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Controlled hypotension for spinal surgery

Received: 28 April 2004
Accepted: 7 May 2004
Published online: 9 June 2004
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Abstract Controlled, deliberate hypotension during anesthesia for major spinal surgery reduces intraoperative blood loss and transfusion requirement. Hypotension may be achieved with increased doses of volatile anesthetic agents or by continuous infusion of vasodilating drugs. Safe application of this technique requires knowledge of the physiology of hemorrhagic shock and close intraoperative monitoring to avoid vasoconstriction and end-organ ischemia.

Keywords Spinal surgery · Deliberate hypotension · Posterior ischemic optic neuropathy · Review

Introduction

Controlled, or deliberate, hypotension has been used for many years as a means of reducing intraoperative blood loss and facilitating surgical exposure. Reduced intraoperative blood pressure leads to a direct reduction in bleeding from surgically injured arteries and arterioles. Venous dilation, in turn, decreases venous bleeding, especially from cancellous bony sinuses that do not collapse when transected. Decreased bleeding improves surgical visualization of the wound, resulting in faster surgeries (in some series) and, thus, further reducing transfusion dependence. Anecdotal reports describing this technique have been published since the 1970s [14, 18], and there have been a number of prospective trials demonstrating the efficacy of deliberate hypotension, alone or in combination with other techniques, at reducing the blood loss and transfusion requirement of major spinal surgery [12, 13, 14, 15, 16, 17, 18, 19, 20].

Because of its simplicity, deliberate hypotension is a widely used technique. The physiology of hemorrhage is complex, however, and the potential consequences of this

approach are not always readily apparent. The ischemic threshold for individual patients (and individual organ systems) cannot be defined prospectively and is not indicated by any existing operating room (OR) monitor. Further, differences in tissue perfusion that are related to different methods of achieving hypotension (anesthetic agents vs pure vasodilators, for example) are not fully understood. Recently published cases describing the occurrence of blindness after spinal surgery have highlighted the potential risks of deliberate hypotension. These have led to renewed interest in determining how broadly this approach can be applied, and what levels of hypotension over what periods of time will be safe for the majority of patients.

Physiology of hemorrhage

Loss of circulating blood volume produces a cascade of physiologic effects intended to preserve perfusion of the heart and brain at the expense of more peripheral organs. Shock is the clinical term for the physiologic disease of hypoperfusion. It is a common finding in patients with on-

going hemorrhage. In the un-anesthetized individual, hemorrhage leads directly to vasoconstriction, with blood pressure falling little until the limits of compensation are exceeded (usually at a total blood loss of 30–40%) [7]. Cardiac output is maintained initially by increased heart rate and contractility, but this may fall substantially before a change in blood pressure occurs. While severe hemorrhage is fatal through the obvious mechanism of decreased cerebral perfusion, lesser degrees of blood loss may still cause death through the accumulated effects of end-organ hypoperfusion, manifesting as the syndrome of multiple-organ-system failure. The “dose” of sub-acute shock is defined by the degree of organ-system hypoperfusion and the length of time it is sustained. Fatal sub-acute shock occurs as the result of too great a failure of peripheral oxygen delivery. Because blood pressure may be sustained by vasoconstriction, even in the face of severe fluid volume loss, this marker in isolation is not a good indicator of the presence or absence of shock and the potential for ischemic injury [5].

Anesthetic agents, particularly induction drugs and volatile gases, interfere with the normal response to hemorrhage. As direct vasodilators, they prevent or reverse compensatory vasoconstriction. Anesthetic agents also inhibit sympathetically mediated increases in heart rate and contractility. This effect makes accurate assessment of the patient’s fluid-volume status both easier and harder. On the one hand, anesthetic inhibition of vasoconstriction will create a more direct link between central filling pressures and blood pressure, meaning that blood loss will more rapidly produce hypotension than in the awake patient with active compensatory mechanisms. On the other hand, decreases in blood pressure that result from decreased intravascular volume may be erroneously attributed to the effects of anesthetic agents, delaying the recognition and treatment of hypovolemia. Hypotension in the operating room must therefore be assessed in terms of both fluid-volume status and anesthetic depth. The patient who is hypotensive primarily from blood loss is very different physiologically from one who is hypotensive due to anesthetic overdose [9]. The former patient is vasoconstricted, with low cardiac output and low blood flow. This patient is at much greater risk for ischemic complications than the patient who is hypotensive due to vasodilatation, who is in a high-flow state with preservation of peripheral oxygen delivery. Blood loss may also be affected by vasomotor tone, independent of blood pressure. Techniques that produce vasodilatation, such as epidural anesthesia, have been reported to reduce blood loss during orthopedic operations, even when normotension is maintained [21]. The goal of minimizing surgical hemorrhage by keeping the blood pressure low is, therefore, best achieved by the use of agents that produce hypotension through vasodilatation in the presence of adequate intravascular volume, rather than through vasoconstriction due to under-replacement of fluid-volume losses.

Monitoring

The anesthesiologist intending to induce and maintain deliberate hypotension during major spinal surgery must obviously observe the blood pressure closely. An arterial catheter and continuous pressure-monitoring system is strongly recommended; this will also allow for frequent blood sampling for laboratory study. The radial artery is the most common site for arterial line placement, because it is technically easy to access, away from the site of surgery and not the sole blood vessel supplying the hand. Femoral artery cannulation is also possible, but the anesthesiologist must take great care that the line remains patent and functional during prone positioning of the patient for surgery. This is also a concern with radial lines if the arms are to be tucked alongside the patient, as for cervical or high-thoracic procedures. Arterial line placement can occur preoperatively or following induction of anesthesia, depending on the need for close pressure monitoring prior to surgery.

As important to deliberate hypotension as close observation of the blood pressure is an accurate understanding of the patient’s fluid-volume status and degree of vasoconstriction. While no single monitor can indicate this with complete reliability, the experienced anesthesiologist will make this assessment based on the integration of several different pieces of data. First is the arithmetic calculation of “ins and outs,” the kind and amount of fluid administered and the estimated fluid lost or consumed, including hemorrhage volume, metabolic maintenance, and insensible losses to interstitial edema and atmospheric evaporation. This is an important exercise to perform, particularly for long cases, but is dependent on both visual estimates (blood loss) and crude approximations (interstitial losses). Even when meticulously calculated, paper estimates of fluid requirements often woefully underestimate actual need.

The second critical piece of data is the patient’s anesthetic requirement: the total dose of narcotic, volatile, and sedative medication maintaining the anesthetized state. It is seductively easy for the inexperienced practitioner to incrementally reduce the anesthetic dose in response to decreases in blood pressure caused by hypovolemia, particularly in the presence of muscle relaxants that prevent patient movement. This runs the obvious risk of allowing intraoperative patient awareness. Even more insidious, however, this may move the patient to a physiologic state of occult shock, in which severe hypovolemia is being compensated for by catecholamine production and peripheral vasoconstriction. As was indicated above, deliberate hypotension achieved by volume contraction and vasoconstriction puts the patient at higher risk for ischemic complications. The patient’s requirement for anesthesia can be established during the early stages of the procedure, prior to significant fluid volume shifts, providing a benchmark for future reference. It is unlikely in any pa-

tient that the appropriate dose of anesthetic agents to balance a given surgical stimulus will be different at the end of the case than it was at the beginning.

A third measure of intravascular volume is the response to fluid administration indicated by direct monitors of central pressure. Central venous pressure (CVP) and pulmonary-artery pressure values in isolation may be difficult to interpret, due to the effects of positive pressure ventilation, prone positioning and anesthetic technique, but serial values over time may be of value. This is particularly true when pressure changes are interpreted following maneuvers likely to change filling volume (increases or decreases in anesthetic depth, fluid administration, bleeding, use of vasoactive agents). If CVP falls during the case, compared to a baseline value established following induction and positioning but prior to hemorrhage, then the patient is most likely hypovolemic. If a fluid bolus increases central pressures but not cardiac output, then the patient is adequately volume replaced. Most commonly, however, fluid administration will increase cardiac output without a significant increase in pressures. This patient has "recruitable perfusion" and – all things being equal – should receive more fluid. In patients with limited cardiac function, an additional variable is added to the equation, and the risk of lowering perfusion through fluid overload and cardiac dysfunction is introduced. Frequent measurement of cardiac output is necessary to guide fluid therapy in these patients, or continuous direct observation of cardiac filling and contractility via trans-esophageal echocardiography.

A fourth way to examine fluid volume is through laboratory assessment of blood chemistry, which should be performed as often as necessary to confirm the adequacy of perfusion. Inappropriately high hematocrit or serum osmolality indicate hypovolemia, while low hematocrit or elevated coagulation times indicate the need for specific blood products. Acute hypoperfusion will be indicated by development of metabolic acidosis, reflected in the pH and base deficit of the arterial blood gas. Hypoperfusion occurring over a period of time will cause a rise in serum lactate, which correlates well with morbidity and mortality due to hemorrhagic shock [1]. Any evidence of systemic acidosis indicates the need for fluid volume administration to improve tissue perfusion, regardless of whether the blood pressure is low or normal. Elevated lactate, indicating an ongoing "oxygen debt," should be regarded as an extremely serious sign in any elective surgical case, and it should mandate immediate correction of the underlying cause and resuscitation of the patient.

Finally, the anesthesiologist must be attentive to monitors of individual organ-system perfusion. Cardiac dysrhythmias and ST-segment changes have been reported as indicators of ischemia during deliberate hypotensive anesthesia in an animal model [11]. Changes in nerve conduction amplitude and latency are monitored during spinal surgery as indicators of procedure-specific over-distrac-

tion of the spinal cord, but global changes may be a warning sign of systemic hypoperfusion. Decreases in urine output indicate decreased renal perfusion, while failure of the pulse oximeter may indicate progressive systemic vasoconstriction. Mixed venous oxygen saturation is a very sensitive marker for the adequacy of perfusion, but is seldom available in the operating room. A fall in end-tidal carbon dioxide concentration is a late indicator of severe hypoperfusion, most likely to occur just moments before complete cardiovascular failure.

Pharmacology

Understanding the physiology of hemorrhage and the necessary monitoring to induce and maintain hypotension safely will allow the experienced provider to apply this technique to most spinal surgery patients. The specific agents used to induce hypotension are almost a secondary consideration, but there have been numerous publications examining the options and attempting to define a difference between the choices. The first level of discrimination is between, on the one hand, those agents that are primarily anesthetics but are capable of producing hypotension, and, on the other hand, those agents that are purely vasoactive.

Anesthetic agents used to induce deliberate hypotension in current practice include the volatile gases (isoflurane, desflurane and sevoflurane) and intravenous sedative medications (thiopental and propofol). For pelvic and lower extremity orthopedic surgery, epidural administration of local anesthetics is a common choice. While adjuvant epidural anesthesia has been used in the past for major spinal surgery, this technique is uncommon today, both because of the technical challenges involved and because of its interference with neuromonitoring and postoperative neurologic assessment. Interference with the ability to measure and compare somatosensory or motor evoked potentials is the principal limitation to using any anesthetic agents to produce hypotension. Pentothol, in fact, is commonly used in patients undergoing complete circulatory arrest or with severe traumatic brain injury as a means of reducing neurologic activity to the lowest possible level [23]. Further, both pentothol and propofol reduce blood pressure in large part through a direct, negative inotropic effect on the heart, which may be less desirable physiologically. The volatile gases produce hypotension mostly through vasodilatation [16], and thus may be more effective at reducing intraoperative blood loss [26]. There is little to discriminate among the different volatile agents in terms of efficacy, although desflurane and sevoflurane may be easier to titrate, due to their faster onset and elimination [2]. Isoflurane begins to depress evoked potentials at a concentration of about 1% (1 MAC) in most patients, but there is substantial variability in this value [25]. Use of volatile gases to produce deliberate hypotension can be

facilitated by the administration of intravenous narcotics to blunt the patient's sympathetic response. Although narcotics have no direct cardiovascular effect and will not produce hypotension as the sole agent in a euvoletic patient, they will potentiate the effects of other anesthetics, allowing a lower blood pressure to be achieved for a given concentration. Deliberate hypotension for spinal surgery is, thus, most commonly achieved by the use of increased concentrations of a volatile agent (typically isoflurane) on top of a balanced general anesthetic.

When higher concentrations of volatile agent are contraindicated, either due to interference with neuromonitoring or to some other patient characteristic, there are a number of other agents that can be used to induce and maintain hypotension. Examples include sodium nitroprusside (SNP), nitroglycerin, trimetaphan, esmolol, nicardipine, and fenoldopam. In the presence of an adequate general anesthetic, any of these agents offers the ability to titrate the patient's blood pressure to any desired level of hypotension, maintain it that way throughout the surgery, and allow it to rapidly return to normal at the end of hemorrhage. Historically, SNP has been the model for direct vasodilatation, but concerns over cyanide toxicity resulting from long periods of administration led first to its combination with the ganglionic-blocking agent trimetaphan [19], and more recently to its abandonment in favor of newer agents. Esmolol (a rapid-acting, beta-blocking agent) and nicardipine (a rapid-acting, calcium channel-blocking agent) have both been used by continuous infusion to maintain hypotension [3]. Each acts through a combination of vasodilatation and negative inotropy. Recent literature examining regional blood flow has suggested that the physiology of hypotension induced by nicardipine may be different than that produced by volatile anesthetics. This implies a variable response on the microcirculatory level [17]. Fenoldopam is a dopaminergic agonist that could in theory preserve splanchnic and renal blood flow during deliberate hypotension, but experience with this agent is still limited [15, 27].

Risks

While deliberate hypotensive anesthesia has a long record of safety, there are a few potential risks that the anesthesiologist must be aware of. Hypotension makes the patient more susceptible to cardiac arrest if a sudden surgical catastrophe, such as massive hemorrhage or tension pneumothorax, occurs. More worrisome for the anesthesiologist is the risk of an ischemic complication that occurs despite the successful application of the planned technique. Because the ischemic threshold of individual organs is impossible to estimate, and because monitoring of perfusion is indirect at best, there have been occasional case reports of complications noted following uneventful anesthetics [22].

New onset or worsening of neurologic deficit below the level of surgery may result from direct injury or overdistraction of the spinal cord, hypoperfusion, or a combination of the two. Continuous electrophysiologic monitoring of either the anterior spinal cord (motor evoked potentials) or posterior cord (somatosensory evoked potentials) is the standard of care for most complex spinal surgeries, and is a sensitive monitor of physiology. Baseline values are obtained prior to anesthetic induction and following surgical positioning. Increase in latency or amplitude of electrical response from baseline should be promptly investigated, and the cause corrected if possible. Electrical evidence of decreased spinal cord function should lead the provider to abandon the hypotensive technique, accepting the potential for increased hemorrhage in exchange for maximizing perfusion.

Myocardial ischemia or infarct is rare following hypotensive anesthesia, and is usually the result of unrecognized hypovolemia and vasoconstriction, anemia, occult coronary disease, or a combination. Sudden desaturation has been described during deliberate hypotension [4], which may put vulnerable patients at risk. Risk factors for decreased myocardial reserve, such as advanced age, diabetes, atherosclerosis, or resting hypertension, are all relative contraindications to deliberate hypotension. These patients may already have flow-limited myocardial perfusion, as well as altered autoregulatory thresholds in other organ systems.

There have been a number of reports describing the development of unilateral or complete visual loss following spinal surgery in the prone position, including a recent presentation by the Anesthesia Closed Claims Project of the American society of Anesthesiologists [8, 13, 22, 28]. Originally thought to be due to pressure on the eye due to careless prone positioning (anterior ischemic optic neuropathy, AION) [25], most cases of blindness are now recognized as the result of posterior ischemia (PION), due to hypoperfusion of the optic nerve [8, 13, 22]. Analysis of published case reports and closed claims suggests that risk factors for this disastrous complication include prolonged surgery, hypotension, and anemia, often in combination. The development of a vasoconstricted – shock – state due to unrecognized hypovolemia is an important risk factor, emphasizing the importance of monitoring tissue perfusion and not just blood pressure when employing a deliberate hypotensive technique.

Summary

The author's recommendations for the conduct of deliberate hypotensive anesthesia are shown in Table 1. While deliberate hypotension has been repeatedly shown to reduce transfusion requirement during major spinal surgery, alone or in combination with hemodilution, cell salvage, erythropoietin administration and other techniques, the risks

Table 1 Recommendations for deliberate hypotensive anesthesia (*CVP* central venous pressure, *SSEP* somatosensory evoked potentials, *PA* pulmonary artery)

Patient selection	Patients scheduled for multilevel or complex spinal surgery with an anticipated risk of homologous blood transfusion
Contraindications	Patients with altered baseline autoregulatory mechanisms (hypertension) or those likely to be vulnerable to ischemic complications (diabetes, coronary artery disease, stroke, chronic renal failure, etc.)
Monitoring	Standard monitors, plus continuous arterial pressure catheter, neurophysiologic monitor (SSEP or motor evoked potential), and monitor of intravascular volume (CVP or PA catheter). Frequent laboratory assay is indicated to monitor hematocrit, coagulation factors, and markers of tissue hypoperfusion (base deficit and lactate)
Positioning	Careful attention to all pressure points, with special attention to the face and eyes. If the arms are to be tucked out of sight, access lines and monitors must be carefully secured
Anesthesia	Balanced general anesthesia using narcotics and inhaled volatile agents. The patient's anesthetic requirement should be established prior to the induction of deliberate hypotension, and used as a baseline for subsequent judgment of volume status
Technique	During surgical dissection and implantation of hardware, maintain systolic blood pressure 20–30% below baseline (80–90 mmHg in normal patients) using (1) an increased concentration of volatile gas or (2) continuous infusion of a rapid-acting vasodilator
Cautions	Deliberate hypotension should be abandoned in the presence of changes in nerve conduction signals, decreased urine output, EKG changes, anemia (HCT <20%) or tissue acidosis

are still difficult to quantify. There are some patient groups, such as Jehovah's Witnesses, in which it may be life-saving [6]. Direct assessment of microvascular perfusion in individual organ systems is not possible, making any decision to deliberately induce hypotension a judgment between the small but serious risks of ischemic complications such as PION or MI and the larger but less severe risks of transfusion. Based on current literature and clinical

practice, moderate degrees of hypotension (80–90 mmHg systolic) are efficacious at reducing blood loss, and safe in any patient without specific risk factors. When employing a deliberate hypotensive technique, the anesthesiologist must be mindful of the physiology of hemorrhage and shock, attentive to direct and indirect markers for hypoperfusion, and responsive to ischemic risk factors such as prolongation of surgery, sudden blood loss, and anemia.

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