Anesthesia For Interventioanl Neuroradiology, Part IV: Intraoperative management, Anticoagulation, Management of neurologic complications, Conclusions

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Abstract

INTRAOPERATIVE MANAGEMENT

INTRAOPERATIVE MANAGEMENT OF VENTILATION

By changing the PaCO2, cerebral blood flow (CBF), cerebral blood volume (CBV), and intracranial pressure can be modified. A high PaCO2 causes cerebral vasodilation, increasing CBF and to a lesser extent CBV. Between a PaCO2 of 18 mmHG and a PaCO2 of 80 mm Hg, CBF varies directly with the PaCO2.

The lowest CBF that can be obtained by hyperventilation occurs at a PaCO2 of 18 - 20 mmHg.

In INR procedures, PaCO2 control has two objectives:

(1) to modify cerebral blood flow:

A decrease in cerebral blood flow is useful for imaging since contrast transit time is decreased, and better contrast visualization will result.

In embolization of intracerebral AVM's, lowering of the CBF can decrease shunt flow and allow the embolic liquid adhesive more time to polymerize inside the AVM, decreasing the possibility of distal embolization. If the neuroradiologist requests it, ventilation can be increased, preferably by increasing respiratory rate, until he or she is satisfied with the velocity of contrast flow, or until the PaCO2 is 20 to 25 mmHg. When controlled hypotension and hyperventilation are used concurrently, cerebral ischemia will occur at a higher level of blood pressure than it would if the PaCO2 was normal. Hyperventilation also augments the CBV- lowering effects of hypotension induced by b-blockers. Accordingly, we avoid using hyperventilation simultaneously with controlled hypotension, and maintain either normocapnia, or mild hyperventilation (PaCO2 30 to 35 mmHg) during controlled hypotension.

(2) to control intracranial pressure

In cases of cerebral edema or in the presence of mass lesions, hyperventilation is used to decrease the ICP. The PaCO2 should be maintained close to 20 mmHG to effect the maximum decrease in CBV. Hyperventilation, however, will not reduce the ICP if there is cerebral venous outflow obstruction. Thus, all constrictions around the neck, such as crossing EKG cables, or gowns, must be loosened.

If possible, the head of the bed should be raised in cases of elevated ICP to facilitate venous outflow.

All inhalation anesthesia agents, to different degrees, cause a dose-dependent increase in CBF; however, when hyperventilation is begun prior to the introduction of the inhaled anesthetic, and the end -tidal anesthetic concentration is kept close to 1 MAC, the ICP is not increased.

To manage ventilation, the PaCO2 can be obtained from a blood gas, or deduced from the PetCO2. Ventilatory adjustments should be made by changing respiratory rate, keeping tidal volume constant, because changes in tidal volume can alter physiologic dead space and change the PetCO2 - PaCO2 relationship. Increasing the ventilation by using large tidal volumes can also elevate intracerebral venous pressure. Most patients for INR are ventilated to a PaCO2 of around 35 mmHg. However, we change the level of ventilation in conjunction with blood pressure control to fit the clinical situation.

DELIBERATE HYPERCAPNIA

According to Young et al,(75) hypercapnia, to a PaCO2 of 50 to 60 mmHg has been used in cases of facial AVM's or dural fistulas to decrease the possibility of inadvertent intracerebral embolization when embolic agents are injected from the venous side into the malformation. The elevated PaCO2 increases CBF and cerebral venous outflow to a greater degree than extracranial venous outflow. Presumably, pressure gradients, clinically demonstrable but not measured, are created that impede extracranial to intracerebral flow.

INTRAOPERATIVE CONTROL OF BLOOD PRESSURE

The most important physiological goals in the management of arterial pressure during INR procedures are to maintain cerebral perfusion pressure, to prevent vessel rupture, and to avoid cerebral edema. A secondary goal, to assist the neuroradiologist to perform the procedure, is met by adjusting hemodynamics. In most INR procedures, a normal or high blood pressure is needed initially to assist in floating the superselective catheter to the lesion. On reaching the lesion, induced hypotension is used for embolization and then, postoperatively, the blood pressure is increased above hypotensive levels, but kept below control.

During the initial diagnostic angiography, the mean arterial pressure (MAP) is kept close to basal levels. Next, during the phase of superselective catheterization, the neuroradiologist often requests an increase in pressure to pre-induction or slightly higher than normal levels, in order to float the thin superselective catheter into place. It is usually not necessary to induce hypertension with vasoactive drips at this time. In our team's experience, all that is needed is a decrease in the depth of anesthesia, but rarely we have to use small boluses of ephedrine or neosynephrine.

Once the radiologist reaches the AVM or aneurysm, we begin controlled hypotension, to a MAP of 50 to 70 mmHg, and continue until embolization is complete. This is the most critical part of the procedure. During AVM embolization with liquid adhesive, we have been empirically using a Valsalva maneuver (to 20mm Hg) during, and for a few seconds after, adhesive injection into the AVM nidus. We believe that the increase in cerebral venous pressure produced by the Valsalva decreases AVM venous outflow, and lessens the amount of distal embolization of the adhesive. It is very critical that the patient does not "buck" during the Valsalva maneuver. Deepening the level of anesthesia may not be well tolerated during controlled hypotension; therefore, we recommend administering an additional dose of muscle relaxant prior to Valsalva in order to avoid movement or coughing during this crucial phase of the procedure. Although no research data is yet available, there is clinical angiographic evidence that a Valsalva maneuver with controlled hypotension, during injection of liquid adhesive, significantly decreases circulation time through AVMs,(76) and allows time for the adhesive to solidify without distal embolization.

After the embolization is complete, we allow the MAP to increase, but keep it 10 - 20 percent lower than basal levels during emergence and in the neurological intensive care unit.

INDUCED HYPOTENSION

We know that many interdependent variables affect autoregulation of cerebral blood flow; yet, clinically, the lowest level of MAP that can be safely induced is not known. In our practice, we use a MAP of 50 mm Hg as the minimum allowable pressure for short periods of induced hypotension. For patients with a history of hypertension, we empirically use a MAP of 70 mmHg as a minimum.

Many techniques are used to induce hypotension. The ideal agent would have a short onset and duration of action, be easily controlled and have no direct action on cerebral autoregulation. The principal agents used to induce hypotension are inhalation agents, b-blockers, vasodilators, ganglionic blocking agents, and calcium channel blocking agents.

Inhalation anesthetic agents are effective and titratable, but increase cerebral blood flow unless hyperventilation is initiated prior to their use. However, when used in combination with other drugs, inhalation agents can be safely used to induce hypotension without increases in ICP.(77)

Esmolol, a short acting b-blocker, does not increase CBF(78) and is effective in bolus doses of 0.5 to 1 mg/kg IV followed by an infusion.

It is our agent of choice for induced hypotension, and is very effective and easily controllable, especially when used in combination with inhalation agents.

Labetalol, a b-blocker with some -adrenergic antagonism is indicated at the end of the procedure in boluses of 10 - 20

mg IV to prevent postoperative hypertension.(79) .

All vasodilators can potentially increase ICP by increasing cerebral blood volume. The magnitude of ICP increase with vasodilators is related to the speed of onset of their effect.(80) Thus, they must be titrated slowly to minimize their effect on ICP. When vasodilators are used in combination with other classes of hypotensive agents, their total dose can be reduced and so can their effect on ICP.(81) Sodium nitroprusside, nitroglycerin, and hydralazine are commonly used. Sodium nitroprusside is a very potent, rapid-onset arterial vasodilator, administered by intravenous infusion, which controls the blood pressure rapidly and effectively. The main drawbacks of sodium nitroprusside are rebound hypertension and tachycardia, which occur unless b-blockers are given concurrently; and cyanide toxicity, which can appear with prolonged therapy. Nitroglycerin, a vasodilator acting principally on capacitance and coronary vessels, is not associated with tachycardia, rebound hypertension, or cyanide toxicity, but is not as effective as sodium nitroprusside. Like sodium nitroprusside, it must be given via a constant infusion, and requires close titration. Hydralazine, an arterial vasodilator, has an onset of 20-30 minutes and, unlike nitroglycerin or sodium nitroprusside, cannot be given by infusion. We do not use it as a single agent for controlled hypotension, but find it useful in the postoperative period, or as an intraoperative adjuvant drug in combination with b-blockers.

In our practice, we use vasodilators only when b-blockers are contraindicated or ineffective, usually in combination with inhalation agents.

Trimetaphan, a ganglionic blocking agent, does not cause an increase in CBV, but has been largely abandoned because it causes mydriasis, which can confuse the neurologic exam; and is associated with a high incidence of tachyphylaxis.

Nicardipine is a calcium channel blocker for intravenous administration by infusion. It has theoretical advantages when used as a hypotensive agent for INR. Although it can increase ICP,(82) it may provide cerebral protection in the event of cerebral ischemia,(83) produces significant decreases in systemic and cerebral vascular resistance with only mild tachycardia, increases coronary blood flow, and causes no myocardial depression.(84) It is administered by intravenous infusion at a rate of 5mg/hr - 50mg/hr. In INR, we have empirically used this drug in several patients, as the sole agent, and in combination with inhalation anesthetics, for induced hypotension and found it to be effective and easy to titrate.

TECHNIQUE OF INDUCED HYPOTENSION FOR INR

Prior to inducing hypotension in the intubated patient, we decrease the level of ventilation to reach a PetCO2 of 30 - 35 mmHg, since cerebral ischemia can result when hypotension is combined with hyperventilation. Once the PaCO2 level is stable, we begin by increasing the inspired concentration of inhaled anesthetic agent. If the end-tidal concentration reaches a level higher than 1 MAC (minimum alveolar concentration) for isoflurane or desflurane, we give a 0.5 - 1 mg/kg bolus of esmolol and start an infusion at 0.5 mg/kg/min while maintaining the inspired concentration of isoflurane or desflurane at 1 MAC or below. The esmolol infusion is then titrated to the desired pressure. In most patients, inhalation agents alone, or in combination with b-blockers, are all that is needed, but calcium channel blocking agents can be added or used alone if necessary. Patients under MAC will require significantly higher doses of b-blockers, vasodilators, or calcium channel blockers, to reach the same level of hypotension when compared to patients under general anesthesia.

When the interventional procedure is completed, the pressure is allowed to drift up by decreasing the dose of the inhalation agent and/or b-blocker. Towards the end of the procedure we administer intermittent boluses of labetalol 5-10 mg IV, or enalaprilat (an injectable converting enzyme inhibitor) 1.25 - 2.5 mg IV, to maintain the blood pressure empirically 10- 20 percent lower than control during the postoperative period. If a vasodilator infusion is being used, it is continued into the postoperative period, titrating the dose down and substituting it as soon as possible with longer- acting antihypertensive drugs.

INDUCED HYPERTENSION

In invasive neuroradiology, controlled hypertension is used in cases of iatrogenic vascular occlusion and in cases of acute thromboembolic stroke. The goal is to increase cerebral perfusion pressure to ischemic areas via collateral circulation through the circle of Willis and external carotid to internal carotid communicators.

If a cerebrovascular occlusion occurs during an INR procedure, induced hypertension may reverse or prevent a neurologic deficit. Several drugs can be used to elevate the blood pressure. Young et al (85) suggest a phenylephrine

drip as a first line agent. It is titrated to empirically increase the pressure 30-40 percent above the baseline,(86) until the neurologic deficit is reversed. Phenylephrine is a pure a-1 agonist vasoconstrictor and causes no direct cardiac stimulation, but can cause coronary artery vasoconstriction and reflex bradycardia. Norepinephrine, a potent inotrope and vasoconstrictor, also causes reflex bradycardia, but is more potent and has a shorter duration of action than neosynephrine, and for this reason is our drug of choice in this situation. If the patient already has bradycardia, dopamine can added to phenylephrine or norepinephrine. If dopamine is used alone, tachycardia often becomes a difficult clinical problem.

Some patients with acute thromboembolic stroke are candidates for intracerebral arterial thrombolysis.(87) They are usually hypertensive, and often have coronary artery disease. An increase in afterload or tachycardia during induced hypertension, places those patients with coronary artery disease at risk for developing coronary ischemia or pulmonary edema and may limit the level of hypertension that is safely tolerated. Monitoring of the ECG for ischemia with ST segment trending, if available, is particularly important in these cases. A pulmonary artery catheter needs to be considered, especially in those with a history of poor myocardial performance.

ANTICOAGULATION

Because of the inherently thrombogenic nature of the embolic materials and catheters, the neuroradiologist often requests systemic heparinization in high risk INR procedures to prevent the formation and propagation of intravascular thrombi. Often, heparinization is continued into the postoperative period, leaving the femoral arterial introducer sheath in place until the heparin is reversed.

The specific indications and duration of heparinization are still controversial, although most centers heparinize when performing temporary balloon occlusion, or superselective catheterization.(88) Heparin can be administered either by intermittent IV boluses or by a bolus followed by a continuous infusion. In addition, heparin is used by the radiologist in the flush solution for the coaxial femoral arterial catheter system.

The insertion of all invasive monitoring should be carried out before heparinization, and a control activated clotting time (ACT) obtained. Care should be taken that no residual heparin from the flush solution of the arterial line contaminates the ACT sample. Heparin can be given on a fixed dose, or titrated using a heparin dose-response curve as described by Bull et al.(89) Since there is individual variation in pharmacokinetics and patient sensitivity to heparin, individual titration and a low initial dose are clearly of value.

Heparin dose is expressed either in milligrams or in international units. An international unit of heparin is defined as that amount required to prolong the coagulation of 1 ml of whole blood for 3 minutes. Since heparin vials are labeled in units per ml, heparin doses should be expressed in units, not milligrams, to avoid confusion.

HEPARINIZATION PROTOCOL FOR INR AT THE METHODIST HOSPITAL

1. Obtain baseline ACT
2. At radiologist's request, administer heparin, 50 units/kg IV push or 0.5 mg/Kg (1mg = 100 units)
3. Begin infusion at a rate of 50 units/kg/hr at radiologist's request
4. After 5 minutes, obtain ACT and construct dose-response curve
5. ACT goal: 2-3 times control
6. Monitor ACT every hour
7. If ACT is < 2 times control, consult radiologist. If continued heparinization is needed, give bolus dose per dose-response curve.
8. Keep protamine, 50 mg, ready for emergency administration
9. For reversal of heparin anticoagulation use protamine, 1 mg per 100 units of heparin, or dose according to heparin dose-response curve.

MANAGEMENT OF NEUROLOGIC COMPLICATIONS (90)

The neurologic complications of INR are an anesthetic emergency. The management of these is similar to that of intraoperative complications in open neurosurgical procedures. There are two main types of immediate vascular complications. One is a vascular occlusion, due to emboli, thrombosis, arterial dissection, or vasospasm, that can lead to brain ischemia or infarction. The second is a vascular

perforation leading to cerebral or subarachnoid hemorrhage. Primary management in either case consists of obtaining control of the airway with endotracheal intubation. Etomidate is our induction agent of choice for intubation since it does not cause cardiovascular depression and may protect the brain.(91) Thiopental is used if the patient is hypertensive and cerebral hemorrhage is suspected. Initially, the most important decision is to differentiate between an occlusive and a hemorrhagic event since the specific treatment for each is different. When a complication occurs, the radiologist should immediately report to the anesthesiologist which one is suspected so that the appropriate treatment can be started immediately.

If the complication is a hemorrhage, the goal is to increase coagulability and decrease bleeding. The initial signs of perforation are angiographic; in the awake patient, the symptoms are similar to those of spontaneous subarachnoid hemorrhage. Heparin is reversed with protamine, in a ratio of 1 mg of protamine to each 100 U of heparin, and monitored with an ACT. Thiopental, in boluses of 100 to 200 mg, is used at this time to decrease the blood pressure and initiate cerebral protection.(92) The blood pressure is kept low, using b-blockers or thiopental, while the radiologist gains control of the bleeding with a balloon, glue, or coils. When control of the bleeding is obtained, the pressure is raised to test for leakage and to maintain cerebral perfusion pressure.

If the problem is an occlusion, the goal is to raise the mean arterial pressure (MAP) with controlled hypertension to increase collateral flow. Thrombolytic therapy may also be indicated in some patients.(93) When this is contemplated, the indications for invasive procedures that could cause bleeding, such as insertion of central lines, pulmonary artery catheters or arterial lines, should be carefully considered.

Further measures include those used to treat brain edema and minimize ischemic insult, such as elevation of the head, hyperventilation, anticonvulsants, and mannitol. To provide some cerebral protection, the patient's temperature is allowed to drift down to 33-35 degrees centigrade, and the blood sugar kept between 150-200 mg percent. In cases of severe brain edema, with a Glasgow coma scale of 9 or less, a pulmonary artery catheter and an intracranial pressure monitor are used, especially if a barbiturate infusion is contemplated.

CONCLUSIONS

Interventional neuroradiology procedures pose many new and interesting challenges for the anesthesiologist. These procedures can provide definitive treatment of some types of cerebral aneurysms, arterio-venous malformations, or fistulae, and provide complementary therapies such as pre-operative embolization of highly vascular tumors, or intraarterial chemotherapy. They are not a variation of a diagnostic cerebral angiogram, but are complex and involved procedures with potential for severe neurologic injury. We consider these procedures "closed head neurosurgery", and as such, we feel that coordination between neuroradiologist and anesthesiologist is essential to safely carry them out. An interventional neuroradiology team should include an interested and expert anesthesiologist who is thoroughly familiar with invasive neuroradiologic procedures, techniques of controlled hypotension, deliberate hypertension, treatment of cerebrovascular complications and management of elevated intracranial pressure.

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