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Neurologic examination in the elderly

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Abstract

Clinical evaluation of neurologic disorders in the elderly requires seeking a thorough history and performing an age-appropriate neurologic examination with special attention to changes that occur with normal aging. The history should be obtained from the patient as well as collateral sources close to the patient to ensure accuracy and should include contextual elements such as medical history, social, economic, and psychological background, as well as an assessment of current functional state beyond activities of daily living. The safety of the patient, including the presence of physical, psychological, and financial threats, should be addressed during the interview. The neurological examination in older adults may need to be modified to circumvent disabilities such as hearing and visual impairment. Some elements of the neurological examination are expected to be affected by the process of aging, including pupillary reactivity, presbyopia, difficulty with ocular pursuit and up-gaze, reduced or absent distal reflexes, slower motor speed, and reduced ability to tandem walk, among others. In addition to a screening neurological assessment, evaluation of older adults with a particular complaint may require additional interview queries and examination manoeuvres. Common symptoms in the elderly include cognitive difficulties, balance and gait disorders, tremors, and neuropathy. A specialized approach to patients with cognitive difficulties must include assessment of each cognitive domain, including attention, executive function, learning andmemory, perceptual–motor function, and social cognition. Balance and gait are essential parts of the neurological examination, and in patients with a history of falls or mobility issues, should become a central part of the evaluation. In patient with tremors, careful observation of the tremor quality (amplitude, frequency, and alleviating/exacerbating factors such as rest, movement, and posture) can aid diagnosis. Evaluation of neuropathy includes determining modality (numbness, tingling, pain, and weakness) and the distribution of symptoms in order to localize the site of nerve injury, which can be supplemented with nerve conduction studies/electromyography, to guide further diagnostic workup and treatment. A combination of detailed history and examination often will suggest a likely underlying neurodegenerative disorder and guide further diagnostic workup to establish a specific diagnosis.

OVERVIEW

The clinical evaluation of neurologic disorders begins with obtaining a detailed history of the disorder and performing a thorough neurologic examination. In evaluating the older

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adult, the clinician must be aware of the factors unique to the geriatric population. In this chapter, we provide an overview of these special considerations, both in obtaining the history and performing the neurologic examination. We then outline several key elements of the history and examination when evaluating common neurologic complaints in older adults, focusing on cognitive decline, balance and gait problems, tremor, and neuropathy.

NEUROLOGIC HISTORY IN OLDER ADULTS

The aphorism, “A careful history will lead to the diagnosis 80% of the time” (Hampton etal., 1975) remains true when evaluating the older adult. Care must be taken to ensure accuracy of the information since cognitive decline, lack of awareness or insight, or patient reticence due to concerns about the potential loss of personal freedom can degrade the accuracy of information. Occasionally, patients or family members assume that symptoms may simply be a normal part of “getting old.” Therefore, the history must be obtained from both the patient and collateral sources who know the patient well. Ideally, the patient and collateral informant should be interviewed independently. When this is not feasible, the informant should be given an opportunity to write down their observations and provide them to the clinician before or after the clinic visit.

The purpose of the clinical interview goes beyond eliciting the primary concerns, i.e., the chief complaint and the history of the present illness. By the end of the interview, the clinician should have a reasonable grasp of the physiologic, social, psychologic, and economic context of the presenting symptoms. The clinical interview can begin with establishing the patient’s cognitive and motor skills prior to the onset of current symptoms by inquiring about where they were born and raised, educational background, and occupational history. Although the majority of older adults may be retired, knowing how they spend their days after retirement— including social engagements, volunteer activity, postretirement work, and entertainment activities—provides valuable information about the patient’s baseline (current) level of cognitive function, motor skills, and level of independence.

Risk factors that may endanger the life of the older adult should be explored during the clinical interview. In addition to obtaining the medical history and identifying comorbidities that are risk factors for disease (e.g., diabetes and hypertension increasing the risk of vascular dementia), the clinician should explore lifestyle factors such as use of alcohol, tobacco, and other substances; medications taken (including nonprescription) to screen for the under-recognized problem of polypharmacy; quality, quantity, and pattern of sleep; diet; and level of physical activity and exercise (Table 5.1).

Assessment of a patient’s functional ability, particularly in carrying out activities of daily living (ADL) and instrumental ADL, can be done efficiently and more reliably using a questionnaire (Table 5.2), which should be filled out by both the patient and their caregiver. A graded scale, rather than a dichotomous (Yes/No) response, provides more information and is more sensitive to change over time. Various indices have been developed, with Katz being the earliest (Katz et al., 1963), and reviewed for their accuracy and ease of use (Mlinac and Feng, 2016; Roedl et al., 2016).

Information about the patient's functional status can be used to assess safety concerns. Patient safety can be threatened through physical injury to self or others from a variety of sources, including access to guns or other weapons, driving or operating heavy machinery, falls when climbing stairs or walking on uneven or cluttered surfaces, and unsupervised administration of medications can result in over- or underdosing. In addition to physical injury, older adults are at risk of economic harm from unsupervised management of finances or by becoming avictim of scams and identity theft. Of course, addressing all the points mentioned above may not be appropriate or feasible in all care settings, and patient safety must be tailored to the clinical venue. A comprehensive safety checklist is provided in Table 5.3.

NEUROLOGIC EXAMINATION IN OLDER ADULTS

The required elements of the neurologic examination differ depending upon the patient's history and suspected potential diagnoses. Many components of the examination can be partially completed during the interview, including assessment of language, speech, behavior, and spontaneous eye and limb movements. We typically walk with our ambulatory patients to the exam room, allowing us to assess gait while conversing with the patient. Vital signs should include orthostatic measurements when there is a history of falls, syncope, or concern for autonomic dysfunction. Generally, the neurologic examination is organized based on the neuroanatomical components of the nervous system. For example, we routinely perform systematic assessment of mental status, cranial nerves, motor and coordination (including standard and tandem gait), deep tendon reflexes, and sensation. Observed focal abnormalities will then guide further detailed neurologic assessment as needed. Table5.4 outlines the pertinent components of a screening neurologic examination in older adults. Examination of patients with cognitive decline, balance and gait difficulty, tremor, and neuropathy is provided in corresponding sections in the following text.

Changes in the neurologic examination with aging

Given the increasing frequency of neurologic disorders with advancing age, a careful neurologic examination is essential to detect signs of disease. However, the clinician must also be mindful of changes in the neurologic examination that occur with aging alone. Whether such findings are "normal" depends on the definitions of normality and disease. Even when a finding is "normal" for age, interventions should be considered to optimize daily function, such as correcting vision and hearing loss. Table 5.5 summarizes relatively common "normal" changes in the neurologic examination with aging.

Pupil size and reactivity lessen with advancing age (Bitsios et al., 1996). Beginning in the mid-40s, the eyes start to lose their ability to focus on near objects, and presbyopia is nearly universal by age 55 (Quinn and Kaye, 2001). Pursuit (tracking) eye movements become less smooth with age, and saccadic intrusions appear (Jenkyn et al., 1985). In addition, there is a symmetrical reduction in upward gaze and convergence with aging. By age 75, upgaze may be limited to less than 10 degrees (Chamberlain,1970). In contrast, reduction in downward gaze can be a sign of neurologic disease such as progressive supranuclear palsy (PSP).

High-frequency hearing loss is common in older adults. Hearing can be assessed at the bedside by the whisper test (Swan and Browning, 1985). This entails standing at arm's length behind the patient, while the patient occludes one ear canal by pressing on and rubbing the tragus in a circular motion. The examiner then whispers a 3-item combination of letters/numbers, such as "4-J-7."Ifthepatientdoesnotrepeatall3correctly, a second combination is used. Inability to repeat 3 of the 6 items constitutes failure of the test. In the absence of occluding cerumen, failing the whisper test correlates with a 30dB hearing loss. This is sufficient to impact social interaction and should have prompt remediation with hearing aids.

A mild increase in muscle tone and slight decreases in muscle bulk and strength are reported with aging (Quinn and Kaye, 2001), but clinically apparent weakness should prompt further evaluation. Decreased vibration sense in the distal lower extremities to the ankles and/or absent ankle jerk reflexes may occur in the absence of a defined neuropathy (Kaye et al., 1994).

"Primitive" reflexes include the palmomental reflex (ipsilateral chin movement elicited by scratching the palm briskly along the thenar eminence), snout reflex (lip pucker in response to a tap above the lips), sustained glabellar reflex (continued blinking after each finger tap between the eyebrows, despite the examiner's instruction not to blink),and grasp reflex (hand clasp in response to a stroke of palm from proximal to distal). Primitive reflexes have also been termed "frontal release signs," but canoccur inolder adults inthe absence offrontal lobe or other neuropathology (van Boxtel et al., 2006). The presence of multiple primitive reflexes increases the likelihood of disease (Isakov et al., 1984; Benassi et al., 1990). Among the primitive reflexes, the grasp reflex is felt to have the greatest association with disease (Kaye et al., 1994; Hogan and Ebly, 1995). However, the grasp reflex lacks diagnostic specificity and can occur in conditions ranging from delirium to dementia.

"Normal" gait changes in the older adult can include a slight stoop, slower speed, and reduced tandem gait ability (Elble et al., 1991; Kaye et al., 1994). However, if these gait changes are accompanied by reduced arm swing, hand tremor, or retropulsion when the patient is pulled from behind, then parkinsonian disorders should be considered.

Neurologic examination in older adults with cognitive impairment

The neurologic examination should be tailored to the specific concern. When there is concern for cognitive impairment, a systematic cognitive assessment is essential (see Section "Approach to Patient With Cognitive Difficulties"). However, the general neurologic examination can also provide helpful clues to the cause of cognitive impairment. Routine vital signs can identify key risk factors for cerebrovascular disease such as hypertension or atrial fibrillation. Orthostatic hypotension without a compensatory pulse increase should raise suspicion for a synucleinopathy (e.g., multiple system atrophy, MSA; dementia with Lewy bodies, DLB).

Olfactory dysfunction, as assessed by standardized "smell cards," is an early finding in Alzheimer's dementia (AD) and particularly DLB (Hawkes, 2006; Williams et al., 2009). Nonneurodegenerative causes of hyposmia include sinus disease and head trauma. The

Argyll Robertson pupil, a small pupil that constricts briskly with accommodation but fails to react to direct light, has long been associated with neurosyphilis. However, this pupillary finding can be due to other midbrain pathologies, particularly given the declining incidence of neurosyphilis in the era of antibiotics (Dacso and Bortz, 1989).

The presence and type of dysarthria aid in the distinction of neurodegenerative disorders. The clinician should note changes in voice quality, speed, pitch, and articulation. Ask the patient to sustain an “ah” sound. Harsh, effortful speech and a poorly sustained “ah” are features of a spastic dysarthria. Potential etiologies include bifrontal cerebrovascular disease or frontotemporal dementia with motor neuron disease. The hypokinetic dysarthria of Parkinson’s disease is monotonous and low in volume, though patients can speak more loudly on request. Speaking rate increases over time with short rushes of speech. Ataxic dysarthria is characterized by fluctuations in pitch and volume, and an abnormal word stress pattern. Not surprisingly, neurodegenerative disorders affecting multiple motor control pathways typically cause a mixed dysarthria. For example, PSP is associated with a hypokinetic and spastic and less prominent ataxic dysarthria (Kluin et al., 1993), and MSA leads to a hypokinetic and/or ataxic, more so than spastic, dysarthria (Kluin et al., 1996).

The neurologic examination should assess for focal neurologic findings that are characteristic of a vascular dementia, such as a visual field cut, hemiparesis, hemisensory deficit, or pathologically increased reflexes. Testing for subtle upper motor neuron findings, such as a pronator drift, should be included. However, individuals with significant cognitive impairment may have difficulty complying with some aspects of the neurologic examination. Potential modifications/substitutions to assess for focal deficits are suggested in Table 5.6.

Involuntary movements can be a clue to the cause of cognitive impairment. DLB may be accompanied by either a parkinsonian tremor or a postural tremor. Tremor is not a feature of AD. Asymmetric limb dystonia is a common and disabling feature of corticobasal degeneration. Symmetric limb and facial dystonia, including blepharospasm, can occur with PSP. Focal myoclonus occurs with corticobasal degeneration, typically in the most affected limb. Multifocal myoclonus can emerge late in the course of AD. Myoclonus accompanies the rapidly progressive dementia of prion disease and may be elicited by a sudden and unexpected stimulus (startle myoclonus). The examiner can test for startle myoclonus by standing behind the patient and unexpectedly making a loud handclap. Excessive fidgetiness is an early manifestation of the choreiform movements of Huntington’s disease. Chorea characteristically interferes with sustained muscle contraction, and thus can be detected by asking the patient to sustain a tight handgrip and observing for intermittent hand relaxation (milkmaid grip). Similarly, the examiner can assess whether the patient can sustain tongue protrusion for 10 s.

Peripheral neuropathy is not a typical feature of dementing illness. However, findings suggesting a sensory neuronopathy in a patient with subacute cognitive changes should prompt an evaluation for a paraneoplastic syndrome,particularly small cell lung cancer with anti-Hu antibodies. Parkinsonism and/or a gait disturbance are prominent features of some causes of cognitive impairment. This is discussed further in Section “Approach to Patient With Balance and Gait Disorders”. Alcohol abuse may present with neuropathy and

cognitive alterations, as may B_{12} deficiency, where neuropsychiatric symptoms may be the predominant cognitive feature (megaloblastic madness).

APPROACH TO PATIENT WITH COGNITIVE DIFFICULTIES

Cognition is a broad term that refers to a set of processes that encompass acquiring new information through our senses, manipulating that information, and using it to modify our interaction with the environment and our behavior (Lezak, 2012). The wide range of human activity that is encompassed by this definition may explain why evaluation of patients with cognitive complaints can be a daunting task. Use of a schema can help ensure systematic approaches to both acquiring information and formulating a clinical impression.

A useful approach is to assess each of the major domains of cognitive function. Many classification schemes of cognitive domains have been suggested (Lezak, 2012), but generally they include the following components: complex attention, executive function, learning and memory, language, perceptual–motor, and social cognition.

Obtaining the history of cognitive decline

Each cognitive domain should be probed by asking questions about day-to-day activities that require proficiency in the corresponding domain (Table 5.7) (Mesulam et al., 2014). Evidence of impairment should prompt the interviewer to seek further information about the acuity of the onset of the deficit, temporal relation to other symptoms, time course and rate of progression, fluctuations of the deficit in time, and other associations as appropriate. The questions should be directed to both the patient and informant, not exclusively the informant, and the answers should always be viewed in the context of expected changes in healthy aging. The features that are cause for concern are those that represent a significant change from the patient’s baseline function (e.g., an English teacher who is now using only single syllable words, or an architect who cannot find his way around the house).

Complex attention

The patient and family members may report a decrease in patience, difficulty concentrating on a task, or distractibility. It is not unusual for older adults to be less tolerant of multiple external stimuli at once, such as grandchildren wanting attention when the subject is trying to watch a TV program. On the other hand, inability to focus one’s attention in the absence of external (or internal) stimuli, such as completing a long sentence, or paying attention to a narrative (in a conversation or when reading), is more suggestive of a decline in attention. In addition, healthy older adults should be able to focus their attention when there is compelling motivation to do so, for example while driving.

Executive function

Assessment of executive function may begin by asking whether the patient continues to engage in familiar complex tasks, such as preparing a multistep meal, undertaking repairs, or operating personal electronic devices or a car. Lack of organization, inability to plan trips or arrange for travel to appointments, inability to go on a trip independently, projects left

unfinished, or lack of initiative to start projects may be reported by family and friends. Any of these should prompt further investigation into possible deficits in executive functions.

Decline in complex attention and executive function are symptoms commonly seen in depression and other mood disorders. For example, lack of initiative, irritability, or inability to concentrate can be due to anhedonia and depression, and on some occasions, the patient's inability to do things as before, can result from a mood disorder. Deciphering the cause and effect is an important step, albeit a challenging one, since more than one cause of the cognitive loss occurs frequently.

Learning and memory

The word *memory* as used by patients and family can have a range of meanings. Occasionally family members of a patient who keep stalking about childhood memories report that the patient's memory is intact. Others may report that the patient no longer "remembers" how to prepare a meal, when the problem is actually with executive function. The clinician must further inquire about the patient's ability to learn new information. In addition, historical information should be probed to make a distinction between a deficit in the encoding versus the retrieval of information. In case of deficits in encoding, as in AD, recall does not improve by providing cues, context, or multiple-choice hints, whereas retrieval deficits (as in subcortical dementias, e.g., vascular dementia or DLB) tend to improve with such cues.

Language

Listening to the patient give the history, as well as noting the responses to direct questions, provides a wealth of information about language abilities. Word-finding difficulties, circumlocution, paraphasic errors, rate of speech, prosody, and attention to grammar should all be noted during the history taking. In addition, any changes in reading and writing, as noted by the patient or family members, can provide information about language deficits.

Perceptual–motor function

Difficulty with directions, especially when the patient has to find an alternate route (e.g., after missing a turn, when traffic is rerouted, or finding the way back to the table in a restaurant after going to the bathroom) implies impairment in visuospatial tasks. Other examples include difficulty reading the time on an analog clock or difficulty recognizing faces (prosopagnosia). Further queries can be aimed at distinguishing a problem with the ventral visual stream (the "what" pathway), e.g., when patients are noticed to have difficulty finding objects that are right in front of them, versus a deficit in the dorsal visual stream (the "where" pathway), when patients are noticed to have difficulty in reaching for objects that they clearly see, or keeping their sight focused on a moving target.

Difficulty with praxis, i.e., performing simple learned tasks such as grooming and dressing, also signals a deficit in perceptual-motor function. Occasionally patients have a hard time assembling objects, or putting them together, such as putting the cover on a barbeque, or assembling a puzzle. These symptoms suggest difficulty with constructional praxis in the

absence of other motor problems (i.e., tremors, neuropathy, and so on), and such an observed change in a person who is described as a handyman or “able to fix anything” is of concern.

Social cognition

Family members and close friends are the primary source of information about social cognition. Disinhibition can manifest as frequently rude, intrusive, or inappropriate comments, overly friendly behavior, or sharing of intimate information with strangers. Hypersexuality can range from making comments with sexual innuendos, to lascivious gestures or touching, to exposing private parts to strangers. Hyperoral behavior includes consuming large quantities of food even when no longer hungry, putting large portions in the mouth, eating nonedible items, and a shift in food preferences toward sweet and high carbohydrate foods. Impairments in social cognition can also led to ritualistic behavior, ranging from repetitive tapping or sounds to more complex compulsions, such as hoarding and shoplifting. Mimicking others’ actions (echopraxia) or words (echolalia) is also observed.

Affect can vary from episodes of unprovoked rage to a blunted affect with loss of empathy and little reaction to emotional events. Thoughts and beliefs may shift toward extremes, such as increased religiosity or a drastic shift in political beliefs, with the patient unable to articulate the reason for the change.

The patient is often unaware of the changes reported by the informants and may deny the changes or blame others, demonstrating lack of insight.

Cognitive examination

In addition to the general neurologic examination detailed earlier, the approach to the patient with cognitive impairment must include direct and objective assessment of each cognitive domain. Formal neuropsychologic evaluation may be indicated, as an extension to the initial assessment, the details of which are outlined in Chapter 7 of this volume. Various screening tests, including the *Mini-Mental Status Examination (MMSE), Montreal Cognitive Assessment (MoCA),* and *St. Louis University Mental Status (SLUMS)* to name a few, provide a structured battery of questions designed to sample the cognitive domains in an efficient manner, given the time constraints of a clinical encounter. It is worth noting that testing of a single cognitive domain in isolation is nearly impossible, as multiple cognitive networks are involved in performing even the most rudimentary tasks. The examiner, therefore, should be familiar with all the cognitive domains that comprise a particular task. As an example, although Trails Bis usually associated with executive function, the task also involves the perceptual–motor domain if done through visual presentation of letters and numbers. Furthermore, all tasks require a sufficient level of alertness and attention for successful completion. Hearing impairment and low vision should also be taken into account. An outline of common office-based approaches to probe the cognitive domains is given in the following text.

Attention

The substrate for all cognitive functions is the level of consciousness and alertness, which allows the patient to participate in the task at hand. The patient's level of alertness should be assessed through observation of their response to external and internal stimuli.

Evaluation of the patient's ability to sustain attention may include testing digit-span; reverse digit-span; reverse spelling; reciting the months of the year backwards; various go/no-go tests; and serial 7's. The examiner can choose the test based on the sensory modalities, education, and cultural background, or patient preference.

Digit span is a relatively pure and easily administered test of attention and correlates with working memory. The digits should be presented distinctly at regular intervals (e.g., 1 s apart), rather than in groups of numbers (e.g., 5–7-4–9, rather than 57–49). Most patients should be able to repeat a sequence of 6±1 digits forward, and 1–2 less in the reverse span (Kipps and Hodges, 2005).

In go/no-go tests, the patient is instructed to give one response to a particular stimulus, and a different response (or no response) to another stimulus. For example, a random sequence of letters is recited by the examiner, and the patient is asked to tap the table upon hearing the letter "A." An advantage of the go/no-go test is that it can be presented in any sensory modality, including visually (show one finger when examiner shows two, and show no finger when examiner shows one), or tactile (lift the right hand when the left hand is touched, but not when the right hand is touched).

Executive function

Executive function is often referenced as a distinct cognitive domain but can be viewed as an extension of "complex attention." Executive function requires the ability to focus one's attention on the task at hand when performing a sequence of tasks, filtering irrelevant or distracting stimuli, making necessary adjustments to performance cues and error signals, and shifting attention to the next task in the sequence.

Executive function can be assessed by testing phonemic fluency, where the patient is asked to produce as many distinct words starting with a particular letter (e.g., "F") in 60s, or semantic fluency, whereby the patient is asked to produce words of a given category, e.g., animals. Normative values for fluency are age-specific, but less than 10 is definitely abnormal.

Another measure of executive function is set shifting,i.e.,theabilitytoswitchbetweenalternatingpatternsorsequences.IntheTrailsBtest,thepatientisasked to connect a series of circles each containing a letter or number, in ascending order, alternating between the letters and numbers. The test can be modified by asking the patient to verbally produce an alternating pattern of letters and numbers in an ascending order, e.g. A-1-B-2-C-3-D-4.

Learning and memory

Assessment of memory begins during the interview process. The patient's ability to recall the details of medical, social, and family history gives the clinician insight into the patient's long-term memory. Their ability to provide details of more recent events such as daily activities, how they got to the clinic, their view of why they have come for the appointment, the weather condition, all give information about short-term memory.

Further testing of memory is often done by a wordrecall test. A list of words (usually 3–5 nouns) is presented at the rate of about one per second. The patient is asked to repeat the words to ensure registration. Registration trials may be repeated to assess the patient's learning curve. Instruction is given to remember the list, in any order. After a short period of distractor tasks (5 min; perhaps other parts of the screening tests in the interval), the patient is asked to recall the word list. If spontaneous recall is not complete, prompts can be offered, either by providing category cues, phonemic cues, or choices. Deficits in encoding of information result in poor recall of words that is not improved by cues or recognition, whereas improved performance with cues or recognition suggests an intact encoding circuit, i.e., hippocampus and related structures, but difficulty with the retrieval circuitry, e.g., frontal lobe or subcortical structures. Difficulty with retrieval (the "tip of the tongue" phenomenon) occurs frequently in aging and is differentiated by the subsequent recall of the information requested.

Visual memory can be tested by asking the patient to copy a visual design, removing the target design, and after a period of time spent performing a distractor task, asking the patient to draw the design from memory. This testing of visual memory probes the nondominant hemisphere, which may otherwise go unnoticed since verbal testing tends to dominate the examination of cognitive function.

Language function

Language is a complex cognitive task. Although its thorough evaluation is beyond the scope of this chapter, there are many excellent resources on this topic (Mesulam et al., 2014; Grossman and Irwin, 2018). Basic evaluation of a patient's language, however, is an essential part of cognitive assessment. Evaluation starts with careful observation of patient's language during the interview process, including the flow of speech (pausing and halting vs a smooth outflow of words), dysarthria, word-finding difficulty, use of general references rather than specific nouns ("thing," "that," "stuff," "there") or describing words (circumlocution) or the use of the object they have been asked to name, without naming it. The patient's response to all questions provides clues about their comprehension as well. Direct evaluation of language should test the basic components of fluency, comprehension, and repetition, as described in Table 5.8. Interpretation of the pattern of deficiencies in these components is provided in Table 5.9.

Perceptual–motor function

Visuospatial assessment is an important part of the mental status examination for the very reason that its deficits are often ignored by the patient and tend to be difficult to detect through casual observation. The most common assessment is asking the patient to copy a

complex figure. The complexity of the figure can range from intersecting pentagrams to a three-dimensional cube to the Rey and Osterrieth complex figure drawing. Such tasks involve wide-ranging brain regions, including the primary association visual cortex, ventral and dorsal streams, and parietal and frontal circuits. It should also be noted that construction of a figure is not purely a nondominant hemisphere task. Lesions of the dominant hemisphere tend to cause over-simplification in copying, whereas the nondominant lesions result in drawing with abnormal spatial relationships among its constituent parts.

Visual perception can be evaluated by asking the patient to locate an object in the room or various tests of simultagnosia, whereby the patient must distinguish between parts and the whole, e.g., when trying to see a large letter “A” made up of small “E’s.”

Constructional dyspraxia and neglect can be detected by asking the patient to draw a familiar object, e.g., clock face, or a named geometric figure. Assessment of parietal lobe circuits can also be done through tests of praxis, that is by asking the patient to demonstrate learned movements and gestures (e.g., use of a hammer). The instructions are provided verbally first, which also requires intact language comprehension. Pantomiming, or imitating the gesture, can isolate the deficit to apraxia. Oral praxis can be assessed by asking the patient to blow out an imaginary candle.

Social cognition

Evaluation of social cognition can be difficult since the clinician often does not know the patient’s personality, and seldom are the norms of social behavior challenged in the relatively sterile settings of the clinic. The best approach is to ask the family members or close friends of the patient about changes in their personality, behavior, or habits, including shopping and eating habits. Formal questionnaires can help guide the assessment, such as the Neuropsychiatric Inventory (Cummings et al., 1994) and the Frontal Behavioral Inventory (Kertesz et al., 1997).

Formulation of clinical impression

Localization of cognitive disorders

The traditional localization approach to neurologic disorders, which was based largely on discrete vascular lesions, has had limited value in cognitive disorders because of the multiple regions utilized in most cognitive behaviors. With the advent of functional neuroimaging, our understanding of brain–behavior relationships has shifted toward disruption of large and distributed neural networks, or connectomes, as the cause of observed symptoms. Our current model assumes that observed dementia syndromes, as an example, represent the set of connectomes that have been disrupted by one or more pathologic processes, hence the observed variability in symptoms from the same pathologic process. Nonetheless, an attempt at mapping the constellation of symptoms onto a general regional distribution of pathology can be helpful in formulating a clinical impression. Table 5.10 outlines the cognitive domains and their corresponding gross neuroanatomical areas.

Temporal patterns of cognitive disorders

As with other neurologic disorders, the tempo of symptom onset and progression is useful in narrowing down the etiology of disease. An acute (hours to days)decline in cognition is more likely to be due to a vascular insult, seizures, trauma, infection, or toxins (endogenous or exogenous). Systemic stress, even brought on by seemingly innocuous conditions such as dehydration, can cause an acute cognitive decline in older adults. Occasionally, change in the environment may present as an acute cognitive decline, e.g., in the case of hospital-induced delirium or sun-downing, or the loss of a partner who was performing most of the ADLs for the couple, and unmask an underlying chronic cognitive disorder.

Most neurodegenerative disorders have an indolent course. The overall progression of these disorders tends to occur over years, which often goes unnoticed until a decompensating event, such as infection or surgery, unmasks the decline. DLB is an exception to this rule; large fluctuations in cognition and level of consciousness is one of the diagnostic criteria for DLB, along with visual hallucinations, REM behaviour disorder, and parkinsonism.

Putting it all together: Temporospatial patterns of neurodegenerative disorders

In patients with chronic progressive cognitive impairment, the etiology can be suggested by the pattern of involved cognitive domains. AD involves multiple domains, most often the medial temporal lobe and posterior parietal regions. Hence, difficulty encoding information (memory loss) and construction dyspraxia are often presenting features. Aphasia may accompany AD, or in some cases be the predominant feature, as in the logopenic variant of Primary Progressive Aphasia. Fluency is variable but by and large preserved. Speech has a paucity of specific nouns, and meaning is conveyed through vague references (“that” or “the thing”) and circumlocution (“that thing you wear that helps you with the time”). Repetition, even of short sentences is difficult, but comprehension remains intact.

Frontotemporal dementia (FTD), on the other hand, has more frontal features, including deficits in social cognition (behavioral variant; bv-FTD), but relatively well-preserved encoding of information, although delayed recall may be diminished due to involvement of frontal circuits. Deficits in executive function also suggest involvement of frontal lobes; however, executive function deficits are common findings in many of the neurodegenerative disorders. A frontal predominance of symptoms can also be seen in vascular dementia. Presence of behavior changes, or features suggesting involvement of anterior temporal lobes, i.e., semantic language difficulties (manifested as difficulty in comprehension of simple words and surface dyslexia yet preserved fluency and repetition) can suggest FTD, whereas visual hallucinations, REM sleep behavior dis- order, fluctuations in cognition/level of consciousness, as well as Parkinsonian features suggest DLB. Vascular dementia lacks a specific cognitive profile and often coexists with other dementia. Isolated subcortical cerebrovascular disease typically affects attention and executive function.

Isolated language deficits at onset are characteristic of the primary progressive aphasias (PPA), and involvement of the dominant hemisphere. Semantic and logopenic variants have been described in the earlier text. Nonfluent/agrammatic PPA presents with impaired fluency, apraxia of speech, and largely intact comprehension. Although comprehension is

largely intact, complex sentences, especially those requiring interpretation of a conjunction (If the lion was killed by the fox, who ends up dead?), present problems.

Difficulty with visuospatial tasks, with little to no involvement of other cognitive domains, should raise suspicion for pathologies involving the posterior brain regions, such as DLB and posterior cortical atrophy. Of course, AD also involves the nondominant hemisphere and can also present with similar features.

The cognitive patterns associated with most common forms of dementia are summarized in Table 5.11. It is important to emphasize that the correlation between the clinical disease and the histopathology is not strict. Terms such as frontotemporal lobar degeneration or corticobasal degeneration, which denote histopathologic diagnoses, should be avoided when formulating a clinical impression in favor of more clinically descriptive terms such as frontotemporal disease, or corticobasal syndrome, given the one-to-many relationship between clinical impression and histopathologic correlates.

APPROACH TO PATIENT WITH BALANCE AND GAIT DISORDERS

Changes to gait are an inevitable part of aging (Pirker and Katzenschlager, 2017; Baker, 2018; Ronthal, 2019). Older adults walk more slowly, take smaller steps, have greater difficulty navigating inclines and declines, and are at greater risk for falls. The reasons for slowed, more cautious gait are often multifactorial, including muscle weakness, reduced vision, pain (often from arthritis), deconditioning or physical inactivity, obesity, and even cognitive impairment (Verghese et al., 2016). In this same volume, Cohen and Verghese (Chapter 22) details the relationship between gait changes and cognitive impairment, highlighting the Motoric Cognitive Risk Syndrome.

Although the multifactorial nature of gait difficulties in the elderly can lead to diagnostic challenges, certain features noted on the examination will suggest specific gait disorders, as summarized in Table 5.12. Parkinsonism is one of the most common contributors to gait difficulties in older adults. The classic gait of idiopathic Parkinson disease (PD) includes shortened strides, stooped posture, reduced arm swing (often asymmetric), festination (progressive acceleration in an effort to maintain balance) and en bloc turning. Patients with PD also have difficulty rising from a chair, initiating gait, maintaining balance when pulled from behind (retropulsion on the pull test), and associated features include generalized bradykinesia, reduced speech volume (hypophonia), cogwheel rigidity on passive movement of the limb, and asymmetrical rest tremor. The presence of a rest tremor, indicating tremorpredominant PD, typically portends a better response to levodopa and slower course of disease. In contrast, individuals with a postural instability gait form or the akinetic-rigid form of PD may have lesser response to dopaminergic agents and a more debilitating and rapid course of disease. The diagnosis and treatment of PD is discussed in greater detail in this volume by Deeb, Nozile-Firth, and Okun (Chapter 14).

Distinguishing among the various causes of parkinsonism, including the atypical parkinsonian disorders, is aided by noting the presence of associated features, as summarized in Table 5.13. The principal atypical parkinsonian disorders are MSA, PSP, cortical basal syndrome (CBS), and DLB. All four typically show a poor response to

levodopa and are more rapidly progressive than PD. Additional signs that suggest atypical parkinsonism over PD are symmetrical parkinsonism at onset, early problems with falls, early and prominent cognitive changes (a frequent presentation of DLB), the absence of a typical rest tremor or presence of an atypical (myo-clonic, intention) tremor, and various cortical signs, including apraxia, aphasia and alien limb. Atypical parkinsonian disorders are covered in greater detail in this volume by Armstrong and McFarland (Chapter 16).

An ataxic gait disorder differs greatly from a parkinsonian gait disorder. Patients will have a lurching wide based unsteady gait with uncoordinated steps, often associated with upper limb ataxia, ataxic dysarthria, and oculomotor findings. While profound ataxia leads to slowed finger tapping (a common neurologic test), it is usually not confused with the bradykinesia of parkinsonism. Gait ataxia accompanied by autonomic features and urinary incontinence raises concern for possible MSA. It is important to recognize that damage at virtually any point of the neuraxis can cause ataxic gait, ranging from normal pressure hydrocephalus (NPH) to profound sensory neuropathy or neuronopathy (van Gaalen and Van de Warrenburg, 2012). A careful examination will narrow the diagnostic possibilities, but brain and cervical spine imaging is often critical in guiding further diagnostic workup. While most later onset ataxia is not genetically based, brain imaging may suggest specific genetic disorders. For example, fragile Xtremor ataxia syndrome (FXTAS), which typically manifests with ataxia, tremor, and cognitive impairment in older men (Hall and Berry-Kravis, 2018), often shows distinctive white matter signal abnormalities in the middle cerebellar peduncles.

When cerebellar ataxia is accompanied by neuropathy and vestibular areflexia, one should consider Cerebellar Ataxia, Neuropathy, and Vestibular Areflexia Syndrome (CANVAS), which was recently discovered to be perhaps the most common sporadic genetic cause of progressive ataxia (Cortese etal., 2019). A careful history is needed to ascertain exposure to cerebellar toxins such as chronic alcohol consumption, anti-neoplastic agents, and lithium (Manto and Perrotta,2018).

Gait difficulty due to distal sensory neuropathy is common in the elderly. It can be associated with distal weakness, even leading to bilateral foot drop, as well as worsened balance and gait when deprived of visual input. Hence, walking at night is more difficult, and a positive Romberg sign on the examination should alert the clinician to rule out causes of sensory neuropathy, including B_{12} deficiency.

Cerebrovascular disease, whether due to clinically manifest stroke or small vessel disease, can present with gait disorder. A hemiparetic gait will have asymmetric features reflecting damage from a manifest stroke, including hyperreflexia and increased tone on the affected side and a tendency to circumduct the affected foot when walking. Extensive subcortical small vessel disease can manifest with symmetrical parkinsonism and gait disorder including hyperreflexia and increased tone. Cervical spine stenosis leading to myelopathy likewise can cause symmetrical lower limb hyperreflexia and spasticity, as can hereditary spastic paraparesis (HSP). HSP, however, usually manifests earlier in adulthood, is slowly progressive, and is often characterized by a distinctive scissors gait.

Frontal gait disorder is accompanied by subcortical or cortical cognitive dysfunction localizing to the frontal lobes. Patient may appear unable to take steps, as if the feet are magnetically trapped, leading to poor gait initiation and a widened base. When lying down, the patient may retain the ability to “pedal the bicycle” with their legs. This gait disorder can occur in vascular parkinsonism, neurodegenerative or cerebrovascular damage to the frontal lobes, or in NPH. NPH is classically associated with the clinical triad of cognitive impairment, gait imbalance, and urinary incontinence. The diagnosis of NPH cannot be established simply by noting enlarged ventricles on brain images, since enlarged ventricles often represent the byproduct of parenchymal damage from neurodegenerative or vascular disorders (Espay etal.,2017).When our clinical suspicion for NPH is high, we refer patients for an inpatient admission for placement of a lumbar drain to rigorously assess the patient’s cognition, gait, and balance in response to withdrawal of cerebrospinal fluid. Only in the setting of a robust response to this intervention do we then consider shunt surgery, which in the right individuals can be highly beneficial (Giordan et al., 2018).

Functional or psychogenic gait disorder can occur in the elderly although it more commonly manifests in younger individuals. Characteristics that suggest a psychogenic gait disorder include distractibility of the impaired gait, fluctuating abnormalities, and profoundly altered gait/balance in the absence of any other neurologic findings, known as astasia abasia. It is important to remember that fear of falling and loss of confidence in one’s balance are common in the elderly, particularly in the presence of sensory neuropathy (Hewston et al., 2018), and should not be confused with psychogenic gait disorder.

“Dizziness” is another common complaint associated with gait concerns in the elderly (Fife, 2017; Alyono, 2018; Jahn, 2019). Uncovering the basis of dizziness begins with carefully evaluating what the patient means by “dizzy.” It can cover everything from feeling unsteady, lightheaded, to a feeling that the room is spinning. More common vestibular causes of dizziness include benign paroxysmal positional vertigo and bilateral vestibulopathy. Whereas the former is often associated with a spinning sensation during an event and worsening of symptoms with the head in a particular position (e.g., right ear down), the latter is instead associated with oscillopsia (jumpy visual fields) when the head is moving, and both are associated with imbalance. Nonvestibular causes of dizziness include sensory neuropathy, orthostatic hypotension, and ataxia. Accordingly, obtaining orthostatic blood pressure measurements is an essential part of the evaluation of dizziness in the elderly.

APPROACH TO PATIENT WITH TREMOR

Tremor increases in prevalence with age but is not expected due to aging alone. The primary types of hand tremor in the older adult are: (1)Exaggerated physiologic tremor, (2) Essential tremor (ET), and (3) Parkinsonian tremor. These can be distinguished by their appearance and modifying factors (Table 5.14). The history should ascertain where and when the tremor occurs, concurrent medications, exacerbating and alleviating factors, and whether there are accompanying parkinsonian symptoms. The hands should be examined for tremor at rest (e.g., hands resting in the patient’s lap), with sustained posture (e.g., arms held outstretched), and with movement (e.g., touching finger to nose or while writing).

Physiologic tremor is a fine, barely visible tremor that occurs in healthy individuals when sustaining a posture or movement. Stress, medications, and toxins can exacerbate this tremor and make it clinically apparent. It responds to removing the causative factor.

ET also occurs with posture and movement but the tremor has a lower frequency than physiologic tremor. ET can also manifest as a head or voice tremor.

The hand tremor is bilateral and worsens with intentional movement, e.g., when bringing objects, such as a cup or spoon, near the mouth. It is often apparent when providing a writing sample, drawing a spiral, or pouring water from one cup to another. Alcohol consumption often ameliorates the tremor, as do benzodiazepines. ET often runs in the family as a dominantly inherited trait but without a clearly defined genetic basis. Late-life ET accompanied by ataxia and cognitive impairment in a male patient should raise concern for FXTAS (Hall and Berry-Kravis, 2018), discussed earlier.

Parkinsonian tremor has a coarse, “pill rolling” appearance (so named because the tremor of the fingers was similar to that of early pharmacists molding a compound into a pill), and is most apparent when the hand is at rest. The tremor lessens with movement but may reemerge after several seconds when a new hand posture is held. Chin tremor may be present. Accompanying parkinsonian findings include bradykinesia and cogwheel rigidity. A unilateral or asymmetric hand tremor is typical with Parkinson’s disease in the initial stages of the disease, whereas drug-induced parkinsonian tremor is often symmetric.

APPROACH TO THE ELDERLY PATIENT WITH NEUROPATHY

Peripheral neuropathy affects an estimated 5%–10% of older adults (Martyn and Hughes, 1997; Hanewinckel et al., 2016). It is important to recognize it given its associated morbidity, including an increased risk of falls. The initial step in evaluating a peripheral neuropathy is identifying the type based on the localization of the nerve injury. The most common types are mononeuropathy, radiculopathy, and polyneuropathy (Callaghan et al., 2015a). These types are distinguished by the anatomic distribution of numbness, weakness, pain, and/or hyporeflexia. Thus, the examination should be sufficiently detailed to determine if the findings correspond to the distribution of a single peripheral nerve (mononeuropathy), nerve root (radiculopathy), or a more generalized peripheral nerve process (polyneuropathy). For example, ankle dorsiflexion weakness(foot drop)can be duetoaperoneal mononeuropathy, L5 radiculopathy, or distal polyneuropathy. Any of these could also affect toe extension and ankle eversion. However, weakness in ankle inversion distinguishesanL5radiculopathyfromaperonealneuropathy, and additional involvement of ankle plantar flexion and toe flexion suggests a more diffuse process. Electromyography (EMG) and nerve conduction velocities (NCT) determination are helpful when the localization is unclear by examination alone.

Peripheral polyneuropathy can be classified further by location (distal or proximal, symmetric or asymmetric) and the involved modalities (motor, sensory, or autonomic). The most common subtype is a distal symmetric polyneuropathy (DSP) (Callaghan et al., 2015b). This manifests with symmetric numbness, tingling, and/or pain beginning in the feet and gradually ascending up the leg over time. DSP is an axon length-dependent process,

such that the fingers start to become symptomatically involved around the time that the lower extremity symptoms have risen to the knees. Weakness also begins distally, but typically occurs late in the course. Examination reveals absent distal reflexes and loss of vibration and pinprick in a “stocking” fashion, or a “stockingglove” distribution when the symptoms have reached the hands as described above. Patients should be asked to walk on their heels in order to assess for weakness in ankle dorsiflexion.

The distal sensory polyneuropathy, or DSP, has numerous potential causes. Diabetes is the most common, accounting for up to half of DSP cases (Hanewinckel et al., 2016). Prediabetes can also cause DSP. Other common causes are chronic excess alcohol, B_{12} deficiency, chronic kidney disease, chemotherapy, and some other medications, paraproteinemia, and hereditary motor and sensory neuropathy, a.k.a. Charcot Marie Tooth (CMT) disease. The latter is distinguished by early motor involvement, with hammertoes and high arches and a positive family history of nondiabetic neuropathy. CMT is classified as either demyelinating or axonal, based on clinical and electrophysiologic features, and this classification helps guide genetic testing.

The recommended diagnostic evaluation for a DSP with a typical clinical course but unknown etiology should have the following tests: a comprehensive metabolic panel, complete blood count (CBC), serum protein electrophoresis with immunofixation, glucose tolerance test, and a test for Vitamin B_{12} level (Callaghan et al., 2015b). Atypical features, such as asymmetry, nonlength predominance, predominant motor or prominent autonomic involvement, or an acute/subacute onset, should prompt further evaluation with an EMG and NCTs, and more extensive laboratory testing based on the clinical scenario. This may include additional testing for nutritional deficiencies (B_6, vitamin E, thiamine, copper), autoimmune diseases (hypothyroidism, lupus, rheumatoid arthritis, Sjogren’s syndrome, sarcoidosis), or infections (HIV, lyme, hepatitis B or C). However, even with extensive testing, there may be no identifiable cause of a peripheral polyneuropathy in up to 25% of cases (Dyck et al., 1981; Callaghan et al., 2014).

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Table 5.1

Pertinent risk factors and lifestyle habits

Medical conditions	Diabetes, hypertension, cardiovascular disease, strokes, chronic infections (e.g., HIV, hepatitis)
Environmental exposures	Drinking water (heavy metal exposure, especially lead and mercury), combustion heaters (CO poisoning)
Substance use	Alcohol, tobacco, illicit, or recreational drugs (including marijuana), ethnic remedies, and contamination of any of these
Polypharmacy	Medications, including supplements and nonprescription drugs (e.g., diphenhydramine for sleep); can be checked against the Beers criteria (Panel BtAGSBCUE, 2019)
Sleep habits	Quantity (time to bed and rise, number of interruptions), quality (refreshed when getting up, daytime sleepiness), and difficulty falling or staying asleep
Diet	Number and content of meals, including salt intake (from canned and processed foods), refined sugar, source (plant versus animal products), and hydration
Exercise	Type of exercise (aerobic, resistance training), frequency, and duration (vs duration of sedentary activities)
Cognitive lifestyle	Reading, writing, music, arts and crafts, games, other hobbies
Socialization	Social network, including family and friends

Table 5.2

Functional assessment in older adults

Activities of daily living
Communicating
Eating
Toileting
Bathing
Dressing
Walking
Transferring (getting in and out of bed, chairs, cars)
Going up and down stairs
Instrumental activities ofdaily living
Taking medications as prescribed
Handling own finances (writing checks, paying bills, balancing checkbook)
Shopping alone (groceries, clothes, and household items)
Playing a game of skill (card games, chess, or working on a hobby)
Preparing simple foods (heating water, making coffee, safely operating a stove)
Preparing a balanced meal
Keeping track of current events
Paying attention to and understanding TV programs, books, or magazines
Remembering appointments, family occasions, holidays
Traveling out of the neighborhood, driving, or arranging transportation
Assessment can be done by rating each activity as: independent, needing assistive devices, needing verbal cues and reminders, needing hands-on help, or totally dependent on others

Table 5.3

Safety checklist

Firearms
Driving
Medication safety (over and under dosing)
Use of heavy or dangerous machinery (lawn mower, table saw, and so on)
Lighting (sufficient lighting to avoid falls)
Floor coverings (area rugs, slippery surfaces)
Stairs and steps (including visual contrast to identify steps)
Burn risks (stove, iron, gas)
Fire safety (ability to heed fire alarms, use fire safety equipment)
Drowning risks (pools, tubs)
Summoning help for emergencies (operating phone, medical alert systems)
Financial safety (sufficient supervision of bills, accounts)
Safeguards against hackers and scammers (by monitoring credit scores, account balances)
Wandering risk (wearing identification bracelet)
Hearing difficulty (ability to hear safety alarms)

Table 5.4

Key components of the screening neurologic examination

Component	Assessments
Mental status	Attention, executive function, language, memory
Cranial nerves	Vision and hearing, eye movements, visual fields, facial symmetry
Motor/Symmetry,	Coordination coordination, and speed of limb use; muscle tone, test for pronator drift, involuntary movements
Sensation	Joint position, double simultaneous stimulation
Reflexes	Pathologic or asymmetric reflexes
Gait	Arising from chair, casual, and tandem gait

Table 5.5

Changes in the neurological examination with aging

Smaller pupil size and reduced reactivity
Decreased near vision (presbyopia)
Breakdown of smooth eye pursuits with saccadic intrusions
Reduced upgaze and convergence
High-frequency hearing loss
Mild increase in muscle tone, decrease in muscle bulk, and subtle reduction in strength
Decreased vibration sense in distal lower extremities
Absent ankle jerk reflexes
Presence of primitive reflexes
Gait with slight stoop, slower speed, and reduced tandem ability

Table 5.6

Modifications in neurological examination if severe cognitive impairment is present

Test	Original method	Modification
Visual fields	Count fingers	Detect hand motion or blink to threat
Eye movements	Track examiner's finger	Track examiner's face or patient's own finger
Strength	Resist movement of examiner	Maintain limbs in antigravity position after release
UMN screen	Pronator drift	Symmetry of arm rolling
Proprioception	Detect joint movements with eyes closed	Train on task with eyes open first and/or perform Romberg
Lower extremity coordination	Heel-knee-shin	Toe to examiner's finger

Table 5.7

Cognitive domains and representative symptoms

Cognitive domain	Clinical observations
Attention	Difficulty focusing on a task, reading, watching a show. Walking into a room and forgetting why
Executive function	Difficulty multitasking, making a schedule/calendar, preparing a full meal, planning a trip
Learning and memory	Forgetting recent events, such as details of holidays and celebrations. Asking repetitive questions
Language	Struggling to find words; talking around words. Speaking in short, grammatically incorrect sentences. Difficulty comprehending spoken or written words. Difficulty reading sight words
Perceptual-motor function	Difficulty finding way when driving (especially if rerouted), finding way back to the table after going to public restroom, telling time on analog clock, performing learned tasks (using knife/fork and tools), recognizing faces
Social cognition	Marked changes in personality, behavior, habits, or beliefs

Table 5.8

Evaluation of language components

Language component	Assessment
Fluency	Word production: phonemic (number of words starting with a particular letter in 1 min) and semantic (nouns in a category, e.g., animals, in 1 min). Confrontational naming (pictures, diagrams, or items in the room). Scene description (e.g., “cookie theft picture”)
Repetition	Sentence repetition, which should include a sentence with semantically vague words and embedded clauses such as: “*I only realized the other day that he is the one who was making all the fuss*”, and a sentence with a clear meaning, such as “*The worms crawl onto the side walk when the pavement gets wet from the rain*”
Comprehension	Definition of simple words, or description of their function. Comprehension of multistep commands. Comprehension of reversed clauses with a connector words: “*point to the ceiling after you point to the ground*”
Reading	Reading and retelling a short story. Reading sight words (“yacht,” “colonel”)
Writing	Write a short sentence (self-generated), and a dictated sentence

Table 5.9

Patterns of language deficits

Component	Semantic PPA	Nonfluent/Agrammatic PPA	Logopenic PPA
Fluency	Intact with respect to rate and effort, but speech content becomes poor with circumlocution and verbal paraphasia. Naming deficits reflect semantic deficits, and do not improve when asked to describe the objects	Impaired rate with increased effort and apraxia of speech	Rate is often preserved, but may be reduced due to word search pauses, which is often compensated by circumlocution and phonemic paraphasia. Naming deficiencies reflect word-finding rather than semantics
Comprehension	Impaired even for single words. No difficulty with grammar	Single word comprehension is intact. Complex sentences and reversed clauses impaired	Single word comprehension is preserved. Difficulty with complex sentences, with reversed clauses
Repetition	Preserved for single words and simple sentences. As the semantic content increases, repetition declines	Preserved	Repetition is variable, and tends to be worse with longer sentences, rather than grammar complexity
Reading/Writing	Surface dyslexia and dysgraphia (write “no” instead of “know”)	Grammar deficits	Preserved

Table 5.10

Neural circuits associated with cognitive domains

Complex attention	**Frontal lobe**
Executive function	Prefrontal including dorsolateral prefrontal cortex (DLPFC)
Learning and memory	*Encoding*: medial temporal lobe, hippocampus, entorhinal cortex
	Consolidation: multiple regions and networks
	Retrieval: Frontal lobe, subcortical projections, as well as hippocampus
Language	Dominant hemisphere
	Fluency: anterior circuits, including frontal and anterior-superior temporal lobe (Grossman et al., 2013)
	Comprehension: posterolateral temporal cortex, as well as left inferior frontal cortex, and anterior cingulate (Cooke et al., 2006)
Perceptual-motor function	Nondominant hemisphere
	Visual perception: nondominant primary visual and association cortices, temporooccipital and ventral stream
	Spatial processing: visual association cortices, occipitoparietal lobes, dorsal stream
	Praxis: bilateral parietal lobes
Social cognition	Prefrontal cortex, anterior temporal lobe

Table 5.11

Principal cognitive domains affected in neurodegenerative disorders

	Complex attention	Executive function	Learning and memory	Language	Perceptual-motor function	Social cognition	Other
Alzheimer		X	X				
Vascular	X	X					
DLB	X	X			X		
PSP	X	X					
CBS	X	X					Apraxia
bv-FTD						X	
nf-PPA				X			
sv-PPA				X			
lp-PPA				X			
PCA					X		

Key: AD, Alzheimer disease; bv-FTD, behavioral variant frontotemporal dementia; CBS, corticobasal syndrome; DLB, dementia with Lewy bodies; lp-PPA, logogenic primary progressive aphasia; nf-PPA, non-fluent/agrammatic primary progressive aphasia; PCA, posterior cortical atrophy; PSP, progressive supranuclear palsy; sv-PPA, semantic variant primary progressive aphasia; VD, vascular dementia.

Table 5.12

Types of gait disorder

Gait disorder	Features
Parkinsonian	Short strides
	Stooped posture
	Reduced arm swing
	Festination
	en bloc turn
Ataxic	Wide-based
	Irregular foot placement
	Upper limb incoordination
	Ataxic speech
Neuropathic	Distal sensory loss
	Lower limb areflexia
	Distal weakness (foot drop)
Hemiparetic	Asymmetric weakness
	Hyperreflexia
	Increased tone
	Limb circumduction
Frontal	“Magnetic” gait
	Poor gait initiation
	Widened base
Functional	Fluctuating
	Distractible
	Astasia abasia

Table 5.13

Features suggesting atypical Parkinsonian disorders

	MSA	PSP	CBS	DLB
Rapid progression	X	X	X	X
Poor response to levodopa	X	X	X	X
Early falls	X	X	X	
Early cognitive changes		X	X	X
Symmetrical parkinsonism	X	X		X
Dysautonomia	X			X
REM sleep behavior disorder	X			X
Irregular tremor	X		X	
Apraxia			X	X
Aphasia			X	X
Supranuclear palsy		X	X	
Cerebellar signs	X			
Laryngeal stridor	X			
Alien limb			X	

Table 5.14

Types of hand tremor and distinguishing features

	Exaggerated physiologic	**Essential tremor**	**Parkinsonian tremor**
Occurrence	Posture, movement	Posture, movement	Resting limb
Frequency	6–12 Hz	5–9 Hz	4–6 Hz
Other findings		Head or voice tremor	Chin tremor, bradykinesia, cogwheel rigidity
Causes	*Drugs*: prednisone, lithium, valproic acid, SSRIs, TCAs, beta adrenergic drugs, caffeine and other stimulants	Familial (genetic)	Dopamine-depleting drugs, including antipsychotics and antiemetics
	Anxiety		Parkinson’s disease
	Hyperthyroidism		
	Alcohol or sedative withdrawal		