Vascular compression of the optic nerves relieved by anastomosis of carotid artery to jugular vein

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The intracranial segment of the optic nerve, crossed near the chiasma by the anterior cerebral artery and fixed anteriorly at its entry into the optic foramen, is particularly vulnerable to compression by vascular deformities of the internal carotid artery or its branches.

Aneurysms arising in this region may be surgically inaccessible directly, because of their size or because they arise from that segment of the carotid between its exit from the cavernous sinus and the anterior clinoid process. Expansion of these aneurysms results in pain and associated blindness or visual field defect due to optic nerve compression.

The orthodox surgery for such aneurysms is carotid ligation. This is a step that cannot be retraced and, if it fails in its objective, it is difficult to justify the very disabling complications.

As an alternative to carotid ligation the blood flow through the carotid artery can be reduced by creating a small arteriovenous shunt between the carotid artery and the jugular vein in the neck. This

technique has been used since 1966 and there have been no complications from the shunting operation in its use for space occupying aneurysms and no long-term effects on the cardiovascular system in the form of alteration of exercise tolerance, cardiac size, or electrocardiographic changes.

This is a report on two cases of aneurysm and one of compression of the optic nerve by a dilated and tortuous ophthalmic artery in a patient suffering from acromegaly.

CASE REPORTS

CASE 1 A 45-year-old woman who had suffered from dizzy turns and fainting attacks for two years experienced a sudden headache one week before her admission to hospital on 10 June 1966; lumbar puncture at the time of admission revealed xanthochromic fluid. The headache persisted and it was noted that she had no useful vision in the left eye.

Bilateral carotid angiography on 20 June 1966 (Fig. 1a and b) showed that the right side was normal; there was a

FIG. 1a and b Case 1. Anteroposterior and lateral angiograms showing the aneurysm arising near the anterior clinoid process.

spontaneous cross-flow of contrast into the left pericallosal artery. On the left side a large irregular aneurysm arose from the upper limb of the left carotid siphon just distal to the anterior clinoid process. This projected upwards and a little medially and the proximal part of the anterior cerebral artery was slightly elevated as it passed across the sac. The adjacent part of the carotid artery was narrow, particularly beyond the aneurysm, and the proximal parts of the anterior and middle cerebral arteries were also attenuated. There was only poor filling of the pericallosal artery, which was normal in position and outline.

At operation on 21 June 1966, a left frontal craniotomy exposed the chiasmatic region. A very large aneurysm 2 cm in length was found beneath the left optic nerve and chiasma, bulging these upwards. The internal carotid artery was very thin and displaced backwards. The anterior clinoid process was removed using a small gouge and, in view of the absence of useful vision in the left eye, the lateral half of the left optic nerve was divided to display the origin of the aneurysm. This was a sessile origin from the carotid artery at the point where the carotid emerged from the cavernous sinus. Temporary occlusion of the exposed common carotid artery did not alter the size of the aneurysm nor did it stop or ease haemorrhage when this occurred later, during dissection of the aneurysm. Three times the blood pressure was reduced to 40 mm systolic using Arfonad and a Mayfield clip applied across the neck of the aneurysm, but as the pressure rose the clip slipped off. The neck of the aneurysm was wrapped around with gauze and the operation concluded. Post-operatively the convalescence was uneventful apart from a generalized epileptic seizure on the ninth post-operative day. The patient did not comment on any change in vision of the left eye, there was still only a vague appreciation of shapes and the division of the outer half of the optic nerve had made no appreciable difference to her.

Consideration was given to the possibility of ligation of the common carotid artery, but this supplied the dominant hemisphere and the narrowing of the internal carotid distal to the aneurysm made the risk of thrombosis considerable, so ligation was considered inadvisable.

It was decided to try to shunt part of the blood flow into the jugular system, thereby reducing the diastolic pressure in the aneurysm and the carotid artery blood flow distal to the shunt.

A small side-to-side anastomosis of the common carotid artery to the jugular vein was made under magnification, diameter 5 mm approximately. The following day sufficient sight had returned to the left eye for reading but with loss of the major part of the nasal field of vision, the result of the section of the optic nerve at the first operation. She has remained well since operation and has returned to her original activities, although occasional dizzy turns and syncopal attacks occur, but these had been present for some years before her surgery. Her blood pressure is 140/85 mm Hg, regular chest radiographs show no increase in heart size, and the electrocardiographs remain normal. Cerebral angiograms were repeated on 14 February 1967 and 2 October 1967, and there was no change in the size of the aneurysm, although it was commented that the upper margin had be-

come more rounded. Visual acuity in October 1968: 6/6 left eye J1 reading types. The visual fields on 4 November 1968 showed the nasal field defect (Fig. 2). This was repeated on 12 February 1969 and showed no change.

FIG. 2 Case 1. Visual field on 4 November 1968. Perimetry. Visual acuity 6/6 and field defect due to surgical lesion of the optic nerve. White object 1/330.

CASE 2 A 44-year-old woman experienced a sudden flash of pain across the top of her head accompanied by a sudden loss of vision in the left eye on 29 June 1968. A lumbar puncture was not done at this time. She was admitted to hospital and cerebral angiography was performed on 5 July 1968 (Fig. 3a, b), and was reported on as follows: 'There is a large aneurysm, ovoid, and about one inch long arising from the proximal part of the left anterior cerebral artery at about its junction with the anterior communicating artery. This fills only from the left and it projects downwards and forwards.'

Her vision in the left eye was very poor by her description, and she was unable to see because of a haze across the eye. On 19 July 1968 visual acuity was 3/60 left, the right eye 6/9-2, fields: right Bjerrum normal, left showed a gross nasal field defect (Fig. 4), perimeter (Fig. 5).

It was felt that exploration of the aneurysm would be a formidable undertaking and the patient herself was opposed to intracranial operation. Carotid ligation was considered, but with such a space occupying aneurysm it was felt that ligation could lead to infarction of the dominant hemisphere.

At operation on 11 July 1968, a small arteriovenous shunt was made using magnification, approximately 3 mm diameter between the common carotid artery and the jugular vein in the neck. The anastomosis was made side to side using 7-0 silk on atraumatic needle.

The following day vision was improved sufficiently for her to read. On 22 July 1968 visual acuity was 6/12 left eye and the visual field defect occupied only a small area in the lower quadrant commencing at 15 degrees.

The visual field studies were repeated on 19 February 1969 (Fig. 6) and perimetry (Fig. 7), the visual acuity being 6/9. She has returned to her work and the visual defect causes her no inconvenience.

CASE 3 A male patient aged 45 years presented on 21 February 1968 with a history of headaches for 15 years mainly over the left eye and with evident features of acromegaly. The pituitary fossa was enlarged but there was no evidence of alteration of visual acuity or visual

FIG. 3a and b Angiography on case 2. Large aneurysm probably arising from anterior cerebral artery.

FIG. 4. Case 2. Pre-operative visual acuity 3/60 and visual field Bjerrum screen white object 1/2000 and 4/2000.

FIG. 5. Case 2. Perimetry 10 July 1968, white object 1/330.

FIG. 6. Post-operative case 2. Bjerrum screen 19 February 1969. 1/2000 white. A very similar field defect was obtained on 22 July 1968.

FIG. 7. Case 2. Perimetry 19 February 1969, 1/330. Visual acuity now 6/9.

field changes. Growth hormone estimations showed a resting level 20 $\mu\text{g/ml}$. (normal range 1.0 to 8.0 $\mu\text{g/ml}$.) and with hypoglycaemia the level rose to 37 $\mu\text{g/ml}$. and suppression with hyperglycaemia down to a mid-normal range.

Air studies and angiography showed no evidence of extension of the tumour above the fossa, so cryogenic destruction of part of the gland was performed by trans-sphenoidal route on 9 May 1968.

This resulted in a profound improvement in the patient's physical appearance and reduction of serum growth hormone to normal levels. Maintenance doses of prednisone were necessary. Headache persisted and for this reason a course of radiotherapy was given but with little improvement.

On 20 January 1969 his admission to hospital was arranged as an emergency. Four days previously he had noticed 'spots' before the right eye followed by a severe headache. The next morning his central vision in the right eye was markedly decreased. Visual fields and acuity were recorded on 21 January 1969 (Fig. 8).

FIG. 8. Case 3. Pre-operative field defect 21 January 1969, right eye 1/2000 and 5/2000. White object (Lumiwand) acuity right 6/9 with some errors.

On the 22nd air studies were performed and these showed good filling of the front end of the third ventricle, which was quite normal. On the 23rd right carotid angiogram was reported: 'considerable tortuosity with some dilation of the right internal carotid artery. The carotid siphon is normally placed. The artery passes abruptly laterally and upwards from the siphon to its bifurcation and there is slight elevation of the proximal portion of the horizontal trunk of anterior cerebral artery.'

The clinical signs and the visual field studies were so strongly suggestive of optic nerve compression that it was decided to explore the region of the optic nerve and chiasma, in spite of negative air studies. At operation on 24 January 1969, right frontal craniotomy was carried out using a large Scoville trephine. There was no tumour extension beyond the sella, but the right optic nerve was seen to be lifted upwards and visibly thinned by a large tortuous ophthalmic artery which could be seen arising lateral to the nerve, passing under it, and appearing as a loop medially before passing forwards beneath the nerve (Fig. 9). The medial part of the optic nerve appeared thin and compressed.

Consideration was given to uncapping the nerve in its foramen, but the compression was proximal to the foramen and uncapping did not offer much promise.

FIG. 9. Sketch of operation showing optic chiasma and on the right side the optic nerve lifted upwards by enlarged tortuous ophthalmic artery.

Post-operatively the possibility of relieving the compression by making an arteriovenous shunt was discussed with the patient, and he decided to try the experiment.

The shunt, approximately 3 mm diameter, was made under magnification using local anaesthetic. Within an hour of operation the patient noticed clearing of the haze over his vision and he could read large print the following day.

Post-operative fields showed contraction of the scotoma and these were repeated on 12 February 1969 (Fig. 10). Visual acuity had improved from 6/9 to 6/6. He has remained well since operation and has returned to work.

FIG. 10. Post-operative visual field changes. Small nasal scotoma relative only with white 1/2000. Visual acuity 6/6.

DISCUSSION

Originally the arteriovenous shunt was conceived as a means of providing a temporary diversion of blood flow to prepare the patient for more definitive surgery. The main advantage, however, appears to be in the space occupying vascular lesions by reducing the tension within cerebral aneurysm. Where pressure effects were the presenting symptoms (facial pain, oculomotor nerve palsy, and compression of the optic nerves) there was improvement.

Similar arteriovenous shunts were tried on a number of cases where subarachnoid haemorrhage was caused by the aneurysm, these patients were too ill to withstand intracranial surgery or were elderly

or hypertensive or suffered from multiple aneurysms. The results in these cases were on the whole disappointing, with the possible exception of the hypertensive group. The series is, however, too small to form a useful basis for comparison with conservatively treated cases, but a review of all cases treated by arteriovenous shunting is planned in the future.

SUMMARY

A technique for relief of pressure effects of intracranial aneurysms is described. This operation involves the construction of a 3 mm arteriovenous shunt between common carotid artery and internal jugular vein in the neck. The shunt has caused no long-term complication in the cases operated upon in 1966 and the improvement in the symptoms due to optic nerve compression has been immediate and promises to be lasting. The procedure is not recommended for treatment of subarachnoid haemorrhage.

Dr. W. J. Burke (neurologist, St. Vincent's Hospital) assisted in making the decision for operation in these cases. Dr. Les Lazarus (endocrinologist and director of the Garvan Institute of Medical Research) conducted the endocrinological management of the acromegalic patient. Dr. Peter Cahill (neuro-radiologist, St. Vincent's Hospital)

reported on the angiograms. Sister Mary Regis (Department of Medical Illustration, St. Vincent's Hospital) and Sister Mary Imelda (Department of Medical Illustration, Lewisham Hospital) were responsible for the photographs.

ADDENDUM

CASE 1 On 25 May 1969 this patient woke with a slight headache which later disappeared. After lunch she was bent over a low bath tub washing her hair under the spout of a bath heater. Her husband heard her call out and when he reached her she was gripping her head and crying with severe headache.

She collapsed and he found it necessary to use mouth to mouth resuscitation until an ambulance arrived. When she reached hospital she was unconscious, but rubbing the left side of her head with her left hand. There was no purposeful movement in the right limbs.

Lumbar puncture revealed heavily blood-stained fluid. She died the following afternoon.

Necropsy confirmed that death was due to subarachnoid haemorrhage from the carotid aneurysm.

It is possible that the awkward position she had adopted to wash her hair had occluded the arteriovenous shunt temporarily and caused a sudden rise in blood pressure and flow in the aneurysm resulting in rupture.

However, there must be both intracranial and extracranial factors responsible for aneurysmal rupture and although the tension within an aneurysm can be reduced by the arteriovenous shunt, this does not necessarily protect against aneurysmal rupture.