Trauma Head & Neck

Jan. 13, 2016

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Staff: Dr. K. Vogt

Objectives

- ***Medical Expert:***

1. Mechanism of primary and secondary brain injuries
2. Definition of intracranial pressure and management of intracranial hypertension
3. Clinical assessment and resuscitation of head injured patient
4. Radiographic investigations in head trauma
5. Indications for surgical intervention in head trauma
6. Clinical assessment, classification and investigation of maxillofacial injuries
7. Clinical assessment and resuscitation of penetrating neck injuries
8. Classification of penetrating neck injuries
9. Radiological assessment of penetrating neck injuries
10. Approach to surgical airway (perc trach, tracheostomy, cricothyreotomy)
11. Selective vs. mandatory exploration of penetrating neck injuries
12. Management of vascular injuries in the neck
13. Management of tracheal and laryngeal injuries from trauma
14. Management of pharynx and esophageal injuries from trauma in the neck
15. Clinical assessment, diagnosis and management of blunt traumatic injuries to the neck

Objectives

- ***Communicator:***

1. Management and function of Trauma Team when dealing with head and neck trauma

- ***Collaborator:***

1. Role of other surgical services in neck trauma (ENT, Thoracic, Vascular)
2. Role of diagnostic imaging in neck trauma

- ***Health Advocate:***

1. Strategies to minimize trauma

- ***Manager:***

1. Local and provincial trauma systems
2. Evidence behind use of trauma systems

- ***Scholar:***

1. Review of some of the most recent seminal papers on topic
2. Technical tips or pearls, access and vascular control

Traumatic brain injuries (TBI) outline

- Case
- Mechanism of primary and secondary brain injuries
- Brain trauma guidelines
- Elevated ICP in TBI
  - Definition
  - When to monitor/how to monitor
  - ICP treatment: 1st, 2nd, 3rd line
- Role of induced hypothermia and craniectomy in refractory elevated ICP in TBI – what is the evidence?

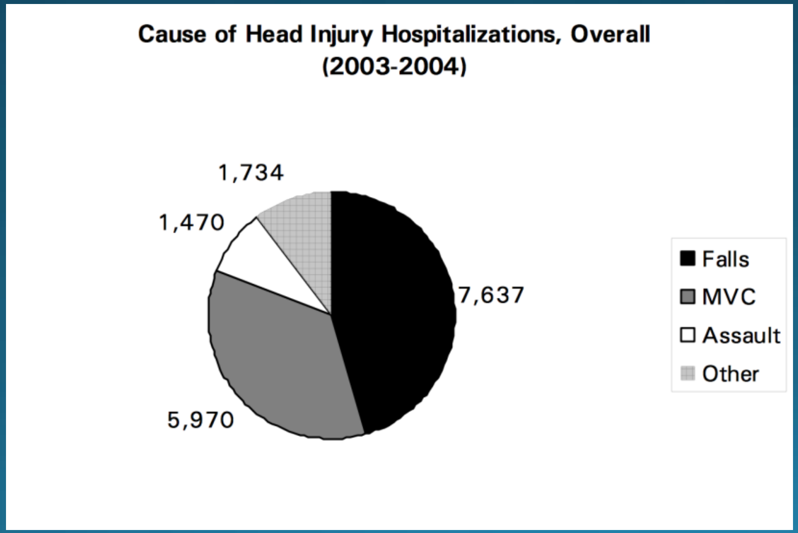
Case 1

- 19y M driving ATV loses control and strikes a telephone pole
  - Transported via land with EMS from peripheral hospital – initially described as hemodynamically stable with GCS 12 (E3V4M5) in periphery -> begins have headache, vomiting and deterioration in LOC

- On arrival @ LHSC,
  - HR 60, BP 160/78, RR 8, SpO_2 90% RA
  - GCS 3
  - Left pupil fixed and dilated 8mm, right pupil 3mm and reactive

Traumatic Brain Injury (TBI)

- Demographics
  - Highest in infants/toddlers(0-4y) and adolescents/youngs adults (15-24)
    - Another peak in elderly (>65y)
  - Higher among men – 2-2.8 : 1 (even higher ratio with severe TBI)
- Other risk factors – lower SES, underlying psych/cognitive disorders
- Mechanism
  1. **Falls**
  2. **MVC**
  3. **Assault**



Pathophysiology of TBI

- Primary brain injuries
  - Intracranial hematoma
    - Subdural hematoma
    - Epidural hematoma
    - Intracerebral hemorrhage (SAH) or contusion
  - Penetrating head injury
    - Tangential
    - Penetrating
    - Perforating
  - Skull fractures
  - Cerebral edema

Pathophysiology of TBI

- Secondary brain injuries -> potentially preventable
  - Hypoxia – $PaCO_2$ < 60mmHg or SpO_2<90%
  - Ischemia
  - Mass effect

Preventing secondary brain injury

- **Avoid hypoxia** (PaO2 <60mm Hg or Spo2<90%)
  - Early intubation if GCS <9 OR lack of appropriate oxygenation or ventilation

- **Avoid hypotension** (SBP <90 mmHg)
  - Hypotension => reduces mean arterial pressure and, as a consequence, cerebral blood flow (CPP = MAP – ICP)
  - Single episode hypotension (SBP<90) shown to be associated with worse outcomes in multiple studies

- **Antipyretics – aggressive treatment of any fever**
  - Fevers increase cerebral metabolic rate -> increases cerebral blood flow, which can raise ICP due to increased blood volume in the cranium

Initial management of TBI patients

- Airway – intubate GCS <=8
  - Needs to be done by skilled practioner

- Breathing – mild hyperventilation ($PaCO_2$ ~35mm) and other measures to reduce ICP before ICP monitoring if one or more clinical signs of herniation:
  - Unilaterally dilated pupil
  - Asymmetric motor examination
  - Declining GCS

- Circulation – keep SBP >90mmHg

Initial management of TBI patients

- Disability (assessment often depends on resuscitated patient who is not hypoxic, hypotensive, hypothermic, hypoglycemic, nor paralyzed)
  - Mild head injury: GCS 14-15
    - 2% chance of elevated ICP and 2% any lesion on CT scan, <0.1% surgical significant lesions
  - Moderate head injuries: GCS 9-13
    - 20% chance elevated ICP, 10% any lesion on CT scan
  - Severe head injury: GCS<9
    - 50% chance of elevated ICP

Epidural hematoma

- Presentation
  - Loss of consciousness – classically brief LOC with recovery of normal consciousness (“lucid interval”), followed by deterioration over period of hours due to continue arterial bleeding/hematoma expansion
  - During deterioration can have:
    - Headache
    - Vomiting
    - Drowsiness
    - Aphasia
    - Seizures
    - Hemiparesis
  - Physical exam: elevated ICP (unilateral dilated pupil) and/or Cushing response

Epidural hematoma

- Dx – CT head – lens-shaped appearance of hematoma

- Management
  - Surgical evacuation of hematoma if:
    - Volume >30cm^3, regardless of GCS
    - Any anisocoria (unequal pupils) or GCS<9, regardless of hematoma size

  - Non-operative an option if:
    - Volume <30cm^2
    - Thickness <15mm
    - Midline shift <5mm, and
    - No coma (GCS>8) and no neuro deficits

Subdural hematoma

- Presentation in acute trauma
  - Coma present at time of injury in 50%
  - Symptoms of elevated ICP (headache, vomiting, anisocoria, dysphagia, cranial nerve palsies, nuchal rigidity, ataxia)
  - Cerebral infarction (ie. stroke), esp. with hematoma in posterior fossa

Subdural hematoma

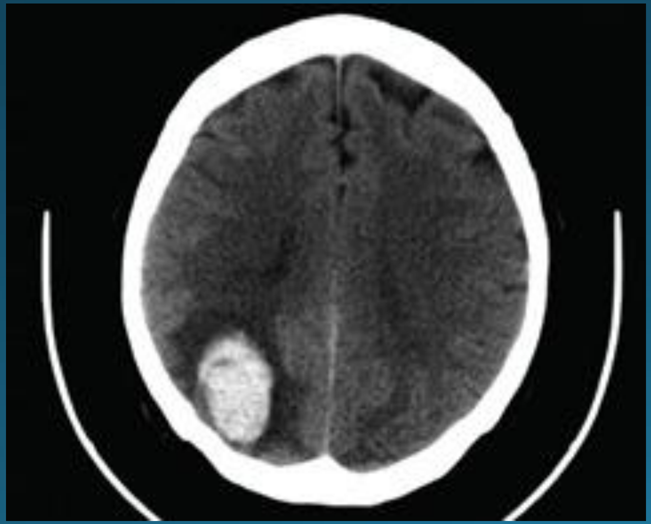
- Dx – CT head – crescent-shaped hematoma

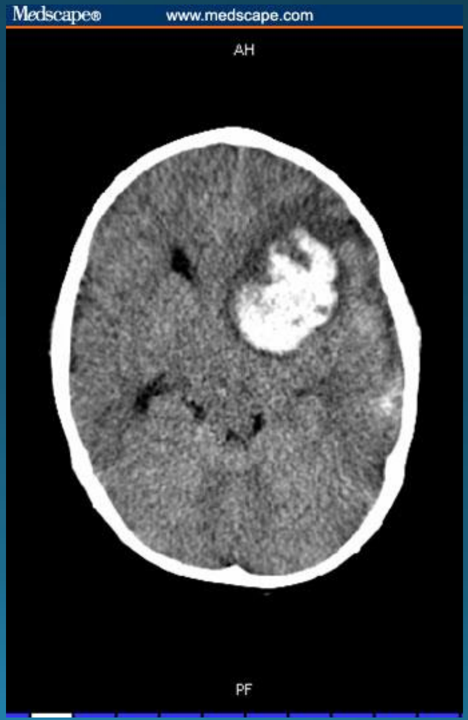
- Management
  - Surgical evacuation if:
    - Thickness >10mm or midline shift >5mm, regardless of GCS
    - If not above, but fixed & dilated pupils OR decline in GCS >=2 points

  - Non-operative – need for serial rpt CT heads, first within 6-8h due to risk of hematoma expansion
    - No RCT available but may be acceptable management if small SDH in stable patients with no deterioration or neuro deficits

Intracerebral hemorrhage

- Presentation
  - Increased ICP – headache, vomiting, seizures, deteriorating LOC, coma
  - Varying neurological deficits depending on location of bleed
- Dx – CT head





Intracerebral hemorrhage

- Craniotomy with surgical evacuation if:
  - Posterior fossa bleed – evidence of mass effect (distortion, dislocation, obliteration of 4th ventricle, compression of basal cisterns, obstructive hydrocephalus)
  - Cerebral hemispheres bleed
    - Hemorrhage >50cm^3
    - GCS 6-8 in frontal or temporal hemorrhage >20cm^3 with midline shift at least 5mm and/or cisternal compression

Penetrating head trauma

- Mortality up to 90%

- types
  - Tangential – glancing injury off of skull

  - Penetrating – projectile enters cranium and remains lodged

  - Perforating – projectile enters and exits cranium, leaving tract

- Predictors of poor outcomes
  - GCS 3-5
  - Hypotension
  - Depressed respiratory rate

Penetrating head trauma

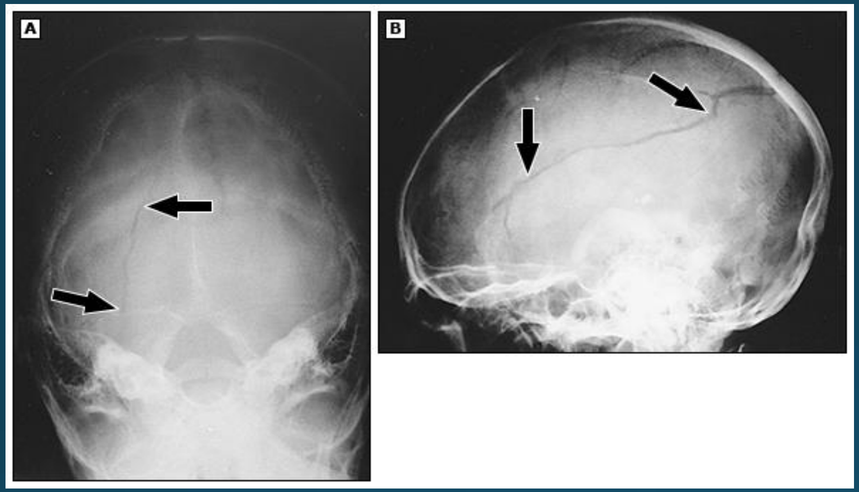
- Management
  - Operative goals are to remove mass effect, control bleeding, control infection, prevent CSF leak, close the scalp
  - Aggressive debridement and removal of deep foreign bodies (bone or bullet fragments) not shown to be effective in preventing delayed infection
  - Prophylactic broad-spectrum abx (cephalosporin) is routine

Skull fractures

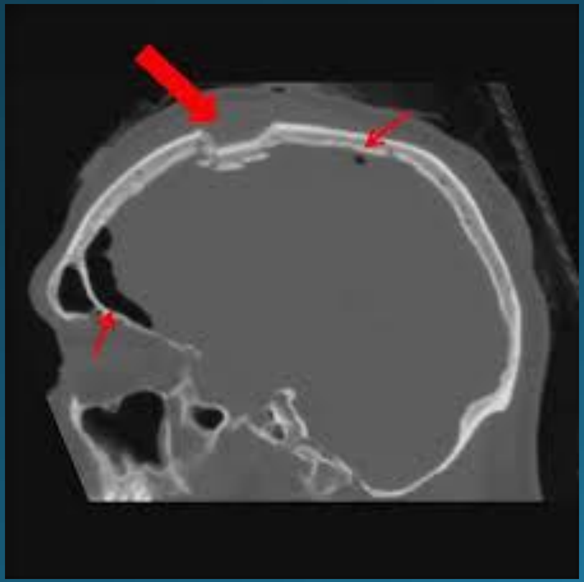
- Presentation/Dx
  - Clinical exam – step deformity, periorbital (racoon eyes) or retroauricular (battle sign) ecchymosis

Skull fractures

- Dx
  - Skull X-rays



  - CT head



Skull fractures

- Open skull fracture
  - Need operative repair if:
    - clinical or radiographic evidence of dural penetration
    - significant intracranial hematoma
    - bone depression >1cm
    - frontal sinus involvement
    - gross cosmetic deformity
    - wound infection
    - pneumocephalus
    - gross wound contamination

Skull fractures

- Closed skull fracture
  - Management:
    - If less than width of skull => non-operative approach

- Principles of surgical repair: elevation of fracture, debridement, closure of dura

Intracranial hypertension

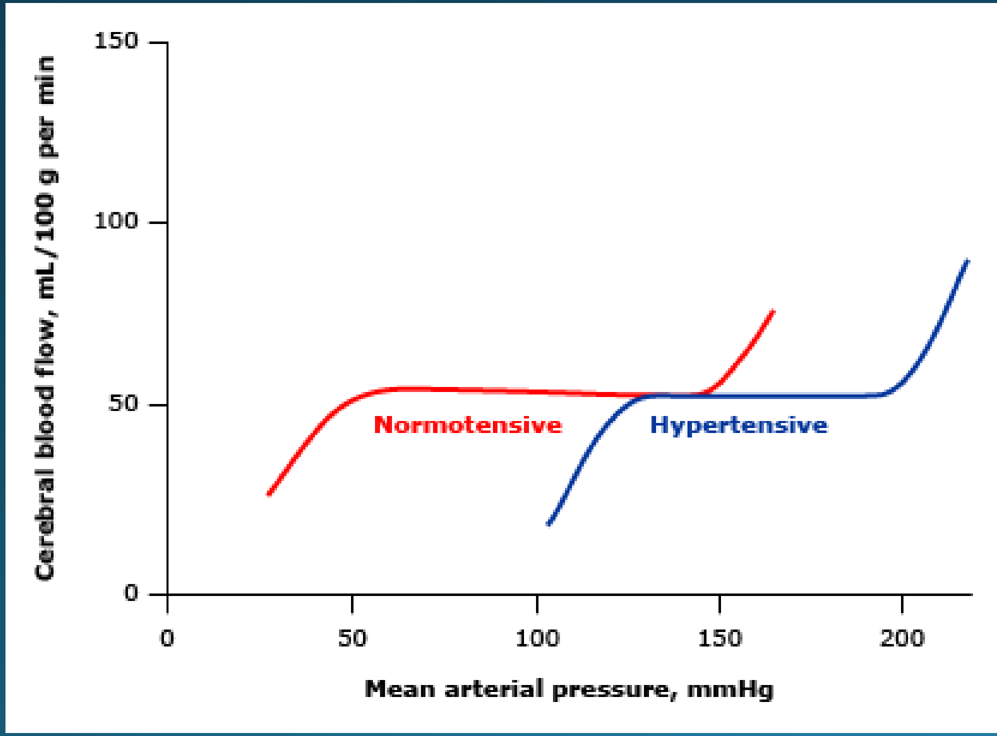
- Intracranial pressure normally <15mmHg
- **Intracranial hypertension >=15mmHg, usually pathologic at >20mmHg**
- Why is ICP important?
  - Cranium has a fixed volume in adult patients (80% parenchyma, 10% CSF, 10% blood)
  - Any increase in the volume one of the above components = increased ICP or mass effect or BOTH -> leads to compression of brain parenchyma and subsequent dysfunction/injury

Intracranial hypertension in TBI

- Most variable intracranial component is the volume of blood
  - Cerebral blood flow (CBF) determines volume of blood flow in the intracranial space
  - CBF = (carotid artery pressure – jugular venous pressure) ÷ cerebrovascular resistance
  - CBF increases with hypercapnia and hypoxia

Intracranial hypertension in TBI

- CBF is normally maintained at a relatively constant level by cerebrovascular autoregulation of CVR over a wide range of CPP (50 to 100 mmHg)
  - process becomes dysfunctional with TBI, small changes in CPP have large effect



Intracranial hypertension in TBI

- CBF difficult to measure -> cerebral perfusion pressure (CPP) is used as a clinical surrogate for the adequacy of cerebral perfusion

- **CPP = MAP – ICP**
  - **Normal CPP usually ~50-60 mmHg**
  - Any increase in ICP results in reduction in CPP (i.e. cerebral perfusion), unless compensated for by increase in MAP

- In TBI, autoregulatory attempt to reduce ICP by decreasing cerebral blood flow eventually leads to ischemia and infarction

ICP Monitoring

- Who needs it in TBI?:

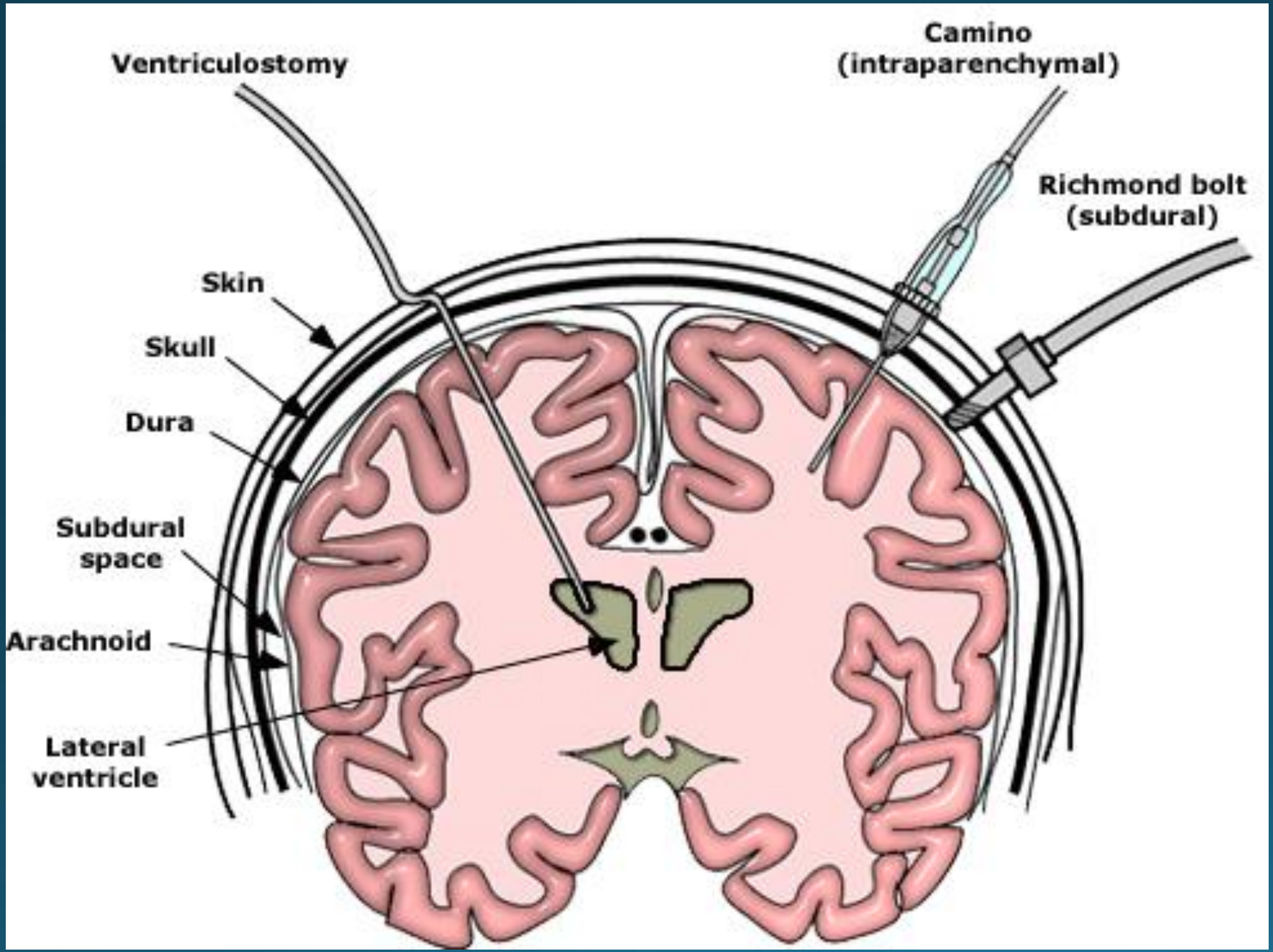
  - GCS<9 with abnormal CT head with evidence of mass effect from hematoma, contusion, swelling

  - Severe TBI (GCS<9) with normal CT head and 2 of:
    - Age >40 years
    - Motor posturing
    - SBP<90mmHg

ICP Monitoring

- Types of monitors – pros/cons of each
  - **Intraventricular – gold standard** – Surgically placed catheter into ventricular system
    - Pro – allow drainage of CSF and can be recalibrated
    - Con – higher infection rate (20%), hemorrhage (2%), technically challenging
  - Intraparenchymal – Fiberoptic transducer into brain parenchyma via small hole in cranium
    - Pro – Lower risk of infection and bleeding (<1%)
    - Con – can't drain CSF and less accurate over longer periods (can't be recalibrated)
  - Subarachnoid – hollow screw through cranium, puncturing through dura
    - Pro – low risk of infection and bleeding
    - Con – often clog and can be unreliable
  - Epidural – rest against dura after placement through cranium
    - Pro – useful in coagulopathic patients due to lower risk of intracerebral hemorrhage (vs. intraventricular or intraparenchymal)

ICP monitors



ICP Monitoring

- Risks of ICP monitoring
  - Infection: intraventricular >>> intraparenchymal > subarachnoid > epidural
    - Highest with intraventricular (20%)
    - Lowest with epidural
  - Intracranial bleed – intraventricular > intraparenchymal > subarachnoid = epidural

Management of increased ICP

- ICP therapy – 1^{st} line therapy
  - **Head elevation – at least 30 degrees**
    - Increases cerebral venous outflow => reducing ICP
    - Avoiding vasovagal maneuvers prevents increases in ICP as well
  - **Cerebral spinal fluid (CSF) drainage via intraventricular drain**
    - CSF removed at 1-2mL/minute at a time at intervals 2-3 min, until ICP goal reached (<20mmHG) OR CSF no longer easily obtained
  - **Sedation – propofol**
    - Propofol easily titrated with short half-life (for neuro reassessment) -> BEWARE hypotension
  - **Analgesia**

Management of increased ICP

- Hyperosmolar therapy
  - Mannitol- can be given in bolus (1g/kg of 20% mannitol over 10-20 min) or as an infusion
    - Side effects – hypotension, excreted renally so risk of ATN (esp. if underlying renal disease)
  - Hypertonic saline – also can be given in bolus or infusion
    - Concentrations vary – 3% to up to 23%
    - Side effects – hypernatremia (worsening coma, sz, SAH)
  - **Mannitol vs. hypertonic saline – hypertonic saline better at controlling ICP?**
    - Meta-analysis of 5 RCTs comparing both in reducing ICP found hypertonic saline had greater efficacy in managing ICP BUT clinical outcomes not examined

Management of increased ICP

- Paralytics
  - Usually run as infusion
  - Agitation common in TBI -> leads to increased ICP
  - Downside – can't perform neuro assessment

- Albumin – associated with worse outcomes in TBI patients

Management of increased ICP

- Hyperventilation
  - Mechanism -> causes reduced CO_2 levels that result in cerebral vasoconstriction that reduces cerebral blood flow and lowers ICP -> potentially can induce cerebral vasoconstriction to levels that cause ischemia/infarction

  - Single RCT – no difference in outcomes at 12 months but worse outcomes at 3 and 6 months
    - Definition: in RCT treatment arm hyperventilation was defined as $PaCO_2$ ~25mmHg (<30mmHg)

  - **Recommendation: use hyperventilation for SHORT periods of time in the face of impending cerebral herniation OR in the presence of elevated ICP refractory to sedation, paralysis, CSF drainage, or osmotic diuresis**
    - May be a role for mild hyperventilation ($PaCO_2$ ~35mmHg) for short periods of time (~30min), especially in situations above

Management of increased ICP

- Refractory elevated ICP
  - Barbiturate coma (3rd line treatment)
    - Mechanism – reduce brain metabolism and cerebral blood flow -> thus reducing ICP
    - Agents – phenobarbital (loading bolus 5-20m/kg, then 1-4mg/kg/h)
    - Monitored with continuous EEG -> aim for burst suppression
    - Side effects
      - Hypotension
      - Inability to perform neuro assessment
      - Intensive monitoring – ICP, EEG, etc.
    - Evidence – controls ICP but no difference in clinical outcomes

Management of increased ICP

- Refractory elevated ICP
  - **Glucocorticoids – found to be HARMFUL in moderate to severe TBI**
    - 10,000 patients randomized within 8h presentation to placebo vs. methylprednisone
    - Methylprednisone treated patients had increased mortality at 2 weeks (21% vs 18%, RR 1.18) and at 6 months (26% vs. 22%, RR 1.15)

Hypothermia in elevated ICP

- Induced hypothermia
  - Multicenter RCT enrolling 387 patients with ICP>20mmHg from 2009-2014
    - Standard care group (mechanical ventilation + sedation)
    - Hypothermia group (standard + induced 32-35C)
    - Primary outcome (extended Glasgow Outcome scale at 6 months)
  - Worse outcomes for hypothermia group
  - "Good outcomes" (GOS-E 5-8) occurred in 26% hypothermia patients and 37% standard care patients (p=0.03) -> trial terminated early due to safety concerns

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 DECEMBER 17, 2015 VOL. 373 NO. 25

Hypothermia for Intracranial Hypertension after Traumatic Brain Injury

Peter J.D. Andrews, M.D., M.B., Ch.B., H. Louise Sinclair, R.G.N., M.Sc., Aryelly Rodriguez, M.Sc., Bridget A. Harris, R.G.N., Ph.D., Claire G. Battison, R.G.N., B.A., Jonathan K.J. Rhodes, Ph.D., M.B., Ch.B., and Gordon D. Murray, Ph.D., for the Eurotherm3235 Trial Collaborators*

1	Death	D
2	Vegetative state	VS
3	Lower severe disability	SD -
4	Upper severe disability	SD +
5	Lower moderate disability	MD -
6	Upper moderate disability	MD +
7	Lower good recovery	GR -
8	Upper good recovery	GR +

Craniectomy in elevated ICP

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 APRIL 21, 2011 VOL. 364 NO. 16

Decompressive Craniectomy in Diffuse Traumatic Brain Injury

D. James Cooper, M.D., Jeffrey V. Rosenfeld, M.D., Lynnette Murray, B.App.Sci., Yaseen M. Arabi, M.D., Andrew R. Davies, M.B., B.S., Paul D'Urso, Ph.D., Thomas Kossmann, M.D., Jennie Ponsford, Ph.D., Ian Seppelt, M.B., B.S., Peter Reilly, M.D., and Rory Wolfe, Ph.D., for the DECRA Trial Investigators and the Australian and New Zealand Intensive Care Society Clinical Trials Group*

Craniectomy in elevated ICP

- Role of decompressive craniectomy on elevated ICP in TBI
  - RCT of 155 patients with severe, diffuse TBI with elevated ICP refractory to other therapies randomized to bifrontal decompressive craniectomy or standard of care within first 72h after injury
  - **Surgery associated with decreased ICP and shorter ICU stay (not overall hospital stay), but worse outcomes on extended Glascow outcome scale at 6 months**
  - Issues:
    - Imbalance in TBI between groups
    - Low threshold for elevated ICP
    - Short window for defining refractory ICP
    - Surgery not representative

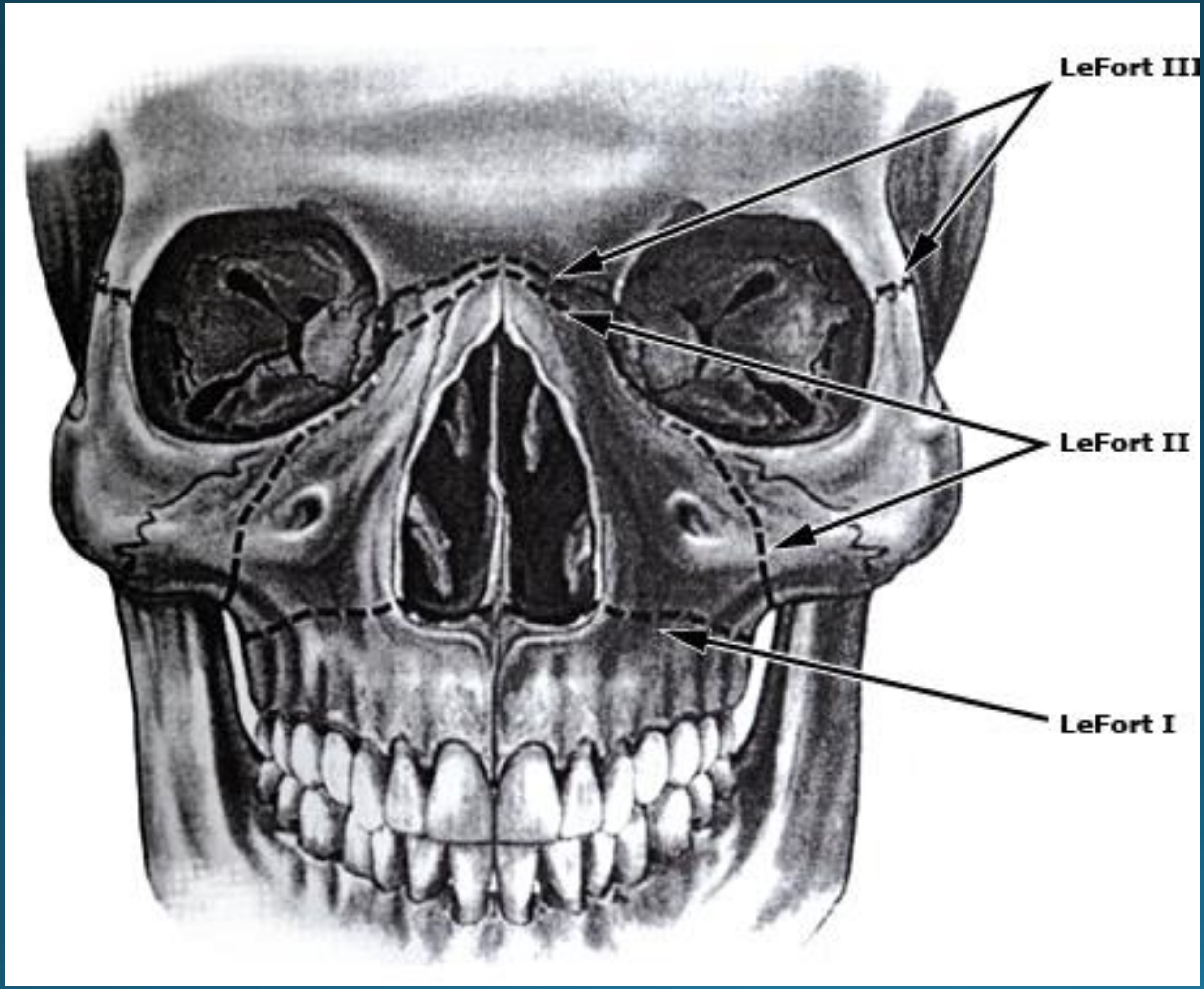
Management of severe TBI

- Role for CPP-based therapy??
  - **Brain Trauma guidelines: CPP ~ 60mmHg is ideal in severe TBI**
    - **Target with fluids and/or vasopressors**
  - Avoid CPP <50mmHg due to associated ischemia and poor outcomes
  - **Avoid aggressive attempts to target CPP>70mmHg with fluids or vasopressors => associated with increase in ARDS**
    - RCT from Roberston *et al*. (1999) comparing effect of CPP-targeted vs. ICP-targeted therapy in severe TBI in 189 patients at 6 months
    - Increase in ARDS (5-fold) compared to ICP-targeted therapy

Maxillofacial trauma

- Lefort classification
  - Lefort I - involve a transverse fracture through the maxilla above the roots of the teeth; injury may be unilateral or bilateral.
  - Lefort II injuries are typically bilateral and involve fractures that extend superiorly in the midface to include the nasal bridge, maxilla, lacrimal bones, orbital floor, and rim. The fracture lines are shaped like a pyramid.
  - Lefort III – injuries (ie, craniofacial dissociation) involve fractures that result in discontinuity between the skull and the face. The fractures begin at the bridge of the nose and extend posteriorly along the medial wall of the orbit and the floor of the orbit, and then through the lateral orbital wall and the zygomatic arch.

Maxillofacial trauma



Maxillofacial trauma

- Presentation
  - Bruising over midface
  - Enophthalmos (posterior displacement of eyeball)
  - Malocclusion of upper teeth (often Lefort I)
  - Diplopia
  - Nasal congestion
  - Epistaxis
  - Anosmia

- On exam
  - Maxillary (I), nasal complex (II), or entire facial (III) mobility during facial stability test
  - increased intercanthal distance
  - Enophthalmos

Maxillofacial trauma

- Lefort fractures – what's important to recognize?
  - **Lefort II & III associated with increased risk of cerebrovascular injury -> screen appropriately with CTA neck**

- Facial fractures that warrant urgent evaluation and admission include:
  - Nasoethmoid fractures, to monitor for cerebrospinal fluid (CSF) leaks and possible complications (eg, meningitis)
  - Zygomatic arch fractures associated with trismus, to monitor for airway complications
  - **LeFort-type fractures of the midface, for surgical repair**
  - Facial fractures in patients with multiple significant injuries

Penetrating neck injury (PNI) outline

- PNI Case 1
- PNI Case 2
- Clinical assessment and resuscitation of penetrating neck injuries
- Classification of penetrating neck injuries – zones vs. compartments
- Management
  - Hard signs to operate
  - Soft signs to investigate
    - Radiological assessment of penetrating neck injuries
    - Selective vs. mandatory exploration of penetrating neck injuries
- Approach to surgical airway (perc trach, tracheostomy, cricothyrotomy)

PNI case 1

- 47 y male migrant worker admitted for suicidal ideation to peripheral hospital, locks himself in washroom and stabs himself in midline neck
  - transferred to LHSC trauma bay

- On arrival
  - HR 90, BP 115/67, RR 22, SpO_2 88%
  - GCS 15

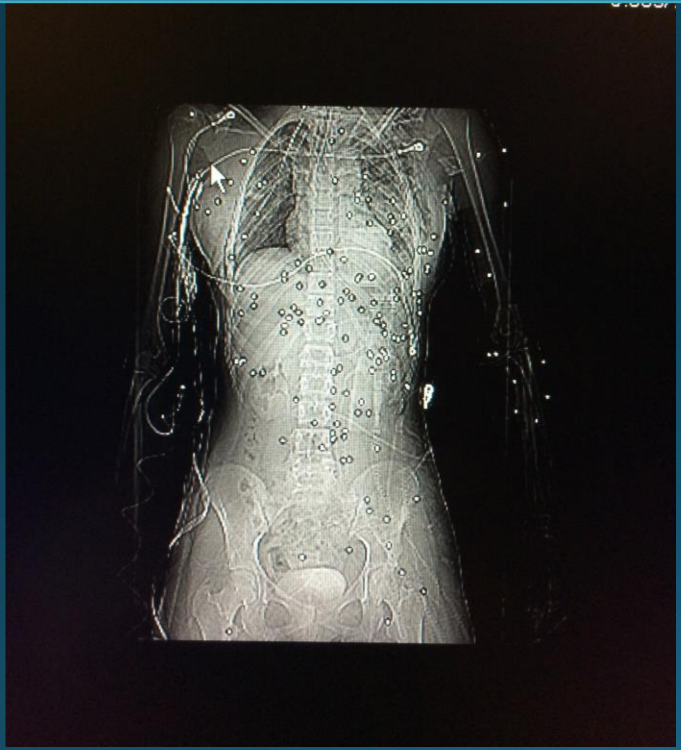
  - Midline zone 2 penetrating neck injury with the following appearance ....

PNI case 1

PNI case 2

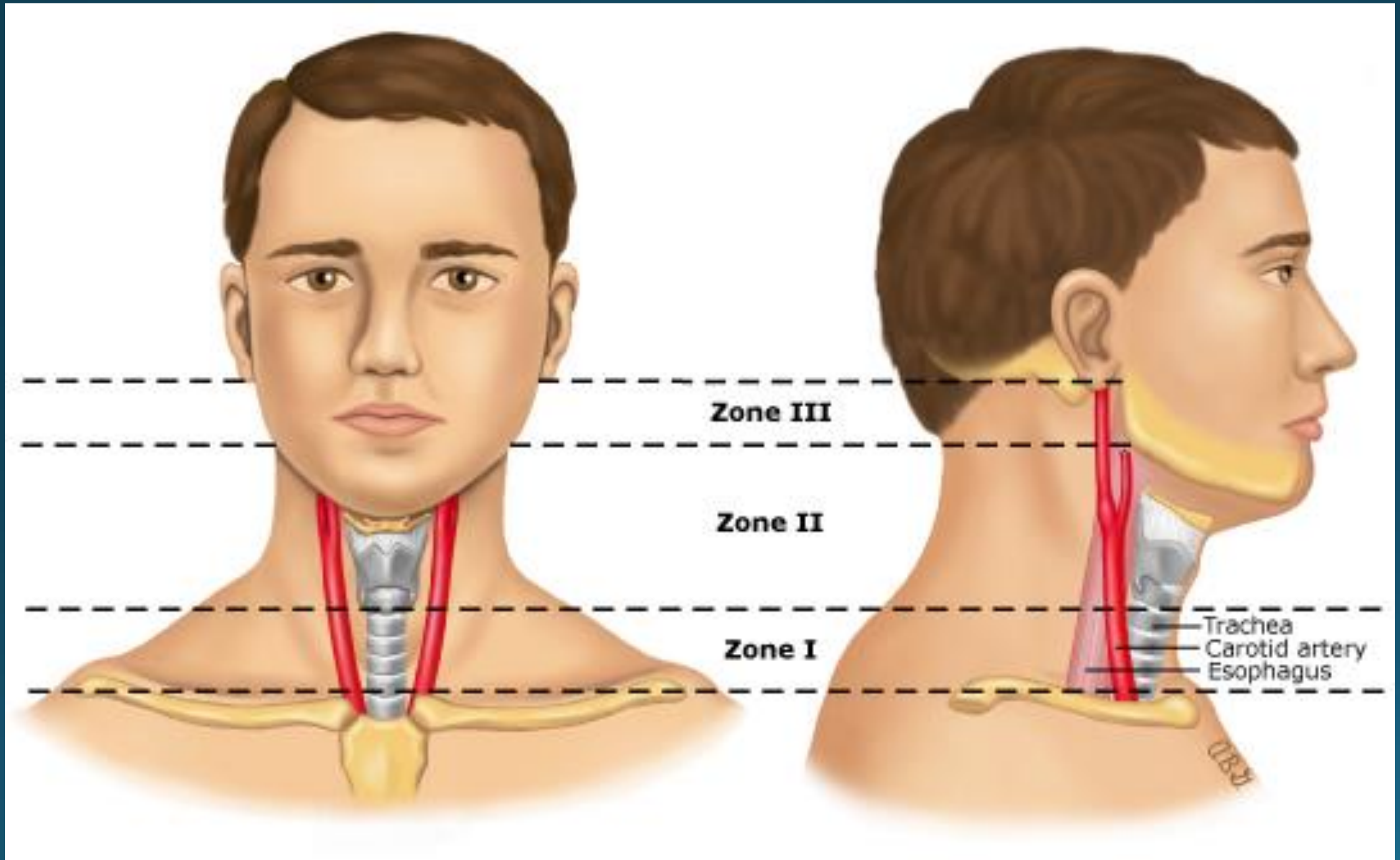
- 19F shot with sawed-off shotgun at distance of 3 ft
- On arrival,
  - HR 95, BP 115/60, SPO_2 96%
  - GCS 9, complaining pain all over
  - No hard signs
  - SC emphysema, crepitus, hemoptysis

PNI case 2



Penetrating neck injuries (PNI)

- Classification of penetrating neck injuries – 3 zones:



PNI Neck Zones

- Zone 1:
  - thoracic outlet vasculature (great vessels), vertebral and proximal carotid arteries
  - lung, trachea
  - thoracic esophagus
  - spinal cord, thoracic duct, major cervical trunks

- Zone 2:
  - jugular veins, vertebral and common carotid arteries, external/internal branches of carotid artery
  - larynx, cervical trachea,
  - cervical esophagus
  - spinal cord, vagus/spinal accessory/hypoglossal nerves

- Zone 3:
  - jugular veins, vertebral arteries, distal internal carotid arteries
  - Pharynx
  - spinal cord, and the facial, glossopharyngeal, vagus, spinal accessory, and hypoglossal nerves.

PNI presentation

- Overt signs ("hard signs)
  - Vascular injury
    - Severe or pulsatile bleeding
    - Expanding hematoma
    - Absent or diminished peripheral pulse
    - Audible bruit or palpable thrill
    - Stroke

  - Airway injury
    - Stridor/severe dyspnea
    - Bubbling from wound track
    - Direct wound to trachea
    - Compression by vascular hematoma

  - Esophagus – none

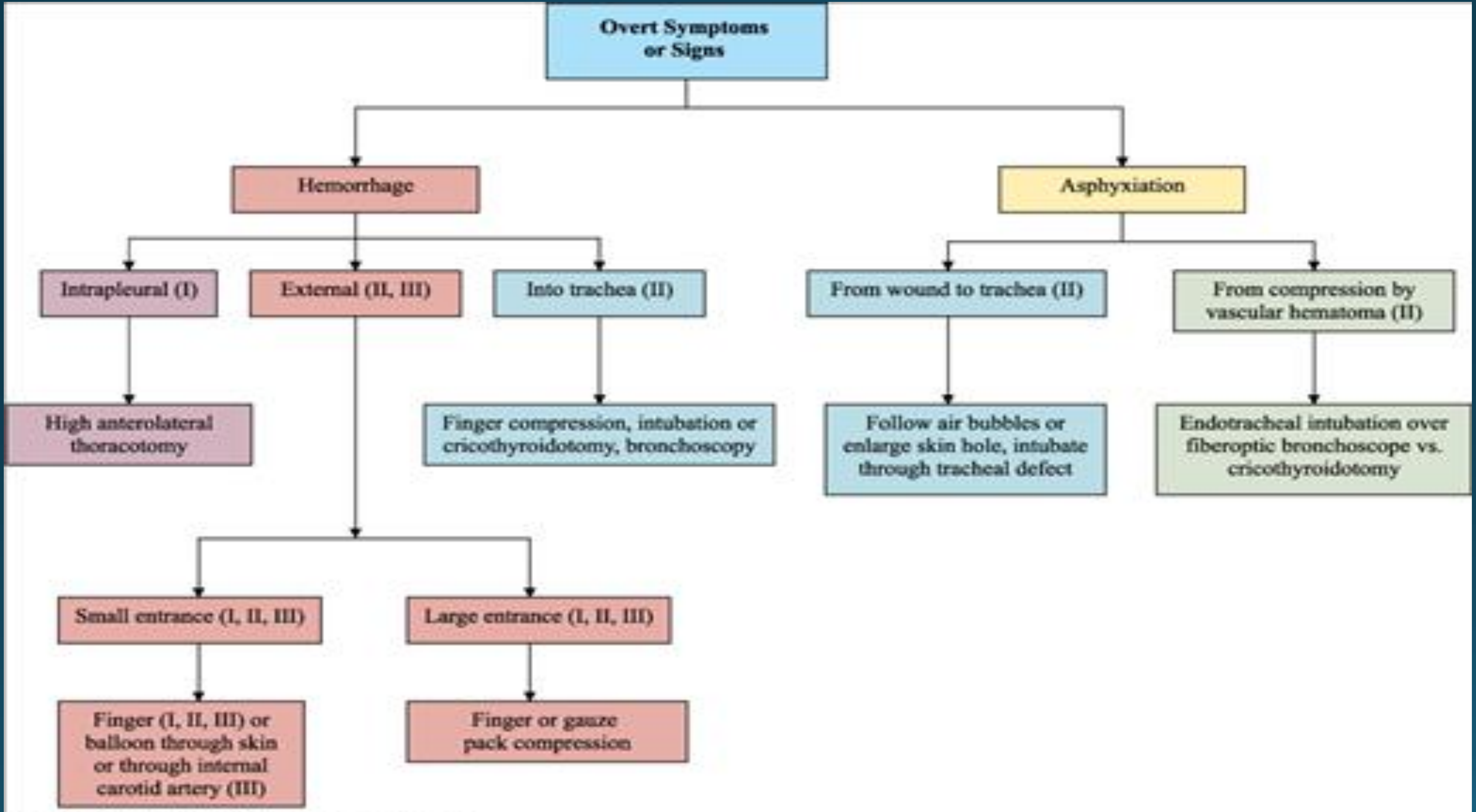
PNI presentation

- Moderate signs ("soft signs")
  - Potential vascular injury
    - Stable hematoma
    - Minimal, non-pulsatile bleeding
    - Proximity
  - Potential aerodigestive injury
    - Hoarseness
    - Dysphonia
    - Hemoptysis
    - Dysphagia/odynophagia
    - Subcutaneous emphysema/crepitus
    - Hematemesis
  - Neurologic – clinical nerve injury

PNI presentation

- Asymptomatic patient – wound with penetration of the platysma muscle

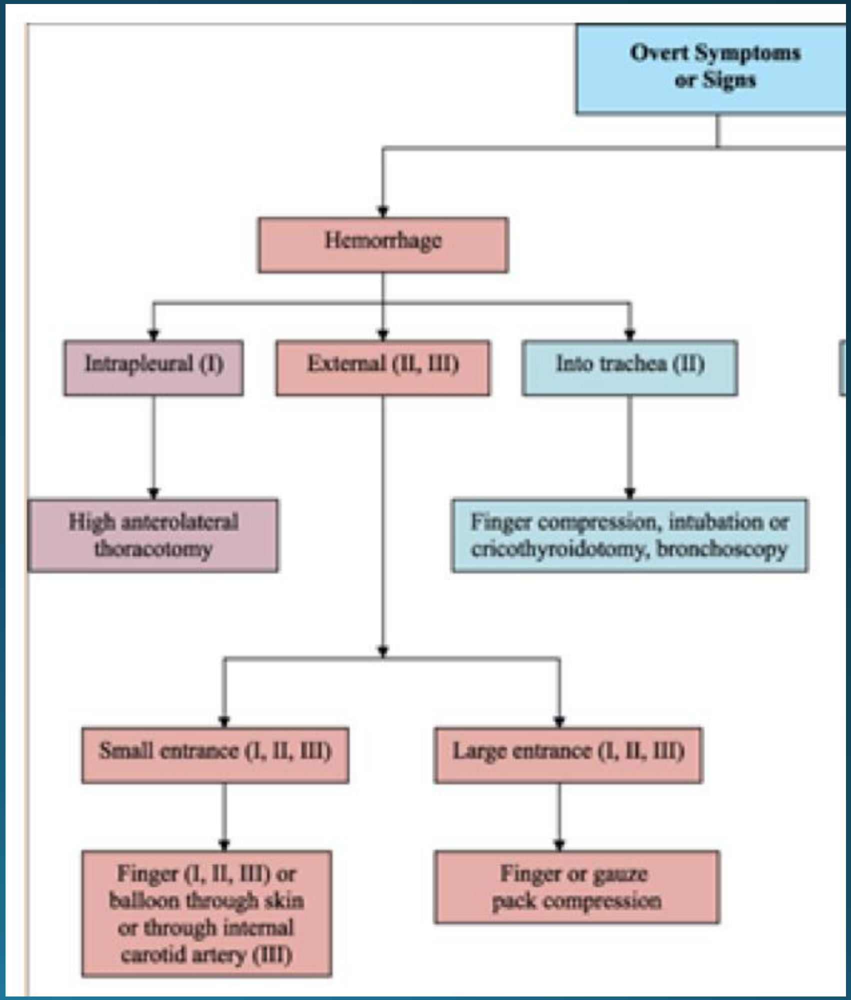
PNI Management – Hard Signs



Source: Mattox KL, Moore EE, Feliciano DV: *Trauma, 7th Edition*: www.accesspharmacy.com

PNI management - hemorrhage

- Hemorrhage
  - Control strategies on way to OR
    - Finger compression/gauze compression
    - Foley balloon
  - Access incisions
    - Zone I – MEDIAN STERNOTOMY or high anterolateral thoracotomy or clavicular incision
    - Zone II – anterior SCM incision, collar incision (if bilateral)
    - Zone III – oblique incision on SCM



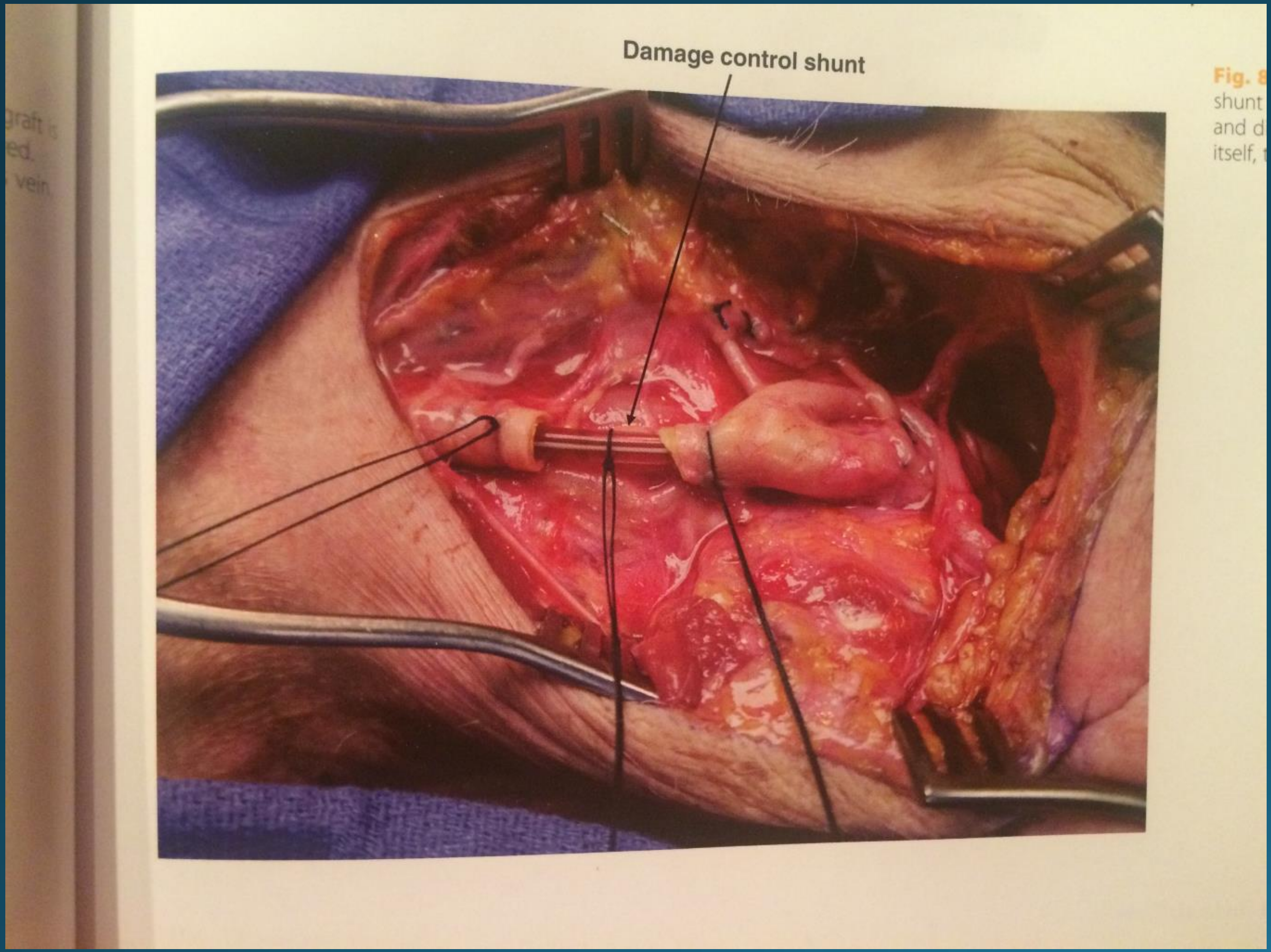
PNI management - hemorrhage

- Foley balloon

PNI management - hemorrhage

- Repair vs shunt
  - Small carotid artery injuries -> primary repair with 5-0 Prolene
  - Larger and/or tissue loss
    - Primary repair not possible without stenosis: patch angioplasty with vein patch or prosthetic graft (Dacron or PTFE) -> running, continuous fashion circumferentially with 5-0 Prolene
    - Destructive injuries: interposition graft
  - Patient unstable: carotid shunt

Carotid shunt



Shunt is secured with silk ties around proximal and distal arterial segments AND shunt itself

Operative incisions

- Anterior sternocleidomastoid (SCM) incision (zone 1 & lateral zone 2 exposure)
  - Incision – anterior border of SCM

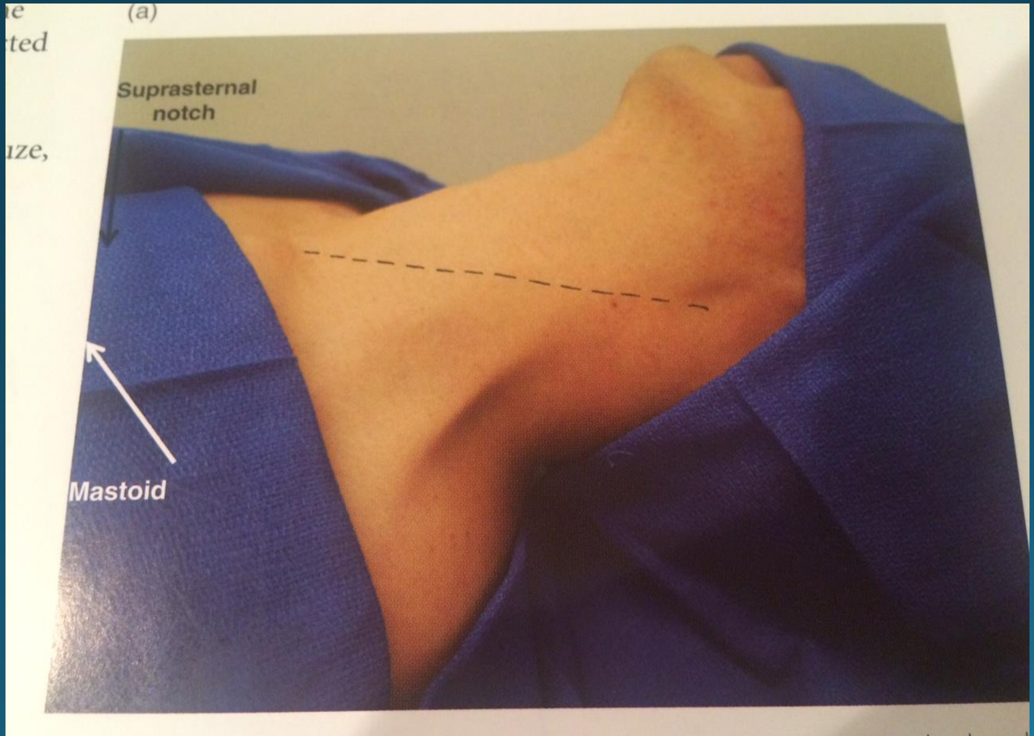
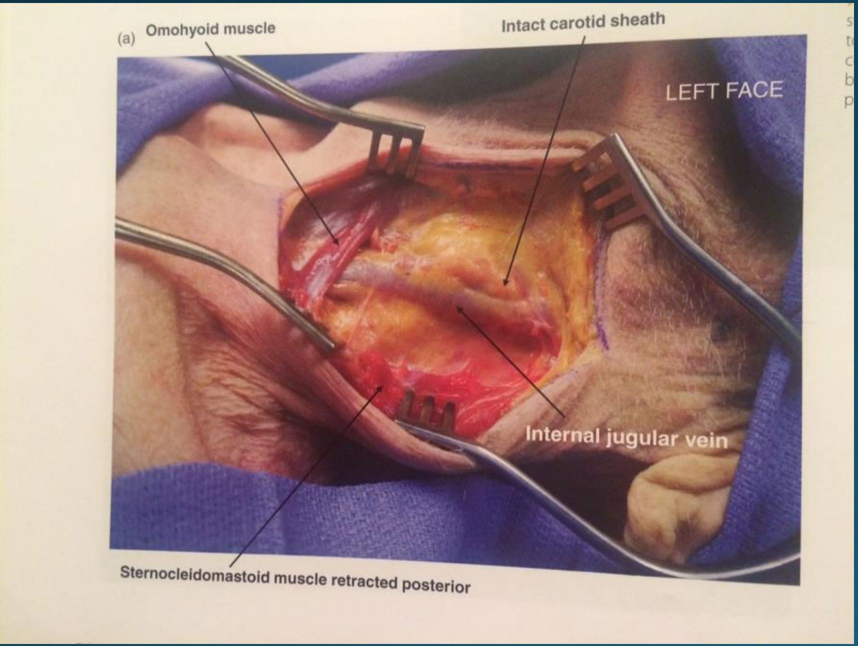
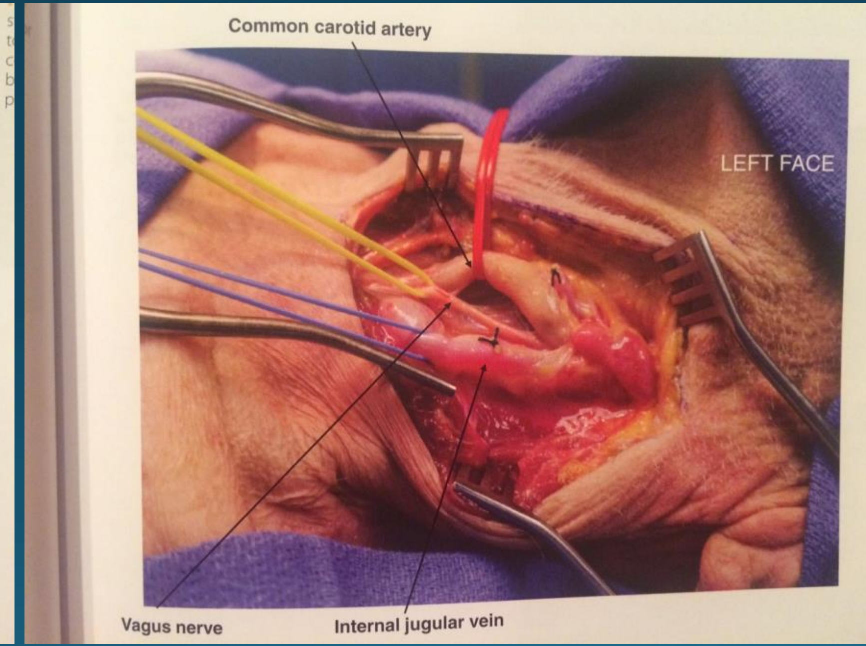


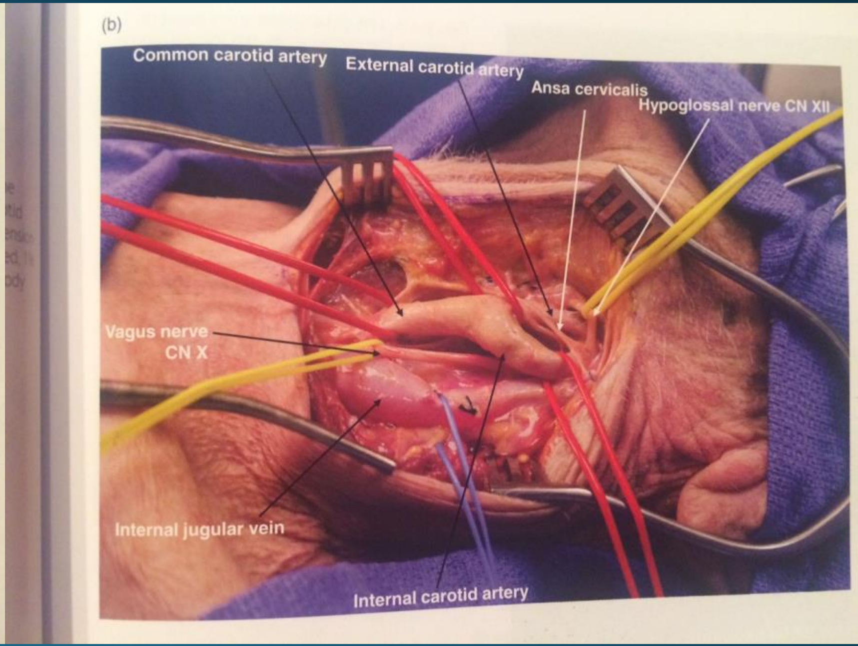
Fig. 7.3(a). Position of patient for a sternocleidomastoid incision: the head

Operative incisions

- Approach to carotid sheath

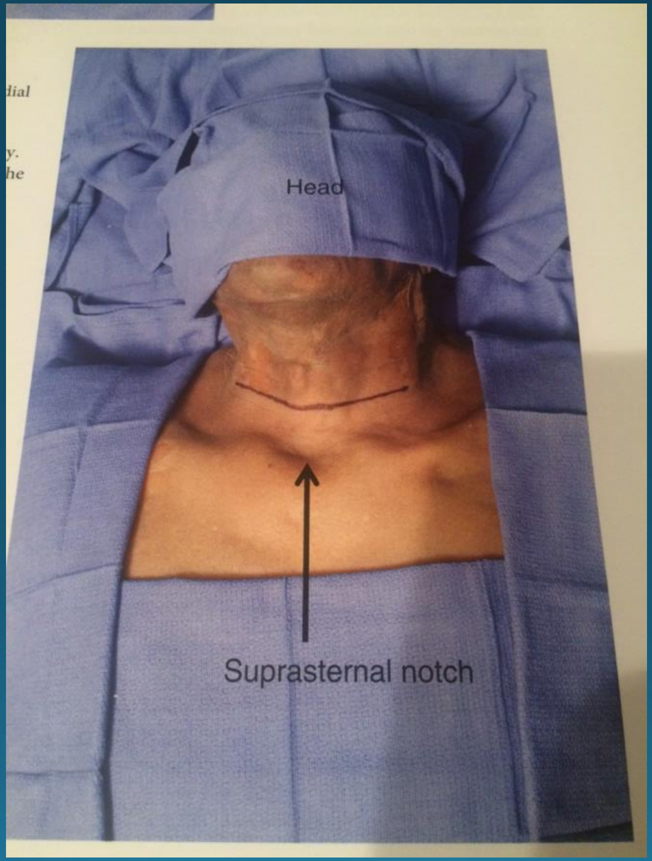






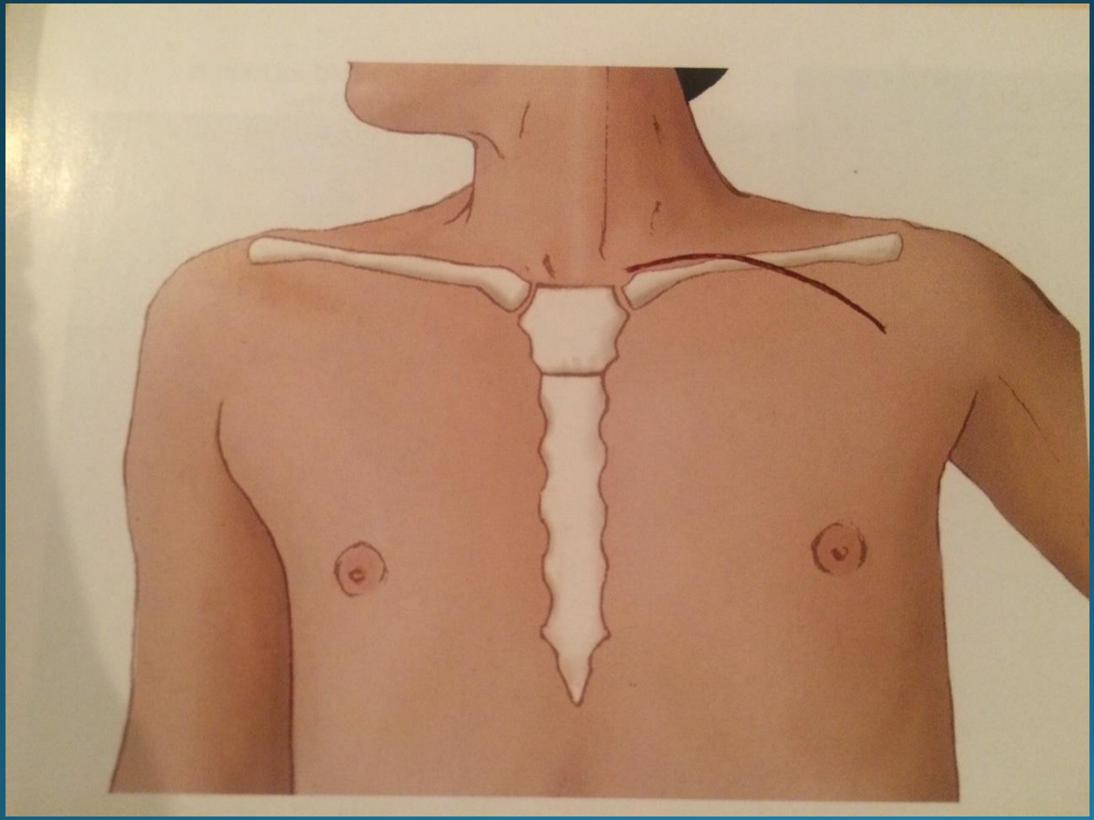
Operative incisions

- Collar incision (midline zone II exposure)
  - Incision made two fingerbreadths above sternal notch with extension to medial aspect of SCMs bilaterally



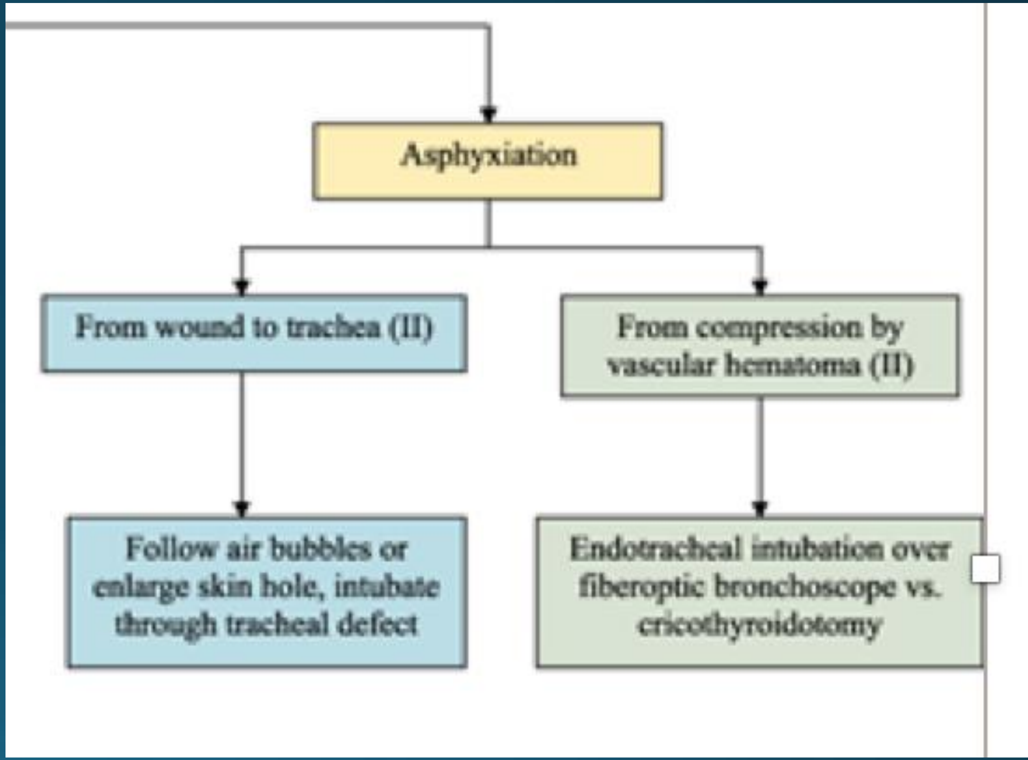
Operative incisions

- Clavicular incision (left and right subclavian exposure)
  - Begins at sternoclavicular junction, extends over medial clavicle, curves downward at middle portion of clavicle into deltopectoral groove

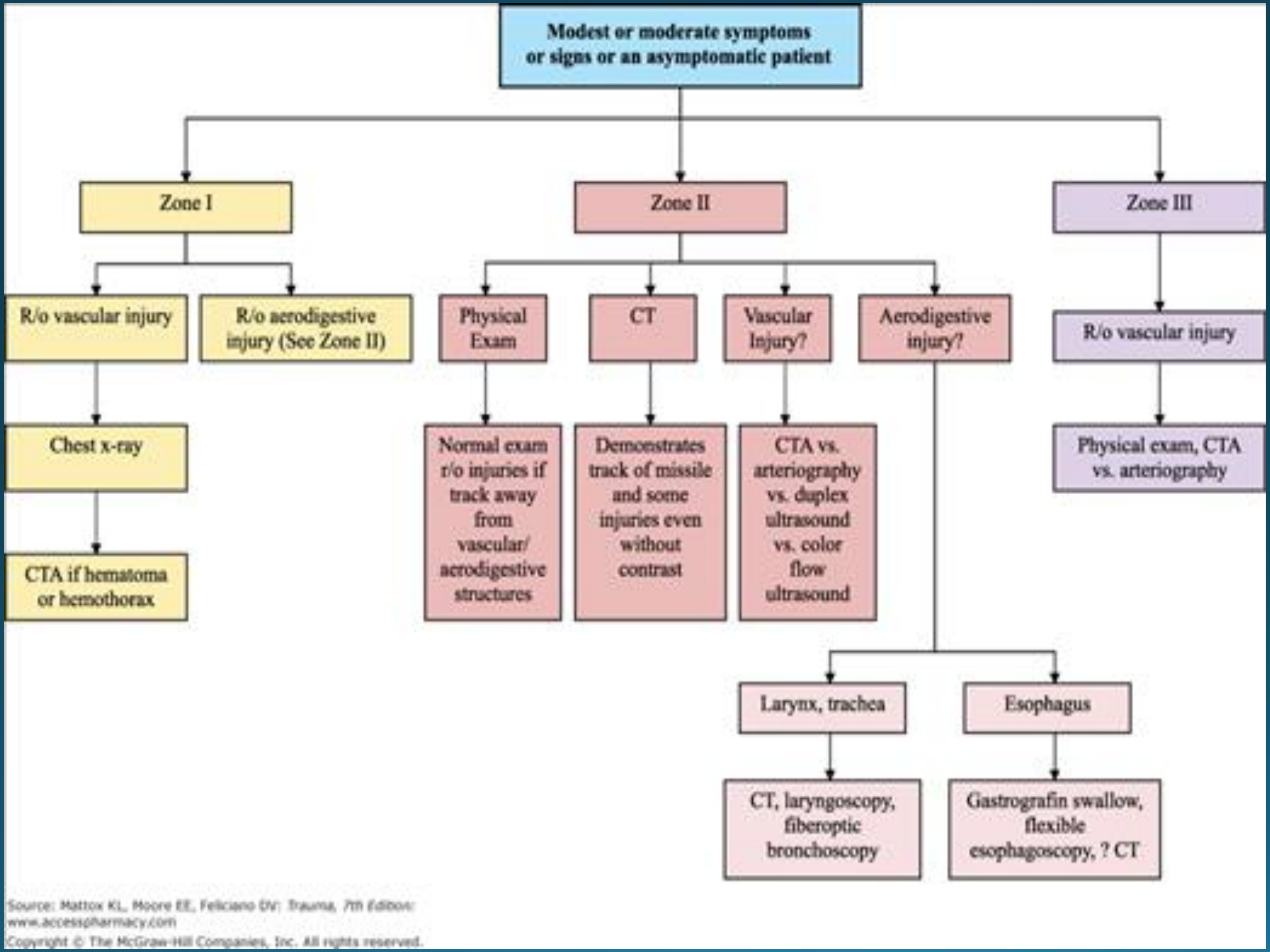


PNI management - asphyxiation

- Asphyxiation – get an airway STAT, low threshold for surgical airway
  - Suspected cricotracheal separation or laryngeal injury
    - ONE attempt at standard endotracheal RSI -> if fails then trach between 2nd-3rd tracheal rings
  - Vascular hematoma causing airway compression
    - ET intubation over bronchoscope vs. cricothryotomy

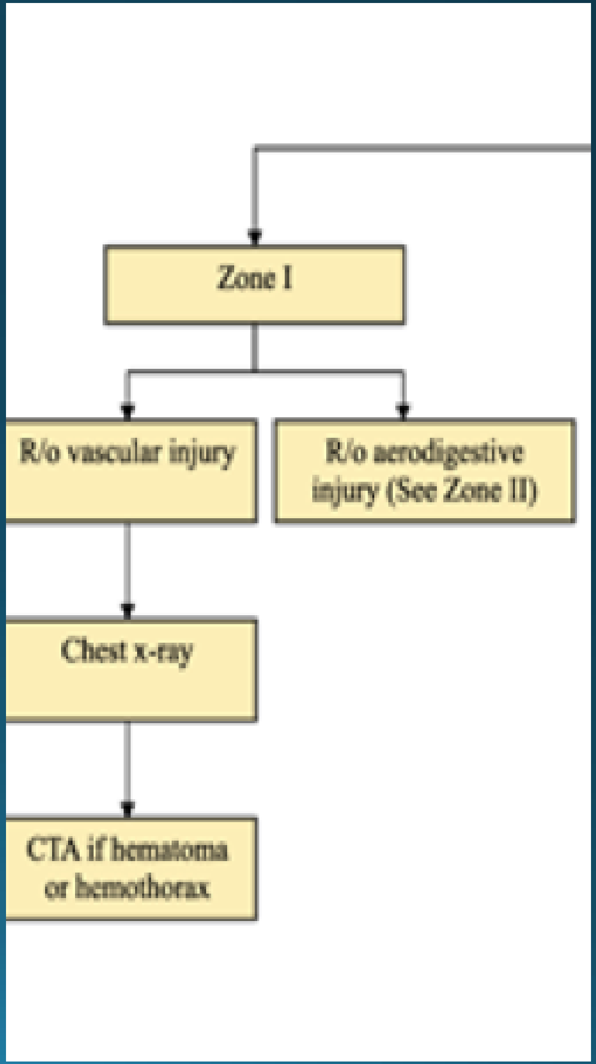


PNI Management – Moderate Signs



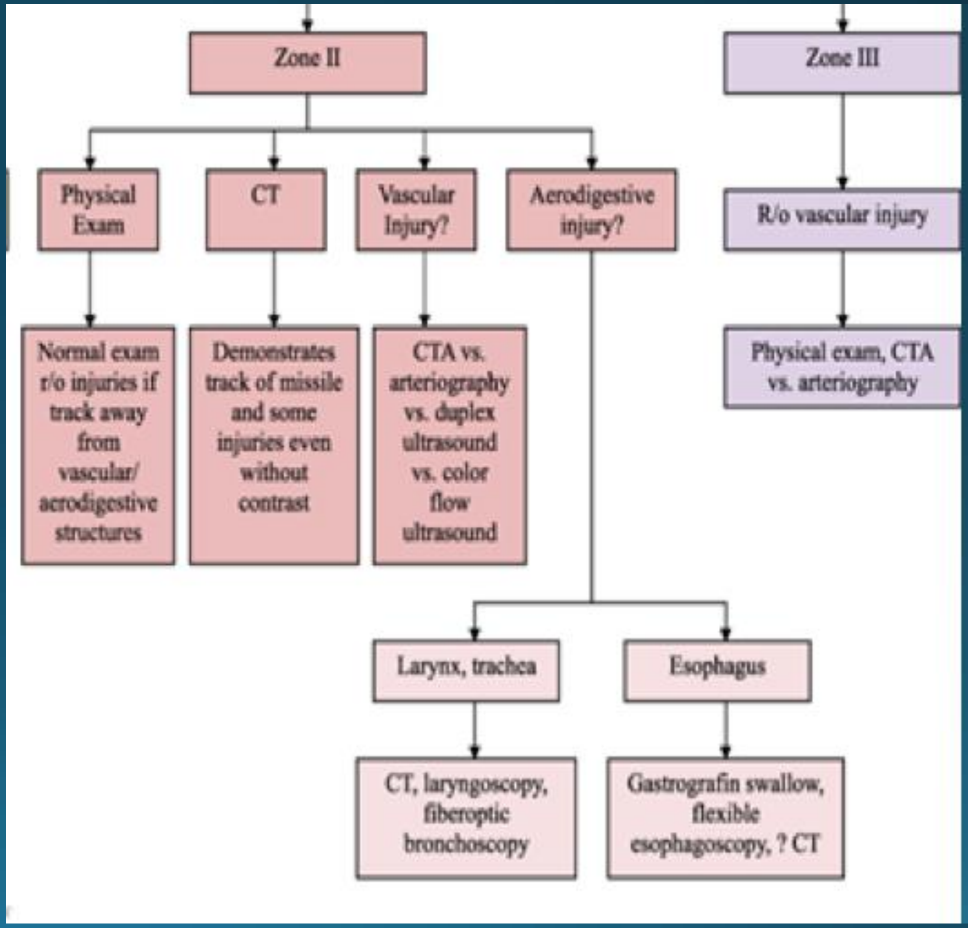
PNI management – Moderate signs

- Zone 1
  - Good retrospective data that normal physical exam and normal CXR almost exclude vascular injury at thoracic outlet
  - Track of wound OR hematoma in proximity to vessels OR hemothorax on CXR => **CTA neck & CT thorax**



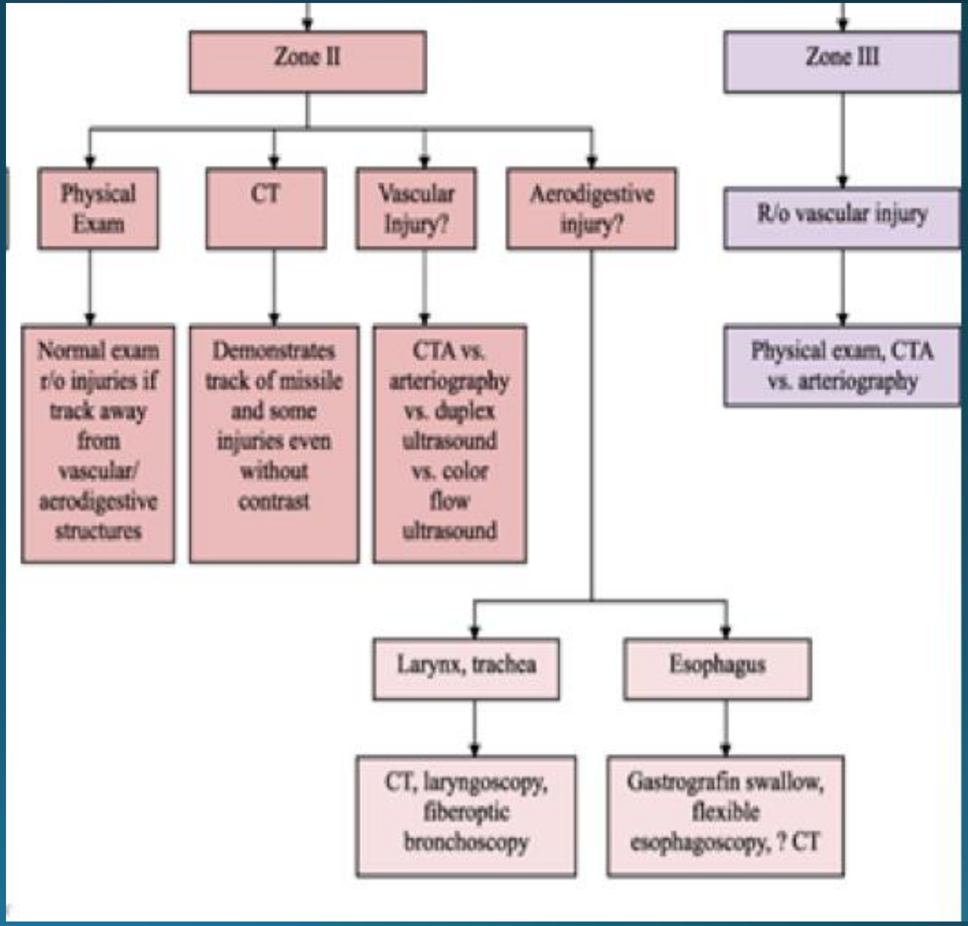
PNI management – Moderate signs

- Zone II
  - Former standard of mandatory exploration of penetrating zone II neck injuries led to 50% negative exploration rate
  - Physical exam alone sufficient in asymptomatic patient for penetrating injuries (deep to platysma)
  - CTA neck and upper thorax – demonstrates trajectory of wound and avoid further tests
    - 100 % sensitive and 95% specific in detecting vascular injuries
  - **Suspected airway injury - Laryngoscopy, bronchoscopy**
  - **Suspected esophageal injury – contrast esophagram + rigid esophagoscopy**



PNI management – Moderate signs

- Zone III
  - **CTA head & neck r/o vascular injury**



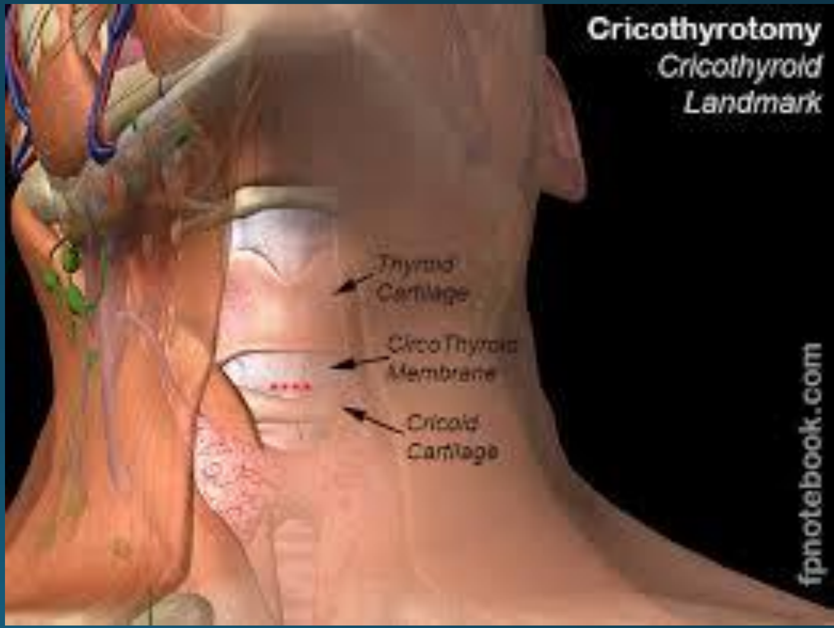
Cricothyrotomy

- Equipment:
  - **ETT and trach tubes (size 6)**
  - **Scalpel**
  - **Tracheal hook**
  - **Retractors**
  - **SUCTION setup**
  - Adequate lighting

- Patient positioning – supine with neck extended (neutral position if c-spine not cleared)

Cricothyrotomy

- Technique
  - Landmark thryoid and cricoid cartilage
  - Stabilize thyroid cartilage with non-dominant hand
  - Make vertical incision over cricothyroid membrane (CTM)
  - Make horizontal stab incision through CTM (lower half to avoid cricothyroid artery)
  - Insert tracheal hook into superior end of incision and retract toward head
  - Insert tube into trachea, inflate balloon, confirm placement (auscultate and end-tidal CO_2)
  - Secure tube to skin



Cricothyrotomy

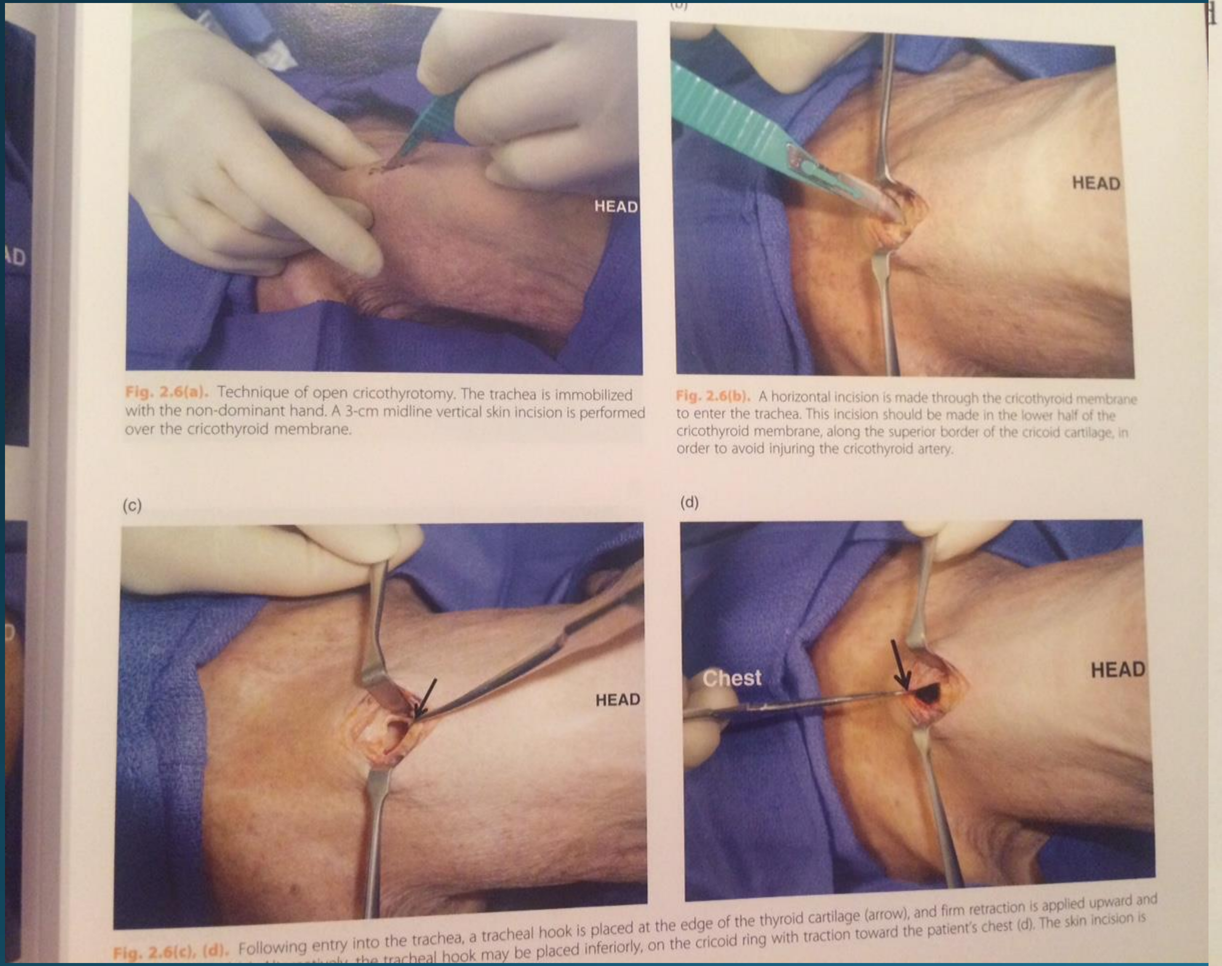
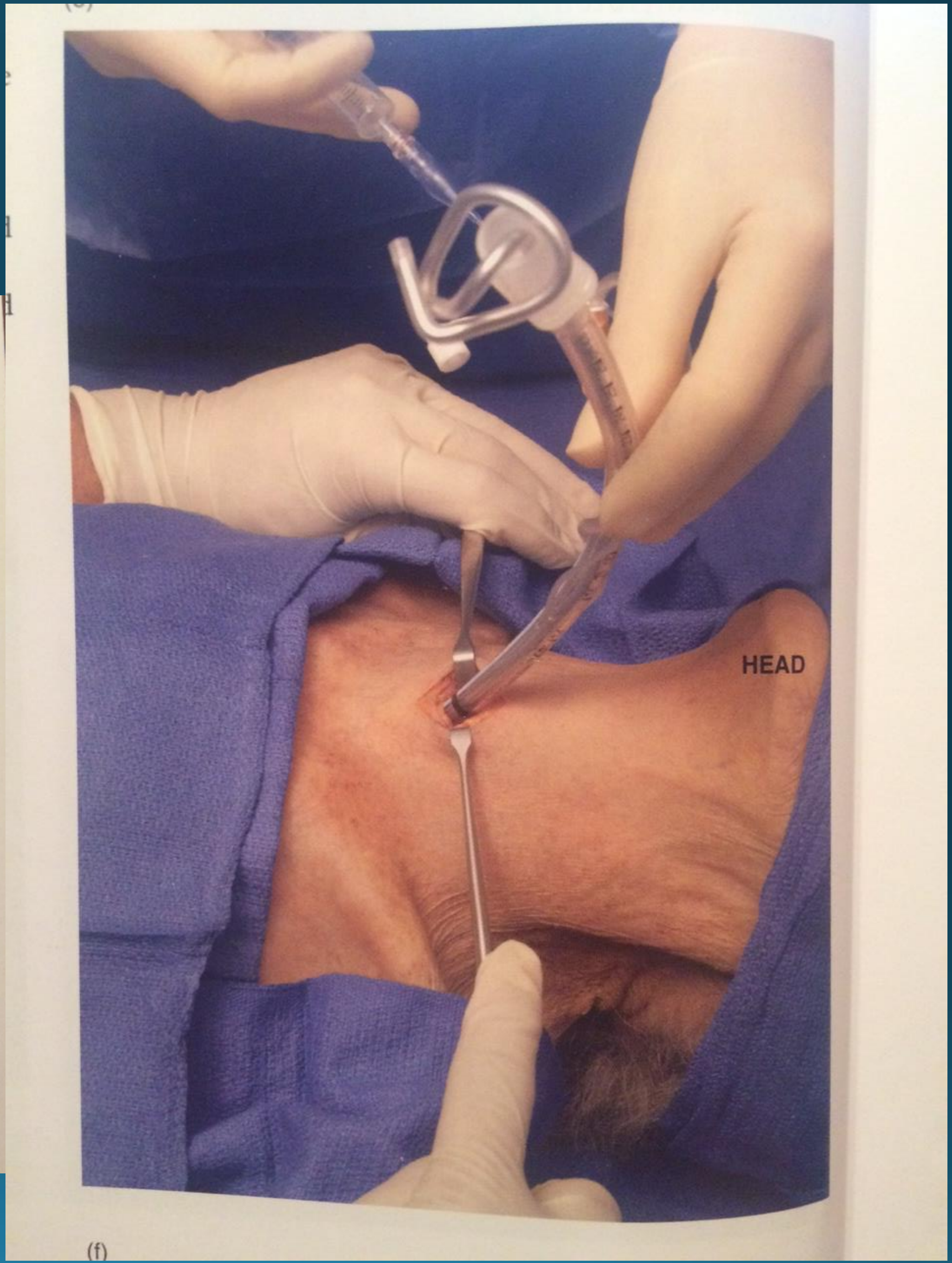


Fig. 2.6(a). Technique of open cricothyrotomy. The trachea is immobilized with the non-dominant hand. A 3-cm midline vertical skin incision is performed over the cricothyroid membrane.

Fig. 2.6(b). A horizontal incision is made through the cricothyroid membrane to enter the trachea. This incision should be made in the lower half of the cricothyroid membrane, along the superior border of the cricoid cartilage, in order to avoid injuring the cricothyroid artery.

Fig. 2.6(c), (d). Following entry into the trachea, a tracheal hook is placed at the edge of the thyroid cartilage (arrow), and firm retraction is applied upward and ... the tracheal hook may be placed inferiorly, on the cricoid ring with traction toward the patient's chest (d). The skin incision is

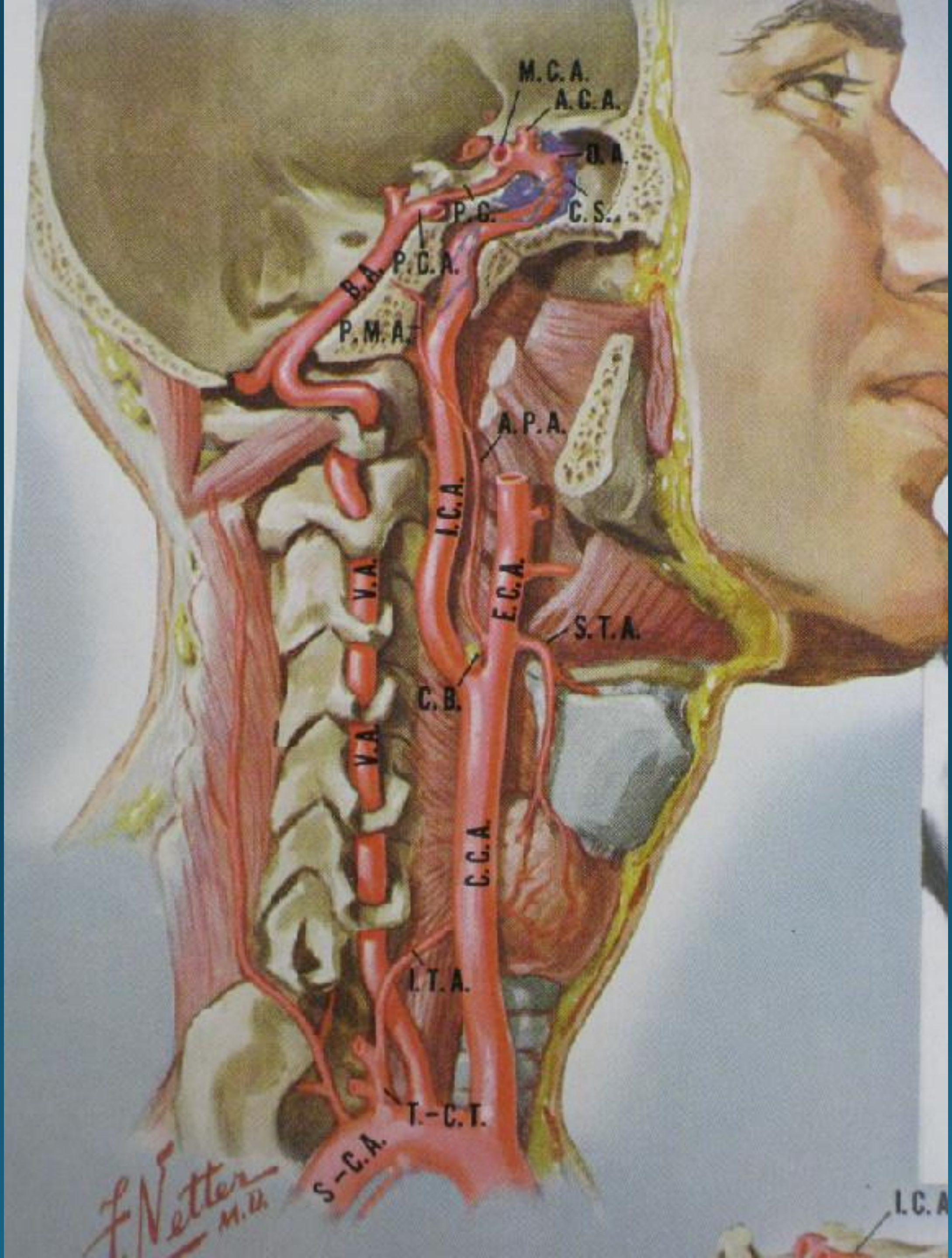


Penetrating neck injuries (PNI)

- Which patient should receive a trach?
  - Lefort fractures
  - Severely injured trauma patient likely to require prolonged ventilation (i.e. severe TBI)

Blunt cerebrovascular injuries

Dr. Vogt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T.-C.T.
S-C.A.
F. Netter M.D.
I.C.A

BVCI: Why do we care?

- Historically diagnosed in ~1% of blunt trauma patients
  - Likely more now

- **Significant** morbidity if undiagnosed
  - Stroke rate as high as 60%

Screening for BCVI

- Who should we screen?
  - Multiple differing recommendations
  - Difficult to know what we should do

ORIGINAL ARTICLE

Western Trauma Association Critical Decisions in Trauma: Screening for and Treatment of Blunt Cerebrovascular Injuries

Walter L. Biffl, MD, C. Clay Cothren, MD, Ernest E. Moore, MD, Rosemary Kozar, MD, Christine Cocanour, MD, James W. Davis, MD, Robert C. McIntyre, Jr., MD, Michael A. West, MD, PhD, and Frederick A. Moore, MD

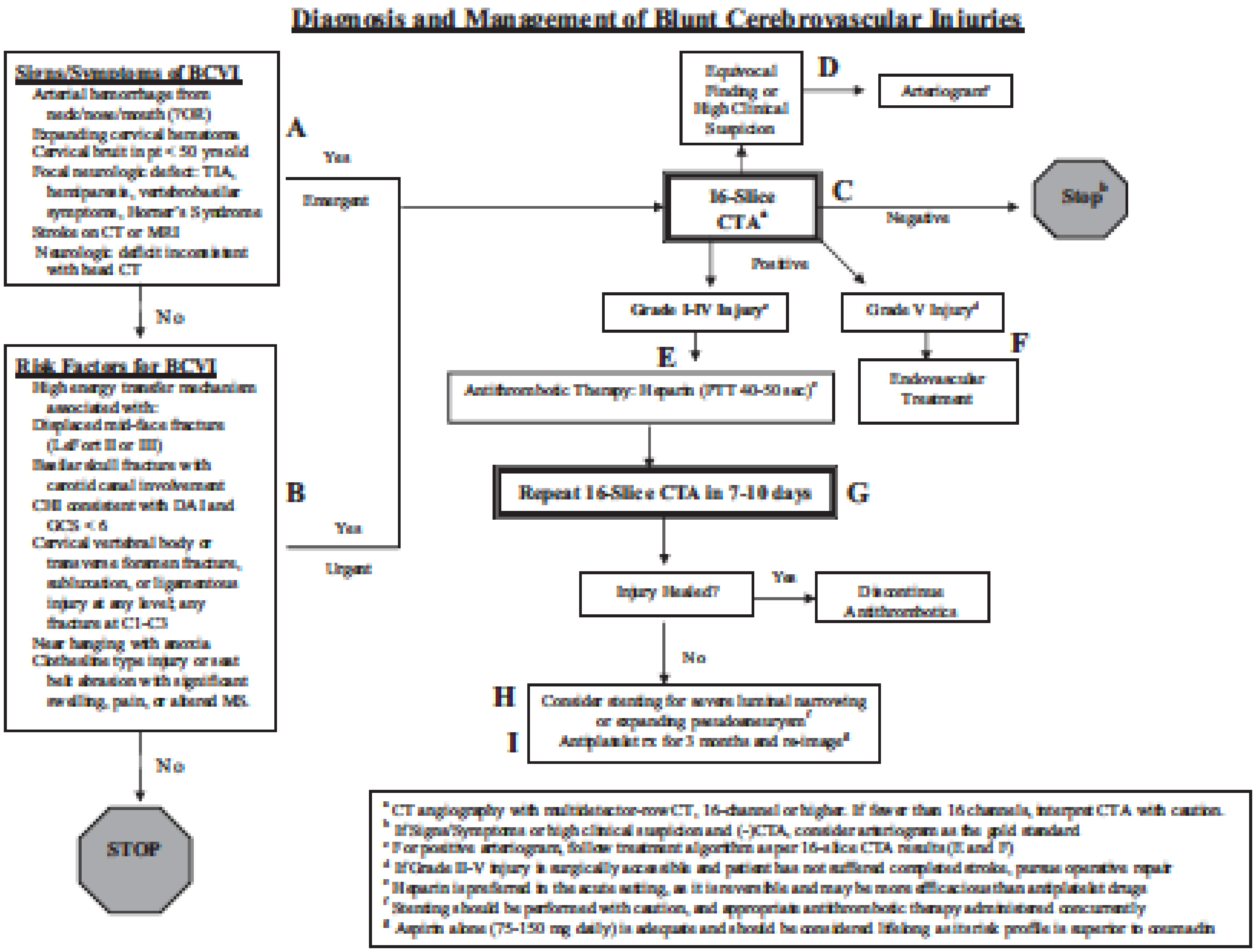

Figure 1. Algorithm for the diagnosis and management of blunt cerebrovascular injuries in adults.

TABLE 1. Screening Criteria for BCVI Adapted From Biffl et al[10] (With Permission)

Screening Criteria for BCVI
Injury mechanism
Severe cervical hyperextension/rotation or hyperflexion, particularly if associated with
Displaced midface or complex mandibular fracture
Closed head injury consistent with diffuse axonal injury
Near hanging resulting in anoxic brain injury
Physical signs
Seat belt abrasion or other soft tissue injury of the anterior neck resulting in significant swelling or altered mental status
Fracture in proximity to internal carotid or vertebral artery
Basilar skull fracture involving the carotid canal
Cervical vertebral body fracture

Table 1. Denver Screening Criteria for BCVI

Signs/symptoms of BCVI
Arterial hemorrhage
Cervical bruit
Expanding cervical hematoma
Focal neurologic deficit
Findings from neurological examination incongruous with head CT scan findings
Stroke on secondary CT scan
Risk factors for BCVI
High-energy transfer mechanism with Le Forte II or III fracture
Cervical spine fracture patterns: subluxation, fractures extending into the transverse foramen, fractures of C1-C3 vertebrae
Basilar skull fracture with carotid canal involvement
Diffuse axonal injury with GCS score <6
Near hanging with anoxic brain injury

Abbreviations: BCVI, blunt cerebrovascular injury; CT, computed tomographic; GCS, Glasgow Coma Scale.

CLINICAL MANAGEMENT UPDATE

Blunt Cerebrovascular Injury Practice Management Guidelines: The Eastern Association for the Surgery of Trauma

William J. Bromberg, MD, Bryan C. Collier, DO, Larry N. Diebel, MD, Kevin M. Dwyer, MD, Michelle R. Holevar, MD, David G. Jacobs, MD, Stanley J. Kurek, DO, Martin A. Schreiber, MD, Mark L. Shapiro, MD, and Todd R. Vogel, MD

EAST guidelines

- Any neurologic abnormality not explained by a diagnosed injury (GCS ≤ 8 with normal CT)
- Epistaxis from a suspected arterial source
- DAI
- C-spine fracture
    - C1 – 3; through transverse foramen; with subluxation or rotational component
- Lefort II or III fractures

What about seatbelt sign

- Not in the EAST guidelines but part of many other criteria

- What is the difference?
  - SYMPTOMS

- Practical answer
  - If it's a true seatbelt sign, you should probably screen

Blunt cerebrovascular injury screening guidelines: What are we willing to miss?

Brandon Robert Bruns, MD, Ronald Tesoriero, MD, Joseph Kufera, MA, Clint Sliker, MD, Adriana Laser, MD, Thomas M. Scalea, MD, *and* **Deborah M. Stein, MD,** *Baltimore, Maryland*

BACKGROUND: Blunt cerebrovascular injury (BCVI) is reported to occur in approximately 2% of blunt trauma patients, with a stroke rate of up to 20%. Guidelines for BCVI screening are based on clinical and radiographic findings. We hypothesized that liberal screening of the neck vasculature, as part of initial computed tomographic (CT) imaging in blunt trauma patients with significant mechanisms of injury, identifies BCVI that may go undetected.

METHODS: As per protocol, patients at risk for significant injuries undergo a noncontrast head CT scan followed by a multislice CT scan (40-slice or 64-slice) incorporating an intravenous contrast-enhanced pass from the circle of Willis through the pelvis (whole-body CT [WBCT] scan). The trauma registry was retrospectively reviewed, and all patients with BCVI from 2009 to 2012 were analyzed. Patients undergoing WBCT scan were then identified, and records were reviewed for BCVI indicators (skull base fracture, cervical spine injury, displaced facial fracture, mandible fracture, Glasgow Coma Scale score ≤ 8, flexion mechanism, hard signs of neck vascular injury, or focal neurologic deficit).

RESULTS: Of 16,026 patients evaluated during the study period, 256 (1.6%) were diagnosed with BCVI. The population consisted of 185 patients with suspected BCVI after WBCT scan. One hundred twenty-nine patients (70%) had at least one indicator for BCVI screening, while 56 (30%) had no radiographic or clinical risk factors; 48 of the 56 patients underwent confirmatory CT angiography of the neck within 71 hours of initial WBCT scan, with 35 patients having 45 injuries.

CONCLUSION: More liberalized screening for BCVI during initial CT imaging in trauma patients clinically judged to have sufficient mechanism is warranted. Using current BCVI screening guidelines leads to missed BCVI and risk of stroke. (*J Trauma Acute Care Surg*. 2014;76: 691–695. Copyright © 2014 by Lippincott Williams & Wilkins)

LEVEL OF EVIDENCE: Diagnostic study, level III.

KEY WORDS: Blunt cerebrovascular injury; screening; BCVI; blunt cervical injury.

Injury grades

TABLE 1. Blunt Carotid and Vertebral Arterial Injury Grading Scale[32]

Injury Grade	Description
I	Luminal irregularity or dissection with <25% luminal narrowing
II	Dissection or intramural hematoma with ≥25% luminal narrowing, intraluminal thrombus, or raised intimal flap
III	Pseudoaneurysm
IV	Occlusion
V	Transection with free extravasation

What if we find an injury?

- We don’t really know
- Current best answer: ASA
- Acceptable answer: Heparin