BASILAR EMBOLISM*

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FOCAL neurological defects due to stenosis or occlusion within the main arterial trunks feeding the brain have become recognized with increasing frequency in recent years. However, until recently, knowledge concerning the effects of occlusion of the internal carotid artery has outstripped that of occlusion within the vertebral-basilar artery system. Both commonly produce ophthalmological features.

It is known that the internal carotid artery may be ligated in young adults with impunity. This depends upon healthy anastomotic connexions around the circle of Willis, particularly *via* the anterior and posterior communicating arteries. In cases in which hemiplegia results from ligation, there is probably an absence of the normal circle of Willis, a situation which Dandy (1944) has shown to be not uncommon.

In the elderly, atheroma and other sclerotic diseases impede the flow both in the main and subsidiary trunks and in the collateral vessels, and in addition predispose to thrombosis. The clinical picture produced depends upon the site and extent of the occluding thrombus and the potential of the collateral circulation.

Occlusion of the internal carotid artery in the neck in such circumstances may lead to focal ischaemic damage at certain sites only in the periphery of its ramifications. Ophthalmological features produced include arcuate field defects from branch retinal artery ischaemia, uniocular blindness from ischaemia of the central retinal artery, and contralateral homonymous hemianopia from ischaemia of the optic tract or of the temporal loop of the optic radiation in the field of the middle cerebral artery (Hollenhorst, 1959; Gordon, 1959).

Moreover, in stenosis or incomplete occlusions, these features may be transient, with complete recovery, to be followed by recurrent episodes, between which the patient is normal. This clinical pattern has become known as the syndrome of internal carotid insufficiency.

On the other hand, complete basilar artery occlusion from thrombosis generally leads to coma, quadriplegia, cerebellar and brain-stem signs, and death.

However, it has come to be recognized that basilar stenosis and incomplete occlusion may result in certain clinical patterns which are analogous to the internal carotid insufficiency syndrome.

In view of the wide distribution of the branches from the basilar artery to the brain-stem, cerebellar, and posterior cerebral fields, the syndrome of

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* Received for publication December 5, 1959.

basilar insufficiency may present different patterns, depending upon which branch suffers the most from ischaemia in the periphery of its ramifications, and which also derives the least reinforcement from the collateral supply of the posterior communicating and middle cerebral arteries.

Common manifestations are homonymous hemianopia, often bilateral, due to occipital ischaemia, and confusion, vertigo, nystagmus, diplopia, dysarthria, and dysphagia due to brain-stem and cerebellar ischaemia (Minor, Kearns, Millikan, Siekert, and Sayre, 1959). These transient recurrent signs indicate basilar stenosis, incomplete occlusion from thrombosis, or complete occlusion with adequate collateral circulation; persistence or progression of signs indicates frank infarction.

Case Report

A man aged 62 years came to the hospital in the early hours of the morning because of the sudden onset of complete blindness about 20 minutes before. He was too distressed and confused to give a coherent story, but the following facts were reported by his son.

History.—From early that evening he had suffered from a severe frontal headache and had vomited several times. This led to a bout of vomiting, after which the patient looked up and asked why the lights had been switched off, stating he could not see light in either eye.

For the previous 6 weeks, he had had almost continual frontal headaches which were severe at night and eased off during the day; there was no relief from ordinary analgesics. He had not suffered from headaches before this. For the last week he had vomited intermittently when the headache was severe.

He was said to suffer from heart trouble and had been under treatment for this for 12 months. From the cardiologist who had been attending him, it was discovered that he had had angina pectoris for 2½ years; and that congestive heart failure had supervened 12 months previously, associated with auricular fibrillation. This had been controlled by digitalis, chlorothiazide, trinitrin, and quinidine. Intermittent quinidine therapy had been necessary to control a variety of cardiac arrhythmias, particularly in the preceding few months. However, quinidine was stopped 2 days before the onset of blindness, as it was thought that it might be causing the headaches and vomiting. There was no history of rheumatic fever.

Examination.—He was a short thickset man in great distress, complaining of loss of sight and severe headache. There was definite confusion in addition to his natural distress.

There was no perception of light in either eye; the pupils were equal, 2 mm. diameter, and reacted briskly to light. The fundi showed normal discs and maculae, and grade I arteriosclerotic vascular changes. There was no reflex blinking to menace. Ocular movements were full, showing continuous conjugate movement of a searching type in all directions. There was no nystagmus.

General Examination.—The blood pressure was 270 mm. Hg systolic, 90 mm. Hg diastolic; pulse 72 per min., often regular, but with episodes of multiple extra-systoles. The heart was clinically a little enlarged and the bruits of mitral and aortic incompetence were present. There were crepitations at both lung bases and slight ankle and sacral oedema. Carotid and peripheral pulses were present and equal on the two sides.

No other definite cranial nerve lesion was found. Both plantar responses were normal, but the left leg showed diminished tendon reflexes and felt flaccid in tone compared with the right leg. There was no neck stiffness and Kernig's test was negative. There was no evidence of cerebellar disturbance.

Laboratory Investigations.—Lumbar puncture showed crystal clear fluid containing 3 lymphocytes per cu. mm., 100 red cells per cu. mm., 20 mg. per cent. protein, and 65 mg. per cent. sugar.

Full blood examination showed packed cell volume of 41 per cent. with 13.3 g. per cent. haemoglobin; the film showed 14,400 white cells with a normal differential count. The erythrocyte sedimentation rate (Westergren) was 52 mm. in 1 hour. The coagulation and prothrombin times were normal.

Radiography of the chest showed the transverse cardiac diameter at the upper limit of normal. The upper mediastinum was widened by the unfolded aortic arch. The lungs appeared moderately congested.

Both the Kahn test and Wassermann reaction were negative.

Electrocardiography showed sinus rhythm with ischaemic changes.

Diagnosis.—Arteriosclerotic heart disease with basilar artery embolism.

Therapy.—Anticoagulant therapy was instituted in hospital, by means of heparin 12,500 units intravenously every 12 hours for 48 hours, together with Dindevan (phenindione) by mouth, the latter to reduce the prothrombin time index to below 20 per cent. Digitalis and chlorothiazide were continued, supplemented by a mercurial diuretic on one occasion. Anticoagulant therapy was discontinued after 7 days.

Course.—The patient was initially very disturbed in hospital, sitting bolt upright in bed and holding his head in distress. Sedation and analgesia enabled him to rest quietly for the day. He remained afebrile.

On the second morning, about 30 hours after the first onset of blindness, he stated that he could see again: testing showed accurate projection of light in each eye and acuity of "counting fingers"; no further vision tests were done at this time. On the following morning the uncorrected vision was 6/12 in the right eye, and 6/9 in the left. The peripheral fields were full on the perimeter (3/330 mm. white).

The patient said he felt well except for some frontal headache. No weakness of the limbs was elicited and his sensation was normal. He remembered the bout of vomiting preceding the blindness, but remembered no more until he found he could see again, some 30 hours later. Whilst in hospital his blood pressure fell to 170/60 mm. Hg.

Discharge.—He was discharged on the ninth day, when the visual acuity was 6/5 in the right eye with +0.5 D sph., and 6/5 in the left eye with +0.25 D sph. He could read N5 with additional +3 D sph. in each eye. The visual fields were full. He has remained without neurological troubles since.

Comment

The differential diagnosis in cases of suspected cortical blindness is of some importance. Bilateral blindness, with normal pupillary light responses and normal fundi, is consistent with either bilateral cortical lesions or hysteria. Symonds and Mackenzie (1957) have emphasized the value of the presence of reflex blinking to menace in hysterical blindness. The possibility of toxic amblyopia had to be considered in this case, as the patient had been treated with digitalis and quinidine. However, no fundal changes suggestive of retinal ischaemia or of retrobulbar neuritis were seen, and the quinidine therapy had ceased 2 days before the onset of blindness; moreover, this patient had been receiving quinidine for some time—whereas toxic amblyopia from quinidine usually occurs soon after starting treatment with the drug.

That the site of the vascular obstruction in this case was not purely peripheral in the calcarine arteries, is shown by the definite confusional state and

slight paresis of the left leg present at the onset, indicating ischaemia over a wider field.

Evidence supporting an embolic aetiology rests upon two features: first, a satisfactory origin for the embolus, and secondly, the sudden onset, short duration, and sudden recovery from the disability. In the first regard, the patient suffered from ischaemic and valvular heart disease, and over the previous months had had a variety of cardiac arrhythmias, including auricular fibrillation. It seems likely that emboli were cast off from the heart at times of change of rhythm, and that minor embolic episodes in the previous weeks explained the headaches and vomiting leading to the ictus itself.

Symonds and Mackenzie (1957) have explained the mechanism of the involvement of both calcarine arteries simultaneously. An embolus or thrombus lodges at the bifurcation of the basilar artery, thereby producing ischaemia throughout the basilar field. To a variable degree this is compensated for by the posterior communicating arteries connected to the posterior cerebral arteries peripheral to the obstruction. Then the embolus fragments, feeding finer emboli into both calcarine branches sufficient to cause ischaemia in the occipital cortex. In favourable cases the whole embolus and its fragments break up, so freeing the circulation, or else collaterals from the middle cerebral artery subserve the affected cortical area. Thus, frank infarction may be avoided.

It is difficult to assess the efficacy of anti-coagulant therapy in this case. It would seem likely that the recovery followed naturally. However, where embolism has occurred, the risk of secondary thrombosis arises, providing justification for the use of anticoagulant therapy to avoid such complication. In the circumstance where recovery from embolism follows rapidly, there is no indication for the continuation of anticoagulant therapy.

Summary

The syndromes of internal carotid and basilar artery insufficiency are briefly described. A case is presented of bilateral cortical blindness due to basilar embolism, which recovered completely. The differential diagnosis of cortical blindness is briefly discussed. The mechanism of production of bilateral cortical blindness is commented upon.

I wish to express my thanks to Dr. Kelvin Lidgett, Honorary Ophthalmic Surgeon to the Victorian Eye and Ear Hospital, Melbourne, for his permission to use the clinical records of this patient, and for his encouragement in the presentation of this report.

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