

NEUROPSYCHIATRIC ASPECTS OF  
TRAUMATIC BRAIN INJURY

# OUTLINE

- ⊕ Introduction
- ⊕ History
- ⊕ Epidemiology
- ⊕ Mechanisms of TBI
- ⊕ Neuropsychiatric sequelae of traumatic brain injury
- ⊕ Acute behavioral consequences of traumatic brain injury
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- ⊕ Laboratory tests
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- ⊕ References



# INTRODUCTION

- ⊕ Traumatic brain injury TBI also known as intracranial injury occurs when an external force traumatically injures to brain
- ⊕ TBI is defined as an non degenerative non congenital insult to the brain from 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

# INTRODUCTION

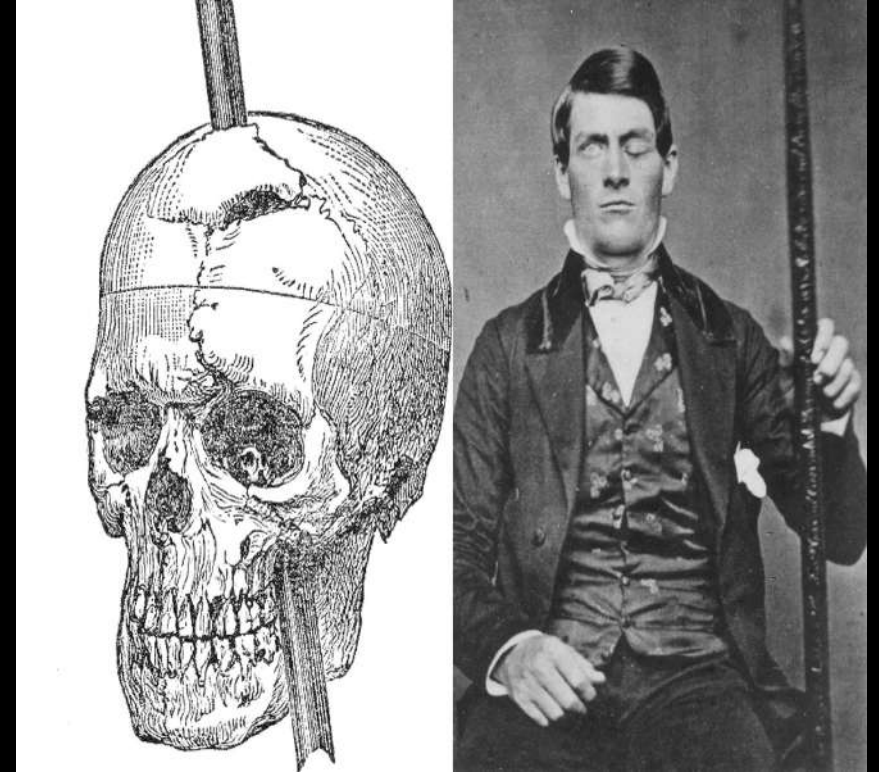
⊕ The disorders that are also seen in patients without brain injury cover the whole spectrum of psychiatric disorders including substance abuse mood anxiety psychotic and personality disorders

# HISTORY

- ⊕ The earliest physical evidence of traumatic brain injury due to assault occurred 1 million years ago
- ⊕ The earliest written evidence of brain injuries was found on the Edwin Smith Papyrus dated 5000 years ago which contained the first 27 head injury records
- ⊕ The Hippocratic Corpus included a treatise on head injury with thoughtful comments on skull fractures, delirium, seizures and coma

# HISTORY

- ✦ *Adolf Meyer* for eg identified a number of disorders that he referred to as the "traumatic insanities"
- ✦ Although he believed that these disorders were determined by a combination of psychological social historical and biological factors he suggested that there may be some unique associations between these disorders and specific lesion locations
- ✦ The most famous case of *frontal lobe injury* however was *Phineas Gage* who suffered a penetrating frontal brain injury after an explosion shot an iron bar through his skull
- ✦ After the injury he was described as childish capricious inconsiderate profane and having poor judgment



# HISTORY

⊕ Analysis of a large series of cases such as the Oxford Collection of Head Injury records suggests that biological variables such as the extent of brain damage, lesion location and the presence of posttraumatic epilepsy were important etiological factors in determining the type and duration of psychiatric syndromes.

# EPIDEMIOLOGY

- ⊕ In the US the annual incidence of closed head injuries admitted to a hospital can be conservatively estimated as 150 per 100 000 population
- ⊕ Incidence of penetrating head injury has been estimated to be 12 per 100 000 According to these rates there are approximately 500 000 new cases each year a significant proportion of which will result in long term disabilities
- ⊕ 80 of TBI patients have mild head injury
- ⊕ 10 have moderate head injury
- ⊕ 10 are categorized as severe

# EPIDEMIOLOGY

- ⊕ Most of these injuries occur among adolescents and young adults
- ⊕ There is also a significant gender difference
- ⊕ Males are two to three times more likely to suffer brain injury than females
- ⊕ Low socioeconomic status constitutes another independent risk factor for TBI
- ⊕ Single greatest risk factor for TBI however is alcohol drug abuse



# EPIDEMIOLOGY

- ⊕ A recent epidemiological study reported that close to one third of brain injury patients had an identifiable alcohol problem before trauma and more than 50% were intoxicated at the time of injury
- ⊕ Transport related cases i.e. motor vehicle accidents and pedestrians hit by vehicles are the most important cause of injury particularly in younger adults
- ⊕ Falls associated with older age are the second most prevalent cause of injury
- ⊕ Assaults especially penetrating injuries involving firearm use as well as sports and recreation related injuries are the next most common causes of TBI



# RISK FACTORS FOR NEUROPSYCHIATRIC DISORDERS

- ⊕ Increasing age
- ⊕ Arteriosclerosis
- ⊕ Alcoholism
- ⊕ Premorbid personality
- ⊕ Marital discord
- ⊕ Poor interpersonal relationships
- ⊕ Problems at work
- ⊕ Financial instability

Lishman WA *Physiogenesis and psychogenesis in the postconcussional syndrome* Br J Psychiatry 1988 153:460-469

# MECHANISMS OF TBI

⊕ Mechanical forces applied to the skull and transmitted to the brain

⊕ This may lead to focal and or diffuse brain damage

⊕ Focal lesions direct



⊕ brain laceration

⊕ contusion

⊕ intracerebral haemorrhage

⊕ subarachnoid or SDH

⊕ ischemic infarct

# MECHANISMS OF TBI

⊕ Diffuse brain injury also results from the differential motion of the brain within the skull causing a shearing and stretching of the axons

⊕ This can produce a wide spectrum of injuries ranging from brief physiological disruption to widespread axonal tearing called diffuse axonal injury *DAI*

Kwentus JA Hart RP Deck ET et al Psychiatric complications of closed head trauma *Psychosomatics* 1985 26:8 15

⊕ In addition to brain damage occurring at the time of the impact secondary damage from several processes may occur during the recovery period

⊕ Hypoxia

⊕ Anemia

⊕ Metabolic abnormalities

⊕ Hydrocephalus

⊕ Intracranial hypertension

⊕ Fat embolism

⊕ SAH

# NEUROPSYCHIATRIC SEQUELAE OF TRAUMATIC BRAIN INJURY

⊕ Cognitive deficits

⊕ Mood disorders

⊕ a Major depression

⊕ b Mania

⊕ Anxiety disorder

⊕ Psychosis

⊕ Apathy

⊕ Behavior or dyscontrol disorder

⊕ a Major variant

⊕ b Minor variant

⊕ Other

⊕ a Sleep disturbances

⊕ b Headache

# CLASSIFICATION OF HEAD INJURY

- ⊕ The most common classification is based on physical trauma *open vs closed head injury* depending on whether or not the skull has been breached
- ⊕ Another important classification system depends on the severity of initial impairment combining the initial Glasgow Coma Scale *GCS* the duration of *LOC* and the duration of posttraumatic amnesia
- ⊕ A *GCS* of 13-15 *LOC* of less than 30 minutes and or *PTA* of less than 1 hour is classified as mild *TBI*
- ⊕ People with moderate *TBI* have a *GCS* of 9-12 *LOC* of 1-24 hours and or *PTA* of 30 minutes to 24 hours
- ⊕ A *GCS* of 8 or less *LOC* of more than 24 hours and or *PTA* of more than 1 day is classified as severe *TBI*

# CLINICAL FEATURES

⊕ *Acute Behavioral Consequences of Traumatic Brain Injury*

⊕ *Chronic Behavioral Consequences*

ACUTE BEHAVIORAL CONSEQUENCES OF  
TRAUMATIC BRAIN INJURY



- ⊕ Acute complications eg brain swelling delayed hematoma or intracranial infection or prolonged postconcussion symptoms
- ⊕ CT and MRI have demonstrated the presence of structural brain lesions in some mild head injury patients who have not experienced clinical complications
- ⊕ Most common consequence of head injury is impairment of consciousness ranging from transient confusion to protracted coma
- ⊕ GCS is commonly used to grade the severity of traumatic brain injury
- ⊕ The scale gives a quantitative estimate of level of consciousness and neurological status based on patterns of eye opening as well as best verbal and motor responses

TBI is characterized by

⊕ Disorientation

⊕ Confusion

⊕ Impaired memory function

⊕ Apathetic withdrawal

⊕ Agitation or

⊕ Severe delirium may also be observed in these patients

- ⊕ There are multiple conditions that may contribute to the development of delirium in TBI patient
- ⊕ Structural brain damage cerebral edema brain hypoxia seizures electrolyte imbalance infections medications eg barbiturates opiates or steroids and drug or alcohol withdrawal
- ⊕ Old age coexistent severe medical disease polypharmacy basal ganglia and right hemisphere lesions have also been shown to be significant risk factors

# POSTTRAUMATIC AMNESIA

- ⊕ PTA occurs during the period when the patient who is usually emerging from coma is disoriented confused and has disrupted memory functioning
- ⊕ Deficits observed in declarative memory i.e. memory of recent events and times affecting both anterograde and retrograde processes
- ⊕ Duration of PTA has been widely used as a measure of TBI severity
- ⊕ Assessed using the Galveston Orientation and Amnesia Test GOAT which evaluates orientation to person place and time as well as awareness of the accident and its consequence

# AGITATION AND AGGRESSION

- ⊕ Among TBI complications agitation is a frequent behavioural problem
- ⊕ Agitation causes potential harm to patients and caregivers interferes with treatments leads to unnecessary chemical and physical restraints increases hospital length of stay delays rehabilitation
- ⊕ Pharmacological treatments are often considered for agitation management following TBI
- ⊕ Several types of agents have been proposed for the treatment of agitation
- ⊕ Some evidence that typical neuroleptics such as haloperidol might have a negative impact upon cognitive recovery

CHRONIC BEHAVIORAL  
CONSEQUENCES

# COGNITIVE DEFICITS

- ⊕ Cognitive deficits are very common among TBI patients
- ⊕ Common after head injury and are classified as delirium dementia amnesic disorder or mild cognitive deficit depending on the variety of symptoms and their time of onset and resolution
- ⊕ Include impairment of arousal attention concentration memory language and executive function
- ⊕ Cognitive outcome depends on a number of factors such as degree of diffuse axonal injury duration of LOC and PTA clinical evidence of brain stem dysfunction at the time of injury and presence and size of focal hemispheric injury

# MEMORY IMPAIRMENT

- ⊕ *Memory functions are also distinctively impaired in TBI patients*
- ⊕ *Memory deficits are the most frequent cognitive disturbances reported by patients and relatives in the chronic phase of TBI*
- ⊕ *Memory dysfunction is characterized by both anterograde and retrograde deficits*



# ATTENTION DEFICITS

- ⊕ Attention deficits are among the most frequent neuropsychological symptoms observed in TBI patients following resolution of PTA
- ⊕ It occurs with both **focal frontotemporal** damage and or diffuse brain injury
- ⊕ They often report problems with concentration difficulty in focusing easy distractibility

## Common language deficits include

- ⊕ Motor and sensory **prosodic** dysfunction
- ⊕ Word finding problems
- ⊕ Difficulty in expressing ideas
- ⊕ **Dysarthria** and **Anomia**
- ⊕ Inability to understand sarcasm or follow simple commands
- ⊕ **Expressive aphasias** are more common compared to receptive aphasias

# EXECUTIVE FUNCTIONS

- ⊕ A prominent defect in control or executive functions has been consistently described in patients surviving severe head injury
- ⊕ Executive functions include goal formation, planning, selection of adequate response patterns, and monitoring of ongoing behavior
- ⊕ The executive dysfunction observed in TBI patients is strongly associated with dysfunction of fronto-subcortical pathways

# DEMENTIA

- ⊕ Dementia is a syndrome defined in the DSM IV TR by impairment of memory and at least one other cognitive domain in the absence of an alteration of consciousness
- ⊕ Dementia due to head trauma is characterized by prominent memory and executive dysfunction with relatively preserved visuospatial and primary linguistic functions
- ⊕ In addition these patients may be severely apathetic and withdrawn and demonstrate markedly slow information processing
- ⊕ Physical examination may reveal the presence of extrapyramidal signs
- ⊕ Chronic subdural hematoma in the elderly may present as a progressive dementia

# DEMENTIA PUGILISTICA

- ⊕ *Dementia pugilistica is another related condition*
- ⊕ *Multiple traumatic brain injury associated with boxing occurs in approximately 20% of professional boxers*
- ⊕ *The diagnosis of this severe complication is dependent upon documenting progressive dementia associated with chronic and repeated brain trauma and unexplainable by an alternative pathophysiological process*
- ⊕ *Pathologically dementia pugilistica shares many characteristics with Alzheimer's disease*

# PERSONALITY CHANGES

- ⊕ TBI patients may experience significant personality changes
- ⊕ These patients have been described as irritable childish inconsiderate capricious anxious or aggressive
- ⊕ They lack foresight and misjudge the consequences of their actions
- ⊕ Disinhibition is a frequent and striking clinical feature that may lead to antisocial behavior
- ⊕ On the other hand they may become apathetic abulic and withdrawn
- ⊕ Sexual disinhibition

# POSTCONCUSSIONAL SYNDROME

⊕ Occurs following *head trauma* usually sufficiently severe to result in loss of consciousness and includes a number of disparate symptoms such as

⊕ *Headache dizziness fatigue irritability difficulty in concentrating and performing mental tasks impairment of memory insomnia and reduced tolerance to stress emotional excitement or alcohol*

⊕ *Symptoms may be accompanied by feelings of depression or anxiety resulting from some loss of self esteem and fear of permanent brain damage*

⊕ *Some patients become hypochondriacal embark on a search for diagnosis and cure and may adopt a permanent sick role*

# MAJOR DEPRESSION

- ⊕ Major depression occurs in approximately 25% of patients with TBI. Feelings of loss, demoralization and discouragement seen soon after injury are often followed by symptoms of persistent dysphoria.
- ⊕ Fatigue, irritability, suicidal thoughts, anhedonia, disinterest and insomnia are seen in a substantial number of patients 6-24 months or even longer after TBI.
- ⊕ Clinical and research studies have also shown that poor premorbid level of functioning and past history of psychiatric illness are major risk factors for depression.



# MAJOR DEPRESSION

- ⊕ Mechanism of depression following head injury is probably due to disruption of biogenic amine containing neurons as they pass through the basal ganglia or frontal subcortical white matter
- ⊕ Presence of left dorsolateral frontal and left basal ganglia lesions is associated with an increased probability of developing major depression
- ⊕ Treatment of depression secondary to TBI is very similar to the treatment of major depressive disorder
- ⊕ It includes antidepressants, psychostimulants and electroconvulsive therapy



# MAJOR DEPRESSION

- ⊕ Choice of medications must be influenced by their side effect profile Agents such as SSRIs are safe and well tolerated
- ⊕ Drugs with anticholinergic effects in general should be avoided
- ⊕ Psychostimulant and even the dopaminergics can be helpful in these cases as they have an antidepressant effect
- ⊕ ECT is a highly effective mode of treatment for BI patients refractory to antidepressants

# Mania

- ⊕ Mania after TBI is less common than depression but much more common than in the general population
- ⊕ It is seen in about 9% of patients
- ⊕ Changes in mood, sleep, and activation may manifest as irritability, euphoria, insomnia, agitation, aggression, impulsivity, and even violent behavior
- ⊕ Positive family history of affective disorder and subcortical atrophy prior to TBI are added risk factors
- ⊕ Mania is often seen in patients with right hemispheric limbic structure lesions

# MANIA

- ⊕ Treatment with anticonvulsants such as carbamazepine or valproate may be more effective than lithium which is not specific to the neuropathology of TBI and may worsen cognitive impairment
- ⊕ Other than this there is little empirical knowledge about the treatment of mania following TBI

# ANXIETY DISORDERS

- ⊕ Anxiety disorders are common in patients with TBI and range in frequency from 11 to 70
- ⊕ All variants of anxiety disorders are seen including GAD, panic disorder, phobic disorders, posttraumatic stress disorder, and obsessive compulsive disorder
- ⊕ TBI patients often experience generalized "free floating" anxiety associated with persistent worry, tension, and fearfulness
- ⊕ Increased activity of the aminergic system and decreased activity of the GABA inhibitory network is the proposed mechanism for the clinical manifestation of anxiety
- ⊕ Right hemispheric lesions are more often associated with anxiety disorder than left-sided lesions

# ANXIETY DISORDERS

- ⊕ Evidence suggests that antidepressants such as SSRIs opioid antagonists such as naltrexone and buspirone are promising in the treatment of anxiety disorders
- ⊕ Benzodiazepines and antipsychotics should be avoided because they cause memory impairment disinhibition and delayed neuronal recovery
- ⊕ Behavioral therapy and psychotherapy are as important as pharmacotherapy in the treatment of anxiety disorders

# SUICIDE

- ⊕ Suicide is considerably increased after head injury and accounted for 14% of all deaths in an 18 year follow up of those with war brain injuries
- ⊕ Change of character alcoholism and interpersonal difficulties are frequently present
- ⊕ An association with lesions in frontal and temporal lobes has been reported

Rerkum RV, Bolago I, Finlayson MA, Lerner S, Link PE. Psychiatric disorders after traumatic brain injury. *Brain Injury* 1996; 10: 319-27

# PSYCHOSES

- ⊕ Psychotic symptoms are not uncommon in TBI patients
- ⊕ A review of the literature by Davison and Bagley revealed that 07-98 of patients with TBI develop schizophrenia like psychosis
- ⊕ Most of these patients do not have a family history of schizophrenia
- ⊕ Other studies have shown that the incidence of head injury pre dating psychotic symptoms in a population of patients with schizophrenia is about 15
- ⊕ Psychotic symptoms following TBI often manifest as frank delusions hallucinations and illogical thinking

# PSYCHOSIS

- ⊕ agitation ideas of reference grimacing silly giggling expression of odd ideas regression and impulsive aggressiveness
- ⊕ Psychotic features may be acute or chronic transient or persistent and may or may not be associated with mood disturbances
- ⊕ Both right and left hemispheres have been implicated in the genesis of psychotic symptoms



# PSYCHOSES

- ⊕ For instance when there is a suggestion of left temporal involvement there may be benefit from the use of an anticonvulsant
- ⊕ Delusional type symptoms that seem more related to cognitive and behavioral impairments from frontal lobe dysfunction can benefit from dopaminergics
- ⊕ Neuroleptics if administered should be given in low doses as animal studies have shown impaired neuronal recovery

# POSTTRAUMATIC EPILEPSY

- ⊕ Early fits within the first week are relatively benign sensitive to prophylactic anticonvulsants and are only weak predictors of later epilepsy
- ⊕ Only about 5% of closed head injuries go on to develop late seizures compared with 30% after an open head injury
- ⊕ The majority of these late seizures start in the first and second year following injury
- ⊕ By the time 5 years have elapsed without seizures any subsequent seizure development may be unrelated to the head injury

# POSTTRAUMATIC EPILEPSY

- ⊕ Children are at increased risk of post traumatic epilepsy. The EEG is generally not a good predictor of post traumatic epilepsy.
- ⊕ Post traumatic epilepsy increases psychiatric morbidity particularly mood disorders, behavioural problems and psychotic illness and may increase the risk of late dementia.
- ⊕ Prophylactic anticonvulsants have no effect on reducing the incidence of late post traumatic epilepsy.
- ⊕ Carbamazepine rather than phenytoin is the drug of choice if an anticonvulsant is needed because it has less effect on cognition.

# APATHY

- ⊕ 10% of patients tend to have apathy without depression and 60% have some degree of apathy and depression following TBI
- ⊕ Apathy refers to a syndrome of *disinterest disengagement lack of motivation and absence of emotional responsivity*
- ⊕ Negative affect and cognitive deficits seen in patients with depression are not seen in patients with apathy
- ⊕ Apathy may be secondary to damage of the mesial frontal lobe
- ⊕ It often responds well to either psychostimulants dextroamphetamine amantadine or bromocriptine

Kant R Duffy JD Divovarnik A The prevalence of apathy following head injury *Brain Inj* 1988 12:87-92

Duffy JD Campbell JJ The regional prefrontal syndromes: a theoretical and clinical overview *J Neuropsychiatry Clin Neuroscience* 1994 6:379-387

# SEXUALITY

- ⊕ Limbic structures *nuclei hypothalamus* and various cortical areas which form part of the neuroanatomic and physiologic substrate of human sexual behaviour may be damaged in head injury resulting in impaired sexuality
- ⊕ Frontal lobe injuries with resultant *disinhibition* *lack of social judgment* *difficulty in modulating* and initiating sexual overtures can profoundly affect the sexual responses

# SLEEP

- ⊕ Disturbances after a head injury have received very little scientific attention despite the fact that several studies indicate that they may occur in 30 to 70 of patients
- ⊕ For individuals with head injury problems falling asleep or maintaining sleep can exacerbate other symptoms such as pain cognitive deficits fatigue or irritability
- ⊕ **Headache** is a common symptom and at times a persistent and disabling sequelae of head injury. It may occur alone or as part of a syndrome

# SUBSTANCE ABUSE

- ⊕ Major risk factors for the occurrence of TBI and also a major determinant of the clinical and psychosocial outcome of TBI patients
- ⊕ Mary R Hibbard and colleagues found that 40% of TBI patients met the DSM IV TR criteria for alcohol abuse or dependence before injury and 28% of the sample were given such diagnoses at the time of the study
- ⊕ A prospective study of 197 TBI patients showed that 42% of these patients had blood alcohol levels consistent with legal intoxication at the time of admission to the emergency department and that blood alcohol levels suggested a history of problem drinking among these patients



# SUBSTANCE ABUSE

⊕ Main findings of this study were that inmates with head injuries had higher levels of alcohol and marijuana use

⊕ Significantly higher frequency of depression anxiety and suicidal thinking as well as more difficulties in concentrating and controlling violent behavior



# POSTTRAUMATIC STRESS DISORDER

⊕ Mayou et al found that PTSD is "not associated with a neurotic predisposition" but is "strongly associated with horrific memories of the accident"

⊕ Women were predisposed to develop PTSD 6 of 10 women versus 2 of 14 men developed PTSD following head injury

⊕ PTSD occurred in 82 of mild head injury patients who had experienced acute stress disorder earlier 1 month post injury but in only 11 of those who did not suffer acute stress disorder

Ohry G, Rattok J, Solomon Z Post traumatic stress disorder in brain injury patients Brain Injury 1996 10:687-95

Bryant RA, Harvey AG Relationship between acute stress disorder and posttraumatic stress disorder following mild traumatic brain injury Am J Psychiat 1998 155:625-9

# LABORATORY TESTS

⊕ Serum markers

⊕ Neuronal eg neuron specific enolase creatine kinase BB cleaved Tau protein

⊕ Glial eg myelin basic protein & 100B proteins

⊕ Hormonal analysis

# NEUROIMAGING

- ⊕ CT and MRI are routinely used for the evaluation of TBI patients
- ⊕ CT is still the most efficient means of detecting surgically treatable hematomas and is the study of choice for evaluating patients with rapid changes in their neurological status
- ⊕ MRI more sensitive in detecting the more prevalent posttraumatic nonhemorrhagic lesions eg cortical contusions and deep white matter lesions and in identifying small subdural collections

# NEUROIMAGING

- ⊕ Diffusion tensor imaging DTI is a sensitive method to study white matter changes after TBI
- ⊕ It has also been shown that DTI reveals changes in the white matter that are correlated with the severity of TBI and with functional outcome at the time of hospital discharge
- ⊕ Overall suggest that DTI may be a powerful technique for in vivo detection of traumatic axonal injury

# NEUROIMAGING

- ⊕ *Magnetic resonance spectroscopy imaging MRS techniques acquire spectra simultaneously over a wide brain region enabling the generation of maps of metabolite distributions*
- ⊕ *Multislice short echo time TE  $H_1$  MRS allows for the "in vivo" evaluation of the metabolic profiles of large brain regions including the neocortex*
- ⊕ *A recent study used proton magnetic resonance spectroscopy  $H_1$  MRS to evaluate the presence of axonal injury at the splenium of the corpus callosum in normal control volunteers and in patients with TBI*

# NEUROIMAGING

⊕ Positron emission tomography PET and functional MRI fMRI might be used to delineate the neural circuits involved in mood and cognitive regulation and assess the effect of brain injury upon these neural circuits

# ELECTROPHYSIOLOGICAL STUDIES

- ⊕ EEG recordings are currently used in trauma intensive care units for monitoring procedures and for brain death diagnosis
- ⊕ EEG is also invaluable in the diagnosis of *status epilepticus* and is the primary diagnostic procedure to localize a *posttraumatic epileptic focus*
- ⊕ Video EEG monitoring and 24 hour ambulatory recordings may be useful in the differential diagnosis of patients presenting with unclear paroxysmal behavioral disturbances



# ELECTROPHYSIOLOGICAL STUDIES

- ⊕ **Quantitative EEG (Q EEG)** currently used as an adjunctive diagnostic technique in the evaluation of slow wave abnormalities associated with brain injuries and in the diagnosis of **posttraumatic temporal lobe epilepsy**
- ⊕ **Polysonnography** permits the diagnosis of atypical sleep disturbances that may occur in TBI patients
- ⊕ Include atypical night terrors, sleep apnea, nocturnal myoclonus, and restless leg syndrome



# COURSE AND PROGNOSIS

- ⊕ Course and prognosis of TBI patients involve the longitudinal analysis of neurological neuropsychological psychiatric and psychosocial variables
- ⊕ The Glasgow Outcome Scale GOS has been widely used as a measure of the long term outcome of TBI patients
- ⊕ It consists of five levels of outcome
  - 1 Death
  - 2 Persistent vegetative state
  - 3 Severe disability conscious but dependent in activities of daily living

# COURSE AND PROGNOSIS

4 Moderate disability disabled but living independently

5 Good recovery mild neuropsychiatric effects but able to resume an otherwise normal life

⊕ The long term outcome of TBI patients is primarily related to severity of brain injury type and location of intracranial lesion patients age and efficacy of acute medical and surgical treatment

⊕ Outcome is also influenced by concurrent factors that include socioeconomic status educational level previous psychiatric disorders eg history of alcohol and or drug abuse personality disorders and premorbid social functioning levels

# COURSE AND PROGNOSIS

- ⊕ The National Institutes of Health NIH Traumatic Coma Data Bank was initiated by the National Institute of Neurological Disorders and Stroke in order to characterize the natural history of traumatic head injury and to evaluate the determinants of recovery
- ⊕ Of 746 severe head injured patients studied in this cohort
- ⊕ 243 patients 325 died
- ⊕ 325 patients 42 were either severely disabled or in a vegetative state
- ⊕ 138 18 had moderate disability
- ⊕ and the remaining 50 7 had a good recovery as measured by the Glasgow Outcome Scale at the time of hospital discharge

# COURSE AND PROGNOSIS

- ⊕ Cognitive disturbances are among the most important factors in long term disability following severe TBI
- ⊕ The TBI group had significantly greater neuropsychological impairment than a control group
- ⊕ During the first year after the injury more than two thirds of relatives experienced moderate to severe degrees of burden as a consequence of the behavioral changes in their family member
- ⊕ Patient motivation and a history of alcohol drug abuse as well as awareness of cognitive and physical impairments have been shown to exert a significant effect on rehabilitation outcome

# TREATMENT OF NEUROPSYCHIATRIC COMPLICATIONS

## Organic Therapies

- ⊕ Patients with brain injury are more sensitive to the side effects of medications especially *psychotropic drugs*
- ⊕ *Start low go slow*
- ⊕ Typical antipsychotic agents such as *haloperidol* have been widely used to treat delirium and agitation in the acute phase of TBI

# TREATMENT

- ⊕ Olanzapine, quetiapine, ziprasidone, and aripiprazole would have similar therapeutic efficacies with more favorable side effect profiles.
- ⊕ However, there have not been systematic studies of the use of atypical antipsychotic agents to treat post-TBI agitation and delirium.
- ⊕ Case report suggested that in a patient with delirium following TBI, the typical mid-potency agent, *lorazepam*, appeared to have a better clinical effect than olanzapine.

# TREATMENT

- ⊕ The data regarding the treatment of mood disorders following TBI are also limited
- ⊕ An 8 week nonrandomized placebo run in trial of sertraline in 15 patients with mild TBI showed statistically significant improvement in psychological distress, anger and aggression as well as in the severity of postconcussive symptoms
- ⊕ Sertraline may also lead to a beneficial effect on cognitive functioning



# TREATMENT

- ⊕ No systematic studies of the treatment of secondary mania
- ⊕ Lithium carbamazepine and valproate therapies have been reported to be efficacious in individual cases
- ⊕ It has been proposed that both lithium and valproate have neuroprotective effects that would certainly constitute an important therapeutic advantage among brain injured populations
- ⊕ However data from the only controlled trial of valproate in TBI fail to identify a beneficial effect on cognitive and functional outcomes



# TREATMENT

- ⊕ The roles of other anticonvulsants such as lamotrigine or topiramate as mood stabilizers have not been tested in TBI populations
- ⊕ A recent case report however reported adequate control of problematic behaviors with lamotrigine treatment
- ⊕ Psychostimulants and other dopaminergically active agents eg methylphenidate dextroamphetamine pergolide and bromocriptine may modestly improve arousal and speed of information processing reduce distractibility and improve some aspects of executive function

# TREATMENT

- ✦ *ECT is not contraindicated in TBI patients and may be considered if other methods of treatment prove to be unsuccessful*
- ✦ *ECT should be administered with the lowest possible effective energy using brief pulse nondominant unilateral currents with an interval of 2 to 5 days between treatments and four to six treatments for a complete course*

# BEHAVIORAL PSYCHOTHERAPEUTIC TREATMENT

- ⊕ Behavioral deficits in self care habits eg feeding or personal hygiene interpersonal skills eg disinhibited behavior problem solving or response to environmental stress may be amenable to behavioral intervention
- ⊕ Behavioral rehabilitation programs shape behavior based on the principles of operant conditioning eg contingency contracts and token economies
- ⊕ Their goal is to increase the patient's repertoire of social and independent living skills generalizing their use from the rehabilitation environment to the more demanding conditions of community life

# BEHAVIORAL PSYCHOTHERAPEUTIC TREATMENT

- ⊕ **Psychological therapies** Personality changes are often resistant to treatment
- ⊕ Psychotherapy at superficial levels may help
- ⊕ Behavioral techniques like social skills training may help in reducing disruptive behaviors
- ⊕ Operant conditioning by rewarding self helping behaviour while belligerent and manipulative behaviour are ignored has proved its utility
- ⊕ Relaxation training is useful in anxiety disorders

# CAREGIVER SUPPORT AND EDUCATION

- ⊕ A caregiver may be the spouse, parent's family member, a friend, or even a professional care provider.
- ⊕ The frequency of psychiatric illness such as anxiety and major depression among care providers of TBI patients ranges from 16 to 51.
- ⊕ Hence, support of the family and of the caregivers is an essential component of treatment of the brain injured patient.
- ⊕ Each family is unique, and so are their problems, and the needs should be addressed individually.

# CAREGIVER SUPPORT AND EDUCATION

The general approach to caregiver support includes

- ⊕ Providing education
- ⊕ Instilling hope
- ⊕ Providing emotional support
- ⊕ If symptoms of anxiety or low mood are persistent recommending professional help
- ⊕ Encouraging the use of available resources such as local and national brain injury association centers
- ⊕ Discussing importance of respite care and the need to have personal time and
- ⊕ Always providing emergency contact numbers

# CONCLUSION

- ⊕ TBI is a complex heterogenous disease that can produce a variety of psychiatric disturbances ranging from subtle deficits in cognition mood and behavior to severe disturbances that cause impairment in social occupational and interpersonal functioning
- ⊕ Evidence to date suggests that depression is the most common comorbidity occurring not merely as a coincidence but seemingly intrinsic to the neuropathologic process
- ⊕ With improvement and sophistication in acute trauma care a number of individuals are able to survive the trauma but are left with several psychiatric sequelae



# CONCLUSION

- ⊕ Treatment should be interdisciplinary and multifaceted with the psychiatrist working in collaboration with the patient caregiver family other physicians and therapists
- ⊕ The goal of treatment should be to stabilize symptoms maximize potential minimize disability and increase productivity socially occupationally and interpersonally



# FUTURE DIRECTIONS

- ⊕ Most of these disorders have not been extensively studied in the TBI population and much research is still needed in this area
- ⊕ The disorders that are unique to brain injury also cover a wide range
- ⊕ Most of these disorders have not been extensively examined in patients with TBI
- ⊕ Because cognitive deficits are the most common consequence of TBI more studies need to be done to prove the effectiveness, tolerance and safety of medications

# REFERENCES

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THANK YOU