STEM-S

ESSENTIAL NOTES

Medical Group

PRESENTED BY
Stem-S

1.APPROACH TO A PATIENT WITH CARDIAC DISEASES

i. JVP Abnormalities

Jugular venous pulse (JVP) reflects **right atrial pressure and right heart hemodynamics**. It provides dynamic information about atrial contraction, ventricular filling, tricuspid valve function, and pericardial pathology. Proper assessment requires the patient to be at 30–45° with identification of the internal jugular venous pulsations.

Normal JVP Waveform

Wave/Descent	Event	Hemodynamic Basis
a wave	Atrial contraction	Right atrial systole
c wave	Tricuspid valve bulging	Early RV systole
x descent	Atrial relaxation	Downward displacement of tricuspid valve
v wave	Atrial filling	Venous return against closed tricuspid valve
y descent	Rapid ventricular filling	Tricuspid valve opening

- Understanding abnormalities requires correlating these components with pathophysiology.

Elevated JVP

An elevated JVP indicates **raised right atrial pressure**.

Causes

- Right ventricular failure
- Pulmonary hypertension
- Tricuspid regurgitation
- Constrictive pericarditis
- Cardiac tamponade
- Fluid overload

2. Splitting of S2

Type of Split	Mechanism	Causes	Key Clinical Clue
Physiological	Inspiration delays P2	Normal finding	Split increases on inspiration
Wide Split	Delayed RV emptying	RBBB, Pulmonary stenosis	Persistent wide split
Fixed Split	Constant RV volume overload	Atrial septal defect	No respiratory variation
Paradoxical (Reversed)	Delayed LV emptying	LBBB, Severe aortic stenosis	Split appears on expiration

3. Third and Fourth Heart Sounds

Sound	Timing	Mechanism	Seen In	Clinical Significance
S3	Early diastole (after S2)	Rapid ventricular filling into dilated ventricle	Heart failure, MR, AR	Volume overload
S4	Late diastole (before S1)	Atrial contraction against stiff ventricle	HTN, AS, HCM	Pressure overload; absent in AF

4. Added Abnormal Sounds

Sound	Timing	Mechanism	Condition
Opening Snap	Early diastole	Sudden tensing of stenotic mitral valve	Mitral stenosis
Ejection Click	Early systole	Abnormal semilunar valve opening	AS (early), PS
Pericardial Knock	Early diastole	Abrupt cessation of ventricular filling	Constrictive pericarditis
Tumor Plop	Early diastole	Movement of atrial myxoma	Atrial myxoma
Pericardial Rub	Systole and diastole	Inflamed pericardial layers rubbing	Acute pericarditis

iv. Approach to Murmur

A cardiac murmur is a **sound produced by turbulent blood flow** across a valve, septal defect, or outflow tract. Proper evaluation requires a systematic approach based on **timing, location, radiation, intensity, pitch, and associated clinical findings**.

Step 1: Determine Timing in Cardiac Cycle

Type	Occurs Between	Common Causes
Systolic murmur	S1 → S2	AS, MR, VSD, HOCM
Diastolic murmur	S2 → S1	AR, MS
Continuous murmur	Throughout systole & diastole	PDA

Timing is the most important initial step because **diastolic murmurs are always pathological**, whereas some systolic murmurs may be innocent.

Step 2: Classify Systolic Murmur

- **Ejection (Mid-systolic) Murmur**

Crescendo–decrescendo pattern.
Seen in:
- Aortic stenosis
- Pulmonary stenosis
- Hypertrophic obstructive cardiomyopathy

Mechanism: Turbulence during ventricular ejection through narrowed outflow tract.

- **Pansystolic (Holosystolic) Murmur**

Uniform intensity throughout systole.
Seen in:
- Mitral regurgitation
- Tricuspid regurgitation
- Ventricular septal defect

Mechanism: Continuous pressure gradient between chambers during systole.

- **KILLIP CLASSIFICATION** (For prognosis associated with post-MI heart failure)

Class	Description	Mortality
I	No HF	<5%
II	S3 gallop/rales/JVP ↑	10–15%
III	Pulmonary edema	20–30%
IV	Cardiogenic shock	40–70%

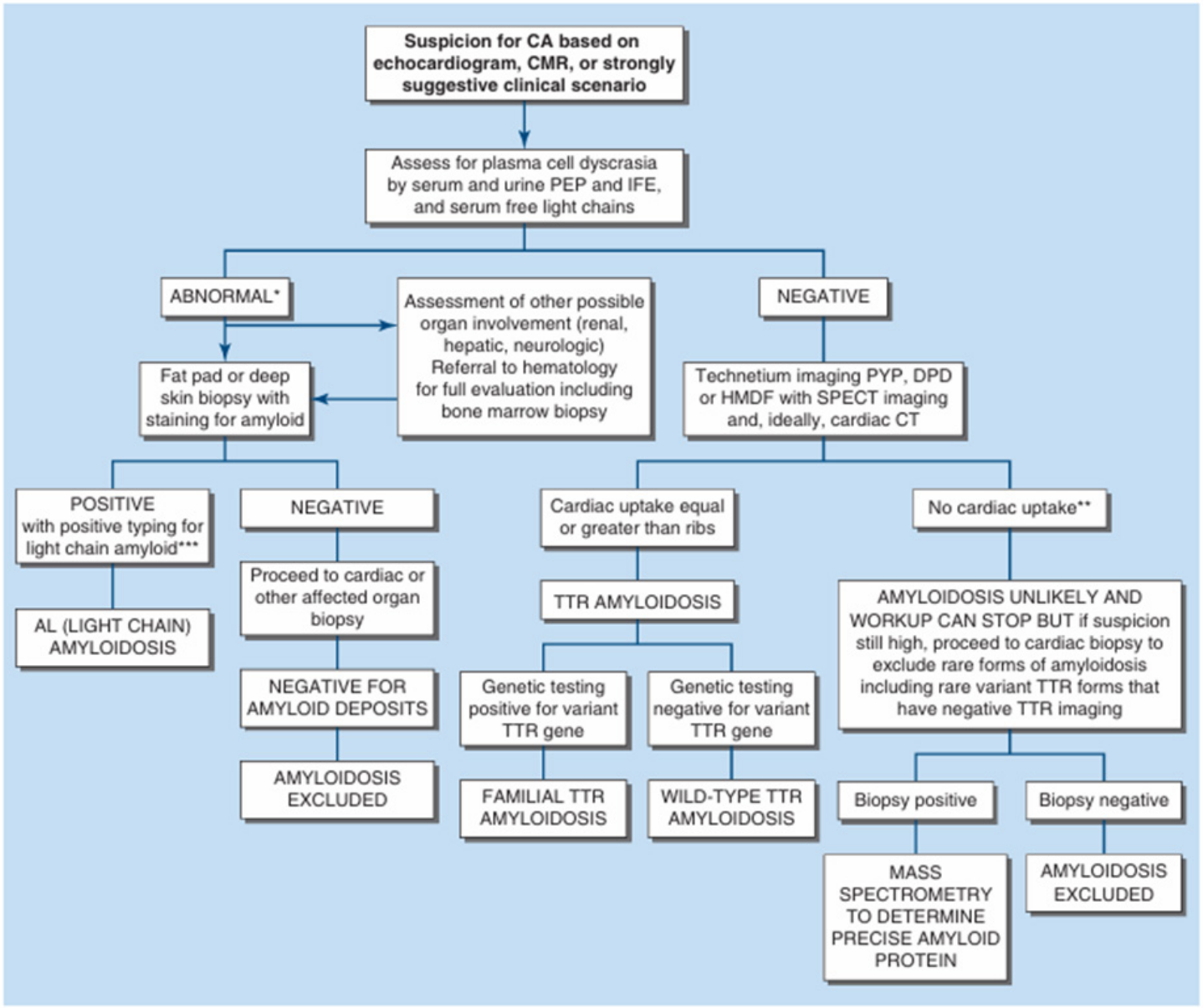
- **CARDIAC BIOMARKERS – KINETICS**

Marker	Rise	Peak	Normalizes	Note
Troponin I/T	3–6 h	12–24 h	7–10 d	Highest sensitivity/specificity
CK-MB	3–6 h	24 h	48–72 h	Detects reinfarction
Myoglobin	1–2 h	6–8 h	24 h	Earliest but nonspecific

COMPLICATIONS OF MI

- **Mechanical**

Characteristic	Ventricular Septal Rupture	Rupture of the Ventricular Free Wall	Papillary Muscle Rupture
Incidence	0.2–3% without reperfusion therapy; 0.2–0.34% with fibrinolytic therapy; ~3.9% in patients with cardiogenic shock	~0.3–1%; fibrinolytic therapy does not reduce risk, primary PCI seems to reduce risk	~0.1–1% (posteromedial more common than anterolateral papillary muscle rupture)
Time course	Bimodal peak within 24 h and 3–5 days (range 1–14 days)	Bimodal peak within 24 h and 3–5 days (range 1–14 days)	Bimodal peak within 24 h and 3–5 days (range 1–14 days)
Clinical manifestations	Chest pain, shortness of breath, hypotension	Anginal, pleuritic, or pericardial pain; syncope; hypotension; restlessness; sudden death	Abrupt onset of shortness of breath and pulmonary edema; hypotension
Physical findings	Harsh holosystolic murmur, thrill, accentuated S_2, pulmonary edema, right and left ventricular failure, cardiogenic shock	Jugular venous distention (≈29%); pulsus paradoxus (≈47%); electromechanical dissociation; cardiogenic shock	Soft murmur in some cases, no thrill, variable signs of RV overload, severe hypotension, edema, cardiogenic shock
Echocardiographic findings	Ventricular septal rupture; left-to-right shunt on color Doppler through septum; RV overload pattern	Pericardial effusion >5 mm; layered/high-acoustic echoes in pericardium (blood clot); direct visualization of tear; signs of tamponade	Hypercontractile LV, torn papillary muscle or chordae tendineae, flail leaflet, severe mitral regurgitation on color Doppler
Right-heart catheterization	Increase in oxygen saturation from RA to RV; large V waves	Ventriculography insensitive; classic tamponade signs; equalization of diastolic pressures in cardiac chambers	No increase in oxygen saturation from RA to RV; large V waves; very high PCWP



x. Cardiac cachexia:

Diagnosis requires ≥5% unintentional weight loss over 6–12 months **and at least *3 of the following 5 criteria:***

1. **Decreased muscle strength**
2. **Fatigue** or reduced exercise tolerance
3. **Anorexia (loss of appetite)**
4. **Low fat-free mass index (lean body mass reduction)**
5. **Abnormal biochemistry**, including any of:
   - ↑ Inflammatory markers (CRP >5 mg/L, IL-6 >4 pg/mL)
   - Anemia (Hb <12 g/dL)
   - Hypoalbuminemia (<3.2 g/dL)

xi. Mechanical circulatory support devices:

Type	Examples	Duration / Purpose
Short-term (Temporary)	Intra-aortic balloon pump (IABP), Impella, TandemHeart, VA-ECMO	Hours to days; used for cardiogenic shock, bridge to decision or recovery
Intermediate-term	CentriMag, paracorporeal VADs	Days to weeks
Long-term (Durable)	LVAD (HeartMate 3, HeartWare HVAD)	Months to years; bridge to transplant or destination therapy

Indications

A. Temporary / Short-Term

- Cardiogenic shock unresponsive to inotropes/vasopressors
- Post-MI shock (Killip IV)
- Fulminant myocarditis / Takotsubo with shock
- Post-cardiotomy LV failure
- Bridge to recovery / bridge to decision / bridge to transplant

B. Long-Term / Durable

- End-stage (NYHA IV) heart failure refractory to GDMT
- Bridge to transplant – awaiting donor heart
- Destination therapy – not a transplant candidate but life expectancy >1 yr
- Bridge to recovery – reversible causes (myocarditis, peripartum)
- Bridge to candidacy – to allow recovery of organ function pre-transplant

Contraindications

Absolute:

- Irreversible end-organ failure (Cr >3 mg/dL, bilirubin >3 mg/dL)
- Severe, irreversible neurological injury
- Active systemic infection / sepsis
- Advanced malignancy or limited life expectancy (<2 yrs)
- Severe bleeding diathesis / coagulopathy

Relative:

- Severe peripheral vascular disease
- Nonadherence / psychosocial limitations
- RV failure (for isolated LVAD)
- Severe pulmonary hypertension (fixed >5 Wood units)

B. Prosthetic Valve IE

1. Staphylococcal PVE (MSSA/MRSA)

Organism	Regimen	Duration
MSSA	Nafcillin/Oxacillin + Rifampin + Gentamicin	**≥6 weeks**, gentamicin 2 weeks
MRSA	Vancomycin + Rifampin + Gentamicin	Same

2. Streptococcal PVE

Regimen	Duration
Penicillin G or Ceftriaxone ± Gentamicin	**6 weeks**

3. Enterococcus PVE

Regimen	Duration
Ampicillin + Ceftriaxone OR Ampicillin + Gentamicin	**6 weeks**

C. HACEK Organisms

Drug	Duration
Ceftriaxone OR Ampicillin-sulbactam OR Fluoroquinolone	**4 weeks (native)** **6 weeks (prosthetic)**

D. Culture-Negative IE

Setting	Regimen
Acute presentation	Vancomycin + Cefepime
Subacute presentation	Vancomycin + Ampicillin-sulbactam

3. Indications for Surgery

Indication	Examples
Heart failure	Severe AR/MR
Uncontrolled infection	Abscess, persistent bacteremia, fungal IE
Prevention of embolization	Vegetation >10 mm + embolic events
Prosthetic valve involvement	Early PVE, dehiscence

Hematocrit (PCV) (fL)	Maturation factor
45	1
35	1.5
25	2
15	2.5

I. Primary cutaneous disorders
   A. Acute and chronic urticaria[a]
   B. Physical urticaria
      1. Dermographism
      2. Solar urticaria[b]
      3. Cold urticaria[b]
      4. Cholinergic urticaria[b]
   C. Angioedema (hereditary and acquired)[b,c]

II. Systemic diseases
   A. Urticarial vasculitis
   B. Hepatitis B or C viral infection, SARS-CoV-2 infection
   C. Serum sickness
   D. Angioedema (hereditary and acquired)

IMPORTANT VALUES:

- Erythropoietin (EPO): ~9–26 mIU/mL
- Serum Ferritin:

Adult male: ~100 µg/L

Adult female: ~30 µg/L

- Serum Iron: ~50-150 µg/dL
- Total Iron-Binding Capacity (TIBC): ~50–150 µg/dL
- Transferrin Saturation (TSAT): ~20–50%

(B) Approach to polycythemia:

Disease	Clinical Manifestations	Histology	Immunopathology	Autoantigens
Pemphigus vulgaris	Oromucosal lesions, flaccid blisters, denuded skin	Acantholytic blister formed in suprabasal layer of epidermis	Cell surface deposits of IgG on keratinocytes	Dsg3 (plus Dsg1 in patients with skin involvement)
Pemphigus foliaceus	Crusts and shallow erosions on scalp, central face, upper chest, and back	Acantholytic blister formed in superficial layer of epidermis	Cell surface deposits of IgG on keratinocytes	Dsg1
Paraneoplastic pemphigus	Painful stomatitis with papulosquamous or lichenoid eruptions that may progress to blisters	Acantholysis, keratinocyte necrosis, and vacuolar interface dermatitis	Cell surface deposits of IgG and C3 on keratinocytes and (variably) similar immunoreactants in epidermal BMZ	Plakin protein family members and desmosomal cadherins
Bullous pemphigoid	Large tense blisters on flexor surfaces and trunk	Subepidermal blister with eosinophil-rich infiltrate	Linear band of IgG and/or C3 in epidermal BMZ	BPAG1, BPAG2
Pemphigoid gestationis	Pruritic urticarial plaques rimmed by vesicles and bullae on trunk and extremities	Teardrop-shaped subepidermal blisters in dermal papillae; eosinophil-rich infiltrate	Linear band of C3 in epidermal BMZ	BPAG2 (plus BPAG1 in some patients)
Dermatitis herpetiformis	Extremely pruritic small papules and vesicles on elbows, knees, buttocks, and posterior neck	Subepidermal blister with neutrophils in dermal papillae	Granular deposits of IgA in dermal papillae	Epidermal transglutaminase
Linear IgA disease	Pruritic papulovesicles on extensor surfaces; occasionally larger arcuiform blisters	Subepidermal blister with neutrophil-rich infiltrate	Linear band of IgA in epidermal BMZ	BPAG2
Epidermolysis bullosa acquisita	Blisters, erosions, scars, and milia on trauma-exposed sites; widespread inflammatory tense blisters may be seen initially	Subepidermal blister that may or may not include a leukocytic infiltrate	Linear band of IgG and/or C3 in epidermal BMZ	Type VII collagen
Mucous membrane pemphigoid	Erosion and/or blistering lesions of mucous membranes and possibly skin; scarring of some sites	Subepidermal blister that may or may not include a leukocytic infiltrate	Linear band of IgG, IgA, and/or C3 in epidermal BMZ	BPAG2, laminin-332, or others

TMPRSS6 (Matriptase-2): Negative Regulator

TMPRSS6, a hepatic transmembrane serine protease, suppresses hepcidin by cleaving membrane-bound hemojuvelin (HJV), thus antagonizing the BMP–SMAD pathway.

Loss-of-function mutations in TMPRSS6 cause iron-refractory iron deficiency anemia (IRIDA), characterized by inappropriately high hepcidin despite iron deficiency.

- Erythroferrone (ERFE): Erythropoietic Suppression of Hepcidin. Erythroferrone is a hormone secreted by erythroblasts in response to erythropoietin (EPO) stimulation (e.g., after hemorrhage or hypoxia).

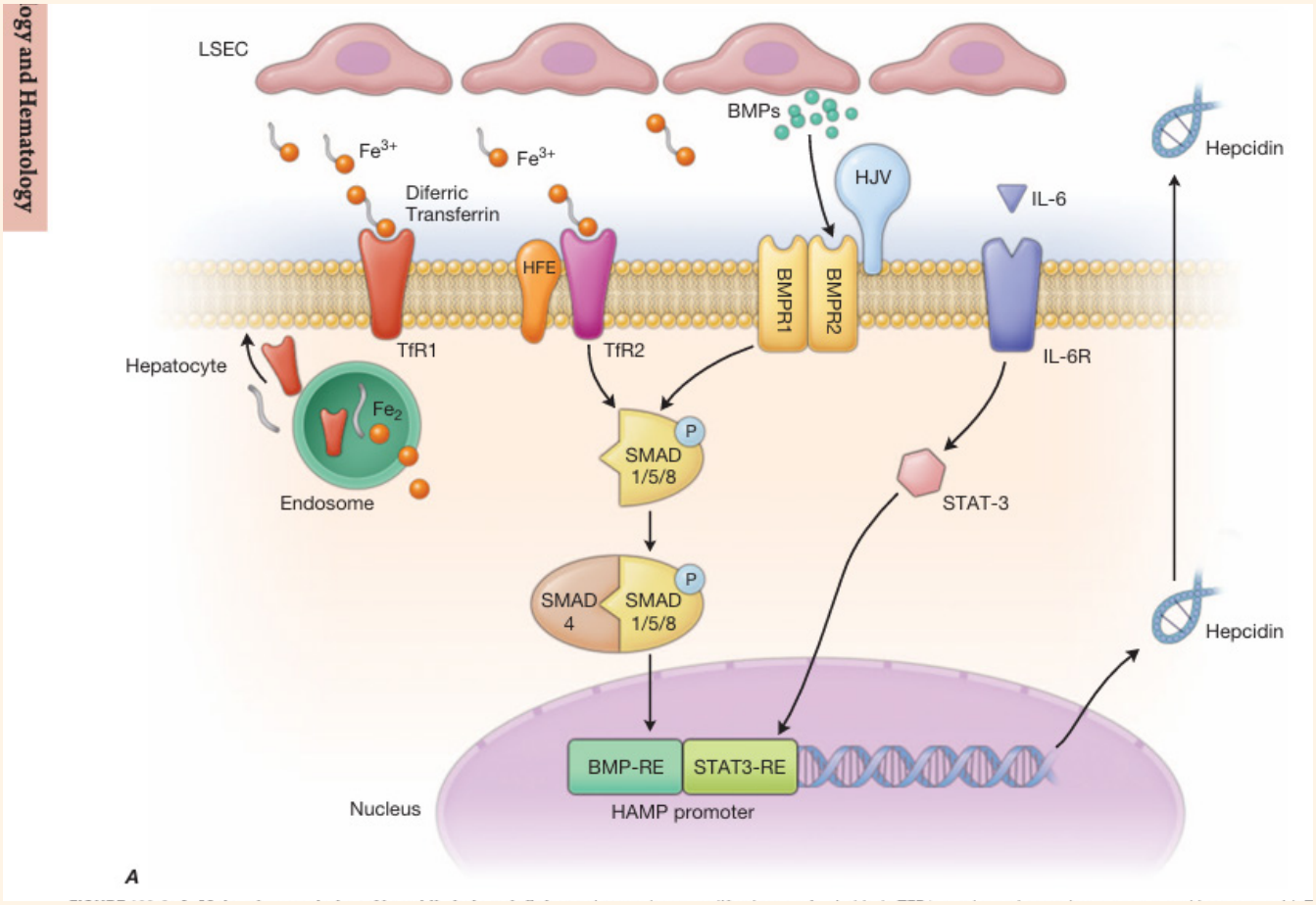
It suppresses hepcidin by binding and sequestering BMPs, thus allowing increased iron mobilization for erythropoiesis.

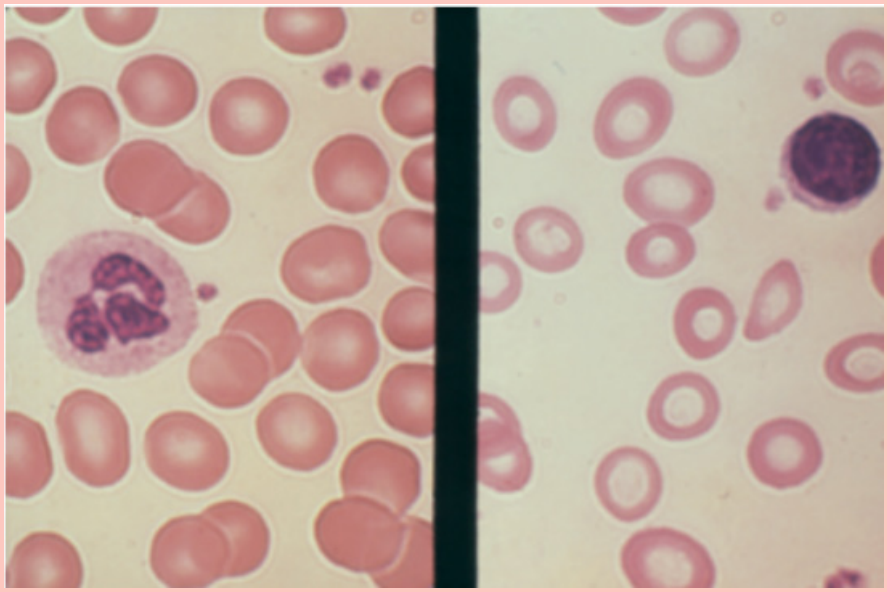
- Other Inhibitors of Hepcidin Expression

Testosterone

Fibrinogen-like protein 1 (FGL1)

Platelet-derived growth factor BB (PDGF-BB)





III.Phases of iron deficiency and their correlation with laboratory studies:

Phase	Serum Ferritin	Serum Iron	TIBC	Transferrin Saturation	Hemoglobin	MCV	Bone Marrow Iron
1. Iron Depletion (Prelatent stage)	↓	Normal	Normal or ↑	Normal	Normal	Normal	↓ or absent
2. Iron-deficient Erythropoiesis (Latent stage)	↓↓	↓	↑	↓	Normal or slightly ↓	↓	Absent
3. Iron Deficiency Anemia (Overt stage)	↓↓↓	↓↓	↑↑	↓↓	↓↓	↓↓ (microcytosis)	Absent

iii. Causes of False (Spurious) High or Low Vitamin B12 Levels

Category	Mechanism / Examples
False High (Pseudoelevated B12)	- Myeloproliferative disorders (↑ transcobalamin I), CML - Liver disease (release of cobalamin from hepatocytes) - Renal failure (impaired clearance of transcobalamin complexes) - Breast, liver, colon cancer
False Low (Pseudodeficiency)	- Folate deficiency (↓ conversion to active methylcobalamin) - Pregnancy or oral contraceptive use (↓ binding proteins) - Multiple myeloma (abnormal binding proteins interfere with assays) - High doses of vitamin C

iv. Causes of Spurious (Artifactually High) MCV/Pseudomacrocytosis

Mechanism	Example / Explanation
Cold agglutinins (CAD)	RBCs clump → analyzer counts clumps as single large cells → ↑ MCV
Hyperglycemia	RBCs swell in vitro due to osmotic shifts
Marked leukocytosis	WBC interference in automated cell counters
Severe reticulocytosis	Reticulocytes are larger → apparent macrocytosis
Delayed sample processing	RBC swelling with time in EDTA tube
Hypernatremia / Hypotonic sample	Osmotic expansion of RBCs

v. Neurological manifestations of B12 deficiency:

Demyelination of CNS and PNS occurs, notably the posterior columns, corticospinal tracts, and peripheral nerves—a condition termed Subacute Combined Degeneration (SACD) of the spinal cord. (MRI- spongy degeneration of spinal cord)These changes are due to defective myelin synthesis caused by impaired conversion of methylmalonyl-CoA to succinyl-CoA.

1. Hypothalamo-Pituitary Disorders

I. HYPOPITUITARISM

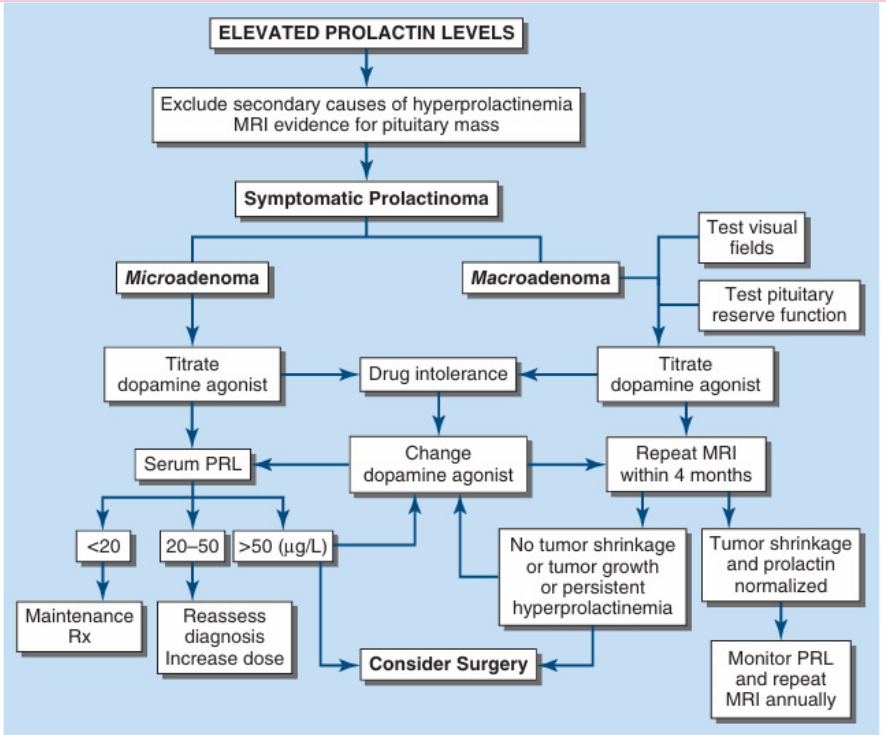
Disorder	Etiology / Pathogenesis	Hormonal Defect	Key Clinical Features	Distinctive Points
Bardet–Biedl Syndrome	Autosomal recessive ciliopathy; hypothalamic dysfunction	Hypogonadotropic hypogonadism (secondary hypogonadism)	Obesity, polydactyly, retinitis pigmentosa, intellectual disability, hypogonadism, renal anomalies	Syndromic obesity with retinal dystrophy + hypogonadism
Prader–Willi Syndrome	Loss of paternal 15q11–q13 (imprinting defect) → hypothalamic dysfunction	GH deficiency, hypogonadotropic hypogonadism	Neonatal hypotonia → hyperphagia → obesity, short stature, small hands/feet, hypogonadism	Hyperphagia + obesity + hypogonadism
Kallmann Syndrome	Failure of GnRH neuron migration (anosmin gene mutation)	Isolated GnRH deficiency → hypogonadotropic hypogonadism	Delayed puberty, anosmia/hyposmia, infertility	Anosmia + delayed puberty
Lymphocytic Hypophysitis	Autoimmune inflammation of pituitary (common in pregnancy/postpartum)	Variable anterior pituitary hormone deficiency	Headache, visual field defects, features of hypopituitarism	Mimics pituitary adenoma; peripartum female
Pituitary Apoplexy	Hemorrhage or infarction of pituitary (often in adenoma)	Acute ACTH deficiency most dangerous	Sudden severe headache, vomiting, visual loss, ophthalmoplegia, hypotension	Neurosurgical emergency; acute adrenal crisis risk

Endocrinology

Disorder	Test	Key Points / Interpretation
Acromegaly	Serum IGF-1	Interpret IGF-1 relative to age- and sex-matched controls
	Oral glucose tolerance test with GH measured at 0, 30, 60 min	Normal subjects suppress GH to <1 µg/L
Prolactinoma	Serum PRL	Exclude medications; if PRL elevated → MRI of sella
Cushing's disease	24-h urinary free cortisol	Ensure urine collection is complete and accurate
	Dexamethasone 1 mg at 11 PM with fasting plasma cortisol at 8 AM	Normal subjects suppress to <5 µg/dL
	Late-night salivary cortisol	Screening test for hypercortisolism
	ACTH assay	Distinguishes adrenal adenoma (ACTH suppressed) from Cushing disease (ACTH normal/elevated)
	CRH stimulation test (peripheral and/or petrosal sinus sampling)	Used mainly to differentiate pituitary vs ectopic ACTH
Gonadotropinoma	Baseline FSH, LH, free α-subunit, ovarian hyperstimulation & estrogen (females), testosterone (males)	Rare; more commonly nonfunctioning adenomas
	TRH stimulation test	Some gonadotropinomas show inappropriate gonadotropin response
TSH-producing adenoma	Free T4, free T3, TSH, free α-subunit	Key feature: inappropriately normal or high TSH with elevated T4/T3

Hormone Deficit	Hormone Replacement
ACTH	Hydrocortisone 10–20 mg/day (divided doses) OR Cortisone acetate 15–25 mg/day OR Prednisone 5 mg/day
TSH	L-Thyroxine 0.075–0.15 mg/day
FSH/LH – Males	Testosterone gel 5–10 g/day, skin patch 5 mg/day, or testosterone enanthate 200 mg IM every 2 weeks
FSH/LH – Females	Conjugated estrogen 0.65–1.25 mg/day for 25 days + Progesterone 5–10 mg/day on days 16–25; Estradiol skin patch 0.025–0.1 mg/week (add progesterone if uterus intact); fertility: menopausal gonadotropins or hCG
GH	Adults: Somatotropin 0.1–1.25 mg SC daily; Children: 0.02–0.05 mg/kg/day
AVP	Intranasal desmopressin 5–20 µg twice daily or Oral 300–600 µg/day

II. PROLACTINOMA:



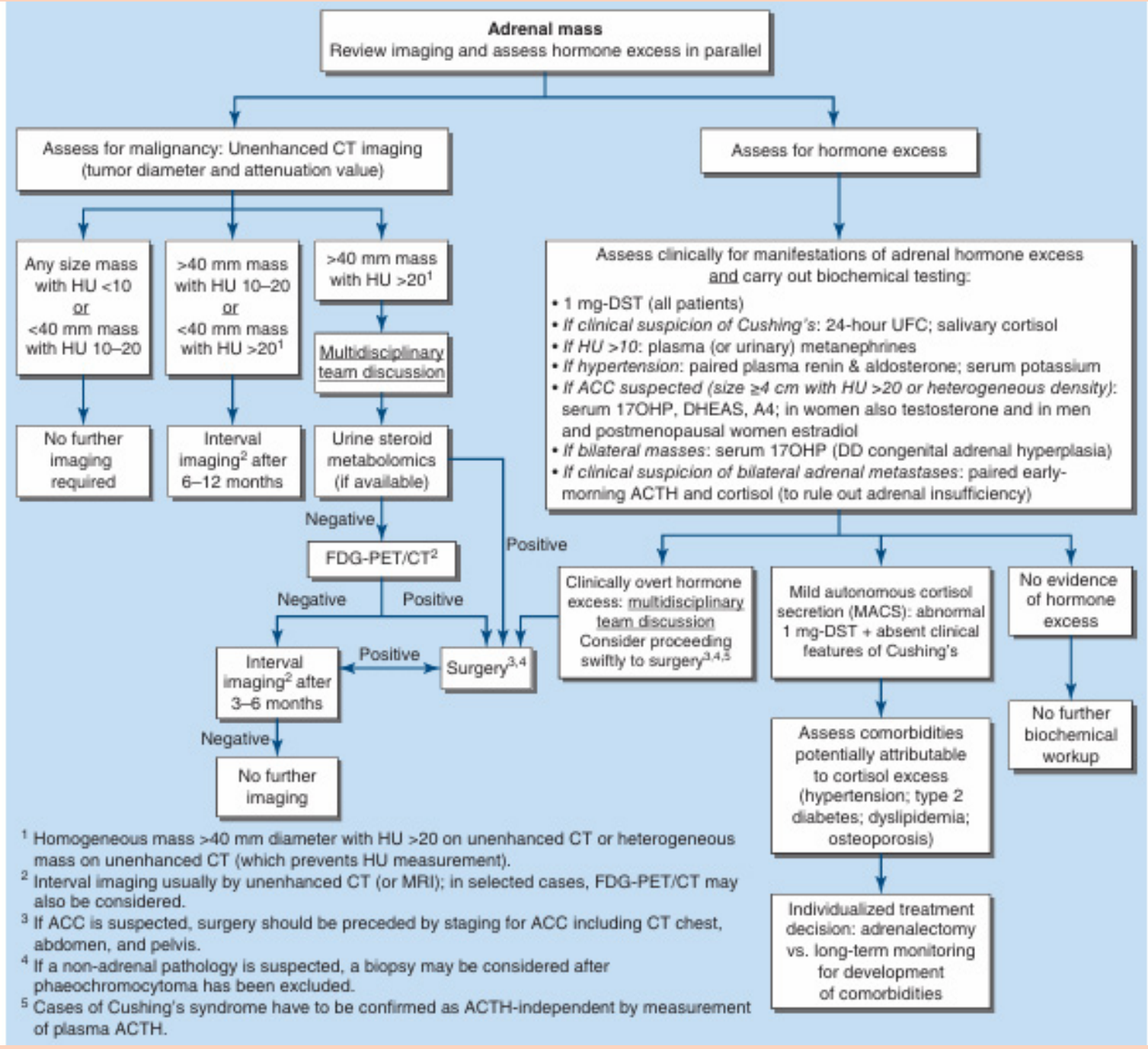
Note: Drug preferred: Cabergoline (0.5-1.0 mg twice weekly) > Bromocriptine (start with 0.625-1.5 mg HS with snack, upto <7.5 mg daily dose). Parknison's disease patients receiving 3 mg daily cabergoline developed cardiac valvular regurgitation.

Feature	Bromocriptine	Cabergoline
Drug class	Ergot-derived dopamine agonist (D2 agonist)	Ergot-derived dopamine agonist (highly selective D2 agonist)
D2 receptor affinity	Moderate	Higher affinity
Half-life	Short (6–8 hours)	Long (~65 hours)
Dosing frequency	Daily (1–2 times/day)	Twice weekly (sometimes once weekly)
Efficacy (PRL normalization)	Effective	More effective than bromocriptine
Tumor shrinkage	Effective	Greater tumor shrinkage
Tolerability	More GI side effects	Better tolerated
Common adverse effects	Nausea, vomiting, orthostatic hypotension, headache	Nausea (less common), dizziness
Risk of valvular heart disease	Rare	Associated with high cumulative doses (mainly Parkinson's doses)

Pre-test Preparation for Accurate ARR:

- Withdraw interfering drugs for ≥2 weeks (4–6 weeks for MR antagonists).
- Normalize potassium and maintain moderate sodium intake.
- Draw sample in the morning after sitting upright for 15–30 minutes.

iii. Incidental adrenal mass:



Erectile Dysfunction

Feature	Details
Definition	Inability to achieve or maintain erection
Prevalence	Increases with age
Major mechanism	Impaired nitric oxide–mediated vasodilation

Causes of Erectile Dysfunction

Category	Causes
Vascular	Atherosclerosis, diabetes
Neurologic	Spinal cord injury
Endocrine	Hypogonadism, hyperprolactinemia
Drugs	SSRIs, beta blockers
Psychogenic	Depression

Evaluation

Investigation	Purpose
Morning testosterone	Screen hypogonadism
Prolactin	Hyperprolactinemia
Thyroid function tests	Thyroid disease
Penile Doppler	Vascular disease
Nocturnal penile tumescence	Psychogenic vs organic ED

Components of the ABCD3I Score

Parameter	Criteria	Points
A – Age	≥ 60 years	1
B – Blood Pressure	≥ 140/90 mmHg at presentation	1
C – Clinical Features	Unilateral weakness = 2 points Speech impairment without weakness = 1 point	2/1
D – Duration of Symptoms	≥ 60 min = 2 points 10–59 min = 1 point	2/1
D – Diabetes Mellitus	Present	1
D₂ – Dual TIA	≥ 2 TIAs within 7 days	2
I – Imaging (MRI/CT)	Acute diffusion-weighted imaging (DWI) lesion = 2 points	2

Total Score Range: 0 – 13

Interpretation:

Score Range	Stroke Risk	Interpretation
0–3	Low	Minimal early stroke risk
4–7	Moderate	Requires prompt outpatient evaluation
≥8	High	High short-term stroke risk; **hospital admission recommended** for urgent evaluation and management

Clinical Utility

- Predicts **2-day, 7-day, and 90-day stroke risk** after a TIA more accurately than ABCD².
- Incorporates **dual TIA events** (recurrence within 7 days) and **DWI-positive lesions**, both of which are strong predictors of early stroke.
- Helps decide **urgency of hospital admission** and **imaging needs**.

2. EPILEPSY:

1. CLINICAL FEATURES:

Terminology:

Term	Definition	Timing	Consciousness	Clinical Features	Localizing Value
Aura	Initial subjective ictal symptom of a focal seizure	At seizure onset	Preserved (by definition)	Epigastric rising sensation, déjà vu, fear, abnormal smell/taste, visual phenomena	High localizing value (e.g., temporal lobe – epigastric rising, fear; occipital – visual aura)
Premonitory Symptoms (Prodrome)	Nonspecific symptoms occurring hours to days before seizure	Before seizure (not ictal)	Fully preserved	Irritability, mood change, headache, sleep disturbance	No localizing value
Automatism	Involuntary, repetitive, purposeless movements during seizure	During seizure (usually focal impaired awareness seizure)	Impaired	Lip smacking, chewing, picking at clothes, fumbling	Suggests temporal lobe (most common)
Ictal Phenomenon	Clinical manifestations occurring during seizure	During seizure	May be preserved or impaired	Motor, sensory, autonomic, psychic symptoms	Depends on seizure focus
Postictal State	Period after seizure with altered brain function	After seizure	Altered (confusion common)	Confusion, drowsiness, Todd's paralysis	May suggest side of focus (e.g., postictal aphasia – dominant hemisphere)
Todd's Paralysis	Transient focal weakness after seizure	Postictal	Altered initially	Temporary hemiparesis	Localizes to contralateral motor cortex

Absence seizures:

Feature	Typical Absence Seizure	Atypical Absence Seizure
Onset and Termination	Sudden onset and sudden termination	Gradual onset and gradual termination
Duration	Short (usually <10–20 seconds)	Longer (often >20 seconds)
Impairment of Consciousness	Brief, complete impairment	More prolonged, less complete impairment
Motor Features	Minimal (eyelid flutter, subtle automatisms)	More prominent tone changes (atonic, tonic components)
Postictal State	No postictal confusion	Mild postictal confusion may occur
EEG Pattern	Generalized 3 Hz spike-and-wave discharges	Slow (<2.5 Hz) spike-and-wave discharges
Association	Childhood absence epilepsy	Symptomatic generalized epilepsy (e.g., Lennox–Gastaut syndrome)
Response to Treatment	Good response to ethosuximide, valproate	Often refractory; treated with valproate, lamotrigine, others

- **Epilepsy syndromes:**

Epilepsy Syndrome	Age of Onset	Seizure Type	EEG Findings	Key Clinical Features
Juvenile Myoclonic Epilepsy (JME)	Adolescence	Myoclonic jerks ± GTC ± absence	4–6 Hz polyspike-and-wave	Early morning myoclonic jerks, lifelong tendency
Lennox–Gastaut Syndrome (LGS)	1–8 years	Multiple types (tonic, atonic, atypical absence)	Slow (<2.5 Hz) spike-and-wave	Developmental delay, refractory seizures
West Syndrome (Infantile Spasms)	3–12 months	Infantile spasms	Hypsarrhythmia	Developmental regression, spasms in clusters
Benign Rolandic Epilepsy (Self-limited epilepsy with centrotemporal spikes)	3–13 years	Focal seizures (face, oropharynx)	Centrotemporal spikes	Nocturnal seizures, good prognosis
Temporal Lobe Epilepsy (TLE)	Any age	Focal impaired awareness	Temporal spikes	Aura (epigastric rising), automatisms
Dravet Syndrome	Infancy	Prolonged febrile + multiple seizure types	Generalized spike-and-wave	Developmental slowing, SCN1A mutation

2.EEG FINDINGS:

Condition / Seizure Type	EEG Pattern	Frequency	Key Feature
Typical Absence Seizure	Generalized spike-and-wave	3 Hz	Abrupt onset and offset
Atypical Absence Seizure	Slow spike-and-wave	<2.5 Hz	Irregular, slower pattern
Juvenile Myoclonic Epilepsy (JME)	Generalized polyspike-and-wave	4–6 Hz	Photosensitive, morning myoclonus
Generalized Tonic–Clonic Seizure	Generalized spike-and-wave / polyspike	Variable	Diffuse synchronous discharge
Focal (Temporal Lobe) Seizure	Anterior temporal spikes/sharp waves	—	Often unilateral
Frontal Lobe Epilepsy	Frontal spikes/sharp waves	—	Brief, may be nocturnal
Benign Rolandic Epilepsy	Centrotemporal spikes	—	Activated during sleep
West Syndrome (Infantile Spasms)	Hypsarrhythmia	Chaotic	High-voltage, disorganized pattern
Lennox–Gastaut Syndrome	Slow spike-and-wave	<2.5 Hz	Background slowing present
Dravet Syndrome	Generalized spike-and-wave / polyspike	—	May be normal early

3.DIFFERENTIAL DIAGNOSIS OF SEIZURE VS SYNCOPE:

Features	Seizure	Syncope
Immediate precipitating factors	Usually none	Emotional stress, Valsalva, orthostatic hypotension, cardiac etiologies
Premonitory symptoms	None or aura (e.g., odd odor)	Tiredness, nausea, diaphoresis, tunneling of vision
Posture at onset	Variable	Usually erect
Transition to unconsciousness	Often immediate	Gradual over seconds
Duration of unconsciousness	Minutes	Seconds
Duration of tonic or clonic movements	30–60 s	Never >15 s
Facial appearance during event	Cyanosis, frothing at mouth	Pallor
Disorientation and sleepiness after event	Many minutes to hours	<5 min
Aching of muscles after event	Often	Sometimes
Biting of tongue	Sometimes	Rarely
Incontinence	Sometimes	Sometimes
Headache	Sometimes	Rarely

4.STATUS EPILEPTICUS (SE)

Definition (ILAE 2015–2022 Consensus)

- **Time point t1:** Continuous seizure activity **≥5 min** (convulsive SE) — *treatment should start.*
- **Time point t2:** Beyond **30 min** → risk of **long-term neuronal injury**.

Types

- **Convulsive SE (generalized tonic–clonic)**
- **Nonconvulsive SE** (complex partial, absence, subtle motor)
- **Refractory SE:** Seizures persist despite **first-line (benzodiazepine)** and **second-line** AEDs.
- **Super-refractory SE:** Continues **>24 h** after anesthetic therapy initiation.

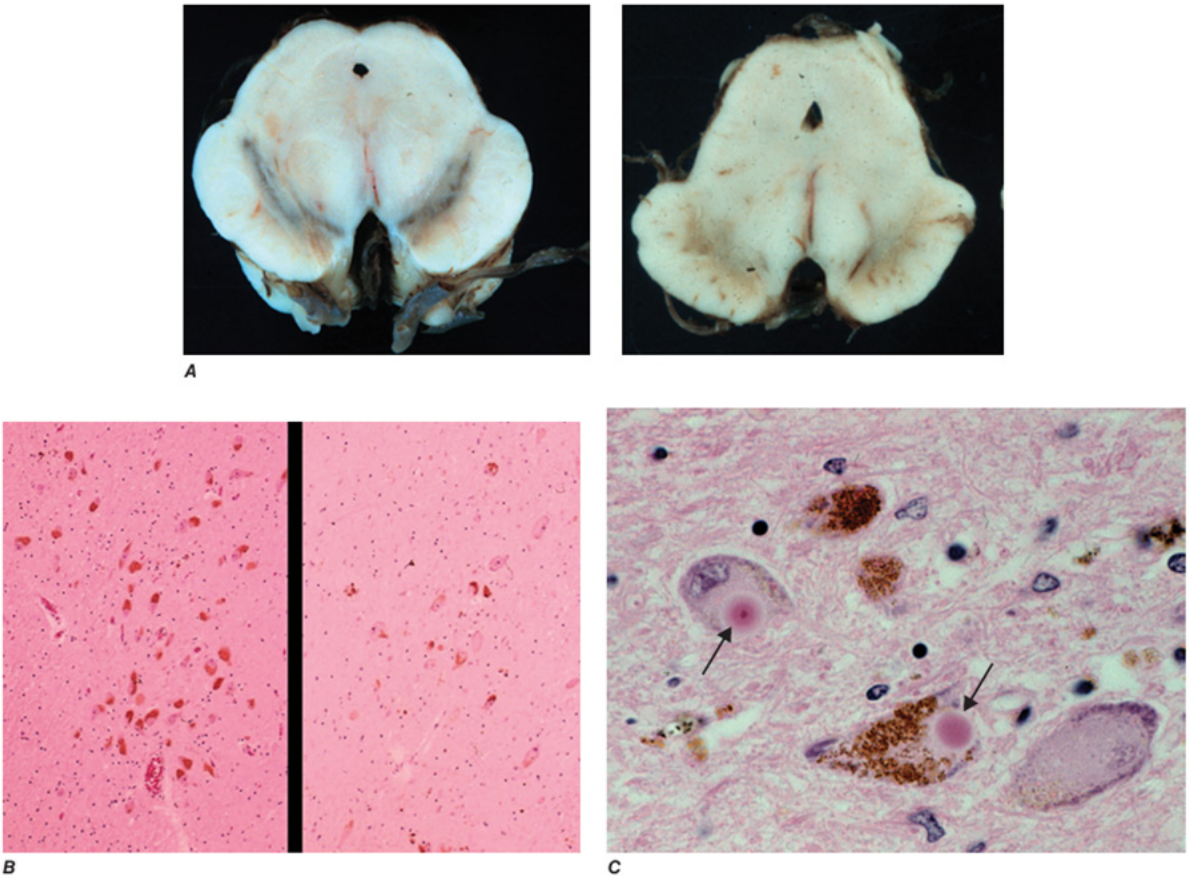


FIGURE 446-1 **Pathologic specimens from a patient with Parkinson's disease (PD) compared to a normal control** demonstrating (***A***) reduction of pigment in SNc in PD (*right*) versus control (*left*), (***B***) reduced numbers of cells in SNc in PD (*right*) compared to control (*left*), and (***C***) Lewy bodies (*arrows*) within melanized dopamine neurons in PD. SNc, substantia nigra pars compacta.

D. RED FLAG SIGNS (Suggesting Atypical or Secondary Parkinsonism)

These features should **raise suspicion against idiopathic PD** and prompt further evaluation for **atypical parkinsonian syndromes** (PSP, MSA, CBD, vascular, drug-induced, etc.).

Red Flag Feature	Possible Alternative Diagnosis
Early falls (<1 year)	PSP
Early autonomic failure (orthostatic hypotension, urinary incontinence)	MSA
Early cognitive decline or visual hallucinations	Dementia with Lewy Bodies
Severe asymmetric dystonia, apraxia, cortical sensory loss	Corticobasal degeneration
Pyramidal signs (spasticity, Babinski)	MSA or vascular parkinsonism
Poor or absent levodopa response	Atypical/secondary parkinsonism
Rapid progression or early gait freezing	PSP, vascular parkinsonism
Ocular movement abnormalities (vertical gaze palsy)	PSP
Early bulbar involvement or cerebellar signs	MSA
Early severe dysautonomia + cerebellar ataxia	MSA-C subtype
Exposure to dopamine blockers (antipsychotics, metoclopramide)	Drug-induced parkinsonism
Stepwise progression, pyramidal signs, vascular risk factors	Vascular parkinsonism

Clinical Presentation	Additional Data Needed for MS Diagnosis
2 or more attacks; objective clinical evidence of ≥2 lesions	None
2 or more attacks; objective clinical evidence of 1 lesion (with reasonable historical evidence of prior attack involving a different CNS site)	**Dissemination in space**, demonstrated by ≥1 T2 lesion on MRI in at least 2 of 4 MS-typical CNS regions (periventricular, juxtacortical/cortical, infratentorial, spinal cord) **OR** wait for a further clinical attack implicating a different CNS site
1 attack; objective clinical evidence of ≥2 lesions	**Dissemination in time**, demonstrated by: • Simultaneous presence of asymptomatic gadolinium-enhancing and non-enhancing lesions at any time **OR** • A new T2 and/or gadolinium-enhancing lesion on follow-up MRI compared with baseline **OR** • Wait for a second clinical attack
1 attack; objective clinical evidence of 1 lesion (clinically isolated syndrome)	**Dissemination in space**, demonstrated by ≥1 T2 lesion in at least 2 of 4 MS-typical CNS regions **AND Dissemination in time**, demonstrated by: • Simultaneous presence of asymptomatic gadolinium-enhancing and non-enhancing lesions **OR** • A new T2 and/or gadolinium-enhancing lesion on follow-up MRI **OR** • Presence of CSF-specific oligoclonal bands
Insidious neurologic progression suggestive of primary progressive MS (PPMS)	**1 year of disease progression** (retrospective or prospective) **PLUS ≥2 of the following:** • Evidence of dissemination in space in the brain based on ≥1 T2 lesions in MS-characteristic regions (periventricular, juxtacortical/cortical, or infratentorial) • Evidence of dissemination in space in the spinal cord based on ≥2 T2 lesions • Positive CSF (CSF-specific oligoclonal bands)

TREATMENT

- **Acute Relapse:**
  - **IV methylprednisolone 1 g/day for 3–5 days**, then oral taper.
  - If steroid-resistant: **Plasma exchange (PLEX)**.

- **CSF FINDINGS**
  - **Oligoclonal IgG bands** in CSF **not present in serum** → marker of intrathecal IgG synthesis (positive in ~85–90%).
  - Mild lymphocytic pleocytosis and ↑ protein.
- **MRI FEATURES**

H-YIELD)T2/FLAIR hyperintense plaques in periventricular, juxtacortical, infratentorial, and spinal cord regions.

- **"Dawson's fingers"** – ovoid periventricular lesions radiating outward along medullary veins.

Acute lesions may enhance with gadolinium.

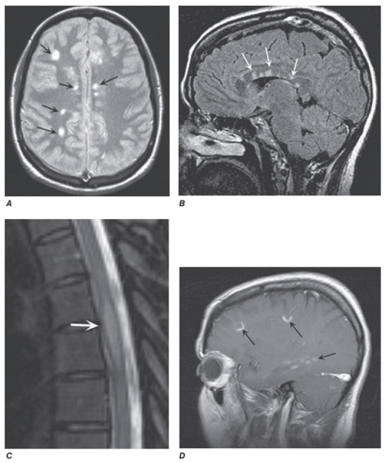


FIGURE 455-3 Magnetic resonance imaging findings in multiple sclerosis (MS). *A.* Axial first-echo image from T2-weighted sequence demonstrates multiple bright signal abnormalities in white matter, typical for MS. *B.* Sagittal T2-weighted fluid-attenuated inversion recovery (FLAIR) image in which the high signal of cerebrospinal fluid (CSF) has been suppressed. CSF appears dark, whereas areas of brain edema or demyelination appear high in signal, as shown here in the corpus callosum (*arrows*). Lesions in the anterior corpus callosum are frequent in MS and rare in vascular disease. *C.* Sagittal T2-weighted fast spin echo image of the thoracic spine demonstrates a fusiform high-signal-intensity lesion in the midthoracic spinal cord. *D.* Sagittal T1-weighted image obtained after the intravenous administration of gadolinium diethylene triamine pentaacetic acid (DTPA) reveals focal areas of blood-brain barrier disruption, identified as high-signal-intensity regions (*arrows*).

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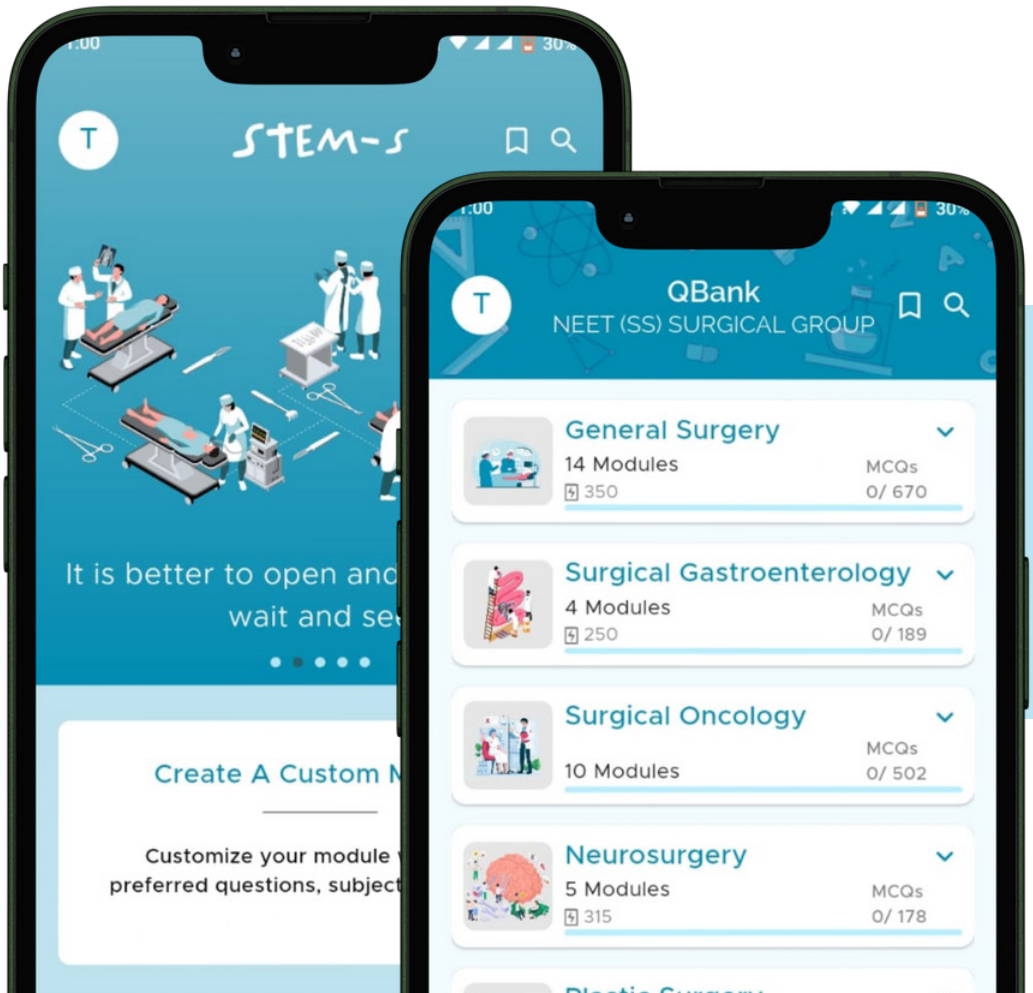

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