ORBITAL VARIX THROMBOSIS*

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INTRODUCTION

ORBITAL VARICES ARE USUALLY CONSIDERED TO BE A CONGENITAL VENOUS malformation of the orbit manifested by intermittent proptosis, induced by changes in venous pressure. They are the most frequently encountered primary orbital venous abnormality and are invariably noted by orbitologists in most series of orbital lesions.[1-9] An occasional feature of orbital varices is sudden thrombosis.[2,4,10-12] We have seen three adult male patients who presented with signs and symptoms suggestive of an acute orbital process who subsequently were found to have thrombosed orbital varices.

CASE REPORTS

CASE 1

A 38-year-old man presented with a 2-week history of diplopia and right orbital pain. Past medical history was unremarkable, with the exception of hemorrhoids. Examination revealed visual acuities of 20/20 OU, 6 mm of right eye proptosis, 3 mm of right hypoophthalmos, a palpable fullness of the right upper eyelid, resistance to retropulsion of the right globe, and slight right conjunctival injection. Diplopia was present in all fields of gaze and there was a marked reduction of elevation of the right eye (Figs 1 to 3). Pupillary, slit lamp, and dilated funduscopic examinations were normal. Intraocular pressures were 20 mm Hg in the right eye and 12 mm Hg in the left eye.

Contact B-scan ultrasonography showed an anechoic mass in the right superior orbit (Fig 4). Computed tomography (CT) of the orbits showed a partially enhancing, intraconal soft tissue mass (Fig 5); hemorrhage was noted within the mass (Fig 6).

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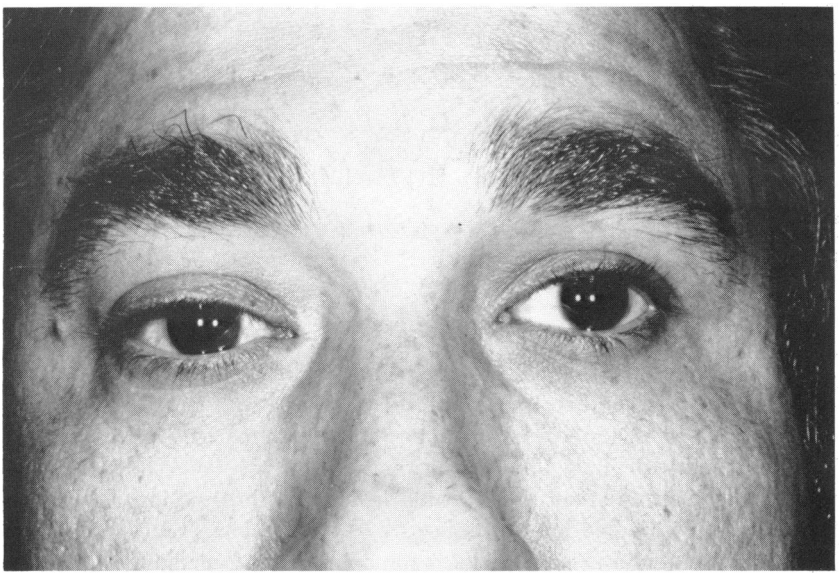

FIGURE 1
External photograph (case 1) showing right hypoophthalmos, and minimal conjunctival congestion.

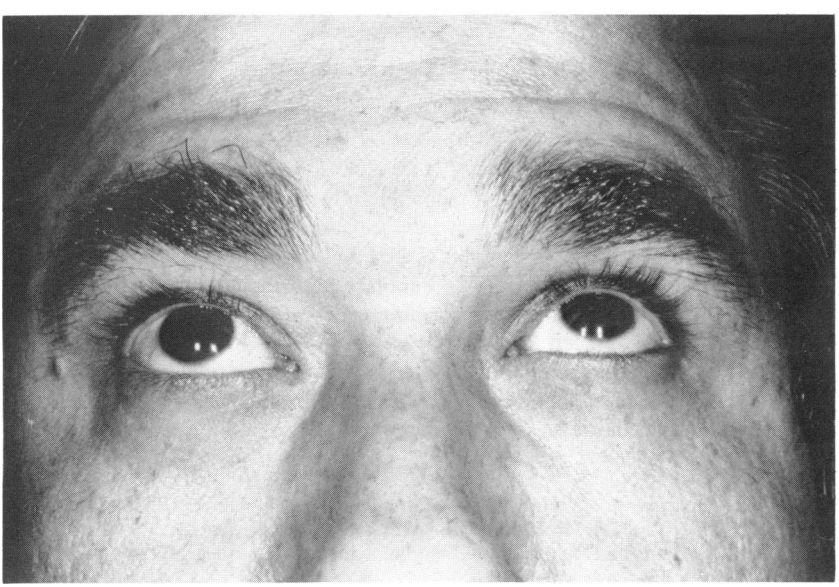

FIGURE 2
External photograph (case 1 - upgaze) showing reduction of elevation of the right eye.

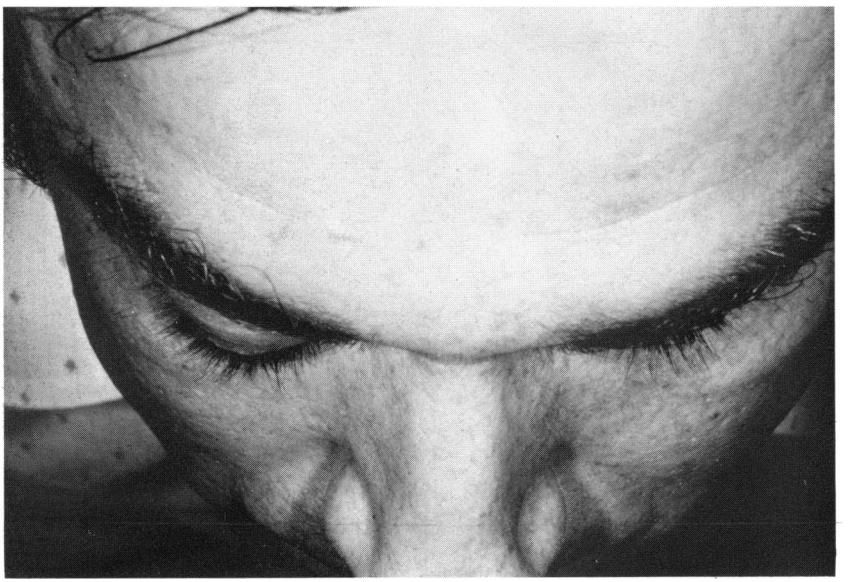

FIGURE 3
External photograph (case 1) showing right proptosis.

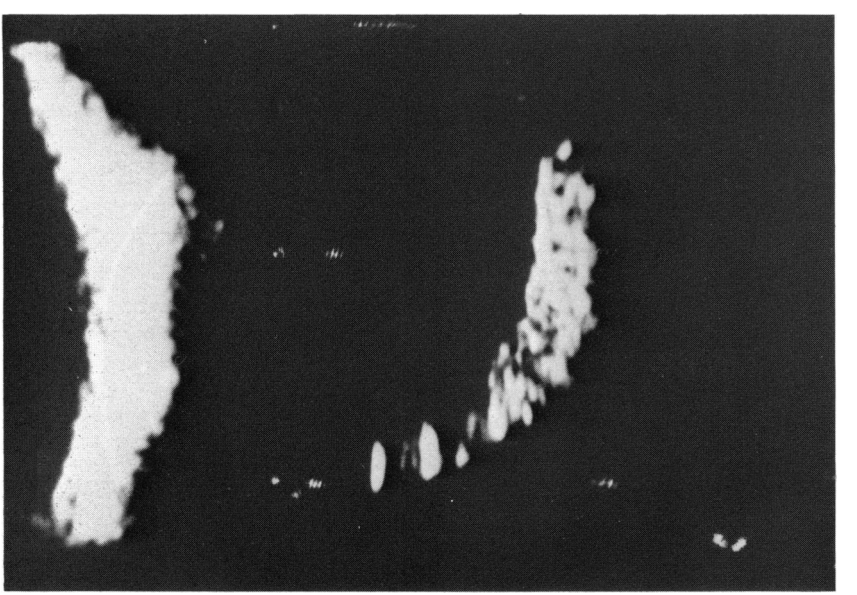

FIGURE 4
Contact B-scan ultrasonogram (case 1) showing anechoic mass in right superior orbit.

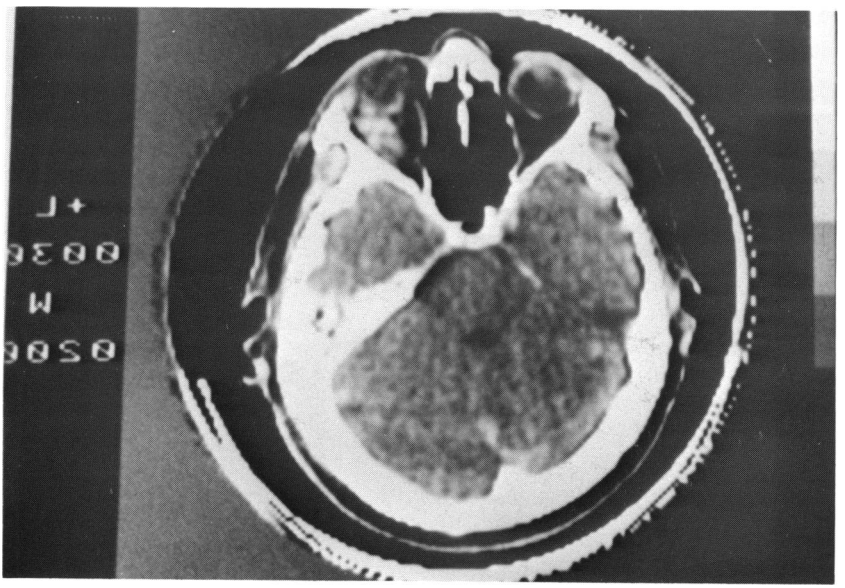

FIGURE 5
CT scan (case 1) showing apartially enhancing intraconal soft tissue mass in the right orbit.

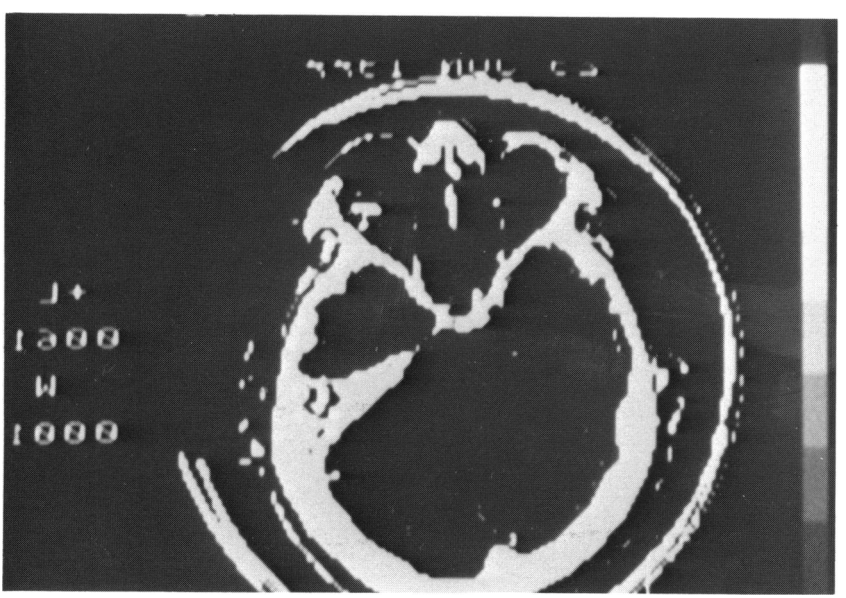

FIGURE 6
CT scan (case 1) using special densitometry settings, showing blood clot within the soft tissue mass.

Nine days after presentation a lateral orbitotomy was performed. At surgery, a large purplish mass was found within the muscle cone. The mass was incised and biopsied anteriorly and a large blood clot was evacuated (Fig 7). The posterior portion of the lesion was left undisturbed. On the first postoperative day the patient developed an orbital hemorrhage, which resolved along with the usual postoperative edema. A contrast enhanced CT scan performed (without the Valsalva maneuver) 4 months after surgery showed no evidence of a right orbital lesion (Fig 8). However, a venogram performed 9 months postoperatively showed a varix arising from the right superior ophthalmic vein (Fig 9).

Two years postoperatively the patient was asymptomatic with visual acuities of 20/20 OU, an 8 prism diopter exophoria at near, and full extraocular motility (Fig 10). No increase in exophthalmometry measurements was noted by the Valsalva maneuver. Twelve years postoperatively, the examination was unchanged. The patient had occasional fullness in the right orbit with straining or bending but no proptosis occurred with the Valsalva maneuver. Exophthalmometry showed 1 mm of right enophthalmos.

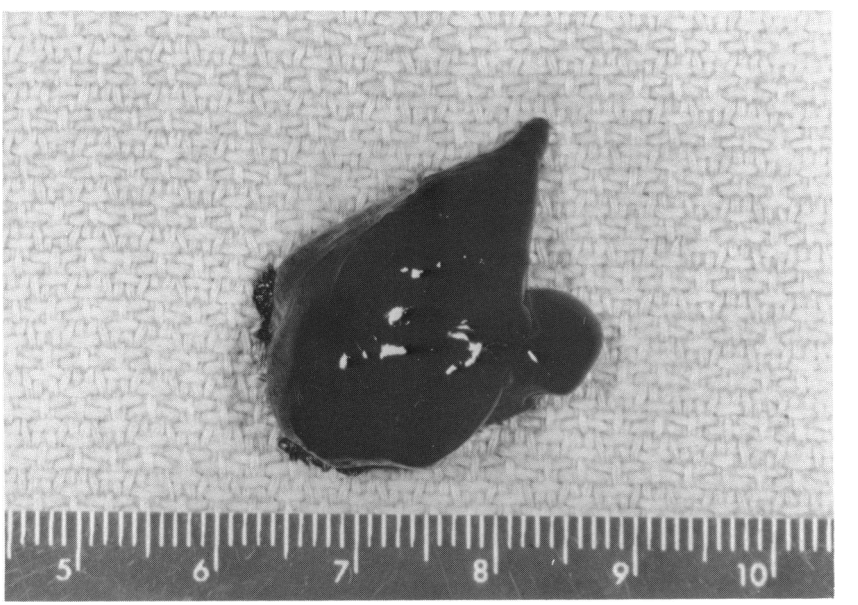

FIGURE 7
Gross photograph of blood clot evacuated from varix (case 1).

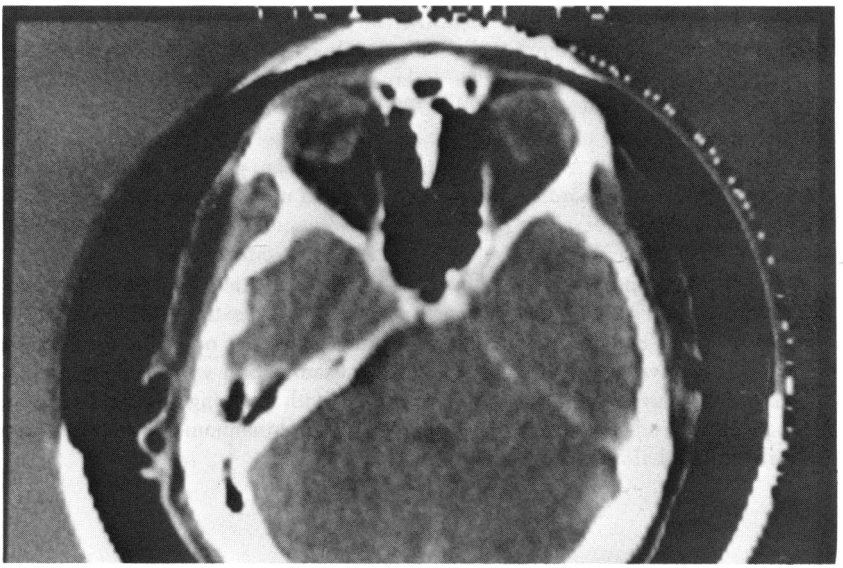

FIGURE 8
Contrast enhanced CT scan (case 1), performed without Valsalva maneuver, 4 months after surgery showing no evidence of right orbital lesion.

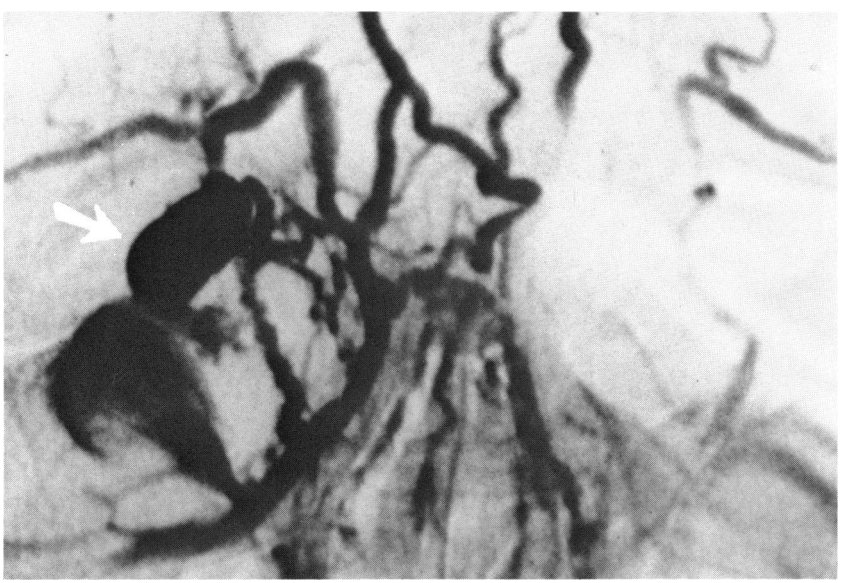

FIGURE 9
Orbital venogram (case 1) performed 9 months after surgery showing varix arising from the right superior ophthalmic vein (*arrow*).

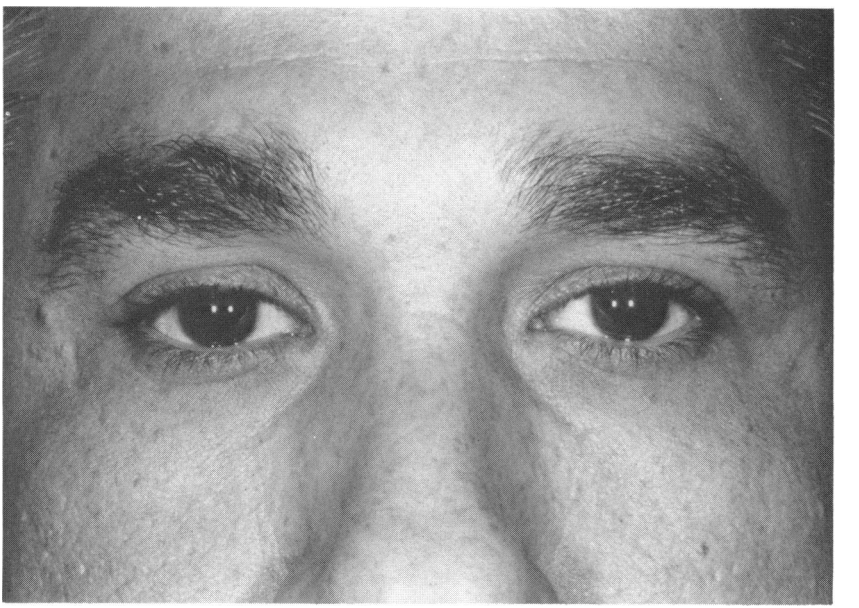

FIGURE 10
External photograph (case 1) 2 years after surgery.

CASE 2

A 45-year-old man presented with a 7-day history of a swollen, painful right eye. He had been struck in the brow area with a beer bottle 27 years previously and for the past 6 years had experienced intermittent proptosis of the right eye. Past medical history was positive only for hemorrhoids.

On examination the visual acuity measured 20/50+ in the right eye and 20/40 in the left eye. There was a well-healed scar in the right brow. The right globe was tender to palpation and resistant to retropulsion. There were 3 mm of proptosis of the right eye (Fig 11) which was unchanged with a Valsalva maneuver. Examination of the pupils, anterior segments by slit lamp, and dilated fundi were all normal. The cup to disc ratio was 0.4 OU, and the intraocular pressures were 27 and 26 mm Hg in the right and left eye, respectively. There was a 6 prism diopter intermittent right exotropia and versions were full. The visual fields were normal.

B-scan ultrasonography showed an anechoic right superior orbital mass (Fig 12). A CT scan showed a large (3.2 × 1.9 cm), dense, well-defined, smooth-walled, posterior, superior, intraconal mass in the right orbit, which appeared to arise from the superior ophthalmic vein (Figs 13 and 14). Hemorrhage was noted within the mass (Fig 15).

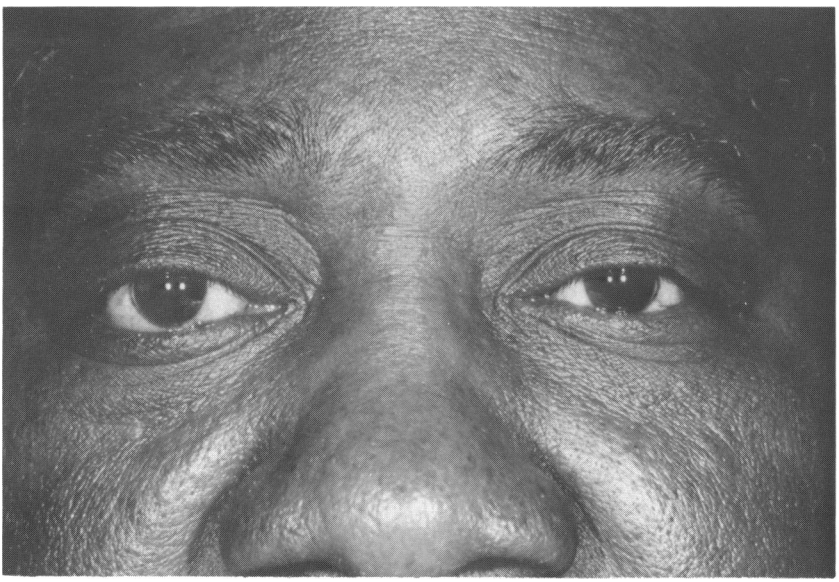

FIGURE 11
External photograph (case 2) showing right proptosis.

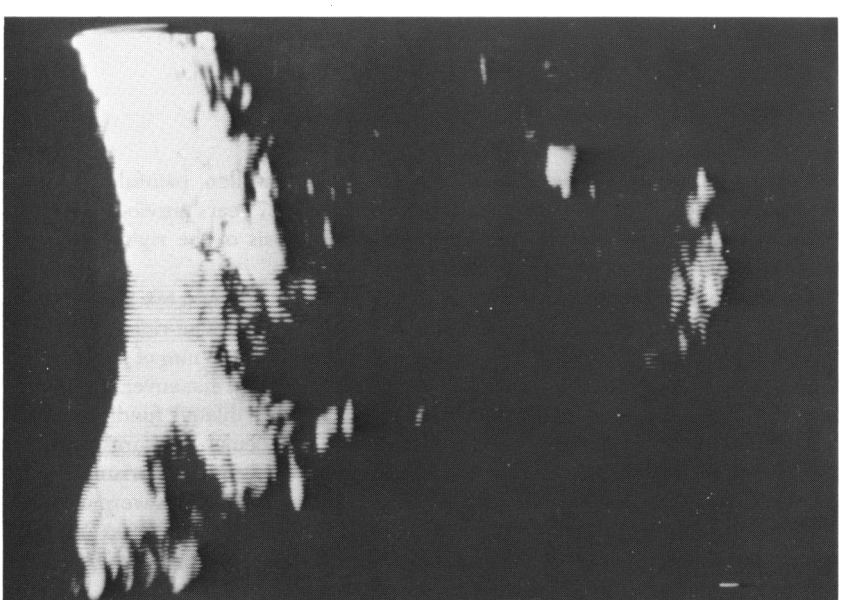

FIGURE 12
Contact B-scan ultrasonogram (case 2) showing an anechoic mass in the right superior orbit.

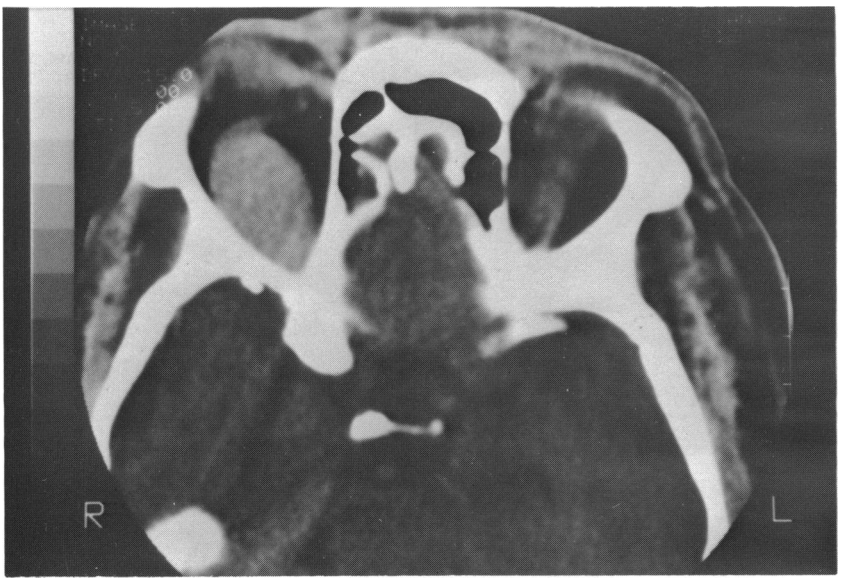

FIGURE 13
Noncontrast enhanced computed orbital tomographic scan (case 2) showing a 3.2 × 1.9 cm dense, well-defined, smooth-walled posterior, superior intraconal mass in the right orbit, arising from the superior ophthalmic vein.

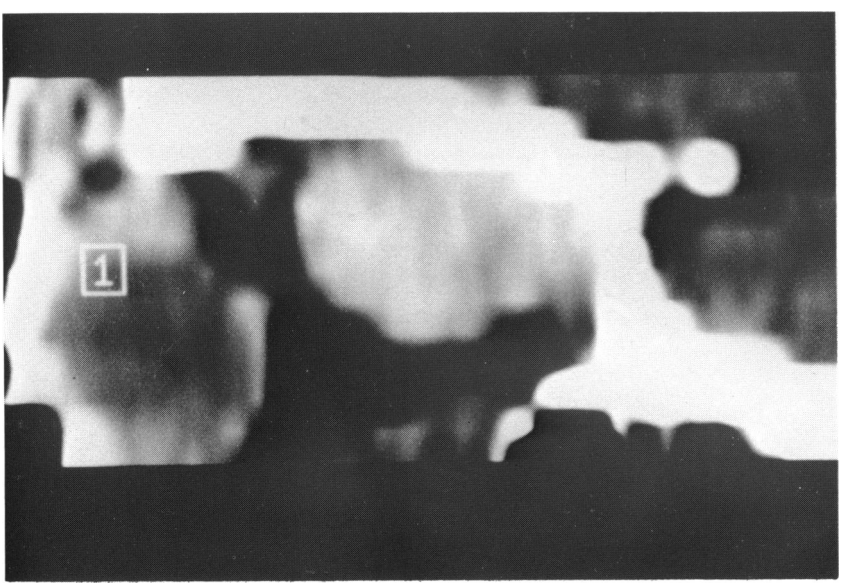

FIGURE 14
Reconstructed saggital CT scan (case 2) showing superior orbital mass.

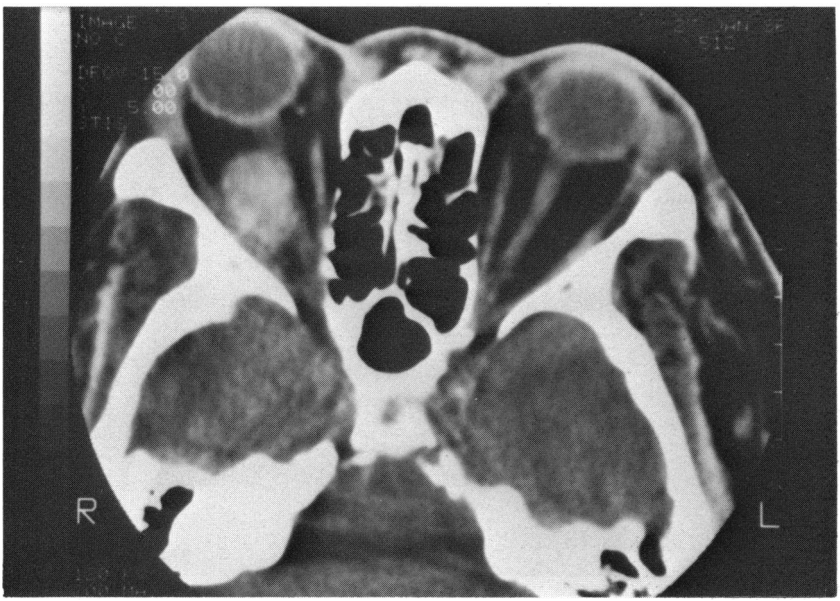

FIGURE 15
Noncontrast enhanced computed orbital tomographic scan (case 2) showing hemorrhage within the orbital mass.

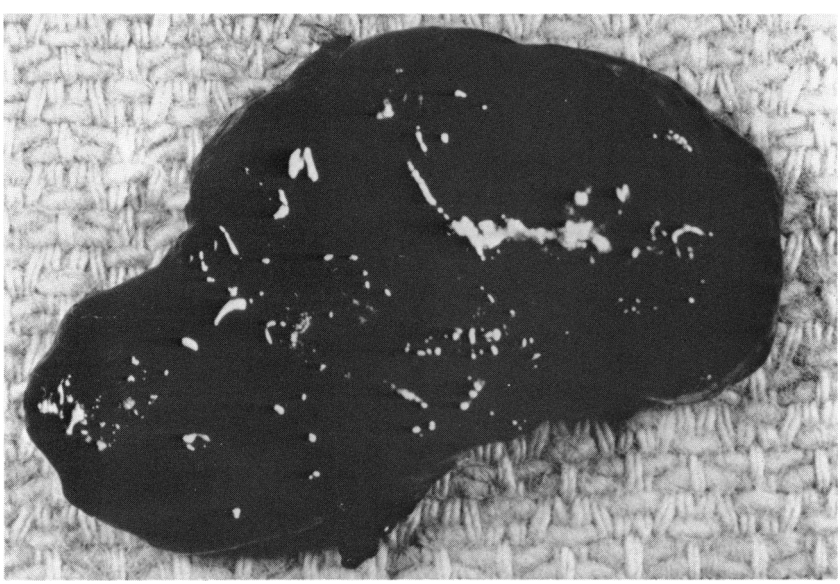

FIGURE 16
Gross photograph of blood clot removed from varix (case 2).

Three days later the right orbit was explored via a orbit approach. A large soft reddish-purple mass arising from the superior ophthalmic vein was noted extending to the apex of the orbit. The varix wall was incised and a large blood clot was removed (Fig 16). The lesion was partially excised and cauterized, leaving the most posterior portion undisturbed.

Examination 12 months postoperatively revealed visual acuities of 20/25− in the right eye and 20/20 in the left eye. The orbitotomy incision was well-healed and versions were full (Fig 17). The intraocular pressure measurements were 13 mm Hg OU on 0.5% timolol, twice a day, OU. The patient stated that he had right sided head pain upon smoking and lying down. The Valsalva maneuver produced no proptosis.

CASE 3

A 75-year-old man presented with a 6-day history of intermittent diplopia, "pressure behind the eyes," and blurred vision. Past medical history was positive only for excision of a basal cell carcinoma of the right temple 5 years prior to presentation.

Visual acuities were 20/30 OU. There was slight right hyperophthalomos (Fig 18), 2 mm of right eye proptosis (Fig 19) and resistance to retropulsion of the right globe. Pupils, anterior segments, and fundi were normal. The intraocular pressures measured 16 mm Hg in the right eye and 15 mm Hg in the left eye. Extraocular muscle movements were full with an exophoria of 2 prism diopters at distance and 11 prism diopters at near.

A contrast enhanced CT scan revealed a large (2.3 × 2.0 cm), well-defined, intraconal, enhancing soft tissue mass extending to the apex of the orbit. A blood clot within the mass could not be identified with certainty. The lesion was located inferior to the optic nerve, and appeared to arise from the inferior ophthalmic vein (Figs 20 and 21).

The patient underwent a lateral orbitotomy 12 days later. The varix wall (Fig 22) was incised and blood clots were evacuated; cautery was applied to obliterate the varix.

Eight months postoperatively the ocular examination revealed visual acuities of 20/40 OU. Exophthalmometry was symmetric. A 2 to 3 prism diopter right hypotropia and a 5 prism diopter right exotropia were noted (Fig 23). The Valsalva maneuver produced no proptosis of the right eye.

PATHOLOGY

Blood clots were evacuated from each patient's lesion at the time of surgery (Figs 7 and 16). The histopathologtic findings were similar in all three cases: recent laminated and organizing thrombi with recanalization were found (Figs 24 to 26). The vessel walls showed thickening, stromal fibrosis and smooth muscle fibers (Figs 27 and 28). The Masson trichrome stain was helpful in identifying the smooth muscle fibers (Figs 29 and 30).

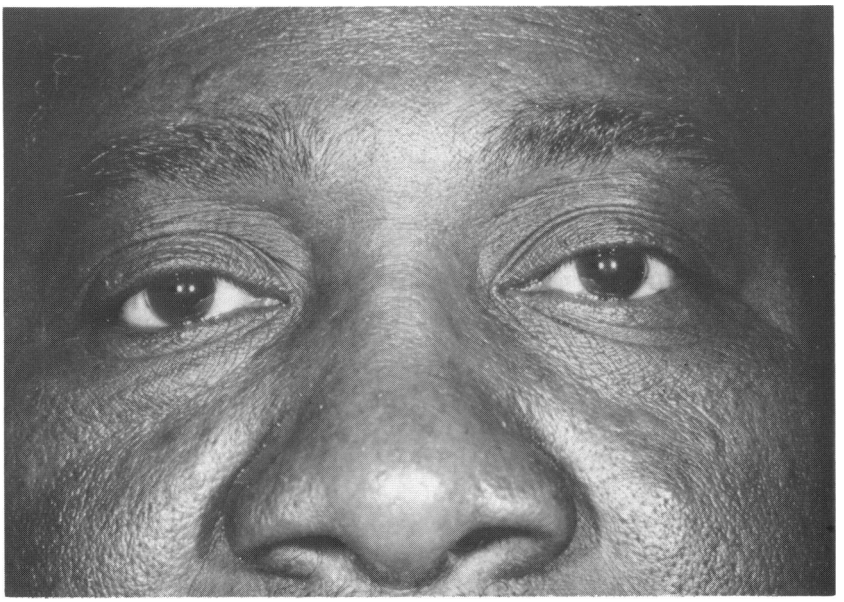

FIGURE 17
External photograph (case 2) 32 months postoperatively.

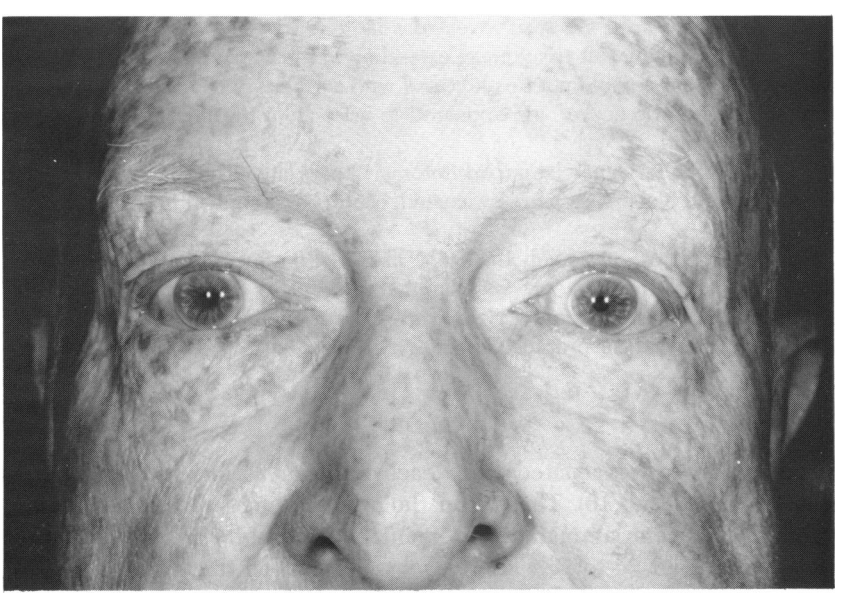

FIGURE 18
External photograph (case 3) showing slight right hyperophthalmos.

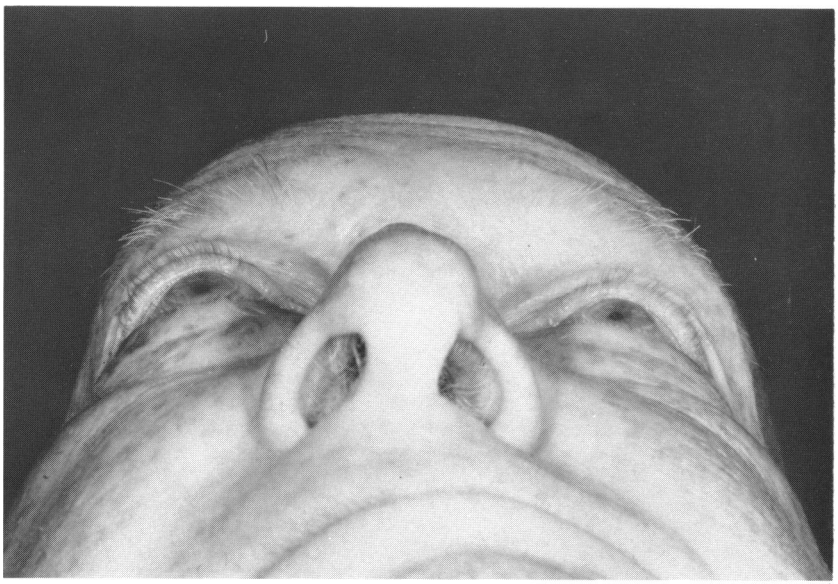

FIGURE 19
External photograph (case 3) showing right proptosis.

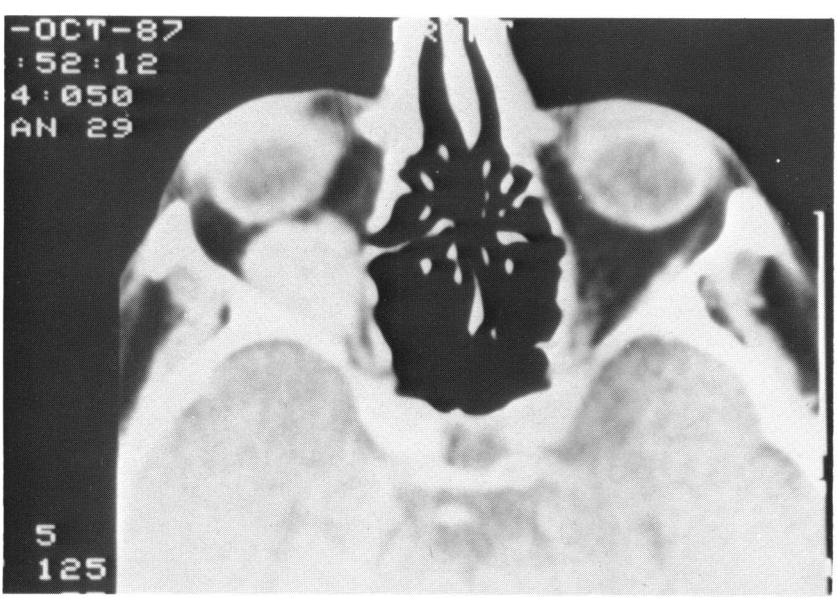

FIGURE 20
Contrast enhanced CT scan showing 2.3 × 2.0 cm well-defined, inferior intraconal enhancing soft tissue mass extending to the apex of the right orbit (case 3).

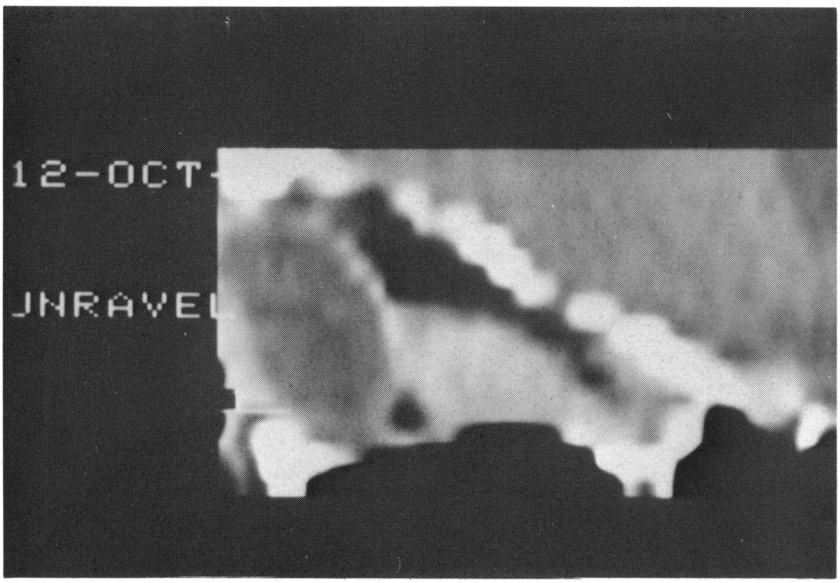

FIGURE 21
Reconstructed saggital CT scan (case 3) showing inferior orbital mass.

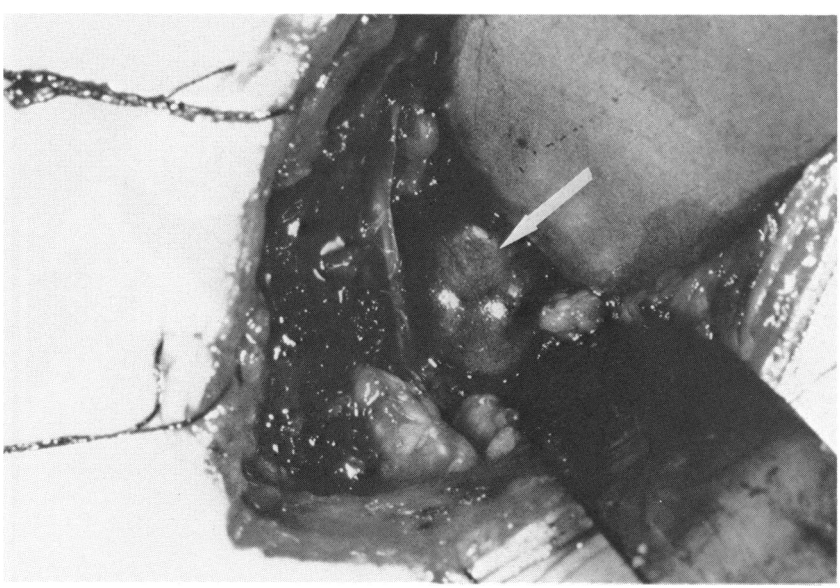

FIGURE 22
Intraoperative photograph (case 3) showing varix (*arrow*).

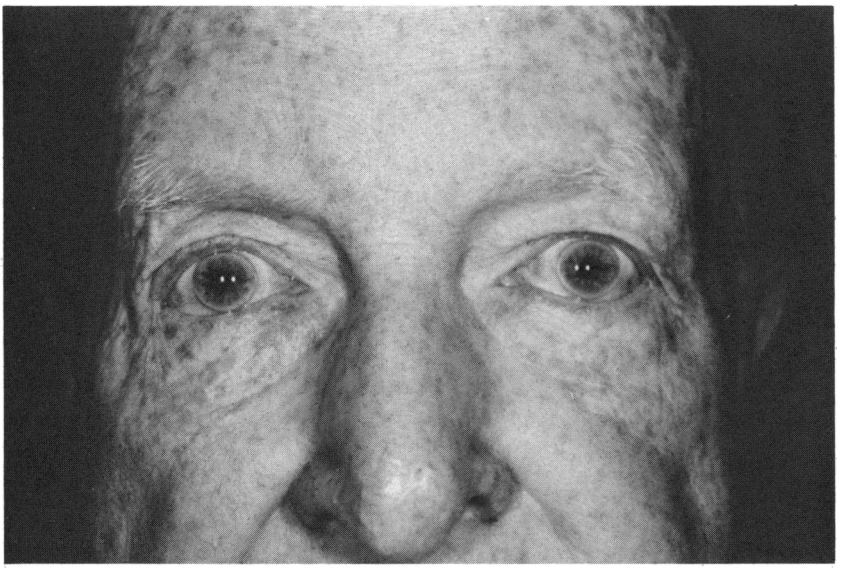

FIGURE 23
External photograph (case 3) 8 months after surgery.

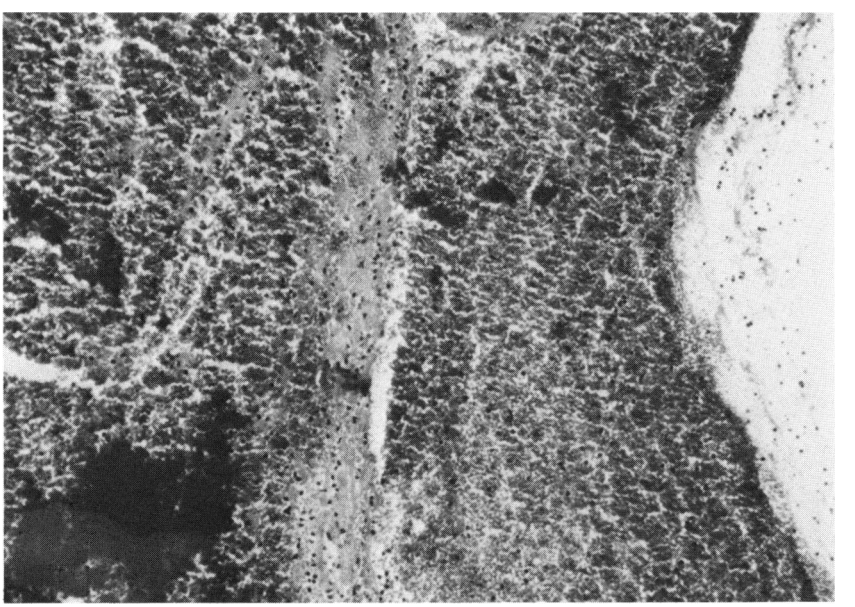

FIGURE 24
Photomicrograph showing laminated recent thrombus (case 1) (H&E, original magnification, ×100).

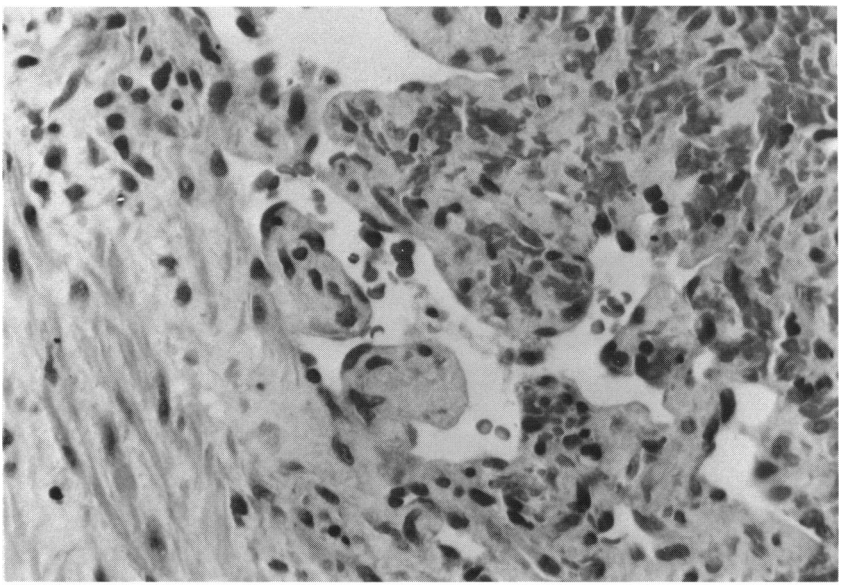

FIGURE 25
Photomicrograph (case 3) showing organizing thrombus with recanalization (H&E, original magnification, ×400).

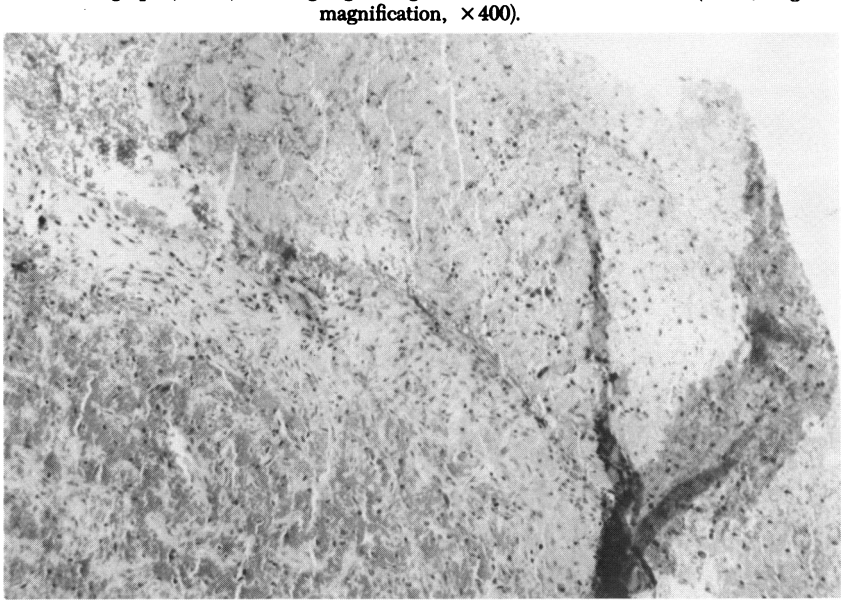

FIGURE 26
Photomicrograph (case 3) showing an organizing thrombus with an overlying more recent fibrin thrombus attached to a vessel wall (H&E, original magnification, ×100).

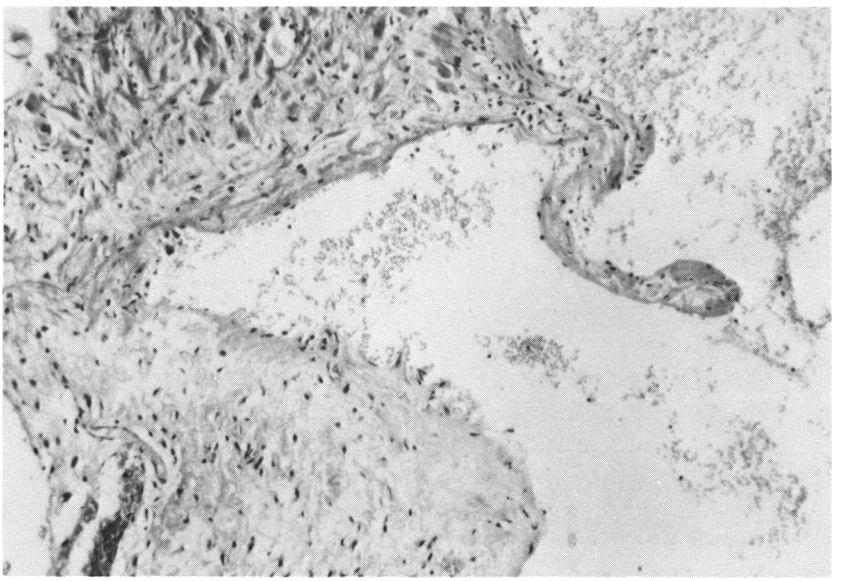

FIGURE 27
Photomicrograph showing an abnormally scarred and thickened vessel wall (case 1) (H&E, original magnification, ×100).

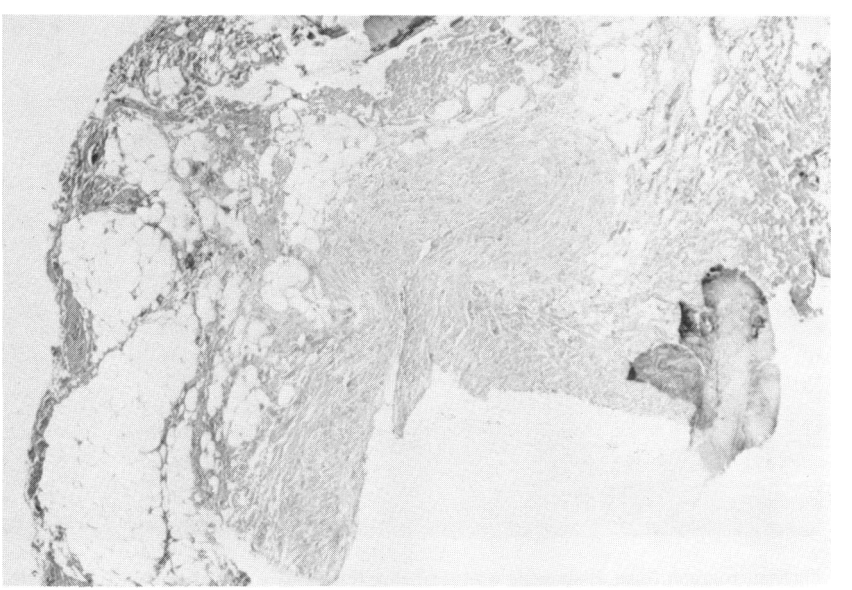

FIGURE 28
Photomicrograph (case 3) showing a thickened variceal vessel wall (H&E, original magnification, ×25).

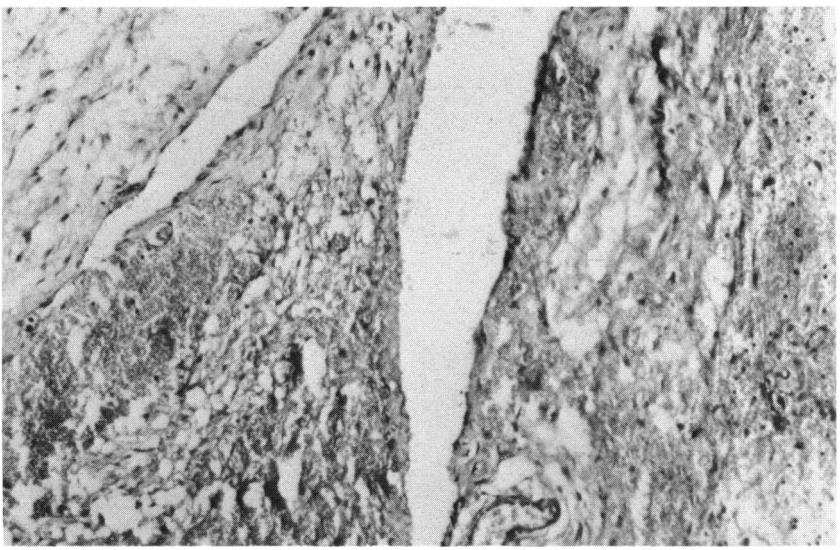

FIGURE 29
Photomicrograph (case 2) showing a variceal vessel wall (Masson trichrome, original magnification, ×100).

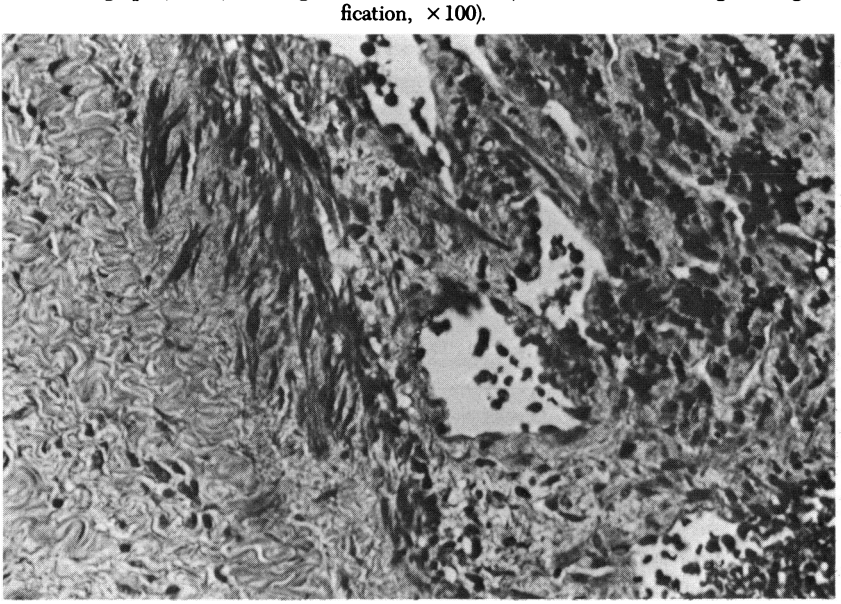

FIGURE 30
Photomicrograph (case 3) showing a recanalizing thrombus attached to a vessel wall. The vessel wall shows a greater amount of collagen than a normal vein. There is scarring of the vessel wall and a reduced amount of smooth muscle (Masson trichrome, original magnification, ×250).

DISCUSSION

Central to the understanding of orbital variceal disease is the fact that the orbital veins (along with all of the veins above the neck) have no valves.[13] As a consequence of this anatomic fact, venous blood may pool in the head and orbital region as a result of venous obstruction. Hence, intermittent proptosis is the clinical feature that is most typically associated with orbital varices. Rapidly reversible proptosis occurs as a result of increased venous pressure in the head from straining or coughing, a dependent position, Valsalva maneuver, or external pressure on the jugular veins.[14] An increase in episcleral venous and intraocular pressures resulting in ipsilateral glaucoma can occur with repeated intermittent elevation of orbital venous pressure from an orbital varix.[15] In addition, stasis of pooled blood in an orbital varix for an extended period of time may result in thrombosis.

Orbital varix thrombosis must be differentiated from other manifestations of orbital venous thrombosis: cavernous sinus thrombosis and superior ophthalmic vein thrombosis.[16-20] The findings of a superior ophthalmic vein thrombosis include: increased intraocular pressure, retinal venous congestion, and conjunctival chemosis, venous dilation, and tortuosity.[20] With a cavernous sinus thrombosis, in addition to the above findings, there is papilledema and massive edema of the orbit and eyelids. The above clinical signs occur secondary to thrombosis or external compression of a functional orbital vein. These clinical changes occur suddenly and represent an acute compromise to the orbital venous drainage system. These findings were not noted in our cases of thrombosed orbital varices.

The varices in our patients had been indolent and possibly thrombosed minimally allowing a collateral venous circulation to develop and adequately maintain the venous drainage demands of the eye and orbit. In addition, a varix is a venous anomaly that does not replace a normally functioning venous drainage system. Our patients presented with acute thrombosis of a varix, simulating a tumor, and the findings on the examination of these patients were due primarily to a mass effect with only minimal congestive signs.

The differential diagnostic possibilities for a thrombosed orbital varix include: hemangioma, lymphangioma, inflammatory orbital pseudotumor, carotid cavernous fistula, dural shunt, or orbital varix without thrombosis. Clinically, the orbital varices in our three cases are probably acquired lesions and are distinct from the vascular abnormalities usually recognized as congenital orbital varices. The clinical, radiographic and ultrasonographic features of an orbital varix thrombosis are sufficiently

characteristic to make the differentiation from the above entities possible.

Rudolf Virchow described the pathophysiology of thrombosis. He cited three predisposing factors: (1) stasis of blood flow (from either pooling or obstruction); (2) injury to the vein wall (from either inflammation, infection or trauma); and, (3) hypercoagulability of blood. Based on Virchow's principles, the following hypothesis is proposed to explain the pathophysiology of orbital varix thrombosis:

Slight loss of tone of the perivenous supporting tissues may lead to the initial development of an acquired varix, in which stasis of blood flow occurs, leading to thrombosis. This is followed by organization of the clot and proximal dilatation of the vessel. Following organization, the thrombus recanalizes, resulting in sluggishly restored flow, which may lead to further thrombosis. Proximal dilatation further enlarges the varix. Ultimately, a large varix forms, which can acutely develop a complete thrombosis.

We believe that the body habitus of our three patients may have contributed to potential stasis of blood flow. All three patients had a stocky build with a "bull-neck." Such individuals are believed to have increased orbital venous pressure. Cataract surgeons are often concerned about an increased risk of expulsive choroidal hemorrhage in patients with this type of body habitus. Since orbital varices become manifest with specific inciting positions or maneuvers, it is possible that these patients developed variceal thrombosis after a prolonged episode of dependence or other activity that maintained an increased venous pressure in their orbits.

We have described three patients who presented as an acute orbital mass secondary to orbital varix thrombosis. The evaluation of patients presenting with these findings should include B-scan ultrasonography which demonstrates an anechoic mass. Although venography currently is not employed routinely in the evaluation of orbital tumors, it is an excellent test for evaluating patients with suspected orbital varices. The venogram well demonstrates orbital varices and may show a filling defect if thrombosis is present in the varix. Either CT scanning (with or without contrast) or magnetic resonance imaging, performed with and without the Valsalva maneuver, will establish the diagnosis of an orbital varix in patients who have intermittent proptosis.[21-23]

All three of our patients were treated surgically and had a good result. However, we suggest that if the diagnosis of orbital varix thrombosis is made clinically it may be appropriate to follow the patient conservatively. The clot can be expected to resorb or recanalize eventually, with improve-

ment in the signs and symptoms.* Although this would lead to a reduction in the orbital mass effect, it is not expected to prevent further episodes from occurring. For patients demonstrating repeated or nonresolving episodes of thrombosis of a known orbital varix, it is appropriate to evacuate the clots surgically (thus decompressing the orbit) and excise and cauterize the anterior portion of the varix in order to prevent recurrence. Other indications for surgery include severe pain, severe proptosis, and compressive optic neuropathy due to orbital mass effect. In most cases it is not possible for the surgeon to completely excise the varix.

SUMMARY

Three patients have been described with a thrombosed orbital varix. The clinical, imaging, surgical, and pathologic features of this disorder are described. A pathophysiologic mechanism has been proposed and this entity has been placed in the spectrum of orbital vascular lesions. Recommendations for the diagnostic evaluation and management of patients with thrombosed orbital varices have been offered.

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*In fact, electrically induced thrombosis has been advocated as a method of surgical management for orbital varices.[24]

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DISCUSSION

DR ROBERT R. WALLER. I would like to thank Doctor Bullock and his colleagues for providing this most interesting and beautifully illustrated paper. To start discussion, I would make four points very briefly. First, Doctor Bullock's patients are not typical for the usual clinical picture for orbital varices of the presumed congenital type. Most orbital varices we see are chronic in behavior and are almost synonymous with intermittent exophthalmos. John Henderson has written about "The transient and evanescent proptosis which has long piqued the attention of clinicians. This 'now you see it/now you don't' phenomenon is the principal sign of presumed congenital orbital varices with transient bulging of the eye and its turgid appendages." None of these patients demonstrated this sign, clinically, presenting instead as an acute orbital process.

Additionally, Doctor Bullock's patients provided no historical evidence of proptosis on command, proptosis associated with exertion or change to a dependent position or proptosis induced by pressure over the jugular vein. Also, two of the three patients were older than most patients observed with the more common form of orbital varix. One patient was 45 and another was 75 years of age. Most orbital varices are identified within the second and third decades of life. Thrombosis was evident at the time of surgery, which is a relatively uncommon feature of presumed congenital orbital varices.

Second, while his patients are not typical for orbital varices, the conclusion must be made that these cases do meet at least the broad definition of orbital varix

if one accepts that this definition is a vascular abnormality with a major venous component. Certainly the venous nature of these three lesions was confirmed at surgery. The etiology of the typical orbital varix is conjectural but the information we have suggests the presence of a congenital aberration in the development of the venous circulation of the orbit. Perhaps Doctor Bullock might comment on his opinion as to whether or not his patients represent acquired lesions of the orbit. More specifically, are the authors comfortable that these patients represent only isolated thrombosed orbital varices rather than a more complex intraorbital or intracranial processes such as low flow dural shunt, one manifestation of which can be venous thrombosis.

Thirdly, most orbital varices which we see are managed conservatively with surgical approaches being taken only when pain, cosmetic disfigurement, and/or a significant threat to vision dictate that something be done. Surgery for orbital varices can be high-risk procedures and the complications of such intervention can include ptosis, ophthalmoplegia, and loss of vision. These complications have been avoided in the present cases by careful and meticulous surgery. I know that Doctor Bullock agrees that we should not conclude from his presentation that the treatment for most orbital varices is surgical. On the other hand, his surgical approach in all three cases seemed appropriate because, in point of fact, all three lesions mimicked an orbital tumor so, obtaining a tissue diagnosis was in order. Perhaps he might comment on what clinical findings, now based on experience with these cases would indicate surgical intervention in the future when orbital varices are suspected.

Finally, we have observed two patients in our practice who presented with painful, superficial nodules palpable in the brow within the neurovascular bundle at the supraorbital notch. The preoperative diagnosis was neuroma. However, at surgery, the nodule was found to be thrombus formation within the supratrochlear vein in both cases. Perhaps Doctor Bullock would comment as to whether or not he has seen such cases and whether these two acquired lesions might well be similar to the deeper lesions he has identified within the orbit.

This was a fascinating paper. I appreciated very much having the opportunity to participate in the discussion.

DR THOMAS R. HEDGES, JR. We are in debt to Doctor Bullock for presenting these patients; this gives me a chance to present a patient I saw 18 years ago.

In October, 1971, I was asked to go to the University of Pennsylvania Hospital to see a 15-year-old girl, who, while playing in a school yard, suddenly developed pain in her right orbit, total visual loss and ophthalmoplegia with proptosis on the right. She had a significant allergic and migraine history and had a known venous anomaly of her upper lid. When I saw her she had proptosis of approximately 6 to 7 mm, total ophthalmoplegia and no light perception. A significant finding was ecchymosis of the lower lid on the affected side with no history of trauma. The right optic disc was choked with a large preretinal hemorrhage. This nerve became totally atrophic with no light perception.

Venography showed bilateral orbital varices and a large cone shaped orbital

hemorrhage on the right. This is obviously an example of spontaneous rupture of an orbital varix.

With the experiences we have had expressed toady I probably would have asked for exploration of the orbit to relieve the pressure on the nerve. Perhaps this case emphasizes some of the points of the discussion.

DR JOHN D. BULLOCK. I appreciate the comments of the discussants.

Yes, these seem to be acquired lesions; we believe that they are different from congenital varices. These patients showed no features of dural shunts. In answer to Doctor Waller's question about the brow lesion, I have had one patient who had a small "tumor" under the brow; it was excised and turned out to be a thrombosis of a vessel. I did not include that in my paper because these others are so much more striking in their presentation than the brow patient.