Anesthesia For The Surgery Of Intracranial Aneurysms: Part IV

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Citation

J Crespo. *Anesthesia For The Surgery Of Intracranial Aneurysms: Part IV*. The Internet Journal of Anesthesiology. 1997 Volume 2 Number 4.

Abstract

Find here Part I, II and III of this review article: Martinez-Chacón Crespo JL: Anesthesia for the Surgery of Intracranial Aneurysms, Part I. The Internet Journal of Anesthesiology 1998; Vol2N1: Published January 1, 1998; Last Updated January 1, 1998. Martinez-Chacón Crespo JL: Anesthesia for the Surgery of Intracranial Aneurysms, Part II. The Internet Journal of Anesthesiology 1998; Vol2N2: Published April 1, 1998; Last Updated April 1, 1998 Martinez-Chacón Crespo JL: Anesthesia for the Surgery of Intracranial Aneurysms, Part III. The Internet Journal of Anesthesiology 1998; Vol2N3: Published July 1, 1998; Last Updated July 1, 1998.

SPECIAL TECHNIQUES

The special techniques used to increase safety during clipping of an aneurysm and to reduce intraoperative cerebral damage by ischemia or hemorrhage are:

- moderate hypothermia,
- controlled hypotension,
- temporary vascular occlusion,
- induced hypertension,
- circulatory arrest during deep hypothermia,
- pharmacological cerebral protection.

INDUCED HYPOTENSION AND TEMPORARY VASCULAR OCCLUSION

Both techniques are used in order to reduce the tension on the wall of the aneurysm thus decreasing the transmural pressure. This will facilitate its dissection and the application of the clip.

Induced hypotension has been the state-of-the-art method to facilitate clipping of aneurysms. However, today induced hypotension is questioned by some authors with respect to temporary vascular occlusion ([138],[139]). Induced hypotension produces hypoperfusion in the organs of those patients with altered cerebral circulation, coronary disease, anemia, severe hypovolemia and advanced age.

Induced hypotension is used during the approach and surgical dissection of the aneurysm. Nevertheless, it is necessary to maintain a blood pressure within the limit of autoregulation of cerebral blood flow (between 50 and 60 mm of Hg mean blood pressure) in order to insure an adequate cerebral perfusion. This range is different in hypertensive patients. Their autoregulation curve is shifted towards the right, requiring higher mean blood pressures in order to maintain an adequate CBF and to avoid zones of ischemic brain tissue. Blood pressure drop should not exceed 40 % of the preoperative mean arterial pressure in this patient population. Induced hypotension is a relative contraindication in hypertensive patients. It might be beneficial to monitor the somatosensoric evoked potentials in such patients in order to prevent reductions in the CBF.

The limit of hypotension is not well defined. Some authors recommend that with intact autoregulation the limit should not be below 50 mmHg. In patients with disturbed autoregulation the limit should not be below 60 mmHg. Other authors think that patients can tolerate reductions of 10 to 20 % of the CBF without danger to the brain and they recommended reducing the mean arterial pressure to 40 mm Hg for short periods of time. There are favorable results with hypotension below 50 mm of Hg ([140]).

Prior to inducted hypotension an adequate intravascular volume should be maintained. During hypovolemia, control

of arterial pressure is difficult. Oliguric hypotension is associated with more tissue dysfunction than normovolemic induced hypotension. The inspired concentration of O2 should be increased during the induced hypotension. Intrapulmonary shunt during hypotension does not appear to be affected by isoflurane.

Relative contraindications for inducted hypotension include:

- cerebral arteriosclerosis,
- valvular cardiac or arteriosclerotic disease,
- significant pulmonary insufficiency,
- renal or hepatic disease.

Induced hypotension can produce coronary ischemia, inhibition of hypoxic pulmonary vasoconstriction, reduction of hepatic or renal blood flow, and hyperglycemia ([141],[142]). Drugs most commonly used for induced hypotension are: adenosine, labetalol, esmolol, trimetaphan, nitroglycerin, prostaglandin E, nipride and isoflurane.

It is not clear if intraoperative rupture of the aneurysm might be caused or prevented by induced hypotension. Iindications for induced hypotension seem also not to be clear. The use of inducted hypotension should be reserved to a thoughtful utilization in intraoperative hemorrhage or for a short period of time during application of a clip .

TEMPORARY VASCULAR OCCLUSION

A method to reduce the transmural pressure of the aneurysm is to occlude the feeding vessel with a temporary clip (Fig. 1). The advantages of the temporary clip are:

- to produce an effective reduction of transmural pressure,
- to reduce the possibility on intraoperative rupture,
- to facilitate clipping,
- to reduce the need for controlled hypotension.

Fig. 1

The time in which temporary vascular ischemia changes to permanent focal cerebral infarct is not defined. Data indicate that temporary occlusion longer then 15 to 20 minutes may result in postoperative cerebral infarct ([143]). Other studies extend the time of vascular occlusion to 120 minutes ([144],[145],[146]). If the vascular occlusion affects the deep nucleus of the central nervous system it is recommended not to go beyond 10 minutes.

Temporary vascular occlusion is a technique initiated several years ago. The goal is to facilitate the surgical manipulation of the aneurysmto and to try to avoid complications that can result from controlled hypotension. It prevents ischemia of zones of cerebral tissue with a compromised circulation. The cerebral areas which depend on the occluded vessel may receive collateral circulation. Increase of mean arterial pressure may also be beneficial.

Risk factors predisposing to neurological deficit after the temporary occlusion are:

1. age (over 61 year old)
2. bad neurological condition before the intervention (degrees III and IV of Hunt and Hess)
3. distal and horizontal basilar segment of the medium cerebral artery (143).

INDUCED HYPERTENSION

Induced hypertension is used during surgery of intracranial aneurysms in order to increase the CBF in potentially ischemic cerebral zones. It is a complementary technique to the temporary vascular occlusion. Both techniques should be used simultaneously.

Cerebral ischemia disturbes autoregulation of CBF. The flow is then pressure dependent. Increase of perfusion pressure improves the CBF and helps to avoid cellular death due to improved collateral circulation. Induced hypertension is therefore used to reduce ischemia during the temporary vascular occlusion. It may also assist treatment of vasospasm.

The following drugs are commolnly used for induced hypertension: phenylephrine, norepinephrine and dopamine ([147],[148]). Phenylephrine is alpha-agonist and does not cause direct cerebral vasoconstriction. The increase of the CBF caused the phenylephrine is due to the increase of systemic blood pressure.

Inducted hypertension can produce edema and cerebral hemorrhage. In addition, it can increase the afterload and cause myocardial ischemia.

HYPOTHERMIA

Hypothermia is another technique used to protect the brain during surgery of intracranial aneurysms. Hypothermia produces a reduction in cerebral metabolism by decreasing cerebral electrical activity. The relationship between the degree of cerebral protection and hypothermia is not lineal. Moderate hypothermia also decreases the production and secretion of substrates associated with cellular damage such as aspartate and glutamate ([149]).

The effects of hypothermia are probably related to the reduction of the oxygen demand (from 7 to 8 % per 1º C), reduction of secretion of neurotransmitters glutamate and asparte (149) and increase in secretion of inhibitors (gamma-aminobutic acid).

Some studies investigating global and focal ischemia in animals demonstrated protective effects using moderate hypothermia (33º-35ºC). Some use moderate hypothermia during temporary vascular occlusion as another way of protecting the brain combined with induced hypertension.

Deep hypothermia (less than 20º C) allows to stop circulation during 60 minutes without neurological damage. Cardiopulmonary bypass is necessary for this technique. It is used in the operation of gigantic aneurysm ([150]).

The complication of deep hypothermia is intracranial bleeding due to coagulopathy. Other complications of hypothermia are cardiac dysrrhytmia, postoperative shiffering, increase of blood viscosity, coagulapathy, and increase in the rate of infections. Further studies are needed before recommending moderate hypothermia as routine technique ([151]).

PHARMACOLOGICAL CEREBRAL PROTECTION

It has been demonstrated in animal models that reduction of CBF under the critical level of 5 - 10 ml/min/100gr in 10 minutes can produce cellular death. No improvement has been demonstrated in some series performed in humans during intracranial aneurysm surgery in regard of morbidity and mortality ([152]). However, several studies demonstrated protective effects of thiopental in humans during cardiopulmonary bypass. Barbiturates have been widely analyzed for the protection of the brain during the aneurysm surgery.

High doses of barbiturates are needed to produce suppression of the cerebral electrical activity and reduction of cerebral metabolism. Such high doses produce a severe cardiovascular depression. This disadvantage should not cause any problems in patients with good cardiac function and normovolemia. However, the use of cerebral protection with barbiturates may be limited in patients with a reduced cardiac function .

An alternative to barbiturates is etomidate. It produces a reduction of cerebral metabolism similar to thiopental with little side effects on ventricular function ([153]). Etomidate can produce suppression of EEG such as thiopental. There is a report which indicates that etomidate provodes a significant cerebral protection during the temporary clipping (153) but the reported incidence of good results (71 %) is not appreciably different to the one reported by the Cooperative Study on the Timing of Aneurysm Surgery ([155]). Etomidate is routinely used in some centers for the temporary arterial occlusion of complex aneurysm.

Propofol produces a significant reduction of CBF, ICP and in a lesser degree of the CMRO2. However, its use as a cerebral protector is not recommended by several authors because its seizure-like activity after anesthesia. Propofol has a hemodynamic profile similar to the barbiturates and etomidate and like them it produces a suppression of the EEG. Animal studies have been confusing in demonstrating its cerebral protecting effects ([156],[157]). More extensive studies are needed regarding propofol before it can be recommended as a cerebral protector.

Isoflurane has been proved to have inferior effects compared to barbiturates ([158]).

PROBLEMS DURING SURGERY INTRAOPERATIVE RUPTURE

The rupture of an aneurysm can take place during induction or during surgery. The incidence of the intraoperative rupture is between 2 and 19%. The appearance of rupture during induction has devastating consequences. The appearance of hypertension with or without bradycardia will suggest the possibility of rupture of the aneurysm. Hemodynamic alterations can be slight in the anesthetized patient. The ultrasound transcranial doppler can be used in order to detect a rupture of an aneurysm after induction .

Should a rupture of an aneurysm occur, one will have to postpone surgery in order to evaluate the prognosis, the neurological state of the patient, and in order to control ICP. Therapy will go towards maintaining cerebral perfusion and controlling the ICP.

If the rupture of the aneurysm takes place during the craniotomy, priority will be to control hemorrhage. Induced hypotension should be applied quickly (average arterial pressure between 40 and 50mmHg or lower if necessary) iIn order to control hemorrhage . Induced hypotension reduces rebleeding and facilitates surgical exposure. Homeostasis can also be obtained by using a temporary or permanent clip. The degree of rebleeding in aneurysms of the anterior circulation can be reduced by manual compression the ipsilateral carotid artery for short periods of time. Induced hypotension for controlling bleeding is associated with worse neurological outcome than temporary clips.

Intraoperative rupture is associated with cerebral swelling, generally refractory to mannitol and corticosteroids. Rupture during induction is associated with higher mortality than during dissection of the aneurysm. Blood looses will be replaced with crystalloid and later, if necessary, with colloids or blood .

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