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RELATIONSHIP DISCLOSURE:
Dr Tang-Wai has provided expert legal testimony for the Canadian Medical Protection Association on a case determining if a patient had cognitive impairment. Dr Freedman serves as a trustee for the World Federation of Neurology and on the editorial boards of *Brain and Cognition* and *Journal of Parapsychology*; has received support from and served on an advisory board for Eli Lilly and Company Canada and receives publishing royalties from Oxford University Press; receives research/grant support from the Alzheimer Society of Canada, Brain Canada Foundation, Centre for Aging and Brain Health Innovation, Canadian Institutes of Health Research, and Westin Brain Institute; receives support from the Behavioural Neurology Physician Recognition Covenant Fund at Baycrest, the Morris Kerzner Memorial Fund, and the Saul A. Silverman Foundation as part of the Canada International Scientific Exchange Program project; and holds stock in companies producing or planning to produce medical marijuana and is listed on a provisional patent related to methods and kits for differential diagnosis of Alzheimer disease.

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Bedside Approach to the Mental Status Assessment

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ABSTRACT

PURPOSE OF REVIEW: This article presents a clinically useful approach to obtaining the history and performing the mental status examination of patients with cognitive, language, or behavioral problems.

RECENT FINDINGS: Laboratory and imaging biomarkers are being developed for accurate diagnosis of neurobehavioral disorders, yet few are currently available for clinical use. Moreover, not all centers have access to these potential tools. Practicing clinicians are therefore left primarily with their skills of history taking and examination. Although geared for research, diagnostic criteria have been refined over the past several years and can nevertheless aid the clinician with the diagnosis of disorders such as mild cognitive impairment, Alzheimer disease, frontotemporal dementia, dementia with Lewy bodies, the primary progressive aphasias, corticobasal syndrome, vascular cognitive impairment, and posterior cortical atrophy. Regularly revised criteria reflect ongoing knowledge gained from in-depth studies of these disorders.

SUMMARY: The focused history and mental status examination remain essential tools for the evaluation and diagnosis of neurologic disorders affecting cognition, language, and behavior.

INTRODUCTION

A thorough history and mental status examination are necessary requirements for the evaluation of the patient with cognitive impairment. Both revolve around the cognitive domains of executive function, attention, memory, visuospatial function, language, and behavior. While the history inquires about the patient's abilities related to these domains as they pertain to his or her life, the mental status examination tests the integrity of these domains in a clinical setting and is, therefore, a formal part of the neurologic examination. The overall goal is to determine if performance on each domain is normal or impaired. Longitudinal assessments provide additional information to determine if a change has occurred.

Memory tests assess the ability to learn new information or recall previously acquired information. The language evaluation involves an assessment of speech output, comprehension, ability to name objects, and ability to repeat words and sentences. It is also important to determine whether the patient has a loss of

grammar, such as the inability to use articles, pronouns, prepositions, or conjunctions. The visuospatial assessment evaluates spatial awareness, visuospatial perception, and a patient's ability to find things in front of him or her. Assessment of executive function evaluates complex processes needed for goal-directed activity that involve task setting and monitoring.[1] Deficits in these processes can lead to problems with performance on tasks that require sequencing, abstraction, and planning.[1] For more information on executive function, refer to the article "Clinical Assessment of Prefrontal Lobe Functions" by Alexandre Henri-Bhargava, MDCM, MScCH, FRCPC; Donald T. Stuss, OC, O Ont, PhD, FRSC, FCAHS, CPsych, ABPP-CN; and Morris Freedman, MD, FRCPC, FAAN,[2] in this issue of *Continuum*. In the appropriate clinical context, other cognitive domains could be evaluated, such as apraxia (defined as the impaired ability to carry out purposeful motor activities despite intact motor function and sensory feedback) or agnosia (defined as the failure to recognize or identify objects despite intact sensory function). For more information on apraxia and agnosia, refer to the article "Apraxia, Neglect, and Agnosia" by H. Branch Coslett, MD, FAAN,[3] in this issue of *Continuum*.

Over the years, mental status tests have evolved from simple screening measures to more in-depth evaluations that are reflective of the increased understanding of the clinical and cognitive features of the various types of dementias (**SDC 1-1**[4-25]; *links.lww.com/CONT/A251*). Initial tests, such as the Blessed Information-Memory-Concentration test (BIMC)[4] or Delayed Word Recall (DWR),[13] were sufficient to detect "dementia." Each successive cognitive assessment has added more cognitive domains, such as in the Mini-Mental State Examination (MMSE)[6] or Behavioural Neurology Assessment (BNA)[20]; become increasingly sensitive to detect milder changes in cognition, such as the Montreal Cognitive Assessment (MoCA)[21] or Toronto Cognitive Assessment (TorCA)[25]; or added assessments of behavior, such as in the Addenbrooke's Cognitive Examination (ACE)/Addenbrooke's Cognitive Examination Revised (ACE-R).[17,18,22] Most of the recent cognitive assessments evaluate memory, orientation, language, visuospatial function, and executive function. In addition, these assessments can objectively monitor the change in a patient's cognition over time.

Although dysfunction in a cognitive domain reflects disruption of neural networks or connectomes,[26] it is clinically useful to localize each cognitive domain/function to a specific area of the brain. Moreover, additional clinical utility exists in determining a patient's relative strengths and weaknesses across the cognitive domains tested, representing the patient's cognitive profile, in addition to determining the overall score. It is the cognitive profile—knowing the most impaired cognitive domain and its localization within the brain—that will help the clinician (in conjunction with the history, neurologic examination, and neuroimaging) determine the underlying cause of cognitive impairment.

This article focuses on the important aspects of the history, identification of the cognitive profile on mental status examination, and localization of the brain lesion based on the results of the cognitive assessment.

OBTAINING A HISTORY

A new specific skill set is not required when obtaining the history of a patient with cognitive or behavioral symptoms but rather a conceptual approach for eliciting specific information about the patient's cognition, behavior, and daily function. This involves obtaining the history from a collateral informant in

KEY POINTS

- The mental status examination tests the integrity of cognitive domains (executive function, attention, memory, visuospatial function, language) in a clinical setting and is, therefore, a formal part of the neurologic examination.
- Longitudinal assessments, combined with the mental status examination, provide additional information to determine if a change has occurred.
- It is clinically useful to determine a patient's cognitive profile (a patient's relative strengths and weaknesses across the cognitive domains tested) as it will help the clinician, in conjunction with the history, elemental neurologic examination, and neuroimaging, to determine the underlying cause of cognitive impairment.
- It is important to obtain the history from a collateral informant in addition to the patient to clarify the presenting symptom, to determine the chronologic progression of the signs and symptoms, and to determine the course of the progression (gradual, fluctuating, or stepwise).
- The collateral historian need not always be a family member but can be anyone who has observed cognitive difficulties, can comment on them by providing specific examples, and can report whether the observed cognitive difficulties have caused any impairment with the patient's usual ability to perform instrumental activities of daily living.

addition to the patient, clarifying the presenting symptom, and determining the chronologic progression of the signs and symptoms. It is also important to determine whether the onset was gradual, acute, or subacute and whether any decline has been gradual, fluctuating, or stepwise.

Importance of a Collateral History

When obtaining the history, it is imperative to try to obtain a collateral history from a person who knows the patient well, since patients with cognitive impairment (eg, due to probable Alzheimer disease [AD]) often are unaware of their own cognitive and functional deficits. The collateral historian need not always be a family member but can be anyone who has observed cognitive difficulties, can provide specific examples, and can report whether the observed cognitive difficulties have caused any impairment with the patient's usual ability to perform instrumental activities of daily living (ADLs). This approach provides the clinician the ability to determine the affected cognitive domains and whether the patient meets the criteria for mild cognitive impairment, in which instrumental ADLs are spared, or dementia, in which these activities are impaired. TABLE 1-1 provides some examples of difficulties that might be

TABLE 1-1 **Sample Comments From Collateral Historians and Disorders to Consider by Presenting Symptom**

First Symptom Noticed	Affected Cognitive Domain	Disorders to Consider
Repeats him/herself; rapidly forgets conversations	Anterograde memory loss	Alzheimer disease
Cannot recall people he/she sees on the street; does not recognize familiar people at a party; cannot recognize his/her own house	Prosopagnosia	Semantic dementia variant of frontotemporal dementia
Cannot align things; has problems seeing, reading; blurry vision; cannot fill out a form; cannot find things in the refrigerator; cannot read a map; misplaces items; gets lost/geographic disorientation	Visuospatial dysfunction	Alzheimer disease (posterior cortical atrophy variant), dementia with Lewy bodies
Inability to fix things	Apraxia, executive dysfunction, visuospatial dysfunction, attentional dysfunction	Corticobasal syndrome, Alzheimer disease, dementia with Lewy bodies, vascular cognitive impairment
Forgets words; describes words, talks around them; mixes up words, mispronounces words; forgets what a word means	Language (anomia)	Primary progressive aphasia (nonfluent, logopenic, or semantic variants)
Sometimes able to do things and sometimes appears more confused and cannot do things	Attention (fluctuations)	Dementia with Lewy bodies
Cannot plan, multitask, or stay on task; must do everything in single steps, cannot combine tasks	Executive dysfunction	Alzheimer disease, vascular cognitive impairment, behavioral variant frontotemporal dementia (behavioral abnormalities must also be present for this diagnosis), dementia with Lewy bodies

reported by the collateral historian, the cognitive domains affected, and some of the disorders to consider based on these symptoms.

Identifying Patient Features

The age when the first symptoms developed determines if the process is either early onset (defined as before age 65) or late onset (defined as age 65 or older). Although not exclusionary, older age of onset likely predicts that a neurodegenerative process is the etiology, while genetic, vascular, infectious, or metabolic causes are more often found in younger patients. However, it is important to note that ischemic cerebrovascular disease is commonly associated with neurodegenerative disorders, such as AD.

Handedness can provide information regarding the lateralization of cognitive functions in the brain. In 95% of right-handed patients, language function is lateralized to the left hemisphere. In contrast, 22% of left-handed patients have an atypical language lateralization within either the right hemisphere or both hemispheres.[27] In addition, it is useful to note a change in use of preferred hand (ie, development of a preference for use of the nondominant hand) as this can imply either weakness or apraxia on the dominant side. Furthermore, slowing of rapid alternating movements of the patient's dominant hand on the neurologic examination should alert the clinician to consider either weakness or extrapyramidal causes.

Education and occupational history provide information about the patient's premorbid level of intelligence and function as well as information that is helpful for interpretation of the cognitive test results. For example, consider a trial lawyer, who ought to perform well on language tests, generating 11 words on letter fluency (producing as many words as possible beginning with a given letter, such as *F*, in 1 minute). Although this may be considered "normal" on the MoCA, it should be considered as possibly impaired as the lawyer might be expected to produce more than 11 words. Depending on the patient's occupation, the clinician may need to alter the line of questioning to determine changes or impairment in instrumental ADLs.

Clarifying the Presenting Symptom and Course of the Illness

At the beginning of the interview, we recommend that the following information be obtained: (1) onset of the condition (insidious or acute), (2) presenting symptom, (3) course of the condition (gradually progressive, stepwise, fluctuating, or improving), and (4) duration. These pieces of information can often identify the underlying dementia syndrome and determine a rational approach to diagnostic investigations (**TABLE 1-2**).

Both the onset of the condition and its duration can be described as acute (minutes to days), subacute (weeks to months), or chronic/insidious (years). When either an acute onset or rapidly progressive duration is reported, the clinician should verify the accuracy of the information as informants can underestimate the duration of illness because of a tendency to link symptom onset to a major life event or illness.[28] It is best to determine a timeline of changes in cognition or behavior so as not to overlook changes that have been incorrectly attributed to normal aging. Posing the simple question "When was the last time the person was able to independently…?" can be useful in this situation.

When asked for the presenting symptoms, the informant often will describe any cognitive deficit as a memory deficit. Therefore, it is important not to interpret each "memory" complaint as an impairment in anterograde (short-term)

KEY POINTS

- The ideal documentation should include the onset of the condition (insidious or acute), presenting symptom, course of the condition (gradually progressive, stepwise, fluctuating, or improving), and duration.
- It is important not to interpret each "memory" complaint as an impairment in anterograde (short-term) memory as it is common for an informant to describe any cognitive deficit as a memory deficit. Instead, ask for examples of the presenting symptom to determine accurately what the informant means by "memory deficits."
- The history must include a description of the patient's cognitive, behavioral, physical, and functional decline.
- Changes in behavior can accompany any patient with cognitive impairment and can occur before the onset of cognitive symptoms, progress with the cognitive symptoms as a major feature of the dementia, or be part of a recognized symptom complex, such as in limbic encephalitis.
- It is best to obtain the chronologic sequence of the changes in a patient's cognitive, behavioral, physical, and functional decline. Although many neurodegenerative dementias have overlapping impairments, the chronologic sequence of events may help determine the type of dementia.

memory; instead, the clinician should ask for examples of the presenting symptom to determine accurately what the informant means by "memory deficits." For example, an informant may report memory deficits, but the examples provided may be indicative of word-finding difficulties and thus indicate an underlying anomia or language impairment as the presenting sign. Otherwise, the rest of the history will overemphasize an amnestic problem that would be suggestive of AD. The course of the condition can be described as progressive, static, fluctuating, or improving. Depending on the course of the disorder, it can further indicate an etiology (TABLE 1-2).

TABLE 1-2

Onset of the Presenting Symptom and Course and Duration of Illness That Will Help Determine Etiology of the Cognitive Change

Onset

- **Acute (seconds to days)**
  - Stroke
  - Infection (viral, bacterial)
  - Metabolic
- **Subacute (weeks to months)**
  - Metabolic
  - Infection (Creutzfeldt-Jakob disease, fungal, spirochete)
  - Endocrine
  - Paraneoplastic
- **Chronic (years)**
  - Neurodegeneration
  - Chronic cerebrovascular disease

Progression

- **Improving**
  - Stroke
  - Infection (viral, bacterial)
  - Metabolic
  - Delirium
- **Static**
  - Stroke (fixed deficit)
- **Fluctuating**
  - Epilepsy
  - Paraneoplastic

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One example of a clarified chief complaint and course is "the *insidious onset* (onset) of *progressive* (course) *anterograde memory decline* (presenting symptom) that has worsened over *2 years* (duration)." This example should indicate to the clinician that the patient most likely has AD, although this must be supported and verified by obtaining the rest of the history. Another example of a chief complaint would be an "*acute onset* (onset) of a *fixed* (progression) *anterograde memory loss associated with prosopagnosia and visual hallucinations* (presenting symptoms) of *1-day duration* (duration)," which would be suggestive of a right posterior cerebral artery infarction.

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- ◇ Metabolic
- ◇ Dementia with Lewy bodies

◆ **Progressive**

- ◇ Neurodegeneration
- ◇ Chronic cerebrovascular disease
- ◇ Infection (Creutzfeldt-Jakob disease)

Duration

◆ **Acute (seconds to days)**

- ◇ Stroke
- ◇ Infection (viral, bacterial)
- ◇ Metabolic

◆ **Subacute (weeks to months)**

- ◇ Metabolic
- ◇ Infection (Creutzfeldt-Jakob disease, fungal, spirochete)
- ◇ Endocrine
- ◇ Paraneoplastic

◆ **Chronic (years)**

- ◇ Neurodegeneration
- ◇ Chronic cerebrovascular disease

First Symptom Noticed

◆ **The presenting symptom often determines the type of dementia (TABLE 1-1)**

◆ **Not all causes of dementia present with true anterograde/short-term memory loss**

What to Obtain in the History

The clinician should obtain a description of the patient's cognitive, behavioral, physical, and functional decline from the informant. The areas of cognitive changes include memory, language, praxis, visuospatial function, attention, and executive function. The first column in TABLE 1-1 lists descriptions from informants (not an exhaustive list) describing the affected cognitive domain. If a cognitive domain has not been addressed spontaneously, then the clinician should inquire about it to determine if a change has

TABLE 1-3 Sample Questions to Probe Each Cognitive Domain

Cognitive Domain	Probes/Sample Questions
Executive function	Does the patient:
	Have difficulty planning/organizing (eg, vacations)?
	Have difficulty multitasking (eg, planning or cooking a meal)?
	Show poor judgment/bad decisions?
	Have difficulty with problem solving?
	Show mental rigidity or inflexibility?
Attention and concentration	Does the patient:
	Lose track of thoughts?
	Have difficulty following TV program or movie?
	Get easily distracted?
Memory	Does the patient:
	Forget names?
	Misplace objects?
	Repeat questions and/or conversations?
	Rapidly forget what is told to him/her?
	Use compensatory strategies (eg, makes notes as reminders)?
	Mix up dates?
	Forget appointments?
	Leave stove on/faucet running?

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occurred that the informant had neither appreciated nor brought up (TABLE 1-3).

Both types of ADLs, instrumental and basic, should be asked about. With impairment in instrumental ADLs, complex skills required to live independently are lost, such as managing finances, driving, preparing meals, using devices, and shopping. Impaired instrumental ADLs are required to satisfy the criteria for dementia. Since every patient is unique and their instrumental ADLs vary, questions should be derived from tasks in their occupation and their usual

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Cognitive Domain	Probes/Sample Questions
Language	Does the patient:
	Have word-finding difficulty (eg, peoples' names, common words/objects)?
	Make speech sound/word errors (phonemic and/or semantic paraphasias)?
	Show comprehension deficits (eg, with complex oral instructions, simple oral instructions, written text)?
	Have pronunciation/articulation deficits?
	Show loss of word meaning?
	Have slurred speech?
	Not always make sense when speaking?
	Have reduced speech?
Visuospatial/geographic orientation	Does the patient:
	Have trouble navigating (eg, gets lost in familiar places, in unfamiliar territory, own household)?
	Have prosopagnosia (ie, trouble recognizing familiar faces, objects/buildings)?
	Have trouble finding objects in a refrigerator or drawer or directly in front of them?
	Have trouble parking the car, resulting in new dents, or drive too close to others?
Personality/behavior	Does the patient:
	Show disinhibition (eg, socially inappropriate behavior; loss of manners or decorum; impulsive, rash, or careless actions)?
	Show changes in social interpersonal conduct/loss of social graces?
	Show apathy or inertia (eg, loss of interest, drive, and motivation; decreased initiation of behavior)?
	Show changes in emotional expression/reactivity/empathy/sympathy (eg, diminished response to other people's needs or feelings; diminished social interest, interrelatedness, or personal warmth)?
	Show changes in eating habits (eg, altered food preferences, binge eating, increased consumption of alcohol or cigarettes, oral exploration or consumption of inedible objects)?
	Show perseverative, stereotyped, compulsive/ritualistic behavior (eg, simple repetitive movements or complex compulsive or ritualistic behaviors)?

function at home. The ability for patients to perform their basic ADLs (eg, grooming, managing hygiene, bathing, eating, dressing) should be determined. Changes in basic ADLs usually occur in the later stages of dementia, and the descriptions range from fully independent to needing reminders, requiring some help, and fully dependent.

Changes in behavior can accompany any patient with cognitive impairment and can occur before the onset of cognitive symptoms,[29] progress with the cognitive symptoms as a major feature of the dementia,[30] or be part of a recognized symptom complex, such as limbic encephalitis.[31] *Mild behavioral impairment* is a recently described condition in which patients who are cognitively normal develop neuropsychiatric symptoms consisting of decreased motivation, affective dysregulation, impulse dyscontrol, social inappropriateness, or abnormal perception or thought.[29] Patients with mild behavioral impairment are at greater risk to develop cognitive decline when compared to patients without neuropsychiatric symptoms. Many patients with dementia will develop changes in their behavior as the dementia progresses. For example, patients with AD can develop apathy or short-temperedness during their illness. Patients with behavioral variant frontotemporal dementia (bvFTD) have early and prominent changes in behavior (apathy, disinhibition, social inappropriateness, indifference) as a hallmark of this disorder.[30] In limbic encephalitis, changes in behavior, such as irritability, depression, or psychotic symptoms, occur characteristically with other symptoms of memory loss, seizures, and sleep changes. This recognizable pattern should alert the clinician to the possibility of a disorder affecting the limbic system, such as a paraneoplastic syndrome.[32]

Chronologic Approach to the History

It is best to obtain the chronologic sequence of the changes in a patient's cognitive, behavioral, physical, and functional decline. Although many neurodegenerative dementias have overlapping impairments, the chronologic sequence of events may help determine the type of dementia. For example, in the amnestic presentation of AD, the sequence of impairment in the cognitive domains would be an initial deficit in memory, then executive function, followed by language, visuospatial function, and, finally, behavior. In contrast, a patient with bvFTD would classically present with impairment in behavior, then executive function, followed by language and then memory. Although the same cognitive domains are affected, it is the sequence of impairment that can help determine the cause. In addition, it has become increasingly clear that certain neurodegenerative syndromes may evolve to incorporate other distinct clinical syndromes, and this is being reflected in revised diagnostic criteria.[33,34] For example, patients presenting with a nonfluent progressive aphasia can progress over time to develop corticobasal syndrome, progressive supranuclear palsy, or bvFTD.[33]

The chronologic approach can also determine if differing pathologies are developing in the same person. Cases have been reported in which patients developed the core features of dementia with Lewy bodies after developing typical AD many years earlier.[35,36] This has implications for management, such as recognition of the relatively high-risk neuroleptic sensitivity in dementia with Lewy bodies.

Ascertainment of the True Cause of the Symptom or Functional Decline

As with the nonspecific presenting symptom of "memory loss," the clinician should elicit additional details when the patient either has difficulties with or is

incapable of performing a specific function. For example, the patient who is no longer able to use a remote control could be demonstrating difficulty with executive function affecting the task setting and monitoring that are required to plan and keep track of the appropriate steps in using the device. Alternatively, visuospatial deficits may be affecting the ability to recognize the remote control itself or its parts, language impairment may be affecting interpretation of what the buttons mean, physical weakness may be limiting the ability to press the buttons, or apraxia may be affecting higher motor control, such as organization or sequencing of movements. A combination of these factors may also exist. The informant will only report that the patient has an inability to perform a task, and it is up to the clinician to determine the reason based on the history and examination.

Other Points to Emphasize in the History

When obtaining a medication history, a list of prescription and over-the-counter medications the patient is taking should be obtained. Many over-the-counter medications, especially those that advertise promotion of sleep, are anticholinergic or antihistaminergic, and both can affect cognition. In addition, the clinician should be mindful of multiple medications used to treat a condition (eg, hypertension); although it may be a medically resistant condition, it could also indicate noncompliance because of forgetfulness that the prescribing physician is unaware of.

Attention should be drawn to medical and neurologic disorders in the past medical history that could affect cognition, such as endocrinopathies, chronic organ failure, and chronic neurologic disorders such as Parkinson disease, multiple sclerosis, and epilepsy. The presence and control of cerebrovascular risk factors should be noted, such as coronary artery disease, hypertension, hypercholesterolemia, transient ischemic attack, and stroke. If the patient has a history of stroke, it is important to determine symptoms to localize the area of the stroke and determine if a direct temporal relationship to cognitive decline is present. A history of concussion or traumatic brain injury must also be obtained. Confusion following recent surgeries may suggest an underlying neurologic disorder affecting cognition combined with limited cognitive reserve. Moreover, caregivers may date the onset of a dementia to a surgical procedure/anesthesia when, likely, the dementing symptoms predate the surgery but were either unnoticed or dismissed. Inquiry regarding the patient's habits should include alcohol consumption, smoking, and illicit drug use.

MENTAL STATUS EXAMINATION

The cognitive domains assessed in many cognitive assessments are memory, orientation, language, visuospatial function, and executive function (**SDC 1-1**; *links.lww.com/CONT/A251*), with each of those functions classically localized to specific areas of the brain. The cognitive assessment should determine not only the domains that are impaired but also the domains on which the patient may perform normally. Although the various tests appear to examine all of these cognitive domains, it is the degree of in-depth evaluation of each domain that differentiates each test, leading to the possibility of either underestimating the impairment or not assessing the involved symptom.[37] For example, the MMSE is an easy and quick-to-administer test but has limitations for detecting mild cognitive impairment and early dementia and for identifying the various types of dementias.[38,39] In addition, some bedside assessments do not examine all domains,

KEY POINTS

- Additional details must be elicited to determine the true cause of the disability when the patient either has difficulties with or is incapable of performing a specific function.
- Be mindful of multiple medications used to treat a condition (eg, hypertension); although it may be a medically resistant condition, it could also indicate noncompliance because of forgetfulness that the prescribing physician is unaware of.
- The cognitive assessment should not only determine the domains that are impaired but also determine the domains on which the patient may perform normally.
- A profile of impaired memory with preservation of function in other domains, together with intact activities of daily living, suggests amnestic mild cognitive impairment.
- Clinicians should use the information obtained in the history to guide them in selecting and interpreting a cognitive assessment as no single cognitive test can accurately diagnose all conditions; each test has some limitations in its sensitivity to detect abnormal function as well as limitations in specificity.

TABLE 1-4 **Localization of Cognitive Domains and Tests to Explore Each Domain**[a]

Cognitive Domain and Localization	General Administration	Additional Office Tests to Consider
Executive function Lateral prefrontal Left frontal: Task setting Right frontal: Monitoring	Executive function can be assessed by examining component processes of tasks that involve task setting and monitoring; for example, on clock drawing, planning the contour size and shape, number placement, and time setting involve task setting, and avoiding duplication of numbers involves monitoring[1]	Trail Making Test[1] Phonemic (letter) word list generation or fluency[1] Luria hand sequences[1] Go/no-go task[1] Similarities[1] Clock drawing[1] Alternating patterns[1]
Attention Frontal	Tests vigilance by asking the patient to perform a single sustained task (eg, serial subtractions, digit span) or when given distractors (eg, letter cancellation)	Serial subtractions[1] Digit span (forward and reverse) [1] Letter cancellation tests[1]
Language	All components of language should be tested individually in patients who present with a primary language disorder; short screening bedside tests may not be sufficient to characterize the disorder, and additional tests are required	
Sentence repetition Left perisylvian area	Ask the patient to repeat words and phrases	Sentence repetition test[40] Montreal Cognitive Assessment sentence repetition[21]; the only caveat, if patients have impaired working memory, they will perform poorly on this test as the sentence has too many words for them to learn and repeat Repetition items of the Toronto Cognitive Assessment (TorCA)[25]
Naming Left temporal lobe for isolated naming deficits	Ask the patient to name various objects with increasing difficulty, for example, naming a *watch*, then *face* (of the watch) or *crystal* (of the watch), then *stem* (of the watch); another example is to ask the patient to name a *shoe*, followed by the *sole* (of the shoe), then *eyelet* or *laces* (of the shoe)	Multilingual Naming Test[41] Boston Naming Test[42]

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Cognitive Domain and Localization	General Administration	Additional Office Tests to Consider
Reading, writing Left parietal/inferior parietal lobule for isolated reading and writing deficits (alexia with agraphia)	Ask patient to write a sentence and read a passage of text, for example from a magazine	Written and oral description of the cookie theft picture
Comprehension[b] Left temporal-parietal	Ask the patient to perform tasks in the office using simple instructions (eg, point to the floor) that increase in complexity (eg, point to the surface that you walk on; point to the floor after pointing to the ceiling)	Sentence comprehension tasks[43] that assesses grammar comprehension
Semantic knowledge Left temporal lobe	Ask the patient to define objects (living and inanimate)	Semantic knowledge test[25,43]
Visuospatial		
Parietal/occipital/temporal	In general, this is assessed by asking patients to copy a figure; can also assess for additional visuospatial functioning such as simultanagnosia and optic ataxia, elements of Balint syndrome; need to ensure that no primary ocular problem is present, such as macular degeneration, that would confound the interpretation	Benson complex figure copy[32] Rey-Osterrieth Complex Figure Test[44] Necker cube copy[11,21] Judgment of Line Orientation test[45] Identifying overlapping figures (Montreal Cognitive Assessment Basic)[46]; assesses simultanagnosia Identifying numbers on the Ishihara plates[47]; assesses simultanagnosia Describing a complex picture, such as the Cookie Theft Picture; assesses simultanagnosia if the patient cannot describe the whole picture but only parts of it Asking the patient to reach out to an object; assesses optic ataxia (in the absence of cerebellar findings) if the patient misses the object[48]
Frontal visuospatial	This is assessed by asking the patient to draw to command (which will be impaired) but patient is able to copy the figure	None

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Cognitive Domain and Localization	General Administration	Additional Office Tests to Consider
Memory Temporal lobe/ hippocampus/medial temporal Dorsomedial thalamus[49] (in cases of stroke)	Verbal or visual memory tests always have two parts: an initial learning task and delayed recall task The learning task employs at least one learning trial and has multiple elements to learn (eg, 3 to 15 words; >10 elements on paragraph/story learning; >10 visual elements) The delayed recall task usually occurs after several minutes and after several distractors are introduced from the learning task so as not to have the patient rehearse the words in between The recall task always involves a free recall without cueing; some tests then allow a cued recall, such as with a category cue Recognition should also be assessed by providing patients with the correct target stimuli and incorrect items and asking them to choose the correct items Impaired delayed recall with impaired recognition suggests a true memory problem with loss of information, whereas impaired delayed recall with good delayed recognition suggests a retrieval problem due to frontal system damage	Bedside assessments generally evaluate verbal memory with three to five words; if additional measures are required to examine further, consider a robust verbal list learning with delayed recall that has 10 to 15 elements, such as the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) Verbal List[14] or Free-Cued Test[50] Testing orientation is also considered as a memory test[25] Visual memory can be assessed by copying a complex figure (see Visuospatial above) followed by recall after a 5- to 10-minute delay; some bedside tests have incorporated this, such as the TorCA
Calculations Left parietal/inferior parietal lobule	This is assessed by asking the patient to perform simple arithmetic (subtraction, multiplication, division or addition), but not using 1, 2, 5, or 10 as this is too easy, or asking from the multiplication tables as this may test memory rather than calculations	Example from the Short Test of Mental Status[11]: 5 × 13 = 65 – 7 = 58 ÷ 2 = 29 + 11 =

[a] Note: Localizations listed in this table represent classic clinical-anatomic relationships; however, brain lesions in other regions may produce similar deficits. For example, although frontal lesions are classically associated with attentional deficits, impaired attention can also be due to right parietal lesions. In addition, although isolated naming deficits can occur following left temporal lesions, impaired naming occurs as part of aphasic disorders due to lesions in the left frontal, temporal, or parietal lobes.

[b] Patients may fail comprehension tests if they have deficits in semantic knowledge. However, patients with semantic knowledge deficits often perform better with phrases than single words because they can benefit from the context, whereas patients with comprehension deficits tend to have more trouble as the length of what they are asked to comprehend increases.

CASE 1-1

A 78-year-old right-handed man with 14 years of education was brought into the clinic urgently for assessment of the acute onset of "confusion." He had been evaluated 4 days previously to assess his 2-year history of the insidious onset of progressive anterograde memory loss that affected his usual instrumental activities of daily living. Neuroimaging at that initial visit revealed bilateral hippocampal and parietal atrophy. At the initial visit, he was diagnosed with Alzheimer disease. He had been well over the intervening 4 days but in the morning, his family noted that he appeared confused and requested an urgent reassessment. His medical history was remarkable for hypertension, type 2 diabetes mellitus, and hypercholesterolemia.

On cognitive testing, he scored 17/38 on the Short Test of Mental Status[11,12]; he had scored 25/38 at the initial evaluation 4 days earlier. The elements of each test and his scores for each area are listed below. His elemental neurologic examination was normal on both visits.

Given the acute clinical change and the results of the clinical assessment at the second visit, he was clinically diagnosed with an acute left parietal stroke and sent to the emergency department for urgent neuroimaging. Brain CT revealed an acute left parietal intracerebral hemorrhage.

Short Test of Mental Status Domain	Score at Initial Visit	Score at Follow-up Visit
Total score	25/38	17/38
Orientation	6/8	5/8
Attention	6/7	4/7
Learning (number of trials)	4/4 (1 trial)	4/4 (2 trials)
Calculations	4/4	0/4
Similarities	2/3	2/3
Construction/drawing	3/4	0/4
Information	4/4	3/4
Delayed recall	0/4	0/4

COMMENT

This case illustrates that cognitive testing can aid with the localization of brain lesions. When comparing the two test results, a marked and focal difference is seen in this patient's ability to perform calculations and draw items, and both functions localize to the left parietal lobe. Given the acute change reported, the clinical conclusion was an acute stroke affecting the left parietal lobe.

and additional tests should be considered. Once the assessment has been completed, the pattern of the patient's cognitive difficulties relative to their overall performance (ie, their cognitive profile) can be used to help determine the etiology of the impairment. For example, a profile of impaired memory with preservation of function in other domains, together with intact ADLs, suggests amnestic mild cognitive impairment.

Classic Localization of Cognitive Domains

Although cognitive function is mediated by networks that span different brain regions, an association exists between each cognitive domain and specific lobes within the brain (TABLE 1-4[40–50]). For example, anterograde memory function is localized to the temporal lobe, specifically the hippocampus or medial temporal lobe. However, it should be noted that lesions in the dorsomedial nucleus of the thalamus can also impair anterograde memory function.[49] Nevertheless, the mental status examination can aid the clinician with lesion localization (CASE 1-1). In addition, some classic syndromes, each with a specific localization, consist of a typical constellation of cognitive symptoms and signs (eg, Gerstmann syndrome and Balint syndrome [TABLE 1-5]) that are found in some neurodegenerative dementias and after focal lesions. By clinically determining the possible damaged areas of brain that may account for the cognitive deficits, the clinician can focus

TABLE 1-5 Classic Eponymous Cortical Cognitive Syndromes

Syndrome	Clinical Components	Office Tests to Consider	Localization	Disorders to Consider
Balint syndrome	Simultanagnosia (the inability to see objects simultaneously)	Ishihara plates[47]; overlapping figures[46]	Bilateral occipitoparietal	Posterior cortical atrophy; watershed infarcts
	Optic ataxia (the inability to reach a target under visual guidance)	Impaired finger-nose (misreaching) in absence of other cerebellar findings[38]		
	Oculomotor apraxia (the inability to purposefully move the eyes to a target)	Test saccades and observe for failure to initiate the movement		
Gerstmann syndrome	Acalculia (the inability to perform arithmetic)	See calculations in TABLE 1-4	Left inferior parietal lobule	Posterior cortical atrophy; left middle cerebral artery infarct
	Right-left difficulties (the inability to recognize the left from right sides)	Ask the patient to demonstrate the right and left sides of parts of their body		
	Agraphia (the inability to write)	See writing in TABLE 1-4		
	Finger agnosia (the inability to discriminate and therefore name the individual fingers of the hand)	Ask the patient to identify fingers		

CASE 1-2

A 55-year-old right-handed man with 17 years of formal education presented with the insidious onset of progressive word-finding difficulties of 3 years in duration. In conversations, he knew that he should know some words, but he had forgotten what they meant. Although he could recognize colleagues, he did not recall their names. He had no change in personality or behavior.

On cognitive testing, he scored 30/38 on the Short Test of Mental Status,[11,12] with the following breakdown of the subitems: orientation 8/8, attention 5/7, learning 4/4 words but in three trials, calculation 4/4, construction/drawing 4/4, information 3/4, and delayed recall 3/4. His elemental neurologic examination was normal.

Additional cognitive testing revealed normal performance on the Rey-Osterrieth Complex Figure Test[44] (a test that involves copy and recall of a complex figure and measures several functions, including visuospatial ability, planning, and visual memory), Logical Memory subtest of the Wechsler Memory Scale[52] (a test of memory in which the patient learns and recalls elements of a story), and Rey Auditory Verbal Learning Test[53] (a test of memory in which the patient learns and recalls a list of unrelated words). However, he obtained 5/30 on the Boston Naming Test, generated seven animals for semantic fluency, and generated 25 words on verbal letter fluency (C, F, L).

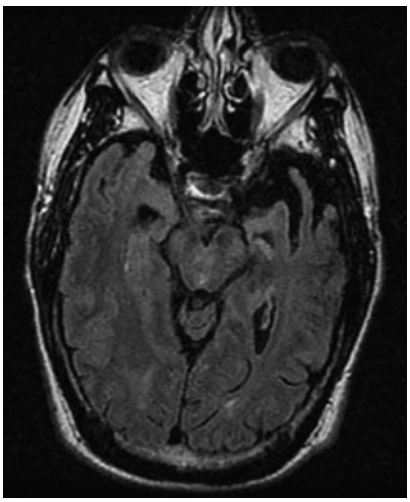

FIGURE 1-1
Axial fluid-attenuated inversion recovery (FLAIR) MRI of the patient in CASE 1-2 showing focal left anterior temporal lobe atrophy.

Brain MRI revealed focal left anterior temporal lobe atrophy (FIGURE 1-1). Brain single-photon emission computed tomography (SPECT) revealed left more than right anterior bitemporal hypoperfusion. A diagnosis of semantic variant primary progressive aphasia was made.

COMMENT

This case illustrates the limitation of certain bedside screening tests and the need to add additional tests to adequately evaluate a patient to reach a diagnosis. The patient had scored reasonably well on the Short Test of Mental Status, losing points for difficulties in learning words as additional points are deducted for learning after one trial. This test also does not adequately evaluate language; therefore, additional language tests were administered as language deficits were the patient's presenting symptom. Furthermore, his deficits were clinically localized to the left temporal lobe, which was confirmed on MRI.

on these areas when reviewing CT or MRI scans of the brain for potential lesions, such as stroke, mass lesion, or focal areas of cortical atrophy.

Selection of an Appropriate Cognitive Test and Knowing Its Limitations

Current bedside cognitive tests provide a structured and easily administered approach for the evaluation of patients with either dementia or mild cognitive impairment[38,51] as well as patients with cognitive complaints who perform normally on testing. In the absence of the clinical history, no single cognitive test can accurately diagnose all conditions, as each test has some limitations in

CASE 1-3

An 80-year-old right-handed man presented with the insidious onset of progressive short-term memory of 2 years in duration. Initially, his family observed that he repeated the same questions and parts of conversations that they had. In addition, he misplaced items around the home.

About 1 year earlier, he could only complete one task at a time and could not multitask. A family member took over the family business, as the patient forgot details of business contracts. His emails got shorter, with shorter sentences, and had spelling and grammatical errors. For example, he wrote "I were doing something." He was still able to drive and cook. He was independent with his basic activities of daily living.

His neurologic examination was normal. Results of his cognitive testing with the Toronto Cognitive Assessment (TorCA)[25] are shown. Brain MRI revealed bilateral hippocampal and mild biparietal atrophy. His presentation was consistent with the diagnosis of Alzheimer disease (AD).

COMMENT

This case illustrates several points. The nature of the presenting symptom suggested a neurodegenerative disorder, likely AD, given the insidious onset, anterograde memory impairment presentation, and progression over years. The history was consistent with a dementia, as it revealed impairments in multiple cognitive domains, including memory (rapid forgetting and repeating the same questions), language (short sentences, agrammatism), and executive function (inability to multitask) associated with a decline in his instrumental activities of daily living (inability to work). Interpretation of cognitive testing revealed impairments in memory (impaired delayed verbal more than visual recall as well as impaired delayed recognition), language (mild impairment in naming), and executive function/attention/working memory (reverse digit span). Memory was most affected compared to the other affected domains. His cognitive profile was predominantly amnestic, with medial temporal lobe localization (impaired delayed recall and impaired delayed recognition). In addition, a discrepancy was seen between letter *F* fluency and semantic animal fluency, with only the latter being impaired. This is consistent with temporal lobe dysfunction. Therefore, multiple pieces of clinical evidence indicated medial temporal lobe pathology. This was supported by the brain MRI findings. Given all the information, the patient's presentation is consistent with AD.

its sensitivity to detect abnormal function and limitations in specificity. The history obtained should guide the clinician on which cognitive assessment to use.

A common approach is to choose a standard cognitive test that is routinely administered as a cognitive screen, such as the MoCA. However, when administering such a cognitive test, it is useful to know the limits of the test. When the test is insufficient because either the patient's symptoms are too mild to be detected by the test or the patient's symptoms are not well assessed by the test (eg, the MMSE is relatively insensitive to deficits in executive function), a

Domain	Test Segment	Results
Memory	Orientation	Normal
	Verbal memory	
	Learning (10 words in three trials)	1, 5, and 7 words on trials 1, 2, and 3, respectively
	Delayed free recall	0/10; impaired
	Delayed recognition recall	17/20; impaired
	Visual memory	
	Delayed free recall	6/17; mild impairment
	Delayed recognition recall	Normal
Attention	Serial subtractions	Normal
	Digit span, forward	Normal
	Digit span, reverse	Mild impairment
Executive function/ attention/working memory	Similarities	Normal
	Letter fluency (*F* words)	13 words (normal >12)
	Trail Making Test Parts A and B	Normal
Visuospatial	Benson complex figure copy	Normal
	Clock drawing	Normal
Language	Naming	Mild impairment
	Sentence repetition	Normal
	Animal fluency	6 words (normal >15)
	Sentence comprehension	Normal
	Semantic knowledge	Normal

KEY POINTS

- When the cognitive test is insufficient because either the patient's symptoms are too mild to be detected by the test or the patient's symptoms are not well assessed by the test, a different test should be chosen or the test should be supplemented with additional bedside tests that would further examine the cognitive issues.
- Common cognitive profiles/patterns include amnestic, executive dysfunction, visuospatial impairment, and language dysfunction.
- In the amnestic pattern, the major difficulty is on tests of delayed recall and recognition.
- In the executive dysfunction or frontal-subcortical pattern, major difficulties on tests include impairments on the Trail Making Test Part B (letter-number sequencing) and in digit span, letter cancellation, phonemic fluency (number of words beginning with a certain letter generated in 1 minute), similarities, or serial subtractions, with relative preservation in the other cognitive domains.
- In the visuospatial impairment pattern, patients have difficulties on tasks that require drawing, whether copying a figure (eg, Benson complex figure, intersecting pentagons) or drawing an object (eg, free-drawn clock).

different test should be chosen or the test should be supplemented with additional bedside tests that would further examine the cognitive issues (TABLE 1-4). For example, if a patient presents with a progressive aphasia and a clinician chooses to administer the MoCA, additional tests should be considered to further evaluate language (CASE 1-2).

One clinically useful bedside test to add to any assessment, if not already included, is semantic or category fluency. Similar to letter or phonemic fluency, the patient is given 1 minute to list as many items in a specific category, such as animals or vegetables, as he or she can, and the score represents the number of items generated. When analyzed together with letter fluency, semantic fluency can provide additional information regarding localization of dysfunction. Letter fluency is impaired with prefrontal lesions, whereas deficits in semantic fluency occur following left temporal lobe lesions. However, the number of words generated on semantic fluency is generally greater than for letter fluency. For example, healthy individuals older than 50 years of age should be able to generate more than 16 animal names in 1 minute and more than 12 words beginning with the letter *F*.[25] Thus when the number of words on the semantic task is abnormally low, it may indicate left temporal lobe dysfunction, such as in AD (CASE 1-3). When the letter fluency is low, it may indicate a prefrontal lesion.

Clock drawing is another easy and quick-to-administer bedside test. Patients are provided with a blank piece of paper with instructions to draw the face of a clock, put in the numbers, and set the hands at 10 after 11.[25] This task is called a free-drawn clock. The clock-drawing test provides useful information about multiple cognitive processes, including executive and visuospatial functions. The executive functions of the clock-drawing test include task setting (defined as planning the size and shape of the contour, placement of the numbers, and time setting) and monitoring (defined as avoidance of duplication, such as numbers or extra hands, and self-correction of errors). Choosing the time at which patients are asked to set the clock affects the sensitivity of clock drawing for detecting executive function deficits. "Ten after 11" (not 11:10) is one of the more sensitive times because the 10 must be recoded so that the minute hand is set to the 2. Patients with frontal lesions tend to have difficulty with this abstraction and make the stimulus-bound error of placing the minute hand on the 10 instead of the 2.[1,54] Visuospatial function is required to place the elements of the clock in the correct location and orientation.

Determining the Cognitive Profile

In addition to the total score (or overall performance) that many bedside tests provide, it is clinically useful to determine the specific areas of impairment (ie, the cognitive profile). Examples and interpretation are shown in TABLE 1-6. Common cognitive profiles/patterns include amnestic, executive dysfunction, visuospatial impairment, and language dysfunction. In a given patient, more than one area can be affected; however, one area is usually disproportionately more affected than other areas. Over time, as the cognitive impairment advances in neurodegenerative dementias, all domains become involved equally in the later stages, and it is difficult, in the absence of clinical history, to determine a profile or etiology.

In the amnestic pattern, the major difficulty is on tests of delayed recall and recognition. Orientation may be impaired as well. This pattern can be seen in amnestic mild cognitive impairment or AD (CASE 1-3).

In an executive dysfunction or frontal-subcortical pattern, major difficulties on tests include impairments on the Trail Making Test Part B (letter-number sequencing) and in digit span, letter cancellation, phonemic fluency (number of words beginning with a certain letter generated in 1 minute), similarities, or serial subtractions, with relative preservation in the other cognitive domains. Disorders that commonly demonstrate this pattern include vascular cognitive impairment (CASE 1-4); normal pressure hydrocephalus; and disorders associated with parkinsonism, such as Parkinson disease dementia (CASE 1-5), dementia with Lewy bodies, progressive supranuclear palsy, Huntington disease, and corticobasal degeneration. Given the multiple differing etiologies that can provide the same cognitive profile, it will be the combination of the history, mental status examination, elemental neurologic examination, and neuroimaging that will determine a final diagnosis.

In the visuospatial impairment pattern, patients have difficulties on tasks that require drawing, whether copying a figure (eg, Benson complex figure, intersecting pentagons) or drawing an object (eg, free-drawn clock). Additional difficulties may be seen on tests that examine other domains but require intact visuospatial skills,[55] such as the Trail Making Test, a test that examines executive function. Patients with visuospatial impairment will have difficulties on the Trail Making Test as they try to find or to "see" the next element in the sequence. Thus, they will take longer to complete the test. Similarly, they may have an impairment in naming, not necessarily due to language impairment but rather to difficulty in accurately perceiving the stimuli being named (CASE 1-6). This is called nonaphasic misnaming.

In patients with language dysfunction, depending on the severity and type of the language dysfunction, difficulties may be seen with naming in association with poor performance on sentence repetition, semantic knowledge, writing, comprehension, and reading and writing (CASE 1-2). For more information on the variants of aphasia, refer to the article "Primary Progressive Aphasia and Stroke Aphasia" by Murray Grossman, MDCM, FAAN, and David J. Irwin, MD,[56] in this issue of *Continuum*. Patients with aphasia generally do very poorly on cognitive assessments that are heavily language based, such as the MMSE and

Cognitive Profiles/Patterns Seen on Cognitive Testing TABLE 1-6

Major Deficit Seen on Testing	Pattern	Example Conditions
Orientation, delayed word recall	Amnestic	Mild cognitive impairment (amnestic), Alzheimer disease
Planning and monitoring, attention, sequencing (eg, three-step command), word list generation for letters	Executive dysfunction, frontal-subcortical dysfunction	Dementia with Lewy bodies, Parkinson disease dementia, vascular dementia
Drawing	Visuospatial impairment	Posterior cortical atrophy, dementia with Lewy bodies, corticobasal degeneration
Naming, repetition, writing	Aphasia	Primary progressive aphasia
Normal testing	Not applicable	Can be seen in behavioral variant frontotemporal dementia, subjective cognitive impairment

CASE 1-4

A 74-year-old right-handed man with 14 years of education presented with a 2-year history of progressive slowness of thought, apathy, and difficulties with multitasking. His past medical history was significant for diabetes mellitus, hypertension, and hyperlipidemia, without any known clinical stroke. He was able to work but was noted to be slow and was at risk of losing his job. He took longer to memorize his shopping list and wrote the items down on a list.

On cognitive testing, he scored 22/30 on the Montreal Cognitive Assessment (MoCA)[21], as shown in the test results. On neurologic examination, he was slow to respond on cognitive testing and had a mild left hemiparesis. He had no signs of parkinsonism. Brain MRI revealed diffuse ischemic white matter changes and old subcortical infarcts (FIGURE 1-2). His presentation was felt to be most consistent with vascular cognitive impairment.

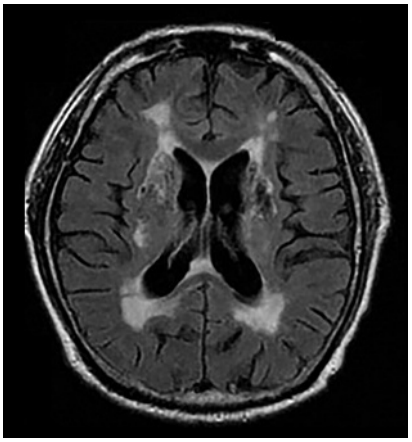

FIGURE 1-2
Axial fluid-attenuated inversion recovery (FLAIR) MRI of the patient in CASE 1-4 showing diffuse ischemic white matter changes and old subcortical infarcts.

COMMENT

This patient with cerebrovascular risk factors developed frontal-subcortical cognitive slowing in the absence of parkinsonism. Cognitive testing primarily revealed impairments in prefrontal functions, specifically impairment on the Trail Making Test Part B and in letter cancellation, letter fluency, and similarities, with preservation of memory. Although his overall performance/score on the MoCA was low, examination into the specific areas of impairment indicate that it was not due to memory impairment and likely not consistent with Alzheimer disease when considering the presentation and the neurologic examination.

Domain/Test Segment	Score
Executive function/visuospatial	
Trail Making Test Part B	0/1
Cube copy	1/1
Clock drawing	3/3
Naming	2/3
Memory	
Learning (5 words)	4 words (trial 1), 5 words (trial 2)
Attention	
Digit span (forward, reverse)	1/2
Letter cancellation	0/1
Serial 7s	2/3
Language	
Sentence repetition	2/2
Letter fluency (*F* words)	0/1 (8 words)
Abstraction	0/2
Delayed recall	5/5
Orientation	6/6

CASE 1-5

An 84-year-old right-handed man with 18 years of formal education presented with a 3-year history of progressive slowness of thought and verbal blocking that caused him to stop working. Seven years ago, 4 years before the onset of the cognitive difficulties, he developed levodopa-responsive parkinsonism, with the onset of resting tremor in his left hand, rigidity, and stooped posture. About 1 year ago, he developed spontaneous nonthreatening visual hallucinations described as seeing small children sitting in the living room.

On cognitive testing, he scored 17/30 on the Montreal Cognitive Assessment (MoCA)[21], as shown below and in FIGURE 1-3. Neurologic examination revealed left more than right rigidity in the arms and legs, resting tremor in his left hand, slow rapid alternating movements with fatiguing, and narrow-based gait with stooped posture, reduced stride length, and absent arm swing. Brain MRI did not reveal any focal cortical atrophy. His presentation was consistent with Parkinson disease dementia.

Domain/Test Segment	Score
Executive function/visuospatial	
Trail Making Test Part B	0/1
Cube copy	0/1
Clock drawing	1/3
Naming	2/3
Memory	
Learning (5 words)	2 words (trial 1), 3 words (trial 2)
Attention	
Digit span (forward, reverse)	2/2
Letter cancellation	0/1
Serial 7s	2/3
Language	
Sentence repetition	2/2
Letter fluency (*F* words)	0/1 (3 words)
Abstraction	2/2
Delayed recall	3/5
Orientation	5/6

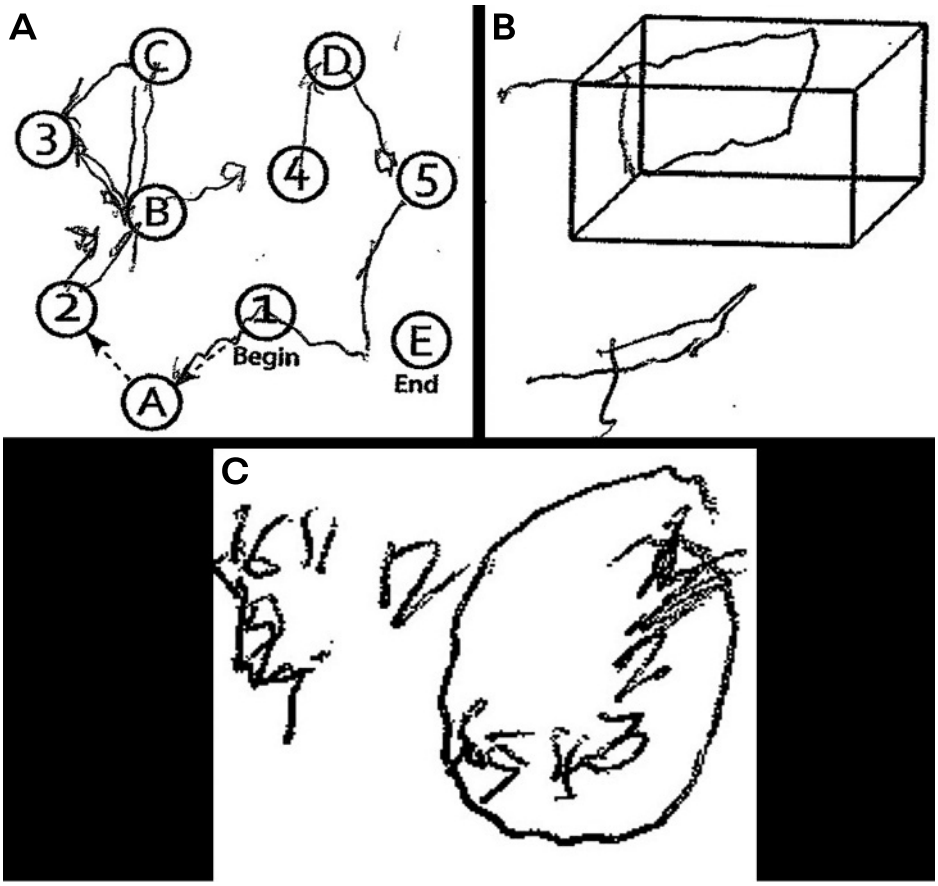

FIGURE 1-3
Performance on the Montreal Cognitive Assessment (MoCA) for the patient in CASE 1-5 demonstrating executive dysfunction on the Trail Making Test Part B (*A*) and visuospatial dysfunction on the rectangle copy (*B*) and clock drawing (*C*).

COMMENT

This case illustrates another example in which cognitive testing revealed primarily executive dysfunction, with relative preservation of memory, similar to the cognitive profile in **CASE 1-4**. However, in this case, an additional visuospatial impairment indicated frontal and parietal dysfunction. With the history and neurologic examination consistent with parkinsonism, the overall presentation is consistent with Parkinson disease dementia. The profile of both executive dysfunction and visuospatial dysfunction is typically seen in Parkinson disease dementia and dementia with Lewy bodies. This case demonstrates that all pieces of information combined, not in isolation of each other, are required to make a diagnosis.

CASE 1-6

A 45-year-old right-handed businesswoman with 14 years of formal education presented with the insidious onset of progressive difficulties seeing, reading, writing, and performing calculations of 2 years in duration. When typing, she could not "find" the letters on her keyboard. She also saw faces of people in trees. She noted difficulties with spelling and had to sound out words to help her. About 1 year before presentation, she started to have difficulty performing simple arithmetic and began to rely on a calculator. She also noted difficulties with reading and had to use her finger to follow a line of text and read letter-by-letter for each word. She had no history of symptoms of parkinsonism or rapid eye movement (REM) sleep behavior disorder. She had been recently diagnosed with major depressive disorder as she had become anxious and depressed in the absence of a prior history of any psychiatric disorder.

On cognitive examination, she scored 21/30 on the Montreal Cognitive Assessment (MoCA)[21], as shown. Additional bedside tests were administered to fully examine her cognitive issues, as shown in test results and in FIGURE 1-4.

Neurologic examination revealed full extraocular movements, normal visual fields with absent visual neglect, absent optic ataxia and ocular apraxia, absent signs of parkinsonism, normal primary sensation, and absent tactile neglect; however, agraphesthesia was noted on both sides and right-left confusion and finger agnosia were present. She had mild difficulties with body placement when sitting on the edge of the examining bed and when navigating toward a chair. Her brain MRI revealed asymmetric left more than right parietooccipital cortical atrophy, normal hippocampi, and no ischemic white matter changes or strokes. She was diagnosed with posterior cortical atrophy.

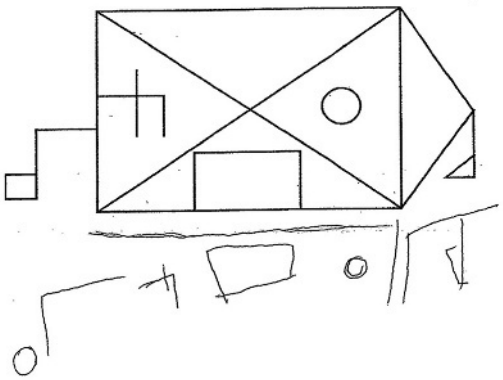

FIGURE 1-4
Drawing performance on the Benson complex figure copy for the patient in CASE 1-6 demonstrating marked visuospatial impairment in copying the figure.

Domain/Test Segment	Score
Executive function/visuospatial	
Trail Making Test Part B	0/1
Cube copy	0/1
Clock drawing	2/3
Naming	2/3
Memory	
Learning (5 words)	5 words (trial 1), 5 words (trial 2)
Attention	
Digit span (forward, reverse)	2/2
Letter cancellation	1/1
Serial 7s	2/3
Language	
Sentence repetition	2/2
Letter fluency (*F* words)	0/1 (3 words)
Abstraction	2/2
Delayed recall	1/5
Orientation	5/6

Test	Score
Trail Making Test (TMT)	
Part A	Stopped after 90 seconds; could only complete 10 out of 25 elements
Part B	Stopped after 4 minutes; could only complete 9 elements
Calculations	0/4
Multilingual Naming Test (MiNT)	13/15
Reading single words	3/12; could only read letter-by-letter and visually; lost track reading on a sheet of paper
Sentence comprehension	Normal
Sentence repetition	Normal
Benson complex figure copy	2/17; impaired
Overlapping figures (from Montreal Cognitive Assessment-B[49])	2/10 objects identified

CONTINUED ON
PAGE 698

KEY POINTS

- In patients with language dysfunction, depending on the severity and type of the language dysfunction, difficulties may be seen with naming in association with poor performance on sentence repetition, semantic knowledge, writing, comprehension, and reading and writing.
- In most cases of dementia due to Alzheimer disease, the general neurologic examination is normal. In other causes of dementia, it is clinically useful to determine the presence of specific extraocular movement abnormalities, upper motor neuron signs, or parkinsonism.

MoCA. It is important to be aware of this so these patients are not misclassified as having a severe dementia.

ELEMENTAL NEUROLOGIC EXAMINATION

The elemental neurologic examination is a necessary component in the evaluation of a patient with dementia. In most cases of dementia due to AD, the general neurologic examination is usually normal. In other causes of dementia, the presence of specific extraocular movement abnormalities, upper motor neuron signs, or parkinsonism can be particularly diagnostically useful (TABLE 1-7). With the history, mental status examination, and the general neurologic examination, a diagnosis or reasonable differential can be determined at the bedside (TABLE 1-8).

CONCLUSION

With limitations on both time and resources, an efficient deductive process is required to obtain and distill the key elements from the history, mental status examination, elemental neurologic examination, and ancillary investigations to determine the etiology of a patient's cognitive impairment. The documentation must contain elements that indicate the timing of the onset, type of progression, presenting symptom, and duration. The history should expand on this and obtain the chronologic progression of the patient's abilities in other cognitive domains, including changes to his or her personality or behavior and ability to function in both instrumental and basic ADLs. Administration of the mental status

CONTINUED FROM PAGE 697

COMMENT

This patient presented with the insidious onset of both a partial Gerstmann syndrome (TABLE 1-5) and visuospatial impairment suggestive of simultanagnosia (TABLE 1-5) that progressed over 2 years, all suggestive of a progressive neurodegenerative disorder affecting, at minimum, the left parietal and occipital regions. Other bedside tests, in addition to the MoCA, were performed to further examine her cognitive symptoms and determine areas of normalcy. Examination of the cognitive testing mirrored her clinical presentation and revealed that her cognitive profile showed predominantly visuospatial impairment with the inability to copy figures and identify overlapping figures (simultanagnosia). Although she also performed poorly on the reading tasks and on the Trail Making Test, this was caused by neither language impairment nor executive dysfunction but rather by her profound visuospatial impairment. Additional tests of her language and executive function were normal. She also demonstrated all elements of Gerstmann syndrome after testing. Examination results concurred with the clinical assessment with a predominant left parietal and bilateral parietooccipital involvement. Brain MRI revealed cortical atrophy in those areas without ischemic changes. All the information in this case was consistent with the diagnosis of posterior cortical atrophy.[40,47]

examination requires that the chosen assessment tool adequately examines the patient's primary symptom in addition to the other cognitive domains. If not, additional bedside tests should be added to the assessment. Alternatively, the clinician can administer a single test that includes a more in-depth assessment than the brief screening measures. Examples of more in-depth assessment tools are the short form of the Behavioural Neurology Assessment[20] for assessment of dementia and the TorCA[25] for assessment of mild cognitive impairment. Synthesis continues with analyzing the patient's performance on cognitive testing to determine his or her cognitive profile. This will then aid in the

Elemental Neurologic Examination Findings in Neurodegenerative Disease TABLE 1-7

Examination Element	Examination Findings	Disorders to Consider
Extraocular movements	Initiation of saccades (indicates an ocular apraxia and can be seen in corticobasal syndrome or posterior cortical atrophy); slow saccade velocity (indicates the possibility of progressive supranuclear palsy); limitation of extraocular movement (indicates the possibility of progressive supranuclear palsy)	Corticobasal syndrome, progressive supranuclear palsy, posterior cortical atrophy
Upper motor neuron signs	Pyramidal distribution weakness (weakness pattern in arms: extensors > flexors; in legs: flexors > extensors); hyperreflexia; presence of a Babinski sign (extensor plantar response)	Cerebrovascular disease (stroke), corticobasal syndrome, intracranial mass lesion
Assessment for parkinsonism	Bradykinesia; bradyphrenia; masked facies; limitation/absence of downgaze (progressive supranuclear palsy); rigidity with or without cogwheeling (distinguish between axial [progressive supranuclear palsy] versus appendicular [Parkinson disease, dementia with Lewy bodies, corticobasal syndrome] rigidity); rest tremor (not typically seen in dementia with Lewy bodies, progressive supranuclear palsy, corticobasal syndrome); fatiguing (rapid alternating movements such as finger tapping, opening/closing hands, festinating gait, progressive hypophonic speech); early postural instability (progressive supranuclear palsy)	Parkinson disease with dementia, dementia with Lewy bodies, corticobasal syndrome, progressive supranuclear palsy, cerebrovascular disease
Assessment for early focal cortical dysfunction	Balint syndrome (some or all components): simultanagnosia (inability to see objects simultaneously), optic ataxia (inability to reach a target under visual guidance); ocular apraxia (inability to move the eyes to a target purposefully)	Posterior cortical atrophy, corticobasal syndrome
	Gerstmann syndrome (some or all components): right-left confusion, finger agnosia, acalculia, dysgraphia	Posterior cortical atrophy, corticobasal syndrome, logopenic progressive aphasia
	Dressing apraxia	Posterior cortical atrophy, corticobasal syndrome
	Ideomotor apraxia	Corticobasal syndrome, Alzheimer disease
	Language (anomia)	Primary progressive aphasias

TABLE 1-8 **Common History, Cognitive Profile, and Neurologic Examination Findings in Neurodegenerative Causes of Dementia**

History/Initial Presentation	Cognitive Profile	General Physical Examination	Cranial Nerves
Rapid forgetting	Amnestic	Normal (including vitals)	Normal
Visuospatial difficulties	Visuospatial	Normal (including vitals)	Normal; visual field cut; visual neglect
Anomia	Anomia; acalculia	Normal	Normal
Slow, executive dysfunction, inattention	Executive dysfunction; slow	Signs of peripheral and cardiovascular disease	Normal
Behavioral changes (apathy or disinhibition)	Normal; executive dysfunction	Normal	Normal
Anomia/circumlocution	Anomia	Normal	Normal
Anomia; loss of semantic knowledge; prosopagnosia	Anomia	Normal	Normal
Visuospatial difficulties, slow, fluctuations	Executive dysfunction; slow; visuospatial	Normal	Normal
Anomia; parkinsonism; apraxia; visuospatial difficulties	Executive dysfunction; slow; language; apraxia; visuospatial dysfunction	Normal	Normal; slow initiation of saccades with normal velocity
Early falls, executive dysfunction	Executive dysfunction; slow; language dysfunction	Normal	Downgaze limitation; slow saccade velocity but normal initiation

localization within the brain. The cases in this article are presented with each piece of information required for the clinical diagnosis. When all the pieces of information concur, a diagnosis can often be made confidently.

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Motor Examination	Deep Tendon Reflexes and Babinski Sign	Gait	Disorder to Consider
Normal	Normal	Normal	Alzheimer disease
Normal	Normal	Normal	Posterior cortical atrophy (Alzheimer disease)
Normal	Normal	Normal	Logopenic variant primary progressive aphasia (Alzheimer disease)
Upper motor neuron pattern of weakness	Hyperreflexia; Babinski sign	Normal; slow; decreased stride length, hemiparetic gait	Vascular cognitive impairment
Normal; upper motor neuron pattern of weakness; lower motor neuron pattern of weakness	Normal; hyperreflexia; Babinski sign	Normal	Behavioral variant frontotemporal dementia with or without motor neuron disease
Normal; upper motor neuron pattern of weakness; lower motor neuron pattern of weakness	Normal; hyperreflexia; Babinski sign	Normal	Nonfluent/agrammatic variant primary progressive aphasia (frontotemporal dementia) with or without motor neuron disease
Normal; upper motor neuron pattern of weakness; lower motor neuron pattern of weakness	Normal; hyperreflexia; Babinski sign	Normal	Semantic variant primary progressive aphasia (frontotemporal dementia) with or without motor neuron disease
Appendicular rigidity with or without resting tremor	Normal	Slow, narrow-based, short strides	Parkinson disease with dementia; dementia with Lewy bodies
Asymmetric rigidity with or without unilateral upper motor neuron signs	Hyperreflexia; Babinski sign	Slow, narrow-based, short strides; hemiparetic gait	Corticobasal degeneration (corticobasal syndrome)
Axial rigidity	Normal	Narrow-based gait with early postural instability and retropulsion	Progressive supranuclear palsy

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