# What is Traumatic Brain Injury (TBI)?

Traumatic brain injury (TBI), a form of acquired brain injury, occurs when a sudden trauma causes damage to the brain. TBI can result when the head suddenly and violently hits an object, or when an object pierces the skull and enters brain tissue. Symptoms of a TBI can be mild, moderate, or severe, depending on the extent of the damage to the brain. A person with a mild TBI may remain conscious or may experience a loss of consciousness for a few seconds or minutes. Other symptoms of mild TBI include headache, confusion, lightheadedness, dizziness, blurred vision or tired eyes, ringing in the ears, bad taste in the mouth, fatigue or lethargy, a change in sleep patterns, behavioral or mood changes, and trouble with memory, concentration, attention, or thinking. A person with a moderate or severe TBI may show these same symptoms, but may also have a headache that gets worse or does not go away, repeated vomiting or nausea, convulsions or seizures, an inability to awaken from sleep, dilation of one or both pupils of the eyes, slurred speech, weakness or numbness in the extremities, loss of coordination, and increased confusion, restlessness, or agitation. [1]

Traumatic brain injury (TBI) is a major public health problem, especially among male adolescents and young adults ages 15 to 24, and among elderly people of both sexes 75 years and older. Children aged 5 and younger are also at high risk for TBI.

Perhaps the most famous TBI patient in the history of medicine was Phineas Gage. In 1848, Gage was a 25-year-old railway construction foreman working on the Rutland and Burlington Railroad in Vermont. In the 19th century, little was understood about the brain and even less was known about how to treat injury to it. Most serious injuries to the brain resulted in death due to bleeding or infection. Gage was working with explosive powder and a packing rod, called a tamping iron, when a spark caused an explosion that propelled the 3-foot long, pointed rod through his head. It penetrated his skull at the top of his head, passed through his brain, and exited the skull by his temple. Amazingly, he survived the accident with the help of physician John Harlow who treated Gage for 73 days. Before the accident Gage was a quiet, mild-mannered man; after his injuries he became an obscene, obstinate, self-absorbed man. He continued to suffer personality and behavioral problems until his death in 1861.

Today, we understand a great deal more about the healthy brain and its response to trauma, although science still has much to learn about how to reverse damage resulting from head injuries.

TBI costs the country more than $56 billion a year, and more than 5 million Americans alive today have had a TBI resulting in a permanent need for help in performing daily activities. Survivors of TBI are often left with significant cognitive, behavioral, and communicative disabilities, and some patients develop long-term medical complications, such as epilepsy.

Other statistics dramatically tell the story of head injury in the United States. Each year:

· approximately 1.4 million people experience a TBI,

· approximately 50,000 people die from head injury,

· approximately 1 million head-injured people are treated in hospital emergency rooms, and

· approximately 230,000 people are hospitalized for TBI and survive. [2]

TBI, a form of acquired brain injury, occurs when a sudden trauma causes damage to the brain. The damage can be focal - confined to one area of the brain - or diffuse - involving more than one area of the brain. TBI can result from a closed head injury or a penetrating head injury. A closed injury occurs when the head suddenly and violently hits an object but the object does not break through the skull. A penetrating injury occurs when an object pierces the skull and enters brain tissue. [2]

Traumatic brain injury (TBI) is a complex injury with a broad spectrum of symptoms and disabilities. The impact on a person and his or her family can be devastating. The purpose of this section is to educate and empower caregivers and survivors of traumatic brain injuries and help understand TBI.

Traumaticbraininjury.com aims to ease the transition from shock and despair at the time of a brain injury to coping and problem solving. Bookmark this site for the latest medical breakthroughs and brain research, the highest quality treatment for brain damage, the symptoms of brain injuries and the nation's best traumatic brain injury rehabilitation centers and resource information. [3]

Traumatic brain injury, often referred to as TBI, is most often an acute event similar to other injuries. That is where the similarity between traumatic brain injury and other injuries ends. One moment the person is normal and the next moment life has abruptly changed.

In most other aspects, a traumatic brain injury is very different. Since our brain defines who we are, the consequences of a brain injury can affect all aspects of our lives, including our personality. A brain injury is different from a broken limb or punctured lung. An injury in these areas limit the use of a specific part of your body, but your personality and mental abilities remain unchanged. Most often, these body structures heal and regain their previous function.

Brain injuries do not heal like other injuries. Recovery is a functional recovery, based on mechanisms that remain uncertain. No two brain injuries are alike and the consequence of two similar injuries may be very different. Symptoms may appear right away or may not be present for days or weeks after the injury.

One of the consequences of brain injury is that the person often does not realize that a brain injury has occurred. [3]

**Traumatic brain injury** (TBI, also called **intracranial injury**) occurs when an external force traumatically injures the brain. TBI can be classified based on severity, mechanism (closed or penetrating head injury), or other features (e.g. occurring in a specific location or over a widespread area). Head injury usually refers to TBI, but is a broader category because it can involve damage to structures other than the brain, such as the scalp and skull.

TBI is a major cause of death and disability worldwide, especially in children and young adults. Causes include falls, vehicle accidents, and violence. Prevention measures include use of technology to protect those who are in accidents, such as seat belts and sports or motorcycle helmets, as well as efforts to reduce the number of accidents, such as safety education programs and enforcement of traffic laws.

Brain trauma can be caused by a direct impact or by acceleration alone. In addition to the damage caused at the moment of injury, brain trauma causes *secondary injury*, a variety of events that take place in the minutes and days following the injury. These processes, which include alterations in cerebral blood flow and the pressure within the skull, contribute substantially to the damage from the initial injury.

TBI can cause a host of physical, cognitive, emotional, and behavioral effects, and outcome can range from complete recovery to permanent disability or death. The 20th century has seen critical developments in diagnosis and treatment which have decreased death rates and improved outcome. These include imaging techniques such as computed tomography and magnetic resonance imaging. Depending on the injury, treatment required may be minimal or may include interventions such as medications and emergency surgery. Physical therapy, speech therapy, recreating therapy and occupational therapy may be employed for rehabilitation. [4]

## Epidemiology

TBI is a leading cause of death and disability around the globe and presents a major worldwide social, economic, and health problem. It is the number one cause of coma; it plays the leading role in disability due to trauma, and is the leading cause of brain damage in children and young adults. In Europe it is responsible for more years of disability than any other cause. It also plays a significant role in half of trauma deaths.

Findings on the frequency of each level of severity vary based on the definitions and methods used in studies. A World Health Organization study estimated that between 70 and 90% of head injuries that receive treatment are mild, and a US study found that moderate and severe injuries each account for 10% of TBIs, with the rest mild.

The incidence of TBI varies by age, gender, region and other factors. Findings of incidence and prevalence in epidemiological studies vary based on such factors as which grades of severity are included, whether deaths are included, whether the study is restricted to hospitalized people, and the study's location. The annual incidence of mild TBI is difficult to determine but may be 100–600 people per 100,000.

## Mortality

In the US, the mortality (death rate) rate is estimated to be 21% by 30 days after TBI. A study on Iraq War soldiers found that severe TBI carries a mortality of 30–50%. Deaths have declined due to improved treatments and systems for managing trauma in societies wealthy enough to provide modern emergency and neurosurgical services. The fraction of those who die after being hospitalized with TBI fell from almost half in the 1970s to about a quarter at the beginning of the 21st century. This decline in mortality has led to a concomitant increase in the number of people living with disabilities that result from TBI.

Biological, clinical, and demographic factors contribute to the likelihood that an injury will be fatal. In addition, outcome depends heavily on the cause of head injury. In the US, patients with fall-related TBIs have an 89% survival rate, while only 9% of patients with firearm-related TBIs survive. In the US, firearms are the most common cause of fatal TBI, followed by vehicle accidents and then falls. Of deaths from firearms, 75% are considered to be suicides.

The incidence of TBI is increasing globally, largely due to an increase in motor vehicle use in low- and middle-income countries. In developing countries, automobile use has increased faster than safety infrastructure could be introduced. In contrast, vehicle safety laws have decreased rates of TBI in high-income countries, which have seen decreases in traffic-related TBI since the 1970s. Each year in the United States about two million people suffer a TBI and about 500,000 are hospitalized. The yearly incidence of TBI is estimated at 180–250 per 100,000 people in the US, 281 per 100,000 in France, 361 per 100,000 in South Africa, 322 per 100,000 in Australia, and 430 per 100,000 in England. In the European Union the yearly aggregate incidence of TBI hospitalizations and fatalities is estimated at 235 per 100,000.

**Demographics**

TBI is present in 85% of traumatically injured children, either alone or with other injuries. The greatest number of TBIs occur in people aged 15–24. Because TBI is more common in young people, its costs to society are high due to the loss of productive years to death and disability. The age groups most at risk for TBI are children age five to nine and adults over age 80, and the highest rates of death and hospitalization due to TBI are in people over age 65. The incidence of fall-related TBI in First World countries is increasing as the population ages; thus the median age of people with head injuries has increased.

Regardless of age, TBI rates are higher in males. Men suffer twice as many TBIs as women do and have a fourfold risk of fatal head injury, and males account for two thirds of childhood and adolescent head trauma. However, when matched for severity of injury, women appear to fare more poorly than men.

Socioeconomic status also appears to affect TBI rates; people with lower levels of education and employment and lower socioeconomic status are at greater risk.

Head injury is present in ancient myths that may date back before recorded history. Skulls found in battleground graves with holes drilled over fracture lines suggest that trepanation may have been used to treat TBI in ancient times. Ancient Mesopotamians knew of head injury and some of its effects, including seizures, paralysis, and loss of sight, hearing or speech. The Edwin Smith Papyrus, written around 1650–1550 BC, describes various head injuries and symptoms and classifies them based on their presentation and tractability. Ancient Greek physicians including Hippocrates understood the brain to be the center of thought, probably due to their experience with head trauma.

Medieval and Renaissance surgeons continued the practice of trepanation for head injury. In the middle Ages, physicians further described head injury symptoms and the term *concussion* became more widespread. Concussion symptoms were first described systematically in the 16th century by Berengario da Carpi.

It was first suggested in the 18th century that intracranial pressure rather than skull damage was the cause of pathology after TBI. This hypothesis was confirmed around the end of the

19th century, and opening the skull to relieve pressure was then proposed as a treatment.

In the 19th century it was noted that TBI is related to the development of psychosis. At that time a debate arose around whether post-concussion syndrome was due to a disturbance of the brain tissue or psychological factors. The debate continues today.

Perhaps the first reported case of personality change after brain injury is that of Phineas Gage, who survived an accident in which a large iron rod was driven through his head, destroying one or both of his frontal lobes; numerous cases of personality change after brain injury have been reported since.
The 20th century saw the advancement of technologies that improved treatment and diagnosis such as the development of imaging tools including CT and MRI, and in the 21st century, diffusion tensor imaging (DTI). The introduction of intracranial pressure monitoring in the 1950s has been credited with beginning the "modern era" of head injury. Until the 20th century, the mortality rate of TBI was high and rehabilitation was uncommon; improvements in care made during World War I reduced the death rate and made rehabilitation possible. Facilities dedicated to TBI rehabilitation were probably first established during World War I. Explosives used in World War I caused many blast injuries; the large number of TBIs that resulted allowed researchers to learn about localization of brain functions. Blast-related injuries are now common problems in returning veterans from Iraq & Afghanistan; research shows the symptoms of such TBI's are largely the same as for TBI's involving a physical blow to the head. [4]

**Traumatic Brain Injury**

Traumatic brain injury (TBI) is a serious public health problem in the United States. Each year, traumatic brain injuries contribute to a substantial number of deaths and cases of permanent disability. Recent data shows that, on average, approximately 1.7 million people sustain a traumatic brain injury annually.

A TBI is caused by a bump, blow or jolt to the head or a penetrating head injury that disrupts the normal function of the brain. Not all blows or jolts to the head result in a TBI. The severity of a TBI may range from “mild,” i.e., a brief change in mental status or consciousness to “severe,” i.e., an extended period of unconsciousness or amnesia after the injury. The majority of TBIs that occur each year are concussions or other forms of mild TBI.

CDC’s research and programs work to prevent TBI and help people better recognize, respond, and recover if a TBI occurs. [5]

**What is Traumatic Brain Injury (TBI)**

Traumatic brain injury (TBI) is a complex injury with a broad spectrum of symptoms and disabilities. The impact on a person and his or her family can be devastating. The purpose of this site is to educate and empower caregivers and survivors of traumatic brain injuries. This site aims to ease the transition from shock and despair at the time of a brain injury to coping and problem solving. Bookmark this site for the latest medical breakthroughs and brain research, the highest quality treatment for brain damage, the symptoms of brain injuries and the nation's best traumatic brain injury rehabilitation centers and resource information. 6

# What are the Different Types of Traumatic Brain Injury?

### What Are the Different Types of TBI?

Concussion is the most minor and the most common type of TBI. Technically, a concussion is a short loss of consciousness in response to a head injury, but in common language the term has come to mean any minor injury to the head or brain.

Other injuries are more severe. As the first line of defense, the skull is particularly vulnerable to injury. Skull fractures occur when the bone of the skull cracks or breaks. A depressed skull fracture occurs when pieces of the broken skull press into the tissue of the brain. A penetrating skull fracture occurs when something pierces the skull, such as a bullet, leaving a distinct and localized injury to brain tissue.

Skull fractures can cause bruising of brain tissue called a contusion. A contusion is a distinct area of swollen brain tissue mixed with blood released from broken blood vessels. A contusion can also occur in response to shaking of the brain back and forth within the confines of the skull, an injury called countercoup. This injury often occurs in car accidents after high-speed stops and in shaken baby syndrome, a severe form of head injury that occurs when a baby is shaken forcibly enough to cause the brain to bounce against the skull. In addition, countercoup can cause diffuse axonal injury, also called shearing, which involves damage to individual nerve cells (neurons) and loss of connections among neurons. This can lead to a breakdown of overall communication among neurons in the brain.

Damage to a major blood vessel in the head can cause a hematoma, or heavy bleeding into or around the brain. Three types of hematomas can cause brain damage. An epidural hematoma involves bleeding into the area between the skull and the dura. With a subdural hematoma, bleeding is confined to the area between the dura and the arachnoid membrane. Bleeding within the brain itself is called intracerebral hematoma.

Another insult to the brain that can cause injury is anoxia. Anoxia is a condition in which there is an absence of oxygen supply to an organ's tissues, even if there is adequate blood flow to the tissue. Hypoxia refers to a decrease in oxygen supply rather than a complete absence of oxygen. Without oxygen, the cells of the brain die within several minutes. This type of injury is often seen in near drowning victims, in heart attack patients, or in people who suffer significant blood loss from other injuries that decrease blood flow to the brain. [2]

### General Trauma

Most TBI patients have injuries to other parts of the body in addition to the head and brain. Physicians call this polytrauma. These injuries require immediate and specialized care and can complicate treatment of and recovery from the TBI. Other medical complications that may accompany a TBI include pulmonary (lung) dysfunction; cardiovascular (heart) dysfunction from blunt chest trauma; gastrointestinal dysfunction; fluid and hormonal imbalances; and other isolated complications, such as fractures, nerve injuries, deep vein thrombosis, excessive blood

clotting, and infections.

Trauma victims often develop hyper metabolism or an increased metabolic rate, which leads to an increase in the amount of heat the body produces. The body redirects into heat the energy needed to keep organ systems functioning, causing muscle wasting and the starvation of other tissues. Complications related to pulmonary dysfunction can include neurogenic pulmonary edema (excess fluid in lung tissue), aspiration pneumonia (pneumonia caused by foreign matter in the lungs), and fat and blood clots in the blood vessels of the lungs.

Fluid and hormonal imbalances can complicate the treatment of hyper metabolism and high ICP. Hormonal problems can result from dysfunction of the pituitary, the thyroid, and other glands throughout the body. Two common hormonal complications of TBI are syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and hypothyroidism.

Blunt trauma to the chest can also cause cardiovascular problems, including damage to blood vessels and internal bleeding, and problems with heart rate and blood flow. Blunt trauma to the abdomen can cause damage to or dysfunction of the stomach, large or small intestines, and pancreas. A serious and common complication of TBI is erosive gastritis, or inflammation and degeneration of stomach tissue. This syndrome can cause bacterial growth in the stomach, increasing the risk of aspiration pneumonia. Standard care of TBI patients includes administration of prophylactic gastric acid inhibitors to prevent the buildup of stomach acids and bacteria. [2]

**Other Names For Mild TBI**

Concussion
Minor head trauma
Minor TBI
Minor brain injury
Minor head injury

**Mild Traumatic Brain Injury is:**

Most prevalent TBI
Often missed at time of initial injury
15% of people with mild TBI have symptoms that last one year or more.
Defined as the result of the forceful motion of the head or impact causing a brief change in mental status (confusion, disorientation or loss of memory) or loss of consciousness for less than 30 minutes.
Post injury symptoms are often referred to as post concussive syndrome. [3]

## Classification

Traumatic brain injury is defined as damage to the brain resulting from external mechanical force, such as rapid acceleration or deceleration, impact, blast waves, or penetration by a projectile. Brain function is temporarily or permanently impaired and structural damage may or

may not be detectable with current technology.

TBI is one of two subsets of acquired brain injury (brain damage that occurs after birth); the other subset is non-traumatic brain injury, which does not involve external mechanical force (examples include stroke and infection). All traumatic brain injuries are head injuries, but the latter term may also refer to injury to other parts of the head. However, the terms *head injury* and *brain injury* is often used interchangeably. Similarly, brain injuries fall under the classification of central nervous system injuries and neurotrauma. In neuropsychology research literature, the term "traumatic brain injury" generally is used to refer to non-penetrating traumatic brain injuries.

TBI is usually classified based on severity, anatomical features of the injury, and the mechanism (the causative forces). Mechanism-related classification divides TBI into closed and penetrating head injury. A closed (also called nonpenetrating, or blunt) injury occurs when the brain is not exposed. A penetrating, or open, head injury occurs when an object pierces the skull and breaches the dura mater, the outermost membrane surrounding the brain.

Head injuries can be classified into mild, moderate, and severe categories. The Glasgow Coma Scale (GCS), the most commonly used system for classifying TBI severity, grades a person's level of consciousness on a scale of 3–15 based on verbal, motor, and eye-opening reactions to stimuli. It is generally agreed that a TBI with a GCS of 13 or above is mild, 9–12 is moderate, and 8 or below is severe. Similar systems exist for young children. However, the GCS grading system has limited ability to predict outcomes. Because of this, other classification systems such as the one shown in the table are also used to help determine severity. A current model developed by the Department of Defense and Department of Veterans Affairs uses all three criteria of GCS after resuscitation, duration of post-traumatic amnesia (PTA), and loss of consciousness (LOC). It also has been proposed to use changes which are visible on neuroimaging, such as swelling, focal lesions, or diffuse injury as method of classification. Grading Scales also exist to classify the severity of mild TBI, commonly called concussion; these use duration of LOC, PTA, and other concussion symptoms.

**Pathological features**

CT scan Spread of the subdural hematoma (single arrow), midline shift (double arrow)
Systems also exist to classify TBI by its pathological features. Lesions can be extra-axial, (occurring within the skull but outside of the brain) or intra-axial (occurring within the brain tissue). Damage from TBI can be focal or diffuse, confined to specific areas or distributed in a more general manner, respectively. However it is common for both types of injury to exist in a given case.

Diffuse injury manifests with little apparent damage in neuroimaging studies, but lesions can be seen with microscopy techniques post-mortem and in the early 2000s, researchers discovered that diffusion tensor imaging (DTI), a way of processing MRI images that shows white matter tracts, was an effective tool for displaying the extent of diffuse axonal injury. Types of injuries considered diffuse include edema (swelling) and diffuse axonal injury, which is widespread damage to axons including white matter tracts and projections to the cortex. Types of injuries

considered diffuse include concussion and diffuse axonal injury, widespread damage to axons in areas including white matter and the cerebral hemispheres.

Focal injuries often produce symptoms related to the functions of the damaged area. Research shows that the most common areas to have focal lesions in non-penetrating traumatic brain injury are the orbit frontal cortex (the lower surface of the frontal lobes) and the anterior temporal lobes, areas that are involved in social behavior, emotion regulation, olfaction, and decision-making; hence the common social/emotional and judgment deficits following moderate-severe TBI. Symptoms such as hemi paresis or aphasia can also occur when less commonly affected areas such as motor or language areas are respectively damaged.

One type of focal injury, cerebral laceration, occurs when the tissue is cut or torn. Such tearing is common in orbit frontal cortex in particular, because of bony protrusions on the interior skull ridge above the eyes. In a similar injury, cerebral contusion (bruising of brain tissue), blood is mixed among tissue. In contrast, intracranial hemorrhage involves bleeding that is not mixed with tissue.

Hematomas, also focal lesions, are collections of blood in or around the brain that can result from hemorrhage. Intracranial hemorrhage, with bleeding in the brain tissue itself, is an intra-axial lesion. Extra-axial lesions include epidural hematoma, subdural hematoma, subarachnoid hemorrhage, and intraventricular hemorrhage. Epidural hematoma involves bleeding into the area between the skull and the dura mater, the outermost of the three membranes surrounding the brain. In subdural hematoma, bleeding occurs between the dura and the arachnoid mater. Subarachnoid hemorrhage involves bleeding into the space between the arachnoid membrane and the pia mater. Intraventricular hemorrhage occurs when there is bleeding in the ventricles.[4]

**Primary and secondary injury**

A large percentage of the people killed by brain trauma do not die right away but rather days to weeks after the event; rather than improving after being hospitalized, some 40% of TBI patients deteriorate Primary brain injury (the damage that occurs at the moment of trauma when tissues and blood vessels are stretched, compressed, and torn) is not adequate to explain this deterioration; rather, it is caused by secondary injury, a complex set of cellular processes and biochemical cascades that occur in the minutes to days following the trauma. These secondary processes can dramatically worsen the damage caused by primary injury and account for the greatest number of TBI deaths occurring in hospitals.

Secondary injury events include damage to the blood-brain barrier, release of factors that cause inflammation, free radical overload, excessive release of the neurotransmitter glutamate (excitotoxicity), influx of calcium and sodium ions into neurons, and dysfunction of mitochondria. Injured axons in the brain's white matter may separate from their cell bodies as a result of secondary injury, potentially killing those neurons. Other factors in secondary injury are changes in the blood flow to the brain; ischemia (insufficient blood flow); cerebral hypoxia (insufficient oxygen in the brain); cerebral edema (swelling of the brain); and raised intracranial pressure (the pressure within the skull). Intracranial pressure may rise due to swelling or a mass effect from a lesion, such as a hemorrhage. As a result, cerebral perfusion pressure (the pressure of blood flow in the brain) is reduced; ischemia results. When the pressure within the skull rises

too high, it can cause brain death or herniation, in which parts of the brain are squeezed by structures in the skull. A particularly weak part of the skull that is vulnerable to damage causing extradural hematoma is the pterion, deep to which lies the middle meningeal artery which is easily damaged in fractures of the pterion. Since the pterion is so weak this type of injury can easily occur and can be secondary due to trauma to other parts of the skull where the impact forces spreads to the pterion. [4]

**Acute stage**

Certain facilities are equipped to handle TBI better than others; initial measures include transporting patients to an appropriate treatment center. Both during transport and in hospital the primary concerns are ensuring proper oxygen supply, maintaining adequate cerebral blood flow, and controlling raised intracranial pressure (ICP), since high ICP deprives the brain of badly needed blood flow and can cause deadly brain herniation. Other methods to prevent damage include management of other injuries and prevention of seizures.

Neuroimaging is helpful but not flawless in detecting raised ICP.A more accurate way to measure ICP is to place a catheter into a ventricle of the brain, which has the added benefit of allowing cerebrospinal fluid to drain, releasing pressure in the skull. Treatment of raised ICP may be as simple as tilting the patient's bed and straightening the head to promote blood flow through the veins of the neck. Sedatives, analgesics and paralytic agents are often used. Hypertonic saline can improve ICP by reducing the amount of cerebral water (swelling); though it is used with caution to avoid electrolyte imbalances or heart failure. Mannitol, an osmostic diuretic, was also studied for this purpose, but such studies have been heavily questioned. Diuretics, drugs that increase urine output to reduce excessive fluid in the system, may be used to treat high intracranial pressures, but may cause hypovolemia (insufficient blood volume). Hyperventilation (larger and/or faster breaths) reduces carbon dioxide levels and causes blood vessels to constrict; this decreases blood flow to the brain and reduces ICP, but it potentially causes ischemia and is therefore only used in the short term.

Endotracheal intubation and mechanical ventilation may be used to ensure proper oxygen supply and provide a secure airway. Hypotension (low blood pressure), which has a devastating outcome in TBI, can be prevented by giving intravenous fluids to maintain a normal blood pressure. Failing to maintain blood pressure can result in inadequate blood flow to the brain. Blood pressure may be kept at an artificially high level under controlled conditions by infusion of norepinephrine or similar drugs; this helps maintain cerebral perfusion. Body temperature is carefully regulated because increased temperature raises the brain's metabolic needs, potentially depriving it of nutrients. Seizures are common. While they can be treated with benzodiazepines, these drugs are used carefully because they can depress breathing and lower blood pressure. TBI patients are more susceptible to side effects and may react adversely or be inordinately sensitive to some pharmacological agents. During treatment monitoring continues for signs of deterioration such as a decreasing level of consciousness.

Surgery can be performed on mass lesions or to eliminate objects that have penetrated the brain. Mass lesions such as contusions or hematomas causing a significant mass effect (shift of intracranial structures) are considered emergencies and are removed surgically. For intracranial hematomas, the collected blood may be removed using suction or forceps or it may be floated off with water. Surgeons look for hemorrhaging blood vessels and seek to control bleeding. In

penetrating brain injury, damaged tissue is surgically debrided, and craniotomy may be needed. Craniotomy, in which part of the skull is removed, may be needed to remove pieces of fractured skull or objects embedded in the brain. De-compressive craniotomy (DC) is performed routinely in the very short period following TBI during operations to treat hematomas; part of the skull is removed temporarily (primary DC). DC performed hours or days after TBI in order to control high intracranial pressures (secondary DC) has not been shown to improve outcome in some trials and may be associated with severe side effects.

**Chronic stage**

Physical therapy will commonly include muscle strength exercise.
Once medically stable, patients may be transferred to a sub acute rehabilitation unit of the medical center or to an independent rehabilitation hospital. Rehabilitation aims to improve independent function at home and in society and to help adapt to disabilities and has demonstrated its general effectiveness, when conducted by a team of health professionals who specialize in head trauma. As for any patient with neurological deficits, a multidisciplinary approach is key to optimizing outcome. Neurologists or Rehabilitation Physicians are likely to be the key medical staff involved, but depending on the patient, doctors of other medical specialties may also be helpful. Allied health professions such as physiotherapy, speech and language therapy and occupational therapy will be essential to assess function and design the rehabilitation activities for each patient. Treatment of neuropsychiatry symptoms such as emotional distress and clinical depression may involve mental health professionals such as therapists, psychologists, and psychiatrists, while neurophysiologists can help to evaluate and manage cognitive.

After discharge from the inpatient rehabilitation treatment unit, care may be given on an outpatient basis. Community-based rehabilitation will be required for a high proportion of patients, including vocational rehabilitation; this supportive employment matches job demands to the worker's abilities. People with TBI who cannot live independently or with family may require care in supported living facilities such as group homes. Respite care, including day centers and leisure facilities for the disabled, offers time off for caregivers and activities for people with TBI.

Pharmacological treatment can help to manage psychiatric or behavioral problems. Medication is also used to control; however the preventive use of anti-epileptics is not recommended. In those cases where the person is bedridden due to a reduction of consciousness, has to remain in a wheelchair because of mobility problems, or has any other problem heavily impacting self-caring capacities, care giving and nursing are critical. [4]

# What are the Treatment and Rehabilitation Options?

**Is there any treatment?**
Anyone with signs of moderate or severe TBI should receive medical attention as soon as possible. Because little can be done to reverse the initial brain damage caused by trauma, medical personnel try to stabilize an individual with TBI and focus on preventing further injury. Primary concerns include insuring proper oxygen supply to the brain and the rest of the body, maintaining adequate blood flow, and controlling blood pressure. Imaging tests help in determining the diagnosis and prognosis of a TBI patient. Patients with mild to moderate injuries

may receive skull and neck X-rays to check for bone fractures or spinal instability. For moderate to severe cases, the imaging test is a computed tomography (CT) scan. Moderately to severely injured patients receive rehabilitation that involves individually tailored treatment programs in the areas of physical therapy, occupational therapy, speech/language therapy, physiatrist (physical medicine), psychology/psychiatry, and social support. [1]

**What Medical Care Should a TBI Patient Receive?**

Medical care usually begins when paramedics or emergency medical technicians arrive on the scene of an accident or when a TBI patient arrives at the emergency department of a hospital. Because little can be done to reverse the initial brain damage caused by trauma, medical personnel try to stabilize the patient and focus on preventing further injury. Primary concerns include insuring proper oxygen supply to the brain and the rest of the body, maintaining adequate blood flow, and controlling blood pressure. Emergency medical personnel may have to open the patient's airway or perform other procedures to make sure the patient is breathing. They may also perform CPR to help the heart pump blood to the body, and they may treat other injuries to control or stop bleeding. Because many head-injured patients may also have spinal cord injuries, medical professionals take great care in moving and transporting the patient. Ideally, the patient is placed on a back-board and in a neck restraint. These devices immobilize the patient and prevent further injury to the head and spinal cord.

As soon as medical personnel have stabilized the head-injured patient, they assess the patient's condition by measuring vital signs and reflexes and by performing a neurological examination. They check the patient's temperature, blood pressure, pulse, breathing rate, and pupil size in response to light. They assess the patient's level of consciousness and neurological functioning using the Glasgow Coma Scale, a standardized, 15-point test that uses three measures - eye opening, best verbal response, and best motor response - to determine the severity of the patient's brain injury. [2]

**What Kinds of Rehabilitation Should a TBI Patient Receive?**

Rehabilitation is an important part of the recovery process for a TBI patient. During the acute stage, moderately to severely injured patients may receive treatment and care in an intensive care unit of a hospital. Once stable, the patient may be transferred to a sub acute unit of the medical center or to an independent rehabilitation hospital. At this point, patients follow many diverse paths toward recovery because there are a wide variety of options for rehabilitation.

Testing by a trained neurophysiologist can assess the individual's cognitive, language, behavioral, motor, and executive functions and provide information regarding the need for rehabilitative services.

In 1998, the NIH held a Consensus Development Conference on Rehabilitation of Persons with Traumatic Brain Injury. The Consensus Development Panel recommended that TBI patients receive an individualized rehabilitation program based upon the patient's strengths and capacities and that rehabilitation services should be modified over time to adapt to the patient's changing needs.* The panel also recommended that moderately to severely injured patients receive rehabilitation treatment that draws on the skills of many specialists. This involves individually

tailored treatment programs in the areas of physical therapy, occupational therapy, speech/language therapy, physiatry (physical medicine), psychology/psychiatry, and social support. Medical personnel who provide this care include rehabilitation specialists, such as rehabilitation nurses, psychologists, speech/language pathologists, physical and occupational therapists, physiatrists (physical medicine specialists), social workers, and a team coordinator or administrator.

The overall goal of rehabilitation after a TBI is to improve the patient's ability to function at home and in society. Therapists help the patient adapt to disabilities or change the patient's living space, called environmental modification, to make everyday activities easier.
Some patients may need medication for psychiatric and physical problems resulting from the TBI. Great care must be taken in prescribing medications because TBI patients are more susceptible to side effects and may react adversely to some pharmacological agents. It is important for the family to provide social support for the patient by being involved in the rehabilitation program. Family members may also benefit from psychotherapy.

It is important for TBI patients and their families to select the most appropriate setting for rehabilitation. There are several options, including home-based rehabilitation, hospital outpatient rehabilitation, inpatient rehabilitation centers, comprehensive day programs at rehabilitation centers, supportive living programs, independent living centers, club-house programs, school based programs for children, and others. The TBI patient, the family, and the rehabilitation team members should work together to find the best place for the patient to recover. [2]

## Treatments for TBI

There are many different kinds of treatments available for patients of Traumatic Brain Injury (TBI).

Initial Treatment stabilizes the individual immediately following a traumatic brain injury.

Rehabilitative Care Center Treatment helps restore the patient to daily life.

Acute treatment of a Traumatic Brain Injury is aimed at minimizing secondary injury and life support.

Surgical Treatment may be used to prevent secondary injury by helping to maintain blood flow and oxygen to the brain and minimize swelling and pressure.

## Initial Treatment

Initial treatment of a Traumatic Brain Injury (TBI) begins upon arrival to a hospital. At the hospital, a team of medical professionals, generally led by the trauma surgeon, will meet the patient. The trauma surgeon, acting as the leader, will direct the team. The trauma staff will initiate resuscitation procedures, monitor the body's vital functions, respond to potential life-threatening changes and coordinate care with other hospital personnel.

The patient may need surgery for injuries. In addition to the trauma surgeon, the surgical staff could include the neurosurgeon, a physician who performs brain and spinal cord surgery; an orthopedic surgeon, a physician who works with broken bones such as fractures of the arms and legs or the spinal column; or a general surgeon.

While the physicians are assessing the patient and the response to treatment, the trauma nurse is caring for the patient: providing resuscitation, stabilization and supportive care. The nurses have the responsibility to coordinate and provide communication within the hospital and with the family.

Once stabilized, the patient will be transferred to a specialized trauma care unit. Care will be provided by the critical care nursing staff. The nursing staff's responsibility is to assess, monitor and interpret vital physiologic or body functions, notify the physician of changes, repeat assessments at regular intervals and provide information for the family. The patient will be monitored for signs of infection and pain.

Other key staff also will play a role on the specialized trauma care unit. The respiratory therapist will help with the initial resuscitation efforts, will provide oxygen therapy, will configure the ventilator settings and will assure proper equipment functioning. In addition, the respiratory therapist will monitor the patient's breathing: looking at blood gas results and listening to the lungs.

In most trauma centers, a psychologist familiar with acute trauma will be part of the team. Using crisis intervention techniques, the psychologist will assist the patient and family in decision-making during crises. The psychologist will provide counseling and education about the injury, as well as assess the cognition of the patient.

A trauma social worker will also work with the family after the injury. Like the psychologist, the social worker will prepare the family emotionally and physically to face the ill or disabled patient. The trauma social worker will assist the family in making plans for the duration of recovery, especially if the recovery progresses slowly. The trauma social worker will encourage the family to consider role and responsibility changes while the patient is ill, including changes in finances and family support. The trauma social worker will also assist the family in discharge planning.

**Rehabilitative Center Treatment**

The families of traumatic brain injury (TBI) victims often have many questions when their loved one is transferred to a rehabilitative care center.

**What happens in rehabilitation?** Similar to the acute care facility, the TBI patient will be cared for by a team of professionals who specialize in the care of trauma victims.

Their goals are to:

- Stabilize the medical and rehabilitation issues related to brain injury and the other injuries.

Prevent secondary complications. Complications could include pressure sores, pneumonia and contractures.
Restore lost functional abilities. Functional changes could include limited ability to move, use the bathroom, talk, eat and think.
The staff will also provide adaptive devices or strategies to enhance functional independence.
The staff will begin to analyze with the family and the patient what changes might be required when the person goes home.

Each day, the patient will participate in therapy. Initially, the patient may require staff assistance for even the simplest activities: brushing teeth, getting out of bed and eating. The patient also may require staff for safety because there is a risk of falling, eloping (trying to get out of the hospital to go home) or getting hurt. The patient may be confused and forgetful.

**The Rehabilitation Team**

The Physiatrist is the team leader in the rehabilitation program. The physiatrist is a physician specializing in physical medicine and rehabilitation. Physiatrists treat a wide range of problems, including the changes after brain injury. The physiatrist will assess and prescribe the treatment and direct the team.

The Neurophysiologist is a key member of the rehabilitation team. The neurophysiologist will assess the patient's changes in thinking and behavior. Changes could include:

Poor memory
Poor attention and concentration
Poor decision-making
Impulsivity
Disorientation
Language and communication abilities
Inability to speak
Inability to understand when spoken to

Many patients are unaware of the changes in the brain and how those changes affect their daily lives. A patient may not understand what has happened and may be distraught by being away from home. Through education and counseling, the neurophysiologist can help assure the patient and the patient's family.

The Rehabilitation Nurse assists patients with brain injury and chronic illness in attaining maximum optimal health, and adapting to an altered lifestyle. The Rehabilitation Nurse provides care for the patient on the nursing unit. The focus of nursing care is on:

Health maintenance
Nutrition
Potential for aspiration
Impaired skin integrity
Bowel and bladder incontinence
Impaired physical mobility
Impaired or limited ability to take care of self
Ineffective airway

Sleep pattern disturbance
Chronic pain
Impaired cognition
Impaired verbal communication and comprehension
Sexual dysfunction

The Physical Therapist works with people with orthopedic problems, such as low back pain, knee injuries or pain reduction. With traumatic brain injury, the PT's job is to minimize or overcome paralyzing effects related to the brain injury. Physical therapists are experts in the examination and treatment of musculoskeletal and neuromuscular problems that affect the abilities to move and function in daily life.

Physical therapists help with transfers to and from the bed when a patient cannot walk alone. They train a person to begin to walk and move more normally. PTs will assess:

Balance
Posture
Strength
Need for a wheelchair, brace or cane
Quality of movement
Spontaneous movement
Coordination of movement
Increased sensation of sensory-motor activities
Pain management

The Occupational Therapist assesses functions and potential complications related to the movement of upper extremities, daily living skills, cognition, vision and perception. OTS helps determine, with the patient, the best ways to perform daily living skills including showering, dressing and personal hygiene. The OT will identify equipment for eating, dressing and bathing.

The OT also will look at skills to prepare the patient for a return to the home. These skills include:

Cooking
Grocery shopping
Banking
Budgeting
Readiness for returning to work by assessing prevocational and vocational skills

**Acute Treatment**

Acute treatment of a Traumatic Brain Injury (TBI) is aimed at minimizing secondary injury and life support.

Mechanical ventilation supports breathing and helps keep the pressure down in the head. A device may be placed surgically in the brain cavity to monitor and help control intracranial pressure.

Medications to sedate and put the individual in a drug-induced coma may be used to minimize

agitation and secondary injury. Seizure prevention medications may be given early in the course and later if the individual has seizures. Medications to control spasticity may be used as the patient recovers function. Behavioral issues also can be treated with medications. Medications for attention problems and aggressive behavior are often tried.

Medications may be used for:

Attention and concentration-amantadine and methylphenidate, bromocriptine and antidepressants.
Aggressive behavior-carbamamazapine and amitriptyline

**Surgical Treatment**

Surgical treatment is often used for patients of Traumatic Brain Injury (TBI).

In closed head injury, surgery does not correct the problem. A bolt or ICP (intracranial pressure) monitoring device may be placed in the skull to monitor pressure in the brain cavity. If there was bleeding in the skull cavity, this may be surgically removed or drained. Bleeding vessels or tissue may need to be repaired. In severe cases, if there is extensive swelling and damaged brain tissue, a portion may be surgically removed to make room for the living brain tissue.

An open head injury confronts doctors with the same issues as a closed head injury; however, in addition, skull fractures may need to be repaired and damaged tissue removed.

The overall goal of all surgical treatment is to prevent secondary injury by helping to maintain blood flow and oxygen to the brain and minimize swelling and pressure.

**Supportive Care Concerns**

The medical staff providing supportive care for the unconscious individual is highly trained and understands how to care for traumatic brain injury (TBI) patients.

TBI patients are monitored with equipment for breathing, heart rhythm, blood pressure, pulse and intracranial pressure.

Sometimes the unconscious individual cannot breathe without assistance. The airway is maintained and breathing occurs though special tubes that help maintain oxygen in the blood. It may be necessary to suction, as to remove thick secretions and keep the air tube clean.

The tube may be located in the mouth or in the neck. If it is in the neck, it is called a tracheotomy tube. Either tube will need to be cleaned daily. A pulse oximeter measures the amount of oxygen the patient is receiving through a device that resembles a finger splint.

After head trauma, seizures can occur. Dilantin is the usual medication administered through the IV to prevent seizures. A tetanus shot also may be given.

Fluid is administered through the IV for nutrition and liquid. The unconscious person cannot eat or drink safely. The need for nutritional support using parenteral (IV) or enteral solutions (a tube placed in the stomach) is determined by a registered dietician and the doctor.

A urinary catheter is put in the bladder for urine collection. The individual is not aware of the need to use the bathroom. The catheter attaches to a bag hanging from the side of the bed.

It is important to maintain the unconscious patient's blood pressure through IV fluid and medication. Ideally, the blood pressure range should be close to 90/70.

The patient is turned and positioned in bed to prevent bedsores because most unconscious people cannot move independently.

The unconscious person may have a compression device wrapped around the legs that resembles a plastic tub mat. This device prevents blood clots. Daily injections are also given to prevent blood clots. [3]

**Recovery**

Recovery from a Traumatic Brain Injury (TBI) varies based on the individual and the brain injury. Attempts at predicting the degree of TBI recovery remain crude. Recovery can be seen months, and even years, after the initial injury. Devastating and fatal injuries can be easier to ascertain than other injuries.

These are the indicators the medical team uses for prognosis:

Duration of Coma. The shorter the coma, the better the prognosis.
Post-traumatic amnesia. The shorter the amnesia, the better the prognosis.
Age. Patients over 60 or under age 2 have the worst prognosis, even if they suffer the same injury as someone not in those age groups.

Recovery of brain function is thought to occur by several mechanisms. Some common theories:

Diaschisis. Depressed areas of the brain that are not injured but linked to injured areas begin functioning again.
The function is taken over by a part of the brain that does not usually perform that task.
Redundancy in the function performed so another area of the brain takes over.
Behavioral substitution. The individual learns new strategies to compensate for deficits. [3]

**Treatment**

It is important to begin emergency treatment within the so-called "golden hour" following the injury. People with moderate to severe injuries are likely to receive treatment in an intensive care unit followed by a neurosurgical ward. Treatment depends on the recovery stage of the patient. In the acute stage the primary aim of the medical personnel is to stabilize the patient and focus on preventing further injury because little can be done to reverse the initial damage caused by trauma. Rehabilitation is the main treatment for the sub acute and chronic stages of recovery. International clinical guidelines have been proposed with the aim of guiding decisions in TBI

treatment, as defined by an authoritative examination of current evidence. [4]

# What are the Signs and Symptoms of Traumatic Brain Injury?

### What Are the Signs and Symptoms of TBI?

Symptoms of a TBI can be mild, moderate, or severe, depending on the extent of the damage to the brain. Some symptoms are evident immediately, while others do not surface until several days or weeks after the injury. A person with a mild TBI may remain conscious or may experience a loss of consciousness for a few seconds or minutes. The person may also feel dazed or not like himself for several days or weeks after the initial injury. Other symptoms of mild TBI include headache, confusion, lightheadedness, dizziness, blurred vision or tired eyes, ringing in the ears, bad taste in the mouth, fatigue or lethargy, a change in sleep patterns, behavioral or mood changes, and trouble with memory, concentration, attention, or thinking.

A person with a moderate or severe TBI may show these same symptoms, but may also have a headache that gets worse or does not go away, repeated vomiting or nausea, convulsions or seizures, inability to awaken from sleep, dilation of one or both pupils of the eyes, slurred speech, weakness or numbness in the extremities, loss of coordination, and/or increased confusion, restlessness, or agitation. Small children with moderate to severe TBI may show some of these signs as well as signs specific to young children, such as persistent crying, inability to be consoled, and/or refusal to nurse or eat. Anyone with signs of moderate or severe TBI should receive medical attention as soon as possible. [2]

### How Does a TBI Affect Consciousness?

A TBI can cause problems with arousal, consciousness, awareness, alertness, and responsiveness. Generally, there are five abnormal states of consciousness that can result from a TBI: stupor, coma, persistent vegetative state, locked-in syndrome, and brain death.

Stupor is a state in which the patient is unresponsive but can be aroused briefly by a strong stimulus, such as sharp pain. Coma is a state in which the patient is totally unconscious, unresponsive, unaware, and unarousable. Patients in a coma do not respond to external stimuli, such as pain or light, and do not have sleep-wake cycles. Coma results from widespread and diffuse trauma to the brain, including the cerebral hemispheres of the upper brain and the lower brain or brainstem. Coma generally is of short duration, lasting a few days to a few weeks. After this time, some patients gradually come out of the coma, some progress to a vegetative state, and others die.

Patients in a vegetative state are unconscious and unaware of their surroundings, but they continue to have a sleep-wake cycle and can have periods of alertness. Unlike coma, where the patient’s eyes are closed, patients in a vegetative state often open their eyes and may move, groan, or show reflex responses. A vegetative state can result from diffuse injury to the cerebral hemispheres of the brain without damage to the lower brain and brainstem. Anoxia, or lack of oxygen to the brain, which is a common complication of cardiac arrest, can also bring about a

vegetative state.

Many patients emerge from a vegetative state within a few weeks, but those who do not recover within 30 days are said to be in a persistent vegetative state (PVS). The chances of recovery depend on the extent of injury to the brain and the patient's age, with younger patients having a better chance of recovery than older patients. Generally adults have a 50 percent chance and children a 60 percent chance of recovering consciousness from a PVS within the first 6 months. After a year, the chances that a PVS patient will regain consciousness are very low and most patients who do recover consciousness experience significant disability. The longer a patient is in a PVS, the more severe the resulting disabilities will be. Rehabilitation can contribute to recovery, but many patients never progress to the point of being able to take care of themselves.

Locked-in syndrome is a condition, in which a patient is aware and awake, but cannot move or communicate due to complete paralysis of the body.

**Advances in imaging and other technologies have lead to devices that help differentiate among the variety of unconscious state.**

Unlike PVS, in which the upper portions of the brain are damaged and the lower portions are spared, locked-in syndrome is caused by damage to specific portions of the lower brain and brainstem with no damage to the upper brain. Most locked-in syndrome patients can communicate through movements and blinking of their eyes, which are not affected by the paralysis. Some patients may have the ability to move certain facial muscles as well. The majority of locked-in syndrome patients do not regain motor control, but several devices are available to help patients communicate.

With the development over the last half-century of assistive devices that can artificially maintain blood flow and breathing, the term brain death has come into use. Brain death is the lack of measurable brain function due to diffuse damage to the cerebral hemispheres and the brainstem, with loss of any integrated activity among distinct areas of the brain. Brain death is irreversible. Removal of assistive devices will result in immediate cardiac arrest and cessation of breathing.

Advances in imaging and other technologies have led to devices that help differentiate among the variety of unconscious states. For example, an imaging test that shows activity in the brainstem but little or no activity in the upper brain would lead a physician to a diagnosis of vegetative state and exclude diagnoses of brain death and locked-in syndrome. On the other hand, an imaging test that shows activity in the upper brain with little activity in the brainstem would confirm a diagnosis of locked-in syndrome, while invalidating a diagnosis of brain death or vegetative state. The use of CT and MRI is standard in TBI treatment, but other imaging and diagnostic techniques that may be used to confirm a particular diagnosis include cerebral angiography, electroencephalography (EEG), transcranial Doppler ultrasound, and single photon emission computed tomography (SPECT). [2]

**What are the Effects of TBI?**

Most people are unaware of the scope of TBI or its overwhelming nature. TBI is a common injury and may be missed initially when the medical team is focused on saving the individual's life. Before medical knowledge and technology advanced to control breathing with respirators

and decrease intracranial pressure, which is the pressure in the fluid surrounding the brain, the death rate from traumatic brain injuries was very high. Although the medical technology has advanced significantly, the effects of TBI are significant.

TBI is classified into two categories: mild and severe.

A brain injury can be classified as mild if loss of consciousness and/or confusion and disorientation is shorter than 30 minutes. While MRI and CAT scans are often normal, the individual has cognitive problems such as headache, difficulty thinking, memory problems, attention deficits, mood swings and frustration. These injuries are commonly overlooked. Even though this type of TBI is called "mild", the effect on the family and the injured person can be devastating.

Severe brain injury is associated with loss of consciousness for more than 30 minutes and memory loss after the injury or penetrating skull injury longer than 24 hours. The deficits range from impairment of higher level cognitive functions to comatose states. Survivors may have limited function of arms or legs, abnormal speech or language, loss of thinking ability or emotional problems. The range of injuries and degree of recovery is very variable and varies on an individual basis.

The effects of TBI can be profound. Individuals with severe injuries can be left in long-term unresponsive states. For many people with severe TBI, long-term rehabilitation is often necessary to maximize function and independence. Even with mild TBI, the consequences to a person's life can be dramatic. Change in brain function can have a dramatic impact on family, job, social and community interaction.

The number of people with Traumatic Brain Injury (TBI) is difficult to assess accurately but is much larger than most people would expect. According to the CDC (United States Centers for Disease Control and Prevention), there are approximately 1.5 million people in the U.S. who suffer from a traumatic brain injury each year. 50,000 people die from TBI each year and 85,000 people suffer long term disabilities. In the U.S., more than 5.3 million people live with disabilities caused by TBI. Patients admitted to a hospital for TBI are included in this count, while those treated in an emergency room or doctor's office are not counted. [3]

**Mild TBI Symptoms**

A traumatic brain injury (TBI) can be classified as mild if loss of consciousness and/or confusion and disorientation is shorter than 30 minutes. While MRI and CAT scans are often normal, the individual has cognitive problems such as headache, difficulty thinking, memory problems, attention deficits, mood swings and frustration. These injuries are commonly overlooked. Even though this type of TBI is called "mild", the effect on the family and the injured person can be devastating.

**Common Symptoms of Mild TBI**

Fatigue

Headaches
Visual disturbances
Memory loss
Poor attention/concentration
Sleep disturbances
Dizziness/loss of balance
Irritability-emotional disturbances
Feelings of depression
Seizures

**Other Symptoms Associated with Mild TBI**

Nausea
Loss of smell
Sensitivity to light and sounds
Mood changes
Getting lost or confused
Slowness in thinking

These symptoms may not be present or noticed at the time of injury. They may be delayed days or weeks before they appear. The symptoms are often subtle and are often missed by the injured person, family and doctors.

The person looks normal and often moves normal in spite of not feeling or thinking normal. This makes the diagnosis easy to miss. Family and friends often notice changes in behavior before the injured person realizes there is a problem. Frustration at work or when performing household tasks may bring the person to seek medical care.

**Severe TBI Symptoms**

Brain injuries can range in scope from mild to severe. Traumatic brain injuries (TBI) result in permanent neurobiological damage that can produce lifelong deficits to varying degrees. Moderate to severe brain injuries typically refer to injuries that have the following characteristics:

Moderate brain injury is defined as a brain injury resulting in a loss of consciousness from 20 minutes to 6 hours and a Glasgow Coma Scale of 9 to 12
Severe brain injury is defined as a brain injury resulting in a loss of consciousness of greater than 6 hours and a Glasgow Coma Scale of 3 to 8
The impact of a moderate to severe brain injury depends on the following:

Severity of initial injury
Rate/completeness of physiological recovery
Functions affected

Meaning of dysfunction to the individual
Resources available to aid recovery
Areas of function not affected by TBI

The impact of a moderate to severe brain injury can include:

**Cognitive deficits including difficulties with:**

Attention
Concentration
Distractibility
Memory
Speed of Processing
Confusion
Perseveration
Impulsiveness
Language Processing
"Executive functions"

**Speech and Language**

not understanding the spoken word (receptive aphasia)
difficulty speaking and being understood (expressive aphasia)
slurred speech
speaking very fast or very slow
problems reading
problems writing

**Sensory**

difficulties with interpretation of touch, temperature, movement, limb position and fine discrimination

**Perceptual**

the integration or patterning of sensory impressions into psychologically meaningful data

**Vision**

partial or total loss of vision
weakness of eye muscles and double vision (diplopia)
blurred vision
problems judging distance
involuntary eye movements (nystagmus)
intolerance of light (photophobia)

**Hearing**

decrease or loss of hearing
ringing in the ears (tinnitus)
increased sensitivity to sounds

**Smell**

loss or diminished sense of smell (anosmia)

**Taste**

loss or diminished sense of taste

**Seizures**

the convulsions associated with epilepsy that can be several types and can involve disruption in consciousness, sensory perception, or motor movements

**Physical Changes**

Physical paralysis/spasticity
Chronic pain
Control of bowel and bladder
Sleep disorders
Loss of stamina
Appetite changes
Regulation of body temperature
Menstrual difficulties

**Social-Emotional**

Dependent behaviors
Emotional ability
Lack of motivation
Irritability
Aggression
Depression
Disinhibition
Denial/lack of awareness [3]

## Symptoms of Traumatic Brain Injury

The broad spectrum of Traumatic Brain Injury (TBI) symptoms and disabilities contribute to the complexity of any TBI. The purpose of this section is to educate and empower caregivers and survivors of traumatic brain injuries and help understand the symptoms and the symptom-grading systems of TBI.

Bookmark this site for the symptoms of brain injuries, the latest medical breakthroughs and brain research, the highest quality treatment for brain damage and the nation's best traumatic brain injury rehabilitation centers and resource information. [3]

## Signs and symptoms

Unequal pupil size is a sign of a serious brain injury.
Symptoms are dependent on the type of TBI (diffuse or focal) and the part of the brain that is affected. Unconsciousness tends to last longer for people with injuries on the left side of the brain than for those with injuries on the right. Symptoms are also dependent on the injury's

severity. With mild TBI, the patient may remain conscious or may lose consciousness for a few seconds or minutes. Other symptoms of mild TBI include headache, vomiting, nausea, lack of motor coordination, dizziness, difficulty balancing, lightheadedness, blurred vision or tired eyes, ringing in the ears, bad taste in the mouth, fatigue or lethargy, and changes in sleep patterns. Cognitive and emotional symptoms include behavioral or mood changes, confusion, and trouble with memory, concentration, attention, or thinking. Mild TBI symptoms may also be present in moderate and severe injuries.

A person with a moderate or severe TBI may have a headache that does not go away, repeated vomiting or nausea, convulsions, an inability to awaken, dilation of one or both pupils, slurred speech, aphasia (word-finding difficulties), dysarthria (muscle weakness that causes disordered speech), weakness or numbness in the limbs, loss of coordination, confusion, restlessness, or agitation. Common long-term symptoms of moderate to severe TBI are changes in appropriate social behavior, deficits in social judgment, and cognitive changes, especially problems with sustained attention, processing speed, and executive functioning. Cognitive and social deficits have long-term consequences for the daily lives of people with moderate to severe TBI, but can be improved with appropriate rehabilitation.

When the pressure within the skull (intracranial pressure, abbreviated ICP) rises too high, it can be deadly. Signs of increased ICP include decreasing level of consciousness, paralysis or weakness on one side of the body, and a blown pupil, one that fails to constrict in response to light or is slow to do so. Cushing's triad, a slow heart rate with high blood pressure and respiratory depression is a classic manifestation of significantly raised ICP. Anisocoria, unequal pupil size, is another sign of serious TBI. Abnormal posturing, a characteristic positioning of the limbs caused by severe diffuse injury or high ICP, is an ominous sign.

Small children with moderate to severe TBI may have some of these symptoms but have difficulty communicating them. Other signs seen in young children include persistent crying, inability to be consoled, listlessness, refusal to nurse or eat, and irritability. [4]

# What are the Causes and Risk Factors? /How Can Traumatic Brain Injury be prevented?

### What Are the Causes of and Risk Factors for TBI?

Half of all TBIs are due to transportation accidents involving automobiles, motorcycles, bicycles, and pedestrians. These accidents are the major cause of TBI in people under age 75. For those 75 and older, falls cause the majority of TBIs. Approximately 20 percent of TBIs are due to violence, such as firearm assaults and child abuse, and about 3 percent are due to sports injuries. Fully half of TBI incidents involve alcohol use.

The cause of the TBI plays a role in determining the patient's outcome. For example, approximately 91 percent of firearms TBIs (two-thirds of which may be suicidal in intent) result in death, while only 11 percent of TBIs from falls result in death. [2]

### How Can TBI be prevented?

Unlike most neurological disorders, head injuries can be prevented. The Centers for Disease Control and Prevention (CDC) have issued the following safety tips* for reducing the risk of suffering a TBI.

Wear a seatbelt every time you drive or ride in a car.
Buckle your child into a child safety seat, booster seat, or seatbelt (depending on the child's age) every time the child rides in a car.
Wear a helmet and make sure your children wear helmets when
riding a bike or motorcycle;
playing a contact sport such as football or ice hockey;
using in-line skates or riding a skateboard;
batting and running bases in baseball or softball;
riding a horse;
Skiing or snowboarding.
Keep firearms and bullets stored in a locked cabinet when not in use.
Avoid falls by
using a step-stool with a grab bar to reach objects on high shelves;
installing handrails on stairways;
installing window guards to keep young children from falling out of open windows;
Using safety gates at the top and bottom of stairs when young children are around.
Make sure the surface on your child's playground is made of shock-absorbing material (e.g., hardwood mulch, sand).

The causes of TBI are diverse. The top three causes are: car accident, firearms and falls. Firearm injuries are often fatal: 9 out of 10 people die from their injuries. Young adults and the elderly are the age groups at highest risk for TBI. Along with a traumatic brain injury, persons are also susceptible to spinal cord injuries which are another type of traumatic injury that can result out of vehicle crashes, firearms and falls. Prevention of TBI is the best approach since there is no cure.

**Mechanisms of Injury:**

These mechanisms are the highest causes of brain injury: Open head Injury, Closed Head Injury, Deceleration Injuries, Chemical/Toxic, Hypoxia, Tumors, Infections and Stroke.

**1. Open Head Injury**
Results from bullet wounds, etc.
Largely focal damage
Penetration of the skull
Effects can be just as serious as closed brain injury

**2. Closed Head Injury**
Resulting from a slip and fall, motor vehicle crashes, etc.
Focal damage and diffuse damage to axons
Effects tend to be broad (diffuse)
No penetration to the skull

**3. Deceleration Injuries (Diffuse Axonal Injury)**

The skull is hard and inflexible while the brain is soft with the consistency of gelatin. The brain is encased inside the skull. During the movement of the skull through space (acceleration) and the rapid discontinuation of this action when the skull meets a stationary object (deceleration) causes the brain to move inside the skull. The brain moves at a different rate than the skull because it is soft. Different parts of the brain move at different speeds because of their relative lightness or heaviness. The differential movement of the skull and the brain when the head is struck results in direct brain injury, due to diffuse axonal shearing, contusion and brain swelling.

*Diffuse axonal shearing:* when the brain is slammed back and forth inside the skull it is alternately compressed and stretched because of the gelatinous consistency. The long, fragile axons of the neurons (single nerve cells in the brain and spinal cord) are also compressed and stretched. If the impact is strong enough, axons can be stretched until they are torn. This is called axonal shearing. When this happens, the neuron dies. After a severe brain injury, there is massive axonal shearing and neuron death.

**4. Chemical / Toxic**

Also known as metabolic disorders
This occurs when harmful chemicals damage the neurons
Chemicals and toxins can include insecticides, solvents, carbon monoxide poisoning, lead poisoning, etc.

**5. Hypoxia (Lack of Oxygen)**

If the blood flow is depleted of oxygen, then irreversible brain injury can occur from anoxia (no oxygen) or hypoxia (reduced oxygen)
It may take only a few minutes for this to occur
This condition may be caused by heart attacks, respiratory failure, drops in blood pressure and a low oxygen environment
This type of brain injury can result in severe cognitive and memory deficits

**6. Tumors**

Tumors caused by cancer can grow on or over the brain
Tumors can cause brain injury by invading the spaces of the brain and causing direct damage
Damage can also result from pressure effects around an enlarged tumor
Surgical procedures to remove the tumor may also contribute to brain injury

**7. Infections**

The brain and surrounding membranes are very prone to infections if the special blood-brain protective system is breached
Viruses and bacteria can cause serious and life-threatening diseases of the brain (encephalitis) and meninges (meningitis)

**8. Stroke**

If blood flow is blocked through a cerebral vascular accident (stroke), cell death in the area

deprived of blood will result

If there is bleeding in or over the brain (hemorrhage or hematoma) because of a tear in an artery or vein, loss of blood flow and injury to the brain tissue by the blood will also result in brain damage. [3]

**TBI Prevention**

Because Traumatic Brain Injury (TBI) cannot be cured, steps must be taken to prevent an injury from occurring. Advice for the prevention of TBI is often common sense.

**TBI Prevention Methods Include:**

- Always wear a seat belt in a motor vehicle
- Use an appropriate child safety seat or a booster
- Never drive under the influence of alcohol or drugs
- Always wear a helmet when on a bicycle, motorcycle, scooter, snowmobile and other open unrestrained vehicles
- Wear a helmet when participating in contact sports
- Wear a helmet when horseback riding
- Wear a helmet while skiing, snowboarding, skating and skateboarding

**Fall Prevention Methods:**

- Use the rails on stairways
- Provide adequate lighting, especially on stairs for people with poor vision or who have difficulty walking
- Place bars on windows to prevent children from falling
- Sit on safe stools
- Do not place obstacles in walking pathways

**Gun Safety:**

- Keep guns locked in a cabinet
- Store guns unloaded
- Store ammunition apart from guns [3]

**Causes**

The most common causes of TBI include violence, transportation accidents, construction, and sports. In the US, falls account for 28% of TBI, motor vehicle (MV) accidents for 20%, being struck by an object for 19%, violence for 11%, and non-MV bicycle accidents for 3%. Bicycles and motor bikes are major causes, with the latter increasing in frequency in developing countries. The estimates that between 1.6 and 3.8 million traumatic brain injuries each year are a result of sports and recreation activities in the US. In children aged two to four, falls are the most common cause of TBI, while in older children bicycle and auto accidents compete with falls for this position. TBI is the third most common injury to result from child abuse. Abuse causes 19% of cases of pediatric brain trauma, and the death rate is higher among these cases. Domestic violence is another cause of TBI, as are work-related and industrial accidents. Firearms and blast

injuries from explosions are other causes of TBI, which is the leading cause of death and disability in war zones.

## Mechanism

### Physical forces

Ricochet of the brain within the skull may account for the coup-countercoup phenomenon.
The type, direction, intensity, and duration of forces all contribute to the characteristics and severity TBI. Forces that may contribute to TBI include angular, rotational, shear, and translational forces.

Even in the absence of an impact, significant acceleration or deceleration of the head can cause TBI; however in most cases a combination of impact and acceleration is probably to blame. Forces involving the head striking or being struck by something, termed *contact* or *impact loading*, are the cause of most focal injuries, and movement of the brain within the skull, termed *noncontact* or *inertial loading*, usually causes diffuse injuries. The violent shaking of an infant that causes shaken baby syndrome commonly manifests as diffuse injury. In impact loading, the force sends shock waves through the skull and brain, resulting in tissue damage. Shock waves caused by penetrating injuries can also destroy tissue along the path of a projectile, compounding the damage caused by the missile itself.

Damage may occur directly under the site of impact, or it may occur on the side opposite the impact (coup and contrecoup injury, respectively). When a moving object impacts the stationary head, coup injuries are typical,

While countercoup injuries are usually produced when the moving head strikes a stationary object. [4]

## Prevention

Protective sports equipment such as helmets can protect athletes from head injury.
Since a major cause of TBI are vehicle accidents, their prevention or the amelioration of their consequences can both reduce the incidence and gravity of TBI. In accidents, damage can be reduced by use of seat belts, child safety seats and motorcycle helmets, and presence of roll bars and airbags. Education programs exist to lower the number of crashes. In addition, changes to public policy and safety laws can be made; these include speed limits, seat belt and helmet laws, and road engineering practices.

Changes to common practices in sports have also been discussed. An increase in use of helmets could reduce the incidence of TBI. Due to the possibility that repeatedly "heading" a ball practicing soccer could cause cumulative brain injury, the idea of introducing protective headgear for players has been proposed. Improved equipment design can enhance safety; softer baseballs reduce head injury risk. Rules against dangerous types of contact, such as "spear tackling" in American football, when one player tackles another head first, may also reduce head injury rates.

Falls can be avoided by installing grab bars in bathrooms and handrails on stairways; removing

tripping hazards such as throw rugs; or installing window guards and safety gates at the top and bottom of stairs around young children. Playgrounds with shock-absorbing surfaces such as mulch or sand also prevent head injuries. Child abuse prevention is another tactic; programs exist to prevent shaken baby syndrome by educating about the dangers of shaking children. Gun safety, including keeping guns unloaded and locked, is another preventative measure. Studies on the effect of laws that aim to control access to guns in the United States have been insufficient to determine their effectiveness preventing number of deaths or injuries. [4]

# What are the Glasgow Coma Scale and the Ranchos Los Amigos Scale?

**Glasgow Coma Scale**

The eye opening part of the Glasgow Coma Scale has four scores:

4 indicate that the patient can open his eyes spontaneously.
3 is given if the patient can open his eyes on verbal command.
2 indicates that the patient opens his eyes only in response to painful stimuli.
1 is given if the patient does not open his eyes in response to any stimulus.

The best verbal response part of the test has five scores:

5 is given if the patient is oriented and can speak coherently.
4 indicates that the patient is disoriented but can speak coherently.
3 means the patient uses inappropriate words or incoherent language.
2 is given if the patient makes incomprehensible sounds.
1 indicates that the patient gives no verbal response at all.

The best motor response test has six scores:

6 means the patient can move his arms and legs in response to verbal commands.
A score between 5 and 2 is given if the patient shows movement in response to a variety of stimuli, including pain.
1 indicates that the patient shows no movement in response to stimuli.

The results of the three tests are added up to determine the patient's overall condition. A total score of 3 to 8 indicates a severe head injury, 9 to 12 indicates a moderate head injury, and 13 to 15 indicates a mild head injury. [2]

**Glasgow Coma Scale**

There are a few different systems that medical practioners use to diagnose the symptoms of Traumatic Brain Injury. This section discusses the Glasgow Coma Scale.

The Glasgow Coma Scale is based on a 15 point scale for estimating and categorizing the outcomes of brain injury on the basis of overall social capability or dependence on others.

The test measures the motor response, verbal response and eye opening response with these values:

**I. Motor Response** 6 - Obeys commands fully 5 - Localizes to noxious stimuli 4 - Withdraws from noxious stimuli 3 - Abnormal flexion, i.e. decorticate posturing 2 - Extensor response, i.e. decerebrate posturing 1 - No response

**II. Verbal Response** 5 - Alert and Oriented 4 - Confused, yet coherent, speech 3 - Inappropriate words and jumbled phrases consisting of words 2 - Incomprehensible sounds 1 - No sounds

**III. Eye Opening** 4 - Spontaneous eye opening 3 - Eyes open to speech 2 - Eyes open to pain 1 - No eye opening

The final score is determined by adding the values of I+II+III.

This number helps medical practioners categorize the four possible levels for survival, with a lower number indicating a more severe injury and a poorer prognosis:

**Mild (13-15)**:

More in-depth discussion on the Mild TBI Symptoms Section.

**Moderate Disability (9-12):**

Loss of consciousness greater than 30 minutes
Physical or cognitive impairments which may or may resolve
Benefit from Rehabilitation

**Severe Disability (3-8):**

Coma: unconscious state. No meaningful response, no voluntary activities

**Vegetative State (Less Than 3):**

Sleep wake cycles
Arousal, but no interaction with environment
No localized response to pain

**Persistent Vegetative State:**

Vegetative state lasting longer than one month

**Brain Death:**

No brain function
Specific criteria needed for making this diagnosis

**Ranchos Los Amigos Scale**

There are a few different systems that medical practitioners use to diagnose the symptoms of

Traumatic Brain Injury. This section discusses the Ranchos Los Amigos Scale.

The Ranchos Los Amigos Scale measures the levels of awareness, cognition, behavior and interaction with the environment.

**Ranchos Los Amigos Scale:** Level I: No Response Level II: Generalized Response Level III: Localized Response Level IV: Confused-agitated Level V: Confused-inappropriate Level VI: Confused-appropriate Level VII: Automatic-appropriate Level VIII: Purposeful-appropriate 5

# Diagnosis and Prognosis

Imaging tests help in determining the diagnosis and prognosis of a TBI patient. Patients with mild to moderate injuries may receive skull and neck X-rays to check for bone fractures or spinal instability. The patient should remain immobilized in a neck and back restraint until medical personnel are certain that there is no risk of spinal cord injury. For moderate to severe cases, the gold standard imaging test is a computed tomography (CT) scan. The CT scan creates a series of cross-sectional X-ray images of the head and brain and can show bone fractures as well as the presence of hemorrhage, hematomas, contusions, brain tissue swelling, and tumors. Magnetic resonance imaging (MRI) may be used after the initial assessment and treatment of the TBI patient. MRI uses magnetic fields to detect subtle changes in brain tissue content and can show more detail than X-rays or CT. Unfortunately, MRI is not ideal for routine emergency imaging of TBI patients because it is time-consuming and is not available in all hospitals.

Approximately half of severely head-injured patients will need surgery to remove or repair hematomas or contusions. Patients may also need surgery to treat injuries in other parts of the body. These patients usually go to the intensive care unit after surgery.

Sometimes when the brain is injured swelling occurs and fluids accumulate within the brain space. It is normal for bodily injuries to cause swelling and disruptions in fluid balance. But when an injury occurs inside the skull-encased brain, there is no place for swollen tissues to expand and no adjoining tissues to absorb excess fluid. This increased pressure is called intracranial pressure (ICP).

Medical personnel measure patients. ICP using a probe or catheter. The instrument is inserted through the skull to the subarachnoid level and is connected to a monitor that registers the patient's ICP. If a patient has high ICP, he or she may undergo a ventriculostomy, a procedure that drains cerebrospinal fluid (CSF) from the brain to bring the pressure down. Drugs that can be used to decrease ICP include Mannitol or barbiturates, although the safety and effectiveness of the latter are unknown. [2]

**Diagnosis**

With moderate or severe traumatic brain injury (TBI), the diagnosis is often self evident. In the presence of other life threatening injuries, which are often the case with motor vehicle

accidents, closed head injury can be missed. The focus is on lifesaving measures.

The patient may be on a ventilator (breathing machine) and sedated and the evaluation for brain injury will be limited until the patient is allowed to emerge from medications and mechanical ventilation. Mild traumatic brain injury may not be diagnosed until the individual begins to have problems in what were once easy tasks or social situations.

Injury to specific areas of the brain will cause certain symptoms. For example, injury to the frontal lobes will cause loss of higher cognitive functions, such as loss of inhibitions leading to inappropriate social behavior. Injury to the cerebellum will cause loss of coordination and balance. The brainstem controls things like breathing and heart rate, as well as arousal. An injury to this area could inhibit any of these processes. **Methods of Diagnosis**

A detailed neurological examination is important and will bring out evidence of brain injury.
Brain imaging with CAT scan, MRI, SPECT and PET scan may be useful.
Cognitive evaluation by a Neurophysiologist with formal neuropsychological testing.
Evaluations by physical, occupational and speech therapists help clarify the specific deficits of an individual.

**Intracranial Pressure**

Intracranial pressure (the pressure in the brain) is controlled through the use of monitoring devices. Doctors place a small bolt in the patient's skull to measure intracranial pressure (ICP). A catheter is attached to the bolt in the brain which connects to a gauge that registers the amount of pressure in the skull. This procedure is most commonly performed on patients with moderate or severe brain injury.

The trauma care staff may try to keep the pressure down by:

Controlling body temperature (keeping the temperature low to normal)
Elevating the head of the bed
Using controlled narcotic sedation to cause paralysis, keeping the person still and comfortable
Ensuring proper breathing
Administering medication including Mannitol
Hypertensive therapies

**Diagnostic Tests**

The medical staff may conduct a number of diagnostic tests to determine what is occurring internally after the accident or illness.

X-rays, CT scans and MRI's of brain are pictures of the inside of the head. The picture will show if there is bleeding and/or swelling, skull fractures and where the damage has been done.

Often, Cervical Spine and other spinal films may be completed. When someone is involved in trauma, the neck and back may also be injured.

EEG: this test shows the presence of brain waves, their intensity and frequency. It is also used to determine if the patient is having seizures. [3]

**Diagnosis**

Diagnosis is suspected based on lesion circumstances and clinical evidence, most prominently a neurological examination, for example checking whether the pupils constrict normally in response to light and assigning a Glasgow Coma Score. Neuroimaging helps in determining the diagnosis and prognosis and in deciding what treatments to give.

The preferred radiological test in the emergency setting is computed tomography (CT): it is quick, accurate, and widely available. Follow-up CT scans may be performed later to determine whether the injury has progressed.

Magnetic resonance imaging (MRI) can show more detail than CT, and can add information about expected outcome in the long-term. It is more useful than CT for detecting injury characteristics such as diffuse axonal injury in the longer term. However, MRI is not used in the emergency setting for reasons including its relative inefficacy in detecting bleeds and fractures, its lengthy acquisition of images, the inaccessibility of the patient in the machine, and its incompatibility with metal items used in emergency care.

Other techniques may be used to confirm a particular diagnosis. X-rays are still used for head trauma, but evidence suggests they are not useful; head injuries are either so mild that they do not need imaging or severe enough to merit the more accurate CT. Angiography may be used to detect blood vessel pathology when risk factors such as penetrating head trauma are involved. Functional imaging can measure cerebral blood flow or metabolism, inferring neuronal activity in specific regions and potentially helping to predict outcome. Electroencephalography and Tran cranial Doppler may also be used.

Neuropsychological assessment can be performed to evaluate the long-term cognitive sequels and to aid in the planning of the rehabilitation. Instruments range from short measures of general mental functioning to complete batteries formed of different domain-specific tests.

**Prognosis**

Prognosis worsens with the severity of injury. Most TBIs are mild and do not cause permanent or long-term disability; however, all severity levels of TBI have the potential to cause significant, long-lasting disability. Permanent disability is thought to occur in 10% of mild injuries, 66% of moderate injuries, and 100% of severe injuries. Most mild TBI is completely resolved within three weeks and almost all people with mild TBI are able to live independently and return to the jobs they had before the injury, although a portion have mild cognitive and social impairments. Over 90% of people with moderate TBI are able to live independently, although a portion require assistance in areas such as physical abilities, employment, and financial managing. Most people with severe closed head injury either die or recover enough to live independently; middle ground is less common. Coma, as it is closely related to severity, is a strong predictor of poor outcome.

Prognosis differs depending on the severity and location of the lesion, and access to immediate, specialized acute management. Subarachnoid hemorrhage approximately doubles mortality.

Subdural hematoma is associated with worse outcome and increased mortality, while people with epidural hematoma are expected to have a good outcome if they receive surgery quickly. Diffuse axonal injury may be associated with coma when severe, and poor outcome. Following the acute stage, prognosis is strongly influenced by the patient's involvement in activity that promotes recovery, which for most patients requires access to a specialized, intensive rehabilitation service.

Medical complications are associated with a bad prognosis. Examples are hypotension (low blood pressure), hypoxia (low blood oxygen saturation), lower cerebral perfusion pressures and longer times spent with high intracranial pressures. Patient characteristics also influence prognosis. Factors thought to worsen it include abuse of substances such as illicit drugs and alcohol and age over sixty or under two years (in children, younger age at time of injury may be associated with a slower recovery of some abilities). [4]

# What are the Post-Traumatic Brain Injury Complications?

### What Immediate Post-Injury Complications Can Occur From a TBI?

Sometimes, health complications occur in the period immediately following a TBI. These complications are not types of TBI, but are distinct medical problems that arise as a result of the injury. Although complications are rare, the risk increases with the severity of the trauma. Complications of TBI include immediate seizures, hydrocephalus or post-traumatic ventricular enlargement, CSF leaks, infections, vascular injuries, cranial nerve injuries, pain, bed sores, multiple organ system failure in unconscious patients, and polytrauma (trauma to other parts of the body in addition to the brain).

About 25 percent of patients with brain contusions or hematomas and about 50 percent of patients with penetrating head injuries will develop immediate seizures, seizures that occur within the first 24 hours of the injury. These immediate seizures increase the risk of early seizures - defined as seizures occurring within 1 week after injury - but do not seem to be linked to the development of /I>post-traumatic epilepsy (recurrent seizures occurring more than 1 week after the initial trauma). Generally, medical professionals use anticonvulsant medications to treat seizures in TBI patients only if the seizures persist.

Hydrocephalus or post-traumatic ventricular enlargement occurs when CSF accumulates in the brain resulting in dilation of the cerebral ventricles (cavities in the brain filled with CSF) and an increase in ICP. This condition can develop during the acute stage of TBI or may not appear until later. Generally it occurs within the first year of the injury and is characterized by worsening neurological outcome, impaired consciousness, behavioral changes, ataxia (lack of coordination or balance), incontinence, or signs of elevated ICP. The condition may develop as a result of meningitis, subarachnoid hemorrhage, intracranial hematoma, or other injuries. Treatment includes shunting and draining of CSF as well as any other appropriate treatment for the root cause of the condition.

Skull fractures can tear the membranes that cover the brain, leading to CSF leaks. A tear between the dura and the arachnoid membranes, called a CSF fistula, can cause CSF to leak out of the

subarachnoid space into the subdural space; this is called a subdural hygroma . CSF can also leak from the nose and the ear. These tears that let CSF out of the brain cavity can also allow air and bacteria into the cavity, possibly causing infections such as meningitis. Pneumocephalus occurs when air enters the intracranial cavity and becomes trapped in the subarachnoid space.

Infections within the intracranial cavity are a dangerous complication of TBI. They may occur outside of the dura, below the dura, below the arachnoid (meningitis), or within the space of the brain itself (abscess). Most of these injuries develop within a few weeks of the initial trauma and result from skull fractures or penetrating injuries. Standard treatment involves antibiotics and sometimes surgery to remove the infected tissue. Meningitis may be especially dangerous, with the potential to spread to the rest of the brain and nervous system.

Any damage to the head or brain usually results in some damage to the vascular system, which provides blood to the cells of the brain. The body's immune system can repair damage to small blood vessels, but damage to larger vessels can result in serious complications. Damage to one of the major arteries leading to the brain can cause a stroke, either through bleeding from the artery ( hemorrhagic stroke ) or through the formation of a clot at the site of injury, called a thrombus or thrombosis, blocking blood flow to the brain ( ischemic stroke ). Blood clots also can develop in other parts of the head. Symptoms such as headache, vomiting, seizures, paralysis on one side of the body, and semi-consciousness developing within several days of a head injury may be caused by a blood clot that forms in the tissue of one of the sinuses, or cavities, adjacent to the brain. Thrombotic-ischemic strokes are treated with anticoagulants, while surgery is the preferred treatment for hemorrhagic stroke. Other types of vascular injuries include vasospasm and the formation of aneurysms .

Skull fractures, especially at the base of the skull, can cause cranial nerve injuries that result in compressive cranial neuropathies. All but three of the 12 cranial nerves project out from the brainstem to the head and face. The seventh cranial nerve, called the facial nerve, is the most commonly injured cranial nerve in TBI and damage to it can result in paralysis of facial muscles.

Pain is a common symptom of TBI and can be a significant complication for conscious patients in the period immediately following a TBI. Headache is the most common form of pain experienced by TBI patients, but other forms of pain can also be problematic. Serious complications for patients who are unconscious, in a coma, or in a vegetative state include bed or pressure sores of the skin, recurrent bladder infections, pneumonia or other life-threatening infections, and progressive multiple organ failure.

**What Disabilities Can Result From a TBI?**

Disabilities resulting from a TBI depend upon the severity of the injury, the location of the injury, and the age and general health of the patient. Some common disabilities include problems with cognition (thinking, memory, and reasoning), sensory processing (sight, hearing, touch, taste, and smell), communication (expression and understanding), and behavior or mental health (depression, anxiety, personality changes, aggression, acting out, and social inappropriateness).

Within days to weeks of the head injury approximately 40 percent of TBI patients develop a host of troubling symptoms collectively called post concussion syndrome (PCS). A patient need not

have suffered a concussion or loss of consciousness to develop the syndrome and many patients with mild TBI suffer from PCS. Symptoms include headache, dizziness, vertigo (a sensation of spinning around or of objects spinning around the patient), memory problems, trouble concentrating, sleeping problems, restlessness, irritability, apathy, depression, and anxiety. These symptoms may last for a few weeks after the head injury. The syndrome is more prevalent in patients who had psychiatric symptoms, such as depression or anxiety, before the injury. Treatment for PCS may include medicines for pain and psychiatric conditions, and psychotherapy and occupational therapy to develop coping skills.

Cognition is a term used to describe the processes of thinking, reasoning, problem solving, information processing, and memory. Most patients with severe TBI, if they recover consciousness, suffer from cognitive disabilities, including the loss of many higher level mental skills. The most common cognitive impairment among severely head-injured patients is memory loss, characterized by some loss of specific memories and the partial inability to form or store new ones. Some of these patients may experience post-traumatic amnesia (PTA), either anterograde or retrograde. Anterograde PTA is impaired memory of events that happened after the TBI, while retrograde PTA is impaired memory of events that happened before the TBI.

Many patients with mild to moderate head injuries who experience cognitive deficits become easily confused or distracted and have problems with concentration and attention. They also have problems with higher level, so-called executive functions, such as planning, organizing, abstract reasoning, problem solving, and making judgments, which may make it difficult to resume pre-injury work-related activities. Recovery from cognitive deficits is greatest within the first 6 months after the injury and more gradual after that.

**The most common cognitive impairment among severely head-injured patients is memory loss, characterized by some loss of specific memories and the partial inability to form or store new ones.**

Patients with moderate to severe TBI have more problems with cognitive deficits than patients with mild TBI, but a history of several mild TBIs may have an additive effect, causing cognitive deficits equal to a moderate or severe injury.

Many TBI patients have sensory problems, especially problems with vision. Patients may not be able to register what they are seeing or may be slow to recognize objects. Also, TBI patients often have difficulty with hand-eye coordination. Because of this, TBI patients may be prone to bumping into or dropping objects, or may seem generally unsteady. TBI patients may have difficulty driving a car, working complex machinery, or playing sports. Other sensory deficits may include problems with hearing, smell, taste, or touch. Some TBI patients develop tinnitus, a ringing or roaring in the ears. A person with damage to the part of the brain that processes taste or smell may develop a persistent bitter taste in the mouth or perceive a persistent noxious smell. Damage to the part of the brain that controls the sense of touch may cause a TBI patient to develop persistent skin tingling, itching, or pain. Although rare, these conditions are hard to treat.

Language and communication problems are common disabilities in TBI patients. Some may experience aphasia, defined as difficulty with understanding and producing spoken and written language; others may have difficulty with the more subtle aspects of communication, such as

body language and emotional, non-verbal signals.

In non-fluent aphasia, also called Broca's aphasia or motor aphasia, TBI patients often have trouble recalling words and speaking in complete sentences. They may speak in broken phrases and pause frequently. Most patients are aware of these deficits and may become extremely frustrated. Patients with fluent aphasia, also called Wernicke's aphasia or sensory aphasia, display little meaning in their speech, even though they speak in complete sentences and use correct grammar. Instead, they speak in flowing gibberish, drawing out their sentences with non-essential and invented words. Many patients with fluent aphasia are unaware that they make little sense and become angry with others for not understanding them. Patients with global aphasia have extensive damage to the portions of the brain responsible for language and often suffer severe communication disabilities.

TBI patients may have problems with spoken language if the part of the brain that controls speech muscles is damaged. In this disorder, called dysarthria, the patient can think of the appropriate language, but cannot easily speak the words because they are unable to use the muscles needed to form the words and produce the sounds. Speech is often slow, slurred, and garbled. Some may have problems with intonation or inflection, called prosodic dysfunction. An important aspect of speech, inflection conveys emotional meaning and is necessary for certain aspects of language, such as irony.

These language deficits can lead to miscommunication, confusion, and frustration for the patient as well as those interacting with him or her.

Most TBI patients have emotional or behavioral problems that fit under the broad category of psychiatric health. Family members of TBI patients often find that personality changes and behavioral problems are the most difficult disabilities to handle. Psychiatric problems that may surface include depression, apathy, anxiety, irritability, anger, paranoia, confusion, frustration, agitation, insomnia or other sleep problems, and mood swings. Problem behaviors may include aggression and violence, impulsivity, disinhibition, acting out, noncompliance, social inappropriateness, emotional outbursts, childish behavior, impaired self-control, impaired self awareness, inability to take responsibility or accept criticism, egocentrism, inappropriate sexual activity, and alcohol or drug abuse/addiction. Some patients' personality problems may be so severe that they are diagnosed with borderline personality disorder, a psychiatric condition characterized by many of the problems mentioned above. Sometimes TBI patients suffer from developmental stagnation, meaning that they fail to mature emotionally, socially, or psychologically after the trauma. This is a serious problem for children and young adults who suffer from a TBI. Attitudes and behaviors that are appropriate for a child or teenager become inappropriate in adulthood. Many TBI patients who show psychiatric or behavioral problems can be helped with medication and psychotherapy.

**Are There Other Long-Term Problems Associated With a TBI?**

In addition to the immediate post-injury complications discussed on page 13, other long-term problems can develop after a TBI. These include Parkinson's disease and other motor problems, Alzheimer's disease, dementia pugilistic, and post-traumatic dementia.

Alzheimer's disease (AD) - AD is a progressive, neurodegenerative disease characterized by dementia, memory loss, and deteriorating cognitive abilities. Recent research suggests an association between head injury in early adulthood and the development of AD later in life; the more severe the head injury, the greater the risk of developing AD. Some evidence indicates that a head injury may interact with other factors to trigger the disease and may hasten the onset of the disease in individuals already at risk. For example, people who have a particular form of the protein Apo lipoprotein E (apoE4) and suffer a head injury fall into this increased risk category. (ApoE4 is a naturally occurring protein that helps transport cholesterol through the bloodstream.)

Parkinson's disease and other motor problems - Movement disorders as a result of TBI are rare but can occur. Parkinson's disease may develop years after TBI as a result of damage to the basal ganglia. Symptoms of Parkinson's disease include tremor or trembling, rigidity or stiffness, slow movement (bradykinesia), inability to move (akinesia), shuffling walk, and stooped posture. Despite many scientific advances in recent years, Parkinson's disease remains a chronic and progressive disorder, meaning that it is incurable and will progress in severity until the end of life. Other movement disorders that may develop after TBI include tremor, ataxia (uncoordinated muscle movements), and myoclonus (shock-like contractions of muscles).

Dementia pugilistic - Also called chronic traumatic encephalopathy, dementia pugilistic primarily affects career boxers. The most common symptoms of the condition are dementia and Parkinsonism caused by repetitive blows to the head over a long period of time. Symptoms begin anywhere between 6 and 40 years after the start of a boxing career, with an average onset of about 16 years.

Post-traumatic dementia - The symptoms of post-traumatic dementia are very similar to those of dementia pugilistic, except that post-traumatic dementia is also characterized by long-term memory problems and is caused by a single, severe TBI that results in a coma. [2]

**Complications**

The relative risk of post-traumatic seizures increases with the severity of traumatic brain injury.

A CT of the head years after a traumatic brain injury showing an empty space where the damage occurred marked by the arrow.
Improvement of neurological function usually occurs for two or more years after the trauma. For many years it was believed that recovery was fastest during the first six months, but there is no evidence to support this. It may be related to services commonly being withdrawn after this period, rather than any physiological limitation to further progress. Children recover better in the immediate time frame and improve for longer periods.

Complications are distinct medical problems that may arise as a result of the TBI. The results of traumatic brain injury vary widely in type and duration; they include physical, cognitive, emotional, and behavioral complications. TBI can cause prolonged or permanent effects on consciousness, such as coma, brain death, persistent vegetative state (in which patients are unable to achieve a state of alertness to interact with their surroundings), and minimally conscious state (in which patients show minimal signs of being aware of self or environment). Lying still for long periods can cause complications including pressure scores, pneumonia or other infections, progressive multiple organ failure, and deep venous thrombosis, which can

cause pulmonary embolism. Infections that can follow skull fractures and penetrating injuries include meningitis and abscesses. Complications involving the blood vessels include vasospasm, in which vessels constrict and restrict blood flow, the formation of aneurysms, in which the side of a vessel weakens and balloons out, and stroke.

Movement disorders that may develop after TBI include tremor, ataxia (uncoordinated muscle movements), myoclonus (shock-like contractions of muscles), and loss of movement range and control (particularly with a loss of movement repertoire). The risk of post-traumatic seizures increases with severity of trauma (image at right) and is particularly elevated with certain types of brain trauma such as cerebral contusions or hematomas. People with early seizures, those occurring within a week of injury, have an increased risk of post-traumatic epilepsy (recurrent seizures occurring more than a week after the initial trauma). People may lose or experience altered vision, hearing, or smell.

Hormonal disturbances may occur secondary to hypopituitarism, occurring immediately or years after injury in 10 to 15% of TBI patients. Development of diabetes insipidus or an electrolyte abnormality acutely after injury indicate need for endocrinologic work up. Signs and symptoms of hypopituitarism may develop and be screened for in adults with moderate TBI and in mild TBI with imaging abnormalities. Children with moderate to severe head injury may also develop hypopituitarism. Screening should take place 3 to 6 months, and 12 months after injury, but problems may occur more remotely.

Cognitive deficits that can follow TBI include impaired attention; disrupted insight, judgment, and thought; reduced processing speed; distractibility; and deficits in executive functions such as abstract reasoning, planning, problem-solving, and multitasking. Memory loss, the most common cognitive impairment among head-injured people, occurs in 20–79% of people with closed head trauma, depending on severity. People who have suffered TBI may also have difficulty with understanding or producing spoken or written language, or with more subtle aspects of communication such as body language. Post-concussion syndrome, a set of lasting symptoms experienced after mild TBI, can include physical, cognitive, emotional and behavioral problems such as headaches, dizziness, difficulty concentrating, and depression. Multiple TBIs may have a cumulative effect. A young person who receives a second concussion before symptoms from another one have healed may be at risk for developing a very rare but deadly condition called second-impact syndrome, in which the brain swells catastrophically after even a mild blow, with debilitating or deadly results. About one in five career boxers is affected by chronic traumatic brain injury (CTBI), which causes cognitive, behavioral, and physical impairments. Dementia pugilistica, the severe form of CTBI, primarily affects career boxer's years after a boxing career. It commonly manifests as dementia, memory problems, and Parkinsonism (tremors and lack of coordination).

TBI may cause emotional or behavioral problems and changes in personality. These may include emotional instability, depression, anxiety, hypomania, mania, apathy, irritability, and anger. TBI appears to predispose a person to psychiatric disorders including obsessive compulsive disorder, alcohol, or substance abuse or dependence, dysthymia, clinical depression, bipolar disorder, phobias, panic disorder, and schizophrenia. Behavioral symptoms that can follow TBI include disinhibition, inability to control anger, impulsiveness, lack of initiative, inappropriate sexual activity, and changes in personality. Different behavioral problems are characteristic of the location of injury; for instance, frontal lobe injuries often result in disinhibition and inappropriate or childish behavior, and temporal lobe injuries often cause irritability and aggression. In patients

who have depression after TBI, suicidal ideation is not uncommon; the suicide rate among these persons is increased 2- to 3-fold.

TBI also has a substantial impact on the functioning of family systems Care giving family members and TBI survivors often significantly alter their familial roles and responsibilities following injury, creating significant change and strain on a family system. Typical challenges identified by families recovering from TBI include: frustration and impatience with one another, loss of former lives and relationships, difficulty setting reasonable goals, inability to effectively solve problems as a family, increased level of stress and household tension, changes in emotional dynamics, and overwhelming desire to return to pre-injury status. Additionally, families may exhibit less effective functioning in areas including coping, problem solving and communication. Psycho education and counseling models have been demonstrated to be effective in minimizing family disruption

In the 1970s awareness of TBI as a public health problem grew, and a great deal of progress has been made since then in brain trauma research, such as the discovery of primary and secondary brain injury. The 1990s saw the development and dissemination of standardized guidelines for treatment of TBI, with protocols for a range of issues such as drugs and management of intracranial pressure. Research since the early 1990s has improved TBI survival; that decade was known as the "Decade of the Brain" for advances made in brain research. [4]

# Glossary of Terms

**Glossary**

Aneurysm - a blood-filled sac formed by disease related stretching of an artery or blood vessel.

Anoxia - an absence of oxygen supply to an organ's tissues leading to cell death.

Aphasia - difficulty understanding and/or producing spoken and written language. (See also non-fluent aphasia.)

Apoptosis - cell death that occurs naturally as part of normal development, maintenance, and renewal of tissues within an organism.

Arachnoid membrane - one of the three membranes that cover the brain; it is between the pia mater and the dura. Collectively, these three membranes form the meninges.

Brain death - an irreversible cessation of measurable brain function.

Broca's aphasia - see non-fluent aphasia.

Cerebrospinal fluid (CSF) - the fluid that bathes and protects the brain and spinal cord.

Closed head injury - an injury that occurs when the head suddenly and violently hits an object but the object does not break through the skull.

Coma - a state of profound unconsciousness caused by disease, injury, or poison.

Compressive cranial neuropathies - degeneration of nerves in the brain caused by pressure on those nerves.

Computed tomography (CT) - a scan that creates a series of cross-sectional X-rays of the head and brain; also called computerized axial tomography or CAT scan.

Concussion - injury to the brain caused by a hard blow or violent shaking, causing a sudden and temporary impairment of brain function, such as a short loss of consciousness or disturbance of vision and equilibrium.

Contrecoup - a contusion caused by the shaking of the brain back and forth within the confines of the skull.

Contusion - distinct area of swollen brain tissue mixed with blood released from broken blood vessels.

CSF fistula - a tear between two of the three membranes - the dura and arachnoid membranes - that encase the brain.

Deep vein thrombosis - formation of a blood clot deep within a vein.

Dementia pugilistica - brain damage caused by cumulative and repetitive head trauma; common in career boxers.

Depressed skull fracture - a fracture occurring when pieces of broken skull press into the tissues of the brain.

Diffuse axonal injury - see shearing.

Dysarthria - inability or difficulty articulating words due to emotional stress, brain injury, paralysis, or spasticity of the muscles needed for speech.

Dura - a tough, fibrous membrane lining the brain; the outermost of the three membranes collectively called the meninges.

Early seizures - seizures that occur within 1 week after a traumatic brain injury.

Epidural hematoma - bleeding into the area between the skull and the dura.

Erosive gastritis - inflammation and degeneration of the tissues of the stomach.

Fluent aphasia - a condition in which patients display little meaning in their speech even though they speak in complete sentences. Also called Wernicke's or motor aphasia.

Glasgow Coma Scale - a clinical tool used to assess the degree of consciousness and neurological functioning - and therefore severity of brain injury - by testing motor responsiveness, verbal acuity, and eye opening.

Global aphasia - a condition in which patients suffer severe communication disabilities as a result of extensive damage to portions of the brain responsible for language.

Hematoma - heavy bleeding into or around the brain caused by damage to a major blood vessel in the head.

Hemorrhagic stroke - stroke caused by bleeding out of one of the major arteries leading to the brain.

Hyper metabolism - a condition in which the body produces too much heat energy.

hypothyroidism - decreased production of thyroid hormone leading to low metabolic rate, weight gain, chronic drowsiness, dry skin and hair, and/or fluid accumulation and retention in connective tissues.

Hypoxia - decreased oxygen levels in an organ, such as the brain; less severe than anoxia.

Immediate seizures - seizures that occur within 24 hours of a traumatic brain injury.

Intracerebral hematoma - bleeding within the brain caused by damage to a major blood vessel.

Intracranial pressure - buildup of pressure in the brain as a result of injury.

Ischemic stroke - stroke caused by the formation of a clot that blocks blood flow through an artery to the brain.

Locked-in syndrome - a condition in which a patient is aware and awake, but cannot move or communicate due to complete paralysis of the body.

Magnetic resonance imaging (MRI) - a noninvasive diagnostic technique that uses magnetic fields to detect subtle changes in brain tissue.

Meningitis - inflammation of the three membranes that envelop the brain and spinal cord, collectively known as the meninges; the meninges include the dura, pia mater, and arachnoid.

Motor aphasia - see non-fluent aphasia.

Neural stem cells - cells found only in adult neural tissue that can develop into several different cell types in the central nervous system.

Neuroexcitation - the electrical activation of cells in the brain; neuroexcitation is part of the normal functioning of the brain or can also be the result of abnormal activity related to an injury.

Neuron - a nerve cell that is one of the main functional cells of the brain and nervous system.

Neurotransmitters -chemicals that transmit nerve signals from one neuron to another.

Non-fluent aphasia - a condition in which patients have trouble recalling words and speaking in complete sentences. Also called Broca's or motor aphasia.

Oligodendrocytes - a type of support cell in the brain that produces myelin, the fatty sheath that surrounds and insulates axons.

Penetrating head injury - a brain injury in which an object pierces the skull and enters the brain tissue.

Penetrating skull fracture - a brain injury in which an object pierces the skull and injures brain tissue.

Persistent vegetative state - an ongoing state of severely impaired consciousness, in which the patient is incapable of voluntary motion.

Plasticity - ability of the brain to adapt to deficits and injury.

Pneumocephalus - a condition in which air or gas is trapped within the intracranial cavity.

Post-concussion syndrome (PCS) - a complex, poorly understood problem that may cause headache after head injury; in most cases, patients cannot remember the event that caused the concussion and a variable period of time prior to the injury.

Post-traumatic amnesia (PTA) - a state of acute confusion due to a traumatic brain injury, marked by difficulty with perception, thinking, remembering, and concentration; during this acute stage, patients often cannot form new memories.

Post-traumatic dementia - a condition marked by mental deterioration and emotional apathy following trauma.

Post-traumatic epilepsy - recurrent seizures occurring more than 1 week after a traumatic brain injury.

Prosodic dysfunction - problems with speech intonation or inflection.

Pruning - process whereby an injury destroys an important neural network in children, and another less useful neural network that would have eventually died takes over the responsibilities of the damaged network.

seizures - abnormal activity of nerve cells in the brain causing strange sensations, emotions, and behavior, or sometimes convulsions, muscle spasms, and loss of consciousness.

Sensory aphasia - see fluent aphasia.

Shaken baby syndrome - a severe form of head injury that occurs when an infant or small child is shaken forcibly enough to cause the brain to bounce against the skull; the degree of brain damage depends on the extent and duration of the shaking. Minor symptoms include irritability, lethargy, tremors, or vomiting; major symptoms include seizures, coma, stupor, or death.

Shearing (or diffuse axonal injury) - damage to individual neurons resulting in disruption of neural networks and the breakdown of overall communication among neurons in the brain.

Stupor - a state of impaired consciousness in which the patient is unresponsive but can be aroused briefly by a strong stimulus.

Subdural hematoma - bleeding confined to the area between the dura and the arachnoid membranes.

Subdural hygroma - a buildup of protein rich fluid in the area between the dura and the arachnoid membranes, usually caused by a tear in the arachnoid membrane.

syndrome of inappropriate secretion of antidiuretic hormone (SIADH) - a condition in which excessive secretion of antidiuretic hormone leads to a sodium deficiency in the blood and abnormally concentrated urine; symptoms include weakness, lethargy, confusion, coma, seizures, or death if left untreated.

Thrombosis or thrombus - the formation of a blood clot at the site of an injury.

Vasospasm - exaggerated, persistent contraction of the walls of a blood vessel.

Vegetative state - a condition in which patients are unconscious and unaware of their surroundings, but continue to have a sleep/wake cycle and can have periods of alertness.

Ventriculostomy - a surgical procedure that drains cerebrospinal fluid from the brain by creating an opening in one of the small cavities called ventricles. [2]

**Definitions Related to TBI**

There are several ways to describe brain injuries. The brain is enclosed in the bony vault of the skull. The cerebrospinal fluid surrounds the brain and, most of the time, protects it from impact with the skull. If there is a rapid force applied to the skull or rapid deceleration of the head, the brain may strike the inside of the bony vault.

Brain tissue may stretch or tear because of the rapid movement. This can injure the nervous tissue of the brain directly. If a projectile such as a bullet enters the skull, it can directly injure the brain.

Below is a list of terms and definitions that refer to the different injuries of TBI.

**Closed Head Injury**- the skull is intact and there is no penetration of the skull. Direct or indirect force to the head can cause this type of injury. This may be caused by rotational and/or deceleration in the case of both direct and indirect force. **Open Head Injury**- penetration of the skull with direct injury to the head.

**Diffuse Axonal Injury**- diffuse cellular injury to the brain from rapid rotational movement. This is often seen in motor vehicle accidents or shaking injuries. The axons are the projections of the brains nerve cells that attach to other nerve cells. They are damaged or torn by the rapid deceleration. The injury is from the shearing force disrupting the axons which compose the

white matter of the brain. **Contusion**- a bruise to a part of the brain. Like a bruise on the body, this is bleeding into the tissue. **Penetrating Trauma**- any object that enters the brain. Causes direct injury by impact and pushing skull fragments into the brain. **Secondary Injury**- swelling and release of chemicals that promote inflammation and cell injury or death. This causes swelling in the brain which may increase the intracranial pressure and prevent the cerebrospinal fluid from draining out of the skull. This causes further increase in pressure and brain damage. If this is not controlled or prevented the brain can herniate (push through) the base of the skull and cause respiratory failure and death. The only way to prevent the primary injury is to prevent the trauma. The prevention of this secondary injury is the focus of the acute medical care after injury.

Secondary Injury Includes:

- Intracranial hemorrhage (bleeding inside the skull)
- Brain swelling
- Increased intracranial pressure (pressure inside the skull)
- Brain damage associated with lack of oxygen
- Infection inside the skull, common with penetrating trauma
- Chemical changes leading to cell death
- Increased fluid inside the skull (hydrocephalus)

**Acquired Brain Injury**- injuries other than congenital, birth trauma, hereditary or degenerative. This includes traumatic brain injury. In the non-traumatic types of acquired brain injury, the brain is usually diffusely injured. These injuries are usually not included in traumatic brain injury but the symptoms span the same spectrum.

Common causes are *anoxia* and *hypoxia*. These are lack of oxygen to the brain and insufficient oxygen to the brain. They can occur because of mechanical problems with breathing, with cardiac arrest or bleeding. Drugs and poisoning can also cause acquired traumatic brain injury. Carbon monoxide poisoning is an example of poisoning that may cause brain injury. [3]

# Research

### What Research is the NINDS Conducting?

The National Institute of Neurological Disorders and Stroke (NINDS) conducts and supports research to better understand CNS injury and the biological mechanisms underlying damage to the brain, to develop strategies and interventions to limit the primary and secondary brain damage that occurs within days of a head trauma, and to devise therapies to treat brain injury and help in long-term recovery of function.

On a microscopic scale, the brain is made up of billions of cells that interconnect and

communicate.

The neuron is the main functional cell of the brain and nervous system, consisting of a cell body (soma), a tail or long nerve fiber (axon), and projections of the cell body called dendrites. The axons travel in tracts or clusters throughout the brain, providing extensive interconnections between brain areas.

One of the most pervasive types of injury following even a minor trauma is damage to the nerve cell's axon through shearing; this is referred to as diffuse axonal injury. This damage causes a series of reactions that eventually lead to swelling of the axon and disconnection from the cell body of the neuron. In addition, the part of the neuron that communicates with other neurons degenerates and releases toxic levels of chemical messengers called neurotransmitters into the synapse or space between neurons, damaging neighboring neurons through a secondary neuroexcitatory cascade. Therefore, neurons that were unharmed from the primary trauma suffer damage from this secondary insult. Many of these cells cannot survive the toxicity of the chemical onslaught and initiate programmed cell death, or apoptosis. This process usually takes place within the first 24 to 48 hours after the initial injury, but can be prolonged.

One area of research that shows promise is the study of the role of calcium ion influx into the damaged neuron as a cause of cell death and general brain tissue swelling. Calcium enters nerve cells through damaged channels in the axon's membrane. The excess calcium inside the cell causes the axon to swell and also activates chemicals, called proteases that break down proteins. One family of proteases, the calpains, are especially damaging to nerve cells because they break down proteins that maintain the structure of the axon. Excess calcium within the cell is also destructive to the cell's mitochondria, structures that produce the cell's energy. Mitochondria soak up excess calcium until they swell and stop functioning. If enough mitochondria are damaged, the nerve cell degenerates. Calcium influx has other damaging effects: it activates destructive enzymes, such as caspases that damage the DNA in the cell and trigger programmed cell death, and it damages sodium channels in the cell membrane, allowing sodium ions to flood the cell as well. Sodium influx exacerbates swelling of the cell body and axon.

NINDS researchers have shown, in both cell and animal studies, that giving specialized chemicals can reduce cell death caused by calcium ion influx. Other researchers have shown that the use of cyclosporine A, which blocks mitochondrial membrane permeability, protects axons from calcium influx. Another avenue of therapeutic intervention is the use of hypothermia (an induced state of low body temperature) to slow the progression of cell death and axon swelling.

In the healthy brain, the chemical glutamate functions as a neurotransmitter, but an excess amount of glutamate in the brain causes neurons to quickly overload from too much excitation, releasing toxic chemicals. These substances poison the chemical environment of surrounding cells, initiating degeneration and programmed cell death. Studies have shown that a group of enzymes called matrix metalloproteinases contribute to the toxicity by breaking down proteins that maintain the structure and order of the extracellular environment. Other research shows that glutamate reacts with calcium and sodium ion channels on the cell membrane, leading to an influx of calcium and sodium ions into the cell. Investigators are looking for ways to decrease the toxic effects of glutamate and other excitatory neurotransmitters.

The brain attempts to repair itself after a trauma, and is more successful after mild to moderate injury than after severe injury. Scientists have shown that after diffuse axonal injury neurons can spontaneously adapt and recover by sprouting some of the remaining healthy fibers of the neuron into the spaces once occupied by the degenerated axon. These fibers can develop in such a way that the neuron can resume communication with neighboring neurons. This is a very delicate process and can be disrupted by any of a number of factors, such as neuroexcitation, hypoxia (low oxygen levels), and hypotension (low blood flow). Following trauma, excessive neuroexcitation, that is the electrical activation of nerve cells or fibers, especially disrupts this natural recovery process and can cause sprouting fibers to lose direction and connect with the wrong terminals.

Scientists suspect that these misconnections may contribute to some long-term disabilities, such as pain, spasticity, seizures, and memory problems. NINDS researchers are trying to learn more about the brain's natural recovery process and what factors or triggers control it. They hope that through manipulation of these triggers they can increase repair while decreasing misconnections.

NINDS investigators are also looking at larger, tissue-specific changes within the brain after a TBI. Researchers have shown that trauma to the frontal lobes of the brain can damage specific chemical messenger systems, specifically the dopaminergic system, the collection of neurons in the brain that uses the neurotransmitter dopamine. Dopamine is an important chemical messenger - for example, degeneration of dopamine-producing neurons is the primary cause of Parkinson's disease. NINDS researchers are studying how the dopaminergic system responds after a TBI and its relationship to neurodegeneration and Parkinson's disease.

The use of stem cells to repair or replace damaged brain tissue is a new and exciting avenue of research. A neural stem cell is a special kind of cell that can multiply and give rise to other more specialized cell types. These cells are found in adult neural tissue and normally develop into several different cell types found within the central nervous system. NINDS researchers are investigating the ability of stem cells to develop into neurotransmitter-producing neurons, specifically dopamine-producing cells. Researchers are also looking at the power of stem cells to develop into oligodendrocytes, a type of brain cell that produces myelin, the fatty sheath that surrounds and insulates axons. One study in mice has shown that bone marrow stem cells can develop into neurons, demonstrating that neural stem cells are not the only type of stem cell that could be beneficial in the treatment of brain and nervous system disorders. At the moment, stem cell research for TBI is in its infancy, but future research may lead to advances for treatment and rehabilitation.

In addition to the basic research described above, NINDS scientists also conduct broader based clinical research involving patients. One area of study focuses on the plasticity of the brain after injury. In the strictest sense, plasticity means the ability to be formed or molded. When speaking of the brain, plasticity means the ability of the brain to adapt to deficits and injury. NINDS researchers are investigating the extent of brain plasticity after injury and developing therapies to enhance plasticity as a means of restoring function.

The plasticity of the brain and the rewiring of neural connections make it possible for one part of the brain to take up the functions of a disabled part. Scientists have long known that the immature brain is generally more plastic than the mature brain, and that the brains of children are

better able to adapt and recover from injury than the brains of adults. NINDS researchers are investigating the mechanisms underlying this difference and theorize that children have an overabundance of hard-wired neural networks, many of which naturally decrease through a process called pruning. When an injury destroys an important neural network in children, another less useful neural network that would have eventually died takes over the responsibilities of the damaged network. Some researchers are looking at the role of plasticity in memory, while others are using imaging technologies, such as functional MRI, to map regions of the brain and record evidence of plasticity.

**In the strictest sense, plasticity means the ability to be formed or molded. When speaking of the brain, plasticity means the ability of the brain to adapt to deficits and injury.**

Another important area of research involves the development of improved rehabilitation programs for those who have disabilities from a TBI. The Congressional Children's Health Act of 2000 authorized the NINDS to conduct and support research related to TBI with the goal of designing therapies to restore normal functioning in cognition and behavior.

**Clinical Trials Research**

The NINDS works to develop treatments that can be given in the first hours after a TBI, hoping that quick action can prevent or reverse much of the brain damage resulting from the injury. A recently completed NINDS-supported clinical trial involved lowering body temperature in TBI patients to 33 degrees Celsius within 8 hours of the trauma. Although the investigators found that the treatment did not improve outcome overall, they did learn that patients younger than 45 years who were admitted to the hospital already in a hypothermic state fared better if they were kept .cool. than if they were brought to normal body temperature. Other ongoing clinical trials include the use of hypothermia for severe TBI in children, the use of magnesium sulfate to protect nerve cells after TBI, and the effects of lowering ICP and increasing cerebral blood flow. [2]

Research

No medication exists to halt the progression of secondary injury, but the variety of pathological events presents opportunities to find treatments that interfere with the damage processes. Neuroprotection, methods to halt or mitigate secondary injury, have been the subject of great interest for their ability to limit the damage that follows TBI. However, clinical trials to test agents that could halt these cellular mechanisms have largely met with failure. For example, interest existed in hypothermia, cooling the injured brain to limit TBI damage, but clinical trials showed that it is not useful in the treatment of TBI. In addition, drugs such as NMDA receptor antagonists to halt neurochemical cascades such as excitotoxicity showed promise in animal trials but failed in clinical trials. These failures could be due to factors including faults in the trials' design or in the insufficiency of a single agent to prevent the array of injury processes involved in secondary injury.

Developments in technologies may provide doctors with valuable medical information. For example, work has been done to design a device to monitor oxygenation that could be attached to a probe placed into the brain—such probes are currently used to monitor ICP. Research is also planned to clarify factors correlated to outcome in TBI and to determine in which cases it is best

to perform CT scans and surgical procedures.

Hyperbaric oxygen therapy (HBO) has been evaluated as an adjunctive treatment following TBI concluding a Cochrane review that its use could not be justified. HBO for TBI has remained controversial as studies have looked for improvement mechanisms, and further evidence shows that it may have potential as a treatment.[4]

# Articles Related to Traumatic Brain Injury

**Experts say more needs to be done for brain-injured**

**Loved ones lobby for bill that would require more help for wounded vets**

By Leo Shane III, Stars and Stripes
Saturday, March 31, 2007

WASHINGTON - Retired Army Sgt. Edward Wade has adjusted to his prosthetic right arm, his nagging foot pain and most of the other ailments caused by a roadside bomb in Iraq three years ago.

What he hasn't been able to get used to is the lingering fogginess in his mind from the brain injury he sustained in that blast.

"Everything comes out very slow and meaningful now," the 28-year-old said, with frequent pauses underscoring his frustration. "Everybody else can do more than one thing at once, but I'm forced to just focus on doing one thing at a time."

Wade and his family joined medical experts on Capitol Hill on Thursday to lobby for more brain injury research, both before troops deploy and after they return home, and for a bill introduced by presidential candidate Sen. Hillary Clinton, D-N.Y., mandating more attention on the issue.

That legislation would require more access to mental rehabilitation for wounded troops through the departments of Defense and Veterans Affairs, better training for family members to care for injured loved ones, and more comprehensive monitoring of all troops for signs of brain trauma.

Health officials said the military has done a good job identifying severe brain trauma cases for troops such as Wade, who was in a coma and near death after his February 2004 injury.

But they added that mild cases of head trauma — concussive explosions that don’t leave visible signs of injury, for example — are not being identified or treated, even though those injuries can lead to long-term problems.

“We’ve seen a rise in [troops] who are having seizures, people who are having trouble controlling their thoughts and memories and emotions,” said Dr. Katherine Henry, director of neurology for the New York University School of Medicine.

“For many of these even with mild brain injuries, they may never return to full brain functions.”

Landstuhl Regional Medical Center in Germany launched a brain injury screening program last May, identifying a third of its war-related patients as suffering from some level of mental impairment.

John Melia, executive director of the Wounded Warrior Project, said screening programs are needed for every deployed service member, as well as some baseline evaluation of those troops before they leave for comparison.

“We really need the government to step up,” he said. “If this country cannot serve severely injured men and women coming back from this conflict, we need to look at ourselves. Unfortunately, to this point we have failed.”

Wade has received some rehabilitation for his brain injuries, but his wife, Sarah, said they’ve had to fight for every evaluation and treatment session. Army officials pressured him to medically retire, she said, and VA officials aren’t equipped to provide consistent and quick care for the new head trauma cases coming out of the war zone.

“I’ve been his driver, his case manager and his primary caregiver,” she said. “Without me and our families there is no way he could have made the recovery he has. But there are plenty of soldiers who don’t have that help.”

**AR'S NEW WOUNDS**
**A Shock Wave of Brain Injuries**
*By Ronald Glasser*
Sunday, April 8, 2007; Page B01
*"We can save you. But you might not be what you were."*

*Neurosurgeon, Combat Support Hospital, Balad, Iraq*

This is the new physics of war. Three 155mm shells, linked together and combined with 100 pounds of Semtex plastic explosive, covered by canisters of butane or barrels of gasoline, can upend a 70-ton tank, destroy a Humvee or blow an engine block through the hood of a truck. Those deadly ingredients form the signature weapon of the war in Iraq: improvised explosive devices, known by anybody who watches the news as IEDs.

Some of the impact of these roadside bombs is brutally clear: Troops are maimed by projectiles, poisoned by clouds of bacteria-laced debris and burned by post-blast flames. But the IEDs have added a new dimension to battlefield injuries: wounds and even deaths among troops who have no external signs of trauma but whose brains have been severely damaged. Iraq has brought back one of the worst afflictions of World War I trench warfare: shell shock. The brain of a soldier

exposed to a roadside bomb is shocked, truly.

About 1,800 U.S. troops, according to the Department of Veterans Affairs, are now suffering from traumatic brain injuries (TBIs) caused by penetrating wounds. But neurologists worry that hundreds of thousands more -- at least 30 percent of the troops who've engaged in active combat for four months or longer in Iraq and Afghanistan -- are at risk of potentially disabling neurological disorders from the blast waves of IEDs and mortars, all without suffering a scratch.

For the first time, the U.S. military is treating more head injuries than chest or abdominal wounds, and it is ill-equipped to do so. According to a July 2005 estimate from Walter Reed Army Medical Center, two-thirds of all soldiers wounded in Iraq who don't immediately return to duty have traumatic brain injuries.

Here's why IEDS carry such hidden danger. The detonation of any powerful explosive generates a blast wave of high pressure that spreads out at 1,600 feet per second from the point of explosion and travels hundreds of yards. The lethal blast wave is a two-part assault that rattles the brain against the skull. The initial shock wave of very high pressure is followed closely by the "secondary wind": a huge volume of displaced air flooding back into the area, again under high pressure. No helmet or armor can defend against such a massive wave front.

It is these sudden and extreme differences in pressures -- routinely 1,000 times greater than atmospheric pressure -- that lead to significant neurological injury. Blast waves cause severe concussions, resulting in loss of consciousness and obvious neurological deficits such as blindness, deafness and mental retardation. Blast waves causing TBIs can leave a 19-year-old private who could easily run a six-minute mile unable to stand or even to think.

Another problem is that these blast-related brain injuries differ from other severe head traumas, and the complexity of treating returning troops with "closed-head" injuries is taxing an already overburdened military health-care system. There is not a neurosurgeon who works in a trauma unit anywhere in the United States who doesn't know what to do when an ambulance brings in a biker who has suffered a severe head injury in a highway accident. The standard care involves using calcium channel blockers to protect damaged nerve cells against further injury, intravenous diuretics to control brain swelling and, if the swelling becomes too great, removal of the top of the skull to allow the brain to swell without increasing neurological damage. This is what surgeons did in the case of ABC News anchor Bob Woodruff, who suffered severe brain injuries from an IED blast in Baghdad last year.

All this works with the common types of severe head injuries, but it does not work with brains damaged by shock waves. Despite the usual interventions and treatments, the majority of blast-injury patients who have neurological damage do not fully recover. There is a growing understanding within the neurosurgical community that blast injuries are different from those caused by penetrating or skull-fracture trauma. It is thought that shock waves damage the brain at a microscopic, sub-cellular level. That's why surgeons who are quite capable of reconstructing the skull of a motorcycle crash victim -- something for which they have been well trained -- struggle to come up with treatment and rehabilitation techniques for the explosion-damaged brains of troops.

"TBIs from Iraq are different," said P. Steven Macedo, a neurologist and former doctor at the Veterans Administration. Concussions from motorcycle accidents injure the brain by stretching or tearing it, he noted. But in Iraq, something else is going on. "When the sound wave moves through the brain, it seems to cause little gas bubbles to form," he said. "When they pop, it leaves a cavity. So you are littering people's brains with these little holes."

Almost as daunting as treating TBI is the volume of such injuries coming out of Iraq. Macedo cited the estimates, gleaned at seminars with VA doctors, that as many as one-third of all combat forces are at risk of TBI. Military physicians have learned that significant neurological injuries should be suspected in any troops exposed to a blast, even if they were far from the explosion. Indeed, soldiers walking away from IED blasts have discovered that they often suffer from memory loss, short attention spans, muddled reasoning, headaches, confusion, anxiety, depression and irritability.

**A Shock Wave of Brain Injuries**

What's baffling is the Pentagon's failure to work with Congress to provide a steady stream of funding for research on TBIs. Meanwhile, the high-profile firings of top commanders at Walter Reed have shed light on the woefully inadequate treatment for troops. In these circumstances, soldiers face a struggle to get the long-term rehabilitation necessary for a TBI. At Walter Reed, Macedo said, doctors have chosen to medicate most TBI patients, even though cognitive rehabilitation, including brain teasers and memory exercises, seems to hold the most promise for dealing with the disorder.

Oddly enough, having more military patients than can be adequately treated is, in terms of warfare, a gruesome kind of success. These are the war injured who once would have been the war dead. And it is the unexpected number of casualties who in a previous medical era would have been fatalities that has sunk the outpatient clinics at Walter Reed and left those in the VA system lost and adrift.

Iraq and Afghanistan, the ratio of wounded service members to fatalities is 16 to 1, if the definition of "wounded" is anyone evacuated from a combat zone. During the Vietnam War, according to the VA, the ratio was 2.6 to 1. U.S. troops no longer die from the kind of injuries that killed many thousands in Vietnam. The majority of combat deaths there occurred right where the soldier was hit. If you were going to die, you were dead before there was any need of a medevac chopper. If you'd had an arm or leg blown off, the chances were that you had also suffered a penetrating chest or abdominal wound and would bleed to death waiting to be taken to the nearest surgical hospital.

But if the bleeding could be staunched and you were still breathing when the medics got to you, the odds on survival were in your favor. The military medicine practiced in Vietnam wasn't so different from what World War II medics practiced: Stop the bleeding and hope for the best until the helicopter shows up.

It wasn't until October 1993, when a U.S. combat assault team rappelled down from a helicopter into a 72-hour gunfight in the streets of Mogadishu, Somalia, that the notion of military medicine changed from basic life support to intensive care. In that siege situation, medics had no choice but to care for a growing number of wounded on their own, because evacuation was impossible. But without clear intensive-care procedures, they ran out of medications and fluids to treat the

most severely injured.

In the civilian world, trauma medicine had progressed throughout the 1970s and '80s, well past the simple expedients of tourniquet, plasma and keeping an airway open. Mogadishu forced the military to abandon the last of its medical practices from Vietnam. It was time to teach the medics a new trade.

Pentagon officials increased the training period for a 91W, or combat medic, from 10 to 16 weeks. Medics now trained on patient simulators that would "bleed to death" if blood loss was not stopped or "suffocate" if chest tubes weren't correctly placed or a tracheotomy wasn't performed within three minutes. Medics learned the new intensive-care theory of "hypotensive resuscitation," in which intravenous fluids are given only in minimal amounts solely to keep the heart pumping, as opposed to the old Vietnam method of keeping blood pressure elevated, which only added to blood loss. Medics today use better-designed tourniquets and hemostatic bandages -- dressings that act to stop bleeding for better hemorrhage control. They administer the latest non-opiate painkillers, which, unlike morphine and Demerol, do not slow breathing. This is the first war in which troops are very unlikely to die if they're still alive when a medic arrives.

Another large part of the 16-to-1 wounded-to-fatality ratio has to do with advances in body armor. Today's body armor is dramatically effective in preventing fatal wounds of the chest and upper abdomen. There is not an orthopedic or general surgeon in Iraq or Afghanistan who hasn't been astonished the first time a trooper with two missing limbs and a traumatic brain injury is carried off in a chopper and the surgeon removing the armor cannot find a scratch from the chin to the groin.

But the unseen damage can be long-lasting. Most of the families of our wounded that I have interviewed months, if not years, after the injury say the same thing: "Someone should have told us that with these closed-head injuries, things would not really get all that much better."

Now in its fifth year, the Iraq conflict is not a war of death for U.S. troops nearly so much as it is a war of disabilities. The symbol of this battle is not the cemetery but the orthopedic ward and the neurosurgical unit. The men and women inside those units have come home alive but missing arms and legs, many unable to see or hear or remember who they were before being hit by a roadside bomb. Survival clearly represents as much of a revolution in military medicine as does the dominance of the suicide bomber and the roadside bomb in the age of "shock and awe." But now both the medical profession and the country are left to play a terrible game of catch-up.

Ronald Glasser is a pediatric nephrologist and the author of " Wounded: Vietnam to Iraq," published last year. From 1968 to 1970, he was deployed at the U.S. Army Hospital at Camp Zama, Japan, treating U.S. soldiers wounded in Vietnam. [11]

**Footnotes:**

**1. http://www.ninds.nih.gov/disorders/tbi/tbi.htm?css=print**
**2. http://www.ninds.nih.gov/disorders/tbi/detail_tbi.htm?css=print**
**3. http://www.traumaticbraininjury.com/content/understandingtbi/tbi-understanding-**

**tbi.html**
**4. http://en.wikipedia.org/wiki/Traumatic_brain_injury**
**5, http://www.cdc.gov/traumaticbraininjury/**
**6. http://www.traumaticbraininjury.com/**