Blunt Carotid Artery Trauma

Report of Two Cases and Review of the Literature

LEONARD P. KRAJEWSKI, M.D.,* NORMAN R. HERTZER, M.D.

From the Cleveland Clinic Foundation, Department of Vascular Surgery, Cleveland, Ohio

Blunt carotid artery trauma is uncommon but has been associated with severe, permanent neurologic deficits in 42% and mortality in 30% of 96 patients previously reported in the English literature. Since neurologic symptoms characteristically develop only after a latent interval and since physical evidence of significant cervical trauma often is absent, diagnosis of nonpenetrating carotid injuries with the use of arteriography usually is delayed until the appearance of obvious, frequently irreversible neurologic complications. Carotid injuries should be suspected in patients who develop monoplegia or hemiplegia following blunt craniocervical trauma, particularly if computerized tomography excludes the presence of intracranial hemorrhage. The cumulative results of a collected series of 96 patients suggest that early surgical correction of blunt carotid injuries is appropriate for patients with transient episodes of cerebral ischemia, strokes in evolution, or mild completed neurologic deficits.

SIGNIFICANT INJURIES of the carotid artery caused by blunt craniocervical trauma are distinctly unusual in comparison to the number of penetrating cervical vascular injuries encountered at urban medical centers. Seventy of the 72 patients with carotid artery trauma described by Rubio and associates[16] had stab or gunshot wounds while only two sustained nonpenetrating injuries. Yamada and associates[20] found that only 51 cases of blunt carotid injury had been reported prior to 1967. In this series, the mortality rate was 38%, and 82% of survivors experienced severe, permanent neurologic deficits. Diagnosis of carotid injuries was delayed in 94% of patients for as long as several days because neurologic symptoms either resembled those of closed head trauma or appeared only after a latent interval. Recommendations concerning the management of documented blunt carotid injuries for the most part have been anecdotal and have included both immediate surgical intervention and nonoperative treatment with the use of dexamethasone and anticoagulants.

Because of our recent experience with two patients who presented with delayed neurologic symptoms following nonpenetrating carotid artery trauma, we reviewed the English literature and found 44 additional cases of this unusual lesion which have been published since the report of Yamada and associates. The following case reports illustrate many of the typical features of blunt carotid injury that have previously been described in the collected series of 96 patients.

Case Reports

Case 1. This 15-year-old boy sustained blunt trauma to the right side of the neck on January 10, 1978, when he struck the support cable of a telephone pole while traveling at 25 mph on a snowmobile. Although he had amnesia for three hours, he remained conscious and was alert and oriented during his initial medical evaluation at a nearby hospital. The neurologic examination was normal, as were plain roentgenograms of the skull and of the cervical and thoracic vertebrae. A large hematoma was still present in the lower right side of the neck, but he had developed no neurologic symptoms by the time he was discharged from the hospital four days later.

Seventeen days after the snowmobile accident, the patient briefly lost consciousness after arising from a chair. He recovered within a few minutes, and the neurologic examination was normal when he was admitted to the Cleveland Clinic Hospital approximately two hours later. A firm, nonpulsatile mass was palpable within the anterior aspect of the right sternocleidomastoid muscle. The right carotid pulse was prominent near the clavicle but was absent at the level of the mandible. A periorbital directional Doppler examination suggested that the right internal carotid artery was occluded.

A transfemoral arch aortogram was obtained. The right common carotid artery was occluded proximal to the carotid bifurcation (Fig. 1). Retrograde flow of contrast medium in the right internal carotid artery demonstrated that the carotid bifurcation was patent (Fig. 2) and suggested that direct vascular reconstruction was

* Current Address: 2101 Forest Avenue, Suite 118, San Jose, California 95128.

Reprint requests: Norman R. Hertzer, M.D., The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, Ohio 44106.

Submitted for publication: September 10, 1979.

0003-4932/80/0300/0341 $00.80

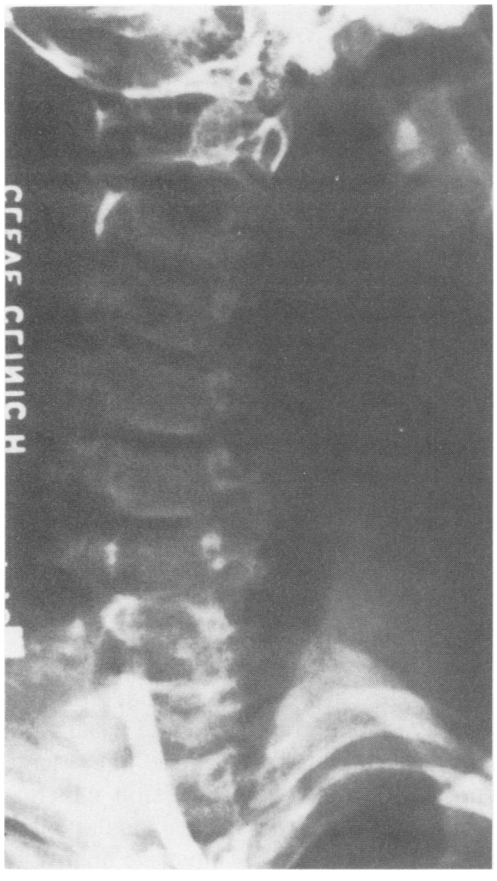

FIG. 1. Case 1, selective right carotid arteriogram. The right common carotid artery is occluded at a point proximal to the carotid bifurcation.

feasible. At the time of operation, a thrombus was found to extend to the carotid bifurcation from an area of disrupted intima in the common carotid artery (Fig. 3). After a temporary carotid shunt had been inserted, an autogenous saphenous vein graft was used to replace the segment of common carotid artery that contained injured intima and adherent thrombus. No complications occurred, and the patient has been well for 15 months following his operation.

Case 2. This 35-year-old man, who had a history of frequent dislocations of the temporomandibular joints, struck the steering wheel and the windshield of his automobile during a collision on March 20, 1978. He had only a mild headache and returned to his home following his initial examination at a nearby hospital. Three days later, he experienced a severe frontal headache and weakness of the left arm and leg. At the time he was admitted to the Cleveland Clinic Hospital, he was found to have bilateral temporomandibular dislocations, left facial weakness, and paresis of the left upper extremity. A peripheral infarction of the left retina was discovered during fundoscopic examination. The superficial temporal and carotid pulses were normal, and no cervical bruits were present. Computerized tomography of the brain was normal.

Three hours after his admission to the hospital, he developed dysarthria and progressive weakness of the left lower extremity. Transfemoral carotid arteriograms were obtained (Fig. 4a and b). Intimal disruption was identified within both internal carotid arteries, and the right internal carotid artery contained a radiolucent filling defect that appeared to represent a thrombus adjacent to the injured intima. Since obvious progression of neurologic deficits could be attributed either to impending occlusion of the right internal carotid artery or to cerebral embolization caused by the thrombus, surgical management of the right internal carotid lesion was undertaken. The carotid bifurcation contained an atheroma which was disrupted near the ostium of the internal carotid artery. A soft thrombus was adherent to the site of intimal injury (Fig. 5). A temporary carotid shunt was inserted, and carotid endarterectomy was performed. Dexamethasone and anticoagulation with sodium heparin were administered for several days following the operation. Weakness involving the left lower extremity improved dramatically, but a left hemianopsia and paresis of the left upper extremity persisted.

Eleven days following the operation, a transfemoral arch aortogram was obtained (Fig. 6). The right carotid bifurcation and the internal carotid artery were normal, and the previous subintimal defect within the left internal carotid artery was hardly perceptible. Another computerized tomogram of the brain eventually demonstrated bilateral cerebral cortical infarcts. The patient continues to have a left hemianopsia and left upper extremity monoparesis one year following his operation, but he is able to walk without disability.

Comment

Both patients described in the case reports sustained intimal tears and superimposed thrombosis following

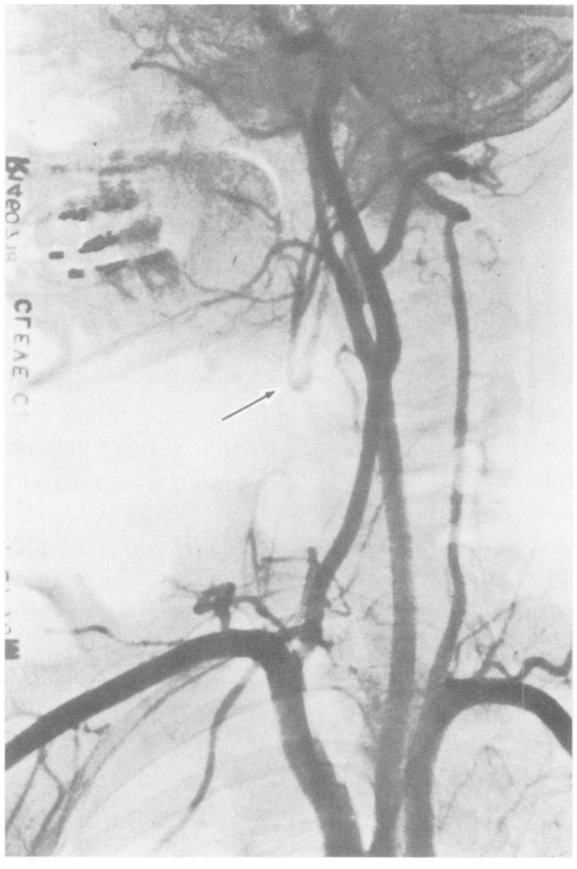

FIG. 2. Case 1, arch aortogram. The right carotid bifurcation (arrow) is opacified by retrograde flow of contrast medium in the right internal carotid artery.

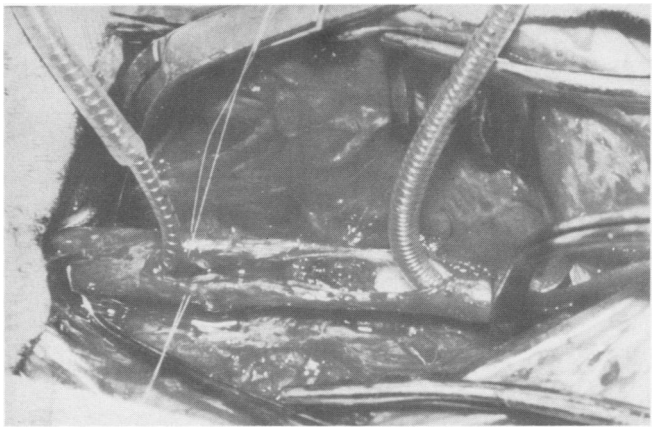

FIG. 3. Case 1, operative photograph. A thrombus is present at the site of intimal injury within a segment of the common carotid artery proximal to the carotid bifurcation.

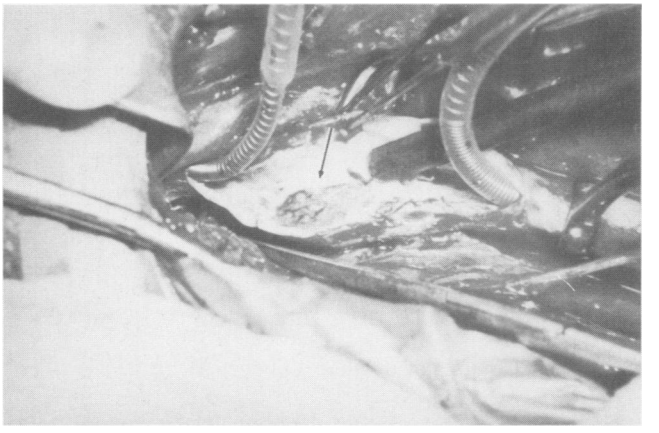

FIG. 5. Case 2, operative photograph. The thrombus which appeared as a radiolucent filling defect in the internal carotid artery during arteriography is indicated by the arrow.

blunt carotid artery trauma. Each developed neurologic symptoms only after a latent interval of several days, and neither was suspected to have a significant cervical vascular injury at the time of his initial medical evaluation. While complete carotid occlusion was responsible for the eventual appearance of symptoms in patient 1, cerebral embolization probably was the cause of bilateral cortical infarcts and focal retinal ischemia in patient 2. The suspected mechanism of injury in patient 2, compression of both internal carotid arteries between the dislocated mandible and the transverse processes of the cervical vertebrae, is particularly unique. To our knowledge, this is the only patient reported to survive bilateral simultaneous carotid artery injuries. We could find only one other similar case in which bilateral carotid artery injuries were confirmed by post mortem examination.[21]

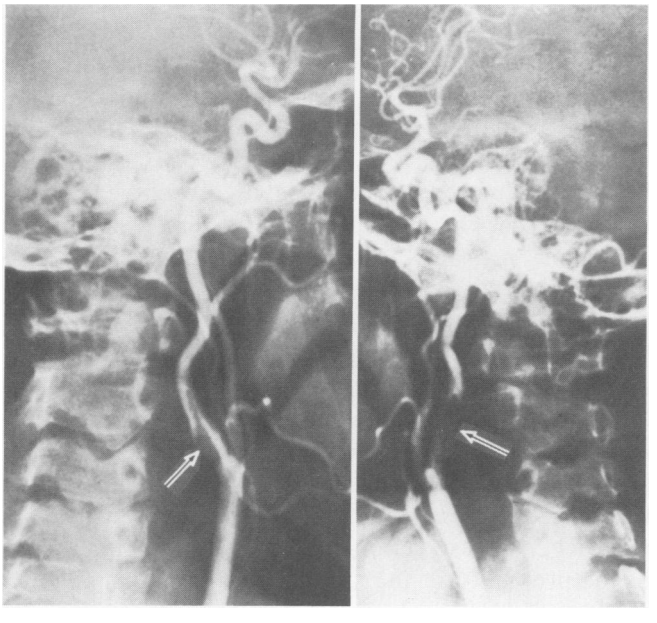

FIG. 4. (a, left) Case 2, selective left carotid arteriogram. An intimal tear (arrow) is present within the internal carotid artery. (b, right) Case 2, selective right carotid arteriogram. A radiolucent filling defect (arrow) represents thrombus at the site of an intimal tear within the internal carotid artery.

Discussion

Blunt injuries comprise only 3% of all cervical carotid trauma[16] but, if unrecognized, are associated with substantial early mortality and a high incidence of severe neurologic complications. We have collected a series of 96 patients with nonpenetrating carotid injuries, including 52 patients described by Yamada and associates in 1967 and an additional 44 patients

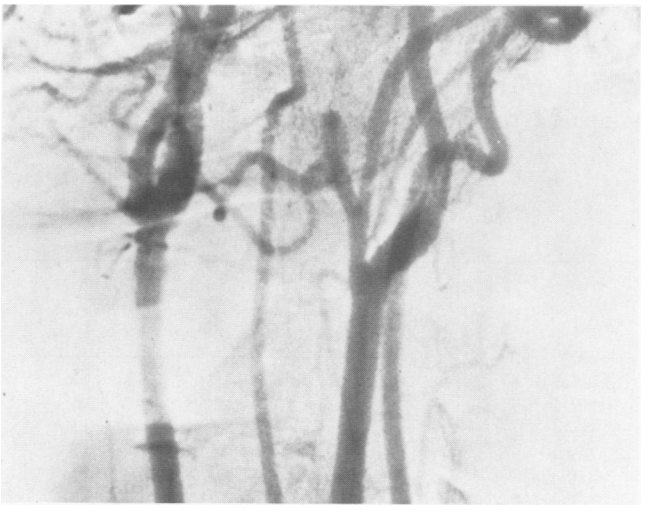

FIG. 6. Case 2, postoperative arch aortogram. The right carotid bifurcation is normal, and disruption of the intima of the left internal carotid artery has nearly resolved.

TABLE 1. *Summary of the Sources of Trauma and Associated Physical Signs in a Collected Series of 96 Reported Patients with Blunt Injuries of the Carotid Artery*[1-7,9-20,22]

	Collected Series	
	Patients	Per Cent
Sources of trauma		
vehicular accident	53	55
fall	16	17
object striking head or neck	14	15
fist fight (brawl)	10	10
boxing	2	2
diagnostic carotid compression	1	1
Total	96	100
Physical signs of trauma		
present	38	40
absent	48	50
not stated	10	10
Total	96	100

TABLE 3. *Sites and Cause of Vascular Trauma in the Collected Series of 96 Patients with Blunt Injuries of the Carotid Artery*

	Collected Series	
	Patients	Per Cent
Site of injuries		
common carotid proximal to bifurcation	7	7
at or near carotid bifurcation	47	49
internal carotid between bifurcation and base of skull	36	38
internal carotid at base of skull	6	6
Total	96	100
Cause of injuries		
no apparent arterial wall injury	14	15
disruption of intima	34	35
intramural hemorrhage	6	6
carotid fibrosis	4	4
not stated	38	40
Total	96	100

subsequently reported in the English literature. The sources of trauma in the collected series are given in Table 1. Vehicular accidents accounted for a majority of blunt carotid injuries, but only 7% of the 96 patients could recall receiving a blow directly to the neck. Because closed head injuries, such as epidural hematoma, subdural hematoma, and cerebral contusion, are far more common, an accurate diagnosis of blunt carotid trauma was considered for only 6% of patients at the time of their hospital admissions. Since 24% of patients did have concomitant closed head trauma and since at least 50% had no physical sign of direct cervical trauma (Table 1), an immediate diagnosis of blunt carotid injury has been exceedingly difficult to determine on the basis of historical or physical evidence during the initial medical examination.

Only 10% of patients with nonpenetrating carotid trauma presented with focal neurologic symptoms[2] compared to 35% of another series of patients with stab or gunshot wounds.[16] Nevertheless, 51 of the 52 patients reported by Yamada and associates eventually developed monoparesis or hemiparesis after a latent interval of several hours to a few days, a characteristic feature of blunt carotid injury (Table 2) that has been confirmed in a number of subsequent reports.[2,3,10,15,17-19,22] While patients with intracranial hemorrhage generally become obtunded by the time focal neurologic signs appear, patients with carotid injuries typically remained alert and oriented,[10] a distinction which may assume considerable diagnostic importance.

TABLE 2. *Delay in Appearance of Neurologic Symptoms Following Blunt Injuries of the Carotid Artery in the Collected Series of 96 Patients*

	Collected Series	
	Patients	Per Cent
Onset of symptoms (hours)		
0-1	6	6
1-4	16	17
4-10	16	17
10-24	22	23
Over 24	34	35
Not stated	2	2
Total	96	100

The sites and proposed cause of carotid injuries in the collected series of 96 patients are given in Table 3. While 75% of penetrating carotid injuries have been reported to occur in the common carotid artery,[16] 93% of nonpenetrating trauma involved the carotid bifurcation or the internal carotid artery. The source of trauma probably is the principal factor that determines the site of carotid injury[2,5,11]: A direct blow to the neck most frequently injures the carotid bifurcation, possibly because of the presence of friable atheromatous disease in this segment; 2) A blow to the head initiating abrupt extension and rotation of the neck to the opposite side may stretch the distal internal carotid artery across the transverse processes of the first three cervical vertebrae. Horner's syndrome may occur in association with such an injury because of the proximity of the superior cervical sympathetic chain to the internal carotid artery at this level[10,19]: 3) Intraoral trauma by foreign objects, especially prevalent in children, may injure the internal carotid artery as it courses posterior to the tonsillar fossa; 4) Basilar skull fractures may lacerate the petrosal

segment of the internal carotid artery, an injury which often is discovered only during postmortem examination.

After determining that several reported cases of blunt carotid injury were associated with fractures of the sternum, Hughes and Brownell[9] produced such fractures in cadaver specimens and found that the depressed sternal fragments tethered the innominate artery so that the distal common carotid artery and the internal carotid artery appeared to be susceptible to sudden stretch injury. Trauma in our collected series often was unimpressive, however, and severe nonpenetrating carotid injuries have been described following ordinarily innocuous events, such as a fall from a bicycle,[3] or careless manipulation of a toothbrush.[15] The diagnosis of blunt carotid injuries in children may be especially difficult because of delay in the appearance of neurologic symptoms following unwitnessed trauma that may have seemed trivial to the victim.

Focal intimal disruption with subsequent thrombosis was documented in 35% of nonpenetrating carotid injuries in the collected series (Table 3). In addition, obscure or inaccessible tears may have been responsible for some, if not most, of the 65% of cases in which the arterial wall injury was never identified.[8] Although carotid atherosclerosis would seem to be a predisposing factor to intimal disruption following blunt cervical trauma, the presence of an atheroma was confirmed in only 14% of patients. Cerebral ischemia caused by progressive carotid thrombosis at the site of intimal injury probably was responsible for the delayed appearance of neurologic symptoms in many patients, but cerebral embolization was also an important consideration.[2,5,9,12] Lower and associates[12] reviewed 20 patients who sustained thrombosis of the middle cerebral artery following blunt trauma and determined that 18 of these patients probably had cerebral emboli from cervical carotid artery injuries.

The diagnosis of carotid injury should be considered whenever an alert patient develops focal neurologic signs during an interval of a few hours to several days following blunt trauma, especially if a cervical hematoma or Horner's syndrome is present. Carotid arteriography is necessary to confirm the diagnosis and to determine whether surgical management of the injury is feasible. While 69% of the 52 patients reported by Yamada and associates underwent arteriography as an initial diagnostic procedure, immediate craniotomy was done without arteriography in another 18% because of an errant diagnosis of epidural or subdural hematoma. In 11% of this series, the diagnosis of nonpenetrating carotid injury was established only after postmortem examination. In comparison, blunt carotid injuries were identified by arteriography in 98% of cases that have been reported since 1967, a trend which probably reflects the aggressive use of arteriography to exclude the presence of intracranial hemorrhage. In many medical centers, computerized tomography currently has supplanted arteriography as the preferred diagnostic investigation when a traumatic intracranial hematoma is suspected. Considering the possibility that nonpenetrating carotid injury may have occurred, patients with distinct neurologic deficits following blunt trauma should undergo arteriography if the results of computerized tomography are normal. In addition, methods for noninvasive assessment of internal carotid blood flow, such as periorbital directional Doppler examination or oculoplethysmography, may be helpful in patients whose symptoms might be associated with progressive carotid thrombosis at the site of intimal disruption.

TABLE 4. *Results of Surgical and Nonoperative Management of Blunt Injuries of the Carotid Artery in the Collected Series of 96 Patients*

	Collected Series	
	Patients	Per Cent
Surgical management		
no neurologic deficit	14	32
minimal deficit	2	4
moderate deficit	5	11
severe deficit	18	40
dead	6	13
Total	45	100
Nonoperative management		
no neurologic deficit	2	4
minimal deficit	2	4
moderate deficit	3	6
severe deficit	22	43
dead	22	43
Total	51	100

The results of surgical and nonoperative management in the collected series of 96 patients are given in Table 4. Death or severe permanent neurologic deficits occurred in 86% of patients receiving nonoperative management and in 53% of those who underwent arterial reconstruction. Thirty-two per cent of patients were asymptomatic following surgical management, but only 4% were asymptomatic if no operation was performed. While the nonoperated group undoubtedly includes some patients who had completed strokes and were not considered to be surgical candidates, it appears that early recognition and repair of nonpenetrating carotid injuries were substantially responsible for improved results in the surgical group. Of the 52 patients collected by Yamada and asso-

ciates in 1967, all 31 patients treated nonoperatively and 71% of 21 patients treated surgically either died or had severe strokes. Of the 44 patients reported since 1967, death or permanent strokes occurred in 65% of 20 patients treated nonoperatively and in 37% of 24 patients treated surgically.

We believe that principles which have been established for management of chronic occlusive extracranial cerebrovascular disease may be applied to treatment of patients with impending carotid occlusion or cerebral embolization caused by nonpenetrating carotid trauma. Patients who have completed hemiplegic deficits should be treated without operation since they have little to gain from carotid reconstruction and could sustain hemorrhagic cerebral infarction following revascularization. However, surgical intervention should be considered for patients with episodes of transient cerebral ischemia, progressive neurologic deterioration (stroke in evolution), and even mild completed neurologic deficits associated with carotid injuries documented by arteriography. Unless the accumulation of thrombus at the site of arterial injury is corrected by operation, such patients may experience additional neurologic complications caused by continued cerebral embolization or progressive carotid occlusion. Even complete thrombosis of the internal carotid artery may be managed successfully if thrombectomy and intimal repair are accomplished within hours after the onset of neurologic symptoms.[5,11,17,22]

References

1. Calceterra TC, Holt GP. Carotid artery injuries. Laryngoscope 1972; 82:321.
2. Crissey MM, Bernstein EF. Delayed presentation of carotid intimal tear following blunt craniocervical trauma. Surgery 1974; 75:543.
3. Fieldhouse J. Posttraumatic thrombotic occlusion of the internal carotid artery. J Oral Surg 1978; 36:539.
4. Flont LM, Snyder WH, Perry MO, et al. Management of major vascular injuries in the base of the neck. Arch Surg 1973; 106:407.
5. Fleming JFR, Petrie D. Traumatic thrombosis of the internal carotid artery with delayed hemiplegia. Can J Surg 1968; 11:166.
6. Garg AG, Gordon DS, Taylor AR, et al. Internal carotid artery thrombosis secondary to closed craniocervical trauma. Br J Surg 1968; 55:4.
7. Gurjian ES, Blaise A, Renato SW, et al. Spasm of the extracranial internal carotid artery resulting from blunt trauma demonstrated by angiography. J Neurosurg 1971; 35:742.
8. Hare RR, Gaspar MR. The intimal flap. Arch Surg 1971; 102: 552.
9. Hughes JT, Brownell B. Traumatic thrombosis of the internal carotid artery in the neck. J Neurol Neurosurg Psychiatr 1968; 31:307.
10. Jernigan WR, Gardner WC. Carotid artery injuries due to closed cervical trauma. J Trauma 1971; 11:429.
11. Little JM, Vanderfield GK, May J, et al. Traumatic thrombosis of the internal carotid artery. Lancet 1969; 2:926.
12. Loar CR, Chadduck WM, Nugent GR. Traumatic occlusion of the middle cerebral artery. J Neurosurg 1973; 39:753.
13. McGough EC, Helfrich LR, Hughes RK. Traumatic intimal prolapse of the common carotid artery. Am J Surg 1972; 123:724.
14. Olafson RA, Christoferson LA. The syndrome of carotid occlusion following minor craniocerebral trauma. J Neurosurg 1970; 33:636.
15. Pitner SE. Carotid thrombosis due to intraoral trauma. N Engl J Med 1966; 274:764.
16. Rubio PA, Ruel JG, Jr, Beall AC, Jr, et al. Acute carotid artery injury: 25 years' experience. J Trauma 1974; 14:967.
17. Silvernail WU, Crowtcher DL, Byrd BR, et al. Carotid artery injury produced by blunt neck trauma. South Med J 1975; 68:310.
18. Towne JB, Neis ND, Smith JW. Thrombosis of the internal carotid artery following blunt cervical trauma. Arch Surg 1972; 104:565.
19. Wilson ES. Thrombosis of the internal carotid artery following non-penetrating craniocervical trauma. N Med Soc NJ 1977; 74:685.
20. Yamada S, Kindt GW, Youmans JR. Carotid artery occlusion due to nonpenetrating injury. J Trauma 1967; 7:333.
21. Yashon D, Johnson AB, II, Jane JA. Bilateral internal artery occlusion secondary to closed head injuries. J Neuro Neurosurg Psychiatr 1964; 27:547.
22. Zilkha A. Traumatic occlusion of the internal carotid artery. Radiology 1970; 97:543.