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TOPIC-WISE

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ANSWERS SOURCED FROM YOUMANS AND WINN NEUROLOGICAL SURGERY, 8th EDITION (2022)

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National Board of Examinations · Neurosurgery (Part-II / Final) · Papers I, II & III

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SOLVED Question Bank

All 350 questions answered · 2020 to 2026

VOLUME ONE

Chapters 1 – 10 · Anatomy, Physiology, Trauma, Tumours, Hydrocephalus,
Aneurysms

ANSWERS SOURCED FROM

Youmans and Winn Neurological Surgery, 8th Edition (2022)

Examination sessions covered	12
Question papers	35
Questions answered in full	350
Topic chapters	21
Distinct textbook pages cited	1429
Page citations in the text	8527

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About this Book

A Clinica Publication · DrNB / DNB Neurosurgery Exit Examination

The DrNB / DNB Neurosurgery exit examination rewards one thing above all: writing complete, structured answers to the questions the National Board actually asks. This book is built around that fact. It gathers **every theory question set between June 2020 and April 2026** — thirty-five papers, three hundred and fifty questions — and answers each one in full, the way it should be written in the answer script.

Questions are not left in paper order. They are grouped **topic-wise into twenty-one chapters**, so that a candidate revising aneurysms, shunts or epilepsy surgery sees every version of the question the examiners have set, side by side, with its session, paper and mark split.

Every answer is written from **Youmans and Winn Neurological Surgery, 8th Edition (2022)**, and each factual block carries the printed page it came from, so any point can be verified and read around in the source. Where the textbook is silent, the answer is still given, clearly flagged, with the source actually used.

The set is published in two volumes. This is **Volume One** (Chapters 1 – 10).

12

exam sessions

35

question papers

350

questions answered

21

topic chapters

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Answers reviewed for accuracy and examination relevance. Suggestions and corrections from readers are welcome through the publisher.

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Inside Volume One

Chapters 1 – 10 · Anatomy · Physiology · Trauma · Tumours · Hydrocephalus · Aneurysms

Chapters in this volume

01	Neuroanatomy & Applied Anatomy	49 questions
02	Neurophysiology, ICP & Cerebral Blood Flow	15 questions
03	Head Injury & Neurotrauma	22 questions
04	Craniovertebral Junction & Spinal Trauma	10 questions
05	Neuro-Oncology: Gliomas, Intra-axial Tumours & Neuropathology	15 questions
06	Skull Base, Meningioma & Orbital Lesions	15 questions
07	Sellar, Suprasellar & Pituitary Region	8 questions
08	Ventricular, Pineal & Posterior Fossa Lesions	10 questions
09	Hydrocephalus, Shunts & CSF Disorders	15 questions
10	Cerebrovascular I: Aneurysms & Subarachnoid Haemorrhage	14 questions
Total in this volume		173 questions

Also in this volume: examination pattern, papers covered and the repeat-frequency statistics.

What every answer gives you

Answered in full

All 350 questions, none skipped, written in the structure the paper asked for.

Textbook-cited

A Youmans & Winn 8e page reference on each factual block for quick verification.

Diagrams

Labelled diagram boxes wherever the question says “draw” or “label”.

Tables

Classify, compare, grade or differentiate — set out as tables to reproduce in the exam.

High-yield focus

Repeat-frequency statistics and the twelve themes to revise first.

Topic-wise

Every version of a recurring question grouped together with session, paper and marks.

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How to use this book

Every question asked in the DrNB / DNB Neurosurgery exit (Part-II, Final) theory examination between June 2020 and April 2026 is answered here — thirty-five papers, three hundred and fifty questions, none omitted.

The source rule

Answers are written from Youmans and Winn Neurological Surgery, 8th Edition (2022), and from nothing else. Each factual block carries the printed page it came from, in the form [YW p.1339]; an e-page of the online edition cites as [YW p.615.e6]. A grey strip at the foot of every answer lists that answer's pages together, so you can go straight to the book and read around the point.

Where the textbook does not cover something — Indian legislation such as the THOTA Act, the biographies of Indian neurosurgeons, a handful of eponymous scales — the answer is not skipped and not guessed. It carries a red NOT IN YOUMANS flag, names the source actually used, and is answered anyway. Twenty-two of the three hundred and fifty answers carry such a flag, most of them for one part only.

Where a current guideline is needed to complete an answer that the textbook leaves short, one tagged line is added, marked in red, for example [Guideline: BTF 4th ed., 2016]. This is deliberately rare. Where the textbook and another source disagree, the textbook is followed.

Reading an answer

Question block

The grey panel carries the question exactly as it was set, with its session, paper, question number and mark split.

Part headings

Purple bars mark each lettered part with its own marks, in the order the paper set them. Answer them in that order in the exam.

Tables

Anything the paper asks you to classify, compare, grade or differentiate is given as a table — write it as a table in the answer book.

DIAGRAM boxes

A gold box appears wherever the question says draw or label. It names every structure the examiner expects to see labelled. Draw it; do not describe it.

Citations

The small brown page numbers are for verification and further reading. Do not copy them into your answer script.

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Examination pattern & papers covered

Three theory papers, each of three hours and 100 marks, each with ten compulsory questions of 10 marks. Papers from August 2021 onward were delivered in the computer-based format with the answer script written offline.

SESSION	PAPERS	No. OF PAPERS	QUESTIONS
JUN 2020	Paper I, Paper II, Paper III	3	30
DEC 2020	Paper I, Paper II, Paper III	3	30
JUN 2021	Paper I, Paper II, Paper III	3	30
DEC 2021	Paper I, Paper II, Paper III	3	30
JUN 2022	Paper I, Paper II, Paper III	3	30
DEC 2022	Paper I, Paper II, Paper III	3	30
APR 2023	Paper I, Paper II, Paper III	3	30
OCT 2023	Paper I, Paper II, Paper III	3	30
MAY 2024	Paper I, Paper II, Paper III	3	30
JAN 2025	Paper I, Paper II, Paper III	3	30
OCT 2025	Paper I, Paper II, Paper III	3	30
APR 2026	Paper I, Paper II	2	20
TOTAL		35	350

April 2026 is represented by Papers I and II; Paper III of that session was not available at the time of compilation. Fifteen pages of the source question papers existed only as scanned images and were transcribed by hand.

STATISTICS

What the Board asks again and again — counted across twelve sessions, 2020 to 2026

A theme is counted once for every question in which it appears; a question can touch more than one theme. Any theme asked five times or more has appeared, on average, in one paper out of every seven.

Table 1 · High-yield repeated themes

THEME	TIMES ASKED	FREQUENCY	SESSIONS
Epilepsy Surgery & Drug-Resistant Epilepsy DEC 2020 · JUN 2021 · DEC 2021 · JUN 2022 · DEC 2022 · OCT 2023 · MAY 2024 · JAN 2025 · OCT 2025 · APR 2026	16		10 of 12
Intracranial Aneurysm — surgery & endovascular JUN 2020 · DEC 2020 · JUN 2021 · APR 2023 · OCT 2023 · MAY 2024 · JAN 2025 · OCT 2025 · APR 2026	11		9 of 12
Newer Antiepileptic Drugs & Phenytoin/Valproate DEC 2020 · JUN 2021 · DEC 2021 · JUN 2022 · DEC 2022 · OCT 2023 · MAY 2024 · JAN 2025 · APR 2026	10		9 of 12
Ventriculoperitoneal Shunt & Shunt Complications JUN 2020 · DEC 2020 · JUN 2021 · JUN 2022 · DEC 2022 · APR 2023 · OCT 2023 · JAN 2025	10		8 of 12
Arteriovenous Malformation (cerebral & spinal) JUN 2020 · JUN 2021 · DEC 2021 · JUN 2022 · APR 2023 · OCT 2023 · MAY 2024 · APR 2026	9		8 of 12
Carpal Tunnel & Entrapment Neuropathies DEC 2020 · JUN 2021 · DEC 2021 · DEC 2022 · OCT 2023 · JAN 2025 · OCT 2025 · APR 2026	9		8 of 12
Hydrocephalus — classification & management JUN 2020 · DEC 2020 · JUN 2021 · JUN 2022 · DEC 2022 · MAY 2024 · OCT 2025 · APR 2026	9		8 of 12
Skull Base Approaches (Kawase, Petrous Apex, Keyhole) JUN 2020 · JUN 2021 · DEC 2021 · JUN 2022 · DEC 2022 · MAY 2024 · OCT 2025	9		7 of 12
Craniosynostosis (syndromic & non-syndromic) DEC 2020 · JUN 2021 · DEC 2022 · OCT 2023 · MAY 2024 · JAN 2025 · APR 2026	8		7 of 12
Craniovertebral Junction Anomaly & Basilar Invagination JUN 2020 · JUN 2021 · DEC 2022 · APR 2023 · MAY 2024 · JAN 2025 · OCT 2025	8		7 of 12
Internuclear Ophthalmoplegia & Brainstem Syndromes APR 2023 · OCT 2023 · MAY 2024 · OCT 2025 · APR 2026	8		5 of 12
Optic Nerve, Chiasma & Visual Field Defects JUN 2020 · JUN 2021 · APR 2023 · OCT 2023 · OCT 2025 · APR 2026	8		6 of 12
Research Methodology, Evidence & Trials JUN 2020 · DEC 2020 · JUN 2021 · DEC 2021 · JUN 2022 · DEC 2022 · APR 2023 · APR 2026	8		8 of 12
Stereotactic Radiosurgery, Gamma Knife & CyberKnife JUN 2021 · DEC 2021 · JUN 2022 · DEC 2022 · APR 2023 · MAY 2024 · JAN 2025	8		7 of 12
Brachial Plexus Injury & Neurotisation JUN 2020 · DEC 2021 · JUN 2022 · OCT 2023 · OCT 2025 · APR 2026	7		6 of 12
Brain Death, Apnea Test & THOTA Act JUN 2022 · APR 2023 · OCT 2023 · MAY 2024	7		4 of 12
Cerebral Blood Flow & Autoregulation DEC 2020 · JUN 2021 · APR 2023 · OCT 2023 · MAY 2024 · APR 2026	7		6 of 12
Encephalocele & Neural Tube Defects DEC 2020 · JUN 2021 · JUN 2022 · OCT 2023 · MAY 2024 · OCT 2025 · APR 2026	7		7 of 12

THEME	TIMES ASKED	FREQUENCY	SESSIONS
ICP Monitoring & Intracranial Pressure JUN 2020 · DEC 2020 · JUN 2022 · APR 2023 · OCT 2023 · MAY 2024 · APR 2026	7		7 of 12
Intramedullary & Intradural Spinal Tumours JUN 2021 · DEC 2021 · DEC 2022 · APR 2023 · OCT 2023 · APR 2026	7		6 of 12
WHO Classification of Gliomas / Recurrent GBM JUN 2020 · DEC 2020 · JUN 2021 · APR 2023 · OCT 2023 · OCT 2025 · APR 2026	7		7 of 12
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History & Contributions to Neurosurgery JUN 2020 · JUN 2022 · OCT 2023 · JAN 2025 · OCT 2025 · APR 2026	6		6 of 12
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Intra-operative Neurophysiological Monitoring (IONM) JUN 2020 · DEC 2021 · APR 2023 · OCT 2023 · OCT 2025 · APR 2026	6		6 of 12
Nanotechnology, Stem Cells & Emerging Therapies JUN 2020 · JUN 2021 · JUN 2022 · DEC 2022 · OCT 2023 · JAN 2025	6		6 of 12
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Chronic & Acute Subdural Haematoma JUN 2021 · DEC 2021 · APR 2023 · MAY 2024	5		4 of 12
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Sodium & Electrolyte Disturbance (SIADH / CSW / DI) DEC 2020 · JUN 2022 · OCT 2023 · JAN 2025 · APR 2026	5		5 of 12
Split Cord Malformation / Diastematomyelia DEC 2021 · JUN 2022 · DEC 2022 · JAN 2025 · OCT 2025	5		5 of 12
Subarachnoid Haemorrhage — grading & management JUN 2020 · JUN 2021 · DEC 2021 · DEC 2022	5		4 of 12

Table 3 · The twelve themes to revise first

01	Epilepsy Surgery & Drug-Resistant Epilepsy	asked 16 times
02	Intracranial Aneurysm — surgery & endovascular	asked 11 times
03	Newer Antiepileptic Drugs & Phenytoin/Valproate	asked 10 times
04	Ventriculoperitoneal Shunt & Shunt Complications	asked 10 times
05	Arteriovenous Malformation (cerebral & spinal)	asked 9 times
06	Carpal Tunnel & Entrapment Neuropathies	asked 9 times
07	Hydrocephalus — classification & management	asked 9 times
08	Skull Base Approaches (Kawase, Petrous Apex, Keyhole)	asked 9 times
09	Craniosynostosis (syndromic & non-syndromic)	asked 8 times
10	Craniovertebral Junction Anomaly & Basilar Invagination	asked 8 times
11	Internuclear Ophthalmoplegia & Brainstem Syndromes	asked 8 times
12	Optic Nerve, Chiasma & Visual Field Defects	asked 8 times

CHAPTER 1

Neuroanatomy & Applied Anatomy

49

QUESTIONS SOLVED

Internuclear Ophthalmoplegia & Brainstem Syndromes

6 questions

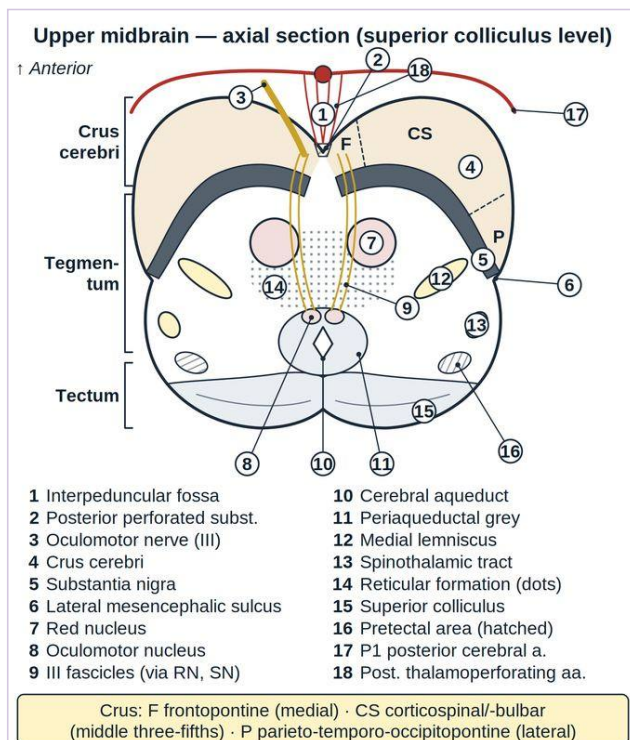
Q A1

APR 2023 · Paper III · Q2

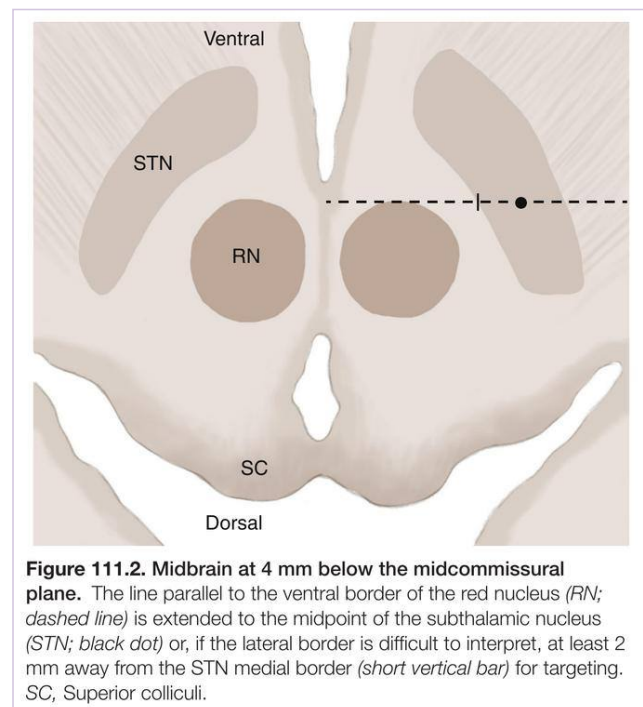
- a) Draw a labelled diagram of transverse section of the upper midbrain. [4]
 b) Parinaud's syndrome and its clinical correlates. [4]
 c) Aqueductoplasty. [2]

ANSWER

(a) Labelled diagram — transverse section of the upper midbrain [4]



Schematic diagram — Upper midbrain — axial section (superior colliculus level)



Original figure: Youmans & Winn 8e, Fig. 111.2, p. 734

- The midbrain is divided by a midline sagittal plane into two cerebral peduncles; each peduncle has an anterior part (crus cerebri or basis pedunculi), an intermediate part (tegmentum) and a posterior part behind the aqueduct (tectum). The substantia nigra and the lateral mesencephalic sulcus separate the crus cerebri from the tegmentum. **YW p.35**
- The oculomotor nerves emerge from the medial side of the crura cerebri in the interpeduncular fossa; the posterior surface of the midbrain shows the superior and inferior colliculi (quadrigeminal plate), and the trochlear nerve exits below the inferior colliculus. **YW p.35**
- The basal view shows, from front to back, the crus cerebri, posterior perforated substance, substantia nigra, tegmentum and tectum. **YW p.24**

- The oculomotor nucleus lies in the periaqueductal grey matter at the level of the superior colliculus; its fascicles pass through the decussating superior cerebellar peduncle, the substantia nigra and the medial cerebral peduncle. **YW p.200.e19**
- The posterior thalamoperforating arteries arise from P1, enter through the posterior perforated substance, interpeduncular fossa and medial crus cerebri, and supply the substantia nigra, red nucleus, oculomotor and trochlear nuclei, mesencephalic reticular formation and pretectum. **YW p.27**

(b) Parinaud's syndrome and its clinical correlates [4]

Definition

Parinaud (dorsal midbrain) syndrome is a constellation of signs produced by a lesion of the dorsal midbrain / posterior commissure region. **YW p.200.e23**

Clinical features

1. **Supranuclear up-gaze palsy** — from involvement of projections from the vertical gaze centres travelling in the posterior commissure. **YW p.200.e23**
2. **Convergence-retraction nystagmus**, often elicited by attempted up-gaze. **YW p.200.e23**
3. **Collier sign** — upper eyelid retraction. **YW p.200.e23**
4. **Pupillary light-near dissociation** — the near response is better than the light response, as also seen in Adie pupil, blind eyes and neurosyphilis (Argyll Robertson pupil). **YW p.200.e23, p.200.e26**

Localising correlates

- A **complete** supranuclear vertical gaze palsy implies a more rostral intrinsic midbrain lesion involving the **rostral interstitial nuclei of the MLF**; causes include bilateral paramedian midbrain-thalamic stroke from top-of-the-basilar occlusion or artery of Percheron occlusion, progressive supranuclear palsy, Niemann-Pick type C and intrinsic tumours. **YW p.200.e23**
- Common causes of the dorsal midbrain syndrome are **stroke, pineal gland tumours, intrinsic brainstem tumours and hydrocephalus**. **YW p.200.e23** Pineal region tumours reach the posterior commissure directly because the pineal gland lies with the posterior commissure ventrally and the habenular commissure dorsally. **YW p.1147** Tectal glioma classically presents with obstructive hydrocephalus at the aqueduct. **YW p.1826**
- It is also a recognised **iatrogenic** complication of endoscopic aqueductoplasty, along with midbrain injury and cranial nerve palsies. **YW p.1733**

(c) Aqueductoplasty [2]

- **Technique:** the bur hole is placed more anteriorly than for a standard ETV. A Fogarty balloon catheter, its tip shaped with a gentle curve (the aqueduct is not straight), is slid into the aqueductal lumen and the balloon carefully dilated; failure to respect the curve risks injury to the tectal plate. A flexible endoscope helps perforate a membranous obstruction, especially a distal one. **YW p.1733**
- **Stenting** is added for patients at high risk of restenosis or with a trapped fourth ventricle; shunted patients with a trapped fourth ventricle and slit lateral ventricles are better served by a suboccipital retrograde aqueductoplasty with stenting. **YW p.1733**
- **Results:** short-term success exceeds 66% in most series, but restenosis is the major problem — a radiographic restenosis rate of 88% at 10 years with 65% needing re-treatment by ETV. Reported complications, especially with long-segment stenoses, are midbrain injury, Parinaud syndrome and cranial nerve palsies. **ETV remains the preferred endoscopic CSF diversion.** **YW p.1733**

Youmans & Winn 8e — pp. 35, 24, 200.e19, 27, 200.e23, 200.e26, 1147, 1826, 1733

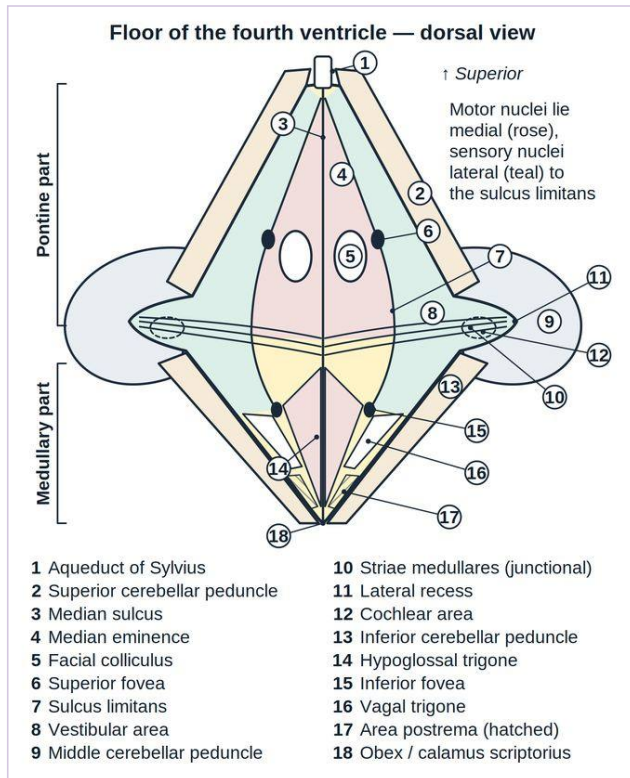
Q A2

OCT 2023 · Paper I · Q4

- a) Draw neat labelled diagram of floor of th fourth Ventricle. [4]
 b) Internuclear ophthalmoplegia. [3]
 c) Draw neat labelled diagram depicting deep venous system of brain. [3]

ANSWER

(a) Neat labelled diagram — floor of the fourth ventricle [4]



Schematic diagram — Floor of the fourth ventricle — dorsal view

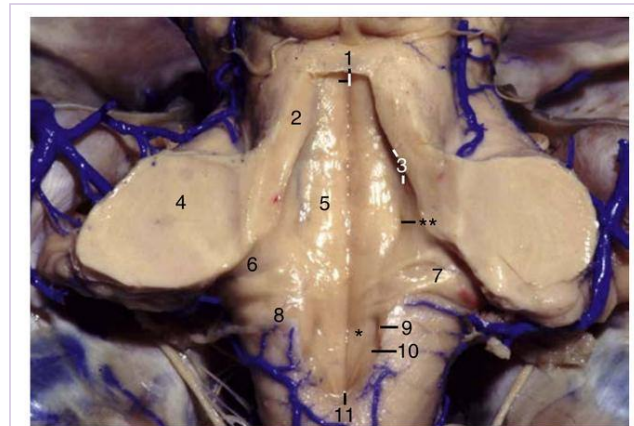


Figure 2.53. Floor of fourth ventricle, posterior view. 1, Median sulcus and median eminence; 2, superior cerebellar peduncle; 3, sulcus limitans and vestibular area; 4, middle cerebellar peduncle; 5, facial colliculus; 6, cochlear area; 7, striae medullary; 8, inferior cerebellar peduncle; 9, inferior fovea; 10, vagal trigone; 11, obex and area postrema; *, hypoglossal trigone; **, superior fovea.

Original figure: Youmans & Winn 8e, Fig. 2.53, p. 39

- The floor of the fourth ventricle is rhomboid. The junctional part, characterised by the medullary striae extending into the lateral recesses, divides it into the larger superior **pontine part** (apex at the aqueduct) and the smaller inferior **medullary part** (apex at the obex); the median sulcus divides it longitudinally. **YW p.39**
- The sulcus limitans divides each half into the raised **median eminence** and the lateral **area vestibularis**; **motor nuclei lie medial and sensory nuclei lateral to the sulcus limitans**. **YW p.39**
- The pontine part carries the two **facial colliculi** on the median eminence, limited laterally by the **superior fovea**. The medullary part has the feather/pen-nib configuration called the **calamus scriptorius**, with the hypoglossal and vagal trigones and the area postrema; the **inferior fovea** lies just lateral to the hypoglossal trigone. **YW p.39**
- The floor is formed by the pons and medulla; the roof is formed above by the superior cerebellar peduncles, superior medullary velum and lingula, and below by the inferior medullary velum, tela choroidea, choroid plexus, uvula and nodule, converging at the **fastigium**. The two open lateral walls are the lateral recesses communicating with the CP angle through the foramen of Luschka. **YW p.36, p.37**

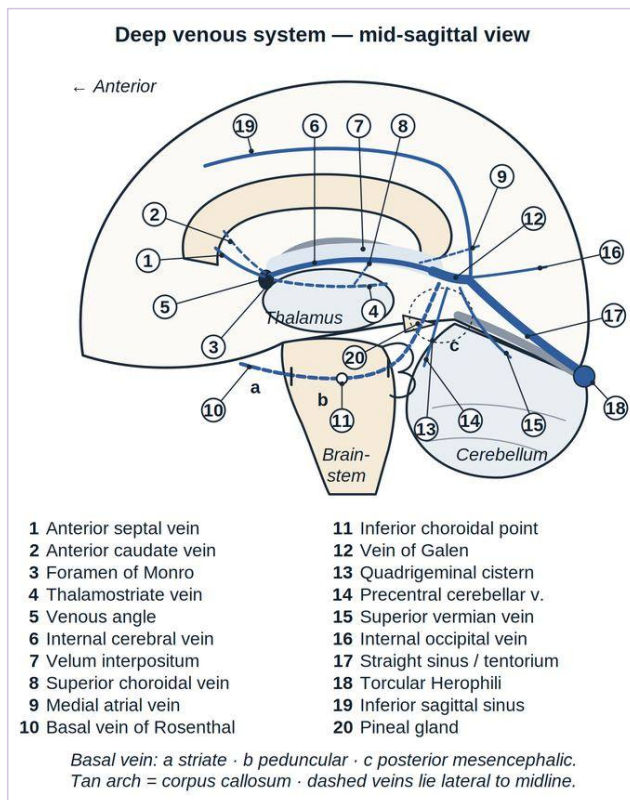
(b) Internuclear ophthalmoplegia [3]

- Mechanism.** INO results from a lesion of the **medial longitudinal fasciculus (MLF)** in the midbrain or pons. The MLF carries the eye-movement command from the sixth nerve nucleus to the **contralateral** third nerve nucleus, so that the ipsilateral lateral rectus and contralateral medial rectus contract together for conjugate

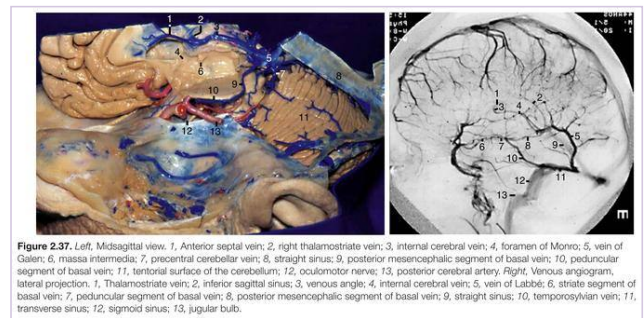
horizontal gaze. **YW p.200.e23**

- **Side of the lesion.** Lesions of the MLF virtually always lie after the immediate decussation of the fibres arising in the sixth nerve nucleus, so the predominant clinical sign — impaired adduction — is **ipsilateral** to the lesion. **YW p.200.e24**
- **Classic signs.** Impaired adduction of the eye ipsilateral to the lesion with **dissociated (abducting) nystagmus of the contralateral eye**. In a subtle INO, ocular movements may look full and the only clue is **adduction lag** — slowing of the adducting eye relative to the abducting eye on horizontal saccades. **YW p.200.e24**
- **Differentiation.** The adduction deficit of INO can usually be **overcome by convergence**, whereas adduction deficits from third nerve palsy or ocular myasthenia cannot. **YW p.200.e24**
- **Causes.** Multiple sclerosis, brainstem stroke, brainstem tumour and fourth ventricular tumour; with stroke the lesion may be tiny and easily missed on imaging. **YW p.200.e24**

(c) Neat labelled diagram — deep venous system of the brain [3]



Schematic diagram — Deep venous system — mid-sagittal view



Original figure: Youmans & Winn 8e, Fig. 2.37, p. 30

- The deep venous system drains four-fifths of the thickness of the cerebrum; the superficial system drains only the outer one-fifth. **YW p.22**
- The velum interpositum, the third layer of the roof of the third ventricle, contains the internal cerebral veins and the medial posterior choroidal arteries. **YW p.20**
- The basal vein of Rosenthal begins below the anterior perforated substance and has striate, peduncular and posterior mesencephalic segments, the last usually draining into the vein of Galen. **YW p.29**
- On the mid-sagittal view the chain is: anterior septal vein and thalamostriate vein → internal cerebral vein at the foramen of Monro → vein of Galen → straight sinus, with the basal vein joining the vein of Galen posteriorly. **YW p.30**
- Lateral ventricular veins drain to either the internal cerebral veins (anterior septal, caudate, thalamostriate, superior choroidal, medial atrial) or the basal vein of Rosenthal (inferior choroidal, inferior ventricular, lateral atrial, amygdalar and hippocampal veins). **YW p.1207**

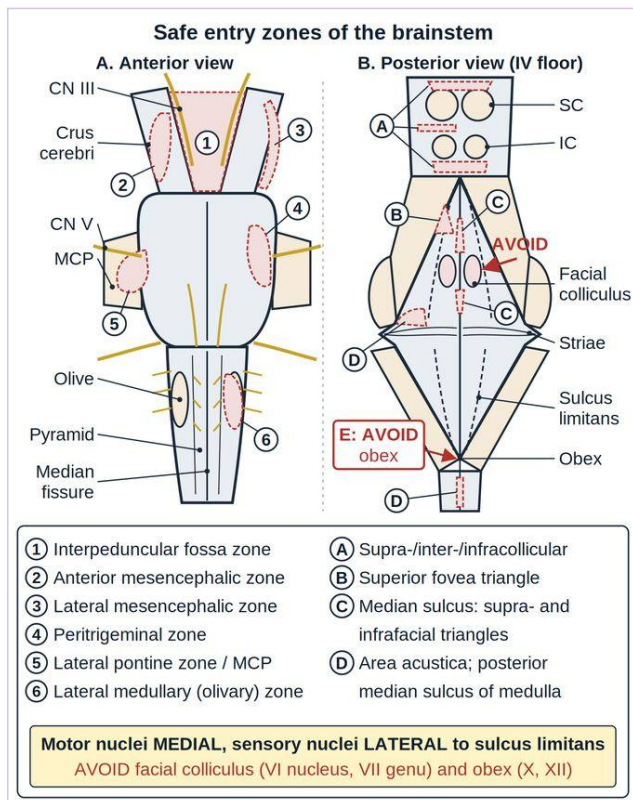
Q A3

OCT 2023 · Paper II · Q3

- a) Draw neat labelled diagrams of safe entry zones of brainstem. [5]
 b) Brain stem auditory evoked potential monitoring. [5]

ANSWER

(a) Labelled diagrams — safe entry zones of the brainstem [5]



Schematic diagram — Safe entry zones of the brainstem

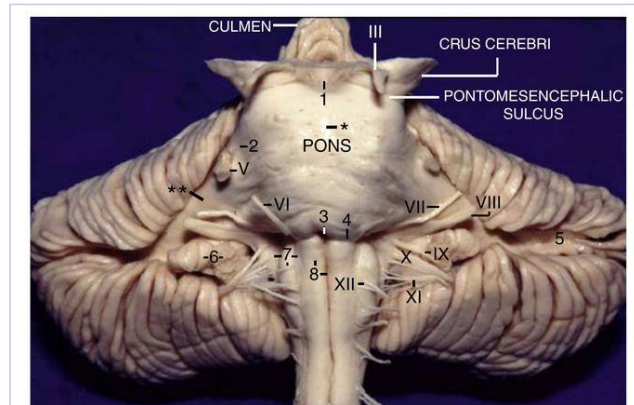


Figure 2.47. Frontal view. 1, Interpeduncular fossa; 2, lateral pontine sulcus; 3, inferior foramen caecum; 4, pontomedullary sulcus; 5, great horizontal or petrosal fissure; 6, flocculus and choroid plexus protruding from foramen of Luschka; 7, supraolivary fossa, olive, and anterolateral sulcus; 8, pyramid and anterior median fissure; *, basilar sulcus; **, middle cerebellar peduncle.

Original figure: Youmans & Winn 8e, Fig. 2.47, p. 36

- For brainstem cavernomas, multiple safe entry sites have been described that avoid critical nuclei and traversing white matter tracts, including the **epitrigeminal, superior fovea triangle, interpeduncular fossa, anterior and lateral mesencephalic, inter-, supra- and infracollicular, and lateral pontine and medullary zones.** YW p.3773
- Entry selection is planned with the **two-point method**: one point in the centre of the lesion, a second where the lesion comes closest to the pial surface; the line joining them determines the approach. The rule has exceptions — pontine lesions are often closest to the ventral surface but are routinely approached through the **middle cerebellar peduncle** to spare the motor pathway. YW p.3772, p.3773
- Once an entry point is chosen the pia is opened sharply and blunt-tipped forceps are used to spread **in the direction of the fibre tracts.** YW p.3773
- The anatomic basis is that motor cranial nerve nuclei lie medial and sensory nuclei lateral to the sulcus limitans on the floor of the fourth ventricle, with the facial colliculi on the median eminence. YW p.39 CNs VI, VII and VIII leave at the superior border of the supraolivary fossa and CNs IX, X and XI along the inferior cerebellar peduncle. YW p.36
- CN VI and VII are especially vulnerable because their nuclei and tracts lie superficially, close to the dorsal surface and the floor of the fourth ventricle; the area around the **obex must be avoided** because of the nuclei of CN X and XII. YW p.1827

- Choosing a safe entry point is the single most critical step in brainstem surgery, and neuronavigation with intraoperative ultrasound is mandatory for its selection. **YW p.1825**

(b) Brainstem auditory evoked potential monitoring [5]

Generators of the waveform

Wave	Presumed generator
I	Acoustic (cochlear) nerve
II	Cochlear nuclei
III	Superior olives
IV	Lateral lemniscus tracts and nuclei
V	Inferior colliculi
VI, VII	Medial geniculate body; auditory radiations to temporal lobe (not clinically useful)

YW p.1284

Principles and interpretation

- Wave I reflects the integrity of the **cochlea and CN VIII**; waves III and V evaluate more **central auditory structures** as surgery progresses. **YW p.201.e9**
- A retrocochlear (space-occupying) lesion **after** the first-order neurons of CN VIII leaves wave I normal but delays or abolishes all subsequent waves; a lesion of the first-order neurons themselves may abolish replicable waveforms altogether. The vestibular schwannoma pattern is poor replicability of waves III and V with increased **I-III and I-V interwave latencies**. **YW p.201.e9**
- The ABR is used preoperatively in suspected CN VIII and low-brainstem disorders, intraoperatively to monitor auditory system integrity, and immediately postoperatively as objective evidence of peripheral auditory integrity before closure. **YW p.201.e9**

Neurosurgical use and limitations

- Patients with **brainstem cavernomas** are routinely monitored with brainstem auditory evoked responses and facial nerve monitoring in addition to motor and somatosensory evoked potentials. **YW p.3772**
- Limitations:** BAERs require averaging over multiple stimulations, so a waveform change may reflect manipulation that occurred seconds to minutes earlier; with large tumours the baseline BAERs are frequently abnormal or absent, precluding their use. **YW p.1284**
- Postoperative deficits are **not always heralded** by a change in monitored potentials — both false-negative and false-positive potentials occur — but any change from baseline requires intraoperative consideration. **YW p.3772**
- Direct cochlear nerve action potential (CNAP)** monitoring, recorded from the nerve proximal to the tumour, gives two negative peaks (N1, N2); change in N1 latency or amplitude suggests neural injury. It responds faster than BAERs, but needs adequate exposure of normal proximal nerve and a bulky electrode, so it is impractical in the middle fossa approach. **YW p.1284**
- Intraoperative monitoring of CN VIII does not achieve the same favourable outcomes as facial nerve monitoring. **YW p.1283, p.1284**

Youmans & Winn 8e — pp. 3773, 3772, 39, 36, 1827, 1825, 1284, 201.e9, 1283

Q A4

MAY 2024 · Paper I · Q4

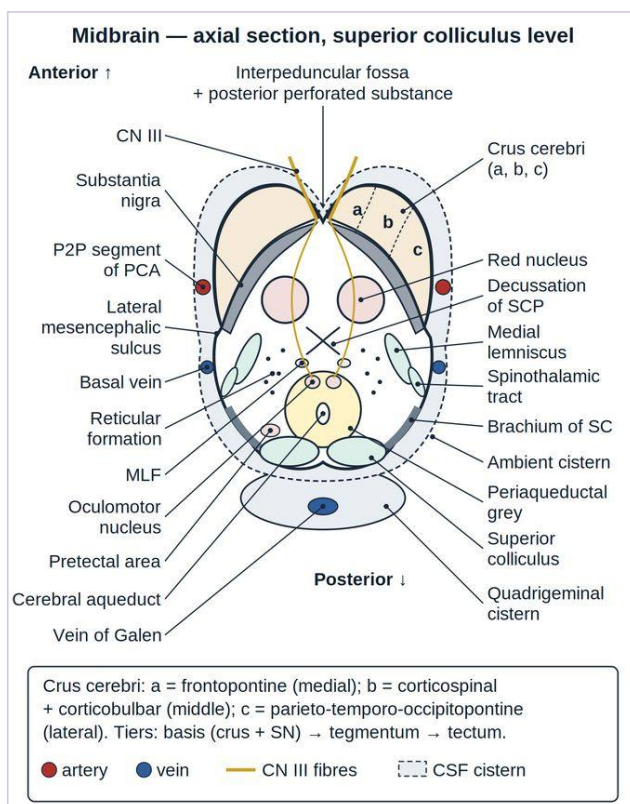
- Internuclear ophthalmoplegia. [3]
- Draw axial section of midbrain at level of tectum. [4]
- Clinical features of intrinsic brain stem lesions. [3]

ANSWER

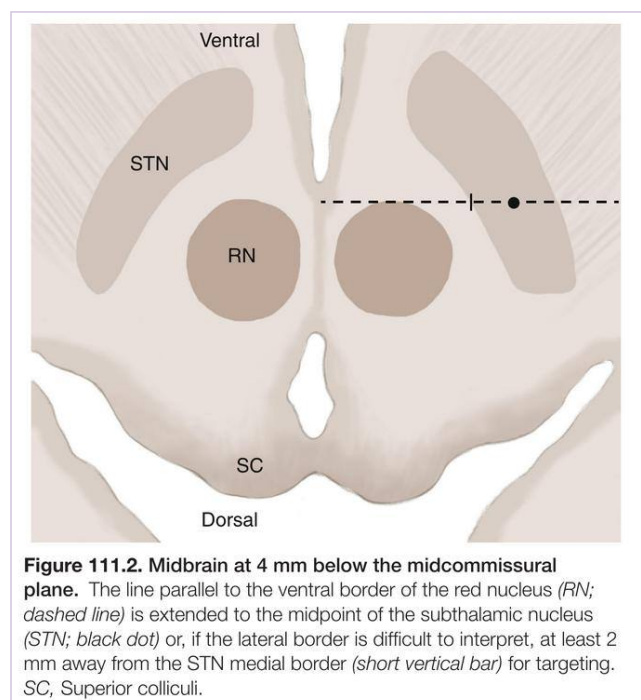
(a) Internuclear ophthalmoplegia [3]

- Lesion:** the medial longitudinal fasciculus (MLF) in the midbrain or pons. The MLF conveys the horizontal gaze command from the abducens nucleus to the **contralateral** oculomotor nucleus, yoking the ipsilateral lateral rectus to the contralateral medial rectus. **YW p.200.e23**
- Signs:** impaired **adduction** of the eye **ipsilateral** to the lesion with **dissociated nystagmus of the contralateral abducting eye**. Because MLF lesions almost always lie after the immediate decussation of the fibres from the sixth nerve nucleus, the adduction deficit is ipsilateral to the lesion. **YW p.200.e24**
- Subtle INO:** eye movements may look full; **adduction lag** (slowing of the adducting eye compared with the abducting eye on horizontal saccades) may be the only clue. **YW p.200.e24**
- Key differentiating sign:** the adduction deficit of INO is usually **overcome by convergence**, unlike a third nerve palsy or ocular myasthenia. **YW p.200.e24**
- Causes:** multiple sclerosis, brainstem stroke (the lesion may be very small and easily overlooked on imaging), brainstem tumour and fourth ventricular tumour. **YW p.200.e24** INO is also seen with craniovertebral junction anomalies, where it may lead to a mistaken diagnosis of mesencephalic or upper pontine disease. **YW p.1961**

(b) Axial section of the midbrain at the level of the tectum [4]



Schematic diagram — Midbrain — axial section, superior colliculus level



Original figure: Youmans & Winn 8e, Fig. 111.2, p. 734

- Each cerebral peduncle is divided into crus cerebri, tegmentum and tectum; the substantia nigra and lateral mesencephalic sulcus separate crus from tegmentum, and CN III emerges from the medial crus in the

interpeduncular fossa. **YW p.35** The basal view demonstrates crus cerebri, posterior perforated substance, substantia nigra, tegmentum and tectum in sequence. **YW p.24**

- The oculomotor nucleus sits in the periaqueductal grey at the level of the superior colliculus; there is a **single midline levator subnucleus** and the **superior rectus subnucleus is contralateral**. **YW p.200.e19**
- The P2P segment runs lateral to the tegmentum in the ambient cistern, parallel and inferior to the basal vein, and the posterior thalamoperforating arteries supply the substantia nigra, red nucleus, oculomotor and trochlear nuclei, mesencephalic reticular formation and pretectum. **YW p.27**

(c) Clinical features of intrinsic brainstem lesions [3]

General pattern

Symptoms correspond to the level of the tumour; intrinsic lesions typically produce **cranial nerve palsies with crossed long-tract signs** (contralateral hemiparesis or sensory loss), often after months to years of minor symptoms. Neck pain and stiffness, cranial nerve palsies, contralateral hemiparesis and hemichorea are commonly seen. **YW p.1825**

By level

Level	Features
Rostral/dorsal midbrain (tectal)	Obstructive hydrocephalus at the aqueduct; dorsal midbrain (Parinaud) syndrome — supranuclear up-gaze palsy, convergence-retraction nystagmus, Collier sign, light-near dissociation YW p.1825, p.200.e23
Midbrain fascicular CN III	Claude syndrome (superior cerebellar peduncle — ataxia); Benedikt syndrome (substantia nigra — contralateral parkinsonism/tremor); Weber syndrome (cerebral peduncle — contralateral hemiparesis) YW p.200.e19
Pons	INO from MLF involvement; CN VI and VII palsies — their nuclei and tracts lie superficially close to the floor of the fourth ventricle YW p.200.e24, p.1827
Dorsally exophytic / fourth ventricle	Hydrocephalus; torticollis in older children from chronic tonsillar herniation YW p.1825
Medulla	Dysphagia, apnoea; injury near the obex affects CN X and XII nuclei YW p.1825, p.1827
Cervicomedullary	Upper and/or lower motor neuron signs YW p.1825

Localising rule

Motor cranial nerve nuclei lie medial and sensory nuclei lateral to the sulcus limitans in the floor of the fourth ventricle. **YW p.39** CNs VI, VII and VIII emerge at the pontomedullary sulcus/supraolivary fossette, and CNs IX, X and XI from the postolivary sulcus, so their palsies localise the lesion rostrocaudally. **YW p.36**

High-yield: an isolated INO plus a tiny "normal-looking" MRI in a young adult is MS until proved otherwise; in an older patient it is a paramedian pontine perforator stroke. **YW p.200.e24**

Youmans & Winn 8e — pp. 200.e23, 200.e24, 1961, 35, 24, 200.e19, 27, 1825, 1827, 39, 36

Q A5

OCT 2025 · Paper I · Q2

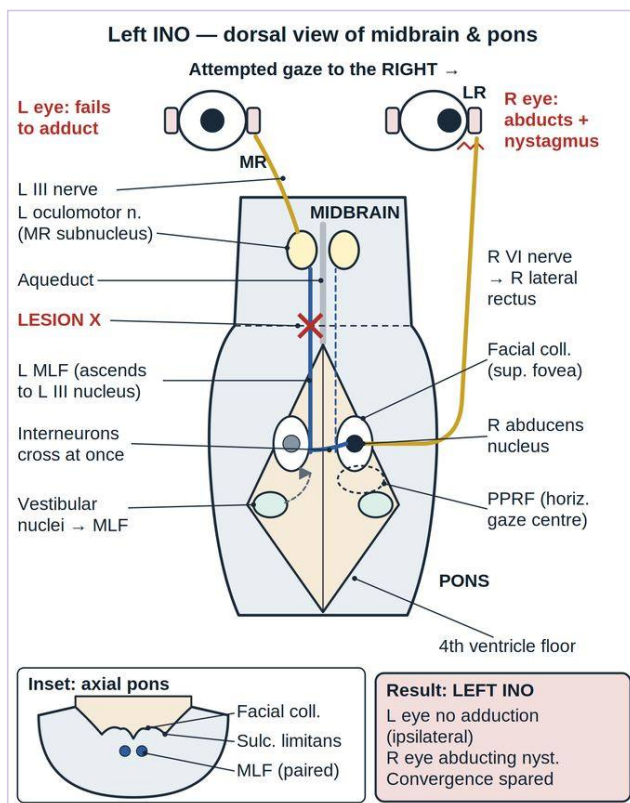
Internuclear ophthalmoplegia:

- Provide anatomical localization with diagram. [5]**
- Clinical examination technique. [3]**
- Discuss associated syndromes. [2]**

ANSWER

(a) Anatomical localisation with diagram [5]**The pathway**

- The **MLF** conveys the eye-movement command from the **sixth nerve nucleus** in the pons to the **contralateral third nerve nucleus** in the midbrain, so that the ipsilateral lateral rectus and the contralateral medial rectus contract simultaneously and the eyes move conjugately in the horizontal plane. **YW p.200.e23**
- The MLF also connects the **vestibular nuclei** (superior/Bechterew, lateral/Deiters, medial/Schwalbe, inferior/Roller — all in the floor of the fourth ventricle) to all the other cranial nerve nuclei, and in particular to CN III, IV, VI and X. **YW p.201.e11**
- Lesions virtually always lie **after the immediate decussation** of the fibres arising in the sixth nerve nucleus, so the adduction deficit is **ipsilateral** to the lesion. **YW p.200.e24**



Schematic diagram — Left INO — dorsal view of midbrain & pons

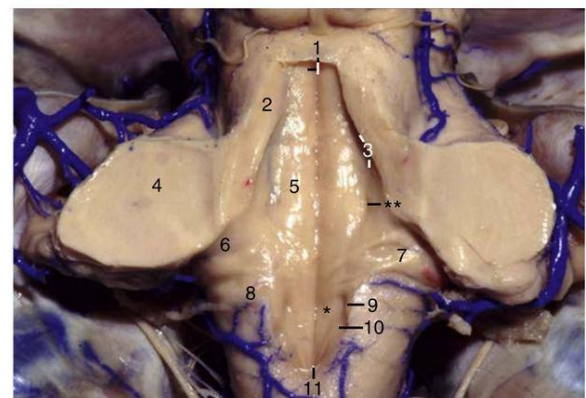


Figure 2.53. Floor of fourth ventricle, posterior view. 1, Median sulcus and median eminence; 2, superior cerebellar peduncle; 3, sulcus limitans and vestibular area; 4, middle cerebellar peduncle; 5, facial colliculus; 6, cochlear area; 7, striae medullary; 8, inferior cerebellar peduncle; 9, inferior fovea; 10, vagal trigone; 11, obex and area postrema; *, hypoglossal trigone; **, superior fovea.

Original figure: Youmans & Winn 8e, Fig. 2.53, p. 39

(b) Clinical examination technique [3]

- Test versions across the full range.** The range of extraocular movement is observed as the patient follows the examiner's fingertip on a prescribed course testing each extraocular muscle. Note whether diplopia is horizontal, vertical or oblique and in which gaze position it is worst — the misalignment is greatest in the field of action of the weak muscle. **YW p.200.e16**
- Look for the classic pair of signs:** impaired adduction of the eye ipsilateral to the lesion and **dissociated**

nystagmus of the contralateral abducting eye. YW p.200.e24

3. **Test horizontal saccades, not just pursuit.** In a subtle INO the versions may appear full; ask the patient to make rapid refixations between two targets and watch for **adduction lag** — the adducting eye moving more slowly than the abducting eye. YW p.200.e24
4. **Test convergence.** Bring a near target to the nose: the adduction deficit of INO is usually **overcome by convergence**, whereas the adduction deficit of a third nerve palsy or ocular myasthenia is not — this single manoeuvre separates INO from its mimics. YW p.200.e24
5. **Exclude mimics.** Confirm the diplopia is **binocular** (resolves on covering either eye); check for fatigable ptosis and orbicularis weakness of ocular myasthenia, which can mimic INO. YW p.200.e16, p.200.e19
6. **Complete the examination** for associated brainstem and cerebellar signs — skew deviation, downbeat and gaze-evoked nystagmus, saccadic dysmetria and cogwheel pursuit. YW p.200.e24

(c) Associated syndromes [2]

- **Multiple sclerosis** — the commonest cause, classically bilateral INO in a young adult. YW p.200.e24
- **Brainstem stroke** — a paramedian perforator infarct; the causative lesion can be very small and easily overlooked on neuroimaging. YW p.200.e24
- **Brainstem tumour and fourth ventricular tumour**, from direct involvement of the paramedian tegmentum. YW p.200.e24
- **Craniovertebral junction anomalies** — INO is not uncommon and leads to misdiagnosis of a mesencephalic or upper pontine disturbance; downbeat nystagmus accompanies strictly compressive lesions at the craniovertebral border with or without Chiari malformation. YW p.1961, p.200.e24
- **Cerebellar and posterior fossa disease** may coexist, adding downbeat nystagmus, saccadic dysmetria, cogwheel pursuit and **alternating skew deviation** that changes direction with lateral gaze. YW p.200.e24
- **Wernicke encephalopathy** — confusion, ophthalmoplegia and ataxia, with T2/FLAIR change in the medial thalami, periaqueductal grey and floor of the fourth ventricle. YW p.200.e23
- **Ocular myasthenia gravis** is the great mimic and must be excluded rather than "associated". YW p.200.e19

Youmans & Winn 8e — pp. 200.e23, 201.e11, 200.e24, 39, 200.e16, 200.e19, 1961

Q A6

APR 2026 · Paper I · Q3

- a) Surgical anatomy of MLF. [5]
- b) Draw coronal section of midbrain at superior colliculus level. [5]

ANSWER

(a) Surgical anatomy of the MLF [5]

Course and position

- The MLF is a paired, compact, **paramedian longitudinal bundle lying immediately ventral to the floor of the fourth ventricle and to the cerebral aqueduct**, running the length of the brainstem from the midbrain to the medulla and into the cervical cord. Its surface marker in the pons is the **median eminence and the facial colliculus**, immediately lateral to the median sulcus; the **sulcus limitans** lies lateral to it. YW p.39
- In the midbrain it lies in the tegmentum ventral to the periaqueductal grey, alongside the oculomotor nucleus; the **rostral interstitial nucleus of the MLF** at the rostral midbrain is the vertical gaze generator, and its bilateral destruction gives a complete supranuclear vertical gaze palsy. YW p.200.e23

Connections

- Conveys the horizontal gaze command from the **abducens nucleus** to the **contralateral oculomotor nucleus (medial rectus subnucleus)**, yoking the ipsilateral lateral rectus to the contralateral medial rectus for

conjugate horizontal gaze. Its fibres **decussate immediately** on leaving the sixth nerve nucleus. **YW**

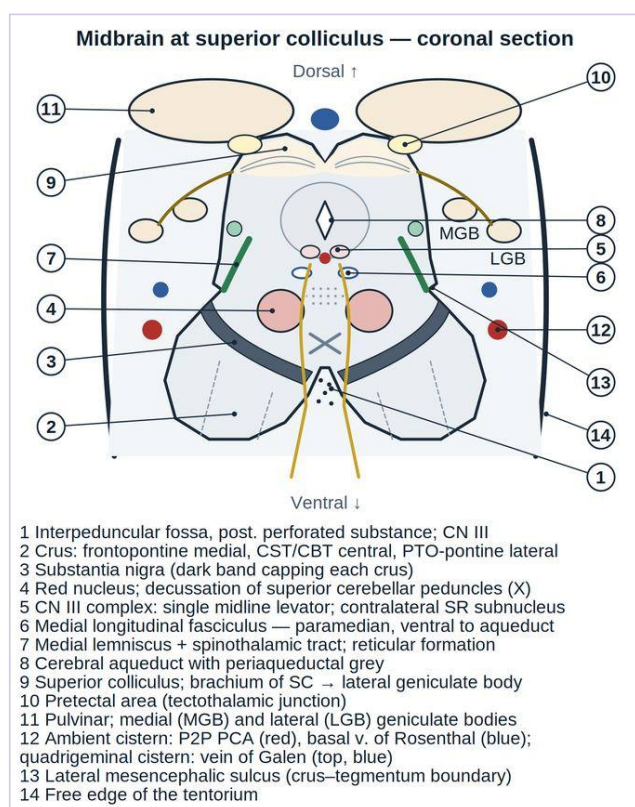
p.200.e23, p.200.e24

- Connects the **vestibular nuclei** (superior/Bechterew, lateral/Deiters, medial/Schwalbe, inferior/Roller, all in the floor of the fourth ventricle) with all the other cranial nerve nuclei, particularly CN III, IV, VI and X. **YW**
p.201.e11

Surgical relevance

- The MLF is the **medial boundary of the superior fovea triangle**, one of the recognised safe entry zones into the dorsal pons; entry zones are chosen precisely to avoid the MLF, the critical nuclei and the traversing white matter tracts. **YW p.3773**
- The **facial colliculus must be identified and avoided** during work on the floor of the fourth ventricle; CN VI and VII are particularly vulnerable because their nuclei and tracts lie superficially close to the floor. **YW**
p.3772 via p.3773 planning, YW p.1827
- Any paramedian dorsal brainstem dissection — telovelar exposure, fourth ventricular tumour removal, dorsal midbrain surgery, aqueductoplasty — risks the MLF; the resulting deficit is INO, ipsilateral adduction failure with contralateral abducting nystagmus, convergence preserved. **YW p.200.e24**
- Because the MLF lies paramedian, **midline splitting of the pontine floor is not safe**; entry must respect the rule that motor nuclei lie medial and sensory nuclei lateral to the sulcus limitans. **YW p.39**

(b) Coronal section of the midbrain at the superior colliculus level [5]



Schematic diagram — Midbrain at superior colliculus — coronal section

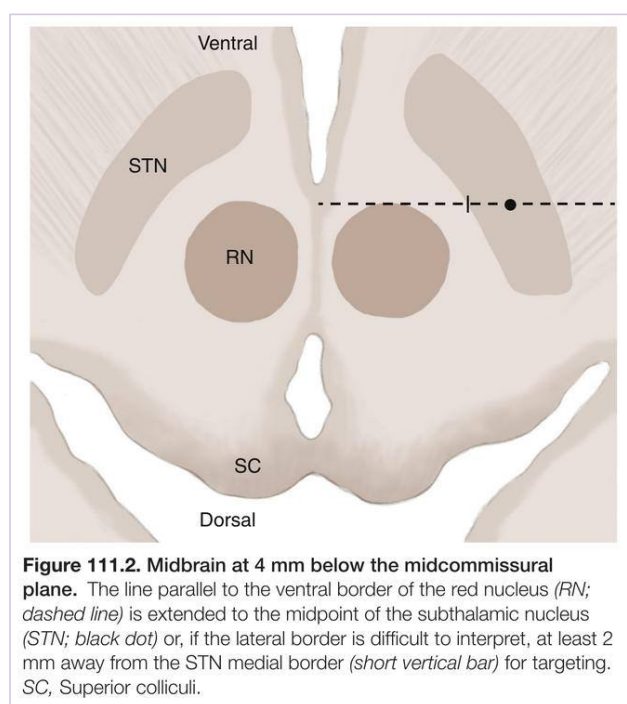


Figure 111.2. Midbrain at 4 mm below the midcommissural plane. The line parallel to the ventral border of the red nucleus (RN; dashed line) is extended to the midpoint of the subthalamic nucleus (STN; black dot) or, if the lateral border is difficult to interpret, at least 2 mm away from the STN medial border (short vertical bar) for targeting. SC, Superior colliculi.

Original figure: Youmans & Winn 8e, Fig. 111.2, p. 734

- The midbrain divides into crus cerebri, tegmentum and tectum; the substantia nigra and lateral mesencephalic sulcus separate crus from tegmentum; CN III emerges medially in the interpeduncular fossa and CN IV below the inferior colliculus. **YW p.35** The corresponding basal-view sequence is crus cerebri, posterior perforated substance, substantia nigra, tegmentum, tectum. **YW p.24**
- The oculomotor nucleus lies in the periaqueductal grey at the level of the superior colliculus, with a single midline levator subnucleus and a contralateral superior rectus subnucleus; the fascicles pass through the

decussating superior cerebellar peduncle, substantia nigra and medial cerebral peduncle. **YW p.200.e19**

- The P2P segment runs lateral to the midbrain tegmentum in the ambient cistern, parallel and inferior to the basal vein, inferolateral to the geniculate bodies and pulvinar, before entering the quadrigeminal cistern; the short circumflex artery terminates at the geniculate bodies and the long circumflex reaches the colliculi. **YW p.27**

Youmans & Winn 8e — pp. 39, 200.e23, 200.e24, 201.e11, 3773, 35, 24, 200.e19, 27

Horner's Syndrome & Sympathetic Pathway

4 questions

Q A7

JUN 2020 · Paper II · Q5 · 3+3+4 marks

Elaborate Horner's syndrome:

- Clinical features.
- Causes.
- Pathway of sympathetic system.

ANSWER

(a) Clinical features [3]

- **Relative miosis** — the affected pupil is the smaller one, and the anisocoria is **most apparent in the dark**, because the sympathetically denervated pupil fails to dilate. **YW p.200.e26**
- **Slight (1–2 mm) ptosis** — from loss of sympathetic supply to the smooth muscle of the eyelid (Müller muscle), which normally contributes a few millimetres of lid elevation; this is why the ptosis of Horner syndrome is partial, unlike the complete ptosis of a third nerve palsy, which paralyses the levator palpebrae superioris. **YW p.200.e26**
- **Anhidrosis** completes the classic triad of ptosis, miosis and anhidrosis. **YW p.1501**
- **Pharmacologic confirmation:** instillation of **0.5% apraclonidine** into both eyes causes the denervated pupil to dilate and the ptosis to resolve, confirming oculosympathetic paresis; cocaine may also be used. Hydroxyamphetamine, previously used to separate preganglionic from postganglionic lesions, is now difficult to obtain, so **imaging of the entire oculosympathetic pathway is required** instead. **YW p.200.e26, p.200.e27**
- **Red flag:** an **acute-onset painful Horner syndrome represents a carotid artery dissection until proved otherwise**. **YW p.200.e26**

(b) Causes [3]

Order of neuron	Site	Causes
First order (hypothalamus → ciliospinal centre of Budge-Waller, C8–T1)	Hypothalamus / brainstem	Stroke, demyelination
	Cervical cord	Trauma, tumour, syrinx, vascular malformation
Second order (cord → superior cervical ganglion)	Cervical cord	Trauma, tumour, syrinx, vascular malformation, cervical spondylosis
	Brachial plexus	Trauma, tumour
	Pulmonary apex	Tumour (e.g. Pancoast), infection (e.g. tuberculosis), cervical rib, vascular malformation

	Neck	Iatrogenic, trauma, tumour
Third order (superior cervical ganglion → carotid plexus → orbit)	Superior cervical ganglion	Trauma, jugular ectasia, iatrogenic
	Internal carotid artery	Dissection , trauma, thrombosis
	Cavernous sinus	Tumour, inflammation, aneurysm, carotid-cavernous fistula, thrombosis
	Other	Cluster headache

YW p.200.e26

- Additional neurosurgically relevant causes: **orbital apex / cavernous sinus infiltration** (e.g. mucormycosis in a diabetic, with proptosis, periorbital oedema and partial ptosis) **YW p.200.e24**; and **iatrogenic Horner syndrome after stellate ganglion block**, in which the classic triad is common and has often been wrongly taken as a sign of an effective block. **YW p.1501**

(c) Pathway of the sympathetic system [4]**The three-neuron oculosympathetic chain**

1. **First-order (central) neuron** — from the **hypothalamus**, descending through the **lateral brainstem and cervical cord** to the **cilio-spinal centre of Budge-Waller at C8–T1**. **YW p.200.e26**
2. **Second-order (preganglionic) neuron** — from the lower cervical cord, **across the lung apex**, to the **superior cervical ganglion**. **YW p.200.e26**
3. **Third-order (postganglionic) neuron** — from the superior cervical ganglion along the **carotid sympathetic plexus** to the **cavernous sinus** and then the **orbit**, to innervate the pupillary dilator (and, via the ophthalmic division, the Müller muscle). **YW p.200.e26**

The general sympathetic outflow

- First-order (preganglionic, cholinergic) neurons of the sympathetic nervous system lie in the **intermediolateral column of the thoracic and upper lumbar spinal cord**. **YW p.2092**
- Efferent fibres exit along the **ventral roots from T1 to L2**, leave as **white rami communicantes**, and enter the **sympathetic chains** on the anterolateral aspect of the vertebral bodies, where most synapse in a sympathetic ganglion; postganglionic fibres then travel to their sites of action. **YW p.1501**
- Preganglionic neurons may instead synapse along **splanchnic (prevertebral) ganglia**, which in turn send axons to the target organs. **YW p.2092**
- **Postganglionic transmitter** is **norepinephrine**, except for the fibres to non-glabrous sweat glands (acetylcholine), renal glomeruli (dopamine) and adrenal chromaffin cells (which secrete epinephrine systemically). **YW p.2092**
- **Cervical ganglia**: three sympathetic ganglia lie in the neck in a narrow fascial plane between the longus colli and the scalene group — superior, middle and inferior. The inferior cervical ganglion is fused with the first thoracic ganglion in about **80%** of people to form the **stellate ganglion**, 1–3 cm long, lying anterior to the C7 transverse process and first rib, immediately posterior to the vertebral artery, posterolateral to the carotid artery and above the pleural dome. It supplies sympathetic fibres to the ipsilateral head and upper limb. **YW p.1501**
- **Visceral afferents** (C fibres) travel through the sympathetic ganglia **without synapsing**, entering the cord through the dorsal roots with their cell bodies in the dorsal root ganglia — which is why a sympathetic block also anaesthetises visceral sensory nerves. **YW p.1501**

High-yield: because cardiac sympathetic fibres ascend the chain to synapse in the cervical ganglia, a **left stellate**

ganglion block is effective in refractory ventricular arrhythmias without producing heart block. YW p.1501

Youmans & Winn 8e — pp. 200.e26, 1501, 200.e27, 200.e24, 2092

Q A8

DEC 2021 · Paper II · Q5

a) Horner's syndrome. [5]**b) Complications of endoscopic third ventriculostomy (ETV). [5]****ANSWER****(a) Horner's syndrome [5]****Definition**

Oculosympathetic paresis produced by a lesion anywhere along the **three-neuron oculosympathetic chain**. YW p.200.e26

Anatomy of the pathway

1. **First-order neuron** — hypothalamus → lateral brainstem and cervical cord → **ciliospinal centre of Budge-Waller (C8–T1)**.
2. **Second-order neuron** — lower cervical cord → across the **lung apex** → **superior cervical ganglion**.
3. **Third-order neuron** — superior cervical ganglion → **carotid sympathetic plexus** → cavernous sinus → orbit → pupillary dilator. YW p.200.e26

Clinical features

- **Miosis**, most obvious in the **dark** (the small pupil is the abnormal one, because it cannot dilate). YW p.200.e26
- **Partial ptosis of 1–2 mm**, because only the smooth muscle of the lid (Müller muscle) is sympathetically innervated; the levator palpebrae superioris, supplied by CN III, is spared. YW p.200.e26
- **Anhidrosis**, completing the triad of ptosis, miosis and anhidrosis. YW p.1501
- Physiologic (benign) anisocoria occurs in up to 20% of normal people, with a difference of less than 1 mm that is constant in light and dark or slightly greater in the dark — the main differential. YW p.200.e26

Causes (by order of neuron)

- **First order:** brainstem/hypothalamic stroke, demyelination; cervical cord trauma, tumour, syrinx, vascular malformation.
- **Second order:** cervical cord lesions, cervical spondylosis, brachial plexus trauma or tumour, pulmonary apex tumour (Pancoast) or infection (tuberculosis), cervical rib, neck trauma, tumour or iatrogenic injury.
- **Third order:** superior cervical ganglion trauma or iatrogenic injury, jugular ectasia, **internal carotid dissection**, thrombosis, cavernous sinus tumour, inflammation, aneurysm, carotid-cavernous fistula; cluster headache. YW p.200.e26

Investigation

- **Pharmacologic testing** with cocaine or **0.5% apraclonidine** confirms the diagnosis — apraclonidine reverses the anisocoria by dilating the denervated pupil and resolving the ptosis. YW p.200.e26, p.200.e27
- Hydroxyamphetamine localisation is no longer practical, so **imaging of the entire oculosympathetic pathway** is required to look for a structural lesion; nonetheless the cause of a chronic isolated Horner syndrome is often never identified. YW p.200.e26, p.200.e27
- **An acute painful Horner syndrome is a carotid dissection until proved otherwise.** YW p.200.e26

(b) Complications of endoscopic third ventriculostomy (ETV) [5]

Overall risk

The overall complication rate varies from **5.8% to 16%**, with permanent morbidity under **3%**. The variation depends chiefly on the indication, the cause of the hydrocephalus and comorbidity, and on centre and surgeon experience. ETV failure itself ranges from **10% to 50%**. **YW p.350**

Major complications to be disclosed at consent

Complication	Reported rate
CSF leak	1%–6%
Meningitis	1%–5%
Cranial neuropathy	1%–2%
Seizure	~1%
Medical and endocrinologic complications	2%–9%
Intraventricular haemorrhage (overall)	1%–8.5%
Catastrophic basilar artery injury	<0.2%

YW p.350

Specific issues

- **Haemorrhage** includes intraventricular bleeding forcing abandonment of the procedure or reoperation when it blocks the ostomy, postoperative haematoma, and catastrophic basilar injury. **YW p.350**
- **Hypothalamic injury** is generally under-reported and includes pathological **weight gain**, diabetes insipidus, amenorrhoea and precocious puberty. **YW p.350**
- **Forniceal damage** is rare, but in one series the rate was as high as 16.6%. **YW p.350**
- **Rapid delayed deterioration** occurs in more than **1 per 200** patients after ETV; 16 reported paediatric cases (mean age 7.6 years) had a mortality of **81%**, and all but one survivor was left disabled. It is uncommon in adults. This is the reason ETV must be treated as a **treatment, not a cure**, with lifelong follow-up. **YW p.350**
- The reported series (Drake, Jenkinson, Hader, Gangemi, Dusick, Kulkarni, Riva-Cambrin) give overall complication rates of 5.8%–16%, with the Hader series reporting 8% for primary ETV rising to **31% when ETV was performed in lieu of shunt revision**. **YW p.351**

Prevention

- Withdraw the endoscope slowly at the end to inspect for haemorrhage and contusion; occlude the cortical tract with fibrin glue or gelatin sponge, or close the dura watertight, to prevent subdural hygroma and CSF leak. **YW p.350**
- External drains and bur-hole reservoirs are controversial — they may promote leakage, infection and pseudomeningocele, and an open drain may prevent the pressure gradient needed to keep the stoma working. **YW p.350**

Youmans & Winn 8e — pp. 200.e26, 1501, 200.e27, 350, 351

Q A9

DEC 2022 · Paper II · Q4

- Various autonomic Nuclei of the Cranial nerves. [5]
- Sympathetic outflow - Anatomy and its supply. [5]

ANSWER

(a) Various autonomic nuclei of the cranial nerves [5]

Parasympathetic (general visceral efferent) outflow

Preganglionic parasympathetic neurons are **cholinergic** and lie **within the brainstem or the sacral spinal cord**; their axons synapse on ganglia closely associated with the target organs, and the postganglionic neurons are also cholinergic. **YW p.2092**

Nerve	Brainstem nucleus / origin	Peripheral ganglion	Target
CN III (oculomotor)	Parasympathetic nucleus in the periaqueductal grey at the superior colliculus	Ciliary ganglion	Pupillary sphincter and ciliary muscle YW p.200.e21
CN VII (facial, via nervus intermedius)	Fibres relay at the geniculate ganglion , "the main parasympathetic output of the facial nerve"	Pterygopalatine (sphenopalatine) ganglion via the greater superficial petrosal nerve; submandibular ganglion via the chorda tympani–lingual nerve	Lacrimal, nasal and palatine glands; submandibular and sublingual glands YW p.63, p.64, p.1501
CN IX (glossopharyngeal)	Youmans states the general visceral efferent parasympathetic fibres arise in the nucleus ambiguus region and pass to the parotid; the lesser superficial petrosal nerve leaves the geniculate region	Otic ganglion	Parotid gland YW p.2236, p.63
CN X (vagus)	Dorsal motor nucleus of the vagus and nucleus ambiguus	Ganglia in the walls of the target viscera	Parasympathetic control of digestive, respiratory and cardiac function YW p.2236

Visceral afferent (sensory) nuclei

- **CN IX**: general visceral afferents from the **carotid body and carotid sinus** synapse in the **inferior glossopharyngeal ganglion** and then in the **inferior visceral sensory portion of the nucleus solitarius**. **YW p.2236**
- **CN X**: visceral afferents have their cell bodies in the **inferior (nodose) ganglion of the vagus** and terminate centrally in the **dorsal nucleus of the vagus**. **YW p.2236**

Mixed cranial autonomic ganglia

- The **sphenopalatine (pterygopalatine, Meckel) ganglion** in the pterygopalatine fossa is composed of sensory, parasympathetic and sympathetic elements: two to three sensory fibres from the maxillary nerve, **parasympathetic fibres from the nervus intermedius of the facial nerve through the greater superficial petrosal nerve**, and **sympathetic fibres from the carotid plexus through the deep petrosal nerve**. It supplies the nose, orbit and part of the face. **YW p.1501**
- The **tympanic (Jacobson) nerve**, a branch of CN IX, forms the tympanic plexus over the promontory of the middle ear. **YW p.65**
- Other cranial autonomic ganglia implicated in pain syndromes include the sphenopalatine ganglion (temporal migraine) and the otic ganglion. **YW p.2236**

Surgical note

The oculosympathetic supply of the pupil is **not** a cranial nerve nucleus — it is the three-neuron chain from the

hypothalamus via the ciliospinal centre and superior cervical ganglion to the orbit; its interruption gives Horner syndrome. **YW p.200.e26**

(b) Sympathetic outflow – anatomy and its supply [5]

Origin

- First-order (preganglionic, cholinergic) sympathetic neurons lie in the **intermediolateral horn of the thoracic and upper lumbar spinal cord**. **YW p.2092**
- Efferent fibres leave along the **ventral roots from T1 to L2**. **YW p.1501**

Course

1. Fibres leave the ventral root as **white rami communicantes** and enter the **sympathetic chains**, which lie on the **anterolateral aspect of the vertebral bodies**. **YW p.1501**
2. Most preganglionic fibres synapse in a **paravertebral (chain) ganglion**; others pass through to synapse in **splanchnic (prevertebral) ganglia**, which then send axons to the target organs. **YW p.1501, p.2092**
3. **Postganglionic fibres** travel to their sites of action; the transmitter is **norepinephrine**, except for fibres to non-glabrous sweat glands (acetylcholine), renal glomeruli (dopamine) and adrenal chromaffin cells (epinephrine, released systemically). **YW p.2092**

Cervical sympathetic anatomy

- Three ganglia lie in the neck in a narrow fascial space between the **longus colli** and the **scalene muscle group**: superior, middle and inferior cervical ganglia. **YW p.1501**
- The inferior cervical ganglion is fused with the first thoracic ganglion in about **80%** of people to form the **stellate ganglion** — 1 to 3 cm long, lying anterior to the **C7 transverse process and first rib**, immediately posterior to the **vertebral artery**, posterolateral to the carotid artery and superior to the **pleural dome**, with the C7 and C8 roots in the immediate vicinity. It supplies sympathetic fibres to the **ipsilateral head and upper extremity**, and cardiac sympathetic fibres ascend the chain to synapse in the cervical ganglia. **YW p.1501**

Functions supplied

- Sympathetic activity governs **vasoconstriction, intestinal motility and piloerection**; sympathetic blockade produces vasodilatation, exploited in complex regional pain syndrome and vasculopathies. **YW p.1501**
- In the lower urinary tract, sympathetic pathways originate in the **T11–L2** segments, travel through the chain to the prevertebral ganglia in the superior hypogastric and pelvic plexuses, and promote **storage** of urine (β -adrenergic detrusor relaxation, α -adrenergic contraction of bladder neck and outlet). **YW p.202.e1**
- Visceral **afferent** C fibres carrying burning/aching visceral pain traverse the sympathetic ganglia **without synapsing** and enter the cord through the dorsal roots, with cell bodies in the dorsal root ganglia. **YW p.1501**

High-yield: iatrogenic **Horner syndrome (ptosis, miosis, anhidrosis) after a stellate ganglion block** is common and has often been wrongly taken as proof of an effective block. **YW p.1501**

Youmans & Winn 8e — pp. 2092, 200.e21, 63, 64, 1501, 2236, 65, 200.e26, 202.e1

Q A10

JAN 2025 · Paper II · Q2

- a) Horner syndrome. [5]
- b) Craniosacral outflow. [5]

ANSWER

(a) Horner syndrome [5]

Definition and anatomy

Oculosympathetic paresis from a lesion anywhere along the **three-neuron oculosympathetic chain**:

1. **First-order neuron** — hypothalamus, down the **lateral brainstem and cervical cord** to the **cilio-spinal centre of Budge-Waller (C8–T1)**.
2. **Second-order neuron** — lower cervical cord, **across the lung apex**, to the **superior cervical ganglion**.
3. **Third-order neuron** — superior cervical ganglion, along the **carotid sympathetic plexus**, through the **cavernous sinus** to the orbit, to the **pupillary dilator**. YW p.200.e26

Clinical features

- **Miosis** — with a sympathetic lesion the ipsilateral pupil is the **smaller** one and the anisocoria is **most apparent in the dark**. YW p.200.e26
- **Partial ptosis (1–2 mm)** — the sympathetic system supplies only the smooth muscle of the lid (**Müller muscle**), which provides a few millimetres of lid elevation; the levator palpebrae superioris (CN III) is unaffected. YW p.200.e26
- **Anhidrosis** — completing the classic triad. YW p.1501
- Differentiate from **physiologic anisocoria**, present in up to 20% of normal people, with less than 1 mm difference that is constant in light and dark. YW p.200.e26

Causes

- **First order:** brainstem/hypothalamic stroke or demyelination; cervical cord trauma, tumour, syrinx, vascular malformation.
- **Second order:** cervical cord lesions, cervical spondylosis, brachial plexus trauma or tumour, pulmonary apex tumour (Pancoast) or tuberculosis, cervical rib, neck trauma, tumour, iatrogenic injury.
- **Third order:** superior cervical ganglion trauma or iatrogenic injury, jugular ectasia, **internal carotid dissection**, carotid trauma or thrombosis, cavernous sinus tumour, inflammation, aneurysm, carotid-cavernous fistula, thrombosis; cluster headache. YW p.200.e26

Diagnosis

- **Pharmacologic testing** with cocaine or **0.5% apraclonidine** confirms oculosympathetic paresis (apraclonidine dilates the denervated pupil and reverses the ptosis). YW p.200.e26, p.200.e27
- Hydroxyamphetamine, previously used to separate pre- from postganglionic lesions, is now hard to obtain, so **imaging of the whole oculosympathetic pathway** is required; an aetiology for a chronic isolated Horner syndrome is often never found. YW p.200.e26, p.200.e27
- **An acute-onset painful Horner syndrome is a carotid artery dissection until proved otherwise.** YW p.200.e26

(b) Craniosacral outflow [5]

Definition

The parasympathetic division of the autonomic nervous system. Preganglionic parasympathetic neurons are **cholinergic** and are located **within the brainstem or the sacral spinal cord**; their axons synapse on **ganglia closely associated with the target organs**, and the postganglionic neurons are also cholinergic. YW p.2092 This contrasts with the sympathetic (thoracolumbar) outflow from the intermediolateral horn of the thoracic and upper lumbar cord. YW p.2092

Cranial component

Nerve	Central origin	Ganglion	Supply
CN III	Parasympathetic nucleus in periaqueductal grey	Ciliary ganglion	Pupillary sphincter, ciliary muscle YW p.200.e21

CN VII (nervus intermedius)	Relays at the geniculate ganglion , "the main parasympathetic output of the facial nerve", giving the greater superficial petrosal nerve anteriorly and the lesser superficial petrosal nerve inferiorly	Pterygopalatine (sphenopalatine) ganglion; submandibular ganglion via chorda tympani + lingual nerve	Lacrimal, nasal, palatine glands; submandibular and sublingual glands YW p.63, p.64, p.1501
CN IX	Parasympathetic general visceral efferents described in Youmans as arising with the nucleus ambiguus	Otic ganglion	Parotid gland YW p.2236
CN X	Dorsal motor nucleus of the vagus and nucleus ambiguus	Ganglia within the walls of the viscera	Parasympathetic control of digestive, respiratory and cardiac function YW p.2236

- Visceral afferents accompany the outflow: CN IX afferents from the carotid body and sinus relay in the inferior glossopharyngeal ganglion and then the **inferior visceral sensory part of the nucleus solitarius**; CN X afferents have their cell bodies in the **nodose (inferior) ganglion** and terminate in the dorsal nucleus of the vagus. **YW p.2236**
- The **sphenopalatine ganglion** is the classic mixed relay: parasympathetic fibres from the nervus intermedius via the greater superficial petrosal nerve, sympathetic fibres from the carotid plexus via the deep petrosal nerve, and sensory fibres from the maxillary nerve. **YW p.1501**

Sacral component

- Preganglionic parasympathetic neurons for the lower urinary tract lie in the **S2–S4** cord segments; cholinergic preganglionic neurons within the **intermediolateral sacral cord** send axons to ganglion cells in the **pelvic plexus and bladder wall**. Postganglionic neurons release **acetylcholine**, acting on **M3 muscarinic receptors** on detrusor smooth muscle to produce bladder contraction. **YW p.202.e1**
- In the spinal grey matter, lamina VII of the intermediate zone contains the intermediolateral nucleus mediating sympathetic innervation and, **in the sacral region, the postganglionic parasympathetic neurons**. **YW p.2398**
- The cauda equina also provides, in part, parasympathetic innervation of the bladder together with sensory innervation of the perineum. **YW p.84**

High-yield: craniosacral = **CN III, VII, IX, X + S2–S4**; parasympathetic ganglia lie **close to (or within) the target organ**, so postganglionic fibres are short — the reverse of the sympathetic arrangement. **YW p.2092**

Youmans & Winn 8e — pp. 200.e26, 1501, 200.e27, 2092, 200.e21, 63, 64, 2236, 202.e1, 2398, 84

Spinal Cord Blood Supply / Artery of Adamkiewicz

4 questions

Q A11

JUN 2020 · Paper I · Q7 · 3+3+4 marks

- Ligamentum denticulatum.
- Blood supply of spinal cord.
- Conus medullaris.

ANSWER

(a) Ligamentum denticulatum [3]

- **Definition and number.** The spinal cord is ensheathed in leptomeninges (pia, arachnoid and dura) and is **attached to the arachnoid and dural sheath by 20 to 22 paired denticulate ligaments.** YW p.2397
- **Origin and attachment.** They are lateral extensions **arising from the pia** that connect the cord to the dura, lying in the coronal plane between the dorsal and ventral nerve roots. YW p.84
- **Relations at the craniovertebral junction.** As the vertebral artery becomes intradural around the inferomedial aspect of the occipital condyle it is **anchored to the craniocervical junction by the denticulate ligament.** YW p.54
- **Relation to the accessory nerve.** In the spine the denticulate ligament lies **anterior to the rootlets of the accessory nerve**; as both ascend they cross, the ligament anchoring to the dural cuff of the vertebral artery while the accessory nerve runs anterosuperiorly to the jugular foramen. Any mobilisation of the ligament (e.g. vertebral artery transposition) must therefore be done **with continuous visual contact with the accessory nerve.** In the upper cervical spine the surgeon must beware of spinal accessory rootlets coursing **superior and anterior** to the ligament in a cephalad direction. YW p.54, p.2551
- **Surgical use.** **Division of the denticulate ligament allows further ventral access** to intradural extramedullary tumours and is a standard manoeuvre to gain a ventral corridor; the cut ligament can also be used to retract the cord gently. YW p.2551

(b) Blood supply of the spinal cord [3]

Longitudinal arteries

- A single **anterior spinal artery (ASA)** ventrally and **paired posterior spinal arteries (PSA)** dorsolaterally; they anastomose in the **Lazorthes basket.** YW p.2400
- The **ASA** forms at the level of the foramen magnum from one branch of each vertebral artery near the vertebrobasilar junction, descends in the **anterior median fissure** and terminates with the filum terminale. **Sulcal arteries** from the ASA perforate the pia to supply the **anterior one-half to two-thirds** of the cord — anterior and lateral corticospinal tracts, anterior horns and anterolateral system — sparing the dorsal columns. YW p.84, p.85, p.2400
- The **PSAs** arise from the vertebral artery or PICA, descend along the posterolateral cord and supply the **posterior third** — dorsal columns, dorsal horns and posterior nerve roots. YW p.85, p.2400

Segmental reinforcement

- The ASA is reinforced by several **anterior segmental medullary arteries**, the largest of which is the **artery of Adamkiewicz**, which typically arises from the left posterior intercostal artery between **T9 and L2.** YW p.84, p.2400
- **Radicular arteries** run with the anterior and posterior roots and supply the nerve roots; unlike segmental medullary arteries they **do not join** the anterior or posterior spinal arteries. YW p.85

Watershed

The thoracic cord between **T4 and T9** is poorly vascularised — the **critical vascular zone** — corresponding to the narrowest part of the spinal canal; in this region the ASA has its smallest diameter and often receives only a single contribution around T7, usually on the left. The midthoracic spine is a watershed between radicular arteries and is therefore vulnerable to ischaemia during hypoperfusion. YW p.84, p.85, p.2400

Veins

PSA territory drains to the midline posterior spinal vein and paired posterolateral spinal veins, and the ASA territory to the midline anterior spinal vein; these form longitudinal plexuses drained by radicular veins into the epidural internal and then external plexuses — the valveless **Batson plexus.** YW p.2400

(c) Conus medullaris [4]

- **Definition.** The **tapered lower end of the spinal cord**. YW p.84
- **Level.** The cord is 40–50 cm long and extends from the foramen magnum to the first or second lumbar vertebral segment; because of the developmental ascent of the cord relative to the vertebral column, the cord terminates as the conus medullaris at **L1–L2 in adults** before fanning into the cauda equina. YW p.84, p.2397
- **Enlargements.** The cord is enlarged from C3 to T1 (cervical enlargement) and from L1 to S2 (lumbar enlargement); the lumbosacral enlargement from L2 to S3 reflects the motor demand of the lower limbs. Lower cord levels have a **higher ratio of grey to white matter** because fewer ascending and descending fibres are present. YW p.84, p.2397
- **Cauda equina.** A bundle of spinal nerves and rootlets arising from the lumbar enlargement and the conus, composed of the **second to fifth lumbar, first to fifth sacral and the coccygeal nerve pairs**. It innervates the pelvic organs and lower limbs, provides sensory innervation of the perineum and, in part, parasympathetic innervation of the bladder. YW p.84
- **Filum terminale.** A delicate strand of **non-neurogenic leptomeningeal tissue** arising from the end of the conus and attaching distally to the dorsal aspect of the **first coccygeal vertebral body**; it stabilises and tethers the lower end of the cord. The cord is attached to the sacrum via the filum terminale. YW p.84, p.2397
- **Blood supply.** The ASA descends to terminate with the filum terminale, and the artery of Adamkiewicz is the **dominant supplier to the lumbosacral cord segments** — its damage can theoretically cause paraplegia from cord infarction. YW p.84, p.85
- **Surgical relevance.** The **sensory dermatomes** of the sacral segments (S2–S4 = perineum) allow clinical separation of conus from cauda equina lesions. YW p.2399

Youmans & Winn 8e — pp. 2397, 84, 54, 2551, 2400, 85, 2399

Q A12

DEC 2021 · Paper I · Q3

- a) Blood supply of spinal cord. [5]
- b) Visual evoked potential (VEP). [5]

ANSWER

NOT IN YOUMANS

part (b) only. Youmans & Winn 8e gives the neurosurgical applications of the visual evoked potential but not its technique or waveform; those lines are answered from standard clinical neurophysiology teaching. Verify before quoting in the exam.

(a) Blood supply of the spinal cord [5]

Longitudinal system

- The cord is supplied by longitudinal vessels: a single **anterior spinal artery (ASA)** running ventrally and **paired posterior spinal arteries (PSA)** running dorsolaterally, which anastomose in the **Lazorthes basket**. YW p.2400
- The **ASA** is formed at the level of the foramen magnum by the union of one branch from each vertebral artery near the vertebrobasilar junction; it descends in the **anterior median fissure** and terminates with the filum terminale. YW p.84, p.2400
- The **PSAs** arise from the vertebral artery or from PICA and descend along the posterolateral surface of the cord. YW p.85, p.2400

Territories

Vessel	Territory
ASA (via sulcal arteries perforating the pia)	Anterior one-half to two-thirds of the cord — anterior and lateral corticospinal tracts, anterior horns, anterolateral (spinothalamic) system; dorsal columns spared YW p.85, p.2400
PSA	Posterior one-third — dorsal columns, dorsal horns, posterior nerve roots YW p.85, p.2400

Segmental supply

- The ASA is reinforced by several **anterior segmental medullary arteries** arising from the aorta, the largest being the **artery of Adamkiewicz**, generally arising **between T9 and L2 on the left**, with considerable variation. **YW p.84, p.2400**
- The PSAs are reinforced by **posterior segmental medullary arteries** and by **radicular arteries** arising from the spinal branches of segmental vessels, entering through the intervertebral foramina and dividing into anterior and posterior radicular branches that travel with the respective roots toward the sulcal artery anastomoses. **Radicular arteries supply the nerve roots and do not themselves join the anterior or posterior spinal arteries.** **YW p.85, p.2400**

Watershed and clinical correlate

- The thoracic cord between **T4 and T9** is the **critical vascular zone**, corresponding to the narrowest part of the canal; the ASA is at its smallest here and often receives only one contribution, around T7 and usually on the left. The midthoracic spine is a **watershed between radicular arteries** and is therefore susceptible to ischaemia during hypoperfusion. **YW p.84, p.85, p.2400**
- Anterior spinal artery syndrome:** paralysis, loss of faecal/urinary continence and loss of pain and temperature below the level, with relative preservation of vibration and proprioception. **YW p.2400**

Venous drainage

PSA territory → midline posterior spinal vein and paired posterolateral spinal veins; ASA territory → midline anterior spinal vein. These form anterior and posterior median longitudinal and lateral longitudinal veins, drained by anterior and posterior radicular veins into the epidural internal plexus and then the external plexus — the valveless **Batson venous plexus**, with extensive anastomoses to the retroperitoneal and thoracic compartments. **YW p.2400**

(b) Visual evoked potential (VEP) [5]

Principle and technique

The VEP is the averaged cortical electrical response recorded over the occipital scalp to a repeated visual stimulus (pattern-reversal checkerboard in a cooperative patient, flash stimulus with goggles in infants, anaesthetised or uncooperative patients). The dominant positive deflection at about 100 ms — **P100** — is measured for **latency** (conduction in the prechiasmal visual pathway) and **amplitude**; each eye is tested separately, and an interocular latency difference indicates a unilateral prechiasmal lesion. (*Technique not described in Youmans 8e.*)

Neurosurgical applications documented in Youmans

- Raised intracranial pressure.** Delay in visual evoked potentials has been observed in patients with increased ICP. In the 1980s the delay was studied in paediatric patients with **hydrocephalus and cerebral oedema** and was found to correlate significantly with ICP. One commercial non-invasive ICP monitor (NIP-200) derives ICP from this principle; its measurements correlated well with lumbar puncture opening pressure and with epidurally measured ICP, and it has been used to guide mannitol therapy in intracerebral haemorrhage. **YW p.294**

- **Craniosynostosis.** VEPs, together with optical coherence tomography, form part of the evaluation of these patients, in whom elevated ICP is a well-recognised concern. **YW p.1679**
- **Optic pathway / hypothalamic glioma and NF1.** Current screening recommendations state that **baseline "screening" neuroimaging or visual-evoked potential measurement of asymptomatic children with normal visual examinations is not warranted**; annual formal ophthalmological examination is used instead in children with NF1 under 8 years. **YW p.1763.e2**

Limitations

- The VEP is a **non-continuous, averaged, operator-dependent** measure; among non-invasive methods of estimating ICP none has provided satisfactory accuracy for detecting ICP change over time, and an **EVD connected to an external strain gauge remains the most reliable, accurate and cost-effective ICP monitor.** **YW p.294**

High-yield: for the exam, VEP in neurosurgery is best framed as (i) an objective test of anterior visual pathway conduction and (ii) an investigational non-invasive surrogate for raised ICP — never a substitute for direct ICP monitoring. **YW p.294**

Youmans & Winn 8e — pp. 2400, 84, 85, 294, 1679, 1763.e2

Q A13

JUN 2022 · Paper II · Q1

- a) Branches of internal carotid artery. [2]
- b) Parts of internal carotid artery. [2]
- c) Parts and branches of posterior cerebral artery. [2]
- d) Artery of Adamkiewicz and its surgical importance. [4]

ANSWER

(a) Branches of the internal carotid artery [2]

Segment	Branches
Cervical	No branches
Petrous	Caroticotympanic artery (at the vertical–horizontal transition, to the tympanic cavity); vidian artery (near foramen lacerum, to the pterygoid canal)
Cavernous	From the posterior bend, the meningohypophyseal trunk = inferior hypophyseal artery + dorsal meningeal artery + tentorial artery (artery of Bernasconi-Cassinari); from the horizontal portion, the inferolateral trunk
Clinoid	No named branch
Supraclinoid	Ophthalmic artery , superior hypophyseal arteries (1–5, to the stalk and anterior lobe), posterior communicating artery (largest branch = premammillary/anterior thalamoperforating artery), anterior choroidal artery , and perforators; terminates by dividing into ACA and MCA

YW p.25, p.26

- The ophthalmic artery arises under the optic nerve, usually from the medial third of the superior surface of the ICA, then passes anterolaterally to become superolateral before entering the optic canal. The inferior hypophyseal artery from the meningohypophyseal trunk supplies the posterior pituitary lobe. **YW p.25**
- The anterior choroidal artery arises from the posterolateral or posterior aspect of the ICA, runs below the optic tract to the temporal horn through the choroidal fissure, and supplies the optic tract, crus cerebri, lateral geniculate body, uncus, optic radiation, globus pallidus, midbrain, thalamus and the retrolenticular and posterior parts of the posterior limb of the internal capsule. **YW p.26**

(b) Parts of the internal carotid artery [2]

The ICA is didactically divided into **five parts: cervical, petrous, cavernous, clinoid and supraclinoid**. YW p.24

1. **Cervical** — carotid bifurcation to the entrance of the carotid canal; no branches. YW p.24
2. **Petrous** — inside the carotid canal, from its entrance to the upper edge of the **petrolingual ligament** at the foramen lacerum; divided into **vertical and horizontal** segments. YW p.24
3. **Cavernous** — upper edge of the petrolingual ligament to the inferior edge of the anterior clinoid process laterally and to the distal dural ring medially; divided into **ascending portion, posterior bend, horizontal portion and anterior bend**. YW p.24, p.25
4. **Clinoid** — from the inferior edge of the anterior clinoid process to the **dural ring**; it is in fact intracavernous, covered laterally by the carotid-oculomotor membrane and the anterior clinoid process. **There is no clinoid segment on the medial side**, where the dural ring separates cavernous from supraclinoid carotid. YW p.25
5. **Supraclinoid** — subdivided by the origins of its branches into the **ophthalmic segment** (ophthalmic artery to PComA), the **communicating segment** (PComA to AChA) and the **choroidal segment** (AChA to the ICA bifurcation). YW p.25

(c) Parts and branches of the posterior cerebral artery [2]

Parts

- **P1** — basilar bifurcation to the junction with the PComA.
- **P2** — PComA to the posterior aspect of the midbrain; subdivided into **P2A** (PComA around the crus cerebri, below the optic tract, AChA and basal vein, up to the posterior margin of the crus) and **P2P** (posterior margin of the crus, lateral to the midbrain tegmentum in the ambient cistern, to the quadrigeminal cistern).
- **P3** — from under the posterior pulvinar in the lateral quadrigeminal cistern to the anterior limit of the anterior calcarine sulcus; often already divided into calcarine and parieto-occipital branches.
- **P4** — the cortical branches. YW p.27
- The **collicular (quadrigeminal) point**, where the two PCAs are closest, marks the posterior limit of the midbrain on angiography. YW p.27

Branches

Posterior thalamoperforating, direct perforating, short and long circumflex, thalamogeniculate, medial and lateral posterior choroidal, hippocampal, inferior temporal, parieto-occipital, calcarine and posterior pericallosal arteries. YW p.27

- **Posterior thalamoperforators** (from P1) supply the anterior and part of the posterior thalamus, hypothalamus, subthalamus, substantia nigra, red nucleus, oculomotor and trochlear nuclei and nerve, mesencephalic reticular formation, pretectum, rostromedial floor of the third ventricle and posterior internal capsule. YW p.27
- **Thalamogeniculate** arteries supply the posterior half of the lateral thalamus, posterior limb of the internal capsule and optic tract. YW p.27
- **Medial posterior choroidal artery** (angiographic "number 3" shape) supplies the crus cerebri, tegmentum, geniculate bodies, colliculi, pulvinar, pineal gland and medial thalamus; the **lateral posterior choroidal artery** supplies the choroid plexus of the atrium and temporal horn and anastomoses with the AChA. YW p.27
- The **splenial (posterior pericallosal) artery** arises from the parieto-occipital artery in 62%. YW p.28

(d) Artery of Adamkiewicz and its surgical importance [4]

CHAPTER 3

Head Injury & Neurotrauma

22

QUESTIONS SOLVED

Chronic & Acute Subdural Haematoma

5 questions

Q C1

JUN 2021 · Paper II · Q10

Management of:

- a) Chronic subdural hematoma. [3]
- b) Normal pressure hydrocephalus. [4]
- c) Cerebral metastasis. [3]

ANSWER

(a) Management of chronic subdural haematoma [3]

Preoperative

- Elderly comorbid patient: cardiorespiratory examination, FBC, U&E, clotting, ECG; treat chest infection and optimise cardiac drugs first. Correct coagulopathy and thrombopathy — essential to reduce operative bleeding and recurrence. **YW p.362**

Conservative

- When operative risk outweighs benefit: asymptomatic small collections (repeat CT in a few weeks) and moribund patients with poor baseline function, managed with elderly-care physicians and an explicit ceiling of care. **YW p.363**

Surgical

- Symptomatic radiologically confirmed CSDH → evacuation; favourable outcome in ~80%. Three techniques: twist-drill craniostomy (<10 mm), bur-hole craniostomy (10–30 mm), craniotomy. **YW p.363**
- Bur-hole craniostomy is first line** — two bur holes ~7 cm apart just above the superior temporal line, irrigate until effluent runs clear, soft subdural drain via the **frontal** bur hole, removed at ~48 h. Drainage cut recurrence 24.3% → 9.3% and 6-month mortality 18.1% → 9.6%. **YW p.363–364**
- Craniotomy/mini-craniotomy for a significant acute component, multiple membranes or recurrence. **YW p.364–365**

Adjuvants

- Avoid dexamethasone** outside research (Dex-CSDH: fewer reoperations but worse 6-month mRS). No case for prophylactic antiepileptics. Early mobilisation, TEDs and LMWH once the drain is out. Recurrence ~10%. **YW p.362, p.365–366**

(b) Management of normal pressure hydrocephalus [4]

Diagnosis

- International and Japanese guidelines require at least one symptom of the triad, Evans index ≥ 0.3 , and exclusion of other explanatory diagnoses. Supportive MRI: periventricular lucency and the **DESH** pattern (enlarged Sylvian fissures, tight vertex sulci). Phase-contrast aqueductal flow, fMRI, MRS, DTI, SPECT and radionuclide cisternography do **not** predict shunt response. **YW p.328**

Predictive tests — mandatory before shunting

Test	Method	Performance
CSF tap test	Remove 30–50 mL by LP; objective gait/cognition before and 2–4 h after	Sens 26–62%, PPV >75%, NPV low (~45%)
External lumbar drainage	10–15 mL/h for 36–72 h; ≥30% gait velocity gain or ≥3–4 MoCA points	~95% sens, 78% spec; infection 2–5%
CSF infusion test	Resistance to outflow (Rout); normal 5–10 mm Hg/mL/min	Sens 50–100%; mainly European

YW p.328, p.330

- A negative tap test does **not** exclude shunt responsiveness — proceed to ELD. **YW p.328**

Treatment

- CSF shunt insertion; no configuration (VP, VA, LP) is proven superior. **ETV gives poorer results and is not recommended** except in older adults with documented obstructive hydrocephalus. There is no effective medical therapy. Properly selected patients improve at least as much as after CSF removal, maintained for years while the shunt works. **YW p.330**

Perioperative

- Multidisciplinary optimisation. Frailty raises complication risk 2.5-fold but frailty indices must **not** exclude a patient from shunting, since iNPH decline is potentially reversible; advanced age alone is not a risk factor. Plan antithrombotic interruption in advance (warfarin 5 days, DOACs 4 days, clopidogrel 5–7 days, aspirin 3–5 days). **YW p.330–331**

(c) Management of cerebral metastasis [3]

- Brain metastases are the commonest intracranial tumour in adults; resection gives clear survival and symptom benefit but adjuvant radiation is needed for local control. **YW p.1187**

Surgery

- Indications: tissue diagnosis, decompression for symptomatic mass effect and vasogenic oedema. Patchell 1990 and a second RCT established resection + WBRT for a **single** metastasis with KPS ≥70. **YW p.1191**
- Lesions ≥3 cm should be resected (SRS gives high local failure and radiation necrosis); SRS suits lesions up to ~1–3 cm. Resection of the dominant lesion in multiple metastases gives survival similar to solitary disease where lesions are accessible and systemic disease is controlled. Brainstem, basal ganglia and thalamic lesions are managed non-operatively. **YW p.1191–1192**

Radiotherapy

- Patchell 1998: WBRT after resection reduced local and distant intracranial failure and neurological death, without an overall survival gain. **YW p.1198**
- Guidelines now recommend **SRS to the resection cavity** after resection of a single metastasis (2017 RCT: lower local recurrence than resection alone), and SRS alone for any number of metastases when cumulative volume is <7 mL; add WBRT if >4 metastases with volume >7 mL, >15 metastases, or lesions unsuitable for resection/SRS. **YW p.1198**
- If WBRT is used, add memantine and consider hippocampal avoidance — 35–52% suffer a 3-SD cognitive drop versus 6–24% after SRS. **YW p.1190–1191**

High-yield: Patchell 1990 = surgery + WBRT beats WBRT alone; Patchell 1998 = WBRT after surgery improves local control only.

Youmans & Winn 8e — pp. 328, 330, 331, 362, 363, 364, 365, 366, 1187, 1190, 1191, 1192, 1198

Q C2

DEC 2021 · Paper I · Q5

Chronic subdural hematoma:

- a) Clinical features. [3]
- b) Pathology. [3]
- c) Management. [4]

ANSWER**(a) Clinical features [3]****Risk factors**

- Advancing age, falls or minor head injury, anticoagulants/antiplatelets, bleeding diatheses, alcohol, epilepsy, low-ICP states, haemodialysis. Only ~60% recall a head injury. Antithrombotic use has nearly doubled since the 1990s (27% in 1990–95 vs 49% in 2011–15) and drives the rising incidence. **YW p.361**

Presentation

- Onset varies from days to weeks; **20–30% are asymptomatic** and found incidentally. **YW p.361**

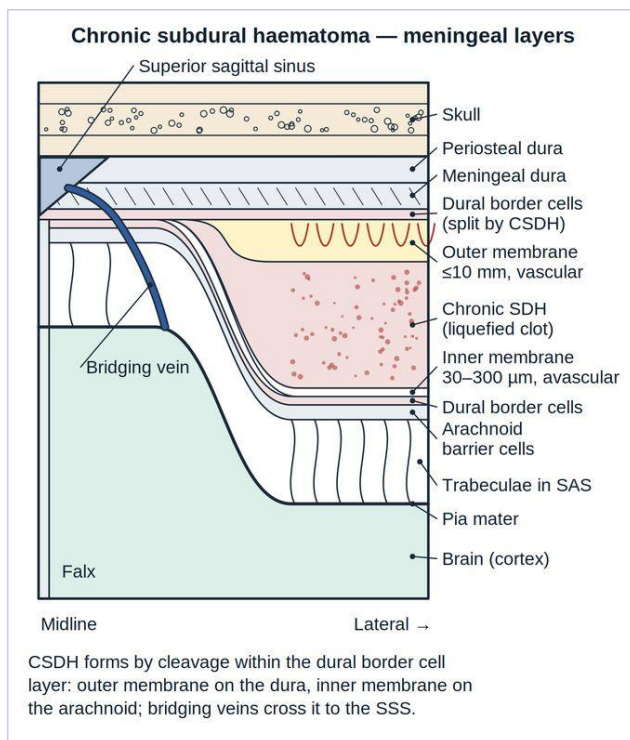
Feature	Frequency (n = 205)
Gait disturbance and falls	55.5%
Mental deterioration	34.0%
Limb weakness	34.0%
Acute confusion	32.1%
Headache	17.2%
Drowsiness or coma	9.6%
Speech impairment	5.7%
Seizure, incontinence, visual disturbance	~0.5–1% each

YW p.362

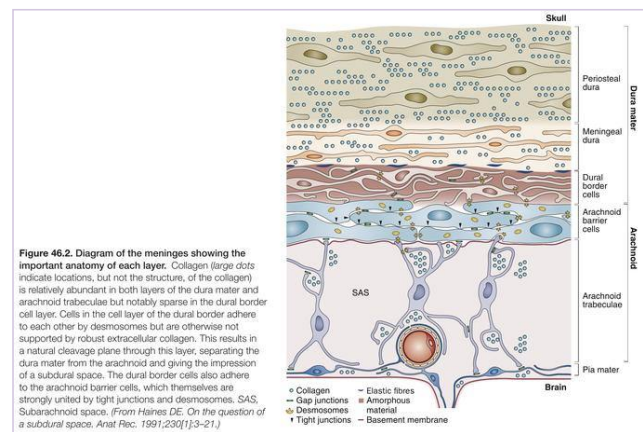
- GCS 13–15 in 80.7%, 9–12 in 12.4%, 3–8 in only 6.9%. Admission GCS and mRS predict prognosis; the Markwalder grade is historically popular but imprecise. **YW p.361–362**

(b) Pathology [3]

- An encapsulated flattened collection over one convexity, bilateral in ~19%, median volume 93–96 mL. It forms **within the dural border cell layer**, not a true anatomical space. Fluid ranges from yellow to dark brown — "coffee, red wine or engine oil". **YW p.355–356, p.361**



Schematic diagram — Chronic subdural haematoma — meningeal layers



Original figure: Youmans & Winn 8e, Fig. 46.2, p. 354

- **Outer membrane:** granulation tissue resembling healing dermis, proliferating from dura and dural border cells with ingrowth of dural capillaries; thin-walled sinusoidal vessels (a few to 80 micrometre) with endothelial fenestrations, gap junctions, incomplete basement membrane and pericyte layer — hence leaky and prone to rebleeding. Also fibroblasts, myofibroblasts, mast cells, eosinophils. **YW p.355–356**
- **Inner membrane:** thin avascular layer of fibroblasts and dural border cells. **YW p.356**
- **Fluid:** resembles serum but with low fibrinogen, plasminogen and alpha-2-antiplasmin and high fibrin degradation products, tPA and plasmin–alpha-2-antiplasmin complex — blood that has clotted then undergone fibrinolysis. Raised IL-6, IL-8, VEGF, bFGF, MMP-2/-9; high IL-6 and IL-8 predict recurrence. **YW p.356–357, p.361**
- **Pathogenesis:** parallels cutaneous wound healing — clotting, inflammation, proliferation with granulation tissue and angiogenesis, then remodelling. Enlargement is chiefly **recurrent haemorrhage** from outer-membrane neovasculature; labelled-erythrocyte studies show fresh daily bleeding accounts for ~7% of volume. The osmotic/oncotic theory is unsupported — CSDH fluid, plasma and CSF have equal osmolality and oncotic pressure. **YW p.356–357**
- **CT stages:** hypodense early, iso/hyperdense when mature, mixed when progressive, low density resolving. Nakaguchi subtypes homogeneous, laminar, separated, trabecular with recurrence 15%, 19%, 36% and 0%. **YW p.357, p.361–362**

(c) Management [4]

Preoperative

- Cardiorespiratory assessment, FBC, U&E, clotting, ECG; medical optimisation. Correct coagulopathy/thrombopathy — platelet function recovers fully **4–6 days after aspirin** and **7–10 days after clopidogrel**; if stable, stop the drug and operate in a few days, otherwise transfuse platelets. Involve haematology/cardiology after recent MI, VTE, stenting or valve replacement. **YW p.362**

Non-operative

- Asymptomatic small collection: reverse coagulopathy, repeat CT in weeks. Moribund/poor baseline: joint elderly-care management, a trial of steroids, ceiling-of-care discussion. **YW p.363**

Operative

- **Bur-hole craniostomy (first line):** two bur holes ~7 cm apart above the superior temporal line, irrigation to clear effluent, soft subdural drain through the **frontal** bur hole, tubing ≥50 cm with bag below head level, removed at ~48 h. The Cambridge RCT showed recurrence 24.3% → 9.3%, 6-month mortality 18.1% → 9.6%, with the survival benefit persisting at 5 years. **YW p.363–365**
- Twist-drill craniostomy for hypodense membrane-free collections in patients unfit for GA; craniotomy/mini-craniotomy for an acute component, multiple membranes or recurrence. **YW p.364–365**

Technique	Successful outcome	Complications	Recurrence	Mortality
Bur-hole craniostomy	79–86%	3.8–9.3%	10.5–12.1%	2.7–3.7%
Twist-drill craniostomy	88–93.5%	2.5–5.5%	14.5–33%	2.9–5.1%
Craniotomy	67.8–80.3%	3.9–12.3%	6.2–19.4%	4.6–12.2%

YW p.365

Postoperative

- **No bed rest** — prescribed bed rest independently predicts poor outcome; mobilise early with TEDs and LMWH once the drain is out. Avoid dexamethasone; statins, tranexamic acid and ACE inhibitors are not routine. **YW p.362–363, p.365**
- Restart anticoagulation individually — 2018 European AF guidance suggests it is safe at ~4 weeks after evacuation absent alcohol abuse or major fall risk; weigh CHA₂DS₂-VASc against HAS-BLED. **YW p.366**
- Complications: recurrence ~10%, postoperative acute SDH, seizures, tension pneumocephalus, subdural empyema 0.2–2.1% (contrast MRI with DWI). **YW p.366**

Youmans & Winn 8e — pp. 355, 356, 357, 361, 362, 363, 364, 365, 366

Q C3

APR 2023 · Paper I · Q5

- Describe the GCS scale and its fallacies. [5]
- Pathogenesis and treatment of acute subdural hematoma. [5]

ANSWER

(a) The Glasgow Coma Scale and its fallacies [5]

The scale

Described in 1974 and universally adopted for grading clinical severity of head injury and other causes of impaired consciousness. **YW p.2969**

Component	Response	Score
Eye opening (E)	Spontaneous / to voice / to pain / none	4 / 3 / 2 / 1
Best motor response (M)	Obeys commands	6
	Localises to pain	5
	Flexion-withdrawal	4
	Flexor (decorticate) posturing	3
	Extensor (decerebrate) posturing	2
	None	1

Best verbal response (V)	Oriented / confused / inappropriate words / incomprehensible sounds / none	5 / 4 / 3 / 2 / 1
Total (E+M+V)		3–15

YW p.2970

- Intubated patients score "T" for verbal and the scale runs 3T–11T. Severity: severe 3–8, moderate 9–13, mild 14–15. The Head Injury Severity Scale refines this to minimal (15, no LOC), mild (14–15 with amnesia/brief LOC), moderate (9–13, LOC $\geq$ 5 min or focal deficit), severe (5–8) and critical (3–4). **YW p.2970, p.2904**

Fallacies

1. **Confounded assessment** — alcohol or drug use, prehospital sedation, paralysis and intubation distort the score. **YW p.2904**
2. **Loss of the verbal component** — intubation removes V, requiring modified notation; the same applies to prelingual children, hence the Paediatric GCS. **YW p.134, p.2970**
3. **Says nothing about pathology** — it cannot reflect the risk of deterioration from swelling or bleeding, so at-risk patients still need enhanced vigilance. **YW p.2904**
4. **Discordance with care pathways** — a third of ICU patients were "mild" by GCS, reflecting frailty, comorbidity or extracranial injury. **YW p.2904**
5. **Unidimensional sum score** — eye and verbal scales discriminate better in mild/moderate injury; for individual patients report the three components separately. **YW p.2904**
6. **The elderly** — pre-existing cognitive impairment gives an abnormal baseline, comorbidity and drugs lower it further, and age-related atrophy allows substantial haematoma expansion before the score falls, so severity is systematically underestimated and abnormal scores are misattributed to pre-existing disease. **YW p.316–317**
7. **Ignores extracranial injury** — present in 35%, driving hypoxia, hypotension, pyrexia and coagulopathy; record AIS/ISS separately. **YW p.2904**

(b) Pathogenesis and treatment of acute subdural haematoma [5]**Pathogenesis**

- ASDH lies between the inner leaf of dura and arachnoid; classically from tearing of bridging veins running to the dural sinuses, but also from superficial cortical vessels or parenchymal lesions rupturing into the space. **YW p.2972, p.3071**
- A brief high-velocity impact with translational inertial loading tears bridging and pial vessels (falls, assaults — 72% of one ASDH series, only 24% road traffic); low-magnitude long-duration acceleration instead propagates deep and causes DAI. **YW p.2969, p.2972**
- **Two pathological types:** (i) contact-force type with contusion/laceration and cortical bleeding, commonest at the temporal pole, producing a **burst lobe**; (ii) convexity type from inertial tearing of bridging veins, with milder underlying damage that is chiefly ischaemic from mass effect and compromised venous outflow. **YW p.2972**
- **Ischaemia is central**, even without marked ICP rise: preoperative CBF <18 mL/100 g/min with CBV halved, ICP 40–80 mm Hg, CPP 10–60 mm Hg, jugular saturation 40–60% — all normalising on evacuation. Patients who "talk and die" are the subset most likely to benefit from decompressive craniectomy. **YW p.2972**
- ASDH affects 11% of all head injuries and up to 30% of severe TBI; up to 80% present with GCS <9 , 30–50% with pupillary abnormality; mortality 50–90%. **YW p.3071**

Treatment

- **Indications (regardless of GCS):** thickness >10 mm or midline shift >5 mm. Below these thresholds operate for GCS fall of ≥ 2 points, anisocoria/fixed dilated pupils, or ICP >20 mm Hg; GCS <9 with a sub-threshold clot warrants ICP monitoring and evacuation if ICP is uncontrollable. **YW p.3063, p.3007, p.3071**

- **Timing is critical:** operation within 2 h of onset of coma gave 47% mortality versus 80% if later; within 4 h, 30% versus 90%. Mean injury-to-surgery time was 170 min in good recoveries and 390 min in poor outcomes. **YW p.3071**
- **Technique:** large unilateral fronto-temporo-parietal craniotomy through a trauma flap with three to five bur holes including a low temporal one; pack sinus-related bleeding with haemostatic agents and tacking sutures rather than dislodging clot over a sinus; dural tenting and tacking stitches to abolish dead space. **YW p.3071**
- **Craniotomy versus primary decompressive craniectomy** is unresolved — the subject of the RESCUE-ASDH trial. **YW p.3071**
- **Critical care:** ICP-directed stepwise therapy — sedation, paralysis, CPP 55–65 mm Hg, EVD, hypertonic saline, temperature control, barbiturates, decompressive craniectomy last. **YW p.3062**

High-yield: thickness >10 mm or MLS >5 mm = operate, whatever the GCS.

Youmans & Winn 8e — pp. 134, 316, 317, 2904, 2969, 2970, 2972, 3007, 3062, 3063, 3071

Q C4

MAY 2024 · Paper II · Q2

- Components of Prothrombin citrate complex and treatment dosages for spontaneous intracranial bleed (PCC). [2]**
- Management of chronic subdural hematoma patient on DAPT (dual antiplatelet drugs). [5]**
- Hemostatic agents in neurosurgery. [3]**

ANSWER

(a) Components of prothrombin complex concentrate and dosage [2]

- **Four-factor PCC contains coagulation factors II, VII, IX and X.** It replaces factor X together with factor VII (extrinsic-pathway thrombin generation) and factor IX (intrinsic-pathway thrombin generation). **YW p.209**
- **Warfarin-related intracranial bleed:** vitamin K 5–10 mg plus FFP 15 mL/kg, with 4-factor PCC 20 U/kg (or rFVIIa). **YW p.3066** An INR-graded alternative is 25 U/kg for INR 2–4, 35 U/kg for INR 4–6, 50 U/kg for INR >6. **YW p.206**
- **Factor Xa inhibitor (rivaroxaban, apixaban, edoxaban, fondaparinux) bleed:** 4-factor PCC 50 IU/kg IV bolus, monitored by PT or anti-Xa assay; alternative is andexanet alfa. **YW p.209, p.3066**
- PCC is first-line for warfarin reversal in the elderly head injury — faster INR normalisation, less haematoma expansion and better functional outcome than FFP. **YW p.317**

(b) Management of chronic subdural haematoma in a patient on dual antiplatelet therapy [5]

Assess the indication for DAPT

- Usually recent MI, coronary stenting, VTE or valve replacement — involve **cardiology and haematology** before stopping. Antiplatelets increase both de novo and recurrent CSDH and are an identified recurrence risk factor, so correction is not optional. **YW p.361–362, p.366**

Timing — the key decision

- Platelet function recovers fully **4–6 days after stopping aspirin** and **7–10 days after clopidogrel**. **YW p.362**
- **Neurologically stable, mild symptoms:** stop both drugs and plan surgery after a few days — the preferred route. **YW p.362**
- **Urgent surgery needed:** transfuse platelets. In the UK CSDH study nearly a third of patients on a single antiplatelet received a perioperative platelet transfusion; median drug-stop-to-operation interval was 1 day with transfusion versus 5 days without. **YW p.362**

Drug	Test	Treatment
Aspirin	PFA-100	Platelets 1–2 U; desmopressin 0.4 micrograms/kg over 30 min
Clopidogrel	Platelet aggregation	Platelets 1–2 U (pooled 2 U); desmopressin 0.4 micrograms/kg over 30 min
Ticagrelor / prasugrel	Platelet aggregation	Platelets 1–2 U (pooled 2 U); desmopressin 0.4 micrograms/kg over 30 min

YW p.3066, p.206

- Desmopressin may alternatively be 0.3 micrograms/kg slow IV 12-hourly; aim for a platelet count $>100 \times 10^9/L$ before incision. **YW p.206, p.208**

Operation and aftercare

- Bur-hole craniostomy with a subdural drain via the frontal bur hole for ~48 h — the drain cuts recurrence from 24.3% to 9.3% and matters especially here, antiplatelet use being itself a recurrence risk factor. Mini-craniotomy if there is a significant acute component or multiple membranes. **YW p.363–366**
- TEDs and LMWH once the drain is removed; mobilise early. **YW p.365**
- Restarting is individualised on indication, age, comorbidity and fall risk with cardiology input; use CHA2DS2-VASc versus HAS-BLED in atrial fibrillation, and the 2018 European guidance considers restarting at ~4 weeks safe absent chronic alcohol abuse or substantial fall risk. **YW p.366**
- Note that the benefit of platelet transfusion for correcting platelet function remains largely unproven, and guidance is to transfuse **only** in patients who will undergo a neurosurgical procedure. **YW p.3066, p.318**

(c) Haemostatic agents in neurosurgery [3]**Generations**

- First generation (single agents):** collagen, fibrinogen, gelatin, thrombin, oxidised regenerated cellulose — in practice Avitene (microfibrillar collagen), bone wax, Gelfoam (porcine gelatin $\pm$ thrombin), Surgicel (oxidised regenerated cellulose). **YW p.211–212**
- Second and third generation (combinations):** FloSeal (bovine gelatin granules with human thrombin), Tisseel and Evicel (thrombin with fibrinogen), EVARREST and TachoSil (not for neurosurgical use). Carboxymethyl cellulose is preclinical. **YW p.211–212**

Neurosurgical cautions

- Surgicel, Gelfoam and FloSeal **must be removed** after haemostasis when used near the spinal cord, in laminectomy, or near the optic nerve/chiasm, because swelling can compress neural structures. **YW p.211**
- Avitene is a foreign protein that tends to exacerbate infection. Thrombin-containing agents provoke antibodies — nearly 20% of neurosurgical patients develop factor V antibodies, with postoperative coagulopathy (INR up to 2.65, PTT up to 65 s); thrombin is contraindicated in those sensitive to its components. **YW p.211**

Use in trauma surgery

- Skull base, diploic vein or diffuse dural ooze: bone wax, fibrin-soaked Gelfoam, FloSeal and pressure. Where a haematoma abuts the sagittal or transverse sinus, leave a layer of clot rather than dislodging it. **YW p.3071**
- Torn dural sinus: Surgicel, Spongostan, Gelfoam or FloSeal with gentle pressure and tacking sutures first, then an onlay patch of galea, temporalis fascia, fascia lata or dura; direct repair or trapping with clips only if these fail. **YW p.3079, p.3071**

In development

- Chitosan (de-acetylated chitin) — haemostatic independent of the coagulation cascade by electrostatically

binding red cells and platelets; mucoadhesive, antimicrobial, resorbable, safe in CNS tissue experimentally. Nanoparticulate agents (PLGA, self-assembling peptide AC5) remain preclinical. **YW p.211**

Youmans & Winn 8e — pp. 206, 208, 209, 211, 212, 317, 318, 361, 362, 363, 364, 365, 366, 3066, 3071, 3079

Q C5

MAY 2024 · Paper III · Q8

- a) Management of an 85 year old male patient with severe traumatic brain injury presenting with GCS score 7/15, Blood pressure 180/60mmHg and Pulse rate 65/min. [4]
- b) Frailty index in elderly. [3]
- c) Role of middle meningeal artery embolisation in chronic subdural hematomas. [3]

ANSWER

(a) Management of an 85-year-old with severe TBI, GCS 7, BP 180/60, pulse 65 [4]

Interpreting the presentation

- GCS 7 = severe TBI. Hypertension with bradycardia is the **Cushing response**, arising from generalised CNS ischaemia or local pressure on the brainstem — it signals raised ICP, the bradycardia being vagally mediated. Treat the ICP, not the blood pressure. **YW p.472.e8**
- Caution with GCS here: pre-existing cognitive impairment gives an abnormal baseline, comorbidity and drugs lower it further, and atrophy allows large haematomas before the score falls. Establish the pre-injury baseline from all available sources. **YW p.316–317**

Immediate

- Airway, breathing, circulation; intubate and ventilate. BTF targets systolic BP >100 mm Hg for ages 50–69 and **>120 mm Hg above 70 years**, though this is not evidence-based in the geriatric population. **YW p.318**
- Immediate non-contrast CT head** — never defer imaging in an older adult with head trauma, particularly on antithrombotics. **YW p.317–318**
- Reverse anticoagulation at once** — atrial fibrillation is present in up to 29% of older adults with TBI. PCC is first-line for warfarin; platelet transfusion for antiplatelets only if a neurosurgical procedure is planned. **YW p.315, p.317–318**

Surgery

- Evacuate by standard criteria: ASDH >10 mm or MLS >5 mm regardless of GCS; EDH >30 mL; GCS 6–8 with frontal/temporal contusion >20 mL and MLS >5 mm or cisternal compression; contusion >50 mL. **YW p.3063**
- Geriatric patients with **GCS >8** gained from craniotomy for acute SDH, but **decompressive craniectomy for intracranial hypertension in the elderly was associated with increased mortality and worse long-term function** in two studies — weigh this carefully at 85. **YW p.318**

Neurocritical care

- ICP monitoring per BTF in comatose patients with abnormal CT. Stepwise protocol: head up 30 degrees, SpO₂ >94%, PaCO₂ 4.5–5.0 kPa, CPP 55–65 mm Hg, Hb >90 g/L, normothermia, propofol and fentanyl; escalate to EVD drainage and 5% saline, then deeper sedation and hypothermia, then barbiturates, with decompressive craniectomy last. Levetiracetam if seizures are suspected. **YW p.3062, p.472.e8**
- Assess baseline frailty, reduce polypharmacy using Beers criteria, involve geriatrics, and discuss goals of care early. **YW p.315–316**

(b) Frailty index in the elderly [3]

- Frailty is a decrement in physiological reserve; with self-rated health it is emerging as a key predictor of outcome in geriatric TBI **independent of age and acute injury characteristics**, and raises perioperative

complication risk ~2.5-fold over age-matched non-frail patients. **YW p.315, p.330**

Trauma-Specific Frailty Index (TSFI) – 15 variables

Domain	Items
Comorbidities	Cancer history; coronary heart disease (MI 1, CABG 0.75, PCI 0.5, medication 0.25); dementia (severe 1, moderate 0.5, mild 0.25)
Daily activities	Help with grooming, money, housework, toileting, walking (wheelchair 1, walker 0.75, cane 0.5)
Health attitude	Feel less useful; feel sad; everything an effort; falls (within last month 1); feel lonely
Function	Sexually active
Nutrition	Albumin <3 g/dL = 1
Score	Sum / 15; frail = TSFI >0.25

YW p.316

- TSFI predicts in-hospital complications, death and discharge to a nursing facility in geriatric trauma. **YW p.315**

Groningen Frailty Indicator

- 15 items in physical (shopping, walking outdoors, dressing, toileting, self-rated fitness, vision, hearing, weight loss, ≥4 medicines), cognitive (memory), social (emptiness, longing for company, abandonment) and psychological (low mood, anxiety) domains; **frail** = **score >3**. Predicts GOS-E at 2–3 years after geriatric mild TBI. **YW p.315–316**

Caveat

- Frailty indices stratify risk but must **not** alone deny an operation whose benefit is potentially reversible — stated explicitly for shunting in iNPH, where frailty and comorbidity do not predict shunt responsiveness. **YW p.330**

(c) Role of middle meningeal artery embolisation in chronic subdural haematoma [3]

Rationale

- The outer neomembrane is granulation tissue fed by dural (middle meningeal) capillaries and is the source both of the fragile neovasculature and of the inflammatory and angiogenic mediators tPA, thrombomodulin, angiotensin-2, VEGF and MMPs; enlargement is driven by recurrent haemorrhage from these vessels. **YW p.355–356, p.361**
- MMA embolisation is hypothesised to **reduce blood supply to the neomembranes and dampen inflammation**, interrupting the rebleeding–fibrinolysis–angiogenesis cycle. **YW p.367**

Current place

- Potentially attractive in **patients with multiple recurrences**; refractory CSDH is defined as two or more recurrences, where a different procedure (mini-craniotomy, subdural–peritoneal shunt or conduit) is otherwise used. **YW p.366–367**
- A few case series report its use in asymptomatic patients or as prophylaxis after primary surgery, outside any research framework. **YW p.367**

The textbook's position

- Its role in preventing or treating recurrence, alone or as an adjunct to surgery, **requires further evaluation in clinical studies**, and it is explicitly named as a field in which robust ethically approved research must

generate evidence before the role can be defined. **YW p.366–367**

High-yield: in Youmans 8e MMA embolisation is investigational, not standard care; bur-hole craniostomy with a subdural drain remains first line.

Youmans & Winn 8e — pp. 315, 316, 317, 318, 330, 355, 356, 361, 366, 367, 472.e8, 3062, 3063

Diffuse Axonal Injury / Traumatic Axonal Injury

5 questions

Q C6

JUN 2020 · Paper I · Q8 · 5+5 marks

About Traumatic brain injury:

- Discuss pathogenesis and pathology of axonal injury.
- Classification of diffuse axonal injury.

ANSWER

(a) Pathogenesis and pathology of axonal injury [5]

Biomechanics

- Rotational acceleration of the unrestricted head delivers **shear, tensile and compressive strains** that dynamically deform brain tissue; contact forces alone cause focal lesions, and purely translational inertial loading causes contusions and subdural haematoma. The large human brain generates high shear strains between regions, and injury concentrates at the **midline** because the falx cerebri and tentorium impede brain motion. **YW p.2969, p.2912.e9**
- Low-magnitude acceleration over a long duration propagates deep and produces DAI (motor vehicle collisions); a brief high-velocity impact tears bridging veins and produces SDH (falls, assaults). Coronal or lateral acceleration produces the most severe DAI, oblique or sagittal the least. **YW p.2969, p.2973**
- Crush injury to a stationary head gives fractures, extra-axial haematoma and contusion but usually no loss of consciousness, because axonal injury is absent and the reticular activating system is undisturbed. **YW p.2926**

Pathogenesis — a delayed, progressive process

- Mechanoporation** of the axolemma, especially at the nodes of Ranvier, allowing unregulated calcium entry. **YW p.2926, p.3007**
 - Calcium-dependent cascades** — calpain activation, mitochondrial swelling with cytochrome c release and caspase activation, bioenergetic depletion, apoptosis. **YW p.2926, p.2912.e10**
 - Cytoskeletal collapse** — calcium-linked dephosphorylation causes neurofilament compaction; tau, dynein and kinesin detach from microtubules. **YW p.2912.e10**
 - Failure of axoplasmic transport** — focal accumulation of neurofilaments, organelles and APP with progressive swelling. **YW p.2912.e10, p.2926**
 - Secondary axotomy at 6–12 hours** — the severed ends retract into bulbs and the distal segment undergoes **Wallerian degeneration** over weeks to months. **YW p.2912.e10, p.2912.e9**
- These changes mature over hours to days, so axonal injury is largely **secondary rather than primary tearing** and is potentially amenable to intervention. **YW p.2973, p.2912.e9**

Pathology

- Macroscopic:** petechial (Strich) haemorrhages at sites of maximal acceleration — corpus callosum, peri-third-ventricular structures (hypothalamus, fornix columns, anterior commissure), internal capsule, basal ganglia, dorsolateral brainstem/midbrain, superior cerebellar peduncles. **YW p.2973**
- Microscopic:** axonal swellings, varicosities and **retraction balls/bulbs** at sites of discontinuity, chiefly on large myelinated fibres, at high density in about 25% of severe head injuries. **YW p.2926, p.2973**

- **Immunohistochemistry:** beta-APP staining is the modern standard — positive within **35 minutes** and almost universal in fatal TBI, whereas silver stains were positive in only ~30%. **YW p.2912.e10**
- **Types:** *multifocal (diffuse) TAI* — bulbs scattered through hemispheric white matter, brainstem and cerebellum; *vascular axonal injury* — clusters of bulbs in a zigzag or geographic pattern around ischaemic brain from raised ICP, which must be distinguished from true TAI. **YW p.2912.e10**
- Retraction bulbs are **non-specific** — also seen beside haematomas, abscesses, tumours and in demyelination; CLARITY work suggests many are varicosities of structurally intact, salvageable axons. **YW p.2912.e9, p.2912.e10**

Nomenclature: because the process may be focal, multifocal or diffuse, **traumatic axonal injury (TAI)** is preferred; "DAI" is recommended as a neuroimaging descriptor when lesions are present in three or more discrete sites. **YW p.2912.e10, p.2973**

(b) Classification of diffuse axonal injury [5]

1. Adams neuropathological grading (1989)

Grade	Localisation of lesion
I	Axonal injury of parasagittal white matter of the cerebral hemispheres
II	Grade I plus a focal lesion in the corpus callosum
III	Grade II plus a focal lesion in the cerebral peduncle (rostral brainstem)

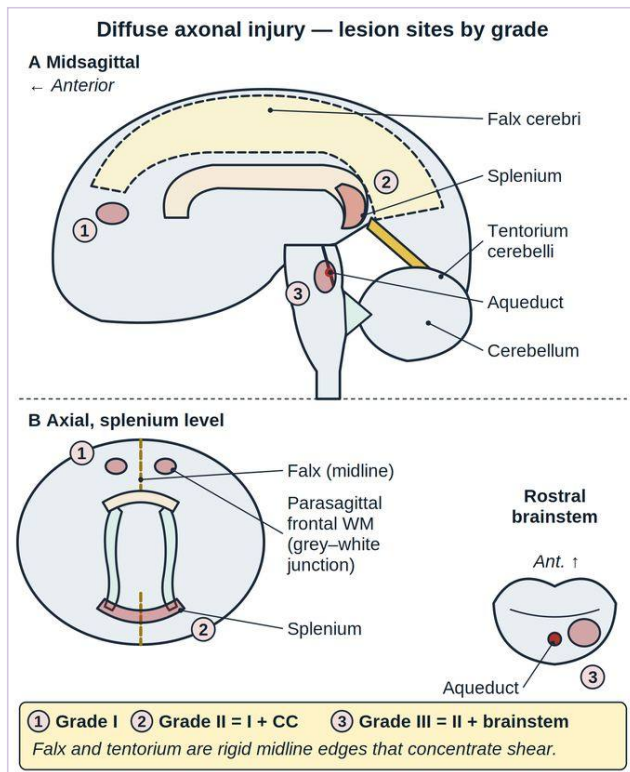
YW p.2973

- Predicts length of coma and persistent neurological deficit. **YW p.2973**

2. The same grading with macroscopic and microscopic detail

- **Grade 1** — widespread axonal damage in corpus callosum, hemispheric white matter, brainstem and cerebellum with **no** macroscopic marker lesion; conventional imaging may be normal.
- **Grade 2** — plus a focal abnormality, usually a small haemorrhage, in the **corpus callosum** (classically the splenium).
- **Grade 3** — plus a small focal lesion in the **rostral brainstem** (dorsolateral rostral pons/midbrain, sometimes with blood in the aqueduct).

Grade 2 and 3 marker lesions may be visible on neuroimaging. **YW p.2912.e9**



Schematic diagram — Diffuse axonal injury — lesion sites by grade

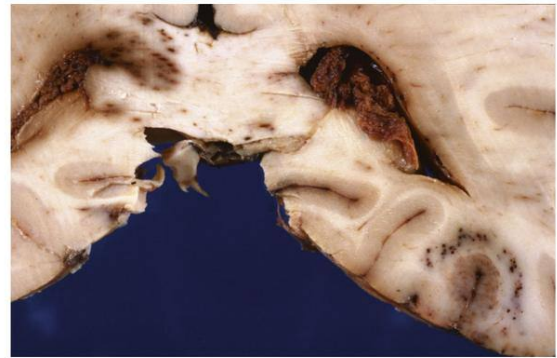


Figure 375.14. Splenium of the corpus callosum showing scattered petechial hemorrhages in diffuse axonal injury grade 2. Note the crescent of subcortical petechial hemorrhages beneath the congested cerebral cortex and the hemorrhage in the occipital horns.

Original figure: Youmans & Winn 8e, Fig. 375.14, p. 2912.e9

3. Clinical grading by duration of coma

Severity	Criterion
Concussion	Traumatic loss of consciousness <6 hours
Mild DAI	Coma 6–24 hours
Moderate DAI	Coma >24 hours without decerebrate posturing
Severe DAI	Coma >24 hours with decerebrate posturing or flaccidity (~50% mortality)

YW p.3007

4. Place within the anatomical classification of head injury

- Diffuse injuries (concussion, DAI — moderate and severe) versus focal injuries (contusion, fracture, herniation, haematoma). YW p.2970, p.2973

High-yield: Adams grades I–III — parasagittal white matter, corpus callosum, cerebral peduncle.

Youmans & Winn 8e — pp. 2912.e9, 2912.e10, 2926, 2969, 2970, 2973, 3007

Q C7

DEC 2020 · Paper I · Q3 · 5+5 marks

- Pathogenesis and pathology of traumatic axonal injury.
- How DAI is graded?

ANSWER

(a) Pathogenesis and pathology of traumatic axonal injury [5]

Terminology

- The syndrome first called diffuse axonal injury is **more appropriately designated traumatic axonal injury**

(TAI) because it may be focal, multifocal or diffuse. There is a spectrum, the severe end correlating with persistent vegetative state and the mild end with concussive syndromes. YW p.2912.e9, p.2912.e10

Mechanism of loading

- The principal loading is **rotational acceleration of the unrestricted head**, producing shear, stretching and compressive strains. The large human brain generates high inter-regional shear strain, and the falx cerebri and tentorium cerebelli impede brain motion — hence the midline concentration of TAI. YW p.2912.e9
- TAI can occur without impact, but many fatal injuries follow impact; non-impact rotational acceleration in a crash may be followed by single or multiple head impacts. Low-magnitude, long-duration acceleration propagates deep and causes DAI; brief high-velocity impact tears bridging veins and causes SDH. YW p.2912.e9, p.2969

Pathogenesis — a progressive secondary process

- Transient **mechanoporation of the axolemma**, particularly at nodes of Ranvier, permitting unregulated calcium entry. YW p.2926, p.3007
 - Calcium initiates **calpain activation**, mitochondrial swelling with cytochrome c release and caspase activation, bioenergetic depletion and apoptosis. YW p.2926, p.2912.e10
 - Calcium-linked dephosphorylation causes **neurofilament compaction**; the cytoskeleton collapses as tau, dynein and kinesin detach from microtubules. YW p.2912.e10
 - Axoplasmic transport fails** — focal accumulation of neurofilaments, membranous organelles and APP with progressive swelling. YW p.2912.e10
 - Over **6–12 hours** the proximal segment disconnects from the distal; severed ends retract as bulbs and the distal segment undergoes **Wallerian degeneration** over weeks to months. YW p.2912.e10
- Proteolysis releases protein fragments with potential as biomarkers; microdialysis shows raised extracellular tau correlating with DTI abnormality. Axons look normal initially — the alterations **mature over hours to days and are therefore potentially treatable**. YW p.2912.e9, p.2912.e10

Pathology

- Gross:** small haemorrhagic marker lesions in deep white matter, corpus callosum (especially splenium) and rostral brainstem; Strich petechial haemorrhages in peri-third-ventricular structures, internal capsule, basal ganglia, dorsolateral midbrain and superior cerebellar peduncles. YW p.2912.e9, p.2973
- Microscopic:** axonal swellings, varicosities, retraction bulbs and axoplasmic "retraction balls" at points of discontinuity, chiefly on large myelinated fibres, at high density in about 25% of severe head injuries. YW p.2926, p.2912.e9
- Immunohistochemistry:** beta-APP has supplanted silver impregnation — positive within **35 minutes** and almost universal in fatal TBI, against ~30% with silver stains; neurofilament proteins and tubulins are alternative targets. YW p.2912.e9, p.2912.e10
- Vascular axonal injury** must be distinguished — bulbs clustered in a zigzag or geographic pattern around ischaemic brain from raised ICP and herniation. YW p.2912.e10
- Retraction bulbs are **non-specific**, also seen beside haematomas, abscesses, tumours and in demyelination; CLARITY suggests many are varicosities of intact axons, implying more salvageable axons and a longer therapeutic window. Long term, APP-positive axons persist for years with intra-axonal amyloid-beta. YW p.2912.e9, p.2912.e10

(b) How is DAI graded? [5]

A. Adams neuropathological grading (1989) — the standard answer

Grade	Lesion	localisation	Imaging	correlate
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I	Parasagittal white matter of the cerebral hemispheres; no macroscopic marker lesion	Conventional imaging often normal
II	Grade I + focal lesion (usually small haemorrhage) in the corpus callosum , classically the splenium	May be visible on MRI
III	Grade II + focal lesion in the cerebral peduncle / rostral brainstem (dorsolateral rostral pons)	May be visible on MRI

YW p.2973, p.2912.e9

- Grading uses combined macroscopic and microscopic lesions, originally by silver impregnation to identify axonal swellings and bulbs; it **predicts length of coma and persistent neurological deficit**. YW p.2912.e9, p.2973

B. Clinical grading by duration of coma

Severity	Criterion
Concussion	Loss of consciousness <6 hours
Mild DAI	Coma 6–24 hours
Moderate DAI	Coma >24 hours without decerebrate posturing
Severe DAI	Coma >24 hours with decerebrate posturing or flaccidity; ~50% mortality

YW p.3007

C. Radiological descriptor

- "DAI" is recommended as a **neuroimaging descriptor when TAI lesions are present in three or more discrete sites**. Coronal/lateral acceleration produces the most severe DAI, oblique or sagittal the least. YW p.2973

D. Caveats

- DAI is primarily a **pathological** diagnosis, classically confirmed only by beta-APP immunostaining at autopsy, so in life the grade is inferred from imaging. YW p.3007
- Less than 20% of TAI is haemorrhagic**, so any haemorrhage-based grade systematically under-grades the injury. Location and severity of axonal injury determine functional recovery. YW p.2966.e13, p.2973

Youmans & Winn 8e — pp. 2912.e9, 2912.e10, 2926, 2966.e13, 2969, 2973, 3007

Q C8

DEC 2021 · Paper I · Q8

Traumatic diffuse axonal brain injury:

- Pathology. [3]
- Radiological features. [3]
- Management. [4]

ANSWER

(a) Pathology [3]

Mechanism

- DAI results from severe angular and rotational acceleration–deceleration delivering shear and tensile forces to axons; severe impairment may occur without gross contusion, laceration or haematoma. Midline concentration reflects restraint of brain motion by the falx cerebri and tentorium cerebelli. YW p.2973,

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CHAPTER 11

Cerebrovascular II: AVM, Fistulae, Stroke & Venous Disease

18

QUESTIONS SOLVED

Arteriovenous Malformation (cerebral & spinal)

5 questions

Q K1

JUN 2020 · Paper II · Q4 · 5+2+3 marks

Spinal AVM:

- a) Classification.
- b) Clinical presentations.
- c) Angiographic finding.

ANSWER

(a) Classification [5]

Current (Spetzler/Barrow) anatomic classification

The first question on angiography is whether an arteriovenous **nidus** is present: no nidus = AVF, nidus = AVM. Lesions are then named by anatomic descriptors (intradural vs extradural, intramedullary vs extramedullary, ventral vs dorsal), giving six categories that guide treatment. **YW p.3779–3780**

Type	Anatomy of the shunt	Classical (Di Chiro) name
1. Extradural AVF	Epidural segment of a radicular artery to the epidural venous plexus, before the artery enters the dural sleeve; engorged epidural veins compress thecal sac; steal	Not separately recognised (Borden I)
2. Intradural dorsal AVF	Radicular artery to a medullary/radicular vein within the dural root sleeve ; commonest spinal vascular malformation	Type I (Borden III)
3. Intradural ventral AVF	Anterior spinal artery to coronal venous plexus in the subarachnoid space, midline	Type IV (perimedullary)
4. Extradural-intradural AVM	Metameric lesion involving bone, muscle, skin, nerve and cord; whole somite = Cobb syndrome	Type III (juvenile)
5. Intramedullary AVM	True cord-parenchymal AVM fed by anterior/posterior spinal arteries with a nidus; compact or diffuse	Type II (glomus)
6. Conus medullaris AVM	Multiple feeders from anterior and posterior spinal arteries, diffuse, large venous ectasia at conus/upper cauda equina	Not recognised

YW p.3780–3783

Anson-Spetzler subtypes of intradural ventral AVF — A: small simple fistula, single anterior spinal artery feeder; B: larger, primary anterior spinal artery feeder plus small adjacent feeders; C: giant fistula, multiple pedicles, immensely engorged venous outflow. **YW p.3781**

Merland subtypes of perimedullary (pial) AVF — I slow flow/mild venous hypertension; II multiple feeders

from the anterior spinal artery, higher flow; III giant fistula with prominent venous dilation. **YW p.3803**

Oldfield biologic classification — dural AVFs; intradural AVMs (cord AVMs: juvenile, glomus; perimedullary AVFs); cavernous angiomas. **YW p.3805**

(b) Clinical presentations [2]

- **Dural (intradural dorsal) AVF** — 80% men, symptoms after 40 years, lower thoracic/lumbar; low back or radicular pain followed by gradual progressive myelopathy in 85% to 95%, with weakness, sensory disturbance, bowel and bladder dysfunction and neurogenic claudication; arms essentially never involved. Untreated, gradual motor and sensory decline over 2 to 3 years. **YW p.3804–3805**
- **Intradural AVM** — mean age 24 years, acute onset in 37%, SAH in 50%, paresis or SAH as first symptom, spinal bruit in 6%, arms affected in 11%. **YW p.3806**
- **Foix-Alajouanine syndrome** — acute deterioration from extreme venous hypertension with subsequent venous thrombosis; a true emergency, reversible if venous hypertension is relieved before thrombosis. **YW p.3805**

(c) Angiographic finding [3]

Selective spinal arteriography remains the **gold standard** and defines four components: type of malformation, spinal level and configuration of fistula/nidus, feeding vessels, and the normal cord vasculature. **YW p.3810**

- **Intradural dorsal (dural) AVF** — injection of the feeder shows the fistulous point in the intervertebral foramen/lateral canal with a single arterialised medullary vein running into a long, tortuous, dilated coronal venous plexus, usually dorsal; flow is slow, so prolonged venous-phase views (~15 seconds) are needed. Nidus lateral in canal in 100%, drainage rostral in 100%. **YW p.3781 YW p.3810 YW p.3811**
- **Intradural ventral AVF** — midline fistulous pouch on AP views (vs lateral pouch in dorsal AVF). **YW p.3781**
- **Intramedullary AVM** — compact tangled glomus nidus within a short cord segment fed by anterior +/- posterior spinal arteries; rapid flow in 80%; associated intranidal/feeding-artery aneurysms in 44%; feeder shared with a medullary artery in 100%. **YW p.3811**
- If arteriography is negative but MRI/CTA shows a dilated coronal plexus, inject the cranial vessels — posterior fossa/tentorial dural AVFs can drain into cord veins and mimic a spinal dural AVF. Also inject both vertebral and the iliac arteries. **YW p.3810–3811**

High-yield: cavernous angiomas of the cord are **angiographically occult** — MRI is definitive. **YW p.3809**

Youmans & Winn 8e — pp. 3779, 3780, 3781, 3782, 3783, 3803, 3804, 3805, 3806, 3809, 3810, 3811

Q K2

APR 2023 · Paper II · Q6

- a) Clinical presentations of cerebral AVMs. [4]
- b) Molecular biology and genetics of AVMs. [2]
- c) Classification of AVM. [4]

ANSWER

(a) Clinical presentations of cerebral AVMs [4]

Presentation falls into five categories: intracerebral haemorrhage, seizures, progressive neurological deficit, headache, and incidental discovery. **YW p.3695**

1. **Haemorrhage** — the commonest and most dangerous. 45% to 72% of patients in modern series present with bleeding; AVMs cause one-third of haemorrhagic strokes in young adults and 38% of intracerebral haemorrhages in patients aged 15 to 45 years. Intraparenchymal bleeding is commonest, then intraventricular

and subarachnoid. First bleed usually at 20 to 40 years. Mortality 10% to 30% and permanent morbidity 20% to 30% per haemorrhage. **YW p.3695**

2. **Seizures** — second commonest with supratentorial AVMs, occurring in almost 70%; seizure without haemorrhage in 25% to 50%; diagnosis made during seizure work-up in 18% to 35%. Mean age of onset 25 years; simple and complex partial types predominate. More frequent with large size, MCA distribution, cortical nidus or feeders, and venous varices. **YW p.3695**
3. **Progressive neurological deficit** — 3% to 10%, from mass effect (direct pressure or perinidal oedema) or ischaemic steal through the high-flow, low-resistance shunt. **YW p.3695**
4. **Headache** — 6% to 14% present with chronic headache without haemorrhage; typically hemicranial, migraine-like. Occipital AVMs are more prone; headache is consistently on the side of the AVM with contralateral visual phenomena, which distinguishes it from classical migraine. **YW p.3695–3696**
5. **Incidental/asymptomatic** — now 10% of modern series (historically under 2%); 40% were asymptomatic in one population-based study. **YW p.3696**

(b) Molecular biology and genetics of AVMs [2]

- AVMs are predominantly **sporadic**, with three morphologic components — feeding arteries, draining veins and a dysplastic nidus of arteriovenous shunts with no interposed brain and no capillary bed. **YW p.3659**
- Classically congenital, but increasing evidence shows up- or down-regulation of multiple **homeobox genes** involved in angiogenesis; AVMs are dynamic and may enlarge, regress, obliterate or recur. **YW p.3659**
- **Somatic activating KRAS mutations** (p.Gly12Val/Asp) activate the MAPK-ERK pathway in brain endothelial cells and are inhibited in vitro by a MEK inhibitor; next-generation sequencing shows KRAS/BRAF mutations in 87.1% of AVMs, raising the prospect of Ras/Raf-directed medical therapy. **YW p.3669**
- Whole-exon sequencing confirms the role of **BMP/TGF-beta** and **VEGF/VEGF-receptor** signalling; microRNA-18a normalises dysfunctional AVM endothelium by lowering VEGF and raising thrombospondin-1. **YW p.3669**
- Proposed cascade: an inciting event (microvascular thrombosis) causes venous hypertension, which elaborates **HIF-1**, then VEGF-driven focal angiogenesis, IL-6 and leukocyte recruitment, and **MMP-9** activation that destabilises the vessel wall and sets the stage for rupture. Tetracyclines are in trial as vasculostatic agents. **YW p.3669**
- **Syndromic associations** — hereditary haemorrhagic telangiectasia (Osler-Weber-Rendu), autosomal dominant, due to mutation in the transforming growth factor beta receptor gene; Sturge-Weber (encephalotrigeminal angiomatosis); Wyburn-Mason (retina, optic nerve, diencephalon AVMs with cutaneous naevi). **YW p.3695 YW p.3659**

(c) Classification of AVM [4]

Histopathologic classification of cerebrovascular malformations (McCormick): true AVM, venous angioma, cavernous malformation, capillary telangiectasia; a possible fifth group is the acquired direct fistulas (dural AVF, carotid-cavernous fistula, vein of Galen malformation). **YW p.3659**

Spetzler-Martin grading — grade = size + eloquence + venous drainage; grades I to V. It predicts risk of surgical resection, not risk of haemorrhage. **YW p.3698 YW p.3677.e2**

Graded feature	Points
Size small (<3 cm)	1
Size medium (3–6 cm)	2
Size large (>6 cm)	3

Non-eloquent adjacent brain	0
Eloquent adjacent brain	1
Superficial venous drainage only	0
Deep venous drainage	1

Eloquent = sensorimotor, language and visual cortex, hypothalamus and thalamus, internal capsule, brainstem, cerebellar peduncles, deep cerebellar nuclei. Deep drainage = any drainage into internal cerebral, basal or precentral cerebellar veins. **YW p.3677.e2 YW p.3699**

Spetzler-Ponce three-tier condensation — class A = SM I-II, class B = SM III, class C = SM IV-V. **YW p.3677.e2**

Supplementary (Lawton-Young) grade, added to the SM grade: age <20 years = 1, 20-40 years = 2, >40 years = 3; diffuse nidus = 1; unruptured = 1, giving a combined range of 2 to 10. **YW p.3677.e8**

Subdivisions of grade III — Spetzler-Martin S2V1E0 (intermediate risk); de Oliveira IIIA (large) treated by embolisation and surgery, IIIB (small with deep drainage or eloquent) treated by radiosurgery. **YW p.3699**

Endovascular scores — AVM Embocure Score (AVMES, 3-10) and the Buffalo score (0-5), based on number and calibre of pedicles, draining veins, nidus size and eloquence. **YW p.3686**

Youmans & Winn 8e — pp. 3659, 3669, 3677.e2, 3677.e8, 3686, 3695, 3696, 3698, 3699

Q K3

OCT 2023 · Paper II · Q9

- Clinical manifestations of spinal AVMs. [4]**
- Spinal AVMs classification. [3]**
- Spinal arachnoiditis - radiology and treatment. [3]**

ANSWER

(a) Clinical manifestations of spinal AVMs [4]

Dural (intradural dorsal) arteriovenous fistula

- Acquired lesion; **80% are men** and symptoms begin **after 40 years**. Strong predilection for the lower thoracic and lumbar spine, so upper-limb involvement is essentially never seen. **YW p.3804**
- History typically of low back or radicular pain, then **gradual progressive myelopathy in 85% to 95%** — increasing weakness, sensory disturbance, bowel and bladder dysfunction. **YW p.3804**
- Neurogenic claudication** — worsening with walking or certain postures; symptoms exacerbated by activity in 70%. This mimics lumbar stenosis, and fistulas of the filum terminale coexist with lumbosacral degenerative disease, causing delayed or missed diagnosis and unnecessary decompressive surgery. **YW p.3804-3805**
- Foix-Alajouanine syndrome** — acute deterioration from extreme venous hypertension with subsequent venous thrombosis. A true emergency: relief of venous hypertension before thrombosis can reverse deficits, failure leads to irreversible cord injury. **YW p.3805**
- Untreated, 20% needed crutches or were non-ambulant within 6 months of onset; by 3 years half were in a wheelchair or on crutches; overall a gradual decline over 2 to 3 years. **YW p.3804-3805**

Intradural (intramedullary/perimedullary) AVM

- Congenital; equal sex incidence; mean age at diagnosis **24 years**; distributed evenly along the cord. **YW p.3805-3806**
- Onset subacute or **acute in 37%**, the most dramatic from haemorrhage: sudden back or suboccipital pain,

meningismus, loss of consciousness, sudden motor deterioration. SAH or intramedullary haemorrhage is the initial imaging finding in about **35%**; children are more prone (acute onset in 84%). **YW p.3805**

- Comparative features: SAH 50% vs 0% for dural AVF; spinal bruit 6%; arms affected 11%; symptoms exacerbated by activity only 15%. **YW p.3806**
- Conus medullaris AVMs uniquely give **both upper and lower motor neuron signs**, with a cauda equina syndrome from compression of exiting roots. **YW p.3783**

Feature	Dural AVF	Intradural AVM
Mean age	46 yr	24 yr
Sex	Predominantly male	Male or female
Onset	Gradual (85%)	Acute (37%)
SAH	0	50%
First symptom	Paresis (44%)	SAH (32%)
Arms affected	0	11%

YW p.3806

(b) Spinal AVMs classification [3]

Spetzler anatomic system — first decide nidus (AVM) vs no nidus (AVF), then use anatomic descriptors. Six categories: **YW p.3780**

Type	Shunt anatomy	Old name
Extradural AVF	Radicular artery to epidural venous plexus	Borden I
Intradural dorsal AVF	Radicular artery to medullary vein in dural root sleeve; commonest	Di Chiro type I
Intradural ventral AVF	Anterior spinal artery to coronal venous plexus, subarachnoid; Anson-Spetzler A/B/C	Di Chiro type IV
Extradural-intradural AVM	Metameric, bone/muscle/skin/nerve; whole somite = Cobb syndrome	Di Chiro type III (juvenile)
Intramedullary AVM	Nidus in cord parenchyma, compact or diffuse	Di Chiro type II (glomus)
Conus medullaris AVM	Multiple anterior and posterior spinal artery feeders, venous ectasia	Not previously recognised

YW p.3780–3783

Oldfield's biologic grouping is also used: dural AVFs; intradural AVMs (cord AVMs - juvenile and glomus; perimedullary AVFs); cavernous angiomas. **YW p.3805**

(c) Spinal arachnoiditis - radiology and treatment [3]

Definition and causes — spinal adhesive arachnoiditis (SAA) is scarring of pial and arachnoid membranes within the thecal sac following chronic inflammation, ranging from focal adhesions to arachnoiditis ossificans. Triggers: infection, myelography, subarachnoid haemorrhage, degenerative lumbar disease, previous spine surgery and intrathecal steroid injection. Latency 14 days to 44 years; predilection for the lumbar spine. **YW p.2604**

Radiology — CT myelography was traditional but **MRI has supplanted it** (sensitivity 92%, specificity 100% vs CT myelography). CISS sequences show dural thickening, syringomyelia and microcystic change better than T2 fast spin echo. Ross and Delamarter grading: **YW p.2604-2605**

Grade	MRI findings
1	Conglomeration of adherent nerve roots centrally within the thecal sac
2	Peripherally adherent roots, "empty thecal sac" appearance, meningeal thickening
3	Soft tissue mass in the canal with obliteration of the subarachnoid space

Findings may be subtle or absent (a single arachnoid web can cause CSF obstruction); myelography then adds information. **YW p.2604-2605**

Treatment — once symptomatic the course is generally progressive. Temporary relief has been reported with oral steroids and with intrathecal hyaluronidase; intrathecal steroid is limited because it can itself cause arachnoiditis. Surgery is for progressive deficit or severe pain: **arachnolysis with duraplasty** is preferred (97% of shunted patients recurred vs 78% stable after arachnoid release and duraplasty). Over-aggressive arachnolysis in severe scarring worsens patients — only 17% with severe scarring stabilised vs 83% with mild scarring, and post-infectious arachnoiditis fares worst. Extensive scarring is better managed by **CSF diversion** (syringosubarachnoid, syringocavitary, syringopleural or lumboperitoneal shunt). **YW p.2604-2606**

Youmans & Winn 8e — pp. 2604, 2605, 2606, 3780, 3781, 3782, 3783, 3804, 3805, 3806

Q K4

MAY 2024 · Paper III · Q10

Please see the imaging study carried out in a 15-year-old female patient presenting to the emergency in an unconscious status after initial headache. Discuss briefly:

- Radiological findings. [2]**
- Role of Gamma knife. [2]**
- Role of Endovascular therapies. [2]**
- Surgical nuances. [2]**
- Postoperative care. [2]**

ANSWER

(Answered for the standard stem: a teenager rendered unconscious after sudden headache by a ruptured cerebral arteriovenous malformation.)

(a) Radiological findings [2]

- Non-contrast CT** — the acute intraparenchymal haematoma with intraventricular extension and hydrocephalus, which explains the unconscious state; also calcification or encephalomalacia from a previous bleed. Contrast CT shows nidus, feeders and draining veins and is a valuable quick study before emergency evacuation. **YW p.3697**
- MRI** — tightly packed "**honeycomb**" of **flow voids** on T1 and T2 with perinidal mass effect and oedema; unhelpful acutely because the haematoma obscures the AVM. **YW p.3698**
- DSA is the gold standard** — size, compact vs diffuse nidus, feeders, drainage, and high-risk features (feeding-artery or intranidal aneurysm, deep venous drainage, venous outflow compromise, perforator supply, deep location). Vessels compressed by clot may appear only on a repeat study weeks later. **YW p.3698**
- AVM rupture causes one-third of haemorrhagic strokes in young adults and 38% of intracerebral haemorrhage at 15 to 45 years. **YW p.3695**

(b) Role of Gamma Knife [2]

- Obliterates by **progressive intimal hyperplasia and nidus thrombosis**. YW p.3711
- **Ideal for AVMs under 3 cm in eloquent or deep locations** where surgical morbidity would be unacceptable, or when comorbidity precludes anaesthesia — here, for a deep or eloquent residual nidus. YW p.3711
- Obliteration is volume dependent: 100% under 4 cm³, under 50% for deep lesions; 64.7% overall in 2236 patients, with a 1.1% annual post-radiosurgery haemorrhage rate. YW p.3711
- **Two disadvantages** — latency of 1 to several years during which bleeding risk persists, and uncertainty of cure. Radiation-induced changes at 6 to 18 months in about 35%, symptomatic in 9%, permanent in 4%; recurrence after apparent cure is described in children. YW p.3711

(c) Role of endovascular therapies [2]

- **Not a stand-alone therapy** and not routine before microsurgery; used for a defined goal — a proximal perinidal aneurysm, an inaccessible feeder, an intranidal aneurysm identified as the source of rupture, deep perforator supply, or dural/transosseous feeders that would bleed at craniotomy. YW p.3639 YW p.3713
- **Palliative embolisation** has not been shown to help: annual haemorrhage 1% in conservatively treated grade IV-V AVMs vs 10.4% in partially treated ones. YW p.3713
- **Curative embolisation** works only in superficial, non-eloquent, low-grade AVMs with few large straight pedicles (92% to 98% cure in grades I-II); overall cure 5% to 30%. YW p.3686 YW p.3713 YW p.3714
- **Before radiosurgery** it lowers obliteration (49.5% vs 70.4% for Gamma Knife alone) and is not recommended for that purpose. YW p.3713

(d) Surgical nuances [2]

- **Timing** — with a ruptured AVM and no major deficit, operate at 4 to 6 weeks so the clot liquefies, oedema settles and dysautoregulated brain recovers; for life-threatening haemorrhage evacuate clot only. Repeat the angiogram close to surgery. YW p.3702
- Intraoperative angiography, radiolucent pin fixation, femoral sheath; large craniotomy with a few centimetres of margin; open dura carefully to protect adherent draining veins. YW p.3702
- **Sequence** — correlate cortex with the angiogram, follow a superficial feeding artery into the sulcus (safer than following the vein), divide feeders close to the nidus after excluding **en passage** vessels. Temporary clipping distinguishes vessels: a vein goes slack and blue, an artery keeps pulsating. YW p.3702
- Circumferential **spiral** dissection, on the AVM loops in eloquent brain, a few millimetres away elsewhere; take the main draining vein last. Deep subependymal perforators retract and resist bipolar — use **Sundt microclips**. Never pack bleeding brain away from the nidus. YW p.3702 YW p.3703

(e) Postoperative care [2]

- At the end of resection raise **mean arterial pressure 15 to 20 mm Hg above baseline for 10 minutes** — any bleeding suggests residual nidus. Then keep blood pressure **at or slightly below normal for 24 hours** and confirm resection with intraoperative angiography. YW p.3703
- **Haemorrhage** is the most immediate and morbid complication, from residual nidus or poor haemostasis; **normal perfusion pressure breakthrough** is predicted by large-calibre, long, high-flow feeders. Also retrograde feeding-artery thrombosis, retrograde venous thrombosis and (rarely) vasospasm. YW p.3711
- Proximal arteries **remodel** over about 7 days for an anterior circulation AVM under 3 cm with a small feeder, longer otherwise; active blood pressure reduction may be needed in that window. Imaging: ICG videoangiography, early CTA, and DSA at 7 days, since a residual nidus with occluded outflow can be invisible immediately. YW p.3677.e10

- **Seizure prophylaxis** — load perioperatively and continue 6 to 12 months after supratentorial resection, weaning if seizure-free with a normal EEG; 15% of previously seizure-free patients develop new seizures. **YW p.3711 YW p.3677.e10**

Youmans & Winn 8e — pp. 3639, 3677.e10, 3686, 3695, 3697, 3698, 3702, 3703, 3711, 3713, 3714

Q K5

APR 2026 · Paper II · Q1 · 10 marks

Management of cerebral AVM-various gradings and treatment.

ANSWER

Natural history — the basis for any decision

Treatment is justified only if its risk is lower than the lifetime risk of the untreated AVM. **YW p.3696** Reported annual haemorrhage rates: Ondra 4%; Hernesniemi 2.4% overall (4.6% in the first 5 years, 1.6% thereafter); Stapf 1.3% for unruptured and 5.9% for AVMs presenting with haemorrhage. **YW p.3696-3697** Risk is stratified by deep venous drainage and deep location — from 0.9% per year (superficial, no deep drainage, never bled) to 34.3% (deep location, deep drainage, prior bleed). **YW p.3697** Cumulative lifetime rupture risk is approximated by **105 minus the patient's age in years**, which justifies a more aggressive stance in the young. **YW p.3701**

Grading systems

Spetzler-Martin grade = size + eloquence + venous drainage (grades I-V). It predicts the risk of resection, not the risk of bleeding. **YW p.3698**

Feature	Points
Size <3 cm / 3-6 cm / >6 cm	1 / 2 / 3
Adjacent brain non-eloquent / eloquent	0 / 1
Venous drainage superficial only / any deep	0 / 1

Eloquent = sensorimotor, language and visual cortex, hypothalamus and thalamus, internal capsule, brainstem, cerebellar peduncle, deep cerebellar nuclei. Deep drainage = internal cerebral, basal vein or precentral cerebellar veins. **YW p.3677.e2 YW p.3699**

Spetzler-Ponce classification (SPC) — class A = SM I-II, class B = SM III, class C = SM IV-V. **YW p.3677.e2**

Supplementary (Lawton-Young) grade — add to the SM grade: age <20 = 1, 20-40 = 2, >40 = 3; diffuse nidus = 1; unruptured = 1. Combined range 2-10. It matters most in SPC class B, where adverse outcome risk is roughly 10%, 20%, 25%, 40% and 65% for grades 4, 5, 6, 7 and 8. **YW p.3677.e8**

Grade III subdivision (de Oliveira) — IIIA large, treated by embolisation plus surgery; IIIB small with deep drainage or eloquent location, treated by radiosurgery. **YW p.3699**

Embolisation-specific scores — AVM Embocure Score (3-10) and Buffalo score (0-5), using number and calibre of pedicles, number of draining veins, nidus size and eloquence. **YW p.3686**

The Spetzler-Martin scale does not capture pattern of arterial supply, compact vs diffuse nidus, feeding-artery aneurysms, or the individual surgeon's experience. **YW p.3699**

Treatment options

Microsurgery — the definitive treatment for accessible AVMs. Ruptured AVMs without major deficit are resected electively at 4 to 6 weeks; for life-threatening haemorrhage only the clot is evacuated. Technique: large craniotomy, superficial feeders divided close to the nidus after excluding en-passage vessels, circumferential spiral dissection, main draining vein taken last, Sundt microclips for deep perforators, intraoperative

angiography. **YW p.3702–3703**

Stereotactic radiosurgery — obliteration by intimal hyperplasia and nidus thrombosis; ideal for AVMs <3 cm in eloquent or deep locations. Obliteration 54% to 65% overall and volume dependent; latency of 1 to several years with persisting bleeding risk (1.1% per year); radiation-induced changes in about 35%, symptomatic in 9%, permanent in about 4%. **YW p.3711**

Endovascular embolisation — adjuvant in most cases. Cure rates 5% to 30% overall (up to 96% when selected for cure, 92% to 98% for SM I-II). Palliative embolisation does not improve natural history (annual haemorrhage 10.4% in partially treated grade IV-V vs 1% conservatively treated). Pre-radiosurgical embolisation reduces obliteration (49.5% vs 70.4%) and is not recommended for that indication. **YW p.3686 YW p.3713–3714**

ARUBA and its critique

Item	Result
Design	Multicentre RCT, unruptured brain AVMs, intervention vs medical management
Enrolment	Revised from 800 to 400; stopped early with 223 patients (109 medical, 114 intervention), mean follow-up ~33 months
Primary endpoint	Any stroke or death: 10% medical vs 29% intervention
By grade	Similar for SM I-II; significantly worse for treated grades III and IV
Mortality	Similar in both arms

YW p.3714–3715 Criticisms: short follow-up in young patients with lifetime cumulative risk; most patients treated by stand-alone endovascular therapy, the highest-risk modality; no stratification of interventional arms; selection and recruitment bias; over-general inclusion criteria. Post-ARUBA surgical series in ARUBA-eligible patients report 93% to 98.1% obliteration with 3.2% to 11.6% permanent morbidity. **YW p.3715**

Management algorithm

- **Unruptured low grade (SM I-II)** — surgery and radiosurgery are equivalent first-line options; observation is not recommended, particularly under 40 years. Embolisation only for a defined target such as a perinidal aneurysm. **YW p.3639**
- **Unruptured high grade (SM III-V)** — radiosurgery first line, single-session or volume-staged; microsurgery +/- embolisation second line or for radiosurgical failure. **YW p.3639**
- **Ruptured low grade** — microsurgery is superior to radiosurgery because of the higher rebleed risk and the enhanced resectability of ruptured lesions. **YW p.3639**
- **Ruptured high grade** — microsurgery is a potential first-line option, with supplementary grading used to quantify surgical risk. **YW p.3639**
- Management pathway also weighs patient age, comorbidity, occupation and lifestyle, and the surgeon's audited results. **YW p.3701**

High-yield: SPC class A (SM I-II) resection carries very low risk and is the benchmark for auditing an early-career cerebrovascular surgeon's practice. **YW p.3677.e2**

Youmans & Winn 8e — pp. 3639, 3677.e2, 3677.e8, 3686, 3696, 3697, 3698, 3699, 3701, 3702, 3703, 3711, 3713, 3714, 3715

Moyamoya Disease & Revascularisation

2 questions

Q K6

OCT 2023 · Paper II · Q2

- a) Differential diagnosis of young stroke. [4]
- b) Various stages of Moya-Moya disease. [3]
- c) Indirect Revascularization procedures. [3]

ANSWER

(a) Differential diagnosis of young stroke [4]

Atherosclerosis of large arteries and small-vessel occlusive disease are **not** common causes in the young, so the differential is different from that of the elderly. YW p.1597

Arterial ischaemic stroke

1. **Cardiac** — congenital or acquired heart disease is the commonest risk factor; cardiac disease accounted for 12% in a large population-based series. YW p.1597
2. **Arteriopathy** (24% in the same series) — arterial dissection, moyamoya disease and vasculitis. Cervical carotid dissection accounts for about 2% of all ischaemic strokes but **20% of strokes in young adults**; classical triad of ipsilateral face/head/neck pain, partial Horner syndrome and TIA or stroke. YW p.1597 YW p.3323
3. **Infection** — 23%; antecedent varicella-zoster infection is associated with arteriopathy and basal ganglia strokes. YW p.1597
4. **Haematological** — sickle cell disease; protein C, protein S or antithrombin III deficiency; activated protein C resistance; factor V Leiden; prothrombin G20210A; MTHFR TT677; antiphospholipid antibody syndrome. YW p.1597–1598
5. **Autoimmune disorders**. YW p.1597
6. Other non-atherosclerotic carotid lesions — fibromuscular dysplasia, radiation-induced stenosis, extracranial carotid aneurysm, carotid body tumour. YW p.3323

Haemorrhagic stroke

- Ruptured **AVM** — causes one-third of haemorrhagic strokes in young adults and 38% of intracerebral haemorrhage at 15 to 45 years, second only to aneurysm. YW p.3695
- Ruptured aneurysm; moyamoya haemorrhage; cavernous malformation. YW p.3695 YW p.1863

Stroke mimics

Metabolic and mitochondrial disease, migraine and seizure. YW p.1597

Investigation: CT or MRI first; MRI is preferred when ischaemia is suspected; add CTA/MRA if dissection is possible. YW p.1597

(b) Various stages of Moyamoya disease [3]

Cerebral angiography is the basis of diagnosis. Youmans presents the staging as the **six-stage classification of Suzuki and Takaku** and describes the progression narratively: from early carotid narrowing, to formation of basal moyamoya collaterals, to the disappearance of these moyamoya vessels, and finally to maintenance of the cerebral circulation only through the external carotid and vertebrobasilar systems. The stage names below are those of the original description cited there. Progression from stage 1 to stage 6 is observed in only a limited number of cases, and **stage 4 is encountered most frequently**. YW p.3363

Stage	Angiographic feature
I	Narrowing of the carotid fork; no moyamoya vessels
II	Initiation of moyamoya — dilated basal collaterals appear with dilated main cerebral arteries

III	Intensification of moyamoya — basal moyamoya most prominent, ACA and MCA filling defective
IV	Minimisation of moyamoya — moyamoya vessels begin to diminish; posterior cerebral artery also becomes occluded; ECA collaterals develop
V	Reduction of moyamoya — further reduction of basal moyamoya, all main cerebral arteries absent from the internal carotid
VI	Disappearance of moyamoya — internal carotid completely occluded; cerebrum supplied only by the external carotid and vertebrobasilar systems

Collateral channels that define these stages are (1) **basal moyamoya** in basal ganglia and thalamus via lenticulostriate, anterior and posterior choroidal and posterior communicating arteries; (2) **ethmoidal moyamoya** via anterior and posterior ethmoidal branches of the ophthalmic artery; and (3) **vault moyamoya** via dural (transdural) arteries. **YW p.3362–3363**

Surgical relevance: revascularisation is offered for symptomatic disease at **any Suzuki stage**, and for asymptomatic disease at **Suzuki II to VI** with evidence of progressive ischaemic change; asymptomatic hemispheres with low stage I to II and normal perfusion are not operated. **YW p.1864 YW p.1866**

(c) Indirect revascularization procedures [3]

Principle — moyamoya spares the external carotid artery, so vascularised ECA-supplied tissue (vessel, muscle, dura, pericranium) is laid on the cortex to induce ingrowth of a new vascular network. Protection is delayed by several weeks while the new vessels grow, but the extent is unpredictable. **YW p.1866 YW p.3365**

Procedures described:

1. **Encephaloduroarteriosynangiosis (EDAS)** — STA pedicle sutured to the pia/dural edges. **YW p.1866**
2. **Encephalomyosynangiosis (EMS)** — temporalis muscle implanted on the lateral brain surface and secured to the dural edges; widely used because a suitable cortical MCA branch is often absent in moyamoya, especially in children, and because gradual revascularisation avoids the hyperperfusion syndrome seen after direct bypass. **YW p.3365–3366**
3. **Encephaloduroarteriomyosynangiosis** and multiple **burr holes** without vessel synangiosis. **YW p.1866**
4. **Vascularised tissue grafts** — muscle, omentum, pericranium. **YW p.1866**
5. **Pial synangiosis** — a modification used in adults and children; in 143 patients strokes fell from 67% before treatment to 7.7% perioperatively and 3.2% after the first year, with a 4.3% long-term rate at 5 years. **YW p.1866**

Selection — indirect techniques are appealing in children because donor and recipient vessels are too small for direct anastomosis, and meta-analyses suggest indirect operations are more durable in the paediatric population. In adults, a direct technique alone or combined with an indirect procedure is the therapy of choice, because indirect methods alone have been reported to be unpredictable or ineffective. **YW p.1866 YW p.3366**

Peri-operative rules — avoid hyperventilation and crying, optimise hydration, keep normocapnia, and operate at a haemodynamically stable period; without these measures ischaemic complications occur in up to 10% or more. **YW p.1866 YW p.3366**

Youmans & Winn 8e — pp. 1597, 1598, 1863, 1864, 1866, 3323, 3362, 3363, 3365, 3366, 3695

Q K7

OCT 2025 · Paper III · Q8 · 3+2+2+3 marks

Moya moya disease: clinical presentation, pathophysiology, diagnostic work up and treatment options.

ANSWER

(a) Clinical presentation [3]

Definition — chronic progressive stenosis of the intracranial ICAs including proximal ACA and MCA with basal collaterals ("puff of smoke"). Bilateral stenosis is required for moyamoya **disease**; unilateral disease or a recognised associated condition makes it moyamoya **syndrome**. Bimodal onset — first decade and ages 30 to 40; female to male nearly 2:1; familial in 10% to 15.4%. **YW p.1861 YW p.3362**

Children — ischaemia (TIA or stroke) in 70% to 80%. In 143 patients: stroke 67.8%, TIA including drop attacks 43.4%, seizures 6.3%, headache 6.3%, choreiform movements 4.2%, haemorrhage 2.8%. TIAs are precipitated by hyperventilation with crying or exertion and by dehydration; headache is migraine-like and refractory. **YW p.1863**

Adults — intracranial bleeding is typical, especially in women (up to 66%), usually intraventricular or periventricular rather than subarachnoid, often repetitive with a 7% annual rebleed rate; only 45% recover well and 7% die, and after rebleeding only 20% recover with nearly 30% mortality. Ischaemia is next commonest and is dominant in Europe and the United States. A sudden fall in cognitive performance from low perfusion is the sole manifestation in about 10% of adults. Infarcts lie in cortico-subcortical watershed or PCA territories in 40%, sparing basal ganglia and thalamus. Saccular aneurysms in 4% to 14%; pregnancy and delivery raise stroke risk. **YW p.3363**

(b) Pathophysiology [2]

- Stenosis results from **intimal smooth muscle hyperplasia plus luminal thrombosis**; the media shows smooth muscle apoptosis with raised caspase-3 and the internal elastic lamina is disrupted. The MCA adjacent to the carotid fork is involved as well as the fork itself. **YW p.1863 YW p.3364**
- Basic FGF, TGF-beta, hepatocyte growth factor and VEGF are raised in the superficial temporal artery and diseased ICA wall, driving angiogenesis and intimal hyperplasia; soluble VCAM-1, ICAM-1, E-selectin and CSF nitric oxide metabolites indicate endothelial activation. Superficial temporal and renal arteries may be similarly affected, so the disease can be systemic. **YW p.3362**
- **Genetics** — autosomal dominant with incomplete penetrance, linked to 17q25.3; the **RNF213** p.R4810K founder mutation is strongly associated (up to 56% of Asian-American patients); loci MYMY1-3 on chromosomes 3, 6, 8 and 17, and ACTA2 R179, BRCC3/MTCP1, GUCY1A3 and SAMHD1 mutations, most affecting smooth muscle cell function. **YW p.1861 YW p.1862 YW p.3363**
- **Haemorrhage mechanisms** — rupture of dilated stressed perforators containing microaneurysms; fibrinoid necrosis of basal ganglia arterial walls; rupture of periventricular microaneurysms at the superolateral wall of the lateral ventricle. **YW p.3363**
- Associations: Asian ancestry, cranial radiotherapy, Down syndrome (26-fold), neurofibromatosis type 1, sickle cell anaemia. **YW p.1862**

(c) Diagnostic work-up [2]

- **CT** — haemorrhage, or hypodensities in cortical watershed zones, basal ganglia, deep white matter or periventricular regions. **YW p.1864**
- **MRI/MRA** — DWI for acute infarct; FLAIR for chronic burden and the **"ivy sign"** (linear sulcal high signal from slow cortical flow, about 80% of cases); reduced ICA, MCA and ACA flow voids with prominent basal ganglia and thalamic collateral flow voids are essentially diagnostic; T1 is sensitive for basal moyamoya. Microbleeds on T2 in 15% to 44% of adults predict later haemorrhage. Angiography is no longer required for definite diagnosis if MRI and MRA (>1 T) fulfil the criteria. **YW p.1864 YW p.3363**
- **Catheter angiography** — still the gold standard for staged surgical planning and progression; four-vessel plus ECA injections; transdural collaterals predict angiogenic potential and cut perioperative stroke by over

40% when used in planning. Complication rate about 1%. YW p.1864 YW p.1865

- **Haemodynamics** — TCD, perfusion CT, xenon CT, PET, MR perfusion, SPECT with acetazolamide challenge, arterial spin labelling. Reduced CBF and reduced CO₂/acetazolamide reactivity in the ICA territory, with raised cerebral blood volume and oxygen extraction fraction, are characteristic. YW p.1865 YW p.3365
- **Other** — EEG showing posterior slowing, hyperventilation "build-up" and the characteristic "re-build-up" (hyperventilation is not recommended in known moyamoya); RNF213/ACTA2 testing, inflammatory markers to exclude vasculitis, aspirin-resistance testing. YW p.1865

(d) Treatment options [3]

Medical — nothing addresses the aetiology. Antiplatelets are used routinely for microthrombi at the stenosis; warfarin rarely; calcium channel blockers for intractable headache and refractory TIAs, avoiding hypotension. Of 651 medically treated patients, 38% later needed surgery for progression. YW p.1865 YW p.3365

Surgical revascularisation is the primary treatment; indications are broad. It prevents stroke but does not arrest the arteriopathy. YW p.1865

- **Direct** — STA-MCA bypass: parietal or frontal STA branch (~1 mm) dissected over 8 to 10 cm, craniotomy 2.5 to 3 cm centred about 6 cm above the external acoustic meatus, anastomosis to a cortical MCA branch with 8 to 10 interrupted 10-0 or 11-0 sutures. STA-ACA (to the medial internal frontal artery) for ACA-territory hypoperfusion with cognitive decline; STA-PCA or OA-PCA for PCA territory. Immediate flow, but risk of hyperperfusion syndrome. YW p.3365
- **Indirect** — EDAS, encephalomyosynangiosis, multiple burr holes, pericranial or omental grafts, pial synangiosis. Delayed, unpredictable collateralisation; preferred in children whose vessels are too small for anastomosis. YW p.1866 YW p.3366
- In adults, direct bypass alone or combined with an indirect procedure is the therapy of choice. YW p.3366

Indications — repeated ischaemic symptoms or reduced regional CBF/perfusion reserve; symptomatic disease at any Suzuki stage; asymptomatic disease at Suzuki II to VI with progressive ischaemic change.

Contraindications: unclear diagnosis, asymptomatic hemisphere at Suzuki I-II with normal perfusion, profound compromise. Peri-operatively keep the patient stable, hydrated and normocapnic, and operate about 48 hours after acetazolamide testing; otherwise ischaemic complications reach 10% or more. Perioperative stroke risk is about 4% per hemisphere. YW p.1866 YW p.3366

Youmans & Winn 8e — pp. 1861, 1862, 1863, 1864, 1865, 1866, 3362, 3363, 3364, 3365, 3366

Carotico-Cavernous Fistula

2 questions

Q K8

APR 2023 · Paper II · Q3

Management of:

- a) Carotico Cavernous Fistula (CCF). [5]
- b) Orbital tumours. [5]

ANSWER

(a) Management of carotico-cavernous fistula (CCF) [5]

Classification

A CCF is either a pathologic shunt between the ICA and a vein of the cavernous sinus region (**direct/true CCF**) or multiple shunts between ICA and/or ECA branches and a vein in the same region (**indirect CCF** — a true dural arteriovenous fistula). Direct vs indirect is the most important distinction for diagnosis and treatment. YW

p.3733

- **Direct** — 80% to 90% follow direct or indirect trauma; the cavernous ICA ruptures easily because it is tethered by the dural rings. Also iatrogenic (transsphenoidal, endoscopic surgery) and spontaneous after rupture of an intracavernous aneurysm or in connective tissue disorders. YW p.3734
- **Indirect** — commoner in women in the sixth and seventh decades, probably following sinus thrombosis; the cavernous sinus is the second commonest dural AVF site after the transverse-sigmoid sinuses. YW p.3736 YW p.3737

Clinical assessment

Direct CCFs present dramatically with the triad of **exophthalmos, chemosis and visual loss** — orbital bruit 80%, proptosis 72%, chemosis 55%, sixth nerve palsy 49%, ophthalmoplegia 24%, visual loss 18%. Auscultate over the eyelid after major facial trauma. Retrograde cortical venous drainage causes haemorrhage and demands urgent treatment. YW p.3734 Dural CCFs are subtler: chemosis 94%, proptosis 87%, raised IOP 60%, cranial neuropathy 54%, reduced acuity 28%. YW p.3737

Diagnosis

CT and MRI give indirect signs (cavernous fullness, proptosis, enlarged venous channels), but **catheter angiography is the gold standard**, including both ICAs and ECAs and the posterior circulation. Localise a high-flow direct fistula with the **Mehring-Hieshima manoeuvre** (ICA injection with gentle common carotid compression) or **Huber's manoeuvre** (vertebral injection with carotid compression); selective ECA injection is essential for dural CCFs. YW p.3734 YW p.3737

Treatment

- **Observation** for indirect CCFs with minimal symptoms and no cortical venous drainage — spontaneous regression is not uncommon; historical intermittent carotid compression is of questionable value. YW p.3737
- **Indications to treat** — progressive or disabling symptoms, raised intraocular pressure refractory to medical treatment, cosmetic deformity, retrograde cortical venous drainage, frank haemorrhage. YW p.3737
- **Endovascular embolisation with coils and/or liquid agents is first line**; direct CCFs transarterially, indirect CCFs transvenously. YW p.3733
- **Direct** — obliterate any cortical venous limb with coils first to avoid redirecting flow into cortical veins; coils as a scaffold plus Onyx is the usual combination, with a non-detachable balloon to identify the fistulous point, act as an abutment and prevent reflux. Covered stents and flow diverters are alternatives but need dual antiplatelets, impractical after trauma. YW p.3736
- **Indirect** — transvenous coils, preferentially via the ipsilateral **inferior petrosal sinus** (catheterisable even if thrombosed); alternatives are the contralateral inferior petrosal sinus, clival or pterygoid plexus, superior or inferior ophthalmic vein (surgical cutdown if needed), cortical veins, or direct percutaneous puncture of the cavernous sinus or through the foramen ovale. YW p.3737 YW p.3738

(b) Management of orbital tumours [5]

Assessment

Presentation is by **visual loss, proptosis or diplopia**; the operative algorithm turns on whether the cause is mechanical/compressive or an optic nerve tumour, whether vision is usable, and whether the lesion is resectable without nerve damage — otherwise decompression with or without biopsy, or radiation. In an 891-case series the commonest lesions were metastasis (185), non-specific orbital inflammation (180), sphenoid-orbital meningioma (135), haemangioma (101), optic nerve or orbital meningioma (60), neurofibroma (39) and optic nerve glioma (39). YW p.1433

Choice of approach

Tumour location, not tumour type, determines the approach, and the rule is never to work across the plane

of the optic nerve. The orbit is read as a clock face about the optic nerve meridian. **YW p.1433 YW p.1442**

Position of tumour	Approach
Lateral (8-10 o'clock)	Lateral orbitotomy (lateral microsurgical approach)
Inferolateral (6-8 o'clock)	Lateral orbitotomy plus zygomatic osteotomy
Superolateral (9-1 o'clock) or intracranial extension	Pterional or fronto-orbital temporal craniotomy +/- orbitozygomatic osteotomy; supraorbital "eyebrow" or transorbital "eyelid crease" as less invasive options
Medial and anterior (1-5 o'clock)	Anterior medial micro-orbitotomy or transconjunctival; transcaruncular for the medial wall
Medial extending posteriorly (1-7 o'clock)	Endoscopic endonasal approach

YW p.1433 YW p.1438 YW p.1439 YW p.1442

Endoscopic endonasal approaches

Three variants — medial-inferior extraconal, transmaxillary extraconal and medial intraconal. The optic nerve is the key landmark: tumours displacing it superiorly and laterally are excellent targets, and medial intracranial extension can be handled endonasally provided the optic nerve and ophthalmic artery stay lateral. Landmarks are the lateral and medial opticocarotid recesses. Intraconal work needs a two-nostril approach with the medial and inferior rectus retracted on vessel loops to open a medial window; reconstruct with a mucosal graft or pedicled nasoseptal flap raised before the posterior septum is transected. **YW p.1439 YW p.1441**

Extent of resection and complications

Tumour type sets the aggressiveness: **optic nerve sheath meningiomas cannot be resected without devascularising the nerve and causing blindness**, whereas haemangiomas are encapsulated and can be removed completely through the right corridor. Complications — retro-orbital haematoma (control the ethmoidal arteries; a retracting ethmoidal artery causes sight-threatening retrobulbar haemorrhage), visual loss, thermal optic nerve injury when drilling (irrigate copiously), CSF leak and meningitis (seal any arachnoid-sinus communication with a vascularised flap), and perioperative diplopia from muscle oedema that usually resolves — exclude true entrapment with thin-slice CT and forced ductions. In 81 endonasal procedures, 2 patients had visual worsening from postoperative haematoma and 3.7% had a CSF leak, with the highest rates in intraconal tumours. **YW p.1441 YW p.1442**

High-yield: approaches are often combined to give 360-degree access to the intraconal and extraconal orbit. **YW p.1442**

Youmans & Winn 8e — pp. 1433, 1438, 1439, 1441, 1442, 3733, 3734, 3736, 3737, 3738

Q K9

MAY 2024 · Paper II · Q8

Cavernous sinus:

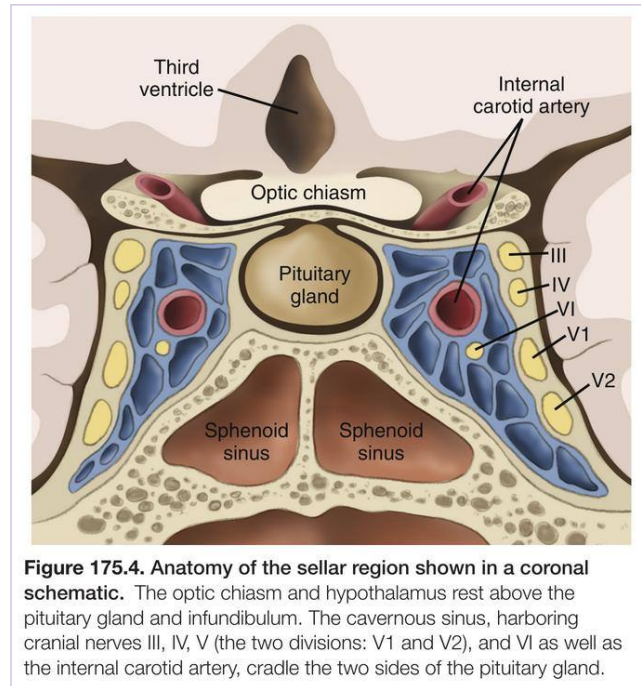
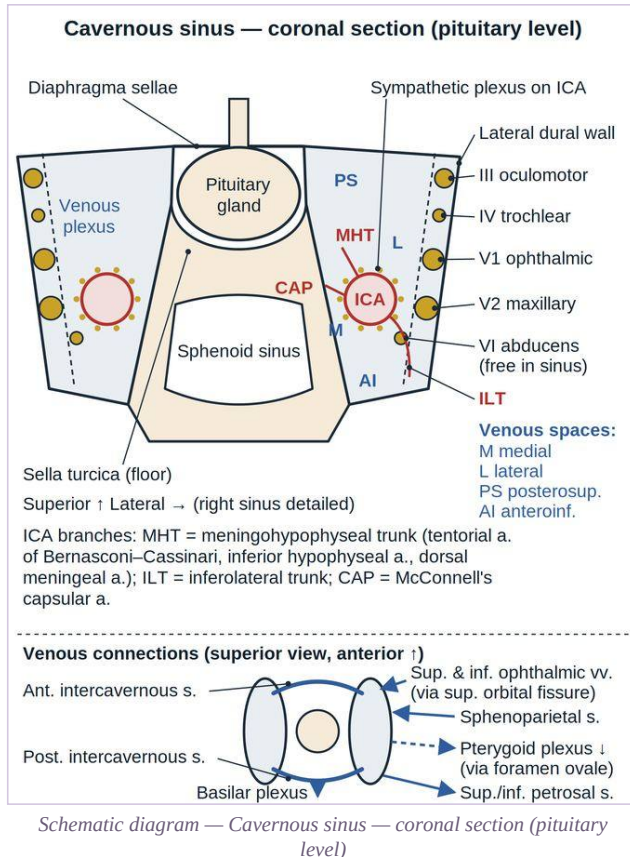
- Draw a section of cavernous sinus showing cranial nerve and vascular structures passing through it. [4]
- Classification and management of carotidocavernous fistula. [6]

ANSWER

(a) Section of the cavernous sinus showing cranial nerves and vascular structures [4]

The cavernous sinus is a dural (extradural) space either side of the pituitary fossa and the base of the greater

sphenoid wing. Because the veins within it are a **plexus** rather than a single venous lake, Parkinson proposed the name **lateral sellar compartment**; this plexiform arrangement explains the compartmentalisation seen in carotid-cavernous fistulas. It contains the ICA, cranial nerves III to VI, fat and a venous plexus. **YW p.70 YW p.3733**



Original figure: Youmans & Winn 8e, Fig. 175.4, p. 1294

Key relations to state under the diagram

- CN III enters between the posterior clinoid process and the tentorial edge through the **oculomotor triangle**, runs at the top of the sinus lateral to the ICA and just below the anterior clinoid process, and is crossed by CN IV 5 to 10 mm before the superior orbital fissure. **YW p.71**
- CN IV becomes superior and medial to CN III, relates to III medially (**supratrochlear triangle**) and to V1 inferiorly (**infratrochlear/Parkinson triangle**), and crosses CN III at the root of the anterior clinoid to reach the superior oblique. **YW p.71**
- CN VI enters through the **Dorello canal** below the petroclival (Gruber) ligament and runs medial and inferior to V1 toward the superior orbital fissure; approached transcranially, V1 obscures it. **YW p.71**
- The space between V1 and V2 is the **anteromedial (Mullan) triangle**. **YW p.71**
- The ICA branches are the meningohipophyseal trunk (present in 90% to 100%), the inferolateral trunk (supplying the nerves of the compartment) and McConnell's capsular artery (20% to 30%). **YW p.3733**
- Venous drainage: anteroinferior space receives the superior and inferior ophthalmic veins; the lateral space receives the sphenoparietal sinus and superficial middle cerebral vein; posteriorly the basilar plexus and superior and then inferior petrosal sinuses; inferolaterally the pterygoid plexus through the foramen ovale; both sides communicate via the intercavernous (circular) sinus. **YW p.3733 YW p.3735**

(b) Classification and management of carotidocavernous fistula [6]

Classification

Primary (Youmans) classification — direct vs indirect:

	Direct (true) CCF	Indirect CCF
Shunt	Cavernous ICA or its immediate branches to cavernous sinus veins	ICA and/or ECA branches to a vein of the cavernous region — a true dural AVF
Flow	High	Low
Cause	80-90% trauma; also iatrogenic, ruptured intracavernous aneurysm, connective tissue disease	Probably preceding sinus thrombosis
Typical patient	Any, after trauma	Women, sixth and seventh decades
Presentation	Classical triad of exophthalmos, chemosis, visual loss; bruit 80%, proptosis 72%, chemosis 55%, CN VI palsy 49%, ophthalmoplegia 24%, visual loss 18%	Chemosis 94%, proptosis 87%, raised IOP 60%, cranial neuropathy 54%, reduced acuity 28%; may regress spontaneously
Access	Transarterial	Transvenous

YW p.3733 YW p.3734 YW p.3736–3737

Barrow classification of carotid-cavernous dural AVFs (Barrow et al., J Neurosurg 1985) — listed in Youmans' table of neurosurgical classification systems: type A is the direct ICA-to-cavernous sinus shunt; types B, C and D are the indirect (dural) shunts, from meningeal branches of the ICA (B), of the ECA (C), or of both (D). YW p.135.e3 YW p.3733

Borden and Cognard classifications apply because indirect CCFs are dural AVFs, and both grade the risk of haemorrhage by the venous drainage pattern: YW p.3741

Borden	Venous drainage
I	Anterograde into the dural sinus and meningeal vein
II	Anterograde into the dural sinus plus retrograde into subarachnoid veins
III	Isolated retrograde drainage into subarachnoid veins (direct, trapped sinus segment, or venous varix/dural lake)

Cognard	Venous drainage
I	Anterograde into the main sinus
IIa	Main sinus with reflux into another sinus
IIb	Main sinus with reflux into cortical veins
IIa+b	Main sinus with reflux into both
III	Direct cortical venous drainage, no venous ectasia
IV	Direct cortical venous drainage with ectasia (>5 mm and 3x the draining vein)
V	Drainage into spinal perimedullary veins

YW p.3742 Any early venous reflux into cortical or spinal perimedullary veins is a **malignant** drainage pattern and is the major predictor of haemorrhage. YW p.3741–3742

Management

- **Diagnosis** — catheter angiography of both ICAs and ECAs plus the posterior circulation;

Mehring-Hieshima and Huber manoeuvres localise high-flow direct fistulas; selective ECA injection is essential in dural CCFs. **YW p.3734 YW p.3737**

- **Observation** — indirect CCFs with minimal symptoms and no cortical venous drainage; spontaneous regression is not uncommon. **YW p.3737**
- **Indications to treat** — progressive or disabling symptoms, refractory raised intraocular pressure, cosmetic deformity, retrograde cortical venous drainage, haemorrhage. Direct CCFs with cortical drainage and any CCF causing haemorrhage need urgent treatment. **YW p.3734 YW p.3737**
- **Endovascular therapy is first line.** Direct CCF: transarterial coils and/or Onyx, obliterating any cortical venous limb first; balloon-assisted technique, covered stents or flow diverters as adjuncts, remembering the dual antiplatelet requirement after trauma. Indirect CCF: transvenous coils +/- Onyx, preferentially via the ipsilateral inferior petrosal sinus, with alternative routes through the contralateral inferior petrosal sinus, clival or pterygoid plexus, superior or inferior ophthalmic vein (surgical cutdown if needed), cortical veins, or direct percutaneous puncture of the cavernous sinus or through the foramen ovale. **YW p.3733 YW p.3736–3738**
- **Outcome** — excellent, with a high likelihood of symptom resolution; complete obliteration with a single endovascular procedure in the majority. **YW p.3733 YW p.3738**

Youmans & Winn 8e — pp. 70, 71, 135.e3, 3733, 3734, 3735, 3736, 3737, 3738, 3741, 3742

Further Topics in this Chapter

9 questions

Q K10

DEC 2020 · Paper II · Q10 · 2+8 marks

Spinal AV fistula's: Classification and management.

ANSWER

(a) Classification [2]

The **Spetzler (modified) classification** asks one angiographic question — is there a nidus? If no, the lesion is a **fistula**; if yes, a **malformation**. Lesions are then named anatomically (extradural/intradural, ventral/dorsal, intramedullary), giving six types. **YW p.3780**

Spetzler type	Old name	Shunt
Extradural AVF	not separately recognised	Epidural radicular artery to epidural venous plexus YW p.3780
Intradural dorsal AVF	Type I, spinal dural AVF	Radicular artery to medullary vein in the dural root sleeve YW p.3781
Intradural ventral AVF	Type IV perimedullary (A/B/C)	Anterior spinal artery to venous network, midline YW p.3781
Extradural-intradural AVM	Type III juvenile/metameric (Cobb)	Whole somite — bone, muscle, skin, cord YW p.3782
Intramedullary AVM	Type II glomus (compact/diffuse)	Nidus in cord, ASA/PSA feeders YW p.3782
Conus medullaris AVM	new category	Multiple ASA + PSA feeders, venous ectasia YW p.3783

Anson-Spetzler subtypes of type IV: **IV-A** single feeder, **IV-B** several dilated feeders, **IV-C** giant fistula with multiple giant feeders. Borden: I antegrade drainage into Batson plexus, II retrograde perimedullary drainage, III meningeal feeder into coronal venous plexus. **YW p.3786**

(b) Management [8]

General principles

- Obliterate the **fistulous point** (AVF) or **nidus** (AVM). General anaesthesia, controlled respiration, heparin to ACT 1.5–2x, superselective catheterisation beyond the branch supplying normal cord. **YW p.3787**
- SSEP/MEP with provocative testing — amobarbital 25–50 mg then lidocaine 20–40 mg; abort that position if SSEP amplitude falls 50%, latency rises >10%, or MEPs disappear. **YW p.3787**
- NBCA is most durable; PVA recanalises (~20% vs ~5% for liquids); coils for large fistulas or as a frame; Onyx off-label, not proven superior. **YW p.3787–3788**
- Postoperatively: NSICU, steroids for cord AVMs, heparin after large-shunt AVFs; angiography at 3 months, 1 year, 3 years. **YW p.3787**

Extradural AVF

Treated almost exclusively endovascularly. Transarterial (occasionally transvenous) closure of a short segment of feeder, the fistula and the proximal draining vein; fibred detachable coils preferred to limit distal migration. Review of 123 cases — obliteration 85% surgical, 84% endovascular, 70% improve. **YW p.3790**

Intradural dorsal AVF (commonest)

Progressive congestive myelopathy with back/leg pain and sphincter dysfunction; 10–20% have acute non-haemorrhagic exacerbations. The fistula must be eliminated to arrest progression. **YW p.3791**

- **Surgery:** unilateral hemilaminotomy, trace the arterialised vein to the dural sleeve, coagulate and sharply divide it, confirm with ICG — low morbidity, high cure. **YW p.3781**
- **Embolisation:** wire-assisted (not flow-directed) microcatheter because flow is slow; drive NBCA into the **proximal draining vein** — under-penetration recurs by collateralisation, over-penetration worsens venous hypertension. A feeder sharing a pedicle with the cord artery must be operated, not embolised. Control runs on the contralateral pedicle and two levels above and below. **YW p.3792**
- Current trend is endovascular first, surgery for failure or recurrence, though surgery has the higher cure rate. **YW p.3791**

Intradural ventral AVF

ASA patency is paramount. **IV-A** usually surgical (ASA barely dilated); **IV-B** NBCA from a wedged microcatheter closing the whole fistula; **IV-C** transarterial or transvenous coil packing of the venous pouch then NBCA. Pooled 213 patients — surgery 88% obliteration (68% improved, 6% worse), endovascular 74% (75% improved, 11% worse). **YW p.3792**

AVMs

Intramedullary AVMs — **microsurgical pial resection** (up to 75% complete), embolisation as adjunct; endovascular cure alone 33% with 13% worsening, but partial embolisation still lowers bleeding rate. **YW p.3781, p.3789** Extradural-intradural (Cobb) lesions are treated **palliatively** by staged embolisation and surgery. **YW p.3782, p.3788** Conus AVMs — aggressive embolisation then microsurgery; 43% improved, 43% stable at 70 months. **YW p.3789**

High-yield: conservative management has no role in spinal dural AVF; embolisation is the fastest option in acute malignant venous congestion (Foix-Alajouanine syndrome). **YW p.3821**

Youmans & Winn 8e — pp. 3780, 3781, 3782, 3783, 3786, 3787, 3788, 3789, 3790, 3791, 3792, 3821

Q K11

DEC 2020 · Paper III · Q7 · 5+5 marks

- a) Pathophysiology and clinical presentation of carotid artery dissection.
- b) Evaluation and management of vertebrobasilar artery dissection.

ANSWER

(a) Pathophysiology and clinical presentation of carotid artery dissection [5]**Epidemiology**

Annual incidence 2.5–3 per 100,000; ~2% of all ischaemic strokes but ~20% of strokes in young adults; mean age ~50 years. YW p.3323, p.3324

Pathophysiology

- A tear in the **tunica intima** lets blood track into the media forming a **false lumen**, which either narrows the true lumen or expands outward as a **dissecting aneurysm** (retains adventitia). A **pseudoaneurysm** has no arterial wall layers — its wall is only the haematoma capsule. YW p.3323
- "Spontaneous" means no direct trauma; dissection after coughing, exercise or routine head movement still counts as spontaneous. Associations — fibromuscular dysplasia, cystic medial necrosis, Marfan syndrome; most are idiopathic. YW p.3323
- Contributors: low type III : type I collagen ratio, mild hyperhomocysteinaemia with low folate, MTHFR TT genotype, raised matrix metalloproteinase-2, impaired endothelium-independent vasodilatation. YW p.3324

Clinical presentation

1. **Triad** — ipsilateral face/head/neck pain, partial Horner syndrome, TIA or stroke. All three in <1/3; **any two should make the diagnosis**. YW p.3323
2. Headache commonest (83%) — frontal, periorbital or periauricular; neck pain infrequent (21%). YW p.3323
3. Focal ischaemia (TIA, stroke or both) 60%. YW p.3323
4. Partial Horner (miosis + ptosis **without** anhidrosis) 50% — oculosympathetic fibres run with the ICA, sudomotor fibres with the ECA. YW p.3323
5. Cervical bruit in >40%. YW p.3323

Angiography

String sign — long, tapered, irregular narrowing beginning 2–3 cm above the bulb, reconstituting at the petrous segment. Intimal flap or double lumen <10%; dissecting aneurysm in ~1/3; occlusion recanalises long-term in 43–47%. YW p.3324

(b) Evaluation and management of vertebrobasilar artery dissection [5]**Evaluation**

- **Clinical:** occipital headache/neck pain with posterior circulation ischaemia; lateral medullary (Wallenberg) syndrome in 13%; bilateral in 22%. **Intracranial** vertebrobasilar dissection causes brainstem ischaemia, **SAH in half of patients**, or both. Fibromuscular dysplasia is the commonest arteriopathy, affecting middle and distal segments; other risks — spinal manipulation, nose blowing, ceiling painting, oral contraceptives, hypertension, migraine, female sex, recent infection. Blunt trauma thromboses the artery most often at C2. YW p.3335
- **Imaging:** MRI/MRA or CT/CTA of head and neck with DWI; CT shows transverse foramen fracture and facet dislocation. YW p.3335, p.3336
- **Catheter angiography** is the gold standard (1% neurological deficit, 0.5% persistent) — arch plus four vessels and collaterals; **dynamic angiography** when symptoms are provoked by neck rotation. YW p.3337
- **QMRA (NOVA)** separates hypoperfusion from embolism; VERITAS showed 70% vs 87% stroke-free

survival at 24 months with low vs normal distal flow. **YW p.3337**

- Exclude mimics — metabolic/drug levels, ECG, Holter, echocardiography, audiometric and vestibular testing. **YW p.3336**

Management

Step	Detail
Supportive	Monitored bed, IV fluids, avoid hyperglycaemia; hypertensive/hypervolaemic therapy if haemodynamically significant YW p.3337
Anticoagulation	Heparin then oral anticoagulation, INR 2–3 for 3–6 months ; re-image at 3 months and continue if the lesion persists, then long-term antiplatelet YW p.3337, p.3338
Antiplatelet	Equivalent alternative — meta-analysis of 26 studies (327 patients) showed no difference in death or disability YW p.3338
Contraindications to anticoagulation	Intracranial dissection with SAH; large infarct with mass effect; intracranial extension YW p.3338
Thrombolysis	Works within hours on clot in both lumens; avoid if SAH from intracranial dissection YW p.3338
Endovascular	For symptoms refractory to (or contraindications to) medical therapy and expanding dissecting aneurysms — angioplasty with balloon-expandable/self-expanding stents, coils or covered stent YW p.3338
Do not treat	Asymptomatic residual aneurysm or luminal irregularity — extracranial dissecting aneurysms do not rupture YW p.3338

Severe stenosis or intraluminal thrombus favours anticoagulation; a large established infarct favours antiplatelet therapy. **YW p.3338**

Youmans & Winn 8e — pp. 3323, 3324, 3335, 3336, 3337, 3338

Q K12

JUN 2021 · Paper II · Q1

Transverse sinus thrombosis:

- a) Aetio-pathogenesis. [3]
- b) Diagnosis. [3]
- c) Management. [4]

ANSWER

(a) Aetio-pathogenesis [3]

Mechanism. Sinus thrombosis raises venous pressure, causing cerebral hypoxia and neuronal ischaemia, blood-brain barrier breakdown, raised intracranial blood volume and intracranial hypertension — a spectrum from oedema to venous infarction and massive haemorrhage. Mechanisms act through the sinus wall, blood chemistry or flow. **YW p.3369**

Causes.

- **Prothrombotic states** in 20–35% (acquired in 34% of the largest series) — protein C/S, antithrombin III and plasminogen defects, factor V Leiden, prothrombin G20210A, lupus anticoagulant, anticardiolipin antibodies, hyperhomocysteinaemia. **YW p.3369, p.3370**

- **Infection** 8.2% — **transverse and cavernous sinus thromboses are the ones most often infective**, from sinusitis, otitis and mastoiditis; *Staphylococcus aureus* commonest, gram-negative rods and *Aspergillus* in chronic forms. YW p.3369
- Pregnancy and puerperium, oral contraceptives (8%, higher with the prothrombin mutation), high altitude, drugs (androgens, danazol, lithium, vitamin A, IVIg, MDMA), malignancy, nephrotic syndrome, polycythaemia, iron-deficiency anaemia, SLE, Behçet disease, inflammatory bowel disease. YW p.3369, p.3370
- Mechanical — trauma, lumbar puncture, epidural blood patch, intracranial hypotension. **About 40% are idiopathic.** YW p.3369, p.3370

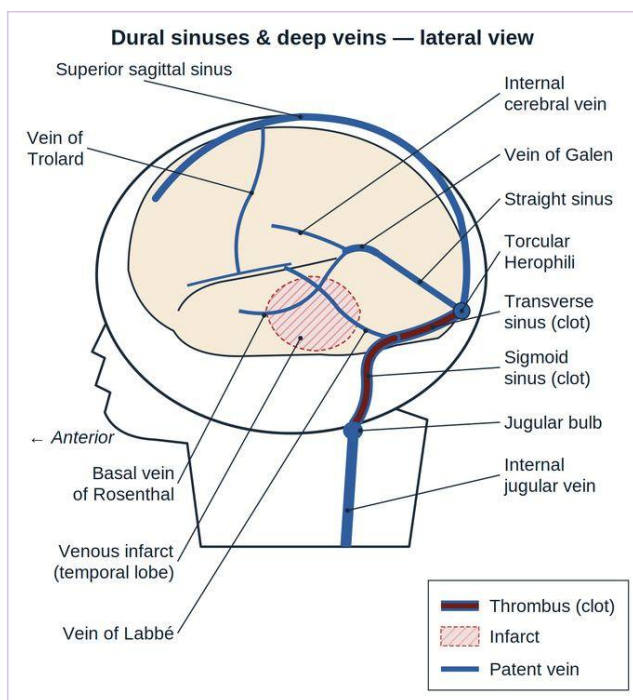
Sites: superior sagittal 62%, **transverse 41–45%**, straight 18%, cavernous 1.3%. Incidence ~5 per million per year, 0.5–1% of strokes, preponderance in women aged 20–40. YW p.3370

(b) Diagnosis [3]

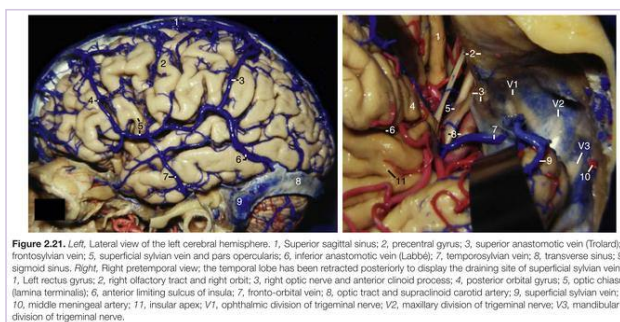
Clinical. Headache earliest and commonest (~80%); papilloedema ~50%, mental status change ~25%, focal deficits 50–75%, seizures ~33%; subacute onset in 70%. **Thrombosis confined to the transverse or sagittal sinus usually presents as isolated intracranial hypertension and is mistaken for IIH.** Transverse sinus CVT may add otalgia, otorrhoea, cervical tenderness and lymphadenopathy from mastoiditis or otitis media; cortical venous extension adds focal deficits and seizures. YW p.3370

Investigation.

1. Bloods with coagulation profile and a **prothrombotic screen.** YW p.3370
2. **CT venography** usually first — dense cord sign ~20% (false-positive in neonates, dehydration, polycythaemia), **empty delta sign** 10–30%; CT false-negative rate 4–25%. YW p.3371
3. **MRI with MRV is the modality of choice and most sensitive;** T2*/SWI show haemorrhage. False positives with a hypoplastic or absent sinus — **coronal source MRV** proves a transverse sinus is occluded rather than hypoplastic. YW p.3371, p.3373
4. Temporal lobe vasogenic oedema with haemorrhage suggests **vein of Labbé** infarction and transverse sinus thrombosis. YW p.3373
5. **DSA** only when MRI is inconclusive or intervention is planned — non-opacification of the sinus in the venous phase, abruptly terminating cortical vein with dilated corkscrew collaterals. YW p.3371



Schematic diagram — Dural sinuses & deep veins — lateral view



Original figure: Youmans & Winn 8e, Fig. 2.21, p. 23

(c) Management [4]

- 1. Treat cause and consequences.** Antibiotics and drainage of purulent collections. Raised ICP — hyperventilation, mannitol or hypertonic saline, CSF drainage, craniectomy; maintain CPP; anticonvulsants for seizures. **YW p.3372**
- 2. Anticoagulation (mainstay).** Heparin to aPTT 2–2.5x normal; ICH or SAH is not an absolute contraindication. Then warfarin or an oral factor Xa inhibitor, **INR 2–3 for 6 months**, lifelong in hypercoagulable states. Meta-analysis of four trials — LMWH or unfractionated heparin gave fewer deaths and less disability than placebo, with no difference between them. **YW p.3372, p.3374**
- 3. Endovascular therapy** for failure of anticoagulation — justified by poor GCS, no response to heparin, rapid worsening, altered mental status, deep (straight sinus) thrombosis or large space-occupying stroke. Transfemoral, transjugular or direct sinus puncture for local thrombolysis or mechanical thrombectomy, with optional venous stenting. **YW p.3375, p.3378**
- 4. Surgery.** Ventriculostomy for CSF diversion and ICP monitoring; **decompressive hemicraniectomy** for refractory intracranial hypertension and impending herniation; open venotomy as last resort. **YW p.3378**

Modality	Evidence
Systemic anticoagulation	Class IIa, level B YW p.3378
Local fibrinolysis	Class IIb, level B YW p.3378
Mechanical thrombectomy	Class IIb, level B YW p.3378
Surgical decompression for refractory raised ICP	Class IIb, level C YW p.3378

Algorithm: MRI T2* + MRV (or CT/CTV) → anticoagulate → improving: oral anticoagulation 3–12 months or lifelong by aetiology; deteriorating with severe mass effect or ICH: decompressive hemicraniectomy, otherwise endovascular therapy. **YW p.3378**

Youmans & Winn 8e — pp. 3369, 3370, 3371, 3372, 3373, 3374, 3375, 3378

Q K13

DEC 2021 · Paper II · Q9

Vein of Galen malformation:

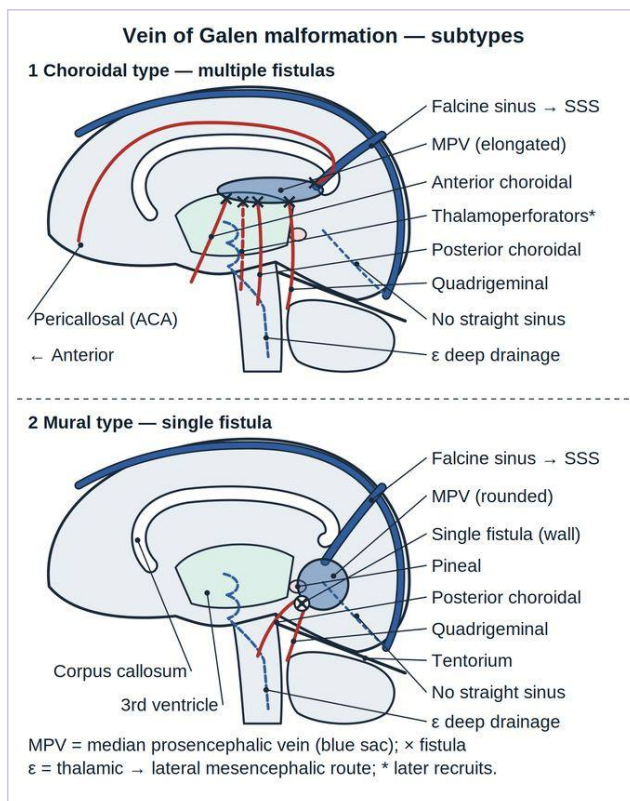
- a) Clinical features. [3]
- b) Radiological features. [3]
- c) Management. [4]

ANSWER

(a) Clinical features [3]

A vein of Galen aneurysmal malformation (VGAM) is a high-flow fistulous malformation draining into the persistent **median prosencephalic vein of Markowski**, not the true vein of Galen. It is <1% of all AVMs and usually presents before 5 years. Two forms — **choroidal** (multiple fistulas, very high flow) and **mural** (one or few fistulas at the vein wall). YW p.1869

- **Neonate (choroidal):** significant **high-output cardiac failure**, worsening over the **first 3–5 days of life** — volume overload of all four chambers, tachycardia, persistent pulmonary hypertension with right-to-left shunting and cyanosis, and **diastolic flow reversal in the descending aorta**. Hepatomegaly, prerenal azotaemia, oliguria and metabolic acidosis are negative prognostic markers. YW p.1875
- **Infant/child (mural, hydrodynamic disorder):** **macrocrania** first, then ventriculomegaly, raised ICP, developmental delay and mental retardation; mural lesions present later with macrocephaly, hydrocephalus or failure to thrive and only mild cardiac failure. Engorged facial veins, exophthalmos and epistaxis from ophthalmic vein drainage; thickened diploe; **tonsillar prolapse** from cerebellar venous congestion — an indication for embolisation, **not** shunting. YW p.1869, p.1878
- **Older child:** cortical venous reflux causes seizures, haemorrhage and focal deficits; chronic venous ischaemia causes bilateral subcortical calcification and atrophy. **Melting brain syndrome** — progressive white-matter destruction with ventriculomegaly but normal ICP — carries a poor prognosis. YW p.1878



Schematic diagram — Vein of Galen malformation — subtypes

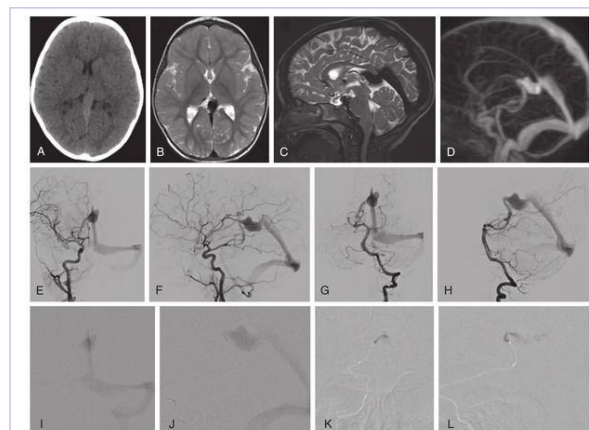


Figure 247.7. A vein of Galen malformation in a 2-year old female patient was incidentally detected during workup for a head injury on CT scan (A) and subsequent MRI brain scans (B, C, D). At 32 months, elective cerebral angiogram and transarterial embolization was performed with complete obliteration. A right internal carotid artery angiogram (E–F) and left vertebral artery (G–H) angiogram show a mural type of vein of Galen aneurysmal malformation. Superselective angiograms of the pedicles performed using a detachable Apollo microcatheter (I–J) and subsequent glue injection at the fistulous location (K–L) are shown. Continued in Fig. 247.8.

Original figure: Youmans & Winn 8e, Fig. 247.7, p. 1876

(b) Radiological features [3]

- **Antenatal ultrasound** may show the midline venous pouch; cardiac enlargement in utero predicted a low neonatal score and death in all four such cases. Fetal and neonatal MRI guides risk stratification and timing. **YW p.1875**
- **Brain parenchyma** (transfontanellar ultrasound, CT, MRI) — venous congestion, encephalomalacia, atrophy, ventricular size, and **bilateral symmetrical subcortical calcification in the transcerebral venous watershed**. **YW p.1878, p.1879**
- **Location and feeders** — subarachnoid, in the **velum interpositum and quadrigeminal cisterns**; anterior and posterior choroidal arteries, anterior cerebral artery via the pericallosal arcade, and quadrigeminal arteries. **YW p.1872**
- **Venous outflow** — the **straight sinus is often absent**, drainage passing through an embryonic **falcine sinus** into the posterior third of the superior sagittal sinus, with persistent occipital and marginal sinuses. Deep drainage takes a thalamic to lateral mesencephalic route with an **epsilon shape on the lateral angiogram**. Progressive jugular bulb, sigmoid or transverse sinus stenosis is typical and drives the hydrodynamic disorder. **YW p.1874, p.1878**
- **Differentiate** from vein of Galen aneurysmal **dilation** (secondary dilation of the true mature vein by a pial AVM or dural AVF, which also drains normal brain) and from a varix with no shunt. **YW p.1869**
- **Diagnostic angiography alone has no place** in a neonate or infant; it is done only when embolisation follows in the same sitting. **YW p.1879**

(c) Management [4]

1. **Medical treatment of neonatal failure.** Diuretics and fluid restriction, ventilation if severe, inotropes (dobutamine, dopamine), phosphodiesterase inhibitors, and nitric oxide with milrinone for pulmonary hypertension; digitalis remains controversial. Goal — control failure so the infant feeds and gains weight. **YW p.1876**
 2. **Timing and selection.** Both umbilical arteries and the vein are catheterised at birth, permitting **transumbilical embolisation without damaging small femoral arteries**. **Significant diastolic flow reversal in the descending aorta on echocardiography is the single most important indicator for early intervention**. **Lasjaunias 21-point neonatal score** (cardiac, cerebral, respiratory, hepatic, renal): **<8 no treatment; 8–12 emergency embolisation; >12 medical management until ~5 months**. Antenatal diagnosis alone is not an indication for emergency neonatal embolisation. **YW p.1876, p.1879**
- Indications for early intervention:** (1) unstable or progressive cardiac failure despite maximal medical therapy; (2) significant macrocrania or hydrocephalus; (3) developmental delay or venous ischaemic change such as calcification; (4) pial venous hypertension. If imaging shows established brain damage, treatment is **not** indicated. **YW p.1879**
3. **Endovascular treatment — the treatment of choice.** Transfemoral **transarterial NBCA embolisation**; medical, surgical and radiosurgical treatment are adjuncts only. In the neonate the aim is not cure but control of failure — about **50% flow reduction** usually improves cardiac status. **Mural** lesions are cured in one or two sessions; **choroidal** lesions need staged embolisation at 3–6 month intervals over years, and complete obliteration is not required to restore hydrovenous equilibrium. **The dilated vein of Galen must not be occluded at the outset**, since deep venous drainage may pass through it. **YW p.1874, p.1876, p.1879**
 4. **Hydrocephalus.** Timely embolisation prevents it. **Avoid ventricular shunting** — it reverses the ventricle-to-sinus gradient, causing brain oedema, enlargement of the draining vein, white-matter calcification, subdural haematoma and slit ventricles. **Third ventriculostomy with embolisation is preferred**; embolisation is deferred several days after shunting to avoid upward cerebellar herniation. Follow-up — monthly head circumference, MRI 3-monthly. **YW p.1878, p.1879**

CHAPTER 16

Functional Neurosurgery, Epilepsy, Pain & Movement Disorders

30

QUESTIONS SOLVED

Epilepsy Surgery & Drug-Resistant Epilepsy

11 questions

Q P1

DEC 2020 · Paper III · Q3 · 4+6 marks

Pre-surgical evaluation & surgical approaches in Pediatric epilepsy.

ANSWER

(a) Pre-surgical evaluation in paediatric epilepsy [4]

When to evaluate

Evaluation begins once pharmacoresistant epilepsy is established — failure to respond to two or more appropriately chosen and used antiepileptic medications. The goal is to localise the epileptogenic zone (EZ) and define its anatomical overlap with eloquent cortex **YW p.503**. Class I evidence (Wiebe, Engel and, in children, the Dwivedi randomised trial) shows surgery is superior to medical therapy despite less well-defined networks and more extratemporal/multifocal disease in children **YW p.2042**.

Mandatory (phase 1) work-up

Modality	What it defines	Paediatric notes
Clinical history + semiology	Symptomatogenic zone, risk factors	Gestational/developmental history, age at onset, genetic testing
Video-EEG (≥ 24 h, ictal + interictal)	Electroclinical hypothesis	Mainstay; both interictal and ictal analysis
3D volumetric epilepsy-protocol MRI (1.5/3 T)	Epileptogenic lesion	Detects malformations of cortical development
Neuropsychology (age-appropriate)	Functional deficit zone, baseline	Essential baseline before resection

These four are the mainstays of diagnostic testing and surgical candidacy **YW p.2041**. The recommended MRI protocol (HARNES) comprises volumetric T1, 3D FLAIR and oblique coronal T2 perpendicular to the hippocampal long axis **YW p.503**.

Secondary (optional/highly recommended) tests

- FDG-PET, ictal SPECT/SISCOM, MEG and fMRI — noninvasive means of generating localisation hypotheses and detecting radiographically occult lesions; chosen by diagnostic considerations, availability and age-appropriate tolerance **YW p.2042**.
- Wada/sodium amytal testing for memory is typically **not** tolerated in younger children or those with autism spectrum disorder or developmental delay; if seizures localise to mesial temporal lobe in a child, the risk to memory is considered outweighed by the risk of intractable seizures **YW p.2042**.
- Results are integrated at a multidisciplinary case conference (epileptologist, epilepsy neurosurgeon, neuropsychologist) with the family counselled that the risks of longstanding epilepsy dwarf the risks of

surgery **YW p.2042.**

Invasive (phase 2)

Indicated when MRI is negative or discordant, when there are multiple potentially epileptogenic lesions, or when the hypothesis involves eloquent cortex **YW p.505**. Robot-assisted SEEG is now favoured for its safety and minimal invasiveness and can determine laterality, regionalise and localise the EZ and expose epileptogenic networks; symmetrical bilateral implantation is used when data are discordant or when neuromodulation is anticipated. Subdural grid/strip plus depth electrodes are used when laterality is known and comprehensive extraoperative mapping is needed, since children tolerate awake craniotomy poorly **YW p.2042–2043**.

(b) Surgical approaches [6]

1. Focal resection / lesionectomy

Commonest surgically remediable paediatric epilepsies are structural. With concordance between electroclinical data and structural pathology, cure rates approach 75% at short-term follow-up. Well-localised type II cortical dysplasias are treated by single-stage craniotomy and excision of the EZ when separate from eloquent cortex; seizure control correlates with extent of resection of the radiographically apparent dysplasia. Type I dysplasias are diffuse, multilobar and MR-negative and do worse **YW p.2043**.

2. Ablation (LITT)

Hypothalamic hamartoma with gelastic seizures is the archetype, effectively treated by stereotactic laser interstitial thermal therapy; LITT is also used for periventricular nodular heterotopia and, with mixed results, for tubers **YW p.2043–2044**.

3. Disconnection — hemispherotomy

Indicated for holohemispheric dysfunction: Rasmussen encephalitis, extensive perinatal/childhood stroke, hemimegalencephaly and Sturge-Weber syndrome. Disconnection is achieved by intraventricular disruption of temporal, parietal and frontal descending outputs, frontal disconnection, complete callosotomy and insular topectomy **YW p.2044**. Hemispherotomy has largely replaced anatomical and functional hemispherectomy, with shorter operating time and much less blood loss **YW p.636**. Small infants with catastrophic epilepsy may be operated very early (from about 4 months) **YW p.637**. In Rasmussen encephalitis a long-term study showed only a palliative effect, with 22% seizure free at 10 years and considerable speech and motor morbidity **YW p.2044**.

4. Callosotomy

Used primarily for atonic (drop) seizures; complete extraventricular section through a small vertex craniotomy, maintaining the plane between the leaves of the septum pellucidum **YW p.2044**.

5. Neuromodulation

VNS and responsive neurostimulation are used where the focus is multifocal, bilateral or in eloquent cortex, and can be combined with resection — e.g. RNS in dominant motor cortex in tuberous sclerosis, and immunotherapy plus VNS/RNS in Rasmussen encephalitis in lieu of hemispherotomy **YW p.2043–2044**.

Outcome

For malformations of cortical development overall, surgery cures just over 50%, with about 70% achieving Engel class I-II; early resection gives better long-term results than deferred surgery **YW p.488.e7**.

High-yield: in children the operation is chosen to fit the network — focus, lobe, hemisphere or circuit — and no single technique is exclusive **YW p.2042**.

Youmans & Winn 8e — pp. 503, 505, 636, 637, 2041, 2042, 2043, 2044, 488.e7

Q P2

DEC 2021 · Paper III · Q9

Drug resistant epilepsy:**a) Preoperative evaluation. [5]****b) Surgical techniques for managing drug resistant epilepsy. [5]****ANSWER****(a) Pre-operative evaluation of drug-resistant epilepsy [5]****Definition first**

Item	Youmans statement
Drug-resistant (pharmacoresistant) epilepsy	Failure of two adequately selected and dosed antiepileptic drugs
Basis	Kwan-Brodie cohort: 47% seizure free on AED 1, 13% on AED 2, 3.7% on AED 3, <=1% thereafter
Implication	Consider surgical therapy once this point is reached

YW p.497. Such patients have less than a 5% chance of successful medical treatment **YW p.667.**

Goal

Localise the epileptogenic zone and define its overlap with eloquent cortex, so that a testable anatomo-electro-clinical (AEC) hypothesis can be framed **YW p.503.**

Non-invasive modalities

Modality	Zone / information defined
Seizure semiology, aura	Symptomatogenic zone — first non-silent area affected, not necessarily onset
Scalp video-EEG in an EMU	Irritative and ictal-onset zones; confirms the diagnosis
High-resolution MRI (HARNESS protocol)	Epileptogenic lesion
FDG-PET	Interictal hypometabolism; useful in MRI-negative temporal cases
Ictal SPECT/SISCOM	Ictal hyperperfusion; best in MRI-negative and FCD
MEG / magnetic source imaging	Interictal source localisation; also eloquent cortex
fMRI (+/- EEG-fMRI)	Language/memory lateralisation; replacing Wada in many centres
Neuropsychology	Functional deficit zone
MRI post-processing (VBM, machine learning)	Converts a "non-lesional" MRI into a lesional one

Video-EEG and high-resolution MRI are the two highest-yield techniques and, with a concordant history, suffice in the majority of cases **YW p.503–504.** Data are integrated at a patient management conference with epileptologists, neurosurgeons, neuroradiologists and neuropsychologists to reach consensus **YW p.504.**

Invasive evaluation

Indicated when (1) MRI is negative or discordant, (2) there are multiple lesions or divergent data, or (3) the hypothesis involves eloquent cortex **YW p.505.** Subdural grids give contiguous cortical coverage and are best for functional mapping adjacent to a surface lesion; SEEG samples deep structures (mesial temporal, insula, cingulate, orbitofrontal, sulcal depths), allows bihemispheric exploration and permits radiofrequency

thermocoagulation, and is now the preferred method in most centres **YW p.505–506**. Wada testing remains relevant where fMRI is inconclusive, and predicts post-operative amnesia when memory asymmetry is reversed **YW p.539**.

(b) Surgical techniques for drug-resistant epilepsy [5]

Category	Procedure	Typical indication	Seizure outcome
Resective	Anteromedial temporal lobectomy	Mesial temporal lobe epilepsy	Engel I in 66% at 10 years
Resective	Selective amygdalohippocampectomy	Mesial temporal epilepsy, dominant side	60-90% seizure free at 3-6 years, equal to ATL
Resective	Frontal resection/lobectomy	Frontal lobe epilepsy	Engel I ~50% (meta-analysis 45.1%)
Resective	Parietal/occipital resection	Posterior quadrant epilepsy	Engel I-II 53-82%
Ablative	MRg laser interstitial thermal therapy	MTLE, FCD, hypothalamic hamartoma, insula	Lower seizure freedom than open surgery but less morbidity
Ablative	Gamma Knife radiosurgery	Hypothalamic hamartoma, MTLE	Engel I 39.6%, Engel I-II 68.8% in HH
Disconnective	Hemispherotomy	Hemispheric damage with hemiparesis	75-90% seizure free in modern series
Palliative disconnective	Corpus callosotomy	Atonic/drop attacks, multifocal epilepsy	80-100% reduction in drop attacks
Palliative disconnective	Multiple subpial transection	Focus in eloquent cortex	Disrupts horizontal spread, preserves columns
Neuromodulation	VNS / ANT-DBS / RNS	Non-resectable or multifocal epilepsy	Palliative, ~50% median reduction at 2 years

Sources: ATL outcome **YW p.601**; SAH equivalence **YW p.603**; frontal series **YW p.607**; parietal series **YW p.611**; LITT trade-off **YW p.631**; radiosurgery for hamartoma **YW p.622**; hemispherotomy **YW p.644**; callosotomy and MST **YW p.615.e3–615.e4**; neuromodulation **YW p.659**.

Choosing between them

Chances of seizure freedom rise when the epileptogenic zone is well visualised and restricted both electrically and semiologically, and is completely resected; palliative and less invasive options are increasingly needed as the surgical population shifts towards MRI-negative and multifocal epilepsy **YW p.667.e1**.

High-yield: two randomised controlled trials establish the superiority of resective surgery over medical therapy in drug-resistant temporal lobe epilepsy — only two patients need be operated for one to become free of disabling seizures **YW p.667.e3**.

Youmans & Winn 8e — pp. 497, 503, 504, 505, 506, 539, 601, 603, 607, 611, 615.e3, 615.e4, 622, 631, 644, 659, 667, 667.e1, 667.e3

Q P3

JUN 2022 · Paper II · Q7

- a) Discuss focal cortical dysplasia classifications. [5]
 b) Discuss diagnostic workup and management of FCDs. [5]

ANSWER

(a) Classification of focal cortical dysplasia [5]

FCDs are regions of cerebral cortex in which tissue architecture and lamination are abnormal. They are a principal cause of epilepsy in infancy and childhood, are all congenital and all highly epileptogenic, with seizures as the presenting symptom YW p.488.e6.

ILAE (Blumcke) classification

ILAE type	Histopathology	Key clinical/imaging features
FCD type I	Abnormal cortical dyslamination — microcolumnar or radial; neurons morphologically normal	Often MRI-negative ("non-lesional"); blurring of grey-white junction from heterotopic neurons in the U-fibre layer; typically posterior temporal or parieto-occipital; onset in early infancy
FCD type II	Abnormal lamination (not usually microcolumnar) plus dysmorphic, megalocytic neurons	Distinctive focal MRI change, often transmantle sign — grey matter line from ventricular wall to the depth of a sulcus
FCD IIa	Dysmorphic neurons, no balloon cells	
FCD IIb	Dysmorphic neurons plus balloon cells	
FCD type III	Type I or II associated with a principal lesion	IIIa hippocampal sclerosis; IIb cortical tumour; IIIc vascular malformation; IIId infarct, porencephalic cyst or other lesion

YW p.488.e6. The same three-tier scheme (type I dyslamination; type II dysmorphic neurons +/- balloon cells; type III with a second lesion) is used to predict differing clinical presentation, topography and surgical response YW p.576.

Genetics

FCD type II is a post-zygotic somatic mutation of mTOR pathway genes; lesion size depends on how early in the 33 mitotic cycles of periventricular neuroepithelium the mutation is first expressed. FCD type II and hemimegalencephaly are now confirmed to be the same disorder genetically, and cortical tubers of tuberous sclerosis closely resemble FCD II. No genetic basis has yet been found for FCD type I YW p.488.e6–488.e7.

(b) Diagnostic work-up and management of FCD [5]

Diagnostic work-up

- Phase 1** — interictal EEG, video-EEG and epilepsy-protocol MRI YW p.488.e7.
- High-resolution MRI** — volumetric T1, 3D FLAIR and oblique coronal T2; the EZ of an FCD may extend well beyond the visible margins of the lesion, at times even to the contralateral hemisphere YW p.503–504.
- Advanced imaging** — FDG-PET (regional hypo/hypermotabolism) and ictal SPECT are particularly useful in FCD and help predict post-operative relapse; fMRI and MEG add functional localisation; voxel-based morphometry and machine-learning surface analysis convert an apparently non-lesional MRI into a lesional one YW p.488.e6, p.504.

4. **Phase 2 invasive monitoring** — strip, grid or depth electrodes with neurobehavioural testing, because there may be multiple lesions of which only some are epileptogenic and the EZ may extend beyond the obvious MCD YW p.488.e7. EEG and ECoG localise the focus but cannot distinguish FCD subtypes; SEEG may show some distinction and reveals lesions not evident on MRI YW p.488.e6.

Management

- **Antiseizure medication first**, but FCD is characteristically drug-resistant.
- **Surgery** — lesionectomy, lesionectomy with resection of surrounding epileptic focus, resection including mesial temporal structures where these have become involved, or hemispherectomy; strategy is individualised to the extent of the malformation and the age of the child YW p.488.e7.
- **Intraoperative ECoG** improves FCD resection in children and adults and is equivalent to subdural grid implantation for identifying resection boundaries without the risks of grid placement YW p.576.
- **Completeness of resection is the single determinant of outcome** — seizure freedom in 70% with complete resection versus 22% with incomplete resection in one cohort of 149 FCD patients; a 10-year paediatric series showed 83% favourable outcome after complete versus 11% after incomplete resection YW p.576.
- **Less invasive options** — LITT offers a low-morbidity alternative when the FCD is deep-seated or adjacent to eloquent cortex, with reported seizure freedom of 50-100% but around 40% in the first series YW p.631.
- **Palliative options** — callosotomy, VNS and responsive neurostimulation where the focus is eloquent, bilateral or undefinable; these rarely cure but significantly reduce seizure frequency YW p.488.e7.

High-yield: type I FCD does worse than type II despite milder histology, because it is more extensive with ill-defined margins, so resection is often subtotal YW p.488.e7. Surgery for FCD should be considered early, not as a last resort YW p.488.e7.

Youmans & Winn 8e — pp. 488.e6, 488.e7, 503, 504, 576, 631

Q P4

DEC 2022 · Paper III · Q9

- Newer anti epileptic drugs. [5]
- Surgical management of epilepsy. [5]

ANSWER

(a) Newer antiepileptic drugs [5]

Generations

Youmans stratifies AEDs by generation. First generation includes bromide, phenobarbital, ethosuximide, phenytoin, benzodiazepines, carbamazepine and valproate. **Second generation:** felbamate, gabapentin, lamotrigine, topiramate, tiagabine, levetiracetam, oxcarbazepine, zonisamide, pregabalin. **Third generation:** lacosamide, rufinamide, vigabatrin, perampanel, eslicarbazepine, brivaracetam and cannabidiol (Epidiolex) YW p.497.

Mechanisms (Table 81.1)

Mechanism	Newer agents
Sodium channel	Eslicarbazepine, lacosamide, lamotrigine, oxcarbazepine, rufinamide, topiramate, zonisamide, felbamate
Calcium channel	Gabapentin, lamotrigine, oxcarbazepine, pregabalin, topiramate, zonisamide
GABA	Gabapentin, pregabalin, tiagabine, topiramate, vigabatrin
Glutamate	Felbamate, oxcarbazepine, topiramate

Carbonic anhydrase	Topiramate, zonisamide
Other	Brivaracetam, lacosamide, levetiracetam, perampanel

YW p.497. Metabolism matters in neurosurgical patients: gabapentin, levetiracetam, pregabalin and vigabatrin are **renally** excreted, the remainder largely hepatic **YW p.498.**

Efficacy and tolerability

Newer generations are **not** more efficacious, but are generally better tolerated **YW p.498.** SANAD (nonblinded randomised trial of carbamazepine, lamotrigine, oxcarbazepine, gabapentin, topiramate) showed lamotrigine best tolerated, with lamotrigine and carbamazepine most efficacious; zonisamide was noninferior to carbamazepine; levetiracetam was better tolerated but carbamazepine/valproate minimally more effective; in the elderly VA study gabapentin and lamotrigine were better tolerated, making lamotrigine an optimal overall choice **YW p.499.**

Practical points

- Levetiracetam is favoured in neurosurgery: no drug-drug interactions, IV formulation, safe in hepatic and (dose-adjusted) renal failure; key adverse effect is **agitation** with mood lability and irritability **YW p.498–499**.
- Newer IV formulations: levetiracetam, lacosamide, brivaracetam; in status epilepticus levetiracetam 60 mg/kg (max 4500 mg) is equivalent to fosphenytoin 20 mg/kg and valproate 40 mg/kg as second line **YW p.499.**
- Lamotrigine needs slow titration (Stevens-Johnson risk) and interacts with valproate **YW p.499.**
- In women of child-bearing age, valproate is the most teratogenic in a dose-dependent manner; lamotrigine and levetiracetam are the most suitable choices in pregnancy. Enzyme-inducing drugs (phenytoin, phenobarbital, carbamazepine, topiramate, oxcarbazepine) reduce hormonal contraceptive efficacy, while hormonal contraception lowers lamotrigine levels **YW p.499–500.**

(b) Surgical management of epilepsy [5]

Prerequisite

Surgery follows a presurgical evaluation that localises the epileptogenic zone and its relation to eloquent cortex — video-EEG plus high-resolution MRI are the highest-yield tests, supplemented by PET, SPECT, MEG, fMRI and, when needed, SEEG or subdural grids **YW p.503.**

Procedures and outcomes

Type	Procedure	Indication	Outcome
Resective	Anteromedial temporal lobectomy	Mesial TLE	Engel class I in 66% at 10 years
Resective	Selective amygdalohippocampectomy	Mesial TLE, spares lateral neocortex	60-90% seizure free; less verbal memory decline in dominant side
Resective	Extratemporal (frontal, parietal, occipital, insular) resection	Lesional/nonlesional ETLE	Frontal Engel I ~50%; parietal Engel I-II 53-82%
Ablative	LITT, radiofrequency thermocoagulation, radiosurgery	MTLE, FCD, hypothalamic hamartoma, PVNH	Lower cure rate than open surgery, lower morbidity
Disconnective	Hemispherotomy	Unihemispheric damage with hemiparesis	75-90% seizure free; 85.7% Engel I for hemispherotomy vs 66.1% Rasmussen technique
Palliative	Corpus callosotomy	Drop attacks, multifocal epilepsy	88.2% seizure control after total, 58.6% after anterior callosotomy

Palliative	Multiple subpial transection	Focus in eloquent cortex	Disrupts horizontal gray-matter spread
Neuromodulation	VNS, ANT-DBS, RNS	Not resectable / failed resection	50% responder rates; not curative

Sources: ATL YW p.601; SAH YW p.603; frontal and parietal series YW p.607, p.611; ablation YW p.631; hemispherotomy technique comparison YW p.644; callosotomy meta-analysis and MST rationale YW p.615.e3–615.e4; neuromodulation YW p.659.

Outcome classification

Report results with the Engel scale (class I free of disabling seizures, II rare disabling seizures, III worthwhile improvement, IV no worthwhile improvement) or the ILAE scale, which grades outcome 1-6 from completely seizure free to more than 100% increase in seizure days YW p.667, p.667.e2.

High-yield: more than 50% remain seizure free beyond 10 years after anterior temporal lobectomy; seizure freedom at 2 post-operative years is the best predictor of long-term outcome YW p.667.e3.

Youmans & Winn 8e — pp. 497, 498, 499, 500, 503, 601, 603, 607, 611, 615.e3, 615.e4, 631, 644, 659, 667, 667.e2, 667.e3

Q P5

OCT 2023 · Paper III · Q3

- Definition of intractable epilepsy. [2]
- Symptomatic zone in epilepsy. [2]
- Side effects of Levetiracetam. [2]
- Types of hemispherectomy and indications. [4]

ANSWER

(a) Definition of intractable (drug-resistant) epilepsy [2]

Element	Statement
Operational definition	Failure of two adequately selected and dosed antiepileptic drugs
Evidence base	Kwan-Brodie Glasgow cohort: 47% seizure free on the first AED, 13% on the second, 3.7% on the third, <=1% thereafter
Consequence	Surgical therapy should be considered once this point is reached

YW p.497. An epilepsy surgery evaluation should follow the diagnosis of pharmacoresistance, defined as failure to respond to two or more appropriately chosen and used antiepileptic medications YW p.503. Such patients have less than a 5% chance of being successfully treated with medication alone YW p.667. The ERSET trial established that unsatisfactory seizure control at **2 years** is sufficient to warrant referral for epilepsy surgery YW p.615.e1.

(b) Symptomatogenic zone [2]

Auras, lateralising signs and ictal semiology all reflect the **symptomatogenic zone**. This does **not** imply that the seizure starts there; rather, it is the first non-silent area of the brain to be affected by the discharge — the seizure may begin in a clinically silent region and only produce symptoms after it propagates YW p.605. Hence the changing signs through the ictus indicate the route of spread, and the symptomatogenic zone must be distinguished from the ictal-onset zone and the true epileptogenic zone, which is the target of resection YW p.503. In practice a left homonymous lower quadrantic visual aura localises to the superior bank of the right calcarine sulcus YW p.504.

(c) Side effects of levetiracetam [2]

- **Key adverse effect: agitation**, presenting as newly acquired mood lability and irritability **YW p.499**.
- Otherwise a favourable profile — no significant drug-drug interactions, available intravenously, and therefore widely used in neurosurgical practice **YW p.499**.
- Renally excreted; dose adjustment (or dosing immediately after dialysis) is required in renal failure, while it remains a good choice in hepatic failure **YW p.498**.
- In a randomised comparison with intravenous phenytoin, levetiracetam trended towards fewer major but more minor side effects **YW p.499**.
- It is one of the preferred agents in women of childbearing age/pregnancy **YW p.499**.

(d) Types of hemispherectomy and indications [4]

Types

Technique	Principle
Anatomic hemispherectomy	Krynauw 1950; removal of the whole hemisphere. Abandoned for high late morbidity — superficial cerebral haemosiderosis, hydrocephalus (52% in the Montreal series, 33% from haemosiderosis) and deaths
Hemicortectomy / hemidecortication	Disconnection achieved by removing all hemispheric cortex
Functional hemispherectomy (Rasmussen)	Removal of two segments — temporal lobe and en bloc central suprasylvian cortex — plus callosotomy and frontal/parietal/occipital disconnection. Mathern variant adds a large central peri-insular resection including basal ganglia
Hemispherotomy / hemispheric deafferentation	Disconnection of the cortical mantle from the basal ganglia bloc with little or no tissue removal: peri-insular (opercular window), transsylvian keyhole deafferentation, and vertical parasagittal (Delalande) / central vertical hemispherotomy

YW p.636. Rasmussen showed anatomic and functional hemispherectomy gave similar seizure freedom (83% vs 85%) but raised-pressure complications fell from 35% to 7% **YW p.636**. Holthausen's 328-case review found hemispherotomy best (85.7% Engel I) compared with the Rasmussen technique (66.1%) and hemicortectomy/hemidecortication (60.7%) **YW p.644**.

Indications

- Severe unihemispheric damage causing drug-resistant epilepsy, usually with hemiparesis, hemianopia and developmental delay **YW p.636**.
- Straightforward for inborn or perinatal unihemispheric lesions presenting in infancy/early childhood with frequent intractable seizures **YW p.637**.
- Catastrophic epilepsy of infancy from severe hemispheric damage or inborn malformation **YW p.637**.
- Specific aetiologies: perinatal infarct/porencephaly, hemimegalencephaly, multilobar cortical dysplasia and polymicrogyria, Sturge-Weber syndrome, Rasmussen encephalitis, hemiconvulsion-hemiplegia-epilepsy syndrome **YW p.637**.
- Also performed in adults with good seizure-freedom rates **YW p.637**.
- **Contraindicated** if the presurgical evaluation cannot show that all typical ictal activity arises from the affected hemisphere; independent contralateral seizures are a relative contraindication **YW p.638**.

High-yield: unavoidable side effects to be consented for are complete hemianopia and loss of fine pincer and

fine foot movement; hydrocephalus needing a shunt occurs in 8-30% **YW p.639, p.644.**

Youmans & Winn 8e — pp. 497, 498, 499, 503, 504, 605, 615.e1, 636, 637, 638, 639, 644, 667

Q P6

OCT 2023 · Paper III · Q7

- a) Magnetoencephalogram. [4]
- b) Sterio EEG advantages. [3]
- c) Callosotomy indications and its side effects. [3]

ANSWER

(a) Magnetoencephalography [4]

Principle

MEG is non-invasive and painless, involving no external magnetic field, electricity, x-rays or radioactivity. It measures the very weak magnetic fields generated by summated postsynaptic **intradendritic** currents of cortical pyramidal cells; parallel dendritic orientation permits spatial summation and synchronous firing temporal summation. Fields are detected by superconducting quantum interference devices (SQUIDS) cooled in liquid helium to about -269 degrees C, more than 300 arranged in a helmet, inside a magnetically shielded room because environmental noise is 10^7 to 10^9 times the brain's field. Co-registering the dipole sources on the patient's MRI gives magnetic source imaging (MSI) **YW p.523.**

Advantages

- Signals are not distorted, filtered or weakened by CSF, meninges, skull or scalp, so simple source models give accurate localisation — spatial resolution reported as less than 5 mm **YW p.523, p.525.**
- Superior spatial sampling: a few hundred sensors covering the whole head versus 20-30 scalp EEG electrodes **YW p.523.**
- Particularly valuable where anatomy is altered (post-operative, post-traumatic) and EEG localisation misleads **YW p.523.**

Limitations

Immobile and expensive (about \$3 million plus a shielded room; 80-100 L helium per week); sensitive to metallic and movement artefact; detects **tangential** sources only; poor sensitivity to deep sources (e.g. cingulate); labour-intensive analysis; and it gives no estimate of source size **YW p.525.**

Clinical use

MEG identifies epileptogenic tissue non-invasively and, with mapping of eloquent cortex, produces neuronavigational maps used intraoperatively. It chiefly provides **interictal** recordings and defines the irritative zone; seizures are captured in only about 10% of studies. It detects interictal spikes in 47% of patients with no EEG spikes, and captures spikes in over 64% of patients with insular epilepsy — a group often misdiagnosed as temporal, where MEG may be decisive in justifying invasive monitoring. Only two indications are reimbursed in the United States: spontaneous epileptiform localisation (CPT 95965) and presurgical mapping of eloquent cortex (CPT 95967). A spikeless MEG does not exclude epileptogenic potential **YW p.525, p.527.**

(b) Advantages of stereo-EEG [3]

1. **Access to deep structures** — mesial temporal, opercular-insular, cingulate, interhemispheric and posterior orbitofrontal regions and the depths of sulci, which subdural grids sample poorly since most cortex is buried in sulci **YW p.505-506.**
2. **Three-dimensional network analysis** — SEEG is not merely a depth-electrode technique but a methodology based on anatomo-electro-clinical analysis describing the 3D spatio-temporal organisation of the epileptogenic zone, sampling the lesion, the hypothesised onset zone and the propagation pathways along

the electrode trajectory YW p.506, p.563.

3. **Extensive and bihemispheric exploration** without a craniotomy, including after a previous failed subdural study, where re-exploration carries the risk of adhesions YW p.505–506.
4. **Therapeutic potential** — radiofrequency thermocoagulation can be delivered through the SEEG electrodes, which is especially effective in periventricular nodular heterotopia YW p.506.
5. **Low morbidity and good yield** — in a 200-patient/2663-electrode series total morbidity was 2.5%, the EZ was localised in 77% and 67.8% of those resected were Engel class I; other large series report haemorrhage in 0.4–2.9% of patients (0.03–0.2% per electrode). Orthogonal implantation is safer and more accurate than oblique trajectories, which deflect against the skull and are longer YW p.567, p.568.

The sole remaining indication favouring subdural grids is the need for contiguous functional mapping of eloquent (especially language) cortex YW p.506.

(c) Corpus callosotomy — indications and side effects [3]

Indications

- **Atonic (drop) attacks** — best results, 80–100% reduction; patients with atonic seizures benefit most and may become free of drop attacks entirely YW p.615.e2, p.615.e3.
- Unresectable or multifocal seizure foci; Lennox-Gastaut syndrome; Rasmussen encephalitis; infantile hemiplegia YW p.615.e2.
- Only after a focal resectable lesion has been excluded, since callosotomy is palliative and never curative YW p.615.e2.
- VNS is now commonly trialled first because it is less invasive; VNS failures remain good callosotomy candidates YW p.615.e2, p.615.e4.

Side effects and complications

- **Approach-related** (2.4–6%): epidural and subdural haematoma, meningitis, deep venous thrombosis; external hydrocephalus if the lateral ventricle is entered YW p.615.e3.
- **Acute disconnection syndrome** in up to 89% after extended callosotomy — mutism, a supplementary-motor-area-like picture with paresis of the non-dominant leg, incontinence and reduced spontaneous speech, apraxia and hypotonia of the non-dominant hand, repetitive grasping and bilateral Babinski responses; worse after single-stage complete section YW p.615.e3.
- **Chronic disconnection syndromes** (45–80%, commoner in adults): alien hand (frontal), poor sound localisation (temporal), tactile dysnomia and pure word blindness (parietal), visual suppression (occipital); permanent interhemispheric sensory dissociation after complete or posterior section; greater language risk with crossed cerebral dominance. Nei's comparison showed better seizure control than VNS but a 3.8% permanent complication rate versus none with VNS YW p.615.e3, p.615.e4.

High-yield: staged (anterior first) callosotomy is preferred to limit disconnection morbidity; complete section gives 88.2% seizure control versus 58.6% for anterior section alone YW p.615.e3.

Youmans & Winn 8e — pp. 505, 506, 523, 525, 527, 563, 567, 568, 615.e2, 615.e3, 615.e4

Q P7

MAY 2024 · Paper III · Q1

- a) Deep Brain Stimulation in Epilepsy. [5]
- b) Lesioning in neurosurgery. [5]

ANSWER

(a) Deep brain stimulation in epilepsy [5]

Rationale and targets

Several major circuits have been targeted for neuromodulation of seizures: the circuit of Papez (anterior thalamic nucleus, mammillary body, hippocampus, entorhinal cortex, cingulate), the cerebellar-cortical circuit, the basal ganglia (caudate, subthalamic nucleus, substantia nigra pars reticulata, centromedian nucleus) and vagal projections **YW p.647**.

Anterior nucleus of thalamus (ANT) — the approved target

The ANT is a key station of the limbic circuit of Papez, making it attractive for modulating thalamocortical excitability and the limbic seizure network **YW p.659**.

SANTE trial — multicentre, randomised, double-blind; 110 adults with refractory focal seizures; bilateral ANT leads randomised to 3 months of stimulation (5 V, 90 microsecond pulse width, 145 Hz, 1 min on / 5 min off) or sham **YW p.659**.

- Implantation effect: 21-22% median reduction in both arms at 1 month.
- Third blinded month: median 40.4% reduction with stimulation vs 14.5% sham ($P = .0017$).
- Open-label: median reduction 41% at 1 year, 56% at 2 years, 69% at 5 years; 50% responder rate 43%, 54% and 68% respectively. Only 16% were seizure free for ≥ 6 months during the first 5 years **YW p.659**.
Bilateral ANT-DBS was FDA-approved in 2018 **YW p.667.e15**.

Technical points

The ANT is small (about 10 x 4 x 5 mm) and surrounded by venous structures; SANTE used a transventricular approach with an 8.2% lead revision rate. Direct MRI-based targeting of the anterior half of the ANT close to the mammillothalamic tract is now favoured over fixed coordinates, responders having contacts nearer the ANT-mammillothalamic junction **YW p.659-660**.

Other targets and comparison

Cerebellar stimulation was abandoned after negative crossover trials; centromedian stimulation showed no significant benefit; caudate stimulation is untested in controlled studies; hippocampal DBS gave $>50\%$ reduction in small series. Haemorrhage and infection occur in about 5%. Responsive neurostimulation, the other intracranial option, gave 44% median reduction at 1 year and 53% at 2 years **YW p.667.e15**.

(b) Lesioning in neurosurgery [5]

Principle

A lesion is a controlled, permanent destruction of a node or tract in a pathological circuit. Modalities are radiofrequency thermocoagulation (classically 70 degrees C for 60 seconds), MR-guided laser interstitial thermal therapy, stereotactic radiosurgery and transcranial MR-guided focused ultrasound; reversible tissue change occurs below 60 degrees C and coagulation between 60 and 100 degrees C **YW p.629, p.773**.

Movement disorders

- **Pallidotomy** — for levodopa-induced dyskinesia, severe wearing-off/on-off fluctuations with rigidity, bradykinesia and tremor, off-period dystonia and off-period gait disturbance. Levodopa responsiveness predicts benefit; atypical parkinsonism must be excluded. Bilateral pallidotomy is generally avoided because of risk to speech, swallowing, cognition and gait **YW p.723**.
- **Subthalamotomy** — physiological basis is STN hyperactivity from indirect-pathway dysregulation; gives greater reduction in dopaminergic medication **YW p.723**.
- **VIM thalamotomy** — the most effective lesion for tremor, superior to GPi and STN lesioning for tremor; extremity tremor responds better than head or voice tremor; little effect on rigidity, bradykinesia or gait. Main complication is intracranial haemorrhage (1.5-6%), plus facial paresis, dysarthria, hemiparesis, dystonia, dyspraxia and sensory loss **YW p.728**.

- **MRgFUS thalamotomy** is incisionless and FDA-approved for unilateral essential tremor and tremor-dominant Parkinson disease, with efficacy similar to other ablative and neuromodulatory techniques **YW p.771**.

Epilepsy

- **LITT** for mesial temporal lobe epilepsy, focal cortical dysplasia, insular epilepsy, hypothalamic hamartoma, tuberous sclerosis, cavernoma and even callosotomy — lower seizure-freedom rate than open resection but reduced complications, reduced morbidity and shorter stay, and it does not preclude later open surgery **YW p.629, p.631**. For hypothalamic hamartoma 97% were seizure free at 1 year in one large series with memory loss in about 1.5% versus up to 15% after open resection **YW p.633**.
- **Gamma Knife radiosurgery** for hypothalamic hamartoma: Engel class I 39.6%, class I-II 68.8% overall and 75% in types I-III, with no permanent neurological deficit **YW p.622**.
- **Radiofrequency thermocoagulation through SEEG electrodes** is a therapeutic use of the diagnostic implant, particularly for periventricular nodular heterotopia **YW p.506**.
- **Disconnective "lesions"** — corpus callosotomy and multiple subpial transection, the latter disrupting horizontal gray-matter spread while sparing the vertical cortical columns **YW p.615.e4**.

Psychiatric and pain

Anterior cingulotomy (interrupting connections between dorsal anterior cingulate, orbitofrontal cortex, amygdala and hippocampus) and anterior capsulotomy (lesioning the anterior limb of the internal capsule) are used for refractory obsessive-compulsive disorder; mean Y-BOCS reduction is 37% for cingulotomy and 57% for capsulotomy, and lesions may be made by radiofrequency, Gamma Knife or MRgFUS **YW p.807**.

High-yield: ablation is irreversible and non-titratable; deep brain stimulation is the reversible alternative and allows safe bilateral treatment **YW p.723, p.778**.

Youmans & Winn 8e — pp. 506, 615.e4, 622, 629, 631, 633, 647, 659, 660, 667.e15, 723, 728, 771, 773, 778, 807

Q P8

MAY 2024 · Paper III · Q2

- Focal cortical dysplasia classifications. [2]**
- Diagnostic work up of a suspected case of FCD. [4]**
- Treatment of FCD. [4]**

ANSWER

(a) Focal cortical dysplasia — classification [2]

ILAE type	Defining histology	Note
I	Abnormal radial/microcolumnar cortical lamination with morphologically normal neurons	Often MRI-negative; grey-white blurring
IIa	Dyslaminar with dysmorphic (megaloctytic) neurons , no balloon cells	Transmantle sign on MRI
IIb	Dysmorphic neurons plus balloon cells	mTOR pathway somatic mutation
IIIa	FCD I or II with hippocampal sclerosis	
IIIb	FCD with a cortical tumour	
IIIc	FCD with a vascular malformation	
IIId	FCD with infarct, porencephalic cyst or other lesion	

All FCDs are congenital and all are highly epileptogenic, seizures being the presenting symptom YW p.488.e6. The ILAE scheme (types I, II and III) describes subtypes with different clinical presentation, topographic localisation and surgical response YW p.576.

(b) Diagnostic work-up of suspected FCD [4]

Phase 1 — non-invasive

1. **Semiology and history** — FCD type I typically arises in posterior temporal or parieto-occipital cortex with seizure onset in early infancy, often with learning disability, cognitive and behavioural disorder YW p.488.e6.
2. **Video-EEG** in an epilepsy monitoring unit for interictal and ictal recording YW p.488.e7.
3. **High-resolution epilepsy-protocol MRI** — volumetric T1, 3D FLAIR, oblique coronal T2. Look for the transmantle sign of FCD II (grey matter extending from ventricular wall to the depth of a sulcus) and for grey-white blurring in FCD I. Remember the epileptogenic zone may extend well beyond the visible lesion margins, occasionally even to the contralateral hemisphere YW p.488.e6, p.504.
4. **FDG-PET and ictal SPECT** — useful in identifying foci not apparent on standard MRI in FCD I, in confirming FCD II lesions and in predicting post-operative relapse; ictal SPECT/SISCOM is most helpful in FCD and MRI-negative focal epilepsy YW p.488.e6, p.504.
5. **MEG and fMRI** for source localisation and eloquent cortex; **MRI post-processing** with voxel-based morphometry and automated surface-based machine-learning detection to convert an apparently non-lesional study into a lesional one YW p.504.

Phase 2 — invasive

Indicated for MRI-negative or discordant cases, multiple potential lesions, or an EZ near eloquent cortex YW p.505. Strip, grid or depth electrodes with neurobehavioural testing are used because only some lesions may be epileptogenic and the EZ may lie outside the obvious malformation YW p.488.e7. EEG and ECoG localise but cannot distinguish FCD types; SEEG may show some distinction and can reveal lesions not evident on MRI, and is well suited to bilateral or multifocal interrogation YW p.488.e6, p.506.

(c) Treatment of FCD [4]

Medical

Antiseizure medication is first-line but FCD is characteristically drug-resistant, so surgical referral should not be delayed — surgery should be considered early in the course and not as a desperate last choice YW p.488.e7.

Surgical options

1. **Simple lesionectomy** for a discrete, non-eloquent lesion YW p.488.e7.
2. **Lesionectomy plus resection of the surrounding epileptic focus** — tailored corticectomy guided by intraoperative ECoG, which significantly improves FCD resection in both children and adults and is equivalent to subdural grids for identifying boundaries without their risks YW p.576.
3. **Resection including mesial temporal structures** where longstanding seizures have rendered them epileptogenic YW p.488.e7.
4. **Hemispherectomy/hemispherotomy** for hemimegalencephaly, which is genetically the same disorder as FCD type II YW p.488.e7.
5. **LITT** — a lower-morbidity option when the FCD is deep-seated or adjacent to eloquent cortex; reported seizure freedom ranges from 50% to 100%, with only about 40% in the first reported series, so it is less effective than open resection YW p.631.
6. **Palliative** — corpus callosotomy, vagus nerve stimulation and responsive neurostimulation where the focus is eloquent, bilateral or undefinable; rarely curative but they reduce seizure frequency significantly YW p.488.e7, p.2043.

Outcome and its determinants

- Complete resection is decisive: 70% seizure free with complete resection versus 22% with incomplete resection in a cohort of 149 FCD patients; 83% versus 11% in a 10-year paediatric series YW p.576.
- Surgery for malformations of cortical development cures just over 50% and gives Engel I-II in about 70%; better outcome is associated with shorter seizure duration, absence of generalised seizures, younger age and total resection YW p.488.e7.
- FCD type I does **worse** than type II despite milder histology, because it is more extensive with poorly defined margins and resection is often subtotal YW p.488.e7, p.576.
- Long-term cognitive function improves post-operatively, especially in FCD type I YW p.488.e7.

High-yield: FCD type II is an mTOR-pathway somatic mutation and is the same entity as hemimegalencephaly; cortical tubers of tuberous sclerosis closely resemble FCD type II YW p.488.e6, p.488.e7.

Youmans & Winn 8e — pp. 488.e6, 488.e7, 504, 505, 506, 576, 631, 2043

Q P9

MAY 2024 · Paper III · Q6

- a) Standard Temporal lobectomy. [3]
- b) Selective amygdalohippocampectomy. [3]
- c) Surgery for non-temporal epilepsy. [2]
- d) Wada test. [2]

ANSWER

(a) Standard (anteromedial) temporal lobectomy [3]

Extent

The classical standard resection of lateral temporal neocortex measured posteriorly from the temporal tip is 4-5.5 cm on the dominant side and 5.5-6.5 cm on the non-dominant side. Modern practice (Spencer modification) spares the superior temporal gyrus regardless of side unless ictal events are proven there, and limits the inferolateral resection to about 3 cm; language mapping is recommended if the resection extends beyond 3 cm posteriorly or must include the STG YW p.596, p.597.

Key technical steps

1. Head rotated and extended, vertex down 10 degrees; expose at least 5 cm of temporal and orbitofrontal lobe.
2. Resect 3-3.5 cm of middle and inferior temporal cortex subpially, preserving the vein of Labbe.
3. Open the temporal horn along the fusiform gyrus; bring in the microscope; resect the anterior uncus to expose the ICA and the knee of the MCA.
4. Identify the choroid plexus and **inferior choroidal point** (velum terminale) and draw the imaginary MCA-choroidal line; staying below it allows safe amygdalar resection without entering the temporal stem or injuring basal ganglia.
5. Elevate the pes hippocampi, coagulate PCA perforators, elevate the fimbria off the thalamus and remove the hippocampus from body to tail at the collateral eminence, preserving the posterior inferior temporal branch of the PCA YW p.598, p.599, p.600.

Complications and outcome

Mortality 0.1-0.5%. Upper quadrantanopia ("pie in the sky") is the commonest deficit, from injury to the Meyer loop, affected in up to 90% of temporal lobectomies; also naming errors, temporalis wasting, frontalis palsy, diplopia, third-nerve palsy and hemiparesis. Engel class I is achieved in 66% at 10 years; unfavourable predictors are rapid seizure spread, non-lesional MRI, PET-EEG discordance and a history of generalised tonic-clonic seizures YW p.597, p.601.

(b) Selective amygdalohippocampectomy [3]

Approaches

- **Transcortical** (Niemeyer 1958, originally transventricular via the middle temporal gyrus; later variants through inferior or superior temporal gyrus) — spares much cortex but transgresses cortex and some temporal stem.
- **Transsylvian** (Wieser and Yasargil 1982) — through the sylvian fissure and temporal insula into the temporal horn; preserves all superficial cortex but transects a large segment of temporal white matter stem.
- **Subtemporal** (Hori 1993) — dissects beneath the temporal lobe to the parahippocampal gyrus and enters the ventricle through the collateral sulcus; avoids both anterolateral cortex and temporal stem and can be done through a 2 cm keyhole craniotomy at the middle fossa floor **YW p.602**.

Technique (subtemporal keyhole)

Supine, head turned 90 degrees, vertex slightly down; 3-4 cm curvilinear incision anterior to the tragus; 2 cm keyhole craniotomy flush with the middle fossa floor; subtemporal CSF egress for relaxation (fixed retractors rarely needed); enter the collateral sulcus to the temporal horn; debulk the amygdala anterosuperior to the choroidal fissure; make the posterior hippocampal cut, free the hippocampus from the choroidal fissure and its feeders, remove it en bloc and chase the tail beyond the tectal plate **YW p.602**.

Evidence

No randomised trial exists. Almost every direct comparison shows **equivalent seizure freedom, 60-90% at 3-6 years**, between SAH and ATL. Cognitive studies mostly favour SAH, the most consistent finding being a smaller decline in verbal memory after dominant-side SAH (31% worse versus 43% after ATL in Clusmann's series), although a meta-analysis found no difference **YW p.603**.

(c) Surgery for non-temporal (extratemporal) epilepsy [2]

Any epilepsy arising outside the temporal lobe. Frontal lobe epilepsy is the second commonest; localisation is hard because the frontal lobe is 35-45% of cortical volume with much buried cortex and seizures propagate widely, so SEEG plus PET, SPECT or MEG is often needed. Resection is limited by motor cortex, SMA and Broca area, and seizure freedom is around 50% against roughly 70% for temporal lobe epilepsy (Englot meta-analysis 45.1%) **YW p.605, p.607**. Parietal series give Engel I-II of 53-82%; commonest deficits are incomplete Gerstmann or hemisensory syndrome, usually temporary, and posterior-quadrant multilobar disease may be disconnected rather than resected **YW p.611**.

(d) Wada test [2]

Intracarotid amobarbital test to determine hemispheric language dominance and whether the contralateral medial temporal lobe can support memory **YW p.538**. Objects and pictures are presented for naming and later recognition during hemianaesthesia monitored by EEG and grip strength; language is tested by repetition, comprehension and reading **YW p.538**. Amobarbital 100-125 mg is standard (historically 150-200 mg); the 2003 shortage led to etomidate, propofol, methohexital and secobarbital, generally 2-6 mg, with equivalent results **YW p.539**. Memory asymmetry of at least 20% indicates the contralateral hemisphere can support learning; **reversed asymmetry** predicts post-operative amnesia. Pass criteria vary from 50% to 75% correct **YW p.538**. A 677-patient series reported adverse events in 11% — encephalopathy 7.2%, seizure 1.2%, stroke 0.6%, dissection 0.4% — with persistent deficit in 0.6% **YW p.539**. It remains relevant before laser ablation of the hippocampus, since hippocampal memory is still disrupted **YW p.539**.

Youmans & Winn 8e — pp. 538, 539, 596, 597, 598, 599, 600, 601, 602, 603, 605, 607, 611

Q P10

JAN 2025 · Paper III · Q6

- a) Vagal nerve stimulation. [5]
b) Types of epilepsy surgery. [5]

ANSWER

(a) Vagus nerve stimulation [5]

Device and mechanism

VNS was the first electrical stimulation therapy for epilepsy approved by the FDA (1997). Coiled stimulation leads are placed around the **left** vagus nerve and connected subcutaneously to a generator below the clavicle. The mechanism remains unclear: vagal sensory afferents project to the nucleus of the solitary tract, which projects broadly to thalamus, amygdala, anterior cingulate and frontal and parietal cortex; release of noradrenaline into the hippocampus is a marker of effective stimulation in limbic seizure models **YW p.647, p.653**. Newer devices (AspireSR) trigger extra stimulation on detecting seizure-related tachycardia, and patients can abort an aura with a hand-held magnet **YW p.653**.

Evidence

Two industry-sponsored randomised double-blind trials compared high with low stimulation.

- **EO3** (114 implanted): high stimulation 0.25-3.0 mA, 20-50 Hz, 500 microsecond pulses, 30-90 s on / 5-10 min off. Seizure reduction -24.5% vs -6.1% ($P = .01$); 31% achieved $\geq 50\%$ reduction but **none** became seizure free.
- **EO5** (196 analysed): high stimulation 30 s every 5 min of 500 microsecond, 30 Hz pulses vs low stimulation 30 s every 3 h of 130 microsecond, 1 Hz pulses; mean total reduction 27.9% vs 15.2% ($P = .04$). Efficacy improves with time — 45% at 12 months and 52% at 18 months **YW p.653**. Long-term, seizure frequency fell 26% at 1 year, 30% at 5 years and 52% at 12 years **YW p.667.e15**.
- The American Academy of Neurology concluded VNS is possibly effective in achieving $>50\%$ seizure reduction in children and recommended it as adjunctive treatment **YW p.653**.

Indications

Palliative adjunctive treatment in drug-resistant epilepsy where resection is not possible or has failed — multifocal epilepsy, foci in eloquent cortex, syndromic epilepsies; commonly trialled before corpus callosotomy because it is less invasive, and callosotomy remains available if VNS fails **YW p.615.e1, p.615.e2**.

Adverse effects

Voice alteration and hoarseness (37.2% with high stimulation in EO3), throat or neck pain, cough, dyspnoea, paraesthesia and headache **YW p.653**. Device-related: infection needing antibiotics or removal, transient left vocal cord paralysis with hoarseness and aspiration, lead fracture, wound erythema, abscess, malfunction and diminished speech volume; electrode removal from the nerve can be difficult. No increase in SUDEP has been identified **YW p.667.e15**.

(b) Types of epilepsy surgery [5]

Category	Procedure	Indication	Outcome
Resective	Anteromedial temporal lobectomy	Mesial TLE	Engel I 66% at 10 years; $>50\%$ seizure free beyond 10 years
	Selective amygdalohippocampectomy	Mesial TLE, especially dominant side	60-90% seizure free at 3-6 years; less verbal memory loss
	Frontal resection/lobectomy	Frontal lobe epilepsy	Engel I $\sim 50\%$; meta-analysis 45.1%

Appendix A · Paper-wise index

Every question in the order it was asked, with the chapter it is answered in.

JUN 2020 · Paper I

10 questions · 100 marks

Q1	Intracranial pressure(ICP): a) Role of ICP monitoring in head injury. b) Various methods of ICP mon...	Ch. B
Q2	Optic nerve (2nd cranial nerve): a) Anatomy. b) Course. c) Clinical localization	Ch. A
Q3	Surgical anatomy of cerebellopontine angle: a) Locations and relations. b) Contents. c) Enumerate va...	Ch. A
Q4	Craniovertebral junction anomaly: a) Anatomy of the CV junction. b) Craniometrics analysis to diag...	Ch. D
Q5	a) Anatomy of blood brain barrier. b) Biological significance of areas devoid of blood brain barriers	Ch. B
Q6	Cerebrospinal fluid: a) Pathway and normal physiological values. b) Pathology due to blockage in fl...	Ch. I
Q7	a) Ligamentum denticulatum. b) Blood supply of spinal cord. c) Conus medullaris	Ch. A
Q8	About Traumatic brain injury: a) Discuss pathogenesis and pathology of axonal injury. b) Classificat...	Ch. C
Q9	Elaborate vein of Galen: a) Tributaries. b) Surgical importance	Ch. A
Q10	About research methodology: a) Blinding in randomized controlled trial. b) Levels of evidence	Ch. U

JUN 2020 · Paper II

10 questions · 100 marks

Q1	Intraoperative Neurophysiologic monitoring: a) Indications of IONM. b) Necessary precautions. c)...	Ch. R
Q2	Discuss about visual field defects with lesions at: a) Chiasm. b) Lateral geniculate body. c) Occipital...	Ch. A
Q3	Normal pressure hydrocephalus: a) Differential diagnosis. b) Programmable shunts. c) Predictors of ...	Ch. I
Q4	Spinal AVM: a) Classification. b) Clinical presentations. c) Angiographic finding	Ch. K
Q5	Elaborate Horner's syndrome: a) Clinical features. b) Causes. c) Pathway of sympathetic system	Ch. A
Q6	Post SAH Vasospasm: a) Theories of pathogenesis of vasospasm in spontaneous SAH. b) Role of cal...	Ch. J
Q7	Endoscopic third Ventriculostomy: a) Indications and procedure. b) Complications	Ch. I
Q8	Ventriculo-peritoneal shunts: a) Sites of lateral ventricular puncture. b) Complications of ventriculo-...	Ch. I
Q9	Ossified Posterior Longitudinal Ligament: a) Classification. b) Pathogenesis. c) Current concepts in...	Ch. N
Q10	Brachial plexus injury: a) Clinical evaluation. b) Draw and label the brachial plexus neatly. c) Curren...	Ch. Q

JUN 2020 · Paper III

10 questions · 100 marks

Q1	Diffusion weighted MRI in Neurosurgery: a) Principles and applications b) Role in brain tumour cha...	Ch. R
Q2	Spinal fusion techniques: a) Techniques of minimally invasive lumbar spine fusion. b) Current conce...	Ch. N
Q3	a) Indications and mechanism of Deep brain stimulation (DBS). b) Advantages of DBS	Ch. P
Q4	Role of stem cells in Neurosurgery: a) Principles. b) Clinical applications. c) Ethical considerations	Ch. R
Q5	Describe Scientific contributions to Neurosurgery: a) Victor Horsley. b) Walter Dandy. c) Cloward	Ch. U
Q6	Tuberculosis of spine: a) Pathophysiology. b) Current techniques in diagnosis. c) Current concepts i...	Ch. O
Q7	Endonasal endoscopic surgery: a) Indications and contraindications. b) Complications. c) Hadad-Bas...	Ch. F
Q8	a) Management options in intracranial aneurysm. b) Recent advances in management of giant intracr...	Ch. J
Q9	Recurrent GBM: a) Diagnosis. b) Indications for surgery c) Timozolamide	Ch. E
Q10	Neurosurgical operating microscope: a) Basic requirements. b) Advanced functions. c) Future deve...	Ch. R

DEC 2020 · Paper I

10 questions · 100 marks

Q1	Draw a labelled diagram of: a) Superior orbital fissure and structures passing through it. b) Floor of f...	Ch. A
Q2	a) Draw a pressure volume curve. b) Define cerebral compliance. c) Effect of cerebral perfusion pres...	Ch. B
Q3	a) Pathogenesis and pathology of traumatic axonal injury. b) How DAI is graded?	Ch. C
Q4	a) The process of apoptosis in brain. b) How apoptosis differs from necrosis?	Ch. B
Q5	a) Innervation of urinary bladder. b) Physiology of micturition	Ch. B
Q6	Hypernatraemia: a) Diagnostic criteria. b) Management. c) Outcome and complications	Ch. T
Q7	Surgical anatomy of anterior cerebral artery: a) Course. b) Segments. c) Branches. d) Clinical effects...	Ch. A
Q8	Phenytoin sodium: a) Mechanism of action. b) Indications. c) Dose. d) Side effects	Ch. T
Q9	Cavernous sinus: a) Anatomy. b) Surgical approaches. c) Parkinson's triangle	Ch. F
Q10	a) Blinding in randomized controlled trial. b) Levels of evidence	Ch. U

DEC 2020 · Paper II

10 questions · 100 marks

Q1	a) Classify Spondylolisthesis. b) Management of Spondylolisthesis	Ch. N
Q2	a) Differentia diagnosis for middle cranial fossa lesions. b) Management of Trigeminal Schwannoma	Ch. F
Q3	Foot drop: different causes, diagnostic evaluation and management	Ch. Q

Q4	Complications of cervical discectomy with relevant diagrams	Ch. N
Q5	a) Types of shunts used for treatment of hydrocephalus. b) Management of slit ventricle syndrome	Ch. I
Q6	Aetiopathology and management of mycotic aneurysms	Ch. J
Q7	a) Differential diagnosis of intraventricular lesions. b) Management and histopathology of choroid p...	Ch. H
Q8	a) What is a depressed skull fracture? b) Management of depressed skull fracture over SSS	Ch. C
Q9	Evaluation and management of tethered cord syndrome	Ch. M
Q10	Spinal AV fistula's: Classification and management	Ch. K

DEC 2020 · Paper III

10 questions · 100 marks

Q1	a) Define trigeminal neuralgia. b) Techniques for management of different neuralgic pain	Ch. P
Q2	Etiology of subdural empyema and its management	Ch. O
Q3	Pre-surgical evaluation & surgical approaches in Pediatric epilepsy	Ch. P
Q4	a) Define tremor. b) Surgical management of tremor	Ch. P
Q5	a) Define non-syndromic craniosynostosis with appropriate diagrams. b) Principles of endoscopic m...	Ch. L
Q6	a) Different categories of thoracic outlet syndrome. b) Management of thoracic outlet syndrome	Ch. Q
Q7	a) Pathophysiology and clinical presentation of carotid artery dissection. b) Evaluation and managem...	Ch. K
Q8	Neurosurgery in COVID-19 pandemic and lesson learnt	Ch. U
Q9	a) Definition and grading of spasticity. b) Treatment options for the management of spasticity	Ch. P
Q10	2016 - WHO classification of Glioblastoma multiforme and its full description	Ch. E

JUN 2021 · Paper I

10 questions · 100 marks

Q1	a) Papez's circuit. b) Area Postrema. c) Optokinetic nystagmus	Ch. A
Q2	Describe briefly microsurgical anatomy of: a) Tentorial hiatus. b) Sellar region	Ch. A
Q3	a) Respiratory centres in the brain. b) Patterns of pathological breathing in brain stem damage	Ch. B
Q4	a) Pituitary apoplexy. b) Neurofibromatosis. c) Ethical issues in a clinical trial	Ch. G
Q5	Facial (VIIth cranial) nerve: a) Nuclei of its origin. b) Intracranial course. c) Supply areas. d) Surgica...	Ch. A
Q6	Management of: a) Jugular foramen lesions. b) Intraspinal lipomas	Ch. F
Q7	Cranio-cerebral missile injuries: a) Pathophysiology and biomechanics. b) Management. c) Complica...	Ch. C
Q8	a) Molecular biology of Medulloblastoma. b) Immunohistochemistry of Pituitary adenomas	Ch. E
Q9	Draw a labelled diagram of: a) Sagittal section of Cranio-vertebral junction. b) Horizontal section of...	Ch. A
Q10	a) Papilloedema. b) Cerebral protection. c) Lacosamide	Ch. B

JUN 2021 · Paper II

10 questions · 100 marks

Q1	Transverse sinus thrombosis: a) Aetio-pathogenesis. b) Diagnosis. c) Management	Ch. K
Q2	Describe systematically: a) Fronto-nasal encephalocele. b) Apert's syndrome	Ch. L
Q3	In a case of Craniopharyngioma in a 8-year-old male child, discuss: a) Clinical features. b) Diagnosis...	Ch. G
Q4	Subarachnoid hemorrhage: a) Grading. b) Complications. c) Risk factors for poor outcome. d) Indica...	Ch. J
Q5	Cervical spondylotic disease: a) Aetio-pathogenesis. b) Natural history. c) Clinical presentations. d) ...	Ch. N
Q6	a) Carpal tunnel syndrome. b) Management of hyperhidrosis	Ch. Q
Q7	a) Surgical approaches to lesions at petrous apex. b) Decompressive craniectomy. c) Types of STA...	Ch. F
Q8	a) Chemotherapy of Glioblastoma multiforme. b) Management of CSF leaks. c) Treatment options fo...	Ch. E
Q9	a) Dumb-bell neurofibromas. b) Hypothalamic hamartomas. c) Choroid plexus papillomas	Ch. N
Q10	Management of: a) Chronic subdural hematoma. b) Normal pressure hydrocephalus. c) Cerebral met...	Ch. C

JUN 2021 · Paper III

10 questions · 100 marks

Q1	Hydatid disease of brain: a) Aetio-pathogenesis. b) Diagnostic evaluation. c) Management	Ch. O
Q2	Stereotactic procedures: a) Principle. b) Types. c) Indications. d) Limitations	Ch. P
Q3	Imaging techniques for: a) Cranio-vertebral junction. b) Eloquent areas of brain. c) Cerebral blood flow	Ch. R
Q4	a) Cerebral microdialysis in traumatic brain injuries. b) Magneto-encephalography. c) Neural plasticity	Ch. R
Q5	Trigeminal neuralgia: a) Definition. b) Aetio-pathogenesis. c) Treatment options	Ch. P
Q6	a) Micro-electrode recording in functional neurosurgery. b) Facial nerve monitoring in vestibular sc...	Ch. R
Q7	A case of spinal tuberculosis: a) Clinical presentations. b) Complications. c) Management	Ch. O
Q8	a) Flow diverters. b) Baclofen pump. c) Programmable shunt	Ch. R
Q9	In relation to the COVID-19 pandemic, discuss: a) Neurological complications of COVID-19. b) Its...	Ch. U
Q10	a) Role of Cyberknife in neurosurgery. b) Cerebral hemispherectomy. c) Key-hole neurosurgery	Ch. S

DEC 2021 · Paper I

10 questions · 100 marks

- Q1 Draw a labelled diagram(s) of: a) Brachial plexus. b) Thalamus and its nuclei Ch. A
- Q2 Sodium valproate: a) Mechanism of action. b) Indications. c) Dose. d) Side effects Ch. T
- Q3 a) Blood supply of spinal cord. b) Visual evoked potential (VEP) Ch. A
- Q4 a) Molecular markers in diagnosis of glioma. b) Pathology of medulloblastoma Ch. E
- Q5 Chronic subdural hematoma: a) Clinical features. b) Pathology. c) Management Ch. C
- Q6 Colloid cyst of third ventricle: a) Clinical features. b) Neuroimaging features. c) Pathology. d) Man... Ch. H
- Q7 Hangman's fracture: a) Classification. b) Clinical features. c) Treatment Ch. D
- Q8 Traumatic diffuse axonal brain injury: a) Pathology. b) Radiological features. c) Management Ch. C
- Q9 Posterior third ventricular tumors: a) Clinical features. b) Radiological features. c) Management Ch. H
- Q10 a) Research hypothesis. b) Bias in scientific study. c) Level of evidence Ch. U

DEC 2021 · Paper II

10 questions · 100 marks

- Q1 a) Various neurotization procedures used in brachial plexus injury. b) Brown Sequard syndrome Ch. Q
- Q2 Acute subarachnoid haemorrhage: a) Clinical features. b) Radiological evaluation. c) Management Ch. J
- Q3 Dysembryoplastic neuroepithelial tumor: a) Clinical features. b) Radiological features. c) Pathology. ... Ch. E
- Q4 Split cord malformation: a) Classification. b) Clinical features. c) Radiological features. d) Management Ch. M
- Q5 a) Horner's syndrome. b) Complications of endoscopic third ventriculostomy (ETV) Ch. A
- Q6 Cervical disc prolapse: a) Clinical features. b) Radiological features. c) Management Ch. N
- Q7 Meralgia paresthetica: a) Etiological factors. b) Clinical features. c) Investigations. d) Management Ch. Q
- Q8 Cavernous hemangioma of cavernous sinus: a) Clinical features. b) Radiological features. c) Manag... Ch. F
- Q9 Vein of Galen malformation: a) Clinical features. b) Radiological features. c) Management Ch. K
- Q10 Dorsal spinal ependymoma: a) Clinical features. b) Radiological features. c) Management Ch. N

DEC 2021 · Paper III

10 questions · 100 marks

- Q1 a) Definition and grading of spasticity. b) Treatment options for the management of spasticity Ch. P
- Q2 Fungal infection of brain: a) Clinical features. b) Radiological features. c) Management Ch. O
- Q3 Extended skull base endoscopic approach in neurosurgery: a) Indications. b) Stages in surgical proce... Ch. F
- Q4 Microvascular decompression: a) Indications. b) Key surgical steps. c) Complications Ch. P
- Q5 Stereotactic radiosurgery (SRS) for arterio-venous malformation (AVM): a) Patient selection. b) Co... Ch. S
- Q6 Deep brain stimulation (DBS): a) Indications. b) Key surgical steps. c) Complications Ch. P
- Q7 a) Robotic neurosurgery. b) Optical coherence tomography Ch. R
- Q8 Discuss various surgical adjuncts used for maximizing extent of tumor resection and minimizing pos... Ch. E
- Q9 Drug resistant epilepsy: a) Preoperative evaluation. b) Surgical techniques for managing drug resistan... Ch. P
- Q10 a) Diffusion tensor imaging (DTI) in neurosurgery. b) Management of post traumatic cerebrospinal f... Ch. R

JUN 2022 · Paper I

10 questions · 100 marks

- Q1 a) Cold Caloric Test : Steps and Interpretations. b) Brun Nystagmus : Pathophysiology and diagnosis Ch. A
- Q2 a) Brain parcellations. b) Connectomes and its importance in neurosurgery Ch. A
- Q3 Describe briefly microsurgical anatomy of: a) Cavernous Sinus. b) Dorsal root entry zone (DREZ) ... Ch. A
- Q4 a) Localisation of various stages of unconsciousness. b) Newer advances in management of Persisten... Ch. B
- Q5 a) Glasgow CT score. b) Adelaide Pediatric Coma Score. c) Pitfalls of Glasgow Coma Score Ch. C
- Q6 a) Neurofibromatosis type 2. b) Os Odontoideum. c) Embryogenesis of C2 vertebra Ch. D
- Q7 a) Trigeminal (5th) Cranial Nerve Nuclei of its origin. b) Intracranial Course. c) Supply areas. d) Su... Ch. A
- Q8 a) Split cord malformations : Classification. b) Limited Dorsal Myeloschisis. c) Secondary neurulati... Ch. M
- Q9 a) Pathophysiology and treatment of Growing skull fractures. b) Compound depressed fractures over ... Ch. C
- Q10 a) Midodrine. b) Fosphenytoin vs Phenytoin. c) Management of brain dead potential organ donor Ch. U

JUN 2022 · Paper II

10 questions · 100 marks

- Q1 a) Branches of internal carotid artery. b) Parts of internal carotid artery. c) Parts and branches of post... Ch. A
- Q2 Draw a schematic cross section diagram of: a) Functional areas (sulci and gyri) of cerebrum seen on l... Ch. A
- Q3 Draw schematic diagram of: a) Brachial plexus and its branches. b) Course of Median nerve, its bran... Ch. A
- Q4 Discuss diagnosis and treatment algorithm for: a) Cerebral vasospasm. b) Cerebral salt wasting Ch. J
- Q5 a) Prerequisites, steps, confounders in Apnea test. b) THOTA Act, 2011 Recommendations Ch. U
- Q6 a) Non neoplastic lesions mimicking CNS tumors. b) Classification of Hippocampal sclerosis in Te... Ch. E

Q7	a) Discuss focal cortical dysplasia classifications. b) Discuss diagnostic workup and management of...	Ch. P
Q8	Discuss advantage and disadvantage of following approaches in management of Petroclival Mening...	Ch. F
Q9	Discuss SLIC and TLICS classifications in spinal trauma and management decisions	Ch. D
Q10	a) CRASH 3 Trial. b) B Ramamurthy. c) SN Bhagwati. d) STITCH Trauma Trial	Ch. U

JUN 2022 · Paper III

10 questions · 100 marks

Q1	Neurocysticercosis: a) Etiopathogenesis. b) Diagnostic evaluation. c) Management	Ch. O
Q2	Lesioning in movement disorders: a) Principle. b) Indications. c) Limitations	Ch. P
Q3	a) Ancillary tests in brain death diagnosis. b) MRS in Neuro-oncology. c) Imaging techniques in do...	Ch. R
Q4	a) ICP monitoring in traumatic brain injuries. b) NIRS (Near infrared spectroscopy) in Neurosurgery...	Ch. R
Q5	Birth plexus injuries: a) Classifications. b) Decision making and timing of surgery. c) Treatment options	Ch. Q
Q6	Osteoporotic D8 vertebral body fracture: a) Discuss diagnostic workup. b) Discuss medical manage...	Ch. N
Q7	a) Endoscopic strategies in management of complex hydrocephalus. b) Endoscopic management of ...	Ch. I
Q8	a) Spinal cord stimulator. b) Antibiotic impregnated ventriculoperitoneal shunts. c) Vertebroplasty. ...	Ch. N
Q9	a) SRS in Neurosurgery. b) Gamma knife in AVMs. c) Proton therapy: Principles and indications	Ch. S
Q10	a) Hirayama Disease: Diagnosis and management. b) Donation after cardiac death	Ch. N

DEC 2022 · Paper I

10 questions · 100 marks

Q1	a) Classification of Meningioma. b) Pathophysiology & Immunohistochemistry of Meningioma. c) M...	Ch. F
Q2	a) Embryology, Morphology & Anatomy of Normal Pituitary Gland. b) Explain Hardy classification ...	Ch. G
Q3	a) Colloid cyst. b) Its Clinical features. c) Colloid cyst management	Ch. H
Q4	Describe Keyhole approaches to Brain Tumour	Ch. F
Q5	a) Hydrocephalus: Pathophysiology and classification. b) Note on Thecopertoneal shunt and its indic...	Ch. I
Q6	Leptomeningeal cyst: a) Classification. b) Approach for Surgery	Ch. C
Q7	a) Endovascular Technique for Tumour Embolization. b) Craniosynostosis	Ch. K
Q8	Classification, Pathophysiology and management of Diffuse axonal injury of Brain	Ch. C
Q9	a) Meta analysis. b) Impact factor	Ch. U
Q10	a) Describe Microsurgical Anatomy of Lateral Ventricle. b) Surgical Approaches to the Tumour of L...	Ch. H

DEC 2022 · Paper II

10 questions · 100 marks

Q1	a) Classification of Occiput-c1 (Cranio-cervical Injury). b) Treatment of C2 fracture	Ch. D
Q2	a) Pathophysiology of SAH. b) Grading of SAH. c) Management of SAH	Ch. J
Q3	a) Stages of Cerebral Abscess. b) Split cord malformation	Ch. O
Q4	a) Various autonomic Nuclei of the Cranial nerves. b) Sympathetic outflow - Anatomy and its supply	Ch. A
Q5	a) Intra-Operative Neuro Physiological monitoring and its indication. b) Various sequences of MRI	Ch. R
Q6	a) Classification of Intradural Tumour. b) Difference between Intradural extramedullary and intrame...	Ch. N
Q7	a) Lipomyelomeningocele. b) Its classification and management	Ch. M
Q8	a) Urodynamic Study. b) Autonomic Bladder	Ch. B
Q9	a) Halfman reflex and its significance. b) Meralgia paresthetica	Ch. Q
Q10	a) Mechanism of action of surgicel. b) Pathophysiology of Arnold-Chiari malformation	Ch. M

DEC 2022 · Paper III

10 questions · 100 marks

Q1	a) Gamma knife. b) Cyber knife. c) SRS	Ch. S
Q2	Role of Nanotechnology in Neurosurgery	Ch. R
Q3	a) What is Intractable pain? b) Surgery for intractable pain	Ch. P
Q4	a) Intra-operative MRI. b) Robotic Stereotactic computer assisted Neurosurgery	Ch. R
Q5	a) Functional Neuro surgery. b) Surgical management of functional neuro surgical diseases	Ch. P
Q6	CSF findings of: a) Pyogenic meningitis. b) Tubercular meningitis. c) SAH	Ch. O
Q7	a) Biplane DSA. b) Endovascular management of Spinal disorders	Ch. K
Q8	a) Spinothalamic Tract. b) Tract of Lissaurers	Ch. A
Q9	a) Newer anti epileptic drugs. b) Surgical management of epilepsy	Ch. P
Q10	Describe various parasitic infection of brain and its clinical manifestation and management	Ch. O

APR 2023 · Paper I

10 questions · 100 marks

Q1	Cranial Nerve II: a) Outline its course. b) Surgical anatomy of the optic chiasma. c) Outline various f...	Ch. A
Q2	a) Draw a labelled diagram of the transverse section at the level of upper pons. b) Outline signs and ...	Ch. A

Q3	Regarding tubercular meningitis: a) Describe the signs and symptoms. b) Diagnostic criterias. c) Co...	Ch. O
Q4	a) Describe cerebral blood flow and its regulation. b) Define brain death and its diagnosis	Ch. B
Q5	a) Describe the GCS scale and its fallacies. b) Pathogenesis and treatment of acute subdural hematoma	Ch. C
Q6	a) Describe various intracranial cystic lesions. b) Surgical technique in removing hydatid cyst. c) Va...	Ch. O
Q7	Regarding ACom aneurysms: a) Describe the surgical anatomy of the region. b) Post operative man...	Ch. J
Q8	a) Discuss ABCDE in the management of trauma patients. b) Describe various clinical trials related t...	Ch. C
Q9	a) Classify and describe Hangman's fracture and their management. b) Evidence based management o...	Ch. D
Q10	a) Describe the recent WHO classification of diffuse gliomas. b) Management of a case of recurrent...	Ch. E

APR 2023 · Paper II

10 questions · 100 marks

Q1	Describe about Chiari malformation: a) Classification. b) Clinical features. c) Management	Ch. M
Q2	Regarding a case of Neurogenic Bladder: a) Pathophysiology and various types. b) Diagnostic evalua...	Ch. B
Q3	Management of: a) Carotico Cavernous Fistula (CCF). b) Orbital tumours	Ch. K
Q4	Regarding NPH: a) Describe the clinical features. b) Diagnostic criterias. c) Various shunts used for t...	Ch. I
Q5	a) Describe MRC motor grading. b) Describe the clinical features and management of spinal intrame...	Ch. N
Q6	a) Clinical presentations of cerebral AVMs. b) Molecular biology and genetics of AVMs. c) Classific...	Ch. K
Q7	A patient presents with sudden headache, right sided ptosis and right sided pupillary dilatation. a) Ou...	Ch. J
Q8	a) Radiobiology and effects of radiation. b) Radiotherapy of gliomas. c) Priciples of Gamma knife	Ch. S
Q9	a) Pathophysiology and differential diagnosis of posterior 3rd ventricular tumour. b) Management al...	Ch. H
Q10	A 43-year-old male with a history of diabetes and hypertension along with bilateral visual impairmen...	Ch. G

APR 2023 · Paper III

10 questions · 100 marks

Q1	Discuss neurological complications of: a) Decompressive craniectomy. b) Deep brain stimulation. c)...	Ch. I
Q2	a) Draw a labelled diagram of transverse section of the upper midbrain. b) Parinaud's syndrome and i...	Ch. A
Q3	a) Percutaneous endoscopic disectomy. b) Dynamic stabilisation of lumbar spine	Ch. N
Q4	a) Techniques of ICP monitoring. b) Trans-cranial Doppler. c) Brain stem evoked potential	Ch. R
Q5	a) Anatomy of cranio-vertebral junction. b) Clinical features of C2 dumbbell neurofibroma. c) Man...	Ch. D
Q6	a) Describe pathophysiology and grading of spasticity. b) Discuss various options of management of...	Ch. P
Q7	Neuropathic pain: a) Definition. b) Etiopathogenesis. c) Management options	Ch. P
Q8	Post Covid-19 CNS fungal infections: a) Causative organisms. b) Factors responsible. c) Clinical pre...	Ch. O
Q9	a) Uses of intraoperative ultrasound. b) Leksell frame and its uses. c) Neuronavigation	Ch. R
Q10	a) Artificial intelligence in neurosurgery. b) Minimally invasive spinal surgery (MISS). c) Psycho-ne...	Ch. R

OCT 2023 · Paper I

10 questions · 100 marks

Q1	a) Blood brain barrier. b) Regulation of cerebral blood flow. c) Intracranial pressure waves	Ch. B
Q2	a) Growing fracture of skull. b) Differential diagnosis of scalp swelling in a 3 year child. c) Aqueduct...	Ch. C
Q3	a) Third ventricular colloid cyst histology. b) Surgical approaches to colloid cyst. c) Differential dia...	Ch. H
Q4	a) Draw neat labelled diagram of floor of th fourth Ventricle. b) Internuclear ophthalmoplegia. c) Dr...	Ch. A
Q5	a) Enumerate lesions of pineal region. b) Symptoms and signs of posterior third ventricular lesion. c)...	Ch. H
Q6	a) Differential diagnosis of unilateral proptosis. b) Pulsatile proptosis. c) Superior orbital fissure syn...	Ch. F
Q7	a) Recent WHO classification of gliomas. b) Management principles of recurrent glioma	Ch. E
Q8	a) Jefferson's fracture. b) Odontoid fractures. c) Cervical traction	Ch. D
Q9	a) Empty sella syndrome. b) Field defects resulting from perichiasmatic lesions. c) J shaped sella causes	Ch. G
Q10	a) Hyponatremia in neurosurgical patients. b) Use of barbiturates in Neurosurgery. c) Diphenyl hydra...	Ch. T

OCT 2023 · Paper II

10 questions · 100 marks

Q1	a) Large head - differential diagnosis. b) Classification of encephaloceles. c) Apert's syndrome	Ch. L
Q2	a) Differential diagnosis of young stroke. b) Various stages of Moya-Moya disease. c) Indirect Revas...	Ch. K
Q3	a) Draw neat labelled diagrams of safe entry zones of brainstem. b) Brain stem auditory evoked poten...	Ch. A
Q4	a) Posterior fossa stroke - neurosurgical treatment options. b) Management of intraoperative aneurys...	Ch. K
Q5	a) Surgical indications in diffuse idiopathic skeletal hyperostosis. b) Skeletal manifestations of fluoro...	Ch. N
Q6	a) Define instability of spine. b) Metals used in spinal implants. c) Contributions of Ralph B Cloward...	Ch. N
Q7	a) Neurosurgical complications of congenital cyanotic heart disease. b) Diagnosis and management o...	Ch. O
Q8	a) Clinical features of Erb's palsy. b) Cubital tunnel syndrome. c) Nerve supply of muscles of first in...	Ch. Q
Q9	a) Clinical manifestations of spinal AVMs. b) Spinal AVMs classification. c) Spinal arachnoiditis - r...	Ch. K
Q10	a) Spinal intramedullary tumors. b) Draw a neat labelled diagram showing safe entry zones of spinal ...	Ch. N

OCT 2023 · Paper III

10 questions · 100 marks

- Q1 a) HIV and brain & spinal cord. b) CJD diagnosis and Neurosurgery. c) Hydatid cyst of CNS : Differ... Ch. O
- Q2 a) Mycotic aneurysm. b) Neurocysticercosis. c) Neurosarcoidosis Ch. O
- Q3 a) Definition of intractable epilepsy. b) Symptomatic zone in epilepsy. c) Side effects of Levetiraceta... Ch. P
- Q4 a) Salient features of Idiopathic Parkinson Disease and differential diagnosis. b) Targets for deep brai... Ch. P
- Q5 a) Ultrasonic surgical aspirator: principles and application. b) Intraoperative ultrasound in neurosurge... Ch. R
- Q6 a) Phantom limb pain. b) DREZ and its importance on Neurosurgery. c) Cordotomy Ch. P
- Q7 a) Magnetoencephalogram. b) Sterio EEG advantages. c) Callosotomy indications and its side effects Ch. P
- Q8 a) IHC markers for glial tumors. b) Liquid biopsy. c) Robotics in neurosurgery Ch. E
- Q9 a) Mimics of braindeath. b) Harvard Brain Death Committee. c) Punishments prescribed in THOTA f... Ch. U
- Q10 a) Features of latest High end Neurosurgical microscope. b) Exoscope. c) Disinfection of endoscope Ch. R

MAY 2024 · Paper I

10 questions · 100 marks

- Q1 a) Draw a neat diagram of axial section of spinal cord at cervicomedullary junction and demonstrate i... Ch. A
- Q2 Draw a neat diagram of floor of 4th Ventricle and discuss following: a) Demonstrate various Cranial... Ch. A
- Q3 a) Draw diagram of Orbit (posterior aspect) showing various foramen and boundaries. b) Structures ... Ch. A
- Q4 a) Internuclear ophthalmoplegia. b) Draw axial section of midbrain at level of tectum. c) Clinical fea... Ch. A
- Q5 a) Enumerate Basal cisterns. b) Draw sagittal section showing various basal cisterns. c) Grading of ci... Ch. A
- Q6 a) Embryology of spinal dysraphism. b) Limited dorsal myeloschisis. c) Timing and dosage of folic ... Ch. M
- Q7 a) Draw a diagram of blood brain barrier. b) Monroe Kellie doctrine. c) Lund hypothesis for cerebral p... Ch. B
- Q8 a) Cerebral autoregulation b) Pathophysiology of cerebral edema Ch. B
- Q9 a) False localizing signs in neurosurgery. b) Enumerate clinical conditions causing false localizing signs Ch. A
- Q10 a) Newer antiepileptic drugs. b) BTF 2016 guidelines on timing and indications of antiepileptic drugs... Ch. T

MAY 2024 · Paper II

10 questions · 100 marks

- Q1 a) Diagnostic criterion of Brain death as per THOTA Act (India). b) Prerequisites and steps of apnea ... Ch. U
- Q2 a) Components of Prothrombin citrate complex and treatment dosages for spontaneous intracranial b... Ch. C
- Q3 a) Arrested hydrocephalus. b) Management of obstructive hydrocephalus. c) Slit ventricle syndrome Ch. I
- Q4 a) Complications of sitting position in Neurosurgery. b) Clinical conditions wherein sitting position i... Ch. T
- Q5 a) Glasgow coma score. b) Pitfalls of Glasgow coma score. c) Adelaide coma score for Children Ch. C
- Q6 CSF rhinorrhea: a) Management of Spontaneous CSF rhinorrhea. b) Management of Post traumatic... Ch. I
- Q7 a) Genetic syndromes and brain tumors. b) Local and intratumoral therapies in high grade gliomas Ch. E
- Q8 Cavernous sinus: a) Draw a section of cavernous sinus showing cranial nerve and vascular structures... Ch. K
- Q9 a) Indications for Skull base approaches in Neurosurgery. b) Kawase's approach with diagrammatic ... Ch. F
- Q10 a) Colpocephaly. b) Holoprosencephaly. c) Syndromic craniosynostosis: various types and management Ch. L

MAY 2024 · Paper III

10 questions · 100 marks

- Q1 a) Deep Brain Stimulation in Epilepsy. b) Lesioning in neurosurgery Ch. P
- Q2 a) Focal cortical dysplasia classifications. b) Diagnostic work up of a suspected case of FCD. c) Tre... Ch. P
- Q3 a) Newer Recommendations of Brain Trauma Foundation in traumatic brain injury management in a... Ch. C
- Q4 a) Pathophysiology of Parkinson's disease. b) Ablative procedures for Parkinson's disease. c) Compl... Ch. P
- Q5 a) Principle of Radiosurgery in arteriovenous malformations. b) Cyberknife in Neurosurgery. c) Prot... Ch. S
- Q6 a) Standard Temporal lobectomy. b) Selective amygdalohippocampectomy. c) Surgery for non-temp... Ch. P
- Q7 Clinical diagnosis in a patient with the following images: a) Radiological diagnosis and expected clin... Ch. L
- Q8 a) Management of an 85 year old male patient with severe traumatic brain injury presenting with GC... Ch. C
- Q9 a) Etiopathogenesis of mycotic cerebral aneurysms. b) Management of mycotic aneurysms Ch. J
- Q10 Please see the imaging study carried out in a 15-year-old female patient presenting to the emergency ... Ch. K

JAN 2025 · Paper I

10 questions · 100 marks

- Q1 a) Graham Teasdale & Penfield contribution to neurosurgery. b) Cerebral concussion Ch. C
- Q2 a) Lucid interval. b) Management of extradural hematoma Ch. C
- Q3 a) Whiplash injury. b) Classification of odontoid fractures Ch. D
- Q4 a) Draw a neat labelled diagram of circle of Willis. b) Course & branches of anterior cerebral artery Ch. A
- Q5 a) Vein of Galen. b) Internal cerebral vein Ch. A
- Q6 a) Hardy's classification of pituitary tumors. b) Diabetes insipidus Ch. G



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Solved Question Bank · 2020 – 2026

Every question set in the DrNB / DNB Neurosurgery exit theory examination from June 2020 to April 2026 — 35 papers and 350 questions — answered in full, arranged topic-wise across 21 chapters, with labelled diagrams and a page citation to Youmans and Winn Neurological Surgery, 8th Edition for every factual point.

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