BCVI BLUNT CEREBROVASCULAR INJURY

DEFINITION OF BCVI

Blunt cerebrovascular injury or blunt carotid and vertebral injury refers to a spectrum of injuries to the major cerebral vessels (cervical carotids, vertebral arteries) due to blunt trauma.

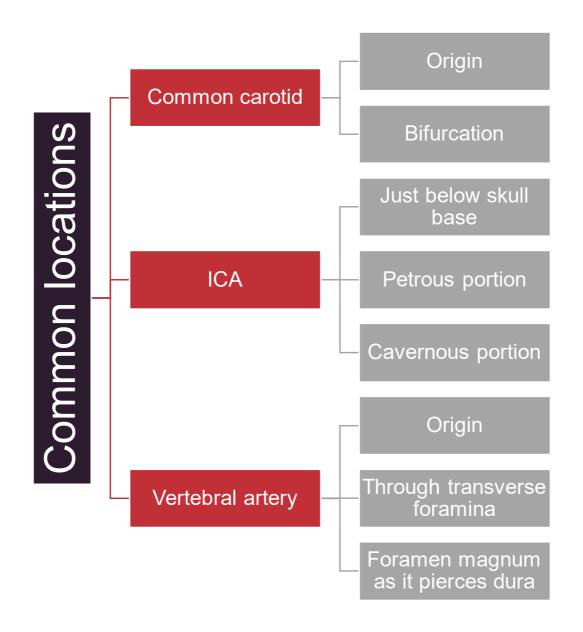
These injuries can occur as a result of polytrauma with a reported incidence of approximately 1-2.7%, although this may be underreported.

PATHOPHYSIOLOGY

BCVI is caused by longitudinal strength of the vessels. This may be due to:

- Acceleration-deceleration causing rotation and hyperextension of the neck
- Stressing the craniocervical vessels
- A direct blow to the neck of base of the skull

BCVI can affect multiple vessels and occurs in typical locations where there is relative fixation.



Denver Criteria

SCREENING FOR BCVI

Signs / Symptoms

- Arterial hemorrhage from neck, nose, or mouth
- Cervical bruit in patient
 50 years old
- Expanding neck hematoma
- Focal neurological deficit
- Neurological deficit incongruent with CT imaging
- Stroke on CT or MRI

Risk Factors

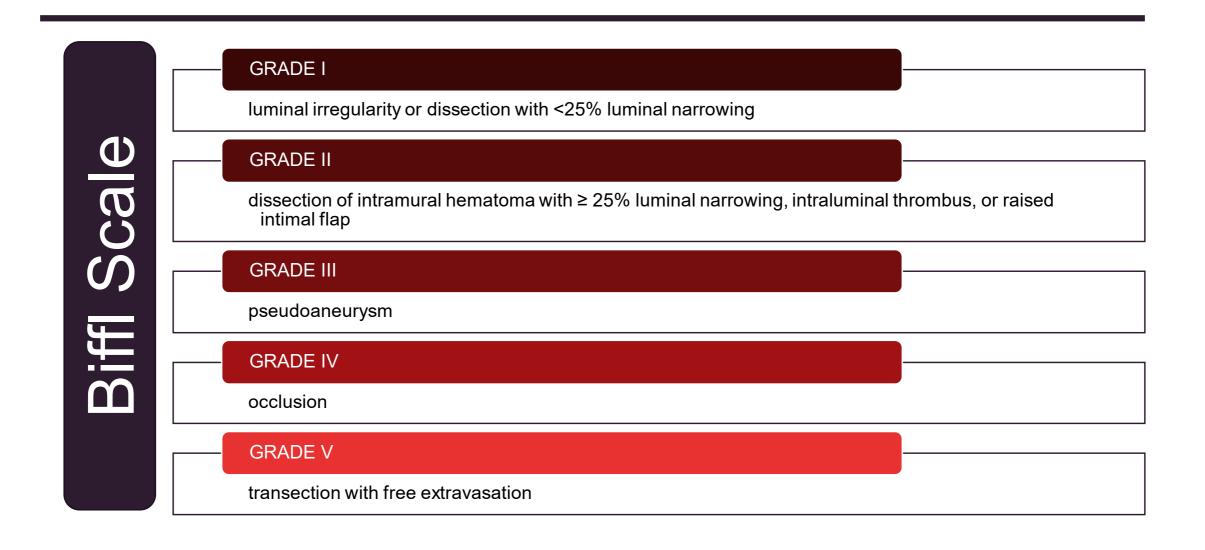
High-energy mechanism plus any risk factor:

- Le Fort II or III fracture
- Mandible fracture
- Complex skull fracture
- Base of skull fracture
- Scalp degloving
- C-spine fracture, subluxation, or ligamentous injury at any level
- Severe TBI with GCS <6
- Clothesline or seatbelt abrasion
- TBI with thoracic injuries
- Thoracic vascular injuries
- Blunt cardiac rupture

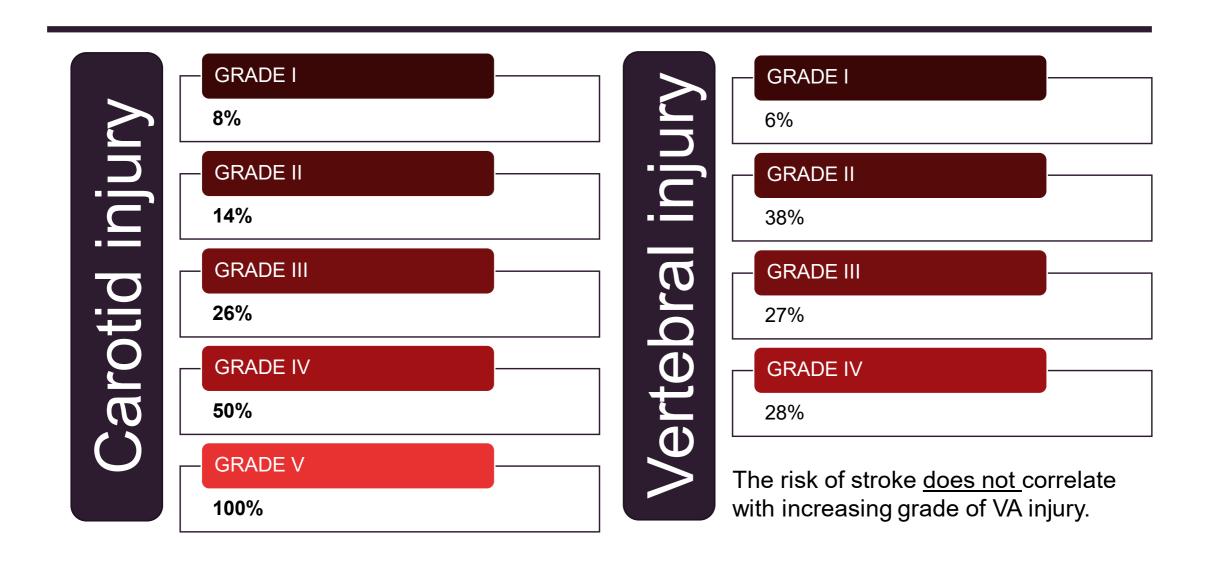
Criteria Memphis Modified

- Base of the skull fracture with involvement of the carotid canal and/or the petrous temporal bone
- Cervical spine fracture
- Neurological exam not explained by neuroimaging
- Horner syndrome
- Le Fort II or III
- Neck soft tissue injury

GRADING BCVI



RISK OF STROKE



TREATMENT

Any medical treatment (antiplatelet or anticoagulation) is likely better than no treatment for the prevention of stroke.

There is a paucity of high-quality evidence to guide the specific choice of medical treatment of asymptomatic BCVI to present stroke and/or promote vessel healing.

In review of the literature, those who received treatment had lower rates of strokes than those who did not but allocation to treatment was often protocolized based on injuries, risks and other patient factors leading to bias in treatment. Those who received no treatment may have had too severe of injuries or may have transition to comfort care.

- Intimal flap injuries have a low stroke risk and high rate of resolution with antithrombotic therapy
 - In one study, one-half of those that were not even treated were healed at follow-up
- Initial treatment with either heparin or antiplatelet therapy is appropriate for grade I injuries
 - No data demonstrating superiority of one over the other
 - Consider heparin in hospitalized patients who may be undergoing surgical procedures
 - Antiplatelet therapy until the lesion is healed
- These lesions do not have flow-limiting potential, additional treatment (surgical or endovascular) is **not** necessary
- Follow-up imaging 7 to 10 days following the injury, or for any change in neurologic status

- Arterial dissections often progress in spite of treatment
- Antithrombotic therapy using heparin, reserving antiplatelet therapy for those who have contraindications to heparin
- Follow-up imaging 7 to 10 days following the injury, or for any change in neurologic status
- Long-term treatment with antiplatelet therapy appears to be adequate for stroke prevention for patients with stable lesions
- Endovascular stenting may be indicated if carotid injury progresses to the point of near-occlusion

- Arterial pseudoaneurysm injuries are less likely to heal compared with lower-grade injuries
- Antithrombotic therapy with heparin, reserving antiplatelet therapy for those who have contraindications to heparin
- In one study, over one-half of pseudoaneurysms remained the same size or enlarged at a mean of six months
 - Intervention is generally warranted once a pseudoaneurysm reaches a size of 1.0 to 1.5 cm or is symptomatic.
 - Surgically accessible carotid artery pseudoaneurysm can be treated:
 - Resection and repair (patch or interposition graft)
 - Endovascular techniques such as stenting or coil embolization

- Injuries with complete arterial thrombosis are associated with high mortality, particularly when the injury is in the carotid artery, and the neurologic outcome is proportional to the degree of neurologic impairment on presentation
- Antithrombotic therapy using heparin, reserving antiplatelet therapy for those with contraindications to heparin
- In one study, 82 percent of grade IV injuries remaining unchanged
 - Over time, a small number of occlusions may recanalize
 - Hypothesized that this may lead to stroke, and that angioembolization may be preventative; there are no data demonstrating any additional benefit in asymptomatic patients
- Since the early follow-up examination is unlikely to change in a manner that will alter treatment, no follow-up scan
- Lifelong antiplatelet therapy for patients with occlusions

- Transection injuries of the carotid artery are associated with high rates of stroke and high mortality
- Hemorrhage from the neck as evidenced by an expanding hematoma (zone II) should be controlled by direct pressure until surgical control, if accessible, or endovascular control can be achieved
- Hemorrhage from the mouth or ears is often indicative of a lesion that will require angioembolization for control

OTHER CONSIDERATIONS

The risks and benefits of antithrombotics must be carefully weight in a polytrauma.

Example 1:

Patient has a grade I splenic laceration with a grade IV ICA BCVI

Example 2:

Patient has a grade IV splenic laceration with a grade I ICA BCVI

While both patients have BCVI, clearly they are not equal. One must also consider upcoming OR trips / procedures that may contraindicate therapy as well as other indications or contraindications for antithrombotic therapy such as recent cardiac stents or a GI bleed, respectively.