ASK THE EXPERTS

What Is Your Threshold for Treating Posterior Circulation Strokes With Thrombectomy and Why?

In the absence of clear guidelines for patients with posterior circulation strokes, our panel weighs the importance of presenting symptoms, infarct location, and other determining factors in their treatment decision-making.

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In our experience, patients who present with posterior circulation stroke are a completely different breed than those with anterior circulation stroke. Other than the presenting National Institutes of Health Stroke Scale (NIHSS) score and baseline function, three major factors—collaterals, amount of infarcted

tissue on presentation, and severity of intracranial atherosclerotic disease—help us decide whether or not to treat posterior circulation stroke patients with mechanical thrombectomy. Compared with their anterior circulation counterparts, we believe collaterals are the most common reason that mechanical thrombectomy has not yet become a proven treatment for these patients. The posterior circulation patient is less likely to have good collaterals, with approximately 25% having poor collaterals and only 36% having good collaterals.¹

Second, there is the issue of the amount of already infarcted tissue during presentation. Unfortunately, when patients present with brainstem infarction on arrival, their outcomes are very poor and there is an increased risk of mortality. Our experience with posterior circulation patients mirrors that of a recent meta-analysis.² The pooled evidence indicated that patients with posterior circulation strokes had worse functional outcomes than those with anterior circulation strokes (odds ratio [OR], 0.79; 95% confidence interval [CI], 0.63–0.98; P = .03). The successful recanalization rate was not statistically significant between the two groups (OR, 1.12; 95% CI, 0.88–1.42; P = .35).²

Finally, knowing whether or not the patient has significant intracranial atherosclerotic disease will help with treatment decision-making. When patients present with acute-on-chronic disease, the case is typically

long and difficult with multiple mechanical thrombectomy attempts, as well as intracranial angioplasty and/or intracranial stenting. This leads you down the road of starting intravenous antiplatelet agents and loading oral agents while balancing the risk of hemorrhage—and all of this while the patient might have received intravenous thrombolytics in the emergency department. One should seriously consider the recent data from the WEAVE study (evaluating the Wingspan stent system [Stryker], the only FDA-approved intracranial

stent), which showed a low ischemic event rate of 2.6% when performed by experienced operators.³ However, the complication rate significantly increased when enrolled patients were treated prior to the required 7-day waiting period.

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Clear guidelines and metrics exist for neuroendo-vascular treatment of large vessel anterior circulation strokes.¹ The same is not true for strokes in the posterior circulation, although pooled experience has led to evidence-based recommendations.² Treatment decisions are heterogeneous and often operator-dependent. I think it is important to segregate posterior circulation strokes into vertebrobasilar lesions and those in the posterior cerebral arteries (PCAs) or other smaller branches.

I have a low threshold for treating basilar or dominant vertebral artery strokes. These are life-threatening lesions that will likely result in severe disability or death if left untreated. I treat these lesions if there is salvageable tissue and a chance for meaningful recovery. I typically extend the time window for which I am willing to treat large vessel occlusions in these locations (when compared with anterior circulation strokes). My main determinant is the radiographic appearance of

the brainstem on presentation.³ If CT or MRI suggests significant, irreversible brainstem infarction, I typically defer on mechanical thrombectomy; otherwise, I am aggressive with treatment.

Although no clear standards for neuroendovascular treatment of PCA strokes exist, I usually make my treatment decisions based on the location of stroke and the presumed impact of the deficit on the presenting individual.^{4,5} If the thrombus is located in the proximal P1 segment of the PCA and the infarct is likely to affect the thalamus, I typically perform mechanical thrombectomy in a young, healthy patient. I have a higher threshold for neuroendovascular treatment when the thrombus is located in the distal PCA and will likely only affect vision (by virtue of a homonymous hemianopsia). I have treated these lesions in young patients and those who rely on their vision for their professional vocation (athlete, musician). Typically, these patients are awake and alert on presentation and can engage in an active discussion about risks and benefits. Smaller branches present unique technical challenges for mechanical thrombectomy, and stroke patients often recover well from occlusions of these branches (anterior inferior cerebellar artery, superior cerebellar artery, posterior inferior cerebellar artery) without endovascular therapy.

- 1. 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 for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association [published erratum appears in Stroke. 2019;50:e440-e441]. Stroke. 2019;50:e344-e418.
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Acute vascular occlusion in the posterior circulation is one of the most challenging clinical scenarios for both patients and physicians. Because there are so many ways symptoms can manifest, diagnosis is frequently delayed. Patients may be asymptomatic, have vague complaints such as unsteadiness, or even rapidly decline into a comatose state. As a result, the differential diagnosis can be quite broad. Early recognition is helpful, especially if patients meet the criteria for intravenous lytic therapy. Sadly, most do not. Endovascular intervention is often the only remaining option for patients with posterior circulation strokes.

Many case reports, series, and small studies have been performed in this patient population, but the 2019 guidelines for the early management of acute ischemic stroke state: "Although the benefits are uncertain, the use of mechanical thrombectomy with stent retrievers may be reasonable for carefully selected patients with [acute ischemic stroke] in whom treatment can be initiated (groin puncture) within 6 hours of symptom onset and who have causative occlusion of the anterior cerebral arteries, vertebral arteries, basilar artery, or [PCAs]."1

Given the state of the data and grave outcomes without (and sometimes even with) intervention, any decisions should be made in consultation with the patients and/or their family members. A guiding principle for our group is that we do not intervene on patients with posterior circulation strokes and absent brainstem reflexes who have been comatose for ≥ 3 hours. For patients who have been comatose for ≥ 3 hours and have some reflexes present or an unclear clinical examination, we perform an urgent diffusion-weighted MRI to determine the extent of tissue injury. Patients with small or incomplete infarcts may benefit from arterial reperfusion therapy.

For noncomatose patients with occlusion of the basilar artery, our evaluation and approach would proceed in a similar fashion to those with acute large vessel occlusion of the anterior circulation.

The anatomy of the occlusion should also be considered carefully. Attention to the vasculature is of utmost importance. Factors to consider in addition to the specific location of the occlusion include patency and flow from the circle of Willis (posterior communicating artery), any fetal anatomy, and a clear understanding of the entire vertebrobasilar system.

A discussion of vascular physiology of basilar artery occlusion could fill up an entire textbook, but a few specific cases merit mention (or debate). Intervention for occlusions at the top of the basilar artery with a low NIHSS score may be dangerous because of the risk of clot fragmentation or distal migration. With time, some patients will stabilize, whereas others will quickly decline. The timing for intervention can be difficult as well. Occlusion of the mid basilar artery is most likely a result of underlying stenosis and presents a number of clinical dilemmas. After thrombectomy, the vessel may reocclude and necessitate emergent angioplasty (with or without stenting). The risk of clot fragmentation is always present, but in this setting, traversing a critical stenosis for additional retrieval may not be possible. In some cases, mid-basilar occlusions spare other vascular territories, and ischemia is primarily to the territories supplied by perforators in that location. A thorough understanding of the supply to and from the posterior inferior cerebellar artery and anterior inferior cerebellar artery can be helpful. The P1 is sometimes the site of primary occlusion (especially after lytics), and other times, distal emboli or migration occur during the process of thrombectomy more proximally. If there is sufficient flow above and below (posterior communicating artery and basilar artery), additional intervention may not be worth the risk.

Our commitment should always be to first do no harm. Not all occlusions warrant intervention.

^{1.} 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 for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association [published erratum appears in Stroke. 2019;50:e440-e441]. Stroke. 2019;50:e344-e418.