Occipital to posterior inferior cerebellar artery bypass surgery

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The results, complications, and technical aspects of occipital to posterior inferior cerebellar artery (PICA) bypass surgery are reviewed. Patients were divided into two groups: those considered to be a high risk for posterior circulation infarct but not disabled by the symptoms or deficits (eight patients), and those moderately or severely disabled at the time of admission (eight patients). Postoperative angiography revealed that 15 of the 16 grafts were patent. In 10 of the 15 patent grafts, the bypass graft served as a sole or major blood supply of the vertebral basilar system; in five patients, flow was limited to the distribution of the PICA. Eight patients achieved full employment or normal activity, six were improved but did not return to full employment, and two patients were unchanged. Ataxia was the major residual deficit in these patients.

KEY WORDS • occipital to PICA bypass • ischemic symptomatology • arterial bypass • vertebrobasilar circulation • anastomosis

Case Material

This operation was performed in 16 patients for a variety of ischemic symptomatology attributable to occlusive disease of the posterior circulation. These symptoms included: progressing stroke, transient ischemic attacks (TIA's), minor brain-stem infarcts, and orthostatic cerebral ischemia. Each patient suffered from more than one symptom complex. The cases comprising this series can be divided into two groups: those considered to be a high risk for a posterior circulation infarct but not disabled by their symptoms or deficits (eight patients), and those moderately or severely disabled at the time of admission (eight patients). Pertinent data regarding each of the patients in these two groups are summarized in Tables 1 and 2.

It is appropriate to review briefly the following three cases, which illustrate major

J. Neurosurg. / Volume 48 / June, 1978

916
Occipital to PICA bypass

### Table 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex</th>
<th>Symptom Complex</th>
<th>Trial AC</th>
<th>Lesion in Intracranial Vertebral Artery</th>
<th>Graft Patency</th>
<th>Operative Complications</th>
<th>Clinical Result†</th>
<th>Residual Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>55, M</td>
<td>TIA, POCI</td>
<td>yes</td>
<td>stenosis stenosis</td>
<td>yes</td>
<td>none</td>
<td>exc</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>69,</td>
<td>TIA, POCI</td>
<td>no</td>
<td>congenital occlusion</td>
<td>yes</td>
<td>small cerebellar infarct</td>
<td>exc</td>
<td>ataxia</td>
</tr>
<tr>
<td>6</td>
<td>61, M</td>
<td>TIA, infarct</td>
<td>yes</td>
<td>occlusion occlusion</td>
<td>yes</td>
<td>aseptic meningitis</td>
<td>exc</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>59, M</td>
<td>TIA</td>
<td>yes</td>
<td>stenosis congenital variant</td>
<td>yes</td>
<td>bleeding in neck muscles; spasm of graft; excellent flow on repeat angiogram, later flow excellent</td>
<td>exc</td>
<td>none</td>
</tr>
<tr>
<td>10</td>
<td>39, M</td>
<td>TIA</td>
<td>yes</td>
<td>stenosis stenosis</td>
<td>yes</td>
<td>poor graft flow on initial postop angiogram, later flow excellent</td>
<td>good</td>
<td>none</td>
</tr>
<tr>
<td>11</td>
<td>47, M</td>
<td>TIA, POCI</td>
<td>yes</td>
<td>stenosis stenosis</td>
<td>yes</td>
<td>none</td>
<td>exc</td>
<td>minimal memory dysfunction</td>
</tr>
<tr>
<td>12</td>
<td>47, M</td>
<td>TIA (rt lat. medullary)</td>
<td>yes</td>
<td>normal occlusion</td>
<td>yes</td>
<td>TIA's unchanged</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>13</td>
<td>35, M</td>
<td>TIA</td>
<td>yes</td>
<td>occlusion small</td>
<td>no</td>
<td>graft thrombosis</td>
<td>unchanged</td>
<td>none</td>
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</table>

*TIA = transient ischemic attack; POCI = primary orthostatic cerebral ischemia; AC = anticoagulants.
†Exc = normal or near-normal function; good = improved, not employed.
‡Small vertebral artery that ended in posterior inferior cerebellar artery and did not communicate with the basilar artery.

complications we have encountered. Additional cases, with ischemic symptomatology and hospital courses more typical of the two groups, have been described previously.14

**Illustrative Cases**

**Case 6**

This 61-year-old man was re-evaluated at the Mayo Clinic in May, 1976, with a primary complaint of syncope and vertigo precipitated by neck movement and positional changes. He had been seen at the Mayo Clinic in 1973 for a lateral medullary infarction from which he recovered with a minimal dissociated sensory deficit involving the left side of the body and the right side of the face. Mild hypertension had been controlled with 1 to 2 Dyazide tablets (triamterene and hydrochlorothiazide) daily and atrial fibrillation with Lanoxin (digoxin), 0.125 mg daily.

**Examination.** Positive findings on readmission included a Horner’s syndrome on the left, nystagmus, incoordination in the left upper extremity, and the dissociated sensory loss described above. Blood pressure was 150/60 standing, and 160/70 lying.

Angiography demonstrated occlusion of the right and the left vertebral arteries just proximal to their entry into the calvaria. Almost all of the posterior circulation was supplied via reflux of contrast medium into the basilar artery from a large right posterior communicating artery. The basilar artery then supplied the cerebellar vessels in a retrograde fashion.

**Operation.** A right occipital to left PICA anastomosis was performed on May 25, 1976. The right occipital artery was selected because it was the larger of the two vessels, and the left PICA because it had filled in a retrograde fashion from the basilar artery on the preoperative angiograms and the communication between the right PICA and the basilar system was not established.

**Postoperative Course.** The night following surgery, this patient was able to turn and move without reproducing his neurological symptomatology. He made a good recovery except for a major febrile response, persisting for 1 week, related to a sterile arachnoiditis
Table 2
Group 2: Patients disabled before surgery

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Symptom Complex</th>
<th>Trial AC Preop</th>
<th>Lesion in Intracranial Vertebral Artery</th>
<th>Graft Patency</th>
<th>Operative Complications</th>
<th>Clinical Result†</th>
<th>Residual Deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55</td>
<td>M</td>
<td>TIA, infarcts, progressing stroke</td>
<td>yes</td>
<td>congenital stenosis</td>
<td>yes</td>
<td>pulmonary</td>
<td>good</td>
<td>ataxia</td>
</tr>
<tr>
<td>2</td>
<td>54</td>
<td>M</td>
<td>TIA, infarct, POCI</td>
<td>yes</td>
<td>congenital occlusion</td>
<td>yes</td>
<td>pulmonary</td>
<td>exc§</td>
<td>minimal ataxia, ataxia, unilateral hearing loss</td>
</tr>
<tr>
<td>3</td>
<td>67</td>
<td>M</td>
<td>TIA, infarct, POCI</td>
<td>yes</td>
<td>occlusion occlusion</td>
<td>yes</td>
<td>hemispheric subdural hematoma</td>
<td>good</td>
<td>ataxia, visual blurring</td>
</tr>
<tr>
<td>4</td>
<td>66</td>
<td>F</td>
<td>infarct, progressing stroke</td>
<td>yes</td>
<td>occlusion occlusion</td>
<td>yes</td>
<td>poor wound healing</td>
<td>exc</td>
<td>ataxia</td>
</tr>
<tr>
<td>5</td>
<td>52</td>
<td>M</td>
<td>TIA, infarct, POCI</td>
<td>yes</td>
<td>occlusion occlusion</td>
<td>yes</td>
<td>postop epidural hematoma</td>
<td>good</td>
<td>ataxia</td>
</tr>
<tr>
<td>6</td>
<td>62</td>
<td>M</td>
<td>infarct, POCI</td>
<td>no</td>
<td>occlusion occlusion</td>
<td>yes</td>
<td>transient increased ataxia</td>
<td>good</td>
<td>ataxia</td>
</tr>
<tr>
<td>7</td>
<td>60</td>
<td>F</td>
<td>TIA, infarct, POCI</td>
<td>yes</td>
<td>occlusion occlusion</td>
<td>yes</td>
<td>none</td>
<td>exc</td>
<td>ataxia</td>
</tr>
<tr>
<td>8</td>
<td>48</td>
<td>M</td>
<td>TIA, infarct, progressing stroke</td>
<td>yes</td>
<td>congenital occlusion variant</td>
<td>yes</td>
<td>pulmonary</td>
<td>good</td>
<td>lower cranial nerve palsies, ataxia</td>
</tr>
</tbody>
</table>

*TIA = transient ischemic attack; POCI = primary orthostatic cerebral ischemia; AC = anticoagulants.
†Exc = normal or near normal function; good = improved, not employed.
‡Small vertebral artery that ends in posterior inferior cerebellar artery and does not communicate with basilar artery.
§Died from myocardial infarction 18 months following operation.

due to blood in the lumbar subarachnoid space. We attributed this accumulation of blood to our failure to place cottonoid pledges around the spinal cord and to seal this space from the blood in the operative field.

Angiography 2 weeks after surgery demonstrated patency of the bypass graft, and repeat angiography performed on November 2, 1976, 6 months after the operative procedure, demonstrated enlargement of the bypass graft with filling of the left vertebral artery and proximal basilar artery from the bypass graft (Fig. 1). The distal basilar artery continued to fill retrograde from the carotid system.

Case 5

This 65-year-old man was first seen at the Mayo Clinic in March, 1976. He had a 6-month history of "dizzy spells" accompanied by syncope. These always occurred when he was standing or active; he could abort an attack by sitting or lying down. He was known to be hypertensive and was on antihypertensive medication. Two months before admission to the hospital the patient had an episode of severe vertigo and ataxia. Subsequent to that date he had persistent ataxia that was progressive with walking and limited his ambulatory distance to about 100 feet. The patient's wife reported some personality disorder and a definite change in his behavior pattern.

Examination. Brachial blood pressures at the time of admission were 140/100 bilaterally with no orthostatic drop. Neurological examination indicated a decrease in alternate motion rate in the right hand as compared to the left and mild truncal ataxia. Retinal artery pressures were equal.

Angiography demonstrated a 90% stenosis of the left internal carotid artery at its origin from the common carotid artery, and a 50%
Occipital to PICA bypass

FIG. 1. Case 6. Angiogram 6 months after bypass surgery. The bypass graft is the primary source of blood flow to the proximal vertebrobasilar system.

stenosis of the origin of the right internal carotid artery at its origin from the common carotid artery. The left vertebral artery was found to end in the PICA. The right vertebral artery was occluded to the level of C-2 and then slowly filled from muscular arterial collaterals, originating in part from the occipital artery.

Operations. The patient underwent left carotid endarterectomy on April 9, 1976; he made an uncomplicated postoperative recovery. A right occipital to PICA bypass procedure was performed on May 13, 1976; upon awakening, the patient was confused and disoriented. Detailed neurological testing was therefore unreliable; however, it was noted that the patient had marked dysmetria, ataxia of the right upper extremity, and a mild truncal ataxia. Blood pressure was recorded as 170/110. The confusion and disorientation persisted along with the ataxia.

Postoperative Course. Angiograms performed on May 18, 1976, indicated that the bypass graft was functional with filling of the PICA, the right vertebral artery, and the basilar artery as far distal as the superior cerebellar artery (Fig. 2). A transurethral resection was necessary on June 1, 1976, because of urinary retention. At discharge on June 11, 1976, the patient was alert and oriented; truncal ataxia had resolved, and a mild right upper extremity ataxia continued.

A follow-up telephone call on February 17, 1977, indicated that the patient had resumed normal activities. He continued to have a mild problem with memory, but this was not major in his judgment. He preferred to swallow liquids and soft foods and avoided solid foods. He complained of a “tightness” in his head but reported that the “dizziness” experienced before surgery was relieved and that he had no problems walking. He enjoys driving the family car for short distances and is leading a normal social life.

Comment

The cause of the confusion and disorientation that followed the bypass procedure in Case 5 is unknown. We had no evidence of air
emboli during the operation and we assume these symptoms reflected the stress of the operation in a man with marginal cerebral function due to diffuse vascular occlusive disease. The transient right cerebellar findings were probably related to our selection of the right occipital artery as a donor vessel in that it already was participating in collateral supply to the posterior circulation. Subsequent to this case, as in Case 6, contralateral donor vessels were often selected on the basis of collateral flow and vessel diameter.

Case 9

This 52-year-old man was evaluated in September, 1976, for complaints of visual blurring and difficulty in walking. Dizziness, vertigo, and dysarthria had begun and persisted for 6 months before his admission. Antihypertensive therapy had been instituted in April, 1976, when the patient's blood pressure was found to be 200/100; marked problems with ambulation subsequently developed. On one occasion he had the sudden onset of vertigo, diplopia, and inability to walk. The diplopia and vertigo eventually subsided, but the patient was left with a persistent degree of ataxia. There were no pyramidal tract signs.

Cerebration and memory gradually deteriorated from April to September, 1976. This was followed by progressive loss in visual acuity with blurring of vision, ataxia, dysarthria, dysphasia, and postural syncope. Ambulatory distance was limited to 50 feet, and then only with assistance.

Examination. Angiography on September 22, 1976, demonstrated multiple occlusions: the left vertebral artery just distal to its origin, the right vertebral artery just proximal to its entrance into the dura, and the left posterior cerebral artery with retrograde filling from the middle cerebral vessels into the distal portions of that vessel. The basilar artery filled retrograde from the right posterior cerebral artery, which in turn filled from collaterals over the surface of the brain as there was no large posterior communicating vessel. There was filling of the left PICA, but no filling of the right PICA. There
was a 75% to 80% narrowing of the left external carotid artery.

Operations. Because of the stenosis of the left external carotid artery the right occipital artery was selected as a donor vessel. The left PICA was used as a recipient vessel. This surgery was performed on September 27, 1976. Postoperatively, the patient was essentially unchanged from his state before surgery, but over the following 5 days there was a gradual deterioration in his level of consciousness, dimness of vision, and syncope on standing. Brachial blood pressure was found to be 180/100 standing, 140/80 sitting. Angiography on October 5, 1976, demonstrated patency of the bypass graft but with severe spasm involving the entire occipital artery in the region of the operation (Fig. 3). A computerized tomography scan on October 6 showed a large epidural hematoma at the site of operation; the wound was immediately re-explored and the hematoma evacuated. A bleeding point in the occipital artery remote from the site of anastomosis was repaired with a clip-graft.

Postoperative Course. Neurological function gradually improved after the second operation. At the time of dismissal in November, 1976, the patient's visual acuity was considerably improved, he had no orthostatic complaints, and he was able to walk unlimited distances with a walker. There were no focal neurological signs or symptoms related to the pyramidal tract. He had considerable truncal and extremity ataxia. Postoperative angiography demonstrated marked enlargement of the occipital artery with good opacification of the entire vertebrobasilar system through the bypass graft (Fig. 4).

The patient returned in July, 1977, with complaints of ataxia and visual blurring that had developed over the past 6 months. There was no change in the neurological examination. Repeat angiography demonstrated good patency of the graft (Fig. 5).

Comment

Our failure to recognize the epidural hematoma earlier in Case 9 is inexcusable,
and underscores the necessity of remembering "the basics" and not ascribing all such deficits to hemodynamic changes, even in patients with severe vascular disease. The severe spasm in the occipital artery on the first postoperative angiogram, at a time when that vessel was embedded in a thick blood clot, suggests that intracranial vessels are not unique in their response to this irritant. The bleeding point that gave rise to this hematoma was difficult to repair with sutures as the wall of the vessel was friable and the actual source was difficult to identify. The clip-graft sealed this bleeding point promptly without distortion or narrowing of the donor graft.

**Surgical Technique**

The patient is placed in the sitting position with the head flexed anteriorly and secured in a pinion headholder. A hockey-stick incision is made (Fig. 6 A) and initially the midline avascular plane is identified and the muscle-ture swept unilaterally from the arch of C-1 and the occiput. The cutting current is not used for the scalp incision but is useful to reflect the deep neck muscles from the occiput as far laterally as the mastoid process (Fig. 6 B). The occipital artery is identified in its muscular plane and is best located by palpation in the mastoid groove just posterior and medial to the mastoid process (Fig. 6 C). This vessel is then dissected free from the surrounding tissue using small blunt scissors. Small branching vessels are coagulated with the bipolar coagulator before their division. This is perhaps the most difficult part of the operation as the vessel is intimately adherent to the surrounding tissue and is much more difficult to isolate than is the superficial temporal artery (Fig. 6 D and E). It is surrounded by a venous plexus and distally joins a fascial sheath shared by the occipital nerve. This vessel is followed to its point of entrance into this muscular bed at the mastoid groove. In its transplanted course it lies at the base of the occiput and follows a straight path from the mastoid groove to the point of anastomosis. It is important to mobilize this vessel as far proximally as possible in order to obtain
Occipital to PICA bypass

Fig. 5. Case 9. Angiogram 9 months after surgery.

Adequate length for the graft. Proximal dissection often permits the resection of the distal 1 to 2 cm of the graft.

A small unilateral suboccipital craniectomy is effected, with a unilateral resection of the arch of C-1 (Fig. 6 F). The dura is then opened in a linear fashion and the margins sutured to adjacent tissue (Fig. 6 G). The medullary loop of the PICA is identified as this vessel passes around the brain stem on its course to the vermis (Fig. 6 H). A small rubber dam is then temporarily placed deep to this artery and the vessel is elevated by suturing the superior end of the rubber dam to the margin of the bone and the inferior end of the dam to muscle or reflected dura (Figs. 6 I and 7). Miniature Mayfield clips are placed on either side of the area selected for arteriotomy. A small linear incision is made in the PICA with a broken razor blade on an appropriate holder. The arteriotomy is extended in both directions with small microscissors. The donor vessel (previously prepared for the anastomosis by resection of excess length, removing excessive soft tissue, and fish-mouthing the end), is sewn to the apex of the arteriotomy with a double-armed 9-0 monofilament nylon suture (Fig. 6 J and K). This initial suture is an important one and is placed in both vessels from the inside to the outside. The remaining portion of the vessel is then anastomosed in a routine fashion by techniques described previously (Fig. 6 L and M). Interrupted sutures are used throughout the closure, 9-0 monofilament nylon is preferred to 10-0 monofilament nylon as the wall of the occipital artery is thicker than the temporal artery and tends to bend the smaller needles provided for 10-0 monofilament suture. Flow is restored by removing the clips on the recipient artery initially and the clip on the donor vessel last. Small bleeding points, if they occur, usually cease within a few minutes from light pressure applied using Gelfoam. However, on occasion it is necessary to place an additional suture if the bleeding does not terminate with light pressure. The temporary rubber dam is removed and a dural graft is.
Fig. 6. For legend see next page.
then sewn into place. Dural closure is facilitated by a separate incision in the lateral wall of the dura for the entrance of the artery into the subarachnoid space (Fig. 7). Nevertheless, a completely water-tight closure is not possible because of the necessity of allowing adequate room for the occipital artery as it passes through the dural opening. Accordingly, a very tight muscle closure is necessary and this in turn is facilitated by retaining a muscular cuff in the transverse portion of the wound and taking the patient out of the “flexed position.” Sutures are left in place for 2 weeks. The transplanted oc-

![Fig. 6. Sketch of surgical procedure. A: A hockey-stick skin incision is made extending above the level of superior nuchal line. B: Deep neck muscles are cut from their insertion, leaving a cuff of tissue for closure. C: The occipital artery is identified in the mastoid groove posterior and superior to the mastoid process. D: The occipital artery is dissected free from adjacent tissue. The vessel lies deep to splenius capitis and longissimus capitis. The dissection is simplified by maintaining this tissue plane. E: The occipital artery, lying free in the muscle bed from which it was dissected. F: A small unilateral suboccipital craniectomy is made, with a unilateral resection of arch of C-1. G: The dura is opened with a straight incision. H: The dura is sutured to the margins of the craniectomy, and the medullary loop of the PICA identified. I: The PICA is elevated by means of a temporary rubber dam. J: The PICA is opened with a linear incision, and the occipital artery fish-mouthed. K and L: Anastomosis is performed with interrupted 9-0 monofilament nylon sutures. M: The completed anastomosis. N: The transplanted course of occipital artery.](image-url)
patients in this group had a progressing deficit and this was arrested in all three patients. There was a major gradual improvement in neurological function in seven of the eight patients, but only three were able to achieve essentially normal function. Symptoms of orthostatic ischemia were relieved. Truncal ataxia and cranial nerve palsy from small brain-stem infarcts limited function in five patients in Group 2. All individuals were able to feed and care for themselves, and were, in the judgment of the family, functioning as useful and important members of their families with normal or good mental function.

Graft Patency

Postoperative angiography demonstrated graft patency in 15 of the 16 cases. Two cases initially considered to be physiologically occluded were subsequently demonstrated to be widely patent on arteriography 6 to 8 weeks later.

Graft Flow

The bypass graft carried the major portion of flow to the posterior circulation in 10 of the 11 cases in which the recipient vessel was the parent trunk of the PICA. In five cases in which, because of anatomical variations, it was necessary to use a branch of the PICA, flow was limited to the PICA and its branches. Three patients underwent repeat angiograms 3 months to 1 year postoperatively. Flow was increased in each of these three cases as compared to the initial postoperative angiogram.

Complications

The complications are detailed in Tables 1 and 2. Only one of these complications resulted in a new fixed neurological deficit (a unilateral hearing loss not present before operation). Two patients had a transient increase in ataxia following surgery. One patient developed bilateral subdural air collections that required burr holes for release 36 hours after the operation. Reoperation was necessary in one patient for a postoperative epidural hematoma. Pulmonary complications were invariably related to lower cranial nerve palsies present before surgery and responded appropriately to standard measures of respiratory therapy.
Occipital to PICA bypass

Discussion

Pathophysiology of Vertebrobasilar Disease

The symptom complexes and vascular pathology for which these patients were operated on are well known. They have been discussed previously in a communication directed to that subject. However, it is appropriate to reemphasize that these patients were not operated on for stenotic lesions at the origins of the vertebral arteries. Infarctions from lesions in these areas are uncommon. The patients reported here had either bilateral lesions of the intracranial portions of the vertebral arteries (occlusion or high-grade stenosis); unilateral stenosis or occlusion in combination with a congenital variant of the opposite vertebral artery; or extracranial vertebral artery disease progressing as high as C-2 and distal to the point of possible flow through collaterals in the neck.

The patterns of occlusive disease and the pathogenesis of symptomaticity in the vertebrobasilar system are different from those in the carotid system. Emboli are thought to be the chief cause of occlusion of the major branches of the internal carotid artery. This does not seem to be the case in the vertebrobasilar system where thrombosis on a preexisting stenosis has been found to be the cause of 90% of basilar artery occlusions and 70% of the intracranial vertebral artery occlusions. Occlusion of a preexisting stenosis has been reported to be uncommon in the extracranial vertebral artery but common in the intracranial portion of that vessel. Occlusions of the posterior cerebral artery are, however, generally embolic in origin and in this regard, these vessels, the terminal branches of the basilar artery and the most likely recipients for emboli, are similar to the major branches of the internal carotid artery. Nevertheless, although atherosclerotic plaques are diffusely distributed throughout the vertebral arteries, in contrast to the carotid system where plaques are often localized near the bifurcation of that vessel and in the siphon area, ulceration of vertebral artery plaques with secondary embolization is uncommon.

Brain-stem infarction also occurs from occlusion of small penetrating branches that arise from the vertebral or basilar arteries. The relative frequency of this cause of brain-stem ischemia, in comparison with occlusion of larger vessels, has not been established. Furthermore, the cause of transient ischemic attacks in the posterior circulation is still unresolved. Two major theories have been proposed: namely, emboli from ulcerated plaques of large vessels to more distal arteries and hemodynamic changes distal to the site of stenosis or occlusion of a large or small penetrating artery. The hemodynamic changes might result from variations in systemic perfusion pressure or from failure of collateral flow.

Analysis of Results

Ataxia was the most common persisting neurological deficit in our patients. No patients had a new brain-stem infarction or an extension of an old infarction after the postoperative period. Two patients have continued to have TIA's but have not sustained an infarction. A fixed neurological deficit was not altered by surgery, but a progressing stroke was arrested and considerable function was restored in three patients with this diagnosis who underwent surgery. Orthostatic symptoms of light-headedness, syncope, and visual blurring were relieved by operation in most patients.

Analysis of Complications

Our primary problems in connection with this operative procedure have been related to the marginal neurological status of these patients before surgery. Respiratory complications have been frequent and usually have resulted from previously impaired cranial nerve function that has made swallowing and handling of secretions difficult for these individuals. Accordingly, we have followed the custom of leaving a nasotracheal tube in place in all individuals in whom there has been impairment in the function of the ninth and tenth cranial nerves before surgery. Surgery with the patient in the sitting position presents risks related to air emboli, hypotension, and convexity subdural collections of fluid or air. However, it is our judgment that these risks are far outweighed by the exposure achieved from this position. All patients should receive adequate blood volume replacement on a unit for unit basis which helps to prevent air emboli by maintaining a high venous pressure and also to avoid hypotension. These patients have areas
in the brain in which autoregulation is no longer preserved, and they are extraordinarily vulnerable to fluctuations in perfusion pressure and cardiac output. Accordingly, it is imperative to maintain an adequate perfusion pressure throughout the operation.

Complications such as epidural hematoma and aseptic meningitis related to blood in the subarachnoid space are not unique to this type of procedure and can be prevented with appropriate measures.

Role of Operation

It is premature to determine at this time the role of this operative procedure. Our results have been modestly encouraging but the series is still too small to form any firm conclusions. It is our experience that these patients represent only a small portion of individuals suffering from posterior circulation ischemic attacks. The role of angiography in the evaluation of such patients has been reviewed by Caplan and Rosenbaum. These authors have indicated that bilateral intracranial occlusions of the vertebral arteries often presented a clinical picture of ataxia, visual disturbances, and altered mentation which generally terminated fatally. We agree with the conclusions of these authors that patients with this clinical triad should be considered for angiography. Additional indications might include orthostatic related ischemic symptoms without a postural decrease in systemic blood pressure, progressing ischemia in the brain stem or cerebellum, and frequent TIA's not altered by antiocoagulation therapy. In our experience, angiograms in patients with a single TIA's involving the posterior circulation are often normal or show only minimal large vessel disease.

References


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