International Journal of Science and Research (IJSR)
ISSN: 2319-7064
ResearchGate Impact Factor (2018): 0.28 | SJIF (2018): 7.426

Anesthetic Considerations for Interventional Radiology in Acute Ischemic Cerebral Stroke (AIS)

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1. Introduction

Acute ischemic stroke (AIS) is the leading cause of adult disability and the third leading cause of death. AIS and transient ischemic attacks are caused by cardiac embolism, cervical atherosclerosis with thrombosis or artery-to-artery embolism, intracranial atherosclerosis leading to thrombosis or hypoperfusion, perforator occlusion due to lipohyalinosis, arteriosclerosis, or embolism, spontaneous or traumatic arterial dissection, vasculitis and venous thrombosis, among others.

The typical ischemic stroke patient loses 1.9 million neurones for each minute they are untreated. Compared with the normal rate of neurone loss in brain ageing, the ischemic brain ages 3.6 yr each hour without treatment. (1,2) Sudden loss of focal brain function is the core feature of the onset of AIS and, until recently, it was considered to be untreatable.

Major advances in endovascular therapy in the past 10 to 15 years have now made treatment a reality, with a potential for remarkable recovery. Timely restoration of cerebral blood flow using reperfusion therapy is presently the most effective maneuver for salvaging ischemic brain tissue that is not already infarcted. The duration of ischemia is a leading predictor of neurological outcome, but with modern penumbral imaging, time alone may not be a necessary parameter for who qualifies for IAT. (3,4,5)

Numerous factors affect neurological outcome and the main risk is that of intracerebral hemorrhage (ICH) - the most feared and devastating complication associated with all treatments for ischemic stroke. The risk of ICH is the single greatest limiting factor for AIS recanalization therapy, because there is no effective treatment for ICH and, when it complicates AIS revascularization treatment, mortality is high.

Stroke specialists, therefore, need to have a thorough understanding of all clinical, anatomical, pathological, and radiological aspects of AIS and the spectrum of ICH to make reasonable decisions regarding intra-arterial therapy (IAT) in every patient.

1.1 Time frame for initiating IAT

The traditional time window for IAT is <6 h and for mechanical embolectomy is 8 h from stroke onset or the last time the patient was known to be normal. Patients with AIS presenting in <3 h should be offered IV tPA. Those patients who do not meet the intravenous tPA criteria due to time window or some other factors may be offered IAT. Intravenous tPA has been recently shown to be safe in a subset of stroke patients up to a maximum of 4.5 h from symptom onset with an 8% benefit in increasing the probability of good outcomes.

1.2 Imaging and Treatment Choice in Cerebral Stroke

A non contrast CT of the head remains the standard of care. If a CT-clinical mismatch is found (i.e., the CT changes are relatively minor compared to the deficit), then therapy may be initiated. The best validated means of determining "minor" CT scan changes is to use the 10-point quantitative topographic CT scan Alberta Stroke Program Early CT Score (ASPECTS). A score of >7 is associated with good neurological outcomes in general, as well as following IAT. This is simple, rapid, and safe without need for X-rays and contrast.

Imaging the penumbra using magnetic resonance imaging or computed tomographic perfusion is currently the most preferred way to identify patients who might benefit. The ischemic penumbra is short-lived, lasting only for a few hours in human patients and reflects the threatened but salvageable tissue surrounding the irreversibly injured or infarcted core. Those patients with no penumbra, even with no necrotic core, may have little to no gain by IAT, and would be exposed to unnecessary risk with treatment. The ideal patient is the one with little to no necrotic core and a large volume of penumbra. These patients may have the most to gain with the least risk, in contrast to patients with a large necrotic core, who even with concurrent large penumbra, have high ICH risk. Revascularized necrotic tissue is prone to hemorrhagic transformation.

Over a third of acute ischemic strokes (AISs) originate from large vessel occlusion (LVO) and consequently have significant risk for severe disability or death. AIS caused by emergent large vessel occlusion (ELVO) such as the internal carotid artery, proximal middle cerebral artery, or basilar artery have low rates of response to intravenous tissue plasminogen activator and, subsequently, poor outcomes.

Rapid mechanical thrombectomy (MT) significantly improves outcomes in anterior circulation. The degree of benefit is profound.

All patients should be ideally treated at a center offering a full spectrum of neuroendovascular care (a level 1 center). Level 1, 2, and 3 centers form a complete stroke system of care, ensuring that proper facility and capabilities are available to deliver this treatment in a safe yet timely fashion. The best option for the management of AIS is to have patients transferred to and treated in high volume, level 1 centers

Patients with mild strokes (National Institutes of Health Stroke Score [NIHSS] < 4) are unlikely to have a visible arterial occlusion and are likely to have a good outcome without therapy. Patients with the most severe strokes (NIHSS > 20) are less likely to benefit from treatment. In these patients, penumbral imaging to identify patients with small ischemic cores and large perfusion deficits, may help select who may benefit from treatment. Patients without obvious large vessel occlusion, but with a clinical syndrome that suggests large vessel ischemia, may have multiple distal cortical branch occlusions that may not be amenable to mechanical recanalization techniques. They are better treated with IV thrombolysis or intra-arterial thrombolytics. Such patients may have seizures or other non stroke causes of the deficits where IAT is unnecessary. The presence of complete internal carotid artery (ICA) occlusion from the bulb to the terminus with MCA occlusion, and no pial collateral flow from the anterior cerebral artery, is a predictor of poor response to IV thrombolysis, even in patients treated in <3 h. In patients with a hyperdense middle cerebral artery sign (MCA), a favorable outcome is doubled and the risk of death reduced by 65% in patients treated with IAT over tPA.

The presence of direct (i.e., communicating arteries) or pial collaterals, the status of the other vessels, the presence of severe tortuosity and aneurysms noninvasive imaging can all be detected by non invasive imaging techniques. Knowledge of these features can save valuable time during the intervention by obviating the need to perform angiograms of the other vessels, or by alerting the interventionist for the need to access the vessel in question with more supportive catheters and devices from the outset. A lesion due to atherosclerotic occlusion can be approached differently from the one due to cardioembolism as the former may be directly stented and the latter approached first with an embolectomy device. (6,7,8,9)

1.3 Treatment Strategies

Acute ischemic stroke intervention (AISI) involves percutaneous endovascular procedures to treat ischemic stroke in adults and children, and may involve

- Mechanical thrombectomy is more effective in securing recanalization, with success rates of 81% to 84%.
- Aspiration.
- Percutaneous transluminal angioplasty.
- Stent implantation.
- Superselective drug infusion using recombinant tissue plasminogen activator (rt-PA).

Intra-arterial therapy (IAT) for acute ischemic stroke refers to endovascular catheter-based approaches to achieve recanalization using mechanical clot disruption, locally injected thrombolytic agents or both.

Recanalization rates correlate with clinical improvement, and it is possible to achieve recanalization in roughly 80% of patients treated with the latest generation catheters.

Large vessel occlusions and those with large thrombus burden (i.e., thrombi longer than 8 mm) are not suited for thrombolytic therapy but amenable to endovascular therapy with percutaneous interventions rather than IV thrombolysis.

1.4 Aim

To achieve a modified Rankin Scale (mRS) (Table 1) score of ≦2 (functional outcome) at discharge and one or three months following endovascular therapy.

Table 1: The Modified Rankin Scale

Description	Score
No symptoms at all	+ 0
No significant disability despite symptoms; able to carry out all usual duties and activities	+ 1
Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance	+ 2
Moderate disability; requiring some help, but able to walk without assistance	+ 3
Moderately severe disability; unable to walk and attend to bodily needs without assistance	+ 4
Severe disability; bedridden, incontinent and requiring constant nursing care and attention	+ 5
Dead	+ 6

1.5 Contraindications to AIS intervention

Unruptured, incidental, non thrombosed aneurysms are not a contraindication. The major contraindications to interventions in AIS are:-

1) Intracerebral hemorrhage (lobar, subdural, intraventicular)
2) Subarachnoid hemorrhage
3) History of intracerebral hemorrhage (ICH)
4) Cerebral arteriovenous malformation or giant thrombosed cerebral aneurysm.
5) Computed tomographic evidence of >1/3 middle cerebral artery (MCA) territory acute infarct or large ischemic core on perfusion imaging.
6) Absence of ischemic penumbra.
7) Uncontrolled hypertension , blood pressure >185/110 mmHg
8) Unknown stroke duration or duration >6 h
9) Thrombocytopenia. Platelet counts <100,000/mm3
10) Bleeding diathesis.
11) International normalized ratio >1.7 (if fibrinolysis is planned)
12) History of Alzheimer's disease or amyloid angiopathy.

2. Pre Intervention Work Up

All patients should have the standard clinical, laboratory, and radiological testing before an intervention is contemplated. Patients must also have a clinical deficit severe enough to warrant intervention, both to avoid the risk of the procedure and to ensure that an intervention is likely to be of benefit. Pre anesthetic evaluation should be *focused and concise* to minimize delays in care. Relevant medical history should be available to the anesthesia team from casualty records and time should not be spent duplicating questions. Electronic records including medications, laboratory results (including blood glucose), electrocardiogram, and imaging results can often be reviewed while waiting for the patient to arrive in the interventional suite. Whenever possible, a personal and family history of problems with anesthesia should be elicited, and an airway assessment should be rapidly performed.

Anesthetic Choice

There are no gold standards for the anesthetic management of AIS. Weighing the risks and benefits for the individual patient, the axiom "time is brain" applies to all aspects of AIS care, and the choice of anesthetic technique in AIS-LVO patients should facilitate swift treatment in the safest manner possible.

Even at centers primarily using moderate sedation, patients may deteriorate clinically prior to, or during the procedure. Immediate access to anesthesia is then necessary to safely complete thrombectomy, under monitored anesthetic care or general anesthesia.

Patients with AIS are often elderly with multiple comorbidities. Their neurological status at time of ictus may vary from almost normal to comatose. Patients may be aphasic and unable to communicate, may be paretic, or may experience vertigo—any of which may cause significant distress. This can make it difficult for the patient to tolerate a procedure that requires lying still for a prolonged period of time. If the patient is unable to remain motionless, it can cause significant degradation in image quality, inability to utilize roadmap functions, and even trauma and vessel damage related to movement of the catheter. Patients' movement during the procedure of EVT can lead to wire perforation resulting in intracranial hemorrhage or vascular injury

There is a preference for general anesthesia, especially for patients with AIS and severely altered mental statuses, inability to reliably protect their airway from aspiration, involuntary sporadic movements, and intolerance to any surgical stimulation. Although GA and immobility allow greater image quality and decrease procedural time, the greatest perceived limitation is a delay in starting the procedure. Airway protection by endotracheal intubation prevents, or at least limits, aspiration and ensures oxygenation (hyperoxia may be desirable). Sedation and neuromuscular blockade prevent movement during critical parts of the procedure. This can facilitate road mapping, clot extraction, angioplasty, and stenting. Temporary apnea is easily accomplished and hemodynamic parameters can be titrated as needed. The use of induced hypotension or even adenosine cardiac arrest to limit hemorrhage in the setting of vessel rupture, is safer under GA; glycemic control can also be better handled by the anesthesia team. Various neuroprotective strategies are better with GA, like the use of propofol, barbiturate burst suppression and therapeutic hypothermia. Transcranial neurosonography, to monitor reperfusion or enhance thrombolysis, requires patient immobility and mandates GA.

Anesthesiologists are intimately involved in sedating, anesthetizing and monitoring the patient, managing hemodynamics, oxygenation, ventilation, glycemic control (140-180mg/dl), and peri-procedure complications, all of which may have a significant effect on the patient's long-term outcome. Choice of anesthetic agents should be based on patient condition, pharmacodynamic and pharmacokinetic properties of the drugs, potential adverse effects, and cost.

Hyperoxia has been suggested as a neuroprotective strategy to salvage acutely ischemic brain tissue and to extend the time window for the administration of thrombolytic drugs. Cerebral blood volume and blood flow within ischemic regions have been shown to improve with high-flow oxygen therapy and the regional cerebral vasoconstrictor responsiveness to 100% oxygen inhalation may be lost or paradoxically reversed in patients with acute hemispheric infarction.

Hypocapnia is associated with poor prognosis in stroke and euvolemia must be ensured. Glucose containing fluids are best avoided unless treating serum glucose values are <50 mg/dL.

Nitrous oxide should be avoided in acute stroke interventions because of concerns for exacerbating any cerebrovascular air emboli entrained during the procedure.

Finally, other factors that could further contribute to excessive vasoconstriction or vasodilation, such as hypocapnia or hypercapnia, should be avoided.

Analgesia is an important component of GA and of conscious sedation. An opioid is typically employed for this purpose, and short-acting opioids such as fentanyl or remifentanil are ideal.

Whatever the anesthetic technique selected, it is critical to remember the race to save the penumbra and avoid delay of therapy. (10,11)

3. Limitations of GA

The limitations for GA are:-

1) Time delays.
2) Danger of propagating cerebral ischemia due to hypoperfusion or other complications.
3) Lack of adequate anesthesia workforce.
4) AIS patients treated with GA had a longer length of stay in the intensive care unit and hospital, higher risk of postoperative complications, and larger infarct volumes. Those treated under GA have a greater than 2-fold risk of poor outcomes and a nearly 2-fold risk of death.
5) Neurotoxicity from the general anesthetic agents themselves. Isoflurane induces cerebral vasodilation and steal blood flow from ischemic areas with poor autoregulation.
6) GA can mask spontaneous or therapy-induced neurological recovery or deterioration.

There is no difference in the incidence of intraprocedural complications attributable to microcatheter or microwire perforation or hemorrhagic complications between intubated and nonintubated patients or between patients who received light or deep sedation. The risk of pneumonia and/or sepsis has been reported to be higher in the intubated patients and in those receiving heavy sedation or pharmacologic paralysis.

4. Monitored Anesthesia Care

Conscious sedation offers the benefits of improved hemodynamic stability and reduced time to induction. MAC groups show a higher incidence of mRS score ≦ 2 at three months and decreased in-hospital mortality compared with these patients in GA group. Spontaneous ventilation with noninvasive positive pressure support using BiPAP to avoid severe hypercarbia and pulmonary hypertensive crisis is advisable. Enough sedation must be exhibited for patients to tolerate the surgical procedure and prevent acute changes in hemodynamic responses to noxious stimuli. Before infiltration of the right groin by surgical team, a 10 mg bolus of propofol followed by propofol infusion started at 30 $ug.kg^{-1}.min^{-1}$ and propofol boluses of 10 mg to supplement the infusion is recommended. Phenylephrine /norepinephrine/vasopressin infusion may be employed to maintain mean arterial pressures (MAPs) between 80 and 100 mmHg.

Once the surgical team has mechanically evacuated the patient's MCA clot, the phenylephrine infusion is discontinued and a nicardipine drip titrated to maintain MAPs between 65 and 85 mmHg. Propofol sedation is stopped upon closure and hemostasis of the right groin entry site.

Dexmedetomidine produces sedation, anxiolysis, and analgesia without significantly depressing respirations and can be a good alternative.

Fentanyl and other i.v. opioids are avoided because of their intrinsic and synergistic potential to depress patient's respiratory efforts when used with propofol sedation. Midazolam is also better avoided as it alters the patient's baseline mental status. Inhaled nitric oxide or prostaglandin therapy can offload the right ventricle. Milrinone, epinephrine, and dobutamine can further increase inotropy but will require a central venous catheter, given their potential to cause phlebitis.

5. Disadvantages of MAC/Conscious Sedation

Sedated patients are prone to physical movement. This may lengthen procedural time and precipitate iatrogenic complications. The cerebral vessels are densely innervated and any excessive dilation or mechanical forces applied to them often result in patient discomfort. Sedated airway is unprotected and may be vulnerable to apneic episodes, which may worsen ischemia. Monitoring cerebral oximetry with near-infrared spectroscopy (NIRS) may noninvasively measure regional cerebral perfusion, recanalization, and desaturations from sedation hypoventilation

Table 1: General anesthesia or local anesthesia for endovascular therapy after acute ischemic stroke

General endotracheal anesthesia	Conscious sedation
PROS	**PRO**
Limits mobility of patients	Monitoring for new neurological deficits
Decreased intra-operative procedure time	Clinical endpoint compared to angiographic recanalization as an endpoint
Limits aspiration	
CONS	**CONS**
Required additional workforce	Increased contrast use
Delay time to start intervention	Increase procedure time
No clinical neurological monitoring	Movement-induced vessel perforation
Anesthesia risks	Emergent intubation may be required
Infrastructure and response time	Aspiration risk
Drop in cerebral perfusion pressure	

Control of Hypertension in TIA

More than 60% of patients who present with ischemic stroke have elevated arterial pressures. This is due to a combination of factors like a Cushing response to a mass effect of edema, autoregulation response in a hypertensive patient, and neuroendocrine response to physiological stress. Within the first 24 h after stroke, arterial pressure declines by about 25% in most patients Presentation with high systemic arterial pressure or low systemic arterial pressure at the time of ischemic stroke are both associated with poor outcome. Lower arterial pressure reduces cerebral edema and the probability of hemorrhagic transformation of the infarct, but it can decrease collateral flow in the penumbral area, especially in the setting of ischaemia and impaired autoregulation.

Thus, when GA is used in acute stroke patients, blood pressure should be strictly controlled, particularly at the time of induction. It is recommended that the systolic blood pressure (BP) be maintained between 140 and 180 mm Hg.

If an arterial cannula is not placed, non invasive blood pressure measurements at least every 3 minutes is recommended.

Both the halogenated anesthetic agents and propofol cause dose-dependent systemic hypotension due to vasodilatation at the time of induction. Propofol reduces cerebral metabolic rate and cerebral blood volume, but causes more hypotension post induction than other induction agents such as etomidate, whose use may be preferred in the setting of AIS. This drop in cerebral perfusion pressure often necessitates the concomitant use of vasopressor agents.

All the halogenated inhalational anesthetic agents (isoflurane, sevoflurane, and desflurane) are cerebral vasodilators and do not maintain the normal coupling of cerebral blood flow with cerebral metabolic rate. Thus, although they suppress the cerebral metabolic rate, they cause a relative cerebral hyperemia. This can be a significant concern in patients with *elevated intracranial pressure*.

Controlled ventilation with hypocapnia can offset this vasodilatory effect of the halogenated inhaled anesthetics.

It is advisable to avoid lowering arterial pressure dramatically during the first 12 h after stroke onset, when collateral circulation compromise is still a concern. The American Heart Association (AHA) recommends **not** to treat systolic blood pressure acutely unless it is above 220 mmHg. In those receiving thrombolytics, the MAP should be kept <135 mmHg (<185/110 mmHg). After complete recanalization, blood pressure should be lowered to the normal or below normal ranges to prevent ICH. In cases of incomplete recanalization, mild hypertension may be desired to maintain collateral flow to the nonperfused regions. If ICH is observed, suspected, or believed to be likely, immediate lowering of blood pressure should be performed. (12,13,14,15,16,17)

Fever in AIS

One third of the patients with AIS are febrile and increased body temperature in the setting of AIS is associated with poor neurological outcome due to increased metabolic demands, enhanced release of neurotransmitters, inflammatory response, and increased free radical production. Treatment includes antipyretic medications and cooling devices.

tPA

Only 3% to 8.5% of stroke patients are treated with IV tissue plasminogen activator (tPA). Less than 50% of patients with LVO treated with tPA experience recanalization after thrombolysis. Thus, there is a need for additional reperfusion strategies.

Patients with AIS presenting in <3 h should be offered IV tPA. Those patients who do not meet the intravenous tPA criteria due to time window or some other factors may be offered IAT. Intravenous tPA has been recently shown to be safe in a subset of stroke patients to a maximum of 4.5 h from symptom onset with an 8% benefit in increasing the probability of good outcomes.

All patients who present within 90 min to 3 hours of symptom onset and with no contraindications to therapy are treated with IV tPA.

Patients who present between 3 and 4.5 hours after stroke onset and have no contraindications may be considered for treatment with IV tPA.

Patients who are not eligible for IV tPA (due to delayed time to presentation or contraindications to tPA therapy such as recent surgery or coagulopathy) can be considered for endovascular therapy.

IV tPA is reasonable in patients whose blood pressure can be lowered safely (to below 185/110 mm Hg) with antihypertensive agents, with the physician assessing the stability of the blood pressure before starting IV tPA. Pressures must be maintained below 180/105 mm Hg for at least the first 24 hours after intravenous tPA treatment.

Dose of tPA is 0.9 to 1.1mg/kg: limited to 90-100 mg within 180 mins-360 mins of symptoms. Doubtful utility of tPA:

a) Patients >80 years old
b) Those taking oral anticoagulants, even with INR ≤1.7
c) Those with a baseline NIHSS score >25
d) Those with a history of both stroke and diabetes mellitus.

6. Post Procedure Management

Postoperative neurological checks should be frequently performed. The occurrence of any headache, with or without worsening of neurological status, should be considered a possible sign of ICH warranting immediate clinical evaluation and emergent CT scan of the brain. The reversal of all antithrombotic and thrombolytic agents should be carried out immediately. If an ICH is detected, urgent neurosurgical consultation must be sought.

Prognosis

Prognosis is affected by various factors such as age of the patients, duration of ischemia, presence of early infarct signs, presenting blood pressure, serum glucose level, underlying dementia, and the presence of collaterals. Large vessel occlusions are the most common cause of severe and fatal AIS. ICA terminus occlusion portends a poor prognosis regardless of recanalization method. By comparison, patients with cervical ICA occlusion, with or without tandem MCA occlusion have a better prognosis. Risk of ICH is greater in patients >80 years of age, hypertension > 185/110 mmHg, elevated serum glucose, prolonged duration of ischemia, absence of collateral blood flow, large infarct size, large clot burden, concurrent use of anticoagulants or other agents, coagulopathy, or aggressive mechanical manipulation during the interventions. A 10% rate of symptomatic ICH after interventional therapy of AIS is considered acceptable

7. Conclusions

In a patient with acute ischemic stroke, time is of essence to save the vulnerable penumbra. The decision on chosing general anesthesia, monitored anesthesia care, or local anesthesia for endovascular procedures should be made with the individual risks and benefits of each patient considered. Arterial pressure should be monitored carefully upon induction of anaesthesia and induction should be expedient to allow for timely endovascular intervention if possible. Target arterial pressures should be systolic readings of 140–180 mm Hg with a reduction in pressure upon recanalization. There are no proven neuroprotective strategies to date for ischemic stroke. (18,19)

References

[1] LozanoR, Naghavi M, ForemanK, et al. Global and regional mortality: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380: 2095–1282
[2] Go AS, Mozaffarian D, Roger VL,et al. Heart disease and stroke statistics—2014 update: a report from the American Heart Association. Circulation 2014;129: e28–2923

[3] Jauch EC, Saver JL, Adams HPJr, et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2013;44: 870–9474
[4] Saver JL, Fonarow GC, Smith EE, et al. Time to treatment with intravenous tissue plasminogen activator and outcome from acute ischemic stroke. J Am Med Assoc2013;309: 2480–85
[5] Del Zoppo GJ, Saver JL, Jauch EC, Adams HP Jr, American Heart Association Stroke Council. Expansion of the time window for treatment of acute ischemic stroke with intravenous tissue plasminogen
[6] Powers WJ, Rabinstein AA, Ackerson T, et al. 2018 Guidelines for the Early Management of Patients With Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke 2018; 49:e46.
[7] Adams HP Jr, Davis PH, Leira EC, et al. Baseline NIH Stroke Scale score strongly predicts outcome after stroke: A report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). Neurology 1999; 53:126.
[8] Adams HP Jr, del Zoppo G, Alberts MJ, et al. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. Stroke 2007; 38:1655.
[9] European Stroke Initiative Executive Committee, EUSI Writing Committee, Olsen TS, et al. European Stroke Initiative Recommendations for Stroke Management-update 2003. Cerebrovasc Dis 2003; 16:311.
[10] David L. McDonagh, DaiWai M. Olson, Junaid S. Kalia, Rishi Gupta, Alex Abou-Chebl, and Osama O. Zaidat. Anesthesia and Sedation Practices Among Neurointerventionalists during Acute Ischemic Stroke Endovascular Therapy. Front Neurol. 2010; 1: 118-124.
[11] Anastasian ZH. Anaesthetic management of the patient with acute ischaemic stroke. British Journal of Anaesthesia 2014;113(S2): ii9–ii16.
[12] Qureshi AI. Acute hypertensive response in patients with stroke: pathophysiology and management. Circulation 2008; 118:176.
[13] Aiyagari V, Gorelick PB. Management of blood pressure for acute and recurrent stroke. Stroke 2009; 40:2251.
[14] Leonardi-Bee J, Bath PM, Phillips SJ, et al. Blood pressure and clinical outcomes in the International Stroke Trial. Stroke 2002; 33:1315.
[15] Bath PM. How to manage blood pressure in acute stroke. J Hypertens 2005; 23:1135.
[16] Berge E. Should high blood pressure be lowered in the acute stroke? J Hypertens 2011; 29:1478.
[17] Oliveira-Filho J, Silva SC, Trabuco CC, et al. Detrimental effect of blood pressure reduction in the first 24 hours of acute stroke onset. Neurology 2003; 61:1047.
[18] Chamorro Á, Dirnagl U, Urra X, Planas AM. Neuroprotection in acute stroke: targeting excitotoxicity, oxidative and nitrosative stress, and inflammation. Lancet Neurol 2016; 15:869.
[19] Neuhaus AA, Couch Y, Hadley G, Buchan AM. Neuroprotection in stroke: the importance of collaboration and reproducibility. Brain 2017; 140:2079.