BONUS STUDY PACKAGE

Printable Flashcards

Clinical Textbook of Neuroradiology
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300 cards, ten for each chapter, built from the Key Concepts that close every chapter. Questions print on the odd pages and answers on the even pages, eight cards to a sheet.

To make cards: print double-sided, flipping on the long edge, at 100% scale (turn off "fit to page"). Cut along the gray lines. Each answer lands on the back of its own question.

To use as a list instead: print single-sided and read each question page with the answer page that follows it; card numbers match on both pages.

F1.1 · Chapter 1 · Question

Which three CT windows are reviewed on every head CT?

F1.2 · Chapter 1 · Question

What was the median effective dose for a routine head CT, and how much did dose vary?

F1.3 · Chapter 1 · Question

How early did diffusion-weighted hyperintensity appear in experimental ischemia compared with T2 change?

F1.4 · Chapter 1 · Question

How is true restricted diffusion separated from T2 shine-through?

F1.5 · Chapter 1 · Question

How does susceptibility-weighted imaging work, and what does blooming do?

F1.6 · Chapter 1 · Question

What is the recommended default arterial spin labeling implementation?

F1.7 · Chapter 1 · Question

When is saline prophylaxis indicated before iodinated contrast?

F1.8 · Chapter 1 · Question

What happened after 4931 group II gadolinium administrations at eGFR below 30?

F1.2 · Chapter 1 · Answer

Median effective dose for a routine head CT was 2 millisieverts in a four-institution study, with a mean 13-fold variation within each study type.

F1.1 · Chapter 1 · Answer

A brain window near 40/80 Hounsfield units, a wider subdural window, and a bone window are all reviewed on every head CT.

F1.4 · Chapter 1 · Answer

True restricted diffusion is dark on the apparent diffusion coefficient map; T2 shine-through is not.

F1.3 · Chapter 1 · Answer

Diffusion-weighted hyperintensity in ischemic cat brain appeared at 45 minutes, hours before T2-weighted change.

F1.6 · Chapter 1 · Answer

Default arterial spin labeling: pseudo-continuous labeling, background suppression, segmented three-dimensional readout.

F1.5 · Chapter 1 · Answer

Susceptibility-weighted imaging combines a phase mask with the magnitude image, and blooming makes blood products look larger than they are.

F1.8 · Chapter 1 · Answer

No nephrogenic systemic fibrosis followed 4931 group II gadolinium administrations at eGFR below 30.

F1.7 · Chapter 1 · Answer

Saline prophylaxis for iodinated contrast is indicated for acute kidney injury or eGFR below 30 mL/min/1.73 m2 in patients not on dialysis.

F1.9 · Chapter 1 · Question

What does dentate nucleus T1 hyperintensity after gadolinium track?

F1.10 · Chapter 1 · Question

How are implants categorized for MRI scanning?

F2.1 · Chapter 2 · Question

Which landmarks locate the central sulcus?

F2.2 · Chapter 2 · Question

Which structures lose definition first in a middle cerebral artery infarct on CT?

F2.3 · Chapter 2 · Question

What does ventricular enlargement out of proportion to the sulci suggest?

F2.4 · Chapter 2 · Question

How often was the circle of Willis entirely complete on time-of-flight MRA?

F2.5 · Chapter 2 · Question

Why does a fetal posterior cerebral artery matter in carotid occlusion?

F2.6 · Chapter 2 · Question

How do perivascular spaces behave on MRI, and where do they cluster?

F1.10 · Chapter 1 · Answer

Implants are scanned by their labeling: MR safe, MR conditional under stated conditions, or MR unsafe.

F1.9 · Chapter 1 · Answer

Dentate nucleus T1 hyperintensity tracks the number of prior gadolinium doses, and tissue gadolinium is dose-dependent even with normal renal function.

F2.2 · Chapter 2 · Answer

The insular ribbon and lentiform nucleus are the first structures to lose definition in a middle cerebral artery infarct on CT.

F2.1 · Chapter 2 · Answer

The hand knob of the precentral gyrus and the right-angle junction of the superior frontal and precentral sulci locate the central sulcus.

F2.4 · Chapter 2 · Answer

An entirely complete circle of Willis was present in 42% of volunteers on time-of-flight MR angiography.

F2.3 · Chapter 2 · Answer

Ventricles enlarged out of proportion to sulci suggest hydrocephalus rather than volume loss.

F2.6 · Chapter 2 · Answer

Perivascular spaces follow cerebrospinal fluid on every sequence and cluster in the basal ganglia, high convexities, and midbrain.

F2.5 · Chapter 2 · Answer

A fetal posterior cerebral artery lets an internal carotid occlusion infarct the occipital lobe.

F2.7 · Chapter 2 · Question

What incidental findings were reported in the Rotterdam Study?

F2.8 · Chapter 2 · Question

How common are pineal cysts of 5 mm or more in adults, and how do they behave?

F2.9 · Chapter 2 · Question

What is the order of the head CT search pattern?

F2.10 · Chapter 2 · Question

Where in the report is the requisition's question answered?

F3.1 · Chapter 3 · Question

What is the sequence of review on noncontrast CT for acute stroke?

F3.2 · Chapter 3 · Question

How is ASPECTS calculated?

F3.3 · Chapter 3 · Question

What did baseline ASPECTS predict, and how reliable was it?

F3.4 · Chapter 3 · Question

What number needed to treat did pooled thrombectomy trials show?

F2.8 · Chapter 2 · Answer

Pineal cysts of 5 mm or more were found in 1.0% of adults and mostly stayed stable or shrank.

F2.7 · Chapter 2 · Answer

In the Rotterdam Study, incidental asymptomatic infarcts were found in 7.2%, aneurysms in 1.8%, and benign tumors in 1.6%.

F2.10 · Chapter 2 · Answer

The report answers the requisition's question in its opening lines.

F2.9 · Chapter 2 · Answer

The search pattern runs brain window, subdural window, bone window, then margins of the field of view.

F3.2 · Chapter 3 · Answer

ASPECTS subtracts one point from 10 for each of ten middle cerebral regions with early ischemic change.

F3.1 · Chapter 3 · Answer

Noncontrast CT first: exclude hemorrhage, then look for insular ribbon loss, lentiform blurring, and a hyperdense vessel.

F3.4 · Chapter 3 · Answer

Pooled thrombectomy trials showed a number needed to treat of 2.6 to reduce disability by one level.

F3.3 · Chapter 3 · Answer

Baseline ASPECTS predicted outcome and symptomatic hemorrhage, with interobserver kappa of 0.71 to 0.89.

F3.5 · Chapter 3 · Question

What were the DAWN functional independence rates and time window?

F3.6 · Chapter 3 · Question

What core and mismatch criteria did DEFUSE 3 use?

F3.7 · Chapter 3 · Question

What does a diffusion lesion without FLAIR change indicate (WAKE-UP)?

F3.8 · Chapter 3 · Question

What did large-core thrombectomy trials show?

F3.9 · Chapter 3 · Question

What did ATTENTION and BAOCHE show for basilar occlusion?

F3.10 · Chapter 3 · Question

What does a stroke CT or CTA report open with?

F4.1 · Chapter 4 · Question

What are the five TOAST stroke subtypes?

F4.2 · Chapter 4 · Question

What do infarcts in several arterial territories suggest?

F3.6 · Chapter 3 · Answer

DEFUSE 3: core under 70 mL and perfusion mismatch ratio of 1.8 or more at 6 to 16 hours.

F3.5 · Chapter 3 · Answer

DAWN: functional independence 49% versus 13% at 6 to 24 hours with clinical-core mismatch selection.

F3.8 · Chapter 3 · Answer

Large-core trials (ASPECTS 3 to 5 or large core volumes) showed benefit from thrombectomy with more hemorrhage.

F3.7 · Chapter 3 · Answer

WAKE-UP: a diffusion lesion without FLAIR change indicates onset within about 4.5 hours.

F3.10 · Chapter 3 · Answer

The report opens with the occluded segment, the ASPECTS, perfusion volumes, and acquisition times.

F3.9 · Chapter 3 · Answer

Basilar occlusion thrombectomy improved function and reduced mortality in ATTENTION and BAOCHE.

F4.2 · Chapter 4 · Answer

Multiple infarcts in several arterial territories suggest a central embolic source.

F4.1 · Chapter 4 · Answer

TOAST subtypes: large-artery atherosclerosis, cardioembolism, small-vessel occlusion, other determined cause, undetermined cause.

F4.3 · Chapter 4 · Question

What do internal border zone infarcts suggest?

F4.4 · Chapter 4 · Question

Which lesions does STRIVE define?

F4.5 · Chapter 4 · Question

How is a lacune distinguished from a perivascular space?

F4.6 · Chapter 4 · Question

What were the NASCET two-year stroke risks for 70% to 99% stenosis?

F4.7 · Chapter 4 · Question

How is carotid near-occlusion recognized?

F4.8 · Chapter 4 · Question

Why does intraplaque hemorrhage matter?

F4.9 · Chapter 4 · Question

How is embolic stroke of undetermined source defined?

F4.10 · Chapter 4 · Question

Which conditions mimic stroke, and which strokes are often chameleons?

F4.4 · Chapter 4 · Answer

STRIVE defines recent small subcortical infarcts, lacunes, white matter hyperintensities, perivascular spaces, microbleeds, and atrophy.

F4.3 · Chapter 4 · Answer

Infarcts along the internal border zone suggest hypoperfusion distal to a severe stenosis.

F4.6 · Chapter 4 · Answer

NASCET: two-year ipsilateral stroke risk 26% with medical care versus 9% with surgery for 70% to 99% stenosis.

F4.5 · Chapter 4 · Answer

A lacune measures 3 to 15 mm and often has a FLAIR rim; a perivascular space does not.

F4.8 · Chapter 4 · Answer

Intraplaque hemorrhage is a vulnerable plaque feature that predicts stroke independent of stenosis.

F4.7 · Chapter 4 · Answer

Near-occlusion is recognized by a collapsed distal internal carotid artery, not by a percentage.

F4.10 · Chapter 4 · Answer

Seizure, hypoglycemia, and migraine mimic stroke; posterior circulation strokes often present as chameleons.

F4.9 · Chapter 4 · Answer

Embolic stroke of undetermined source is a nonlacunar infarct without proximal stenosis or a cardioembolic source.

F5.1 · Chapter 5 · Question

How does hematoma appearance evolve from acute to subacute?

F5.2 · Chapter 5 · Question

What method estimates hematoma volume quickly at the bedside?

F5.3 · Chapter 5 · Question

Which ICH score components are read from CT?

F5.4 · Chapter 5 · Question

What are the sensitivity and specificity of the spot sign for expansion?

F5.5 · Chapter 5 · Question

Which noncontrast CT signs predict hematoma expansion?

F5.6 · Chapter 5 · Question

How often was a macrovascular cause found in adults aged 18 to 70 with ICH?

F5.7 · Chapter 5 · Question

What defines probable CAA under Boston 2.0?

F5.8 · Chapter 5 · Question

Which white matter features count in Boston 2.0?

F5.2 · Chapter 5 · Answer

ABC/2 estimates hematoma volume in under a minute with excellent reliability.

F5.1 · Chapter 5 · Answer

Acute hematoma: hyperdense on CT, centrally hypointense on T2; subacute: T1 hyperintensity from the periphery inward.

F5.4 · Chapter 5 · Answer

Spot sign: 91% sensitivity and 89% specificity for hematoma expansion.

F5.3 · Chapter 5 · Answer

ICH score components read from CT: infratentorial origin, volume of 30 cm3 or more, intraventricular hemorrhage.

F5.6 · Chapter 5 · Answer

A macrovascular cause was found in 23% of adults aged 18 to 70 years; early CT angiography found 17%.

F5.5 · Chapter 5 · Answer

Swirl, blend, black hole, hypodensity, irregular shape, and island signs predict expansion and poor outcome.

F5.8 · Chapter 5 · Answer

Boston 2.0 white matter features: severe centrum semiovale perivascular spaces or multispot white matter hyperintensities.

F5.7 · Chapter 5 · Answer

Boston 2.0 probable CAA: two strictly lobar hemorrhagic lesions, or one plus a white matter feature.

F5.9 · Chapter 5 · Question

Which technical factors affect microbleed counts?

F5.10 · Chapter 5 · Question

What do lobar versus deep microbleeds suggest?

F6.1 · Chapter 6 · Question

How sensitive was CT for subarachnoid hemorrhage within six hours of onset and overall?

F6.2 · Chapter 6 · Question

What are the sensitivity and specificity of the Ottawa SAH Rule?

F6.3 · Chapter 6 · Question

What Fisher CT finding predicted severe vasospasm?

F6.4 · Chapter 6 · Question

What does modified Fisher grade 4 combine, and what does it predict?

F6.5 · Chapter 6 · Question

When does vasospasm peak after subarachnoid hemorrhage?

F6.6 · Chapter 6 · Question

What were the ISAT 1-year outcomes for coiling and clipping?

F5.10 · Chapter 5 · Answer

Lobar microbleeds suggest amyloid angiopathy; deep microbleeds suggest hypertensive arteriopathy.

F5.9 · Chapter 5 · Answer

Microbleed counts depend on sequence, resolution, field strength, and postprocessing.

F6.2 · Chapter 6 · Answer

Ottawa SAH Rule: 100% sensitivity and 15.3% specificity in alert patients with headache peaking within 1 hour.

F6.1 · Chapter 6 · Answer

CT within six hours of headache onset identified all subarachnoid hemorrhages in a large cohort; overall sensitivity was 92.9%.

F6.4 · Chapter 6 · Answer

Modified Fisher grade 4 combines thick cisternal blood with intraventricular hemorrhage and carries the highest vasospasm risk.

F6.3 · Chapter 6 · Answer

Fisher: thick cisternal clots or layers of 1 mm or more predicted severe vasospasm in 23 of 24 cases.

F6.6 · Chapter 6 · Answer

ISAT: dependency or death at 1 year 23.7% with coiling versus 30.6% with clipping.

F6.5 · Chapter 6 · Answer

Vasospasm peaks between days 4 and 14 after hemorrhage.

F6.7 · Chapter 6 · Question

What is the prevalence of unruptured aneurysms in adults without comorbidity?

F6.8 · Chapter 6 · Question

What factors make up the PHASES score?

F6.9 · Chapter 6 · Question

What is the course of perimesencephalic hemorrhage with negative angiography?

F6.10 · Chapter 6 · Question

What does convexity subarachnoid hemorrhage suggest by age?

F7.1 · Chapter 7 · Question

What is an arteriovenous malformation, and how does it look on T2?

F7.2 · Chapter 7 · Question

Which features determine the surgical grade of an AVM?

F7.3 · Chapter 7 · Question

What are the Borden dural fistula types?

F7.4 · Chapter 7 · Question

What hemorrhage rates did Cognard report for type III and IV fistulas?

F6.8 · Chapter 6 · Answer

PHASES: population, hypertension, age, size, earlier hemorrhage, and site predict 5-year rupture risk.

F6.7 · Chapter 6 · Answer

Unruptured aneurysm prevalence is about 3.2% in adults without comorbidity.

F6.10 · Chapter 6 · Answer

Convexity subarachnoid hemorrhage suggests reversible vasoconstriction in the young and amyloid angiopathy in the old.

F6.9 · Chapter 6 · Answer

Perimesencephalic hemorrhage with negative angiography has a benign course.

F7.2 · Chapter 7 · Answer

Surgical grade uses size, venous drainage, and eloquence of adjacent brain.

F7.1 · Chapter 7 · Answer

An arteriovenous malformation is a nidus shunting arteries to veins without capillaries; flow voids form a honeycomb on T2.

F7.4 · Chapter 7 · Answer

Cognard: hemorrhage in 40% of type III and 65% of type IV fistulas.

F7.3 · Chapter 7 · Answer

Borden types: I sinus or meningeal drainage, II with retrograde cortical drainage, III cortical drainage only.

F7.5 · Chapter 7 · Question

What is the dangerous feature of a dural arteriovenous fistula?

F7.6 · Chapter 7 · Question

What are the MRI and angiographic features of a cavernous malformation?

F7.7 · Chapter 7 · Question

When is cavernous malformation hemorrhage risk highest?

F7.8 · Chapter 7 · Question

Why are developmental venous anomalies not resected?

F7.9 · Chapter 7 · Question

What is the annual hemorrhage rate of unruptured AVMs?

F7.10 · Chapter 7 · Question

What were the ARUBA outcomes?

F8.1 · Chapter 8 · Question

What are the imaging signs of cervical artery dissection?

F8.2 · Chapter 8 · Question

What did CADISS show about recurrence and diagnostic confirmation?

F7.6 · Chapter 7 · Answer

Cavernous malformations have a hemosiderin rim, mixed central signal, and normal angiography.

F7.5 · Chapter 7 · Answer

Cortical venous drainage is the dangerous feature of a dural fistula.

F7.8 · Chapter 7 · Answer

Developmental venous anomalies drain normal brain and are not resected.

F7.7 · Chapter 7 · Answer

Five-year hemorrhage risk from a cavernous malformation is highest after presentation with hemorrhage or a deficit and in the brainstem.

F7.10 · Chapter 7 · Answer

ARUBA: death or stroke in 10.1% with medical management versus 30.7% with intervention.

F7.9 · Chapter 7 · Answer

Annual hemorrhage rate of unruptured arteriovenous malformations is about 2.2%.

F8.2 · Chapter 8 · Answer

CADISS: stroke recurrence 2% overall; central review did not confirm dissection in 52 of 250 patients.

F8.1 · Chapter 8 · Answer

Dissection: flame-shaped or eccentric narrowing beyond the bulb and a bright crescent of mural hematoma on fat-suppressed T1.

F8.3 · Chapter 8 · Question

How does vessel wall MRI separate plaque, vasculitis, and vasoconstriction?

F8.4 · Chapter 8 · Question

How does reversible vasoconstriction behave, and what triggers it?

F8.5 · Chapter 8 · Question

What are the incidence and relapse rate of primary angiitis of the CNS?

F8.6 · Chapter 8 · Question

How do moyamoya disease and moyamoya syndrome differ?

F8.7 · Chapter 8 · Question

What is the ivy sign?

F8.8 · Chapter 8 · Question

What proportion of strokes is caused by cerebral venous thrombosis?

F8.9 · Chapter 8 · Question

Which MR venography technique is more reliable?

F8.10 · Chapter 8 · Question

Which features predict poor outcome after cerebral venous thrombosis?

F8.4 · Chapter 8 · Answer

Reversible vasoconstriction resolves within 3 months; more than half the cases follow delivery or vasoactive drugs.

F8.3 · Chapter 8 · Answer

Vessel wall MRI: eccentric plaque, concentric enhancing vasculitis, nonenhancing vasoconstriction.

F8.6 · Chapter 8 · Answer

Moyamoya disease narrows the intracranial internal carotid arteries; moyamoya syndrome adds an associated condition.

F8.5 · Chapter 8 · Answer

Primary angiitis has an incidence of 2.4 per million person-years and relapses in one fourth.

F8.8 · Chapter 8 · Answer

Cerebral venous thrombosis accounts for 0.5% to 3% of strokes.

F8.7 · Chapter 8 · Answer

The ivy sign is sulcal FLAIR hyperintensity from slow leptomeningeal collateral flow.

F8.10 · Chapter 8 · Answer

Deep venous involvement and hemorrhage predict death or dependence after venous thrombosis.

F8.9 · Chapter 8 · Answer

Contrast-enhanced MR venography is more reliable than time-of-flight venography.

F9.1 · Chapter 9 · Question

How sensitive were the Canadian CT Head Rule high-risk factors?

F9.2 · Chapter 9 · Question

What are the New Orleans criteria?

F9.3 · Chapter 9 · Question

How do epidural and subdural hematomas differ in shape and suture relation?

F9.4 · Chapter 9 · Question

What thickness or shift indicates evacuation of an acute subdural hematoma?

F9.5 · Chapter 9 · Question

How are isodense subdural hematomas detected?

F9.6 · Chapter 9 · Question

Where does diffuse axonal injury favor?

F9.7 · Chapter 9 · Question

How did CT compare with T2-weighted MRI for nonhemorrhagic shear lesions?

F9.8 · Chapter 9 · Question

How did mortality change across Marshall diffuse injury categories?

F9.2 · Chapter 9 · Answer

New Orleans criteria: headache, vomiting, age over 60, intoxication, short-term memory deficit, trauma above the clavicles, seizure.

F9.1 · Chapter 9 · Answer

Canadian CT Head Rule high-risk factors were 100% sensitive for need for neurological intervention.

F9.4 · Chapter 9 · Answer

Evacuate an acute subdural hematoma over 10 mm thick or with more than 5 mm of shift.

F9.3 · Chapter 9 · Answer

Epidural hematomas are biconvex and respect sutures; subdural hematomas are crescentic and cross sutures.

F9.6 · Chapter 9 · Answer

Diffuse axonal injury favors the gray-white junction, corpus callosum splenium, and dorsolateral brainstem.

F9.5 · Chapter 9 · Answer

Isodense subdural hematomas are found by displaced gray-white junction and effaced sulci.

F9.8 · Chapter 9 · Answer

Marshall diffuse injury mortality rose from 10% in category I to more than 50% in category IV.

F9.7 · Chapter 9 · Answer

Nonhemorrhagic shear lesions: CT sensitivity 17.7% versus 93.3% on T2-weighted MRI in one prospective series.

F9.9 · Chapter 9 · Question

Which CT elements carry prognostic value in traumatic brain injury?

F9.10 · Chapter 9 · Question

How are blunt cerebrovascular injuries graded, and what rises with grade?

F10.1 · Chapter 10 · Question

What are the main types of intracranial herniation?

F10.2 · Chapter 10 · Question

What is the earliest sign of uncal herniation?

F10.3 · Chapter 10 · Question

What is the Kernohan notch phenomenon?

F10.4 · Chapter 10 · Question

Where do Duret hemorrhages occur, and what causes them?

F10.5 · Chapter 10 · Question

Which structures does profound adult hypoxic-ischemic injury affect?

F10.6 · Chapter 10 · Question

How does diffusion restriction evolve after hypoxic injury?

F9.10 · Chapter 9 · Answer

Blunt cerebrovascular injury grades run from intimal injury to transection, and stroke risk rises with grade.

F9.9 · Chapter 9 · Answer

Prognostic CT elements: basal cisterns, shift, traumatic subarachnoid or intraventricular blood, mass lesions.

F10.2 · Chapter 10 · Answer

Early uncal herniation effaces the ipsilateral suprasellar and ambient cisterns.

F10.1 · Chapter 10 · Answer

Intracranial herniation types: subfalcine, transtentorial (ascending or descending), and tonsillar.

F10.4 · Chapter 10 · Answer

Duret hemorrhages in the pons and midbrain follow central descending herniation.

F10.3 · Chapter 10 · Answer

Kernohan notch compression of the opposite cerebral peduncle causes weakness on the side of the mass.

F10.6 · Chapter 10 · Answer

Diffusion restriction after hypoxic injury peaks in days and pseudonormalizes after about a week.

F10.5 · Chapter 10 · Answer

Profound adult hypoxic-ischemic injury affects deep gray nuclei, cortex, hippocampi, and cerebellum.

F10.7 · Chapter 10 · Question

What is the reversal sign?

F10.8 · Chapter 10 · Question

How is imaging used after cardiac arrest?

F10.9 · Chapter 10 · Question

What does delayed post-hypoxic leukoencephalopathy spare?

F10.10 · Chapter 10 · Question

What role do ancillary flow studies play in death by neurologic criteria?

F11.1 · Chapter 11 · Question

What are the three adult-type diffuse glioma types?

F11.2 · Chapter 11 · Question

Which molecular change makes an IDH-mutant astrocytoma grade 4?

F11.3 · Chapter 11 · Question

Which molecular features make an IDH-wildtype diffuse astrocytic tumor a glioblastoma?

F11.4 · Chapter 11 · Question

What did the T2-FLAIR mismatch sign predict in its derivation set?

F10.8 · Chapter 10 · Answer

Imaging after cardiac arrest is interpreted within a multimodal prognostic assessment.

F10.7 · Chapter 10 · Answer

The reversal sign is diffuse hemispheric hypodensity with relatively dense thalami, brainstem, and cerebellum.

F10.10 · Chapter 10 · Answer

Death by neurologic criteria is clinical; ancillary flow studies support it when the examination is incomplete.

F10.9 · Chapter 10 · Answer

Delayed post-hypoxic leukoencephalopathy spares the subcortical U-fibers.

F11.2 · Chapter 11 · Answer

CDKN2A/B homozygous deletion makes an IDH-mutant astrocytoma grade 4.

F11.1 · Chapter 11 · Answer

Adult diffuse gliomas: astrocytoma IDH-mutant; oligodendroglioma IDH-mutant and 1p/19q-codeleted; glioblastoma IDH-wildtype.

F11.4 · Chapter 11 · Answer

T2-FLAIR mismatch: all 15 positive cases were IDH-mutant, non-codeleted in the derivation set.

F11.3 · Chapter 11 · Answer

Molecular features (TERT, EGFR, +7/-10) make an IDH-wildtype diffuse astrocytic tumor a glioblastoma.

F11.5 · Chapter 11 · Question

What are the specificity and sensitivity of the T2-FLAIR mismatch sign?

F11.6 · Chapter 11 · Question

What share of malignant primary brain and CNS tumors is glioblastoma?

F11.7 · Chapter 11 · Question

Which T1 sequences does the standardized Brain Tumor Imaging Protocol use?

F11.8 · Chapter 11 · Question

How did radiotherapy with temozolomide change two-year glioblastoma survival?

F11.9 · Chapter 11 · Question

What did INDIGO show for vorasidenib?

F11.10 · Chapter 11 · Question

Which prior studies should glioma surveillance scans be compared with?

F12.1 · Chapter 12 · Question

What are the signs that a mass is extra-axial?

F12.2 · Chapter 12 · Question

How common is meningioma among primary brain and CNS tumors?

F11.6 · Chapter 11 · Answer

Glioblastoma made up 50.9% of malignant primary brain and CNS tumors in the US registry.

F11.5 · Chapter 11 · Answer

The mismatch sign has near-perfect specificity but low sensitivity; criteria must be applied strictly.

F11.8 · Chapter 11 · Answer

Radiotherapy with temozolomide raised two-year survival from 10.4% to 26.5% in glioblastoma.

F11.7 · Chapter 11 · Answer

The standardized Brain Tumor Imaging Protocol uses matched precontrast and postcontrast 3D T1 images.

F11.10 · Chapter 11 · Answer

Compare surveillance scans with baseline and nadir, not only the previous study.

F11.9 · Chapter 11 · Answer

INDIGO: vorasidenib extended median progression-free survival from 11.1 to 27.7 months.

F12.2 · Chapter 12 · Answer

Meningioma is the most common intracranial tumor and made up 40.8% of primary brain and CNS tumors in the US registry.

F12.1 · Chapter 12 · Answer

Extra-axial signs: CSF cleft, displaced vessels, buckled gray matter, broad dural base.

F12.3 · Chapter 12 · Question

Is a dural tail specific for meningioma?

F12.4 · Chapter 12 · Question

Which molecular changes define CNS WHO grade 3 meningioma?

F12.5 · Chapter 12 · Question

How did DOTATATE PET compare with MRI for detecting meningioma tissue?

F12.6 · Chapter 12 · Question

How often did incidental meningiomas become symptomatic, and which features were associated?

F12.7 · Chapter 12 · Question

What do Simpson grades describe?

F12.8 · Chapter 12 · Question

What are the imaging features of solitary fibrous tumor?

F12.9 · Chapter 12 · Question

How are epidermoid and arachnoid cysts separated on MRI?

F12.10 · Chapter 12 · Question

What does a ruptured dermoid cyst look like?

F12.4 · Chapter 12 · Answer

TERT promoter mutation and CDKN2A/B deletion define CNS WHO grade 3 meningioma.

F12.3 · Chapter 12 · Answer

A dural tail is common in meningioma but not specific.

F12.6 · Chapter 12 · Answer

Incidental meningiomas: symptoms developed in 8.1% under monitoring; edema and a diameter of 3 cm or more were associated.

F12.5 · Chapter 12 · Answer

DOTATATE PET detected meningioma tissue with 90% sensitivity versus 79% for MRI.

F12.8 · Chapter 12 · Answer

Solitary fibrous tumor: narrow base, flow voids, bone erosion without hyperostosis.

F12.7 · Chapter 12 · Answer

Simpson grades describe extent of resection and predict recurrence.

F12.10 · Chapter 12 · Answer

Ruptured dermoid cysts scatter T1-hyperintense fat droplets through the subarachnoid space.

F12.9 · Chapter 12 · Answer

Epidermoid cysts restrict diffusion and fail to suppress on FLAIR; arachnoid cysts do neither.

F13.1 · Chapter 13 · Question

How common are CNS metastases among adult malignant brain tumors?

F13.2 · Chapter 13 · Question

Which primary cancers account for most brain metastases?

F13.3 · Chapter 13 · Question

Where do brain metastases lodge, and how is their edema described?

F13.4 · Chapter 13 · Question

Which imaging technique detects more small metastases?

F13.5 · Chapter 13 · Question

What is recommended for one to four unresected brain metastases?

F13.6 · Chapter 13 · Question

How did adding whole-brain radiotherapy to radiosurgery affect cognition at 3 months?

F13.7 · Chapter 13 · Question

What are the imaging features of primary CNS lymphoma?

F13.8 · Chapter 13 · Question

What should be done with corticosteroids when lymphoma is suspected?

F13.2 · Chapter 13 · Answer

Lung, breast, and melanoma account for 67% to 80% of brain metastases.

F13.1 · Chapter 13 · Answer

CNS metastases are the most common malignant brain tumors in adults; incidence is reported at 9% to 17%.

F13.4 · Chapter 13 · Answer

Thin-section 3D postcontrast T1 imaging detects more small metastases than thick 2D slices.

F13.3 · Chapter 13 · Answer

Metastases lodge at the gray-white junction with edema out of proportion to size.

F13.6 · Chapter 13 · Answer

Cognitive deterioration at 3 months: 63.5% with radiosurgery alone versus 91.7% with added whole-brain radiotherapy.

F13.5 · Chapter 13 · Answer

Radiosurgery alone is recommended for one to four unresected metastases in many patients.

F13.8 · Chapter 13 · Answer

Withhold corticosteroids before biopsy when lymphoma is suspected, if clinically safe.

F13.7 · Chapter 13 · Answer

Primary CNS lymphoma: periventricular, homogeneous enhancement, restricted diffusion, low perfusion.

F13.9 · Chapter 13 · Question

Which imaging detects leptomeningeal metastasis?

F13.10 · Chapter 13 · Question

How do abscess and tumefactive demyelination rims differ?

F14.1 · Chapter 14 · Question

How does RANO measure enhancing disease?

F14.2 · Chapter 14 · Question

Which MRI is the baseline in RANO 2.0 for newly diagnosed glioma?

F14.3 · Chapter 14 · Question

How common is pseudoprogression after glioblastoma chemoradiotherapy, and when?

F14.4 · Chapter 14 · Question

What is required to call progression within 12 weeks of radiotherapy?

F14.5 · Chapter 14 · Question

How do antiangiogenic agents affect enhancement, and what should be followed?

F14.6 · Chapter 14 · Question

What are the timing and appearance of radiation necrosis?

F13.10 · Chapter 13 · Answer

Abscess: smooth thin rim and central restricted diffusion; tumefactive demyelination: incomplete rim.

F13.9 · Chapter 13 · Answer

Postcontrast FLAIR and whole-spine MRI detect leptomeningeal metastasis.

F14.2 · Chapter 14 · Answer

RANO 2.0 uses the postradiotherapy MRI, not the postoperative MRI, as baseline in newly diagnosed glioma.

F14.1 · Chapter 14 · Answer

RANO uses the sum of perpendicular diameter products of measurable enhancing lesions, at least 10 mm in both dimensions.

F14.4 · Chapter 14 · Answer

Progression within 12 weeks of radiotherapy requires confirmation by repeat MRI or histopathology.

F14.3 · Chapter 14 · Answer

Pseudoprogression affects 20% to 30% of glioblastomas after chemoradiotherapy, mostly within 12 weeks.

F14.6 · Chapter 14 · Answer

Radiation necrosis: months to years after treatment, soap-bubble enhancement, waxing and waning course.

F14.5 · Chapter 14 · Answer

Antiangiogenic agents decrease enhancement by reducing permeability; follow FLAIR for nonenhancing recurrence.

F14.7 · Chapter 14 · Question

How accurate is perfusion imaging for recurrence versus treatment effect?

F14.8 · Chapter 14 · Question

Which PET tracer class is preferred for glioma assessment?

F14.9 · Chapter 14 · Question

What does iRANO recommend after immunotherapy?

F14.10 · Chapter 14 · Question

Which pituitary complication can checkpoint inhibitors cause?

F15.1 · Chapter 15 · Question

How do microadenomas appear on dynamic contrast imaging?

F15.2 · Chapter 15 · Question

What size separates microadenoma from macroadenoma?

F15.3 · Chapter 15 · Question

What did WHO 2022 rename pituitary adenomas?

F15.4 · Chapter 15 · Question

Which Knosp grades indicate cavernous sinus invasion?

F14.8 · Chapter 14 · Answer

Amino acid PET is superior to FDG PET for glioma assessment.

F14.7 · Chapter 14 · Answer

Pooled perfusion accuracy for recurrence versus treatment effect is about 90% sensitivity and 85% to 88% specificity.

F14.10 · Chapter 14 · Answer

Checkpoint inhibitors can cause hypophysitis with pituitary and stalk enlargement.

F14.9 · Chapter 14 · Answer

iRANO: confirm progression within 6 months of immunotherapy if the patient is clinically stable.

F15.2 · Chapter 15 · Answer

Microadenoma: smaller than 10 mm; macroadenoma: 10 mm or larger.

F15.1 · Chapter 15 · Answer

Dynamic contrast imaging shows microadenomas as hypoenhancing foci within early-enhancing gland.

F15.4 · Chapter 15 · Answer

Knosp grades 3 and 4 indicate cavernous sinus invasion; grades 0 and 1 do not.

F15.3 · Chapter 15 · Answer

WHO 2022 renamed pituitary adenomas pituitary neuroendocrine tumors, classified by lineage.

F15.5 · Chapter 15 · Question

How often are pituitary adenomas found at autopsy?

F15.6 · Chapter 15 · Question

What is the follow-up MRI schedule for pituitary incidentalomas?

F15.7 · Chapter 15 · Question

What does a large sellar mass with mildly raised prolactin usually represent?

F15.8 · Chapter 15 · Question

How do adamantinomatous and papillary craniopharyngiomas differ?

F15.9 · Chapter 15 · Question

What are the features of pituitary apoplexy, and what treatment starts immediately?

F15.10 · Chapter 15 · Question

How do germinomas and pineal parenchymal tumors treat pineal calcification?

F16.1 · Chapter 16 · Question

When should CT precede lumbar puncture in suspected meningitis?

F16.2 · Chapter 16 · Question

Which sequence is sensitive for leptomeningeal enhancement in meningitis?

F15.6 · Chapter 15 · Answer

Incidentaloma follow-up: MRI at 6 months for macro and 1 year for micro lesions.

F15.5 · Chapter 15 · Answer

Pituitary adenomas are found in about 10.6% of autopsies.

F15.8 · Chapter 15 · Answer

Adamantinomatous craniopharyngioma: cystic, calcified, CTNNB1; papillary: solid, BRAF V600E, adults.

F15.7 · Chapter 15 · Answer

Stalk compression raises prolactin modestly; a large mass with mild hyperprolactinemia is usually not a prolactinoma.

F15.10 · Chapter 15 · Answer

Germinoma engulfs pineal calcification; pineal parenchymal tumors disperse it.

F15.9 · Chapter 15 · Answer

Pituitary apoplexy: sudden headache, visual loss, ophthalmoplegia; start glucocorticoids immediately.

F16.2 · Chapter 16 · Answer

Postcontrast FLAIR is sensitive for leptomeningeal enhancement in meningitis.

F16.1 · Chapter 16 · Answer

Do not delay antibiotics for imaging; CT before lumbar puncture only when a mass or raised pressure is suspected.

F16.3 · Chapter 16 · Question

How does diffusion separate subdural empyema from sterile effusion?

F16.4 · Chapter 16 · Question

What does a mature brain abscess look like on MRI?

F16.5 · Chapter 16 · Question

Why does an abscess abutting the ventricle matter?

F16.6 · Chapter 16 · Question

What is the distribution of herpes simplex encephalitis, and when is it treated?

F16.7 · Chapter 16 · Question

What are the imaging features of tuberculous meningitis?

F16.8 · Chapter 16 · Question

What is an absolute criterion for neurocysticercosis?

F16.9 · Chapter 16 · Question

What do PML lesions look like on MRI?

F16.10 · Chapter 16 · Question

What establishes a diagnosis of PML?

F16.4 · Chapter 16 · Answer

Mature abscess: smooth thin rim, thinner medially, T2-hypointense capsule, central restricted diffusion.

F16.3 · Chapter 16 · Answer

Subdural empyema restricts diffusion; sterile effusions do not.

F16.6 · Chapter 16 · Answer

Herpes encephalitis: medial temporal lobe and insula, basal ganglia spared; treat on suspicion.

F16.5 · Chapter 16 · Answer

Abscess abutting the ventricle risks intraventricular rupture and ventriculitis.

F16.8 · Chapter 16 · Answer

A scolex within a cyst is an absolute criterion for neurocysticercosis.

F16.7 · Chapter 16 · Answer

Tuberculous meningitis: basal cistern enhancement, hydrocephalus, and perforator infarcts.

F16.10 · Chapter 16 · Answer

JC virus in CSF plus consistent clinical and imaging findings is diagnostic of PML.

F16.9 · Chapter 16 · Answer

PML: asymmetric subcortical white matter lesions involving U fibers with little mass effect.

F17.1 · Chapter 17 · Question

How is dissemination in space shown in multiple sclerosis?

F17.2 · Chapter 17 · Question

Which location did the 2024 McDonald criteria add?

F17.3 · Chapter 17 · Question

What shape, size, and orientation do typical MS lesions have?

F17.4 · Chapter 17 · Question

What are the length and area limits of typical MS cord lesions?

F17.5 · Chapter 17 · Question

What is the central vein sign, and how often is it seen in MS lesions?

F17.6 · Chapter 17 · Question

What is the significance of four or more paramagnetic rim lesions?

F17.7 · Chapter 17 · Question

What is the core MS sequence, and what measures activity?

F17.8 · Chapter 17 · Question

How is PML surveillance done in high-risk natalizumab patients?

F17.2 · Chapter 17 · Answer

The 2024 McDonald criteria add the optic nerve as a fifth location.

F17.1 · Chapter 17 · Answer

Dissemination in space: typical lesions in at least two of periventricular, juxtacortical or cortical, infratentorial, and spinal cord regions.

F17.4 · Chapter 17 · Answer

MS cord lesions span less than two segments and less than half the cord area.

F17.3 · Chapter 17 · Answer

Typical lesions are ovoid and at least 3 mm; periventricular lesions are perpendicular to the ventricles.

F17.6 · Chapter 17 · Answer

Four or more paramagnetic rim lesions are associated with earlier disability.

F17.5 · Chapter 17 · Answer

Central vein sign: thin line or dot in two planes; seen in about 80% of MS lesions.

F17.8 · Chapter 17 · Answer

Natalizumab: surveil for PML with abbreviated FLAIR and diffusion MRI in high-risk patients.

F17.7 · Chapter 17 · Answer

3D FLAIR is the core sequence; new or enlarging T2 lesions measure activity.

F17.9 · Chapter 17 · Question

What did dimethyl fumarate do in radiologically isolated syndrome?

F17.10 · Chapter 17 · Question

Which red flags suggest an MS mimic?

F18.1 · Chapter 18 · Question

What are the core clinical characteristics of NMOSD?

F18.2 · Chapter 18 · Question

What does seronegative NMOSD require?

F18.3 · Chapter 18 · Question

What are the features of longitudinally extensive transverse myelitis?

F18.4 · Chapter 18 · Question

Why recommend aquaporin-4 antibody testing when NMOSD is suspected?

F18.5 · Chapter 18 · Question

What are the imaging features of MOGAD?

F18.6 · Chapter 18 · Question

What does ADEM require, and what are its lesions like?

F17.10 · Chapter 17 · Answer

Red flags for mimics: U-fiber sparing, long cord lesions, central callosal snowballs, absent central veins.

F17.9 · Chapter 17 · Answer

Radiologically isolated syndrome: dimethyl fumarate reduced the risk of a first clinical event.

F18.2 · Chapter 18 · Answer

Seronegative NMOSD needs optic neuritis, myelitis with LETM, or area postrema syndrome.

F18.1 · Chapter 18 · Answer

NMOSD core characteristics: optic neuritis, transverse myelitis, area postrema, brainstem, diencephalic, and cerebral syndromes.

F18.4 · Chapter 18 · Answer

Some MS therapies may worsen NMOSD; recommend aquaporin-4 antibody testing.

F18.3 · Chapter 18 · Answer

LETM spans three or more segments, favors the central cord, and may extend into the medulla.

F18.6 · Chapter 18 · Answer

ADEM requires encephalopathy; lesions are large, bilateral, and of similar age.

F18.5 · Chapter 18 · Answer

MOGAD: bilateral anterior optic neuritis, perineural enhancement, conus lesions, H sign.

F18.7 · Chapter 18 · Question

What MRI finding is required for definite antibody-negative limbic encephalitis?

F18.8 · Chapter 18 · Question

What does MRI usually show in anti-NMDA receptor encephalitis, and what search follows?

F18.9 · Chapter 18 · Question

What does neurosarcoidosis look like, and where should biopsy be taken?

F18.10 · Chapter 18 · Question

What is CLIPPERS?

F19.1 · Chapter 19 · Question

Which patients with cognitive impairment should be imaged, and how?

F19.2 · Chapter 19 · Question

How is the medial temporal atrophy scale scored?

F19.3 · Chapter 19 · Question

What establishes a biological diagnosis of Alzheimer disease?

F19.4 · Chapter 19 · Question

How often did ARIA with edema or effusion occur with lecanemab and donanemab?

F18.8 · Chapter 18 · Answer

Anti-NMDA receptor encephalitis often has a normal MRI; search for ovarian teratoma.

F18.7 · Chapter 18 · Answer

Definite limbic encephalitis without antibodies requires bilateral medial temporal FLAIR signal.

F18.10 · Chapter 18 · Answer

CLIPPERS: curvilinear punctate pontine enhancement responsive to corticosteroids.

F18.9 · Chapter 18 · Answer

Neurosarcoidosis: basal leptomeningeal and hypothalamic enhancement; biopsy nonneural tissue when possible.

F19.2 · Chapter 19 · Answer

Medial temporal atrophy scale: 0 to 4, based on choroid fissure, temporal horn, and hippocampal height.

F19.1 · Chapter 19 · Answer

Image every new cognitive impairment at least once, preferably with MRI, to exclude treatable causes.

F19.4 · Chapter 19 · Answer

Lecanemab: ARIA with edema or effusions in 12.6%; donanemab: 24.0%.

F19.3 · Chapter 19 · Answer

An abnormal Core 1 biomarker, such as amyloid PET or plasma p-tau217, establishes Alzheimer disease.

F19.5 · Chapter 19 · Question

How do ARIA rates with lecanemab vary by APOE ε4 genotype?

F19.6 · Chapter 19 · Question

What are the lecanemab exclusion and MRI monitoring rules?

F19.7 · Chapter 19 · Question

Which atrophy pattern supports probable behavioral variant FTD?

F19.8 · Chapter 19 · Question

What imaging features suggest dementia with Lewy bodies?

F19.9 · Chapter 19 · Question

Which MRI signs suggest progressive supranuclear palsy?

F19.10 · Chapter 19 · Question

Which MRI findings suggest multiple system atrophy?

F20.1 · Chapter 20 · Question

What is the typical imaging appearance of PRES?

F20.2 · Chapter 20 · Question

In which clinical settings does PRES occur?

F19.6 · Chapter 19 · Answer

Exclude patients with more than four microhemorrhages; MRI before the 5th, 7th, and 14th infusions.

F19.5 · Chapter 19 · Answer

ARIA rates with lecanemab: 5.4% in APOE ε4 noncarriers, 10.9% in heterozygotes, 32.6% in homozygotes.

F19.8 · Chapter 19 · Answer

Dementia with Lewy bodies: relatively preserved medial temporal lobes, low DAT uptake, cingulate island sign.

F19.7 · Chapter 19 · Answer

Behavioral variant FTD: frontal and anterior temporal atrophy supports a probable diagnosis.

F19.10 · Chapter 19 · Answer

MSA: putaminal, pontine, and middle cerebellar peduncle atrophy; hot cross bun sign.

F19.9 · Chapter 19 · Answer

PSP: midbrain atrophy with hummingbird and morning glory signs.

F20.2 · Chapter 20 · Answer

PRES settings: blood pressure fluctuation, renal failure, cytotoxic drugs, autoimmune disease, eclampsia, transplantation.

F20.1 · Chapter 20 · Answer

PRES: bilateral subcortical vasogenic edema, mainly parieto-occipital, with increased diffusion.

F20.3 · Chapter 20 · Question

Which atypical PRES features are compatible, and which predicts worse outcome?

F20.4 · Chapter 20 · Question

Where does Wernicke encephalopathy appear, and when is thiamine given?

F20.5 · Chapter 20 · Question

What are the features of osmotic demyelination?

F20.6 · Chapter 20 · Question

How long may imaging lag symptoms in osmotic demyelination?

F20.7 · Chapter 20 · Question

What T1 finding is classic in hepatic encephalopathy, and why?

F20.8 · Chapter 20 · Question

What does acute hyperammonemia look like on MRI?

F20.9 · Chapter 20 · Question

Which structures do carbon monoxide and methanol target?

F20.10 · Chapter 20 · Question

What lesions does metronidazole cause, and how do they evolve?

F20.4 · Chapter 20 · Answer

Wernicke encephalopathy: medial thalami, mammillary bodies, periaqueductal gray; give thiamine on suspicion.

F20.3 · Chapter 20 · Answer

Hemorrhage, enhancement, and restricted diffusion are compatible with PRES; restriction predicts worse outcome.

F20.6 · Chapter 20 · Answer

Imaging in osmotic demyelination may lag symptoms by up to 2 weeks; repeat an early normal study.

F20.5 · Chapter 20 · Answer

Osmotic demyelination: rapid sodium correction, biphasic course, central pontine trident lesion.

F20.8 · Chapter 20 · Answer

Acute hyperammonemia: cortical swelling with restricted diffusion in the insula and cingulate.

F20.7 · Chapter 20 · Answer

Hepatic encephalopathy: T1-hyperintense globus pallidus from manganese deposition.

F20.10 · Chapter 20 · Answer

Metronidazole: symmetric dentate nucleus, dorsal brainstem, and splenial lesions, reversible after stopping the drug.

F20.9 · Chapter 20 · Answer

Carbon monoxide targets the globi pallidi; methanol the putamina.

F21.1 · Chapter 21 · Question

What Evans index indicates ventricular enlargement?

F21.2 · Chapter 21 · Question

Which ventricles dilate in aqueductal stenosis?

F21.3 · Chapter 21 · Question

What are the components of DESH, and how specific and sensitive is it?

F21.4 · Chapter 21 · Question

How does the callosal angle differ in NPH and atrophy?

F21.5 · Chapter 21 · Question

What are the sensitivity and specificity of the CSF tap test?

F21.6 · Chapter 21 · Question

What imaging is urgent in suspected IIH, and are its signs pathognomonic?

F21.7 · Chapter 21 · Question

What does the SEEPS mnemonic stand for in intracranial hypotension?

F21.8 · Chapter 21 · Question

What Bern score indicates a high probability of a spinal leak?

F21.2 · Chapter 21 · Answer

Aqueductal stenosis dilates the lateral and third ventricles with a normal fourth ventricle.

F21.1 · Chapter 21 · Answer

Evans index above 0.3 indicates ventricular enlargement.

F21.4 · Chapter 21 · Answer

A steep callosal angle supports normal pressure hydrocephalus; atrophy widens it.

F21.3 · Chapter 21 · Answer

DESH: ventriculomegaly, dilated Sylvian fissures, tight high-convexity sulci; specific but not sensitive.

F21.6 · Chapter 21 · Answer

IIH: urgent MRI and venography to exclude mass and sinus thrombosis; imaging signs are not pathognomonic.

F21.5 · Chapter 21 · Answer

CSF tap test sensitivity 58% and specificity 75%; nonresponders may still improve with a shunt.

F21.8 · Chapter 21 · Answer

Bern score: 5 or more points indicates high probability of a spinal leak.

F21.7 · Chapter 21 · Answer

SIH brain findings, SEEPS: subdural collections, enhancement, engorgement, pituitary hyperemia, sagging.

F21.9 · Chapter 21 · Question

What is the first-line imaging for spontaneous intracranial hypotension?

F21.10 · Chapter 21 · Question

How are CSF-venous fistulas found?

F22.1 · Chapter 22 · Question

How does the ILAE classify seizures?

F22.2 · Chapter 22 · Question

What is the recurrence risk after a first unprovoked seizure?

F22.3 · Chapter 22 · Question

For which epilepsies is routine structural MRI endorsed?

F22.4 · Chapter 22 · Question

What sequences make up the essential six epilepsy protocol?

F22.5 · Chapter 22 · Question

What are the MRI signs of hippocampal sclerosis?

F22.6 · Chapter 22 · Question

Which ILAE type of hippocampal sclerosis has the best surgical outcome?

F21.10 · Chapter 21 · Answer

CSF-venous fistulas lack an epidural collection; seek them with decubitus CT or digital subtraction myelography.

F21.9 · Chapter 21 · Answer

First-line SIH imaging: contrast-enhanced brain MRI and whole-spine MRI.

F22.2 · Chapter 22 · Answer

Recurrence risk after a first unprovoked seizure is 21% to 45% within 2 years.

F22.1 · Chapter 22 · Answer

ILAE seizure classes: focal, generalized, unknown, unclassified; focal seizures by state of consciousness.

F22.4 · Chapter 22 · Answer

Essential six: 3D T1 at 1 mm isotropic, T2 and FLAIR at 3 mm or less in two hippocampal planes, susceptibility.

F22.3 · Chapter 22 · Answer

Routine structural MRI is endorsed for new-onset focal and generalized epilepsy.

F22.6 · Chapter 22 · Answer

ILAE type 1 hippocampal sclerosis (CA1 and CA4) has the best surgical outcome.

F22.5 · Chapter 22 · Answer

Hippocampal sclerosis: volume loss, T2 hyperintensity, lost internal architecture, enlarged temporal horn.

F22.7 · Chapter 22 · Question

What are the most common lesions in epilepsy surgery series?

F22.8 · Chapter 22 · Question

What are the MRI signs of FCD type II, and which sign indicates type IIb?

F22.9 · Chapter 22 · Question

What does a DNET look like on MRI?

F22.10 · Chapter 22 · Question

What are typical peri-ictal MRI changes?

F23.1 · Chapter 23 · Question

What are the orbital compartments?

F23.2 · Chapter 23 · Question

What are the Chandler stages of orbital infection?

F23.3 · Chapter 23 · Question

What does a subperiosteal abscess look like?

F23.4 · Chapter 23 · Question

Which muscles and parts of muscles does thyroid eye disease affect?

F22.8 · Chapter 22 · Answer

FCD type II: thickening, blurring, FLAIR signal; transmantle sign indicates type IIb.

F22.7 · Chapter 22 · Answer

Surgical series: hippocampal sclerosis 36.4%, tumors 23.6%, cortical malformations 19.8%.

F22.10 · Chapter 22 · Answer

Peri-ictal changes: cortical and pulvinar restricted diffusion, nonterritorial, transient.

F22.9 · Chapter 22 · Answer

DNET: bubbly, T2-bright cortical lesion without enhancement or edema.

F23.2 · Chapter 23 · Answer

Chandler stages: preseptal cellulitis, orbital cellulitis, subperiosteal abscess, orbital abscess, cavernous sinus thrombosis.

F23.1 · Chapter 23 · Answer

Orbital compartments: preseptal, extraconal, intraconal, optic nerve sheath complex, and globe.

F23.4 · Chapter 23 · Answer

Thyroid eye disease: muscle belly enlargement sparing tendons; inferior and medial recti most often.

F23.3 · Chapter 23 · Answer

Subperiosteal abscess: lentiform collection along the lamina papyracea next to ethmoid sinusitis.

F23.5 · Chapter 23 · Question

What does apical crowding in thyroid eye disease indicate?

F23.6 · Chapter 23 · Question

How does orbital myositis differ from thyroid eye disease?

F23.7 · Chapter 23 · Question

How does optic neuritis length and location suggest MS or NMOSD?

F23.8 · Chapter 23 · Question

What did oral prednisone alone do in the Optic Neuritis Treatment Trial?

F23.9 · Chapter 23 · Question

What does a cavernous venous malformation of the orbit look like?

F23.10 · Chapter 23 · Question

How do optic nerve glioma and sheath meningioma differ in shape?

F24.1 · Chapter 24 · Question

When is temporal bone CT used, and when is MRI used?

F24.2 · Chapter 24 · Question

Where does pars flaccida cholesteatoma begin, and what does it erode?

F23.6 · Chapter 23 · Answer

Myositis enlarges a single muscle including its tendon.

F23.5 · Chapter 23 · Answer

Apical crowding in thyroid eye disease indicates risk of compressive optic neuropathy.

F23.8 · Chapter 23 · Answer

Oral prednisone alone increased new optic neuritis episodes in the Optic Neuritis Treatment Trial.

F23.7 · Chapter 23 · Answer

Optic neuritis: short anterior lesions suggest MS; long posterior lesions with chiasm suggest NMOSD.

F23.10 · Chapter 23 · Answer

Optic nerve glioma: fusiform nerve enlargement; sheath meningioma: tram-track enhancement.

F23.9 · Chapter 23 · Answer

Cavernous venous malformation: ovoid intraconal mass with progressive enhancement from a point.

F24.2 · Chapter 24 · Answer

Pars flaccida cholesteatoma begins in Prussak space and erodes the scutum and ossicles.

F24.1 · Chapter 24 · Answer

Temporal bone CT for conductive loss, chronic otitis, and trauma; MRI for sensorineural loss and the angle.

F24.3 · Chapter 24 · Question

How accurate is non-EPI DWI for recurrent cholesteatoma?

F24.4 · Chapter 24 · Question

What is the size limit of non-EPI DWI for cholesteatoma?

F24.5 · Chapter 24 · Question

How much more likely are otic capsule-violating fractures to cause sensorineural loss?

F24.6 · Chapter 24 · Question

What are the imaging features of vestibular schwannoma?

F24.7 · Chapter 24 · Question

What are the imaging features of a cerebellopontine angle meningioma?

F24.8 · Chapter 24 · Question

How does serviceable hearing change after radiosurgery for vestibular schwannoma?

F24.9 · Chapter 24 · Question

How is sudden sensorineural hearing loss imaged?

F24.10 · Chapter 24 · Question

What symptoms does superior canal dehiscence cause, and how is it confirmed?

F24.4 · Chapter 24 · Answer

Non-EPI DWI misses cholesteatoma under 3 mm; follow up with serial imaging.

F24.3 · Chapter 24 · Answer

Non-EPI DWI for recurrent cholesteatoma: sensitivity about 90%, specificity about 95%.

F24.6 · Chapter 24 · Answer

Vestibular schwannoma: centered on the porus, enters the canal, acute angles with bone.

F24.5 · Chapter 24 · Answer

Otic capsule-violating fractures: 25 times more likely to cause sensorineural hearing loss.

F24.8 · Chapter 24 · Answer

Serviceable hearing after radiosurgery: 80% at 1 year, 23% at 10 years.

F24.7 · Chapter 24 · Answer

Meningioma: broad dural base, obtuse angles, calcification, hyperostosis, dural tail.

F24.10 · Chapter 24 · Answer

Superior canal dehiscence causes sound- and pressure-induced vertigo; confirm on Pöschl and Stenvers reformats.

F24.9 · Chapter 24 · Answer

Sudden sensorineural hearing loss: no routine head CT; MRI or ABR for retrocochlear disease.

F25.1 · Chapter 25 · Question

How does EPOS2020 classify chronic rhinosinusitis?

F25.2 · Chapter 25 · Question

Does uncomplicated acute sinusitis need imaging?

F25.3 · Chapter 25 · Question

What does a preoperative sinus CT report cover?

F25.4 · Chapter 25 · Question

How specific was the presence of two of seven CT variables for invasive fungal sinusitis?

F25.5 · Chapter 25 · Question

Which signs suggest invasive fungal sinusitis?

F25.6 · Chapter 25 · Question

What MRI pattern suggests inverted papilloma, and what suggests malignancy?

F25.7 · Chapter 25 · Question

What are the features of juvenile nasopharyngeal angiofibroma?

F25.8 · Chapter 25 · Question

How do chordoma and chondrosarcoma differ?

F25.2 · Chapter 25 · Answer

Uncomplicated acute sinusitis does not need imaging.

F25.1 · Chapter 25 · Answer

EPOS2020: chronic rhinosinusitis is primary or secondary, localized or diffuse.

F25.4 · Chapter 25 · Answer

Invasive fungal sinusitis: any two of seven CT variables gave 100% specificity in one model.

F25.3 · Chapter 25 · Answer

Preoperative CT: ostiomeatal units, Keros depth, lamina papyracea, anterior ethmoidal artery, Onodi cells.

F25.6 · Chapter 25 · Answer

Inverted papilloma: convoluted cerebriform pattern on MRI; focal loss suggests malignancy.

F25.5 · Chapter 25 · Answer

Periantral fat infiltration and the black turbinate sign suggest invasive fungal sinusitis.

F25.8 · Chapter 25 · Answer

Chordoma: midline clival, very T2 bright; chondrosarcoma: off midline, chondroid calcification.

F25.7 · Chapter 25 · Answer

Juvenile angiofibroma: adolescent boys, sphenopalatine foramen, intense enhancement; avoid biopsy.

F25.9 · Chapter 25 · Question

What does classical trigeminal neuralgia require?

F25.10 · Chapter 25 · Question

Why is MRI recommended in all trigeminal neuralgia?

F26.1 · Chapter 26 · Question

How is the space of origin of a suprahyoid neck mass identified?

F26.2 · Chapter 26 · Question

How do carotid space masses displace fat, and what do carotid body tumors do?

F26.3 · Chapter 26 · Question

How are cervical nodal levels defined?

F26.4 · Chapter 26 · Question

What is the best size criterion for nodal metastasis?

F26.5 · Chapter 26 · Question

What is the significance of nodal necrosis of 3 mm or larger?

F26.6 · Chapter 26 · Question

What did AJCC 8 change for head and neck cancer?

F25.10 · Chapter 25 · Answer

MRI is recommended in all trigeminal neuralgia because no clinical feature excludes a secondary cause.

F25.9 · Chapter 25 · Answer

Trigeminal neuralgia: classical needs vascular compression with morphologic root change.

F26.2 · Chapter 26 · Answer

Carotid space masses push the parapharyngeal fat anteriorly; carotid body tumors splay ICA and ECA.

F26.1 · Chapter 26 · Answer

Parapharyngeal fat displacement identifies the space of origin of a suprahyoid neck mass.

F26.4 · Chapter 26 · Answer

Minimal axial diameter of 10 mm (11 mm subdigastric) is the best size criterion for nodal metastasis.

F26.3 · Chapter 26 · Answer

Nodal levels I to VII follow imaging-based landmarks integrated with AJCC and AAO-HNS systems.

F26.6 · Chapter 26 · Answer

AJCC 8: separate staging for HPV-associated oropharyngeal cancer; extranodal extension added to N.

F26.5 · Chapter 26 · Answer

Nodal necrosis of 3 mm or larger was seen only in tumor-bearing nodes.

F26.7 · Chapter 26 · Question

How does HPV status affect oropharyngeal cancer survival?

F26.8 · Chapter 26 · Question

What does NI-RADS link its categories to?

F26.9 · Chapter 26 · Question

What did PET-CT surveillance after chemoradiotherapy achieve?

F26.10 · Chapter 26 · Question

When does an incidental thyroid nodule on CT need ultrasound?

F27.1 · Chapter 27 · Question

How does a herniation differ from a bulge?

F27.2 · Chapter 27 · Question

How do extrusion and protrusion differ?

F27.3 · Chapter 27 · Question

What does the Pfirrmann scale grade?

F27.4 · Chapter 27 · Question

How do Modic type 1 and type 2 changes differ?

F26.8 · Chapter 26 · Answer

NI-RADS links categories 1 to 4 with surveillance, inspection, biopsy, or treatment.

F26.7 · Chapter 26 · Answer

HPV-positive oropharyngeal cancer: 3-year survival 82.4% versus 57.1%.

F26.10 · Chapter 26 · Answer

Incidental thyroid nodule on CT: ultrasound if 1 cm or larger under 35, 1.5 cm or larger at 35 or older, or suspicious.

F26.9 · Chapter 26 · Answer

PET-CT surveillance after chemoradiotherapy avoided most neck dissections with noninferior survival.

F27.2 · Chapter 27 · Answer

Extrusion: displaced material exceeds its base in at least one plane; protrusion: base is wider.

F27.1 · Chapter 27 · Answer

Herniation involves less than 25% of the disc circumference; a bulge involves more than 25%.

F27.4 · Chapter 27 · Answer

Modic type 1: low T1, high T2, fibrovascular; type 2: high T1, fatty marrow.

F27.3 · Chapter 27 · Answer

Pfirrmann grades disc degeneration I to V on T2-weighted images with good reliability.

F27.5 · Chapter 27 · Question

What separates Modic type 1 change from discitis?

F27.6 · Chapter 27 · Question

Which lumbar stenosis grades predict failure of conservative treatment?

F27.7 · Chapter 27 · Question

Which root does a far lateral L4-L5 herniation compress?

F27.8 · Chapter 27 · Question

When is surgery recommended for degenerative cervical myelopathy?

F27.9 · Chapter 27 · Question

What defines cervical stenosis grade 3, and what predicts poorer outcome?

F27.10 · Chapter 27 · Question

When is imaging indicated for acute low back pain?

F28.1 · Chapter 28 · Question

What is the first imaging step in acute myelopathy?

F28.2 · Chapter 28 · Question

How do MS and NMOSD cord lesions differ?

F27.6 · Chapter 27 · Answer

Lumbar stenosis grade C and D by dural sac morphology predict failure of conservative treatment.

F27.5 · Chapter 27 · Answer

Intact endplates and a dehydrated disc separate Modic type 1 from discitis.

F27.8 · Chapter 27 · Answer

Surgery is recommended for moderate and severe degenerative cervical myelopathy.

F27.7 · Chapter 27 · Answer

Far lateral herniation at L4-L5 compresses the exiting L4 root.

F27.10 · Chapter 27 · Answer

Uncomplicated acute low back pain needs no imaging; consider it after 6 weeks or for red flags.

F27.9 · Chapter 27 · Answer

Cervical stenosis grade 3 includes cord signal change; T1 hypointensity predicts poorer outcome.

F28.2 · Chapter 28 · Answer

MS cord lesions are short and peripheral; NMOSD lesions are long and central.

F28.1 · Chapter 28 · Answer

Exclude cord compression first with whole-spine MRI in acute myelopathy.

F28.3 · Chapter 28 · Question

What enhancement pattern suggests spinal cord sarcoidosis?

F28.4 · Chapter 28 · Question

What are the clinical and MRI features of spinal cord infarction?

F28.5 · Chapter 28 · Question

What are the features of a spinal dural arteriovenous fistula, and what may worsen it?

F28.6 · Chapter 28 · Question

What do intramedullary tumors almost always do?

F28.7 · Chapter 28 · Question

How do ependymoma and astrocytoma of the cord differ?

F28.8 · Chapter 28 · Question

What are the features of a spinal hemangioblastoma?

F28.9 · Chapter 28 · Question

What cord signal pattern do B12 deficiency, copper deficiency, and nitrous oxide cause?

F28.10 · Chapter 28 · Question

What enhancement pattern suggests spondylotic myelopathy?

F28.4 · Chapter 28 · Answer

Spinal cord infarction: nadir within 12 hours; initial MRI normal in 24%; owl eyes in 65%.

F28.3 · Chapter 28 · Answer

Sarcoidosis: central canal and dorsal subpial enhancement forming a trident sign.

F28.6 · Chapter 28 · Answer

Intramedullary tumors almost always expand the cord and enhance.

F28.5 · Chapter 28 · Answer

Dural AV fistula: older men, cord edema, dorsal flow voids, enhancement; corticosteroids may worsen it.

F28.8 · Chapter 28 · Answer

Hemangioblastoma: dorsal enhancing nodule with large syrinx; consider von Hippel-Lindau disease.

F28.7 · Chapter 28 · Answer

Ependymoma: central, well defined, homogeneous enhancement; astrocytoma: eccentric and ill defined.

F28.10 · Chapter 28 · Answer

Spondylotic myelopathy: pancakelike enhancement just below the stenosis; avoid biopsy.

F28.9 · Chapter 28 · Answer

B12, copper deficiency, and nitrous oxide: symmetric dorsal column inverted V signal.

F29.1 · Chapter 29 · Question

What are the sensitivity and negative predictive value of NEXUS?

F29.2 · Chapter 29 · Question

What are the high-risk factors of the Canadian C-Spine Rule?

F29.3 · Chapter 29 · Question

When can the collar be removed in obtunded adults?

F29.4 · Chapter 29 · Question

What does the SLIC system score?

F29.5 · Chapter 29 · Question

What are the AOSpine thoracolumbar injury types?

F29.6 · Chapter 29 · Question

How should fractures in an ankylosed spine be managed on imaging?

F29.7 · Chapter 29 · Question

Which MRI findings are most sensitive for spondylodiscitis?

F29.8 · Chapter 29 · Question

What are the features of tuberculous spondylitis?

F29.2 · Chapter 29 · Answer

Canadian C-Spine Rule: high-risk factors include age 65 or older, dangerous mechanism, and paresthesias.

F29.1 · Chapter 29 · Answer

NEXUS: sensitivity 99.0% and negative predictive value 99.8% for cervical spine injury.

F29.4 · Chapter 29 · Answer

SLIC scores morphology, discoligamentous complex, and neurologic status.

F29.3 · Chapter 29 · Answer

Obtunded adults: collar removal is conditionally recommended after a negative high-quality CT.

F29.6 · Chapter 29 · Answer

Fractures in an ankylosed spine are unstable; image the whole spine.

F29.5 · Chapter 29 · Answer

AOSpine: type A compression, type B tension band, type C translation injuries.

F29.8 · Chapter 29 · Answer

Tuberculous spondylitis: subligamentous spread, disc sparing, large thin-walled abscesses.

F29.7 · Chapter 29 · Answer

Spondylodiscitis: paraspinal or epidural inflammation (97.7%) and disc enhancement (95.4%) are most sensitive.

F29.9 · Chapter 29 · Question

What are the SINS score categories?

F29.10 · Chapter 29 · Question

Which ESCC grades indicate high-grade cord compression?

F30.1 · Chapter 30 · Question

How are malformations of cortical development grouped?

F30.2 · Chapter 30 · Question

Why can infant MRI miss focal cortical dysplasia, and when is it repeated?

F30.3 · Chapter 30 · Question

What defines Chiari I malformation, and how often is there a syrinx?

F30.4 · Chapter 30 · Question

What does Chiari II malformation accompany?

F30.5 · Chapter 30 · Question

What defines Dandy-Walker malformation?

F30.6 · Chapter 30 · Question

What are the brain findings of NF1, and what was NF2 renamed?

F29.10 · Chapter 29 · Answer

ESCC grades 2 and 3 indicate high-grade cord compression on axial T2-weighted images.

F29.9 · Chapter 29 · Answer

SINS: 0 to 6 stable, 7 to 12 potentially unstable, 13 to 18 unstable.

F30.2 · Chapter 30 · Answer

Myelination in infants can hide focal cortical dysplasia; repeat MRI after about age 2.

F30.1 · Chapter 30 · Answer

Cortical malformations: proliferation (dysplasia II, hemimegalencephaly), migration (heterotopia, lissencephaly), postmigration (polymicrogyria).

F30.4 · Chapter 30 · Answer

Chiari II accompanies open spinal dysraphism.

F30.3 · Chapter 30 · Answer

Chiari I: tonsils at least 5 mm below the foramen magnum, crowding, small posterior fossa; syrinx in 65%.

F30.6 · Chapter 30 · Answer

NF1: optic pathway glioma and focal areas of signal intensity; NF2 renamed NF2-related schwannomatosis.

F30.5 · Chapter 30 · Answer

Dandy-Walker: hypoplastic rotated vermis with cystic fourth ventricle and enlarged posterior fossa.

F30.7 · Chapter 30 · Question

What are the imaging findings of tuberous sclerosis, and where does SEGA arise?

F30.8 · Chapter 30 · Question

What do the PECARN rules identify?

F30.9 · Chapter 30 · Question

How do profound and partial asphyxia injury patterns differ in term infants?

F30.10 · Chapter 30 · Question

What is the leading cause of fatal head injury under 2 years?

F30.8 · Chapter 30 · Answer

PECARN rules identify children at very low risk of clinically important brain injury.

F30.7 · Chapter 30 · Answer

Tuberous sclerosis: tubers, radial migration lines, subependymal nodules; SEGA near the foramen of Monro.

F30.10 · Chapter 30 · Answer

Abusive head trauma is the leading cause of fatal head injury under 2 years; exclude mimics.

F30.9 · Chapter 30 · Answer

Term profound asphyxia injures thalami, putamina, and perirolandic cortex; partial asphyxia injures watershed zones.