	52.9	6.9	–/96	10	58	30	1.9

Abbreviations: EBRT, external beam radiotherapy; FFP, freedom from progression; FSRT, fractionated stereotactic radiotherapy; IMRT, intensity-modulated radiotherapy; PFS, progression-free survival; RT, radiotherapy.
* Crude percentage rather than actuarial probability.
† Excluding atypical or malignant meningiomas.
§ Percentage of patients with improvement, by symptom.

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sue exposed to retreatment is very small. The radiation dose that is required for good tumor control is close to the tolerance of normal brain tissue, and normal cells repair radiation injury to some extent, but never completely.

Radiotherapy Techniques

Radiation therapy implies multiple treatments, or "fractions," given over time, as opposed to radiosurgery, which generally refers to a high dose of highly focused radiation given in a single fraction. Fractionated radiosurgery refers to very focused radiation given daily (or much less commonly every other day) over two to five sessions. In many centers and publications, there is no distinction between single-fraction and fractionated radiosurgery, but much less information is available about results and toxicity of fractionated radiosurgery for meningiomas as opposed to single-fraction radiosurgery or "fully fractionated" radiotherapy to ~50.4 to 54 Gy at 1.8 Gy per daily fraction, 5 days per week over 6 weeks. The term *fractionated stereotactic radiotherapy* generally implies fully fractionated treatment but with superior daily treatment setup accuracy compared with conventional radiotherapy. The primary subject of this chapter is fully fractionated radiation therapy.

In preparation for radiotherapy, most patients will have a thermoplastic mask made to establish a reproducible position for daily treatment. Marks are placed on the mask to guide daily alignment on the treatment machine. A treatment-planning computed tomographic (CT) scan is performed with the patient in the mask. This CT scan is normally fused with a diagnostic magnetic resonance imaging (MRI) scan in the treatment planning computer. The target for radiotherapy is then outlined, first delineating the gross tumor including any associated bone hyperostosis or extracranial tumor extent, referred to as the gross tumor volume (GTV), or tumor bed and any residual gross disease. Then margin is added as desired for microscopic disease extension, referred to as the clinical target volume (CTV), perhaps 0 to 5 mm for benign meningioma but up to ~1 cm for atypical or malignant meningioma. It may be preferable to use nonuniform margins, with smaller margins extending into brain and larger margins along the dura. Unlike the treatment planning for gliomas, it is not necessary to include edematous brain adjacent to the original tumor site within the target volume when treating atypical or malignant meningiomas. Finally, additional margin is added to account for daily setup variation, perhaps 2 to 5 mm depending on the frequency and accuracy of treatment setup verification on the treatment machine, referred to as the planning target volume (PTV). Critical normal tissues are also outlined as needed to ensure adequate sparing of these structures when possible, such as the eyes, lenses, optic nerves, optic chiasm, pituitary gland and stalk, brain stem, and cochlea.

A treatment plan is developed on the computer to encompass the target while limiting the dose to surrounding normal tissues. Radiotherapy is typically given at 1.8 Gy per daily fraction, 5 days weekly, to a total dose of ~50.4 to 54.0 Gy for benign meningiomas and ~59.4 Gy for atypical or malignant meningiomas prescribed at the 90 to 95% isodose line normalized to a maximum dose of 100% (**Fig. 40.1**).

Multiple radiation techniques of varying complexity are available for radiotherapy of meningiomas. In its simplest form, radiation therapy can be given via two opposed rectangular or shaped radiation fields, such as opposed laterals. However, modern radiotherapy for meningiomas typically consists of either three-dimensional conformal radiation or intensity-modulated radiotherapy (IMRT), which may be delivered in a variety of ways, such as step-and-shoot fields or helical or arc rotation. Three-dimensional conformal radiotherapy and step-and-shoot IMRT techniques generally employ multiple radiation beams aiming at a focal point within the target region from a variety of angles, with shaping of each beam around the target volume from the beam's eye view. In the case of three-dimensional conformal radiotherapy, there is uniform beam intensity across the

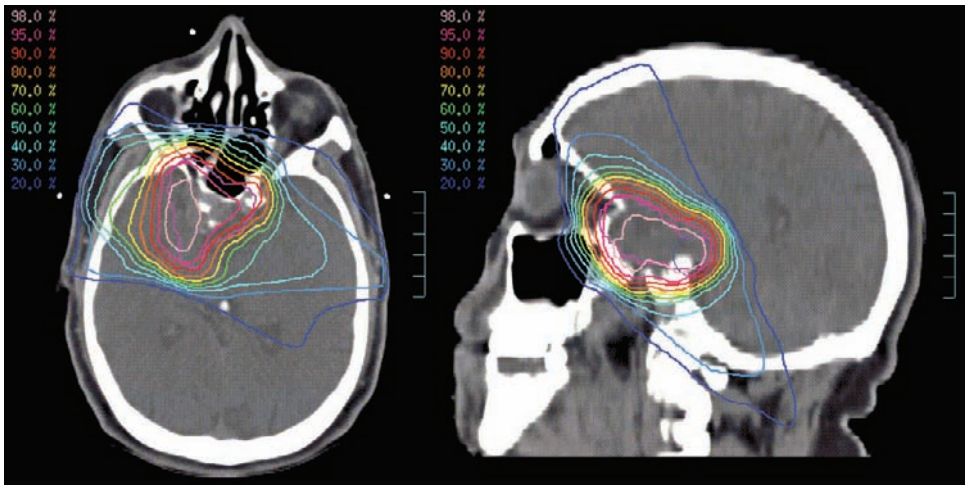


Fig. 40.1 Axial and sagittal images of a three-dimensional conformal radiotherapy plan for a large benign meningioma in the right cavernous sinus with extension into the sella and suprasellar region, extensively contacting but not compressing the optic chiasm. A dose of 50.4 Gy at 1.8 Gy per daily fraction was prescribed at the 95% isodose contour, normalized so that the maximum dose was 100%. The 98, 95, 90, 80, 70, 60, 50, 40, 30, and 20% isodose contours are shown. Care was taken to ensure that the 98% "hot spot" was away from the optic nerves and chiasm to lessen the risk of late visual complications.

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cross section of each beam, whereas each IMRT beam has varying dose intensity such as a complex checkerboard pattern. The varying dose intensities are calculated by inverse planning in a treatment planning computer and created by computer control of "multileaf collimators" 1, 3, 5, or 10 mm wide that move during the course of treatment to create the intensity pattern. IMRT often makes possible more complex dose distributions for complexly shaped targets or complex dose constraints. Compared with three-dimensional conformal radiotherapy, IMRT requires more stringent quality assurance and machine tolerances to ensure accurate and safe treatment delivery, and there may be a tendency for more heterogeneous dose ("hot spots") within the treated volume. An awareness of this dose heterogeneity is especially important around critical structures such as the optic nerve, chiasm, or brain stem; extra care must be taken to avoid hot spots within critical structures if they are included within the planning target volume, or the risk of complications may increase dramatically.

Proton beam or heavy particle radiotherapy may also be used to treat meningiomas. These beams have steep dose-fall beyond the distal edge of a target compared with conventional photon radiotherapy; this feature may allow for improved radiation dose distributions. This is of less importance for benign meningiomas that are treated to ~54 Gy than for more radioresistant tumors, such as chordomas and chondrosarcomas, requiring higher doses. Nevertheless, heavy particle therapy could be put to good use in many cases, such as allowing for improved sparing of pituitary gland, pituitary stalk, or chiasm when tumors lie very close to or in contact with these structures.

Results

The results of radiotherapy with or without prior subtotal resection are presented in **Table 40.2**, ordered by year of publication.[11–15,17–19,22–30] Overall, the 5-year freedom from progression probabilities ranged from 79 to 97% with 5- and 10-year progression-free survival probabilities of 76 to 95% and 83 to 96%, respectively. The majority of the tumors treated were benign, with some series including small numbers of patients with atypical or malignant histology. Multiple reports include a mixture of patients treated postoperatively after subtotal resection and those treated at the time of recurrence or progression following gross total or subtotal resection. Also, patients with only a clinical and imaging diagnosis of meningioma are increasingly being treated and included in reports of radiotherapy for meningioma. Outcomes appear to be the same for patients treated with radiotherapy for presumed meningioma as for those with a surgical diagnosis of benign meningioma.[11–14]

Results from **Table 40.1** and **Table 40.2** are plotted graphically in **Fig. 40.2** with the year of publication on the x-axis and probability of tumor freedom from progression or patient progression-free survival at 3 to 5 or 8 to 10 years on the y-axis. Note that the results of external beam radiotherapy (EBRT) appear to be trending upward over time. In support of this observation, Goldsmith et al reported 5-year progression-free survival probabilities of 77% versus 98% (p = 0.002) for patients with benign meningioma treated before 1980 versus 1980 or later, before versus after the availability of CT or MRI-based treatment planning and more sophisticated radiotherapy techniques that facilitated improved tumor coverage and normal tissue sparing.[22]

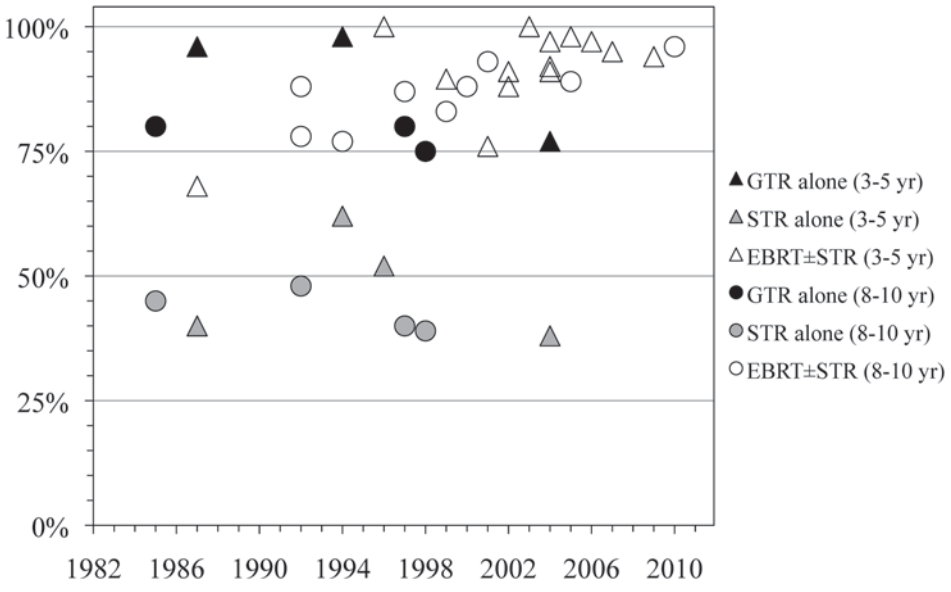


Fig. 40.2 Scatter plot of freedom from progression or progression-free survival probabilities versus year of publication for the series included in **Table 40.1** and **Table 40.2**. Triangle symbols denote 3- to 5-year results, and circles denote 8- to 10-year results. Results of gross total resection (GTR) alone are shown in black, subtotal resection (STR) alone in gray, and external beam radiotherapy (EBRT) with or without prior subtotal resection in empty symbols. Note that the results of EBRT appear to be improving gradually over time.

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Freedom from progression and progression-free survival are significantly worse for atypical and malignant histologies. Goldsmith et al observed 5-year progression-free survival of 89% versus 48% ($p = 0.001$) and overall survival probabilities of 85% versus 58% ($p = 0.02$) for 117 patients with benign meningioma versus 23 with malignant meningioma, respectively.[22] Pourel et al reported progression-free survival of 95% at 5 years and 81% at 8 years for 26 patients with known benign meningiomas, whereas all seven patients with atypical or malignant meningiomas had tumor recurrence or progression before 2 years, and all but one died by 42 months after radiotherapy ($p < 0.001$).[26] Milker-Zabel et al reported 5- and 10-year progression-free survival probabilities of 90.5% and 89% for benign and unknown histology versus 89% and 67% for atypical meningioma treated with fractionated stereotactic radiotherapy ($p < 0.002$)[14] and 5-year progression-free survival probabilities of 96% versus 78% for complex skull-base benign versus atypical meningiomas treated with IMRT.[24]

Multiple authors have also described poorer results for patients treated at the time of tumor recurrence or progression rather than immediately postoperatively. For example, Pourel et al reported 5-year progression-free survival of 90% after adjuvant radiotherapy given postoperatively versus 73% after salvage radiotherapy given at the time of recurrence or progression following initial surgery ($p = 0.37$).[26] Milker-Zabel et al noted relapse rates of 4.7% after radiotherapy given for primary disease versus 10% after radiotherapy given at the time of recurrence or progression ($p = 0.06$).[14] The significance of this finding is unclear given the potential of selection bias; patients who had tumor recurrence or progression are probably a poorer-prognosis subset of all patients treated surgically. In addition, some tumors transform over time to a more malignant histology and may be atypical or malignant at the time of radiotherapy for recurrence or progression despite a prior surgical diagnosis of benign histology. We recommend making the decision regarding adjuvant versus delayed salvage radiotherapy based on clinical factors, electing upfront postoperative treatment in patients with atypical or malignant histology, symptoms related to residual tumor, or disease likely to cause significant morbidity in the event of tumor growth (such as tumor adjacent to or compressing chiasm or brain stem). We tend to elect surveillance in patients with asymptomatic residual benign meningioma in locations where tumor progression would not be expected to be symptomatic, postponing radiotherapy or radiosurgery until there is imaging evidence of disease progression.

Toxicity and Complications

Toxicity of radiotherapy may be acute or late. Expected acute toxicities of modern radiotherapy include fatigue, which generally resolves within 1 or several months after treatment, and hair loss in the treatment portals, with normal hair regrowth 3 to 6 months after treatment unless a higher dose to the scalp was necessitated by tumor invasion of bone close to the skin surface, in which case alopecia may be permanent. Treatment of tumors close to mucosa or the auditory canal may cause temporary mucositis or otitis. Some patients develop headache or slight nausea during radiotherapy, which generally responds to pain or antiemetic medications or to a course of steroids.

Serious complications of radiotherapy tend to be from late toxicity and may be permanent. The risk of serious late toxicity ranges from 0 to 20% in **Table 40.2**, with a patient-weighted average of 3.5%. Depending on the tumor location, patients may be at low risk of developing partial vision loss or blindness, hormonal deficits, cerebrovascular accidents, hearing loss or other cranial neuropathies, cataract, brain or brain stem necrosis, or neurocognitive decline. In some papers, the authors linked complications to cases of reirradiation or doses clearly exceeding known tolerance levels currently employed.[17,24,30] Another potential serious complication of radiotherapy is the risk of induction of a second tumor or malignancy or of tumor transformation to a more malignant histology. These risks are quite small; none of the papers cited in this chapter reported a case of second malignancy attributable to radiotherapy or of an excessive risk of tumor transformation among patients treated with radiotherapy.

Conclusion

- Meningiomas tend to remain stable or shrink slowly after radiotherapy.
- Symptomatic improvement of cranial nerve deficits is relatively common after radiotherapy, despite the fact that there may be little or no visible tumor shrinkage.
- Success of radiotherapy for meningiomas is generally measured by tumor freedom from progression or patient progression-free survival.
- For benign meningiomas or presumed meningiomas not amenable to safe complete resection or radiosurgery, modern conformal radiotherapy to ~50 to 54 Gy at 1.8 Gy per daily fraction yields a 10-year progression-free survival probability of ~88 to 96%, with ~3.5% risk of serious late complications.
- Results are significantly poorer for atypical and malignant meningiomas.
- Radiotherapy is preferred over radiosurgery for tumors larger than ~3 to 4 cm in diameter and for tumors less than 2 to 3 mm away from the optic nerve or chiasm.
- Radiotherapy after subtotal resection yields progression-free survival comparable to that of gross total resection.
- Radiotherapy alone without surgical resection yields progression-free survival equivalent to that of subtotal resection plus radiotherapy.
- Common radiotherapy techniques include three-dimensional conformal radiotherapy, IMRT, or fractionated stereotactic radiotherapy (FSRT).

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- Proton therapy and to a lesser extent heavy particle therapy are becoming more available.
- For benign meningioma, it is unclear whether outcomes are compromised by treating with radiotherapy at the time of progression rather than upfront; clinical judgment should be exercised. We recommend upfront radiotherapy for residual tumor that could be expected to cause important symptoms in the event of tumor growth.

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Chapter 41

Stereotactic Radiosurgery for Meningiomas: Techniques and Results

Douglas Kondziolka, David Mathieu, Ricky Madhok, John C. Flickinger, and L. Dade Lunsford

Introduction

This textbook addresses the many issues surrounding meningiomas. From anatomical and pathological considerations to imaging and natural history, clinical context based on age, adjuvant therapies, and surgical resection stratified by brain or spine location, one meningioma may be very different from another. These common tumors pose a variety of therapeutic challenges because of these issues. Over the past century, craniotomy and tumor resection along with the tumor's dural base became the preferred approach for the majority of symptomatic patients with intracranial tumors.[1] Over time, the morbidity of resection was reduced with microneurosurgical technique, better anesthesia, neuronavigation, and improved medical care. These achievements have been discussed. However, because these usually benign tumors may be associated closely with critical neural or vascular structures, complete resection may not be feasible.[2–5] Meningiomas adjacent to venous sinuses may be resectable only at the risk of major neurological deficits caused by venous injury. Elderly or infirm patients often seek alternative approaches. This chapter reviews our experience in the care of over one thousand patients with intracranial meningiomas using stereotactic radiosurgery. We have found that our approach provides safe and effective management of these tumors when properly selected.

Clinical Indications

During our first 20 years of experience, radiosurgery was performed on 1191 intracranial meningiomas. Our first report was in 1991, when we detailed results from the first 50 patients.[6] We recently reviewed results from the initial 972 patients with 1045 tumors managed over the initial 18 years.[7] The brain locations of these tumors are shown in **Table 41.1**. The decision to perform radiosurgery was made in patients with residual or recurrent smaller volume tumors after prior resection, those with symptomatic primary tumors in locations associated with higher risk for resection, those with concomitant medical illnesses or advanced age, those in younger patients who chose radiosurgery over other available options, and those in younger patients with minimal symptoms or who were asymptomatic but chose against observation. There are some relative contraindications or exclusion criteria for radiosurgery. These include large tumor volume (mean diameter > 3.5 cm), tumors with symptomatic optic nerve or chiasmal compression, optic nerve sheath tumors with preserved vision, elderly patients with asymptomatic tumors, or tumors with atypical imaging features and no prior histologic diagnosis.

The detailed results from 982 tumors (94%) were available for analysis. Five hundred and four patients (51%) had had no prior treatment, and prior resections were usually partial removals (84%). A solitary tumor was present in 818 patients, and multiple tumors were found in 161. Twenty-eight patients had neurofibromatosis type 2. Tumor pathology was studied in 511 residual or recurrent tumors. Prior radiotherapy (RT) had been delivered to 54 patients (48 after a resection, and six had RT alone); two patients had prior radiosurgery elsewhere. Eight patients had received chemotherapy. Twenty-five tumors (2.5%) developed after prior fractionated irradiation.

Radiosurgery was performed under local anesthesia with mild sedation as necessary, using a Leksell Gamma Knife (Elekta, Inc., Norcross, GA). We have used the Gamma Knife models U, B, C, 4C, and Perfexion for our patients with meningiomas. Radiosurgery was targeted with stereotactic computed tomographic (CT) guidance (before 1992), and with magnetic resonance imaging (MRI) since.

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Table 1 Gamma Knife Radiosurgery for 1045 Intracranial Meningiomas

Location	Number of Tumors
Posterior fossa	
Petroclival	122
Petrous ridge	66
Foramen magnum	22
Other	42
Middle fossa	
Cavernous sinus	306
Sphenoid wing	32
Other	13
Anterior fossa	
Olfactory groove	29
Planum sphenoidale	29
Anterior clinoid	17
Parasellar	13
Convexity	126
Other	
Parasagittal	113
Tentorial notch	40
Torcular	6
Falcine	47
Intraorbital	13
Intraventricular	9

A mean of 7.5 isocenters were used to provide conformal radiosurgery. The dose received by adjacent critical structures was determined, and selective beam blocking used if necessary to restrict the dose fall-off (**Fig. 41.1**). We delivered a mean dose to the tumor margin of 14 Gy and a mean maximum dose of 28 Gy. The mean tumor volume in this series was 7.4 mL. The mean volume receiving ≤ 12 Gy was 8.4 mL. Radiation doses were prescribed to the 50% isodose volume for 886 tumors (85%) (**Fig. 41.2**). For tumors near the optic nerve or chiasm, the average maximal optic dose was 6.4 Gy. After radiosurgery, patients were discharged home within 24 hours, and in recent years on the same day. The median follow-up in this study was 4 years; 842 patients were still living (86%). Follow-up past 5, 7, 10, and 12 years was obtained in 327, 190, 90, and 41 patients, respectively.

Tumor Response to Radiosurgery

Imaging studies after radiosurgery showed that 407 tumors had regressed, 454 were unchanged, and 96 had enlarged, for a raw tumor control rate of 90% (**Fig. 41.3**). Imaging and clinical follow-up were requested for all patients but were not complete for all.

Adjuvant Radiosurgery

Based on prior histopathology, we had 424 World Health Organization (WHO) grade I meningiomas in this series, and 384 were available for study. We found that 172 tumors had regressed, 186 were unchanged, and 26 had enlarged, for a tumor control rate of 93%, at a median of 4 years. Ninety-one percent of patients were either improved (n = 21), or unchanged (n = 341) clinically. Imaging follow-up past 8 and 10 years was obtained in 79 and 53 tumors, respectively, both with a control rate of 91% (**Figs. 41.4** and **41.5**). Past 10 years, 45 patients were stable, six were improved, and two were worse. For grade I meningiomas, the 5-, 10-, and 15-year actuarial tumor control rates were 97 + 1.2%, 87.2 + 4.4%, and 87.2 + 4.4%. Disease-specific survival was 98.9 + 0.5%, 96.2 + 1.9%, and 96.2 + 1.9%, respectively.

Of 56 WHO grade II tumors, 54 were available for review. Sixteen had regressed, 11 were unchanged, and 27 had enlarged for a tumor control rate of 50%, at a median of 2 years. During follow-up, 72% of these patients were stable clinically. Of 31 WHO grade III tumors, 29 were reviewed. We found that four regressed, one was stable, and 24 later enlarged, for a tumor control rate of 17% at a median of 15 months.

Primary Radiosurgery

There were 536 tumors that received radiosurgery as primary management without a prior biopsy (**Fig. 41.2**). All patients had typical imaging features. We were able to evaluate later serial images for 488 tumors. We found that 215 tumors had regressed, 256 were unchanged, and 19 had enlarged, for a tumor control rate of 97%, at a median of 4 years. Ninety-three percent of patients with these tumors either improved neurologically (n = 87), or remained unchanged (n = 380). Imaging follow-up past 8 and 10 years was obtained in 49 and 22 tumors, respectively, with control rates of 94 and 95%. Past 10 years, 16 patients were stable, three were improved, and three were worse.

Subsequent surgical resection was performed in 51 patients (5.2%) due to local tumor growth or increased symptoms, at an average time of 35 months. Additional fractionated radiotherapy was used in 2.9%, and further radiosurgery was performed in 41 patients (4%), usually for new tumors, at an average of 4 years. No patient developed a radiation-induced tumor.

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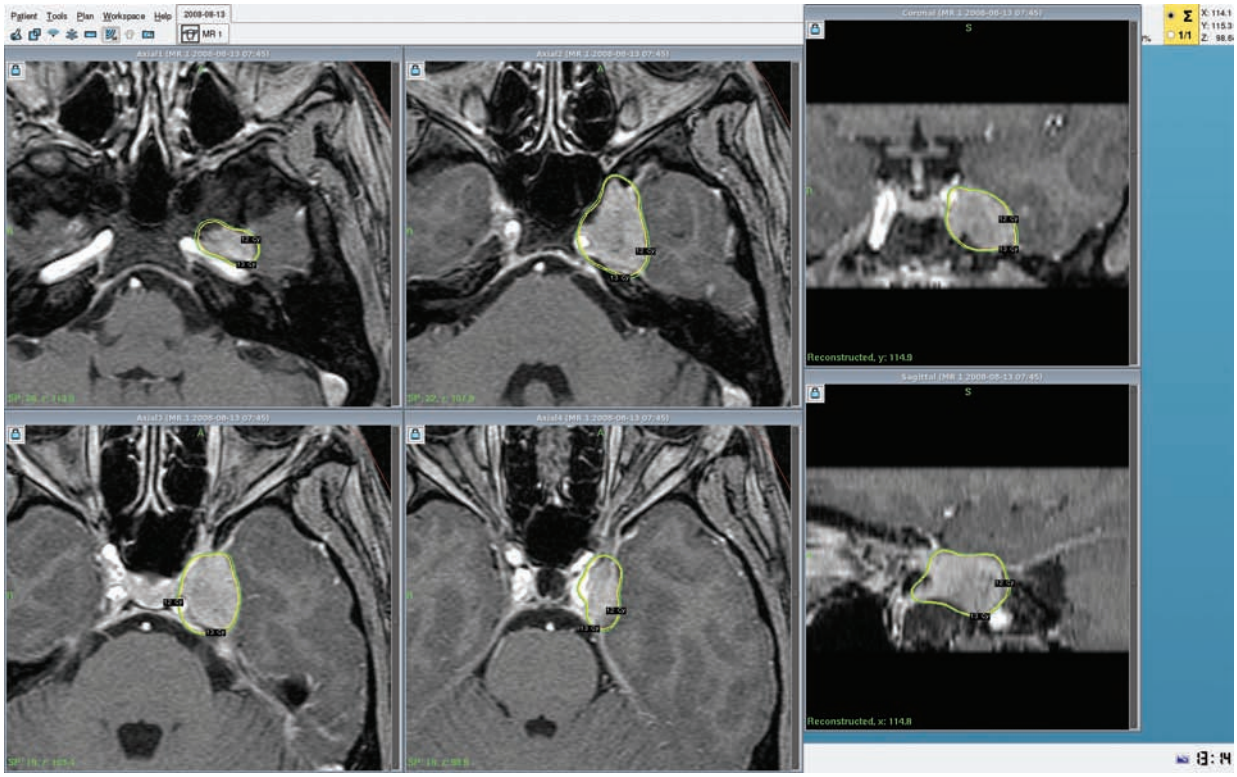


Fig. 41.1 Gamma knife radiosurgical plan in a young woman with a left cavernous sinus meningioma who presented with headache and mild diplopia. Multiple isocenters were used to create this dosage plan for a tumor margin dose of 13 Gy.

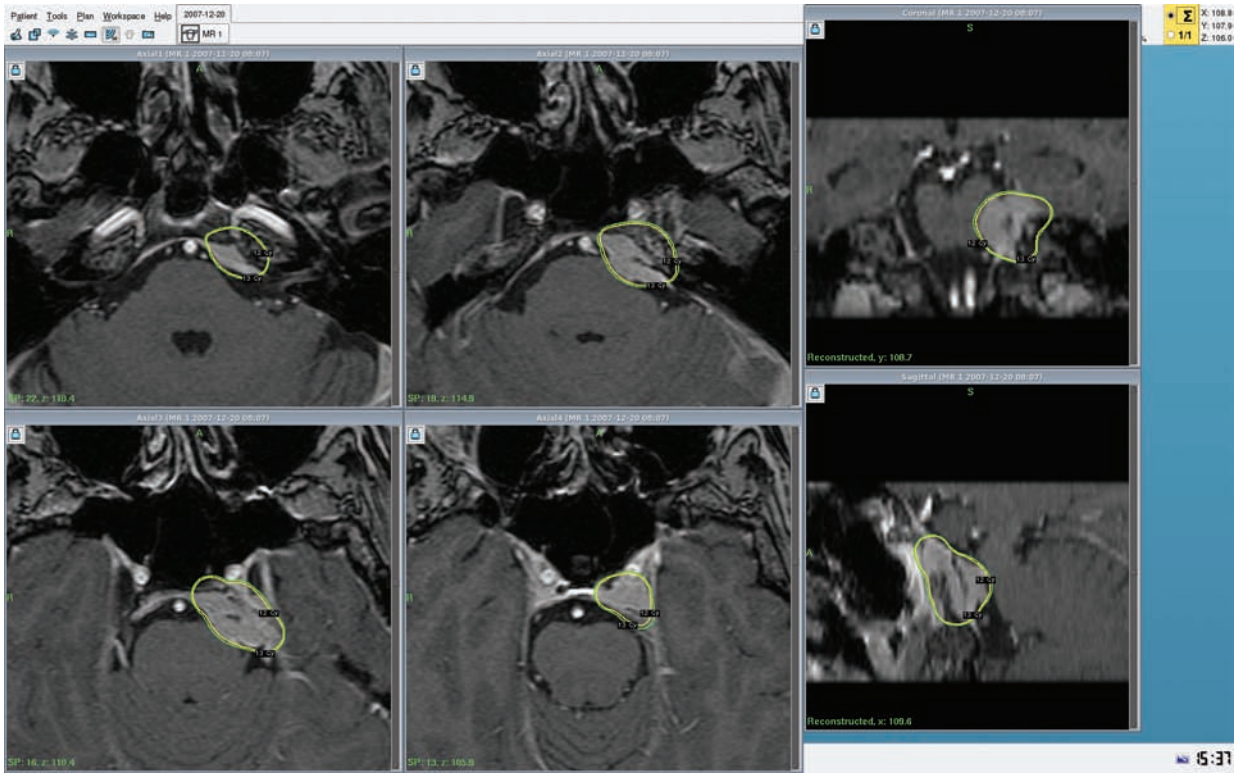


Fig. 41.2 Gamma knife radiosurgical plan for a left petroclival meningioma with mild indentation of the brain stem surface. Multiple 8 and 4 mm isocenters were used.

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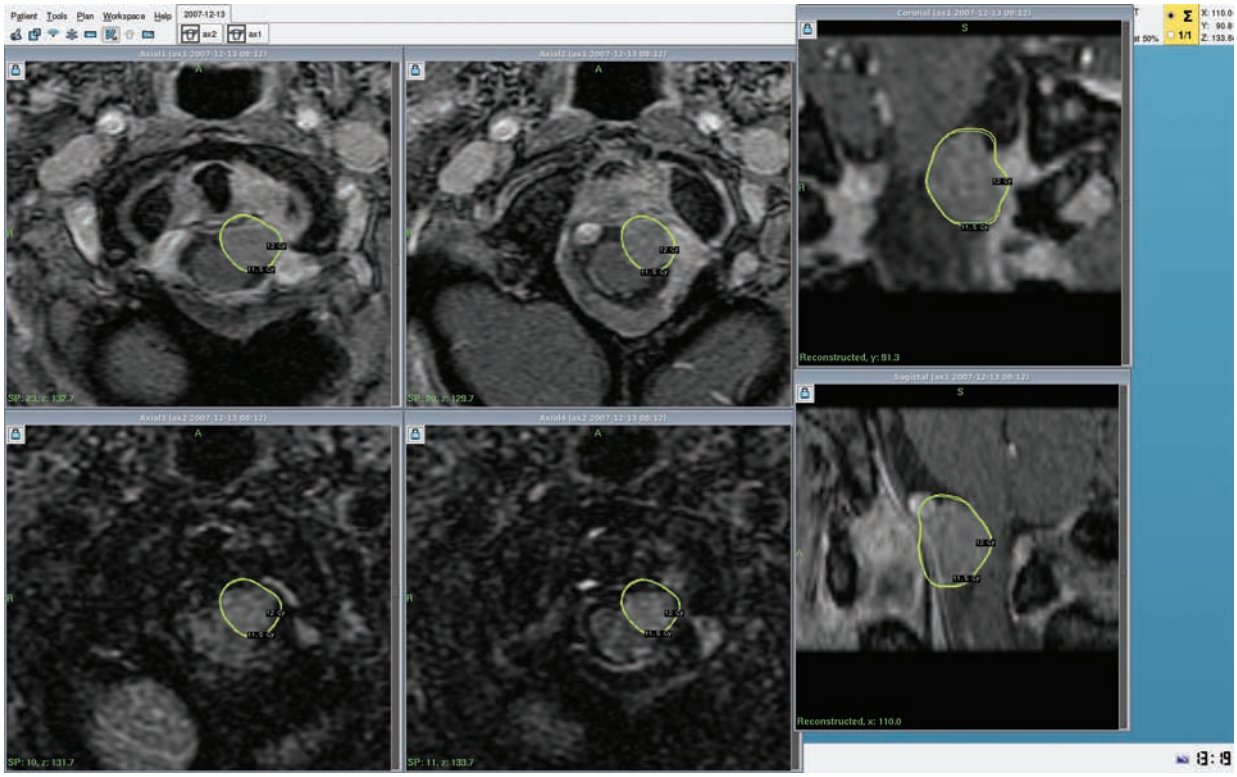


Fig. 41.3 Gamma knife radiosurgical plan for a foramen magnum meningioma in a 77-year-old woman that had enlarged after a period of observation. The tumor margin dose was 11.5 Gy. Tumor regression was noted in follow-up.

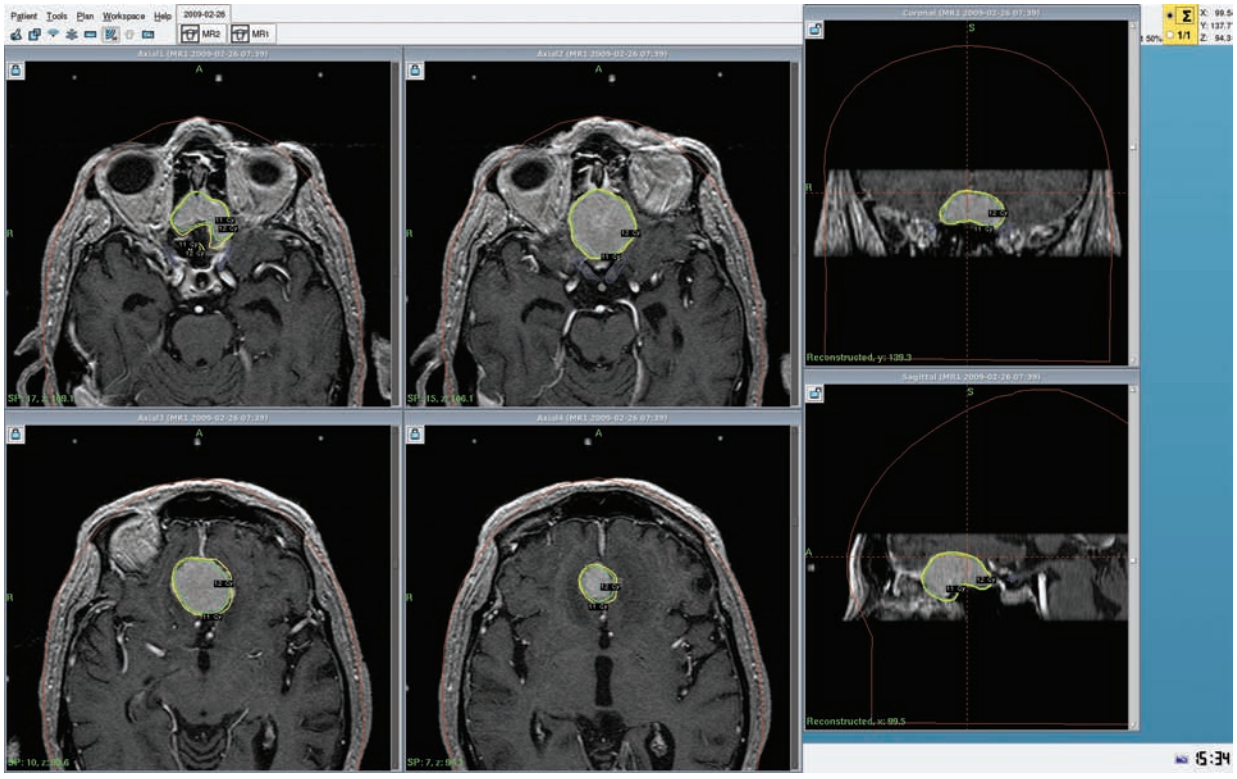


Fig. 41.4 Gamma knife radiosurgical plan in an 80-year-old man with a meningioma from the anterior fossa floor. The tumor had caused headache and mild confusion. The tumor margin dose was 12 Gy.

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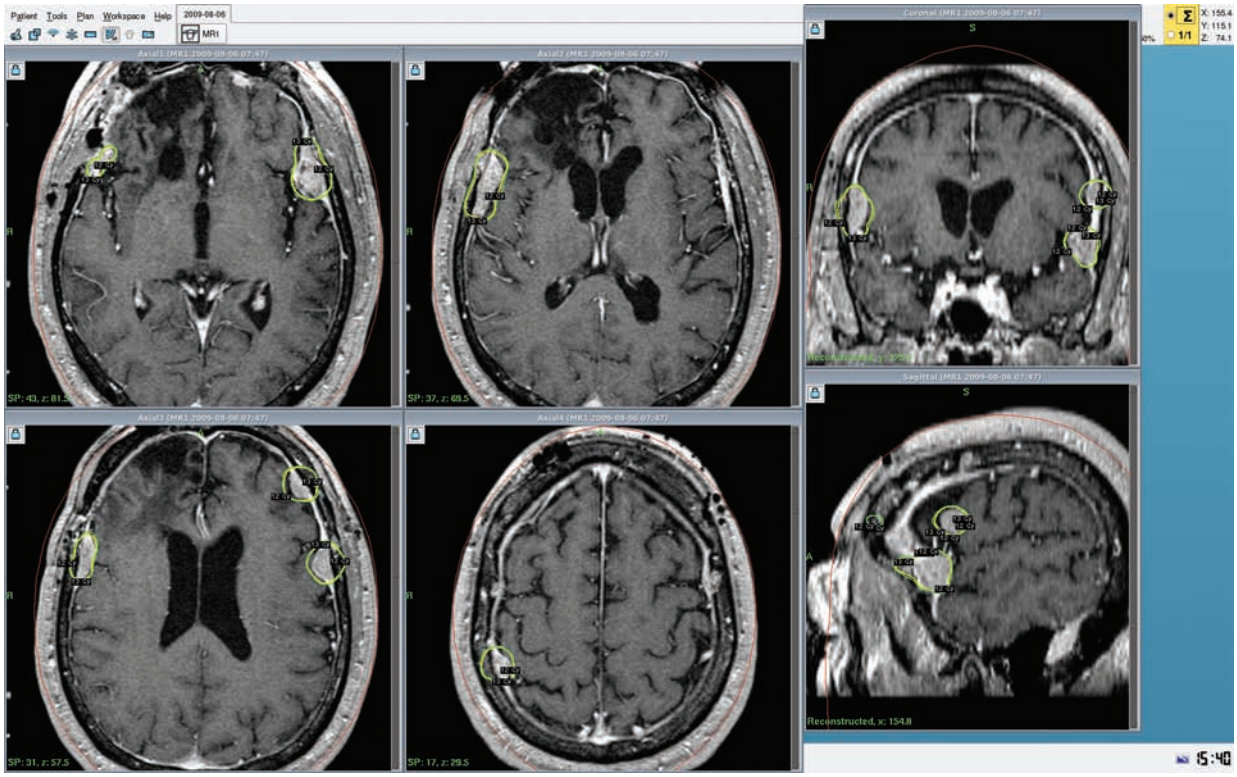


Fig. 41.5 Gamma knife radiosurgical plans for a man with multiple meningiomas after prior resections. Five separate tumors underwent radiosurgery. All were asymptomatic but had grown on serial imaging studies.

The Morbidity of Radiosurgery

After radiosurgery, all patients returned to their preoperative activities immediately. Postradiosurgery nausea or other immediate symptoms were rare, although one patient developed pneumonia 1 week after radiosurgery and died. A complication attributed to radiosurgery was observed in 76 patients (7.7%), at an average time of 11 months. The morbidity rates for cavernous sinus location and parasagittal locations were 6.3 and 9.7%, respectively. For cavernous sinus tumors, these included cranial nerve deficits in 12 patients. Four of these had diplopia, and two had trigeminal neuropathy (one having neuralgic pain). Six patients had decreased visual acuity and four of these had prior treatment that included multiple resections (n = 3), multiple radiosurgeries,[2] radiation therapy (n = 3), or chemotherapy (n = 1).

The 10- and 15-year actuarial complication rates were both 9.1%. Specifically, hydrocephalus was found in 0.4%, a cranial nerve deficit in 3.4%, headaches in 2.2%, seizures in 2.4%, a motor deficit in 1.4%, and a sensory deficit in 0.3%. Complete improvement was noted in 35% of patients who sustained morbidity. Imaging showed interval development of tumoral signal abnormalities determined by long relaxation time MRI in 6.2% of patients, with 4% of the total series having symptomatic imaging changes at an average of 8 months. A ventriculoperitoneal shunt was placed in five patients (0.5%), at an average of 20 months.

Multivariate Analyses

The results of multivariate analyses of survival, disease-free survival, and local control showed that poorer overall survival significantly correlated with increasing WHO grade 0 to III (0 for no tissue diagnosis) [$p < 0.00001$], and increasing volume [$p = 0.04$]. Poorer intracranial tumor control correlated with increasing WHO grade 0 to III, increasing tumor volume, and with multiple meningiomas.

Univariate and multivariate analyses of postradiosurgery complications and new imaging changes on MRI were studied. Both volume and 12 Gy volume significantly correlated with the development of postradiosurgery complications and new T2 changes in univariate testing ($p \leq 0.0001$), whereas in multivariate analysis, volume correlated with complications.

Indications for Radiosurgery

Over the last two decades, the indications for meningioma radiosurgery have expanded to patients with newly diagnosed as opposed to recurrent or residual tumors after initial resection[6,8–23] (**Fig. 41.4**). How good are neurosurgeons in making the diagnosis of a benign meningioma based on imaging alone? In long-term follow-up, we found that the initial imaging-based diagnosis of a benign meningioma was not correct in 2.3% (usually proven to be

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a higher-grade meningioma).[24] As primary management, radiosurgery is not appropriate for patients with large meningiomas, for patients with significant symptomatic mass effect, and for patients with optic nerve sheath tumors and preserved vision. Although some surgeons advocate tumor debulking in symptomatic patients (who are then simply followed to see if the residual tumor enlarges), the literature substantiates high recurrence and progression rates after such management. Condra et al found a 70% rate of tumor progression after subtotal resection without fractionated radiation therapy.[25] Other groups have noted the value of postresection irradiation.[26,27] A recent series found a mean 4 mm annual rate of tumor growth after partial resection of a basal meningioma.[28] We advocate that the entire neoplasm be treated using the most appropriate modalities (when safe and feasible), including complete resection, resection plus radiosurgery for residual tumors, or radiosurgery alone for small-volume tumors in critical locations. We believe that this strategy will provide better long-term outcomes than managing only a portion of the tumor.

Donald Simpson's often-cited report described meningioma recurrence rates based on the degree and description of the resection.[29] He reported a 9% recurrence rate after complete resection of the tumor and its neoplastic dural base, a 19% recurrence rate when the tumor was resected and the dural base only coagulated, a 29% recurrence rate when the tumor itself was removed but the dura could not be excised or cauterized, and an approximate 40% recurrence rate when only a subtotal resection was performed. In the modern era, even higher recurrence rates have been noted[30] (**Fig. 41.5**). Pollock et al studied 136 patients after resection and 62 after radiosurgery with a mean follow-up of 64 months.[31] Their recurrence rates after resection were remarkably similar to the findings of Simpson over 40 years prior. Tumor recurrence was more frequent in the resection group (12% vs 2%; $p = 0.04$). There was no difference in recurrence rates for patients after a Simpson grade I resection, but radiosurgery provided more durable control after grade II ($p < 0.05$) or grades III and IV resections ($p < 0.001$).[31] One could therefore conclude that the results of radiosurgery for a smaller tumor are equal to or better than after a resection, depending on the degree of resection expected. Thus for a resectable smaller-volume meningioma (i.e., convexity, falcine, or anterior fossa location), what should be the main strategy? Other patient factors besides brain location must be considered. We believe that our patient series indicates that radiosurgery can be an effective and safe option for such patients.

Patients undergoing outpatient radiosurgery have not been exposed to the risks related to an open surgical exposure, brain retraction, anesthesia, or a prolonged hospital stay. However, is this of value? In a prior review of 5- to 10-year outcomes in our initial patients, we surveyed patients for their own opinions regarding outcomes.[32] Sixty-five patients answered our questions regarding employment. Twenty-seven patients (42%) were employed at the time of radiosurgery and 20 remained so (74%). Of 35 patients not employed at radiosurgery, five (14%) resumed employment. Patients described their overall level of activity as remaining unchanged in 65%, being increased in 8%, or being decreased in 27%. Radiosurgery was described as a "successful" procedure by 67 of 70 patients (96%).[32]

In conclusion, stereotactic radiosurgery is a powerful biological approach for a patient with an intracranial meningioma. It is well tolerated and can be performed on an outpatient basis.[7] Excellent long-term outcomes can be expected in the majority of patients.

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Chapter 42

Chemotherapy for Intracranial Meningiomas

Marc C. Chamberlain

Introduction

Meningiomas are extraaxial central nervous system (CNS) tumors most often discovered in middle to late adult life. They have a female predominance. Ninety percent of meningiomas are benign, 6% are atypical, and 2% are malignant.[1–4] The majority of patients diagnosed with a symptomatic meningioma undergo surgical resection to relieve neurological symptoms.[1–4] Complete surgical resection is often curative. For the majority of incompletely resected or recurrent tumors not previously irradiated, radiotherapy is administered.[1–4] Radiotherapy may be administered as either conventional external beam irradiation or stereotactically by linear accelerator (LINAC), Leksell Gamma Knife (Elekta, Inc., Norcross, GA) or Cyberknife (Accuray, Sunnyvale, CA) radiosurgery. Advocates of stereotactic radiotherapy have suggested this therapy in lieu of surgery, particularly in poor surgical risk patients, patients with meningiomas in eloquent or surgically inaccessible locations, and patients of advanced age. In the subpopulation of patients with an unresectable meningioma and in whom all other treatments (radiotherapy) have failed, hormonal-chemotherapy-targeted therapy may be considered.[5–30] Notwithstanding limited data, hydroxyurea has been modestly successful in patients with recurrent meningiomas and is frequently the agent of first choice in patients with refractory and recurrent meningiomas of all grades. Emerging targeted therapies may prove useful in refractory meningiomas and are discussed in the context of relevant biology and ongoing clinical trials.

Medical Treatment

Background

Hormonal therapy, chemotherapy, or targeted therapy in the context of treating patients with meningioma is confined to recurrent disease otherwise considered inoperable or refractory to radiotherapy. These various therapies in other cancer settings have been administered in four contexts (i.e., neoadjuvant, chemoradiation, adjuvant, and salvage). When administered before surgery to downstage cancer, such as breast or head and neck cancer, such therapy is termed neoadjuvant. When administered with concomitant radiotherapy, as in the upfront treatment of glioblastoma, it is referred to as chemoradiation. Adjuvant therapy is used with hormonal, chemo-, or targeted therapy and follows resection, as in breast or lung cancer. Lastly, salvage therapy is administered for recurrent disease after failure of up-front treatment usually including surgery, radiotherapy, and chemotherapy. As well, therapy may be conceptually considered as cytotoxic (resulting in net cell kill and radiographic shrinkage) or cytostatic (antiproliferative and radiographically disease stabilizing). At present and as is discussed here, there are no compelling data to suggest a role for hormonal, chemo-, or targeted therapy for meningiomas in the context of neoadjuvant or adjuvant therapy or as a component of chemoradiation. Consequently, these therapies are confined to patients with recurrent meningiomas refractory to radiotherapy and considered inoperable. In addition, contemporary medical therapy of recurrent meningiomas remains at best cytostatic and of relatively short durability (i.e., palliative) when compared with outcomes following surgical resection or radiotherapy. The management of recurrent meningioma has utilized reoperation, radiotherapy, or stereotactic radiosurgery. However, there exists a subset of patients with recurrent meningioma desirous of further treatment in whom both surgical and radiotherapy options have been exhausted. It is in these patients that chemo-hormonal-targeted therapy may be considered.

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Hormonal Therapy

Both epidemiological (female predominance) and biochemical evidence (70% of meningiomas are progesterone receptor positive and 10 to 30% are estrogen receptor positive) suggest meningioma growth may be hormone dependent (**Table 42.1**).[1–9] Additionally, ~60% of meningiomas show staining of prolactin receptors.[1–9] As a consequence, a variety of hormonal therapies have been utilized in the treatment of recurrent benign meningiomas not amenable to further surgery or radiotherapy. The oral progesterone agonist megestrol acetate (megace) was used in a small trial of nine patients with no observed response.[7] Subsequently, in a trial of 14 patients, the progesterone antagonist mifepristone (RU-486) was utilized.[6] Five objective minor responses were seen, though availability of mifepristone limited further study. The Southwest Oncology Group (SWOG) completed a study of mifepristone for unresectable meningiomas (198 total patients, of whom 160 were evaluable).[6] The results did not support a role for RU-486 as compared with placebo (median progression-free survival was 10 months in the RU-486 arm and 12 months in the placebo arm). In addition, SWOG reported on a phase 2 trial of 21 meningioma patients treated with oral tamoxifen, an estrogen receptor antagonist.[5] One patient achieved a partial response, two patients had a minor response, and six patients had stable disease for > 6 months.

There has been interest in the role of growth hormone (GH) on meningiomas since the initial observation that the incidence of meningiomas may be increased in patients with acromegaly.[10] GH secreted by the pituitary gland stimulates the synthesis of insulin-like growth factor-1 (IGF-1) in the liver, and together GH and IGF-1 facilitate normal growth. Growth hormone receptors (GHRs) are ubiquitous in meningiomas, and inhibition of the GHR decreases tumor growth in vitro.[1–4,10] Pegvisomant, a pegylated GH analogue that acts as a competitive antagonist of the GHR, significantly inhibited the growth of meningioma xenografts in nude mice.[10] Tumor IGF-1 concentrations did not vary with pegvisomant treatment, and there was no autocrine IGF-1 production by the tumors. The antitumor effect was thought to be a consequence of decreased IGF-1 in the circulation and/or surrounding host tissues. Direct blockade of the GHR on meningioma cells may contribute to the antitumor effect as well. Whether pegvisomant can inhibit meningioma growth in patients remains to be established.

Table 42.1 Receptors Identified in Meningiomas

Progesterone
Androgen
Glucocorticoid
Growth hormone
Somatostatin
Epidermal-derived growth factor
Insulin-like growth factors 1 and 2
Transforming growth factor-β
Interferon-α
Fibroblast growth factor-1
Estrogen
Prolactin

Biochemotherapy

Recombinant interferon-α (IFN-α) has been found to inhibit the growth of cultured human meningioma cell lines in vitro.[11,12] Four small reports, two in abstract, have been published.[12] In the largest report, 35 patients with recurrent unresectable and previously irradiated meningiomas were treated. IFN-α was considered a reasonable alternative therapy for refractory recurrent meningioma based on the data already presented, in addition to evidence suggesting antiproliferative, immunomodulatory, and antiangiogenic properties.[12] Toxicity was modest in this trial and consisted mainly of fatigue that resulted in dose reduction (seven patients), institution of psychostimulant medication (10 patients), and early discontinuance of therapy (three patients).[12] Based on neuroradiographic response, it appears that IFN-α functions as a cytostatic antimeningioma agent because there was no evidence of either a complete or partial radiographic response; rather, the best response seen was stable disease. Nonetheless, meaningful palliation was achieved, as reflected by a 6-month progression-free survival (PFS) of 54%, surpassing the study primary objective of 40% PFS at 6 months. Although no radiographic responses were seen, 74% demonstrated stable disease with a median PFS of 7 months (6-month and 12-month PFS were 54% and 31%, respectively). Median overall survival was 8 months (range 3 to 28 months).

Hydroxyurea, an oral ribonucleotide reductase inhibitor, arrests meningioma cell growth in the S phase of the cell cycle and induces apoptosis.[13–17] In a preliminary report by Schrell et al, hydroxyurea (1000 to 1500 mg/day; 20 mg/kg/day) decreased tumor size in three patients with recurrent benign meningiomas and prevented recurrent disease for 24 months in a patient with a completely resected malignant meningioma.[13] Several more recent studies (**Table 42.2**) suggest that hydroxyurea has modest activity; responses are uncommon but some patients appear to have disease stabilization.[14–17] Additionally, hydroxyurea has been demonstrated to have modest and acceptable toxicity in patients with recurrent meningioma manifested primarily as fatigue and treatment-related anemia. SWOG conducted a phase 2 study to further evaluate the role of hydroxyurea in meningiomas (SWOG-S9811). This study is closed to accrual but the final results are not yet available. Problematic with the various hydroxyurea trials, however, is that many pa-

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Table 42.2 Hydroxyurea for Recurrent Meningioma

First Author	No. (benign)	Prior Radiotherapy	Response	Median TTP (months)	Toxicity (≥ grade 3)
Newton[15]	17(13)	41%	SD (88%)	20	25% (15%)
Mason[14]	20 (16)	40%	SD (60%)	30	15%
Rosenthal[17]	15 (5)	7%	SD (73%)	10	27% (20%)
Hahn[9]	21 (4)	21 (concurrent)	SD (52%)	14	53% (0%)
Loven[16]	12 (8)	50%	SD (8%)	13	33% (25%)

Abbreviation: SD, standard deviation.

tients had not failed radiotherapy or that radiotherapy was administered concurrently.

Rengel has demonstrated that the calcium channel antagonists verapamil, nifedipine, and diltiazem can block in vitro and in vivo meningioma growth at clinically relevant doses.[18] However, only modest growth inhibition was exhibited in the tumors in these studies with calcium channel antagonists alone. Additionally, many authors have shown augmented growth inhibition by adding calcium channel antagonists to traditional chemotherapies in other tumor types.[18] Calcium channel antagonists seem to exert the majority of their antitumor effects by inhibiting calcium-dependent secondary messenger systems.[18] Recently, Jensen has demonstrated that the use of verapamil or diltiazem with hydroxyurea enhances the growth inhibition of meningiomas seen with these drugs in vitro and in vivo. A clinical trial combining hydroxyurea and verapamil testing the aforementioned hypothesis for recurrent meningioma has recently opened and is actively accruing patients (**Table 42.3**).

In a recent prospective phase 2 study of temozolomide (a DNA-damaging agent that methylates the O^6 position of guanine), an oral agent with modest toxicity and known activity in a variety of gliomas, no clear activity was seen in patients with refractory meningioma no longer considered operable or having progressed following radiotherapy.[19] The schedule of temozolomide (TMZ) attempted to optimize tumor drug exposure by exploiting a chronic daily schedule (75 mg/m^2/day for 42 days administered every 56 days) permitting a nearly 2.5-fold increase in TMZ dose intensity as compared with the standard 5-day schedule given every 28 days.[19] Notwithstanding these presumed pharmacological benefits, no patients demonstrated PFS at 6 months, the primary study objective. As a consequence, the study was terminated following enrollment of the first 16 patients. Based on the results of this study, TMZ does not appear to have activity against recurrent meningiomas, suggesting a need for further phase 1/2 chemotherapy trials for this common primary brain tumor.

A recent prospective phase 2 study used CPT-11 (Camptosar [Pfizer, Inc., New York, NY]; irinotecan), an intravenous topoisomerase 1 inhibitor with modest toxicity and activity in intermediate-grade glial tumors, for refractory recurrent World Health Organization (WHO) grade I meningiomas progressing despite prior surgery and radiotherapy.[20,21] The trial was based on significant activity seen in vitro, suggesting antimeningioma activity.[20] In addition, this study differs from hydroxyurea-based chemotherapy trials of recurrent meningioma mentioned earlier in that all patients had evidence of progressive disease despite previous surgery (50% of patients underwent a second resection) and radiotherapy (all patients received prior external beam radiotherapy and 35% were treated in addition with stereotactic radiotherapy). Though only 50% of patients had reconfirmation of initial pathology, it seems reasonable to assume that the patients in this series are representative of patients with treatment-refractory recurrent meningiomas. The schedule of CPT-11 utilized (once every 3 weeks) is one of two commonly used; however, no differences with respect to tumor response between weekly versus every-3-weeks schedules of CPT-11 administration are seen.[21] Toxicity was manageable using this CPT-11 schedule, as reflected by no patient requiring a CPT-11 dose reduction, no delays in therapy, and no treatment-related deaths. This suggests that this regimen may be used with moderate toxicity not otherwise affecting treatment. Nonetheless, only a single patient demonstrated PFS at 6 months, the primary study objective. As a consequence, the study was terminated following enrollment of the first 16 patients. Based on the results of this study, CPT-11 does not appear to have activity against recurrent WHO grade I me-

Table 42.3 Recurrent and Refractory Meningiomas, Phase 2 trials in the United States

Hydroxyurea + verapamil
Hydroxyurea + imatinib
Pasireotide
Sunitinib
Vatalanib
Bevacizumab

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ningiomas, suggesting a need for further trials with other agents for this common primary brain tumor. However, because no aggressive or malignant meningiomas were treated, these results should not be extrapolated to this patient population.

Multidrug chemotherapy trials for recurrent meningiomas, whether aggressive, malignant, or refractory to surgery and radiotherapy, are scant.[8,9,22] The best-documented chemotherapy regimen (cyclophosphamide, adriamycin, and vincristine) has been used primarily in an adjuvant setting for the treatment of malignant meningiomas; however, without a control group, the results are difficult to interpret.[22] Other published regimens do not report response rates, length of response, or toxicity data and therefore should be regarded as investigational.[8,9,22] Unpublished data from a small number of patients from a phase 2 SWOG trial for aggressive meningeal tumors and malignant meningiomas with ifosfamide/mesna disulfide did not show initial promise.

Targeted Therapy

In contrast to the increasing understanding of the molecular pathogenesis and biology of systemic malignancies, including gliomas, relatively little is known about the molecular pathogenesis of meningiomas and the critical molecular changes promoting meningioma growth.[1–4,9,23] Overexpression of several growth factors, including platelet-derived growth factor (PDGF), epidermal growth factor (EGF), and vascular endothelial growth factor (VEGF) and their receptors, and signal transduction pathways, such as the Ras/mitogen-activated protein kinase (MAPK), phosphatidylinositol-3-kinase (PI3K)-Akt, and phospholipase C (PLC)-γ1-protein kinase C (PKC) pathways, have been implicated, but their relative significance is largely unknown.[1–4,9,23] As a result, the most important molecular targets for meningioma-targeted therapy remain unknown.

PDGF is a fundamental driver of cell proliferation in normal development and in a variety of pathological conditions, including cancer.[1–4,9,23] Accumulating evidence suggests that PDGF plays an important role in meningioma growth.[1–4,9,23] The majority of meningiomas of all histological grades express PDGF ligands AA and BB and the PDGF-β (PDGF-β) receptor. Expression levels appear higher in atypical and malignant meningiomas than in benign meningiomas. Laboratory data suggest that an autocrine PDGF loop supports meningioma cell growth and maintenance. These data suggested a sound rationale for testing PDGF inhibitors in meningioma patients. Imatinib is a potent inhibitor of the Bcl-Abl, PDGF-α and β receptors, and c-Kit tyrosine kinases.[24] Its ability to inhibit PDGF receptors (PDGFr) with an IC_{50} of 0.1 μM suggested that it may have therapeutic potential in meningiomas. The North American Brain Tumor Consortium (NABTC) conducted a phase 2 study of imatinib in patients with recurrent meningiomas.[24] Patients were stratified into two cohorts: (1) benign meningiomas or (2) atypical and malignant meningiomas. Because imatinib is metabolized by the cytochrome P450 system, patients could not be receiving enzyme-inducing antiepileptic drugs. Patients initially received 600 mg/day of imatinib; the dose was increased in the second cycle to 800 mg/day if no significant toxicity was observed in the first cycle. Twenty-three patients were enrolled into the study (13 benign meningiomas, five atypical meningiomas, and five malignant meningiomas). Although the treatment was well tolerated, imatinib had minimal activity. Of the 19 patients evaluable for response, 10 progressed at the first scan and nine were stable. There were no radiographic responses. Overall median PFS was 2 months (range 0.7 to 18 months); 6-month PFS was 29.4%. For benign meningiomas, median PFS was 3 months; 6-month PFS was 45%. For the atypical and malignant meningiomas, median PFS was 2 months; 6-month PFS was 0%. A study of 22 patients with recurrent meningioma treated with hydroxyurea and imatinib has recently completed and results are pending (**Table 42.3**).

The EGF receptor (EGFr) is overexpressed in more than 60% of meningiomas.[1–4,9,23] EGF and transforming growth factor-α (TGF-α) activate these receptors and stimulate meningioma growth in vitro, supporting the concept that activation of EGFr in human meningiomas by autocrine/paracrine stimulation may contribute to their proliferation. Increased TGF-α immunoreactivity in meningiomas has been associated with aggressive growth. The NABTC has conducted two trials of EGFr inhibitors in recurrent meningiomas using either gefitinib (Iressa, AstraZeneca Pharmaceuticals, Wilmington, DE; 500 mg/day) or erlotinib (Tarceva, OSI Pharmaceuticals, Inc., Melville, NY; 150 mg/day).[25] A total of 25 patients were entered on trial. In both studies, the drugs were well tolerated; the main toxicities were the expected adverse effects of rash and diarrhea. Nonetheless, there were no objective responses and PFS-6 was 25%. Based on the results of this study, neither EGFr inhibitor appears to have significant activity against recurrent meningioma.

Although EGFr monoclonal antibodies have been effective for some systemic malignancies (e.g., cetuximab in colorectal and head and neck cancer), these agents have generally not been used for primary brain tumors because of the concern regarding the ability of these agents to penetrate the blood–brain barrier (BBB) in sufficient concentrations to produce a therapeutic effect. Because the BBB is not a factor in drug delivery to meningiomas, it is possible that monoclonal antibody-based therapy may be effective in these tumors. To date, very few studies have evaluated EGFr monoclonal antibodies in recurrent meningiomas.[26] In a phase 1 study of a murine monoclonal antibody against EGFR in nine patients with either recurrent gliomas or meningiomas, treatment was reasonably well tolerated. No radiographic responses were detected, but efficacy data are difficult to interpret in a study with so few subjects. Currently, several anti-EGFr monoclonal antibodies are undergoing evaluation for other malignancies, including cetuximab, panitumumab, EMD 72000, nimotuzumab, and mAb 806. Trials of these agents in meningiomas likely will emerge and be combined with correlative studies examining whether the

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antibodies can achieve therapeutic concentrations in vivo and inhibit meningioma EGFr activity in vivo.

Many tumors express somatostatin receptors, providing a molecular rationale for utilizing long-acting somatostatin analogues for therapeutic and diagnostic purposes.[27,28] ^{111}Indium-DTPA octreotide is useful for in vivo imaging of somatostatin receptor–positive neuroendocrine tumors as well as meningiomas.[28] Among brain tumors, meningiomas show the highest frequency of somatostatin receptor expression.[27] The largest study, comprising 52 patients with intracranial meningiomas studied by somatostatin scintigraphy, indicated a 90% positive rate of detection.[28] In another study by the same group, postoperative somatostatin scintigraphy, was complementary to cranial magnetic resonance imaging (MRI) and in many instances (> 50%) of cases suggested residual tumor not evident by MRI.[28] A previous study evaluated as a treatment escalating doses of long-acting analogue octreotide in three patients with unresectable meningiomas.[28] No objective radiographic response was demonstrated; however, therapy was limited to a median of 7 weeks of therapy. A second report, communicated in letter format, described relief of headache in three patients with unresectable meningiomas.[28] A final paper described objective visual improvement without radiographic tumor shrinkage in a patient with a sellar meningioma.[28] Somatostatin receptors, especially the sst2A subtype, are present on most meningiomas, although their functional role remains unclear.[27] The addition of somatostatin inhibits meningioma growth in vitro in most studies but increases meningioma proliferation in some. In the largest trial, 16 patients with recurrent meningiomas (progressive after prior surgery and radiotherapy) shown to overexpress somatostatin receptors by octreotide scintigraphy were treated with monthly long-acting somatostatin (Sandostatin LAR [Novartis, Basel, Switzerland]).[28] Thirty-one percent of the patients demonstrated a partial radiographic response and 44% achieved PFS at 6 months with minimal toxicity. New somatostatin analogues with higher affinity may offer a novel, relatively nontoxic alternative treatment for patients with recurrent meningiomas. SOM230C (pasireotide) is a novel, intramuscularly administered, long-acting somatostatin analogue with a wider somatostatin receptor spectrum (including subtypes 1, 2, 3, and 5) and higher affinity (particularly for subtypes 1, 3, and 5) than the sustained-release somatostatin described earlier. A phase 2 trial for patients with recurrent or progressive meningiomas using pasireotide (60 mg intramuscular once every 4 weeks) has opened and is accruing patients (**Table 42.3**).

Meningiomas are highly vascular tumors that derive their blood supply predominantly from meningeal vessels supplied by the external carotid artery, with additional supply from cerebral pial vessels.[1–4,9,23,29,30] Inhibition of angiogenesis has become an increasingly important approach to treating cancer.[1–4,9] VEGF plays a central role in tumor angiogenesis, and there is increasing evidence that inhibition of VEGF or VEGF receptors (VEGFr) can lead to significant antitumor effects. Inhibition of VEGF with the anti-VEGF antibody bevacizumab (Avastin, Genentech, Inc., South San Francisco, CA) has significantly improved survival in several malignancies, including colorectal, lung, and breast cancer.[1–4,9] Inhibitors of VEGFr, such as sorafenib (Nexavar, Bayer HealthCare Pharmaceuticals, Berkeley, CA) and sunitinib (Sutent, Pfizer, Inc., New York, NY), have also prolonged survival in renal cell carcinoma and gastrointestinal stromal tumors (GISTs).[1–4,9] VEGF and VEGFr are expressed in meningiomas, and the level of expression increases with tumor grade.[9] VEGF expression is increased twofold in atypical meningiomas, and 10-fold in malignant meningiomas compared with benign meningiomas. VEGF also plays an important role in the formation of peritumoral edema, which adds to the morbidity of these tumors.[1–4,9] Inhibitors of VEGF and VEGFr are promising agents in meningiomas, with the potential not only to inhibit angiogenesis but also to decrease peritumoral edema (**Table 42.3**). Studies evaluating inhibitors of angiogenesis in meningiomas are limited. Two small trials reported in abstract utilized small molecule inhibitors for recurrent meningioma.[29,30] In one trial of antiangiogenic inhibition, sunitinib, a multifunctional tyrosine kinase inhibitor with anti-VEGFr and PDGFr activity, with 20 recurrent or inoperable meningioma patients accrued to study, 1/17 (5%) showed a partial radiographic response and 13/17 (65%) demonstrated stable disease.[29] Of all patients studied, nearly one third required dose modification due to drug-related toxicity. Median PFS is 7 months; however, accrual is incomplete because the study is designed to assess 20 grade I meningioma patients and 20 grade II or III meningioma patients. A second trial of 25 patients with recurrent meningioma of any grade, utilizing PTK787 (vatalanib), another multifunctional tyrosine kinase inhibitor with anti-VEGFr activity, reported 25% stable disease as the best response.[30] This trial has recently closed and outcome data are being collated. Another trial just opening will study recurrent meningioma of all grades treated with single-agent bevacizumab (Avastin, Genentech), a VEGF ligand monoclonal antibody, in a prospective phase 2 design.

Conclusion

In summary, meningiomas are benign extraaxial CNS tumors, which when symptomatic are typically treated with definitive surgical resection. Several trials of chemotherapeutic and hormonal agents for progressive or recurrent meningioma have been reported and are ongoing. The aforementioned studies should be interpreted with caution because no large cohorts have been studied nor has the therapy been shown to cause regression of disease. Also, "stability" must be scrutinized carefully given the natural biology and inherent slow rate of growth of these tumors. Furthermore, these studies have not consistently treated patients having failed both surgery and radiotherapy. Clearly there is a need to develop new biological, genetic, and chemotherapeutic options for recurrent meningiomas that have exhausted surgical and radiation treatment options. Future treatments will

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be based upon an improved understanding of the molecular biology of meningiomas, better in vitro models, and novel therapeutics, including chemotherapeutic agents based on mechanism of action, monoclonal antibodies targeted to meningioma cell-surface receptors, and targeted therapies that either antagonize surface receptors or ligands involved in cell growth and molecular agents that interfere with cell signaling.

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XI

Special Considerations

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Chapter 43

Meningioma Surgery: Experience, Volume of Care, and Patient Outcome

Fred G. Barker II, Patrick J. Codd, and William T. Curry Jr.

Harvey Cushing, the father of modern neurosurgery and the man who coined the word *meningioma*, decided early in his surgical career that he was going to devote special attention to treating and studying brain tumors. As he gained experience in his chosen specialty, he felt even more strongly that surgical progress had come to depend on surgeons' subspecialization.[1] As he wrote in 1905, justifying the creation of the new field called neurological surgery, "Are practice of hand and concentration of thought to go for nothing?"[2] Geoffrey Jefferson identified this confidence as central to Cushing's surgical philosophy as a whole:

> He was almost frightening in his belief that whatever the lesion was (unless hopelessly malignant) he could always show vastly better results in the next five-year period, frightening because such confidence seemed to invoke the wrath of the gods. Yet time proved that he was right.[3]

Time *has* proved that he was right: Cushing's own career in meningioma surgery proved to be a powerful demonstration of the cumulative effects of surgical experience on patient outcomes. By concentrating on brain tumor surgery and the perioperative care of brain tumor patients, he was able in the first half of his career (1908 to 1926) to show a 47% reduction in long-term mortality after meningioma craniotomy compared with Victor Horsley's results in the decades before him. In the second half of his career, Cushing's patients' mortality was further reduced by an additional 41%.[4]

The idea that surgical experience might influence patient outcomes has been studied using several related paradigms. As predictors of patient outcome, we can potentially study: first, the current volume of care (surgical caseload) for a provider (surgeon or hospital); second, the cumulative lifetime experience of the surgeon (the surgical learning curve); third, whether the surgeon has had special training, has gained certification in a given field, or belongs to specialized professional societies; fourth, the degree to which a surgeon concentrates his or her efforts on a certain type of surgery (specialization); and fifth, whether the surgeon practices as part of a specialized team (which may be multidisciplinary). This chapter summarizes the evidence for these effects in meningioma surgery and allied operations.

Surgical Caseload and the Volume-Outcome Effect

The observation that patients who receive care from high-volume providers typically have better outcomes compared with those treated by low-volume providers is called the volume-outcome effect.[5,6] The volume-outcome effect was first observed in neurosurgery in relation to aneurysm care[7] and has been subsequently confirmed for many other types of neurosurgical procedures, including craniotomy for many individual types of tumor.[8–15]

Because demonstration of the volume-outcome effect requires knowledge of outcomes across many providers (hospitals or surgeons), it is usually studied using administrative databases. This has some specific consequences that affect the design and interpretation of volume-outcome studies. First, outcome measures in such studies are not usually disease-specific. This means that mortality, adverse hospital discharge disposition, or cost measures, such as length of stay or hospital charges, are more likely to be the endpoint of an administrative database study, rather than extent of resection, specific neurological outcomes, or disease-free survival. Second, volume-outcome studies are nonrandomized. This means that if patients who obtain care from low-volume providers differ systematically from those who are cared for by high-volume providers, the study will be biased. Accurate risk

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adjustment for such "casemix differences" are critical in volume-outcome studies. Third, use of administrative databases also typically forces the use of coding systems that have not been designed to study specific diseases, and may have other shortcomings. In meningioma studies, for example, one consequence is that many studies cannot distinguish hemiparesis on admission from weakness that results from surgery. Vision loss is likely to be crudely coded as affecting one or both eyes, rather than specific information about acuity or fields. Many complications and adjunct procedures, such as embolization, may be systematically undercoded in administrative databases.

Despite these drawbacks, studies on short-term outcomes after meningioma surgery[8,12] have shown better patient outcomes after surgery at higher-volume hospitals or from higher-volume surgeons. Curry et al[8] studied 15,028 admissions for meningioma craniotomy in the United States from 1988 to 2000. In multivariate analysis adjusted for multiple casemix factors, they found significantly lower in-hospital mortality after surgery at higher-volume hospitals ($p = 0.01$) (**Fig. 43.1**). There was a 26% reduction in mortality for each 10-fold increase in hospital case volume. The highest-volume quintile of hospitals in this study had 24 or more meningioma craniotomies per year. Bateman et al reported similar results in a cohort of adult meningioma patients treated from 1998 to 2002.[12] Surgeon volume had no effect on mortality in the study by Curry et al, but both hospital and surgeon caseload were significantly ($p < 0.001$) related to less frequent adverse hospital discharge disposition (i.e., other than directly home). For most individual complications studied (such as hematomas, thrombotic complications, and transfusions), there were no differences between high- and low-volume providers. Paradoxically, neurological complications were more frequent with high-volume hospitals and surgeons. This could reflect a more complex case mix treated by high-volume providers. Only 6% of neurological complications were followed by in-hospital death at high-volume hospitals, compared with 19% at low-volume hospitals; this effect (better "rescue" at high-volume hospitals) has been identified in studies of general surgical procedures as well.[16]

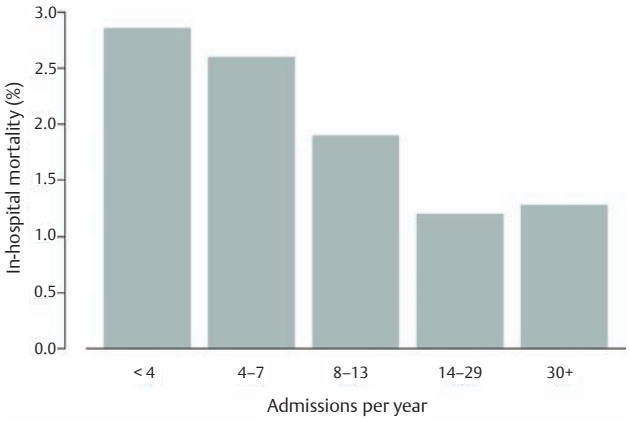


Fig. 43.1 Bar graph showing in-hospital mortality as a function of hospital annual case volume after craniotomy for meningioma in the United States, 1988 to 2007. Hospitals were grouped into quintiles by volume; mortality was lower at larger hospitals. Data from Nationwide Inpatient Sample (Healthcare Cost and Utilization Project, Agency for Healthcare Research and Quality, Rockville, MD), using methods from Curry et al.[8]

Other studies of short-term outcomes after brain tumor craniotomies have identified similar volume-outcome effects after surgery for primary brain tumors,[9] metastases,[11] acoustic neuromas,[17,18] adult brain tumors as a whole,[13–15] and pediatric brain tumors.[10] Most brain tumor volume-outcome studies to date have been from the United States, although in other medical areas the effect has been demonstrated in many different national health care systems.

The volume-outcome effect has usually been explained as a combination of the well-established existence of a learning curve for most surgical procedures ("practice makes perfect," see later discussion) and from the systematization of care in the form of formal checklists or less formal habits that accompany any frequent activity.[19] Other possible explanations for better outcomes at higher-volume hospitals include "structural" factors describing hospital resources, such as the presence of advanced operating room technology, dedicated intensive care units and intensivists, 24-hour resident coverage, and the number and level of training of nurses.[20–22] Finally, the "perfect makes practice" or "selective referral" effect describes the ability of established high-volume centers to attract good-prognosis patients from a broad area, whereas lower-volume hospitals are more likely to receive the sickest, most emergent patients with a given diagnosis from a limited geographical catchment area.[23] For example, lower-volume meningioma centers and surgeons treated a significantly higher proportion of patients with emergent or urgent presentation in the study by Curry et al (although conversely, complex patients with neurofibromatosis 2 tended to receive care at high-volume referral centers).[8]

Some surgeon-specific factors may also help to explain the volume-outcome effect. Integrated Medical Learning (IML) results from the 2007 CNS meeting demonstrated that surgeons with high-volume tumor practices displayed significantly greater knowledge about current neurooncology publications.[24] Of four questions in this survey study on current literature knowledge, 54% of surgeons who saw fewer than 10 brain tumor patients per year scored zero correct, whereas 75% of surgeons who saw 50 brain tumor patients or more per year answered all questions correctly.

Regardless of the causes of the volume-outcome effect, can it be used as a tool for improving the quality of meningioma surgery in a population? Most answers to this type of question for other specific operations have taken one of two forms. First, demonstration of a volume-outcome effect for a procedure is often followed by a call to "regionalize" the procedure by restricting its performance to high-volume centers.[25,26] Such initiatives have been performed by government mandate for some expensive, high-risk procedures, with mixed results.[27–30] Regionaliza-

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tion can cause disadvantageous, unintended consequences, such as increased difficulty coordinating care with local primary providers, longer travel time for patients, and the general deterioration of skills in the general community when emergency care is necessary without transfer to regional centers; in addition, some patients simply prefer to receive care close to home.[31–34] Careful study of both intended and unintended consequences is necessary before mandating regionalization of a procedure.[35] That said, spontaneous regionalization of some neurosurgical procedures, including meningioma surgery, seems to be already taking place in the United States, following trends in other major surgical procedures such as cancer surgery.[36,37] Between 1988 and 2000, the median annual hospital caseload of meningioma craniotomies in the United States increased from six to 10 cases per year; in 2007, the increase had continued to 15 per year (Barker FG, unpublished data, Nationwide Inpatient Sample [NIS], Healthcare Cost and Utilization Project, Agency for Healthcare Research and Quality, Rockville, MD). In 1988, 10% of meningioma patients underwent surgery at hospitals where there was no other meningioma craniotomy that year, compared with 3.5% in 2000 (2.8% in 2007; Barker FG, unpublished data, NIS).[8] Similar spontaneous centralization (i.e., not in response to government mandate) took place in the United States between 1988 and 2000 for pediatric tumor craniotomies[10] and craniotomies for adult primary brain tumor.[9] The forces causing these shifts in practice have not been studied.

The other general way to use the volume-outcome effect to improve care is to study processes of care that characterize high-volume centers and care delivered by specialists[38] and use them to improve care at low-volume centers.[39,40] This is sometimes called "floating all boats." It is possible that the periodic published reports of "good outcomes at low-volume aneurysm centers"[41–44] may result from conscious or unconscious applications of this strategy. At the individual practitioner level, a parallel is the high-quality care delivered by low-volume surgeons who have participated in a formal credentialing process, as in the context of a clinical trial.[45–48] For meningioma care, processes that characterize high-volume providers have not been studied, nor have there been published reports of high-quality care achieved specifically at low-volume centers. In general terms, one example of possible meningioma process research would be to study the use of a fixed protocol for meningioma surgery resembling the "surgical checklists" that have been studied in general surgery.[19] Postcraniotomy infection has been shown to be more common in brain tumor craniotomy than after other craniotomies,[49] and a standardized protocol ensuring timely administration of antibiotics[50] could easily be developed and tested. Protocols to retain patients through long-term follow-up periods after surgery could also be studied.

The demonstration that higher-volume providers are associated with better outcomes offers the possibility to use volume as a surrogate for quality of care, allowing investigators to ask questions about social variables (such as race and income) that could predict better access to high-quality care for meningiomas. In 2003, Curry et al demonstrated that high-volume meningioma surgery was associated with white race, private insurance, residence in higher-income areas, and lesser general medical comorbidity in the United States, 1988 to 2000.[8] These disparities have demonstrable impact on patient outcomes: In the United States, 1988 to 2004, the same group demonstrated higher inpatient mortality for black patients compared with whites after meningioma craniotomy (2.7% vs 1.9%).[51] The difference in mortality was significant when meningioma results were pooled with results for primary brain tumors, metastases, and acoustic neuromas, with an odds ratio of 1.6-fold higher mortality for blacks compared with whites ($p < 0.001$).[51]

Meningiomas and the Surgical Learning Curve

The surgical learning curve is the progressive improvement in patient outcomes that results when a single surgeon gains larger experience with a specific surgical procedure.[52] The acquisition of skills through repeated practice has been studied in many fields and probably has both cognitive and physical performance domains. Research has suggested that the aspect of practice that contributes most to improved performance is "directed practice," in which an individual attempts tasks beyond the present capacity and learns from mistakes.[53–55] Clearly, generalizing this model to clinical surgery could be associated with more frequent poor outcomes early in an individual's cumulative experience. Based on results in other fields, expert performance is not typically expected to be attained until after 10,000 hours or 10 years of repeated practice. However, the existence of significant learning after many fewer hours than this has been demonstrated for some surgical procedures. For example, when physicians experienced in endovascular procedures learned aneurysm coiling, improved results were seen after as few as five cases.[56] In the International Subarachnoid Aneurysm Trial (ISAT) study, a minimum of 30 endovascular cases was required for physicians to participate in the trial.[57]

Lee and Sade studied the learning curve in a single surgeon's meningioma practice of 600 cases over 13 years.[58] They noted progressive improvement during the series, from a surgical complication rate of 22% in the first third of the series to 13.5% in the last third, as well as improved outcomes—92% Glasgow Outcome Scale 5 in the last third of the series, compared with 83.5% in the first third. Major neurological complications were experienced by 12.5% of patients in the first third of the series and 4% in the final third. The study did not show a "plateau" in results during the series, leaving open the possibility of further improvement with additional experience. This parallels Cushing's progressively improved results during his career mentioned earlier. In a study on 28 tuberculum sellae meningiomas, Kitano et al reported better visual results in the latter part of the series, but the authors changed technique during the study period, which could also have accounted for the results.[59] Importantly, studies

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have shown a nationwide trend over time toward progressively decreasing in-hospital mortality for meningioma craniotomies, presumably at least partially because of improvements in diagnosis and perioperative care (**Fig. 43.2**). This broad trend must be taken into account when interpreting learning curve studies.[8]

Although these are the only "learning curve" studies known to the authors that are specific to meningioma surgery, some studies of learning curves for other types of brain tumor surgery have also been reported. Several studies have shown better endocrine cure rates and decreased complications for experienced pituitary surgeons.[60,61] Both hearing[62] and facial nerve results[63–65] after acoustic neuroma surgery have been shown to reflect learning curves, with results improving after 20 to 100 cases.

When surgery is performed by a team rather than a single surgeon, it may be possible for an experienced team to train a novice surgeon without an increase in adverse events. For example, one acoustic neuroma team trained a new neurosurgeon who joined the group by starting with easier cases and always having a more experienced member available for intraoperative advice at the start of the series. No increase in adverse events was seen, which the authors attributed to the graded learning process.[66]

Other Measures of Surgical Experience

Other measures of surgical experience, or of concentration of a provider's effort on a given topic, include whether the surgeon has had special training or has gained certification in a given field (or belongs to specialized professional societies); the degree to which a surgeon concentrates his or her efforts on a certain type of surgery (specialization); and whether the surgeon practices as part of a specialized team (which may be multidisciplinary). To our knowledge, no studies have investigated meningioma surgery outcomes according to provider experience measured by these criteria. For completeness, we will briefly summarize related literature, where available.

In malignant brain tumor surgery, pediatric tumor specialists had better extent of resection in medulloblastoma and malignant glioma cases in a Children's Cancer Group study, with dedicated pediatric neurosurgeons and American Society for Pediatric Neurosurgery members being more likely to remove more than 90% of the tumor and to leave less than 1.5 cc of residual tumor than were general neurosurgeons ($p < 0.05$).[67] This study combined features of a specialization study ("dedicated pediatric neurosurgeons," defined as the designated pediatric specialist for their center) and a certification study (i.e., specialty society members). The differences seen were modest, and when the same data were analyzed by number of cases performed by individual surgeons, stronger correlations with extent of resection were seen. In contrast to this study, Latif et al reported that adult malignant glioma patients who received operations by a single specialist surgeon did not have better survival than similar patients operated at the same center by other nonspecialist neurosurgeons.[68] Other studies on general surgical topics have demonstrated that increased surgeon specialization correlates modestly with decreased mortality after a variety of surgical procedures.[69] Studies in breast cancer and other malignancies have shown higher patient satisfaction and better long-term survival after specialist, multidisciplinary team management of the initial tumor.[70–72] Although it has been suggested that this might be true for brain tumor treatment as well,[73] studies actually seeking to demonstrate such an effect are lacking in brain tumors to date.

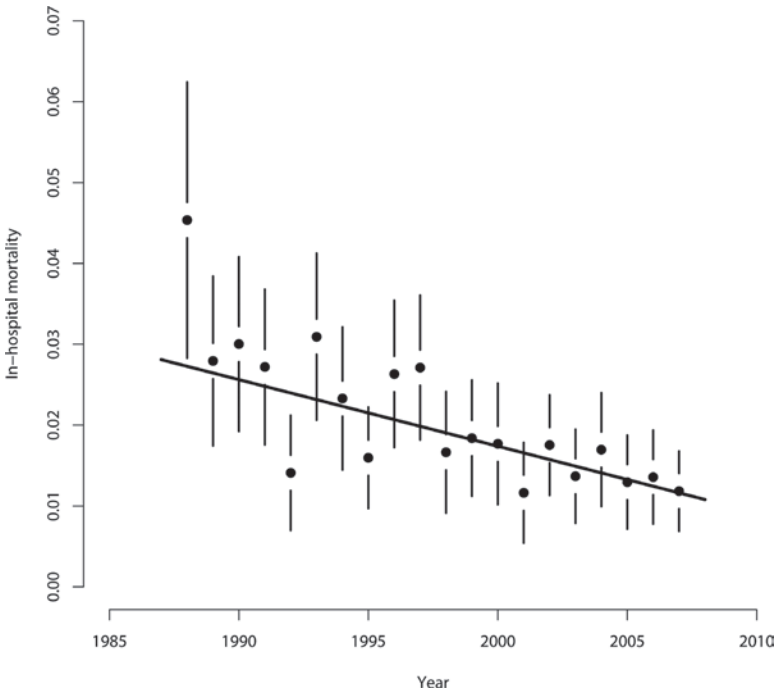


Fig. 43.2 Graph showing trend over time for in-hospital mortality in U.S. nonfederal hospitals, 1988 to 2007. Vertical bars show 95% confidence intervals around the point estimate for each year. Solid regression line shows the reduction in mortality over time, which was statistically significant ($p < 0.001$). Data from Nationwide Inpatient Sample (Healthcare Cost and Utilization Project, Agency for Healthcare Research and Quality, Rockville, MD), using methods from Curry et al.[8]

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Conclusion

Evidence suggests that patients have better outcomes after tumor treatment by high-volume, specialized providers for a broad range of tumor types. Studies available to date that specifically address meningioma care seem to follow the same pattern. The reasons for the observed advantage from specialized care have not yet been defined, and future research is likely to focus on the processes that characterize high-volume meningioma care. Better definition of these processes offers the potential to improve care for many meningioma patients in the future.

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Chapter 44

Outcomes and Quality of Life after Surgery for Meningiomas

Abel Po-Hao Huang, Khalid Medani, and Peter M. Black

Because surgical treatment is the mainstay for most intracranial meningiomas, it is important for neurosurgeons to appreciate the surgical outcome and quality of life after meningioma surgery. Advances in microsurgical technique, neuroimaging modalities, neuroanesthesia, and perioperative intensive care have improved surgical outcome substantially in recent decades. Therefore, most of the surgical outcomes in this chapter are cited from contemporary literature (2000 to present) to depict the outcome of contemporary microsurgery.

Outcomes are usually measured in surgical morbidity, mortality, time to recurrence, and quality of life. Extent of resection, tumor grade, proliferative markers, and tumor location are significant factors in predicting the surgical outcome; we therefore address each of these in detail. It is also important to acknowledge the increasing use of multimodality treatment, including radiation therapy, in the management of certain meningiomas, such as cavernous sinus and petroclival meningiomas. Finally, the importance of quality of life is discussed, with emphasis on the fact that comprehensive evaluation of outcome after meningioma surgery is crucial.

Because surgical resection has long been the preferred definite treatment for most intracranial meningiomas, it is important for neurosurgeons to appreciate the outcome and quality of life after surgery for these patients. In contemporary practice, the goal of surgery for intracranial meningiomas is to achieve as extensive a resection as possible while minimizing neurological morbidity.

Figure 44.1 shows the parameters and interrelationship for surgical, tumor, and patient outcome. In general, the most common factors that influence *surgical outcome* include the comorbidity and age of the patient, the size and location of the tumor, and the presence and severity of neurological deficit. The measurable parameters for *patient outcome* include the Karnofsky performance scale (KPS), quality of life (QOL) assessments, biopsychosocial status, complication, and disease status. It is known that a patient may have poor functional outcome or QOL after apparently successful surgery. This fact stresses the importance of addressing biopsychosocial factors to achieve best outcome. *Complications* include both surgical and medical complications. Surgical complications include infection, hemorrhage, cerebrospinal fluid (CSF) leakage, and wound problems. Medical complications include deep vein thrombosis, seizure, infection, and pulmonary embolism. We expand more on this topic at the end of the chapter, especially with regard to QOL. *Surgical outcome* is reported in the form of extent of resection, complications, morbidity, and mortality in most series. Factors that influence surgical outcome include tumor location, consistency, and vascularity; and the surgeon's experience, philosophy, and technique. It is apparent that surgical outcome and other treatment modalities influence *disease outcome*. This includes recurrence rate, progression-free survival, and overall survival. This is related to the extent of resection, World Health Organization (WHO) grade of the tumor, and proliferative markers. Disease outcome is perhaps the greatest determinant of patient outcome; it is a compilation of all other factors.

It is also necessary to acknowledge that the recent advances in radiosurgery and radiation therapy have created a paradigm shift in the management of intracranial meningiomas. This is especially true with the contemporary management of optic nerve sheath meningiomas, cavernous sinus meningiomas, and petroclival meningiomas. Multimodality treatment is often applied to achieve satisfactory functional outcome and tumor control.

Given that extent of resection, tumor grade, proliferative markers, and tumor location are significant factors in predicting outcome and recurrence; we begin by addressing each of these in detail.[1,2]

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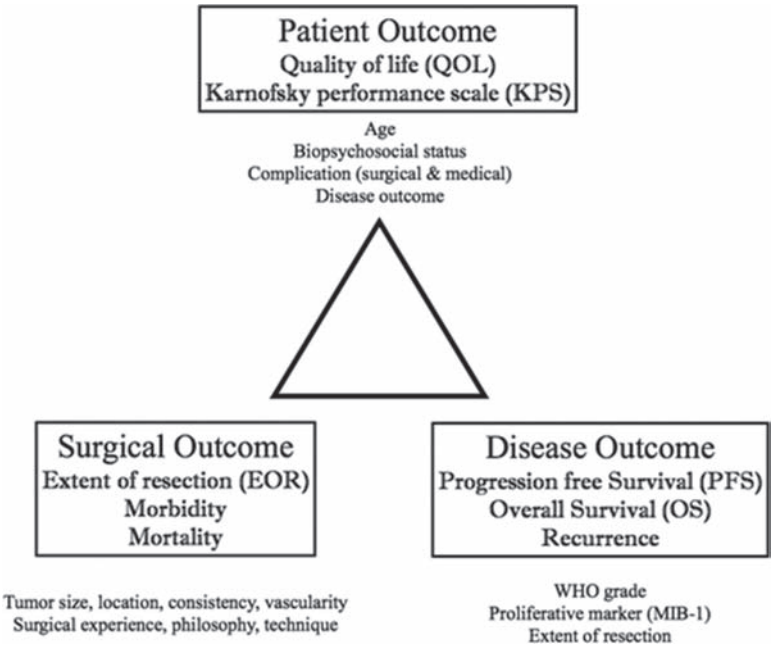


Fig. 44.1 Parameters used for patient, surgical, and disease outcome and the relationship between outcomes.

Extent of Resection as Prediction of Outcome

Simpson demonstrated in 1957 that the extent of surgical resection correlated well with late tumor recurrence.[3] However, his data were gathered long before microsurgery and present techniques and are based on the surgeon's assessment of resection. He reported a 9% recurrence rate after total resection along with the dural base (grade I resection), a 19% recurrence rate after total resection with the dural base cauterized (grade II), a 29% recurrence rate when the tumor was removed but the dura could not be resected (grade III), and an ~40% recurrence rate when only subtotal resection was performed (grade IV). Subsequent studies in the magnetic resonance imaging (MRI) era with a follow-up period longer than 5 years have found higher recurrence rates; a 70% recurrence rate after subtotal resection, and 16% and 20% recurrence rates after Simpson grade I and II resections, respectively.[4,5] Given this high recurrence rate even after Simpson grade I resections, some authors have recommended "grade 0" resection of a normal dural margin 2 cm wide.[6,7] Subsequent retrospective reviews using this surgical method for convexity meningiomas have found no recurrences and no increase in morbidity with this aggressive approach at 5 years.[8] However, this approach is impossible in meningiomas other than convexity tumors.

Tumor Grade and Proliferative Markers as Predictors of Outcome

The WHO classification of brain tumors distinguishes three grades of meningiomas: the common type (WHO grade I), the atypical type (WHO grade II), and the anaplastic type (WHO grade III).[9] Using WHO criteria, 81% of intracranial meningiomas were benign, with a 12% 5-year recurrence rate; 15% were atypical, with a 41% 5-year recurrence rate; and 4% were anaplastic brain-invasive with a 56% 5-year recurrence rate after gross total resection.[10] In one study of the brain-invasive anaplastic type meningiomas, the 5-year mortality rate was 83% and median survival was only 1.4 years.[11] Benign and atypical brain-invasive lesions showed similar 5-year mortality rates of ~25% and between 10 and 14 years median survival. Similar to benign grade I meningiomas, gross total resection of atypical or anaplastic meningioma is associated with lower recurrence rates than with subtotal resection.[11] Once recurrence develops, prognosis is poor because of the high likelihood of treatment failure.[12]

The histopathology is not solely decisive for the outcome with respect to tumor control.[13] Molecular markers of prognostic value have been reported, and most studies have shown that an elevated MIB-1 index is useful for assessing growth rate and may be predictive of recurrence and poor outcome.[1,12] It has also been demonstrated that the CDKN2A deletion, along with a 9p21 deletion, is a predictor of malignant progression, increased recurrence, and poor survival.[14]

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Surgical Outcome of Meningiomas in Different Locations

Comparing surgical outcome for meningiomas at different sites is difficult because the nomenclature, surgical indication, approach, philosophy, policy for adjuvant radiation, definition of the extent of resection, recurrence, management of recurrence, and follow-up period all differ significantly. The results provided in this chapter are outcomes from centers of excellence in managing intracranial meningiomas and are intended to provide general information for clinicians and patients in terms of surgical outcome.

Surgical morbidity and mortality have declined steadily as a result of the refinements of microsurgical technique, advancement in neuroanesthesia, and improved perioperative intensive care. Therefore, most of the surgical outcomes in this chapter are cited from contemporary references (2000 to present). **Table 44.1** provides contemporary surgical outcome according to location of intracranial meningioma. Outcomes with multimodality treatment for petroclival and cavernous sinus meningiomas are provided, given that multimodality treatment is the current trend in management of these difficult lesions.

Joung and Lee formulated a "CLASS" algorithm based on *c*omorbidity, tumor *l*ocation, patient *a*ge, tumor *s*ize, and neurological *s*igns and *s*ymptoms to assess the risk of meningioma surgery.[15] Tumor location was classified into low-, moderate-, and high-risk groups. The low-risk group included convexity, lateral and middle sphenoid wing, and posterior petrous meningiomas. The moderate-risk group included olfactory groove, planum sphenoidale, tentorial (lateral/paramedian), parasagittal, falcine, intraventricular, cerebellopontine angle, posterior/lateral foramen magnum, and parasigmoid/paratransverse sinus meningiomas. The high-risk group included clinoidal, medial sphenoid ridge, cavernous sinus, tuberculum sellae, tentorial (medial/incisural), ventral petrous, anterior/lateral foramen magnum, and petroclival meningiomas. Professor Kawase and his group (Adachi et al) at Keio University have created a simple algorithm, the "ABC" system, for assessing risk in skull base meningioma surgery based on preoperative image findings.[16] This system takes *a*ttachment/size, *a*rterial involvement, relation to *b*rain stem, and *c*ranial nerve involvement into account and was found to predict the probability of radical surgical removal.

Olfactory Groove and Planum Sphenoidale Meningiomas

Hentschel and DeMonte reported 13 patients with olfactory groove meningiomas surgically treated at the M.D. Anderson Cancer Center in 2003.[17] Complete resection, including hyperostotic bone and dura of the cribriform plate and any extension into the ethmoid sinuses, was achieved in 11 patients. There was no surgically related complication and there was no recurrence in a mean follow-up period of 2 years. However, this is too short an interval to be definitive. Nakamura et al reported 82 cases of olfactory groove meningiomas surgically treated by the senior author Samii.[18] Gross total tumor resection was accomplished in 92% of the cases. The overall recurrence rate was 4.9% in a median follow-up period of 5.3 years. It has been reported that the cranial base and paranasal sinuses are sites of predilection for recurrence of olfactory groove meningiomas.[19] Complications included subdural hygroma (17.6%), seizure (11.8%), hydrocephalus (5.8%), hemorrhage (2.9%), and brain edema (2.9%). The perioperative mortality was 4.9%.

Surgical outcome of patients with preoperative visual impairment or mental status changes related to these lesions is frequently satisfying[20]; Nakamura et al reported preoperative visual disturbance improved in 55% of patients, dementia improved in 62.9% of patients, and concentration difficulties improved in 68.3% of patients.[18] Hentschel and DeMonte reported 100% improvement in mental dysfunction and 83% improvement in visual disturbance.[17] Preservation of olfactory function was more likely in cases with tumor size less than 3 cm and normal preoperative function.[21]

Tuberculum Sellae Meningiomas

Tuberculum sellae meningiomas present a special challenge because of their proximity to arteries of the anterior circulation, optic apparatus, and hypothalamus. The overall morbidity and mortality rate associated with resection of these tumors has decreased substantially in modern series. Total resection can be achieved in 76.4—93% of the cases, with a 15 to 20% incidence of nonvisual morbidity and 0 to 8.7% mortality.[22–29] Preservation of vision is the most important goal of surgery, and improvement in vision has been reported in 40 to 80% of the patients.[24,29–31] Nakamura et al reported 72 cases of tuberculum sellae meningiomas surgically treated by the senior author Samii.[32] Total resection was achieved in 91.7% of the patients. The perioperative mortality rate was 2.8%. Postoperative visual improvement was seen in 65% of patients. Visual improvement was dependent on the duration of preoperative visual symptoms but not on preoperative visual acuity or tumor size. Patients with a visual symptom duration of less than 6 months tend to recover more than those with a duration longer than 1 year. The overall recurrence rate was 2.8% during a mean follow-up time of 3.8 years. Although most patients will have favorable visual outcome, it is noteworthy that visual deterioration has been reported in 17 to 20% of patients receiving surgery for these lesions.

The long-term visual outcome for these patients has improved substantially compared with early series. However, tumor recurrence is still common, even when gross total resection was achieved. One study demonstrated a 39% recurrence rate with a mean follow-up time of 10.7 years.[33] Patients with tumor recurrence are likely to lose vision in at least one eye and are unlikely to have visual

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Table 44.1 Contemporary Surgical Outcome According to Meningioma Location*

Location	Rate of Total Excision	Recurrence	Morbidity	Mortality	Functional Improvement	Surgically Related Complications	Comments
Olfactory groove/ planum sphenoidale	85–100% (54, 141)	0–4.9% with mean follow-up period of 2–5.28 years (54, 87)	0–31.3% (54, 87, 127)	0–4.9% (54, 87, 127)	Mental status improvement 100%, visual improvement 83% (54)	CSF leakage, seizure, infection, decreased visual acuity, anosmia, infarction, hemorrhage	All hyperostotic bone should be removed with the dura of the anterior skull base to minimize the risk of recurrence (54) Preservation of olfactory function was likely if preoperative function was normal and if the tumor was smaller than 3 cm in diameter (135)
Tuberculum sellae	76.4–93% (10, 35, 43, 48, 86, 98)	1.4–4.2% with mean follow-up period of 2.5–4.3 years (43, 48, 96)	25–45% (5, 43, 48, 57, 96, 121)	0–8.7% (10, 35, 48, 57, 86)	Visual function improvement 37.8–80% (10, 43, 86, 121)	CSF leakage, seizure, anosmia, hypopituitarism, decreased visual acuity, infarction	Visual deterioration in 10–30% of patients (10, 43, 48, 98, 121) Tumor extension into the optic canal is common and required optic canal deroofing to achieve maximal visual outcome (121)
Convexity	95% (81)	4.3% with mean follow-up period of 2.3 years (81)	9.4% (81)	0 (81)	—	Motor deficit, infarction, infection, hematoma	5-year recurrence rate for benign meningiomas was 1.8%, atypical meningiomas 27.2%, and anaplastic meningiomas 50% (81)
Parasagittal and falcine	63.2–93% (18, 39, 126)	4–24% with mean follow-up period of 5.0–8.0 years (18, 39, 126)	8–10% (18, 39)	0–3% (18, 39, 126)	55.6% had improved neurologically (18)	Motor deficit, brain swelling, infarction, infection, hematoma	5.5% had a new deficit (18)
Medial sphenoid ridge	58–87% (1, 71, 115)	0–9% with mean follow-up period of 3.1–12.8 years (1, 71, 115)	5.7–13% (1, 71, 115)	0 (1, 71, 115)	63–75% had improvement of visual function (71, 115)	Cranial nerve palsy (CN 3, 4, 6) visual impairment, vascular injury	Cavernous sinus involvement is associated with less favorable visual outcome, less total resection, and higher recurrence rate after surgery (84)
Clinoidal	54.5–86.7% (71, 107, 130)	3.8–15% with mean follow-up period of 3.1–4.5 years (71, 107, 130)	4–29% (71, 107, 130)	0 (71, 130)	Visual function improvement 71–75% (71, 130)	Oculomotor palsy, trochlear palsy, infection, hydrocephalus, cerebral edema, seizure	Al-Mefty identified three distinct groups on the basis of the site of tumor origin and the presence/absence of the arachnoidal plane between the tumor and the ICA. These factors are significantly related to surgical difficulty, resectability, and outcome (4)
Cavernous sinus	0 (75, 133)	5–5.7% with mean follow-up period of 2.3–3.8 years (75, 133)	7.5–15% (75, 133)	0 (75, 133)	Recover rate of preoperative cranial nerve dysfunction 20% (75)	Cranial nerve dysfunction, CSF leakage, stroke, hematoma, infection, and pituitary dysfunction	Recurrence, morbidity and mortality of multimodality treatment (excision of extracavernous component, radiation for intracavernous component) are shown here. Worsening of preoperative neurological status in 31.7% of the patients, but improvement was evident in 23% (104)

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Petroclival	0–48% (9, 60, 72, 89, 122)	5–29% with mean follow-up period of 3.9–8.5 years (60, 89, 122)	20.3–47% (9, 60, 72, 89, 122)	0–0.7% (9, 60, 72, 89, 122)	Most patients had improvement of KPS at 1 year after surgery, except for those with preop KPS less than 70 (89). 14.8–78.6% of the preop cranial nerve deficits improve (89, 97)	Cranial nerve palsy (esp. CN IV, CN VII), motor deficit, hydrocephalus, CSF leakage, infection, infarction, hematoma	Recurrence, morbidity and mortality of multimodality treatment are shown here. Risk of cranial nerve deficits increased with prior resection, preoperative cranial nerve deficit, tumor adherence to neurovascular structures, and fibrous tumor consistency. The risk of paresis or ataxia increased with prior resection and tumor adherence (72)
Cerebellopontine angle	82–86.1% (11, 83, 113, 132, 138)	3.9–7.5% with mean follow-up period of 3.0–6.0 years (11, 83, 113, 132, 138)	10.4–35.7% (11, 83, 113, 132, 138)	0–5% (11, 83, 113, 132, 138)	5.5–34.8% had hearing improvement (11, 85, 113, 138)	Cranial nerve palsy (esp. CN VII, VIII), hydrocephalus, CSF leakage, infection, hematoma	Result depends on tumor location with regard to IAC (83, 113, 138) Functional preservation of facial nerve was 60–86%. Hearing preservation was achieved in 67–90.8% (11, 83, 113, 132, 138)
Foramen magnum	67–96% (8, 13, 20, 23, 25, 47, 62)	0–5.5% with follow-up period of 3.6–6.1 years (8, 13, 47, 62)	5.9–27% (8, 13, 20, 23, 25, 47, 62)	0–4% (8, 13, 20, 23, 25, 47, 62)	Improvement of preop deficit 75–100% (8, 13, 88). Low chance of lower cranial nerve functional recovery, however (13)	Lower cranial nerve palsy, vascular injury, CSF leakage, hydrocephalus	Factors associated with morbidity: anterior located tumor, smaller tumor size, vertebral artery encasement, extradural extension, adhesion in recurrent cases, absence of arachnoid plane (25)
Jugular foramen	50–100% (7, 45, 109, 120)	0–16.6% with mean follow-up period of 2.5–6.5 years (7, 45, 109, 120)	30–61.5% (7, 45, 109, 120)	0–20% (7, 45, 109, 120)	Recovery of preoperative lower cranial deficit usually does not occur (7, 45, 109, 120)	Cranial nerve palsy (esp. lower cranial nerve), CSF leakage, hydrocephalus	New lower cranial nerve dysfunction occurs in 27–61.5% (95, 120)
Tentorial	77–91.3% (12, 24, 30)	0–8.6% with mean follow-up period of 4.5–5.9 years (12, 24, 30)	9.7–55% (12, 24, 30)	0–3.7% (12, 24, 30)	All pre-operative complaints and neurological deficit resolved postoperatively (108)	Cranial nerve palsy, hydrocephalus, CSF leakage, infarction, brain edema, infection, hematoma	Most of the falcotentorial or peritorcular meningiomas could not be removed completely due to frequent sinus infiltration. Tumors of the inner dural ring might be difficult to resect completely due to tight adherence to brain stem, cranial nerves, or venous system (12)
Intra-ventricular	87.5–93.7% (15, 16, 73, 82)	0–8.3% with mean follow-up period of 3.0–5.8 years (16, 73, 82)	6.2–33.3% (15, 16, 73, 82)	0–8.3% (15, 16, 73, 82)	Headache, vertigo, and dysphasia resolve in all patients. 75% had motor recovery, 75% had improvement in ataxia, 71.4% had improvement in hemianopia, 25% had improvement in cognitive dysfunction (73)	Seizure, visual field defect, motor weakness, speech dysfunction, memory impairment, disconnection syndrome, hydrocephalus	—

Abbreviations: CSF, cerebrospinal fluid; IAC, internal auditory canal; ICA, internal cerebral artery; KPS, Karnofsky performance scale.
*Operative morbidity is reported according to authors.

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improvement with subsequent surgery or radiation therapy. Therefore, postoperative patients should undergo long-term, serial clinical and radiological examination to allow early detection and management of recurrences. Some have reported the extended transsphenoidal approach for resection of these tumors.[34] This procedure is suitable for small midline lesions, without major vessel encasement or parasellar extension. Improvement of vision was seen in 75% of the cases, and 93.1% had gross total tumor removal. However, the difficulty in reconstructing the cranial base dural and bone defects has to be overcome because the postoperative cerebrospinal fluid leakage rate was as high as 28.6%.[34]

Optic Nerve Sheath Meningiomas

It has been shown that fractionated stereotactic radiotherapy should be considered the treatment of choice for the majority of patients with these tumors because of its capability of achieving excellent local control and improved/stable visual function in more than 80% of patients. This is especially true for patients with progressive functional loss and most patients with some degree of functional loss at presentation. The proper timing for patients with no or slight vision loss at presentation should be further investigated, however.[35]

Surgical management includes biopsy, optic nerve sheath fenestration, and tumor excision. Because surgical management is rarely applied for these tumors, we will only review the indication and result in brief. Biopsy, which was used in the past, frequently leads to functional impairment. Due to the characteristic clinical presentation, funduscopic and radiological findings of these tumors, biopsy is currently regarded as unnecessary for the clinical diagnosis. Optic nerve sheath fenestration has been shown effective in arresting progressive visual loss by some, whereas some others found it unsuccessful.[35,36] Some even report this procedure might result in orbital invasion of the tumor, which necessitates orbital exenteration. This approach has therefore largely been abandoned. Tumor excision was a frequently practiced treatment approach in the last decade. Orbital approaches provide adequate resection for selected localized cases, whereas most cases require a transcranial approach to provide complete tumor resection up to the optic chiasm. Dutton reviewed 148 surgically treated cases in 1992 and reported that the transcranial approach is associated with zero mortality, 30% morbidity, and a recurrence rate of 25%.[37] Importantly, improvement of vision occurred in only 5% of patients, and 94% of patients suffered from visual deterioration. In most cases, blindness occurred due to the interruption of pial vessels and ischemic injury to the optic nerve, given that these tumors and the optic nerve share the same blood supply. Currently, transcranial surgery might be indicated for tumors with intracranial spread or continuously progressing large tumors in nonfunctioning eyes to prevent major intracranial spread.

Convexity Meningiomas

The overall prognosis for these tumors is excellent, with minimal morbidity and mortality. In our series of 163 surgically treated patients reported in 2008, the operative mortality rate was 0%. The incidence of new neurological deficit was 1.7%, and the overall complication rate precluding medical complications was 9.4%.[38] It has been stressed that preservation of the cerebral venous system is a key to successful outcome in the surgical management of convexity meningioma. The 5-year recurrence rate for benign meningiomas was 1.8%, atypical meningiomas 27.2%, and anaplastic meningiomas 50%.[38] Because total excision could be achieved in all cases, the recurrence rate was largely dependent on the grade of the tumor.

Parasagittal and Falcine Meningiomas

Overall, the surgical morbidity for these tumors is around 8 to 10% and the mortality is less than 3%.[39–41] Surgical complications include hematoma, brain swelling, infarction, and infection.

Management of sinus involvement is an important issue. In 2006, Sindou and Alvernia reported 92 patients who underwent aggressive resection of the invaded superior sagittal sinus with venous reconstruction and postoperative anticoagulation.[41] They reported a 4% recurrence rate with a mean follow-up period of 8 years, which is low compared with the rates from other series (4% vs 11 to 24%). In their study, there was 3% mortality, and 8% of the patients had permanent neurological deficit, likely due to venous infarction. DiMeco et al reported 108 patients harboring parasagittal meningiomas that invaded the superior sagittal sinus who were surgically treated at their institute in 2004.[40] They recommend that if the sinus is invaded, it can be opened for resection of the tumor to attempt to preserve the patency of the sinus. Complete resection of the tumor and sinus can be performed in patients with sinus obstruction. With this approach it is not necessary to reconstruct the sinus, and tumor recurrence rate was 13.9% with a median follow-up period of 6.6 years. The mortality rate was 1.8%, and the complication rate was 10.1%. In our series of 46 consecutives cases reported in 2008, we proposed a less aggressive surgical approach; tumor was resected up to the sinus wall, and the sinus was left intact.[39] Residual tumor was followed up and treated with radiosurgery at recurrence. Although this approach led to a substantial number of patients with postoperative residual tumors (36.8%), only three WHO grade I meningiomas (7.7%) progressed. The recurrence-free survival was 94.7% at 5 years. In our series, 55.6% of patients with a preoperative neurological deficit had recovery, and 5.5% had a new deficit. One elderly patient died of a pulmonary embolus within 1 month of surgery.

The recent advance in radiosurgical technique provides an alternative treatment paradigm to radical surgery; subtotal resection with adjuvant radiosurgery or

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with radiosurgery alone. Few studies have focused on the parasagittal meningioma in this regard. However, there is accumulating evidence showing that midline meningiomas have a higher incidence of symptomatic brain edema after radiosurgery, which might be related to venous thrombosis.[42] Recently, it has been shown that this effect is seen less in patients with parasagittal meningioma treated with fractionated radiotherapy.[43]

Lateral and Middle Sphenoid Ridge Meningiomas

The complication rates and mortality for these lesions are lower than those for medial sphenoid ridge and clinoidal meningiomas. Basso et al reported surgical outcome for 23 middle and 59 lateral sphenoid wing meningiomas.[44] For lateral sphenoid wing meningiomas, 89.8% of the patients had good outcome with no sequelae, 8.5% had minor sequelae, 1.7% had major sequelae, and 1.7% mortality. For middle sphenoid wing meningiomas, 68.6% of the patients had good outcome with no sequelae, 26% had minor sequelae, 13% had major sequelae, and 8.7% mortality. Of note, mortality is usually related to vascular injury with cerebral infarction or edema. They reported a recurrence rate of 18.18% at 10 years. Recurrence may arise from hyperostosis and osseous involvement of these tumors.[45] Because the chance of total resection during second surgery is low, every attempt should be aimed at gross total resection, including the hyperostotic bone during the first surgery.[46]

Medial Sphenoid Ridge Meningiomas

Medial sphenoid ridge meningiomas are surgically challenging lesions due to the frequent involvement of the optic apparatus, cranial nerves, blood vessels, and cavernous sinus. However, substantial improvement in surgical outcome has been achieved in the last decade. Approximately 66 to 87% of these tumors can be totally resected with 4 to 18% morbidity and zero mortality. Improvement of vision is seen in 63 to 75% of the patients. This is in contrast to early series, where mortality was as high as 15 to 43% and the rate of total resection ranged from 23 to 50%. Russell and Benjamin reported 35 surgically treated patients in 2008; gross total resection was achieved in only 69% of cases due to frequent cavernous sinus involvement. The overall morbidity rate was 18% and there was no mortality. Improvement of vision was achieved in 63% of cases. A 9% recurrence rate was observed during a median follow-up of 12.8 years.[47] The authors also stress that frequent tumor involvement of the arteries of anterior circulation mandates meticulous management to avoid major morbidity. Nakamura et al reported 108 surgical cases in 2006.[48] They divided patients into two groups; those with cavernous sinus involvement and those without. Those with cavernous sinus involvement had less favorable visual outcome, less total resection, and higher recurrence rate after surgery. In their study, recurrence with tumor progression after surgery was observed in 7.7% of tumors without cavernous sinus involvement and 27.5% of tumors with cavernous sinus involvement during a mean follow-up period of 5.8 years.

Recurrence is a major concern in the management of these patients, and several studies have shown medial sphenoid ridge/clinoidal meningiomas have one of the highest recurrence rates among all intracranial meningiomas. Recent series (2000 to present) have demonstrated a significant decrease in recurrence rate compared with older series (1980 to 1999) (0 to 11% vs 12 to 36%). The substantial improvement in surgical outcome is likely related to use of the operating microscope, skull base techniques, appreciation of the meningioma–arachnoid plane, preservation of perforators around the optic apparatus, and dedicated perioperative intensive care.

Clinoidal Meningiomas

Clinoidal meningiomas and medial sphenoid meningiomas are clinically and anatomically similar, with surgical difficulties aforementioned. Thanks to the advance of skull base surgery and postoperative care, surgical mortality and morbidity for clinoidal meningioma have also been greatly diminished. Tobias et al reported 26 surgical cases in 2003.[49] Total resection was achieved in 77% of the patients, and 76.9% of the patients had improvement of preoperative visual impairment. None of the benign meningiomas recurred during a mean follow-up period of 3.6 years. In most series, patients also experience postoperative visual improvement (32 to 76.9%). The rate of total resection is 59 to 86.7%, and the recurrence rate is in the range of 3.8 to 15%.[49–51] The morbidity ranges from 4 to 29%, and mortality is zero.

Al-Mefty identified three distinct groups of clinoidal meningiomas on the basis of the site of tumor origin and the presence of the arachnoid plane between the tumor and the internal carotid artery. These factors are significantly related to surgical difficulty, resectability, and outcome.[52] Group 1 tumors were those encasing and directly attaching to the adventitia of the internal carotid artery, without an arachnoidal plane between the tumor and the vessel. In this group, total resection was not possible in any patient. Group 2 consisted of tumors with a separate arachnoidal plane between the tumor and the internal carotid artery, which facilitated total removal. Group 3 tumors were those originating at the optic foramen, for which total resection could be achieved in all patients.

Cavernous Sinus Meningiomas

Most cavernous sinus meningiomas have both an intracavernous and an extracavernous component. The extracavernous component can be surgically resected with good outcome. Surgery for the intracavernous portion of the tumor, despite its feasibility, is associated with high morbidity, low rate of complete resection, and substantial recurrence rate (5 to 13%).[53–57] Total resection was only achieved in 20

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to 76% of the patients who underwent aggressive surgery with a cavernous sinus opening technique.[53–55] Aggressive surgical removal of these tumors is associated with an increased incidence of morbidity and mortality and does not provide a better rate of tumor control. This is presumably due to the frequent tumor infiltration of cranial nerves and vessels within the cavernous sinus.[58] The potential complications of cavernous sinus surgery for meningiomas include cranial nerve dysfunction, CSF leaks, stroke, hematoma, infection, and pituitary dysfunction. As a general rule, preoperative cranial nerve dysfunction has a low rate of recovery (10 to 14%), and most patients with new postoperative deficits recover. New cranial nerve dysfunction occurs in 12 to 41% of the patients.[53–57]

Recently, more patients have been treated with radiosurgery, either alone or as adjuvant therapy, with a cranial nerve complication of 0 to 22.5% and a progression rate of 5 to 17.7%. Many surgeons adopt a multimodality approach that consists of removal of the extracavernous portion and application of radiotherapy/radiosurgery to the intracavernous component. Maruyama et al reported the multimodality management outcome of 40 cases of cavernous sinus meningiomas in 2004.[59] Radiosurgery alone was given to tumors confined to the cavernous sinus and distant from the optic apparatus and the brain stem. Tumors attached to or compressing the optic apparatus and brain stem that were larger than 3 cm were treated with combined subtotal resection and radiosurgery. The tumor control rate was 94.1% at 5 years. Improvement of preoperative cranial nerve dysfunction was observed in 20% of the patients. Other studies using similar combined therapy also yield low morbidity (13 to 15%) and high tumor control rate (92 to 95%).[60]

In summary, growth control and preservation of neurological functions are the primary goals in the management of cavernous sinus meningiomas. Multimodality treatment is important to achieve such goals. Treatment algorithms and strategies are being formulated, and further results on long-term outcome will provide us more evidence for management.[59,61]

Petroclival Meningioma

Petroclival meningiomas are among the most difficult skull base lesions, and the history of surgical treatment for these tumors reflects the evolution of modern skull base surgery. A systematic approach to these tumors based on their size, location, brain stem involvement, preoperative neurological deficit (especially hearing function), venous anatomy, and radiographic appearance optimizes the extent of resection and minimizes perioperative morbidity and mortality. One study focused on the preoperative radiological findings and their relationship to surgical outcome. In 70 patients, tumor size, brain stem compression, and tumor extension laterally to the internal auditory canal did not influence either the extent of resection or the long-term surgical outcome.[62] In contrast, supratentorial extension to the middle fossa and downward involving the caudal cranial nerves was significantly related to surgical radicality and outcome. Radiological evidence of infiltration and peritumoral edema at the brain stem surface were important factors that influenced extent of resection.

In recent years, several reports have demonstrated marked improvement of the surgical outcome of petroclival meningiomas.[63–68] Regardless of the surgical approach used, the major determinants of the ability to achieve an excellent resection with a low rate of morbidity are the presence of an intact arachnoid plane, the consistency of the tumor, and involvement of critical neurovascular structures.[63,65] The goal of surgery is complete tumor removal, if possible, without causing functional decline of the patient. This has substantial morbidity of 40 to 63% and a recurrence rate of 5 to 29%.[63–68] Complete resection is usually not possible because of the severe adherence to or invasion of the brain stem, or encasement of the adjacent vasculature or cranial nerves. Contemporary series show that the morbidity ranges from 22 to 47%, and mortality is in the range of 0 to 17%.[63–68] The most common morbidity is cranial nerve deficit, especially the facial nerve or trochlear nerve, which occurs in 28.6 to 30% of patients.[63,66,67] With regard to functional improvement, 14.8 to 78.6% of the preoperative cranial nerve deficits improve to some degree, with patients receiving subtotal resection having the highest probability of recovery.[67] Most patients had improvement of KPS at 1 year after surgery, except for those with a preoperative KPS less than 70.[66]

Although Simpson grade I resection is possible in a substantial number of patients, subtotal resection with or without radiosurgery has become the preferred approach to reduce the morbidity and improve the quality of life.[63–66] The policy of management of cavernous sinus involvement by these tumors has changed considerably over the years because of the good results of radiation, as discussed previously, which preclude aggressive resection of the prepontine component and its associated morbidities. Knowing when to resect the tumor completely and when and where to leave some behind is based on the experience and philosophy of the surgeon and the expectation of the patient.

Stereotactic radiosurgery continues to play an emerging role in the treatment of petroclival meningiomas. Long-term control has been reported to be as high as 97%.[69] Tumor volumes were stable or decreased in 92 to 97% of patients. Neurological improvement occurred in 14 to 48% of the patients, and cranial nerve deficits occurred in 8 to 12% of patients.[69–72] Therefore, stereotactic radiosurgery provides effective management of small or medium-sized petroclival meningiomas and is an alternative to microsurgery. For tumors with larger volume, fractionated stereotactic radiotherapy or multisession stereotactic radiosurgery might be an alternative treatment option.[73,74]

Cerebellopontine Angle Meningiomas

Wu et al presented the result of 82 surgically treated cases in 2005, which demonstrated that the functional outcome after surgery is significantly related to the pre-

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cise location of the cerebellopontine angle meningioma.[75] According to the anatomical relationship with the posterior petrous bone and the internal auditory canal (IAC), posterior petrous meningiomas were classified into three types in their study: type I, located laterally to the IAC; type II, located medially to the IAC, which might extend to the cavernous sinus and clivus; and type III, extensively attached to the posterior surface of the petrous bone, which might envelop the seventh and eighth cranial nerves. Overall, total resection was achieved in 83% of cases. Anatomical preservation of the facial nerve was possible in 97.5%, whereas functional preservation was achieved in 81% of the patients. The rate of hearing preservation was 67%. All type I tumors were completely resected, and the rate of anatomical preservation of the facial nerve was 100% and functional preservation was 93%. In terms of type II lesions, 75% of patients received total resection; the rate of anatomical preservation of facial nerve was 97% and functional preservation was 75%. For type III lesions, 73% were totally resected. The rate of anatomical preservation of the facial nerve in patients with this tumor type was 95%, whereas functional preservation was 73%. Other studies have demonstrated similar results; good results can be achieved with tumors located posterior or superior to the internal acoustic canal compared with tumors with a premeatal location or intrameatal involvement.[76–78]

Functional facial nerve preservation can be achieved in 66 to 86% of patients and hearing preservation in 67 to 90.8% of the patients.[75–77,79,80] It is noteworthy that recovery of hearing is also possible, even in patients with profound hearing impairment.[76] Meningioma arising from the internal acoustic canal is rare and presents a distinct category. Surgical outcome is favorable for these lesions, with 71.4% of hearing preservation and 95.2% functional facial nerve preservation.[81]

In terms of radiosurgery, serviceable hearing was preserved in 73% of patients who had serviceable hearing before treatment.[82] However, 54% of patients with tinnitus at presentation continued to experience it in long-term follow-up.[81]

Foramen Magnum Meningiomas

These tumors are surgically challenging due to the proximity of the brain stem, lower cranial nerves, and vertebral artery. Factors associated with morbidity include an anteriorly located tumor, smaller tumor size (more difficult to access), vertebral artery encasement, extradural extension, adhesion in recurrent cases, and absence of arachnoid plane.[83] Bassiouni et al reported their surgical result for 25 patients with foramen magnum meningiomas.[84] Simpson grade II resection was achieved in 96% of patients. Permanent morbidity and mortality rates were 8% and 4%, respectively. No tumor recurrence was observed after a mean follow-up period of 6.1 years.

In general, total resection can be achieved in 67 to 100% of cases in the modern neurosurgical era, with relatively low morbidity (0 to 22%) and mortality (0 to 6%).[83–90] Operative morbidity is related to the precise location and extension of the tumor. Although vertebral artery encasement occurs in 38 to 59% of patients, meticulous microsurgical technique usually avoids the complication of vascular injury. Most preoperative deficits, such as ataxia and sensory and motor deficit, recover. However, lower cranial nerve palsy rarely recovers. Lower cranial nerve palsy is the most common morbidity and should be treated aggressively to avoid serious pulmonary infection and mortality.[85] The presence of preoperative lower cranial nerve dysfunction is associated with better prognosis, presumably due to previous adaptation. In contrast, new-onset postoperative lower cranial palsy has worse recovery.

Minimally invasive approaches (transoral transclival approach and endoscopic approach) have been reported with a high rate of total excision, but the difficulty of dural repair and CSF leakage are the main drawbacks of these approaches.[88,91]

Jugular Foramen Meningiomas

Primary jugular foramen meningiomas are extremely rare tumors, with less than 100 cases reported in the English literature. Gross total resection can be achieved in only ~36 to 84.6% of patients due to the frequent infiltration of the lower cranial nerves, cavernous sinus involvement, posterior inferior cerebellar artery (PICA) encasement, the need to preserve dominant jugular bulb, and adherence or invasion of the brain stem.[92–96] Therefore, the recurrence rate is high, in the range of 12.5 to 25% according to some series.[92,97,98] Surgery for these tumors is associated with 20 to 50% morbidity and 0 to 20% mortality.[92–96] Hearing function can be preserved, and facial nerve outcomes are favorable in many patients. The most common postoperative complication is the lower cranial nerve deficit occurring in ~57 to 61.5% of patients.[92–96] In cases with preoperative dysfunction, compensation is usually achieved by contralateral innervation. Most of these patients will require assistance and rehabilitation with regard to swallowing, airway protection, and phonation, and many might benefit from vocal cord medialization.[93]

Cerebellar Convexity Meningiomas

Pure cerebellar convexity meningiomas arising from the dura over the posterior convexity of the cerebellum are rare, and there is no large series reported. Most cerebellar convexity meningiomas are inferior peritorcular meningiomas (arising from the inferior wall of the torcular herophili or the medial portion of the transverse sinus) or parasinual meningiomas arising in the angle between the petrous and convexity dura (these tumors may involve the wall of the sigmoid sinus or lateral transverse sinus). Roberti et al reported 161 cases of surgically treated posterior fossa meningiomas, among which 14 cases were cerebellar convexity meningiomas.[98,99] Gross total resection was achieved in 77% of patients with 8.3% morbidity

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and no mortality. The overall recurrence rate was 14.2% during a 19-month mean follow-up period. Sinus involvement may account for the low rate of complete excision and high recurrence rate. Mismanagement of the involved sinus may lead to a catastrophic outcome, however.

Tentorial Meningiomas

Tentorial meningiomas are a heterogeneous group consisting of medial incisural meningioma, falcotentorial meningioma, paramedian meningioma, peritorcular meningioma, and lateral meningioma. Depending on the extension of the tumor and the major sinus involvement, various supra-, infratentorial, and combined approaches are applied for excision of these tumors. Overall, surgery for these tumors is associated with 9.7 to 55% morbidity and 0 to 3.7% mortality.[100–105] Total resection could be achieved in 77 to 91.3% of cases. Bassiouni et al reported 81 surgically treated tentorial meningiomas.[100] Simpson grade I and II resection was achieved in 91% of cases, with a recurrence rate of 8.6% during a 3.5-year follow-up period. Overall, permanent surgical morbidity was 19.8% and surgical mortality was 2.5%. The authors reported that most of the falcotentorial or peritorcular meningiomas could not be removed completely due to sinus infiltration. Tumors of the inner dural ring might also be difficult to resect completely due to tight adherence to the brain stem, cranial nerves, or venous system.[100]

A major issue in the surgical treatment of these tumors is resection of the venous sinuses to achieve complete tumor resection. There is general agreement that a completely occluded and thus nonpatent sinus can be resected safely. The proper management of the tumor invading a patent sinus is a controversial issue. Some recommend excision of the involved venous sinus when preoperative imaging reveals a dominant contralateral transverse sinus or evidence of venous collateral formation.[106–108] In some cases, reconstruction of the venous sinus from dural leaves or with a saphenous venous graft might be necessary.[107] With these aggressive venous sinus resection approaches, the recurrence rate is still as high as 16 to 21%.[107,109,110] Therefore, some suggest preservation of the infiltrated sinus. This approach is associated with fewer complications and a recurrence rate of 8.6 to 25.9%.[100,101] It has also been demonstrated that subtotal removal of these tumors can be associated with a long progression-free period and high quality of life.[111]

Intraventricular Meningiomas

Intraventricular meningiomas are relatively rare tumors representing 1 to 5% of all intracranial meningiomas.[112] Depending on the precise location and associated hydrocephalus, different surgical approaches are used to excise these tumors. Complications may include seizure, visual field defect, motor weakness, speech dysfunction, memory impairment, disconnection syndrome, and hydrocephalus. In recent series, gross total tumor excision was achieved in 87.5 to 93.7% of cases, with 6.2 to 25.0% morbidity and 0 to 8.3% mortality.[112–115] Liu et al reported 25 surgically treated cases.[114] Compared with preoperative deficit, 75% had motor recovery, 75% had improvement in ataxia, 71.4% had improvement in hemianopia, and 25% had improvement in cognitive dysfunction. In terms of functional recovery, headache, vertigo, and dysphasia resolved in all patients. Gross total resection was achieved in 87.5% of patients, and the recurrence rate was 8.3% in follow-up from 6 months to 15 years.[114] Others have reported no recurrence in cases with complete resection.[113]

Meningioma Surgery in the Elderly

Meningioma surgery in the elderly is an important issue to address with regard to surgical outcome, given that most intracranial meningiomas occur in the elderly, with a peak incidence between 60 to 70 years.[116] Many have reported the increased morbidity and mortality associated with meningioma surgery in elderly patients; morbidity ranges from 11.3 to 52% and mortality 1.8 to 45%.[117,118]

Recent studies have shown the importance of patient selection, which greatly reduces the mortality and morbidity in these patients.[119] Using the Karnofsky performance scale, American Society of Anesthesiology Class, location of tumor, and the peritumoral edema (SKALE) grading system, surgery for elderly patients with a SKALE grade above 8 can be performed with a morbidity of 9.4% and a 3-month mortality of 1.4%. Data from a case-control prospective study of 114 patients undergoing meningioma resection by a single surgeon have shown that medical and surgical complication rates were 7% and 5.2% in elderly patients, compared with 8.8% and 3.5% in younger patients.[120] Mortality within the first 30 postoperative days was zero for the young patients, and 1.7% in the elderly group. This study provided level 3 evidence that meningioma surgery in the elderly can be performed safely in experienced hands.

Radiosurgery/Radiotherapy versus Surgery

Stereotactic radiosurgery has been considered an alternative treatment option for small to moderate-sized benign meningiomas.[6,54,87,100,112] Tumor control rates of intracranial meningiomas with various locations were reported to be as high as 93%.[121,122] One retrospective study comparing the efficacy of radiosurgery to that of surgical resection compared outcomes between 528 patients who underwent surgical resection versus 170 patients who underwent radiosurgery at the Mayo Clinic.[123] Recurrence or progression occurred in 12% of patients in the surgical resection group, which is more frequent than the 2% rate in the radiosurgery group. The 3- and 7-year progression-free survival rates for patients having Simpson grade I surgical resections were 100% and 96%, respectively, comparable to the 100% and 95% obtained with radiosurgery. On the other hand, Simpson grade II resections produced 91% and 82% 3- and 7-year

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progression-free survivals, less than obtained with radiosurgery. This study provides level 3 evidence supporting radiosurgery in the management of patients with small to moderate-sized meningiomas without symptomatic mass effect, especially those for which an incomplete resection is anticipated.

Because resection of skull base meningiomas is often limited owing to involvement of critical neurovascular structures, radiosurgery is an appealing treatment option. Within the last decade, radiosurgery has gained increasing importance as an adjunct treatment after incomplete resection and as an alternative treatment to open surgery.[69–72] Recent studies on the long-term result of radiosurgery for skull base meningiomas have confirmed that it is safe and effective, with a 92 to 100% tumor control rate and 13 to 45% clinical improvement.[69,124] Therefore, radiosurgery should be considered a treatment option for patients with small to medium-sized skull base meningiomas. For tumors of a larger size, fractionated stereotactic radiotherapy or multisession stereotactic radiosurgery may also provide safe and effective treatment.[74,125]

Quality of Life after Intracranial Meningioma Surgery

Traditional outcome studies regarding intracranial meningioma surgery usually use recurrence, progression, morbidity, and mortality as outcome parameters. Comprehensive outcome assessment should also consider neuropsychological status and QOL. QOL is a multidimensional concept comprising physical, psychological, and social phenomena; this is probably the reason that different measures of QOL often lead to different results.[73,78,126] Therefore, direct comparison between studies of the complex measure QOL is difficult, and the most appropriate method for interpreting QOL data remains an area of intense debate.[78,127] In the study by Krupp et al, the combination of neuropsychological tests and a structured interview was used in an effort to increase information about QOL and to enable a comparison of both objective and subjective indices of QOL.[128]

Although surgery for meningioma leads to long-term recovery, which is different from other primary brain tumor, it raises concerns about postoperative neurological and neuropsychological deficits.[129] While most literature supports that most patients have good QOL after surgery for meningioma, some studies report a significant portion of patients with poor QOL after surgery.[66,128,130] This is especially true for skull base meningiomas.[131–133] Lower cranial nerve dysfunction seems to contribute significantly to the poor QOL in these cases.[133]

Studies focusing on QOL after meningioma surgery are still scarce. Review of the literature shows that 14 to 35% of patients do not regain premorbid working ability after meningioma surgery.[128,130,134,135] In most series, age of the patient significantly influenced cognitive performance, and one study also demonstrated rapid decline after 55 years of age.[128] Most studies have also found that young patients tend to have a long period of recovery despite successful surgery. This phenomenon also seems to prohibit their rehabilitation with regard to work and social life. Most studies also show that younger patients were less confident about their cognitive performance status than older patients, with a significant discrepancy between subjective complaints and test results.[128,130,136,137] Some believe that younger patients held unrealistic expectations about their vulnerability and long-term recovery.

Patients with good cognitive performance may be affected by a subjective impression of cognitive impairment or fear of failing. This phenomenon should be considered and taken into account as a part of appropriate and comprehensive rehabilitation programs. Rehabilitation with an assertiveness training program may help to overcome depression and the possibility of social isolation. It has been shown that depressive coping is generally more common among females and persons living as singles.[128,138]

In summary, comprehensive evaluation of outcome after meningioma surgery is crucial to prevent poor long-term outcome after an apparently successful surgery. Assessments should include standardized neuropsychological tests and a structured interview.[128] The use of more homogeneous study end points, such as disease-, age-, and health-related QOL may be necessary to enable valid comparison of results from different studies in the future.

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