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Topical Review

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Care of the Post-Thrombectomy Patient

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Multiple lines of evidence supporting endovascular stroke treatment (EVT) have propelled this treatment modality as the standard of care[1] with increased anticipated and observed utilization. In parallel, it has been recognized that optimal outcomes are reliant on processes across the disease continuum from first medical contact, prehospital triage, primary hospital treatment, interfacility transfer, destination hospital care, preprocedural planning, reperfusion therapy, post-thrombectomy care, and rehabilitation. In this review, we focus specifically on considerations in the care of the post-thrombectomy patient. In particular, we address the common and critical complications as they occur over the spectrum of care from preprocedural, intraprocedural, and postprocedural management (Figure).

Key Issues in Patients Undergoing EVT

Complications after EVT can be broadly considered as being inherent to issues surrounding the stroke itself (hyperglycemia, temperature dysregulation, arrhythmias, hemodynamic instability, aspiration, respiratory failure, and infection), as well as issues commonly encountered in all critically ill patients, such as stress ulcers, pressure ulcers, and peripheral venous thrombosis. Complications can also be secondarily related to the procedure (hemorrhagic conversion and malignant edema) or direct complications (volume overload, arteriotomy site complications, vessel perforation, vasospasm, device retention, and vessel reocclusion). Adverse events may further be different in reperfused versus nonreperfused patients. For instance, reperfusion injury is not a feature of nonreperfused patients, whereas infarct growth is. Key management decisions include blood pressure (BP) parameters, glucose target, fluid balance, medication use (ie, antithrombotics, anticoagulants, and antiepileptics), as well as necessity and timing of surgical interventions (eg, craniectomy, extraventricular drain placement, tracheostomy, and parenteral gastrostomy). Given the broad spectrum and complex nature of adverse events, the postoperative care should be directed in an intensive care unit with stroke-specialized nursing and physicians.

Contrast-Related Considerations

Patients undergo noninvasive radiocontrast media-based studies before catheter-based angiography, including single-phase or multiphase computed tomographic (CT) angiography to document an occlusion, determine collateral quality, and estimate core infarct. They may undergo CT perfusion (CTP) to estimate core infarct and salvageable tissue. These studies typically involve ≈70 mL of radiocontrast media for CT angiography head/neck and 80 mL of radiocontrast media for CTP. Occasionally, patients undergoing interfacility transfer may undergo repeated studies to determine interval vessel recanalization. Subsequent catheter-based angiography may then involve a range of radiocontrast media utilization depending on the complexity of the case. The volume is typically less than that used for CT angiography/CTP studies, and the additional saline flush utilized during angiography does not generally lead to volume overload in adults. However, this volume may be a concern in patients with heart failure or in the pediatric population.

Allergies to iodinated radiocontrast media are estimated to occur with a prevalence of 1% to 12% with severe reactions comprising only 0.01% to 0.2% of all reactions.[2] Prophylactic regimens may reduce the frequency of adverse reactions, and at our center, we routinely use a periprocedural emergency protocol consisting of 50 mg of intravenous diphenhydramine and 200 mg of intravenous methylprednisolone. Serious reactions can include severe bronchospasm, facial/laryngeal edema, and hemodynamic instability (hypotension and bradycardia or tachycardia; Table).

Contrast-induced nephropathy is substantially less common than widely believed. It is a more delayed complication of contrast exposure that may typically manifest with rising serum creatinine within the first 24 hours of exposure and can peak at day 3 or 5. Recent data have emphasized that there is little evidence that modern (low osmolar, nonionic) radiocontrast media is nephrotoxic in the typical stroke patient requiring thrombectomy.[3] It remains possible that contrast nephropathy was relevant with older agents. Most patients with rising creatinine after a radiocontrast-enhanced imaging study harbor additional comorbidities that also may contribute

Received July 6, 2018; final revision received September 12, 2018; accepted September 17, 2018.

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(*Stroke*. 2018;49:2801-2807. DOI: 10.1161/STROKEAHA.118.021640.)

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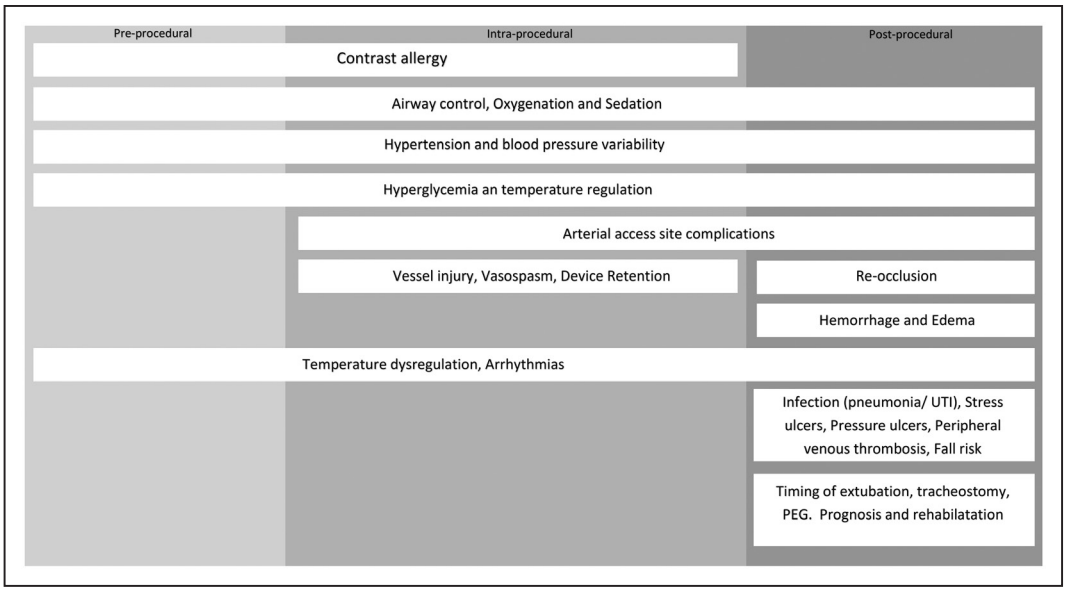


Figure. Adverse events encountered during various stages of care in the post-thrombectomy patient. PEG indicates percutaneous endoscopic gastrostomy; and UTI, urinary tract infection.

to nephrotoxicity. In general, hydration and adjustment of electrolyte imbalance is helpful with dialysis becoming a necessity in a minority of cases. Creatinine typically normalizes by 7 to 10 days.

Arterial Access-Site Complications

Catheter-based angiography is typically performed via the transfemoral approach; however, this route may not be feasible in patients with severe femoral, external iliac, or aortic disease or in cases where catheterization of the target vessel may be difficult with resultant prolonged groin-to-recanalization times. Alternative sites of access include the arm (radial, brachial, or axillary arteries), particularly in cases of vertebral-basilar occlusions, as the vertebral artery can be more directly accessed. In select cases, particularly older, hypertensive patients with left carotid lesions, access from the aortic arch may be challenging, and a direct transcervical approach may be indicated. Femoral puncture should typically occur at the common femoral artery as a puncture that is too high may lead to increased risk of retroperitoneal hemorrhage (particularly if

Table. Complications and Treatment Considerations in the Post-Thrombectomy Patient

Complication	Treatment
Contrast allergy	Pretreat with 50 mg IV diphenhydramine and 200 mg IV methylprednisolone; supplemental oxygen; intravenous epinephrine, diphenhydramine (H1 antagonist), hydrocortisone, and ranitidine (H2 antagonist); intravenous fluid resuscitation and epinephrine infusion (if hypotensive); intubation (if severe laryngeal edema)
Access-site hematoma and pseudoaneurysm	Establish hemostasis, reestablish arterial flow, vascular repair, initiate blood transfusion, thrombin injection, or direct surgical repair of symptomatic pseudoaneurysms, watchful waiting (if stable)
Vessel vasospasm	Remove catheter/device, infusion of calcium channel blocker (5–10 mg of intra-arterial verapamil, 2.5 mg of intra-arterial nicardipine), balloon angioplasty (if severe)
Vessel perforation	Reduce SBP to <140 mm Hg, reverse coagulopathies, transfuse platelets if indicated, watchful waiting (if extravasation is self-limited), inflate balloon at site of extravasation to achieve hemostasis; if extravasation persists, advance microcatheter to the site of perforation and deliver liquid embolic agent (Onxy, glue) or coils to achieve hemostasis. If tear is discrete, vessel patency may be preserved, but in most cases, vessel sacrifice is necessary; postprocedural CT to assess for hemorrhage, edema, and hydrocephalus; Extensive intraventricular hemorrhage and hydrocephalus may warrant CSF diversion with EVD; extensive parenchymal hematoma with mass effect and edema may warrant clot evacuation and decompressive craniectomy.
Vessel dissection or intrinsic residual stenosis	If not flow limiting or not severely narrowing, medical therapy with antithrombotics and permissive hypertension; if high concern for vessel occlusion, consider acute stent placement with loading of intravenous antiplatelet agents (eg, 180 μg/kg of eptifibatide) and postprocedural loading with 325 mg aspirin and 600 mg clopidrogel via enteral access
BP control	If reperfusion achieved, aim for moderate or normal BP goal of SBP <140 or 160 mm Hg; if reperfusion not achieved, permissive hypertension with SBP <180 or 220 mm Hg
Glucose control	Aim for normoglycemia (<180 mg/dL or 10 mmol/L)
Temperature control	Aim for euthermia; benefit of hypothermia remains unproven
Tracheostomy	Consider early in patients with severe bulbar weakness or large infarct with midline shift

BP indicates blood pressure; CSF, cerebrospinal fluid; CT, computed tomography; EVD, extraventricular drain; IV, intravenous; and SBP, systolic blood pressure.

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the posterior arterial wall is punctured inadvertently) as there is no underlying bone to facilitate effective manual compression. In cases of low puncture, there is a higher risk of arteriovenous malformation (<0.1%) or pseudoaneurysm formation (1%). These lesions typically manifest with a pulsatile bruit and groin site pain and swelling. Most cases can be managed with observation and serial sonography; more symptomatic cases may require thrombin injection or direct surgical repair.[4]

Access-site hemostasis may be a challenge as patients are often exposed to intravenous thrombolysis (IVT) or heparin products, and the access site may be 6F or larger in size. Stasis may require prolonged manual compression several centimeters proximal to the arteriotomy site (typically 30–60 minutes) or closure device deployment (eg, 6F or 8F Angio-Seal vascular closure device). Patients are then kept supine for 2 to 6 hours, depending on the risk of access-site hemorrhage. Occasionally, it may be necessary to reverse coagulopathies. Rarely, embolization or placement of a covered stent may be required. During vessel cannulation, advancement of the needle or guidewire in a vessel plane may lead to retrograde dissection formation. These dissections are typically self-resolving and asymptomatic because blood flow does not keep the dissection flap open. Anterograde dissections, however, can propagate further and lead to vessel occlusion and ultimately limb ischemia. Limb ischemia may also occur because of thrombus formation or intravascular closure device failure. Serial neurovascular assessment in the postprocedural setting is important to the early identification of access-site complications.

Puncture at a high location or of the posterior wall can increase the risk of retroperitoneal hemorrhage (incidence, <3%). Early signs can include back pain, bruising of the flank (Grey Turner sign), abdominal distention with periumbilical ecchymosis (Cullen sign), hypotension, and tachycardia. Late complications can include a pressure-induced femoral neuropathy because of hematoma compression of the nerve in the psoas sheath. This can be a devastating evolution in a patient with stroke, preventing return to walking and interfering with rehabilitation. Diagnosis is typically confirmed by noncontrast CT of the pelvis. Addition of contrast or the use of ultrasound imaging, however, may additionally identify a pseudoaneurysm or vessel irregularity. Examination of the femoral nerve function is a critical postoperative task. Initial management is fluid resuscitation with crystalloids and blood transfusion as needed. In refractory cases of hemodynamic instability, surgical evacuation and vessel repair may be indicated. Surgical evacuation or radiological aspiration of the hematoma may also be indicated to decompress the femoral nerve.

Intraprocedural Vessel Injury, Nonrecanalization, and Vessel Reocclusion

One of the most devastating complications encountered during EVT is vessel perforation with concomitant extravasation. The incidence is likely lower in the modern era with advent of safer devices and less-traumatic aspiration catheters. Perforation rates in randomized controlled trails have varied from 0.9% to 4.9% with lack of uniform definitions likely accounting for the variability.[5] In a recent multicenter analysis of 1599 patients undergoing stent retriever thrombectomy, intraprocedural perforation occurred in 1% of all cases.[6] A majority of the perforations involved distal locations (63%). Mortality was high (63%), but 25% of patients were able to achieve favorable functional outcome suggesting that perforation is not universally devastating. Hemostasis can be achieved with BP control, reversal of coagulopathy, balloon inflation, embolization of the perforation site, or vessel sacrifice. Immediate postprocedural imaging is important to assess the need for hyperosmolar therapy, extraventricular drain placement, or craniectomy.

A more common and typically more benign intraprocedural complication encountered is catheter- or device-associated vasospasm. This often resolves after removal of the catheter but may occasionally require infusion of intra-arterial calcium channel blockers. It is customary to confirm brisk anterograde transit times and improvement of the vasospasm. Persistent or refractory spasm with delayed transit times may indicate an underlying dissection. These lesions do not usually require revascularization with stent placement unless there is concern for hemodynamic impairment in the distal vascular bed. Often, antithrombotic agents or more liberal postprocedural BP parameters are the only measures used.

Unsuccessful or incomplete recanalization can lead to continued infarct growth. Permissive hypertension may be necessary to allow collateral flow to sustain ischemic penumbra.[7] After successful revascularization, a subset of patients is at risk of vessel reocclusion, particularly patients with underlying hypercoagulable state or those harboring an intrinsic lesion. Close, frequent neurological monitoring, especially in the first 24 to 48 hours, is essential for early identification and treatment. Immediate initiation of anticoagulation may be considered in selected vulnerable patient populations if post-treatment scan demonstrates no hemorrhage or large infarct. In patients with untreated, intrinsic lesions, medical management should be instituted with dual antiplatelet therapy and permissive hypertension. If clinical fluctuation or deterioration occurs, acute stenting may be indicated.

In rare circumstances, device fracture and retention can occur. In such cases, retrieval is the typical first step. This can be accomplished with the use of retrieval snare devices.[8] However, this may not be possible, and postprocedural anticoagulation may be necessary. The manufacturer should be contacted, and the retained material should be reviewed for compatibility before exposing the patient to magnetic resonance imaging testing.

Airway Control, Oxygenation, and Sedation

Given the nature of the neurological injury, patients suffering an acute stroke are prone to agitation, noncooperation, dysphagia, aspiration, and respiratory failure. The current American Heart Association guidelines recommend maintaining oxygen saturation >94% in all patients with acute ischemic stroke (AIS).[1] Intubation may be necessary for airway protection or electively pursued before EVT. There is no general consensus on the optimal method of anesthesia (general, conscious sedation, or local anesthetic) with a small number of largely single-center randomized trials

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suggesting both methods are safe and feasible with no marked difference in clinical outcomes. Prospectively obtained registry and nonrandomized data cohorts have reported higher rates of poor outcomes in patients undergoing general anesthesia with increased rates of pneumonia, prolonged intubation, and worse neurological outcome (principally in patients with induction-related hypotension). Advocates of general anesthesia cite theoretical benefits of decreasing metabolic demands in the anesthetized patient, lower patient discomfort, as well as the advantages of a more controlled environment while performing the procedure although it is not clear what the advantages of such a controlled environment are from a patient outcome perspective. The optimal strategy likely varies by patient and endovascular team and should account for the patient's individual challenges and treating center expertise. Regardless of the approach, it is clear that even transient prerecanalization BP decreases are harmful[9] and should be avoided. Postprocedural extubation should be prioritized to try to reduce the risk of ventilator-associated pneumonia.

Early or prolonged intubation may be anticipated in patients with decreased level of consciousness or in patients with impaired secretion control (eg, with midline shift or brain stem/thalamic involvement). A subset of patients may benefit from early tracheostomy, and randomized controlled trials are ongoing to address this area of uncertainty.[10]

BP Management Pre-EVT and Post-EVT

Hemodynamic parameters in the patient with AIS are complex and dynamic. Initial BP is often elevated to augment blood flow via collaterals to the ischemic tissue. Both low and high baseline systolic BP (SBP) are associated with poor functional outcome. Although it is reasonable to treat symptomatic hypotension and hypovolemia, there are no data to support routine use of induced hypertension. Higher admission SBP is an independent predictor of increased final infarct volume and lower likelihood of good outcome.[11] In a post hoc analysis of the MR CLEAN trial (A Randomized Trial of Intra-Arterial Treatment for Acute Ischemic Stroke), higher baseline SBP was associated with higher risk of symptomatic intracranial hemorrhage (ICH); however, baseline BP did not impact EVT effectiveness.[12] Interestingly, the investigators find a U-shaped association because both low and high baseline SBP were associated with poor functional outcome. Details about the trajectory of BP during or after EVT were not available, and so, no conclusions could be drawn on the target post-EVT BP. Before IVT/EVT, it is recommended to target an SBP <185 mm Hg and diastolic BP <110 mm Hg.

The optimal BP post-EVT remains an area of uncertainty. In a single-center analysis of 88 patients who underwent EVT, large fluctuations in the post-EVT BP from the mean were found to be predictive of worse outcomes.[13] In patients with incomplete reperfusion (Thrombolysis in Cerebral Infarction 2a or less) or receiving IVT alone, it is judicious to maintain an upper BP limit of <180/110 mm Hg. In patients with complete or nearly complete reperfusion, a stricter SBP upper limit of 160 or 140 mm Hg may be considered to reduce the likelihood of hemorrhagic conversion or reperfusion injury. In a single-center analysis of 217 patients who underwent EVT, a 10-mm Hg increment in maximum SBP during the first 24 hours post-EVT was associated with a lower likelihood of functional independence and higher likelihood of mortality at 3 months.[13] Patients with intensive or moderate BP ranges had lower likelihood of mortality compared with the permissive hypertension group. Given the retrospective nature of this analysis, BP might simply be a marker of underlying prognosis, making it difficult to establish a cause-effect relationship between post-EVT BP and outcomes. Additionally, extremely low BPs may be detrimental to maintaining perfusion if there are persistent filling defects (ie, non-Thrombolysis in Cerebral Infarction 3 flow). Randomized trials of post-thrombectomy BP management are needed.

Given the absence of randomized control trial data, it seems rational to set a systolic upper limit of 140 to 160 mm Hg post-complete or near-complete reperfusion. In patients with tandem lesions who undergo adjunctive extracranial stent placement or in patients with intrinsic lesions who undergo intracranial stent placement, a more intensive target BP may be warranted to mitigate the possibility of reperfusion injury.

Hyperglycemia Is Associated With Worse Outcomes After AIS

Hyperglycemia is common after AIS in both diabetic and nondiabetic patients and is associated with poor outcomes. In patients undergoing IVT, hyperglycemia on admission is associated with lower rates of successful recanalization[14] and higher rates of symptomatic ICH[15] with subsequent increased risk of poor clinical outcome.[16] In patients undergoing EVT, hyperglycemia has similarly been demonstrated to be associated with poor outcomes and higher likelihood of symptomatic ICH.[17] Hyperglycemia, however, has not been shown to impact the likelihood of complete reperfusion suggesting that the detrimental impact is independent of quality of reperfusion.

Although it is clear that hyperglycemia leads to worse outcomes after AIS, no clinical trial data to date support the benefit of tight glucose control in altering outcomes, although this is being investigated in the ongoing SHINE trial (Stroke Hyperglycemia Insulin Network Effort).[18] In the absence of trial data, the current American Heart Association guidelines recommend treating hyperglycemia to achieve target glucose levels of 140 and 180 mg/dL (7.7–10.0 mmol/L) during the first 48 hours post-ictus with careful attention to avoiding hypoglycemic episodes.[1] There is no current indication that hyperglycemia should be considered a contraindication for mechanical thrombectomy.

Temperature Control After AIS

Elevated temperature in the stroke population can be attributed to hyperthermia (abnormality of homeostasis) versus fever (a physiological response to pathogens). In the case of fever, treatment is focused on temperature control along with addressing the underlying source of infection. In the case of hyperthermia, the source is felt to be central or the injured brain itself (eg, irritated hypothalamus and systemic inflammatory response), and temperature control may be the sole option after the exclusion of potential fever sources (eg,

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infection, drug, venous clot). Numerous studies have demonstrated that elevated temperature leads to worse outcomes after stroke (increased infarct growth, higher disability, and more deaths).[19] Harmful effects include increase in metabolic demand, alteration of protein function, release of neurotoxic excitatory neurotransmitters, and production of free radicals. Temperature control can be achieved by identifying and treating the source, initiating antipyretics, infusing cold saline, and utilizing cooling devices. Although euthermia should be the goal, the clinical benefit of hypothermia in patients with acute stroke remains unproven and is currently being investigated.

Management of Post-EVT Hemorrhage and Edema

Neurological decline after EVT should be evaluated to assess for hemorrhagic conversion and mass effect. Although EVT has been shown to reduce final infarct, subsequent edema,[20] and need for decompression, close monitoring is necessary for early identification of patients at high risk for malignant edema and mass effect-associated clinical decline. In rare situations, reocclusion (particularly in the setting of intrinsic disease) or new occlusion may occur, and repeat vessel imaging should be considered.

There is no established guideline on the need or timing of routine post-EVT neuroimaging in clinically stable patients not receiving IVT. In a pooled meta-analysis of patients enrolled in the SWIFT PRIME (Solitaire FR With the Intention for Thrombectomy as Primary Endovascular Treatment for Acute Ischemic Stroke Clinical Trial), EXTEND-IA (Extending the Time for Thrombolysis in Emergency Neurological Deficits — Intra-Arterial Trial), REVASCAT (Randomized Trial of Revascularization With Solitaire FR® Device Versus Best Medical Therapy in the Treatment of Acute Stroke due to Anterior Circulation Large Vessel Occlusion Presenting Within 8 Hours of Symptom Onset), and ESCAPE (Small Core and Anterior Circulation Proximal Occlusion With Emphasis on Minimizing CT to Recanalization Times Trial) trials, the rates of symptomatic hemorrhage (2.4% versus 2.8%) and parenchymal hematoma (9.2% versus 8%) were no different in patients managed with EVT versus medical therapy alone.[21] Similar to patients with IVT, it is reasonable to obtain imaging at 24 hours to assess for hemorrhage and guide the timing of antiplatelets or anticoagulants. In some cases, magnetic resonance imaging or dual-energy CT can be useful to distinguish between hemorrhage and iodinated contrast extravasation. In the absence of hemorrhagic conversion, it is recommended to initiate antithrombotic monotherapy and deep venous thrombosis (DVT) chemoprophylaxis. In patients requiring anticoagulation, the timing is dependent on the stroke burden (ie, projected risk for hemorrhagic conversion) and the urgency of anticoagulation (eg, secondary prevention in the setting of atrial fibrillation versus a more pressing need to treat a deep venous thrombosis or pulmonary embolism). At our center, when indicated, we typically initiate anticoagulation if there is small stroke burden (<30 cc) on the postprocedural scan. In patients with moderate infarct (30–70 cc), anticoagulation is typically held for 2 to 4 weeks, and in patients with large infarct (>70 cc), anticoagulation is typically held for 4 to 6 weeks. The timeline should be tailored to individual patient risk-benefit profile and is variable from center to center based on provider preference.

When postreperfusion hemorrhage is encountered, it is important to first classify the severity of the bleed. A commonly used classification scheme is the Heidelberg bleeding classification criteria,[22] which includes the presence of hemorrhage on CT or magnetic resonance head within 48 hours after reperfusion treatment: HI1 (small petechiae), HI2 (more confluent petechiae), parenchymal hematoma (PH)1 (≤30% of the infarcted area with mild mass effect), and PH2 (>30% of the infarcted area with significant mass effect). BP control should be initiated to prevent further hemorrhage expansion. At our center, we suspend the use of deep venous thrombosis chemoprophylaxis for 24 to 48 hours after serial imaging demonstrates hemorrhage stability. In patients with PH1 or PH2 hemorrhage, reversal of alteplase can be considered (particularly if the fibrinogen level is <100 mg/dL) with cryoprecipitate, fresh-frozen plasma plus other blood product with adequate fibrinogen content; the choice may be related to rapid availability and the patient's capacity to tolerate a volume load. Other medication-related coagulopathies should target the relevant parameters: prothrombin complex concentrate and vitamin K for warfarin-related elevated international normalized ratio; protamine for heparin-related elevated partial thromboplastin time; antifibrinolytic agents, desmopressin acetate, prothrombin complex concentrates and antibodies to reverse the effects of dabigatran (idarucizumab), and the oral direct factor Xa inhibitors (andexanet alfa).

In patients with pretreatment antithrombotic exposure, there does not seem to be higher risk of symptomatic ICH.[23] It may be reasonable to discontinue the use of antiplatelets once hemorrhagic conversion is noted. At our center, we routinely suspend aspirin for 5 to 7 days if there is post-treatment hemorrhage and there are no contraindications. Again, the risks and benefits of continuing antiplatelets should be considered at the individual patient level, and practice may vary from center to center. For example, in a patient with active acute coronary syndrome or with a newly placed carotid or intracranial stent with a stable, asymptomatic hemorrhage, the benefits of continuing dual antiplatelets will outweigh the theoretical harm of suspending these drugs. At present, there are no data to support platelet transfusions in patients with ICH exposed to antithrombotic agents. In patients who were previously anticoagulated without evidence of post-thrombectomy hemorrhage, there is typically no indication for reversal.

Nutrition and Additional Poststroke Care

Aspiration precautions should be exercised to minimize aspiration pneumonia, particularly in patients with oropharyngeal weakness or poor mentation. Similar to tracheostomy placement, the timing of percutaneous endoscopic gastrostomy tube placement can be challenging. In the FOOD trial (The Feed or Ordinary Diet), patients with feeding via nasogastric tube versus percutaneous endoscopic gastrostomy tube had better functional outcomes suggesting that early percutaneous endoscopic gastrostomy placement may be not be a beneficial routine approach.[24]

There are no randomized data on the impact of most poststroke care specific to the EVT population, and at present,

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similar practices should be used in all populations.[1] In the critically ill patients with stroke, the usual measures should be instituted, including initiation of a proton pump inhibitor (to minimize the formation of gastrointestinal ulcers), commencement of deep venous thrombosis prophylaxis (chemoprophylaxis or intermittent pneumatic compression stockings in patients with concern for ICH), upkeep of a routine bowel regimen, and introduction of longer term stroke prevention measures (treatment of hypertension, lipid lowering, smoking cessation, diabetes mellitus management, lifestyle measures counseling).

Rehabilitation, Recovery, and Prognostication

Early neurological improvement (as measured by the National Institutes of Health Stroke Scale at 24 hours and day of discharge or the modified Rankin Scale at day of discharge) is a strong predictor of functional outcome at 90 days. In patients with persistent disability, early mobilization has been traditionally recommended to reduce poststroke complications. In the AVERT trial (A Very Early Rehabilitation Trial After Stroke), patients were randomized to early (<24 hours), frequent, higher dose therapy versus early, lower dose activity. The patients who received early therapy had no difference in mortality but had lower rates of favorable outcome (46% versus 50%; P=0.04).[25] Although further trials are necessary to understand the exact timing and dosing of rehabilitation, it seems that it is more efficacious to initiate activity at day 2 versus day 1 poststroke.

After EVT, prognostication is important in guiding which aggressive medical measures or surgical therapies should be pursued. The individual patient goals and wishes are often related to chance of long-term functional independence. The Pittsburgh Outcomes After Stroke Thrombectomy scale was developed to predict 3-month outcomes in anterior circulation large vessel occlusion stroke patients with incorporation of postintervention variables.[26] By factoring in age, final infarct volume, and presence of PH1/2, the score can reliably predict 3-month clinical outcome at a relatively early stage after the intervention. In addition to age, outcome after posterior circulation stroke seems to be more dependent on stroke location (ie, brain stem) rather than total stroke burden as demonstrated by the Pittsburgh Outcomes After Stroke Thrombectomy Vertebrobasilar score.[27] Utilization of Pittsburgh Outcomes After Stroke Thrombectomy, Pittsburgh Outcomes After Stroke Thrombectomy Vertebrobasilar, and other similar clinical tools can be helpful in guiding family decision-making.

Conclusions

With increasing utilization of EVT, there is a parallel need for expertise and familiarity with the periprocedural and postprocedural care of this complex patient population. Given that a large number of patients do not achieve functional independence or survive despite EVT, it is paramount to identify treatable factors that may improve functional outcomes. Complications related to the procedure itself are rare but can lead to devastating consequences, and awareness and vigilance in a highly monitored environment is essential to augmenting good outcomes. Multiple areas of uncertainty remain and will be the focus of future research, including understanding ideal post-EVT BP parameters and optimal blood glucose management. New treatments and strategies will need to be developed to prevent and mitigate malignant edema and hemorrhagic conversion, particularly as the benefit of EVT is being investigated in additional populations, such as patients with salvageable tissue, despite large baseline core infarcts.

Disclosures

Dr Molyneaux served on a scientific advisory board and as a consultant for Biogen. Dr Hill received research support from Medtronic LLC, Stryker LLC, NoNO, Inc, Boehringer Ingelheim, the Calgary Stroke Program, the Hotchkiss Brain Institute, the University of Calgary, the Heart and Stroke Foundation of Alberta, Heart and Stroke Foundation of Alberta, Northwestern Territories and Nunavut, and Alberta Innovates Health Solutions; personal fees from Merck; and nonfinancial support from Hoffmann-La Roche, Canada. He has a pending patent related to systems and methods for assisting in decision-making and triaging for patients with acute stroke (US patent office No. 62/086,077) and holds stock ownership in Calgary Scientific, Inc. Dr Jovin acts as an investor/advisor at Route 92 Medical, Anaconda Biomed, FreeOX Biotech, Blockade Medical, and Silk Road Medical and as a consultant at Cerenovus and Stryker Neurovascular. The other authors report no conflicts.

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KEY WORDS: blood pressure ■ edema ■ glucose ■ hemorrhage ■ thrombectomy

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