Blindness due to aneurysm of anterior communicating artery

With recovery following carotid ligation

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Aneurysms of the anterior communicating artery are an uncommon cause of visual, olfactory, mental, endocrine, and sphincteric disturbance. In this paper is presented a patient with a giant anterior communicating aneurysm, causing headache, severe constriction of visual fields, and diminution of visual acuity. Transient diabetes insipidus, mental change, and urinary retention were also noted. Improvement occurred after ligation of the right common carotid artery.

Case history

A 54-year-old woman presented in July, 1965, with a 5-week history of blurred vision and transient pain in the right eye. She could just count fingers held 6 in. from the right eye and the visual acuity in the left eye was 6/36. Both optic discs appeared normal but the right pupil reacted sluggishly to light, accommodation, and convergence. Bjerrum screening showed a normal visual field for the left eye but the right eye showed complete inferior nasal quadrant and incomplete inferior temporal quadrant defects (Fig. 1). No other neurological abnormality was found. In particular there was no olfactory, mental, endocrine, or hypothalamic disturbance. The blood pressure was normal (150/95) and no cranial bruits were heard.

FIG. 1 *Inferior nasal and temporal quadrant defects in the right eye (July, 1965)*

Radiographs of the skull were normal. The blood Wassermann and Kahn reactions were negative. A right carotid angiogram (Dr. G. F. Swann) showed a large aneurysm of the anterior communicating artery with good filling on cross-compression (Fig. 2). Surgery was not thought advisable at that stage.

FIG. 2 Right carotid angiogram, showing aneurysm of anterior communicating artery (July, 1965)

September, 1967 For 3 weeks the patient had noticed pain and blurred vision in the left eye, but thought the sight of the right eye had not changed. Examination revealed optic atrophy in the right fundus with a visual acuity of less than 6/60 (6/9 corrected). The visual acuity in the left eye was also less than 6/60 and could not be improved. Considerable reduction in both visual fields had occurred (Figs 3 and 4). No further neurological deficit was found.

FIGS 3 and 4 Considerable reduction in visual fields in both eyes (September, 1967)

Plain skull radiographs showed asymmetry of the pituitary fossa with erosion of the posterior clinoid processes. Bilateral carotid angiography (Dr. G. F. Swann) showed that the aneurysm had considerably enlarged (6 cm. in transverse diameter compared with 4 cm. in 1965) (Fig. 5).

FIG. 5 Bilateral carotid angiography, showing enlargement of aneurysm (September, 1967)

Occlusion of the right common carotid artery for 15 min. produced no clinical change (after Matas, 1909).

Operation (October, 1967) The artery was doubly ligated on October 24, 1967 (Mr. I. R. McCaul).

After operation the vision continued to deteriorate, and 3 weeks post-operatively there was no perception of light in the left eye. The right eye showed a worsening field defect (Fig. 6). The patient's name was placed on the Register of Blind Persons.

FIG. 6 Deterioration in visual field in right eye (November, 1967)

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<i>Control</i>	<i>Insulin</i>					
	+15 min.	+30	+45	+60	+75	+90
63	60	16	22	41	47	69

<i>Control</i>	<i>Insulin</i>		
	+ 30 min.	+ 60	+ 90
18.5	19.0	48.5	48.5

<i>Control</i>	<i>Insulin</i>			
	+30 min.	+60	+90	+120
5	7	35	12	6

January, 1968 i.e. 3 months after the operation, the patient's vision and headache suddenly began to improve; about the same time, the diabetes insipidus remitted and has not recurred. No further mental change has been noted.

October, 1968 Visual improvement has continued and in October, 1968, the acuity was 6/5 (corrected) in the left eye and fingers held at 12 in. could be counted with the right eye. The visual field of the left eye had become full and that of the right eye had considerably increased, although there was still a nasal hemianopia (Fig. 7). The patient's name was removed from the Register of Blind Persons. The atrophic appearance of the right optic disc was unchanged and the right pupillary reactions remained sluggish.

FIG. 7 Improvement in visual fields was noted in October, 1968, but there was still nasal hemianopia in the right eye

Discussion

Norlén and Barnum (1953) have estimated that there is a 1 per cent. incidence of intracranial aneurysm in the general population. Of these, 20 to 25 per cent. affect the anterior communicating artery. Intracranial aneurysms frequently present with headache, subarachnoid haemorrhage, or epilepsy. They may cause sudden or gradual symptoms by compressing the brain stem, cerebral hemispheres, or cranial nerves. Aneurysms of the anterior cerebral or anterior communicating arteries may also cause mental, olfactory, sphincteric, hypothalamic, or endocrine disturbances, although these are unusual (Jefferson, 1937; Meadows, 1951). Symptoms due to pressure by an anterior communicating aneurysm on the optic nerves or chiasm are also uncommon. Jefferson (1937) reviewed 66 cases of amblyopia and field defect caused by intracranial aneurysms at various sites and described twelve of his own. Six of his cases showed optic nerve or chiasmal compression although the anterior communicating artery was not the site of the lesion. Each presented with a history similar to that described above, *viz.* headache, acute diminution in visual acuity, and field defects. Similar cases were described by Dailey, Holloway, Murto, and Schlezinger (1964) and Cullen, Haining, and Crombie (1966).

It seems clear that direct compression of the optic nerve by the aneurysm is not the sole cause of the loss of vision. Distortion of the optic nerve at the foramen plays an important

part as does interference with the blood supply of the proximal optic nerve or the chiasma. The latter point was brought out by Richardson and Rose (1965), who described a patient who died from pituitary apoplexy. Symmetrical small haemorrhages were found in the optic nerve at the point where they were crossed by the anterior communicating artery.

The diagnosis is usually confirmed, but not invariably if clotting has occurred, by bilateral carotid arteriography. This procedure may not only confirm the size, shape, and position of the lesion, or show up some other unsuspected vascular anomaly, but it is also of importance, when planning surgery, in determining to what extent each carotid system supplies the aneurysm (Norlén and Barnum, 1953). This is especially important when carotid ligation is contemplated. The Matas test is also of value in this connexion. Plain skull radiography may show erosion of the dorsum sellae or calcification in the wall of the aneurysms. The testing of pituitary function may reveal an unsuspected latent hypopituitarism necessitating corticosteroid cover for operation. In the patient described, symptoms of diabetes insipidus developed after surgery. Anterior lobe function was found to be normal but ideally this should have been assessed preoperatively (Nieman, Landon, and Wynn, 1967).

Recently reported series deal primarily with surgery after subarachnoid haemorrhage (Robertson, 1949; Rogers, 1949; Elvidge and Feindel, 1950; Falconer, 1951; Poppen, 1951; Wechsler, Gross, and Cohen, 1951; Bassett, List, and Lemmen, 1952; Black and German, 1953; Brackett, 1953; Logue, 1956). Little or no mention is made of the treatment of visual disturbances *per se* but no visual deterioration was reported after carotid ligation. Most neurosurgeons seem to favour the direct approach for anterior communicating aneurysm with a view to clipping the neck of the lesion. There is a risk of venous thrombosis but frontal lobe infarction is more likely after carotid ligation, except when the aneurysm fills equally well from both sides (Poppen, 1951).

There remains a small group of cases such as that described, with a supraclinoid aneurysm adjudged too large for direct surgery. When such lesions are causing severe pain or impairment of vision, carotid ligation would seem to be indicated (Meadows, 1951; Norlén and Olivecrona, 1953). There are a few reports of patients who, as in the case described, showed considerable improvement after such treatment (Birley and Trotter, 1928; Jefferson, 1937; Krayenbühl, 1946; Höök and Norlén, 1964; Shealy and Kaufman, 1965). Ligation of the common, rather than of the internal carotid, artery seems less likely to produce a hemiparesis. Occasionally spontaneous improvement may occur, presumably by clot formation (Conway, 1926). It is well recognized that symptoms due to chiasmal compression by pituitary tumours may spontaneously improve, probably after infarction.

The mechanism of improvement after carotid ligation is obscure. Reduction of the pressure in the aneurysm, with or without subsequent thrombosis, presumably allows partial recovery of the compressed structures. Oedema is possibly lessened and arterial or venous channels may re-open, especially if surgery is performed sufficiently early after an exacerbation in symptomatology. A reduction in pressure may also possibly cause a slight shift in position of the lesion. The earlier the pressure is reduced the more neurological recovery may be expected.

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