Aneurysms of the ophthalmic segment

A clinical and anatomical analysis

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The clinical, radiographic, and anatomical features in 80 patients with ophthalmic segment aneurysms were reviewed, and were categorized according to a presumed origin related to the ophthalmic (41 cases) or superior hypophyseal (39 cases) arteries. There was a marked female predominance (7:1) and high incidence of multiple aneurysms (45%). Clinical presentations included subarachnoid hemorrhage in 23 cases (29%) and visual deficits in 24 (30%); five patients exhibited both hemorrhage and visual loss. Twenty-eight aneurysms were incidentally identified.

Ophthalmic artery aneurysms arose from the internal carotid artery (ICA) just distal to the ophthalmic artery, pointed superiorly or superomedially, and (when large) deflected the carotid artery posteriorly and inferiorly, closing the siphon. Abnormalities relating to vision were not identified until the aneurysm realized giant proportions. The optic nerve was typically displaced superomedially, which restricted contralateral extension until late in the clinical course; unilateral nasal field loss was seen in 12 patients. Nine patients had bilateral ophthalmic artery aneurysms which were often clipped via a unilateral craniotomy.

Superior hypophyseal artery aneurysms arose just above the dural ring from the medial bend of the ICA, at the site of perforator origin to the superior aspect of the hypophysis, and had no direct association with the ophthalmic artery. The carotid artery was usually located lateral or superolateral relative to the aneurysm. These lesions could extend medially beneath the chiasm (suprasellar variant), producing a clinical and computerized tomography picture similar to a pituitary adenoma, or they could extend ventrally to burrow beneath the anterior clinoid process (paraclinoid variant).

Preoperative categorization of these lesions according to their likely branch of origin provides excellent correlation with visual deficits and operative findings, and has allowed the author to clip 52 of 54 lesions, with very low operative or visual morbidity.

KEY WORDS • aneurysm • ophthalmic artery • internal carotid artery • superior hypophyseal artery • anatomical study

Ophthalmic artery aneurysms have been defined as those arising from the medial or anteromedial wall of the internal carotid artery (ICA) in the segment between the ophthalmic artery and posterior communicating artery (PCoA). This segment, termed the “ophthalmic segment,” is the longest subarachnoid portion of the carotid artery, from which there is generally considered to be only one named branch, the ophthalmic artery. Many aneurysms from this segment, however, have no direct relationship to this named branch. As a result, these lesions are often described according to their relationship to adjacent anatomical landmarks, producing such varying terms as paraophthalmic, supraophthalmic, infraophthalmic, proximal ICA, global, paraclinoid, supraclinoid, subchiasmal, parachiasmal, suprachiasmal, dorsal, and ventral aneurysms.

In this report, the clinical, anatomical, and radiographic characteristics of 80 patients with aneurysms arising from the ophthalmic segment have been analyzed. Based upon this study, new terminology has been developed that returns these lesions to the more traditional nomenclature of association with an arterial branch, one that also helps to explain the various clinical and technical characteristics of aneurysms with different sizes and locations within the segment.

Anatomy of the Ophthalmic Segment

The ophthalmic segment begins as the ICA penetrates the dura beneath the anterior clinoid process to enter...
the subarachnoid space. There are two major arterial bends in this segment. The first occurs as the carotid artery turns sharply posteriorly after penetrating the dura to enter the subarachnoid space (Fig. 1a and b). The second is a gentler medial-to-lateral curve as the artery approaches its terminal bifurcation (Fig. 1c and d). After spanning a distance of approximately 1 to 1.5 cm, the segment ends at the origin of the PCoA.\textsuperscript{21} The ophthalmic segment is known to have several branches. The first, largest, and best known is the ophthalmic artery. This vessel usually arises from the dorsal or dorsomedial surface of the ICA just after the carotid artery enters the subarachnoid space.\textsuperscript{24,38,46} The ophthalmic artery typically originates immediately beneath the lateral aspect of the overlying optic nerve, and subsequently travels through the optic canal to reach the orbit. During surgery, the origin of this branch often cannot be visualized until the anterior clinoid is removed.

Several large perforating vessels also arise from the ophthalmic segment, the largest of which is the superior hypophyseal artery.\textsuperscript{8,49} These perforators may be quite large and supply the dura around the cavernous sinus,
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FIG. 2. Drawings defining the terminology relating to ophthalmic segment aneurysms. a: Ophthalmic artery (Oph Art) aneurysms (1) arise from the dorsal internal carotid artery (ICA) just distal to the Oph Art. Superior hypophyseal artery (Sup Hyp Art) lesions arise independent of the Oph Art, and include paraclinoid (2a) and suprasellar (2b) variants. b: The paraclinoid variant (A) arises from the inferomedial ICA surface, burrows down toward the carotid cave above the ring, and is deflected ventrally by the dura of the lateral sellar wall. PComArt = posterior communicating artery. c: The suprasellar variant (A) exceeds these ventral confines and expands directly into the suprasellar space below the optic chiasm.

Terminology

Most intracranial saccular aneurysms derive their name from an intimate hemodynamic association with the branch of a parent vessel (such as the PCoA or the anterior choroidal artery). As outlined by Rhoton, most saccular aneurysms arise from a curve of the parent vessel, in the angle between it and a significant arterial branch, and point in the direction that flow in the parent artery would have continued if the curve had not been present. To conform to these rules, ophthalmic segment aneurysms have been divided herein into two large categories, depending on the relationship of the aneurysm neck with the arterial branches within the segment (Fig. 2).

Aneurysms arising in clear relation to the ophthalmic artery are termed ophthalmic artery aneurysms (Fig. 3). These lesions arise from the ICA just distal to the origin of the ophthalmic artery, and initially project dorsally or dorsomedially from the carotid artery surface to the lateral half of the optic nerve. Those originating more distally within the segment invariably incorporate the perforating branches to the hypophysis, and are herein called superior hypophyseal artery aneurysms (Figs. 4 and 5). Superior hypophyseal artery aneurysms are further subdivided into paraclinoid and suprasellar variants, largely based on the direction of enlargement as limited by the size of the pocket medial to the carotid artery and the height of the lateral sellar wall. The paraclinoid variant (Figs. 2b and 4) projects primarily inferiorly or inferomedially toward and beneath the anterior clinoid process, while the suprasellar...
variant (Figs. 2c and 5) extends medially or superomedially above the diaphragma sellae into the suprasellar space. Rarely, the paraclinoid type may remain lateral as it enlarges, and may reach giant proportions without compression of the visual pathways. Because the space beneath this carotid segment is limited, however, most paraclinoid lesions will eventually project toward the suprasellar space as they enlarge, at which time they exhibit features of both the paraclinoid and suprasellar variants. For categorization purposes, aneurysms exhibiting both paraclinoid and suprasellar extensions are herein classified according to their largest component (generally suprasellar).

Ophthalmic segment aneurysms were also divided according to size, based on estimates of maximal diameter of their external surface. Those measuring 4 to 9 mm were considered small, those between 10 and 24 mm were considered large, and those 25 mm or greater were considered giant.  

Summary of Cases

Patient Population

Within the last 9 years, approximately 700 patients with intracranial aneurysms were treated at our institution. The series described here comprised 80 (11%) of these patients who were diagnosed as harboring at least one ICA aneurysm arising between the origins of the ophthalmic artery and the PCoA. The series included 41 patients with ophthalmic artery aneurysms and 39 with superior hypophyseal artery aneurysms. The average age at presentation was 54 years. There was a striking (86%) female predominance. Thirty-six (45%) of the 80 patients had additional aneurysms elsewhere.

Clinical Presentation

Fifty-two patients presented with specific symptoms directly attributable to their ophthalmic segment an-
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![Image](https://example.com/aneurysms.png)

**Fig. 4.** Superior hypophyseal artery aneurysms (paraclinoid variants). a: Lateral arteriogram of a small superior hypophyseal artery aneurysm (A). The lesion appears to arise ventrally, opposite the ophthalmic artery (Oph Art) origin, and points toward cavernous sinus. The aneurysm lies below the level of the anterior clinoid process, but is still within subarachnoid space. PComArt = posterior communicating artery. b: Lateral arteriogram of a large superior hypophyseal artery aneurysm (A). The lesion arises just above the dural ring (arrow) and projects ventromedially seemingly into the cavernous sinus. Note the opening of the carotid siphon.

Aneurysms, such as subarachnoid hemorrhage or visual changes. The remaining 28 patients included 20 with nonspecific symptoms (such as headache, incidental computerized tomography (CT) findings, or transient ischemic attacks (TIA's) in which the ophthalmic segment lesion may have had some relationship to the symptoms) and eight with symptoms (such as subarachnoid hemorrhage, SAH) related to another aneurysm, who in the course of their evaluation were found to have a second, incidental ophthalmic segment lesion.

**Subarachnoid Hemorrhage.** Twenty-eight patients experienced SAH from their ophthalmic segment aneurysm; 23 of these presented with hemorrhage only and the other five also had visual changes at the time of bleeding. Fourteen lesions were ophthalmic artery aneurysms including three small, six large, and five giant lesions. Fourteen superior hypophyseal artery aneurysms bled; nine of these were suprasellar variants (three small, two large, and four giant) and five were paraclinoid variants (three large and two giant). Overall, SAH developed from six small, 11 large, and 11 giant aneurysms within the segment; no small paraclinoid aneurysms caused SAH.

**Visual Loss.** Twenty-nine patients had visual symptoms that were clearly related to their ophthalmic segment aneurysm: 24 had only visual symptoms at the time of presentation and the other five were found to have visual deficits after presenting with SAH (see next section). Aneurysms smaller than 1 cm did not produce visual signs regardless of any association with bleeding. Six large aneurysms (four ophthalmic artery and two superior hypophyseal artery suprasellar variants) produced visual deficits, three of which presented with SAH. The remaining 23 patients exhibited visual deficits caused by giant aneurysms, including 16 ophthalmic artery lesions and seven superior hypophyseal artery lesions (suprasellar type). No aneurysm that remained purely paraclinoid presented with visual symptoms, regardless of size or association with SAH.

Visual field testing was initially performed at the bedside in a manner described by Trobe and Glaser; this was especially important in those patients presenting with SAH. With one eye closed, each patient was asked to visually fixate on the examiner's nose, while two quadrants of the visual fields of the open eye were simultaneously stimulated paracentrally with the examiner's fingers or red-colored objects. The patient was then asked to compare the clarity and color of the two objects without altering central fixation. In this manner, relative visual field loss was easily demonstrated which otherwise might have been overlooked if only one field had been examined at a time. Subsequent formal visual field measurements confirmed the accuracy of this testing method in almost all instances.

The types of visual field loss were usually quite predictive of the aneurysm origin (Figs. 6 and 7). Overall, 17 patients exhibited unilateral visual defects, all but two of whom harbored ophthalmic artery aneurysms. In 12 of these 17 cases, the visual field defect was primarily nasal, usually involving the inferior nasal fields first (Fig. 6 upper and center). In three others, a different pattern was seen: one patient had an inferior altitudinal defect, in one the remaining vision was restricted to light perception only in the affected eye, and one had a poorly recorded unilateral deficit. Two patients with a superior hypophyseal artery aneurysm exhibited a mild ipsilateral superior temporal field loss.
FIG. 5. Superior hypophyseal artery aneurysms (suprasellar variants).  

a: Arteriogram, anteroposterior view, of a small superior hypophyseal artery aneurysm (A) projecting medial above the diaphragma sellae in this patient with subarachnoid hemorrhage.  
b: Arteriogram of a giant superior hypophyseal artery aneurysm (A), lateral view. In this projection, the lesion resembles that in Fig. 4b, arising from the internal carotid artery (ICA) wall opposite to the ophthalmic artery origin (black arrow). The white arrow marks the paraclinoid (ventral) bulge where the aneurysm reaches the carotid ring ventromedially.  
c: Drawing of a giant superior hypophyseal artery aneurysm (A), lateral view. Note the aneurysm medial to the ICA (white arrow), which is displaced laterally toward the clinoid process (Clin P). The lesion expands into the suprasellar space and does not sharply angulate the optic nerve at the falciform ligament. The black arrow marks the ventral bulge where the aneurysm wall merges with the cavernous sinus dura and ring. PComArt = posterior communicating artery.  
d: Arteriogram of a giant superior hypophyseal artery aneurysm (A), anteroposterior view, showing extension of the lesion into the suprasellar space (same case as illustrated in b). The arrow marks the paraclinoid (ventral) bulge lateral to the sella, indicating a common origin with paraclinoid variants.  
e: Drawing of a giant superior hypophyseal artery aneurysm (A), dorsal view. Note the suprasellar extension beneath the chiasm, with the pituitary stalk (Pit Stalk) easily displaced and allowing extension across midline. The arrow marks the paraclinoid bulge lateral to the sella, which must be incorporated in a clip down to the level of the dural ring.  
f: Computerized tomography scan with contrast enhancement of the giant aneurysm illustrated in b and d. Note the intrasellar mass resembling a pituitary tumor. A = aneurysm.
Fig. 6. Visual field loss in patients with ophthalmic segment aneurysms. 

Upper: Inferior nasal quadrant defect in the left eye of a patient with a giant ophthalmic artery aneurysm. 

Center: Inferior and superior nasal visual field defect in the left eye of another patient secondary to a giant ophthalmic artery aneurysm. 

Lower: Severe ipsilateral nasal visual field defect on the left plus contralateral superior temporal field loss due to a giant ophthalmic artery aneurysm in a third patient.
Twelve patients had bilateral visual deficits. Of these patients, four had bitemporal and five had homonymous visual field defects; these defects were associated with superior hypophyseal artery aneurysms in seven cases. The remaining three patients, each with a giant ophthalmic artery aneurysm, exhibited ipsilateral blindness and a contralateral visual field loss.

Subarachnoid Hemorrhage and Visual Loss. Five patients had ophthalmic segment aneurysms that produced both SAH and visual loss. Two had hemorrhages from ophthalmic artery lesions directly into the optic nerve; both exhibited dense central and nasal field loss. Two other patients had diffuse SAH without focal extension through the optic nerve. The visual field loss was confined to the ipsilateral superior temporal quadrant in one patient (with a superior hypophyseal artery lesion) and to the nasal field in the other (with an ophthalmic artery lesion). The fifth patient presented with ipsilateral blindness and contralateral temporal field loss. She suffered a fatal SAH from a giant ophthalmic artery aneurysm shortly after admission, and operative or autopsy visualization of the optic nerves or chiasm was not possible. No patient with SAH from an ophthalmic segment aneurysm exhibited Terson's syndrome.54

Nonspecific Symptoms. Twenty patients had vague or “other” types of symptoms that might have been related to their ophthalmic segment lesion, including headaches alone (eight patients), tinnitus, hearing loss, and vertigo (four patients), seizures (two patients), and TIA's or bruits (six patients). Eight of these patients also had at least one other vascular lesion (an arteriovenous malformation, another aneurysm, or arteriosclerotic lesion) at the time of presentation, thus making it impossible to attribute all symptoms solely to the ophthalmic segment aneurysm. Eight patients had symptomatic aneurysms at other sites and, during their evaluation, were also found to have at least one aneurysm within the ophthalmic segment.

Aneurysm Multiplicity. Thirty-six (45%) of the 80 patients with one ophthalmic segment lesion also had at least one other intracranial aneurysm. Of the 52 patients who presented with symptoms specific for their ophthalmic segment aneurysm (SAH or visual deficits), 21 (40%) harbored at least one other aneurysm elsewhere. The most common locations of the other aneurysms included the contralateral ophthalmic segment (six cases), the cavernous sinus (five cases), and the PCoA (three cases). Fourteen of 28 patients presenting with nonspecific symptoms had one or more aneurysms in addition to the ophthalmic segment lesion; these included the eight patients presenting with symptoms referable to another aneurysm. The most common second aneurysm location in these circumstances was the PCoA (six cases) and the ophthalmic segment (two cases).

Radiology. Hemorrhage from ophthalmic segment aneurysms usually appeared in the subarachnoid space within the anterior chiasmatic cisterns. Of the patients whose original CT scans were available for analysis, nine had hemorrhage extending beyond the subarachnoid space, including four with bleeding into the orbitofrontal gyri, three with subfrontal and intraventricular hemorrhage, and two with pure intraventricular bleeds. In two patients with subfrontal bleeding the aneurysm had ruptured through the ipsilateral optic nerve. Although many of the patients were quite ill from their SAH, only one presented with an intraparenchymal clot large enough to be life-threatening.

Small aneurysms were not well visualized on CT or magnetic resonance (MR) imaging. In patients presenting with visual loss, however, the aneurysm was invariably defined by one of these modalities. The lesion often appeared much larger on CT than the angiographically apparent lumen size, indicating a significant incidence of partial luminal thrombosis. Calcifications were common in giant lesions.

Ophthalmic Artery Aneurysms. Arteriographically, ophthalmic artery aneurysms arose from the dorsal or dorsomedial surface of the proximal ophthalmic segment, just distal to the ophthalmic artery origin (Fig. 3). With expansion, the aneurysm elevated the inferior and lateral aspects of the optic nerve, creating a notch or flattened area on its anterior medial surface which can often be seen on angiography (Fig. 3b).23

Fig. 7. Drawing of ophthalmic segment aneurysms affecting visual system, dorsal view. The left side shows the effect of an ophthalmic artery aneurysm. The lateral portion of the optic nerve is elevated and displaced medially, leading to a monocular nasal field defect (pathway marked by double arrows). The initial defect is often first noted inferiorly, due to pressure on the superior optic nerve surface by the sharp edge of the falciiform fold (black arrow) (see Fig. 6 upper and center). With further enlargement, the anterior knee of Wildbrand (white arrow) is compressed, leading to a contralateral superior temporal defect (junctional hemianopsia, see Fig. 6 lower). The right side is affected by a superior hypophyseal artery aneurysm. The lesion expands beneath the chiasma, beyond the optic nerve-falciiform fold junction, producing visual field defects similar to those associated with a pituitary tumor.

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On CT and MR imaging, ophthalmic artery aneurysms were usually unilateral, with the optic nerve serving as an effective tether to prevent significant expansion across the midline until late in the clinical course (Fig. 3c). Magnetic resonance imaging often delineated the position of the optic nerve, and was useful in confirming the compression primarily affecting the nerve rather than the chiasm. As the aneurysm was limited in its superior growth by the optic nerve, the carotid artery was often deflected inferiorly, with “closing” of the carotid siphon (Fig. 3b).

Superior Hypophyseal Artery Aneurysms. Arteriographically, small superior hypophyseal artery aneurysms were invariably noted on the inferior or inferior medial surface of the ICA just opposite and slightly distal to the ophthalmic artery origin, and there was often some question on initial interpretation as to whether the aneurysm was actually within the subarachnoid space (Figs. 4a and 5a). The paraclinoid variant remained lateral to the sella, burrowing beneath and medial to the ICA under the anterior clinoid process (Figs. 2b and 4). As the aneurysm enlarged, the ICA was often deflected laterally and superiorly, thereby “opening” the siphon.

When suprasellar expansion occurred (apparently dependent on the position of the aneurysm origin relative to the lateral sellar wall), the aneurysm enlarged without significant restrictions beneath the chiasm (Fig. 5). Many large and giant superior hypophyseal artery aneurysms had two “scalloped” basal surfaces, one in the paraclinoid region where the aneurysm originated, and the second more medially as the aneurysm extended over the diaphragma sellae into the suprasellar space (Fig. 5b to e), thus supporting the common origin of the two variants.

When superior hypophyseal artery aneurysms expanded into the suprasellar space (suprasellar variant), they often created a CT picture resembling a pituitary tumor (Fig. 5f). In contrast to ophthalmic artery aneurysms, these lesions extended medially beneath the chiasm rather than the optic nerve, thereby allowing bilateral visual changes, especially when reaching giant proportions. The pituitary stalk, offering little resistance to the contralateral expansion of the aneurysm, was markedly deflected to the other side (Fig. 5e).

Operative Results

Fifty-four patients in this series underwent surgery by the author, with the results summarized in Table 1. The others either received surgery by other physicians within our department or did not undergo a surgical procedure.

General Outcome. Overall, 47 patients (87%) had excellent results (no deficits) or good outcomes (minimal dysfunction compatible with normal life activities). Four patients (7%) had poor outcomes from ischemic injury, one directly from the operative procedure (see Complications section) and three from the consequences of vasospasm. Three patients died (6%), none apparently directly related to the intracranial operative procedure (see below). Of the seven cases with major morbidity or mortality, four were directly attributable to ischemia from vasospasm. All patients presenting with nonspecific symptoms had excellent results.

Visual Outcome. In 23 of the author’s patients visual deficits were recognized preoperatively. Four of these patients came to clinical attention after SAH. Two patients with large ophthalmic artery aneurysms had hemorraghes into and through ipsilateral optic nerve. Both had marked ipsilateral loss of visual acuity and nasal field deficits, and both improved after surgery. The two other patients had compressive visual deficits unrelated to direct intraneural injury, and both improved after aneurysm clipping. The remaining 19 patients presented with visual loss as their only neurological complaint. In all but two of these, the lesions were giant aneurysms. The visual deficits were improved following the procedure, often dramatically, in 13 patients and were unchanged in six. When present preoperatively, a dense inferior nasal defect often persisted.

Overall, 17 of 23 patients who presented with visual loss had improvement following clipping and decompression of their aneurysm. However, three others who did not have documented visual deficits preoperatively had diminished acuity and/or field loss postoperatively (see Complications section).

TABLE 1

Operative results in 54 patients with ophthalmic segment aneurysms

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<tr>
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* This series includes the author’s cases only. SDH = subdural hematoma; TIA = transient ischemic attack; ICAO = internal carotid artery occlusion.

† Includes only patients with preoperatively recognized visual deficits.

‡ Patients without recognized preoperative visual deficits.
Complications

Deaths. There were three postoperative deaths. The first occurred in a patient with a giant calcified superior hypophyseal artery aneurysm and severe, rapidly progressive visual loss. At surgery, the aneurysm was deemed unclippable, and she awakened without new deficits. She later underwent a cervical carotid artery clamping procedure under systemic heparin to minimize thromboembolic complications. She developed an unrecognized posterior fossa subdural hematoma and died from tonsillar herniation and brain-stem compression.

A second patient did quite well for 6 days after clipping of a ruptured giant superior hypophyseal artery aneurysm, but then died suddenly from an autopsy-proven massive pulmonary embolus. The third patient died of florid, unrelenting vasospasm that was refractory to volume expansion and hypertensive therapy. Arteriography and subsequent autopsy documented excellent clip placement and severe generalized ischemic damage.

Ischemia. Seven patients developed cerebral ischemia after surgery for an ophthalmic segment aneurysm. In four patients ischemia was due to vasospasm following SAH; one died (see above) and the other three exhibited mild to moderate deficits as long-term sequelae (poor outcomes).

In the other three instances, the postoperative ischemia was clearly related to the surgical procedure. One, a 65-year-old woman with progressive bilateral blindness from a 4.5-cm ophthalmic artery aneurysm, suffered a permanent deficit. She had undergone a cervical carotid ligation 15 years previously. At surgery, she developed bleeding from the base of the aneurysm, requiring prolonged temporary clipping of the intracranial ICA. She subsequently sustained an aphasia secondary to parietal lobe infarction. This case was treated very early in the series (the anterior clinoid process was not removed), and emphasized strongly the need for proximal exposure before aneurysm manipulation. The two remaining patients, both with giant calcified aneurysms producing visual loss, experienced transient contralateral weakness postoperatively, which resolved within 24 hours. A hemostat was used to crack the eggshell-like coverings of the aneurysm so as to allow closure of the clip. In both situations, the carotid artery thrombosed early after surgery, presumably secondary to narrowing by the clip or by incorporation of atheroma into the vessel lumen.

Visual Sequelae. Six patients suffered deterioration of their visual status after surgery. In three instances, the visual fields and/or acuity were decreased. Two of these patients did not report any preoperative visual deficit and were not tested by the bedside method described above. Both had large ophthalmic artery aneurysms which significantly distorted the ipsilateral optic nerve medially and superiorly. The falciform ligament was not cut in either case prior to optic nerve manipulation. Postoperatively, both patients had a residual ipsilateral inferior nasal quadrant defect. The third patient, also without preoperative visual signs, had a giant aneurysm arising from the opthalmic artery. After surgery, she had marked visual acuity diminution (20/100), presumably due to excessive optic nerve or perforator manipulation.

Three other patients had transient sixth nerve palsies which resolved within a few weeks. The cranial nerves within the cavernous sinus were presumably disturbed either by clinoid removal or by the clip blades as they were advanced proximally beyond the aneurysm neck.

Postoperative Infection. Two patients developed postoperative infections. One of these had a delayed epidural abscess associated with frontal sinusitis, and the other exhibited a meningitis-like syndrome (with negative cultures) 4 weeks after surgery. Both infections resolved without sequelae after appropriate therapy.

"Unclippable" Lesions. Two lesions were judged unclippable because of marked calcification within their walls. One was in a woman with headaches only who had a large paraclinoid aneurysm burrowing beneath the anterior clinoid process. Because of extensive atherosclerosis and calcification of the walls of both the aneurysm and the adjacent ICA, it was deemed safer to wrap this lesion rather than clip it. The patient awoke from surgery without deficit.

The aneurysm in the second patient (mentioned above) could probably have been clipped had the case been treated later in the author's experience, when the lesion would have been managed with a combination of barbiturate anesthesia, temporary ICA clipping, and intra-aneurysmal calcification removal. Due to the previously mentioned unpredictability of using a hemostat in creating a surgical neck in these calcified lesions, this maneuver is now avoided if possible. Instead, the aneurysm is temporarily trapped, and the laminated calcific walls are removed through an incision into the interior of the aneurysm. The resultant neck is much more pliable and accepting of the clip, with less risks of parent vessel compromise by fractured or displaced calcification or atheroma.

Discussion

The level of the anterior clinoid process (as defined either by lateral x-ray or direct operative visualization) has traditionally been used to label aneurysms as supraclinoid (within the subarachnoid space, and therefore "clippable") or infraclinoid (within the cavernous sinus, and "unclippable"). While the need for clinoid removal may be implied in such terms, "clippability" or liability to rupture and to SAH should not be aligned with this anatomical landmark. Instead, these features are more appropriately associated with the aneurysm's relationship to the dural carotid "ring."

Differential Diagnosis

Generally, aneurysms that originate near the anterior clinoid process do not violate the dural ring. Those
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arising below the ring are extradural in location, and originate within either the cavernous sinus itself or the clinoid space (carotid cave). Small anterior intracavernous or clinoid space lesions may be quite difficult to differentiate from superior hypophyseal artery aneurysms. As suggested by Kobayashi, et al., some of these lesions represent cases in which a superior hypophyseal artery arose from the clinoid rather than the ophthalmic segment of the carotid artery. A similar situation is known to occur with the ophthalmic artery origin. 8,9,38,40.

Most larger intracavernous or clinoid segment aneurysms are readily diagnosed by radiographic or clinical findings, and their association with an arterial branch site, if initially present, is obscured by the lesion's mass. As these lesions enlarge, they will be capped by dura extending from the ring (formerly considered the roof of the cavernous sinus), and thus will have a double-layered reinforcement (aneurysm wall plus dura) that reduces the risks of hemorrhage and limits significant suprasellar extension. When these lesions do erode upward through the dura to reach the subarachnoid space, they do so adjacent to the ring, not through it, and will have a thin "collar" of dura around the base of the subarachnoid projection.

Direct intervention into the cavernous sinus or clinoid space requires greater anatomical mastery before surgery in this area can become routinely successful. Proximal exposure is much more difficult to obtain, as the ring does not define the aneurysm's proximal extent. Clindoid aneurysms probably account for some lesions formerly termed "ophthalmic aneurysms" which at surgery were considered "unclippable" or catastrophically ruptured. Due to their lower risks of hemorrhage, many of these lesion types are perhaps best managed by continued clinical observation or by carotid ligation and bypass considerations.

Extensive removal of the clinoid processes of the ICA are foreshortened or when the anterior clinoid process is elongated, a PCoA aneurysm may be mistaken for an ophthalmic segment lesion. However, PCoA aneurysms arise well distal to the dural ring, project posteriorly, posterior-laterally, or laterally, and often point slightly downward. Oculomotor nerve compression is frequently encountered, a clinical finding that is rarely, if ever, seen with ophthalmic segment lesions.

Operative Technique

Since the large majority of clinoid region aneurysms, especially those with "supraclinoid" projection, arise from the ophthalmic segment above the dural ring, these lesions should receive the greatest attention. Although most subarachnoid aneurysms arise from the ophthalmic segment of the carotid artery, some of these lesions represent cases in which a superior hypophyseal artery arose from the clinoid rather than the ophthalmic segment of the carotid artery. A similar situation is known to occur with the ophthalmic artery origin. 8,9,38,40.

Most larger intracavernous or clinoid segment aneurysms are readily diagnosed by radiographic or clinical findings, and their association with an arterial branch site, if initially present, is obscured by the lesion’s mass. As these lesions enlarge, they will be capped by dura extending from the ring (formerly considered the roof of the cavernous sinus), and thus will have a double-layered reinforcement (aneurysm wall plus dura) that reduces the risks of hemorrhage and limits significant suprasellar extension. When these lesions do erode upward through the dura to reach the subarachnoid space, they do so adjacent to the ring, not through it, and will have a thin “collar” of dura around the base of the subarachnoid projection.

Proximal visualization of the carotid and ophthalmic arteries remains mandatory for both types of aneurysms (ophthalmic artery and superior hypophyseal artery), and is facilitated by removal of the anterior clinoid process and optic canal roof and lateral wall. Initially, only the clinoid process itself was removed, and this was undertaken from an intradural approach. Currently, the posterior roof of the orbit, the lesser wing of the sphenoid bone covering the superior and medial surfaces of the superior orbital fissure, and the base of the clinoid process are all removed extradurally. In unruptured aneurysms, the anterior clinoid process can be removed extradurally but, with ruptured aneurysms or whenever fusion between the anterior and posterior clinoid processes is shown on CT scans, the clinoid tip is removed intradurally after direct visualization of the lesion.

Extensive removal of the clinoid process exposes the clinoid space (carotid cave), a pocket of variable size (usually approximately 5 mm) that houses a segment of the carotid artery (clinoid segment) below the dural ring but outside the main venous channels of the cavernous sinus (Fig. 1). Sectioning the carotid ring circumferentially allows excellent exposure of this ICA segment, thus providing enough proximal exposure to allow temporary carotid clipping if required. Bleeding from this region is quite easily controlled with bone wax, Gelfoam, or Surgicel. Although the carotid artery was exposed proximally near the cavernous sinus or neck in most instances, the use of temporary ICA clipping was uncommon. If anticipated, however, barbiturate protection was initiated by bolus injection and maintained throughout the duration of vessel compromise. Somatosensory evoked potential recordings were also utilized to monitor cerebral function and perfusion, and were quite useful in several instances in predicting carotid artery compromise from improper clip placement.

Optic nerve displacement, if not already done by the aneurysm, was often necessary to visualize the proximal neck. The falciiform ligament was sectioned before any aneurysm dissection was undertaken in all later cases. This structure forms a knife-like edge against the super-
FIG. 8. Drawings showing intraoperative views of an ophthalmic artery (Oph Art) aneurysm (pterional approach). A: The anterior clinoid process (Clin) has been removed and the falciiform fold sectioned to allow visualization of the Oph Art-aneurysm (A) junction and provide proximal intracranial control of the internal carotid artery (ICA) should temporary clipping be necessary. ON = optic nerve. B: A side-angled clip is placed parallel to the long axis of the ICA. The aneurysm is then aspirated, and the carotid artery inspected for patency.

rior aspect of the optic nerve, and mobilization of the nerve against it may further increase visual morbidity, especially in ophthalmic aneurysms that already exhibit clinical visual compression at this site.

The typical surgical view (from a pterional approach) for an ophthalmic artery aneurysm is shown in Fig. 8. Longer straight or side-angled clips would satisfactorily obliterate most ophthalmic artery lesions while sparing the ophthalmic artery. Broad necks are commonplace in larger lesions, and are best secured by placing the clip parallel to the parent (ICA) vessel. Clips placed more perpendicular are often ineffective in collapsing the aneurysm, and the clip jaws would occasionally open and be propelled backward with each arterial pulsation. On several occasions, fenestrated clips incorporating the optic nerve were utilized.

From a pterional view, small superior hypophyseal artery aneurysms might not initially be visible to the surgeon, being hidden by the overlying ICA and anterior clinoid process. Superior hypophyseal artery lesions are usually easily obliterated with a fenestrated clip, the blades of which pass over and then run parallel to the ICA, spanning the distance between the PCoA and the dural ring (Fig. 9). As with ophthalmic artery aneurysms, a second clip was often useful to control the aneurysm. After clipping, the aneurysm must be collapsed and the clips adjusted accordingly to ensure carotid patency. Sparing of medial perforators (the superior hypophyseal arteries) should be attempted in all cases but is not always possible. The PCoA, displaced posteriorly across the back wall of the aneurysm, must be seen and preserved.

Although no perforators off the ophthalmic segment supply brain parenchyma, some (the superior hypophyseal arteries) reach the optic chiasm, and every attempt should be made to spare them from the surgical clip. Because the pituitary stalk receives blood supply from both sides, endocrine deficits secondary to unilateral interruption of these branches are rarely noticed. No patient in our series has developed hypopituitarism, but several had visual loss that was unimproved or somewhat worsened following surgery; these deficits were perhaps secondary to blood supply alterations to the optic nerve or chiasm.

The female predominance and incidence of bilaterality and multiplicity in patients harboring aneurysms involving the ophthalmic segment is quite high (Table 1). Because of their superior or medial projection, small ophthalmic segment aneurysms can often be clipped from a contralateral approach between or behind the optic nerves. In six patients in this series a small asymptomatic contralateral ophthalmic artery aneurysm was clipped during a procedure to secure another lesion elsewhere. This capability may be quite important when deciding which side to treat first in a patient harboring bilateral lesions, one of which arises from the ophthalmic segment. In general, the craniotomy should be carried out on the side of the
Aneurysms of the ophthalmic segment

![Diagram showing intraoperative views of a superior hypophyseal artery aneurysm (pterional approach). A: The anterior clinoid process has been removed to expose the ventral-medial surface of the internal carotid artery (ICA) to the level of the dural ring. A fenestrated clip passes over the broadened ICA wall. A = aneurysm; ON = optic nerve. B: The clip is carefully placed parallel to the ICA, with the fenestration reconstructing the carotid lumen. The butt of the clip must spare the posterior communicating artery (PComArt), while the tips of the clip are advanced to the ventral border of the ring. The aneurysm is then aspirated and the carotid artery inspected for patency.]

symptomatic aneurysm. The surgeon may then choose to explore the opposite carotid artery, with plans to obliterate the contralateral lesion if feasible. Superior hypophyseal artery aneurysms are often more difficult to visualize from a contralateral approach than ophthalmic artery lesions. The author does not advise clipping large or giant lesions from the contralateral side except in emergency situations.

**Risks of Rupture**

Many ophthalmic segment lesions have a “double” protective layer that may reduce liability to hemorrhage. As an ophthalmic artery aneurysm expands upward and medially, its fundus encounters and eventually is capped by the optic nerve, which may explain the higher frequency of lesions reaching large or giant proportions without bleeding.\(^{15,16,22,23}\) Similarly, superior hypophyseal artery aneurysms initially expand medially or inferomedially toward the dura of the lateral sellar wall and cavernous sinus roof, with the fundus somewhat protected in a way similar to those arising below the dural ring.\(^{28}\) This double insulation provides some rationale for the selection of operation for unruptured aneurysms in this area. Those that originate below the dural ring or remain purely paraclinoid appear to have a lower hemorrhage rate when small (no ruptures in this series). Large lesions, or those with medial suprasellar extension, appear to bleed with higher frequency.

**Conclusions**

Ophthalmic artery aneurysms arise from the superomedial surface of the ICA just distal to the ophthalmic artery, and point upward. The optic nerve is deflected superiorly and medially, leading to angulation against the sharp edge of the falciform ligament. An inferior nasal visual field defect may initially go unnoticed by the patient because the corresponding fields in the opposite eye are unaffected. Eventually, acuity and the entire nasal field of the ipsilateral eye will be affected, followed by superior temporal field loss in the contralateral eye secondary to compression of the anterior knee of Wildbrand.\(^{32}\) The stretched optic nerve inhibits extension across the midline until late in the clinical course. By that time, severe ipsilateral visual loss or blindness has occurred.

Loss of vision in this pattern is so often produced by ophthalmic artery aneurysms (classified as described herein) that it is often possible to diagnose the exact type of supraclinoid aneurysm at the bedside prior to reviewing the radiographic studies. The pattern of visual loss reported here provides some support for Jefferson’s earlier conclusions about supraclinoid aneurysms compressing the visual pathways: that monocular or junctional hemianopsia are the most common types of visual field loss produced by these lesions.\(^{15,16,28,29}\)

In contrast, superior hypophyseal artery aneurysms project medially and inferiorly, just above the carotid ring, at the site of the perforators supplying the pituitary...
stalk, surrounding dura, and optic chiasm. As they enlarge, their walls become adherent to the dura of the sella, diaphragma sellae, and lateral cavernous sinus wall. Although the arteriogram may suggest otherwise, these lesions rarely if ever project into the cavernous sinus, and the walls of the two structures can be separated.\textsuperscript{11,39,40} In large and giant varieties, the ICA is displaced slightly laterally and superiorly toward the surgeon, and the neck of the lesion is often so wide and long that the entire carotid wall appears to be incorporated (Fig. 9).

When suprasellar extension occurs, the entire optic apparatus is elevated, with the accompanying pressure directed more toward the chiasm rather than the optic nerve. Sharp angulation against the falx cerebri is not typically seen with this aneurysm variety, and visual field deficits are more closely akin to those associated with pituitary tumors.\textsuperscript{45,46} The hypophyseal stalk is usually adherent to the posterior and medial surface of the enlarging lesion, and is not a significant impediment to extension across the midline.

Almost all ophthalmic segment aneurysms are clip-\textsuperscript{able}, but larger lesions often require exposure of the proximal carotid artery within the clinoid space for accurate clip placement. The labeling of these lesions as “unclip\textsuperscript{able}” should be withheld until clinical removal and exploration has been performed by a surgeon familiar with the anatomy of the dural ring and paraclinoid area. Clipping and aneurysm aspiration allow immediate and effective decompression of the visual system and eliminate rebleeding, while preserving carotid artery blood flow to the hemisphere.\textsuperscript{16,23,28} Carotid artery ligation or balloon embolization may be useful in selected instances, when surgery has been unsuccessful in securing the lesion.\textsuperscript{17,20,26}

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