Amnestic disorders

The amnestic disorders are syndromes characterised by memory impairment (anterograde and/or retrograde amnesia), which are caused by a general medical condition or use substance, and where delirium and dementia have been excluded as causative of the amnesia. Amnestic disorders may be transient or chronic (< or > 1mth). Amnestic conditions usually involve some or all of the following neuroanatomical structures: frontal cortex; hippocampus and amygdala; dorsomedial thalamus; mamillary bodies; and the periaqueductal grey matter (PAG). In terms of neurochemistry, glutamate transmission at the NMDA receptor is often implicated in amnesia, mainly due to its role in memory storage in the limbic system (LTP). ***A number of amnestic disorders are recognised:***

Wernicke's encephalopathy :

An acute syndrome, with a classic tetrad of symptoms (ataxia, ophthalmoplegia, nystagmus, and acute confusional state), caused by thiamine depletion, usually related to alcohol abuse, and associated with pathological lesions in the mamillary bodies, PAG, thalamic nuclei, and the walls of the 3rd ventricle.

Korsakoff psychosis :

Amnesia and confabulation associated with atrophy of the mamillary bodies, usually following Wernicke's encephalopathy (rarer causes include: head injury; hypoxia brain injury; basal/temporal lobe encephalitis; and vascular insult).

Vascular disease:

Insults to the hippocampus (especially involving the posterior cerebral artery or basilar artery) may result in an amnestic disorder. Other regions implicated include: parietal-occipital junction; bilateral medio-dorsal thalamus; basal forebrain nuclei (e.g. aneurysm of the anterior communicating artery).

Head injury:

An open or closed head injury involving acceleration or deceleration forces may result in injury to the anterior temporal poles (as this structure collides with the temporal bone). Anterograde or post-traumatic amnesia (PTA) is prominent with retrograde amnesia relatively absent. Prognosis is related to length of PTA-better prognosis associated with PTA of less than 1wk.

HSV encephalitis:

Affects medial temporal lobes and results in deficits in STM storage.

Temporal lobe surgery:

Bilateral damage or surgery to the medial temporal lobes results in an inability to store new short-term memories (e.g. patient HM) (see box opposite).

Hypoxic brain damage:

Hypoxia following asphyxia from CO poisoning, near drowning, etc, may damage sensitive CA1 and CA3 neurons in the hippocampus. This results in problems with STM storage.

MS:

40% of patients have some amnesia due to plaques in the temporal lobes and diencephalon resulting in difficulty with recall.

Alcohol blackouts (palimpsest):

Significant alcohol intoxication may lead to amnesia for the period of intoxication. This usually only occurs in the context of chronic alcohol misuse.

ECT:

There may be a period of mild anterograde and/or retrograde amnesia for several hours following administration of ECT. More rarely there may be ongoing patchy memory loss for up to 6 - 9 mths.

Transient global amnesia (TGA):

This is a syndrome of amnesia lasting 6 - 24 hrs caused by transient ischaemia of the temporal lobes and/or diencephalon. It is more common over 50 yrs and may occur in the context of hypertension or migraine. Differential

diagnosis includes dissociative disorders and malingering, and diagnosis is often unclear.

Other causes of amnesia:

Substances (benzodiazepines, anticholinergics); space occupying lesions (e.g. tumours); hypoglycaemia.

Psychiatric aspects of head injury :

Head injuries are unfortunately common in a world characterised by mass use of motor vehicles and widely misused alcohol. The peak incidence of head injury is between the ages of 15 and 24 years and improved medical care has resulted in large numbers of individuals surviving with neuropsychiatric consequences.

There are important acute psychiatric effects of head injury:

Acute psychological effects of head injury

Most significant head injuries are closed and involve a period of loss of consciousness (which may extend from brief concussion to prolonged coma). On recovery of consciousness there are often memory deficits.

Amnesia is classified in terms of:

- **Post-traumatic amnesia (PTA) includes the period of injury and the period following injury (until normal memory resumes). Apparently normal behaviour often occurs during this period. PTA may end abruptly.**
- **Retrograde amnesia (RA) includes the period between the last clearly recalled memory prior to the injury and the injury itself. It is usually a dense amnesia that is brief, lasting seconds or minutes, and shrinks with time.**

Acute post-traumatic delirium (PTD)

A state that may follow severe head injury and occurs as the individual begins to regain consciousness. This is sometimes also called â€کpost-traumatic psychosis and is characterised

by prolonged and variable confusion, with or without behavioural symptoms, anxiety, affective lability, paranoia, delusional misinterpretation, and hallucinations.

Factors associated with increased psychiatric morbidity following head injury:

- **Increased duration of loss of consciousness**
- **Increased duration of PTA**
- **Increased duration of PTD**
- **Increased age, arteriosclerosis, and alcoholism**
- **Increased area of damage**
- **Increased neurological sequelae (focal deficits, epilepsy, etc.)**
- **Dominant or bilateral hemisphere involvement**

There are important chronic psychiatric effects of head injury: **A number of chronic syndromes are recognised following head injury:**

- **Cognitive impairment Especially after closed head injuries with PTA lasting >24 hours. There may be focal cognitive deficits such as amnesia, or diffuse problems including slowing, apathy, affective blunting, decreased concentration, executive difficulties, amnesia, and affective lability. Catastrophic reactions and emotional incontinence may occur. In its severest form the cognitive impairment may present as a dementia post-traumatic dementia. If symptoms are severe, it is particularly important to exclude NPH, SDH, or coexisting DAT.**
  Treatment: antipsychotics; stimulants.
- **Personality/behavioural changes Personality changes are most likely after head injury to the orbito-frontal lobe or anterior temporal lobe. Frontal lobe syndrome is characterised by disinhibition, impulsivity, irritability, and aggressive outbursts.**

Treatment may include b-blockers (e.g. atenolol), carbamazepine.

- **Psychoses A schizophrenia-like psychosis with prominent paranoia is associated with left temporal injury, while affective psychoses (esp. mania in 9% patients) are associated with right temporal or orbito-frontal injury. There is also an increased prevalence of schizophrenia post head injury (-2.5% develop the disorder).**
  Treatment Cautious use of antipsychotics (risk of seizure), anticonvulsants.
- **Neurotic disorders Depressive illness is most common but anxiety states (including PTSD) are common sequelae. Persistent depression and anxiety occur in roughly 1/4 of head injury survivors. Suicide risk is also higher post head injury.**
  Treatment: SSRIs; ECT.
- **Post-traumatic syndrome Also called post-concussional syndrome. This is a common phenomenon after head injury. The main symptoms are: headache; dizziness; insomnia; irritability; emotional lability; increased sensitivity to noise, light, etc; fatigue; poor concentration; anxiety; and depression. Although this syndrome was previously thought to be a purely psychological phenomenon (since in many cases the injury was minor), it is now recognised that it probably involves a complex interplay of both organic and non-organic factors.**

Factors influencing psychiatric disability and prognosis:

- **Mental constitution i.e. vulnerability due to genetics, temperament (premorbid personality: increased risk in histrionic, hypochondriacal and dependent personalities), IQ (â€کcerebral reserveâ€™), age.**

- **Emotional impact of injury i.e. extent of psychological trauma.**
- **Setting, circumstances, and repercussions of injury.**
- **Iatrogenic factors.**
- **Home and social environment (including gain issues).**
- **Compensation and litigation issues (including gain issues).**
- **Post-traumatic epilepsy (PTE) occurs in 5% closed and 30% open head injuries (usually during first year) and worsens prognosis.**
- **Size and location of brain damage: frontal, temporal, dominant side worse.**

Sequelae in children:

Less psychopathology after head injury due to increased brain plasticity. Recovery may continue for up to 5 years after injury (as opposed to 2 years in adults). Problems are generally behavioural in nature and include aggression, delinquency and ADHD-like syndromes.

Punch-drunk syndrome:

Boxers may develop diffuse injury to the cortex, basal ganglia, and cerebellum, giving rise to extra-pyramidal symptoms or a subcortical dementia. Pathology shows cerebral atrophy and neuro-fibrillary tangles.