SURGICAL TREATMENT OF ARTERIOSCLEROTIC OCCLUSION OF COMMON CAROTID ARTERY

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(Received for publication April 24, 1956)

In 1905 Chiari 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 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 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 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. Recently, Johnson and Walker 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. 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 et al. 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 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, and cervical sympathectomy 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. Strully 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. 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 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 Thorotrust 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.
Operation. On March 8, 1956, under intravenous Pentothal and endotracheal inhalation anesthesia, cooling of the cranium and brain was attempted by immersing the head in crushed ice. After about 30 minutes this was discontinued, primarily because there was no available method of estimating the degree of local tissue cooling. An oblique incision was made in the neck at the level of the arterial obstruction and the carotid bifurcation was exposed. The occlusive process in the common carotid was located and found to be calcified and fixed, involving only the terminal 2 cm. of the common carotid. A strong systolic thrill was palpated in the external and internal carotids, where the arterial tension was palpably lower than in the common carotid. The aneurysmal dilatation of the internal carotid was demonstrated.

A polyvinyl shunt, with needle points at both ends, was used to by-pass the carotid circulation during the period of occlusion (Fig. 2). With the external carotid temporarily occluded, internal carotid flow was maintained by means of the shunt while the atheromatous plaque was removed from the vessel (Fig. 3). The intervening segment of artery was occluded by arterial clamps and a transverse incision was made in the carotid bulb. The occluding calcified arteriosclerotic plaque was peeled out of the lumen by thromboendarterectomy and the arteriotomy was sutured transversely (Fig. 4). Arterial flow was restored after 9 minutes of carotid clamping during which period the shunt continued to function satisfactorily. After arterial repair was complete, strong pulses without a thrill were palpable in the external and internal carotids. The incision was closed without drainage.

Postoperative Course. Soon after awakening from anesthesia the patient announced that the head noises were gone. During the first 12 hours after operation, mild weakness of the right side with thickened speech was present, but these symptoms rapidly resolved, and his recovery was uneventful. He was discharged 6 days
later with slight impairment of motion and sensation in the right hand. Four weeks later examination revealed normal pulsation in the left common and external carotid arteries without evidence of a thrill or bruit. The slight residual weakness in the right hand was improving and function was almost normal.

**DISCUSSION**

Although our patient demonstrated no neurologic deficit prior to operation, the results of the carotid occlusion test indicated that cerebral circulation was precariously reduced. Under such circumstances even slight reduc-
tion in cardiac output or periods of hypotension from any cause may produce temporary hemiplegia from ischemia even in the absence of complete carotid occlusion. Furthermore, the narrow lumen of the carotid artery could easily have been occluded acutely by development of a very small thrombus. If operation had been postponed until permanent damage to cerebral tissue had occurred, little could have been expected from restoration of normal carotid circulation. Thus, operation was undertaken in a stage of the disease when carotid occlusion was incomplete. In cases in which symptoms remain transient even though carotid occlusion is complete, operation may also be successful.

Operations upon the carotid artery require temporary occlusion of the vessel during the vascular reconstruction and certain technical measures to protect the brain during the period of temporary ischemia must be employed. The Matas carotid occlusion test provides good indication of the tolerance to temporary ischemia and should always be done before operation in evaluation of such patients. In those patients with total thrombosis of the carotid artery in whom symptoms are transient, the test should be negative, and if operation is done, temporary clamping of the carotid produces no additional cerebral ischemia. On the other hand, when the occlusive process is incomplete, as was the situation in our patient, measures to protect the brain from ischemic damage may be vital to success of the operation. The solution of this technical problem may be achieved either by use of temporary shunts.
or by hypothermia. The external shunt used in our patient probably permitted a successful result after 9 minutes of carotid occlusion since the Matas test had demonstrated that neurologic changes on an ischemic basis otherwise appeared after 30 seconds of carotid compression. Arterial flow through one normal carotid artery under basal conditions is estimated to be approximately 250 cc. per minute, but in our patient this rate was probably much less. Measurements on the maximum rate of flow of saline through the shunt at 80 mm. Hg was determined to be 125 cc. per minute. Although this rate of flow is only 50 per cent of estimated basal flow through one carotid artery, reduced flow undoubtedly can protect the brain from serious damage under these conditions. The brief occurrence of right hemiplegia during the first few hours after operation may have been caused by mild ischemic damage, and indicates that the margin of safety with this method may not be sufficient. Perhaps enlarging the lumen of the shunt would have been desirable in this case. The focal residual neurologic change in the right hand also may have been caused by a small cerebral embolus produced in the shunt, although the shunt had been soaked in heparin solution and revealed no evidence of thrombosis. Nevertheless, this complication may indicate the need for continued systemic heparinization after such operations.

Hypothermia may provide another means of solving the problem of cerebral ischemia during temporary carotid occlusion. Hypothermia reduces the metabolic rate of the cerebral tissue and thus reduces the requirements for oxygen and arterial circulation. On the basis of experimental observations hypothermia appears to exert a definite protective influence against ischemic damage.9,10,12,13 Similar protective influence has been demonstrated in use of hypothermia during temporary occlusion of vascular supply to the spinal cord.5,14 It is further significant that in the only previously reported successful case of restoration of carotid circulation the procedure was done under hypothermia without any neurologic sequelae. Unfortunately, Eastcott et al.5 in reporting that case made no mention of a Matas test before operation and it is possible that the patient would have recovered even without hypothermia. Whether local hypothermia of the brain by external cooling is feasible or not is questionable, but it is doubtful that the local cooling was effective in our patient. Local hypothermia of the brain by cooling of the carotid blood has been used clinically and provides a method of producing selective hypothermia to the brain without cooling the entire patient.9 General body hypothermia was not used in our patient but is undoubtedly a very useful adjunct, particularly in younger patients when the general physical condition permits. The combination of external shunts plus hypothermia offers the maximum protection of the brain during operation on the carotid artery and should be considered the method of choice.

SUMMARY

Arteriosclerotic occlusion of the common or internal carotid arteries is not uncommon and may produce neurologic symptoms of varying types and severity. Onset may be explosive in character, producing hemiplegia, or may
be insidious, producing slowly progressive or transient symptoms. Prompt recognition of the disease and localization of the occlusive lesion permits early treatment and may prevent permanent neurologic symptoms. Surgical treatment with restoration of effective carotid circulation should be employed in all patients if irreversible cerebral damage has not already resulted.

A case is reported of incomplete left common carotid artery occlusion in a 71-year-old man whose only symptoms were annoying swishing sounds in the head. At operation thromboendarterectomy was performed, reconstructing the artery while a temporary external shunt was used to provide cerebral circulation.

REFERENCES


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