SURGICAL TREATMENT OF ARTERIOSCLEROTIC OCCLUSION OF COMMON CAROTID ARTERY

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In 1905 Chiari\(^2\) first described a clinical syndrome characterized by temporary hemiparesis, aphasia, or transient loss of consciousness caused by spontaneous occlusion of the internal carotid arteries. Ten years later Hunt\(^7\) again called attention to the same disease and symptom complex and emphasized the importance of careful examination of the cervical carotid vessels in all patients presenting symptoms of possible vascular origin. Not until 1937, however, when Moniz\(^15\) introduced an efficient method of carotid arteriography was accurate diagnosis of the condition made possible, and since that time the disease is being recognized with increasing frequency. The condition is not rare, as evidenced by Hultquist’s\(^8\) study of 3,500 autopsies, in which he examined the carotid vessels and evidence of occlusion was found in 91 instances. The majority occurred in the sixth and seventh decades and the condition was about twice as common in men as in women.\(^10\) Recently, Johnson and Walker\(^8\) were able to assemble 107 reported clinical cases. In the great majority the occlusive process occurs in the region of the carotid sinus but in some cases occurs more distally, even as high as the intracranial portion of the internal carotid artery. Interestingly enough, the lesion is 6.5 times more common in the left carotid than the right.\(^18\) In a few cases the lesion is embolic in origin, but the predominant cause is atheroma, which may develop prematurely. The carotid sinus thus appears to be similar to the aortic bifurcation and left coronary artery as a site of predilection for arteriosclerotic occlusion.

Symptomatology may vary widely in severity and mode of onset. Webster\(\textit{et al.}\)^{19} described four methods of clinical onset for the disorder: (1) explosive, simulating stroke; (2) slowly progressive, characterized by remissions and recurrences (which may suggest multiple sclerosis); (3) with visual symptoms including transient blindness; (4) without signs or symptoms. In addition to the neurologic manifestations already mentioned, Shapiro and Peyton\(^15\) described convulsions and head noises. The latter were described as a soft roaring noise, sensations of escaping steam in the ear, whistling sound or the sound of a watch ticking. Presumably the wide variation of incapacity produced by carotid artery occlusion is related to the acuteness of the occlusive process, presence of collateral vascular supply to the brain, and the completeness of the occlusion.

Several methods of surgical treatment have been advocated and tried
in a few cases, usually with unsatisfactory results. Arterectomy has been performed in the hope that removal of the involved segment might reduce reflex spasm in the cerebral vessels,¹ and cervical sympathectomy⁸,¹⁸ or denervation of the carotid sinus was advocated for a similar purpose, but in both instances results were disappointing. Definitive treatment by restoration of carotid arterial continuity has been attempted in relatively few cases. Strully¹⁷ attempted thromboendarterectomy for thrombosis of the internal carotid artery in the neck, but was unable to establish a retrograde flow from cerebral vessels and finally ligated the carotid. Although excision of the thrombosed segment with insertion of an arterial homograft would obviously be an effective method, interestingly enough, successful results have not been reported.⁴ Indeed, only one satisfactory result from restoration of arterial continuity has been recorded by Eastcott et al., who used direct anastomosis of the vessel to the common carotid artery after excision of the partially occluded carotid bifurcation and ligation of the external carotid. After operation the patient was neurologically normal and relieved of intermittent attacks of hemiplegia.

This report is concerned with a case of incomplete occlusion of the common carotid artery at the bifurcation producing annoying head noise and with the method of surgical treatment by means of endarterectomy.

CASE REPORT

D.W., a 71-year-old white man, was admitted to Methodist Hospital on March 4, 1956, complaining of a swishing noise in his left ear. The noise first appeared 4 months previously and had steadily increased in intensity. It was synchronous with heart beat and was most annoying during periods of accelerated heart rate. The noise was accentuated by slight flexion of the head on the neck, particularly if his head were on a pillow, thus interfering with sleep. He denied episodes of numbness, tingling, or unilateral loss of vision, but described dizziness on sitting or standing up. The dizziness was transient, usually lasting less than 1 minute.

Examination. Blood pressure was 150/80 mm. Hg. Normal pulsations were palpable in the common carotid arteries, and a soft thrill was present over the left carotid bifurcation. The left superficial temporal pulse was slightly weaker than the right. On auscultation a loud bruit was audible under the angle of the mandible and was transmitted upward over the superficial temporal artery. Compression of the carotid obliterated the thrill and bruit. Temporary occlusion of the left common carotid artery for 30 seconds (Matas test) produced dizziness, weakness, numbness and tingling sensations in the entire right half of the body, and partial aphasia. All peripheral arterial pulsations were normal.

Laboratory studies revealed a normal hemogram and urinalysis. Electroencephalograms were within the range of normal, and the pattern showed no appreciable change during temporary left carotid occlusion.

Cerebral arteriography was done using Thorotrast solution and showed a partial occlusion of the left common carotid artery beginning just proximal to the bifurcation, with satisfactory filling of the internal and external divisions (Fig. 1). Localized aneurysmal dilatation of the internal carotid artery was demonstrated distal to the stenosis.