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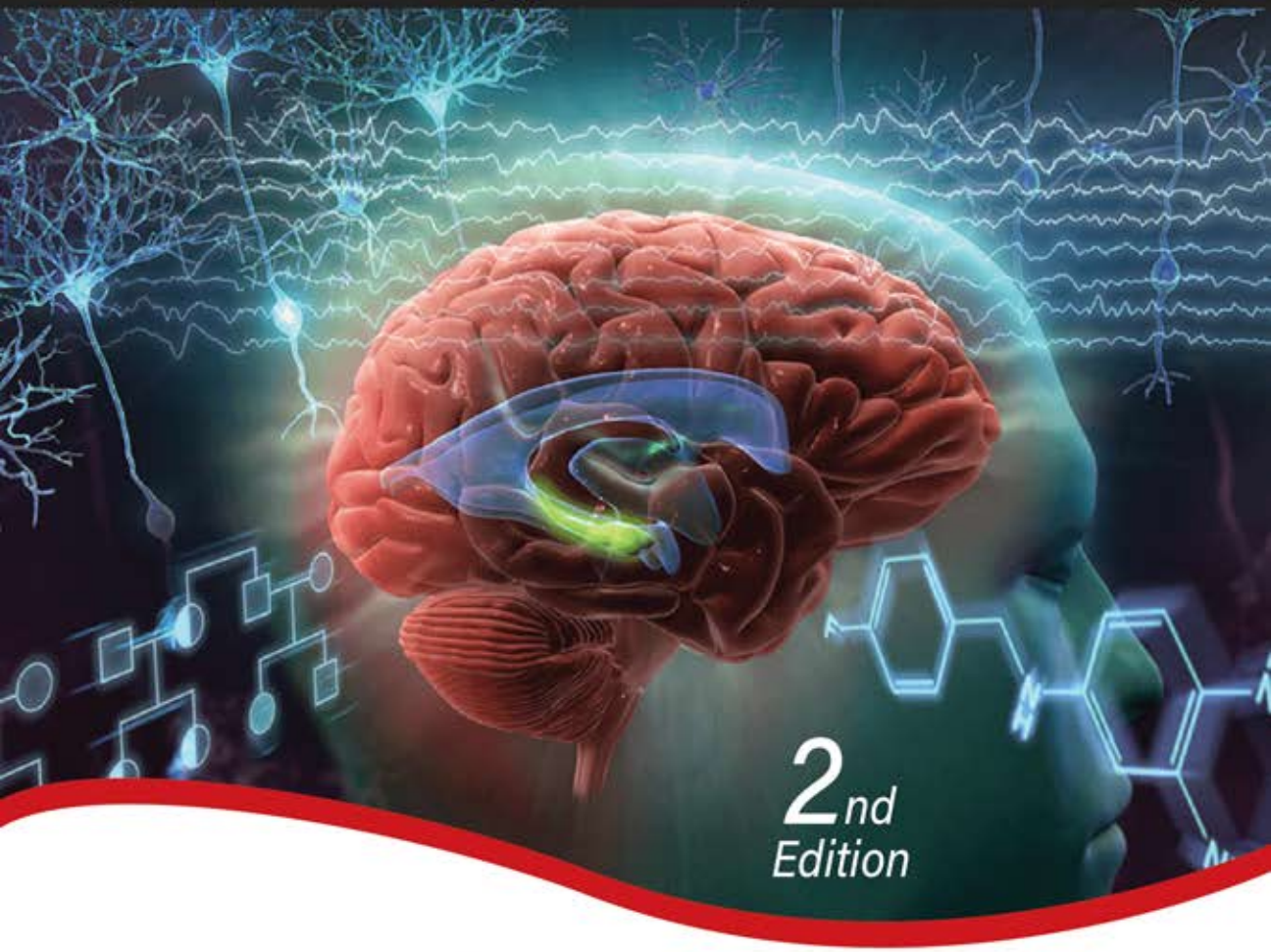

2nd Edition

Ward Rounds in Clinical NEUROLOGY

Long and Short Cases

Ravi Yadav
Girish Baburao Kulkarni

JAYPEE

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CHAPTER

3

Approach to a Case of Dementia

Faheem Arshad

CASE STUDY

A 66-year-old right-handed gentleman, multilingual, with 16 years of education, a teacher by occupation, presented with progressive cognitive complaints of (1) forgetfulness of 3 years' duration, (2) wayfinding difficulty for 2 years, (3) difficulty in wearing clothes for 2 years, (4) behavioral disturbances since one and a half years, (5) speaking difficulty since 1 year, and (6) difficulty in handling tools since 1 year.

History of Present Illness

The patient's symptoms began with an insidious onset of forgetfulness 3 years before presentation. His wife reported that he would repeatedly ask the same questions, frequently misplaced personal items, and would search for them. He would forget passwords, appointments, and conversations and could not concentrate. It has progressed to the point that he has difficulty remembering recent events (e.g., he could not recall attending his nephew's wedding ceremony 2 months ago), and he repeatedly asks for food and medications despite having taken them once. Occasionally, he tends to forget the dates as well. Currently, he is unaware of the current events in the news. He stopped doing his checkbook and filing his taxes a year ago. His wife has been handling his work and administering medications for the past year.

One year after the symptom onset, the patient began having trouble finding his way home while driving. This progressed to the extent where he began to experience problems at home, such as entering the kitchen instead of the bathroom and requiring assistance.. His challenges in wayfinding further progressed until he ultimately got lost in the house he had lived in for 10 years. His wife also noticed that he used to get confused while wearing his shirt and sometimes used to wear it inside out. He also had difficulties using his mobile phone and pressing the digits in the correct order. For one and a half years, there was a slight change in his personality in the form of a progressive loss of interest in his previous hobbies, irritability, adamant behavior, and lack of concern for his family members. Six months later, he developed difficulty speaking, causing frequent pauses in conversation and hesitations in initiation. Sentence structure in emails was abnormal, but his comprehension was intact. He had trouble retrieving the names of relatives. For the last 6 months, he has had clumsiness and difficulty using scissors and activities such as combing, brushing, and holding a pen for writing, followed by the inability to even draw or write

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simple signs. His memory worsened. Further, he had difficulty recognizing familiar faces and could not plan. He could not perform everyday activities independently and depended on family members for his daily activities.

There was no history suggestive of compulsive or ritualistic behaviors, food faddism, disinhibitions, visual or auditory hallucinations, repetitive behaviors, weakness on any side, imbalance or falls, tremors, twitching of muscles, dream enactment behavior, fluctuations in sensorium, seizures, myoclonus, insomnia, snoring, bladder/bowel incontinence, or swallowing difficulty. They denied any history of depression, weight loss, chronic diarrhea, trauma, headache, fever, cough, breathlessness, rash, oral or genital ulcers, high-risk behavior, exposure to toxins, drugs, smoking, alcohol intake, etc.

Family history: There was no history of dementia, psychiatric illness, or any other illness in the family.

Medical history: The patient was diagnosed with hypertension 5 years ago and has been on antihypertensives since then. There were no other comorbid conditions such as diabetes, hypothyroidism, cardiac, respiratory, or kidney illness.

Personal history: The patient consumes a mixed diet. He had a normal appetite and bowel and bladder habits. Premorbid history, as reported by his wife, shows that the patient was a high-functioning person working as a schoolteacher.

■ Examination

Systemic Examination

Respiratory, cardiovascular, per abdomen, and musculoskeletal systems were normal. On examination, the patient was of normal built; weight: 65 kg; height: 165 cm; body mass index (BMI): 23.8 kg/m^2; pulse rate (PR): 82 bpm regular synchronous with other pulses; blood pressure (BP): 130/80 mm Hg, no postural drop; respiratory rate (RR): 20 breaths/min; no pallor, icterus, cyanosis, edema, lymphadenopathy, rash, neurocutaneous markers, ulcers, or signs of chronic toxin exposure.

Central Nervous System Examination

The patient was conscious, cooperative, and oriented to person but not to time and place. Rapport was established with the patient. The affect was normal. He had socially appropriate behavior and relatively normal insight. Mini-mental status examination score was 19/30. He was unaware of the date, month, hospital, and city name. He lost points for serial 7s, recalled two words, was able to remember one with the cue, had difficulty in comprehension, and could not draw intersecting pentagons **(Fig. 1)**.

His detailed mental status examination revealed the following:

Attention: Impaired digit span (digit forward four and digit backward three), trail making test A (TMT-A) was normal (<90 seconds), TMT-B (>3 minutes) was impaired, impaired serial subtraction. *Memory*: Anterograde memory—impaired learning [10/30 on three trials Kolkata Verbal Learning Test (KVLT)], impaired recall (3/10), mild improvement on delayed recognition, intact visual, remote and semantic memory. *Language assessment*: The spontaneous speech was laborious, hypophonic with mild initiation difficulty and word finding pauses, mildly impaired naming, normal semantic association tasks, unable to repeat multisyllabic words or phrases, intact comprehension and reading, able to write sentences but poorly formed letters and some spelling errors. His phonemic fluency was reduced, total F,A,S words 14 (normal > 36) and category fluency animals 11 (normal > 16). *Praxis*: Markedly apraxic, unable to copy meaningless

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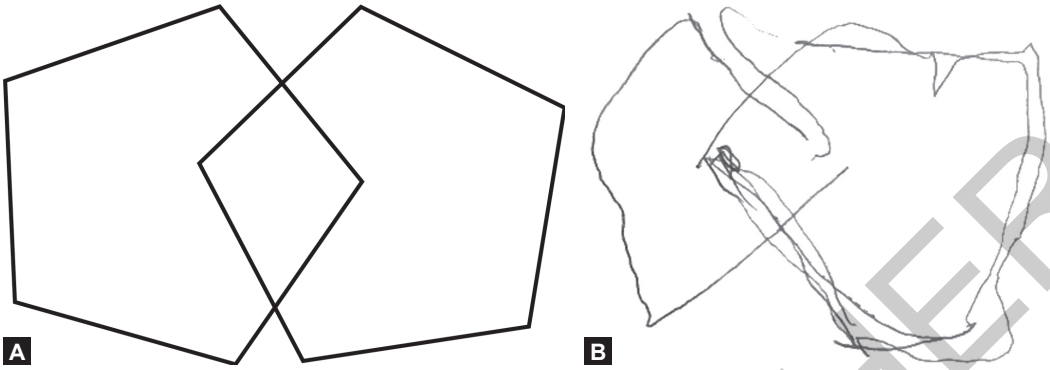

FIGS. 1A AND B: Copying intersecting pentagons. (A) Normal (B) Spatial distortions and errors in perspective.

gestures, especially with the right hand, able to copy meaningful gestures and improved with imitation, able to identify tools to complete a task, no orobuccal or eyelid apraxia. *Calculation*: Normal simple calculation with impaired complex verbal and written calculation. *R–L orientation*: Mildly impaired. *Finger identification*: Normal. *Visuospatial skills*: Normal line bisection and letter cancellation, impaired construction ability, unable to copy loops or cubes, poorly organized clock drawing, mild dressing apraxia, mildly impaired topographic orientation. *Visuoperceptual skills*: Able to match, but poor on copying line diagrams, able to identify unusual views of objects, poor on letter identification, dot counting, normal silhouette and real object identification, able to match dissimilar objects with the same function, and also able to match palm tree with pyramids. *Optic aphasia*: Absent. *Face recognition*: Able to identify famous faces. *Color perception*: Able to name, point, match, or sort colors and preserved conceptual color naming. *Simultagnosia*: Able to identify global letters made of small letters, normal cookie theft picture description. *Optic ataxia and oculomotor apraxia*: Able to reach for visually defined objects and shifting gaze to commands. *Visual orientation*: Able to judge the distance of two objects, no difficulty in dot position discrimination or number location. *Frontal lobe tests*: Normal graphic sequencing, motor Luria, mildly impaired proverb interpretation, normal go-no-go test, no environmental dependency, normal judgment and insight, normal Stroop test, able to identify emotions using pictures of facial affect (POFA) and primitive reflexes were absent except for palmomental reflex. *Cranial nerves*: Visual acuity was 6/9 in both eyes and 6/6 with pinhole. Fundus and visual fields by confrontation were normal. Direct and indirect light reflex was present. Extraocular movements were normal in all directions of gaze, and saccades were slow with normal pursuits and optokinetic nystagmus (OKN). Other cranial nerves were normal.

Motor examination: Normal bulk all over, increased tone in the right upper limb suggestive of rigidity, power was normal, deep tendon reflexes were normal, and bilateral plantar reflexes were flexor. There were no involuntary movements. *Sensory system*: All the Sensations were normal in all for limbs, including joint position and vibration. *Cerebellar system*: Finger-Nose test coordination, heel-knee shin test, and alternate supination pronation of forearm and hand were normal. *Gait*: There was no postural instability. However, he had difficulty starting or turning, hesitations while walking, mild problems in the tandem walk, and mild posturing of the right upper limb. There was no alien limb phenomenon. *Cortical sensations*: These were normal for two-point discrimination, astereognosis, graphesthesia, tactile localization, topognosis, and simultaneous bilateral stimulation.

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■ Summary of the Case

This 66-year-old man, with hypertension for 5 years, presents with an insidious onset, progressive cognitive decline in the form of inattention, episodic memory impairment, mild language problems, lack of empathy, mild apathy, visuospatial disorientation, dressing apraxia, and ideomotor apraxia. On examination, he had impaired attention, executive dysfunction, anterograde memory loss, effortful agrammatic language disturbances, ideomotor apraxia, acalculia, impaired visuospatial skills, apperceptive agnosia, mild frontal lobe deficits, and parkinsonism. The patient became dependent on family members for all his daily activities.

■ Symptom Analysis and Localization of the Case

In evaluating this patient, the goal is to determine the cognitive domain involved and use that information to assess the integrity of neural systems (structures involved) that can be affected by various systems (pathophysiology and etiology).

Forgetfulness (Memory)

A detailed clinical history from a reliable caregiver is the most helpful tool in evaluating a patient with forgetfulness. A valuable framework for analyzing memory complaints divides memory into several separate domains. Episodic memory is personally experienced events. It comprises anterograde (newly encountered information) or retrograde (past events) components and depends on the hippocampal–diencephalic system. Another critical system involves memory for word meaning and general knowledge (semantic memory), the key neural correlate being the left anterior temporal lobe. Working memory (or attention) is the capacity that allows us to retain information for a few seconds, and the neural substrate is the dorsolateral prefrontal cortex. Memory loss and learning impairment are out of proportion to other cognitive disturbances known as amnestic syndrome. Generally, both anterograde and retrograde memory loss occur in parallel, such as Alzheimer's disease (AD) or head injury, but dissociations occur. Hippocampal damage may cause relatively pure anterograde amnesia, particularly in herpes simplex encephalitis, focal temporal lobe tumors, or infarction. Confabulation in Korsakoff's syndrome might be grandiose or delusional but more often involves the misordering and fusion of real memories that are retrieved out of context. A transient amnesic syndrome with pronounced anterograde and variable retrograde amnesia is seen in transient global amnesia (TGA), while memory lacunes and repeated brief episodes of memory loss suggest transient epileptic amnesia. Based on history, it is possible to ascertain the involvement of either episodic memory or attention, as well structural localization within the brain. Pathophysiology and etiology should be determined after localization because these domains can be involved in a variety of conditions **(Flowchart 1)**.

Bedside tests to assess memory:

- Digit span backward
- Verbal learning test/story recall
- Visual memory
- Remote memory
- Semantic battery test
- Rey–Osterrieth figure test (nonverbal memory)

Wayfinding Difficulty (Visuospatial Ability)

Visuospatial processing relies upon networks that compose the "dorsal stream," which includes parietal areas specialized for processing motion and spatial relationships. This is also referred to as the "where pathway." Higher-order visual processing also relies on a "ventral stream," which

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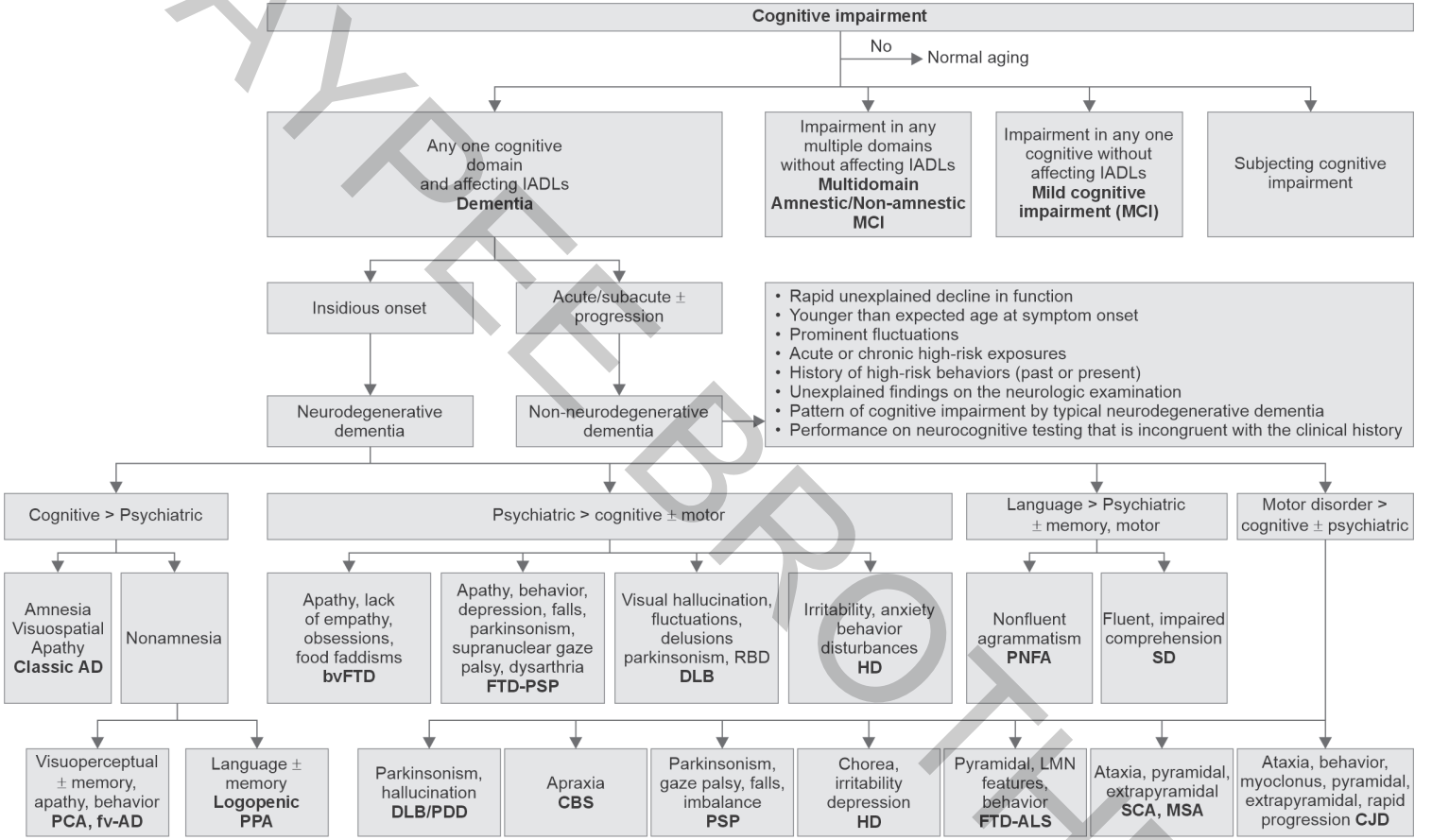

FLOWCHART 1: Algorithmic approach for identifying different types of dementia.

(AD: Alzheimer's disease; bvFTD: behavioral variant frontotemporal dementia; CBS: corticobasal syndrome; CJD: Creutzfeldt–Jakob disease; DLB: dementia with Lewy bodies; fv-AD: frontal variant Alzheimer's disease; FTD-ALS: frontotemporal dementia with amyotrophic lateral sclerosis; FTD-PSP: frontotemporal dementia with progressive supranuclear palsy; HD: Huntington's disease; IADLs: instrumental activities of daily living; LMN: lower motor neuron; MSA: multiple system atrophy; PCA: posterior cortical atrophy; PDD: Parkinson's disease dementia; PNFA: progressive nonfluent aphasia; PPA: primary progressive aphasia; PSP: progressive supranuclear palsy; RBD: rapid eye movement sleep behavior disorder; SCA: spinocerebellar ataxia; SCI: subjective cognitive impairment; SD: semantic dementia)

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includes inferior temporal areas specialized for processing visual features of an object, face, or scene. Lesions that disrupt right parietal regions and their networks may produce impaired visuospatial perception, such as the inability to appreciate the location, orientation, or motion and clinical syndrome of hemineglect. Wayfinding difficulty can also be due to frontal lobe involvement (intention resulting in aimless wandering). Ictal wandering is almost paroxysmal and accompanied by altered sensorium or confusional state or can be secondary to dissociative fugue (sudden-onset wandering purposefully away from home and failure to remember important aspects of their identities such as their name, address, occupation—lasts for a period of hours to days—recovery is spontaneous).

Bedside tests for visuospatial ability:

- Figure copying
- Drawing to command (clock drawing, daisy in a flower pot)
- Complex figure test [modified Taylor complex figure test (MTCF)]
- Line bisection test
- Target cancellation test
- Visual object and space perception (VOSP)

Dressing Apraxia (Perceptual-motor)

Impairment in dressing oneself, so-called dressing apraxia, usually reflects a complex visuospatial deficit rather than an actual apraxic problem. Putting on a shirt requires alignment of body parts and mental rotation, which depend on the nondominant hemisphere (posterior parietal lobe). Patients with frontal lobe involvement may have a different disorder of dressing, which consists of mis-sequencing of garments, such as wearing underpants on top of trousers, or perseveration, such as wearing multiple shirts, one over the other.

Test to assess dressing apraxia:

- Hand over a shirt (that has been turned upside down with one sleeve inside out) and observe for errors and smoothness of putting on the dress.

Behavior Disturbances (Social Cognition)

Social cognition refers to cognitive processes, empathy [orbitofrontal cortex (OFC) and and anterior cingulate cortex (ACC)—mainly right frontotemporal network], theory of mind, and emotion processing [OFC, insula] required to perceive, interpret, and integrate social stimuli with given contextual clues for successful social interactions. Changes in personality and behavior consist of a lack of concern about oneself and others, often with childish egocentric behavior, and lack of inhibition (a ventromedial portion of the OFC), with a tendency to react impulsively (OFC). Patients may become rigid and have difficulty appreciating humor or sarcasm. Other behavioral disturbances include lack of spontaneity [apathy (ACC)], reduced initiation of conversation and activity, self-care, changes in eating or sexual behavior, stereotyped patterns of behavior, imitation and utilization behavior (OFC), delusions, and hallucinations.

Tests and questionnaires to assess social cognition and frontal lobes:

- Frontal Assessment Battery (FAB)
- Neuropsychiatric Inventory (NPI)
- Frontal Systems Behavior Scale (FrSBe)
- Interpersonal Reactivity Index (IRI) for empathy
- POFA for emotion recognition
- Reading the Mind in the Eyes test for Theory of Mind (ToM)
- Awareness of Social Inference Test (TASIT)

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Speaking Difficulty

Speech impairments may result from disrupted lower-order neural and muscular mechanisms for implementing sound production (nonaphasic speech impairments: dysphonia or dysarthria) or central language processing (aphasia). Dysarthria is the abnormal articulation of spoken language. There are at least six types of dysarthria. (1) In spastic dysarthria, speech is typically strained, slow, effortful, and monotonous. Bilateral upper motor neuron lesions compromising the corticobulbar tracts are the cause. (2) In flaccid dysarthria, speech is soft, low pitched, thick, slurred, nasal quality, and hesitant with failure of correct enunciation, and all sounds and syllables may be indistinct. Causes include myopathies, myasthenia gravis, and lower motor neuron disease (MND). (3) Ataxic dysarthria causes a lack of smooth coordination of the tongue, lips, pharynx, and diaphragm. Speech is slow, slurred, irregular, labored, and jerky. Words are pronounced with irregular force and speed, with involuntary variations in loudness and pitch, leading to an explosive quality. There are unintentional pauses, which cause words and syllables to be erratically broken. Excessive separation of syllables and skipped sounds in words produce a disconnected, faltering staccato articulation (scanning speech). Causes include cerebellar strokes, tumors, multiple sclerosis, and cerebellar degeneration. (4) Hypokinetic dysarthria, the typical speech pattern in Parkinson's disease, is notable for decreased and monotonous loudness and pitch, rapid rate, and occasional consonant errors. (5) Hyperkinetic dysarthria is characterized by marked variation in rate, loudness, and timing, with distortion of vowels, harsh voice quality, and occasional sudden stoppages of speech. Causes include Huntington's disease and dystonia musculorum deformans. (6) Mixed dysarthria involves a combination of the other five types. One common cause is amyotrophic lateral sclerosis.

Disturbances in language may manifest as difficulty speaking or understanding spoken language, but reading and writing are also usually impacted. A complex neurocognitive network, usually located in the left hemisphere, subserves the capacity for human language. The language areas comprise the posterior inferior frontal gyrus (area 44—Broca's area) that has adjacent motor neurons subserving the mouth and larynx and controls the output of spoken language and syntax. The posterior two-thirds of the superior temporal gyrus (area 22—Wernicke's area) receives information from the auditory cortex and accesses a network to assign word meanings.

The angular gyrus (in the inferior parietal lobe) subserves the perception of written language in association with the graphomotor output regions in the frontal lobe and insula, which is integral to normal articulation. Finally, the arcuate fasciculus includes perisylvian areas which connect Wernicke's and Broca's areas and is responsible for repetition. In addition, subcortical structures such as the thalamus, basal ganglia, or adjoining white matter can result in aphasia. The presentation of aphasia varies according to underlying etiology—acute or subacute presentation with relatively fixed deficits is likely due to structural disease. These include ischemic stroke (most common), hemorrhagic stroke, neoplasm, abscess, encephalitis, and other infections and traumatic brain injury. Transient episodes of aphasia may occur with transient cerebral ischemia (TIA), migraine, and seizures (either an ictal or a postictal phenomenon). Progressive aphasia can be a manifestation of neurodegenerative diseases such as primary progressive aphasias.

Tests to assess language:

- Western Aphasia Battery
- Semantic association task
- Boston Naming Test
- Frenchay Aphasia Screening Test (FAST)

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Difficulty Handling Tools (Apraxia)

The inability to perform a learned motor act despite intact sensory and motor function is termed apraxia. It should be distinguished from other causes of impaired movement secondary to weakness, spasticity, ataxia, extrapyramidal disturbances, impaired sensory deficits, hemispatial neglect, spatial or object agnosia, or abnormal movements such as tremor, myoclonus, or dystonic posturing. Apraxia typically presents difficulty dressing, feeding, or bathing, not explained by clear motor deficits. It is crucial to describe apraxia by region and to provide a description of impaired performance, recording both spatial and sequencing errors on several different types of tasks. Common praxis errors include using the wrong object or body part to perform a task. Many patients who cannot perform motor tasks to command can do so spontaneously or by imitation. The ability to perform learned motor movements (ideomotor praxis) can be assessed by asking the patient to perform increasingly complex motor tasks. The patient may be asked to demonstrate how to use an object (such as a comb, hammer, or fork) both with and without the object in their hands. Asking the patient to perform a step-by-step series of coordinated tasks, such as "Take this piece of paper, fold it in half, and place it in the envelope," can be used to assess ideational praxis (the ability to carry out a sequential set of actions toward a final goal).

Localization of Apraxia

Localization for each type of apraxia is ideomotor apraxia—left premotor/supplementary motor area and left inferior parietal lobe; ideational apraxia—posterior dominant region (left parietotemporal, frontal and basal ganglia); limb kinetic apraxia—contralateral frontal lobe; orofacial apraxia—left parietal, inferior frontal, deep frontal white matter, insula, and basal ganglia; eyelid opening apraxia—right hemisphere or bilateral hemispheric lesions and basal ganglia diseases; apraxia of speech—left inferior frontal, insula; gait apraxia—bilateral medial frontal lesions.

Test to assess apraxia: Tulia apraxia test

■ Differential Diagnosis

After symptom analysis and finalizing the structure involved, differentials should be considered first based on "pathophysiology" depending on history, duration of symptoms, associated systemic findings, and examination. Pathophysiology could be vascular, infective, inflammatory, trauma, toxins, autoimmune, metabolic, iatrogenic, nutritional, endocrine, structural, systemic diseases, neurodegenerative, and inherited neurometabolic disorders. The etiology can be accordingly delineated based on the pathophysiology and specific symptoms **(Table 1)**.

ALGORITHMIC APPROACH TO COGNITIVE IMPAIRMENT

Neurodegenerative dementias comprise a group of distinct neuropsychological disorders that reflect the topographic selectivity of the underlying pathology. It is essential to correctly diagnose the type of dementia as management and long-term outcomes differ for each subtype of dementia. Thus, salient features for each dementia subtype should help in characterization. Before concluding an irreversible cause (neurodegenerative) of cognitive impairment, reversible causes should be ruled out. Dementia should be distinguished from symptoms of memory loss or personality and behavioral changes that are nonprogressive or provoked by underlying systemic illness, medication, psychiatric disorder, or structural pathology. Therefore, strategies are required to recognize potentially treatable causes of dementia (non-neurodegenerative) and differentiate them from primary dementias (neurodegenerative dementias). This can be accomplished by

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TABLE 1: List of non-neurodegenerative dementia—potentially reversible causes.

Vascular causes • CADASIL • CAA • Primary angiitis of CNS • Secondary CNS vasculitis • Vascular cognitive impairment	*Iatrogenic/medications* Steroids, BZDs, anticholinergics, first-generation antihistaminic, valproate, antipsychotics, antidepressants	*Inherited metabolic disorders* • Wilson's disease • NBIA spectrum • Mitochondrial disorders • X-linked adrenoleukodystrophy • Metachromatic leukodystrophy • Sialidosis types 1 and 2 • Niemann–Pick disease type C • Gaucher's disease type 3 • Adult neuronal ceroid lipofuscinosis • Homocystinuria • Hartnup's disease • Propionic acidemias • Ornithine transcarbamylase deficiency • Arginase deficiency • Fahr's disease • Kufs disease • Cerebrotendinous xanthomatosis • Adult polyglucosan disease • Aceruloplasminemia • Porphyrias • Lesch–Nyhan syndrome • Refsum's disease • ALPS
Infections • CNS tuberculosis • HIV-associated neurocognitive disorder • PML • Neurosyphilis • Herpes encephalitis • Whipple's disease • SSPE • Lyme's disease	*Nutritional* • Vitamin B12 deficiency • Thiamine deficiency (Wernicke's encephalopathy) • Vitamin E deficiency • Niacin deficiency • Vitamin D deficiency	
Inflammatory/demyelination • Multiple sclerosis • Neurosarcoidosis • Neuro-Behçet's disease • Neuro-Sjögren's syndrome • CNS lupus	*Endocrine disorders* • Hypo- and hyperthyroidism • Diabetes • Hypo- and hyperparathyroidism • Addison's disease • Cushing's disease	
Toxins • Alcohol-related dementia • Heavy metals (Al, Bi, lead, arsenic, mercury) • CO poisoning	*Systemic diseases* • Pulmonary disease (COPD) • Hepatic insufficiency • Renal failure • Cardiac dysfunction	
Trauma • Chronic traumatic encephalopathy	*Structural brain lesions* • Subdural hematoma • Extradural hematoma • Tumors (space-occupying lesions) • Metastasis	
Autoimmune • Autoimmune dementia/encephalitis • Hashimoto's encephalitis • Paraneoplastic encephalopathy	*Other causes* • Depression • Sleep apnea syndrome • Epilepsy • Frontotemporal brain sagging syndrome • Encephalitis sequelae • Systemic infections (UTI)	
Metabolic • Hypo- and hypernatremia • Hypo- and hypercalcemia • Hypo- and hyperglycemia		

(ALPS: autoimmune lymphoproliferative syndrome; BZDs: benzodiazepines; CAA: cerebral amyloid angiopathy; CADASIL: cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy; CNS: central nervous system; COPD: chronic obstructive pulmonary disease; HIV: human immunodeficiency virus; NBIA: neurodegeneration with brain iron accumulation; PML: progressive multifocal leukoencephalopathy; SSPE: subacute sclerosing panencephalitis; UTI: urinary tract infection)

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implementing a universal approach to dementia evaluation that focuses on active monitoring for atypical features in history, examination, and investigations that may indicate a reversible cause of dementia **(Flowchart 1)**.

WHAT IS DEMENTIA?

The term "dementia," also known as neurocognitive disorder (NCD) [according to Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria], is an umbrella term, not one specific disease but rather a group of symptoms that occur as a result of a disease. It is characterized by a decline in cognition involving one or more cognitive domains (learning and memory, language, executive function, complex attention, perceptual-motor, and social cognition). The deficits must represent a decline from the previous level of function and are significant enough to interfere with daily living activities and independence. Thus, it severely impacts memory, behavior, thinking, and social abilities to interfere with one's activities of daily living and social autonomy. Cognitive change is often noted before the onset of frank dementia. The term mild cognitive impairment (MCI) is used when objective decrements exceed those expected from usual aging, yet do not impair the activities of daily living.

Worldwide, around 55 million people are living with dementia, and there are nearly 10 million new cases every year, with over 60% living in low- and middle-income countries. As the proportion of older people in the population is increasing in every country, this number is expected to rise to 78 million in 2030 and 139 million in 2050.

In 2013, dementia was categorized as an NCD in DSM-5. NCD is further subdivided into minor NCD and major NCD. The DSM-5 details six cognitive domains that may affect both minor and major NCDs. These cognitive domains (and their associated warning signs) include:

A. *Complex attention*: It involves sustained attention, divided attention, selective attention, and information-processing speed. [Signs: Patient has difficulty in environments with multiple stimuli and has difficulty holding new information in mind (recalling phone numbers or address just given)].
B. *Executive function*: It involves planning, decision-making, working memory, responding to feedback, error correction, overriding habits, and mental flexibility. (Signs: Patient is unable to perform both familiar and complex tasks. He needs to rely on others to plan instrumental activities of daily living or make decisions. He has problems with abstract thinking and displays loss of initiative as well as poor judgment).
C. *Learning and memory*: It involves immediate memory, recent memory (free recall, cued recall, and recognition memory), and long-term memory. (Signs: Patient repeats self in conversation, often with the same conversation. He cannot keep track of a short list of items when shopping or of plans for the day. He requires frequent reminders to orient the task at hand, confusion about time and place, and repetitive behavior).
D. *Language*: It involves expressive language (spontaneous speech, naming, fluency, grammar, repetition) and receptive language. (Signs: Patient has significant difficulties with expressive or receptive language. He often uses general terms such as "that thing" and "you know what I mean." In severe impairment, he may not recall names of closer friends and family).
E. *Perceptual-motor-visual perception, praxis*: It involves picking up the telephone, handwriting, and using a fork/spoon. [Signs: Patient has significant difficulties with previously familiar activities (using tools or driving a motor vehicle) and navigating in familiar environments].
F. *Social cognition*: It involves recognition of emotions and behavioral regulation, social appropriateness in dress, grooming, and topics of conversation. [Signs: Patient may have changes in behavior (shows insensitivity to social standards or make decisions without regard

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