Notable Grand Rounds
of the
Michael & Marian Ilitch Department of Surgery

Wayne State University
School of Medicine

Detroit, Michigan, USA

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UPDATE ON BLUNT CEREBROVASCULAR INJURIES (BCVI)

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About Notable Grand Rounds

These assembled papers are edited transcripts of didactic lectures given by mainly senior residents, but also some distinguished attending and guests, at the Grand Rounds of the Michael and Marian Ilitch Department of Surgery at the Wayne State University School of Medicine.

Every week, approximately 50 faculty attending surgeons and surgical residents meet to conduct postmortems on cases that did not go well. That “Mortality and Morbidity” conference is followed immediately by Grand Rounds.

This collection is not intended as a scholarly journal, but in a significant way it is a peer reviewed publication by virtue of the fact that every presentation is examined in great detail by those 50 or so surgeons.

It serves to honor the presenters for their effort, to potentially serve as first draft for an article for submission to a medical journal, to let residents and potential residents see the high standard achieved by their peers and expected of them, and by no means least, to contribute to better patient care.

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Update on Blunt Cerebrovascular Injuries (BCVI)

A Notable Grand Rounds Presentation
Wayne State University School of Medicine

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This paper has been adapted from an oral presentation.

Blunt Cerebrovascular Injuries (BCVI)

In the 1990s, blunt cerebrovascular injuries were thought to have unavoidable devastating neurologic outcomes, but over the last 30 years, new understanding has led to early screening, diagnosis, and preemptive antithrombotic management.

Incidence, Types, & Screening

BCVI is diagnosed in 1% to 3% of blunt trauma admissions. Historically BCVI was thought to be more rare (less than 0.1%) and the neurologic sequelae were attributed to primary head injury. Once BCVI was recognized as a vascular source, its identification tripled. Most patients become symptomatic more than 12 hours after injury. Early identification thus leads to a therapeutic window of opportunity for treatment before stroke.

There are two types of BCVI: Carotid artery injuries (CAI) and vertebral artery injuries (VAI). CAI have an average untreated stroke rate of 50%, increasing in proportion to injury grade. VAI have an untreated stroke rate 20-25%. The anatomy of the cerebrovascular vessels and mechanism of hyperextensions

with contralateral rotations effect on the carotid and vertebral artery are shown in **Figs. 1a and 1b**.

Screening protocols based on injury patterns and mechanisms of injury identify asymptomatic patients to initiate treatment prior to neurologic sequelae. Early antithrombotic therapy reduces stroke rate and prevents neurologic morbidity.

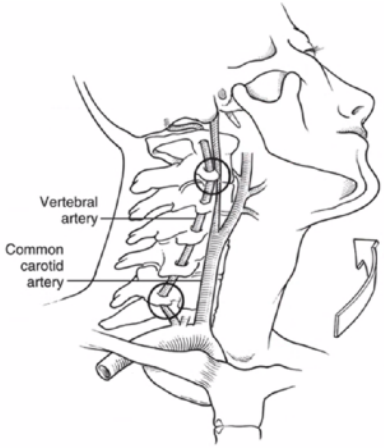

Fig. 1a. Anatomy of the cerebrovascular vessels

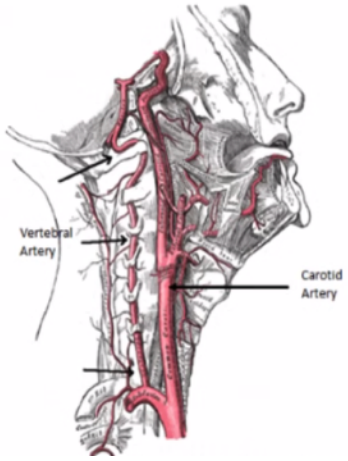

Fig. 1b. Mechanism of hyperextensions

Objectives

This paper aims to discuss BCVI as it is understood and treated today; specifically:

- Signs and symptoms
- Diagnostic imaging
- Injury grading scale
- Incidence and treatment

The 2020 EAST guidelines for the evaluation and management of blunt cerebrovascular injury and the 2021 AAST systemic review of the treatment of asymptomatic BCVI are discussed. An appendix provides an illustrative case study.

Mechanism of Injury

Three mechanisms may be involved in BCVI: (1) A direct blow to the neck, (2) hyperextension with contralateral rotation, and (3) direct injury to the artery from fracture of sphenoid or petrous bones. (**Fig. 2**)

Hyperextension with contralateral rotation is the most common cause of CAI, resulting in the stretching of the carotid artery over the lateral articular process of C1-C3. Direct blows to the neck may occur from sports activities or from a motor vehicle crash, in which case the patient may have incorrectly positioned the seatbelt and presents with seatbelt sign on the neck.

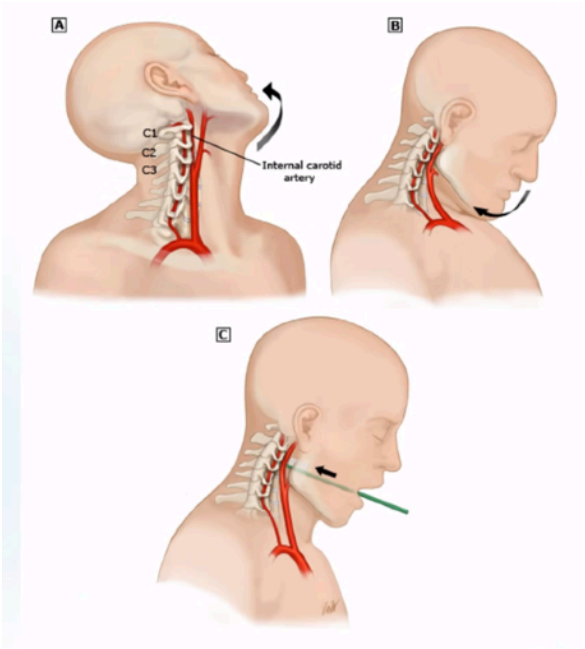

Fig. 2. Mechanism of BCVI. (A: Cervical hyperextension with contralateral rotation; B: Cervical hyperflexion directly compressing the carotid artery between the mandible and the upper cervical vertebrae causing intimal disruption or hematoma; C: Falling with a hard object [e.g., a tooth- brush] in the mouth, injuring the internal carotid artery at the angle of the jaw.

Regardless of the injury mechanism, there is an intimal disruption of the carotid or the vertebral artery. The intimal tear exposes subendothelial collagen and becomes a nidus for platelet aggregation that may lead to thrombosis or emboli and stroke. The disruption can also cause sub-intimal dissection, luminal narrowing, acute occlusion, or pseudoaneurysm (causing compression or a nidus for embolization). The highest grade is a free rupture, causing intracranial or extracranial hemorrhage or development of arteriovenous fistula.

The majority of injuries are at the base of the skull and are difficult to identify on imaging.

Signs and Symptoms of BCVI

Symptoms resemble a stroke and may depend highly on the patient's pre-existing physiology, collateral circulation (an incomplete Circle of Willis) and underlying cerebrovascular disease.

CAI typically present with aphasia if the dominant hemisphere is involved, or hemineglect if the non-dominant hemisphere is affected. Symptoms of VAI can be more subtle: They may include ataxia, dizziness, vomiting, facial or body analgesia, and visual field defects. A rare third type of BCVI—carotid-cavernous fistula (**Box 1**, top right)—may present with orbital pain, exophthalmos, chemosis, and conjunctival hyperemia.

Signs of BCVI are active arterial hemorrhage from the neck, mouth, nose, or ear; expanding cervical hematoma; cervical bruit in a patient <50 years old, and focal or lateralizing neurological defects.

While some patients may show symptoms within an hour, most have a latent period between 12 to 75 hours. Rarely, it can extend to years. The physician's goal should be to diagnose BCVI during the "silent period" prior to the onset of stroke.

Box 1: Carotid Cavernous Fistula

The carotid-cavernous fistula (CCF) is a rare abnormal connection between the carotid artery and/or its branches and a large vein called the cavernous sinus. The cavernous sinus is located behind the eye.

A direct CCF often occurs days or weeks after a closed head injury. Patients present with the classic triad of chemosis (red eye syndrome), pulsatile exophthalmos (abnormal protrusion of the eyeball), and ocular bruit (blood flow sounds coming from the eye). Proptosis, diplopia, and visual loss may result with these fistulas. (See **Fig. 3 below**.)

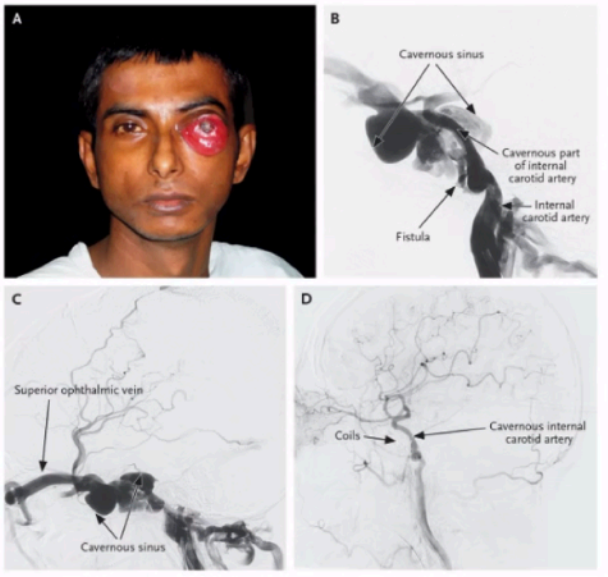

Fig. 3. Signs of carotid-cavernous fistula *(see Box 1)* in a 26 year-old male patient with a history of blunt trauma to the head two months earlier. He presented with rapidly progressing eye swelling, pain and diminished vision in the left eye (A). The carotid fistula (B) affects the cavernous sinus The eye swells because the superior ophthalmic vein (C) behind the eye drains into the cavernous sinus.

The current screening algorithm, which includes patients considered high risk based on their injury pattern and patients with associated injuries, captures between 80-95% of all BCVI; however, clinical suspicion should be used to screen for at-risk patients based on their mechanism.

Ongoing studies advocate for including all patients with cervical spine fractures or high energy transfer mechanism based upon the high morbidity risk of a missed injury compared to the low morbidity risks of imaging.

Denver Screening Criteria

The Denver Screening Criteria for Blunt Cerebrovascular Injuries (**Table 1,** opposite page) are the prevalent screening criteria.

Diagnostic Imaging

Historically, four-vessel digital subtraction arteriography (DSA) was the gold standard. It was labor intensive and therefore unavailable at smaller hospitals. It has since been replaced by computed tomographic arteriography (CTA), which reduces time to BCVI identification and permits earlier treatment. Studies from Harborview and Medical College of Virginia using 16 slice CTA demonstrated concordance with DSA confirmatory tests.

The optimal CTA scanner is 64 slice or greater. The resolution of 1-4 slice CTA is too poor for diagnosing BCVI. Magnetic resonance angiography (MRA) has too low sensitivity and specificity for BCVI and takes too long to obtain. Ultrasound (US) cannot visualize the base of the skull, the patient may be wearing a cervical collar limiting usefulness, and is operator-dependent.

DSA is required if suspicion is high yet noninvasive testing is negative or equivocal.

Repeat CTA imaging for BCVI should be carried out at 7-10 days for change in neurologic status. The reason is that half of Grade I BCVI patients completely heal and can stop antithrombotic therapy. In contrast, fewer than 10% of Grade II, III, IV patients heal, and 12% have an injury progression .

Injury Grading Scale and Rate of Stroke

Varied luminal irregularities comprise BCVI, including dissection, occlusion, transection, and pseudoaneurysms. They are graded according to the Denver grading scale (**Table 2**, below).

Grade 1	Irregularity of the vessel wall or a dissection/intramural hematoma with less than 25% luminal stenosis
Grade 2	Intraluminal thrombus or raised intimal flap is visualized, or dissection/ intramural hematoma with 25% or more luminal narrowing
Grade 3	Pseudoaneurysm
Grade 4	Vessel occlusion
Grade 5	Vessel transsection

Table 2. Denver Grading Scale

Figs. 4 and 5 (p. 6) show two imaging modalities that illustrate the various grades.

Untreated injuries have an overall stroke rate of 20%. The stroke rate for CAI increases proportionately with increasing injury grade, whereas the rate for VAI has a more consistent stroke rate of 20%. (**Table 3**, p. 7))

Treatment & Therapy

If the injury occurs in a surgically accessible area of the carotid artery, operative management via resection and primary end-to-end anastomosis is considered if the distal extent of the injury-associated thrombus can

Signs/symptoms of BCVI	Arterial hemorrhage
	Cervical bruit in patient > 50 years of age
	Expanding cervical hematoma
	Focal neurologic deficit
	Neurologic examination incongruous with head CT scan findings
	Stroke on secondary CT scan
Risk factors for BCVI	High-energy transfer mechanism associated with:
	Displaced mid-face fracture (LeFort II or III)
	Mandible fracture
	Complex skull fracture basilar skull fracture/occipital condyle fracture
	Severe TBI with GCS < 6
	Cervical spine fracture, subluxation, or ligamentous injury at any level
	Near hanging with anoxic brain injury
	Clothesline-type injury or seat belt abrasion with significant swelling, pain, or altered MS TBI with thoracic injuries
	Scalp degloving
	Thoracic vascular injuries
	Blunt cardiac rupture
	Upper rib fractures

Table 1. Denver Screening Criteria for Blunt Cerebrovascular Injuries BCVI: Blunt cerebrovascular injury; CT: computed tomography; MS: mental status; TBI: traumatic brain injury.) *Courtesy* Denver Health Medical Center.

be controlled. However, operative intervention is rare because most are high within the carotid canal at the base of the skull or in the foramen transversarium (transverse process of the cervical vertebra that contains the vertebral vessels) (**Fig. 6**, p. 7).

The morbidity and mortality associated with untreated BCVI make expectant management an unacceptable strategy unless significant contraindications to antithrombotic therapy exist.

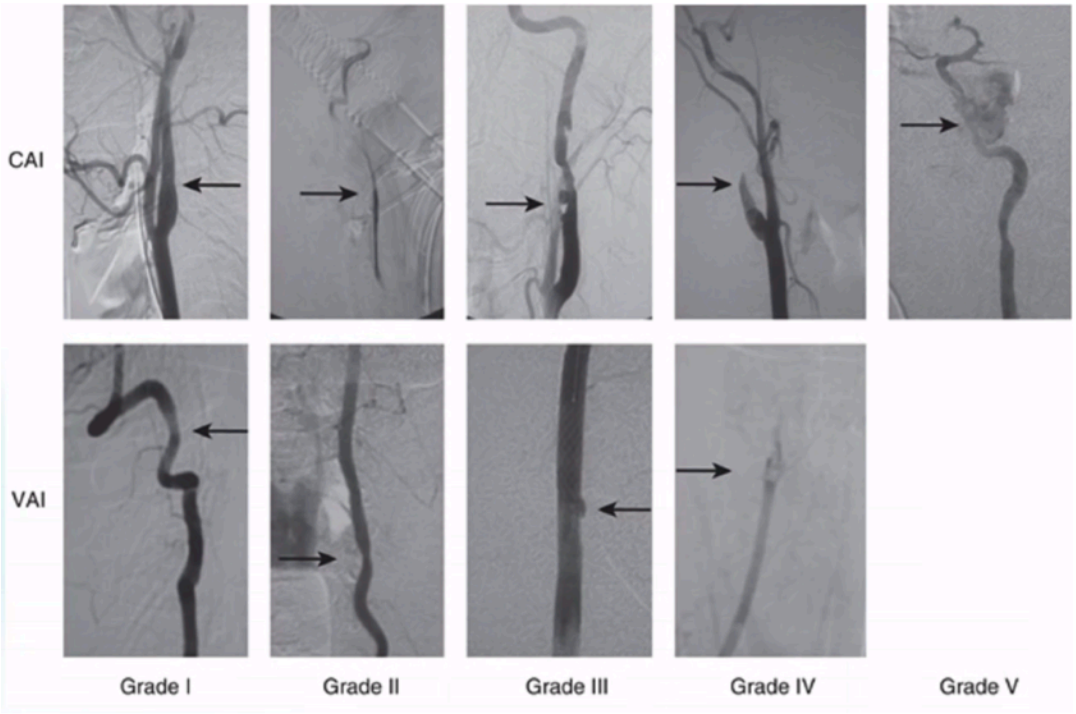

Fig. 4. DSA illustrating BCVI grades I -V. Arrows denote the injury location.

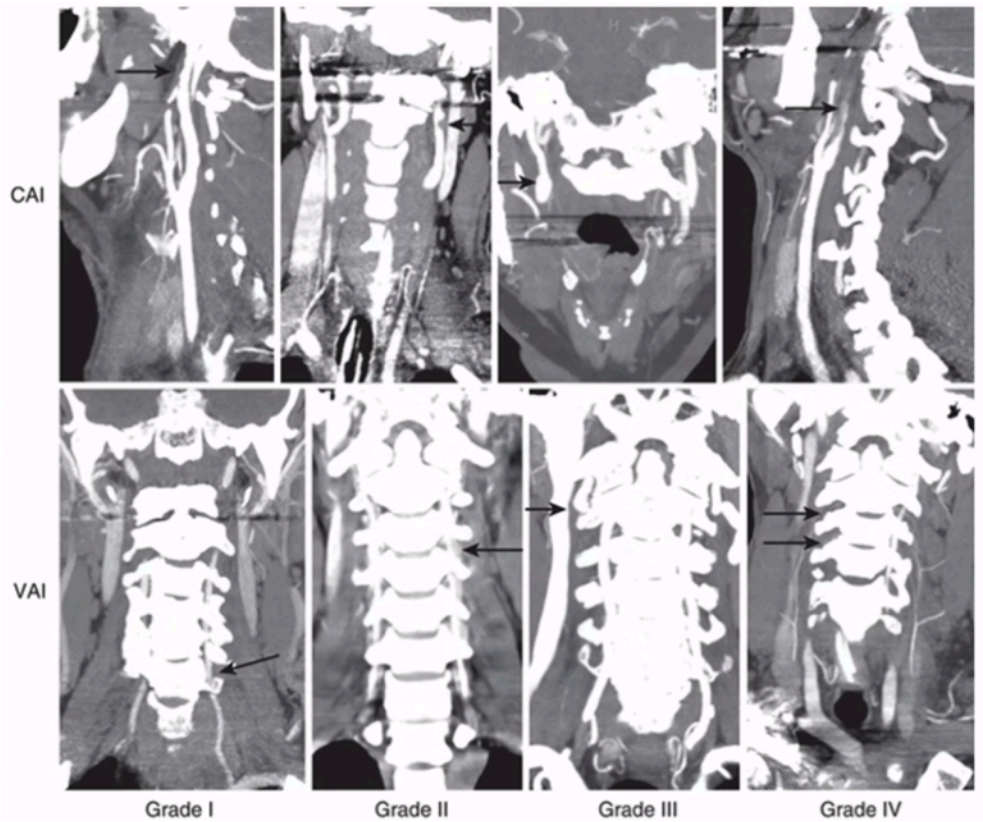

Fig. 5. CTA illustrating BCVI grades I -IV. Arrows denote the injury location.

Injury	Grade of Injury	Stroke Rate (%)	
Carotid artery injury	I	3	
	II	14	
	III	26	
	IV	50	
	V	100	
Vertebral artery injury	I	6	
	II	38	
	III	27	
	IV	28	
	V	100	

Table 3. Stroke rate by BCVI injury grade

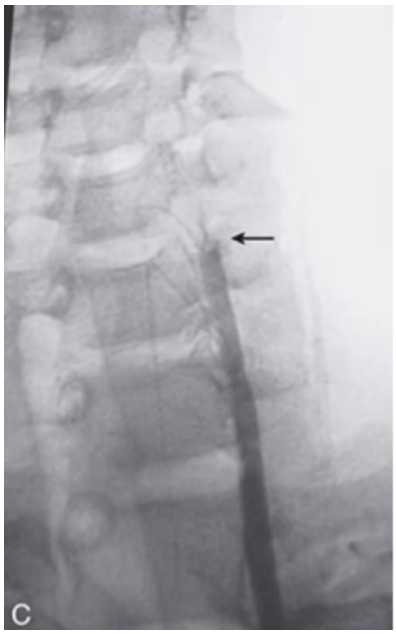

Fig. 6. Vertebral artery occlusion in the foramen transversarium

Endovascular stents are not a main treatment but are sometimes used in individual cases with symptoms from severe flow-limiting stenosis, AV fistula, enlarging pseudoaneurysm, and as a salvage maneuver for Grade V injuries.

Antithrombotic Treatment

In earlier Western Trauma studies, patients treated with anticoagulation had much improved outcomes compared to those not treated. 45% had good neurologic status and anticoagulation was independently associated with survival and improvement in neurologic outcome.

In the acute phase, intravenous (IV) heparin is administered at 15 U/kg/hr, no bolus, titrated to partial thromboplastin time of 40-50 seconds. However, there are subtle but important nuances in trauma cases. If the patient has a closed head injury or intraparenchymal hemorrhage, neurosurgery must be consulted regarding heparin. Treatment in patients with pelvic fractures and retroperitoneal hemorrhage should not be started until coagulation status is normal, physiologic stability is achieved, and transfusion is not required. IV heparin can always be held or reversed if necessary.

There is some current controversy regarding the ideal antithrombotic therapy—whether it is anticoagulation or antiplatelet agents. The latter are easier to administer but some recent research favors anticoagulant agents especially in higher grade lesions.

Thromboelastography (**TEG**) testing can demonstrate variable platelet response and platelet mapping may be an effective test to assess treatment in individual patients.
A Memphis study of polytrauma patients found no worsening of brain injuries or pro-

gression of solid organ injuries compared to operative intervention. A Houston group had similar findings. This suggests that early therapeutic antithrombotic therapy can be safe in polytrauma patients. These studies are retrospective (prospective studies would clearly be unethical since it is known that patients who are diagnosed with BCVI early and treated with antithrombotic agents almost universally avoid ischemic neurologic events).

Treatment duration is usually 6 months, a somewhat arbitrary duration based on an assumption of re-endothelialization. Long term studies are lacking but persistent injuries on repeat CTA may be treated with lifelong aspirin.

Evaluation and Management of BCVI

The Denver Health Medical Center published the BCVI screening algorithm shown in **Fig. 7** (opposite page) in 2011.[1]

A practice management guideline from the Eastern Association for the Surgery of Trauma (EAST)[2] identified 4 "PICO" questions: (P) population, (I) intervention, (C) comparator and (O) outcome. It used the GRADE methodology[3] to evaluate evidence. Data from studies that were relevant to each question underwent meta-analysis. Outcomes were stroke, mortality, length of stay, bleeding, infection, post-procedural complications, contrast-induced nephropathy, hospital costs, worsening of bleeding, need for delayed operative intervention, detection of injury, missed injury, and readmission.

PICO 1: In adult patients with blunt polytrauma, should a screening protocol vs no screening protocol be used to detect BCVI2?

Six retrospective studies looked at implementation of an institutional BCVI screening protocol on the detection of BCVI. One was the 1998 Biffl study[4] which found that the incidence of detection increased from 0.1% 0.86% with screening protocols. A 2001 study by Kerwin liberalized screening of patients at high risk for BCVI and found an incidence of 1.1%, with 91% of all BCVIs detected.

The resulting **strong recommendation** was to use a screening protocol for the detection of BCVI in adult patients with blunt polytrauma.

PICO 2: In adult patients with high-risk cervical spine injuries should a screening CTA be performed to detect BCVI2?

High-risk injuries were defined by the Denver Group as upper cervical spine (C1–C3) fractures, subluxation, and cervical spine fractures that extend into the transverse foramen. The resulting **strong recommendation** was to screen with CTA in this group. However, low-risk injuries, defined by the Memphis group as any cervical spine injury, carried only a **conditional recommendation** to screen with CTA in this group, which had an incidence of BCVI of 2-9%, and take individual patient factors into consideration.

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[1] *Source*: Blunt Cerebrovascular Injuries: Screening and Diagnosis - Scientific Figure on ResearchGate. Available from: https://www.researchgate.net/figure/The-Denver-Health-Medical-Center-BCVI-screening-guideline_fig1_330423966 [accessed 9 Aug, 2023]

[2] J Trauma Acute Care Surg. 2020 Jun;88(6):875-887). This is an update from 2010 guidelines.

[3] See the GRADE Handbook at https://gdt.gradepro.org/app/handbook/handbook.html

[4] Biffl WL, Moore EE, Ryu RK, Offner PJ, Novak Z, Coldwell DM, Franciose RJ, Burch JM. The unrecognized epidemic of blunt carotid arterial injuries: early diagnosis improves neurologic outcome. Ann Surg. 1998 Oct;228(4):462-70. doi: 10.1097/00000658-199810000-00003. PMID: 9790336; PMCID: PMC1191517.

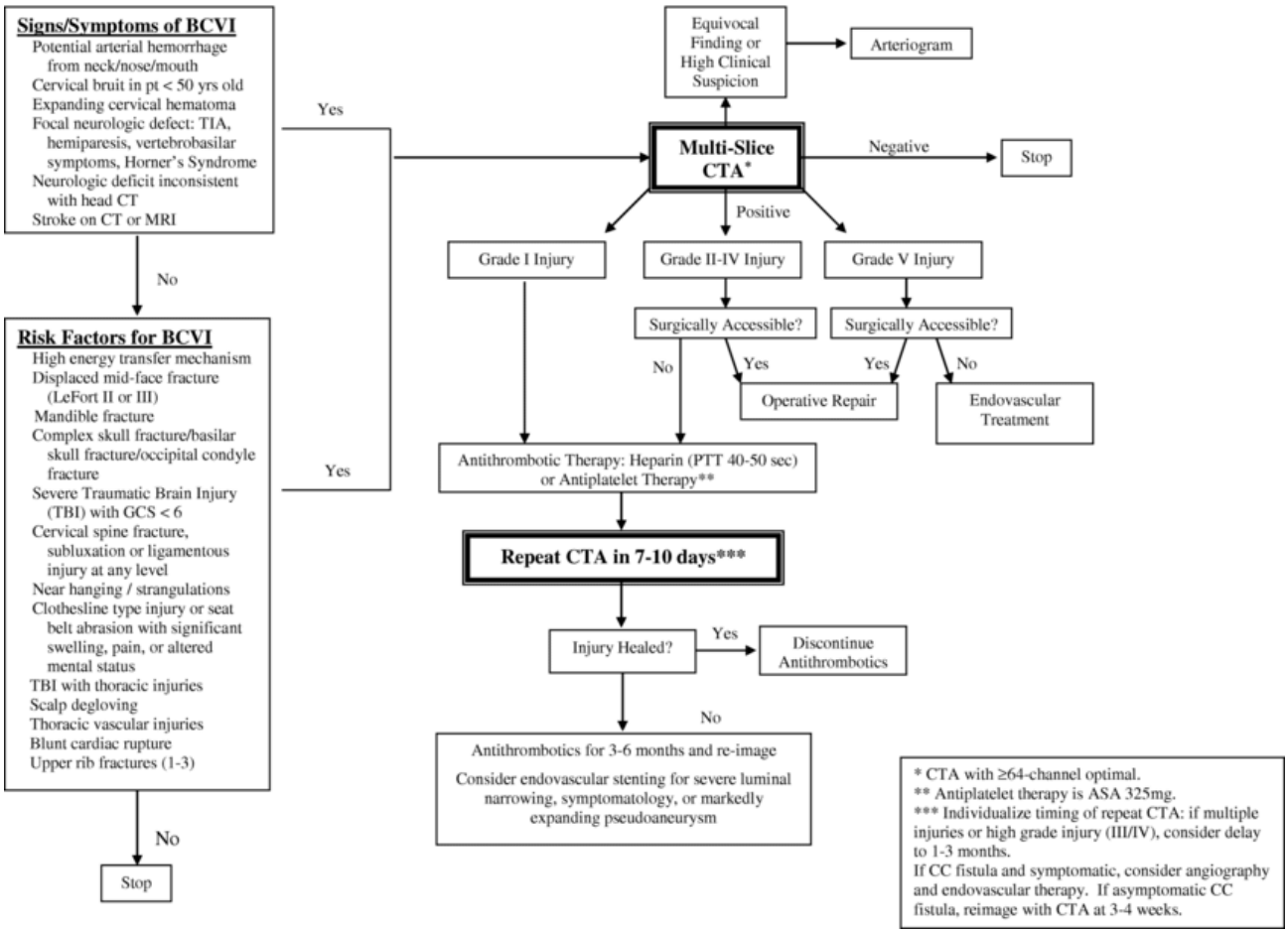

Fig. 7. The Denver Health Medical Center BCVI screening guideline.

PICO 3: In adult patients diagnosed with BCVI, should antithrombotic therapy* vs *no antithrombotic therapy be administered to prevent stroke or mortality?

An analysis of 10 comparative studies resulted in a **strong recommendation** for the use of antithrombotic therapy (the analysis did not differentiate among aspirin, Plavix, or Coumadin) to decrease the incidence of stroke and mortality. It should be started as soon as possible commensurate with safety. There should be a multidisciplinary discussion where patients have concomitant injuries that could exacerbate or worsen bleeding.

PICO 4: In adult patients with a Grade II or Grade III BCVIs, should routine endovascular stenting (as an adjunct to antithrombotic therapy (ATT) vs ATT alone be performed to reduce the risk of stroke or mortality?

Early reports suggested a role for stenting in Grade II injuries with luminal narrowing and Grade III injuries to decrease the risk of embolism or rupture of pseudoaneurysm. More recent studies, however, demonstrated high-risk complications such as in-stent thrombosis leading to increased stroke risk. The result was a **strong recommendation** against routine stenting as an adjunct to ATT in adult patients with Grade II and Grade III BCVI to reduce the risk of stroke.

Practice Management Guidelines

Fig. 8 (next page) illustrates a practice management guideline developed by EAST. It was referenced in an American Association for the Surgery of Trauma (AAST) systemic review published in 2021[5] whose objective was to determine if a specific treatment resulted in lower stroke rates and improved vessel healing in asymptomatic BCVI. Study eligibility criteria were studies that reported a comparison of any treatment for BCVI and stroke or vessel healing rates. The main outcomes were stroke and healing rates.

The review found that major trauma center guidelines recommend antithrombotic treatment but evidence regarding specific agents, dose, and duration had not been established. (The study was not a meta-analysis—it was reported results based on specific treatment modalities.)

It concluded that any medical treatment (ASA, Plavix, heparin, warfarin) was likely better than no treatment for the prevention of stroke, but noted the paucity of high-quality evidence to guide treatment choice or promote vessel healing. Many patients in the studies reviewed received no treatment either because TBI precluded antithrombotic therapy or their injuries were so devastating that the focus was on the patient's comfort rather than survival.

Three studies addressed vessel healing, most often in Grade I injuries. Their low rate of stroke and high rate of vessel healing suggested a need for more nuanced assessment of risks and plan for antithrombotic therapy.

Conclusions

The diagnosis and treatment of BCVI have evolved over the past three decades. Stroke rates decreased from 37% (1980s) to 1-5% in 2015 (latest available data) and BCVI is now diagnosed in 1-3% of blunt trauma admissions.

Comprehensive evaluation and screening has resulted in the early diagnosis of BCVI in the asymptomatic period and allowed prompt initiation of treatment. However, there may be opportunities for screening to go further to include all patients with high energy mechanism.

Treatment with antiplatelet agents or anticoagulation reduces BCVI-related stroke. Nevertheless, most evidence is retrospective given the rarity of the condition and well-designed multi-institutional studies will be required to determine "best" treatment and clearly identify which patients have true counterindications to antithrombotic therapy.

* * *

[5] Treatment of asymptomatic blunt cerebrovascular injury (BCVI): a systematic review. J Trauma Surg Acute Care Open. 2021 Apr 26;6(1):e000668. doi: 10.1136/tsaco-2020-000668. eCollection 2021.

Appendix: Case Study of BCVI

A 50 year old male presents after high speed MVA as trauma code. Pt was restrained driver. Car lost control and t-boned another car on I-75 at high-way speeds. Airbags deployed. Questionable LOC. Patient intoxicated.

Pt evaluated in trauma bay:
Vitals stable
Intoxicated, cooperative, GCS 15, no neurologic defects noted
Lungs clear, heart regular
Mild abdominal pain, FAST negative
Chest ray clear, no fractures
Pelvis ray no fractures
Pulses strong and long bones intact, no other injuries
Pt undergoes CT Imaging of Head, Cervical Spine, Chest, Abdomen & Pelvis per trauma protocols

Review of imaging:
CT Head and Cervical Spine (non contrast) - negative
CT abdomen / pelvis shows free fluid, no solid organ injury

Pt has worsening abdominal pain and undergoes operative intervention for small bowel injury.

Next morning (> 24 hrs post accident) - patient complains of dizziness and blurry vision
CT Head - acute stroke
CT Arteriogram - blunt cerebrovascular injury of the vertebral artery

Did the patient meet criteria for screening for BCVI on initial trauma bay evaluation?
If screening would have been done, could his stroke have been prevent-ed?

* * *