Contemporary outcomes of vertebral artery injury

Daniel M. Alterman, MD, Richard E. Heidel, PhD, Brian J. Daley, MD, Oscar H. Grandas, MD, Scott L. Stevens, MD, Mitchell H. Goldman, MD, and **Michael B. Freeman, MD,** *Knoxville, Tenn*

Objective: **Vertebral artery injury (VAI) associated with cervical trauma is being increasingly recognized with more aggressive screening. Disparate results from previous literature have led to uncertainty of the significance, natural history, and optimal therapy for VAI.**

Methods: **To understand the natural history and treatment outcomes from our experience, we performed a retrospective, single-center review from a level I trauma center for the previous 10 years of all VAI. Injuries were identified from search of an administrative trauma database, a resident-run working database, and all radiology dictations for the same period. All VAI were classified according to segmental involvement, Denver grading scale, and laterality. Analysis of associated injuries, demographics, neurologic outcome, mortality, length of stay, treatment plan, and follow-up imaging was also performed.**

Results: **Fifty-one patients with VAI were identified from 2001 to 2011 from a total of 36,942 trauma admissions (0.13% incidence). Associated injuries were significant with an average New Injury Severity Score of 29.6. Penetrating trauma occurred in 14%. Cervical spine fracture was present in 88% with VAI. Diagnosis was obtained with computed tomographic angiography (CTA) in 95%. Screening was prompted by injury pattern or high-risk mechanism in all cases. Injuries classified according to the Denver grading scale were grade I = 24%, grade II = 35%, grade III = 4%, grade IV = 35%, and grade V = 2%. Distribution across segments included V1 = 18%, V2 = 67%, V3 = 31%, and V4 = 6%. Only one posterior circulation stroke was attributable to VAI. Overall mortality was 8%, with each mortality being associated with significant other organ injuries. Treatment rendered for VAI was antiplatelet therapy (50%), observation (31%), warfarin (17%), and stent (2%). There were no significant differences between treatment groups on any variable with the exception of body mass index (*P* = .047). Follow-up was obtained for 13% (n = 6) of survivors. The CTA demonstrated injury stability in four patients and resolution in two patients. Accuracy of the administrative trauma database was 53% compared with 96% for the resident-run working database.**

Conclusions: **Neurologic sequelae attributable to VAI were rare. Grade of VAI or vertebral artery segment did not correlate with morbidity. We did not observe any differences in short-term outcomes between systemic anticoagulation and antiplatelet therapy. Of those patients seen at follow-up, injury resolution or stability was documented by CTA. A conservative approach with either observation or antithrombotic therapy is suggested. If the natural history of VAI includes a very low stroke rate, then therapies with a lower therapeutic index, such as systemic anticoagulation, in the severely injured trauma patient are not supported. Our search strategy urges awareness of the limitations of administrative databases for retrospective vascular study. (J Vasc Surg 2013;57:741-6.)**

Vertebral artery injury (VAI) is a rare entity. Screening for VAI after blunt trauma has yielded an incidence of 0.24% to 2%.[1-4] When more specific populations are screened for VAI (head injury or blunt cervical spine trauma), up to 20% are found to have associated VAI.[3] The incidence and natural history of morbidity attributable to VAI are unclear. There is a wide range of reported stroke and death rate after VAI, with published reports disparate in their screening and diagnostic criteria.[3-7] Sequelae of VAI include posterior cerebral circulation stroke and death.[8,9] The options for treatment of VAI are observation, antiplatelet therapy, anticoagulation, endovascular therapy, surgical repair, or various combinations of these methods. The consensus of the present heterogeneous literature is that symptomatic blunt VAI should be treated with systemic anticoagulation, provided there is no contraindication to the proposed therapy.[3] Asymptomatic blunt VAI can be managed with either anticoagulation or antiplatelet therapy.[3] The role of endovascular therapy, such as stent placement, is evolving but may be indicated for enlarging pseudoaneurysms or symptomatic accessible dissection.[8] With reports on VAI containing small patient volumes, these recommendations have been extrapolated in part from the literature on blunt carotid artery injury. At this time, there is no level 1 or 2 evidence available to guide the management of various grades of VAI. Equally vexing is the uncertainty concerning targeted screening guidelines for VAI as well as the optimal method for imaging and the accuracy of these examinations. Most centers use some derivation of the Denver, Memphis, or Biffl modified criteria to prompt targeted screening for VAI. As various centers have applied more aggressive

From the Department of Surgery, Graduate School of Medicine University of Tennessee.

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Reprint requests: Daniel M. Alterman, MD, Department of Surgery, Graduate School of Medicine University of Tennessee, 1924 Alcoa Highway, Box U-11, Knoxville, TN 37920-6999 (e-mail: dalterma@mc.utmck.edu).

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screening and utilized computed tomographic angiography (CTA), there has been increased recognition of VAI. This presents a clinical conundrum because the optimal management of the varied grades of VAI is at best unclear, the clinical sequelae are rare, and the proposed therapies have low therapeutic indices in this population. The purpose of this study was to analyze our experience with VAI over a 10-year period in a level I trauma center. We sought to better understand the natural history and treatment outcomes of VAI from our experience.

METHODS

After approval was obtained from the Institutional Review Board, a retrospective 10-year search (2001-2011) of all trauma admissions to a level I trauma center was performed for VAI using the *International Classification of Diseases, Ninth Revision* codes in the institutional administrative trauma database. The search strategy was then implemented by querying the radiology dictation database, which searches that same time period for the key words "vertebral artery dissection" or "vertebral artery injury." The results of the two queries were compared. The results of the search were validated in a resident-run database of trauma inpatients maintained since 2008. We compared the accuracy of these three different search strategies. Study demographic, diagnostic, therapeutic, and outcome data were obtained via our institution's administrative and clinical database, chart review, and imaging review. Data points collected included basic demographics, body mass index (BMI), mechanism of injury, New Injury Severity Score, survival, admission Glasgow Coma Scale (GCS) score, presence of associated vertebral column, brain or carotid artery injury, length of stay, discharge disposition, and treatment rendered. VAI was classified by segment location (V1-V4 segments), laterality, and Denver radiologic grading scale.[7] The Denver radiologic grading scale of blunt cerebrovascular injury is composed of five grades. Grade I is irregularity of the vessel wall or dissection with <25% stenosis. Grade II is irregularity of vessel wall or dissection with ≥25% stenosis. Grade III represents pseudoaneurysm. Grade IV indicates vessel occlusion. Grade V indicates vessel transection. The practice at our institution is to obtain CTA of the neck to screen for VAI based upon the Denver screening criteria.[3,5] Over the 10-year study period, our facility upgraded to 64-row multidetector CT technology (Lightspeed VCT; GE Medical Systems, Milwaukee, Wisc) with advanced reformatting software. Specific documentation of the indication for VAI screening was not available and was left to the discretion of the treating surgeon. During the study time period, our institution's practice had been to screen for cervical vascular injury by the Denver criteria. Treatment modality was classified as observation, antiplatelet therapy, systemic anticoagulation, surgical intervention, or endovascular intervention. Neurologic outcome was classified by the presence of stroke on imaging, spinal cord injury, and cognitive function by the Rancho Los Amigos scale. The Rancho Los Amigos scale is a clinical scoring tool that indicates cognitive function on a scale from 1 to 10, with 10 being independent and purposeful and 1 having no response to stimuli. Hospital and intensive care unit (ICU) length of stays and discharge destination (either home or long-term care facility) were recorded. Stroke was defined as a focal neurologic deficit lasting more than 24 hours with an associated lesion detected on CT or magnetic resonance imaging. After identification of VAI, inpatient repeat neurologic imaging was not obtained unless the patient had signs or symptoms of stroke. Consultation with the neurology department was obtained only for those patients who were subsequently diagnosed with stroke.

Of the patients who returned for follow-up, imaging was analyzed for resolution, stability, or worsening of VAI. We compared baseline characteristics and outcome data for active treatment vs observation, anticoagulation vs antiplatelet therapies, presenting GCS 3-5 vs GCS 6-15, and Denver injury grade I-III vs grade IV-V. Descriptive and frequency statistics were run on each continuous and categorical variable. Skewness and kurtosis statistics were run on each continuous variable to meet the assumption of normality for each analysis. Any continuous variable that had a skewness or kurtosis statistic above an absolute value of 2.0 was considered to not have a normal distribution. The Levene test was used to meet the assumption of homogeneity of variance. Any analysis with $P < .05$ was considered to have violated this assumption. Independent samples *t*-tests were used to compare treated vs nontreated patient groups, patients treated with anticoagulation vs antiplatelet drugs, patients with GCS score between 3 and 5 vs those with GCS score between 6 and 15, and patients with Denver grade between I and III vs those with Denver grade between IV and V. When any violation of a statistical assumption occurred, a nonparametric Mann-Whitney *U*-test was used. The χ^2 tests were used to compare groups on categorical variables, and unadjusted odds ratios were calculated for any significant findings. All analyses were conducted using SPSS version 19 (IBM, Chicago, Ill), and statistical significance was assumed at $P < .05$.

RESULTS

For the 10-year period from 2001 to 2011, there were 36,942 trauma admissions to the University of Tennessee Medical Center in Knoxville, a level I trauma center. The combined results of our different search strategies yielded 51 patients with VAI. Characteristics of this cohort are given in Table I. The distribution of injuries across vertebral artery segments V1-V4 is shown in the Fig. The distribution for VAI injury grade according to the Denver radiologic scale was grade I (n = 12), grade II (n = 18), grade III (n = 2), grade IV (n = 18), and grade V (n = 1). Four deaths were attributed to associated injuries other than VAI. One isolated posterior circulation stroke occurred and was attributable to VAI, for a stroke rate of 1.9%. This occurred in a 29-year-old man with a blunt injury and no associated vertebral column or carotid artery injury. The injury involved segments V2 and V3. He was

Table I. Patient background characteristics (N = 51)

	Mean	*Standard deviation*
Age, years	41.8	17.4
Body mass index	26.8	6.5
Penetrating mechanism	13.7%	—
GCS score	11.6	5.1
New Injury Severity Score	25.4	14.4
Male sex	78%	—
Cervical spine fracture	88%	—
Single-level cervical spine	29%	—
Traumatic brain injury	22%	—
Carotid artery injury	9.8%	—
Rancho Los Amigos score	7.4	1.3
Spinal cord injury	26%	—
ICU length of stay, days	14.9	21.9
Hospital length of stay, days	23	34
Discharge to long-term facility	41%	—

GCS, Glasgow Coma Scale; *ICU,* intensive care unit.

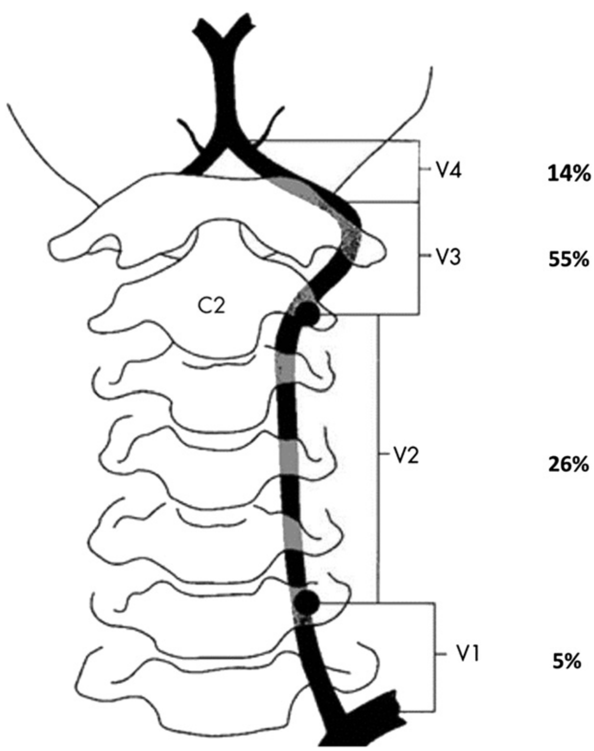

Fig. Distribution of vertebral artery segment injuries.

treated with systemic anticoagulation therapy and discharged home after a hospital stay of 12 days with improving neurologic function. Of the 51 VAIs, 26 occurred on the right, one bilaterally, and 24 on the left. The only posterior circulation stroke occurred with an injury of the right vertebral artery.

Treatments were antiplatelet therapy (n = 26), observation (n = 15), systemic anticoagulation (n = 9), and stent placement (n = 1). Skewness and kurtosis statistics found nonnormal distributions for the Rancho Los Amigos score at discharge, hospital length of stay, and ICU length of stay variables, so nonparametric tests were used for those analyses. Univariate comparison of the 15 patients with VAI who were observed vs the 36 who were not demonstrated a significant difference in BMI. Patients with an injury and a lower BMI tended to be observed more often with a threshold between 24 and 28 BMI.

Table II. Comparison of patients with vertebral artery injury based upon admission GSC score

	GCS 3-5 (N = 13)	*GCS 6-15 (N = 38)*
Age, years	38.3 (13.8)	43 (18.7)
Body mass index	28 (7.2)	26.3 (6.3)
New Injury Severity Score[a]	37 (13.1)	21.5 (12.8)
Denver radiologic grade	2.7 (1.2)	2.5 (1.3)
Rancho Los Amigos Score[b]	6.5 (2.1)	7.8 (0.7)
ICU length of stay, days[b]	25.6 (29)	10 (17)
Hospital length of stay, days[b]	33 (27)	19.5 (36)

GCS, Glasgow Coma Scale; *ICU,* intensive care unit.
Values are given as mean (standard deviation).
[a]$P < .001$.
[b]$P < .05$.

Antiplatelet therapy for VAI was either aspirin or clopidogrel and rarely with dual therapy. Systemic anticoagulation consisted of unfractionated heparin infusion or therapeutic dalteparin injections, followed by 3 to 6 months of warfarin therapy targeted to maintain an international normalized ratio of 2 to 3. No significant differences were found between patients treated with anticoagulation drugs vs antiplatelet medications. In the one patient treated with endovascular techniques, a self-expanding stent was placed for management of vertebral artery dissection. No significant differences were found between patients with less severe Denver scores (I-III) vs more severe (IV-V). Comparison of those presenting with GCS 3-5 vs GCS 6-15 are given in Table II. Significant differences were found between the GCS groups, with patients in the GCS 3-5 range having higher New Injury Severity Scores ($P <$ .001), higher Denver grades ($P = .008$), and longer stays in the hospital ($P = .03$) and the ICU ($P = .021$). Patients in the GCS 3-5 group were also 7.31 times more likely (95% confidence interval [CI], 1.61-33.22) to have a penetrating mechanism of injury vs a blunt mechanism, 5.12 times more likely (95% CI, 1.78-14.69) to have an associated traumatic brain injury, 11.69 times more likely (95% CI, 1.43-95.37) to have an associated carotid injury, and 2.19 times more likely (95% CI, 1.21-3.96) to be discharged to a long-term care facility. Spinal cord injury was present in 13 patients, of whom 11 had quadriplegia. There were no significant relationships between the frequency of spinal cord injury and VAI grade or treatment strategy. Associated carotid artery injury was found in five patients, representing 10% of the study population, all of whom had penetrating injury by gunshot wound. These five patients with a penetrating mechanism had VAI Denver injury grades II, III, III, IV, and V. Of the five carotid artery injuries, one had operative repair, one had a covered stent for distal internal carotid artery injury, and the other three were treated with antiplatelet therapy and observation.

Follow-up imaging was available for six of the 47 survivors and was completed between 2 and 5 months after discharge. Of these six follow-up imaging studies, one showed resolution of the injury (Denver grade II injury across segments V1-V3 treated with 3 months of warfarin without any neurologic sequelae), and the other five demonstrated stability of the injury. The Denver grades of the five stable injuries were grade I (n = 3), grade II, and grade III. Whether follow-up imaging was obtained at alternative sites is not known.

Among the three different databases used to identify patients with VAI, the resident-run database had the highest percentage of agreement with the other two databases for the same period. Search of the radiology database for the term "vertebral artery injury" found 88 occurrences, of which 15 were injuries. Search for the term "vertebral artery dissection" found 52, of which 13 were injuries. The original search output from the administrative trauma database yielded 48 patients, with only 27 documented VAIs. The overall accuracy of this database was 53%. Comparatively, the resident-run database was more accurate for the same time period, identifying 28 of 29 VAIs from the time period from 2008 to 2011.

DISCUSSION

The current consensus for management of blunt VAI suggests an aggressive approach with antithrombotic therapy to prevent neurologic sequelae in the posterior cerebral circulation. There is wide agreement that high-grade symptomatic VAI should be treated.[3] Anticoagulation has been the most commonly used treatment method for high-grade symptomatic VAI with a blunt mechanism, but a growing number of reports have used endovascular management.[8] When anticoagulation is contraindicated, largely due to associated injuries, antiplatelet therapy has been recommended.[3] The advantage of unfractionated heparin infusion in the early phase after injury is the potential to reverse its effects more rapidly compared with antiplatelet agents. Similar to previous series, we found VAI to be rare, with an incidence of 0.13% (51 injuries identified over 36,942 trauma admissions). In contrast to previously reported stroke rates, we found only one documented posterior cerebral infarction in our cohort. As in previous series, the Denver grade of VAI was not associated with the degree of morbidity.[5] No differences were found in patient characteristics when controlling for treatment strategy, outcome data, or Denver grade, with the exception of a slightly lower BMI associated with observation. When controlling for GCS severity, we met the expectations of more severe injury scores having longer hospital lengths of stay. Of note, the VAI grade was not significantly different for GCS <6. This was surprising because this is part of the widely accepted screening criteria. With a variety of alternative and more frequent diagnoses impacting the GCS, we would call this parameter into question as an indicator for VAI screening. Although follow-up was limited, patients who were reimaged demonstrated either healing or stability of the VAI with no detrimental changes in neurologic function noted.

Stroke rates of approximately 20% have been reported for varying grades of VAI.[7] However, Miller et al[4] reported an overall stroke rate of 2.6% among 64 patients with VAI. It is important to note that half of these reported strokes had no VAI-specific therapy and consisted of only four patients.[4] A previous series reported by Biffl et al[5] noted no correlation between VAI radiographic injury grade and outcome. Previous series have reported higher rates of stroke (5%-24%) associated with VAI; however, several of them included patients with associated traumatic brain injury and carotid artery injury.[5,7,10] Often, it is not clear whether associated strokes are in the posterior circulation distribution or attributable to another concurrent mechanism of injury. Our study suggests a low incidence of stroke with VAI. Individual stroke risk from VAI may be better assessed by evaluating the flow of the contralateral vertebral artery, patency of the circle of Willis, and the carotid system. Unilateral vertebral artery hypoplasia is found in up to 10% of patients and may influence treatment decisions.[3] The left vertebral artery is dominant in 50% and the right is dominant in approximately 25%. The remaining 25% have vertebral arteries of similar caliber.[11] In healthy subjects, the vertebral artery may occupy between 8% and 85% of the transverse foramen.[12] These congenital and anatomic differences in the native vessels may create diagnostic uncertainty when presented with an associated cervical spine fracture because a hypoplastic vertebral artery may be mistaken for a dissection. It also can be hypothesized that any injury to the dominant vertebral artery is more likely to result in stroke. This result was noted by Biffl et al,[5] with 88% of posterior circulation ischemic events occurring in patients with a left VAI. Our series of VAI was divided equally between left and right, with the only posterior circulation event occurring with a right VAI.

Many questions remain concerning VAI, including optimal screening criteria for VAI as well as the optimal imaging study. In our series, VAI was diagnosed in 92% of patients using CTA as the sole diagnostic modality. Our study suggests that CTA can identify patients with VAI. Screening with CTA is able to rapidly identify those with VAI, but vertebral artery dominance or hypoplasia may influence its sensitivity. When is reimaging indicated to assess injury healing, and how should the information obtained from reimaging guide further management? We were unable to address this due to low follow-up. The optimal treatment of asymptomatic VAI is unclear at this time. The critical question that is difficult to adequately answer from the current literature relates to the risk of stroke from VAI. This is difficult to address given the wide range of reported morbidity and mortality associated with different grades and mechanisms of VAI. Due in part to the rarity of VAI, no randomized prospective trials have addressed these questions. Most evidence guiding the management of VAI is class 3 data based upon case series from single institutions. Spontaneous vertebral artery dissection is a well-described cause of stroke in young people and currently is managed with anticoagulation. Our series demonstrated equivalent short-term neurologic outcomes between treatment with systemic anticoagulation or antiplatelet therapy.

The questions concerning optimal imaging, treatment modality, and stroke risk assessment cannot be answered from our data because the data were collected in retrospective fashion from a small cohort. Several limitations exist in our data. Our series is retrospective and from a single institution, follow-up is limited, and there were a small number of injuries. A low follow-up rate makes drawing conclusions about neurologic outcomes, whether clinical or radiographic, difficult. The study population was largely composed of young injured patients who have a historically low rate of outpatient follow-up. The rarity of this injury and the low follow-up rate of this population have relegated the management of VAI to class 3 or 4 level of evidence. Understanding the decision process that guided selection of a particular treatment strategy was hampered by the retrospective nature of the review.

Use of administrative databases for retrospective research has many known limitations.[13] Database research for abdominal aortic aneurysm treatment has reported coding accuracies between 52% and 97%.[14] We found an accuracy of 53% for the administrative trauma database with respect to VAI compared with electronic search of radiology dictations and a resident-run working database for similar time periods. It is possible that our search terms were not sensitive to alternative diagnostic labels, causing us to miss those reports. It is possible that VAI is not identified if a single search modality is used; we used three search strategies to reduce this potential error. Our data urge caution regarding reliance on administrative databases for vascular surgery research. Correlating these types of databases with other local search strategies, such as our use of the radiology dictation system or a resident-run database, was useful.

We noted a very low incidence of VAI and found our neurologic outcomes were unaffected by the grade of VAI. The vertebral artery segment involved and the grade of injury did not seem to influence outcome. Our outcomes with treatments of observation, antiplatelet agents, or anticoagulation were similar, but this conclusion is limited by a small heterogeneous retrospective cohort. The only patient with a posterior circulation event in our series presented with signs of stroke that prompted immediate imaging and diagnosis. From our experience, VAI may have a more benign natural history than previously suggested, particularly with a blunt intimal injury, and may support a less aggressive treatment strategy. This conclusion is based upon a cohort having a mean hospital length of stay of 23 days and a very low follow-up rate. If the natural history of VAI includes a very low stroke rate, then therapies with a lower therapeutic index, such as systemic anticoagulation, in the severely injured trauma patient are not supported.

AUTHOR CONTRIBUTIONS

Conception and design: DA, MF
Analysis and interpretation: MG, SS, DA, RH, BD, OG, MF
Data collection: DA
Writing the article: DA, MF, RH, BD
Critical revision of the article: MG, SS, BD, OG, MF
Final approval of the article: MF
Statistical analysis: RH
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Overall responsibility: DA, MF

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DISCUSSION

Dr Gregory Modrall (*Dallas, Tex*). I congratulate Dr Alterman on expertly presenting the Knoxville group's experience with 51 traumatic vertebral artery injuries. These are rare injuries, so a series of this size must be viewed as an important opportunity to learn. In their series, the majority of patients were managed nonoperatively with antiplatelet therapy or anticoagulation. Only two patients received endovascular therapies, and no patient required an open operation. In their experience, vertebral artery injuries proved to be a relatively innocuous injury, as only one patient suffered a posterior circulation stroke, and no patient died as a consequence of a vertebral artery injury. I have four basic questions for the authors:

1. Can the authors surmise why their stroke rate was so much lower than several of the previous papers in the literature on this topic? Is there something different about the mechanisms of injury or anatomic extent of injury to explain this difference? This is an important question because a 20% stroke rate paints a far different picture of these injuries than a 2% stroke rate.
2. Your management approach was nonoperative in most cases, which yielded a relatively low stroke rate. Based on your experience, are there any vertebral artery injuries that you believe should be managed preferentially with early endovascular or surgical intervention?
3. From your manuscript, I could not decipher whether there were any bleeding pseudoaneurysms encountered. If so, how were those injuries managed?
4. Your series recapitulated a common theme in a series of traumatic injuries—poor long-term follow-up. Only about 40% of the surviving patients were ever seen by a vascular surgeon in follow-up, and only six patients had follow-up imaging. For a patient with an asymptomatic vertebral artery occlusion, a lack of follow-up may not be problematic. However, a lack of follow-up could be dangerous for a patient with an untreated vertebral artery pseudoaneurysm. Should the unpredictable follow-up of these patients warrant consideration for early treatment of the subset with vertebral artery pseudoaneurysms?

Again, I congratulate the authors on a fine presentation, and I thank the Society for the privilege of discussing this important paper.

Dr Daniel M. Alterman. Thank you, Dr Modrall, for your thoughtful questions and time. In terms of your first question relating to our incidence and stroke rate, the literature on vertebral artery injury is quite heterogeneous. To our knowledge, we do have the largest series of vertebral artery injury to analyze them apart from blunt carotid artery injury. Previous reports have analyzed blunt carotid and vertebral artery injury together, and it is difficult to interpret their reported stroke rates of the contribution from associated cranial trauma, carotid trauma, or vertebral artery sequelae. Dr Biffl reported 38 patients all diagnosed with four-vessel cerebral angiography, and he reported a stroke rate of 24%. This is often quoted. It is possible that smaller series with different screening methods where the screening was driven by stroke symptoms may have been subject to a type 2 beta error. We did confirm the findings of a more recent series by Miller where he reported 50 patients with vertebral artery injury diagnosed by CTA with no stroke. Our center is very aggressive with CT angiography screening, and it is possible that we are identifying many injuries that would not have been previously recognized. On a further note, the CTA is very sensitive and may initially cause overestimation of an injury. It is possible that many of the patients in our series that are labeled in other series are false positives, and this would dramatically affect the conclusions and treatment algorhythms, so we plan to evaluate this further with blinded analysis.

In terms of your second question related to operative indications, we believe that bleeding or expanding pseudoaneurysms should prompt treatment.

In terms of your third question, intervention for bleeding, points well taken. We did find two injuries that were treated with a stent. One was a bare metal stent placed for dissection.

Your question regarding the poor follow-up is an important point in the trauma patient and affects your disposition and long-term care. Forty percent of the patients in our series were seen after discharge, and we did have six with follow-up imaging. In this demographic, it is well known that there is poor follow-up. This is a point that we could pursue further in the future; however, we cannot assume that any intervention would make a difference in a patient that is stable at discharge, and the unreliable nature of this population may argue against therapies with lower therapeutic index such as systemic anticoagulation.

Dr Kenneth J. Cherry (*Charlottesville, Va*). In our system, we expend a great deal of resources to evaluate vertebral artery injury that includes a neurology consult and serial imaging studies. It is interesting that you found that these injuries may have a more benign natural history than we thought.

Dr Alterman. You raise an excellent point. About 10% to 20% of people have a hypoplastic vertebral artery, and almost 50% have a dominant one. If you have an ipsilateral cervical fracture with imaging that detects a vertebral artery with a smaller caliber, how do you evaluate this? If this finding occurs after hours in the setting of multiple other injuries, it will tip the balance toward labeling this a traumatic dissection. It is possible that many of these "injuries" are false positives and have caused us to either underestimate the morbidity or even overestimate it since the true denominator is unknown. A future step would involve review of these images in a blinded fashion to see what influence the circumstances of the study affect the radiologic diagnosis as well as our basic understanding of normal vertebral artery anatomy.