Chapter 10
Frontal Lobe/Executive Functioning

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Abstract The frontal lobes represent a large area, consuming approximately one-third of the cortical surface of the brain. This area is involved directly and indirectly across a wide spectrum of human thought, behavior and emotions. The irony of the frontal lobes may best be described as the area of the brain we know the most about but understand the least. For example, frontal lobe functioning involves simple motor skills (both gross and fine), complex motor skills, sequenced motor skills, inhibition of motor skills and automatic motor skills, and these may be the simplest of the functions of the frontal lobes. The frontal lobes also subsume what is collectively referred to as executive skills. These functions include attention, reasoning, judgment, problem solving, creativity, emotional regulation, impulse control and awareness of aspects of one's and others' functioning. In this chapter, we will briefly discuss the anatomy of the frontal lobes, the basic and complex functions of the frontal lobes, and the informal assessment of frontal lobe functions.

Key Points and Chapter Summary

- Frontal lobes include a large area of the cortex and are involved directly or indirectly in most brain functions involving cognition, behavioral, and motor skills
- Frontal lobe damage can have profound effects on attention, memory, language, problem solving/reasoning, and general comportment (personality/social behaviors, etc.)
- Frontal lobe damage can be grouped into three syndromes determined by anatomical regions involved and associated with characteristic cognitive,

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Key Points and Chapter Summary (continued)

behavioral and/or mood symptoms: (1) Dorsolateral, (2) Orbitofrontal, and (3) Medial frontal syndromes
- Behavioral and personality changes are often the most profound change seen in Frontal Lobe injuries and are not well measured by standardized tests
- Cognition may be minimally impaired on standardized tests administered in controlled environments that minimize distraction and maximize motivation, particularly on tests which emphasize previously acquired knowledge

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Anatomy of the Frontal Lobes

The frontal lobes represent the cerebral cortex anterior of the central sulcus, and accounts for 1/3 of the entire human neocortex, but represents more than a third of the cortical surface. The frontal lobe has been described in multitude of systems and areas, but we will review the frontal lobes in terms of basic functional organizations. The frontal lobes can be divided into three broad categories: (1) Primary motor cortex, (2) Premotor and supplementary motor cortex, and (3) Prefrontal cortex (see Figs. 10.1–10.3). The prefrontal cortex is often subdivided into three
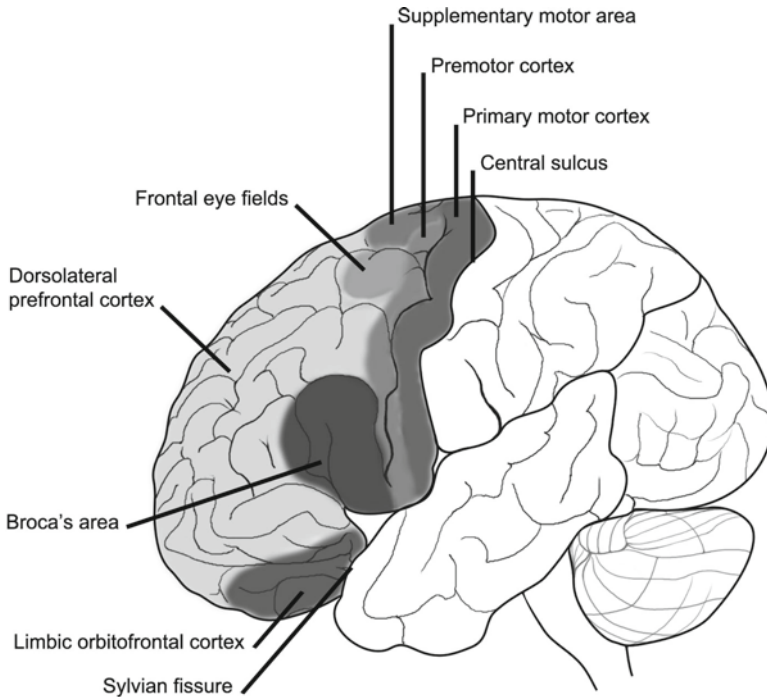

Fig. 10.1 Lateral view of the frontal lobe including primary motor, premotor, visual eye field, Broca's areas (Brodmann's area 44) and prefrontal region
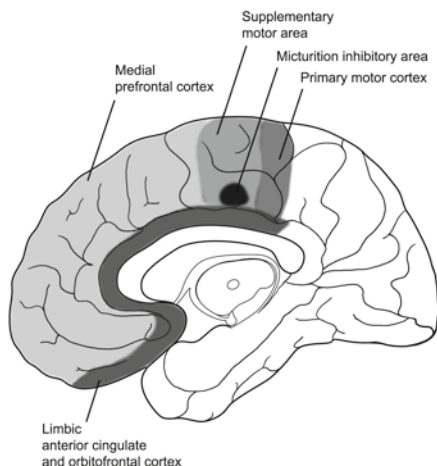
Fig. 10.2 Mesial view of the frontal lobe including primary motor, supplementary motor, micturation center, and prefrontal cortex/anterior cingulate
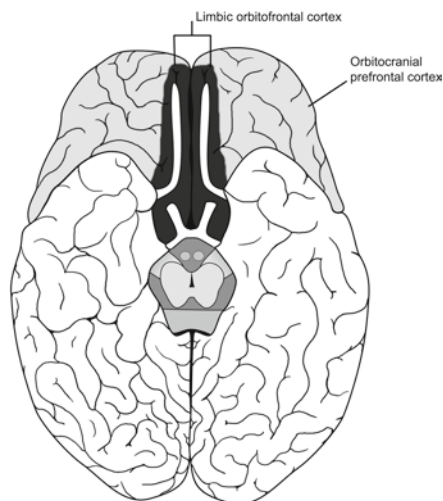

Fig. 10.3 Orbitofrontal (inferior frontal) view of the frontal lobe

functional domains, although some authors report two prefrontal functional domains. The three traditional prefrontal domains are: (a) dorsolateral prefrontal, (b) orbitofrontal (inferior or ventral frontal lobe), and (c) the medial frontal/anterior cingulate. The prefrontal cortex derived its name because this area of the frontal lobe received inputs (afferent fibers) from the dorsomedial nucleus of the thalamus. The prefrontal cortex also has extensive afferent and efferent connections to the temporal, parietal, and occipital lobes as well as reverberating (input and output) fibers to subcortical regions, including the basal ganglia, thalamus, hypothalamus, and tegmentum. The prefrontal projects to, but does not receive input from, the basal ganglia. Combined, the frontal lobe has classically been divided into six functional subdivisions (considering Broca's area, a separate subdivision of the premotor/supplementary area like the Frontal Eye Fields would yield seven subdivisions) (see Mesulam 2000; Salloway et al. 2001, for reviews):

1. Primary motor cortex
2. Premotor (supplementary) motor cortex
   (a) Broca's area
3. Frontal Eye Fields
4. Dorsolateral Frontal
5. Orbitofrontal (Inferior) Frontal
6. Medial frontal/anterior cingulate

Below, we will briefly review the functional neuroanatomy of each subdivision, and how lesions of the frontal lobes may present symptomatically within each functional area, and conclude with an overview of neuropsychological assessment for frontal lobe functions.

Primary Motor Cortex

The primary motor cortex is the most posterior aspect of the frontal lobes (precentral gyrus), and contains a motor "humunuclus" representing a symatotopic representation of motor function for the contralateral body that is upside down (e.g., head towards the temporal lobe while the trunk is near the superior convexity and the legs are represented within the medial aspect of the pre-central gyrus lying within the interhemispheric fissure) (see Figs. 10.1, 10.2, 10.4, 10.5 and 10.6). The primary motor cortex is frequently termed the "motor strip" and is Brodmann's area 6. The primary motor cortex has efferent projections to the spinal cord and cranial nerve nuclei as well as the basal ganglia and red nuclei, forming part of the corticospinal or corticobulbar tracts, respectively. The initiation of the corticospinal and corticobulbar tracts is the premotor cortex (see below and Chap. 3). The primary motor cortex receives input from the premotor/supplementary motor cortex areas.

Lesions involving the primary motor cortex will result in contralateral motor weakness. Initially, the motor weakness may present as a flaccid hemiplegia (complete lack of motor strength), but strength will often recover to some extent, particularly if premotor and supplementary motor areas are preserved. Larger lesions may resolve into a spastic hemiparesis and smaller lesions may resolve into incoordination and mild hemiparesis which can be difficult to identify without careful examination.

Primary Facial Motor Cortex

The primary motor area involved in facial control (recall the upper part of the face is innervated bilaterally by the facial nerve) has some unique aspects summarized below. The primary motor cortex of the face is just superior to the perisylvian fissure and anterior to the central sulcus. Each hemisphere controls the contralateral half of the face (facial region above the eyes is controlled by both contralateral cortical and ipsilateral cranial nerve function). Focal damage to the language dominant (left) primary motor facial area is typically described as resulting in an expressive deficit (impaired receptive language but intact comprehension) thought to reflect an *oral apraxia*, along with contralateral hemiplegia of the lower face (Kolb and Whishaw 2009). The oral apraxia is the inability to coordinate the muscle movements necessary for speech production. Expressive speech deficits can also include agraphia (inability to write), thought to reflect damage to the closely situated supplementary area for fine motor movements of the hand. However, focal lesions can result in an initial global aphasia (impaired expressive and receptive speech). Patients with surgical removal of pre- and post-central gyrus involving the facial area have demonstrated recovery of facial expression usually within a month of surgery. However, recovery of speech is more gradual, and while speech production grossly recovers, more careful evaluation has revealed more profound residual impairments of generative verbal fluency, phonetic discrimination, spelling, and figural fluency. Remarkably, individuals with focal damage to the nondominant

(right) primary facial motor cortex have exhibited chronic deficits in figural fluency to a greater extent than individuals with more extensive prefrontal nondominant (right) frontal damage. Deficits in verbal (and possibly figural) generative fluency might represent deficits in the motor preplanning needed for these tasks (Salloway et al. 2001).

Rule of thumb: Divisions of the frontal lobe

- Primary Motor
- Premotor/supplementary motor
  - Frontal Eye Fields
  - Broca's area
- Prefrontal
  - Dorsolateral
  - Orbitofrontal
  - Medial frontal/anterior cingulate

Premotor and Supplemental Motor

The premotor and supplemental motor cortex areas are involved in fine motor movements and sequenced motor movement such as writing or fastening buttons.
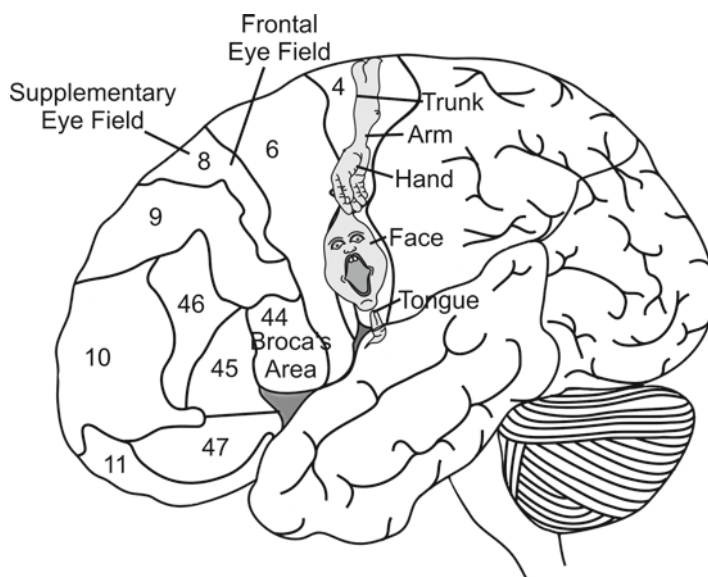

Fig. 10.4 Left dorsolateral prefrontal cortex including Brodmann's areas of dorsolateral cortex
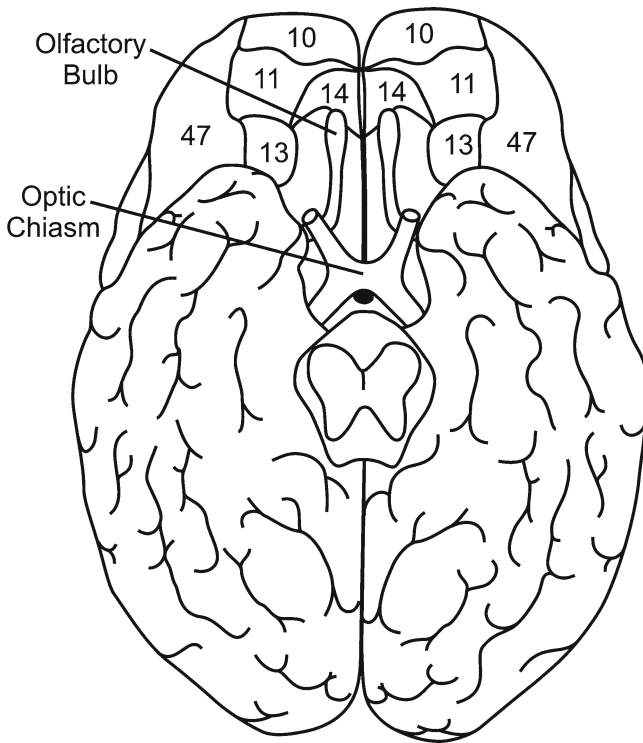
Fig. 10.5 Orbitofrontal/inferior frontal prefrontal cortex, including Brodmann's areas making up orbitofrontal areas

The premotor and supplementary motor cortices lie just anterior to the primary motor cortex, and includes Brodmann's areas 6 and 8. While many areas of the brain are involved in producing smooth, coordinated motor movements (i.e., the cerebellum and basal ganglia; see Chap. 3 for more details), the unique aspect of

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Rule of thumb: Primary motor cortex

- Mediates contralateral Motor Movement
- Receives inputs from cerebellum, basal ganglia, supplemental motor cortex
- Projections form part of corticospinal and corticobulbar tracts
- Lesions produce contralateral motor weakness (hemiplegia or hemiparesis)
- Facial Primary Motor cortex is unique
  - Dominant hemisphere lesions cause expressive aphasia features (oral apraxia) with long-standing residual deficits in: generative verbal fluency, phonemic awareness, and spelling

the premotor/supplemental motor frontal cortex appears critical in two ways: (1) involved in acquiring novel motor skills which have not become overlearned, and (2) sequencing necessary motor movements. The premotor/supplementary area has projections directly to the cortico spinal and corticobulbar tracts, but *primarily* have connections to and from the basal ganglia. There are also projections to the primary motor cortex and thalamus. In addition to basal ganglia and thalamus, premotor and supplementary motor cortices receive input (afferent tracts) from the parietal and dorsolateral cortex. Thus, premotor and supplementary motor areas are able to execute complex motor actions and continually adjust and fine tune motor activity. Perhaps a simplistic example is the motor movement necessary for learning to ride a bicycle. Initially, it takes much more effort to focus on the conscious motor movements necessary for balance, propulsion and steering. This motor movement is not initially automatic, and the sequence of balance, pedal and steer is difficult to master and initially requires substantial prefrontal resources. With experience, these motor and sequencing aspects evolve into an automatic sequence of motor skills resulting in a complex behavior. As this occurs, the premotor cortex is less involved and other areas of the brain (cerebellum, parietal cortex) are more involved.

Lesions to the premotor and supplementary motor areas 6 (not involving the frontal eye fields, area 8) typically will result in motor apraxias of the contralateral body/limb, and not hemiparesis. Individuals will also have difficulty synthesizing sensory information into complex motor movements and complex motor sequencing will be incoordinated and may appear "choppy" or clumsy. However, simple motor movements remain fluid (not ataxic).

Broca's Area (Brodmann's Area 44 and 45)

Language production is also a function of the frontal lobes. For most individuals, the left lateral premotor area of the frontal lobe (i.e., Broca's area, Brodmann's area 44 and 45, see Chap. 3) controls expressive language. Lesions in this area can produce expressive aphasias or more subtle language impairment such as decreased verbal fluency and writing (see also Chaps. 7 and 16) or word-finding deficits (dysnomia). The right (nondominant) frontal lobe is less concerned with the actual production of speech, but rather contributes to expressive language *prosody* (see also Chaps. 7 and 16). Prosody refers to the vocal amplitude, tone and inflection that communicate nonsemantic meaning in vocal expressions such as emotion, questioning, confidence, lethargy, etc.

Lesions to Broca's area in the dominant hemisphere will result in loss of expressive speech. Depending upon the extent of damage, repetition may also be disrupted. Lesions to the nondominant hemisphere result in difficulties with expressive prosody (expressive aprosdy, see Chaps. 7 and 12 for details). Briefly, speech may sound monotone to others. Several different patterns of aphasia have been identified and extensively studied. These are reviewed in detail in Chap. 12, see also Chap. 7.

Rule of thumb: Pre-motor and supplementary cortex

- Anterior to primary motor cortex
- Beginning of the corticospinal and corticobulbar tracts
- Involved in production of complex movements and motor programming
- Connections with basal ganglia, parietal cortex, primary motor cortex, thalamus, and dorsolateral prefrontal cortex
- Lesions result in apraxias and discoordinated movement
- Broca's area, the expressive language center, in dominant hemisphere
- Frontal Eye fields involved in volitional eye movements and visual attention

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Frontal Eye Fields

The premotor cortex also includes a region referred to as the frontal eye fields, which are involved in voluntary eye movements and fixation of gaze important for novel visually guided activities and visual attention. The frontal eye fields direct visual focus to central elements in an environment that allows us to successfully execute sequences of behaviors. The frontal eye fields (Brodmann's areas 8 and 8A) are anterior to the primary motor cortex. This area of supplementary motor cortex has afferent and efferent tracts to regions of the brain important for controlling eye movements, including the posterior parietal regions and the superior colliculus. As a simplistic act of dressing illustrates, one must first search out articles of clothing, locate each in space, plan motor movement to get each article, manipulate each article prior to putting it on and then successfully execute dressing, including fastening, buttoning and zipping. While many of these behaviors can become automatic through overlearning and repetition, the frontal eye fields must direct vision to each aspect for a successful behavior to be completed. Contrast this simple example with the visual demands in extracting or debulking a tumor located in the frontal lobe of a patient and you quickly become aware of the demands placed on the frontal eye fields every day.

Prefrontal Cortex (Dorsolateral, Orbitofrontal, and Medial Frontal/Cingulate Gyrus)

The Dorsolateral, Orbitofrontal (inferior or ventral frontal lobe) and Medial Frontal/Cingulate gyrus (anterior portion) areas compose the prefrontal cortex. The prefrontal cortex is comprised of Brodmann's areas 8, 9, 10, 44, 45, 46, and 47 on the lateral side, Brodmann's areas 10, 11, 12, 13, 14, and part of 45 on the ventral (inferior) side, and Brodmann's areas 8, 9, 10, 11, 13, and 32 on the mesial (medial) side (see Figs. 10.4–10.6, respectively). These areas have been
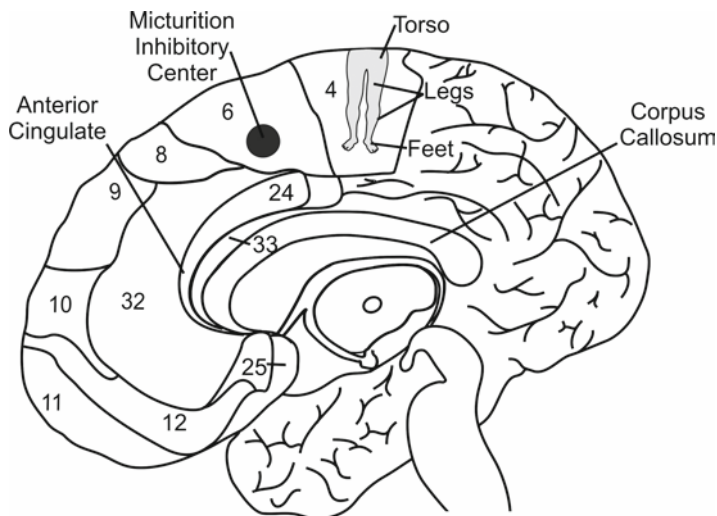
Fig. 10.6 Mesial frontal/anterior cingulate cortex including Brodmann's areas of the mesial frontal/
anterior cingulate

subdivided into various regions, most commonly; the dorsolateral, orbitofrontal (inferior or ventral frontal lobe) and medial frontal/cingulate gyrus (anterior portion) areas. Another classification scheme involves the anterior prefrontal, medial prefrontal, and ventrolateral prefrontal regions. The roles and demarcations of these frontal lobe areas are less definitively agreed upon and more difficult to describe. Part of this difficulty arises from these functions being impaired or affected to differing degrees by injury to the frontal lobe cortex. These functions are often collectively referred to as "executive functions" and pertain to high level or complex aspects of cognitive, behavioral and emotional aspects of human behavior. These functions are necessarily complex and dependent on many regions of the brain. The proceeding description will necessarily be general and intended to give examples of the frontal lobe influences on these cognitive, behavioral and emotional aspects of frontal lobe functions. Table 10.1 summarizes the role of the frontal lobes.

Symptoms of Frontal Lobe Dysfunction: The "Frontal Lobe" Patient

It is not uncommon for a health care provider to mention "oh, that individual is pretty frontal" or "that individual has a frontal lobe syndrome". But what does that mean, and how is that determined? We will begin the behavioral syndrome review of frontal lobe disease with an overview of more generalized or diffuse prefrontal dysfunction

Table 10.1 Frontal lobe and executive functions

Domain	Region	Deficit
Motor	Left motor strip	Right gross, fine and coordination motor deficits
	Right motor strip	Left gross, fine and coordination deficits
	Left pre-motor	Poor contralateral sequencing and novel motor acquisition skills
	Right pre-motor	Poor contralateral sequencing and novel motor acquisition skills
	Frontal eye field	Contralateral voluntary eye movement/coordination
Cognitive	Diffuse frontal	Attentional deficits in sustained and voluntary alternation of attentional focus. Learning and retrieval.
	Posterior left frontal	Expressive language, naming, word finding, fluency
	Anterior left frontal	Verbal reasoning, verbal problem solving, sequencing, reduced generative verbal fluency
	Posterior right frontal	Expressive prosody
	Anterior right frontal	Visuospatial reasoning, visual problem solving, sequencing
Behavior/Emotional	Orbital frontal	Disinhibition, poor social skills, impulsivity, hyperactivity, emotional overreactivity.
	Dorsolateral frontal	Impaired problem solving, concrete, environmental dependency (stimulus bound behaviors), perseveration, poor sequencing, lack of self-monitoring for errors and self-correction. Impaired memory with reduced working memory, poor memory for temporal sequence of events, poor retrieval strategies with intact recognition.
	Medial frontal	Apathy, akinetic, mutism, leg weakness contralateral to lesion (may be bilateral). Abulia, apathy, socially disengaged, indifference, lack of initiation, emotionally underreactive with intermittent dysregulation.

and then turn to a description of selected "Frontal Lobe Syndromes." Table 10.1 summarizes the predominant roles of the frontal lobes and associated deficits.

Prefrontal Cortices (General/Diffuse Symptoms)

Many cognitive functions are mediated either directly or indirectly by the frontal lobes (dorsal lateral, medial, and orbital cortex) (see Kolb and Whishaw 2009; Lezak et al. 2004; Mesulam 2000; Salloway et al. 2001, for reviews). An overriding function involves many voluntary aspects of attention. Specifically, the ability to attend to relevant aspects of our environment and inhibit being distracted by incidental environmental stimuli is an important cognitive function of the frontal lobes. Failure of this function often has devastating results for individuals and usually is manifest as *tangentiality* or *circumloquaciousness* in language or distractibility in performing other tasks. The extent of the influence that voluntary attentional control can produce on other observed or measured skills should not be underestimated. In fact, voluntary attentional control is a prerequisite skill in everything from speaking to cooking and dressing. This function is frequently tested in terms of simple attention, sustained attention and voluntary rapid alternation of attention.

Environmental dependency (and *utilization behaviors*) can often be observed in patients with frontal lobe damage. Patients with environmental dependency respond in the usual way to a stimulus regardless of the appropriateness of the environmental situation. Environmentally bound behaviors can be initiated by an object, persons or situations. They may also be initiated by their own poorly inhibited thought processes. For example, patients may respond sexually to personnel who look at them and smile or respond angrily to personnel who make a request of them. Similarly, environmental cues such as the counter at a nursing station may solicit a patient to order auto parts because of its similarity to an auto parts store. Despite instruction not to shake an examiner's hand, this behavior can be elicited (hand shaking) by offering to shake the patient's hand. An internal trigger, such as a need to urinate, can elicit a patient to urinate in a potted plant. *Utilization behaviors* are a subtype of environmental dependency, and reflect the spontaneous use of an object without apparent need or desire. Examples of utilization behaviors can often be easily initiated by having a hairbrush, toothbrush, pen/pencil, comb, or cup/glass within reach of a patient with frontal lobe damage. Patients with utilization behaviors will, despite directions to not touch the items, reach out for the object(s) and begin using the object(s). For example, a patient may begin brushing his/her teeth without toothpaste or a sink or begin brushing his/her hair. Patients may "drink" from an empty cup or begin writing on a desk with a pen/pencil. These patients often demonstrate remorse for inappropriate behavior or verbal recognition of inappropriate behavior when their behavior is confronted, but will be unable to inhibit the behavior if the environmental trigger presents again.

Autonoetic awareness (Tulving 2002) is a term to describe self-awareness which has been defined as the autobiographical temporal continuum which is able to affect behavior through one's past personal experiences and goals. Patients with prefrontal damage in general, but particularly those with orbitofrontal damage, frequently exhibit deficits in autonoetic awareness (self-awareness) such that they have difficulty with self-regulation of behavior. Patients are unable to reference behaviors to past experiences or goals.

Memory functioning can be adversely affected by lesions of the prefrontal cortex. These are reviewed within each frontal lobe functional domain below. However, a hemispheric difference in memory functions involving frontal lobe regions has been identified and termed *Hemispheric Encoding and Retrieval Asymmetry* (HERA; see Tulving et al. 1994). This model posits the left prefrontal cortex is more involved during encoding of episodic and semantic memory material and less involved during retrieval. The right prefrontal cortex (and insula and parietal cortices) are thought to be more involved in retrieval of episodic memory material.

Paratonia (*Gegenhalten)* may be found with bilateral frontal lobe damage. Paratonia is presentation of increasing muscle tone to oppose efforts to passively move the limb by someone else. This can often be misinterpreted as purposeful noncompliance. Paratonia (gegenhalten) is load- and velocity-dependent resistance, and is outside the control of the patient. While it may be found following bilateral diffuse frontal lobe lesions, it can also be present following bilateral basal temporal lobe damage and more diffuse brain damage, including dementias and encephalopathies.

Several *neuropsychiatric syndromes* are associated with frontal lobe damage. *Personality changes* are commonly reported, particularly with orbitofrontal damage but also with dorsolateral or medial frontal damage (see below). Orbitofrontal personality changes are frequently described as overactive/manic, uncaring, narcissistic, and pleasure seeking. Commonly, these individuals are described as disinhibited and impulsive. Emotionally, patients generally have poor emotional regulation, but have a tendency to be overly reactive, in which their emotional response to a situation is often much greater than what might be anticipated or warranted. Individuals with orbito-frontal damage have also been labeled as *pseudopsychopathic* because of their personality changes noted above and apparent disregard for the feelings of others. Patients with dorsolateral or medial frontal damage may be perceived as indecisive, lazy, amotivated, apathetic, or passive. Some may be incorrectly described as "depressed." Indeed, the dorso-lateral syndrome has been referred to as *pseudo-depressive* or *apathetic* due to their appearance of indifference and abulia. Emotionally, patients with dorso-lateral damage tend to exhibit a propensity toward emotional underreactivity with variability or fluctuations when they do become emotionally engaged. Frequently, these individuals will exhibit emotional dysregulation that fluctuates from indifference to overreaction. Patients with mesial frontal damage also exhibit indifference, but exhibit more akinetic qualities, in which these patients may just sit motionless for hours and be potentially mute. These individuals have been incorrectly diagnosed as having catatonic schizophrenia. Other neuropsychiatric syndromes associated with frontal lobe damage include *reduplicative paramnesia* and *Capgras syn-*

drome. Reduplicative paramnesia is the delusion that a place or an object (clothes, furniture, house, food, etc.) has been exactly duplicated. Capgrass syndrome is the "imposter" delusion, such that the patient believes a person (or persons) has (have) been duplicated, and the person claiming to be the person is an imposter.

Finally, several frontal lobe reflexes (or release signs) normally present in infants are considered pathological in adult patients. While typically not present in adults, some of the reflexes may be present and the presence of one should not be interpreted as pathognomic of frontal lobe dysfunction. Among the reflexes, the grasp reflex is less often encountered in normal adults. If frontal release signs are present, there likely is frontal lobe dysfunction. The frontal release signs include: Glabbelar, Grasp, Palmomental, Root, Snout, and Suck reflexes which are briefly described:

- *Glabbelar reflex*. Failure to extinguish eye blink response to gentle tapping to the center of the forehead right above the nose.
- *Grasp reflex*. Perhaps the most helpful frontal release sign, as it is fairly specific of frontal lobe injury, and has localizing value to the contralateral supplementary motor area located in the medial frontal lobe. The grasp reflex occurs when the hand grasps onto an object (or examiner's finger). It is elicited by stroking the inside palm in a distal motion towards the base of the fingers. One may also stroke the proximal surface of the fingers (towards the palm). The grasp can be quite strong, allowing the person's torso to be lifted up from a lying position. Release may be voluntary or in some cases, takes considerable effort to release.
- *Palmomental reflex*. Ipsilateral contraction of the muscle of the chin (mentalis muscle) occurring to an unpleasant stimulus of the thenar eminence (body of the palm just proximal to the thumb). The ipsilateral corner of the mouth may also contract. The stimulus eliciting the reflex is started at the lower wrist and up the base of the thumb. The stimulus can be a tongue depressor or the handle of a reflex hammer.
- *Root reflex*. The turning of the patient's head ipsilateral to the side of the cheek that is lightly stroked. It is associated with the suck reflex in its adaptability for infants to breast feed.
- *Snout reflex*. The puckering of the lips to make a "snout" when the top lip is gently tapped (percussed). Typically, the Snout reflex can be elicited by gently tapping on the center of the upper lip when the lips are closed with your finger.

Rule of thumb: Frontal lobe reflexes

- Frontal Lobe Reflexes are normal in very young children and are suppressed through development
- Frontal Lobe reflexes are often seen in acute injuries or in severely impaired patients, but may not be seen in sub-acute populations or less severely impaired patients who continue to have cognitive, behavioral or motoric frontal lobe symptoms

• *Suck reflex*. Sucking movements of the lips when the lips are generally stroked or touched. The sucking movement can be elicited by stroking the upper or lower corners of the mouth.

Following a brief review of symptoms reflecting more generalized or diffuse dysfunction, we will review the traditional three prefrontal syndromes: (1) dorsolateral, (2) orbitofrontal, and (3) medial frontal. Another viewpoint that specifies two frontal lobe syndromes: (a) *syndrome of frontal abulia* and (b) *syndrome of frontal disinhibition* (e.g., Mesulam 2000) will be reviewed as well. The syndrome of frontal abulia has been associated with the dorsolateral frontal lobe syndrome while the syndrome of frontal disinhibition collapses the orbitofrontal and medial frontal syndromes together, as features of both are often observed in a patient (Mesulam 2000).

Rule of thumb: Frontal lobe syndromes

• Dorsolateral damage=dysexecutive syndrome also pseudodepressed syndrome
  – Poor problem solving, abulia/amotivational, perseverative, stimulus bound
• Orbitofrontal (inferior/ventral frontal)=Disinhibited/pseudopsychopathic syndrome
  – Disinhibited, emotional lability, impulsivity, lack of social graces, personality changes, poor smell discrimination
• Medial Frontal/Cingulate gyrus=akinetic/apathetic syndrome
  – Akinetic, amotivation, mute (if bilateral), leg weakness, urinary incontinence

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Frontal Lobe Syndromes: Traditional Three (3) Syndrome Model

The three traditional frontal lobe regions have neuroanatomical connections to discrete areas of shared basal ganglia and thalamic nuclei structures. Each of the three systems have segregated tracts, but operate in parallel and have multiple areas where input and output of each region is directed to other parts of the prefrontal cortex and brain. The three systems are thought to process distinct cognitive and emotional information incorporating multiple sensory and motor information from other brain regions.

Dorsolateral Prefrontal (Dysexecutive or Frontal Convexity) Syndrome

Patients often appear distractible, apathetic and "depressed," but also have difficulty reasoning, problem solving, shifting attention, and maintaining a behavior for completion of a task (impersistence). Patients may exhibit environmental dependency and memory problems. The memory problems (detailed below) often reflect

difficulty remembering the temporal sequence of when events occurred (as opposed to forgetting something occurred altogether). Learning rate is often slow, and the patient may not remember some things due to reduced working memory/attention but also reduced retrieval. Depending upon extent of lesion, motor weakness of the upper extremity contralateral to the lesion may be present.

Sequencing, problem solving and reasoning is impaired. The patient can have considerable difficulty with three-step motor sequencing along with sequencing figures or shapes (see Appendix). Problem solving and reasoning is concrete, but tends to be worse with divergent reasoning (reasoning that requires many possible solutions or answers) than convergent reasoning (drawing similarities or solutions from two or more things). For example, patients would have problems listing the uses for a brick. Besides the obvious use for construction, other uses might include a door stop, stepping stones, hammer, a paper weight, an exercise tool, etc. While many frontal lobe patients have difficulty with divergent reasoning, fewer problems may be found on the convergent reasoning tasks on many intelligence tests. Patients with dorsolateral damage have difficulty solving problems that require sequential steps for problem resolution. Failure reflects difficulty selecting the series of appropriate steps to reach a problem resolution. Their responses in such situations are often simplistic and fail to appreciate the complexities involved in the situation. For example, a patient discovering his/her refrigerator has quit working may recognize the need to have it repaired, but will not spontaneously appreciate the need to use, relocate or otherwise dispose of the contents. The patient will, however, answer correctly when prompted about the contents.

Insight and judgment is often poor. Individuals typically do not appreciate the depth or extent of their cognitive compromises. While patients are able to appreciate or recognize direct failure, they have a diminished capacity to appreciate the degree or extent of their deficits and anticipate the impact it is likely to have on their future performance despite previous failure. This often leads to an astonishing repetition of attempts and failures that are resistant to making adjustments to future attempts. They may exhibit perseveration or difficulty switching sets when they become engaged. Alternatively, persistence in completing a task (particularly one they are not interested in for themselves) can be reduced, especially when distracters are present in the environment. As an example, a patient spouse may say the patient will start a task if asked, but will get easily distracted and will not finish the task unless repeated requests and redirection to complete the task is given. Patients often exhibit environmental dependency. Verbal output is often reduced, and phrase length is typically shorter.

Memory can be worsened due to the disruption of several systems involved in learning and memory. First, working memory may be reduced and the patient more distractable. Working memory reflects two general processes, the maintenance of information in an attentional "store" and, the ability to manipulate this "online" material. Second, strategies for active learning and efficient encoding and retrieval may be disrupted. Third, memory for facts or events may be out of temporal sequence. Memory encoding of unrelated material is often poor, exhibiting a reduced learning curve. Spontaneous recall is often impaired, particularly for material not semantically (contextually) organized. Alternatively, encoding of material

provided to the patient already semantically (contextually) organized that makes sense to the patient can improve encoding and retrieval. Recognition cues often improves recall. However, the temporal organization of memory is also frequently disrupted. Thus, patients may remember something happening a month ago as occurring yesterday. While the recall of events and situations may be perfectly reasonable, review of the patient's medical chart and/or report from a reliable collateral informant (spouse, adult child, friend, etc.) will quickly identify the poor temporal organization of memory.

Patients frequently appear to be emotionally blunted, apathetic, and abulic (hence the term "pseudodepressed"). However, when emotionally aroused, patients often exhibit difficulty regulating emotional expression, appearing to both over- and underreact to various situations. Patients will not be as behaviorally disinhibited as patients with orbito-frontal syndrome, they demonstrate attentional and initiation deficits that are no less impairing.

This presentation may reflect the dorsolateral (dysexecutive or frontal convexity) syndrome. Because of prominent deficits in problem solving, reasoning, and switching sets, this is known as the Dysexecutive syndrome. It has also been termed the pseudo-depressed syndrome due to patients often exhibiting apathy and abulia.

Anatomy: The dorsolateral cortex is anterior to the premotor and supplementary cortices and is also termed the frontal convexities, and includes Brodmann's areas 9 and 46 (see Fig. 10.4). Some also include the lateral aspects of Brodmann's area 10 (the anterior prefrontal or frontopolar region). This area of the cortex predominately has reciprocal projections from the posterior parietal and superior temporal sulcus. The dorsolateral prefrontal area also projects to the basal ganglia, thalamus, cingulate gyrus, and superior colliculus. The dorsolateral frontal region projects to the dorsolateral caudate nucleus which projects to the lateral and medial globus pallidus, which projects to the dorsomedial and ventral anterior thalamic nuclei and back to the prefrontal cortex. Note the dorsolateral cortex does not receive inputs from the striatum directly, but rather projections from the thalamus. The dorsolateral prefrontal areas are involved in complex human cognitive and behavioral func-

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Rule of thumb: Dorsolateral frontal lobe damage=Dysexecutive syndrome

- Poor problem solving (concrete and rigid)
- Poor organizational strategies
- Impaired set-shifting, perseveration, and impersistence
- Memory may be disrupted
    - Reduced working memory, encoding/retrieval strategies, and temporal organization (order). Retrieval improved with recognition cues.
- Apathy and psychomotor slowing. Poor motivation/Abulia
- Decreased emotional range. May appear uncaring or emotionally unresponsive
- Contralateral upper extremity weakness

tions for making decisions, problem solving, sequencing and organizing behaviors. It is also associated with some attention and memory functions.

Causes of damage to the dorsolateral cortex are often the result of blunt trauma, occlusion of the anterior branch of the middle cerebral artery (or hemorrhage of ACA or MCA affecting the frontal convexities) and some neurodegenerative diseases (frontotemporal dementias). Frontal lobe tumors may also affect this area.

Orbitofrontal (Disinhibited/Pseudopsychopathy) Syndrome

Patients with lesions differentially affecting the orbitofrontal (the inferior or ventral surface of the frontal lobe) region will appear disorganized, behaviorally disinhibited, impulsive and overactive and often display emotional dysregulation. These individuals often demonstrate impulsivity and poor inhibition of behavior including inappropriate sexual behavior, inability to inhibit verbal outbursts and socially improper behavior. These individuals appear to have little empathy for others and will say and do things which may (at worst) appear purposeful efforts to hurt the feelings of others and (at best) appear uncaring. Patients may start new (or re-initiate) bad habits or addictive behaviors (e.g., start smoking, drinking, gambling) and may break social rules and norms to attain desired reinforcers. Work has found a functional distinction between the lateral and medial orbitofrontal areas. Lateral orbitofrontal regions are associated with the evaluation of punishment which leads to changes in behavior. Medial orbitofrontal regions are associated with the evaluation of reinforcers (primary or secondary) including the learning and memory for the reward value of reinforcers. In addition, a posterior and anterior distinction has also been made, such that the anterior orbitofrontal area is more involved in evaluation of more complex (secondary) reinforcers (such as money, social recognition, etc.) whereas the posterior orbitofrontal regions are more associated with primary reinforcers (e.g., gustatory).

Patients with orbitofrontal damage often exhibit poor judgment, and their behaviors are often governed by seeking reinforcers (often to extremes) and faulty reasoning initiated by environmental cues such as an object, person or situation. They may also be initiated by their own poorly inhibited thought processes. For example, patients may respond sexually to personnel who look at them and smile or respond angrily to personnel who make a request of them. Similarly, environmental cues such as the counter at a nursing station may solicit an orbitofrontal (disinhibited syndrome) patient to order auto parts because of its similarity to an auto parts store. These patients often demonstrate remorse for inappropriate behavior or verbal recognition of inappropriate behavior when their behavior is confronted. They tend to be hyperverbal and have difficulty with sustained attention. These individuals have little insight into how their behavior may affect others. These individuals may freely urinate in public or walk into other people's house to use the restroom, or to get desired things such as food, drugs, or money.

Memory functions are *not* consistently disrupted with lesions restricted to the orbitofrontal area. However, memory is often disrupted *if* damage includes the basal forebrain structures/septum, such that patients exhibit a classically described

dense amnesia (Irle et al. 1992; Zola-Morgan and Squire 1993). Thus, damage restricted to the orbitofrontal region does *not* produce traditional memory impairment, which occurs when basal forebrain/septal structures are involved (Irle et al. 1992; Zola-Morgan and Squire 1993). The amnesia occurring with septal damage includes both antegrade as well as a temporally graded retrograde amnesia (see Chap. 9). Encoding is reduced due to poor attention. Learning rate is often deficient with a flat learning curve. Episodic memory is generally impaired, but recall of various events may appear (incorrectly) to be quite vivid reflecting confabulation. This confabulation does not appear to be a purposeful attempt to deceive, and is outside the person's awareness. That is, the examiner should not believe he/she has been purposely deceived by a patient with basal forebrain/septal damage.

Patients with lesions restricted to the orbitofrontal regions may perform normally on most traditional neuropsychological tests. Frequently, behavioral observation and report from reliable informants can provide needed information. However, patients do exhibit more risk-taking behaviors, are less likely to adjust their behavior to feedback, and perform poorly on tasks of behavioral disinhibition/emotional regulation (e.g., Frontal Systems Behavior Scale; FrSBe, Grace and Malloy 2001) and modulation of reward-related behaviors (i.e., Iowa Gambling test; Bechara et al. 2000).

The orbitofrontal area (and superior temporal sulcus) is important when individuals make judgments about others' personality characteristics based on their physical characteristics (Winston et al. 2002). Patients with ventral medial (orbitofrontal) lesions (right more than left) have difficulty appreciating deception (Stuss et al. 2001; Rowe et al. 2001).

Emotionally, patients with orbitofrontal dysfunction may exhibit difficulty regulating emotional expression when emotionally aroused and appear to overreact emotionally at various times. Individuals may present with a "hollow" jocularity termed *Witzelsucht*. This presents as an inappropriate humor and/or laughing, often with the patient making inappropriate jokes about self or others. Another common feature of the orbitofrontal syndrome is *anosmia* (or, more correctly, lack of smell discrimination). Because patients often exhibit disinhibition, impulsivity, hyperactivity, lack of insight or empathy for others, emotional lability, and distractability, predominant damage to the orbitofrontal cortex has been called the disinhibited or pseudopsychopathic syndrome.

Anatomy: The orbitofrontal lobe (i.e., the entire ventral or inferior surface of the frontal lobes) incorporates Brodmann's areas 10, 11, 12, 13, and 14, and has been further subdivided into several specific functional regions termed the lateral and medial orbitofrontal areas (see Fig. 10.5). The orbitofrontal area is very complex, and has connections with areas throughout the brain including all sensory modalities as well as limbic structures. The main connections are from the temporal lobe (superior temporal cortex, inferior temporal cortex, and amygdala) as well as parietal lobe (somatosensory cortex), insula (gustatory cortex), and pyriform (olfactory) cortex. There are also connections to the medial temporal lobe structures, cingulate gyrus, thalamus (medial dorsal and intralaminar nuclei), and hypothala-

mus. Projections of the orbitofrontal area to the hypothalamus and amygdala allow this area to influence the autonomic nervous system. The orbitofrontal area is implicated in a vast array of cognitive, emotional, and somatosensory functions, such as behavioral inhibition, emotional regulation, social cognition, memory, and smell discrimination (Frith and Frith 2003; Lezak et al. 2004; Mesulam 2000; Siegal and Varley 2002). Social cognition is an important aspect of behavior, allowing one to interact in complex social networks. An important aspect to social cognition is the ability to appreciate or attribute the mental perspectives to other people, termed *Theory of Mind* (ToM) (Frith and Frith 2003; Siegal and Varley 2002; Stuss et al. 2001). Neuroanatomic organization for ToM has been purported to involve amygdala, temporo-parietal junction, orbitofrontal, and medial frontal regions. While theory of mind (appreciate mental perspectives of others) was thought to be particularly mediated by medial frontal function (e.g., Frith and Frith 2003; Siegal and Varley 2002), other data argue the medial frontal lobes are not involved (Bird et al. 2004). We include social cognition and appreciation of others' mental function here, rather than medial frontal lobe (below), because some recent data do not support the involvement of the medial frontal lobe.

Lesions to the orbitofrontal area is often *not* limited to one focal area, but rather the typical causes of damage to the orbitofrontal area tends to result in diffuse damage to the inferior and ventral frontal lobe areas. The subcortical basal forebrain as well as the medial frontal lobe areas can also be affected. Thus, patients may also present with features of the medial frontal (akinetic-apathetic) syndrome detailed below. Damage is often caused by acceleration-deceleration closed head traumatic brain injuries (which often also affect the anterior temporal lobes as well), neurodegenerative diseases (frontotemporal dementias), brain tumor, and/or hemorrhagic stroke of an aneurysm of the ACoA (anterior communicating artery) or an ACA (anterior cerebral artery).

Rule of thumb: Orbitofrontal (inferior/ventral) Frontal lobe damage = Disinhibited or pscheduopsychopathic syndrome

- Disinhibited
  - Hyperactive, intrusive, pressured behavior
- Poor impulse control
  - Loss of social insight, poor situational awareness
- Distractible
  - Focus on single thing and unable to selectively guide attention away from competing stimuli
- Emotional lability/emotional dysregulation
  - Septal/basal forebrain damage can result in amnesia with confabulation

Medial Frontal (Akinetic/Apathetic) Syndrome

Patient symptoms of medial frontal lobe damage often include akinesia, lethargy, not spontaneously initiating behavior, and can appear indifferent to painful stimuli. Memory can be severely disrupted, with a dense antegrade amnesia. Bilateral lesions can result in an akinetic and mute state. Unilateral lesions often result in an incomplete akinetic state, with the patient regaining some self-initiated behaviors. Left medial frontal lesions affecting the anterior cingulate can present with features of a transcortical motor aphasia. Patients with medial frontal lesions often lack insight or awareness and frequently are described as having decreased arousal in general. These patients will often not initiate behavior or speech themselves. In some cases, these patients will be observed to remain in postures likely to be extremely uncomfortable for prolonged periods without complaint or attempt to change positions. Urinary and bowel incontinence can be present, and patients exhibit little concern about the incontinence, making little effort to clean themselves unless given prompt(s).

Memory functions can be severely disrupted (e.g., Bird et al. 2004), reflecting damage to septal region/basal forebrain structures sometimes affected by more extensive orbitofrontal lesions. Memory impairment associated with medial frontal (akinetic-apathetic) syndrome is an amnesia with antegrade as well as a temporally graded retrograde amnesia (see Chap. 8). Encoding is poor and the patient often exhibits a flat learning curve. Episodic memory is generally impaired. Some semantic and nondeclarative memory may be intact. Confabulation is frequently present. Like confabulation associated with more extensive orbitofrontal damage, confabulation is not purposeful, and lacks intent to purposefully deceive the examiner.

Patients with mesial frontal/anterior cingulated damage often demonstrate restricted emotional responses and appear disengaged from their environment. Individuals may exhibit little (no) interest in family or friends, exhibiting indifference and apparent lack of concern. Generally, patients will appear dull and unmotivated, but may respond if requested to perform specific behaviors. Patients may exhibit lower extremity weakness contralateral to the side of the lesion (bilateral leg weakness if damage was bilateral). If the corpus callosum is damaged, the patient may also exhibit the so-called alien hand syndrome if the dominant hemisphere is affected. The left extremities may not be under the volitional control of the patient, and the left hand may reach for objects and/or explore the immediate environment outside the apparent control of the patient.

Anatomy: The medial frontal cortex includes the cortex between the two frontal hemispheres, anterior to the primary motor strip, which includes the anterior portion of the cingulate gyrus and includes Brodmann's areas 24, 25, and 32 (see Fig. 10.6). This area of the cortex has connections with the temporal cortices, particularly the amygdala (anterior cingulate) and hippocampus (more posterior cingulated) along with the hypothalamus. Reciprocal connections are with lateral prefrontal cortex (Brodmann's areas 8,9,10, and 46), orbitofrontal cortex (Brodmann's area 47), parahippocampal gyrus, amygdala, insula, and claustrum. Afferent projects are from entorhinal and perirhinal cortex and hippocampus,

thalamus, and tegmentum. Projections are also to substantia nigra pars compacta, subthalamic nucleus, hypothalamus, globus pallidus, and thalamus. This area of the brain is implicated in attention, behavioral inhibition, initiation and motivation, motor function (lower extremities), social cognition, including theory of mind, memory, mood, and autonomic (visceral) systems (e.g., Frith and Frith 2003; Lezak et al. 2004; Mesulam 2000: Siegal and Varley 2002; Stuss et al. 2001).

Rule of thumb: Medial frontal lobe damage = Akinetic/apathetic syndrome

- Akinetic and apathetic
- Little initiation of movement or speech
- Lack of interest and indifference
- Emotional blunting
- Memory can be impaired (amnesia with confabulation)
- Incontinence (bladder and sometimes bowel)
- Leg weakness

Prefrontal Syndromes: Two Syndromic Model

Below, we briefly review a two-syndromic model of prefrontal syndromes: (1) syndrome of frontal abulia and (2) syndrome of frontal disinhibition (e.g., Mesulam 2000).

(a) *Syndrome of Frontal Abulia*. This syndrome description is the same for that of the dorsolateral frontal lobe syndrome. Generally, patients exhibit poor problem solving, concrete reasoning, stimulus bound behaviors, lack of creativity, reduced (or no) initiative, apathy, and emotional blunting. Patients have difficulty planning and sequencing activities and exhibit deficits in strategic decision making in light of anticipated consequences for making various decisions.

(b) *Syndrome of Frontal Disinhibition*. This syndrome description reflects the fact that frontal lobe damage to the orbitofrontal area often also involves some aspects of the medial frontal lobe and basal forebrain, resulting in a general pattern of behavioral disinhibition, behavioral impulsivity, lack of judgment, reduced insight and foresight, and inability to delay gratification. Like orbitofrontal syndrome patients, the syndrome of frontal disinhibition may also include increased energy level and emotional reactivity. The individual's sleep–wake cycle can be disrupted, and they may not exhibit remorse for their behavior.

Bedside Assessment of Frontal Lobe Functions

General Assessment Issues

Frontal lobe damage can produce a range of deficits from subtle to grossly overt. The complexity of the frontal lobe involvement in many tasks both directly and indirectly means that their effect cannot only run the gamut from noticeable only to those who knew the patient well, to obvious to everyone in the environment but can also include functions which have an impact on other cognitive areas. For example, impairment in frontal lobes can produce simple, sustained and complex voluntary attention deficits which in turn have a discernable impact on seemingly unrelated tasks as object naming, copying a geometric figure or holding a conversation without becoming tangential or circumloquacious.

Assessment of frontal lobe functioning begins with good history taking and includes queries of both the patient and someone else who knows them well such as a parent, spouse, relative or friend. The content of this interview should include an assessment of changes in the cognitive, behavioral, and emotional functioning noted previously in this chapter. This discussion should inquire directly about change in these areas as there is considerable naturally occurring variability in behavior, cognition and emotional expressiveness across individuals. Table 10.2 outlines areas of inquiring for frontal lobe assessment. It is important to gain collateral information on these functions, as patient awareness of the existence or extent of the change in these areas of functioning is frequently diminished. If changes are noted, it is often helpful to try to establish an estimate of how much change has occurred and the frequency with which it occurs. It may be informative to ask both patients and collateral informants to give an estimate of current performance using 100% as a baseline and estimating the current level of functioning relative to that baseline. When using this table, we ask patients and a caregiver to rate how much change (percent) has occurred in the areas noted in Table 10.2, and this is written down in the column "Frequency/Duration/Severity." This may also highlight the discrepancy between the patient's perception of change and the perceptions of others who know them well.

In addition to a history of change, many of the functions of frontal lobe deficits can be assessed bedside or informally in the outpatient clinic. While these techniques can yield important information, these functions can also be more precisely measured through formal, standardized, psychometrically evaluated means. The informal assessment of frontal lobe functions can often lead to the decision for referral or further formal assessment of frontal lobe functions identified or suspected to be impaired in the brief examination. Table 10.3 gives some examples of areas of assessment and assessment items.

Motor and Sequencing Skills

Patients with frontal lobe damage often have difficulty with fine motor skills and sequencing motor skills. They may exhibit difficulty both with tasks requiring

Table 10.2 Interview assessment of Frontal Lobe changes

Area	Change	Frequency/duration/severity[a]
Activity/energy	Hypoactive	
	Hyperactive	
Initiation	Hypo initiative (Abulic)	
	Disinhibited	
Social function	Decreased social skill	
	Social imperturbability	
	Social avoidance	
	Social disinhibition	
Emotional responsiveness	Overreactive	
	Underreactive	
	Rapidly variable	
Attention	Poor sustained attention	
	Difficulty switching attention	
	Perseveration	
Language	Disorganized	
	Unresponsive to questions asked	
	Lapses from attentional difficulty	
Memory	Increased variability/inconsistency	
	Poor detail recall	
	Difficulty with temporal order in recall	
Reasoning/sequencing	Difficulty sequencing tasks such as cooking, repair, or other frequent activities	
	Concrete understanding, inability to see from others perspective	
	Knows conceptually, but cannot problem-solve the solution, step or steps to resolve novel problem	

[a]Column can be used as part of a working guide to note (write down) extent of change in domains listed. We have used percentile change, ranging from no change (around zero%) to total change (100%) change in that function/behavior. In some situations, it may be appropriate to not ask percent change, but rather a qualitative description, such as "no, small, medium, or large" change in function has occurred

sustained rapid motor responses as well as sequenced, novel motor skills. Patients may also have difficulty with simple sequencing tasks, exhibiting perseveration. Patients can be asked to rapidly tap their thumb and index finger. They should be instructed to perform this after being shown the examiner performing the task. Observations should be made regarding their ability to sustain their fine motor speed for 15 seconds to ensure that there is no gross slowing or increasing rigidity as indicated by progressively smaller taps. A second task is to ask them to perform a novel three-step sequenced task (Luria's manual sequencing task) after being shown through modeling. In this case the examiner places his hand on a table first making a fist, then with palm flat (slap), then on the side (i.e., Karate chop). Figure 10.7 illustrates these movements. The patient is asked to mimic this "fist–palm–side" sequenced movement. After success or failure with one hand, the other hand

should be tested. While it is not unusual for some patients (especially elderly) to require repeated modeling to acquire the appropriate sequence, if more than two trials are necessary, a sequencing deficit should be suspected. The patient should be able to complete three complete motor sequences without error.

Rapid motor sequencing can be assessed by asking patients to rapidly alternate hands from palm up to palm down. The examiner asks the patient to observe him (the examiner) doing the task and then requests that the patient mimic the task. The examiner places both hands down on a surface (typically a table or top of legs if seated) and then alternatingly lifts and rotates each hand to be palm up and then palm down. Each hand is rotated palm up and palm down and then the alternate hand is rotated. The alternate rotating is then increased in speed and should be sustained in rapid succession for 10–15 seconds. Patients should be expected to master the sequence of movements rapidly (one or two modeling trials) and be able to sustain the sequence for the duration of the task. If patients have difficulty, the examiner may try to teach the task by adding the verbal label, "all the way over, all the way back," to their demonstration to gauge if verbal cueing or prompting assists in acquiring or maintaining the task performance. If labeling the examiner's modeling of the behavior is unsuccessful, the examiner can take the patient's hands and rotate them and verbalize, "all the way over, all the way back," to see if kinetic cueing is effective in allowing them to master the task. In general, patients are expected to be able to rapidly master the task after being shown a demonstration. Failure to be able to perform rapid alternation or sustain rapid alternations over a 10- to 15-seconds timeframe should be considered an abnormal performance and reason for further psychometric investigation.

Rule of thumb: Bedside assessment

- Assess sustained attention with and without distraction
- Assess impulse control by conflicting verbal and behavioral gesture (i.e. say "Don't shake my hand" while simultaneously extending the hand)
- Assess perseveration by using repeating drawings such as loops or Ramparts
- Assess sequencing by asking patient to repeat three-step sequence or rapidly alternate hand movements

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Perseveration and deficits in set-shifting may also be identified in having patients complete an alternating sequence such as ramparts (see Fig. 10.7) running half-way across the page. Frontal lobe patients often fail to alternate between rectangle and triangle (often with the patient perseverating making linked triangles or linked rectangles only). Similarly, an alternating pattern of cursive "m"s and "n"s can be used to elicit perseveration in frontal lobe patients (see Fig. 10.7). The patient is asked to complete the pattern, beginning where the examiner stopped. The task is evaluated based on the patient's ability to appropriately alternate and not repeat "m" or "n". A final task is for the examiner to draw (outside of the examinee's vision) a series of large figures with 3 loops each. The patient is asked to
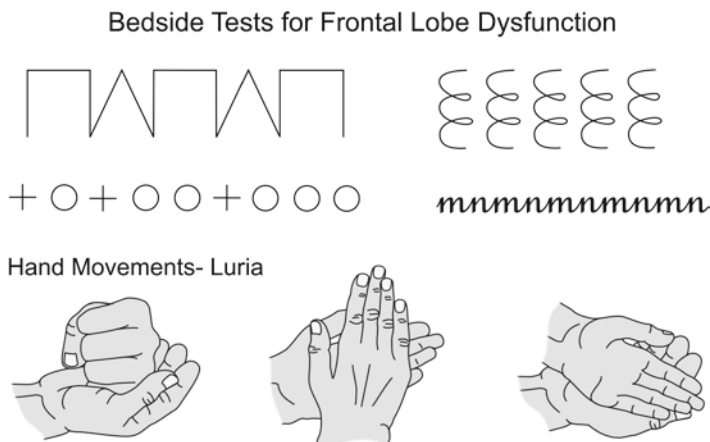
Bedside Tests for Frontal Lobe Dysfunction

Fig. 10.7 Luria's figures and sequencing tasks. Figures from left to right include ramparts, repeating loops, alternating +'s with increasing O's and alternating cursive M's and N's

complete making the looped figures until reaching the end of the page. Failure reflects having more or less than exactly three loops making up each of the figures. It is not unusual for patients with frontal lobe damage to make the figures with successively more loops (see Fig. 10.7).

Assessing Attention. Multiple aspects of attention can be impaired in frontal lobe patients (see also Chap. 6). They may have difficulty with simply attending to relevant stimuli in their environment without distraction, sustaining attention over time or in tasks which require them to switch voluntary attention rapidly. Simple attention can be evaluated by asking them to watch your finger as you move it slowly back and forth horizontally. They should be informed to keep their head still and track the examiner's finger with their eyes. This should be sustained for 15 seconds. The examiner can then add distraction to the task by prompting a discussion or purposefully diverting their gaze away from the patient. If the patient fails to maintain their voluntary gaze on the examiner's finger under either circumstance, the instructions can be repeated that the examiner wants them to maintain attention on the moving finger no matter what distractions are present. Patients with frontal lobe damage will often demonstrate difficulty with persistent voluntary attention and either lose attention to the task or have difficulty sustaining attention when confronted with verbal or visual competing stimuli in their environment.

Assessing Impulsivity/Disinhibition. The ability to initiate or inhibit a behavior is integrally linked to frontal lobe integrity. Patients with frontal lobe injuries often demonstrate changes in their ability to spontaneously initiate appropriate behavior or inhibit the enacting of overlearned or high frequency behavior. These deficits are most frequently observed early in the course of traumatic or acute injuries and progressively worsen in degenerative diseases involving the frontal lobes. These behaviors can be tested in examination of the patient in several ways. Perhaps the

easiest way is to use contradictory verbal commands and physical gesture. In such a circumstance, the examiner would tell the patient not to take an object or shake a hand that is offered. This is typically done by offering an object to the patient such as a pen, cup, or paper while simultaneously telling them, "Don't take this." A patient who spontaneously takes the object should be asked to repeat what the instructions were and then given a second trial. Failure on a second trial would indicate difficulty with inhibiting the behavior. A second task which emphasizes a more subtle disinhibition is referred to as the *go–no-go* task. In this task, the examiner first instructs the patient to hold up one finger when the examiner holds up one finger and hold up two fingers when the examiner holds up two fingers (checking for cooperation and sufficient motor/sensory function). Complete a minimum of two trials in random order of holding up one and then two fingers. Once this is mastered, the examiner then instructs patients to hold up two fingers when the examiner holds up one finger, and to display one finger when the examiner displays two fingers. The examiner displays alternating one and two fingers increasingly rapidly. The patient is evaluated on accuracy of their responses, the consistency of accuracy (i.e., can they maintain response set), how quickly they respond (should be decreasing delay as task is learned), and their spontaneous recognition and correction of errors. Once their responses have stabilized with several correct responses, the examiner randomly alternates holding up one or two fingers and assesses the patient's ability to respond correctly. This sequence of trials should include at least one series in which the same number of fingers is held up repeatedly to allow a habitual response to be established from the patient, at which point the number of fingers displayed by the examiner is switched and the patient's ability to suppress what had become an overlearned response can be gauged. To establish this overlearned response, four to five trials with display of the same number of fingers by the examiner are typically required. Patients are typically expected to make some errors early in learning this task, but quick mastery is expected. Rapid recognition and correction of errors is expected.

Abstract Reasoning. Both verbal and nonverbal abstract reasoning can be impaired by frontal lobe injury. These patients tend to have greatest difficulty with divergent abstract reasoning tasks compared to relatively intact convergent reasoning. The conceptual difference between the two being the increased demand in divergent reasoning tasks to escape a single, sometimes concrete (right/wrong) answer of convergent reasoning and attempt to enact creative, multi-solution divergent solutions to a stated problem. Verbally, patients can be asked to list the similarities of a set of things and then be asked to list their differences. Both the similarities and differences should demonstrate an understanding of multiple ways the two are similar and different. The examiner can prompt for the other ways the objects are similar or different but should not provide answers. Examples that can be used may include a dog and a wolf, a shark and a whale, a house of representatives and a senate, a house and a hotel (see Table 10.3). The patient should be able to give 2–3 ways each pair is similar and different and be evaluated based on the quantity of responses, organization of responses and the quality of the explanation they can make for their answers.

Again, frontal lobe patients would be expected to have difficulty with switching from similarities to differences and providing adequate number of responses and/or organization of responses. Patients can also be asked to state what they might do in a situation such as their refrigerator quits working or they notice that their pet's behavior changes. Responses should include recognition that these may have multiple causes and that they can be systematically ruled out but may ultimately require seeking additional help (see Table 10.3).

Rule of thumb: Disrupted functions associated with frontal lobe
- Divergent reasoning more impaired than convergent reasoning
  - For example, test is to "tell me as many uses of a soda pop bottle
- Poor inhibition
  - Impulsive. Unable to perform "go–no-go" task.
- Set-shifting and perseveration
  - Unable to sequence simple squares and triangles on a page or put three, and only three loops in a series of three looped "3"-shaped figures on a page

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Table 10.3 Verbal reasoning and cognitive flexability assessment

Object pairs	Similarities	Differences
Dog–wolf	Canine	Domesticated–wild
	Social	Geographically widespread–restricted
	Physical characteristics (legs, fur)	Size variable–typically large
	Carnivores	Seeks human contact–avoids human contact
	Mammals	
	Multiple pups	
Shark–whale	Live in water	Size
	Alive (breath oxygen)	Fish–mammal
	Swim	Bones–cartilage
	Widespread	Lungs–gills
	Similar physical characteristics (i.e., fins, skin, organs, teeth)	Horizontal swimming motion–vertical swimming motion
House of Representatives–Senate	Part of congress	Population proportioned–two per state
	Legislative branch	
	Elected	4-year term–6-year term
	Represent constituencies	Many more representatives–100 senators
	Work at capital	
	Pass bills	Rules and procedures differ

Table 10.3 (continued)

Cognitive Flexability Problems	Potential Solutions
Refrigerator repair	Check power supply
	Secure alternate food storage
	Call repairman
	Check breaker box/circuit breaker
	Inspect power cord, electrical outlet
	Check with neighbors about power supply
Pet behavior	Check food supply
	Check for injuries
	Check temperature
	Recall past routine (diet, interactions, elimination)
	Inspect physically
	Call vet
	Call knowledgeable friend
	Offer desired object (treat, toy)

Object	Alternative Uses
Coat hanger	Hang clothes
	Probe
	Fastener
	Toothpick
	Guide wire
	Skewer
	TV antenna
	Open car door lock
	Holder, extender
	Sculpting
	Form for object (i.e., lamp shade)
	Made into tongs or a rod for reaching/grabbing objects
	Scraping tool
Brick	Building material
	Water saver device for commode tank
	Message delivery system
	Door stop
	Paper weight
	Hammer
	Stepping stone
	Landscaping
	Art object
	Motor vehicle chock
	Speed bumps
	Self-defense

Visual divergent reasoning can be assessed either by asking the patient to creatively list as many uses for a common object such as a coat hanger, knife or brick or asked to draw as many shapes/designs using four straight lines of similar length that touch as possible. In the first task, the patient should be able to generate at least 3–4 nontraditional uses for each object. Responses are not judged for the quality of the object as a substitute for another purpose or object, but rather for the presence of divergent abstraction and creativity. For example, a brick might be used as a paper weight, a door stop, a water-saving (displacement device) device in a commode tank, or an exercise device, etc (see Table 10.3). In the latter task, patients could draw a sequence, two Xs, a series of crosses, or a series of triangles with intersecting lines. Again, the quality of the designs should not be judged, but rather the diversity and number. Patients should be able to spontaneously draw 4–6 designs in less than a minute. Failures can be inquired as to what they found difficult about the task. Possible solution sets for a bedside figure fluency task is presented in Fig. 10.8.
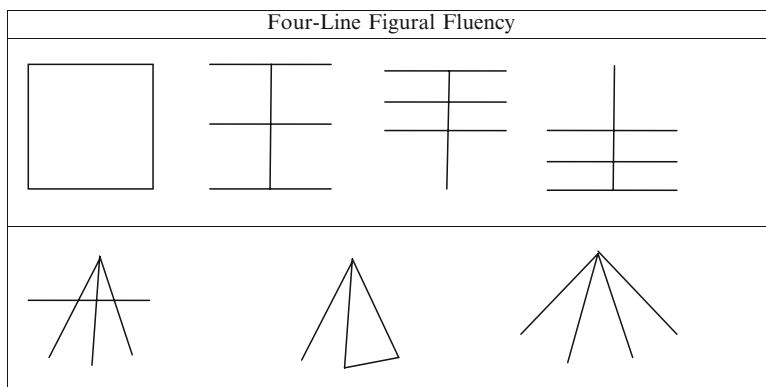

Fig. 10.8 Examples of solutions to a figural fluency task making at least six unique designs with four lines that touch

References and Suggested Further Reading

Bechara, A., Tranel, D., & Damasio, H. (2000). Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. *Brain, 123*, 2189–2202.

Bird, C. M., Castelli, F., Malik, O., Frith, U., & Husain, M. (2004). The impact of extensive medial frontal lobe damage on "theory of mind" and cognition. *Brain, 127*, 914–928.

Frith, U., & Frith, C. D. (2003). Development and neurophysiology of mentalizing. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences, 358*, 459–473.

Grace, J., & Malloy, P. F. (2001). *Frontal systems behavior scale (FrSBe): Professional manual*. Lutz: Psychological Assessment Resources.

Kolb, B., & Whishaw, I. (2009). *Fundamentals of human neuropsychology* (6th ed.). New York: W.H. Freeman.

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.

Mesulam, M. (2000). *Principals of behavioral and cognitive neurology* (2nd ed.). New York: Oxford University Press.

Rowe, A. D., Bullock, P. R., Polkey, C. E., & Morris, R. G. (2001). "Theory of mind" impairments and their relationship to executive functioning following frontal lobe excisions. *Brain, 124*, 600–616.

Salloway, S. P., Malloy, P. F., & Duffy, J. D. (2001). *The frontal lobes and neuropsychiatric illness*. Arlington: American Psychiatric Publishing, Inc.

Stuss, D. T., Gallup, G. G., Jr., & Alexander, M. P. (2001). The frontal lobes are necessary for "theory of mind". *Brain, 124*, 279–286.

Stuss, D. T., & Knight, R. (2002). *Principles of frontal lobe function*. New York: Oxford University Press.

Tulving, E. (2002). Episodic memory: From mind to brain. *Annual Review of Psychology, 53*, 1–25.

Tulving, E., Kapur, S., Craik, F. I., Moscovitch, M., & Houle, S. (1994). Hemispheric encoding/retrieval asymmetry in episodic memory: Positron emission tomography findings. *Proceedings of the National Academy of Sciences of the United States of America, 91*, 2016–2020.

Winston, J. S., Strange, B. A., O'Doherty, J., & Dolan, R. J. (2002). Automatic and intentional brain responses during evaluation of trustworthiness of faces. *Nature Neuroscience, 5*, 277–283.