

Insight

Neuroanatomy of Behavior After Brain Injury or You Don't Like My Behavior? You'll Have to Discuss That With My Brain Directly.

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Introduction

“Any given behavior [e.g., watering a plant, dressing] is the product of a myriad of complex neurophysiological and biochemical interactions involving the whole brain” (Lezak, 1995, p. 45). However, the “...disruption of complex behavior by brain lesions occurs with such great anatomical regularity that inability to understand speech, to recall recent events, or to copy a design, for example, can often be predicted when the site of the lesion is known” (Lezak, 1995, p. 46). The ability to *localize* a lesion based upon the disruption of behavior led scientists during the early nineteenth century to

specialization is acknowledged, the relative degree of involvement of a specific region may vary and there may be interactions with other regions of the brain. The advent of functional neuroimaging such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and single photon emission computerized tomography (SPECT) has allowed researchers to observe the dynamic interactions of the brain while a person is engaging in a specific behavior (Goldberg, 2001). In 1997, Shadmehr and Holcomb (as cited in Goldberg, 2001) studied the activation of brain areas using PET scans

always the simple result of willfulness and deliberation, it is possible to oversimplify the organic explanation for behavioral change after brain injury. As in the above example, multiple brain areas may be involved in the execution of even a simple behavior. Furthermore, brain lesions after an injury are not typically isolated or contained within predictable boundaries (*focal*). Rather, closed head injuries, particularly severe injuries, are usually *diffuse* (spread out). More than one area is involved, some to a greater or lesser extent.

Understanding the neurological basis of behavior extends well beyond identifying brain regions that are associated with a particular syndrome.

take the concept to an extreme: cognitive abilities (reading, mental processing), personality traits (courage, recklessness), and attitudes (affection, contempt) were attributed to specific parts of the brain (Goldberg, 2001).

while the subject learned a complex motor skill. During the early learning stages, the right prefrontal cortex was activated. During the late training stages, activation shifted to multiple areas scattered throughout the brain.

It is clear to modern researchers that brain structure is organized in a more complex fashion than the scientists of the early nineteenth century believed. While a certain degree of *regional*

Regional specialization is implied in describing behavioral syndromes associated with brain lesions, however we should proceed with caution. Just as it is inaccurate to assume that behavior is

Understanding the neurological basis of behavior extends well beyond identifying brain regions that are associated with a particular syndrome. Behavior also is dependent on complex neural networks that are influenced by a balance or imbalance of neurotransmitters (refer to “Medications and Behavior” in this issue of *Premier Outlook*). While some neurotransmitters are associated with specific brain areas, most are scattered throughout the various structures of the brain. As Goldberg (2001, p. 28) remarks, “The brain can be thought of as the coupling of two highly complex organizations, structural and chemical. This

coupling leads to an exponential increase in the system's overall complexity."

Behavior following injury is also influenced by a host of other factors. Environmental factors, stage of development, effects of normal grieving, personality characteristics prior to the injury, and personality disturbance that may develop after the injury due to coping or adjustment problems contribute to the person's behavioral pattern (Hibbard et al., 2000; Prigatano, 1999). Part of the problem in studying behavior following brain injury is in defining, recording, and evaluating the behavior because there are so many subtle variations (Kolb & Wishaw, 1990). Each individual surviving a brain injury presents with a unique constellation of behaviors.

Changes after Brain Injury

Subsequent to an injury to the brain, an individual may experience physical, cognitive, and/or behavioral and emotional changes.

Physical Changes

Physical changes as a result of injury to the brain include the following:

- Hemiplegia or hemiparesis (paralysis or weakness of one side of the body)
- Spasticity
- Tremors
- Hearing loss
- Seizures
- Double vision
- Visual field cuts
- Changes in sensory perception
- Fatigue
- Ataxia (problems with balance or coordination)
- Dysphagia (problems swallowing)
- Dysarthria (problems with articulation)
- Autonomic dysfunction (disregulation of the stress reaction)
- Apraxia (inability to carry out purposeful movement)

Physical changes are typically observable and the connection between a physical consequence and an associated behavior change is usually obvious. For example, a person may no longer play basketball due to a left-sided hemiparesis (weakness). Activities may be reduced due to fatigue.

Cognitive Changes

Changes in cognitive functioning also may result in changes in behavior. Cognitive changes after a brain injury may include problems in the following areas:

- Level of consciousness
- Attention/concentration
- Memory
- Expressive language (spoken or written)
- Receptive language (understanding what is said or written)
- Constructional ability (copying 2- or 3-dimensional designs)
- Orientation (knowing who, what, when, where and why)
- Abstract thought
- Planning
- Organizing
- Insight
- Generalization
- Flexibility
- Problem solving
- Speed of mental processing
- Academic skills
- Right-left orientation

An altered level of consciousness results in confused behavior. This is observed mostly during the initial stages of recovery. However, other cognitive problems may persist. Needless to say, problems in most of the areas listed will have an impact on emotional and behavioral functioning. For example, social interactions are affected by one's ability to attend in individual or group discussions. Inability to plan and organize may negatively affect productivity and lead to frustration. Lack of insight and generalization interfere with one's ability to

learn from social and behavioral mistakes and to apply lessons learned.

Emotional/Behavioral Changes

Behavioral changes as a direct result of injury to the brain may present on an ongoing basis or only under specific circumstances (i.e., when in stressful situations or when expected to perform socially). They may coexist with normal intelligence and physical recovery, but interfere with normal adjustment (Strub & Black, 1988). The following are changes in behavior that may present subsequent to brain injury:

- Agitation (excessive restlessness)
- Lack of cooperation
- Inability to tolerate frustration
- Aggression, anger, or hostility
- Emotional lability (extreme & inappropriate fluctuations in mood)
- Distortions of reality
- Obsessions or compulsions
- Loose associations
- Tangentiality (answers to questions are obliquely related or completely unrelated)
- Egocentrism
- Decreased social skills
- Lack of initiation and motivation
- Perseveration (repeating an idea or action over and over)
- Disinhibition
- Impulsivity

Studies show that during the acute stage of recovery, 35 to 96 percent of patients exhibit agitated behavior. One to 15 years after injury, irritability and bad temper occurred in 31 to 71 percent of patients who had severe traumatic brain injury. It is important to emphasize the characteristics of aggressive outbursts resulting from brain injury. The outbursts tend to be reactive, non-reflective, and non-purposeful, particularly during the early stages of recovery. They also tend to be volatile and sporadic (Silver, Anderson, & Yudofsky, 2003).

It is clearly understood that there is most often an underlying organic cause to the behavioral changes described above. In some cases, however, behavioral changes may be referred to as *functional*, that is, no organic condition can be identified to account for the behavior. The clinician should always first consider the possibility of an organic condition since behavioral and cognitive problems may present without physical findings. It is not uncommon for a lesion to show up later on autopsy (Lezak, 1995).

Each individual with brain injury presents with a unique combination of symptoms. Despite this variability, several *clinical syndromes* involving disturbances in mood, personality, and emotional reaction following brain injury have been identified (Strub & Black, 1993). Following is a description of these *clinical syndromes*. The list is, by no means, exhaustive. It is hoped, however, that the information presented will enhance the reader's understanding of behavioral change secondary to traumatic brain injury.

Clinical Syndromes Involving Behavioral Disturbance

Frontal Lobe Syndromes

Following damage to the frontal lobes (see Figure 1), an individual may present with a frontal lobe syndrome. In these syndromes a person may retain normal, or near-normal, intellectual and neurological functioning and may perform well on psychological tests that measure specific abilities in isolation. Ability to function may decline significantly, however, when the individual is expected to coordinate these skills in an organized, goal-directed process. In general, the frontal lobe syndromes are almost purely behavioral, although cognition may be affected in specific ways and in some injuries (Strub & Black, 1988). Strub and Black (1993, p. 16) describe individuals with frontal lobe syndrome as having "an intellect without social and emotional guidance."

While behavior may be outgoing, the person exhibiting frontal lobe syndrome gives a false impression of interest and productivity. These individuals have lost their interest in the environment and productive social drive. They fail to perform on the job, to maintain normal family relations, or even to maintain personal cleanliness (Strub & Black, 1993). Variation in behavioral presentation is observed depending upon the exact location of the lesion (as described below), however, "underlying the behavior of all patients with significant frontal lobe damage is an unfortunate lack of motivation, inability to plan ahead, and poor judgment" (Strub & Black, 1988, p. 286).

Individuals with injury to the frontal lobes may have significant problems when asked to change from one activity to another or to perform a repetitive sequence of actions. They tend to *perseverate*, that is, to repeat the action or verbalization over and over without moving on to the next required action, activity, or topic.

An additional consequence of frontal lobe damage is the inability to stay on track. Incidental environmental distract-

tions and internal associations can easily result in *derailment* of thought processes (slipping off track from one topic to another) similar to patterns seen in individuals with Attention Deficit Disorder/Attention Deficit Hyperactivity Disorder (ADD/ADHD). This is also similar to the *tangentiality* seen in schizophrenia in which answers to questions are unrelated. Both ADD/ADHD and schizophrenia are considered frontal lobe disorders (Goldberg, 2001). *Utilization* or *field-dependent behavior* is another example of the distractibility seen with frontal lobe injury. The individual may drink from an empty cup, put on glasses that do not belong to him/her, or enter through a door just because these items are there and not because the action makes sense. In extreme cases, the individual engages in direct imitation of speech (*echolalia*) or action (*echopraxia*). When asked, "Where are you from?" they may reply, "Where am I from, Chicago," (Blumenfeld, 2002; Goldberg, 2001).

Although it is most frequently seen in individuals with right hemisphere lesions, severe frontal lobe damage may result in a debilitating condition, referred to as *anosagnosia*. In this condition, the

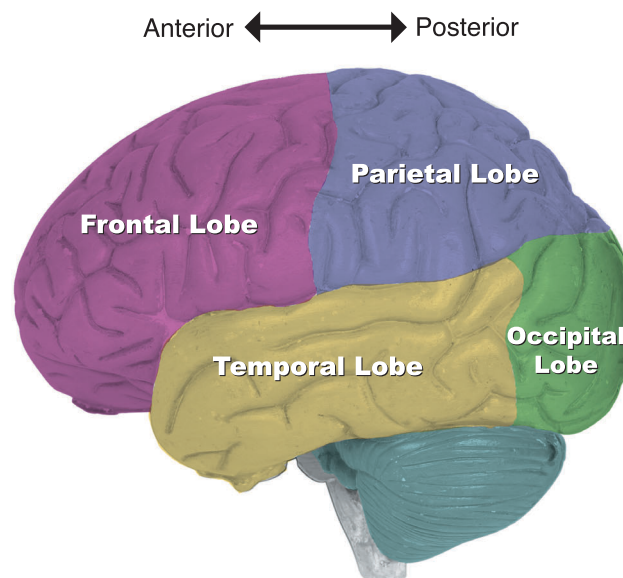


Figure 1. Four lobes of the cerebral cortex

individual lacks awareness of any impairment or deficits he/she may have and insists that everything is fine. In contrast to being “in denial,” in which it is assumed that the individual comprehends the deficit, but “chooses” to look the other way, with anosognosia insight into the illness or injury is genuinely lost. The person may “not have the slightest inkling that his life had been catastrophically and irreversibly changed by the illness” or injury (Goldberg, 2001, p. 136).

The frontal lobes are considered to be relatively vulnerable, as they are affected in more brain disorders than any other part of the brain. Frontal lobe functioning appears to have a low threshold for injury or “breakdown.” Goldberg (2001) suggested that the richness of the frontal lobes’ connections contribute to their unique vulnerability.

The Prefrontal Cortex

The frontal lobes were the last to evolve and are considered to be unique to humans (relative to other species) in terms of their level of development. According to Korbinian Brodmann (as cited in Goldberg, 2001), the *prefrontal cortex* (the front part of the frontal lobes) (see Figure 2) accounts for 29% of the total cortex in humans. It only accounts for 17% of total cortex in the chimpanzee, 11.5% in the gibbon (skinny Asian monkeys with long arms) and the macaque (Asian monkeys with short tails), 8.5% in the lemur, 7% in the dog, and 3.5 % in the cat.

The prefrontal cortex plays the main role in goal formation and in developing a plan of action to reach those goals. It coordinates the skills needed and applies them in order, then evaluates the success or failure of the action based upon the intention. It has been likened to the CEO of a large corporation, coordinating and integrating the activities of other important brain structures (e.g., those responsible for perception, memory, survival decisions, emotion, vital internal

states/homeostasis, activation and arousal, etc.). It is considered the “best-connected part of the brain” (Goldberg, 2001, p. 35). Injury to the prefrontal cortex interferes with ability to plan and to anticipate the consequences of action (Goldberg, 2001).

Following is a description of the distinct behavioral patterns observed after damage to the *dorsolateral* and *orbitofrontal* areas of the prefrontal cortex (see Figure 2).

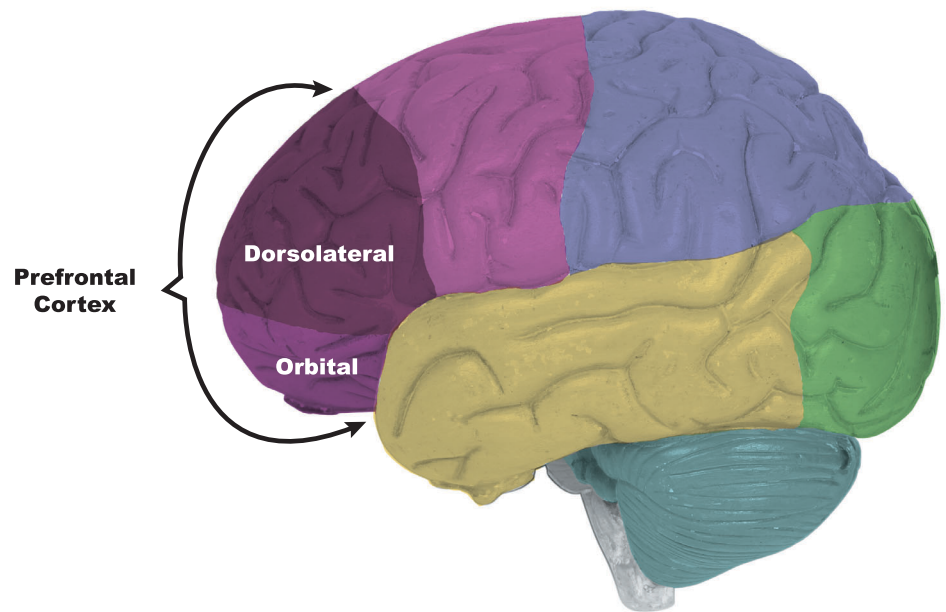


Figure 2. Prefrontal cortex with dorsolateral and orbitofrontal areas.

Dorsolateral syndrome.

When the dorsolateral area of the prefrontal cortex is severely injured, the individual may exhibit extreme inertia and an inability to initiate behaviors. He/she may be extremely passive, not bothering to eat or drink or to attend to other needs. The behavior is referred to as *abulic*, or having a tendency to stare passively and to respond only after a long delay. As described in “Psychological Factors Affecting Behavior: Depression after Brain Injury” in this issue of *Premier Outlook*, this apathy may be misdiagnosed as depression and psychiatric treatment may be erroneously undertaken with no effect. The person lacks the sad mood and sense of misery that a depressed person has.

Rather, the person with dorsolateral syndrome has a *flat affect* (or lack of expression) and appears indifferent (Goldberg, 2001). He/she appears to be unresponsive to external events, either good or bad. This indifference may be apparent across settings, including work and family. Attention is also disrupted and the individual is easily distracted (Strub & Black, 1988). There are problems with irritability, however, it is short-lived (Strub & Black, 1993). Individuals with

prefrontal dorsolateral syndrome may also have problems with mental flexibility, that is, perseveration interferes with their ability to efficiently shift thinking when environmental changes signal the need to alter their behavior (Kolb & Wishaw, 1990; Strub & Black, 1988). There may be problems terminating an activity once started. For example, hand-over-hand guidance may be required to engage in a drawing task. Once engaged, the individual may continue to draw the image over and over again until his hand is removed from the page. Goldberg (2001) has referred to this pattern as *reverse inertia*.

The previous examples are extreme, however, after even mild injury to the dorsolateral area, there may be signs of indifference and lack of drive. If the changes are subtle, it may be difficult for family members or professionals to recognize the problem as neurological. Rather, it may be perceived as a “change in personality.”

Orbitofrontal syndrome

In contrast to the passivity seen in dorsolateral syndrome, in the orbitofrontal syndrome the individual does what they feel like doing at any point in time, without concern for social taboos or legal prohibitions. Personality changes may include a cheerful lack of concern about the illness, inappropriate joking, and other disinhibited behaviors (Blumenfeld, 2002). However, the individual is frequently aggressive and irritable. Emotional expression may fluctuate between euphoria and rage. There is no evidence of ability to control impulses (Goldberg, 2001; Silver et al., 2003). It has been suggested that the damage results in a failure of the frontal lobes to inhibit, process, or modulate sudden discharges of emotion from the limbic system (Silver et al., 2003). Behavioral patterns may appear that are considered antisocial and are quite inconsistent with the individual’s behavior prior to his/her brain injury or illness. Examples may include stealing, lying, sexual aggression/inappropriateness, use of profanity, selfishness, boastfulness, overly jocular and off-color humor, and immaturity. As mentioned, frequently, there is even a failure to recognize that the actions are morally wrong. While some actually engage in criminal behaviors, most appear as “lacking in inhibitions, ‘loose’ but harmless” (Goldberg, 2001, p. 140). Goldberg (2001) described a patient that was brought for an evaluation after he had bought 100 horses “on an impulse.” There may be problems with diagnosis if an adequate history is not taken since the symptoms may resemble psychopathic behavior.

Direct damage or disconnection between the orbitofrontal area (as well as other areas in the region) and limbic structures may result in an individual’s tendency to *confabulate*. Feinberg and Giacino (2003, p. 363) define confabulation as “an erroneous statement that is made without a conscious effort to deceive.” The individual may make minor errors in content or order or they may make statements that are bizarre and impossible. Some researchers describe confabulation in terms of the need to cover a gap in memory. Confabulation is associated with a disruption of the retrieval process for memories that have been formed. While actual memory impairment commonly accompanies confabulation, the two conditions do not always occur together. Also, while executive dysfunction (judgment problems, disinhibition, inability to shift mental set) frequently accompanies confabulation, it is not a necessary component.

Limbic Syndromes

Structures Involved in Emotion and Behavior

The limbic system consists of a number of structures deep within the brain that are involved in instinctual and emotional

behavior (see Figure 3). Clearly defining the regions that encompass these structures is complicated and controversial. For example, portions of the frontal and temporal lobes are considered to be a part of the limbic system, due in part to their loaded connections, but also because of the classic emotional changes that occur if there is damage to the area. The issue is complicated even further: the signal may originate in the limbic system; however, other parts of the cortex contribute to the emotional experience. The process of planning and working toward goals (a frontal lobe function) involves an interaction with limbic system structures and the emotions they generate. For example, planning for and studying for an important test is accomplished by the added component of emotion (in this case fear of failure, of losing a job, etc.) (Strub & Black, 1988).

The amygdala and hippocampus, which are considered part of the limbic system but also part of the temporal lobes, help to regulate interactions with the external world that are associated with survival (e.g., knowing when to fight/attack, escape from danger, copulate, and ingest). The amygdala helps provide a rapid emotional assessment of a situation

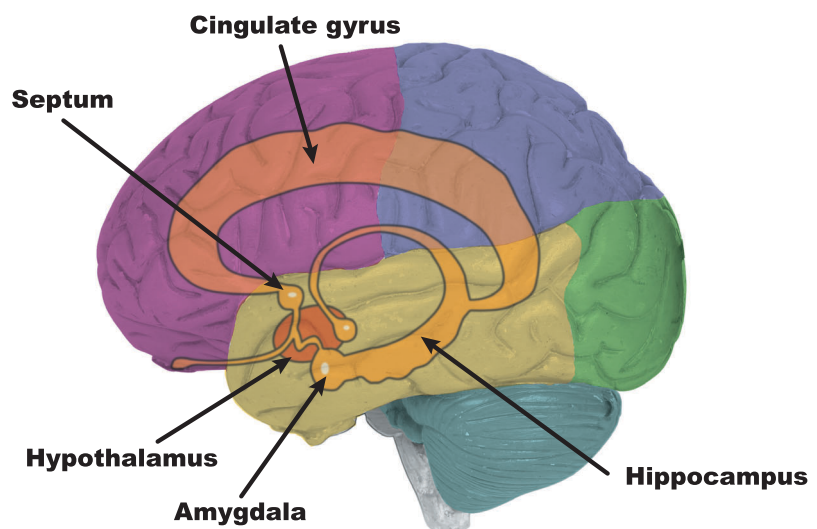


Figure 3. Limbic systems structures associated with emotion and behavior (view of structures located medially or toward the middle/internal area of the brain).

in regard to survival (Goldberg, 2001). It should be noted that the hypothalamus is involved in the *fight or flight* process as a mediator (Strub & Black, 1988). The amygdala is associated with negative emotions such as sadness, hate, anger, and fear. The effects of specific medicines that reduce anxiety in humans have been localized to sites within the amygdala (LaBar & LeDoux, 2003).

Aggressive behavior may result from neuronal excitability in limbic system structures. Silver and colleagues (2003) explain that *subconvulsive stimulation or kindling* of the amygdala results in permanent changes to the neuron. That is, the cell may become more excitable. This process can result in activation of the amygdala, causing enhanced emotional reactions (e.g., outrage at insignificant events) (Silver et al., 2003).

The septal nuclei and cingulate gyrus are associated with positive emotions. The experiences of pleasure, as well as fear and anguish, can be evoked when these particular structures in the limbic system are electrically stimulated. Pharmacological studies have shown that neurons involved in the experience of pleasure, also are involved in endorphin (endogenous opiate) production (Goldberg, 2001).

Each person's basic temperament has a basis in brain physiology and structure. It has been suggested that the functions of temperament (activity, drive level, need for attention, degree of satisfaction gained from reward, and mood) involve interactions with the limbic and arousal systems, the learning process, and socialization (Strub & Black, 1988).

Specific Limbic Syndromes

When functioning of specific areas of the hypothalamus is compromised by lesion, overeating, aggressiveness, and

confusional behavior result. Other patients may present with manic behavior, sloppiness, and paranoia. These behavioral signs are usually accompanied by endocrine or autonomic disorders such as an abnormal response to temperature change, menstrual abnormalities, and diabetes insipidus. Damage to other parts of the hypothalamus may cause the individual to develop anorexia. Again, when this behavioral pattern is observed, endocrine and autonomic changes will also appear (Strub & Black, 1988).

Damage to the limbic structures that are located deep in the frontal and temporal lobes may cause behaviors that are similar to psychiatric diseases. A variety of *pseudopsychiatric* states may be observed, such as depression or schizophreniform psychosis (Strub & Black, 1988).

Kluver-Bucy Syndrome has been associated with damage to limbic system structures, although temporal lobe structures are involved. Behavioral changes include placidity, increased oral tendencies, altered eating habits, amnesia, hypersexuality, and *visual agnosia* (inability to recognize objects by sight, in spite of adequate vision), although the full syndrome is not usually seen. These behavioral patterns are generally only seen during the initial stages of recovery following trauma. Strub and Black (1988, p. 291) described a patient who would "attack female staff members sexually, fight with male staff members, and eat anything he could get his hands on including his medical chart."

Temporal Lobe Syndromes

Temporal lobe injury has been associated with *affective* (emotional) disturbance. As discussed above, the temporal lobes include two structures that are part of the limbic system: the hippocampus and the amygdala. If these structures are damaged,

emotional and personality changes may occur (Strub & Black, 1988). In humans, temporal lobe dysfunction is associated with *hyposexuality* (decreased sex drive). This is particularly apparent in patients with focal epilepsy (brain seizures that originate from localized, irritative lesions) of the temporal lobes (Carlson, 1991). It should be noted, however, that the mating drive originates in deep frontal lobe structures, very close to limbic system structures. The drive appears to be modified and inhibited by the action of temporal and limbic structures. In humans, sexual behavior is also influenced by learned social behavioral patterns (Strub & Black, 1988).

Temporal lobe epilepsy has been associated with specific personality characteristics. The individual with temporal lobe epilepsy may tend to overemphasize trivia or meaningless details of daily life. Other symptoms include, "pedantic speech, egocentricity, perseveration on discussion of personal problems (sometimes referred to as "sticky," because one is stuck talking to the person), paranoia, preoccupation with religion, and proneness to aggressive outbursts" (Kolb & Wishaw, 1990, p. 453). It should be noted that few people present with all of these traits. Hyposexuality, hypersexuality, and a variety of odd or bizarre sexual behaviors have been associated with seizure activity (Strub & Black, 1988).

Lateralized Lesions

The brain is divided into two hemispheres, the right and the left (see Figure 4). Regions of the brain are described as anterior (toward the front) and posterior (toward the back). Brain areas are also described as lateral (out to the sides). Consequently, when lesions are lateralized, they are either in the right or the left hemisphere (that is, the right or left side). The two hemispheres have a similar appearance, however,

each has functional differences from the other. Following is a description of the differences in emotional behavior of individuals with left and right hemisphere lesions.

Left Hemisphere

In terms of emotion and behavior, individuals with left hemisphere damage may present with an intense anxiety reaction when they begin to fail on a task that was within their capability prior to the injury. This has been described as a *catastrophic reaction*, an excessive, disruptive, and momentary emotional reaction. The reaction may include tearfulness and agitation, however, the individual may regain his composure when the expectation or task is removed (Strub & Black, 1988).

There is also a high incidence of depression and anxiety in individuals with anterior left hemisphere damage. Depression appears to reflect awareness of deficit. Anxiety may present as undue cautiousness, over sensitivity to disability, and a tendency to exaggerate impairment. The prognosis is generally better for these individuals, however, because they are willing to compensate for deficits and

make adjustments in their living situations. Posterior left hemisphere lesions tend to result in indifference and paranoia, rather than anxiety and depression. Since there is a diminished capacity for awareness of deficit with left posterior lesions, the individual appears to be spared the agony of depression (Lezak, 1995; Strub & Black, 1988).

Right Hemisphere

In contrast, individuals with right hemisphere lesions demonstrate a lack of emotional response and apathy (similar to those with frontal lesions described previously). There is an altered appreciation for humorous situations (the response may be exaggerated or none at all), they may have problems identifying the emotional tone of someone's voice or facial expression, and they are more likely to take risks than to be cautious (Lezak, 1995; Strub & Black, 1988). These individuals are less likely to experience dissatisfaction with themselves and less likely to be aware of their mistakes compared to those individuals with left hemisphere lesions. This may be referred to as an *indifference reaction* (denying or making light of deficits). The emotional and behavioral patterns also vary in right hemisphere lesions, depending upon whether the damage is anterior or

posterior. Those with anterior right hemisphere lesions are described as inappropriately cheerful, but lacking in drive. For those with posterior right hemisphere lesions, the individual tends to experience depression. In contrast to anterior left hemisphere lesions, however, with posterior right hemisphere lesions there is a tendency to be apathetic, with a low mood that does not appear to arise from awareness of deficits. Rather, it appears to result from the secondary effects of diminished self-awareness and social insensitivity. For example, lacking awareness of impairment, the person may set unrealistic goals and frequently fail. Their lack of self-awareness and insensitivity make them difficult to live with and more likely to be rejected by others than individuals with left hemisphere anterior lesions. Depression takes longer to develop and is likely to be an evolving reaction to the secondary problems described. When it does develop, it can be more chronic, debilitating, and difficult to treat. It should be emphasized that, while there may be problems processing emotional communication, these individuals do experience emotions as much as persons without lesions/injury (Lezak, 1995).

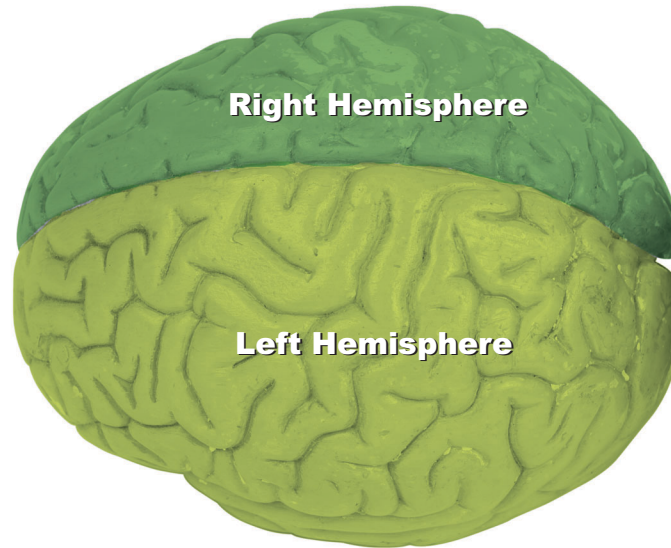


Figure 4. Cerebral hemispheres (view from the top of the brain).

Clearly, the goal of rehabilitation professionals, educators, and family members is to teach and help an individual build adaptive coping and social skills that may have been lost as a direct result of the injury. Understanding and empathy are prerequisites for accomplishing this goal.

Another group of syndromes associated with right hemisphere injury or disease is referred to as *misidentification*. In misidentification syndromes, the individual incorrectly identifies and reduplicates people, places, objects, or events. One case study reported by Feinberg and Roane (2003b) involved a woman who was convinced that her family members had been replaced by imposters. Misidentification most frequently occurs with right hemisphere lesions when there are also *bifrontal* (both the right and left portions of the frontal lobes) lesions. Right hemisphere lesions can cause left *hemineglect*, a behavioral syndrome associated with attention to one side only. For example, the person may ignore objects in their left visual field, but may attend to them if their attention is strongly drawn to that side. They may draw a clock face without filling in the numbers on the left side of the clock or fail to shave the left side of the face (Blumenfeld, 2002).

As mentioned previously, *anosagnosia* is most often seen in right hemisphere lesions, however, the syndrome may occur with left hemisphere and frontal lobe disorders. The individual may be completely unaware of neurological defects or illness that have affected the left side of the body. For example, one may be cortically blind or paralyzed on the left side of the body and be unaware that there is a deficit. They may be perplexed as to why they are in the hospital or fail to comprehend that an affected limb even belongs to them. Efforts by others to demonstrate the impairment are futile (Feinberg & Roane, 2003a). Anosagnosia, as described for persons with right hemisphere lesions, almost always presents during the acute phase after injury, when the individual is experiencing confusional behavior (Strub & Black, 1988).

Conclusion

In the rehabilitation setting we are frequently asked whether a problem behavior or behavioral pattern is a direct result of the brain injury. Is there an underlying organic cause for the behavior or is it deliberate, willful, or manipulative? As described here, there are behavioral patterns and syndromes which have been identified as having an organic basis and which can be attributed to the brain injury itself. However, the answer to the question of whether a particular behavior is a direct result of injury is quite complex. Frequently, we don't know for certain. When we don't know, our default approach should be to consider that the behavior is either directly or indirectly associated with the injury. A person with brain injury may engage in a spectrum of deliberate behaviors (adaptive and maladaptive) for the purpose of seeking control when control is threatened (e.g., stress reaction), to meet a basic need, when others are not listening, when all else fails, out of frustration due to other impairments associated with the injury (memory deficits, disorientation, physical limitations), etc. For an individual with a brain injury, more often than not, control is threatened in ways that it was never threatened prior to the injury. Basic needs in terms of interpersonal relationships are frequently not being met. Coping strategies that used to work

are either unavailable after the injury or are no longer effective. In this sense, problem behavior may be indirectly associated with consequences of the injury, but may be deliberate and willful.

Clearly, the goal of rehabilitation professionals, educators, and family members is to teach and help an individual build adaptive coping and social skills that may have been lost as a direct result of the injury. Understanding and empathy are prerequisites for accomplishing this goal. The intent of this article is to build greater understanding for behavioral patterns that have an identified organic cause. However, the list of syndromes described is by no means exhaustive and a great deal of knowledge is yet to be gained in this area. Furthermore, this article does not begin to describe the array of behaviors that are an indirect result of an injury: those that are a product of frustration or lack of resources in getting basic needs met. Behavioral response to injury is influenced by individual differences and characteristics, as well as educational, vocational, or life experiences. Finally, it is important to recognize that all humans, injured or not, engage in maladaptive behavior on occasion due to "basic human error," "trying to get one's way," or "the maturation process." Brain and behavior is a complex topic indeed!



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