Invited Review Article

Blood Pressure Management Following Large Vessel Occlusion Strokes: A Narrative Review

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Stroke is one of the leading causes of mortality and morbidity worldwide. Intravenous tissue Plasminogen Activator (tPA) and Mechanical Thrombectomy (MT) comprise the two major treatments for acute ischemic stroke. tPA has been used for more than two decades and guidelines for hemodynamic management following tPA administration are well established. However, MT is a relatively newer therapy, and there is a paucity of evidence regarding hemodynamic management following large vessel occlusion (LVO) strokes. The important tenets guiding the pathophysiology of LVO strokes include understanding of cerebral autoregulation, collateral circulation and blood pressure variability. In this narrative review, we discuss the current American Heart Association - American Stroke Association (AHA/ASA) guidelines for early management of acute ischemic stroke during the different phases of illness encountered at different locations of a hospital including the emergency room (ER), the neuro-interventional suite (IR) and the intensive care unit (ICU). There is emerging evidence with regards to post-recanalization blood pressure management following LVO strokes. Future research directions will include real time blood pressure variability assessments, identifying the extent of impaired autoregulation, and providing guidelines related to range and personalized blood pressure trajectories for patients following LVO strokes.

Glossary of Terms:
AHA/ASA – American heart association/ American Stroke Association; AIS -acute ischemic stroke; ASPECT - Alberta stroke program early CT; ER – emergency room; ICU – intensive care unit; MT- mechanical thrombectomy; MAP – mean arterial pressure; SBP – systolic blood pressure; LVO – large-vessel occlusion; EMS – emergency medical services; NIHSS - National Institute of Health Stroke Scale; MCA – middle cerebral artery; SNACC - Society for Neuroscience in Anesthesia and Critical Care; tPA – tissue plasminogen activator; ESO - European Stroke Organization; SNIS – Society of Neurointerventional Surgery; sICH – symptomatic intracranial hemorrhage; MERCI - Mechanical Embolus Removal in Cerebral Ischemia; MR CLEAN - Multicenter
Introduction

Stroke is the second leading cause of death affecting six million people every year world-wide. The global burden of ischemic stroke has increased by 37% between 1990 to 2010. Per CDC figures, stroke is the fifth leading cause of death and a leading cause of disability in the US. Strokes add 34 Billion USD to US healthcare cost every year. Ischemic stroke is the most common subtype comprising 87% of all types of strokes (including subarachnoid and intracranial hemorrhages) in the United States. Intra-venous tPA was US FDA (United States Food and Drug Administration) approved for treatment of acute ischemic stroke in 1996 after the National Institute of Neurological Disorders and Stroke (NINDS) trial and has been the main stay of treatment ever since. After unsuccessful initial attempts involving intra-arterial treatment, the first endovascular clot retriever, Mechanical Embolus Removal in Cerebral Ischemia (MERCI) retriever was FDA-approved in 2004. After a series of negative trials, largely attributed to study design errors, the Multicenter Randomized Clinical Trial of Endovascular Treatment for Acute Ischemic Stroke in the Netherlands (MR CLEAN), showed the benefit of Mechanical Thrombectomy (MT) in LVO strokes in 2015. Such benefit was subsequently confirmed by several other LVO stroke trials including SWIFT PRIME (Solitaire™ With the Intention For Thrombectomy as PRIMary Endovascular Treatment), EXTEND-IA (Extending the Time for Thrombolysis in Emergency Neurological Deficits - Intra-Arterial), ESCAPE (Endovascular Revascularization With Solitaire Device Versus Best Medical Therapy in Anterior Circulation Stroke Within 8 Hours), THRACE (Trial and Cost Effectiveness Evaluation of Intra-arterial Thrombectomy in Acute Ischemic Stroke), REVASCAT (Endovascular Revascularization With Solitaire Device Versus Best Medical Therapy in Anterior Circulation Stroke Within 8 Hours), and THRACE (Trial and Cost Effectiveness Evaluation of Intra-arterial Thrombectomy in Acute Ischemic Stroke). MT has become the recommended standard for anterior circulation LVO strokes presenting within six hours of symptom onset, having a favorable radiological Alberta stroke program early CT (ASPECT) score, and National Institutes of Health Stroke Scale (NIHSS) 6 or greater. More recently, after the publication of the DEFUSE3 (Endovascular Therapy Following Imaging Evaluation for Ischemic Stroke 3) trial and the DAWN (Clinical Mismatch in the Triage of Wake Up and Late Presenting Strokes Undergoing Neurointervention With Trevo) trial in 2018, the time window for efficacy of MT was extended to 16 to 24 hours, using perfusion imaging to a smaller group of patients. MT for acute LVO strokes is a relatively new therapy compared to tPA and its peri-procedural guidelines have evolved with emerging evidence. We continue to learn about hemodynamic management following LVO strokes as we gather new data. In the interim, the AHA-ASA guidelines recommend blood pressure goals of lower than 180/105 mm Hg following reperfusion, similar to the guidelines following tPA administration. However, the recanalization rates of these treatments are not similar. Studies have shown that tPA is able to achieve recanalization rates of 17-38%, while MT is able to achieve 70-90% recanalization. Therefore, similar BP goals can not necessarily apply to both post-recanalization phase after tPA administration and MT. The purpose of this review is to examine the key concepts related to patho-physiology of LVO strokes, current evidence on post-recanalization BP management, and to provide recommendations on modified management strategies in light of the current evidence. Hemodynamics and Pathophysiology of LVO Strokes

Impaired Autoregulation

In a normal brain, the blood vessels constrict in response to high pressures and dilate in response to low pressures to maintain a constant perfusion to the cerebrovascular capillary bed. This phenomenon is called cerebral autoregulation and is an essential homeostatic process that allows the brain to receive adequate blood even in the setting of fluctuating systemic blood pressures, albeit, within a range of 60-150mm Hg. This range is shifted rightward in chronic hypertensive patients due hypertrophy of cerebral vessel walls. The auto-regulatory curve shifts downwards in a setting of hypocapnia, and shifts upwards with a narrow plateau in hypercapnia. Cerebral autoregulation is lost following an acute ischemic stroke.
LVO strokes involve acute occlusion of blood vessels in the anterior circulation including the Internal Carotid, the Middle Cerebral and the Anterior Cerebral Arteries; as well as the posterior circulation including the Vertebral, the Basilar and the Posterior Cerebral Arteries. Such occlusion results from thrombosis in an atherosclerotic vessel or from an embolus originating from the heart or the cervical blood vessels. Following an acute stroke, cerebral autoregulation is known to be impaired, increasing the dependence of cerebral perfusion on systemic pressures. Therefore, a peak in systemic blood pressure in the setting of pre-existing vaso-dilatation in the ischemic tissue places the infarcted tissue at risk of hemorrhagic transformation. This could result from luxury perfusion in the ischemic core as well as reperfusion injury following re-canalization. Surrounding the ischemic core, tissue with marginal blood flow, called the penumbra is also susceptible to these changes. On the other hand, brief and drastic blood pressure drops in the first 24 hours of an acute ischemic stroke has been shown to contribute to the loss of penumbral tissue. Interestingly, the blood vessels in the contralateral side of the ischemia are also dilated in order to perfuse the penumbra through collateral circulation. Cerebrovascular pressure reactivity (PRx) and tissue oxygenation measurements using near-infrared spectroscopy have been reported to estimate auto-regulation index in stroke patients.

Collateral Circulation
In the setting of ischemic stroke the core of the infarct experiences irreversible damage, but the penumbra in the periphery of the infarct can be salvaged. The penumbra receives blood flow via collaterals. Collateral circulation has an anatomic aspect, as well as a physiologic aspect. The anatomic aspect of the collateral vessels can be quantified using a multi-phase CT angiogram or a conventional cerebral angiogram. A multiphase CT angiography collateral score ranges from 0 indicating no visible collateral vessels to a maximum of 3 indicating extensive collateral vessels. The collateral circulation is dynamic, and its physiological aspect is difficult to quantify. A radiographic filling delay of one second or less during contrast injection in a diagnostic angiogram indicates the presence of robust collateral circulation. The extent of collateral circulation depends on genetic factors, chronicity of the vascular lesion, physiologic parameters such as perfusion pressure and capacity for autoregulatory dilatation. For example, an elderly patient with long standing atherosclerosis may have extensive anatomic collaterals given chronicity of the lesion. However, in contrast, a younger patient may have better physiological collateral circulation resulting from greater capacity for autoregulatory dilatation. A perfusion scan indicates the adequacy of collaterals to keep the ischemic tissue viable. The presence of extensive and robust collaterals have been associated with a higher likelihood of recanalization and reduced infarct volume even with treatment failure.

Blood Pressure Variability
Blood Pressure variability in the setting of an acute ischemic stroke is due to a complex interplay between autonomic regulation and arterial wall mechanical properties. Over 60% of patients with acute ischemic strokes experience hypertension post-infarct and managing blood pressure peaks and fluctuations are clinically difficult to interpret due to an existing evidence gap regarding expected blood pressure trajectory for each degree of recanalization. For example, incomplete recanalization may result in increased intracranial vascular resistance and subsequent blood pressure variability. Likewise, patients who present with less blood pressure variability experience better short-term and long-term functional outcomes. Blood pressure variability can be measured using several statistical formulae including standard deviation (SD), coefficient of variation, successive variation (SV) and average real variation (ARV). Unfortunately, even time-weighted averages of relatively high-frequency BP data appear to be insufficient in presenting the successive variability. While a few measures like SD are dependent on mean blood pressure, parameters like SV and ARV take into account point to point variations. These parameters also take into account rapid peaks in BP.

Blood Pressure Trajectory
A study looking at the natural history of blood pressure following MT in sixty-eight patients with LVO strokes was reported by in 2016. It was observed that there is an initial drop in BP in all patients within first 8 to 12 hours, which is followed by a plateau phase of a steady BP trajectory. The initial drop was greater in recanalized patients compared to the non-recanalized patients. Recent reports have identified five distinct blood pressure trajectories following LVO strokes and have proposed how these trajectories may determine 90-day functional outcomes in patients. These trajectories were named as low, moderate, moderate to high, high to moderate and high. The last two trajectories were associated with higher odds of an unfavorable 90 day-outcome. Higher blood pressure trajectories may represent either a re-occlusion or an impaired perfusion dynamic in the affected tissue.
Current Guidelines
Due to the distinct pathophysiology underlying the problem, it makes more sense to discuss the current guidelines under the three phases following an acute ischemic stroke: (a) Pre-recanalization phase or Initial Assessment phase encountered in the emergency department, (b) Re-recanalization phase or procedure phase, when the patient is in the angiography suite for an interventional neuro-vascular approach to treat thrombectomy, and (c) Early post-recanalization phase, the first 24-hour following thrombectomy, when the patient is managed in the ICU after the procedure. (Fig. 2)

Pre-recanalization Phase (Initial Assessment/Admission Phase)
A drop in systolic blood pressure of >50 mmHg over 24 hour period or an acute drop of >30 mmHg may worsen overall outcomes. 54 Acute drop in BP in this study was defined as the largest drop in BP between a measurement and one immediately preceding it. BP data was acquired every 15 minutes in first 2 hours after presentation, every 30 minutes in the next 6 hours and hourly until the first 24 hours after presentation. 55 Also, in another study, a drop in systolic blood pressure to less than 110 mm Hg, was shown to result in increased mortality. 56

The 2019 AHA/ASA guidelines for early management of acute ischemic stroke introduced a few new recommendations regarding blood pressure management. The recommendations discussed in this section are Class I recommendations unless stated otherwise. Table 1 summarizes the meaning of each level of recommendation. In patients presenting with higher blood pressures, the blood pressure should be lowered to <185/110 mm Hg before fibrinolytic treatment. A class IIa recommendation is proposed to maintain blood pressure ≤ 180/105 mm Hg for patients with planned MT, who did not receive tPA. The efficacy of drug-induced hypertension is not established (Class IIb). Post tPA treatment, the blood pressure should be maintained <180/105 mm Hg for the first 24 hours. 54 In patients with presenting blood pressure ≥ 220/110 mm Hg, it is customary to identify any co-morbid conditions including concomitant acute coronary event, acute heart failure, aortic dissection, postfibrinolysis sICH, or preeclampsia/eclampsia, which will require lowering blood pressure. In the absence of any of the above comorbidities, the benefit of initiating or reinitiating anti-hypertensive treatment is not reported. Therefore, AHA/ASA guidelines propose a class II recommendation to lower the blood pressure only minimally, <15% of baseline in these patients. For patients who present with blood pressure <220/110 mm Hg, who did not receive tPA or MT and did not have any comorbidities, the guidelines recommend against lowering the blood pressure from baseline. 54 (Figure 2)

Re-canlization Phase (MT Procedure Phase)
The Society of Neuroscience in Anesthesiology and Critical Care (SNACC) recommends SBP to be maintained between 140 and 180 mmHg during MT. 57 Abrupt drops in blood pressure > 40% of baseline as well as a drop in mean arterial pressure (MAP) below 70 mm Hg have been associated with poor outcomes. 58 The type of anesthesia used during MT could possibly confound outcomes. Initial retrospective observational studies suggested that conscious sedation may render better outcomes when compared to general anesthesia which may delay recanalization. 59-61 However, in recent prospective trials, where general anesthesia and conscious sedation were compared in randomized studies, such as, Sedation vs. Intubation for Endovascular Stroke Treatment (SIESTA) and General or Local Anesthesia in Intra-arterial Therapy (GOLIATH), general anesthesia was associated with a higher rate of favorable 90-day functional outcomes. Even though the time to groin puncture was slightly higher in general anesthesia in the GOLIATH trial, the overall time to recanalization was improved. 62-64 Looking pragmatically, as long as there is an aggressive blood pressure control as described above, general anesthesia and conscious sedation, may remain the clinical equipoise for procedural anesthesia care during MT.

Post-recanalization Phase
In the absence of a randomized controlled trial (RCT) data for post-recanalization BP management following MT, AHA-ASA guidelines propose a class II recommendation to keep SBP ≤ 180/105 during the procedure and in the first 24 hours following the procedure. For completely recanalized patients, the SBP could be kept <180/105 mm Hg (Class II b). 54 In spite of the guidelines, the current practice is to keep the SBP ≤ 140 mm Hg to 160 mm Hg as used in the DAWN trial. 55 65, 66 Also, a more intensive BP control is intuitively pursued in patients with adjunct extracranial or intra-cranial lesions treated with stents to minimize reperfusion injury. 67 AHA/ASA guidelines based SBP goal is arbitrarily set higher in patients with incomplete reperfusion to ensure perfusion to the penumbra through collaterals. However, we now see emerging evidence for post-recanalization BP management that may require incorporation into the subsequent AHA/ASA guidelines. An initial retrospective study in 2017 reported that higher blood pressures may relate to worse outcomes and a 10 mm Hg increase in maximum SBP within the first 24 hours after MT was an independent predictor of both worse functional independence and increased mortality at 3 months. 66 This study also found that moderate blood pressure control <160/90 mm Hg during the first 24 hours post-MT was associated with lowest 3-month mortality when
compared to the groups with intensive blood pressure control <140/90 mmHg as well as with permissive hypertension (BP <180/105 mmHg following tPA or MT, or <220/110 mmHg in patients without any acute intervention). The criticism to this study was that it was a retrospective study and it was unclear if blood pressure was the cause for poor outcomes, or a mere indicator. The first multi-center prospective study in this regard is the Blood Pressure after Endovascular Therapy for Ischemic Stroke, BEST trial which enrolled 485 patients in 12 centers across the US from November 2017 to July 2018. The study showed that a peak SBP of >158 mm Hg within the first 24 hours post re-canalization increased the likelihood of poor 90-day functional status. Most patients in this sample were fully re-canalized (Thrombolysis in Cerebral Infarction score, TICI 2b or 3 score). There is no clear evidence regarding blood pressure goals in partially re-canalized or non-recanalized patients. Intuitively, these patients may require higher blood pressure goals to ensure perfusion to the penumbra. Our group has demonstrated poor discharge outcomes among the successfully re-canalized patients who have sustained hypoperfusion for >12 hours as well as those with higher blood pressure variability in the first 24 hours following MT. Similarly, in a post-hoc analysis of the BEST trial data, the patients with higher blood pressure variability within the first 24 hours had poor 90-day functional outcomes.

**Blood Pressure measurement and Anti-hypertensives**

As per AHA/ASA guidelines, blood pressure should be measured every 15 minutes in the first 2 hours following tPA or MT, every 30 minutes until the first 6 hours, and only hourly thereafter. AHA/ASA recommends a few fast-acting anti-hypertensives with short lasting effect to reach the above discussed blood pressure goals. These include intravenous scheduled bolus doses of Labetalol, Hydralazine or Enalaprilat, and continuous drip forms of Nicardipine and Clevidipine. These anti-hypertensives have demonstrated clinical equipoise and therefore selection may be based on ease of availability. Simultaneously, escalating oral or per tube use of antihypertensives allows blood pressure to be maintained within the goal range recommended. For blood pressure levels refractory to these treatments, or diastolic blood pressure >140 mmHg, intravenous Nitroprusside can be used in the continuous drip form.

Looking at the opposite end of the spectrum, we acknowledge that lower blood pressure levels can also be harmful. There are ongoing trials to examine the effect of induced hypertension using peripherally acting vasopressors like phenylephrine to ensure better perfusion to the penumbra. There is no conclusive data in favor of such treatment (Class IIb), meaning it is possible to improve low blood pressure levels with vasopressors, however the effects of pressor-based normalized blood pressure on clinical outcomes are not known at this point.

**Summary**

In summary, the current AHA/ASA guidelines recommend a blood pressure < 185/110 mm Hg before fibrinolytic treatment, and to maintain blood pressure ≤ 180/105 mm Hg in the first 24 hours thereafter if the patient received tPA and/or MT. However, in the light of BEST trial, and reflecting on parameters used in the DAWN trial, we propose the following Blood Pressure goals.

(a) For patients who are not recanalized (TICI 0), blood pressure limits of ≤ 180/105 should be permitted if these patients received tPA. One can be more liberal regarding blood pressure if these patients did not receive tPA and attempt to reduce the blood pressure by 15% of baseline only if presenting blood pressure is greater than 220/110 mm Hg. In patients who did not receive tPA, and could not be recanalized, induced hypertension is a consideration.

(b) For patients who are partially recanalized (TICI 1, 2a), the blood pressure goal should be ≤ 160/90 mm Hg. Higher SBP up to 180 mm Hg may be permitted in cases of neurological deterioration. It is noteworthy that higher blood pressure variability and disruption of autoregulation may persist longer in these patients. Acute blood pressure drops in this group of patients may be detrimental.

(c) For patients who are completely recanalized (TICI 2b, 3), blood pressure goals should be ≤ 160/90 mm Hg. Greater blood pressure variability may be a predictor of poor outcomes in these patients. Therefore, acute peak and fall in blood pressure should be avoided in this group.

(d) More intensive blood pressure goals (<140 mm Hg) may be required for patients who are fully re-canalized but have symptomatic intracerebral hemorrhage (ICH) or ICH on imaging. A significant, but slow drop in blood pressure is seen in the early post-recanalization phase and more in successfully recanalized patients as compared to non-recanalized patients. Thereafter, a plateau in blood pressure is achieved after 10-12 hours of re-canalization. The physiological mechanism behind this phenomenon is unclear. There is emerging evidence that blood pressure trajectories following recanalization may be guided by the level at which autoregulation is functioning. While traversing these trajectories, the blood pressure varies from point to point. Post-recanalization phase blood pressure variability has been shown to determine discharge disposition as well as 90-day functional outcomes.
Hemodynamic management of LVO stroke patient requires close blood pressure monitoring and its careful management for at least the first 24 hours after MT. Emerging research utilizing continuous, noninvasive methods to identify cerebral autoregulatory ranges, and the use of multimodal monitoring to assess real time blood pressure variability may guide the personalized management of blood pressure. In this narrative review, we aimed to summarize the blood pressure management guidelines for the care of LVO stroke patients with specific emphasis on blood pressure variability.

REFERENCES
3. Center for disease control and prevention national center for health statistics. 2020
6. Zivin JA. Acute stroke therapy with tissue plasminogen activator (tpa) since it was approved by the u.S. Food and drug administration (fda). Ann Neurol. 2009;66:6-10


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**Fig. 1. Cerebral Autoregulation Curve.**

Note the cerebral autoregulation curve and auto-regulatory plateau in the normal brain (1). The curve 2 depicts upward shift with a narrower plateau in hypercapnia, curve 3 depicts rightward shift in chronic hypertension, and curve 4 depicts a downward shift in hypocapnia. Plots 5 and 6 show the loss of autoregulation in ischemic penumbra and core respectively. [Modified from Meng & Gelb, and prepared by Dr. S. Das]

**Fig. 2. Blood Pressure goals and anti-hypertensive treatment options in different phases of a Large Vessel Occlusion stroke.**

(tPA- tissue Plasminogen Activator, BP- Blood Pressure, MT- Mechanical Thrombectomy, TICI- Thrombolysis in Cerebral Infarction Score, Rx- Treatment) [Modified from Vitt, Trillanes, & Hemphill, and prepared by Dr. S. Das]
### Table 1. Classes of Recommendation

<table>
<thead>
<tr>
<th>Class of Recommendation</th>
<th>Interpretation</th>
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<tbody>
<tr>
<td>I</td>
<td>Presence of evidence, general agreement or both that a given treatment is useful or effective.</td>
</tr>
<tr>
<td>II a</td>
<td>Presence of conflicting evidence, a divergence of opinion, or both about the usefulness/efficacy of a procedure or treatment. However, usefulness is well established.</td>
</tr>
<tr>
<td>II b</td>
<td>Presence of conflicting evidence, a divergence of opinion, or both about the usefulness/efficacy of a procedure or treatment. The usefulness is not well-established.</td>
</tr>
<tr>
<td>III</td>
<td>No benefit. Not recommended.</td>
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<tr>
<td>IV</td>
<td>Potentially harmful.</td>
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### Table 2. Blood Pressure Management in Large Vessel Occlusion Strokes

<table>
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<tr>
<th>Current Recommendations</th>
<th>Reference</th>
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<tr>
<td><strong>Initial Management</strong></td>
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<tr>
<td>• BP levels up to 220/120mmHg permitted to allow perfusion to ischemic site</td>
<td>Powers, WJ et al Stroke, 2018</td>
</tr>
<tr>
<td>• BP needs to be lowered to &lt;185/110mmHg for the tPA and MT candidates</td>
<td></td>
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<tr>
<td>• Hypotension worsens outcomes</td>
<td></td>
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<tr>
<td>o BP drops up to 15% considered within safety limits</td>
<td></td>
</tr>
<tr>
<td>o SBP drop of &gt;50 &amp; acute SBP drop &gt;30mmHg may worsen functional outcomes</td>
<td>Silver, B et al J Neurol Science, 2008 Maier, B et al J Am Heart Assoc, 2017</td>
</tr>
<tr>
<td>o SBP&lt;110mmHg is associated with increased mortality</td>
<td></td>
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<tr>
<td><strong>Revascularization Procedure (Mechanical Thrombectomy)</strong></td>
<td>Talke, PO et al J Neurosurg Anesthesiol, 2014</td>
</tr>
<tr>
<td>• Maintain SBP 140-180mmHg during the procedure</td>
<td></td>
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<tr>
<td>• MAP ≥ 70mmHg at all times</td>
<td></td>
</tr>
<tr>
<td>• Maximum up to 40% of drop in BP to be allowed for hypertensive patients</td>
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</tbody>
</table>
| • If successful reperfusion (TICI 2b/3) maintain SBP<160mmHg, but if hemorrhagic conversion, maintain SBP<140mmHg | Mistry, EA et al Stroke, 2020  
Jovin, TG et al Int J Stroke, 2017  
Nogueira, RG NEJM, 2018 |
<table>
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<tbody>
<tr>
<td>• In case of incomplete reperfusion (TICI 0-2a), maintain BP &lt;180/105mmHg for at least 24h</td>
<td>Mistry, EA et al Stroke, 2020</td>
</tr>
<tr>
<td>• Induced hypertension can be considered for incomplete reperfusion patients</td>
<td></td>
</tr>
<tr>
<td>• In case of end organ hypoperfusion or change in neurologic examination, consider maintaining BP at a higher range</td>
<td>Powers, WJ et al Stroke, 2018</td>
</tr>
</tbody>
</table>

BP: blood pressure; SBP: systolic blood pressure; MAP: mean arterial blood pressure; tPA: tissue plasminogen activator; MT: mechanical thrombectomy; TICI: thrombolysis in cerebral infarction scale