THE ROLE OF ACUTE CARE NURSE PRACTITIONER IN THE MANAGEMENT OF
BLUNT CEREBROVASCULAR INJURY

by

Kaori Bird

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SIGNED: Kaori Bird

APPROVAL BY MASTER'S PROJECT DIRECTOR

This Master's Project has been approved on the date shown below:

Leslie Ritter, PhD, RN, FAAN     Date:
Associate Professor
ACKNOWLEDGMENTS

Leslie Ritter PhD, RN, FAAN
Associate Professor
College of Nursing and Department of Neurology
University of Arizona

Ted Rigney PhD, ACNP-BC, FAANP
Assistant Director, Nurse Practitioner Program & Clinical Associate Professor
College of Nursing
University of Arizona
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ABSTRACT

Blunt carotid artery injury (BCI) and blunt vertebral artery injury (BVI) are collectively termed blunt cerebrovascular injury (BCVI). BCVI is associated with a risk of cerebrovascular events, or stroke. Initial signs of BCVI may be minimal, vague, or even delayed and diagnosis of BCVI are often complicated or masked by the presence of concomitant traumatic injuries associated with BCVI. BCVI has been increasingly diagnosed using standardized protocols to promptly and accurately detect injury and manage these patients in an effort to reduce the devastating neurologic sequelae of a missed injuries. Currently, there is no consensus or guidelines which direct outline the definitive management of BCVI. This article reviews current knowledge on evaluation strategies and optimal management of patients with BCVI. Acute care nurse practitioners (ACNP) care for patients with a variety of complex injuries who have unique, specialized needs, such as neurological injury with traumatic brain injury and multiple organ involvement. ACNPs are increasingly recognized as an integral part of the trauma care delivery system today. To our knowledge, the role of the ACNP in the management of patients with BCVI has not been previously described. Given the complexity and diverse pathophysiology encountered in the trauma patient population as well as the evolving roles and responsibilities of ACNPs, the information on BCVI presented in this article may positively impact clinical practice in a variety of settings. In order to help improve outcomes of patients with BCVI, the role and significance of the ACNP in the evaluation of trauma patients with suspected BCVI and key features of ACNP in management of patients with BCVI will be discussed as well as an algorithm for the initial triage and management is presented for use by the ACNP.
CHAPTER 1: CASE PRESENTATION

The patient is 32 y.o. female who was a restrained driver of a vehicle that was traveling at approximately 40mph. Her vehicle struck the driver's side of a car that pulled out in front of her. There was an airbag deployment and 15 inch intrusion into the front passenger compartment of her vehicle. She felt “hazy” immediately after the crash, but denied losing consciousness. She remembered the entire event and she was ambulatory at scene. Initially she was complaining of neck pain with active range of motion and diffuse lower back tenderness. She has no significant medical or surgical history and she does not drink alcohol, smoke cigarettes, or take illicit substances. In the emergency department, vital signs were normal except for a slightly elevated heart rate. Physical examination was unremarkable except for mild cervical spine tenderness on palpation and mild tenderness along 6-8th right ribs without bony crepitus. It was difficult to determine whether her neck pain was mid-line tenderness or para-spinal muscle pain from the physical exam. There were no other obvious signs of trauma and the neurological examination was unremarkable including no muscle weakness or paresthesias. The computed tomography (CT) scan of the neck without contrast was unremarkable and her cervical collar was subsequently removed. She had no difficulty breathing and her right-sided thoracic pain improved during the course of the emergency department stay. She was discharged to home from the emergency department with a diagnosis of cervical strain and instructed to take ibuprofen for pain and follow up with her primary care provider the next day.

Three days later, she followed up with her primary care physician, who requested medical records from the emergency department visit and ordered a head CT scan without contrast for a complaint of headache. Eight days after the accident, she returned to the emergency department
with complaint of right sided weakness and a persistent headache since that morning. She woke with a headache around 6:00am, feeling unwell, she went back to sleep. She woke 3 hours later with right-sided facial droop and right-sided hemiparesis. Her boyfriend reported that she had sounded “like she was drunk” on the phone 3 days prior to that morning.

In the emergency department, she was alert and oriented x3 with expressive aphasia and dysarthria. Physical exam revealed normal reflexes and sensation in all extremities with motor strength 3/5 in right upper extremity and 4/5 in right lower extremity. Cranial nerve II through XII were grossly intact except for right facial droop and right facial weakness. A head and neck CT with angiography (CTA) showed left carotid artery (ICA) dissection and right ICA pseudoaneurysm. She was diagnosed with cerebrovascular accident and transferred to another facility for further care and an immediate consultation with an interventional neuroradiologist.

A head and neck CTA obtained 42 days following the initial event showed stable and improved findings compared to the previous CTA. It revealed encephalomalacia within the left middle cerebral artery (MCA) distribution consistent with subacute and chronic infarction and an interval development of complete patency of the proximal left MCA. There was an increased patency of the intracranial portion of the left ICA although it was abnormally stenotic while the cervical left ICA showed persistent stenosis. The right cervical ICA pseudoaneurysm was unchanged.
CHAPTER 2: INTRODUCTION

Although blunt traumatic injuries are not generally thought of as a cause of stroke, there is a real and high risk for cerebrovascular events among patients who have sustained blunt traumatic injuries, especially of the head and neck. Blunt carotid artery injury (BCI) and blunt vertebral artery injury (BVI) are collectively termed blunt cerebrovascular injury (BCVI). BCVI is associated with a stroke risk of approximately 50% in patients with BCI and about 20-25% in those with BVI (Kirkpatrick, A., Evans, D., & Meredith, W. 2009). Compared to penetrating carotid injuries, patients with blunt carotid injuries overall are found to have worse functional outcomes due to delayed recognition and treatment (Martin et al., 2005). BCVI is most frequently associated with motor vehicle collisions and other mechanisms of injury including pedestrian struck by a vehicle, motorcycle collisions, direct blow to the neck, falls, strangulation, hanging, and sports accidents (Arthurs & Starnes, 2008). A cerebrovascular injury due to BCVI complicates traumatic, often multiple, injuries, leading to high morbidity and mortality.

As the opening case study has illustrated, missed injuries and delayed diagnosis of BCVI lead to serious complications such as stroke and permanent neurological disability. A number of patients without any risk factors for BCVI may escape detection. Subtle injuries may be missed and asymptomatic patients at risk for stroke may be left untreated if initial evaluation is based solely on risk factors, signs and symptoms, or physical exam findings. Although these injuries have been reported to be uncommon, BCVI has been increasingly diagnosed using standardized protocols to promptly and accurately detect injury and manage these patients in an effort to reduce the devastating outcome of an overlooked injuries. While the diagnosis and treatment of BCVI have evolved significantly during the past two decades, there is still controversy regarding
what the best diagnostic and management strategies should be, posing significant challenges to trauma clinicians. This article reviews current knowledge on evaluation strategies and optimal management of patients with BCVI. In addition, the role and significance of the acute care nurse practitioner (ACNP) in the evaluation of trauma patients with suspected BCVI and key features of ACNP in management of patients with BCVI will be discussed. Last, in order to help improve outcomes of patients with BCVI, an algorithm for the initial triage and management is presented for use by the ACNP.
CHAPTER 3: EPIDEMIOLOGY OF BCVI

There is a paucity of data regarding the epidemiology of blunt neck trauma. The incidence of serious complications related to BCVI following blunt trauma may be underreported because initial signs and symptoms of such injuries are often indistinguishable from accompanying injuries and may go unrecognized. The prevalence of BCVI is 0.5-1.55% of all blunt trauma cases and it has been reported as high as 2.7% (Mutze, S., Rademacher, G., Matthes, G., Hosten, N., & Stengel, D. 2005). Notably, the incidence of BCVI is much higher among patients with extensive basilar skull fractures, cervical fractures, cervical soft tissue hematomas, and facial fractures (Resnick, D. K., Subach B. R., Marion, D. W. 1997, Biffi et al., 2002, Miller et al., 2002, Kerwin et al., 2001, McKinney et al., 2007). Demographic data relating to BCVI is limited, but it is likely to be similar to that of other traumatic injuries, primarily male, relatively young age, and those who are injured in motor vehicle collisions and other traumatic events (Cothren et al., 2004, Stein, D. M. Boswell, S., Sliker, C. W., Lui, F. Y. & Scalea, T. M. 2009).

The number of reported cases of BCVI has climbed in the recent years, most likely due to increased awareness among clinicians and use of standardized screening protocols for these injuries in severely injured patients (Sliker, C. W., Shanmuganathan, K., & Mirvis, S. E. 2008). In a recent study by Eastman and colleagues, 4216 trauma patients were screened using a modified Denver screening criteria designed for patients at risk for BCVI (Eastman, A. L., Chason, D. P., Perez, C. L., McAnulty, A. L., Minei, J. P. 2006). Blunt trauma patients who met at least one criterion were deemed to be at risk and underwent a screening computed tomographic angiography (CTA). In this study, the authors found 162 at risk patients and BCVI was found in 46 of these patients, yielding a prevalence rate of 28.4% in the screened population,
and an incidence of 1.25% in the overall study sample (Eastman et al, 2006). Alarming news is that this relatively small percentage of patients with BCVI has a morbidity rate secondary to BCVI-related strokes ranging from 25-58% and mortality rate of 25-59% (McKevitt, E. C., Kirkpatrick, A. W., Vertesi, L., Granger, R. & Simon, R. K. 2002, Arthurs & Starnes, 2008, Stein, et al., 2009).
CHAPTER 4: PATHOPHYSIOLOGY AND MECHANISM OF INJURY

The neck contains many vascular as well as other vital structures such as airway, nerves and spinal cord. The neck contains many vascular as well as other vital structures such as airway, nerves and spinal cord. These structures are enclosed by layers of superficial fascia and deep cervical fascia, but they are relatively unprotected and vulnerable to traumatic injury. The common carotid artery bifurcates to the external carotid and internal carotid at the level of C4. The internal carotid artery extends through 4 segments, cervical, petrous, cavernous, and supraclinoid, and supplies the branches of the anterior cerebral artery and the middle cerebral artery (Bouthillier, A., van Loveren, H. R. & Keller, J. T. 1996) (Figure1).

Figure 1. Arterial circulation of the neck and brain. Copyright 2010 by Kaori Bird.
The internal carotid artery gives rise to the ophthalmic artery, anterior cerebral artery, anterior choroidal artery, middle cerebral artery, and posterior cerebral artery (Adams, H. P., del Zoppo, G. J., & von Kummer, R. (2006). The vertebral arteries arise from the subclavian arteries at the level of C6 and they proceed superiorly through the transverse foramen of each cervical vertebra of C2-6 and travels across the posterior arch of C1 before entering the foramen magnum to join the basilar artery at the base of the medulla oblongata (Figure 2).

Figure 2. Description of the neck vessels. Note. From www.heart-vessels.com. Reprinted with permission.

The basilar artery terminates as it bifurcates into the left and right posterior cerebral arteries. The two vertebral arteries and the basilar artery are together called the vertebrobasilar system, which supplies blood to the posterior part of circle of Willis and anastomoses with the anterior part of
the circle of Willis, which is supplied by the internal carotid arteries (Figure 3).

Figure 3. The vertebral artery, a branch of the subclavian, presents cervical, vertebral, suboccipital, and intracranial parts. Note. From Dartmouth Medical School Basic Human Anatomy by O'Rahilly. Reprinted with permission.

The sites of BCVIs can be anywhere along the carotid and vertebral arteries, but more frequently occur in certain areas (Singh, R. R., Barry, M. C., Ireland, A. & Bouchier Hayes, D. 2004, Sliker, 2008, Ringer A. J., Matern, E., Parikh, S. & Levine, N. B. 2009) (Table 1).

Table 1. Common sites of BCVI.

<table>
<thead>
<tr>
<th>Injury</th>
<th>Common sites of injury</th>
</tr>
</thead>
</table>
| BCI    | - The distal ICA where the artery can stretch over the lateral bony masses of the C3 - C4 vertebrae  
|        | - Transverse processes near of the C1-C3 vertebral bodies  
|        | - The petrous segment of the temporal bone, near the carotid canal  
|        | - The cavernous segment of the ICA  
|        | - The proximal portion at the base of the skull, near the foramen lacerum |
Injuries affecting multiple blood vessels in the head and neck are not uncommon and have been reported in 18-38% of patients with BCVI (Sliker, 2008). In other cases, BCVI may be accompanied by injuries to the external carotid arteries and other arterial branches in the head and neck. A dissection of the carotid artery can extend through the carotid canal and involve the intracranial arteries.

Energy transfer during a traumatic injury can result in a variety of insults and possible vascular injury such as a direct blow to the blood vessel, or laceration of the vessel by adjacent bone fractures. Direct energy with compressive forces, acceleration-deceleration forces, stretching or tearing forces to the neck can cause extreme hyperextension, hyperflexion, and rotation (Arthurs & Starnes, 2008, Sliker 2008, Ringer et al., 2009) (Table 2).

Table 2: Common mechanisms of injury of BCVI.

<table>
<thead>
<tr>
<th>Injury</th>
<th>Mechanism</th>
<th>Resulting injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>BCI</td>
<td>Cervical hyperextension</td>
<td>Stretches the carotid artery over the lateral articular processes of C1 through C3 at the base of the skull</td>
</tr>
<tr>
<td></td>
<td>Lateral flexion</td>
<td></td>
</tr>
<tr>
<td>BCI</td>
<td>Cervical hyperflexion</td>
<td>Compresses the carotid artery between the spine and the mandible</td>
</tr>
<tr>
<td>BCI</td>
<td>Mandible fractures</td>
<td>Posteriorly displaced mandible crushes the internal carotid artery between the mandible and spine</td>
</tr>
<tr>
<td>BVI</td>
<td>Cervical spine subluxation</td>
<td>Stretches the relatively fixed vertebral artery over the adjacent bony structures</td>
</tr>
<tr>
<td></td>
<td>Cervical spine dislocation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cervical spine fracture at the C1 - C3 (esp. fractures involving transverse foramen)</td>
<td></td>
</tr>
<tr>
<td>BVI</td>
<td>Occipital condyle dislocation</td>
<td>Dislocated occipital condyle crushes the vertebral artery against C1</td>
</tr>
</tbody>
</table>
Available data regarding specific injuries associated with BCVI are somewhat inconclusive. BCVI can result from direct cervical trauma, traumatic blow to the neck, intraoral trauma, craniocervical junction injury, basilar skull fracture involving the petrous or sphenoid portions of the carotid canal, or displacement of bone fragments from basilar skull fractures or cervical spine fractures (Biffl et al., 1999, Miller et al., 2002, McKeveit et al., 2002). Traumatic injuries frequently associated with BCVI include closed head injuries, facial fractures, cervical spine fractures, spinal cord injury, basilar skull fractures, carotid canal or vertebral foramen fractures, and major thoracic injuries (McKinney et al., 2007, Ringer et al., 2009, Stein et al., 2009).

McKinney and colleagues (2007) studied the incidence of BCVI and found that patients with carotid canal or vertebral foramen fractures and severe cervical subluxations on CT had a high rate of BCVI (McKinney et al., 2007). Later, Ringer and colleagues (2009) confirmed these earlier findings and reported a high frequency of BCVI in patients with cervical spine fractures, mid-face fractures, fractures of skull base after high-velocity trauma. These investigators also found that lateralizing neurological deficit consistent with hemispheric ischemia, but unexplained by accompanying injuries, was a better predictor of BCVI than a decreased mental status such as Glasgow Coma Scale (GCS) score of less than 6 (Ringer et al., 2009). There was low frequency of BCVI in patients with isolated thoracic injuries and soft tissue injuries of the neck (Ringer et al., 2009). Contrary, Stein and colleagues (2009) found Le Fort I or II facial fractures, skull base fractures, cervical spine fractures or cervical spinal cord injury, significant traumatic brain injury, and major thoracic injury were all risk factors for BCVI (Stein et al., 2009). Specifically, BCI was associated with severe facial fractures, skull base fractures,
traumatic brain injury, and major thoracic injuries whereas BVI were associated with cervical spine fractures or spinal cord injury (Stein et al., 2009). In another recent study, investigators found that cervical spine fracture, basilar skull fracture, and mandible fracture were more predictive of BCVI than major thoracic injury (Berne, J. D., Cook, A., Rowe, S. A. & Norwood, S. H. (2010). Therefore, high-velocity blunt trauma associated with unexplained, often lateralizing, neurological deficits on intracranial imaging and fracture of the cervical spine, midface, or skull base, are indications for screening neck CTA for BCVI while isolated major thoracic injuries or soft tissue injuries of the neck may not.

Although energy caused by high-velocity trauma sufficient to result in vascular injury of the neck often suffices to produce injuries to the bone, BCVI has been reported to occur after a low-energy or seemingly trivial trauma secondary to preexisting blood vessel and bony abnormalities. There is data suggesting patients with the following predisposing medical conditions may be at increased risk for BCVI, including hypertension, Marfan syndrome, Ehlers-Danlos syndrome Type IV, autosomal dominant polycystic kidney disease, osteogenesis imperfecta Type I, fibromuscular dysplasia, syphilis, arteriopathies, and Erdheim cystic medial necrosis (Ringer et al., 2009).

Injury to the arterial intima exposes subendothelial collagen. Platelets readily adhere to this thrombogenic surface, and a platelet plug forms at the site of intimal damage. This can cause an occlusion from thrombosis formation. Platelet aggregates at the site of intimal disruption causes luminal stenoses and occlusions, which lead to reduced arterial blood flow to the brain, resulting in downstream infarction (Singh et al., 2004). Dissection may begin as an intimal tear within the wall of a blood vessel, which allows circulating blood under high arterial pressure to
penetrate into the arterial wall and split, or “dissect” its layers, resulting in an intramural hematoma and luminal stenosis (Singh et al., 2004).

Pseudoaneurysm may form as blood leaks from the injured artery into the surrounding tissue through the adventitia, creating a hematoma outside the arterial wall. This blood-filled cavity may eventually clot to seal the blood leak from the parent artery. However, it can also expand, compressing surrounding structures such as nerves or it can eventually rupture out of the tissue enclosing it and extravasate (Singh et al., 2004). For example, an enlarging pseudoaneurysm within the cavernous segment of the ICA, which is typically associated with basal skull fractures, can result in a caverous sinus syndrome and compression of certain cranial nerves or it may rupture and create a cavernous arteriovenous fistula. A medial tear of the ICA with rupture and hemorrhage into the sphenoid sinus can cause massive and life-threatening epistaxis (Krigs et al., 2008).

Initial intimal injury can also progress to arteriovenous fistula (AVF) formation. The most common traumatic AVF are carotid-cavernous fistulae. Tearing of the internal carotid artery (ICA) which is relatively fixed within the cavernous sinus by fibrous trabeculae can result in rupture of the ICA into the cavernous sinus and fistulization while injury to the external carotid artery (ECA) and the adjacent vein can also result in a ECA AVF (Krings, T., Geibprasert, S. & Lasjaunias, P. L. 2008). In these injuries, elevated venous pressure may result in orbital and ocular abnormalities including extraocular muscles dysfunction, proptosis, and subchoroidal effusions and abnormal diversion of blood flow into these fistulas may lead to cerebral ischemia (Sliker, 2008). With a large expanding arterial dissection or active hemorrhage from arterial transection, hemodynamic disturbances can occur rapidly, leading to cerebral ischemia or fatal
exsanguination (Singh et al., 2004). When a small intimal flap progresses to a dissection, occlusion, or becomes a possible source of emboli to the cerebral circulation, there may be a delay between time of original trauma and appearance of thromboembolism and resulting cerebral ischemia.
CHAPTER 5: CLINICAL PRESENTATION

Obvious signs of BCVI may include hemorrhage from the mouth, nose, ears, or open wounds or a large, expanding cervical hematoma; however, signs of BCVI are often minimal, vague, or even delayed. Common symptoms of BCVI are headache, neck pain, and manifestations of cerebral or retinal ischemia (Singh et al., 2004). “Carotid injuries typically present with contralateral sensory or motor deficit, and vertebral injuries present with ataxia, vertigo, emesis, and possible visual field deficits” (Arthurs & Starnes, 2008, pp1232). Carotid cavernous fistulas may present as orbital pain, blurred vision, pulsatile exophthalmos or proptosis, chemosis, hyperaemia, cranial nerve III, IV, or VI palsies, cerebral edema, and seizure (Cothren & Moore, 2005, Arthurs & Starnes, 2008) (Figure 4).

Figure 4. Neurovascular anatomy: Internal carotid artery. Note. From Loyola University Medical
In essence, the signs and symptoms of neurologic events in the carotid and vertebrobasilar circulations reflect the individual vascular territories of the arteries or their branches (Adams, et al., 2006) (Table 3).

Table 3: Clinical presentation according to the carotid artery and vertebrobasilar circulations.

<table>
<thead>
<tr>
<th>Areas supplied</th>
<th>Branches supplied &amp; Signs/symptoms of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Carotid artery circulation</strong></td>
<td>• Ophthalmic artery → Amaurosis fugax</td>
</tr>
<tr>
<td>Cortex, lobar white matter, deep structures of the</td>
<td>• Middle cerebral artery → contralateral</td>
</tr>
<tr>
<td>cerebral hemisphere, eyes</td>
<td>hemiplegia (arm and face &gt; leg), hemiparesis,</td>
</tr>
<tr>
<td></td>
<td>homonymous hemianopia, aphasia (if left</td>
</tr>
<tr>
<td></td>
<td>hemisphere is affected)</td>
</tr>
<tr>
<td></td>
<td>• Anterior cerebral artery → frontal lobe</td>
</tr>
<tr>
<td></td>
<td>symptoms, hemiplegia (leg &gt; face and arm)</td>
</tr>
<tr>
<td><strong>Vertebrobasilar circulation</strong></td>
<td>• Posterior cerebral artery → visual</td>
</tr>
<tr>
<td>Brain stem, cerebellum, cerebral hemisphere,</td>
<td>disturbance/loss</td>
</tr>
<tr>
<td>thalamus, inner ear.</td>
<td>• Posterior inferior cerebral artery</td>
</tr>
<tr>
<td></td>
<td>• Anterior inferior cerebral artery</td>
</tr>
<tr>
<td></td>
<td>• Internal auditory artery</td>
</tr>
<tr>
<td></td>
<td>• Superior cerebellar artery</td>
</tr>
<tr>
<td></td>
<td>• Penetrating branches</td>
</tr>
<tr>
<td></td>
<td>Disruption of blood flow to vertebral artery,</td>
</tr>
<tr>
<td></td>
<td>basilar artery, SCA, AICA, PICA → brain stem</td>
</tr>
<tr>
<td></td>
<td>dysfunction</td>
</tr>
</tbody>
</table>

Diagnosis of BCVI may be often complicated by the presence of concomitant traumatic injuries associated with BCVI, such as basilar skull fracture, serious facial fractures, spinal cord injury, thoracic injury, traumatic brain injury, and diffuse axonal injury, all of which may mask the signs and symptoms of BCVI (Arthurs & Starnes, 2008). Other major traumatic injuries, such as chest and abdominal injuries, extremity injuries, and pelvic fractures make the evaluation of BCVI challenging. These injuries may also cause significant hemodynamic instability and resulting cerebral ischemia (McKevitt et al., 2002, Biffl et al., 1999). Therefore, subtle physical
exam findings that are inconsistent with radiographic results as well as less recognizable signs of BCVI, such as cervical bruit in patients younger than 50 years of age, unexplained central or lateralizing neurologic deficit, transient ischemic attack, pulsatile tinnitus, and Horner's syndrome should increase a clinician's index of suspicion for BCVI (Biffl, W. L., Moore, E. E., Offner, P. J., & Burch, J. M. 2001).

BCVI have also been reported in the absence of associated signs and symptoms and other associated traumatic injuries (Biffl et al., 1999). Thus, dilemma and challenge arise when patients present with a significant mechanism of injury as in motor vehicle crash without any immediate or obvious evidence of an injury. Some patients with BCVI are asymptomatic or exhibit no signs of clinically significant ischemia, while others develop signs and symptoms over hours, or weeks to months after the initial traumatic event depending on the natural progression of the injury (Biffl et al. 2001, Arthurs & Starnes, 2008). This asymptomatic period in patients with BCVI, who initially did not receive treatment, has been reported to as long as 72 hours, although some data has shown it may be up to 14 years (Cothren et al., 2005). Therefore, the initial absence of physical findings does not preclude the possibility of BCVI and the need for further observation and diagnostic testing. The following section discusses the key elements and recommendations of management of BCVI based on available data.
CHAPTER 6: GUIDELINES FOR DIAGNOSIS AND MANAGEMENT OF BCVI

The first trauma practice guidelines for management of BCVI were developed by the Eastern Association for the Surgery of Trauma (EAST) in 2007. They were based on a literature review of 179 articles written between 1965 to 2005 (Bromberg et al., 2010). Key questions addressed in the guidelines included what population should be screened, what diagnostic modality should be used, and what is the appropriate treatment and follow-up for BCVI. Due to lack of randomized, prospective trials and scientific evidence, the EAST group did not establish convincing, strong recommendations, stating “change in the diagnosis and management of this injury constellation is rapid due to technological advancement and the difficulties inherent in performing randomized prospective trials in this patient population” (Bromberg et al., 2010, pp. 471). The American Association for the Surgery of Trauma (AAST) and the American Association of Neurological Surgeons (AANS) have not issued recommendations or definitive guidelines. The EAST guidelines support the use of an injury grading scheme proposed originally by the Denver group, Biffl and colleagues in 1999 (Biffl et al., 1999, Biffl, W. L., Egglin, T., Benedetto, B., Gibbs, F., & Cioffi, W. G. 2006). The use of the injury grading scale allows accurate, unified characterization of BCVI and determine prognosis and approach to appropriate therapy. The next section will examine the injury grading system for BCVI.

The injury grading scale

The Denver injury grading scale was developed in an effort to standardize reporting of BCVI, evaluate the variety of presentations, predict the risk of stroke, and guide treatment options (Biffl et al., 1999, Biffl, W. L., Egglin, T., Benedetto, B., Gibbs, F., & Cioffi, W. G. 2006). It consists of 5 injury grades that are based on angiographic findings (Table 4). Once
detected, BCVI can be triaged using the injury grading scale.

Table 4: Denver injury grading scale.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>Irregularity of the vessel wall or a dissection/intramural hematoma with less than 25% luminal narrowing</td>
</tr>
<tr>
<td>Grade II</td>
<td>A raised intimal flap, a dissection, intraluminal thrombus, or intramural hematoma with greater than 25% luminal stenosis, or hemodynamically insignificant AVFs</td>
</tr>
<tr>
<td>Grade III</td>
<td>Pseudoaneurysm</td>
</tr>
<tr>
<td>Grade IV</td>
<td>Vessel occlusion</td>
</tr>
<tr>
<td>Grade V</td>
<td>A transection with active extravasation, or hemodynamically unstable AVFs</td>
</tr>
</tbody>
</table>

Radiographic manifestations of BCVI are diverse. An aneurysm may appear as a blood-filled dilation, or a bulge of a blood vessel, a pseudoaneurysm may appear as a narrowing of the native arterial lumen, with resulting compromise of arterial flow, and an AVF may appear as an abnormal pattern of blood vessels (Sliker, 2008) (Figure 5).
Intraluminal thrombi may be seen at the site of intimal injury or within the lumen of a pseudoaneurysm (Sliker, 2008). Findings on angiography may include irregularity of the vessel wall, as well as characteristic findings such as stenosis with a “string sign” or tapered “flame shaped” occlusions (Singh et al., 2004, pp. 578).

The mortality rate is 100% in Grade V BCI, whereas the highest mortality rate of 31% in BVI is reported with Grade I with many of these deaths most likely attributed to severe head injury encountered in both BCI and BVI (Biffl et al., 2001). The prevalence of stroke in BCI increases by increasing grade scale while strokes in BVI tend to occur more consistently across all grades of injury, ranging from 6 – 40%, with the exception of 100% stroke rate in Grade V for both BCI and BVI (Biffle et al., 2001, Biffle et al., 2002) (Table 5).

Table 5: Stroke rate by the Denver injury grading scale.

<table>
<thead>
<tr>
<th>Injury grade</th>
<th>Stroke rate</th>
<th>Injury grade</th>
<th>Stroke rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>BCI I</td>
<td>3%</td>
<td>BVI I</td>
<td>6%</td>
</tr>
<tr>
<td>BCI II</td>
<td>11 - 14%</td>
<td>BVI II</td>
<td>38 - 40%</td>
</tr>
<tr>
<td>BCI III</td>
<td>26 - 33%</td>
<td>BVI III</td>
<td>13 - 27%</td>
</tr>
<tr>
<td>BCI IV</td>
<td>44 - 50%</td>
<td>BVI IV</td>
<td>28%</td>
</tr>
<tr>
<td>BCI V</td>
<td>100%</td>
<td>BVI V</td>
<td>100%</td>
</tr>
</tbody>
</table>

Biffle and colleagues (2002) found that nearly half of the Grade I and Grade II injuries healed completely while some portion of the Grade I and Grade II injuries (8% and 43% respectively)
that did not receive treatment progressed to pseudoaneurysm upon reevaluation with cerebral angiography 7-10 days following the injury. The goal of the care of blunt trauma patients with possible BCVI is detection of an injury in order to allow prompt intervention in the hope of reducing morbidity and mortality. The challenge of the initial management of these patients lies in systematic evaluation and accurate diagnosis of BCVI using effective screening criteria and radiographic study. However, there is no consensus on the ideal screening criteria to use to identify BCVI when managing blunt trauma patients, what diagnostic tool and when to order such test.

Screening criteria and diagnostic strategy

The initial assessment of blunt trauma patients with possible BCVI includes prompt evaluation to detect and treat life-threatening injuries and careful attention to the identification of associated injuries, as well as the assessment of the signs and symptoms and risk factors for BCVI. Unfortunately, multiple factors compromise prompt diagnosis of BCVI. Obtaining a complete neurological examination is often difficult in trauma patients due to the effects of medications, presence of alcohol or other substances, or respiratory failure with need for immediate intubation. Patients with BCVI can present with delayed signs and symptoms, with no obvious significant associated injuries, or be asymptomatic with potentially silent course of progression, resulting in devastating outcomes. In fact, nearly 20% of patients with BCVI have none of the risk factors or exhibit delayed onset of symptoms ranging from 1 day to 7 months following initial injury (Biffl et al., 1999, Kerwin, et al., 2001). Thus, clinicians are challenged with identifying patients at greatest risk for BCVI as well as detecting these injuries in patients who are asymptomatic.
In an effort to facilitate early diagnosis of BCVI, researchers from two major trauma centers in Denver and Memphis have developed criteria based on signs, symptoms, and risk factors associated with BCVI, the Denver criteria and the Memphis criteria respectively (Table 6).

Table 6: Commonly used screening criteria for BCVI

<table>
<thead>
<tr>
<th>Denver criteria</th>
<th>Memphis criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Signs and symptoms of BCVI</strong></td>
<td>• Cervical spine fracture</td>
</tr>
<tr>
<td>• Hemorrhage from mouth, nose, ears of potential arterial origin</td>
<td>• Neurological exam not explained by brain imaging</td>
</tr>
<tr>
<td>• Cervical bruit in patient &lt; 50 yo</td>
<td>• Horner's syndrome</td>
</tr>
<tr>
<td>• Expanding cervical hematoma</td>
<td>• LeFort II or II fracture pattern</td>
</tr>
<tr>
<td>• Focal neurologic deficit (transient ischemic attack, Horner's syndrome, hemiparesis, vertebrobasilar syndrome)</td>
<td>• Basilar skull fracture with involvement of the carotid canal</td>
</tr>
<tr>
<td>• Neurologic exam incongruous with head CT scan findings</td>
<td>• Neck soft tissue injury (seatbelt sign or hanging or hematoma)</td>
</tr>
<tr>
<td>• Stroke on CT scan or MRI</td>
<td></td>
</tr>
<tr>
<td><strong>Risk factors for BCVI</strong></td>
<td></td>
</tr>
<tr>
<td>High-energy transfer mechanism with:</td>
<td></td>
</tr>
<tr>
<td>• Displaced mid-face fracture (LeFort II or III fracture)</td>
<td></td>
</tr>
<tr>
<td>• Cervical spine fracture (subluxation, transverse foramen involvement, any C1-C3)</td>
<td></td>
</tr>
<tr>
<td>• Basilar skull fracture with carotid canal involvement</td>
<td></td>
</tr>
<tr>
<td>• Diffuse axonal injury with GCS &lt;6</td>
<td></td>
</tr>
<tr>
<td>• Near hanging with anoxic brain injury</td>
<td></td>
</tr>
</tbody>
</table>

The modified Denver screening criteria

- Lateralizing neurologic deficit (not explained by CT head)
- Infarct on CT head scan
- Expanding cervical haematoma
- Massive epistaxis
- Anisocoria / Horner’s syndrome
- GCS ≤ 8 without significant CT findings
- Cervical spine fracture
- Basilar skull fracture
• Severe facial fracture (LeForte II or III)
• Seatbelt sign above the clavicle
• Cervical bruit or thrill in patient below 50 years old

The Denver criteria risk stratify patients with injuries secondary to high-velocity trauma mechanism based on signs and symptoms, and risk factors for BCVI. If any of one or more of these criteria are present in a trauma patient presenting with blunt injury, further investigation with an diagnostic imaging is recommended. Using the Denver criteria, 4.8% of all blunt trauma patients were screened and 18% of these patients were found to have BCVI (Biffl et al., 1998).

The Memphis criteria consider trauma patients who have sustained an associated injury to be at high risk for BCVI. The Memphis group has found that nearly all BVI were associated with cervical spine subluxations and fractures involving transverse foramen in their research findings (Miller et al., 2002). Using the Memphis criteria, 3.5% of all blunt trauma patients were screened and 29% of these patients were found to have BCVI (Miller et al., 2002). Thus, the Memphis criteria is slightly more restricted screening protocol with a higher screening yield, potentially limiting the number of negative diagnostic studies. The Denver group has later proposed the modified Denver screening criteria, which incorporates both Denver and Memphis criteria and additional research findings (Eastman et al., 2006, Eastman, A. L., Muraliraj, V., Sperry, J. L., & Minei, J. P. 2009).

While the EAST guidelines did not support the use of single screening criteria, the authors concluded that patients at risk for BCVI can be identified before the onset of neurologic symptoms using screening protocols, thus all blunt trauma patients at risk for BCVI should be screened for diagnostic evaluation (Broombert et al., 2010) (Table 7). Additional evidence based on randomized, prospective studies is necessary in order to unify the screening criteria (i.e.
mechanism of injury, associated injuries, signs and symptoms, and risk factors) in both research and clinical settings (Biffl et al., 1998, Miller et al., 2002, Cothren & Moore, 2005, Cothren et al., 2007).

Table 7: Criteria for screening and risk factors by EAST

<table>
<thead>
<tr>
<th>Level II recommendations:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Patients presenting with any neurologic abnormality that is unexplained by a diagnosed injury should be evaluated for BCVI.</td>
</tr>
<tr>
<td>• Blunt trauma patients presenting with epistaxis from a suspected arterial source after trauma should be evaluated for BCVI.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Level III recommendations:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic patients with significant blunt head trauma as defined below are at significantly increased risk for BCVI and screening should be considered. Risk factors are as follows:</td>
</tr>
<tr>
<td>• Glasgow Coma Scale score 8</td>
</tr>
<tr>
<td>• Petrous bone fracture</td>
</tr>
<tr>
<td>• Diffuse axonal injury</td>
</tr>
<tr>
<td>• Cervical spine fracture particularly those with (i) fracture of C1 to C3 and (ii) fracture through the foramen transversarium</td>
</tr>
<tr>
<td>• Cervical spine fracture with subluxation or rotational component</td>
</tr>
<tr>
<td>• LeFort II or III facial fractures</td>
</tr>
</tbody>
</table>

Diagnostic strategies for BCVI

Current controversy surrounds the optimal diagnostic strategy for BCVI, the indications for instituting standardized protocols, the benefits of using such liberal screening criteria, especially in asymptomatic patients, and whether early diagnosis actually improves overall outcomes. Being a relatively rare injury with numerous, nonspecific risk factors, a large number of patients may need to be screened to produce high diagnostic yield and such broad inclusion criteria may result in a number of potentially unnecessary tests being performed, hence increasing the cost of health care (Mayberry, J. C., Brown, C. V., Mullins, R. J. & Velmahos, G. C. 2004, Berne et al., 2006). The impact of BCVI, the degree of neurological and functional
deficits, and mortality among patients who survive BCVI are difficult to determine because it is often unclear how much of the disability is directly attributable to BCVI and how much is due to concomitant head injury or other associated injuries.

Opponents of broad selection criteria have questioned the clinical and cost effectiveness of liberal screening protocols and argued that the specificity of screening asymptomatic patients and its diagnostic yield must be improved before instituting liberal screening protocols. They have suggested that these injuries are infrequent, the potential neurological event associated with BCVI may be minimal, and even if present, neurologic sequelae is not always preventable (Mayberry et al., 2004). They have cautioned that liberal screening protocols should not be instituted until further data becomes available due to potential risks associated with diagnostic tests, substantial resources required for screening, lack of clearly defined optimal treatment strategies, and absence of level I evidence in the current literature (Mayberry et al., 2004).

However, there have been limited studies evaluating protocols using restricted screening criteria and data from these studies have been inconclusive regarding the preponderance of the evidence supporting the futility of standardized protocols using liberal screening criteria of asymptomatic patients at risk for BCVI (Arthus & Starnes, 2008).

Overall cost-effectiveness of instituting screening protocols is difficult to estimate, but the cost of long-term rehabilitation, morbidity and mortality, and lost productivity due to neurological events is substantial. An analysis involving a large number of patients (total number of patients with BCI = 454) found BCI was associated with more severe functional disability in terms of feeding, communication, and mobility at discharge as compared to patients with similar blunt trauma injuries other than BCI, most frequently being severe head injury (Martin et al.,
The authors concluded that patients with BCI had worse neurological disability than those with penetrating carotid artery injury and BCI was independently associated with poor functional outcome at discharge (Martin et al., 2005). Available data have also shown that higher incidence of BCVI among patients who were screened for the injury as compared to those who were not screened (Arthus & Starnes, 2008). Furthermore, comprehensive screening for BCVI using standardized protocols has resulted in the early diagnosis of asymptomatic high risk patients and allowed prompt treatment to decrease the incidence of neurological sequelae (Cothren et al., 2005). Therefore, limiting screening criteria results in a decrease in the rate of detection of occult injuries, potentially leading to increased risk of morbidity and mortality. More studies are needed to determine the evidence-based, optimal screening protocols and diagnostic strategies for BCVI.

### Diagnostic modalities

With advances in technology, the current literature is filled with studies advocating computed tomography angiography (CTA) as a valid diagnostic alternative to digital four-vessel cerebral angiography (FVCA) of the head and neck for evaluating the vasculature of the head and neck. Available data is inconclusive regarding the best modality for the screening and diagnosis of BCVI. The EAST guidelines suggest that (8-slice or better) multidetector CTA may be equivalent to FVCA and should be considered as an initial screening tool for detecting BCVI while reserving FVCA for the definitive diagnosis of BCVI (Bromberg et al., 2007). The advantages and disadvantages of diagnostic modalities are summarized in the table below (Table 8).
Table 8. Advantages and disadvantages of commonly used diagnostic modalities for BCVI.

<table>
<thead>
<tr>
<th></th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVCA</td>
<td>• Allows excellent visualization of;</td>
<td>• Risk for serious complications</td>
</tr>
<tr>
<td></td>
<td>- the proximal and distal neck vessels</td>
<td>• Requires an insertion of an intra-arterial catheter and injection of a small amount of iodine contrast</td>
</tr>
<tr>
<td></td>
<td>- bilateral vessel injuries and associated vessel injuries</td>
<td>• $$ $$</td>
</tr>
<tr>
<td></td>
<td>- the intracranial circulation</td>
<td>• Resource intensive (requires interventional radiology suite and interventional radiologist)</td>
</tr>
<tr>
<td></td>
<td>- intracranial thromboembolic material</td>
<td></td>
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</tr>
<tr>
<td>CT</td>
<td>• Produces 3-D images of intracranial arteries (esp. 16-slice or better</td>
<td>• Requires a skilled radiographer and accurate and timely interpretation by an experienced neuroradiologist</td>
</tr>
<tr>
<td></td>
<td>multidetector scanner)</td>
<td>• Factors that hinder accurate visualization;</td>
</tr>
<tr>
<td></td>
<td>• Noninvasive, minimal complications</td>
<td>- location of certain injuries (the petrous and cavernous ICA and the vertebral arteries between the transverse foramina of the 2nd cervical spine and the base of the skull)</td>
</tr>
<tr>
<td></td>
<td>• Fast</td>
<td>- injuries that mimic BCVI (vasospasm)</td>
</tr>
<tr>
<td></td>
<td>• $$</td>
<td>- artifacts (movement, dental work, metallic objects)</td>
</tr>
<tr>
<td></td>
<td>• Often part of an initial evaluation of trauma patients</td>
<td>- presence of artherosclerosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- normal variations in anatomy</td>
</tr>
<tr>
<td></td>
<td>64-slice mlultidetector CT</td>
<td>- inappropriate timing of contrast administration</td>
</tr>
<tr>
<td></td>
<td>• Requires less doses of contrast to obtain the image of the large</td>
<td></td>
</tr>
<tr>
<td></td>
<td>anatomical areas than the 16-slice scanner does</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
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</tr>
<tr>
<td>Carotid Duplex</td>
<td>• Evaluates narrowing of the ICA and its branches in the neck</td>
<td>• Highly operator dependent</td>
</tr>
<tr>
<td></td>
<td>• Noninvasive, no risk for complications</td>
<td>• Not readily available in a trauma situation</td>
</tr>
<tr>
<td></td>
<td>• $</td>
<td>• Limited evaluation of small lesions or nonocclusive injuries with preserved flow (small intimal flaps, pseudoaneurysms, stenosis &lt;60%)</td>
</tr>
<tr>
<td></td>
<td>• ? role in follow-up assessment of previously detected BCVI</td>
<td>• Fails to adequately visualize the</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TCD</td>
<td>ICA at the base of the skull, where high incidence of BCVI occurs (low sensitivity for BCI)</td>
<td></td>
</tr>
<tr>
<td>-----</td>
<td>------------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Examines the major intracranial arteries at the base of the brain</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• It can detect turbulence and blood flow abnormalities</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• ? role in follow-up assessment of previously detected BCVI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Imaging impossible in approx. 15% of patients due to their anatomy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Difficulty visualizing the intracranial and posterior circulation</td>
<td></td>
</tr>
<tr>
<td>MRA</td>
<td>• Allows visualization of intracranial and extracranial arteries from the arch of the aorta to the circle of Willis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Used to evaluate suspected arterial stenosis, occlusions, aneurysms, dissection, and vascular malformations</td>
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</tr>
<tr>
<td></td>
<td>• Minimal risk for complications</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Extremely rare incidence of allergic reactions to gadolinium contrast agent as compared to iodine-based contrast used in conventional angiography)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Gadolinium contrast agent does not cause kidney damage</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Can be performed along with MRI</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Not readily available in trauma situation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Time-consuming</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Highly motion sensitive</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• $$$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Impracticality in trauma situation due to incompatibility with ventilator, orthopedic fixation or traction device, pacemaker, or aneurym clips</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Poor sensitivity and specificity of MRA/MRI as compared to FVCA</td>
<td></td>
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</tbody>
</table>

FVCA is an angiographic study of the four blood vessels (i.e. carotid and vertebral arteries) under fluoroscopy. Four-vessel cerebral angiography, or sometime called digital subtraction angiography, is a computer-augmented form of contrast angiography that obtains digital blood flow images of the cranial vessels as an iodine-based intravenous contrast agent courses through a blood vessel. The computer subtracts bone and soft tissues from the image, thereby improving visualization of blood vessels (Figure 6).
Computed tomographic angiography (CTA) is now believed to be even superior to FVCA owing to fewer complications, cost and increasing practicability. Since 16-slice multidetector CT scanners became readily available in the United States in 2003 and the imaging technology of CTA has advanced rapidly, many trauma centers now have 16-slice or better scanners (Biffl et al., 2002, Miller et al., 2002). This has resulted in substantial improvement in resolution and 3-D reconstruction capabilities and a growing body of evidence supports the reliability and accuracy of CTA with the latest generation imaging techniques using 16-slice or better scanner for
detecting BCVI, as compared to earlier findings using 4-slice multidetector CT scanner (Biffl et al., 2006, Berne et al., 2006, Utter, G. H., Hollingworth, W., Hallam, D. K., Jarvik, J. G. & Jurkovich, G. J. 2006, Malhotra et al., 2007, Eastman et al., 2006) (Figure 7).

Figure 7. Reconstructed image - 64-slice multidetector CT angiography showing right ICA dissection. Copyright 2010 by Kaori Bird.

The EAST guidelines currently recommend CTA using at least 8-slice multidetector technology, preferably 16-slice or better multidetector CT scanner. Recent studies have reported improved sensitivity and specificity with 16-slice scanner CTA for detecting BCVI and significantly less time (from 31.2 hours to 2.6 hours) from admission to diagnosis of BCVI-related strokes by using the CTA-based screening as compared to FVCA (Berg et al., 2005, Eastman et al., 2006, Eastman et al., 2009).
CT imaging is often part of the work-up of trauma patients, so CTA of the head and neck can be easily obtained without additional contrast administration, radiation exposure, or “road trips” to the CT suite. When only the diagnosis of BCVI is of clinical interest, it is appropriate to obtain a targeted head and neck multidetector CTA protocol using 16-slice or better scanner. Whole-body multidetector CTA as part of trauma imaging studies has been shown to demonstrate comparable accuracy to head and neck multidetector CTA in detecting BCVI (Sliker et al., 2008, Stein et al., 2009). Whole-body CTA has an advantage of the capacity to rapidly obtain contrast-enhanced large anatomical segments from the head to the pelvis with minimal intravenous contrast material dose. At present, routine use of whole-body or neck multidetector CT angiography in multitrauma patients who do not meet the screening criteria is not currently recommended. Whole-body multidetector CTA may be performed when there is a history and mechanism of trauma with possible multiple injuries, thus imaging of the cervical spine, chest, abdomen, and pelvis is also necessary. Some investigators still recommend a complete visualization of cervical and intracranial imaging with head and neck CTA in patients who meet screening criteria for BCVI (Ringer et al., 2009). Sensitivity and specificity of whole-body multidetector CTA in diagnosing BCVI have not yet been well-established. Thus, patients with abnormal findings on whole-body multidetector CTA should undergo additional confirmatory imaging study with FVCA.

Diagnosing BCVI by CTA is not without a challenge. Additional planes may be necessary if poor or suboptimal imaging technique results in poor visualization of the arteries (Malhotra et al., 2007, Sliker, 2008). Confirmatory FVCA should be obtained before subjecting a patient to treatment that has potential risks because some data suggest that the false positive rates of 16-
slice multidetector CTA may be significant (Berg et al., 2005, Mlhotra et al., 2007, Sliker, 2008, Bloomberg et al., 2010). As CT technology continues to improve and 64-slice or better CT scanners are being introduced, it is possible that the performance and accuracy of CTA will surpass FVCA in diagnosis of BCVI in near future. The modern 64-slice multidetector CT scanner can obtain complex images of the skull, aorta, and craniocervical vasculature, as well as reconstructions of the cervical vertebra and upper thorax from one examination (McKinney et al., 2007). Using more accurate CTA technology available today, potential savings and benefits of CTA as a diagnostic test may be even greater. Until further evidence from prospective studies verifying the accuracy of CTA, FVCA should be still considered the gold standard confirmatory test for diagnosing BCVI (Bromberg et al., 2007, Adams et al., 2006, Sliker, 2008).

Alternative diagnostic modalities include carotid duplex scanning, transcranial doppler ultrasonography (TCD), and magnetic resonance angiography (MRA). Carotid duplex scanning is a vascular ultrasound study which can be used to evaluate ICA stenosis, but it has very limited ability to visualize the skull base and intracranial circulation, making this modality less ideal for diagnosis of BCVI (Malhotra et al., 2007, Arthus & Starnes, 2008). TCD has been used to assess changes in blood flow velocity and vasospasm before the actual neurologic signs in patients with subarachnoid hemorrhage (Adams et al., 2006). At present, the role of TCD as part of the trauma and emergency evaluation has not been established and this is not adequate screening modality for BCVI. There may be a role of ultrasound studies such as carotid duplex scanning and TCD as a modality for follow-up assessment of previously detected BCVI (Singh et al., 2004).

Contrast-enhanced MRA obtains blood flow imaging after an injection of an intravenous contrast agent. Gadolinium, a non-ionic element, is currently used to achieve better visualization
of structures when compared to un-enhanced studies. MRI of the brain, which is often obtained along with the MRA, offers many anatomical projections and assessment of intracranial architecture, allows identification of ischemic infarction, and obtains information on the extent of cerebral infarction, associated soft tissue and ligamentous injury, and age or thickness of mural thrombus (Adams et al., 2006, McKinney et al., 2007). The available studies have demonstrated poor sensitivity and specificity of MRA/MRI as compared to FVCA (Miller et al., 2002, Bromberg et al., 2010). MRA may be a useful adjuvent diagnostic tool, but is not considered as the sole screening modality for evaluation of blunt trauma patients suspected of BCVI.

Current treatment strategies

There is no consensus or level I recommendations for the optimal treatment of BCVI (surgical intervention, endovascular therapy, or antithrombotic therapy), the appropriate angicoagulation agent (antiplatelet, clopidrel, heparin, or warfarin), the duration and end point of antithrombotic therapy. Evidence suggest that early diagnosis and treatment reduce the morbidity and mortality associated with BCVI as well as management of blunt trauma patients using standardized screening protocols and treatment guidelines have been shown to reduce the time from admission to diagnosis of BCVI (Cothren et al., 2005, Eastman et al., 2009, Bromberg et al., 2010). Although evidence suggests that treatment of BCVI does improve outcome and prevent many cases of cerebrovascular infarctions, a portion of such neurological sequelae may not be entirely preventable while a number of patients with BCVI may not be candidates for available treatment due to contraindications (Stein et al., 2009). The following table summarizes treatment strategies for each injury grade of BCVI (Table 9).
Table 9: Summary of current treatment strategies.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Treatment strategies</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Grade I | • Symptomatic patients: Antithrombotic therapy with heparin or aspirin (some authors recommend anticoagulation with heparin over aspirin unless contraindicated)  
• Asymptomatic patients: Antithrombotic therapy with aspirin or heparin                                                                                                                                                                                                               | → Heparin or antiplatelet therapy appear equivalent  
→ Heparin infusion without a bolus  
→ A guideline for aPTT goal has not been determined  
→ No recommendations regarding the specific length of antithrombotic therapy  
→ Heparin to warfarin conversion with INR goal 2-3 for the duration of 3-6 months  
→ Antithrombotic therapy for 3-6 months is recommended unless follow-up studies demonstrate complete healing                                                                                                    |
| Grade II| • Antithrombotic therapy with heparin or aspirin  
• Consider surgical repair if accessible or endovascular therapy if inaccessible (stent placement, coil embolization)                                                                                                                                                                         | → Antithrombotic therapy for 3-6 months is recommended unless follow-up studies demonstrate complete healing  
→ Wait several days to perform an invasive procedure due to an increased risk of stroke from manipulation of angiocatheter in the acutely injured vessel during the initial 48-72 hours  
→ Periprocedural antiplatelet therapy (aspirin +/- clopidogrel) for at least 4-6 weeks (+ possible lifelong aspirin therapy) if an endovascular stent placement unless contraindicated                                                                 |
| Grade III| • Antithrombotic therapy with heparin or aspirin  
• Consider invasive therapy, especially if symptomatic or persistent pseudoaneurysm  
• Endovascular therapy for inaccessible lesions, coil embolization                                                                                                                                                                                                                       | → Pseudoaneurysm rarely resolve with no therapy or heparin therapy, so consider invasive therapy  
→ Wait several days to perform an invasive procedure due to an increased risk of stroke from manipulation of angiocatheter in the acutely injured vessel                                                                 |
to excluding pseudoaneurysm from the circulation and/or stenting to maintain vascular patency
- Surgery repair if accessible
during the initial 48-72 hours
→ Periprocedural antiplatelet therapy (aspirin +/- clopidogrel) for at least 4-6 weeks (+ possible lifelong aspirin therapy) if an endovascular stent placement unless contraindicated

Grade IV
- Consider surgical repair if accessible
- Consider anticoagulation therapy
- May consider endovascular therapy with stenting to maintain vascular patency if not candidates for surgical repair or anticoagulation therapy
→ Grade IV lesions rarely improve with antithrombotic therapy
→ Periprocedural antiplatelet therapy (aspirin +/- clopidogrel) for at least 4-6 weeks (+ possible lifelong aspirin therapy) if an endovascular stent placement unless contraindicated

Grade V
- Transection with free extravasation may require immediate operative repair if surgically accessible.
- Endovascular therapy may or may not be possible
- Consider surgical repair or endovascular embolization to occlude the parent artery in unstable AVF
→ 100% stroke rate and poor outcomes

**Surgical repair**

The EAST guidelines make a weak recommendation for operative repair for patients with early neurologic deficit, an accessible carotid injury, and persistent pseudoaneurysm despite antithrombotic therapy with high risk for subsequent embolization or rupture (Bromberg et al., 2010). Surgical intervention may also be considered for a large expanding cervical hematoma, and selected cases of surgically accessible Grade II, III, IV lesions (Singh et al., 2004). Other factors such as worsening neurological symptoms despite medical therapy as well as poor collateral circulation to the brain may support the decision to pursue surgical intervention (Singh et al., 2004). Several surgical techniques such as cervical (extracranial) to intracranial bypass, carotid ligation or balloon occlusion, resection, reconstruction with grafts, or carotid
endarterectomy may be considered to repair the lesions (Schievink, W. I., Piepgras, D. G., McCaffrey, T. V. & Mokri, B. 1994).

Surgical repair is difficult due to the fragile nature of the aneurysm and often complicated by extension of the injury to the base of the skull. Complications such as cranial nerve neuropraxia due to extensive surgical exposure of the artery as well as stroke and restenosis have been reported (Schievink et al., 1994). Direct surgical repair with high cervical approach has been particularly associated with complications such as damage to the pharyngeal and superior laryngeal branches of the vagus nerve, cranial nerve palsies and deafness (Singh et al., 2004). Patients are at increased risk for surgical complications if there is severe blockage in other blood vessels that supply blood to the brain, such as the carotid artery on the other side. An increased risk of developing cerebral ischemia and intracranial aneurysm later in life in patients who undergone surgical repair has been reported (Singh et al., 2004). Surgery carries high risk for morbidity and may be best performed by an experienced vascular surgeon. Due to technical demands and complications, surgical intervention may be best reserved for those patients who are symptomatic, but cannot be managed effectively with other therapy. Hemodynamically unstable traumatic arteriovenous fistula (AVF) may require individualized management based on symptoms and radiological findings and may include medical management, surgical repair or endovascular therapy.

**Antithrombotic therapy**

Outcomes of patients with BCVI have dramatically improved since the recognition of antithrombotic agent as effective treatment in reducing thromboembolic sequelae associated with BCVI. The evidence supports early treatment with antithrombotic therapy for patients with
Grade I and II injuries who do not have contraindications (Bromberg et al., 2010). Prompt diagnosis of BCVI and treatment with antithrombotic therapy have been shown to decrease the ischemic neurologic events and improve outcome in patient with BCVI when compared with no intervention (Fabian et al., 1996, Biffl et al., 1998, Miller et al., 2001, Cothren et al., 2004, Cothren et al., 2005). The EAST guidelines recommend either heparin or antiplatelet therapy for treatment of BCVI (Bromberg et al., 2010). Efficacy of anticoagulation therapy and antiplatelet therapy has been found equivocal. Antithrombotic agents are particularly effective at treating lesions in surgically inaccessible areas. There may be a role of prophylactic therapy with antithrombotic agent, or at the very least protective effect from possible ischemic neurologic events in asymptomatic patients. However, antithrombotic therapy are often ineffective in resolving Grade IV lesions while it may be unsuitable for Grade V transection injuries or cerebral infarctions. Contraindications to antithrombotic therapy have not been clearly defined. Factors such as presence of traumatic brain injury, complex cervical spine fractures, evidence of massive stroke on admission, solid organ injuries, multiple orthopedic injuries as well as patient's overall condition should be evaluated by the attending interdisciplinary team in order to determine the appropriateness of initial treatment for BCVI (Stein et al., 2009, Eastman et al., 2009). Antithrombotic therapy may be initiated after the stabilization of concomitant injuries in selected cases.

Heparin is currently first line therapy for BCVI and is thought to stabilize and resolve clot formed as a result of BCVI through intrinsic fibrinolytic mechanisms and prevents further thrombosis (Fabian et al., 1996, Stein et al., 2009). Early initiation of anticoagulation therapy with heparin has been shown to reduce neurological morbidity and mortality in both
symptomatic and asymptomatic patients with angiographically-confirmed BCVI (Fabian et al., 1996, Biffl et al., 1998, Miller et al., 2001). Intravenous heparin infusion should be started without a bolus with PTT goal of 50-60 seconds (some authors recommend PTT goal of 40-50 seconds) and heparin therapy converted to warfarin therapy to target INR of 2 to 3 for the duration of 3 to 6 months (Biffl et al., 2002, Eastman et al., 2009, Bromberg et al., 2010). No definitive target PTT is established by the EAST guidelines at this time. However, systemic heparin therapy is often contraindicated in trauma patients due to coexisting injuries. Serious bleeding complications have been reported with systemic anticoagulation with heparin therapy for BCVI (Biffl et al., 1999, Wahl, W. L., Brandt, M. M., Thompson, B. G., Taheri, P. A. & Greenfield, L. J. 2002, Edward et al., 2007). Intracranial hemorrhage, blunt aortic injury, recent surgical wounds, solid organ injury, and skeletal or soft tissue injury with high risk for hemorrhage are considered as contraindications to heparin therapy.

Unsuitability of heparin therapy in many trauma patients, fueled by the introduction of new antiplatelet agents (e.g. clopidogrel), has led to investigations of the efficacy of antiplatelet agents as an alternative to heparin. Evidence-based guidelines for antithrombotic therapy of BCVI based on prospective, randomized trials has not been established to this date. Current practice on antiplatelet therapy for trauma patients with BCVI is largely extrapolated from available data on medical patients with noncardioembolic ischemic stroke or transient ischemic attack (TIA). Results from the recent Cochrane review involving medical patients with carotid artery dissections found that equivocal efficacy between aspirin and heparin and suggested aspirin with a lower risk of hemorrhage among the medical population (Lyrer, P. & Engelter, S. 2003). Although results have been somewhat inconclusive, aspirin monotherapy (50 to 325
mg/d), the combination of aspirin and extended-release dipyridamole (ER-DP) (Aggrenox), or clopidogrel monotherapy (75mg/day) are all considered acceptable options in preventing ischemic neurlogic events for these non-trauma population (Bhatt et al., 2006, Kennedy et al., 2007, Adams et al., 2008). The combination therapy with aspirin plus clopidogrel has been recommended for medical patients with carotid artery disease, who also have concomitant symptomatic coronary artery disease, recent coronary stenting, and severe peripheral arterial disease (Adams et al., 2008).

The EAST guidelines recommend antiplatelet agents for patients with relative contraindications to heparin (Bromberg, et al., 2010). Antiplatelet therapy, particularly in patients with contraindications to systemic anticoagulation therapy with heparin, may be as effective as heparin therapy in reducing incidence of stroke associated with BCVI (Miller et al., 2001, Miller et al., 2002, Wahl et al., 2002, Edward et al., 2007). Antiplatelet therapy with aspirin (325mg/day) or clopidogrel (75mg/day) should be recommended for BCVI. For patients with concomitant traumatic brain injury and/or spinal cord injury, antiplatelet agent, particularly aspirin monotherapy, may be considered after the stabilization of these injuries, although safety of such therapy has not been well established (Stein et al., 2009). Safety profile of aspirin and clopidogrel appears equivalent while there is insufficient data to determine effects of Aggrenox in trauma patients with BCVI at this time. The addition of clopidogrel to aspirin increases the risk of bleeding complications in trauma patients with BCVI who have sustained concomitant injuries or who may need to undergo invasive procedures and is not recommended as a routine therapy (Bhatt et al., 2006).

The duration and end-point of antiplatelet therapy for prevention of thromboembolic
events associated with BCVI are not well established. In the absence of concomitant injuries or contraindications, antiplatelet therapy should be started immediately. Long-term antithrombotic therapy (i.e. 3-6 months) is recommended unless follow-up studies demonstrate complete healing. The end-point of therapy in some patients may be an evidence of healed lesion on follow-up cerebral angiography, which would eliminate a future risk of progressing to a more severe injury. Side effects and costs of medications, comorbid illnesses as well as patient's compliance with medication regimen, and ability to maintain long-term follow-up with health care provider will likely influence decisions regarding antithrombotic therapy.

**Endovascular therapy**

Endovascular therapy offers a less invasive option than surgical open repair for patients with high surgical risks or contraindications for other treatment. Endovascular therapy may be indicated for chronic dissections that progress and limit blood flow and Grade I and II injuries which progress to pseudoaneurysms despite antithrombotic therapy, as well as completely occlusive lesions and some cases of vessel transection (Fabian et al., 1996, Edward et al., 2007). Enlarging pseudoaneurysms have a high risk of rupture and endovascular embolization with coil, balloon, or glue may be used to occlude bleeding from the parent artery. Endovascular stenting and coil embolization have been performed successfully to treat Grade III pseudoaneurysms as well as Grade IV injuries (Klein et al., 1997, Biffl et al., 1999, Berne, et al., 2008, Stein et al., 2009).

The EAST guidelines recommend endovascular therapy with subsequent antiplatelet therapy for unresolving Grade III injuries (Bromberg et al., 2010). However, current available data regarding the efficacy and safety of endovascular therapy for BCVI is insufficient, thus the
controversy surrounding its use. Recent studies have shown that endovascular stenting coupled with early antithrombotic therapy is both safe and effective in patients with severe dissections or pseudoaneurysms, particularly Grade III BCI because these lesions are often located at the skull base below or within the carotid canal of the petrous bone and surgically inaccessible (Edward et al., 2007, Berne et al., 2008). There is an increased risk of embolization during angiography and stenting (Cothren et al., 2004). Thus, endovascular stent placement should be delayed for 7-10 days in selected asymptomatic, stable patients to allow the lesion to stabilize, heal without treatment, or improve on antithrombotic therapy.

The ideal antithrombotic therapy for endovascular stenting, how long the patients should be kept on such therapy, and long-term patency rate of endovascular stents for BCVI have not been well established. The current practice in periprocedural antithrombotic therapy in endovascular stent therapy for BCVI is extrapolated from the experience gained in percutaneous coronary interventions for acute coronary syndromes and carotid artery stenting for atherosclerotic disease and prevention of ischemic stroke in these patients (Bhatt et al., 2001, Yadav et al., 2004, Chaturvedi & Yadav, 2006, Kramer, J., Abraham, J., Teven, C. M., & Jones, P. A. 2007). Studies involving patients with BCVI treated with endovascular stents have demonstrated an increased rate of thrombotic events associated with early discontinuing antiplatelet therapy and potential beneficial effects of antiplatelet therapy with a regimen of aspirin plus clopidogrel for a minimum of 6 weeks and aspirin daily for life (Duane, T. M., Parker, F., Stokes, G. K. Parent, F. N., & Britt, L. D. 2002, Edward et al., 2007). Based on these evidence, all patients who have received endovascular stents for BCVI should receive periprocedural antiplatelet therapy with aspirin and/or clopidogrel for at least 4 to 6 weeks and
possible lifelong aspirin therapy unless contraindicated. At this time, there is insufficient data to
determine whether other antipletelet agent (e.g. Aggrenox) or intravenous GPIIB/IIIA inhibitor
(e.g. eptifibatide) is an alternative therapy (Kramer et al., 2007).

Complications of endovascular therapy include bleeding, thromboembolism, dissection,
restenosis, fracture or compression of the stent, and complications related to the dye used during
the angiogram. The addition of clopidogrel to aspirin does increase the risk of hemorrhagic
complications in trauma patients and might impair hemostasis in patients undergoing other
invasive procedures. Trauma patients with BCVI are best cared for at trauma centers with
neurosurgeon or interventional neuroradiologist who has extensive experience with endovascular
stenting (Edward et al., 2007). Post-stent management of patients with BCVI includes
antithrombotic therapy and close monitoring to prevent complications and reduce vascular risk
factors for thromboembolic events and re-stenosis. The modern endovascular therapy technology
and skilled practitioners, coupled with effective antithrombotic therapy have improved outcome
and made this approach an alternative treatment strategy for BCVI. Evidence regarding safety
and feasibility of endovascular therapy is preliminary, and durability and long-term ability of this
therapy to prevent stroke are still under investigation.

Outcomes and follow-up

There is no consensus on definitive follow-up and management of BCVI and the EAST
guidelines lack level I recommendations regarding how to best monitor the patient's response to
therapy. Based on available data on outcomes observed in studies involving patients with BCVI,
investigators and experts have made following recommendations (Table 10).
Table 10: Findings on follow-up angiography.

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<th>Grade</th>
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| Grade I | • 14% risk of developing pseudoaneurysms (Biffl et al., 2002)  
• Nearly half healed completely, thus did not require additional antithrombotic therapy based on follow-up angiography at 7-10 days (Biffl et al., 2002)  
• 72% improved on follow-up angiography at 4 months, thus discontinuing antithrombotic therapy (Edward et al., 2007) |
| Grade II | • 47% risk of developing pseudoaneurysms (Biffl et al., 2002)  
• A small % at high risk of thrombosis (Biffl et al., 2002)  
• Nearly 1/3 required surgical intervention or endovascular therapy due to formation of pseudoaneurysms, unstable, or worsening pseudoaneurysms based on follow-up angiography at several months (Edward et al., 2007)  
• Nearly half healed completely, thus did not require additional antithrombotic therapy based on follow-up angiography at 7-10 days (Biffl et al., 2002)  
• 66% improved on follow-up angiography at 4 months, thus discontinuing antithrombotic therapy (Edward et al., 2007) |
| Grade III | • A small % at high risk of thrombosis (Biffl et al., 2002)  
• Nearly 90% required surgical intervention or endovascular therapy due to formation of pseudoaneurysms, unstable, or worsening pseudoaneurysms based on follow-up angiography at several months (Edward et al., 2007)  
• Initial injury did not resolve on follow-up angiography at 7-10 days (Biffl et al., 2002) |
| Grade IV | • Initial injuries did not resolve on follow-up angiography at 7-10 days (Biffl et al., 2002)  
• Majority of injuries remain occluded and unchanged at 4 months |

Currently, there are no consensus for the follow-up criteria, specific timing intervals, or type of imaging for BCVI. A cost-risk-benefit profile of follow-up imaging in asymptomatic patients with angiographically confirmed cases of BCVI has not been entirely established (Biffl et al., 2000, Kerwin et al., 2001, Miller et al., 2001). The EAST guidelines recommend follow-up imaging using FVCA within 7-10 days after injury in Grade I-III BCVI to monitor the
progression of the untreated injury or the response to initial treatment (level II recommendation) (Biffl et al., 2002, Bromberg et al., 2010). Both untreated and treated BCVI have been shown to either progress or regress over the course of days or weeks. Grade I injuries are at risk for progressing to pseudoaneurysms and Grade II lesions are even more likely to progress to pseudoaneurysms or developing thromboembolic event (Biffl et al., 2002). Thus, symptomatic or asymptomatic, patients with Grade I and II BCVI should be closely monitored for ischemic symptoms due to risks of progression to pseudoaneurysms and resultant thromboembolic events or hemorrhage due to rupture.

Follow-up imaging should be obtained for all patients diagnosed with BCVI, and may be considered for patients who meet at least one of the screening criteria for BCVI. Reevaluation of the injury with follow-up imaging at 7-10 days or whenever patient's condition changes, close monitoring and prompt adjustment in antithrombotic therapy, and re-evaluation for alternative interventions such as surgical repair and endovascular therapy are warranted for all patients with BCVI. Follow-up FVCA should be not be performed before 7 days post injury due to risk of angiography-related complications such as an embolization of an unstable thrombi (Biffl et al., 2002, Bromberg et al., 2007). In patients receiving pharmacological treatment, FVCA should be performed in 3 to 6 months to determine whether these medications can be discontinued although less invasive imaging modalities such as CTA or MRA may also be considered when appropriate for follow-up.
CHAPTER 7: THE ROLE OF ACNP IN THE CARE OF TRAUMA PATIENT WITH POSSIBLE BCVI

ACNPs are increasingly recognized as an integral part of the trauma care delivery system in many institutions in the United States. Historically nurse practitioners have worked in rural areas where they were initially supplied to assist the shortage of physicians, but the scope and practice setting have expanded to include a variety of clinical settings, such as major trauma centers and acute care hospitals (Sole, M. L., Hunkar-Huie, A. M., Schiller, J. S. & Cheatham, M. L. 2001). Today, ACNPs care for patients with a variety of complex injuries who have unique, specialized needs, such as neurological injury with traumatic brain injury and multiple organ involvement (Sole et al., 2001, Yeager S., Shaw, K. D., Casavant, J., & Burns S. M. 2006).

The benefits of utilizing nurse practitioners in a hospital trauma service have been documented in the literature as early as in 1980 by a landmark study by Spisso and colleagues (Spisso, J., O'Callaghan, C., McKennan, M., & Holcroft, J. 1980) and more recently by Jarrett & Emmett (2009). Data from recent studies also found that pediatric trauma patients who received care by the group of pediatric trauma nurse practitioners had a shorter length of stay, higher patient satisfaction, and positive clinical and functional outcomes compared to patients who received care by the traditional treatment team without nurse practitioners (Fanta et al., 2006, Schweer, L., Cook, B. S., & Fanta, K. B. 2004). The effectiveness of the ACNP role in managing patients at risk for clinical deterioration and benefits of preventing complications have been demonstrated in terms of both financial and clinical outcomes (Russell, D., VorderBruegge, M., & Burns, S. 2002, Yeager et al., 2006). Despite these positive findings, only a handful of researchers have explored the topics of advanced practice nursing in trauma care and little has
been published regarding the role of the ACNP in the care of specific injuries in the trauma population. ACNP's managing neurosurgical patients in acute care settings and management strategies of patients with suspected cervical spine injuries by advanced practice nurses in the emergency department have been discussed in recent nursing journals (Yeager et al., 2006, Ramirez & Flarity, 2009).

Responsibilities of ACNP in the care of trauma patients are diverse and vary depending on practice settings. Specific services may include assessment and management of traumatic injuries, interpretation of diagnostic data, cervical spine clearance, wound care, collaboration with physicians, ongoing trauma evaluation and management of complications, meeting psychosocial and educational needs, and coordination of discharge (Spisso et al., 1980, Sole et al., 2001, Schweer et al., 2004). As expert clinicians, ACNPs function beyond direct patient care by developing and implementing protocols, sharing knowledge and collaborating with colleagues, and evaluating institutional standards to improve specific care provided to patients (Yeager et al., 2006, Chakravarthy, 2008). ACNPs in acute care settings are often physically present at bedside as a core member of trauma service, intensive care team, or neurosurgical unit to evaluate patients and provide immediate care (Yeager et al., 2006). Trauma patients in many institutions are followed by nurse practitioners along the continuum from initial resuscitation to discharge, and even after discharge during follow-up in outpatient clinic (Chakravarthy, 2008, Jarrett & Emmett, 2009). Thus, ACNPs are in the ideal position to provide consistent, high-quality care to the trauma patients and meet their complex needs.

To our knowledge, the role of the ACNP in the management of patients with BCVI has not been previously described. Given the complexity and diverse pathophysiology encountered
in the trauma patient population as well as the evolving roles and responsibilities of ACNPs, the information on BCVI presented in this article may positively impact clinical practice in a variety of settings. ACNPs equipped with advanced clinical knowledge can effectively manage trauma patients with BCVI and are perfectly situated to institute evidence-based protocols in consultation with the attending interdisciplinary team, which may include trauma surgery, neurosurgery, neurology, neuroradiology, vascular surgery, interventional radiology, and nursing staff.

The ACNP may encounter trauma patients with BCVI in many settings, including during an initial encounter at a trauma center, in an emergency department in a non-trauma center or in a rural area, or in inpatient hospital settings. Due to the shortening of hospital lengths of stay, the ACNP may also see these patients during follow-up imaging and evaluation at rehabilitation facility or or in an outpatient clinic after discharge. High index of suspicion for BCVI, comprehensive assessment of injuries, evaluation of patients with history of trauma, and use of evidence-based screening criteria and diagnostic tools are key features of ACNP in approaching patients at risk for BCVI. Following a diagnosis of BCVI, ACNP should carefully select patients who have increased risk of developing devastating neurological consequences and thus, benefit from immediate interventions, close observation, and long-term follow-up. Side effects and costs of medications, comorbid illnesses, patient's compliance with medication regimen, and ability to maintain long-term follow-up with health care provider will all likely influence decisions regarding specific therapy. ACNP has an important role in management of preexisting psychosocial-medical problems, reduction of risk factors for strokes, and injury prevention. ACNP uses holistic approach to evaluate such factors as preexisting psychosocial issues as well
as medical problems in these trauma patients. The unique role of ACNP as an independent clinician and a collaborator with bedside nurses and other health care providers allows prompt diagnosis and treatment of patients suspected of BCVI. If the patient with BCVI suddenly deteriorates, the ACNP may order appropriate diagnostic tests and interventions, collaborate with the interdisciplinary team, and initiate consultation with interventional neuroradiologist or vascular surgeon.

An algorithm presented in this article was developed to assist practicing ACNPs in care of trauma patients with possible BCVI (Figure 8). It summarizes the available data based on literature review. Throughout the algorithm, the role and important features of ACNP in the management of trauma patients with possible BCVI have been integrated. The first part of the algorithm summarizes the important elements of BCVI including signs and symptoms, and risk factors for BCVI and criteria for screening trauma patients. It is designed to help select optimal diagnostic tool and implement appropriate treatment based on available evidence and clinical presentation of individual patient. It guides ACNP in risk stratification and evaluation of benefits and risks, as well as factors to be considered when determining initial management strategies. This is the second part of the algorithm is designed to assist ACNP in selecting appropriate therapy based on the Denver injury grading scale. It explains recommendations for pharmacologic therapies, endovascular intervention, appropriate periprocedural care and follow-up care. These would guide discharge planning and patients and family education. The algorithm should guide practitioners in a variety of clinical settings -- identifying patients at risk for BCVI, managing those diagnosed with BCVI, selecting and implementing appropriate treatment, managing post-operatively or post-endovascular therapy, monitoring for complications,
providing follow-up care, educating patients and family, and coordinating comprehensive discharge plan. Successful integration of the role of ACNP in the care of trauma patients with BCVI will improve resource utilization, decrease lengths of stay, increase patient satisfaction, and improve patient outcome.
The Role of ACNP in the Management of Trauma Patients with Possible BCVI

**Signs and Symptoms of BCVI**
- Neurologic findings unexplained by brain imaging or other injuries or diagnoses (herniation or central neurologic deficit, TIA or NHT, Horner's syndrome, anosmia, GCS ≤ 8)
- Massive epidural or subdural hemorrhage from a suspected arterial source after blunt trauma
- Evidence of stroke on brain imaging
- Cervical bruising in patient ≤ 30 yo
- Expanding cervical hematomas

**Risk Factors for BCVI**
- Asymptomatic patients with significant blunt head trauma with following risk factors:
  - GCS ≤ 8
  - Petrous bone fracture
  - Basilar skull fracture with carotid canal involvement
  - Dense second injury
  - Cervical spine fracture particularly those with (i) fracture of C1 to C3 and (ii) fracture through the transverse foramen
  - Cervical spine fracture with subluxation or rotational component
  - Displaced midface fracture (Le Fort II or III)
  - Cervical type neck injury with significant anterior neck soft tissue injury (seethrough sign above the clavicle, near-hanging, or significant cervical hematoma)
  - Injury mechanism compatible with severe cervical hyperextension, rotation, hyperflexion, or intramural trauma

**Equivocal or abnormal findings**
- Screen for BCVI with brisk or better (16-slice preferable) CTA
- Observe with frequent neurovascular checks

**Benefits > Risks of diagnostic four-vessel cerebral angiography (FVCA) AND available interventions (Consider transcatheter antithrombotic therapy?)**

**Diagnostic Therapeutic FVCA**

- **Grade I**: Irregularity of the vessel wall or a dissection intramural hematoma with less than 25% luminal narrowing
  - **Tx:** Consider: Antithrombotic therapy with heparin, aspirin (100 mg daily), or clopidogrel (75 mg daily)
  - Heparin infusion at INR goal 2-3
  - Consider warfarin with INR goal 2-3 for duration of 3-6 months
  - Consider transcatheter embolization of angiography

- **Grade II**: A raised intimal flap, a dissection, intramural hematoma, or intramural hematoma with greater than 25% luminal stenosis, or hemodynamically significant AVFs
  - **Tx:** Consider: Antithrombotic therapy with heparin, aspirin, or clopidogrel
  - Surgical repair if accessible or endovascular therapy if inaccessible

- **Grade III**: Pseudoaneurysm
  - **Tx:** Consider: Antithrombotic therapy with heparin, aspirin, or clopidogrel
  - Invasive therapy, especially if symptomatic or persistent (rarely resolve with observation or heparin therapy alone)
  - Endovascular therapy for inaccessible lesions
  - Embolization to exclude pseudoaneurysm from the circulation and/or stenting to maintain vascular patency

- **Grade IV**: Vessel occlusion
  - **Tx:** Consider: Anticoagulation + endovascular therapy
  - Surgical repair if accessible

- **Follow-up FVCA in 1-10 days**
  - Discontinue antithrombotic therapy if evidence of healed injury. If not, continue antithrombotic therapy for 3-6 months
  - Obtain FVCA in 3-6 months and re-evaluate the course of therapy (life-long aspirin therapy). *CTA or MRA may be considered for follow-up imaging.

The key features of ACNP include patient/family education, staff education, and coordination of comprehensive discharge plan.
Figure 8. FIGURE 8. The role of ACNP in the management of trauma patients with possible BCVI. Copyright 2010 by Kaori Bird.
CHAPTER 8: CONCLUSION

Patients with BCVI can initially present with seemingly minor injuries. However, as the opening case has illustrated, these injuries can lead to debilitating neurological complications or even death if left undetected. While patients with BCVI make up a small percentage of the trauma patient population, the high morbidity and mortality associated with BCVI demands immediate and long-term resources as well as coordinated care in order to prevent permanent physical and neurological disability. The consequence of undiagnosed BCVI and subsequent functional loss in this primarily young adult population cannot be overstated. Initial management of trauma patients should include prompt recognition and evaluation of patients who are at risk for BCVI, accurate diagnosis, risk stratification of neurological consequences, identification of therapeutic options, and careful selection of patients who would benefit from appropriate interventions. Close observation and serial neurological and neurovascular examinations of patients who are at risk of BCVI are the key factors in improving detection of this injury which carries risk of serious sequelae. ACNP's involved in the care of trauma patients should understand the mechanism and nature of injury unique to BCVI and manage these injuries using an evidence-based screening protocol and algorithm that are appropriate for his/her clinical practice.

Currently, there is no consensus or guidelines which direct outline the definitive management of BCVI. This article has underscored the importance of prompt screening and diagnosis of BCVI, analysis of risks and benefits of available therapies, and initiation of appropriate treatment and follow-up care in hopes of reducing complications and mortality from BCVI. ACNP must integrate the knowledge gained and the algorithm presented in this article
into clinical settings to guide practice and implement strategies to improve outcomes. In conclusion, more studies are needed to determine the evidence-based screening protocols, ideal diagnostic strategies, and optimal treatment for BCVI. Furthermore, there is need for randomized, prospective study to determine the long-term clinical course of patients with BCVI, angiographic progression of these injuries, functional outcomes of these patients, and efficacy of various therapies. The impact of ACNP in the management of trauma patients with BCVI remains to be seen and this is an opportunity for ACNP to get involved in research and process improvement projects. ACNP may have an important role in improving patient compliance and follow-up surveillance through staff training as well as patient and family education. As expert clinicians, ACNP may function beyond direct patient care by evaluating institutional standards or developing and implementing protocols to improve specific care provided to trauma patients with possible BCVI. ACNP may take initiatives in future research to examine the positive impact of preventative approach and health promotion by ACNP and to investigate the role of ACNP in trauma settings, which may include effective management of preexisting diseases and reduction of risk factors for future strokes.
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