Medial Sphenoid Wing Meningioma

Approximately ~15-20% of all meningiomas arise from the sphenoid wing, with about half of these arising from the medial portion of the wing.

Medial sphenoid wing meningiomas are a heterogeneous group of tumors originating from the anterior clinoid and the medial third of the lesser sphenoid wing. This group includes both globular and hyperostotic en plaque tumors (also called “spheno-orbital” meningiomas). Spheno-orbital meningiomas will be discussed in the Lateral Sphenoid Wing Meningioma chapter. There are no specific pathologic or genetic features for medial sphenoid wing meningiomas. Some of these tumors are caused by ionizing radiation.

Surgical management of medial sphenoid wing meningiomas is challenging because of the closely associated critical neurovascular structures along the parasellar region. Meningiomas can originate from any part of the meninges along the clinoid process or lesser sphenoid wing and grow medially, so clinical presentation and technical details of surgical treatment vary accordingly.

Sphenoid wing meningiomas can be divided into three main groups based on the site of their origin: those arising from the anterior clinoid and medial third of the sphenoid wing; those arising from the middle and lateral sphenoid wing; and en plaque meningiomas of the sphenoid wing. In this chapter, I will discuss techniques for resection of globular meningiomas of the anterior clinoid and medial portions of the sphenoid wing.
The Simpson scale remains the most practical method to predict the risk of meningioma recurrence following resection.

### Table 1: Simpson Scale for Prediction of Meningioma Recurrence after Surgery

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<th>Simpson Grade</th>
<th>Completeness of Resection</th>
<th>10 yr Recurrence</th>
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<tr>
<td>I</td>
<td>Complete with associated dura and bone removal</td>
<td>9%</td>
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<tr>
<td>II</td>
<td>Complete with coagulation of dural attachment</td>
<td>19%</td>
</tr>
<tr>
<td>III</td>
<td>Complete without dural coagulation</td>
<td>29%</td>
</tr>
<tr>
<td>IV</td>
<td>Subtotal resection</td>
<td>40%</td>
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### Classification

Anterior clinoid meningiomas are further classified into three following subgroups based on their site of origin along the anterior clinoid. Each group offers a unique set of technical difficulty for microsurgery, but all three typically involve both the internal carotid artery (ICA) and the optic apparatus and potentially the oculomotor nerve.

As the ICA emerges from the cavernous sinus inferior and medial to the anterior clinoid process, it passes through the subdural space between the inner and outer (or upper and lower) dural rings where 1-2mm of its segment lacks arachnoidal covering. Meningiomas arising around this short segment are classified as Group 1 clinoidal meningiomas.
Figure 1: A lateral view of the cavernous sinus and clinoidal segments of the right ICA. Note the short ICA segment between the upper and lower dural rings where group 1 clinoidal meningiomas arise from (image courtesy of AL Rhoton, Jr).

As Group 1 tumors grow, they typically engulf the ICA, grow distally toward the ICA bifurcation and encase the proximal middle cerebral artery. Because they lack an intervening arachnoidal plane, they are densely adherent to the adventitia of the ICA, rendering dissection difficult and resulting in lower rates of surgical cure. Group 1 tumors also typically involve the optic nerve and chiasm, but an arachnoid plane invests the tumor in this region, facilitating dissection. Group 1 tumors frequently invade the cavernous sinus.

Group 2 clinoidal meningiomas arise from the superior and lateral aspects of the anterior clinoid dura. These tumors often engulf the ICA as they grow, but are invested by the arachnoidal layers of the carotid cistern, creating accessible dissection planes. Additionally, these tumors own arachnoidal dissection planes within the region of the optic nerve and chiasm. Cavernous sinus invasion is common.
These tumors are therefore more amenable to aggressive safe resection than group 1 tumors.

Group 3 clinoidal meningiomas arise from the optic foramen and extend into the optic canal. Because of their site of origin and growth pattern, group 3 tumors become symptomatic earlier than Group 1 and 2 tumors and are substantially smaller at the time of their diagnosis. These tumors are invested by arachnoid membranes in the area of the ICA, but because they originate away from the chiasmatic cistern, there is typically no obvious arachnoid plane between the tumor and the optic apparatus. As a result, surgical cure is less common and the risk of postoperative visual decline is more real.

The tumors arising from the middle portion of the sphenoid wing grow very large before their clinical presentation. They cause significant mass effect on the temporal lobe, and if they have enough medial extension, they cause visual disturbance. Smaller lesions without medial extension can be treated like convexity meningiomas after resection of the sphenoid wing.

**Diagnosis**

The most common clinical presentation of clinoidal and medial sphenoid wing meningiomas are headaches and visual disturbance such as blurred vision, visual field deficit, or optic atrophy (resulting from optic apparatus compression) or diplopia (resulting from oculomotor nerve distortion).

Tumors that invade the cavernous sinus or superior orbital fissure may cause additional cranial neuropathies. Large tumors with middle cranial fossa extension compressing the temporal lobe or brainstem result in seizures or hemiparesis, respectively. Such tumors may also cause cognitive and memory deficits, personality changes, and
dysphasia.

Tumor-induced hyperostosis of the sphenoid wing and lateral orbit may present with proptosis, diplopia, and orbital pain. *En plaque* meningiomas of the sphenoid wing, also called spheno-orbital meningiomas, present with such ocular manifestations. These tumors can invade the lateral wall of the cavernous sinus, superior orbital fissure, floor of the middle cranial fossa, and the extracranial infratemporal fossa.

**Evaluation**

A thorough history and physical exam with particular attention to the symptoms and signs mentioned above are required. Thin-cut or high-resolution magnetic resonance (MR) imaging, while including fat suppression sequences through the orbits, can assess orbital involvement.

Angiographic evaluation with MR angiography or computed tomography (CT) angiography determines the meningioma’s relationship to the surrounding vasculature and their degree of encasement. However, these studies are rarely necessary as the T2-weighted MR images are adequate for identification of relevant vasculature. The bone windows on CT angiography also determine the extent of tumor-infiltrated hyperostosis.

Catheter angiography can demonstrate the utility of preoperative embolization and estimates the robustness of collateral blood supply via a temporary balloon occlusion test if the ICA is encased and at a high risk of operative injury. However, I advocate subtotal removal of this benign tumor in an attempt to preserve the ICA. With the availability of radiosurgery, the associated ischemic risks of a more aggressive resection are not warranted.
I do not believe endovascular embolization is necessary for most meningiomas as they can be devascularized early during exposure by aggressive resection of the sphenoid wing and anterior clinoid as well as cauterization of the involved dura.

A thorough neuro-opthalmologic and endocrinologic assessment should be performed as part of evaluation for all symptomatic parasellar tumors, including meningiomas.

Figure 2: Medial sphenoid wing meningiomas can present
different set of technical challenges based on their involvement of the medial neurovascular structures and the encasement of the carotid artery’s perforating vessels. A medial sphenoid wing meningioma with minimal medial extension is shown (upper images). The Sylvian middle cerebral artery branches drape over the superior pole of the tumor. A more true medial sphenoid wing/clinoidal meningioma with significant medial extension and encasement of the ICA is also included (lower images).

Figure 3: A group 3 or right-sided optic foramen meningioma is demonstrated. The strategic location of this mass leads to its early discovery due to the associated relatively rapid course of visual deterioration.

**Indications for Procedure**

Surgical resection is the mainstay of treatment for medial sphenoid wing meningiomas. Stereotactic radiosurgery is an option for asymptomatic small tumors without mass effect, but the proximity of
highly radiosensitive optic chiasm and nerves often precludes its use. Observation is also a reasonable treatment plan for small incidental tumors.

Figure 4: Coronal and axial views of a middle/medial sphenoid wing meningioma with its typical relationship to the surrounding vascular structures is demonstrated. More prominent evidence of optic apparatus compression is usually present.

**Preoperative Considerations**

Computed tomography (CT) measures the extent of bony invasion or hyperostosis. This information is important for intraoperative navigation to guide gross total resection of the involved bone and achieving Simpson scale 1 outcome. This CT data also determines the potential need to prepare a custom implant preoperatively to reconstruct the area of resected bone.

Preoperative understanding of how the tumor has distorted the normal vasculature is beneficial to avoid catastrophic vascular injury. Furthermore, significant vascular encasement at the skull base
highlights the need for planned subtotal resection as small caliber ICA perforating arteries are highly vulnerable to arterial injury and dissection during tumor excision. Magnetic resonance (MR) images provide the necessary information.

A lumbar drain can decompress the brain early and allow for an obstructed extradural clinoidectomy to release the affected optic nerve before the tumor is manipulated.

**Operative Anatomy**

Familiarity with the paraclinoid vascular and optic apparatus anatomy in addition to bony morphology is important.

![Osteology of the anterior and middle cranial base](image)

**Figure 5**: Osteology of the anterior and middle cranial base is shown. Note the lesser sphenoid wing, anterior clinoid process and surrounding bony structures (image courtesy of AL Rhoton, Jr). **Extradural clinoidectomy can expose the base of the tumor early and facilitate its devascularization.** Furthermore, extradural
optic nerve decompression protects the nerve early before any intradural tumor manipulation places the nerve at risk of traction injury.

Figure 6: Different anatomical views of the anterior clinoid processes, cavernous sinus, and their associated neurovascular structures. The dura is removed over the right anterior clinoid process (images courtesy of AL Rhoton, Jr). Most meningiomas enter the optic canal medial to the nerve
because of the availability of a potential space there. The oculomotor nerve is at risk of injury during clinoidectomy and tumor resection. Medial sphenoid wing meningiomas may infiltrate the cavernous sinus; however, this portion of the tumor should be left behind because of the risk of operating within the cavernous sinus.

**RESECTION OF MEDIAL SPHENOID WING MENINGIOMA**

Most medial sphenoid wing meningiomas can be resected through the extended pterional craniotomy. If the lesion harbors a significant suprasellar component, the orbitozygomatic craniotomy affords an excellent exposure of the suprasellar extent of the tumor with minimal frontal lobe retraction. Tumors with intraorbital extension also require an orbitozygomatic/orbital osteotomy to expose the orbit, remove the tumor and correct the proptosis. I use the extended pterional craniotomy with extradural clinoidectomy for >90% of medial sphenoid wing meningiomas.

The use of prophylactic perioperative antiepileptic medications is controversial. I prefer to administer a loading dose of this medication at surgery and continue the medication for 7 days postoperatively. In the absence of any seizure within the perioperative period, this medication is tapered off around 1 week after surgery. If the patient suffers from any seizure activity during the perioperative period, the dose may be increased and continued for 6 months to 1 year.

Since larger tumors fill the opticocarotid cisterns and often prevent early cerebrospinal fluid drainage for brain relaxation, I implant a lumbar drain after induction of the anesthesia to promote brain relaxation. This relaxation is important for 1) making extradural clinoidectomy possible despite the tumor overlying the medial sphenoid wing, 2) early extra- and intradural aggressive tumor devascularization and disconnection through mobilization of the
tumor base away from the skull base before its debulking.

For giant tumors with significant edema and mass effect, CSF drainage should be conducted judiciously and gradually, preferably after dural opening to avoid transtentorial herniation. Overdrainage of cerebrospinal fluid at the outset of surgery can also potentially make dissection of the Sylvian fissure more difficult.

Please refer to the Extrudal Clinoidectomy chapter for further details regarding the initial steps of the operation after craniotomy. Hyperostotic clinoid process can be challenging to safely remove, as the bone is very resistant to drilling. The optic nerve should be skeletonized and carefully protected during heavy drilling using ample amount of irrigation fluid.

Hypertrophied clinoid processes can distort the normal anatomy of the optic foramen/canal. I use the assistance of intraoperative CT navigation to localize the foramen/canal. Once the clinoidectomy is complete, the tumor’s base along the dura over the sphenoid wing and clinoid process is thoroughly devascularized extradurally.

Once the above steps are complete, I open the dura in a crescent shape and expose the meningioma following an anterior sylvian fissure split.

**INTRADURAL PROCEDURE**

Slow egress of CSF via the lumbar drain achieves desirable brain relaxation.
Figure 7: Exposure of the tumor through a left-sided extended pterional craniotomy after extradural clinoidectomy is shown. In this case, the large tumor extended laterally through the Sylvian fissure. Following ~40cc of gradual CSF drainage through the lumbar drain, in 10cc aliquots, the tumor is mobilized away from the lateral sphenoid wing dura and its more medial dural attachments coagulated. This important maneuver completes a critical step in the operation that leads to thorough devascularization of the tumor and significantly expedites the later steps of dissection by minimizing the need to frequently interrupt tumor dissection/removal to obtain hemostasis.
Figure 8: I continue tumor devascularization along the anterior cranial fossa while keeping the approximate location of the optic nerve in mind to avoid its heat injury. CSF drainage, Sylvian fissure split and strategic use of the hand held suction device obviate the need for fixed retractors.
Figure 9: Enucleation and debulking of firm tumors is conducted using an ultrasonic aspirator (left image) while softer tumors are debulked using bipolar electrocautery, suction apparatus and pituitary rongeurs. Next, I gently draw upon the tumor capsule to cause its collapse into the debulked core of the tumor (right image). It is critical to stay inside the tumor capsule. Violation of the capsule places the vulnerable adherent medial cerebrovascular structures at risk. Vicinity of the ultrasonic aspirator to the vessels, even without an immediate contact, can lead to irreparable vascular injury. This device should be used away from the critical vascular structures.

Figure 10: At this juncture, after some tumor debulking to create more working space, I further split the distal aspect of Sylvian fissure and identify the M2 branches draped over the superior
and posterior poles of the tumor capsule. I also gently mobilize the tumor capsule posteriorly along the sphenoid wing in an attempt to find or estimate the location of the ICA at the skull base. These latter two maneuvers help me approximate the route of the MCA branches, including the M1, along the medial tumor capsule-my blind spot.
Figure 11: All MCA vessels are sharply dissected off of the tumor capsule and protected with the use of cottonoids once mobilized (upper image). Blunt dissection should be avoided.
when possible. **Most importantly,** the feeding arteries of the tumor and the vital *en passage* vessels are clearly identified before their fate is decided. Pieces of papaverine-soaked Gelfoam are used to periodically bathe the small *en passage* vessels for relief of their vasospasm. Higher magnification intraoperative view demonstrates dissection of the M2 branches away from the tumor (T)(lower image).

Although vascular encasement is common on imaging in these tumors, most often, the arachnoidal plane between the tumor and the MCA branches remains intact enough to dissect the vessel free from the tumor. If the tumor is too adherent for this maneuver, a small sheet of tumor must be left on the vessels for their protection and prevention of vasospasm.
Figure 12: It is important to carefully mobilize the anterior frontal pole of the tumor in order to identify the optic nerve and ICA at the level of the skull base (upper image). Following the contour
of sphenoid wing medially, one can localize the approximate location of the optic canal and the ICA. In the lower intraoperative photo, the frontal portion of the tumors is removed and the location of the optic nerve and carotid artery is appreciated at the tip of the suction device. Residual coagulated tumor is present along the tentorium.

Figure 13: Gentle mobilization of the medial capsule and sharp dissection will uncover the optic nerve and proximal ICA. The falciform ligament is incised to untether the optic nerve. The posterior communicating artery can be seen originating from the posterior wall of ICA. This artery is an indicator for the general location of the oculomotor nerve. If the tumor is very adherent to the nerves or vessels, aggressive manipulation and blunt dissection must be avoided and a sheet of tumor left behind. Despite gentle handling of the tumor around the oculomotor
nerve and tentorium, most patients will suffer from transient third and fourth nerve palsy after surgery. Coagulation of the tentorium around these nerves should be minimized as much as feasible.

Figure 14: Next, I mobilize the posterior tumor capsule away from the temporal lobe. The base of the tumor along the anterior middle fossa is disconnected. I prefer to say “there it is” and be wrong 100 times, rather than say “there it was” and be right once. Neurovascular structures (more specifically, the posterior communicating artery, anterior choroidal arteries and the oculomotor nerve) are displaced and can be found in very unexpected locations. They are in harm’s way during aggressive coagulation in face of bleeding. The medial
arachnoid membranes over the basal cisterns and brainstem are left untouched.

Figure 15: It is essential to maintain the arachnoid planes along the entire circumference of the tumor capsule. To prevent infarcts, I preserve every perforating artery and minimize its manipulation. After gross total tumor resection, the infiltrated dura along the medial sphenoid wing is cauterized. The neurovascular anatomy at the end of resection is demonstrated.

The optic canal is then explored with a fine ball-tip dissector. If tumor is identified in this location, the falciform ligament is divided further
and the optic nerve unroofed to allow intracanalicular tumor extraction. Aggressive removal of attached tumor from the optic nerve can disrupt the nerve’s blood supply and worsen visual deficits. If the tumor is not readily separable from the nerve, a thin sheet of tumor must be left on the nerve and the optic canal generously unroofed. Careful microsurgery around the sensitive oculomotor nerve is necessary to avoid permanent cranial nerve paresis. The cavernous sinus is not entered.

**In menigioma surgery, the first operation is the best opportunity for surgical cure.** Therefore, safe aggressive tumor removal is an appropriate operative philosophy. However, if the tumor is adherent to the proximal ICA and encases this portion of the artery, a thin sheet of tumor must be left behind. Dissection of adherent tumor in this region invariably leads to injury to the small perforators originating from the medial wall of the ICA, including the posterior communicating and anterior choroidal arteries.

Unfortunately, I have suffered from the agony of this complication. One of my patients suffered from an infarct in the posterior limb of the internal capsule, causing hemiplegia, after removal of a giant medial sphenoid wing meningioma. I therefore advise against aggressive manipulation of the attached encasing tumor along the skull base.
Figure 16: The optic nerve is decompressed, but the adherent firm/calcified tumor encasing the vasculature is left behind to avoid injury to the perforating arteries (upper image). The lower intraoperative photo demonstrates the anterior choroidal artery or one of the perforators (arrow) encased by the tumor. This
piece of the tumor was not manipulated.

**Additional Considerations**

Dissection of fibrous tumors can be challenging and alternative techniques are necessary to mobilize the tumor from the optic nerve and the carotid artery.

Figure 17: The fibrous capsule of this medial sphenoid wing meningioma that was resistant to mobilization was removed by
dividing the tumor into two fragments parallel to the long axis of the ICA. The proximal carotid artery and optic nerve were first identified at the skull base (upper photo). The tumor was subsequently divided along the axis of the ICA (lower photo). This division facilitated mobilization and removal of the anterior and posterior fragments of the tumor.

**Case Example**

This patient presented with right-sided visual decline and was diagnosed with a large medial sphenoid wing meningioma.
Figure 18: The MR images of the first row demonstrate the mass and associated orbital roof hyperostosis. Extradural clinoidectomy decompressed the optic nerve early. The distal MCA branches were dissected and protected (second row). As dissection continued toward the skull base, the tumor was divided along the ICA; this maneuver facilitated tumor mobilization (last row, left image). The optic nerve was found distal in its foramen and generously released via removal of the intracanalicular portion of the tumor (last row, right image).

**RESECTION OF OPTIC FORAMEN MENINGIOMA**

Removal of optic foramen meningiomas is more straightforward as these tumors are discovered when they are small. They do not encase the vasculature. However, they can adhere to the optic apparatus.
Figure 19: A right optic foramen, group 3 meningioma, is demonstrated (top image). Extradural clinoidectomy unroofs the optic nerve (middle photo) in preparation of intradural opening of the falciform ligament and dissection of the tumor within the optic canal. The extracanalicular extent of the tumor along the medial aspect of the nerve is shown upon dural opening and elevation of the frontal lobe (lower image).
Figure 20: A Karlin blade (Symmetric Surgical, Antioch, TN) is used to cut the falciform ligament on the side of the tumor toward the surgeon (top image). The extracanalicular component of the tumor is dissected away from the nerve using sharp techniques and delivered using pituitary rongeurs (bottom photos).
Figure 21: The small perforating vessels to the chiasm are protected (top image) while an angled dissector mobilizes the more intracanalicular portion of the tumor around the medial optic nerve within the operative blind spot (middle image). Angled straight dissector inspects the distal part of the canal to ensure complete decompression of the canal; this finding is also verified using a microsurgical mirror (lower images).

**Closure and Postoperative Care**

A small piece of temporalis muscle is used to plug the extradural space at the site of clinoidectomy to prevent a postoperative CSF leak. The lumbar drain is removed at the end of the operation. Postoperative care is similar to the one for patients with other skull base meningiomas.

Postoperative vasospasm of the MCA branches is a significant risk and should be timely considered in the differential diagnosis of delayed postoperative neurologic decline. Imaging using a CT angiogram is warranted.

**Pearls and Pitfalls**

- A thorough extradural sphenoid wing resection and
Clinoidectomy leads to an opportunity to devascularize the tumor and decompress the optic nerve early in the procedure.

- Early tumor devascularization minimizes bleeding during the demanding microsurgical steps of the operation and keeps the operative field pristine. Avoidance of bipolar coagulation around the medial neurovascular structures is lifesaving.

- The critical neurovascular structures are along the medial capsule and therefore within the blind spot of the surgeon. Central tumor debulking and careful mobilization of the tumor capsule are key maneuvers to avoiding complications.

- All vessels should be treated with utmost respect and a small sheet of adherent tumor must be left behind. The perforators along the ICA at the skull base are nonforgiving.

DOI: [https://doi.org/10.18791/nsatlas.v5.ch05.3](https://doi.org/10.18791/nsatlas.v5.ch05.3)

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**References**


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