Clinical Assessment of Dysexecutive Syndromes


Dysexecutive syndromes result from damage to the anterior regions of the brain and present as a combination of disinhibition, disorganization, or apathy.

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This activity offers CE credits for:
1. Physicians (CME)
2. Other

ACTIVITY GOAL
This article provides an overview of executive cognition and a review of the functional neuroanatomy of the frontal-subcortical system as well as approaches for evaluating executive cognition in the office.

LEARNING OBJECTIVES
At the end of this CE activity, participants should be able to:
1. Understand the various subcategories of dysexecutive syndrome.
2. Define the role that the frontal-subcortical system plays in dysexecutive syndrome.
3. Assess symptoms and differentiate between the 3 dysexecutive syndromes.

TARGET AUDIENCE
This continuing medical education activity is intended for psychiatrists, psychologists, primary care physicians, physician assistants, nurse practitioners, and other health care professionals who seek to improve their care for patients with mental health disorders.

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This article focuses on CNS dysfunction that produces dysexecutive syndromes. Dysexecutive syndromes result from damage to the anterior regions of the brain and present as a combination of disinhibition, disorganization, or apathy. We provide an overview of executive cognition, a central function of the anterior regions; review the functional neuroanatomy of the frontal-subcortical system, the neural circuitry that supports executive cognition; and offer ways to evaluate executive cognition in the office.

**Overview of executive cognition**

Executive cognition is an elusive metacognitive function that serves many purposes. As modern humans evolved over millions of years, we acquired a selective advantage in hostile environments—we became socialized and thereby benefited from the cooperative efforts of the groups we formed. Over time our brain increased in size, favoring expansion of the frontal system, which allowed humans to form progressively more sophisticated social networks. What began as small bands of hunter-gatherers eventually grew into early agrarian communities; city-states; and modern-day societies, clubs, and organizations.

We developed the ability to learn from experience and to communicate verbally and nonverbally using facial expressions, gestures, and tone of voice. Our social environment increased in complexity: cultures developed and introduced rules, limitations, and consequences for our behavioral choices. Throughout our evolution, our expanding neurology for executive cognition provided flexibility to manage our responses to the social environment. We have thus acquired the ability to exert influence over events taking place around us and over our own actions within the social context.

Executive cognition can be broken down into smaller cognitive elements. These include the ability to monitor the environment; identify relevant stimuli; devise plans of action to engage the environment; anticipate the outcomes of each option on the basis of previous experience, select an option, implement it, monitor it on the basis of anticipated result, and modify it as needed to achieve the intended outcome; and remember the experience. This system permits us to align our behaviors with the social context and includes motivation, organization, and self-regulation. This can involve simple acts (e.g., when to eat) or more complicated challenges (e.g., interviewing for a job). The ability to maintain sufficient motivation, organization, and social comportment serves as the foundation for achieving social success.

The ability to succeed socially depends on our capacity to conceptualize the self (self-awareness) and to mentalize (i.e., to have a theory of mind). Self-awareness is necessary to evaluate our behaviors in comparison with social norms; patients with frontal lobe lesions have impaired introspection and self-reflection.

Theory of mind refers to the ability to conceptualize the mental and emotional states of others as analogous to but distinct from our own. This allows us to predict and understand the actions of others, and it enables us to empathize.

According to Heatherton, numerous positron emission tomography and functional magnetic resonance imaging (fMRI) studies indicate that these functions involve the medial prefrontal cortex. It may be that self-conceptualization engages posterior cingulate cortical circuits along with some contribution from the precuneus. The temporoparietal junction and temporal lobes are essential cortical regions contributing to mentalization.

On a neuroanatomical level, our ability to conceive of a theory of mind may be dependent on our capacity to conceptualize the self. Work done by Heatherton and Mitchell and colleagues indicates that the ventral regions of the medial prefrontal cortex are involved in conceptualizing the self. Conceptualizing others is attributed to neuronal activation in the dorsal regions of the medial prefrontal cortex. Findings from fMRI studies suggest that subjects who are asked to anticipate the emotional state of individuals perceived as having shared values, interests, and motivations show activation in the ventral prefrontal regions.

Working memory is an important and well-established executive function. This type of memory enables us to analyze information. Desimone identified an area in the dorsolateral convexity of rhesus monkeys that appears to function as a mental scratch pad that may provide the ability to mentally hold and thus manipulate information. Prabhakaran and colleagues have confirmed the existence of similar working memory functions in the lateral prefrontal cortex of humans. These functions free us from the consequences of acting primarily on impulse and expand our ability to
navigate successfully in more complex environments.
In an effort to build upon the working memory model, Fuster proposed that a primary function of the executive cognitive system is for “the integration of sensory information and motor acts into novel, complex, and purposive behavioral sequences.” Functions such as these permit us to efficiently and effectively deal with social situations. This is likely accomplished through at least 4 fundamental cognitive operations:

• Control of attentional resources (selective attention)
• Use of a template for provisional short-term (working) memory
• Development of response strategies (preparatory set)
• Feedback control of the selected response (monitoring)

The prefrontal cortex is also the repository of “executive memory” and harbors representations of plans and schemas of behavior and language. Mesulam expands on the model of executive cognition by emphasizing the ability of the prefrontal cortex, through its cortico-cortical and cortico-subcortical connections, to engage other neural systems in the brain in the service of adaptive behavior. Consequently, we are able to efficiently utilize emotions, sensations, and memories to deal with our environment. Human behavior is likely to be adaptive when we are able to suppress our “default modes” of behavior. He identifies our default modes as innate tendencies to be stimulus-driven, impulsive, perseverative, and emotionally shallow.

Lesions located anywhere within the extended networks of the frontal-subcortical system can produce similar deficits. In other words, when the physical integrity of a circuit is critically disrupted anywhere, by any cause, the entire circuit will fail and the functional contribution of that circuit to behavior will be diminished or lost entirely. Some combination of the default modes of behavior will be released and expressed. The patient becomes less autonomous, and his or her behavior is driven by impulsive and ineffective responses to social experiences.

**Functional neuroanatomy of executive cognition**

The underlying neurology of executive cognition is represented by one of the principal organizing neural networks of the human brain, the frontal-subcortical system. Alexander and Crutcher identified a series of neural loops involving frontal cortex anterior to the central sulcus of Rolando (prefrontal) and subcortical gray structures including the caudate nucleus, the globus pallidus, and the dorsomedial nucleus of the thalamus. Cortical and subcortical regions were linked by long white matter tracts in the anterior region of the brain. These loops, with their gray matter nodes, are capable of efficiently processing enormous amounts of information. A total of 5 loop circuits were delineated, 3 of which support executive cognition: the orbitofrontal, the mesial frontal, and the dorsolateral convexity. Information from broad regions of the cortex is quickly funneled to much smaller subcortical areas that reconnect with the larger cortical regions. There is limited “cross-talk” between these circuits, and the loops appear to remain segregated.

The prefrontal, or frontal-subcortical, system has a unique place in the CNS in that it receives highly processed information from the other heteromodal regions of the brain, including the parietal and temporal areas. These are cortical regions where various sensory modalities (eg, visual, auditory) are built up into more complete inner templates of our environment. Parietal information is carried along the superior longitudinal fasciculus. The parietal regions can be conceived of as our cognitive tool kit; they include language functions, overlearned motor acts (praxis), mathematical abilities, constructional abilities, and perception of 3-dimensional space.

Emotional responses are processed by the frontal system through input from the limbic system. The limbic system provides rich connections to the prefrontal system via the uncinate bundle, especially to the posterior orbitofrontal region. Afferent information from the limbic system includes emotional and motivational cues that can bias our selection of options and, ultimately, our behavior. Auditory information from the rostral region of the superior temporal gyrus is carried anteriorly to the orbitofrontal cortex, which appears to mediate behavioral responses to emotionally relevant auditory stimuli. Similarly, object-related visual information is carried through the uncinate bundle. This likely permits executive processing of visually triggered emotions. Emotions that are specifically social in nature (eg, love, embarrassment, pride, admiration) appear to be processed in the anterior cingulate cortex and the medial prefrontal cortex.

The modulatory centers for the attentional matrix located in the brain stem communicate with the frontal system through the reticular activating system. This system, which originates in the reticular core and connects to the reticular nucleus of the thalamus, utilizes acetylcholine to modulate cortical arousal (ie, level of consciousness). Cholinergic neurons located in the nucleus basalis respond selectively to novel and emotionally relevant stimuli and project diffusely throughout the cortex. This
system can serve as a means to facilitate storage of new information for future reference. Cortical tone can be further modulated by noradrenergic projections from the locus coeruleus and activated by motivational cues. Serotonergic neurons within the dorsal raphe nuclei appear to enhance our ability to respond to the behavioral relevance of environmental stimuli. Dopaminergic input to the cortex originates in the ventral tegmental area and nucleus accumbens. This mesocortical system is sensitive to the relationship between expectation and reward and is capable of reinforcing behaviors associated with reward.

Schmahmann and Sherman identified participation of the cerebellum in executive cognition. Substantial connections link both the limbic system and the prefrontal cortex with cerebellar structures. This cerebrocerebellar network facilitates smooth and efficient executive cognition, perhaps in an analogous manner to the motor functions of the cerebellum.

**Dysexecutive syndromes**

This system of distributed neural networks involving prefrontal cortical and subcortical gray matter linked by white matter tracts is vulnerable to a broad range of pathology. The cortical component can be adversely affected by degenerative conditions such as β-amyloid (neuritic plaque) or α-synuclein (Lewy body) accumulation, formation of neurofibrillary tangles or Pick bodies (tauopathy), spongiform changes, stroke, or trauma.

The subcortical gray structures can be damaged by genetic trinucleotide repeats (Huntington disease), dopamine depletion (Parkinson disease), copper accumulation (Wilson disease), neurofibrillary tangle formation (progressive supranuclear palsy), or lacunar strokes, in addition to other, less common conditions. White matter can be damaged by, among other things, demyelinating conditions such as multiple sclerosis, infarcts, or diffuse axonal injury.

These various forms of neural damage can produce specific prefrontal syndromes. Disorders that affect the physical or neurochemical balance within these networks will alter the otherwise stable expression of personality and/or cognition. Thus, the first sign of the development of prefrontal systems disease is usually a personality change or a cognitive change.

The term “frontal lobe syndrome” has become synonymous with acquired changes in personality, and there is no doubt that focal lesions involving the prefrontal cortical mantle produce predictable changes in behavior and executive cognition. However, sufficient clinicopathological evidence exists that lesions distant from the cortex, those that involve the cortico-striato-pallido-thalamo-cortical circuit, will produce similar clinical findings. Hence the term “dysexecutive syndrome” is a more appropriate designation for these patients.

In clinical practice, lesions are rarely confined to one functional division within the prefrontal system. Therefore, patients will usually present with features of more than one symptom cluster. With this in mind, 3 distinct dysexecutive syndromes have been identified, each associated with the discrete cortical region that participates in the neural network.

**Orbitofrontal disinhibition syndrome.** This behavioral syndrome that results from damage to the orbitofrontal cortex or circuit often caused by a coup-contrecoup brain injury is characterized by the onset of inappropriate sexual, aggressive, or vulgar behavior. The posterior orbitofrontal region has rich connections with the paralimbic cortex and the limbic system. Thus, this executive network is heavily involved with the elaboration and integration of mood and limbic drives into successful social behavior.

Hunger, joy, sexual arousal, fear, and anger all serve to bias our attention and behavior toward gratifying the particular subjective sensation. However, the manner in which one goes about seeking and achieving gratification can mean the difference between effectively settling a dispute and committing murder. The ability to anticipate the consequences of various behavioral options and to hold our impulses in check until we choose a particular course of action is a highly adaptive skill. Damage to this system results in a loss of good social judgment. The patient becomes impulsive, aggressive, tactless, shallow, and remorseless. In the primary care physician’s office, a patient with orbitofrontal disinhibition syndrome may appear irritated by the physician’s questions and would refuse to cooperate with much of the physical examination despite having no sound reason for doing so.

Alternatively, the patient may express inappropriate familiarity or friendliness, asking inappropriate questions or even making a sexual proposition. He may demonstrate inappropriate silliness known as witzelsucht. The key clinical observation with the dysexecutive syndromes is the acquired nature of these behaviors. They often represent a distinct departure from the patient’s premorbid interactive style.

The best illustration of the orbitofrontal disinhibition syndrome is provided by Harlow in his classic description of Phineas Gage in 1868. Gage is perhaps the most famous neuropsychiatric patient of all
Patients with orbitofrontal disinhibition syndrome are often considered to be antisocial or manic. Typically, however, there is no premorbid history of sociopathic behaviors or additional symptoms of mania, such as diminished need for sleep, pressured speech, or grandiose delusions. **Dorsal convexity dysexecutive syndrome.** This cognitive syndrome is characterized by the onset of disorganization in the setting of frontal systems damage and usually renders a person unable to live independently because he is unable to adequately perform activities of daily living, such as housework, meal preparation, taking medications, managing money, grocery shopping, and telephone or technology use. Otherwise, these patients appear to be perfectly intelligent and usually retain excellent verbal skills.

The high-level executive cognition mediated by the dorsal convexity circuit, as described by Milner and Petrides,\(^{12}\) involves cognitive flexibility, temporal ordering of events, planning, and learning from experience. In a sense, this system operates our cognitive tool kit, located in the posterior parietal lobes. The product of this system is an organized approach to our daily responsibilities, such as managing finances, maintaining nutrition, budgeting time, communicating, and applying our intellect adaptively and successfully to our constantly changing environment. Damage to this system results in an overall loss of the ability to independently manage functions of daily living. Patients can no longer balance their checking accounts or maintain good hygiene or nutrition. They fall prey to telephone marketers or mail lottery schemes. Typically, a family member takes over their checking account and financial management. They usually have a limited variety of food items in their pantries. A “tea and toast diet” is often a reflection of diminished executive management of the many steps involved in maintaining proper nutrition. Family members worry and check in on these patients frequently, despite their seemingly good physical health.

In the office, they provide simple and concrete explanations for their progressively poorer decisions, such as “I don’t trust repairmen” or “I’ll get around to doing that.” They cannot adequately answer open-ended questions and often respond by rephrasing the physician’s question as an answer. Their responses may also be rather long-winded and convey little content, indicating an inability to think succinctly. Perseveration, tangentiality, and circumstantial thinking are common, making it difficult for the examiner to efficiently collect information from the patient. These persons can be either easily distractible or quite perseverative with an inability to “switch set.” Their family members occasionally find them to be exasperating, because they appear to be more capable of independent functioning than they actually are.

Patients with dysexecutive syndrome may be able to perform within normal limits on tests designed to quantify learned knowledge or sensorimotor skills, such as the Mini-Mental Status Examination or the Wechsler intelligence and memory scales. They do, however, demonstrate impairment on tests that emphasize problem-solving skills, such as the Wisconsin Card Sorting Test or the Tower of London.\(^{13,14}\)

**Apathy syndrome.** This behavioral syndrome is characterized by a loss of spontaneity and activity. The mesial frontal region is histologically complex and thus least amenable to designation as a distinct functional entity. Cytoarchitectonic studies of the region demonstrate the anterior and medial extensions of cingulate, orbitofrontal, and dorsomedial cortical regions. Thus, it is likely that there is no predominant executive function subserved solely by this area. However, published reviews of cases of anterior cerebral artery territory infarction find a high prevalence of post-stroke apathy or hypobulia.\(^{15,16}\)

Kang and Kim\(^{16}\) found that strokes located in the frontal pole, cingulate gyrus, and superior medial frontal gyrus were associated with apathy. Thus, this region may represent to some degree a functional bridge between thought and action. The behavioral product of this loosely defined system is typically termed “motivation.”

These patients often appear to have depression. Their lack of spontaneity and limited interest in hygiene are noteworthy. However, they deny subjective sadness or hopelessness and have no insomnia or loss of appetite. Family members become progressively more frustrated by their inertia and occasionally accuse them of being lazy or uncaring. In the office, the patients will typically be dressed plainly. They sit quietly and tend to speak only when asked direct questions. They are...
capable of answering questions accurately but use few words. They have no explanation for their progressive loss of motivation. They lack animation but have no motor signs of parkinsonism beyond their blandness.

Because there is no specific formal test to examine motivation, the clinical impression is of utmost importance with this syndrome. This impression should be based on observation of the patient in the office, the clinical interview, and conversation with collateral informants. The patient will often struggle to answer questions about what provokes anger or other strong emotions that they now lack.

**Office evaluation**

The most efficient way to assess executive cognition is an office visit. This is a very simplified approach but one that will help the busy clinician screen for dysexecutive syndromes. The clinician may, if time permits, offer the patient the comprehensive Executive Interview (EXIT25). The Frontal Assessment Battery can also be offered.

If dysexecutive syndrome is suspected, the patient can be referred to a neuropsychologist for further testing. In this instance, the clinician should specifically ask the neuropsychologist to test the patient’s executive cognition. Otherwise the patient may receive a cognitive battery that does not fully examine executive cognition, and the diagnosis may be missed or the severity may be underestimated.

The initial inquiry should be conducted with the patient and, whenever possible, with collateral informants—spouses and adult children are ideal. The inquiry consists of 3 questions:
1. Has the patient become more disorganized of late (eg, managing a checking account, keeping up with laundry and dishwashing, maintaining adequate food supplies)?
2. Has the patient developed a personality change and become more vulgar or silly?
3. Has the patient become more apathetic or neglectful of late?

The patient is then asked to perform 5 executive cognitive tasks:
1. The Ozeretski Alternating Motor Sequence: The patient is asked to place one hand palm down on a table with the other hand making a fist and simultaneously alternate the open and closed position of the hands in a smooth and steady manner; repeat several times.

Patients with frontal systems dysfunction cannot perform the sequence accurately. They may open and close both hands together, at the same time, or do one at a time without being able to do them simultaneously. They may also begin to add movements such as flapping their hands or flexing and extending their elbows. (The commonly used 3-step sequence of fist-palm-side is too complicated and is likely to produce false-positive findings.)

2. The Go/No Go Task: The clinician faces the patient and asks him to place both hands in his lap; the clinician says, “When I touch my nose, then you raise your finger. If I raise my finger, then you touch your nose.” This task is done in a series of rounds with the examiner and patient replacing their hands on their lap after each round.

Patients with frontal systems dysfunction cannot perform this task accurately. It requires an intact ability to suppress the high probability or default response of mimicry. Initially, they often have difficulty in learning the simple rules. Subsequently, they will demonstrate a partial or complete tendency to copy the examiner’s movement despite knowing the rules.

3. Letter Fluency Test: Patients are asked to produce as many words as possible in 1 minute without using proper nouns and beginning with the letter “A.” Many patients do not remember what makes a noun “proper,” so explain to them: “No names of people, like Adam, or places like Alabama, just regular words beginning with A.” Patients should produce approximately 15 words in 1 minute.

Patients with frontal systems dysfunction have problems with this task. It requires the executive cognitive ability to retrieve stored information within the limits of a “rule.” They may become confused and start saying “apple, ball, chair . . .,” or they may simply struggle to name more than 2 or 3 words beginning with “A.” They may also perseverate on 1 or 2 words. Disinhibited patients may say “ass” and smirk at their cheekiness.

4. Draw a circle 6 inches in diameter on a blank (not lined) piece of paper, and ask the patient to turn it into a clock by drawing in the numbers. If he completes this satisfactorily, then ask him to draw in the hands so that the clock says “10 past 11.” This test requires the executive skills of planning to properly space the numbers around the circle, monitoring performance for errors, resisting distraction, and set shifting for the symbolic meaning of minutes as opposed to hours on a clock face.

Patients with frontal systems dysfunction may have difficulty with “shifting set” from drawing numbers to using hands and numbers to tell time. They may be drawn to the stimulus of the number “10” and place a hand on it, instead of the number “2” (10 minutes). Some patients will simply write
“10 past 11” on their otherwise normal clock. Alternatively, more progressive dysexecutive problems will render patients unable to plan the clock properly and space the numbers evenly. They may place the numbers backward or write numbers beyond “12.” When patients are followed up annually, the clinician will see progressive deterioration in the quality of their ability to draw a clock.

5. Draw a “Ramparts Design” (Figure) and ask the patient to copy it. Have the patient continue his copy all the way across the page. Many times patients can successfully copy the design while drawing directly underneath it but will make mistakes when they no longer have the visual cue readily available.

Patients with frontal systems dysfunction will be unable to accurately shift set between the square and triangle and will tend to perseverate on one or the other. Those with more severe dysexecutive syndrome will simply draw directly on the examiner’s drawing.

Since executive cognition is so complex, no single office test will suffice as an acceptable screen. Most patients are capable, at least early on in their course, to perform some of the battery well. Often it is the impression and frustrations of collateral informants that are the most sensitive, especially early in the course of illness. Although the series of tests presented here has not been statistically validated, it is sensitive enough to detect early signs of frontal systems dysfunction and can lead to further workup.

Patients in whom dysexecutive syndromes are suspected can be evaluated with a more focused history, a thorough neurological examination, and judicious use of neuroimaging, leading to a diagnosis. In this way, clinicians may make more timely diagnoses of these typically vexing syndromes and prevent an escalating cycle of patient decline and caregiver stress.

Additional tests that are often a valuable part of a neuropsychological evaluation that the clinician should be aware of include the following:

• The Wisconsin Card Sort Test: This test is designed to study a patient’s ability to think abstractly and switch set. The patient must sort cards and deduce the rules by which they should be sorted (eg, color, symbol, number) from the examiner’s feedback. The rules change periodically throughout the test. The test can be scored in a number that quantifies the ease with which the examinee adapts to the new rules. Patients with frontal lobe injury make more perseverative errors and have trouble shifting to changing rules or “switching set.”

• The Stroop Test: This test provides information about a patient’s ability to maintain attention and inhibit certain responses. It takes advantage of the fact that it takes people longer to name a color than to read a word. And it proves especially difficult to read a name of a color when it is written in ink of a mismatched color.

The test also shows a slowing effect as patients’ reading times slow the longer they are involved in the task, and this phenomenon lends to the test’s sensitivity. It appears to have a high sensitivity in patients with closed head injury, who will continue to do poorly despite good recovery in many areas. One caveat is that patients with pronounced attention difficulty may find the test arduous and unpleasant.

• Trail Making Test: This test was developed by US Army psychologists as part of the Army Individual Test Battery. It incorporates visual, spatial, and motor skills as well as attention, set shifting ability, and complex conceptual thinking. In part A, the patient must connect consecutively numbered circles. In part B, the circles have letters and numbers and the patient must alternate between letter and numerical sequences. The patient is asked to do the task quickly. This test is quite sensitive to impairments in executive cognition. Patients with emotional disturbances can perform slower on this test because of avolition and/or apathy.

• Tower of London: This test evaluates the ability to plan, which involves organization, and the ability to make choices, to look ahead, and to conceptualize change. Memory is also necessary. Beads are placed on a series of pegs, and the participant must move them from one position to another in the fewest possible moves. Multiple versions are possible with different degrees of difficulty and number of moves required.

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References:


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