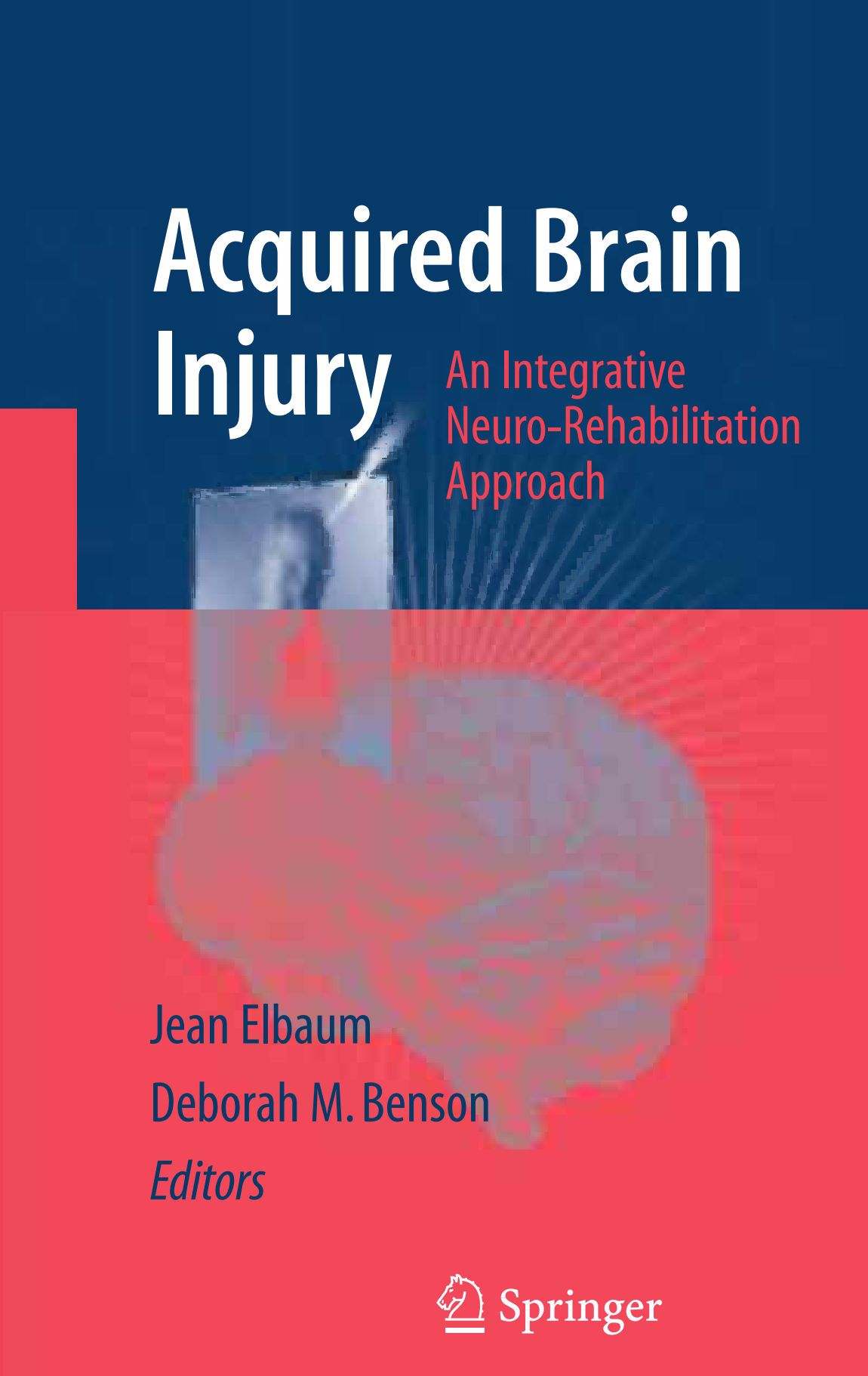
Acquired Brain Injury

An Integrative Neuro-Rehabilitation Approach



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Introduction

JEAN ELBAUM AND DEBORAH M. BENSON

Crimmins (2000) marveled at the greatness of the "three pound-blob" that is our brain and control system. As seasoned clinicians in the field of neuro-rehabilitation, we still marvel each day at the resilience of the brain and at the exciting recoveries that we attempt to facilitate in survivors of acquired brain injuries (ABIs). We observe the survivor who used to have frequent and severe behavioral outbursts each hour now remain calm and focused throughout the day. We note the survivor who once was a major safety risk due to lack of insight now act as our ally motivating other survivors by his experiences. We see survivors who were admitted to our rehabilitation program with a multitude of challenges, broken and vulnerable, discharged each week to productive, meaningful activities, competent and compensating for their residual weaknesses.

On the other hand, we've encountered a disillusioning number of situations in which distraught survivors and family members find themselves in crisis, sometimes years after the injury. The survivor with a preexisting psychiatric illness, that goes undiagnosed and untreated after his brain injury, resulting in psychiatric hospitalization for a suicide attempt a few years after discharge from acute rehabilitation. The woman with chronic pain that prevents her from returning to work, despite the significant gains she demonstrated in physical and cognitive functioning during her rehabilitation stay. The bright college student whose mild brain injury went unrecognized, who never received rehabilitative services, and whose premature return to school resulted in failure, depression, and the onset of substance abuse.

From both the successes and failures of our rehabilitation efforts, we have learned that the best way to achieve positive outcomes for our clients and families is by ensuring a comprehensive, integrated approach; one which spans the continuum of care, allowing us to support our survivors and families from the earliest stages of recovery, throughout their rehabilitation and beyond.

We have become highly aware of the value of, and need for, such a team approach to neuro-rehabilitation; including both highly trained specialists (e.g., the neuro-urologist, neuro-optometrist), as well as holistically oriented coordinators (e.g., case managers, discharge planners), who will assume very different, yet interwoven, roles in the rehabilitation of the individual post-ABI. While the benefits

of this comprehensive approach may be apparent, the challenges of ensuring coordination and integration of care across each of these components/specialists are significant. The survivor and family must know that their care is being coordinated as well as the purpose and function of each of their care providers. Equally important, all rehabilitation team members must be knowledgeable about the different roles of their interdisciplinary colleagues, and maintain open communication that crosses multidisciplinary borders.

Thus, the goal of this text is to provide an introduction to many of the key members of the neuro-rehabilitation team, including their roles, approaches to evaluation, and treatment. The book was written for interdisciplinary students of neuro-rehabilitation as well as practicing clinicians interested in developing their knowledge of other discipline areas. It may also be of interest to survivors, caregivers, and advocates for persons with acquired brain injury, to help explain and unravel the mysteries and complexities of the rehabilitation maze. Case examples were included in each chapter to help illustrate real life challenges. Dimancescu (Chapter 2) describes the role of the neurosurgeon in treating clients post acquired brain injuries and highlights the importance of providing educational information to families to help reduce feelings of confusion and powerlessness. Rosenberg, Simantov, and Patel (Chapter 3) and Duarte and Fishman (Chapter 4) describe the central roles of physiatry and neurology in diagnosing and treating clients post ABI. They highlight the importance of team collaboration and discuss topics such as neuroplasticity, spasticity management, medical complications, headaches, seizures, and sleep disorders. Aprile and Reilly (Chapter 9) review the specific challenges of the neuro-rehabilitation nurse in addressing the needs of the individual recovering from brain injury. Kearney et al. (Chapter 12) and Kramer, Shein and Napolitano (Chapter 13) discuss the essential roles of the occupational therapist, and the speech/language pathologist on the neuro-rehabilitation team. Megna (Chapter 11) reviews the importance of conducting vestibular evaluations for clients with dizziness and balance difficulties post-ABI, so that appropriate treatment can be rendered. Karlovsky and Badlani's chapter on neuro-urology (Chapter 5) involves a review of the common urological and sexual difficulties post-ABI as well as treatment strategies. Han (Chapter 8) describes common visual difficulties post-ABI and the role of the neuro-optometrist. Scicutella (Chapter 6), Benson and Pavol (Chapter 7), and Elbaum (Chapter 14) discuss the emotional, behavioral, and cognitive challenges of clients post-ABI and the importance of addressing these difficulties through an integration of counseling, neuro-cognitive intervention, and proper medication management. The specific challenges of families and ways to meet their needs effectively through appropriate interventions are reviewed in a separate chapter (Chapter 15). Finally, Tovell (Chapter 10) reviews the key role of the case manager in coordinating the complex and varied aspects of treatment for individuals with ABI. The text ends with a discussion of life after neuro-rehabilitation, including long-term challenges for clients and factors that influence outcome.

We wish to thank, above all, the many survivors and families, whose hard work, perseverance, and resilience serves as a continual source of inspiration to us, as

well as a reminder of how we must continue to strive to improve our services and supports, not only as rehabilitation professionals, but as a community and society, for survivors of brain injury and their families. We also would like to thank our professional colleagues, whose passion, enthusiasm, and devotion to the field of neuro-rehabilitation allow us to continue to push ourselves as a team, and raise the bar in order to provide the best care we can offer. And we offer thanks to our administrative support staff, who rarely get the credit for our successes and achievements, but whose "behind the scenes" efforts are the glue that holds the complex structure of our programs together.

Reference

Crimmins, C. (2000) *Where is the Mango Princess?* New York: Vintage Books.

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Neurosurgery and Acquired Brain Injury
An Educational Primer

MIHAI D. DIMANCESCU

Introduction

Injuries of the nervous system are particularly frightening to clients and families because of the many unknowns that still revolve around nervous system function, and because of the potential for resulting life-long disabilities or functional deficits. Recovery from brain injury is best achieved with the full participation of the patient and/or his or her family. To this end, each patient and involved family member needs to have an understanding of basic brain anatomy, physiology and pathology, as well as recuperative abilities, expressed as clearly as possible in understandable language. Because the organization of the brain is extremely complex and since an understanding of the brain and types of possible injuries is not part of our elementary, high-school, or even college education, teaching the patient and family is an ongoing process throughout treatment and rehabilitation. It behooves the neurosurgeon to provide as much of that education as possible during the acute care period of time, and to prepare the patient and family for the rehabilitation process during which the therapists will continue to provide education. The latter phase should also include preparation for re-integration into the community or for long-term care.

The nervous system consists of the brain, the spinal cord, and the peripheral nerves. While the neurosurgeon is usually involved in the care of any part of the nervous system, this chapter will address only injuries of the brain. The basic information required by an injured individual and/or his family to understand the injury, its implications and its treatment will be introduced in the following pages.

Anatomy

The **brain** is a soft mass weighing about two and a half pounds, fairly tightly packed in a three-layered skin known as the **meninges** (Truex & Carpenter, 1971). The inner or **pial** layer is translucent and is firmly adherent to the brain. Over the pia, the middle or **arachnoid** layer is extremely thin and is separated from the pia by a narrow space containing a clear colorless fluid called **cerebrospinal fluid**

(CSF). The outermost layer is the **dura mater**, thick and tough, easily separable from the arachnoid, with several folds to be identified later (Truex & Carpenter, 1971). The brain and its coverings are contained in a hard, closed box, the **skull**. The only opening out of the skull is at the skull base where the brain connects with the spinal cord through the **foramen magnum** (large opening) (Truex & Carpenter, 1971). If a brain is removed from the skull and the outer layer of meninges—the dura—is peeled off, the brain surface is noted to have multiple folds or **convolutions** and grooves or **sulci** coming together in a large mushroom like structure sitting on a narrow stalk—**the brain stem**. The large mushroom-like portion has **two halves**, the left brain and the right brain, separated by a deep groove at the bottom of which is a bridge of brain connecting the two halves. A fold of the dura extends down the groove and is called the **falx**. The main body of the brain is separated from a lower smaller portion of the brain—**the cerebellum**—located just behind the brain stem. Another fold of the dura called the **tentorium** separates the two parts of the brain (Brodal, 1969; Standing, 2005; Truex & Carpenter, 1971). The brain shares its space inside the skull with blood vessels—**arteries and veins**—and with the cerebrospinal fluid (CSF). A normal brain contains 140 to 170 cc (4.7 to 5.7 oz) of CSF manufactured in four almost slit-like cavities in the brain called **ventricles**. The brain produces approximately one cupful of fluid every 24 hours. The entire structure—brain, meninges, blood vessels, cerebrospinal fluid, and skull—is perched at the very top of the spinal column (Rouviere et al., 1962; Standing, 2005; Watson, 1995).

The basic anatomical functional unit of the brain is the **neuron**. Billions of neurons are located in several layers near the surface of the brain. This is the **gray matter**. Other neurons are packed in clusters deep in the brain, called **basal ganglia**. Each active neuron has about 80,000 connections with neurons around it. The connections occur at microscopic contact points known as **synapses**. Longer connections between the neurons and deeper parts of the brain travel in bundles through the **white matter** (Dimancescu, 2000; Standing, 2005; Truex & Carpenter, 1971). At the subcellular level, each neuron contains multiple structures that manufacture chemicals and provide energy. Around the neurons are trillions of smaller support cells—the **glial cells** (Brodal, 1969). With special staining techniques in the laboratory, these structures can be seen under a microscope and constitute the cellular anatomy of the brain.

Physiology

The brain has autonomic, sensory, motor, and cognitive functions. In very simple terms, autonomic functions are located deep in the brain, in the **midbrain** and in the **brain stem**; sensory functions in the back parts of the brain, **occipital**, **parietal**, and **posterior temporal** lobes; motor function in the **frontal** lobes; and cognitive functions, including memory, concentration, and emotions are more diffusely represented, requiring integration of both sensory and motor functions of the brain (Dimancescu, 1986, 2000; Rouviere et al., 1962). The cerebellum,

or hindbrain, is mainly involved in coordination and modulation of movement as well as balance. The right brain controls the left side of the body and the left brain controls the right side. Right-handed individuals are left-brain dominant. Speech centers are mostly in the left brain.

To function smoothly, **sensory** information has to be provided to the brain. Sensations include smell, vision, taste, hearing, and tactile senses. The tactile senses include light touch, pressure, temperature, vibration, and pain. Other sensations are sent to the brain from sensors providing information related to joint positions or to the various organs in the body. The sensations travel to the brain along sensory nerves, into the spinal cord and up to the brain (Victor & Roper, 2001; Wilkins & Rengachay, 1996). To avoid a chaotic bombardment of information into the brain, a wonderful apparatus exists in the brain stem called the **reticular system**. Its function is to filter sensory information as it enters the brain and to allow through only that information required by the brain at any given moment (Rouviere et al., 1962; Wilkins & Rengachay, 1996).

The neurons receiving sensory information integrate the data and initiate transmission of information to the **motor** parts of the brain that trigger an appropriate movement or series of movements. Such movements may be very gross, including movements of the trunk, shoulder or hips or may be very fine movements such as writing, playing a musical instrument, eye movements, or talking. The smoothness or accuracy of each movement is dependent on the quality of the sensory information received (if there is no feeling in a hand and the eyes are blindfolded, it will be impossible to write or to find an object on a table) and on appropriate modulation by the cerebellum to avoid over- or undershooting. Certain parts of the brain are able to learn patterns of movement such as picking up glass and pouring water from a pitcher, complex athletic movements, and playing musical instruments. Thus, a command can be given for a complex patterned movement without having to break the movement down into its components (Andrews, 2005; Victor & Roper, 2001; Wilkins & Rengachay, 1996). The healthy brain has the capacity to process enormous amounts of sensory information and to provide a very large variety of motor responses or activities.

Autonomic functions of the brain emanate from deep brain and brain stem areas. Classified in this category are blood pressure, heart rate, breathing and digestive functions. Their deep location makes them the best protected of the many brain functions (Brodal, 1969; Rouviere et al., 1962; Wilkins & Rengachay, 1996).

The most complex function of the brain and the one that distinguishes humans from all other living creatures is **cognitive function**. Cognition is the ability to be aware of oneself and of one's condition, to concentrate, to analyze and to synthesize information consciously, to imagine and to create, to remember and to retrieve memories. Memories are stored throughout the brain in many neurons and are thus visual or olfactory, tactile or auditory, motor or emotional, or different combinations. Memories can be simple, such as a single smell, or complex, such as a whole series of events. One memory can trigger another. While it is recognized that memory is stored in neurons in the form of proteins, and that some memories are for short-term periods and other memories are long term, the process

of memory retrieval still remains mysterious. How the brain is able to use given information to create new information or ideas is also unknown (Brodal, 1969; Truex & Carpenter, 1971; Victor & Roper, 2001).

Most of us are familiar with computers and in many ways the brain functions like a computer—a very complex computer that human inventiveness has not yet been able to match. Each neuron is like a computer microchip, with all the microchips able to communicate with each other, but without any input into the computer, there is no output. Furthermore, the input has to be appropriate: wrong information in, equals wrong information out. The output is triggered by an event—with computers the event is the touch of a key or of several keys. However, a computer functions electronically. The brain functions through a combination of chemical reactions and electrical impulses triggered by chemical changes, too complex for further explanation here (Dimancescu, 1986, 2000; Guyton & Hall, 2006).

For a computer to function, a source of energy is needed—electricity. Energy for the brain comes from oxygen. Oxygen is the brain's fuel, brought to the neurons by the flow of blood. No oxygen is stored in the brain; consequently a good flow of blood is required for the brain to receive the needed oxygen to provoke the appropriate chemical reactions. In addition good nutrition is necessary to supply the building blocks of the tissues and to supply the basic chemicals that allow the various chemical reactions to take place. An appropriate balance of proteins, fats, sugars, minerals, and vitamins is needed to assure a healthy functioning brain (Dimancescu, 1986, 2000; Guyton & Hall, 2006).

The anatomical and physiological overview described represents a summary of the extremely complex brain anatomy and physiology. It is hoped that the information provided is sufficient to understand some of the basics of what happens to the brain when an injury occurs.

Injuries to the Brain

The two most common mechanisms of injury to the brain are the application of a mechanical force or the interruption of a normal supply of oxygen. Occasionally the two mechanisms occur together.

Mechanical Force Injuries

Blows to the head are the type of force most commonly associated with brain injury. The simplest of these may be a simple bump on the head on an overhead cabinet, or a punch to the head, accidentally or intentionally. Other times the blow may be forceful, as in a fall striking the head against the ground, or hitting one's head against a tree while skiing, falling off a bicycle, a skateboard or rollerblades, or being struck by a falling object such as a tree branch, a brick, or an overhead fixture. Greater forces are transmitted to the brain in hammer, crowbar, poolstick, or lead-pipe attacks, or in automobile, motorcycle, or motorboat accidents or any accident where speed is involved and a rapid deceleration occurs. All of these

types of injuries may occur in the home, the workplace or during travel and may be commonplace or recognized work hazards. Some are criminal in nature, others are related to negligence or carelessness and still others are unavoidable (Dimancescu, 1979, 1995, 2000). Yet another type of mechanical force injury includes penetrating injuries such as a bullet wound, shards of metal, axe or pick wounds, harpoon injuries, imbedded bone fragments or wounds caused by any other hard object that penetrates the brain (Dimancescu, 2000; Rowland, 2005a).

Occasionally mechanical force is applied to the brain from within, without any external blows being exerted against the head. Such forces occur with spontaneous hemorrhages (bleeding) into or around the brain. Such hemorrhages may result from a ruptured aneurysm, a weak spot on an artery around the base of the brain, or from a ruptured arterio-venous malformation—an abnormal tangle of weak arteries and veins. Other times bleeding may occur into a brain tumor or may result from the use of blood-thinning medications (aspirin, warfarin, plavix) that can also worsen any bleeding resulting from a blow to the head. Bleeding into the brain as a result of high blood pressure is not uncommon (Dimancescu, 1995).

The type of injury suffered by the brain after a blow to the head or following a spontaneous hemorrhage depends in part on the degree of injury, the location or locations of the injuries, and the site and size of the hemorrhage. Associated factors intervene as well in determining the effect of the injury, such as age, coexisting disease or illness, nutritional state, fitness, medications, illicit drugs, and injuries to other parts of the body such as might occur in a serious motor vehicle accident or in a fall from a great height.

The least serious injury to the brain resulting from a blow to the head is a **concussion**, defined as a brief period of loss of consciousness lasting a few minutes following which there may or may not be a period of memory loss (amnesia), and with no brain abnormalities noted on any diagnostic testing (Rowland, 2005a,b; Victor & Roper, 2001). A more serious injury is the **cerebral contusion** or hemorrhagic contusion, usually associated with a brief loss of consciousness, frequently accompanied by some weakness of an arm or a leg on one side of the body or by mental changes such as poor attention span and sometimes speech difficulties, all of which are usually, though not always, temporary. Diagnostic tests show areas of bruising of the brain (Rowland, 2005a,b; Victor & Roper, 2001). Some injuries to the brain consist of brain swelling or **edema** without any noted bruises or hemorrhages. The edema may be short-lived or prolonged, very focal or diffuse, and associated with minimal deficits or with serious brain dysfunction such as coma. More serious brain injuries cause bleeding or hemorrhages that may occur in different locations defined by the anatomy and the relationship of the hemorrhages to the three layers of the meninges covering of the brain.

Working from the surface down into the depths of the brain, the most superficial hemorrhage is an **epidural hematoma**, located between the skull and the dura or outermost meningeal layer. An epidural hematoma results from a ruptured vein or artery. The latter are usually more serious because the higher blood pressure in an artery causes more bleeding and a larger clot. Epidural hematomas in the temporal region (just above and in front of the ear) can be lethal, causing sudden death, one to

two hours after an injury (Rowland, 2005a). A hemorrhage between the next two layers is a **subdural hematoma**, in the space between the dura and the arachnoid. Acute subdural hematomas have a very high mortality rate, usually because of the size of the blood clot that covers a large surface, compressing the brain and accompanying underlying injury of the brain itself. Occasionally an *acute subdural hematoma* may be silent, without any clinical signs, but over a period of one to two months, liquefies, increases in size and becomes a *chronic subdural hematoma* with mild to severe signs and symptoms. Occasionally subdural hematomas occur without any known blow to the head in individuals taking blood-thinning medications (Rowland, 2005a). Bleeding into the third space of the meninges, under the arachnoid layer, is a **subarachnoid hemorrhage**. In this type of bleeding, the blood spreads through the cerebrospinal fluid and insinuates into the grooves of the brain. The most serious of these types of hemorrhages are not from blows to the head, but from a spontaneous rupture of a weak spot *(aneurysm)* on a blood vessel. They may or may not be serious and may or may not have devastating consequences, but all spontaneous subarachnoid hemorrhages are potentially lethal (Rowland, 2005a). Any hemorrhage into the meat of the brain is known as an **intracerebral** or **intraparenchymal hemorrhage**. These blood clots may be deep or superficial, may be large or small, may be near or removed from vital structures, may be relatively inconsequential or devastating, frequently leaving an individual with long-term signs and symptoms. They may result from blows to the head or may result from hypertension *(high blood pressure)* (Rowland, 2005a). The final location of a hemorrhage may be in one or more of the ventricles, the narrow cavities of the brain that manufacture cerebrospinal fluid. These are **intra-ventricular hemorrhages**, represented by a few drops of blood in the CSF or by massive bleeding, casting the ventricles and impeding the flow of CSF (Rowland, 2005a). Each of the described injuries may occur in isolation or in combination.

Interruption of Oxygen Supply

The brain does not store any oxygen, yet it is totally dependent on oxygen to function. If the brain is totally deprived of oxygen for two minutes, the brain dies. Many situations occur where oxygen is deprived to parts of the brain *(focal anoxia)* or where the oxygen supply is diminished but not totally cut off *(hypoxia).* Focal anoxia or hypoxia may occur without mechanical blows to the head but are frequently associated with mechanical force injuries. Conversely, spontaneous occurrences also frequently result in internal mechanical force injuries (Dimancescu, 1996). Spontaneous situations accompany arteriosclerosis (hardening of the arteries) with narrowing of the arterial openings and decreased blood flow to areas of the brain *(ischemic stroke).* Atheromatous plaques on the carotid artery sometimes result in formation of clots that travel into the arteries of the brain and shut down the blood supply and oxygen supply to a specific area of the brain. That is commonly known as a **stroke** (or *embolic stroke)* (Dimancescu, 1996). Clots forming on abnormal heart valves may also travel into the arteries of the brain causing embolic strokes. Heart attacks requiring prolonged resuscitation efforts result in very

weak blood flow to the brain during the resuscitation process, seriously decreasing the amount of oxygen delivered to the brain. The net result is a diffuse decrease in oxygen supply affecting the entire brain *(diffuse hypoxia)* (Dimancescu, 1996). Strangulation, suffocation, near drowning, and smoke inhalation all deprive the lungs of breathed-in oxygen, thereby decreasing the amount of oxygen in the blood and causing a diffuse hypoxia (Rowland, 2005a; Truex & Carpenter, 1971). Focal hypoxia and diffuse hypoxia result in chemical changes that in turn result in edema or brain swelling. Swelling compresses brain cells and small blood vessels feeding the brain, adding a mechanical compressive component to the hypoxic or anoxic component of the injury (Rowland, 2005a; Truex & Carpenter, 1971). Conversely, primary mechanical injuries to the brain cause compression of blood vessels surrounding the blood clot or hemorrhage, resulting in a focal decrease of blood supply or oxygen to that area of the brain. A vicious cycle is frequently initiated compounding the initial effect of the injury and explaining why some individuals progressively worsen during the days following a blow to the head. As bleeding or swelling increases, pressure in the skull increases (Dimancescu, 2000). Ischemic and embolic strokes are not traditionally associated with brain injuries but the end result is the same—the brain is injured and its functions are impaired.

Signs and Symptoms of Brain Injury

Brain injury causes signs and symptoms related to levels of consciousness, breathing, vital signs, pupillary function, motor function, sensory function, and autonomic function.

1. *Levels of consciousness* change with increasing degrees of brain injury or with increasing pressure within the skull. A normal individual is considered to be alert, but as consciousness becomes impaired, the individual becomes *lethargic,* then *obtunded*, then *stuporous,* and finally *comatose,* in a light, moderate, or deep coma (Dimancescu, 2000; Victor & Roper, 2001).
2. *Breathing* also changes with increasing intracranial pressure. One of the first signs of increased intracranial pressure is *hyperventilation,* a rapid breathing rate representing the brain's effort to blow off CO_2 and thereby cause constriction of blood vessels and a decrease in the volume of blood in the head. As the condition worsens, the breathing pattern changes to one of regularly increasing amplitude of each breath followed by a progressively decreasing amplitude in repeating cycles; this is known as *Cheyne–Stokes breathing.* The next phase, called *Kussmaul breathing,* is ominous, indicating impairment of brain stem function and consists of an inspiration followed by a pause then an expiration followed by a pause and this cycle repeats itself. An even more ominous respiratory pattern is *agonal breathing*, in which very irregular breaths are followed by pauses of varying lengths (Rowland, 2005a; Victor & Roper, 2001).
3. *Vital signs* consisting of blood pressure and heart rate are modified by increased intracranial pressure, with a decreasing heart rate and an increasing blood pressure

noted. This is known as the *Cushing response* (Rowland, 2005a,b; Victor & Roper, 2001).

4. *The pupils* of the eyes are always examined following brain injury. *Dilatation* of one pupil that does not constrict when a bright light is shined in the eye is an indication of increased pressure on the same side of the brain as the dilated pupil. When both pupils are dilated and fixed to light stimulation, increased intracranial pressure is bilateral, secondary to bleeding or to swelling affecting both right and left hemispheres of the brain (Rowland, 2005a,b; Victor & Roper, 2001).

5. *Changes in motor function* follow a similar progression reflecting a worsening condition, starting with weakness, then paralysis on one side of the body, opposite the side of the brain injury. Weakness or paralysis of both sides reflects bilateral brain injury. As pressure in the skull increases, abnormal reflexive movements develop, known as *decorticate* or *decerebrate* posturing. In the former, either spontaneously or to stimulation, the arm flexes over the chest and the hand turns inward. In the latter the arm extends stiffly by the side, inwardly rotated. In both conditions, the leg extends stiffly with the foot and toes pointing downward. In some patients, seizures or convulsions represent an irritation of the surface of the brain as a result of the injury sustained (Rowland, 2005c; Victor & Roper, 2001).

6. *Sensory function* is the least reliable parameter to observe since it can only be fully assessed in an alert and cooperative individual; therefore, other than responses to pain in an injured person with an altered level of consciousness, sensory function is not very helpful in determining the degree of injury to the brain (Rowland, 2005c; Victor & Roper, 2001).

7. *Autonomic function* impairment is usually manifested by a rapid heart rate and profuse sweating (Rowland, 2005a,c; Victor & Roper, 2001).

8. *The Glasgow Coma Scale* is a rapid bedside assessment tool developed 30 years ago by two neurosurgeons (see Table 2.1). The score provides a measure of the severity of the brain injury and enables the nurses and physicians to follow the patient's progress over the days following the injury (Dimancescu, 2000). The

TABLE 2.1. Glasgow Coma Scale*

Best Eye Response (4)	Best Verbal Response (5)	Best Motor Response (6)
1. No eye opening	1. No verbal response	1. No motor response
2. Eye opening to pain	2. Incomprehensible sounds	2. Extension to pain
3. Eye opening to verbal commands	3. Inappropriate words	3. Flexion to pain
4. Eyes open spontaneously	4. Confused	4. Withdrawal from pain
	5. Oriented	5. Localizing pain
		6. Obeys commands

*The GCS is scored between 3 and 15, with 3 being the worst and 15 the best. It is composed of three parameters : Best Eye Response, Best Verbal Response, Best Motor Response, as shown above. Note that the phrase "GCS of 11" is essentially meaningless, and it is important to break the figure down into its components, such as E3V3M5 = GCS 11. A Coma Score of 13 or higher correlates with a mild brain injury, 9 to 12 is a moderate injury, and 8 or less a severe brain injury.

Source: Teasdale G., Jennett B., *Lancet* (ii) 81–83, 1974.

score measures the ability to open the eyes, to vocalize or to speak, and to move the limbs. The scores range from 1 to 4 for eye opening, 1 to 5 for vocalization and speech, and 1 to 6 for the best motor movement, with 1 representing the worst score, i.e., absence of activity. The lowest possible total score is a 3, indicating extremely severe brain injury. The highest score of 15 is near normal. A score of 8 or less indicates coma.

Testing

The initial testing when a brain-injured individual first comes to the emergency room is a bedside examination to assess the ability to breathe and to measure the blood pressure, which indicates adequacy of blood circulation and can detect possible blood loss from associated injuries. Once a good airway has been established (sometimes requiring insertion of a tube and placement on a respirator), blood loss has been controlled (sometimes requiring a transfusion), and blood pressure has been stabilized, a CT scan is performed. This computerized image of the skull and the brain will show whether any contusions or hemorrhages have occurred and where they are located, may show skull fractures if any are present, and will indicate the existence of edema of the brain. Any foreign bodies in the brain will also be visualized. Sometimes, after testing is complete, an intracranial pressure monitor will be inserted by the neurosurgeon through a tiny opening in the skull. If there is clinical, CT scan or intracranial pressure monitoring evidence of increased intracranial pressure, intravenous medications will be started emergently in an attempt to reduce swelling and pressure (Rowland, 2005a; Victor & Roper, 2001; Wilkins & Rengachay, 1996).

Treatment

The role of the neurosurgeon following brain injury is to do everything reasonably possible to *assure the survival* of the individual and to try to minimize the long-term effects of the injury. The neurosurgeon's intervention starts in the emergency room. Based on the bedside examination and the CT scan results, the neurosurgeon will decide if an intracranial pressure monitor needs to be inserted, and what intravenous medications need to be administered to attempt to reduce swelling. In addition, if the individual needs to be on a respirator to support the breathing mechanism, the neurosurgeon may request that the respirator rate be set faster than the normal rate of breathing to blow off CO_2 and further reduce intracranial pressure. Every effort is made from the onset to *decrease pressure in the skull*. If the pressure increases too much, the brain becomes more compressed, causing further injury, and with high intracranial pressure, blood flow to the brain is impaired, the normal blood pressure being insufficient to overcome the increased intracranial pressure. A certain amount of pressure inside the skull is normal for everyone, but if the intracranial volume increases because of a blood clot or because of swelling,