PAPER

Anticoagulation Is the Gold Standard Therapy for Blunt Carotid Injuries to Reduce Stroke Rate

C. Clay Cothren, MD; Ernest E. Moore, MD; Walter L. Biffl, MD; David J. Ciesla, MD; Charles E. Ray, Jr, MD; Jeffrey L. Johnson, MD; John B. Moore, MD; Jon M. Burch, MD

Hypothesis: Aggressive screening, early angiographic diagnosis, and prompt anticoagulation for blunt carotid artery injuries (CAIs) improves neurologic outcome.

Design: From January 1, 1996, through December 31, 2002, there were 13 280 blunt trauma admissions to our level I center, of which 643 underwent screening angiography for blunt CAI on the basis of a protocol including injury patterns and symptoms. Patients without contraindications underwent anticoagulation immediately for documented lesions.

Setting: A state-designated, level I urban trauma center.

Patients: Of the 643 patients undergoing screening angiography, 114 (18%) had confirmed CAI.

Intervention: Early angiographic diagnosis and prompt anticoagulation.

Main Outcome Measures: Diagnosis, stroke rate, and complications stratified by method of intervention.

Results: A CAI was identified in 114 patients during the 7-year study period; the majority were men (71%), with a mean ± SD age of 34 ± 1.3 years and a mean ± SD Injury Severity Score of 29 ± 1.5. Seventy-three patients underwent anticoagulation after diagnosis (heparin in 54, low-molecular-weight heparin in 2, antiplatelet agents in 17); none had a stroke. Of the 41 patients who did not receive anticoagulation (because of a contraindication in 27, symptoms before diagnosis in 9, and carotid coil or stent in 5), 19 patients (46%) developed neurologic ischemia. Ischemic neurologic events occurred in 100% of patients who presented with symptoms before angiographic diagnosis and those receiving a carotid coil or stent without anticoagulation.

Conclusions: Our prospective evaluation of blunt CAIs suggests that early diagnosis and prompt anticoagulation reduce ischemic neurologic events and their disability. The optimal anticoagulation regimen, however, remains to be established.

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From the Departments of Surgery (Drs Cothren, E. E. Moore, Ciesla, Johnson, J. B. Moore, and Burch) and Radiology (Dr Ray), Denver Health Medical Center and the University of Colorado Health Sciences Center, Denver; and Department of Surgery, Rhode Island Hospital/Brown Medical School, Providence (Dr Biffl).

DURING THE PAST DECADE, the management of blunt cerebrovascular injuries (BCVIs) has undergone a marked evolution. Originally thought to be a rare occurrence, BCVIs are now diagnosed in approximately 1% of patients with blunt trauma.[1-3] The relative epidemic of BCVI is due to the institution of aggressive screening protocols based on injury mechanism and associated injuries. The goal of such screening protocols is to identify these injuries in asymptomatic patients and implement preventive treatment before devastating neurologic sequelae take place. Initially, BCVIs were thought to have unavoidable serious neurologic outcomes, but early reports suggested that anticoagulation might decrease these events.[4-6] If untreated, carotid artery injuries (CAIs) are associated with a stroke rate up to 50%, depending on injury grade, with increasing stroke rates correlating with increasing grades of injury.[3] More current studies have suggested that early anticoagulation in patients with BCVIs reduces stroke rates and resultant neurologic morbidity[1,3,7,8]; however, no randomized studies have been completed.

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A recent review by Miller et al[2] of the Memphis group suggests that early diagnosis and anticoagulation in CAI showed no improvement in stroke rate compared with their earlier study[2]. Although heparin has been proposed as the gold standard treatment in these patients, with this recent report, we

questioned this supposition. The expense and risk associated with screening for CAIs is not justified if the outcome is not altered with treatment. Therefore, we analyzed our recent experience with anticoagulation, as we hypothesize that early diagnosis and prompt anticoagulation are effective.

METHODS

Denver Health Medical Center, Denver, Colo, is a state-certified and American College of Surgeons–verified level I regional trauma center and an integral teaching facility of the University of Colorado Health Sciences Center. Beginning in 1996, we instituted a comprehensive screening protocol for blunt CAIs based on injury mechanism, injury patterns, or symptoms (**Table 1**). Presenting neurologic symptoms include transient ischemic attacks, reversible ischemic neurologic deficits, and ischemic infarction or stroke; all of these are categorized as ischemic neurologic events (INEs).

We have prospectively observed these patients since initiation of the screening protocol in 1996. Four-vessel cerebrovascular angiography remains the standard screening test for patients at risk for BCVI and is performed as soon as possible after evaluation of life-threatening injuries. Angiography is performed via the femoral approach by an attending interventional radiologist. Digital subtraction techniques are used to study the aortic arch and carotid and vertebral vessels. Patients' injuries are classified according to their angiographic appearance by means of our grading scale (**Table 2**).[9] Follow-up angiography is performed 7 to 10 days after initial diagnosis to evaluate the status of the injury and need for further therapy.

As the vast majority of injuries are inaccessible by surgery, anticoagulation has been used. Once a CAI is identified, anticoagulation with systemic heparin is initiated with a continuous infusion of heparin sodium at 15 U/kg per hour, without a loading dose; heparin drips are titrated to achieve a partial thromboplastin time between 40 and 50 seconds. If the patient has a contraindication to heparin, antiplatelet agents (aspirin, 325 mg/d, and clopidogrel bisulfate, 75 mg/d) or subcutaneous heparin (5000 U of unfractionated heparin sodium twice daily or 5000 U of dalteparin sodium daily) are administered, in consultation with our neurosurgical colleagues.

Patients diagnosed as having blunt CAIs from January 1, 1996, through December 31, 2002, were identified from our trauma registry database. One patient undergoing operative repair of a common carotid injury was excluded from the analysis. The Colorado Multi-institutional Review Board approved this study.

Table 1. Potential Screening Criteria for BCVI

Signs/Symptoms of BCVI	Risk Factors for BCVI
Arterial hemorrhage	High-energy transfer mechanism with:
Cervical bruit	Le Fort II or III fracture
Expanding cervical hematoma	Basilar skull fracture with carotid canal involvement
Focal neurologic deficit	Diffuse axonal injury with GCS score <6
Neurologic examination results incongruous with findings on head CT scan	Cervical spine fracture
Stroke on secondary CT scan	Near-hanging with anoxic brain injury

Abbreviations: BCVI, blunt cerebrovascular injury; CT, computed tomographic; GCS, Glasgow Coma Scale.

Table 2. Grading Scale for Carotid Artery Injuries

Grade	Description
I	Irregularity of vessel wall or dissection/intramural hematoma with <25% luminal stenosis
II	Intraluminal thrombus or raised intimal flap visualized, or dissection/intramural hematoma with ≥25% luminal narrowing
III	Pseudoaneurysm
IV	Vessel occlusion
V	Vessel transection

RESULTS

STUDY POPULATION

During the study period, 13 280 patients sustaining blunt trauma were admitted to our level I trauma center. According to our standard BCVI screening protocol, 643 patients underwent diagnostic angiography and 114 patients (18%) were identified as having CAIs, for an overall incidence of 0.86% of blunt trauma admissions. Eighty-one patients (71%) were men, and the mean±SD age was 34±1.3 years. The mechanism of injury was motor vehicle related in 90 patients (79%), 13 patients (11%) sustained a fall, and the remaining 10% of injuries resulted from other mechanisms (snowboarding, horseback riding, bicycle collisions, and assault).

Most patients were multiply injured. Associated injuries included intracranial or facial trauma in 82 patients (72%), thoracic injury in 56 patients (49%), intra-abdominal injury in 40 patients (35%), extremity injuries in 45 patients (39%), pelvic fractures in 20 patients (18%), and spine fractures or spinal cord injuries in 41 patients (36%). The mean±SD Injury Severity Score was 29±1.5, and Glasgow Coma Scale score, 9±0.5.

BLUNT CAIs

Seventy-eight patients had unilateral carotid injuries, 50 patients with right-sided injuries and 28 patients with left-sided injuries, while 36 patients had bilateral injuries, for a total of 150 CAIs. Twenty-five patients (22%) also had associated vertebral artery injuries. Eighty-nine injuries were grade I, 30 were grade II, 25 were grade III, 3 were grade IV, and 3 were grade V (**Table 3**).

STROKE RATE STRATIFIED BY TREATMENT

Seventy-three asymptomatic patients with CAIs received 1 of 3 types of anticoagulation: systemic heparin, subcutaneous low-molecular-weight heparin, or antiplatelet agents (aspirin and clopidogrel). Systemic heparin was administered in 54 patients (74%), low-molecular-weight heparin in 2 patients (3%), and antiplatelet agents in 17 patients (23%) (Table 3). None of the 73 asymptomatic patients with CAIs who received anticoagulation developed an INE.

Forty-one patients did not receive anticoagulation; of these, 19 patients (46%) had an INE (Table 3). Of the 27 asymptomatically screened patients who did not re-

Table 3. Stratification of Patients by Treatment Modality and CAI Grade

Intervention (Stroke Rate)	Patient's Grade of CAI, No. of CAIs				
	I	II	III	IV	V
Anticoagulation in asymptomatic patients (n = 73)					
Heparin (0/54)	38	17	11	1	0
LMWH (0/2)	2	0	0	0	0
Antiplatelet agent (0/17)	16	1	3	0	0
No anticoagulation (n = 41)					
Contraindication to anticoagulation (5/27)	31	7	3	2	0
Symptoms before angiography (9/9)	2	5	5	0	0
Carotid coil/stent (5/5)	0	0	3	0	3

Abbreviations: CAI, carotid artery injury; LMWH, low-molecular-weight heparin.

Table 4. Patient Treatment and Outcomes Related to CAI Grade

Presentation (INE Rate)	CAI Grade	Time to Diagnosis, h	Treatment After Diagnosis	Outcome
Contraindication to anticoagulation (5/27)	I	72	SQ heparin sodium	Improved
	I	192	SQ heparin	Improved
	Bilateral I	24	Aspirin, clopidogrel bisulfate	Improved
	Bilateral II	72	SQ heparin	Improved
	IV	24	Aspirin	Cerebral edema, death
Symptoms before angiography (9/9)	I	240	Aspirin	Improved
	II	14	Heparin	Improved
	II	48	Heparin	Improved
	II	24	Heparin	Improved
	III	24	Heparin	Resolved
	Bilateral III	36	Heparin	Improved
	I and III	18	None	Pulmonary failure, death
	II	10	None	Cerebral edema, death
	II and III	36	None	Cerebral edema, death
Carotid coil/stent (5/5)	Bilateral III	48	Stent, heparin	Improved
	Grade III	144	Stent	Care withdrawn, death
	Grade V	Admission	Coil	Exsanguinated, death
	Grade V	Admission	Coil	Cerebral edema, death
	Grade V	Admission	Coil	Cerebral edema, death

Abbreviations: CAI, carotid artery injury; INE, ischemic neurologic event; SQ, subcutaneous.

ceive anticoagulation after diagnosis, 5 patients (19%) had an INE. The contraindication for anticoagulation in these 5 patients was intracranial hemorrhage in 4 (3 subdural hematomas and 1 subarachnoid hemorrhage) and a complex pelvic fracture requiring embolization and operative pelvic packing in 1. Nine patients presented with neurologic symptoms, consisting of hemiparesis (6 patients), aphasia (2 patients), or mental status changes (1 patient), before diagnostic angiography. Five patients underwent interventions during angiography; coil placement was performed in 3 patients (2 for a carotid-cavernous fistula, 1 for active bleeding), and 2 patients with pseudoaneurysms (grade III carotid injuries) underwent early stent placement (postinjury days 2 and 6). All 5 of these patients had an INE. In the 9 patients who presented with neurologic findings before angiography, the average time of onset to symptoms after the original insult was 50 hours (range, 10-240 hours). In the 5 asymptomatic patients not receiving anticoagulation who had a stroke, the average time to symptoms was 77 hours (range, 24-192 hours). The individual CAI grade, time to diagnosis, and therapy for patients who developed an INE are summarized in **Table 4**.

COMPLICATIONS AND OUTCOME

In patients who had an INE, either before or after diagnosis of CAI by angiography, the neurologic outcome varied (Table 4). In the 5 patients who were screened while asymptomatic but had a contraindication to anticoagulation, 4 patients improved neurologically after INE; of these patients, 3 were treated with subcutaneous heparin and 1 with aspirin and clopidogrel. One patient with a grade IV CAI had a hemispheric stroke with concurrent postinjury cerebral edema and subsequently died, presumably of herniation. Of the 9 patients with neurologic symptoms before angiography, 3 patients died; 1 died of overwhelming pulmonary failure, while the 2 patients with hemispheric strokes experienced cerebral edema, uncontrolled elevations of intracranial pressure, and subsequent herniation. Of the 6 remaining patients, all showed neurologic improvement (5 treated with heparin, 1 with aspirin), with 1 showing eventual complete recovery. Five patients were treated with either carotid embolization (3 patients) or early stenting (2 patients). Of those undergoing embolization, 1 patient exsanguinated during interventional radiology, and 2 pa-

tients subsequently died of cerebral edema and herniation. One patient who received carotid stents was treated with heparin; 1 patient's condition improved, and the other patient's family withdrew care because of the severity of his associated head injury. Stroke-related mortality in this study was 32% (6 of 19 patients), with an overall mortality of 14% (16 of 114 patients).

Complications of angiography included hematomas of the catheter entry site in 2 patients, neither requiring operative intervention, and 1 stroke after screening angiography. This patient underwent screening because of his mechanism of injury, with associated basilar skull fracture and complex facial fractures. He had a right middle cerebral artery stroke after angiography, with resultant left hemiplegia. At discharge to an acute inpatient rehabilitation program, he was beginning to show improvement in motor function.

COMMENT

Cerebral ischemia after blunt CAI occurs in up to 50% of untreated patients, with significant attendant neurologic morbidity and mortality.[3,10-12] A recent study by Miller et al[2] reported a stroke rate of 33% despite aggressive screening, early identification, and anticoagulation for CAIs. Although heparin has been recommended as the gold standard therapy,[2,5-7] after the Miller et al report we retested our own hypothesis that early anticoagulation reduces the stroke rate after diagnosis of CAI. It is imperative to document improved outcomes with treatment; otherwise the expense and risk associated with screening for CAI is not justified.

Initially, recognition of blunt CAI followed neurologic symptoms of cerebral ischemia.[5-8,13] In early studies of BCVI, more than 90% of patients were symptomatic at the time of angiography.[7,8] Identification of CAI in symptomatic patients resulted in the recognition of the clinically critical and interventionally advantageous "latent period" associated with these injuries.[2,6] This latent period is confirmed in the current study's small group of patients who presented with or developed neurologic findings; the average time to onset of symptoms after the original insult was 50 hours in those without diagnosed injury and 77 hours in those with a diagnosed injury and contraindications to anticoagulation. Although surgeons were initially hesitant about anticoagulation in patients with CAI caused by associated injuries,[13] anticoagulation was shown to improve neurologic outcomes in symptomatic patients with CAI.[7] Therefore, diagnosis of these injuries before symptom onset was sought with the idea that anticoagulation in asymptomatic patients might prevent neurologic sequelae.

Screening of asymptomatic patients was instituted through the use of mechanism of injury, constellation of injury patterns, and symptoms. Although the ideal screening protocol remains debated, several groups have identified risk factors and injury patterns associated with CAI.[1,2,5,7,8,14] Our current indications for screening angiography in asymptomatic patients include high-energy transfer mechanism with Le Fort II or III fracture, basilar skull fracture with carotid canal involvement, diffuse axonal injury with Glasgow Coma Scale score less than 6, C-spine fractures, or near-hanging with anoxic brain injury.

Angiography remains the gold standard for screening patients at risk. Others have advocated duplex scanning,[5,12,15] computed tomographic (CT) angiography,[16,17] and magnetic resonance angiography[18,19] for diagnosis of CAI. However, studies have shown that each of these imaging options remains suboptimal for evaluation.[2,3,6,20,21] Although angiography is invasive and utilizes hospital resources, the possibility of missed injuries is real. Complications related to angiography include puncture-site hematoma, bleeding complications, and angioembolic phenomena. In this study, puncture-site hematoma occurred in 2 patients, or 0.3% of all angiograms, and 1 patient (0.1%) had a stroke after screening angiography.

Subsequent to the institution of aggressive screening protocols, CAIs have been diagnosed in an alarming number of patients with blunt trauma. This injury occurred in 0.86% of trauma admissions during the 7-year study period, consistent with our previous study numbers, after initiation of the screening protocol, which ranged from 0.86% to 1.1% of trauma admissions.[3,8,9] During the past 7 years, 643 angiograms have been performed according to our protocol; 114 patients with 150 CAIs were identified, producing yield rates of 18% (for patients) and 23% (for injuries). As noted in previous studies,[2,3] the majority of patients are multiply injured, emphasizing the need for standard screening protocols and a high index of suspicion; our results echo these previous findings, as our patients had a mean Injury Severity Score of 29 and most sustained intracranial or facial trauma and thoracic or abdominal trauma.

The first large single-institution study by the Memphis group reported neurologic morbidity of 37% and mortality of 31%,[7] concordant with previous smaller studies' estimates of morbidity rates up to 48% and mortality of 23%.[5,6,12] Early diagnosis and anticoagulation in asymptomatic patients with CAIs appeared to reduce stroke rate,[2,3,5,6] although no randomized trials have been completed. This study confirms that early diagnosis is critical and that prompt anticoagulation remains the cornerstone for prevention of impending neurologic disasters.

Seventy-three patients diagnosed as having CAIs received anticoagulation with heparin, low-molecular-weight heparin, or antiplatelet agents; remarkably, none of these patients experienced an INE. All grades of injury were represented in this group, but the majority of grade II to IV injuries were treated with heparin, our gold-standard therapy based on previous study findings. Although heparin appears to have improved outcomes in patients with neurologic symptoms,[1,3] the ideal choice of anticoagulation in asymptomatic patients remains under debate. As noted by the Memphis group, asymptomatic patients treated with either heparin or aspirin have markedly lower stroke rates than those untreated.[1] On the basis of our previous work that shows no significant difference between antiplatelet and heparin treatment of asymptomatic patients with CAIs,[3] we are currently enrolling patients in a randomized prospective study to compare heparin with aspirin-clopidogrel in the acute treatment of asymptomatic grade I to III BCVIs. An additional

area of study is the long-term anticoagulation choice, warfarin sodium vs aspirin-clopidogrel, for the proposed 6 months of treatment.

Forty-one patients did not receive anticoagulation, 19 (46%) of whom suffered neurologic sequelae. Of note, 3 of these patients had grade I injuries, reminding us that what may appear to be an innocuous injury may in fact have profound consequences if ignored. This also supports the theory that the predominant mechanism of stroke after blunt injury is embolic rather than occlusive. Hence, any breach of the endothelium is a potential source of emboli and should be appropriately treated. Five (19%) of the 27 patients with contraindications to anticoagulation had INEs, while 9 patients presented with neurologic findings before diagnostic angiography. These findings raise 2 fundamental questions. First, in multiply injured patients with relative contraindications to anticoagulation, what is the optimal risk-benefit intervention? Second, in patients manifesting neurologic symptoms before angiography, what steps can be taken to ensure timely diagnosis of BCVI during the asymptomatic latent period?

The majority of patients who have BCVIs are multiply injured, often with solid-organ injuries, complex pelvic fractures, or intracranial hemorrhage. In this group of patients, anticoagulation for a CAI is potentially problematic. This topic has not been addressed definitively to date. With only 2 patients in this series experiencing complications from visceral bleeding, and neither requiring intervention, perhaps a more aggressive anticoagulation protocol should be used. In patients with stable CT scan findings of intracranial hemorrhage, we initiate anticoagulation after concordance with our neurosurgical colleagues.

Clearly, diagnosis and treatment of CAIs during the latent period is critical to prevent neurologic devastation. Therefore, despite an aggressive screening protocol, why did 9 of our patients present with neurologic findings before diagnosis? Two patients were transferred to our facility specifically for angiography after development of neurologic symptoms; in these cases, education on screening criteria for BCVIs at referring hospitals is the solution. Six patients had injuries that fit the screening criteria; the time to angiography becomes questionable in this group. One patient did not undergo angiography until 10 days after injury because the results of his neurologic examination were believed to be consistent with intracranial pathologic findings diagnosed by CT scan. The other 5 patients developed symptoms within 36 hours (at 10, 14, 18, 24, and 36 hours) before angiography. Because of intense demand for interventional radiology in our level I trauma center, we agree to defer screening angiography to daytime hours. These results suggest that we need prompt angiography in all patients. The final patient is most frustrating. This patient was involved in a motor vehicle collision and sustained only a head laceration; he was discharged from the observation unit after 12 hours without incident. At home he fell asleep and, on awakening, was noted to have left hemiparesis. Angiography was performed emergently and disclosed a grade II injury, and fortunately his neurologic findings improved after heparin treatment. This is the only patient in our series who had a stroke that would not have been identified by screening criteria and timely angiography.

Although carotid stenting has been suggested and used for grade II CAIs with marked flow compromise, persistent grade III CAIs, and grade V CAIs, its precise role remains a topic for future exploration. Because of the increased risk of emboli during angiography and stenting, we advocate a 7- to 10-day delay before stent placement after initial diagnosis of CAI. Since the institution of this policy, there have been no subsequent events with carotid stent placement.

In sum, our ongoing evaluation of blunt CAIs, and that of the Memphis group, suggests that early diagnosis and prompt anticoagulation reduce stroke and its disability. We submit that these data support continued aggressive screening, although a less labor-intensive diagnostic tool is needed. Although no asymptomatic patient treated with anticoagulation had a stroke, the optimal anticoagulation regimen is not yet established. In patients with relative contraindications to anticoagulation—intracranial hemorrhage, high-grade solid-organ injury, and complex pelvic fractures—more precise guidelines for timing and type of anticoagulation must be developed. The role of carotid stents in the treatment of CAI remains unanswered. Education of trauma surgeons in the screening criteria for BCVI, need for diagnostic diligence, and prompt anticoagulation in patients at risk will ultimately reduce devastating neurologic sequelae.

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Corresponding author: C. Clay Cothren, MD, Department of Surgery, Denver Health Medical Center, 777 Bannock St, Mail Code 0206, Denver, CO 80204 (e-mail: clay.cothren@dhha.org).

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DISCUSSION

David B. Hoyt, MD, San Diego, Calif: I congratulate Dr Cothren for an excellent presentation, Dr Moore and the Denver group for bringing this important subject forward once again.

This paper is important for at least 3 reasons: First, it is yet another wake-up call that we are underdiagnosing blunt carotid injuries. Most of the trauma centers in the United States still have the alarm button on snooze regarding this issue.

Second, anticoagulation seems to prevent stroke, whereas stroke, once it occurs, is associated with a mortality of about one third of all patients. This has been found in virtually every study that has ever looked at blunt carotid injuries. Again, aggressively searching for these injuries is essential. Amazingly, in their series, one third of the patients had bilateral carotid dissections. You can argue whether starting heparin or some other anticoagulant is correct, but it would appear from this and the data from Memphis that heparin saves lives.

Third, the newest kid on the block, if you will, angiographic stenting or coiling, though theoretically helpful, particularly in the patient in whom there is a contraindication to anticoagulation, is almost invariably in their experience associated with some complication, including stroke. We need to step back and reevaluate the role of stenting and ask, is there is any role in this group of patients?

I have 3 questions for the authors. First, given the slow adoption of these aggressive screening procedures in other trauma centers and the improved quality of CT reconstruction, is CT angiography a reasonable alternative even though in past studies it has been shown to be not as sensitive? Should it be reevaluated? Perhaps duplex and CT angiography will be an alternative to conventional angiography. Second, which presenting signs and symptoms are the highest yield for actual injuries, and have you done any subsequent analysis as your series has enlarged to really refine screening criteria? What is the injury pattern that predicts this kind of patient? Is there an injury pattern that predicts bilateral injuries? And then, finally, what was the basis of choosing heparin vs low-molecular-weight heparin vs antiplatelet therapy?

You mentioned in the manuscript that you are embarking on a prospective trial. What should we do in the meantime until there are new data? Should there be a moratorium on stenting these patients until there are better data? Again, it really is time to turn the alarm off and get out of bed with regard to this injury. We need to have a better screening technique, but we now have at least 2 groups in this country that have shown us that there is about a 10-fold incidence of this injury if you look for it carefully.

Dr Moore: It's a pleasure to have Dr Hoyt to discuss our paper. Dave's report in 1990 from San Diego is one of the first to suggest that heparin would improve outcome in these patients. The first question related to emerging improvements in CT angiography as well as magnetic resonance angiography and duplex scanning. To date, the comparative studies with these modalities have not proven them to be adequately sensitive. Specifically, CT angiography with reconstruction and magnetic resonance angiography with gadolinium have yielded sensitivities of only 50% in both the carotid and vertebral beds. They also have about a 5% false-positive rate. Distressing in our experience, 8% of grade I carotid injuries and 19% of grade I vertebral injuries led to a stroke without anticoagulation. These minor injuries are going to be a challenge if we omit angiography. However, in institutions in which angiography is not available, certainly these diagnostic modalities are better than ignoring the possibility.

With regard to presenting signs and symptoms of BCVIs, of course, the most predictable is neurologic symptoms in the middle cerebral artery distribution. High-risk injury patterns include basilar skull fractures through the foramen lacerum and Le Fort II or III fractures. With 2 or more of these risk factors, the carotid injury rate exceeds 50%. Moreover, there is a risk of associated vertebral artery injury that compounds the situation. For example, cervical fractures involving the foramen transversarium have an incidence of vertebral injury exceeding 65%. In reference to bilateral BCVIs, as you might suspect, frontal impact with Le Fort III fractures is the highest risk. Finally, addressing the basis for anticoagulation, this has not been very scientific. We began systemic heparin based on the Memphis series. We did reduce our partial thromboplastin time target to 40/50 seconds to minimize bleeding complications and, in fact, have been successful in that arena. However, low-molecular-weight heparin is appealing at more therapeutic levels. In patients who have prohibitive risks for anticoagulation, we used aspirin and clopidogrel as an alternative, and, serendipitously, we found that this appeared equally protected. In fact, that is the basis of a current randomized study in our institution in which grade I to III injuries are randomized to either heparin or antiplatelet therapy. The issue of stents for grade III injuries is a real conundrum. In the literature, there are reports of devastating complications associated with carotid pseudoaneurysms, but frankly, we have not observed these. I need to clarify 1 point; the 2 events that we described were in an era before we started presumptive anticoagulation. Subsequently, we have convinced ourselves that intervention for stenting should be delayed at least 7 days because the clot in the early period is prone to embolization. We currently have 19 patients who have had stenting done under anticoagulation. In this group, we have had 1 significant early event, and, alarmingly, 3 patients have had late occlusions. Thus, if stents are used, we agree they should be applied selectively until more data are available.

Thomas H. Cogbill, MD, La Crosse, Wis: I commend the Denver group for the careful and systematic way in which they have studied the screening and management of blunt cerebrovascular injuries. We witnessed another excellent analysis today. I have 2 questions for the authors: What is your present protocol for duration of therapy? I am aware that you will frequently perform follow-up angiographic evaluation of identified injuries. How have these follow-up studies affected the type and duration of your continued therapy?

Dr Moore: We are pleased to hear your review of our material, acknowledging your leadership on the Western Trauma Multicenter group that brought this entity into focus in 1994. At this point, we currently advocate warfarin for 3 months, but again, this is with little scientific basis. It may be too long for the grade I or II injuries, but too short for the persistent grade III injuries. The arrival of new antiplatelet agents may offer a safer alternative. As to the follow-up angiography, these data are available in our *Annals of Surgery* report last year. We routinely perform follow-up angiography between 7 and 10 days. Remarkably, 70% of our grade I injuries and 10% of our grade II injuries have resolved by 7 days. Consequently, we can then terminate anticoagulation and do not subject these patients to the obligatory risk. Conversely, 8% of grade I injuries and 43% of the grade II injuries progress into pseudoaneurysms, and it is conceivable that these patients are candidates for more prolonged therapy or stenting.

Clayton H. Shatney, MD, Aptos, Calif: Dr Moore, I am trying to compare apples to apples here in your data. It looks to me that the comparison groups would be the 73 asymptomatic patients who got anticoagulation vs the 27 patients who were asymptomatic and not anticoagulated, because as I understand from the presentation, the other 14 patients were symptomatic or had manipulation. And the reason that those 27 were not anticoagulated was there was some contraindication to anticoagulation. So my question is, what were the reasons that they could not be anticoagulated, and were some of those reasons, perhaps, contributory to those 5 who had a stroke and not necessarily to the fact that they did not get anticoagulated?

Dr Moore: Our described study group transcends a period before we began aggressive screening and presumptive anticoagulation. Thus, a number of these patients were not anticoagulated because that was not our practice before 1996. Subsequently, patients were not anticoagulated because of associated injuries. The most consistent reason for withholding anticoagulation is intracranial hemorrhage. But our neurosurgeons are generally comfortable if the hemorrhage is stable for greater than 24 hours.

Merril T. Dayton, MD, Buffalo, NY: As I was listening to the presentation this morning, it occurred to me that the only difference between group 2, which had the high stroke rate, and group 1 was that group 2 had a contraindication for anticoagulation, and I wondered as I listened to the presentation, is there possibly a selection bias regarding group 2 and the fact that perhaps they were more severely injured predicts a greater risk to have strokes anyway? Would you just address the possibility of a bias in that group 2, since it obviously wasn't a randomized cohort?

Dr Moore: It is important to acknowledge these were not matched groups. In fact, many of those without anticoagulation were prohibited because of associated injuries, and they theoretically would be prone to early thrombosis because of the coagulation abnormalities associated with acute trauma. However, we submit that the lack of ischemic events in the 73 anticoagulated patients is compelling and, frankly, feel it would be unethical to embark on a randomized study without anticoagulation in the control group.

On behalf of my coauthors, we appreciate the opportunity to share this material with you and thank you for your interest.