Blunt cerebrovascular injuries: Redefining screening criteria in the era of noninvasive diagnosis

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Author Disclosures: All authors have nothing to disclose.

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Submitted: September 1, 2011, Revised: October 4, 2011, Accepted: October 17, 2011.
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Presented at the 2011 American Association for the Surgery of Trauma Annual Meeting, Chicago, Illinois.
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DOI: 10.1097/TA.0b013e31823de8a0

BACKGROUND: Screening for blunt cerebrovascular injuries (BCVIs) and early treatment has virtually eliminated injury-related strokes. Screening protocols developed in the 1990s captured ~80% of ultimately identified BCVI. With the availability of noninvasive diagnosis with computed tomographic angiography, broader indications for screening seem warranted. The purpose of this study was to identify injury patterns of patients with BCVI that are not currently recommended screening criteria.

METHODS: Our prospective BCVI database, initiated in 1997, was queried through December 2010. Indications for screening, injury mechanism, and outcomes were analyzed. Patients younger than 18 years were excluded.

RESULTS: During the 14-year study period, 585 BCVIs were identified in 418 patients (66% men; age, 40 years ± 0.7 years). Eighty-three (20%) patients with BCVI did not have standard screening criteria; 66% were asymptomatic at diagnosis. Injury patterns in these patients included mandible fracture (27 patients), complex skull fractures (21 patients), traumatic brain injury with thoracic trauma (6 patients), scalp degloving (6 patients), and great vessel or cardiac injuries (4 patients). Other injuries (11 patients) and no injuries (8 patients) were identified in the remainder. Of the 307 asymptomatic patients who received antithrombotic treatment, one patient suffered stroke (0.3%) and one patient a transient ischemic attack (0.3%).

CONCLUSIONS: A significant number of patients suffering BCVI are not captured by current screening guidelines. Screening for BCVI should be considered in patients with mandible fractures, complex skull fractures, traumatic brain injury with thoracic injuries, scalp degloving, and thoracic vascular injuries. (*J Trauma.* 2012;72: 330–337. Copyright © 2012 by Lippincott Williams & Wilkins)

LEVEL OF EVIDENCE: II, prognostic study.

KEY WORDS: Cerebrovascular injury; carotid artery injury; vertebral artery injury; trauma; antithrombotics; screening criteria; guidelines.

Screening for blunt cerebrovascular injuries (BCVIs) and early antithrombotic therapy in patients with identified injuries has virtually eliminated injury-related strokes. Some patients with BCVI present with symptoms of cerebral ischemia within an hour of injury; unfortunately, treatment in these patients is often unsuccessful.[1,2] Fortunately, the majority of patients with BCVI have a latent period between their original injury and the onset of neurologic events. This timeframe can range from hours up to 14 years, but the majority develops symptoms within 72 hours.[1,3–9] Diagnosing BCVI during this "silent period" provides the opportunity for treatment before neurologic sequelae. These facts provide the basis for developing guidelines for BCVI screening, based on patient injury patterns and mechanism of injury, to identify these injuries in asymptomatic patients. The Memphis group[10] and our group[11] developed screening protocols in the 1990s, but these screening criteria only capture 75% to 85% of patients ultimately identified with BCVI.[11–13] BCVI screening protocols have remained relatively unchanged over the past decade since their institution. With the availability of noninvasive diagnosis with computed tomographic angiography (CTA), broader indications for screening have been suggested. The purpose of this study was to identify injury patterns of patients with BCVI that are not currently recommended by standard screening criteria.

TABLE 1. Denver Screening Criteria for BCVI

Signs/symptoms of BCVI
Arterial hemorrhage
Cervical bruit
Expanding cervical hematoma
Focal neurologic deficit
Neurologic examination incongruous with head CT scan findings
Stroke on secondary CT scan
Risk factors for BCVI
High-energy transfer mechanism with:
LeForte II or III fracture
Cervical-spine fracture patterns: subluxation, fractures extending into the transverse foramen, and fractures of C1–C3
Basilar skull fracture with carotid canal involvement
Petrous bone fracture
Diffuse axonal injury with GCS score <6
Near hanging with anoxic brain injury

GCS, Glasgow Coma Scale.

METHODS

Denver Health Medical Center is a state-certified and American College of Surgeons-verified Level I regional trauma center and integral teaching facility of the University of Colorado, Denver. We initiated a screening protocol for BCVI based on injury mechanism, injury patterns, or symptoms in 1996 (Table 1). Screening criteria were modified in 2005 after our evaluation of the relationship of cervical spine fracture patterns and BCVI.[14] Although we initially screened all patients with cervical spine fractures except spinous process fractures, we currently screen only patients with evidence of subluxation, fractures extending into the transverse foramen, and fractures of any part of C1-C3. The decision to screen a patient for BCVI outside the noted guidelines was attending dependent.

We currently use multislice CTA as our standard screening test for patients at risk for BCVI. Dedicated neuroradiologists overread every CTA performed for BCVI screening. Digital subtraction four-vessel angiography was used to evaluate patients for the first 10 years of the study period. Patients' injuries were classified according to the Denver grading scale[7] (Table 2). Once a BCVI was identified, antithrombotic therapy was started unless contraindicated by an associated injury. The modified anticoagulation protocol involves initiating systemic heparin using a continuous infusion of heparin at 15 U/kg/h, without a loading dose;[7,15] heparin drips were titrated to achieve a partial thromboplastin time between 40 seconds and 50 seconds. Alternatively,

TABLE 2. Denver Grading Scale for BCVI

Grade I: irregularity of the vessel wall or a dissection/intramural hematoma with <25% luminal stenosis
Grade II: intraluminal thrombus or raised intimal flap is visualized, or dissection/intramural hematoma with 25% or more luminal narrowing
Grade III: pseudoaneurysm
Grade IV: vessel occlusion
Grade V: vessel transection

antiplatelet agents (aspirin 325 mg/d and/or clopidogrel 75 mg/d) were administered at the discretion of the surgical attending.

Our prospective BCVI database was queried from the time of initiation in January 1997 to January 2011. Indications for screening, injury mechanism, BCVI grade, treatment, and outcomes over the 14-year period were analyzed. Patients younger than 18 years were excluded. Statistical analysis was performed using SAS for Windows (SAS Institute, Cory, NC). The Colorado multi-institutional review board approved this study.

RESULTS

During the 14-year study period, 585 BCVIs were identified in 418 patients (66% men; mean age, 40 years ± 0.7 years). BCVIs diagnosed included 158 patients with carotid injuries, 198 patients with vertebral artery injuries, and 62 patients with combined carotid and vertebral injuries. Of the 418 patients with BCVI, 313 (75%) patients were evaluated based on previously defined screening guidelines (Table 3), 83 (20%) patients with BCVI did not have our defined screening criteria, and 22 (5%) of these patients presented with neurologic findings.

Injury patterns that triggered screening for BCVI in these 83 patients are presented in Table 4. The predominant single injury that triggered screening was a mandible fracture (27 patients). Skull fractures with or without extension into the orbits were identified in 21 patients; fracture patterns included any basilar skull fracture (not just those extending to the carotid canal), occipital condyle fractures, and frontal skull fractures. Six patients with a reported high mechanism of injury with traumatic brain injury (TBI) and thoracic trauma were identified. Scalp degloving was the screening trigger in six patients. Injuries to the great vessels or blunt cardiac rupture were the indication for BCVI evaluation in four patients. The remaining 11 patients had a myriad of injuries including the following: (1) ejection motor vehicle collision (MVC) with maxillary sinus fracture and thoracic injuries; (2) thrown from horse with epidural hemorrhage and intraparenchymal hemorrhage; (3) 10 foot fall with neck pain, clavicle fracture, and a scalp laceration; (4) assault with subdural hemorrhage and multiple facial and neck lacerations; (5) head-on MVC into a semi with maxilla fracture and multiple lower extremity fractures; (6) unrestrained MVC with movement from back to front seat with intraventricular hemorrhage; (7) fall with epidural hematoma with midline shift; (8) bike accident with T7 fracture, clavicle fracture, complaints of neck pain; (9) fall with subarachnoid hemorrhage; (10) head-on MVC at 40 mph with a nasal fracture; and (11) fall with subarachnoid hemorrhage. Eight patients had no identified injuries aside from their BCVI, and all were symptomatic at the time of diagnosis. Table 5 illustrates the injury grades identified in all patients compared with the study population.

TABLE 3. Patients With BCVI Stratified by Screening Indication

Current Screening Guidelines	Number of Patients Diagnosed With BCVI
Cervical spine fx patterns	223
Basilar skull fx with carotid canal involvement	33
Diffuse axonal injury with GCS score <6	23
LeFort II or III fx	20
Neurologic examination incongruous with head CT scan	6
Hanging with anoxic brain injury	5
Expanding cervical hematoma	3

fx, fracture; GCS, Glasgow coma scale.

TABLE 4. Patients With BCVI Without Standard Screening Indications Stratified by Injury

	Asymptomatic Patients (n = 55)	Patients With Stroke (n = 28)
Mandible fx	21	6
Any basilar skull fx/occipital condyle fx	12	3
Frontal skull fx and orbit fx	6	0
High-mechanism injury with TBI and thoracic injuries	2	4
Scalp degloving	4	2
Great vessel or cardiac injury	4	0
Other	6	5
No injuries	0	8

TABLE 5. Comparison of Injury Grades for All Patients With BCVI Versus Those With No Screening Indications

	Injury Grade	CAI All Patients (n = 418)	CAI No Screening Indications (n = 83)	VAI All Patients (n = 418)	VAI No Screening Indications (n = 83)
No stroke patients	I	125	29	128	11
	II	35	5	44	4
	III	43	16	31	3
	IV	4	2	62	1
	V	3	0	2	0
Stroke patients	I	22	5	9	5
	II	16	12	9	3
	III	18	11	7	0
	IV	2	2	10	1
	V	6	0	1	1

Eight injuries were not specified.
CAI, carotid artery injury; VAI, vertebral artery injury.

Of the 307 asymptomatic patients who received antithrombotic treatment, one patient suffered stroke (0.3%) and one patient a transient ischemic attack (0.3%). In the study group, 28 (34%) patients suffered a stroke. Mean time to stroke was 44 hours ± 12 hours with nine patients manifesting neurologic symptoms within 6 hours of injury. Death in three patients in study group was attributed to their BCVI-related stroke.

DISCUSSION

Blunt trauma patients continue to be diagnosed with BCVI who do not have injuries consistent with current screening guidelines. Diagnosis is often because of the "gestalt" of the trauma surgeon who orders the imaging, based on mechanism and constellation of injuries that are consistent with cervical hyperextension, and "we should screen this patient although they do not fit classic criteria" judgment. Alternatively, some centers perform screening if the patient is undergoing CTA of the chest or imaging of the head and neck. Unfortunately, there are also patients who are diagnosed with BCVI with the onset of neurologic symptoms, often >12 hours after injury. Better qualifying the screening triggers that do not fit the "standard published criteria" would aid less experienced clinicians in the diagnosis of these injuries and could prevent stroke in those asymptomatic BCVI patients treated early.

Although BCVI were initially recognized >30 years ago,[16] routine screening for these injuries was not initiated until the late 1990s. At that time, screening for BCVI was advocated because of the recognition of both an asymptomatic period and that specific patterns of injuries were associated with BCVI.[3,10,11,17] The asymptomatic postinjury period has been inferred based on the time to onset of symptoms in patients with diagnosed injuries who did not receive antithrombotic therapy. This timeframe seems to range from hours up to 14 years, but the majority seems to develop symptoms within 10 hours to 72 hours.[3,5–7] The goal is to diagnose BCVI during this latent period before the onset stroke. Multiple studies affirm that if you diagnose these injuries during the asymptomatic period, early treatment with antithrombotics virtually eliminates neurologic sequelae.[1,5,18,19] The Memphis group showed a reduction in stroke rate for carotid artery injury from 64% in untreated patients to 6.8% in patients treated with antithrombotics (either anticoagulation or antiplatelet agents), and for vertebral artery injury a reduction from 54% to 2.6% in treated patients.[19] Their most recent published series demonstrates a postdiagnostic stroke rate of 4% with a combination of medical and endovascular therapy used.[20] Our group's recent published series demonstrated a stroke rate of 0.3% in 282 patients with BCVI treated with antithrombotics, whereas untreated patients had an overall stroke rate of 21%.[1] This study had two treated patients who suffered neurologic ischemia, one of whom had transient symptoms, for an overall stroke incidence of 0.2% in treated patients.

Screening indications for BCVI have been debated over the past decade. In fact, the controversy runs the full spectrum; some have questioned the utility of aggressive screening,[2] whereas others have argued that the screening criteria should be broadened.[21–23] But the Memphis and Denver groups have agreed on a recommended screening profile. However, our early series that described these risk factors also noted 20% of patients with BCVI had none of these high-risk injury patterns.[11] The recent analyses from the Memphis[12] and Shock Trauma[13] groups corroborate that ~20% of patients with BCVI have no conventional screening criteria. It is for this reason that the majority of screening protocols include the phrase "injury mechanism" as a viable screening trigger.

While reviewing the current literature, it is difficult to deduce a single screening protocol, but the agreed upon high-risk factors are reasonably concurrent. Our published screening criteria include patients with signs of BCVI (arterial hemorrhage, cervical bruit, expanding cervical hematoma, focal neurologic deficit, neurologic examination incongruous with head computed tomographic (CT) scan findings, and stroke on secondary CT scan); screening is also performed in patients with risk factors for BCVI, which includes LeFort II or III fracture, specific cervical-spine fracture patterns (subluxation, fractures extending into the transverse foramen, and fractures of C1-C3), basilar skull fracture with carotid canal involvement, petrous bone fracture, diffuse axonal injury with Glasgow Coma Scale score <6, and near hanging with anoxic brain injury.[1,11] The Memphis group's standard criteria for angiographic screening includes patients with skull base fractures, Horner's syndrome, cervical spine fractures, LeFort II or III fractures, neck soft tissue injuries, and neurologic examination not consistent with radiologic findings.[12,24] More recently, they seem to be using CTA imaging to screen a larger cohort of patients including those with loss of consciousness, altered mental status, periorbital or supramastoid ecchymosis, any potential cervical spine or neck injury, and any potential facial fracture or facial deformity.[12] Recent publications by the Western Trauma Association and the Eastern Association for the Surgery of Trauma incorporate elements from both the Memphis and Denver groups' original guidelines,[25,26] but do not extrapolate beyond these initial screening criteria.

Expanding screening criteria for BCVI to capture all injuries is attractive, particularly with the improved accuracy and rapid acquisition of noninvasive screening modalities.[27–32] In addition to the injury patterns previously identified, several groups have published on the association of BCVI with mandible fractures, TBI, and thoracic injuries.[13,22,23,30] Our current evaluation supports these recommendations. The most common injury observed was a mandible fracture, observed in 33% of the study group. Broadening our screening guideline to include all basilar skull fractures or occipital condyle fractures rather than only those with extension to the carotid canal would have captured an additional 15 patients. Patients with a high mechanism of injury and TBI plus thoracic injuries were noted in six patients. Three potential screening indications that have not previously been reported to our knowledge that emerged from this analysis included complex frontal skull fractures with

orbit involvement, scalp degloving injuries, and cardiac or great vessel injuries.

This study underscores the importance of clinical acumen. Injury pattern alone does not direct screening; rather, the incorporation of mechanism combined with patient injuries determines optimal imaging. A recent analysis of injury pattern modeling using the National Trauma Data Bank adult blunt trauma cohort indicated that 96% of patients would have to be screened to identify all BCVIs if injury patterns alone were used.[33] Although CTA is a noninvasive imaging option, there is still a small risk of radiation and intravenous contrast exposure.[34] One emerging option is the use of whole-body multidetector computed tomographic scan which uses a single contrast bolus for craniocervical and thoracic evaluation in addition to standard CT scan imaging of the head, neck, and thorax. Although early reports are encouraging,[13,35,36] this modality is not adequately investigated. Perhaps most importantly, CTA allows early diagnosis and therapeutic intervention in asymptomatic patients to prevent stroke.[27] In treated patients, our stroke rate was 0.2%, the lowest reported in the literature.

Unfortunately, there is a group of patients who will present with neurologic symptoms within 1 hour to 2 hours of admission; these patients cannot be effectively screened and treated because of the early onset of neurologic sequelae. Fortunately, however, this group of patients represents the minority of patients with BCVI. The majority of patients have a latent period that permits timely diagnosis and intervention. In our study, those patients not presenting with symptoms had a mean time to stroke of 44 hours, with only 9 patients manifesting neurologic symptoms within 6 hours of injury. Therefore, screening in the at-risk population should be aggressively pursued and treatment appropriately initiated based on associated injuries and physiologic indices.

This study has its limitations. This was not a randomized controlled trial. As such, the decision to screen a patient for BCVI outside the published guidelines from our institution was attending dependent. The number of patients with any single injury pattern identified in this study was small. Without the denominator of total number of patients screened with each new identified injury pattern, we are unable to determine the overall yield of the expanded screening criteria. However, with the significant mortality of BCVI-related stroke, one could argue that expanded screening policies with associated clinical judgment are appropriate and timely. We did not analyze renal insufficiency in these patients with

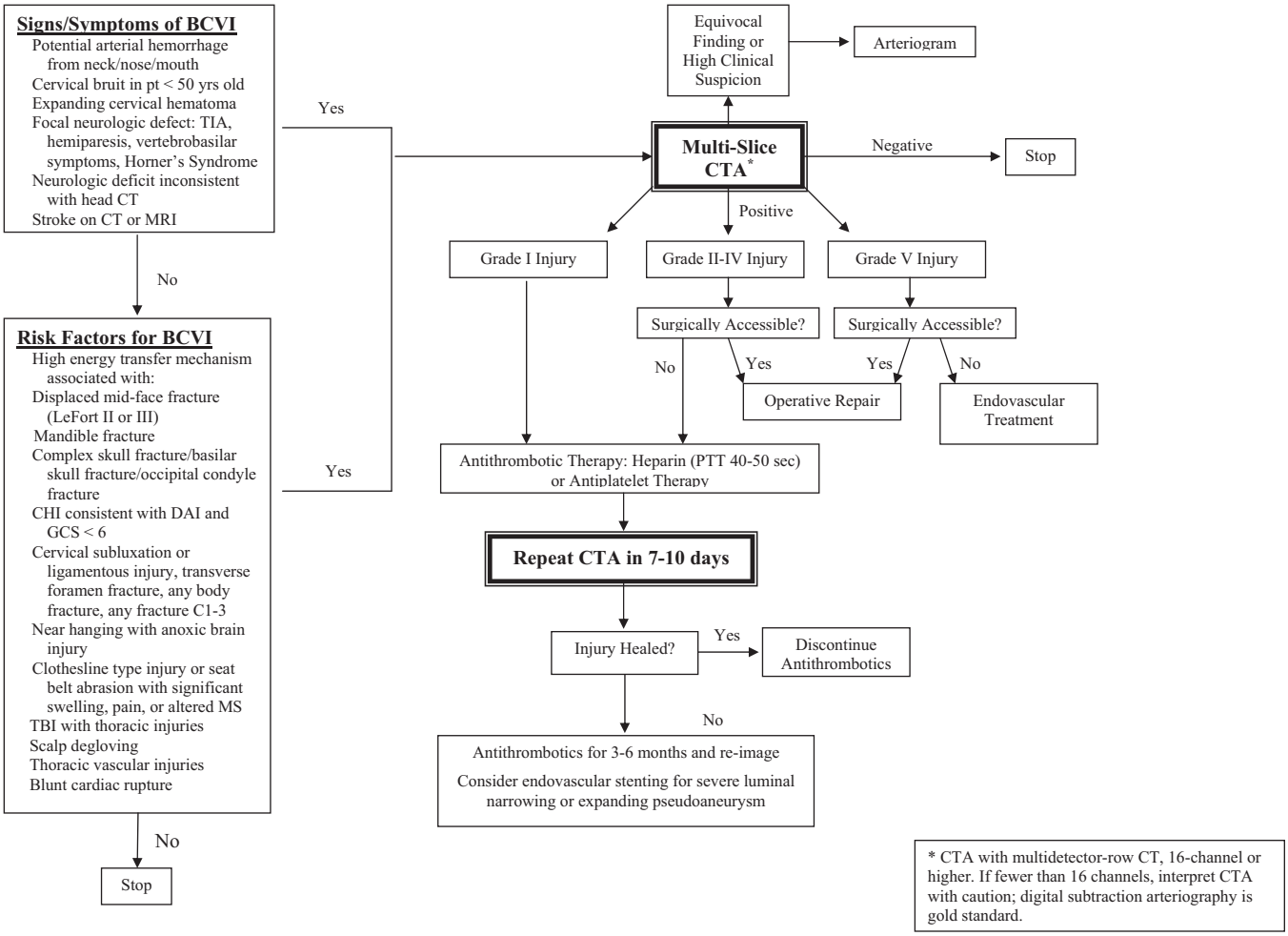

Figure 1. The new Denver Health Medical Center BCVI screening guideline.

multiple injuries, and hence cannot correlate the potential for contrast induced nephropathy. To our knowledge, no patient required dialysis because of contrast-related renal failure. Finally, children were excluded from this study as they may have different injury profiles and screening indications.[37]

Diagnosis and treatment of BCVI continues to evolve. With BCVI diagnosed in >1% of blunt trauma patients and the recognition of a clinically silent period, diagnosis of these injuries should be aggressively pursued. Stroke in asymptomatic patients can almost universally be avoided with antithrombotic treatment. With 20% of patients suffering BCVI not captured by current screening guidelines, clinical acumen and a high index of suspicion based on injury mechanism and associated injuries should prevail. Screening for BCVI should be considered in patients with mandible fractures, complex skull fractures or cranial injuries, and upper thoracic injuries with TBI. BCVI screening algorithms should be modified to include these expanded criteria (Fig. 1).

AUTHORSHIP

C.C. Burlew, W.L.B., and E.E.M. designed this study and collected, analyzed, and interpreted the data. C.C. Burlew, and E.E.M. prepared the manuscript, which was critically reviewed by W.L.B., C.C. Barnett, J.L.J. and D.B.

DISCLOSURE

The authors declare no conflict of interest.

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DISCUSSION

Dr. Timothy C. Fabian (Memphis, Tennessee): The Denver group is to be congratulated for continuing to chip away at this interesting and fairly important lesion. We're not yet identifying 100% of the blunt cerebrovascular lesions, but we're getting closer.

Dr. Burlew has presented a large series of patients who were studied with the purpose of identifying injury patterns in patients with cerebrovascular injuries that aren't currently in their institutional screening criteria.

Consistent with their previous work and work done in Memphis and other places, the group from Denver demonstrated that up to 5% of patients present with stroke, about 75% are asymptomatic, and 20% do not meet standard screening criteria. Those 20% of patients are the group we must identify early so we can hopefully prevent strokes with appropriate treatment.

We can quibble about the best method for definitive diagnosis, arteriogram versus CTA, or the optimal treatment strategy, anticoagulation with or without stenting, but that's not what this paper is about. It's about screening trauma patients for this potentially devastating injury. It is about identifying that 20% of patients with cerebrovascular injuries who don't meet standard screening criteria. It is about finding this 20% before they have a stroke. The authors did this by describing other injury patterns associated with BCVI, injuries such as mandible fractures, skull and scalp injuries, great vessel or cardiac injuries and a potpourri of others.

I have really only two questions. One, why not screen patients with any cervical spine fracture? You screened those with C1 to C3 injuries based on the previous 2007 study. But even in that study you identified eight patients or 3% with lower cervical spine fractures who had BCVI.

Last year at this meeting Dr. Emmett from our institution showed that 9% of patients with cerebrovascular injuries would have been missed had we screened only those with upper cervical fractures. In fact, the 3% increase of BCVI for lower cervical spine fractures in your previous study is similar to your new criteria of TBI with thoracic injuries which is 4%. Please help me understand why we shouldn't screen patients with C4 to C7 cervical spine fractures.

My second question deals with the matter of simplicity. As you know we use CTA as a screening tool in Memphis and not for definitive diagnosis. The patients who get CTA are those with the standard injury patterns screening we all agree on, and CTA is done on patients who otherwise would receive a CT of their head, thus, the screening criteria become much simpler.

While your screening guideline is impressive, the patients you added all would likely be receiving a head CT. That is the common denominator between your institution and our institution's attempts to find this important missing 20%. Please comment on this fairly simplistic approach. We don't get very complicated in Memphis.

Once again, our Denver colleagues have carefully reviewed their patients in an attempt to identify the 20% of patients who are missed by standard screening criteria. This is an important piece of work. Again, they continue to chip away and have added a tremendous amount to improved outcomes in these patients.

Dr. Gregory J. "Jerry" Jurkovich (Seattle, Washington): I have three specific questions: One, could you describe the mechanisms of injury related to mandible fractures since that was the largest category of patients who were not usually screened?

We have included mandible fractures that occur as a result of motor vehicle crashes in our screening protocol but not those that occur as assault. Does it make a difference of how the mandible fracture occurred?

Number two, regarding the use of post-injury recognition anticoagulation, are you using aspirin or Plavix or Coumadin or heparin? What do you specifically mean by "anticoagulation" ?

And in theory could you just put everyone who has any trauma to the head and neck and at any risk of blunt cerebrovascular injury on aspirin and then stop all the screening?

Third question, can you tell us a little bit more about the patients who had a stroke? How sure were you that the stroke was related to their blunt cerebrovascular injury and not a primary neurologic or cardiac event and not to their cerebrovascular injury? Maybe the age of the people who had a stroke might help with that.

Dr. Clay Cothren Burlew (Denver, Colorado): Thank you very much Dr. Fabian. Your contributions in this field encourage us to continue our ongoing research. I'd also like to thank Dr. Croce for kindly reviewing our manuscript.

Regarding your first question, Dr. Fabian, about screening all patients with cervical spine fractures - this is a screening criteria that is often debated. We previously screened patients with upper cervical spine fractures, those with subluxation or those with transverse foramen involvement. Now that we've shifted to CTA rather than angiography for routine screening, perhaps broader criteria for screening cervical spine fractures is indicated. That being said, I wouldn't necessarily screen a patient who has an isolated spinous process fracture. I think expanding our criteria and incorporating mechanism is exactly how we identify the 20% of patients who don't currently fit screening guidelines. For example, a patient with a high mechanism of injury, an orbit fracture, a C6 fracture, and upper rib fractures, should be screened. I think a middle ground on screening cervical spine fractures is probably where we stand.

Regarding overall screening criteria, I gather from your comments that all patients undergoing head CT scan at your institution also have a CTA performed. We still use mechanism of injury and injury patterns to determine need for BCVI screening, recognizing that some of our CTAs are obtained in a delayed fashion based upon identified injuries. In this era of health care, with the Senate currently debating financial cuts for the medical field, some element of cost-consciousness in evaluating patients with imaging studies is warranted. We have not adopted a universal screen-everybody-that-comes-in-the-front-door, which is probably the only way we will capture 100 percent of these injuries.

Dr. Jurkovich, your question about mandible fractures and should we screen patients with multiple types of mech-

anisms. We do screen patients with mandible fractures following an assault, and several of our patients had identified carotid or vertebral artery injuries. I assume that these patients have had a hyperextension injury in relation to the assault or other mechanism.

Regarding antithrombotics, our current practice at Denver Health is to treat patients acutely with intravenous heparin. Occasionally we will have a patient with an identified BCVI that is going to be discharged 24 hours later and we will start those patients on aspirin. But the majority of our patients are multiply injured and our neurosurgeons, particularly, are not keen on antiplatelet agents; we treat patients with heparin in the acute phase, since it is easier to correct if a bleeding complication occurs, and as the patient is about to be discharged we transition them to aspirin. Theoretically, you could give everyone an aspirin rather than screen them, but I tend to think diagnosis followed by heparin is the way to go.

Regarding whether or not the stoke is related to their BCVI rather than an antecedent event is an excellent question. Admittedly, I would assume that the 30-year old with a stroke is most likely related to their diagnosed carotid or vertebral injury versus atherosclerotic disease or embolic phenomenon; however, I can't say that is 100% confirmed.