# Traumatic Brain Injury

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# Outline

- Epidemiology
- Classification
- Types
- Physical Exam
- Emergency room management
- IC-HTN and monitoring
- ICU protocols and management for raised ICP
- Outcome

# Epidemiology

- TBI is the leading cause of death and disability worldwide
- Incidence of TBI in Canada – 15 000/year

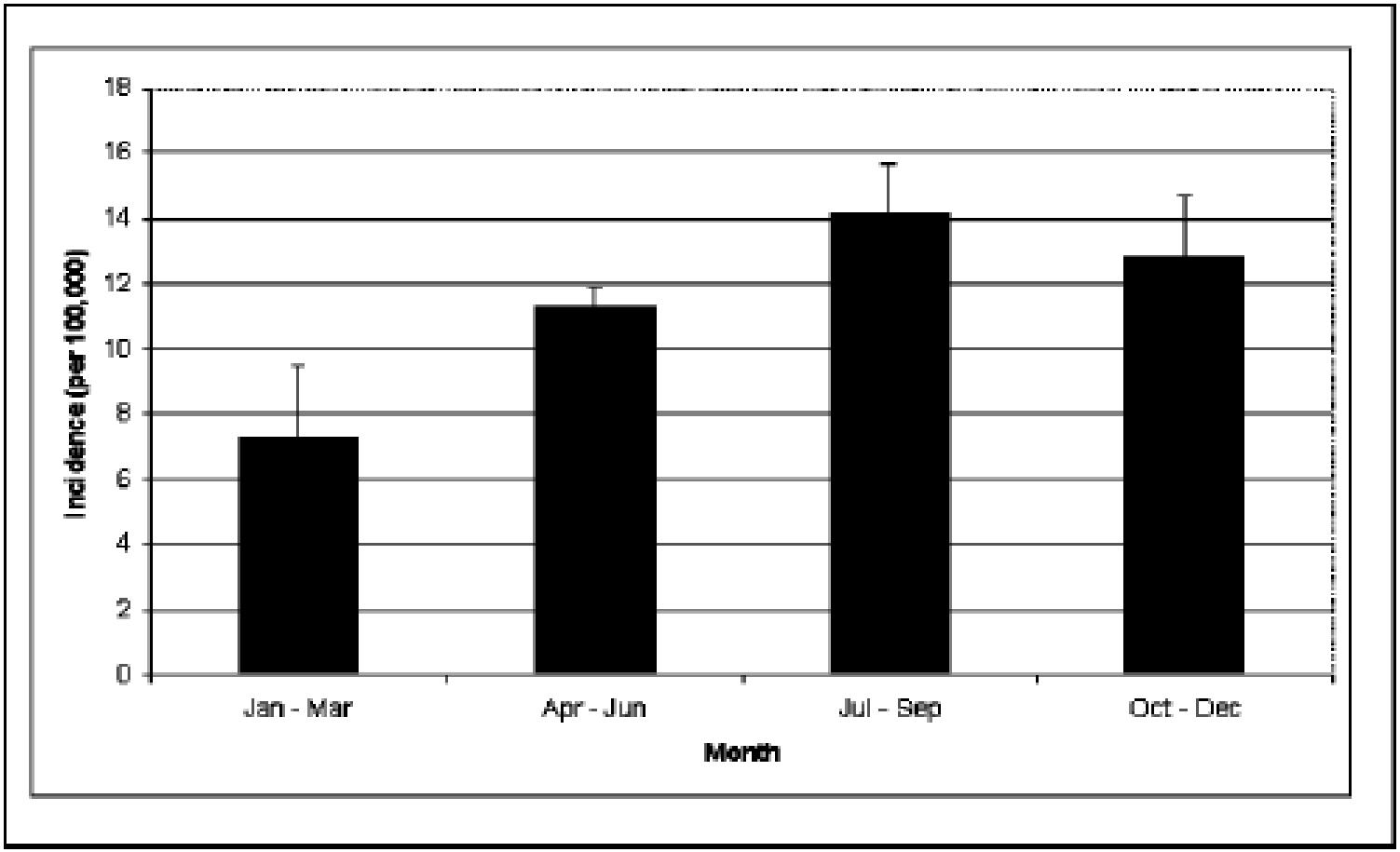
# Causes of TBI in Calgary

**Table 1: Characteristics of severe traumatic brain injury by etiology of injury in the Calgary Health Region (April 1999 to March 2002).**

| Primary accident etiology | Number of cases (%) | Mean Age (±SD) Years | Intentional Causality (%) | Overall Number of Deaths (%) |
|---|---|---|---|---|
| Motor vehicle crash | 113 (47) | 39 (±19) | 1 (1) | 48 (42) |
| Fall or jump | 81 (34) | 55 (±21) | 6 (8) | 38 (47) |
| Struck by object or animal | 29 (12) | 33 (±8) | 21 (54) | 7 (24) |
| Other | 19 (8) | 38 (±14) | 11 (75) | 15 (79) |
| Total | 242 (100) | 44 (±20) | 39 (17) | 108 (45) |

Zygun ‘05

# Quarterly Incidence of TBI in Calgary

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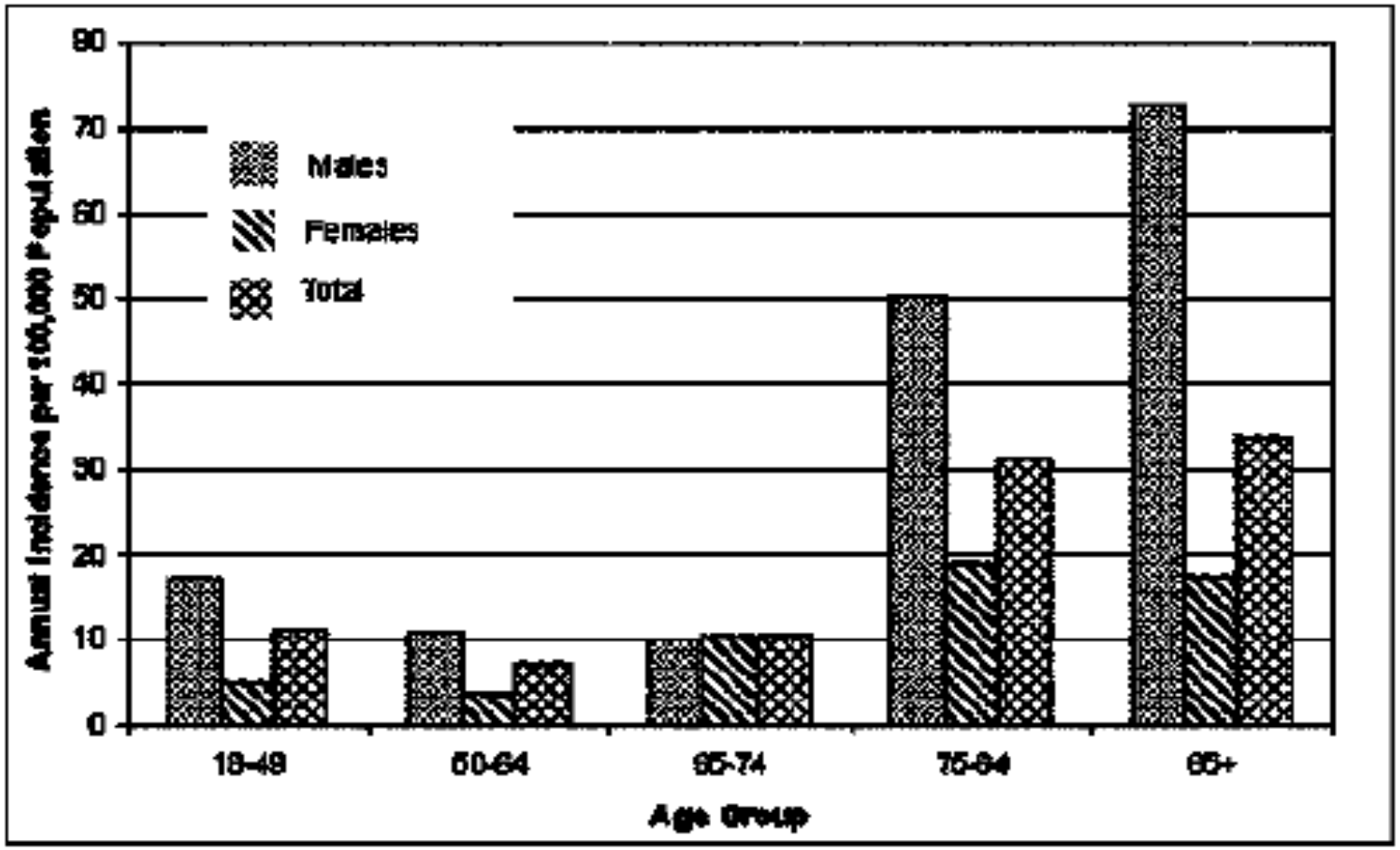
# Age and Gender in TBI in Calgary




Zygun ‘05

# Classification of TBI

| **Severity** | **GCS** |
|---|---|
| Mild | 14 – 15 |
| Moderate | 9 - 13 |
| Severe | 3 - 8 |

# How does trauma cause brain injury?

- Primary – impact damage
  - Contusions, lacerations, bone fragmentation, DAI, brainstem contusion
- Secondary
  - Intracranial hematomas, edema, hypoxemia, ischemia (especially due to elevated ICP and/or shock)

# Delayed deterioration after TBI

- Occurs in 15% of TBI patients. Causes:

1. Intracranial hematoma (EDH/SDH/contusion)
2. Posttraumatic brain swelling
3. Hydrocephalus
4. Tension pneumocephalus
5. Seizure
6. Metabolic abnormalities
7. Vascular events (DST, Carotid dissection, fat embolism
8. Meningitis
9. Shock

# Types of TBI - Concussion

1. Concussion:
   - Alteration of consciousness as a result of closed head injury
     - Grade 1 (mild) – posttraumatic amnesia <30min with no LOC
     - Grade 2 (mod) – PTA > 30min with LOC < 5min
     - Grade 3 (severe) – PTA > 24hrs with LOC > 5min
   - Confusion, amnesia, incoordination/ataxia, emotional lability, delayed response, slurred speech

# Types of TBI – Concussion (con't)

- Return to play following concussion:
  - General rule: a symptomatic player should not return to competition
  - Grade 1: may return if symptoms resolve in 15min
  - Grade 2: return in 1 week if symptoms resolved
  - Grade 3: go to hospital, do not return to sports for at least 2 weeks

# Types of TBI - Contusion

2. Contusion: Radiologic diagnosis, area of high (hemorrhagic) or low (edema) density on CT
   - Due to direct impact of brain on bony prominences of skull (temporal, frontal, occipital poles)
   - Can be the coup itself, of the contracoup.

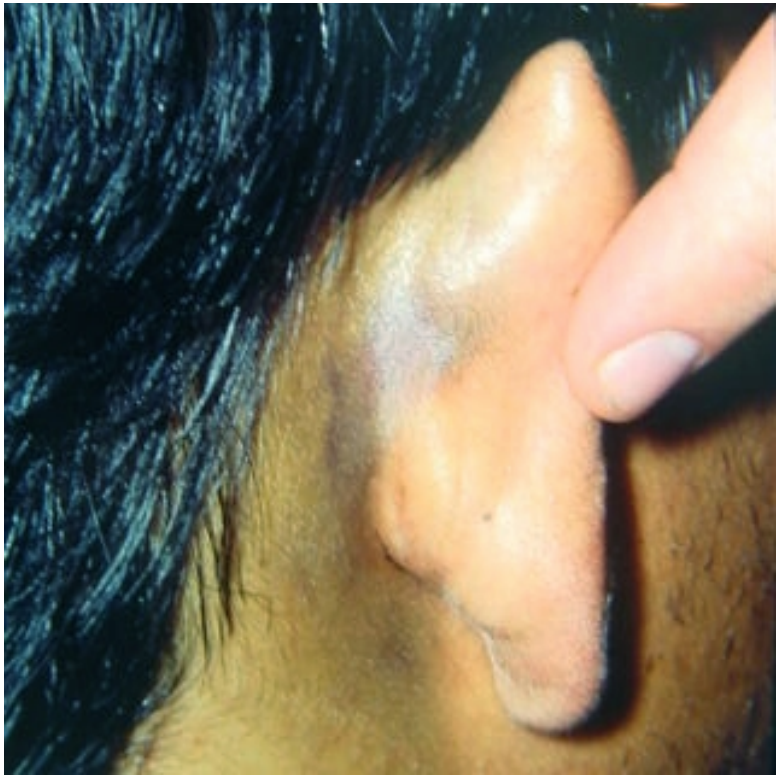
# Types of TBI - DAI

3. Diffuse Axonal Injury: a primary lesion of rotational acceleration/deceleration head injury
   - Lesions typically in white matter - can have hemorrhagic foci in corpus callosum and brain stem
   - Microscopic evidence of injury to axons
   - Often causes coma immediately after injury in absence of space occupying lesion on CT (although DAI can also occur with SDH/EDH)

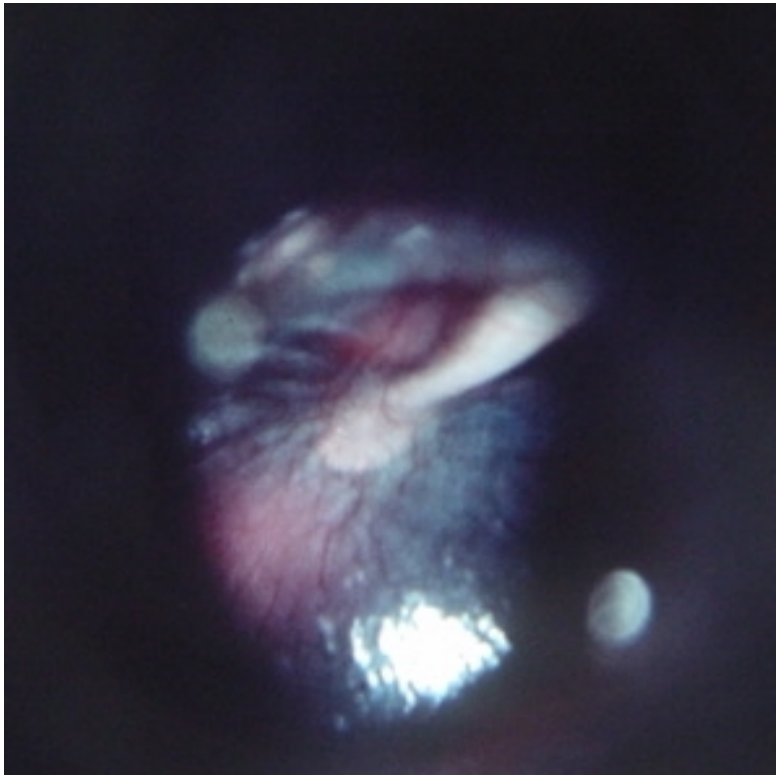
# NSx Exam in Brain Trauma

- If pt is awake, do full neuro exam
- If pt is comatose, determine GCS and do…
  - Inspection
    - Racoon eyes, battle sign, CSF rhinorrhea/otorrhea, hemotympanum, facial fractures
  - Reflexes
    - Pupils, corneals, gag

Battle sign



Hemotympanum



# TBI Management in ED

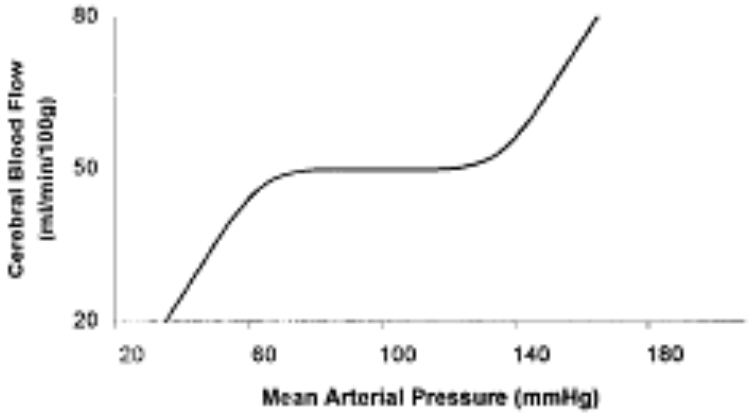
1. ABCs
   - Intubate if GCS < 8, ventilate to correct hypoxia, infuse fluids to correct hypotension
   - Remember – hypotension is rarely attributable to head injury
2. Ensure the pt is fully recovered from chemical paralysis and/or sedation before performing a neuro exam
3. Because hyperventilation can exacerbate cerebral ischemia, it should not be used prophylactically (only use if CT or clinical signs of IC-HTN are present – if used, use only briefly)
4. Prophylactic anticonvulsants are only recommended for 1 week post injury (have not been shown to decrease morbidity or mortality)

# TBI Management in ED (con't)

5. Mannitol (0.5-1.0g/kg bolus, peak effect 20min)
   - Only use if patient is adequately volume-resuscitated
   - Indications:
     - Evidence of IC-HTN (pupils asymmetric or dilated, posturing, deterioration in neuro exam)
     - Evidence of mass effect (e.g. hemiparesis)
     - After CT, if a lesion associated with increased ICP is identified
     - After CT, if pt going to OR
     - To assess "salvageability" – pt with no evidence of brainstem function, look for return of brainstem reflexes

# Intracranial Pressure (review)

- CPP = MAP – ICP




- Normal CPP for adult is > 50 mmHg
- Normal ICP for adult is < 10 -15 mmHg
- Cerebral autoregulation is a mechanism whereby large changes in systemic BP produce only small changes in CBF
  - Note: Autoregulation is impaired in trauma (i.e. CBF will be dependent on CPP)

# Causes of IC-HTN

1. Cerebral edema
2. Hyperemia – normal response to head injury, especially days 1-5
3. Traumatically induced masses, either initial or delayed (eg EDH, evolving contusion)
4. Hydrocephalus
5. Hypoventilation (causes hypercarbia and vasodilation)
6. Systemic hypertension
7. Venous sinus thrombosis
8. Increased muscle tone and valsalva
9. Status epilepticus

- Note: Secondary rise in ICP (days 3-10) can be due to delayed hematoma formation/enlargement, cerebral vasospasm, ARDS causing hypoventilation, delayed edema formation, hyponatremia

# Indications for ICP Monitoring

- GCS < or equal to 8 (after cardiopulmonary resuscitation) and either:
  - Abnormal admitting CT head

  OR
  - A normal CT, but with 2 or more of:
    1. Age > 40
    2. SBP < 90 mmHg
    3. Decerebrate or decorticate posturing (uni or bilateral)

# Methods for ICP Monitoring

1. EVD
   - Also allows drainage of CSF
   - Can be re-zeroed/calibrated
2. Codman (intraparenchymal ICP monitoring)
   - Cannot be re-zeroed
   - Good choice if small ventricles
3. Licox
   - 3 ports – temperature, O2 sats, and a port for a Codman

- Note: Insertion of an ICP monitor in trauma is *contraindicated* in coagulopathy

# Adjunct to ICP monitoring

- Jugular venous monitoring
  - Jugular venous O2 saturation (SJO2) sampled from jugular bulb gives additional data on cerebral perfusion
  - Use intermittent sampling
  - Multiple venous desaturations (<50%) or sustained/profound desaturation episodes are associated with poor outcome

# ICU Management of TBI

- Canadian Neurocritical Care Group – founded in 1992 by Drs. Draga Jichici and David Zygun
  - *An organized approach with special training, experience and protocols for treating various conditions produces improved outcomes and is cost-effective*
  - *Collaboration of disciplines is optimal*

# Management of TBI in the ICU

- General:
  - HOB at 30 degrees
    - Reduces ICP by enhancing venous outflow (but also reduces mean carotid pressure – therefore no net change in CBF)
    - Onset is immediate
  - Keep neck straight, avoid tight trach tape
    - Constriction of jugular venous outflow increases ICP
  - Enteral nutrition as per protocol
  - Stockings for DVT prophylaxis (at least initially)
  - Seizure prophylaxis (Dilantin)

# Management of TBI in the ICU (con't)

- ICU physiologic goals:
  - CPP 60-70 mmHg (Greenberg recommends > 70)
    - Use fluids and pressors if needed
  - MAP > 80 mmHg
  - ICP < 20 mm Hg
    - Prevents "plateau waves" from compromising CBF and causing cerebral ischemia
  - PaCO2 35-40 mmHg
  - PaO2 > 80-120 mmHg
    - Hypoxia may cause further ischemic brain injury
  - Hgb > 90 g/dl
    - Transfusion of pRBC improves brain tissue oxygenation (Zygun '09)
  - Temp 36.0 – 37.5 (euthermia)
    - Fever is a potent stimulus to increase CBF, may also increase plateau waves
    - If Tylenol is not enough, use intravascular cooling
  - Euvolemia (fluid balance positive 0.5 – 1.0L/24hrs)
  - Avoid hyper or hypoglycemia
    - Hyperglycemia causes brain tissue acidosis (Zygun '04)
    - Low brain tissue pH worsens outcome in TBI (Zygun '04)

# Treatment of ICP – ICU protocol

- If ICP > 20 mmHg – initial measures
  - Fentanyl 25-150ug/hr, propofol 0-50ug/kg/hr for full sedation
    - Sedation blunts the elevations in ICP that occur with movement/straining/agitation
  - EVD drainage
    - Reduces intracranial volume

# Treatment of ICP – ICU protocol (con't)

- If previous not sufficient, then add…
  - Paralyze with pancuronium
    - Same principle as sedation
  - Cool to 35 degrees
    - The effect on outcome of hypothermia is being studied (NABISH)
    - Monitor for drop in cardiac index, decrease plts, pancreatitis
    - Avoid shivering – raises ICP
  - Osmol therapy:
    - Hypertonic Saline 5% 2 ml/kg q2h prn for ICP > 20 mm Hg for 10 minutes (serum Na never >160)
    - Alternate: Mannitol 0.25 - 1g/kg q6h prn
      - Lowers ICP by causing immediate plasma expansion (reduces blood viscosity – improved rheology – which increases CBF and O2 delivery), and has a longer osmotic effect to draw edema fluid from cerebral parenchyma
  - Consider mild hyperventilation (paCO2 30-34)
    - Reducing CO2 causes cerebral vasoconstriction, thus reducing CBV and lowering ICP
      - But vasoconstriction also decreases CBF, especially in first 24hrs
      - So, only use for brief periods or as a last line measure if prolonged

# Treatment of ICP – ICU protocol (con't)

- If still not sufficient, then add…
  - Cool to 34 degrees
  - Pentobartital coma
    - Lowers ICP but limited by hypotensive side effects
  - Consider decompressive craniectomy
    - Removal of portion of calvaria, and/or large areas of contused brain, and/or temporal or frontal lobectomy
    - Duroplasty is mandatory

# Outcome

- Anything from complete recovery to death
- Older age, pre-injury unemployment, pre-injury substance abuse and more disability at rehabilitation discharge are important predictors of long-term disability (Murray ’07)
- For people who have sustained a TBI and survived the initial acute phase of care, a considerable increased risk of excess hospitalizations, increased lengths of stay in hospital care and increased rates of physician claims remained across almost every year of follow-up regardless of the level of injury severity (Cameron ’08)

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