

# Traumatic Brain Injury



National Institute of Neurological Disorders  
and Stroke  
National Institutes of Health

Hope Through Research

**Cover:** Gary Weinstein of Takoma Park, Maryland, pictured with his wife Julie Wiatt and his son Zak, suffered a severe head injury when a ball shattered his skull while he was coaching a baseball team practice in the spring of 2001. A year after his injury, he returned to work and is back to coaching.

This pamphlet was written and published by the National Institute of Neurological Disorders and Stroke (NINDS), the United States' leading supporter of research on disorders of the brain and nerves, including traumatic brain injury. NINDS, one of the U.S. Government's National Institutes of Health in Bethesda, Maryland, is part of the Public Health Service within the U.S. Department of Health and Human Services.

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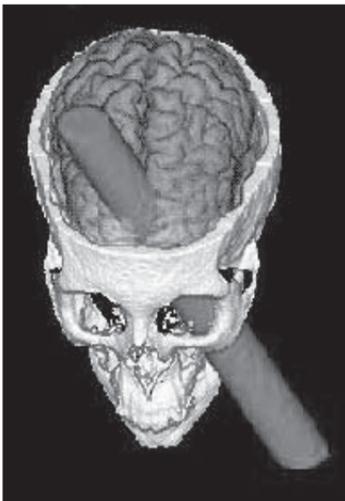
**Information Resources**

**Traumatic Brain Injury Research Centers**  
(see pocket inside back cover)

## Introduction

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



This computer-generated graphic shows how, in 1848, a 3-foot long, pointed rod penetrated the skull of Phineas Gage, a railway construction foreman. The rod entered at the top of his head, passed through his brain, and exited his skull by his temple. Gage survived the accident but suffered lasting personality and behavioral problems.

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 \$48 billion a year, and between 2.5 and 6.5 million Americans alive today have had a TBI. 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 270,000 people experience a moderate or severe TBI,
- approximately 70,000 people die from head injury,
- approximately 1 million head-injured people are treated in hospital emergency rooms,
- approximately 60,000 new cases of epilepsy occur as a result of head trauma,
- approximately 230,000 people are hospitalized for TBI and survive, and
- approximately 80,000 of these survivors live with significant disabilities as a result of the injury.

## What is a Traumatic Brain Injury?

TBI, also called acquired brain injury or simply head 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.

## 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.

\* Terms in Italics are defined in the Glossary, page 36.

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.

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



Transportation accidents involving automobiles, motorcycles, bicycles, and pedestrians account for half of all TBIs and are the major cause of TBIs in people under age 75.

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 firearm TBIs (two-thirds of which may be suicidal in intent) result in death, while only 11 percent of TBIs from falls result in death.

## 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.

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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 “*contrecoup*.” 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, contrecoup 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.

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## 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.

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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



Medical professionals take great care in moving and transporting head-injured patients. Often back-boards and neck restraints are used to immobilize patients and prevent further injury.

## Glasgow Coma Scale

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

- 4 indicates 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.

measures — eye opening, best verbal response, and best motor response — to determine the severity of the patient's brain injury.

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.

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 *intra-cranial 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



A computed tomography scan creates X-ray images of the head and brain. This imaging test can show bone fractures, hemorrhages, hematomas, contusions, brain tissue swelling, and tumors, and can help determine the prognosis of TBI patients.

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.

## How Does a TBI Affect Consciousness?

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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.

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*“Advances in imaging and other technologies have led to devices that help differentiate among the variety of unconscious states.”*

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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 cerebral hemispheres of 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).

## 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).

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A medical technician carefully monitors a patient undergoing an electroencephalogram, a diagnostic technique used to detect abnormalities in brain waves.

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 *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.

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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 semiconsciousness 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.

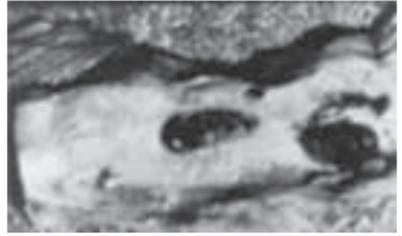
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Pain is a common symptom of TBI and can be a significant complication for conscious patients in the period immediately following a TBI. Headache is the most common form of pain experienced by TBI patients, but other forms of pain can also be problematic. 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.

## **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.



In addition to head and brain injuries, most TBI patients have injuries in other parts of the body — a condition called polytrauma. The formation of blood clots deep inside a vein (shown above), referred to as deep vein thrombosis, is one of the many medical complications that may occur with TBI.

Trauma victims often develop *hypermetabolism* 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 hypermetabolism 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.

## What Disabilities Can Result From a TBI?

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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

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*“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.”*

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the injury and more gradual after that.

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.

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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*



Communication disabilities such as language and speech problems are common among TBI patients. Above, a speech therapist uses a mirror in her work, performing exercises to help a patient relearn speaking skills.

*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 pugilistica*, 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 apolipoprotein 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



Dementia pugilistica is a condition caused by repetitive blows to the head over a long period of time. Symptoms of the disorder, which primarily affects career boxers, include dementia and parkinsonism.

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).

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*Dementia pugilistica* — Also called chronic traumatic encephalopathy, dementia pugilistica 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 pugilistica, 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.

## 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 subacute 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.

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

\* National Institutes of Health Consensus Development Conference Statement, October 26–28, 1998. *Rehabilitation of Persons with Traumatic Brain Injury*. Bethesda, MD, September 1999.



Rehabilitation therapy is an important part of the recovery process of TBI patients. Above, a physical therapist works with a patient to help improve her ability to walk.

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.

# 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.**

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Certain precautions, such as wearing a helmet when riding a bicycle or motorcycle and wearing a seatbelt when driving or riding in a car, greatly reduce the risk of TBI.

\*From the CDC, Department of Health and Human Services (<http://www.cdc.gov/safeusa/home/tbi.htm>).

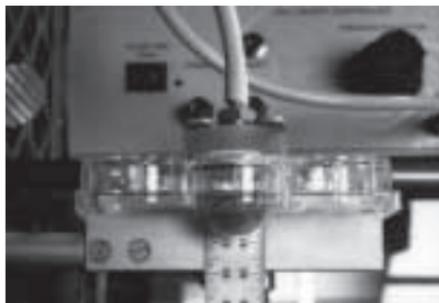
- **Avoid falls by**
  - using a step-stool with a grab bar to reach objects on high shelves;
  - installing handrails on stairways;
  - installing window guards to keep young children from falling out of open windows;
  - using safety gates at the top and bottom of stairs when young children are around.
- **Make sure the surface on your child’s playground is made of shock-absorbing material (e.g., hardwood mulch, sand).**

## What Research is the NINDS Conducting?

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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 interven-

tions 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.



NINDS grantees developed a device that simulates the condition of trauma in cells in culture. The device allows researchers to control the degree of injury to the cells and then study the biological and physiological responses those cells have to trauma. The device can also serve as a tool to test the effects of pharmaceutical agents.

**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 cyclosporin 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.



A researcher studies an image generated by an immunofluorescent microscope to understand cell death in the hippocampus, the section of the brain that controls memory. The hippocampus is frequently affected in TBI patients. This technology allows the researchers to study what happens to injured cells at the molecular level, and to look for ways to interrupt the chain of chemical events that causes permanent brain damage after a severe head injury.

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.

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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 misconceptions 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 misconceptions.

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

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*“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.”*

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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.

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.

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### **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 Celcius 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.

## Where Can I Find More Information?

The National Institute of Neurological Disorders and Stroke is the Federal government's leading supporter of biomedical research on brain and nervous system disorders, including TBI. The NINDS conducts research in its own laboratories at the National Institutes of Health in Bethesda, Maryland, and supports research at institutions nationwide. The address for the Institute, as well as information on other organizations that offer various services to those affected by TBI, is provided on the Information Resources card enclosed in the back pocket of this brochure. Information on the NINDS and its research programs is also available on the World Wide Web at *www.ninds.nih.gov*.

## 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.

***hypermetabolism*** — 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 non-invasive 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.

***Wernicke's aphasia*** — see fluent aphasia.

## Information Resources

The National Institute of Neurological Disorders and Stroke, a component of the National Institutes of Health, is the leading federal supporter of research on brain and nervous system disorders. The Institute also sponsors an active public information office. For information on head injury or other neurological disorders, contact the Institute's Brain Resources and Information Network (BRAIN) at:

### **BRAIN**

P.O. Box 5801  
Bethesda, MD 20824  
301-496-5751  
800-352-9424  
*www.ninds.nih.gov*

In addition, a number of private organizations offer a variety of services and information that can help those affected by TBI. They include:

### **Acoustic Neuroma Association**

600 Peachtree Parkway  
Suite 108  
Cumming, GA 30041  
*www.anausa.org*  
770-205-8211

### **Brain Injury Association**

105 North Alfred Street  
Alexandria, VA 22314  
*www.biausa.org*  
703-236-6000  
800-444-6443

### **Brain Trauma Foundation**

523 East 72nd Street  
8th Floor  
New York, NY 10021  
*www.braintrauma.org*  
212-772-0608

**Family Caregiver Alliance**

690 Market Street

Suite 600

San Francisco, CA 94104

*www.caregiver.org*

415-434-3388 (in California: 800-445-8106)

**National Rehabilitation Information  
Center (NARIC)**

1010 Wayne Avenue

Suite 800

Silver Spring, MD 20910-5633

*www.naric.com*

301-562-2400

800-346-2742

**National Stroke Association**

9707 East Easter Lane

Englewood, CO 80112-3747

*www.stroke.org*

303-649-9299

800-STROKES (787-6537)

**National Institute on Disability  
and Rehabilitation Research (NIDRR)**

600 Independence Avenue, S.W.

Washington, DC 20013-1492

*www.ed.gov/offices/OSERS/NIDRR*

202-205-8134

**“Traumatic Brain Injury: Hope Through Research,”  
2002, NINDS**

## Head Injury Research Centers: August 2002

To find better ways to prevent and treat head injury, the NINDS research program supports a broad spectrum of studies by investigators at leading biomedical research institutions across the country. Information on research activities at these centers may be obtained by contacting the principal investigators listed below.

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