Hypothermia and cardiac arrest in the treatment of giant aneurysms of the cerebral circulation and hemangioblastoma of the medulla

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Hypothermia and elective cardiac arrest may be of benefit during surgery of some technically difficult lesions of the brain. Eight giant aneurysms and one hemangioblastoma of the medulla were operated on with no mortality, using hypothermia and cardiac arrest. All but one aneurysm were excluded from the cerebral circulation, and the hemangioblastoma appeared to be completely excised. Careful attention to clotting factors, depth of hypothermia, and time of bypass were important factors in the success of this series.

**KEY WORDS** • hypothermia • cardiac arrest • giant aneurysm • hemangioblastoma

Some lesions of the brain present technical problems not easily solved by standard neurosurgical approaches. We have recently treated 10 cases of giant aneurysms (larger than 2.5 cm in maximum diameter) and one with hemangioblastoma of the medulla. Two patients with giant aneurysms died of massive hemorrhage preoperatively. The remaining eight giant aneurysms and the hemangioblastoma appeared to be best treated microsurgically in combination with hypothermia and elective cardiac arrest. Thanks to technical advances in pump oxygenators and heat exchange pumps, as well as the recent availability of multiple clotting factors and an increased understanding of hypothermic physiology, the use of hypothermia and elective cardiac arrest as an adjunct to neurosurgery has become far safer and less time-consuming than in years past.

Although a number of neurosurgical centers pioneered hypothermic cardiac arrest techniques 15 to 20 years ago as an aid to intracranial aneurysm surgery, these techniques fell into disuse by neurosurgeons for a number of reasons: the combination of hypothermia and extracorporeal circulation led to multiple clotting problems; frequently the patient had to be packed in ice and there were difficulties in controlling temperature drift; and the procedure was very time-consuming. As Drake, et al., correctly noted: “The use of cardiopulmonary bypass and deep hypothermia in the surgical treatment of ruptured intracranial aneurysms creates enough additional hazards so that it is probably not warranted in cases in which the aneurysm can be obliterated by more conventional means.” Moreover, with increased use of the operating microscope, a better understanding of the proper timing for surgical intervention, and better neuroanesthesia, the surgical morbidity with intracranial aneurysms has become quite acceptable without hypothermia and circulatory arrest.

Cardiac surgeons, however, have persisted in refining hypothermic circulatory arrest in order to repair complex congenital heart defects and facilitate aortic arch replacement. These techniques have allowed us to carry out definitive surgery on eight giant aneurysms and remove one hemangioblastoma from the medulla.

**Clinical Material**

The cases are summarized in Table 1. Of the 10 patients with aneurysms, six (60%) bled at some time in their clinical course, two fatally. Five aneurysm
patients suffered from mass effect, two having seizures. Three of these patients also bled. One patient presented with emboli from the giant aneurysm sac.

Five of the aneurysms originated from the middle cerebral artery (MCA), two from the basilar caput, one from the junction of the vertebral and basilar arteries, and two from the internal carotid artery (ICA) at the ophthalmic artery. One patient had symmetrical giant MCA aneurysms. The right-sided aneurysm was thought to be the source of the subarachnoid hemorrhage (SAH) in this case, and was repaired first. The opposite side was operated on electively 2 months later, also under hypothermic arrest. The hemangioblastoma arose from the medulla at the posterior end of the fourth ventricle, filling the vallecula. The patient presented with sudden hemorrhage into the fourth ventricle.

**Surgical Technique**

The surgical technique evolved from previous cases of aortic arch replacement performed by the Stanford Cardiac Surgery Department. Monitored variables include the electrocardiogram, direct radial artery pressure, central venous pressure, expiratory CO₂ and O₂, urine output, and nasopharyngeal and rectal temperatures. Premedication is restricted to a short-acting narcotic, such as fentanyl. Other usual agents, such as diazepam, Phenergan (promethazine), and scopolamine, should be avoided because of the duration of their postoperative effects.

Anesthesia is induced by intravenous narcotics infused over 10 to 60 minutes (Demerol (meperidine), 3 to 9 mg/kg, or fentanyl, 6 μg/kg) and pancuronium, 1.3 mg/kg. Surface cooling is initiated immediately after induction of anesthesia, by means of a thermal blanket. Nitroprusside infusion is used to assure maximum vasodilation, with chlorpromazine (total less than 10 mg, with 1 mg or less per dose) when nitroprusside requirements exceed 2 μg/kg/min. Thipental, 30 mg/kg, is infused over 30 to 90 minutes, just fast enough to complete the infusion before switching to the bypass circulation.

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**TABLE 1**

*Clinical summary in 11 cases in this series*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Symptoms</th>
<th>Lesion</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>46, M</td>
<td>SAH, progressive dementia, seizures, severe headache</td>
<td>giant lt MCA aneurysm (4 cm)</td>
<td>excellent; no more seizures; headache improved; mentation good</td>
</tr>
<tr>
<td>2</td>
<td>29, M</td>
<td>hemorrhage, 4th ventricle</td>
<td>hemangioblastoma of medulla</td>
<td>excellent; total tumor removal; no neurological deficit</td>
</tr>
<tr>
<td>3</td>
<td>38, F</td>
<td>SAH</td>
<td>giant rt MCA aneurysm (3 x 3 cm)</td>
<td>excellent; no neurological deficit</td>
</tr>
<tr>
<td>4</td>
<td>38, F</td>
<td>asymptomatic</td>
<td>giant lt MCA aneurysm (3 x 2 cm)</td>
<td>good; transient short-term memory deficit, cleared with time</td>
</tr>
<tr>
<td>5</td>
<td>44, M</td>
<td>seizures</td>
<td>giant basilar aneurysm (3 x 3 cm)</td>
<td>good; initial hemiparesis; 3rd nerve palsy; dysphasia; all cleared</td>
</tr>
<tr>
<td>6</td>
<td>57, M</td>
<td>TIA's</td>
<td>giant lt MCA aneurysm (2 x 3 cm)</td>
<td>excellent; no neurological deficit</td>
</tr>
<tr>
<td>7</td>
<td>59, F</td>
<td>SAH</td>
<td>giant basilar aneurysm (3 x 4 cm)</td>
<td>good; 3rd nerve palsy, clearing; slipped clip but late total thrombosis of aneurysm presenting with rt homonymous hemianopia 6 mos postop</td>
</tr>
<tr>
<td>8</td>
<td>48, F</td>
<td>SAH</td>
<td>giant right MCA aneurysm (3 x 2 cm)</td>
<td>excellent; no neurological deficit; postop pulmonary embolus</td>
</tr>
<tr>
<td>9</td>
<td>62, F</td>
<td>progressive visual loss, first in lt eye, then in rt</td>
<td>giant ICA aneurysm (2.5 x 3 cm)</td>
<td>excellent; no neurological deficit; some return of vision on lt; complete clearing on rt</td>
</tr>
<tr>
<td>10</td>
<td>67, F</td>
<td>dizziness &amp; ataxia</td>
<td>giant aneurysm at junction of right vertebral &amp; basilar</td>
<td>patient fell &amp; broke hip while awaiting surgery; fatal hemorrhage while recovering from hip pinning</td>
</tr>
<tr>
<td>11</td>
<td>50, F</td>
<td>progressive visual loss</td>
<td>giant lt carotid ophthalmic aneurysm (4.5 x 3 cm)</td>
<td>fatal hemorrhage during angiography</td>
</tr>
</tbody>
</table>

* SAH = subarachnoid hemorrhage; MCA = middle cerebral artery; TIA = transient ischemic attack; ICA = internal carotid artery.
Sufficient blood is withdrawn through a second radial artery cannula to give an estimated packed cell volume (PCV) on bypass of approximately 20% (for a 70-kg patient with a starting PCV of 40%, remove three units of whole blood, adding one unit of packed cells to 1700 ml of bypass prime). A complex algorithm and PCV sampling during withdrawal of the blood are used. Chilled normal saline, with 4 to 8 mEq KCl/liter, is infused to maintain central venous pressure constant (approximately 10 torr). A typical total infusion before bypass is 5 liters.

Cooling is begun after the patient is asleep to reduce cardiovascular stress. The patient is undraped when possible. The cooling blanket is used (infusate at 4°C), and all intravenous solutions are chilled to 4°C. Minute ventilation is maintained with high gas flows to reduce rebreathing and heat conservation. As noted, aggressive vasodilation is used to accelerate heat exchange. At the time of initiation of bypass circulation, the patient's temperature is 30°C to 27°C.

During cooling, minute ventilation is maintained. In our earlier patients, modest CO₂ was added (1% to 2%) when body temperature fell below 30°C to keep arterial pCO₂ above 27 torr. However, clinical experience dictated progressively lower arterial pCO₂ with cooling. Our experience agrees with that of Rahn, et al.³⁰ the appropriate pCO₂ is such that pH is 7.42 or slightly greater, when the blood sample is at 37°C.²⁰,²³ The patient's pCO₂ will decrease approximately 40% per 10°C cooling. End-tidal values are typically 3 to 5 torr higher than true arterial values, but arterial samples are necessary for repeated calibration because of individual variation. We have not measured cerebral O₂ consumption during these cases.

Neurosurgical exposure is begun immediately. While cooling proceeds, the lesion is exposed and meticulous hemostasis attained. Multiple dural tenting sutures are used to control epidural bleeding. Cannulation is carried out as shown in Fig. 1. Heparin (300 IU/kg) is given, and a No. 28 French catheter passed via the right femoral vein to the right atrium. A second catheter is placed at the iliac bifurcation through the left femoral vein. A No. 22 French Bardic catheter is inserted into the right femoral artery for arterial return. Since the left femoral vein is occluded by a catheter, the left femoral artery is clamped. Cardiopulmonary bypass is then instituted at a flow of approximately 40 ml/kg/min, and the patient cooled to the target temperature (17°C to 20°C). As the patient approaches the target temperature, the bypass perfusate is warmed to the target temperature (starting about 10°C cooler), so that actual nasopharyngeal temperature is within 1°C of target temperature during arrest, and temperature drift is less than 1°C. Because of rapid cooling, heart temperature is difficult to estimate, but progressive bradycardia and fibrillation are observed at a nasopharyngeal temperature of 27°C to 23°C. Cooling occurs at an approximate rate of

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**Fig. 1.** Schematic diagram of the bilateral groin cannulation for extracorporeal circulation.
0.2°C/min, circulatory arrest being initiated when the target temperature is reached. Table 2 summarizes the total perfusion time, arrest time, and lowest core temperature in the nine operations. Just prior to arrest, additional muscle relaxant is administered (pancuronium, 0.04 mg/kg). The patient is positioned with the head elevated above heart level so that when the pump is stopped and the venous lines are opened, the available blood volume is drained into the pump reservoir, markedly reducing brain bulk and collapsing the aneurysm or vascular tumor.

Following the neurosurgical procedure, the pump is restarted while the operative field is inspected to be certain the lesion has been truly dealt with and no major arterial bleeders are left unsecured. There is always some capillary and venous oozing that is best left alone until all clotting factors are restored. The patient is then rewarmed to 34° to 36°C nasopharyngeal temperature, using the heat exchanger with the prime about 10°C above patient temperature (to a maximum of 40°C), and a warming blanket. 21 The patient’s heart either resumes a normal sinus rhythm spontaneously or is converted by an external shock of maximum of 40°C and a warming blanket. 21 The patient’s heart either resumes a normal sinus rhythm spontaneously or is converted by an external shock of 300 W/sec when cardiac temperature is estimated to be at 28°C or above. At this time, the nasopharyngeal temperature may be only 23°C. Right atrial pressure is increased to normal as soon as the heart can function effectively (usually within 10 minutes after defibrillation), adding pulsatile flow and cardiac output to pump flow. Sodium nitroprusside and chlorpromazine are again used, in similar doses, to maximize vasodilation and improve blood flow distribution. We have used a pulsed flow device,* but with the above measures it appears to offer little advantage. Furosemide is administered in progressive doses (beginning with 5 mg) if a urine output of 200 ml/hr or more does not commence within 20 minutes of restoring physiological blood pressure and flow above 30°C.

Bypass is discontinued when the rewarming temperature is reached, and decannulation is performed. Only sufficient blood volume is returned from the pump to the patient to stabilize the patient until reversal can begin; the remaining perfusate is spun down and the red cells subsequently readministered. Protamine sulfate (10 mg/1000 units of initial heparin dose) is given immediately after decannulation, normalizing the activated clotting time. Platelets (six units), fresh frozen plasma (four units), calcium chloride (1 to 2 gm), the patient’s unchilled blood, and Factor IX complex (Proplex, two units) are usually then infused in approximately that order. Bleeding from the brain is usually minimal prior to restoring clotting factors, but epidural bleeding may be copious despite the dural tenting sutures. The bleeding comes under control with the administration of the clotting factors. When the operative field is dry, the craniotomy is closed. It is important to realize that the usual laboratory screen can suggest complete clotting correction without Factor IX, but ooze continues. Typically, both the laboratory and clinical impression suggest normal coagulation when the patient’s temperature has returned to 37°C (about 3 to 5 hours after the patient arrives in the intensive care unit).

An epidural telemetered intracranial pressure (ICP) monitor is then inserted, usually through a separate burr hole just anterior to the coronal suture, preferably on the nondominant side. 22 After the insults of hypothermia and the anesthetic agents used, the patient will sleep from 8 to 48 hours; the ICP monitor is essential to follow patients during this period. In one case, it alerted us to a rise in ICP from temporal lobe swelling that responded to intravenous mannitol. In our most recent patients, with strict control of pH added to the previously mentioned criteria, extubation has been performed the evening of surgery without difficulty, although subtle affect disturbances may persist for 48 hours.

**Operative Results**

Eight patients had nine surgical procedures, performed under hypothermia and elective cardiac arrest (Table 1). There was no operative mortality. There were six postoperative complications. Five were transient and one (in Case 7) was permanent, a stroke that occurred 6 months postoperatively due to late thrombosis of the residual sac of an aneurysm. Of the five transient complications, two were small pulmonary emboli occurring 1 and 3 months postoperatively in two patients, one in an aneurysm patient and the other in the patient with the hemangioblastoma. We assume the emboli arose from cannulated femoral

* Datascope pulsatile assist device from Datascope Corp., Paramus, New Jersey.
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veins, although no sign of thrombophlebitis was present in either patient. Both had preoperative prolonged bed rest and antifibrinolytic therapy (aminocaproic acid (Amicar) 24 gm/day) before their neurological status cleared sufficiently to allow surgical intervention. Both patients recovered without sequelae following anticoagulation therapy for 3 months. One patient sustained a small intracerebral hematoma, seen on the postoperative computerized tomography (CT) scan. It appeared to be just under the site of the frontal lobe retractor. The patient was unharmed and the hematoma resolved without treatment. Two patients, both with giant basilar artery aneurysms, had postoperative ipsilateral third nerve palsy and contralateral hemiparesis. One also had a mild dysphasia. All these last complications have cleared, save for one third nerve palsy that has not yet completely resolved.

Postoperative angiography was performed in all the aneurysm patients. The aneurysm was excluded from the cerebral circulation in all but one case (Case 7). In this case, angiography showed that the aneurysm clip had slipped off. The aneurysm, however, was thrombosed except for a channel between the basilar and left posterior cerebral arteries. We thought that we could not improve upon this result so nothing further was done. Six months later, the patient presented with the sudden onset of a right homonymous hemianopsia. An infusion CT scan showed total non-filling of the aneurysm with an infarct of the left occipital pole, presumably secondary to thrombosis of the residual channel between the basilar and left posterior cerebral arteries. The patient has since shown some opening out of the right visual field but some, presumably permanent, visual field loss remains. A 30-month follow-up period of the patient with the hemangioblastoma shows no evidence of recurrence either clinically or on CT scan. The five patients who were employed before the onset of their illness have all returned to work.

Discussion

Giant aneurysms are uncommon, occurring in only 3% to 5% of patients suffering from intracranial aneurysms.3,15,17 Those giant aneurysms arising from the ICA at the base of the skull appear to have a benign course,10 whereas those arising from arteries within the subarachnoid space seem to be more lethal, presenting with hemorrhage4,16,17,20 or as a mass.10,20 Direct surgical attack on these lesions has been difficult.2,16,17,28 The size of the aneurysm and the difficulty in occluding the sac while still maintaining patency in the parent vessel and its branches make these lesions a formidable surgical challenge. Many times the surgeon must settle for a proximal ligation, trapping, or investing procedure, or even abandon the attempt without definitive treatment.

In 1969, Bull2 presented the radiological appearance and clinical course of 22 patients with giant aneurysms presenting at the National Hospital, Queen Square, London. Six patients had probably bled from their aneurysm (27.3%). Thirteen aneurysms were on the carotid circulation, the remainder were vertebral-basilar. Five patients were treated by carotid ligation, with two deaths. One patient had both a carotid ligation and an intracranial approach and died. A total of five patients had surgical excision, with only one survivor, and that patient was left with hemiplegia. Two patients were explored, but the surgeon thought nothing could be done to the aneurysm; one died within the year. One patient succumbed to vertebral ligation for a vertebral aneurysm believed to be inoperable after intracranial exploration. In nine patients, the lesion was thought to be inoperable and nothing was done. Three of these patients died within the year. The majority of the survivors were seriously handicapped. In all, 13 patients were operated on, with eight deaths (61.5%). Bull concluded that “the most disappointing aspect was the treatment.”

Heiskanen and Nikki9 discussed large intracranial aneurysms, but of their 15 cases, only three carotid aneurysms (one a carotid-cavernous fistula) could be classified as “giant.” One patient died of unrelated causes; the other two survived carotid ligation with fair results in one (the patient with carotid-cavernous fistula) and a poor result in the second.

Morley and Barr15 reviewed 28 patients with giant aneurysms. Eleven were intracavernous carotid aneurysms. The authors believed that these intracavernous lesions were relatively innocuous, save for local pressure on third, fourth, fifth, and sixth cranial nerves, and probably did not require treatment, whereas the giant aneurysms located within the subarachnoid space were quite dangerous. Eight of the 17 extracavernous aneurysms bled (47%). Seven of these 17 patients died, four due to SAH. Only four patients had a direct attack upon the aneurysm. Two did well, one did poorly, and one died. Five of the other patients were explored, but the lesion was thought to be inoperable. It was the feeling of the authors that direct attack with obliteration of the aneurysm was not usually feasible, and they recommended, “more out of despair than enthusiasm,” carotid ligation when the aneurysm arose on the carotid vascular tree.

Extending that observation to the vertebrobasilar circulation, Drake6 reported the results of vertebral or basilar artery occlusion in the treatment of 21 patients with giant aneurysms of the posterior circulation. Three died in the acute stage of their illness, and two
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succumbed later to recurrent hemorrhage. Two more were thought to have a poor surgical result, yielding a combined morbidity and mortality of 33%. Fourteen were classified as good or excellent results, but in only six was the aneurysm believed to be completely thrombosed.

Recently, Drake\textsuperscript{4} reviewed his entire series of giant aneurysms. Of the 174 lesions, 66 were on the carotid system, and 108 were vertebrobasilar. Subarachnoid hemorrhage occurred in 36\% of these patients. Interestingly, one-third of these had mural thrombi, sometimes massive, within the aneurysm sac. Drake classified 71.5\% of these cases as good to excellent results, 13\% had poor outcomes, and 15.5\% died. Aneurysmal neck occlusion, however, could be accomplished in only 38.5\% of the cases. Thirteen cases had exploration only, while the remainder had a number of procedures, including wrapping,\textsuperscript{6} intraluminal thrombosis with hair or wire,\textsuperscript{7} a proximal occlusion,\textsuperscript{7} or trapping.\textsuperscript{15} Extracranial-intracranial bypass was used prior to proximal occlusion in some cases, and it was thought to confer a measure of protection to the patient. An ingenious delayed proximal arterial occlusion was performed in a number of patients in the awake state using an arterial snare applied to the parent artery during surgical exploration when it was clear that the aneurysm could not be ligated at its neck.

Other cases have been reported of giant MCA aneurysms that were treated by superficial temporal artery-MCA anastomosis, followed by proximal occlusion of the MCA.\textsuperscript{1} One must gamble in these cases that the fresh anastomosis can supply sufficient blood to the middle cerebral arterial tree to prevent ischemia. Other surgeons have simply excised the giant aneurysm, sacrificing the parent artery, sometimes with an acceptable result.\textsuperscript{26} Sundt and Piepgras\textsuperscript{29} reviewed 80 cases of giant aneurysms occurring in 594 operations for aneurysms (13.5\%). They had a very commendable 4\% mortality and 14\% morbidity. Direct clipping or excision was accomplished in 55 patients (69\%), and the remainder had proximal ligation with or without an extracranial-intracranial bypass or a trapping procedure. Results with aneurysms of the carotid circulation were quite good, but less so with posterior circulation aneurysms. In the 15 cases with posterior circulation aneurysms, the morbidity was 30\% and the mortality 7\%. Morbidity of basilar head aneurysms was 50\%.

Onuma and Suzuki\textsuperscript{17} reviewed 32 giant aneurysms. Seven were infracarotid in location. In their series, 72\% presented with SAH. Twenty-four patients underwent a direct approach to the aneurysm, and four had a carotid ligation. Some cases were operated on under mild hypothermia (without cardiac arrest). In 10 cases, the lesion could be extirpated after the neck was ligated. In 12 cases, the surgeon had to settle for partial ligation plus reinforcement of the aneurysm by muscle wrapping. Two patients had to have a trapping procedure and neither survived. Overall, the mortality for direct surgery was 20.8\% and the morbidity was 37.5\%.

Sonntag, \textit{et al.},\textsuperscript{28} reviewed the experience of the Neurosurgical Service at New England Medical Center. They reported 13 cases of extracavernous large aneurysms (1.5 to 2.5 cm in diameter); 69 of these patients presented with SAH. Eight cases were approached directly, but only in five cases could the aneurysm be clipped. There were three deaths (37.5\%), two after actual clipping and one following exploration only.

Although we have so far used hypothermia and circulatory arrest in only eight cases of giant aneurysm, the lack of mortality and the low morbidity are encouraging. We believe that the success of the procedure is due to collapse of the aneurysm, such that the vascular anatomy at the base of the aneurysm can be more easily appreciated. The parent artery and its branches can be identified and preserved. In the cases we have operated on, there has really been no aneurysm neck to ligate but, rather, the parent vessel goes into the base of the sac at one point and it or its branches emerge at another point. Not infrequently, these two points are separated by 1 cm or more of thick-walled aneurysm base (Fig. 2). With the aneurysm inflated by blood, the base is usually hidden from view and can only be seen by retracting or rotating the sac. Ligation or clipping of the base of the expanded lesion is difficult because there is no clear demarcation of the sac from the parent artery (Fig. 3). Clips applied to the sac tend to slip down to occlude the parent vessel or its branches. Ligation carries the risk of kinking the artery of origin and causing either immediate or delayed occlusion. Rupture of the sac during dissection tends to obscure the anatomy even more, frequently leading to a trapping procedure, often with disastrous consequences for the patient.

With circulatory arrest, dissection of the aneurysm from the brain and its separation from adherent arterial branches can be safely and rapidly done. Under the operating microscope, the anatomy can be clearly defined and a decision made whether to open the aneurysm and carry out an endaneurysmorrhaphy\textsuperscript{27} (preferred if there is a large amount of laminated clot within the sac) or obliterate the aneurysm with a ligature or a series of clips. The ligature or clips can be precisely applied, since the sac is collapsed, to
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Fig. 2. Case 5. Left: Angiogram of a giant basilar aneurysm showing the long segment of the basilar artery from which the aneurysm originated (arrows). Right: The basilar artery after the aneurysm was clipped. Some residual ectasia remains but continuity is maintained. The base of the aneurysm had to be left because brain-stem perforating arteries arose from it.

Fig. 3. Case 9. Left: Angiogram of a giant internal carotid artery (ICA) aneurysm that is totally obscuring its origin. Center: Oblique view to demonstrate the lengthy segment of the ICA incorporated into the aneurysm base. Right: Postoperative angiogram after ligation of the aneurysm showing continuity of the ICA. Postoperative vasospasm is also present.
maintain patency in the parent artery and continuity with its branches (Fig. 4).

As Drake noted, we have also found that massive thrombosis within an aneurysm sac does not confer immunity to bleeding. One of our patients with a large vertebrobasilar junction aneurysm (Case 10) was shown on arteriography to have a mural thrombus that occluded all but a small crescent of aneurysm near the neck (Fig. 5). She presented with ataxia from brain-stem compression. Unfortunately, she broke her hip from a fall. While awaiting convalescence from her hip surgery, the aneurysm ruptured and she died. A second patient died of aneurysm rupture during angiography (Case 11). She had never bled previously. One must, we believe, abandon the notion that giant aneurysms only rarely bleed.

Hemangioblastomas of the brain stem are not common. We have had experience with only three such cases. Two, operated on in the standard way, could not be completely removed and resulted eventually in the death of the patients, both young adults. Following the lead of Patterson and Fraser, we were able to completely remove the last such tumor and the patient incurred no neurological deficit. With the patient in a semisitting position under hypothermia and circulatory arrest, the tumor, initially very vascular and friable, became a pale, shrunken mass that separated without too much difficulty from the medulla. Dissection with bipolar forceps under the operating microscope allowed for occlusion of all feeding vessels to the lesion at their point of entry without disturbing blood supply to the medulla. With a completely bloodless field, there is no need to use a suction apparatus during the dissection within the medulla, which minimizes damage.

The bleeding tendency seen in patients following extracorporeal circulation can be due to a number of defects in the clotting scheme. In any individual case, it is usually believed due to one or several of the following disorders: 1) thrombocytopenia and abnormal platelet function; 2) inadequate correction of heparinization; and 3) inhibition (mainly dilutional) of coagulation Factors I, II, V, VII, X, and XIII. The addition of deep hypothermia further inhibits clotting by delaying all the enzyme-mediated steps in the coagulation cascade.

Biological abnormalities in clotting occur in 68% to 100% of cases during and after use of extracorporeal
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For the cardiac surgeon, however, only 2% to 3% of these patients exhibit clinically significant bleeding.

Neurosurgical patients, however, are intolerant of postoperative bleeding so that intracranial hemostasis must be absolute. Drake reported that several catastrophic bleeding episodes occurred during surgery with hypothermic cardiac arrest, causing him to abandon the procedure. We have, therefore, adopted a “shotgun” approach to replacing clotting factors since the more sophisticated clotting studies are not reliably available in time to affect replacement therapy during surgery. In addition, laboratory evidence of adequate replacement is sometimes associated with bleeding, which can be corrected by further factor administration. Accurate reversal of heparin and platelet infusions are probably the two most important corrections, and the effect can be measured rapidly with the activated clotting time and platelet counts. Heparin levels in the blood of greater than 0.2 IU/ml were found in 58% of patients operated on with extracorporeal circulation and deep hypothermia following neutralization with protamine. There was also a heparin rebound in 34% of these patients 3 hours after the procedure that required additional protamine.

Infusion of fresh frozen plasma and the patient’s own warm blood helps correct the clotting factor deficit which, with the exception of Factor V, is primarily dilutional. The administration of concentrated clotting factors (Proplex) is also useful. Fibrinolysis may be accentuated with hypothermia, although the reason for this is not clear. In order to minimize clotting deficits, the depth and duration of hypothermia and bypass should be the least that affords adequate protection during definitive surgery. Therefore, as much of the exposure and preliminary dissection as can be safely done should be completed before cardiopulmonary bypass begins.

In contrast to the standard neurosurgical approaches, the use of hypothermia and cardiac arrest in these difficult lesions has, as other authors have noted, allowed a clearer assessment of the anatomy involved and a far more precise and controlled treatment. Hopefully, the encouraging results in this small group of patients will be borne out in a larger series.

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