Internal Carotid Artery Redundancy is Significantly Associated With Dissection.

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Internal Carotid Artery Redundancy Is Significantly Associated With Dissection

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Background and Purpose Redundant internal carotid arteries have been considered a risk factor in tonsillectomy, adenoidectomy, and surgical treatment of peritonsillar abscess and also a potentially treatable cause of stroke. However, an association between internal carotid artery redundancy and spontaneous dissection has not yet been clearly demonstrated.

Methods We reviewed, for spontaneous carotid artery dissection, records of all patients admitted to our institution during the period from 1986 through 1992 with the diagnosis of stroke or transient ischemic attack. We also reviewed 108 percutaneous cerebral arteriograms performed between September 1992 and December 1992 for presence of carotid artery redundancies.

Results Thirteen patients exhibited spontaneous dissection. Of these, 8 of 13 (62%) patients and 13 of 20 (65%) internal carotid arteries, viewed to the siphon, had significant redundancies, kinks, coils, or loops. Of 108 consecutive arteriograms of patients without dissection, in which 187 internal carotid arteries were viewed to the siphon, there were 20 (19%) patients and 22 (12%) of 187 vessels with significant redundancy. Five patients in the dissection group and 2 in the nondissection group had bilateral internal carotid artery redundancy ($P=.0019$ and $P=.0001$, respectively).

Conclusions We found a significant correlation between internal carotid artery redundancy and dissection, particularly if redundancy is present bilaterally. (***Stroke.* 1994;25:1201-1206.**)

Key Words • carotid arteries • dissection • risk factors

Divergent opinions have been expressed concerning the clinical significance of redundant internal carotid arteries (ICAs). When first described in 1741, these aberrant vessels were referred to as anatomic curiosities.[1] They were assigned clinical significance when appreciated as a risk factor in tonsillectomy, adenoidectomy, and surgical treatment of peritonsillar abscess. Later these vessels were considered a potentially treatable cause of stroke,[2] but indications for surgery, natural history, and association with stroke remained controversial.[3-9] An association between ICA redundancy and spontaneous dissection has not been clearly demonstrated. However, one report in the neurosurgical literature raised the question of an association between ICA redundancy and stroke based on findings of ICA coils in two of nine cases of spontaneous dissections of the extracranial ICA.[10] In a review of ICA dissection by Gee et al,[11] three of four patients with ICA dissection were found to have significant ICA redundancy. Some of these redundant vessels were only considered redundant with repeated arteriogram after recanalization was demonstrated noninvasively.

Because there appeared to be an association between ICA dissection and redundancy, we examined the prevalence of carotid artery redundancy in patients with and without dissection.

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Subjects and Methods

We retrospectively reviewed, for spontaneous ICA dissection, the records of all patients admitted to our hospital between January 1986 and December 1992 with the diagnosis of stroke or transient ischemic attack who received noninvasive carotid artery testing and/or cerebral angiography. An ICA dissection was defined arteriographically by the "classic string" sign in the partially occluded vessels or by a pointed tapered occlusion of the artery. The arteriograms of these patients were evaluated for ICA redundancy in the same manner as the nondissection group. Spontaneous dissection was defined as dissection with no obvious clinical cause or with "trauma" deemed trivial and unlikely to cause dissection. We eliminated only one case of bilateral dissection resulting from severe trauma due to motor vehicle accident. None of the patients with dissection had arteriographic evidence of fibromuscular dysplasia. Representative case subjects are described in Table 1.

We also reviewed 108 consecutive percutaneous transfemoral cerebral arteriograms, performed at our institution between September 1992 and December 1992, for the presence of carotid artery redundancies. The demographics of the control case groups are displayed in Table 2. Redundant vessels were classified as loops, coils, and kinks (Fig 1). Kinks produce a sharp bend in the vessel with an angle $\leq 90°$, coils produce a 360° turn in the vessel, and loops produce a C- or S-shaped deformity with two turns in the vessel with angles $\leq 90°$.

All arteriographic images in the dissection group were reviewed by two examiners (J.E.C. and A.D.R.-G.). The best select films, showing the pathology of dissection, were reviewed and accepted by all authors. Representative cases are shown in Figs 2 and 3. The nondissection group was evaluated by an independent examiner (P.J.B.). All redundant carotid arteries were traced and the results examined for consistency by all authors.

Differences in prevalence between the nondissection and dissection groups were analyzed using a χ^2 test or Fisher's

TABLE 1. Clinical Description of Internal Carotid Artery Dissection in 13 Consecutive Patients Seen at Lehigh Valley Hospital and the Presence or Absence of Redundancy

Pt	Sex/Age, y	History	Signs	ICA
1	M/40	HT	Anosognosia, L hemiplegia, R Horner's syndrome, sensory loss L face	R, D, C L, ND, C
2	F/39	HA, BCP, AC-I	Lethargic, anosognosia, L hemiplegia, R hemianesthesia	R, D, C L, ND, C
3	M/45	Previous stroke, S/P aneurysm repair, transient L hemiparesis	Normal exam	R, D, C L, ND, C
4	F/81	R amaurosis fugax	Normal exam	R, D, loop L, D, NV
5	M/62	HT, DM, CAD, asymptomatic	Normal exam	R, ND, K L, D, NV
6	F/49	Transient R amaurosis fugax, diplopia	Lethargic, R hemiplegia, R afferent pupil, bilateral Babinski, general hyperreflexia	R, ND, NR L, D, NR
7	M/56	Stroke, S/P OHS	R hemiplegia	R, ND, NR L, D, NV
8	M/47	Transient L hemiparesis	Normal exam	R, D, NV L, ND, NR
9	F/65	Transient R hemiparesis	Normal exam	R, ND, NR L, D, NV
10	M/74	DM, HT	R hemiparesis, aphasia	R, D, NV L, ND, NR
11	M/49	CAD, VA	R hemiparesis, aphasia	R, ND, C L, NV
12	F/54	Migraine, HT	L hemiparesis	R, D, NV L, ND, K
13	M/45	CAD	Gerstmann's syndrome, aphasia	R, ND, C L, D, C

Pt indicates patient; ICA, internal carotid artery; HT, hypertension; HA, headache; BCP, birth control pills; AC-I, Arnold Chiari I; S/P, status post; L, left; R, right; DM, diabetes mellitis; CAD, coronary artery disease; OHS, open heart surgery; VA, ventricular arrhythmia; D, dissection; C, coil; ND, no dissection; NV, not visualized above dissection; K, kink; and NR, no redundancy.

TABLE 2. Demographic Comparison Between Dissection Patients and Control Subjects for Age, Diabetes, Smoking, Hypertension, and Reason for Study

	Control Subjects (n=108)	Dissection Patients (n=13)	*P*
Men	76 (70.4)	8 (61.5)	
Women	32 (29.6)	5 (38.5)	.733
Age range, y	39-89	39-81	
Age, y (mean±SEM)	68.0±0.8	54.3±3.6	.002
Diabetes	35 (32.4)	4 (30.8)	1.000
Smoker	64 (59.3)	4 (30.8)	.049
Hypertension	75 (69.4)	4 (30.8)	.011
Reason for study			
Transient ischemic attack	37 (34.3)	4 (30.8)	.829
Asymptomatic carotid disease	35 (32.4)	1 (7.7)	.063
Stroke	29 (26.9)	8 (61.5)	.021
Syncope	3 (2.8)	0	.709
Seizure	1 (0.9)	0	.893
Vascular mass	1 (0.9)	0	.893
Aneurysm	1 (0.9)	0	.893

Numbers in parentheses are percentages.

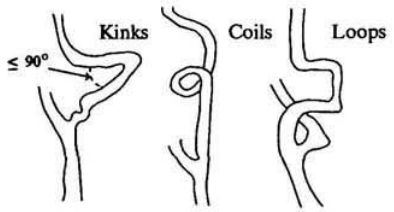

FIG 1. Drawings of kinks, coils, and loops as seen in our patient population.

exact test when one or more of the expected frequencies were less than 5. A McNemar test was used for testing differences in correlated proportions. All probability values are two-sided unless otherwise noted. We set the α-levels for statistical significance at $P<.01$.

Results

In the dissection group, 8 of 13 (62%) patients and 13 of 20 (65%) vessels visualized to the siphon had redundancies. In 6 of the dissected vessels, the segment distal to the dissection was not visualized. Five patients (38.5%) had bilateral redundancies. The distribution of redundancy type in the 13 redundant vessels was as follows: 2 loops, 9 coils, and 2 kinks.

The prevalence of ICA redundancy in the nondissection group was 20 of 108 (19%) patients and 22 of 187 (12%) vessels visualized. There were only 2 of 108 (2%)

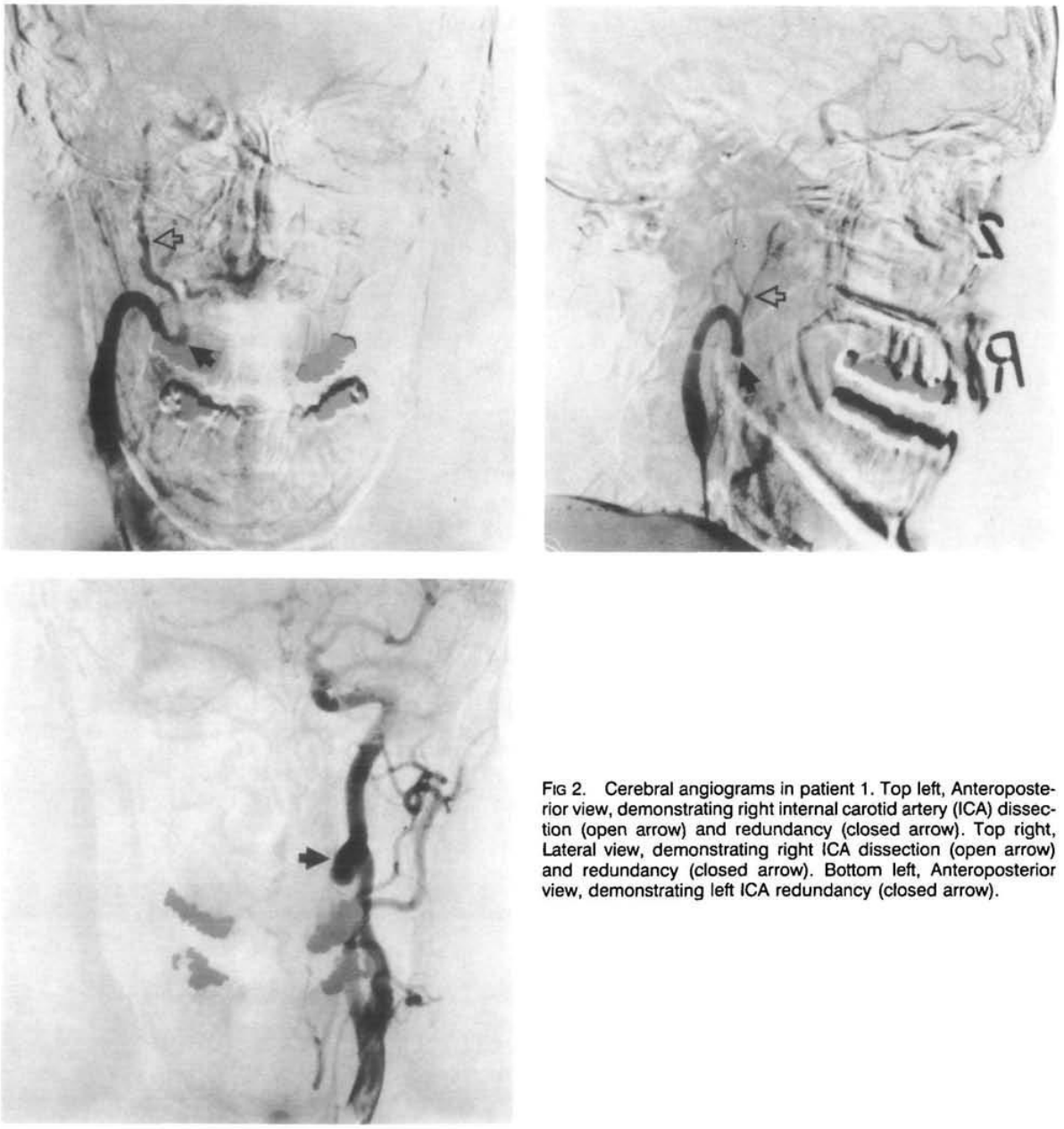

FIG 2. Cerebral angiograms in patient 1. Top left, Anteroposterior view, demonstrating right internal carotid artery (ICA) dissection (open arrow) and redundancy (closed arrow). Top right, Lateral view, demonstrating right ICA dissection (open arrow) and redundancy (closed arrow). Bottom left, Anteroposterior view, demonstrating left ICA redundancy (closed arrow).

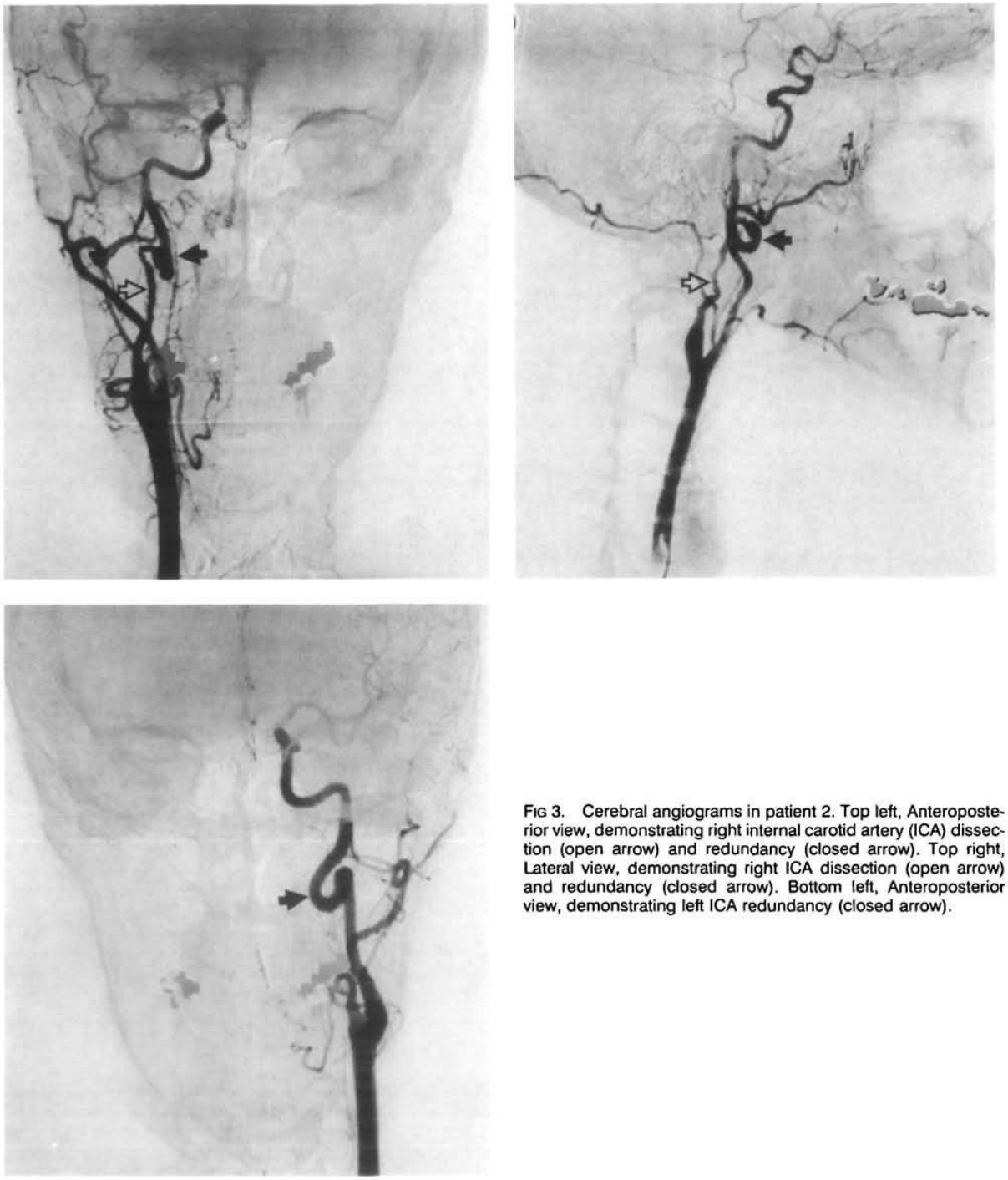

FIG 3. Cerebral angiograms in patient 2. Top left, Anteroposterior view, demonstrating right internal carotid artery (ICA) dissection (open arrow) and redundancy (closed arrow). Top right, Lateral view, demonstrating right ICA dissection (open arrow) and redundancy (closed arrow). Bottom left, Anteroposterior view, demonstrating left ICA redundancy (closed arrow).

patients with bilateral redundancy. Complete occlusion without dissection was found in 24 of 108 (22%) patients. Of these, 6 patients had redundancy in the visualized, nonoccluded vessel (25%). Five vessels were not imaged by the arteriographer at the time of the arteriogram. The distribution of redundancy type in the 22 redundant vessels was as follows: 3 loops, 10 coils, and 9 kinks.

A comparison between a control group of patients and the dissection patients is given in Table 2. With the exception of age (60.8±0.8 versus 54.3±3.6 years, $P=.002$), the control patients and dissection patients are equivalent in regard to sex ratio, incidence of diabetes, smoking, hypertension, transient ischemic attack, asymptomatic carotid disease, stroke, syncope, seizure, vascular mass, and aneurysm ($P=.738$, $P=1.000$,

TABLE 3. Comparison Between Nondissection and Dissection Populations for Internal Carotid Artery Loops, Coils, and Kinks

	Nondissection	Dissection	*P*
Patients with redundant ICA	20/108 (18.5)	8/13 (61.5)	.0019*
Patients with bilateral redundancy	2/108 (1.9)	5/13 (38.5)	.0001*
Vessels with redundancy	22/187 (11.8)	13/20 (65.0)	.0093†
Types of redundancy			
Loops	3/22 (13.6)	2/20 (10.0)	.750*
Coils	10/22 (45.5)	9/20 (45.0)	.976‡
Kinks	9/22 (40.9)	2/20 (10.0)	.035*
Patients with occlusion, nondissection, and redundancy			
Redundancy contralateral to occlusion			6/24
Redundancy in nondissection population without occlusion			14/79
			P=.430‡

ICA indicates internal carotid artery. Numbers in parentheses are percentages.
*Fisher's exact test.
†McNemar test.
‡χ^2 test with 1 *df*.

P=.049, P=.011, P=.829, P=.063, P=.021, P=.709, P=.893, P=.893, and P=.893, respectively) (Table 2).

Comparisons between the dissection group and nondissection groups demonstrated significant differences in patients with redundant ICAs (P=.0019, Fisher's exact test), patients with bilateral redundancy (P=.0001, Fisher's exact test), and the number of redundant vessels in each group (P=.0093, McNemar test) (Table 3). There was no significant difference between the two groups and subgroups of redundancy types in regard to loops, coils, and kinks (P=.750 [Fisher's exact test], P=.976 [χ^2 test], and P=.035 [Fisher's exact test], respectively). As shown in Table 3, there was no significant difference between the nondissection group with and without one vessel occluded and incidence of ICA redundancy (P=.430, χ^2 test).

Discussion

The prevalence of ICA redundancy in the general population is not known. Estimates of the prevalence of kinks and coils in the general population range from 4% to 66%, including autopsy series, and 3% to 5% of those people undergoing carotid endarterectomy in angiographic series.[1,3] We found ICA redundancy in 19% of patients in a consecutive angiographic series. In contrast to this, we found a 62% prevalence of ICA redundancy in our population of patients with carotid dissections. There appears to be a relation between ICA redundancy and dissection, particularly if the redundancy occurs bilaterally.

Perhaps similarities in the pathogenesis of ICA redundancy and dissection explain this relation. ICA redundancy is postulated to have several possible etiologies.[1] It may be congenital, related to differential growth of the spine and descent of the great vessels into the chest. Redundancy has been observed in fetuses and infants; it is frequently bilateral when it occurs in children. Another possible etiology of ICA redundancy is that it is related to atherosclerotic degeneration of the ICA. Finally, it has been suggested that hemodynamic effects and lateral forces on the ICA, which is fixed at both ends, cause the vessel to lose its elasticity and become redundant with age.[12] Other authors have found that the relation to aging and hypertension remains unclear.[13]

We were concerned that because 23% of the patients in our control group had blunt, occluded vessels, there may have been a redundancy distal to the occlusion that was not visualized and hence was underreported in this group. If this were the case, however, control subjects with blunt occlusion would be expected to have a higher prevalence of carotid redundancy on the contralateral side, as was found in our dissection series. Since this was not our observation, it would appear that redundancy is not associated with nondissection occlusion.

The pathophysiology of carotid dissection can be divided into traumatic and spontaneous.[10,14] Traumatic dissection is often the result of direct injury to the carotid artery. Although minor trauma may be suggested, the etiology of spontaneous dissection is not always clear. Speculated causes for spontaneous ICA dissection include fibromuscular dysplasia, hypertension, Marfan's syndrome, syphilis, arteriopathies, and cystic medial necrosis.[15-18] Disintegration of the supportive structure of the ICA appears to be a possible common factor leading to redundancy and predisposing to dissection.

In summary, ICA redundancy and dissection are significantly associated. We need to know more regarding the pathophysiology of redundant ICAs and their natural history before establishing a causal relation.

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References

1. Busuttil R, Memsic L, Thomas D. Coiling and kinking of the carotid artery. *Vasc Surg*. 1989;2:1406-1411.
2. Patel K, Clauss R. Carotid artery coils and kinks. In: Ernst CB, Stanley JC. *Current Therapy in Vascular Surgery*. 2nd ed. Philadelphia, Pa: BC Decker, Inc; 1991:159-165.
3. Collins P, Orecchia P, Gomez E. A technique for correction of carotid kinks and coils following endarterectomy. *Ann Vasc Surg*. 1991;5:116-120.
4. Eikelboom B, Lawson J, Moll F, Taks C, Vermeulen F. The hemodynamic significance of carotid coiling. *J Cardiovasc Surg*. 1988;29:437-440.
5. Leipzig T, Dohrmann G. The tortuous or kinked carotid artery: pathogenesis and clinical consideration. *Surg Neurol*. 1986;25:478-486.
6. Weibel J, Fields W. Tortuosity, coiling, and kinking of the internal carotid artery: relationship of morphological variation to cerebrovascular insufficiency. *Neurology*. 1965;65:462-468.
7. Daskalakis M. Coiling of the carotid arteries. *South Med J*. 1987;80:387-389.
8. Chino E. A simple method for combined carotid endarterectomy and correction of internal carotid artery kinking. *J Vasc Surg*. 1987;6:197-199.
9. Mukherjee D, Inhara T. Management of the tortuous internal carotid artery. *Am J Surg*. 1985;149:651-655.
10. Luken M, Ascherl G, Correll J, Hilal S. Spontaneous dissecting aneurysms of the extracranial internal carotid artery. *Clin Neurosurg*. 1979;26:353-375.
11. Gee W, Kaupp H, McDonald K, Lin F, Curry J. Spontaneous dissection of internal carotid artery. *Arch Surg*. 1980;115:944-949.
12. Dobrin P, Schwarcz T, Baker W. Mechanism of arterial and aneurysmal tortuosity. *Surgery*. 1988;104:568-571.
13. Metz H, Murray-Leslie R. Kinking of the internal carotid artery in relation to cerebrovascular disease. *Lancet*. 1961;1:424-426.
14. Zelenock G, Huber T. Carotid artery dissection. In: *Current Therapy in Vascular Surgery*. 2nd ed. Philadelphia, Pa: BC Decker, Inc; 1991:150-154.
15. Friedman W, Day A, Quisling R, Sypert G, Rhoton A. Cervical carotid dissecting aneurysms. *Neurosurgery*. 1980;7:207-214.
16. Garcia-Merino J, Gutierrez J, Lopez-Lozano J, Marquez M, Lopez F, Liano H. Double lumen dissecting aneurysms of the internal carotid artery in fibromuscular dysplasia: case report. *Stroke*. 1983;14:815-817.
17. Mokri B, Sundt T, Houser W, Piepgras D. Spontaneous dissection of the internal carotid artery. *Ann Neurol*. 1986;19:126-138.
18. Bradac G, Kaernbach A, Bolk-Weischedel D, Finck G. Spontaneous dissecting aneurysm of cervical cerebral arteries. *Neuroradiol*. 1981;21:149-154.