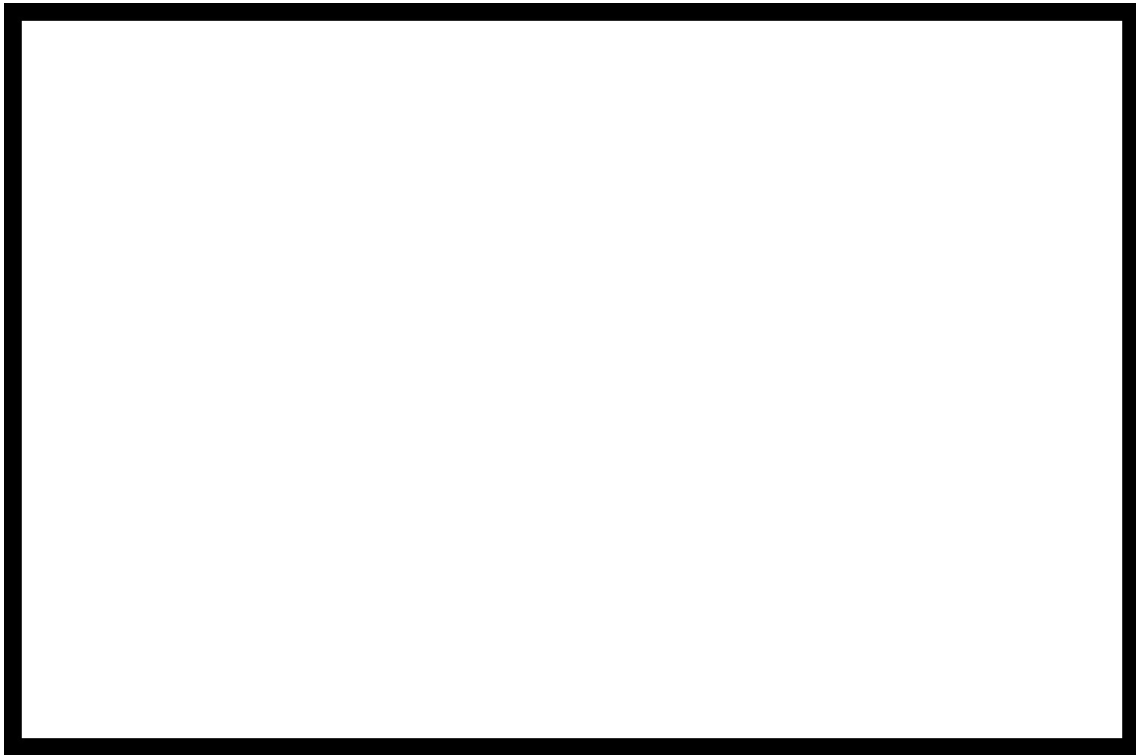


LIVING WITH A BRAIN INJURY

ABI training module I

Version: 5/2024

A rocky road



Instructions and suggested activities for this module:

- Please read and study this module.
- Since this module includes a lot of medical terms, the ABI program director would like to hold a couple of phone conversations with you so that you feel more comfortable with the content. Please contact the ABI program administrator at (801) 809-5391.

Objectives of this module:

- Understand the consequences of brain injury and how it can affect a person.
 - Understand the distinction between acquired brain injury (ABI) and traumatic brain injury (TBI).
 - Understand brain functions.
 - Understand the interactions of behavior, environment, and brain chemistry.
 - Understand the impact of drugs and alcohol
-

Brain injury facts & statistics

Every 9 Seconds

someone in the U.S. a brain injury

More than 3.6 million

people sustain an ABI each year.

At least 2.8 million

people sustain a TBI each year.

Typical causes of ABI Include:

- Electric shock
- Infectious disease
- lightning strike
- oxygen deprivation (hypoxia/anoxia)
- Toxic exposure
- Near drowning
- Seizure disorder
- Tumor
- Substance abuse / overdose
- Stroke

- **AN ACQUIRED BRAIN INJURY (ABI)** is any injury to the brain that is not hereditary, congenital, degenerative, or induced by birth trauma.
- **TRAUMATIC BRAIN INJURY (TBI)**, is type of ABI. A TBI is caused by trauma to the brain from an external force.
- The number of people who sustain TBIs and do not seek treatment is **UNKNOWN**.

FIVE-YEAR OUTCOMES OF PERSONS WITH (TRAUMATIC BRAIN INJURY) TBI:*

Died..... 22%
Became worse..... 30%
Same..... 22%
Improved..... 26%

Data are US population estimates based on the TBIMS National Database.

**Data refer to people 26 years of age and older who received inpatient rehabilitation services for a primary diagnosis of TBI.*

<https://www.cdc.gov/traumaticbraininjury/moderate-severe/index.html>

BRAIN INJURY AS A CHRONIC HEALTH CONDITION:

Research has shown that brain injury can evolve into a lifelong health condition that impairs the brain and other organ systems and may persist or progress over a person's lifetime.

AT LEAST 5.3 MILLION AMERICANS LIVE WITH A TBI-RELATED DISABILITY. THAT'S ONE IN EVERY 60 PEOPLE.

The statistics above are provided by the Brain Injury Association of America. More information about the Brain Injury Association of America can be found on their [website](#).

Understanding brain injury

There are hundreds of different types of brain injuries.

Traumatic brain injury (TBI)

A traumatic brain injury is an injury to the brain, not of degenerative or congenital nature, that is caused by an external physical force that may produce a diminished or altered state of consciousness. The result is an impairment of cognitive abilities or physical functioning. It can also result in the disturbance of behavioral or emotional functioning.

There are two types of *traumatic* brain injuries:

Closed brain injury- This means that the injury occurred without physical penetration of the skull. Although the brain is not directly “touched” during the injury, the brain floats in fluid within the skull, and can bounce and twist within the skull, resulting in bruising and tearing from sharp bone on the inside of the skull. There are a variety of forces that cause damage in a closed brain injury. They include:

- Damage at the site of the blow (coup).
- The brain is compacted by molding of the bone inward.
- Pressure on the brain at the time of the coup may force the brain against the opposite side of the skull, producing additional contusions (counter-coup).
- Movement may cause a twisting or shearing of the nerve fibers, producing microscopic lesions.
- Bruises and strains may produce bleeding (hemorrhage), once the blood is trapped in the skull, this mass exerts pressure on surrounding structures.
- Blows to the head can produce edema (brain swelling), which could cause another source of pressure on the brain.

Open brain injury- This means that due to some type of blunt-force trauma, the skull was fractured and/or penetrated. Examples of this could be a gunshot to the head or having severe fractures due to a motorcycle or automobile accident. No two traumatic brain injuries are the same. This is because for each individual, different areas of the brain are affected based on their particular injury. Other factors that affect the outcome are possible medical complications, the age of the person, current and former health status, and preexisting intellectual and personality characteristics.

Brain damage can result due to the direct or indirect causes noted below:

Primary injury: acceleration-deceleration movement:

The skull is pretty hard and inflexible. The brain is rather soft, like firm Jell-O. When the skull makes a rapid movement, it moves as a solid object. The brain, encased inside the skull, moves at a different rate than the skull because it is soft. In addition, different parts of the brain move at different speeds due to their relative lightness or heaviness. The differential movement of the skull and brain when the head is struck results in direct brain injury from three main causes: Axonal Shearing, Contusion (bruising), and brain swelling (Cerebral Edema).

- 1. Axonal shearing:** When the brain is slammed back and forth inside the skull after a head trauma, it is alternatively compressed and stretched because of its soft, Jello-like structure. The long, fragile axons of the neurons that make up the brain are also compressed and stretched. If the impact is severe enough, axons can be stretched until they are torn. This is called Axonal Shearing. If this occurs, the neuron dies. After a severe traumatic brain injury, there is massive axonal shearing and neuron death. This is a major cause of brain damage after a traumatic brain injury and cannot be directly treated.

2. Brain swelling (Cerebral edema): The brain also swells after a severe trauma, just like any other part of the body. This is a major cause of brain damage after traumatic brain injury. Severe swelling can cause death by compressing the brain stem. Brain swelling can lead to neuron damage by squeezing the cells or from ANOXIA caused by disrupting the flow of blood and oxygen to the brain.

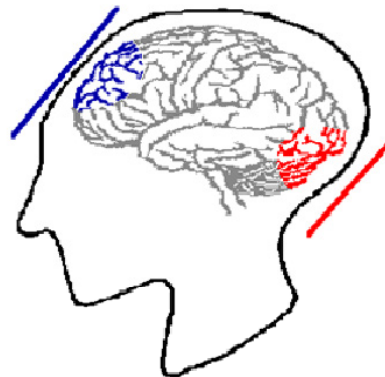
3. Contusion: The brain's violent slamming against the bones of the skull can also result in contusions, bruising, and bleeding. This results in the tearing of small blood vessels. It can also result in the death of neurons and is the second leading cause of neuron death after a traumatic brain injury. Small contusions are usually not treated (concussion) if blood flow is interrupted.

Coup - Contrecoup:

After a traumatic brain injury, contusion can occur anywhere in the brain. Often, most of the contusions will be on the underside of the frontal and temporal lobes because of the irregular and rough texture of the bones underlying these parts of the brain. In addition, specific sites of bruising and contusion can occur at the site of the blow to the head ("coup") and the site directly opposite to where the blow was struck on the head ("contrecoup").

The coup injury is caused when the head is stopped suddenly and the brain rushes forward. It only gets injured by hitting in the side of the skull but is also damaged as it rubs against all the inner ridges.

The contrecoup injury is caused when the brain bounces off the primary surface and impacts against the opposing side of the skull. Again, additional injury occurs as the brain again rubs against all the inner ridges.



Hematomas: If the blood vessels damaged by the impact inside the skull are large enough, they may bleed and create a pool of blood or hematoma. A hematoma can cause brain injury by directly damaging the neurons it comes in contact with or by squeezing neurons through increased pressure in the brain due to its volume. The treatment for a hematoma is to surgically drain it, if possible.

Secondary injury:

Delayed secondary injury at the cellular level is recognized as a major contributor to the ultimate tissue loss that occurs after brain injury. A cascade of physiologic, vascular, and biochemical events is set in motion in injured tissue. This secondary tissue damage is at the root of most of the severe, long-term deficits a person with brain injury may experience. Procedures that minimize this damage can be the difference between recovery to a normal or near-normal condition or permanent disability.

Diffuse blood vessel damage has been increasingly implicated as a major component of brain injury. Depending on the severity of the trauma, early changes include an initial rise in blood pressure, an early loss of the automatic regulation of cerebral blood vessels, and a transient breakdown of the blood-brain barrier. Vascular changes peak at approximately 6 hours post-injury but can persist for as long as 6 days. The clinical significance of these blood vessel changes is still unclear, but may relate to delayed brain swelling that is often seen, especially in younger people.

Much of the ultimate loss in brain function may be caused not by the injury itself, but by an uncontrolled vicious cycle of biochemical events set in motion by the trauma. The control of this complex cascade of cellular events remains one of the most important challenges in the acute management of brain injury. As with diffuse axonal injury, it offers a potential therapeutic window of opportunity during which brain swelling and nerve cell death may be prevented during the first few hours after an injury has been sustained.

Secondary intracranial insults

In the minutes and hours after a brain injury, a variety of other damage may occur:

- Hematoma (epidural, subdural and/or intracerebral)
- Brain swelling/edema
- Increased intracranial pressure
- Cerebral vasospasm
- Intracranial infection
- Epilepsy (seizures)

In addition to primary injury to the brain, traumatic brain injury can also result in two main secondary problems that can cause additional brain injury or complications: seizure disorders and Hydrocephalus.

Seizure disorders: A secondary effect of traumatic brain injury can be a seizure disorder, caused by a specific injury that leads to a disruption in the electrical activity of the brain. Seizure disorders can occur immediately, soon, or much later after a traumatic brain injury. Seizures are usually treated by anticonvulsant drugs.

Hydrocephalus: The flow of cerebral spinal fluid in the ventricles of the brain can sometimes be blocked or disrupted after a traumatic brain injury. When this happens, the fluid being constantly made in the ventricles can accumulate in the brain, causing increased pressure. This problem is called Hydrocephalus and can be a serious secondary effect of a traumatic brain injury. Hydrocephalus is treated by inserting a needle valve into the ventricles to draw off the fluid down a tube into the abdominal cavity. This procedure, called a Ventriculo-Peritoneal Shunt, reduces the pressure inside the brain.

Secondary systemic insults

Secondary systemic insults (outside the brain) that may lead to further damage to the brain are extremely common after brain injuries of all grades of severity, particularly if they are associated with multiple injuries. Thus people with brain injury may have combinations of low blood oxygen, high blood pressure,

heart and lung changes, fever, blood coagulation disorders, and other adverse changes at recurrent intervals in the days following brain injury. These occur when the normal regulatory mechanism by which the cerebrovascular vessels can relax to maintain an adequate supply of oxygen and blood during such adverse events is impaired as a result of the original trauma.

Common forms of secondary systemic insults are listed below:

- Hypoxemia (Low blood oxygen)
- Arterial hypotension (high or low blood pressure)
- Hypercarbia (carbon dioxide accumulation)
- Severe hypocarbia (low carbon dioxide)
- Pyrexia (fever) Hyponatremia (low sodium)
- Anemia (low iron)
- Abnormal blood coagulation Lung changes
- Cardiac (heart) changes
- Nutritional (metabolic) changes

Acquired brain injury (ABI)

An injury to the brain that is not hereditary, congenital, or degenerative. These types of injuries to the brain can occur and are not considered "traumatic." For instance, if someone nearly drowns and loses oxygen to their brain, they may well have the same issues to deal with as someone who has been injured by physical force. However, loss of oxygen (anoxia), is not considered a traumatic brain injury. Non-traumatic injury would also include illnesses such as encephalitis (swelling of the brain), stroke, brain tumors, and high fever.

Acquired brain injury Includes:

Stroke

The brain has the most complex system of blood vessels in the body. Weighing only about three pounds, it requires about twenty percent of the blood flow to keep it alive. Only a few minutes without sufficient blood flow can cause irreversible death of neurons. If the blood flow is blocked through a stroke (also called Cerebrovascular Accident or CVA), cell death in the area deprived of blood will result. If there is bleeding in or over the brain (Hemorrhage or Hematoma) because of a tear in an artery or vein, loss of blood flow and injury to the brain tissue by the blood will also result in brain damage.

Infections

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

Anoxia and hypoxia

If the blood flow is depleted of oxygen, irreversible brain injury from ANOXIA (no oxygen) or HYPOXIA (reduced oxygen) can result in just a few minutes. Anoxia and hypoxia are often caused by heart attacks, respiratory failure, drops in blood pressure, and a low oxygen environment. This type of brain injury can result in severe cognitive and memory deficits.

Metabolic disorders

Brain damage due to metabolic disorders can result when harmful chemicals damage the neurons. Such injury can be caused by being exposed to toxins like insecticides and solvents.

The spectrum of brain injury

Mild brain injury

A mild brain injury, also known as “concussion,” is one in which there is only a brief or momentary loss of consciousness, if any, without any major complications such as a hematoma. Often, people with mild brain injury do not even go to a hospital. However, a relatively subtle amount of reversible brain damage occurs, even after a mild concussion. This is often followed by “post-concussion syndrome” that can include temporary headaches, dizziness, mild mental slowing and fatigue. The most important element in the management of mild brain injury is recognizing that the symptoms are real and can be treated. Symptoms of mild brain injury almost always improve over 1-3 months. Another important element is proper management of the resulting fatigue, with a gradual return to normal activities and/or work over time.

Moderate brain injury

A moderate brain injury results in a loss of consciousness usually lasting only minutes or a few hours followed by a few days or weeks of confusion. It may be accompanied by brain contusions or hematomas. Persons sustaining a moderate brain injury will usually have cognitive and psychosocial impairments that can last for many months. However, with treatment these individuals are generally able to make a nearly complete recovery.

Severe brain injury

Severe brain injury almost always results in prolonged unconsciousness or coma lasting days, weeks or even longer. Persons in a coma appear to be asleep, but cannot be awakened and there is no meaningful response to stimulation. Such persons often have brain contusions, hematomas or damage to the nerve fibers or axons, and some may have suffered from anoxia. Although persons who sustain a severe TBI can make significant improvements in the first year after injury and can continue to improve at a slower pace for many years, they will often be left with some permanent physical, behavioral and/or cognitive impairments.

Consequences of brain injury

No two traumatic brain injuries are the same, just as no two individuals are identical. This is because different areas of the brain are affected with each injury and the effects are multiple. Other factors that affect the outcome are possible complications, the age of the person, current and former health status, and preexisting intellectual and personality characteristics.

Traditionally, the changes a person experiences are divided into three main areas: physical, cognitive (related to thinking), and behavioral/psychological (related to personality and emotions). Some changes are common after a head injury, but it is important to note that they are not universal. Not only do the nature and context of the brain injury differ from one situation to the next, but so do the life experiences, personalities, lifestyles, and personal resources of each individual. Thus, the results of injury can vary dramatically from one person to the next.

Brain function

The brain controls all of the body's functions, from the vital processes of breathing and sensing, to thinking and judgment as well as emotional reactions to everyday events (Swiercinsky, Price, and Leaf, 1993). The brain has three important interconnected areas: the brainstem, the cerebellum, and the cortex.

Brainstem - The brainstem connects the brain with the spinal cord. All information from the body to the brain and from the brain to the body must go through the brainstem. It also controls consciousness, arousal, and vital functions such as breathing, respiration and pulse.

Cerebellum - The cerebellum is attached to the rear of the brain and controls coordination of movement, muscle use, and balance. It is in a more protected area of the brain than the brainstem.

Cortex - The cortex is the largest area of the brain and is where most thinking functions occur. The cortex is divided into right and left hemispheres. For the most part, the left hemisphere is usually dominant and controls verbal functions such as speaking, reading, writing and calculating. The right hemisphere controls functions that are more visual in nature, such as memory, drawing, or copying. In general, left and right hemispheres control opposite sides of the body. Each hemisphere is further divided into four lobes, each of which have specialized functions.

Frontal lobe - (in the front of the brain, behind the forehead) is one important component of the brain's emotional control center. It can be thought of as a "gatekeeper" that controls language, how to solve problems, what to say or not say in social situations, judgment, planning, and impulse control.

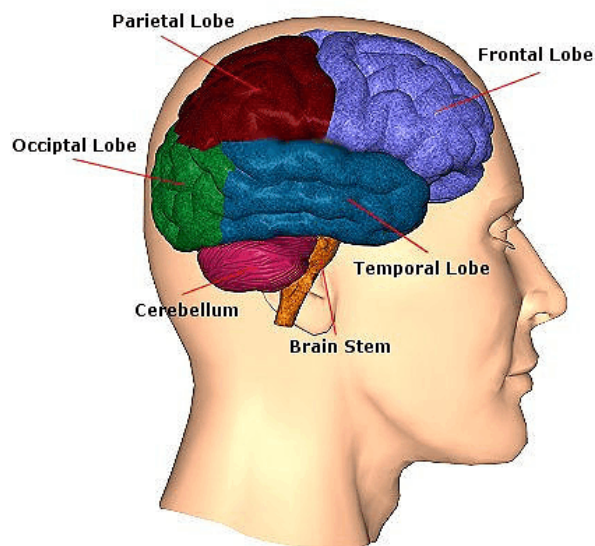
Parietal lobe - (one in each hemisphere) located just behind the brain's mid line. The parietal lobe is an important site for most intellectual activities such as reading, writing, and arithmetic. Things such as making sense out of pictures, diagrams, and reading maps are controlled by the parietal lobes.

Temporal lobes - are located in the mid section of each hemisphere, on either side of the head. Among the functions controlled by these lobes are memory, hearing, language, sequencing, and musical ability. Finally, the occipital lobe is the primary site of visual perception and is located at the very back of the brain.

Reticular formation - contains fibers that enter into or connect with all major parts of the brain. These fibers, or nerve centers, help control muscle tone and complex reflexes. The reticular activating system controls wakefulness and mechanisms allowing us to be alert and react to changes.

Limbic system- which includes the thalamus and the hypothalamus, is literally a system of structures that run along the center of the brain and help mediate both memory and emotional behaviors. The thalamus is a major sensory correlation center. It is a critical "switching" station for processing incoming and outgoing brain messages. Focusing of attention and concentration as well as retrieval of memory information are controlled here. The hypothalamus controls appetite, sexual arousal and thirst.

While different areas of the brain "specialize" to a large degree, they are all connected structurally and functionally in very complex ways. Veins, neurons, nerve fibers and other vital tissues connect each part.



Surface view

Frontal lobe (motor, creativity, emotional reactions)

Parietal lobe (body senses, orientation, visual & spatial perception)

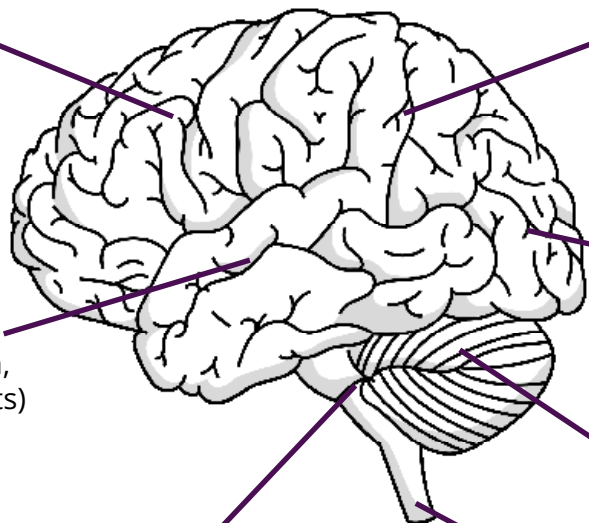
Temporal lobe (hearing, music, understanding speech, memory for non-verbal events)

Occipital lobe (vision)

Cerebellum (Muscular coordination)

Brain stem (regulation of blood pressure, heartbeat, respiration)

Spinal cord



Midline view

Corpus callosum (connection hemisphere)

Limbic system (emotions and learning)

Thalamus (sensory relay)

Hypothalamus

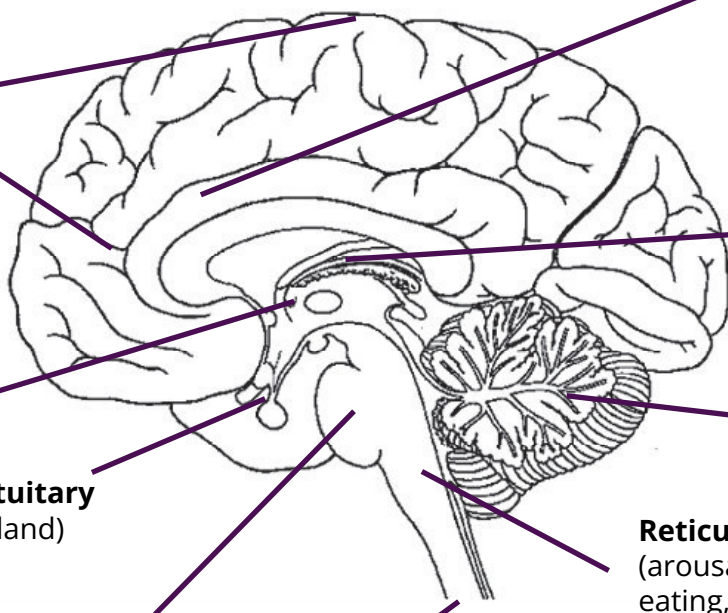
Cerebellum

Pituitary (Gland)

Reticular formation (arousal, consciousness, eating, sleeping patterns, drowsiness and attention)

Brain stem

Spinal cord



Comas & unconsciousness

A coma is a profound or deep state of unconsciousness. The affected individual is alive but is not able to react or respond to life around him/her. Comas may occur as an expected progression or complication of an underlying illness or as a result of an event such as head trauma.

Unconsciousness is a state in which the affected person cannot be roused. It is caused by temporary or permanent damage to brain function. There is reduced activity of the nerve cells and fibers in the part of the brain stem called the reticular formation. The most common causes of unconsciousness are stroke, epilepsy, drug overdose, head injury, cardiac arrest, poisoning, diabetes, and alcohol. There are various levels of unconsciousness. If the person responds to sound and touch, then the state is only light as in a faint. If the level of response is low, then the person is more deeply unconscious.

It is common for a person who has experienced a brain injury to have been in a coma due to the trauma to their brain, or at the very least have had a concussion or a period of unconsciousness. All individuals with traumatic brain injury who are initially in a coma will emerge from the coma. Some people will progress and ultimately have a good recovery. Some will emerge but have significant disabilities, and others will be in what is known as the minimally conscious state or the vegetative state for years. In the vegetative state, people may appear to be awake and may even open their eyes and look about the room, but are otherwise unresponsive. A variety of treatments and techniques may be used to care for these people and prevent complications.

Medical management

Medical management may involve sensory stimulation programs, positioning programs, medications, surgery, nutrition, hygiene, and various other interventions. Professional staff can include physicians, neurologists, surgeons, nurses, and many others. Seizures, hypertension, hydrocephalus, aspiration pneumonia, urinary tract infections, hormonal abnormalities, and skin ulcers are some of the potential problems that a person in a coma may experience.

Medication

Medication might be used to treat seizure disorders, infections, muscle spasticity, hypertension, and swelling, to name only a few of the possible reasons. In some cases, medication might be prescribed that has the potential to increase the coma duration, but decrease the swelling in the brain, therefore decreasing the overall extent of damage to the brain tissue.

Physical results

Many of the physical challenges a person with a brain injury experiences may be evident. Others may be more subtle and present at differing times. Some of the more common physical challenges caused by brain injury include:

Balance and coordination - The ability to use appropriate righting and equilibrium reactions to maintain and upright position. It is usually tested in sitting and standing positions.

Fatigue - Physical or mental weariness resulting from exertion.

Hemiplegia or hemiparesis - Paralysis or weakness of one side of the body as a result of injury to neurons carrying signals to muscles from the motor areas of the brain.

Perceptual losses - The ability to make sense of what one sees, hears, feels, tastes, or smells. Perceptual losses are often very subtle.

Seizure - An uncontrolled discharge of nerve cells that may spread to other cells nearby or throughout the entire brain. It may be associated with loss of consciousness, loss of bowel and bladder control, and tremors. It may also cause aggression or other behavioral change.

Visual field defect - Inability to see objects located in a specific region of the field of view ordinarily received by each eye. Often the blind region includes everything on the right half or left half of the visual field.

Mental health/chemical health - The use of drugs and alcohol after brain injury is a controversial and hotly debated issue. The excitatory or depressing effects of alcohol are more extreme after brain injury. Smaller doses of alcohol yield more powerful effects than before. Additionally, alcohol consumption is known to kill brain cells. People with brain injuries must decide if the additional brain damage is an acceptable risk.

Behavior and cognition

Behavior

Behavior problems are acts that are either dangerous or have a negative effect on an individual's rehabilitation or community reintegration. The brain controls our thought processes and all bodily functions. In short, it controls behavior. When the brain is injured, behavior often is affected. Unfortunately, many changes in behavior following brain injury are not adaptive or acceptable to other people.

Behavior problems can be a difficult hurdle for individuals, as well as their families and friends. Behavior challenges can appear in many forms. Problems with social skills and interactions with other people account for the majority of behavior problems. Other, more severe, behavior problems can also appear following brain injury. Aggression toward others, self-injury, property destruction, tantrums, yelling, and cursing can cause serious concerns for families, friends, teachers, co-workers, and others. Behavior problems can interfere with rehabilitation and be an obstacle to community reintegration.

Many people experience increased agitation for a period of days, weeks, or months following brain injury. This agitated state is a natural result of the damage to the brain and the disruptive effect that it has on a person's general functioning. In many cases, the agitation goes away as the brain learns to function in its newly acquired injured condition. But in some cases, agitation can be a lifelong condition that results in significant behavior problems if not treated.

Damage to the frontal lobe, common in motor vehicle crashes and assaults, can cause specific behavior problems. The frontal lobe controls decision-making, judgment, and other executive functions. Often when the frontal lobe is damaged, an individual has great difficulty tolerating frustration or over stimulation. This can lead to feelings of agitation and, sometimes, to yelling, cursing, aggression, property destruction, and/or other such behaviors.

Approaches can vary greatly because professionals in contrasting disciplines receive different types of training. Even the field of psychology contains many different schools of thought, many with differing theories. Treatment approaches based on one theory can vary in fundamental ways from treatment approaches based on a different theory. Some psychologists believe that changing the way people feel about themselves and their behavior will result in behavior problems not occurring. Others recommend giving individuals an opportunity to discuss their problems and how they might relate to childhood experiences. Still other professionals feel that the best method of treating behavior problems is to place the individual in a positive and supportive environment.

There are no simple, universal solutions to treating behavior problems. Every person with behavior problems is an individual whose situation is unique. Many individuals have occasional inappropriate behaviors that are not dangerous and do not impede their rehabilitation or their ability to function. The best way to identify a behavior problem is to consider an action's severity and how often it occurs. Behavioral and emotional difficulties cannot be separated from the cognitive difficulties that accompany ABI. Ninety-nine times out of 100 when there is a behavioral problem it is tied to a cognitive problem.

Cognition

The definition of cognition is “the mental process or faculty of knowing, including aspects such as awareness, perception, reasoning, language, memory and judgment.” Thus, cognition includes all of the brain’s mental input and output, from basic activities like using language and arithmetic during a trip to the grocery store, to complex decisions like selecting between two job offers, to the creativity of writing a poem or song, to being able to understand things from another person’s perspective and maintain an emotionally intimate relationship with them.

Part of cognition, memory is much more than simply a passive storage system for knowledge. Memory is a set of active processes that encode information. Memory “packages” the information so that it is easier to remember and can be associated with related items already in memory. Memory also involves storing information, which includes constantly re-arranging what is stored so that new knowledge is integrated with what is already in storage, and locating and retrieving information as it is needed. For example, cognition assists memory by helping to identify what is important to remember, thereby freeing you of having to recall everything. A few types of brain injury, such as viral infections deep within the brain, can impair memory without affecting other aspects of cognition. However, in the vast majority of cases, memory impairment is part of a larger cognitive impairment. In fact, many symptoms of brain injury that appear to be memory problems on the surface really are secondary consequences of cognitive deficits. For example, impaired attention and concentration can reduce the amount of information a person takes in, such that even if they have perfect memory, only a portion of the original information will be remembered.

The brain is a person’s organ for thought, emotion and behavior. Injury can disrupt any or all of these brain functions, including the brain’s ability to integrate functions and produce complex behavior.

As you are reading this information, you are using your perceptual abilities to see the printed page, your language abilities to make sense of what you see, your memory encoding and storage so that some of what you read will remain with you afterwards, your concentration to keep all the things going on around you (and inside you) from distracting you, your capacity to form intentions and plans—so that it was you who decided to do this at this time—and your mental flexibility, so that if you smell smoke or hear a baby crying you can re-prioritize your plans and stop reading in order to do something that is more urgent.

Therefore, in asking, “What types of cognitive impairments are associated with brain injury?” The answer is that everything a human being does can be affected by brain injury. While everything can be influenced by brain injury, different diseases and injuries to the brain certainly do produce their own characteristic sets of symptoms. Keep in mind, however, that even though the disease/injury process (i.e., stroke, traumatic brain injury, anoxia, infection, tumor) and the area of the brain that is involved may be known, the ability to predict the types of cognitive and behavioral problems that will result is good, but by no means perfect. The only way to identify reliably the specific impairments of any given person with brain injury is through careful interview and examination of that person and—equally as important—through detailed interviews with the person’s loved ones and care providers.

Cognitive impairments also can affect personality and emotion. For example, having a considerate and empathetic personality requires the ability to think about someone else’s needs, feelings and desires and—at the same time—think and experience one’s own needs, feelings and desires.

Consider a generous, considerate and empathetic person who sustains a brain injury and is left with a deficit in the ability to manage two lines of thought at the same time. This person may appear to have become selfish and self-centered when, in fact, he/she may retain their former considerate personality but not have the cognitive ability to implement their intentions. If you point out an instance of selfishness to the person in the above example, he/she may experience remorse and guilt and truly be puzzled about why they are behaving in ways that are uncharacteristic of them.

The experience of having cognitive impairment in itself is frightening and discouraging and often leads to depression and anxiety. While such reactions can be a source of great pain and despair for the person and his/her loved ones, it is important to keep in mind that they also are positive signs, indicating that the person now has the cognitive ability to be aware of his/her situation.

A common scenario is for the person to be in a good mood early in recovery when self-awareness is still very impaired; and then as self-awareness improves, the individual enters a phase of depression. A competent cognitive treatment program will pay close attention to such emotional issues and will have the capacity to provide psychological and psychiatric treatment as needed.

Some common problem cognitive issues after brain injury include:

Orientation - Individuals with diffuse or bilateral damage are more likely to experience problems with orientation to time, place, and person. External cues—such as calendars, bulletin boards, watches with beepers, pictorial systems for identifying persons and places—and other commonly used objects can be helpful. Orientation difficulties lead to a great deal of confusion for the individual, but consistency and structure in every aspect of life can keep him/her better oriented.

Attention/concentration - Arousal and attention are important for processing information. Attention and concentration skills are needed to support most of the other cognitive functions on this list. For individuals with brain injury, it can be overwhelming to try to maintain attention, make sense of information, integrate it and use it appropriately.

Overload-breakdown of comprehension - Fatigue plays havoc with comprehension. Trying to manage too much information at once inevitably leads to overload. Too much stimulation in the environment (i.e., a noisy classroom, bright lights, blaring music, large crowds of people) can cause overload and shut down for the individual with brain injury.

Reasoning and problem solving - Solving problems—even simple ones—in the course of daily living may be problematic for some individuals with brain injury. A spilled cup of coffee may create a major problem-solving dilemma and result in an odd reaction (e.g., throwing the cup in the trash instead of getting a cloth to mop up the spill). Many poor decisions are made when a person is unable to weigh various options and their effects before determining an action plan. For the individual with brain injury, it is often very difficult to organize and sequence information to solve problems responsibly.

Organizational skills - As injury to frontal lobes of the brain often disrupts organizational skills, individuals may experience many problems in organizing both information and tasks. An external cue may need to identify the problem areas and develop strategies to enhance organization (e.g., organization trays labeled to note the items kept there, cue cards for various activities to enable greater independence, “a place for everything and everything in its place” and strategies for breaking down tasks or information into manageable steps).
Rate of processing- Slowed processing of information creates problems for individuals when extra time is required to formulate a response or manage a cluster of information, particularly while under pressure.

Rate of performance - When the ability to perform tasks is slowed by injury, this decreased performance must be a consideration when setting realistic goals particularly in developing a student's Individual Education Plan (IEP) and vocational planning.

Perseveration - Many individuals with brain injury have trouble shifting their attention and find themselves “trapped” in one area of focus. There is a tendency to repeat a response or activity after it is no longer needed. External cueing or redirection may help shift the focus to another topic.

Staying on task/topic - Distractibility is a major problem for some individuals with brain injury and external cueing devices (e.g., cue cards, beepers, task lists) may be helpful. Some persons may need external redirection to get back on task. Safety can be compromised; for example, when a housewife browning meat for a casserole is distracted by a telephone call, a fire can erupt as a result of the disruption causing her to forget the task at hand. On the job, a worker distracted by other activities in the environment may be unable to resume his/her work without external cueing.

Initiation/motivation - Families often express their concern that an individual is a “couch potato.” Often this is a direct result of damage in areas of the brain responsible for initiating action or a plan. Some suggested choices of activities or assistance in starting an activity may be all that is needed to get the family member with brain injury moving and occupied productively.

Generalization - Most individuals with brain injury need help transferring skills learned in one setting (i.e., a rehabilitation program) back into a home setting. In a job setting, a promotion to a new position may prove unsuccessful without assistance to “retrain” learned skills to a different setting. Upon completion of rehabilitation, a well-executed discharge plan will make this transition easier and more successful.

Agitation - A diminished level of frustration is very common after severe brain injury. The offending stimuli may be external and obvious or internal and less obvious. Well planned strategies are very helpful to offset this problem and families are advised to seek both medical evaluation to rule out seizure activity or other possible medical problems and neuropsychological evaluation to learn more about the individual’s deficits and methods for managing the agitation.

Fatigue/stress - Understanding the fatigue level of the person can be helpful in many difficult situations. Many individuals with brain injury are unable to maintain attention, concentration and skill levels, as well as behavioral control for extended periods without time out to revitalize. This can be a problem for children and adolescents in school and persons on the job if they are expected to maintain attention for long periods of time without respite. Once a person’s fatigue-ability is identified and understood, special accommodations often may be arranged and contribute to improved performance.

Memory - Memory dysfunction is possibly the most common residual effect of brain injury and one that families generally find the most troubling. Memories of events that occurred prior to the injury usually are retained but new information and recent events may not be accessed easily. Additionally, there often is a problem with retrieving information on an as-needed basis. To retrieve or recall information one must have the ability to initiate; sustain and switch attention; recognize relevant and irrelevant information; rehearse, organize and apply it to the task or question at hand. These are all very complex cognitive abilities and ones that often are impaired as a result of brain injury.

The most practical method for helping an individual with cognitive deficits is to help him/her devise strategies to compensate for these deficits. Providing a structured and consistent setting, at least initially, can be very helpful. Compensatory strategies may include calendars, daily journals, watches with easily set alarms, post-it notes, telephone dialers, stove top timers, cue cards, task cards that organize and sequence various tasks the individual is expected to perform and many other tools developed to increase the individual’s ability to control those aspects of his/her life that can be managed as independently as possible.

Interactions of behavior, environment, and brain chemistry

Convergence of disciplines

When students of the brain learned that neurons communicate through chemical messengers, the stage was set for developing a new area of inquiry: behavioral pharmacology. This new discipline was based on a simple logic--if the communication system of brain cells is mediated by specific chemicals, then compounds that interact with these chemicals should change the messages. Consider, for example, that the behavior of drinking when thirsty might be controlled by the release of acetylcholine by certain brain cells. It should be possible to artificially stimulate this system by adding acetylcholine from another source. In the same way that Otto Loewi was able to change the rate of the heart beating in the second beaker, it should be possible to get a non-thirsty animal to drink by administering the appropriate drug. Conversely, it should be possible to prevent a thirsty animal from drinking by giving a drug that blocks the chemical messenger that is being released by the brain cells. These and other more complicated forms of behavior were simply substituted for the physiological test objects (e.g., heart, spleen, pupil, etc.) that were used by Elliot, Dale, Loewi and other pioneers in the field. The experiments worked, and a new area of research was born.

The ability to change behavior by altering brain chemistry underlined the importance of objective analysis of behavior. Behavior is more than a beating heart or a contracting eye muscle, and methods for the reliable observation of behavior were clearly needed. At about the same time that the early experiments in pharmacology were being conducted, a psychologist named B. F. Skinner was formulating a new approach for the study of behavior, which he called the analysis of operant behavior. This approach was published in a book entitled *Behavior of Organisms* (Skinner 1938) a book, which became a benchmark in the study of behavior. The basic principles involved the careful control of the animal's environment and the measurements were limited strictly to the observable, objective responses of the animal (e.g, lever presses or key pecks.) Unobservable(s) such as fear, hunger, or thirst were specifically excluded from this system of analysis.

Skinner's system for the objective analysis of behavior was eagerly embraced by the students of the new pharmacology. The precision of the chemically specific transmitter systems was mirrored by the precision of the operant method of behavioral analysis. The convergence of these two systems became synonymous with behavioral pharmacology, and set forth the basic principle of the discipline: Specific changes in brain chemistry produce specific changes in behavior.

The combination of operant analysis of behavior with pharmacological methods formed a powerful tool for researchers. It is an efficient and effective methodology for the development and screening of new drugs and, to a somewhat lesser extent, for the characterization of drug effects on behavior. But it is not enough. If we are to understand the broader implications of the chemistry of behavior, our considerations must go well beyond the effects of drugs on behavior.

Dynamics of brain chemistry and behavior

Behavior has no clear beginning or end. The analysis of behavior starts out innocently enough to describe the interactions of the organism with the environment. More specifically, it is the interaction of the organism's brain with the environment. The environment includes not only the outside world, but also the organism's internal environment. Of course, the brain is a part of that internal environment and the behavior itself becomes a part of the environment. Lest we become tempted to pursue the logical proof that the universe is made up of behavior, let us return to some more direct issues to illustrate that these considerations are not just idle philosophical musings--we must understand the implications of these interactions in order to appreciate the dynamics of brain chemistry and behavior. These interactions are presented as six principles for understanding behavioral pharmacology:

Principle 1. Changes in brain chemistry produce changes in behavior

This is perhaps the most straightforward principle and, as indicated in our previous discussion, the one that has guided most of the research in behavioral pharmacology. Manipulation of the chemical system that controls behavior will change behavior.

Principle 2. Changes in behavior produce changes in brain chemistry

This principle is a bit more subtle and offers the opportunity to confuse cause and correlation. The fact that behavioral change is correlated with the chemical changes that produced it is simply a restatement of principle 1. The important point here is that behavioral change can actually produce changes in brain chemistry. One type of change is an increase in the efficiency of the chemical system that produces the behavior (analogous to increased muscle efficiency with exercise). This change may, in turn, produce changes in related chemical systems that were not directly involved in the first bit of behavior.

Principle 3. Changes in the environment produce changes in behavior

This principle is the simple definition of behavior and requires little in the way of explanation. The major point that needs to be made is that the environment is quite extensive. It includes not only the relationships and contingencies of the external world, but also the internal milieu--blood pressure, gastrointestinal activity, level of energy stores, memory of past experiences, etc. Until recently, the internal environment has been downplayed by the "black box" approach of experimental psychology.

Principle 4. Changes in behavior produce changes in the environment

In some sense, the only role of behavior is to change the environment. In the simplest case, the behavior is operant and results in opened doors, captured prey, warmed cockles and the like. But just as the environment was expanded in the preceding paragraph, so must our notions of the effects of behavior be expanded to include, for example, changes in the internal environment either directly (as in the case of autonomic responses to a fear arousing situation) or indirectly (as in the case of nutritional changes).

Principle 5. Changes in the environment produce changes in brain chemistry

We begin to complete the circuit through brain, behavior, and environment by noting that environmental changes can produce changes in brain chemistry. In some cases, the environment has tonic influences on brain chemistry as exemplified by responses to seasonal changes, temperature fluctuations, lighting changes and so forth. Other environmental changes are more closely interactive with behavior, and include responses to crowding, members of the opposite sex, complexity of the physical and behavioral environment, etc. These and many other types of environmental manipulations have been shown to alter the status of the neuro-chemical transmitter systems.

Principle 6. Changes in brain chemistry produce changes in the environment

On the surface, this seems to be the least likely of the principles. Changes in brain chemistry obviously cannot directly perform operands like opening doors. It can, however, produce significant changes in the internal environment and set the stage for such operands to occur.

The listing of these six principles is a formal way of stating the major considerations that must accompany our study of behavioral pharmacology. We do not recommend that you commit these principles to memory, because individually they represent an artificial analysis of the situation. There is a single statement that embodies all of these principles:

“Brain chemistry, behavior, and environment have interpenetrating effects.”

This statement is the major theme of Skinner’s book, and emphasizes the need to appreciate the complexities of the nervous system. Yes, drugs change behavior. But the effect of a drug can be altered by the organism’s behavior, which in turn has been produced by current and past changes in the environment. Drugs do not possess some essence that magically induces a change in behavior. They act through the normal channels of our physiological response to the environment. As human organisms in a complex environment, we are fortunate that these interactions are complicated. As students of behavior, these physiological interactions are pushed to their limits in our feeble attempts to understand them. Do not despair; the thrill is in the pursuit.

Note:

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Source: <http://www.rci.rutgers.edu/~lwh/drugs/>

Drugs and alcohol (see also module VI- substance abuse)

The pre-injury lifestyle of a person with a brain injury may have included use of drugs and alcohol, either habitually or recreationally. Families are generally unprepared for the negative effects created when drugs and alcohol are consumed after brain injury. Abuse of alcohol and illegal substances frequently occurs post-injury. It can also occur in the life of an individual after they have acquired a brain injury, when it was not present in their pre-injury lifestyle.

Things to remember:

- When alcohol and/or other substances were used prior to the brain injury without appropriate intervention and guidance, the problem is usually exaggerated after the injury.
- Even though abuse was a problem before the brain injury and may even have contributed to the injury, therapeutic intervention involving both the individual and caregivers during rehabilitation enhances management of substances post-injury.
- All treating professionals should discuss the risks associated with the use of alcohol or substances after TBI and its effect on cognitive functioning. In the absence of professional instructions, the individual with brain injury typically assumes that drinking and/or drug use is as harmless as they thought pre- injury.
- It is not uncommon for substance abuse to surface some months or years after injury as an emotional response to depression when life does not return to “normal.” Reestablishing relationships with old friends and the desire to return to pre-injury social settings where substances were used can sabotage rehabilitation goals.

Some suggested strategies for caregivers coping with addiction or pre-addictive behaviors include:

- Don't forbid consumption without risking the “dare me” syndrome. Casually and frequently discuss the hazards of using alcohol/substances and their effect on cognitive functioning.
- Enlist the assistance of a doctor. Information from a medical professional is more meaningful. The doctor should emphasize the danger inherent with the use of drugs and alcohol in conjunction with anti-convulsions and other prescription drugs used by some individuals with brain injury.
- Contact the National Brain Injury Association, the Utah State Brain Injury Association, or your ABI Support Coordinator for articles and other helpful information about brain injury and addictive disorders.
- If there's a need to “fit in” when others are consuming alcohol, try cocktail- sized glasses, or add a twist, cherry, or olive to a non-alcoholic beverage. Many people in bars and restaurants order “virgin” drinks (without alcohol), so it's socially acceptable.
- Take a strong stand. Impress upon the “old gang” that use of alcohol/substances post-injury could be very dangerous and expect their cooperation in helping the person with the brain injury stay healthy and safe.
- When the problem is related to depression, take inventory of what the individual is doing (or not doing) during the day that contributes to their depression. Use your imagination to find something that stimulates and motivates the individual to see the world around them. Busy people have less reliance on “crutches.”
- Once an addiction problem is identified and the individual is willing to get help, get into AA or Narcotics Anonymous (NA). The selection of a sponsor is important and that person needs to be made aware of brain injury and its consequences.
- Addiction is a major public health problem and difficult to control after ABI. Treatment works but requires that lessons learned be reinforced on an ongoing basis.

Alcohol

Alcohol is a central nervous system depressant and acts as a mild anesthetic and tranquilizer. It is toxic in large quantities. When a person drinks alcohol, the alcohol is absorbed by the stomach, enters the blood-stream, and goes to all the tissues. The effects of alcohol are dependent on a variety of factors, including a person's size, weight, age, and sex, as well as the amount of food and alcohol consumed. The dis- inhibiting effect of alcohol is one of the main reasons it is used in so many social situations. Other effects of moderate alcohol intake include dizziness and talkativeness. The immediate effects of a larger amount of alcohol

include slurred speech, disturbed sleep, nausea, and vomiting. Alcohol, even at low doses, significantly impairs the judgment and coordination required to drive a car safely. Low to moderate doses of alcohol can also increase the incidence of a variety of aggressive acts, including domestic violence and child abuse. Hangovers are another possible effect after large amounts of alcohol are consumed; a hangover consists of headache, nausea, thirst, dizziness, and fatigue.

Prolonged, heavy use of alcohol can lead to addiction (alcoholism). Sudden cessation of long term, extensive alcohol intake is likely to produce withdrawal symptoms, including severe anxiety, tremors, hallucinations, and convulsions. Long-term effects of consuming large quantities of alcohol, especially when combined with poor nutrition, can lead to permanent damage to vital organs such as the brain and liver. In addition, mothers who drink alcohol during pregnancy may give birth to infants with fetal alcohol syndrome. These infants may suffer from mental retardation and other irreversible physical abnormalities. In addition, research indicates that children of alcoholic parents are at greater risk than other children of becoming alcoholics.

Alcoholism, also known as alcohol dependence, is a disease that includes the following four symptoms:

1. Craving--A strong need, or urge, to drink.
2. Loss of control--Not being able to stop drinking once drinking has begun.
3. Physical dependence--Withdrawal symptoms, such as nausea, sweating, shakiness, and anxiety after stopping drinking.
4. Tolerance--The need to drink greater amounts of alcohol to get "high." Like many other diseases, alcoholism is chronic, meaning that it lasts a person's lifetime; it usually follows a predictable course; and it has symptoms. The risk for developing alcoholism is influenced both by a person's genes and by his or her lifestyle.

Symptoms of alcohol use include:

1. slurred speech
2. lack of coordination
3. decreased attention span
4. impaired judgment

Alcohol and other drugs can cause or worsen a wide range of medical problems. For example, alcohol increases the risk of some stomach and heart diseases. Some people get gastritis (inflammation of the stomach lining) or high blood pressure because of drinking too much alcohol.

Drugs

Drug abuse is the use of illicit drugs or the abuse of prescription or over-the-counter drugs for purposes other than those for which they are indicated or in a manner or in quantities other than directed. Drug dependence (addiction) is a compulsive use of a substance despite negative consequences, which can be severe. Drug abuse is simply excessive use of a drug or use of a drug for purposes of which it was not medically intended.

Physical dependence on a substance (needing a drug to function) is not necessary or sufficient to define addiction. There are some substances that don't cause addiction, but do cause physical dependence (for example, some blood pressure medications). Other substances cause addiction but not classic physical dependence (cocaine withdrawal, for example, doesn't have symptoms like vomiting and chills; it is mainly characterized by depression).

Types of drugs and the consequences of their use

Stimulants

Stimulants (for example, cocaine, “crack,” amphetamines) give a temporary illusion of enhanced power and energy. As the initial elevation of mood fades, however, a depression emerges. Stimulant abuse can lead to serious medical problems:

- Heart attacks—even in young people with healthy hearts
- Seizures
- Strokes
- Violent, erratic, anxious, or paranoid behavior

Marijuana

The most widespread and frequently used illicit drug, marijuana is associated with the following:

- Short-term memory loss
- Accelerated heartbeat
- Increased blood pressure
- Difficulty with concentrating and information processing
- Lapses in judgment
- Problems with perception and motor skills

Heroin

Heroin, which can be smoked, eaten, sniffed, or injected, produces an intense—but fleeting—feeling of pleasure. Serious withdrawal symptoms begin, however, after 4 to 6 hours:

- Chills
- Sweating
- Runny nose and eyes
- Abdominal cramps
- Muscle pains
- Insomnia
- Nausea
- Diarrhea

Hallucinogens

Hallucinogens are drugs such as LSD (“acid”) or the new “designer” drugs (for example, “ecstasy”) that are taken orally and cause hallucinations and feelings of euphoria.

Dangers from LSD include stressful “flashbacks”—re-experiencing the hallucinations despite not having taken the drug again, sometimes even years later. Excessive use of ecstasy, combined with strenuous physical activity, can lead to death from dehydration or an exceptionally high fever.

Inhalants

Inhalants are breathable chemicals—for example, glue, paint thinner, or lighter fluid. They are commonly abused by teenagers because they are easy to obtain and because they produce mind altering effects when “sniffed” or “huffed.” These chemicals reach the lungs and bloodstream very quickly and can be deadly. High concentrations of inhalant fumes can cause heart failure or suffocation. Long-term abuse of inhalants can cause permanent damage to the nervous system.

Sedatives

Sedatives are highly effective medications prescribed by physicians to relieve anxiety and to promote sleep. Unfortunately, harmful effects can occur when they are taken in excess of the prescribed dose or without a physician’s supervision, such as when they are obtained illegally.

Combining sedatives with alcohol or other drugs greatly increases the likelihood of death by overdose. Women who abuse sedatives during pregnancy may deliver babies with birth defects (for example, cleft palate) who may also be physically dependent on the drugs.

Frequently used prescription drugs for people who have brain injuries

Anticonvulsants

Anticonvulsants (i.e., carbamazepine, valproic acid, phenytoin, phenobarbital, tiagabine, lamotrigine, gabapentin, topiramate) act to prevent abnormal firing patterns of neurons. This can occur as a result of direct injury to the cell or due to chemical changes around the cell. These seizures either can be generalized or focal events. Focal seizures may involve sensory, motor or behavioral regions of the brain. One way in which anticonvulsants (i.e., benzodiazepines, barbiturates, valproic acid) may prevent seizures is by increasing the activity of an inhibitory neurotransmitter, GABA. They also may decrease the firing rates by preventing the “snowball” effect of seizure production called kindling (i.e., carbamazepine).

Anticonvulsants can be used not only to prevent seizures, but also to:

- decrease irritability,
- improve frustration tolerance,
- decrease headache and
- stabilize mood swings.

Balance problems also may respond to certain anticonvulsants. Once these anticonvulsant medications are prescribed, follow-up blood testing may be required to ensure that the concentrations of medication in the blood falls within the therapeutic range. This is the level required to inhibit seizures in 95% of persons. These tests also may involve assessment of liver function and blood counts (CBC) to monitor potential toxicity of these agents.

Side effects commonly encountered with these agents include: fatigue (barbiturates, benzodiazepines), dizziness (phenytoin, carbamazepine) and gastrointestinal irritation (valproic acid). Abruptly stopping these medications without medical guidance can result in severe seizures and even death.

Antidepressants

Antidepressants were first developed in the 1940s, and many refinements have occurred in the years since. Types of antidepressants include monoamine oxidase inhibitors (MAOI), tricyclics (TCA), heterocyclics and specific serotonin re-uptake inhibitors (SSRI). Novel antidepressants also have been developed which have combination effects. MAOIs (i.e., phenelzine, tranylcypromine) act by slowing the breakdown of neurotransmitters at the synapse (the junction where neural impulses are transmitted). The agents currently available require strict dietary control to prevent toxic reaction, which will elevate blood pressure to lethal levels. MAOIs tend to increase energy but may cause insomnia, even at low dosages. Prescription of these agents must be supervised closely to prevent accidental drug-drug interaction (i.e., avoiding meperidine, decongestants, diet pills).

TCAs (i.e., amitriptyline, imipramine, desipramine, nortriptyline, protriptyline, clomipramine) are related closely to antihistamines and possess many of the same characteristics. They act by decreasing the reabsorption of neurotransmitters into the releasing neuron (“re-uptake inhibition”). No dietary restrictions are necessary with TCAs. They act to increase two neurotransmitters—serotonin and norepinephrine. Onset of action generally is two to four weeks after treatment is started. This allows the development of certain blood concentrations and then for the agent to cross into the neuron. Periodic assessment of blood level is useful to ensure an effective concentration. Side effects with TCAs largely are caused by their antihistaminic and anticholinergic properties. They tend to be more sedating and commonly induce initial sleep improvement. They also tend to cause dry mouth, delayed urination, sexual dysfunction, constipation and lightheadedness. These side effects also can assist in alleviating some types of post traumatic dizziness.

Some cardiac changes may be evident, including increased heart rate and, rarely, skipped beats. TCAs also may lower the seizure threshold after brain injury. These medications can be used for:

- explosive episodes,
- emotional instability,
- headache relief,
- chronic pain management,
- insomnia,
- post-traumatic stress disorder and
- typical depressive symptoms.

SSRIs (i.e., fluoxetine, fluvoxamine, sertraline, paroxetine, nefazadone, citalopram) are the newest agents in this class. SSRIs prevent the reabsorption of serotonin into the releasing neuron and increase its availability to the next neuron downstream. These powerful medications have a more rapid action onset. Usually, they have no cardiac side effects. Principle side effects relate to nausea, dizziness, fatigue and, occasionally, tremor. SSRIs also may cause sexual dysfunction. Interaction with anticonvulsants also can influence seizure threshold.

Novel antidepressants combine serotonin re-uptake inhibition with norepinephrine re-uptake inhibition (i.e., venlafaxine) or dopamine blockade (i.e., amoxapine). Side effects are similar to other agents. However, amoxapine may cause involuntary movements as can neuroleptics. Bupropion causes re-uptake inhibition of serotonin, norepinephrine and dopamine. It may be associated with overstimulation or seizures.

Anti-anxiety agents

Anti-anxiety agents (i.e., lorazepam, diazepam, alprazolam) exert their effect by increasing the inhibitory neurotransmitter, GABA. This then slows the firing rates of all neurons in the region. For thousands of years, alcohol has been used to do this as well. Currently used agents primarily are benzodiazepines, although barbiturates still are prescribed. The effect of these agents is to reduce the individual's awareness of environmental stress and disrupt memory of the events. Buspirone acts to decrease the impact of environmental events on aggression through interference with serotonin activity in the hippocampal/amygdala (memory processing) regions of the brain. Side effects of GABA-potentiating agents include:

- sedation,
- short-term memory disruption,
- muscle relaxation and
- development of tolerance.

They act to raise the seizure threshold and have some use as secondary anticonvulsants. These agents cannot be stopped without medical supervision as they can result in severe withdrawal delirium, including potentially lethal seizures. The use of ethanol with these agents greatly increases their sedating properties and can result in slowing or stopping breathing. Short-term use is appropriate if closely supervised by a physician.

Neuroleptics

Neuroleptics (i.e., chlorpromazine, haloperidol, thioridazine, risperidone, pimozide) act by blocking the transmission of dopamine-stimulated nerve impulses. They rarely are used for agitation and aggressive behavior, as studies have shown that they may slow the recovery rate after brain injury. Neuroleptics may be required in severe cases of delusional thinking or hallucinations. Other similar medications are used to decrease nausea and vomiting and enhance the effect of narcotic pain relievers. Side effects include:

- abnormal involuntary movements,
- weight gain,
- low blood pressure,
- lowered seizure threshold, and
- decreased memory.

Permanent movement disorders can be seen. Newer agents such as clozapine, olanzepine, ziprasidone and quetiapine are less likely to cause movement problems, although lowered production of blood cells can be observed with clozapine.

Antiparkinson agents

AntiParkinson Agents (i.e., levodopa, amantadine, bromocriptine, pergolide, pramipexole, ropinirole,

benztropine) act to increase dopamine activity or decrease cholinergic activity at the synapse. This may be beneficial in certain types of amotivational syndromes and initiation deficits. They are used to increase endurance – both cognitive and physical – and improve swallowing in certain individuals. They also can improve initiation and mood. Side effects include:

- agitation,
- nausea,
- blood pressure changes and
- headache.

High dosages also may induce hallucinations or paranoid delusions.

Psychostimulants

Psychostimulants (i.e., methylphenidate, dextroamphetamine, pemoline) are used to:

- decrease daytime drowsiness,
- increase attention and concentration and
- increase mood temporarily.

They act by increasing the release of already-produced norepinephrine and dopamine from storage areas of the neuron. Their onset of action is within hours, and their duration is usually less than 24-hours (with the exception of Pemoline). Long-term use must be monitored closely by a physician, because of the abuse potential and possible lowering of the seizure threshold. These agents also can trigger paranoid thoughts and insomnia. Modafinil is a new agent that is useful in combating fatigue associated with neurological dysfunction.

Anticholinergic agents

Anticholinergic Agents (i.e., meclizine, scopolamine) may be used to increase tolerance for certain types of dizziness, increase endurance and relieve insomnia at the beginning of the night. The ability of these agents to lower seizure threshold and to cause dry mouth, constipation and confusion at high doses requires close monitoring.

Antihypertensives

Antihypertensives are used for headache management, aggressive behavior and impulsivity. Beta-blockers (i.e., propranolol, atenolol) were the first of this class to be used successfully. Side effects include lowered heart rate and blood pressure. The agents cannot be used in persons at risk for hypoglycemia, as they mask the physical complaints. Certain medications (i.e., propranolol) also may increase depressive symptoms. Alpha blocking agents (i.e., clonidine) are used to decrease impulsivity and blood pressure. Calcium channel blockers (i.e., verapamil) have been used to treat migraine headaches after brain injury. Their primary side effects include light headedness and constipation.

Narcotic antagonists

Narcotic antagonists (i.e., naltrexone) are a class of medications that block the brain's naturally produced opiates (endorphins) from attaching at receptor sites in the brain. These agents can be used to decrease self-injurious behavior, bulimic symptoms (binging and purging on food) and suicidality. These agents may decrease the craving for alcohol in those individuals with alcoholism. Side effects include potential liver irritation, confusion and headache.

Botox (aotulinum toxin type A)

This unique agent that is injected into muscle, prevents the release of chemical transmitters that cause muscles to contract. While other chemicals (i.e., phenol, local anesthetics) have been used to reduce this increased contraction (spasticity), Botox provides symptomatic relief of spasticity within three to seven days of injection. Repeated dosing may be necessary to achieve the desired reduction in tone.

[Click here to see the National Institute of Health web site](#) for detailed information on specific medications.

Effects of food, alcohol, and caffeine on medications

Certain foods, beverages, alcohol, caffeine, and even cigarettes can interact with medicines. This may make them less effective or may cause dangerous side effects or other problems. Drug interactions may make a drug less effective, cause unexpected side effects, or increase the action of a particular drug. Some drug interactions can even be harmful. Read the label every time a Non-prescription or prescription drug is taken. You can reduce the risk of potentially harmful drug interactions and side effects with a little bit of knowledge and common sense.

Drug interactions fall into three broad categories:

- Drug-drug interactions occur when two or more drugs react with each other. This drug-drug interaction may cause you to experience an unexpected side effect. For example, mixing a drug you take to help you sleep (a sedative) and a drug you take for allergies (an antihistamine) can slow your reactions and make driving a car or operating machinery dangerous.
- Drug-food/beverage interactions result from drugs reacting with foods or beverages. For example, mixing alcohol with some drugs may cause you to feel tired or slow your reactions.
- Drug-condition interactions may occur when an existing medical condition makes certain drugs potentially harmful. For example, if you have high blood pressure you could experience an unwanted reaction if you take a nasal decongestant.

General medication interactions

Antihistamines (Allegra, Claritin, Benadryl)

It is recommended that prescription antihistamines be taken on an empty stomach to increase their effectiveness. Some antihistamines may increase drowsiness and slow mental and motor performance. Use caution when operating machinery or driving.

Arthritis & pain medications (Tylenol)

For rapid relief, these medications should be taken on an empty stomach because food may slow the body's absorption of acetaminophen. Avoid or limit the use of alcohol because chronic alcohol use can increase your risk of liver damage or stomach bleeding. If you consume three or more alcoholic drinks per day talk to your doctor or pharmacist before taking these medications.

Non-steroidal anti-inflammatory drugs (Aspirin, Aleve, Ibuprofen)

Because these medications can irritate the stomach, it is best to take them with food or milk. Avoid or limit the use of alcohol because chronic alcohol use can increase your risk of liver damage or stomach bleeding. If you consume three or more alcoholic drinks per day talk to your doctor or pharmacist before taking these medications. Buffered aspirin or enteric coated aspirin may be preferable to regular aspirin to decrease stomach bleeding.

Narcotic analgesics (Percocet, Demerol, Vicodin, Tylenol #2, #3, or #4)

Avoid alcohol because it increases the sedative effects of the medications. Use caution when motor skills are required, including operating machinery and driving.

Anti-anxiety drugs (Valium, Xanax)

Alcohol may impair mental and motor performance (e.g., driving, operating machinery). Caffeine may cause excitability, nervousness, and hyperactivity and lessen the anti-anxiety effects of the drugs.

Anti-depressants (Paxil, Zoloft, Prozac)

Although alcohol may not significantly interact with these drugs to affect mental or motor skills, people who are depressed should not drink alcohol. These medications can generally be taken with or without food.

**Contributors to this module**

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<https://www.biausa.org/>

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<https://dspd.utah.gov/>

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