SURGICAL RECONSTRUCTION OF OCCLUDED CERVICAL CAROTID ARTERY

REPORT OF A SUCCESSFUL CASE WITH 4-YEAR FOLLOW UP AND THREE EXAMPLES WITHOUT SUCH TREATMENT*

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The clinical syndrome of occlusive disease of the carotid arterial system has been well described. Thrombotic disease of the carotid tree cephalad from the aortic arch, including temporal and ophthalmic branches, has also been attributed in rare instances to infection or trauma. Narrowing of the lumen can advance so insidiously that recurrent and progressive neurologic deficit may be overlooked or not ascribed to decrements of carotid-cerebral blood flow. Successful direct treatment of the abnormality depends on its prompt recognition and evaluation to ascertain the possible value of surgical reconstruction of the affected artery.

CASE REPORTS

Case 1. A 64-year-old man (right-handed) was admitted to hospital on Feb. 21, 1950, having experienced drooping of the right side of the mouth and weakness of both legs 8 years previously, lasting 8–9 months. He had had a fainting spell 5 years back with loss of consciousness. Three weeks before hospitalization he became drowsy and his tongue and mouth pulled to the left, clearing rapidly. One week before admission the left arm became weak and vision began to fail, beginning with diplopia and declining to perception of light and gross movements of the hand within 3–4 days. (The sketchy history probably omitted other interval episodes of neurological difficulty.)

Examination. There was poor comprehension because of difficulty in language. The patient was mentally dull but oriented and able to perform simple commands. He could not read letters 4 inches high but named a few objects adequately visualized. There were nystagmoid movements of the eyes on lateral gaze; pupils and fundi were normal; fields of vision revealed large central scotomata surrounding points of fixation and extending beyond blind spots. There was weakness of the left lower face and left arm, with slight increase in deep reflexes generally; plantar reflexes were equivocal; sensation was grossly intact. Blood pressure was 220/100.

Laboratory Studies. Hemoglobin was 16 gm. per cent, hematocrit 50 per cent, and count of white blood cells 8,800/c.mm. Cerebrospinal fluid was slightly xantho-
chronic; protein was 60–80 mg. per cent. Hinton was negative, blood urea nitrogen 16 mg. per cent, and alkaline phosphatase 3.5 K.A. units (normal). Electrocardiogram showed frequent premature beats and indication of hypertension. Roentgenograms of skull and chest were unremarkable.

Course. Bilateral percutaneous carotid arteriography was carried out at one stage using 35 per cent Diodrast. Upon injection of the left carotid the anteroposterior view showed the internal carotid dividing into its anterior and middle branches as well as the contralateral anterior and middle branches filling via the anterior communicating artery, all vessels being in normal relationship. Upon injection of the right side there was no filling of the internal carotid in both anteroposterior and lateral projections although the needle was shown well placed in the common trunk with filling of the external carotid system. Following arteriography pupillary inequality developed and the patient was responsive only to painful stimuli. Subsequent spinal puncture revealed a pressure of 140 mm. and slightly yellow cerebrospinal fluid. He remained comatose and expired 3 days later.

Autopsy disclosed advanced atherosclerosis involving all major cerebral arteries. There was extensive and complete obstruction of the right internal carotid but the anterior and middle cerebral arteries on both sides were open; both posterior communicating arteries were extremely narrowed and questionably patent. The brain showed fresh thrombotic occlusion obliterating the full length of a tortuous basilar artery, superimposed on arteriosclerotic plaques with patchy chronic ischemic infarction and acute softening in both occipital and both temporal lobes, thalami, pons and cerebellum. Multiple old infarcts with cystic degeneration were noted in the internal capsule, putamen, and globus pallidus on each side. Generalized arteriosclerosis was widespread in major arteries elsewhere.

Comment. The brain of this man had achieved remarkable toleration to progressive occlusion of the right carotid artery in the face of extensive cerebral arteriosclerosis by the development of compensatory supply through the contralateral circle of Willis and probably meningeal anastomoses and the homolateral ophthalmic artery as well. Cerebrovascular survival during the last 3 days of life apparently was maintained almost entirely by the left carotid artery. The extensive old capsular and lenticulai lesions bilaterally came as a surprise in view of the relatively minor degree of neurologic loss recorded during his hospitalization. Basilar narrowing was undoubtedly in progress for several years before it was rendered complete by ill-conceived carotid arteriography. Competent observers, including several neurologists and neurosurgeons and two ophthalmologists, suspected a prechiasmal or retrobulbar lesion, perchance a neoplasm of the middle fossa. The possibility of occlusive carotid or cerebrovascular disease was not mentioned by any one of them; apparently no one chose to palpate the carotids in the neck. In retrospect, absent pulsation might have been noted on the right side. Two-stage angiography should then have been performed at 24-hour interval using the less irritating Thorotrast; or surgical exploration of the right cervical carotid system might have disclosed the lesion and sufficient healthy subcranial segment of internal carotid artery available above the site of thrombosis to permit reconstruction. We suggest that such an at-