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The Devastating Potential of Blunt Vertebral Arterial Injuries

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Objective
To formulate management guidelines for blunt vertebral arterial injury (BVI).

Summary Background Data
Compared with carotid arterial injuries, BVIs have been considered innocuous. Although screening for BVI has been advocated, particularly in patients with cervical spine injuries, the appropriate therapy of lesions is controversial.

Methods
In 1996 an aggressive arteriographic screening protocol for blunt cerebrovascular injuries was initiated. A prospective database of all screened patients has been maintained. Analysis of injury mechanisms and patterns, BVI grades, treatment, and outcomes was performed.

Results
Thirty-eight patients (0.53% of blunt trauma admissions) were diagnosed with 47 BVIs during a 3.5-year period. Motor vehicle crash was the most common mechanism, and associated injuries were common. Cervical spine injuries were present in 71% of patients, but there was no predilection for cervical vertebral level or fracture pattern. The incidence of posterior circulation stroke was 24%, and the BVI-attributable death rate was 8%. Stroke incidence and neurologic outcome were independent of BVI injury grade. In patients treated with systemic heparin, fewer overall had a poor neurologic outcome, and fewer had a poor outcome after stroke. Trends associated with heparin therapy included fewer injuries progressing to a higher injury grade, fewer patients in whom stroke developed, and fewer patients deteriorating neurologically from diagnosis to discharge.

Conclusions
Blunt vertebral arterial injuries are more common than previously reported. Screening patients based on injury mechanisms and patterns will diagnose asymptomatic injuries, allowing the institution of therapy before stroke. Systemic anticoagulation appears to be effective therapy: it is associated with improved neurologic outcome in patients with and without stroke, and it appears to prevent progression to a higher injury grade, stroke, and deterioration in neurologic status.

Blunt vertebral arterial injury (BVI) has historically been considered an uncommon event of relative insignificance. There have been many case reports describing BVI-associated cerebrovascular accidents, both ischemic and hemorrhagic, but clinical series of vertebral artery injuries have been composed primarily of penetrating injuries. In the seven largest reports, only 8 (4%) of 195 total reported patients had sustained blunt mechanisms; these 8 patients as a group had reasonably good outcomes, leading to the belief that BVI is relatively innocuous.[1–7] This concept was further supported in small series dealing specifically with blunt injuries.[8–14]

In 1996 we adopted an aggressive screening protocol for blunt carotid arterial injuries.[15] Using four-vessel cerebral arteriography, we began identifying a significant number of BVIs in addition to blunt carotid injury. Fabian et al[16] previously demonstrated the benefits of systemic heparin therapy in treating patients with blunt carotid injury; our experience with asymptomatic patients affirmed the Memphis data.[15] Extrapolation of blunt carotid injury treatment principles to BVI, however, has not been supported. Col-

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lectively, the consensus in the literature to date is that asymptomatic patients with narrowing, irregularity, or occlusion of the vertebral artery do not require treatment.[1–7,11,12] The purpose of this study was to analyze our experience and formulate a rational diagnostic and therapeutic approach to BVI.

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. Since mid-1996, patients selected for cerebral arteriography to exclude BVI or blunt carotid injury have been identified and entered into a prospectively maintained database. Detailed analysis of these patients' records was approved by the Colorado Multi-Institutional Review Board.

Diagnosis

The diagnosis of BVI or blunt carotid injury is confirmed by four-vessel cerebral arteriography. Digital subtraction techniques are used, and all studies include the aortic arch and cerebral vessel origins. Injured patients undergo emergent arteriography for any of the following signs or symptoms suggestive of cerebrovascular injury: hemorrhage from the mouth, nose, ears, or wounds of potential arterial origin; expanding cervical hematoma; cervical bruit in a patient younger than 50 years; evidence of cerebral infarction on computed tomography; and unexplained or incongruous central or lateralizing neurologic deficit, transient ischemic attack (TIA), amaurosis fugax, or Horner syndrome. In addition, at-risk asymptomatic patients (i.e., those exhibiting no suggestive signs or symptoms for BVI or blunt carotid injury) undergo prompt angiographic evaluation. The criteria for screening arteriography include an injury mechanism compatible with severe cervical hyperextension/rotation or hyperflexion, particularly if associated with displaced midface or complex mandibular fracture, or closed head injury consistent with diffuse axonal injury of the brain; near-hanging resulting in cerebral anoxia; seat belt abrasion or other soft tissue injury of the anterior neck resulting in significant cervical swelling or altered mental status; basilar skull fractures involving the carotid canal; and cervical vertebral body fracture or distraction injury, excluding isolated spinous process fractures. Follow-up arteriography is performed within 7 to 10 days when possible to evaluate the efficacy of the initial therapy. Recently, magnetic resonance angiography (MRA) has been used selectively in asymptomatic patients with preexisting renal disease, with all abnormal findings confirmed arteriographically.

Injury Grading

Cerebrovascular injuries are classified according to our previously described grading scale[17]:

- **Grade I:** arteriographic appearance of irregularity of the vessel wall or a dissection/intramural hematoma with less than 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:** pseudoaneurysms
- **Grade IV:** vessel occlusions
- **Grade V:** transections

Treatment

Systemic heparin is administered to patients who have no contraindications. A continuous infusion of unfractionated heparin is initiated at 15 U/kg/hr, without bolus dosing, and is adjusted to maintain the partial thromboplastin time at 40 to 50 seconds. Patients with relative contraindications to systemic heparin are given an antiplatelet agent, low-dose subcutaneous heparin, or low-molecular-weight heparin. Patients with absolute contraindications to systemic heparin are observed without specific treatment. One patient—the first in the series—had embolization of an occluded vertebral artery.

Outcome

Neurologic function of surviving patients is classified as severe deficit (institutionalized or requiring assistance at home with activities of daily living), mild deficit (independent in activities of daily living but with residual cognitive or sensorimotor deficit), or normal.

Data Analysis

Data were analyzed using Microsoft Excel version 7.0 software (Microsoft, Redmond, WA). Statistical analysis was performed on an IBM-compatible personal computer using StatMost 32 for Windows 95 (DataMost Corp., Sandy, UT) and SPSS 9.0 for Windows (SPSS, Inc., Chicago, IL). Means of continuous data were compared using the Student *t* test and are expressed as mean ± standard error of the mean. Categorical data were compared using the Fisher exact test or chi-square analysis, where appropriate.

RESULTS

Patients

From May 1996 through October 1999, 7,205 patients were admitted to our institution after suffering blunt trauma. Vertebral arterial injuries were diagnosed by cerebral arteriography in 38 patients, for an incidence of 0.53% among

all patients admitted with blunt trauma. The left vertebral artery was injured in 27 (71%) and the right vertebral artery in 20 (53%); 9 patients (24%) suffered bilateral BVI. Twelve patients (32%) had associated blunt carotid injury. The mean age of the patients was 38.9 ± 2.3 years (range 16–77). Males made up 66% of the total group (25 patients).

Injury Mechanism and Associated Injuries

The mechanism of injury was a motor vehicle crash in 22 (58%), a fall in 5 (13%), and a pedestrian struck in 4 (11%); other mechanisms were uncommon and are listed in Table 1. The mean injury severity score was 25.5 ± 2.7 (median 22, range 9–75). Patients with associated blunt carotid injury had a higher mean injury severity score (33.1 ± 5.0, median 32) than those without blunt carotid injury (22.0 ± 3.0, median 18) ($P = .056$). Associated injuries, which were present in 35 (92%) patients, included injury to the spine in 27 (71%), chest and extremities in 17 (45%) each, head in 13 (34%), abdomen in 9 (24%), and pelvic fracture in 7 (18%) patients.

Cervical spine injuries were found in 27 patients (71%); the injuries are detailed in Table 1. Nine (33%) of the 27 patients had fractures at multiple vertebral levels. There did not appear to be a preponderance of injuries at any particular level: fractures were present at C1 in five, C2 in eight, C3 in three, C4 in two, C5 in nine, C6 in seven, and C7 in five patients. One patient suffered craniocervical dislocation, and one had ligamentous damage. The foramen transversarium was fractured in 6 (22%) of the 27 patients with cervical spine injury. Five patients (19%) had vertebral body subluxations, and five had facet dislocations.

Vertebral Arterial Injury Grade, Treatment, and Outcome

Table 1 lists the initial injury grade at diagnosis and the follow-up arteriographic injury grade. Initial lesions were grade I in 25, grade II in 9, grade III in 3, and grade IV in 10 instances. Twenty-one of the 47 injuries were reimaged; only 3 had improved, whereas 11 were unchanged and 7 had worsened. With the use of systemic heparin, there was a trend toward preventing arteriographic progression of the injuries: of 16 patients so treated and restudied, 2 (13%) improved, 10 (63%) remained unchanged, and 4 (25%) progressed to a higher grade. In contrast, of five patients who did not receive heparin, one (20%) improved and one remained unchanged, whereas 3 (60%) worsened ($P = .18$).

Twelve (32%) of the patients were neurologically normal at discharge. Twelve had mild deficits: four were attributable to spinal cord injuries, two to brain injuries, one to blunt carotid injury, one to brachial plexus injury, and four to the BVI. Seven (18%) patients had severe deficits at discharge, which were attributed to BVI in two patients and spinal cord injury in the other five. Of the seven deaths (18% of the total group), three were due to BVI, two to brain injury, one to blunt carotid injury, and one to multiple organ failure. Excluding patients whose outcome was influenced primarily by multiple organ failure or brain, carotid artery, spinal cord, or brachial plexus injury, the remainder were stratified by treatment. The outcome for those who received heparin versus those who did not is summarized in Table 2. Whereas 60% of those not receiving heparin had a poor outcome (death or severe deficit), only 6% of those given heparin had a poor outcome ($P < .05$). Moreover, deterioration in neurologic status from diagnosis to discharge occurred in 60% of patients not treated with heparin versus 19% of those treated systemically ($P = .11$) (Table 3).

Neurologic outcome of patients was stratified by injury grade (Table 4). There was no correlation between injury grade and outcome. This also held true when the analysis was limited to BVI-attributable complications and death (i.e., excluding patients whose outcome was influenced primarily by multiple organ failure or brain, carotid artery, spinal cord, or brachial plexus injury) (Table 5).

Ten patients suffered strokes; one of them had coexistent blunt carotid injury and sustained an infarction in the middle cerebral artery distribution. The nine (24% of the total group) who had posterior circulation strokes are highlighted in Table 6. There was no correlation between injury grade and stroke: the worst injury grade identified before stroke was I in three patients, II in two, III in one, and IV in three. The stroke incidence by grade was 19% for grade I, 40% for grade II, 13% for grade III, and 33% for grade IV. Neither bilaterality of BVI nor the presence of blunt carotid injury predicted stroke: 2 (17%) of 12 patients with coexistent blunt carotid injury had stroke, 3 (33%) of 9 with bilateral BVI had stroke, 0 of 4 with both blunt carotid injury and bilateral BVI had stroke, and 4 (19%) of 21 with isolated unilateral BVI had stroke. Two of the three with grade I BVI who suffered stroke had coexistent blunt carotid injury. Of unclear significance is the fact that eight (88%) of the nine patients who had stroke had left-sided BVI. Two patients had a dominant vertebral artery by arteriography; neither of them suffered stroke, even though the dominant artery was injured in both cases (and bilateral injuries were present in one of them). Seven patients had one vertebral artery that was more than 1.5 times the diameter of the contralateral vertebral artery. In three of the seven patients, the larger artery was injured, and one of them had a stroke; in the other four, the smaller artery was injured, and one of them had a stroke. Only one patient had a premonitory TIA before stroke. The mean time from injury to stroke was 4.3 days (range, 8 hours to 12 days); seven strokes (78%) occurred more than 48 hours after injury. Three patients were receiving heparin when the stroke occurred, and three were treated with heparin after the stroke. There may be a protective effect of heparin to prevent stroke: of 21 patients treated with heparin while asymptomatic, 3 (14%) had stroke; in contrast, 6 (35%) of 17 patients suffered stroke without

Table 1. INJURY MECHANISM, CERVICAL SPINE INJURY, VERTEBRAL ARTERIAL INJURY GRADE, TREATMENT, AND OUTCOME

Age/ Sex	Injury Mechanism	Cervical Spine Fx	BVI Grade*	Treatment	Follow-Up BVI Grade	Stroke	Outcome	Attributable M&M
16 M	Rollover MVC	C3 lamina; C5 FT; C5/6 Fx/subluxation	R IV	Embolization	—	—	Severe deficit	Spinal cord
48 M	Train vs. auto	C1 lateral mass	L III	Heparin	L III	—	Mild deficit	Brain
32 M	Tornado vs. trailer	Ligamentous injury	R I	Heparin	—	Yes	Dead	BVI
49 M	Auto vs. pedestrian	—	R III	Heparin	R III	—	Normal	—
35 M	MVC, near-hanging	—	L I	Heparin	—	Yes	Mild deficit	BVI
54 M	Rollover MVC	C4/5 facet Fx/dislocation	R I, L IV	Heparin	R I, L IV	—	Normal	—
77 M	Auto vs. pedestrian	C7 spinous process	R I, L I	Antiplatelet	—	—	Dead	Multiple organ failure
20 F	MVC	—	R I, L I	Heparin	—	Middle cerebral artery	Dead	BCI
32 F	MVC	—	R I, L I	Heparin	—	—	Dead	Brain
42 M	Auto vs. pedestrian	Craniocervical dislocation	R I, L I	Observation	—	—	Dead	Brain
32 M	20′ fall	C6 body, lateral mass; C7 body, transverse process	L I	Heparin	Healed	—	Severe deficit	Spinal cord
48 M	MVC	C5 facet; C6 pedicle/lamina/FT; C7 facet	L I	Observation	—	—	Mild deficit	Spinal cord
57 M	Assault	—	L I	Antiplatelet	—	Yes	Severe deficit	BVI
30 M	Snowboard fall	—	R IV	Heparin	—	—	Normal	—
35 M	10′ fall	—	L I	Heparin	—	—	Normal	—
37 M	MVC	Occipital condyle; C1 lateral mass	L I	Antiplatelet	L III	—	Severe deficit	Spinal cord
41 M	Motorcycle crash	—	L II	Heparin	—	—	Mild deficit	BCI
42 M	MVC	C5 body Fx/dislocation, FT	L IV	Observation	—	—	Normal	—
27 M	Rollover MVC	C2 body/pedicle/lamina/ FT	L I	Observation	—	—	Normal	—
31 F	Rollover MVC	C2 hangman's/FT	L II	Heparin	L II	Yes	Mild deficit	BVI
21 F	MVC	C1/2 facet dislocation; type III odontoid	L I	Observation	Healed	—	Mild deficit	Spinal cord
37 F	MVC	C1 posterior arch Fx	R II, L II	Heparin	R II, L II	Yes	Mild deficit	BVI
18 F	MVC	C5 pedicle/lamina	R II	Heparin	R III	—	Normal	—
33 M	Tree vs. pedestrian	C3 facet/lamina; C3/4 facet disruption	L IV	Observation	—	Yes	Dead	BVI
20 M	Auto vs. pedestrian	C2 body/facet; C3 lamina/pedicle; C4/5 facet	R I	Heparin	R I	—	Normal	—
48 F	Rollover MVC	C5 facet; C6 facet Fx/ dislocation	R IV	Antiplatelet	—	—	Mild deficit	Spinal cord
33 F	MVC	—	L III	Heparin	L III	—	Mild deficit	Brain
49 M	Rollover MVC	C2 hangman's	R IV, L IV	Heparin	—	Yes	Mild deficit	BVI
57 M	Tornado vs. tractor	C5/6 lateral mass Fx/subluxation	R IV, L II	Heparin	R IV, L III	—	Mild deficit	Spinal cord
44 F	MVC	C2 hangman's	R I	Heparin	—	—	Normal	—
25 F	Rollover MVC	C5 lamina; C6 body; C7 body/lamina Fx	R I, L I	Observation	R II, L III	Yes	Dead	BVI
25 M	20′ fall	C1 posterior arch; C2 body/facet/pedicle/ lamina; C2/3 subluxation; C6/7 spinous process	L II	Heparin	L III	—	Normal	—
25 M	Fall from horse	—	R I	Heparin	R III	—	Mild deficit	Brachial plexus
63 M	8′ fall	C1 anterior & posterior arch	R IV	Heparin	—	Yes	Mild deficit	BVI
66 M	MVC	C2 type III odontoid/FT	L II	Heparin	—	—	Normal	—
49 F	MVC	C6/7 facet lock/subluxation	L II	Antiplatelet	L II	—	Severe deficit	Spinal cord
24 F	30′ fall	—	R I	Heparin	Healed	—	Normal	—
41 F	Rollover MVC	C6/7 facet lock/subluxation	L I	Antiplatelet	—	—	Severe deficit	Spinal cord

BCI, blunt carotid artery injury; BVI, blunt vertebral artery injury; FT, foramen transversarium; Fx, fracture; M&M, morbidity and mortality; MVC, motor vehicle crash.
* Injury grades: I = irregularity of the vessel wall, dissection/intramural hematoma with <25% luminal stenosis; II = intraluminal thrombus or raised intimal flap, or dissection/intramural hematoma with ≥25% luminal narrowing; III = pseudoaneurysm; IV = occlusion.

heparin therapy ($P = .13$). All of the stroke victims' complications and death were attributed to the BVI, and the outcome was better in those treated with heparin. Of the six stroke victims who were treated with heparin, only one had poor outcome (death); all three not treated with heparin had poor outcome (two died and one was discharged with severe

Table 2. NEUROLOGIC OUTCOME IN PATIENTS WITHOUT SIGNIFICANT CONFOUNDING INJURIES, STRATIFIED BY TREATMENT

	Neurologic Outcome			
Treatment	Normal	Mild Deficit	Severe Deficit	Dead
Heparin	10	5	0	1
No heparin	2	0	1	2

deficits) ($P < .05$). Two patients sustained hemorrhagic strokes while receiving heparin therapy. There were no other bleeding complications associated with systemic anticoagulation.

DISCUSSION

Blunt vertebral arterial injuries remain poorly characterized. The literature is replete with case reports of BVI: participation in athletics (aerobics,[18] boxing,[19] football,[20] jockeying,[21] jogging,[22] judo,[23] paddleball,[24] skiing,[22] swimming,[25] volleyball,[26] and wrestling[27]), being bitten by a dog,[28] undergoing chiropractic cervical manipulation,[29] coughing,[30] "bottoms-up" drinking,[24] getting dressed in a tight diving suit,[22] "head banging" to music,[31] moving furniture,[24] parking a car,[22] roller coaster riding,[32] scolding a child,[33] seizing,[34] vomiting,[24] performing yard work,[24] and practicing yoga[35] have all been associated with BVI. Unfortunately, such bizarre case reports, although interesting, do not provide a scientific foundation on which to establish management policies. A relative paucity of institutional series has similarly precluded the formulation of sound practice guidelines for BVI. We have accumulated and presented herein a relatively large single-institution series, the analysis of which provides insight into several issues pertaining to the diagnosis and treatment of BVI.

Foremost in dealing with such an unusual but potentially devastating injury is determining whether the injury can be detected and effectively treated before complications occur. During the past several years, recognition of an association between BVI and cervical spine injuries has prompted investigators to screen for BVI in that clinical setting. In the first such prospective investigation, Louw et al[10] performed arteriography on patients who had facet joint dislocations of the cervical spine. Nine (75%) of their 12 patients were found to have vertebral artery occlusions, and consequently they suggested that a distraction-flexion injury was the most common cause of BVI. Woodring et al[14] performed arteriography on eight patients who had fractures involving the foramen transversarium of the cervical spine; seven (88%) had BVI. Willis et al[13] combined the strategies, screening patients with cervical trauma resulting in subluxation from a "locked" or "perched" facet, facet destruction, or foramen transversarium fracture. Of 26 patients undergoing arteriography for these indications, 12 (46%) were found to have BVI. More recently, investigators have screened for BVI using noninvasive testing. Friedman et al[8] performed MRA on 37 patients with cervical spine trauma; the results were abnormal for BVI in 9 patients (24%). Giacobetti et al[9] performed MRA on 61 patients with cervical spine trauma and found evidence of BVI in 12 (20%) of them. Weller et al[12] chose a more selective strategy, performing MRA only in patients with foramen transversarium fractures; they found BVI in 4 (33%) of 12 patients.

Our experience with screening patients for BVI has yielded similar results. We were purposely inclusive in establishing our blunt carotid injury and BVI screening

Table 3. CHANGE IN NEUROLOGIC STATUS IN PATIENTS WITHOUT SIGNIFICANT CONFOUNDING INJURIES, STRATIFIED BY TREATMENT

	Change in Neurologic Status		
Treatment	Improved	Unchanged	Worse
Heparin	8	5	3
No heparin	1	1	3

Table 4. NEUROLOGIC OUTCOME IN PATIENTS, STRATIFIED BY INJURY GRADE

	Neurologic Outcome			
Injury Grade	Normal	Mild Deficit	Severe Deficit	Dead
I	5 (31%)	3 (19%)	3 (19%)	5 (31%)
II	1 (20%)	3 (60%)	1 (20%)	0
III	3 (38%)	3 (38%)	1 (13%)	1 (13%)
IV	3 (33%)	3 (33%)	2 (22%)	1 (11%)

Table 5. NEUROLOGIC OUTCOME IN PATIENTS WITHOUT SIGNIFICANT CONFOUNDING INJURIES, STRATIFIED BY INJURY GRADE

	Neurologic Outcome			
Injury Grade	Normal	Mild Deficit	Severe Deficit	Dead
I	5 (63%)	1 (13%)	1 (13%)	1 (13%)
II	1 (33%)	2 (67%)	0	0
III	3 (75%)	0	0	1 (25%)
IV	3 (50%)	1 (17%)	1 (17%)	1 (17%)

Table 6. STROKE PATTERN ATTRIBUTED TO BLUNT VERTEBRAL ARTERIAL INJURY

BVI Grade	BCI Grade	Stroke Area	Time Postinjury	Treatment at Time of Stroke	Outcome
L I	R I	Brain stem	6 d	Heparin	Death
L I	R I, L I	Pons	18 h	None	Mild deficit
L I	—	Cerebellum	6 d	None	Severe deficit
L II	—	Cerebellum	8 h	None	Mild deficit
R II, L II	—	Cerebellum	7 d	Heparin	Mild deficit
L IV	—	Brain stem	9 d	None	Death
R IV, L IV	—	Occipital	2 d	Heparin	Mild deficit
R II, L III	—	Brain stem	12 d	None	Death
R IV	—	Cerebellum, medulla	7 d	None	Mild deficit

BCI, blunt carotid injury; BVI, blunt vertebral artery injury; d, days; h, hours.

criteria and have screened virtually all patients sustaining cervical vertebral fractures or distraction injuries. In a 28-month period, 12 (39%) of 31 patients with cervical spine injuries were found to have BVI by arteriography; further, extensive analysis of injury mechanisms and patterns revealed that cervical spine injuries were the only independent predictor of BVI.[36] The present series illustrates, however, that there is no specific cervical vertebral fracture pattern that stands out among the rest in association with BVI. Of the 27 patients with cervical spine injuries, only 6 (22%) had foramen transversarium fractures, and 5 (19%) each had subluxations and facet dislocations. In fact, had we adhered to the screening criteria of Willis et al, we would have missed 25 (66%) of the patients with BVI. We believe this justifies including all cervical spine injuries in a screening protocol.

Carrillo et al[37] asserted that blunt cerebrovascular injuries cannot be predicted based on clinical grounds, including injury mechanism or pattern. To an extent, the present series supports that premise. We noted no particular injury mechanism that is distinctly high risk (although being caught in a tornado seems to put one at a statistical disadvantage) (see Table 1). Internal carotid artery injuries seem to be associated primarily with cervical hyperextension. In contrast, Nibu et al[38] demonstrated in a cadaver study that lateral bending and axial rotation, but not flexion or extension, are associated with significant vertebral artery stretching. Whereas fibromuscular dysplasia is a risk factor for "spontaneous" dissections or injuries that follow trivial trauma,[39] only one patient in our series had the condition. Indeed, the diverse case reports cited[18–35] illustrate the difficulty in predicting BVI in every affected patient. However, to date we have been successful in identifying asymptomatic BVI as well as blunt carotid injury based on our inclusive set of screening criteria, and we know of no BVI missed by our protocol. Although our analysis of risk factors[36] revealed cervical spine injury to be the only independent risk factor for BVI, 11 (29%) of our patients did not have cervical spine injury, and 5 (13%) overall had neither signs or symptoms nor cervical spine injury. Therefore, we intend to continue screening using the same criteria. That said, it is important to identify the optimal diagnostic test for screening purposes.

Cerebral arteriography is the gold standard for the diagnosis of BVI and blunt carotid injury. Unfortunately, it is invasive and resource-intensive. Its risks include complications related to catheter insertion (1–2% hematoma, potential arterial pseudoaneurysm), contrast administration (1–2% renal dysfunction, potential allergic reaction), and stroke (<1%).[40] Duplex scanning has been proposed as the procedure of choice for imaging the cerebral vasculature, but there has been limited experience in imaging BVI. Sturzenegger et al[22] reported a diagnostic yield of 79% for any abnormalities on duplex scanning in cases of vertebral artery dissection and noted that there is no pathognomonic ultrasonographic finding. Duplex requires a stenosis of 60% or greater to detect flow disturbances and, thus, does not detect grade I or II injuries reliably; further, the bony vertebral foramina interfere with imaging of the complete vessel.

A modality recently promoted for blunt cervical artery injuries is computed tomographic angiography (CTA).[41] In their series, Rogers et al[41] reported five BVIs identified by CTA; however, the sensitivity of the test for BVI is unknown. In our experience, CTA has a sensitivity of 86% compared with arteriography in diagnosing blunt carotid injury.[42] Given the course of the vertebral artery, it is likely that bony artifact could obscure an injury. Despite its convenience (many of these patients will have alternative indications for CT scanning), CTA cannot be considered a reliable screening test at this time.

Of all the noninvasive screening modalities, MRA holds the greatest promise for supplanting cerebral arteriography. It has been studied by several groups and found to demonstrate injuries satisfactorily.[8,9,12] Although Levy et al[43] reported lower sensitivity for MRA and magnetic resonance imaging (20% and 60%, respectively) compared with angiography, subsequent technical refinements could improve this. Advantages of MRA include the capability to image the remainder of the head and neck simultaneously and

detect cerebral infarction earlier than CT scanning, while avoiding contrast.[44] Major impediments are a lack of timely availability at many institutions and the incompatibility of ventilatory and orthopedic fixation equipment with the magnet. A final decision on the utility of MRA awaits controlled comparisons with arteriography.

Although we have been successful in identifying a large number of asymptomatic BVIs, we have witnessed significant BVI-related complication and death rates. In six previous reports describing screening for BVI, a total of 55 patients were diagnosed with injuries.[8–10,12–14] Nine (16%) of the 55 patients had symptoms, with complete resolution in 6. The remaining three symptomatic patients had suffered strokes; two had gradual resolution of symptoms, and one died. Thus, the overall stroke rate was 5% and the death rate 2% in screening series.[8–10,12–14] Our patients fared worse: the stroke incidence was 24% and the overall death rate 18%, with a BVI-attributable death rate of 8%. There are several potential explanations for the worse outcome. First, we have an active trauma outreach program and a statewide trauma system; 42% of our patients with BVI were transferred from other institutions, including three patients after suffering stroke. Second, most of our patients suffered multisystem trauma, including one third with brain injury and one third with blunt carotid injury. It is likely the combination of injuries contributes to stroke or death, particularly in the presence of associated shock. Third, it is possible that BVI-attributable complications have been underestimated in previous studies: vertebrobasilar symptoms may be subtle or nonspecific and thus attributed to brain or spinal cord injury rather than BVI.[45] It is also possible that BVI is overlooked in patients who die suddenly after head or neck trauma. Potsch and Bohl[46] reported 15 such cases, emphasizing that standard autopsy techniques may miss BVI. Opeskin and Burke[47] similarly reported 25 autopsy cases of BVI and described special techniques used in the diagnosis.

Finally, our treatment may have resulted in complications. Indeed, two patients suffered hemorrhagic strokes while receiving anticoagulation to treat BVI. This latter fact should not dissuade clinicians from treating BVI with anticoagulation, however: our analysis suggests overall neurologic benefits associated with systemic heparin therapy.

An unexpected finding from our analysis was that neurologic complications were not related to injury grade. In our experience, the stroke incidence associated with BVI (24%) is no different than the stroke incidence we found in association with blunt carotid injury (21%).[17] In the case of blunt carotid injury, stroke incidence increases with injury grade,[17] but there is no such correlation with BVI injury grade (see Table 6). It has been speculated that nonocclusive injuries (grade I–III) are potentially more dangerous than grade IV injuries. This is presumably because, in general, collateral circulation (i.e., the contralateral vertebral artery) is sufficient to compensate for an interruption in vertebral artery flow, whereas a nonocclusive intimal injury may promote platelet thromboembolization and consequent infarction. However, in the case of blunt carotid injury, the stroke rate is higher in occlusive than nonocclusive injuries, presumably because the internal carotid circulation is less able to compensate for an interruption in flow.

Treatment of BVI remains controversial. In the past, general guidelines for the management of vertebral artery injuries have focused on controlling hemorrhage, arteriovenous fistulas, and pseudoaneurysms surgically or by interventional radiologic techniques (balloon occlusion or embolization), while observing occlusion, narrowing, or mild intimal irregularity.[1–7,11,12] Extrapolating such tenets from penetrating trauma is probably not appropriate; further, our data contest the notion that BVIs are innocuous injuries. Fabian et al[16] have demonstrated the efficacy of systemic heparin therapy in improving neurologic outcome in patients with blunt carotid injury, and we have applied this treatment to BVI. Although relatively small patient numbers limit the statistical power of our data, we find them compelling. Systemic heparin improves the neurologic outcome in the group as a whole and in those suffering stroke. In addition, it may prevent the arteriographic progression of lesions, protect against postinjury stroke, and prevent neurologic deterioration from treatment to discharge. However, systemic anticoagulation introduces risks of hemorrhagic complications, especially in multisystem trauma victims. Although we previously reported a 54% incidence of bleeding complications in a similar patient population,[15] we have reduced that to 10% in the present series by avoiding bolus heparin dosing and targeting a lower partial thromboplastin time.

Two recent papers[48,49] have challenged the use of systemic heparin in treating blunt cerebrovascular injuries, suggesting antiplatelet therapy or even observation as viable alternatives. However, their conclusions were based on retrospective analyses of small numbers of patients, without respect to injury grade. As an alternative to heparin, antiplatelet therapy makes intuitive sense given the presumed pathophysiology of BVI and the subsequent risk of thromboembolic stroke. In fact, we are enrolling patients in a prospective, randomized trial comparing heparin with antiplatelet therapy in the treatment of grade I injuries. However, we believe that an untreated control group would be inadvisable in light of the available data and therefore will use patients with absolute contraindications to anticoagulation as our controls.

There has been discussion of preemptive embolization of BVI as primary therapy. This is based on the premise that occlusion of one vertebral artery is generally well tolerated. The presence of vertebral artery agenesis has been found to be 1.8% to 3.1% in autopsy studies,[50] and unilateral vertebral artery dominance is uncommon: only two (5%) of our patients were found to have a dominant vertebral artery, and neither suffered stroke. Hoshino et al[51] performed unilateral vertebral artery ligation on 15 patients without adverse sequelae on long-term follow-up. However, routine embolization introduces the risk that the contralateral vertebral

artery may acquire transient flow disturbances, resulting in infarction. As noted above, axial rotation of the head may result in significant stretching of the vertebral artery and possible intimal injury.[38] In addition, extension-rotation of the head may result in decreased blood flow.[52] Hoshino et al cautioned that the contralateral vertebral artery should not be smaller than the ligated artery and that test occlusion should be performed before ligation to minimize the risk of infarction after vertebral artery ligation. We found no correlation between the size discrepancy of the vertebral arteries and stroke—in other words, in patients in whom the larger vertebral artery was injured, the stroke incidence was no higher than when the opposite was true. The fact that 88% of our patients with stroke had left vertebral artery injuries (compared with 44% having right vertebral artery injuries) is intriguing. Although it may well represent coincidence, Yi-Kai et al[52] found that right vertebral arteries had more insufficiency of blood supply than left vertebral arteries, potentially making them less able to compensate for left-sided injuries.

In sum, we have confirmed that BVI is not a rare event and may be diagnosed in an asymptomatic phase through screening. Screening, however, must include more than just patients with cervical spine injuries. To identify injuries after apparently trivial trauma, unilateral headache or posterior neck pain, particularly if sudden, sharp, severe, and unlike previous pain, must be considered potential signs of BVI.[53] Arteriography is the gold standard for diagnosis, but MRA may become an acceptable alternative. Adverse clinical outcomes are more common than previously reported, but the optimal treatment remains to be established. Our experience suggests that anticoagulation improves the neurologic outcome.

References

1. Blickenstaff KL, Weaver FA, Yellin AE, et al. Trends in the management of traumatic vertebral artery injuries. Am J Surg 1989; 158:101–106.
2. Demetriades D, Theodorou D, Asensio J, et al. Management options in vertebral artery injuries. Br J Surg 1996; 83:83–86.
3. Golueke P, Sclafani S, Phillips T, et al. Vertebral artery injury: diagnosis and management. J Trauma 1987; 27:856–865.
4. Landreneau RJ, Weigelt JA, Megison SM, et al. Combined carotid-vertebral arterial trauma. Arch Surg 1992; 127:301–304.
5. Meier DE, Brink BE, Fry WJ. Vertebral artery trauma: acute recognition and treatment. Arch Surg 1981; 116:236–239.
6. Reid JDS, Weigelt JA. Forty-three cases of vertebral artery trauma. J Trauma 1988; 28:1007–1012.
7. Yee LF, Olcott EW, Knudson MM, Lim RC Jr. Extraluminal, transluminal, and observational treatment for vertebral artery injuries. J Trauma 1995; 39:480–486.
8. Friedman D, Flanders A, Thomas C, Millar W. Vertebral artery injury after acute cervical spine trauma: rate of occurrence as detected by MR angiography and assessment of clinical consequences. AJR 1995; 164:443–447.
9. Giacobetti FB, Vaccaro AR, Bos-Giacobetti MA, et al. Vertebral artery occlusion associated with cervical spine trauma: a prospective analysis. Spine 1997; 22:188–192.
10. Louw JA, Mafoyane NA, Small B, Neser CP. Occlusion of the vertebral artery in cervical spine dislocations. J Bone Joint Surg [Br] 1990; 72:679–681.
11. Parent AD, Harkey HL, Touchstone DA, et al. Lateral cervical spine dislocation and vertebral artery injury. Neurosurgery 1992; 31:501–509.
12. Weller SJ, Rossitch E Jr, Malek AM. Detection of vertebral artery injury after cervical spine trauma using magnetic resonance angiography. J Trauma 1999; 46:660–666.
13. Willis BK, Greiner F, Orrison WW, Benzel EC. The incidence of vertebral artery injury after midcervical spine fracture or subluxation. Neurosurgery 1994; 34:435–442.
14. Woodring JH, Lee C, Duncan V. Transverse process fractures of the cervical vertebrae: are they insignificant? J Trauma 1993; 34:797–802.
15. Biffl WL, Moore EE, Ryu RK, et al. The unrecognized epidemic of blunt carotid arterial injuries: early diagnosis improves neurologic outcome. Ann Surg 1998; 228:462–470.
16. Fabian TC, Patton JH Jr, Croce MA, et al. Blunt carotid injury: importance of early diagnosis and anticoagulant therapy. Ann Surg 1996; 223:513–525.
17. Biffl WL, Moore EE, Offner PJ, et al. Blunt carotid arterial injuries: implications of a new grading scale. J Trauma 1999; 47:845–853.
18. Stahmer SA, Raps EC, Mines DI. Carotid and vertebral artery dissections. Emerg Med Clin North Am 1997; 15:677–698.
19. Plant JR, Butt JC. Laceration of vertebral artery. An historic boxing death. Am J Forensic Med Pathol 1993; 14:61–64.
20. Garg BP, Ottinger CJ, Smith RR, Fishman MA. Strokes in children due to vertebral artery trauma. Neurology 1993; 43:2555–2558.
21. Fletcher J, Davies PT, Lewis T, Campbell MJ. Traumatic carotid and vertebral artery dissection in a professional jockey: a cautionary tale. Br J Sports Med 1995; 29:143–144.
22. Sturzenegger M, Mattle HP, Rivoir A, et al. Ultrasound findings in spontaneous extracranial vertebral artery dissection. Stroke 1993; 24: 1910–1921.
23. Lannuzel A, Moulin T, Amsallem D, et al. Vertebral-artery dissection following a judo session: a case report. Neuropediatrics 1994; 25:106–108.
24. Provenzale JM, Morgenlander JC, Gress D. Spontaneous vertebral dissection: clinical, conventional angiographic, CT, and MR findings. J Comput Assist Tomogr 1996; 20:185–193.
25. Tramo MJ, Hainline B, Petito F, et al. Vertebral artery injury and cerebellar stroke while swimming: case report. Stroke 1985; 16:1039–1042.
26. DeBehnke DJ, Brady W. Vertebral artery dissection due to minor neck trauma. J Emerg Med 1994; 12:27–31.
27. Rogers L, Sweeney PJ. Stroke: a neurologic complication of wrestling. A case of brainstem stroke in a 17-year-old athlete. Am J Sports Med 1979; 7:352–354.
28. Miller SJ, Copass M, Johansen K, Winn HR. Stroke following Rottweiler attack. Ann Emerg Med 1993; 22:262–264.
29. Lee KP, Carlini WG, McCormick GF, Albers GW. Neurologic complications following chiropractic manipulation: a survey of California neurologists. Neurology 1995; 45:1213–1215.
30. Herr RD, Call G, Banks D. Vertebral artery dissection from neck flexion during paroxysmal coughing. Ann Emerg Med 1992; 21:88–91.
31. Egnor MR, Page LK, David C. Vertebral artery aneurysm: a unique hazard of head banging by heavy metal rockers. Case report. Pediatr Neurosurg 1991–92; 17:135–138.
32. Biousse V, Chabriat H, Amarenco P, Bousser MG. Roller-coaster–induced vertebral artery dissection. Lancet 1995; 346:767.
33. Dragon R, Saranchak H, Lakin P, Strauch G. Blunt injuries to the carotid and vertebral arteries. Am J Surg 1981; 141:497–500.
34. Young CA, Chadwick DW, Humphrey PR. Extracranial vertebral artery dissection following tonic-clonic seizure. J Neurol Neurosurg Psychiatry 1991; 54:365–366.
35. Russell WR. Yoga and vertebral arteries. Br Med J 1972; 1:685.

36. Biffl WL, Moore EE, Offner PJ, et al. Optimizing screening for blunt cerebrovascular injuries. Am J Surg 1999; 178:517–522.
37. Carrillo EH, Osborne DL, Spain DA, et al. Blunt carotid artery injuries: difficulties with the diagnosis prior to neurologic event. J Trauma 1999; 46:1120–1125.
38. Nibu K, Cholewicki J, Panjabi MM, et al. Dynamic elongation of the vertebral artery during an in vitro whiplash simulation. Eur Spine J 1997; 6:286–289.
39. Eachempati SR, Sebastian MW, Reed RL II. Posttraumatic bilateral carotid artery and right vertebral artery dissections in a patient with fibromuscular dysplasia: case report and review of the literature. J Trauma 1998; 44:406–409.
40. Biffl WL, Moore EE, Elliott JP, et al. Blunt cerebrovascular injuries. Curr Prob Surg 1999; 36:507–599.
41. Rogers FB, Baker EF, Osler TM, et al. Computed tomographic angiography as a screening modality for blunt cervical arterial injuries: preliminary results. J Trauma 1999; 46:380–385.
42. Biffl WL, Moore EE, Mestek M. Computed tomographic angiography as a screening modality for blunt cervical arterial injuries: a cautionary note [letter]. J Trauma 1999; 47:438–439.
43. Levy C, Laissy JP, Raveau V, et al. Carotid and vertebral artery dissections: three-dimensional time-of-flight MR angiography and MR imaging versus conventional angiography. Radiology 1994; 190:97–103.
44. Moseley ME, Cohen Y, Mintorovitch J, et al. Early detection of regional cerebral ischemia in cats: comparison of diffusion- and T2-weighted MRI and spectroscopy. Magn Reson Med 1990; 14:330–346.
45. Cohen SM. Clinical manifestations and evaluation of patients with ischemic cerebrovascular disease. In: Rutherford RB, ed. Vascular Surgery, 4th ed. Philadelphia: WB Saunders; 1995:1474–1480.
46. Potsch L, Bohl J. Traumatic lesion of the extracranial vertebral artery: a noteworthy potentially lethal injury. Int J Legal Med 1994; 107:99–107.
47. Opeskin K, Burke MP. Vertebral artery trauma. Am J Forensic Med Pathol 1998; 19:206–217.
48. Eachempati SR, Vaslef SN, Sebastian MW, Reed RL II. Blunt vascular injuries of the head and neck: is heparinization necessary? J Trauma 1998; 45:997–1004.
49. Colella JJ, Diamond DL. Blunt carotid injury: reassessing the role of anticoagulation. Am Surg 1996; 62:212–217.
50. Thomas GI, Anderson KN, Hain RF, et al. The significance of anomalous vertebral-basilar artery communications in operations on the heart and great vessels. Surgery 1959; 46:747–757.
51. Hoshino Y, Kurokawa T, Nakamura K, et al. A report on the safety of unilateral vertebral artery ligation during cervical spine surgery. Spine 1996; 21:1454–1457.
52. Yi-Kai L, Yun-Kun Z, Cai-Mo L, Shi-Zhen Z. Changes and implications of blood flow velocity of the vertebral artery during rotation and extension of the head. J Manipulative Physiol Ther 1999; 22:91–95.
53. Sturzenegger M. Headache and neck pain: the warning symptoms of vertebral artery dissection. Headache 1994; 34:187–193.

Discussion

DR. TIMOTHY C. FABIAN (Memphis, Tennessee): This is truly an exceptional contribution to the surgical literature. I am somewhat envious in that for the last few years we have been considering such a study, but have not undertaken it. Perhaps, however, we are somewhat lucky since the Denver group has provided outstanding direction for future retrospective as well as prospective evaluations of this interesting lesion.

The fact that 40% of cervical spine injuries have blunt vertebral artery injury is absolutely astounding. In my critical analysis of the manuscript, I could find no criticism. I will ask but a few questions:

First, would you tell us your anticoagulation protocol with heparin relative to dosage and duration? And do you continue with Coumadin or antiplatelet therapy following discharge, and for how long?

Two, considering your very aggressive approach to screening angiography, what is your complication rate? In the past year we had a patient with screening cerebral angiography that was normal, but who sustained a vertebral dissection which resulted in a major cerebellar stroke with catastrophic outcome.

Three, what is the rate of yield for cerebral vascular injury studies, i.e., how many angiograms are performed for positive study?

Four, what was the number of blunt carotid injuries over this same time interval, so we can have a relative incidence of the two disease processes?

Finally, could you speculate a little more on the adequacy of CT and MR angiography for cerebral vascular diagnostics for trauma?

DR. J. DAVID RICHARDSON (Louisville, Kentucky): I queried our trauma registry for blunt vertebral artery injuries, and found six patients over a 9-year period. We probably actually had more blunt trauma admissions than the authors did in that period of time, and so, trying to look at what the difference is, I think it is possible that we weren't looking for them with the kind of aggressive protocol described today.

We have been had an interest in blunt carotid artery injuries, but have not identified a group at major risk for them. We see injuries that are devastating in patients who have no external signs. We have other patients who have garroting injuries or bruises of the neck with no carotid injury. We would appreciate any other information you have on who you screen.

The second major point that I think the authors made very well is that vertebral injuries are not innocuous in their experience. They had nine patients with. We certainly have not identified anything like that proportion of patients with posterior circulation strokes. So again, whether that is purely and simply a recognition problem or a different experience, I think, is a little bit hard to know.

I have always been bothered a little bit by the heparin recommendation. We have certainly used it for carotid injuries. Some patients clearly seem to benefit from it. But we do find that the patients we cannot heparinize for other reasons, such as coexisting head injuries, seem to do worse. Why does heparin work, and do you use it for all injuries?

DR. GEORGE M. WATKINS (Tampa, Florida): The Immitron-like superfast CAT scans have been very efficacious in quickly and noninvasively diagnosing chronic vertebral artery disease. Do you have such available in Denver, and have you used such or do you now plan to, since you show that it is very important to diagnosis?

And second, vitamin E has been shown to cause regression of acquired coronary artery disease—low dose vitamin E, 200 mg a day. Do you give this routinely to your patients who have an injury and have stenosis, or just as routine?

DR. WALTER L. BIFFL (Closing Discussion): Our anticoagulation protocol has evolved over time. Presently, we do not bolus patients with heparin. We use systemic unfractionated heparin at 15 units per kilogram per hour to target a PTT of 40 to 50 seconds. By

evolving to this, we have reduced our anticoagulation-related complications from over 50% to under 10%.

The complication rates associated with arteriography: we have performed between 350 and 400 screening arteriograms over the last 3½ to 4 years. We did an interim analysis after 250 and at that point had had two strokes, but haven't had any more, so—two strokes out of 350 to 400 arteriograms. Both of them involved manipulation of catheters within the injured vessel lumina. Both were in the carotid circulation. Unfortunately, one was also in a lawyer.

So we have taken from that a couple of messages. One is to stay at the origin of the vessel if at all possible and not try to do anything with intravascular ultrasound. And if stents are considered, not to try to place them until that injury has matured a little bit.

We also had three puncture site hematomas, but no arterial pseudoaneurysms.

There are fewer medical risks of renal toxicity, we have not realized.

The yield, I think I mentioned we have had about 350 angios to get these 38 vertebral injuries. Over the same period of time, the incidence of carotid injuries has been almost exactly twice that of vertebral injuries. We have used arteriography because one thing we have not had access to is magnetic resonance angiography. We have just acquired a scanner and will start a prospective analysis comparing the two. We did look at duplex scanning in a previous multicenter trial, found that it missed some high carotid lesions, suspect it to probably miss more vertebral lesions, and so we have not looked at that as a screening study.

CT-angio was looked at prospectively at our center some time ago. We found also sensitivity about 86% with that, and continue to use arteriography, as we are most comfortable that that's going to pick up even the most minor injuries.

Dr. Richardson, we agree that the patients at risk are hard to recognize. We analyzed our data about a year ago to look at independent predictors of carotid and vertebral injuries. Out of an analysis of 249 patients, we found that the only independent predictor of vertebral injuries was a cervical spine fracture. For carotid injuries, again, to get into the study a patient had to have the injury mechanism that I described, a hyperextension or near hanging or direct cervical blow. We found four independent predictors, and that was a patient coming in with a Glasgow score of 6 or less associated with those mechanisms, diffuse axonal brain injury, petrous bone fractures, and Le Fort II or III fractures. Those seem to be high-risk predictors, but 20% of the patients had none of those. So we are still continuing the screening with the entire criteria.

The strokes in this series: nine patients had stroke, and three were on heparin at the time and still sustained stroke. Six others were not. Three got heparin after their stroke, and only one of the six who got heparin had a poor outcome.

How does heparin work? We wish we knew that. We assume that it's preventing the further aggregation of platelets at the injury site. Virtually any intimal injury is going to cause platelet aggregation. We were able to get autopsy data on a limited number of patients whom we had studied. In particular, there were three patients who had grade I injuries by arteriogram, but all of them had either intimal disruption or thrombosis of the vessel at the time of autopsy. So either the arteriograms are undercalling them or there is occult progression of the injuries between the time of diagnosis and either event or death.

I have mentioned CT scanning. It sounds like we don't have as nice a CT scanner as you, Dr. Watkins, but we are going to be studying MRA. We think that that's the study that's going to potentially take over from arteriography. Finally, vitamin E—that's an interesting idea. We have not tried it, but we might.