# Neurotrauma


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MISSING LINK

- Neurocritical care provides comprehensive neurological support for patients with life-threatening neurological and neurosurgical illnesses by integrating and balancing the management of both the brain and the other organs.

# Manifestations of psychiatric disorders timing????

- During the stay in intensive care unit (ICU) but also after transfer from ICU and several months after discharge from hospital.
- Part of psychiatric disorders is caused by organic or toxic causes (metabolic disturbances, electrolyte imbalance, withdrawal syndromes, infection, vascular disorders and **head trauma**).
- Nevertheless some authors estimate that they are due to the particular environment of ICU.
- Particularities of these units are: a high sound level (noise level average between 50 and 60 dBA), the absence of normal day-night cycle, a sleep deprivation, a sensory deprivation, the inability for intubated patients to talk, the pain provoked by some medical procedures, the possibility to witness other patients' death.

# The Role of Psychiatry in the Management of Acute Trauma Surgery Patients

John K. Findley, M.D.; Kathy B. Sanders, M.D.; and James E. Groves, M.D.

Half of all trauma patients in the general hospital setting are estimated to have a preexisting psychiatric disorder, and in patients with penetrating wounds, the figure approaches 90%.[1,2] This association is not surprising since psychiatric disorders produce behavioral patterns that often result in traumatic events. Depending on the hospital (and their various definitions of abuse, dependence, intoxication, etc.), studies have repeatedly shown that one quarter to one half of all acute trauma patients are intoxicated with alcohol or other drugs,[3–10] and more than a third meet criteria for at least 1 other psychiatric disorder.[11] Substance abuse or dependence disorders are often associated with mood disorders in this population. In one sample of acute traumatic burn patients, more than two thirds had preexisting psychopathology, of which more than half suffered from major depression and were more likely to have sustained burns in the setting of risk-taking behavior.[12]

***Conclusions:*** The ability to identify and treat coexisting psychopathology requires trauma surgeons to routinely incorporate a psychiatrist into their evaluation and treatment algorithm. Such a change in physician awareness and motivation hinges on a psychiatrist's visible presence (even if brief) and regular, active participation in the emergency department.

*(Primary Care Companion J Clin Psychiatry 2003;5:195–200)*

# Questionnaire Items: Trauma Surgeons' Attitudes Towards Psychiatric Consultation

1. Trauma patients are more difficult to manage than other patients in the ED.[b]
2. I find it more stressful to work with trauma patients than with other patients in the ED.
3. When the stressed family members of trauma patients arrive in the ED, I generally feel I do not have time to discuss the treatment plan with them.
4. If one of our psychiatrists addressed specific psychosocial concerns of trauma patients (eg, grief, death and dying), this would free up time for me to address evolving medical/surgical issues.
5. When trauma patients are "out of control," I consider consulting psychiatry.
6. When trauma patients have positive toxicology screens (for alcohol or other substances of abuse), I think about consulting the psychiatry service.
7. If I express anger to a trauma patient, I think about consulting the psychiatry service.
8. When imparting "bad news" to trauma patients, I think about consulting the psychiatry service.
9. If a trauma patient appears overwhelmed by his/her trauma and by its treatment, I think about consulting the psychiatry service.
0. Having a skilled psychiatrist work directly with the trauma service may be helpful in the management of trauma patients.

Review Article

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2005, Vol. 2, No. 1, pp. 13-21

# Neuropsychiatric Sequelae of Head Injury

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the injury. It is therefore essential to increase awareness of these sequelae so that psychological intervention is planned as early as possible, in hopes of improving function and limiting disability. In this paper we briefly review neuropsychiatric disorders following head injury.

# Prevalence Neuropsychiatric Sequelae of Head Injury (figures in percentages)

| Sequelae | Chatterjee Kishore [17] (n=37) | Keshavan (n=60) | Sabhesan et (n=134) | Chaudhury et et al (n=146) |
|---|---|---|---|---|
| Cognitive | 16.2 | 3 | 2.2 | 3.4 |
| Personality | 8 | - | 7.5 | 4.8 |
| Psychoses | 18.9 | 1 | 14.9 | 8.9 |
| Neuroses & others | - | 47 | 12.7 | 20.5 |
| Total | 43.2 | 51 | 29.1 | 37.7 |

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# Relative risk

- Highest relative risk was for major depression, with an RR of 7.5.
- Bipolar disorder also had a high RR of 5.3.
- Anxiety disorders was 2.0,
- Panic disorder with an RR of 5.8.
- Schizophrenia and substance abuse were close to or less than 1.0, suggesting either no, or a minor, increased risk for these disorders .

# Psychiatric disorders and traumatic brain injury

- TBI " silent epidemic"
- Rates of depression (13.9%) and panic disorder (9%)
- Risk factors : Young age low educational level, alcohol use, previous TBI, psychiatric disorder, low Glasgow Outcome scale
- Finland: 48.3% psychiatric disorder in post trauma patients
- Fann (northwest United nations):severe to moderate TBI 49% psychiatric disorders and 34% in milder ones.

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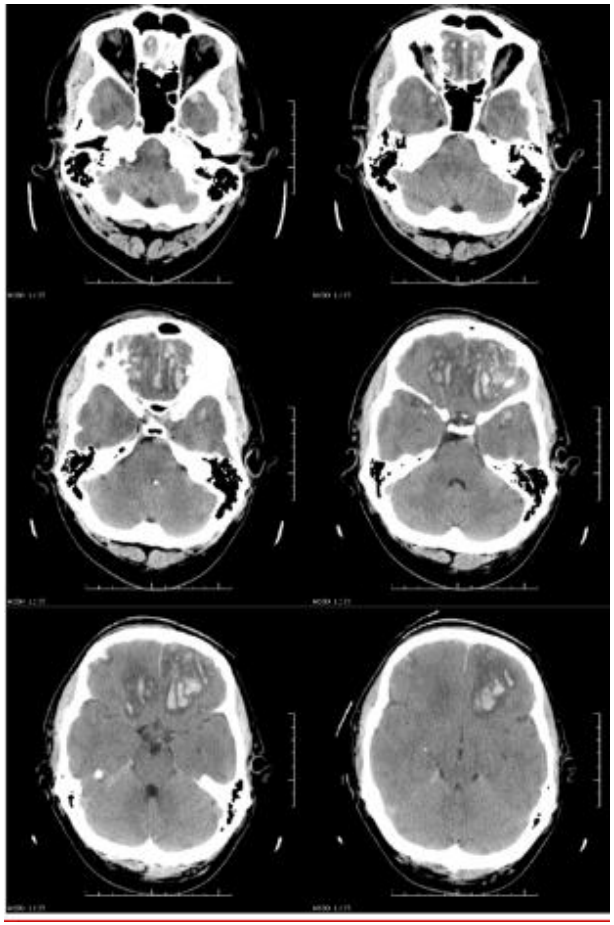
It has been proposed, at a theoretical level, that the rupture of neural circuits involving the prefrontal cortex, amygdala, hippocampus, basal ganglia, and thalamus may be related to the development of depression due TBI. During traumatism, diffuse axonal injury and damage located precisely in the frontal and anterior temporal regions are frequent, which may be an explanation for the high rate of mood disorders among these patients (Jorge and Starkstein 2005). The classic monoaminergic hypothesis of depression may also be useful for explaining depressive symptoms due TBI. Low levels of serotonin, for example, are classically associated to emotional changes, disinhibition and aggression, which are common symptoms of mood disorders after TBI. Disturbances in the neurotransmission systems, including serotonin, glutamate and dopamine were described in animal models and in TBI patients (Soblosky et al 1992; Jorge and Starkstein 2005).

Common series indicates that early identification and intervention on emotional and behavioral disturbances may also improve the life quality of these patients (Rapoport et al 2003).

# Neuropsychiatric Effects of Traumatic Brain Injury

- In somebody with a severe psychotic illness that develops 3 months after a traumatic brain injury with no loss of consciousness, one can be fairly confident that the illness is not a direct consequence of the effects of the brain injury on delusion formation.
- If posttraumatic amnesia (PTA) lasts less than 1 week, a reasonably good outcome is expected; if PTA lasts longer than 1 month, significant disability is likely.
- In patients with severe brain injury, a typical clinical picture consists of significant cognitive impairment—particularly in the domains of attention and concentration, psychomotor speed, memory, and executive function—as well as fatigue and problems with motivation.



CT brain scans within 2 days of a head injury show multiple bilateral hemorrhagic contusions in medial orbital frontal lobe and anterior temporal lobe. The 35-year-old had been found wandering with a skull fracture. He had been disoriented and agitated for several days. He was convinced that he had pressing appointments that he needed to get to. Despite no neurological signs, the patient was disorganized, showed poor insight, and was hospitalized. He improved markedly after about 2 months and subsequently returned to work.

# Specific Vulnerability

- Sites of to contusions are **the medial orbital frontal lobe and the anterior temporal lobes** .
- Areas where contusions rarely occur include the primary motor, somato-sensory, and visual cortex.
- areas of the brain concerned with social function and decision making are particularly vulnerable

# Importance of Neuropsychometric assessment

- Can be useful in defining the severity of cognitive impairment and any areas of particular impairment
- Accurate interpretation of a patient's post injury performance.
- Make sure that tests of executive function have been done
- Normal neuropsychometric test results do not rule out brain injury

# Neuropsychiatric assessment

- Severity of brain injury.
- the likely outcomes attributable to direct effects of brain injury can be determined,
- any mismatch between these and what is observed can be attributed to psychological reactions or independent events

# Severity of brain injury is measured by the following

- Glasgow Coma Scale (used soon after injury)
- Duration of loss of consciousness
- Duration of post traumatic amnesia (PTA), ie, the interval between the injury and the return of continuous day-to-day memories

# Risk factors neuropsychiatric disturbances after head injury

- Increasing age, arteriosclerosis, and alcoholism. These delay the reparative process within the central nervous system.
- Premorbid personality also a plays a significant role in the process of rehabilitation,
- "The late effects of head injury can only be properly understood in the light of a full psychiatric study of the individual patient... it is not only the kind of injury that matters, but the kind of head".
- Similarly, factors such as marital discord, poor interpersonal relationships, problems at work, or financial instability are important contributors to the neuropsychiatric disability.

# Impact of Trauma

- Brain damage occurring at the time of the impact,
- Secondary damage from several events like hypoxia, hemodynamically significant cerebral vasospasm, anemia, metabolic abnormalities, hydrocephalus, intracranial hypertension, fat embolism, and subarachnoid hemorrhage.
- Other delayed effects include release of excitatory amino acids, oxidative free-radical production, release of arachidonic acid metabolites, nitric oxide synthase-2, and disruption of neurotransmitters like monoamines and serotonin, dopamine, and acetylcholine
- Hypoxia may lead to free radical and excitotoxic neurotransmitter release
- Head injury may cause contusional injuries affecting brain regions involved in the mediation of mood, especially “along the temporal lobes and frontal cortex”

# Focal and/or diffuse brain damage

- Focal lesions often result from a direct blow to the head and include brain laceration, contusion, intracerebral hemorrhage, subarachnoid or subdural hemorrhage, and ischemic infarct
- Contusion occurs directly beneath or contralateral to the site of impact, commonly referred to as coup and contre-coup injury
- Most common in the orbital–frontal area and the temporal tips, where acceleration/deceleration forces cause the brain to impact on the bony protuberances of the skull.

- Contusions are areas of cerebral bruising particularly involving gray matter, whereby blood leaks into the extravascular space. The contusion results in cell death and local loss of tissue.

# Diffuse brain injury

- Results from the differential motion of the brain within the skull, causing a shearing and stretching of the axons.
- Wide spectrum of injuries, ranging from brief physiological disruption to widespread axonal tearing, called diffuse axonal injury (DAI).
- Usually observed in the corpus callosum and in the brainstem

- Diffuse axonal injury affects white matter anywhere throughout the cerebrum and brain stem. It may be followed by generalized atrophy with ventricular enlargement this may take a few weeks or months to develop.
- Diffuse axonal injury in the brain stem is usually responsible for the slurred speech and severe ataxia that are seen in some severely disabled patients after TBI.

- Few disorders ……..

# Cognitive and behavioral sequelae

- Difficult to know the extent to which brain injury is a factor in aggressive behavior.
- Relies on a good objective history of the behavior before and after the injury, along with an assessment of the likelihood of significant brain injury, which depends in part on the location of the injury.
- It is not easy to discern the origins of aggression (constitutional vs brain injury) from its phenomenology

# Cognitive impairment

- Patients with severe brain injury: a typical clinical picture consists, in the domains of attention and concentration, psychomotor speed, memory, and executive function, as well as fatigue and problems with motivation.
- The patient is likely to be self-centered, thoughtless, and crude in social relationships.
- He or she may show disinhibited behavior that is often sexual.
- Agitation and repetitive purposeless behaviors may also be present.
- Lability of mood is common; patients are often described as childish or moody.

# Cognitive outcome

- Depends on a number of factors: 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

# Mild traumatic brain injury/post concussional

- Glasgow Coma Scale score of more than 12; loss of consciousness, less than 30 minutes; PTA, less than 24 hours.
- Symptoms include headaches, fatigue, dizziness, depression, and difficulties with concentration and memory, which are often complicated by anxiety symptoms related to travel and posttraumatic stress disorder.
- Patients with long-standing symptoms, the extent and severity of the symptoms suggest that the illness is a form of somatization disorder

# Development of agitation : warning

- The patient may have thrown off some fat emboli from a fractured femur or
- Be in urinary retention.
- Agitation may also be the first sign of infection entering through a cerebrospinal fluid leak.

# Treatment

- Main task is to ensure the safety of the patient and others
- Family and caregivers may be a helpful resource if they can spend time with the patient.
- First stage of management is a review of the patient's medical and surgical recovery.
- Intoxication from medication may be to blame, and in some, agitation is a manifestation of craving because of substance abuse at the time of the injury.

# Depression

- Incidence: 15-33%
- Lack of energy (29%), irritability (28%), insomnia, lack of appetite, decreased involvement in activities
- It is associated with executive dysfunction, negative affect, and prominent anxiety symptoms. Feelings of loss, demoralization, and discouragement
- Depression occurs more frequently with left dorsolateral frontal and left basal ganglia lesions.
- The 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.
- Psychological impairments in excess of the severity of injury and poor cooperation with rehabilitation are strong indicators of a persistent depressive disorder

# Mania

- After head injury is less common than depression
- But much more common than in the general population supporting the contributory role of trauma in its etiology.
- It is seen in about 9% of patients.
- Often with lesions in right-sided limbic or limbic related structures.
- In post-traumatic mania irritable mood is more common than euphoric mood. 50% of post-traumatic manic patients have abnormal EEG.
- Changes in mood, sleep, and activation may manifest as irritability, euphoria, insomnia, agitation, aggression, impulsivity, and even violent behavior.

# Suicide

- 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.

# Anxiety disorders

- Eneralized anxiety disorder, panic disorder, phobic disorders, posttraumatic stress disorder, and obsessive–compulsive disorder are common after TBI and range in frequency from 11% – 70%.
- 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.

# Psychoses

- 4% to 8.9% of individuals who sustain head trauma
- Abnormalities due to trauma in temporal areas. Less consistent, but also common, are in the frontal lobes
- Interest to clinicians and neuroscientists for three reasons:
  - Usually a latency between the head injury and presentation of psychotic symptoms, thus the appearance of psychosis is often unexpected and puzzling;
  - There are diagnostic issues as some people who develop PSTHI(Psychosis secondary to head injury) have family histories of psychotic disorder, while many others do not; and
  - The disorder has conceptual relevance to understanding schizophrenia spectrum disorders

- Latencies of less than one year have been associated with diffuse injuries, paranoid symptoms and visual hallucinations.
- By contrast, patients with longer latencies before the onset of symptoms were found to have localized damage to the temporal lobe and presence of epilepsy

# Post traumatic Stress Disorder (PTSD):

- PTSD is “not associated with a neurotic predisposition” but is “strongly associated with horrific memories of the accident”.
- PTSD did not occur, in subjects who lost consciousness during the head injury or who were amnesic for the event.
- Women were predisposed to develop PTSD>than Men.
- 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.

# Personality change

- Two main variants of frontally-mediated changes in personality and emotions:
  - Pseudopsychopathic seen with lesions in the orbitobasal aspects of the frontal lobes manifesting with euphoria, impulsiveness and inadequate actions, which may be superimposed on a background of disinhibition, and
  - Pseudodepressed after lesions of the convexity regions of the frontal lobes manifesting with narrowing of interests and generalised emotional indifference, superimposed on a background of general inhibition and torpidity.
- Medial frontal syndrome is characterized by akinesia, sparse verbal output and incontinence.

# Treatment

- Rehabilitation: Cognitive Retraining, Psychological therapies, Family Therapy

- Pharmacological intervention: Anti-convulsants, Anti-depressants, Psychostimulants, Dopaminergic Agents Hypnotics,

# Overview of treatment

## Treatment guidelines for patients who have had a TBI

Obtain baseline assessments and wait to see whether problems remit spontaneously

Start low and go slow with drug dosing

Continue medication only if there is good evidence of benefit

Avoid polypharmacy

Choose drugs with:

- Low potential for adverse effects, particularly sedative/cognitive, anticholinergic, and extrapyramidal
- Low potential for lowering seizure threshold and for inducing drug interactions

Avoid short-acting anxiolytics, which are likely to reinforce unwanted behavior in patients with agitation

TBI, traumatic brain injury.

# Case # 1

- 28/ Male RTA with Polytrauma (Crush injuries to Rt Upper and Lower Limb, Pelvic Fracture etc.)
- Presents With Irrelevant Talk, Aggression, Uncooperativeness.
- Clinical Examination Reveals No Head Injury Co-related by Imaging.
- Conventional Sedatives(BZDs) not effective.

# Importance of History

- History Revealed Pt had 1 Month H/O Overtalkativeness, Overactivity, Irritability, Overexpenditure, Grandiose Behaviour, Poor Sleep. Accident itself caused due to extremely rash driving.
- During this period he was Consuming Alcohol and Cannabis everyday.
- Diagnosis Of Bipolar Disorder currently in Mania was made and started on Haloperidol.

# Stroke

- Can have varied Presentations
  - Confusion, Disorientation, Irrelevant Talk, Restlessness, Aggression etc. (Most often Related to the area of involvement)
  - Low Mood, Anxiety, Decreased Interactions, Negative Thought, Resisting interventions esp. Physiotherapy, Poor Sleep etc. (Post Stroke Depression)

# Common Comorbidities and Complications

- Aphasias (have to be diagnosed, as often very distressing for the pt and would require additional intervention like Speech Therapy).
- Urinary tract infections and Associated Delirium
- Electrolyte Imbalance
- Parkinsons Disease, Dementia.

# Case # 2

- 65/M with MCA Infarct (Correlated clinically and with imaging)
- Clinical examination reveals Global Aphasia.
- Presented to Psychiatrist with irritability, uncooperativeness, tear fullness, poor sleep and appetite, refusal to do Physiotherapy and Speech Therapy.
- Diagnosis of Post Stroke Depression was made and Addition of Sertraline gave Dramatic Response within 5-6 days.

# Case # 3

- 50/M with history of Alcohol Dependence since several years admitted for GI complaints suddenly became Irrelevant, started Hallucinating, Developed tremors, profuse sweating on Day 2 of Admission in ward.
- On examination all signs of Delirium Tremens were present (Tremors, Autonomic Hyperactivity, Visual Hallucinations etc.) Pt noticed to have multiple bruises all over his body.
- On enquiring regarding same relatives reported “He falls all the time when he is drunk”
- CT scan of brain revealed Chronic SDH.
- Important to be thorough. Had the scan not been done, and if pt was only treated for alcohol withdrawal the outcome would have been different !!

# Lets build up the missing link

Thank you……


# Ready for a relay race?

**It is always possible to do better…**

- Metabolic encephalopathy (ME) represents a syndrome of temporary or permanent disturbance of brain functions that occurs in different diseases and varies in clinical presentation.

- Manifested in a range from very mild mental disorders to deep coma and death.
- Most common causes of ME are: hypoxia, ischemia, systemic diseases and toxic agents.
- ME is the most frequent in elderly people who have previously been exhausted by chronic illnesses and prolonged stay in bed.
- ME is a very common complication in patients treated in intensive care units.
- Treatment and prognosis of the disease are varied and depend on aetiology, as well as on the type and severity of clinical presentation.
- Mortality of patients with septic encephalopathy ranges from 16-65%, while the one-year survival of patients with encephalopathy and liver cirrhosis is less than 50%.