NCPD 2.0 ANCC CONTACT HOURS

TERESA FERREIRA/SHUTTERSTOCK

Acute ischemic stroke or migraine with aura? Triage considerations

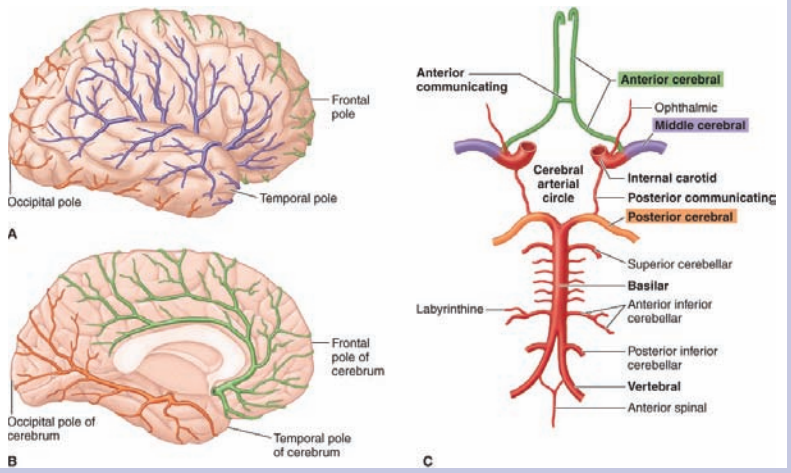
BY ANGELA COLLINS-YODER, PhD, RN, CCNS, ACNS BC, SCRN, AND BETTY KEY, EdD, MSN, RN, CCRN

Abstract: This article discusses the complex relationship between acute ischemic stroke and migraine with aura, and critical nursing interventions.

Keywords: ischemic stroke, migraine with aura, thrombectomy, visual aura

MB, A 26-YEAR-OLD White female with a 13-year history of migraines with visual aura, was brought to the ED with complaints of a severe headache and change in mental status. She had difficulty answering orientation questions, photophobia, diplopia, partial hemianopia, ataxia, left arm and left leg drift, and dysarthria. She needed repeated verbal stimulation to be aroused. Her initial National Institutes of Health Stroke Score (NIHSS) was 12, so a stroke alert was called. She is a nonsmoker, currently not sexually active, and not using birth control. Two and one-half hours prior to ED admission, it was reported that she was symptom-free. Two hours prior to admission, she started getting a headache and took one dose of her selective serotonin 5-HT(1B/1D) agonist (triptan) as prescribed.

The anterior and posterior circulations of the brain



Note the internal carotid artery, the vertebral arteries, basilar artery, and the distribution patterns of the anterior (green), middle (purple), and posterior cerebral (orange) arteries. A. Right lateral view of right cerebral hemisphere. B. Medial view of left cerebral hemisphere. C. Inferior view.

Source: Dirette DP, Gutman SA. Occupation Therapy for Physical Dysfunction. 8th ed. Philadelphia, PA: Wolters Kluwer; 2021.

Acute ischemic strokes (AIS) make up 87% of all strokes, making it the fifth leading cause of death in the United States.[1-3] AIS occurs when blood flow is absent in a portion of the brain, cutting off the oxygen supply to the neuronal cells. Patients having an acute stroke, either ischemic or hemorrhagic, demonstrate neurologic deficits that point to the region of brain ischemia and infarction. The sooner a patient with an AIS receives treatment with a fibrinolytic or mechanical thrombectomy, the better the outcome. About 20% of patients evaluated for stroke signs and symptoms actually have stroke mimics, the third most common of which is migraine with aura (MA).[2,4-6] MA is associated with a twofold increased risk of AIS.[3-5] This article discusses the complex relationship between AIS and MA, and critical interventions.

Migraine affects 12% of the US adult population, of which 25% have MA.[4,5] Aura is a set of sensory symptoms, typically visual, that occurs prior, during, or after a migraine headache.[4,6-8] MA is a subtype of recurrent headaches that is fully reversible and includes neurological, visual, and other sensory symptoms.[4,5,9] It can present as a neurologic deficit, making it a challenge to differentiate between the two neurologic disorders. The triage dilemma, therefore, is if a patient's signs and symptoms represent an MA or an AIS.[5,7] Treatment options for these disorders are very different. Identifying a patient's history of MA can help the stroke team pursue additional testing and identify an optimal treatment plan.[5-7] (See *Triage questions to distinguish MA from AIS.*)

Pathophysiology of AIS and MA

The common denominator in both AIS and MA is altered perfusion to a portion of the brain. Insufficient blood flow leads to neurologic deficits.[7-9] In AIS, this is due to a thrombus or embolism occluding a cerebral artery. Depending on the area of the brain the artery supplies, the signs and symptoms will present as stroke syndromes pointing to the portion of the brain that is hypoxic.[2] The area of injured hypoxic brain cells is called the stroke penumbra. The goal of AIS treatment is to rapidly resume adequate perfusion to the cells within the stroke penumbra by fibrinolytic drugs or clot extraction. Rapid restoration of cerebral perfusion often improves functional outcomes.[2,3]

The precise pathophysiology of migraine headaches with and without aura is not fully understood. A recent new drug classification has demonstrated that in MA, blood flow is disrupted by an abnormal genetic neuropeptide regulation in certain cerebral arteries.[5,6] One neuropeptide found in a migraine attack is calcitonin gene-related peptide (CGRP). When coupled with the receptor, CGRP activates the trigeminal nerve and cervical nerve pathway, causing pain and meningeal irritation.[5,6] Additional mechanisms in MA include fluctuations in hormone levels and injuries to the neurons from repeated vascular inflammation called sterile inflammation. Radiologic studies performed at the initiation of an MA indicate that the hypothalamus releases neuroendocrine hormones associated with headache onset and duration.[5,10,11] This atypical neurovascular chemistry causes diminished perfusion to the posterior circulation of the brain, leading to neurologic impairment. In addition, the patient experiences disabling pain and nausea. Treatment goals for MA headaches are to administer medication to halt the abnormal neurochemistry, resolve the pain and nausea, and return normal neurologic functioning.[12] Diminished cerebral perfusion may cause loss of brain tissue in a prolonged MA episode.[10,11]

Differences in signs and symptoms

Stroke signs and symptoms are assessed using the mnemonic "BE

FAST": **(B) balance** such as ataxia upon movement; **(E) eyes** because visual problems like diplopia, blind spots, and blurred vision are linked to the posterior circulation; **(F) face** since anterior circulation strokes may cause drooping of one side of the face; drift and/or pronation when both **(A) arms** are extended palms up at shoulder height; absent, slurred, or nonsensical **(S) speech**; and the **(T) time** last known well (LKW), which applies to all stroke types. The majority of AIS involve the anterior circulation, commonly the middle cerebral artery.[3,13,14] Posterior circulation strokes account for approximately 20% to 25% of AIS in all age ranges (See *The anterior and posterior circulations of the brain*).[13]

MA signs and symptoms are correlated with the anatomy of the posterior circulation of the brain along the trigeminal nerve pathways.[5,10] Clinically silent infarcts are associated with MA, often in the cerebellar region.[6,15] Posterior ischemic strokes may have subtle assessment findings such as abnormalities in vision, balance, and speech (dysarthria).[16] If the patient can communicate, they should be asked if the aura experience is typical or atypical of past migraines. If a patient with a history of MA presents with signs and symptoms of anterior circulation compromise, the patient is triaged as having an acute stroke.[11,12,15]

Sample patient case

MB's computed tomography (CT) scan of the head demonstrated no hemorrhage and her NIHSS score remained 12. Her BP was 160/88, heart rate was 104, respirations were 14/minute, and pulse oximetry reading was 97% on room air. The neurologist also ordered computed tomographic angiography (CTA) and a CT perfusion study to help determine if the neurologic signs and symptoms represented AIS or MA. CTA demonstrated a basilar artery partial occlusion at the level of the origin of the anterior inferior cerebellar artery (AICA), which is an AIS. The stroke care team reviewed the fibrinolytic checklist and noted no contraindications to fibrinolytic therapy.[2] The risks, benefits, and alternatives were explained to the patient and her family, and consent for treatment was obtained. A weight-based alteplase bolus and infusion were then administered. The patient's NIHSS score improved from 12 to 9,which is still within moderate stroke range.

Stroke alert

When a stroke alert is called, all resources, personnel, and equipment needed to make optimal decisions and interventions for the patient immediately become available.[2] The zero on the stroke timeline is established as LKW, which is the time prior to hospital arrival when the patient was last known to be without the signs and symptoms of the current stroke or at their base-

Triage questions to distinguish migraines with aura (MA) from acute ischemic strokes (AIS)[5-7]

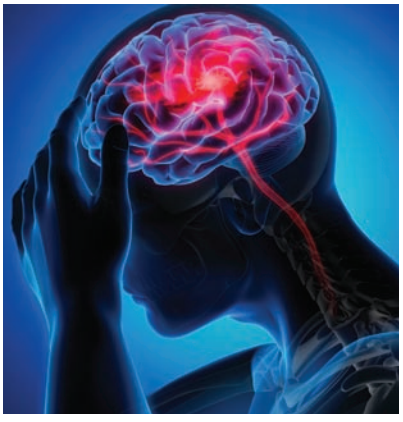
Question	Rationale
Which was first—the headache or the change in function?	Knowing if the headache happened first can help clarify the clinical diagnosis. In stroke, the initial signs involve a sudden change in or loss of neurological function. MA typically have presenting symptoms of visual changes and muscle tightness of the neck.
Is the aura typical or atypical of the patient's past headaches?	People with MA have a two-fold increased risk for stroke compared with those without aura. Individuals typically know the normal progression of their headaches.
To the family member: Is this typical or atypical of the patient's past headache episodes?	AIS is both a clinical and radiologic diagnosis. Family members may see changes in the patient that the patient is unaware of.
Are there additional risk factors of smoking or use of hormonal birth control?	These additional risk factors can increase the likelihood of an AIS.
Is the patient receiving radiation therapy to the head or neck?	A "stroke-like" headache called SMART (stroke-like migraine after radiation therapy) and neurologic decline are associated with radiation therapy to the brain.
Has the patient been treated for a recent sinus infection?	This may be associated with a sinus vein thrombosis, which may cause cerebral ischemia and increased intracranial pressure. Headache is the typical presenting complaint.
Is the patient possibly pregnant?	This could be a clinical diagnosis of posterior reversible encephalopathy syndrome in which vasogenic edema associated with pre-eclampsia and eclampsia causes neurologic decline.

line state of health. The LKW is the start of the treatment decision clock for the window of interventions. Upon arrival to the ED, patients should be assessed for the following: ABCs, vital signs, hypoglycemia (a frequent stroke mimic), pregnancy status as appropriate, and arrival NIHSS score. In some facilities, these assessments are completed by emergency medical services allowing the patient to go directly to the CT scanner.[2,13,16,21] An emergent CT scan of the head should be obtained within 20 minutes of ED arrival and interpreted within 45 minutes of ED arrival. The CT results, along with the initial patient data, are critical in deciding whether the stroke is ischemic or hemorrhagic.[16]

If the stroke is ischemic, the patient's focused history is used to determine if there are contraindications to fibrinolytic therapy and if the patient is within the 3-hour window from symptom onset (4.5 hours in select patients).[22] The focused history in a patient with MA should also include whether the patient has taken an abortive migraine medication and the patient's response to any abortive migraine medication taken. If the health history of a patient with MA includes factors such as smoking, hypertension, diabetes, and obesity, the probability of an acute stroke increases.[3,5] Pregnancy or hormonal changes can trigger MA episodes, thus it is important to ask related questions to a woman of childbearing age.[12]

Elevated BP greater than or equal to 185 systolic or 110 diastolic must be decreased prior to fibrinolytic therapy.[23] If indicated, the healthcare provider may also order CTA, which helps to visualize cerebral vessels to identify a large vessel occlusion.[13,22] A CT perfusion study can also outline the location and extent of the penumbra. The rationale for using the CT perfusion study is to ensure that a mechanical thrombectomy option is offered if viable ischemic tissue is present in the penumbra.[2,24-26] The interventional neurosurgeon or radiologist determines if a mechanical thrombectomy, in addition to fibrinolytic, would be beneficial.[21] Mechanical thrombectomies are indicated when, for example, larger cerebral vessels are occluded and if there is a large area of penumbra to be rescued.[22-24] CTA is performed in patients with stroke signs and symptoms that occurred within the past 24 hours to maximize the potential use of mechanical thrombectomy for better patient outcomes.[14,25]

MB's CTA demonstrated that she would benefit from a mechanical thrombectomy. Consent was obtained, and she was taken to the endovascular suite while the fibrinolytic infusion was in progress.[25] The interventional neurosurgeon removed a 9 mm thrombus. MB's NIHSS score improved to 3, which is in the range of a minor stroke, and she was transferred to the neuroscience CCU.



Healthcare providers must be vigilant in determining the risk of hemorrhagic conversion, the most feared complication of fibrinolytic therapy.

Critical care interventions

The most important interventions in the first 72 hours are assessment and reassessment for systemic and intracranial bleeding, changes in neurologic status, and pulse checks (which include femoral access site and pedal pulses).[2] Bleeding can be either external from puncture sites or internal. Healthcare providers must be vigilant in determining the risk of hemorrhagic conversion, the most feared complication of fibrinolytic therapy that occurs in 2%-7% of patients.[22-24] Hemorrhagic conversion of an AIS typically occurs within the first 36 hours after administration of the fibrinolytic agent.[20,24] Hemorrhagic conversion is often characterized by a patient's rapid neurologic deterioration. However, in those patients with a high NIHSS, neurologic deterioration may be identified through subtle declines.

Acute care stroke centers have protocols in place for when to notify the healthcare provider and for specific assessment intervals.[2,23,27,28] The American Heart Association guidelines recommend assessing patients every 5-15 minutes immediately after fibrinolytic therapy for changes in neurologic status, including NIHSS, I.V. site patency, and vital sign stability.[16,25,28] (See *Recommended interventions with fibrinolytic therapy*.[27-29]) These intervals continue until vital signs are stable and there are no neurologic status changes. Patients who require mechanical thrombectomy will need additional assessment of the puncture site and distal pulses.[29] Potential bleeding will also be assessed by lab values and vital sign changes.[24,25,29] If the patient has a history of a coagulopathy or anticoagulation use, additional coagulation studies will be monitored.[23-26,29]

Nurses must quickly determine problems that may impact patient outcomes after an AIS such as dysphagia, fever, fall risk, depression,

Recommended interventions with fibrinolytic therapy[27-29]

Assessment	Interval	Rationale
Perform neurologic assessments NIHSS Glasgow Coma Scale	Prior to fibrinolytic Every 15 minutes during the fibrinolytic infusion Continue every 15 minutes after end of fibrinolytic administration for 1st hour Every 30 minutes for next 6 hours Hourly from the 8th to the 24th hour post administration	To quickly intervene with additional measures and prevent additional loss of neural tissues
Observe for major and minor bleeding from multiple sites Major: intracranial, retroperitoneal, gastrointestinal, or genitourinary hemorrhages Minor: puncture sites, gums, hematomas, ecchymoses Bleeding precautions: Assess areas of legs under sequential pressure devices for bruising. Consider rotation of automatic BP cuff. Minimize blood draws.	Prior to fibrinolytic Every 15 minutes during the fibrinolytic infusion Continue every 15 minutes after end of fibrinolytic administration for the 1st hour Every 30 minutes for next 6 hours Hourly from the 8th to the 24th hour post administration	To quickly intervene and prevent or decrease blood loss
Obtain BP and use protocol medications to keep BP below 180 systolic and 105 diastolic.	Prior to infusion; treat with medications to minimize hemorrhagic conversion Every 15 minutes during the fibrinolytic infusion Continue every 15 minutes after fibrinolytic administration for 1st hour Every 30 minutes for next 6 hours Hourly from the 8th hour post administration until 24 hours	Maintaining BP within these ranges has been associated with fewer ischemic to hemorrhagic stroke conversions.
Monitor for signs and symptoms of angioedema or anaphylaxis.	Vital signs and physical assessment are done at each frequent assessment interval during the 24-hour time frame FDA drug labeling states that this serious drug reaction is most commonly noted during and up to 2 hours after infusion. Case reports show that some patients were also on concurrent angiotensin-converting enzyme inhibitors.	This is a rare but serious adverse drug reaction to fibrinolytics. Protocols to quickly respond to these reactions are recommended.
Reassess radiologic findings.	With any significant change in neurologic assessment and at 24 hours prior to initiation of medications that increase risk of bleeding such as antiplatelet agents or anticoagulants	To assess risk and benefits of additional interventions
Assess I.V. site for patency, infiltration, or extravasation. If extravasation occurs, terminate the infusion and apply local therapy following acute care policy.	Prior to fibrinolytic administration Every 15 minutes during the fibrinolytic infusion Continue every 15 minutes after end of fibrinolytic administration for 1st hour Every 30 minutes for next 6 hours Hourly from the 8th to the 24th hour post administration	For the patient to receive optimal dosage and prevent local tissue damage

and seizures.[2,25,27-29] Patients who have posterior strokes, such as MB, are at risk for silent aspiration and need a swallowing screen and an evaluation by a speech therapist. Preventing aspiration decreases the incidence of pneumonia after a stroke.[28] Fever increases oxygen demand in the stroke penumbra, creating an imbalance that can lead to a neurologic decline. Prompt intervention to keep the patient temperature within a normothermic target of 98.6° F (37°C) improves stroke functional outcomes.[2,29]

Post-stroke protocols should include rehabilitation services to evaluate changes in cognition associated with depression and possible seizures. Rehabilitation services must intervene as early as possible to improve the patient's functional status. Healthcare providers assess a modified Rankin score (mRs) that helps quantify the impact of the stroke on the patient's functional ability to return to normal activities of daily living after being discharged. The Rankin score will be reassessed after 90 days in the neurologist's office.

Stroke education with the entire interdisciplinary team should be seamless throughout the acute care episode. Education starts in the community, which should be informed about early recognition of stroke signs and calling 911 to activate EMS. Telling the dispatcher that the patient is having signs and symptoms of stroke helps in making the optimal dispatch to the geographically closest stroke-certified ED.

In MB's case, her family brought her to the ED without recognizing the seriousness of her "headache." The ED healthcare providers can explain the purpose of the CT scan as they also screen for contraindications to the fibrinolytic. The critical care or stroke unit nurse should explain the rationale for BP monitoring and any additional medications that are administered. Explaining aspiration precautions, such as dietary interventions, related to dysphagia is a joint effort by the speech therapist, nurses, and neurologists.[2,29] Prior to discharge, the rationale for each medication, any needed modification of stroke risk factors, in particular, BP monitoring, and all needed follow-ups should be reinforced by the teach-back method.[30,31] Providing patients with printed materials that they can review after hospital discharge has been linked to patient adherence. Stroke teams must also include a social worker or case manager who can evaluate any needed community resources to optimize patient adherence with postdischarge plans.

Patient case: Discharge and outcomes

MB was discharged with an NIHSS score of 3 and a mRs of 1, indicating no significant disability. She was able to carry out all usual activities despite some speech difficulties. She will continue having speech therapy in the outpatient setting. She will follow up with her neurologist for post-AIS care as well as modification of prevention therapies for her migraine with aura symptoms.[17,26,30] She will monitor her BP more closely and keep a migraine headache diary to assist in differentiating MA from AIS symptoms.[28,29,31] She was also advised to have her primary healthcare provider contact the neurologist prior to the initiation of any birth control that may involve hormone changes.[28] The patient expressed her gratitude to the healthcare team and surprise that a stroke can occur in someone her age, noting that she thought that it was just another headache. ■

REFERENCES:

1. Centers for Disease Control and Prevention. Stroke facts. www.cdc.gov/stroke/facts.htm.

2. Kiffon K, Livesay S, Wessol J. *AANN Comprehensive Review for Stroke*. 2nd ed. Chicago, IL: American Association of Neuroscience Nurses; 2020.

3. Virani SS, Alonso A, Benjamin EJ, et al. Heart disease and stroke statistics—2020 update: a report from the American Heart Association. *Circulation*. 2020;141(9):e139-e596. doi:10.1161/CIR.0000000000000757.

4. Migraine Research Foundation. Migraine facts. https://migraineresearchfoundation.org/about-migraine/migraine-facts/.

5. Otlivanchik O, Liberman AL. Migraine as a stroke mimic and as a stroke chameleon. *Curr Pain Headache Rep*. 2019;23(9):63. doi:10.1007/s11916-019-0801-1.

6. Terrin A, Toldo G, Ermani M, Mainardi F, Maggioni F. When migraine mimics stroke: a systematic review. *Cephalalgia*. 2018;38(14):2068-2078. doi:10.1177/0333102418767999.

7. Qubty W, Patniyot I. Migraine pathophysiology. *Pediatr Neurol*. 2020;107:1-6. doi:10.1016/j.pediatrneurol.2019.12.014.

8. Iyengar S, Ossipov MH, Johnson KW. The role of calcitonin gene-related peptide in peripheral and central pain mechanisms including migraine. *Pain*. 2017;158(4):543-559. doi:10.1097/j.pain.0000000000000831.

9. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. *Cephalalgia*. 2018;38(1):1-211. doi:10.1177/0333102417738202.

10. Goadsby PJ, Holland PR. An update: pathophysiology of migraine. *Neurol Clin*. 2019;37(4):651-671. doi:10.1016/j.ncl.2019.07.008.

11. Purdy RA, Diener H-C. Migraine mimicking stroke: What to do? *Cephalalgia*. 2018;38(14):1995-1997. doi:10.1177/0333102418768087.

12. Vilela P. Acute stroke differential diagnosis: stroke mimics. *Eur J Radiol*. 2017;96:133-144. doi:10.1016/j.ejrad.2017.05.008.

13. Sparaco M, Ciolli L, Zini A. Posterior circulation ischemic stroke-a review part II: imaging and acute treatment. *Neurol Sci*. 2019;40(10):2007-2015. doi:10.1007/s10072-019-03936-x.

14. Yahya T, Jiani MH, Khan SU, et al. Stroke in young adults: current trends, opportunities for prevention and pathways forward. *Am J Prev Cardiol*. 2020;3:100085. doi:10.1016/j.ajpc.2020.100085.

15. Mayer SA, Viarasilpa T, Panyavachiraporn N, et al. CTA-for-all: impact of emergency computed tomographic angiography for all patients with stroke presenting within 24 hours of onset. *Stroke*. 2020;51(1):331-334. doi:10.1161/STROKEAHA.119.027356.

16. American Heart Association. *Advanced Cardiac Life Support Provider Manual*. 17th ed. Dallas, TX: American Heart Association; 2021.

17. Ersoy A, Yasar H, Mertoglu C, et al. Is ischemia associated with the formation of white matter lesions in migraine? *Clin Neurol Neurosurg*. 2020;193:105770. doi:10.1016/j.clineuro.2020.105770.

18. Dodick DW. A phase-by-phase review of migraine pathophysiology. *Headache*. 2018;58(suppl 1):4-16. doi:10.1111/head.13300.

19. Amatangelo MP. Cryptogenic stroke: anatomy of the stroke work-up. *Crit Care Nurs Clin North Am*. 2020;32(1):37-50. doi:10.1016/j.cnc.2019.11.008.

20. Gil-Gouveia R, Martins IP. Cognition and cognitive impairment in migraine. *Curr Pain Headache Rep*. 2019;23(11):84. doi:10.1007/s11916-019-0824-7.

21. Oskoui M, Pringsheim T, Holle-Managan Y, et al. Practice guideline update summary: acute treatment

of migraine in children and adolescents. *Headache*. 2019;59(8):1158-1173. doi:10.1111/head.13628.

22. Keigher KM. Large vessel occlusion in the acute stroke patient: identification, treatment, and management. *Crit Care Nurs Clin North Am*. 2020;32(1):21-36. doi:10.1016/j.cnc.2019.11.007.

23. Demaerschalk B, Kleindorfer D, Adeoye O, et al. Scientific rationale for the inclusion and exclusion criteria for intravenous alteplase in acute ischemic stroke: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2017;48:e343-e361. doi:10.1161/STR.0000000000000152.

24. Yaghi S, Willey JZ, Cucchiara B, et al. Treatment and outcome of hemorrhagic transformation after intravenous alteplase in acute ischemic stroke. *Stroke*. 2017;48:e343-e361. doi:10.1161/STR.0000000000000152.

25. Jahan R, Saver JL, Schwamm LH, et al. Association between time to treatment with endovascular reperfusion therapy and outcomes in patients with acute ischemic stroke treated in clinical practice. *JAMA*. 2019;322(3):252-263. doi:10.1001/jama.2019.8286.

26. The American Headache Society position statement on integrating new migraine treatments into clinical practice. *Headache*. 2019;59(1):1-18. doi:10.1111/head.13456.

27. Murray NM, Ke M, Yee A, et al. ICU interventions in ischemic stroke patients treated using liberalized IV-tPA criteria. *J Stroke Cerebrovasc Dis*. 2019;28(9):2488-2495. doi:10.1016/j.jstrokecerebrovasdis.2019.06.015.

28. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines. *Stroke*. 2019;50(12):e344-e418. doi:10.1161/STR.0000000000000211.

29. Amatangelo MP, Thomas SB. Priority nursing interventions caring for the stroke patient. *Crit Care Nurs Clin North Am*. 2020;32(1):67-84. doi:10.1016/j.cnc.2019.11.005.

30. Orr SL, Friedman BW, Christie S, et al. Management of adults with acute migraine in the emergency department: the American Headache Society evidence assessment of parenteral pharmacotherapies. *Headache*. 2016;56(6):911-940. doi:10.1111/head.12835.

31. Jillella DV, Calder CS, Uchino K, et al. Blood pressure and hospital discharge outcomes in acute ischemic stroke patients undergoing reperfusion therapy. *J Stroke Cerebrovasc Dis*. 2020;29(11):105211.

Angela Collins-Yoder is a clinical professor at the University of Alabama's Capstone College of Nursing and a critical care clinical nurse specialist at Ascension Sacred Heart. Betty Key is an assistant professor at Samford University in Birmingham, Ala.

The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.

DOI-10.1097/01.NURSE.0000806152.65968.41

For 114 additional continuing professional development articles related to emergency care topics, go to nursingcenter.com/CE.

Lippincott® NursingCenter®

NCPD Nursing Continuing Professional Development

INSTRUCTIONS

Acute ischemic stroke or migraine with aura? Triage considerations

TEST INSTRUCTIONS

- Read the article. The test for this nursing continuing professional development (NCPD) activity is to be taken online at **www.nursingcenter.com/CE/nursing**. Tests can no longer be mailed or faxed.
- You'll need to create an account (it's free!) and log in to access My Planner before taking online tests. Your planner will keep track of all your Lippincott Professional Development online NCPD activities for you.
- There's only one correct answer for each question. A passing score for this test is 7 correct answers. If you pass, you can print your certificate of earned contact hours and access the answer key. If you fail, you have the option of taking the test again at no additional cost.
- For questions, contact Lippincott Professional Development: 1-800-787-8985.
- Registration deadline is December 6, 2024.

PROVIDER ACCREDITATION

Lippincott Professional Development will award 2.0 contact hours for this nursing continuing professional development activity.

Lippincott Professional Development is accredited as a provider of nursing continuing professional development by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 2.0 contact hours. Lippincott Professional Development is also an approved provider of continuing nursing education by the District of Columbia, Georgia, and Florida, CE Broker #50-1223. Your certificate is valid in all states.

Payment: The registration fee for this test is $21.95.