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Vertical Diplopia and Ptosis from Removal of the Orbital Roof in Pterional Craniotomy

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Purpose: To describe a newly recognized clinical syndrome consisting of ptosis, diplopia, vertical gaze limitation, and abduction weakness that can occur after orbital roof removal during orbito-zygomatic-pterional craniotomy.

Design: Case series.

Participants: Eight study patients (7 women), 44 to 80 years of age, with neuro-ophthalmic symptoms after pterional craniotomy.

Methods: Case description of 8 study patients.

Main Outcome Measures: Presence of ptosis, diplopia, and gaze limitation.

Results: Eight patients had neuro-ophthalmic findings after pterional craniotomy for meningioma removal or aneurysm clipping. The cardinal features were ptosis, limited elevation, and hypotropia. Three patients also had limitation of downgaze and 2 patients had limitation of abduction. Imaging showed loss of the fat layers that normally envelop the superior rectus and levator palpebrae superioris. The muscles appeared attached to the defect in the orbital roof. Ptosis and diplopia developed in 2 patients despite Medpor titanium mesh implants. Deficits in all patients showed spontaneous improvement. In 2 patients, a levator advancement was required to repair ptosis. In 3 patients, an inferior rectus recession using an adjustable suture was performed to treat vertical diplopia. Follow-up a mean of 6.5 years later revealed that all patients had a slight residual upgaze deficit, but alignment was orthotropic in primary gaze.

Conclusions: After pterional craniotomy, ptosis, diplopia, and vertical gaze limitation can result from tethering of the superior rectus—levator palpebrae superioris complex to the surgical defect in the orbital roof. Lateral rectus function sometimes is compromised by muscle attachment to the lateral orbital osteotomy. This syndrome occurs in approximately 1% of patients after removal of the orbital roof and can be treated, if necessary, by prism glasses or surgery. Ophthalmology 2015;122:631-638 © 2015 by the American Academy of Ophthalmology.

The development of the fronto-temporal-sphenoidal approach has greatly facilitated neurosurgical access to lesions around the base of the skull.1–5 It is also known as the pterional approach because it involves the conjunction of the frontal, parietal, sphenoid, and temporal bones. In a refinement of the pterional approach, an orbitozygomatic craniotomy was added to allow a more tangential approach to lesions in the anterior cranial fossa, such as aneurysms and meningiomas.6–9 After surgery, a permanent defect often remains in the orbital roof, bringing orbital contents into direct contact with meningeal tissue at the base of the frontal lobe.

Over the past 15 years, we have encountered 8 patients with a constellation of neurovisual findings that constitute a new clinical syndrome caused by orbito-zygomatic-pterional craniotomy. The patients had various combinations of vertical diplopia, limited upgaze, restricted downgaze, and ptosis after neurosurgical exploration of the anterior cranial fossa. Some patients also had a horizontal component to their gaze misalignment with limited abduction. This article describes the neuro-ophthalmic complications of pterional craniotomy involving the orbit, with emphasis on the likely mechanism, prevention, and treatment.
temporalsis fascia to protect the facial nerve. After the zygoma and the superior and lateral orbital rims were exposed, the periorbita was dissected away from the inner surface of the orbital wall. The temporalis muscle was reflected inferiorly. A pterional craniotomy was performed with a Medtronic (Minnepolis, MN) equipped with a footplate attachment. After the bone flap was removed, the dura was opened and tacked up circumferentially to the bone edge.

At this point, a series of osteotomy cuts with a reciprocating saw were made to release the superior and lateral orbital wall as a single unit. These consisted of a linear, parasagittal cut in the medial orbit, a perpendicular cut in the posterior orbit extending medially, and a cut in the lateral orbit wall from the inferior orbital fissure to the pterion (Fig 1). Additional bone was removed from the orbital roof as necessary using a Lempert rongeur. The intraorbital contents were retracted from the orbital roof with a malleable retractor to guard them from injury during the saw cuts. Moist Telfa pads or cottonoid sponges were placed over the orbital tissues for protection. The dura was opened in a curvilinear fashion over the orbital roof to provide access to the tumor or aneurysm tissues for protection. The dura was opened in a curvilinear fashion to close the orbital roof defect. The temporalis muscle and its fascia were protected with cottonoid sponges, the levator palpebrae superioris complex was damaged (Fig 4B). In addition, the lateral rectus appeared displaced toward the temporalis muscle.

Prism glasses were prescribed but were unsatisfactory for restoration of fusion in primary gaze. An 8-mm recession of the inferior rectus muscle was performed 16 months after the pterional craniotomy using an adjustable suture technique. Forced ductions under anesthesia just before the muscle surgery showed restriction of elevation. After surgery, the patient was able to fuse in primary gaze.

**Results**

**Patient 1**

Patient 1 is a 55-year-old man who underwent clipping of a ruptured anterior communicating artery aneurysm. The patient reported vertical diplopia immediately after the operation. One year later, there was nearly complete absence of elevation (Fig 2). In primary position, there were 30 prism diopeters (PD) of right hypotropia and 12 PD of right esotropia. In upgaze, there were 45 PD of right hypotropia. In downgaze he could fuse. Horizontal gaze was full, except for slight limitation of abduction in the right eye. Surprisingly, there was no appreciable ptosis.

A 3-dimensional CT reconstruction showed a 2-cm defect in the roof of the orbit (Fig 3). An oblique CT image revealed that the superior rectus muscle followed an abnormal trajectory to the orbital apex. It was tented upward in mid course, where it appeared adherent to soft tissue filling the bony defect. A coronal T1-weighted MRI scan showed disruption of the superior rectus and levator palpebrae superioris muscles (Fig 4A). The normal fat plane between the 2 muscles could not be identified. In addition, the layer of fat that usually surrounds the 2 muscles was absent. A contrast-enhanced image confirmed that the superior rectus—levator palpebrae superioris complex was damaged (Fig 4B).

In addition, the lateral rectus appeared displaced toward the temporalis muscle.

Table 1. Summary of Patients with Neuro-ophthalmic Complications Resulting from Orbital Roof Removal During Pterional Craniotomy

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yrs)</th>
<th>Gender</th>
<th>Neurosurgical Lesion</th>
<th>Motility Deficit</th>
<th>Primary Gaze Deviation</th>
<th>Ptosis</th>
<th>Follow-up/Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55</td>
<td>M</td>
<td>Anterior communicating artery aneurysm</td>
<td>Right elevation &amp; abduction</td>
<td>30 PD right hypotropia &amp; 12 PD right esotropia</td>
<td>No</td>
<td>Left inferior rectus recession</td>
</tr>
<tr>
<td>2</td>
<td>69</td>
<td>F</td>
<td>Right sphenoid wing meningioma</td>
<td>Right elevation &amp; depression</td>
<td>Orthotropia</td>
<td>Yes</td>
<td>Levator repair</td>
</tr>
<tr>
<td>3</td>
<td>58</td>
<td>F</td>
<td>Right sphenoid wing meningioma</td>
<td>Right elevation</td>
<td>20 PD right hypotropia</td>
<td>Yes</td>
<td>Prism glasses</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>F</td>
<td>Right supraclinoid internal carotid artery aneurysm</td>
<td>Right elevation &amp; depression</td>
<td>Right hypotropia</td>
<td>Yes</td>
<td>No light perception right eye, no intervention</td>
</tr>
<tr>
<td>5</td>
<td>53</td>
<td>F</td>
<td>Anterior communicating artery aneurysm</td>
<td>Right elevation &amp; depression</td>
<td>10 PD right hypotropia</td>
<td>Yes</td>
<td>Right inferior rectus recession &amp; levator repair</td>
</tr>
<tr>
<td>6</td>
<td>55</td>
<td>F</td>
<td>Anterior communicating artery aneurysm</td>
<td>Left elevation</td>
<td>Orthotropia</td>
<td>Yes</td>
<td>No treatment needed</td>
</tr>
<tr>
<td>7</td>
<td>54</td>
<td>F</td>
<td>Left middle cranial fossa</td>
<td>Left elevation &amp; abduction</td>
<td>25 PD left hypotropia &amp; 25 PD left esotropia</td>
<td>Yes</td>
<td>Left inferior &amp; medial rectus recession</td>
</tr>
<tr>
<td>8</td>
<td>80</td>
<td>F</td>
<td>Left sphenoid wing meningioma</td>
<td>Left elevation &amp; depression</td>
<td>40 PD left hypertropia</td>
<td>Yes</td>
<td>Monitoring</td>
</tr>
</tbody>
</table>

F = female; M = male; PD = prism diopeters.

Table 1. Summary of Patients with Neuro-ophthalmic Complications Resulting from Orbital Roof Removal During Pterional Craniotomy

Immediately after surgery, the patient had complete right ptosis and little vertical eye movement. Ten weeks later, the ptosis was still complete. There was 80% elevation and 30% depression of the globe. Six months later, the right palpebral fissure measured 5 mm. The eyelid just cleared the pupil and the patient reported no diplopia in primary gaze. However, there was still limitation of
vertical ductions, with corresponding diplopia. A levator muscle advancement was performed to correct the ptosis.

The patient was examined again 8 years after meningioma resection. She reported diplopia only on upgaze. The ductions in the right eye were full, except for 90% elevation with 10 PD of hypotropia in upgaze. A coronal flair MRI scan showed direct contact between the levator palpebral superioris and the basal meninges of the frontal lobe, with peaking of the muscle (Fig 5A). Abnormal gadolinium enhancement of the basal meninges was seen, which merged with the muscle complex (Fig 5B). The dark cleft representing the bony orbital roof was absent on the right side. The layer of adipose tissue that normally insulates the muscle complex from the orbital roof also was absent.

**Patient 3**

Patient 3 is a 58-year-old woman who underwent removal of a right sphenoid wing meningioma via a right fronto-temporal-sphenoidal craniotomy. The orbital roof and lateral wall were removed to the orbital fissures using a rongeur. The optic canal then was exposed under the operating microscope using a drill. After tumor removal, FloSeal hemostatic matrix (Baxter, Deerfield, IL) was applied to the undersurface of the frontal lobe. The defect in the orbital roof was closed with a titanium mesh Medpor implant.

Examination 2 months after surgery showed 5 mm of ptosis in the right eye. There was only 50% elevation of the right eye, with 20 PD of hypotropia in primary gaze. An orbital MRI scan showed disruption of the superior rectus/levator muscles and loss of the fat layer separating them from the Medpor implant. Four months after surgery, the ptosis had resolved completely. There was a persistent elevation paresis, with 7 PD of right hypotropia in primary gaze. Prism glasses were prescribed. By 5 months after surgery, the patient had regained fusion in primary gaze without prisms.

The patient was evaluated 7.5 years after the meningioma resection. There was still slight limitation of elevation, manifested by 10 PD of right hypotropia on upgaze.

**Patient 4**

Patient 4 is a 44-year-old woman in whom vision loss developed in the right eye from a 2.5-cm right supraclinoid internal carotid artery aneurysm. The lesion was clipped via a pterional craniotomy. The optic strut was drilled out to expose the siphon of the internal carotid artery. In addition, a superior orbitotomy was performed. Four clips were required to obliterate the aneurysm.

Immediately after the surgery, the patient had no light perception in the right eye. The eyelids were closed because of periorbital edema. Despite treatment with high doses of glucocorticoids, there was no recovery of visual function. One month after surgery, there were 5 mm of ptosis. Ductions showed 50% elevation and 90% depression. In primary gaze, there was a right hypotropia. A CT scan showed extensive postoperative changes, clip artifacts, and a defect in the orbital roof.

Examination 9 years later showed slightly limited elevation of the right globe. There were 20 PD of sensory exotropia.

**Patient 5**

Patient 5 is a 53-year-old woman who experienced a subarachnoid hemorrhage from a 4-mm anterior communicating artery aneurysm. Four days later, she underwent a right orbital-pterional craniotomy. A portion of the orbital roof extending back to the anterior clinoid process was removed. In addition, a small part of the gyrus rectus was removed to improve exposure. After the lesion...
was clipped, the orbital bone was repositioned using titanium plates and screws. The defect in the orbital roof was left open.

Examination 8 months later showed 4 mm of ptosis of the right eyelid and only 50% elevation of the globe. Ocular alignment was achieved with a 12-PD base-up prism in the right eye. An MRI examination of the orbits showed loss of the fat signal in the superior orbit and alteration in the appearance of the superior rectus and levator muscles. The patient declined prism glasses. Ocular alignment was restored by performing a 5-mm recession of the inferior rectus muscle using an adjustable suture technique. The ptosis was repaired by advancement of the levator palpebrae superioris.

The patient was contacted by telephone 12 years later. She reported double vision only on extreme upgaze.

Patient 6

Patient 6 is a 55-year-old woman who experienced a subarachnoid hemorrhage from a 7-mm anterior communicating artery aneurysm. It was clipped by a left orbital–pterional approach, with removal of additional bone from the back of the orbit with a rongeur. A small portion of the left gyrus rectus was sacrificed to improve exposure of the base of the aneurysm. After the aneurysm was clipped, the bone flap and orbital osteotomy were replaced using cement plates and screws, without closure of the defect in the orbital roof.

Examination 2 months after surgery revealed 1 to 2 mm of ptosis of the left upper eyelid. Ductions were intact except elevation, which showed only 90% of the normal range. Alignment was orthotropic in primary gaze, but a small left hypotropia developed in upgaze. No treatment was necessary. The only neuroimaging available after surgery was an axial CT scan obtained on the first postoperative day. The orbital images were not of adequate quality to determine the cause of her elevation deficit.

A follow-up examination 12 years later showed persistence of a small elevation deficit in the left eye with vertical diplopia on extreme upgaze.

Patient 7

Patient 7 is a 54-year-old woman who experienced slowly progressive vision loss in the left eye. An MRI scan showed a large
sphenoid wing meningioma compressing the left optic nerve. She underwent a left pterional craniotomy with removal of the lateral and superior orbital walls. The orbital roof was left open after closure of the craniotomy.

Examination 6 weeks after surgery showed a left frontalis muscle palsy, 50% elevation deficit, and a nearly total abduction deficit. In primary gaze, there were 25 PD of left hypotropia and 25 PD of left esotropia. An MRI examination showed a large defect in the left orbital roof and postoperative changes in the appearance of the lateral rectus and the levator palpebrae superioris and superior rectus muscles.

One year after removal of the meningioma, the patient underwent an 8-mm recession of the inferior rectus muscle and an 8-mm recession of the medial rectus muscle to correct her diplopia. An adjustable suture technique was used for both muscles. She could not be reached to obtain follow-up information.

**Patient 8**

Patient 8 is an 80-year-old woman who reported gradual proptosis of the left eye. An MRI scan showed a left sphenoid wing meningioma. It was resected via a left pterional craniotomy with a frontozygomatic and supraorbital osteotomy. After tumor removal, the orbital roof defect was reconstructed with a titanium mesh Medpor implant.

The day after surgery, the patient reported vertical double vision. There was eyelid swelling with ptosis. Globe elevation was reduced slightly and depression was absent. Forced ductions showed restriction of depression. There were 40 PD of left hypertropia in primary gaze. A CT scan obtained 2 days after surgery showed air in the left orbit (Fig 6A). There was soft tissue incarcerated in the supraorbital bone incision, with the levator palpebrae superioris and superior rectus muscles pulled upward. A more posterior cut (Fig 6B) showed a vertically distorted superior rectus–levator complex. The roof defect has been closed with a Medpor implant (white arrows), but incompletely.

**Discussion**

The pterional approach has undergone numerous modifications to improve access to the sellar region and the anterior cranial fossa. The most critical advance has been the discovery that temporary removal of the superior and lateral walls of the orbit can reduce the brain retraction required for exposure of lesions, such as sphenoid wing meningiomas and circle of Willis aneurysms. Violation of the orbit, however, carries the risk of disturbing eyelid function, globe motility, and ocular alignment. We report 8 patients with neurovisual symptoms that occurred immediately after pterional craniotomy. All but 1 patient had ptosis,
presumably from compromise of the levator palpebrae superioris. Every patient had limitation of globe elevation, most with a corresponding hypotropia. The mechanism is uncertain, but imaging showed attachment of the superior rectus to the defect in the orbital roof (Figs 3–6). We propose that adhesion of the muscle to the orbital roof may reduce its ability to rotate the globe in a manner similar to the action of a posterior fixation suture.10 Physical injury during surgery also may cause paresis of the superior rectus muscle. Three patients also displayed restricted globe depression. In these individuals, scarring of the superior rectus may have restricted downward eye rotation. In addition, 2 patients had limited abduction with esotropia. In surgical decompression also may cause paresis of the superior rectus muscle. Three patients also displayed restricted globe depression. In these individuals, scarring of the superior rectus may have restricted downward eye rotation. In addition, 2 patients had limited abduction with esotropia. In these cases, removal of the lateral orbital wall seemed to cause adhesion of the lateral rectus muscle (Fig 3).

Kaeser and Klainguti11 were the first to recognize the problem of vertical diplopia resulting from pterional craniotomy. They described a single patient with upgaze limitation and corresponding hypotropia, with adhesion of the superior rectus and levator palpebrae to the orbital roof defect. In the neuorsurgery literature, a review of 75 patients who underwent pterional surgery identified 2 cases (2.6%) of extraocular muscle restriction and 3 cases (4.0%) of ptosis.12 The true rate of neuro-ophthalmic complications after pterional craniotomy is difficult to estimate. We encountered 8 patients with diplopia and ptosis over a 15-year period, during which 980 pterional craniotomies with orbital takedown were performed by 2 neurosurgeons. This corresponds to a 0.81% rate of neurovisual complications but does not include patients whose symptoms resolved before referral to our clinic or who were evaluated elsewhere.

Restriction or paresis of eye muscles are common sequelae after any event that results in fracture of an orbital wall. The most common cause is a blowout fracture of the orbital floor or medial wall resulting from blunt trauma.13–16 Diplopia may occur from muscle entrapment, neuromuscular trauma, or extraocular muscle scarring. Kushner17 emphasized that the vertically acting muscle can show either restriction or paresis. In surgical decompression for thyroid eye disease, bony apertures are left deliberately in the orbital walls. Diplopia and gaze limitation occur in 2% to 43% of cases.18–25 Vertical diplopia and ptosis have been reported in patients with fibrous dysplasia after removal of the orbital roof and lateral wall.26–27 Diplopia also has been described in patients undergoing medial and superior orbital peristeum removal for frontal sinus, anterior skull base, and craniofacial procedures.28–30 In such cases, diplopia is believed to result from tethering of the superior oblique tendon or malposition of the trochlea. Finally, postoperative diplopia has been reported after breach of the orbital wall during endoscopic paranasal sinus surgery.30–32

Although Yasargil8 was among the first to remove the orbital roof to improve exposure, he later discarded this strategy after he recognized the risk of postoperative diplopia. Removal of bone has the advantage of reducing the amount of frontal lobe retraction required to expose a lesion during neurosurgery.33 Even with bone removal, some patients show persistent frontal lobe imaging abnormalities from retraction (Fig 5A). The gyrus rectus can be removed to gain better exposure, but one must balance the loss of brain tissue against the risks posed by removal of the orbital roof.34–37 It is likely that orbital roof removal will remain a necessary tactic for some pterional operations. Therefore, it is important to devise methods to minimize postoperative diplopia and ptosis. When possible, the orbital periosteum should be left intact to create a barrier between the dura mater and the orbital contents. In a recent study of orbital decompression in thyroid eye disease, Mainville and Jordan38 reported only an 11.8% rate of diplopia when the periorbita was left intact, compared with a 40.0% rate when it was violated. The orbital fat overlaying the superior rectus–levator complex should be disturbed as little as possible. Animal models have confirmed that changes in fat localization and subsequent tethering of tissues can lead to extraocular movement dysfunction.39,40

After the orbital bone fragment is removed, additional roof often is resected using a rongeur. This technique should be used sparingly to minimize the orbital roof defect. Extraocular muscles have a natural tendency to adhere to dura mater because it is histologically identical to sclera. In fact, extraocular muscle flaps have been exploited to repair bony orbital defects.41 By limiting the size of the bony defect, one may reduce the risk of muscle adhesion to the orbital roof and frontal meninges.

An implant is sometimes used to close orbital wall defects, although it is not proven that leaving a defect open results in a higher incidence of diplopia. Implants have been constructed from a wide variety of materials, including autologous bone, cartilage, dura mater, hydroxyapatite, titanium mesh, polydioxanone, polyethylene, polytetrafluoroethylene, nylon, glass, silastic rubber, and polyester.42 Porous implants become incorporated into surrounding tissues, which seems to reduce the rate of extrusion, migration, and fibrous encapsulation.43,44 However, which material is optimal for orbital wall reconstruction remains unclear. Two patients in our series demonstrated ptosis and diplopia, despite repair with a Medpor titanium mesh implant.

In all 8 cases, ptosis and limited ductions improved in the months after surgery. Prism glasses were prescribed to alleviate diplopia. Surgical correction of ptosis or diplopia was necessary in half the patients. Rather than operating to free the adherent superior rectus muscle, we performed inferior rectus muscle recession using an adjustable suture technique. Ptosis was repaired by advancement of the levator complex should be disturbed as little as possible. Animal models have confirmed that changes in fat localization and subsequent tethering of tissues can lead to extraocular movement dysfunction.39,40

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References


Desai et al. Diplopia and Ptosis in Pterional Craniotomy
Footnotes and Financial Disclosures

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Abbreviations and Acronyms:
CT = computed tomography; MRI = magnetic resonance imaging; PD = prism diopters.

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