Carotid Artery Injuries Caused by Blunt Trauma

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Carotid artery injuries caused by blunt trauma often cause thrombosis and delayed neurologic deficits, and are associated with mortality rates of up to 40%. In this series of 17 patients with blunt trauma of the carotid, three had no symptoms, ten patients had limb paresis and four had severe neurologic deficits. The wounds were identified by arteriography; repair was attempted in 15 patients, and successful in eight. All eight patients with successful repair were improved or normal after surgery, but only two out of nine patients without repair improved, and four died. The mortality rate for the series was 23%, but only 14% in the patients who had carotid surgery. This experience suggests that repair is safe and effective in patients with carotid injuries in whom prograde flow continues and only mild neurologic deficits are present. In contrast, patients with complete occlusion, severe neurologic problems, and altered consciousness are not likely to be helped by attempts at revascularization.

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CAROTID ARTERY INJURIES caused by blunt trauma often result in thrombosis and delayed neurologic deficits.[3,12,13,17] In 85% of reported cases the extracranial internal carotid artery (ICA) or carotid bifurcation is involved, and an intimal tear or mural contusion is thought to be the underlying cause of thrombotic occlusion.[5,18] As Yamada, et al. reported, the usual evolution of symptoms is slower than might be expected after major arterial trauma, and the subsequent delay in diagnosis and treatment may unfavorably influence the prognosis.[6,18]

Jernigan and Gardner described certain clinical features that may assist in making a diagnosis of blunt carotid injury[10] (Table 1). These patients often have other injuries, and assessing neurologic deficits may be difficult in the presence of associated closed head trauma. Early separation of these patients from those with carotid artery injuries is important since restoration of carotid blood flow prior to the onset of irreversible neurologic damage is necessary if the reported mortality rate of 40% is to be reduced.[2,16]

Materials and Methods

The study group consisted of 17 patients who had sustained blunt injuries to the common or internal carotid arteries. The patients were divided into three groups according to the severity of the neurologic deficit. Group I included three patients who had no symptoms; Group II consisted of ten patients who developed limb paresis or mild hemiparesis, and Group III included four patients with severe neurologic deficits. The arterial wounds were identified by preoperative or operative arteriography and surgical repair was attempted in 15 patients (Table 2).

Results

The results are summarized in Table 2. A delay in the onset of symptoms was noted in eight patients, but the more severely injured patients generally exhibited symptoms earlier in their course. Six patients developed a neurologic deficit within one hour after injury, and four of the six had severe symptoms including alterations in consciousness. The onset of symptoms was delayed 24 hours or longer in four patients. One patient developed transient attacks of cerebral ischemia two years after the injury.

Localized intimal tears were found in four patients, diffuse intimal damage in three, and contusion in four. Two patients had spasm with occlusion, and the pathology was not identified in the remaining four patients. Simple intimal tears were repaired by direct suture. In the injuries caused by hyperextension, multiple areas of intimal and mural damage were present, and resection and vein graft interposition was the procedure of choice. Figure 1 and 2 are examples of this more extensive injury, often associated with clots. Ligation was performed in seven patients because the internal carotid artery could not be cleared of all thrombi. Carotid exploration was not undertaken in two patients because the artery was completely occluded on arteriography, and the patients were comatose.

Four of the 17 patients died from cerebral causes, for an overall mortality rate of 23%. Fifteen patients underwent carotid exploration, and the carotid artery was ligated in seven, with two deaths (14% mortality

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rate). Eight carotid repairs were performed, and there were no deaths in this group. The final results are shown in Table 3.

Discussion

In separate reports concerned with the management of penetrating cervical trauma Thal and colleagues and Bradley suggested that the eventual outcome depends largely on the extent of the initial neurologic damage.[2,16] They found that patients with mild or no neurologic deficits could be operated on with confidence and relative safety. In contrast, those patients who had severe neurologic deficits associated with alterations in consciousness, and who also had total interruption of prograde carotid flow were not improved by carotid repair. This approach was also recommended by Liekweg and Greenfield after a survey of the literature.[11] The problems posed by vascular injuries of the carotid arteries caused by blunt trauma are somewhat different, and as Towne and Yamada, et al. pointed out, the extent of injury to the artery is frequently more severe, more widespread, and in many cases the diagnosis is more difficult to establish.[17,18]

With penetrating cervical trauma the injury is obvious, but this is not the case with patients who have sustained blunt trauma; in fact, half of the patients in the collected series reported by Yamada, et al. had no external signs of cervical trauma, and only 10% developed neurologic symptoms during the first hour.[18] The reason for the delayed onset of symptoms is speculative, but as clots accumulate in the damaged vessel, thromboembolism is likely.[5,9] Although any of the carotid artery injuries may culminate in thrombotic occlusion, the diffuse intimal tears secondary to hyperextension injuries apparently are more likely to cause extensive clotting. Mural contusion can cause acute thrombosis, but may result in a progressive stenosis and produce symptoms several years later.[5,7]

Crissey and Bernstein have described four types of craniocervical injury believed to produce carotid artery

TABLE 1. *Carotid Injuries—Blunt Trauma Clinical Features*

Hematoma of lateral neck
Horner's syndrome
Transient attack of ischemia
Lucid interval
Limb paresis in an alert patient

Modified from Jernigan and Gardner, 1971.

TABLE 2. *Pertinent Data on 17 Patients with Blunt Trauma to the Carotid Artery*

Age	Cause	Onset Symptoms	Neurological Symptoms	Artery	Pathology	Operation	Condition at Follow-up
21	Crush	None	None	Rt. CC	Intimal tear	Dacron graft	Normal
58	MVA	2 yr	TIA	Lt. CC	Mural stenosis	Dacron graft	Normal
26	Blow	8 hr	Arm Paresis	Lt ICA	Multiple intimal tears	Vein graft	Normal
46	MVA	Minutes	History of unconsciousness	Lt. ICA	Multiple intimal tears	Vein graft	Normal
28	Blow	None	None	Lt. ICA	Contusion	Resection and anastomosis	Normal
56	MVA	Minutes	Hemiparesis	Rt. ICA	Intimal, tear, clot	Thrombectomy, suture	Normal
16	Fell	24 hr	Hemiparesis	Rt. ICA	Spasm, occlusion	Ligation (no back flow)	Arm paresis
42	MVA	Minutes	Hemiparesis	Lt. ICA	Intimal tear, occlusion	Thrombectomy, suture	Hand paresis
36	Blow	Minutes	Hemiplegia	Lt. ICA	Occlusion	Thrombectomy	Hemiparesis
62	Fell	20 hr	Hemiparesis	Rt. ICA	Occlusion	Ligation (distal clots)	Hemiparesis
49	Fell	48 hr	Hemiparesis	Rt. ICA	Contusion, occlusion	Ligation (no back flow)	Hemiparesis
21	Blow	24 hr	Hemiparesis	Lt. ICA	Contusion, intimal tears	Ligation (no back flow)	Hemiparesis
36	Blow	18 hr	Hemiparesis	Lt. ICA	Contusion, occlusion	Ligation (no back flow)	Hemiplegia
28	Blow	Minutes	Hemiparesis	Lt. ICA	Contusion, clot	Ligation (no back flow)	Death
19	Blow	12 hr	Hemiparesis, coma	Rt. ICA	Contusion, occlusion	Ligation (no back flow)	Death
54	MVA	Minutes	Hemiplegia, coma	Lt. ICA	Occlusion	None	Death
22	MVA	Minutes	Coma	Rt. ICA	Occlusion	None	Death

Blow = assault, beating.
MVA = motor vehicle accident.
ICA = internal carotid artery.
CC = common carotid artery.

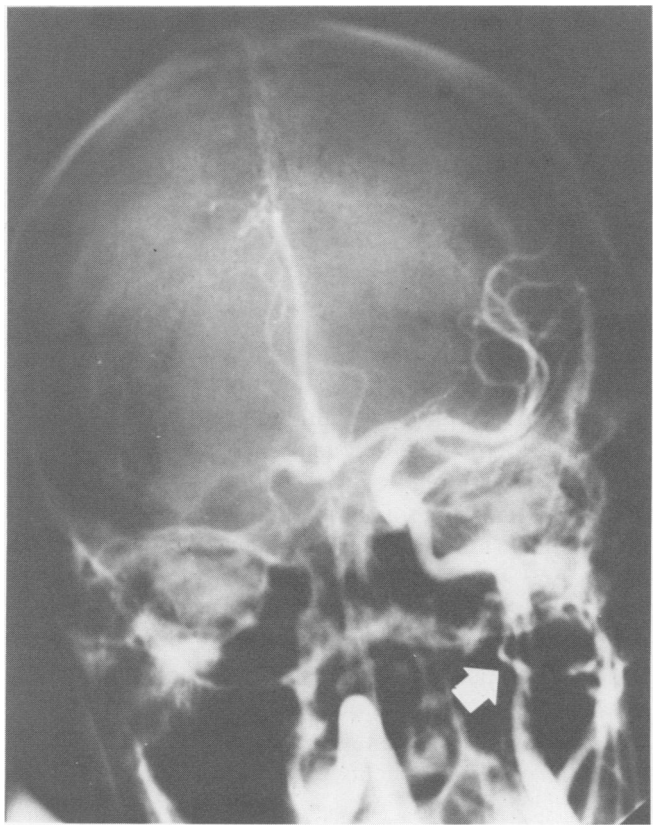

FIG. 1. Preoperative carotid arteriogram demonstrating diffuse arterial damage and intraluminal clot.

injuries.[5] 1) In approximately half of the patients a direct blow to the neck is though to be the cause of the arterial injury; 2) A blow to the side of the head or face can cause rotation and hyperextension of the head and neck, and thus stretch the carotid artery across the bodies of the first and second cervical vertebrae, or across the transverse processes of the third cervical vertebra. 3) Intraoral trauma has also been reported to be a cause of blunt carotid injury. 4) Basal or other skull fractures can damage the intrapetrous portion of the internal carotid artery.

Since the initial symptomatology may not be distinct, and because a thorough knowledge of the location and extent of the injuries is essential to successful treatment, arteriography is very helpful in managing these patients.[1,3,6,19] With modern techniques cerebral arteriography causes very few complications and in patients who are strongly suspected of having sustained blunt cervical trauma, or who have any of the symptoms described by Jernigan and Gardner (Table 1). Arteriographic examination of the extracranial vessels should be employed early in the diagnostic evaluation. Blunt cervical trauma usually causes single vascular injuries, but multiple arterial wounds have been reported and therefore all four of the extracranial vessels should be studied.[2,4] Radiographic visualization of the intracranial circulation is also recommended, especially if neurologic deficits are present or if extracranial or intracranial bypass grafts are being considered. If extensive intracerebral embolization has supervened prior to repair, revascularization is unlikely to be of substantial benefit and may be contraindicated.[17] The consensus of opinion from the reported studies of carotid trauma support surgical repair of all carotid injuries in patients who have either no neurologic deficit, or have only a mild deficit.[2,11,16] This decision is easier to reach when the arterial injury is bleeding briskly, but may be more elusive when there is complete carotid occlusion and there are no neurologic symptoms. In this situation technical problems encountered during surgery could conceivably produce brain damage, although this has been rare in the reported experience.

With the advent of better methods of diagnosis, patient selection and vascular repair, ligation of the carotid artery is rarely indicated if it is patent.[11] In a few patients the arterial damage may be so extensive that reconstruction cannot be accomplished. If the distal backflow and the distal carotid blood pressure are adequate, carotid ligation will probably be safe. This is

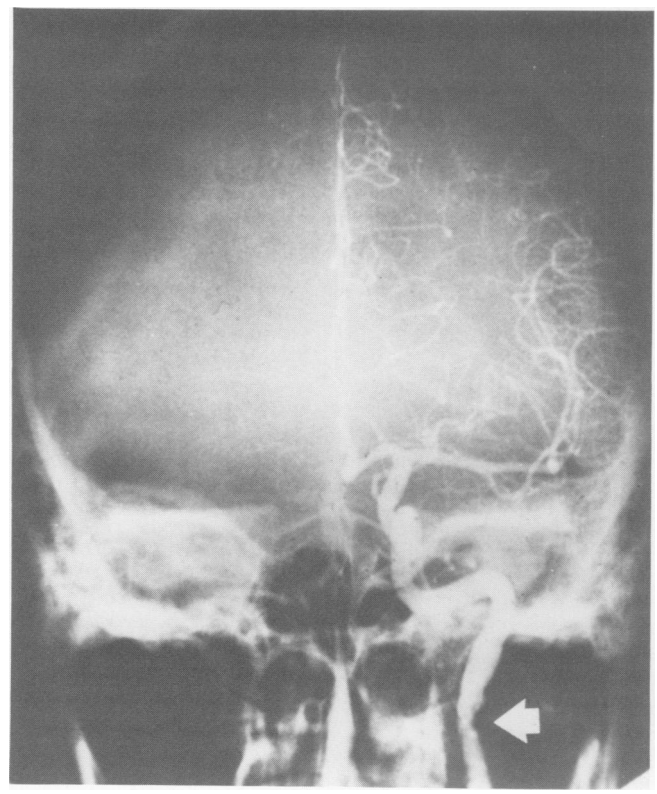

FIG. 2. Carotid arteriograms illustrating multiple intimal tears and mural contusion caused by hyperextension.

TABLE 3. *Neurologic Results*

	Normal	Improved	Same	Worse	Died
Group I (3)	3				
Group II (10)	3	2	3	1	1*
Group III (4)		1			3*

* No repair.

not invariably true, because even if initially tolerated distal internal carotid artery thrombosis may lead to a stroke.[14] It has been suggested that heparin can be used to control the rate and extent of carotid thrombosis following occlusion, or proximal ligation. A lower stroke rate has been found in a small series of patients treated with heparin while awaiting definitive surgery for occlusive disease.[8]

When inaccessible lesions of the intrapetrous portion of the internal carotid artery are present, extracranial to intracranial bypass grafts may be considered.[15] Recent clinical experiences suggest that despite the rather formidable nature of such operations, they may be useful in special circumstances, especially if there are reasons to believe that the collateral circulation to the brain is inadequate.

Thrombectomy and simple suture may be possible in selected cases, but because of the importance of securing a smooth intraluminal surface resection and anastomosis of the injured artery is favored. Hyperextension injuries are especially prone to produce diffuse intimal damage of the distal internal carotid artery, and in most of these cases resection and interposition of a saphenous vein graft or an arterial autograft is recommended. Failure to completely repair the vessel or to remove all the distal clot is very likely to culminate in additional neurologic damage, and perhaps death.[17,18] Following completion of the arterial repair a carotid arteriogram should be obtained to assess the distal intracranial circulation and to be certain that no residual clots are present in the carotid system.

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