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**Anaesthesia and haemodynamic management of acute ischemic stroke patients before, during and after
endovascular therapy**

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Abstract: Endovascular therapy (EVT) is now standard of care for eligible patients with acute ischemic stroke caused by large vessel occlusion in the anterior circulation. EVT can be performed with general anaesthesia (GA) or with monitored anaesthesia care, involving local anaesthesia with or without conscious sedation (LA/CS). Controversies remain regarding the optimal choice of anaesthetic strategy and observational studies suggested poorer functional outcome and higher mortality in patients treated under GA, essentially because of its haemodynamic consequences and the delay to put patients under GA. However, these studies are limited by selection bias, the most severe patients being more likely to receive GA and recent randomised trials and meta-analysis showed that protocol-based GA compared with LA/CS is significantly associated with less disability at 3 months. Unlike for intravenous thrombolysis, few data exist to guide management of blood pressure (BP) before and during EVT but arterial hypotension should be avoided as long as the occlusion persists. BP targets following EVT should probably be adapted to the degree of recanalisation and the extent of ischemia. Lower BP levels may be warranted to prevent reperfusion injuries even if prospective haemodynamic management evaluations after EVT are lacking.

Keywords: Acute ischemic stroke; endovascular therapy; general anaesthesia; monitored anaesthesia care; haemodynamics; blood pressure

1. Background

Stroke is a major global health issue with an overall growing incidence, particularly in low to middle income countries [1]. Approximately 15 million of people worldwide are victims of stroke each year, there are more than 25 million stroke survivors across the world and stroke is the leading cause of acquired disability among adult population and the second leading cause of dementia after Alzheimer disease [1, 2]. In France, stroke affects around 150,000 patients each year, leading to more than 100,000 hospitalisations and 30,000 deaths [3, 4].

Approximately 85% of stroke patients have acute ischemic stroke (AIS), and timely restoration of cerebral blood flow (CBF) using reperfusion therapies is the mainstay of AIS treatment. For eligible patients presenting within 4.5 hours since last known well, the recommended first-line therapy is intravenous thrombolysis (IV-TL) with tissue plasminogen activator (tPA) [5, 6]. tPA reduces rate of functional dependence in up to one third of patients and results in a better functional prognosis. In addition, some trials suggest that patients who have ischemic stroke and salvageable brain tissue on perfusion imaging may also benefit from IV-TL between 4.5 and 9.0 hours, despite higher occurrence of cerebral haemorrhage [7]. However, many AIS patients do not benefit from this treatment because of its restricted therapeutic window or a contraindication to fibrinolytic administration [8, 9].

More recently, pivotal randomised trials and meta-analysis showed that intra-arterial therapy with endovascular therapy (EVT), performed within 6 to 12 hours of last known well in large cerebral vessels of anterior circulation, leads to significant improved functional outcomes, without increasing the risk of cerebral haemorrhage [10-17] (Table 1). In addition, two randomised trials demonstrated that MT for AIS can even benefit patients with favourable mismatch pattern on perfusion imaging until 16 to 24 hours after stroke onset [18, 19], and MT has now become the standard of care for AIS patients with anterior circulation large vessel occlusion (LVO) [5, 6].

However, there remain several unresolved issues regarding the optimal management of AIS patients eligible for EVT, particularly regarding the optimal anaesthetic and haemodynamic management. Therefore, the aim of this review article is to discuss available data concerning anaesthesia and haemodynamic management before, during and after EVT for AIS to assist specialised as well as non-specialised anaesthesiologists in charge of AIS patients.

2. Cerebral pathophysiology after AIS

In healthy condition, the cerebral blood flow (CBF) is adapted to meet local metabolic demand and any prolonged reduction in CBF will be responsible for neuronal dysfunction. If CBF remains low, irreversible injury will lead to infarction (cellular death). However, cerebral ischemia is a focused phenomenon and ischemic

penumbra has been widely described as a severely hypoperfused, functionally impaired, but still viable parenchyma which surrounded core infarction [20]. While the ischemic core is permanently damaged, ischemic penumbra can regain its function if rapidly reperfused and restoring CBF within penumbra in a timely manner is the cornerstone of recanalisation therapies (“time-is-brain”).

Normally, CBF is maintained thanks to a protective mechanism known as cerebral autoregulation (CA), whereby resistance level of cerebral arteries adapts to maintain a constant blood flow [21, 22]. An increase in systemic blood pressure (BP) will be compensated by a vasoconstriction (higher resistance level) while a drop in systemic blood pressure will induce vasodilatation (lower resistance level) [21, 22]. CBF is therefore maintained across a range of systemic blood pressures, as long as other determinants of cerebral vessels resistance are constant (i.e., partial pressure in carbon dioxide). Outside this range of pressure CBF becomes pressure-dependent and every change in systemic pressures will be followed by a proportional change in CBF [21, 22].

Studies evaluating the association between changes in CBF in proportion to modifications in BP have various results, but most of them show that drops in BP will reduce CBF and worsen the ischemia. CA within the penumbra is often impaired, thus making CBF directly dependent on systemic pressures within a highly vulnerable area [23], and both the magnitude and the duration of arterial hypotension have been shown to be harmful after an AIS [23, 24]. Additional factors may also play a role in the reduction of cerebral perfusion in the context of AIS, such as the quality and the number of collateral vessels or comorbidities (i.e., chronic hypertension) [24, 25], justifying a tailored management of blood pressure changes after AIS.

Reperfusion can contribute to significant brain damages in the form of oedema, haemorrhagic transformation or worsening ischemia [26, 27]. After recanalisation, there is often a sharp increase in CBF due to the impairment of CA and the release of vasodilatory substances [28]. This hyperperfusion state has been well described in both animal and human models by using perfusion imaging and favours vasogenic oedema and haemorrhagic transformation [29, 30], especially in this context of potential loss of blood brain-barrier integrity. On the other hand, there can also exist a hypoperfusion state after MT that can worsen cerebral ischemia, called no-reflow phenomenon, which is due to white substance and inflammatory injuries [31].

3. Current emergency treatment of acute ischemic stroke

3.1 Intravenous thrombolysis

Alteplase administration is recommended as the first-line treatment in patients with disabling stroke symptoms regardless of age and stroke severity [5, 6]. The safety and the efficacy of this treatment given within 4.5 hours of symptoms onset (or the last time to be known well) are supported by many trials and confirmed by an extensive community experience in many countries (Figure 1) [32]. IV-TL is also reasonable for patients with mild stroke presenting within the same time period and may be beneficial for wake-up stroke (unknown time

of onset) or selected AIS patients between 4.5 and 9.0 hours (Figure 1) [7, 33]. However, IV-TL suffers from limitations: Alteplase administration is limited by a restricted therapeutic window, the probability for complete recanalisation is influenced by the site of occlusion and it is lower for proximal occlusion [34], and there are contraindications to Alteplase administration (i.e., coagulopathy, use of direct oral anticoagulants, severe head trauma < 3 months, infective endocarditis, etc.) [5, 6].

3.2 Endovascular therapy

Early intra-arterial treatment with third-generation EVT devices (stent retrievers and suction catheters) is now indicated for patients with AIS due to LVO, regardless of whether they receive IV-TL for the same ischemic event [5, 6, 35]. Patients presenting within 6 hours of last known well with LVO of anterior circulation (internal carotid artery and/or the first segment [M1] of the middle cerebral artery [MCA]) should benefit from MT as quickly as possible (Figure 1) [5, 6]. In carefully selected patients, MT may also be reasonable for AIS with more distal anterior occlusions (i.e., MCA segment M2) or posterior circulation strokes within the same time period [5, 6]. Further prospective data are yet warranted. For selected patients who meet DEFUSE 3 or DAWN eligibility criteria (including favourable mismatch pattern on perfusion imaging), MT may be used during an extended time period (until 16 to 24 hours after last known well) (Figure 1) [5, 18, 19].

The access to EVT requires a critical step of patient's conditioning and supposes to choose an anaesthetic strategy. An EVT can be performed under general anaesthesia (GA) or with monitored anaesthesia care (MAC). MAC include local anaesthesia (LA), conscious sedation (CS) and the combination of the two (LA/CS). The anaesthesia team may be involved from the beginning of the outset or may be asked to assist in the event of a procedural complication or a MAC failure (i.e., agitation requiring conversion to GA). Because anaesthesia may affect the functional prognosis as well as the technical goal of MT, which is to obtain a successful reperfusion (modified thrombolysis in infarction scale [mTICI] $\geq 2b$) [36], the choice of an anaesthetic technique for MT has to be individualised and based on the technical performance of the procedure, patient risk factors and clinical characteristics that should be elicited during the pre-anaesthesia evaluation [5].

4. Phase I: initial assessment and supportive care

Pre-anaesthesia evaluation must be precise but also concise to minimise delays in care and should be conducted in close communication with other caregivers (i.e., intensivists, neurologists, neuroradiologists) within multidisciplinary stroke teams. Although it is recommended to perform some diagnostic tests (i.e., electrocardiogram, troponin), none should delay the initiation of recanalisation therapies and only an assessment of blood glucose must precede TL-IV [5, 6]. All patients admitted to the hospital with suspected acute stroke should receive brain imaging and a comprehensive intra and extracranial neurovascular evaluation on arrival (Figure 1) [5, 6]. In most cases, non-contrast computed tomography (CT) seems sufficient to detect acute intracerebral haemorrhage (ICH) and avoid antithrombotic treatments. Diffusion-weighted imaging (DWI)

sequences from magnetic resonance imaging (MRI) is more sensitive but its routine use in all AIS patients is not currently recommended [5, 6, 37]. Advanced CT and MRI technologies may be useful in cases of atypical presentation or diagnostic doubt and able to distinguish between brain tissue that is irreversibly infarcted and that which is potentially salvageable [5, 6], and perfusion imaging allows selection of patients who are likely to benefit from MT until 16 to 24 hours (Figure 1) [5, 6, 18, 19].

4.1 Specificities of the anaesthesia evaluation for stroke patients eligible to mechanical thrombectomy

EVT is an emergency procedure, requiring focused and concise pre-anaesthesia evaluation as well as rapid communication and smooth coordination between the stroke team, interventional radiologist, and anaesthetist. In addition to general considerations related to the conduct of anaesthesia in an emergency setting, numerous pre-anaesthesia concerns are more specific to AIS patients (Figure 2) [38-40]. The impairment caused by an AIS as well as its evolution should be assessed by using dedicated scale such as the National Institutes of Health Stroke Scale [41]. Because many AIS patients are over 60 years of age and the risk of stroke increases with age, AIS patients often have chronic health conditions that put them at high risk for complication, such as coronary disease, chronic hypertension or chronic obstructive pulmonary disease. Stroke itself can cause serious life-threatening complications such as myocardial infarction, adrenergic cardiomyopathy, arrhythmias, coma or seizure. The interventional radiology suite is often an off-site and less familiar location, which has limitations in terms of both physical space and range of anaesthetic equipment. Finally, the anaesthetist also has to consider the need for protective lead clothing, which can restrict movements and hinder the realisation of a GA. At this time, no detailed guidelines regarding the anaesthesia evaluation of EVT candidates exist and such tools should be settled locally by multidisciplinary teams according to local resources and staffs.

4.2 Supportive care and conditioning of AIS patients

4.2.1 Supportive care

Initial blood pressure management: Hypertension is very common in AIS with a large cohort study showing that 3 out of 4 patients had a SBP greater than 140 mmHg [42]. Severe hypertension is also relatively frequent with approximately 10% of AIS patients presenting with SBP > 200 mmHg and hypertension has been identified as risk factor for unfavourable outcome due to increased risk of ICH after thrombolysis, increased rate of early recurrence and cerebral oedema [24, 43, 44]. Elevated BP after AIS is multifactorial (i.e., chronic hypertension, stroke localisation, pain or agitation, etc.) but may also represent a compensatory mechanism to maintain cerebral perfusion in the case of severe AIS (i.e., those with poor quality of collateral vessels) and the risk of hypotension probably outweighs that of elevated BP as long as the occlusion of the cerebral artery persists. Low BP levels may indeed compromise penumbra perfusion and several studies have shown that there is a higher risk of poor outcome and/or mortality for low SBP during the acute phase of AIS [24, 44-46]. For

patients who have elevated BP and are otherwise eligible for IV-TL, it is only recommended to carefully lower their BP so that their systolic blood pressure (SBP) is < 185 mmHg and their diastolic blood pressure (DBP) is < 110 mmHg (Grade I, [5]) (Figure 3). Both high and low baseline BP seem to be harmful in patients eligible to EVT [24, 47, 48]. In such patients it is consequently reasonable to maintain BP under the same levels before the procedure (Grade IIa, [5]) (Figure 3), but arterial hypotension before MT should be avoided [5, 47, 48], even if the optimal BP that should be maintained before MT is still unknown. No studies have addressed the treatment of low BP in patients with AIS, there are no data to guide volume and duration of parenteral fluid delivery and no studies have compared different isotonic fluids. Only a systematic analysis of 12 studies comparing colloids with crystalloids showed no differences in terms of functional outcome [49].

Airway and respiratory management: Airway support and ventilatory assistance are recommended for the treatment of patients with acute stroke who have decreased consciousness or who have bulbar dysfunction that causes compromise of the airway (Grade I, [5]). Supplemental oxygen should be provided to maintain oxygen saturation > 94% (Grade I, [5]) but is not recommended in non-hypoxic patients with AIS (Grade III: No benefit, [5]).

Temperature: A retrospective and multicentric cohort study showed that peak temperature in ICU during the first 24 hours (< 37 °C and > 39 °C) was associated with an increased risk of in-hospital death compared with normothermia in 9,366 patients with AIS [50]. Consequently, causes of hyperthermia (temperature > 38°C) should be identified and treated, and antipyretic medications should be administered to lower temperature in hyperthermic patients with stroke (Grade I, [5]). Induced hypothermia is often discussed in the context of acute brain injury (i.e., head trauma). However, the benefit of induced hypothermia for treating patients with ischemic stroke is not well established and hypothermia should be offered only in the context of ongoing clinical trials (Grade IIb, [5]).

Blood glucose: High blood glucose levels during the first 24 hours increase the risk of cerebral haemorrhage and are associated with worse outcomes [51]. Therefore, it is recommended to treat hyperglycaemia to achieve blood glucose levels in a range of 140 to 180 mg.dL⁻¹ and to closely monitor to prevent hypoglycaemia (blood glucose < 60 mg.dL⁻¹) (Grade IIa, [5]).

4.2.2 Conditioning of AIS patients eligible to EVT

Standard monitoring (i.e., electrocardiogram, pulse oximeter, non-invasive blood pressure, temperature) should be used in all AIS patients. Capnography is a useful monitor for patients under either GA or deep to moderate sedation. During MAC, capnography can be used to monitor respiratory function, to diagnose airway obstruction and to avoid hypercapnia. Intra-arterial catheter (IAC) is an invasive and continuous BP assessment that allows a tighter haemodynamic management. However, IAC placement should not delay the initiation of the treatment and direct arterial pressure can be transduced from the arterial introducer placed for the

procedure. Two peripheral intravenous catheters prior to MT should be placed to facilitate rapid fluid administration, sedative agents and/or dedicated administration of vasoactive medications (Figure 4).

5. Phase II: during the mechanical thrombectomy

5.1 Is monitored anaesthesia care the gold standard for mechanical thrombectomy?

5.1.1 Rational to promote LA/CS over GA

LA/CS has been widely used in studies validating the efficacy of MT (90.9% in the ESCAPE study and 63% in the SWIFT PRIME study [12, 13]) and is often associated with favourable functional outcome. GA is potentially responsible of a significant drop in BP and the time to realise it can delay the timing of recanalisation: the effect of MT treatment in the ESCAPE study was greater than the one in the MR CLEAN trial with fewer patients treated with GA (9.1% *versus* 37.8%) and a post hoc analysis of the MR CLEAN study showed a 51% (95% CI, 31-86) decrease in the effect of treatment under GA [52]. A meta-analysis of individual patient data confirmed these results, but included studies primarily focused on the efficacy and the safety of EVT, and not on the anaesthetic strategy itself [53].

In recent years, many studies have focused specifically on anaesthesia for EVT and most have shown that GA was associated with a poorer functional prognosis. As early as 2010, Abou-Chebl et al. showed that GA was an independent factor of poor functional outcome and mortality [54]. Between 2010 and 2017, more than 20 studies evaluating the impact of anaesthesia on patient outcome showed similar results. First systematic reviews and meta-analysis comparing the different anaesthetic approaches for MT confirmed these results [55-57]. For example, Brinjikji et al. showed that GA was associated with a significant lower odds of favourable functional outcome (OR, 0.58; 95% CI, 0.48-0.54) and that GA patients had higher odds of 90-day mortality (OR, 2.02; 95% CI, 1.66–2.45), vascular complications (OR, 1.43; 95% CI, 1.01–2.03), and respiratory complications (OR, 1.70; 95% CI, 1.22–2.37) [57]. In addition, the recanalisation rate was not significantly different between both groups and patients treated under GA had more often symptomatic intracranial haemorrhage (OR, 1.31; 95% CI, 1.01-1.70) [57]. For the authors, main arguments against GA were the hemodynamic effects of GA and the delayed groin puncture in comparison with LA/CS [56, 57].

5.1.2 Methodological issues of early studies evaluating anaesthesia for EVT

Yet, most of studies evaluating anaesthesia for MT after AIS were observational and retrospective. Some of them included a majority of patients treated under sedation [58, 59], sometimes endovascular procedures were not performed using stent retriever or thromboaspiration [60, 61], some studies included patients presenting AIS of posterior circulation [59, 62], details regarding the anaesthetic protocol and/or the type of endovascular treatment were not provided [63], details about the cardiovascular history of patients were

missing [64], the conversion rate of LA/CS to GA was highly variable and sometimes unspecified [65, 66] and, finally, it was often the most severe patients (higher NIHSS score) who received GA (selection bias).

For example, Davis et al. compared patients treated with GA or LA/CS and showed that the proportion of patients with favourable outcome was 15% with GA *versus* 60% with LA/CS ($P < 0.001$) and that the lowest systolic BP (SBP) was correlated with GA ($r = -0.7$, $P < 0.001$). Patients with severe stroke (i.e., NIHSS > 15) were more often treated with GA, supporting the idea of selection bias (confounding by indication) [62].

5.1.3 Arterial hypotension under conscious sedation

In addition, hypotension may occur under conscious sedation, especially if the anaesthetic agents used are the same as those used for GA (i.e., propofol). In the AnStroke study, a recent randomised trial evaluating the impact of anaesthesia on functional outcome after EVT, 60% of patients treated with CS (remifentanyl target control infusion) had a decrease in MAP of 20 mmHg or more and 79% of them required the use of vasopressor [67]. Although the study protocol included specific BP objectives leading to early introduction of vasopressors, these results show that a significant number of patients may have arterial hypotension under sedation. This effect may be more pronounced in studies where patients under CS received the same anaesthetic agents as those used for GA, only at lower dose.

Whalin et al. also showed that patients treated with CS could be hypotensive and that this hypotension was deleterious. They retrospectively studied 256 patients with MAC. Patients received dexmedetomidine (0.5 to 1.0 $\mu\text{g}\cdot\text{kg}^{-1}$ then 0.5 to 1.0 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$), eventually with an injection of fentanyl and/or midazolam. The haemodynamic objectives were to maintain a SBP > 140 mmHg and a mean blood pressure (MBP) > 70 mmHg. In this study, SBP decreased by 35.5 mmHg (IQR, 17-56), a decrease of 22.8% (IQR, 13-34), and MBP decreased by 24.7 mmHg (IQR, 13-39), a decrease of 23.3% (IQR, 13-34). One hundred and thirty-four (52%) patients received vasopressors. The authors pointed out that the duration of hypotension under CS would potentially be longer than under GA, the anaesthesiologist waiting to introduce vasopressor agents in non-intubated patients [68]. Therefore, optimisation of haemodynamic stability may be more important than only focusing on the type of anaesthesia [69].

5.2 Benefit/risk balance and definition of general anaesthesia

GA is required in obvious situations such as coma or respiratory failure. GA may reduce the risk of aspiration and may prevent patient movement, making the procedure safer and more technically feasible. The longer delay to put patients under GA could be compensated by a shorter procedural time: in the SIESTA trial, the duration of MT was 111.6 min with GA and 129.9 min with LA/CS ($P = 0.04$) [70]. Similarly, Vukasinovic et al. demonstrated that a well-organised workflow is associated with reasonable delay in performing GA (5 to 10 minutes), with no impact on final recanalisation time and no difference in functional outcome [71]. Moreover,

starting a procedure under LA/CS in patients presenting risk factors of sedation failure (i.e., agitation, conscious impairment, expected long procedure) exposes them to the risk of emergency GA conversion (in unfavourable conditions) with relatively high doses of anaesthetics that increase the risk of hypotension [72]. The benefit/risk balance of GA *versus* MAC for EVT is exposed in Table 2.

5.3 General anaesthesia with haemodynamic objectives

5.3.1 Which blood pressure thresholds during EVT

Many studies have shown that low BP during MT for stroke is associated with a poor functional outcome [62, 68, 73, 77]. Some of them concerned only patients treated with GA [74,76], patients treated with GA or LA (or mild sedation) [62, 77], or patients under MAC sedation with dexmedetomidine [68,73]. A systematic review evaluating the influence of per procedural BP on functional outcome after MT also suggests that BP drops during MT may be associated with a worse functional outcome, and three types of BP drops were found to be predictors of poor functional outcome at 3 months: > 40% drop in mean arterial pressure compared with baseline (odds ratio = 2.8; [1.09-7.19]; $P = 0.032$), lowest mean arterial pressure before recanalisation (odds ratio = 1.28; [1.01-1.62] per 10 mm Hg drop below 100 mm Hg; $P = 0.04$), and MAP drops (odds ratio = 4.38; [1.53-12.6] for drops > 10%) (Figure 3) [78].

5.3.2 Randomised trials evaluating anaesthesia for EVT

Recently three randomised controlled clinical trials comparing patients treated with EVT either under GA or LA/CS have been published [67, 70, 79] (Table 3). They all included strict haemodynamic objectives (i.e., SBP targets) during the procedure. The Swedish AnStroke study showed that the functional outcome at 90-day (mRS) was similar regardless of the anaesthetic management [67], the German SIESTA study did not show any difference in the improvement of the 24-hours NIHSS score between the GA group and the sedation group [70], and the Danish GOLIATH study did not show a significant impact of anaesthesia on the growth of the infarction size measured on MRI at 2/3-day post EVT [79].

In addition, these studies showed numerous positive signals in favour of GA. In both the SIESTA and the GOLIATH trials, the recanalisation rate was significantly higher in the GA group (89% *versus* 80.5% and 76.9% *versus* 60.3%, respectively) [70, 79]. In these same two studies, and despite being a secondary endpoint, the 90-day functional outcome assessed by the modified Rankin score was significantly better and, in the GOLIATH trial, the final volume of infarction was significantly smaller in the GA group (22.3 ml *versus* 38 ml, $P = 0.04$) [79]. Furthermore, while the time to groin puncture was slightly prolonged in the GA group, the overall time to reperfusion was significantly decreased in the GOLIATH study [79]. These studies highlighted the relevance of adopting an aggressive haemodynamic management during MT for AIS. While arterial hypotension (drops in MBP > 20%) were more frequent under GA, there was no significant difference in large falls in MBP (i.e., drops

in MBP > 40% or MBP < 70 mmHg) [67, 79]. Finally, risk of LA/CS failure was not negligible and conversion into GA reached 15.6% in the AnStroke trial [67]. However, these results should be interpreted with caution. First, these are small and monocentric studies including fewer than 150 patients each. Then, all these trials included meticulous haemodynamic protocols, based on the recommendation of the Society of Neuroscience in Anaesthesiology and Critical Care [80], which incentivised early introduction of vasopressors (even for non-GA patients).

Both anaesthetic regimens seem to be reasonable approaches as long as there is a strict control of haemodynamic parameters, such as BP level, and an organisation to minimise delays in care before the recanalisation (Figure 4). Recent reviews and meta-analysis confirmed that the use of either GA or CS during MT for AIS of anterior circulation does not yield significantly different rates of functional independence at 3 [81,83]. Furthermore, a systematic review and meta-analysis of individual patient data from SIESTA, AnStroke and GOLIATH trials was recently published [84]. As a primary outcome measure, mean 3-month mRS was better in the GA group compared to the CS group (2.8 [95% CI, 2.5-3.1] vs 3.2 [95% CI, 3.0-3.5], $P = 0.02$). Similarly, 3-month functional independence (mRS 0-2) was better under GA vs CS (49.2% vs. 35.1%; OR, 2.16 [95% CI, 1.31-3.54]; $P = 0.003$). For the authors, the clinical benefits associated with GA may be primarily related to the higher rate of reperfusion in the GA group (mTICI \geq 2b: 72.7% vs 63.2%; OR, 1.84 [95% CI, 1.12-3.01]; $P = 0.02$), although more hypotension and BP variability occurred in patients who received GA. Finally, despite a slightly longest time for groin puncture in the GA group, time to reperfusion did not differ between groups, neither did procedural complications such as vessel perforation or nosocomial pneumonia. However, the more favourable outcome associated with GA were no longer significant in the case of conversion from CS to GA (11.5%), and further research is needed to identify risk factors that could predict when primary GA is necessary to reduce the number of emergency conversions.

5.3.3 Management strategies for arterial hypotension during mechanical thrombectomy

In the case of low BP, a preventive use of diluted vasopressor such as norepinephrine (dilution of 5 to 10 $\mu\text{g.ml}^{-1}$) should be made, as suggested by Futier et al. for general surgery [85], rather than using neosynephrine or fluid expansion alone. One of the two peripheral intravenous catheters placed for MT should be dedicated to the administration of vasoactive medications (See 4.2.2, Figure 4).

6. Phase III: following revascularisation

6.1 Blood pressure after EVT

Recent society guidelines recommend to maintain a BP < 180/105 for at least 24 hours after cerebral recanalisation therapy (Figure 3) [5], and some neurocritical care experts suggest the systolic BP to be < 140

mmHg and the mean BP to be > 70 mmHg to ensure sufficient perfusion of ischemic brain while mitigating risks of haemorrhagic transformation and reperfusion injuries [86].

High BP levels over the first 24-48h after MT have been identified as a risk factor for intracranial haemorrhage and worse functional outcome, regardless of the recanalisation status [87, 88]. In addition, both elevations in BP and BP variability are more common in AIS patients with incomplete recanalisation and have been correlated with haemorrhagic transformation and unfavourable outcome, whereas BP often spontaneously declines after a successful reperfusion [89, 90]. However, it is not clear whether significant high (or variable) BP levels are themselves risk factors for brain injury or simply a marker of stroke severity because of the observational character of these studies and data concerning the rapidity and the extent to which the BP should be lowered without compromising brain perfusion are lacking. A randomised controlled trial comparing two BP levels after recanalisation is ongoing (NCT03160677).

6.2 “One size fits all” versus “tailored” blood pressure target

A “one size fits all” BP target could make sense from a pathophysiological standpoint after IV-TL because (1) individual recanalisation status is often unknown in the clinical setting and (2) the rate of recanalisation is often low (approximately 20%) in studies including angiography [91]. Conversely, MT is associated with recanalisation in 70-80% of cases and this can be confirmed directly during the procedure by using the Thrombolysis in Cerebral Infarction (TICI) score [36]. In recanalised patients, the risk of reperfusion injury at BP levels close to 180/105 probably outweighs the risk of hypoperfusion at lower BP targets and several studies have shown that patients with complete reperfusion have a better functional prognosis at lower BP levels (Figure 3) [92,94].

Indeed, elevated BP and increased CBF may be associated with cerebral hyperperfusion syndrome (CHS), characterised by headaches, haemorrhagic transformation and epileptic seizures [95]. The exact mechanism of CHS is still imperfectly understood, but CHS may result of cerebral vessel injuries and loss of CA (see Part 2.) [29, 30, 96]. In the case of AIS patients who suffer CHS following complete recanalisation, carefully lowering systolic BP < 140 mmHg might be safe as long as there is no associated severe ($\geq 70\%$) carotid (or basilar) stenosis (Figure 3) [24]. Short-acting and easy-to-use antihypertensive agents (i.e. labetalol - α and β blocker - or nicardipine - dihydropyridine calcium channel blocker-) are preferable in the acute setting to rapidly achieve haemodynamic goals and avoid prolonged hypotension [24]. As with patients presenting CHS, patients with hypoperfusion (i.e., no-reflow phenomena) should benefit from a tailored BP target where induced hypertension may be warranted [97].

Therefore, physicians could systematically adopt tailored BP targets after MT based on individual conditions such as the degree of recanalisation, the presence of an associated carotid (or basilar) stenosis or the fluctuation of the neurological examination in the context of changes in systemic BP. Even if there is no

consensus regarding the management of haemodynamic disturbances after MT, such conditions could be identified with multiple available modalities, including cerebral blood flow sonography, cerebral perfusion imaging and measures of the cerebral oxygenation [98, 99].

7. Conclusion

EVT studies bring new clinical context of acute recanalisation after AIS and confirmed the importance of BP management for the procedure. Arterial hypotension should be avoided as long as the occlusion persists and BP targets following EVT should probably be adapted to the degree of recanalisation and the extent of ischemia. Even if prospective haemodynamic management evaluations after EVT are lacking, lower BP levels may be warranted to prevent reperfusion injuries after EVT. Among patients with AIS of anterior circulation eligible to EVT, the use of protocol-based GA with haemodynamic objectives are significantly associated with less disability at 3 months. However, LA/CS is a viable approach in some patients and further research is warranted to identify risk factors that could predict when primary GA is necessary to reduce the number of emergency conversions.

Ethics committee approval

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Disclosure of interest

The authors declare that they have no competing interest.

Availability of data and material

Data sharing is not applicable to this review article as no datasets were generated or analysed during the current study.

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Legends of figures

Figure 1. Early management and emergency treatment of acute ischemic stroke (AIS). *Disabling stroke defined by a National Institute of Health Stroke Score (NIHSS) > 6. **Cerebral non-contrast computed tomography (NCCT) scanning is sufficient in most patients because of the detection of intracerebral haemorrhage (ICH). Diffusion-weighted magnetic resonance imaging (DW-MRI) is more sensitive and may guide the management of some patients with negative NCCT (i.e., puzzling clinical presentations). ***Mechanical thrombectomy may be reasonable for other carefully selected patients (i.e., posterior strokes). According to: American Heart Association/American Stroke Association 2018 Guidelines for the Early Management of Patients with Acute Ischemic Stroke [5]. ASPECTS: Alberta Stroke Program Early Computed Tomography Score; BP: blood pressure; CTP: CT perfusion; ICA: internal carotid artery; IV: intravenous; MCA: middle cerebral artery; mRS: modified Rankin score.

Figure 2. Clinical preanesthetic considerations of ischemic stroke patients eligible to endovascular therapy. *The National Institutes of Health Stroke Scale (NIHSS) is a widely used tool for neurologic examination in the setting of AIS [5, 6, 41]. The score identifies disabling stroke (NIHSS > 6) and is strongly correlated to stroke outcome, even it does not capture all stroke-related impairments and may underestimate the severity of posterior circulation strokes. COPD: Chronic obstructive pulmonary disease; mRS: modified Rankin score.

Figure 3. Blood pressure (BP) management before, during and after endovascular therapy (EVT) for acute ischemic stroke. Dashed lines: no consensus regarding the optimal BP threshold.

According to: American Heart Association/American Stroke Association 2018 Guidelines for the Early Management of Patients with Acute Ischemic Stroke [5], Society for Neuroscience in Anesthesiology and Critical Care Expert Consensus Statement: Anesthetic Management of Endovascular Treatment for Acute Stroke [80]. IV: intravenous; TICI: Thrombolysis in Cerebral Infarction (≥ 2 b: successful reperfusion).

Figure 4. Anaesthesia and conditioning of acute ischemic stroke patients eligible to endovascular therapy (EVT). EVT can be performed with general anaesthesia (GA), or with monitored anaesthesia care (MAC), including local anaesthesia with or without conscious sedation (LA/CS). The choice of anaesthesia for EVT has to be individualised and based on the technical performance of the procedure, patient risk factors and clinical

characteristics [5]. Arterial hypotension should be avoided, regardless of the anaesthesia used. *diluted norepinephrine (NE): 5 to 20 $\mu\text{g}\cdot\text{ml}^{-1}$. **i.e., dexmedetomidine: 0.5 to 1.0 $\mu\text{g}\cdot\text{kg}^{-1}$ then 0.5 to 1.0 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$, or fentanyl or midazolam [68]. ***i.e., propofol target control infusion (TCI) (site effect): [3.0-6.0 $\mu\text{g}\cdot\text{ml}^{-1}$] and remifentanyl TCI (site effect): [3.0-6.0 $\text{ng}\cdot\text{ml}^{-1}$] with suxamethonium or rocuronium (1 $\text{mg}\cdot\text{kg}^{-1}$). BP: blood pressure; CNAP: continuous non-invasive arterial pressure; etCO₂: End-tidal carbon dioxide concentration; IAC: intra arterial catheter; ICA: ICA: internal carotid artery; IV: intravenous access; RSI: rapid sequence induction; SatO₂: arterial oxygen saturation.

Tables

	MR CLEAN Berkhemer OA et al. ¹⁰ 2015	EXTEND-IA Campbell BC et al. ¹¹ 2015	ESCAPE Goyal M et al. ¹² 2015	SWIFT PRIME Saver JL et al. ¹³ 2015	REVASCAT Jovin TG et al. ¹⁴ 2015	THRACE Bracard S et al. ¹⁵ 2016	DAWN Nogueira RG et al. ¹⁹ 2017	DEFUSE 3 Albers GW et al. ¹⁸ 2018
Study type, centres	RCT, multicentric	RCT, multicentric	RCT, multicentric	RCT, multicentric	RCT, multicentric	RCT, multicentric	RCT, multicentric	RCT, multicentric
Comparison	EVT or IA-TL +/- IV-TL vs IV-TL	EVT or IA-TL + IV-TL vs IV-TL	EVT +/- IV-TL vs IV-TL	EVT + IV-TL vs IV-TL	EVT +/- IV-TL vs IV-TL	EVT + IV-TL vs IV-TL	EVT vs medical	EVT vs medical
Recruitment period	2010 to 2014	2012 to 2014	2013 to 2014	2012 to 2015	2012 to 2014	2010 to 2015	2014 to 2017	2016 to 2017
Size; N (MT vs control)	500 (233 vs 267)	70 (35 vs 35)	316 (165 vs 150)	196 (98 vs 98)	206 (103 vs 103)	414 (204 vs 208)	206 (107 vs 99)	182 (92 vs 90)
Follow-up, days	90	90	90	90	90	90	90	90
Inclusion criteria	- Age > 18 y - 6 h to MT - Anterior circulation LVO - NIHSS >2	- Age ≥ 18 y - 6 h to groin - < 8 h - Anterior circulation LVO - mRS 0-1 - mismatch on perfusion	- Age > 18 y - < 12 h - ICA/MCA LVO - NIHSS > 5 - Barthel ≥ 90 - ASPECTS > 6 - CT collateral score good or	- Age 18–80 y - 6 h to groin - ICA/M1 LVO - mismatch on perfusion imaging - NIHSS 8–29 - mRS 0–1	- Age 18–80 y - 8 h to groin - LVO ICA/M1 - NIHSS ≥ 6 - mRS 0–1	- Age 18–80 y - IV-TL < 4 h - MT < 5 h - NIHSS 10–25 - ICA, M1, superior 1/3 basilar occlusion	- Age ≥ 18 - No IV-TL - NIHSS ≥ 10 - mRS 0-1 - Time last seen well: 6-24 h - < 1/3 MCA territory	- Age 18-90 - NIHSS ≥ 6 - femoral puncture within 6-16 h - mRS ≤ 2 - ICA or M1 occlusion

		imaging	intermediate				- ICA and/or MCA M1 - Clinical Imaging mismatch	and target mismatch profile on CT perfusion or MRI
1° end point	- mRS shift analysis at 90 d, adjusted OR, 1.67; 95% CI, 1.21–2.3; mRS of 0–2 in 32.6% vs 19.1%	- Median reperfusion at 24 h: 100% vs 37%, adjusted OR, 4.7 (95% CI, 2.5–9) - Decrease in NIHSS of 8 points or NIHSS 0–1 at 3d: 80% vs 37%, adjusted OR, 6 (95% CI, 2–18)	- mRS shift analysis at 90 d; adjusted OR, 3.1 (95% CI, 2–4.7)	- mRS shift analysis at 90 d (mRS 5 and 6 combined), $P < 0.001$	- mRS shift analysis at 90 d (mRS 5 and 6 combined), adjusted OR, 1.7 (95% CI, 1.05–2.8)	- mRS 0–2 at 90 d: 53% vs 42%, $P = 0.028$	- 90-day disability assessed by utility weighted mRS: 5.5 +/- 3.8 vs 3.4 +/- 3.1, Adjusted difference 2.0; 95% CI, 1.1-3.0, PPS > 0.999 - mRS 0-2 at 90 d: 49% vs 13%, Adjusted Difference 33%, 95% CI, 21%-44%, PPS > 0.999	- mRS shift analysis at 90 d: unadjusted OR 2.77; 95% CI, 1.63-4.70; $P = 0.0002$
2° end point(s), if any	- TICl 2b/3: 59% - Median time to reperfusion: 332 min (IQR, 279–394)	- TICl 2b/3: 86% - Median time to reperfusion: 248 min (IQR, 204-277)	- TICl 2b/3: 72% - Median time to reperfusion: 241 min (IQR, 176-359)	- Functional independence at 90-day: 60% vs 35% ($P < 0.001$)	- TICl 2b/3: 66% - Median time to reperfusion: 355 min (IQR, 269–430)	- TICl 2b/3: 69% - Median time to reperfusion: 250 min (IQR 210–290)	90-day mRS 0-2 (1) 6-12 h 55.1% vs 20.0%, PPS > 0.99 (2) 12-24 h 43.1%	- mRS 0-2 at 90-day: 44.6% vs 16.7%, Relative risk 2.67; 95% CI

			- Median time CT to groin puncture: 51 min (IQR, 39–68)	- TICI 2b/3: 88% - Median time to reperfusion: 332 min (IQR, 279–394) - Reperfusion at 24 h: 83% vs 40% ($P < 0.001$)			vs 7.4%, PPS > 0.99	1.60-4.48; $P < 0.0001$
Safety end point	- Death: 21% vs 22% ($P = 0.75$) - sICH: 7.7% vs 6.4% ($P = 0.24$)	- Death: 9% vs 20%, adjusted OR, 0.45 (95% CI, 0.1–2.1) - sICH: 0 vs 6%	- Death: 10.4% vs 19%; adjusted rate ratio, 0.5 (95% CI, 0.3–0.8) - sICH: 3.6% vs 2.7%; adjusted rate ratio, 1.2 (95% CI, 0.3–4.6)	- Death: 9% vs 12%, adjusted rate ratio: 0.74 (95% CI, 0.33–1.68) - sICH: 0 vs 3%	- Death: 18% vs 16%; adjusted risk ratio, 1.2 (95% CI, 0.6–2.2) - sICH: 2% vs 2%; adjusted risk ratio, 1.0 (95% CI, 0.1–7)	- Death: 12% vs 13%, $P = 0.7$ - sICH: 2% vs 2%, $P = 0.71$	- Mortality at 90-day: 19% vs 18%, $P = 1.00$ - sICH: 6% vs 3%, $P = 0.50$	Mortality at 90-day: 14% vs 26%; $P = 0.053$ sICH: 6.5% vs 4.4%; $P = 0.75$
GA, no. (%)	88/233 (37.8)	12/35 (36)	15/165 (9.1)	36/98 (37)	7/103 (6.7)	69/204 (49)	11/107 (10)	26/92 (28%)
Comments, limitations	- First RCT to demonstrate benefit of EVT - Low reperfusion	- Perfusion imaging criteria (mismatch) - Low ability to	- Benefit on mortality of EVT - Large treatment	- Low ability to generalise results (narrow inclusion criteria)	- Large treatment window - Lowest GA rate	- Long duration of trial (5 years) - No additional imaging criteria	- Benefit of MT > 6-24 h with clinical and imaging criteria - Large proportion	- Benefit of MT > 6-16 h with clinical and imaging

rate - Low favourable neurological outcome (mRS 3 [2-5] vs 4 [3-5])	generalise results (narrow inclusion criteria) - Halted early after MR CLEAN results - Small number of patients	window - Collateral and penumbra assessment - Halted early after MR CLEAN results	- Halted early after MR CLEAN results - Small number of patients	- Halted early after MR CLEAN results - Small number of patients	(i.e., perfusion) - 3 mo mRS non-blinded - Halted early after MR CLEAN results - Cost-effectiveness	of patients with wake-up strokes - Stopped early for efficacy at first planned interim analysis - Bayesian approach	criteria - Stopped early at first interim analysis
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Table 1. Positive randomised clinical trials (RCT) demonstrating the benefit of endovascular therapy (EVT) with medical management over medical management alone for anterior circulation acute ischemic stroke. ASPECTS: Alberta Stroke Program Early CT Score; CI: confidence interval; CT: computed tomography; GA: general anaesthesia; IA-TL: intra arterial thrombolysis; ICA: internal carotid artery; IQR: interquartile range; IV-TL: intravenous thrombolysis; LVO: large vessel occlusion; MCA: middle cerebral artery (M1: first segment); MRI: magnetic resonance imaging; mRS: modified Rankin score; NIHSS: National Institutes of Health Stroke Scale; OR: odds ratio; PPS: posterior probability of superiority; sICH: symptomatic intracerebral haemorrhage; TICI: Thrombolysis In Cerebral Infarction.

Monitored anaesthetic care (MAC)*	<ul style="list-style-type: none"> - Relative haemodynamic stability - Shorter time to procedure initiation - Allow clinical evaluation during the procedure and sooner after EVT - Reduced risk of postoperative respiratory complications 	<ul style="list-style-type: none"> - No protection of the airway - Lower comfort for the neuro-interventionalist - Higher risk for technical complications (i.e., vessel dissection) - Emergency conversion to GA in unfavourable condition in the case of MAC failure - Arterial hypotension may occur under conscious sedation 	<ul style="list-style-type: none"> - First-line choice in the absence of a formal indication to GA and convert to GA if needed (Sequential approach**)
General anaesthesia (GA)	<ul style="list-style-type: none"> - Lower risk for patient movement - Improved procedure safety and operator comfort - Secured airway, controlled ventilation (PaCO₂) - No emergency conversion to GA - Shorter procedure time 	<ul style="list-style-type: none"> - Haemodynamic effects of anaesthetic drugs - Delayed groin puncture and treatment initiation - No real time neurologic evaluation - Complications of mechanical ventilation (i.e., pneumonia, delayed extubation) 	<ul style="list-style-type: none"> - Start with GA in the case of: severe agitation (i.e., aphasia, pain), consciousness impairment, seizures, acute respiratory failure or impaired airway protection

Table 2. Pros and cons analysis of anaesthetic approaches for endovascular therapy (EVT) in acute ischemic stroke patients. *Local anaesthesia +/- conscious sedation. ** Step-by-step approach: start the procedure with MAC then convert to GA if needed (i.e., agitated patient, MT related technical issues). PaCO₂: partial pressure of carbon dioxide.

	Schönenberg et al., SIESTA ⁷⁰ 2016	Löwhagen et al., AnStroke ⁶⁷ 2017	Simonsen et al., GOLIATH ^{78, 79} 2018
Primary outcome	Early neurological improvement (24-hour NIHSS)	mRS after 3 mo	Infarct growth after 48 to 72 h
N, number (%) (GA vs CS)	150 (73 [48.6] vs 77 [51.4])	90 (45 [50] vs 45 [50])	128 (65 [58.8] vs 63 [49.2])
Centers	Single-centre Germany	Single-centre Sweden	Single-centre Denmark
Inclusion period	2014 to 2016	2013 to 2016	2015 to 2017
Inclusion criteria	- Age \geq 18 - NIHSS $>$ 10 - Anterior circulation	- Age \geq 18 - NIHSS \geq 10 (14: left-sided occlusion) - Anterior circulation - $<$ 8 h stroke onset	- Age \geq 18 - NIHSS $>$ 10 - Anterior circulation - $<$ 6 h stroke onset - mRS \leq 2
Age, mean (SD or IQR), y (GA vs CS)	71.8 (12.9) vs 71.2 (14.7)	73 (65-80) vs 72 (66-82)	71.0 (10) vs 71.8 (12.8)
Women, number (%) (GA vs CS)	25 (34.2) vs 35 (45.5)	19 (42) vs 22 (49)	29 (44.6) vs 33 (52.4)
Hypertension, number (%) (GA vs CS)	53 (72.6) vs 54 (70.1)	27 (60) vs 22 (49)	39 (60) vs 32 (50.8)
Initial NIHSS, mean (IQR) (GA vs CS)	17 (13-20) vs 17 (14-20)	20 (15.5-23) vs 17 (14-20.5)*	18 (13-21) vs 17 (15-21)
IV alteplase, number (%) (GA vs CS)	46 (63.0) vs 50 (64.9)	33 (73.3) vs 36 (80)	50 (76.9) vs 46 (73.0)

Anaesthesia	IV, low-dose, short-acting analgesics and sedatives in the CS group (same medication at higher dose in the GA group or additional medications)	GA: induced by propofol and remifentanyl, maintained with sevoflurane and remifentanyl; CS: remifentanyl	GA: rapid sequence intubation (suxamethonium, alfentanil and propofol), maintained with propofol and remifentanyl; CS: fentanyl, propofol
Blood pressure objectives (mmHg)	SBP: 140 to 160	SBP: 140 to 180	SBP: > 140 MBP: > 70
Follow-up, days	90	90	90
Primary outcome (GA vs CS)	NS	NS	NS
Secondary clinical outcomes* (GA vs CS)	- Favourable mRS after 3 mo: 27 (37%) vs 14 (18.2%)	NS	- OR for a shift to a lower mRS: 1.91 (95% CI, 1.03-3.56)
Logistics, mean (SD or IQR)* (GA vs CS)	-Door-to-arterial puncture time, min: 75.6 (29.3) vs 65.6 (19.9) -Duration of EVT, min: 111.6 (62.5) vs 129.9 (62.5)	NS	-NRI suite-to-groin puncture, min: 24 (20-27) vs 15 (12-20)
Feasibility of MT, number (%)* (GA vs CS)	- TICI \geq 2 b: 65 (89.0) vs 62 (80.5) - Patient movement: 0 (0) vs 7 (9.1)	- Poor angiography quality: 0 (0) vs 6 (13.3) - Patient movement: 0 (0) vs 15 (33.3)	- TICI \geq 2 b: 50 (76.9) vs 38 (60.3)
Safety, number (%)* (GA vs CS)	-Hyper/hypothermia: 24 (32.9%) vs 7 (9.1%) -Delayed extubation: 36 (49.3%) vs 5 (6.5%) -VAC: 10 (13.7%) vs 3 (3.9%)	NS	NS
Haemodynamic during the procedure, number (%)* (GA vs CS)	NS	- Lower MBP under GA - MBP drops \geq 20%: 41 (93) vs 26 (60) - Vasoactive drugs: 43 (98) vs 34 (79)	- Lower SBP and MBP under GA - MBP drops \geq 20%: 57 (87.7) vs 22 (34.9)
Converted to GA, number (%)	11/73 (14.3)	7/45 (15.6)	4/65 (6.3)

Table 3. Comparison of randomised controlled trials evaluating the impact of anaesthesia on functional prognosis after endovascular therapy (EVT) for acute ischemic stroke (AIS). *Statistically significant ($P < 0.05$). CS: conscious sedation; GA: general anaesthesia; IQR: interquartile range; mRS: modified Rankin score; National Institutes of Health Stroke Scale; NIHSS: MBP: mean blood pressure; NRI: neuro-interventional; NS: non-significant; OR: odds ratio; SBP: systolic blood pressure; SD: standard deviation; TICI: Thrombolysis in Cerebral Infarction; VAC: Ventilation-associated complications.

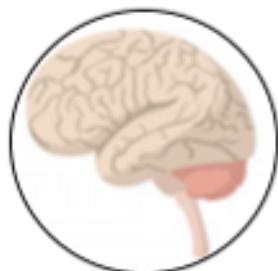


▪ **Risk factors**

- Cardiovascular risk factors (i.e., chronic hypertension, diabetes)³⁸
- Antihypertensive agents, dehydration, hypovolemia

▪ **Clinical complications**

- Angina and myocardial infarction³⁹
- Adrenergic cardiopathy, cardiogenic shock, acute lung edema
- Poorly tolerated arrhythmias⁴⁰

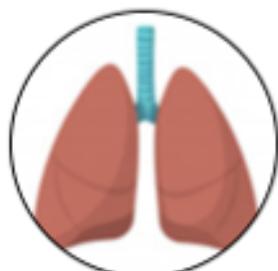


▪ **Risk factors**

- Prior neurological status impaired (mRS >1)
- Severity of stroke related deficits (NIHSS)*⁴¹

▪ **Clinical complications**

- Decreased level of consciousness, seizure
- Pain, confusion, agitation (i.e., secondary to aphasia, urinary globe)
- Spine injury (i.e., stroke related fall)

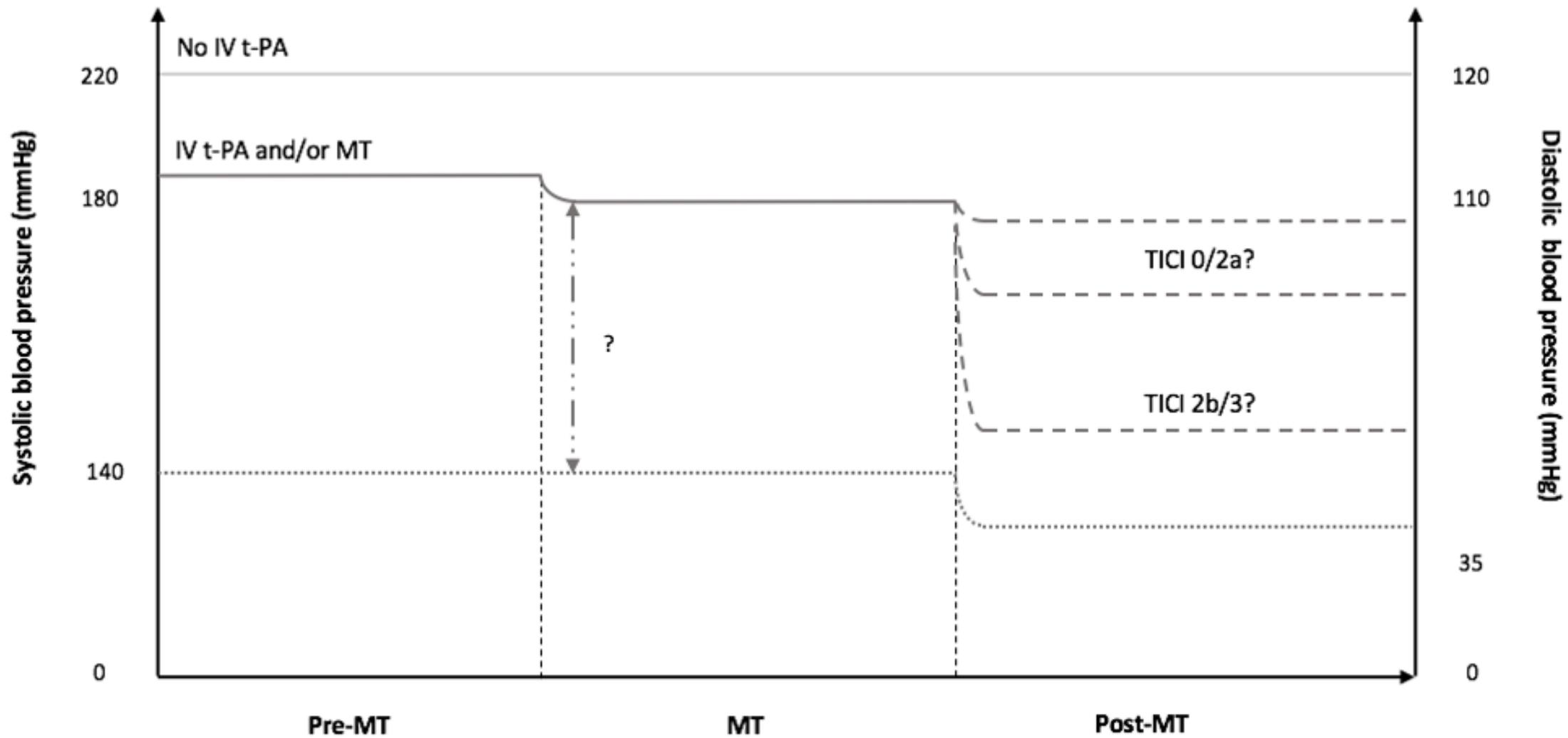


▪ **Risk factors**

- Impaired respiratory function, chronic lung disease (i.e., COPD)

▪ **Clinical complications**

- Inability to protect the airway (i.e., decreased consciousness, bulbar dysfunction)
- Vomiting, inhalation, acute respiratory failure (i.e., bihemispheric or vertebrobasilar ischemia, increased intracranial pressure)



Angio Suite arrival

Patients conditioning

CNAP, ECG, SatO₂, 2 IV, 1.0L cristalloids +/- diluted NE*

1. Consciousness impairment (dominant hemisphere)?
2. Acute respiratory failure (bulbar injury)?
3. Agitation (pain, aphasia, confusion)?
4. Long/technical procedure (ICA, posterior stroke, dissection, >6h)?

No

**LA/CS
First**

1. Perform LA (lidocaine)
2. +/- associated with CS**
3. Capnography
4. +/- IAC or EVT arterial introducer
5. No central line

Yes

GA

1. Perform GA*** +/- RSI
2. Intubation and analgosedation
3. Capnography
4. +/- IAC or EVT arterial introducer
5. No central line

Safety criteria for conversion to GA

1. Decreased consciousness
2. Vomiting
3. Agitation
4. Technical complications

Per procedural objectives

1. Sedation: calm, no movements
2. BP [mmHg]: systolic >140, mean >70
3. etCO₂ [mmHg]: 40-45
4. SatO₂ [%]: 95-98
5. Glucose level [mg.dl⁻¹]: 140-180