Now with 620 questions—more than 250 completely new to this edition—this review guide has been thoroughly revised to reflect current science and clinical knowledge. With improved diagnostic-quality images, an emphasis on new drugs, and added chapters devoted to anatomy, clinical trials and ethics, neuro-ophthalmology, and case studies, this comprehensive review covers the full range of topics tested on the ABPN vascular neurology certification and MOC exams. Vascular Neurology Board Review is an engaging, active method to gauge proficiency and identify gaps for further study. Questions and answers with detailed rationales address a broad mix of topics including basic science, pharmacology, epidemiology and prevention, recovery and rehabilitation, and recognition, evaluation, and treatment of cerebrovascular diseases and associated clinical problems. Each answer is accompanied by a relevant reference to guide further study. The book is a must-have review tool for anyone taking the vascular neurology subspecialty exam, and for physicians who want to enhance their understanding of stroke and stroke-related issues and concerns.

Key Features:
- Contains 620 board-style questions and answers with rationales and references
- Covers all topic areas on the ABPN content outline for vascular neurology boards and the MOC exam
- 85 images reinforce key diagnostic points and build interpretive skills
- 5 new chapters
- All questions reviewed and updated to include the latest scientific, clinical, and treatment information
- Includes downloadable ebook to broaden study options

Recommended Shelves Category: Neurology
Vascular Neurology Board Review
Vascular Neurology Board Review: Questions and Answers

Second Edition

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To my late husband, Clark H. Millikan, MD, a giant in the field of vascular neurology.
Nancy Futrell, MD

To those in my past and present who have given me the strength to complete this edition.
Dara G. Jamieson, MD
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Since the publication of the first edition of Vascular Neurology: Questions and Answers, the science and clinical knowledge in this field have grown exponentially. The first group of us who sat for the board exam have now taken the first MOC exam, which included topics not in the original exam, such as questions on medical ethics. We have had multiple requests from candidates for both exams to produce an undated book in the practice exam format, and this second edition has been written to fulfill that need.

The second edition has been expanded from 512 questions to over 600 questions. There are more than 250 new questions. We have included new sections on ethics and neuro-ophthalmology. Some outdated questions from the first edition were omitted and most other questions were completely reworked in view of changes in the basic and clinical sciences over the past 10 years. Every question in the second edition has been reviewed multiple times for accuracy and we have worked diligently to avoid errors, with the addition of collaborators to add another layer to the review process.

We have improved the quality of the images. Image quality is a chronic complaint on the board exam. This is likely (at least in part) due to the expectation that we, as vascular neurologists, must interpret images produced with both cutting-edge technology and some older technologies with less-than-perfect resolution and patient positioning, along with motion artifacts. The point of reviewing images in this book and in the board exam is that (a) the images are of diagnostic quality, and (b) systematic review of the images should lead the reader (examinee) to the correct diagnosis.

This book is written as a study guide/self-evaluation tool to prepare for the ABPN exam and MOC exam in vascular neurology. It is representative of the areas tested on the exam, including the standard clinical and basic science of stroke and some of the esoterica that appear on the exam. The book should also serve as a study guide for any neurologist, internist, or family practitioner interested in expanding his or her knowledge in this important field.

The authors have found this practice exam question-and-answer format to be an effective and engaging study method, as opposed to a didactic review or summary reader. We find it useful also to identify areas of weakness that require further study. Explanatory answers with appropriate references are included to facilitate learning. Most of the references are relatively recent, but for certain topics, we have included the classic references, such as Niels Lassen’s work on incomplete cerebral ischemia and Anthony Furlan’s classic description of retinal ischemia presenting as visual change in bright light.

As with the first edition, the authors and the collaborators have learned a tremendous amount in the process of preparing this learning tool for our colleagues. We hope this book will provide you with an interesting and rewarding learning tool.

Nancy Futrell
Share

Neurovascular Imaging

QUESTIONS

1. Restricted diffusion following stroke is most directly associated with
   A) Hemorrhagic transformation of acute stroke
   B) Cytotoxic edema
   C) Vasogenic edema
   D) Interruption of the blood–brain barrier
   E) T2 shine-through

2. Which statement is true about diffusion weighted imaging (DWI) in patients with sudden onset of focal neurological symptoms?
   A) DWI images are easily degraded by motion.
   B) DWI images are not useful for tissue plasminogen activator (tPA) decisions, as imaging acquisition time is prolonged.
   C) DWI images are both sensitive and specific for hyperacute cerebral infarction.
   D) Restricted diffusion is seen on DWI within 10 minutes following acute cerebral infarction.
   E) If there is no corresponding restricted diffusion on DWI, tPA should not be offered to a patient with a sudden onset of neurological deficit 2 hours previously, as negative DWI implies this is a stroke mimic.

3. Which statement is true about diffusion weighted images (DWIs) and perfusion weighted images (PWIs)?
   A) With occlusion of large or medium cerebral arteries, the lesion seen on DWI is generally larger than that seen on PWI at 3 hours.
   B) With the occlusion of penetrating arteries, the size of the lesion on DWI is generally smaller than that on PWI at 3 hours.
   C) Gadolinium contrast is necessary to obtain PWI and DWI.
   D) Lesions on DWI become progressively smaller, with minimum lesion size seen at 2 to 3 days.
   E) The ischemic penumbra refers to tissue hypoperfusion without restricted diffusion, representing potentially salvageable tissue.

4. Which statement is correct about the sequence of changes on MRI following acute ischemic stroke?
   A) Acute ischemic lesions are seen on T2 weighted images (T2WIs) earlier than they are seen on DWIs.
   B) Acute ischemic lesions are seen on T2WIs earlier than cytotoxic edema is seen on T1WIs.
   C) Contrast enhancement of ischemic lesions demonstrates the development of cytotoxic edema.
   D) T2WI has a high degree of sensitivity for acute stroke within the first 4 hours.
   E) Signal changes on diffusion weighted images (DWIs) are more sensitive than signal changes on T1WI for the detection of acute ischemic changes.

ANSWERS TO THIS SECTION CAN BE FOUND ON PAGE 214
5. Which of the following findings on MRI/MR angiography (MRA) confirm a diagnosis of Fabry disease in a 40-year-old stroke patient?
   - A) Vertebrobasilar ectasia
   - B) Posterior circulation infarcts
   - C) Pulvinar hypointensity on T1 weighted imaging (T1WI)
   - D) Extensive white matter disease
   - E) None of the above

6. Which of the following is a definite contraindication to CT angiography (CTA)?
   - A) Shellfish allergy
   - B) Claustrophobia
   - C) Cardiac pacemaker
   - D) Pregnancy
   - E) All of the above

7. Which statement is true about a hyperdense artery sign on head CT without contrast?
   - A) The hyperdense artery sign most likely represents atherosclerosis.
   - B) The sensitivity of the hyperdense artery sign for acute thrombus is 80%.
   - C) Sensitivity is best with 5-mm slices.
   - D) The specificity of the hyperdense artery sign for acute thrombus is 96%.
   - E) The hyperdense artery sign cannot be considered a reasonable surrogate marker to confirm the diagnosis of ischemic stroke.

8. Which statement is true about imaging diagnosis of cerebral venous sinus thrombosis (CVST)?
   - A) Digital subtraction angiography (DSA) is generally necessary to make a diagnosis of CVST.
   - B) The “empty delta sign” seen on noncontrast CT is often present with superior sagittal sinus (SSS) thrombosis.
   - C) Flow characteristics of the jugular bulbs often produce a false-positive suggestion of occlusion.
   - D) Acute CVST is reliably detected on MRI with T2 weighted images (T2WIs).
   - E) Subacute CVST is reliably detected on T1 images with contrast, with precontrast scans not needed.

9. Please answer both questions 9 and 10 before referring to the explanatory answer. MRI images (A) to (D) are most compatible with:

   ![MRI Images](A) FLAIR  ![MRI Images](B) DWI
10. The images in question 9 in the preceding text show lesions that

A) Generally result in permanent infarction
B) Generally are reversible if proper treatment is administered
C) Occur most often in women
D) Are most likely due to cerebral ischemia
E) Generally resolve completely in the brainstem but not in the cortex

11. Match the arrows with the structures. Each answer can be used only once, but some answers will not be used. Match arrows 1 through 5 with the following answers:

A) Middle cerebral artery
B) Vein of Galen
C) Jugular vein
D) Sigmoid sinus
E) Superior sagittal sinus
F) Transverse sinus
G) Straight sinus
H) Internal cerebral vein
I) Vertebral artery
J) Basilar artery
12. An infant of African descent was adopted by a couple in the United States. The birth parents were both healthy, and the birth mother used no alcohol or drugs (prescription or recreational) during the pregnancy. Pregnancy and delivery were normal. In the first 3 to 4 months, the infant met developmental milestones normally, although it was noted that she reached for things preferentially with her left hand. As her motor skills progressed, decreased fine motor skills and posturing in the right upper extremity were noted. At 6 months, she was referred by her primary for a stroke diagnosed by CT scan. She was alert and attentive to her environment. Vision seemed intact. She had hypertonia, posturing, and decreased fine motor control in the right hand with mild hypertonia in the right leg. The most likely diagnosis is

A) Prenatal or perinatal left middle cerebral artery (MCA) ischemic stroke
B) Schizencephaly
C) Prenatal or perinatal intracranial hemorrhage
D) Congenital absence of the left MCA
E) Arachnoid cyst

13. Cerebral microbleeds (CMBs)
   A) Are easily detected on gradient-recalled echo (GRE) T2* weighted MRI sequences
   B) Indicate focal acute hemorrhage
   C) Are independent of increasing age
   D) Are independent of hypertension
   E) Are associated with increased serum cholesterol

14. A 74-year-old male presented for evaluation of “amyloid angiopathy.” He had a long-standing seizure disorder and was on both phenytoin (Dilantin) and gabapentin (Neurontin). He has a history of gastrointestinal (GI) bleed and frequent nosebleeds. His wife noted that he had “slowed down,” had memory problems, and his mood seemed less stable. This MRI was obtained, and the patient was referred to a vascular neurologist. This disorder:
   A) Has an autosomal dominant inheritance pattern
   B) Affects only the brain
   C) Essentially always has normal findings on CT angiography (CTA) of the brain
D) Presents most often with focal neurologic deficits
E) All of the above

15. The patient described in question 14 has three children. The recommended advice to the children is
A) No testing unless symptoms develop
B) MRI of the brain with and without contrast
C) Cerebral angiography
D) Computed tomography of the brain with and without contrast
E) CT angiography (CTA) or MR angiography (MRA) of the brain

16. Match the stage of a cerebral hematoma on the left with the type of blood product that is predominant at that stage on the right.
A) Hyperacute (<24 hours) 1. Methemoglobin in macrophages
B) Acute (1–3 days) 2. Extracellular methemoglobin
C) Early subacute (3–7 days) 3. Deoxyhemoglobin
D) Late subacute (1–2 weeks) 4. Hemosiderin
E) Chronic (>1 month) 5. Oxyhemoglobin

17. Compared to gray matter, fresh (hyperacute <24 hours) blood in the brain can be
A) Hyperintense on T1 and hyperintense on T2
B) Hyperintense on T1 and isointense on T2
C) Isointense on T1 and hyperintense on T2
D) Isointense on T1 and hypointense on T2
E) Hyperintense on susceptibility weighted imaging (SWI) and hyperintense on T2

18. Match the blood product on the left with the MRI appearance on the right. Each blood product has 2 correct answers, one for T1 and one for T2.
A) Oxyhemoglobin 1. Hypointense on T1
B) Deoxyhemoglobin 2. Isointense on T1
C) Intracellular methemoglobin 3. Hyperintense on T1
D) Extracellular methemoglobin 4. Hypointense on T2
E) Hemosiderin 5. Hyperintense on T2
19. The timing of a hemorrhage seen on MRI
   A) Is simple and precise
   B) Is based on the imaging properties of oxygen bound to hemoglobin
   C) Is frequently complicated by the overlapping signal characteristics of hemoglobin breakdown products along with variable maturation rates within the hematoma
   D) Is only understood by neuroradiologists

20. Which statement is most likely correct regarding a patient with the diffusion weighted imaging (DWI) images in the following text?
   A) The most likely etiology is a vertebral artery dissection.
   B) The patient had sudden onset of altered mental status, vertical gaze palsy, and memory impairment.
   C) The patient had embolic infarcts involving three intracranial arteries.
   D) The patient most likely has metastatic lung cancer.
   E) The patient is most likely to respond to aspirin for secondary prevention.

21. This is an image from a patient who presented with a chief complaint of 10 minutes of “loss of vision in the right eye,” which resolved completely. Neurological examination was normal. Carotid duplex, transesophageal echocardiogram (TEE), and 48-hour heart monitor were normal. Based on the MR angiography (MRA) images, which of the following treatment options is most appropriate acutely for this patient?
Neurovascular Imaging

ANSWERS

1. The answer is B. Restricted diffusion of water molecules occurs within minutes following loss of tissue perfusion, with the main contributor being cytotoxic edema. Cytotoxic edema is the earliest form of edema, which is correlated with cellular swelling. Vasogenic edema occurs following breakdown of the blood-brain barrier, where fluids from the intravascular space traverse into the extracellular space in the brain. This starts after 4 to 6 hours of ischemia. Vasogenic edema also produces changes in diffusion, although the signal may be either hypointense or hyperintense because of the T2 contribution to the diffusion-weighted images (DWIs). T2 shine-through is a phenomenon that can cause a false-positive impression of hyperacute stroke in an old lesion, unless appropriate correlation is performed with exponential images and apparent diffusion coefficient (ADC) maps. This is again because of the T2 contribution to the image, which is removed in exponential images. (Schaefer PW, Copen WA, Lev MH, Gonzalez RG. Diffusion-weighted imaging in acute stroke. Neuroimag Clin N Am. 2005;15:503–530.)

2. The answer is C. In a patient with acute onset of focal neurological symptoms, DWI has high sensitivity (88%–100%) and specificity (88%–100%) for hyperacute cerebral infarction. DWI is not specific for acute cerebral infarcts in the absence of the history of an acute onset of a focal neurological deficit, as other disorders can have restricted diffusion (herpes encephalitis, diffuse axonal injury from trauma, some multiple sclerosis [MS] lesions, etc.). DWI is resistant to motion artifact and has short acquisition times, with imaging times ranging from seconds to 2 minutes. Restricted diffusion has been seen on images in animals as early as 10 minutes, but the earliest lesions in human stroke begin closer to 30 minutes. (The reason for this difference is not entirely clear, but it may be related to the high-field Tesla research magnets that were used in these animal studies.) The lack of a lesion on DWI does not always rule out an acute stroke, particularly with small lesions in the brainstem or deep gray matter nuclei. (Schaefer PW, Copen WA, Lev MH, Gonzalez RG. Diffusion-weighted imaging in acute stroke. Neuroimag Clin N Am. 2005;15:503–530.)

3. The answer is E. The core of an infarct, which is irreversibly damaged, is tissue with both low perfusion and restricted diffusion. The ischemic penumbra is the area surrounding the core, which has low perfusion but has no restricted diffusion. This tissue is potentially salvageable if perfusion is restored, but it will become infarcted if hypoperfusion continues. With an ischemic penumbra, the defect on DWI is smaller than the defect on PWI, also known as diffusion/perfusion mismatch. The penumbra is seen in moderate-to-large infarcts and is generally not seen in small infarcts of penetrating arteries. In these smaller infarcts, DWI and PWI images are generally similar in size. Gadolinium is used for obtaining PWI, but not DWI. Lesions on DWI in medium-to-large infarcts generally increase with time over the first 2 to 3 days. (Schaefer PW, Copen WA, Lev MH, Gonzalez RG. Diffusion-weighted imaging in acute stroke. Neuroimag Clin N Am. 2005;15:503–530.)
4. **The answer is E.** DWI is the sequence most sensitive for hyperacute ischemic stroke. T1WI shows morphologic changes of cellular swelling due to cytotoxic edema between 2 and 4 hours. Changes in T2WI and later changes on T1WI are due to vasogenic edema with breakdown of the blood–brain barrier (BBB). These appear on T2WI at about 8 hours and on T1WI between 16 and 24 hours. Contrast enhancement of ischemic lesions is also due to breakdown of the BBB, but is generally first seen after 24 hours. (Schwam LH, Koroshetz WJ, Sorensen AG, et al. Time course of lesion development in patients with acute stroke: serial diffusion- and hemodynamic-weighted magnetic resonance imaging. *Stroke.* 1998;29:2268–2276.)

5. **The answer is E.** Pulvinar hyperintensity (not hypointensity) has been reported as “pathognomonic,” but this has been questioned after it was not seen in any of a group of 21 patients with confirmed Fabry disease. Vertebrobasilar ectasia, posterior circulation infarcts, pulvinar hyperintensity on T1WI, and extensive white matter disease are all features seen in some patients with Fabry disease, but they are not specific enough to separate patients with Fabry disease from other young stroke patients. (Fazekas F, Enzinger C, Schmidt R, et al. Brain magnetic resonance imaging findings fail to suspect Fabry disease in young patients with an acute cerebrovascular event. *Stroke.* 2015;46:1548–1553.)

6. **The answer is D.** Intravenous contrast should not be used on pregnant women. Cardiac pacers and claustrophobia can be contraindications to MRI but not to CT. Shellfish allergy is often thought to be a contraindication to iodine contrast, but that is not always the case. A patient with “shellfish allergy” should be questioned carefully as to the nature of the reaction to shellfish. Some causes of shellfish intolerance do not necessarily represent iodine allergies. (Pasternak JJ, Williamson EE. Clinical pharmacology, uses, and adverse reactions of iodinated contrast agents: a primer for the non-radiologist. *Mayo Clin Proc.* 2012;87(4):390–402.)

7. **The answer is D.** The hyperdense artery sign is 96% specific for an acute thrombus or embolus. When it is present, it can be considered a reasonable surrogate marker for a compromised vessel and an ischemic stroke. Atherosclerosis can also produce hyperintensity in arteries, but the signals are not as homogeneous as the hyperdense artery sign. Calcified plaques have an inhomogeneous appearance with some calcium signal. The sensitivity for detection of a hyperdense artery is increased with thinner slices, 3-mm slices. Although specificity is high, sensitivity is only 54%. Thus, many occluded arteries are NOT hyperdense, so the lack of a hyperdense artery sign does not rule out an occlusion. (Mair G, Boyd EV, Chappell FM, et al. Sensitivity and specificity of the hyperdense artery sign for arterial obstruction in acute ischemic stroke. *Stroke.* 2015;46:102–107.)

8. **The answer is C.** As the jugular bulbs frequently have turbulent flow, this may produce a “pseudo occlusion.” MR venogram (MRV) and CT venogram (CTV) are both reliable for the diagnosis of CVST. DSA is rarely required. The “empty delta sign” can be seen with SSS thrombosis, but this is with a contrast enhanced CT rather than a noncontrast CT, as contrast surrounds an “empty” triangular filling deficit. Acute thrombus is so hypointense on T2WI that it may mimic a flow void. Because subacute thrombus is bright on T1WI, precontrast images are important. (Osborn AG. *Osborn’s Brain: Imaging, Pathology and Anatomy.* Salt Lake City, UT: Amirsys; 2013.)

9. **The answer is B.** Acute hypertensive encephalopathy produces MRI changes known as PRES, posterior reversible encephalopathy syndrome. It is also known as reversible posterior leukoencephalopathy syndrome (RPLS). PRES is not limited to hypertensive encephalopathy, but also has other causes. Most, but not all, patients with PRES do have hypertension as a component of their clinical picture. Image A shows hyperintensity of the posterior portion of the medulla, with no corresponding restricted diffusion on DWI (B). Images C and D show relatively symmetrical hyperdense areas in the basal ganglia. Classic lesions in acute PRES are bilateral lesions with some degree of symmetry, with hyperintensity on T2 and fluid-attenuated inversion recovery (FLAIR)
and no restricted diffusion. The posterior circulation is most often involved, but lesions can also be seen in the anterior circulation. Bilateral occipital lesions are present frequently, but occipital involvement is not invariable as in this case. Status epilepticus can occasionally produce an imaging picture similar to PRES, as can hypoglycemia, but the clinical picture will be obvious in those patients. Global hypoxia/ischemia can present with bilaterally symmetrical hyperintensity on T2, but the location is most often basal ganglia and cerebellum, and restricted diffusion will be present. Cerebral vasculitis can present with variable distribution of lesions, but there will be restricted diffusion in acute lesions. Although MS has hyperintense lesions on T2WI without restricted diffusion, the bilateral symmetry of lesions in multiple structures is not consistent with MS. (Osborn AG, Salzman KL, Barkovich AJ. Diagnostic Imaging: Brain. 3rd ed. Salt Lake City, UT: Amirsys; 2016.)

10. The answer is B. This image is from a preeclamptic woman with severe hypertension and posterior reversible encephalopathy syndrome (PRES)—please refer to question 7 in Chapter 15 for the history. These are the films of that patient. If blood pressure is normalized in a timely fashion, the clinical and imaging sequelae generally resolve. Cortical lesions are more likely to be permanent than brainstem white matter lesions. PRES is easily differentiated by acute cerebral ischemia, as restricted diffusion is almost always present with acute ischemia and is not present with PRES. PRES occurs more often in women than in men. This case is atypical, as there is less posterior involvement than in most cases, but it illustrates the important concept that lesions in PRES have significant variability in location. (Osborn AG, Salzman KL, Barkovich AJ. Diagnostic Imaging: Brain. 3rd ed. Salt Lake City, UT: Amirsys; 2016.)

11. The answer is 1A, 2B, 3G, 4J, 5F. This is a CT angiography (CTA)/CT venogram (CTV) of a patient with an extracranial-intracranial (EC-IC) bypass. The CTA images arteries and veins simultaneously, unlike catheter angiography with an arterial phase and a venous phase. In this situation, the superior temporal artery enters the intracranial space via the surgical skull defect and anastomoses with the middle cerebral artery (MCA), representing an EC-IC bypass. The other vessels are standard normal anatomy. (Osborn AG. Osborn’s Brain: Imaging, Pathology and Anatomy. Salt Lake City, UT: Amirsys; 2013.)

12. The answer is B. Of note, on the CT scan, the cortical ribbon can be seen posterior to the cerebrospinal fluid (CSF) density, being more obvious on the MRI. With an MCA stroke, the cortical ribbon in the MCA territory would be destroyed, with the borders of the infarct being subcortical white matter. This is an example of schizencephaly with an open lip lined with gray matter or cortical ribbon. As with pediatric stroke, focal neurologic deficits are frequently not notable for several months. As motor development progresses, asymmetry becomes more obvious. (Osborn AG, Salzman KL, Barkovich AJ. Diagnostic Imaging: Brain. 3rd ed. Salt Lake City, UT: Amirsys, 2016.)
13. The answer is A. CMBs are focal areas of signal loss on GRE T2* weighted MRI sequences due to focal hemosiderin deposition from previous small bleeds. It should be noted that susceptibility weighted imaging (SWI) is the state of the art for the detection of hemosiderin, but at the time of this writing, GRE is still performed widely, particularly in community hospitals, and remains sensitive for hemosiderin. Vascular neurologists will be seeing GRE images in the clinic for some time, including on previous studies brought in by patients. CMBs are believed to be due to previous extravasation of blood associated with bleeding-prone small vessel disease. Increasing age and blood pressure, as well as decreased cholesterol, increase the risk for CMBs. (Cheng AL, Batool S, McCreary CR, et al. Susceptibility-weighted imaging is more reliable than T2*-weighted gradient-recalled echo MRI for detecting microbleeds. Stroke. 2013;44:2782–2786.)

14. The answer is A. This is an MRI of a patient with HHT—hereditary hemorrhagic telangiectasia, also known as Osler–Weber–Rendu syndrome. This disorder is inherited as an autosomal dominant. This patient had a history of epistaxis, which is the most common presentation. He also had telangiectasias on his lips and a history of GI bleed. Multiple areas of chronic hemorrhage were present on MRI. CTA of the brain often reveals arteriovenous malformations (AVMs), which can be multiple. The brain imaging findings seen here are seen most frequently with cerebral amyloid angiopathy (CAA), but the clinical history with the association of systemic hemorrhages (GI blood and nosebleeds) is not consistent with CAA, which is limited to the brain. (Osborn AG, Salzman KL, Barkovich AJ. Diagnostic Imaging: Brain. 3rd ed. Salt Lake City, UT: Amirsys; 2016.)

15. The answer is B. Because of the autosomal inheritance pattern of hereditary hemorrhagic telangiectasia (HHT) and the potentially devastating consequences in those patients with HHT with cerebral arteriovenous malformations (AVMs), screening of first-degree relatives is recommended. The test of choice is MRI with and without contrast. Invasive or noninvasive vascular imaging then may be appropriate in patients with abnormal MRIs, depending on the findings. This screening should also be done in patients with systemic evidence of HHT, including mucocutaneous and visceral telangiectasias. In this case, the patient’s sister had a history of epistaxis and hemorrhagic changes on MRI. We recommended that his daughter come in for evaluation, but she declined. Two years later, she suffered a disabling hemorrhage from a cerebral AVM. (Osborn AG, Salzman KL, Barkovich AJ. Diagnostic Imaging: Brain. 3rd ed. Salt Lake City, UT: Amirsys; 2016.)

16. The answers are A5, B3, C1, D2, E4. The process of transformation of hemoglobin following intracerebral hemorrhage is oxyhemoglobin, deoxyhemoglobin, intracellular methemoglobin, extracellular methemoglobin, and hemosiderin. The time frames of this transformation are approximate. (Osborn AG, Salzman KL, Barkovich AJ. Diagnostic Imaging: Brain. 3rd ed. Salt Lake City, UT: Amirsys; 2016.)

17. The answer is C. On T1, fresh blood can be either hypointense or isointense. On T2, fresh blood is hyperintense, but a fresh hematoma will frequently have a surrounding rim of hypointensity representing early deoxygenation (formation of oxyhemoglobin). On SWI, hyperacute hemorrhage will have a hypointense ring. (Osborn AG, Salzman KL, Barkovich AJ. Diagnostic Imaging: Brain. 3rd ed. Salt Lake City, UT: Amirsys; 2016.)

18. The answers are A2,5; B2,4; C3,5; D3,5; E1,4. The T1 appearance of a hematoma progresses from isointense to hyperintense to hypointense. The T2 appearance of a hematoma progresses from hyperintense to hypointense, then again to hyperintense, followed by the long-term hypointensity. (Osborn AG, Salzman KL, Barkovich AJ. Diagnostic Imaging: Brain. 3rd ed. Salt Lake City, UT: Amirsys, 2016.)