

Chapter 60

Brain-Injured Patients

Clark Watts, MD, JD, FCLM, and
T. Walter Harrell, PhD, ABPP

Primary Causes of Brain Impairment: Diagnosis
Traumatic Brain Impairment

Legal Considerations

When referencing the brain, the general term *injury* should be considered in its broadest context. The brain is considered injured when it sustains pathology from whatever cause. Although this is the context in which the term will be used in this chapter, the primary focus will be on the traumatically brain-injured patient because most of the medicolegal implications of brain injury apply to this group of patients.¹

It is very important for the legal practitioner to understand how the physician will arrive at a diagnosis in these patients, through the process of creating a differential diagnosis. Equally important for the legal practitioner representing brain-injured patients is an understanding of how the brain recovers, and how the injury and the recovery are quantitated. Of additional importance to the legal practitioner is an awareness of obstacles to coverage of brain injuries.

PRIMARY CAUSES OF BRAIN IMPAIRMENT: DIAGNOSIS

General Considerations

Prior to any discussion of the differential diagnosis of primary causes of brain impairment, it is helpful to understand how one arrives at a differential diagnosis. A differential diagnosis is simply a listing, usually by probabilities, but often without mathematical designations, of diseases that it is reasonable to consider in a person suffering from brain impairment. The process of arriving at the differential diagnosis is a relatively simple one, but often poorly understood. It begins, as with all contacts between physicians and patients, with a history and elicitation of signs and symptoms from the patient and a physical examination. This is followed by a correlation of the signs and symptoms with the anatomy and physiology of the portion of the brain that seems to relate to those signs and symptoms. The process of time over which the signs and symptoms have been present is factored in, and the most likely disease categories, based on general pathology, are then extracted from the process. Confirmation of the conclusions at this point is obtained by laboratory tests and, finally, a differential diagnosis of specific pathology is created.

The Process

Signs and Symptoms

Symptoms are those complaints the patient presents to the physician. *Signs* are those findings the physician elicits by physical examination. In eliciting the signs and symptoms of a patient with suspected brain disease, it is important to keep in mind that the brain could express itself in response to disease in only a few ways. The brain may respond to disease by an alteration of the *mental status* of the patient. Usually, the alteration of mental status is in the level of consciousness. The patient may appear conscious and be awake and alert, lethargic, or obtunded. Or the patient may present in or deteriorate into an unconscious state. An important subset of the mental status examination is a search for any derangements of intellect, orientation, self-awareness, or memory.

The patient with impaired brain function may present with *motor* symptomatology. Most patients with this group of signs and symptoms will be noted to have certain patterns of paresis, or weakness of muscle function. Some will present, however, without significant weakness, but instead will have abnormal movements created by disorders of the nervous system such as spasticity or seizure disorders. The muscles may be flaccid, unusually rigid, or uncoordinated in action. The abnormal movements may be noted during voluntary or involuntary activity.

Pain, such as headache, is the most common form of *sensory* complaint. The patient may also complain of abnormal sensation, as with parasthesia, or electric-like painful phenomena, or numbness, the presence of dulled sensation. The complaint may be present spontaneously, or only when the physician, in examining the patient, obtains an admission of the symptom. Other sensory complaints may involve visual or hearing difficulties.

Disturbances of *language* are common with brain impairment. Language disorders can be categorized in several ways, but generally they can be placed into three separate groups, called aphasias. In expressive aphasia the person has, as the name implies, trouble expressing her or himself, that is, trouble making coherent, understandable sentences. The person suffering from receptive aphasia has difficulty receiving communication input and processing it into meaningful language. The verbal expressions of these

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individuals may appear normal, even quite articulate, but they have no relationship to input received. The person with global aphasia has elements of both and, in the worst cases, may be mute. In a traumatically induced brain injury, the patient may present with what is referred to as cognitive language impairment; typically manifested by problems with verbal fluency, semantic or phonemic paraphasias, and/or word-finding difficulties, oftentimes in the context of normal conversational speech.

Finally, afflictions of the brain may also reveal associated mental disorder, including signs and symptoms concerned with emotions, disturbances of reality, memory problems, complex ideational dyspraxia, and/or alterations of self-image.

It is rare that the patient will present with a single group of symptoms; more often there will be a constellation of symptoms. For example, a patient presenting with injury involving the left side of the brain may complain of lethargy, headaches, numbness and weakness of the right arm and leg, blindness in certain portions of the field of vision, expressive aphasia, and depression.

Considerations of Anatomy/Physiology

The brain consists of four parts, which are connected anatomically and physiologically. The largest mass of the brain is the *cerebrum*, composed of the two lateral cerebral hemispheres. Each has a frontal lobe anteriorly, a parietal and a temporal lobe laterally, and an occipital lobe posteriorly. The two frontal lobes in association with structures connecting the two cerebral hemispheres in the midline (portions of the limbic “lobe”) are functionally related to personality, emotion, and self-image. The posterior aspects of each frontal lobe provide voluntary motor function for the opposite side of the body, whereas the anterior aspects of both parietal lobes provide conscious sensory function to the opposite side of the body. Brain auditory function is served by temporal lobes, as is memory when the temporal lobes are interacting with the frontal lobes. In most individuals, voluntary and conscious speech function is located in the left frontotemporoparietal area of the left cerebral hemisphere, whereas visual-spatial orientation function is lateralized to the right cerebral hemisphere, particularly the right parietal lobe.

The second element of the brain is the *cerebellum*, located posterior, beneath the cerebrum. This paired structure is responsible, primarily, for involuntary actions of coordination.

Descending down from the middle of the base of the paired cerebral hemispheres, passing anterior to the cerebellum on its way into the spinal canal where it continues as the spinal cord, is the *brainstem*, the third part. It serves as a major pathway for nervous impulses to leave the brain and enter the spinal cord, and to pass from the spinal cord to the brain.

Finally, there are the *cranial nerves*, which pass from the various other elements of the brain to structures peripheral to the skull. They conduct impulses to the brain that provide the senses of vision, smell, taste, and hearing; the voluntary functions of the face, such as mastication and

sensation; and certain automatic functions of the body, such as rhythmicity of the heart and autonomic bowel function.

An example of the importance of considerations regarding localization can be seen in the patient who complains of visual disturbances. If that patient were also complaining of weakness in the left hand, one would consider a lesion in the right cerebral hemisphere that is affecting the nerve fibers of vision as they pass from the eyes in front to the occipital lobe in the posterior aspects of the cerebral hemisphere where vision is recognized. On the other hand, if the patient with visual disturbances were also complaining of problems with smell or taste, one might look more anteriorly, to the region of the eyes, where the eyes are more closely associated with nasal and oral mucosa from which taste emanates.

Considerations of Time

In arriving at a differential diagnosis, one must not only consider the patient's signs and symptoms and anatomical/physiological correlations, but one must also factor in the time course over which the symptoms and the signs are present. For example, the patient may very well have a headache precipitated by a minor episode of head trauma. The headache would come on suddenly coincident with the trauma and persist appropriately. A headache similar in intensity and location, however, may have gradually developed over several weeks or months in a patient with a brain tumor. Likewise, a brain tumor may cause hand weakness progressively and slowly over several months, whereas a stroke, secondary to cerebral vascular embolization, may cause a sudden onset of hand weakness.

The Differential Diagnosis

After the physician works through the process of analysis in considering the patient's presenting signs and symptoms, the anatomical and physiological localization of the suspected lesion, the time course for the development and presentation of the suspected lesion, and the general pathological nature of the suspected lesion, the etiology of the brain impairment may preliminarily be placed into one or more categories of diseases from which a more specific differential diagnosis may be extracted. Below, definitions and examples of the major categories of neurological diseases will be presented. Then, the traumatic diseases will be categorized in order to illustrate the process of developing a specific differential diagnosis.

Brain impairment may occur as a result of disease categorized as follows: genetic, congenital/developmental, degenerative/metabolic, infectious, traumatic, neoplastic, vascular, immunological, psychogenic, and idiopathic. As with any arbitrary classification, overlapping of categories may occur, as will be apparent in the following discussion.

Genetic Disorders

Most of the primary diseases of the brain associated with genetic disorders are characterized by an underlying error of metabolism. To understand this concept, it is helpful to

look at one of the earliest recognized and best understood primary brain disorders produced by a genetic abnormality, phenylketonuria. This disorder, untreated, is seen primarily in children and is highlighted by mental retardation, seizures, and imperfect hair pigmentation, and is transmitted as an autosomal recessive condition. Due to a well-defined genetic disorder, the gene necessary for the activation of an enzyme, phenylalanine hydroxylase, is disturbed and the enzyme is almost completely lacking. As a result, the normal conversion of phenylalanine to tyrosine does not occur. Instead, phenylalanine is converted to phenylpyruvic acid, phenylacetic acid, and phenylacetylglutamine. With the accumulation of these metabolites in the brain, there is interference with normal maturation of the brain, neurofibers within the brain are not properly myelinated (a process of normal insulation), and other widespread and diffuse anomalies develop. Fortunately, in children born with this disorder, urine and blood levels of phenylalanine rise in the first few days and weeks of life and can be detected by a simple screening test.

Congenital/Developmental Disorders

In general, congenital/developmental disorders are those created by a deleterious effect of the environment, either in utero or following birth, upon the developing brain. Some years ago, a number of the genetic disorders were placed in this category. However, as specific abnormalities in the genome have been identified, the corresponding disorders have been removed from this category. The term cerebral palsy refers to a general condition caused by a number of different environmental insults to the developing brain. While its most common presentation is a spastic weakness of all four extremities, some children may experience mental retardation and seizure disorders. The characteristic of this type of congenital disorder is that it is not progressive, although it may appear to be so as the child grows and becomes progressively more disabled in comparison with his or her peers. The etiological insult may occur before birth, in the perinatal period, or in the first few years of life. Cerebral palsy is believed to be caused by any number of insults including abnormal implantation of the ovum, maternal diseases, threatened but aborted miscarriages, external toxins, or metabolic insults such as maternal alcohol ingestion.

Degenerative/Metabolic Disorders

The category of diseases termed degenerative/metabolic disorders is usually reserved for those conditions that develop in individuals with previously normal brain development. It is appropriate today to exclude conditions with known genetic bases, even though they express themselves later in life, such as Huntington's chorea, or conditions that are congenital or developmental and, as noted previously, appear to progress as the affected individual is compared with developing peers. Alzheimer's disease was at one time believed to be a classic condition in this category. Individuals in the prime of their senior years develop dementia associated with specific neuropathological changes in the brain of unknown etiology. The dementia occurs much earlier than would be expected based simply on senility. Aside from Alzheimer's

disease, the most studied degenerative/metabolic disease of the nervous system is Parkinsonism. This condition is characterized by a progressive uncontrollable tremor with an associated dementia. The motor disability created by the tremor often progresses much more rapidly than the dementia, so that patients, well aware of their limitations, suffer substantial depression. For some reason, certain cells within the brain are unable to manufacture an appropriate amount of the agent, dopamine, which is metabolically necessary for cell function. The etiology of the condition in most patients today is not at all clear. A syndrome identical to Parkinsonism has been described in a group of drug abusers who have used *n*-methyl-4-phenyl-1,2,3,6-tetrahydropyridine, both intravenously and by inhalation. In addition, a Parkinson-like presentation is seen in neuropsychiatric patients who develop a condition known as tardive dyskinesia in response to use of antipsychotic medication.

Infectious Diseases

Most known infectious agents have been reported to cause infections of the brain. Meningitis is a term used to refer to infections of the coverings of the brain, whereas encephalitis is used to refer to an infection of the substance of the brain. In addition to the generalized widespread infectious processes these terms suggest, localized brain infections, or abscesses, can also occur. This category of disease lends itself to a simplified discussion of how the physician might use the earlier presented scheme of analysis. A patient who has the fairly rapid development of brain impairment associated with a fever might be considered to have a disease within this category. If widespread impairment ensues that is characterized by nonfocal deficits and suppression of the mental status of the patient, one might consider meningitis or encephalitis. If, however, the disease process appears to be focal in nature, resulting in a partial paralysis (e.g., hemiparesis or weakness on one side of the body), one might consider the presence of a more focal infectious process such as a brain abscess.

Traumatic Disorders

The traumatic category of diseases encompasses everything associated with acute brain trauma. This includes not only diseases caused by disruption of brain tissue, but also diseases caused by systemic illnesses secondary to the traumatic episode, whether or not this trauma directly involves the head. For example, not uncommonly, following trauma to the head, the patient experiences a period of apnea, or diminished respiratory effort. If this is not corrected quickly, the patient may suffer hypoxia, or a lack of oxygen, which can damage brain cells. A more comprehensive discussion of traumatic brain disease appears later in this chapter to provide more details of the application of principles for defining a specific differential diagnosis of primary brain impairment.

Neoplastic Disorders

The category of neoplastic diseases contains all tumors that are progressive in development, whether benign or malignant. It includes those tumors that arise primarily within

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the brain and those that metastasize to the brain from extracranial sites. As suggested by the foregoing comments, it is traditional to describe tumors as benign or malignant. A benign tumor is one that grows more slowly, does not extend beyond the confines of the tumor mass itself, and does not metastasize or spread through the vascular system. A malignant tumor is a more aggressive tumor. It has a shorter time course and may spread to other parts of the brain or the body by way of the vascular stream. The malignant tumor characteristically results in death in a shorter period of time than the benign tumor. However, this concept may be deceiving in that a histologically benign tumor, placed in a critical location within the brain, may cause death quicker than a malignant tumor placed within the brain in a location that is not as critical. All tumors cause impairment by one of two mechanisms. They may produce direct pressure on the surrounding brain. Additionally, they develop a volume that cannot be accommodated safely within the fixed cranial vault. Consequently, generalized increased intracranial pressure occurs, which adversely affects the flow of blood to sensitive areas of the brain not directly contiguous with the mass itself.

Vascular Disorders

Most diseases in the vascular category affect the blood vessels directly or indirectly. Primarily congenital or developmental conditions, such as aneurysms and arteriovenous malformations, can produce sudden brain impairment by hemorrhage. Arteriosclerosis of the vessels, a degenerative/metabolic condition, may cause sudden impairment by creating an occlusion of the vessels, causing death of tissue from lack of circulating oxygen and nutrients. Occlusions may also occur with embolization of cerebral vessels by arteriosclerotic debris from other sites such as diseased heart valves.

Immunological Disorders

Immunological diseases are caused by disturbances of the immune system. Multiple sclerosis is such a condition. It is characterized by repeated and progressive bouts of demyelination of nerve cells and their axons, extensions of nerve cells that connect with other cells. These extensions ordinarily contain an insulating material called myelin. As a result of disturbances in the immune system not completely understood, the myelin is recognized by the body as a foreign substance and is placed under lymphocytic attack and destruction. Presentation of the patient will depend on the area of the brain affected, with virtually any combination of signs and symptoms possible.

Psychogenic Disorders

The category of psychogenic diseases refers to those recognized and characterized as diseases of the mind associated with personality disorders, disturbances of emotion, and problems of self-image that may or may not be related to one of the preceding categories. Certainly, patients may be depressed as a result of head trauma or disabling conditions such as Parkinsonism.

Iatrogenic and Idiopathic Disorders

Iatrogenic diseases are those produced as a result of treatment by the physician. A patient who develops a blood clot following surgery for a brain tumor due to inadequate hemostasis by the surgeon has developed an iatrogenic hemorrhage.

Idiopathic diseases are those for which there is no known, or reasonably suspected, etiology. As a result of dramatic recent advances in the neurosciences, especially in neuroimaging, these are now few in number.

TRAUMATIC BRAIN IMPAIRMENT

The Diagnosis

There are at least 15 states that have a statutory definition of traumatic brain injury. A definition is also written into federal law that describes the disability for educational purposes. A traumatic brain injury is damage to the brain caused by an external force impinging upon the head and brain. The Traumatic Brain Injury Model Systems (a group of 17 national centers funded by the National Institute on Disability and Rehabilitation Research (NIDRR)), have established diagnostic criteria for traumatic brain injury relative to level of consciousness.² A person is said to have suffered a traumatic brain injury if there was documented loss of consciousness, amnesia for any period of time, and the Glasgow Coma Scale (GCS) was less than 15 during the initial 24 hours following the trauma. The GCS is used by medical personnel to determine the depth of coma. Scale scores range from 3 to 15, with 3 being the poorest score. It measures the best eye, verbal, and motor response to command or stimulation. A score of 13 or better is indicative of a mild brain injury, 9 to 12 is considered moderate, and 8 or below reflects a severe brain injury.³

The diagnosis of traumatic brain disease usually begins with the identification of a traumatic episode resulting in either blunt or penetrating head injury.⁴ A traumatic brain injury is further subdivided into "closed" versus "open" head injury. Closed refers to a nonpenetrating force, while open refers to the skull being penetrated. Penetrating head injuries generally produce less of a problem in the differential diagnosis because the penetrating object (usually a bullet) will produce primary brain disruption and hemorrhage. More challenging is the establishment of a differential diagnosis of traumatic brain disease following blunt trauma.

During blunt trauma, the brain is subjected to forces secondary to acute acceleration and deceleration of the brain within the skull, which is itself undergoing acute acceleration and deceleration. As the result of these forces, a number of pathological processes ensue. The brain may be "stunned" by relatively minor head injury without any anatomical or pathological changes, producing the so-called "concussion." Renewed interest in this condition has occurred because of the exquisite detail of neuroimaging created by MRI. Some believe that through this modality previously unrecognized changes in the limbic lobe, the medial temporal lobe, and the upper brainstem may occur in concussion, accounting for the characteristic findings of

transient loss of consciousness, some degree of retrograde amnesia, and difficulty with mental energy (e.g., lack of motivation), which may exist for weeks or months following the injury. Blood vessels, including both arteries and veins, may be torn, resulting in hemorrhage. This hemorrhage may occur exterior to the brain or within the brain substance. Some portions of the brain may move through greater distances than other portions of the brain, creating shearing injuries at the interface of these moving areas—not too dissimilar from the activity at the fault line during an earthquake. Brain tissue may be disrupted. Mentioned earlier is the fact that, following some severe head injuries, apnea, or loss of normal respiration, may ensue, resulting in hypoxia and other metabolic changes that cause direct injury to nerve cells.

With both CT scanning and MRI scanning, intracranial hemorrhages following head trauma are easily identified. Epidural hemorrhages are arterial in nature and are located beneath the skull but external to the most outer membrane lining the brain, the dura mater. These hemorrhages are usually associated with a skull fracture that lacerates an artery lying between the dura mater and the skull. The hemorrhage may develop rapidly over a period of 2 to 3 hours, creating increased intracranial pressure and focal pressure on the brain. Recovery is excellent in patients who are operated on with evacuation of the hematoma prior to developing coma, whereas the prognosis is extremely poor in someone who develops coma prior to surgery.

The subdural hematoma forms beneath the dura mater but external to the arachnoid membrane, which is the intermediate covering of the brain. The blood usually comes from torn veins, and develops more slowly than an epidural hematoma. It is less well localized and is often associated with other injuries to the brain because the force required to tear veins is actually greater than the force required to cut an artery following a skull fracture, leading to an epidural hematoma. As the result of the more widespread brain injury that is associated with an acute subdural hematoma, the mortality rate for subdural hematomas is higher than that for acute epidural hematomas in that more patients with acute subdural hematomas are comatose at the time of surgery. Often associated with acute subdural hematomas are intracerebral hematomas, blood clots within the substance of the brain. Although rarely an indication for surgery, their presence does adversely affect prognosis.

Subarachnoid hemorrhage (SAH) occurs between the arachnoid membrane and the surface of the brain (the pia mater). It is rarely focal in presentation and may occur with minor head injuries. While SAH, in and of itself, rarely produces primary brain impairment, it may be associated with the development, days or weeks later, of hydrocephalus, which is caused by the excessive accumulation of cerebral spinal fluid that has been normally produced within the brain but is unable to be absorbed normally because of the presence of blood in the subarachnoid space.

Focal injuries produced by shearing forces within the brain are rarely severe. However, if they are widespread

throughout the brain, they can produce significant brain impairment, which is treatable only through the provision of primary support to the patient during the recovery and rehabilitation process. Prognosis of the patient with this condition depends on the location and how widespread the lesions are.

Hypoxia and other adverse metabolic stresses suffered by the brain in the posttraumatic period are a major cause of death or residual disability. When brain cells are subject to these stresses, they may continue to function relatively normally (or lack of function cannot be detected clinically); they may die and the patient will suffer permanent deficit; or they may live but not function normally. It will require time for cells to rejuvenate to recover from the insult, and to begin functioning again.

Posttraumatic Epilepsy

A discussion of head injuries is not complete without some mention of posttraumatic epilepsy. The condition is due to the creation of hyperexcitable areas in the brain by the underlying disorder, or the removal of the normal inhibition of excitable brain by the disorder. Although epilepsy may be focal in presentation, it is such a generalized nonspecific response to trauma that it has little value in distinguishing the underlying brain pathology.

The Treatment

As alluded to previously, treatment⁵ of the brain-injured patient begins with the establishment of a differential diagnosis: a general differential diagnosis based upon the history and physical and a more specific differential diagnosis based upon laboratory studies, including brain imaging. The general principles for the treatment of brain-injured patients are relatively uniform regardless of the etiology of the injury.

Treatment of the initial injury may require the surgical débridement of skull fractures and brain lacerations or the evacuation of hematomas. The causes of secondary injury fall into two general categories: loss of vital metabolic substrates, and compression. Failure to adequately oxygenate the patient or to maintain adequate blood pressure will result in poor delivery of oxygen, glucose, and other essential nutrients to the brain, which will result in further cell injury and death. Local compression by bone fragments or hematomas may cause direct injury to nervous system tissue, or may impede the flow of blood to nervous system tissue. Secondary injury may result in cerebral edema, or the excessive accumulation of fluid both in injured cells and in the interstitial space between the cells. Due to the nondistensible nature of the skull, this may lead to increase in intracranial pressure which may further cause direct brain injury or injury secondary to the interference of cerebral blood flow.

It is important in certain patients, particularly those who are unconscious from head injury or who have evidence of hematomas or cerebral edema on imaging studies, to have the intracranial pressure monitored through the use of various surgically implantable intracranial

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monitoring devices. A medication useful in the treatment of cerebral edema is intravenous mannitol, which will remove interstitial edema in the brain.

Other medications may be useful in the treatment of the brain-injured patient. Antibiotics may be helpful, especially if the brain has been contaminated by an open wound. There is some evidence that the prophylactic use of anticonvulsants during the first week after a disruptive brain injury may reduce the rate of seizure activity following trauma. While at one time, steroids such as Decadron were routinely used in patients with traumatic brain injury, this drug is no longer recommended for this use.

Brain Recovery

At one time it was believed that brain cells either functioned or did not function—an all-or-none phenomenon.⁶ Increasingly, it is becoming obvious that such cells may function at various levels of activity, depending on influences from surrounding cells.⁷ This is an explanation for oftentimes dramatic changes that are observed during the initial phase of recovery. During the initial period of recovery, more and more brain cells move from the idling state to the active state. As they do, they exert their influence on surrounding cells, increasing the activity of the cell pool and thus improving the neurological status of the patient. In addition to spontaneous neurological recovery, a secondary mechanism for improvement is that of relearning. A patient's "weakness" may improve because of the increase in activity of the cell pool as mentioned earlier, or as the result of more efficient utilization of the existing cell pool through rehabilitation efforts. It is believed, with regard to the first phenomenon, that 90% of the ultimate recovery of neurological function will be seen within the first 6 months following injury and the remaining 10% will be seen in the next 1.5 years. The time frame for the second phenomenon is related to learning and developing, and thus can theoretically occur over a lifetime.

A major factor in the rehabilitation of brain-injured patients is the management of the patient's behavioral and emotional state. The frontal lobe structures that are involved in emotional and behavioral control or regulation are oftentimes injured as a result of trauma. As the brain-injured patient recovers and becomes aware of his or her limitations, or attempts to adjust to a disabled body and mind, emotional consequences are common. The combination of brain-based emotional dyscontrol and adjustment difficulties or reactive dysphoria makes clinical management extremely difficult, especially in light of altered mental abilities and memory problems. The World Health Organization describes a health condition with respect to the body, the individual, and society.⁸ They describe the impairment as the body condition (in our discussion the brain injury). The disability is the expression of the impairment with respect to the body (e.g., posttraumatic seizure disorder, hemiparesis, etc.). Finally, the handicapping condition reflects how the disability impacts social or environmental penetration; it is the social definition of the disability. These distinctions are useful in describing

how a person recovers from a brain injury and the types of residual sequelae that are anticipated or are the focus of rehabilitation efforts.

Rehabilitation is typically phased according to the need of the patient and their level of cognitive functioning. Once the patient is medically stabilized, they can begin to receive rehabilitation. The patient's ability to participate in treatment is typically determined by an assessment of cognitive functioning. A commonly used scale is the Rancho Los Amigos-Revised, which focuses on the quality of response a person can make and the degree of assistance they require due to impaired mental functioning.⁹ The scale goes from Level I, which reflects a generalized (often delayed) response and total assistance, to Level X, reflecting appropriate responses and modified independence. The patient's capacity to participate will determine whether they are in need of subacute or acute medical rehabilitation, postacute or transitional rehabilitation, and/or long-term supported living. These stages of rehabilitation are distinguished by their focus (from disability to handicapping condition) and independence in basic and more higher-level activities of daily living (ADLs). Most individuals who suffer from a severe traumatic brain injury will require some degree of support for the remainder of their life due to persistent neuropsychological deficits.

Neuropsychology is a specialization in psychology that focuses on brain-behavior relationships. Its counterpart in neurology is behavioral neurology. In addition to working with patients who have a known or suspected disease or injury to the brain, neuropsychologists evaluate patients to determine the nature of their disability and handicapping condition following brain trauma. The history of neuropsychology dates to the 1940s when soldiers returning from war were noted to perform in the normal range on standardized intelligence tests but were severely disabled from a cognitive standpoint. There was a call to apply psychometric theory to the measurement of these brain-based changes. Before the advent of the remarkable imaging techniques that we enjoy today, neuropsychological testing was used to determine the area of the brain that had been injured. Today, the localization of lesions by neuropsychological evaluation is considered anachronistic, and neuropsychological testing is predominately used to determine the nature and extent of a disability and to translate the results into a useful prescription for care or rehabilitation effort. Typically, a neuropsychological evaluation will consist of standardized observations of intelligence, cognition, memory, language, psychomotor and sensoriperceptual functioning, and behavioral and emotional competence. The neuropsychologist will then describe any deficits or pathology with respect to the known injury to the brain as evidenced by the patient's history and neuroimaging studies. Brain impairment can be seen in the absence of identifiable lesions on imaging studies, but typically the results would be considered to reflect premorbid functioning in the absence of a history of altered consciousness indicated in the medical record or in retrospective questioning of the patient.

Neuropsychological testing is useful but with a note of caution. Although some believe that, through careful

neuropsychological testing, it is possible to quantitate neuropsychological abnormalities in patients with no deficits on neurological examination and neuroimage evaluation, others believe that adequate research has not been conducted to establish standards for such distinctions.¹⁰ In evaluating these matters, the lack of correlation between neuropsychological testing and neuroimaging confirmation of underlying residual brain impairment may indicate preinjury evidence of neuropsychological abnormalities. Neuropsychological test results stand on their own merits, but any change in functioning that is being asserted should be made on the basis of the known medical history and indirect evidence of neuropsychological functioning, such as school grades, community/vocational achievement, and mentor/work evaluations.

LEGAL CONSIDERATIONS

In general, the legal considerations for the brain-injured patient vary little from those generally present in common and statutory law related to personal injury torts and contracts. Issues of informed consent generally concern the incompetent. These issues are relevant when considering a brain injury survivor's capacity to consent to care, contract for services (including legal), etc. In most cases, a temporary or permanent guardianship will be required due to the survivor's persistent neuropsychological deficits. Increasingly, patients, particularly the elderly, have created advance directives, either a durable power of attorney or a living will, which may come into play if they remain vegetative or minimally responsive. The laws related to these matters are generally state-specific.¹¹ The exception is the Patient Self-Determination Act of 1990, a federal law mandating that hospitals that receive federal funds must inform patients of their right to create advance directives and to have them followed.¹²

The question often arises as to how to handle a matter of termination of treatment to include termination of life support in patients who do not have advance directives. This is of particular note in situations where the patient is in the persistent vegetative state with no hope of recovery and, potentially, years of survival. The U.S. Supreme Court in *Cruzan v. Director* held that, although a competent adult has a right to terminate treatment, the state may establish the standard of proof in matters involving the incompetent patient such as one in the persistent vegetative state.¹³ In *Cruzan* the Supreme Court upheld the state of Missouri's requirement that the proof be "clear and convincing."

A recent and much discussed case was that of Terri Schiavo, a young woman who became in a persistent vegetative case after cardiac arrest. She had no advanced directives, and a major dispute developed between her husband and her parents over her interests regarding artificial nutrition and hydration. Before resolution of the case, the executive, legislative, and judicial branches of both Florida and the United States Government became involved and the case became shamelessly politicized.¹⁴

Most distressing and costly in terms of dollars and emotional capital is the patient in a persistent comatose state.

While a number of these states have been defined, the one with the most recent exhaustive study is the persistent vegetative state.¹⁵ This patient is unconscious; the patient is not aware of his environment nor can he or she react appropriately to that environment. There is a restitution of primitive brainstem responses such as a normal sleep-wake cycle, facial grimacing (including smiling), and eye movement ("doll's eye" in response to movement in the room). To the lay person, these behaviors appear to reflect conscious awareness and obvious responses to environmental stimuli. The diagnosis of persistent vegetative state typically is not made in the absence of serial imaging studies over time that clearly show deterioration of neural substrate and the lack of volitional response in the patient. This diagnosis is distinguished from the "minimally responsive" patient who is capable of demonstrating volitional responses to stimuli, but who has severe physical and mental disabilities that limit their responsiveness.

Of major concern to head-injured patients and their families is denial of coverage under health insurance policies.¹⁶ Often the portion of the policy that relates to chronic care or rehabilitation is ambiguously worded. As can be anticipated from the previous discussion, there are no bright lines between acute care, rehabilitation, and custodial care.¹⁷ These terms and phrases are often self-defined after the fact by adjusters to provide denial. It is important that legal practitioners help medical practitioners understand the legal implications of conclusions such as "no further medical required" or "medically stabilized." It is ironic that a spinal cord patient is provided with durable medical goods and equipment and medical and therapeutic services with no expectation that they will be fully independent or walk again, yet a brain injury survivor, who has injured the same organ system, is relegated to the status of "custodial care" to deny coverage for the types of support services they need as a result of their injury. A severe brain injury will necessitate some degree of support for the remainder of the individual's lifetime due to reduced cognitive, behavioral, and emotional competence. A severe brain injury requires planning for a lifetime. The costs associated with attendant or supervisory support needed to keep the survivor safe dwarf all other financial considerations. In the absence of a diagnosis of persistent vegetative state, there are no authoritative data upon which to reduce life expectancy, a consideration when evaluating need and managing resources for a lifetime. A lifetime of need will likely require the establishment of a permanent guardianship, medical special needs trust, and application for and establishment of public benefits.

Endnotes

1. A number of excellent treatises are available to which the reader may refer to expand the knowledge of the material in this chapter. Especially recommended are (a) R.K. Narayan, J.E. Wilberger, Jr. & J.T. Povlishock, *Neurotrauma* (McGraw-Hill, New York, 1996), (b) G.T. Tindall, P.R. Cooper & D.L. Barrow (eds.), *The Practice of Neurosurgery*, 3 vols. (Williams & Wilkins, Baltimore, 1996), and (c) L.P. Roland (ed.), *Merritt's Textbook of Neurology*, 9th ed. (Williams & Wilkins, Baltimore, 1995).

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2. See <http://www.tbims.org/combi/> (accessed December 2005).
3. See <http://www.trauma.org> (accessed December 2005) for discussion, also texts cited *supra*, note 1.
4. Space does not permit a discussion of the special circumstances surrounding the diagnosis and treatment of brain injury in the neonate and the very young child. A comprehensive review of this subject may be found in A. Towbin, *Brain Damage in the Newborn and its Neurological Sequels: Pathologic and Clinical Correlation* (PRM, Danvers, MA, 1998).
5. In addition to the texts referenced (*supra* note 1), the legal practitioner might wish to review *Guidelines for the Management of Severe Head Injury* published by the American Association of Neurological Surgeons, Chicago; telephone: 708-692-9500.
6. For a comprehensive review of this subject, especially of the role that rehabilitation plays, see the report of the NIH Consensus Development Panel on Rehabilitation of Persons with Traumatic Brain Injury, *Rehabilitation of Persons with Traumatic Brain Injury*, 282 J.A.M.A. 974 (1999).
7. P. Bach-y-Rita, *Recovery from Brain Damage*, 6 J. Neuro. Rehab. 191-199 (1992).
8. For a full description, see their website, <http://www.who.int/en/> (accessed December 2005).
9. There are a number of scales that are used to measure outcomes or to track changes in response to rehabilitation efforts following traumatic brain injury. Consult <http://www.trauma.org>, <http://calder.med.miami.edu/pointis/tbipro/NEUROPSYCHOLOGY/func.html> (both accessed December 2005), or *supra* note 2 for a general description.
10. As evidenced by the conflicting positions contained in the following references, it behooves any lawyer representing clients with brain injuries to become familiar with the subject matter of neuropsychological testing: (a) M.D. Lezak, *Neuropsychological Assessment*, 3d ed. (Oxford University Press, New York, 1995), (b) G.P. Prigatano, *Principles of Neuropsychological Rehabilitation* (Oxford University Press, New York, 1999).
11. See A.D. Liberson, *Advance Medical Directives* (Clark, Broadman, Callaghan, New York, 1992). See also Chapter 23, this volume.
12. Omnibus Budget Reconciliation Act of 1990, Pub. L. 101-508, §4206, 4751.
13. *Cruzan v. Director, Missouri Dept. of Health*, 110 S.Ct. 2841 (1990).
14. See L.O. Gostin, *Ethics, the Constitution, and the Dying Process: The Case of Theresa Marie Schiavo*, 293 J.A.M.A. 2403-2407 (2005). (This presents is a good summary of the various executive, legislative, and judicial actions in this case).
15. See the publications by the Multi-Speciality Task Force, *Medical Aspects of the Persistent Vegetative State*, 330 N. Engl. J. Med. 1499, 1572 (1994).
16. See, e.g., S. McMath, *Insurance Denial for Head and Spinal Cord Injuries: Stacked Deck Requires Health Care Reform*, 10 HealthSpan 7-11, 1993 (July/Aug.), and C. Rocchio, *Social Security Continued Disability Review Requires Action*, 2 TBI Challenge 4 (1998), published by the Brain Injury Association.
17. See *Anderson v. Blue Cross/Blue Shield*, 907 F. 2d 1072 (11th Cir. 1990).