CNA Exam Review: Traumatic Injuries of the Brain

Studying Materials

Self-Learning Package: Overview of Adult Traumatic Brain Injuries:

Overview

- Pathophysiology and manifestations of traumatic brain injury
- Classification of brain injury
- Treatment options
- Goals for the TBI client
- Complications
- Nursing strategies
Pathophysiology and Manifestations of Traumatic Brain Injury

Mechanism of Injury: Penetrating
- Goes through skull and penetrates brain
- Primary injury:
  - Brain tissue laceration
  - Contusions
  - Shearing
- Secondary injury happens immediately
  - Cerebral edema
  - Tissue hypoxia
  - Tissue hypoxia
- Severity depends upon
  - Speed
  - Size
  - Shape
  - Direction
  - Location
  - Path travelled

Mechanism of Injury: Blunt Trauma
- Contact phenomena
- Result of head motion
  - Acceleration/deceleration
    - Compression
    - Tension
    - Shearing
    - Rotation
Mechanism of Injury: Blunt Trauma - Contact Phenomena

Contact Phenomena: Direct result of an object striking the head

Velocity of impact → extent of the injury

Low velocity
- Scalp lacerations
- Extradural hematoma
- Contusions
  - Restricted to scalp and skull

High velocity
- Skull fractures
- Intracerebral hemorrhage
  - Includes scalp, skull, and injuries to the brain

Blunt Injury: Coup & Contrecoup

Coup injuries occur at the site of impact and is usually the result of an object striking the head.

Contrecoup injury occurs at the opposite side or at the rebound site of impact.
**Pathophysiology:**
Sudden changes in velocity followed by a sudden stop
- Strain on cerebral tissue
  - Compression – where tissue is pushed together
  - Tension – where there is traction on the tissue
  - Shearing – where there is a parallel sliding motion of the planes of an object
  - Rotation (angular acceleration)

**Manifestations:**
- Acute subdural hematomas
- Diffuse axonal injury
- Contusions
- Lacerations
- Necrosis
- Areas commonly affected:
  - Inferior frontal & temporal lobes
  - Fronto & temporal poles.

**Classification of Brain Injury**
TBI severity of injury requires practitioners to look at the GCS value after initial resuscitation

<table>
<thead>
<tr>
<th>GCS Value</th>
<th>Severity</th>
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<tbody>
<tr>
<td>&lt; 8</td>
<td>Severe</td>
</tr>
<tr>
<td>9 - 12</td>
<td>Moderate</td>
</tr>
<tr>
<td>&gt; 12</td>
<td>Mild</td>
</tr>
</tbody>
</table>

Also considered in severity estimation:
- CT scan abnormalities
- Mechanism of injury
- Duration of post-traumatic amnesia
Primary Versus Secondary Injury

**Primary injury** is the initial "physical brain injury sustained at the moment of impact, leads to physical disruption of neurons, tearing of blood vessels and/or inadequate perfusion of the brain." (Woodward & Mestecky, 2011, p532)

Impacts neurons, brain tissue and/or blood supply

- **Neurons**
  - are fragile, axons delicate
  - shearing forces break the neuron axons.
- **Brain tissue**
  - torn and bruised by violent movements
- **Blood vessels**
  - on and within brain can tear and bleed

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**Secondary injury** is injury that happens subsequent to the primary event and can be divided into two areas

1. **Damage**
   - Lack of blood and O2 to cells,
   - Involves multiple metabolic mechanisms
   - Results in cytotoxic edema & inflammatory response

2. **Insults**
   - Preventable and treatable
   - Hypoxia, hypotension (SBP < 90), and delays in appropriate surgical management

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**Classification of Brain Injury**

<table>
<thead>
<tr>
<th>Area</th>
<th>Focal Damage</th>
<th>Diffuse Damage</th>
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<tbody>
<tr>
<td>Head</td>
<td>Scalp Lacerations, Contusions, Hematomas, Subgaleal Hematomas</td>
<td>Diffuse axonal injury, Concussion, Hypoxic-Ischemic damage, Diffuse brain swelling (edema)</td>
</tr>
<tr>
<td>Face</td>
<td>Fractures</td>
<td></td>
</tr>
<tr>
<td>Meninges</td>
<td>Dural tear, between skull &amp; Brain</td>
<td></td>
</tr>
<tr>
<td>Brain</td>
<td>Contusions, Intracerebral haemorrhage, Penetrating brain injury</td>
<td></td>
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</tbody>
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Damage can be classified as focal or diffuse
Diffuse Axonal Injury

Pathophysiology
- acceleration–deceleration/rotational forces stretch & shearing neurons
- Interfering with neuronal transmission
- Mainly at the junction of white and grey matter

Manifestations
- DAI visible on MRI
- Multiple small cerebral contusions on CT
- Varied neurological signs and symptoms

Classifications Of DAI
Made According To Coma Length & Brain Stem Signs

<table>
<thead>
<tr>
<th>Coma Length</th>
<th>Brain Stem Signs</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 24 hours</td>
<td>Begins to follow commands by 24 hours</td>
<td>Death uncommon</td>
</tr>
<tr>
<td>&gt; 24 hours</td>
<td>No permanent signs</td>
<td>Incomplete recovery in survivors</td>
</tr>
<tr>
<td>Post-traumatic</td>
<td>Permanent signs</td>
<td>Decortication, decerebration, death or severe disability</td>
</tr>
</tbody>
</table>

Hemorrhage

5 areas:
- Epidural
- Subdural
- Subarachnoid
- Intracerebral
- Intraventricular
Hemorrhage: Epidural

**Epidural** (epi = on, upon)
- Between skull and dura
- Uncommon (.05 to 6%)
- 85% are arterial (middle meningeal artery)
- Can be venous
- Pulls dura away from skull
- Creates egg appearance (ovoid)
- Pressure and mass effect leads to the symptoms displayed

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Hemorrhage: Epidural

**“Talk and Die”**

- Classic: momentary unresponsiveness, followed by period of lucidity… then sudden deterioration
  - 60% will not lose consciousness
  - Drowsiness → lethargy → coma
  - Brain herniates
  - Most common s/s: headache, vomiting, seizures, unilateral hyperreflexia with positive Babinski sign & elevated ICP
  - Other: Ipsilateral dilated, sluggish, fixed pupil, hemiparesis, hemiplegia, decortication, decerebration, resp distress, death

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Hemorrhage: Subdural

**Subdural** (sub = below, under)
- Bleeding between the arachnoid layer and dura
- 30% of post traumatic bleeds
- Mainly tearing of bridging veins
- Also tearing of small cortical arteries, contusions, and bleeding into chronic subdural hematomas
- Can be
  - Acute: up to 48 hours
  - Subacute: 2 days to 2-3 weeks
  - Chronic: > 3 weeks – several months
- Classified based on blood and fluid
  - Acute – clotted blood
  - Subacute – mix of clotted and fluid blood
  - Chronic – is composed of fluid only

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Hemorrhage: Subdural

Chronic SDH – headache which progresses in severity, slow cerebration, confusion, drowsiness, possibly seizure, pupillary edema, sluggish ipsilateral pupillary response, hemiparesis

Hemorrhage: SAH

Bleeding into the space below the subarachnoid membrane
- Seen with severe TBI
- Blood vessels stretched/torn during injury
- On CT not always visible
- Rely on presentation & brain injury for Dx

Manifestations
- Neurological deterioration
- Meningeal irritation
- Intracranial HTN
- Focal ischemia
- Localized cerebral edema
- Vomiting
- Thrombosis of blood vessels

Hemorrhage: Intracerebral

Pathophysiology:
- 4-15% of brain injured pts
- Bleeding into the cerebral parenchyma
- Related to contusions
- Occur like contusions
- Frontal & temporal lobes
- Expanding space occupying lesions
- Can be delayed (hours to days)
- Poor outcomes
- Increased mortality

Manifestations:
- Headache
- Decreased LOC to deep coma
- Hemiplegia
- Ipsilateral dilated pupil
- Transtentorial herniation
Hemorrhage: Intraventricular

Pathophysiology:
- Setting of severe TBI
- Secondary to SAH
- Extension of ICH

Manifestations:
- Related to increased ICP
- Meningeal irritation
- Stiff neck
- Ipsilateral pupil dilatation
- Deteriorating LOC
- Hemiparesis
- Hemiplegia
- Posturing

Concussions

Concussion = Mild traumatic brain injury

Pathophysiology:
- A primary injury
- Mechanisms of injury
  - Linear acceleration/deceleration
    - Grey matter most affected
  - Rotational
    - Axon shearing
    - Stress/strain on tissue, vasculature
    - Traumatic axonal injury
  - Blast
    - Blast waves (acceleration/deceleration)

Sports injury concussion grading scale
American Academy of Neurology

Grade 1
- Confusion transient
- No loss of consciousness
- Mental status abnormalities resolve < 15 min

Grade 2
- Confusion transient
- No loss of consciousness
- Symptoms/abnormalities > 15 min

Grade 3
- Any loss of consciousness (seconds – mins)
Concussion

WHO definition of MTBI
“Acute brain injury resulting from mechanical energy to the
head from external forces” (AANN, 2011)

Manifestations:
- Confusion
- Disorientation
- Loss of consciousness < 30 mins
- Post traumatic amnesia < 24 hours
- Transient s/s (focal, seizures)
- Intracranial lesion (no OR)
- GCS of 13 -15 30 mins following event

Contusions

“Bruising of the brain”

Pathophysiology:
- Blunt or penetrating trauma
- Swelling, bleeding, inc. ICP
- Generally frontal and temporal lobes

Manifestations:
- Depend on the size, amount of swelling and location.
- Change in level of consciousness,
- Seizures,
- Disorientation
- Headache
- Vomiting
- Signs of increased intracranial pressure.
- CT/MRI scans – small diffuse bleeding and edema.
- 24 hour CT - increased bleeding +/- localized cerebral edema

Skull Fractures

Linear
- Single fracture line on skull

Depressed
- Skull fracture with depressed fragments, tearing of dura & scalp
Skull Fractures

Causes: Motor vehicle crash, assault, sports injuries.
Facial fractures are described as Le Forte I, II or III.

**Le Forte I Fracture**
- Most common type
- Usually occur to an unrestrained driver
- Along the maxilla bone
- Manifestations:
  - Gross malocclusion
  - Intra-oral ecchymosis
  - Epistaxis
  - Airway edema (soft palate tissue, or tongue).
- Le Forte II & III fractures present a higher risk for bleeding as the internal maxillary artery could tear and bleed into the sinuses.
- Csf leak
- Tear may have occurred in the parotid duct gland

**Le Forte II Fracture**
- Mid-face separation
- Fracture between the malar and maxilla bones & across the nasal bone
- Also involves the orbit and ethmoid bones.
- Manifestations:
  - Dishpan face
  - Wrinkled bridge of the nose
  - Severe epistaxis
  - Edema along the fracture lines
  - Possible CSF leak

**Le Forte III Fracture**
- Craniofacial disruption
- Fracture of malar and the nasal bone
- Manifestations:
  - Malocclusion
  - Facial edema
  - Free-floating maxilla
  - CSF leak
  - Severe epistaxis
  - Likely severe airway compromise

Skull Fractures: Basilar

Presentation depends upon location of fracture:

**Anterior Fossa:**
- Rhinorrhea
- Naso-ocular
- Oculomotor palsy

**Middle Fossa:**
- Hemotympanum
- Otorrhea
- Vertigo
- Battle’s sign (causal post)
- Unilateral hearing loss
- Nausea

**Posterior Fossa:**
- Compression of the brainstem
  - Hypotension
  - Tachycardia
  - Alteration in respirations

Fracture involves base of skull. Can be linear or depressed.
Treatment Options and Goals for Clients with TBI

TBI Initial Management

- Avoid hypotension
- Avoid hypoxia

In the field

- Complete/rapid physiological resuscitation
  - MAP > 90, (CPP > 70)
    - Consider hypertonic saline
  - Maintain patent airway & adequate oxygen supply
    - Intubating for GCS under 9 if airway or oxygenation are inadequate
  - Immediately immobilize the head and neck
**TBI Initial Management: In the ER/Trauma Center**

**Goals:** resuscitation, stabilization, establish a diagnosis

- Placement of lines and devices
- Resuscitation & stabilization
- Diagnostic testing
- Decision re: need for emergency neurosurgery
- Labs/diagnostics
- Initial “Brain Resuscitation”

Ct is the gold standard for Dx of TBI … can show depressed skull fractures, EDH, SDH, ICH, contusion, and most DAL... if operable lesion present pt will go to the OR... if not then will go to the ICU

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**TBI Initial Management: In the ICU**

**Goals:**
1. Manage intracranial hypertension
2. Maintain adequate cerebral oxygen delivery meeting cerebral metabolic needs
3. Prevent secondary brain injury
4. Prevent other systemic problems that increase mortality, morbidity, and increased costs

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**Figure 1: Traumatic Signs and Symptoms of Clinical Practice Guidelines for the Relative Management of Adults with Severe TBI**
Nursing Management of Severe TBI

A Word About CPP

Normal:
- MBP = 70 – 90
- ICP = 10
- CPP = 60 – 80

CPP < 50 = significant risk for brain ischemia
CPP is arguably more important than ICP

Normal:
- MBP = 70 – 90
- ICP = 10
- CPP = 60 – 80

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Rehabilitation

Goals: Prevention, maintenance, restoration

- Cognitive deficits - debilitating to recover from
- Wide impact
- Complete assessment needed
  - Staff observation
    - Assessment tools
      - The Mini Mental State Examination
      - Neurobehavioural Cognitive Status Examination
      - Rancho Los Amigos Scale
  - Identify deficits
  - Plan strategies

Nurses role:

- Identify realistic goals & expectations
- Interventions for cognitive problems
  - see - Rancho Los Amigos Scale
- Communicate/explain behaviour to families
  - what to expect
  - how to interact
- Support family’s concerns
- D/C referrals for follow up care

Palliative Care

6,857 head injury patients died in hospital 2003/2004

Goals:

- Patient comfort
  - No respiratory distress (RR < 25)
  - No agitation, grimacing, discomfort, pain
- Support family
  - Offer pastoral support
  - Allow as much time as possible with patient
Complications, Nursing Strategies, Pharmacology

Seizures

Description
- 15% to 20% of patients with severe head injury.
- Early post trauma seizures = days 0-7
- Late post trauma seizures = after 7
- dramatically increase CBF, ICP, \( O_2 \) consumption

Pharmacology
- Anticonvulsants effective to prevent early post trauma seizures, but not delayed post trauma seizures.
- In the first week following a severe TBI, Phenytoin offers a temporary reduction in seizures.
- Other anti seizure drugs include:
  - Phenytoin
  - Carbamazepine
  - Valproic Acid
  - PHENobarbital

Drugs to stop acute seizures:
- Benzodiazepines: (Lorazepam, Diazepam, Midazolam)
- If very hard to manage use general anesthesia in the ICU

Nursing strategies
- Maintain seizure precautions
  - Intubated/airway management
  - Suction equipment available
  - Padded side rails left up when pt unattended
  - Protect pt from injury as able

- Observe for seizure activity
  - TBI - 50% may be subclinical (CEEG)
  - May not be evident if sedated or on paralytics
  - CEEG
  - watch for tachycardia, Bp instability, intracranial HTN,

- Monitor anticonvulsant drug levels
Cerebrospinal Fluid Leak

**Description**
- Commonly seen with basilar skull fractures

**How can you tell if it's CSF?**

**s/s**
- post nasal drip
- sweet or salty taste in their mouth
- coughing or clearing of throat
- visible drainage from ear or nose (esp. if basal skull fracture)
- Usually CSF and blood leak
- CSF has higher chloride concentration than serum
- Halo sign

**Self healing or patch**

**Nursing strategies**
- HOB 30 degrees
- Instruct not to: blow nose, drink via straw, drink hot liquids
- Do not attempt to clean nose or ears
- Cotton just outside ear (absorb otorrhea, replacing frequently)
- Do not block the flow of CSF
- Notify MD of CSF leak
- Avoid inserting any nasal tubes (select oral if needed)

**Pharmacology**
- Team may consider antibiotics
hydrocephalus

- Abnormal accumulation of CSF within the ventricles
- Caused by tumors, TBI, SAH
- CSF's path blocked
- Rx: drain placement

Airway/Respiratory Compromise

**Description**
- Ineffective airway clearance
- Atelectasis
- Pneumonia (aspiration, bacterial)
- Hypoxemia
- Ventilator dependency
- Neurogenic pulmonary edema
- ARDS
- Pulmonary embolus

**Nursing strategies**
- Assess ABC's
- Avoid increased CO2 and decreased O2
- Give O2
- Ventilate
- Limit suctioning to 10 sec
- Preoxygenate with 100% O2
- Turn frequently
- Physio
- VAP (ventilator acquired pneumonia) bundle

Airway/Respiratory Compromise

**Pharmacology**
- Antibiotics for pneumonia or aspiration
- Sedation for ventilation
- Anticoagulants
  - Heparin
  - Fragmin/Dalteparin
- Chlorhexidine mouth decontamination
- PUD prophylaxis
  - Pantoloc/Pantoprazole
Increased Intracranial Pressure

Description
Sustained elevation in ICP >15
Malignant hypertension is ICP >20

General strategies
Level 1 evidence
- Maintain ICP < 20
- EVD
Level 2 evidence
- Do not hyperventilate!
- Use sedation (avoiding coughing & agitation) Propofol
- Mannitol
- Elevate HOB 30 degrees
- Maintain normal temperature
Level 3 evidence
- Remove/loosen rigid cervical collars
- Intensive insulin therapy

Brain 80%
Blood 10%
CSF 10%

Other nursing interventions:
- Avoid obstructing venous return
  - Position head neutral
  - Loosen anything restricting the neck
- Respiratory problems
  - Airway obstruction
  - Hypoxia
  - hypercapnia
- Treat agitation and pain
- Treat hypernatremia, anemia, seizures

10 – 15 % of severe TBI patients have refractory intracranial hypertension

Treatments for Refractory ICH
- Moderate hyperthermia
- Hyperonic saline
- High dose barbiturates (paralysis)
- Hyperventilation

Surgical interventions
- Resection of mass lesions
- Cerebrospinal fluid drainage
- Decompressive craniectomy

Pharmacology
- Propofol
- Tylenol
- Insulin
- Mannitol
Infection

Description
Infection of initial injury, incision, drain, or line sites, or nosocomal infection. Hyperthermia increases metabolic rate, leading to ischemia for the injured brain.

Nursing strategies
- Universal precautions
- Strict aseptic technique
- Follow culture and dressing protocols
- VAP bundle

Pharmacology
- Antibiotics
- Tylenol

Fluid and Electrolyte Imbalances

Description
- Electrolyte imbalance
- SIADH
- Cerebral salt wasting
- Diabetes Insipidus
- Hyperglycemia
- Sepsis

Pharmacology
- Insulin
- Tylenol
- Antibiotics

Replacement electrolytes, DDAVP/vasopressin (in setting of DI)

Nursing strategies
- Monitor electrolytes, Bg, Ca, Phos, Mg, serum and urine osmolality, urine specific gravity, ongoing core temp, for sx's of sepsis
- Identify and treat underlying fluid and electrolyte abnormalities
- Maintain euvolemia
- Tightly control glucose
- Mannitol
- Keep normothermic
- Rx sepsis

Cerebral Edema

Vasogenic Oedema
- disruption of the blood brain barrier
- Leaky capillaries
- Setting of:
  - brain tumors
  - cerebral abscesses
  - maturing cerebral contusions
  - cerebral hemorrhages

Cytotoxic Oedema
- lack of ATP, cellular swelling
- Setting of:
  - Cerebral ischemia
  - Cerebral hypoxia
Cerebral Edema
- Vasogenic and cytotoxic edema can be seen together
- Edema is proportional to severity of injury
- Maximum edema in 2 - 4 days
- Gradually subsides
- Secondary injury can worsen edema and extend timeline

Nursing strategies:
- Are the same as those aimed at reducing ICP and maintaining adequate CPP

Depression
Feeling of sadness and self-depreciation accompanied by difficulty thinking and the ability to go about usual activities and carry on with usual responsibilities

s/s and classic behaviours include:
- Sad expressionless face
- Flat affect
- Listlessness
- Lack of interest in others/environment
- Possible crying spell
- Sense of hopelessness
- Have a difficult seeing a resolution to their situation
- May contemplate suicide

Nursing strategies:
- Take all allusions made to suicide seriously
- Consider psychiatric consultation/help
- Uncover reasons for depression
- Identify, address, and treat reasons
- Drug therapy
- Suicide assessment
- Suicide precautions
Importance of Injury Prevention Programs and Risk Reduction

Head Injury Prevention
- Aged 0 to 19
  - admissions decrease of 53%
  - 10,589 down to 4,966
- Aged 20 to 39
  - admission decrease of 45%
  - deaths after admission decrease 14%
- Aged 40 to 59
  - admissions decrease 9%
  - Deaths increased 15% (193 to 222)

Injury Prevention

Mechanism of injury
- Falls
- MVC
- Sports-related injuries
- Whiplash/pro hockey
- Blast injury

Prevention: Education
- Car restraint systems
- Distracted driving
- Riding in back of pickups
- Advocate use of helmets
- Violence/suicide prevention
- Water safety
- Falls prevention
- Athletes/parents – equipment, s/s

Child health promotion
- Encourage to follow sport/coach rules
- Good sportsmanship
- Helmets, mouth guards, padding
- Coaches check equipment
- Playground safety
- Teach parents ways to deal with stress
- Hospital based parent education

(AANN, 2011a)
References


