Surgical alternatives in the treatment of cavernous sinus aneurysms

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Direct surgery on aneurysms in the cavernous sinus is a formidable technical procedure. The intimate relationship of the intracavernous carotid artery to the venous structures and to the cranial nerves makes surgical access difficult at best. Thirty-two of 356 aneurysm patients presented with symptomatic aneurysms originating from the intracavernous internal carotid artery. Twenty-one patients had aneurysms contained entirely within the cavernous sinus, and in 11 others the aneurysms arose within the cavernous sinus and extended into the subarachnoid space. Of the purely intracavernous aneurysms there were five small aneurysms (< 25 mm) and 16 giant (≥ 25 mm) aneurysms. Fifteen patients with purely intracavernous lesions had superior orbital fissure syndrome, and six had a variety of other symptoms. Of 11 patients with subarachnoid extension, five had a subarachnoid hemorrhage (Grade I or II), five had ipsilateral visual loss, and one had periorbital pain. The aneurysms were treated as follows: Group 1 received progressive ligation of the internal carotid artery in the neck with a Selverstone clamp and a superficial temporal artery-middle cerebral artery (STA-MCA) anastomosis (purely intracavernous in nine, and with subarachnoid extension in one); Group 2 underwent trapping of the internal carotid artery and a deep STA-MCA anastomosis (purely intracavernous in seven); and Group 3 had direct clipping of the aneurysm (purely intracavernous in five, and with subarachnoid extension in 10).

The cavernous sinus was entered directly through its roof by a pterional craniotomy with radical removal of the optic canal, lesser sphenoid wing, and lateral and superior orbital walls. Proximal control of the internal carotid artery was obtained through a cervical incision. Two patients in Group 1 developed transient neurological deficits, which resolved. Two patients in Group 2 developed a cerebral infarction, one of whom died; in both of these patients, the anastomosis was completed after the internal carotid artery occlusion. Two patients in Group 3 progressed from marked visual loss to blindness of the same side, and one developed an intraventricular hemorrhage during induction of anesthesia and died without surgery. It is proposed that a direct approach to symptomatic aneurysms in the cavernous sinus is the best initial alternative. When this approach is not feasible, a trapping procedure preceded by a high-flow extracranial-intracranial anastomosis may be considered. Although the authors have been able to clip aneurysms of various sizes, this has not been possible in all patients. Further work is needed in this area.

KEY WORDS • cavernous sinus • aneurysm • giant aneurysm • clipping • anastomosis, vascular

Neurysms in the cavernous sinus region produce mechanical changes that affect the cranial nerves contained within the confines of the venous sinus, causing periorbital pain, ptosis, and paresis of the third, fourth, and sometimes the sixth cranial nerves, and producing a superior orbital fissure syndrome. The aneurysms frequently exceed 25 mm in diameter, and are therefore classified as giant aneurysms. Because of the intimate relationship in the cavernous sinus of the internal carotid artery (ICA) to the third through sixth cranial nerves and because of potential bleeding from the cavernous sinus itself, direct clipping of these aneurysms has been considered undesirable. Parkinson reported a direct approach to the sinus with the aid of cardiac arrest, and Dolenc described an approach to cavernous sinus aneurysms through an extradural route.

In this study, 32 patients with aneurysms arising from the intracavernous ICA and treated through an intra-
Surgical treatment of cavernous sinus aneurysms

TABLE 1
Clinical course of 10 patients treated with surface STA-MCA anastomosis and Selverstone clamp

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>History</th>
<th>Preoperative Findings</th>
<th>Postoperative Findings</th>
<th>Follow-Up Period (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>CT Scan</td>
<td>Aneurysm Site</td>
<td>Size (mm)</td>
</tr>
<tr>
<td>1</td>
<td>F, 68</td>
<td>retro-ocular pain, od rt eye; paresis of rt III, IV, V, VI</td>
<td>temporal mass</td>
<td>cavernous</td>
<td>25</td>
</tr>
<tr>
<td>2</td>
<td>F, 47</td>
<td>retro-ocular pain, od rt eye; diplopia; paresis rt UE</td>
<td>temporal mass</td>
<td>cavernous</td>
<td>35</td>
</tr>
<tr>
<td>3</td>
<td>M, 65</td>
<td>headache; paresis of rt II, IV, VI</td>
<td>temporal mass</td>
<td>cavernous</td>
<td>39</td>
</tr>
<tr>
<td>4</td>
<td>M, 23</td>
<td>diplopia; paresis of rt VI</td>
<td>temporal mass</td>
<td>cavernous</td>
<td>40</td>
</tr>
<tr>
<td>5</td>
<td>M, 60</td>
<td>diplopia; paresis of rt II, IV, V</td>
<td>temporal mass</td>
<td>cavernous</td>
<td>30</td>
</tr>
<tr>
<td>6</td>
<td>F, 74</td>
<td>rt orbital pain; diplopia; paresis of rt III, IV, VI</td>
<td>parasealar mass</td>
<td>cavernous</td>
<td>40</td>
</tr>
<tr>
<td>7</td>
<td>F, 56</td>
<td>rt orbital pain; diplopia; paresis of rt VI</td>
<td>temporal mass</td>
<td>cavernous</td>
<td>35</td>
</tr>
<tr>
<td>8</td>
<td>M, 79</td>
<td>lt frontal pain; syncope; diplopia; paresis of lt III, V</td>
<td>temporal mass</td>
<td>cavernous</td>
<td>35</td>
</tr>
<tr>
<td>9</td>
<td>F, 58</td>
<td>lt orbital pain; SAH</td>
<td>temporal mass</td>
<td>parasealar</td>
<td>25</td>
</tr>
<tr>
<td>10</td>
<td>F, 63</td>
<td>lt face numbness; diplopia; paresis of lt III, VI</td>
<td>temporal mass</td>
<td>cavernous</td>
<td>25</td>
</tr>
</tbody>
</table>

* Roman numerals denote cranial nerves (CN's); superficial temporal artery-middle cerebral artery (STA-MCA); CT = computed tomography; UE = upper extremity; TIA = transient ischemic attack; SIADH = syndrome of inappropriate anti-diuretic hormone release; PRIND = prolonged reversible ischemic neurological deficit; SAH = subarachnoid hemorrhage.
† Cavernous = intracavernous location; mixed = intracavernous location with subarachnoid extension.

Summary of Cases
Clinical Material

Of 356 aneurysm patients treated at Henry Ford Hospital from October, 1978, through June, 1987, 32 presented with aneurysms arising entirely from the intracavernous ICA. Among these 32 patients, the aneurysms were contained entirely within the cavernous sinus in 21 cases; in the remaining 11 cases the aneurysms arose entirely from the carotid artery in the cavernous sinus and extended into the subarachnoid space. Of the aneurysms contained purely within the cavernous sinus, five were small (< 25 mm) and 16 were giant in size (≥ 25 mm). Fifteen patients with an aneurysm contained entirely within the cavernous sinus presented with a superior orbital fissure syndrome; two had ischemic symptoms, two had severe orbital pain, one had ipsilateral visual loss, and one had a subarachnoid hemorrhage (SAH). Of the 11 patients with cavernous aneurysms extending into the subarachnoid space, five presented with SAH (Grade I or II), five had ipsilateral visual loss, and one had severe ipsilateral orbital pain. Patients with incidentally found asymptomatic aneurysms arising from the intracavernous ICA did not undergo surgery. All operations were performed under barbiturate anesthesia through a pterional approach. Preoperatively, a lumbar subarachnoid drain was inserted and intravenous furosemide (40 mg) and mannitol (1 gm/kg) were given to facilitate brain retraction.34 Proximal arterial control was first obtained in every patient by placing a Selverstone clamp on the ICA distal to the common carotid artery bifurcation in the neck. This clamp may be used to obtain partial or total arterial occlusion during aneurysm dissection.

Surgical Procedures

Patients with symptomatic intracavernous aneurysms were treated in the following manner. In 10 patients (Group I, Table 1), a Selverstone clamp was placed at the origin of the ICA in the neck and closed to decrease the carotid artery flow to 10% of its initial value.32,33 At the same operation, a superficial temporal artery (STA)-to-middle cerebral artery (MCA) anastomosis was completed on the cortical surface.8 The ICA was then progressively occluded during the first 7 days after surgery using the Selverstone clamp. Nine patients in Group 1 had purely intracavernous aneurysms, and one had a cavernous aneurysm with subarachnoid extension.

The ICA was then progressively occluded during the first 7 days after surgery using the Selverstone clamp. Nine patients in Group 1 had purely intracavernous aneurysms, and one had a cavernous aneurysm with subarachnoid extension. In seven patients (Group 2, Table 2), an artery-to-artery anastomosis of the largest branch of the STA to a proximal trunk of the MCA, just distal to the bifurcation, was performed initially.9 This was followed immediately by trapping of the ICA, performed by ligating the ICA in the neck and clipping it intracrani-
### TABLE 2
Clinical course of seven patients treated with trapping and proximal EC-IC anastomosis*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>History</th>
<th>CT Scan</th>
<th>Aneurysm Size (mm)</th>
<th>Site of Aneurysm†</th>
<th>Karnofsky Score</th>
<th>Outcome</th>
<th>Karnofsky Score</th>
<th>Complications</th>
<th>Follow-Up Period (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F, 82 rt orbital pain; diplopia; paresis of rt III, IV, V, VI</td>
<td>temporal mass parasellar mass</td>
<td>30</td>
<td>cavernous</td>
<td>90</td>
<td>lt hemiplegia</td>
<td>50</td>
<td>cerebral infarction</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>F, 47 headaches; diplopia; paresis of rt III, IV, VI</td>
<td>temporal mass parasellar mass</td>
<td>30</td>
<td>cavernous</td>
<td>80</td>
<td>asymptomatic</td>
<td>90</td>
<td>none</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>F, 77 diplopia; paresis of rt III, IV, VI</td>
<td>temporal mass parasellar mass</td>
<td>40</td>
<td>cavernous</td>
<td>90</td>
<td>confusion</td>
<td>70</td>
<td>none</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>F, 48 diplopia; visual loss lt eye</td>
<td>temporal mass parasellar mass</td>
<td>25</td>
<td>cavernous</td>
<td>90</td>
<td>asymptomatic</td>
<td>90</td>
<td>none</td>
<td>96</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>F, 56 rt orbital pain; diplopia; paresis of rt VI</td>
<td>temporal mass parasellar mass</td>
<td>35</td>
<td>cavernous</td>
<td>90</td>
<td>asymptomatic</td>
<td>90</td>
<td>none</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>F, 64 dysphasia; paresis of rt UE</td>
<td>temporal mass parasellar mass</td>
<td>25</td>
<td>cavernous</td>
<td>80</td>
<td>asymptomatic</td>
<td>90</td>
<td>none</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>F, 58 diplopia; paresis of rt III; SAH</td>
<td>temporal mass parasellar mass</td>
<td>70</td>
<td>cavernous</td>
<td>80</td>
<td>died</td>
<td>0</td>
<td>cerebral infarction</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

* Roman numerals denote cranial nerves; EC-IC = extracranial-intracranial; CT = computerized tomography; UE = upper extremity; SAH = subarachnoid hemorrhage.

† Cavernous = intracavernous location.

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### TABLE 3
Clinical course of 15 patients treated with direct clipping*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>History</th>
<th>CT Scan</th>
<th>Aneurysm Size (mm)</th>
<th>Site of Aneurysm†</th>
<th>Karnofsky Score</th>
<th>Outcome</th>
<th>Karnofsky Score</th>
<th>Complications</th>
<th>Follow-Up Period (mos)</th>
</tr>
</thead>
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<tr>
<td>1</td>
<td>M, 44 SAH, Grade 1; neck stiffness</td>
<td>paracellar mass normal mass</td>
<td>50</td>
<td>mixed</td>
<td>70</td>
<td>transient con-fusion</td>
<td>90</td>
<td>none</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>F, 57 visual loss, both eyes</td>
<td>paracellar mass normal mass</td>
<td>25</td>
<td>mixed</td>
<td>80</td>
<td>asymptomatic</td>
<td>100</td>
<td>none</td>
<td>72</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>F, 58 visual loss, rt eye</td>
<td>paracellar mass normal mass</td>
<td>22</td>
<td>cavernous</td>
<td>60</td>
<td>asymptomatic</td>
<td>90</td>
<td>none</td>
<td>52</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>F, 43 SAH, Grade 3; lt central facial palsy; confused visual loss, rt eye</td>
<td>paracellar mass normal mass</td>
<td>25</td>
<td>mixed</td>
<td>90</td>
<td>transient con-fusion</td>
<td>90</td>
<td>none</td>
<td>79</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>F, 62 visual loss, both eyes</td>
<td>paracellar mass normal mass</td>
<td>30</td>
<td>mixed</td>
<td>90</td>
<td>blind, lt eye</td>
<td>80</td>
<td>optic nerve infarct</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>F, 79 visual loss, both eyes</td>
<td>SAH normal</td>
<td>5</td>
<td>mixed</td>
<td>100</td>
<td>asymptomatic</td>
<td>100</td>
<td>none</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>F, 54 SAH, Grade 1</td>
<td>SAH normal</td>
<td>25</td>
<td>mixed</td>
<td>100</td>
<td>asymptomatic</td>
<td>100</td>
<td>none</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>F, 58 lt orbital pain</td>
<td>SAH normal</td>
<td>15</td>
<td>mixed</td>
<td>40</td>
<td>hemiparesis</td>
<td>60</td>
<td>none</td>
<td>68</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>F, 66 SAH, Grade 3; paresis of rt III; lt hemiparesis</td>
<td>SAH normal</td>
<td>16</td>
<td>mixed</td>
<td>80</td>
<td>asymptomatic</td>
<td>100</td>
<td>none</td>
<td>75</td>
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<tr>
<td>10</td>
<td>F, 51 SAH, Grade 2; confused</td>
<td>SAH normal</td>
<td>15</td>
<td>mixed</td>
<td>100</td>
<td>asymptomatic</td>
<td>100</td>
<td>none</td>
<td>36</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>F, 43 rt orbital pain</td>
<td>SAH normal</td>
<td>8</td>
<td>cavernous</td>
<td>100</td>
<td>asymptomatic</td>
<td>100</td>
<td>none</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>F, 55 lt orbital pain</td>
<td>SAH normal</td>
<td>10</td>
<td>cavernous</td>
<td>100</td>
<td>asymptomatic</td>
<td>100</td>
<td>none</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>M, 41 lt hemihypesthesia</td>
<td>paracellar mass normal mass</td>
<td>15</td>
<td>mixed</td>
<td>90</td>
<td>blind, rt eye</td>
<td>80</td>
<td>optic nerve infarct</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>F, 61 visual loss, rt eye</td>
<td>paracellar mass normal mass</td>
<td>25</td>
<td>mixed</td>
<td>70</td>
<td>improved</td>
<td>80</td>
<td>sepsis</td>
<td>72</td>
<td></td>
</tr>
</tbody>
</table>

* Roman numerals denote cranial nerves; CT = computerized tomography; SAH = subarachnoid hemorrhage; IVH = intraventricular hemorrhage.

† Cavernous = intracavernous location; mixed = intracavernous location with subarachnoid hemorrhage extension.

Both procedures were completed during the same operative intervention; no venous grafts were used. All seven patients in Group 2 had purely intracavernous aneurysms. Fifteen patients (Group 3, Table 3) underwent direct clipping of the aneurysm. Of these patients, five had purely intracavernous aneurysms and 10 had intracavernous aneurysms with subarachnoid extension.

Direct clipping of aneurysms arising within the cavernous ICA is performed through a limited pterional craniotomy (Fig. 1). The most lateral portion of the
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Fig. 1. Diagrams of surgical dissection of aneurysms arising from the intracavernous internal carotid artery (ICA). Upper Left: Through a pterional craniotomy, the roof of the orbit, the anterior clinoid process, and the lateral wall of the orbit are removed with a high-speed drill to expose the intracavernous ICA. Upper Right: The cavernous sinus is entered by a direct incision of the dura over the third and fourth cranial nerves. Bleeding from the sinus is controlled with oxidized cellulose packing. The cranial nerves are displaced laterally and the aneurysm neck is dissected under direct vision. When the aneurysm is large, it may be necessary to identify the first branch of the trigeminal nerve at its origin from the gasserian ganglion and follow it into the cavernous sinus in a manner similar to that described above. The sixth nerve usually lies immediately medial and inferior to the first branch of the trigeminal nerve. Lower Left: Once the aneurysm neck is exposed, a clip may be placed directly on the lesser wing of the sphenoid is removed extradurally, and the dura is opened in a semicircular manner based pterionally. The Sylvian fissure is opened widely, the temporal lobe tip is freed of its venous attachments, and the dura over the roof of the orbit, anterior clinoid, and lateral orbital wall is resected intradurally. The bone portions of these structures are removed with a high-speed drill to expose the cavernous ICA and the optic nerve as they pass through the dural foramen and the optic canal (Fig. 1 upper left). The dural sleeve covering the optic nerve is incised to free the optic nerve from its subarachnoid location to the insertion of the anulus of Zinn. The third and fourth cranial nerves and the first division of the trigeminal nerve are then dissected from their subarachnoid portion into and through the cavernous sinus to the superior orbital fissure (Fig. 1 upper right). The dissection begins through the roof of the cavernous sinus, starting with the third nerve and the fourth and first division of the fifth nerve are identified sequentially in a craniocaudal direction. Entering the roof of the sinus permits immediate identification of the ICA with preservation of the anatomical relationship of the artery to the nerves. The ring of dura that surrounds the ICA as it enters the subarachnoid space is transected, moving from the cavernous sinus area to the subarachnoid space. The gradual dissection of the cranial nerves and ICA allows progressive control of bleeding from the cavernous sinus with gentle packing with oxidized cellulose or direct coagulation of the venous channels visualized under the microscope. With distal and proximal control, the carotid artery may be occluded temporarily under barbiturate (2 mg/kg) and lidocaine (1 mg/kg) protection so that the aneurysm may be clipped. This procedure was carried out in the 15 Group 3 patients.

Two patients in Group 1 developed transient neurological deficits which reversed over the course of a week. Two patients in Group 2, in whom ICA occlusion preceded the anastomosis, developed a cerebral infarction, and one of these patients died. In Group 3, two patients who had severe visual deficits preoperatively deteriorated after surgery and became blind in the affected eye. One patient in Group 3 developed an intraventricular hematoma during induction of anesthesia and died without an operation. Complete resolution of symptoms was seen in the rest of the patients and to date they remain well.

Discussion

Aneurysms arising from the cavernous ICA represent a major surgical challenge because of the intimate relationship of the ICA to the cranial nerves contained
within the sinus, and because the ICA is surrounded by the cavernous sinus from its point of exit from the petrous bone to its subarachnoid location.\(^8,10,11,13,22,23,44\)

In anatomical studies performed in cavernous sinuses obtained from cadavers that were selectively injected with resin and decomposed, Parkinson\(^32\) described the cavernous sinus as a series of interconnecting veins contained within the leaves of the parietal dura located around the sella turcica. Dujovny, \(et\ al.,\)\(^13\) reported distinct structures which resemble well-developed vessels and fit best with Parkinson’s description of a venous plexus.

The cranial nerves enter the region of the cavernous sinus in a rostral caudal direction from the margin of insertion of the tentorium cerebelli on the anterior clinoid process, where the third nerve enters, to the area adjacent to the base of the lesser sphenoid wing next to the clivus, where the sixth nerve pierces the dura of the cavernous sinus.\(^13,22,23,32,44\) "The third and fourth cranial nerves are located in the superior portion of the sinus and travel parallel in their course toward the superior orbital fissure, where they exit the cavernous sinus. The first division of the trigeminal nerve arises from the gasserian ganglion and joins the lateral and inferior wall of the cavernous sinus next to the ICA as the artery enters the cavernous sinus from its intrapetrous location. This nerve then follows an upwardly oblique direction until it joins the third and fourth nerves as they exit the sinus into the superior orbital fissure. The sixth nerve is located below and slightly medial to the first branch of the trigeminal nerve, with which it courses parallel to the superior orbital fissure. The triangular space within the cavernous sinus (bordered superiorly by the third and fourth cranial nerves, inferiorly by the first division of the fifth and by the sixth cranial nerves, and posteriorly by the dural wall) is known as Parkinson’s triangle. Parkinson\(^32\) advocated the direct approach to lesions arising from the ICA in the cavernous sinus through this triangle.

Since copious bleeding is frequently encountered when the cavernous sinus is opened, Parkinson\(^32\) combined his direct approach to the sinus with the simultaneous use of extracorporeal circulation and cardiac arrest. The distinct advantages of this method include a completely dry surgical field, easy dissection, and preservation of the cranial nerves and ICA in the cavernous sinus, freedom of rapid and precise movement because of the clarity of the anatomical structures, and the ability to obliterate fistulas or aneurysms by direct clipping of their neck facilitated by the absence of arterial pressure. The disadvantages include the need for a second surgical team, the need for a second operative field and all its inherent surgical morbidity, and the need for full heparinization and its reversal when the procedure has been completed. Abundant intracranial bleeding may develop once cardiac function has been reestablished as a result of the heparin effect and/or the platelet damage caused by the extracorporeal circulation.\(^2\)

Profound hypothermia is required for myocardial and cerebral protection before complete cardiac arrest can be started; numerous dysrhythmic problems, including ventricular tachycardia and ventricular fibrillation,\(^2\) have been reported from its use. Since the safe time for complete cardiac arrest is limited to 1 hour, Parkinson’s\(^32\) combined procedure conducted in the confines of the cavernous sinus should be completed within that time.\(^2\) In most cases, Parkinson found this time limit to be sufficient; however, if a problem arises and it is necessary to work on the carotid artery for longer than 1 hour, myocardial and cerebral ischemic complications might develop from prolonged cardiac arrest.

An alternative to the management of aneurysms in the cavernous sinus is ligation of the ICA in the neck.\(^1,27,30\) However, Roski, \(et\ al.,\)\(^35\) and others\(^17,26,30,31,33,34\) reported a high incidence of ipsilateral cerebral infarctions after selective occlusion of the ICA. Spetzler, \(et\ al.,\)\(^38,39\) found that a combined procedure involving ICA ligation in the neck followed by a cortical extracranial-intracranial (EC-IC) anastomosis was successful in the management of giant ICA aneurysms. However, Diaz, \(et\ al.,\)\(^5\) observed the development of transient episodes of cerebral ischemia during the progressive occlusion of the Selverstone clamp placed on the cranial ICA, in spite of a functional cortical EC-IC anastomosis, and recommended that the ICA be occluded under full heparinization over the course of 1 week. The management of Group 1 patients in the present series was based on Spetzler’s treatment of giant ICA aneurysms. The development of ischemic complications in Group 1 patients after the occlusion of the ICA with a functional cortical EC-IC bypass motivated the search for other forms of treatment that would allow abrupt occlusion of the ICA, would provide greater arterial flow to the brain, and would not be associated with ischemic complications.

Diaz, \(et\ al.,\)\(^9\) found that a cortical STA-MCA anastomosis fails to reach its optimum size in over 80% of patients for the first 6 to 12 weeks after the procedure. As an alternative, Little, \(et\ al.,\)\(^24\) reported that the anastomosis of a short vein graft from the STA to the MCA produces a high-flow shunt with a high degree of patency. Diaz, \(et\ al.,\) also noted that an anastomosis of the STA to a proximal branch of the MCA produced a high-flow system with a high degree of patency from the 1st week after surgery. The combination of either of these high-flow bypass procedures with simultaneous ICA occlusion in the neck would therefore seem appropriate to manage otherwise surgically inaccessible carotid artery aneurysms. However, there have been reports of patients with giant aneurysms treated with a proximal arterial ligation and a cortical EC-IC anastomosis who developed fatal SAH.\(^18,25\) The trapping or double ligation of the ICA in the neck and intracranially beyond the aneurysm would limit the potential for bleeding from the aneurysm after a high-flow anastomosis procedure.
Surgical treatment of cavernous sinus aneurysms

Group 2 patients in the present series underwent the combined trapping of the ICA with a simultaneous high-flow anastomosis beyond the carotid artery occlusion. Most patients tolerated the procedure well, but two developed cerebral infarctions, and one of them died after the operation. In this patient, a direct approach to the aneurysm through the cavernous sinus was first attempted unsuccessfully, and resulted in severe bleeding from the aneurysm. The aneurysm was trapped to control the bleeding and an EC-IC anastomosis was then performed. However, the patient developed a massive cerebral infarction and died. To prevent this complication, it would be reasonable to perform an EC-IC anastomosis prior to a direct approach to a giant cavernous aneurysm. If the aneurysm could not be ligated directly, the ICA may then be trapped safely under the protection of the established high-flow anastomosis. No patient has developed a cerebral infarction when this procedure has been used.

Dolenc reported a direct attack on the ICA within the cavernous sinus without the need for cardiac arrest. Through an extradural pterional or subtemporal approach, the lesser sphenoid wing is exposed from the anterior clinoid process to its most lateral extent. The lesser sphenoid wing, the orbital roof, and the anterior clinoid process are then removed to expose the cavernous portion of the ICA. Proximal arterial control is gained by dissecting out the intrapetrous portion of the ICA lateral to the gasserian fossa. A small dural incision is made to expose the subarachnoid portion of the ICA to gain distal arterial control. The cavernous sinus is then opened, and control of any venous bleeding is obtained by packing the cavernous sinus with oxidized cellulose. Control of bleeding from the ICA is easily obtained by temporarily occluding the artery between the exposed proximal and distal sites. To enter the cavernous sinus, Dolenc prefers to first dissect and mobilize the third and fourth cranial nerves and the first division of the trigeminal nerve free of the ICA.

The advantages of Dolenc's approach include the ability to enter the sinus without the need for cardiac arrest, gradual and progressive control of sinus bleeding, the ability to identify and preserve cranial nerve integrity with early dissection and mobilization, and the ability to control any arterial bleeding by ligating the ICA whenever necessary. The disadvantages include difficulty in exposing the intrapetrous portion of the ICA through a subtemporal approach, the need for a large craniotomy to obtain access to the temporal region and petrous ICA, and the need for significant brain retraction to expose the cavernous sinus and the petrous ICA located deep under the temporal lobe. Dissection of the lateral petrosal area may be associated with injury to the greater petrosal nerve and loss of lacrimal gland function or, if the tension on the nerve is marked, the development of a temporary or permanent seventh nerve palsy. Dissection of the intrapetrous ICA is difficult because the bone canal is relatively narrow and is bordered by the gasserian fossa medially and the semicircular canals laterally. The space around the artery is small and a periarterial venous plexus surrounds the ICA. The potential for injury to the semicircular canals, the trigeminal nerve, the facial nerve, or the ICA itself makes this approach undesirable.

On anatomical dissections of the cavernous sinus performed on sphenoid bone blocks, Dujovny, et al., reported the roof of the sinus as the most direct route to the area of the cavernous ICA where aneurysms usually arise. Dujovny's report served as the basis for the pterional transdural approach to cavernous sinus aneurysms. The intracranial dissection is performed through a limited pterional craniotomy with minimal brain retraction. Proximal arterial control is gained approaching the ICA in the neck through a small submandibular incision. A transdural approach facilitates the direct observation of the brain, clear exposure and mobilization of the optic nerve, and preservation of the third and fourth cranial nerves and the first division of the trigeminal nerve which may be dissected from their subarachnoid portion into and through the cavernous sinus to the superior orbital fissure. The dissection performed from the roof of the cavernous sinus permits easy access to the ophthalmic artery origin and its course into the orbit, gradual control of venous bleeding from the sinus, and clear exposure of the ICA within the sinus under controlled conditions. Aneurysms arising from the horizontal and ascending portions of the cavernous ICA are accessible through this approach, and their obliteration is feasible (Fig. 1 lower left). Since the area of the lateral temporal fossa is not approached, none of the potential problems previously described for this approach (temporal lobe swelling, seizures, carotid artery trauma, seventh nerve injury, and others) are of concern. The potential risk of dissection of the ICA in the neck is considerably less than that associated with exposure of the intrapetrous portion of the ICA.

The problems that have prevented direct occlusion of some aneurysms in the cavernous carotid artery include calcifications or atheromatous plaques contained within the wall of the ICA or the aneurysm itself, loss of definition of the ICA wall because of overexpansion by the aneurysm (making the entire wall a fusiform aneurysm), technical inability to place fenestrated clips to reconstruct a lumen on the ICA, and inability to place a clip or a ligature around the aneurysm neck because of the intimate adherence of the aneurysm to the base of the skull. It is possible that our inability to obliterate some of those aneurysms through a direct approach has resulted from our initial hesitation to remove enough bone from the sphenoid wing adjacent to the cavernous sinus. A more radical exposure of the entire cavernous carotid area will likely increase our ability to address this problem.

In patients with asymptomatic intracavernous aneurysms discovered incidental to other cerebrovascular problems, it would seem unreasonable to pursue a direct operative approach because of the potential intrinsic
morbidity associated with the operation. Perhaps one may consider the use of a percutaneous balloon occlusion of the aneurysm or of the ICA as a possible alternative in the management of symptomatic aneurysms. However, balloon techniques do not have widespread application or sufficient reliability to make them the preferred method of treatment for intracavernous aneurysms. Balloon techniques were not used in this series of patients with cavernous sinus aneurysms because these techniques were not available at Henry Ford Hospital during this time. We would now consider obliterating the ICA with a balloon, or possibly occluding the aneurysm by means of this technique. However, for large aneurysms in the cavernous sinus, a balloon would act as a mass and may not relieve the symptoms of compression. Trapping and evacuation of the aneurysm, plus an EC-IC anastomosis, may be a better therapeutic alternative for these patients.

Conclusions

For symptomatic aneurysms arising from the intracavernous ICA, several points should be considered. The direct approach to aneurysms arising from the ICA in the cavernous sinus is probably the best initial alternative in the management of these patients. From our experience, it may be concluded that aneurysms less than 15 mm in diameter may be clipped directly. For aneurysms larger than 15 mm, a decision must be made between trapping the ICA intracranially and in the neck and performing a high-flow EC-IC anastomosis to the proximal MCA with the STA, or performing a venous graft. Whenever the ICA is trapped, an EC-IC anastomosis performed prior to the carotid artery occlusion would provide the area distal to the occlusion with immediate flow and prevent the development of cerebral ischemia as the trapping procedure is completed. In situations where the initial attempt at clamping the aneurysm is unsuccessful, a high-flow EC-IC anastomosis followed by either ICA trapping or ICA occlusion by a detachable balloon may be considered once the patient has recovered from anesthesia. In patients with giant aneurysms, it would be reasonable to proceed with trapping of the aneurysm from the onset, preceded by a high-flow EC-IC anastomosis performed during the same procedure. A cortical EC-IC anastomosis does not seem to be a reasonable alternative, since it does not provide the middle cerebral circulation with immediate consistent high flow. Since the present series is small, it would be unwise to conclude that the direct attack on aneurysms in the cavernous sinus is the only or the best alternative in their management. However, the success reported with the direct approach to these lesions makes further studies of this approach warranted.

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References

3. Bendetti A, Curri D: Direct attack on carotid ophthalmic and large internal carotid aneurysms. Surg Neurol 8:49–54, 1977
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