Carotid Kinking as a Cause of Cerebral Insufficiency

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Transverse enlargement of vessels is well recognized and is known to result in aneurysmal formation. Most often this condition coexists with arteriosclerotic degeneration of the aorta or the major arteries. Of equal importance and more frequently recognized is the clinical entity of elongation and tortuosity of the aorta and the major arteries, secondary to degenerative changes of the blood vessels. In patients who have conspicuous elongation of the aorta or arteries, tortuosity is often demonstrable.

In the carotid artery, elongation and tortuosity are fairly common. Actually, for some time, the frequency of such findings prevented their recognition as significant factors in cerebral insufficiency. In 1925, Kelly postulated that increased tortuosity of the carotid artery was a result either of embryologic error or of arteriosclerotic change. With the increasing use of arteriography, many patients have been found to have twisted or kinked carotid arteries, and the relation of this finding to cerebral insufficiency has become apparent; however, attempts at surgical correction were for so long unsuccessful that lesions of this type came to be considered untreatable by surgical means.

Carotid insufficiency because of tortuosity of the artery has recently been diagnosed at John Sealy Hospital in several acutely ill patients with strokes. In most cases, surgical repositioning of the artery brought about a favorable outcome. Case reports of two of these patients are cited. The purpose of this paper is to describe the clinical course in the two patients with arterial insufficiency secondary to kinking of the arteries to the brain and to discuss the methods used in diagnosis and treatment.

Case Histories

Case 1

A 66-year-old white woman, was admitted to the hospital on October 31, 1960. Ten days before admission she began to hear a roaring noise in the left ear, described as being comparable to that of a train. She had been quietly watching a television program when the noise began. The patient was unable to hear other sounds because of it. Three or four years earlier, the patient had had transient episodes of similar sensations, but they lasted, at most, for only a few hours and would disappear spontaneously. On this occasion, she also had dizziness and loss of vision in the left eye.

She had also previously noted a recurrent painless pulsating mass on the left side of the neck. During the episode in October the mass was evident. It protruded almost an inch but it was not sore and she had no disturbances of sensation. Earlier, when the mass had appeared, she had consulted a physician, but always, by the time she reached him, the lesion would no longer be visible. Physicians not unnaturally assumed that the patient was imagining the protrusion, although her husband verified the presence of the mass on each occasion.

The patient had had recent experience of memory loss and of spells of dizziness but recalled no trauma to the head or neck, ear infections, headaches, or visual, sensory, or motor disturbances. For the preceding 5 years she had been receiving treatment for hypertension.

On physical examination she was found to have a blood pressure of 185/110 mm. Hg in both upper extremities, respirations 24 per minute, and pulse 64 per minute. No ocic abnormalities were noted, and, although she had grade-II hypertensive retinopathy, there were no appreciable differences in the fundi. The roaring that she heard was continuous but it did not correspond to the pulse. Carotid pulsations were full and equal, bilaterally. No masses in the neck were palpable.

An extremely loud thrill was audible over the angle of the mandible on the left side, and there was a faint murmur or thrill on the right. Pressure for a period of 7 seconds over the left common carotid artery resulted in sensations of numbness in the right arm and leg. Except for slight cardiac enlargement, the findings on physical examination were essentially negative.

All findings were within normal limits on the
laboratory studies, which included lumbar puncture. Chest and skull films with Stenver's views were normal; visual fields were normal; and ophthalmodynamometric examination suggested no significant difference in the ophthalmic arterial pressures. Perception deafness in the left ear and fairly good hearing in the right were discerned on audiographic study.

Bilateral percutaneous carotid arteriograms were then made under general anesthesia. A complete loop of the left (figs. 1 and 2) and a sharp kink in the right internal carotid artery were demonstrable (a 360-degree turn in the left and two 90-degree turns on the right). After the anesthesia, the patient had flaccid paralysis of the right forearm and hand, with only limited use of the muscles at the elbow.

After thorough evaluation it was decided to explore the left carotid artery. General anesthesia and external hypothermia were utilized for operation on the next day. The artery was found to be acutely kinked, approximately 3 to 4 cm. distal to the bifurcation of the left common carotid artery. Simultaneous pressure determinations proximal and distal to the area of kinking demonstrated a gradient of 30 mm. Hg pressure, after the sheath had been opened and the arteries were freed. By gentle unkinking of the artery at the time the determinations were being made, the pressure gradient could be made to disappear completely. When the patient's head was placed differently, the loop would protrude laterally, anterior to the sternocleidomastoid muscle, which probably explained the mass described by the patient. The artery was pulled downward and a portion of the sternocleidomastoid muscle was freed by dissection and placed under the artery to prevent kinking, without disruption of the continuity of the vessel. The head was placed in various positions, each for several minutes at a time, and the pupils were observed for changes in size. No gradient was observed on pressure determinations.

In the period immediately after operation, the patient was free from the roaring sensation, and, with physiotherapy, she regained gradually some function of the right hand. The patient was discharged on November 18 and has had no further episodes of dizziness. Her eyesight is improved and she states that her memory is much better.
Case 2

A 71-year-old Negro woman entered the hospital June 6, 1960, because of recurrent attacks of syncope that had occurred during a period of 4 years. She received treatment for hypertension but the therapy did not relieve the intermittent attacks. During the weeks before admission, the patient had observed that the episodes occurred at any time she turned her head sharply to the right. There was no history of motor or sensory loss and no confusion.

On physical examination the patient, who was taking antihypertensive agents, was found to be normotensive. Although obese, she was extremely active and alert. When, on instruction, she turned her head sharply to the right, she would become dizzy or would faint completely and fall. Pressure for 10 seconds over the right common carotid artery would result in the same sensation that was incurred by turning her head quickly to the right. Except for slight enlargement of the heart the findings on examination were otherwise uninformative.

Bilateral carotid arteriograms demonstrated tortuosity of the right internal carotid artery. A pulsatile mass, 3 by 3 cm. in diameter, was discernible in the right supraclavicular fossa. The possibility of a carotid aneurysm was considered. The remainder of the laboratory findings were essentially normal except for slight electrocardiographic evidence of left ventricular hypertrophy.

At exploration the right common carotid artery was found to be greatly elongated. When the head was rotated to the right side, the right common carotid artery would become acutely kinked, and the kink would be released when the head was placed in a neutral position (figs. 3 and 4). Simultaneous pressure determinations were made from the right common carotid and the right subclavian arteries. The patient was found to have a 20-mm. Hg pressure gradient when the artery was kinked, with disappearance of the gradient when the kink was released. The artery was too long to transplant superficially, so that a graft was inserted from the innominate artery to the distal part of the common carotid. Pulsation in the graft was strong with the head in all positions. The wound was closed loosely.

In the postoperative period the patient was able to turn her head sharply to the right or left without any dizziness or syncope. She has now been observed for 8 months and has experienced no further difficulty in this respect.

Discussion

Arterial insufficiency as a result of kinking of the carotid artery has not received much attention in the past, probably because tor-
tuosity is a common finding in individuals without other signs of cerebral insufficiency. Labeling of this entity as nonsurgical was probably a result of the poor outcome of early attempts to shorten the arteries. The authors believe, however, that surgical repositioning of twisted arteries can offer definite relief to some patients with cerebral arterial insufficiency. Because arterial kinking can be demonstrated easily and safely when arteriography is properly employed, this type of study can be recommended for use in all patients who show any indications that kinking might be the cause of stroke. Similarly, when arterial tortuosity is demonstrated, relocating the carotid artery outside the sheath is a simple procedure that can, if necessary, be done under local anesthesia.

**Diagnosis**

As in most diseases, the history is of prime importance. Typically, the patient complains of having had multiple transient strokes, possibly induced by changes in the position of the head, or extreme positions of the head. More recently patients have been observed whose symptoms seemed to be associated with the administration of antihypertensive agents.

Cerebral damage is evaluated at the physical examination. Palpation of the carotid artery, both externally and laterally in the pharynx, may sometimes but not always be informative. The vessels should be palpated with the patient’s head in all possible positions. There may be areas of tenderness near the carotid sheath. If one carotid artery is palpable, nothing but trouble can be gained by occluding the other; however, if both are palpable, occlusion of the artery on the side shown to be insufficent can be a helpful procedure if correctly performed. The patient should be seated, and looking into the examiner’s eyes. The subject is asked to report any unusual feeling, and, when he does so, the artery is immediately released. In any case, the artery should be compressed no longer than 10 seconds. This procedure is most helpful in ruling out other causes for cerebral insufficiency besides unilateral carotid occlusion, and often a patient will state that the exact symptoms are reproduced by this maneuver. The patient is then instructed to rotate the head to the extreme right and left, as well as to the extreme positions of flexion and extension. Patients are told to maintain the extreme position for a period of 15 to 20 seconds to determine if any of the previously experienced sensations will result. It is important that the examiners remain on each side, so that, if syncope occurs, it will not result in a fall. Instruction of the patient to return the head to a neutral position at the first evidence of dizziness or neurologic sensation is necessary, in some instances, to prevent a syncopal episode.

The preliminary study of all patients is predicated upon the fact that an acute episode of cerebral vascular insufficiency could occur during the procedure. For this reason, patients are checked regularly for blood pressure, state of consciousness, and neurologic deficit. A 2-hour check has been found to be extremely helpful, since two patients have had complete hemiparesis during hospitalization while they were being tested for cerebral vascular insufficiency. Circulation was re-established in both instances by emergency surgical procedures. If, however, acute insufficiency should last for longer than 6 to 8 hours, damage to the brain would often be irreparable, and reestablishment of cerebral flow would not then result in return of function.

Cardiovascular and cardiopulmonary evaluation is done routinely, in addition to electroencephalographic study. When the electroencephalogram is made, continuous tracings are obtained with the patient’s head in the extreme positions, but only if one of the physicians on the Cardiovascular Service who is totally familiar with the problem can accompany the patient. In three instances, electroencephalographic changes were observed when a patient with kinking of the internal arteries turned his head in the extreme position. Complete neurologic evaluation is made in each case, after which the neurologist decides whether carotid arteriographic study should be done.

Percutaneous carotid arteriography has proved to be most useful in diagnosis. This
type of study is done, with rare exceptions, under local anesthesia, so that any change in the patient’s neurologic status may be quickly evaluated. This examination can be both safe and informative if several precautions are observed: 1. Multiple punctures of the artery are to be condemned. 2. Repeated unsuccessful attempts to position the needle correctly may result in development of large hematomas, with some degree of external compression of the artery. 3. The smallest amount of dye that permits adequate study should be used. 4. The study should make possible a thorough evaluation of the extracranial and intracranial arteries. 5. Films should be made with the patient in flexion and extension to determine if there is any change in the position of the artery during this maneuver. Threading a polyethylene cannula through the needle is most helpful in prevention of damage during positioning. 6. After the study the patient should be closely observed for 24 hours in order that any neurologic deficit or change in state of consciousness may be discerned.

Correction

Under either local or general anesthesia, the entire carotid sheath is exposed. The artery should be palpated thoroughly, with the head in all positions, before the sheath is opened, since release of the binding fibers will often correct the occlusive link. Pressure determinations should be taken proximal and distal to the suspected occlusion whenever feasible. The sheath is then opened and the artery is freed upward. Usually the kink can be corrected by superficial transplantation of the artery, with a slip of sternocleidomastoid muscle inserted beneath to anchor the artery in the desired position. Repeated pressure determinations will indicate how much of the obstruction is alleviated. The patient’s head should then be moved into different positions to see if the kinking will recur. The carotid sheath is not closed, and the soft tissues are closed only very loosely in order to allow drainage. It is important to explore the entire carotid tree, as the kink may be located in any of a number of sites, as illustrated by case 2, in which the carotid was kinked at its origin.

Summary

With proper diagnosis based on history, physical findings, and arteriographic and pressure studies, it has become apparent that carotid artery kinking is a more common cause of strokes than was formerly believed. Because kinking is easily corrected, every patient with stroke deserves examination sufficient for determination of whether the carotid arteries are kinked. Two cases are cited in which multiple syncopal attacks were relieved when flow past a kink in the common carotid artery was reestablished.

Acknowledgment

The authors are indebted to Dr. Glenn Draeger for neurologic evaluation of all patients and for his advice and counsel regarding arteriography and surgery.

Addendum

Sixteen additional patients with insufficiency secondary to kinking have been seen since submitting this article.

References

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Circulation. 1962;25:849-853
doi: 10.1161/01.CIR.25.5.849

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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