

Anesthesia For The Surgery Of Intracranial Aneurysms: Part III

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Abstract

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INTRAOPERATIVE MANAGEMENT PREMEDICATION

Grade III -IV patients (see table 1) with reduction in the level of consciousness do not require preoperative sedation. In patients with grades I - II the preoperative visit can be sufficient to calm the patient. A strong premedication can produce respiratory depression with hypercapnia and increase of cerebral blood flow (CBF) and intracranial pressure (ICP). Premedication is used to reduce the hemodynamic response associated with anxiety. Normally small doses of benzodiazepines are sufficient for predication.

Figure 1

Table 1. Clinical Grades according to the Hunt and Hess Scale

Grade	Criteria	Index of Perioperative Mortality (%)
0	Aneurysm is not ruptured	0-5
I	Asymptomatic or with minimal headache and slight nuchal rigidity	0-5
II	Moderate to severe headache, nuchal rigidity, but no neurologic deficit other than cranial nerve palsy	2-10
III	Somnolence, confusion, medium focal deficits	10-15
IV	Stupor, hemiparesis medium or severe, possible early decerebrate rigidity, vegetative disturbances	60-70
V	Deep coma, decerebrate rigidity, moribund appearance	70-100

MONITORING

CARDIOVASCULAR MONITORING

Strict control of blood pressure is mandatory during anesthesia for patients with ruptured intracranial aneurysms in order to prevent rebleeding. Oscillations of cerebral perfusion pressure (CPP) and ICP have to be avoided in order to stabilize transmural pressures of the aneurysm. On the other hand, one will have to avoid hypotension which can cause cerebral ischemia.

Monitoring of the arterial pressure must be invasive in order to obtain control of hemodynamics and dosage of vasoactive drugs. Arterial pressure has to be monitored prior to induction of anesthesia. Insertion of the arterial catheter can be performed in local anesthesia combined with mild sedation (benzodiazepines and narcotics). There is controversy regarding the need for invasive monitoring prior to induction because of the degree of anxiety that can cause rebleeding of the aneurysm.

Measurement of central venous pressure (CVP) is necessary for handling intravascular volume. This is especially important after clipping of aneurysms. Hypervolemia needs then to be maintained in patients with risk of vasospasm. In addition, volume status has to be controlled if hyperosmolar and diuretic substances are administered. It must be taken into account that there is no strict correlation between CVP and ventricular pressures at the end of the diastole (92). Monitoring of the pressure in the pulmonary artery is useful in handling hemodynamic responses resulting from barbiturate coma. The selection between central venous catheter or pulmonary artery catheter depends on the patient. The pulmonary artery catheter is especially useful in patients with a history of coronary illness.

Routine monitoring should include heart rate, electrocardiogram, pulseoximetry, esophagus stethoscope,

and continuous ETCO₂. Some stethoscopes incorporate a thermometer in order to monitor temperature.

NEUROLOGICAL MONITORING

The intraoperative neurological monitoring of evoked potentials is useful in detecting the presence of cerebral ischemia associated with temporary vascular occlusion during aneurysm surgery. Somatosensory evoked potentials during temporary vascular occlusion do not detect with good reliability ischemia in the cortical and subcortical motor zones or in other topographically not represented regions. There is an incidence of 38 to 60 % of false positives and 5 to 34 % of false negatives results (^{93, 94}). Both, auditive evoked potentials as well as somatosensory evoked potentials can be used if patients suffer an aneurysm of the posterior circulation (^{95, 96}).

Intraoperative electroencephalography can also help to detect ischemia during cerebral aneurysm surgery.

INTRACRANIAL PRESSURE MONITORING

Some groups monitorize the ICP with an intraventricular catheter (⁹⁷) because there is a high probability of increase in ICP after a subarachnoidal hemorrhage (SAH). Drainage of the cerebrospinal fluid (CSF) in these cases can improve the patients condition and the intraoperative handling.

CONTINUOUS ETCO₂ MONITORING

Hypercapnia may cause cerebral vasodilatation and increase ICP.

Monitoring of Diuresis:

- Placing an urinary catheter is mandatory with the use of mannitol.

INDUCTION

The main anesthetic goals during cerebral aneurysm surgery:

- To facilitate surgery by creating good intraoperative conditions.
- To reduce the risk of intraoperative rupture of the aneurysm.
- To provide a quiet and stable awakening.
- To maintain an adequate cerebral perfusion.

Pressure changes in intracranial hemodynamics during induction can result in a rupture of the aneurysm. The goal

of the anesthetic handling is to minimize hemodynamic alterations.

Laryngoscopy, intubation and all the drugs applied in a short period of time can provoke fluctuations in pressures of cerebral perfusion that can alter the cerebral hemodynamics and consequently the stability of the transmural pressure of an aneurysm.

The most commonly used agent for induction is thiopental. Thiopental reduces the cerebral O₂ consumption and the cerebral blood flow thus reducing the intracranial volume and the ICP. A dose of 3 - 6 mg/kg does not reduce the hemodynamic responses to external stimuli. Such a dose is associated with a reduction in perfusion pressure of 10 - 15 %. Therefore, one has to supplement anesthesia with opioids (fentanyl 5 -10 mg/kg), lidocaine (1.5 - 2 mg/kg), esmolol (0.5 mg/kg), or labetalol (10 - 20 mg) and/or inhaling agents 90 seconds before laryngoscopy in order to reduce the transmural pressure of the aneurysm (^{98, 99}). The use of alfentanil (25 - 50 mg/kg) and sufentanil (0.5 -1.0 mg/kg) in neurosurgical patients is controversial due to the publication of some reports which indicated an increase of ICP and CBF following these drugs (^{100, 101}).

Propofol (1.5 - 2.5 mg/kg) offers similar advantages as etomidate (0.1 - 0.2 mg/kg). Propofol produces a significant reduction of arterial pressure. The minimal cardiotoxicity of etomidate makes this an attractive drug for this kind of surgery. Etomidate and propofol are drugs which produce similar effects in regard of reduction of transmural pressure and cerebral metabolism, thus being a reasonable alternative to thiopental.

The rupture of an aneurysm during induction is associated with a mortality rate of 75% and has an incidence of 1 - 2 % (¹⁰²).

SAH can damage autoregulation and provoke vasospasm. Combined with hypotension during induction it could produce neurological deficits.

Neuromuscular blockers do normally not cross the blood-brain barrier. It is necessary to choose a muscle relaxant that provides greatest hemodynamic stability and has the least possible effects on cerebral hemodynamics. Vecuronium has little effect on the ICP and CBF and is consequently the first choice in cerebral surgery (¹⁰³).

Succinylcholine is reported to produce an increase in ICP (^{104, 105}), attributed to its activity at the muscle spindle. It can

be reduced by defasciculation. Succinylcholine can also produce hyperkalemia. Serious arrhythmia have been reported after administration of succinylcholine in cerebral aneurysm surgery (¹⁰⁶).

Laryngoscopy and intubation can produce a strong sympathetic stimulus which can be very dangerous for patients with intracranial aneurysms. During induction, once complete relaxation is obtained, anesthesia should be supplemented with doses of opioids, thiopental and/or lidocaine before laryngoscopy. The use of betablockers and vasodilators can protect against the effects of intubation, but they can also raise the ICP. An alternative is the use of isoflurane with hyperventilation before intubation. The careful combination of iv drugs and volatile anesthetics can produce a quiet induction with hemodynamic stability and stable ICP during the intubation (¹⁰⁷).

MAINTENANCE

The effects of the drugs used for maintenance of anesthesia in the surgery of intracranial aneurysms should lead to:

- Systemic and cerebral hemodynamic stability.
- Cerebral relaxation and facilitation of surgical exposure.
- Cerebral protection from ischemia .
- Reduction of formation of vasogenic cerebral edema and control of ICP.
- Maintain normovolemia or moderate hypervolemia.
- Maintain isotonicity of plasma.
- Normalization of blood sugar.
- Obtain a quick awakening in order to be able to make an early neurological test.

The drugs normally used are nitrous oxide with oxygen, narcotics such fentanyl, isoflurane as inhaling agent, propofol and vecuronium as a non-depolarizing muscle relaxant . The comparison of three anesthetic techniques for resection of intracranial masses (isoflurane/fentanyl vs. nitrous oxide/fentanyl vs. propofol/fentanyl) demonstrated equal results between the three groups (¹⁰⁸).

The use of nitrous oxide can increase the ICP due to increase of the CBF (^{109, 110}). The use of nitrous oxide in cerebral

surgery is controversial.

In order to obtain an sufficient cerebral relaxation it is a necessary position the head adequately. The head should be slightly raised above the right auricle, without excessive rotation and flexion in order to avoid jugular compression and facilitate venous drainage.

In the acute phase of the SAH, draining the CSF during interventions represents an effective way for cerebral relaxation. It is contraindicated to drain lumbar CSF in cases of high ICP prior to opening the dura mater. A quick reduction of the CSF pressure can lead to cerebral herniation and to an increase of the transmural pressure of the aneurysm with resulting potential of rebleeding.

Painful stimuli may lead to hypertension. It is therefore necessary to raise the level of anesthesia with thiopental, opioids, inhaling agents and/ or sympathetic blockers before placing a craniostat . The hemodynamic response to the placement of the pins of the craneostat can also be reduced by prior infiltration with local anesthetic (¹¹¹).

Administration of crystalloids can compensate for small blood losses and maintain urine output. Severe hypovolemia in patients with an SAH is associated with cerebral ischemia and neurological deficit due to vasospasm (¹¹²). The old practice of dehydrating, used for years, has been replaced with new neuroanesthesia practices. Some authors recommend the use of prophylactic hypervolemic therapy before and during the clipping of the aneurysm in order to try to increase the CBF and to reduce the effects of perioperative vasospasms (^{113, 114}). Before clipping an aneurysm it is necessary to maintain normovolemia and to have an adequate blood pressure and cardiac output. After clipping the aneurysm moderate hypervolemia must be maintained, keeping the CVP higher than normal. Crystalloids, hypertonic or isotonic solutions are used to prevent hypoosmolality of the plasma that can lead to cerebral swelling. The osmolality of the plasma has more effect on the cerebral edema than oncotic pressure.

A moderate hemodilution to hematokrits of 30 to 35 % improves cerebral perfusion (^{115, 116}). This is controversial. The reduction in O₂ content can be compensated with the increase of cerebral blood flow.

Administration of intraoperative mannitol reduces the intracranial volume and facilitates cerebral retraction. The time of administration of mannitol will depend on the ICP. The doses of mannitol vary between 0.25 to 1 g/kg,

depending on the urgency of reducing the ICP and the estimated duration of this reduction. Quick administration produces a significant but temporary reduction of the systematic vascular resistance (¹¹⁷). Mannitol is administered after the bone flap is raised in order to avoid the risk of tearing the bridging veins due to premature retraction of the brain. It is also recommended to administer mannitol only after opening of the dura mater since an increase of intravascular volume with a decrease of ICP may increase the transmural pressure of the aneurysm with the potential danger of rupture (¹¹⁸). Mannitol produces, due to its osmotic effect, a rapid increase of intravascular volume with increase of cerebral blood flow and reduction of the ICP. The effect of mannitol begins to reduce the ICP and the intracranial volume after 5 -15 min and has a peak effect after 30 - 45 min. It can be used at high doses (2 g/kg) to increase the CBF and produce cerebral protection during temporary vascular occlusion. High doses of mannitol produce electrolytic alterations such as a temporary increase of serum potassium and reduction of sodium (^{119, 120}). The effect of mannitol can be increased by adding furosemide at doses of 0.1 to 1.0 mg/kg iv. Electrolytes should be monitored. Mannitol can produce an overcharge of volume in patients with deterioration of the ventricular function.

Lumbar drainage reduces the intracranial volume and the need for osmotic diuretics with the advantage of not increasing intravascular volume. It should be performed with the dura open in order to avoid changes of intracranial pressure which could affect the transmural pressure of the aneurysm. The speed of drainage of the CSF should be approx. 5 ml/min in order to avoid hypertension and reflex bradycardia.

During the intervention, the patient should be kept in normoglycemia. Cerebral ischemia can be aggravated in the presence of hyperglycemia (^{123, 124}). Occurrence of neurologic deficit and focal cerebral ischemia associated with hyperglycemia has been demonstrated in experimental models (124). Solutions containing glucose should not be used.

Hyperventilation producing hypocapnia has been a routine practice in neuroanesthesia. Recently, there has been an increasing interest regarding the varieties and applications of hypocapnia in the preoperative period. Hyperventilation is the simplest and most accessible method for the anesthesiologist to influence cerebral hemodynamic. Hyperventilation produces three effects:

- A reduction of cerebral mass due to reduction of the cerebral blood volume with a reduction of the CBF.
- Redistribution of blood flow from normal regions to ischemic regions.
- A compensating effect for cerebral acidosis due to an increase of extracellular pH (¹²⁵).

Hyperventilation has two practical applications in neuroanesthesia:

- As an specific or prophylactic treatment of intracranial hypertension during induction and during the surgical period prior to the opening of the dura mater.
- After the opening of the dura mater in order to provide better surgical access and less pressure on the separators on the cerebral tissue. There is no established method to quantify the degree of relaxation obtained. It has been proposed to measure pressure on the separators but it has not been developed (^{126, 127}).

CO₂ is a powerful modulator of the cerebral vascular resistance. It diffuses quickly through the blood-brain barrier modulating the pH of the extracellular liquid and acting over arteriolar resistance. Vasoconstriction induced by hypocapnia develops within 6 to 10 hours (¹²⁸). Prolonged hyperventilation has been suggested to result in hypersensitivity of the vessels to the PaCO₂. In normotensive patients the CBF is modified by 2 to 4 % for each mm of Hg of variation of the PaCO₂.

Increasing PaCO₂ from 40 to 80 mm of Hg doubles the CBF. Reducing the PaCO₂ from 40 to 20 mm of Hg reduces the CBF in half. Arteriolar tone determined by the systemic arterial pressure and modulates the effect of the PaCO₂ over CBF. A moderate hypertension reduces the capacity of the cerebral circulation to respond to the variation of the PaCO₂, and serious hypertension abolishes it completely (¹²⁹). At the same time, the PaCO₂ changes the capacity for CBF autoregulation. A PaCO₂ lower than 20 mm of Hg can provoke cerebral ischemia due to vasoconstriction and displacement of the Hb dissociation curve to the left (^{130, 131}). Such extreme levels of hypocapnia should never be reached.

Improvement in the collateral perfusion could be

demonstrated by using induced hypertension and hypocapnia in patients undergoing carotid endarterectomy (¹³², ¹³³, ¹³⁴, ¹³⁵, ¹³⁶).

Deep hypocapnia should never be used blindly during craniotomy. It is necessary to evaluate the adequacy of cerebral relaxation based in the needs of the surgeon. In addition, objective physiological parameters such as the pressure of the separators, its effect on the cerebral electrical activity or the local CBF, and the arteriovenous difference of the O₂ contents (jugular or transcranial oximetry) are of importance. In absence of direct methods of evaluation of cerebral circulation, blood gases should be monitored at regular intervals and the Petco₂ should be measured continuously. It is reasonable to maintain a Paco₂ as high as possible compatible with intraoperative conditions. Objective measures of global and regional cerebral perfusion should be soon available for controlled handling of the Paco₂ (125).

Hyperventilation in cerebral aneurysm surgery should start after opening the dura mater. An average hypocapnia of 30 to 35 mm of Hg can be maintained thereafter. Patients should be kept in normocapnia during induced hypotension or after securing the aneurysm, and normocapnia should also be maintained in patients who suffer vasospasm (¹³⁷).

AWAKENING AND RECOVERY

External stimuli during emergence from anesthesia such as repeated suction of secretions of the oropharynx, fast movements of the bandaged head, and extubation should be reduced or anticipated as much as possible. Lidocaine 1.5 mg/kg or opioids (fentanyl 0.5 to 1mg/kg) before extubation may help to reduce coughing.

Arterial pressure of more than 10 to 20% of the preoperative value are beneficial after clipping the aneurysm. Pressures above these figures should be avoided. When a hypertensive response occurs during/after extubation, antihypertensive drugs like nifedipine, hydralazine or beta-blockers can be used.

The neurological exploration must be performed promptly in patients with grades I and II. Patients with higher grades (including alteration in the level of consciousness prior to the intervention) should not be awakened and kept intubated until they can maintain a free airway with adequate ventilation and a stable neurological state.

The patient should be transferred to a recovery room monitored and watched by personnel experienced in

handling neurosurgical patients.

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