ANNALS OF SURGERY
Vol. 223, No. 5, 513–525

Blunt Carotid Injury

Importance of Early Diagnosis and Anticoagulant Therapy

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Objective
The incidence, associated injury pattern, diagnostic factors, risk for adverse outcome, and efficacy of anticoagulant therapy in the setting of blunt carotid injury (BCI) were evaluated.

Summary Background Data
Blunt carotid injury is considered uncommon. The authors believe that it is underdiagnosed. Outcome is thought to be compromised by diagnostic delay. If delay in diagnosis is important, it is implied that therapy is effective. Although anticoagulation is the most frequently used therapy, efficacy has not been proven.

Methods
Patients with BCI were identified from the registry of a level I trauma center during an 11-year period (ending September 1995). Neurologic examinations and outcomes, brain computed tomography (CT) results, angiographic findings, risk factors, and heparin therapy were evaluated.

Results
Sixty-seven patients with 87 BCIs were treated. Thirty-four percent were diagnosed by incompatible neurologic and CT findings, 43% by new onset of neurologic deficits, and 23% by physical examination (neck injury, Horner's syndrome). There were 54 intimal dissections, 11 pseudoaneurysms, 17 thromboses, 4 carotid cavernous fistulas, and 1 transected internal carotid artery. Thirty-nine patients had follow-up angiograms. Mortality rate was 31%. Of 46 survivors, 63% had good neurologic outcomes, 17% moderate, and 20% bad. Logistic regression analysis demonstrated heparin therapy to be associated independently with survival ($p < 0.02$) and improvement in neurologic outcome ($p < 0.01$).

Conclusions
Blunt carotid injury is more common than appreciated, seen in 0.67% of patients admitted after motor vehicle accidents. Therapy with heparin is highly efficacious, significantly reducing neurologic morbidity and mortality. Heparin therapy, when instituted before onset of symptoms, ameliorates neurologic deterioration. Liberal screening, leading to earlier diagnosis, would improve outcome.

After Verneuil's first description of a case of blunt carotid injury (BCI) in 1872,[1] the entity was thought to occur rarely. The diagnosis has been made with increasing frequency in the 20th century, with the advent of the automobile and resultant high-speed collisions. Regardless of that development, BCI still is considered very uncommon. We believe that it occurs much more frequently than is generally appreciated.

Angiography is the current standard for definitive diagnosis of BCI. The diagnosis usually is suspected by neurologic findings that do not correlate with computed tomography results, or the development of a focal neurologic deficit at some point after initial evaluation. Outcome is likely compromised once a deficit develops. Predicting which patients who sustain blunt injury are at greatest risk might permit a focussed diagnostic screening program, which might identify asymptomatic lesions. Otherwise, liberal screening will be necessary to diagnose lesions before the compromise of cerebral blood flow.

Two therapeutic approaches have been applied. A surgical approach relies on either resection of the injured vessel with grafting, or extracranial–intracranial bypass. An anticoagulation approach relies on systemic heparin therapy to diminish propagation of clot and decrease embolization. Anticoagulation has received increasing support in recent years because many lesions are not surgically accessible in a safe fashion. However, because of the relative infrequency of the lesion, anticoagulation has never been proven to be an effective form of therapy.

The current study was conducted to evaluate the incidence of BCI at our institution. Lesions were stratified according to type of arterial injury. Factors impacting on diagnosis, neurologic outcome, and death were evaluated. The efficacy of anticoagulation therapy in various subgroups was analyzed, and statistical analyses were performed.

METHODS

Patients with BCI treated from December 1984 through September 1995 at a level I trauma center were identified through the trauma registry. The charts were reviewed for demographics and data related to mechanism and associated injury patterns. Brain computed tomography (CT) findings, neurologic findings at admission, and subsequent alterations in the neurologic examination were evaluated to analyze those factors that led to diagnosis of BCI. Both initial and follow-up carotid angiograms were reviewed. The neurologic outcome was evaluated according to neurologic status at the time of diagnosis, angiographic findings, and whether the patient received anticoagulation therapy with heparin at the time of diagnosis. Potential risks for mortality or neurologic deterioration were evaluated: Injury Severity Score, score on the Glasgow Coma Scale (GCS), shock, closed head injury (CHI), major neurologic deficit at time of diagnosis, and age.

Definitions of Neurologic Deficit and Neurologic Outcome

Neurologic deficit at diagnosis was defined as follows:

Minor: normal, or no *focal* neurologic deficit (neck trauma, Horner's syndrome, altered mental status with normal CT);
Major: focal neurologic deficit (paresis or plegia), GCS ≤ 8.

Neurologic status at hospital discharge and follow-up was defined as follows:

Good: normal or minimal disability;
Moderate: monoparesis or hemiparesis with independent function;
Bad: hemiplegia, GCS ≤ 8.

Radiographic Examinations

Brain scans were performed on either a helical GE Advantage (GE Medical Systems, Milwaukee, WI) or Somatom Plus (Siemens, Iselan, NJ) with and without intravenous contrast at 10-mm intervals. Angiography was performed with intra-arterial injection and digital subtraction technique. When a lesion was identified, a four-vessel study generally was performed to evaluate for synchronous lesions and cross-fill from the other cerebral vessels in event of an injury. All diagnoses were reached by consensus opinion by attending physicians of radiology, neurosurgery, and trauma surgery departments.

Treatment

Anticoagulation with heparin was the treatment for the majority of patients. The aim of heparin therapy was a partial thromboplastin time of 40 to 50 seconds. After 1 to 3 weeks of heparin, warfarin was initiated and maintained for 3 to 6 months. The target for warfarin therapy was a prothrombin time of 15 to 18 seconds or an international normalized ratio of 1.8. Anticoagulation was withheld in some patients believed to have significant associated intracranial or extracranial pathology, and in a

Presented at the 107th Annual Session of the Southern Surgical Association, December 3–6, 1995, Hot Springs, Virginia.
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Accepted for publication January 17, 1996.

Table 1. ASSOCIATED INJURIES IN 67 PATIENTS WITH BLUNT CAROTID INJURY

	Number	Percent
Closed head	32	48
Cervical spine	4	6
Chest	13	19
Abdominal	16	24
Pelvis	6	9
Lower extremity	21	31
None	2	3

few instances in which carotid thrombosis was not believed to warrant therapy. Those patients with carotid cavernous arteriovenous fistula as their form of BCI were managed with balloon occlusion.

Statistical Analysis

Univariate analysis was performed by chi square and Fisher's exact tests for discrete variables, and Student's t test for continuous variables. A logistic regression analysis was performed with multiple variables, including risk factors and heparin therapy, to determine the independent significance of factors affecting neurologic improvement, deterioration, and death. Patients treated with carotid ligation or balloon occlusion were excluded from all analyses involving heparin to eliminate bias introduced by these therapies. The level of confidence was defined as $p < 0.05$.

RESULTS

Over the nearly 11-year period of the study, there were 67 patients, receiving diagnoses of 87 BCIs, treated at the Presley Regional Trauma Center. Bilateral injuries were present in 20 patients (30%). The mechanisms of injury included: 55 motor vehicle accidents (82%), 5 motorcycle accidents (7%), 4 assaults (6%), and 3 other mechanisms (5%). During the same time period, there were 20,349 patients admitted for blunt mechanisms of injury, and 10,040 of these were injured in automobile accidents. The incidence of BCI was 1 in every 304 (0.33%) blunt injuries, and 1 in every 150 (0.67%) automobile accidents. The incidence has been consistent over time.

Forty-three (64%) patients were male and 24 (36%) were female. The average age of the patients was 34 years, with a range of 16 to 79 years. They had moderate severity of injury, with a mean Injury Severity Score of 26 (range, 15–50). The average GCS was 10. All but two of the patients had some associated injury. Table 1 illustrates the total associated injuries.

Diagnosis

The initial brain CT was normal in 37 patients. A mass lesion was present on CT examination in 18 patients. Five CT examinations demonstrated evidence of regional brain ischemia. The circumstances that prompted clinical suspicion and angiographic diagnosis for BCI were: 1) physical findings demonstrating soft-tissue injury to the anterior neck in 9 patients (14%); 2) a neurologic examination that was not compatible with brain CT in 23 patients (34%); 3) development of a neurologic deficit subsequent to hospital admission (with or without CT changes) in 29 patients (43%) and; 4) Horner's syndrome in 6 patients (9%). The average interval from injury to definitive diagnosis (angiography) was 53 hours (range, 2–672 hours). Initial neurologic findings that were incompatible with CT findings included hemiparesis (13 patients), hemiplegia (6 patients), monoparesis (3 patients), monoplegia (1 patient), aphasia/dysphasia (3 patients), and Horner's syndrome (5 patients). The neurologic deficits that developed subsequent to the initial examination included hemiparesis (10 patients), monoplegia (6 patients), depressed affect (5 patients), hemiplegia (4 patients), monoparesis (3 patients), and aphasia (1 patient).

Initial Angiographic Findings

All but one of the 67 patients underwent angiography. That patient had a transected internal carotid artery (ICA) near the foramen lacerum, discovered at the time

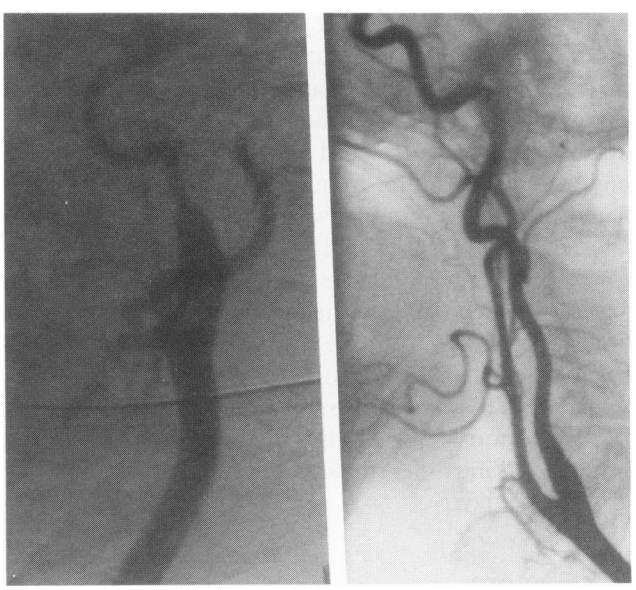

Figure 1. The carotid arteriogram on the left demonstrates internal carotid artery (ICA) dissection; that on the right demonstrates ICA pseudoaneurysm.

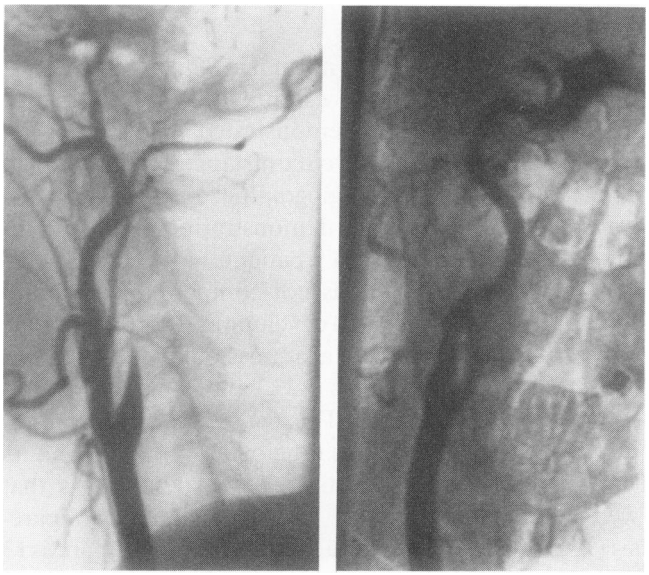

Figure 2. The carotid arteriogram on the left demonstrates internal carotid artery thrombosis; that on the right demonstrates carotid cavernous sinus fistula.

of craniotomy for evacuation of hematoma. There were 86 angiographically demonstrated lesions in the remaining 66 patients (20 bilateral). The 86 lesions included 54 dissections, 11 pseudoaneurysms (all had associated dissections), 17 thromboses, and 4 carotid cavernous sinus fistulas. Five arterial injuries occurred in the common carotid artery. Of these, three were associated with direct blows to the neck. The remaining 81 injuries were to the ICA, beginning anywhere from the bifurcation to the base of the skull. The usual pathology of these lesions is initial intimal disruption. The intimal disruption leads to dissection with or without eventual thrombosis, or to the development of pseudoaneurysm formation. Representative angiograms for the four angiographic lesions are shown in Figures 1 and 2. Dissections generally extended 2 cm or more, frequently ending near the base of the skull. The injuries were distributed fairly evenly between left and right sides.

The neurologic outcome at the time of hospital discharge is correlated with the various constellation of arterial injury types in Table 2. Of the 46 survivors, 63% had good neurologic outcome, 17% had moderate outcome, and 20% had bad outcome. Our results with carotid cavernous sinus fistula were poor; otherwise, there were no striking differences correlating outcome with arterial pathology.

Risk Factors

Shock at the time of admission (systolic blood pressure < 90 mmHg), age, major neurologic deficit at the time of diagnosis, carotid thrombosis, Injury Severity Score, and associated brain injury all were considered potential risks for mortality or neurologic impairment. Groups with and without these potential risks were compared by univariate statistical analysis. Those results are shown in Table 3. Significant increases in mortality were noted in those with shock and coma. Only coma and thrombosis demonstrated significant impact on neurologic deterioration.

Treatment

The patient with the transected ICA discovered at the time of craniotomy had the vessel ligated. He suffered brain death due to severe brain injury and ischemia 3 days later. All four cases of carotid cavernous sinus fistula (3 patients) were managed by balloon occlusion. Two of these patients died and one had aphasia and right hemiplegia. One patient had a high pseudoaneurysm that was thought to represent an impending carotid cavernous fistula. She underwent balloon occlusion of her ICA, followed by heparin therapy. Two days later, she had her heparin stopped because of hemorrhage from her tracheostomy, and she underwent sternotomy for suspected tracheoinnominate fistula. The exploration was negative, but she died of a massive stroke believed to be related to stoppage of the heparin and her transient hypotension from the tracheal hemorrhage. No autopsy was performed.

The remaining 62 patients sustained *partial* disruption of the arterial wall. Forty-seven (76%) of those received

Table 2. NEUROLOGIC STATUS AT THE TIME OF HOSPITAL DISCHARGE CORRELATED WITH ARTERIAL INJURY

	No. of Patients	Status			
		Good	Moderate	Bad	Dead
Dissection	25	14	4	1	6
Pseudoaneurysm	8	2	0	1	5
Thrombosis	11	5	2	4	0
2 × dissection	11	6	1	2	2
2 × occlusion	1	0	0	0	1
Dissection/ pseudoaneurysm	3	2	0	0	1
Dissection/occlusion	4	0	1	0	3
CCF	2	0	0	0	2
2 × CCF	1	0	0	1	0
ICA transection	1	0	0	0	1
Total	67	29	8	9	21

CCF = carotid cavernous sinus fistula; ICA = internal carotid artery.

Table 3. UNIVARIATE ANALYSIS OF RISK FACTORS FOR DEATH AND NEUROLOGIC DETERIORATION*

	Death	Deteriorate
Shock	0.03	0.14
Major deficit	0.19	0.12
GCS ≤ 8	0.04	0.03
Any CHI	0.30	0.99
Age	0.21	0.08
ISS	0.74	0.72
Thrombosis	0.53	0.03

GCS = Glasgow Coma Scale; CHI = closed head injury; ISS = injury severity score.
* Numbers are p values.

heparin at the time of diagnosis, and the others received either no therapy (8), aspirin (6), or surgery (1). The operated patient had an ICA pseudoaneurysm and dissection 2 cm distal to the bifurcation. Because this was a surgically accessible lesion, the patient was not anticoagulated. He underwent pseudoaneurysm resection with reconstitution of flow via transposition of the external to the internal carotid. He developed hemiplegia, apparently due to occlusion of the repair. He regained significant function, and currently has improving hemiparesis. Table 4 demonstrates the neurologic outcome at the time of hospital discharge, according to whether heparin was used for treatment. A significantly better outcome ($p < 0.01$) was associated with heparin as opposed to no heparin. Because this outcome could have been biased by a greater percentage of patients with minor neurologic deficits at diagnosis ($n = 19$) being treated with heparin as opposed to those with major deficits ($n = 43$), patients evaluations were done separately. Those results are shown in Figure 3. Heparin significantly improved outcomes in both groups. Deterioration was ameliorated in patients with minor deficits, and improvement was enhanced in those with major deficits. Analysis of the impact of heparin therapy on outcome associated with the previously described risk factors also was performed. Those results are illustrated in Table 5. Heparin significantly improved survival with all risk factors, including those previously noted to have a significantly negative impact on survival (shock and coma; Table 3). Heparin significantly improved neurologic function associated with all factors, except "any CHI."

The 20 patients with bilateral injuries deserve discrete analysis as a group. One of these was the patient with bilateral carotid-cavernous fistulae, leaving 19 with partial arterial disruption. The distribution of injuries is shown in Table 2. The most common indication for carotid arteriogram was a brain CT inconsistent with the physical examination (11), followed by neurologic change (4), neck injury (3), and Horner's syndrome (1). Minor deficits were present in nine at the time of diagnosis and major deficits in 10. Of these 19 patients with bilateral arterial disruption; there was one patient who had neurologic symptoms that could be attributed to both carotids. This patient had bilateral upper-extremity weakness without spinal cord injury. She had bilateral dissections, was treated with heparin for 21 days, and returned to normal. The other 18 patients had symptoms from only one of the injured carotids. Fourteen of these patients were treated with heparin. None of these patients developed symptoms from the previously asymptomatic carotid artery. Overall mortality in these 19 patients with bilateral injuries was 37%. Mortality for the patients treated with heparin was 20% compared with 100% for those who did not receive heparin ($p < 0.01$). All deaths were due to stroke.

Logistic regression analyses were performed to determine those factors that are associated independently with death or survival and improvement or deterioration in neurologic status. Table 6 contains the results from those multivariate analyses. Heparin therapy is the only independently significant factor associated with improvement in neurologic outcome. Heparin and GCS were significantly associated with survival.

There were six complications related to heparin therapy. One patient had gastrointestinal hemorrhage, which resolved with decreasing heparin. One patient required relaparotomy for what was believed to be further hemorrhage from a recently repaired liver injury. The laparotomy was negative. The patient died of a massive stroke believed to be related to discontinuation of heparin therapy in association with abdominal re-exploration. One was the aforementioned patient with balloon occlusion of her ICA and subsequent tracheal hemorrhage. Subdural hematomas developed in two patients, requiring evacuation (one in hospital receiving heparin and one at home receiving warfarin). Both underwent craniotomy

Table 4. COMPARISON OF THE NEUROLOGIC OUTCOME AT DISCHARGE OR DEATH WITH OR WITHOUT HEPARIN ANTICOAGULATION

	Neurologic Outcome		
Treatment	Good	Moderate	Bad*
Heparin	26†	7	14†
No heparin	3	1	11

* Includes deaths.
† $p < 0.01$ good *vs.* bad outcome.

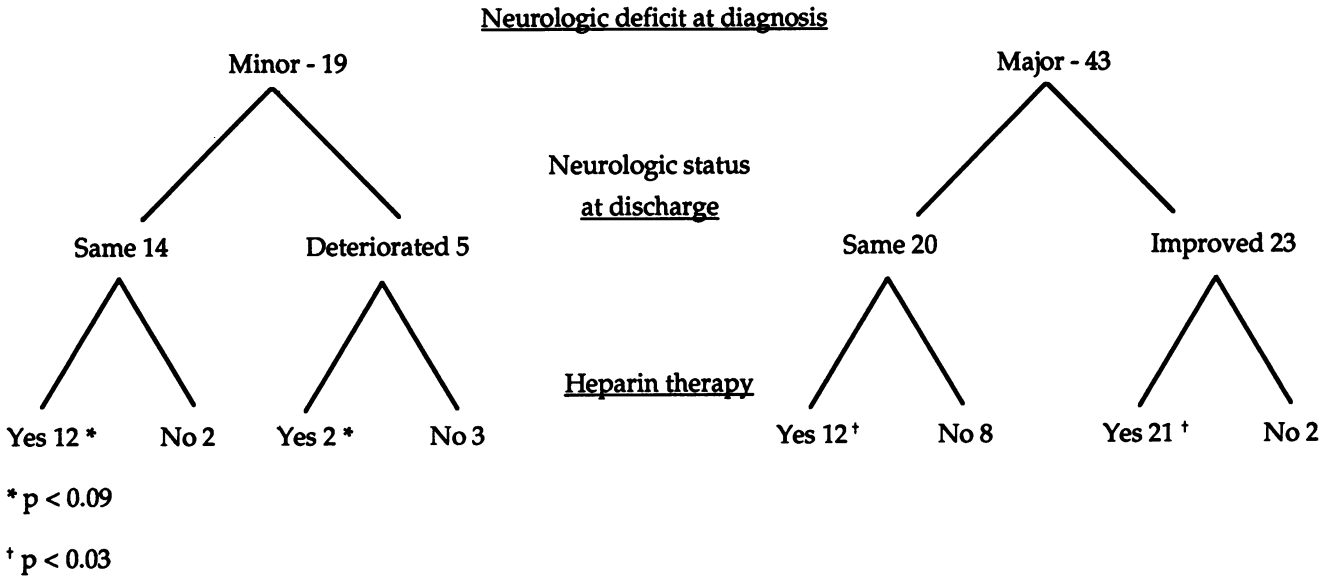

Figure 3. Correlation of neurologic status at hospital discharge or death with heparin therapy and with neurologic deficit at the time of initiation of heparin.

with subdural evacuation and had good neurologic outcomes. One patient had worsening intraventricular hemorrhage, which was treated by decreasing the heparin, and had an uneventful recovery.

Mortality

There were 21 deaths (31%). Sixteen of these deaths (76%) were directly related to strokes due to the BCI. Three patients died of CHI, and two died of sepsis and multiple-system organ failure.

Follow-Up

Thirty-nine of the 46 survivors (53 arterial injuries) had angiographic follow-up. Results of follow-up angiography are shown in Table 7. The mean follow-up interval was 172 days (range, 5– 1310 days). Of those with dissection, 62% reverted to normal and 29% developed pseudoaneurysms. None of the six patients with pseudoaneurysms reverted to normal, and one patient's artery thrombosed. One patient with thrombosis recanalized, and that individual had been treated with heparin and warfarin.

Table 5. MODIFICATION OF THE IMPACT OF RISK FACTORS ON OUTCOME BY ADDITION OF HEPARIN*

	Survival	Improvement
Shock	0.03	0.05
Major deficit	0.05	0.03
GCS ≤ 8	0.03	0.03
Any CHI	0.05	0.14
Age	0.48	0.39
ISS	0.62	0.11
Thrombosis	0.60	0.50

GCS = Glasgow Coma Scale; CHI = closed head injury; ISS = injury severity score.
* Numbers are p values.

DISCUSSION

Blunt carotid injuries generally are believed to be quite uncommon, if not rare lesions. Until the past decade, the majority of reports in the medical literature basically were case reports, with large series being in the vicinity of ten patients accumulated over one to two decades. The trend toward regionalization of trauma care in the United States and many other countries around the world has led to a number of uncommon injury types being filtered into fewer referral centers. In 1990, we noted that there were a total of 96 cases of BCI reported in the literature up to 1980, and that during the ensuing decade, there were another 75 cases reported.[2] In the subsequent 5 years, we have identified 242 additionally reported cases,[3–40] which, along with the 67 cases contained in this study, yield 480 BCI patients from all of these reports. Are there more cases being recognized, or has the funneling effect provided enough cases at individual institutions to stimulate interest in evaluation and publication? Both phenomena are likely involved. However, we believe that BCI remains an underdiagnosed problem.

Table 6. INDEPENDENT OUTCOME PREDICTORS IDENTIFIED BY LOGISTIC REGRESSION ANALYSIS

	Survival*	Odds Ratio	Improvement*	Odds Ratio
GCS	0.056	1.176 (0.996–1.389)	0.118	1.196 (0.955–10.497)
Heparin	0.015	4.936 (1.364–17.999)	0.006	13.158 (2.115–81.880)

GCS = Glasgow Coma Scale.
* Numbers are p values.
Confidence intervals in parentheses.

Davis and colleagues have reported an incidence of 0.08% among blunt trauma victims admitted to trauma centers in the San Diego area.[10] We found an incidence of 0.33%, four times greater than that report. A recent multicenter study involving 11 trauma centers reported on 49 patients (60 arterial injuries) over a 6-year period.[9] Those results, compared with the current study, would support the suggestion that many injuries are not recognized.

Issues relating to diagnosis are salient because the current data demonstrate that early diagnosis (before significant symptoms) affords improved outcome, and that outcome is improved because of the positive effect associated with therapy. The most likely explanation for our relatively large experience with BCI is the aggressive diagnostic approach of our neurosurgical colleagues. They evaluate blunt trauma admissions that have evidence of even mild head injury. The potential for carotid injury is considered in nearly all cases. Thus, mild deficits are sought, with or with significant head injury, and pursued by angiography if the CT scan does not clearly demonstrate intracranial pathology accounting for the deficit. Horner's syndrome is produced by stretching of the sympathetic plexus and interruption of the superior sympathetic ganglion, which is the basic mechanism that produces the ICA intimal injury. Prompt recognition followed by angiography will yield good outcomes, especially in the absence of associated brain injury. Blunt carotid lesions can be missed easily in the face of significant associated CHI (CHI). The data from this study would support that an aggressive diagnostic approach in head injury contributes to some degree for a higher yield. In this study, 48% of patients had CHI (mean GCS in that group = 8.6), whereas others have reported that 15% to 20% of patients with BCI have CHI.[18,30] In the study with 15% head injury, patients had a mortality of 7% for BCI, compared with our mortality of 31%.[30] The multicenter study with 49 patients stratified patients by GCS: 49% were $\geq$ 13, 14% were 8 to 12, and 37% were $\leq$ 7.0. Our study had fewer ($p < 0.06$) scores $\geq$ 13 (31%), and more ($p < 0.01$) with scores of 8 to 12 (38%). Unfortunately, although we are probably not missing many of these injuries, most are not being picked up until they have produced symptoms associated with a compromise in hemispheric blood supply. Twenty-two percent of our patients had no evident compromise of flow (9 diagnosed by soft-tissue injury and 6 by Horner's syndrome), and those patients with minimal deficit have better outcomes than those with major deficit. The reverse of that observation is that approximately 70% were diagnosed after they have had major deficits, and deficit developed in 43% after hospital admission. There are two potential approaches to enable diagnoses before progression of symptoms: 1) definition of a target population identified as being high risk for BCI and 2) broad scale diagnostic screening programs. There are major problems with either of these approaches.

The probable mechanism of injury for most *internal* carotid injuries (94% of all BCI) is rapid deceleration, with resultant hyperextension and rotation of the neck,

Table 7. INITIAL ANGIOGRAPHIC FINDINGS COMPARED WITH THOSE AT FOLLOW-UP ANGIOGRAPHY IN THE 39 PATIENTS (53 ARTERIES) WITH FOLLOW-UP STUDIES

Initial		Follow-Up			
Lesion	Number	Normal	Dissection	Pseudoaneurysm	Thrombosis
Dissection	42	26	4	12	0
Pseudoaneurysm	6	0	0	5	1
Thrombosis	5	0	1	0	4

which stretches the ICA over the upper cervical vertebrae, producing an intimal tear.[41] Some probably heal spontaneously, but others must be dissected, with or without pseudoaneurysm formation, and some thrombose. This mechanism of injury occurs in a very high percentage of automobile accidents in which that information can be obtained reliably. Furthermore, although CHI and cervical spinal injury occur in a higher percentage of patients with BCI (Table 1) than in all patients with blunt trauma, there are both a high number of patients with CHI or cervical spine injury who do not have BCI, and there are a substantial number with BCI without either of these associated injuries. Thus, targeting a select group in which to pursue arteriographic evaluation does not appear practical.

A broad-scale screening program is probably the best way to diagnose asymptomatic lesions. However, conventional arteriography is not a practical approach for mass screening. Duplex Doppler examination has been reported accurate for the diagnosis in 12 of 14 BCIs.[9] Lesions high in the ICA will be difficult to evaluate by Doppler. Although that approach is appealing, it would require a substantial commitment of resources and personnel. At a time when resource utilization is coming under increasing scrutiny, there probably are many institutions that would have difficulty making that commitment. Magnetic resonance angiography also may have a place for wide-scale screening in the future, but costs and resource availability currently are prohibitive. Until such technological avenues are open, it is doubtful that there will be substantial improvement in earlier diagnosis and subsequent outcome of BCI.

The optimal therapeutic approach has been in question. In earlier reports, the options of operative management *versus* anticoagulation were discussed. However, it became apparent that a high percentage of ICA lesions were anatomically unfavorable for repair because of either location near the base of the skull or extent of distal dissection in lesions originating nearer the bifurcation. In recent years, a trend toward management with anticoagulation has developed.[10,18] However, until now, the efficacy of anticoagulation has not been established.

The rationale for systemic anticoagulation has been to 1) minimize clot formation at the site of intimal injury; 2) decrease further propagation of clot which has formed, allowing the internal fibrinolytic systemic to dissolve the clot, and; 3) prevent embolization of clot from the sac of pseudoaneurysms.[42] The follow-up angiograms from this study demonstrated that 62% of dissections reverted to normal, demonstrating that repair occurs in association with anticoagulation. It also has been unclear whether anticoagulation is responsible for neurologic improvement, as opposed to the natural history being spontaneous improvement in some patients.

We believe that the data contained in this study unequivocally demonstrate the benefit of anticoagulation in nearly all groups with partial wall disruption. Patients with minor and major neurologic deficits at the time of diagnosis improve with heparin (Fig. 3). Heparin significantly reduced the mortality associated with the risks of shock and associated brain injury, provided significant neurologic improvement, and prevented neurologic deterioration in association with those risk factors (Tables 4 and 5). In the 15 patients with bilateral ICA injuries treated with heparin who had symptoms produced by only one side, no sequelae developed from the opposite side after initiation of anticoagulation. Finally, logistic regression analysis demonstrated heparin to be associated independently with both improved survival and neurologic outcome. The exclusion of the carotid ligation and balloon occlusion patients from all heparin-related statistical analyses (see Methods) eliminates any bias for heparin because all these patients had bad outcomes.

The distribution of pathology according to dissection with narrowing, pseudoaneurysm, and thrombosis in this series is similar to that reported by others.[9,43] There are differences in the pathologic groups, which probably has therapeutic implications. Over time, 62% of nonocclusive dissections resolve with anticoagulation. However, in our experience, there was no resolution among pseudoaneurysms on follow-up angiography. Furthermore, 29% of initial dissections developed pseudoaneurysms on follow-up angiography (Table 7, Fig. 4). The lack of pseudoaneurysm resolution also was reported by Mokri.[23] He found that of 14 pseudoaneurysms, only 3 decreased in size or resolved. It also was noted that many of these were the source of emboli, producing neurologic injury several weeks and even years after injury. Those findings would imply that patients with pseudoaneurysms should receive anticoagulants until they resolve. Perhaps more importantly, it opens the question of whether nonresolving pseudoaneurysms should undergo surgical correction. There was one report dealing with surgical repair of nine post-traumatic pseudoaneurysms in eight patients. Surgery was selected because all were producing symptoms related to embolic ischemia.[43] Excision with reversed saphenous vein grafting was performed in five, and four required extracranial–intracranial bypass. Most of the patients had residual deficits from the inciting ischemic event, but otherwise did fairly well.

What other lesions should be considered for primary surgical therapy? It has been suggested that primary surgical therapy with resection is appropriate for management of those common carotid and proximal internal carotid injuries resulting from direct trauma to the neck in the absence of fixed neurologic deficits.[44] We do not

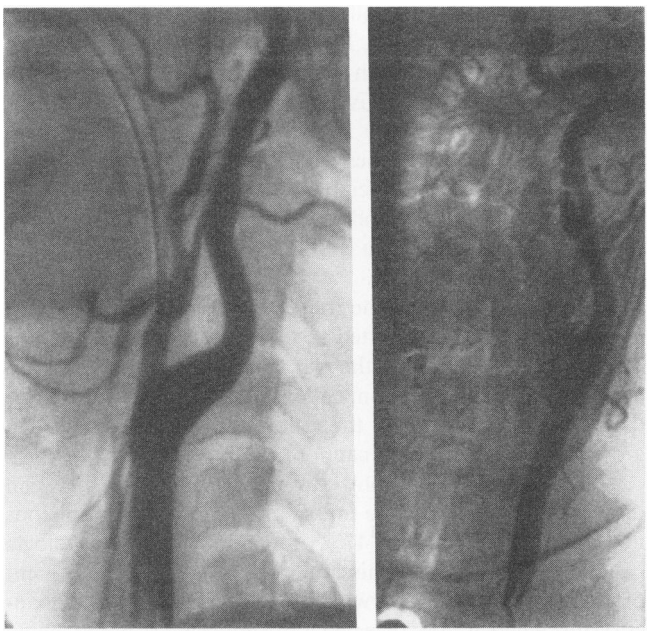

Figure 4. Initial arteriogram (left) demonstrates internal carotid artery dissection; the follow-up arteriogram (right) at 1 month demonstrates pseudoaneurysm formation.

disagree with that approach. Localized, low ICA dissections also are candidates for surgery, although these usually are not uncovered at that early stage. Advances in diagnostic techniques may discover lesions earlier, producing a higher percentage that are amenable to surgery.

Insertion of intravascular stents may have some role to play in the management of BCI. They may find a place in the treatment of pseudoaneurysms. There have been three cases reported of stent placement for carotid dissection and aneurysm (2 ICA, 1 common carotid).[45,46] Controlled trials clearly are required; platelet emboli have quite different significance in the carotid circulation compared with the systemic.

Although one of five patients with an occluded ICA recanalized with long-term anticoagulation in our series, this probably is uncommon. Another follow-up angiography study revealed no recanalization in five BCI occlusions.[23] However, 7 of 11 unilateral thromboses in this study had good (5) or moderate (2) neurologic outcomes (Table 2). Those patients had satisfactory cross-circulation through relatively intact circles of Willis. Some patients with bad outcomes probably have borderline cross-filling, which is compromised, in association with blood loss or hypotension, to such a degree that they develop cerebral ischemic insults. We would speculate that some of these patients might benefit from thrombolytic therapy in association with anticoagulation. Innovative approaches, such as stent placement and thrombolytic therapy, may improve outcomes in selected groups of patients. Controlled trials of these modalities would appear to be justified.

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Discussion

DR. DAVID V. FELICIANO (Atlanta, Georgia): Dr. Fabri, Secretary Copeland, Members, and Guests. It is a real privilege for me to discuss this review of blunt injuries of the carotid artery, the largest series ever reported in the English literature.

Dr. Fabian and his colleagues in the Presley Regional Trauma Center, Memphis, have retrospectively reviewed the records of 67 patients with 87 blunt injuries treated over a nearly 11-year period. To give you some perspective on the incredible number of patients in this series, as Tim commented, the San Diego County series had 14 dissections over 5 years. The Western Trauma Association reported 60 injuries from 11 institutions over 6 years, and the eight major trauma centers in the North Carolina Trauma Registry described only 27 injuries over a recent 6-year period.

With an incidence of one injury in every 150 victims of a motor vehicle crash admitted to their center, Dr. Fabian has clearly demonstrated that the rest of us are seriously underdiagnosing this lesion. The only alternative hypothesis would be that the carotid arteries of the residents of the Memphis area are particularly stiff. And I cannot really speak to that.

The need for angiography was evenly distributed between those with overt soft tissue injury to the neck or a suspicious neurologic examination in 47%, or those in whom a neurologic deficit developed after admission, in 43%. Seventy-five percent of the angiographic lesions were either dissections or pseudoaneurysms, whereas 20% revealed a thrombosis. As noted, heparin was found to independently and significantly improve outcome in patients with minor or major neurologic deficits.

There is a wealth of information in this study, but I would like Tim just to clarify a few points, because this study is such a large series.

Tim, you had an extraordinary incidence of these injuries, as we both commented on. And many of the angiograms that were done, 34%, were based on a neurologic examination which you say in the manuscript was not compatible with the CT of the brain. Are the rest of us who never diagnoses this injury doing incomplete neurological examinations on our patients with craniocerebral trauma? Or, as you hinted in the manuscript, are your neurosurgeons just simply much more aggressive about obtaining angiograms?

Second, the use of heparin in multiply injured trauma patients has traditionally been contraindicated, and you had six patients in the series with complications from heparin. Tim, when would you not use heparin, despite a positive carotid arteriogram? And would you then choose operation for a surgically accessible lesion?

Third, what did you do with the 29% of carotid dissections that became pseudoaneurysms on angiographic follow-up? There was no comment in the manuscript about what happened to these patients long term when they still had a surgically correctable lesion.

And, finally, in the group of 16 patients who died of strokes, were these primarily the patients who had a late diagnosis based on new neurologic findings developing after admission? In other words, were these the patients in whom heparin would be expected to have the least effect?

I really appreciate the opportunity of the floor, and I commend this manuscript to all of you. Thank you.

DR. KENNETH L. MATTOX (Houston, Texas): Classic, frequently referenced papers are but a small percentage of the many presentations each year—perhaps 0.6% of the 20,000 scientific papers each year.