Published in final edited form as:

Ophthalmology. 2015 March ; 122(3): 631–638. doi:10.1016/j.opththa.2014.09.011.

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

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Abstract

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

Design—Case series.

Participants—Eight study patients, ages 44 – 80 years, 7 female, with neuro-ophthalmic symptoms after pterional craniotomy.

Methods—Case description of eight 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 two had limitation of

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There are no conflicts of interest pertaining to this study

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abduction. Imaging showed loss of the fat layers which normally envelop the superior rectus/levator palpebrae superioris. The muscles appeared attached to the defect in the orbital roof. Ptosis and diplopia developed in two patients despite Medpor titanium mesh implants. Deficits in all patients showed spontaneous improvement. In two patients a levator advancement was required to repair ptosis. In three 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 is sometimes compromised by muscle attachment to the lateral orbital osteotomy. This syndrome occurs in about 1% of patients after removal of the orbital roof and can be treated, if necessary, by prism glasses or surgery.

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, orbito-zygomatic 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 eight 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 following 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.

MATERIALS AND METHODS

This study was approved by the UCSF Institutional Review Board and adhered to the tenets of the Declaration of Helsinki. The electronic patient records of a single neuro-ophthalmologist (JCH) from 1999 to 2013 were searched to identify patients who were referred after pterional craniotomy for either ptosis, diplopia, or limitation of eye movements. Patients with a malignancy, benign tumor invading the orbit, or cranial nerve palsy were excluded. There were eight patients who met the search criteria (Table). In each case a portion of the orbital roof had been removed to gain better access to the anterior cranial fossa during the pterional approach. The operative report, CT or MRI imaging, intracranial pathology, neurovisual examination, ocular deviation, and degree of ptosis were reviewed. The subsequent course of treatment was recorded. Patients were contacted and invited to return to the clinic to document their long term outcome.

The operative technique varied from case to case, depending on the lesion being resected. The patient was placed supine on the operating room table with the head stabilized in a Mayfield head holder and turned 45° to the side. A pterional skin incision was made with a scalpel and clips were applied to the skin edges for hemostasis. The scalp was elevated anteriorly and the superficial temporal fascia was dissected as a free flap from the deep temporalis fascia to protect the facial nerve. Once the zygoma and the superior and lateral orbital rims were exposed, the periorbital was dissected away from the inner surface of the orbital wall. The temporalis muscle was reflected inferiorly. A pterional craniotomy was performed with a Midas Rex drill saw 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 medio-laterally, and a cut in the lateral orbital 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 located underneath the frontal lobe.

After lesion resection the dura was closed with running #4-0 Dexon suture. The pterional bone flap and orbital bone fragment were replaced with hydroxyapatite cement, plates, and screws. In some patients a Medpor titanium mesh was used to close the orbital roof defect. The temporalis muscle and its fascia were re-approximated with interrupted #3-0 Dexon sutures and the galea was closed with running #2-0 Dexon suture. A subgaleal drain was placed and brought out through a separate skin incision.

RESULTS

CASE DESCRIPTIONS

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. A year later there was nearly complete absence of elevation (Fig. 2). In primary position there were 30 prism-diopters of right hypotropia and 12 prism-diopters of right esotropia. In upgaze there were 45 prism-diopters 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 three-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 upwards in mid-course, where it appeared adherent to soft tissue filling the bony defect. A coronal T1-weighted MR scan showed disruption of the superior rectus and levator palpebrae superioris muscles (Fig. 4A). The normal fat plane between the two muscles could not be identified. In addition, the layer of

fat that usually surrounds the two 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 towards 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, employing an adjustable suture technique. Forced ductions under anesthesia just prior to the muscle surgery showed restriction of elevation. After surgery, the patient was able to fuse in primary gaze.

Patient 2 is a 69-year-old woman with a history of vision loss in the right eye from a sphenoid wing meningioma. The lesion was resected via a right fronto-temporal-sphenoidal craniotomy. The orbital roof was removed up to the superior orbital fissure using rongeurs. Difficulty was encountered owing to abnormal thickness of the sphenoid bone. The Midas Rex drill was used, requiring bipolar cautery, bone wax, and Surgicel to achieve hemostasis. Although the orbital contents were protected with cottonoid sponges, the levator palpebrae superioris and superior rectus muscles were damaged.

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 eight years after meningioma resection. She reported diplopia only on upgaze. The ductions in the right eye were full, except for 90% elevation with 10 prism-diopters of hypotropia in upgaze. A coronal Flair MR image showed direct contact between the levator palpebral superioris/superior rectus muscle 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 was also absent.

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 was then exposed under the operating microscope using a drill. After tumor removal, Floseal hemostatic matrix was applied to the undersurface of the frontal lobe. The defect in the orbital roof was closed with a titanium mesh Medpor implant.

Examination two months after surgery showed 5 mm of ptosis in the right eye. There was only 50% elevation of the right eye, with 20 prism-diopters of hypotropia in primary gaze. An orbital MR 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 completely resolved. There was a persistent elevation paresis, with 7 prism-diopters of

right hypotropia in primary gaze. Prism glasses were prescribed. By five 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 prism-diopters of right hypotropia on upgaze.

Patient 4 is a 44-year-old woman who developed vision loss 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 following the surgery the patient had no light perception in the right eye. The eyelids were closed because of peri-orbital 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 post-operative changes, clip artifacts, and a defect in the orbital roof.

Examination nine years later showed slightly limited elevation of the right globe. There were 20 prism-diopters of sensory exotropia.

Patient 5 is a 53- year-old woman who suffered 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. Following surgery she developed hydrocephalus which required placement of a ventriculoperitoneal shunt.

On examination eight months later there were 4 mm of ptosis of the right eyelid and only 50% elevation of the globe. Ocular alignment was achieved with 12 prism-diopters base-up prism in the right eye. An MR of the orbits showed loss of the fat signal in the superior orbit and alteration in the appearance of the superior rectus/levator muscles. The patient objected to 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 is a 55-year-old woman who suffered 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 two months after surgery revealed 1–2 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 post-operative day. The orbital images were not adequate in 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 is a 54-year-old woman female who developed 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.

On examination six weeks after surgery there was a left frontalis muscle palsy, 50% elevation deficit, and a nearly total abduction deficit. In primary gaze there were 25 prism-diopters of left hypotropia and 25 prism-diopters of left esotropia. MR imaging showed a large defect in the left orbital roof and post-operative changes in the appearance of the lateral rectus and the levator palpebrae superioris/superior rectus.

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

The day following surgery the patient reported vertical double vision. There was eyelid swelling with ptosis. Globe elevation was slightly reduced and depression was absent. Forced ductions showed restriction of depression. There were 40 prism-diopters of left hypertropia in primary gaze. A CT scan obtained two 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/superior rectus muscles pulled upwards. A more posterior cut (Fig. 6B) showed the Medpor implant, with vertical distortion of the muscles. Three months following surgery there was a persistent depression deficit with a left hypertropia measuring 10 prism-diopters.

DISCUSSION

The pterional approach has undergone numerous modifications to improve access to the sellar region and the anterior cranial fossa.⁸ 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.⁹ 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 one 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 may also 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 downwards eye rotation. In addition, two patients had limited abduction with esotropia. In these cases, removal of the lateral orbital wall appeared to cause adhesion of the lateral rectus muscle (Fig. 3).

Kaeser and Klainguti were the first to recognize the problem of vertical diplopia from pterional craniotomy.¹¹ 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 neurosurgery literature, a review of 75 patients who underwent pterional surgery identified two (2.6%) cases of extraocular muscle restriction and three cases (4.0%) of ptosis.¹² The true rate of neuro-ophthalmic complications following 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 two 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 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 etiology is a blowout fracture of the orbital floor or medial wall from blunt trauma.^{13–16} Diplopia may occur from muscle entrapment, neuromuscular trauma, or extraocular muscle scarring. Kushner has emphasized that the vertically acting muscle can show either restriction or paresis.¹⁷ In surgical decompression for thyroid eye disease, bony apertures are left deliberately in the orbital walls. Diplopia and gaze limitation occur in 2–43% 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 has been also been described in patients undergoing medial and superior orbital periosteum removal for frontal sinus, anterior skull base and craniofacial procedures.^{28, 29} In such cases, diplopia is believed to result from tethering of the superior oblique tendon or malposition of the trochlea. Lastly, post-operative diplopia has been reported after breach of the orbital wall during endoscopic paranasal sinus surgery.^{30–32}

Although Yasargil was among the first to remove the orbital roof to improve exposure, he later discarded this strategy once he recognized the risk of post-operative diplopia.⁸ 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 colleagues reported only an 11.8% rate of diplopia when the periorbital was left intact, compared with a 40.0% rate when it was violated.³⁸ 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 extra-ocular movement dysfunction.^{39, 40}

After the orbital bone fragment is removed, additional roof is often 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 appears to reduce the rate of extrusion, migration, and fibrous encapsulation^{43, 44}. However, it remains unclear which material is optimal for orbital wall reconstruction. Two patients in our series developed ptosis and diplopia, despite repair with a Medpor titanium mesh implant.

In all 8 cases, ptosis and limited ductions improved in the months following 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 palpebrae superioris. Normal function can be achieved for patients in primary gaze, but follow-up data collected a mean of 6.5 years after surgery revealed slight limitation of globe elevation or depression in every patient.

Acknowledgments

Funding Support: National Eye Institute and Research to Prevent Blindness.

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Figure 1. Pterional craniotomy (gray shading) and orbito-zygomatic craniotomy (yellow shading) allow removal of two separate bone pieces for optimal exposure of lesions in the anterior cranial fossa. Additional bone (dashed line) is often removed piecemeal from the orbital roof, leaving a defect.

Figure 2.

Patient 1. Limited elevation in the right eye, with 30 prism-diopters of hypotropia in primary gaze following pterional craniotomy. There is essentially no ptosis. Horizontal gaze shows slight limitation of abduction in the right eye, producing a small esodeviation.

Figure 3.

Patient 1. (A) CT reconstruction revealing right orbital roof defect (B) Oblique cut demonstrating tenting of the superior rectus/levator palpebrae superioris complex (white arrow). (C) Corresponding image through the other orbit showing normal trajectory of the muscle complex (black arrow).

Figure 4. Patient 1. (A) Coronal T1-weighted MR showing abnormal right superior rectus and levator palpebrae superioris. The normal fat signal that surrounds the muscles on the left side (white arrows) is disrupted on the right side. (B) Contrast-enhanced image showing bowing of the right lateral rectus (black arrow).

Figure 5.

Patient 2. **(A)** Eight years after surgery, coronal flair MR showing direct contact between the right superior rectus/levator palpebrae superioris and the basal meninges of the frontal lobe, with peaking of the muscle complex (white arrow). Note abnormal bright signal in frontal white matter from brain retraction during pterional craniotomy. **(B)** Post-gadolinium image showing abnormal enhancement of the basal meninges (black arrowheads), which is contiguous with the superior rectus/levator complex. On the normal side the dark contour representing orbital roof is intact (white arrowheads), along with the fat cushion between the orbital roof and muscle complex.

Figure 6.

Patient 8. **(A)** CT showing saw cut (black arrow) in the left orbital roof with incarcerated soft tissue that appears to draw upwards the superior rectus/levator palpebrae superioris complex (see insets comparing normal and altered sides). **(B)** More posterior cut, showing vertically distorted superior rectus/levator complex. The roof defect has been closed with a Medpor implant (white arrows), but incompletely.

Table

Summary of patients with neuro-ophthalmic complications from orbital roof removal during pterional craniotomy.

Patient	Neurosurgical Lesion	Motility Deficit	Primary Gaze Deviation	Ptosis	Follow-up/Intervention
1) 55♂	Anterior Communicating Artery Aneurysm	Right Elevation & Abduction	30 Right Hypotropia & 12 Right Esotropia	No	Left Inferior Rectus Recession
2) 69♀	Right Sphenoid Wing Meningioma	Right Elevation & Depression	Orthotropia	Yes	Levator Repair
3) 58♀	Right Sphenoid Wing Meningioma	Right Elevation	20 Right Hypotropia	Yes	Prism Glasses
4) 44♀	Right Supraclinoid Internal Carotid Artery Aneurysm	Right Elevation & Depression	Right Hypotropia	Yes	No Light Perception Right Eye, No Intervention
5) 53♀	Anterior Communicating Artery Aneurysm	Right Elevation	10 Right Hypotropia	Yes	Right Inferior Rectus Recession & Levator Repair
6) 55♀	Anterior Communicating Artery Aneurysm	Left Elevation	Orthotropia	Yes	No Treatment Needed
7) 54♀	Left Middle Cranial Fossa Meningioma	Left Elevation & Abduction	25 Left Hypotropia & 25 Left Esotropia	Yes	Left Inferior & Medial Rectus Recession
8) 80♀	Left Sphenoid Wing Meningioma	Left Elevation & Depression	20 Left Hypertropia	Yes	Monitoring