Special Techniques and Concepts in Anesthesia

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ANESTHESIA AND THE PREGNANT PATIENT

Postpone all elective surgery until 6 weeks following delivery. The risks of teratogenesis and/or preterm labor are too high to consider elective surgery prior to delivery.

Depending on the severity or emergent nature of an operative procedure, fetal and uterine monitoring may be warranted (e.g., in women who are more than 16 weeks pregnant) throughout the surgery, with plans and preparation for an emergency cesarean section (C-section) if necessary.

Later in pregnancy, the goal is to prevent fetal asphyxia by maintaining a maximum delivery of oxygen to the mother and, therefore, the fetus via the placenta. Maintain maternal blood pressure to ensure adequate placental perfusion. In evaluating laboratory values, the total blood volume will be increased; therefore, one would expect a reduction in hemoglobin and hematocrit values. Blood gases normally show a respiratory acidosis.

Use left uterine displacement (LUD) to prevent compression on the aorta when the mother is in the supine position (in women who are more than 16–20 weeks pregnant).

Tocolytics are drugs that suppress onset of premature labor and should be available when giving anesthesia on any pregnant patient. They include the following medications:

- Magnesium sulfate: first-line agent; calcium antagonist.
- Ritodrine: beta-2 agonist; decreases levels of free calcium
- Terbutaline: beta-2 agonist
- Calcium-channel blockers: nifedipine, verapamil

Anesthetics in Pregnancy

General anesthesia helps to block uterine contractions. Use lower doses of all medications. Short-acting agents are preferred and minimize overall exposure to all anesthetic agents.

Narcotics cross the placental barrier and affect the fetus.

Muscle relaxants do *not* cross the placental barrier as readily as narcotics.

Avoid benzodiazepines (owing to the increased incidence of deformities in the fetus) and nitrous oxide in any pregnant patient.

Regional anesthesia is preferred because it minimizes fetal exposure to the anesthetic agents and reduces the risk of maternal aspiration.

AORTIC CROSS-CLAMPING, SPINAL CORD ISSUES, AND AORTIC SHUNTING

Application and Withdrawal of an Aortic Cross-Clamp

The higher the cross-clamp on the aorta (at the suprarenal or supra-celiac levels), the more severe the effects. Fewer hemodynamic changes occur when cross-clamping the infra-renal aorta.

Prior to Clamp Placement

The systolic blood pressure (SBP) should be 90 mm Hg prior to clamp placement. Minimize the effects of clamping with nitroprusside, nitroglycerin, betablockers, fenoldopam, nicardipine, and/or inhalational agents.

After Clamp Is Applied

Marked hypertension occurs in the proximal aortic segment (above the clamp).

- MAP increases by 40%.
- Acute elevations in left ventricular pressure occur.
- Cardiac output is decreased.
- CVP increases by approximately 4 mm Hg.
- Left atrial pressure (LAP) and pulmonary capillary wedge pressure (PCWP) increase 12 mm Hg or more.
- Coronary blood flow increases by 40%.
- Systemic vascular resistance (SVR) increases by 100%.
- Levels of catecholamines, renin, and angiotensin increase, leading to vasoconstriction.

Hypotension occurs in the distal aortic segment (below the clamp), with MAP decreasing by 15%. Spinal blood flow decreases:

Proximal and distal clamp placement to isolate the diseased aortic segment may include critical intercostal vessels that provide flow to the cord; this loss is not compensated for by distal perfusion. The following measures are directed at protecting the spinal cord when the cross-clamp is on:

- Maintain MAP at 40–60 mm Hg.
- Use somatosensory evoked potentials (SSEPs) to monitor dorsal column function (sensory tracts). *Anterior cord function (i.e., motor tracts) is not monitored with SSEP*.
- Motor evoked potentials (MEPs) can accurately monitor anterior horn function. However, *muscle relaxants cannot be given* when monitoring MEPs.
- Hypothermia (decreasing core temperature to 33–34°C) will lower the patient's metabolic rate. It can be accomplished with application of ice and administration of cold blood. Care must be taken, as the myocardium becomes irritable at 32°C.
- Spinal drains can be used to relieve pressure. The pressure exerted by the cerebrospinal fluid (CSF) increases during cross-clamping.
- Give steroids to further protect the spinal cord.

Renal and intestinal blood flow decreases:

To prevent renal failure and gut ischemia, keep the patient's BP up with good perfusion. Give Mannitol (0.5 mg/kg), furosemide, and IV infusion of low-dose dopamine or fenoldopam for renal protection. Even if aortic shunting is used, the distribution changes in renal blood flow make these interventions prudent.

Before Clamp Is Removed

Check the volume load. The patient may need vasopressors prior to removal of the cross-clamp.

Aortic Cross-Clamp Removal Effects

Declamping shock is a possibility. Severe hypotension—a decline in BP of as much as 70%—may occur due to hypovolemia (combined with bleeding), with this abrupt decrease in after load. The release of vasodilating acid metabolites from the ischemic lower body into the general circulation (noted as an increase in $ETCO_2$), the release of vasodilator substances, and an increase in the vascular space all cause a severe decrease in ventricular preload. Declamping shock is also associated with hyperkalemia and hypocalcemia.

Removal of the cross-clamp may also lead to decreased contractility and cardiac output. The severity of this effect is influenced by the duration of clamping, existence of adequate preload, and influence of circulating drugs.

Treat cross-clamp removal effects by decreasing the anesthetic levels, decreasing or discontinuing vasodilators, and administering volume (crystalloids, colloids, cell saver, blood products), and calcium chloride. May also need to give phenylephrine, epinephrine, or norepinephrine to maintain the BP. Increasing the ventilation rate will help decrease acidosis, though it may be necessary to give sodium bicarbonate as well. The surgeon may need to remove the cross-clamp slowly to minimize these effects.

Renal blood flow decreases by 50% when the cross-clamp is removed. To compensate for this effect, keep the patient hydrated and keep the BP within normal limits.

- The spinal cord must also be protected from severe hypotension *after* removing the aortic cross-clamp by maintaining the BP within normal limits.

SPINAL CORD INJURY AND AORTIC CROSS-CLAMPING

Spinal cord perfusion in the thoracolumbar area is derived from the artery of Adamkiewicz (the principal arterial supply of the anterior spinal cord). The artery of Adamkiewicz joins the anterior spinal artery in sending flow to the lower thoracic and lumbar segments T8–L4: the celiac artery at T12 (gastric, splenic, hepatic branches), the superior mesenteric artery at L1, and the inferior mesenteric artery at L3. The lateral aortic branches are the suprarenal and renal branches at L1 and the gonadal branchat L2. The posterolateral artery contains the inferior phrenic and lumbar branches. Maintaining perfusion in these areas can be important, particularly in thoracoabdominal aneurysm repair, where the cross-clamp is applied above these structures.

The most feared major complication of aortic cross-clamping is paraplegia from prolonged spinal cord ischemia as a consequence of hypotension and surgical interruption of the blood supply to the artery of Adamkiewicz. Along with paraplegia, there is a risk of mesenteric/bowel ischemia/infarction, renal ischemia/failure, and hepatic ischemia. Coagulopathy can occur if thoracolumbar blood flow is decreased when systemic pressures are low. Maintaining a distal perfusion pressure mean of 70 mm Hg or higher will minimize the incidence of paraplegia and organ ischemia/failure.

As far as aortic cross-clamp time goes, a simple rule applies: Shorter is better. The duration of aortic cross-clamping is directly related to the risk of complications; cross-clamping the aorta for more than 30 minutes increases their incidence.

Spinal Cord Protection During Aortic Cross-Clamping

Protective measures during aortic cross-clamping aim to stabilize cell membranes, prevent release of chemical mediators, and scavenge oxygen-free radicals. These measures are summarized here:

- Thiopental IV
- Intrathecal Papaverine (3 mL of 1% strength)
- Corticosteroids (methylprednisone)
- Magnesium sulfate
- Mannitol
- Betablockers

- Drain 20–25 mL CSF prior to clamping to decrease intrathecal pressure.
- Maintain MAP at greater than 70 mm Hg intraoperatively and postoperatively.

The MAP distal to the aortic clamp is decreased and the distal spinal cord will be at risk of ischemia. Several techniques can increase the distal arterial perfusion pressure, such as the placement of a simple shunt (a shunt is placed above and below the cross-clamp) or a partial (femoral vein to femoral artery) or full cardiopulmonary bypass (left atrium to femoral artery). Heparin is given with these techniques.

Administration of nitroprusside can be detrimental to spinal cord perfusion by decreasing systemic vascular resistance and shunting blood away from the spinal cord vessels and collateral branches.

Cross-clamping the aorta below the left common carotid increases the proximal systemic pressure, which in turn increases the CSF pressure. This increased CSF pressure can increase the incidence of paraplegia and careful drainage of CSF can be beneficial.

Spinal cord perfusion pressure (SCPP) = anterior spinal artery pressure (distal aortic MAP) − CSF pressure

To increase SCPP, either increase the distal aortic MAP or lower the CSF pressure. SCPP should be maintained at a level higher than 15 mm Hg.

Hypothermia at 32–24°C decreases spinal cord oxygen requirement, decreases tissue metabolism, and increases tolerance to anoxia.

It is important to prevent hyperglycemia when aortic cross-clamping is used. Maintain blood glucose levels between 80 and 120 mg/dL.

Prolonged latency or reduced amplitude of SSEPs and MEPs indicates spinal cord ischemia, although this relationship is not always reliable. SSEPs monitor sensory neurons in dorsal root ganglia to the posterior column; MEPs monitor motor neurons in the anterior horn cells and is more sensitive to ischemia.

Aortic Shunt

Placement of a shunt may be used to bypass the cross-clamped aorta. In this case, the proximal end of the shunt is placed in the ascending aorta and the distal end in the descending thoracic aorta past the aneurysm. Shunt flow should be approximately 2.5 L/min, with distal MAP being maintained at more than 70 mm Hg.

Nevertheless, the spinal cord and kidneys cannot be assumed to be "protected" when a shunt or bypass is used. Even with these measures, atherosclerosis may prevent significant flow to the kidneys and spinal cord.

Distal shunt placement advantages include the ability to attenuate proximal hypertension by sending blood down past the clamps via the shunt. This approach may lead to perfusion of the vascular beds distal to clamp. The placement of such a shunt minimizes the risk of paraplegia, attenuates metabolic acidosis, and relieves hypotension.

BLOOD PRODUCTS

Whole blood: 500 mL. Contains red blood cells (RBCs), plasma, white blood cells (WBCs), and platelets, along with 63 mL anticoagulant/preservative. Given to increase red cell mass and plasma volume.

Packed red blood cells (PRBCs): 300 mL. Contains RBCs, WBCs, platelets, and some plasma. Each unit will raise the hematocrit (HCT) by one third or hemoglobin (HGB) by 1 g/dL. PRBCs are deficient in Factors V and VIII.

RBC washed: 175 mL. Has no plasma; has increased RBC mass; confers a reduced risk of allergy to plasma proteins.

RBC leukocyte poor: 250 mL. Has no plasma; has increased RBC mass; confers a reduced risk of febrile reaction due to leukocyte antibodies.

Platelets: 50 mL/unit. Don't put on ice or heat, as such a temperature change affects platelet function. Consists of platelets and plasma; each unit will increase the platelet count by 7500 to 10,000. Controls bleeding associated with decreased platelet number or function.

- A male older than age 18 can receive any Rh and type.
- A female of child-bearing age must receive Rh-appropriate platelets but any type is acceptable.
- A child younger than age 18 must receive an exact match in terms of Rh and type.

Fresh frozen plasma (FFP): 200 mL. Must be ABO compatible with the recipient's RBCs; Rh matching need not be considered. FFP reverses the effects of Coumadin (warfarin). FFP contains all coagulation factors in normal amounts; it has no platelets, leukocytes, or RBCs. FFP is made from plasma removed from a unit of whole blood and frozen; it is not a concentrate of clotting factors. One unit of FFP increases clotting factors by 2%.

Cryoprecipitate: 15 mL/unit. Contains fibrinogen F1, von Willebrand's Factor VII, and fibrin F13. Cryoprecipitate is given for hemophilia and hypofibrinogenemia; it is also given to correct factor deficiencies. One bag contains 100 units of Factor VIII and 250 mg of fibrinogen.

Autologous blood does not need a filter.
Scavenged cell saver blood (BRAT) needs a filter.
Platelets can use regular tubing or blood tubing.
PRBC, cryoprecipitate, and FFP can use blood tubing.
All products can be put through a fluid warmer except platelets,
which should not be cooled or warmed.

CARCINOID SYNDROME

Carcinoid syndrome refers to an array of symptoms that occur when a proliferation of cells secrete several vasoactive substances (e.g., serotonin, bradykinin, histamine, prostaglandins, and polypeptide hormones) from malignant carcinoid tumors. These tumors arise from endocrine cells situated in the ileum, although they can also arise from anywhere in the gastrointestinal (GI) tract, pancreas, gonads, or the bronchi. Serotonin release causes a syndrome of episodic cutaneous flushing, diarrhea, bronchospasm, supraventricular dysrhythmias, hyperglycemia, and valvular heart disease and, less commonly, asthma.

The current treatment of choice for significant cardiovascular complications or bronchospasm with suspected carcinoid syndrome is:

- Octreotide (Sandostatin) IV neutralizes serotonin, gastrin, insulin, glucagon, and vasoactive intestinal peptide (VIP). It is a universal inhibitor of GI motility that acts by binding to somatostatin receptors in the GI tract.
- Histamine (H_1 and H_2) blockers help the histamine-related symptoms.
- Phenoxybenzamine and phenothiazines control flushing from bradykinin release.
- Glucocorticoids, indomethacin, and nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit the bradykinin system and control flushing.
- Aminophylline and steroids ease bronchospasm (secondary to bradykinin).
- Ondansetron can relieve nausea and diarrhea.
- Digitalis and diuretics may help with congestive heart failure secondary to valve malfunction.

Carcinoid Crisis

Carcinoid crisis occurs in patients with 5-HIAA levels greater than 200mg/day and is precipitated by stressful events (e.g., anesthesia, manipulation, chemotherapy, hepatic artery embolization). Patients

can experience flushing, severe abdominal pain, explosive diarrhea, CNS depression, CV instability, profound hypotension, CV collapse, severe hypertension, tachycardia, and bronchospasm.

Treat carcinoid crisis with Octreotide 50–100 mcg IV over 30 minutes, fluid resuscitation, and direct-acting vasopressors.

Surgical Resection of Carcinoid Tumor

Intraoperative management includes the following measures:

- Hydrate the patient well and consistently maintain blood volume.
- Monitor the CVP and urine output.
- Monitor the blood pressure by arterial line.
- Place a pulmonary artery catheter if the patient has advanced cardiac disease.
- Avoid drugs that release histamine, such as morphine and atracurium.
- Use succinylcholine with caution: It increases intra-abdominal pressure, but has been used safely in these surgeries.
- Check glucose levels intermittently, maintain glucose levels between 80–120 mg/dL.

Postoperative treatment consists of chemotherapy with 5-fluoruracil (5-FU) and doxorubicin.

CARDIOPULMONARY BYPASS;

Why Off-Pump Is Done

As the serious complications associated with traditional coronary artery bypass graft (CABG) and cardiopulmonary bypass (CPB) were increasingly identified, and cardiac stabilization devices were developed, performing coronary bypass without the use of cardiopulmonary bypass found new popularity.

Some of the many complications of traditional CABG and CPB include microembolization with resultant strokes and cerebral dysfunction; platelet consumption or sequestration and endothelial dysfunction, causing coagulopathy and anemia requiring blood product transfusion; aortic cannulation; systemic inflammatory response and activation of fibrinolysis; sternal wound infections; and respiratory insufficiency. There is a higher incidence of renal failure after CPB as the kidneys operate best with pulsatile flow.

On-Pump Issues

Cardiopulmonary bypass allows the heart muscle to be still while the patient's blood is diverted, oxygenated, and then reperfused. CPB can be either total or partial. Three components of CPB are hemodilution, hypothermia, and anticoagulation; these three components contribute to the complications associated with CPB.

CPB must be established so that blood can be taken from the body and returned. Blood is usually diverted from the body at the right atrium, the femoral vein, or the inferior vena cava. Blood is usually returned to the body by an artery—ascending aorta, femoral, or subclavian.

Weaning off bypass may need to be gradual if the patient's blood pressure is too low. The perfusionist will partially occlude the venous line, allowing the heart to fill; the arterial flow from the pump is then decreased and ejection begins. It is also possible to use "parallel circulation" (the patient's own circulation along with bypass support) during CPB. Time spent in parallel circulation is directly related to the risk of myocardial dysfunction.

If the patient has increased BP, the bypass flow should be shut off completely.

If the patient has decreased BP, it is recommended to maintain partial CPB (parallel circulation) and begin appropriate infusions. Continue to assess blood pressure and contractility, titrate volume, and give appropriate drugs during further attempts to reduce CPB flow.

CEREBRAL PROTECTION

Cerebral protection: $\downarrow$ CPP = $\downarrow$ CBV = $\downarrow$ ICP = $\downarrow$ CBF

Cerebral blood flow (CBF) is normally auto regulated with the body maintaining a mean arterial pressure (MAP) between 50 and 150 mm Hg (newer

data suggest that this range is 70–150 mm Hg); vascular resistance changes in response to pressure changes. Patients who have shifted to the right (chronically hypertensive patients in whom auto regulation occurs in a higher MAP range) have adapted to higher pressures and will not tolerate hypotension.

To protect the patient's brain, it is essential to maintain good cerebral blood flow, normocapnia, blood sugar levels, oxygenation levels, and carrying capacity.

Pharmacologic Methods of Cerebral Protection

Cerebral Vasoconstrictor

Cerebral metabolism may be reduced by administration of a *barbiturate* (e.g., sodium thiopental [Pentothal]), which is a potent cerebral vasoconstrictor that decreases CBF, cerebral blood volume, $CMRO_2$ (cerebral metabolic rate of oxygen), and ICP. The cerebral metabolism reduction dose of thiopental is 4 mg/kg (2–5 g range dose). Pentothal is the only drug proven to save an ischemic brain. Barbiturates are thought to enhance gamma-aminobutyric acid (GABA) activity and antagonize the N-methyl-D-aspartate receptor, thereby reducing ischemic excitotoxicity.

Propofol and etomidate are also potent cerebral vasoconstrictors. The use of this technique must be weighed against the potential complications if the symptoms of cerebral ischemia are present, because the possible hypotension can compound the ischemia.

Burst Suppression

Barbiturates are also used for burst suppression because they follow a pattern of initial EEG activation followed by dose-related depression. Eventually high doses lead to lengthening periods of suppression alternating with periods of activity. Pentothal burst suppression doses consist of 10–30 mg/kg total dose and can reduce EEG electrical activity (in a dose-dependent manner) by as much as 50%.

Propofol will induce burst suppression in a dose-dependent fashion and has the potential to be as beneficial as thiopental. Furthermore, this agent is metabolized quickly, providing a more predictable wake-up time for the patient.

See Chapter 9, Special Techniques, "Neurophysiologic Monitoring," for more information.

Fluid restriction is rarely used to lower ICP for purposes of cerebral protection, as it can cause hypovolemia, resulting in hypotension with anesthesia induction.

Loop diuretics used for cerebral protection include furosemide 0.3 mg/kg (or 10–20 mg IV), Edecrin, or Bumex. These medications decrease blood volume; the onset of action occurs in 10–30 minutes. Lasix (furosemide) lowers BP by reducing intravascular volume and cardiac output; it may require as long as 30 minutes to lower ICP.

An *osmotic diuretic*, Mannitol 0.5–2 g/kg (or 25–50 g) increases urinary output, acts as a free-radical scavenger, and decreases ICP. The surgeon will often order a specific amount in total to be given, such as 50 g mannitol. (25% is 12.5 g in 50 mL) to be given over 15–30 minutes. Mannitol crystallizes and should be drawn up through a filter needle; an administration set with a filter should be used for infusions containing 20% or more of this agent. Mannitol acts within 10–15 minutes and its effect last as long as 2 hours.

Mannitol can cause a triphasic hemodynamic response:

Phase 1: transient (1–2 minutes) hypotension after rapid administration. Mannitol causes vasodilation, the extent of which is dependent on the dose and the rate of administration.

Phase 2: transient increase in blood volume, cardiac index, and PCWP. The maximum increase occurs shortly after termination of infusion.

Phase 3: 30 minutes after infusion, blood volume returns to normal; PCWP and cardiac index drop to below normal levels because of peripheral vascular pooling. Due to mannitol's potent diuretic effect, excessive administration of this agent may result in systemic hypotension and cerebral hypoperfusion.

Mannitol can also have a biphasic effect on ICP:

Phase 1: transient increase in ICP due to increased CBF and CBV. This increase in blood volume and ICP may be attenuated by giving Lasix beforehand.

Phase 2: maximal reduction of ICP within 10 minutes of administration, an effect that persists for 3–4 hours. Mannitol increases serum osmolarity, which creates an osmotic gradient across an intact blood—brain barrier (BBB), drawing water out of the brain parenchyma. (e.g., from interstitial and intracellular spaces into the intravascular system).

Corticosteroids stabilize cell membranes, prevent release of chemical mediators, decrease edema, and act as free radical scavengers. These agents also reduce swelling around tumors, improve pulmonary compliance, and decrease pulmonary vascular resistance. Although it may take many hours or days before the ICP is reduced, the advantage associated with use of corticosteroids is that these agents may restore the BBB. Examples include Decadron 10 mg IV or methylprednisolone (also known as methylprednisone or SoluMedrol) 1 g IV or 30 mg/kg.

While administration of steroids is useful in reducing cerebral edema, the associated hyperglycemia can be detrimental to the ischemic brain. Insulin infusions should be used to help reduce blood glucose to normoglycemic levels when corticosteroids are given.

Other pharmacologic methods of cerebral protection include *judicious use of anesthetic agents* and good skeletal *muscle relaxation. Antiseizure medication* may also confer this kind of protection, such as Dilantin (phenytoin) 1 gram IV piggyback drip. Give Dilantin very slowly as an IV medication, as its administration can cause profound hypotension and arrhythmias. Seizure activity produces increases in both CBF and $CMRO_2$ by "supranormal" energy demand.

Magnesium may act as a free-radical scavenger; it may exert beneficial effects during brain ischemia by dilating cerebral vascular smooth muscle.

Calcium-channel blockers such as nimodipine may exert beneficial effects during brain ischemia by acting on cerebral vascular smooth muscle.

Interventions in Cerebral Protection

A simple mnemonic to remember in terms of cerebral protection is the "30-31-32 rule": keep the head of bed (HOB) up 30 degrees—hematocrit ≥ 31%—body temperature at 32°C.

Carbon dioxide (hypercarbia) is a potent cerebral vasodilator. Low normal $ETCO_2$ (30–34 mm Hg) is ideal. At that level, CBF is decreased by approximately 10%. CBF decreases 4% if the $PaCO_2$ decreases from 34 to 33 mm Hg.

Hyperventilation is still the fastest way to decrease brain bulk by blowing off carbon dioxide; decreases in carbon dioxide cause cerebral arterial vasoconstriction and reductions in CBF and CBV. Vasoconstriction occurs so quickly with hyperventilation because CO_2 crosses the BBB without limitation. The effect may last only 6–8 hours before the body's balancing mechanisms kick in (metabolic; bicarbonate levels change). One disadvantage of hyperventilation is that the decrease in CBF can cause ischemia in brain tissue.

Hyperventilation Benefits

- Cerebral vasoconstriction and reductions in CBF, $CMRO_2$, CBV, and ICP.
- Increased pH decreases acidosis.
- Inverse steal to feed injured tissue.
- Decreased brain bulk.
- Reduction in brain tissue bulk increases exposure for surgeon.

Hyperventilation should not be used in patients with possible focal ischemia because of the "steal" phenomenon: During periods of cerebral ischemia, blood flow is increased in *normal* areas of the brain but not in ischemic areas.

A mild decrease in *body temperature* to 32–33°C is most effective in providing cerebral protection; it decreases both basal and metabolic requirements.

$CMRO_2$ decreases 7% for every 1°C decrease in temperature. This is due to a reduction in the transmembrane ion flux and basal energy expenditure, which produces a neuron-sparing effect. To use this approach to provide cerebral protection, passively expose patient to decrease body temperature, or lay the patient on a cooling blanket but turn it on only if necessary. At the end of the procedure, use a warming blanket to prevent shivering. Hypothermia can cause platelet dysfunction.

Blood viscosity can also be used to provide cerebral protection. The optimal hematocrit range is 30–33. Anemia increases CBF; polycythemia decreases CBF. A hematocrit of 33 decreases viscosity, which in turn decreases CBF but still provides good oxygen-carrying capacity.

Postural changes (i.e., raising the head of the bed up to approximately 30 degrees) are associated with increased venous drainage and a lower ICP. Keep the patient's neck straight to facilitate venous outflow.

Planned CSF drainage from a ventricular catheter or lumbar drain can also be used to protect the brain. The collection device is placed at a scale that allows for drainage. The drainage device is placed at a certain level (say, 15 cm H_2O), and the CSF drains only when pressure is above that level. A scale too high would impede drainage, whereas a scale too low would allow too much CSF to flow out. Strict parameters of CSF removal, including how much and how often, have been established. If a lumbar drain is inserted in a patient with increased ICP, there is a chance the brain tissue will herniate through the foramen magnum.

With this technique, a ventriculostomy drain to monitor ICP or drain CSF is placed in lateral horn of the lateral ventricle near the foramen of Monroe in the brain. The transducer is leveled at the tragus of the ear (or external auditory meatus). A ventriculostomy catheter can be used to drain CSF, although one should never remove more than 10 mL at a time or as much as 50 mL in total.

Normotension alterations—namely, induced hypotension—is implemented only at surgeon's request; it is rarely used for cerebral protection purposes (hypertension causes an increase in CBV). Deliberate hypotension may be utilized as a means to decrease blood loss and hyperemic complications (cerebral edema or hemorrhage).

Good oxygenation (hyperoxia) is based on the following premises: A PaO_2 less than 60 mm Hg profoundly increases CBF and causes anaerobic metabolism. The condition worsens ischemia by releasing free radicals and worsening neurologic injury. Studies show that a marked improvement in the survival of cortical neurons occurs if PaO_2 kept above 200 mm Hg.

Blood glucose concentration is also important during episodes of ischemia. It is thought that hyperglycemia, in the presence of hypoxia, will increase intracellular acidosis. Insulin has a direct protective effect on ischemic neural tissue. Insulin and glucose infusions should be used to bring the patient to a normoglycemic level.

Deep hypothermic circulatory arrest (DHCA) for cerebral protection is discussed later in this chapter.

COMMON ALLERGIES, ASTHMA, AND HISTAMINE IN THE OPERATING ROOM

Agents Associated with Anaphylaxis

- #1 Muscle relaxants: rocuronium, succinylcholine, atracurium
- #2 Latex
- #3 Antibiotics: penicillin, cephalosporin, vancomycin
- #4 Colloids
- #5 Hypnotic/induction agents: propofol, thiopental, midazolam

Asthmatic Patients

Drugs to avoid with asthmatic patients include beta-2 blockers (e.g., propranolol, labetalol) and histamine-releasing drugs. Histamine-releasing anesthetic drugs include trimethaphan (Arfonad), succinylcholine, mivacurium, atracurium, opioids (MSO_4), Demerol, thiopental, and curare.

Histamine release from mast cells results in bronchospasm, hypotension from peripheral vasodilation, and skin flushing. The effects of histamine can be minimized by slow opioid infusion, adequate IV volume, or pretreatment with H_1 or H_2 antagonists. True antibody-mediated allergic reactions are extremely rare. Histamine release is associated with signs and symptoms including flushing, itching, and even wheezing. Hypotension can occur; it is especially likely to occur in a hypovolemic patient, in whom it may lead to orthostatic hypotension. Nausea, vomiting, and retching due to stimulation of chemoreceptor trigger zone (parasellar region lateral medulla) by the opioid are possible as well and may be exacerbated by movement (e.g., vestibular stimulation).

Histamine blockers come in two varieties: H_1 blockers, such as Benadryl (diphenhydramine), and H_2 blockers, such as Tagamet, Zantac, Axid, and Pepcid.

DEEP HYPOTHERMIC CARDIAC ARREST

Deep hypothermic cardiac arrest (DHCA) is used in pediatric intracardiac defect surgery (e.g., transposition of great arteries, hypoplastic left heart syndrome), major adult vascular procedures such as aortic arch reconstruction, removal of hepatic and renal tumors, intracranial surgery, and cardiac surgery. The goal in using DHCA is to lower body temperature, thereby lowering the patient's metabolic rate and oxygen demand to the point where blood flow and oxygen delivery can be completely stopped for an extended period of 45–60 minutes.

Initiation of cooling is done once cardiopulmonary bypass is established. Pharmacologic cerebral protection is implemented prior to initiating hypothermic arrest.

The perfusionist decreases the core body temperature to 14–18°C; that temperature is maintained until approximately 20 minutes prior to circulatory arrest. The period of hypothermic circulatory arrest with minimal cerebral complications is approximately 45 minutes at 18°C and 1 hour at 15°C. In addition to the cooling implemented by the perfusionist, the anesthetist packs the patient's head in ice, over towels (to protect the skin, nose, eyes, and earlobes from frostbite). The head temperature is monitored with a nasal or tympanic probe. The best estimate of the brain temperature is measured in the nasopharyngeal area. The rectal temperature measures the body temperature.

The following monitors are commonly used in DHCA:

- Right radial arterial line
- Right femoral arterial line
- Pulmonary artery catheter
- Separate CVP line to monitor retrograde cerebral flow
- Transesophageal echocardiography (TEE) monitor

Cerebral Protection with Hypothermic Blood Flow

Both antegrade cerebral perfusion (ACP) and retrograde cerebral perfusion (RCP) allow for continued hypothermic blood flow to the brain while maintaining a bloodless field.

Antegrade Cerebral Perfusion

ACP is a technique utilized to maintain selective cerebral perfusion during total-body DHCA. Once DHCA has been established, depending on the surgeon's aortic cannula placement (generally through the carotid or brachiocephalic artery), the cannula is either removed or clamped. The dissected/aneurysmal aorta is opened. Ballooned retrograde perfusion catheters are then placed into the innominate artery (to access the right carotid) and directly into the left carotid; these catheters are attached to individual pump heads on the CPB machine. A flow rate of 500–700 mL/min of cold blood at 20°C is delivered; thus the brain is perfused with cold blood while the patient is maintained in DHCA. Distal and arch vessels are anastomosed to the replacement aortic tube graft;

ACP is terminated and the balloon catheters are deflated and removed. The new graft is flushed and de-aired with trickle blood flow from the arterial cannula. Once the new graft is de-aired, total body perfusion is reinitiated and rewarming begins.

ACP has several notable advantages. For example, cerebral flow provides uniform cooling in a normal flow direction, and metabolic substrate delivery tends to have more favorable outcomes. ACP has also been shown to be a more effective cerebral protector than retrograde flow. Disadvantages include the complexity of the procedure, the presence of an extra cannula on the surgical field, an increased risk of emboli, problems with fragile vessels, and the difficulty level of performing this procedure.

Retrograde Cerebral Perfusion

RCP, with deep hypothermic circulatory arrest, will increase the safe arrest time. Due to the greater fragility of the venous system (compared to the arterial system), RCP is maintained at a pressure of 20–25 mm Hg at 8–14°C (flow rates depend on pressures but are usually approximately 500–800 mL/min). The patient is placed in the steep Trendelenburg position to ensure brain perfusion.

After DHCA is initiated, the arterial perfusion line is connected to the SVC cannula and low perfusion flow is initiated backward or retrograde through the brain. The brain is perfused in retrograde fashion with cold, oxygen-rich blood with 100% oxygen saturation via SVC to jugular vein; the blood will come back through the carotid and innominate arteries and is recovered by suction.

Advantages of RCP include effective global cerebral cooling; decreased cerebral embolization; ability to wash out air bubbles, embolic debris, and metabolic waste products; use of simpler system; avoidance of fragile vessels; and delivery of oxygen and nutritional substrates to brain tissue. Moreover, use of RCP decreases strokes and neurologic injury.

Disadvantages of RCP include increased cerebral edema from over-perfusion; the fact that substrate delivery is *not* reliable, as only 20–60% of the brain is perfused with retrograde flow; ability of venous valves to alter flow; ability of collateral veins to divert the flow; and ability of drainage from the RCP catheter to affect the surgical view.

Anesthesia Considerations with DHCA

- Prolonged effect of anesthesia agents due to slowed drug metabolism and elimination
- Decreased minimal alveolar concentration (MAC) of inhalational agents
- Need for a narcotic, amnestic, and nondepolarizing muscle relaxant (NDMR) when the patient is rewarmed to 34°C.
- Positioning and padding must be meticulous as DHCA leaves the patient more susceptible to nerve and tissue injury
- Monitor urine output every 30 minutes and report it to the perfusionist

Surgical Considerations with DHCA

DHCA provides for a bloodless field. There is also no need for aortic cross-clamping, thereby reducing the risks associated with embolization sequelae.

Systemic Effects from DHCA

Cerebral Effects

- Rapid core cooling to 15–18°C. EEG becomes flat at 18°C.
- Protection with metabolic suppression occurs by severe reduction of body temperature, reducing intracellular enzymatic reactions and thereby reducing oxygen demand and cerebral blood flow.
- Fewer cerebral intracellular enzymatic reactions.
- Proportionate reduction in cerebral oxygen requirements and blood flow.
- Brain tissue protectant—preserves pH and ATP stores.
- Decreased cerebral oxygen consumption. The patient is able to tolerate anoxia longer.

- Edema, which can occur after hypothermia due to cellular membrane permeability.
- An isoelectric EEG and bispectral analysis monitor (BIS) reading of 0—used to gauge the degree of hypothermia necessary to prevent neurologic damage. Different areas of the brain have varying abilities to tolerate a lack of oxygen: Some regions tolerate very short ischemic times, whereas others can sustain function for longer periods under these conditions. The amount of time the brain can tolerate ischemia depends on its tissue energy stores and the rate of energy consumption. The rate of energy consumption depends on temperature, metabolic rate, brain activity, and the use of anesthetics.
- Hypothermia: $CMRO_2$ is decreased by approximately 7% per 1°C decrease in temperature. A mild body temperature decrease to 32–340°C is most effective; it decreases both basal and metabolic requirements.
- Blood viscosity: Optimal hematocrit is 30–35.
- Injection of Papaverine into subdural space—can be done to dilate the anterior spinal artery to preserve blood flow.

Cardiovascular Effects

- Oxyhemoglobin dissociation curve: shifts to the left with oxygen less readily available to the tissues. The reduced oxygen demand from core cooling usually mitigates this effect.
- Decreased heart rate.
- Increased SVR with decreased cardiac output and decreased LV compliance.
- Prolonged refractory period.
- Heart: most susceptible to ventricular fibrillation when the body is warmed to 30°C.

Hematological Effects

- Coagulopathy from the profound cooling—due to clotting factor and platelet dysfunction and activation (of platelets) with release of granules. Hemodilution is needed to prevent increased viscosity of the patient's blood at these extremely cold temperatures, which also helps increase the tissue perfusion.
- Cerebral macro-circulatory obstruction occurrence from coagulopathy.
- Impaired enzymatic activity of clotting factors, promoting abnormal clot formation; a slowed coagulation cascade; decreased platelet number and function. DHCA contributes to greater blood product requirements.
- Decreased heparin metabolism.
- Hyperglycemia: Requires monitoring of glucose levels, maintain between 80–120 mg/dL.

Metabolic Effects

- Change of 1°C = 7% metabolic decrease.
- Carbon dioxide production—decreased due to the decreased metabolic rate. CO_2 levels should be adjusted to maintain a $PaCO_2$ of 40 mm Hg to help prevent cerebral injury.
- Metabolic acidosis due to peripheral hypoperfusion; increased production of lactic acid with cold body temperature. $ETCO_2$ levels will rise with the reperfusion after blood flow is restored.

DOUBLE-LUMEN ENDOBRONCHIAL TUBE

One-lung ventilation allows for the collapse of the operative lung; it facilitates surgical exposure. With a double-lumen endobronchial tube (DLEBT), the following settings are used:

- Proximal tracheal cuff: 10–20 mL air (high volume, low pressure)
- Bronchial cuff: 3 mL only

The tube and stylet should be coated liberally with a water-soluble lubricant (or spray).

DLEBT Sizing in Adult Patients

- Height of 170 cm (approximately 5 feet, 5 inches): 29 cm at central incisors; advance or withdraw the tube by 1 cm for each 10-cm height difference.

< 165 cm (less than 5 feet, 3 inches): 35–37 Fr

165–179 cm (5 feet, 3 inches to 5 feet, 8 inches): 37–39 Fr

> 179 cm (greater than 5 feet, 8 inches): 39–41 Fr

- Females: typically use DLEBT 35–39; *place the largest device you can to decrease resistance*
- Males: typically use DLEBT 39–43

DLEBT Sizing in Pediatric Patients

	Preemie	Birth	6 mo	1 y	2 y	3 y	4 y	5 y	6 y	7 y	8 y	10 y	12 y	> 15 y
kg	1.5	3.5	7	10	12	14	17		20		25	33	40	50+
lb	3.5	7.5	15	22	26	31	37	40	44	49	55	73	88	
DLEBT size											26	26–28	32	35

For all patients, it is important to *remove the stylet before rotating and advancing the DLEBT to avoid tracheal or bronchial lacerations.* Once the tube is thought to be placed properly, the tracheal cuff should be inflated and equal ventilation of both lungs established. (Is it through the vocal cords and in trachea?)

To check for proper position, go into the tracheal lumen first. In pediatric patients, use a *pediatric flexible fiber optic bronchoscope.*

Obtain a baseline ABG on two-lung ventilation; remeasure ABG after 15 minutes on one-lung ventilation.

When going from two-lung to one-lung ventilation:

- Go to 100% FiO_2.
- Decrease the tidal volume.
- Increase the respiratory rate.
- Clamp the tube to the non-dependent lung.
- Open the pop-cap to the air.
- Avoid peak airway pressures greater than 40 mm Hg.

In case of decreasing SaO_2 with DLEBT with one-lung ventilation:

- Notify the surgeon. Discuss which modality is most advantageous for the patient and least likely to interfere with the surgery.
- Administer 100% FiO_2.
- Check tube position and make sure the cuff has not moved.
- Deliver continuous positive airway pressure (CPAP) on the *deflated* lung (surgical lung).
- Deliver positive end-expiratory pressure (PEEP) on the inflated lung.
- Jet ventilation may be helpful with low driving pressure and an increased respiratory rate.
- Allow the patient to desaturate to a certain level, have the surgeon stop the procedure, inflate both lungs and oxygenate the patient to the desirable level, and then clamp lung again (until the patient desaturates again).
- Band the pulmonary artery (PA) to stop blood flow to the surgical lung. This step can be taken only if the surgical team is performing a total pneumonectomy.

Indications and Contraindications for DLEBT

Absolute indications (to isolate one lung from the other): Infection, massive hemorrhage, bronchopleural fistula, bronchopleural cutaneous fistula, washout for alveolar proteinosis, bronchial disruption or trauma, unilateral cyst, or bullous. Video-assisted thoracoscopic surgery (VATS) requires lung separation and is becoming the most common indication for one-lung ventilation.

Relative Indications: Surgical exposure for thoracic aortic aneurysm; pneumonectomy; upper, middle, or lower lobectomy; or esophageal surgery.

Contraindications: Airway lesion, poor laryngeal visualization, or patients so unstable that short periods of apnea would be life-threatening.

EMBOLISM

Venous Air Embolism

Air embolism is the abnormal presence of air or carbon dioxide in the vena cava and right atrium, resulting in obstruction of the flow of blood through the heart. *Venous air embolism* (VAE) is related to a mass of foamy bubbles that interfere with the right heart venous outflow tract; blood cannot get into the heart (venous return) and, therefore, cannot get into the lungs because of all the air. Cardiac output will fall and circulatory collapse may result. One-third of 1 mL air can cause problems; *50 mL air is lethal*. The latter condition causes air lock in heart (air cannot flow into lungs) accompanied by a foamy, incompressible mass.

The physiologic consequences of VAE depend on the volume of air, the rate of air entry, and the presence or absence of a patent foramen ovale (PFO). A PFO can facilitate passage of air into arterial circulation (paradoxical air embolism), especially when the normal transatrial (left→right) pressure gradient is reversed. Reversal of this gradient is favored by hypovolemia and perhaps by PEEP. Potential end-organ damage can also occur from paradoxical air emboli to the microvascular circulation.

VAE can occur when the pressure within an open vein is subatmospheric. This pressure gradient develops when the surgical site is higher than the right atrium (approximately 2-torr pressure difference for 1-inch difference in height). These conditions may exist in any position (and during any procedure) whenever the wound is above the level of the heart:

- Sitting craniotomies (highest incidence of VAE, at 25–50%), especially with open bone.
- Severe barotrauma associated with mechanical ventilation.
- Open sinuses.
- Open, large veins.
- Cement impaction in orthopedic surgeries, when air is forced into vessels.
- Sitting position.
- Side-lying posterior fossa procedures (Brain surgery is dealing with highly vascular membranes; e.g., the dura can entrain air when cut.).
- Low central venous pressures (especially from positioning the legs too low)
- Inadvertent opening of a large-bore venous catheter
- Accidental IV bolus of air.
- Traumatically, as by a puncture wound

Nitrous oxide can markedly accentuate the effects of even small amounts of air.

Monitors for VAE

The monitors for VAE are presented here in order of decreasing sensitivity.

Transesophageal echocardiography (TEE): Transesophageal two-dimensional echocardiography is the most sensitive intraoperative monitor but some consider it "too sensitive" due to the potential for false positives. TEE "sees" every bubble. Some argue though that detecting even small amounts of venous air embolism is important because it allows surgical control of the entry site before additional air is entrained. TEE has the added benefits of detecting the amount of bubbles and their transatrial passage, as well as evaluating cardiac function. TEE is expensive and requires trained personnel to be continually available for interpretation. There is also potential for injury to the esophagus or larynx; there have been reports of esophageal rupture and recurrent laryngeal nerve injury with TEE use.

Precordial Doppler: The monitor is placed over the right atrium ventricle at the second

through sixth intercostal spaces at the right sternal border. Interruption of regular swishing of the Doppler signal by sporadic roaring sounds or a high-pitched whoosh (called mill-wheel murmur) indicates venous embolism. Even 0.25–0.5 mL of air can be detected. Precordial Doppler is considered the best air embolism monitor, because it is sensitive but does not generate a lot of false positives.

$ETCO_2$ monitoring: A sudden decrease in expired carbon dioxide may indicate VAE.

Signs of VAE

The following signs also have air emboli as a differential diagnosis. *These signs can occur with anaphylaxis, acute myocardial infarction, and pulmonary embolism*.

- Sudden hypotension
- Decreased SaO_2/PaO_2
- Change in heart sounds, cardiac murmur
- EKG changes and dysrhythmias; tachycardia
- Sudden decrease in expired carbon dioxide ($ETCO_2$): decreases due to a fall in cardiac output and increased dead space
- Appearance of nitrogen in the expired gas
- Increased pulmonary artery pressure (PAP) and central venous pressure (CVP)
- Sudden appearance of vigorous spontaneous ventilation despite continuing mechanical ventilation

Signs of VAE are often not apparent until large amounts of air have been entrained. Rapid entrainment of large amounts of air can produce sudden circulatory arrest by obstructing right ventricular outflow.

Treatment of VAE

- Inform everyone.
- Immediately cease insufflation and release the pneumoperitoneum.
- Hyperventilate with 100% oxygen; discontinue nitrous oxide if it is being used.
- The surgeon will flood the surgical field with normal saline and apply bone wax.
- Aspirate the central venous line/PA line in an attempt to retrieve the entrained air. Note that the aspirate may appear foamy.
- Occlude the neck veins; discontinue pressurized gas.
- Place the patient in steep Trendelenburg or left lateral position.
- Vasopressors should be given to correct hypotension.
- Give intravascular volume infusion to increase central venous pressure.
- Use PEEP/CPAP to increase CVP (although there are some arguments against doing this).
- Undertake cardiopulmonary resuscitation, if necessary.

Prevention of VAE

Prevent hydrostatic gradient development by limiting positions where the operative site is above the right atrium. Maintain vigilance for VAE in high-risk situations.

Carbon Dioxide Gas Embolism

There is a risk of carbon dioxide embolus during a laparoscopic procedure; gas may enter the circulation through any opening in an injured vessel. This type of embolism is the most dangerous complication associated with laparoscopy. It develops principally during induction of a pneumoperitoneum. With carbon dioxide embolus, "gas lock" occurs in the vena cava and right atrium; venous return is obstructed. Blood cannot go into pulmonary artery. Cardiac output will fall and circulatory collapse may result.

Signs of Gas Embolism

Signs of carbon dioxide embolus include a sudden decrease in expired carbon dioxide ($ETCO_2$; $ETCO_2$ *decreases* due to the fall in cardiac output and increased dead space), unexplained tachycardia, EKG changes and dysrhythmias, sudden hypotension,

increased PAP and CVP, a change in heart sounds, a cardiac murmur, the appearance of nitrogen in the expired gas, and decreased SaO_2/PaO_2.

Treatment of Gas Embolism

- Immediately cease insufflation and release the pneumoperitoneum.
- Position the patient in a steep head-down and left lateral decubitus position.
- Hyperventilate with 100% O_2.
- Aspirate gas if a CVP catheter is in place.

Fat Embolism Syndrome

Fat embolism syndrome (FES) is a rare clinical condition in which embolized and circulating fat particles are deposited in the pulmonary capillary beds and brain tissue and lead to multisystem dysfunction in the skin, lungs, blood, and brain. Microvascular plugging of these fat droplets produces local ischemia, causing the release of inflammatory mediators and platelet aggregation.

Implicated procedures and causes include long bone fractures, multiple fractures with pelvic injury, total joint replacement, intra-abdominal surgery, liposuction, bone marrow transplant, and burns. A factor increasing the risk of FES is aggressive reaming or nailing of the bone's medullary cavity. Mobilized fat particles occur to some degree in most long bone fractures.

Symptoms may be subclinical or masked by general anesthesia; manifestations of FES may occur 12–72 hours after injury.

Signs and Symptoms of FES

FES has three main symptoms: dyspnea, confusion, and petechiae. All together, the following signs can be seen with FES:

Skin

- Petechiae (transient cutaneous pin-point sized red dots in the axilla, conjunctiva, neck, shoulders, chest, arms)

Lungs

- Dyspnea
- Increased $PaCO_2$
- Decreased PaO_2 and oxygen saturation
- Decreased $ETCO_2$ and arterial hypoxemia
- ARDS (interstitial edema, alveolar collapse, decreased compliance)
- Sudden increase in peak inspiratory pressures

Neurological

- Level of consciousness (LOC) changes
- Confusion
- Seizures
- Coma

Heart

- EKG changes (dysrhythmias, tachycardia, ischemic changes)

Blood

- Thrombocytopenia
- Anemia
- Coagulopathy
- Disseminated intravascular coagulation (DIC)
- Increased lipase
- Triglyceride levels
- Fat globules in urine and sputum
- Decreased serum calcium levels (calcium binds with free fatty acids)

Diagnostic Tests for FES

- Fat globules in urine and sputum
- Complete blood count: anemia and thrombocytopenia
- Increased lipase and triglyceride levels
- Low calcium levels (calcium binds with free fatty acids)

Supportive Measures for FES

- Adequate hydration
- Oxygenation

- Early splinting of fracture especially long bones
- Give albumin to provide free fatty acid binding sites

High-dose steroids have not been proved effective for treatment of FES.

HEMODIALYSIS CATHETER ACCESS AND FLUSH GUIDELINES

This section outlines the authors' practice policy and is just one example of dialysis catheter access guidelines. Check your institution's policy for your specific guidelines.

- A physician's order *must* be written on the chart before the provider can access a dialysis catheter.
- Use a dialysis catheter *only* if you are unable to get any other vascular access.
- Use sterile technique when accessing these ports!
- Wear sterile gloves and clean the ports using sterile technique. Cleanse with Betadine solution for 30 seconds on the caps. Aspirate out the heparin flush and discard it; flush the port with normal saline (NS); and connect the port to the NS fluids.
- Have the circulator order heparin 5000 units/mL from the pharmacy so you will have it ready. If the dialysis catheter is *not* your only access, then you should discontinue IV fluids in the OR and flush with the previously described heparin solution. If this catheter is your *only* access, then take the heparin with you; the hemodialysis catheter can be flushed in the PACU.

HIGH-FREQUENCY JET VENTILATION

There is no bulk flow of gases with high-frequency jet ventilation (HFJV); it is referred to as coaxial flow in the larger airways. Following are the usual setting ranges for HFJV:

Rate = 60–600 cycles per minute (cpm)

- Lower cpm: < 60 cpm will increase inspiratory time (improves CO_2 elimination)
- Extremely high cpm: > 400 cpm may decrease inspiratory time (worsens CO_2 elimination)

Tidal volume (VT) = less than dead space (2–5 mL/kg)

CO_2 = eliminated by passive expirations through an open system/exhalation takes place continuously

ETT with special connector; attaches to any tube with a 15-mm connector

Factors Affecting Ventilation during HFJV

Driving Pressure

Driving pressure (DP) is measured in pounds/square inch (psi). It is the pressure of the force of inhaled gases: air/O_2 or N_2O/O_2 (*not* inhalation agents). In the Venturi effect, velocity of airflow increases through a small orifice, resulting in a decreased pressure; this effect causes a decrease in the pressure at the outflow of the jet injector, and gas will be entrained, increasing the tidal volume at 10–20%. This volume must be humidified for cases where surgery lasts more than 45 minutes or in ICUs.

Percent Inspiratory Time with HFJV

Increasing the inspiratory to expiratory ratio (I:E) ratio will increase tidal volume and minute ventilation. The range is usually set between 30% and 50%. Minute ventilation is most dependent on DP and the I:E ratio.

Jet Catheter Size and Configuration

The bigger the catheter, the greater the jet volume and minute ventilation. It is best to have two side holes for entrained volume: jet velocity or tip velocity from the nozzle.

Cycle Rates

Generally, changing the cycle rate does not change minute ventilation. The exception occurs when delivering HFJV at rates < 60 cpm and > 400 cpm.

FiO_2

The FiO_2 percent used is a matter of choice and depends on the case and technique employed. Blenders are used for O_2, air, or N_2O.

Humidity

Humidity can be delivered using a small saline bag and infusion line with some ventilators. The recommended rate is 15 cc/h 0.9% NS or 0.45% NS. This technique may not be used in the OR; it is associated with fog buildup if the surgeon is working with airway scopes.

LAPAROSCOPIC ISSUES

Laparoscopic surgery involves the introduction of a laparoscope through a single or multiple ports placed through a body wall into a cavity. Usually several 2- to 3-cm slits are made across the skin surface where ports are placed, specimens retrieved, and surgery performed. Laparoscopic surgery is done for exploration, diagnosis, and treatment purposes. To visualize an internal surface, the external body surface is distended away from the internal organs by establishing insufflation with carbon dioxide.

With abdominal laparoscopy, the parietal wall is distended away from the internal organs by establishing a pneumoperitoneum with CO_2. Before insufflating the area, the anesthetist inserts an orogastric or nasogastric tube and suctions out the stomach contents. A urethral catheter should also be placed by the OR staff.

When the trocar pierces a body cavity, the anesthetist should monitor viewing screens to look for any internal trauma (i.e., the trocar piercing an internal organ).

Insufflation pressures used are usually between 5 mm Hg and 14–16 mm Hg and can decrease cardiac output, venous return, and the lung's functional residual capacity (FRC). Increased abdominal pressure from insufflation can also stimulate vagal nerve activity, and the patient's heart rate can become bradycardic to asystolic. Notify the surgeon if the patient becomes severely bradycardic; team members can decrease insufflation pressures a little or completely until the heart rate increases. IV glycopyrrolate may need to be given to increase the heart rate. If glycopyrrolate is ineffective, IV atropine should be given. Have epinephrine 1:10,000 immediately available for severe bradycardia unresponsive to vagolytics medications.

Carbon dioxide gases can increase end-tidal carbon dioxide ($ETCO_2$) but should level out after 30–45 minutes. This increase can be offset by increasing the minute ventilations to keep the $ETCO_2$ in appropriate ranges (32–39 mm Hg). If $ETCO_2$ continues to rise, check for subcutaneous emphysema; the insufflation pressure may need to be reduced and ventilation increased dramatically.

Once pneumoperitoneum is achieved, setting the ventilator on *pressure-controlled ventilations* can help decrease airway pressures while maintaining oxygenation, lung expansion, and $ETCO_2$ while the pneumoperitoneum lasts. Tidal volumes may need to be decreased to allow adequate pneumoperitoneum. The anesthetist must be constantly aware of the presence of the pneumoperitoneum. The surgeon may decrease or desufflate at any time; if the ventilator is set on pressure ventilation and the pneumoperitoneum is lost, the patient's lung volumes may increase dramatically.

The patient may be moved from Trendelenburg position to reverse Trendelenburg position several times during surgery to change internal organ positioning so as to facilitate organ exposure for surgeon. If the patient's arms are secured on padded armboards, be sure to secure both the forearm and the upper arm to eliminate the chance of the arms rolling off the boards.

Urine output tends to diminish with insufflation of the abdomen. Nausea and vomiting are more common following laparoscopic procedures.

Patients may complain of right shoulder/shoulder blade pain after a laparoscopic surgery. This effect is thought to be due to carbon dioxide

insufflation causing chemical irritation to the diaphragm.

Carbon dioxide gas embolism, pneumothorax, pneumomediastinum, subcutaneous emphysema, and hypercarbia with ensuing acidosis are potential risks of CO_2 insufflation. Air can enter the thorax through weakened areas in the diaphragm. Immediate chest tube decompression is required for any clinical evidence of a tension pneumothorax.

Effects of Abdominal Laparoscopic Surgery on the Respiratory System

Intraperitoneal insufflation of CO_2 creates a pneumoperitoneum that results in ventilatory and respiratory changes:

- Decreases thoraco-pulmonary compliance.
- Decreases lung compliance by 30–50%.
- During uneventful laparoscopy, $PaCO_2$ progressively increases and reaches a plateau 15–30 minutes after insufflation. The increase in $PaCO_2$ depends on the intra-abdominal pressure.
- Once the pneumoperitoneum is created and kept constant, compliance is not affected by patient tilting or changes in minute ventilation.

Respiratory Complications from Abdominal Insufflation

Endobronchial Intubation

Cephalad displacement of the diaphragm during pneumoperitoneum also results in cephalad movement of the carina; this effect may occur even in laparoscopic cases in which the patient is in a head-up position. If the patient's oxygen saturation decreases after insufflation of the abdomen, check for bilateral breath sounds. The tracheal tube can go into the right mainstem bronchus, with the pressure of insufflation pushing the diaphragm upward. If breath sounds are decreased on the left side, pull the endotracheal tube back until breath sounds are audible bilaterally and retape the endotracheal tube.

Pneumothorax

Pneumothorax may occur due to a defect in the diaphragm or as a result of a pleural tear. If rupture of preexisting bullae occurs in the lung, causing air to escape into the thorax, the pneumothorax will not resolve spontaneously. A thoracentesis (pleural tap) must be done to remove the air in the pleural space. Capnothorax (carbon dioxide in the pleural space) without pulmonary trauma will spontaneously resolve 30–60 minutes after ex-sufflation. Treatment of CO_2 or venous air pneumothorax consists of adding PEEP and reducing all intra-abdominal pressure.

Carbon Dioxide Gas Embolism

See the discussion of this condition presented earlier in this chapter.

CO_2-Subcutaneous Emphysema

This condition develops secondary to extraperitoneal insufflation. It results in increased carbon dioxide levels after $ETCO_2$ has plateaued. Hypercapnia becomes unresponsive to adjustment of ventilation. To resolve this condition, laparoscopy should be temporarily interrupted to allow for CO_2 elimination.

MASK (VENTILATOR) STRAP FACIAL INJURIES

Direct pressure on the patient's brow from the ventilator face mask can cause hair loss or nerve damage. The facial nerve can also be affected, specifically at the following branches (all branches are listed):

- Temporal branch. Supraorbital pressure, especially by the ETT connector, can cause eye pain, photophobia, and/or forehead numbness.
- Zygomatic branch.
- Buccal branch. This branch innervates the orbicularis oris around the mouth; damage to this branch can cause loss of motor ability (e.g., no puckering of lips).

- Mandibular branch. Damage to this branch can cause motor loss and minimal sensory loss to the mandible.
- Cervical branch.

METHYLMETHACRYLATE BONE CEMENT

Methylmethacrylate (MMA) is a self-polymerizing bone cement that is used to secure a prosthesis inside the bone/joint. It is part of an exothermic reaction that leads to cement hardening and expansion against prosthetic components. This expansion can exert pressure in excess of 500 mm Hg in the intramedullary space. MMA can embolize fat, clot, air, cement, marrow, and bone chips. Its reaction may also trigger tissue thromboplastin, causing small clots to travel to the lung. Unpolymerized monomer can be absorbed into the circulation. Pressurization of the bone canal with cement is also associated with hemodynamic changes.

Complications of MMA Use

The following problems are associated with MMA use:

- **Hypotension**
  - MMA may be a direct vasodilator/myocardial depressant.
  - Severity: usually related to volume (could result from a pulmonary embolism [PE] or anaphylactoid reaction).
  - Onset: 30 seconds to 10 minutes.
  - Termination: usually spontaneous within 5 minutes.
  - Treatment: adequate hydration to full support.
- **Desaturation**
  - Result: In one study, 34% patients had desaturation with a baseline FiO_2 of 33%.
  - Treatment: increase FiO_2 to at least 50% during cementing.
- **Fat embolism**
  - Result: arterial hypoxemia, ADRS, coagulopathy, fever confusion, coma, seizures; petechiae on the neck, shoulders, and chest.
  - Treatment: supportive, oxygenations, corticosteroids, immobilize long bones.
- **Ambient contamination: fumes**
  - MMA should be mixed in a vented hood.
  - Liquid monomer is highly flammable.
  - Contact lenses and other plastics can be affected by MMA vapors.
  - Vapors can cause irritation of the eyes and respiratory tract.
- **Mortality**
  - Deaths can occur intraoperatively or postoperatively due to pulmonary embolism.
  - PE may result from the impact from surgical instrumentation and the expansion/pressure of the cement against the femoral shaft.

MMA use can also cause significant increases in PVR and pulmonary wedge pressure and decreases in SVR, CO, and MAP pressures. Hypotension, hypoxia, cardiovascular collapse and arrest, and pulmonary embolus following prosthesis insertion have been reported.

Prevention of MMA-Related Complications

- Better surgical lavage helps to avoid complications.
- Maintain vigilance for potential problems.
- Use 100% oxygen (FiO_2) when using MMA.

MIXED VENOUS OXYGEN CONTENT

Mixed venous oxygen content (MVO_2), also known as *saturated venous oxygen content* (SVO_2), is an index of cardiac output and overall tissue perfusion. Its ongoing measurement allows minute-to-minute assessment of total tissue oxygen balance (delivery versus consumption) at the tissue level.

MVO_2 varies directly with cardiac output, hemoglobin levels, and oxygen saturation. It varies inversely with tissue oxygen requirements and oxygen consumption (VO_2). Normal MVO_2 is considered to be in the range of 65–75%; 25% extracted

and utilized in body tissues. In other words, under normal conditions, if 1000 mL of oxygen is available, 350 mL of oxygen is used by the body's tissues and 650 mL is returned to the lungs; thus the normal SVO_2 is 65%.

Increased MVO_2 may be caused by a wedged Swan-Ganz catheter (common cause), increased FiO_2, methemoglobinemia, sepsis, hypothermia, an elevated cardiac output with left to right shunts, neuromuscular paralysis (muscles less active), or excessive inotropic drugs.

Decreased MVO_2 may be caused by a decreased hemoglobin level, a low oxygen saturation (arterial hypoxia), low cardiac output with myocardial damage, congestive heart failure, hypovolemia, hypoxia, orinadequate pulmonary gas exchange. It may also reflect increased tissue demand owing to malignant hyperthermia, thyroid storm, shivering, fever, exercise, or agitation.

If the MVO_2 falls below 30%, the oxygen balance is compromised and anaerobic metabolism ensues.

INTRAOPERATIVE NEUROLOGIC MONITORING BY THE ELECTROPHYSIOLOGIST

1. EEG (electroencephalography):
   - BIS
   - PSA 4000
2. Evoke potentials:
   - Somatosensory (SSEP)
   - Auditory (BAER)
   - Visual (VEP)
   - Dermatome (trigeminal)
   - Motor (MEP)
3. Electromyography.
4. Wake-up test.

NEUROPHYSIOLOGIC MONITORING

Electrophysiological measurements are done to detect adequate cerebral perfusion and proper neuronal functioning; protect and monitor the functional integrity of "at risk" neural structures; monitor the effects of anesthetic agents and other CNS drugs; and identify pathophysiologic conditions that can alter neurologic function. Such monitoring allows for intraoperative detection of cerebral ischemia, which may require a change in surgical technique to improve or restore perfusion. It can also be indicated when temporary occlusion of a vessel is planned, to determine the duration of tolerance, or for titration of anesthetic agents when pharmacologic metabolic suppression is desired.

The impact of anesthetic agents on neurophysiologic monitoring increases with the number of synapses in the pathway being monitored. This relationship arises because all anesthetic agents produce their effects by altering neuronal excitability via changes in synaptic function or axonal conduction.

ELECTRO ENCEPHALOGRAPHY

EEG has long been regarded as the "gold standard" for assessing cerebral ischemia during cerebrovascular procedures and is used as a guide for tolerable hypotensive techniques. The EEG is a recording of unstimulated brain waves (cortical activity) that results from spontaneous, continuous electrical activity of the brain; it measures electrical activity of the neurons of the cerebral cortex. Thus EEG may be used as a marker for detection of ischemia due to inadequate cerebral blood flow (CBF). The character of the EEG waves depends on the level of metabolic activity of the cerebral cortex and level of wakefulness.

EEG involves continuous intraoperative monitoring. The EEG technician reads the EEG monitor and analyzes the summative numerical value. Changes in the EEG are characterized by *alterations in both frequency and amplitude* as the cerebral cortex becomes increasingly ischemic. Changes range from subtle changes, such as mild loss of beta/theta activity with a mild increase in delta frequencies, to isoelectric recordings. Summation of all electrical activity and conversion into characteristic

waveforms (see Figure 3-1) is of key interest to the EEG technician:

- Beta waves: presence of increased mental stimulation and with eye opening; 12–42 Hz
- Alpha waves: typical of an awake, resting patient with the eyes closed; 8–12 Hz
- Theta waves: occur during general anesthesia and in healthy children while sleeping; 4–8 Hz
- Delta waves: occur in deep sleep, general anesthesia, and organic brain disease; less than 4 Hz

EEG is now used for the detection of depth of anesthesia (via BIS) technology.

Cerebral Blood Flow

Normal CBF is 50 mL/100 g/min.

CBF $\geqslant$ 20 mL/100 g/min: normal amplitude and latency components of cortical evoked potentials are maintained.

CBF $<$ 18 mL/100 g/min: has been associated with significant alterations in EEG signaling. Amplitude measures decline to 50% with CBF of approximately 16 mL/100 g/min; waveforms are completely abolished at levels below 12 mL/100 g/min.

CBF $<$ 6 mL/100 g/min: irreversible changes in EEG signaling.

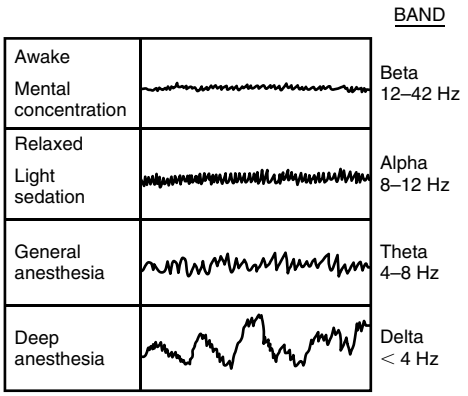


Figure 3-1 *EEG Waves, Bands*

Mean Arterial Pressure

50 mm Hg: EEG slows

25–40 mm Hg: flat EEG

$<$ 20 mm Hg: irreversible damage in normothermic patient

Other Uses of EEG

EEG can be used intraoperatively as a means by which to detect abnormal activity such as spike and wave interictal events and epileptiform discharge. These types of recording are commonly performed before and after resection of epileptic foci or temporal lobectomy. *Don't use propofol in these cases, as this agent raises the seizure threshold.*

EEG monitoring is especially useful to evaluate cerebral perfusion with clamping of major vessels. Is also used as a guide with use of hypotensive surgical techniques.

EEG Burst Suppression

Monitored by an EEG, drug-induced burst suppression is a reversible decrease in cortical neuronal metabolic function ($CMRO_2$). It is used as a cerebral protection technique. Burst suppression pattern also occurs with ischemic encephalopathy.

The characteristic signal of EEG burst suppression is often recognized with deepening of anesthesia. This pattern consists of high voltage periods of bursts and low voltage periods of suppression, each lasting from 1.5 to 6 seconds. During suppression, low-voltage mixed frequency activity can be seen. EEG findings during the suppression phase are not isoelectric.

EEG burst suppression may be seen with any anesthetic when combined with hypothermic technique.

- **General anesthetics:** Potent inhaled gases (isoflurane, sevoflurane, enflurane, desflurane) follow the basic anesthesia-related EEG pattern. Burst suppression occurs at approximately

1.5 MAC for isoflurane, desflurane, and sevoflurane. Enflurane shows burst suppression at 2–3 MAC. Sevoflurane and enflurane bursts can turn into epileptic seizure activity.

- **Barbiturates and propofol:** These drugs follow a pattern of initial EEG activation followed by dose-related depression. Eventually high doses lead to lengthening periods of suppression alternating with periods of activity (burst suppression). Pentothal burst suppression doses are in the range of 10–30 mg/kg total dose. Barbiturates reduce EEG electrical activity (in a dose-dependent manner) by as much as 50%.
- **Ischemic brain damage:** Hypoxia leads to slowing of EEG activity, with electrical activity potentially ceasing altogether depending on the severity of the event. Cerebral blood flow below 18 mL/100 g/min has been associated with significant alterations in EEG signaling.
- **Hypothermia:** Complete EEG suppression occurs at 15–18°C. Additive effects are noted with other suppressive factors (e.g., inhaled gases, barbiturates, ischemia).
- **Hyperventilation:** Hyperventilation can activate excitable seizure foci (burst).
- **Ventilation/oxygenation:** Hypoxemia can result in evoked potential deterioration.

Burst suppression on EEG can be multifactorial in origin, given that we utilize many classes of drugs and other intraoperative factors influence the EEG.

Note that the background EEG activity of neonates is much less regular than that for older children and adults.

The following medications are *not* associated with burst suppression: ketamine, benzodiazepines, opiates, halothane.

BISMonitor: Bispectral Analysis

Bispectral analysis requires fewer electrodes and gives a global assessment of EEG rather than evaluation of specific areas of the brain. It is used by anesthesia specialists to assess the level of hypnosis and possible awareness. This technique does not measure anesthetic depth.

Measurements are calculated over 15–30 seconds. A time delay may occur in rapidly changing states.

Electrode placement is provided via a prepackaged electrode setup, which is applied to the patient's forehead at the temple. The machine sets itself up automatically and tests conduction.

Advantages of BIS

- Reduced risk of awareness
- Better management of responses to surgical stimulation
- Faster wake-up
- More cost-effective use of drugs

BIS readings are affected by electrocautery, pacer spikes, and patient movement.

BIS Data Interpretation

100: awake

70: light hypnotic effects

60: moderate hypnotic effects

40: deep hypnotic effects

0: EEG suppression

Under general anesthesia, 40–60 is the desired range.

There is no guarantee the patient will have no awareness or recall. Research indicates that levels above 70 have an increased risk of recall.

BIS is not affected by neuromuscular paralysis.

PSA 4000: Patient State Analysis

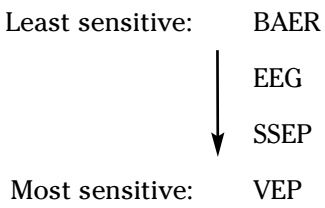
PSA 4000 analyzes a four-channel processed EEG continuously over regions of the brain. This technology uses quantitative EEG; it removes artifacts—both physiologic and environmental—as well.

EVOKED POTENTIALS

Evoked potentials (EP), measured from either the cortex or periphery, are generated in response to

some stimulus or behavior. This information is valuable for monitoring monitor the functional integrity of the ascending (sensory) or descending (motor) pathways.

How sensitive are evoked potentials to anesthesia?



In general, evoked potentials differ in their sensitivity to anesthetic agents depending on the neurologic pathways involved and the agent being used. The impact of anesthetic agents on neurophysiologic monitoring increases with the number of synapses in the pathway being monitored. This relationship arises because all anesthetic agents produce their effects by altering neuronal excitability via changes in synaptic function or axonal conduction.

Sensory Pathways: Ascending

Somatosensory Evoked Potentials

While best known for monitoring during spinal surgery, somatosensory evoked potentials (SSEP) have been used to monitor for ischemia in cortical tissue. They are used to determine the adequacy of collateral blood flow, tolerance of vessel occlusion, and tolerance of hypotensive technique during intracranial surgery. A change in SSEP cortical amplitude is the most sensitive indication of ischemia.

For SSEP monitoring, the stimulating electrodes are placed at a peripheral nerve while the response is recorded at the contralateral sensory cortex via scalp electrodes. *Because the pathway is afferent, it goes* ***into*** *the brain from the periphery.* Use of multiple recording sites allows for coverage along the entire neural axis, from peripheral stimulus to the primary somatosensory cortex.

The choice of electrode placement for recording evoked potentials from the scalp will be dictated by the site of stimulation (see Figure 3-2). The critical electrode for detecting the evoked potential after stimulation of the tibial nerve must be placed over the primary sensory cortex at the midline of the scalp. If the ulnar nerve is stimulated, the electrode must be placed over the primary sensory cortex, somewhat laterally from midline.

The integrity of the gracilis and cuneatus tracts of the posterior spinal cord is assessed by SSEP. If posterior cord or brain ischemia is present, transmission of action potentials through the posterior cord or brain will be diminished, thereby reducing the intensity and delaying the arrival of action potentials in reaching the cerebral cortex. Stimulation of a peripheral nerve results in an ascending volley of action potentials, which travels ipsilaterally via the fasiculus gracilis or cuneatus in the dorsal columns and first synapses at the dorsal column nuclei at the cervico-medullary junction. At this point the volley crosses the midline via the medial lemniscal pathway, traverses the brain stem, and synapses at the thalamus. From here, the volley ultimately synapses in the primary somatosensory cortex or post-central gyrus.

Noninvasive measurement of the brain stem's response to repetitive stimulation of a distal sensory peripheral nerve is possible with SSEP. This technology assesses the integrity of the sensory pathway in the posterior column of the spinal cord. SEEP monitoring measures sensory perception and does not ensure intact motor response. For SSEP monitoring to be useful, the surgery must place neural tissue at risk and options for intervention must exist.

The most common peripheral nerves monitored are the median (wrist), common peroneal (knee), or the posterior tibial (ankle). SSEP monitoring measures both latency (how long) and amplitude (how big) of the waveform. A change in SSEP cortical amplitude is the most sensitive indicator of ischemia or some interruption in the posterior

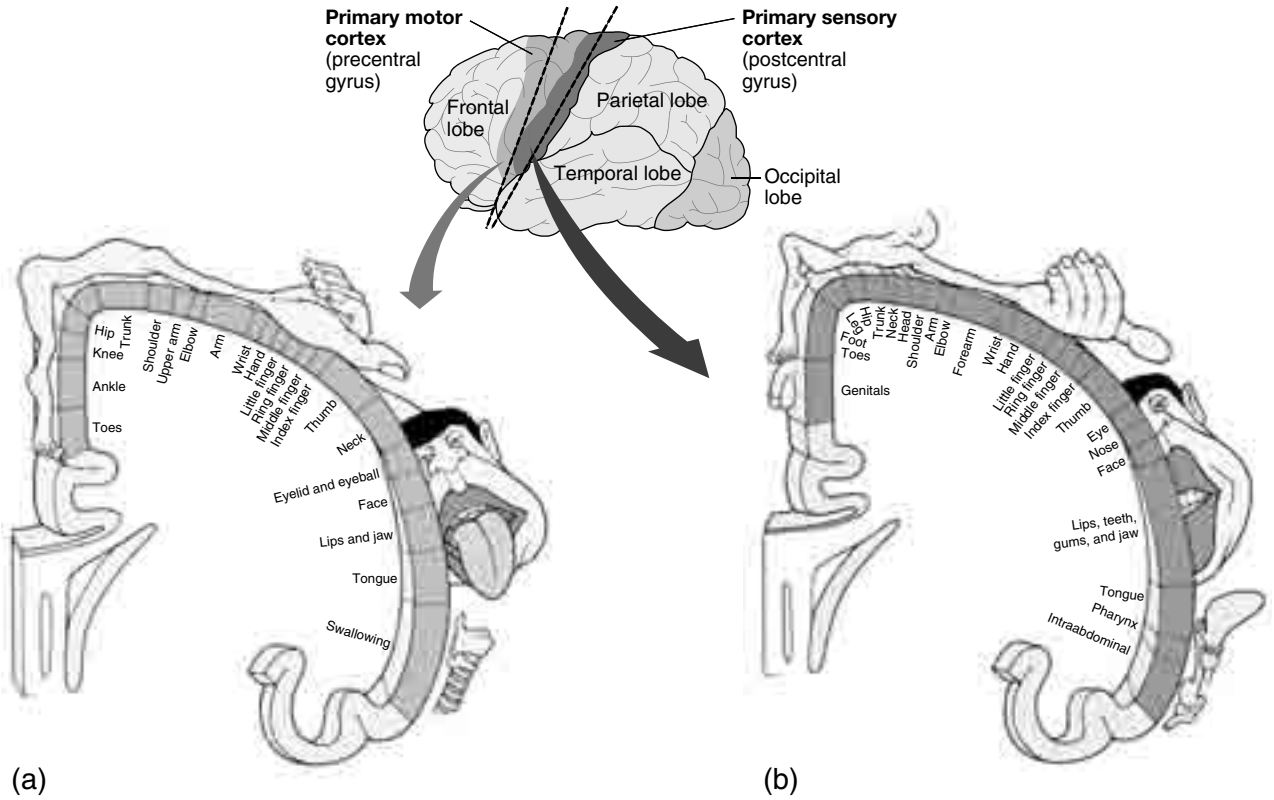


Figure 3-2 *Primary Motor (a) and Sensory (b) Areas of the Cerebrum*

column pathway. A greater than 50% decrease in amplitude or a greater than 10% increase in latency indicates an interruption of the posterior spinal cord pathway. Elevated ICP is associated with reductions in amplitude and increases in latency of cortically generated SSEPs; by virtue of its effect on cortical structures, this increase in ICP produces a pressure-related decrease in cortical SSEP responses.

SSEP monitoring is used to determine the adequacy of collateral blood flow, tolerance of vessel occlusion, and tolerance of hypotensive technique during intracranial surgery. While it is best known for its utility during spinal surgery (especially spinal fusion), this type of monitoring has also been used to check for ischemia in cortical tissue during surgery in the posterior fossae (especially that requiring retraction of the brain stem), during surgery near the primary somatosensory cortex and during supratentorial surgeries. Moreover, it is valuable in assessing lumbar and sacral nerve roots in posterior interbody fusion, lumbar fusion with instrumentation, and cauda equine surgeries; during carotid endarterectomy, scoliosis repair, aortic aneurysm repair, and spinal cord tumor surgeries; in cervical surgery with instrumentation; and in cardiac procedures where cardiopulmonary bypass is used.

Anesthetic Agents and SSEP Monitoring

It is essential to communicate changes in anesthesia level to neurophysiology staff. It is best to keep anesthesia at constant levels, especially during critical points of surgery. Neurophysiology personnel will

determine which agents may or may not be used and what their dose limits are.

SSEP monitoring offers intermediate sensitivity to anesthetics. In general, an anesthetic technique involving the use of a low-dose inhalational agent (at "half-MAC": minimum alveolar concentration.) along with a narcotic infusion and nitrous oxide at 66% with oxygen at 33% provides the most stable and optimal conditions for monitoring nerve function.

Volatile Anesthetics N_2O and inhaled anesthetics have the greatest effects to SSEP. Nevertheless, all anesthetic agents depress SSEP amplitude in dose-dependent manner, including nitrous oxide. This effect varies from isoflurane (most potent) to enflurane (intermediate potency) to halothane (least potent). Sevoflurane and desflurane have similar effects to isoflurane when the patient is at a steady state; however, because of their more rapid onset and offset of effect (both are more insoluble than isoflurane), they may appear to be more potent during periods when concentrations are increasing.

Good SSEP monitoring is compatible with 0.5–1 MAC isoflurane and 60% N_2O and oxygen, after which the waveforms are lost.

Systemic Factors

Blood pressure: Mean arterial pressures below the threshold for cerebral auto regulation via blood loss or drug effects result in progressive decreases in amplitude until loss of the waveform occurs (but no changes in latency). Hypotension considered "safe" combined with surgical manipulation can result in spinal cord ischemia.

Temperature: Hyperthermia leads to decreased amplitude of SSEPs, with loss of waves occurring at 42°C.

Blood gases/hematocrit: Hypoxia and severe drops in HCT have been reported to lead to decreased amplitude of SSEPs.

IV Agents

- Barbiturates cause dose-dependent reduction of SSEP amplitude and increased latency, but waveforms are still present at doses high enough to suppress the EEG.
- Propofol decreases SSEP amplitude and increases SSEP latency.
- Opioids cause dose-dependent decreased amplitude and increased latency. High-dose fentanyl (60 mcg/kg) is still compatible with SSEP monitoring. Morphine has effects similar to those of fentanyl. Avoid large boluses at moments of potential neural compromise. Meperidine can decrease or increase SSEP amplitude.
- Benzodiazepines also decrease SSEP amplitude. Midazolam has no effect on latency.
- Droperidol has varying effects on SSEPs but can decrease their amplitude.
- Drugs that can increase amplitude include etomidate (can be used to enhance SSEPs), ketamine, meperidine, and methohexital.
- Muscle relaxants have no effect on SSEPs. However, these agent cannot be used if the team is evaluating motor evoked potentials.

Brain Stem Auditory Evoked Responses

Brain stem auditory evoked responses (BAER), also known as brain stem auditory evoked potentials (BAEP), record data from the scalp that are generated in response to an auditory stimulus applied to the ear via an external or insert earphone. This placement monitors posterior fossa and brain stem activity for cranial nerve VIII (auditory). BAERs are generated in response to depolarization of the cochlear nerve and are processed almost entirely in the three divisions of the brain stem. Such a potential can arise secondary to compression or ischemia to the nerve or brain stem. BAERs are reflected as changes in both amplitude and latency in high-frequency deflections termed

Jewett waves. Each wave has a specific site of generation along the auditory axis and can provide the interpreter with information about specific sites of pathology.

BAERs are very resistant to (essentially unaffected by) anesthetics. BAER monitoring is a good choice for surgeries on posterior fossae or in which the brain stem at risk; it is also effective in cases involving acoustic neuroma, vestibular nerve resection, posterior fossae masses, vertebral-basilar aneurysms, brain stem lesions (meningioma), or posterior circulation aneurysms.

Visual Evoked Potentials

Visual evoked potentials (VEP) are recorded from the scalp in response to a visual stimulus provided in the form of either a flash or a changing checkerboard pattern. In the operating room, a flash stimulus is delivered via either a strobe lamp or fiber optic leads or goggles fitted very close to the patient's eyes. A potential is recorded from electrodes placed over the occipital cortex. Changes in latency, phase, and amplitude of the VEP can be used to assess the visual pathway integrity. The primary visual pathway consists of the retina (receptor), the optic nerves/tracts (pathway), and the pathway from the thalamus to the primary visual cortex.

VEPs are extremely (most) sensitive to anesthetic agents; they are obliterated with inhaled agents. VEP monitoring is useful for surgery near the optic pathway, especially during pituitary surgery; surgery targeting supra- and infra-sellar tumors; surgery focused on optic nerve tumors and decompression; surgery on occipital cortical masses; and pallidotomy, which involves destruction of part of the globus pallidus, a region of the brain involved with the control of movement.

Dermatome Evoked Potentials

Sensory evoked potentials can also be useful in evaluating a single segmental (dermatome) nerve root function; this technique focuses on dermatome evoked potentials (DEP), also known as trigeminal evoked potentials. Stimulation of a single dermatome results in the depolarization of a single nerve root and subsequent ascending conduction of a signal via the dorsal column pathways to the primary somatosensory cortex. SSEPs and other evoked potentials assess a mixed nerve root and do not provide information on individual nerve roots. Input to the spinal cord via, for example, the median nerve is distributed over several nerve root levels.

In DEP monitoring, electrodes are placed several centimeters apart within a single dermatome. The key problem with this technique is that overlap exists between dermatomes. Anatomical pathways and alarm criteria with regard to changes in DEPs are the same as with SSEPs.

Dermatome monitoring is used during surgery on lesions of specific nerve roots, brachial plexus explorations, and spina bifida/tethered cord releases.

Motor Pathways: Descending

Motor Evoked Potentials

Motor evoked potential (MEP) monitoring tests for the adequacy of perfusion of ventral spinal cord and the motor pathways. MEPs monitor activation of motor pathways (proximal to the surgical site) while recording motor responses (distal to the surgical site) from the arms and legs. They are recorded as nerve action potentials from peripheral nerves or from the distal musculature (compound muscle action potential). Once these potentials are activated, information flows to the ipsilateral thalamus and subsequently to lower motor neurons after crossing over at the level of the brain stem. These same pathways can be activated via stimulation of the brain stem or stimulation of the spinal cord directly.

MEP monitoring is used in procedures involving anterior approaches to the spinal cord, abdominal aortic aneurysms, scoliosis repair, intradural and

intramedullary spinal cord tumors, spinal decompression and fusion.

Anesthesia during MEP monitoring:

- MEPs recorded from the musculature are abolished easily by halogenated inhalational agents.
- Transcranially elicited MEPs are interfered with markedly by anesthetics.
- *Muscle relaxants cannot be used with MEP monitoring.* Muscle relaxation will reduce the ability to detect nerve irritation and quantify functional integrity.
- When recordable, MEPs may occur only at low concentrations (e.g., 0.2–0.5%).
- The effect of the inhalational agent is likely the result of depression of synaptic transmission either in the anterior horn cell synapses on motor neurons or in the cortex at the level of the interneuronal connections.
- As with the barbiturates, midazolam produces a prolonged, marked depression of MEPs.

ELECTROMYOGRAPHY

Electromyography (EMG) provides real-time information about the integrity of the cranial nerves and their underlying brain stem nuclei, the muscles innervated by cranial nerves, and the muscles innervated by spinal nerve roots. EMG monitoring is especially useful if there is any potential for inadvertent resection of cranial nerves V, VII, IX, and X during brain surgery. It is typically recorded by placing bipolar pairs of needle electrodes in the muscle groups of interest.

An alarm criterion for EMG recording is simply the presence of a signal. A baseline or "normal" situation is the absence of spontaneous muscle activity. Different grades of spontaneous activity results in different levels of alert (i.e., neurotonic discharges or injury potentials). Significant change is often unilateral and typically abrupt; it is not necessarily correlative to a particular surgical maneuver.

Neuromuscular junction stimulation is essential for EMG. Thus one cannot use this type of monitoring when muscle relaxants have been administered; one can use total intravenous anesthesia (TIVA) and inhalation at low doses, however. EMG monitoring is especially used for facial nerve and some spinal surgeries; it can be used in such cases to detect muscle and nerve disorders. Profound neuromuscular blockade will prevent recording of EMG activity during MEP recordings.

MEP cranial nerve monitoring can be used during procedures related to acoustic neuromas, microvascular decompression, posterior fossae tumors, facial nerve surgery, spinal surgery, skull base procedures, and ENT procedures (tympanomastoidectomy, cochlear implants). It is also useful for peripheral nerve explorations, including brachial plexus explorations, sciatic nerve explorations, and lumbosacral explorations and fusions (e.g., pedicle screw fusions, tethered cord releases, dorsal rhizotomy, and interbody cage fusions).

WAKE-UP TEST

The wake-up test is intended to measure the patient's motor function. The surgeon will indicate when a patient should be awakened during surgery. The patient will be asked to wiggle his or her toes on command upon wakening from anesthesia and will then be put back to sleep.

Desflurane, which offers a quick wake-up profile, is a volatile anesthesia option that is commonly used in today's OR. If nerve paralysis is used, it is allowed to resolve before wake-up occurs. Use of small amounts of propofol and a narcotic can help keep the patient sleepy (and not coughing and bucking), yet still able to follow commands. Dexmedetomidine is also helpful in that patients remain sedated but easily awaken to voice commands.

It is important for patients to have thorough preoperative teaching and explanation to prepare them for having a "wake-up test." The patient should not be in severe pain and will be immediately

put back under general anesthesia after checking motor function.

ANESTHETICS AND BODY STATES INFLUENCING INTRAOPERATIVE NEUROLOGIC MONITORING

The effects of anesthetic agents result from one of two mechanisms of action:

- Inhibition of synaptic pathways
- Indirect action on pathways by changing the balance of inhibitory and excitatory influences

While most anesthetics depress evoked response amplitude and increase latency, some anesthetic agents (etomidate, methohexital, ketamine) enhance both SSEP and MEP amplitudes. This phenomenon is thought to occur via a mechanism whereby inhibition is attenuated.

Intravenous induction and sedation agents interact at a number of different receptors. For example, barbiturates, etomidate, propofol, and benzodiazepines primarily act by enhancing the inhibitory effects of gamma-aminobutyric acid (GABA). Binding and activation of the GABA-a receptor results in an increase in chloride conductance and a subsequent hyperpolarization resulting in synaptic inhibition.

Some intravenous agents work by blocking the excitatory effects of glutamate via antagonism of a variety of receptor subtypes: NMDA, kainate, and quisqualate. For example, ketamine appears to have its major action by inhibiting the NMDA receptor and subsequently reducing sodium flux and intracellular calcium levels. Other intravenous anesthetic agents activate opioid receptors (mu, kappa, and delta).

Ketamine can enhance cortical SSEP amplitude and MEP amplitude. Its effects on subcortical and peripheral SSEP responses are minimal. Although this agent is ideal for cases involving intraoperative monitoring, increases in intracranial pressure are associated with ketamine, and this medication can produce hallucinations in some patients.

Thiopental, a popular barbiturate induction agent, reasults in a transient decrease in amplitude and an increase in latency of cortical responses occurring immediately after induction. Minimal effects are seen on the subcortical and peripheral responses.

MEPs are unusually sensitive to barbiturates, with a prolonged effect being observed when these agents are used. *Methohexital* is the exception: It has been found to increase the amplitudes of cortical SSEPs.

Midazolam, a benzodiazepine, has desirable properties of amnesia. When administered at moderate doses, it produces a mild depression of cortical SSEPs. Like the barbiturates, midazolam produces a prolonged, marked depression of MEPs.

Like ketamine, *etomidate* increases the amplitude of cortical SSEP components and increases the amplitude of MEPs.

Like thiopental, *propofol* produces amplitude depression in cortical SSEPs with rapid recovery after termination of infusion due to rapid metabolism. MEPs have demonstrated a similar depressive effect on response amplitude.

The *neuromuscular blocking agents* act at the acetylcholine receptors found at the neuromuscular junction. Because muscle relaxants exert the majority of their action at the neuromuscular junction, they have little effect on electrophysiologic recordings such as SSEPs, which are not derived from muscle activity. However, profound neuromuscular blockade will prevent recording of EMG activity during MEP recordings. Partial neuromuscular blockade has the benefit of reducing a substantial portion of patient movement and facilitates surgical procedures when muscle relaxation is needed for retraction of tissues.

Nitrous oxide reduces SSEP cortical amplitude and increases latency when used alone or when combined with halogenated inhalational agents or opioid agents. When anesthetic agents are

compared at equipotent concentrations, nitrous oxide produces more profound changes in cortical SSEPs and MEPs than any other inhalational agent.

Opioids depress electro excitability by increasing inward potassium ion (K^+) current and depressing outward sodium ion current via a G-protein mechanism linking the receptors to ion channels. The effects of opioid analgesics (e.g., alfentanil, fentanyl, remifentanil, sufentanil) on SSEPs and MEPs are weaker than those associated with inhalational agents. The opioids produce minimal changes in spinal or subcortical SSEP recordings, although there is some depression of amplitude and an increase of latency in the cortical responses. Spinal application of morphine or fentanyl for postoperative pain management produces minimal changes in SSEPs and MEPs.

Blood Flow

Local factors may produce regional ischemia not predicted by systemic blood pressure. For example, during spinal surgery, the effects of hypotension may be aggravated by spinal distraction, such that an acceptable limit of systemic hypotension cannot be determined without monitoring. Regional effects include peripheral nerve ischemia from positioning, tourniquets, or vascular interruption secondary to vasospasm. MEPs and SSEPs are both sensitive to spinal cord events produced by vascular ischemia (carotid cross-clamping) or mechanical compression. These types of potentials may show differential sensitivity to ischemic events.

Intracranial Pressure

Elevated ICP is associated with reductions in amplitude and increases in latency of SSEPs, leading to a loss of brain stem responses with uncal herniation. For MEPs, a gradual increase in the onset latency occurs with a gradual complete abolishment of responses.

Blood Rheology

Changes in hematocrit can alter both oxygen-carrying capacity and blood viscosity. Maximum oxygen delivery is thought to occur with a midrange hematocrit (30–32%). Evoked response changes with hematocrit are consistent within this optimal range.

Ventilation/Oxygenation

Hypoxemia can result in evoked potential deterioration before other clinical parameters show any changes. Alterations in carbon dioxide levels are known to affect spinal cord and cortical blood flow. Remarkable SSEP changes occur when the CO_2 tension is extremely low, suggesting that excessive vasoconstriction may produce ischemia (< 20 mm Hg).

Temperature

SSEP and MEP changes can be observed with hypothermia. These changes are consistent with those seen with ischemia and anesthesia, in that they are more significant in the cortically recorded responses as opposed to the subcortical potentials. MEPs exhibit a gradual increase in onset latency due to slowed conduction time along the neural axis. An increase in activation threshold latencies is also seen.

POSTOPERATIVE VISUAL LOSS

No single factor has been identified as a cause of postoperative visual loss (POVL), although several risk factors are commonly observed:

- Eye pressure (small risk)
- Elevated central venous pressure from retarded drainage from ophthalmic veins
- Hypotension, hypovolemia: may be partially causative
- Increased intraocular pressure (IOP):
  - In the prone position the risk of POVL increases dramatically if: surgery lasts

more than 5 hours and if in a prolonged head down (Trendelenburg) position causing decreased venous outflow from cranium.
    - With administration of increased amounts of IV fluids
    - With increased intra-abdominal pressure (increases epidural vein pressure)
- Increased serum blood glucose levels: affects neurons
- Hypothermia: a decreased temperature increases the viscosity of blood, decreasing blood flow to the eye.
- Repeat spine surgery in the prone position.
- Preoperative donation of blood; hematocrit low; anemic patient.
- Smoking, obesity

Preoperative/Anesthetic Management

- Blood replacement should be timely to maintain hematocrit.
- IV fluid balance should be maintained carefully. Infusion of colloid is an alternative to infusion of multiple liters of crystalloid. Avoid hemodilution.
- Keep the patient's head at or above the level of the heart.
- Avoid abdominal compression.
- Keep blood glucose within "tight control": serum blood glucose 80–120 mg/dL.
- Pad the eyes
- Urinary output should be maintained at a rate of 1 mL/kg/h or more.
- Conduct surgery in the minimal amount of time when the patient is in the prone position.
- Maintain an adequate mean arterial blood pressure; maintain adequate perfusion pressure to the optic nerve.

PULMONARY HYPERTENSION

Normal pulmonary circulation is a high-flow, low-pressure circuit. Progressive increases in pulmonary vascular resistance (PVR) initially result in right ventricular hypertrophy (RVH) but will eventually lead to right heart failure, defined as pulmonary artery pressures that exceed 30/10 mm Hg. Pulmonary hypertension may be caused by obstruction of the vascular bed, pulmonary vasoconstriction, chronic hypoxia, collagen vascular disease, sickle cell disease, congenital heart disease, portal hypertension, high altitude, allergic alveolitis, or acidosis.

Treatment

- Identify and treat the underlying cause.
- Reduce vascular tone.
- Optimize right ventricular function.

Anesthesia Management if the Patient Has Pulmonary Hypertension

- Pre-oxygenation is crucial, as the patient will have a decreased FRC. A decreased FRC subjects the patient to rapid oxygen desaturation and hypoxemia.
- Inspired oxygen concentration may be lowered to maintain a SpO_2 greater than 91% to reduce the risk of O_2 toxicity.
- Avoid barotrauma with mechanical ventilation; ventilation should be maintained with decreased tidal volumes and increased respiratory rate.

TOURNIQUET ISSUES

The primary objective of tourniquet use in surgical settings is the creation of a bloodless operative field; this is achieved by applying circumferential pressure on the arterial and venous circulation. In certain situations, tourniquets may be useful for preventing the undesirable escape of vascular fluids into body areas or to confine local anesthetics to an extremity. Orthopedics and plastic surgery are two specialties that frequently utilize pneumatic tourniquets.

Prior to Tourniquet Inflation

Check to make sure the antibiotic has been given before the Esmarch bandage and the tourniquet are applied. An adhesive occlusive dressing should be applied over any open skin before padding and the tourniquet are applied. Wrapping the skin (e.g., with cuff padding, cast padding, or soft cotton wrap) to protect it from the tourniquet itself helps to prevent damage to the skin and subcutaneous tissue.

Multiple sizes of tourniquets should be available to ensure an adequate size to encircle the limb with enough overlap in the cuff—minimum 3 inches and maximum 6 inches. The overlap itself should lie on the outside of the extremity to avoid nerve sheath compression. Palpate the patient's distal extremity pulses before inflation to assess baseline pulses.

The cuff should be positioned over the largest amount of soft tissue. The cuff should not apply pressure to the bend of the elbow or of the knee when inflated.

Elevate the operative extremity to drain blood passively, and then wrap extremity tightly with an Esmarch bandage (or equivalent).

Tourniquet Inflation

It is the surgeon's responsibility to dictate the tourniquet pressure setting. The setting is based on the patient's systolic blood pressure, age, and limb size. Usually, the inflation setting is 50–100 mm Hg greater than the patient's systolic pressure. The inflation pressure maximum is usually 300 mm Hg in the upper extremity and 400 mm Hg in the lower extremity.

Over-pressurization may cause pain at the tourniquet cuff site; muscle weakness; compression injuries to blood vessels, nerve, muscle, or skin; or extremity paralysis. *Under-pressurization* may result in blood in the surgical field, passive congestion of the limb, shock, and hemorrhagic infiltration of a nerve.

- Pale coloring is indicative of adequate exsanguination.
- Extensive mottling is indicative of inadequate exsanguination.

Tourniquet Pain

After inflation of the pneumatic tourniquet for 30–60 minutes, patients may experience "tourniquet pain," accompanied by an increase in heart rate and blood pressure. Pain should be assessed and managed. A sympathetic response can occur even when the patient is under general anesthesia. Tourniquet pain frequency occurs, with intensity depending on the type of anesthesia administered: IV regional > epidural > spinal > general anesthesia.

Treatment of tourniquet pain includes regional analgesics, opioids, hypnotics for a patient under local anesthesia, and change in sedation technique. Local hypothermia appears to be a safe and effective method of decreasing the adverse effects of tourniquet ischemia and allowing continuous tourniquet inflation time to extend safely beyond the customary 2-hour limit.

While the tourniquet is inflated, be mindful of how much narcotic is administered for tourniquet pain. As soon as the tourniquet is deflated, this noxious stimulus will be removed and presence of excessive narcotic may affect the return of spontaneous ventilation for patients under general anesthesia.

Tourniquet Time: Safe Duration

Tourniquets may be applied for a maximum of 2 hours. Tourniquets have been used up to 4 hours with intermittent deflation of the cuff. The *minimum time of application should be 20 minutes* to prevent release of the local anesthetic into the general circulation.

Damage from tourniquet use can affect the extremity vessels, nerves, or muscle. Direct cuff pressure can cause ischemia with muscle dysfunction. Any damage to vessels, nerves, and skeletal

muscle is usually reversible for tourniquet inflations of 1–2 hours.

Deflation of the Tourniquet

When the tourniquet is deflated, a decrease in blood pressure occurs as blood is shunted to the extremity. Products of anaerobic metabolism and newly released acid metabolites enter the circulation upon deflation of the tourniquet cuff, causing transient increases in end-tidal carbon dioxide, metabolic acidosis, and a decrease in oxygen saturation. The time for clearance of the metabolites depends on the patient's physiologic status, the extremity involved, and the duration of tourniquet inflation.