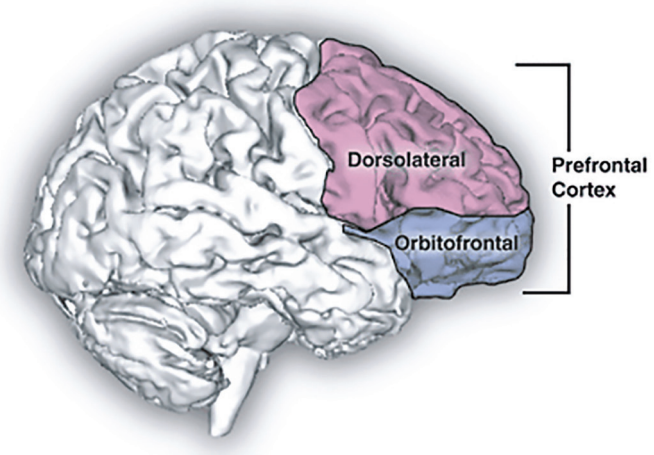
CHAPTER ELEVEN

Neuropsychology of Executive Functions

Happiness is an imaginary condition, formerly attributed by the living to the dead, now usually attributed by adults to children and by children to adults. (Thomas Szasz, author of *The Myth of Mental Illness* (1961).)

The executive functions are a broadly defined domain of neurocognitive behavior that has defied consensus dissection into its constituent processes (Lenartowicz, Kalar, Congdon, & Poldrack, 2010; Packwood, Hodgetts, & Tremblay, 2011). The term executive derives from the function of top executives in an organization. What the top executives do for a company (planning, goal-setting, and so forth), the executive functions do for an individual. The executive functions are mediated by frontal association cortex that has three major neuroanatomical subdivisions: dorsolateral, orbitofrontal, and ventromedial (Alvarez & Emory, 2006). Dorsolateral and orbitofrontal frontal cortex are pictured in Figure 11.1 and ventromedial frontal cortex is shown in Figure 11.3. All three divisions are involved in executive functioning. Association frontal cortex is not modality (i.e., visual, auditory) specific, nor strongly lateralized (i.e., language processing in the dominant hemisphere vs. visual-perceptual processing in the non-dominant), but there is a tendency for language-format executive function tasks to be processed more by the dominant hemisphere and visual-perceptual format executive function tasks to be processed more by the non-dominant hemisphere.

FIGURE 11.1 Dorsolateral and orbitofrontal frontal cortex



Source: "Prefrontal Cortex," by Natalie M. Zahr, PhD and Edith V. Sullivan, Ph.D. 2008, courtesy National Institutes of Health

The term executive function is often used synonymously with frontal lobe function, but this is an over-simplification. The frontal lobes also encompass primary and secondary motor cortices, Broca's area, the frontal eye-fields (involved in eye-gaze; when injured the patient has a fixed eye-gaze toward the side of the lesion), and other regions not designated as frontal association cortex. Executive function

impairment may also result from lesions in subcortical regions of the brain that disrupt input to the association cortex of the frontal lobes in a disconnection syndrome.

Frontal association cortex is sometimes referred to as *prefrontal cortex*. This is a term that dates from the late 1880s, being originally used to refer to that region of the frontal lobes anterior to primary and secondary motor cortex that was "silent" (i.e., produced no observable change in behavior) when stimulated electrically. In modern times the term *prefrontal cortex* has no consistent definition. It is most often used to refer to dorsolateral frontal association cortex, but also has been construed broadly to include orbitofrontal and ventromedial regions of frontal association cortex as well (Preuss, 1995).

Some characterizations of executive functions have used a unitary neurocognitive concept, such as working memory (Baddeley, 1986) or attention. As used in the neuropsychological literature the term attention has multiple meanings, although all definitions are encompassed within the rubric of a limited capacity, executive system. Single executive mechanism models have failed to account for the complexity of behavior governed by frontal association cortex. Alternatively, more complex fractionation schemes have failed to generate consensus classification. A recent literature review of 60 studies identified 68 different subcomponents of executive functions that were reduced to 18 by using statistical techniques and removing semantic overlap (Packwood et al., 2011).

TABLE 11.1 Subcomponents of executive functions and the associated impairment that occurs with brain injury.

Subcomponent of executive functions	Impairment
Self-evaluation of strengths and weaknesses	Anosognosia.
Speed of information processing	Slow information processing speed.
Capacity limitation within working memory*	Limited information processing capacity (difficulty with multitasking).
Resistance to distraction	Difficulty staying focused on task.
Impulsivity	Acting without thinking.
Inhibition	Disinhibited behavior in sex/aggression; utilization behavior.
Goal setting	Lack of congruence between verbal statements and actions.
Initiation	Lack of spontaneity (apathy).
Planning	Difficulty planning for the future.
Cognitive flexibility	Perseveration (repetition of same mistakes).
Organization	Disorganized thinking.
Abstraction	Concrete thought.
Emotional responsiveness	Flat affect

* The term "working memory" in the executive function domain is the same as "short term memory" in the memory domain. Working memory is used in the executive function domain to emphasize its limited capacity.

Table 11.1 shows some widely accepted subcomponents of executive functions, with the proviso that this list is not exhaustive. The general gist of these executive functions is of supervisory cognitive processes involving the most complex level of human behavior. The dorsolateral part of frontal association cortex is most associated with mediating the behaviors on this list. This region is conceived as

operating as a top-down (i.e., exerting control over other parts of the brain), limited capacity system. Orbitofrontal cortex is more linked to social behavior and also processes the pleasantness/averseness of stimuli (Hayes & Northoff, 2011; Wallis & Kennerley, 2010). Ventromedial cortex links cognition with emotion (Gasquoine, 2013). All three subdivisions of frontal association cortex have extensive bidirectional interconnections with other cortical and subcortical structures.

Executive Impairment Following Structural Brain Injury

The very first case of executive impairment following structural brain injury is probably *the* most famous patient in the history of clinical neuropsychology, namely Phineas Gage. At the age of 25 he was employed in Vermont as a railroad construction foreman in charge of firing explosives. On September 13, 1848 an accidental explosion blew a tamping iron through his check and out the top of his head and into the air. He was conscious at the accident scene and could ambulate and communicate. He was treated by a local physician who initially noted no major neurocognitive complaints until at about three weeks post-injury when Phineas developed a frontal brain abscess and was described as demented (Macmillan, 1996). He had lost the sight in one eye and otherwise made a good recovery in terms of sensory, motor, memory, and language skills, but he was given to outbursts of profanity, was unable to follow through on plans, and exhibited other behavioral changes such that friends stated he "was no longer Gage" (Harlow, 1868, p. 340).

Figure 11.2 shows a hypothetical reconstruction of Gage's injury. His skull is preserved at the Warren Anatomical Medical Museum of the Countway Library of Medicine, Harvard Medical School, in Boston, Massachusetts. The extent of his brain injury has been estimated in modern times to primarily include bilateral ventromedial injury.

FIGURE 11.2 Hypothetical reconstruction of the injury to Phineas Gage

Source: "Skull Diagram of Phineas Gage," by John M. Harlow, M.D., 1868, via Wikimedia

BOX 11.1 WHO NEEDS REHAB AFTER BRAIN INJURY?

Despite the absence of any type of brain injury rehabilitation program, Phineas Gage made a relatively good recovery and returned to work first "exhibiting himself" (for which there was not much interest) and later driving stagecoaches (Macmillan & Lena, 2010). He pursued the latter occupation for eight years, seven of which were spent in Chile. He died of an unknown illness on May 21, 1860.

One of the other notable features of the Phineas Gage case was that his injury occurred 13 years before Broca (1861) made the first localization of a complex neurocognitive function within the brain. As noted by De Renzi (1996, p. 123): "It is a recurrent feature of the history of science that for every discovery attributed to an author a forerunner will sooner or later be found."

Executive function impairments that follow structural brain injury include (see Table 11.1): anosognosia (unique in that it requires co-occurring neurocognitive impairment in any one or more of the five domains); slow information processing speed; limited information processing capacity (difficulty with multitasking); distractibility (difficulty staying on task); impulsivity (acting without thinking); disinhibited behaviors, especially pertaining to sexuality or aggression; lack of congruence between verbal statements and actions; lack of spontaneity (apathy); difficulty planning for the future; inflexibility and perseveration (repetitions of the same mistake); disorganized thinking; concrete thinking; and flat affect. Only some of these skills (e.g., processing speed; information processing capacity) can be measured by pencil and paper neuropsychological tests of executive function.

Severe executive impairment can result in *utilization behavior,* whereby the patient will automatically use objects placed before them without instructions to use them (Chapados & Petrides, 2014). For example, if a pitcher of water and an empty glass is placed in front of the patient without any instructions they will fill the glass and take a drink. Similarly, if the patient is presented with a telephone they will pick up the receiver even through it is not ringing. Utilization behavior is indicative of massive brain injury, whereby the patient cannot inhibit his/her automatic response to common stimuli.

More subtle aspects of executive functioning (e.g., planning) are very difficult to assess in a clinician's office and evaluation may have to be based chiefly on the report of a family member or significant other who is familiar with the patient, both pre- and post-injury. There is no "gold standard" neuropsychological measure of orbitofrontal or ventromedial impairment (Zald & Andreotti, 2010).

Interaction of Cognition and Emotion

Managing our emotional responses are part of executive functioning. Emotion is a psychological concept distinct from cognition but there exists a bidirectional relationship between emotion and cognition that is forged primarily in ventromedial frontal cortex. While cognition, specifically the executive functions, can involve conscious thought, emotional behavior is triggered at the unconscious or automatic level of information processing.

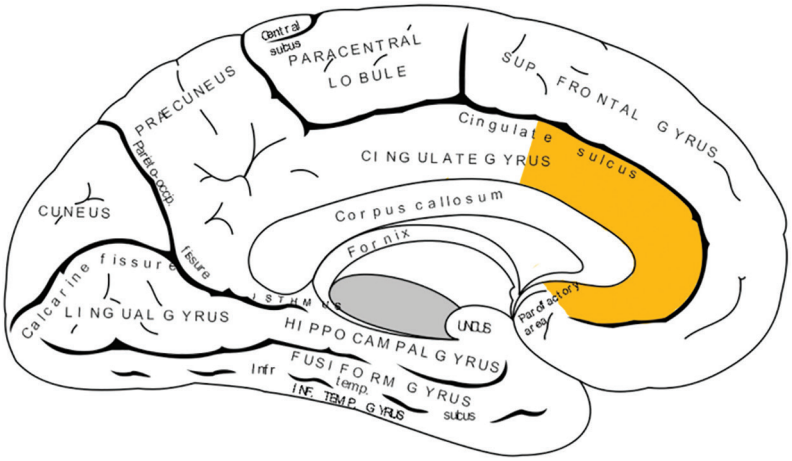
Many adjectives are used to describe our emotions (e.g., angst; passion; remorse; terror; worry), but a fundamental set was first proposed by Darwin (1872/1965) based upon the range of different facial expressions in animals and man. This scheme gives six different basic emotions: (a) fear; (b) anger; (c) sadness; (d) happiness; (e) disgust; and (f) surprise (Ekman & Friesen, 1971).

There is no emotion classification scheme that is based upon distinct physiological responses within the body. This was noted by the father of American psychology William James over 130 years ago. Since that time, numerous unsuccessful attempts have been made to distinguish emotional states on the basis of differing physiological response patterns.

The physiological change associated with the experience of all six basic emotional states involves activation of the sympathetic branch of the autonomic nervous system (see Figure 5.13). Individuals label emotional states differently not by interpreting variation in the internal physiological reaction, but by inferences based upon the behavioral circumstances associated with the increased generalized physiological response (James, 1884). As illustration, if alone in a dark car park late at night activation of the sympathetic branch of the autonomic nervous system is interpreted as fear or anxiety, but if cut off by another car while driving the same physiological response in interpreted as anger.

This is known as the *James-Lange theory* of emotion whereby the brain interprets the physiological reaction and labels the emotional state on the basis of social and behavioral cues (Lange is Carl Lange, a Danish physiologist who developed this idea independently about the same time [1885] as James). The basic concept of the James-Lange theory of emotion is still accepted in modern times (Dalgleish, 2004).

FIGURE 11.3 Ventromedial frontal cortex includes anterior cingulate cortex and adjacent regions superior to the corpus callosum



Source: "Gray 727 Fusiform Gyrus," by Henry Vandyke Carter, from *Anatomy of the Human Body* by Henry Gray, 1918, adapted by Mysid and was_a_bee, via Wikimedia

The contribution of cognition to the experience of emotion and vice versa is mediated through anterior cingulate cortex, a structure in the ventromedial frontal association area that lies immediately superior to the corpus callosum and forms part of the limbic system (see Figure 11.3). Functional neuroimaging studies have shown that anterior cingulate cortex is especially active during cognitively challenging conditions involving conflict, uncertainty, errors, task-switching, and during challenging physical conditions that disturb homeostasis like hunger, thirst, exercise, and hypoglycemia (Gasquoine, 2013). Such challenging cognitive tasks and physical conditions promote activation of the sympathetic branch of the autonomic nervous system that in turn activates insular cortex and anterior cingulate cortex. Insular cortex (see Figure 5.5) acts as primary cortex for the reception of interoceptive information from our internal state, including changes brought about by activation of the sympathetic branch of the

autonomic nervous system (Gasquoine, 2014a). Anterior cingulate cortex then serves to use perceptions of environmental factors to interpret these sensory signals.

Emotions like anxiety are well-known to exert bidirectional effects on executive functioning (e.g., Aarts & Pourtois, 2010). For example, when anxiety reduces efficiency on cognitive tasks, it has been theorized that effortful executive function control in dorsolateral frontal cortex is increased to maintain optimal performance (Eysenck, Derakshan, Santos, & Calvo, 2007).

Decreased Emotional Responsiveness after Structural Brain Injury

It is generally assumed by medical professionals that structural brain injury *should* result in increased emotional distress (anxiety, depression, and/or hostility: Gainotti, 2011) but in reality, this is often not the case. Many patients with structural brain injury are emotionally indifferent and/or have a *flat affect*. Such decreased emotional responsiveness has been, respectively, associated with: (a) anosognosia; and (b) injury to certain locations within the brain.

Ansognosia and anosodiaphoria (indifference)

In cases of bilateral brain injury, such as in trauma, post-injury levels of emotional distress (anxiety, depression, and/or anger) are unrelated to severity of structural brain injury (e.g., Gasquoine & Gibbons, 1994; Prigatano, 1987) or neuropsychological test scores (e.g., Gasquoine, 1997b). In contrast, positive correlations have been found between these levels and patient self-reported awareness of brain injury-related neurocognitive impairment with multiple etiologies, including stroke (Lamb, Anderson, Saling, & Dewey, 2013), Alzheimer disease (Mograbi & Morris, 2013), and traumatic brain injury (Carroll & Coetzer, 2011; Gasquoine, 1992). Many patients with structural brain injury show reduced awareness of neurocognitive impairment (anosognosia) and are emotionally indifference. This indifference reaction was termed *anosodiaphoria* (Babinski, 1914). Although this is correlational evidence, it is consistent with the idea that the indifference reaction results from anosognosia failing to trigger the brain's error detection system in anterior cingulate cortex (Gasquoine, 2016a).

Anosognosia is not associated with injury to any one region of the brain. It can be viewed as resulting from a disconnection syndrome disrupting feedback between injured brain regions governing specific behavioral change (producing the impairment of which the patient has reduced awareness) and the brain's error detection system. Anosognosia is greatest in the acute stages after brain injury.

Anosognosia is conceived as a direct result of the brain injury that impairs the ability of the patient to accurately self-evaluate their impaired neurocognitive skills. There may also be a psychological (non-brain injury-related) component to the emotional indifference in some cases. This is referred to as *denial*. It is well established that neurologically intact adults frequently overestimate their levels of competence across a broad range of behavioral domains (Dunning, Health, & Suls, 2004). Similarly, denial of illness-related information occurs in neurologically intact patient populations with heart disease, cancer, and spinal cord injury (Kortte & Wegener, 2004).

Denial originated with Sigmund Freud as a process that guarded the ego from unacceptable repressed thoughts in psychiatric patients. Later his daughter, Anna Freud, defined denial as a psychological

defense mechanism used to avoid emotional distress by keeping adverse information from conscious awareness (A. Freud, 1936). In denial the patient has explicit impairment-related knowledge available but it is excluded from consciousness. In contrast, neuropathological (i.e., stemmings from the structural injury to the brain) conceptions of anosognosia imply that the missing explicit impairment-related knowledge is unavailable to conscious awareness. Behaviorally, it is not possible to distinguish between psychological (denial) and neuropathological (anosognosia) contributions to reduced awareness of neurocognitive impairment (Halligan, 2006), and both may coexist in any one case.

Flat affect (placidity)

Structural injury in certain brain regions consistently results in decreased emotional responsiveness. This was first associated with the Klüver-Bucy syndrome (see Box 11.2). Placidity, or loss of the fear response was one of six behavioral changes noted by Klüver and Bucy (1939) after they removed both temporal lobes in monkeys. This finding helped initiate the psychosurgical use of bitemporal lobectomy for psychotic behaviors in refractory psychiatric patients (Obrador, 1947). The effect of this type of psychosurgery was "disappointing" (Scoville, Dunsmore, Liberson, Henry, & Pepe, 1953, p. 362) and the treatment was never popularized.

Terzian and Dalle Ore (1955) described the first human case of Klüver-Bucy syndrome following bitemporal lobectomy for seizure control. There was a change in the patient's emotional behavior post-surgery such that he did not exhibit fear and rage reactions, yet prior to the surgery he was given to violent outbursts. Similarly, the famous amnesia case of H. M. who also underwent bitemporal lobectomy for seizure control showed post-surgical placidity (Corkin, 1984). The placidity is often described in humans as *flat affect* and can be regarded as a more severe form of decreased emotional responsiveness than an indifference reaction. It is thought to result from two possible mechanisms of structural brain injury: (a) direct bilateral injury to the amygdala or other limbic structures; or (b) disconnection of those limbic structures from frontal lobe structures. In the second mechanism limbic structures are intact, but are "disinhibited" (Gerstenbrand, Poewe, Aichner, & Saltuari, 1983, p. 417) by disconnection from frontal structures.

The limbic structures whose direct injury is most associated with flat affect are anterior cingulate cortex and the amygdala (see Figure 5.9). Pioneering research in monkeys led to bilateral removal of anterior cingulate cortex as yet another form of psychosurgery (Ward, 1948). This form of psychosurgery is still used to this day to treat a variety of mental disorders including obsessive-compulsive disorder, emotional concomitants of chronic pain, depression, and substance abuse, but its effectiveness is still debated (Brotis, Kapsalaki, Paterakis, Smith, & Fountas, 2009).

The amygdala is considered to process the "threat" value of stimuli. It has been implicated in fear conditioning, an extension of the classical conditioning paradigm of Pavlov, whereby neutral stimuli come to elicit fear reactions when paired with fear producing stimuli (LeDoux, 1995). Overgeneralization within this paradigm is considered responsible for disorders like generalized anxiety disorders whereby patients become fearful of innocuous stimuli and situations. Normally, anxiety is an adaptive response that prevents people from engaging in behavior that has an associated element of risk. Anxiety functions to promote avoidance behavior that in many situations can be highly adaptive. Anxiety disorders arise when the emotional reaction promotes the avoidance of behaviors that have little risk and individuals come to avoid so many situations that it impairs their ability to function.

The most common activity that people fear is public speaking. Public speaking does not have any associated physical risk, but it could result in social embarrassment. The second most common activity that people fear is death.

BOX 11.2 KLÜVER-BUCY SYNDROME

Klüver and Bucy (1939) performed bitemporal lobectomies on 16 rhesus, java, and cebus monkeys that produced six behavioral changes subsequently collectively known as the Klüver-Bucy syndrome: (a) visual agnosia; (b) hyperorality: a tendency to place edible and inedible objects in the mouth; (c) hypermetamorphosis (distractibility): a compulsory urge to respond to visual objects; (d) hypersexuality: increased auto, hetero and homosexual behaviors; (e) altered diet: increased food intake; and (f) placidity: loss of the fear response. Similar findings had been reported 50 years earlier following bilateral temporal lobectomy in monkeys (Brown & Schäfer, 1888), but for some unclear reason they were ignored (Nahm, 1997).

After the first human case of Klüver-Bucy syndrome (excepting hyperorality) was described (Terzian & Dalle Ore, 1955) there were multiple case reports from a wide range of etiologies including: herpes simplex encephalitis; traumatic brain injury; Pick disease; Huntington disease; Alzheimer disease; anoxia; and bithalamic infarction. All these etiologies are capable of producing bilateral temporal lobe injury. Despite the multiple etiologies, reported cases are very rare. Most human cases exhibit a subset of the six classic symptoms with at least three being required for the syndrome to be defined (Lilly, Cummings, Benson, & Frankel, 1983). Neurocognitive impairments commonly associated with temporal lobe dysfunction in humans, especially amnesia and Wernicke's aphasia, frequently co-occur with Klüver-Bucy symptomatology.

Heightened Emotional Distress after Structural Brain Injury

While some level of increased emotional distress is to be expected when an individual experiences a structural brain injury (Gainotti, 2011) in some cases the level of increase is excessive, negatively affecting the patient's ability to function. Location of injury has been investigated as a factor in producing this emotional distress. In a classic paper on the relationship between location of brain injury and post-injury emotional distress, Gainotti (1972) found a higher incidence of post-injury "catastrophic" reactions following unilateral left than right cerebral hemisphere injury (62% and 10%, respectively). In contrast, indifference reactions were more common following unilateral right than left hemisphere injury (38% and 11%, respectively). This was interpreting as showing support for the right cerebral hemispheric control of emotion (the right hemisphere being uninjured when catastrophic reactions occur in left hemisphere injury).

Confounding this interpretation is that unilateral hemispheric injury produces vastly different neurocognitive impairments, namely language impairment with unilateral left hemisphere injury and visual-perceptual impairment with unilateral right hemisphere injury. Thus, it is unclear if the different emotional reactions observed after left and right cerebral hemispheric injury result from the location of the brain injury or from patient reactions to differing neurocognitive impairments. Loss of language skills may be more emotionally distressing than loss of visual-perceptual skills, as the former are more overt.

Nowadays, it is considered that the location of the injury is not so important in producing heightened levels of emotional distress after structural brain injury. Instead, there are three other possible explanations: (a) pseudobulbar affect; (b) preexisting emotional distress; or (c) over-reaction to injury.

Pseudobulbar affect

Pseudobulbar affect is a disorder of emotional expression without accompanying disturbance of feelings that follows structural brain injury. It is characterized by short involuntary crying or laughing spells that occur unpredictably. First described by Darwin (1872/1965), this behavior is known by a number of other names (e.g., emotional lability). The patient will typically report that they do not feel sad when they cry but are unable to control the tears. It frequently accompanies Broca's aphasia (Gainotti, 1972), where the patient cannot express their level of emotional distress, leading family members to interpret the tears as a sign that their relative is severely emotionally distressed. The mechanism of brain injury that results in pseudobulbar affect is unclear although injury to the pathway between the cerebellum and cerebral cortex has been implicated (Parvizi, Anderson, Martin, Damasio, & Damasio, 2001).

Preexisting emotional distress

Heightened levels of emotional distress are common in the general population. Anxiety disorders in particular are one of the most common mental disorders, with onset early in life and a chronic course. While estimates vary considerably, it is thought that the worldwide prevalence is about 10% (Baxter, Scott, Vos, & Whiteford, 2013). If an individual with an anxiety disorder has a brain injury, their anxiety will likely persist after the injury. Similarly, for the other negative emotions of depression and anger.

Over-reaction to injury

In some cases of mild brain injury, the patient over-reports the extent of the neurocognitive impairment they have sustained without chance of apparent financial gain. It is often described as *symptom exaggeration* as patients typically experience a milder version of the symptoms, rather than inventing novel ones from scratch. Such patients are described as being *preoccupied* with their symptoms. They typically also report high levels of emotional distress (Gasquoine, 1997a).

All disease states have similar phenomena. Historically known as hypochondriasis, in the *DSM*-5 (American Psychiatric Association, 2013) the relevant categories are somatic symptom disorder and illness anxiety disorder in the somatoform disorders category. In these disorders, the causes are presumed to be psychological (unconscious to the patient). Rule-out causes include conscious compensation-seeking (e.g., from worker's compensation or disability payments) or avoidance of unwanted activities (these are known as malingering). The diagnosis of hypochondriacal reactions is fraught with difficulty and there have been many cases where medical advances subsequently provided a legitimate medical explanation for the apparent mental disorder.

Summary

There is no universally accepted fractionation scheme for the executive functions. They are mediated by frontal association cortex that has three major neuroanatomical subdivisions: dorsolateral, orbitofrontal, and ventromedial. Association frontal cortex is not modality specific, nor strongly lateralized, but there is a tendency for language-format executive function tasks to be processed more by the dominant hemisphere and visual-perceptual format executive function tasks to be processed more by the non-dominant hemisphere.

The executive functions as mediated by dorsolateral frontal association cortex operate as a top-down (i.e., exerting control over other parts of the brain), limited capacity system. Examples of subcomponents include: working memory; goal setting; planning; inhibition; and speed of processing. Orbitofrontal cortex is more linked to the control of social behavior and also processes the pleasantness/averseness of stimuli. Ventromedial cortex links cognition with emotion.

The most famous case of brain injury producing executive impairment was Phineas Gage. After his 1848 traumatic injury that affected ventromedial frontal association cortex bilaterally, he was given to outbursts of profanity and was unable to follow through on plans.

Emotion is a psychological concept that is distinct from cognition but there exists a bidirectional relationship between emotion and cognition forged primarily in ventromedial frontal association cortex. One popular emotion classification scheme based on the identification of distinct and universally recognized facial expressions gives six different emotions: (a) fear; (b) anger; (c) sadness; (d) happiness; (e) disgust; and (f) surprise. The James-Lange theory of emotion holds that physiological changes associated with the sympathetic branch of the autonomic nervous system mediates all six emotions and individuals differentiate emotional states not by the nature of the internal physiological reaction, but by interpreting the accompanying behavioral circumstances.

The contribution of cognition to the experience of emotion and vice versa is mediated through anterior cingulate and insular cortices. Anterior cingulate cortex is especially active during cognitively and physically challenging conditions that increase sympathetic autonomic nervous system activity levels to facilitate the behavioral response. Insular cortex acts as primary cortex for the reception of interoceptive information from our internal state, including activation of the sympathetic branch of the autonomic nervous system. Anterior cingulate cortex then serves to consciously interpret these automatic sensory signals.

Some increase in emotional distress (anxiety, depression, and/or hostility) is expected after structural brain injury, but often the emotional reaction of the patient is either decreased or increased over what is expected. Decreased emotional responsiveness is known as anosodiaphoria (indifference) or flat affect. These reactions have been, respectively, associated with: (a) anosognosia, resulting from failure of the brain's error detection system, possibly associated with a psychological reaction of denial; and (b) injury to anterior cingulate cortex and/or the amygdala. Heightened levels of emotion distress can also occur after structural brain injury with three possible explanations: (a) pseudobulbar affect that is characterized by short involuntary crying or laughing spells that occur unpredictably. It is an impairment of emotional control distinct from emotional feeling; (b) preexisting emotional distress; or (c) with mild brain injury, pre-occupation with and exaggeration of self-reported symptoms.