Anaesthesia for Interventional Neuroradiology

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Anaesthesia for Interventional Neuroradiology

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13.1 Interventional Neuroradiology and the Neuroteam

Interventional neuroradiology (INR) or endovascular neurosurgery is a specialty emerged as a hybrid of traditional neurosurgery and neuroradiology. It has its role in the management of neurovascular diseases and other neurosurgical conditions, by delivering therapeutic drugs and devices through endovascular or percutaneous access [1].

INR always needs some kind of anaesthesia or sedation and is considered as part of the non-operating room anaesthesia (NORA). NORA generally defines every anaesthetic regimen performed out of the classical operating theatre to serve non-surgical procedures, which are rapidly developing and have both diagnostic and therapeutic purposes [2].

Although, interventional procedures cause much less tissue trespass than surgical operations, anaesthetists must deal with some specific challenges of this domain. Locations may not be adequately organized or equipped to host interventional procedures and manage potential emergencies [2]; interventional suite personnel are

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often not used to cooperate with anaesthetists and therefore may not be aware of their needs; anaesthetists may not be fully trained to deal with NORA challenges, and they may not be familiar to many new interventional techniques [3].

These criticisms make patient management more difficult, while patients with acute brain injuries are particularly frail and need a multidisciplinary, well-integrated management and shared therapeutic plans. According to the well-developed neurosurgical literature, the patient outcome may be improved by the presence of a neuroteam able to focus on common priorities and to share specific competencies. The neuroteam is made by neuroradiologist, neurosurgeon, neuroanaesthetist, neurointensivist, neurologist and well-trained nursing personnel. The neuroanaesthetist differs from general anaesthetist for his qualified experience in treating patients with acute neurologic injury and knowledge of goals and methods of endovascular intervention. In such organization, the neuroanaesthesia department takes care of patients before, during and after the procedure, delivering both anaesthesiologic and intensive care services. Therefore, anaesthesia is essential for INR activity and whole patient management.

This chapter focuses on the anaesthesiologic peri-procedural management of patient undergoing INR procedures. After a rapid introduction to the field of application of INR, anaesthetic issues are described sorting them in two different sections:

- General considerations applicable to all INR procedures
- Specific considerations inherent to the three most important and frequent INR procedures: aneurysm coiling, arteriovenous malformations (AVMs) and fistulae (AVFs) embolization and acute ischaemic stroke thrombectomy

13.2 Introduction to Interventional Neuroradiology

Neuroradiology procedures can be classified in two major groups, as shown in Table 13.1 [1].

Table 13.1 Main neuroradiology procedures

Diagnostic procedures
• Cerebral and spinal cord angiography • Carotid occlusion test • Super-selective anaesthesia functional examination
Therapeutic procedures
Closing or occluding procedures • Endovascular treatment of aneurysms • Embolization of AVMs and AVFs • Preoperative tumour embolization Opening procedures • Chemical and mechanical thrombolysis in acute ischaemic stroke (AIS) • Angioplasty and stenting of intra- and extracranial atherosclerotic vessel disease • Angioplasty and stenting of vasospasm

13.3 Generic Considerations About Anaesthesia for INR

13.3.1 Management of Patients During Transport

Patients with neurovascular diseases or injuries may have highly unstable conditions and rapidly deteriorating neurological status. Therefore, before transfer, some precautions should be taken:

1. Carefully organize logistic details, staff and equipment for the route.
2. Adequately alert the receiving ward or operating room.
3. Consider each patient's clinical condition, especially neurologic injury and haemodynamic and respiratory status.

Two personnel, with at least one certified in advanced cardiovascular life support, should be present during transport [4].

13.3.2 Radiation Exposure Risk and Protection

The major source of radiation is the X-ray tube, but leakage through the collimators and radiation scattered from surfaces surrounding the patient's head are two other minor sources [5]. The amount of exposure responds to the inverse square law: radiation intensity decreases proportionally with the inverse of the square of the distance from the source of radiation.

Therefore, correct radiation protection depends on the following:

- Stay as far as possible [4]; use connected multiparameter monitor from remote locations
- Wearing protections, particularly lead aprons (at least 0.5 mm thickness), thyroid shields, protective eyewear and radiation exposure badges [5, 6]
- Providing movable lead glass screens [4]

13.3.3 Equipment and Logistic Organization

The American Society of Anesthesiology (ASA) produced a statement on the minimal necessary equipment and organization for NORA. In each location there should be:

- A reliable source of oxygen sufficient for as long as the entire procedure
- An adequate source of aspiration system
- A reliable removal system for anaesthetic gases (if used)
- A bag valve mask, adequate anaesthesia drugs, supplies and equipment
- Adequate monitoring systems and anaesthesia machine

- An emergency cart with defibrillator and emergency drugs
- Sufficient space for equipment and personnel and to allow rapid access to the patient [7]

In the INR suite, imaging devices need to rotate around the patient's head without any restriction [4]. For this reason logistic organization of the INR suite differs from that of surgical settings: anaesthetist and anaesthetic equipment cannot be close to the patient's head, but they are placed at the opposite end of the table, thanks to extensions on tubes and breathing circuit [4, 8].

13.3.4 Patient Monitoring

As stated by the ASA, each procedure requires online monitoring of the following physiologic variables: oxygenation, ventilation, circulation and body temperature [9].

To ensure adequate oxygen delivery, oxygenation must be controlled by measuring the concentration of O_2 in the inspired gas mixture and by pulse oximetry [9]. Capnometry is important in every anaesthetic regimen, as a method of control of ventilation and circulation, but it is especially important in INR because variations in arterial pressure of CO_2 can be induced to modify cerebral blood flow (CBF) and intracranial pressure (ICP). The sampling port of a nasal cannula provides CO_2 measuring when patients are not intubated.

Standard monitoring with ECG, non-invasive blood pressure (NIBP) and pulse oximetry are essential for every INR procedure. Invasive blood pressure monitoring may be required by the type of procedure or by the patient condition (e.g. aneurysm coiling with risk of elevated ICP), but it is not mandatory [9]. For example, diagnostic angiography does not require invasive BP monitoring, while the Society for Neuroscience in Anesthesiology and Critical Care (SNACC) recommends invasive BP measurement unless it delays the procedure [10].

Invasive BP monitoring may facilitate the prevention or management of cerebrovascular complications of INR procedures, and it is advocated when an anaesthetist must deliberately modify BP during the procedure.

Temperature monitoring and warming devices are required: there is a lack of evidence on body temperature management in INR; normothermia is recommended in agreement with the well-developed observation that peri-procedural hypothermia and post-procedural shivering have deleterious effect both in surgical patients and patients with acute brain injury [11].

13.3.4.1 Intraoperative Neurologic Status Monitoring

Neurological monitoring deserves special considerations. Interventional procedures may be complicated by haemorrhagic or occlusive events that worsen patient's outcome. It is important to detect cerebrovascular complications early enough to allow corrective intervention. During conscious sedation it is possible to examine the neurological status while general anaesthesia or comatose status does not allow a direct assessment of neurological function [1].

Electrical activity is the first function lost after regional interruption of cerebral blood flow. Therefore, it has been proposed the use of electrophysiological monitoring (evoked potentials) or non-invasive cerebral oxygenation by near-infrared spectroscopy (NIRS). Indeed there is a time window between electric failure and ion pump failure with irreversible damage [12]. Some large retrospective studies highlighted that evoked potentials are able to detect neurological damage and improve the patient outcome [13–16]. ICONA (Italian Consensus in Neuroradiological Anesthesia) recommendations reach a B grade of consensus strength for evoked potentials, and their use should be integrated in the perioperative management of patient indicated to INR [17].

Due to the limited evidence available and the weak results obtained, NIRS reaches a C grade of consensus strength in ICONA recommendations, so its use in clinical practice is still controversial [17].

13.3.5 Complete Preoperative Evaluation

A complete preoperative evaluation includes:

1. History of actual illness and its systemic effects [8]
2. Neurological deficits and Glasgow Coma Scale (GCS).
3. Symptoms of raised ICP: if present, the neuroanaesthetist must maintain an adequate MAP to ensure sufficient CPP.
4. Previous neurosurgical procedures.
5. Renal function, history of radiographic contrast reactions (contrast nephropathy and allergy) and other risk factors. Contrast and flush fluid injections may be dangerous for renal function [6, 8].
6. Allergy and medication history [6].

A phenomenon yet well-described is the re-emergence of prior fixed neurologic deficits in patients who undergo anaesthesia [18, 19]. There is no explanation for this observation, but it can complicate neurological status evaluation during the procedure [5]. This requires to carefully perform neurological examination before the procedure, with assessment of GCS, pupil size and reactivity and any focal deficits [8].

All patients should have preoperative blood tests:

- Full blood count, haemoglobin and haemo group for the risk of bleeding [6].
- Urea and creatinine for renal function.
- Electrolyte abnormalities may contribute to patient's level of consciousness [6].
- Coagulation screen with basal ACT is fundamental because anticoagulation is required during and after INR procedures to avoid thromboembolic complications [5].
- Glycaemia should be assessed being a source of secondary brain injury [20].

13.3.6 Choice of Anaesthetic Technique

There is no evidence for a superior anaesthetic choice for all different INR procedures. Therefore recommendations of neuroanaesthesia for surgical procedures are often applied to INR [5], and anaesthetic regimen is chosen in relation to the disease, patient condition and procedure.

Among anaesthetic techniques for INR, general anaesthesia (GA) and conscious sedation (CS) are the main choices. Table 13.2 resumes their principal pros and cons.

13.3.6.1 Reduced Time Delay

Some INR procedures need immediate treatment; reducing time delay is particularly important for endovascular treatment of AIS. The phrase "time is brain" was first used by Saver to emphasize that cerebral tissue is rapidly and irreversibly damaged over time after stroke [21].

In this setting, GA may cause some delay, due to the time needed for induction of anaesthesia and endotracheal intubation. However several studies demonstrated that there is no difference in time delay from patient arrival at the hospital and revascularization among the two anaesthetic techniques [22–29]. This is explained by the fact that procedure duration is longer in CS regimen rather than in GA regimen because of patient movements [23], compensating time delay for endotracheal intubation of patients undergoing GA [27, 30].

13.3.6.2 Patient Immobility, Comfort and Analgesia and High-Quality Imaging

GA provides patient immobility and analgesia allowing patient comfort and safe device deployment into the vasculature with reduced risk of iatrogenic vascular

Table 13.2 Resumes principal pros and cons of general anaesthesia and conscious sedation. Modified from Anastasian 2014

General anaesthesia	Conscious sedation
Pros	
Patient immobility, comfort and analgesia	Reduced time delay
High-quality imaging	Haemodynamic stability
Airway protection	Intraoperative neurological monitoring
Control of haemodynamic and respiratory variables	Rapid post-procedure recovery and short monitoring period
Cons	
No intraoperative neurological monitoring	Risk of emergency conversion
Time delay	Patient's movements, discomfort and pain
Haemodynamic instability	Poor-quality imaging
Long post-procedure recovery and initiation of a critical care pathway	Prolonged procedure time
	Risk of aspiration
Respiratory complications due to intubation	Risk of vessel injury from endovascular device

injury. Besides, patient immobility increases imaging quality and decreases the need for repeating imaging acquisition, for contrast fluid administration and for time to achieve the procedure [4, 5].

Patients in conscious sedation have always a certain degree of discomfort from lying in the supine position for a long time, from burning pain due to contrast injection and from headache due to distention or traction on cerebral vessels. Discomfort increases patient's movements that in turn oblige to repeat imaging acquisition and so lengthens the procedure [5].

GA is obviously mandatory in patients confused who have involuntary movements or are uncooperative [8]

13.3.6.3 Haemodynamic Stability and Maintenance of Adequate CPP

GA allows easier deliberate modifications of BP during the procedure, but it may also produce haemodynamic instability: hypertension during induction (stimulation of oropharynx and larynx) and emergence (cough and strain) and hypotension during maintenance due to higher doses of anaesthetics and analgesics than CS [1].

CS instead may allow avoidance of all these haemodynamic consequences typical of GA, ensuring more stable pressure values during all the different procedure phases [4].

13.3.6.4 Airways and Respiration Management

GA ensures careful airway protection, and it reduces the need for acting on airways during the procedure. CS, instead, exposes to the risks of gastric aspiration in non-fasted patients, and, if any problem occurs, emergent intubation may be more risky [4, 6].

GA allows tightly monitoring of ventilatory variables reducing the risk of hypoxemia and hypercapnia [4] and allowing deliberate adjustments [5, 31].

13.3.6.5 Anaesthetic Drugs

The anaesthetic regimens for GA in neuroradiology are total intravenous anaesthesia (TIVA) with propofol and opioids or balanced anaesthesia with the use of volatile anaesthetic such as sevoflurane together with opioids. Remifentanil is the opioid of choice for its pharmacodynamic and pharmacokinetic features and its half-life context independence [32].

Propofol causes systemic hypotension and reduces cerebral blood flow (CBF), intracranial pressure (ICP) and metabolic demand. Hence, TIVA is preferred when patients have elevated ICP or have elevated risk of intracranial hypertension. Sevoflurane, instead, ensures systemic hemodynamic stability and is associated with a more rapid and smoother emergence from anaesthesia than propofol [33], but volatile anaesthetics induce cerebral vasodilatation, increase CBF and do not allow neurophysiological monitoring during procedure [34, 35]. In these cases, TIVA may be the regimen of choice for maintenance of anaesthesia.

Both TIVA and inhaled anaesthesia are useful for rapid titration of arterial pressure if deliberate brief time of hypotension is needed.

The use of sevoflurane strengthens neuromuscular block and spare in neuromuscular blocker dose. This is associated with a lower incidence of post-operative residual curarization and complications.

13.3.7 Management of Blood Pressure

Haemodynamic stability is crucial in all procedures of INR. It is important to assess the baseline BP and ascertain the likely autoregulatory range [4]. Some phases of these procedures may require manipulation of the arterial pressure, with deliberate hypertension or hypotension [4].

Deliberate hypertension may be necessary during intraprocedural arterial occlusion, due to intentional balloon inflation or thrombotic clot formation. Increasing mean arterial pressure (MAP) up to 30–40% above the baseline is necessary to maintain CBF through collateral circles or through a partially occluded artery. Deliberate hypertension may be achieved with multiple vasoactive agents such as phenylephrine or ephedrine under ECG monitoring and considering the risk of haemorrhage [4, 5].

In other situations, it may be necessary to induce deliberate hypotension to define cerebrovascular reserve before carotid occlusion procedure, to decrease blood flow into AVM before intraarterial liquid embolization or to temporarily decrease BP in the immediate management of cerebral haemorrhage [5].

13.3.8 Management of Anticoagulation, Platelet Inhibition and Reversal

Many INR procedures require the use of anticoagulant and/or antiplatelet drugs. Anticoagulation is necessary to prevent thrombotic complications during INR procedures. Unfractionated heparin (UFH) is most commonly used. The initial dosage is 50–70 UI/kg to achieve an activated clotting time (ACT) of two- to threefold preheparin, and then UFH can be given continuously or as intermittent boluses [5]. When procedures end or in case of intraprocedural haemorrhage, it is necessary to reverse heparin effects. The first-line therapy for the reversal of anticoagulation with UFH is protamine: 1 mg for each 100 UI heparin given. It is important to remember that protamine administration is not uneventfully. This drug must be administered slowly, because it increases pulmonary vascular resistance and decreases systemic vascular resistance, with the risk of pulmonary hypertension and systemic hypotension, respectively [1, 5]. A fast infusion of protamine may cause cardiogenic shock. Besides, administering more protamine than the necessary has a pro-haemorrhagic effect.

Antiplatelet drugs, including aspirin, P2Y12 inhibitors and glycoprotein IIb/IIIa antagonists, are often needed in IR procedures, particularly those involving stent placement. Clopidogrel is a P2Y12 antagonist commonly used for stent placement or stent-assisted aneurysm coiling. Because of pharmacogenomic differences in

response to clopidogrel, prasugrel may function as an alternative P2Y12 antagonist in these patients [4].

13.3.9 Rapid, Smooth Recovery

After INR, the aim is to rapidly wake up the patient to perform early neurological evaluation. Besides, it is necessary to achieve a smooth recovery to avoid ICP surges or haemorrhage due to cough and strain.

Maintenance of anaesthesia with sevoflurane is associated with a more rapid recovery from anaesthesia in INR [33]. Waking up patients with minimal dose of remifentanil infusion or extubating patient with a deep plain of anaesthesia and the presence of spontaneous breathing are possible emergence strategies. However, the choice of technique is less important than maintenance of patient stability [6].

ICONA recommendations highlight that, after an INR procedure without any complication or major comorbidities, there is no need to routinely admit all patients to an intensive care unit, but continuous observation for 1–4 h must be ensured [17]. Patients who have had neurological complications, instead, need to be transferred to neuro-intensive care settings for continued sedation and ventilation.

13.3.10 Management of Procedure-Specific Complications

Endovascular procedures may have some severe peri-procedural complications (Table 13.3).

Cerebrovascular complications may be catastrophic, and efficacious management with good outcomes depends upon well-planned strategies and a rapid and clear communication between anaesthetist and operators, to define the haemorrhagic or occlusive nature of the problem [4, 5].

In all cases, the anaesthesiologist must preserve pulmonary gas exchange by ensuring a secure airway and by ventilating 100% oxygen [4, 5].

Table 13.3 Summarizes peri-procedural complications of endovascular procedures. Modified from Perritt and Mahalingam 2014

Cerebrovascular complications	Peripheric or systemic complications
Haemorrhagic • Aneurysms, AVM and AVF bleeding or re-bleeding • Vessel injury/rupture	Contrast induced • Anaphylaxis • Contrast nephropathy
Occlusive • Thromboembolism • Arterial dissection • Vasospasm • Intravascular device or material migration/ displacement	Haemorrhage • Puncture site • Groin haematoma • Retroperitoneal haematoma

13.3.10.1 Vascular Occlusion

In the setting of vascular obstruction or intraoperative vasospasm, the goal is to increase distal perfusion by MAP augmentation to 30–40% above baseline to increase oxygen delivering by collateral blood flow. Simultaneously, intraarterial direct thrombolysis may be performed considering patient comorbidity: it should be titrated to a neurological or angiographic endpoint [4, 5, 8]. The operator may also try to achieve the recanalization through emergent angioplasty, stenting, mechanical lysis or other endovascular treatments (e.g. intraarterial vasodilator injection for vasospasm) [4].

Prevention of vascular occlusion implies the use of anticoagulant drugs during the procedure (as UFH in flush fluid) to reduce incidence of intraoperative thromboembolism and preoperative calcium channel blockers for catheter-induced vasospasm [5].

13.3.10.2 Intracerebral Haemorrhage

Intracerebral haemorrhages may be either spontaneous from aneurysm or AVM/AVF rupture or caused by endovascular devices or by inadequate anaesthetic management (BP surges) [6, 36].

Headache, nausea, vomiting and vascular pain are typical signs of intracranial haemorrhages in conscious patients. When GA is performed or in comatose patients, the Cushing response (sudden bradycardia and hypertension) or the evidence of contrast extravasation on fluoroscopy may be the only signs of a haemorrhage [5].

In haemorrhagic emergency, anaesthetist must rapidly reverse anticoagulation and consider to induce transient hypotension [1]. Heparin must be antagonized with protamine on the basis of original UFH doses and serial ACT measurements [5, 8]. Antiplatelet drugs do not have specific antidotes: intravenous desmopressin may be used to decrease the effect of antiplatelet drugs, but platelet transfusion is the standard therapy for reversal of effects [37, 38]. The use of specific clotting factors may be considered in cases of life-threatening bleeding uncontrolled with platelet transfusion therapy [39].

Secondary, BP may be controlled by deepening anaesthesia or using antihypertensive drugs, such as IV labetalol [8].

The INR team should find the bleeding site and try to stop the haemorrhage by endovascular treatment, to complete the procedure. Rarely, a patient needs to be transported in the operating room for a rescue craniotomy and vascular clipping [5, 8, 36].

Haemorrhages may produce sudden ICP elevations with the risk of herniation syndromes. The initial management includes hyperventilation (that rapidly reduces cerebral blood volume), head elevation to 15–30°, steroids, intravenous mannitol boluses and burst suppression [4, 36]. When hydrocephalus has developed and neurological condition is deteriorating, a ventricular or lumbar catheter may be placed in the INR suite to monitor and drain CSF, as recommended by the ICONA [8, 17].

13.4 Anaesthetic Implications for Each Specific INR Procedure

13.4.1 Aneurysms

Endovascular coiling is now considered the treatment of choice for many ruptured or unruptured aneurysm of anterior and posterior cerebral circulation compared with neurosurgical clipping, because coiling is associated to a 5-year reduced mortality compared to surgical clipping [40].

GA is preferred over CS during endovascular coiling, because of perceived improved imaging quality and patient safety. However, CS may be performed safely in patient with ruptured cerebral aneurysms and low-grade SAH (WFNS grades 1–2; see Table 13.4), in order to reduce GA complications and allow frequent neurological evaluation [5].

The main goal for the anaesthesiologist is to maintain haemodynamic stability and adequate cerebral perfusion and oxygenation. It is important to maintain systolic arterial pressure (SAP) <160 mmHg during all phases of anaesthesia and to avoid BP surges that might cause aneurysmal rupture. For this objective, Propofol is usually used for the induction of anaesthesia in combination with remifentanil, alfentanil or fentanyl. For the maintenance of anaesthesia, sevoflurane may be the volatile anaesthetic of choice: up to 1 minimal alveolar concentration (MAC), the cerebral circulation responsiveness to CO_2 is preserved, and CBF/cerebral metabolic rate for O2 (CMRO2) coupling is maintained [4]. Propofol is associated with reduced CBF, ICP and CMRO2. Among short acting opioids, remifentanil provides stable haemodynamic and allows more rapid recovery from anaesthesia [8].

In SAH patients some important complications may occur, cardiopulmonary damage and vasospasm being the most threatening.

Neurogenic stress cardiomyopathy (NSC) may complicate several types of severe acute brain injury, and it is mainly a consequence of the catecholamine storm released in the acute phase. NSC presents with ECG or left ventricular wall motion abnormalities, myocardial necrosis enzyme and brain natriuretic peptide elevation [5, 41]. Respiratory complications occur in up to 20–80% of patients with SAH and include pulmonary oedema (cardiogenic or neurogenic or mixed), acute lung injury, acute respiratory distress syndrome and pneumonia. Both NSC and respiratory complications increase mortality and morbidity [42, 43].

Table 13.4 WFNS grading score

Grade	GCS	Motor deficit
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present or absent
5	3–6	Present or absent

Vasospasm is a potential complication of SAH. It may occur 7–10 days after aneurysm rupture and resolves spontaneously after 21 days. Vasospasm is one possible cause of delayed cerebral ischemia (DCI). The arterial narrowing is probably caused by the contact of oxyhaemoglobin with the abluminal side of vessels. To prevent vasospasm, euvolemia should be maintained, and oral nimodipine (60 mg every 4 h) should be administered to all patients with SAH for a period of 21 days [44–46]. When vasospasm develops, maintenance of euvolemia and induction of hypertension is indicated to treat ischemia, unless BP is high at baseline or cardiac conditions preclude it [46]. Patients with symptomatic vasospasm and not rapid response to deliberate hypertension may be treated with cerebral angioplasty and/or selective intraarterial vasodilators [46].

13.4.2 AVMs and AVFs

AVMs are classified as cerebral or dural. Cerebral AVMs are congenital and consist of a nidus of abnormal vessels containing arterial inflow and venous outflow, often in absence of capillary component. Dural AVMs are acquired, often due to venous dural sinus stenosis or occlusion, with opening or recanalization of a potential fistulous tract due to venous hypertension. Endovascular embolization alone is rarely sufficient, and subsequent surgery and radiotherapy are generally requested to complete the treatment.

GA is the preferred technique for the treatment of AVM and AVF, due to enhanced vessel visualization, lack of patient movement, and possible need for deliberate hypotension or cardiac arrest to counteract venous hypertension in dural fistulae [4]. In some phases it may be necessary to induce deliberate hypertension or hypotension. Hypertension is necessary in case of vascular occlusion, to achieve the maintenance of cerebral perfusion through collateral vessels. Deliberate hypotension may be helpful, because it reduces flow into AVMs and allows glue adherence to the tissues, avoiding unwilling migration [4]. Anaesthesia plane can be tailored to manipulate arterial pressure and induce hypotension. Both propofol bolus and temporarily increase of sevoflurane's inspiratory concentration are useful in reducing BP for a brief time. Short-acting vasoactive agents may be used in addition to achieve hypotension. Transient asystole produced by adenosine administration or rapid ventricular pacing is an option in selected patients [47].

After AVM exclusion, cerebral hyper-perfusion and consequent oedema and haemorrhage may result from abrupt restoration of cerebral blood flow to chronically hypo-perfused vascular beds that have lost their autoregulatory capacity [48, 49]. So, maintenance of systolic BP 20–30% below the basal values during the recovery phase may be protective in these cases.

Because of the embolization, there is always a risk of morbidity and mortality. The most common complication associated with occipital AVM endovascular treatment is the visual field loss [50].

13.4.3 Stroke

Intravenous thrombolysis with rtPA within 4.5 h of symptom onset is now widely accepted as the mainstay of early treatment for AIS [51, 52]. Intraarterial methods of recanalization have been introduced to increase the number of patients treated and the efficacy of the early treatment [53]. Indications to endovascular treatment are increasing, and this claims for some considerations on the best anaesthesiologic management.

Medical literature reported association between GA and higher mortality and poorer neurological outcome compared to CS, but this observation is sustained by poor-quality evidences [22, 25, 27, 54, 55]. More recently, other retrospective studies and the first two RCTs published do not demonstrate any superiority of CS compared to GA [23, 24, 56–59].

There is yet no agreement among authors about the influence of anaesthetic regimen on the efficacy in achieving recanalization. Although Brinjikji and colleagues state that GA is associated with reduced probability of revascularisation compared with CS [22], some other authors did not observe this difference [24, 56], and others have shown a greater success with GA [60].

In several studies, GA was associated with a higher rate of respiratory complications due to the invasiveness of endotracheal ventilation [22–24, 54], but not all authors agree on this statement [61]. On the other side, one of the major disadvantages of CS is patient movements and risk for iatrogenic vascular complications caused by intraarterial devices. However, most of the studies available do not highlight a higher incidence of these complications in CS group with regard to GA group [22, 24–26, 54, 61].

So far, there is no consensus on the best anaesthesiologic regimen for endovascular treatment of AIS. AHA/ASA guidelines suggest preferring CS over GA [62], but they have been produced before the publication of SIESTA, AnStroke and GOLIATH trials [23, 24, 60]. Therefore the choice of anaesthetic regimen must be individualized on patient clinical characteristics, in communication with the neurointerventionalists [10].

In 2014 the SNACC developed a series of recommendations for the anaesthesiologic management of patients who undergo endovascular treatment for AIS [10].

13.4.3.1 Haemodynamic Management

The SNACC recommends to maintain SAP between 140 and 180 mmHg and diastolic blood pressure (DAP) below 105 mmHg [10]. These targets derive from the observation that 150 mmHg is the pressure value associated with the best outcome for a patient with acute ischaemic stroke [63].

Three clinical trials demonstrated that it is beneficial to reduce arterial BP after 24 h from stroke onset, when the effect of high BP on the ischaemic penumbrae lessens [64–66].

13.4.3.2 Respiratory Gas Exchange

Recommendations by the SNACC suggest to maintain $SpO_2 > 92\%$, $PaO_2 > 60$ mmHg and $PaCO_2$ between 35 and 45 mmHg [10].

Hypoxemia is a common event after AIS for altered control of breathing, weakness of respiratory muscles, neurogenic or cardiogenic pulmonary oedema and pulmonary embolism [52]. An adequate oxygen delivery must be ensured but hyperoxemia should be avoided, because it induces cerebral vasoconstriction, excitotoxic injury and radical oxygen species (ROS) formation [52].

13.4.3.3 Body Temperature

The SNACC suggests to maintain body temperature between 35 and 37 °C and to treat febrile patients with antipyretics and cooling devices [10]. Fever has been demonstrated to increase death or dependency risk in patients with AIS [67].

Medical evidence currently do not recommend deliberate hypothermia for a patient with AIS, because it is not associated with better outcomes [68, 69].

13.4.3.4 Glycaemia

The SNACC suggests maintaining glycaemia between 70 and 140 mg/dL [10].

13.5 Conclusion

For an efficient and high-quality management, patients with acute neurologic injury should be admitted in high-volume centres with a dedicated neuroteam. In such centres the anaesthesia department is increasingly involved in the treatment of patients requiring an INR procedure [70].

In the perioperative care of these patients, the main goals to improve outcome are haemodynamic stability, maintenance of adequate CPP, avoidance of secondary insults, patient immobility, rapid management of complications and smooth rapid recovery.

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