Blunt Cerebrovascular Injury
Practice Management Guidelines

East Practice Management Guidelines Committee

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Scope of the Problem:

Blunt injury to the carotid or vertebral vessels (blunt cerebrovascular injury – BCVI) is diagnosed in approximately 1/1000 (0.1%) patients hospitalized for trauma in the United States.[1] However the vast majority of these injuries are diagnosed following the development of symptoms secondary to central nervous system ischemia with a resultant neurologic morbidity of up to 80% and associated mortality of up to 40%.[2] When asymptomatic patients are screened for BCVI the incidence rises to 1% of all blunt trauma patients.[3] Key issues that need to be addressed in the diagnosis and management of BCVI include what population (if any) merits screening for asymptomatic injury, what screening modality is best, what is the appropriate treatment for BCVI (both symptomatic and asymptomatic) and what constitutes appropriate follow-up for these injuries.

Process:

Identification of references

A computerized search of the National Library of Medicine/National Institute of Health, Medline database was performed utilizing citations from 1965 to 2005 inclusive. The search terms "cerebrovascular trauma," or "carotid artery" or "vertebral artery" AND wounds and injuries (mesh heading), AND "blunt" limited to the English language returned approximately 1500 citations. Titles and abstracts were reviewed to determine relevance and isolated case reports, small case series, editorials, letters to the editor, and review articles were eliminated. The bibliographies of the resulting full text articles were searched for other relevant citations and these were obtained when appropriate. One hundred sixty two articles were selected for review and of these 60 met criteria for inclusion and are excerpted in the attached evidentiary table.

Quality of the references

The Eastern Association for the Surgery of Trauma "Utilizing Evidence Based Outcome Measures to Develop Practice Management Guidelines: A Primer" was utilized as the quality assessment instrument applied to the development of this protocol.[4] Articles were classified as Class I, II, or III according to the following definitions:

Class I: Prospective, randomized, controlled trial (there were no Class I articles reviewed)

Class II: Clinical studies in which the data was collected prospectively, and retrospective analyses which were based on clearly reliable data. Types of studies so classified include: observational studies, cohort studies, prevalence studies, and case control studies. There were 23 Class II studies identified.

Class III: Studies based on retrospectively collected data. Evidence used in this class includes clinical series, database or registry reviews, large series of case reviews, and expert opinion. There were 37 Class III studies identified.

Establishment of recommendations

A committee consisting of 10 trauma surgeons was convened to review the data and establish these recommendations using these definitions:[5]

Level 1: The recommendation is convincingly justifiable based on the available scientific information alone. This recommendation is usually based on Class I

data, however strong Class II evidence may form the basis for a Level 1 recommendation, especially if the issue does not lend itself to testing in a randomized format. Conversely, low quality or contradictory Class I data may not be able to support a Level 1 recommendation.

No Level 1 guidelines were supported by the literature.

Level 2: The recommendation is reasonably justifiable by available scientific evidence and strongly supported by expert opinion. This recommendation is usually supported by Class II data or a preponderance of Class III evidence.

Seven Level 2 guidelines were establish by the literature.

Level 3: The recommendation is supported by available data but adequate scientific evidence is lacking. This recommendation is generally supported by Class III data. This type of recommendation is useful for educational purposes and in guiding future clinical research.

Nine Level 3 guidelines are proposed for this topic.

Recommendations

Question addressed: What patients should be screened for blunt cerebrovascular injury?

Level 1: No Level 1 recommendations can be made.

Level 2:

1. Patients presenting with any neurologic abnormality that is unexplained by a diagnosed injury should be evaluated for BCVI.

2. Blunt trauma patients presenting with epistaxis from a suspected arterial source following trauma should be evaluated for BCVI.

Level 3:

1. Asymptomatic patients with significant blunt head trauma as defined below are at significantly increased risk for BCVI and screening should be considered.

   Risk factors:

   - GCS ≤8
   - Petrous bone fracture
   - Diffuse axonal injury
   - Cervical spine fracture
   - Fracture through the foramen transversum
   - Lefort II or III facial fractures

2. Pediatric trauma patients should be evaluated using the same criteria as the adult population.

Question addressed: What is the appropriate modality for the screening and diagnosis of BCVI?

Level 1: No Level 1 recommendations can be made.

Level 2:

1. Diagnostic four vessel cerebral angiography (FVCA) remains the gold standard for the diagnosis of BCVI.
2. Duplex ultrasound is **not** adequate for screening for BCVI.
3. CT angiography with a 4 (or less)-slice multidetector array is neither sensitive nor specific enough for screening for BCVI.

Level 3:

1. Multi-slice (8 or greater) multidetector CTA has the same rate of detection for BCVI when compared to historic control rates of diagnosis with FVCA and should be considered as a screening modality in place of FVCA.

Question: How should BCVI be treated? This references a grading scheme proposed by Biffl et al.[6]

Grading scale

Grade I – intimal irregularity with <25% narrowing
Grade II – dissection or intramural hematoma with >25% narrowing
Grade III – pseudoaneurysm
Grade IV – occlusion
Grade V – transection with extravasation

Level 1: No Level 1 recommendations can be made.

Level 2:

1. Barring contraindications, Grade I and II injuries should be treated with antithrombotic agents such as aspirin or heparin.

Level 3:

1. Either heparin or antiplatelet therapy can be used with seemingly equivalent results. A number of authors still recommend heparinization if there is no contraindication, reserving anti-platelet agents for those patients with relative contraindications to heparinization.
2. If heparin is selected for treatment, the infusion should be started without a bolus and titrated to an aPTT of 50-60 sec.
3. In patients in whom anticoagulant therapy is chosen conversion to warfarin titrated to a PT INR of 2-3 for 3-6 months is recommended.

4. Grade III injuries (pseudoaneurysm) rarely resolve with observation or heparinization and invasive therapy (surgery or angio-interventional) should be considered. *N.B.* carotid stents placed without subsequent anti-platelet therapy have been noted to have a high rate of thrombosis in this population.[7]
5. In patients with an early neurologic deficit and an accessible carotid lesion operative or interventional repair should be considered to restore flow.
6. In children who have suffered an ischemic neurologic event, aggressive management of resulting intracranial hypertension up to and including resection of ischemic brain tissue has improved outcome as compared to adults and should be considered for supportive management.

Question addressed: For how long should antithrombotic therapy be administered?

No recommendations can be made for this question.

Question addressed: How should one monitor the response to therapy?

Level 1: No Level 1 recommendation can be made.

Level 2:

1. Follow-up angiography is recommended in Grade I-III injuries. In order to reduce the incidence of angiography-related complications this should be performed after 7 days post injury.

Level 3: There are no Level 3 guidelines for this question.

Scientific Foundation:

Screening and Diagnosis

Symptomatic patients that undergo FVCA for the indications of unexplained neurologic symptoms or arterial epistaxis the diagnosis of BCVI is made in a significant percentage of cases (38-100%) and is clearly recommended as a reason to pursue the diagnosis.[8, 9, 10]

Screening asymptomatic patients at risk for BCVI is more controversial. Multiple studies have indicated that patients with BCVI often present hours to days prior to the onset of symptoms.[11, 12, 13] Failure to identify and treat these injuries can result in significant mortality and morbidity.[14] It is clear that screening for BCVI by essentially any modality can diagnosis BCVI prior to the onset of symptoms at rates up to 10 times higher than previously identified.[15] On the basis of this data a number of individuals recommend screening blunt trauma patients at risk for BCVI using 4-vessel cerebral angiography as the diagnostic modality.[16, 17, 18, 19] There is some countervailing opinion.

In a database review of thirty-five thousand patients Mayberry determined that only 17 were diagnosed with BCI of which 11 became symptomatic. Of these only 2

were asymptomatic for over 2 hours post admission, and of these 2, only 1 met criteria for screening. Based on this data Mayberry et al concluded that screening was futile in light of the inability to diagnose the injury prior to the development of symptoms.[20] The majority of the available data does not support this finding. The preponderance of the evidence supports the recommendation that patients at risk for BCVI can be identified and diagnosed prior to the onset of symptoms with the application of an appropriate screening modality.

Criteria for screening/Risk factors

The mechanism of BCVI seems to be associated with cervical hyperextension and rotation, hyperflexion, or direct blow.[21] The factors that are most closely associated with the finding of BCVI are direct evidence of neurologic deficits as noted above. In asymptomatic patients a number of factors have been associated with increased risk of BCVI. Biffl and colleagues performed linear regression analysis of a liberally screened patient population (N =249)and found that there were four independent risk factors for BCAI. These were: 1) GCS<6, 2) Petrous fracture, 3) Diffuse axonal injury, and 4) LeFort II or III fracture. Patients who had one risk factor had a risk of 41% for BCAI. This risk increased to 93% in the presence of all 4 factors. The only risk factor for BVAI was presence of cervical spine fracture. However 20% of patients diagnosed with BCVI selected for screening by the criteria in Table 1 did not have the independent risk factors identified by regression analysis indicating that broad selection criteria are necessary to prevent missed injuries.[22] Cothren retrospectively reviewed patients with BVAI and found that complex cervical spine fractures involving subluxation, fracture into the foramen transversarium, or C1 to C3 fractures were closely associated with this injury.[23] In a prospective review of screening with DFVCA Cothren et al utilized criteria similar to that proposed by Biffl and modified to incorporate those specific cervical spine fracture patterns shown to increase risk of BVAI to select patients for evaluation (Table 2). Seven hundred and twenty-seven patients (4.6%) of all blunt trauma patients were studied and 244 were diagnosed with BCVI for a screening yield of 34%.[24] An isolated cervical seat belt sign without other risk factors and normal physical exam has failed to be identified as an independent risk factor in two retrospective studies and should not be utilized as the sole criteria to stratify patients for screening.[25, 26]

Table 1

Injury mechanism
- Severe cervical hyperextension/rotation or hyperflexion, particularly if associated with
  - Displaced midface or complex mandibular fracture
  - Closed head injury consistent with diffuse axonal injury
- Near hanging resulting in anoxic brain injury

Physical signs
- Seat belt abrasion or other soft tissue injury of the anterior neck resulting in significant swelling or altered mental status

Fracture in proximity to internal carotid or vertebral artery
- Basilar skull fracture involving the carotid canal
- Cervical vertebral body fracture

Screening Criteria for BCVI adapted from Biffl et al[9] (with permission)

Table 2

Signs/symptoms of BCVI
- Arterial hemorrhage
- Cervical bruit
- Expanding cervical hematoma
- Focal neurological deficit
- Neurologic examination incongruous with CAT scan findings
- Ischemic stroke on secondary CAT scan

Risk factors for BCVI
- High-energy transfer mechanism with
  - Lefort II or III fracture
  - Cervical spine fracture patterns: subluxation, fractures extending into the transverse foramen, fractures of C1-C3
  - Basilar skull fracture with carotid canal involvement
  - Diffuse axonal injury with GCS ≤ 6
  - Near hanging with anoxic brain injury

Denver Modification of Screening Criteria for BCVI adapted from Cothren et al[59] (with permission)

Screening Modality

Duplex Sonography

Multiple studies have shown that duplex sonography is not sensitive enough for screening for BCVI with an overall sensitivity from as low as 38.5%[27] to as high as 86% (the latter for carotid injuries alone).[28, 29] Duplex US cannot be recommended as a screening modality for BCVI.

Angiography

Arguments have been made that DFVCA, in an appropriate group is safe, sensitive, and cost effective. Biffl et al report a 27% rate of positive screening angiogram when asymptomatic patients were screened according to the criteria in Table 1.[30] Cothren[31] utilized DFVCA in 727 asymptomatic patients that met screening criteria (Table 2) in which he found 244 patients with injury (34% screening yield). In patients who were initially asymptomatic and could **not** have antithrombotic therapy there was a 21% (10/48) rate of ischemic neurologic event (INE) whereas in those treated with either heparin, low molecular weight heparin, or antiplatelet agents only one of 187 had an INE. Using this internal data Cothren estimated that the identification and treatment of asymptomatic BCVI in these 187 patients prevented 32 strokes. This comes at an expense (charge data) of $6500 per angiogram for a total of approx. $154 000 per stroke avoided. Cothren concludes that this is cost-effective and screening with DFVCA should be pursued. The argument against the utilization of DFVCA (aside from that against screening *per se*) is that it is expensive (approx $1500)[32], carries an inherent risk of stroke (1-2%)[33] and is impractical to apply at many institutions.[34]

Magnetic Resonance Angiography

In so far as MRA is non-invasive and requires no contrast administration MRA/MRI has been gaining popularity as an alternative to DFVCA for the diagnosis of BCVI. Although a number of studies describe the use of MRA to identify BCVI [35, 36, 37, 38] at this time the few direct studies that do exist indicate that sensitivity and specificity is significantly lower than that of DFVCA. In a (albeit small) direct comparison of MRA vs. angiography Miller et al found a sensitivity of 50% for CAI and 47% for VAI.[39] Levy also reported a significantly lower sensitivity for MRI and MRA than angiography for the diagnosis of BCVI.[40] It seems that, based on this data MRA cannot be recommended as the sole modality for the screening of BCVI.

Computed Tomographic Angiography

Early CT angiography with 1 to 4 slice scanners is not sensitive enough to qualify as an adequate screening modality for BCVI. In a prospective study of CTA on a single slice scanner vs. DFVA Biffl et al report a sensitivity and specificity of 68% and 67% respectively.[41] Similarly Miller et al compared 4-slice CTA vs. DFVCA and showed that CTA performed poorly with a sensitivity of 47% for CAI and 53% for VAI.[42] Sensitivity and specificity seems to improve in direct relationship to improvements in technology, however. In a prospective study which included images obtained from single, four and eight-slice scanners Bub reports improvement in image quality and concomitant improvement in sensitivity and specificity as the number of detectors increases. The overall results for the mixed population (reported as ranges from different observers) was

83-92% sensitivity and 88-92% specificity for the carotid artery and 50-60% sensitivity and 90-97% specificity for the vertebral artery.[43] Berne et al screened patients with 4-slice and, later, 16-slice scanner CTA in a study in which only positive CTA studies underwent confirmatory angiography showing an overall sensitivity (for symptomatic BCVI) and specificity of 100% and 94% respectively. Interestingly the incidence of BCVI detected went up from 0.6% with the earlier machine to 1.05% with the newer device, approaching historic incidence of BCVI as diagnosed by DFVCA and the comparative specificity improved from 90.8% to 98.7%.[44] In a follow-up study Berne et al screened patients for BCVI solely with a 16-slice scanner. In this prospective study Berne showed that the detected incidence of BCVI goes up threefold when changing from a 4-slice scanner to a 16-slice scanner with a resulting incidence of 1.2% which is similar to that found by screening with DFVCA.[45] In a similar study in which only positive 16-slice CTA studies were followed by DFVCA, Biffl et al reversed an earlier recommendation[46] that CTA was not adequate for screening for BCVI reporting a sensitivity of 100% for symptomatic BCVI.[47] Schneidereit and colleagues report similar findings and give a diagnosed incidence for BCVI of 1.4% using a 16-slice scanner.[48] Although these studies are interesting obviously a true sensitivity can only be obtained via direct comparison between CTA and DFVCA. At this time only one study has directly compared 16-slice CTA vs. angiography for screening for BCVI. Eastman et al performed 162 CTAs followed by 146 confirmatory DFVA studies (12 patients refused consent, 4 were discharged, and 6 died of non-neurologic causes prior to the study being obtained). Twenty carotid injuries and 26 vertebral artery injuries were identified with one false negative CTA (a grade I vertebral artery injury) for a screened population incidence of 28.4% and an overall incidence of 1.25%. The overall sensitivity, specificity, positive predictive value, negative predictive value, and accuracy were 97.7%, 100% 100%, 99.3%, and 99.3% respectively.[49]

Blunt cerebrovascular injuries in children: There is a relative paucity of information on the screening, diagnosis, and management of BCVI in children and what is available primarily consists of isolated case reports and small case series. In one review of the National Pediatric Trauma Registry (NPTR) Lew and colleagues found an overall incidence of 0.03%, which is lower than that of the adult trauma population and speculated that it may be due to the increased elasticity of the younger children's blood vessels. They did note that another possibility was that the difference was secondary to decreased detection in children and the retrospective nature of the study. Children under six years of age seemed to be at higher risk, making up 73% of patients with BCVI whereas they made up only 36% of the registry patients. Chest trauma (in particular clavicle fracture) and severe head injury (basilar skull fracture, intracranial hemorrhage) were associated with a higher risk of BCVI in the pediatric population.[50] In a case review of 5 patients with BCI Duke and Partington[51] recommend initial treatment of the arterial injury to be the same as in adults. Where recommendations differ is that they go on to recommend aggressive management of intracranial hypertension in children up to and including resection of infracted tissue due to improved outcome in pediatric patients in contradistinction to the dismal outcome of post-ischemic intracranial hypertension in adults.

Treatment of BCVI

Surgery – a number of studies from the 80's and 90's have concluded that if individuals have minimal or no symptoms and an accessible carotid lesion they do well with operative intervention and therefore recommend repair of any more than minor intimal irregularities.[52,53,54] However most of these studies also note that if patient present with profound neurologic deficit, revascularization does not improve outcome. In all studies that have compared ligation v. repair, those patients that do not have a profound deficit do much better with repair.[55, 56] Karlin for example found a 7.8% mortality in patients undergoing repair v. 50% in those undergoing ligation and that, furthermore, those patients who did not have a deficit prior to surgery did not develop one if revascularized.[57] Finally a vast majority of these studies including Richardson[58] indicate that if the patient presents with a dense neurologic deficit, neither operation nor anticoagulation improves outcome. All of these studies however were of Class III quality.

Anticoagulation – there have been a number of studies attempting to evaluate the impact of antithrombotic agents on the progression or development of sequellae of BCVI. As is not unexpected the results have been somewhat contradictory but the weight of the evidence seems to support the administration of antithrombotic agents to those patients with BCVI who do not have contraindications for such. A series of retrospective studies[59, 60, 61, 62] found that administration of antithrombotic agents reduces the rate of neurologic sequellae after BCVI. Fabian also indicated that mortality also improves with heparinization in this population. Although there has not been a direct, controlled comparison of heparinization vs. antiplatelet agents (aspirin or clopidigrel) in the prevention of CVA after BCVI, a number of studies performed subgroup analysis in an attempt to address this question. In one of these studies Biffl[63] compared those patients treated with ASA v. heparin and found a trend towards reduction in CVA for those treated with heparin (1% v. 9% p=0.07) however in studies by Wahl,[64] Cothren,[65] and a second study by Biffl,[66] failed to demonstrate a difference in outcome between the two modalities. In these previously mentioned studies both Cothren and Biffl still recommend heparinization as first line therapy for those patients without contraindications, reserving antiplatelet agents for those not deemed to be candidates for anticoagulation.

Serious bleeding complications can accompany aggressive anticoagulation regimens. In a mixed population of patients with both blunt and penetrating carotid injury Nanda[67] found that, in patients with a pre-existing intra-cerebral hemorrhage, anticoagulation resulted in worsening in 2/3. Extracranial hemorrhage is another frequent complication of systemic heparinization in polytrauma patients. For example in a previously mentioned study Biffl[68] noted that bleeding which required either transfusion or cessation of heparin was encountered in 54% of patients prompting him to recommend a conservative protocol for the initiation and maintenance of the heparin infusion and tight control of aPTT to within 40-50 seconds in a later study.[69]

Angiointerventional therapy – There have been several preliminary, Class III studies that have indicated the safety and feasibility of catheter directed therapy to include embolization of pseudoaneurysms and stenting of intimal injuries.[70, 71, 72, 73] A more recent Class II study by Cothren[74] indicated that the carotid artery occlusion rate in patients who underwent stenting is much higher than that of patients with BCAI who

were treated with antithrombotic agents alone. This resulted in a rate of complications (3 CVA and one subclavian artery dissection) of 21% in stented patients v. 5% in non-stented patients (no one who was received anticoagulation suffered a CVA). The author goes on to add that the reason for this may be that patients who had undergone stenting were then treated with heparin and not anti-platelet agents and recommends a study to evaluate this.

Monitoring response to therapy – In a Class II study, Biffl[75] found that follow-up angiography changes management in 61% of BCVI, particularly in that Grade 1 and 2 injuries often go on to complete healing or to form a pseudoaneurysm within 7-10 days. The author went on to note that the complication rate of angiography was significantly higher if the follow-up procedure was performed within 7 days and recommends that at least that amount of time be allowed to lapse prior to follow-up angiography.

Future Directions.

Screening – Blunt cerebrovascular injury is a rare entity (though not as rare as formerly thought), which requires a high index of suspicion to identify prior to the onset of symptoms. The clinical and cost-effectiveness of a screening program depends on both disease-specific, test specific, and organizational issues as well as the utility (or futility) of the treatment modalities available. Further prospective investigation is necessary to further refine the screening criteria so as to maximize the disease incidence in the screened population which will increase accuracy and decrease costs.

Treatment – the optimum modality for the treatment of BCVI is as yet undetermined. Prospective studies will be necessary to compare invasive intervention v. anticoagulation. Furthermore the optimal anticoagulation regimen is as yet unknown in terms of agent (anti-platelet v. heparinoid v. warfarin) as well as the duration and endpoint of therapy. Cleary there is room for further study in this regard. In light of the relative rarity of the disease entity, systematic, multi-institutional studies will be required to answer this question.

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Evidentiary Table

First Author	Year	Reference	Data Class	Conclusions/Comments
Ahmad HA	1999	Cervicocerebral artery dissections. J Accid Emerg Med. 1999;16:422-424	III	**Design:** Retrospective review of 18 mixed traumatic and non-traumatic cases . **Findings:** 1. 61% of patients develop symptoms >24 hours 2. 71% of patients presented with normal head CT . **Recommendations:** 1. Most patients present with delayed neurologic deficits and therefore high risk groups should undergo arteriography. 2. Minimal adverse outcomes related to use of anticoagulation, therefore medical therapy advised.
Batnitzky S	1983	Cervical internal carotid artery injuries due to blunt trauma. Am J NeuroRadiol. 1983;4:292-295	III	**Design:** Retrospective review of 21 cases of blunt carotid injury. **Findings:** 1. Greater than 50% had delayed presentation (from 3 hrs to 4 days). 2. 20% presented with no external trauma. **Recommendations:** 1. Angiography is the definitive radiologic procedure to rule out blunt carotid injuries. 2. Angiography should be performed in all patients in whom blunt carotid injury is suspected.
Berne JD	2001	The high morbidity of blunt cerebrovascular injury in an unscreened population: more evidence of the need for	III	**Design:** Registry review, identified 30 patients over 4 years.

		mandatory screening protocols. J Am Coll Surg. 2001;192:314-321		**Findings:** 1. Blunt cerebrovascular injury is uncommon (0.48% of all blunt trauma admissions) but lethal (59% mortality), particularly when diagnosis is delayed. 2. Most deaths (80%) are directly attributable to the BCVI and not to associated injuries. 3. Chest injury, rib fractures, and basilar skull fracture were significant predictors of BCAI 4. Closed head injury, basilar skull fracture, and rib fractures were significant predictors of BCVI **Recommendations:** 1. Aggressive screening based on mechanism of injury, associated injuries, and physical findings are justified to minimize morbidity and mortality. 2. Head & chest injuries may serve as markers for BCVI.
Berne JD	2004	Helical computed tomographic angiography: an excellent screening test for blunt cerebrovascular injury. J Trauma. 2004;57:11-19	II	**Design:** Prospective screening to identify BCVI with helical CTA using a four-slice scanner initially and then 16 slice. All positive CTAs were followed by angiography. All the negative CTA patients were followed by physical exam during admission and none manifested symptoms of BCVI. They did not perform angiography in patients with negative CTA. Screening was based on following injuries a. Basilar skull fracture b. C-spine injury c. Severe facial fracture d. Hematoma or bruise to neck e. GCS < 8 f. Lateralizing neurological signs **Findings:** 1. Incidence of BCVI diagnosed with CTA was 0.6% 2. A combination of 4 and 16-slice CTA was

				found to have a sensitivity of 100%, specificity of 94% PPV 37.5%, NPV 100% for clinically important BCVI **Recommendations:** 1. Diagnostic screening with CTA accurately identifies all clinically significant BCVI. 2. FVCA is impractical as a screening mechanism at most institutions
Berne JD	2006	Sixteen-slice multi-detector computed tomographic angiography improves the accuracy of screening for BCVI	II	**Design:** Prospective screening protocol initiated based on injury criteria which led to CTA using a 16-slice scanner. Positive, equivocal, and suspicious studies were followed up with FVCA. Patients with negative studies were followed clinically. This is a subset of an earlier group that was then compared to CTA with a 4-slice scanner. **Findings:** 1. Incidence of BCVI diagnosed with 16-slice CTA was 1.2% (same as historic controls screened with FVCA) as compared to 0.38% with 4-slice CTA. 2. No patient with an initial negative CTA went on to develop symptoms. 3. Mortality improved from 59% to 29% with the initiation of screening. **Recommendations:** 3. Diagnostic screening with 16-slice CTA accurately identifies all clinically significant BCVI. 4. Screening for BCVI is indicated as it can decrease BCVI-related mortality.
Biffl WL	1998	The unrecognized epidemic of blunt carotid arterial injuries: early diagnosis improves neurologic outcome. Ann Surg. 1998;228:462-470	III	**Design:** Retrospective registry review of 15,331 blunt trauma patients. Compared unscreened population (prior to 1996) to screened population. **Findings:** 1. Incidence of BCI prior to screening was 0.1% (all symptomatic). 2. Incidence of BCI post screening was 0.86% of which 72% were asymptomatic at the time

				of diagnosis. 3. There is a trend to neurologic improvement in symptomatic BCI patients treated with heparin. 4. Hemorrhagic complications of anticoagulation are common in the trauma population. **Recommendations:** 1. Aggressive screening for BCI based on injury patterns is warranted. 2. Early institution of heparin therapy is indicated (with a target aPTT of 40-50). 3. Follow-up angiography should be withheld until at least 7 days post injury.
Biffl WL	1999	Blunt carotid arterial injuries: implications of a new grading scale. J Trauma. 1999;47:845-853	II	**Design:** Initially retrospective review followed by prospective protocol. **Findings**: 1. A grading scale is proposed – see text. 2. Grade I injuries a. 7% of progressed to Grade 2 or higher b. there was no difference in healing in patients given either heparin or antiplatelet agents. c. 3% risk of stroke if untreated. 3. Grade II injuries a. 10% healing rate with heparin. There was no comparison made to antiplatelet agents or to no treatment. b. 70% progressed to higher grade injury on repeat angiogram. c. 11% stroke rate if untreated. 4. Grade III injuries a. 8% healed with heparin initially. One occluded. b. 33% stroke rate if untreated.

				c. If GI or II progressed to III none healed. 5. Grade IV injuries a. none healed with medication alone b. 44% stroke rate if untreated. 6. Grade 5 (transection) – 100% mortality **Recommendations:** 1. Repeat angiogram at or after 10 days to evaluate for evolving or healed lesion. 2. Grade II injuries should be treated with heparin anticoagulation. 3. Grade III injuries a. surgical repair is front-line therapy in accessible lesions b. stenting for BCAI is risky in the acutely injured artery and should be delayed 7 days c. endovascular stents planed in traumatized arteries should be treated adjunctively with full systemic anticoagulation. 4. Grade IV injury – treat with heparin anticoagulation to prevent stroke.
Biffl WL	1999	Optimizing screening for blunt cerebrovascular injuries. Am J Surg. 1999;178:517-522	II	**Design:** Prospective observational study in which 249 patients meeting certain screening criteria underwent DFVCA. **Screening Criteria:** 1. Neurologic signs of BCVI 2. Injury mechanism a. Severe cervical hyperextension/rotation or hyperflexion particularly if associated with i. Displaced midface or complex mandibular fracture ii. Closed head injury consistent with diffuse axonal injury b. Near-hanging resulting in anoxic

				brain injury 3. Signs a. Seat-belt abrasion or other soft tissue injury of the anterior neck resulting in significant swelling or altered mental status 4. Fracture in proximity to internal carotid or vertebral artery a. Basilar skull fracture involving the carotid canal b. Cervical vertebral body fracture **Findings:** 1. Incidence of BCVI in screened population was 34% 2. In patients screened for symptoms incidence was 70%. 3. In asymptomatic patients incidence was 27%. 4. Linear regression analysis identified these risk factors for BCVI a. GCS ≤6 b. Petrous bone fracture c. Diffuse axonal injury d. Lefort II or III fractures e. Cervical spine fracture (specifically for BVAI) **Recommendation:** 1. Screening angiography based on the above criteria is indicated to identify BCVI.
Biffl WL	2000	The devastating potential of blunt vertebral arterial injuries. Ann Surg. 2000;231:672-681	III	**Design:** Retrospective review of prospectively collected data. **Findings:** 4. Incidence of BVI was 0.53% 5. Stroke incidence in BVI was 24%, Mortality 18%, BVI-attributable mortality 8% 6. Neurologic complications were not associated with injury grade. 7. Trend to improvement in neurologic outcome with anticoagulation.

				8. Cervical spine injury is independently associated with BVAI. **Recommendations:** 1. Screening for BCVI is indicated and should include all those with cervical injury, unilateral headache, and posterior neck pain when sudden, severe, and unlike previous pain. 2. Arteriography is the gold standard for diagnosis of BCVI 3. Anticoagulation improves neurologic outcome.
Biffl WL	2002	Noninvasive diagnosis of blunt cerebrovascular injuries: a preliminary report. J Trauma. 2002;35:850-856	II	**Design:** 46 asymptomatic patients selected by application of a previously reported screening algorithm underwent both arteriogram and either CTA (single slice scanner) or MRA. **Findings:** 1. CTA: 7/23 false negatives and had 8/23 false positives (sensitivity 68%, specificity 67% PPV 65%, NPV 70%). 2. MRA had 1/11 false negatives , 4/7 false positives (sensitivity 75%, specificity 67% PPV 43%, NPV 89%). 3. Both CTA and MRA failed to reliably identify Grade I, II, and III injuries. **Recommendations:** 1. Angiography remains the gold standard for the screening and diagnosis of BCVI at the time of this publication 2. If DFVCA is unavailable CTA or MRA should be used to screen for BCVI in patients at risk.
Biffl WL	2002	Treatment-related outcomes from blunt cerebrovascular injuries. Importance of routine follow-up arteriography. Ann Surg. 2002;235:699-707	II	**Design:** A retrospective review of a prospectively collected database. **Findings:** 1. Incidence of BCVI is found to be 1.55% with a screening protocol.

				2. In patients diagnosed with BCAI f/u angiography showed healing of grade I injuries 57% in 7-10 days and 8% grade II (allowed cessation of Rx). However 8% GI and 43% GII injuries progressed to pseudoaneurysm. 3. Grade III and IV injuries rarely changed in early follow-up (93% and 82% unchanged respectively). 4. 23% of BCAI and 20% BVAI developed an INE and risk of INE increased with grade of injury. 5. Trend towards improvement of neurologic outcome in both heparin v. ASA (Stroke rate was 1% on heparin and 9% on ASA p=0.07) and heparin v. no therapy but not statistically significant. 6. There was a complication rate of 22% with anticoagulation. 20/22 bleeds were on aggressive therapeutic protocol (bolus dose followed by PTT 60-80) this was 20/47pts (46%). Subsequently a less aggressive protocol (no bolus and goal PTT of 40-50) resulted in only a 4% incidence (2/53 patients) of bleeding complications. **Recommendations:** 1. Follow up angiography is recommended at 7-10 days because findings that will require a change in management are likely. 2. Anticoagulation is recommended for the treatment of BCVI in those patients without contraindication. A non-aggressive heparin protocol is suggested. 3. Grade IV injuries are unlikely to improve without intervention.
Biffl WL	2006	Sixteen-Slice CT-angiography is a reliable noninvasive screening test for clinically significant blunt cerebrovascular injuries	II	**Design:** Prospective evaluation of 16-slice CTA in a screening role. A positive CTA was confirmed with DFVCA. Patients with a negative CTA were followed clinically.

				Findings: 1. No patient with a negative CTA developed neurologic signs of BCVI 2. False positive rate of 1.2% with CTA. 3. the most liberal screening protocol continues to miss clinically significant BCVI **Recommendations:** 1. 16-slice CTA is a reliable noninvasive screening test for clinically significant BCVI.
Bub LD	2005	Screening for BCVI: Evaluating the accuracy of Multidetector CTA	III	**Design:** Retrospective review **Findings:** 1. When evaluating data obtained by pooling images obtained by either a 4 and 8 slice CT scanner, the sensitivity and specificity of CTA for CAI was 88% and 94% and for VAI was 50% and 95% respectively. 2. The 8 slice CT scanner showed improved images subjectively. **Recommendations:** 1. Angiography continues to have higher sensitivity and specificity when compared to 4 and 8-slice CTA. 2. Imaging sensitivity will likely improve with newer generation technology.
Carrillo EH	1999	Blunt carotid artery injuries: difficulties with the diagnosis prior to neurologic event. J Truama. 1999;46:1120-1125	III	**Design:** Review of 21,428 patient registry which identified 30 injured patients. **Findings:** 1. Incidence of symptomatic BCAI is 0.14% 2. 23% presented with neurologic symptoms with normal head CT. 3. No injuries were identified based on angiography in asymptomatic patients with a normal head CT. 4. Duplex US missed 1/3 injuries in which it was utilized. **Recommendations:**

				1. Screening of asymptomatic patients is not justified. 2. Duplex scanning is not useful for the diagnosis of BCVI. 3. A complex treatment algorithm is proposed which recommends: a. Surgical repair in accessible lesions without thrombosis. b. Anticoagulation in inaccessible lesions without thrombosis or contraindication. c. Antiplatelet therapy for inaccessible lesions with contraindication to anticoagulation. d. Antiplatelet therapy v. anticoagulation for thrombosed vessels. e. Endovascular embolization for certain lesions
Cogbill TH	1994	The spectrum of blunt injury to the carotid artery: a multi-center perspective. J Trauma. 1994;37:473-439	III	**Design:** Retrospective review of 49 patients (60 injuries) from 11 institutions. **Findings:** 1. Neurologic symptoms may develop after blunt carotid injury in a delayed fashion 2. Injuries with complete arterial thrombosis are associated with high mortality and poor neurologic outcome in proportion to the initial degree of neurologic impairment. 3. Sensitivity of Duplex US is 86%. 4. Injury specific mortality was 19%. **Recommendations:** 1. Surgical repair is indicated for the treatment of pseudoaneurysms in accessible locations. 2. Systemic anticoagulation is the primary method of treatment for arterial dissections in the absence of a pseudoaneurysm or complete thrombosis. 3. The optimal method of management for arterial thrombosis remains poorly defined. 4. Balloon occlusion effectively treats carotid-

				cavernous fistula.
Coldwell DM	2000	Treatment of posttraumatic internal carotid arterial pseudoaneurysms with endovascular stents. J Trauma. 2000;48:470-472	III	**Design:** Case series of 14 patients with blunt carotid pseudoaneurysms treated with metallic endoprostheses and anticoagulation. **Findings:** 1. No patients developed neurologic symptoms post stenting. 2. 12/14 patients showed complete healing at 2 month follow-up. The other 2 patients were healed at the 4 month follow-up. 3. One patient had intimal hyperplasia and 10% stenosis at 3-month follow-up. **Recommendations:** 1. Endovascular stenting with metallic endoprostheses followed by anticoagulation is safe and effective in the treatment of carotid pseudoaneurysm.
Colella JJ	1996	Blunt carotid injury: reassessing the role of anticoagulation. Am Surg. 1996;62:212-217	III	**Design:** Retrospective database review which identified 20 patients with BCAI. **Findings:** 1. 10/12 patients treated with heparin survived with normal neurologic function. 2. 2 patients died while on heparin, one from infarct progression and one from a new infarct. 3. 2 patients were treated with antiplatelet therapy (aspirin, 325mg/day) and survived without deficit 4. 2 patients received no therapy of which one survived without associated deficit. The other died of massive left middle cerebral artery infarction. **Recommendation:**. 1. Patients without contraindication to heparin should be heparinized, however "with careful patient selection, a delay in the initiation of heparin therapy, no therapy, or aspirin

				therapy, may all be appropriate in the initial management."
Cothren CC	2004	Anticoagulation is the gold standard therapy for blunt carotid injuries to reduce stroke rate. Arch Surg. 2004:139:540-546	II	**Design:** Prospectively collected, observational study, non-randomized. **Findings:** 1. Incidence of BCAI is 0.86% of blunt trauma patients undergoing a screening protocol. 2. In patients treated with either a) systemic heparin, b) subcutaneous low-molecular-weight heparin, or c) antiplatelet agents no-one developed an ischemic neurologic event (INE). 3. Of 27 asymptomatic patients with BCVI that did not receive anticoagulation secondary to contraindications, 5 (19%) developed an INE. **Recommendations:** 1. Asymptomatic patients with BCAI and without contraindication to anticoagulation should be anticoagulated to reduce the incidence of INE.
Cothren CC	2005	Carotid artery stents for BCVI: Risks exceed benefits.	II	**Design:** Prospectively collected database of patients with CAI treated with stenting. Post stenting patients were placed on therapeutic warfarin. Stent patients received follow-up angiography. Patients treated with antithrombotic agents alone were followed clinically. **Findings:** 1. 45% of patients who underwent carotid stenting had documented occlusion v. 5% of patients receiving antithrombotic agents alone. However only 2/23 received post-stent antiplatelet agents (18 received heparin to warfarin, 3 received nothing). 2. There was a 21% procedure-related complication rate associated with stenting. **Recommendation:** 1. Carotid stenting should be performed in

				selective cases and antithrombotic agent therapy remains the cornerstone of treatment for posttraumatic pseudoaneurysms.
Cothren CC	2005	Screening for blunt cerebrovascular injury is cost effective	III	**Design:** Retrospective review of a prospectively collected database. **Findings:** 1. An aggressive screening program using FVCA per protocol identified 244 patients with BCVI (34% of those selected for screening). 2. Extrapolating from previously obtained data on the utility of treating asymptomatic BCVI with anticoagulation the authors estimate that this prevented 32 ischemic neurologic events (INE). 3. Further extrapolating based on previously obtained data in which the mortality of patients with and without INE was 18% and 7% respectively the authors estimate that this prevented 3.2 lives. 4. Based on charges of $6,500 per angiogram the authors report a charge of $146,672 per INE avoided or $1,476,719 per life saved. **Recommendations:** 1. Screening of selected at risk patients for BCVI with angiography is cost effective "not only in terms of pure dollars to the institution but also from a patient and family perspective." 2. Surgeons caring for the multiply injured should screen for carotid and vertebral artery injuries in high-risk patients.
Davis JW	1990	Blunt carotid artery dissection: incidence, associated injuries, screening and treatment. J Trauma. 1990;30:1514-1517	III	**Design:** Retrospective review, multi-institutional. **Findings:** 1. The rate of blunt carotid dissection was found to be 0.08% in an unscreened population of blunt trauma patients. 2. Carotid duplex identified all 5 injuries in

				which it was utilized for screening. 3. Combination of head injury + facial fractures or head injury + C-spine injury had an increased risk of BCI. **Recommendations:** 1. Duplex scan appears to be a useful screening test in patients at increased risk for BCD. 2. A positive duplex scan should be followed by angiography of the aortic arch with selective studies of the carotid arteries.
DiPerna CA	2002	Clinical importance of the "seat belt sign" in blunt trauma to the neck. Am Surg. 2002;5:441-445	III	**Design:** Retrospective review of 131 patients who presented with cervical seat belt sign and subsequently underwent duplex ultrasonography. **Findings:** 5. One patient was found to have a BCI by duplex scan (incidence of 0.76%). This patient had presented with lateralizing signs. 6. No initially asymptomatic patient (50 patients) was found to have an injury by duplex scan, nor did they develop symptoms. **Recommendations:** 2. A cervical seat belt sign should not serve as a sole indicator for evaluation of the carotid artery in the absence of other pertinent signs or symptoms. **Note:** based on the small number of asymptomatic patients (50) and the estimated 1.5% incidence of BCVI in a screened asymptomatic population it is likely that no injuries were present in the asymptomatic group.
Duke BJ	1996	Blunt carotid injury in children. Ped Neurosurg. 1996;25:188-193	III	**Design:** Retrospective review of affiliated hospital databases. Five patients with BCI were identified. **Findings:** 1. No patient was anticoagulated because they all were identified after the development of

				large infarctions. 2. One patient died secondary to bilateral carotid thrombosis. 3. 3 of 5 patients developed elevation of intracranial pressure (ICP) of which 2 required pentobarbital coma and resection of the infarcted portion of brain. All of these patients survived to hospital discharge. **Recommendations:** 1. In light of the better outcomes of children with elevations in ICP following ischemic stroke as compared to adults, aggressive ICP management including resection of infarcted tissue is indicated should intractable intracranial hypertension develop.
Duke BJ	1997	Treatment of blunt injury to the carotid artery by using endovascular stents: an early experience. J Neurosurg. 1997;87:825-829	III	**Design:** Case series of 6 patients who were treated with stenting for worsening pseudoaneurysm on repeat angiography. Stents were anticoagulated with heparin followed by coumadin for 8 weeks followed by aspirin for 1 additional month. **Findings:** 1. Five of the patients had healing on repeat angiography at 2 months. The sixth had healing at 7 months. 2. There was no stenosis or thrombosis of the stents in this series (follow up 2-7 months). 3. Two patients had complications of anticoagulation. One required anticoagulation to be discontinued and this patient was changed to aspirin with no embolic complications. **Recommendations:** 1. The use of endovascular stents may provide a safe and effective alternative to surgery and medical therapy for the treatment of BCIs.
Eachempati SR	1998	Blunt vascular injuries of the head and neck: is heparinization necessary? J Trauma. 1998;45:997-1004	III	**Design:** Retrospective database review. 23 patients with BCVI identified over 9 year period. Extremely heterogeneous treatments and outcomes.

				Findings: 1. Carotid canal fractures and CHI raise risk of BCVI. 2. 5/7 patients treated solely with antiplatelet agents had minimal or no deficit upon discharge. 3. Heparin showed no benefit and produced a 2/13 (16%) complication rate (but only 4/24 patients had heparin initiated within 48 hrs of injury). 4. No patient suffered a fatal outcome or worsened neurologically after diagnosis. **Recommendations:** 1. Suggested a prospective, randomized, multi-institutional trial of heparin v. antiplatelet therapy. 2. Despite findings above, recommended anticoagulation with heparin followed by warfarin for 3-6 months followed by lifelong antiplatelet therapy. 3. Screening angiogram recommended for: a. carotid canal fractures b. neck hematomas c. focal neurological deficits d. deficits not attributable to intracranial findings on CTH
Eastman, AL	2005	Computed tomographic angiography for the diagnosis of blunt cervical vascular injury: Is it ready for primetime?	II	**Design:** Prospective, blinded observational study. Patients were screened for BCVI with 16-slice CTA followed by DFVCA. 162 patients were screened with CTA. 16 did not receive arteriogram secondary to refusal of consent (12) or early discharge (4). 6 patients died from non-neurologic causes prior to angiogram and were excluded. **Note:** this is the only study in which both positive and negative CTA were followed with angiography. **Findings:** 1. 46 BCVIs were identified in 43 patients (20

				BCAIs, 26 BVAIs) for an overall incidence of 1.25% and an incidence of 28.4% in the screened population. 2. For BCVI overall the sensitivity, specificity, PPV, NPV, and accuracy of 16-slice CTA was 97.7%, 100%, 99.3%, and 99.3% respectively. 3. Sensitivity of CTA for BCAI was 100%. Specificity for BCAI was 100%. 4. Sensitivity of CTA for BVAI was 96.1%. Specificity for BVAI was 100%. **Recommendations:** 1. 16-channel, multislice CTA is an effective and sensitive diagnostic test modality for the detection of BCVI.
Fabian TC	1996	Blunt Carotid Injury. Importance of early diagnosis and anticoagulant therapy. Ann Surg. 1996;223:513-525	III	**Design:** A retrospective review of a trauma registry which identified 67 patients with 87 BCAIs over 11 years. **Findings:** 1. Most common indication for angiography was neurologic exam inconsistent with CTH. 2. 57 patients received heparin, 8 no therapy, 6 aspirin, 1 surgery. When compared use of heparin anticoagulation was associated with better neurologic outcome and higher survival than no treatment. 3. 16 or the 21 deaths were directly related to strokes due to BCI. 4. There were six complications of heparin therapy. **Recommendations:** 1. Patients with partial arterial disruption or thrombosis should be treated with heparin anticoagulation. 2. Anticoagulation should be continued as long as abnormality of artery persists.
Fakhry SM	1988	Cervical vessel injury after blunt trauma. J Vasc Surg. 1988;8:501-508	III	**Design:** Retrospective review of 10 patients with 18 blunt cervical vessel injuries over 12 years.

				Findings: 1. All patients diagnosed after the development of symptoms. 2. 80% developed symptoms in a delayed fashion (> 1 hour post admission). 3. 40% of patients had more than one injured vessel. **Recommendations:** 1. Complete FVCA is recommended because of risk of multiple vessel injuries. 2. Patients with BCVI with a fixed deficit and inaccessible lesions can be safely treated with heparin.
French BN	1988	Cranial computed tomography in the diagnosis of symptomatic indirect trauma to the carotid artery. Aust N Z J Surg. 1988;58:651	III	**Design:** Retrospective case series. Documents the natural history of untreated or undiagnosed CAI. **Findings:** 1. The presence of focal neurologic deficits not explained by head CT suggest possible carotid artery injury. **Recommendations:** 1. Patients with focal neurologic deficit unexplained by head CT or ischemic findings on head CT should undergo evaluation for BCVI.
Friedman D	1995	Vertebral artery injury after acute cervical spine trauma: Rate of occurrence as detected by MR angiography and assessment of clinical consequences. Am J Roentgenol. 1995;164:443-447	II	**Design:** Prospective, non-randomized protocol in which all patients with C-spine injury underwent MRI and MRA of cervical spine. No confirmatory angiography. **Findings:** 1. Incidence of BVAI in major cervical trauma was 24% by MRA. 2. With complete motor and sensory deficits the incidence was 50%. 3. Only 1 patient had neurologic sequellae of BVAI (cerebellar stroke secondary to bilateral thrombosis).

				4. Only one patient had treatment directed at the BVAI (heparin). **Recommendations:** 1. MR imaging should be utilized to screen for vascular injury in the acutely injured cervical spine.
Giacobetti RF	1997	Vertebral artery occlusion associated with cervical spine trauma: A prospective analysis. Spine. 1997;22:188-192	II	**Design:** Prospective protocol in which all patients presenting with cervical spine injury underwent MRI/MRA. No confirmatory angiography was performed. **Findings:** 1. The incidence of BVAI following C-spine injury as detected by MRA is 19.7%. 2. 3/12 pts had symptoms related to the BVAI, all resolved on anticoagulation therapy (heparin, followed by coumadin for 3 months). **Recommendations:** 1. Patients with cervical spine injury, particularly with flexion distraction or flexion compression injuries should undergo evaluation of their vertebral vessels.
Halbach VV	1993	Endovascular treatment of vertebral artery dissections and pseudoaneurysms. J Neurosurg. 1993;79:183-191	III	**Design:** Review of endovascular treatment of 16 patients with symptomatic VA dissections and pseudoaneurysms of a mixture of traumatic and spontaneous etiologies. **Findings:** 1. Excellent technical success is reported but no neurologic outcomes are reported. **Recommendations:** 1. Angioembolization is useful in the treatment of symptomatic VA dissections and aneurysms.

Hellner D	1993	Blunt trauma lesions of the extracranial internal carotid artery in patients with head injury. J Craniomaxillofac Surg. 1993;21:234-238	III	**Design:** Retrospective review of 18 patients over 22 years. **Findings:** 1. Development of symptoms was usually delayed for a period ranging from 4 hours to 1 month. 2. Bilateral lesions were common (50%) 3. Outcome was generally poor consisting of only 6 with normal neurologic outcome, 5 deaths and 7 with hemiparesis. **Recommendations:** 1. When a carotid lesion is suspected bilateral angiography should be performed.
Hughes KM	2000	Traumatic carotid artery dissection: a significant incidental finding. Am Surg. 2000;11:1023-1027	III	**Design:** Retrospective review of patients who had incidental findings of BCAI when being screened for c-spine injury with MRI. Patients were subsequently treated medically. **Findings:** 1. The incidence of incidental CAI is 3.7%. 2. 1/2 patients who underwent observation alone died. 3. No patient treated medically (4 given heparin followed by warfarin, 1 treated with aspirin) had infarction or hemorrhagic complication. **Recommendations:** 1. MRI/MRA screening of the head and neck should be instituted for patients who fit the profile for occult carotid injury. 2. Medical therapy provides excellent results.
Kerwin AJ	2001	Liberalized screening for blunt carotid and vertebral artery injuries is justified. J Trauma. 2001;51:308-314	II	**Design:** Prospectively collected data utilizing liberal screening criteria for the detection of asymptomatic BCVI in high risk patients based on the following injuries: a. Anisocoria b. Mono or hemiparesis c. Neurologic symptoms unexplained by head d. Basilar skull fracture near carotid artery

				e. Fracture of foramen transversarium f. CVA, TIA g. Massive epistaxis h. Severe flexion/extension injury of the C-spine i. Massive facial fracture j. Neck hematoma **Findings:** 1. Of the 48 patients screened, 21(44%) had BCVI (19 BCAI and 10 BVAI). 2. Overall incidence was 1.1%. 3. 5 patients of 1941 total blunt trauma victims developed delayed symptoms and had been missed by screening criteria. 4. Incidence with selected findings a. CVA, TIA, massive epistaxis : 100% b. Fracture through foramen transversarium: 60%. c. Unexplained hemiparesis: 44% d. Basilar skull fracture: 42% e. Unexplained neurologic exam: 38% f. Anisocoria: 33% g. Severe facial fracture 0%. 5. 43% of BCVIs were identified prior to the development of neurologic symptoms. 6. No difference in outcome was demonstrated between those patients treated with heparin, aspirin, or observation. **Recommendations:** 1. Liberal screening is justified. 2. They will continue to anticoagulate with heparin followed by warfarin in patients without contraindication. (Expert opinion) 3. In patients with contraindications they recommend aspirin. (Expert opinion)
Kraus RR	1999	Diagnosis, treatment and outcome of blunt carotid arterial injuries. Am J Surg. 1999;178:190-193	III	**Design:** Retrospective review of 16 patients with blunt carotid artery injuries.

				Findings: 1. Limited (n=5), although favorable experience with Duplex scan. 2. Anticoagulation had better outcome than observation or therapeutic coiling **Recommendations:** 1. Anticoagulation is recommended for the treatment of BCAI in patients without contraindications.
Lévy C	1994	Carotid and vertebral artery dissections: Three-dimensional time-of-flight MR angiography and MR imaging versus conventional angiography. Radiology. 1994;190:97-103	II	**Design:** Prospective protocol in which MRI versus MRA was evaluated in angiographically confirmed BCVI in 18 patients. **Findings:** 1. MRA found to be superior to MRI with sensitivity of 95% and specificity of 99% overall. 2. For vertebral injuries specifically MRA was only 60% sensitive. **Recommendations:** 1. MRA is a reliable noninvasive method for use in the diagnosis and follow-up of extracranial internal carotid artery dissection. 2. Conventional angiography is recommended in the assessment of VAI. 3. Conventional MRI is not as sensitive as MRS for the evaluation of BCVI.
Lew SM	1999	Pediatric blunt carotid injury: a review of the national pediatric trauma registry. Ped Neurosurg. 1999;30:239-244	III	**Design:** Review of the National Pediatric Trauma Registry and thorough review of the adult literature. **Findings:** 1. There is an increased incidence of BCVI in children with chest trauma, combined head & chest trauma, basilar skull fractures, intracranial hemorrhage, and clavicle fractures. 2. Children & adults are similarly susceptible to the adverse sequellae of BCVI.

				3. Incidence of BCVI was found to be 0.03% (15/57,659). 40% (6) had neurologic complication. **Recommendations:** 1. Screen, diagnose, and treat children for BCVI similarly to adults.
Louw JA	1990	Occlusion of the vertebral artery in cervical spine dislocations. J Bone Joint Surg Br. 1990;72:679-681	II	**Design:** All patients with cervical spine facet dislocations were evaluated for BVAI with angiography. **Findings:** 1. Incidence of vertebral artery occlusion in patients with unilateral facet dislocation was 80%. 2. Incidence with bilateral dislocation was 71.4%. 1 of 5 had bilateral occlusions. **Recommendations:** 1. Vertebral artery angiography is indicated in all patients with neurological deficit above the level of potential spinal cord injury. 2. Evaluation of the vertebral arteries should be considered in all patients with cervical facet dislocation.
Martin RF	1991	Blunt trauma to the carotid arteries. J Vasc Surg. 1991;14:789-795	III	**Design:** Retrospective review of 8 patients over 10 years. **Findings:** 1. 4 patients were found incidentally when screening for aortic injury with angiography. 2. 3 patients were treated operatively, two which had asymptomatic occlusion of the CCA and one had recurrent TIA. All had complete neurologic recovery or remained asymptomatic. 3. Only one patient was treated with heparin and did not develop symptoms. 4. 4 patients were observed without specific treatment one of which had a persistent dense hemiplegia.

				Recommendations: 1. Angiography is recommended for screening in patients being screened for possible aortic injuries. 2. Angiography is recommended if neurological exam does not correlate with intracranial findings. 3. Surgery is recommended for those with accessible lesions. (Expert opinion) 4. Anticoagulation (unless contraindicated) is recommended for small intimal lesions. (Expert opinion)
Mayberry JC	2004	Blunt carotid artery injury. The futility of aggressive screening and diagnosis. Arch Surg. 2004;139:609-613	III	**Design:** Retrospective multi-institutional review. **Findings:** 1. Incidence of BCVI was found to be 0.05%. 2. Only 2 of 11 patients who had sequellae of BCVI presented in a delayed fashion and only one of these had risk factors that might have prompted screening. 3. 6 were found incidentally and none of these patients developed an ischemic event. **Recommendations:** 1. A cost benefit analysis should be done before trauma surgeons accept an aggressive screening protocol as the standard of care.
McKevitt EC	2002	Blunt vascular neck injuries: diagnosis and outcomes of extracranial vessel injury. J Trauma. 2002;53:472-476	III	**Design:** Retrospective review of 22 patients identified over 8 years. **Findings:** 1. 31 BCVI were identified in 22 patients for an overall incidence of 0.075%. 2. 8/12 patients with occult injuries developed subsequent stroke in a delayed fashion. 3. 25% of the occult injuries died secondarily (in whole or part) due to the vascular injury. 4. Multivariate analysis identified GCS $\leq$ 8 and thoracic AIS $\geq$ 3 as risks for BCVI.

				Recommendations: 1. Patients with risk factors for BCVI should undergo screening.
McKevitt EC	2002	Identifying patients at risk for intracranial and extracranial blunt carotid injuries. Am J Surg. 2002;183:566-570	III	**Design:** Retrospective review of British Columbia trauma registry to identify injury patterns that increase risk of BCAI, looking at both extracranial arterial injury (ECAI) and intracranial arterial injuries (ICAI). **Findings:** 1. 35 carotid injuries were identified in 28 patients (over 8 years). Incidence was 0.09 % of blunt trauma admissions. 18 had ECAI, and 10 with ICAI, 7 bilateral injuries. 2. 56% of ECAI and 11% ICAI were occult. 3. Independent risk factors for ECAI were high ISS, GCS $\leq$ 8, and thorax AIS $\geq$3. 4. Independent risk factors for ICAI GCS $\leq$ 8 and facial fracture. 5. All patients with ICAI did poorly and were often symptomatic on initial presentation. **Recommendations:** 1. Limited screening resources should focus on risk factors for occult extracranial injury: namely, low GCS and significant thoracic trauma as ECAI is more likely to benefit from identification prior to the onset of symptoms.
Miller PR	2001	Blunt cerebrovascular injuries: Diagnosis and treatment. J Trauma. 2001;51:279-286	III	**Design:** Retrospective review. **Findings:** 1. Stroke rates vary with injury type in untreated patients: a. dissection 14% b. occlusion 90% c. carotid-cavernous fistula 67% d. pseudoaneurysm 50% 2. Patients Treated with heparin had better outcomes in CAI and VAI (stroke rate). 3. Pts treated with ASA (aspirin) had worse

				outcome in terms of discharge Glasgow Outcome Score. 4. Pseudoaneurysms do not improve with or without anticoagulation or antiplatelet agents **Recommendations:** 1. Patients with BCVI other than pseudoaneurysm should be treated with anticoagulation.
Miller PR	2002	Prospective screening for blunt cerebrovascular injuries. Analysis of diagnostic modalities and outcomes. Ann Surg. 2002;236:386-395	II	**Design:** Prospective evaluation of a screening protocol for BCVI. **Findings:** 1. Aggressive screening of patients with blunt head & neck trauma identifies an incidence of BCVI in 1.03% of blunt admissions. 2. Early identification & treatment significantly reduces stroke rates in patients with VAI but not with CAI. **Recommendations:** 1. CTA & MRA are inadequate for screening; conventional angiography remains the standard for diagnosis. 2. Aggressive screening for BCVI is indicated to identify patients prior to the development of symptoms. 3. Treatment of patients with asymptomatic BCVI is indicated to prevent progression to INE.
Mutze, S	2005	Blunt CVI in patients with blunt multiple trauma: Diagnostic accuracy of duplex doppler US and early CTA	II	**Design:** Prospective observational study. **Findings:** 1. Accuracy of Duplex US for detection of BCVI (clinical f/u) was only 38.5% if you include deaths as having BCVI (worst case scenario) it went down to 12.2%. 2. CTA with 4 slice scanner had a sensitivity of 100% and specificity of 98.6% (worst case including early deaths as having BCVI decreased sensitivity to 73.3%.

				Recommendations: 1. Duplex US is not useful in the diagnosis of BCVI. 2. CT angiography with 4 slice scanner is useful in the diagnosis of BCVI.4
Nanda A	2003	Management of carotid artery injuries: LSU Shreveport experience. Surg Neurol. 2003;59:184-90	III	**Design:** Retrospective review of 23 patients with carotid injury of mixed blunt and penetrating etiology. **Findings:** 1. 2/4 patients with ICH treated with heparin had worsening of the ICH. **Recommendations:** 1. BCVI patients with ICH should not be treated with heparin anticoagulation.
Parent AD	1992	Lateral cervical spine dislocation and vertebral artery injury. Neurosurgery. 1992;31:501-509	III	**Design:** Case series of quadriplegic patients with cervical spine injuries, all at C5-C6 with subluxation with some element of lateral vertebral displacement. **Findings:** 1. In 12 patients found to have VA injury over 12 years all were seen to have lateral subluxation on C-spine films. Conclusion lateral subluxation puts you at high risk of VAI. However no numerator ("real incidence" of VAI) or denominator (# with subluxation) is noted. **Recommendations:** 1. Spinal stabilization is recommended early. 2. The artery may need ligation or embolization to prevent ischemic sequellae.
Parikh AA	1997	Blunt carotid artery injuries. J Am Coll Surg. 1997;185:80-86	III	**Design:** A retrospective chart review looking at patients with BCI. **Findings:** 1. Incidence of BCAI was found to be 0.24%. 2. Head + chest injuries increase risk of BCAI by 14X.

				3. Patients who underwent anticoagulation of any type had less morbidity however the complication rate was 40%. **Recommendations:** 1. The combination of head and chest injury should raise the index of suspicion for BCAI. 2. Anticoagulation is indicated as it is associated with the least morbidity.
Perry MO	1980	Carotid artery injuries caused by blunt trauma. Ann Surg, 1980;192:74-77	III	**Design:** Retrospective case series of 17 patients described with blunt carotid injury. **Findings:** 1. Morbidity and mortality lower with surgical repair v. observation, especially those with prograde flow and only mild neurological deficit. **Recommendations:** 1. Repair of the injured vessel is safe and effective in patients with carotid injuries in whom prograde flow continues and there is only mild neurologic deficit present. 2. Repair is not indicated if there is complete occlusion, severe neurologic deficit and altered consciousness.
Prall JA	1998	Incidence of unsuspected blunt carotid artery injury. Neurosurgery. 1998;42:495-499	II	**Design:** Prospective observational study in which patients that were to have thoracic aortography also underwent angiography of the neck for identification of occult BCAI. **Findings:** 1. The incidence of asymptomatic BCAI in 119 patients screened was 2.5%. Overall BCAI in patients undergoing aortogram (symptomatic and not) BCI incidence was 3.5%, and .32% in all blunt trauma patients. 2. All patients identified prior to symptoms were anticoagulated and none developed symptoms 3. 6/7 patients with BCAI not identified by

				screening developed a delayed neurologic event **Recommendations:** 1. All patients undergoing evaluation for blunt aortic injury should be screened for BCAI 2. Patients with asymptomatic BCAI should be anticoagulated to prevent INE.
Punjabi AP	1997	Diagnosis and management of blunt carotid artery injury in oral and maxillofacial surgery. J Oral Maxillofac Surg. 1997;56:1388-1396	III	**Design:** Retrospective review of 10 patients with BCAI – all patients had symptoms at presentation. **Findings:** 1. Symptomatic BCI is found in 0.31% of blunt trauma patients and 1.2% of CHI patients 2. Presentations associated with BCAI are: **a.** neck tenderness/hematoma **b.** ipsilateral Horner's syndrome **c.** TIA **d.** focal neurologic deficit **e.** progressive limb paresis 3. Duplex U/S missed 2/3 injuries in which it was used to screen for BCAI. 4. Anticoagulation was associated with improved neurologic outcome. **Recommendations:** 1. All patients undergoing evaluation for blunt aortic injury should be screened for BCAI 2. Anticoagulation is recommended as the most beneficial therapeutic modality.
Rogers FB	1999	Computed tomographic angiography as a screening modality for blunt cervical arterial injuries: preliminary results. J Trauma. 1999;43:280-385	II	**Design:** Retrospective review of prospectively collected data before and after a screening protocol using CTA was instituted. The type of CT scanner was not identified. CTA not used consistently and patients with negative CTA received only clinical follow-up (no angiography). **Findings:** 1. The use of CTA increased the detected incidence of BCAI from 0.06% to 0.19% 2. 23% of patients who were found to have

				BCAI presented without neurologic deficit. 3. There was a 16% complication rate (2/12 patients) related to anticoagulation. 4. No patient with negative CTA developed stroke. 5. No patients went on to completed stroke in the post CTA group. **Recommendations:** 1. The inclusion of CTA in the admission work-up for patients at risk for BCAI is safe and effectively increases the frequency of diagnosis of this injury.
Rozycki GS	2002	A prospective study for the detection of vascular injury in adult and pediatric patients with cervicothoracic seat belt signs. J Trauma. 2002;52:618-624	II	**Design**: Prospective, non-randomized study of 131 patients with seat-belt signs after blunt trauma. 4 patients found with BCVI. **Findings**: 1. Cervical or thoracic seat belt sign is associated with a 3% risk of BCVI. **Recommendations:** 2. The presence of a cervical or thoracic seat belt sign should raise the suspicion for the presence of occult vascular injury and in the presence of an abnormal physical exam should prompt diagnostic evaluation.
Schneidereit NP	2006	Utility of screening for blunt vascular neck injuries with computed tomographic angiography.	II	**Design:** Prospective, non-randomized study of a CTA screening protocol utilizing an 8-slice CT scanner. Negative studies did not undergo confirmatory conventional angiography. 10 of 33 abnormal scans also did not undergo confirmatory conventional angiography; four of these were treated based on the CTA alone, 3 patients were thought to have a false positive CTA and were followed with observation, and 3 had minimal injury to a vertebral artery and were followed with observation. **Findings:** 1. The incidence of BCVI as detected by 8-slice CTA was 1.1% (as confirmed by DFCA) or

				1.4% based on CTA alone (including those 4 patients treated based on the CTA alone). 2. The incidence of delayed stroke rate and injury spec mort went down from 67% to 0% and 38% to 0% from pre to post-screening period. 3. Of the 23 confirmatory angiograms, 8 were found to be falsely positive. 4. The only significant predictor of BCVI by linear regression analysis was cervical spine injury. **Recommendations:** 1. Liberal screening utilizing 8-slice CTA is recommended to identify BCVI prior to neurologic event.
Wahl WL	2002	Antiplatelet therapy: an alternative to heparin for blunt carotid injury. J Trauma. 2002;52:896-901	III	**Design:** Retrospective registry review of 22 patients diagnosed with BCAI. 7 of which were treated with heparin and 7 treated with antiplatelet agents **Findings:** 1. There was no difference in neurologic outcome between groups. 2. Heparin-treated patients had significantly higher bleeding risk (4 patients had bleeding complication of heparin v. none on antiplatelet agents). **Recommendations:** 3. Use antiplatelet therapy in patients who are at high risk for bleeding complications from either intracranial or major torso injuries, and reserve heparin for those with fairly isolated blunt carotid injuries not amenable to surgical intervention or if crescendo neurologic symptoms are present.
Weller SJ	1999	Detection of vertebral artery injury after cervical spine trauma using magnetic resonance angiography. J Trauma. 1999;46:660-666	II	**Design:** Prospective non-randomized review of a screening protocol utilizing MRI/MRA for the detection of BVAI in patients with evidence of cervical fracture or dislocation.

				Findings: 1. Of 38 patients with cervical spine injury there were 4 vertebral artery injuries identified. 2. All BVAI were associated with fracture through the ipsilateral foramen transversarium. 3. All patients found to have BVAI were treated initially with antiplatelet agents and 2 were systemically anticoagulated. 4. No patient developed INE. **Recommendations:** 1. Patients with cervical spine injury, particularly fracture through the foramen transversarium are at high risk for BVAI and should undergo diagnostic evaluation for this injury.
Willis BK	1994	The incidence of vertebral artery injury after midcervical spine fracture or subluxation. Neurosurgery. 1994;34:435-442	II	**Design:** Prospective non-randomized observational study in which all patients presenting with an unstable cervical spine injury or fracture through the foramen transversarium underwent angiography of the vertebral arteries. **Findings:** 1. In this population the incidence of BVAI was found to be 46%. 2. No patient had neurologic dysfunction secondary to the BVAI. 3. Patients with non-occlusive injury (3) were treated with anticoagulation. 2 went on to heal on this therapy. 1 patient had an enlarging pseudoaneurysm and was converted to aspirin with subsequent healing. **Recommendations:** 1. Vertebral angiography should be considered before surgical reduction & stabilization in patients sustaining significant subluxation

				(more than 1 cm) or fracture involving the lateral masses or foramen transversarium, particularly when a comminuted fracture of the foramen transversarium is present.
Woodring JH	1993	Transverse process fractures of the cervical vertebrae: are they insignificant? J Trauma. 1993;34:797-802	III	**Design:** Retrospective chart review of 216 patients with cervical spine injury of which 8 were found to have transverse process fractures into the vertebral foramen. **Findings**: 1. Of the 8 patients with fracture into the vertebral foramen who underwent angiography 7 had BVCAI. 2. 2 of these seven had clinical evidence of vertebral-basilar artery stroke. 3. No difference in outcome between those 4 of the 7 patients who were anticoagulated versus the 3 were not. **Recommendations:** 1. Albeit the incidence of BVAI in patients with fracture into the vertebral foramen is 88%, in light of the low incidence of symptomatic injury the authors recommend reserving angiography for patients with symptoms of vertebral-basilar artery stroke.
York G	2005	Association of internal carotid artery injury with carotid canal fractures in patients with head injury	III	**Design:** Retrospective review of patients found to have carotid canal (CC) fracture who subsequently underwent cerebral angiography within 7 days. **Findings**: 1. The presence of CC fracture had a sensitivity, specificity, PPV, and NPV of 60%, 67%, 35%, and 85% for the detection of BCAI respectively. 2. This was not significantly better than other CT findings not typically associated with BCAI such as cerebral contusion or sphenoid air-fluid level. **Recommendations:**

				1. CC fracture is not useful as a single risk factor for the identification of BCAI.