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3

ICP-IAN 100 Questions in Neurological Practice

Office Neurology, Neuro Muscular and Functional Neurology



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SECTION 1

Office Neurology

Section Editors: Lakshmi Narasimhan Ranganathan, Biman Kanti Ray

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Q1. How and when to start medications for dementia?

Ans. Dementia is defined as an acquired disorder characterized by a progressive decline in cognition that affects at least one or more of the six defined cognitive domains (language, learning and memory, executive function, perceptual-motor function, complex attention, and social cognition). Alzheimer's disease (AD) is the most common cause of dementia and most medications for dementia have been predominantly studied in patients with AD. The two major group of drugs for dementia include central cholinesterase inhibitors (ChEIs) which increase the levels of cerebral acetylcholine (donepezil, rivastigmine, and galantamine) and a non-competitive NMDA receptor antagonist (memantine). Studies have shown that these agents delay disease progression, enhance cognitive functions and improve activities of daily living. At present the recommendation is to offer medications to all patients with dementia at the point of initial diagnosis **(Flowchart 1)**. Medical therapy of dementia not only includes administration of the dementia drugs but also addresses the coexistent neuropsychiatric issues and risk factor control **(Flowchart 2)**. Choosing the right agent depends on the clinical profile of the patient (disease severity), pharmacological properties of the agent and the adverse effect profile **(Table 1)**. The EFNS-ENS/EAN guideline published in 2015 favors the combination of memantine with ChEI over monotherapy in patients with moderate-to-severe dementia.

Currently 2 Food and Drug Administration (FDA)-approved amyloid targeted monoclonal antibodies are available (lecanemab and donanemab) and their use is recommended in patients with mild disease (MMSE >22, MoCA >17 or CDR 0.5 to 1) with documented amyloid pathology (amyloid PET scan or lumbar puncture).[1-3]

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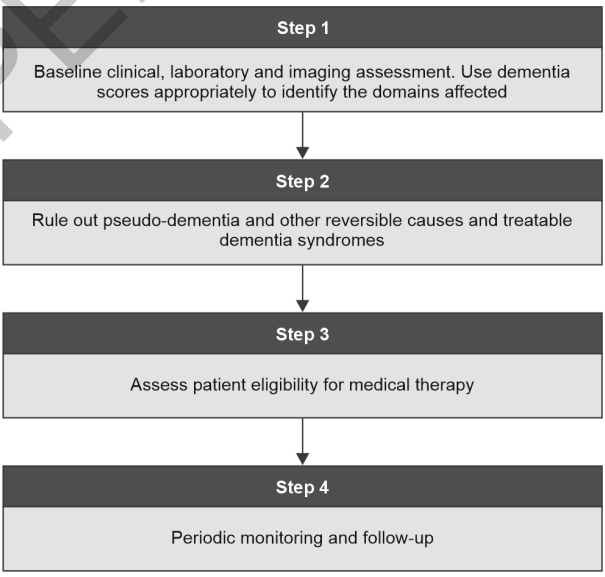
TABLE 1: Anti-dementia drugs, their dosing and application.

Agent	Dosing	Adverse effect	Clinical application
Donepezil	5–10 mg once daily oral medication	• Gastritis, diarrhea, and GI dysfunction • Galantamine and rivastigmine to be avoided in the setting of renal/liver disease	Mild-to-moderate AD
Galantamine	• 4 mg twice daily to 12 mg twice daily oral medication (immediate release) • 8 mg once daily to 24 mg once daily oral medication (extended release)		
Rivastigmine	• 1.5 mg twice daily to 6 mg twice daily oral medication • Transdermal patch • 4.6 mg/24 h • 9.5 mg/24 h • 13.3 mg/24 h		
Memantine	• 5 mg once daily to 10 mg twice daily oral medication (immediate release) • 7 mg once daily to 28 mg once daily oral medication (extended release)	Confusion, dizziness, drowsiness, and headache	Moderate-to-severe AD

Classification of dementia:
Mild: MMSE 19–26; MoCA 12–16; CDR 1
Moderate: MMSE 10–18; MoCA 4–11; CDR 2
Severe: MMSE <10; MoCA <4; CDR 3
(AD: Alzheimer's disease; CDR: clinical dementia rating score; MMSE: mini-mental scale examination, MoCA: Montreal cognitive assessment)



FLOWCHART 1: Approach to management of a patient with dementia.

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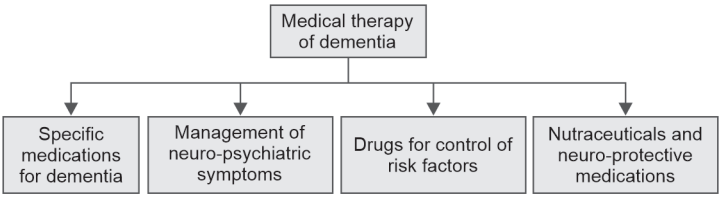

FLOWCHART 2: Medical management of a patient with dementia.

Q2. Antipsychotic for neurology practice—how to choose the right agent?

Ans. The term "organic psychosis" is used to refer to hallucinations and psychotic symptoms in the presence of an underlying pathology. Acute organic psychosis is commonly observed in individuals with metabolic derangements, febrile infection/sepsis, prolonged hospitalization/ICU stay as well as in individuals with a primary neurological illness such as stroke, neurodegenerative disorders (like Parkinson's disease and frontotemporal dementia). As neurologists we commonly encounter these patients and a basic understanding of the various antipsychotic agents is mandatory to choose the right agent and improve patient care.

Dopamine is the key neurotransmitter concerned with hallucinations and psychosis and dopaminergic dysfunction involving the meso-cortical and meso-limbic pathways is believed to play the major role (dopamine hypothesis of schizophrenia). Almost all antipsychotics work by reducing the dopaminergic activity either directly or indirectly and based on their mechanism of actions are classified as typical (1st generation) or atypical (2nd generation) antipsychotics **(Flowchart 3)**. The decision to choose the agent is based on the clinical profile of the patient and adverse drug profile of the agent. Accordingly certain medications are preferred or deferred in specific scenarios as depicted in **Table 2**.[4-7]

Q3. How to approach a patient with suspected cerebrospinal fluid (CSF) rhinorrhea?

Ans. Cerebrospinal fluid (CSF) rhinorrhea refers to abnormal leak of the CSF through the nose as result of cranial CSF leaks due to a bony defect. The major causes for CSF rhinorrhea include craniofacial trauma and cranial/endonasal surgeries. Spontaneous leak without preceding trauma or surgery can be seen in patients with increased intracranial pressure such as idiopathic intracranial hypertension (IIH), obstructive sleep apnea (OSA) and intracranial tumors. Patients typically complain of a clear watery nasal discharge. The discharge is typically clear and not sticky and has sweet/salty taste when associated with postnasal drip. Recurrent meningitis and orthostatic headache are other scenarios where CSF rhinorrhea needs to be suspected.

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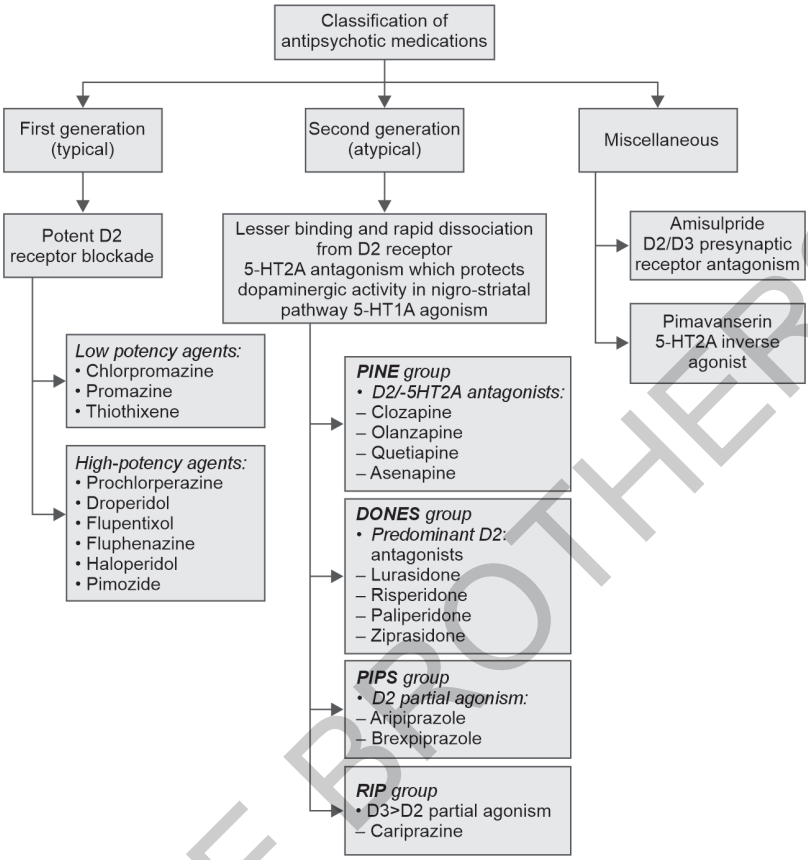

FLOWCHART 3: Classification of antipsychotic agents.

TABLE 2: Suggested choice of antipsychotic based on patient's clinical profile.

Clinical profile	Preferred agents	Agents to be avoided
Risk of acute extrapyramidal syndrome	Quetiapine, olanzapine, and aripiprazole	Typical antipsychotics
Dyslipidemia, metabolic syndrome, and hyperglycemia	Lurasidone, aripiprazole, and amisulpride	Quetiapine and olanzapine
Risk of QT prolongation	Lurasidone and aripiprazole	Quetiapine, olanzapine, and clozapine
Sedation to be minimized or avoided	Paliperidone, aripiprazole, and amisulpride	Quetiapine and olanzapine
Cytopenia and risk of bone marrow suppression		Clozapine
Coexistent hyperkinetic movement disorder	Risperidone and haloperidol	
Risk of akathisia	Quetiapine, olanzapine, and aripiprazole	Typical antipsychotics
Coexistent parkinsonism	Pimavanserin, quetiapine, olanzapine, and clozapine	Typical antipsychotics

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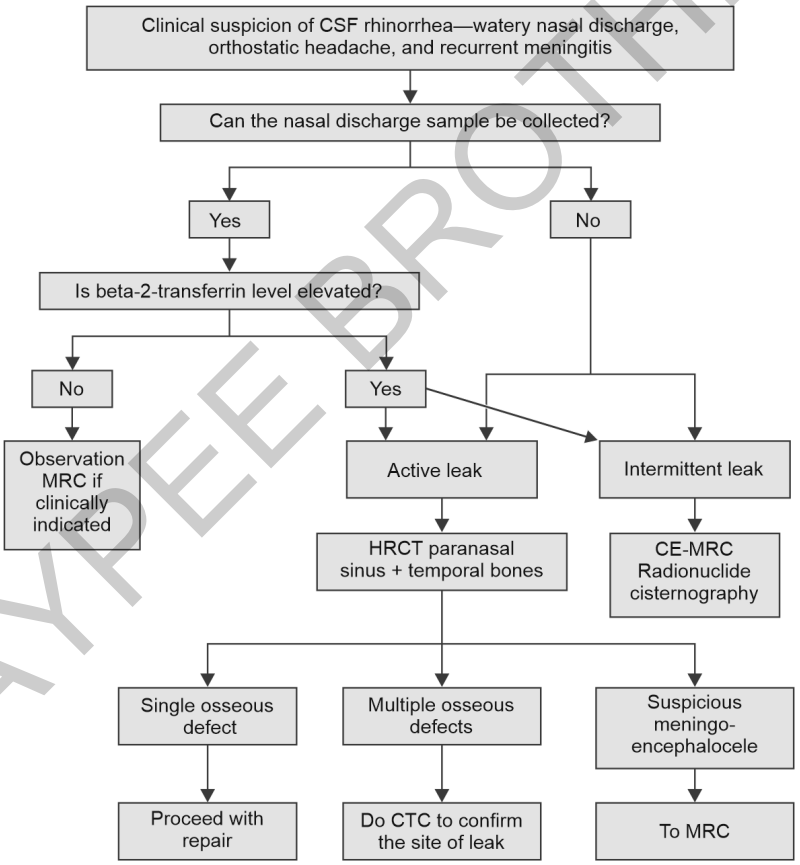
Once suspected the first preferred investigation is to collect the fluid and assess for beta-2-transferrin by immunohistochemical assays. Beta-2-transferrin in present in CSF, perilymph, and vitreous humor. The quantity of perilymph is too less while the contamination of nasal secretions with vitreous humor is extremely rare making the presence of beta-2-transferrin in nasal discharge highly sensitive for CSF rhinorrhea (100%) with specificity ranging between 70 and 95%.

Once CSF rhinorrhea is established the next step is to look for the location of leak and plan surgical repair accordingly. Several imaging modalities are used to identify the leak and their advantages and disadvantages are summarized in **Table 3**. An algorithmic approach to suspected CSF rhinorrhea is provided in **Flowchart 4**.[8-11]



FLOWCHART 4: Algorithmic approach to a patient with suspected cerebrospinal fluid (CSF) leak.

(CE-MRC: contrast-enhanced MR cisternography; CTC: CT cisternography; MRC: MR cisternography)

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TABLE 3: Various imaging modalities used in the assessment of cerebrospinal fluid (CSF) rhinorrhea.

Modality	Technique	Advantages	Disadvantages	Accuracy
Hight resolution CT (HRCT) of paranasal sinuses including temporal bones	Isotropic, volumetric, thin submillimeter sections with multiplanar reformations	• Excellent delineation of bony anatomy • Most useful in patients with active CSF leaks • Assists intraoperative navigation via endoscopy	• Failure to ascertain concomitant dural defect in the setting of multiple bony defects • Failure to differentiate mucus secretions from CSF collections in paranasal sinuses	84–95% sensitivity 57–100% specificity
MR cisternography (MRC)	Steady state free precision (SSFP) technique	• Demonstrates CSF track between the subarachnoid space and extracranial space • Demonstrates indirect signs of CSF leak and signs of increased intracranial pressure • Can delineate contents herniating through bony defect	Poor visualization of bony window (to always interpret along with HRCT findings)	94% sensitivity
CT cisternography (CTC)	Image acquisition following intrathecal injection of non-ionic iodinated contrast and placing the patient prone in Trendelenburg position for 5 minutes	Primary role in evaluating patients with multiple surgeries/fractures/bony defects to confirm the site of leak	• Useful only in active CSF leaks • Invasive nature • Increased radiation exposure • Complications related to intrathecal administration	85–92% sensitivity (in active CSF leaks) 40% sensitivity (in intermittent/ inactive CSF leaks)
Contrast-enhanced MR cisternography (CE-MRC)	Image acquisition following intrathecal injection of gadolinium-based contrast agent and image acquisition after about 1–2 hours.	• Clear delineation of site of CSF leak • Can show site of leak in inactive or intermittent CSF leaks • Depiction of meningoceles	• Poor visualization of bony window • Invasive nature • Complications related to intrathecal administration • Lack of FDA approval for intrathecal administration of the gadolinium contrast agents	92–100% sensitivity (in active CSF leaks) 70% sensitivity (in intermittent/ inactive CSF leaks) 80% specificity
Radionuclide cisternography	Image acquisition after intrathecal administration of Technetium—99 or Indium—111	Especially useful in intermittent CSF leaks where other modalities have failed to help	• Low accuracy and suboptimal localization • Invasive nature • Complications related to intrathecal administration • Complications related to exposure to radioactive agents	• Accuracy 70% in active leaks • Accuracy <28% in inactive leaks

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Q4. How to evaluate for organic causes in a patient with new onset psychiatric illness?

Ans. In patients presenting with an acute or new onset neuropsychiatric dysfunction (such as psychosis/hallucinations, mood fluctuations, behavior changes, depression, and suicidal thoughts) investigations to identify underlying (organic) causes are warranted. It is not possible to do every investigation for all patients presenting with neuropsychiatric symptoms. Still a basic understanding of the psychiatric manifestations of neurological and systemic disorders helps in clinical decision-making. One major difference between the approach of a traditional neurologist and a traditional psychiatrist is that neurological illness has a one-to-one relationship between the symptomatology, pathology, and region of the nervous system which is lacking in most psychiatric illnesses.

Clinical features which suggest an underlying medical disorder in a new onset psychiatric illness include abrupt onset of symptoms, presentations in extremes of age, prominent visual hallucinations, associated neurological features such as gait dysfunction, seizures, or extrapyramidal symptoms, a clinical phenotype not fixing into a defined psychiatric syndrome, fluctuating cognition and behavior (may suggest delirium), cognitive impairment disproportionate and atypical for the psychiatric illness, fever, progressive weight loss, and high-risk behavior (substance abuse). Certain neurological disorders such as central nervous system (CNS) vasculitis and autoimmune encephalitis [anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis] can have isolated psychiatric symptoms and require a high degree of clinical suspicion for further evaluation.

Some common neurological and systemic disorders and their psychiatric manifestations are summarized in **Table 4**. Over the last few years, patients with non-convulsive status epilepticus masquerading as delirium and new onset psychiatric illness have been increasingly reported. Also, the clinical spectrum of immune encephalitis (autoimmune and paraneoplastic encephalitis) is being increasingly widened to include patients with isolated psychiatric manifestations. The spectrum of neuropsychiatric manifestations reported in NMDAR encephalitis is summarized in **Box 1**.[12-16]

Q5. Discuss the management of sudomotor disorders.

Ans. Sudomotor function refers to sweat production and release which plays a key role in thermoregulation. Sweat gland is innervated by thin unmyelinated Type-C sympathetic fibers which utilize acetylcholine as the neurotransmitter. The central control for sweat production is regulated at the pre-optic anterior hypothalamus and is influenced by cortical centers and several external/internal factors. Sudomotor dysfunction is commonly encountered in small fiber neuropathies. However, diseases affecting any part of the neuraxis can lead to sudomotor dysfunction **(Flowchart 5)**.

The diagnosis of sudomotor dysfunction is complicated by the failure to demonstrate straightforward clinical findings. In most cases sudomotor

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TABLE 4: Neuropsychiatric manifestations of common neurological and medical disorders.

Disorder		Psychiatric manifestations
Traumatic brain injury		• Depression and psychosis • Behavior changes, impulsivity, and anti-social personality • Mood disorders, apathy • Substance abuse • Anhedonia, poor sleep and appetite, and chronic fatigue • Anxiety disorder (PTSD, GAD)
Vascular diseases of the brain Infarct, hemorrhage		• Cognitive impairment • Depression and dementia • Pseudo-bulbar affect • Anxiety disorder • Amotivation and impaired judgement
Neurodegenerative disorders	PD	• Depression and anxiety • Psychosis and hallucinations • Impulse control disorder
	Dementia syndromes	• Depression, apathy, and anxiety • Psychosis and hallucinations • Behavior changes, impulsivity, and anti-social personality • Anhedonia
Multiple sclerosis		• Demoralization and depression • Cognitive impairment and chronic fatigue • IEED • Pseudobulbar affect
Epilepsy		• Cognitive impairment • Depression, anxiety, and mania • Psychosis and hallucinations • Suicidal ideation • Forced normalization
CNS infection/inflammation Chronic meningitis Viral encephalitis CNS vasculitis Immune mediated encephalitis HIV/AIDS and neurosyphilis Prion diseases SREAT		• Delirium • Psychosis and hallucinations • Behavior changes, impulsivity, and anti-social personality • Anhedonia, poor sleep and appetite, and chronic fatigue • Cognitive impairment

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Disorder		Psychiatric manifestations
Metabolic disorders Dyselectrolytemia Hypothyroidism Renal failure/ liver failure Acute intermittent porphyria Wilson's disease Hyperglycemia CO_2 narcosis		• Delirium • Depression and mania • Behavior changes • Psychosis and hallucinations • Apathy and anhedonia
Drugs/toxins	Medications (steroids, sympathomimetics, CNS stimulants, anti-seizure medications, and dopaminergic agonists)	• Psychosis and hallucinations • Mania • Suicidal ideation • Delirium
	Alcohol intoxication	• Psychosis • Behavior changes • Delirium tremens • Addiction and withdrawal features
	Recreational agents (cannabis, cocaine, marijuana, LSD, and hallucinogens)	• Psychosis and hallucinations • Mania • Suicidal ideation • Delirium • Depression • Anti-social behavior
	Heavy metal toxicity (lead, mercury, and arsenic)	• Psychosis and hallucinations • Mania • Delirium

(AIDS: acquired immunodeficiency syndrome; CNS: central nervous system; GAD: generalized anxiety disorder; HIV: human immunodeficiency virus; IEED: involuntary emotional expression disorder; LSD: lysergic acid diethylamide; PD: Parkinson's disease; PTSD: post-traumatic stress disorder; SREAT: steroid-responsive encephalopathy associated with autoimmune thyroiditis)

BOX 1: Diagnostic clues and spectrum of clinical presentation of anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis.

- *S*leep dysfunction
- *E*xcitement, disinhibition, or manic behavior alternating with depressive behavior
- *A*ggression and/or agitation
- *R*apid onset
- *C*hildren and young adult predominant involvement
- *H*istory of no prior psychiatric disease
- *F*luctuating catatonia
- *N*egative and positive symptoms at presentation
- *M*emory impairment
- *D*ecrease in verbal output/mutism
- *A*ntipsychotic intolerance
- *R*igidity, hyperthermia, and coma similar to neuroleptic malignant syndrome
- Antibody in serum or CSF

Mnemonic—SEARCH for NMDAR antibody

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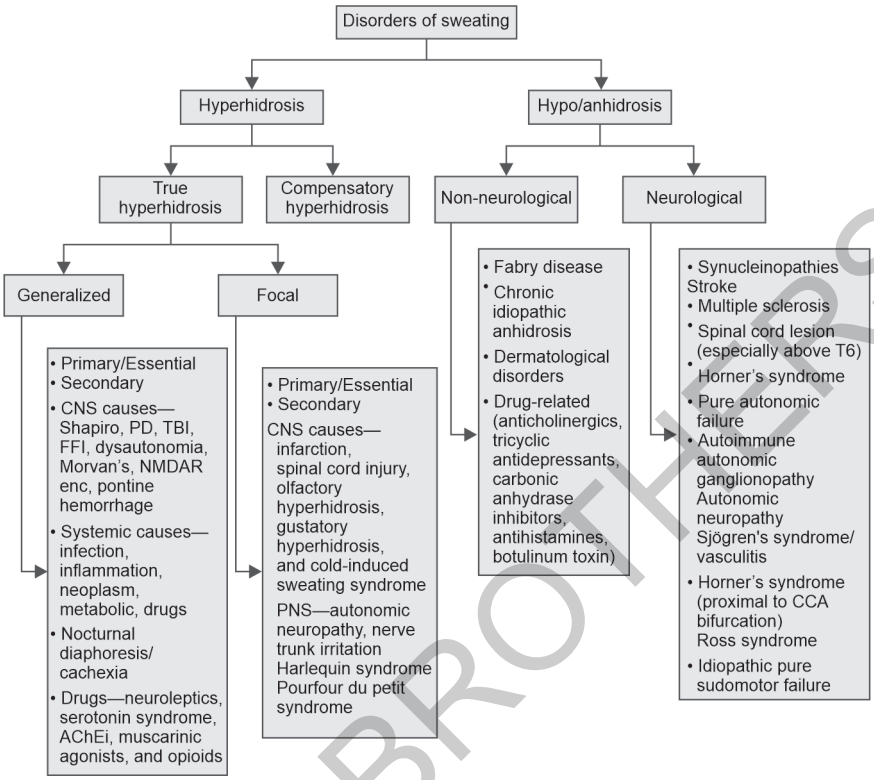
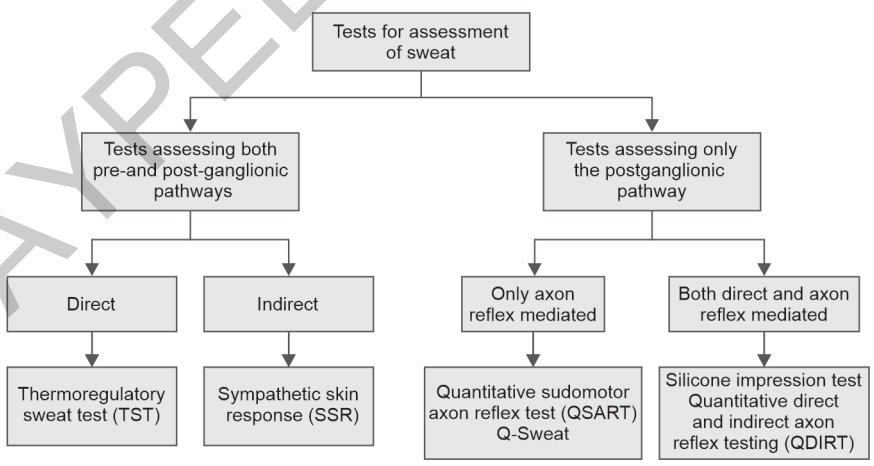


FLOWCHART 5: Classification of sudomotor disorders.

(AChEi: acetylcholinesterase inhibitors; CCA: common carotid artery; FFI: fatal familial insomnia; PD: Parkinson's disease; TBI: traumatic brain injury)



FLOWCHART 6: Tests for assessment of sweating.

dysfunction is a part of the neurological illness. Specialized demonstration of sudomotor function (or dysfunction) can be achieved by the use of specialized diagnostic modalities **(Flowchart 6 and Table 5)**.

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TABLE 5: Summary of the various diagnostic modalities for assessment of sweating.

	TST	SSR	QSART	Silicone impression	QDIRT
Stimulus	Whole body heating	Electrical current delivered to a peripheral sensory nerve	Iontophoresis of acetylcholine	Iontophoresis of pilocarpine or acetylcholine	Iontophoresis of acetylcholine
Response	Anatomic distribution of sweating over the anterior body surface	Change in electrodermal potential over the palms or soles	Latency, magnitude, and duration of sudomotor output in the four limb sites	Size and number of individual sweat droplets over a defined area of skin	• Similar to QSART • No requirement of sweat capsule • Sweat production photographed after application of dye–alizarin red
Site of lesion detection	Pre and post ganglionic	Pre and post ganglionic	Post ganglionic axonal only	Post ganglionic direct and axonal	Post ganglionic direct and axonal
Sudomotor assessment	Direct	Indirect	Direct	Direct	Direct
Need for specialized equipment	Yes	No	Yes	Yes	Yes
Objectivity	Poor	Good	Good	Good	Good
Standardized procedure	Yes	Yes	Yes	No	No
Time consumption	60–120 minutes	5 minutes	15–20 minutes	15 minutes	15 minutes
Technical interference	Low (manual calculation)	Very high	Low (correct marking)	High (proper placement)	Very low
Repeatability/ reproducibility	Yes	Poor (habituate)	Yes	Yes	Yes
Sampling sites	Excellent	Limited	Limited	Limited –may be better	Limited –may be better

(QDIRT: quantitative direct and indirect axon reflex testing; QSART: quantitative sudomotor axon reflex test; SSR: sympathetic skin response; TST: thermoregulatory sweat test)

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TABLE 6: Treatment modalities for sudomotor dysfunction (hyperhidrosis).	
Local modalities	• Antiperspirants (aluminum chloride 15–25%) • Tap water iontophoresis (for palmar/plantar sweating) • Botulinum toxin injection
Systemic medications	• Anticholinergic drugs (glycopyrrolate) • Tricyclic antidepressants (amitriptyline) • Beta blockers • Centrally acting sympatholytic drugs (clonidine) • Calcium channel blockers (diltiazem)
Surgical interventions	• Endoscopic thoracic sympathectomy in sweating–disorders of the upper quadrant • Axillary curettage, liposuction for axillary hyperhidrosis

Management of sudomotor dysfunction involves identification of the underlying disorder and directed therapies specific to the underlying etiology. In addition, complications related to sudomotor dysfunction and trophic changes need special care and attention. The various treatment modalities for hyperhidrosis are summarized in **Table 6**. The management options for hypo/anhidrosis are limited. Patients may be encouraged to use moisturizer, avoid agents such as anticholinergics/antihistaminergics which can further worsen the hypo/anhidrosis, and perform exertional activities in a cool, well-ventilated environment. In patients with autoimmune neuropathy and acquired idiopathic generalized anhidrosis (AIGA) steroid therapy may be beneficial.[17-23]

Q6. Discuss the role of vestibular sedatives for common vestibular disorders—BPPV, vestibular migraine, and PPPD.

Ans. Vertigo is a commonly observed neurological symptom where the patient perceives a feeling of movement, a sensation as if the external world were revolving around the patient (objective vertigo) or as if he himself were revolving in space (subjective vertigo). Several diseases can lead to vertigo and these are broadly classified into central and peripheral causes **(Table 7)**.

The commonly used medication for acute vertigo/vestibular syndrome includes the vestibular sedative agents and antiemetic agents **(Table 8)**. These agents are most useful in acute setting and provide early symptomatic relief without altering the underlying pathophysiology. In fact, prolonged use of vestibular sedatives is not recommended as it tends to affect the central adaptive mechanisms. Robust data exists regarding the use of vestibular sedatives in patients with vestibular neuritis and acute vestibular hypofunctioning. The role of vestibular sedatives in select disorders such as benign paroxysmal positional vertigo (BPPV), vestibular migraine, and persistent postural-perceptual dizziness (PPPD) is unknown.

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