COMPREHENSIVE MOCK EXAM 9

Supplemental to: Vascular Neurology Board Review 2026-2027

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ABPN Vascular Neurology Certification Examination — 220 questions, computer-based single-best-answer format, domain-weighted per the official content specifications

Board-Review Questions

1. A 69-year-old woman develops sudden global aphasia and right hemiplegia 80 minutes ago. Noncontrast CT shows no hemorrhage, and CTA demonstrates a left internal carotid terminus occlusion. She has no thrombolysis contraindication and is being prepared for thrombectomy. Which treatment sequence is most appropriate?

A. Lower systolic pressure below 130 mm Hg before initiating either intravenous or endovascular reperfusion therapy

B. Defer thrombolysis until thrombectomy is completed and give it only if substantial occlusion remains

C. Observe for spontaneous improvement because proximal occlusions frequently recanalize without reperfusion therapy

D. Give aspirin immediately and postpone endovascular therapy until a repeat examination documents persistent disability

E. Administer intravenous thrombolysis promptly and continue directly to endovascular thrombectomy without waiting for improvement

2. A 76-year-old man presents 3 hours after abrupt left facial weakness and dysarthria. His NIHSS score is 3, but the residual dysarthria is mild and does not interfere with communication or swallowing. CT shows no hemorrhage and CTA shows no occlusion. Which acute strategy best reflects current practice?

A. Use early antiplatelet therapy rather than routine intravenous thrombolysis for a clearly nondisabling minor stroke

B. Withhold antithrombotic therapy for 24 hours because a small infarct may not yet be visible on CT

C. Arrange emergent cerebral angiography because a low NIHSS score can conceal a distal thrombectomy target

D. Give intravenous thrombolysis because any measurable neurologic deficit is automatically considered disabling

E. Begin therapeutic heparin because minor stroke has the highest short-term risk of artery-to-artery embolism

3. A 64-year-old woman has isolated severe expressive aphasia 2 hours after onset. Her NIHSS score is only 4, CT excludes hemorrhage, and no contraindication to thrombolysis is present. Which principle should determine treatment?

A. Only motor deficits qualify as disabling when deciding whether to give an intravenous thrombolytic

B. The aphasia is disabling despite the low NIHSS score, so rapid intravenous thrombolysis should be considered

C. Advanced perfusion imaging is required before thrombolysis whenever the NIHSS score is below 6

D. Thrombolysis should be postponed until the aphasia persists for at least 60 minutes without improvement

E. A score below 5 defines a nondisabling stroke and therefore excludes intravenous thrombolysis

4. A 71-year-old man with a disabling hemispheric stroke arrives 2 hours after onset. The stroke team can use either alteplase or tenecteplase, and the patient has no contraindication to either agent. Which statement best reflects the 2026 acute ischemic stroke guideline?

A. Tenecteplase is recommended only after failed alteplase because comparative trials did not support first-line use

B. Alteplase remains the only guideline-endorsed thrombolytic because tenecteplase is restricted to clinical trials

C. Either alteplase or tenecteplase is an accepted intravenous thrombolytic option within the 4.5-hour window

D. Tenecteplase is preferred only when symptom onset is unknown and perfusion mismatch is demonstrated on MRI

E. Alteplase should be avoided in thrombectomy candidates because its infusion interferes with endovascular workflow

5. An otherwise eligible patient with acute ischemic stroke receives tenecteplase. Which regimen is the standard stroke dose used in contemporary practice?

A. Tenecteplase 1.00 mg/kg as a single intravenous bolus, with a maximum dose of 100 mg

B. Tenecteplase 0.10 mg/kg as a 60-minute infusion, with a maximum dose of 50 mg

C. Tenecteplase 0.40 mg/kg as a single intravenous bolus, with a maximum dose of 40 mg

D. Tenecteplase 0.90 mg/kg with ten percent as a bolus and the remainder over one hour

E. Tenecteplase 0.25 mg/kg as a single intravenous bolus, with a maximum dose of 25 mg

6. A 58-year-old man arrives 80 minutes after sudden aphasia and right hemiparesis. CT shows no hemorrhage, glucose and platelets are acceptable, and thrombolysis is planned. Repeated blood pressures are 190-194/110-114 mm Hg. What should be done next?

A. Use a rapidly titratable intravenous antihypertensive to reduce pressure below 185/110 mm Hg before thrombolysis

B. Give an oral long-acting antihypertensive and reconsider thrombolysis after several hours of observation

C. Exclude the patient permanently from thrombolysis because the presenting pressure exceeded the treatment threshold

D. Administer the thrombolytic immediately because treatment benefit outweighs this degree of hypertension

E. Lower systolic pressure below 120 mm Hg before thrombolysis to minimize the risk of reperfusion hemorrhage

7. A 59-year-old woman has an M2 occlusion and disabling aphasia. She receives intravenous thrombolysis, and her blood pressure is 176/101 mm Hg afterward. What blood-pressure target is generally used during the first 24 hours after thrombolysis?

A. Permit blood pressure up to 220/120 mm Hg because autoregulation is impaired after thrombolysis

B. Maintain mean arterial pressure below 60 mm Hg to reduce the risk of hemorrhagic transformation

C. Maintain blood pressure below 180/105 mm Hg while avoiding unnecessary intensive lowering

D. Target systolic blood pressure below 140 mm Hg even after complete reperfusion because lower is always better

E. Maintain systolic blood pressure below 120 mm Hg regardless of recanalization or neurologic examination

8. A patient has complete TICI 3 reperfusion after thrombectomy for an M1 occlusion. The postprocedure systolic blood pressure is 154 mm Hg and the examination is stable. Which blood-pressure strategy is most appropriate?

A. Avoid routine intensive lowering below 140 mm Hg solely because complete reperfusion was achieved

B. Immediately lower systolic pressure to 100-110 mm Hg because complete reperfusion abolishes perfusion dependence

C. Allow systolic pressure to remain above 220 mm Hg because thrombectomy removes the risk of hemorrhagic transformation

D. Induce hypertension with vasopressors to a systolic pressure above 200 mm Hg after every successful thrombectomy

E. Use nitroprusside to reduce systolic pressure below 120 mm Hg during the first postprocedural hour

9. A 68-year-old woman awakens with dense right hemiparesis. She was last known well 8 hours earlier. MRI shows a small DWI lesion with minimal corresponding FLAIR signal, and CTA shows no thrombectomy target. Which finding is most relevant to possible intravenous thrombolysis?

A. Wake-up stroke requires a witnessed onset time before any intravenous thrombolytic can be considered

B. The absence of a thrombectomy target makes all reperfusion treatment inappropriate after 4.5 hours

C. A normal serum glucose level proves that the infarct is too old for intravenous thrombolytic treatment

D. The low FLAIR signal indicates completed infarction and therefore predicts no benefit from reperfusion therapy

E. A favorable DWI-FLAIR mismatch can identify selected unknown-onset patients for extended-window thrombolysis

10. A 62-year-old man presents 6 hours after a witnessed onset with disabling aphasia. CTA shows no large-vessel occlusion, while perfusion imaging demonstrates a small ischemic core and substantial salvageable tissue. Which approach is most appropriate?

A. Begin full-dose heparin because perfusion mismatch indicates an actively propagating intracranial thrombus

B. Consider extended-window intravenous thrombolysis using validated perfusion-based selection when no contraindication is present

C. Treat only with aspirin because a favorable perfusion profile is useful solely for prognostication, not selection

D. Withhold reperfusion because intravenous thrombolysis is never considered after 4.5 hours from a witnessed onset

E. Perform thrombectomy despite the absence of an occluded artery because perfusion mismatch alone defines an EVT target

11. A 74-year-old man develops acute basilar artery occlusion with quadriparesis and dysarthria. He arrives 11 hours after last known well with NIHSS 16 and no extensive irreversible brainstem infarction. Which treatment is most appropriate?

A. Proceed with endovascular thrombectomy because selected basilar occlusion with NIHSS at least 10 benefits within 24 hours

B. Defer thrombectomy unless the NIHSS exceeds 25 because lower scores do not represent severe posterior stroke

C. Give therapeutic heparin as definitive treatment because basilar thrombi are usually cardioembolic rather than atherosclerotic

D. Wait for coma before intervention because thrombectomy benefit in basilar occlusion is limited to deeply unconscious patients

E. Use aspirin alone because posterior-circulation thrombectomy has not shown benefit beyond six hours

12. A 79-year-old woman has an ICA terminus occlusion 7 hours after onset. CT perfusion shows a large ischemic core but also tissue at risk, and she was previously independent. Which statement best reflects modern thrombectomy selection?

A. Any large core is an absolute contraindication because reperfusion cannot improve outcome once ASPECTS is reduced

B. Large-core thrombectomy is recommended only when the patient is younger than 50 years and has no vascular risk factors

C. A large core favors intravenous heparin rather than thrombectomy because endovascular reperfusion increases edema uniformly

D. Thrombectomy should be delayed until 24 hours to determine whether the large core remains radiographically stable

E. A large ischemic core no longer automatically excludes thrombectomy when clinical and imaging features otherwise support treatment

13. A 66-year-old man with suspected large-vessel occlusion is picked up in a region where the nearest primary stroke center is 8 minutes away and an EVT-capable center is 15 minutes away. Interhospital transfer from the primary center is often delayed more than 90 minutes. Which prehospital principle is most appropriate?

A. Local system performance may justify direct transport to the EVT-capable center when transfer delays are substantial

B. Emergency medical services should wait for symptom improvement before choosing a destination for possible thrombectomy

C. Every suspected stroke must first go to the nearest thrombolysis-capable hospital regardless of local transfer performance

D. All suspected strokes should bypass nearby hospitals and travel to the most distant comprehensive center in the state

E. Prehospital triage should be based only on patient age because clinical LVO scales are too inaccurate to use

14. A city is considering a mobile stroke unit with onboard CT and thrombolysis capability. Which expected benefit best supports implementation when resources and case volume are appropriate?

A. Mobile units primarily improve outcomes by providing prolonged blood-pressure observation before hospital transport

B. Their main advantage is avoiding intravenous thrombolysis until patients reach a comprehensive stroke center

C. Mobile stroke units eliminate the need for hospital-based thrombectomy evaluation in patients with proximal occlusion

D. Earlier diagnosis and thrombolytic treatment can reduce treatment delays compared with conventional emergency transport

E. Onboard CT allows paramedics to exclude every stroke mimic without physician or telemedicine support

15. A 61-year-old woman with disabling stroke receives alteplase. A trainee suggests adding eptifibatide during the infusion to improve arterial recanalization. What is the best response?

A. Add therapeutic heparin routinely because thrombolysis alone cannot prevent immediate thrombus reformation in cerebral arteries

B. Add argatroban whenever NIHSS exceeds 10 because combined therapy has become standard for disabling stroke

C. Delay alteplase and use dual antiplatelet therapy instead because combination antithrombotic regimens are safer

D. Do not add routine adjuvant antithrombotic therapy because it has not improved outcomes with intravenous thrombolysis

E. Add eptifibatide routinely because glycoprotein IIb/IIIa inhibition reduces symptomatic intracranial hemorrhage after alteplase

16. A 57-year-old man presents with acute left hemiparesis 90 minutes after onset. Glucose is 278 mg/dL. CT shows no hemorrhage, and he otherwise meets criteria for reperfusion. Which glucose strategy is most appropriate?

A. Delay all reperfusion until glucose is lowered to exactly 100 mg/dL with a continuous insulin infusion

B. Maintain glucose above 250 mg/dL because hyperglycemia supplies metabolic substrate to the ischemic penumbra

C. Use intensive insulin to maintain 80-130 mg/dL because this improves functional outcome after acute ischemic stroke

D. Ignore glucose management entirely because dysglycemia has no association with outcome after cerebral ischemia

E. Treat marked hyperglycemia reasonably while avoiding intensive 80-130 mg/dL control that increases hypoglycemia risk

17. A 45-year-old woman presents with sudden right hemiparesis and aphasia. Point-of-care glucose is 34 mg/dL. What should be done first?

A. Correct the hypoglycemia immediately and reassess the focal neurologic deficits after glucose normalizes

B. Treat with intravenous antiseizure medication because hypoglycemia-induced focal deficits usually represent nonconvulsive status

C. Give intravenous thrombolysis immediately because profound hypoglycemia confirms acute cerebral ischemia

D. Start therapeutic anticoagulation because severe hypoglycemia is strongly associated with cardioembolic stroke

E. Withhold glucose until vascular imaging is complete because dextrose can worsen reperfusion injury in ischemic tissue

18. A 70-year-old man has a generalized seizure at the onset of a stroke syndrome. After the seizure ends, he remains aphasic with right hemiplegia, and CTA shows a left M1 occlusion. Which interpretation is best?

A. The patient should receive antiseizure medication and no vascular treatment until the weakness persists for 24 hours

B. Persistent aphasia after a seizure proves primary epilepsy even when vascular imaging shows an acute proximal occlusion

C. The residual deficit should be presumed Todd paresis because seizures at onset exclude reperfusion treatment

D. The M1 occlusion is probably chronic because acute arterial occlusions rarely provoke cortical seizures at symptom onset

E. Seizure at onset does not exclude acute ischemic stroke when persistent deficits match a documented arterial occlusion

19. A 63-year-old woman with sudden aphasia improves from NIHSS 12 to 4 while in the emergency department, but she still cannot understand spoken commands. How should this improvement affect thrombolysis eligibility?

A. Persistent disabling aphasia remains an indication to consider thrombolysis despite substantial spontaneous improvement

B. Aphasia becomes nondisabling once motor strength is normal, so antiplatelet therapy alone is required

C. Thrombolysis should be delayed until the deficit worsens again because improving stroke cannot recur after treatment

D. Only the admission NIHSS matters, so the current neurologic examination should not influence reperfusion decisions

E. Any improvement of more than 50 percent in NIHSS is an absolute contraindication to thrombolytic treatment

20. A 60-year-old man with acute ischemic stroke has a platelet count of 78,000/μL and a right M1 occlusion. CT shows no hemorrhage. Which management principle is most appropriate?

A. Exclude both thrombolysis and thrombectomy because the procedures share the same platelet-count contraindications

B. Give platelet transfusion solely to raise the count above 100,000/μL and then administer thrombolysis routinely

C. Avoid routine intravenous thrombolysis for severe thrombocytopenia while still evaluating the patient for mechanical thrombectomy

D. Proceed with full-dose intravenous thrombolysis because an M1 occlusion makes the platelet count clinically irrelevant

E. Use systemic heparin instead of reperfusion therapy because thrombocytopenia prevents procedural arterial access

21. A 72-year-old woman with atrial fibrillation took apixaban 3 hours ago and now has a disabling ischemic stroke. Renal function is normal, and the drug effect is presumed clinically significant. What is the safest general approach to intravenous thrombolysis?

A. Ignore the timing of the last dose if the NIHSS score is at least 10 and the CT shows no hemorrhage

B. Withhold routine intravenous thrombolysis while clinically significant factor Xa inhibitor activity is present

C. Give full-dose thrombolysis because direct factor Xa inhibitors do not increase intracranial bleeding during reperfusion therapy

D. Administer half-dose thrombolysis because reducing the fibrinolytic dose reliably offsets active anticoagulant effect

E. Reverse apixaban routinely and immediately give thrombolysis because this sequence is established as standard care

22. A 58-year-old man on warfarin for a mechanical valve presents 2 hours after onset of disabling hemiparesis. INR is 1.4. CT shows no hemorrhage. Which statement is most accurate regarding intravenous thrombolysis?

A. Warfarin use requires reversal with vitamin K before every thrombolysis decision even when the INR is low

B. A subtherapeutic INR at or below the accepted threshold may permit thrombolysis if all other criteria are satisfied

C. Mechanical-valve anticoagulation makes thrombectomy mandatory and prevents use of intravenous reperfusion in all cases

D. Any current warfarin prescription permanently excludes intravenous thrombolysis regardless of measured anticoagulant effect

E. Thrombolysis is safe only when the INR is exactly 1.0 because any elevation predicts symptomatic hemorrhage

23. A 67-year-old woman presents 2 hours after acute aphasia. She underwent an uncomplicated lumbar puncture yesterday. CT is negative for hemorrhage. Which approach best describes thrombolysis decision-making?

A. Recent procedures require individualized bleeding-risk assessment rather than automatic exclusion based only on the procedure name

B. Lumbar puncture has no bleeding relevance, so the recent procedure should never be considered in treatment decisions

C. Thrombolysis should be delayed for exactly seven days after every invasive procedure regardless of anatomic site

D. The recent lumbar puncture makes therapeutic heparin safer than thrombolysis for acute disabling ischemic stroke

E. Any lumbar puncture within 30 days is an absolute and universal contraindication to intravenous thrombolysis

24. A 73-year-old man presents with acute left neglect and hemiparesis. Noncontrast CT reveals a small amount of chronic-appearing subdural blood without mass effect that was documented unchanged on imaging one month ago. Which issue is most important before intravenous thrombolysis?

A. Ignore the CT finding because only intraparenchymal blood matters when considering intravenous thrombolytic treatment

B. Give thrombolysis immediately because chronic subdural blood cannot increase the risk of post-treatment hemorrhage

C. Proceed only if the NIHSS score exceeds 15 because high neurologic severity overrides all intracranial bleeding concerns

D. Determine whether any intracranial blood is active or clinically relevant because acute intracranial hemorrhage precludes thrombolysis

E. Replace thrombolysis with therapeutic anticoagulation because anticoagulation is safer in the presence of subdural blood

25. A 55-year-old man develops sudden aphasia while already hospitalized after abdominal surgery. The nurse activates a stroke alert within 5 minutes. Which systems practice most directly improves the chance of timely reperfusion?

A. Delay vascular imaging until laboratory testing is completely resulted because hospital-acquired stroke is usually hemorrhagic

B. Wait for the primary surgical team to complete rounds before obtaining neurologic imaging or activating consultants

C. Use a standardized in-hospital stroke pathway with immediate imaging and parallel assessment of reperfusion eligibility

D. Require a formal neurology clinic referral before CT because inpatient symptoms have a higher rate of stroke mimics

E. Transfer the patient to the emergency department before beginning any stroke evaluation or treatment decisions

26. A 52-year-old man has a distal medium-vessel occlusion causing disabling hand weakness and aphasia. The center is considering thrombectomy. Which statement best captures current decision-making?

A. Treatment should be individualized because evidence for distal or medium-vessel thrombectomy is less definitive than for proximal LVO

B. Only intravenous heparin is appropriate because distal arterial thrombi cannot be reached by modern thrombectomy devices

C. The patient should be excluded from all reperfusion because distal occlusions never produce functionally disabling deficits

D. Thrombectomy is universally mandatory for every M3 or A3 occlusion regardless of deficit severity and infarct burden

E. Distal occlusions are never treated endovascularly because catheter access beyond M1 is considered contraindicated

27. A 78-year-old woman with an M1 occlusion has a baseline modified Rankin Scale score of 3 because of severe osteoarthritis but lives at home and manages her own medications. Which statement best describes thrombectomy candidacy?

A. Only patients who are fully asymptomatic before stroke can benefit from endovascular reperfusion of large-vessel occlusion

B. A baseline disability score of 3 requires withholding both thrombolysis and thrombectomy regardless of the acute deficit

C. Thrombectomy should be offered only if the patient can be expected to achieve a modified Rankin Scale score of zero

D. Prestroke disability should prompt individualized assessment rather than automatic exclusion when meaningful recovery to baseline is possible

E. Any prestroke modified Rankin Scale score above 1 is an absolute contraindication to mechanical thrombectomy

28. A patient with an M1 occlusion has an ASPECTS of 4 at 5 hours after onset and was independent before stroke. Which interpretation is most appropriate in the modern EVT era?

A. Low ASPECTS suggests a large core but may still be compatible with thrombectomy benefit in selected patients

B. An ASPECTS below 6 proves that the entire MCA territory is irreversibly infarcted and thrombectomy cannot help

C. Low ASPECTS should trigger immediate anticoagulation rather than endovascular reperfusion because edema is inevitable

D. ASPECTS is used only to diagnose intracranial hemorrhage and has no role in estimating early ischemic injury

E. An ASPECTS of 4 is favorable imaging that predicts a very small infarct core and minimal risk of edema

29. A patient has abrupt cortical blindness and memory difficulty. CTA shows a proximal posterior cerebral artery occlusion without basilar occlusion. Which clinical issue is most important when considering reperfusion?

A. A low NIHSS score requires withholding intravenous thrombolysis even when the visual deficit prevents independent function

B. Posterior cerebral artery strokes are considered nondisabling unless they produce hemiplegia or aphasia on examination

C. The deficit may be highly disabling even with a modest NIHSS score because posterior deficits are underweighted by the scale

D. NIHSS fully captures visual and memory disability, so a low score reliably proves that reperfusion cannot benefit the patient

E. PCA occlusions are always caused by migraine and should not be evaluated with vascular imaging or reperfusion pathways

30. A 68-year-old man with an acute M1 occlusion receives thrombectomy and develops sudden severe headache, vomiting, and neurologic decline 30 minutes later. What is the most appropriate immediate test?

A. Give an additional thrombolytic dose before imaging because reocclusion is the most likely cause of deterioration

B. Start full-dose heparin immediately because worsening after thrombectomy usually represents catheter-related embolization

C. Obtain an urgent noncontrast head CT to evaluate for intracranial hemorrhage and other acute complications

D. Wait six hours for diffusion MRI because CT cannot detect clinically important post-thrombectomy hemorrhage

E. Perform carotid ultrasound first because abrupt postprocedural decline most often reflects extracranial restenosis

31. A 75-year-old woman with a large MCA infarction becomes progressively somnolent 36 hours after onset. CT shows increasing hemispheric edema with midline shift. What management should be considered urgently?

A. Give high-dose corticosteroids because vasogenic edema is the primary mechanism of swelling after large infarction

B. Start full-dose anticoagulation to prevent extension of the infarct while waiting for edema to resolve spontaneously

C. Avoid surgery in all patients older than 60 because hemicraniectomy has no survival benefit in this age group

D. Obtain neurosurgical evaluation for decompressive hemicraniectomy while providing intensive neurocritical care

E. Begin prolonged prophylactic hyperventilation as definitive therapy because it reverses malignant edema without surgery

32. A patient with a large cerebellar infarction develops declining consciousness, obstructive hydrocephalus, and brainstem compression. Which treatment is most appropriate?

A. Urgent suboccipital decompressive surgery, often with CSF diversion when hydrocephalus is present

B. High-dose dexamethasone alone because posterior fossa infarct swelling is predominantly vasogenic rather than cytotoxic

C. Therapeutic anticoagulation because decompressive surgery increases the risk of converting ischemia to hemorrhage

D. Observation with serial examinations because cerebellar swelling rarely causes life-threatening posterior fossa compression

E. Lumbar puncture to relieve pressure because it safely treats obstructive hydrocephalus caused by cerebellar infarction

33. A 65-year-old patient with acute ischemic stroke is not receiving thrombolysis or thrombectomy. Blood pressure is 214/116 mm Hg, and there is no aortic dissection, acute coronary syndrome, encephalopathy, or other hypertensive emergency. Which approach is most appropriate?

A. Leave any pressure untreated even if systolic values exceed 240 mm Hg because cerebral autoregulation is always absent

B. Immediately reduce systolic pressure below 120 mm Hg because all acute ischemic strokes benefit from intensive lowering

C. Induce hypotension to a mean arterial pressure below 60 mm Hg to reduce the metabolic demand of ischemic tissue

D. Avoid rapid aggressive reduction and use modest blood-pressure lowering only when clinically indicated in the acute phase

E. Treat with sublingual nifedipine because abrupt pressure reduction improves collateral flow around the infarct core

34. A 70-year-old woman receives alteplase for acute ischemic stroke. When should aspirin generally be started if follow-up imaging shows no hemorrhage?

A. Seven days after thrombolysis because earlier antiplatelet therapy uniformly causes symptomatic intracranial bleeding

B. Only after hospital discharge because antiplatelet therapy is unnecessary during the acute stroke admission

C. After approximately 24 hours, once post-thrombolysis imaging excludes intracranial hemorrhage

D. Immediately during the thrombolytic infusion because aspirin prevents early reocclusion without increasing bleeding

E. Within 15 minutes of alteplase completion regardless of symptoms or follow-up brain imaging findings

35. A 63-year-old man has a minor noncardioembolic stroke and is started on aspirin plus clopidogrel. What is the best rationale for limiting dual therapy to a short course?

A. Aspirin should always be stopped after one week because chronic aspirin use increases ischemic stroke recurrence in all patients

B. Long-term dual therapy is avoided only because it prevents future carotid endarterectomy from being technically feasible

C. The benefit is concentrated early after minor stroke, while prolonged dual therapy increases bleeding without added long-term benefit

D. Dual therapy is useful solely for pain control in the acute phase and has no effect on early recurrent ischemic events

E. Clopidogrel becomes completely inactive after several weeks because platelet P2Y12 receptors permanently downregulate with treatment

36. A 49-year-old woman arrives 50 minutes after a disabling stroke. She is 10 weeks pregnant, and CT shows no hemorrhage. Which principle best guides consideration of reperfusion?

A. Pregnancy requires individualized maternal-fetal risk assessment but is not an automatic exclusion from reperfusion for a disabling stroke

B. Pregnancy is an absolute contraindication to both intravenous thrombolysis and mechanical thrombectomy at every gestational age

C. Only aspirin can be used because any reperfusion therapy predictably causes fetal intracranial hemorrhage and pregnancy loss

D. Reperfusion should be delayed until delivery because maternal neurologic disability does not affect pregnancy-related treatment decisions

E. Mechanical thrombectomy is forbidden because iodinated contrast exposure is more dangerous than untreated large-vessel occlusion

37. A 14-year-old boy presents with sudden aphasia and hemiparesis from an M1 occlusion at a pediatric center with neurointerventional expertise. Which statement best reflects contemporary guidance?

A. Pediatric stroke treatment should wait for 24 hours of observation because most large-vessel occlusions recanalize spontaneously

B. Select pediatric patients with disabling large-vessel occlusion may be considered for thrombectomy after expert multidisciplinary evaluation

C. Mechanical thrombectomy is contraindicated in everyone younger than 18 because pediatric arteries cannot safely accommodate devices

D. Children with arterial ischemic stroke should receive only aspirin because reperfusion evidence in adults cannot inform pediatric care

E. Endovascular treatment is reserved exclusively for cerebral venous thrombosis and should not be used for pediatric arterial occlusion

38. A thrombectomy candidate is undergoing CT angiography. Which imaging finding most directly identifies the endovascular target?

A. Mild bilateral carotid bulb plaque without hemodynamically significant stenosis or intracranial vessel occlusion

B. An old lacunar infarct in the contralateral basal ganglia with no corresponding acute neurologic deficit

C. Diffuse chronic white-matter hyperintensity without a focal vascular occlusion or acute perfusion deficit

D. A small isolated cortical microbleed on susceptibility imaging without evidence of acute arterial obstruction

E. A proximal intracranial arterial occlusion in a vessel that is technically accessible for thrombectomy

39. A patient with an M1 occlusion has excellent leptomeningeal collateral filling on CTA. What is the main physiologic significance of this finding?

A. Collateral vessels increase intracranial pressure and therefore accelerate infarct growth in every large-vessel occlusion

B. Good collateral filling indicates the lesion is venous rather than arterial and should be treated with anticoagulation

C. Collateral flow can sustain threatened tissue temporarily and slow infarct-core expansion before reperfusion

D. Collateral status has no relationship to tissue survival because only the exact clock time determines infarct progression

E. Good collaterals prove the occluded artery has already recanalized and make thrombectomy unnecessary

40. A patient with a carotid terminus occlusion has a small core and a large perfusion deficit. What does the mismatch primarily represent?

A. A region of completed infarction that has already lost all metabolic activity and cannot respond to reperfusion

B. Blood-brain barrier disruption that specifically predicts symptomatic intracranial hemorrhage after every thrombectomy procedure

C. A zone of chronic small-vessel ischemic change that develops over years and is unrelated to the acute occlusion

D. Hypoperfused tissue that is not yet irreversibly infarcted and may be salvageable with timely reperfusion

E. Normal oligemic tissue with increased flow reserve that does not depend on collateral circulation for survival

41. A patient with a severe anterior-circulation stroke has an ASPECTS of 9. What does this score most strongly suggest?

A. The score proves that the patient has no cerebral ischemia because values above 8 are considered completely normal

B. Only limited early ischemic change is visible in the scored MCA regions on noncontrast CT

C. Extensive early ischemic change involves nearly the entire MCA territory and indicates a very large core

D. The scan demonstrates diffuse subarachnoid hemorrhage because ASPECTS is primarily a hemorrhage grading scale

E. The patient has complete basilar artery occlusion because high ASPECTS specifically localizes posterior circulation stroke

42. A 69-year-old man with acute stroke has fluctuating right weakness that worsens whenever systolic blood pressure falls after nitrates are given for chest discomfort. CTA shows critical left ICA stenosis with poor collaterals. What mechanism best explains the fluctuation?

A. Pressure-dependent cerebral perfusion distal to severe stenosis is producing hemodynamic ischemia

B. Cerebral venous hypertension is reducing cortical drainage because arterial stenosis cannot produce perfusion-dependent deficits

C. Migraine aura is most likely because ischemic deficits do not vary with changes in systemic hemodynamic pressure

D. Repeated embolization from atrial fibrillation is occurring because deficits in cardioembolic stroke always fluctuate with blood pressure

E. The symptoms represent postictal weakness because Todd paresis typically worsens when systemic pressure decreases

43. A 72-year-old woman has recurrent transient right-hand weakness when standing, with brief involuntary jerking of the hand but preserved awareness. Severe left carotid occlusive disease is present. Which diagnosis best explains the episodes?

A. Hemiballismus from a chronic subthalamic infarct because posture-induced episodes are typical of basal ganglia disease

B. Psychogenic nonepileptic events because cerebral ischemia cannot produce positive motor phenomena such as jerking

C. Cervical radiculopathy because transient hand shaking after standing is usually caused by positional nerve-root compression

D. Limb-shaking transient ischemic attacks from hemodynamic hypoperfusion distal to severe carotid disease

E. Focal motor seizures because any rhythmic limb movement with preserved awareness is necessarily epileptic in origin

44. A patient treated with thrombolysis develops acute tongue swelling and stridor. The swelling is predominantly contralateral to an insular infarct, and the patient takes an ACE inhibitor. What is the most likely complication?

A. Anaphylaxis to iodinated contrast because asymmetric tongue swelling is specific for contrast allergy after CT angiography

B. Bulbar weakness from pontine infarction because true angioedema never occurs after fibrinolytic therapy for stroke

C. Acute epiglottitis because thrombolysis does not influence bradykinin metabolism or upper-airway swelling

D. Glossopharyngeal neuralgia because unilateral tongue symptoms after stroke are usually a painful cranial neuropathy

E. Orolingual angioedema associated with thrombolysis, with ACE-inhibitor use increasing the risk

45. A patient receiving alteplase suddenly develops severe headache, vomiting, and worsening hemiparesis. What is the best immediate response?

A. Wait until the scheduled 24-hour CT because imaging sooner does not alter management after intravenous thrombolysis

B. Continue the infusion because early neurologic worsening usually represents reperfusion and predicts a favorable outcome

C. Give aspirin immediately because platelet activation is the most common cause of deterioration during thrombolytic treatment

D. Stop the thrombolytic infusion if still running and obtain emergent brain imaging while preparing hemorrhage management

E. Begin therapeutic heparin before imaging because early recurrent embolism is more common than treatment-related hemorrhage

46. A stroke patient has persistent fever of 39.2°C during the first day after infarction. Which management is most appropriate?

A. Use prophylactic broad-spectrum antibiotics for every stroke patient regardless of infection signs or aspiration risk

B. Identify and treat the cause of fever and use antipyretic measures rather than allowing sustained hyperthermia

C. Start corticosteroids because post-stroke fever is usually caused by autoimmune cerebral edema rather than infection

D. Induce mild hyperthermia because higher temperature increases oxygen delivery to the ischemic penumbra after stroke

E. Ignore fever unless it exceeds 41°C because moderate hyperthermia has no relationship to neurologic outcome

47. A patient with hemispheric ischemic stroke cannot safely swallow water during bedside screening. What is the best next step to reduce aspiration risk?

A. Give thin liquids only because they are easier to swallow than thickened liquids in patients with acute dysphagia

B. Insert a permanent feeding gastrostomy immediately because post-stroke dysphagia almost never improves in the first weeks

C. Avoid speech-language evaluation because dysphagia screening has no effect on pneumonia or nutritional complications

D. Allow a regular diet because early oral feeding improves neurologic recovery even when bedside screening is failed

E. Keep the patient NPO until formal swallowing assessment determines a safe route for nutrition and medications

48. A patient with large ischemic stroke is immobile and cannot receive pharmacologic VTE prophylaxis on the first day because of bleeding concerns. Which measure is appropriate?

A. Use graduated compression stockings alone because they are proven superior to pneumatic compression after acute stroke

B. Use intermittent pneumatic compression to reduce venous thromboembolism risk while reassessing pharmacologic prophylaxis

C. Begin full-dose therapeutic anticoagulation solely for immobility because prophylactic dosing is ineffective in stroke patients

D. Avoid all mechanical prophylaxis because limb compression increases cerebral embolization after ischemic stroke

E. Place an inferior vena cava filter routinely because immobile stroke patients have an inevitable risk of pulmonary embolism

49. A patient develops a small asymptomatic petechial hemorrhagic transformation within an ischemic infarct on follow-up MRI. Which statement is most accurate?

A. Asymptomatic petechial transformation has no relevance to timing of antithrombotic therapy in any clinical circumstance

B. Management depends on hemorrhage type, symptoms, infarct mechanism, and antithrombotic indication rather than the imaging label alone

C. Every petechial transformation requires permanent cessation of all antiplatelet and anticoagulant therapy regardless of indication

D. Petechial hemorrhage is equivalent to a large parenchymal hematoma and always requires emergent surgical evacuation

E. Any hemorrhagic transformation proves that the original event was primary intracerebral hemorrhage rather than ischemic stroke

50. A 66-year-old right-handed man has sudden inability to speak, right lower facial weakness, and weakness of the right arm greater than the leg. Sensation is reduced on the right, and gaze is deviated leftward. Which vascular territory is most likely involved?

A. The right posterior inferior cerebellar artery territory involving the lateral medulla and inferior cerebellum

B. The left posterior cerebral artery territory involving the dominant occipital cortex and splenium

C. The right posterior cerebral artery territory involving the occipital lobe and medial temporal structures

D. The left anterior cerebral artery territory involving the medial frontal and paracentral cortical regions

E. The left middle cerebral artery territory, particularly the superior division and adjacent deep branches

51. A 72-year-old woman develops left leg weakness much greater than arm weakness, urinary urgency, and marked abulia without aphasia. Which artery is most likely affected?

A. The left posterior cerebral artery supplying the occipital lobe, thalamus, and inferomedial temporal lobe

B. The right posterior inferior cerebellar artery supplying the lateral medulla and inferior cerebellar hemisphere

C. The basilar artery supplying the ventral pons and multiple long descending motor pathways bilaterally

D. The right middle cerebral artery supplying the lateral frontal, parietal, and temporal cerebral cortex

E. The right anterior cerebral artery supplying the medial frontal lobe and paracentral lobule

52. A 58-year-old man has sudden vertigo, vomiting, ipsilateral facial numbness, contralateral body pain-temperature loss, dysphagia, hoarseness, and ipsilateral Horner syndrome. Which lesion best explains the syndrome?

A. A medial pontine infarction affecting corticospinal fibers and the abducens fascicle near the midline

B. A dominant angular gyrus infarction causing alexia, acalculia, and hemisensory neglect with cortical aphasia

C. A pure thalamic infarction involving the ventral posterior nucleus without lower cranial nerve dysfunction

D. A lateral medullary infarction, usually from vertebral or posterior inferior cerebellar artery disease

E. A medial frontal infarction in the anterior cerebral artery territory producing leg-predominant motor weakness

53. A 70-year-old woman develops right hemiparesis and loss of vibration and proprioception on the right, with tongue deviation to the left. Which vascular syndrome is most likely?

A. Left medial medullary infarction involving corticospinal fibers, medial lemniscus, and hypoglossal pathways

B. Right thalamic infarction involving the ventral posterior nucleus and internal medullary lamina

C. Left lateral medullary infarction involving nucleus ambiguus, spinothalamic fibers, and sympathetic pathways

D. Right lateral pontine infarction involving the facial nucleus, middle cerebellar peduncle, and spinothalamic tract

E. Left occipital infarction involving the primary visual cortex and splenium without long-tract motor findings

54. A patient is awake and can move the eyes vertically but has quadriplegia, anarthria, and bilateral facial weakness after a basilar artery event. What syndrome is most likely?

A. Cerebellar mutism from bilateral superior cerebellar peduncle injury with preserved limb strength and facial movement

B. Wernicke aphasia from dominant posterior temporal injury causing fluent but meaningless speech and poor comprehension

C. Akinetic mutism from bilateral anterior cingulate injury causing loss of initiation and reduced spontaneous behavior

D. Locked-in syndrome from bilateral ventral pontine injury with preserved consciousness

E. Vegetative state from diffuse cortical injury with loss of awareness despite preserved sleep-wake cycles

55. A 63-year-old man suddenly cannot form new memories and repeatedly asks the same questions. He also has a right superior quadrantanopia. MRI shows an infarct in the left inferomedial temporal lobe. Which artery is most likely involved?

A. The right middle cerebral artery supplying the lateral parietal cortex and superior temporal region

B. The left posterior inferior cerebellar artery supplying the lateral medulla and inferior cerebellar hemisphere

C. The right anterior choroidal artery supplying the posterior limb of the internal capsule and optic tract

D. The left anterior cerebral artery supplying the medial frontal cortex and anterior corpus callosum

E. The left posterior cerebral artery supplying the medial temporal and occipital regions

56. A 69-year-old woman develops pure left-sided numbness involving face, arm, and leg without weakness, aphasia, neglect, or visual field loss. MRI shows a small acute infarct. Which location is most likely?

A. The left inferior frontal gyrus in the dominant middle cerebral artery territory

B. The right precentral gyrus involving the primary motor cortex of the face and upper limb

C. The right ventral posterior thalamus supplied by a small penetrating artery

D. The left lateral medulla involving spinothalamic and trigeminal sensory pathways on opposite sides

E. The right occipital lobe involving the primary visual cortex without thalamic sensory relay nuclei

57. A 62-year-old man has dysarthria and clumsy right-hand movements without sensory loss, aphasia, or visual symptoms. Which lacunar location is classically associated with this syndrome?

A. The left hippocampus supplied by posterior cerebral artery branches to the medial temporal lobe

B. The right occipital cortex supplied by the posterior cerebral artery and calcarine branches

C. The left angular gyrus supplied by the inferior division of the middle cerebral artery

D. The right lateral medulla supplied by the posterior inferior cerebellar artery and vertebral branches

E. The left basis pontis or adjacent internal capsule supplied by penetrating arteries

58. A 73-year-old patient has sudden left hemiparesis with no sensory loss, aphasia, neglect, visual field deficit, or cranial nerve findings. MRI shows a 9-mm acute infarct in the posterior limb of the right internal capsule. What mechanism is most likely?

A. Venous thrombosis causing hemorrhagic cortical infarction and vasogenic edema in a parasagittal distribution

B. Reversible cerebral vasoconstriction causing multifocal segmental narrowing of large and medium cerebral arteries

C. Artery-to-artery embolism from an ipsilateral carotid plaque lodging in a superficial cortical MCA branch

D. Occlusion of a single deep penetrating artery from small-vessel lipohyalinosis or microatheroma

E. Cardioembolism from atrial fibrillation selectively occluding a distal posterior cerebral artery cortical branch

59. MRI after a transient episode of aphasia shows several small acute infarcts scattered in both cerebral hemispheres and the cerebellum. Which mechanism should be strongly suspected?

A. An embolic source proximal to multiple vascular territories, such as the heart or aortic arch

B. Isolated severe stenosis of one distal M2 branch producing repeated infarcts confined to one cortical territory

C. Hypertensive small-vessel lipohyalinosis because it characteristically produces simultaneous infarcts in many arterial territories

D. A focal cerebral venous sinus thrombosis because venous infarcts usually respect several unrelated arterial territories

E. A single lenticulostriate penetrating-artery occlusion causing one lacunar infarct in the basal ganglia

60. A 78-year-old woman has an embolic-appearing ischemic stroke. Initial ECG shows sinus rhythm, echocardiography is unrevealing, and no significant cervicocephalic stenosis is found. What is the most appropriate next diagnostic strategy?

A. Stop the etiologic evaluation because a normal admission ECG excludes clinically meaningful paroxysmal atrial fibrillation

B. Start lifelong anticoagulation empirically because every cryptogenic cortical stroke is presumed to be cardioembolic

C. Use prolonged rhythm monitoring to look for paroxysmal atrial fibrillation that may not appear on a single ECG

D. Perform routine catheter angiography solely to search for a cerebral aneurysm as the cause of an embolic infarct

E. Diagnose small-vessel disease because the absence of a source on initial testing makes a lacunar mechanism most likely

61. A 70-year-old man with nonvalvular atrial fibrillation has a recent ischemic stroke and no mechanical heart valve or moderate-to-severe rheumatic mitral stenosis. Which long-term antithrombotic strategy is generally preferred?

A. Use no antithrombotic therapy once sinus rhythm is restored because prior atrial fibrillation no longer confers embolic risk

B. Use aspirin alone because antiplatelet therapy provides stroke prevention equivalent to anticoagulation in atrial fibrillation

C. Use aspirin plus clopidogrel indefinitely because dual antiplatelet therapy has lower embolic risk than any anticoagulant

D. Use warfarin in every patient with atrial fibrillation because direct oral anticoagulants are contraindicated after ischemic stroke

E. Use a direct oral anticoagulant rather than warfarin when no contraindication to a DOAC is present

62. A 66-year-old woman with a mechanical mitral valve has an ischemic stroke while her INR is subtherapeutic. Which long-term anticoagulant is appropriate for prevention of recurrent valve-related embolism?

A. Aspirin alone is adequate because mechanical valve emboli are platelet-rich and do not respond to anticoagulation

B. Clopidogrel plus aspirin should replace anticoagulation because dual antiplatelet therapy lowers prosthetic-valve thrombosis risk

C. Apixaban is preferred because direct factor Xa inhibition is safer and more effective for all mechanical valves

D. Dabigatran is preferred because direct thrombin inhibition prevents valve thrombosis better than vitamin K antagonism

E. Warfarin with valve-specific INR management remains the indicated oral anticoagulant

63. A 62-year-old man has an ischemic stroke 5 days after a large anterior myocardial infarction. Echocardiography shows a mobile left ventricular apical thrombus. Which mechanism best explains the stroke?

A. Artery-to-artery embolism from carotid plaque because ventricular thrombi cannot embolize to the cerebral circulation

B. Cerebral venous thrombosis caused by low cardiac output and impaired dural sinus drainage after myocardial infarction

C. Reversible cerebral vasoconstriction caused by ventricular remodeling and increased intracranial sympathetic tone

D. Cardioembolism from thrombus formed over akinetic infarcted ventricular myocardium

E. Small-vessel lipohyalinosis caused by the transient catecholamine surge associated with acute myocardial infarction

64. A 54-year-old woman has a cryptogenic nonlacunar stroke. Transesophageal echocardiography shows a patent foramen ovale with a large right-to-left shunt and atrial septal aneurysm. No atrial fibrillation, arterial stenosis, or other cause is found. Which prevention option is reasonable?

A. Use lifelong warfarin in every patient with a PFO because closure has not reduced recurrent stroke in randomized trials

B. Ignore the PFO because right-to-left shunts cannot transmit venous thrombi into the arterial cerebral circulation

C. Perform carotid endarterectomy because cryptogenic stroke with PFO is most often caused by occult ipsilateral carotid plaque

D. Close the PFO only if the index infarct is a classic lacunar lesion caused by a single penetrating artery occlusion

E. Discuss percutaneous PFO closure plus antiplatelet therapy because the patient is younger and the PFO has high-risk features

65. A 74-year-old man with hypertension and diabetes has a 7-mm thalamic lacunar infarct and is incidentally found to have a small PFO. Which interpretation is most appropriate?

A. The PFO proves that the thalamic lesion is venous infarction rather than an arterial lacunar infarct

B. The PFO is likely incidental because the infarct pattern and risk factors strongly support small-vessel disease

C. The PFO is the presumed cause because any intracardiac shunt overrides competing vascular mechanisms after ischemic stroke

D. Percutaneous closure is mandatory because lacunar infarction is the stroke subtype most strongly linked to paradoxical embolism

E. The patient should receive lifelong anticoagulation solely because a PFO is present even without venous thrombosis or atrial fibrillation

66. A 59-year-old woman has a noncardioembolic ischemic stroke and LDL cholesterol of 146 mg/dL. Which lipid strategy is generally appropriate for secondary prevention?

A. Treat lipids only when LDL exceeds 190 mg/dL because lower values do not influence secondary cerebrovascular risk

B. Stop lipid-lowering therapy once the index stroke has stabilized because long-term treatment does not affect recurrence

C. Avoid statins after ischemic stroke because lowering LDL increases recurrent ischemic events more than it reduces vascular risk

D. Use fibrate monotherapy because triglyceride lowering has greater evidence than LDL reduction for recurrent stroke prevention

E. Use high-intensity statin therapy and pursue substantial LDL reduction, often to below 70 mg/dL in atherosclerotic disease

67. A 67-year-old man with prior ischemic stroke has persistent blood pressure around 152/88 mm Hg despite adherence to one antihypertensive. What prevention principle is most appropriate?

A. Stop antihypertensive therapy because cerebral autoregulation remains permanently dependent on elevated systemic pressure after infarction

B. Intensify long-term blood-pressure control because hypertension is a major modifiable risk factor for recurrent stroke

C. Treat blood pressure only when symptoms recur because asymptomatic hypertension does not influence future cerebrovascular risk

D. Use short-acting agents intermittently rather than sustained therapy because chronic control increases small-vessel ischemic events

E. Allow chronic systolic pressure above 150 mm Hg because permissive hypertension should continue indefinitely after ischemic stroke

68. A 63-year-old woman with prior ischemic stroke continues to smoke one pack of cigarettes daily. Which counseling approach has the strongest prevention rationale?

A. Recommend switching to fewer cigarettes because reducing daily smoking by half eliminates most recurrent stroke risk

B. Avoid cessation medications because nicotine replacement and other therapies are contraindicated after any ischemic stroke

C. Recommend complete smoking cessation and offer structured behavioral and pharmacologic support rather than advice alone

D. Focus only on lipid control because smoking has little independent association with recurrent cerebrovascular events

E. Wait until six months after stroke before discussing smoking because early cessation efforts interfere with neurologic rehabilitation

69. A 71-year-old man has a TIA from 80% symptomatic left internal carotid stenosis. He is neurologically stable and has no major surgical contraindication. Which prevention strategy is generally preferred?

A. Treat only with aspirin because revascularization provides no benefit once stenosis exceeds 70 percent

B. Perform carotid endarterectomy relatively early in addition to intensive medical therapy

C. Use anticoagulation alone because severe extracranial carotid stenosis is usually caused by fibrin-rich intracardiac emboli

D. Delay any carotid procedure for at least six months because early intervention has a higher recurrent stroke risk than observation

E. Use intracranial angioplasty because extracranial carotid endarterectomy does not reduce ipsilateral stroke in symptomatic disease

70. A 76-year-old man has 75% symptomatic carotid stenosis and heavy calcification with a tortuous aortic arch. He is otherwise a good surgical candidate. Which feature tends to favor endarterectomy over transfemoral carotid stenting?

A. Age over 70 eliminates the benefit of carotid revascularization regardless of stenosis severity or symptom status

B. Aortic-arch tortuosity lowers the chance of catheter embolization because devices remain farther from the cerebral circulation

C. Advanced age and difficult aortic-arch anatomy increase embolic and access risks with transfemoral stenting

D. A surgically accessible carotid bifurcation makes endarterectomy impossible because open surgery cannot treat cervical plaque

E. Severe calcification reduces perioperative risk from transfemoral stenting and therefore strongly favors catheter treatment

71. A 70-year-old woman with prior neck radiation has symptomatic 80% carotid stenosis at a surgically difficult high cervical location. Which consideration may favor a stenting approach?

A. Endarterectomy is mandatory for every symptomatic stenosis above 70 percent regardless of neck anatomy or surgical risk

B. Radiation-associated stenosis is never atherosclerotic and therefore does not benefit from any form of carotid revascularization

C. High cervical lesions are easier to expose surgically than low bifurcation lesions and therefore strongly favor open surgery

D. Carotid stenting is contraindicated after radiation because irradiated vessels cannot tolerate endovascular devices or antiplatelets

E. Hostile surgical anatomy and prior neck radiation can increase endarterectomy difficulty and may favor carotid stenting

72. A 65-year-old man has 75% asymptomatic carotid stenosis found during evaluation of a carotid bruit. He is receiving intensive risk-factor therapy and has no neurologic symptoms. What is the most appropriate general principle?

A. Perform urgent endarterectomy within 24 hours because any asymptomatic stenosis above 70 percent carries the same risk as a recent TIA

B. Begin warfarin because asymptomatic carotid plaque causes stroke primarily through fibrin emboli that do not respond to antiplatelets

C. Ignore the stenosis because asymptomatic carotid disease never causes future ipsilateral ischemic stroke under any circumstance

D. Schedule intracranial stenting because treatment of asymptomatic extracranial disease requires a device beyond the carotid siphon

E. Optimize medical therapy and consider revascularization only after individualized assessment of procedural risk, life expectancy, and plaque risk

73. A 60-year-old man has a TIA attributable to 80% intracranial M1 atherosclerotic stenosis. Which initial prevention strategy is preferred?

A. Use aggressive medical therapy with risk-factor control and short-term dual antiplatelet therapy rather than first-line stenting

B. Refer for extracranial carotid endarterectomy because M1 stenosis is best treated by removing cervical carotid plaque

C. Perform intracranial angioplasty and stenting immediately because device therapy has lower early stroke risk than medical treatment

D. Use lifelong warfarin because intracranial atherosclerotic stenosis responds better to anticoagulation than antiplatelet therapy

E. Provide aspirin alone without risk-factor modification because blood pressure and lipids do not influence intracranial plaque recurrence

74. A patient with symptomatic severe intracranial atherosclerotic stenosis asks why blood pressure and lipid goals are emphasized so strongly. What is the best explanation?

A. Lowering LDL and blood pressure works mainly by dissolving the fixed stenosis within hours rather than modifying long-term vascular risk

B. Intensive medical therapy is used only because antiplatelet drugs have no role in symptomatic intracranial atherosclerotic disease

C. Risk-factor control matters only for coronary disease because intracranial plaque progression is unrelated to hypertension or dyslipidemia

D. The recurrence risk reflects an active systemic atherosclerotic process that is reduced by aggressive control of modifiable vascular factors

E. The primary mechanism is congenital arterial narrowing, so medical therapy cannot influence recurrent stroke risk after the first event

75. A 68-year-old woman has an embolic-appearing stroke without atrial fibrillation, PFO, significant arterial stenosis, or another identified source after a complete evaluation. Which antithrombotic strategy is recommended for an embolic stroke of undetermined source?

A. Use lifelong aspirin plus clopidogrel because dual antiplatelet therapy is the standard long-term treatment for all cryptogenic strokes

B. Give warfarin plus aspirin because combining anticoagulation and antiplatelet therapy prevents both cardiac and arterial emboli

C. Withhold antithrombotic therapy until a source is found because treatment can obscure future diagnostic testing

D. Start a direct oral anticoagulant empirically because all embolic-appearing strokes are presumed occult atrial fibrillation

E. Use antiplatelet therapy rather than empiric anticoagulation in the absence of a defined anticoagulation indication

76. A 52-year-old man presents with painful partial Horner syndrome followed by a small left hemispheric ischemic stroke. CTA shows a tapered irregular narrowing of the left cervical internal carotid artery. What is the most likely diagnosis?

A. Atherosclerotic carotid stenosis because painful Horner syndrome is typical of calcified plaque at the carotid bifurcation

B. Cervical internal carotid artery dissection with mural injury and thromboembolic ischemia

C. Cerebral venous sinus thrombosis because cervical arterial tapering is caused by elevated venous pressure in the cavernous sinus

D. Reversible cerebral vasoconstriction because RCVS primarily produces a single tapered extracranial carotid stenosis and Horner syndrome

E. Giant cell arteritis because it commonly causes isolated cervical carotid mural hematoma with a characteristic tapered lumen

77. A 44-year-old woman has a vertebral artery dissection after minor neck trauma and a small cerebellar infarct. Which secondary prevention principle is most appropriate?

A. Perform prophylactic surgical ligation of the dissected vertebral artery because endovascular and medical treatments are ineffective

B. Use individualized antithrombotic therapy, commonly an antiplatelet or anticoagulant for a limited period, based on bleeding and ischemic risk

C. Perform routine emergent vertebral artery stenting in all dissections because medical therapy does not prevent recurrent embolization

D. Use no antithrombotic therapy because cervical dissections heal spontaneously and treatment increases recurrence risk

E. Continue dual antiplatelet therapy indefinitely because recurrent dissection risk remains uniformly high for the rest of life

78. A 48-year-old man with cervical artery dissection asks whether ischemic stroke risk is confined to the first few hours after neck pain begins. Which answer is most accurate?

A. Stroke can occur days after dissection symptoms begin, with risk highest early and many events occurring within the first week

B. Ischemic complications are exceptionally rare in cervical dissection because the lesion causes pain but not luminal thrombus

C. Stroke risk is delayed until a pseudoaneurysm exceeds two centimeters and compresses the adjacent sympathetic chain

D. Stroke occurs only at the exact moment the arterial wall tears, so later neurologic symptoms must have another cause

E. The risk begins only after one month because mural hematoma must organize before embolization can occur

79. A 37-year-old woman with spontaneous carotid dissection has marked joint hypermobility, translucent skin, and a family history of arterial rupture. What additional consideration is appropriate?

A. Avoid any genetic or systemic evaluation because all spontaneous dissections have the same idiopathic mechanism

B. Diagnose giant cell arteritis because joint hypermobility and fragile skin are characteristic inflammatory arteritis findings

C. Evaluate for an underlying connective-tissue or arteriopathy syndrome when clinical features strongly suggest one

D. Treat with chronic corticosteroids because connective-tissue fragility in dissection is caused by autoimmune vascular inflammation

E. Assume the dissection is purely traumatic because inherited connective-tissue disorders never affect cervical arteries

80. A 61-year-old man has recurrent embolic strokes despite no atrial fibrillation. Transesophageal echocardiography shows a 6-mm mobile aortic arch plaque with superimposed thrombus. Which mechanism is most plausible?

A. Artery-to-artery embolization from complex aortic arch atheroma into the cerebral circulation

B. Small-vessel lipohyalinosis because aortic plaques cannot embolize into branch vessels arising from the arch

C. Cerebral venous thrombosis because aortic atheroma raises intracranial venous pressure and produces hemorrhagic infarction

D. Carotid dissection because mobile aortic plaque is a secondary response to cervical arterial mural hematoma

E. Moyamoya disease because arch atheroma causes progressive bilateral terminal internal carotid stenosis and basal collaterals

81. A 65-year-old patient with embolic stroke has a mobile left atrial appendage thrombus on transesophageal echocardiography but no atrial fibrillation captured on telemetry. Which conclusion is most appropriate?

A. Antiplatelet therapy alone is sufficient because intracardiac thrombi are composed mainly of platelets rather than fibrin

B. The intracardiac thrombus itself establishes a high-risk cardioembolic source even without documented atrial fibrillation

C. The thrombus is incidental because cardioembolism requires electrocardiographic proof of atrial fibrillation during the stroke admission

D. The stroke should be classified as small-vessel disease because atrial thrombi cannot embolize unless the ventricular ejection fraction is reduced

E. Carotid endarterectomy is indicated because left atrial appendage thrombus usually forms in response to ipsilateral carotid stenosis

82. A 58-year-old woman has recurrent strokes and a transthoracic echocardiogram showing a suspected left atrial mass. Which test is most useful to further characterize a possible left atrial appendage thrombus or other cardioembolic source?

A. Transesophageal echocardiography provides better visualization of the left atrium, appendage, valves, and aortic arch

B. Electroencephalography is preferred because cardioembolic sources are detected by intermittent cerebral electrical slowing

C. Lumbar puncture provides the most direct evidence of a left atrial thrombus through measurement of CSF D-dimer

D. Carotid duplex ultrasound is the best test because it directly images the left atrial appendage through the neck vessels

E. Routine noncontrast head CT can determine whether a left atrial mass is thrombus, tumor, or normal anatomic variant

83. A 69-year-old man has recurrent transient monocular blindness in the right eye and 85% right internal carotid stenosis. What mechanism most likely causes the visual episodes?

A. Bilateral optic tract ischemia from basilar stenosis because each optic tract carries visual information from one eye only

B. Papilledema from raised intracranial pressure because carotid disease commonly produces transient visual obscurations through venous congestion

C. Retinal ischemia from emboli or hypoperfusion in the ophthalmic circulation supplied by the ipsilateral carotid artery

D. Migraine aura because carotid stenosis cannot produce transient retinal ischemia without a permanent visual deficit

E. Occipital cortex ischemia from the contralateral posterior cerebral artery because monocular visual loss localizes posterior to the chiasm

84. A 64-year-old woman has a brief episode of aphasia and right arm weakness that completely resolves. Diffusion MRI shows a small acute cortical infarct. Which terminology is most accurate?

A. This is a transient ischemic attack because the neurologic deficit resolved in less than 24 hours

B. This is a stroke mimic because transient symptoms cannot produce diffusion-restricted brain injury

C. This is a silent infarct because silent lesions are defined by symptoms that resolve before imaging is performed

D. This is an ischemic stroke because tissue infarction is present despite transient clinical symptoms

E. This is a reversible ischemic neurologic deficit, a separate modern category between TIA and completed stroke

85. A 55-year-old man has a high-risk TIA with transient aphasia and weakness. CT is normal. Which diagnostic approach is most appropriate in the emergency setting?

A. Perform urgent brain and vascular imaging and initiate rapid etiologic evaluation because early recurrent stroke risk is substantial

B. Begin empiric lifelong anticoagulation without vascular or cardiac testing because all high-risk TIAs are cardioembolic

C. Discharge without imaging because complete symptom resolution makes a vascular cause unlikely and recurrence risk negligible

D. Delay evaluation for one month because vascular imaging during the first days often overestimates carotid and intracranial stenosis

E. Order only an EEG because transient focal deficits are more likely epileptic than ischemic in patients with a normal CT

86. A patient with a high-risk TIA has no hemorrhage, no atrial fibrillation, and no indication for anticoagulation. Which early antithrombotic strategy is often appropriate when standard criteria are met?

A. Withhold antiplatelets until an MRI proves tissue infarction because antithrombotic therapy is unnecessary for true TIA

B. Use triple antiplatelet therapy because combining aspirin, clopidogrel, and dipyridamole provides the best early safety

C. Use aspirin plus clopidogrel indefinitely because the recurrent risk after a TIA remains equally high for life

D. Start therapeutic heparin and then lifelong warfarin because transient deficits imply an unstable intracardiac thrombus

E. Use a short course of dual antiplatelet therapy followed by single antiplatelet therapy

87. A 68-year-old woman has a deep hypertensive lacunar infarct and no significant large-artery stenosis or cardioembolic source. Which long-term antithrombotic therapy is appropriate?

A. Use a direct oral anticoagulant because small penetrating artery disease is primarily caused by fibrin-rich embolism

B. Use no antithrombotic therapy because lacunar infarcts are caused by vessel-wall disease rather than luminal thrombosis

C. Use single antiplatelet therapy for secondary prevention together with aggressive vascular risk-factor control

D. Use warfarin plus aspirin because combined therapy is required whenever hypertension contributes to the stroke mechanism

E. Use lifelong aspirin plus clopidogrel because lacunar stroke responds best to chronic dual platelet inhibition

88. A 72-year-old patient has an ischemic stroke while taking aspirin consistently. No atrial fibrillation or other anticoagulation indication is found. Which principle is most appropriate before simply escalating antithrombotic therapy?

A. Add warfarin immediately because any stroke occurring on aspirin proves an occult cardioembolic mechanism

B. Use indefinite aspirin plus clopidogrel because recurrent stroke on one antiplatelet automatically requires chronic dual therapy

C. Reassess stroke mechanism, medication adherence, interactions, and vascular risk factors before changing treatment empirically

D. Stop all antithrombotic therapy because recurrent stroke demonstrates pharmacologic resistance that cannot be overcome

E. Increase aspirin to the highest possible dose because platelet inhibition and stroke prevention improve linearly with dose

89. A stroke clinic audits 200 patients with cryptogenic stroke and finds that prolonged rhythm monitoring is ordered in only 35%, despite a local target of 80%. What is the best next quality-improvement step?

A. Identify workflow barriers and implement a standardized monitoring pathway with prospective measurement of completion rates

B. Focus solely on physician education without changing ordering workflows because systems changes rarely affect test completion

C. Conclude that the target is unrealistic and stop collecting data because practice variation is expected in cryptogenic stroke

D. Order implantable monitors for every stroke patient regardless of mechanism so that the measured percentage reaches 100%

E. Remove rhythm monitoring from the audit because process measures should never be linked to diagnostic quality after stroke

90. A 59-year-old woman with stroke asks whether exercise matters after discharge. She can walk safely with supervision and has no cardiac contraindication. What counseling is best?

A. Recommend unrestricted vigorous exercise immediately because supervision and gradual progression are unnecessary after ischemic stroke

B. Delay all activity until every neurologic deficit resolves because rehabilitation before complete recovery increases recurrent stroke risk

C. Recommend avoiding exercise for one year because increased heart rate after stroke raises the risk of cerebral reperfusion injury

D. Encourage regular, progressive physical activity tailored to ability because exercise and reduced sedentary time support vascular risk reduction

E. Advise only passive range-of-motion exercises because aerobic activity does not influence recurrent vascular risk or functional recovery

91. A 62-year-old man with recurrent vascular events reports that he frequently misses evening medications because of a complicated regimen and shift work. Which intervention is most likely to improve prevention?

A. Add more medications without changing the schedule because a larger regimen compensates for missed doses through redundancy

B. Use shared problem-solving to simplify the regimen and build reminder or support systems around the patient's daily routine

C. Emphasize that recurrent stroke is inevitable unless the patient independently changes behavior without additional support

D. Switch every medication to an injectable formulation because injections are always easier to adhere to than oral treatment

E. Document nonadherence and discharge the patient from follow-up because medication-taking behavior is outside the clinician's role

92. A 67-year-old man has severe symptomatic intracranial stenosis and recurrent symptoms because he continues to smoke and has uncontrolled blood pressure despite prescriptions. Which management priority is most important?

A. Proceed directly to intracranial stenting because failure to control risk factors proves medical therapy has biologically failed

B. Stop antihypertensive treatment because higher pressure reduces the chance of embolization from an intracranial atherosclerotic plaque

C. Intensify adherence-focused risk-factor management rather than moving directly to intracranial stenting as a substitute for medical therapy

D. Use warfarin in place of antiplatelets because persistent smoking converts intracranial atherosclerosis into a cardioembolic disorder

E. Avoid further counseling because lifestyle factors no longer influence recurrence once intracranial stenosis exceeds 70 percent

93. A patient with a noncardioembolic stroke is taking aspirin and clopidogrel long term without a specific indication for extended dual therapy. What is the main concern?

A. Dual therapy is contraindicated only because it produces severe neutropenia in nearly every patient treated beyond 30 days

B. Long-term dual therapy causes rebound hypercoagulability because clopidogrel converts irreversibly into a platelet agonist after several weeks

C. Aspirin and clopidogrel together prevent statins from lowering LDL and therefore accelerate atherosclerotic plaque progression

D. The combination is ineffective because the two drugs block exactly the same platelet receptor and provide no additional inhibition

E. Chronic dual antiplatelet therapy increases major bleeding risk without sufficient added stroke-prevention benefit for most patients

94. A 69-year-old woman with atrial fibrillation and prior ischemic stroke is taking apixaban and aspirin without coronary stent, recent acute coronary syndrome, or another antiplatelet indication. Which approach is generally preferable?

A. Replace both drugs with clopidogrel because P2Y12 inhibition prevents both cardioembolic and atherosclerotic stroke mechanisms

B. Use anticoagulation alone because combining an antiplatelet with anticoagulation usually adds bleeding without stroke-prevention benefit

C. Add a second antiplatelet because triple therapy provides the greatest net benefit in stable atrial fibrillation after stroke

D. Continue both drugs indefinitely because every patient with atrial fibrillation and prior stroke requires dual-pathway inhibition

E. Stop apixaban and continue aspirin because antiplatelet therapy is safer and equally effective for atrial-fibrillation embolism

95. A 73-year-old man with atrial fibrillation has a small ischemic stroke and is temporarily off anticoagulation. Which factor should most influence the timing of anticoagulation resumption?

A. Wait exactly 30 days after every ischemic stroke because earlier anticoagulation uniformly causes symptomatic intracranial bleeding

B. Base timing only on the admission INR because infarct size and hemorrhagic transformation do not affect bleeding risk

C. Avoid anticoagulation permanently after any cerebral infarct because recurrent embolic risk is lower than intracranial bleeding risk

D. Balance early recurrent embolic risk against hemorrhagic transformation risk using infarct size, severity, imaging, and clinical stability

E. Restart full-dose anticoagulation at exactly 24 hours after every ischemic stroke regardless of infarct volume or hemorrhage

96. A 70-year-old man has symptomatic 90% carotid stenosis and a large completed ipsilateral MCA infarct with severe edema and hemorrhagic transformation. Which principle best guides timing of carotid revascularization?

A. Delay revascularization until the large infarct and hemorrhagic risk stabilize because very early surgery may be hazardous

B. Perform immediate endarterectomy within hours because severe stenosis always outweighs any risk from a large unstable infarct

C. Use carotid stenting immediately because intracranial hemorrhagic transformation eliminates the bleeding risks of dual antiplatelet therapy

D. Ignore the carotid lesion because severe symptomatic stenosis no longer contributes to recurrence once the index infarct is large

E. Perform bilateral endarterectomy because unilateral symptomatic disease predicts equal short-term risk from the contralateral carotid artery

97. A patient undergoes carotid endarterectomy and several days later develops severe ipsilateral headache, seizures, and a lobar intracerebral hemorrhage despite a widely patent carotid artery. What complication is most likely?

A. Posterior reversible encephalopathy because carotid surgery causes isolated posterior vasogenic edema without hypertension

B. Recurrent carotid embolism because a patent postoperative artery commonly produces hemorrhage through distal cholesterol emboli

C. Reversible cerebral vasoconstriction because carotid surgery uniformly triggers diffuse segmental intracranial vasospasm

D. Cerebral hyperperfusion syndrome after abrupt restoration of flow to a chronically hypoperfused vascular bed

E. Cerebral venous sinus thrombosis because endarterectomy blocks jugular venous drainage and causes lobar hemorrhagic infarction

98. A 65-year-old woman has high-grade left carotid stenosis and recurrent right arm weakness specifically when standing or after antihypertensive dosing. Perfusion imaging shows poor cerebrovascular reserve. Which mechanism best explains the attacks?

A. Cerebral venous thrombosis because orthostatic symptoms are caused by reduced drainage through the superior sagittal sinus

B. Hemodynamic failure distal to the stenosis rather than repeated plaque embolization

C. Small-vessel lipohyalinosis because lacunar syndromes reliably appear and disappear with small changes in systemic pressure

D. Cardioembolism from occult atrial fibrillation because embolic events are characteristically provoked by changes in posture

E. Focal seizures because perfusion-dependent weakness and low vascular reserve indicate epileptic cortical irritability

99. A 61-year-old man has a cryptogenic cortical stroke and frequent premature atrial contractions with left atrial enlargement but no documented atrial fibrillation. Which statement is most accurate?

A. Premature atrial contractions exclude cardioembolism because true atrial fibrillation suppresses all ectopic atrial beats

B. Atrial cardiopathy may increase embolic suspicion, but anticoagulation is not automatically indicated without a validated treatment target

C. Atrial cardiopathy should be ignored because structural atrial abnormalities have no relationship to thromboembolic risk

D. Empiric warfarin plus aspirin is required because combining therapies is the only way to address uncertain embolic mechanisms

E. Left atrial enlargement alone proves atrial fibrillation and mandates lifelong anticoagulation after any ischemic stroke

100. A patient with a cortical embolic stroke has a cardiac tumor attached to the interatrial septum and moving through the mitral valve during diastole. What is the most likely lesion?

A. Papillary fibroelastoma of the aortic valve

B. Left ventricular thrombus

C. Atrial septal aneurysm

D. Infective vegetation

E. Left atrial myxoma

101. A 46-year-old woman has fever, a new murmur, splinter hemorrhages, and multiple acute infarcts in different arterial territories. Blood cultures are positive for Staphylococcus aureus. What is the most likely stroke mechanism?

A. Septic embolization from infective endocarditis

B. Reversible cerebral vasoconstriction

C. Hypertensive lipohyalinosis

D. Primary cerebral vasculitis

E. Cerebral venous sinus thrombosis

102. A patient with active infective endocarditis presents within the thrombolysis window with a disabling ischemic stroke. Which principle is most appropriate?

A. Use full-dose heparin as the safest acute alternative because anticoagulation prevents mycotic aneurysm rupture during embolic stroke

B. Delay brain imaging because CT cannot distinguish septic embolism from ordinary cardioembolic ischemic stroke

C. Intravenous thrombolysis is preferred because infected emboli dissolve more readily than sterile fibrin thrombi after alteplase

D. Give thrombolysis only after starting antibiotics because one antimicrobial dose immediately normalizes the hemorrhagic risk

E. Routine intravenous thrombolysis is generally avoided because septic emboli carry an increased risk of intracranial hemorrhage

103. A 57-year-old man with ischemic stroke has a prosthetic valve, fever, and persistent bacteremia. Transesophageal echocardiography shows a mobile vegetation. Which additional cerebrovascular complication should be considered if he develops sudden severe headache?

A. Rupture of an infectious intracranial aneurysm causing subarachnoid or intraparenchymal hemorrhage

B. Hypertensive deep hemorrhage because infectious endocarditis selectively weakens lenticulostriate arterioles in the basal ganglia

C. Spontaneous epidural hematoma because septic emboli preferentially lodge in meningeal arteries outside the dura

D. Dural arteriovenous fistula because endocarditis uniformly creates pathologic shunts between meningeal arteries and venous sinuses

E. Cerebral amyloid angiopathy because prosthetic-valve infection rapidly deposits beta-amyloid in cortical arterioles

104. A 61-year-old man with a bicuspid aortic valve has an ischemic stroke and severe calcific aortic stenosis but no atrial fibrillation, vegetation, or intracardiac thrombus. Which conclusion is most appropriate?

A. Calcific aortic stenosis automatically establishes a high-risk cardioembolic source requiring lifelong anticoagulation after stroke

B. Aortic stenosis excludes atherosclerotic stroke because valvular obstruction prevents emboli from reaching the cerebral circulation

C. The valvular disease may coexist with stroke but should not be assumed causal without a plausible embolic lesion or mechanism

D. The patient should receive dual antiplatelet therapy indefinitely because calcific valve debris is uniquely resistant to single therapy

E. Carotid imaging is unnecessary because any structural aortic valve abnormality has greater causal probability than cervical atherosclerosis

105. A 69-year-old woman has an ischemic stroke and echocardiography reveals an ejection fraction of 25% with global hypokinesis but no ventricular thrombus or atrial fibrillation. Which antithrombotic principle is most appropriate?

A. No antithrombotic therapy should be used because reduced ejection fraction increases bleeding risk more than embolic risk

B. Any ejection fraction below 35% mandates lifelong anticoagulation because ventricular stasis always creates occult thrombus

C. Low ejection fraction alone does not automatically justify anticoagulation after stroke without another established indication

D. Warfarin plus aspirin is required because systolic dysfunction is simultaneously a cardioembolic and platelet-mediated mechanism

E. Antiplatelet therapy is contraindicated because heart failure converts all ischemic strokes into fibrin-rich cardiac emboli

106. A 68-year-old man has recurrent strokes despite negative vascular imaging and no documented atrial fibrillation. An implantable loop recorder eventually captures 12 hours of atrial fibrillation. What is the most important management consequence?

A. The newly documented atrial fibrillation provides a specific indication for oral anticoagulation if bleeding risk permits

B. Carotid endarterectomy is indicated because atrial fibrillation is a marker of occult carotid plaque instability rather than an embolic source

C. Anticoagulation should be combined permanently with dual antiplatelet therapy because cryptogenic stroke requires maximal antithrombotic intensity

D. The finding does not change treatment because atrial fibrillation must be present during the exact moment of stroke onset to be causal

E. The patient should remain on aspirin alone because short episodes of atrial fibrillation cannot meaningfully increase embolic risk

107. A patient with embolic stroke has severe atherosclerotic plaque in the aortic arch and moderate ipsilateral carotid stenosis. Which approach best supports causal attribution?

A. Integrate infarct distribution, plaque morphology, side and territory, and competing sources rather than assigning cause by lesion severity alone

B. Always label the event cardioembolic because aortic plaque is anatomically closer to the heart than the carotid artery

C. Ignore infarct pattern because brain imaging cannot help distinguish embolic sources in patients with multiple vascular abnormalities

D. Classify the stroke as small-vessel disease whenever more than one potential source is present because causality cannot be estimated

E. Always choose the numerically greatest percentage stenosis because the largest measured lesion is necessarily the causal source

108. A 56-year-old woman with recurrent embolic stroke has no arterial stenosis or atrial fibrillation. She is newly diagnosed with metastatic pancreatic cancer and has markedly elevated D-dimer. What mechanism should be considered?

A. Hypertensive lipohyalinosis because malignancy preferentially produces deep penetrating-artery occlusion through chronic arteriolar fibrosis

B. Moyamoya disease because pancreatic adenocarcinoma causes bilateral terminal carotid stenosis with basal collateral formation

C. Reversible cerebral vasoconstriction because elevated D-dimer directly triggers multifocal intracranial arterial vasospasm

D. Cerebral amyloid angiopathy because metastatic cancer accelerates cortical beta-amyloid deposition and lobar microhemorrhage

E. Cancer-associated hypercoagulability causing arterial thromboembolism or nonbacterial thrombotic endocarditis

109. A 68-year-old man presents with sudden left hemiparesis. CT shows a 22-mL right putaminal intracerebral hemorrhage. Systolic blood pressure is 198 mm Hg, and he is awake without signs of herniation. Which blood-pressure strategy is most appropriate?

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

B. Induce hypertension above 220 mm Hg to preserve perihematomal perfusion because autoregulation is completely absent

C. Leave systolic pressure near 200 mm Hg because acute lowering consistently worsens hematoma expansion and outcome

D. Reduce systolic pressure immediately below 100 mm Hg because maximal lowering prevents all early hematoma growth

E. Use only oral antihypertensive medication because intravenous agents increase blood-pressure variability after hemorrhage

110. A 74-year-old woman with warfarin-associated intracerebral hemorrhage has an INR of 3.4. Which reversal strategy is most appropriate?

A. Give idarucizumab because it rapidly binds warfarin and reverses vitamin K antagonist anticoagulant activity

B. Use protamine sulfate because it neutralizes the reduced vitamin K-dependent clotting factors caused by warfarin

C. Give aspirin because platelet inhibition counteracts vitamin K antagonist activity in the coagulation cascade

D. Give 4-factor prothrombin complex concentrate plus intravenous vitamin K for rapid and sustained warfarin reversal

E. Use fresh frozen plasma alone and withhold vitamin K because vitamin K prevents later anticoagulation restart

111. A 79-year-old man taking dabigatran presents with a large lobar intracerebral hemorrhage 2 hours after his last dose. Which specific reversal agent is indicated?

A. Idarucizumab, a monoclonal antibody fragment that binds dabigatran

B. Vitamin K, which rapidly restores thrombin inhibition caused by direct oral anticoagulants within minutes

C. Protamine sulfate, which directly binds dabigatran and reverses its renal clearance-independent anticoagulant effect

D. Desmopressin, which neutralizes circulating dabigatran by increasing endothelial release of von Willebrand factor

E. Andexanet alfa, a decoy factor Xa protein used specifically to bind and neutralize dabigatran

112. Medication reconciliation in a patient with acute spontaneous ICH shows rivaroxaban taken earlier the same day. The team wants a drug-specific reversal strategy for the anticoagulant effect. Which option best fits that goal?

A. Idarucizumab is preferred because it binds all direct oral anticoagulants regardless of their coagulation target

B. Vitamin K should be given because it directly restores factor Xa activity suppressed by apixaban within minutes

C. Protamine sulfate is the standard antidote because it neutralizes oral factor Xa inhibitors as effectively as heparin

D. Platelet transfusion reverses apixaban because the drug acts primarily by blocking platelet aggregation rather than coagulation

E. Andexanet alfa can reverse clinically significant apixaban or rivaroxaban activity in life-threatening bleeding

113. A 70-year-old man with spontaneous ICH was taking aspirin but has a normal platelet count and does not require emergency neurosurgery. Should he receive prophylactic platelet transfusion?

A. Yes; platelet transfusion is preferred to blood-pressure treatment because expansion is driven mainly by aspirin-mediated dysfunction

B. Yes; platelet transfusion is routinely required because any aspirin exposure produces irreversible intracranial bleeding until platelets are replaced

C. Yes; transfuse platelets only to raise the platelet count above 500,000/μL because supranormal counts reduce hematoma expansion

D. No; routine platelet transfusion for aspirin-associated ICH without an operative indication may worsen outcome

E. No; aspirin has no effect on platelet function after ingestion and therefore never contributes to hemorrhage risk

114. A 63-year-old man with a 35-mL frontal ICH is somnolent but has no clinical seizure. Continuous EEG shows no epileptiform activity. Which antiseizure strategy is appropriate?

A. Give benzodiazepines around the clock because sedation reduces cerebral metabolism and prevents hematoma expansion

B. Treat prophylactically only with valproate because it has been proven to improve functional outcome after spontaneous ICH

C. Begin phenytoin for at least six months because all cortical ICHs produce delayed epilepsy if prophylaxis is omitted

D. Do not give routine prophylactic antiseizure medication in the absence of clinical or electrographic seizures

E. Start two antiseizure drugs because somnolence after ICH should be assumed to represent nonconvulsive status epilepticus

115. A patient with lobar ICH becomes intermittently unresponsive with subtle right facial twitching. What is the most appropriate diagnostic test?

A. Continuous EEG monitoring to detect electrographic seizures or nonconvulsive status epilepticus

B. Lumbar puncture because subtle facial movements after hemorrhage most strongly suggest bacterial meningitis

C. Electromyography because intermittent facial twitching is best localized to a peripheral facial nerve disorder

D. Routine outpatient EEG several weeks later because acute electrographic seizures do not influence inpatient management

E. Carotid ultrasound because episodic unresponsiveness after ICH is usually caused by recurrent carotid embolization

116. A 71-year-old woman with ICH is hemiplegic and unable to walk. Which venous thromboembolism prophylaxis should be initiated on the day of diagnosis?

A. Graduated compression stockings alone are preferred

B. Full-dose unfractionated heparin should begin immediately

C. An inferior vena cava filter should be placed routinely

D. Intermittent pneumatic compression should begin early in immobile patients

E. No prophylaxis should be used during the entire hospitalization

117. A 66-year-old man with stable spontaneous ICH has no hematoma expansion on repeat CT at 30 hours and remains immobile. Which prophylaxis can now reasonably be considered?

A. Systemic thrombolysis because immobility-associated venous thrombosis is best prevented by maintaining continuous fibrinolysis

B. Therapeutic-dose anticoagulation because any stable repeat CT eliminates the risk of recurrent intracranial bleeding

C. Low-dose unfractionated heparin or low-molecular-weight heparin for DVT prevention after stability is documented

D. Aspirin plus clopidogrel because antiplatelet drugs are more effective than heparin for venous thromboembolism prevention

E. No pharmacologic prophylaxis until six weeks because low-dose heparin always causes hematoma expansion in spontaneous ICH

118. A 58-year-old woman with a 25-mL thalamic ICH has worsening edema but no herniation. A consultant suggests high-dose dexamethasone. What is the best response?

A. Corticosteroids should not be used for routine treatment of perihematomal edema in spontaneous ICH

B. Methylprednisolone should replace osmotic therapy because corticosteroids rapidly shrink the hematoma itself through vasoconstriction

C. Corticosteroids are required for every deep hemorrhage because they prevent intraventricular extension and obstructive hydrocephalus

D. Dexamethasone is first-line therapy because perihematomal edema is primarily caused by steroid-responsive vasogenic inflammation

E. Steroids should be given only when systolic blood pressure exceeds 180 mm Hg because they reduce pressure and edema simultaneously

119. A patient with ICH is receiving scheduled hypertonic saline every four hours despite normal intracranial pressure and no clinical evidence of herniation. Which principle is most appropriate?

A. Prophylactic mannitol should continue until the hematoma completely resolves on imaging several weeks after the hemorrhage

B. Hyperosmolar therapy is contraindicated even during impending herniation because it invariably causes rebound cerebral edema

C. Continuous prophylactic hyperosmolar therapy has no established outcome benefit and should not be used routinely

D. Hypertonic saline prevents rebleeding by increasing serum sodium and therefore should replace antihypertensive treatment

E. Scheduled hyperosmolar therapy is required for all ICH because subclinical intracranial hypertension always precedes neurologic deterioration

120. CT in a deteriorating patient shows a 24-mL posterior fossa hematoma centered in the cerebellum, with effacement of the fourth ventricle and new obstructive hydrocephalus. Which intervention is most appropriate now?

A. Observation alone because cerebellar hemorrhage rarely causes life-threatening brainstem compression or obstructive hydrocephalus

B. High-dose corticosteroids because posterior fossa hemorrhage responds better to medical edema treatment than to surgery

C. Lumbar puncture because removing spinal CSF safely relieves obstructive hydrocephalus from a posterior fossa mass

D. Systemic anticoagulation because cerebellar hemorrhage with hydrocephalus usually represents venous thrombosis rather than primary bleeding

E. Urgent surgical evacuation of the cerebellar hematoma with management of hydrocephalus

121. A 74-year-old woman has a 17-mL cerebellar ICH but remains awake. There is mild fourth-ventricular compression without hydrocephalus. Which modern surgical consideration is most relevant?

A. A cerebellar hematoma volume above about 15 mL is itself a factor supporting immediate surgical evacuation

B. A patient without hydrocephalus cannot benefit from evacuation because surgery works only by reducing ventricular obstruction

C. Surgery is never considered until the patient becomes comatose because volume alone has no relationship to posterior fossa risk

D. The 15-mL threshold applies only to lobar supratentorial hemorrhage and not to posterior fossa hematomas

E. All cerebellar hemorrhages smaller than 30 mL should be treated with prophylactic corticosteroids rather than surgery

122. A 65-year-old man has a 45-mL superficial lobar ICH causing progressive hemiparesis but no herniation. Which statement best summarizes minimally invasive evacuation?

A. The main proven benefit of minimally invasive surgery is elimination of all long-term disability among survivors with lobar hemorrhage

B. Surgical evacuation should never be considered unless the hemorrhage is cerebellar because supratentorial procedures have no evidence base

C. Minimally invasive evacuation is contraindicated because catheter-based removal uniformly increases mortality compared with medical treatment

D. Every supratentorial ICH larger than 20 mL requires immediate open craniotomy because medical therapy cannot achieve survival

E. Minimally invasive evacuation can reduce mortality in selected supratentorial ICH, but functional-outcome benefit is less certain

123. A 77-year-old woman with lobar ICH has numerous strictly lobar cerebral microbleeds and cortical superficial siderosis on MRI. Which underlying arteriopathy is most likely?

A. Moyamoya disease

B. Cerebral amyloid angiopathy

C. Primary CNS vasculitis

D. Cerebral venous thrombosis

E. Hypertensive arteriolosclerosis

124. A 76-year-old man with probable cerebral amyloid angiopathy has brief recurrent spreading numbness of the hand and face lasting several minutes. MRI shows cortical superficial siderosis without acute infarction. What is the most likely explanation?

A. CAA-related transient focal neurologic episodes associated with superficial cortical blood products

B. Classic lacunar TIAs from lipohyalinosis because spreading positive sensory symptoms specifically localize a penetrating artery occlusion

C. Migraine aura because superficial siderosis has no relationship to recurrent transient focal symptoms in older adults

D. Focal seizures can be excluded because CAA never irritates cortex or produces positive motor and sensory phenomena

E. Hemodynamic carotid TIAs because superficial siderosis is a marker of critically reduced extracranial carotid perfusion reserve

125. A 60-year-old man with long-standing hypertension has a spontaneous pontine hemorrhage. MRI also shows deep microbleeds in the basal ganglia and thalami. Which small-vessel pathology is most likely?

A. Cerebral venous thrombosis

B. Cerebral amyloid angiopathy

C. Hypertensive arteriolosclerosis

D. Reversible cerebral vasoconstriction

E. Moyamoya disease

126. A 51-year-old woman has a lobar ICH with no hypertension. CTA shows a compact tangle of vessels with an early draining vein. What is the most likely cause?

A. Developmental venous anomaly because DVAs are high-flow lesions that commonly rupture into large lobar hematomas

B. Dural venous sinus thrombosis because sinus occlusion creates a focal nidus with direct early drainage into a cortical vein

C. A cavernous malformation because cavernomas show early arterial filling and prominent draining veins on routine CTA

D. Cerebral amyloid angiopathy because amyloid vessels form a compact high-flow nidus with early venous drainage

E. An arteriovenous malformation producing a high-flow arteriovenous shunt

127. A 48-year-old man with unexplained lobar ICH has a negative CTA but persistent suspicion for a small vascular malformation. Which test provides the highest spatial and temporal resolution for detecting an occult shunting lesion?

A. Catheter cerebral angiography

B. Carotid duplex ultrasound

C. Noncontrast head CT

D. Electroencephalography

E. Routine skull radiography

128. A 73-year-old woman with severe ICH has an ICH score of 4. Her family asks whether this score means treatment should be withdrawn. What is the best response?

A. Use the score for severity communication and prognosis, but not as the sole basis for limiting life-sustaining treatment

B. Recommend withdrawal because an ICH score of 4 is a validated directive to stop aggressive medical and surgical care

C. Ignore the score completely because severity scales have no prognostic value and should never be discussed with families

D. Use the score only to determine whether the patient can receive antihypertensive therapy, not for prognosis or communication

E. Convert the ICH score directly into an individual certainty of death because group-level prediction applies exactly to each patient

129. A patient with ICH has a preexisting do-not-attempt-resuscitation order. Which statement is most appropriate?

A. A DNAR order can be expanded to other treatment limits without discussion because patients who refuse CPR also refuse intensive care

B. DNAR status means all antihypertensive, neurosurgical, nutritional, and rehabilitation treatments should be withheld automatically

C. The order should be ignored because advance preferences have no role in acute neurologic emergencies with uncertain prognosis

D. DNAR status requires immediate comfort-only care because intracerebral hemorrhage has a high mortality rate regardless of severity

E. A DNAR order addresses cardiopulmonary resuscitation and should not automatically limit other indicated stroke treatments

130. A 62-year-old woman with moderate ICH is medically stable at 18 hours. Which rehabilitation approach is most appropriate?

A. Delay rehabilitation until all weakness resolves because therapy before spontaneous recovery interferes with cortical reorganization

B. Use fluoxetine routinely to improve motor recovery because selective serotonin reuptake inhibitors consistently enhance post-ICH function

C. Begin coordinated rehabilitation assessment early, but avoid very aggressive mobilization during the first 24 hours

D. Keep the patient on strict bed rest for two weeks because any movement during the first days increases hematoma expansion

E. Begin high-intensity walking within six hours because very early aggressive mobilization reduces mortality after spontaneous ICH

131. A hospital reviews ICH care and finds large hour-to-hour blood-pressure variability despite achieving the average target. Why is this a quality concern?

A. Smooth sustained blood-pressure control is favored because variability may contribute to hematoma expansion and worse outcome

B. Only the lowest recorded pressure matters because peak systolic values have no relationship to hematoma growth or clinical outcome

C. Variability should be increased intentionally with intermittent bolus therapy because continuous infusions cause cerebral vasospasm

D. Blood pressure is relevant only before CT diagnosis and has no influence on subsequent hematoma expansion once hemorrhage is confirmed

E. Blood-pressure variability is desirable because alternating hypertension and hypotension improves perfusion to the perihematomal region

132. CTA performed after confirmed aneurysmal subarachnoid hemorrhage identifies the ruptured anterior communicating artery aneurysm. The patient has been stabilized in the neurocritical care unit. Which next step most directly reduces the risk of catastrophic early rebleeding?

A. Delay vascular imaging for one week because early aneurysm treatment increases rebleeding risk before vasospasm begins

B. Urgently identify and secure the ruptured aneurysm, preferably within 24 hours, while providing neurocritical care

C. Start therapeutic anticoagulation because diffuse cisternal blood most often reflects thrombosis of a cerebral venous sinus

D. Treat only the headache because aneurysmal subarachnoid hemorrhage usually stops bleeding permanently after the initial rupture

E. Begin routine hypervolemic therapy before aneurysm treatment because prophylactic volume expansion prevents early rebleeding

133. A patient presents 12 hours after a classic thunderclap headache. Noncontrast CT is read as normal, but clinical suspicion for subarachnoid hemorrhage remains high. Which diagnostic approach is appropriate?

A. Wait for recurrent headache before further testing because additional evaluation after a normal CT increases false-positive diagnoses

B. Exclude subarachnoid hemorrhage completely because a normal CT at any time after headache onset has perfect sensitivity

C. Diagnose migraine immediately because thunderclap headache cannot occur without visible blood on the first CT scan

D. Start anticoagulation empirically because a normal CT makes cerebral venous thrombosis more likely than any arterial disorder

E. Pursue additional evaluation, such as lumbar puncture or vascular imaging according to timing and local diagnostic pathway

134. A 56-year-old man with confirmed aneurysmal SAH has a ruptured anterior communicating artery aneurysm. Which medication should be started early to improve neurologic outcome related to delayed cerebral ischemia?

A. Enteral nimodipine

B. Prophylactic induced hypertension

C. Oral corticosteroids

D. Intravenous magnesium

E. High-dose statin therapy

135. A 61-year-old woman with aneurysmal SAH becomes somnolent and develops enlarged ventricles with transependymal CSF flow. What is the most appropriate treatment?

A. Give high-dose dexamethasone because hydrocephalus after SAH is caused primarily by steroid-responsive ependymal inflammation

B. Place an external ventricular drain to treat acute obstructive hydrocephalus when clinically indicated

C. Start therapeutic heparin because enlarged ventricles indicate thrombosis of the deep cerebral venous system rather than CSF obstruction

D. Observe until vasospasm resolves because acute hydrocephalus usually improves spontaneously and drainage increases rebleeding risk

E. Perform lumbar puncture repeatedly because spinal CSF drainage is always safer than ventricular drainage after aneurysmal SAH

136. A patient with aneurysmal SAH is neurologically stable on day 7, then develops new right arm weakness and aphasia. CT shows no rebleeding or hydrocephalus. What complication is most likely?

A. Delayed cerebral ischemia related to vasospasm or other post-SAH perfusion disturbance

B. Recurrent migraine aura because delayed focal deficits after SAH are rarely caused by cerebral perfusion abnormalities

C. Acute bacterial meningitis because focal aphasia and arm weakness are the most specific signs of post-SAH meningeal infection

D. Cerebral amyloid angiopathy because delayed deficits one week after aneurysm rupture reflect newly deposited cortical amyloid

E. Todd paralysis because every new focal deficit after SAH should be presumed postictal even without clinical or EEG seizure

137. A patient with aneurysmal SAH develops symptomatic delayed cerebral ischemia and has no cardiopulmonary contraindication. Which hemodynamic strategy can be beneficial?

A. Raise blood pressure therapeutically while maintaining euvolemia to improve cerebral perfusion

B. Lower systolic pressure below 100 mm Hg because vasospastic arteries dilate when transmural pressure is reduced substantially

C. Induce prophylactic hypotension because lowering arterial pressure reduces vasospasm severity and improves collateral flow

D. Create marked hypervolemia with several liters of fluid because routine triple-H therapy prevents pulmonary and cerebral complications

E. Use diuretics to maintain a negative fluid balance because relative hypovolemia reduces the risk of cerebral edema and DCI

138. A neurologically intact patient with secured aneurysmal SAH is receiving prophylactic hypervolemia despite normal volume status. What is the best recommendation?

A. Restrict fluids severely because dehydration prevents cerebral edema and therefore lowers delayed cerebral ischemia risk

B. Use daily albumin boluses because colloid-induced hypervolemia reduces vasospasm more effectively than enteral nimodipine

C. Continue hypervolemia because excess intravascular volume is the only established method for preventing delayed cerebral ischemia

D. Stop routine hypervolemic augmentation and maintain euvolemia because prophylactic hypervolemia adds morbidity without proven benefit

E. Add prophylactic vasopressors because all patients should remain hypertensive throughout the entire 21-day vasospasm window

139. A 53-year-old man with high-grade aneurysmal SAH has a poor neurologic examination that limits detection of vasospasm. Which monitoring tools can help identify delayed ischemia risk?

A. Serial lumbar puncture because CSF opening pressure is the most specific measure of delayed cerebral ischemia after aneurysm treatment

B. Daily noncontrast skull radiographs because arterial vasospasm produces visible calcific narrowing during the second week

C. Repeated nerve-conduction studies because worsening cortical perfusion is first detected by changes in peripheral motor amplitudes

D. Routine carotid duplex alone because extracranial flow velocities reliably measure intracranial vasospasm in every cerebral artery

E. Transcranial Doppler, CTA or CT perfusion, and in selected cases continuous EEG or invasive monitoring

140. A patient with aneurysmal SAH has persistently elevated middle cerebral artery velocities on transcranial Doppler. What does this finding most directly suggest?

A. The artery has necessarily recanalized from an embolic occlusion because high velocity proves a completely normal vessel diameter

B. The patient has cerebral venous thrombosis because venous outflow obstruction specifically accelerates arterial TCD velocity

C. The aneurysm has reruptured because TCD velocity directly measures the volume of new subarachnoid blood

D. Increasing flow velocity can indicate angiographic vasospasm in the monitored artery when interpreted with ratios and clinical context

E. Hydrocephalus is worsening because ventricular enlargement is the main determinant of middle cerebral artery flow velocity

141. A 60-year-old woman with aneurysmal SAH has a witnessed generalized seizure on day 1. Which antiseizure approach is most appropriate?

A. Use continuous benzodiazepine sedation instead of an antiseizure drug because sedation prevents delayed cerebral ischemia and rebleeding

B. Do not treat the seizure because early seizures after SAH are benign and never recur or worsen secondary brain injury

C. Use lifelong phenytoin because any seizure after SAH predicts permanent epilepsy and phenytoin improves cognitive outcome

D. Treat the new-onset seizure, commonly for about 7 days, while avoiding routine long-term therapy unless another indication emerges

E. Give prophylactic antiseizure medication to every SAH patient indefinitely because seizure risk remains uniformly high for life

142. A neurologically intact patient with low-grade aneurysmal SAH has no seizure, intraparenchymal hemorrhage, hydrocephalus, or cortical infarction. Should prophylactic antiseizure medication be started routinely?

A. No; routine prophylaxis is not recommended in low-risk patients without seizures

B. No; antiseizure drugs are contraindicated even after a witnessed seizure because they worsen cerebral vasospasm

C. Yes; prophylactic treatment should continue until follow-up angiography proves complete aneurysm obliteration and no vasospasm

D. Yes; every aneurysmal SAH requires at least one year of phenytoin regardless of hemorrhage grade or cortical injury

E. Yes; prophylaxis is mandatory because nimodipine lowers the seizure threshold and should always be paired with an anticonvulsant

143. A 50-year-old woman with aneurysmal SAH develops severe hyponatremia and appears volume depleted. Which management principle is most appropriate?

A. Ignore sodium unless seizures occur because electrolyte abnormalities do not influence neurologic status after aneurysmal hemorrhage

B. Use loop diuretics to create negative fluid balance because reduced circulating volume lowers the risk of delayed cerebral ischemia

C. Correct sodium while preserving intravascular volume, because hypovolemia can worsen cerebral perfusion and delayed ischemia risk

D. Treat only with oral free water because SAH-related hyponatremia is caused by dehydration and requires hypotonic replacement

E. Restrict fluids aggressively because all hyponatremia after SAH represents SIADH and volume contraction protects against vasospasm

144. A patient with aneurysmal SAH develops pulmonary edema, reduced ejection fraction, and diffuse T-wave inversion without obstructive coronary disease. What mechanism is most likely?

A. Bacterial myocarditis because subarachnoid blood commonly seeds the myocardium and produces transient ventricular dysfunction

B. Cardiac amyloidosis because acute SAH accelerates transthyretin deposition and causes reversible systolic failure within hours

C. Pulmonary embolism because right-heart strain specifically produces diffuse T-wave changes and left ventricular apical ballooning

D. Neurogenic stunned myocardium from catecholamine-mediated cardiac injury after severe SAH

E. Chronic hypertensive cardiomyopathy because longstanding pressure overload typically causes abrupt reversible dysfunction after aneurysm rupture

145. A 58-year-old man with a ruptured posterior communicating artery aneurysm has the lesion secured by coiling. Follow-up imaging shows a small residual neck. Which principle is most appropriate?

A. Stop all vascular risk management because aneurysm treatment removes the need to address smoking or hypertension afterward

B. No additional imaging is needed after any coil procedure because treated aneurysms cannot recur or regrow once flow is reduced

C. Repeat imaging only if a new thunderclap headache occurs because asymptomatic remnants have no future rupture or retreatment implications

D. Perform prophylactic whole-brain radiation because residual aneurysm tissue is best prevented from enlarging by vascular irradiation

E. Continue cerebrovascular imaging surveillance because residual, recurrent, or de novo aneurysms can affect future management

146. A 42-year-old woman with two first-degree relatives who had aneurysmal SAH asks about screening. Which statement is most appropriate?

A. Only people with prior ischemic stroke should be screened because family history does not meaningfully influence aneurysm risk

B. Familial clustering with at least two affected first-degree relatives can justify discussion of noninvasive aneurysm screening

C. A normal scan at one time guarantees that de novo aneurysms cannot develop later in a person with strong familial risk

D. Screening is never appropriate in asymptomatic relatives because intracranial aneurysms cannot be detected before they rupture

E. Annual catheter angiography is mandatory for every person with a single distant relative who had any type of intracranial hemorrhage

147. A 31-year-old woman is 10 days postpartum and develops progressive headache, papilledema, and a focal seizure. Noncontrast CT is nondiagnostic. Which test is most appropriate to evaluate for cerebral venous thrombosis?

A. Routine EEG alone because headache with seizure after delivery is most often primary epilepsy rather than vascular disease

B. Lumbar puncture before venous imaging because opening pressure confirms the location and extent of dural sinus thrombosis

C. Noncontrast skull radiography because thrombosed venous sinuses become calcified and visible within the first postpartum weeks

D. CT venography or MR venography to assess the dural sinuses and cerebral veins

E. Carotid duplex ultrasound because postpartum thrombosis predominantly affects the extracranial internal carotid arteries

148. A 28-year-old woman with superior sagittal sinus thrombosis has a small hemorrhagic venous infarct. Which treatment is generally indicated?

A. Begin therapeutic anticoagulation despite the hemorrhagic venous infarction unless a specific contraindication exists

B. Withhold anticoagulation because any intracranial blood makes anticoagulant treatment of venous thrombosis uniformly unsafe

C. Give platelet transfusion and aspirin because venous thrombi are platelet-rich and respond better to antiplatelet therapy than heparin

D. Perform immediate decompressive craniectomy in every hemorrhagic CVT because anticoagulation cannot prevent thrombus propagation

E. Use intravenous thrombolysis as routine first-line therapy because systemic fibrinolysis is safer than heparin in venous infarction

149. A pregnant patient is diagnosed with cerebral venous thrombosis. Which anticoagulant is generally preferred during pregnancy?

A. No antithrombotic therapy because pregnancy-associated CVT usually recanalizes spontaneously after the first trimester

B. Therapeutic low-molecular-weight heparin because it does not cross the placenta

C. Warfarin throughout pregnancy because vitamin K antagonists are safest for the fetus and require no dose adjustment

D. Rivaroxaban because direct factor Xa inhibitors have the strongest pregnancy safety data and routinely replace heparin

E. Aspirin alone because anticoagulation is contraindicated during pregnancy even when cerebral venous thrombosis is confirmed

150. A patient with cerebral venous thrombosis worsens despite adequate anticoagulation and develops impending herniation from a large hemorrhagic venous infarct. Which intervention can be lifesaving?

A. Give high-dose corticosteroids because venous infarct edema is an inflammatory process that responds better than arterial stroke edema

B. Use prophylactic carotid stenting because improving arterial inflow is the standard treatment for herniation caused by venous outflow obstruction

C. Stop all anticoagulation permanently because deterioration proves that venous thrombosis is no longer contributing to the brain injury

D. Decompressive craniectomy for malignant mass effect while continuing mechanism-directed CVT management

E. Perform lumbar puncture to rapidly reduce intracranial pressure because mass lesions do not create pressure-gradient risk in CVT

151. A 35-year-old woman with CVT remains comatose and worsens despite anticoagulation. The multidisciplinary team considers catheter-based therapy. Which statement is most accurate?

A. Catheter therapy is contraindicated in CVT because venous sinuses cannot be safely instrumented under any circumstance

B. Systemic thrombolysis should replace anticoagulation in all severe CVT because randomized evidence shows universal functional benefit

C. Endovascular treatment is useful only for chronic asymptomatic sinus occlusion and not for acute neurologic deterioration

D. Endovascular therapy may be considered as rescue treatment in selected severe deteriorating CVT, but it is not routine first-line therapy

E. Endovascular thrombectomy is mandatory for every CVT because anticoagulation alone cannot reopen dural venous sinuses

152. Three days after using a sympathomimetic decongestant, a 32-year-old woman experiences several abrupt, maximal-intensity headaches separated by symptom-free intervals. Vascular imaging shows alternating areas of intracranial arterial constriction and dilatation. Which disorder best explains this pattern?

A. Primary CNS vasculitis

B. Reversible cerebral vasoconstriction

C. Moyamoya disease

D. Atherosclerotic intracranial disease

E. Cerebral amyloid angiopathy

153. A patient with probable reversible cerebral vasoconstriction syndrome is taking a serotonergic antidepressant and recently used cocaine. Which management principle is most appropriate?

A. Perform intracranial stenting in every narrowed segment because the vasoconstriction is fixed and rarely resolves spontaneously

B. Start high-dose glucocorticoids because immunosuppression shortens RCVS and prevents arterial rupture in most patients

C. Remove vasoactive triggers and provide supportive care while monitoring for ischemic or hemorrhagic complications

D. Continue cocaine in small doses because abrupt withdrawal increases sympathetic tone and worsens cerebral vasoconstriction

E. Begin lifelong anticoagulation because multifocal arterial narrowing is caused by thrombus rather than dysregulated vascular tone

154. A 29-year-old woman one week postpartum has severe hypertension, headache, seizures, and visual loss. MRI shows symmetric parieto-occipital vasogenic edema without restricted diffusion in most lesions. What is the most likely diagnosis?

A. Posterior reversible encephalopathy syndrome

B. Cerebral venous thrombosis

C. Osmotic demyelination syndrome

D. Bilateral posterior cerebral artery infarction

E. Progressive multifocal leukoencephalopathy

155. A patient with hypertensive PRES has blood pressure 220/130 mm Hg and ongoing visual symptoms. What is the best treatment principle?

A. Avoid antihypertensive treatment because PRES represents ischemia and therefore requires permissive hypertension until MRI normalizes

B. Reduce blood pressure carefully with titratable therapy while treating seizures and the underlying trigger

C. Use thrombolysis because posterior vasogenic edema is caused by multifocal small-artery occlusion and should be reperfused

D. Give high-dose anticoagulation because severe hypertension in PRES produces venous sinus thrombosis as the primary mechanism

E. Normalize systolic pressure below 100 mm Hg immediately because rapid maximal lowering is required to reverse vasogenic edema

156. A 39-year-old woman has recurrent ischemic strokes, bilateral terminal internal carotid stenosis, and a network of basal collateral vessels on angiography. Which diagnosis is most likely?

A. Reversible cerebral vasoconstriction syndrome

B. Cerebral amyloid angiopathy

C. Moyamoya vasculopathy

D. Fibromuscular dysplasia

E. Giant cell arteritis

157. A patient with symptomatic adult moyamoya has recurrent ischemic events despite conservative medical therapy. Which treatment most directly addresses the underlying hemodynamic problem?

A. Carotid endarterectomy because removing plaque at the cervical bifurcation restores flow through the terminal intracranial carotid arteries

B. Intracranial balloon angioplasty of every narrowed segment because moyamoya stenoses respond durably to routine endovascular dilation

C. Long-term anticoagulation alone because thromboembolism from atrial fibrillation is the primary mechanism of moyamoya ischemia

D. Surgical cerebral revascularization using direct or indirect bypass techniques

E. High-dose corticosteroids because terminal carotid narrowing in moyamoya is caused by an active granulomatous vasculitis

158. A 34-year-old woman has carotid and vertebral artery dissections. Renal artery imaging shows alternating stenoses and dilatations with a 'string-of-beads' appearance. What systemic arteriopathy is most likely?

A. Cerebral amyloid angiopathy

B. Antiphospholipid syndrome

C. Takayasu arteritis

D. Atherosclerosis

E. Fibromuscular dysplasia

159. A 46-year-old man has weeks of progressive headaches, cognitive slowing, and recurrent multifocal ischemic strokes. Angiography shows irregular stenoses, CSF has lymphocytic pleocytosis and elevated protein, and secondary causes are excluded. Which diagnosis is most likely?

A. Atherosclerotic intracranial disease

B. Moyamoya disease

C. Cerebral amyloid angiopathy

D. Reversible cerebral vasoconstriction syndrome

E. Primary angiitis of the central nervous system

160. A patient is being evaluated for possible primary CNS vasculitis, but the history is dominated by recurrent thunderclap headaches and the CSF is normal. Which alternative should rise on the differential?

A. Primary CNS lymphoma

B. Reversible cerebral vasoconstriction

C. Multiple sclerosis

D. CADASIL (NOTCH3 arteriopathy)

E. Hypertensive small-vessel disease

161. A 70-year-old woman with CAA develops a small atraumatic convexity subarachnoid hemorrhage over the frontal sulci without basal cistern blood. Which association is most plausible?

A. Carotid dissection because cervical mural hematoma directly leaks into the subarachnoid space over the ipsilateral convexity

B. Cortical superficial siderosis and transient focal neurologic episodes related to cerebral amyloid angiopathy

C. A ruptured basilar tip aneurysm because aneurysmal SAH characteristically confines blood to a single high-convexity sulcus

D. Hypertensive pontine arteriopathy because deep perforator rupture usually produces isolated superficial sulcal blood over the frontal lobe

E. Moyamoya disease because basal collateral rupture never causes deep hemorrhage and exclusively produces convexity subarachnoid blood

162. A 33-year-old woman with thunderclap headaches has a small convexity subarachnoid hemorrhage and multifocal arterial vasoconstriction. Which unifying diagnosis is most likely?

A. Hypertensive arteriopathy because deep perforator disease produces sulcal blood and rapidly reversible large-artery narrowing

B. Primary CNS vasculitis because inflammatory vasculitis usually presents with repeated thunderclap headaches and completely normal CSF

C. Aneurysmal subarachnoid hemorrhage because ruptured aneurysms routinely cause diffuse reversible beading of multiple intracranial arteries at presentation

D. Cerebral amyloid angiopathy because CAA is the most common cause of thunderclap headache and multifocal vasoconstriction in young adults

E. Reversible cerebral vasoconstriction syndrome complicated by convexity subarachnoid hemorrhage

163. A 24-year-old man with sickle cell disease has an acute ischemic stroke. Which disease-specific treatment is an important early priority in addition to standard stroke care?

A. Routine platelet transfusion because sickled erythrocytes cause stroke by inducing severe thrombocytopenia in cerebral vessels

B. Delay all transfusion until several weeks after stroke because changing blood viscosity acutely increases recurrent cerebral ischemia

C. Use long-term aspirin alone because transfusion does not reduce recurrent cerebrovascular risk in sickle cell disease

D. High-dose corticosteroids because sickle-cell stroke is caused primarily by autoimmune cerebral vasculitis rather than anemia and vasculopathy

E. Urgent transfusion therapy to reduce the proportion of sickled hemoglobin and improve oxygen delivery

164. A 9-year-old child with sickle cell disease has elevated transcranial Doppler velocities but no prior overt stroke. What is the purpose of this finding?

A. It proves an acute cerebral infarct has already occurred and therefore requires emergent thrombectomy despite no symptoms

B. It indicates cerebral venous thrombosis because high arterial Doppler velocity is caused primarily by impaired dural sinus drainage

C. It identifies children at increased stroke risk who may benefit from preventive chronic transfusion therapy

D. It is a normal adaptation to anemia and has no relationship to future stroke risk in children with sickle cell disease

E. It mandates carotid endarterectomy because elevated intracranial velocities arise from cervical carotid plaque in pediatric patients

165. A 39-year-old woman has migraine with aura, early-onset lacunar strokes, mood symptoms, and a parent with similar disease. MRI shows anterior temporal pole and external capsule white-matter lesions. Which diagnosis is most likely?

A. Primary CNS vasculitis

B. Cerebral amyloid angiopathy

C. Moyamoya disease

D. CADASIL (NOTCH3 arteriopathy)

E. Fabry disease

166. A 45-year-old man has early strokes, neuropathic pain in the hands and feet, angiokeratomas, renal dysfunction, and left ventricular hypertrophy. Which disorder should be suspected?

A. Cerebral amyloid angiopathy

B. Antiphospholipid syndrome

C. CADASIL

D. Moyamoya disease

E. Fabry disease

167. A 36-year-old woman has recurrent small-vessel strokes and cerebral microhemorrhages. Her mother had porencephalic cysts and kidney disease. Which genetic vasculopathy should be considered?

A. Fabry disease (GLA deficiency)

B. Moyamoya arteriopathy

C. Cerebral amyloid angiopathy

D. COL4A1/COL4A2-related arteriopathy

E. CADASIL (NOTCH3 arteriopathy)

168. A 52-year-old man received head-and-neck radiation 15 years ago and now has accelerated bilateral carotid stenosis with a TIA. Which mechanism is most likely?

A. Reversible cerebral vasoconstriction because remote radiation causes transient segmental narrowing that disappears within weeks

B. Cerebral amyloid angiopathy because ionizing radiation primarily deposits beta-amyloid in cervical carotid arteries rather than brain vessels

C. Radiation-induced vasculopathy with premature arterial fibrosis and atherosclerotic change

D. Fibromuscular dysplasia because radiation creates the classic noninflammatory medial fibroplasia pattern in renal and cervical arteries

E. Moyamoya disease because all post-radiation stenosis is confined to terminal intracranial carotid arteries with basal collaterals

169. A 29-year-old woman with antiphospholipid syndrome has recurrent arterial ischemic strokes despite no conventional vascular risk factors. Which mechanism best explains the events?

A. Moyamoya disease

B. Antiphospholipid syndrome

C. Small-vessel lipohyalinosis

D. Cerebral amyloid angiopathy

E. Reversible cerebral vasoconstriction

170. A 35-year-old woman with prior ischemic stroke has persistent high-risk antiphospholipid antibody positivity and recurrent arterial thrombosis. Which long-term antithrombotic principle is most appropriate?

A. No long-term therapy is indicated once the acute stroke resolves because antiphospholipid antibodies do not predict recurrent thrombosis

B. Dual antiplatelet therapy should replace anticoagulation because combining aspirin and clopidogrel neutralizes pathogenic antibodies directly

C. Aspirin alone is always sufficient because antiphospholipid-related arterial events are purely platelet-mediated rather than thrombotic

D. A direct oral factor Xa inhibitor is preferred in triple-positive APS because trials show lower arterial recurrence than warfarin

E. Vitamin K antagonist anticoagulation is generally favored over a direct oral anticoagulant for high-risk thrombotic antiphospholipid syndrome

171. A 33-year-old woman develops an ischemic stroke 4 days after an uncomplicated cesarean delivery. Which physiologic change increases her vascular risk in the postpartum period?

A. The puerperium eliminates venous stasis immediately, so postpartum stroke is unrelated to coagulation or vascular complications

B. Estrogen falls after delivery and therefore prevents cerebral venous thrombosis and arterial embolism during the first six weeks

C. Platelet function is absent after cesarean delivery, making hemorrhage the only plausible cerebrovascular event during the postpartum period

D. Persistent pregnancy-related hypercoagulability continues into the puerperium and raises arterial and venous thrombotic risk

E. Pregnancy causes a prolonged systemic bleeding tendency after delivery that makes thrombosis less likely than at any other adult age

172. A 40-year-old woman with ischemic stroke uses an estrogen-containing oral contraceptive and smokes heavily. No other source is found. What prevention counseling is most appropriate?

A. Continue both exposures because neither smoking nor estrogen meaningfully affects ischemic stroke risk in premenopausal women

B. Stop smoking but continue estrogen because exogenous estrogen does not influence arterial or venous thrombosis under any circumstance

C. Address both smoking and estrogen exposure because the combination can amplify thrombotic vascular risk

D. Continue smoking if the contraceptive is stopped because tobacco-related cerebrovascular risk disappears below age 50

E. Replace estrogen with a higher-dose formulation because stronger hormonal suppression reduces thrombotic risk after ischemic stroke

173. A 67-year-old man survives a moderate MCA stroke and has persistent depression, reduced participation in therapy, and no active suicidality. Why should depression be addressed directly?

A. Depression should be ignored for the first year because early emotional symptoms always resolve spontaneously as motor recovery occurs

B. Post-stroke depression can impair quality of life, engagement, and recovery and should be screened for and treated

C. Depression is an expected neurologic deficit that cannot respond to psychotherapy or medication and therefore needs no specific treatment

D. Treating depression is avoided because all antidepressants substantially increase recurrent ischemic stroke risk regardless of drug class

E. Mood symptoms matter only if psychosis is present because depression alone has no effect on rehabilitation participation or caregiver burden

174. A 71-year-old woman with stroke is medically stable but has persistent neglect, unsafe transfers, and severe dysphagia. Her family requests immediate discharge home without services. What is the best response?

A. Keep the patient hospitalized indefinitely because any residual neurologic deficit makes discharge to home or rehabilitation inappropriate

B. Arrange a feeding tube and home discharge without therapy assessment because nutritional access eliminates the need for rehabilitation planning

C. Discharge immediately because family preference always overrides clinical assessment of aspiration, fall, and caregiver safety risks

D. Explain the specific safety risks and use multidisciplinary discharge planning to identify the safest setting consistent with the patient's goals

E. Refuse to discuss home discharge because clinicians alone determine disposition and patient or family values should not influence planning

175. A 69-year-old man with a large stroke lacks decision-making capacity. His previously completed advance directive states that he would accept temporary ventilation and rehabilitation but would not want prolonged dependence if recovery is impossible. How should the team use this information?

A. Withdraw all treatment immediately because any mention of avoiding dependence means the patient rejected temporary intensive care as well

B. Apply substituted judgment by aligning current treatment with the patient's documented values while reassessing prognosis over time

C. Allow the family to choose care based solely on their own preferences because the patient's prior stated values are no longer relevant

D. Use the admission severity score as the sole decision rule because prognostic models are more authoritative than individualized preferences

E. Ignore the directive because advance preferences do not apply to acute neurologic illness when the exact outcome remains uncertain

176. A patient with limited English proficiency is being asked to consent to a high-risk neurovascular procedure. The patient's adult child offers to translate. What is the best approach?

A. Use the adult child as the sole interpreter because family members understand the patient's values better than trained interpreters

B. Proceed without interpretation because emergency neurologic procedures do not require meaningful informed consent when time is limited

C. Ask the child to sign consent in place of the patient even though the patient has decision-making capacity and wants to decide personally

D. Avoid discussing risks because detailed information through an interpreter may increase anxiety and delay the procedure

E. Use a qualified medical interpreter and speak directly with the patient while incorporating family support if the patient wishes

177. A competent patient declines a recommended intervention after accurately explaining the expected benefits, risks, and alternatives. The clinician believes the choice is unwise. What is the most appropriate response?

A. Avoid documenting the refusal because a written record could increase liability if the patient's condition later worsens

B. Override the refusal because physicians may substitute their judgment whenever a neurologic treatment has substantial expected benefit

C. Ask family members to authorize the procedure despite the patient's capacity because disagreement with clinicians proves impaired judgment

D. Respect the informed refusal, document the discussion, and continue to offer appropriate care consistent with the patient's goals

E. Discharge the patient immediately from all care because refusal of one recommended treatment ends the therapeutic relationship

178. A neurologist realizes that a medication dose entered earlier in the day was ten times higher than intended, but the pharmacy caught the error before administration. What is the best response?

A. Wait to see whether a similar error reaches a patient before changing the ordering process or reporting the event

B. Avoid discussing the event with the team because near misses are too common to provide useful quality-improvement information

C. Correct the order, disclose the near miss through appropriate safety channels, and examine the system factors that allowed it

D. Delete the order without reporting it because no medication reached the patient and therefore no safety event occurred

E. Blame the pharmacist for not recognizing the intended dose because intercepted prescribing errors are pharmacy responsibility

179. A hospital implements a new protocol intended to reduce door-to-imaging time. Which outcome best measures whether the protocol achieved its operational goal?

A. Ask one clinician whether imaging feels faster because subjective impressions are more reliable than time-stamped workflow data

B. Measure hospital mortality alone because a process metric is unnecessary when the intervention targets speed rather than treatment content

C. Compare the median door-to-imaging time before and after implementation while monitoring balancing safety measures

D. Stop measurement once the protocol is launched because repeated data collection can itself change clinician behavior

E. Count only the number of protocol pages distributed because dissemination is equivalent to successful process improvement

180. A diagnostic test has high sensitivity but modest specificity. How should a negative result generally be interpreted when the disease is reasonably prevalent in the tested population?

A. A negative result strongly confirms disease because highly sensitive tests generate mostly true-positive results and few true negatives

B. Sensitivity determines only the false-positive rate, so it has no relationship to the meaning of a negative test result

C. A negative result can be ignored because test sensitivity influences only screening cost and not diagnostic probability

D. A negative result is useful for lowering the probability of disease because highly sensitive tests have few false negatives

E. High sensitivity guarantees that every positive result is correct regardless of disease prevalence or test specificity

181. A test has 95% specificity. What does this statistic mean?

A. Five percent of affected patients will test positive because specificity measures the true-positive fraction among diseased patients

B. Among people without the target condition, about 95% will have a negative test result

C. Among people with the condition, about 95% will have a positive result regardless of disease prevalence

D. Among all positive results, about 95% represent true disease in every population in which the test is used

E. The test reduces the pretest probability by 95 percentage points whenever the result is negative

182. A randomized trial reports a relative risk of 0.80 with a 95% confidence interval of 0.62 to 1.03 for its primary binary outcome. Which interpretation is most accurate?

A. The point estimate suggests benefit, but the confidence interval includes no effect, so conventional statistical significance is not established

B. The trial is statistically significant because most of the confidence interval lies below 1.0 even though the interval crosses 1.0

C. The confidence interval means 95 percent of individual patients will have a relative risk between 0.62 and 1.03

D. The treatment increases risk by 80 percent because any relative risk below 1.0 represents harm to the intervention group

E. The result proves a 20 percent benefit in every patient because the point estimate alone determines both significance and certainty

183. A meta-analysis shows substantial heterogeneity among included studies. What is the best first response when interpreting the pooled estimate?

A. Examine clinical and methodological differences among studies before assuming one pooled effect applies uniformly

B. Select only the study with the largest effect because heterogeneity indicates that smaller effects are likely due to measurement error

C. Convert every outcome into a p value because statistical significance removes the need to examine study design differences

D. Assume publication bias is the sole cause because heterogeneity cannot arise from genuine differences in populations or treatment effects

E. Ignore heterogeneity because pooling automatically eliminates differences in patient populations, interventions, and outcome definitions

184. A patient with an implanted medical device needs MRI. The exact device model is unknown. What is the safest next step?

A. Proceed with MRI because all modern implanted devices are automatically safe at any field strength and sequence parameters

B. Verify the device identity and MRI-conditional requirements before scanning rather than assuming compatibility

C. Cancel all future MRI permanently because an unidentified device can never be investigated or cleared for conditional scanning

D. Use gadolinium contrast because contrast administration prevents magnetic interaction between the scanner and implanted devices

E. Perform the scan only after turning off the hospital's monitoring equipment because external monitors cause the main implant-related risk

185. A patient with advanced chronic kidney disease requires contrast-enhanced vascular imaging for a potentially life-threatening neurologic condition. What is the best general principle?

A. Balance the immediate diagnostic benefit against contrast-related risk and use the lowest-risk effective imaging strategy without reflexively withholding necessary imaging

B. Select gadolinium or iodinated contrast solely by cost because kidney function does not affect contrast-agent risk profiles

C. Withhold all contrast-enhanced imaging because kidney disease makes contrast risk more important than any acute neurologic diagnosis

D. Use double the usual contrast dose because impaired renal clearance reduces vascular enhancement and otherwise makes imaging nondiagnostic

E. Delay imaging until kidney function normalizes spontaneously because urgent neurologic decisions should never influence contrast choices

186. A patient becomes acutely agitated and tries to leave before an urgent evaluation is complete. Which step is essential before overriding the patient's decision?

A. Assess decision-making capacity for the specific choice and address reversible causes of impaired cognition or communication

B. Use the patient's age as the primary capacity criterion because adults over 65 require surrogate consent for high-risk neurologic decisions

C. Determine capacity from orientation questions alone because a patient who knows the date necessarily understands treatment consequences

D. Assume incapacity because agitation by itself proves the patient cannot understand or appreciate any medical decision

E. Ask security to restrain the patient immediately because clinicians may override refusal whenever testing is medically advisable

187. A clinician must communicate uncertain prognosis after a severe neurologic injury. Which approach is best?

A. Avoid discussing uncertainty because acknowledging limits of prediction undermines confidence in the treatment team

B. Use a prognostic score as an exact individual forecast and omit the patient's values because objective numbers should drive decisions

C. Wait until recovery is complete before discussing prognosis because early conversations cannot contribute to shared decision-making

D. Give the most pessimistic outcome as a certainty because families need a single prediction to make timely decisions

E. Describe the range of plausible outcomes, what is known and uncertain, and how upcoming clinical milestones may refine prognosis

188. A patient with impaired consciousness has no available surrogate and needs an immediately lifesaving procedure. There is no time to obtain court authorization. What ethical principle supports treatment?

A. Emergency implied consent permits necessary treatment when delay threatens life and the patient cannot decide

B. The procedure must always be withheld until a legally appointed guardian can sign consent regardless of imminent risk

C. The treating physician becomes the patient's permanent surrogate and may make all future decisions without review or documentation

D. Consent requirements disappear for the entire hospitalization once one emergency procedure is performed under implied consent

E. The hospital administrator must provide clinical consent because administrative authority replaces patient autonomy in all emergencies

189. A neurologist receives an imaging report containing an unexpected critical finding after the patient has already left the department. What is the best systems response?

A. Notify only billing staff because documentation of communication is less important than coding the additional diagnosis correctly

B. Use a closed-loop process to ensure the responsible clinician and patient are reached and the needed follow-up is completed

C. Place the report in the electronic record and assume that passive availability guarantees the critical finding will be addressed

D. Wait until the next scheduled visit because contacting patients about unexpected findings increases anxiety and should be avoided

E. Delete the finding from the report if it is unrelated to the original indication because incidental results create unnecessary responsibility

190. A hospital has repeated delays because outside imaging cannot be viewed when transferred patients arrive. Which systems change is most likely to improve care?

A. Delay transfer until every image is formally interpreted twice at the referring hospital because destination review provides no additional value

B. Stop accepting transferred patients because technical barriers cannot be meaningfully reduced through standardized workflows

C. Repeat every outside scan automatically because duplication is more reliable than building image-sharing systems across hospitals

D. Require families to transport printed films because patient-carried images are the most dependable way to support emergency transfer

E. Develop interoperable image-transfer and prearrival review workflows with clear ownership for troubleshooting failures

191. A patient asks whether a medical test can be ordered solely because the clinician is curious, even though the result would not change diagnosis, treatment, prognosis, or counseling. Which principle best applies?

A. Avoid explaining the test because unnecessary testing is acceptable as long as the patient does not know it lacks a clear purpose

B. Testing should have a meaningful clinical purpose because low-value studies can create false positives, cost, and downstream harm

C. Order multiple additional tests simultaneously because broad testing reduces the probability of incidental findings and diagnostic uncertainty

D. Use testing only when it is guaranteed to be normal because unexpected results increase follow-up work and therefore should be prevented

E. Order the test because more information is always beneficial even when it cannot influence any patient-centered decision

192. A trainee presents a patient's examination findings but omits the patient's baseline function and goals when recommending an invasive treatment. What additional information is most important?

A. Only the imaging abnormality matters because patient goals and baseline function introduce subjective information into evidence-based care

B. Age alone should replace functional history because chronological age is the most accurate predictor of benefit from every neurologic treatment

C. The trainee should avoid discussing prognosis because treatment selection should never incorporate the patient's preferred quality of life

D. Family preferences should automatically replace the patient's values because relatives are better positioned to judge acceptable disability

E. Baseline independence, comorbidities, patient values, and acceptable outcomes should be integrated with disease severity when making treatment decisions

193. A 38-year-old woman has recurrent unilateral throbbing headaches with photophobia, nausea, and visual scintillations that gradually spread across the field over 20 minutes before headache begins. Neurologic examination is normal between attacks. Which diagnosis is most likely?

A. Migraine with visual aura

B. Giant cell arteritis

C. Occipital transient ischemic attack

D. Focal occipital seizure

E. Aneurysmal subarachnoid hemorrhage

194. A 74-year-old woman has a new unilateral temporal headache, jaw claudication, scalp tenderness, and transient monocular visual loss. ESR and CRP are elevated. What should be done immediately?

A. Give a triptan because new unilateral headache with visual symptoms in an older adult is most consistent with late-onset migraine

B. Begin anticoagulation because jaw claudication indicates carotid dissection with thromboembolic retinal ischemia

C. Wait for temporal artery biopsy before treatment because corticosteroids given first make the diagnosis impossible to confirm

D. Treat with aspirin alone because the visual symptoms are caused by platelet emboli rather than inflammatory arterial occlusion

E. Start high-dose glucocorticoid treatment promptly while arranging confirmatory evaluation for giant cell arteritis

195. A 29-year-old woman has sudden excruciating unilateral orbital pain with lacrimation, nasal congestion, and marked restlessness. Episodes last 45 minutes and occur twice nightly for several weeks. What is the most likely diagnosis?

A. Cluster headache

B. Trigeminal neuralgia

C. Carotid transient ischemic attack

D. Subarachnoid hemorrhage

E. Migraine with aura

196. A 70-year-old man has recurrent episodes of right hand jerking that march to the forearm over 30 seconds, followed by five minutes of weakness. The events are stereotyped and not related to standing. Which diagnosis is most likely?

A. Limb-shaking transient ischemic attacks

B. Migraine aura with motor symptoms

C. Transient global amnesia

D. Focal motor seizure with Todd paralysis

E. Convulsive syncope

197. A patient with acute brain injury has persistent unexplained depressed consciousness without convulsive movements. Which test is most useful for detecting nonconvulsive status epilepticus?

A. Single-lead cardiac telemetry

B. Routine carotid duplex ultrasonography

C. Continuous EEG monitoring

D. Electromyography

E. Lumbar puncture

198. A 62-year-old woman has a first unprovoked focal seizure. MRI shows a remote cortical infarct. Which factor increases the likelihood of seizure recurrence and may support starting antiseizure medication?

A. Age over 60 because late-onset seizures are almost always isolated events without an underlying epileptogenic lesion

B. No family history of epilepsy because family history is required for recurrence after a first focal seizure

C. Normal electrolyte values because absence of a metabolic trigger makes future seizures less likely than after provoked events

D. The seizure lasting less than two minutes because brief duration eliminates recurrence risk from structural brain disease

E. A structural cortical lesion that provides an enduring epileptogenic substrate

199. A 65-year-old man with prior stroke snores loudly, has witnessed apneas, morning headaches, and excessive daytime sleepiness. Which diagnosis should be evaluated because it also affects vascular risk?

A. REM sleep behavior disorder

B. Obstructive sleep apnea

C. Restless legs syndrome

D. Fatal familial insomnia

E. Narcolepsy type 1

200. A patient with suspected obstructive sleep apnea has significant cardiopulmonary disease and recent neurologic injury. Which diagnostic strategy is generally most appropriate?

A. Use pulse oximetry alone because oxygen desaturation can distinguish obstructive from central apneas and quantify sleep stages accurately

B. Make the diagnosis from snoring history alone because formal sleep testing does not alter treatment selection or pressure settings

C. Use EEG without respiratory channels because cortical arousals are sufficient to calculate the apnea-hypopnea index

D. Delay testing indefinitely because sleep-disordered breathing after neurologic illness cannot be treated until all deficits have resolved

E. Use attended polysomnography when comorbidity makes a simple home sleep apnea test less suitable

201. A patient with moderate obstructive sleep apnea asks about treatment after stroke. Which management is most appropriate?

A. No treatment is needed because OSA influences daytime sleepiness but has no relevance to hypertension or vascular health

B. Avoid positive airway pressure because treating nocturnal obstruction lowers blood pressure and therefore increases recurrent cerebral ischemia

C. Use sedative-hypnotic medication as first-line therapy because deeper sleep prevents upper-airway collapse more effectively than airway pressure

D. Offer positive airway pressure therapy and address weight, sleep position, alcohol, and other contributing factors as appropriate

E. Treat only with supplemental oxygen because oxygen corrects the obstructive events and arousals even when the airway remains closed

202. A 63-year-old man develops fever, confusion, aphasia, and focal seizures. MRI shows temporal lobe edema and hemorrhagic signal abnormality. What diagnosis should be treated empirically while PCR testing is pending?

A. Herpes simplex virus encephalitis

B. West Nile encephalitis

C. Neurosyphilis

D. Acute ischemic stroke from small-vessel lipohyalinosis

E. Progressive multifocal leukoencephalopathy

203. A patient with bacterial meningitis develops acute focal deficits and MRI shows multifocal infarcts. Which vascular complication can explain this finding?

A. Infectious cerebral vasculopathy or vasculitis causing arterial thrombosis and ischemic infarction

B. Moyamoya disease because bacterial infection produces permanent bilateral terminal carotid occlusion within hours of meningeal inflammation

C. Fibromuscular dysplasia because meningeal infection causes medial fibroplasia and string-of-beads narrowing in cerebral arteries

D. Reversible cerebral vasoconstriction because bacterial meningitis causes only transient sympathetic vasospasm and never inflammatory arterial injury

E. Cerebral amyloid angiopathy because meningitis rapidly deposits beta-amyloid in cortical vessels and causes multifocal ischemia

204. A 42-year-old patient with advanced HIV has weeks of progressive hemiparesis and cognitive change. MRI shows multifocal nonenhancing white-matter lesions without mass effect, and JC-virus PCR is positive. What is the most likely diagnosis?

A. Herpes simplex encephalitis

B. Primary CNS lymphoma

C. Progressive multifocal leukoencephalopathy

D. Acute disseminated encephalomyelitis

E. Subacute ischemic stroke

205. A 70-year-old man on anticoagulation falls and strikes his head. He initially feels well but six hours later develops headache, confusion, and left weakness. CT shows a crescent-shaped extra-axial collection crossing suture lines. What is the diagnosis?

A. Acute subdural hematoma

B. Cerebral venous thrombosis

C. Epidural hematoma

D. Aneurysmal subarachnoid hemorrhage

E. Lobar intracerebral hemorrhage

206. A patient with acute cervical spinal cord compression develops rapidly progressive quadriparesis and urinary retention. MRI shows a large epidural mass compressing the cord. What is the most important management principle?

A. Perform lumbar puncture because reducing CSF pressure below the lesion directly decompresses the cervical spinal cord

B. Use thrombolysis because acute quadriparesis from a spinal epidural process is best treated as an arterial ischemic stroke

C. Obtain urgent spine surgical evaluation because progressive cord compression is a neurologic emergency

D. Delay intervention for several weeks because spinal cord deficits recover best after edema stabilizes before decompression

E. Treat only with aspirin because cord compression produces ischemia through platelet emboli rather than mechanical injury

207. A 58-year-old man has sudden severe back pain followed by bilateral leg weakness, loss of pain and temperature below the umbilicus, and preserved vibration sense. MRI shows an anterior spinal cord infarct. Which artery is most likely involved?

A. The artery of Adamkiewicz only

B. The anterior spinal artery

C. A vertebral artery

D. A middle cerebral artery

E. A posterior spinal artery

208. A 72-year-old woman has sudden painless loss of vision in the right eye with a relative afferent pupillary defect and a pale retina with a cherry-red spot. What is the diagnosis?

A. Occipital lobe infarction

B. Optic neuritis

C. Central retinal vein occlusion

D. Central retinal artery occlusion

E. Migraine aura

209. A 68-year-old man develops acute binocular horizontal diplopia. The left eye cannot abduct, but the right eye moves normally. Which lesion is most likely?

A. A right trochlear nerve palsy

B. A left third cranial nerve palsy

C. A right internuclear ophthalmoplegia

D. A left optic neuropathy

E. Left sixth-nerve palsy

210. A 57-year-old woman has vertigo lasting seconds when turning in bed. Dix-Hallpike testing provokes a brief latency followed by fatigable torsional-upbeating nystagmus. What is the most likely diagnosis?

A. Meniere disease

B. Posterior-canal BPPV

C. Cerebellar hemorrhage

D. Lateral medullary infarction

E. Vestibular neuritis

211. A patient has acute continuous vertigo, nausea, and gait imbalance. The head impulse test is normal, nystagmus changes direction with gaze, and a vertical skew deviation is present. What does this HINTS pattern suggest?

A. Benign positional vertigo

B. Central vestibular lesion

C. Meniere disease

D. Orthostatic hypotension

E. A peripheral vestibular neuritis

212. A malnourished patient with alcoholism develops confusion, gait ataxia, and ophthalmoplegia. What treatment should be given promptly?

A. Parenteral thiamine before or with glucose to treat suspected Wernicke encephalopathy

B. High-dose folate alone because folate deficiency causes the acute triad of ophthalmoplegia, ataxia, and encephalopathy

C. Intravenous glucose alone because carbohydrate administration rapidly corrects the underlying thiamine deficiency without supplementation

D. Naloxone because opioid intoxication is the classic cause of ophthalmoplegia with broad-based gait and memory impairment

E. Levodopa because dopamine deficiency in the basal ganglia produces acute Wernicke-like ocular motor and cognitive symptoms

213. A patient with severe hyponatremia is corrected from 108 to 132 mEq/L in 12 hours. Several days later, the patient develops dysarthria, dysphagia, and quadriparesis. MRI shows central pontine T2 hyperintensity. What is the diagnosis?

A. Guillain-Barré syndrome

B. Acute basilar artery thrombosis

C. Wernicke encephalopathy

D. Osmotic demyelination syndrome

E. Posterior reversible encephalopathy syndrome

214. A 52-year-old man is found unconscious with pinpoint pupils and respiratory depression. Neurologic examination improves rapidly after naloxone. What is the most likely cause?

A. Severe hypoglycemia

B. Basilar artery occlusion

C. Pontine hemorrhage

D. Organophosphate poisoning

E. Opioid intoxication

215. A 73-year-old man has progressive impairment in episodic memory over three years, followed by word-finding difficulty and disorientation. MRI shows disproportionate hippocampal and medial temporal atrophy. What syndrome is most likely?

A. Alzheimer-type major neurocognitive disorder

B. Dementia with Lewy bodies

C. Vascular cognitive impairment

D. Normal pressure hydrocephalus

E. Behavioral-variant frontotemporal dementia

216. A 68-year-old woman has cognitive decline characterized by slowed processing, impaired set shifting, gait difficulty, and urinary urgency. MRI shows extensive confluent white-matter disease and multiple lacunes with relatively preserved hippocampal volume. Which diagnosis is most likely?

A. Alzheimer disease

B. Subcortical vascular cognitive impairment

C. Semantic-variant primary progressive aphasia

D. Dementia with Lewy bodies

E. Frontotemporal dementia

217. A patient after a right hemispheric stroke repeatedly denies left-sided weakness and attempts to stand despite dense hemiplegia. What neurobehavioral syndrome is present?

A. Anosognosia for hemiplegia

B. Akinetic mutism

C. Prosopagnosia

D. Alexia without agraphia

E. Capgras syndrome

218. A 34-year-old woman develops painful monocular visual loss followed months later by a cervical myelitis. MRI shows demyelinating lesions, and CSF has oligoclonal bands. Which diagnosis is most likely?

A. CADASIL

B. Fibromuscular dysplasia

C. Multiple sclerosis

D. Cerebral amyloid angiopathy

E. Moyamoya disease

219. A 27-year-old woman has a week of otherwise unexplained intractable hiccups and vomiting, followed months later by a longitudinally extensive cervical myelitis. Serum aquaporin-4 IgG is positive. Which diagnosis best unifies these attacks?

A. Ischemic optic neuropathy with myelopathy

B. Neuromyelitis optica spectrum disorder

C. CADASIL-associated arteriopathy

D. Spinal dural arteriovenous fistula

E. Relapsing-remitting multiple sclerosis

220. A 59-year-old man with small-cell lung cancer develops subacute severe anterograde memory loss, seizures, and medial temporal T2 hyperintensity. What diagnosis should be considered?

A. Bilateral posterior cerebral artery infarction

B. Herpes simplex encephalitis

C. Alzheimer disease

D. Transient global amnesia

E. Paraneoplastic limbic encephalitis

Answers

1. E. A disabling stroke with a proximal large-vessel occlusion requires rapid reperfusion. When intravenous thrombolysis is indicated, planned thrombectomy is not a reason to delay or omit it; the patient should receive thrombolysis while the endovascular pathway proceeds in parallel. Waiting for response wastes time, and excessive blood-pressure reduction can compromise penumbral perfusion.

2. A. The important distinction is disability, not the numerical NIHSS score alone. For clearly nondisabling minor ischemic stroke within the thrombolysis window, contemporary guidance favors antiplatelet therapy, often short-term dual therapy when standard criteria are met, rather than routine thrombolysis. Disabling language, visual, gait, or dominant-hand deficits would change the decision.

3. B. A low NIHSS score does not necessarily mean a nondisabling stroke. Severe aphasia can prevent independent communication and work and is therefore disabling even when the total score is small. Within 4.5 hours, eligible patients with disabling deficits should be treated rapidly without requiring advanced imaging simply because the NIHSS is low.

4. C. The 2026 guideline endorses either alteplase or tenecteplase for eligible patients in the standard 4.5-hour thrombolytic window. Tenecteplase has practical advantages because it is given as a bolus, but the key board point is that both agents are guideline-supported options. Choice of agent does not justify delaying reperfusion.

5. E. The accepted acute ischemic stroke regimen is tenecteplase 0.25 mg/kg given as one intravenous bolus, capped at 25 mg. The higher 0.4 mg/kg regimen has not shown the same safety-benefit profile for stroke. The 0.9 mg/kg bolus-plus-infusion regimen describes alteplase dosing, not tenecteplase.

6. A. Blood pressure must be below 185/110 mm Hg before intravenous thrombolysis. A short-acting, titratable intravenous agent can be used, and successful control allows treatment if other criteria are satisfied. Overly aggressive reduction is unnecessary and may reduce perfusion to threatened brain tissue.

7. C. After intravenous thrombolysis, the usual ceiling is below 180/105 mm Hg for the first 24 hours. Recent evidence does not support routine intensive systolic lowering below 140 mm Hg after thrombolysis and suggests that overly aggressive reduction can be harmful after thrombectomy. Smooth control without hypotension is the goal.

8. A. The 2026 acute stroke guideline specifically discourages intensive systolic reduction below 140 mm Hg after EVT, even with complete reperfusion, because trials have not improved outcome and some have suggested harm. Blood pressure should be managed within accepted postreperfusion ranges while avoiding both severe hypertension and hypotension.

9. E. Selected patients with unknown onset can be treated using tissue-based imaging rather than clock time alone. A DWI-positive lesion with little FLAIR change suggests relatively recent ischemia and can support thrombolysis when other eligibility criteria are met. Absence of an EVT target does not eliminate the possibility of intravenous reperfusion.

10. B. Contemporary guidance supports thrombolysis in selected patients 4.5 to 9 hours from onset when advanced imaging demonstrates favorable tissue mismatch. The imaging identifies potentially salvageable brain despite the later clock time. Thrombectomy still requires an appropriate arterial target rather than mismatch alone.

11. A. Current evidence supports EVT for appropriately selected basilar artery occlusion within 24 hours, particularly when NIHSS is at least 10 and imaging does not show extensive irreversible injury. Posterior-circulation stroke can deteriorate catastrophically, so waiting for coma is inappropriate. Anticoagulation does not replace reperfusion of an occluded basilar artery.

12. E. Randomized trials have expanded EVT eligibility to selected patients with larger ischemic cores who previously would have been excluded. Core size remains important for prognosis and patient selection, but it is not an automatic disqualifier. Decisions integrate clinical severity, baseline function, time, occlusion site, and imaging.

13. A. The 2026 guideline emphasizes local systems of care rather than a universal destination rule. If a nearby thrombolysis-capable hospital has rapid transfer pathways, stopping there may be efficient; when transfer is predictably prolonged and the additional travel is modest, direct transport to an EVT-capable center can be reasonable. The objective is fastest effective reperfusion.

14. D. Mobile stroke units can perform rapid imaging, specialist assessment, and thrombolysis before hospital arrival, shortening time to treatment. They do not replace thrombectomy-capable hospitals or eliminate diagnostic uncertainty. Their value depends on local infrastructure, geography, and the ability to integrate them into the broader stroke system.

15. D. Trials of adjuvant antithrombotic drugs such as argatroban or eptifibatide given with IV thrombolysis have not shown clinical benefit sufficient to support routine use. Combining potent antithrombotic effects can also increase bleeding risk. Standard reperfusion therapy should be delivered without unproven add-on regimens.

16. E. Hyperglycemia is associated with worse stroke outcomes, but intensive normalization to 80-130 mg/dL has not improved functional outcome and increases severe hypoglycemia. Reasonable treatment of substantial hyperglycemia is appropriate without delaying indicated reperfusion. Hypoglycemia must also be avoided because it can injure the brain and mimic stroke.

17. A. Severe hypoglycemia is a classic reversible stroke mimic and must be corrected promptly. Persistent focal deficits after normalization can still represent true stroke and should then be evaluated urgently for reperfusion. The immediate priority is correcting a dangerous metabolic abnormality that may entirely explain the examination.

18. E. Seizures can occur at the onset of cortical ischemic stroke. The key is whether the persisting deficit is attributable to ischemia rather than a transient postictal state; a concordant M1 occlusion strongly supports stroke. Appropriate reperfusion should not be withheld solely because a seizure occurred at onset.

19. A. Rapid improvement is not itself a reason to withhold thrombolysis if a disabling deficit remains. Functional consequences such as severe aphasia can be profound even with a low residual NIHSS score. Treatment decisions should be based on the current disability and eligibility criteria rather than percentage improvement alone.

20. C. A platelet count below 100,000/μL generally precludes routine IV thrombolysis because of bleeding risk. Mechanical thrombectomy has different eligibility considerations and may still be

appropriate for a disabling LVO after individualized procedural assessment. Contraindications to one reperfusion modality should not automatically be transferred to the other.

21. B. Recent direct factor Xa inhibitor exposure with active anticoagulant effect creates important bleeding risk for IV thrombolysis. Timing, renal function, and when available drug-specific testing help determine whether clinically significant activity persists. An LVO may still be treated with thrombectomy when appropriate, because EVT does not have identical exclusions.

22. B. Warfarin use is not an automatic exclusion when the INR is sufficiently low; an INR up to 1.7 is generally within the accepted thrombolysis threshold if other criteria are met. The measured anticoagulant effect, not the medication name alone, guides this decision. Reversal solely to enable thrombolysis is not routine practice.

23. A. Many historical exclusions are relative and depend on the site, timing, hemostasis, and consequences of bleeding. A recent dural puncture deserves careful individualized assessment but is not best handled by a simplistic universal rule. The clinician must balance hemorrhagic risk against the disability expected from untreated stroke.

24. D. Acute intracranial hemorrhage is a contraindication to IV thrombolysis, while remote stable findings require careful characterization. The immediate task is to establish whether the collection represents active or unstable bleeding and to integrate its clinical significance. Stroke severity does not override a true intracranial hemorrhage contraindication.

25. C. In-hospital stroke is often delayed by fragmented workflows. A dedicated stroke alert pathway should trigger rapid bedside assessment, imaging, and parallel review of contraindications and thrombectomy candidacy. The patient does not need to pass through the emergency department before receiving time-sensitive stroke care.

26. A. Evidence for EVT is strongest for established proximal large-vessel targets and selected basilar occlusion. Distal and medium-vessel thrombectomy remains more dependent on anatomy, disability, core size, technical feasibility, and evolving trial data. A disabling deficit may justify consideration, but a universal rule would overstate current evidence.

27. D. The relevant outcome for a patient with prestroke disability may be return to the prior level of function, not achievement of perfect independence. Baseline disability influences prognosis and goals of care but does not by itself create a universal EVT exclusion. Decisions should incorporate prior quality of life, comorbidity, stroke severity, imaging, and patient preferences.

28. A. ASPECTS is a semiquantitative estimate of early ischemic change in the anterior circulation, with lower scores generally indicating larger core injury. Large-core trials have shown that selected patients with low ASPECTS can still benefit from thrombectomy. The score should inform selection and prognosis rather than function as an automatic exclusion.

29. C. The NIHSS underrepresents several posterior-circulation deficits, including visual field loss, gait ataxia, and cognitive problems. Functional disability should therefore be judged directly rather than inferred from a low score. Reperfusion decisions still depend on time, imaging, vessel target, and treatment-specific evidence.

30. C. Abrupt neurologic deterioration after reperfusion requires immediate evaluation for intracranial hemorrhage, contrast-related findings, reocclusion, edema, and other complications. Noncontrast CT is rapid and highly useful for detecting significant hemorrhage. Empiric antithrombotic escalation before imaging could worsen an unrecognized bleed.

31. D. Malignant hemispheric infarction can produce fatal mass effect over the first several days. Early neurosurgical assessment for decompressive hemicraniectomy is appropriate in selected patients, including carefully selected older adults, with discussion of likely disability among survivors. Corticosteroids do not treat cytotoxic stroke edema, and prolonged hyperventilation is only a temporizing measure in emergencies.

32. A. Swollen cerebellar infarction can rapidly compress the brainstem and obstruct the fourth ventricle. Neurologic deterioration, hydrocephalus, or brainstem compression warrants urgent neurosurgical decompression, with ventricular drainage used when appropriate. Lumbar puncture can be dangerous in the setting of posterior fossa mass effect.

33. D. In ischemic stroke patients not receiving reperfusion, permissive hypertension is often allowed unless pressure is extremely high or another medical indication requires treatment. Abrupt large reductions can worsen cerebral perfusion. When treatment is necessary, a modest controlled reduction is preferred rather than normalization to low values.

34. C. Antiplatelet therapy is generally withheld during the first 24 hours after IV thrombolysis, and follow-up brain imaging is obtained before starting it. This reduces the risk of compounding an unrecognized treatment-related hemorrhage. Earlier aspirin is used in non-thrombolysed ischemic stroke when appropriate, but the timing differs after lytic therapy.

35. C. Early recurrent risk after minor stroke or high-risk TIA is front-loaded, so short-term dual antiplatelet therapy can provide meaningful early protection. Extending aspirin plus clopidogrel long term increases hemorrhagic risk and generally does not add sufficient ischemic benefit. Long-term therapy is usually a single antiplatelet agent unless another indication exists.

36. A. A severe maternal stroke threatens both patient and pregnancy. Reperfusion decisions during pregnancy should be individualized with obstetric input, considering stroke severity, treatment benefit, radiation and medication exposure, and bleeding risk. Pregnancy alone should not create a blanket prohibition when the expected maternal benefit is substantial.

37. B. The 2026 acute ischemic stroke guideline includes pediatric recommendations and acknowledges potential benefit of endovascular therapy in selected children with appropriate anatomy and disabling

LVO. Evidence is less extensive than in adults, so experienced multidisciplinary evaluation is essential. Age alone does not make reperfusion impossible.

38. E. Thrombectomy treats an occluded artery, so vascular imaging must demonstrate a suitable arterial target. Core and collateral imaging influence selection, especially in later windows, but they do not substitute for identifying the occlusion itself. Chronic incidental findings do not create an endovascular indication.

39. C. Leptomeningeal collaterals provide alternative arterial supply around an occlusion and can preserve penumbral tissue, producing slower infarct growth in some patients. They do not prove recanalization and do not eliminate the need for reperfusion when a treatable occlusion remains. This explains why patients with similar onset times can have very different core volumes.

40. D. Perfusion-core mismatch is an imaging approximation of ischemic penumbra: tissue with reduced flow that has not yet crossed the threshold to irreversible infarction. A larger mismatch can support late-window reperfusion selection when used with validated criteria. It is not simply chronic white-matter disease or a direct measure of hemorrhage risk.

41. B. ASPECTS begins at 10 and subtracts points for early ischemic change in defined MCA regions. A score of 9 indicates limited visible early injury, though it does not prove the absence of ischemia or quantify all tissue at risk. Lower values generally reflect more extensive established injury.

42. A. Critical inflow stenosis and poor collateral circulation can make downstream perfusion pressure-dependent. A fall in systemic pressure may then reduce cerebral blood flow below the ischemic threshold and worsen deficits. This pattern supports hemodynamic compromise rather than a purely embolic or epileptic mechanism.

43. D. Limb-shaking TIAs are a distinctive manifestation of severe hemodynamic cerebrovascular insufficiency. Episodes are often precipitated by standing or exertion and may resemble focal seizures, but they reflect critically reduced perfusion reserve. Recognition matters because the underlying large-artery disease carries substantial stroke risk.

44. E. Orolingual angioedema is an uncommon but potentially dangerous complication of thrombolysis and is associated with bradykinin pathways; ACE-inhibitor use increases risk. It may be asymmetric and associated with insular involvement. Airway assessment and prompt treatment are essential because progression can be rapid.

45. D. Sudden headache, vomiting, hypertension, or neurologic deterioration during or shortly after thrombolysis raises concern for symptomatic intracranial hemorrhage. The infusion should be stopped if ongoing, urgent imaging obtained, and blood products or antifibrinolytic reversal strategies prepared as indicated. Empiric antithrombotic escalation is unsafe until hemorrhage is excluded.

46. B. Fever is associated with worse neurologic outcome and should prompt evaluation for infection, aspiration, urinary sources, drug reactions, or central fever. Antipyretic treatment and cause-specific

management are appropriate. Routine antibiotics without evidence of infection do not prevent post-stroke pneumonia and expose patients to unnecessary harm.

47. E. Dysphagia is common after stroke and increases aspiration and pneumonia risk. Patients who fail a swallow screen should remain NPO until a trained assessment identifies a safe diet or alternative route for medications and nutrition. Many patients improve, so immediate permanent gastrostomy is not routinely necessary.

48. B. Intermittent pneumatic compression reduces DVT risk in immobile stroke patients and is useful when pharmacologic prophylaxis is delayed or contraindicated. Graduated compression stockings alone have not shown the same benefit. Full-dose anticoagulation or routine IVC filter placement is not justified for prophylaxis without another indication.

49. B. Hemorrhagic transformation ranges from small petechiae without mass effect to symptomatic parenchymal hematoma. Its clinical significance and impact on antithrombotic timing vary accordingly. The decision should integrate symptoms, size, mass effect, embolic risk, and the reason antithrombotic therapy is needed rather than applying a universal rule.

50. E. Aphasia localizes the dominant hemisphere, while face and arm weakness greater than leg weakness strongly suggests the MCA distribution. Conjugate gaze toward the lesion is also typical of a large hemispheric MCA syndrome. ACA infarction would more often emphasize contralateral leg weakness and abulia.

51. E. ACA infarction preferentially affects the contralateral leg because the leg motor cortex lies on the medial hemisphere. Medial frontal injury can also produce abulia, impaired initiation, and urinary dysfunction. The absence of aphasia and relative sparing of face and arm support this localization.

52. D. The combination of nucleus ambiguus dysfunction, crossed sensory findings, ipsilateral Horner syndrome, and vestibulocerebellar symptoms is classic for lateral medullary syndrome. Vertebral artery or PICA pathology is the usual vascular substrate. Medial brainstem lesions would produce a different cranial nerve and long-tract pattern.

53. A. Medial medullary syndrome classically combines contralateral weakness, contralateral loss of dorsal-column modalities, and ipsilateral hypoglossal dysfunction. The lesion involves the pyramid, medial lemniscus, and hypoglossal fascicle or nucleus. Lateral medullary infarction instead causes dysphagia, vertigo, ataxia, and crossed pain-temperature loss.

54. D. Bilateral ventral pontine infarction can interrupt corticospinal and corticobulbar tracts while sparing the reticular activating system and vertical eye movements. The patient is conscious but cannot move or speak, communicating through preserved eye movements. Distinguishing this state from coma is essential for prognosis and communication.

55. E. The PCA supplies inferomedial temporal structures important for memory and the occipital visual cortex. A dominant PCA infarction can therefore combine memory dysfunction with a contralateral visual field deficit. ACA and MCA lesions would produce different cortical syndromes.

56. C. A small thalamic lacune in the ventral posterior sensory relay can produce pure hemisensory stroke without cortical signs. Absence of aphasia, neglect, visual field loss, or motor deficit supports a subcortical localization. Cortical sensory syndromes more often include neglect, extinction, astereognosis, or other higher-order findings.

57. E. Dysarthria-clumsy hand syndrome is a classic lacunar syndrome caused by a small deep infarct, often in the pons or internal capsule. The lack of cortical signs or a hemisensory syndrome supports a penetrating-artery lesion. Cortical and posterior fossa alternatives would produce additional localizing findings.

58. D. A small deep infarct producing a classic pure motor lacunar syndrome is most consistent with intrinsic penetrating-artery disease. Hypertension and diabetes contribute to lipohyalinosis and microatheroma. Embolic mechanisms more often produce cortical signs or multiple infarcts, although overlap is possible.

59. A. Acute infarcts in multiple anterior and posterior circulation territories suggest an embolic shower from a source upstream of those arterial beds, particularly the heart or aortic arch. Small-vessel disease typically produces single deep lesions, not synchronous territorial scatter. The diffusion pattern therefore helps prioritize etiologic investigation.

60. C. Paroxysmal AF can be intermittent and easily missed on a single ECG or short telemetry period. Longer external or implantable monitoring increases detection when no other cause is identified. Empiric anticoagulation without documented AF or another indication has not proved beneficial for unselected embolic stroke of undetermined source.

61. E. For most patients with nonvalvular AF who require stroke prevention, DOACs are preferred over warfarin because of favorable efficacy and bleeding profiles. Important exceptions include mechanical valves and moderate-to-severe rheumatic mitral stenosis. Antiplatelet therapy does not provide equivalent protection from AF-related embolism.

62. E. Mechanical heart valves remain an important exception to the usual preference for DOACs in atrial fibrillation. Vitamin K antagonist therapy with warfarin is required, using an INR target appropriate to valve type and patient factors. DOACs have not demonstrated acceptable safety and efficacy for mechanical valve thrombosis prevention.

63. D. A large anterior MI can produce apical akinesis and stasis, favoring left ventricular thrombus formation. A mobile thrombus can embolize into the systemic circulation and cause cerebral infarction. This mechanism is directly demonstrated by the echocardiographic finding and differs from atherosclerotic or small-vessel stroke.

64. E. PFO closure can reduce recurrence in selected younger patients with nonlacunar cryptogenic stroke after a thorough evaluation excludes other causes, especially when high-risk PFO anatomy is present. The presumed mechanism is paradoxical embolism through a right-to-left shunt. Closure is not appropriate when a more likely stroke mechanism has been identified.

65. B. PFOs are common in the general population and can be incidental. An older patient with conventional small-vessel risk factors and a classic lacunar infarct has a plausible alternative mechanism that lowers the likelihood that the PFO is causal. PFO closure trials focused on selected patients with nonlacunar cryptogenic stroke after exclusion of competing etiologies.

66. E. Aggressive lipid lowering is a core component of secondary prevention, especially in atherosclerotic stroke. High-intensity statins reduce major vascular events, and additional agents may be needed when LDL remains above target. The goal is durable vascular risk reduction rather than short-term treatment during hospitalization only.

67. B. Permissive hypertension is an acute-stroke concept, not a chronic secondary-prevention strategy. Long-term control of hypertension is among the most effective interventions for reducing recurrent stroke and other vascular events. Targets should be individualized, but persistent values in this range generally warrant better control.

68. C. Smoking increases vascular risk and cessation is an important component of secondary prevention. Successful behavior change is more likely with structured support, counseling, and appropriate pharmacotherapy than with brief advice alone. Risk reduction requires sustained cessation rather than merely reducing the number of cigarettes.

69. B. Symptomatic severe extracranial carotid stenosis is a high-risk, treatable cause of recurrent stroke. Carotid endarterectomy provides substantial benefit in appropriate surgical candidates and is generally performed relatively early once the patient is stable. Intensive medical management remains necessary before and after revascularization.

70. C. Choice between carotid endarterectomy and stenting depends on age, anatomy, comorbidity, and local expertise. Older age and complex arch anatomy can increase periprocedural embolic risk with transfemoral stenting, often favoring endarterectomy when surgical risk is acceptable. The decision is individualized rather than based on stenosis percentage alone.

71. E. Carotid revascularization modality is individualized. Prior radiation, restenosis after endarterectomy, or surgically inaccessible high lesions can make open exposure more difficult and may favor an endovascular option when procedural risks are acceptable. Patient age, arch anatomy, and local expertise still matter.

72. E. The absolute stroke risk of asymptomatic carotid stenosis has fallen with modern intensive medical therapy. Revascularization may still be considered in carefully selected patients with low

procedural risk and sufficient life expectancy, but it is not an automatic emergency. Shared decision-making should include the uncertain incremental benefit over contemporary medical care.

73. A. For severe symptomatic intracranial atherosclerotic stenosis, aggressive medical management is first-line. This includes intensive vascular risk-factor control and short-term dual antiplatelet therapy in appropriate patients. Routine early intracranial stenting produced excess periprocedural harm in trials and is not the preferred initial strategy.

74. D. Intracranial atherosclerosis is part of a systemic vascular disease process strongly influenced by hypertension, lipids, diabetes, smoking, and lifestyle. Aggressive medical management lowers recurrent risk even though it does not mechanically remove the stenosis overnight. The strategy combines risk-factor control with antithrombotic therapy and adherence support.

75. E. Trials of empiric anticoagulation for unselected ESUS did not reduce recurrent stroke enough to justify the bleeding risk. In the absence of documented AF, ventricular thrombus, venous-to-arterial shunt with another indication, or another anticoagulation target, antiplatelet therapy is used. Continued etiologic monitoring remains important.

76. B. Neck or head pain plus partial Horner syndrome is a classic clue to internal carotid dissection because sympathetic fibers run along the carotid wall. The injured artery can develop mural hematoma, stenosis, pseudoaneurysm, and thrombus that embolizes intracranially. CTA or MRA can demonstrate the characteristic arterial abnormality.

77. B. Most cervical artery dissections are managed medically with individualized antithrombotic therapy for a finite period, while recurrent events and arterial healing are monitored. Evidence does not establish a universal superiority of anticoagulation over antiplatelet therapy. Stenting is reserved for selected circumstances rather than routine use.

78. A. Dissection can generate thrombus and emboli over time rather than only at the instant of wall injury. Many ischemic events occur during the first days, and a substantial majority occur within the first week, although later events are possible. New focal symptoms after recent dissection therefore require urgent assessment.

79. C. Most cervical dissections do not reveal a monogenic disorder, but striking features of connective-tissue fragility or a strong family history should prompt targeted evaluation. Identifying an underlying arteriopathy can affect counseling, surveillance, and management of vascular risk. It should not be replaced by empiric immunosuppression.

80. A. Complex aortic arch atheroma, especially when thick or mobile, can serve as a source of artery-to-artery embolism into the cerebral vessels. The finding is particularly relevant when infarcts are embolic in pattern and no stronger source is identified. Risk-factor treatment and antithrombotic strategy are individualized.

81. B. A demonstrated intracardiac thrombus is a direct embolic source and does not require simultaneous documentation of atrial fibrillation to be clinically meaningful. It may arise in atrial cardiopathy, intermittent AF, or other conditions. The finding changes secondary prevention because anticoagulation is generally indicated when safe.

82. A. TEE offers superior visualization of several high-risk cardioembolic structures, including the left atrial appendage, atrial septum, valves, and proximal aorta. It is particularly useful when transthoracic imaging is nondiagnostic or when a structural source is suspected. Vascular ultrasound and brain imaging cannot directly characterize an intracardiac mass.

83. C. Transient monocular vision loss localizes anterior to the optic chiasm, most often to the retina or optic nerve. Ipsilateral carotid plaque can embolize through the ophthalmic artery or reduce perfusion, producing amaurosis fugax. Occipital ischemia causes homonymous, not monocular, field loss.

84. D. The contemporary tissue-based definition distinguishes TIA from ischemic stroke by the presence of infarction rather than symptom duration alone. If acute infarction is demonstrated, the event is an ischemic stroke even when symptoms resolve rapidly. A true TIA is transient neurologic dysfunction caused by focal ischemia without acute infarction.

85. A. TIA can precede disabling stroke, with risk especially front-loaded in the first hours and days. Evaluation should rapidly identify infarction, cervicocephalic stenosis, atrial fibrillation, and other treatable causes while secondary prevention begins. Symptom resolution is not a reason to defer urgent workup.

86. E. In selected patients with early-arriving minor stroke or high-risk TIA, short-term aspirin plus clopidogrel reduces early recurrent ischemic events. The benefit is time-limited, and long-term dual therapy increases bleeding. The regimen is not a substitute for anticoagulation when a high-risk cardioembolic source such as AF is identified.

87. C. For noncardioembolic lacunar stroke, long-term single antiplatelet therapy and risk-factor control are standard. Chronic dual antiplatelet therapy increases bleeding without providing enough additional prevention benefit. Anticoagulation is reserved for a separate indication such as atrial fibrillation.

88. C. A recurrent stroke while prescribed aspirin is not a diagnosis. The event may reflect nonadherence, a new cardioembolic source, large-artery disease, small-vessel disease, or another mechanism that calls for different prevention. Mechanism-directed evaluation is more useful than reflexively adding medications that increase bleeding risk.

89. A. Quality improvement uses measured gaps to redesign processes and then reassess performance. A standardized order set, referral pathway, ownership assignment, and follow-up tracking can address missed monitoring more reliably than education alone. The intervention should remain clinically targeted rather than inflating the metric through indiscriminate testing.

90. D. Physical activity is an important component of secondary prevention and recovery. The program should be individualized to neurologic deficits, fall risk, and cardiopulmonary status, often with rehabilitation support. Prolonged sedentary behavior is undesirable, but unsupervised maximal exertion is not the goal.

91. B. Medication adherence is a major determinant of secondary prevention and is often shaped by regimen complexity, cost, routines, beliefs, and support. Collaborative simplification and practical reminder strategies address the actual barrier more effectively than blame or adding complexity. Effective counseling is patient-centered and specific.

92. C. Aggressive medical management for intracranial atherosclerosis depends on actually achieving risk-factor control and adherence. Persistent smoking and uncontrolled hypertension represent modifiable treatment gaps, not proof that guideline-directed medical therapy has failed. Stenting is not a routine shortcut around unaddressed preventive factors.

93. E. Dual antiplatelet therapy has a defined short-term role in selected minor stroke, high-risk TIA, and severe symptomatic intracranial stenosis. For most other secondary-prevention situations, long-term dual therapy adds bleeding risk without enough ischemic benefit. A single antiplatelet agent is usually preferred after the short high-risk interval.

94. B. Anticoagulation is the key preventive therapy for AF-related embolism. In the absence of another clear antiplatelet indication, adding aspirin usually increases bleeding without meaningfully reducing recurrent stroke. Combination therapy should therefore be reserved for specific cardiovascular indications and used for the shortest appropriate duration.

95. D. Anticoagulation timing after AF-related stroke is individualized. Small stable infarcts often permit earlier resumption, while large infarcts, mass effect, or hemorrhagic transformation may justify delay. The decision weighs the competing hazards of recurrent embolism and intracranial bleeding rather than following a single interval for every patient.

96. A. Although symptomatic carotid stenosis is generally treated relatively early, timing must account for infarct size, neurologic stability, edema, and hemorrhagic transformation. A large unstable infarct increases perioperative risks, including hyperperfusion and hemorrhage. The intervention should be delayed until the brain and patient are sufficiently stable.

97. D. Hyperperfusion syndrome can follow carotid revascularization when chronically impaired autoregulation is exposed to a sudden increase in perfusion pressure. Headache, seizures, focal deficits, edema, and intracerebral hemorrhage can occur. Careful postoperative blood-pressure management helps reduce risk.

98. B. Positional or pressure-related focal symptoms in the setting of severe inflow disease and impaired reserve suggest hemodynamic ischemia. The downstream territory is dependent on systemic pressure

and collateral flow. Embolic events generally are not reproducibly triggered by standing or modest blood-pressure changes.

99. B. Markers of atrial cardiopathy can suggest an atrial substrate associated with embolic risk and may justify closer rhythm monitoring. However, treatment should not leap from an association to automatic anticoagulation without a supported indication. The distinction between risk marker and actionable mechanism is important in cryptogenic stroke.

100. E. Atrial myxoma classically arises from the interatrial septum, often near the fossa ovalis, and can be mobile enough to prolapse toward the mitral valve. Tumor fragments or associated thrombus can embolize systemically and cause stroke. Surgical removal is usually required after stabilization and appropriate evaluation.

101. A. The systemic infectious findings plus a new murmur and multifocal embolic infarcts strongly support infective endocarditis with septic emboli. These strokes may be complicated by hemorrhage, mycotic aneurysm, or abscess. Management focuses on antimicrobial therapy and valve-related decisions; reperfusion choices require special caution because bleeding risk differs from routine cardioembolism.

102. E. Infective endocarditis is associated with septic arteritis, hemorrhagic conversion, and mycotic aneurysms, making IV thrombolysis unusually hazardous. Mechanical thrombectomy may be considered in selected LVO cases at experienced centers, but treatment must be individualized. Standard lytic pathways should not be applied without accounting for the infectious vascular pathology.

103. A. Infective endocarditis can cause infectious intracranial aneurysms through septic embolic injury of the arterial wall. Rupture may present with sudden severe headache and intracranial hemorrhage. New hemorrhagic symptoms in this setting require urgent vascular imaging and multidisciplinary management.

104. C. Not every cardiac abnormality found during stroke workup is causal. High-risk sources include atrial fibrillation, intracardiac thrombus, mechanical valves under certain conditions, vegetation, or tumor. Calcific valvular disease without a demonstrated embolic mechanism should be interpreted in the context of the entire etiologic evaluation.

105. C. Severe LV systolic dysfunction increases vascular risk, but in sinus rhythm without intracardiac thrombus it does not by itself create a universal anticoagulation indication. Therapy should target the identified stroke mechanism and follow heart-failure guideline-directed treatment. Demonstrated thrombus or atrial fibrillation would change the antithrombotic strategy.

106. A. Prolonged rhythm monitoring can convert an undetermined stroke into a mechanism-directed diagnosis. Clinically meaningful AF changes secondary prevention because oral anticoagulation is much more effective than antiplatelet therapy for preventing AF-related embolism. Combination therapy is not routinely required unless another indication exists.

107. A. Stroke mechanism assignment often requires synthesis rather than a single test result. Infarct distribution, lesion morphology, anatomic plausibility, timing, and competing sources all contribute to causal probability. Overcalling an incidental lesion can lead to inappropriate prevention, while undercalling a treatable source can miss an opportunity to reduce recurrence.

108. E. Active adenocarcinoma can create a strong systemic prothrombotic state and is associated with multifocal arterial embolism and sterile valvular vegetations. Multiple-territory infarcts and markedly elevated coagulation markers heighten suspicion. The finding should prompt mechanism-specific evaluation rather than assuming conventional atherosclerosis.

109. A. For many patients with mild-to-moderate spontaneous ICH and presenting systolic pressure in the 150-220 mm Hg range, early smooth lowering toward 140 mm Hg is reasonable. Very aggressive reduction below about 130 mm Hg may be harmful, and large fluctuations are undesirable. The goal is controlled reduction that limits expansion without causing hypoperfusion.

110. D. Warfarin-associated ICH requires immediate reversal. Four-factor PCC rapidly restores vitamin K-dependent clotting factors, while intravenous vitamin K provides sustained correction as the PCC effect wanes. Idarucizumab reverses dabigatran, and protamine is used for heparin rather than warfarin.

111. A. Idarucizumab is the specific reversal agent for dabigatran and can rapidly neutralize its anticoagulant effect. Andexanet targets factor Xa inhibitors, while vitamin K reverses warfarin and protamine reverses heparin. Reversal should occur promptly in life-threatening intracranial bleeding.

112. E. Andexanet alfa is a specific reversal agent for factor Xa inhibitors such as apixaban and rivaroxaban. Four-factor PCC may also be used in some settings when a specific antidote is unavailable or unsuitable, but it is not a drug-specific binder. Idarucizumab is specific for dabigatran.

113. D. Routine platelet transfusion in spontaneous ICH associated with antiplatelet use can be harmful when there is no emergency surgical indication or severe thrombocytopenia. Management instead focuses on blood pressure, reversal of true anticoagulants when present, and neurosurgical assessment when indicated. Platelet strategy can differ when urgent surgery is planned.

114. D. Seizures should be treated when they occur, and continuous EEG is useful when mental status is unexplained or fluctuating. Routine prophylactic antiseizure medication has not improved long-term seizure control or functional outcome in patients without evidence of seizures. Unnecessary sedating drugs can also obscure the neurologic examination.

115. A. Fluctuating consciousness and subtle motor phenomena after ICH raise concern for electrographic seizures, which may be missed on a brief examination. Continuous EEG is the appropriate tool to capture intermittent or nonconvulsive events. Detecting seizures matters because they should be treated, unlike routine prophylaxis in seizure-free patients.

116. D. Intermittent pneumatic compression is recommended early for immobile ICH patients. Graduated compression stockings alone are ineffective as sole prophylaxis. Low-dose heparin

prophylaxis may be considered after hematoma stability, often 24-48 hours after onset, but full-dose anticoagulation is not used simply for immobility.

117. C. Once the hematoma is stable, low-dose pharmacologic VTE prophylaxis can be considered, commonly after 24-48 hours in appropriate patients. The dosing is prophylactic, not therapeutic, and is combined with mechanical measures when indicated. Timing is individualized to bleeding stability and thrombotic risk.

118. A. Corticosteroids do not improve outcome in spontaneous ICH and are not recommended for routine treatment of perihematomal edema or elevated intracranial pressure. Acute ICP management may involve head positioning, osmotic agents for clinical deterioration, CSF diversion, or surgery depending on the situation. Steroids are appropriate for some tumors and inflammatory conditions, not primary ICH edema.

119. C. Hyperosmolar agents are used as targeted therapy for elevated intracranial pressure or impending herniation, not as routine prophylaxis in stable ICH. Scheduled treatment without a clinical indication has not improved outcomes and can cause electrolyte, renal, and volume complications. Therapy should respond to physiologic need.

120. E. Cerebellar ICH with neurologic deterioration, brainstem compression, hydrocephalus, or a sufficiently large volume warrants urgent surgical evacuation to reduce mortality. Ventricular drainage may be needed, but treating hydrocephalus alone may not address the posterior fossa mass. Lumbar puncture is dangerous when a posterior fossa lesion is causing pressure gradients.

121. A. The posterior fossa has limited space, so relatively modest hematoma volumes can become dangerous. Contemporary ICH guidance includes cerebellar volume greater than about 15 mL among indications supporting immediate evacuation, alongside deterioration, brainstem compression, and hydrocephalus. Clinical context and neurosurgical expertise remain essential.

122. E. Trials of minimally invasive approaches have shown mortality benefit in selected supratentorial ICH populations, while consistent improvement in functional independence has been harder to demonstrate. Surgery is therefore not a universal requirement for every lobar hemorrhage. Location, volume, neurologic status, timing, and local expertise shape selection.

123. B. CAA is characterized by beta-amyloid deposition in cortical and leptomeningeal arterioles, predisposing older adults to lobar ICH, strictly lobar microbleeds, and cortical superficial siderosis. Hypertensive arteriopathy more often affects deep perforating territories such as basal ganglia, thalamus, pons, and cerebellum. MRI distribution is therefore etiologically informative.

124. A. CAA can cause transient focal neurologic episodes, sometimes called amyloid spells, associated with cortical superficial siderosis or convexity subarachnoid blood. Symptoms may spread over minutes and can mimic TIA, migraine aura, or focal seizures. Correct recognition matters because the hemorrhagic substrate affects antithrombotic decisions.

125. C. Hypertension-related arteriolosclerosis affects deep perforating vessels and commonly causes ICH or microbleeds in the basal ganglia, thalamus, pons, and cerebellum. This distribution contrasts with the strictly lobar pattern typical of CAA. The imaging pattern can therefore support the likely hemorrhage mechanism.

126. E. An AVM consists of abnormal arteries connected to veins through a nidus without a normal capillary bed, producing early venous drainage on angiography. This high-flow architecture can rupture and cause intracranial hemorrhage. Cavernous malformations are low-flow lesions and are typically angiographically occult.

127. A. DSA remains the reference standard when detailed characterization of an intracranial vascular lesion is required, particularly when noninvasive imaging is negative but suspicion remains high. It can show small feeding arteries, the nidus or fistulous point, venous drainage, and flow dynamics. The decision should still consider procedural risk and pretest probability.

128. A. Severity scores summarize population-level risk and can support communication, triage, and research, but they are not deterministic for an individual. Early self-fulfilling withdrawal of treatment can bias outcomes. Decisions about life-sustaining therapy should integrate the patient's values, neurologic trajectory, comorbidities, and response to treatment.

129. E. A DNAR order is specific to attempted resuscitation after cardiopulmonary arrest unless broader limitations are explicitly documented. It should not be interpreted as a blanket decision against surgery, ventilation, antibiotics, nutrition, rehabilitation, or other appropriate care. Treatment decisions should reflect the patient's known values and goals through shared decision-making.

130. C. Early multidisciplinary rehabilitation planning is important after ICH, and selected activities may begin after the initial stabilization period. However, very early aggressive mobilization within the first 24 hours can worsen outcomes. Routine fluoxetine has not improved functional recovery and can increase adverse events such as fractures.

131. A. ICH management emphasizes not only the target pressure but also the manner in which it is achieved. Smooth, sustained control with minimal variability is associated with better physiologic stability and may reduce hematoma expansion. Protocols should therefore avoid repeated overshoot and rebound from poorly titrated treatment.

132. B. Rebleeding is a major early cause of death after aneurysmal SAH. Rapid vascular imaging and definitive aneurysm occlusion, ideally within 24 hours, are priorities alongside airway, blood pressure, hydrocephalus, and other critical-care needs. Prophylactic hypervolemia is not recommended.

133. E. CT sensitivity for SAH is highest early and depends on timing, scanner quality, and interpretation. When suspicion remains substantial after a negative scan, additional testing is warranted rather than simply closing the diagnosis. The exact pathway can include lumbar puncture and/or vascular imaging depending on the clinical setting.

134. A. Early enteral nimodipine improves functional outcome after aneurysmal SAH and is standard therapy. Routine statins and intravenous magnesium are not recommended solely for vasospasm prevention. Hemodynamic augmentation is used for symptomatic delayed cerebral ischemia rather than prophylactically in every patient.

135. B. Acute hydrocephalus is common after SAH because blood obstructs CSF pathways and impairs absorption. Declining consciousness with ventricular enlargement warrants urgent CSF diversion, typically with an external ventricular drain. Aneurysm status and drain management require coordinated neurocritical and neurosurgical care.

136. A. Delayed cerebral ischemia typically occurs several days after SAH and is an important cause of secondary neurologic injury. New focal deficits or reduced consciousness require evaluation for DCI while also excluding rebleeding, hydrocephalus, seizures, metabolic problems, and infection. Vasospasm is an important contributor but DCI is a broader clinical syndrome.

137. A. For symptomatic DCI, induced hypertension can improve perfusion through narrowed vessels or collateral pathways in selected patients. Euvolemia should be maintained; prophylactic hypervolemia is not beneficial and can cause cardiopulmonary harm. Hemodynamic augmentation is a treatment for symptoms, not a routine preventive intervention.

138. D. Modern SAH care targets euvolemia rather than routine hypervolemia. Prophylactic volume expansion can cause pulmonary edema, cardiac stress, and other complications without improving functional outcomes. Hemodynamic augmentation is reserved for clinically significant delayed cerebral ischemia in appropriate patients.

139. E. When the examination is limited, multimodal monitoring can provide indirect evidence of vasospasm or threatened cerebral perfusion. TCD, CTA, and CTP are commonly used, while cEEG or invasive monitors may add information in selected high-grade cases. No single test replaces clinical assessment, and findings must be interpreted in context.

140. D. Vasospasm narrows the arterial lumen and can increase measured Doppler flow velocity. Interpretation also considers the extracranial-to-intracranial velocity relationship and the clinical examination, because hyperemia can also increase velocity. TCD is therefore a useful surveillance tool rather than an isolated diagnostic verdict.

141. D. New-onset seizures after aneurysmal SAH should be treated, and a short treatment course is often appropriate in the acute period. Routine prophylaxis for all patients is not recommended, though it may be considered in selected high-risk cases. Phenytoin is generally avoided because it has been associated with excess morbidity.

142. A. Routine seizure prophylaxis is not recommended for all SAH patients. It may be considered in higher-risk situations such as ruptured MCA aneurysm, high-grade SAH, hydrocephalus,

intraparenchymal hemorrhage, or cortical infarction. Treatment is clearly indicated when clinical or electrographic seizures actually occur.

143. C. Hyponatremia after SAH can reflect natriuresis and volume depletion, and hypovolemia may worsen cerebral perfusion. Management should correct the electrolyte disturbance while maintaining euvolemia rather than reflexively imposing fluid restriction. Volume status, urine studies, renal function, and ongoing losses help guide therapy.

144. D. Severe SAH can trigger a catecholamine surge that causes transient myocardial injury, arrhythmias, ECG changes, and stress cardiomyopathy-like ventricular dysfunction. This can complicate blood-pressure and volume management during the vasospasm window. Coronary disease should still be considered when clinically appropriate.

145. E. Post-treatment imaging is important when a residual aneurysm or recurrence is possible, particularly after endovascular therapy. Surveillance can guide retreatment decisions and identify growth of other aneurysms in selected patients. Long-term risk-factor counseling remains relevant because aneurysm treatment does not eliminate systemic vascular risk.

146. B. A strong family history, particularly two or more affected first-degree relatives, increases the prevalence of intracranial aneurysms and can support screening discussion. Noninvasive MRA or CTA is generally preferred for screening rather than routine invasive angiography. The interval and need for repeat imaging depend on risk factors and prior results.

147. D. Postpartum hypercoagulability, headache, papilledema, and seizures are classic clues to CVT. CTV and MRV are the main confirmatory vascular imaging tests. Lumbar puncture may show elevated pressure but cannot define the thrombosed venous anatomy and should not delay appropriate imaging.

148. A. Anticoagulation is the cornerstone of acute CVT treatment even when hemorrhagic venous infarction is present, because the hemorrhage results from venous outflow obstruction and anticoagulation prevents thrombus propagation. Parenteral heparin is commonly used initially. Surgery or endovascular rescue is reserved for selected severe cases.

149. B. LMWH is generally preferred for treatment of venous thromboembolism during pregnancy because it does not cross the placenta and has extensive obstetric experience. Warfarin is teratogenic during pregnancy, and DOAC safety data are insufficient for routine use. The maternal danger of untreated CVT is substantial.

150. D. Severe CVT can cause large hemorrhagic venous infarction, edema, and herniation. Decompressive surgery can be lifesaving in appropriately selected deteriorating patients. Anticoagulation addresses the underlying thrombosis, while surgery addresses the immediate mass effect; these therapies are complementary rather than mutually exclusive.

151. D. Most CVT is treated with anticoagulation. Endovascular thrombectomy or local thrombolytic approaches are reserved for selected patients with severe clinical deterioration despite appropriate

medical therapy, especially when the alternative is catastrophic progression. Evidence does not support routine first-line intervention for all CVT.

152. B. RCVS typically presents with recurrent thunderclap headaches and multifocal segmental vasoconstriction that reverses over time. Triggers can include postpartum state and vasoactive medications or drugs. Ischemic stroke, convexity SAH, and intracerebral hemorrhage can complicate the syndrome.

153. C. RCVS management centers on eliminating vasoactive triggers, controlling symptoms, and avoiding treatments that may worsen the syndrome. Glucocorticoids are not beneficial and may be associated with worse outcomes. Calcium-channel blockers are often used for headache, but the key mechanism is reversible dysregulation of vascular tone rather than inflammatory vasculitis.

154. A. PRES presents with headache, seizures, encephalopathy, and visual symptoms, commonly in the setting of severe hypertension, eclampsia, renal failure, or immunosuppressive therapy. MRI typically shows vasogenic edema, often posterior predominant. Treatment addresses blood pressure and the underlying trigger while controlling seizures.

155. B. PRES usually reflects failure of autoregulation and endothelial dysfunction with vasogenic edema. Severe hypertension should be lowered in a controlled manner to avoid hypoperfusion, while seizures and precipitating conditions are treated. The syndrome is not an indication for thrombolysis or empiric anticoagulation.

156. C. Moyamoya is a progressive steno-occlusive arteriopathy centered on the terminal internal carotid arteries and proximal circle of Willis, with development of fragile basal collateral networks. Patients can present with ischemic or hemorrhagic stroke. Diagnosis integrates characteristic vascular imaging and exclusion of secondary moyamoya syndromes.

157. D. Moyamoya causes progressive intracranial steno-occlusion with impaired cerebral perfusion, and surgical revascularization is the principal disease-directed treatment for symptomatic patients. Bypass creates new routes for cerebral blood flow. Routine angioplasty is not durable for the characteristic diffuse arteriopathy, and the disorder is not treated as inflammatory vasculitis.

158. E. Fibromuscular dysplasia is a nonatherosclerotic, noninflammatory arteriopathy that commonly affects renal and cervicocephalic arteries. Medial fibroplasia produces the classic string-of-beads appearance, and affected patients have increased risk of dissection and aneurysm. Recognition should prompt evaluation of other vascular beds when appropriate.

159. E. PACNS can produce subacute headache, encephalopathy, seizures, and multifocal ischemic lesions with inflammatory CSF and irregular vascular narrowing. RCVS more often has recurrent thunderclap headache and usually normal or near-normal CSF. Diagnosis may require vessel-wall imaging, angiography, or biopsy after careful exclusion of mimics.

160. B. RCVS and PACNS can both show arterial narrowing, but the clinical tempo differs. Recurrent thunderclap headache, common vasoactive triggers, and normal or near-normal CSF strongly favor RCVS, whereas PACNS more often has subacute progressive symptoms and inflammatory CSF. Correct distinction matters because glucocorticoids may worsen RCVS.

161. B. In older adults, atraumatic convexity SAH can occur with CAA and may coexist with cortical superficial siderosis and transient focal neurologic episodes. This pattern differs from aneurysmal SAH, which typically involves basal cisterns. Other causes of convexity SAH include RCVS, CVT, and PRES depending on age and context.

162. E. RCVS can produce both ischemic and hemorrhagic complications, including small convexity SAH early in the course. Recurrent thunderclap headache plus multifocal reversible vasoconstriction is the key pattern. Age and clinical tempo make CAA and PACNS less likely in this scenario.

163. E. Acute stroke in sickle cell disease requires rapid multidisciplinary management, and transfusion is a key disease-specific intervention to reduce HbS burden and improve oxygen-carrying capacity. Exchange transfusion is often preferred when feasible, especially in severe presentations. Long-term prevention may include chronic transfusion and treatment of associated vasculopathy.

164. C. Transcranial Doppler screening transformed primary stroke prevention in children with sickle cell disease. Abnormally high velocities identify a high-risk cerebrovascular state, and chronic transfusion reduces first-stroke risk. The test is a risk-stratification tool rather than proof of an existing acute infarction.

165. D. CADASIL is an autosomal dominant small-vessel arteriopathy caused by NOTCH3 variants. Migraine with aura, subcortical ischemic events, mood or cognitive symptoms, and characteristic anterior temporal and external capsule lesions are important clues. Family history can be absent in de novo cases but is often informative.

166. E. Fabry disease is an X-linked lysosomal storage disorder caused by deficient alpha-galactosidase A activity and glycosphingolipid accumulation. Neuropathic pain, angiokeratomas, renal disease, hypertrophic cardiomyopathy, and premature stroke are classic manifestations. Diagnosis can lead to disease-specific therapy and family screening.

167. D. COL4A1 and COL4A2 encode type IV collagen in vascular basement membranes. Pathogenic variants can cause a spectrum including small-vessel ischemia, microbleeds, intracerebral hemorrhage, porencephaly, ocular findings, and systemic manifestations. A multigenerational pattern with congenital or early-onset vascular disease is an important clue.

168. C. Remote radiation can injure large and small vessels, accelerating fibrosis, endothelial dysfunction, and atherosclerotic stenosis years later. The distribution often corresponds to the irradiated field. Secondary prevention and revascularization decisions can be complicated by hostile tissue planes and diffuse vascular disease.

169. B. Antiphospholipid syndrome is an acquired autoimmune thrombophilia associated with arterial thrombosis, venous thrombosis, and pregnancy morbidity. Stroke may occur in young adults without conventional atherosclerotic risk factors. Management depends on the thrombotic history and antibody profile and often requires long-term anticoagulation for arterial events.

170. E. High-risk thrombotic APS, particularly with arterial events or triple-positive antibodies, is generally treated with a vitamin K antagonist rather than a DOAC because recurrent arterial thrombosis has been a concern with DOACs. Management should involve hematology or rheumatology when appropriate and incorporate bleeding risk and recurrent-event history.

171. D. Pregnancy and the postpartum period are hypercoagulable states, and thrombotic risk remains elevated after delivery. CVT, arterial ischemic stroke, RCVS, hypertensive disorders, and cardiomyopathy are all important considerations in postpartum neurologic symptoms. The differential should be driven by the clinical syndrome rather than assuming a single mechanism.

172. C. Smoking and estrogen-containing contraceptives are modifiable vascular risk factors, and together they can increase thrombotic risk more than either exposure alone. Secondary prevention should address avoidable prothrombotic factors while offering safe contraceptive alternatives. Counseling should be individualized and nonjudgmental.

173. B. Depression is common after stroke and can worsen quality of life, adherence, social participation, and rehabilitation engagement. Screening and treatment with psychotherapy, medication, or both can improve patient-centered outcomes. Management should also assess suicidality, cognition, caregiver stress, and social support.

174. D. Discharge planning should be multidisciplinary and patient-centered. The team should explain concrete risks, assess caregiver capacity and home environment, and discuss rehabilitation options while respecting the patient's values and decision-making authority. Shared decision-making does not mean ignoring preventable safety hazards or excluding the patient and family from choices.

175. B. When a patient lacks capacity, surrogates and clinicians should use the patient's known values and prior expressed preferences to guide decisions. The directive supports a time-limited trial of treatment rather than automatic withdrawal or indefinite escalation. Prognosis should be updated as the clinical course clarifies the likelihood of returning to an acceptable level of function.

176. E. Meaningful informed consent requires communication the patient can understand. A qualified medical interpreter reduces errors and preserves confidentiality, while clinicians should address the patient directly rather than shifting the conversation to family. Family members can participate when the patient wants them involved, but they should not replace professional interpretation for complex consent.

177. D. Decision-making capacity includes understanding, appreciation, reasoning, and communicating a choice; it does not require agreeing with the clinician. A capacitated patient may refuse even beneficial

treatment. The clinician should ensure the refusal is informed, document it, and continue other care rather than coercing or abandoning the patient.

178. C. Near misses are valuable safety signals because they reveal vulnerabilities before harm occurs. The immediate error should be corrected, and reporting should support a systems analysis of dose defaults, order-entry design, workflow, and verification. A just-culture approach focuses on preventing recurrence rather than concealing the event or assigning reflexive blame.

179. C. Quality improvement requires a measure linked to the process being changed. Median door-to-imaging time directly tests the operational aim, while balancing measures can detect unintended harms such as inappropriate imaging or workflow disruption. Dissemination is an implementation step, not proof of effectiveness.

180. D. Sensitivity is the proportion of affected patients who test positive; a highly sensitive test therefore misses relatively few true cases. A negative result can substantially reduce disease probability, particularly when the test is applied appropriately. Predictive value still depends on pretest probability and specificity.

181. B. Specificity is the true-negative fraction among people who do not have the condition. It is not the same as positive predictive value, which varies with prevalence and pretest probability. Sensitivity, not specificity, describes the true-positive fraction among affected patients.

182. A. For a relative risk, the null value is 1.0. The point estimate of 0.80 is compatible with a 20% relative reduction, but the interval crossing 1.0 means the data are also compatible with no difference at the conventional two-sided 0.05 threshold. Clinical importance and precision should still be considered separately from statistical significance.

183. A. Heterogeneity can reflect real differences in populations, interventions, comparators, outcomes, bias, or chance. The pooled estimate is less informative if clinically dissimilar studies are combined without understanding those differences. Subgroup or sensitivity analyses may help, but they should be interpreted cautiously and preferably be prespecified.

184. B. MRI safety depends on the specific device, components, implantation status, field strength, and required scanning conditions. Device records, radiographs, manufacturer information, or specialty consultation may be needed before proceeding. Guessing can expose the patient to heating, displacement, malfunction, or inappropriate device programming.

185. A. High-stakes diagnostic decisions require weighing competing risks rather than applying blanket exclusions. The team should consider kidney function, contrast type and dose, alternative imaging, hydration when appropriate, and the consequence of missing or delaying the diagnosis. In an emergency, the neurologic benefit of timely vascular imaging may outweigh a manageable renal risk.

186. A. Capacity is decision-specific and depends on the ability to understand information, appreciate consequences, reason about options, and communicate a choice. Delirium, intoxication, pain, language

barriers, and other reversible factors should be addressed when possible. Temporary restriction may be justified in a true emergency when capacity is absent, but it requires a careful basis.

187. E. Neurologic prognosis often evolves over time. Clear communication should distinguish population-level estimates from individual certainty, name important uncertainties, and identify what future information may change expectations. This supports shared decision-making without offering false precision or avoiding difficult conversations.

188. A. When a patient lacks capacity, no surrogate is available, and delay would create serious imminent harm, clinicians may provide necessary emergency treatment under implied consent. The exception is limited to the emergency and does not erase the obligation to seek a surrogate or formal decision-making process for later nonurgent decisions.

189. B. Critical and unexpected results require reliable closed-loop communication. The system should identify who is responsible, confirm receipt, contact the patient when appropriate, and track completion of the next step. Passive posting in the chart can fail when multiple teams assume someone else will act.

190. E. Interhospital transfer depends on timely access to clinical information and imaging. Reliable electronic image exchange, prearrival review, and defined escalation pathways reduce redundant scans and treatment delay. A systems solution is preferable to repeatedly exposing patients to avoidable delays and duplicate testing.

191. B. Diagnostic testing is most useful when the result can alter probability, management, prognosis, or counseling. Low-value testing can generate incidental findings, cascades, cost, anxiety, and procedural risk without improving outcomes. Thoughtful test selection is part of patient safety and resource stewardship.

192. E. Neurologic interventions often trade survival, disability, procedural risk, and uncertain recovery. Baseline function and patient values therefore belong alongside anatomy and disease severity in decision-making. Evidence informs options, but patient-centered care requires understanding which outcomes the patient considers acceptable.

193. A. Migraine aura typically consists of positive neurologic symptoms that build or migrate over minutes, often followed by a characteristic migraine headache. TIA more often causes abrupt negative symptoms such as field loss, while occipital seizures are usually briefer and more rapidly evolving. The long stereotyped history and normal interictal examination also support migraine.

194. E. Giant cell arteritis can cause irreversible visual loss through ischemic optic neuropathy or retinal ischemia. When clinical suspicion is high, glucocorticoids should begin immediately and diagnostic confirmation should follow; treatment should not wait for biopsy. Age, jaw claudication, inflammatory markers, and new headache strongly distinguish this from migraine.

195. A. Cluster headache causes severe strictly unilateral orbital or temporal pain with ipsilateral cranial autonomic features and restlessness, often in circadian clusters. Individual attacks usually last 15-180 minutes. Migraine tends to last longer and patients often prefer stillness rather than pacing.

196. D. A stereotyped Jacksonian march followed by transient focal weakness is characteristic of a focal motor seizure with Todd paresis. Limb-shaking TIAs are more often precipitated by standing or exertion in severe occlusive disease and usually lack a classic march and postictal pattern. EEG and vascular evaluation can be tailored to the context.

197. C. Nonconvulsive seizures may have little or no visible motor activity, especially in critically ill patients. Continuous EEG is the appropriate diagnostic tool when unexplained depressed or fluctuating consciousness raises concern. A short routine EEG can miss intermittent events, making longer monitoring useful in selected patients.

198. E. A remote cortical lesion, epileptiform EEG abnormalities, or other evidence of an enduring predisposition can raise recurrence risk after a first unprovoked seizure. A prior cortical infarct is a common structural substrate in older adults. Treatment decisions balance estimated recurrence risk, medication adverse effects, occupation, driving, and patient preference.

199. B. OSA causes repetitive upper-airway obstruction, intermittent hypoxemia, sleep fragmentation, and sympathetic activation. It is common after stroke and is associated with hypertension and vascular risk. Diagnostic testing is usually performed with polysomnography or an appropriate home sleep apnea test depending on patient factors.

200. E. Home sleep apnea testing is useful in selected uncomplicated patients, but attended polysomnography provides broader physiologic monitoring when major comorbidities, hypoventilation, central apnea, or complex neurologic disease are concerns. Testing helps distinguish event type and severity and supports appropriate therapy.

201. D. Positive airway pressure is the standard treatment for many patients with clinically significant OSA and can improve sleepiness and breathing. Weight management and other behavioral measures are also important when relevant. Supplemental oxygen alone does not correct upper-airway obstruction or sleep fragmentation.

202. A. HSV encephalitis often affects the temporal and inferior frontal lobes and can produce fever, encephalopathy, aphasia, seizures, and hemorrhagic necrosis. Intravenous acyclovir should be started promptly when suspicion is substantial because delay worsens outcome. CSF PCR is highly useful, but treatment should not wait for the result.

203. A. Meningitis can injure cerebral arteries through inflammatory vasculopathy, thrombosis, or vasospasm, leading to ischemic stroke. Infarcts may be multifocal and can complicate severe bacterial or tuberculous meningitis. Treatment must address the infection while managing neurologic complications and raised intracranial pressure when present.

204. C. PML is caused by JC-virus reactivation in immunosuppressed patients and produces progressive focal deficits with multifocal demyelinating white-matter lesions, often without major mass effect. Immune restoration is the central treatment strategy in HIV-associated PML. The tempo and imaging differ from acute vascular infarction.

205. A. Subdural hematoma results from bleeding into the space between dura and arachnoid, classically from torn bridging veins. On CT it is crescentic and can cross sutures, unlike a biconvex epidural hematoma. Older age, anticoagulation, and cerebral atrophy increase risk even after seemingly minor trauma.

206. C. Rapidly progressive spinal cord compression can cause irreversible neurologic injury and requires urgent diagnosis and cause-specific treatment, often surgical decompression. The exact therapy depends on whether the lesion is hematoma, abscess, tumor, or another process. Lumbar puncture and cerebral thrombolysis do not relieve a cervical compressive lesion.

207. B. Anterior spinal artery syndrome affects the anterior two-thirds of the cord, including corticospinal and spinothalamic pathways, while often sparing posterior column modalities. Sudden pain and weakness can occur with aortic disease, surgery, hypotension, or embolic causes. The arterial anatomy explains the dissociated sensory pattern.

208. D. CRAO causes acute retinal ischemia with profound monocular vision loss, a relative afferent pupillary defect, retinal whitening, and a cherry-red fovea. It is an ocular vascular emergency and should trigger urgent stroke-like evaluation for embolic sources and giant cell arteritis when clinically appropriate.

209. E. The abducens nerve innervates the lateral rectus, so an isolated sixth nerve palsy causes impaired abduction and horizontal diplopia worse when looking toward the affected side. Causes include microvascular ischemia, raised intracranial pressure, brainstem lesions, and compressive processes. The ocular motor pattern localizes the lesion before etiology is considered.

210. B. Posterior-canal BPPV produces brief position-triggered vertigo with characteristic torsional-upbeating nystagmus on Dix-Hallpike testing, often with latency and fatigability. Persistent spontaneous vertigo, severe gait ataxia, focal deficits, or central nystagmus features should raise concern for a posterior circulation process instead.

211. B. In the acute vestibular syndrome, a normal head impulse, direction-changing gaze-evoked nystagmus, or skew deviation points toward a central lesion. When performed by trained clinicians in the right clinical syndrome, HINTS can be highly useful for identifying posterior circulation stroke. It should not be applied to brief episodic positional vertigo.

212. A. Wernicke encephalopathy results from thiamine deficiency and classically affects ocular motor function, gait, and mental status, though the full triad may be incomplete. Thiamine should be given

promptly, especially before or with carbohydrate, because glucose utilization can worsen thiamine depletion. Treatment is low risk and delay can cause irreversible injury.

213. D. Overly rapid correction of chronic severe hyponatremia can cause osmotic demyelination, classically affecting the central pons and sometimes extrapontine structures. Symptoms are often delayed and may include dysarthria, dysphagia, altered consciousness, and quadriparesis. Prevention depends on controlled correction and careful monitoring.

214. E. The triad of depressed consciousness, respiratory depression, and miosis is classic for opioid toxicity, and rapid response to naloxone strongly supports the diagnosis. Structural brainstem lesions can also produce small pupils and coma, so persistent focal findings or incomplete response should prompt urgent neuroimaging.

215. A. Alzheimer disease typically begins with progressive episodic memory impairment associated with medial temporal and hippocampal dysfunction, later involving language, visuospatial skills, and orientation. Vascular cognitive impairment more often has a temporal relationship to vascular injury or a subcortical executive pattern, although mixed pathology is common in older adults.

216. B. Subcortical vascular cognitive impairment often produces executive dysfunction, slowed processing, gait impairment, and mood or urinary symptoms in association with lacunes and extensive white-matter disease. Memory can be impaired but may reflect retrieval and executive problems rather than a primary storage deficit. Mixed vascular and neurodegenerative pathology remains common.

217. A. Anosognosia is impaired awareness of a neurologic deficit and is especially associated with right hemispheric lesions and neglect networks. It has major safety implications because the patient may not appreciate fall risk or need for assistance. Rehabilitation and discharge planning should account for impaired insight.

218. C. A relapsing CNS syndrome with optic neuritis, myelitis, characteristic MRI lesions, and CSF oligoclonal bands strongly supports multiple sclerosis. Vascular disorders can mimic some focal deficits but do not produce the same inflammatory demyelinating pattern. Accurate distinction prevents inappropriate antithrombotic treatment for a nonvascular disease.

219. B. Aquaporin-4 IgG strongly supports neuromyelitis optica spectrum disorder. NMOSD can cause longitudinally extensive myelitis, severe optic neuritis, and area postrema attacks with otherwise unexplained hiccups or vomiting. Recognizing this inflammatory syndrome is important because it requires immunotherapy rather than vascular reperfusion or antithrombotic treatment.

220. E. Limbic encephalitis can present subacutely with memory loss, seizures, psychiatric symptoms, and medial temporal MRI abnormalities. Small-cell lung cancer is associated with several paraneoplastic antibodies and immune-mediated neurologic syndromes. Evaluation includes autoimmune and infectious testing while treating seizures and the underlying immune process.