Current endovascular strategies for posterior circulation large vessel occlusion stroke: report of the Society of NeuroInterventional Surgery Standards and Guidelines Committee

Yasha Kayan,[1] Philip M Meyers,[2] Charles J Prestigiacomo,[3] Peter Kan,[4] Justin F Fraser,[5] on Behalf of the Society of NeuroInterventional Surgery

[1]Neurointerventional Radiology, Abbott Northwestern Hospital, Minneapolis, Minnesota, USA
[2]Neurointerventional Surgery, Columbia Presbyterian Hospital, New York City, New York, USA
[3]Neurological Surgery, University of Cincinnati, Cincinnati, Ohio, USA
[4]Department of Neurosurgery, Baylor College of Medicine, Houston, Texas, USA
[5]Neurological Surgery, University of Kentucky, Lexington, Kentucky, USA

Correspondence to
Dr Justin F Fraser, Neurological Surgery, University of Kentucky, 800Rose St Room MS105A, Lexington, KY40536, USA; Jfr235@uky.edu

Received 22 February 2019
Revised 17 April 2019
Accepted 22 April 2019
Published Online First 18 May 2019

To cite: Kayan Y, Meyers PM, Prestigiacomo CJ, *et al*. *J NeuroIntervent Surg* 2019;**11**:1055–1062.

ABSTRACT

Background The aim of this publication is to provide a detailed update on the diagnosis, treatment, and endovascular techniques for posterior circulation emergent large vessel occlusion (pc-ELVO).

Methods We performed a review of the literature to specifically evaluate this disease and its treatments.

Results Data were analyzed, and recommendations were reported based on the strength of the published evidence and expert consensus.

Conclusion While many questions about pc-ELVO remain to be studied, there is evidence to support particular practices in its evaluation and treatment.

INTRODUCTION

Posterior circulation strokes account for approximately 20% of all strokes, but posterior circulation emergent large vessel occlusions (pc-ELVO) are rare, representing only 1% of all ischemic strokes, and 5% of all ELVOs.[1–3] These strokes are often devastating events. Good clinical outcomes occur in approximately 20% despite advanced care, and the rate of good clinical outcome after revascularization has been lower than for anterior circulation stroke.[4–6] A number of reasons account for this disparity. Unlike hemispheric ischemia, basilar artery occlusion can mimic other clinical conditions, resulting in delays to clinical neurological evaluation and identification.[7] There may be an extended prodrome that can potentially last days to months.[8 9] Furthermore, the neurologic 'real estate' involved in brainstem ischemia is more eloquent; a small completed infarction can be clinically devastating. Etiologies for posterior circulation ischemia include thromboembolism (often in younger age), intrinsic atherosclerosis (usually sixth/seventh decades of life), dissection with basilar embolism (often in trauma), and vasculitidies.[8 10] These factors all require consideration in the assessment of acute stroke referable to the posterior circulation.

Basilar artery occlusion (BAO) syndrome presents as a subtype of posterior circulation stroke. While there is large practice variability among interventionalists on time limits for thrombectomy in this syndrome, good clinical outcomes have been demonstrated up to 50 hours from last known well.[11] Stroke severity at presentation is an independent predictor of patient outcome.[12] However, good outcomes appear to depend, at least in part, on revascularization of the occlusion.[13 14] Lindsberg *et al* report good clinical outcomes with recanalization in 38% versus 2% without recanalization.[15] From the Helsinki Stroke Thrombolysis Registry, Sairanen *et al* report good clinical outcomes with recanalization in 86% versus 14% without recanalization.[7] Similarly, Kumar *et al* report 1.5 times decreased dependency and two times decreased mortality with recanalization than without it.[14]

With these data in mind, the aims of this document include: (1) to review data on the natural history and outcomes from pc-ELVO and (2) to provide guidelines and recommendations on acute intervention regarding pc-ELVO (figure 1). Recommendations follow the American College of Cardiology/American Heart Association (ACC/AHA) classification of recommendation and level of evidence.[16]

DIAGNOSIS

Clinical presentation

The clinical presentation of posterior circulation strokes can be non-specific. Common symptoms can include loss of consciousness, headache, nausea, vomiting, dizziness, double vision, hearing loss, slurred speech, vertigo, imbalance, and unilateral extremity weakness. Physical examination may also commonly reveal ataxia, nystagmus, and visual field defects.[17]BAO vary based on thrombus location. Top of the basilar syndrome includes somnolence, peduncular hallucinosis, convergence nystagmus, skew deviation, oscillatory eye movements, retraction and elevation of the eyelids, and vertical gaze paralysis. Mid-basilar occlusions can result in the various pontine syndromes. Proximal basilar occlusions can result in 'locked in' syndrome.[17]

Prehospital ELVO scales

Recent prehospital stroke scales were designed for anterior circulation strokes. In the evaluation of the Los Angeles Motor Scale, sensitivity and specificity in 119 patients to detect ELVO were 81% and 89%, respectively, but excluded posterior circulation strokes.[18]Rapid Arterial Occlusion Evaluation was found to have a similar ELVO sensitivity and specificity (85% and 65%, respectively) in a cohort of 357 patients, but only 7% had posterior circulation stroke.[19] While it has recently been shown that adding the finger to nose test to the Cincinnati Prehospital Stroke Scale significantly increased

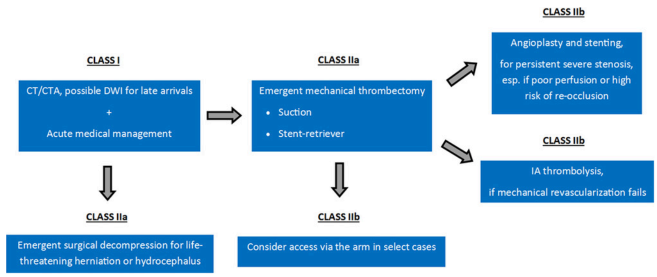

Figure 1 Flow chart illustrating the workflow from the summary of recommendations. CTA, CT angiography; DWI, diffusion weighted imaging; IA, intra-arterial.

the recognition of posterior circulation strokes by paramedics in a cohort of 789 consecutive ischemic stroke cases, pc-ELVO remains difficult to detect in the field.[20]

Limitations of the NIHSS in pc-ELVO

Limitations of the National Institutes of Health Stroke Scale (NIHSS) include a focus on limb and speech impairments and less emphasis on cranial nerve lesions.[21] In a prospective cohort of 310 stroke patients not treated with thrombolysis, baseline NIHSS cut-off for a favorable 3 month outcome (modified Rankin Scale (mRS) score of ≤2) was significantly lower in patients with posterior circulation stroke compared with anterior circulation stroke.[22] Indeed, patients with pc-ELVO can have an NIHSS score of 0, where symptoms may be headache, vertigo, and nausea only. The most common neurological sign in a series of 20 NIHSS 0, diffusion weighted imaging (DWI) positive patients was truncal ataxia.[23] Therefore, the physician must maintain a high index of clinical suspicion for this condition in the face of subtle symptoms.

Clinical confounders and differential diagnosis

Posterior circulation strokes presenting to an emergency department may be associated with a delay in neurology evaluation, and door to needle time for intravenous tissue plasminogen activator (IV tPA) is often significantly longer compared with anterior circulation strokes. This is likely related to difficulty in timely recognition due to non-specific symptoms.[7] The cause of BAO may not be immediately apparent on imaging. The differential diagnosis for etiology includes thromboembolism, cardiogenic or artery to artery embolism, or in situ thrombosis of vertebrobasilar stenosis due to atherosclerotic disease or, more rarely, dissection.

In a Korean series of 62 patients with acute basilar occlusion who underwent stent retriever thrombectomy, 15 (24.1%) also underwent intracranial angioplasty and stenting for an underlying atherosclerotic stenosis. Proximal basilar occlusions were more likely to be related to atherosclerosis, and distal occlusions were more likely thromboembolic. Bilateral thalamic infarction on pretreatment DWI was less common in patients with atherosclerotic stenosis (0% vs 27.7%, P=0.027). Otherwise, patients with and without underlying atherosclerotic stenosis who underwent endovascular therapy had similar outcomes.[24]

Natural history and outcomes with medical treatment

Initially described in 1946, BAO was considered universally fatal.[25] While there are limited data on BAO outcomes prior to the era of thrombolysis, data from 82 patients at three centers reported in 2005 show a case fatality of 40% and a poor outcome in almost 80% of patients.[@@@] Zeumer *et al* first described successful basilar artery thrombolysis in 1982, and Hacke *et al* reported the clinical outcome benefit of revascularization in a series of 65 patients 6 years later in 1988.[26 27] Advancement in non-invasive imaging permitted understanding of variances in outcome. The New England Stroke Registry showed that 29% of patients with posterior circulation strokes died or suffered severe disability; however, individuals with embolic strokes fared worse than those with in situ atherosclerosis.[8] However, reported mortality ranges from 45% to 86%.[4 15 28] Predictors of poor outcome include older age, higher NIHSS score, lack of recanalization, atrial fibrillation, intracranial hemorrhage, and posterior circulation Alberta Stroke Program Early CT Score (pc-ASPECTS) ≤8.[29]

As with anterior circulation strokes, hemorrhagic conversion is related to the overall volume of the infarction, use of anticoagulants (eg, heparin) in the peri-ictal setting, and administration of fibrinolytics. In a small study that reviewed 37 patients with posterior circulation strokes who were administered heparin, 11% developed hemorrhagic conversions. All patients who worsened sustained a proximal to mid-basilar artery occlusion and cerebellar infarction.[30] In a retrospective review of almost 1000 patients in the TIMS-China registry, multivariate analysis suggested a lower 3.2% incidence of symptomatic hemorrhagic conversion in 124 patients with posterior circulation strokes compared with 7.7% in patients with anterior circulation strokes.[31] Moreover, a higher proportion of functional independence was noted after hemorrhagic conversion in patients with posterior circulation strokes when compared with patients with anterior circulation strokes (64% vs 53%, OR 2.33, 95% CI 1.40 to 3.89).

IMAGING

Computed tomography

Non-contrast CT is used to exclude hemorrhage and evaluate for other intracranial abnormalities, including life-threatening edema and mass effect in the posterior fossa. Beam hardening artifact often limits assessment of structures in the posterior fossa but vessel hyperdensity, particularly a hyperdense basilar artery, is sometimes present.[32] In a series of 95 patients with posterior circulation infarction who underwent CT brain scan with CT angiography (CTA), 14 had partial or complete BAO. Of the 14, 10 (83%) had a hyderdense basilar artery on non-contrast CT. A hyperdense basilar artery was a significant independent predictor of a poor outcome at 6 months (mRS >2, OR 5.6, 95% CI 1.1 to 33.3). A hyperdense basilar artery had a sensitivity of 71% and specificity of 98% as a predictor of basilar occlusion.[33] Diagnostic accuracy may be improved by measuring vessel density (optimal cut-off is 40–42 Hounsfield units).[34]

CTA has become the primary method used to identify large vessel occlusion in most stroke patients.[35–39] Because posterior circulation strokes were excluded from the major randomized controlled trials for thrombectomy, the evidence for CTA in the posterior circulation is limited but the logic remains compelling. In addition to identifying the site of occlusion, it can have prognostic significance. In a retrospective review of 15 patients with vertebrobasilar stroke treated with thrombectomy, a 6 point posterior circulation CTA vascular collateral score as well as patency of the distal third of the basilar artery correlated with good outcomes, defined by mRS score of ≤3 at 3 months.[40] Several studies, including a series of 21 patients and another of 104 patients, found that the presence of bilateral posterior communicating arteries on pretreatment CTA was associated with more favorable outcomes after endovascular recanalization.[41 42] In the Basilar Artery International Cooperation Study

(BASICS) study, evaluation of comatose patients with basilar occlusions using CTA showed the extent of brainstem ischemia on source images was related to mortality and poor outcome.[43] The use of CTA in patients with possible BAO has also been found to be cost effective.[44]

Historically, the use of CT perfusion (CTP) in the posterior circulation has been of limited value. However, the prospective, multicenter, observational Dutch Acute Stroke Study (DUST) of 88 patients with suspected posterior circulation stroke found that CTP adds additional diagnostic value, primarily in the form of higher sensitivity (74%) and negative predictive value (80%) compared with non-contrast CT (31% and 61%, respectively) and CTA (33% and 62%, respectively). Large vessel occlusions were not specifically evaluated.[45] Another study found no difference between detection of infratentorial and supratentorial lesions using CTP; however the duration of stroke onset to imaging was long (540 min), likely favoring identification of ischemic changes, and thus sensitivity, with non-enhanced CT alone.[46] In BASICS, a small minority of patients were evaluated using CTA and CTP (27 of 592, 4.6%). Mean transit time was abnormal in 93%. Cerebral blood volume correlated with a risk of death. For three patients with pc-ASPECTS <8, all had died at 1 month compared with 6 of 23 patients with a cerebral blood volume pc-ASPECTS ≥8 (RR 3.8, 95% CI 1.9 to 7.6).[47]

Magnetic resonance imaging

In ENDOSTROKE, an observational registry that included 148 consecutive patients with basilar occlusion who underwent mechanical thrombectomy, the use of MRI prior to intervention was an independent predictor of good clinical outcome.[6] Spin-echo MRI can be used to evaluate for potential edema and mass effect in the posterior fossa, although non-contrast CT is more readily obtained at most medical centers in the acute setting. The extent of signal hyperintensity of the basilar artery on FLAIR imaging has been associated with mortality in a retrospective review of 20 basilar occlusion patients.[48] DWI may be the most sensitive test for evaluation of ischemia but can initially be normal in 6–10% of cases, twice as often as DWI in the anterior circulation.[49–51]

pc-ASPECTS, originally described using CTA source images,[52] may also be calculated using DWI. For reference, pc-ASPECTS assigns 10 points to the entire posterior circulation, with 1 point each subtracted for early ischemic change in the right or left thalamus, cerebellum, or posterior cerebral artery territories (total 6 points), and 2 points each for early ischemic change in any part of the midbrain or pons (total 4 points). The extent of restricted diffusion used to calculate pc-ASPECTS was an independent predictor of unfavorable outcome (mRS ≥3, OR 0.40, 95% CI 0.23 to 0.67) in a retrospective study of 132 patients.[52,53] Nagel *et al* reported a retrospective study of 50 patients with acute basilar occlusion, showing that the DWI pc-ASPECTS ≥8 was the only independent predictor for favorable outcome (OR 3.9, 95% CI 1.4 to 11.7).[54] In 35 patients who underwent mechanical thrombectomy for basilar occlusion using MRI based selection criteria, DWI pc-ASPECTS was significantly different between those patients with a good outcome (mRS 0–2) compared with patients who suffered a poor outcome (7.8±1.6 vs 5.4±1.8, P=0.001).[55] DWI pc-ASPECTS has also been found to be useful in predicting outcome in patients with acutely symptomatic basilar stenosis and not occlusion.[56]

There is little in the literature specifically looking at MR perfusion in the posterior circulation. In five patients with acute basilar occlusion receiving intra-arterial thrombolytics, significant diffusion–perfusion mismatch (mean 73%, range 49–99%) was visualized in all patients with pretreatment imaging, suggesting the presence of salvageable tissue. Final infarction volumes were smaller than pretreatment perfusion volumes, suggesting tissue salvaged by thrombolysis.[57]

CT and CTA are recommended in the evaluation of patients with suspected pc-ELVO, with possible DWI for late arrivals, as institutionally defined, to exclude those with a poor pc-ASPECTS or large core infarction (AHA Class I, Level of Evidence C-LD).

ACUTE PATIENT CARE

Guidelines applicable to ischemic strokes of the anterior circulation apply when managing patients with pc-ELVO.[58] As in any acute care setting, careful and frequent assessment of the patient's airway, breathing, and circulation is required, particularly given the potential fluctuating nature of posterior circulation strokes. Due to the brain territory involved, patients with pc-ELVO are at high risk for loss of consciousness, airway compromise, respiratory depression, and autonomic instability.

Respiratory

In a series of 153 patients with stroke complicated by hypoxemia, Rowat *et al* demonstrated increased mortality and a greater risk of post-stroke hypoxemia.[59] Loss of sensation to the pharynx and hypopharynx, along with reduction in reflexes, increases the risk of airway compromise.[60] Central breathing abnormalities and associated hypoxia have also been shown to be frequent complications of posterior circulation stroke.[61]

Given poorer outcomes in stroke patients with hypoxia, administration of supplemental oxygen to maintain oxygen saturation above 94% may be useful.[58] While the least invasive route for oxygen supplementation is preferred, in the setting of potential airway compromise or focal brainstem related deficits, endotracheal intubation and ventilation is warranted. Given the aspiration risk, this may reduce the number of hospital acquired pneumonias and potentially aid in the management of intracranial pressure.[62,63] Unfortunately, it is important to note that the outcome for patients who undergo intubation is poor.[64]

Although there is some evidence that patients with anterior circulation strokes and no evidence of hypoxia can benefit from being placed in the supine or decubitus position, the high risk of aspiration in patients with pc-ELVO would preclude placing the patient in the supine position.[65] Thus in patients with a risk of elevated intracranila pressure (ICP) or aspiration risk, the head of the bed should be elevated to between 15 and 30°.[66]

Cardiovascular

Posterior circulation ELVO patients should have frequent, if not continuous, blood pressure monitoring as it serves as a potential indicator of imminent ICP elevation. There is a delicate balance between blood pressure adequate to perfuse the brainstem and increased cardiac work resulting in myocardial ischemia or elevated ICP. Studies have demonstrated improved outcomes in patients whose systolic blood pressure ranged from 121 to 200 mm Hg and diastolic blood pressures ranging from 81 to 110 mm Hg.[67–69] The current recommendation not to lower blood pressure within the first 24 hours of a stroke unless it exceeds 220/120 mm Hg or there are concomitant medical indications to do so, is reasonable.[58]

Endocrinology

Hyperglycemia is common in the acute ischemic stroke population, with some studies reporting an elevated admission blood glucose level in >40%.[70] There are no data with regards to hyperglycemia specifically in posterior circulation stroke. Poor clinical outcomes have been reported in patients with elevated blood glucose levels in the setting of symptomatic intracerebral hemorrhage after intravenous fibrinolysis using recombinant tPA, and increase in infarction volume as documented on MRI.[70] A randomized trial to evaluate the effects of hyperglycemia in stroke failed to reach a conclusion as the trial was stopped early.[71] At present, it is recommended that blood sugar be maintained between 140 and 180 mg/dL in accordance with the American Diabetic Association guidelines for hospitalized patients. It is also suggested that subcutaneous insulin protocols can be used safely and effectively in patients with stroke without the need for intravenous insulin protocols.[58]

Fever

No specific data exist for the incidence of hyperthermia (>37.6°C) in patients presenting with posterior circulation stroke.[72] However, hyperthermia in the setting of acute stroke has been associated with poor neurological outcome.[73] A recent meta-analysis demonstrated a twofold increase in short term mortality in hyperthermic patients within 24 hours of ictus.[74] Administration of aspirin or acetaminophen can achieve normothermia in patients presenting with temperatures <38°C.[75] However, hypothermia protocols in the setting of pc-ELVO as a neuroprotectant have not been assessed.

General guidelines for the acute medical management of patients with ischemic stroke also apply to the subgroup of pc-ELVO patients (AHA Class I, Level of Evidence C-LD).

Surgical considerations

The most significant surgical concern in patients presenting with pc-ELVO is the immediate and delayed risk of herniation secondary to cerebellar infarction or hemorrhage. The small volume of the posterior fossa and the eloquence of brainstem structures contribute to the risk of catastrophic herniation and fulminant clinical deterioration, respectively. Mass effect on the brainstem by an infarcted cerebellar hemisphere or non-communicating hydrocephalus secondary to occlusion of the cerebral aqueduct may also result in a fluctuating neurological examination and require repeat imaging.

Although mass effect can peak on the third day post-infarct, it has been reported to occur throughout the first week. Approximately 25% of patients will develop mass effect causing rapid clinical deterioration.[76 77] Ventricular or aqueductal obstruction causes hydrocephalus in up to 20% of patients with cerebellar stroke.[76] Thus it is imperative to identify and aggressively treat patients with evidence of mass effect and hydrocephalus, as 85% of patients progressing to coma die without intervention.[78]

Conservative measures such as elevating the head of bed, the use of osmotic diuretics, and hyperventilation provide only transient benefit. There is no consensus on the treatment of patients with hydrocephalus in this setting. Placement of an external ventricular drain alone carries a risk of upward herniation as well as continued mass effect on the brainstem.[78] Thus suboccipital craniectomy, durotomy, and duraplasty to reduce the mass effect on the brainstem and restore CSF drainage should be considered early for these patients. Half of patients progressing to coma treated with suboccipital decompression have good outcomes. It has been demonstrated in several independent case series that rapid surgical intervention in the setting of acute clinical deterioration can improve outcome.[79]Randomized controlled trials addressing surgery in this patient population are unlikely to be performed due to the potential for herniation in a control group. In a rare prospective study on this topic, preoperative Glasgow Coma Scale was a major predictor of good clinical outcome.[80] As such, early neurosurgical consultation should be sought for surgical decompression *before clinical deterioration occurs.*

Emergent surgical decompression in patients with cerebellar infarct exhibiting mass effect with life threatening sequela is reasonable(AHA Class IIa, Level of Evidence C-LD).

ENDOVASCULAR MANAGEMENT

Mechanical revascularization

There are no randomized controlled trials of mechanical thrombectomy for acute stroke due to BAO. A retrospective analysis of 34 consecutive patients undergoing thrombectomy for acute BAO showed that the Solitaire stent retriever and the ACE reperfusion catheters had a higher recanalization rate (92%) compared with older devices (24%). The procedures were also faster with the newer devices (88±31 min vs 126±58 min).[81] Successful recanalization has been found not to depend on thrombus length and can be achieved despite a high clot burden.[82] While mechanical thrombectomy in the basilar artery has been associated with more procedural complications than in anterior circulation strokes,[83] failure of successful recanalization was a strong predictor of mortality in a prospective registry of 117 consecutive patients with BAO treated with either stent retrievers or large bore distal aspiration catheters. In the setting of recanalization, a lower pc-ASPECTS (OR 1.71, 95% CI 1.19 to 2.44) and angioplasty/stenting of the basilar artery (OR 4.71, 95% CI 1.34 to 16.54) were independent predictors of mortality after thrombectomy.[84] A meta-analysis of 31 studies summarizing 1358 patients with basilar occlusions treated with fibrinolysis (either intravenous or intra-arterial recombinant tPA) and/or mechanical thrombectomy found data insufficient to generate evidence based medical recommendations; however, the mechanical thrombectomy group had a significantly higher rate of independence (mRS 0–2) than fibrinolysis without mechanical thrombectomy at 90 days (P<0.001).[85]

Emergent mechanical thrombectomy is reasonable for pc-ELVO patients in order to maximize the chance of a good clinical outcome (AHA Class IIa, Level of Evidence B-NR).

Suction thrombectomy

A retrospective review of 436 acute ischemic stroke patients who underwent mechanical thrombectomy using A Direct Aspiration First Pass Technique (ADAPT[86]) included 57 (13%) patients with vertebrobasilar occlusions. The posterior circulation group had a similar likelihood of good outcome compared with the anterior group (42.9% vs 43.2%, respectively).[87] In a prospective study of 100 patients with BAO treated with mechanical thrombectomy, successful reperfusion was a strong predictor of favorable outcome at 90 days (OR 4.57, 95% CI 1.24 to 16.87, P=0.023). Of the 100 patients, 46 patients were treated using ADAPT and 54 patients primarily with stent retrievers. ADAPT achieved a significantly higher rate of complete reperfusion (OR 2.59, 95% CI 1.14 to 5.86, P=0.021) with shorter mean procedure duration (median 45 min, IQR 34 to 62 min vs 56 min, IQR 40 to 90 min; P=0.05). The rate of periprocedural complications was also lower with ADAPT (4.3% vs 25.9%, P=0.003).[84]

Suction thrombectomy is a reasonable form of mechanical thrombectomy for the emergent treatment of pc-ELVO patients (AHA Class IIa, Level of Evidence B-NR).

Stent retriever thrombectomy

Several retrospective series have examined the use of stent retrievers to treat BAO. A retrospective review of 161 consecutive stent retriever thrombectomy patients comparing 24 basilar occlusions with 137 anterior circulation occlusions found a significantly higher 90 day mRS score in the basilar group (4.2 vs 3.0, P=0.003). Mortality was also higher in the basilar group (16.6% vs 5.8%, P=0.001). Nonetheless, among the patients with BAO, successful recanalization was associated with a more favorable mRS at 90 days (P=0.027).[88] In 50 consecutive patients who underwent stent retriever thrombectomy for acute basilar occlusion, multivariate analysis of factors potentially related to prognosis found that only low initial NIHSS score (OR 0.82, 95% CI 0.709 to 0.949, P=0.008) and high DWI pc-ASPECTS (OR 1.854, 95% CI 1.012 to 3.397, P=0.045) were significant independent predictors of good outcome (mRS 0–2) at 90 days[12]. In 69 patients treated with Solitaire AB thrombectomy for acute posterior circulation stroke caused by large intracranial vessel occlusion, stroke subtype (intracranial atherosclerotic disease vs embolism, OR 0.101, 95% CI 0.020 to 0.501, P=0.005) and pc-ASPECTS on DWI (≥6 vs <6, OR 7.335, 95% CI 1.495 to 36.191, P=0.014) were independent predictors of good clinical outcome at 90 days.[89]

Stent retriever thrombectomy is a reasonable form of mechanical thrombectomy for the emergent treatment of pc-ELVO patients (AHA Class IIa, Level of Evidence B-NR).

Angioplasty and stent angioplasty

In situ thrombosis of an underlying atherosclerotic stenosis is the cause of BAO in some patients, most commonly in Asian populations. For symptomatic intracranial stenosis due to atherosclerotic disease, both Stenting versus Aggressive Medical Therapy for Intracranial Arterial Stenosis (SAMMPRIS) and Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSYLVIA) trials failed to show superior results with stent angioplasty compared with medical therapy alone.[90 91] However, these studies looked at secondary stroke prevention in typically less acute settings. For acute BAO presenting with severe neurological deficits, the risk of major morbidity and death are high, and angioplasty and stenting of an associated plaque may be lifesaving. This is particularly true when there is flow limitation or thrombectomy related endothelial injury resulting in poor reperfusion or a high risk of re-occlusion.[92]

Coronary and intracranial balloon catheters and stents have been applied anecdotally to the treatment of refractory acute cerebral artery occlusion with stroke with some success since 1987.[16] In a series of 13 patients at a single Chinese center who underwent combined mechanical thrombectomy with angioplasty and stenting, 46% had a good functional outcome, with an mRS score of at least 2 at 90 days.[92] In the anterior circulation, for patients with acute atherosclerotic occlusion, it has even been suggested that primary angioplasty and stenting may be superior to thrombectomy.[93] Both intracranial angioplasty/stenting and intra-arterial infusion of a glycoprotein IIb/IIIa inhibitor were found to be safe and effective in treating underlying severe atherosclerotic stenosis in acute stroke patients with ELVO in a series of 140 patients in Korea, although only 1 in 5 patients in this study had basilar occlusions.[94] Also, a higher stroke complication rate has been noted in angioplasty and stenting of perforator bearing arteries in the posterior circulation versus *the anterior circulation.*[95]

Angioplasty and stenting may be considered for pc-ELVO if there is a persistent severe stenosis following thrombectomy, particularly if there is poor reperfusion or a perceived high risk of re-occlusion(AHA Class IIb, Level of Evidence C-EO).

Intra-arterial fibrinolysis

Case series and the available trial data point to the benefits of revascularization in appropriately selected patients, but selection of patient likely to benefit remains a challenge. Prior to 2006, all reports of basilar recanalization were case series. The Australian Urokinase Trial enrolled 16 patients with BAO but was halted prematurely, and showed no overall treatment benefit, although recanalization was associated with good outcome (38% vs 2%).[96] Meta-analysis of uncontrolled studies showed no benefit to treatment.[15] In Interventional Management of Stroke 3 (IMS-3), five patients enrolled had vertebrobasilar occlusions, but there were no outcome differences in this small group.[97] In the ENDOSTROKE multicenter registry, 148 consecutive patients with BAO were enrolled. By this time, stent retrievers were available for stroke treatment. Thirty per cent received some form of intra-arterial fibrinolysis in combination with mechanical methods. In addition, 84% of patients underwent thrombectomy using first generation thrombectomy devices, including the concentric MERCI retriever or Penumbra suction catheter. Overall, 34% achieved good outcome. The mortality rate was 35%. Lower NIHSS predicted good outcome, but revascularization did not. Thrombolysis in Cerebral Infarction 2b–3 recanalization was achieved in 79% but did not predict good outcome.[6] Similarly, the BASICS study did not identify the superiority of intra-arterial treatment over intravenous fibrinolysis in the 619 patients entered into the registry.[5]

Subsequently, between 2006 and 2015, the BASICS trial enrolled 38 consecutive patients with BAO to intra-arterial treatment; 71% also received intravenous fibrinolysis before intra-arterial therapy. Mechanical thrombectomy was performed in 30 patients (84%) while 7 patients (18%) received intra-arterial urokinase without thrombectomy. Adequate recanalization was achieved in 34 patients (89%) while good clinical outcome occurred in only 19 patients (50%). There was no association between good outcome and patent collateral circulation in the BASICS trial. Symptomatic intracranial hemorrhage occurred in two patient (5%). The authors concluded that the rate of good outcomes is comparable with the intra-arterial treatment group in the Multicenter Randomized Clinical trial of Endovascular treatment for Acute ischemic stroke in the Netherlands (MR CLEAN) *trial.*[98]

Intra-arterial pharmacologic thrombolysis may be considered for pc-ELVO if mechanical revascularization fails(AHA Class IIb, Level of Evidence C-LD).

Most commonly, cerebrovascular procedures, including mechanical thrombectomy, are performed using a transfemoral approach. Recently, a series of nine cases of basilar thrombectomy from a radial approach were reported with technical success, defined as Thrombolysis in Cerebral Infarction 2b or 3, achieved in 89% of cases. Average puncture to revascularization time was 35.8 min. There were no complications related to radial artery catheterization. The authors advocate for radial access as a firstline approach for basilar thrombectomy given the anatomical advantages.[99] There is little in the literature specifically for brachial access in the setting of pc-ELVO.[100] Disadvantages of transbrachial or transradial access include the theoretical possibility of having to use smaller catheters as well as less operator familiarity with the technique in an acute setting.

For the endovascular management of pc-ELVO, transbrachial or transradial access may be considered as an alternative to the traditional transfemoral route given anatomic advantages(AHA Class IIb, Level of Evidence C-EO).

SUMMARY OF RECOMMENDATIONS

- CT and CTA are recommended in the evaluation of patients with suspected pc-ELVO, with possible DWI for late arrivals, as institutionally defined, to exclude those with a poor pc-ASPECTS or large core infarction *(AHA Class I, Level of Evidence C-LD)*.
- General guidelines for the acute medical management of patients with ischemic stroke also apply to the subgroup of pc-ELVO patients *(AHA Class I, Level of Evidence C-LD)*.
- Early surgical decompression in patients with cerebellar infarct exhibiting mass effect with life threatening sequela is reasonable *(AHA Class IIa, Level of Evidence C-LD)*.
- Emergent mechanical thrombectomy is reasonable for pc-ELVO patients in order to maximize the chance of a good clinical outcome *(AHA Class IIa, Level of Evidence B-NR)*.
- Suction thrombectomy is a reasonable form of mechanical thrombectomy for the emergent treatment of pc-ELVO patients *(AHA Class IIa, Level of Evidence B-NR)*.
- Stent retriever thrombectomy is a reasonable form of mechanical thrombectomy for the emergent treatment of pc-ELVO patients *(AHA Class IIa, Level of Evidence B-NR)*.
- Angioplasty and stenting may be considered for pc-ELVO if there is a persistent severe stenosis following thrombectomy, particularly if there is poor reperfusion or a perceived high risk of re-occlusion *(AHA Class IIb, Level of Evidence C-EO)*.
- Intra-arterial pharmacologic thrombolysis may be considered for pc-ELVO if mechanical revascularization fails *(AHA Class IIb, Level of Evidence C-LD)*.
- For the endovascular management of pc-ELVO, transbrachial or transradial access may be considered as an alternative to the traditional transfemoral route given anatomic advantages *(AHA Class IIb, Level of Evidence C-EO)*.

Collaborators A S Arthur, S Ansari, B Baxter, K Bulsara, G Dabus, K Fargen, D Frei, C D Gandhi, S Hetts, M S Hussain, M V Jayaraman, R P Klucznik, S K Lee, W J Mack, R McTaggart, T Leslie-Mazwi, J Milburn, M Mokin, J Mocco, A T Patsalides, G L Pride, R M Starke, P J Sunenshine, G Toth, Michael Chen.

Contributors YK, PMM, and CJP were the primary authors and, as such, were responsible for overseeing construction of the document outline, gathering of evidence, and drafting of the manuscript. PK acted as a representative of the Board of the Society of NeuroInterventional Surgery, and provided commentary and editing on behalf of the Board and of the Society as a whole. JFF, as senior author, was responsible for organizing the writing group, overseeing the outline construction, facilitating communication between the writing group and the Standards and Guidelines Committee, drafting and editing of the manuscript, and the submission/proofing for publication.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial, or not-for-profit sectors.

Disclaimer This literature review ('Review') is provided for informational and educational purposes only. Adherence to any recommendations included in this review will not ensure successful treatment in every situation. Furthermore, the recommendations contained in this review should not be interpreted as setting a standard of care, or be deemed inclusive of all proper methods of care nor exclusive of other methods of care reasonably directed to obtaining the same results. The ultimate judgment regarding the propriety of any specific therapy must be made by the physician and the patient in light of all the circumstances presented by the individual patient, and the known variability and biological behavior of the medical condition. This review and its conclusions and recommendations reflect the best available information at the time the review was prepared. The results of future studies may require revisions to the recommendations in this review to reflect new data. SNIS does not warrant the accuracy or completeness of the review and assumes no responsibility for any injury or damage to persons or property arising out of or related to any use of this review or for any errors or omissions.

Competing interests JFF is an equity interest holder for Fawkes Biotechnology, LLC, and a consultant for Stream Biomedical and Medtronic Neurovascular. PK is a consultant for Stryker and Medtronic Neurovascular. YK is a consultant for Penumbra and for Medtronic Neurovascular. PMM is a consultant for Stryker, Medtronic, Penumbra, and Siemens. A S Arthur is a consultant for Balt, Johnson and Johnson, Leica, Medtronic, Microvention, Penumbra, Scientia, Siemens, and Stryker. He has research support from Cerenovus, Microvention, Penumbra, and Siemens. He is a shareholder in Bendit, Cerebrotech, Endostream, Magneto, Marblehead, Neurogami, Serenity, Synchron, Triad Medical, and Vascular Simulations. B Baxter is a consultant for Penumbra, Medtronic, Stryker, Metactive, and 880 Medical. He has stock in Penumbra, and stock options in Viz.ai. He has ownership interest in Route 92 and Marblehead. Dr Chen is a consultant for Medtronic, Stryker, Genentech, Imperative Health, Microvention, and Cerenovus. G Dabus is a consultant for Medtronic, Microvention, Penumbra, Stryker, and Cerenovus. He is a shareholder in InNeuroCo. K Fargen is a consultant for Cerebrotech. D Frei is a consultant for Genentech, Penumbra, and Stryker. He has stock ownership in Penumbra. S Hetts has a Royalty Agreement with Penumbra. He contracts for Core Imaging Lab for Stryker and MicroVention. He has a research contract with Siemens. He is on Data Safety and Monitoring Boards for DAWN, ARISE2, and PHIL trials. He has grant support through NCI and NIBIB. M S Hussain is a consultant for Cerenovus. M V Jayaraman is a speaker for Medtronic. R P Klucznik is a consultant for Medtronic and Microvention. W J Mack is a consultant for Rebound Therapeutics, Viseon TSP, Medtronic, Penumbra, and Stream Biomedical. He is an investor in Cerebrotech, Endostream, Viseon, and Rebound Therapeutics. R McTaggart is a consultant for Stryker. J Milburn is a consultant for Stryker and Penumbra. M Mokin is a consultant for Canon Medical, Cerebrotech, and Imperative Care. J Mocco receives research support from Stryker, Penumbra, Medtronic, and Microvention. He is a consultant for Imperative Care, Cerebrotech, Viseon, Endostream, Rebound Therapeutics, and Vastrax. He is an investor/shareholder in BlinkTBI, Serenity, NTI, Neurvana, and Cardinal Consulting. G L Pride is a consultant for Sequent and Microvention-Terumo. He is on the Data Safety and Monitoring Board for Web-IT Study (Cerenovus) and for the Pulserider NAPA trial. R M Starke receives research support from NREF, Joe Niekro Foundation, Brain Aneurysm Foundation, Bee Foundation, NIH, Miami Clinical and Translational Science Institute, National Center for Advancing Translational Sciences, and the National Institute on Minority Health and Health Disparities. He is a consultant for Penumbra, Abbott, Medtronic, and Cerenovus. P J Sunenshine is a consultant for Medtronic. G Toth is a consultant for Dynamed EBSCO.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Greving JP, Schonewille WJ, Wijman CA, *et al*. Predicting outcome after acute basilar artery occlusion based on admission characteristics. *Neurology* 2012;78:1058–63.
2 Mattle HP, Arnold M, Lindsberg PJ, *et al*. Basilar artery occlusion. *Lancet Neurol* 2011;10:1002–14.
3 Smith WS, Lev MH, English JD, *et al*. Significance of large vessel intracranial occlusion causing acute ischemic stroke and TIA. *Stroke* 2009;40:3834–40.
4 Schonewille WJ, Algra A, Serena J, *et al*. Outcome in patients with basilar artery occlusion treated conventionally. *J Neurol Neurosurg Psychiatry* 2005;76:1238–41.
5 Schonewille WJ, Wijman CA, Michel P, *et al*. Treatment and outcomes of acute basilar artery occlusion in the Basilar Artery International Cooperation Study (BASICS): a prospective registry study. *Lancet Neurol* 2009;8:724–30.
6 Singer OC, Berkefeld J, Nolte CH, *et al*. Mechanical recanalization in basilar artery occlusion: the ENDOSTROKE study. *Ann Neurol* 2015;77:415–24.
7 Sarraj A, Medrek S, Albright K, *et al*. Posterior circulation stroke is associated with prolonged door-to-needle time. *Int J Stroke* 2015;10:672–8.
8 Voetsch B, DeWitt LD, Pessin MS, *et al*. Basilar artery occlusive disease in the New England Medical Center Posterior Circulation Registry. *Arch Neurol* 2004;61:496–504.
9 Ferbert A, Brückmann H, Drummen R. Clinical features of proven basilar artery occlusion. *Stroke* 1990;21:1135–42.
10 Demel SL, Broderick JP. Basilar occlusion syndromes: an update. *Neurohospitalist* 2015;5:142–50.
11 Grigoriadis S, Gomori JM, Grigoriadis N, *et al*. Clinically successful late recanalization of basilar artery occlusion in childhood: what are the odds? Case report and review of the literature. *J Neurol Sci* 2007;260:256–60.
12 Yoon W, Kim SK, Heo TW, *et al*. Predictors of good outcome after stent-retriever thrombectomy in acute basilar artery occlusion. *Stroke* 2015;46:2972–5.
13 Bouslama M, Haussen DC, Aghaebrahim A, *et al*. Predictors of good outcome after endovascular therapy for vertebrobasilar occlusion stroke. *Stroke* 2017;48:3252–7.
14 Kumar G, Shahripour RB, Alexandrov AV. Recanalization of acute basilar artery occlusion improves outcomes: a meta-analysis. *J Neurointerv Surg* 2015;7:868–74.
15 Lindsberg PJ, Mattle HP. Therapy of basilar artery occlusion: a systematic analysis comparing intra-arterial and intravenous thrombolysis. *Stroke* 2006;37:922–8.
16 Brückmann HJ, Ringelstein EB, Buchner H, *et al*. Vascular recanalizing techniques in the hind brain circulation. *Neurosurg Rev* 1987;10:197–9.
17 Nouh A, Remke J, Ruland S. Ischemic posterior circulation stroke: a review of anatomy, clinical presentations, diagnosis, and current management. *Front Neurol* 2014;5:30.

18 Nazliel B, Starkman S, Liebeskind DS, *et al*. A brief prehospital stroke severity scale identifies ischemic stroke patients harboring persisting large arterial occlusions. *Stroke* 2008;39:2264–7.
19 Pérez de la Ossa N, Carrera D, Gorchs M, *et al*. Design and validation of a prehospital stroke scale to predict large arterial occlusion: the rapid arterial occlusion evaluation scale. *Stroke* 2014;45:87–91.
20 Oostema JA, Chassee T, Edberg A, *et al*. Abstract TMP69: Educating paramedics on the "finger-to-nose' test improves recognition of posterior stroke. *Stroke* 2018;49:ATMP69.
21 Harrison JK, McArthur KS, Quinn TJ. Assessment scales in stroke: clinimetric and clinical considerations. *Clin Interv Aging* 2013;8:201–11.
22 Sato S, Toyoda K, Uehara T, *et al*. Baseline NIH Stroke Scale Score predicting outcome in anterior and posterior circulation strokes. *Neurology* 2008;70:2371–7.
23 Martin-Schild S, Albright KC, Tanksley J, *et al*. Zero on the NIHSS does not equal the absence of stroke. *Ann Emerg Med* 2011;57:42–5.
24 Lee YY, Yoon W, Kim SK, *et al*. Acute basilar artery occlusion: differences in characteristics and outcomes after endovascular therapy between patients with and without underlying severe atherosclerotic stenosis. *AJNR Am J Neuroradiol* 2017;38:1600–4.
25 Kubik CS, Adams RD. Occlusion of the basilar artery; a clinical and pathological study. *Brain* 1946;69:73–121.
26 Hacke W, Zeumer H, Ferbert A, *et al*. Intra-arterial thrombolytic therapy improves outcome in patients with acute vertebrobasilar occlusive disease. *Stroke* 1988;19:1216–22.
27 Zeumer H, Hacke W, Kolmann HL, *et al*. [Local fibrinolysis in basilar artery thrombosis (author's transl)]. *Dtsch Med Wochenschr* 1982;107:728–31.
28 Labauge R, Pages M, Marty-Double C, *et al*. [Occlusion of the basilar artery. A review with 17 personal cases (author's transl)]. *Rev Neurol* 1981;137:545–71.
29 Strbian D, Sairanen T, Silvennoinen H, *et al*. Thrombolysis of basilar artery occlusion: impact of baseline ischemia and time. *Ann Neurol* 2013;73:688–94.
30 Kang K, Yoon BW. Symptomatic intracerebral hematomas in posterior circulation stroke patients anticoagulated with heparin. *J Thromb Thrombolysis* 2006;21:249–55.
31 Tong X, Liao X, Pan Y, *et al*. Intravenous thrombolysis is more safe and effective for posterior circulation stroke: Data from the Thrombolysis Implementation and Monitor of Acute Ischemic Stroke in China (TIMS-China). *Medicine* 2016;95:e3848.
32 Ehsan T, Hayat G, Malkoff MD, *et al*. Hyperdense basilar artery. An early computed tomography sign of thrombosis. *J Neuroimaging* 1994;4:200–5.
33 Goldmakher GV, Camargo EC, Furie KL, *et al*. Hyperdense basilar artery sign on unenhanced CT predicts thrombus and outcome in acute posterior circulation stroke. *Stroke* 2009;40:134–9.
34 Connell L, Koerte IK, Laubender RP, *et al*. Hyperdense basilar artery sign-a reliable sign of basilar artery occlusion. *Neuroradiology* 2012;54:321–7.
35 Berkhemer OA, Fransen PS, Beumer D, *et al*. A randomized trial of intraarterial treatment for acute ischemic stroke. *N Engl J Med* 2015;372:11–20.
36 Goyal M, Demchuk AM, Menon BK, *et al*. Randomized assessment of rapid endovascular treatment of ischemic stroke. *N Engl J Med* 2015;372:1019–30.
37 Campbell BCV, Mitchell PJ, Kleinig TJ, *et al*. Endovascular therapy for ischemic stroke with perfusion-imaging selection. *N Engl J Med Overseas Ed* 2015;372:1009–18.
38 Saver JL, Goyal M, Bonafe A, *et al*. Stent-retriever thrombectomy after intravenous t-PA vs. t-PA alone in stroke. *N Engl J Med* 2015;372:2285–95.
39 Jovin TG, Chamorro A, Cobo E, *et al*. Thrombectomy within 8 hours after symptom onset in ischemic stroke. *N Engl J Med Overseas Ed* 2015;372:2296–306.
40 Da Ros V, Meschini A, Gandini R, *et al*. Proposal for a vascular computed tomography-based grading system in posterior circulation stroke: a single-center experience. *J Stroke Cerebrovasc Dis* 2016;25:368–77.
41 Goyal N, Tsivgoulis G, Nickele C, *et al*. Posterior circulation CT angiography collaterals predict outcome of endovascular acute ischemic stroke therapy for basilar artery occlusion. *J Neurointerv Surg* 2016;8:783–6.
42 Maus V, Kalkan A, Kabbasch C, *et al*. Mechanical thrombectomy in basilar artery occlusion: presence of bilateral posterior communicating arteries is a predictor of favorable clinical outcome. *Clin Neuroradiol* 2019 29 1 153 160.
43 Pallesen LP, Khomenko A, Dzialowski I, *et al*. CT-angiography source images indicate less fatal outcome despite coma of patients in the Basilar Artery International Cooperation Study. *Int J Stroke* 2017;12:145–51.
44 Beyer SE, Hunink MG, Schöberl F, *et al*. Different imaging strategies in patients with possible basilar artery occlusion: cost-effectiveness analysis. *Stroke* 2015;46:1840–9.
45 van der Hoeven EJ, Dankbaar JW, Algra A, *et al*. Additional diagnostic value of computed tomography perfusion for detection of acute ischemic stroke in the posterior circulation. *Stroke* 2015;46:1113–5.
46 Lee IH, You JH, Lee JY, *et al*. Accuracy of the detection of infratentorial stroke lesions using perfusion CT: an experimenter-blinded study. *Neuroradiology* 2010;52:1095–100.
47 Pallesen LP, Gerber J, Dzialowski I, *et al*. Diagnostic and prognostic impact of pc-ASPECTS applied to perfusion CT in the Basilar Artery International Cooperation Study. *J Neuroimaging* 2015;25:384–9.
48 Gawlitza M, Quäschling U, Hobohm C, *et al*. Hyperintense basilar artery on FLAIR MR imaging: diagnostic accuracy and clinical impact in patients with acute brain stem stroke. *AJNR Am J Neuroradiol* 2014;35:1520–6.
49 Bulut HT, Yildirim A, Ekmekci B, *et al*. False-negative diffusion-weighted imaging in acute stroke and its frequency in anterior and posterior circulation ischemia. *J Comput Assist Tomogr* 2014;38:627–33.
50 Oppenheim C, Stanescu R, Dormont D, *et al*. False-negative diffusion-weighted MR findings in acute ischemic stroke. *AJNR Am J Neuroradiol* 2000;21:1434–40.
51 Simonsen CZ, Madsen MH, Schmitz ML, *et al*. Sensitivity of diffusion- and perfusion-weighted imaging for diagnosing acute ischemic stroke is 97.5%. *Stroke* 2015;46:98–101.
52 Puetz V, Sylaja PN, Coutts SB, *et al*. Extent of hypoattenuation on CT angiography source images predicts functional outcome in patients with basilar artery occlusion. *Stroke* 2008;39:2485–90.
53 Tei H, Uchiyama S, Usui T, *et al*. Posterior circulation ASPECTS on diffusion-weighted MRI can be a powerful marker for predicting functional outcome. *J Neurol* 2010;257:767–73.
54 Nagel S, Herweh C, Köhrmann M, *et al*. MRI in patients with acute basilar artery occlusion-DWI lesion scoring is an independent predictor of outcome. *Int J Stroke* 2012;7:282–8.
55 Son S, Kim YW, Oh MK, *et al*. Initial factors affecting the clinical outcome after successful recanalization via MR-based mechanical thrombectomy in patients with acute ischemic stroke due to basilar artery occlusion. *J Neurointerv Surg* 2016;8:889–93.
56 Lee W-J, Jung K-H, Ryu YJ, *et al*. Acute symptomatic basilar artery stenosis: MR imaging predictors of early neurologic deterioration and long-term outcomes. *Radiology* 2016;280:193–201.
57 Ostrem JL, Saver JL, Alger JR, *et al*. Acute basilar artery occlusion: diffusion-perfusion MRI characterization of tissue salvage in patients receiving intra-arterial stroke therapies. *Stroke* 2004;35:e30–4.
58 Jauch EC, Saver JL, Adams HP, *et al*. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2013;44:870–947.
59 Rowat AM, Dennis MS, Wardlaw JM. Hypoxaemia in acute stroke is frequent and worsens outcome. *Cerebrovasc Dis* 2006;21:166–72.
60 Aviv JE, Martin JH, Sacco RL, *et al*. Supraglottic and pharyngeal sensory abnormalities in stroke patients with dysphagia. *Ann Otol Rhinol Laryngol* 1996;105:92–7.
61 Milhaud D, Popp J, Thouvenot E, *et al*. Mechanical ventilation in ischemic stroke. *J Stroke Cerebrovasc Dis* 2004;13:183–8.
62 Doggett DL, Tappe KA, Mitchell MD, *et al*. Prevention of pneumonia in elderly stroke patients by systematic diagnosis and treatment of dysphagia: an evidence-based comprehensive analysis of the literature. *Dysphagia* 2001;16:279–95.
63 Bushnell CD, Phillips-Bute BG, Laskowitz DT, *et al*. Survival and outcome after endotracheal intubation for acute stroke. *Neurology* 1999;52:1374–81.
64 Foerch C, Kessler KR, Steckel DA, *et al*. Survival and quality of life outcome after mechanical ventilation in elderly stroke patients. *J Neurol Neurosurg Psychiatry* 2004;75:988–93.
65 Schwarz S, Georgiadis D, Aschoff A, *et al*. Effects of body position on intracranial pressure and cerebral perfusion in patients with large hemispheric stroke. *Stroke* 2002;33:497–501.
66 Wojner-Alexander AW, Garami Z, Chernyshev OY, *et al*. Heads down: flat positioning improves blood flow velocity in acute ischemic stroke. *Neurology* 2005;64:1354–7.
67 Leonardi-Bee J, Bath PM, Phillips SJ, *et al*. Blood pressure and clinical outcomes in the International Stroke Trial. *Stroke* 2002;33:1315–20.
68 Okumura K, Ohya Y, Maehara A, *et al*. Effects of blood pressure levels on case fatality after acute stroke. *J Hypertens* 2005;23:1217–23.
69 Vemmos KN, Tsivgoulis G, Spengos K, *et al*. U-shaped relationship between mortality and admission blood pressure in patients with acute stroke. *J Intern Med* 2004;255:257–65.
70 Williams LS, Rotich J, Qi R, *et al*. Effects of admission hyperglycemia on mortality and costs in acute ischemic stroke. *Neurology* 2002;59:67–71.
71 Gray CS, Hildreth AJ, Sandercock PA, *et al*. Glucose-potassium-insulin infusions in the management of post-stroke hyperglycaemia: the UK Glucose Insulin in Stroke Trial (GIST-UK). *Lancet Neurol* 2007;6:397–406.
72 Sung CY, Lee TH, Chu NS. Central hyperthermia in acute stroke. *Eur Neurol* 2009;62:86–92.
73 Azzimondi G, Bassein L, Nonino F, *et al*. Fever in acute stroke worsens prognosis. *Stroke* 1995;26:2040–3.
74 Prasad K, Krishnan PR. Fever is associated with doubling of odds of short-term mortality in ischemic stroke: an updated meta-analysis. *Acta Neurol Scand* 2010;122:404–8.
75 Sulter G, Elting JW, Maurits N, *et al*. Acetylsalicylic acid and acetaminophen to combat elevated body temperature in acute ischemic stroke. *Cerebrovasc Dis* 2004;17:118–22.
76 Koh MG, Phan TG, Atkinson JL, *et al*. Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. *Stroke* 2000;31:2062–7.

77 Jauss M, Krieger D, Hornig C, *et al*. Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. *J Neurol* 1999;246:257–64.
78 Edlow JA, Newman-Toker DE, Savitz SI. Diagnosis and initial management of cerebellar infarction. *Lancet Neurol* 2008;7:951–64.
79 Ayling OGS, Alotaibi NM, Wang JZ, *et al*. Suboccipital decompressive craniectomy for cerebellar infarction: a systematic review and meta-analysis. *World Neurosurg* 2018;110:450–9.
80 Tsitsopoulos PP, Tobieson L, Enblad P, *et al*. Surgical treatment of patients with unilateral cerebellar infarcts: clinical outcome and prognostic factors. *Acta Neurochir* 2011;153:2075–83.
81 Fahed R, Di Maria F, Rosso C, *et al*. A leap forward in the endovascular management of acute basilar artery occlusion since the appearance of stent retrievers: a single-center comparative study. *J Neurosurg* 2017;126:1578–84.
82 Shu L, Riedel C, Meyne J, *et al*. Successful recanalization in acute basilar artery occlusion treated with endovascular therapy is independent of thrombus length. *J Neurointerv Surg* 2017;9:1047–52.
83 Alonso de Leciñana M, Kawiorski MM, Ximénez-Carrillo Á, *et al*. Mechanical thrombectomy for basilar artery thrombosis: a comparison of outcomes with anterior circulation occlusions. *J Neurointerv Surg* 2017;9:1173–8.
84 Gory B, Lapergue B. Erratum. Mechanical thrombectomy in basilar artery occlusion: influence of reperfusion on clinical outcome and impact of the first-line strategy (ADAPT vs stent retriever). *J Neurosurg* 2018:1–10.
85 Wyszomirski A, Szczyrba S, Tomaka D, *et al*. Treatment of acute basilar artery occlusion: Systematic review and meta-analysis. *Neurol Neurochir Pol* 2017;51:486–96.
86 Turk AS, Spiotta A, Frei D, *et al*. Initial clinical experience with the ADAPT technique: a direct aspiration first pass technique for stroke thrombectomy. *J Neurointerv Surg* 2014;6:231–7.
87 Alawieh A, Vargas J, Turner RD, *et al*. Equivalent favorable outcomes possible after thrombectomy for posterior circulation large vessel occlusion compared with the anterior circulation: the MUSC experience. *J Neurointerv Surg* 2018;;10:735–40.
88 Hu SY, Yi HJ, Lee DH, *et al*. Effectiveness and safety of mechanical thrombectomy with stent retrievers in basilar artery occlusion: comparison with anterior circulation occlusions. *J Korean Neurosurg Soc* 2017;60:635–43.
89 Luo G, Mo D, Tong X, *et al*. Factors associated with 90-day outcomes of patients with acute posterior circulation stroke treated by mechanical thrombectomy. *World Neurosurg* 2018;109:e318–28.
90 Chimowitz MI, Lynn MJ, Derdeyn CP, *et al*. Stenting versus aggressive medical therapy for intracranial arterial stenosis. *N Engl J Med* 2011;365:993–1003.
91 SSYLVIA Study Investigators. Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSYLVIA): study results. *Stroke* 2004;35:1388–92.
92 Gao F, Lo WT, Sun X, *et al*. Combined use of mechanical thrombectomy with angioplasty and stenting for acute basilar occlusions with underlying severe intracranial vertebrobasilar stenosis: preliminary experience from a single Chinese center. *AJNR Am J Neuroradiol* 2015;36:1947–52.
93 Yang D, Lin M, Wang S, *et al*. Primary angioplasty and stenting may be superior to thrombectomy for acute atherosclerotic large-artery occlusion. *Interv Neuroradiol* 2018;24:412–20.
94 Kang DH, Yoon W, Kim SK, *et al*. Endovascular treatment for emergent large vessel occlusion due to severe intracranial atherosclerotic stenosis. *J Neurosurg* 2018;22:1–8.
95 Nordmeyer H, Chapot R, Aycil A, *et al*. Angioplasty and stenting of intracranial arterial stenosis in perforator-bearing segments: a comparison between the anterior and the posterior circulation. *Front Neurol* 2018;9:533.
96 Macleod MR, Davis SM, Mitchell PJ, *et al*. Results of a multicentre, randomised controlled trial of intra-arterial urokinase in the treatment of acute posterior circulation ischaemic stroke. *Cerebrovasc Dis* 2005;20:12–17.
97 Broderick JP, Palesch YY, Demchuk AM, *et al*. Endovascular therapy after intravenous t-PA versus t-PA alone for stroke. *N Engl J Med* 2013;368:893–903.
98 van Houwelingen RC, Luijckx GJ, Mazuri A, *et al*. Safety and outcome of intra-arterial treatment for basilar artery occlusion. *JAMA Neurol* 2016;73:1225–30.
99 Oselkin M, Satti SR, Sundararajan SH, *et al*. Endovascular treatment for acute basilar thrombosis via a transradial approach: Initial experience and future considerations. *Interv Neuroradiol* 2018;24:64–9.
100 Horton TG, Kalapos P, Cockroft KM. Brachial artery approach for endovascular treatment of posterior circulation intracranial vascular disease: technique and application in 5 cases. *J Stroke Cerebrovasc Dis* 2012;21:68–74.
101 Kistler JP, Buonanno FS, DeWitt LD, *et al*. Vertebral-basilar posterior cerebral territory stroke--delineation by proton nuclear magnetic resonance imaging. *Stroke* 1984;15:417–26.
102 Hasso AN, Stringer WA, Brown KD. Cerebral ischemia and infarction. *Neuroimaging Clin N Am* 1994;4:733–52.
103 Cho TH, Nighoghossian N, Tahon F, *et al*. Brain stem diffusion-weighted imaging lesion score: a potential marker of outcome in acute basilar artery occlusion. *AJNR Am J Neuroradiol* 2009;30:194–8.
104 Karameshev A, Arnold M, Schroth G, *et al*. Diffusion-weighted MRI helps predict outcome in basilar artery occlusion patients treated with intra-arterial thrombolysis. *Cerebrovasc Dis* 2011;32:393–400.
105 Mourand I, Machi P, Nogué E, *et al*. Diffusion-weighted imaging score of the brain stem: A predictor of outcome in acute basilar artery occlusion treated with the Solitaire FR device. *AJNR Am J Neuroradiol* 2014;35:1117–23.
106 Renard D, Landragin N, Robinson A, *et al*. MRI-based score for acute basilar artery thrombosis. *Cerebrovasc Dis* 2008;25:511–6.
107 Haussen DC, Oliveira RA, Patel V, *et al*. Functional independence following endovascular treatment for basilar artery occlusion despite extensive bilateral pontine infarcts on diffusion-weighted imaging: refuting a self-fulfilling prophecy. *Interv Neurol* 2016;5:179–84.
108 Takeuchi S, Suzuki G, Takasato Y, *et al*. Prognostic factors in patients with primary brainstem hemorrhage. *Clin Neurol Neurosurg* 2013;115:732–5.
109 Caplan LR, Wityk RJ, Glass TA, *et al*. New England medical center posterior circulation registry. *Ann Neurol* 2004;56:389–98.
110 Nikić PM, Jovanović D, Paspalj D, *et al*. Clinical characteristics and outcome in the acute phase of ischemic locked-in syndrome: case series of twenty patients with ischemic LIS. *Eur Neurol* 2013;69:207–12.
111 Morrison LJ, Gent LM, Lang E, *et al*. Part 2: Evidence evaluation and management of conflicts of interest: 2015 American Heart Association Guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 2015;132(18 Suppl 2):S368–82.