Abstract

The critical-care patient requiring anesthesia is at high risk for life-threatening complications during the perianesthesia period. Preoperative stabilization can be difficult, and the decision to induce anesthesia can be guided by goal-directed therapies. During the perioperative period, close monitoring of vital parameters will guide clinical interventions. The recovery period is just as critical as the induction period, and diligent monitoring and supportive care during that time should not be undervalued.
Anesthetizing the critical-care patient can be a daunting task for the veterinary nurse anesthetist. Balancing an appropriate surgical anesthetic plane with the inherent cardiopulmonary depressive effects of injectable and inhalant anesthetics can be clinically challenging. This article provides tips for how to use a balanced multimodal approach to support the critical-care patient through the pre-, peri-, and postoperative periods.

**PREOPERATIVE PERIOD**

Deciding when to anesthetize a critically ill patient is subjective and will depend on the patient’s response to stabilization attempts. Stabilizing the critical-care patient is similar to stabilizing the systemic inflammatory response syndrome (SIRS)/sepsis patient. Goal-directed therapy should be used to tailor treatments to the patient’s clinical needs (**TABLE 1**).

**Fluid Therapy**

Critical-care patients benefit from early fluid resuscitation because of hypovolemia, dehydration, and hemostatic dysregulation leading to reduced oxygen delivery to the tissues. Early treatment

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**Take-Home Points**

- Preoperative patient stabilization will help set up the anesthetist for perioperative success.
- When possible, vascular access (e.g., peripheral IV catheters, multilumen central lines, arterial catheters) should be obtained before anesthesia is induced.
- Large-volume fluid resuscitation is detrimental in the long term; thus, balanced fluid support includes use of blood products and vasopressor therapy.
- Monitoring equipment (e.g., electrocardiography equipment, pulse oximeter, blood pressure cuff) should be used during the induction period.
- Following a balanced multimodal anesthetic plan will reduce the need for inhalant anesthetics and subsequently limit cardiovascular depression.
- Vasopressor therapy may be needed to maintain a mean arterial pressure of >60 mm Hg.
- Ventilatory support (i.e., positive-pressure ventilation) may be needed.
- Recovery is a critical period, and supportive care and monitoring should continue while the patient is moved out of the surgical suite.
with crystalloid fluids, specifically goal-driven administration via small-volume resuscitation, can aid in oxygen delivery and arterial blood pressure management. Small-volume resuscitation is based on the concept that fluid therapy is driven by reaching clinical goals. Example goals include decreased heart rate in the tachycardic patient or a mean arterial blood pressure (MAP) of >60 mm Hg in the hypotensive patient. For small-volume resuscitation, isotonic crystalloid fluid boluses of 5 to 10 mL/kg are administered while carefully monitoring for response to fluid therapy.

Intravascular longevity of crystalloid fluids is short; only an estimated 70% to 80% remains in the vasculature after 30 minutes; thus, the response of severely ill patients to initial crystalloid therapy may be poor or short-lived. However, care should be taken to avoid large-volume resuscitation as continued administration of fluid boluses can damage the endothelial glycocalyx (endothelial cell lining) and lead to vascular leakage and edema formation. Use of hypertonic crystalloids (e.g., hypertonic saline) may promote an osmotic shift of fluid from the interstitial space into the intravascular space, with effects lasting 2 to 4 hours; continued isotonic fluid therapy will be needed to prevent interstitial dehydration.

Blood Product Administration
Blood products serve a multitude of purposes, including treating anemia and hypoproteinemia, acting as a natural colloid prolonging intravascular volume, and treating coagulopathies. Fast preoperative administration of packed red blood cells (pRBCs) to anemic patients, to maintain a packed cell volume (PCV) greater than 20%, is preferable but not always attainable in very large canine patients or in feline patients when pRBC units are a fixed volume. Preoperatively, patients with a severe coagulopathy should receive a transfusion of fresh frozen plasma (FFP), often administered as a slow bolus to critical-care patients, depending on volume status.

### BOX 1

**Diluted Vasopressor Calculations**

- **Patient weight:** 25 kg
- **Drug:** Dobutamine 12.5 mg/mL
- **Dose:** 10 µg/kg/min running at 10 mL/hr for 2 hr

**Calculations**

1. \( \frac{(25 \text{ kg} \times 10 \text{ µg/kg/min} \times 60 \text{ min/hr})}{1000 \text{ µg/mg}} = 15 \text{ mg/hr dobutamine} \)
2. \( \frac{15 \text{ mg/hr}}{12.5 \text{ mg/mL}} = 1.2 \text{ mL/hr dobutamine} \)
3. \( 1.2 \text{ mL/hr} \times 2 \text{ hr} = 2.4 \text{ mL total dobutamine} \)
4. \( 2 \text{ hr} \times 10 \text{ mL/hr} = 20 \text{ mL diluent} - 2.4 \text{ mL dobutamine} = 17.6 \text{ mL diluent} \)

### Table 1: Common Treatments for Stabilizing Critical-Care Patients

<table>
<thead>
<tr>
<th>THERAPY</th>
<th>RECOMMENDED DOSAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FLUIDS</strong></td>
<td></td>
</tr>
<tr>
<td>Crystalloid fluid bolus</td>
<td>5–10 mL/kg over 5–15 min</td>
</tr>
<tr>
<td>Hypertonic saline</td>
<td>3–5 mL/kg over 15 min</td>
</tr>
<tr>
<td>Dextrose</td>
<td>0.5–1 mL/kg over 10–15 min</td>
</tr>
<tr>
<td><strong>BLOOD PRODUCTS</strong></td>
<td></td>
</tr>
<tr>
<td>pRBC</td>
<td>10–20 mL/kg over 1–4 hr</td>
</tr>
<tr>
<td>FFP (for volume)</td>
<td>1–2+ mL/kg over 1+ hr</td>
</tr>
<tr>
<td>FFP (for coagulation factors)</td>
<td>5–10 mL/kg over 1–4 hr</td>
</tr>
<tr>
<td><strong>VASOPRESSORS</strong></td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>5–15 µg/kg/min</td>
</tr>
<tr>
<td>Dopamine</td>
<td>1–10 µg/kg/min</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>0.2–1 µg/kg/min</td>
</tr>
</tbody>
</table>

*FFP = fresh frozen plasma; pRBC = packed red blood cells*
The author prefers to use FFP as a natural colloid for SIRS/sepsis patients to maintain intravascular volume and correct hypotension secondary to hypovolemia. Studies of humans indicate that administration of plasma reduces proinflammatory mediators and endothelial glycocalyx degradation in patients with sepsis. FFP may be administered to septic patients as a small-volume bolus of 1 to 2 mL/kg, followed by a continuous-rate infusion (CRI) at 1 to 2+ mL/kg.

Vasoactive Drug Support
Early use of vasopressors reduces death rates. Patients should be euhydrated; however, for critical-care patients, that is not always achievable. Choice of vasoactive drug should be tailored to the desired clinical response; however, for the critical-care patient, use is primarily geared toward increasing arterial blood pressure.

Vasoactive drugs act primarily on 4 adrenergic receptors: β1 receptor agonists, which increase heart rate and contractility; β2 receptor agonists, which cause vasodilation; and α1 and α2 receptor agonists, which cause vasoconstriction. Vasopressors pose a risk for phlebitis if administered perivascularly and thus should always be diluted. Diluent options include sodium chloride or dextrose 5% in water.

Dobutamine
Dobutamine has strong β1 effects along with some β2 and α1 effects (strong changes to heart rate and contractility and thus cardiac output). Dobutamine is used primarily for patients that have poor cardiac output but do not need strong augmentation of blood pressure. An example of a critical-care patient that may benefit from dobutamine is one with uncontrolled dilated cardiomyopathy needing to undergo anesthesia. In cats, dobutamine should be used with caution and at lower doses (5 µg/kg/min) as it can cause seizures.

Dopamine
Dopamine is a precursor to norepinephrine and has strong β and α activity (e.g., stimulating the release of norepinephrine). It also uniquely has dopamine receptor activity, which causes arterial vasodilation. The effects of dopamine are dose dependent. Low doses (1 to 3 µg/kg/min) lead primarily to dopaminergic arterial vasodilation; moderate doses (5 to 10 µg/kg/min) lead primarily to the β effects of increased heart rate and contractility; and high doses (>15 µg/kg/min) lead primarily to the α effects of vasoconstriction.

Norepinephrine
Norepinephrine has primarily α effects leading to vasoconstriction and may variably have weak β1 activity leading to changes in heart rate and contractility, which depend on patient status. Because hypovolemic animals tend to already be vasoconstricted, further vasoconstriction may decrease venous return and thus cardiac output. Norepinephrine may be used with
dopamine if both vasoconstriction and strong inotropic effects are required.\(^9\) For SIRS/sepsis patients, norepinephrine is the vasopressor of choice.\(^10\)

**Epinephrine**

Epinephrine is a strong $\beta$ and $\alpha$ agonist, causing profound vasoconstriction and strong changes in heart rate and contractility and thus in cardiac output. Unfortunately, epinephrine can also cause unwanted negative side effects (e.g., sinus tachycardia, ventricular arrhythmias, hyperlactatemia).\(^8\) Clinical use of epinephrine is primarily reserved for use when alternative vasopressor therapy has failed.

**Vascular Access**

Critical-care patients require advanced vascular access because they often require multiple CRIs and undergo repeated blood sampling. The author’s guideline is American Society of Anesthesiologists (ASA, asahq.org) status minus 1 for vascular access sites (with a minimum of 1). Before anesthesia induction, ASA 5 patients should undergo placement of a multilumen central venous catheter, if clinically possible. Central multilumen catheters provide the ability to run multiple infusions at once into different lumens, administer hyperosmolar solutions (e.g., high percentage dextrose) with decreased risk for phlebitis, and collect serial blood samples (FIGURE 1). Vascular access in critical-care patients can be extremely difficult, and difficulty may increase after administration of vasopressors.

**Venous Access**

Common venous access sites in canine and feline patients include the cephalic and cephalic accessory veins, the medial and lateral saphenous veins, and the jugular veins. Uncommon venous access sites for patients of both species include the dorsal pedal veins; in dogs, unusual sites include the auricular vein (conformation dependent) and the dorsal metacarpal veins.

**Blood Sampling**

Improper sampling technique can lead to fatal clinical decisions; thus, correct sampling technique with a multilumen central catheter is paramount.\(^3\) Because of the risk of falsely elevated blood glucose levels when administering high-percentage dextrose dilutions, 1 lumen should not be used for dextrose infusions, if possible, and should be reserved strictly for sampling. In addition, during sampling, all dextrose infusions should be paused to prevent incorrect measurements of glucose concentrations.

**Arterial Access Sites**

Depending on predisposing risk factors, critically ill anesthesia patients may benefit from arterial catheter placement for monitoring invasive blood pressures and collecting serial arterial blood samples. Although

**FIGURE 2.** (A) Lingual arterial catheter and (B) invasive blood pressure displayed on monitor in a critical-care patient.
technically challenging, particularly in critical-care patients with preexisting hypotension, the ability to have direct, real-time arterial blood pressure measurements and to assess and monitor ventilation/oxygenation status will help guide more rapid clinical decisions. Typical placement sites include the metatarsal, coccygeal, auricular, radial, and lingual arteries (the downside of lingual artery placement is that the catheter will need to be removed before recovery) (FIGURE 2). Placement of an arterial catheter before anesthesia induction can be attempted but, depending on the patient and placement site, may not be tolerated. Attempts may be repeated after induction, as clinically necessary. For arterial catheters in cats, care should be taken to remove them as soon as clinically possible as they can potentially lead to increased ischemic injury at the catheter site.\(^1\)

Anesthesia Planning
Anesthesia of critical-care patients should be multimodal (a balance of different drugs with different mechanisms of action administered by various routes). Drug selection will vary from case to case but should be patient-specific. Examples include preexisting renal or hepatic disease limiting medication clearance (e.g., renal metabolism of ketamine in the cat) and hepatic clearance of midazolam in patients with liver dysfunction.\(^2\) Concurrent drug therapy should also be considered (e.g., recent administration of steroids and nonsteroidal anti-inflammatory drugs). In general, inhalant anesthetics cause significant cardiovascular depression, and use in critical-care patients should be limited; multimodal anesthesia should be used instead. Species-specific issues should be considered (e.g., infusions of propofol injectable emulsion in cats and their intolerance of benzyl alcohol, the use of lidocaine in cats and negative cardiovascular effects).\(^3\) When using inhalant anesthetics for critical-care patients, keep in mind that minimum alveolar concentration is decreased when CRIIs are concurrently administered.

Surgical Suite and Patient Preparation

Suite Preparation
Before the patient undergoes anesthesia induction, the surgical suite should be completely prepared to reduce total anesthetic time as well as the possibility of distraction during the critical perianesthetic period. Preparation should include readying the sterile table and having all potential surgical equipment available in the room, leak testing and setting up the anesthesia machine and monitoring equipment, and setting up quick access to potential emergency drugs. For critical-care patients, careful forethought can help reduce the likelihood of treatment delays. Potentially needed blood products and administration pumps, fluid/syringe pumps, extension sets, and Y-ports should be readily available; for severe cases, a dose of emergency drugs may even be predrawn. If a surgical ventilator is available, it should be outfitted with the correct size bellows and be quickly accessible in the event of apnea.

Patient Preparation
For patients with known or suspected gastric distension, preplacement of a nasogastric tube to allow for gastric emptying may reduce the risk for life-threatening aspiration during induction.

Anesthesia Induction
The most critical time during the perioperative anesthetic period is induction. Administration of premedication and induction drugs diminishes the patient’s ability to continue the compensatory mechanisms that are responding to critical disease processes. There is no safe induction combination, and
the patient should be monitored and supported very closely during induction.

**Preoxygenation**
Before anesthesia induction, critical-care patients should be preoxygenated for 5 to 10 minutes, particularly brachycephalic breeds and patients that may require a prolonged intubation time (e.g., cats). Preoxygenation increases oxygen reserves to reduce the risk for hypoxemia during the induction period.

**Monitoring**
During induction, the critical-care patient requires intensive monitoring and support. In routine clinical practice, the standard order of procedures is often inducing anesthesia, positioning the patient for preparation of the surgical site, and then applying monitoring equipment. However, for the critical-care patient, monitoring equipment (e.g., electrocardiography [ECG] leads, a pulse oximeter, a blood pressure cuff) should be placed before induction and an initial reading taken or direct arterial monitoring should be initiated. A recent minimum database, performed preferably in the past 10 to 15 minutes, should be available. A minimum database includes a PCV, total protein (TP), and blood glucose and lactate levels. Before induction begins, hypoglycemia should be treated with 0.5 to 1 mL/kg of 50% dextrose diluted 1:3, and patients with persistent hypoglycemia should be evaluated for the need of 2.5+% dextrose supplementation in their maintenance fluids. PCV/TP readings can help drive clinical decisions on fluid balance during the perioperative period because patients with a high PCV/TP level are probably dehydrated and may be more responsive to fluid therapy to correct hypotension. Elevated blood lactate levels indicate anaerobic metabolism and thus continued poor oxygen delivery.

**Induction Agents**
Common induction medication choices include premedication with a pure µ opioid and a benzodiazepine (e.g., methadone, fentanyl, midazolam) (**TABLE 2**). Pure µ opioids allow for optimal pain control, and benzodiazepines assist with sedation. That combination, known as neuroleptanalgesia (the combination of an opioid with an anxiolytic), may be adequate for intubating the critical-care patient. If additional sedation is needed to achieve swift intubation, a titratable induction agent (e.g., propofol, alfaxalone) may be necessary. Propofol induces anesthesia when titrated over 60 to 90 seconds, and its effect lasts approximately 10 minutes. Negative side effects, including dose-dependent apnea and transient vasodilatory hypotension, may be more prevalent in the critical-care patient. Alfaxalone, like propofol, induces anesthesia with slow titration over 1 to 2 minutes; clinical effects last approximately 10 minutes. Previous research suggested that alfaxalone caused less respiratory depression than propofol, although recent research has shown a similar degree of respiratory depression. Clinically, both alfaxalone and propofol cause dose-dependent apnea and cardiovascular depression.

**Induction Follow-up**
After intubation, positioning the patient in the surgical

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**TABLE 2** Common Induction Drug Dosages

<table>
<thead>
<tr>
<th>DRUG</th>
<th>DOSAGE</th>
</tr>
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<tbody>
<tr>
<td><strong>BENZODIAZEPINES</strong></td>
<td></td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.2 mg/kg IV</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.2 mg/kg IV</td>
</tr>
<tr>
<td><strong>OPIOIDS</strong></td>
<td></td>
</tr>
<tr>
<td>Methadone</td>
<td>0.2 mg/kg IV</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>5 µg/kg IV</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.1 mg/kg IV</td>
</tr>
<tr>
<td><strong>INDUCTION</strong></td>
<td></td>
</tr>
<tr>
<td>Propofol</td>
<td>3–5 mg/kg IV, titrated</td>
</tr>
<tr>
<td>Alfaxalone</td>
<td>1–3 mg/kg IV, titrated</td>
</tr>
</tbody>
</table>

**TABLE 3 Dosages for Perioperative Constant-Rate Infusions to Reduce Inhalant Anesthetics for Critical-Care Patients**

<table>
<thead>
<tr>
<th>INJECTABLE ANESTHETIC</th>
<th>DOSAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fentanyl</td>
<td>5–20 µg/kg/hr</td>
</tr>
<tr>
<td>Methadone</td>
<td>0.05–0.2 mg/kg/hr</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.1–0.3 mg/kg/hr</td>
</tr>
<tr>
<td>Ketamine</td>
<td>2–20 µg/kg/min</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>For pain control 20–50 µg/kg/min</td>
</tr>
<tr>
<td></td>
<td>For ventricular arrhythmias 50–80 µg/kg/min</td>
</tr>
<tr>
<td></td>
<td>Propofol 200–400 µg/kg/min</td>
</tr>
<tr>
<td></td>
<td>Alfaxalone 6–9 mg/kg/hr</td>
</tr>
</tbody>
</table>
suite and complete surgical preparation can follow. Ventilation should be carefully monitored as critical-care patients may require ventilatory support with positive-pressure ventilation (PPV) after induction. Additional monitoring equipment should be swiftly placed, including a capnograph for end-tidal carbon dioxide (ETCO₂) monitoring. Ideally, an assistant can help during the preparation period so the anesthetist can focus on patient monitoring.

PERIOPERATIVE PERIOD

Anesthetic Plane Maintenance
Maintaining anesthesia should be focused on keeping the patient in a light surgical plane and quickly troubleshooting any changes in vital parameters. Although general anesthesia is commonly maintained with inhalants, use of a balanced multimodal protocol should decrease cardiovascular depression. CRIs should be used to reduce anesthetic gas requirements (TABLE 3). CRIs that control analgesia include opioids (e.g., fentanyl, methadone) and lidocaine (in the canine patient). Ketamine, a dissociative drug, is an excellent anesthesia supplement for reducing the need for cardiodepressive anesthetics in dogs and cats.¹⁷ Use of CRIs will allow for reduced concentrations, and potentially elimination, of inhalant anesthetics, as well as titratable management of negative side effects (e.g., anesthesia-induced hypotension). The author prefers to use multimodal total intravenous anesthesia for critical-care patients (FIGURE 3).

Anesthesia Monitoring
Monitoring should include ECG, pulse oximetry, blood pressure, capnography, and temperature measurements. For respiratory-compromised patients, arterial blood gas monitoring may be helpful; trends and abrupt changes will alert the anesthetist to changes in patient stability and the need for swift intervention.

Electrocardiography

Ventricular Tachycardia
Critically ill canine patients are at risk for sudden development of fatal arrhythmias. Ventricular tachycardia requires quick intervention with an appropriate antiarrhythmic (e.g., 2 mg/kg lidocaine bolus).

Ventricular Arrhythmias
Ventricular arrhythmias associated with hypotension should also be addressed; a lidocaine CRI may help control this arrhythmia while also providing analgesia and free radical scavenging.

Premature Ventricular Contractions
Premature ventricular contractions are common in the critical-care patient but, unless associated with tachycardia or hypotension, do not always require intervention.

Tachycardia
Tachycardia in the critical-care patient may be secondary to inappropriate anesthetic depth or analgesic control. Thus, care should be taken to prevent deep anesthetic planes. In addition, sinus tachycardia may be a compensatory response to hypovolemia and hypotension, which should be corrected with fluid therapy and blood product replacement.
Bradyarrhythmias
Bradyarrhythmias are more common in feline patients and should be addressed with an anticholinergic (e.g., glycopyrrolate, atropine). Glycopyrrolate (0.011 mg/kg IV) has a slower onset but longer duration of action and can be used to address mild bradycardia. Atropine (0.02 to 0.04 mg/kg IV) has a faster onset and shorter duration of action and should be used for critical emergency bradycardia.\textsuperscript{18}

Pulse Oximetry
For patients receiving 100% oxygen, the pulse oximetry reading should be 100%. However, pulse oximetry is often inaccurate in the critical-care patient due to weak pulses and hypotension. Because of inaccuracies of pulse oximeters in the critical-care patients, the anesthetist should also monitor oxygen delivery parameters (e.g., mucous membrane color, capillary refill time). Even in the event of inaccurate readings, the pulse oximeter can be used to monitor response to therapy. Identifying trends in the critical-care patient may be beneficial as abrupt changes in the pulse oximetry readings may indicate imminent cardiac arrest or severe hypotension.

Blood Pressure Monitoring
Hypotension in anesthetized critical-care patients is common. Blood pressure should be monitored closely, and when needed, MAP should be swiftly restored to >60 mm Hg. Fluid boluses of 5 mL/kg may be administered over a 5-minute period to determine whether hypotension is responsive to fluid therapy (and thus secondary to hypovolemia). If normotension is achieved but short-lived after a fluid bolus, higher fluid rates may be beneficial. A dose of hypertonic saline may be administered to draw fluids into the intravascular space from the interstitial space. Colloids (e.g., FFP) can be administered to increase intravascular volume and may be preferred over large-volume fluid resuscitation.\textsuperscript{1} Hypotension secondary to hemorrhage should be controlled with administration of pRBCs. Monitoring blood loss during surgery can be helpful for determining rate and quantity of blood product administration.

For patients experiencing continued or severe hypotension, vasopressors should be administered to prevent MAP from falling below 60 mm Hg and risking acute kidney injury.

Capnography and Ventilatory Support
The most important piece of monitoring equipment available for the critical-care patient’s anesthetist may be the capnograph. Although other forms of monitoring equipment may be prone to error, the capnograph reading provides accurate, real-time insight as to the patient’s ventilatory and cardiovascular status. Abruptly decreased $ETCO_2$ should immediately alert the anesthetist to imminent cardiopulmonary arrest. High $ETCO_2$ readings can indicate hypoventilation, and the anesthetist should provide ventilatory support via PPV. If already providing ventilatory support, increasing the respiratory rate and/or tidal volume will help return the $ETCO_2$ to normal.

Positive-Pressure Ventilation
PPV is delivered primarily by using a surgical ventilator, although in the absence of a ventilator, PPV can be performed manually. Surgical ventilators connect to the anesthesia machine and use the anesthetic gas, oxygen, and tubing to deliver positive-pressure breaths to the patient. Positive-pressure breaths are delivered by increased pressure in the ventilator forcing air into the patient’s lungs. That process differs from a natural patient-initiated breath, in which the diaphragm and intercostal muscles move to cause decreased pleural pressure and consequently decreased alveolar pressure to below atmospheric pressure, causing air to enter the lungs.\textsuperscript{19}

Each positive-pressure breath increases intrathoracic pressure, which can decrease venous return. Decreased venous return equals decreased stroke volume, which means decreased cardiac output (stroke volume times heart rate) and potentially decreased blood pressure, particularly in the already hemodynamically compromised patient.\textsuperscript{20} Thus, for hypotensive patients, peak inspiratory pressures should be lowered and the expiratory phase should be prolonged. \textbf{TABLE 4} and \textbf{FIGURE 4} show suggested initial ventilator settings.

\begin{table}[h]
\centering
\caption{Initial Ventilator Settings}
\begin{tabular}{|l|l|}
\hline
\textbf{PARAMETER} & \textbf{SETTING} \\
\hline
Respiratory rate & 5–15 breaths/min \\
Peak inspiratory pressure & 10–20 cm H$_2$O \\
Tidal volume & 10–15 mL/kg \\
\hline
\end{tabular}
\end{table}
Positive End-Expiratory Pressure
For patients in which ventilation is sufficient but pulse oximetry numbers are low despite adequate oxygen delivery, adding a positive end-expiratory pressure (PEEP) valve may be considered. Recumbent patients are likely to be predisposed to compression atelectasis and thus experience a ventilation/perfusion mismatch. Ventilation/perfusion mismatch is caused when areas of the lungs are not ventilated (alveoli are not receiving air) but are perfused (alveolar capillaries are receiving blood), leading to sections of desaturated blood being shunted past the lungs and back to the heart.

In addition to compression atelectasis, patients receiving a high fraction of inspired oxygen (e.g., those being administered 100% oxygen) may also experience absorption atelectasis. Absorption atelectasis results from the alveoli being flooded with a high fraction of inspired oxygen, causing increased oxygen pressure in the alveoli and thus faster absorption from the alveoli into the pulmonary capillaries. Collapse occurs when this rate is faster than alveolar refilling.

Both compression and resorption atelectasis can be treated with use of a PEEP valve. PEEP valves prevent airway pressure from reaching 0 and prevent alveolar collapse by maintaining a positive airway pressure, thus allowing more alveoli to participate in gas exchange. High levels of PEEP hinder CO₂ exchange and may lead to alveolar overdistension in non–gravity-dependent alveoli. A PEEP of 2.5 to 5 cm H₂O is usually sufficient for alveolar recruitment, except for patients with severe pulmonary disease.

Temperature
The critical-care patient is particularly prone to loss of thermostatic regulation and to hypothermia. Controlling hypothermia will require multiple warming measures (e.g., warming blanket devices [HotDog, hotdogwarming.com; Bair hugger, bairhugger.com], fluid warmers, limb insulation devices such as socks and plastic wrap, warm lavage fluids). Recovery of hypothermic patients will be slow, and oxygen offload in peripheral tissues may be impaired.

Cardiopulmonary Arrest
Cardiopulmonary arrest is often preceded by a sudden development of an arrhythmia and rapid decline in ETco₂. When arrest or impending arrest is suspected, RECOVER (Reassessment Campaign on Veterinary Resuscitation, recoverinitiative.org) guidelines should be followed. All anesthetic agents should be ceased; inhalant gas should be turned to 0, and CRI pumps should be stopped. If performing abdominal surgery, the surgeon can pierce the diaphragm and begin direct cardiac compressions. If PPV is not being administered, it should be initiated at 10 breaths/min to 20 cm H₂O pressure. The emergency drugs epinephrine and atropine should be given intravenously, quickly followed by emergency reversal agents; opioids are reversed with naloxone, and benzodiazepines are reversed with flumazenil (TABLE 5). Survival to discharge is reported to be as high as 47% for patients that underwent anesthetic death; thus, cardiopulmonary arrest is a possible outcome.

<table>
<thead>
<tr>
<th>TABLE 5 Emergency Drugs for Dogs and Cats²³</th>
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<tbody>
<tr>
<td>DRUG</td>
</tr>
<tr>
<td>Epinephrine</td>
</tr>
<tr>
<td>Atropine</td>
</tr>
<tr>
<td>Naloxone</td>
</tr>
<tr>
<td>Flumazenil</td>
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<tr>
<td>Lidocaine</td>
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![FIGURE 4. Surgical ventilator used for positive-pressure ventilation during anesthesia.](image-url)
resuscitation efforts should be conducted according to clients’ wishes until all efforts have been exhausted.22

POSTOPERATIVE
The second potentially most critical period for the critically anesthetized patient is recovery. Cardiopulmonary arrest can rapidly occur if monitoring, oxygen supply, and CRIs are discontinued as the patient is moved from the surgical suite to the recovery location. Complications can be minimized if the patient fully recovers and is extubated while in the surgical suite. Use of mobile IV poles to continue running supportive fluids and CRIs and, if available, mobile anesthesia monitoring equipment is also recommended. Performing a recheck minimum database during the postoperative period will help guide continued clinical decisions.

SUMMARY
Anesthetizing critical-care patients requires diligence and knowledge. Preparing and anticipating intraoperative complications will lead to swift response times and better patient outcomes.

References