REVIEW ARTICLE

Concerns and challenges during anesthetic management of aneurysmal subarachnoid hemorrhage

Kamath Sriganesh,
Sudhir Venkataramaiah

Department of Neuro Anesthesia, National Institute of Mental Health and Neurosciences, Bengaluru, Karnataka, India

Address for correspondence:
Dr. Kamath Sriganesh,
Department of Neuro Anesthesia, National Institute of Mental Health and Neurosciences, Bengaluru, Karnataka, India.
E-mail: drsri23@rediffmail.com

ABSTRACT

Anesthetic management of patients with aneurysmal subarachnoid hemorrhage is challenging because of the emergency nature of the presentation, complex pathology, varied intracranial and systemic manifestations and need for special requirements during the course of management. Successful perioperative outcome depends on overcoming these challenges by thorough understanding of pathophysiology of Subarachnoid hemorrhage, knowledge about associated complications, preoperative optimization, choice of definitive therapy, a good anesthetic and surgical technique, vigilant monitoring and optimal postoperative care. Guidelines based on randomized studies and provided by various societies are helpful in the routine management of these patients and wherever there is a lack of high quality evidence, the available data is provided for practical management.

Key words: *Anesthetic management, ruptured aneurysm, subarachnoid hemorrhage, surgical clipping*

INTRODUCTION

Aneurysmal subarachnoid hemorrhage (aSAH) is an emergency neurological condition with a very high mortality (>25%) and significant morbidity (>50%) among the survivors.[1] These patients generally present during odd working hours, are clinically unstable, and require emergency intervention by the medical team including anesthesiologist. The acute nature of presentation and need for emergency interventions for managing such a complex pathology along with associated serious co-morbidities pose a unique challenge to the anesthesiologist during the perioperative period. A detailed knowledge of the pathophysiology of aSAH, treatment options, requirement of special monitoring and dedicated care will help overcome these challenges inherent in managing such patients. This review attempts to address the key challenges and concerns that anesthesiologists face during surgical management of ruptured intracranial aneurysm.

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METHODS

A thorough PubMed and Medline search was conducted for all publications with the key words "aSAH," "anesthesia," "cerebral protection," "intraoperative aneurysm rupture (IOAR)," "complications," "monitoring" in neurosurgical patients. Complete manuscripts were studied and only those that reported on human subjects in English language and published in the last 10 years from January 2004 to December 2013 were included. Publications were not restricted to only prospective randomized trials but also included guidelines, retrospective studies and case series/reports. Papers involving endovascular aneurysm management, unruptured aneurysm surgery, aneurysm management in special situations like pregnancy, children and giant aneurysm were excluded.

Concerns/challenges in patients with intracranial aneurysm

1. Systemic and intracranial effects of ruptured aneurysm.
2. Full brain during surgery.
3. Monitoring for ischemia and providing cerebral protection during temporary vessel occlusion (TVO).
4. Postclipping evaluation of circulation.

5. IOAR.
6. Hemodynamic manipulation.
7. Smooth induction and early recovery.
8. Detection and management of complications.

Systemic and intracranial effects of ruptured aneurysm

The intracranial effects of aSAH causing death and disability are from vasospasm, direct effects of the initial bleed, increased intracranial pressure (ICP) and rebleeding.[2] Patients with good aSAH grades are generally hemodynamically stable and less prone for perioperative adverse events. The diagnosis of aSAH itself may not be a challenge as the classical presentation is sudden severe headache described as "worst headache ever experienced in life." Other manifestations such as brief loss of consciousness, sentinel headache, nausea and vomiting, photophobia, neck stiffness, seizures and focal deficits may however cause diagnostic confusion.[3] Two most commonly used grading systemsto evaluate and stratify the risk and prognosis in aSAH are shown in Tables 1 and 2.[4,5] They help the anesthesiologist to anticipate and prepare for the likely challenges that might manifest during the perioperative period. Non-contrast computed tomography (CT) brain is the cornerstone for diagnosis of aSAH. The outcomes are significantly different in patients with different Fischer grades [Table 3]; with poor grades having increased ICP, focal deficits, significant systemic changes and poor outcome.[6]

Table 1: Modified Hunt and Hess grading

Grade	Criteria
0	Unruptured aneurysm
I	Asymptomatic or minimal headache and slight nuchal rigidity
II	Moderate-severe headache, nuchal rigidity, cranial nerve palsy
III	Drowsiness, confusion, or mild focal deficit
IV	Stupor, severe hemiparesis, vegetative disturbance
V	Deep coma, decerebrate rigidity, moribund appearance

Hypertension, diabetes, arteriosclerosis, chronic pulmonary disease, or vasospasm assigns patient to next less favorable category

Table 2: World federation of neurological surgeons' grading

Grade	GCS score	Motor deficit
I	15	Absent
II	14-13	Absent
III	14-13	Present
IV	12-7	Present/absent
V	6-3	Present/absent

GCS: Glasgow Coma Scale

Table 3: Fischer grading of aSAH based on admission CT scan

Grade	CT finding(s)
1	No blood detected
2	Diffuse thin layer of subarachnoid blood (vertical layers <1 mm thick)
3	Localized clot or thick layer of subarachnoid blood (vertical layers ≥1 mm thick)
4	Intracerebral or intraventricular blood with diffuse or no subarachnoid blood

aSAH: Aneurysmal subarachnoid hemorrhage; CT: Computed tomography

The knowledge from digital subtraction angiography (DSA) guides both definitive therapy and perioperative management based on the number, size and location of aneurysm and status of collateral circulation and vasospasm. Hemogram, renal and hepatic function, electrolytes, coagulation status, electrocardiogram (ECG), echocardiogram and chest radiograph provides information about systemic effects of aSAH. Transcranial Doppler (TCD) and ICP monitoring also help evaluation and management.

Systemic physiologic derangements are common after aSAH and require detailed evaluation and optimization. Agitation, stress, unwarranted mobilization, straining, intramuscular injection, pain and fever can increase hemodynamic stress and predispose to rebleeding. Blood pressure (BP) management is crucial to prevent occurrence of stroke, reduce rebleeding, and maintain cerebral perfusion. Risk of rebleeding in the 1st 24 h is as high as 14%. A practical goal is to maintain systolic BP <160 mmHg till obliteration of aneurysm.[1] Nicardipine, labetalol and sedation have been used to control hypertension. Almost 90% patients demonstrate some ECG changes attributable to neuro-cardiac response to sudden increase in ICP or intracranial bleed. These changes may be difficult to differentiate from that of myocardial ischemia. Severe arrhythmias (prolonged QT interval) and ischemic changes (ST changes) can result in increased hemodynamic complications, ischemic deficits, increased neurological intensive care unit (NICU) stay and mortality. Hypotension can also occur after aSAH and is a manifestation of myocardial dysfunction. It manifests from excessive catecholamine levels and myocardial band necrosis and is seen with poor grade aSAH.[7]

Pulmonary complications occur in about 80% of patients with aSAH. Massive catecholamine release during ictus can cause pulmonary hypertension, increased hydrostatic pressure and pulmonary edema, increasing the risk of morbidity and mortality.[8] Myocardial dysfunction and treatment for vasospasm with hypervolemia can aggravate this complication. Poor grade patients can aspirate oro-pharyngeal and gastric contents into the lungs due to depressed consciousness. Intubation and ventilation may be required in such patients to both protect the airway and to stabilize respiratory parameters.

Hyponatremia is a common complication occurring in one-third of aSAH patients. This is secondary to syndrome of inappropriate diuretic hormone (SIADH) or cerebral salt wasting (CSW). CSW occurs from increased natriuretic peptide secretion and causes hyponatremia with diuresis and natriuresis, reduces total blood volume and increases risk of vasospasm. SIADH manifests as euvolemic hyponatremia with concentrated urine from excessive ADH secretion. CSW is managed by administering isotonic fluids and fludrocortisone while SIADH is corrected with fluid restriction. Severe and refractory hyponatremia may warrant hypertonic saline administration. Other electrolyte disturbances in these patients include hypomagnesemia, hypokalemia and hypocalcemia.[8] Therefore, intravascular volume and electrolyte status should dictate type and quantity of fluids, with a goal to maintain euvolemia and normal electrolyte function. Persistent hyperglycemia (>200 mg/dl for >2 consecutive days) increases the likelihood of poor outcome after aSAH.[9] Fever is a common occurrence (70%) especially in poor grades, contributes to adverse outcome and may not always respond to conventional treatment.[8]

Planning of intervention-coiling or clipping

Evaluation of treatment modality is important to anticipate accompanying challenges. In general, surgically managed patients include those with parenchymal hematoma and large aneurysm, while endovascular therapy is preferred in elderly, patients with significant co-morbidity, poor grades and basilar artery aneurysm.[1] This knowledge will help in formulating appropriate anesthetic plan and prepare for intervention-related adverse events. The International Subarachnoid Aneurysm Trial showed better outcomes with endovascular treatment compared to surgery.[10] Since then, increasing number of patients are managed by endovascular technique shifting the anesthetic management outside the operating room.

Timing of surgery

Time from rupture to treatment is a crucial factor in determining outcome.[8] Practice of delayed surgery to avoid edematous brain has been replaced by early surgery to minimize risk from rebleeding and vasospasm. Mahaney *et al.* in their analysis of intraoperative hypothermia for aneurysm surgery trial (IHAST) data observed that patients operated early (day 0-2) or late (day 7-14) fared significantly better than those operated during intermediate phase (day 3-6).[11] Change in surgical strategy has posed challenge to the anesthesiologists as increasing number of patients are operated during off-work hours in inadequately optimized state with less expert help.

Challenges during anesthetic induction and intubation

General goals include smooth induction and hemodynamic control to prevent rebleeding. Propofol or thiopentone in liberal doses attenuates hemodynamic response and rebleeding risk in good grade patients. In poor grade patients with raised ICP, dose reduction is required as induction hypotension can compromise cerebral perfusion. Intubation should be smooth and swift to minimize hemodynamic stimulation as rupture of the aneurysm during intubation is associated with poor outcome.[12] Difficult intubation may be encountered when a large internal carotid artery (ICA) aneurysm protrudes into the oral cavity.[13] The risk from hemodynamic and hypoxic stress associated with repeated attempts at intubation can have an adverse bearing on the outcome.

Maintenance of anesthesia

Both intravenous and inhalational anesthetic technique may be used for maintenance keeping in mind the objectives of stable intraoperative hemodynamics, early smooth recovery and effect on special monitoring techniques. Cerebral perfusion increases with isoflurane when compared with propofol without increase in ICP in aSAH.[14] Hypocapnia is not essential in good grade patients as it can reduce ICP and increase transmural pressure within aneurysmal sac predisposing it to rupture. In poor grade patients, hyperventilation however is beneficial to reduce ICP and provide lax brain.

Challenges from a full brain

Brain laxity is crucial to obtain good surgical access to the aneurysm without causing IOAR or compromising underlying brain from excessive retractor pressure. This is important as early surgery risks a tense/full brain and dissection without adequate exposure can result in IOAR. Both 20% mannitol and 3% hypertonic saline are suitable osmotic agents for intraoperative brain relaxation in the dose of 2-4 ml/kg. Head end elevation, avoiding jugular venous compression, avoiding high concentration of inhalational agents and nitrous-oxide and mild hyperventilation are other measures to achieve a lax brain. If full brain persists, additional measures like moderate hyperventilation, switching to intravenous anesthetic maintenance and release of cerebrospinal fluid might be helpful.

Monitoring for ischemia and cerebral protection during temporary vessel occlusion

Whenever possible, direct clipping is preferred. However, when it is not possible or anticipated to be difficult, TVO is performed to facilitate peri-aneurysmal dissection and safe permanent occlusion of the aneurysm. Hypotension, used earlier, reduced the pressure within the aneurysmal neck and facilitated clipping. However, hypotension in an already injured brain increased ischemic complications.[15] TVO proximal to the aneurysm and occasionally distal to it when collateral circulation is good, reduced the pressure and flow into the aneurysmal sac and facilitated safe

clipping without compromising perfusion to other parts of the brain and minimized risk of IOAR.[16] However, if the TVO duration is prolonged (>20 min), it predisposes distal areas of the brain to ischemia. In general, a TVO time of 5 min followed by reperfusion for 5 min before repeat TVO is ideal. As it may not always be possible to adhere to these timelines, cerebral protective measures may be required. Induced hypertension, and/or suppression of cerebral activity with hypothermia and/or pharmacological agents help reduce cerebral metabolism and improve tolerance to ischemia. Suzuki *et al.* recommended Sendai cocktail (Mannitol, Vitamin E and steroid) for cerebral protection during TVO.[17] Kim and Park compared the effect of thiopentone and etomidate on brain protection using burst suppression during TVO and observed that both the drugs produced similar duration (11 min) and magnitude of burst suppression. They suggested repeat administration of either drug if TVO duration is prolonged.[18] The IHAST trial could not demonstrate difference in neurological outcome[19] or cardiovascular events[20] between hypothermic (33°C) and normothermic (36.5°C) patients. However, another study compared temperatures of the deteriorated and non-deteriorated patients at different stages during aneurysm surgery. When the patients were divided into hypothermic (<34.5°C) and normothermic groups (>34.5°C) during TVO, hypothermic group tended to have a lower incidence of neurological deterioration at 24 h.[21] Hindman *et al.* in their *post-hoc* analysis of IHAST trial observed that neither hypothermia (upto 33°C) nor pharmacological protection (with thiopentone or etomidate) affected short and long term neurologic outcomes of 441 patients who underwent TVO. Patients with temporary clip duration of >20 min (13% of total patients) had less favorable outcome despite cerebral protection.[22] In good grade patients, based on the existing evidence, maintenance of normal physiology (normotension, euglycemia, normocarbia, and normoxia) without additional cerebral protective interventions is sufficient. In poor grade patients with inadequate cross circulation and where dissection or aneurysmal anatomy is difficult (anticipated prolonged TVO) and potential for IOAR is high, pharmacological protection may be beneficial. In addition, passive hypothermia may be permitted and BP maintained 20% above the preclip level.

Monitoring during aneurysm surgery

Somato-sensory evoked potential (SSEP) has been used to predict postoperative stroke in patients undergoing aneurysm clipping. A <15 ml/100 g/min regional cerebral blood flow resulted in a drop of SSEP amplitude to 50% of baseline. SSEP monitoring helps in detecting effect of changes in the anesthetic depth, TVO, hemodynamic changes and surgical manipulation. It allows detection of cerebral ischemia, facilitates timely corrective measures and predicts postoperative neurological deficits. Wicks *et al.* in their study on 691 patients, found that in unruptured aneurysms, irreversible SSEP changes were associated with 80% stroke rate. In ruptured aneurysms, however, irreversible changes were associated with 42% stroke rate.[23] Motor evoked potential (MEP) is sensitive for predicting motor deficits following aneurysm surgery with permanent loss of MEP predicting postoperative motor impairment.[24] Bispectral index (BIS) monitoring might facilitate identification of lower limit of cerebral autoregulation during aneurysm surgery and help maintain safe level of BP to prevent ischemic insult.[25] BIS and electroencephalogram not only help in detecting ischemic changes during TVO, but also help in titrating anesthetic to achieve metabolic endpoint.

Postclipping evaluation

Another challenge during aneurysm surgery is to ensure noninclusion of normal vessel/perforators within the clip and perform complete aneurysmal isolation. This is done with either intraoperative microvascular Doppler (IMD) or indocyanine green video-angiography (ICG-VA) as they are simple and safe. Anesthesiologists administer ICG and also help perform IMD. ICG-VA appropriately assessed vessel patency and aneurysm obliteration in 93.5% of 109 aneurysms clipped.[26] However, ICG can cause transient oxygen desaturation.[27] IMD use confirms aneurysm isolation and patency of parent vessel and branching arteries. Hui *et al.* observed that clip repositioning was required based on IMD findings in 24% of aneurysms clipped in 91 patients and concluded that IMD could reduce the rate of residual aneurysm and unanticipated vessel stenosis.[28]

Challenges during intraoperative aneurysm rupture and its management

Intraoperative aneurysm rupture is a nightmare for both surgeon and the anesthesiologist. It results in rapid and significant physiological derangement. While securing the aneurysm quickly and safely is the primary focus of the surgeon, anesthesiologist needs to protect the brain from possible ischemia during the unplanned prolonged TVO and correct hemodynamic changes. TVO provides a blood-less field and helps placement and/or readjustment of permanent clip. Delay can result in significant blood loss causing hemodynamic instability and puts both distal and remote neurons to ischemic risk. A transient low-normal BP may be permitted if IOAR occurred during clip application. This facilitates clip placement in presence of a bloody field. Also pharmacological protection might not be feasible during IOAR when both intravascular volume and BP is low. Intravascular volume resuscitation with isotonic fluids and blood should be aggressively pursued. Though IOAR is not always associated with poor neurological outcome,[29] the outcome is likely to be

poor if the IOAR occurs during early part of surgery, as there is delay in securing the aneurysm and physiological changes are ongoing.

Hemodynamic manipulation postclipping

Cerebral vasospasm can occur from surgical handling of vessels and also from earlier SAH. Hence, BP must be maintained about 20% above the preclipping range to maintain cerebral perfusion through the spastic vessels and reduce ischemic complications. In addition, maintaining normovolemia (central venous pressure [CVP] of 10) and normal oxygenation (hemoglobin of 10 g%) is desirable. Various drugs have been used to achieve and maintain hypertension including norepinephrine, phenylephrine and dopamine. However, excessive hypertension can cause intracranial hematoma.[30]

Challenges during recovery from anesthesia

Recovery and extubation depends on the preoperative status and intraoperative events. Poor preoperative aSAH grade, prolonged TVO and severe intraoperative vasospasm are possible indications for postoperative ventilation. These patients are likely to deteriorate necessitating vigilant monitoring. Good grade aSAH patients can be successfully extubated on table using standard extubation criteria. Anesthetic maintenance should permit swift but smooth extubation for early neurological assessment with minimal hemodynamic fluctuation. If there is undue delay in recovery, anesthetic factors should be excluded before surgical cause is considered and evaluated.

Detection and management of complications

Patient should be ideally monitored in the NICU for at least 1st 24 h after surgery. Anticonvulsants, osmotherapy and nimodipine must be continued. Hydrocephalus, vasospasm, seizures, and electrolyte disturbances can occur necessitating close observation and prompt management. One of the major challenges in the management of aSAH is identifying potential or ongoing perfusion deficits. Ischemic insults can occur following ictus, or due to raised ICP, hypotension and vasospasm. Early identification and appropriate treatment of postictal intracranial (ICP, TCD flow velocities) and cardiovascular (cardiac output, ECG, BP, CVP) changes is possible in dedicated NICU and is crucial for improving outcomes. Heuer *et al*. observed that raised ICP (>20 mmHg) occurred in >50% of patients after aSAH and was associated with poor outcomes. Factors associated with raised ICP included poor clinical and radiological grades of aSAH, intraoperative brain swelling, parenchymal and intraventricular bleed and rebleeding.[31] Failure of cerebral autoregulation has been shown in patients with aSAH even before vasospasm sets in and contributes to delayed ischemic neurological deficits (DIND) along with vasospasm.[32]

Vasospasm is an important cause for mortality following aSAH affecting as many as 70% of patients. It usually occurs between 4th and 21st days of aSAH and is responsible for DIND and cerebral infarcts. Risk factors for vasospasm include age, Fischer grade and volume of blood. Vasospasm should be suspected when there is neurological deterioration (headache, new/worsening focal deficits or decline in sensorium). Serial TCD examination is a noninvasive, repeatable bedside tool to predict vasospasm before clinical presentation. It also helps in assessing effect of therapeutic interventions. Mean cerebral blood flow velocity (CBFV) in anterior circulation of >120 cm/s and >60 cm/s in posterior circulation is suggestive of vasospasm. A Lindegaard ratio (ratio of middle cerebral artery to extracranial ICA) of >3 and >6 is indicative of mild-moderate and severe vasospasm respectively and differentiates vasospasm from hyperperfusion. An increase in CBFV of >50% in 24 h is also predictive of vasospasm.[33] Other techniques for diagnosing vasospasm include perfusion CT scan and DSA. Nimodipine is the mainstay for both prevention and management of aSAH. Hypotension occurs when it is concurrently administered with mannitol. Triple H (hypertension, hypervolemia and hemodilution) therapy results in clinical improvement when administered within 2 h of neurological deterioration.[34] Recent evidence however suggests increased pulmonary and cardiac complications with this therapy. Therefore, only hypertension component of triple H therapy is currently recommended.[35] Other treatment modalities for vasospasm include magnesium targeted to serum level of 2-2.5 mmol/l (blocks Calcium and n-methyl-D-aspartate [NMDA] channels and reduces vasospasm), statins (decreases microthrombi, causes NMDA antagonism, fibrinolysis and immunomodulation), erythropoietin (by promoting hematopoiesis, increasing BP and neuroprotective mechanism), stellate ganglion block, intra-arterial nimodipine and balloon angioplasty.[36]

Anemia is common in patients with aSAH (50% incidence) and is associated with reduction in cerebral oxygenation and poor outcome.[37] Hemoglobin of 10-11 g% is ideal in these patients. However, red blood cell (RBC) transfusion has been shown to increase medical complications including infection by 3-fold.[38] Hence, any benefit from correction of anemia with RBC transfusion has to weighed against its possible adverse complications.

Acute neurological deterioration occurs in >40% of aSAH patients and hence close monitoring is required. Factors associated include age, timing of surgery, Fisher grade, preoperative interventions such as ventriculostomy, intraoperative BP, ST segment changes and blood loss, duration of TVO and difficulty in aneurysm exposure. Of the patients who had neurological deterioration only 50% had good outcome at 3 months.[39]

The guidelines relevant to the anesthesiologists in the day-to-day perioperative management of patients with ruptured intracranial aneurysm given by various societies are summarized in the Table 4.[40-42]

CONCLUSION

Patients with aSAH are challenging to manage and require a thorough knowledge about the pathophysiology of aSAH and treatment options. Vigilant peri-operative monitoring, adherence to good surgical and anesthetic technique and prompt detection and management of complications are likely to improve the outcome. At present, there is paucity of high quality data regarding most aspects of intraoperative anesthetic management of aneurysm surgery. Furthermore, it is not possible currently to have evidence based on the randomized clinical trials for all concerns and dilemma affecting the clinical management. This review

Table 4: Summary of the guidelines provided by various societies regarding perioperative management of aneurysmal subarachnoid hemorrhage

Guidelines	Diringer *et al.*[40]	Bederson *et al.*[41]	Steiner *et al.*[42]
Cardiopulmonary Complications and monitoring	Baseline cardiac assessment with serial enzymes, electrocardiography, and echocardiography is recommended. Cardiac output monitoring may be useful in patients with hemodynamic instability or myocardial dysfunction. In case of pulmonary edema or evidence of lung injury, euvolumia should be targeted avoiding excessive fluid intake and judicious use of diuretics. Standard management of heart failure is indicated while maintaining CPP/MAP		Monitoring should consist of atleast continuous ECG monitoring, GCS, focal deficits, blood pressure and temperature at least every hour
Fluid and electrolyte management	Fluid restriction should not be used to treat hyponatremia. Treatment with hydrocortisone or fludrocortisone limits natriuresis and hyponatremia. Hypertonic saline solutions can be used to correct hyponatremia. Serum glucose should be maintained between 80 and 200 mg/dl. Administration of corticosteroids is not recommended in acute SAH		Intravenous fluids of 3 L/day (0.9% isotonic saline) should be administered aiming for normovolemia. Monitoring of electrolytes, glucose and white blood cell count at least every other day should be done. Hyperglycemia over 10 mmol/l should be treated
Hemodynamic monitoring and management	Central venous lines should not be placed solely to obtain CVP measures and fluid management based solely on CVP measurements is not recommended. The goal should be maintaining euvolemia. Isotonic crystalloid is the preferred agent for volume replacement. In patients with a persistent negative fluid balance, use of fludrocortisone or hydrocortisone may be considered. Patients clinically suspected of DCI should undergo a trial of induced hypertension. Blood pressure augmentation should progress in a stepwise fashion with assessment of neurologic function at each MAP level to determine if a higher blood pressure target is appropriate. The choice of vasopressor should be based on the other pharmacologic properties of the agents. If nimodipine administration results in hypotension, then dosing intervals should be changed to more frequent lower doses. If patients with DCI do not improve with blood pressure augmentation, a trial of inotropic therapy may be considered	Blood pressure should be monitored and controlled to balance the risk of stroke, hypertension-related rebleeding, and maintenance of CPP. Administration of large volumes of hypotonic fluids and intravascular volume contraction should generally be avoided after SAH. Monitoring volume status in certain patients with recent SAH using some combination of CVP, pulmonary artery wedge pressure, fluid balance, and body weight is reasonable, as is treatment of volume contraction with isotonic fluids. The use of fludrocortisone acetate and hypertonic saline is reasonable for correcting hyponatremia	Hypertension should not be treated unless it is extreme; limits for extreme blood pressures should be set on an individual basis, taking into account age of the patient, pre-SAH blood pressures and cardiac history; systolic blood pressure should be kept below 180 mmHg, only until securing of ruptured aneurysm, to reduce risk for rebleeding. This may be achieved by applying analgesics and nimodipine. If systolic pressure remains high despite these treatments further lowering of blood pressure should be considered. If blood pressure is lowered MAP should be kept >90 mmHg
Anesthetic and intraoperative management	Cautious blood pressure elevation to improve perfusion might be attempted, weighing potential risks and benefits in unsecured ruptured aneurysm. Measures should be taken to minimize blood loss. Patients should receive packed RBC transfusions to maintain hemoglobin concentration above 8-10 g/dl	Minimizing the degree and duration of intraoperative hypotension during aneurysm surgery is indicated. There are insufficient data on pharmacological strategies and induced hypertension during temporary vessel occlusion to make recommendations, but there are instances when their use may be considered reasonable. Induced hypothermia may be a reasonable in some cases but is not routinely recommended	

CPP: Cerebral perfusion pressure; MAP: Mean arterial pressure; SAH: Subarachnoid hemorrhage; CVP: Central venous pressure; DCI: Delayed cerebral ischemia; RBC: Red blood cell; ECG: Electrocardiogram; GCS: Glasgow Coma Scale

specifically attempted to address the day-to-day challenges faced by anesthesiologist in the perioperative management of patients with aSAH.

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