FINAL MBBS · GENERAL MEDICINE · NEUROLOGY CASES

A E T C M

Neurology Case Sheet

Writing format — from history taking to the final diagnosis

How to take, examine, write and present a neurology long case — with the theory behind every step, a fully worked hemiplegia case, the NIH Stroke Scale, and how to read CT and MRI in ischaemic and haemorrhagic stroke.

Using this book › | Localisation — UMN vs LMN › | Stroke essentials › | History taking › | General examination ›
Higher functions › | Cranial nerves › | Motor system › | Sensory system › | Cerebellum, gait, meninges ›
Model case sheet › | Summary & diagnosis › | NIHSS › | GCS · mRS · ICH score › | CT — ischaemia vs bleed ›
MRI — DWI, FLAIR, SWI › | Investigations & management › | Viva questions › | Video & image atlas ›

TAP A TOPIC TO JUMP TO IT

WHERE? anatomical › **WHAT?** pathological › **WHY?** aetiological › **HOW MUCH?** functional

WATCH THE VIDEOS · TAP TO PLAY ON YOUTUBE

▶ Cranial nerve examination | ▶ Sensory examination | ▶ Stroke — CNS case discussion

STROKE SCORES & SCANS · TAP TO OPEN

NIHSS scale ↗ | NIHSS calculator ↗ | CT — ischaemic stroke ↗ | MRI — DWI infarct ↗ | CT/MRI — brain bleed ↗

All links — atlas ›

AETCM
Academy for Emergency, Trauma & Critical care Medicine

By

Dr. Gireesh Kumar K.P.

For final-year MBBS students, interns and teachers of clinical medicine

Neurology Case Sheet

History taking · Examination · NIHSS · CT & MRI · Final diagnosis

Dr. Gireesh Kumar K.P.

Academy for Emergency, Trauma & Critical care Medicine (AETCM) · First edition · October 2026

How to use this case sheet

The neurology long case is where examiners test whether you can **think like a clinician**. Every finding you elicit must answer one of four questions: **where** is the lesion (anatomical diagnosis), **what** is the lesion (pathological), **why** did it happen (aetiological) and **how much** has it disabled the patient (functional). This book walks you through that process in the order you will actually do it at the bedside.

Section A gives the **minimum theory** needed to localise a lesion. Sections B–D are the **proforma** — what to ask, what to examine and why it matters. Section E is a **complete case sheet** written the way a final-year student should write it, followed by the **diagnosis format**. Sections G and H cover the **NIH Stroke Scale** and the **CT/MRI of ischaemic and haemorrhagic stroke** — the two things examiners now expect every student to know.

Tap to watch and see

Green buttons ▶ Video play the AETCM teaching videos on YouTube. Maroon CT ↗, purple MRI ↗ and gold Score ↗ buttons open real scans and scoring tools on Radiopaedia, NINDS and MDCalc. A one-page atlas with QR codes is included for the printed copy.

The patient in Section E is a composite teaching case. Drug doses and time windows follow current AHA/ASA and European Stroke Organisation guidance and Indian practice; always check your institution's stroke protocol. CT and MRI drawings in this book are schematics — tap the buttons to see the real images.

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SECTION A · THEORY

Theory for the Bedside

Just enough neuroanatomy to localise any lesion

1. The motor pathway in one paragraph

The corticospinal (pyramidal) tract begins in the **motor cortex** (precentral gyrus, homunculus — face and arm laterally, leg on the medial surface supplied by the ACA), descends through the **corona radiata** and the **posterior limb of the internal capsule** (fibres tightly packed — a small lesion gives a dense hemiplegia), the **cerebral peduncle** (midbrain), the **basis pontis** and the **medullary pyramids**, where ~85 % of fibres **decussate** and descend as the lateral corticospinal tract to synapse on the **anterior horn cell**. Everything above the anterior horn cell is the **upper motor neuron (UMN)**; the anterior horn cell, root, plexus and peripheral nerve form the **lower motor neuron (LMN)**.

Feature	UMN lesion	LMN lesion
Distribution	Groups of muscles (pyramidal pattern — extensors of UL, flexors of LL weaker)	Individual muscles / myotome / nerve territory
Wasting	Absent (late disuse atrophy only)	Marked, early
Fasciculations	Absent	May be present (anterior horn cell)
Tone	Increased — spasticity, clasp-knife (flaccid in the first days — 'neural shock')	Decreased — flaccid
Power	Weak	Weak
Deep tendon reflexes	Exaggerated; clonus	Diminished or absent
Plantar response	Extensor (Babinski)	Flexor or mute
Superficial reflexes	Abdominal and cremasteric lost	Preserved unless segment involved

▶ Babinski sign ↗ ▶ Ankle clonus ↗ ▶ Pronator drift ↗

2. Localising a hemiplegia — the level of the lesion

Level	Clues	Classical example
Cortex	Face + arm > leg (MCA) or leg > arm (ACA); aphasia (dominant), neglect (non-dominant), cortical sensory loss, seizures, hemianopia	Left MCA infarct — right hemiplegia + Broca/ global aphasia
Internal capsule	Dense, equal face–arm–leg weakness; no cortical signs; ± hemisensory loss, hemianopia (posterior limb)	Lacunar pure motor stroke; hypertensive capsular bleed
Midbrain	Ipsilateral III nerve palsy + contralateral hemiplegia	Weber syndrome
Pons	Ipsilateral VI / VII (LMN) palsy + contralateral hemiplegia; gaze palsy towards lesion	Millard–Gubler, Foville syndromes
Medulla	Ipsilateral XII palsy + contralateral hemiplegia; or lateral medullary signs	Medial medullary (Dejerine); Wallenberg
Spinal cord (high cervical)	Ipsilateral weakness, no cranial nerve signs; sensory level; Brown-Séquard pattern	Cord hemisection

Examiner's rule — crossed hemiplegia

Cranial nerve palsy on **one** side with hemiplegia on the **opposite** side = **brainstem** lesion. The cranial nerve involved tells you the level (III — midbrain, VI/VII — pons, XII — medulla). A **UMN** facial palsy on the **same** side as the hemiplegia = lesion **above** the facial nucleus (capsule or cortex).

3. Stroke essentials

Stroke is an acute focal (sometimes global) neurological deficit of vascular origin lasting >24 h or causing death, or with imaging evidence of infarction. A **TIA** is a transient episode of focal dysfunction from ischaemia **without** infarction on imaging (tissue-based definition). About **80–85 %** of strokes are **ischaemic** and 15–20 % **haemorrhagic** (intracerebral haemorrhage, subarachnoid

haemorrhage); in India the proportion of haemorrhage is higher because of uncontrolled hypertension. Clinical features **cannot** reliably separate infarct from bleed — **imaging is mandatory**.

Artery	Clinical picture
MCA (most common)	Contralateral face + arm > leg weakness and sensory loss; homonymous hemianopia; gaze deviation towards lesion; aphasia (dominant) or neglect / anosognosia (non-dominant)
ACA	Contralateral leg > arm weakness; urinary incontinence; abulia, frontal release signs, grasp reflex
PCA	Contralateral homonymous hemianopia with macular sparing; alexia without agraphia (dominant); thalamic sensory loss
Lacunar (penetrating arteries)	Pure motor, pure sensory, sensorimotor, ataxic hemiparesis, dysarthria–clumsy hand; no cortical signs
Vertebrobasilar	Vertigo, diplopia, dysarthria, dysphagia, ataxia, crossed signs, quadriparesis, coma ('locked-in' with basilar occlusion)

TOAST aetiology of ischaemic stroke	Clues
1. Large-artery atherosclerosis	Carotid bruit, >50 % stenosis on Doppler/CTA; diabetic, smoker, dyslipidaemia
2. Cardioembolism	AF, recent MI, rheumatic MS, prosthetic valve, LV thrombus, endocarditis; multiple territories
3. Small-vessel occlusion (lacunar)	Hypertension, diabetes; lacunar syndrome; infarct <15 mm
4. Other determined cause	Young stroke — dissection, vasculitis, APLA, hypercoagulable state, CVT, drugs
5. Undetermined	Two causes, or none found after full work-up (ESUS)

Oxfordshire (Bamford) class	Criteria	Prognosis
TACS — total anterior circulation	All three: hemiparesis/hemisensory loss, homonymous hemianopia, higher cortical dysfunction	Worst — ~60 % dead at 1 year
PACS — partial anterior circulation	Two of the three, or cortical dysfunction alone	High early recurrence
LACS — lacunar	Pure motor / sensory / sensorimotor / ataxic hemiparesis	Best survival
POCS — posterior circulation	Brainstem / cerebellar signs, isolated hemianopia	Intermediate

▶ **Watch: stroke case discussion**

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SECTION B · HISTORY TAKING

The Neurology Proforma — History

What to ask, in what order, and why it matters

1. Particulars and chief complaints

Item	What to record	Why it matters
Name, age, sex	Exact age	Young (<45 y) stroke → look for cardiac, vasculitic, prothrombotic causes
Handedness	Right / left; which hand to write, eat, throw	Decides the dominant hemisphere — needed to interpret aphasia
Occupation, education	Job, years of schooling	Baseline for higher functions; functional impact; occupational toxins
Residence, informant	Place; reliable bystander	Aphasic / drowsy patients need a bystander history
Chief complaints	In the patient's words, in chronological order, with duration	e.g. 'Weakness of right upper and lower limb — 3 weeks; difficulty in speaking — 3 weeks'

2. History of presenting illness — the questions that localise

Ask about	Details	Interpretation
Mode of onset	Sudden (seconds–minutes), acute (hours), subacute (days), chronic (weeks–months); activity at onset	Sudden = vascular; on waking = thrombotic; during exertion with headache/vomiting = bleed; subacute = infection/demyelination; chronic progressive = tumour, compressive
Time of onset / last seen normal	Exact clock time	Decides eligibility for thrombolysis (≤4.5 h) and thrombectomy (≤24 h)
Progression	Maximal at onset, stepwise, progressive, improving	Stepwise = recurrent emboli / thrombosis in evolution; improving = favourable
Distribution of weakness	Side; UL vs LL; proximal vs distal; face	Face+arm > leg = MCA cortex; leg > arm = ACA; equal = capsule
Higher functions	Speech, comprehension, reading, writing, memory, behaviour	Aphasia = dominant cortex; neglect = non-dominant parietal
Cranial nerve symptoms	Vision loss, diplopia, deviation of mouth, drooling, nasal regurgitation, dysphagia, hoarseness, vertigo, hearing loss	Brainstem involvement; bulbar palsy → aspiration risk
Sensory symptoms	Numbness, paraesthesia, pain; distribution	Thalamic pain (Dejerine–Roussy); sensory level → cord
Cerebellar	Unsteadiness, swaying, tremor during reaching	Posterior circulation stroke
Raised ICP	Headache, vomiting, blurring of vision, altered sensorium	Large infarct with oedema, haemorrhage, tumour
Seizures, LOC	Type, duration, post-ictal state	Cortical lesion; Todd's paralysis is a stroke mimic
Bladder and bowel	Retention, urgency, incontinence	Bilateral frontal (ACA) or spinal cord lesion
Negative history	Fever, neck pain, trauma, chest pain, palpitations	Rules out meningitis, dissection, subdural haematoma, cardioembolism

3. Past, personal, family and treatment history

History	Ask about	Why it matters
Past	Hypertension, diabetes, IHD, AF, rheumatic heart disease, previous stroke/TIA, seizures, TB, HIV, syphilis, migraine	Risk factors and aetiology (TOAST)
Drugs	Antihypertensives (adherence), antiplatelets, anticoagulants (INR), OCPs, statins	Non-adherence → stroke; anticoagulant → bleed risk and reversal

History	Ask about	Why it matters
Personal	Smoking (pack-years), alcohol (units), diet, sleep, snoring (OSA), drug abuse	Modifiable risk factors
Family	Stroke, IHD, diabetes, hypertension at young age, sudden death	Premature atherosclerosis; inherited thrombophilia; CADASIL
Menstrual / obstetric	Pregnancy, puerperium, abortions, OCP use	Cerebral venous thrombosis; APLA syndrome (recurrent abortions)
Socio-economic	Family support, house (stairs, toilet), income	Rehabilitation and discharge planning

Summary of history — write it before you examine

Close the history with a 3-line summary that already contains a **provisional anatomical and aetiological diagnosis**: *"A 62-year-old right-handed hypertensive diabetic smoker with sudden-onset right hemiparesis and non-fluent speech, maximal at onset, without headache or vomiting — likely a left MCA territory ischaemic stroke."* The examination is then done to confirm or refute this hypothesis.

SECTION C · GENERAL EXAMINATION

General Examination

The vascular and systemic clues to the cause of the stroke

Look for	Significance
Consciousness, orientation, build, nutrition, bedsores	Severity; nursing care
Pallor, icterus, cyanosis, clubbing, lymphadenopathy, oedema	Anaemia, endocarditis (clubbing), malignancy, TB
Pulse — rate, rhythm, all peripheral pulses, radio-radial delay	**AF** = cardioembolism; absent pulses = Takayasu arteritis / atherosclerosis
BP — both arms, supine	Hypertension (cause, and management decisions); arm difference >20 mmHg = subclavian stenosis
Carotids — palpate one at a time; auscultate for **bruit**	Carotid atherosclerosis
Respiratory rate and pattern, SpO_2, temperature	Aspiration pneumonia; Cheyne–Stokes in large lesions
Fundus	Hypertensive/diabetic retinopathy, papilloedema, emboli (Hollenhorst plaques)
Skin and eyes	Xanthelasma, arcus (dyslipidaemia), livedo reticularis (APLA), neurocutaneous markers
Heart (brief CVS exam)	Murmur of MS/MR, prosthetic click, cardiomegaly — source of emboli

SECTION D · CNS EXAMINATION

Higher Mental Functions

Consciousness, orientation, memory, speech — tested first, written first

Function	How to test	Record as
Consciousness	Observe; GCS (E, V, M)	Conscious / drowsy / stuporous / comatose; GCS E4V5M6 = 15
Orientation	Time, place, person	Oriented to time, place and person
Attention	Serial 7s, digit span, months backwards	Attention preserved
Memory	Immediate (3 words), recent (breakfast), remote (school, wedding)	Immediate, recent and remote memory intact
Intelligence, calculation	General knowledge; simple sums	Appropriate for education
Mood, behaviour, hallucinations	Observe; ask	Emotional lability is common after stroke
MMSE / MoCA	Screening for cognitive impairment	MMSE ≤23/30 suggests cognitive impairment

Speech — the most examined higher function

First decide whether the problem is **dysphonia** (voice — X nerve, larynx), **dysarthria** (articulation — UMN, LMN, cerebellar, extrapyramidal) or **aphasia** (language — dominant cortex). Then test the six components of language: **fluency**, **comprehension**, **repetition**, **naming**, **reading** and **writing**.

Aphasia	Fluency	Comprehension	Repetition	Lesion
Broca (expressive)	Non-fluent, effortful, telegraphic	Intact	Impaired	Inferior frontal gyrus (MCA superior division)
Wernicke (receptive)	Fluent, paraphasias, jargon	Impaired	Impaired	Superior temporal gyrus (MCA inferior division)
Global	Non-fluent	Impaired	Impaired	Large MCA stem / perisylvian
Conduction	Fluent	Intact	**Severely impaired**	Arcuate fasciculus / supramarginal gyrus
Transcortical motor	Non-fluent	Intact	**Preserved**	Watershed ACA–MCA, anterior
Transcortical sensory	Fluent	Impaired	**Preserved**	Watershed MCA–PCA, posterior
Anomic	Fluent, word-finding pauses	Intact	Intact	Angular gyrus; any lesion; recovery stage

Examiner's tip — dysarthria types

Spastic (pseudobulbar — slow, strained, 'hot-potato'), **flaccid** (bulbar — nasal, breathy), **ataxic** (scanning, explosive — cerebellar), **hypokinetic** (soft monotone — Parkinson's). Ask the patient to say 'British Constitution', 'West Register Street', 'pa-pa-pa' (lips), 'ta-ta-ta' (tongue), 'ka-ka-ka' (palate).

SECTION D · CNS EXAMINATION

Cranial Nerves

Twelve nerves, examined in order — one line each in the case sheet

▶ Watch: cranial nerve examination | UMN vs LMN facial palsy ↗

Nerve	How to test	Abnormal findings
I Olfactory	Each nostril separately with familiar non-irritant smell (coffee, soap)	Anosmia — frontal tumour, head injury, Kallmann
II Optic	Acuity (Snellen / finger counting), colour (Ishihara), fields by confrontation, pupillary reflexes, fundus	Homonymous hemianopia (optic radiation / occipital), RAPD, papilloedema, optic atrophy
III, IV, VI	Ptosis, pupils, ocular movements in 'H' pattern, diplopia, nystagmus, gaze	III: ptosis, 'down and out', dilated pupil; VI: failure of abduction; conjugate gaze deviation towards cortical lesion (away from pontine lesion)
V Trigeminal	Sensation in V1, V2, V3; masseter/temporalis; jaw deviation; corneal reflex; jaw jerk	Jaw deviates to side of LMN lesion; brisk jaw jerk = bilateral UMN above pons
VII Facial	Raise eyebrows, close eyes tight, show teeth, blow cheeks; taste anterior 2/3	**UMN**: lower face only (forehead spared — bilateral cortical supply); **LMN**: whole half of face, Bell's phenomenon
VIII Vestibulocochlear	Whisper / finger rub, Rinne, Weber; nystagmus; head impulse if vertigo	Sensorineural deafness; vestibular nystagmus
IX, X	Voice, cough, swallow; palate movement on 'aah'; gag reflex	Uvula deviates to **normal** side in unilateral X palsy; nasal regurgitation
XI Accessory	Shrug shoulders (trapezius), turn head against resistance (SCM)	Weak SCM turns head away from the weak side
XII Hypoglossal	Tongue at rest (wasting, fasciculations), protrusion, side-to-side	Tongue deviates **towards** the side of LMN lesion; in UMN lesion towards the hemiplegic side

Bulbar vs pseudobulbar palsy

Bulbar (LMN — IX, X, XII nuclei): wasted fasciculating tongue, absent gag, nasal speech, normal jaw jerk (MND, GBS, syringobulbia). **Pseudobulbar (bilateral UMN)**: small spastic tongue, brisk jaw jerk, spastic dysarthria, emotional lability (bilateral strokes, MND, MS).

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SECTION D · CNS EXAMINATION

Motor System

Bulk · Tone · Power · Reflexes · Coordination · Gait · Involuntary movements

Step	Method	Record
Attitude	Observe the patient lying	Wernicke–Mann posture: UL flexed, adducted, internally rotated; LL extended, foot plantar-flexed
Bulk	Inspect; measure circumference (10 cm above/below olecranon, 18 cm above / 10 cm below tibial tuberosity)	Compare sides; wasting suggests LMN
Tone	Passive movement at wrist, elbow, knee, ankle; knee roll; pronator catch	Spasticity (clasp-knife, velocity-dependent) vs rigidity (lead-pipe, cog-wheel) vs hypotonia
Power	Each muscle group against resistance; pronator drift; Medical Research Council grade	e.g. Right UL proximal 2/5, distal 1/5; Right LL 3/5
Deep tendon reflexes	Jaw, biceps (C5,6), supinator (C5,6), triceps (C7,8), knee (L3,4), ankle (S1); reinforcement	Grade 0 absent, 1+ diminished, 2+ normal, 3+ brisk, 4+ with clonus
Superficial reflexes	Abdominal (T7–T12), cremasteric (L1), plantar (S1)	Abdominal absent on hemiplegic side; extensor plantar
Clonus	Ankle (sudden sustained dorsiflexion), patellar	Sustained clonus (>3 beats) = UMN
Coordination	Finger–nose, heel–knee–shin, dysdiadochokinesia	Cannot be tested if power <3/5 — write 'could not be assessed'
Gait	Walk, turn; tandem; Romberg	Hemiplegic circumduction gait; ataxic; spastic; high-stepping
Involuntary movements	Tremor, chorea, athetosis, myoclonus, fasciculations	Hemiballismus = subthalamic nucleus

▶ Pronator drift ↗ ▶ Babinski sign ↗ ▶ Ankle clonus ↗ ▶ Hemiplegic gait ↗

MRC grade	Definition
0	No contraction
1	Flicker or trace of contraction
2	Active movement with gravity eliminated
3	Active movement against gravity
4	Active movement against gravity and some resistance (4−, 4, 4+)
5	Normal power

SECTION D · CNS EXAMINATION

Sensory System

Primary and cortical sensations — compare side to side, distal to proximal

▶ Watch: sensory system examination

Modality	Tract	Test with
Pain	Lateral spinothalamic	Disposable pin — never a hypodermic needle
Temperature	Lateral spinothalamic	Test tubes with warm (40–45 °C) and cold water
Crude touch	Anterior spinothalamic	Cotton wisp, dabbed not dragged
Fine touch, two-point discrimination	Dorsal column	Divider — fingertip normal 2–4 mm
Vibration	Dorsal column	128 Hz tuning fork on bony points, distal to proximal

Modality	Tract	Test with
Joint position	Dorsal column	Small movements of the terminal phalanx, sides of the digit; Romberg
Cortical sensations	Parietal cortex	Stereognosis, graphaesthesia, two-point discrimination, sensory localisation, sensory extinction — test only if primary sensations are intact

Pattern of sensory loss	Site of lesion
Glove and stocking	Peripheral polyneuropathy
Dermatomal	Root
Sensory level on the trunk	Spinal cord
Dissociated (pain and temperature lost, touch preserved) — cape distribution	Syringomyelia / central cord
Ipsilateral dorsal column + contralateral spinothalamic loss	Brown-Séquard (cord hemisection)
Crossed: ipsilateral face + contralateral body	Lateral medulla (Wallenberg)
Hemianaesthesia of face, arm and leg, all modalities	Thalamus / posterior limb of internal capsule
Cortical sensory loss with intact primary sensation	Parietal cortex

SECTION D · CNS EXAMINATION

Cerebellum, Gait, Meninges, Skull and Spine

Complete the CNS — the steps students forget

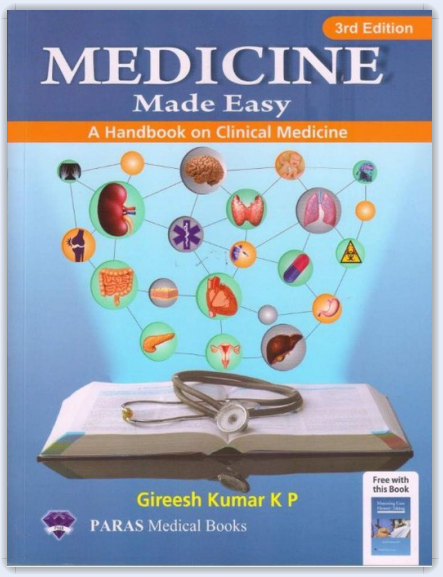
System	Examine	Abnormal
Cerebellar	Nystagmus, scanning speech, finger–nose (intention tremor, past-pointing), dysdiadochokinesia, heel–shin, rebound, pendular knee jerk, tandem gait	Ipsilateral signs in cerebellar hemisphere lesions; truncal ataxia in vermis lesions — remember 'DANISH'
Meningeal signs	Neck stiffness, Kernig's sign, Brudzinski's sign	Meningitis, subarachnoid haemorrhage
Skull and spine	Shape, scars, bruit over orbit/skull, tenderness, deformity, gibbus	Head injury, AVM, Pott's spine
Autonomic	Postural BP fall, sweating, bladder	Diabetic autonomic neuropathy, cord lesion
Other systems	CVS, respiratory, abdomen (brief)	Source of emboli; aspiration pneumonia; DVT; bedsores

Trauma & Critical care Medicine

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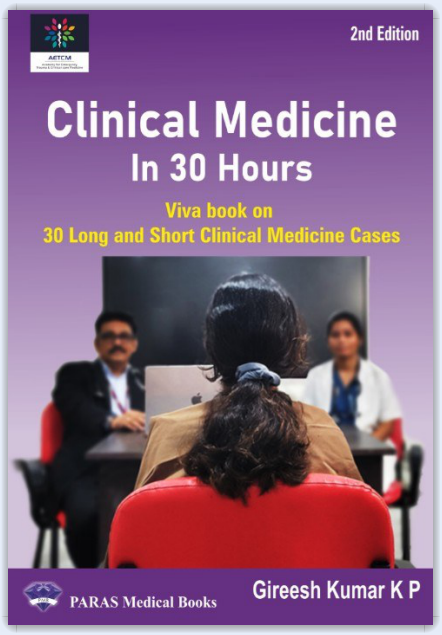
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SECTION E · MODEL CASE SHEET

Right Hemiplegia with Expressive Aphasia

A complete case sheet written the way a final-year MBBS student should write it

History

Particulars	Mr. R. K., 62 years, male, right-handed, retired bus driver, educated up to 10th standard, resident of Ernakulam district. Informant — patient (partly) and wife, reliable.
Date of admission	Admitted 3 weeks ago; examined on day 21.
Chief complaints	1. Weakness of right upper and lower limb — 3 weeks 2. Difficulty in speaking — 3 weeks 3. Deviation of mouth to the left — 3 weeks

History of presenting illness. The patient was apparently normal 3 weeks ago, when at about 7 a.m., while drinking tea, he **suddenly** dropped the cup from his right hand and was unable to stand because of weakness of the right lower limb. The weakness was **maximal at onset** and has **not progressed**; since the second week he is able to move the right leg in bed and stand with support. His wife noticed that he could **understand** what was said and obey commands but could **speak only a few words with great effort**; he became frustrated when he could not express himself. She also noticed deviation of the angle of the mouth to the left and drooling of saliva from the right angle. He was brought to hospital **5 hours** after onset (outside the thrombolysis window).

No headache, vomiting, loss of consciousness or seizures at onset. No blurring or loss of vision, double vision, difficulty in swallowing, nasal regurgitation or change of voice. No vertigo or unsteadiness. No numbness or tingling. No bladder or bowel disturbance. No fever, neck pain, trauma, chest pain or palpitations. No similar episodes or transient weakness in the past.

Past history. Known hypertensive for 10 years on amlodipine 5 mg — **irregular** treatment for one year. Type 2 diabetes for 6 years on metformin 500 mg twice daily. No history of IHD, rheumatic fever, AF, previous stroke, TB or seizures.

Personal history. Smoker — 20 beedis/day for 35 years (35 pack-years); occasional alcohol. Mixed diet, high salt intake. Normal sleep; snores. Bladder and bowel habits normal before the illness.

Family history. Father had a stroke at 65 years. Mother diabetic. **Socio-economic:** lives with wife and son in a two-storey house; toilet upstairs.

Summary of history

A 62-year-old right-handed hypertensive (irregular treatment) diabetic smoker presented with **sudden-onset right hemiparesis**, **non-fluent speech with preserved comprehension** and deviation of the mouth to the left, maximal at onset, without features of raised intracranial pressure — suggesting an **ischaemic stroke in the left MCA (superior division) territory**.

General examination and vitals

Finding	In this patient	Significance
Consciousness	Conscious, cooperative; GCS E4V(aphasic)M6	Record V as 'aphasic' rather than giving a falsely low score
Build, nutrition	Moderately built, BMI 26 kg/m²	Overweight
Pallor, icterus, cyanosis, clubbing, lymphadenopathy, oedema	Absent	—
Pulse	84/min, regular, all peripheral pulses felt, no radio-radial delay	No AF — cardioembolism less likely (Holter needed)
BP	160/96 mmHg right arm, 158/94 left arm, supine	Uncontrolled hypertension
Carotids	Soft bruit over left carotid	Left ICA atherosclerosis — large-artery aetiology
RR, SpO_2, temperature	18/min, 97 % room air, afebrile	No aspiration pneumonia
Fundus	Arteriovenous nipping, no papilloedema	Hypertensive retinopathy grade II
Others	Arcus, no xanthelasma; no bedsores	Dyslipidaemia likely

CNS examination

Component	Finding	Interpretation
Higher functions	Conscious, oriented (by gestures and yes/no); memory intact; **non-fluent, effortful, telegraphic speech**; comprehension of simple and complex commands intact; repetition impaired; naming impaired; reading aloud impaired, reading comprehension preserved; writing could not be tested (right hand)	**Broca's (expressive) aphasia** — left inferior frontal gyrus → **left hemisphere dominant**
Cranial nerves	I — normal. II — acuity 6/9 both eyes, fields full by confrontation, pupils equal and reactive. III, IV, VI — full movements, no nystagmus, no gaze deviation. V — normal. **VII — right lower facial weakness, forehead spared**. VIII — normal. IX, X — palatal movement normal, gag present. XI — normal. **XII — tongue deviates to the right**, no wasting	**Right UMN facial and hypoglossal palsy** — lesion above the nuclei, same side as hemiplegia
Motor — bulk	Normal, no wasting; arm and calf circumference equal	UMN pattern
Tone	**Increased** right UL (clasp-knife at elbow) and LL (knee, ankle)	Spasticity — stage of neural shock is over
Power	**Right UL**: shoulder 2/5, elbow 2/5, wrist and fingers 1/5. **Right LL**: hip 3/5, knee 3/5, ankle 2/5. Left side 5/5	**Arm > leg**, distal > proximal → MCA territory (cortex / corona radiata)

Reflex	Right	Left
Jaw jerk	Normal	—
Biceps	3+	2+
Supinator	3+	2+
Triceps	3+	2+
Knee	3+	2+
Ankle	3+ with ankle clonus (5 beats)	2+
Abdominal	Absent	Present
Plantar	**Extensor**	Flexor

Component	Finding	Interpretation
Coordination	Left side normal; right side could not be tested because of weakness	—
Gait	Walks a few steps with support — **circumduction** of right leg, right arm held flexed	Hemiplegic gait
Sensory	Pain, temperature, touch, vibration and joint position normal on both sides; cortical sensations intact on the left (right hand could not be tested reliably)	No significant sensory deficit — parietal lobe and thalamus spared
Cerebellar	No nystagmus; left side normal	—
Meninges, skull, spine	No neck stiffness; Kernig's negative; skull and spine normal	—
Other systems	CVS — S1 S2 normal, no murmur, apex not displaced. RS — normal. Abdomen — normal	No cardiac source of emboli clinically

60-second presentation

"Mr. R. K., a 62-year-old right-handed retired bus driver, hypertensive on irregular treatment, diabetic and a 35 pack-year smoker, presented with sudden-onset weakness of the right upper and lower limbs, difficulty in speaking and deviation of the mouth, maximal at onset, 3 weeks ago, without headache, vomiting, seizures or loss of consciousness. On examination he is conscious with Broca's aphasia, right UMN facial and hypoglossal palsy, and right spastic hemiparesis — arm worse than leg — with brisk reflexes, ankle clonus and an extensor plantar; sensations are preserved. BP is 160/96 mmHg and there is a left carotid bruit. My diagnosis is right spastic hemiparesis due to an ischaemic stroke in the left MCA (superior division) territory, probably large-artery atherosclerosis, with hypertension, diabetes and smoking as risk factors."

SECTION F · DIAGNOSIS

How to Write the Final Diagnosis

Four layers — where, what, why, how much

Layer	Question	In this patient
Clinical (syndromic)	What is the deficit?	Right spastic hemiparesis (UL > LL) with right UMN VII and XII palsy and Broca's aphasia
Anatomical	Where is the lesion?	Left cerebral hemisphere — cortical/subcortical frontal lobe, left MCA superior division territory
Pathological	What is the lesion?	Infarction (sudden onset, no raised-ICP features) — to be confirmed by CT/MRI
Aetiological	Why did it happen?	Large-artery atherosclerosis (TOAST 1) — left ICA disease suspected (bruit)
Risk factors	What predisposed?	Hypertension (uncontrolled), type 2 diabetes, smoking, family history, likely dyslipidaemia
Functional	How disabled?	mRS 4 (cannot walk without help); NIHSS at admission 12
Complications	What else?	None at present — watch for aspiration, DVT, bedsores, shoulder pain, depression

Write it in one sentence

Final diagnosis: Right spastic hemiparesis with right UMN facial and hypoglossal palsy and Broca's aphasia due to **acute ischaemic stroke in the left MCA (superior division) territory**, aetiology large-artery atherosclerosis (TOAST 1), with uncontrolled systemic hypertension, type 2 diabetes mellitus and smoking as risk factors; NIHSS 12 at admission; current functional status mRS 4.

Differential diagnosis of a sudden hemiplegia

Condition	Points for	Points against in this patient
Intracerebral haemorrhage	Hypertensive, sudden onset	No headache, vomiting or drop in consciousness — but only CT can exclude it
Cardioembolic stroke	Sudden, maximal at onset, cortical signs	Regular pulse, no murmur — still needs ECG/Holter and echo
Subdural haematoma	Elderly, hemiparesis	No trauma, sudden not fluctuating onset
Todd's paralysis	Post-ictal weakness	No seizure; deficit persistent
Hypoglycaemia	Diabetic on drugs	Check capillary glucose in every stroke — corrects within minutes
Brain tumour / abscess	Hemiparesis	Gradual onset expected; no fever
Cerebral venous thrombosis	Headache, seizures, young women, puerperium	Male, no headache

If the CT shows a bleed — how the diagnosis changes

*"Right dense hemiplegia (face, arm and leg equally) with right UMN facial palsy and right hemianaesthesia due to **acute spontaneous intracerebral haemorrhage in the left basal ganglia / internal capsule** with/without intraventricular extension, aetiology **hypertensive vasculopathy**; GCS 13; ICH score 1; haematoma volume ~18 mL."* Headache, vomiting, early drowsiness, very high BP and a deficit that worsens over the first hours favour haemorrhage clinically.

SECTION G · STROKE SCORES

NIH Stroke Scale (NIHSS)

11 items, 15 scored elements, total 0–42 — administer in order, score the first response

Official NIHSS — NINDS ↗ | NIHSS calculator — MDCalc ↗ | ▶ Watch: stroke case

Item	Test	Scoring
1a Level of consciousness	Observe	0 alert · 1 arousable by minor stimulation · 2 needs repeated/painful stimulation · 3 reflex responses or unresponsive
1b LOC questions	Month and age	0 both correct · 1 one correct · 2 neither (aphasic or stuporous = 2; intubated/dysarthric = 1)
1c LOC commands	Open/close eyes; grip and release non-paretic hand	0 both · 1 one · 2 neither
2 Best gaze	Horizontal eye movements only	0 normal · 1 partial gaze palsy · 2 forced deviation
3 Visual fields	Confrontation, all quadrants	0 none · 1 partial hemianopia · 2 complete hemianopia · 3 bilateral (cortical blindness)
4 Facial palsy	Show teeth, raise eyebrows, close eyes	0 normal · 1 minor (flat nasolabial fold) · 2 partial (lower face) · 3 complete (upper + lower)
5a/5b Motor arm (L, R)	Arms at 90° (sitting) or 45° (supine) for 10 s	0 no drift · 1 drift · 2 some effort against gravity · 3 no effort against gravity · 4 no movement · UN = amputation/joint fusion
6a/6b Motor leg (L, R)	Leg at 30° supine for 5 s	0 no drift · 1 drift · 2 some effort against gravity · 3 no effort against gravity · 4 no movement
7 Limb ataxia	Finger–nose, heel–shin	0 absent · 1 one limb · 2 two limbs (score 0 if weakness explains it)
8 Sensory	Pinprick to face, arm, trunk, leg	0 normal · 1 mild–moderate loss · 2 severe/total loss (coma = 2)
9 Best language	Describe picture, name objects, read sentences	0 normal · 1 mild–moderate aphasia · 2 severe aphasia · 3 mute, global aphasia
10 Dysarthria	Read word list	0 normal · 1 mild–moderate · 2 severe/unintelligible · UN = intubated
11 Extinction / inattention	Double simultaneous stimulation (visual, tactile)	0 none · 1 inattention in one modality · 2 profound hemi-inattention (>1 modality)

NIHSS total	Severity	Practical meaning
0	No stroke symptoms	—
1–4	Minor stroke	Consider DAPT for 21 days if non-disabling (NIHSS ≤3)
5–15	Moderate	Thrombolysis if within window; screen for large-vessel occlusion
16–20	Moderate–severe	LVO likely (NIHSS ≥6 → CT angiography for thrombectomy)
21–42	Severe	High risk of oedema, herniation and death

NIHSS of the model case at admission = 12

1a 0 · 1b 2 (aphasic) · 1c 0 · gaze 0 · fields 0 · face 2 · R arm 3 · L arm 0 · R leg 2 · L leg 0 · ataxia 0 · sensory 0 · language 2 · dysarthria 1 · extinction 0 = **12 (moderate stroke)**. Note that the NIHSS weighs left-hemisphere (language) strokes more heavily — a right hemisphere stroke of the same size scores lower.

Other scores you must know

Glasgow Coma Scale	Response	Score
Eye opening (E)	Spontaneous 4 · to sound 3 · to pressure 2 · none 1	E 1–4
Verbal (V)	Oriented 5 · confused 4 · words 3 · sounds 2 · none 1 (record 'T' if intubated, 'A' if aphasic)	V 1–5

Glasgow Coma Scale	Response	Score
Motor (M)	Obeys 6 · localises 5 · normal flexion 4 · abnormal flexion 3 · extension 2 · none 1	M 1–6

GCS — official site ↗

Modified Rankin Scale (mRS)	Description
0	No symptoms
1	No significant disability; carries out all usual activities
2	Slight disability; looks after own affairs without help
3	Moderate disability; needs some help but walks without assistance
4	Moderately severe; cannot walk or attend to bodily needs without help
5	Severe; bedridden, incontinent, constant care
6	Dead

ICH score (Hemphill)	Points
GCS 3–4 = 2 · GCS 5–12 = 1 · GCS 13–15 = 0	0–2
Haematoma volume ≥30 mL (ABC/2)	1
Intraventricular extension	1
Infratentorial origin	1
Age ≥80 years	1
30-day mortality: 0 → 0 %, 1 → 13 %, 2 → 26 %, 3 → 72 %, 4 → 97 %, 5 → 100 %	Total 0–6

ABC/2 — haematoma volume at the bedside

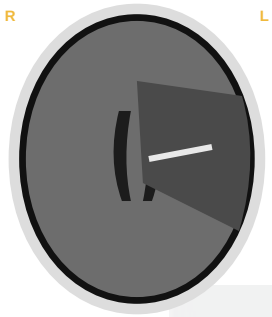
On the CT slice with the largest bleed, **A** = longest diameter (cm), **B** = diameter perpendicular to A, **C** = number of slices showing blood × slice thickness (cm). Volume (mL) = **A × B × C ÷ 2**.

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SECTION H · IMAGING

CT in Stroke — Ischaemia versus Bleed

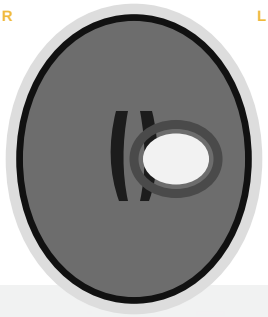
Non-contrast CT is the first test in every stroke — its job is to exclude haemorrhage



Wedge hypodensity, L MCA territory

Ischaemic stroke (schematic)

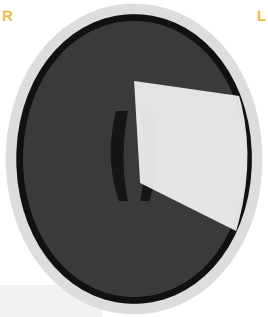
Dense MCA ↗ Insular ribbon ↗



Hyperdense clot, L basal ganglia

Intracerebral haemorrhage (schematic)

BG bleed ↗ With IVH ↗



DWI bright + ADC dark = acute infarct

MRI DWI — acute infarct (schematic)

DWI/ADC ↗ Pontine ↗

Time from onset	CT appearance of an infarct
0–6 h (hyperacute)	Often **normal**; **hyperdense MCA sign** (thrombus); **loss of insular ribbon**; obscured lentiform nucleus; loss of grey–white differentiation; sulcal effacement
6–24 h	Ill-defined hypodensity in an arterial territory becomes visible
1–7 days (acute)	Well-defined wedge-shaped hypodensity involving grey and white matter; **mass effect** peaks at 3–5 days; risk of haemorrhagic transformation
2–3 weeks	**Fogging** — infarct may become transiently isodense
Months (chronic)	CSF-density cavity (encephalomalacia), ex-vacuo dilatation of the adjacent ventricle, Wallerian degeneration

Stroke progression 0 → 24 h ↗ Insular ribbon — article ↗ ASPECTS ↗

ASPECTS — scoring early ischaemic change in the MCA territory

Start with **10** and subtract 1 for each region with early ischaemic change: caudate, lentiform, internal capsule, insula, and M1–M6 cortical regions at two axial levels. **ASPECTS ≥6** is generally used to select patients for thrombectomy; a score of **7 or less** carries a sharp increase in dependence and death.

Haemorrhage on CT	Appearance
Acute (hours–3 days)	**Hyperdense** (60–80 HU) well-defined mass with a thin hypodense rim of oedema; may extend into ventricles (IVH) causing hydrocephalus
Subacute (1–6 weeks)	Density falls from the periphery inwards — becomes isodense, then hypodense; ring enhancement with contrast
Chronic	Hypodense slit-like cavity
Location clue	**Hypertensive**: putamen (most common), thalamus, pons, cerebellum. **Lobar** bleed in elderly: cerebral amyloid angiopathy. Young / atypical: AVM, aneurysm, tumour, CVT, drugs — needs CTA/MRI
'Spot sign' on CTA	Contrast extravasation within the clot — predicts haematoma expansion

Haemorrhagic stroke — basal ganglia ↗ Bleed with intraventricular extension ↗

SECTION H · IMAGING

MRI in Stroke — DWI, FLAIR, SWI

MRI is more sensitive for infarction, posterior fossa lesions and old microbleeds

Sequence	Acute ischaemic infarct	Use
DWI (diffusion-weighted)	**Bright within minutes** of onset — cytotoxic oedema restricts water movement	Most sensitive for acute infarct (posterior fossa, lacunes)
ADC map	**Dark** (low ADC) — confirms true restriction; pseudonormalises at 1–2 weeks	DWI bright + ADC dark = acute; DWI bright + ADC bright = T2 shine-through (old)
FLAIR	Becomes bright after ~4.5–6 h	**DWI–FLAIR mismatch** (DWI positive, FLAIR negative) suggests onset <4.5 h — used in wake-up stroke
SWI / GRE (T2*)	Dark 'blooming' of thrombus (susceptibility vessel sign)	Detects haemorrhage, microbleeds, haemorrhagic transformation
MRA / perfusion	Vessel occlusion; penumbra (perfusion–diffusion mismatch)	Selects patients for late thrombectomy

MCA infarct — DWI/ADC ↗ | Pontine infarct — DWI ↗

Haemorrhage on MRI — ageing blood

Stage	Time	Haemoglobin	T1	T2
Hyperacute	<24 h	Intracellular oxyHb	Iso	Bright
Acute	1–3 days	Intracellular deoxyHb	Iso	Dark
Early subacute	3–7 days	Intracellular metHb	Bright	Dark
Late subacute	1–4 weeks	Extracellular metHb	Bright	Bright
Chronic	>4 weeks	Haemosiderin (rim)	Dark	Dark

Mnemonic

'It Be, Iddy Biddy, BaBy, Doo Doo' — read the letters in pairs as T1 then T2 (I = iso, B = bright, D = dark): hyperacute I-B, acute I-D, early subacute B-D, late subacute B-B, chronic D-D. On SWI/GRE all stages except very early hyperacute blood **bloom dark**.

Haemorrhage on MRI ↗ | BG bleed — CT and MRI ↗ | Thalamic bleed — MRI ↗

Feature	Ischaemic stroke	Haemorrhagic stroke
Onset	On waking / at rest (thrombotic); sudden (embolic)	During activity, often with headache and vomiting
Consciousness	Usually preserved (except large/brainstem)	Early drowsiness common
Progression	Maximal at onset or stepwise	Worsens over minutes–hours (haematoma expansion)
BP	Raised	Often very high
CT	Normal early; later hypodense wedge in a vascular territory	**Hyperdense** immediately
MRI	DWI bright, ADC dark	SWI/GRE blooming; T1/T2 by age
Treatment	Thrombolysis/thrombectomy, antiplatelets	BP lowering, reverse anticoagulation; antiplatelets withheld

SECTION I · INVESTIGATIONS & MANAGEMENT

Investigations and Management

What to order and what to say when the examiner asks 'how will you treat?'

Investigation	Purpose
Capillary blood glucose (at once)	Exclude hypoglycaemia — the commonest treatable mimic
Non-contrast CT brain (door-to-CT <20 min)	Exclude haemorrhage; early ischaemic signs; ASPECTS
CT angiography ± perfusion (head and neck)	Large-vessel occlusion, carotid stenosis, penumbra — thrombectomy decision
MRI brain with DWI, FLAIR, SWI; MRA	Confirm infarct, posterior fossa, wake-up stroke, microbleeds
CBC, RFT, electrolytes, coagulation (PT/INR, aPTT), lipid profile, HbA1c	Before lysis (platelets, INR); risk factors
ECG, Holter monitoring, troponin	AF, MI
2D echo (± transoesophageal)	LV thrombus, valve disease, PFO, endocarditis
Carotid Doppler	Symptomatic stenosis 50–99 % → endarterectomy / stenting
Young stroke work-up	ESR, ANA, APLA, homocysteine, protein C/S, antithrombin, VDRL, HIV, CSF, MRV for CVT

Acute ischaemic stroke

- **ABC, airway protection, swallow screen** before oral intake; keep SpO_2 >94 %; treat fever and hyperglycaemia (target 140–180 mg/dL).
- **IV thrombolysis within 4.5 h** of last known well: **tenecteplase 0.25 mg/kg** (max 25 mg) single bolus, or **alteplase 0.9 mg/kg** (max 90 mg) — 10 % as bolus, rest over 60 minutes. BP must be <185/110 before and <180/105 for 24 h after.
- **Mechanical thrombectomy** for large-vessel occlusion (ICA, M1, basilar) up to 6 h, and in selected patients up to 24 h with imaging selection.
- Without lysis, do not lower BP unless >220/120 mmHg (permissive hypertension).
- **Aspirin** 150–325 mg within 24–48 h (24 h after thrombolysis). Minor stroke (NIHSS ≤3) or high-risk TIA: aspirin + clopidogrel for 21 days, then single antiplatelet.
- **High-intensity statin** (atorvastatin 40–80 mg); DVT prophylaxis (intermittent pneumatic compression); early mobilisation, physiotherapy, speech therapy.
- **Malignant MCA infarct**: decompressive hemicraniectomy within 48 h in patients ≤60 years.
- **Secondary prevention**: BP <130/80, diabetes control, stop smoking; anticoagulation for AF (DOAC); carotid endarterectomy for symptomatic stenosis 70–99 % within 2 weeks.

Acute intracerebral haemorrhage

- Lower systolic BP to about **140 mmHg** (target range 130–150) within an hour; avoid dropping below 130 mmHg.
- **Reverse anticoagulation**: warfarin — IV vitamin K + 4-factor prothrombin complex concentrate; dabigatran — idarucizumab; factor Xa inhibitors — andexanet alfa or PCC. Stop antiplatelets.
- Head end elevation 30°, treat seizures (no prophylaxis), normoglycaemia, normothermia, DVT prophylaxis with compression devices.
- **Neurosurgery**: cerebellar haemorrhage >3 cm or with brainstem compression/hydrocephalus → evacuation; external ventricular drain for hydrocephalus; selected lobar bleeds.

SECTION J · VIVA VOCE

Frequently Asked Viva Questions

Short, exam-ready answers

Q1 What are the four parts of a neurological diagnosis?
Anatomical (where), pathological (what), aetiological (why) and functional (how much) — plus risk factors and complications.

Q2 Why is the forehead spared in a UMN facial palsy?
The part of the facial nucleus supplying the upper face receives corticobulbar fibres from both hemispheres; the lower face receives only contralateral fibres.

Q3 Why is tone reduced in the first days after a stroke?
Neural (cerebral) shock — temporary loss of descending facilitation; spasticity develops over days to weeks.

Q4 What is the pyramidal pattern of weakness?
Extensors of the upper limb and flexors of the lower limb are weaker than their antagonists — producing the flexed arm and extended leg posture.

Q5 How do you localise a lesion with left III palsy and right hemiplegia?
Left midbrain (cerebral peduncle) — Weber syndrome.

Q6 Which artery supplies the internal capsule?
Lenticulostriate branches of the MCA (most), anterior choroidal artery (posterior limb), recurrent artery of Heubner (anterior limb).

Q7 Why is the leg spared in MCA stroke?
The leg area of the motor cortex is on the medial surface of the hemisphere, supplied by the ACA.

Q8 What is Babinski's sign?
Dorsiflexion of the great toe with fanning of the other toes on stroking the lateral sole — extensor plantar response, indicating a UMN lesion (normal in infants <1 year).

Q9 Name the causes of an absent ankle jerk with extensor plantar.
Subacute combined degeneration (B12), Friedreich's ataxia, MND, taboparesis, conus lesion, diabetes with stroke.

Q10 What are the components of the NIHSS?
LOC (1a–c), gaze, visual fields, facial palsy, motor arms, motor legs, ataxia, sensory, language, dysarthria, extinction — 15 elements, 0–42.

Q11 What NIHSS score makes you suspect large-vessel occlusion?
NIHSS ≥6 — proceed to CT angiography for possible thrombectomy.

Q12 What is the time window for IV thrombolysis?
Within 4.5 hours of the time last known well.

Q13 Give contraindications to thrombolysis.
Intracranial haemorrhage on CT, recent major surgery/head injury (3 months), prior ICH, BP >185/110 uncontrolled, platelets <1 lakh, INR >1.7, therapeutic anticoagulant, glucose <50 mg/dL, large hypodensity on CT (>1/3 MCA).

Q14 What is the earliest CT sign of MCA occlusion?
The hyperdense MCA sign (thrombus in the artery), followed by loss of the insular ribbon and obscured lentiform nucleus.

Q15 What is 'fogging'?
A 2–3-week-old infarct can become transiently isodense on CT and be missed.

Q16 Which MRI sequence detects an infarct earliest?
Diffusion-weighted imaging — bright within minutes, with a dark ADC.

Q17 What is DWI–FLAIR mismatch?
DWI positive but FLAIR negative lesion — the stroke is probably <4.5 h old; used to select wake-up strokes for thrombolysis.

Q18 Common sites of hypertensive haemorrhage?
Putamen (commonest), thalamus, pons, cerebellum, subcortical white matter.

Q19 What is the ICH score?
GCS, haematoma volume ≥30 mL, IVH, infratentorial origin, age ≥80 — total 0–6, predicts 30-day mortality.

Q20 How is haematoma volume calculated?
ABC/2 — the product of the three diameters in cm divided by 2.

Q21 What is a lacunar infarct?
A small (<15 mm) deep infarct from occlusion of a single penetrating artery, usually from hypertensive lipohyalinosis.

Q22 What is Wallenberg syndrome?

Lateral medullary syndrome (PICA / vertebral) — ipsilateral facial pain/temperature loss, Horner's, ataxia, dysphagia, hoarseness; contralateral body pain/temperature loss; no hemiplegia.

Q23 What is a 'stroke in the young' and how do you work it up?

Stroke <45 years — look for cardioembolism (RHD, PFO), dissection, vasculitis, APLA, prothrombotic states, CVT, drugs, homocystinuria.

Q24 What is the role of aspirin in ICH?

None in the acute stage — it is withheld; restarting antiplatelets later is individualised.

Q25 Name stroke mimics.

Hypoglycaemia, Todd's paralysis, migraine with aura, subdural haematoma, tumour, encephalitis, functional disorder, Bell's palsy (isolated LMN VII).

Q26 What is the mRS used for?

Measuring global disability after stroke; mRS 0–2 is a 'good outcome' in trials.

Q27 When is decompressive hemicraniectomy indicated?

Malignant MCA infarction in patients ≤60 years, within 48 hours of onset.

Q28 What is Todd's paralysis?

Transient post-ictal focal weakness lasting hours (up to 48 h) after a seizure.

Q29 What are the complications of stroke?

Aspiration pneumonia, DVT/PE, bedsores, urinary infection, seizures, depression, shoulder–hand syndrome, contractures, falls, haemorrhagic transformation.

Q30 What is the secondary prevention of stroke?

Antiplatelet (or anticoagulant in AF), statin, BP <130/80, diabetes control, smoking cessation, carotid endarterectomy for symptomatic 70–99 % stenosis.

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VIDEO & IMAGE ATLAS

Watch the Examination, See the Scans

AETCM teaching videos on YouTube, real CT and MRI cases from Radiopaedia, and scoring tools from NINDS and MDCalc

Cranial nerve examination
Video

Sensory examination
Video

Stroke — CNS case discussion
Video

NIHSS — official NINDS page
Score

NIHSS calculator — MDCalc
Score

Glasgow Coma Scale — official site
Score

ASPECTS — Radiopaedia
Score

Hyperdense MCA sign — CT
CT

Dense MCA + loss of insular ribbon
CT

Stroke progression on CT (0 → 24 h)
CT

MCA infarct — CT + DWI/ADC
MRI

Pontine infarct — DWI/ADC
MRI

Hypertensive basal ganglia bleed — CT + MRI
CT+MRI

Bleed with intraventricular extension
CT

Haemorrhagic stroke — basal ganglia CT
CT

Thalamic bleed — CT + MRI
MRI

Haemorrhage on MRI — ageing blood
MRI

Babinski sign — video
Video

Ankle clonus — video
Video

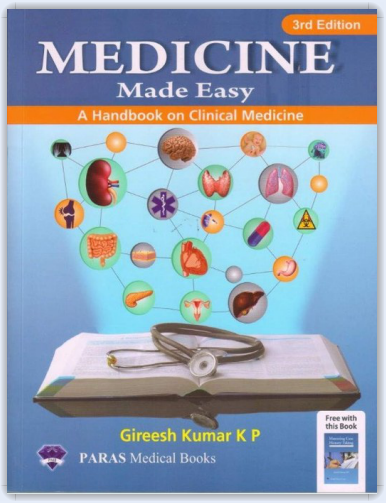
Pronator drift — video
Video

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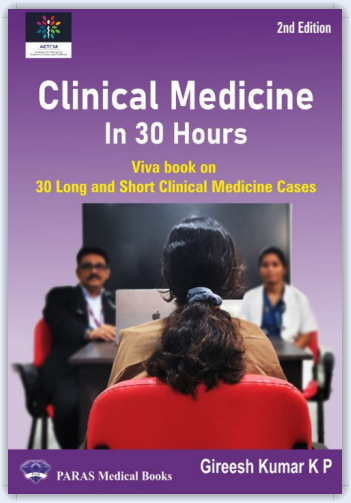
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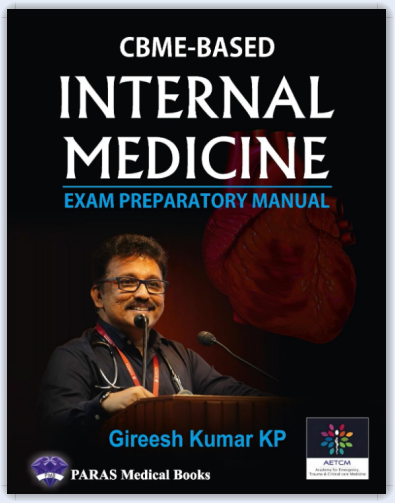
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