

Stroke Associated with Elongation and Kinking of the Carotid Artery:

Long-Term Follow-Up

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CAROTID ELONGATION and tortuosity was recognized by otolaryngologists decades ago because of the hazards posed by the carotid artery bulging into the pharynx during the course of tonsillectomy.^{8,9,15,18,26,27} However, it remained for Riser *et al.*²³ in 1951 to recognize the association between carotid kinking and cerebrovascular insufficiency and to obtain relief of "crises of vertigo" in their patient using a piece of the sheath of the sternocleidomastoid muscle to fix the vessel and make it "describe a turn with a large radius." They postulated that changing head position might lead to ischemia. The first internal carotid resection was reported by Hsu and Kisten¹⁴ to have been done for fear the buckled segment would rupture. Unfortunately, thrombosis of the anastomotic suture line led to a fatal outcome. In December 1958, we discussed before this Association a paper by Bahnson *et al.*¹ We reported three cases of transient hemiparesis associated with elongation and kinking of the internal carotid artery treated by carotid resection which were subsequently published in detail.²² A fourth case of carotid tortuosity, which at that time was asymptomatic and under observation, was also presented. Since all four of these patients were followed until death, we felt it would be of interest to present their ultimate course with an analysis of the remainder of our series of 138 patients with carotid elongation and kinking treated surgically.

Case Reports

Case 1. A 69-year-old hypertensive woman noted numbness and weakness of the entire right side of the body on April 29,

1958. Carotid arteriogram (Fig. 1) showed overt kinking of the left internal carotid artery. This was confirmed at operation under general anesthesia on May 12, 1958 (Fig. 2) by which time neurologic changes had cleared. A 2 cm. segment of the left common carotid artery was resected which relieved the kinking (Fig. 3) as confirmed by a postoperative arteriogram (Fig. 4). Following recovery she remained in Savannah for several years and then moved to Nashville, Tennessee, where she continued to remain asymptomatic though hypertensive till 1968. She began to have "fainting spells" which were believed to be due to hardening of the arteries. On April 25, 1970, 2 months of frequent episodic dizziness and confusion culminated in the development of *left* hemiplegia. She recovered slightly in the hospital and was transferred 17 days later to an extended care facility where she died July 30, 1970 at the age of 83 years. Arteriograms were not done in the hospital and autopsy was not performed.

Case 2. On September 4, 1958, a 75-year-old normotensive man was admitted with a history of several years of intermittent dizzy spells. A transient ischemic episode 2 months previously had involved the right side and then complete right hemiparesis with aphasia had occurred that day which had begun to clear by the time of admission to the hospital some 4 hours later. Spinal tap revealed clear fluid. Carotid arteriograms showed corkscrew-type elongation of the right internal carotid just below the skull, while on the left there was elongation with a transverse band of radiolucency across the vessel about 2 cm. above the bifurcation (Fig. 5). This proved to be due to a kink in the artery (Fig. 6) and resection of the bifurcation, which contained a non-obstructing plaque, was carried out under local anesthesia. End-to-end anastomosis of the common to the internal carotid was made. Postoperatively the residual neurologic changes gradually cleared over a period of several weeks. He remained asymptomatic until January 28, 1963. On this date he was involved in an automobile accident in which he received a severe blow to the right side of his head. In the presence of unconsciousness and paralysis of the left side, bilateral carotid arteriograms showed no change of the left carotid circulation from that seen on the previous postopera-

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tive study. On the right there was found a large subdural hematoma which was evacuated surgically by Dr. Charles Usher, Jr. Initial response was good, but on February 21 it was necessary to plicate the inferior vena cava because of multiple pulmonary emboli. Thereafter the neurologic state gradually improved, but he died April 29, 1963 while still in the hospital of congestive heart failure secondary to infarction of the left ventricle and complicated by pneumonia in all lobes of both lungs. Autopsy also showed focal necroses in both frontal lobes of the brain with cerebral edema.

Case 3. A 68-year-old man was admitted October 11, 1958 with paralysis of the right upper extremity with associated aphasia which had occurred 4 days previously. Partial clearing had occurred. Spinal tap yielded clear fluid, and carotid arteriograms showed marked elongation of both internal carotid arteries without overt kinking (Fig. 7). The left carotid bifurcation, which contained a small non-obstructing plaque, was resected under local anesthesia. Anastomosis was performed from the common to the internal carotid artery. Over a period of several weeks there was complete resolution of the neurologic deficit. He remained well until 1960 when transurethral resection of the prostate was complicated by postoperative retinal hemorrhage in the right eye. Chronic atrial fibrillation then developed, but he remained active until December 19, 1963, when he developed hemiparesis involving the left side. No definitive evaluation was carried out, and he died January 1, 1964.

Case 4. The case of an asthmatic man, who was 67 years old

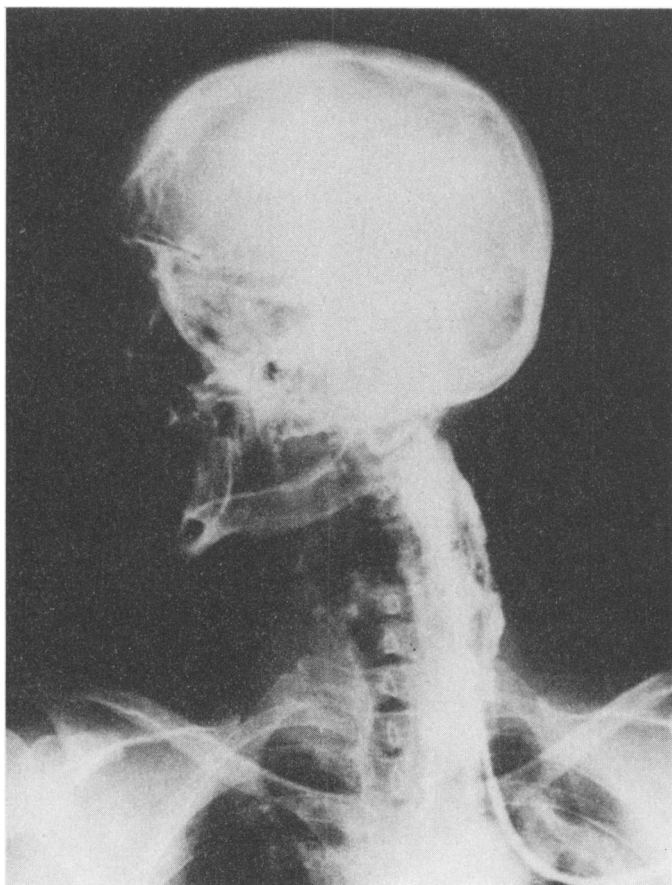


FIG. 1. Case 1. Left carotid arteriogram shows overt kink of internal carotid artery.



FIG. 2. Case 1. Internal carotid kink as seen at operation.

at the time of arteriogram in June 1957 (Fig. 8), was reported to this Association as an asymptomatic case of internal carotid tortuosity. He was seen on several occasions for other conditions but he remained neurologically asymptomatic until October of 1960 when he was admitted to the hospital in mild congestive heart failure which was controlled with diuretics. On the night of October 19, he had an episode of unconsciousness with associated cyanosis and bronchospasm which passed without neurologic residual. The following night a similar episode began with a convulsive seizure. He was left with spastic changes which were bilateral at first but remained longer in the right upper extremity. On carotid arteriogram the following day, there was marked elongation and tortuosity of the left internal carotid artery similar to that demonstrated initially on the right side. At exploration

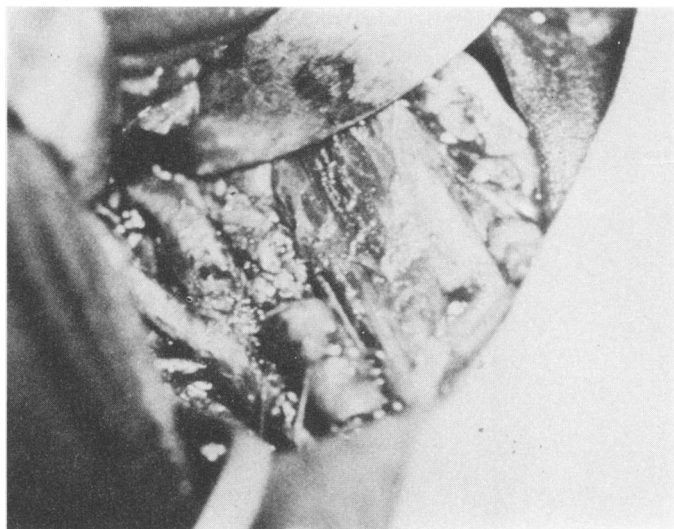


FIG. 3. Case 1. After resection of segment of common carotid (seen at side of anastomosis) the kink is eliminated.

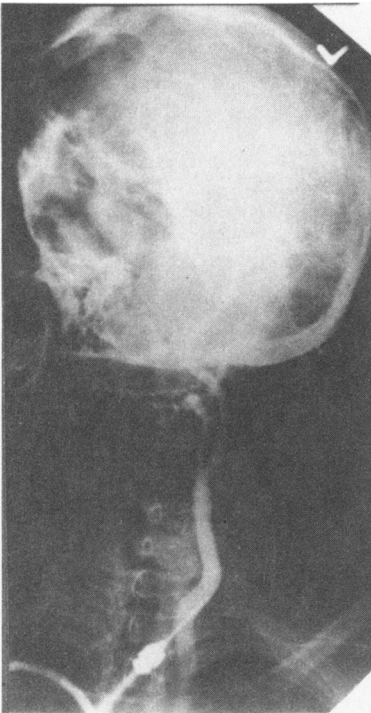


FIG. 4. Case 1. Postoperative arteriogram confirm elimination of kink.

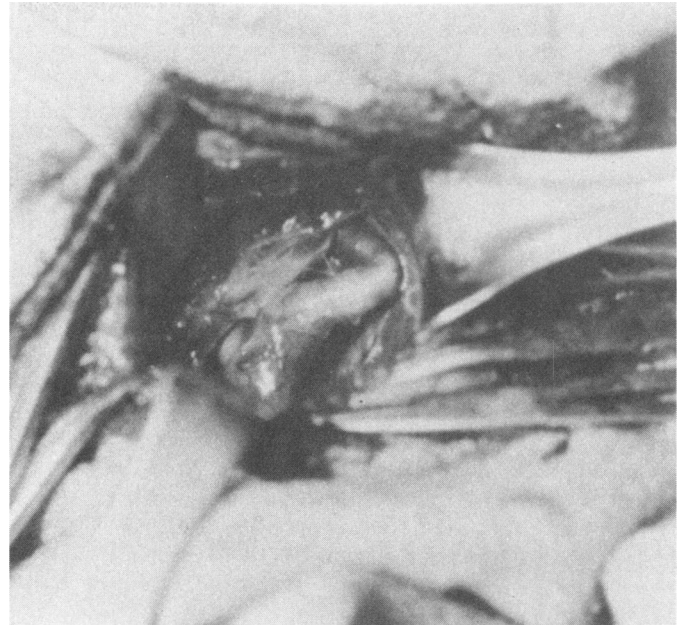


FIG. 6. Case 2. At operation, vessel is found to be kinked at point of arteriographic lucency.

under local anesthesia, approximately 5 cm. excess length was estimated to be present in the kinked and looped internal carotid artery. The proximal internal carotid artery was resected and the end of the vessel above was anastomosed into the side of the common carotid 2 cm. below the bifurcation after the fashion of Lorimer.¹⁷ Postoperatively he had no further seizures but developed increasingly severe bronchospasm with cyanosis and tachypnea, then coma, followed by death on October 23. Autopsy showed massive bilateral pneumonia.

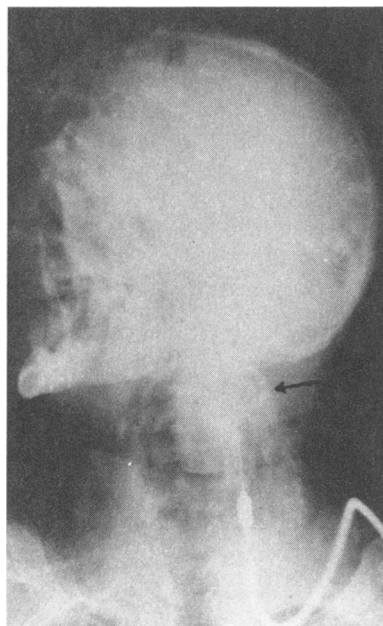


FIG. 5. Case 2. Arrow points to band-like lucency seen across artery.

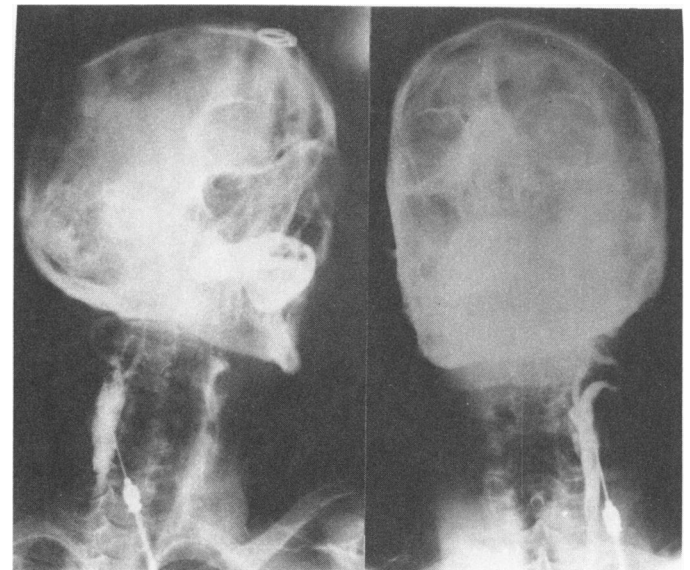


FIG. 7. Case 3. A, Right and B, Left, carotid arteriograms showing bilateral marked elongation.

Analysis of these cases plus the remainder of our series, a total of 138 patients upon whom 149 operations were performed, has been carried out. The youngest patient was 45 years of age; the oldest 93. One hundred and one of the patients were between 56 and 75 years of age. There were 66 men and 72 women. One hundred and twenty-six were Caucasian and 12 were Negro. Blood pressure on initial examination was greater than 160 systolic or 90 diastolic or both in 60 per cent of the

patients. Arteriographic evaluation of the internal carotid arteries showed that elongation was bilateral in 102 patients. It was unilateral in 20 patients and was not determined in eight. In 56 instances, an arteriosclerotic plaque was associated with elongation, and in three the plaque was stenosed and was the primary cause of symptoms. There was no significant plaque associated in 90 instances. The indication for operation (Table 1) was transient ischemic attack in 56 operations, stroke in 47 operations, vertigo in 21, blackout in 19, progressive mental deterioration in four and asymptomatic pulsating retropharyngeal mass in one. The term *transient ischemic attack* as used here includes an attack in which there is a focal neurologic deficit due to selective ischemia of one portion of the brain. Vertigo and blackout represent transient ischemic attacks in which the ischemia is diffuse and which differ in the degree of severity of the ischemia. They are classified separately for purposes of analysis. In two of the patients with stroke the condition involved first the one side and then the other. Of the patients who had vertigo, two had opposite arteries operated upon for one of the other indications.

As for the procedure which was carried out (Table 2), the common carotid artery was resected in seven instances, in three of which endarterectomy of the bifurcation was carried out concomitantly. The carotid bifurcation was resected with end-to-end anastomosis of the internal to the common carotid artery in 106 instances. Three of these required supplemental endarterectomy of the proximal internal carotid. In 34 instances, transplantation of the artery under a sternomastoid muscle flap was carried out, and in two a plastic procedure to the artery was done. The right side was operated on in 62 patients and the left in 65. Both sides were operated on in 11 patients. In each instance evaluation of the availability of

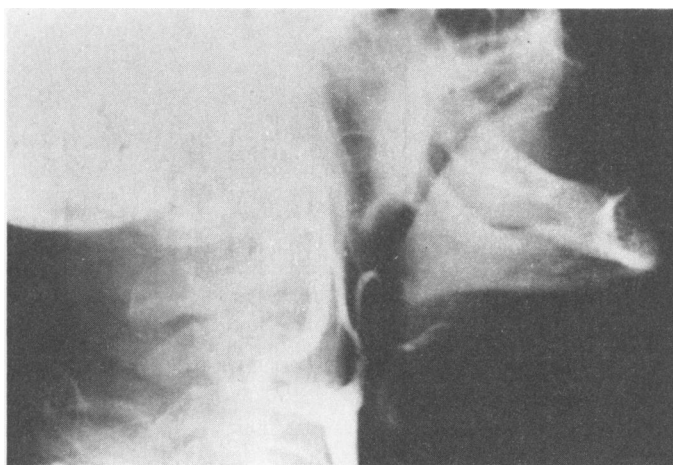


FIG. 8. Case 4. Right carotid arteriogram shows elongation of internal carotid which presented as aneurysmoid pulsation in neck.

TABLE 1. *Indication For Surgery*

T. I. A.	56
Stroke	47
Vertigo	21
Blackout	19
Progressive Mental Deterioration	5
Pulsating Retropharyngeal Mass	1

collateral flow by compression of the common carotid artery on the side to be operated for a period of 2 minutes was carried out. The occurrence of air hunger, aphasia, vertigo or more severe neurologic deficit was considered a positive test and occurred on 49 occasions, of which five were negative on the opposite side. The test was negative 95 times and, of course, five of these were positive on the opposite side. Two patients changed from positive preoperatively to negative on the table, and one changed from negative preoperatively to positive on the table. Anesthesia used (Table 3) was general with hypothermia in 16 operations, general with hypothermia plus shunt one time, general with shunt alone one time and general anesthesia without any measure to provide collateral flow was carried out five times. A total of 23 operations were performed under general anesthesia. The procedures under hypothermia and/or with shunt were employed early in the series in patients in whom the carotid compression test was positive. Local anesthesia was employed in 126 operations, and since August of 1966 all except four operations have been done under local anesthesia, carrying out transplantation in those who were positive to compression.

In the assessment of results a grade of excellent, good, fair or poor was assigned. Excellent signifies complete recovery from any neurologic deficit and remaining asymptomatic since. Good indicates a case in which the course of the disease has been unequivocally altered but in which minor symptoms may persist, such as occasional slight vertigo in a patient who was experiencing blackouts, or failure to recover totally from the neurologic deficit of a stroke. A grade of fair was assigned when the patient was better than preoperatively, but continued to have symptoms which were troublesome. Poor was used to denote patients who died in the hospital or who failed to improve. They have been analyzed individually as to the cause of failure. A single grade was assigned to patients who had both sides operated on for the same indication. If the two sides were operated upon for sep-

TABLE 2. *Operative Procedure*

Resection Common Carotid	7
Resection Carotid Bifurcation	106
Carotid Transplant	34
Arterioplasty	2

TABLE 3. Anesthesia*

General Alone	5
General plus Hypothermia	16
General plus Hypothermia with Shunt	1
Local with Shunt	1
Local	126

* Since 1966, local anesthesia has been used in all but four cases.

arate indications, a separate grade was given for each. Except for hospital deaths (Table 4), patients who were available for follow-up for less than 1 year were excluded from final assessment. The basis of evaluation included personal interview and examination, evaluation by the family physician, or questionnaire answered by the patient or his family, or combinations of these. Follow-up assessment was carried out in 107 patients. Analysis of the series as a whole shows our results to be excellent in 28 per cent, good in 44 per cent, fair in 10 per cent and poor in 18 per cent. When viewed with respect to indication for operation (Table 5), it becomes apparent that the best results were obtained in those operated for transient ischemic attacks (TIA), where the grade was excellent or good in 82 per cent of the cases. Worst results, conversely, were found in those operated upon for progressive mental deterioration, although the one good result obtained in this group was significant, occurring in a condition generally considered to be hopeless.

Discussion

In the 14-year period since our original presentation, a number of authors have reported the clinical relationship of carotid elongation to cerebrovascular insufficiency.^{2,4,5,7,10-13,17,19-21,25,28,29} In addition Brice³ and Derrick⁶ have investigated the relationship between kinking and reduction of flow in arteries.

Concerning its etiology, carotid elongation in children has given credence to the theory of its congenital origin due to failure of embryonic absorption of the third aortic arch as postulated by Kelley.¹⁶ However, the failure to develop symptoms of cerebrovascular insufficiency in any

TABLE 4. Cause of Death

	In Hospital	Late
Original Stroke	2	4
Contralateral Stroke		3
Other CVA	1	3
Cardiac	3	5
Cancer		3
Pulm. Embolism		1
Suicide		1
Pneumonia		1
Unknown		7
Total	6	28

TABLE 5. Results*

Tia (38)	E	17	45%
	G	14	
	F	2	
Stroke (38)	P	5	21%
	E	8	
	G	13%	
	F	6	
Vertigo (16)	P	8	21%
	E	2	
	G	12%	
	F	2	
Blackout (12)	P	4	26%
	E	3	
	G	25%	
	F	1	
PMD (5)	P	0	60%
	E	0	
	G	1	
	F	1	
Total Series			
	E	8	28%
	G	67%	44%
	F	8%	10%
	P	0	18%

* Includes follow-up study from 1-14 years. Except for hospital deaths short-term results are not included.

of our patients before the age of 45, many of whom still had no atherosclerotic narrowing of the collateral vessels, would tend to suggest an acquired condition. Additionally, the familiar progressive elongation of the aorta and other arteries, and the demonstration by Henley *et al.*¹² of degenerative changes in the walls of the involved carotid arteries in adults, tend to support the latter theory. As we have developed experience with this entity over the years, it has become apparent that in many instances it is selective elongation of the artery to a greater extent than that of its adventitia that leads to the tendency to buckle and kink. This in itself explains the rarity of obstructive symptoms in children in whom a congenital elongation should involve the muscularis and adventitia equally.

Since the outset we have been convinced that the development of symptoms depends upon the fortuitous positioning of the head in such a way that the vessels are kinked rather than merely elongated. While we have

seen three patients with complete obstruction due to kinking and many with lesser degrees of overt kinking on arteriograms, we also look for elongation which might logically be expected to produce kinking though such is not present at the time of the arteriogram. Roberts *et al.*²⁴ demonstrated that the neutral position of the head is the one most favorable to full cerebral blood flow. It is in this position that most angiograms are made. Roberts also demonstrated in a group of cadavers of all age groups that flow ceased in at least one of the four vessels supplying the brain at some point throughout the range of "normal" positions in every subject studied. The older the patient, the more readily was flow occluded. Kinking must be considered as a dynamic affair with symptoms dependent not only on the degree but on the time interval involved. In our series, the patient frequently awakened with neurologic deficit. Presumably it is easier for the vessel to remain in an unfavorable position for extended periods during sleep.

We have placed great reliance upon the carotid compression test preoperatively to estimate the relative safety of temporary occlusion of carotid flow. We feel that it is always best to resect the carotid bifurcation if it contains a plaque, however small. In 94 resections in which the compression test was negative, the only permanent neurologic deficit occurred before the vessel was to be occluded, presumably due to middle cerebral thrombosis. The test should be repeated on the table before committing oneself to resection because in one instance in this series the test changed from negative preoperatively to positive on the table. If the test is positive, then procedures requiring occlusion of the vessel should be avoided if possible, for in 18 resections under such circumstances significant increase in neurologic deficit occurred twice despite general anesthesia with hypothermia.

Analysis of our poor results (Table 6) suggests that in a significant number of patients, kinking of the carotid arteries was not responsible for the clinical symptoms. Death in the hospital of three patients from myocardial infarction, occurring after uneventful operation under local anesthesia, emphasizes the need for careful evaluation of the cardiac status. Obviously Stokes-Adams attacks can lead to many of the symptoms included as indications for operation. Hypertension *per se* did not seem to mediate against a good result, but hypertensive encephalopathy must be considered as a cause of vertigo or TIA, and cerebral hemorrhage must be eliminated as a cause of stroke. Diffuse intracerebral atherosclerosis as the primary cause of symptoms is especially difficult to eliminate, since it is so difficult to demonstrate angiographically and is present coincidentally with some frequency. We have learned to defer operation in the patient with a severe neurologic deficit until it becomes apparent

TABLE 6. *Analysis of Poor Results*

Tia (5)	Unimproved	2
	Operative Complication (SBE)	1
	Hospital Death	2
	(a) Myocardial Infarct	
	(b) Pneumonia	
Stroke (8)	Failure to Recover from Stroke (2 Died in Hospital)	5
	Hemorrhage into Infarct	1
	Recurrent Stroke (Kink Not Relieved by Transplant)	1
	Hospital Death (Myocardial Infarct)	1
Vertigo (4)	Unimproved	2
	Hospital Death	2
	(a) Myocardial Infarct	
	(b) Cerebral Hemorrhage	
PMD (3)	Unimproved	2
	Hemiparesis on Table	1

that significant improvement will occur, for carotid surgery is prophylactic and not therapeutic.

Summary and Conclusion

Our experience with 149 operations performed on 138 patients with carotid elongation and kinking associated with symptoms of cerebrovascular insufficiency has been reported. The condition is felt to represent an acquired degenerative process which permits relatively greater elongation of the muscular wall of the artery than its adventitia. Symptoms depend upon the fortuitous positioning of the head to cause kinking and upon the time it remains so. The condition is bilateral in the great majority of patients. Hypertension, heart disease and cerebral atherosclerosis are frequently present as well and must be eliminated as primary causes for symptoms. Cerebral vascular insufficiency due to carotid elongation must remain a diagnosis by exclusion, but when such has been accomplished the very high percentage of good results should lead to a firm recommendation of an operative procedure which can be done in almost every instance under local anesthesia with virtually no risk and minimal morbidity. We prefer resection of the carotid bifurcation with end-to-end anastomosis in patients who tolerate compression of the common carotid artery for 2 minutes without symptoms. For those who cannot, transplantation of the carotid under a sternomastoid muscle flap has been a satisfactory alternative in most instances.

References

1. Bahnson, H. T., Spencer, F. C. and Quattlebaum, J. K. Jr.: Surgical Treatment of Occlusive Disease of the Carotid Artery. *Ann. Surg.*, **149**:711, 1959.
2. Barnes, W. T. *et al.*: Carotid Insufficiency Due to Elongation and Kinking of the Internal Carotid Artery. *Penn. Med. J.*, **68**:41, 1965.
3. Brice, J. G., Dowsett, D. J. and Lowe, R. D.: Haemodynamic Effects of Carotid Artery Stenosis. *Br. Med. J.*, **2**:1363, 1964.

4. Culligan, J. A.: Buckling and Kinking of the Carotid Vessels in the Neck. *Minnesota Med.*, **43**:678, 1960.
5. Derrick, J. R.: Carotid Kinking and Cerebral Insufficiency. *Geriatrics*, **18**:272, 1963.
6. Derrick, J. R., Estess, M. and Williams, D.: Circulatory Dynamics in Kinking of the Carotid Artery. *Surgery*, **58**:381, 1965.
7. Derrick, J. R. and Smith, T.: Carotid Kinking as a Cause of Cerebral Insufficiency. *Circulation*, **25**:849, 1962.
8. Edington, G. H.: Tortuosity of Both Internal Carotid Arteries. *Br. Med. J.*, **2**:1526, 1901.
9. Fisher, A. G. T.: Sigmoid Tortuosity of the Internal Carotid Artery and Its Relation to the Tonsil and Pharynx. *Lancet*, **2**:128, 1915.
10. Freeman, T. R. and Lippitt, W. H.: Carotid Artery Syndrome Due to Kinking: Surgical Treatment in Forty-four Cases. *Am. Surg.*, **28**:745, 1962.
11. Gass, H. H.: Kinks and Coils of the Cervical Carotid Artery. *Surg. Forum*, **9**:721, 1959.
12. Henley, W. S., Cooley, D. A., Gordon, W. B. Jr. and DeBakey, M. E.: Tortuosity of the Internal Carotid Artery. Report of Seven Cases Treated Surgically. *Postgrad. Med.*, **31**:133, 1962.
13. Hohf, R. P.: The Clinical Evaluation and Surgery of Internal Carotid Insufficiency. *Surg. Clin. North Am.*, **47**:71, 1967.
14. Hsu, I. and Kisten, A. D.: Buckling of the Great Vessels. *AMA Arch. Int. Med.*, **98**:712, 1956.
15. Jackson, Joseph L.: Tortuosity of the Internal Carotid Artery and Its Relation to Tonsillectomy. *Can. Med. Assoc. J.*, **29**:475, 1933.
16. Kelly, A. B.: Tortuosity of the Internal Carotid Artery in Relation to the Pharynx. *J. Laryngol. Otol.*, **40**:15, 1925.
17. Lorimer, W. S. Jr.: Internal Carotid Artery Angioplasty. *Surg. Gynecol. Obstet.*, **113**:783, 1961.
18. McKenzie and Woolf, C. F.: Carotid Abnormalities and Aneurysm. *J. Laryngol. Otol.*, **73**:596, 1959.
19. Metz, H., Murray-Leslie, R. M., Bannister, R. G., Bull, J. W. and Marshall, J.: Kinking of the Internal Carotid Artery in Relation to Cerebrovascular Disease. *Lancet*, **1**:424, 1961.
20. Najafi, H., Javid, H., Dye, W. S., Hunter, J. A. and Julian, O. C.: Kinked Internal Carotid Artery. *Arch. Surg.*, **89**:134, 1964.
21. Parrott, J. C.: Internal Carotid Artery Insufficiency. *Am. J. Surg.*, **108**:777, 1964.
22. Quattlebaum, J. K. Jr., Upson, E. T. and Neville, R. L.: Stroke Associated with Elongation and Kinking of the Internal Carotid Artery. *Ann. Surg.*, **150**:824, 1959.
23. Riser, M., Gerard, J. and Ribaut, L.: Dolichocarotide interne avec syndrome vertigineux. *Rev. Neurol.*, **85**:145, 1951.
24. Roberts, B., Hardesty, W. H., Holling, H. E., Reivich, M. and Toole, J. F.: Studies on Extracranial Cerebral Blood Flow. *Surgery*, **56**:826, 1964.
25. Rundles, W. R. and Kimbell, F. D.: The Kinked Carotid Syndrome. *Angiologica*, **20**:177, 1969.
26. Skillern, P. G.: Anomalous Internal Carotid Artery and Its Significance in Operations on Tonsils. *JAMA*, **60**:172, 1913.
27. Smith, G. M.: Tortuosity of the Internal Carotid Artery. *Br. Med. J.*, **1**:1601, 1902.
28. Spencer, W. J.: Pseudostroke: Acute Cerebrovascular Insufficiency with Congenital Carotid Kinking. *JAMA*, **186**:76, 1963.
29. Weibel, J. and Fields, W. S.: Tortuosity, Coiling, and Kinking of the Internal Carotid Artery II. *Neurology*, **15**:462, 1965.

DISCUSSION

DR. ALTON OCHSNER, JR. (Metairie): I think this is such a significant paper that many surgeons in this audience will be looking now for these carotid elongations and kinks. I would like to point out, however, that our neurologist confreres, with rare exceptions, think they have no significance at all. Of course, I do not agree with them; I agree with Dr. Quattlebaum that they are significant.

In the last 4 years we have had 40 cases not associated with any significant stenosis in the carotid artery. A third of these are my own cases; the others are cases of two young men that I assisted, one of whom, Dr. Ricardo Del Real, is now practicing here in Boca Raton.

From this experience, I think, particularly because there may be those who will be interested in looking into this subject further in terms of diagnosis and treatment, I would like to make a few cogent remarks.

First, the only way this can be diagnosed is by angiography, and one does not look for murmurs, because there should not be any murmurs. Angiograms must be performed on people with symptoms of cerebrovascular insufficiency without murmurs.

[Slide] This is a continuous condition, I think, with beginning elongation and folding back upon itself, and eventually complete looping or kinking. It is in these early stages that the symptoms are most likely to be seen. This complete looping is seen as an incidental finding sometimes, and may lead the neurologists to believe that none of this is important. This is, of course, one of the few diseases that I know of in which in the late stages the symptoms are less.

[Slide] In taking angiograms, one must recognize that this condition is sometimes seen only in one plane, and it is usually the AP projection. This is a lateral projection of the carotid artery,

and if that is all you had, you would miss this condition. However, there is a kink, or an uncoiling which can lead to kinking, when the X-ray is taken in the AP position.

[Slide] This is just another example of the same thing. In the lateral position there is no evidence of kinking. In the AP position there is a kink.

[Slide] This is just one patient with X-rays in AP, with the head turned to one side and the head turned to the other side. The kinking is seen in the AP position when the head is turned to one side, but not when the head is turned to the other side. So taking the X-ray in two planes may be necessary to completely rule in or rule out this condition.

The amount of kinking or coiling can be lessened or greatedened by the position of the head, and sometimes if you want to take real effort, [slide] you can actually demonstrate the occlusion. This is an AP view of an uncoiled, potentially kinkable internal carotid artery, with the head extended.

[Slide] This is the same patient with the head flexed, and the blood flow is cut off there.

Regarding comments about treatment, we would have to take issue with the authors about the use of local anesthesia. We think that general anesthesia is important in the management of this condition, because if the kinking is to be completely overcome, the internal carotid artery must be dissected up to the base of the skull. Sometimes the kink will not be found except at the very base of the skull. In order to do this, you have to have retraction on the base of the skull, and I think it is too much of a hardship on both the patient and the surgeon to do it under local anesthesia.

Do not pull on the carotid! I think part of the etiology of this condition is the lack of elastic tissue, and I can assure you that traction on the carotid will put it apart. I have done this in two instances. We recovered from it, but I would not want anybody