Definition of Traumatic Brain Injury

Traumatic brain injury (TBI) is a non-degenerative, non-congenital insult to the brain from an external mechanical force (possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions) with an associated diminished or altered state of consciousness.

Scale of the Problem

UK (DOH 2001/Jennett 1986)
- 1.5 million patients attend A&E departments with head injuries yearly
- Of these 10 – 30% will be admitted and 2,000 adults a year suffer serious impairment which remain with them forever.

USA (Nat Head Injury Foundation 1992)
- Over a million patients per year sustain head injuries severe enough to require hospitalisation
- Of these, 30,000 – 50,000 persons have such serious intellectual and behavioural dysfunction that they are unable to return to normal life
**Epidemiology Of TBI**

**Annual incidence of TBI**

@ 300 /100,000

- severe 8
- moderate 18
- mild 274

- large variation: - M.F 2.5 : 1
- 66% < 30 yo

↑'d with: - urban/social deprivation
- poor academic performance
- poorer social/vocational background
- previous HI
- drug and alcohol abuse
- forensic history/psychopathology

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**Grading of TBI**

<table>
<thead>
<tr>
<th>GCS</th>
<th>Mild</th>
<th></th>
<th>Moderate</th>
<th>9 – 12</th>
<th></th>
<th>Severe</th>
<th>3 – 8</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>at scene</td>
<td></td>
<td></td>
<td>at A&amp;E</td>
<td></td>
<td></td>
<td>lowest within 24 hours</td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td>lowest within 48 hours</td>
</tr>
<tr>
<td>GCS</td>
<td>Mild</td>
<td>&lt; 60mins</td>
<td></td>
<td>Moderate</td>
<td>&lt; 24hrs</td>
<td></td>
<td>Severe</td>
</tr>
</tbody>
</table>

**Glasgow Coma Scale (3 – 15)**

Eye opening
- Spontaneous = 4
- To speech = 3
- To painful stimulation = 2
- No response = 1

Motor response
- Follows commands = 6
- Makes localizing movements to pain = 5
- Makes withdrawal movements to pain = 4
- Flexor (decorticate) posturing to pain = 3
- Extensor (decerebrate) posturing to pain = 2
- No response = 1

Verbal response
- Oriented to person, place, and date = 5
- Converses but is disoriented = 4
- Says inappropriate words = 3
- Says incomprehensible sounds = 2
- No response = 1

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**Epidemiology Of TBI**

**Prevalence**

- prevalence of severe disability post-HI 150 /100,000

- ↑ing prevalence - much improved surgical care in last 30yrs , 90% of 1970’s severe TBI deaths would now live.

**Epidemiological problems**

- poor data collection - many mild TBI don’t present
- ↑ >50% TBI in-pats not ICD coded
- out-dated figures - though increased safety features, more car usage and more assaults
Causes Of Traumatic Brain Injury

- Road Traffic Accidents 45%
- Domestic/Industl Accidents 25%
- Sports/Leisure Injury 12%
- Assaults 10%
- Other 8%

Common Sites Of Injury

Neuropsychiatric Brain Regions

*Begler et al (2005)* used Voxel Based Morphology to compare neurobehaviourally challenged TBI patients to controls.

Areas highlighted were:
- frontal lobes and sub-cortical white matter
- deeper midline structures including the basal ganglia
- rostral brainstem
- temporal lobes and hippocampi
### Primary Injuries

**Forces**
- Penetrating vs Non-penetrating (open/closed)
- Acceleration vs Non-acceleration (coup/contrecoup)
- Shear or linear vs Rotational (inertia)

**Damage**
- Contusion
- Laceration
- Haemorrhage
- Diffuse Axonal Injury

### Secondary Injuries

- Haemorrhage (EDH, SDH, ICH, IVH, SAH)
- Oedema
- Raised Intracranial Pressure
- Ischaemia
- Hypoxia
- Hydrocephalus
- Infection
- Pneumocephalus
- Brain herniation

### Complicating Injuries Elsewhere

- Hypoxia - respiratory arrest
- Hypotension - shock
- Hypovolaemia - blood loss
- Metabolic Disturbance - iatrogenic
Delayed Complications

- Post Traumatic Epilepsy (10x in severe TBI)
- Hydrocephalus
- Unrecognised skull fracture -Infection/Abcess
  - Pneumocephalus
- DVT

Post Traumatic Amnesia (PTA)

A period of relatively clear consciousness but with disorientation and marked confusion - (as opposed to delerium – which will normally be treated by neurosurgeon/ neurologist/ physician)

- the time from the moment of injury to the time of resumption of normal continuous memory (latterly can get “islands of memory”)
- length of PTA used as guide to severity of insult
- theoretically traumatic brain injury term, but similar phases seen after stroke and other neurological conditions

Characteristics of a Patient in PTA

<table>
<thead>
<tr>
<th>Cognitive Features</th>
<th>Behavioural Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disorientated to time and place</td>
<td>Restlessness</td>
</tr>
<tr>
<td>Confusion</td>
<td>Agitation/irritation</td>
</tr>
<tr>
<td>Confused speech content</td>
<td>Aggression (verbal and physical)</td>
</tr>
<tr>
<td>Impaired memory</td>
<td>Easily fatigued</td>
</tr>
<tr>
<td>Easily distracted</td>
<td>Wandering</td>
</tr>
<tr>
<td>Poor concentration</td>
<td>Absconding</td>
</tr>
<tr>
<td>Slow information processing</td>
<td></td>
</tr>
<tr>
<td>Easily overloaded</td>
<td></td>
</tr>
<tr>
<td>Perseveration (stuck on a thought)</td>
<td></td>
</tr>
</tbody>
</table>
Management of PTA

1. Exclude physical causes: infection, ↓Na, hydrocephalus, iatrocauses (medication/fluids), alcohol/drug withdrawal, pain
2. Keep communication simple and slow, closed questions, non-confrontational, allow for pauses and slow processing
3. Review the environment: not over-stimulating, space to wander, familiar possessions, regular staff, small no. of visitors, plenty of rest, avoid day/night disruption
4. Would “1:1”/RMN nursing help?

Management of PTA

Medication
1. Avoid medication whenever possible: - calmer environment/side room, consider “special” nursing or transfer, ??MHA
2. Propranolol, Carbamazepine - less sedative properties
3. Buspirone, Lorazepam – can ↑ disinhibition & agitation
4. Neuroleptics as last resort, - use atypicals (Olanzapine, Risperidone), smallest dose possible, discontinue asap

Recovery

Timescale
- Inverse exponential improvement for first 6 – 12 months
- Then slower improvement over next 4 years
- Can improve up to 15 years post event
- 5% show late deterioration
- Life expectancy is approximately normal

BUT - multitude of variables in patients with TBI has resulted in no objective measures being found that are of clinical significance in predicting the recovery of an individual patient
Recovery
6 months after injury

Of the notional 18 moderate and 8 severe TBIs:
- 2 will have severe disabilities/coma
- 6 will have moderate disabilities
- 18 will have made a good physical recovery

BUT Less than 1 in 6 patients with severe TBI who were in employment prior to the injury return to work by 5 years

Factors influencing TBI sequelae and disability

• Area of brain affected
• Extent of injury

• Behaviour and personality premorbidly
• Levels of education and income
• Support of family, friends and employers
• Use of mind altering substances
• Response to injury and expectations (patient and others)

Sensorimotor Sequelae 1

Vision
• Optic nerve blindness, acuity defects
• Optic pathways field defects
• Cranial nerves eye movement disorders, diplopia, blurred vision
• (Retinal detachment/haemorrhage)
**Sensorimotor Sequelae 2**

**Speech, Swallow, Taste, Smell**

- Dysarthria phonation problems
- Dyspraxia speech motor coordination
- Dysphagia sensory and motor arms of swallow
- G/pharyngeal N taste and tongue sensation
- Olfactory N smell and taste

- Complications of tracheostomy, nasogastric (NG) and Percutaneous Endoscopic Gastrostomy (PEG) tubes

**Sensorimotor Sequelae 3**

**Hearing and Balance**

- Organ of Conti sensorineural (often bilateral)
- Middle ear conductive (blood/ossicle disruption)
- Periph/central tinnitus
- Labyrinth benign positional vertigo
- Central speech comprehension

**Incontinence**

**Sensorimotor Sequelae 4**

**Motor**

- Paralysis / motor weakness/spasticity/contractures
- Ataxia / loss of fine movement

**Sensory**

- Tactile sensation problems
- Proprioception problems
- Sensory inattention
- Anosognosia
- Neuropathic pain
Neuropsychiatric Sequelae

Cognitive, behavioural and personality defects give rise to most of the morbidity and most impair the capacity of the brain-injured to return to work and maintain social activities.

Failure in the individual’s premorbid role and status at home and at work frequently leads to a downward spiral of increasing demoralisation, social withdrawal and eccentricity, distress within the family, and a further decline in self-image.

Of the moderate and severely injured groups – virtually all will be left with some neurobehavioural disability.

Cognitive Sequelae

- Attention and concentration difficulties
- Poor memory
- Slow processing
- Poor planning and problem solving
- Problem with complex tasks (cooking, eating & dressing)
- Intellectual changes (abstraction, reasoning etc.)
- Communication problems (perseveration, word finding, comprehension, reading, writing)
- Being overwhelmed in stimulating environments
- Visuospatial difficulties
- Fatigue

Personality changes

- Exaggeration of traits (suspiciousness, anxiety, disruptiveness, inflexibility)
- Irritability, aggression & violence (up to 70% in severe head injuries)
- ‘Childishness’, emotional lability, restlessness
- Decreased social contact, decreased spontaneity, poverty of ideas
- Disinhibition, impulsivity
- Apathy
- ↓↓ libido (sometimes increased)
### Psychiatric Sequelae

**Depression**
- 25-60% of TBI suffer depressive episode within 8 years
- significant number have premorbid history of depression
- linked to poorer cognitive, functional & social outcomes
- ? cholinergic/adrenergic imbalance

BUT: - be careful of demoralisation, grief reaction as well as "frontal lobe" symptoms such as apathy, anhedonia and cognitive slowing

**Treatment** – SSRIs, (Citalopram), ?neurotrophic
CBT/psychotherapy dependant on Exec. Function

**Mania/ Bipolar Disorder**
- much less common than depression
- distinguish from “frontal” symptoms of disinhibition, fatuous behaviour and poor self-awareness
- can be rapid-cycling

**Treatment**  Carbamazepine, Valproate, Lithium

**Suicide**
- 4 x rate in normal Australian population
- 23% of TBI out-patients in Liverpool had clinically significant suicidal ideation
- requires careful history taking with particular regard to the degree of hopelessness

**Anxiety**
All forms of anxiety disorder have ↑ incidence (but small samples/studies and wide variation):
- GAD 9-24%, OCD 6-15%, panic disorder 6-14%, PTSD 6-19%

*BUT* large overlap with symptoms of Post-Concussive Syndrome; eg headache, dizziness, blurred vision, irritability, sensitivity to light and noise, poor concentration and attention

PTSD-like syndrome despite amnesia of event/trauma

**Treatment**  - antidepressant –SSRI (Citalopram/Sertraline)
- 2nd line – atypical antipsychotics, ?Pregabalin
- CBT + education
Psychiatric Sequelae

**Psychosis**
- Symptoms can occur:
  1. During Post Traumatic Amnesia (PTA)
  2. As a complication of P T Epilepsy
  3. Within TBI-related mood disorders
  4. In chr. schizophrenia-like syndrome
     - Associated with left-sided temp. lobe lesions

- Early psychosis frequently characterised by paranoia and delusional misidentification syndromes, eg reduplicative amnesias, Capgras & Fregoli synd.

**Treatment**
- Depends on accurate diagnosis and etiology of symptoms
- Check for:
  - Premorbid psychiatric diagnosis/family history
  - Seizure disorder (time course/rapid resolution)
  - Mood disorder
  - Concurrent or past substance abuse

Psychiatric Sequelae

**Psychosis (contd)**
- Medication:
  - PTA – try to avoid medication
  - PTE – anti-convulsant
  - Mood – antidepressant, (if sedation required consider trazadone)
    (+/- antipsychotic in initial stages, +/− anti-cycling agent)
  - Psychotic syndrome – antipsychotic – eg quetiapine, olanzapine
    (+/- substance abuse treatment)

**But:**
- Be aware of confusion between s/s of antipsychotics and symptoms of TBI eg: ↑ seizures, ↓ motor function/gait, ↓ arousal, ↓ speed of processing

**Also:**
- In TBI: ↑ sensitivity to antipsychotics, ↑ risk of TD, ↓ neuroplasticity

Psychiatric Sequelae

**Substance Abuse**
- At 50% of all TBI involve use of alcohol (25% intoxicated)
- Majority of these have pre-injury patterns suggestive of addictive drinking
- Presence of alcohol → longer duration of unconsciousness
  - Challenging behaviour in acute stages
  - Cognitive/overall outcome, ↑ mortality
- TBI sequelae of ↓ impulse control and anxiety lead to ↑ abuse

**Treatment**
- Initial withdrawal management
  - Counselling and group work (but poor compliance due to ↓ cogn functions/memory, ↓ impulse control, ↓ awareness, overstimulation of group, poor social functioning)
  - Therefore: structured setting, approp. cogn. level, use of “buddy”
Psychiatric Sequelae

Dementia
Some studies suggest either ↑ frequency or earlier onset of dementia in TBI sufferers, but overlap of diagnostic features (eg impairment of memory and other cogn.function)
- both associated with abnormal amyloid and Tau deposition
- ApoE – e4 allele linked to SDAT and poor outcomes in TBI and stroke

Treatment – following recovery from TBI and stable cogn function, any evidence of cognitive decline should be aggressively investigated, as not normal course for TBI

Consequences To The Family
Affective states of close relatives deteriorates very markedly between 1 and 5 years;
After 1yr, most families feel they are coping well
By year 5, most families feel they are not
It is the changes in personality and behaviour rather than physical disabilities that are blamed for this change

*Brooks et al 1986*