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The Unrecognized Epidemic of Blunt Carotid Arterial Injuries

Early Diagnosis Improves Neurologic Outcome

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Objective
To determine the benefit of screening for blunt carotid arterial injuries (BCI) in patients who are asymptomatic.

Summary Background Data
Blunt carotid arterial injuries have the potential for devastating complications. Published studies report 23% to 28% mortality rates, with 48% to 58% of survivors having permanent severe neurologic deficits. Most patients have neurologic deficits when the injury is diagnosed. The authors hypothesized that screening patients who are asymptomatic and instituting early therapy would improve neurologic outcome.

Methods
The Trauma Registry of the author's Level I Trauma Center identified patients with BCI from 1990 through 1997. Beginning in August 1996, the authors implemented a screening for BCI. Arteriography was used for diagnosis. Patients without specific contraindications were anticoagulated. Endovascular stents were deployed in the setting of pseudoaneurysms.

Results
Thirty-seven patients with BCI were identified among 15,331 blunt-trauma victims (0.24%). During the screening period, 25 patients were diagnosed with BCI among 2902 admissions (0.86%); 13 (52%) were asymptomatic. Overall, eight patients died, and seven of the survivors had permanent severe neurologic deficits. Excluding those dying of massive brain injury and patients admitted with coma and brain injury, mortality associated with BCI was 15%, with severe neurologic morbidity in 16% of survivors. The patients who were asymptomatic at diagnosis had a better neurologic outcome than those who were symptomatic. Symptomatic patients who were anticoagulated showed a trend toward greater neurologic improvement at the time of discharge than those who were not anticoagulated.

Conclusions
Screening allows the identification of asymptomatic BCI and thereby facilitates early systemic anticoagulation, which is associated with improved neurologic outcome. The role of endovascular stents in the treatment of blunt traumatic pseudoaneurysms remains to be defined.

Blunt carotid arterial injuries (BCI) have the potential for devastating complications. In 1980, Krajewski and Hertzer[1] reviewed the literature and reported data on 96 patients, including the early series of Yamada et al.[2] Simultaneously, Perry et al.[3] described a series of 17 patients from Cornell University. These studies established the danger of BCI, with a collective 28% mortality rate, and 58% of the survivors having permanent severe neurologic deficits. More recent multicenter reviews corroborated mortality and morbidity rates of 23% and 48%, respectively, and also suggested that the incidence of BCI is approximately 1 in 1000 patients' requiring hospitalization after blunt trauma.[4–7] As a result of its relative infrequence, most institutions have limited experience with BCI; consequently, many issues remain unresolved. In 1996, Fabian et al.[8] published the largest single-institution series to date. Among their 67 patients with BCI, mortality was 31% and neurologic morbidity was 37%. This seminal report demonstrated improved neurologic outcome associated with the use of sys-

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temic anticoagulation. Unfortunately, 93% of the patients in the Memphis series already had neurologic symptoms at the time of BCI diagnosis. Recognizing that BCI often manifests after a latent period of variable duration, we hypothesized that aggressive screening would identify BCI in the asymptomatic phase or with mild neurologic changes, allowing institution of therapy and thereby decreasing neurologic morbidity. Herein we report our experience with BCI during the past 8 years, including the most recent period in which we screened patients for BCI aggressively.

METHODS

Patients

Denver Health Medical Center is a certified urban Level I Trauma Center with Pediatric Commitment and serves as the Rocky Mountain Regional Trauma Center for Colorado and adjoining regions. The number of trauma admissions during the study period (January 1990–December 1997) was stable, with a mean of 3200 patients per year, of which 86% resulted from blunt injury mechanisms. Our trauma registry records patients at the time of their hospitalization and was used to identify patients diagnosed with BCI.

Diagnosis

In all cases, the diagnosis of BCI was confirmed angiographically. Patients with hemorrhage of presumed carotid origin; cervical bruits; signs or history of external cervical trauma with altered mental status; lateralizing neurologic deficits including hemiparesis, transient ischemic attacks, amaurosis fugax, or Horner's syndrome; neurologic deficits incongruous with computed tomography (CT) scan findings; or evidence of cerebral infarction on CT underwent four-vessel cerebral angiography. In August 1996, we began to screen patients who were asymptomatic for BCI. Screening criteria included an injury mechanism compatible with severe hyperextension or flexion and rotation of the neck; significant soft-tissue injury of the anterior neck; cervical spine fracture; displaced midface fracture or mandibular fracture associated with a major injury mechanism; and basilar skull fracture involving the sphenoid, mastoid, petrous, or foramen lacerum. Follow-up angiography was performed within 7 to 10 days, when possible, to evaluate efficacy of the initial therapy.

Treatment

Before 1994, patients were managed expectantly with one exception; this patient who had an accessible lesion underwent graft replacement of the injured common carotid artery. Subsequent to 1994, patients with surgically inaccessible lesions and no contraindications underwent anticoagulation systemically.[4] A continuous infusion of heparin was administered to maintain the partial thromboplastin time at 60 to 70 seconds. Patients who had a specific contraindication to systemic anticoagulation were given antiplatelet agents, low-dose subcutaneous heparin, or were not treated. Self-expanding endovascular stents (Wallstent, Schneider, Minneapolis, MN) were deployed for persistent pseudoaneurysms.

Outcome

Neurologic function of the patients who survived was classified in the following manner: major deficit (institutionalized or requiring assistance with at least some activities of daily living), minor deficit (independent in activities of daily living but with residual sensorimotor or cognitive deficit), or normal.

Statistics

Data were managed with Microsoft Excel V. 7.0 software (Microsoft, Redmond, WA). Statistical analysis was performed on an IBM-compatible personal computer using SPSS V. 8.0 for Windows (SPSS, Chicago, IL). Continuous data are expressed as mean ± the standard error of the mean. Means of continuous data were compared using Student t test. Categorical data were compared using Fisher exact test or chi square analysis, where appropriate.

RESULTS

Incidence of BCI

During the 8-year study period we admitted 15,331 patients who experienced blunt trauma; 37 patients were diagnosed with BCI, for an overall incidence of 0.24%. Of the 37 patients, 28 (76%) were male and the mean age was 35.1 ± 2.8 years (range, 12–81 years). The average injury severity score was 28.1 ± 3.4 (range, 9–75 years). Mechanism of injury was motor vehicle crash in 15 (41%), fall and pedestrian struck in 5 (14%) each, motorcycle crash in 4 (11%), and other mechanisms (ski, horseback, bicycle and construction accidents; assault, and near-hanging) in 8 patients (22%). Associated injuries included closed head injury in 24 (65%), facial fractures in 22 (60%), thoracic injuries in 19 (51%), basilar skull fractures in 12 (32%), extremity fractures in 12 (32%), intraabdominal injuries in 11 (30%), and pelvic fractures in 6 (16%). Only two patients (5%) had cervical spine fractures, and six patients (16%) had no significant associated injuries.

A variety of injury types were observed; 22 patients had unilateral BCI (14 right, 8 left) and 15 (41%) had bilateral injuries. One was localized in the proximal internal carotid artery (ICA), and the remainder involved the ICA at or above the base of the skull. Dissection alone was present in 20 patients and pseudoaneurysm alone in 2. Six patients had combined dissection and pseudoaneurysm, four had carotid occlusion, three had carotid-cavernous sinus (C-C) fistula

Table 1. BCI SIGNS AND SYMPTOMS AT DIAGNOSIS, STRATIFIED BY YEAR

Signs/Symptoms	1990	1991	1992	1993	1994	1995	<8/96	>8/96	1997
Lateralizing deficit	1	1	1	0	0	2	3	0	5
Hemorrhage	0	1	1	1	0	1	0	0	2
Coma	0	0	0	0	0	0	0	1	4
Asymptomatic	0	0	0	0	0	0	0	4	9

BCI = blunt cartoid arterial injuries.

(one in combination with bilateral dissections), and two had carotid rupture.

Before the institution of aggressive screening in August 1996, we identified 12 BCI among 12,429 patients (0.1%). All 12 patients diagnosed with BCI had lateralizing signs or symptoms at the time of diagnosis (Table 1). During the subsequent aggressive screening period, we identified 25 patients with BCI among 2902 patients (0.86%). Only 7 (28%) of these 25 patients had lateralizing signs or symptoms. Five additional patients had severely depressed mental status with concomitant head injury, which in the past would not have prompted further diagnostic evaluation. Of the 24 patients who were symptomatic, 7 (29%) first manifested symptoms 24 or more hours postinjury, and 2 became symptomatic after 1 week. The remaining 13 patients in the series constitute the "asymptomatic" group. Of these, seven patients had subtle alterations in mental status (*e.g.*, confusion, lethargy), and six were asymptomatic neurologically. Of the seven with subtle neurologic changes, five had abnormal brain CT scans (swelling, contusion, or subdural hematoma), which easily explained their mental status changes. Table 2 displays neurologic outcome stratified by symptoms at the time of diagnosis of BCI. The presence of symptoms at the time of BCI diagnosis was associated with worse neurologic outcome ($p < 0.05$).

Management of BCI

One patient had an accessible lesion repaired via operative graft replacement. Twenty-four patients (65%) underwent systemic anticoagulation with heparin; 10 of these had additional endovascular stent placement on postinjury day 2 through 30. Eleven patients (30%) had no therapeutic intervention. Two patients had attempted embolization of a C-C fistula but exsanguinated during the procedure. Neurologic outcome stratified by the use of anticoagulation is displayed in Table 3. Four patients sustained devastating injuries and died within 24 hours; they were excluded from the analysis because treatment would not have made a difference in their outcome. There was no statistical difference in the outcome attributable to the use of systemic anticoagulation. The symptomatic patient group was analyzed separately to determine the effect of anticoagulation on the change in neurologic function from diagnosis to discharge (Table 4). Again, the patients who died within 24 hours of injury were excluded from the analysis. There was a trend toward greater improvement among those patients treated with systemic anticoagulation. While anticoagulated, 13 of 14 patients (93%) improved or were unchanged, compared with 4 of 6 (67%) of those who were not anticoagulated.

Of the 13 patients who were asymptomatic, 10 underwent anticoagulation; 9 of the 10 were discharged with normal neurologic examinations. The tenth patient was a 49-year old man wearing a helmet who was injured in a bicycle accident. On presentation, he was confused but had no lateralizing neurologic findings. Based on his injury mechanism(a direct fall on his face with cervical hyperextension) and the finding of a subarachnoid hemorrhage on CT scan, arteriography was performed to exclude BCI. He had a right ICA dissection with pseudoaneurysm and underwent anticoagulation. There was initial reluctance to deploy an en-

Table 2. NEUROLOGIC OUTCOME STRATIFIED BY SYMPTOMS

Symptoms	Dead	Major Deficit	Minor Deficit	Normal
Asymptomatic* n = 13	1	1	0	11
Symptomatic n = 24	7	6	5	6

* $p < 0.05$ compared with symptomatic group.

Table 3. NEUROLOGIC OUTCOME STRATIFIED BY TREATMENT, EXCLUDING EARLY DEATHS

Treatment	Dead	Major Deficit	Minor Deficit	Normal
Anticoagulation n = 24	1	6	4	13
No Anticoagulation n = 9	3	1	1	4

Table 4. CHANGE IN NEUROLOGIC FUNCTION FROM DIAGNOSIS TO DISCHARGE IN PATIENTS PRESENTING WITH SYMPTOMS, EXCLUDING EARLY DEATHS, STRATIFIED BY TREATMENT

Treatment	Worse	Same	Improved
Anticoagulation n = 14	1	4	9
No Anticoagulation n = 6	2	2	2

dovascular stent in the acutely injured artery, so the procedure was postponed until postinjury day 2. At that time, while the arterial catheters were being manipulated (before stent placement), he had an embolic stroke and was eventually discharged with major neurologic deficit. Excluding this patient, none of the nine patients who were asymptomatic developed an ischemic cerebral infarction while undergoing anticoagulation. Three patients who were asymptomatic did not undergo systemic anticoagulation. One underwent partial anticoagulation, was placed on aspirin, and was discharged with a normal neurologic examination; the other two had contraindications to anticoagulation and thus were not treated. Neither of the two developed symptoms of cerebral ischemia, but one died of multiple organ failure. That patient, a 77-year old pedestrian, was struck by an automobile traveling at high speed and sustained bilateral ICA dissections. Although neurologically asymptomatic, he had sustained severe multisystem trauma including a C-7 fracture, major liver injury, and unstable pelvic fracture. His BCIs were not treated because his associated injuries precluded anticoagulation. He never developed neurologic deficits but ultimately died of multiple organ failure on postinjury day 12. Table 5 summarizes of the neurologic outcome stratified by treatment for asymptomatic and symptomatic patients.

Twenty patients had follow-up angiography between 7 and 10 days postinjury. Four patients initially diagnosed with a carotid dissection and who underwent anticoagulation were found to have a new pseudoaneurysm; six patients had persistent pseudoaneurysms. One patient had a persistent dissection and one a persistent occlusion. One patient had progressive stenosis associated with dissection. Two had improved dissections, and five had resolved injuries.

Overall, 8 of the 37 patients in our series died. Four patients died within 24 hours, two died of massive head trauma and the other two secondary to profuse hemorrhage from a ruptured carotid artery. One patient with carotid occlusion and C-C fistula had a stroke with severe intracranial hypertension and was declared brain-dead on postinjury day 3. Another patient had massive head trauma and was declared brain-dead on postinjury day 11. One patient sustained carotid occlusion and had a severe stroke. After 46 days in a persistent vegetative state, care was withdrawn. The final death was a result of multiple organ failure, as described above. Excluding the three patients who died from massive brain injury, the mortality rate associated with BCI was 15%.

Complications of Treatment

Two patients had complications of angiography. One patient developed a significant groin hematoma after sheath removal, prompting a blood transfusion. A second patient, described above, had an asymptomatic BCI and had a stroke during arteriography. He ultimately was discharged but was disabled because of poor neurologic recovery.

One patient had a complication related to endovascular stenting. She had a combined right ICA dissection and pseudoaneurysm and was anticoagulated. Heparin was discontinued on postinjury day 4, however, when she had evidence of blood loss and was found to have a ruptured hepatic hematoma. The ICA injury was stented and aspirin was administered. Five days later, she had an ipsilateral transient ischemic attack but was later discharged with normal neurologic function.

Hemorrhagic complications of systemic anticoagulation were common. Of the 24 patients who were fully anticoagulated, 13 (54%) had evidence of rebleeding during anticoagulation, necessitating transfusions, cessation of heparin, or both. The source of hemorrhage was the nasopharynx in four, gastrointestinal tract in three, head in three, facial fracture site, liver and retroperitoneum in one each, and one occult source. Although the mean maximum measured partial thromboplastin time for these 13 patients exceeded the therapeutic target (87.2 seconds), that of the group who did not have bleeding complications was similarly high (97.4 seconds).

DISCUSSION

Blunt carotid arterial injuries have long been regarded as potentially devastating, yet rare, events. Early reports estab-

Table 5. NEUROLOGIC OUTCOME STRATIFIED BY SYMPTOMS AND TREATMENT

Symptoms/Treatment	Dead	Major Deficit	Minor Deficit	Normal
Asymptomatic-Anticoagulation	0	1	0	9
Asymptomatic-No Anticoagulation	1	0	0	2
Symptomatic-Anticoagulation	1	5	4	4
Symptomatic-No Anticoagulation	6	1	1	2

lished mortality rates of 28%, with 58% of survivors experiencing severe neurologic sequelae.[1–3] Subsequent multicenter reviews confirmed these disconcerting morbidity and mortality rates and reported an incidence of 0.08% to 0.17% among patients admitted for blunt trauma.[4–7] Given the relative infrequence of BCI, many issues remain unresolved. These include mechanism and pathophysiology, natural history, optimal screening and diagnostic tests, and the treatment of patients who were asymptomatic and symptomatic. In 1996, Fabian et al.[8] published the largest single-institution BCI experience and demonstrated improved neurologic outcome when patients were fully anticoagulated with heparin. Unfortunately, 93% of their patients already had neurologic symptoms at the time of diagnosis, and the ultimate outcome included a mortality rate of 31% and neurologic morbidity rate of 37%. The authors speculated that early diagnosis of BCI would be advantageous. Recognizing the potential to affect the outcome of these patients, we adopted a more aggressive approach to early diagnosis and screening for asymptomatic BCI at our institution in mid-1996.

Delayed recognition was common in previous series, with most patients diagnosed after the onset of neurologic deficits. There are several reasons for this, as suggested by Welling et al.[9] First, most of these patients have multisystem trauma, with critical injuries demanding immediate attention. In addition, head injury may preclude a meaningful neurologic examination that would demonstrate a lateralizing sign. Our experience certainly reflects this confounding scenario, because severe associated injuries, particularly closed head injuries, were common. Conversely, BCI may not be suspected in the setting of minor associated injuries. Again, our experience demonstrates the importance of a high index of suspicion because 16% of patients had no other injuries. Finally, the signs and symptoms of BCI often present in a delayed fashion. To make the early diagnosis of BCI, it is imperative that the trauma surgeon recognize the signs and symptoms of the injury. Diagnostic studies are indicated in the face of hemorrhage of presumed carotid origin; cervical bruits; signs or history of external cervical trauma with altered mental status; lateralizing neurologic deficits including hemiparesis, transient ischemic attacks, amaurosis fugax, or Horner's syndrome; neurologic deficits incongruous with CT scan findings; or evidence of cerebral infarction on CT. Early diagnosis of the symptomatic lesion can potentially minimize cerebral ischemia and ultimately optimize neurologic outcome via early therapy.

Fortunately, there is often a window of opportunity to make the diagnosis and institute therapy. A latent period has long been recognized as a unique feature of BCI. The intima is torn, providing a thrombogenic surface for platelet aggregation and a portal of exit for a dissecting column of blood. Dissection with luminal narrowing, partial thrombosis with embolization, or complete thrombotic occlusion evolve over time. Krajewski and Hertzer,[1] in their study of 96 patients, found that 58% of patients first manifested symptoms 10 or more hours after the injury; 36% became symptomatic 24 hours or more postinjury. Perry et al.[3] similarly reported 23% of patients first showing symptoms more than 24 hours after injury. Mokri et al.[10] reported that 9 of 18 BCI patients developed symptoms more than 3 days postinjury, with 1 injury manifesting 14 years later. In 1990, Fabian et al.[11] reported that 33% of their patients had a neurologic change more than 12 hours postinjury and said, "We must do better than apply the threadbare platitude of 'a high index of suspicion.'" However, in their 1996 study[8] they reported a mean time to diagnosis of 58 hours.

Recently, the Louisville group described their experience with 24 BCI.[12] All patients had neurologic symptoms at the time of diagnosis, and the authors concluded that "there was *no* reliable means to suspect this injury prior to neurologic or CT abnormalities." We respectfully challenge this premise. We believe the following injuries and injury mechanisms warrant diagnostic investigation for BCI: an injury mechanism compatible with hyperextension and rotation of the neck, which might stretch the ICA over the transverse processes of C1-C3; severe flexion and rotation of the neck, which can compress the ICA between the angle of the mandible and the vertebral column or injure it over a prominent styloid process; a direct blow to the neck; significant soft-tissue injury of the anterior neck; cervical spine fracture; significant, displaced midface fracture or mandibular fracture; and basilar skull fracture involving the sphenoid, mastoid, petrous or foramen lacerum. Although we cannot retrospectively analyze the accuracy of our screening criteria, we have been astonished by a preliminary review of the recent series: nearly 20% of patients identified by these criteria (12 of 56) have had arteriographically proven BCI. The other asymptomatic injury was detected during incidental carotid arteriography performed at the time of thoracic aortography.

The current diagnostic gold standard remains cerebral arteriography. Unfortunately, its invasive nature and potential technical and mechanical complications preclude indiscriminate application. It is usually not difficult to justify the procedure when arteriography is indicated for other reasons (*e.g.*, to rule out aortic arch injury or embolize bleeding sites), although total contrast dye load is a factor in renal toxicity. However, as we adopt alternatives to arch aortography as a screening procedure for thoracic aortic injuries, we must carefully weigh the risks and benefits of cerebral arteriography individually. Although contrast agents are becoming safer, there is still a finite risk associated with securing arterial access and imaging the cerebral vasculature. As described above, two of our patients had complications related to arteriography. For these reasons, alternative diagnostic modalities have been or are presently being investigated in this arena. Duplex scanning has been advocated to diagnose BCI,[13] but it does not detect subtle dissections. Moreover, it does not adequately image the carotid

vasculature at the skull base and above. Magnetic resonance angiography is an attractive noninvasive modality;[14] however, it is not universally available and is often not a practical option in the injured patient with multisystem injuries. Helical CT angiography offers many potential advantages over arteriography, including noninvasiveness, shorter examination times, and the ability to manipulate the image in three dimensions.[15] Furthermore, most patients with multisystem injuries undergo CT imaging for other indications. However, preliminary studies in our institution have not found CT angiography sufficiently sensitive to detect subtle intimal injuries. Given the unpredictable nature of BCI, we do not believe CT angiography is advisable until more is known about the natural history of various morphologic lesions.

The optimal management of BCI remains to be established, but there is a consensus for early active treatment. Early reports recommended surgical therapy, either ligation or repair, for accessible injuries.[1–3,7,16–18] However, as the Louisville group[17] pointed out, there are two primary patterns of BCI—a direct blow to the neck, injuring the proximal ICA—or an injury at the base of the skull or higher. The former is accessible for repair; the latter is not and is predominate in most series of BCI, including ours. In recent series, nonoperative or selective management has been widely supported.[4,5,8,9,11,12,19,20] Anticoagulation has become standard therapy, based on a multicenter review[4] and with scientific validity established by the Memphis study.[8] We cannot draw any firm conclusions regarding heparin therapy from our overall experience because we did not randomize matched groups. However, we noticed a trend toward neurologic improvement from diagnosis to discharge among patients who were symptomatic and treated with heparin. Unfortunately, 54% of our patients experienced hemorrhagic complications related to anticoagulation. This will continue to be a problem in multiply injured patients, until safer anticoagulants become available. Despite the lack of correlation between highest partial thromboplastin time and bleeding complications, we have reduced our targeted partial thromboplastin time to 40 to 50 seconds for heparin therapy in multiply injured patients, as suggested by Fabian et al.[8] In general, we did not stop the anticoagulation for hemorrhagic complications.

Pseudoaneurysms deserve special consideration. Although most dissections heal during anticoagulation therapy, a substantial number will form pseudoaneurysms. In our study, four patients with dissections (31%) developed pseudoaneurysms while on heparin therapy. This rate is similar to the Memphis experience,[8] in which 29% of patients with dissection developed pseudoaneurysms on follow-up angiography. Extensive experience with peripheral vascular trauma has shown that pseudoaneurysms, although potentially dangerous, may be managed nonoperatively because they have been witnessed to resolve in long-term follow-up studies.[21] In contrast, carotid arterial pseudoaneurysms do not typically resolve, even with heparin therapy. In addition to noting the appearance of new pseudoaneurysms with heparin therapy, Fabian et al.[8] reported no resolution among 6 primary pseudoaneurysms; Mokri[22] described resolution of just 1 of 14 pseudoaneurysms of the ICA. Moreover, pseudoaneurysms of the carotid artery have been associated with stenosis, thrombosis, embolization, and rupture causing cerebral infarction, intracranial bleeding, arteriovenous fistula, and even fatal hemothorax.[23] Thus, we submit that a more aggressive approach is justified once a carotid arterial pseudoaneurysm is recognized. Endovascular stenting provides an alternative treatment in the setting of pseudoaneurysms.[24] We previously reported our preliminary experience with stents[25,26] and have continued to gain experience with their use. This technology must be used with caution because of the potential complications as described above. An additional consideration is the need for full anticoagulation after stent placement. Experience with stenting for atherosclerotic carotid disease suggests that antiplatelet agents may be sufficient to prevent thrombosis or embolization.[27] However, our one stent-related complication occurred in a patient in whom heparin was terminated due to remote bleeding, and aspirin alone was administered. The patient sustained a transient ischemic attack 3 days later. Thus, we cannot recommend anything less than full anticoagulation to cover stents for traumatic pseudoaneurysms until more data become available.

Overall, our incidence of BCI was 0.24% for the 8-year study period. This is similar to the recent reported rates from Memphis,[8] Cincinnati,[19] and Miami.[28] From 1990 through mid-1996 we documented BCI in 0.1% of blunt-trauma admissions, and all were symptomatic at the time of diagnosis. Since August 1996 when we began aggressive screening, we have found an unprecedented 0.86% BCI among blunt trauma admissions, with most asymptomatic at diagnosis. In a sense, we have created an epidemic of BCI in our institution. In 1997 alone, we managed 20 injuries, which was more than the previous 7 years combined. Further, we have diagnosed 10 more cases in the first quarter of 1998 (not included in this study). Many of these patients are symptomatic. Their increasing presentation to our trauma center may be the result of a growing Trauma Outreach Program and the recognition of our center as having expertise with these injuries. However, the growing number of patients who are asymptomatic begs the question, are we in a position to alter the natural history of these injuries, or have we simply exposed more patients to the risks of diagnostic and therapeutic interventions? Based on data presented herein, we cannot answer this definitively. However, the patients who are identified early as asymptomatic and are anticoagulated have a superior outcome compared with those identified after the onset of symptoms (Table 2). Furthermore, no patient who is asymptomatic has experienced neurologic deterioration during heparin therapy. Ex-

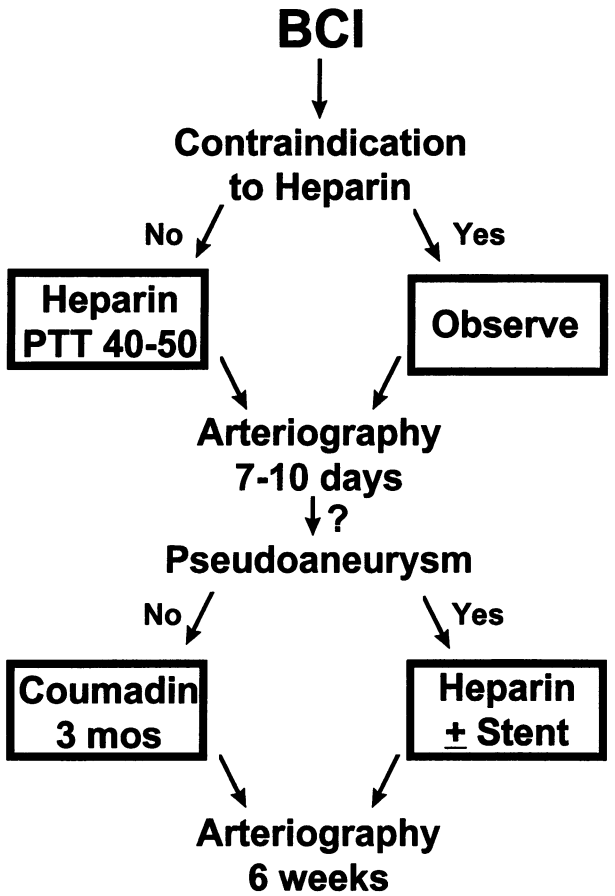

Figure 1. Protocol for the management of blunt internal carotid arterial injuries (BCI).

cluding those dying of massive brain injury and patients admitted with coma and brain injury, the mortality rate associated with BCI was 15%, with severe neurologic morbidity in 16% of survivors. This compares favorably with previous reports.[1–7]

Based on our experience and that recently reported by the Memphis and Louisville groups, we submit aggressive screening for BCI based on injury patterns is warranted. However, several important clinical issues remain unresolved. The precise injury patterns and relative cerebrovascular risks needs to be defined. Furthermore, the optimal diagnostic screening test remains to be identified, with consideration of the relative risk–benefit profile. Finally, we must determine the best means of treating BCI. Although it did not reach statistical significance, the use of heparin was associated with a trend toward improved outcome in patients who were symptomatic. In addition, no patient who was asymptomatic developed new neurologic deficits during heparin therapy. Thus, we believe early institution of heparin therapy is indicated. However, the role of endovascular stenting remains unclear. Consequently, we have initiated a prospective study (Figure 1) in which patients who have a contraindication to anticoagulation serve as the "control" group, and all others with documented BCI are systemically anticoagulated. Because the acutely injured endothelium is susceptible to angiography-related injury, follow-up arteriography is not performed until at least 7 days postinjury. If a pseudoaneurysm is identified then patients are randomized to continued anticoagulation with or without endovascular stenting. Patients will be reassessed by carotid arteriography at 6 weeks postinjury. All remaining patients will be anticoagulated for 3 months.

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Discussion

DR. MALCOLM O. PERRY (Dallas, Texas): Fewer than half the patients with carotid injuries have external evidence of such injuries. Most of these injuries are detected after the patient has a TIA or stroke. The authors advocate aggressive screening to treat these serious injuries which have a dismal natural history. They described six indications for arteriography. Surprisingly, under this protocol, as Dr. Biffl said, nearly 20% of the patients had a positive arteriogram. As Dr. Spencer indicated earlier, this is a remarkable finding in this group of patients. As I counted the numbers 13 patients were asymptomatic, 11 remained normal. As I read the manuscript which you so kindly supplied to me, of the 20 patients with neurologic deficit, you treated 14 with anticoagulants. And nine of them became normal or improved, but two of six symptomatic patients improved without anticoagulation. The mortality rate is still high, 22%. From your study it appears that anticoagulation is helpful in certain patients. I think that the eventual outcome in most of these people is still determined by the severity of the neurologic damage prior to treatment. Moreover, anticoagulation can't be used in many trauma patients, and as you described, complications were frequent and sometimes serious. I do not believe that published data support anticoagulation as the preferred method of treatment for accessible carotid lesions. The best results are obtained when carotid injuries are surgically repaired. This has been confirmed again in a recent study by Dr. Hobson and his associates in a pooled data analysis of 1316 patients. This has also been my experience. Dr. Biffl, I have three questions for you. How did the patients do after their anticoagulants were discontinued because of the complications, which were 54% in your series? Sixty-five percent of your patients had closed head injuries and a number of the group had skull fractures. How were you able to separate the underlying cause of deficits in this difficult group of patients and determine what to do? Finally, for which of these carotid injuries would you recommend surgical repair? It has been my position for a long time that most of these injuries should be repaired if they can be reached. I congratulate you and Dr. Moore and your associates. You continue to make significant contributions in the management of trauma.

DR. WALTER L. BIFFL (Denver, Colorado): Unfortunately, over 50% of the patients suffered hemorrhagic complications during anticoagulation. This presented quite a dilemma. We were hesitant to discontinue anticoagulation, for fear of stroke. Our feeling was that we could manage the bleeding either by transfusion or surgically correcting the bleeding, but that we could not treat a worsening stroke. In general we would decrease the heparin dose. In patients with head injuries, it was difficult to determine the source of the neurologic deficit. I believe one reason why these injuries may be underrecognized is because patients present with serious neurologic compromise and a concomitant head injury, and the head injury is presumed to be the cause. We feel that if a carotid injury is present we can treat it and potentially minimize the neurologic damage or prevent further neurologic damage emanating from the carotid artery. We agree that accessible lesions should be surgically repaired. Unfortunately in our experience, only 1 out of 37 was in a location that was amenable to surgical repair. All the remaining dissections and pseudoaneurysms involved the vessel at or above the base of the skull.

DR. BEN EISEMAN (Denver, Colorado): My question is whether the over-the-shoulder strap of a seat belt that runs across the base of the neck might contribute to this newly recognized "epidemic" of blunt carotid artery injuries following automobile accidents. We frequently find significant hematomas and swelling at the base of the neck following such injuries. This challenging paper will raise the obvious question of the optimum way to screen such trauma victims for unsuspected carotid artery injuries. This will have significant medical, legal, and economy importance. Might examination of eyegrounds for evidence of microthrombi be of value, particularly in the elderly? Finally, it is a source of enormous pride to be present when Dr. Biffl in the mid-point of his residency presents such an important paper in such a professional manner to this Association. I predict this will be only the first of others to follow in future years. Occasions such as this make academic surgery so wonderfully rewarding.

DR. WALTER L. BIFFL (Denver, Colorado): The question regarding seat belt injuries is an interesting one. In fact, we anticipated shoulder harnesses would prove to be a risk factor for injury. However, of 15 patients injured in motor vehicle accidents, only 2 were actually wearing their seat belt at the time of the accident. Of those two, both were drivers with a left-sided shoulder belt; one had a left-sided injury and one had a right-sided injury. The person who had the left-sided injury actually had gone off the highway down 60 feet into a tree and sustained a near-hanging by his seat belt. Thus, we have not found seat belts to be an independent risk factor. Our screening procedure of choice is arteriography. Ultrasound does not reliably detect the distal carotid lesions that we are seeing. In a previous multi-center trial in which we participated, ultrasound missed 14% of injuries. In our series, that would have been five or six patients with missed carotid injury. We have tried helical CT and thus far have found that it is not sensitive in detecting subtle intimal injuries. Unfortunately, we can't predict at this time who is going to have a stroke and who is not, based on the initial injury. Thus, we treat every injury indiscriminately. We are presenting evaluating our screening criteria for their individual predictive value. To date, we have found injuries in 20% of the patients identified for screening. An earlier study from our institution, currently in press, screened the carotids in all patients undergoing thoracic aortography to rule out blunt aortic injury.