

UNIVERSITY OF CINCINNATI MEDICAL CENTER

Guidelines on the Management of Blunt Cerebrovascular Injury (BCVI)

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References: Bromberg et al. J Trauma 2010;68:471

Burlew et al. Surg Clin N Am 2011;91:217

Morton et al. J Neurosurg 2014;120:1446

Tables: Three

Algorithms: Three

MANAGEMENT OF BLUNT CEREBROVASCULAR INJURY (BCVI)

Purpose: This guideline will describe the screening, diagnosis and management of patients with suspected BCVI.

Scope: This protocol will be applicable to all patients presenting with suspected BCVI

Background: The incidence of BCVI in the United States is reported to affect 1 in 1000 hospitalized trauma patients. This incidence has increased to 1% in past decades primarily because of the significant improvements in non-invasive imaging that detect asymptomatic injuries to the carotid and vertebral arteries. When detected as a symptomatic lesion, neurologic morbidity can approach 80% with an associated mortality of almost 40%. The current literature only provides moderate guidance as to the screening and management strategies of this patient population. This guideline addresses the methods by which patients with suspected BCVI should be screened, the modalities used to screen them and the subsequent short-term and long-term management strategies.

Guidelines:

1.0 Evaluation and Screening

- 1.1 Emergency Room Evaluation – Patients presenting with a suspicion of BCVI based on injury characteristics (**Table 1.1.A**) or patients with neurologic signs and symptoms inconsistent with the presenting injury should be imaged and screened for BCVI (**Table 1.1.B**). “Asymptomatic” patients with significant head trauma and findings consistent with a high force of impact should also be considered and screened for BCVI (**Table 1.1.B**).

TABLE 1.1.A

Mechanisms suspicious for BCVI
HIGH ENERGY IMPACT ASSOCIATED WITH: GCS \leq 8 LeFort II or III fractures Skull base fracture (e.g. petrous fracture) Diffuse Axonal Injury High C-spine fracture (Occipital condyle, C1-C3) Cervical vertebral body fracture or fracture through foramen transversarium Subluxation or ligamentous injury at any level Significant thoracic/cardiac blunt force trauma

TABLE 1.1.B

Signs/Symptoms suspicious for BCVI
Arterial bleeding from nose, mouth, neck Audible neck bruit Skull base fracture (e.g. petrous fracture) Base of Skull Fractures High C-spine fracture Expanding neck hematoma Focal neurological deficit to include TIAs, Horner’s syndrome, Vertebrobasilar syndrome

- 1.2 **Imaging** – In the setting of suspected BCVI at UCMC, a 64-slice CT Angiogram (CTA) of the head and neck to include the arch is warranted unless absolutely contraindicated (patient is unstable). This study should be obtained as quickly as is feasible and is dependent on the condition of the patient. In the setting of equivocal results from the CTA, a four-vessel angiogram may be indicated (**Figure 1.0.A**).
- 1.3 **Consultation Services** – Upon suspicion of symptomatic BCVI, upon identification of BCVI on non-invasive imaging in the asymptomatic population, or upon equivocal results on non-invasive imaging studies, the Neurotrauma Service (Neurosurgery on-call) shall be contacted. A formal consult shall be performed, Neurovascular/Endovascular will be engaged, and a plan will be documented and the patient shall be followed for the course of the patient’s hospitalization. In the setting of an asymptomatic patient that suddenly becomes symptomatic, the neurosurgery resident AND the endovascular attending should be contacted for immediate patient evaluation.

2.0 *Management*

BCVI should be graded by the Denver Grading Scale for Blunt Cerebrovascular Injuries (**Table 2.1.A**)

TABLE 2.1.A

2.1 **Asymptomatic Patient** – Presence of BCVI in the absence of neurological exam or imaging

Denver Grading Scale for BCVI
Grade I: Intimal irregularity with < 25% luminal narrowing
Grade II: Dissection/intramural hematoma and >25% narrowing
Grade III: Pseudoaneurysm
Grade IV: Occlusion
Grade V: Transection with extravasation

consistent with stroke (**Figure 2.1.A**)

A **Grade I asymptomatic injury** should be managed with antithrombotic therapy. Aspirin 325mg po/pr should be initiated unless there are relative contraindications as deemed by the consensus of the trauma, neurotrauma and neurovascular teams. Other anti-platelet agents can be considered in the setting of ASA allergy. Response to ASA administration can be performed via a platelet aggregation assay (e.g. VerifyNow) within 24 hours of the initial dose of aspirin. In the setting of contraindications to aspirin, the first line treatment to be considered in the acute phase should be heparin as it can easily be titrated on and off.

Heparin should be initiated at the low dose no bolus protocol with a PTT goal of 60-80. Once the patient is stable and is not at risk of hemorrhagic complications, patients being treated with heparin can be converted to ASA. Patients should be observed for any changes in neurological status over the course of their hospitalization. Non-invasive repeat imaging should be performed in 7 days if the patient remains asymptomatic to determine if there is interval resolution, or worsening of the lesion.

A **Grade II asymptomatic injury** should be managed with antithrombotic therapy as in Grade I asymptomatic injuries above. Additionally, if the luminal narrowing measures > 70%, consideration should be given towards performing a CT perfusion study +/- four-vessel angiogram to determine vascular reserve and collateral flow. If there is radiographic evidence of thrombus, heparin should serve as a first-line therapy unless contraindicated. In addition, heparin may be appropriate in cases of severe luminal narrowing or irregularity which carry a higher risk of thrombus formation. Non-invasive repeat imaging should be performed in 7 days if the patient remains asymptomatic to determine if there is interval resolution, or worsening of the lesion. If the thrombus has resolved, a transition from heparin to aspirin should be considered. If no resolution consider long-term anticoagulation.

A **Grade III asymptomatic injury** should be managed with anti-platelet therapy unless there are contraindications. Consideration should be given towards performing a four-vessel angiogram to determine vascular reserve, collateral flow, better characterize the lesion, and safety of anticoagulation therapy.

A **Grade IV asymptomatic injury** should be managed with anti-platelet therapy. If there are contraindications to aspirin or there is a clinical concern for the development of stump emboli, heparin should be considered. Additionally, a CT perfusion study +/- four-vessel angiogram should be performed to determine vascular reserve and collateral flow.

2.2 Symptomatic Patient – **Symptomatic BCVI** is defined as neurologic exam or imaging consistent with stroke (**Figure 2.2A**)

Grade I patients should undergo MRI brain (stroke protocol) to characterize the extent of infarct and presence of hemorrhagic change. Patients should all be started on antiplatelet therapy, unless there are contraindications. If heparin is indicated, it should be initiated with the low dose, no bolus protocol with a target PTT of 60-80. If patient cannot be monitored clinically, repeat parenchymal imaging (MRI, CT) in 3 days to detect the potential accumulation of infarcts should be performed. Transition from antiplatelet to anticoagulation should be considered if evidence of failed aspirin therapy on repeat imaging. In all cases, follow-up vascular imaging should be performed in 7 days.

Grade II - IV should be managed as symptomatic Grade I patients above. In addition, patients should undergo CT perfusion and/or four-vessel angiography. Patients with recurrent or unstable neurological symptoms shall be considered for open or endovascular repair as per the Neurovascular/Endovascular team. When contacting the Neurovascular/Endovascular team for repair, considerations should be made as to safety of long-term Plavix (or equivalent) therapy.

Grade V patients require emergent open reconstruction.

3.0 Disposition

3.1 In-hospital

All patients with symptomatic BCVI should be admitted. Those with asymptomatic BCVI Grade II with 70% stenosis, Grade III or IV need admission.

- 3.1.1 **Asymptomatic patients** -- Patients that have an isolated, asymptomatic BCVI may be triaged to a step-down unit for one day and then to the floor if no significant changes occur. Polytrauma patients in the setting of asymptomatic BCVI should be triaged according to their principal injuries. The Neurovascular team shall continue to follow these patients in consultation with the primary service.
- 3.1.2 **Symptomatic patients** – Patients with neurologic findings referable to BCVI and without significant polytrauma may be transferred to the Neurosurgical/Neurovascular service for further management. Polytrauma patients in the setting of symptomatic BCVI should be triaged according to their principal injuries. The Neurovascular team shall continue to follow these patients in consultation with the primary service.
- 3.1.3 **Symptomatic patients that undergo open surgical or endovascular treatment** – Patients shall be admitted to the Neurosurgery/Neurovascular team for management unless they have significant polytrauma requiring the expertise of the Surgical ICU faculty and nursing staff. In such instances, the Neurovascular team shall follow closely with the Trauma service.

- 3.2 Out-patient – All patients, upon discharge, shall have an appointment made with a member of the neurovascular team (the Neurovascular /Endovascular attending of record) within 2 weeks of discharge unless otherwise indicated. Follow-up imaging shall be discussed and recommended on discharge.

Table 1.1.A

FIGURE 1.0.A EVALUATION AND SCREENING FOR SUSPECTED BCVI

Table 1.1.B

Mechanisms suspicious for BCVI
HIGH ENERGY IMPACT ASSOCIATED WITH:
GCS \leq 8
LeFort II or III fractures
Skull base fracture (e.g. petrous fracture)
Diffuse Axonal Injury
High C-spine fracture (Occipital condyle, C1-C3)
Cervical vertebral body fracture or fracture through foramen transversarium
Subluxation or ligamentous injury at any level
Significant thoracic/cardiac blunt force trauma

Signs/Symptoms suspicious for BCVI
Arterial bleeding form nose, mouth, neck
Audible neck bruit
Skull base fracture (e.g. petrous fracture)
Base of Skull Fractures
High C-spine fracture
Expanding neck hematoma
Focal neurological deficit to include TIAs, Horner's syndrome, <u>Vertebrobasilar syndrome</u>

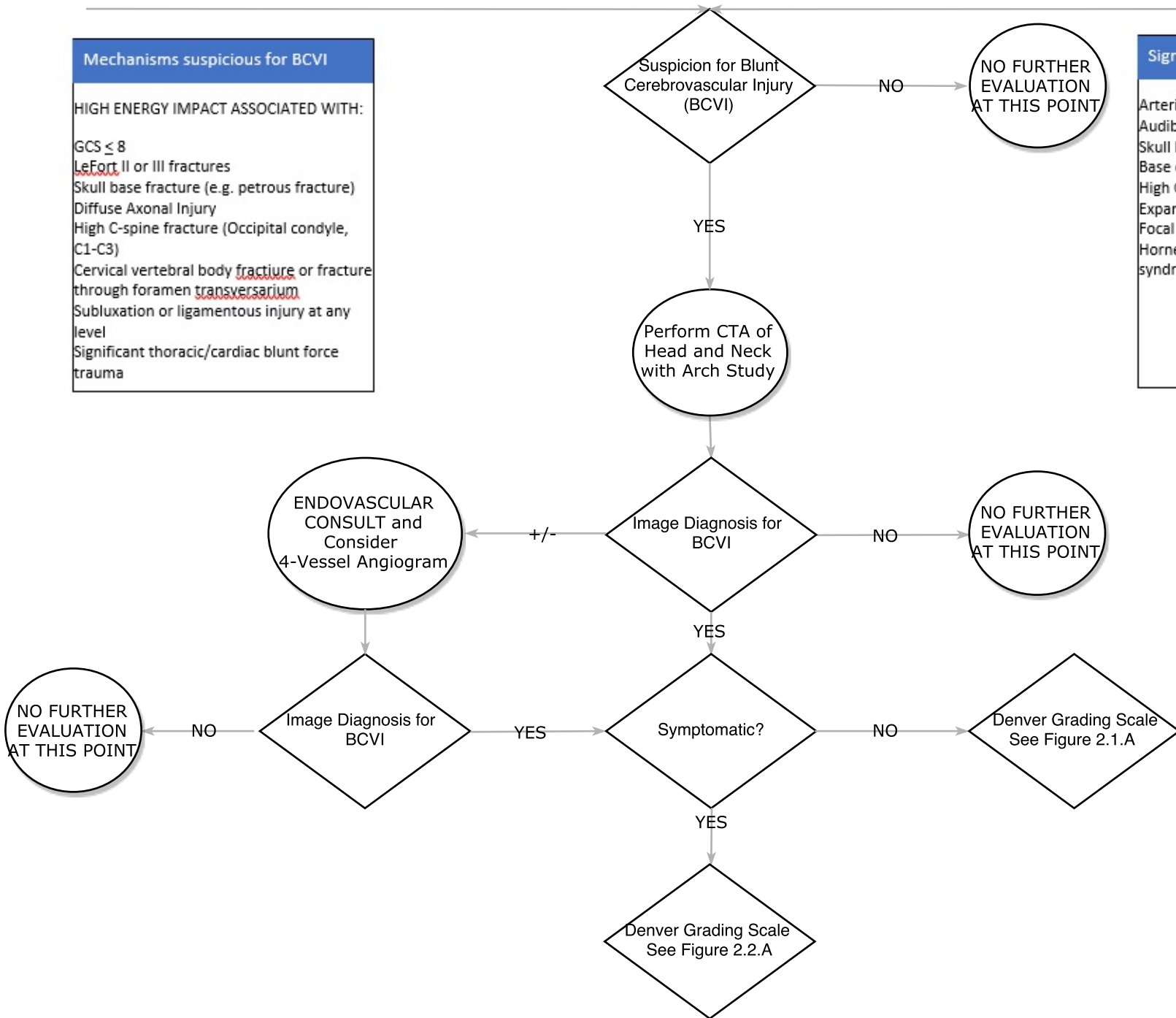


FIGURE 2.1.A MANAGEMENT OF ASYMPTOMATIC BCVI

** Route of administration for ASA can be po or 300mg pr
If ASA is clinically contraindicated, low-dose
no-bolus heparin should be initiated*

*** If Thrombus resolves on 7-day follow-up
imaging, transition to ASA 325mg*

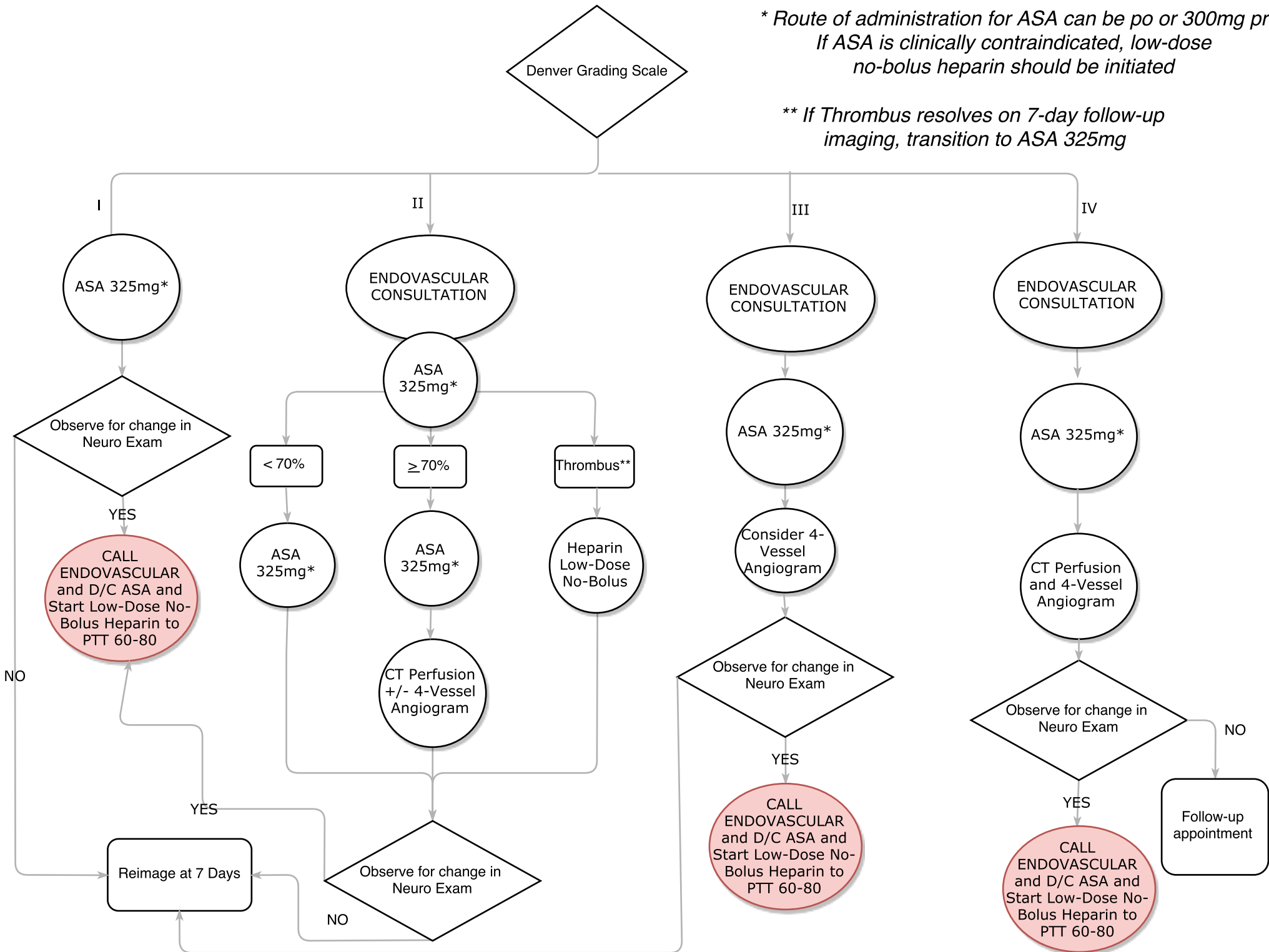


FIGURE 2.2.A MANAGEMENT OF SYMPTOMATIC BCVI

