COMPREHENSIVE MOCK EXAM 6

Supplemental to: Vascular Neurology Board Review 2026-2027

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ABPN Vascular Neurology Certification Examination — 220 single-best-answer questions, five options (A-E), domain-weighted to the official content specifications

Board-Review Questions

1. A 71-year-old right-handed man develops sudden aphasia and dense right hemiparesis 95 minutes ago. CT shows no hemorrhage or large established infarct. CTA shows a left M1 occlusion. His blood pressure is 168/92 mm Hg, glucose is normal, and he takes no anticoagulant. Which acute treatment strategy is most appropriate?

A. Give intravenous thrombolysis promptly and proceed to thrombectomy evaluation without waiting for clinical response

B. Observe for one hour to determine whether the deficit improves before reperfusion therapy

C. Withhold thrombolysis because a large-vessel occlusion requires thrombectomy alone

D. Lower systolic pressure below 140 mm Hg before considering any reperfusion treatment

E. Give aspirin and defer reperfusion because the CT does not yet show infarction as the preferred approach in this clinical setting

2. A patient with an otherwise eligible disabling ischemic stroke is being transferred for thrombectomy. Which principle should guide use of intravenous thrombolysis before transfer?

A. Use thrombolysis primarily after a repeat CT demonstrates a completed infarct

B. Reserve thrombolysis primarily for patients in whom thrombectomy has already failed

C. Do not delay indicated intravenous thrombolysis solely because thrombectomy is planned

D. Delay thrombolysis until the receiving center confirms groin-puncture availability

E. Omit thrombolysis whenever any intracranial occlusion is visible on CTA

3. A 63-year-old woman presents 70 minutes after abrupt isolated left-hand numbness and mild facial sensory loss. NIHSS is 2, the deficits are judged nondisabling, CT excludes hemorrhage, and CTA shows no large-vessel occlusion. Which treatment approach best fits contemporary acute stroke practice?

A. Give intravenous thrombolysis because any measurable NIHSS deficit is disabling as the preferred approach in this clinical setting

B. Withhold all antithrombotic treatment for 24 hours because ischemia is not visible on CT

C. Use early antiplatelet therapy, typically short-course dual antiplatelet therapy when eligibility criteria are met

D. Perform immediate mechanical thrombectomy despite absence of an occluded target artery

E. Start therapeutic heparin to prevent early neurologic worsening from presumed embolism

4. A patient with a minor nondisabling noncardioembolic ischemic stroke has no hemorrhage and no indication for anticoagulation. Which early prevention strategy is most appropriate?

A. Continue aspirin plus clopidogrel indefinitely because recurrence risk remains highest for life as the preferred approach in this clinical setting

B. Use aspirin plus dipyridamole plus clopidogrel as routine triple therapy as the preferred approach in this clinical setting

C. Begin warfarin immediately because all early recurrent strokes are presumed embolic as the preferred approach in this clinical setting

D. Avoid antiplatelet therapy until a follow-up MRI proves infarction as the preferred approach in this clinical setting

E. Begin a short course of aspirin plus clopidogrel when the patient meets minor-stroke criteria, then continue single antiplatelet therapy

5. A 58-year-old man with a disabling acute ischemic stroke arrives 2 hours after onset. He has no contraindication to thrombolysis and is a thrombectomy candidate. Which thrombolytic regimen is a contemporary evidence-based option?

A. Tenecteplase 0.4 mg/kg as an infusion over 60 minutes, maximum 90 mg

B. Urokinase 1 million units as routine first-line systemic thrombolysis

C. Streptokinase 1.5 million units because it has the longest stroke evidence base

D. Tenecteplase 0.25 mg/kg as a single intravenous bolus, maximum 25 mg

E. Alteplase 0.25 mg/kg as a single intravenous bolus, maximum 25 mg

6. A hospital is revising its thrombolysis pathway to simplify treatment before transfer. Which practical advantage of tenecteplase is most relevant?

A. It has no risk of symptomatic intracranial hemorrhage after treatment

B. It eliminates the need to assess blood pressure before thrombolytic treatment

C. It can be administered as a single intravenous bolus rather than a one-hour infusion

D. Use tenecteplase without additional anticoagulant assessment after a recent factor Xa inhibitor dose if renal function is normal

E. It requires no screening for intracranial hemorrhage before administration

7. A 66-year-old man with a disabling stroke is otherwise eligible for intravenous thrombolysis, but his blood pressure is 198/116 mm Hg. What is the most appropriate next step?

A. Give thrombolysis immediately because treatment delay is more dangerous than this blood pressure

B. Lower blood pressure to below 185/110 mm Hg with a rapidly titratable agent, then give thrombolysis if otherwise eligible

C. Administer oral long-acting antihypertensives and reassess for thrombolysis the next morning

D. Lower systolic blood pressure to below 120 mm Hg before giving thrombolysis as the preferred approach in this clinical setting

E. Defer thrombolysis because an initial pressure above 185/110 mm Hg predicts excessive bleeding risk even if later controlled

8. A thrombolysis-treated patient has stable neurologic findings after reperfusion. What blood-pressure ceiling is generally used during the first 24 hours after intravenous thrombolysis?

A. Maintain systolic blood pressure below 120 mm Hg in all patients

B. Maintain diastolic blood pressure below 60 mm Hg regardless of symptoms

C. Maintain blood pressure below 180/105 mm Hg given the clinical findings and context described

D. Target a mean arterial pressure below 55 mm Hg to reduce hemorrhage risk

E. Permit blood pressure up to 220/120 mm Hg without treatment

9. A 74-year-old woman awakens with aphasia and right weakness. She was last known well 7 hours earlier. MRI shows a small diffusion-restricted lesion with little corresponding FLAIR hyperintensity and no hemorrhage. Which interpretation is most appropriate?

A. Unknown onset time automatically excludes all intravenous reperfusion treatment as the best diagnosis in this clinical setting

B. The absence of hemorrhage alone makes most wake-up stroke eligible for thrombolysis as the best diagnosis in this clinical setting

C. MRI mismatch is used primarily to decide whether aspirin should be withheld as the best diagnosis in this clinical setting

D. The imaging mismatch can identify selected patients who may still benefit from intravenous thrombolysis despite unknown onset time

E. FLAIR hyperintensity must exceed the diffusion lesion before thrombolysis is considered as the best diagnosis in this clinical setting

10. A hospital evaluates a patient 8 hours after last known well with a disabling deficit and no thrombectomy target. Which strategy can extend thrombolysis consideration beyond a simple clock-time rule?

A. Use a normal noncontrast CT alone as proof that all brain tissue is salvageable

B. Base treatment primarily on the patient being younger than 80 years as the preferred approach in this clinical setting

C. Use validated tissue-based imaging selection such as diffusion-FLAIR mismatch or favorable perfusion mismatch

D. Require a visible hyperdense artery sign before considering thrombolysis as the preferred approach in this clinical setting

E. Treat primarily if the NIHSS score is exactly 6 or lower in this clinical context as the preferred approach in this clinical setting

11. A 69-year-old woman presents with aphasia and right hemiparesis 3 hours after onset. She took apixaban for atrial fibrillation 4 hours ago. Renal function is normal. Which additional information is most directly relevant to intravenous thrombolysis eligibility?

A. Whether an echocardiogram shows left atrial enlargement at this time given the clinical findings and context described

B. Whether the patient has a carotid bruit on the symptomatic side as the next step given the clinical findings and context described

C. Whether clinically significant factor Xa inhibitor activity is absent based on timing and, when available, an appropriate drug-specific assay

D. Whether aspirin was taken at any point during the previous month for this patient given the clinical findings and context described

E. Whether LDL cholesterol is below 70 mg/dL at presentation in this clinical context given the clinical findings and context described

12. An otherwise eligible stroke patient has taken a direct oral anticoagulant very recently and has evidence of active anticoagulant effect. What is the safest general approach to intravenous thrombolysis?

A. Withhold routine intravenous thrombolysis while clinically significant anticoagulant activity is present

B. Use intra-arterial heparin instead of thrombectomy when a large-vessel occlusion is present

C. Give thrombolysis at half dose because anticoagulation lowers the required fibrinolytic dose

D. Ignore anticoagulant exposure if the presenting NIHSS score exceeds 10 as the preferred approach in this clinical setting

E. Reverse most direct oral anticoagulant and immediately give thrombolysis as a standard strategy

13. A 52-year-old man with sudden aphasia and hemiparesis has a platelet count of 72,000/µL. CT shows no hemorrhage. Which finding is most important for intravenous thrombolysis?

A. The normal CT proves that bleeding risk is negligible despite thrombocytopenia

B. Thrombocytopenia is relevant primarily after thrombectomy, not before thrombolysis

C. The platelet count is below the accepted safety threshold for routine intravenous thrombolysis

D. The presence of aphasia makes thrombolysis preferred regardless of platelet count

E. The young age overrides laboratory contraindications to thrombolysis

14. A patient with a disabling ischemic stroke has a platelet count of 85,000/µL but also has an M1 occlusion and favorable imaging for thrombectomy. Which approach is most appropriate?

A. Delay all treatment until the platelet count spontaneously exceeds 150,000/µL as the preferred approach in this clinical setting

B. Give full-dose thrombolysis and cancel thrombectomy once the infusion starts as the preferred approach in this clinical setting

C. Avoid routine intravenous thrombolysis because of thrombocytopenia while still evaluating candidacy for mechanical thrombectomy

D. Transfuse platelets primarily to make the patient eligible for immediate thrombolysis as the preferred approach in this clinical setting

E. Avoid all reperfusion therapy because thrombolysis and thrombectomy have identical hematologic exclusions

15. A 60-year-old woman arrives with abrupt dysarthria and left weakness. Point-of-care glucose is 31 mg/dL. What is the best immediate step?

A. Withhold glucose because correction can worsen ischemic penumbra given the clinical findings and context described

B. Correct the hypoglycemia promptly and reassess the neurologic deficit before labeling the event as persistent ischemic stroke

C. Start long-term anticoagulation because hypoglycemia implies cardioembolism given the clinical findings and context described

D. Give intravenous thrombolysis immediately because low glucose confirms cerebral ischemia given the clinical findings and context described

E. Perform carotid endarterectomy before correcting the metabolic abnormality given the clinical findings and context described

16. A patient with suspected stroke becomes neurologically normal immediately after intravenous dextrose corrects severe hypoglycemia. What is the most appropriate interpretation?

A. The episode was most likely a metabolic stroke mimic rather than completed cerebral infarction

B. The response indicates that thrombolysis should still be administered prophylactically

C. The response confirms a large-vessel occlusion that spontaneously recanalized

D. The response establishes nonconvulsive status epilepticus as the cause

E. The response proves a transient ischemic attack from carotid stenosis

17. A 77-year-old man has a generalized seizure at the moment of sudden onset of left hemiplegia. After the seizure stops, he remains aphasic with dense left weakness, and CTA shows a right M1 occlusion. What is the best interpretation?

A. Any seizure at onset proves the residual deficit is Todd paralysis given the clinical findings and context described

B. Treat the persistent deficit as postictal Todd paresis unless it lasts several hours despite the concordant vascular occlusion

C. The M1 occlusion is incidental because seizures occur primarily with venous infarction given the clinical findings and context described

D. The persistent deficit should be observed for 24 hours before vascular treatment given the clinical findings and context described

E. Seizure at onset does not exclude ischemic stroke when persistent deficits are attributable to a documented vascular occlusion

18. Which management principle is most appropriate when a seizure occurs at stroke onset but a disabling vascular deficit persists afterward?

A. Treat the underlying ischemic stroke when the residual deficit is judged due to ischemia and reperfusion criteria are met

B. Assume all postictal weakness is benign and avoid vascular imaging as the preferred approach in this clinical setting

C. Use therapeutic anticoagulation as the first-line treatment for postictal weakness as the preferred approach in this clinical setting

D. Give chronic antiseizure medication before any reperfusion decision is made as the preferred approach in this clinical setting

E. Delay thrombectomy until an electroencephalogram excludes most epileptiform discharge as the preferred approach in this clinical setting

19. A 67-year-old woman presents with severe aphasia that improves substantially over 20 minutes but remains disabling because she still cannot name common objects or follow multistep commands. CT shows no hemorrhage. How should the improvement affect thrombolysis decision-making?

A. Improvement requires waiting until the entire 4.5-hour window has elapsed as the best mechanistic explanation for these findings

B. Persistent disabling deficit remains a reason to consider thrombolysis despite partial spontaneous improvement

C. Thrombolysis is indicated primarily if the NIHSS score increases after arrival as the best mechanistic explanation for these findings

D. Aphasia is nondisabling whenever motor strength is normal as the best mechanistic explanation for these findings

E. Any early improvement is an absolute reason to withhold thrombolysis as the best mechanistic explanation for these findings

20. A patient improves from NIHSS 14 to NIHSS 5 within 30 minutes, but the remaining deficit is dense homonymous hemianopia preventing safe ambulation and driving. Which principle is most appropriate?

A. Use the percentage improvement in NIHSS as the sole thrombolysis criterion as the preferred approach in this clinical setting

B. Withhold reperfusion because an NIHSS score of 5 generally predicts a nondisabling deficit regardless of the specific symptoms

C. Wait for complete spontaneous resolution before deciding on acute therapy as the preferred approach in this clinical setting

D. Treat primarily if a repeat scan shows hemorrhagic transformation as the preferred approach in this clinical setting

E. Base reperfusion decisions on whether the remaining deficit is disabling, not simply on the lower NIHSS score

21. A 73-year-old man presents 2 hours after onset of a disabling left hemispheric syndrome. Noncontrast CT shows subtle loss of gray-white differentiation in a small portion of the left insula but no hemorrhage. What is the best interpretation?

A. Any visible ischemic change is equivalent to intracranial hemorrhage as the best diagnosis in this clinical setting

B. Early ischemic change on CT does not by itself preclude thrombolysis when there is no large completed infarct and other criteria are met

C. Subtle ischemic change mandates immediate decompressive hemicraniectomy as the best diagnosis in this clinical setting

D. Thrombolysis requires a completely normal CT without any early ischemic signs as the best diagnosis in this clinical setting

E. A small insular change proves the stroke is older than 24 hours in this clinical context as the best diagnosis in this clinical setting

22. Why is noncontrast head CT obtained urgently before intravenous thrombolysis in most acute stroke patients?

A. To exclude intracranial hemorrhage and identify major established injury or alternative pathology that changes treatment

B. To measure carotid stenosis without additional vascular imaging as the best mechanistic explanation for these findings

C. To prove that most ischemic stroke already has visible hypodensity as the best mechanistic explanation for these findings

D. To determine the exact embolic source before reperfusion begins as the best mechanistic explanation for these findings

E. To establish the patient's long-term cognitive prognosis before treatment as the best mechanistic explanation for these findings

23. A 66-year-old man with prestroke mRS 0 presents 4 hours after onset with NIHSS 18. CTA shows an ICA terminus occlusion. CT demonstrates a large ischemic core but no hemorrhage. Which statement best reflects modern thrombectomy evidence?

A. Large-core patients should receive heparin instead of endovascular reperfusion

B. Selected patients with large ischemic cores can still benefit from mechanical thrombectomy

C. A large core is an absolute contraindication to thrombectomy in most time window

D. Thrombectomy is beneficial primarily when ASPECTS is 10 with no visible ischemia

E. A visible core requires waiting for spontaneous recanalization before treatment

24. A thrombectomy-capable center is revising its protocol for anterior-circulation large-vessel occlusion. Which change best reflects current evidence?

A. Use intravenous heparin as a preferred bridge before most thrombectomy given the clinical findings and context described

B. Allow selected large-core patients to undergo endovascular evaluation rather than excluding them categorically

C. Restrict thrombectomy to patients younger than 50 years regardless of imaging given the clinical findings and context described

D. Exclude all patients with any visible infarct on noncontrast CT given the clinical findings and context described

E. Require complete absence of diffusion restriction before thrombectomy given the clinical findings and context described

25. A 70-year-old woman presents 11 hours after she was last known well with a disabling anterior-circulation syndrome and an ICA terminus occlusion. Imaging suggests salvageable tissue despite an established core. What is the most appropriate next step?

A. Give aspirin alone because any stroke discovered after 6 hours is completed

B. Wait for MRI at 24 hours to determine whether spontaneous recovery occurs

C. Proceed with late-window thrombectomy evaluation using clinical and imaging selection

D. Use medical therapy alone because thrombectomy benefit is insufficient once a patient presents beyond the traditional 6-hour window

E. Use carotid endarterectomy as the emergency treatment for the intracranial occlusion

26. Which principle most accurately describes selection for thrombectomy 6 to 24 hours after last known well?

A. Use validated clinical and imaging criteria to identify patients likely to benefit despite the delayed presentation

B. Require a normal MRI with no diffusion lesion before considering intervention given the clinical findings and context described

C. Exclude all wake-up strokes because onset time is uncertain given the clinical findings and context described

D. Use the severity of intracranial stenosis rather than tissue imaging as the main determinant of late-window thrombectomy eligibility

E. Use age alone as the decisive criterion and ignore infarct burden given the clinical findings and context described

27. A 64-year-old man presents 9 hours after onset with coma, quadriparesis, and dysarthria. CTA shows a basilar artery occlusion; NIHSS is 18, baseline mRS was 0, and posterior-circulation imaging shows limited established injury. Which treatment is most appropriate?

A. Give prophylactic antiseizure medication as the primary reperfusion strategy

B. Perform carotid endarterectomy because posterior circulation is supplied by the carotids

C. Delay intervention until 24 hours to see whether collateral flow is sufficient

D. Treat with aspirin alone because posterior-circulation occlusions are not thrombectomy targets

E. Perform urgent endovascular thrombectomy evaluation for basilar artery occlusion

28. A patient with basilar artery occlusion is being considered for thrombectomy. Which clinical feature most strengthens the expected benefit from intervention?

A. A normal neurologic examination despite the vascular finding as the preferred approach in this clinical setting

B. An incidental basilar stenosis with no neurologic symptoms as the preferred approach in this clinical setting

C. A remote history of migraine without current focal deficits as the preferred approach in this clinical setting

D. A chronic occlusion with extensive completed bilateral pontine infarction as the preferred approach in this clinical setting

E. A severe disabling posterior-circulation syndrome with a treatable basilar occlusion and limited established infarction

29. A 57-year-old woman presents 3 hours after onset with NIHSS 9, aphasia, and right arm weakness. CTA shows an occlusion of a proximal dominant M2 branch supplying much of the left lateral frontal lobe. ASPECTS is 9 and baseline mRS was 0. Which statement is most appropriate?

A. Thrombectomy is indicated primarily for isolated posterior cerebral artery occlusion as the preferred approach in this clinical setting

B. Mechanical thrombectomy is a reasonable treatment consideration for this selected proximal dominant M2 occlusion

C. The patient should undergo carotid endarterectomy instead of intracranial reperfusion

D. Endovascular therapy requires waiting until the NIHSS score exceeds 20 as the preferred approach in this clinical setting

E. All M2 occlusions are too distal for endovascular therapy regardless of symptoms

30. A stroke center is deciding whether to transfer a patient with a proximal dominant M2 occlusion and disabling deficits. What is the best systems approach?

A. Wait for the infarct to become visible on CT before contacting a thrombectomy center as the preferred approach in this clinical setting

B. Transfer primarily if intravenous thrombolysis is generally contraindicated as the preferred approach in this clinical setting

C. Delay transfer until a repeat CTA proves the thrombus has enlarged into M1 as the preferred approach in this clinical setting

D. Keep the patient locally because no M2 lesion can ever be treated endovascularly as the preferred approach in this clinical setting

E. Transfer promptly for endovascular evaluation when the patient meets reasonable clinical and imaging selection criteria

31. A 72-year-old man with mild isolated sensory loss has a distal M3 branch occlusion on CTA and NIHSS 2. What is the most appropriate interpretation regarding thrombectomy?

A. Proceed with thrombectomy because a distal arterial occlusion is visible despite the mild nondisabling neurologic deficit

B. Any visible intracranial occlusion mandates immediate thrombectomy

C. Routine thrombectomy is not established for this mild distal-medium-vessel presentation

D. Distal M3 occlusion has stronger thrombectomy evidence than proximal M1 occlusion

E. The absence of motor weakness is a reason to start systemic heparin instead

32. Which principle best prevents overuse of thrombectomy in distal occlusions?

A. Use thrombectomy mainly to prevent future atherosclerosis rather than to restore acute flow given the clinical findings and context described

B. Perform thrombectomy whenever CTA detects any arterial narrowing for this patient given the clinical findings and context described

C. Require evidence that the occlusion site, deficit severity, tissue status, and expected functional benefit justify the procedural risk

D. Ignore deficit severity because angiographic anatomy alone determines benefit given the clinical findings and context described

E. Treat most distal occlusion before considering intravenous therapy given the clinical findings and context described

33. A patient with acute left ICA cervical occlusion and tandem left M1 occlusion presents within 3 hours with NIHSS 17. Which statement is most accurate?

A. The cervical lesion makes intracranial thrombectomy technically not routinely feasible given the clinical findings and context described

B. Tandem lesions should be observed because reperfusion increases distal embolization in all patients

C. primarily carotid endarterectomy can restore flow during the acute stroke given the clinical findings and context described

D. Withhold intravenous thrombolysis because a cervical ICA lesion lowers recanalization and increases bleeding risk during tandem-lesion treatment

E. Tandem cervical and intracranial occlusions can still be treated with endovascular reperfusion in appropriately selected patients

34. During thrombectomy for a tandem ICA-MCA occlusion, how should the cervical carotid lesion generally be approached?

A. Cross the cervical lesion and defer treatment of it even when the lesion significantly limits access or antegrade flow

B. Give full-dose systemic anticoagulation as the primarily accepted treatment for the cervical lesion

C. Perform emergency open endarterectomy during the same thrombectomy procedure in most case as the best diagnosis in this clinical setting

D. Stent most cervical lesion before confirming an intracranial occlusion as the best diagnosis in this clinical setting

E. Individualize angioplasty or stenting decisions based on access, flow, lesion morphology, bleeding risk, and antithrombotic needs

35. A 65-year-old man has an NIHSS score of 16 and right M1 occlusion. Noncontrast CT shows early ischemic changes in the right MCA territory with ASPECTS 8. What does the ASPECTS score indicate?

A. The patient has an 80% cervical carotid stenosis by CT criteria

B. The hemorrhage volume is approximately 8 mL by the ABC/2 method

C. There are limited early ischemic changes, with most scored MCA regions still preserved

D. Eight of ten posterior-circulation regions are already infarcted

E. There is diffuse subarachnoid blood in eight basal cistern compartments

36. How should ASPECTS be used in modern thrombectomy decision-making?

A. Use it as one measure of established ischemic injury within a broader clinical and imaging assessment

B. Use it primarily after thrombectomy to grade reperfusion success given the clinical findings and context described

C. Use it to determine whether atrial fibrillation requires anticoagulation

D. Use an ASPECTS score below 6 as a stand-alone exclusion from thrombectomy regardless of other large-core selection factors

E. Use it to replace vascular imaging when an occlusion is suspected given the clinical findings and context described

37. CT perfusion in a patient with an M1 occlusion estimates an ischemic core of 25 mL and a critically hypoperfused region of 90 mL. What does this pattern most strongly suggest?

A. A substantial volume of hypoperfused tissue remains outside the estimated core and may represent salvageable penumbra

B. Perfusion mismatch proves the stroke is caused by small-vessel lipohyalinosis given the clinical findings and context described

C. The entire 90 mL region is already irreversibly infarcted given the clinical findings and context described

D. The 25 mL core represents normal tissue with increased blood flow given the clinical findings and context described

E. The imaging pattern rules out any benefit from reperfusion therapy given the clinical findings and context described

38. Which limitation should be remembered when interpreting automated CT-perfusion maps?

A. Perfusion maps eliminate the need to inspect noncontrast CT or CTA as the preferred approach in this clinical setting

B. Core and penumbra estimates are model-dependent and should be reconciled with the clinical syndrome and source images

C. Automated maps are infallible and should override obvious clinical-imaging mismatch as the preferred approach in this clinical setting

D. Treat the automated core estimate as fixed and definitive without reconciling it with the source images or clinical syndrome

E. Perfusion imaging directly identifies the cardiac source of embolism as the preferred approach in this clinical setting

39. A 68-year-old woman has successful thrombectomy with TICI 3 reperfusion. Her postprocedure systolic pressure is 162 mm Hg. Which approach is most appropriate?

A. Stop all blood-pressure monitoring after angiographic reperfusion is confirmed at this time as the preferred approach in this clinical setting

B. Allow systolic pressure above 220 mm Hg because the artery is now open as the next step as the preferred approach in this clinical setting

C. Immediately lower systolic pressure to 100 mm Hg to minimize reperfusion injury for this patient as the preferred approach in this clinical setting

D. Avoid forcing systolic pressure below 140 mm Hg solely because reperfusion was successful; use a reasonable post-thrombectomy target and prevent marked hypertension or hypotension

E. Induce hypotension to reduce cerebral edema in the reperfused territory in this clinical context as the preferred approach in this clinical setting

40. Why can overly aggressive blood-pressure reduction after thrombectomy be harmful?

A. Assume that lowering blood pressure after thrombectomy directly increases the risk of large-vessel reocclusion

B. Reperfused brain universally develops systemic hypertension to prevent seizures as the best mechanistic explanation for these findings

C. Blood pressure has no effect on cerebral perfusion after mechanical recanalization as the best mechanistic explanation for these findings

D. Post-thrombectomy brain no longer receives blood through collateral pathways as the best mechanistic explanation for these findings

E. Previously ischemic tissue may still have impaired autoregulation and depend on adequate systemic pressure for microvascular perfusion

41. A 49-year-old man has a massive right MCA infarction. At 36 hours he becomes progressively somnolent; CT shows increasing edema, 10-mm midline shift, and compression of the right lateral ventricle. What is the most appropriate treatment?

A. Use hyperosmolar therapy alone and defer surgical evaluation unless fixed pupillary changes develop

B. Use therapeutic anticoagulation because the edema is presumed to reflect ongoing arterial thrombosis

C. Perform urgent carotid revascularization first to improve perfusion of the already infarcted hemisphere

D. Treat with high-dose corticosteroids as the principal therapy for postischemic cerebral edema

E. Urgent neurosurgical evaluation for decompressive hemicraniectomy

42. A patient with a large MCA infarct is at risk for malignant edema. Which management step is most important before clinical herniation occurs?

A. Maintain continuous prophylactic hyperosmolar therapy to a high sodium target before clinical or radiographic swelling develops

B. Monitor closely in a setting capable of rapid neurologic reassessment and obtain early neurosurgical consultation when swelling risk is high

C. Delay family discussion until after fixed bilateral pupils develop at this time as the preferred approach in this clinical setting

D. Give corticosteroids routinely to most large hemispheric infarct for this patient as the preferred approach in this clinical setting

E. Avoid repeat imaging because edema is diagnosed primarily clinically as the next step as the preferred approach in this clinical setting

43. A 62-year-old woman with a large cerebellar infarction develops declining consciousness. CT shows fourth-ventricular compression and obstructive hydrocephalus. What is the best next step?

A. Urgent neurosurgical management with suboccipital decompression, with CSF diversion as needed

B. Perform carotid endarterectomy because hydrocephalus implies carotid disease

C. Give full-dose heparin to shrink the swollen cerebellar hemisphere

D. Observe because cerebellar infarcts rarely cause life-threatening mass effect

E. Treat with corticosteroids alone and repeat CT in one week as the preferred approach in this clinical setting

44. Why is isolated ventricular drainage potentially insufficient in a patient with a swollen cerebellar infarct and marked posterior-fossa mass effect?

A. It prevents any subsequent suboccipital decompression from being performed

B. It may relieve hydrocephalus without correcting brainstem compression from the swollen cerebellum

C. It treats the infarct mechanism but not the associated atrial fibrillation

D. It irreversibly closes the basilar artery by lowering intracranial pressure

E. Avoid ventricular drainage because upward transtentorial herniation is expected to outweigh any benefit from treating hydrocephalus

45. A 55-year-old man develops sudden vertigo, dysarthria, bilateral facial weakness, quadriparesis, and then inability to move except for vertical eye movements. He remains awake and can communicate by blinking. Where is the lesion most likely located?

A. Cervical spinal cord causing quadriparesis with loss of vertical eye movement control

B. Bilateral occipital lobes causing cortical blindness without motor pathway involvement

C. Medial frontal lobes causing akinetic mutism with preserved limb movement as the best diagnosis in this clinical setting

D. Ventral pons involving corticospinal and corticobulbar tracts with preserved tegmental arousal pathways

E. Peripheral neuromuscular junction causing fatigable weakness and ptosis as the best diagnosis in this clinical setting

46. A patient with basilar artery thrombosis appears unresponsive but intermittently moves the eyes vertically to command. What is the most important bedside action?

A. Treat the patient as deeply comatose and continue sedation without testing a voluntary eye-response channel

B. Assess horizontal gaze only and regard preserved vertical eye movements as a brainstem reflex

C. Assume profound coma from the absent limb and verbal responses without testing command following

D. Establish a reliable eye-movement communication code and assess consciousness before assuming coma

E. Attribute the eye movements to subtle seizure activity before establishing whether they are reproducibly voluntary

47. A 68-year-old man develops pure left face-arm-leg weakness without aphasia, neglect, visual field loss, or sensory symptoms. MRI shows a small acute infarct in the right posterior limb of the internal capsule. Which mechanism is most likely?

A. Thrombosis of the superior sagittal sinus with venous infarction

B. Occlusion of a small penetrating artery related to lipohyalinosis or microatheroma

C. Rupture of a cortical arteriovenous malformation as the best diagnosis in this clinical setting

D. Embolic occlusion of the left posterior cerebral artery as the best diagnosis in this clinical setting

E. Bilateral vertebral dissection causing cerebellar ischemia as the best diagnosis in this clinical setting

48. A patient with a recent lacunar stroke has no atrial fibrillation and no large-artery stenosis. Which long-term antithrombotic strategy is generally appropriate?

A. Withhold antithrombotic therapy because small-vessel disease is nonthrombotic

B. Begin warfarin because small-vessel strokes are usually occult cardioembolic events

C. Use lifelong dual antiplatelet therapy because lacunes have the highest platelet burden

D. Use single antiplatelet therapy rather than indefinite aspirin-clopidogrel dual therapy

E. Use full-dose low-molecular-weight heparin indefinitely as the preferred approach in this clinical setting

49. A 61-year-old woman develops sudden hemisensory loss affecting the left face, arm, and leg without weakness or cortical signs. MRI shows a small right thalamic infarct. Which stroke syndrome is most likely?

A. Lateral medullary syndrome from posterior inferior cerebellar artery infarction

B. Pure sensory lacunar syndrome from thalamic perforator ischemia

C. Cerebellar hemisphere infarction causing ipsilateral limb ataxia

D. Anterior cerebral artery syndrome causing abulia and leg weakness

E. Dominant temporal lobe infarction causing Wernicke aphasia

50. A patient develops persistent burning dysesthesia and allodynia months after a right thalamic stroke. Which mechanism best explains the pain syndrome?

A. Peripheral demyelination of the median and ulnar nerves from the thalamic lesion

B. Deafferentation and maladaptive central sensory processing after injury to thalamocortical pathways

C. Ongoing arterial occlusion causing repeated acute infarction most time the skin is touched

D. Spinal cord compression caused by remote cerebral edema as the best diagnosis in this clinical setting

E. Neuromuscular junction failure triggered by central sensory loss as the best diagnosis in this clinical setting

51. A 59-year-old right-handed man has a left posterior cerebral artery infarct involving the medial occipital lobe and splenium. He can write a sentence but cannot read what he just wrote. Which syndrome is present?

A. Hemispatial neglect from a nondominant parietal infarct

B. Pure motor aphasia from an isolated pontine lesion

C. Broca aphasia with impaired speech production and preserved repetition

D. Alexia without agraphia in this setting as the best diagnosis in this clinical setting

E. Gerstmann syndrome from angular gyrus dysfunction as the best diagnosis in this clinical setting

52. What anatomic disconnection produces alexia without agraphia after a dominant PCA infarct?

A. Visual information from the intact right occipital cortex cannot cross the damaged splenium to reach dominant-hemisphere language regions

B. Somatosensory input is unlikely to cross the medulla to reach the thalamus as the best mechanistic explanation for these findings

C. Auditory input from the cochlea is unlikely to reach bilateral temporal cortices as the best mechanistic explanation for these findings

D. Motor output from the dominant frontal lobe is unlikely to reach the contralateral corticospinal tract as the best mechanistic explanation for these findings

E. Vestibular information is unlikely to reach the ipsilateral cerebellar hemisphere as the best mechanistic explanation for these findings

53. A 72-year-old woman has sudden left leg-predominant weakness, urinary urgency, abulia, and reduced spontaneous speech. Which arterial territory is most likely involved?

A. Right anterior cerebral artery territory involving medial frontal structures

B. Left anterior inferior cerebellar artery territory involving the lateral pons

C. Right posterior inferior cerebellar artery territory involving the lateral medulla

D. Right superior cerebellar artery territory involving the cerebellar hemisphere

E. Left posterior cerebral artery territory involving the dominant occipital lobe

54. A patient with bilateral ACA infarction is awake but minimally initiates speech or movement despite preserved basic motor capacity. Which network dysfunction best explains this behavior?

A. Interruption of lower motor neurons in the facial nucleus as the best mechanistic explanation for these findings

B. Blockade of neuromuscular transmission at the motor endplate as the best mechanistic explanation for these findings

C. Destruction of the primary visual cortex causing cortical blindness as the best mechanistic explanation for these findings

D. Loss of vestibular nuclei causing oscillopsia and vertigo as the best mechanistic explanation for these findings

E. Disruption of medial frontal and anterior cingulate circuits responsible for initiation and motivated behavior

55. A 64-year-old woman has fluent speech with severely impaired comprehension and repetition after a left hemispheric stroke; motor strength is nearly normal. Which vascular distribution is most likely?

A. Right PCA territory affecting the nondominant occipital cortex as the best diagnosis in this clinical setting

B. Superior division of the left MCA affecting frontal motor-language cortex

C. Inferior division of the left middle cerebral artery affecting posterior temporal language cortex

D. Left PICA territory affecting lateral medullary structures as the best diagnosis in this clinical setting

E. Right ACA territory affecting medial frontal leg motor cortex as the best diagnosis in this clinical setting

56. A left MCA stem occlusion produces global aphasia, right hemiplegia, right hemisensory loss, and right homonymous hemianopia. Why is the syndrome more extensive than an isolated division occlusion?

A. The syndrome requires simultaneous bilateral ACA occlusion as the best diagnosis in this clinical setting

B. A proximal MCA stem lesion mainly compromises deep lenticulostriate territory while sparing both cortical divisions

C. The stem occlusion compromises both superior and inferior MCA divisions and often deep perforator flow

D. The stem lesion selectively injures the cerebellar peduncles as the best diagnosis in this clinical setting

E. A proximal MCA stem lesion selectively affects posterior temporal cortex and spares frontal and deep motor pathways

57. A 76-year-old man with severe bilateral carotid stenosis becomes hypotensive during surgery and later has bilateral proximal arm weakness greater than hand weakness. MRI shows borderzone infarcts between ACA and MCA territories. Which mechanism is most likely?

A. Hemodynamic failure in distal arterial territories during systemic hypotension

B. A single embolus to a distal posterior cerebral artery branch

C. Venous sinus thrombosis causing increased intracranial pressure

D. Transient cortical spreading depression from migraine aura

E. Rupture of a thalamic perforator producing a deep hemorrhage

58. A patient with critical carotid disease has recurrent limb-shaking episodes precipitated by standing and relieved by lying down, without epileptiform activity. What management priority is most appropriate?

A. Induce lower blood pressure to reduce the frequency of attacks as the next step as the preferred approach in this clinical setting

B. Treat as focal epilepsy with escalating antiseizure medication alone as the preferred approach in this clinical setting

C. Evaluate for severe hemodynamic cerebrovascular insufficiency and optimize cerebral perfusion while addressing the causative arterial disease

D. Diagnose psychogenic events because posture changes the symptoms in this clinical context as the preferred approach in this clinical setting

E. Use long-term sedatives to suppress involuntary movement at this time as the preferred approach in this clinical setting

59. A 46-year-old woman presents with sudden neck pain followed by right Horner syndrome and transient left arm weakness. CTA shows a tapered narrowing of the right cervical internal carotid artery. What is the most likely diagnosis?

A. Cavernous sinus thrombosis causing bilateral orbital congestion

B. Posterior communicating artery aneurysm causing isolated third-nerve palsy

C. Cervical internal carotid artery dissection as the best diagnosis in this clinical setting

D. Temporal arteritis causing chronic bilateral visual loss

E. Atherosclerotic intracranial MCA stenosis without cervical vessel injury

60. A patient with extracranial carotid dissection has a small ischemic stroke and no contraindication to antithrombotic therapy. Which long-term strategy is generally reasonable?

A. Withhold all antithrombotic therapy because dissection is a bleeding disorder as the preferred approach in this clinical setting

B. Use antithrombotic therapy with either an antiplatelet or anticoagulant approach individualized to the patient, then reassess healing

C. Use prolonged dual antiplatelet therapy for a year regardless of whether follow-up imaging shows arterial healing

D. Treat primarily with corticosteroids because the lesion is inflammatory as the preferred approach in this clinical setting

E. Perform emergency carotid endarterectomy in most dissection for this patient as the preferred approach in this clinical setting

61. A 41-year-old man develops sudden severe occipital neck pain after vigorous exercise, followed by vertigo, dysarthria, and right-sided limb ataxia. CTA shows an irregular tapered stenosis of the left vertebral artery. What is the most likely diagnosis?

A. An isolated vestibular neuritis without cerebrovascular disease

B. A left temporal lobe seizure producing persistent posterior-circulation signs

C. Extracranial vertebral artery dissection causing posterior-circulation ischemia

D. A chronic lacunar syndrome from penetrating-vessel lipohyalinosis

E. A spontaneous deep putaminal hemorrhage from hypertension

62. A patient with vertebral artery dissection has a small cerebellar infarct and remains neurologically stable. Which management approach is usually appropriate?

A. Use corticosteroids because the arterial wall lesion is presumed autoimmune as the preferred approach in this clinical setting

B. Use indefinite anticoagulation without interval vascular imaging because recurrent risk remains high after the dissection heals

C. Use antithrombotic therapy and vascular follow-up, reserving intervention for selected recurrent or flow-limiting cases

D. Place an intracranial stent in most dissection regardless of symptoms as the preferred approach in this clinical setting

E. Avoid all antithrombotic therapy until the artery has completely healed as the preferred approach in this clinical setting

63. A 47-year-old woman with hypertension at a young age has a cervical CTA showing alternating stenoses and dilatations of both internal carotid arteries without atherosclerotic plaque. Which diagnosis best fits?

A. Fibromuscular dysplasia in this setting as the best diagnosis in this clinical setting

B. Takayasu arteritis with diffuse inflammatory aortic wall thickening

C. Atherosclerosis with calcified carotid bulb plaque

D. Moyamoya disease with terminal intracranial ICA occlusion

E. Cerebral amyloid angiopathy involving cortical arterioles

64. What additional vascular assessment is reasonable after diagnosing cervicocephalic fibromuscular dysplasia?

A. Assume the disease is confined to one carotid artery and avoid further vascular assessment

B. Screen for involvement of other vascular beds and associated intracranial aneurysm according to clinical context

C. Start lifelong immunosuppression because the lesion is granulomatous vasculitis as the most informative choice for this evaluation

D. Use therapeutic anticoagulation indefinitely even without dissection or ischemia

E. Perform carotid endarterectomy for most beaded segment as the most informative choice for this evaluation

65. A 54-year-old man has two cryptogenic left MCA embolic strokes. CTA repeatedly shows a thin shelf-like projection from the posterior wall of the left carotid bulb without significant atherosclerotic plaque. What is the most likely lesion?

A. Carotid body tumor producing an enhancing bifurcation mass

B. Cervical carotid dissection with a long tapered flame-shaped occlusion

C. Fibromuscular dysplasia with multifocal string-of-beads changes

D. Ulcerated calcific atherosclerotic plaque with high-grade luminal stenosis

E. Carotid web in this setting as the best diagnosis in this clinical setting

66. A patient with recurrent ipsilateral embolic strokes has a clearly implicated carotid web despite antiplatelet therapy. Which management option may be considered at an experienced center?

A. Carotid revascularization by endarterectomy or stenting after individualized multidisciplinary review

B. Escalate antiplatelet intensity alone because the structural carotid lesion does not change management

C. Switch to long-term anticoagulation without addressing the recurrent embolic source at the carotid bulb

D. Continue the same antiplatelet regimen despite recurrent ipsilateral embolic events and no competing source

E. Perform an intracranial bypass procedure even though the embolic lesion is extracranial at the carotid bulb

67. A 70-year-old man has a nondisabling left hemispheric ischemic stroke. CTA shows 80% stenosis of the left cervical internal carotid artery at the bulb. He is medically stable and has no major surgical contraindication. What is the preferred secondary-prevention strategy?

A. Delay carotid treatment for several months in most nondisabling stroke

B. Early carotid endarterectomy plus intensive medical therapy when perioperative risk is acceptable

C. Indefinite anticoagulation alone because carotid plaque is a cardiac embolic source

D. Perform intracranial stenting of the MCA despite the cervical lesion as the preferred approach in this clinical setting

E. Observe without revascularization because the stroke has already occurred

68. Which factor most favors carotid endarterectomy over carotid artery stenting for routine treatment of symptomatic carotid stenosis?

A. A restenotic lesion after prior endarterectomy with hostile neck anatomy

B. A history of severe radiation injury making open neck surgery hazardous

C. A heavily calcified tortuous aortic arch that complicates endovascular access

D. A need to avoid any incision regardless of comparative stroke risk

E. Older age with anatomy suitable for surgery and acceptable perioperative surgical risk

69. A 59-year-old woman has symptomatic 75% internal carotid stenosis but previously received extensive neck radiation and has severe scarring around the carotid bifurcation. Which revascularization strategy deserves particular consideration?

A. Long-term thrombolytic infusion through the carotid artery

B. Open endarterectomy is preferred regardless of hostile neck anatomy

C. Emergency intracranial bypass for an extracranial carotid bulb lesion

D. Carotid artery stenting as an alternative to high-risk open surgery

E. No revascularization because prior radiation eliminates stroke recurrence risk

70. Before carotid artery stenting, which medication strategy is generally necessary?

A. Stop statin therapy before the procedure to reduce concern for peri-procedural plaque instability

B. Use peri-procedural anticoagulation alone instead of antiplatelet therapy to prevent acute stent thrombosis

C. Hold antiplatelet therapy before stenting to reduce access-site bleeding, then restart it several days later

D. Use warfarin monotherapy because thrombin inhibition is sufficient to prevent platelet-mediated stent thrombosis

E. Provide appropriate peri-procedural antiplatelet therapy to reduce acute stent thrombosis

71. A 64-year-old woman has a recent TIA attributed to 80% intracranial M1 stenosis. There is no atrial fibrillation. Which initial strategy is best supported?

A. Carotid endarterectomy despite the lesion being intracranial as the preferred approach in this clinical setting

B. No antithrombotic treatment because stenosis itself is not embolic as the preferred approach in this clinical setting

C. Primary intracranial stenting for most symptomatic stenosis before medical therapy

D. Long-term warfarin because intracranial atherosclerosis is an anticoagulant indication

E. Aggressive medical management with antiplatelet therapy and intensive control of vascular risk factors

72. A patient with severe symptomatic intracranial atherosclerotic stenosis asks why stenting is not the routine first treatment. What is the best explanation?

A. Intracranial arteries is unlikely to technically be stented under any circumstances as the preferred approach in this clinical setting

B. Assume aggressive medical therapy rapidly reverses the fixed stenosis, making stenting unnecessary for anatomic reasons

C. Avoid intracranial stenting because the antiplatelet therapy required for a stent is contraindicated after ischemic stroke

D. Randomized evidence showed substantial periprocedural risk with routine intracranial stenting compared with aggressive medical therapy

E. Avoid intracranial stenting mainly because delayed restenosis is the principal risk rather than early periprocedural stroke or hemorrhage

73. A 61-year-old man has a high-risk TIA with transient aphasia and right arm weakness lasting 35 minutes. CT excludes hemorrhage, and there is no atrial fibrillation. He presents 3 hours after symptom onset. Which early antithrombotic strategy is appropriate?

A. Short-course dual antiplatelet therapy when no contraindication exists, followed by single antiplatelet therapy

B. Warfarin immediately even before a cardioembolic source is identified as the preferred approach in this clinical setting

C. Full-dose heparin for all high-risk TIAs regardless of mechanism as the preferred approach in this clinical setting

D. No antithrombotic treatment because the symptoms have resolved as the preferred approach in this clinical setting

E. Long-term triple antiplatelet therapy to maximize platelet inhibition as the preferred approach in this clinical setting

74. What is the main reason dual antiplatelet therapy is limited to a short course after selected minor stroke or high-risk TIA?

A. The drugs become completely ineffective after the first 24 hours as the preferred approach in this clinical setting

B. The early reduction in ischemic recurrence is front-loaded, whereas bleeding risk accumulates with prolonged dual therapy

C. Long-term dual therapy prevents statins from lowering LDL cholesterol as the preferred approach in this clinical setting

D. Aspirin causes standard thrombocytopenia if combined for more than three days as the preferred approach in this clinical setting

E. Clopidogrel converts to an anticoagulant after several weeks as the preferred approach in this clinical setting

75. A 67-year-old woman with an atherosclerotic ischemic stroke has an LDL cholesterol of 132 mg/dL and no statin intolerance. Which lipid-lowering strategy is most appropriate?

A. Use a low-intensity statin primarily if LDL exceeds 200 mg/dL as the next step as the preferred approach in this clinical setting

B. Treat cholesterol primarily with dietary change after a symptomatic stroke as the preferred approach in this clinical setting

C. Begin high-intensity statin therapy and target substantial LDL reduction, often to below 70 mg/dL in atherosclerotic disease

D. Start warfarin instead of lipid lowering because LDL is a coagulation marker as the preferred approach in this clinical setting

E. Avoid statins because they increase recurrent ischemic stroke risk as the preferred approach in this clinical setting

76. A stroke patient remains above the LDL goal despite adherence to a maximally tolerated high-intensity statin. What is a reasonable next step?

A. Add nonstatin lipid-lowering therapy such as ezetimibe, with further escalation based on residual risk and LDL level

B. Replace lipid therapy with dual anticoagulation as the next step as the preferred approach in this clinical setting

C. Use chronic corticosteroids to lower vascular inflammation as the preferred approach in this clinical setting

D. Stop the statin and accept the elevated LDL because combination therapy has no role

E. Delay additional treatment until another stroke occurs at this time as the preferred approach in this clinical setting

77. A 58-year-old man is seen 2 months after a lacunar stroke. His home blood pressures average 154/92 mm Hg. Which intervention has the greatest importance for recurrent-stroke prevention?

A. Permit chronic systolic pressures above 170 mm Hg to preserve cerebral perfusion as the preferred approach in this clinical setting

B. Use thrombolysis whenever blood pressure exceeds 160 mm Hg as the preferred approach in this clinical setting

C. Avoid antihypertensive treatment because lacunar strokes are unrelated to hypertension

D. Intensify long-term blood-pressure control with an individualized target commonly below 130/80 mm Hg if tolerated

E. Treat blood pressure primarily during the first 24 hours after the stroke as the preferred approach in this clinical setting

78. A patient with recent ischemic stroke is neurologically stable but has persistent hypertension during follow-up. Which management principle is best?

A. Rely on occasional emergency intravenous treatment instead of daily therapy

B. Stop antihypertensives whenever clinic pressure reaches 140 mm Hg as the preferred approach in this clinical setting

C. Treat primarily diastolic pressure and ignore systolic hypertension as the preferred approach in this clinical setting

D. Avoid home monitoring because it is unlikely to inform stroke prevention as the preferred approach in this clinical setting

E. Use sustained outpatient blood-pressure control and address adherence, lifestyle, and medication barriers

79. A 74-year-old woman with nonvalvular atrial fibrillation has a recent small ischemic stroke and no hemorrhagic transformation. Which long-term therapy most effectively reduces cardioembolic recurrence?

A. Aspirin alone because anticoagulation is not effective for atrial fibrillation as the preferred approach in this clinical setting

B. Long-term aspirin plus clopidogrel as a superior substitute for anticoagulation as the preferred approach in this clinical setting

C. No antithrombotic therapy because embolic risk falls after the first stroke as the preferred approach in this clinical setting

D. Oral anticoagulation, usually with a direct oral anticoagulant when no valvular or other contraindication is present

E. Carotid endarterectomy even when no carotid stenosis is present as the preferred approach in this clinical setting

80. A patient with a mild atrial-fibrillation-related ischemic stroke has a small infarct and no hemorrhage. What principle guides when anticoagulation can be started?

A. Wait exactly six months after most ischemic stroke before anticoagulating for this patient given the clinical findings and context described

B. Start full anticoagulation during the first minute of most stroke regardless of infarct size given the clinical findings and context described

C. Balance the immediate risk of recurrent embolism against hemorrhagic transformation risk, allowing earlier treatment after small mild infarcts than after large infarcts

D. Use dual antiplatelet therapy indefinitely instead of ever starting anticoagulation given the clinical findings and context described

E. Base timing primarily on the patient's age and ignore imaging in this clinical context given the clinical findings and context described

81. A 69-year-old man has an embolic-appearing cortical infarct. Inpatient telemetry and a 24-hour Holter show no atrial fibrillation, and no other cause is found. Which next diagnostic strategy is most appropriate?

A. Use prolonged ambulatory rhythm monitoring to increase detection of paroxysmal atrial fibrillation

B. Conclude that atrial fibrillation has been adequately excluded after normal telemetry and a 24-hour Holter

C. Repeat carotid imaging at short intervals instead of extending rhythm monitoring despite normal vascular studies

D. Begin empirical anticoagulation for presumed occult atrial fibrillation without documenting the arrhythmia

E. Repeat transthoracic echocardiography alone rather than pursue longer-duration rhythm surveillance

82. What is the practical value of an implantable cardiac monitor after selected cryptogenic strokes?

A. It directly images the left atrial appendage for thrombus given the clinical findings and context described

B. It measures carotid plaque thickness during daily activity given the clinical findings and context described

C. It provides long-duration rhythm surveillance that can detect infrequent asymptomatic atrial fibrillation episodes

D. It replaces all echocardiography and vascular imaging in stroke evaluation given the clinical findings and context described

E. It prevents atrial fibrillation by pacing the ventricle continuously given the clinical findings and context described

83. A 43-year-old man has a nonlacunar embolic ischemic stroke. Extensive vascular and rhythm evaluation reveals no alternative cause. Transesophageal echocardiography shows a patent foramen ovale with large right-to-left shunt and atrial septal aneurysm. Which strategy is reasonable?

A. Use antiplatelet therapy alone without considering closure despite high-risk PFO anatomy and no alternative cause

B. Start indefinite warfarin because any PFO with a shunt should be treated as a proven cardioembolic source

C. Consider transcatheter PFO closure plus antiplatelet therapy after multidisciplinary confirmation that the PFO is likely causal

D. Treat the PFO as incidental because paradoxical embolism is not a recognized mechanism of ischemic stroke

E. Recommend PFO closure on the basis of the echocardiographic finding alone without considering age or competing causes

84. Which feature makes a PFO more likely to be causally related to a cryptogenic stroke?

A. Advanced age with multiple strong atherosclerotic risk factors given the clinical findings and context described

B. Persistent atrial fibrillation documented before the stroke given the clinical findings and context described

C. Severe ipsilateral carotid stenosis explaining the cerebral infarct given the clinical findings and context described

D. Large right-to-left shunt or associated atrial septal aneurysm in a younger patient without competing vascular causes

E. A classic lacunar infarct from hypertensive small-vessel disease given the clinical findings and context described

85. A 62-year-old man with a recent anterior MI has an embolic stroke. Echocardiography shows a mobile apical left ventricular thrombus. Which secondary-prevention strategy is most appropriate once neurologically safe?

A. Use antiplatelet therapy alone because the ventricular thrombus is expected to organize without further embolization

B. Use dual antiplatelet therapy instead of anticoagulation because the embolic source followed myocardial infarction

C. Therapeutic anticoagulation for the ventricular thrombus, with duration guided by cardiac recovery and repeat imaging

D. Treat the patient with carotid revascularization despite no evidence that the embolic source is cervical carotid disease

E. Defer antithrombotic therapy until repeat echocardiography confirms that the ventricular thrombus persists

86. Why can a large anterior myocardial infarction predispose to left ventricular thrombus and stroke?

A. Myocardial ischemia produces amyloid deposition in cortical arterioles as the best mechanistic explanation for these findings

B. Apical akinesis increases cerebrospinal fluid viscosity and causes lacunes as the best mechanistic explanation for these findings

C. Anterior MI directly narrows the cervical carotid artery through vasospasm as the best mechanistic explanation for these findings

D. The infarct causes cerebral venous sinus thrombosis by increasing intracranial pressure as the best mechanistic explanation for these findings

E. Apical akinesis creates blood stasis over an injured endocardial surface, promoting thrombus formation and systemic embolization

87. A 56-year-old woman with a mechanical mitral valve develops an ischemic stroke after warfarin interruption. Which long-term antithrombotic strategy is required?

A. Use aspirin alone because prosthetic valves are not embolic sources as the preferred approach in this clinical setting

B. Stop all anticoagulation after a first ischemic event in this clinical context as the preferred approach in this clinical setting

C. Resume vitamin K antagonist anticoagulation when neurologically safe because mechanical valves require warfarin-class therapy

D. Switch to a direct oral anticoagulant to reduce intracranial bleeding while maintaining equivalent protection of the mechanical valve

E. Treat with clopidogrel monotherapy as a complete substitute for warfarin as the preferred approach in this clinical setting

88. A patient with a mechanical mitral valve has a large infarct with small hemorrhagic transformation. What is the key management challenge?

A. Use long-term antiplatelet therapy instead of anticoagulation because any hemorrhagic transformation makes future anticoagulation too risky

B. Start a direct oral anticoagulant immediately because it is unlikely to worsen intracranial hemorrhage as the preferred approach in this clinical setting

C. Balance very high valve-thrombosis and embolic risk against the danger of enlarging intracranial bleeding when restarting anticoagulation

D. Control blood pressure and defer anticoagulation until the mechanical valve can be surgically replaced

E. Use thrombolysis daily until the hemorrhage resolves in this clinical context as the preferred approach in this clinical setting

89. A 39-year-old man with fever, a new murmur, and positive blood cultures develops an acute embolic MCA stroke. Echocardiography shows a mitral vegetation. Which statement about intravenous thrombolysis is most appropriate?

A. Treat the stroke with intravenous thrombolysis because the vegetation is small and blood cultures are already positive

B. Infective endocarditis is a major contraindication to routine intravenous thrombolysis because of hemorrhagic complications including mycotic vascular lesions

C. Use intravenous thrombolysis if the embolus is presumed septic because infected thrombi may be fibrin rich

D. Treat endocarditis-associated ischemic stroke like routine cardioembolism if the head CT shows no hemorrhage

E. Proceed with thrombolysis after starting antibiotics because antimicrobial therapy immediately normalizes vascular bleeding risk

90. A patient with infective endocarditis has a proximal large-vessel occlusion and severe deficits. Which reperfusion option may still be considered at an experienced center?

A. Mechanical thrombectomy after individualized assessment despite the thrombolysis contraindication

B. Avoid thrombectomy because manipulation of septic emboli is presumed to create unacceptable hemorrhagic risk

C. Routine full-dose systemic thrombolysis followed by anticoagulation given the clinical findings and context described

D. Emergency carotid endarterectomy regardless of occlusion location given the clinical findings and context described

E. High-dose steroids as the primary method of clot removal given the clinical findings and context described

91. A 35-year-old woman with systemic lupus erythematosus has recurrent arterial ischemic strokes, persistent lupus anticoagulant, and high-titer anticardiolipin antibodies. Which diagnosis is most likely?

A. Mitochondrial encephalopathy as the primarily cause of arterial thrombosis

B. Thrombotic thrombocytopenic purpura without laboratory evidence of hemolysis

C. Isolated migraine aura with incidental autoantibodies as the best diagnosis in this clinical setting

D. Cerebral amyloid angiopathy in a young adult as the best diagnosis in this clinical setting

E. Antiphospholipid syndrome in this setting as the best diagnosis in this clinical setting

92. A patient with definite antiphospholipid syndrome has had an arterial ischemic stroke. Which long-term therapy is commonly favored for secondary prevention?

A. No antithrombotic therapy because the autoantibodies are primarily laboratory markers

B. Vitamin K antagonist anticoagulation, individualized to the patient's thrombotic and bleeding risk

C. Chronic intravenous thrombolysis is needed to suppress autoantibody production

D. Use aspirin alone after the arterial event because anticoagulation is reserved for venous manifestations of antiphospholipid syndrome

E. Prefer a direct oral anticoagulant over warfarin because its lower intracranial bleeding risk outweighs concerns in high-risk antiphospholipid syndrome

93. A 66-year-old man with metastatic pancreatic cancer develops multifocal acute infarcts in several arterial territories. Telemetry is unrevealing, and echocardiography shows sterile-appearing mitral vegetations. What is the most likely mechanism?

A. Primary demyelination producing vascular-territory lesions as the best diagnosis in this clinical setting

B. Cancer-associated hypercoagulability with nonbacterial thrombotic endocarditis

C. Hypertensive lipohyalinosis affecting one penetrating artery

D. Migraine aura causing diffusion restriction in multiple territories

E. A single carotid plaque embolus explaining bilateral anterior and posterior infarcts

94. A patient with active cancer has recurrent embolic strokes attributed to nonbacterial thrombotic endocarditis. Which management principle is most appropriate?

A. Treat the underlying malignancy and use therapeutic anticoagulation when bleeding risk permits

B. Use antiplatelet therapy alone because sterile valvular vegetations are platelet rich and do not require systemic anticoagulation

C. Give prolonged antibiotics because all valvular vegetations are infectious

D. Avoid anticoagulation because active malignancy is considered a contraindication even when recurrent NBTE embolism is present

E. Perform carotid endarterectomy even without carotid disease as the preferred approach in this clinical setting

95. A 12-year-old child with sickle cell disease develops acute right hemiparesis and aphasia. What disease-specific treatment should be initiated urgently in addition to standard stroke stabilization?

A. Use standard antiplatelet therapy alone because the neurologic deficit is managed the same as an adult atherosclerotic stroke

B. Give simple packed-cell transfusion only when hemoglobin is below 7 g/dL rather than urgently reduce the HbS fraction

C. Increase the hematocrit substantially with transfusion without limiting blood viscosity or the proportion of sickled hemoglobin

D. Wait for confirmatory transcranial Doppler testing before initiating disease-specific acute stroke treatment

E. Rapid red blood cell transfusion, preferably exchange transfusion when feasible, to reduce the proportion of sickled hemoglobin

96. Which screening strategy reduces first-stroke risk in children with sickle cell anemia?

A. Use annual brain MRI alone and reserve transcranial Doppler for children who already have focal neurologic symptoms

B. Use transcranial Doppler to identify high cerebral arterial velocities and institute chronic transfusion therapy when high-risk thresholds are met

C. Use extracranial carotid ultrasound as the primary screening test for the intracranial vasculopathy of sickle cell disease

D. Start chronic aspirin when cerebral arterial velocities rise instead of instituting transfusion-based prevention

E. Use routine EEG screening to identify silent cerebral ischemia and determine the need for chronic transfusion

97. A 32-year-old woman at 24 weeks of pregnancy develops a disabling left MCA ischemic stroke within 2 hours of onset. CT shows no hemorrhage and she otherwise meets reperfusion criteria. Which principle is most appropriate?

A. Perform cesarean delivery before any neurologic intervention regardless of gestational age

B. Pregnancy alone should not automatically exclude potentially beneficial reperfusion therapy for a disabling ischemic stroke

C. primarily aspirin can be used acutely because all reperfusion crosses the placenta as the preferred approach in this clinical setting

D. Pregnancy is an absolute contraindication to both thrombolysis and thrombectomy as the preferred approach in this clinical setting

E. Delay all stroke treatment until fetal viability is reached in this clinical context as the preferred approach in this clinical setting

98. A pregnant patient has an ICA terminus occlusion with severe deficits and favorable imaging. What is the most appropriate systems response?

A. Use primarily prophylactic-dose heparin because mechanical treatment is contraindicated

B. Prefer medical reperfusion alone and defer thrombectomy because fetal radiation exposure is considered prohibitive during pregnancy

C. Activate endovascular stroke treatment promptly while coordinating obstetric and anesthesia expertise

D. Transfer primarily to an obstetric unit without stroke-intervention capability

E. Wait for spontaneous recanalization to avoid any maternal procedure as the preferred approach in this clinical setting

99. A 29-year-old woman 10 days postpartum develops progressive headache, a focal seizure, and left leg weakness. CT venography shows a superior sagittal sinus filling defect. What is the diagnosis?

A. Aneurysmal subarachnoid hemorrhage from a posterior communicating artery aneurysm

B. Primary tension headache related to sleep deprivation as the best diagnosis in this clinical setting

C. Cerebral venous sinus thrombosis in this setting as the best diagnosis in this clinical setting

D. Hypertensive lacunar infarction of the internal capsule as the best diagnosis in this clinical setting

E. Carotid dissection causing a unilateral retinal syndrome as the best diagnosis in this clinical setting

100. Which imaging study is most useful for confirming suspected cerebral venous sinus thrombosis when routine CT is nondiagnostic?

A. Repeat noncontrast head CT because evolving venous thrombosis is reliably diagnosed by parenchymal changes alone

B. CT venography or MR venography demonstrating impaired venous sinus opacification or flow

C. Obtain catheter cerebral arteriography as the routine first confirmatory test before any noninvasive venous imaging

D. Use carotid duplex ultrasonography because extracranial arterial flow can serve as a surrogate for dural sinus patency

E. Use routine brain MRI without dedicated venographic sequences and infer sinus thrombosis from edema pattern alone

101. A 36-year-old woman has cerebral venous thrombosis with a small hemorrhagic venous infarct and no contraindication to anticoagulation. What is the recommended acute treatment?

A. Give platelet transfusion as the primary treatment for the thrombosed sinus

B. Perform carotid endarterectomy to restore venous drainage as the preferred approach in this clinical setting

C. Therapeutic anticoagulation despite the hemorrhagic venous infarct

D. Withhold anticoagulation because any intracranial blood makes it generally contraindicated

E. Use primarily acetazolamide because anticoagulation is unlikely to improve venous thrombosis

102. A patient with cerebral venous thrombosis deteriorates despite adequate anticoagulation and develops coma from worsening venous congestion. What additional treatment may be considered at an expert center?

A. Continue anticoagulation alone despite coma and progressive mass effect because invasive rescue therapy has no established role

B. Endovascular therapy or decompressive surgery in carefully selected severe rescue situations

C. Place an arterial intracranial stent to improve venous outflow despite the thrombosis being in the venous circulation

D. Stop anticoagulation when neurologic deterioration occurs because progression implies anticoagulant treatment failure

E. Use systemic thrombolysis as the routine rescue strategy without catheter-based selection or surgical assessment

103. A 30-year-old pregnant woman with cerebral venous thrombosis requires anticoagulation. Which agent is generally preferred during pregnancy?

A. Low-molecular-weight heparin in this setting given the clinical findings and context described

B. Aspirin alone because anticoagulants are contraindicated in pregnancy

C. A direct oral factor Xa inhibitor because fetal safety is fully established

D. Warfarin throughout pregnancy because it does not cross the placenta

E. No antithrombotic therapy until after delivery given the clinical findings and context described

104. After delivery, a woman with pregnancy-associated cerebral venous thrombosis asks about duration of anticoagulation. What is the best general approach?

A. Switch to dual antiplatelet therapy because venous thrombosis is unlikely to recur postpartum as the preferred approach in this clinical setting

B. Use intermittent thrombolysis during future pregnancies instead of prophylaxis as the preferred approach in this clinical setting

C. Continue anticoagulation for a finite course based on whether the event was provoked and on persistent thrombophilic risk

D. Continue lifelong anticoagulation in most pregnancy-associated case regardless of risk

E. Stop anticoagulation immediately after the placenta is delivered as the preferred approach in this clinical setting

105. A 43-year-old woman develops recurrent thunderclap headaches over one week. CTA shows multifocal segmental narrowing and dilatation of several intracranial arteries. She recently started a serotonergic drug. What is the most likely diagnosis?

A. Moyamoya disease with chronic terminal ICA occlusion and basal collaterals

B. Atherosclerotic intracranial stenosis confined to one plaque-bearing segment

C. Fibromuscular dysplasia limited to the renal arteries as the best diagnosis in this clinical setting

D. Reversible cerebral vasoconstriction syndrome as the best diagnosis in this clinical setting

E. Primary angiitis of the central nervous system with steadily progressive encephalopathy

106. Which management step is most appropriate for suspected reversible cerebral vasoconstriction syndrome?

A. Perform intracranial stenting of most narrowed segment at this time as the best diagnosis in this clinical setting, based on the information provided

B. Remove vasoactive triggers, control severe symptoms and blood pressure, and avoid unnecessary glucocorticoids while monitoring for ischemic or hemorrhagic complications

C. Use lifelong anticoagulation because all vasoconstriction is thrombotic in this clinical context as the best diagnosis in this clinical setting

D. Start high-dose glucocorticoids routinely because they accelerate vascular recovery as the best diagnosis in this clinical setting

E. Induce systemic hypotension to force vasodilation as the next step as the best diagnosis in this clinical setting, based on the information provided

107. A 52-year-old man has weeks of headache, cognitive decline, and recurrent multifocal ischemic lesions. CSF shows lymphocytic pleocytosis and elevated protein. Angiography shows multifocal narrowing, and no systemic vasculitis is found. Which diagnosis is most likely?

A. Embolic stroke from isolated atrial fibrillation without inflammatory findings

B. Hypertensive arteriopathy confined to deep perforating vessels

C. Primary angiitis of the central nervous system as the best diagnosis in this clinical setting

D. Reversible cerebral vasoconstriction syndrome with normal inflammatory studies

E. Cerebral amyloid angiopathy causing primarily lobar hemorrhage

108. When primary CNS vasculitis is strongly suspected but the diagnosis remains uncertain, which step can provide the most definitive evidence?

A. Lumbar spine MRI because cerebral vessels is unlikely to be biopsied

B. Targeted brain and leptomeningeal biopsy of an abnormal region when feasible

C. Serum troponin measurement because PACNS is a cardiac disease

D. Routine EEG because vasculitis is defined by seizure activity

E. A normal carotid ultrasound because it excludes intracranial inflammation

109. A 28-year-old woman has recurrent TIAs and ischemic strokes. Angiography shows bilateral narrowing of the terminal internal carotid arteries with a network of fine basal collateral vessels. What is the diagnosis?

A. Fibromuscular dysplasia of the extracranial cervical carotids

B. Moyamoya arteriopathy in this setting as the best diagnosis in this clinical setting

C. Isolated posterior reversible encephalopathy syndrome

D. Cerebral venous sinus thrombosis with collateral cortical veins

E. A single embolic MCA occlusion with normal terminal ICAs

110. A symptomatic adult with moyamoya has recurrent ischemic events and impaired cerebrovascular reserve. Which treatment offers the best chance of durable risk reduction?

A. Long-term corticosteroids because moyamoya is primarily inflammatory

B. Intracranial stenting of all stenotic segments as routine first-line therapy

C. Aggressive blood-pressure lowering to minimize flow through collateral vessels

D. No intervention because collateral vessels eliminate future ischemic risk

E. Surgical revascularization using direct or indirect bypass techniques at an experienced center

111. A 63-year-old man presents with a 24-mL spontaneous putaminal hemorrhage and systolic blood pressure of 188 mm Hg. He is awake with moderate hemiparesis. Which blood-pressure approach is most appropriate?

A. Avoid acute blood-pressure lowering because preserving perihematomal perfusion is more important than reducing expansion risk at this pressure

B. Reduce systolic pressure immediately to below 90 mm Hg as the preferred approach in this clinical setting

C. Use intermittent large boluses that cause rapid pressure swings as the preferred approach in this clinical setting

D. Avoid blood-pressure treatment until the hematoma resolves on MRI as the preferred approach in this clinical setting

E. Lower systolic pressure smoothly toward approximately 140 mm Hg while avoiding overshoot below about 130 mm Hg

112. Which blood-pressure characteristic is particularly desirable during acute ICH management?

A. Smooth, sustained control with minimal variability rather than repeated peaks and abrupt drops

B. Persistent severe hypertension to maintain perihematomal perfusion as the preferred approach in this clinical setting

C. Wide oscillations because they improve clot contraction as the preferred approach in this clinical setting

D. Profound hypotension to eliminate all risk of expansion as the preferred approach in this clinical setting

E. No monitoring once the first antihypertensive dose is given as the preferred approach in this clinical setting

113. A 75-year-old man taking warfarin presents with a cerebellar intracerebral hemorrhage. INR is 3.1. What is the most appropriate immediate reversal strategy?

A. Give fresh frozen plasma alone and omit vitamin K because it acts too slowly

B. Wait for the INR to fall spontaneously before reversing anticoagulation

C. Administer four-factor prothrombin complex concentrate plus intravenous vitamin K

D. Administer idarucizumab because it reverses vitamin K antagonists

E. Administer andexanet alfa because it is the specific antidote to warfarin

114. Why is vitamin K given with prothrombin complex concentrate in warfarin-associated ICH?

A. PCC has no effect on coagulation until vitamin K is metabolized as the best mechanistic explanation for these findings

B. Vitamin K reverses platelet inhibitors rather than warfarin as the best mechanistic explanation for these findings

C. The combination is used primarily to prevent seizures after hemorrhage as the best mechanistic explanation for these findings

D. PCC provides immediate factor replacement, while vitamin K restores endogenous factor synthesis for sustained correction

E. Vitamin K directly dissolves the intracerebral hematoma for this patient as the best mechanistic explanation for these findings

115. A 72-year-old woman taking dabigatran develops a spontaneous lobar intracerebral hemorrhage shortly after her last dose. Which reversal agent is specific for dabigatran?

A. Andexanet alfa for this patient given the clinical findings and context described

B. Vitamin K at this time given the clinical findings and context described

C. Protamine sulfate in this clinical context

D. Idarucizumab in this setting given the clinical findings and context described

E. Four-factor PCC as the drug-specific antidote

116. A patient with dabigatran-associated ICH has severe renal failure and persistent drug effect after initial reversal. Which additional modality can remove dabigatran because of its relatively low protein binding?

A. Administer additional four-factor prothrombin complex concentrate as the preferred method to physically remove dabigatran

B. Use repeated activated-charcoal doses despite the last dabigatran ingestion occurring many hours earlier

C. Hemodialysis in selected refractory circumstances because dabigatran has relatively low protein binding and can be removed by dialysis

D. Use plasma exchange because removing plasma proteins is the most efficient way to clear dabigatran

E. Perform exchange transfusion because replacing circulating blood is more effective than dialysis for dabigatran clearance

117. A 79-year-old man taking apixaban presents with a large spontaneous intracerebral hemorrhage 3 hours after his last dose. Which reversal approach is most directly targeted to the anticoagulant mechanism?

A. Delay reversal because direct oral anticoagulants is unlikely to be counteracted

B. Use protamine because apixaban is a form of low-molecular-weight heparin

C. Use idarucizumab because it binds all direct oral anticoagulants as the preferred approach in this clinical setting

D. Use vitamin K because apixaban blocks vitamin K recycling as the preferred approach in this clinical setting

E. Use a factor Xa inhibitor reversal strategy such as andexanet alfa when appropriate and available

118. What is the most important timing principle in a life-threatening factor Xa inhibitor-associated ICH?

A. Treat primarily with platelet transfusion because factor Xa inhibitors act on platelets given the clinical findings and context described

B. Normalize LDL cholesterol before addressing anticoagulation in this clinical context given the clinical findings and context described

C. Reverse clinically significant anticoagulant effect promptly while simultaneously controlling blood pressure and arranging neurocritical care

D. Delay reversal until the exact embolic indication for apixaban is forgotten given the clinical findings and context described

E. Wait for repeat CT to show expansion before reversing anticoagulation given the clinical findings and context described

119. A 74-year-old woman taking aspirin has a spontaneous deep intracerebral hemorrhage and is not scheduled for emergency neurosurgery. Which statement about platelet transfusion is most accurate?

A. Give platelet transfusion to reduce hematoma expansion because the patient was taking aspirin before the hemorrhage

B. Give desmopressin plus platelet transfusion routinely to all aspirin-associated ICH regardless of procedural plans

C. Routine platelet transfusion is not beneficial and may be harmful in aspirin-associated spontaneous ICH without an operative indication

D. Transfuse platelets whenever aspirin exposure is documented, even when no emergency neurosurgical procedure is planned

E. Use platelet transfusion as a general reversal strategy for antiplatelet-associated ICH because laboratory platelet function is reduced

120. A patient with aspirin-associated ICH is going urgently to neurosurgery for hematoma evacuation. How should antiplatelet-related hemostasis be approached?

A. Coordinate individualized perioperative reversal or hemostatic measures with neurosurgery rather than applying the nonoperative platelet-transfusion rule blindly

B. Apply the nonoperative evidence against platelet transfusion unchanged even when an emergency neurosurgical procedure is planned

C. Use warfarin to restore platelet function before surgery at this time as the preferred approach in this clinical setting

D. Give systemic thrombolysis to counter aspirin before incision in this clinical context as the preferred approach in this clinical setting

E. Delay emergency surgery for several days to allow aspirin-related platelet dysfunction to recover spontaneously

121. A 68-year-old man has a 4-cm cerebellar hemorrhage with worsening somnolence, brainstem compression, and hydrocephalus. What is the best next step?

A. Manage with close neurocritical observation alone because the patient remains spontaneously breathing

B. Treat the hydrocephalus with ventricular drainage alone and defer posterior fossa decompression despite brainstem compression

C. Use hyperosmolar therapy alone and postpone surgery unless fixed pupillary abnormalities appear

D. Urgent posterior fossa surgical evacuation with management of hydrocephalus

E. Start therapeutic anticoagulation because the hemorrhage may have originated from a venous infarct

122. Why is cerebellar hemorrhage treated more aggressively surgically than many similarly sized supratentorial hemorrhages?

A. Cerebellar hematomas are treated surgically mainly because posterior-fossa arterial collateral pressure makes rebleeding more frequent

B. The cerebellum directly controls systemic coagulation factor synthesis as the best mechanistic explanation for these findings

C. The confined posterior fossa allows relatively small increases in volume to compress the brainstem and obstruct CSF pathways

D. Cerebellar blood is more toxic because it contains higher iron concentrations as the best mechanistic explanation for these findings

E. Posterior-fossa hematomas should be evacuated because their surgical accessibility is favorable even in the absence of mass effect

123. A 66-year-old man with long-standing hypertension develops acute left hemiplegia and hemisensory loss. CT shows a right putaminal hemorrhage. Which small-vessel pathology is most closely associated with this location?

A. Aneurysmal SAH from the anterior communicating artery

B. Venous sinus thrombosis causing a parasagittal hemorrhagic infarct

C. Rupture of a cortical developmental venous anomaly

D. Hypertensive arteriopathy of deep perforating vessels

E. Cerebral amyloid angiopathy confined to superficial cortical vessels

124. Which long-term intervention most effectively lowers recurrent deep hypertensive ICH risk?

A. Routine prophylactic platelet transfusion

B. Long-term dual antiplatelet therapy as the preferred approach in this clinical setting

C. Chronic high-dose corticosteroids as the preferred approach in this clinical setting

D. Repeated lumbar puncture to lower intracranial pressure

E. Sustained blood-pressure control as the preferred approach in this clinical setting

125. An 81-year-old woman with mild cognitive impairment has a spontaneous right parietal lobar hemorrhage. MRI shows numerous strictly lobar microbleeds and cortical superficial siderosis. Which diagnosis is most likely?

A. Moyamoya disease causing terminal ICA narrowing as the best diagnosis in this clinical setting

B. Cerebral amyloid angiopathy in this setting as the best diagnosis in this clinical setting

C. Hypertensive arteriopathy as the sole explanation for strictly lobar hemorrhage markers

D. Multiple sclerosis causing cortical microhemorrhages as the best diagnosis in this clinical setting

E. Cerebral venous sinus thrombosis without venous imaging abnormality

126. A patient with probable cerebral amyloid angiopathy has no compelling indication for antithrombotic therapy. Which prevention priority is most appropriate?

A. Optimize blood pressure and avoid unnecessary antithrombotic exposure that could increase lobar hemorrhage risk

B. Use dual antiplatelet therapy to prevent recurrent transient focal neurologic episodes attributed to cortical ischemia

C. Maintain higher chronic blood pressure to preserve cortical perfusion in regions affected by amyloid angiopathy

D. Begin anticoagulation because recurrent lobar hemorrhage reflects embolic occlusion of fragile cortical vessels

E. Treat stereotyped transient focal episodes as TIAs and escalate antithrombotic therapy without reviewing blood-sensitive MRI

127. A 72-year-old woman has a sudden brief episode of spreading tingling from the right fingers to the face followed by a small left convexity subarachnoid hemorrhage on MRI, without aneurysm. She has cortical superficial siderosis. What is the most likely underlying disorder?

A. Cerebral amyloid angiopathy with a convexity subarachnoid hemorrhage and transient focal neurologic episode

B. Bacterial meningitis causing diffuse basal cisternal exudate as the best diagnosis in this clinical setting

C. Aneurysmal subarachnoid hemorrhage from the basilar tip as the best diagnosis in this clinical setting

D. Carotid dissection causing isolated retinal ischemia as the best diagnosis in this clinical setting

E. Hypertensive deep perforator rupture into the basal ganglia as the best diagnosis in this clinical setting

128. A patient with CAA-related transient focal neurologic episodes is mistakenly treated as having recurrent TIAs. What is the main danger of escalating antithrombotic therapy?

A. Escalating antithrombotic therapy may suppress superficial bleeding and reduce recurrence of cortical superficial siderosis

B. It converts amyloid angiopathy into hypertensive arteriopathy

C. It may increase the risk of recurrent lobar or superficial intracranial bleeding

D. Antithrombotic therapy mainly increases hemorrhage by provoking reversible cerebral vasoconstriction in amyloid-affected arteries

E. It prevents MRI from detecting future microbleeds as the preferred approach in this clinical setting

129. A 27-year-old man has a seizure and small lobar hemorrhage. MRI and angiography show a compact nidus with feeding arteries and early draining veins. What is the diagnosis?

A. Hypertensive perforator microaneurysm in the basal ganglia

B. Dural venous sinus thrombosis without an arteriovenous connection

C. Cavernous malformation with no high-flow shunt

D. Developmental venous anomaly without an arterial component

E. Brain arteriovenous malformation as the best diagnosis in this clinical setting

130. How should an unruptured brain AVM generally be managed?

A. Use individualized multidisciplinary assessment of hemorrhage risk, AVM anatomy, age, symptoms, and treatment risk rather than a universal intervention rule

B. Use lifelong anticoagulation to prevent AVM rupture in this clinical context given the clinical findings and context described

C. Favor observation because procedural risk exceeds natural-history risk for most unruptured AVMs regardless of lesion anatomy

D. Resect most unruptured AVM immediately regardless of location or grade for this patient given the clinical findings and context described

E. Treat primarily with thrombolysis if a feeding artery thromboses at this time given the clinical findings and context described

131. A 52-year-old woman develops the worst headache of her life, vomiting, and brief loss of consciousness. CT shows diffuse basal cisternal subarachnoid blood. What is the most likely cause?

A. A lacunar infarct of the internal capsule as the best diagnosis in this clinical setting

B. Rupture of an intracranial saccular aneurysm as the best diagnosis in this clinical setting

C. Hypertensive rupture of a deep lenticulostriate arteriole into the putamen

D. Cerebral amyloid angiopathy causing an isolated lobar hemorrhage

E. A chronic subdural hematoma from bridging-vein tearing

132. Why does aneurysmal rupture produce prominent basal cisternal subarachnoid blood?

A. Cortical amyloid deposition preferentially ruptures arteries beneath the midbrain as the best mechanistic explanation for these findings

B. Cerebellar edema mechanically squeezes blood from intact arteries as the best mechanistic explanation for these findings

C. High-pressure arterial bleeding enters the CSF-filled subarachnoid space surrounding the circle of Willis and basal arteries

D. Venous hypertension forces blood through the choroid plexus into the ventricles primarily as the best mechanistic explanation for these findings

E. A deep perforator intracerebral hemorrhage commonly dissects into the basal cisterns and produces the same SAH pattern

133. A patient with aneurysmal SAH has the aneurysm secured on day 1 and is neurologically stable. Which medication should be given routinely to improve neurologic outcome?

A. Use prophylactic intravenous magnesium as the routine medication proven to improve neurologic outcome after SAH

B. Enteral nimodipine given routinely after aneurysmal SAH to improve neurologic outcome and reduce the impact of delayed cerebral ischemia

C. Use long-term dual antiplatelet therapy to prevent delayed cerebral ischemia after the aneurysm is secured

D. Use routine therapeutic heparin infusion to prevent vasospasm-related arterial thrombosis

E. Use prophylactic high-dose corticosteroids to reduce meningeal inflammation and delayed ischemia

134. What is the principal pathophysiologic reason delayed cerebral ischemia after SAH is not equivalent to angiographic vasospasm alone?

A. Delayed ischemia is caused exclusively by hydrocephalus after most SAH for this patient as the best mechanistic explanation for these findings

B. Microvascular dysfunction, cortical spreading depolarizations, impaired autoregulation, inflammation, and macrovascular narrowing can all contribute

C. primarily seizures cause neurologic decline after the aneurysm is secured as the next step as the best mechanistic explanation for these findings

D. Treat angiographic vasospasm as an epiphenomenon that does not materially contribute to delayed cerebral ischemia

E. Delayed ischemia is simply recurrent aneurysm rupture in all cases at this time as the best mechanistic explanation for these findings

135. A patient with aneurysmal SAH becomes progressively drowsy. CT shows acute enlargement of the lateral and third ventricles. What is the most appropriate treatment?

A. Urgently place an external ventricular drain to treat symptomatic acute hydrocephalus and provide controlled CSF diversion

B. Treat with hyperosmolar therapy alone because ventricular enlargement is caused primarily by diffuse cerebral edema

C. Use systemic anticoagulation to improve CSF circulation by reducing intraventricular fibrin burden

D. Observe with serial examinations because ventricular enlargement usually resolves after aneurysm securing without drainage

E. Use acetazolamide as the definitive acute therapy to suppress CSF production in a deteriorating patient

136. How does subarachnoid blood cause communicating hydrocephalus?

A. Blood products impair CSF resorption at arachnoid granulations and can obstruct CSF pathways

B. Aneurysm rupture causes the choroid plexus to stop producing CSF as the best mechanistic explanation for these findings

C. Subarachnoid blood increases CSF production by the choroid plexus, making overproduction the principal cause of hydrocephalus

D. Cortical ischemia directly shrinks the ventricles and traps CSF as the best mechanistic explanation for these findings

E. Vasospasm of the carotid artery blocks CSF flow through the foramen magnum

137. A 49-year-old woman has an aneurysmal SAH from a ruptured anterior communicating artery aneurysm. After stabilization, what is the most important definitive step to reduce early rebleeding risk?

A. Use aspirin alone because platelet inhibition seals the aneurysm neck

B. Use blood-pressure control and short-term antifibrinolytic therapy alone because the rupture site usually seals without urgent aneurysm occlusion

C. Induce hypertension to tamponade the ruptured aneurysm given the clinical findings and context described

D. Wait several weeks for vasospasm risk to pass before treating the aneurysm

E. Secure the ruptured aneurysm promptly by endovascular coiling or microsurgical clipping

138. Why is the risk of rebleeding highest before an aneurysm is definitively secured?

A. Vasospasm mechanically tears most ruptured aneurysm again at this time as the best mechanistic explanation for these findings

B. The aneurysm wall immediately becomes thicker after rupture and is unlikely to hold blood as the best mechanistic explanation for these findings

C. The initial rupture site is temporarily sealed by a fragile clot that can be disrupted by arterial pressure and fibrinolysis

D. Rely on elevated CSF pressure to stabilize the rupture site sufficiently that definitive aneurysm treatment can be delayed

E. Platelets is unlikely to enter the subarachnoid space under any circumstance as the best mechanistic explanation for these findings

139. On day 7 after aneurysmal SAH, a patient develops new aphasia and right arm weakness. CT shows no rebleeding, and CTA demonstrates severe left MCA vasospasm. What is the most appropriate next step after ensuring adequate volume status?

A. Stop nimodipine because vasospasm proves it has failed in this clinical context as the preferred approach in this clinical setting

B. Induce blood-pressure augmentation as clinically appropriate and consider endovascular rescue therapy if deficits persist

C. Restrict fluids and lower blood pressure aggressively to prevent rebleeding as the preferred approach in this clinical setting

D. Begin full-dose anticoagulation to dissolve the constricted arterial segment as the preferred approach in this clinical setting

E. Start prophylactic corticosteroids as the primary treatment as the preferred approach in this clinical setting

140. What causes large-artery narrowing after aneurysmal SAH?

A. Oxyhemoglobin and blood-breakdown products trigger prolonged smooth-muscle constriction, endothelial dysfunction, and inflammatory signaling in exposed arteries

B. Platelet deficiency prevents arterial walls from maintaining diameter in this clinical context as the best mechanistic explanation for these findings

C. Venous thrombosis directly compresses most basal artery from outside at this time as the best mechanistic explanation for these findings

D. The aneurysm mechanically pulls adjacent arteries closed as it shrinks for this patient as the best mechanistic explanation for these findings

E. CSF loss causes arteries to collapse because intracranial pressure becomes negative as the best mechanistic explanation for these findings

141. A 65-year-old man has pulsatile tinnitus and progressive cognitive slowing. Angiography shows meningeal arterial feeders draining directly into a dural venous sinus with retrograde cortical venous reflux. What is the diagnosis?

A. Cerebral venous sinus thrombosis without any arteriovenous connection

B. Cavernous malformation with low-flow capillary channels

C. Dural arteriovenous fistula in this setting as the best diagnosis in this clinical setting

D. Brain arteriovenous malformation with an intraparenchymal nidus

E. Developmental venous anomaly without arterial shunting

142. Why does cortical venous reflux increase hemorrhage risk in a dural AV fistula?

A. The fistula converts cortical veins into muscular arteries with atherosclerotic plaque as the best mechanistic explanation for these findings

B. Retrograde flow eliminates venous pressure and collapses the cortical veins as the best mechanistic explanation for these findings

C. Venous reflux directly causes amyloid deposition in small cortical arteries as the best mechanistic explanation for these findings

D. The shunt blocks platelet production and causes systemic coagulopathy as the best mechanistic explanation for these findings

E. Arterialized high-pressure flow is transmitted retrogradely into thin-walled cortical veins, producing venous hypertension and rupture risk

143. A 38-year-old woman has recurrent brief focal seizures. MRI shows a small lobulated lesion with mixed-signal blood products and a hemosiderin rim, described as a popcorn-like lesion. What is the most likely diagnosis?

A. Developmental venous anomaly with primarily a caput medusae pattern

B. Cerebral cavernous malformation as the best diagnosis in this clinical setting

C. High-flow arteriovenous malformation with early draining veins

D. Atherosclerotic aneurysm of the cervical carotid artery

E. Dural arteriovenous fistula with cortical venous reflux

144. Why are cavernous malformations often angiographically occult?

A. Iodinated contrast is chemically repelled by hemosiderin as the best mechanistic explanation for these findings

B. The lesion is perfused primarily during seizures and otherwise disappears as the best mechanistic explanation for these findings

C. Cavernous malformations contain no blood vessels as the best mechanistic explanation for these findings

D. The lesion contains very low-flow sinusoidal vascular channels without a high-flow arterial shunt

E. The lesion is located outside the cranial circulation entirely as the best mechanistic explanation for these findings

145. A 44-year-old woman has an incidental MRI finding of medullary veins converging on a prominent collecting vein, without hemorrhage or associated cavernous malformation. What is this lesion?

A. Cortical venous sinus thrombosis

B. High-grade dural arteriovenous fistula

C. Developmental venous anomaly

D. Ruptured saccular aneurysm as the best diagnosis in this clinical setting

E. Moyamoya collateral network

146. Why can surgical obliteration of an isolated developmental venous anomaly be dangerous?

A. Removing the DVA invariably ruptures an intracranial aneurysm as the next step as the best mechanistic explanation for these findings

B. The anomalous collector may provide essential venous drainage for otherwise normal brain, so occlusion can cause venous infarction

C. Its walls contain autonomic fibers required for consciousness at this time as the best mechanistic explanation for these findings

D. The DVA supplies the entire arterial circulation of the hemisphere as the best mechanistic explanation for these findings

E. The anomaly controls cerebrospinal fluid production in this clinical context as the best mechanistic explanation for these findings

147. A 71-year-old man has a large mobile aortic arch plaque measuring 6 mm on transesophageal echocardiography after an embolic stroke. Which mechanism can produce cerebral ischemia?

A. Small-vessel lipohyalinosis produced locally in the thalamus by the arch plaque

B. Venous embolization from the aorta through the superior sagittal sinus as the best mechanistic explanation for these findings

C. Direct compression of the brainstem by the aortic arch as the best mechanistic explanation for these findings

D. Artery-to-artery embolization of thrombus or atheromatous debris from the aortic arch into great vessels

E. Failure of CSF absorption because of aortic calcification as the best mechanistic explanation for these findings

148. What makes a complex aortic arch plaque especially emboligenic?

A. Ulceration or superimposed mobile thrombus creates material that can detach into cerebral arterial circulation

B. The arch secretes clotting factors into the carotid artery given the clinical findings and context described

C. Calcification prevents any thrombus formation and increases embolization primarily by vasospasm

D. Aortic plaques embolize mainly through cerebral veins given the clinical findings and context described

E. Plaque lowers intracranial pressure and causes watershed infarction directly given the clinical findings and context described

149. A 67-year-old woman has an embolic-appearing ischemic stroke. Standard vascular imaging, echocardiography, and rhythm monitoring reveal no clear source. Which statement best reflects current secondary prevention for embolic stroke of undetermined source?

A. All ESUS is covert atrial fibrillation and therefore requires warfarin as the most informative choice for this evaluation

B. Do not empirically anticoagulate every ESUS patient in the absence of a defined anticoagulation indication

C. ESUS patients should receive no antithrombotic therapy as the most informative choice for this evaluation

D. All ESUS is carotid disease and requires endarterectomy as the most informative choice for this evaluation

E. Routine triple antiplatelet therapy is superior to further diagnostic evaluation

150. Why did the ESUS concept not translate into a universal anticoagulation strategy?

A. ESUS is defined by the presence of intracranial hemorrhage at this time as the preferred approach in this clinical setting, based on the information provided

B. Anticoagulants is unlikely to prevent any form of arterial embolism as the next step as the preferred approach in this clinical setting

C. The phenotype includes heterogeneous mechanisms such as occult atrial disease, nonstenotic plaque, aortic disease, and other embolic sources with different responses to therapy

D. All embolic strokes arise from infectious endocarditis in this clinical context as the preferred approach in this clinical setting

E. most ESUS patient actually has small-vessel lipohyalinosis for this patient as the preferred approach in this clinical setting

151. A 72-year-old man with long-standing hypertension and diabetes has multiple small deep infarcts, confluent white-matter hyperintensities, and cerebral microbleeds in the basal ganglia. Which vasculopathy best explains the pattern?

A. Reversible cerebral vasoconstriction syndrome as the best mechanistic explanation for these findings

B. Moyamoya disease with terminal ICA stenosis as the best mechanistic explanation for these findings

C. Hypertensive cerebral small-vessel arteriopathy as the best mechanistic explanation for these findings

D. Dural arteriovenous fistula with cortical venous reflux

E. Cerebral amyloid angiopathy limited to cortical and leptomeningeal vessels

152. How does chronic hypertension damage penetrating cerebral arterioles?

A. It deposits beta-amyloid selectively in cortical arterioles for this patient given the clinical findings and context described

B. It obstructs venous sinuses and secondarily narrows arterioles given the clinical findings and context described

C. It causes congenital absence of smooth muscle in most perforator given the clinical findings and context described

D. Persistent pressure stress promotes lipohyalinosis, fibrinoid degeneration, wall thickening, and loss of autoregulatory integrity

E. It transforms penetrating arteries into high-flow arteriovenous shunts given the clinical findings and context described

153. An 84-year-old man has recurrent lobar hemorrhages and numerous strictly lobar cerebral microbleeds. Which pathologic process is most likely?

A. Inflammatory destruction of the basilar artery by infection

B. Fibromuscular dysplasia of renal and cervical medium arteries

C. Beta-amyloid deposition in small cortical and leptomeningeal vessel walls

D. Chronic thrombosis of the superior sagittal sinus as the best diagnosis in this clinical setting

E. Hyaline change limited to deep lenticulostriate arteries

154. Why does cerebral amyloid angiopathy preferentially produce lobar rather than deep hemorrhage?

A. The cortex has higher CSF pressure than the thalamus at this time as the best mechanistic explanation for these findings

B. Deep gray nuclei are protected because they do not receive arterial blood as the best mechanistic explanation for these findings

C. Amyloid selectively accumulates inside cerebral veins of the basal ganglia as the best mechanistic explanation for these findings

D. Amyloid accumulates mainly in cortical and leptomeningeal arterioles rather than the deep perforating vessels affected by hypertensive arteriopathy

E. Lobar brain tissue has no capillaries and therefore bleeds from large arteries as the best mechanistic explanation for these findings

155. A 73-year-old woman with a large cardioembolic MCA infarct develops petechial blood within the infarct bed 48 hours later without clinical worsening or mass effect. What is this finding?

A. A primary hypertensive intracerebral hemorrhage unrelated to the infarct

B. Cerebral venous thrombosis causing a remote hemorrhagic lesion

C. Aneurysmal subarachnoid hemorrhage confined to basal cisterns

D. Hemorrhagic transformation of ischemic infarction given the clinical findings and context described

E. A chronic cavernous malformation incidentally discovered

156. Why is recently infarcted brain prone to hemorrhagic transformation after reperfusion?

A. Infarction increases platelet production inside the brain as the next step as the best mechanistic explanation for these findings

B. Reperfusion converts cerebral arteries into veins for this patient as the best mechanistic explanation for these findings

C. Ischemia disrupts the blood-brain barrier and damages microvascular endothelium, so restored flow can leak into injured tissue

D. Collateral vessels become calcified and mechanically fragment as the best mechanistic explanation for these findings

E. Ischemic neurons secrete vitamin K antagonists into the circulation as the best mechanistic explanation for these findings

157. A patient with a massive MCA infarct develops progressive hemispheric swelling over 48 hours. What type of edema predominates early in ischemic stroke?

A. Cytotoxic edema from failure of cellular ion pumps and intracellular water accumulation

B. Pure vasogenic edema from intact ion pumps and extracellular protein leakage primarily

C. Interstitial edema caused exclusively by venous sinus thrombosis

D. Hydrocephalic edema caused by overproduction of CSF given the clinical findings and context described

E. Osmotic edema caused by systemic hypernatremia given the clinical findings and context described

158. Why do corticosteroids fail to treat malignant ischemic cerebral edema effectively?

A. Corticosteroids increase arterial oxygen content too much for this patient as the best mechanistic explanation for these findings

B. The dominant early process is cytotoxic swelling from cellular energy failure rather than steroid-responsive vasogenic edema from tumor or inflammation

C. Ischemic edema occurs primarily inside the ventricles where steroids is unlikely to enter as the best mechanistic explanation for these findings

D. Corticosteroids invariably cause acute carotid dissection at this time as the best mechanistic explanation for these findings

E. Steroids directly activate tissue plasminogen activator in brain tissue as the next step as the best mechanistic explanation for these findings

159. A patient with a proximal MCA occlusion has surprisingly mild deficits and small infarct core despite several hours of occlusion. Which factor most likely explains this?

A. Robust collateral flow through leptomeningeal arterial connections

B. Increased intracranial venous pressure preventing infarction

C. Complete absence of cerebral metabolic demand as the best diagnosis in this clinical setting

D. Loss of all pial arterial anastomoses as the best diagnosis in this clinical setting

E. A functionally incomplete circle of Willis prevents collateral recruitment and therefore explains the unexpectedly mild deficit

160. How do leptomeningeal collaterals protect brain during a large-vessel occlusion?

A. Microglia synthesize red blood cells inside the infarct in this clinical context given the clinical findings and context described

B. Distal branches of neighboring arterial territories provide retrograde flow across pial anastomoses into the threatened territory

C. The occluded artery expands downstream and creates new blood cells given the clinical findings and context described

D. Cerebral veins reverse direction and become oxygenated arteries given the clinical findings and context described

E. CSF carries oxygen directly from the lungs to ischemic cortex as the next step given the clinical findings and context described

161. A patient with advanced mucinous adenocarcinoma has recurrent cerebral infarcts in multiple territories and markedly elevated D-dimer. Which stroke mechanism is most characteristic?

A. Mechanical compression of intracranial arteries by abdominal tumor as the best mechanistic explanation for these findings

B. Systemic cancer-associated hypercoagulability with thromboembolism or nonbacterial thrombotic endocarditis

C. Primary amyloid deposition triggered by tumor markers as the best mechanistic explanation for these findings

D. Cortical spreading depression caused by chemotherapy in most case as the best mechanistic explanation for these findings

E. Pure hypertensive small-vessel lipohyalinosis as the primarily cancer-related mechanism

162. How can mucin-producing adenocarcinoma promote thrombosis?

A. Mucin dissolves fibrin and therefore causes primarily bleeding for this patient given the clinical findings and context described

B. Adenocarcinoma causes cerebral vasodilation that eliminates clot formation given the clinical findings and context described

C. Tumor-derived procoagulant and inflammatory signals activate coagulation, platelets, and endothelium, creating a systemic thrombotic state

D. Tumors prevent tissue factor expression in the circulation in this clinical context given the clinical findings and context described

E. Cancer cells lower platelet reactivity below normal as the next step given the clinical findings and context described

163. A child with sickle cell anemia has markedly elevated transcranial Doppler velocities in the distal internal carotid and proximal MCA. What do these high velocities signify?

A. Venous sinus thrombosis because TCD measures primarily venous flow given the clinical findings and context described

B. Low cardiac output with reduced cerebral blood flow in this clinical context given the clinical findings and context described

C. Normal childhood hyperemia with no relationship to stroke for this patient given the clinical findings and context described

D. Aneurysmal subarachnoid hemorrhage in the absence of symptoms given the clinical findings and context described

E. Severe flow acceleration through stenotic or high-resistance intracranial vessels associated with increased stroke risk

164. What vascular mechanism contributes to large-artery stenosis in sickle cell disease?

A. The disease causes congenital absence of the circle of Willis at this time as the best mechanistic explanation for these findings

B. Sickle cells form permanent plaques identical to cholesterol atherosclerosis as the best mechanistic explanation for these findings

C. Low-flow venous thrombosis directly compresses most intracranial artery as the best mechanistic explanation for these findings

D. Chronic hemolysis, endothelial dysfunction, inflammation, anemia-related high flow, and intimal proliferation injure intracranial arteries

E. Beta-amyloid deposition selectively narrows terminal carotid arteries in childhood as the best mechanistic explanation for these findings

165. A 78-year-old man has prolonged severe hypotension during cardiac arrest. MRI later shows bilateral cortical borderzone infarcts. Which mechanism best explains this distribution?

A. Global cerebral hypoperfusion preferentially injures distal arterial watershed zones

B. A single embolus selectively occludes most borderzone artery simultaneously

C. Hypertension causes rupture of distal cortical arteries without hemorrhage

D. Migraine aura creates permanent infarction primarily at arterial boundaries

E. Venous thrombosis directly produces symmetric arterial watershed lesions

166. Why are watershed regions especially vulnerable to systemic hypotension?

A. They contain no capillaries and depend on diffusion from CSF for this patient as the best mechanistic explanation for these findings

B. They are supplied exclusively by cerebral veins as the next step as the best mechanistic explanation for these findings

C. Their neurons lack mitochondria under normal conditions in this clinical context as the best mechanistic explanation for these findings

D. They are supplied by distal branches with the lowest perfusion pressure and therefore lose flow first when driving pressure falls

E. They receive blood primarily during systole and none during diastole as the best mechanistic explanation for these findings

167. A 58-year-old woman is found somnolent with vertical gaze palsy and memory impairment. MRI shows bilateral paramedian thalamic infarcts with a small rostral midbrain lesion. Which vascular variant is most likely involved?

A. Superior sagittal sinus thrombosis limited to the frontal lobes

B. Posterior inferior cerebellar artery dissection

C. Single recurrent artery of Heubner occlusion as the best diagnosis in this clinical setting

D. Bilateral middle cerebral artery stem occlusion

E. Artery of Percheron occlusion in this setting

168. Why can one small arterial occlusion cause bilateral thalamic infarction in the artery of Percheron variant?

A. The variant eliminates all thalamic collateral circulation from the PCA as the best mechanistic explanation for these findings

B. Both thalami are supplied primarily by the basilar artery trunk in all people as the best mechanistic explanation for these findings

C. One perforator crosses the corpus callosum to supply both occipital lobes as the best mechanistic explanation for these findings

D. A single dominant perforator supplies paramedian territories on both sides instead of separate paired arteries

E. The thalami share a single venous sinus that becomes arterialized as the best mechanistic explanation for these findings

169. A 63-year-old man develops sudden bilateral leg weakness, loss of pain and temperature below the umbilicus, urinary retention, and preserved vibration sense. MRI shows central anterior spinal cord diffusion restriction. What is the syndrome?

A. Anterior spinal artery infarction as the best diagnosis in this clinical setting

B. Guillain-Barre syndrome with peripheral demyelination

C. Cauda equina compression affecting primarily lower motor neurons

D. Brown-Sequard syndrome from hemicord trauma as the best diagnosis in this clinical setting

E. Posterior spinal cord infarction affecting dorsal columns primarily

170. Why can the anterior spinal cord be vulnerable during severe aortic hypotension or aortic surgery?

A. CSF pressure directly closes the posterior spinal arteries first as the next step as the best mechanistic explanation for these findings

B. The anterior cord has no arterial blood supply under normal conditions for this patient as the best mechanistic explanation for these findings

C. Aortic surgery causes amyloid deposition in spinal perforators within minutes as the best mechanistic explanation for these findings

D. Its long longitudinal arterial supply depends on a limited number of radiculomedullary feeders, making some segments sensitive to reduced perfusion

E. The spinal cord is perfused primarily by the internal carotid arteries at this time as the best mechanistic explanation for these findings

171. A 45-year-old woman has recurrent ischemic strokes and headaches. High-resolution vessel-wall MRI shows concentric enhancement of several intracranial arterial segments, and CSF is inflammatory. Which process is most consistent with these findings?

A. Cerebral venous sinus thrombosis causing arterial wall enhancement

B. A fixed calcified atherosclerotic plaque with no inflammatory activity

C. A normal age-related change without clinical significance given the clinical findings and context described

D. Simple embolic occlusion from atrial fibrillation without vessel-wall inflammation

E. Inflammatory intracranial vasculopathy such as primary CNS vasculitis

172. What biologic process produces ischemic stroke in primary CNS vasculitis?

A. Inflammation increases collateral flow enough to prevent infarction as the best mechanistic explanation for these findings

B. Transmural vascular inflammation damages endothelium and narrows or thromboses affected arteries, reducing downstream perfusion

C. Inflammation produces vasodilation and steal rather than luminal narrowing or thrombosis as the dominant ischemic mechanism

D. Autoantibodies directly dissolve cerebral myelin without affecting vessels as the best mechanistic explanation for these findings

E. Amyloid deposition universally occludes veins before arteries as the best mechanistic explanation for these findings

173. A 45-year-old woman develops a sudden maximal-at-onset headache while lifting weights. Neurologic examination is normal. Noncontrast CT performed 10 hours later is negative. Which diagnosis must remain a priority despite the normal examination?

A. Aneurysmal subarachnoid hemorrhage as the best diagnosis in this clinical setting

B. Medication-overuse headache from daily analgesics

C. Idiopathic intracranial hypertension without papilledema

D. Chronic migraine without aura as the best diagnosis in this clinical setting

E. Tension-type headache related to muscle contraction

174. A 37-year-old woman has recurrent thunderclap headaches over five days. Initial head CT is normal. Which vascular study is most useful for evaluating a suspected reversible cerebral vasoconstriction syndrome?

A. Repeat noncontrast head CT alone because arterial vasoconstriction is reliably inferred from evolving parenchymal changes

B. Use lumbar puncture as the primary test because a normal CSF profile confirms reversible cerebral vasoconstriction

C. CTA or MRA of the intracranial arteries, with repeat vascular imaging if early studies are nondiagnostic

D. Use carotid duplex ultrasonography as the principal vascular study even though the suspected narrowing is intracranial

E. Obtain brain MRI without arterial imaging and diagnose RCVS from headache pattern alone if no infarct is present

175. A patient with probable reversible cerebral vasoconstriction syndrome has severe recurrent thunderclap headaches after exposure to a sympathomimetic decongestant. Which management step is most appropriate?

A. Stop the vasoactive trigger and use supportive therapy while monitoring for ischemic or hemorrhagic complications

B. Start high-dose glucocorticoids routinely because they improve RCVS outcome as the best diagnosis in this clinical setting

C. Use lifelong warfarin because the syndrome is caused by emboli as the best diagnosis in this clinical setting

D. Perform angioplasty of most narrowed artery during the first headache as the best diagnosis in this clinical setting

E. Induce systemic hypotension to reduce arterial wall stress as the best diagnosis in this clinical setting

176. A 68-year-old man with a remote cortical stroke has a focal seizure with right-arm jerking followed by 20 minutes of right-arm weakness that fully resolves. CTA shows no new occlusion. What is the most likely explanation for the transient weakness?

A. A persistent new left MCA infarction despite complete resolution

B. Hemiplegic migraine with no relation to the witnessed seizure

C. Limb-shaking TIA from critical carotid stenosis triggered by standing

D. Todd postictal paresis in this setting as the best diagnosis in this clinical setting

E. Acute cervical myelopathy causing unilateral arm weakness

177. A patient remains confused with subtle intermittent right facial twitching after a lobar intracerebral hemorrhage. Which test is most appropriate to evaluate for nonconvulsive or subtle ongoing seizures?

A. Continuous or prolonged electroencephalographic monitoring

B. Transcranial Doppler for embolus detection primarily

C. Routine echocardiography to detect ictal discharges

D. Carotid duplex ultrasonography as the preferred approach in this clinical setting

E. Lumbar puncture to measure opening pressure

178. A patient has a single early focal seizure during the first day after an acute cortical ischemic stroke and returns to baseline. Which antiseizure strategy is most appropriate?

A. Give chronic benzodiazepines to prevent any future cortical irritability as the preferred approach in this clinical setting

B. Use prophylactic antiseizure medication in most ischemic stroke patient as the preferred approach in this clinical setting

C. Stop all stroke prevention medications because antiseizure drugs replace them as the preferred approach in this clinical setting

D. Start lifelong dual antiseizure therapy after most early post-stroke seizure as the preferred approach in this clinical setting

E. Treat the acute seizure if needed, but do not assume lifelong antiseizure medication is required after a single provoked early seizure

179. A 59-year-old man with a recent ischemic stroke snores loudly, has witnessed apneas, morning headaches, and daytime sleepiness. Which comorbidity is most likely?

A. REM sleep behavior disorder as the primary cause of snoring

B. Narcolepsy with cataplexy for this patient

C. Fatal familial insomnia at this time as the best diagnosis in this clinical setting

D. Obstructive sleep apnea in this setting as the best diagnosis in this clinical setting

E. Restless legs syndrome causing upper-airway collapse

180. A stroke survivor has high clinical suspicion for obstructive sleep apnea. Which test provides the most definitive physiologic assessment when a comprehensive study is needed?

A. Carotid ultrasound during sleep as the most informative choice for this evaluation

B. Electroencephalography without respiratory channels

C. Lumbar puncture with opening pressure measurement

D. Transesophageal echocardiography at midnight

E. Overnight polysomnography as the most informative choice for this evaluation

181. A patient with ischemic stroke is found to have moderate obstructive sleep apnea. Which intervention is most appropriate?

A. Offer positive airway pressure therapy and address adherence barriers along with vascular risk-factor control

B. Use supplemental oxygen alone as a standard substitute for airway therapy as the preferred approach in this clinical setting

C. Start anticoagulation when obstructive sleep apnea is moderate because the sleep disorder independently implies a cardioembolic mechanism

D. Prescribe chronic sedatives to deepen sleep and suppress arousals as the preferred approach in this clinical setting

E. Avoid treatment because sleep apnea has no cardiovascular relevance as the preferred approach in this clinical setting

182. A 34-year-old man with untreated HIV develops subacute headache, fever, confusion, and multifocal ischemic strokes. CSF shows lymphocytic pleocytosis, and vascular imaging shows multifocal intracranial narrowing. Which broad mechanism should be considered?

A. Isolated hypertensive lipohyalinosis as the primarily explanation

B. Idiopathic lacunar disease unrelated to immune status

C. Cerebral amyloid angiopathy in a young adult

D. Infectious or inflammatory cerebral vasculopathy

E. Pure cardioembolism with no relation to the infection

183. A patient with suspected varicella-zoster virus cerebral vasculopathy has multifocal strokes and inflammatory CSF but no rash. Which testing strategy is most useful?

A. CSF testing for intrathecal VZV antibody together with PCR when appropriate

B. Carotid duplex because VZV affects primarily extracranial arteries

C. Routine urine culture as the primary confirmation test as the preferred approach in this clinical setting

D. Serum VZV IgG alone because nearly all adults with prior exposure have the diagnosis

E. EEG because viral vasculopathy is defined by epileptiform discharges

184. A patient has a convincing diagnosis of varicella-zoster virus vasculopathy causing ischemic strokes. Which therapy is most appropriate?

A. No antiviral therapy because vascular infection is self-limited as the best diagnosis in this clinical setting

B. Carotid endarterectomy because viral arteritis is confined to the bulb

C. Long-term dual antiplatelet therapy as the primarily disease-specific treatment

D. Chronic warfarin primarily to suppress viral replication as the best diagnosis in this clinical setting

E. Intravenous acyclovir, with adjunct corticosteroid considered in selected inflammatory cases

185. A 58-year-old man develops acute quadriparesis after a hyperextension neck injury. MRI shows central cervical cord injury without fracture, and arm weakness is greater than leg weakness. What syndrome is most likely?

A. Brown-Sequard syndrome from hemicord injury as the best diagnosis in this clinical setting

B. Locked-in syndrome from ventral pontine infarction

C. Cauda equina syndrome affecting lumbosacral roots

D. Anterior spinal artery infarction with preserved vibration sense

E. Central cord syndrome in this setting as the best diagnosis in this clinical setting

186. A patient has abrupt paraplegia and a sensory level after thoracic aortic repair. Which MRI sequence is particularly helpful for supporting acute spinal cord infarction?

A. Use contrast-enhanced cervical spine MRI without diffusion-weighted sequences and infer infarction from cord swelling alone

B. Diffusion-weighted imaging of the spinal cord together with conventional T2-weighted sequences

C. Use susceptibility-weighted brain MRI to look for cerebral microbleeds as the main confirmation of spinal cord ischemia

D. Use spinal MR angiography alone because visualization of a normal anterior spinal artery excludes infarction

E. Use conventional T1-weighted spine imaging alone because acute spinal cord infarction does not affect diffusion signal

187. A patient with acute cervical spinal cord compression from an epidural hematoma develops rapidly progressive weakness. What is the most appropriate next step?

A. Use thrombolysis to dissolve the epidural blood collection as the preferred approach in this clinical setting

B. Observe for spontaneous recovery because compression does not threaten permanent injury

C. Perform carotid stenting to improve spinal perfusion as the preferred approach in this clinical setting

D. Urgent neurosurgical evaluation for decompression and correction of any coagulopathy

E. Start long-term anticoagulation to shrink the hematoma as the preferred approach in this clinical setting

188. A 71-year-old man develops sudden painless monocular blindness lasting 8 minutes with complete recovery. Funduscopic examination is normal afterward. Which diagnosis is most likely?

A. Retinal detachment because vision fully recovers within minutes

B. Amaurosis fugax from transient retinal ischemia as the best diagnosis in this clinical setting

C. Retinal migraine producing recurrent monocular positive and negative visual symptoms without an embolic source

D. Anterior ischemic optic neuropathy producing a persistent altitudinal defect rather than a brief fully reversible episode

E. Occipital lobe seizure because each eye has separate cortical representation

189. A patient has acute vertigo, nausea, and gait unsteadiness with continuous spontaneous nystagmus. Which bedside examination is useful in experienced hands for distinguishing central from peripheral acute vestibular syndrome?

A. The HINTS examination including head impulse, nystagmus pattern, and test of skew

B. Tinel sign at the wrist in this clinical context as the best diagnosis in this clinical setting

C. Romberg testing as the primarily required component as the best diagnosis in this clinical setting

D. The Mini-Mental State Examination alone as the best diagnosis in this clinical setting

E. Straight-leg-raise testing as the next step as the best diagnosis in this clinical setting

190. A 66-year-old woman has a retinal artery occlusion with sudden persistent monocular visual loss that began 90 minutes ago. What is the most appropriate systems response?

A. Assume the event is benign if the fellow eye sees normally in this clinical context as the preferred approach in this clinical setting

B. Start chronic corticosteroids without checking age or inflammatory symptoms as the preferred approach in this clinical setting

C. Schedule routine ophthalmology follow-up in several weeks because the event is not a stroke equivalent as the preferred approach in this clinical setting

D. Treat the event as an acute ischemic stroke emergency and obtain immediate stroke-center evaluation for reperfusion eligibility and embolic source workup

E. Delay vascular evaluation until permanent optic atrophy develops at this time as the preferred approach in this clinical setting

191. A patient with a right occipital infarct has a left homonymous hemianopia but retains central vision bilaterally. Which mechanism can explain macular sparing?

A. Macular sparing occurs because central visual fibers project bilaterally to both occipital cortices and therefore resist unilateral injury

B. The macula projects exclusively to the cerebellum rather than visual cortex as the best mechanistic explanation for these findings

C. Dual or collateral blood supply to the occipital pole, including contribution from MCA branches in some individuals

D. Central vision is mediated primarily by the superior colliculus in adults as the best mechanistic explanation for these findings

E. The optic nerves cross twice before reaching the occipital lobe as the best mechanistic explanation for these findings

192. A 64-year-old man with chronic alcohol use develops confusion, gait ataxia, and ophthalmoplegia during a hospitalization for stroke. Which diagnosis should be treated immediately?

A. Hepatic myelopathy without encephalopathy

B. Miller Fisher syndrome despite no areflexia or neuropathy

C. Normal-pressure hydrocephalus developing within hours

D. Pure cerebellar infarction as the primarily possible cause

E. Wernicke encephalopathy requiring immediate thiamine treatment

193. A patient with sudden focal deficits has severe hyponatremia and fluctuating confusion. MRI shows no acute infarct. Which approach best distinguishes metabolic encephalopathy from stroke?

A. Assume any electrolyte abnormality proves the symptoms are nonvascular as the preferred approach in this clinical setting

B. Use serum sodium as a direct marker of cerebral artery occlusion at this time as the preferred approach in this clinical setting

C. Avoid repeat examination because metabolic deficits is unlikely to coexist with stroke as the preferred approach in this clinical setting

D. Attribute the persistent focal deficits to metabolic encephalopathy and stop vascular evaluation once severe hyponatremia is identified

E. Correct the metabolic derangement while continuing focused neurologic assessment and vascular imaging when focal deficits remain unexplained

194. A malnourished patient with possible Wernicke encephalopathy requires intravenous glucose for severe hypoglycemia. What is the best thiamine strategy?

A. Withhold glucose until thiamine levels return from the laboratory as the preferred approach in this clinical setting

B. Give parenteral thiamine promptly, ideally before or with glucose, without delaying lifesaving glucose treatment

C. Avoid thiamine because it can precipitate cerebral edema as the preferred approach in this clinical setting

D. Use oral thiamine initially because gastrointestinal absorption is sufficient even when acute Wernicke encephalopathy is suspected

E. Give folate alone because it replaces thiamine function as the preferred approach in this clinical setting

195. A 73-year-old man with extensive subcortical ischemic disease develops slowed processing, impaired executive function, gait difficulty, and emotional lability with relatively preserved recognition memory early in the course. Which syndrome is most likely?

A. Subcortical vascular cognitive impairment as the best diagnosis in this clinical setting

B. Normal cognition expected after small-vessel ischemic disease

C. Frontotemporal dementia with prominent early disinhibition and no vascular disease

D. Typical early Alzheimer disease with isolated episodic memory storage failure primarily

E. Lewy body dementia defined by early visual hallucinations and parkinsonism

196. A stroke survivor reports cognitive decline affecting finances and medication management. What is the most appropriate evaluation?

A. Use NIHSS alone because it fully measures executive and memory function for this patient as the preferred approach in this clinical setting

B. Avoid collateral history because caregiver observations are unreliable by definition as the preferred approach in this clinical setting

C. Diagnose dementia from MRI white-matter disease without clinical assessment as the preferred approach in this clinical setting

D. Use bedside cognitive screening followed by formal neuropsychological assessment when deficits or functional consequences require detailed characterization

E. Order carotid ultrasound as a substitute for cognitive testing as the next step as the preferred approach in this clinical setting

197. A patient with vascular cognitive impairment has poorly controlled hypertension, diabetes, sedentary lifestyle, and recurrent lacunar strokes. Which intervention is most important?

A. Treat primarily memory symptoms and ignore recurrent vascular events as the preferred approach in this clinical setting

B. Use antipsychotic medication as the primary disease-modifying treatment as the preferred approach in this clinical setting

C. Avoid blood-pressure control because cognition depends on chronic hypertension as the preferred approach in this clinical setting

D. Stop exercise because activity increases cerebral metabolic demand as the preferred approach in this clinical setting

E. Intensively manage vascular risk factors and prevent additional strokes while providing cognitive and functional rehabilitation

198. A 29-year-old woman develops optic neuritis and a longitudinally extensive transverse myelitis. Serum aquaporin-4 IgG is positive. Which diagnosis is most likely?

A. Multiple sclerosis with a classic short-segment cord lesion pattern

B. Neuromyelitis optica spectrum disorder as the best diagnosis in this clinical setting

C. Hypertensive arteriopathy at this time as the best diagnosis in this clinical setting

D. Cerebral amyloid angiopathy as the next step as the best diagnosis in this clinical setting

E. Moyamoya disease in this clinical context as the best diagnosis in this clinical setting

199. A patient with subacute encephalopathy, faciobrachial dystonic seizures, and hyponatremia is suspected of having autoimmune encephalitis. Which test is particularly relevant?

A. Transcranial Doppler embolus monitoring as the definitive test as the most informative choice for this evaluation

B. A single normal head CT that excludes encephalitis in this clinical context as the most informative choice for this evaluation

C. Serum and CSF neuronal antibody evaluation, including LGI1 antibodies, interpreted with MRI, EEG, and clinical findings

D. Carotid duplex as the sole diagnostic test for this patient as the most informative choice for this evaluation

E. D-dimer alone because autoimmune encephalitis is a thrombotic disorder as the most informative choice for this evaluation

200. A patient has probable autoimmune encephalitis with frequent seizures and progressive memory loss after infection and structural causes have been reasonably excluded. What is an appropriate initial treatment?

A. Perform carotid endarterectomy to remove the source of neuronal antibodies at this time as the preferred approach in this clinical setting

B. Treat primarily with aspirin because antibodies are platelet-mediated in this clinical context as the preferred approach in this clinical setting

C. Use thrombolysis because autoimmune inflammation is caused by arterial clot as the preferred approach in this clinical setting

D. Avoid immunotherapy until irreversible dementia develops as the next step as the preferred approach in this clinical setting

E. Begin immunotherapy such as high-dose corticosteroids, IVIG, or plasma exchange while treating seizures and screening for an associated tumor when indicate

201. A right-handed patient has acute inability to speak fluently, impaired repetition, preserved comprehension, and right lower facial weakness. Which cerebral hemisphere is most likely dominant for language?

A. Cerebellar hemisphere as the next step

B. Left hemisphere in this setting as the best diagnosis in this clinical setting

C. Right hemisphere in nearly all right-handed adults

D. Nondominant parietal cortex primarily as the best diagnosis in this clinical setting

E. Medulla without cerebral involvement

202. A patient with an acute stroke has left-sided neglect, extinction to double simultaneous stimulation, and constructional apraxia. Which hemisphere is most likely affected?

A. Right nondominant parietal hemisphere

B. Cervical spinal cord as the best diagnosis in this clinical setting

C. Medial medulla in this clinical context

D. Left dominant frontal operculum

E. Bilateral primary visual cortices primarily

203. A patient follows commands by looking up and down but cannot move the limbs or speak. Which finding most strongly indicates preserved consciousness?

A. Reproducible voluntary eye movements or blinking to command

B. Lack of verbal output in this clinical context

C. Absence of limb movement for this patient given the clinical findings and context described

D. Need for mechanical ventilation given the clinical findings and context described

E. Extensor posturing to pain as the next step

204. A patient has suspected large-vessel ischemic stroke. Which imaging sequence provides the fastest routine assessment of intracranial and cervical arterial occlusion in most emergency departments?

A. Lumbar spine MRI in this clinical context as the most informative choice for this evaluation

B. CT angiography from the aortic arch through the intracranial circulation

C. Routine skull radiography as the next step as the most informative choice for this evaluation

D. Carotid auscultation without imaging as the most informative choice for this evaluation

E. Noncontrast head CT alone for this patient as the most informative choice for this evaluation

205. A patient with suspected embolic stroke has a normal transthoracic echocardiogram, but concern remains for left atrial appendage thrombus and aortic arch plaque. Which study can provide better visualization of these structures?

A. Carotid duplex ultrasonography

B. Routine electroencephalography

C. Transesophageal echocardiography

D. Lumbar puncture at this time

E. Noncontrast head CT as the most informative choice for this evaluation

206. In a stroke patient, MRI shows diffusion restriction with corresponding low ADC signal. What does this combination most strongly indicate?

A. Normal white matter myelination as the next step given the clinical findings and context described

B. A purely vascular flow artifact in most case given the clinical findings and context described

C. Chronic encephalomalacia with freely diffusing water given the clinical findings and context described

D. Acute cytotoxic edema consistent with recent infarction in the appropriate clinical context

E. Calcification that is unlikely to represent ischemia given the clinical findings and context described

207. A stroke survivor smokes one pack per day and wants the single most effective strategy for reducing tobacco-related vascular risk. What should be offered?

A. Chronic dual antiplatelet therapy as a substitute for quitting tobacco as the preferred approach in this clinical setting

B. Switching to cigars because they do not increase stroke risk as the preferred approach in this clinical setting

C. Advice to reduce to half a pack without a quit plan for this patient as the preferred approach in this clinical setting

D. No intervention because smoking cessation after stroke is too late as the preferred approach in this clinical setting

E. Behavioral counseling combined with evidence-based smoking-cessation pharmacotherapy when not contraindicated

208. A patient with a large dominant-hemisphere stroke has severe aphasia but appears to understand simple questions. His spouse answers every question for him. What is the best communication approach?

A. Use complex multi-part questions to test the patient efficiently as the next step as the preferred approach in this clinical setting

B. Address the patient directly, use simple supported communication, allow extra response time, and involve the spouse without bypassing the patient

C. Assume inability to speak means inability to understand or choose in this clinical context as the preferred approach in this clinical setting

D. Speak primarily to the spouse because aphasia eliminates decision-making capacity as the preferred approach in this clinical setting

E. Avoid visual aids because they can bias the patient at this time as the preferred approach in this clinical setting

209. Family members ask whether a patient with a massive hemispheric infarct will "recover completely." Prognosis is uncertain. Which response is best?

A. State that no useful prognosis can ever be discussed after stroke as the next step given the clinical findings and context described

B. Explain what is known, describe the range of likely outcomes and key uncertainties, and revisit prognosis as the clinical course evolves

C. Give primarily a mortality percentage without discussing function or uncertainty given the clinical findings and context described

D. Avoid discussing uncertainty because it undermines trust at this time given the clinical findings and context described

E. Promise complete recovery to preserve hope for this patient given the clinical findings and context described

210. A patient with mild cognitive impairment declines carotid surgery after hearing the risks and benefits in plain language and accurately explains the consequences of refusal. What is the best next step?

A. Ask the family to consent in place of a capable patient as the preferred approach in this clinical setting

B. Label the patient incompetent because the choice differs from the recommendation

C. Respect the informed decision, document the discussion, and continue optimal medical therapy

D. Discontinue all stroke prevention because surgery was declined as the preferred approach in this clinical setting

E. Override the decision because the clinician believes surgery is beneficial

211. A neurologist realizes that a thrombolytic dose was entered incorrectly but the error was caught before administration and no harm occurred. What is the most appropriate action?

A. Hide the event because no medication reached the patient for this patient as the preferred approach in this clinical setting

B. Blame the pharmacist without reviewing the system in this clinical context as the preferred approach in this clinical setting

C. Disclose the near miss through appropriate safety channels and help analyze how the ordering process allowed the error

D. Wait for a similar error to cause harm before reporting at this time as the preferred approach in this clinical setting

E. Alter the chart so the incorrect order is unlikely to be traced as the next step as the preferred approach in this clinical setting

212. A clinician is asked to enroll an incapacitated stroke patient in a research study for which the clinician is also a paid investigator. What is the most important professional obligation?

A. Disclose the conflict of interest and follow independent consent and ethics safeguards for surrogate authorization

B. Avoid institutional review because emergency neurology is exempt from ethics standards

C. Use clinical consent for thrombectomy as consent for unrelated research as the most informative choice for this evaluation

D. Enroll the patient automatically because research may help future patients as the most informative choice for this evaluation

E. Conceal the financial relationship because it may confuse the surrogate as the most informative choice for this evaluation

213. A patient with stroke has a documented do-not-resuscitate order. A consultant suggests that the DNR means the team should also withhold antibiotics and rehabilitation. What is the best response?

A. Accept the suggestion because DNR means comfort care primarily in most case given the clinical findings and context described

B. Clarify that a DNR order limits resuscitation after cardiopulmonary arrest and does not automatically restrict other beneficial treatments

C. Convert the order to full code without discussing it with the patient or surrogate given the clinical findings and context described

D. Ignore the patient's stated goals and ask the family to choose instead given the clinical findings and context described

E. Cancel all diagnostic testing because DNR eliminates treatment goals given the clinical findings and context described

214. A stroke service finds that its median door-to-needle time has risen from 42 to 61 minutes. What is the best first quality-improvement step?

A. Assume slower treatment reflects more complex patients and take no action

B. Replace the entire team before examining the process given the clinical findings and context described

C. Stop tracking door-to-needle time because metrics can create pressure

D. Set a lower target without measuring any intermediate steps given the clinical findings and context described

E. Map the current workflow and identify where delays occur using time-stamped process data

215. A hospital implements a new stroke-alert protocol and wants to know whether it improves treatment speed without increasing complications. Which evaluation is most appropriate?

A. Use stroke-alert volume as the primary effectiveness measure and defer treatment-time and safety measures until later

B. Track prespecified process and outcome measures before and after implementation, including balancing safety measures

C. Measure primarily staff satisfaction because clinical outcomes are unnecessary as the preferred approach in this clinical setting

D. Avoid measuring hemorrhage because adverse outcomes may discourage adoption as the preferred approach in this clinical setting

E. Assume the protocol works if one patient is treated rapidly as the preferred approach in this clinical setting

216. A neurologist notices several recent posterior-circulation strokes were initially misdiagnosed as peripheral vertigo. Which response best reflects practice-based learning?

A. Adopt routine MRI for all acute dizziness presentations rather than improve bedside examination and targeted imaging selection

B. Stop evaluating patients with vertigo to reduce diagnostic errors as the next step given the clinical findings and context described

C. Review the cases for common diagnostic failures, identify knowledge or workflow gaps, and implement targeted education or decision support

D. Attribute most miss to individual carelessness and avoid system review given the clinical findings and context described

E. Wait for national guidelines to change before examining local performance given the clinical findings and context described

217. A rural hospital identifies a patient with a disabling large-vessel occlusion who is receiving intravenous thrombolysis. The nearest thrombectomy center is 70 minutes away. What is the best systems approach?

A. Keep the patient locally because transfer cancels the benefit of thrombolysis as the preferred approach in this clinical setting

B. Arrange immediate transfer to the thrombectomy-capable center while thrombolysis proceeds, using a predefined transfer pathway

C. Wait for the thrombolytic infusion to finish and reassess several hours later before calling

D. Send the patient to the nearest hospital regardless of thrombectomy capability as the preferred approach in this clinical setting

E. Transfer primarily if the patient first develops malignant edema in this clinical context as the preferred approach in this clinical setting

218. A stroke program wants to reduce disparities in thrombectomy access for patients from distant communities. Which intervention is most likely to help?

A. Require all patients to obtain outpatient specialist approval before transfer as the best diagnosis in this clinical setting

B. Rely on individual clinicians to improvise transfer decisions each time as the best diagnosis in this clinical setting

C. Avoid collecting demographic data because disparities is unlikely to be measured objectively as the best diagnosis in this clinical setting

D. Restrict thrombectomy referrals to patients who live near the comprehensive center as the best diagnosis in this clinical setting

E. Develop regional triage, tele-stroke, transport, and transfer protocols with continuous measurement of access by geography and population

219. A comprehensive stroke center repeatedly receives transfer patients without imaging discs or electronic image access, causing thrombectomy delays. What is the most effective systems solution?

A. Require printed radiology reports as a complete substitute for source images as the most informative choice for this evaluation

B. Ask each family to photograph the CT images on a phone as the most informative choice for this evaluation

C. Create a standardized image-sharing and transfer checklist integrated into the referring and receiving workflows

D. Repeat all imaging automatically without attempting transfer of prior studies as the most informative choice for this evaluation

E. Accept the delays because outside hospitals is unlikely to share images as the most informative choice for this evaluation

220. During a nationwide contrast shortage, a hospital must preserve CTA access for time-critical stroke care. Which systems principle is most appropriate?

A. Allocate contrast on a first-come basis regardless of emergency need as the preferred approach in this clinical setting

B. Stop vascular imaging for all patients until the shortage ends as the preferred approach in this clinical setting

C. Allow individual clinicians to compete informally for remaining supply as the preferred approach in this clinical setting

D. Use transparent prioritization criteria based on clinical urgency and likely impact on patient outcome

E. Reserve all contrast for elective studies scheduled months earlier as the preferred approach in this clinical setting

Answers

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1. A. This patient has a disabling acute ischemic stroke within the intravenous thrombolysis window and a proximal large-vessel occlusion. Eligible patients should receive intravenous thrombolysis without delaying endovascular evaluation; one therapy is not a reason to postpone the other. Waiting for improvement wastes time, and a normal early CT does not exclude ischemia. Aggressive blood-pressure lowering below 140 mm Hg is not required before reperfusion.

2. C. When intravenous thrombolysis is indicated, planned thrombectomy is not a reason to withhold or delay it. Bridging therapy preserves the opportunity for earlier reperfusion while transfer and endovascular preparation occur. The key operational principle is parallel rather than sequential decision-making.

3. C. For a clearly nondisabling minor ischemic stroke without a thrombectomy target, routine intravenous thrombolysis does not provide the same benefit as it does for disabling deficits. Early antiplatelet therapy, including short-course dual antiplatelet therapy in appropriate minor stroke or high-risk TIA patients, reduces early recurrence. Therapeutic heparin and thrombectomy are not indicated in this scenario.

4. E. Short-term dual antiplatelet therapy is useful in selected early minor ischemic stroke or high-risk TIA because recurrence risk is front-loaded. The benefit does not justify indefinite dual therapy, which increases bleeding risk. Anticoagulation is reserved for a defined indication such as atrial fibrillation.

5. D. Tenecteplase at 0.25 mg/kg, capped at 25 mg, is an accepted thrombolytic option for eligible acute ischemic stroke patients and is operationally convenient because it is given as a single bolus. The higher tenecteplase dose is not the standard stroke regimen. Urokinase and streptokinase are not routine systemic therapies for acute ischemic stroke.

6. C. Tenecteplase has a longer half-life and can be delivered as a single bolus, which can simplify door-in-door-out workflows. It does not remove standard safety screening, blood-pressure requirements, or hemorrhage risk. Recent anticoagulant exposure still requires careful eligibility assessment.

7. B. Blood pressure above the thrombolysis threshold should be treated with a rapid, titratable intravenous agent. If it can be reduced below 185/110 mm Hg and other criteria are satisfied, thrombolysis can proceed. The initial elevated reading is not by itself a permanent exclusion, but treatment should not be given while pressure remains above the threshold.

8. C. After intravenous thrombolysis, blood pressure is generally maintained below 180/105 mm Hg during the first 24 hours. Excessive lowering can compromise cerebral perfusion, while uncontrolled hypertension increases hemorrhagic risk. The target therefore balances reperfusion safety with perfusion needs.

9. D. In selected wake-up or unknown-onset strokes, tissue-based imaging can identify patients likely to have relatively recent ischemia despite an uncertain clock time. A diffusion-positive, FLAIR-negative or minimally positive pattern is one established selection approach. Imaging selection does not make all unknown-onset strokes eligible; clinical and safety criteria still apply.

10. C. Extended or unknown-window thrombolysis relies on imaging that estimates tissue age or identifies salvageable penumbra, not on age or NIHSS cutoffs alone. A normal CT is useful to exclude hemorrhage but is not sufficient to define all patients as candidates. Tissue-based selection is the key concept.

11. C. Recent direct factor Xa inhibitor exposure raises concern for systemic anticoagulant activity and bleeding with thrombolysis. Eligibility depends on the timing of the last dose, renal clearance, and whether meaningful drug activity can be excluded with an appropriate assay when available. Aspirin use, carotid bruit, LDL level, and atrial size do not resolve the immediate thrombolysis safety question.

12. A. Active therapeutic anticoagulant effect is an important bleeding hazard for intravenous thrombolysis. There are selected circumstances in which laboratory testing or specific reversal considerations may alter decisions, but routine thrombolysis in the presence of substantial active DOAC effect is avoided. A large-vessel occlusion may still be treatable with thrombectomy if the patient otherwise qualifies.

13. C. Severe thrombocytopenia materially increases bleeding risk with systemic thrombolysis; a platelet count below 100,000/μL is generally considered a contraindication to routine intravenous thrombolysis. The normal CT only excludes current intracranial hemorrhage and does not eliminate systemic bleeding risk. Disabling symptoms do not override major safety exclusions.

14. C. Intravenous thrombolysis and mechanical thrombectomy do not share every contraindication. A low platelet count may preclude routine systemic thrombolysis, yet a patient with a treatable large-vessel occlusion can still be considered for endovascular therapy. The decision should preserve potentially beneficial reperfusion options rather than treating the exclusions as identical.

15. B. Hypoglycemia can closely mimic focal stroke and is rapidly reversible. Immediate correction is essential, followed by neurologic reassessment; persistent focal deficits after correction can still represent true ischemic stroke and should be evaluated urgently. Treating the reversible mimic first does not mean abandoning reperfusion evaluation if deficits remain.

16. A. Complete resolution of focal deficits with correction of profound hypoglycemia strongly supports a metabolic mimic. Stroke and hypoglycemia can coexist, so persistent or recurrent deficits would require renewed vascular evaluation. Prophylactic thrombolysis is not appropriate after the neurologic syndrome has resolved through correction of the metabolic cause.

17. E. A seizure can occur at the onset of acute ischemic stroke, especially with cortical involvement. Persistent focal deficits plus a concordant large-vessel occlusion strongly favor ongoing ischemia rather than isolated Todd paralysis. Reperfusion decisions should be based on the likelihood that the residual deficit is ischemic and on standard safety criteria.

18. A. Seizure at onset is not an automatic barrier to acute reperfusion. The key distinction is whether the persistent deficit is better explained by ischemia than by a transient postictal state. Concordant imaging and a disabling syndrome support treating the stroke rather than allowing the seizure to create an avoidable delay.

19. B. Rapid improvement is not itself a contraindication if the patient is left with a disabling neurologic deficit. The relevant question is the functional impact of what remains, not whether the NIHSS score has fallen. Persistent aphasia can be profoundly disabling even without major weakness.

20. E. NIHSS magnitude and functional disability are related but not identical. A lower score can still represent a disabling deficit such as aphasia, hemianopia, or dominant-hand weakness. Acute treatment

decisions should therefore incorporate the patient-specific functional consequences of the residual deficit.

21. B. Noncontrast CT is primarily used to exclude hemorrhage and identify extensive established infarction or other contraindicating pathology. Subtle early ischemic changes are common and do not automatically exclude intravenous thrombolysis. Extensive completed infarction raises different risk-benefit concerns, but a small early change is not synonymous with hemorrhage.

22. A. The immediate role of noncontrast CT is safety triage: exclude hemorrhage and detect large established infarction or another structural diagnosis. Ischemic stroke can be present despite a nearly normal early CT. Etiologic workup and long-term prognostication should not delay time-sensitive reperfusion decisions.

23. B. Randomized trials have expanded the population that can benefit from endovascular therapy to include selected patients with larger established cores. Core size influences prognosis and selection but is no longer an automatic exclusion. Decisions integrate time, occlusion site, disability, baseline function, imaging, and overall clinical context.

24. B. Large-core thrombectomy trials broadened treatment eligibility, so categorical exclusion based solely on a large core is outdated. Imaging still matters because it helps define the amount of established injury and expected benefit, but it should inform rather than automatically terminate evaluation. Patient selection remains individualized.

25. C. Mechanical thrombectomy can benefit selected patients well beyond 6 hours when they have an appropriate large-vessel occlusion and favorable clinical-imaging profile. Late-window treatment is based on tissue and outcome selection rather than a rigid 6-hour cutoff. Emergency carotid surgery is not the treatment for an intracranial terminus embolus.

26. A. Late-window thrombectomy selection relies on the presence of a treatable occlusion and a profile suggesting meaningful benefit from reperfusion. Imaging estimates established injury and remaining tissue at risk, while clinical factors establish severity and baseline function. Unknown or delayed onset no longer automatically excludes treatment.

27. E. Contemporary randomized evidence supports thrombectomy for appropriately selected patients with severe basilar artery occlusion, including selected presentations beyond the early window. A severe deficit, good baseline function, and limited established posterior-circulation injury favor intervention. Aspirin alone does not address the high-risk occlusion.

28. E. Thrombectomy benefit is greatest when there is a clinically meaningful acute syndrome, a causative occlusion, and enough viable tissue to make reperfusion useful. Incidental or chronic vascular findings without an acute disabling syndrome do not justify emergency thrombectomy. Extensive completed injury also reduces the potential benefit.

29. B. Current evidence and guideline recommendations support thrombectomy consideration in selected patients with a proximal dominant M2 occlusion, especially with disabling deficits, favorable imaging, and early presentation. Distal occlusion location is not an all-or-none concept; vessel dominance and clinical severity matter. Carotid surgery does not treat an acute M2 embolus.

30. E. For selected proximal dominant M2 occlusions, endovascular treatment can be reasonable. Transfer decisions should therefore be made early, before infarction progresses, and should not depend on thrombus migration or failure of intravenous thrombolysis. Timely access to endovascular expertise is the systems priority.

31. C. Endovascular benefit is well established for selected proximal large-vessel occlusions and has expanded to some additional occlusion sites, but routine thrombectomy for mild distal-medium-vessel occlusions is not established. Clinical severity, eloquence, vessel size, and trial evidence matter. A small distal occlusion with mild nondisabling symptoms should not be treated as equivalent to an ICA terminus occlusion.

32. C. Thrombectomy is an invasive reperfusion treatment whose benefit depends on a treatable acute occlusion and meaningful threatened function. Distal vessel caliber and lower deficit severity can reduce expected benefit and increase technical risk. Appropriate selection therefore balances anatomy, clinical disability, imaging, and evidence rather than angiographic abnormality alone.

33. E. Tandem occlusions are challenging but are common enough to be a recognized thrombectomy scenario. Endovascular teams can address the intracranial occlusion and, when necessary, the cervical lesion using individualized strategies. The cervical component does not automatically negate the benefit of reperfusion.

34. E. Acute tandem-lesion management is individualized because carotid stenting can improve access and flow but may require antiplatelet therapy that influences hemorrhagic risk. Some lesions need angioplasty or stenting, while others can be crossed or deferred. There is no universal requirement to stent or never stent.

35. C. ASPECTS is a 10-point semiquantitative score for early ischemic change in the MCA territory on noncontrast CT. Points are subtracted for affected regions, so a score of 8 reflects relatively limited established change. It is not a hemorrhage, carotid-stenosis, or hematoma-volume scale.

36. A. ASPECTS summarizes early ischemic burden and helps estimate how much tissue is already injured. Modern thrombectomy evidence includes selected patients with lower scores, so ASPECTS should inform rather than dictate treatment in isolation. Occlusion site, time, baseline function, severity, and other imaging features remain important.

37. A. A perfusion mismatch reflects tissue that is markedly hypoperfused but not yet included in the estimated infarct core. This can identify patients with potentially salvageable penumbra for reperfusion

treatment, particularly in extended windows. Perfusion imaging is a selection tool, not a direct test of stroke mechanism.

38. B. Automated perfusion processing is useful but not perfect. Motion, delay, poor bolus, chronic stenosis, and algorithmic thresholds can distort estimates, so clinicians should review the source data and determine whether the maps fit the clinical and vascular findings. No single automated number should replace integrated interpretation.

39. D. After successful thrombectomy, excessive blood-pressure lowering can worsen outcome, and contemporary guidance cautions against intensive targets below 140 mm Hg as a routine strategy. Blood pressure should be controlled smoothly while avoiding both severe hypertension and hypotension. Reperfusion does not end the need for physiologic monitoring.

40. E. Even after angiographic reperfusion, microcirculatory flow can remain vulnerable and cerebral autoregulation may be impaired. Excessive systemic pressure reduction can therefore decrease perfusion to injured tissue. The goal is stable, individualized control rather than reflexive intensive lowering.

41. E. Malignant hemispheric infarction can produce life-threatening edema and herniation. Decompressive hemicraniectomy performed in an appropriate time window can substantially reduce mortality and improve the chance of survival with meaningful function in selected patients. Steroids do not treat cytotoxic stroke edema, and anticoagulation or carotid surgery does not reverse the mass effect.

42. B. Large territorial infarcts can deteriorate over the first several days as edema evolves. Early recognition, intensive monitoring, repeat imaging when indicated, and timely neurosurgical involvement are critical because decompression works best before irreversible herniation injury. Routine steroids and indiscriminate prophylactic hyperosmolar therapy do not replace surveillance.

43. A. Swollen cerebellar infarction can rapidly compress the brainstem and obstruct the fourth ventricle. Surgical decompression is lifesaving in deteriorating patients, and ventricular drainage may be required for hydrocephalus, typically in conjunction with definitive decompression when posterior-fossa mass effect is present. Observation alone is unsafe in a deteriorating patient.

44. B. Obstructive hydrocephalus is only one component of dangerous cerebellar swelling. The swollen tissue itself can compress the brainstem, so CSF diversion may not address the principal mass effect. Neurosurgical management must consider decompression of the posterior fossa, not just ventricular size.

45. D. This is the classic locked-in syndrome from a ventral pontine lesion, often due to basilar artery ischemia. Consciousness and vertical eye movements are preserved because the reticular activating system and vertical gaze pathways are relatively spared, while corticospinal and corticobulbar fibers are interrupted. The syndrome must be distinguished from coma.

46. D. Locked-in patients can be fully conscious despite profound paralysis. Detecting reproducible vertical eye movement or blinking to command changes prognosis, communication, and ethical

decision-making. A careful consciousness assessment is therefore essential before labeling the patient comatose or unresponsive.

47. B. A small internal-capsule infarct producing a pure motor syndrome is a classic lacunar stroke pattern. The usual mechanism is disease of a penetrating artery from lipohyalinosis or microatheroma, strongly associated with chronic hypertension and diabetes. Cortical embolic syndromes typically add aphasia, neglect, visual, or other cortical signs.

48. D. Noncardioembolic lacunar stroke is generally managed with antiplatelet therapy and aggressive vascular risk-factor control. Long-term aspirin plus clopidogrel increases bleeding without providing sufficient recurrent-stroke benefit in this setting. Anticoagulation is not indicated without a separate cardioembolic indication.

49. B. A small thalamic infarct can interrupt ascending sensory pathways and produce a pure hemisensory syndrome without weakness or cortical dysfunction. The pattern is a classic lacunar presentation. Other listed syndromes would produce additional brainstem, cortical, frontal, or cerebellar findings.

50. B. Central post-stroke pain can follow thalamic or other lesions of central somatosensory pathways. Loss of normal sensory input and maladaptive central processing can create painful dysesthesia, allodynia, or hyperalgesia in the affected body region. The symptoms are neuropathic rather than evidence of recurrent infarction with each painful stimulus.

51. D. Alexia without agraphia classically results from dominant occipital cortex injury plus disruption of visual information crossing through the splenium to language areas. Writing remains intact because the language network itself can still generate written output, but visual input cannot reach it normally. The syndrome localizes to the dominant posterior cerebral artery territory.

52. A. The dominant occipital lesion causes a right visual field defect, while splenial involvement disconnects visual information from the intact nondominant occipital cortex from the dominant language network. This disconnection impairs reading while sparing the ability to formulate and write language.

53. A. Anterior cerebral artery infarction affects the medial frontal and parietal lobes, including leg motor-sensory areas and circuits involved in initiation and motivation. Leg-predominant weakness plus abulia strongly suggests ACA territory ischemia. The posterior-circulation territories listed produce different brainstem, cerebellar, or visual syndromes.

54. E. Bilateral medial frontal and anterior cingulate injury can cause profound abulia or akinetic mutism. The problem is impaired initiation rather than primary weakness or reduced arousal. ACA territory infarction can therefore produce striking behavioral change out of proportion to elementary motor deficits.

55. C. An inferior-division dominant MCA infarct can affect posterior temporal and inferior parietal language areas, producing fluent aphasia with impaired comprehension. Superior-division dominant

MCA infarction more often causes nonfluent aphasia with face-arm weakness. The absence of major weakness further supports an inferior-division syndrome.

56. C. A proximal MCA stem occlusion can threaten broad lateral hemispheric cortex from both divisions as well as lenticulostriate territories, creating combined language, motor, sensory, and visual deficits. A division-level occlusion typically produces a more restricted cortical syndrome.

57. A. Watershed infarcts occur in junctional zones between major arterial territories and can result from systemic hypoperfusion, especially when proximal arterial disease limits reserve. Bilateral proximal arm weakness can occur with ACA-MCA borderzone injury. The distribution and hypotensive trigger support a hemodynamic mechanism.

58. C. Limb-shaking TIA is a hemodynamic phenomenon caused by critically reduced cerebral perfusion, often from severe carotid occlusive disease. Postural precipitation and relief with recumbency are important clues. Management focuses on avoiding hypoperfusion and evaluating definitive vascular treatment rather than treating the movements as primary epilepsy.

59. C. Neck pain, partial Horner syndrome, and ipsilateral cervical ICA narrowing are classic for carotid dissection. The sympathetic fibers around the carotid can be disrupted, while artery-to-artery embolism or hemodynamic compromise can cause cerebral ischemia. A tapered or flame-shaped stenosis supports the diagnosis.

60. B. For most extracranial cervical artery dissections with ischemia, antithrombotic therapy is used to reduce recurrent embolic risk. Evidence has not established a universal superiority of anticoagulation over antiplatelet therapy, so choice is individualized according to thrombus burden, bleeding risk, infarct size, and other factors. Most dissections heal or remodel over time.

61. C. Vertebral artery dissection often presents with acute posterior neck or occipital pain followed by vertebrobasilar ischemic symptoms. A tapered stenosis or mural abnormality on vascular imaging supports the diagnosis. The combination of pain and focal posterior-circulation deficits is much more specific than isolated peripheral vertigo.

62. C. Most extracranial vertebral dissections with ischemia are managed medically with antithrombotic therapy and follow-up imaging. Endovascular treatment is reserved for selected circumstances such as recurrent ischemia despite therapy, critical hemodynamic compromise, or specific anatomic complications. Routine stenting is unnecessary.

63. A. Fibromuscular dysplasia is a nonatherosclerotic, noninflammatory arteriopathy that commonly affects renal and cervicocephalic arteries. The classic angiographic appearance is a string-of-beads pattern from alternating stenosis and dilatation. It is associated with arterial dissection and aneurysm.

64. B. Fibromuscular dysplasia can involve multiple vascular beds and is associated with aneurysm and dissection. Evaluation often extends beyond the initially identified artery, particularly to renal and

intracranial circulation. Treatment depends on manifestations rather than the angiographic diagnosis alone.

65. E. A carotid web is a shelf-like intimal projection at the carotid bulb, often considered an intimal variant of fibromuscular dysplasia. It can create flow stasis and recurrent artery-to-artery embolism despite little conventional stenosis. Recurrent ipsilateral embolic stroke in a younger patient is a classic clinical clue.

66. A. Recurrent stroke associated with an ipsilateral carotid web suggests a persistent embolic source. Although evidence is less extensive than for atherosclerotic carotid stenosis, revascularization is often considered in selected recurrent cases. The lesion is structural rather than inflammatory.

67. B. Symptomatic severe extracranial carotid stenosis carries substantial early recurrence risk. Carotid endarterectomy provides clear benefit in appropriately selected patients and is generally performed relatively early after a nondisabling event once the patient is stable. Intensive medical therapy remains essential before and after surgery.

68. E. For many standard-risk symptomatic patients, especially older individuals, carotid endarterectomy has a lower periprocedural stroke risk than transfemoral stenting. Stenting can be useful when surgery is high risk because of prior radiation, restenosis, inaccessible anatomy, or other factors. Choice depends on age, anatomy, comorbidity, and local expertise.

69. D. Prior neck radiation can create hostile surgical anatomy and increase cranial nerve and wound complications. Carotid stenting may therefore be a reasonable alternative when revascularization is indicated and endovascular anatomy is suitable. The underlying symptomatic stenosis still requires aggressive medical therapy.

70. E. Carotid stents are thrombogenic surfaces, so peri-procedural antiplatelet therapy is important to reduce acute thrombotic complications. The exact regimen depends on procedure type and patient risk, but stopping antiplatelet therapy is generally counterproductive. Statins and other vascular risk-factor treatments remain part of comprehensive care.

71. E. Symptomatic intracranial atherosclerotic disease is initially treated with aggressive medical therapy, including antiplatelet therapy, high-intensity lipid lowering, blood-pressure control, diabetes and lifestyle management, and short-term dual antiplatelet therapy in selected severe recently symptomatic stenosis. Routine early intracranial stenting has higher procedural risk and is not first-line.

72. D. Intracranial stenting is technically feasible, but early randomized trials demonstrated excess periprocedural stroke and hemorrhage compared with intensive medical treatment. Selected rescue or refractory situations may be considered at experienced centers, but routine first-line stenting is not justified.

73. A. Early recurrence risk after a high-risk TIA is greatest in the first days to weeks. Short-term aspirin plus clopidogrel is beneficial in appropriately selected noncardioembolic high-risk TIA or minor stroke, but prolonged dual therapy increases bleeding. Etiologic evaluation must proceed rapidly in parallel.

74. B. The balance of benefit and harm changes over time. Dual therapy meaningfully lowers very early recurrent ischemic events in selected patients, but continued exposure increases major bleeding without proportional additional stroke prevention. The strategy is therefore intentionally time-limited.

75. C. High-intensity statin therapy reduces recurrent vascular events after atherosclerotic ischemic stroke, and lower LDL targets are commonly pursued in patients with established atherosclerotic disease. If LDL remains above goal despite maximally tolerated statin, additional agents such as ezetimibe or a PCSK9 inhibitor may be considered.

76. A. Residual LDL elevation despite high-intensity statin therapy is an opportunity for additional risk reduction. Ezetimibe is commonly added first, and PCSK9-directed therapy can be considered in very high-risk patients who remain above target. Secondary prevention should be proactive rather than waiting for recurrence.

77. D. Hypertension is one of the most important modifiable risk factors for both ischemic stroke and intracerebral hemorrhage. After the acute phase, sustained blood-pressure reduction substantially lowers recurrence risk, including in small-vessel disease. Targets are individualized, but values around or below 130/80 mm Hg are commonly pursued when tolerated.

78. E. Secondary prevention depends on consistent long-term risk-factor control, not episodic emergency treatment. Home measurements, adherence assessment, lifestyle interventions, and rational medication adjustment help achieve stable control. Both systolic and diastolic pressure matter, with systolic hypertension particularly important in older adults.

79. D. Atrial fibrillation is a major cardioembolic source, and oral anticoagulation is substantially more effective than antiplatelet therapy for secondary prevention. Direct oral anticoagulants are generally preferred in nonvalvular AF because of favorable efficacy and intracranial bleeding profiles. Timing after the acute stroke is individualized to infarct size and hemorrhagic risk.

80. C. Anticoagulation timing after AF-related stroke is a risk-balance decision. Small mild infarcts with no hemorrhagic transformation can often be anticoagulated relatively early, whereas large infarcts or hemorrhagic transformation usually justify more delay. A rigid one-size-fits-all interval is not appropriate.

81. A. Paroxysmal atrial fibrillation can be intermittent and missed on short monitoring. Longer external monitoring or an implantable cardiac monitor increases diagnostic yield after otherwise unexplained embolic stroke. Demonstrating AF matters because it changes secondary prevention from antiplatelet therapy to anticoagulation.

82. C. Occult AF may occur rarely and without symptoms, so detection probability rises with monitoring duration. Implantable monitors can surveil for months to years and identify episodes that shorter studies miss. They are diagnostic tools; they do not directly prevent arrhythmia or replace structural cardiac evaluation.

83. C. In appropriately selected younger patients with a nonlacunar cryptogenic stroke and high-risk PFO features, closure reduces recurrent stroke compared with medical therapy alone. Selection is critical because PFOs are common incidental findings. The workup must exclude more plausible stroke causes before attributing causality.

84. D. PFO causality is more plausible when the patient is younger, the infarct is embolic, alternative causes are absent, and high-risk anatomic features are present. Competing mechanisms such as atrial fibrillation, severe carotid disease, or lacunar small-vessel disease make an incidental PFO less likely to be responsible.

85. C. A mobile LV thrombus after anterior MI is a direct systemic embolic source. Anticoagulation is used to prevent recurrent embolism, with timing after stroke individualized to infarct size and hemorrhagic risk and duration guided by thrombus resolution and ventricular function. Antiplatelet therapy alone is not equivalent for an established ventricular thrombus.

86. E. Ventricular thrombus formation follows Virchow principles: blood stasis from akinesis, endocardial injury, and a prothrombotic milieu. A thrombus in the left heart can embolize through the aorta to cerebral arteries. The mechanism is cardioembolic rather than carotid or venous.

87. C. Mechanical prosthetic valves require vitamin K antagonist anticoagulation; direct oral anticoagulants are not appropriate substitutes. After an ischemic stroke, the timing of resumption depends on infarct size and bleeding risk, but the long-term indication remains strong. Aspirin alone does not provide adequate protection for a mechanical mitral valve.

88. C. This scenario creates competing high-stakes risks. Delayed anticoagulation increases valve thrombosis and systemic embolism, but restarting too early can worsen hemorrhagic transformation. Management is individualized with serial imaging and multidisciplinary input rather than abandoning the mechanical-valve indication.

89. B. Ischemic stroke from infective endocarditis carries increased hemorrhagic risk because septic emboli can injure arterial walls and coexist with mycotic aneurysms or hemorrhagic transformation. Routine intravenous thrombolysis is therefore avoided. Mechanical thrombectomy may still be considered in selected large-vessel occlusion cases.

90. A. The bleeding concerns that preclude routine intravenous thrombolysis do not automatically preclude mechanical clot retrieval. Selected endocarditis patients with disabling large-vessel occlusion can undergo thrombectomy after careful assessment. Infection treatment and cardiac management remain essential in parallel.

91. E. Persistent antiphospholipid antibodies plus clinical arterial thrombosis fulfill the core pattern of antiphospholipid syndrome. It can occur with SLE or independently and can cause arterial or venous thrombotic events. Repeated embolic-appearing strokes in a young patient are an important clue.

92. B. High-risk antiphospholipid syndrome with arterial thrombosis is generally managed with warfarin-class anticoagulation rather than relying on a direct oral anticoagulant, particularly in triple-positive disease. Intensity and adjunct antiplatelet decisions depend on recurrence and bleeding risk. The antibodies represent a clinically meaningful prothrombotic state.

93. B. Advanced adenocarcinoma can produce a marked hypercoagulable state and sterile valvular vegetations known as nonbacterial thrombotic endocarditis. Emboli can shower multiple cerebral territories, producing recurrent multifocal infarction. The pattern is particularly suggestive when no infectious or conventional cardiac source is found.

94. A. NBTE reflects a systemic prothrombotic state. Anticoagulation is commonly used to reduce further embolization, while treatment of the underlying cancer is central to controlling the driver of thrombosis. Infection must be excluded, but sterile vegetations do not require prolonged antibiotics.

95. E. Acute ischemic stroke in sickle cell disease requires rapid reduction of circulating HbS and improvement of oxygen-carrying capacity, typically with exchange transfusion when feasible. This addresses the disease-specific pathophysiology and recurrence risk. Childhood stroke in sickle cell disease is not usually caused by atherosclerotic carotid plaque.

96. B. Transcranial Doppler identifies children with sickle cell anemia who have markedly elevated intracranial flow velocities and high stroke risk. Chronic transfusion therapy in this high-risk group dramatically reduces first-stroke occurrence. The strategy is one of the clearest examples of effective primary stroke prevention in a specific hematologic disease.

97. B. Pregnancy complicates risk assessment but does not make severe maternal ischemic stroke untreatable. Alteplase has minimal placental transfer, and thrombectomy can be performed with radiation-minimizing techniques when indicated. Decisions should weigh maternal neurologic benefit against obstetric bleeding and fetal considerations in a multidisciplinary manner.

98. C. Maternal neurologic outcome is central to fetal and maternal health. When thrombectomy is indicated, treatment should not be withheld solely because of pregnancy; fetal radiation exposure can be minimized and is generally far below deterministic-harm thresholds. Multidisciplinary planning should occur in parallel rather than delaying reperfusion.

99. C. Postpartum hypercoagulability, progressive headache, seizure, focal deficit, and a venous sinus filling defect are classic for cerebral venous thrombosis. Venous outflow obstruction can cause intracranial hypertension, venous infarction, and hemorrhage. Presentation often differs from abrupt arterial stroke.

100. B. Venous imaging is required to demonstrate the thrombosed sinus or cortical vein. CT venography and MR venography are standard approaches, with source-image review important because flow artifacts can mimic thrombosis. Routine EEG may evaluate seizures but does not diagnose the venous occlusion itself.

101. C. Hemorrhage caused by venous hypertension is not a reason to withhold anticoagulation in cerebral venous thrombosis. Anticoagulation prevents thrombus propagation and facilitates recanalization while treating the underlying prothrombotic state. The balance differs from a primary spontaneous intracerebral hemorrhage.

102. B. Most CVT responds to anticoagulation, but rare patients deteriorate from extensive thrombosis, mass effect, or refractory intracranial hypertension. Endovascular thrombectomy/thrombolysis or decompressive surgery can be considered as rescue therapy in selected life-threatening cases. These are not routine first-line interventions.

103. A. Low-molecular-weight heparin is commonly preferred for venous thrombosis during pregnancy because it is effective and does not cross the placenta. Warfarin is teratogenic during pregnancy, and direct oral anticoagulants lack sufficient pregnancy safety data. Antiplatelet therapy alone is inadequate for acute CVT.

104. C. Pregnancy-associated CVT is often a provoked event, so anticoagulation is usually continued for a defined period extending through the higher-risk postpartum interval. Longer treatment depends on persistent thrombophilia, recurrence, or other risk factors. Future pregnancy planning should include individualized recurrence-prevention counseling.

105. D. Recurrent thunderclap headache with multifocal reversible arterial narrowing strongly suggests RCVS. Common associations include vasoactive drugs, postpartum state, and sympathetic surges. Unlike primary CNS vasculitis, CSF is often normal or near-normal and the clinical onset is characteristically abrupt.

106. B. RCVS management is primarily supportive and trigger-directed. Calcium-channel blockers are often used for headache or vasoconstriction symptoms, although proof of outcome benefit is limited. Glucocorticoids can worsen RCVS and should not be reflexively used as if the syndrome were inflammatory vasculitis.

107. C. PACNS often presents subacutely with headache, encephalopathy, focal deficits, and multifocal ischemic abnormalities. CSF is commonly inflammatory, helping distinguish it from RCVS. Vascular imaging and vessel-wall MRI may support the diagnosis, but biopsy remains important when uncertainty is substantial.

108. B. Brain and leptomeningeal biopsy can demonstrate transmural vascular inflammation and help exclude infection, lymphoma, amyloid-related angiitis, and other mimics. Angiographic beading is not

specific, so tissue diagnosis is valuable when treatment would require prolonged immunosuppression and the clinical picture is uncertain.

109. B. Moyamoya is characterized by progressive stenosis or occlusion of the terminal ICAs and proximal branches with development of fragile basal collaterals. It can cause ischemic events from hypoperfusion and embolic mechanisms as well as intracranial hemorrhage from collateral rupture. The angiographic appearance is distinctive.

110. E. Moyamoya is a progressive flow-limiting arteriopathy, and surgical revascularization can augment cerebral blood flow and reduce future ischemic risk. Medical management alone may not correct the hemodynamic problem in symptomatic patients with impaired reserve. Avoiding dehydration and hypotension is also important.

111. E. In mild-to-moderate spontaneous ICH with systolic hypertension, early smooth reduction toward roughly 140 mm Hg can be beneficial and is generally safe, while excessive lowering below about 130 mm Hg may be harmful. Stable control with titratable agents is preferred to large fluctuations. Clinical context and extreme presenting pressures may require individualized targets.

112. A. Blood-pressure variability is associated with worse outcomes in ICH. Titratable intravenous therapy aims for gradual, stable reduction while avoiding both recurrent hypertensive surges and excessive hypotension. The goal is controlled hemodynamics, not simply achieving a single isolated measurement.

113. C. Warfarin-associated ICH requires rapid reversal. Four-factor PCC replaces vitamin K-dependent clotting factors quickly, while intravenous vitamin K provides sustained reversal as the PCC effect wanes. Delaying reversal permits continued hematoma expansion risk.

114. D. PCC acts rapidly but its infused factors have finite half-lives. Vitamin K addresses the underlying warfarin-mediated inhibition of clotting-factor synthesis, maintaining reversal after the replacement factors decline. The therapies are complementary rather than redundant.

115. D. Idarucizumab is a monoclonal antibody fragment that binds dabigatran and rapidly neutralizes its anticoagulant effect. Andexanet is directed against factor Xa inhibitors, protamine reverses heparin, and vitamin K reverses vitamin K antagonists. Rapid reversal is a priority in anticoagulant-associated ICH.

116. C. Dabigatran is more dialyzable than factor Xa inhibitors because of relatively low protein binding. Idarucizumab is first-line specific reversal, but dialysis can be considered in selected situations with renal failure or persistent high drug levels. This is an adjunctive rescue concept rather than routine management.

117. E. Apixaban is a direct factor Xa inhibitor. Andexanet alfa is a specific reversal agent, while four-factor PCC is also used in many protocols when specific reversal is unavailable or unsuitable. Idarucizumab is specific to dabigatran, vitamin K to warfarin, and protamine to heparins.

118. C. Anticoagulant-associated ICH can expand early, so reversal should be initiated promptly when meaningful drug activity is likely. Blood-pressure control, repeat imaging, neurosurgical assessment when relevant, and general critical care proceed in parallel. Waiting for documented expansion sacrifices preventable time.

119. C. Randomized evidence does not support routine platelet transfusion for spontaneous antiplatelet-associated ICH in patients not undergoing emergency surgery and suggests potential harm. Management instead emphasizes blood-pressure control, general neurocritical care, and treatment of specific complications. Operative contexts may require separate individualized hemostatic planning.

120. A. The evidence against routine platelet transfusion applies most directly to nonoperative spontaneous ICH. Emergency surgery creates a different hemostatic problem, and perioperative strategies should be individualized with the surgical team. The broader principle is to match reversal therapy to the clinical context rather than use a universal rule.

121. D. Cerebellar ICH can cause rapid deterioration through brainstem compression and obstructive hydrocephalus. Surgical evacuation is recommended for patients with neurologic decline, significant mass effect, hydrocephalus from ventricular obstruction, or sufficiently large hematomas. Posterior-fossa anatomy leaves little reserve for expansion.

122. C. The posterior fossa is a tight compartment. Hematoma and edema can quickly compress the fourth ventricle and brainstem, causing hydrocephalus and sudden neurologic decline. This anatomy lowers the threshold for surgical intervention when mass effect is clinically significant.

123. D. Chronic hypertension produces lipohyalinosis and fibrinoid change in deep penetrating arterioles. Typical hemorrhage sites include basal ganglia, thalamus, pons, and cerebellum. Lobar hemorrhage in an older patient is more suggestive of cerebral amyloid angiopathy, though hypertension can coexist.

124. E. Hypertension is the dominant modifiable risk factor for recurrent deep ICH. Long-term pressure control reduces both hemorrhagic and ischemic vascular events. Antithrombotics may be indicated for other reasons but are not substitutes for blood-pressure treatment.

125. B. Strictly lobar microbleeds, cortical superficial siderosis, and lobar ICH in an older adult strongly suggest cerebral amyloid angiopathy. Amyloid deposition weakens small cortical and leptomeningeal vessels and creates a high risk of recurrent lobar hemorrhage. Cognitive impairment commonly coexists.

126. A. CAA carries substantial recurrent lobar hemorrhage risk, especially when superficial siderosis or numerous lobar microbleeds are present. Blood-pressure control is important, and antithrombotic therapy should be used only when a sufficiently strong competing indication exists. Decisions require individualized ischemic-versus-hemorrhagic risk assessment.

127. A. In older adults, nonaneurysmal convexity SAH and stereotyped spreading sensory or motor episodes can be manifestations of cerebral amyloid angiopathy. These transient focal neurologic

episodes may reflect cortical irritation from superficial bleeding and can mimic TIA. Recognizing the pattern prevents reflexive escalation of antithrombotic therapy.

128. C. CAA-related episodes can be caused by superficial hemorrhagic pathology rather than ischemia. Increasing antiplatelet or anticoagulant intensity without recognizing the mechanism may expose the patient to additional intracranial bleeding. MRI blood-sensitive sequences are therefore important in the diagnostic evaluation.

129. E. An AVM is a high-flow arteriovenous shunt with a nidus, feeding arteries, and early venous drainage. It can present with intracranial hemorrhage, seizure, headache, or focal neurologic deficit. Angiography defines architecture important for treatment planning.

130. A. Unruptured AVM management is nuanced because procedural risks can exceed natural-history risk in some patients, while selected lesions may be reasonably treated. Decisions integrate prior hemorrhage, size, location, venous drainage, symptoms, age, and the risks of surgery, embolization, and radiosurgery. There is no one-size-fits-all strategy.

131. B. Diffuse basal cisternal SAH with a thunderclap headache is aneurysmal until proved otherwise. CT angiography or catheter angiography is used to identify the responsible aneurysm. Early complications include rebleeding, hydrocephalus, seizures, and delayed cerebral ischemia.

132. C. Most saccular aneurysms arise near branch points of the circle of Willis. When they rupture, arterial blood rapidly spreads through adjacent basal subarachnoid cisterns and can extend into fissures and ventricles. The distribution therefore reflects the arterial location and communication of CSF spaces.

133. B. Nimodipine is standard therapy after aneurysmal SAH because it improves neurologic outcome, particularly by reducing the impact of delayed cerebral ischemia. Its benefit is not explained solely by reversing angiographic vasospasm. Hypotension should be avoided so the drug can be continued when possible.

134. B. DCI is a multifactorial brain-injury syndrome. Large-artery vasospasm is important, but microcirculatory impairment, autoregulatory failure, inflammation, and spreading depolarizations also contribute. This explains why treating vessel caliber alone does not fully prevent delayed ischemic injury.

135. A. Acute hydrocephalus after SAH can reduce consciousness and requires urgent CSF diversion when symptomatic. An external ventricular drain both treats elevated pressure and allows monitoring. The aneurysm should be secured promptly because CSF drainage can alter transmural pressure and rebleeding risk before definitive aneurysm treatment.

136. A. Blood and inflammatory debris can interfere with CSF circulation and absorption, especially at arachnoid granulations, and intraventricular clot can also cause obstructive components. The resulting ventricular enlargement raises intracranial pressure and can impair arousal.

137. E. Rebleeding risk is highest early after aneurysmal SAH and is associated with major mortality. Prompt aneurysm occlusion by coiling or clipping prevents recurrent arterial bleeding. Choice of technique depends on aneurysm anatomy, patient factors, associated hematoma, and local expertise.

138. C. Initial hemostasis at a ruptured aneurysm is unstable. A platelet-fibrin clot can temporarily seal the defect, but systemic arterial pressure and endogenous fibrinolysis can disrupt it. Definitive exclusion of the aneurysm from the circulation is therefore critical.

139. B. Symptomatic delayed cerebral ischemia from vasospasm is treated by maintaining euvolemia and augmenting cerebral perfusion pressure when appropriate; refractory focal deficits may be treated with intra-arterial vasodilators or angioplasty. Hypovolemia and aggressive pressure lowering can worsen ischemia. Nimodipine is generally continued if tolerated.

140. A. Breakdown products from subarachnoid blood alter vascular smooth muscle, nitric oxide signaling, endothelin pathways, inflammation, and endothelial function. These processes promote delayed arterial constriction and impaired perfusion. The biology is more complex than a simple mechanical compression phenomenon.

141. C. Dural AV fistulas are abnormal shunts between meningeal arteries and dural venous sinuses or cortical veins. Cortical venous reflux is an important high-risk feature associated with intracranial hemorrhage and neurologic deficits. Pulsatile tinnitus is a common lower-risk presentation depending on location.

142. E. Dural fistulas with cortical venous drainage expose normally low-pressure veins to arterialized pressure and impaired outflow. Venous hypertension can cause congestion, edema, infarction, and rupture. This hemodynamic feature is central to risk stratification.

143. B. Cavernous malformations are low-flow vascular lesions composed of dilated capillary channels without intervening normal brain. Recurrent microhemorrhage produces mixed-age blood products and a hemosiderin rim on MRI. Angiography is often negative because flow is slow.

144. D. Cavernous malformations lack the rapid arteriovenous transit that makes AVMs conspicuous on catheter angiography. Their abnormal capillary spaces fill slowly, so MRI blood-product characteristics are far more informative than conventional angiography.

145. C. A developmental venous anomaly is a common benign variant in which small medullary veins converge into a collector vein, often described as a caput medusae. Most are asymptomatic and serve as normal venous drainage for surrounding brain. They should generally not be surgically removed.

146. B. DVAs are venous drainage variants rather than pathologic high-flow shunts. The surrounding brain may depend on the collector for normal outflow. Interrupting it can produce congestion and venous infarction, which is why incidental DVAs are typically left alone.

147. D. Complex aortic arch atheroma can be an embolic source, particularly when plaque is thick, ulcerated, or mobile. Embolic material can enter the brachiocephalic or carotid circulation and produce multifocal cortical infarcts. The mechanism is systemic arterial embolization.

148. A. Plaque surface disruption and mobile thrombus increase the likelihood that material will break free under pulsatile arterial flow. The cerebral circulation is exposed because the great vessels originate directly from the arch. Stable, small, calcified plaque generally has lower embolic potential than mobile complex plaque.

149. B. Large trials did not show that empiric anticoagulation improves outcomes for unselected ESUS compared with antiplatelet therapy and did increase bleeding in some settings. The appropriate response is continued etiologic evaluation and mechanism-specific treatment when a source is identified. ESUS is a syndrome, not a single mechanism.

150. C. ESUS groups patients by an imaging and workup phenotype rather than a single causal pathway. Different underlying sources have different biology and treatment responses, so one empiric therapy cannot be expected to benefit all patients. Precision depends on identifying the most likely mechanism.

151. C. Deep lacunes, deep microbleeds, and white-matter disease in a hypertensive patient reflect arteriolosclerosis of penetrating vessels. Lipohyalinosis, fibrinoid change, and microatheroma can produce ischemic lacunes and deep hemorrhagic lesions. The distribution distinguishes this pattern from the strictly lobar markers of CAA.

152. D. Chronic pressure and endothelial injury remodel small penetrating arteries, narrowing their lumens and weakening their walls. The same small-vessel process can therefore produce occlusive lacunes, white-matter ischemia, and deep microhemorrhage or ICH.

153. C. Cerebral amyloid angiopathy is caused by deposition of beta-amyloid in cortical and leptomeningeal vessel walls. Vessel fragility leads to lobar hemorrhage, cortical superficial siderosis, and lobar microbleeds. It also overlaps biologically with Alzheimer disease but is a distinct vascular pathology.

154. D. The anatomic distribution of amyloid deposition determines the hemorrhage pattern. CAA targets superficial cortical and leptomeningeal vessels, whereas hypertensive arteriopathy more strongly affects penetrating vessels supplying basal ganglia, thalamus, pons, and cerebellum.

155. D. Hemorrhagic transformation ranges from small petechial bleeding to space-occupying parenchymal hematoma within ischemic tissue. It is particularly common after large infarcts, cardioembolism, and reperfusion. Clinical significance depends on the type, volume, mass effect, and neurologic change.

156. C. Energy failure and inflammation injure endothelial tight junctions and the basal lamina. When blood flow returns, fragile microvessels may extravasate blood into the infarct. Larger infarct burden and severe barrier disruption increase the risk of clinically significant parenchymal hematoma.

157. A. Acute energy failure stops ATP-dependent ion transport, causing sodium and water to accumulate intracellularly in neurons and glia. This cytotoxic edema begins within minutes. Blood-brain barrier disruption later adds vasogenic components, contributing to mass effect in large infarcts.

158. B. Steroids are useful for vasogenic edema associated with tumors and some inflammatory disorders, but ischemic edema originates primarily from cellular ionic failure and evolving infarction. They therefore do not reverse the underlying mechanism and can cause systemic complications.

159. A. Collateral circulation can maintain perfusion to threatened tissue when an artery is occluded. Patients with strong leptomeningeal collaterals often have slower infarct growth, smaller cores, and a longer window in which reperfusion can help. Collateral quality is a major source of interpatient variability.

160. B. Pial arterial networks connect distal branches of major cerebral arteries. When one territory loses direct inflow, pressure gradients can drive blood from adjacent territories across these connections. This collateral flow can delay irreversible ischemic injury.

161. B. Active cancer can drive coagulation through tissue factor, mucins, inflammatory signaling, platelet activation, and endothelial injury. This may produce venous or arterial thrombosis and sterile valvular vegetations. Multifocal embolic infarcts and high coagulation markers are common clues.

162. C. Malignancy alters multiple components of hemostasis. Tumor products and host inflammatory responses can activate coagulation pathways and platelets, while endothelial injury further promotes thrombosis. The result can be recurrent embolic stroke even without a conventional cardiac source.

163. E. In sickle cell anemia, progressive intracranial arteriopathy and compensatory flow changes can produce high TCD velocities. Specific velocity thresholds identify children at high first-stroke risk and guide chronic transfusion therapy. The test is a validated physiologic screening tool.

164. D. Sickle cell vasculopathy is multifactorial. Recurrent endothelial injury from sickling, hemolysis, inflammation, abnormal nitric oxide biology, and hemodynamic stress promotes intimal hyperplasia and progressive stenosis, especially in distal ICAs and proximal cerebral arteries.

165. A. Watershed zones lie at the distal margins of major arterial territories and are vulnerable when systemic perfusion pressure falls. Severe global hypotension therefore can cause bilateral borderzone ischemia, especially in patients with impaired vascular reserve. The distribution reflects perfusion gradients rather than one focal embolus.

166. D. Pressure progressively falls along the arterial tree, making distal borderzone tissues most dependent on adequate systemic driving pressure and collateral support. When blood pressure drops severely, these regions cross ischemic thresholds before better-perfused central territories.

167. E. The artery of Percheron is a single perforating artery arising from one PCA that supplies both paramedian thalami and sometimes the rostral midbrain. Its occlusion produces a distinctive bilateral thalamic syndrome with altered consciousness, memory impairment, and vertical gaze abnormalities.

168. D. In the Percheron configuration, bilateral paramedian thalamic perfusion depends on one arterial trunk. Occlusion therefore produces symmetric lesions from a single focal event, an important clue when vascular imaging does not show a large proximal occlusion.

169. A. Anterior spinal artery infarction damages the anterior two-thirds of the cord, affecting corticospinal and spinothalamic pathways while often sparing dorsal-column vibration and proprioception. Abrupt onset and diffusion restriction support a vascular rather than inflammatory or compressive cause.

170. D. The anterior spinal artery is reinforced by segmental radiculomedullary vessels, including the artery of Adamkiewicz in the thoracolumbar region. Disruption of these feeders or systemic hypoperfusion can critically reduce cord perfusion, particularly in watershed segments.

171. E. Concentric vessel-wall enhancement in the right clinical context can support inflammatory vasculitis, particularly when accompanied by inflammatory CSF and multifocal ischemia. Vessel-wall MRI is not perfectly specific, so infection, atherosclerosis, RCVS, and other mimics must still be considered.

172. B. Inflammatory infiltration can disrupt endothelial function, thicken and weaken vessel walls, promote thrombosis, and narrow lumens. Depending on vessel size and severity, the result can be multifocal infarction, hemorrhage, or encephalopathy. This mechanism distinguishes vasculitis from purely embolic disease.

173. A. A thunderclap headache is a vascular emergency until dangerous secondary causes are excluded. A normal examination does not rule out subarachnoid hemorrhage, and CT sensitivity declines as time from onset increases. When suspicion remains after a negative CT obtained outside the highest-sensitivity early window, additional evaluation such as lumbar puncture or vascular imaging may be necessary.

174. C. RCVS is diagnosed by demonstrating segmental cerebral arterial narrowing in the appropriate clinical context and by showing reversibility over time. CTA or MRA is a practical initial study, though early imaging can be normal because vasoconstriction may evolve. Catheter angiography is reserved for selected uncertain or severe cases.

175. A. Removing vasoactive triggers is central in RCVS. Supportive care, pain control, avoidance of severe hypertension or hypotension, and close monitoring for stroke or hemorrhage are appropriate; calcium-channel blockers are often used for symptoms. Glucocorticoids are not routine therapy and may worsen the course.

176. D. Todd paresis is a transient focal neurologic deficit after a seizure and can mimic acute stroke. The witnessed focal motor seizure, short-lived weakness, and complete recovery support a postictal mechanism. In real-time acute care, stroke must still be excluded when uncertainty remains because seizure and stroke can coexist.

177. A. After acute brain injury, seizures may be electrographic with minimal motor manifestations. Continuous EEG has higher yield than a short routine recording when altered mental status persists or subtle repetitive movements raise concern. Vascular and cardiac tests do not directly detect seizure activity.

178. E. Early seizures are acute symptomatic events provoked by recent brain injury and do not automatically establish epilepsy. Long-term treatment depends on recurrence risk, seizure burden, cortical injury, EEG findings, and patient factors. Routine prophylactic antiseizure medication for all ischemic stroke patients is not recommended.

179. D. Obstructive sleep apnea is common after stroke and is associated with hypertension, atrial fibrillation, recurrent vascular risk, and impaired recovery. Loud snoring, witnessed apneas, and daytime sleepiness are classic clues. Formal sleep testing is appropriate when clinically feasible.

180. E. Polysomnography measures airflow, respiratory effort, oxygen saturation, sleep stage, and related variables and remains the reference standard for detailed assessment of sleep-disordered breathing. Home sleep apnea testing can be appropriate in selected uncomplicated adults, but it provides less comprehensive information.

181. A. Positive airway pressure is standard therapy for clinically significant obstructive sleep apnea and can improve sleepiness and physiologic stress. Adherence often requires mask fitting, education, and follow-up. Treatment complements rather than replaces management of hypertension, weight, atrial fibrillation, and other vascular risks.

182. D. Infections can injure cerebral arteries directly or through secondary inflammation and produce multifocal stenosis, thrombosis, or aneurysm formation. In an immunocompromised patient with inflammatory CSF and multifocal vascular lesions, infectious vasculopathy is a key diagnostic category. Specific causes include varicella-zoster virus, syphilis, tuberculosis, fungal infection, and others depending on context.

183. A. VZV vasculopathy can occur without a rash. CSF PCR may be positive early but can become negative later, whereas intrathecal anti-VZV antibody can improve diagnostic sensitivity in subacute disease. Serum seropositivity alone is nonspecific because prior VZV exposure is common.

184. E. VZV vasculopathy is treated with intravenous acyclovir because active viral infection of the arterial wall drives the disease. Some clinicians add a short corticosteroid course when vessel-wall inflammation is substantial, but prolonged steroid monotherapy is inappropriate. Standard stroke prevention is added according to mechanism and bleeding risk.

185. E. Central cord syndrome typically follows cervical hyperextension, especially in a spondylotic canal, and produces disproportionately greater upper-extremity than lower-extremity weakness. Sensory and bladder dysfunction vary. The pattern is spinal rather than cerebral and can mimic bilateral anterior-circulation weakness.

186. B. Spinal cord diffusion restriction can support an acute infarct, though technical artifacts make interpretation more challenging than in brain MRI. T2 signal may be initially subtle and can evolve over time. Imaging also excludes compressive causes that require different urgent treatment.

187. D. Progressive neurologic deficit from an epidural hematoma is a surgical emergency. Rapid decompression can prevent irreversible cord injury, and anticoagulant reversal or correction of coagulopathy may be necessary. The management is fundamentally different from arterial ischemic stroke thrombolysis.

188. B. Transient painless monocular visual loss in an older vascular-risk patient is retinal TIA until proved otherwise. Embolism from ipsilateral carotid plaque is an important cause, though cardiac and ophthalmic etiologies also exist. The episode requires urgent vascular evaluation because early stroke risk can be substantial.

189. A. In continuous acute vestibular syndrome, a properly performed HINTS examination can help identify central causes such as cerebellar or brainstem stroke. A normal head impulse, direction-changing gaze-evoked nystagmus, or skew deviation is concerning for a central lesion. The examination should be used only by clinicians trained in its performance and interpretation.

190. D. Acute retinal artery occlusion is an ischemic emergency and a marker of systemic vascular risk. Selected very early patients may be considered for reperfusion protocols at expert centers, and all require urgent evaluation for carotid, cardiac, and inflammatory causes as appropriate. Routine delayed outpatient handling misses both vision and recurrent-stroke opportunities.

191. C. Macular sparing after PCA infarction is classically attributed to variable collateral or dual arterial supply of the occipital pole, often with contribution from MCA branches. The phenomenon is vascular-anatomic rather than evidence that central vision bypasses the visual cortex.

192. E. Wernicke encephalopathy is caused by thiamine deficiency and can present with any combination of confusion, gait ataxia, and ocular motor abnormalities. The classic triad is often incomplete, so treatment should not wait for laboratory confirmation. Hospitalized patients with malnutrition or alcohol use are at particular risk.

193. E. Metabolic abnormalities can mimic or worsen focal neurologic deficits, but they can also coexist with true stroke. The safest approach treats the reversible metabolic problem while continuing time-sensitive evaluation when a persistent focal syndrome remains. Diagnosis should not be anchored to one laboratory abnormality.

194. B. Thiamine should be given promptly in suspected deficiency, especially when carbohydrate administration may increase metabolic demand. However, emergent glucose for severe hypoglycemia should never be delayed. Parenteral thiamine is preferred initially because absorption may be unreliable in high-risk patients.

195. A. Vascular cognitive impairment from subcortical small-vessel disease often causes executive dysfunction, slowed information processing, impaired attention, gait disturbance, and mood change. The profile can differ from the prominent early episodic memory storage deficit of typical Alzheimer disease, although mixed pathologies are common in older adults.

196. D. Post-stroke cognitive disorders require assessment of multiple domains and real-world function. Brief screening can identify concern, while formal neuropsychological testing defines strengths and deficits and helps distinguish aphasia, neglect, mood, and executive dysfunction. Collateral history is often crucial because insight may be impaired.

197. E. Preventing additional cerebrovascular injury is central to management of vascular cognitive impairment. Blood-pressure control, diabetes management, physical activity, smoking cessation, lipid treatment, and mechanism-specific stroke prevention reduce future injury. Cognitive rehabilitation and treatment of mood, sleep, and functional problems are also important.

198. B. Aquaporin-4 IgG strongly supports neuromyelitis optica spectrum disorder, which commonly causes severe optic neuritis and longitudinally extensive myelitis. The disorder is immune-mediated and differs in treatment and prognosis from multiple sclerosis. Acute presentations can occasionally mimic vascular events when deficits are abrupt.

199. C. LGI1 encephalitis often presents with faciobrachial dystonic seizures, memory impairment, and hyponatremia. Antibody testing in serum and CSF supports the diagnosis, but results must be interpreted alongside the phenotype, MRI, and EEG because false positives and test limitations exist. Early recognition matters because immunotherapy can be highly effective.

200. E. Autoimmune encephalitis is treated with immunotherapy, often beginning with corticosteroids, IVIG, plasma exchange, or combinations depending on severity and antibody syndrome. Seizures and autonomic complications require concurrent treatment, and paraneoplastic antibodies or phenotypes prompt tumor evaluation. Delay can worsen long-term cognitive outcome.

201. B. Language is left-hemisphere dominant in the large majority of right-handed adults. The combination of nonfluent aphasia and right facial weakness localizes to dominant frontal cortex, typically in the left MCA superior division. Handedness is not an absolute rule, but it strongly informs pretest probability.

202. A. Hemispatial neglect is most strongly associated with nondominant, usually right, parietal and attention-network injury. Extinction and constructional deficits support a cortical attention syndrome

rather than primary visual or spinal dysfunction. These deficits may be underrepresented by motor-focused assessments.

203. A. Consciousness cannot be inferred from motor output alone. Locked-in syndrome can eliminate speech and limb movement while preserving awareness and vertical eye movements. Demonstrating command-following through an alternative motor channel is therefore critical.

204. B. CTA rapidly identifies large-vessel occlusion, tandem lesions, stenosis, and relevant vascular anatomy and can be obtained immediately after noncontrast CT. Noncontrast CT is essential for hemorrhage exclusion but does not reliably define all arterial occlusions. Rapid vascular imaging is central to thrombectomy triage.

205. C. Transesophageal echocardiography places the probe close to the left atrium, interatrial septum, valves, and thoracic aorta, improving visualization of several potential embolic sources. It is more invasive than transthoracic echocardiography and should be used when the result can change management.

206. D. Diffusion restriction with low ADC reflects restricted water motion from cytotoxic cellular swelling and is a sensitive marker of acute ischemic injury. Interpretation still requires clinical correlation because seizures, abscess, and other processes can also restrict diffusion. Chronic infarcts usually show facilitated rather than restricted diffusion.

207. E. Smoking cessation materially lowers recurrent vascular risk. Combining counseling with pharmacotherapy such as nicotine replacement, varenicline, or bupropion generally improves quit rates over advice alone. Treatment should address dependence directly rather than compensating with additional antithrombotic medication.

208. B. Aphasia affects communication but does not automatically eliminate comprehension or decision-making capacity. Supported communication, yes/no formats, gestures, writing, pictures, and extra time can reveal preferences and understanding. Family members are valuable partners, but the patient should remain the primary person addressed whenever possible.

209. B. High-quality prognostic communication is transparent about both evidence and uncertainty. Families benefit from ranges of possible functional outcomes, factors that will clarify prognosis, and scheduled reassessment. False certainty can distort decisions, while refusing to discuss prognosis abandons a central clinical need.

210. C. Decision-making capacity is task-specific and depends on understanding, appreciation, reasoning, and ability to communicate a choice, not on agreeing with clinicians. A capable patient may refuse recommended treatment. The team should document informed refusal and continue other appropriate care.

211. C. Near misses reveal system vulnerabilities and are valuable patient-safety data. Professional practice includes transparent internal reporting, factual documentation when appropriate, and

participation in process improvement. A nonpunitive safety culture focuses on preventing recurrence while maintaining accountability.

212. A. Financial and professional conflicts must be disclosed and managed so that patient welfare and voluntary research participation remain primary. Incapacitated patients require legally appropriate surrogate consent or an approved emergency research pathway. Clinical treatment consent does not automatically authorize unrelated research participation.

213. B. DNR status concerns cardiopulmonary resuscitation unless broader treatment limits are explicitly documented. Patients can simultaneously decline CPR and request antibiotics, stroke prevention, rehabilitation, or other treatments consistent with their goals. Orders should be interpreted according to the actual preferences they represent.

214. E. Quality improvement begins by defining the performance gap and understanding the process that produces it. Time-stamped data can reveal delays in triage, imaging, laboratory decisions, consent, medication preparation, or communication. Interventions should then target the actual bottlenecks and be remeasured.

215. B. Improvement work should measure both the intended benefit and unintended harms. Door-to-imaging and door-to-needle times are process measures, while symptomatic hemorrhage, inappropriate activations, and outcomes can serve as balancing or outcome measures. Repeated measurement distinguishes durable improvement from anecdote.

216. C. Practice-based learning uses real clinical outcomes to identify patterns, compare care with evidence, and improve future decisions. Case review may reveal failures in acute vestibular examination, imaging selection, escalation, or supervision. Targeted change is more useful than blame or indiscriminate testing.

217. B. Large-vessel occlusion requires parallel systems of care. Intravenous thrombolysis should not delay transfer for endovascular evaluation, and transfer should not wait for proof of thrombolysis failure. Prearranged protocols reduce door-in-door-out time and preserve the chance for thrombectomy.

218. E. Equitable stroke systems require deliberate regional design. Tele-stroke consultation, EMS destination protocols, rapid imaging transfer, clear acceptance pathways, and monitoring of treatment times can reduce geographic and demographic barriers. Measuring access is essential for knowing whether interventions work.

219. C. Reliable image transfer is a process-design problem. Standardized electronic exchange, clear ownership, and a transfer checklist can eliminate recurrent delays and unnecessary repeat imaging. Source images are often essential for thrombectomy selection and procedural planning.

220. D. Resource scarcity requires explicit, ethically defensible allocation based on urgency, benefit, and feasible alternatives. Time-critical stroke vascular imaging can directly determine access to

thrombectomy and often has no equivalent substitute. Transparent criteria reduce arbitrary or inequitable decisions.