

TURN THE GAS OFF—WE'RE DONE! RECOVERY FROM ANESTHESIA



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Did you know that most adverse events associated with anesthesia occur during recovery? That's right; of the three anesthetic periods—induction, maintenance, and recovery—the recovery period is often the most critical. Why? Many factors can play a role: the unrecognized residual effects of anesthetic agents, the termination of oxygen and fluid support, and, perhaps most importantly, a lack of monitoring or personnel.

In anesthetized people, one study found a 26% overall complication rate when intraoperative and postoperative complications were combined. Of the 26%, only 3% occurred intraoperatively and 23% occurred in recovery.¹ Contrary to what might be expected, these complications did not occur primarily in severely compromised patients but in patients only mildly to moderately compromised (American Society of Anesthesiologists classification ASA II to ASA III—*Table 1*).¹ Unfortunately, without a thorough physical examination and appropriate chemistry and ancillary tests (e.g., an electrocardiogram or thoracic radiographs) before induction, mild disease that might change an ASA I patient to an ASA II or III can be easily overlooked. This oversight can put patients at extreme risk for postanesthetic complications.

The most common complications in anesthetized people include respiratory compromise (15.2%), cardiovascular abnormalities (12.3%), and excessive pain (7.2%).² Respiratory complications include general respiratory depression, upper airway dysfunction, apnea, and hypoxemia-hypercarbia, while cardiovascular complications include hypotension and arrhythmias. One veterinary study from a major referral center reported an overall anesthetic complication rate of 12% in dogs and 10.5% in cats, with respiratory and cardiovas-

Table 1: American Society of Anesthesiologists classification of patients undergoing anesthesia

ASA I:	A normal, healthy patient
ASA II:	A patient with mild systemic disease (compensated cardiac disease, mild fever)
ASA III:	A patient with severe systemic disease (moderate dehydration, anemia, cachexia, hypovolemia)
ASA IV:	A patient with severe systemic disease that is life-threatening
ASA V:	A patient not expected to live

Table 2: Monitoring recommendations for the recovery period

Heart rate and rhythm
Pulse strength
Mucous membrane color and capillary refill time
Respiratory rate, rhythm, and depth (using thoracic excursions as a guide)
Core body temperature
ECG
Blood pressure (e.g., using oscillometric or Doppler methods)
Pulse oximetry (if possible)
End-tidal CO ₂ or arterial blood gases (in select cases)
Urinary output (in select cases)
Blood glucose and serum electrolytes (in select cases)

cular complications most common.³ In a Canadian study, the most common causes of anesthetic complications in dogs and cats were respiratory and cardiovascular dysfunction, and complications occurred in both the maintenance and recovery phases of anesthesia.⁴ And a British study of anesthetic risk in small animals found that 25% of all fatalities occurred postoperatively.⁵ Since mammals are affected similarly by anesthetic drugs, it is not surprising that postanesthetic complications in veterinary patients are similar to those in people.

In addition, hypothermia should be considered in veterinary patients. Because of the rapid heat loss associated with a small body mass and large body surface area, veterinary patients often become hypothermic under anesthesia. Hypothermia can cause a wide array of side effects and should be prevented when possible.

Optimally, postoperative complications can be minimized by thorough preoperative assessment and stabilization and appropriate intraoperative management and support. However, attentive monitoring and support well into the recovery period is imperative for a successful anesthetic outcome. Although young, healthy patients may recover rapidly with a minimum amount of support required, aged and compromised patients may recover slowly and require an extended monitoring period (*Table 2*). Regardless of the duration, patients should be monitored until they are fully recovered from anesthesia. After anesthesia, sleep from sedatives and analgesics is acceptable, but residual anesthesia is not. The difference is that patients still anesthetized are not rousable when stimulated, while patients that are sleeping comfortably due to analgesia and light sedation are rousable with stimulation.

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Table 3: Cardiovascular effects of commonly used anesthetic drugs

Cardiovascular component affected	Drug(s)	Effect
Heart rate	Ketamine, tiletamine	Increased rate
	Propofol, alpha-2 agonists	Decreased rate
Contractility	Inhalant anesthetic agents, propofol, thiobarbiturates	Decreased contractility
	Ketamine, tiletamine	Indirect increase in contractility (direct decrease in contractility)
Preload	Acepromazine, isoflurane, sevoflurane	Vasodilators = decreased preload (decreased cardiac return)
Afterload	Alpha-2 agonists, halothane, ketamine, tiletamine	Vasoconstriction = increased afterload
Relaxation	Ketamine, halothane	Impaired relaxation

Respiratory complications

All anesthetic agents cause some degree of dose-dependent respiratory depression (both depression of respiratory rate and tidal volume), and this depression is not eliminated when the vaporizer is turned off. Instead, residual depression lasts well into the recovery period and, when unrecognized, is one of the major contributors to postanesthetic morbidity and mortality. Furthermore, both upper and lower airway dysfunction occur in recovery, and either condition can lead to hypoxemia and hypercarbia.

Upper airway complications include laryngeal dysfunction, paralysis, edema, and collapse of the soft palate or other tissues into the airway. Brachycephalic patients and patients who have suffered traumatic intubation are likely to experience postoperative upper airway dysfunction. All patients should be left intubated until they are swallowing vigorously, and patients with suspected upper airway dysfunction should remain intubated until they will no longer tolerate the endotracheal tube. Patients should not be excessively stimulated for extubation. Excessive stimulation may rouse the patient enough to allow extubation but not enough to ensure adequate ventilation. Lower airway dysfunction includes atelectasis and ventilation-perfusion mismatch, both of which can contribute to impaired ventilatory function. Airway disease, age, and

anesthesia of long duration all contribute to lower airway dysfunction.

In order to prevent respiratory complications, patients should be closely monitored during recovery. Respiratory rate and volume (as judged by thoracic excursions) and mucous membrane color should be assessed routinely. As people transition from 100% oxygen during anesthesia to 21% oxygen after anesthesia, hemoglobin in the red blood cells (RBCs) rapidly desaturates,⁶ and we can assume that the same phenomenon also occurs in veterinary patients. Pulse oximetry may be useful during recovery to detect RBC desaturation, and supplemental oxygen should be provided if desaturation is expected or suspected. Oxygen can be delivered through the endotracheal tube or through a nasal cannula if the patient is already extubated.

Cardiovascular complications

Hypotension and arrhythmias are anesthetic complications that can occur in both human and veterinary patients. Hypertension may also occur in people, but it is rarely a problem in veterinary medicine unless linked to a specific cause (e.g., pain or a hypertensive disease, such as hyperthyroidism). Most anesthetic agents, including inhalant agents, cause a dose-dependent decrease in systemic arterial blood pressure through a variety of mechanisms

(Table 3). As with anesthetic-induced respiratory depression, anesthetic-induced cardiovascular depression is not eliminated when the vaporizer is turned off; instead, function improves over time. In veterinary medicine, blood pressure is not routinely measured in the recovery period, so we are unsure of the incidence of hypotension in dogs recovering from anesthesia. However, since most of the commonly used anesthetic drugs cause hypotension, we should expect residual hypotension to occur in our patients during the recovery period.

Arrhythmias may occur secondary to a whole array of anesthesia-related factors, including the arrhythmogenic effects of the drugs themselves, hypoxia, and hypercarbia. As with hypotension, cardiac arrhythmias may occur more often than we recognize. In a study of healthy dogs anesthetized with either isoflurane or propofol, arrhythmias occurred in the first 24 hours after anesthesia in 56 out of 60 dogs, although the overall number of arrhythmias per patient was low.⁷

To detect cardiovascular complications, both blood pressure and electrocardiogram (ECG) monitoring should continue well into the recovery period whenever possible. Intravenous fluid therapy should be continued in hypovolemic and hypotensive patients, and positive inotropic agents such as dopamine or dobutamine should be

Table 4: Analgesic drug dosages commonly used during anesthetic recovery*

Drug	Class	Dosage	Comments
Morphine	Opioid, full agonist	0.25 to 2.0 mg/kg IM, SQ in dogs; 0.1 to 0.3 mg/kg IM, SQ in cats	Potent opioid, may cause excitement in cats
Morphine	Opioid, full agonist	0.05 to 0.2 mg/kg/hr in dogs (use sedation in cats)	Run as constant rate infusion (CRI)
Hydromorphone	Opioid, full agonist	0.05 to 0.2 mg/kg IM, SQ, IV in dogs and cats	Potent opioid, may cause hyperthermia in cats
Hydromorphone	Opioid, full agonist	0.05 to 0.1 mg/kg/hr in dogs; 0.01 to 0.05 mg/kg/hr in cats	Run as CRI
Fentanyl	Opioid, full agonist	5 to 20 µg/kg/hr in dogs and cats	Potent opioid, short duration, run as CRI
Buprenorphine	Opioid, partial agonist	0.01 to 0.03 mg/kg IM, SQ, IV in dogs and cats; buccally in cats	Long-lasting analgesia, minimal sedation
Butorphanol	Opioid, agonist-antagonist	0.2 to 0.4 mg/kg IM, SQ, IV in dogs and cats	Short-lasting analgesia, mild sedation
Medetomidine	Alpha-2 agonist	1 to 5 µg/kg IV, IM, SQ in dogs and cats	Moderate analgesia, moderate sedation
Ketamine	NMDA-receptor antagonist	2 to 10 µg/kg/min in dogs and cats	Run as CRI with a true analgesic agent
Lidocaine	Local anesthetic agent	2 to 4 mg/kg local block in dogs and cats	May need to sedate patient to perform block
Lidocaine	Local anesthetic agent	25 µg/kg/min	Run as CRI
Bupivacaine	Local anesthetic agent	1 to 2 mg/kg local block in dogs and cats	May need to sedate patient to perform block. Do not give IV.
Carprofen, deracoxib	NSAIDs	See individual drug recommendations	Other NSAIDs are not FDA-approved for surgical pain in dogs

*Not all of the drugs or dosages referenced are approved by the FDA. Please check drug labels or www.fda.gov/cvm for more information on individual drugs.

available for patients in which hypotension persists despite appropriate fluid therapy. Specific antiarrhythmic therapy should also be available to treat arrhythmias that develop. Drugs used to treat hypotension and arrhythmias have been reviewed elsewhere.⁸

Uncontrolled pain, stress, and excitement

Uncontrolled pain in recovery is a problem in both veterinary and human patients.⁹⁻¹¹ Unfortunately, uncontrolled pain can become a pathology in itself, producing such adverse effects as tachycardia, hypertension, tachypnea, gastric ulcerations, ileus, decreased renal function, catabolism, altered hemostasis, and impaired wound healing.¹² Furthermore, pain can cause

excitement as the patient regains consciousness, resulting in a turbulent recovery.¹³ In fact, it can be difficult to differentiate the effects of pain from “emergence delirium” caused by the anesthetic drugs themselves. Fortunately, differentiation really isn’t necessary since excitement, regardless of the cause, is not appropriate in the recovery period. Excitement with subsequent physiologic stress can lead to respiratory and hemodynamic compromise, and all forms of stress should be treated.¹⁴⁻¹⁵

Drugs that provide both sedation and analgesia should be considered. Options in this category include opioids and alpha-2 agonists. Opioids are often the first choice because of their analgesic potency. However, if opioid therapy is already being used and the

patient remains painful, alpha-2 agonists should be considered. These drugs augment the analgesia provided by opioids and decrease the anesthesia-induced stress response. Alpha-2 agonists are often used in human medicine to control excitement and pain following recovery from anesthesia.¹⁵ If pain persists after initial treatment, other possible analgesic protocols include local anesthetic blockade and constant rate infusions. See the drug recommendations and dosages in *Table 4*.

Appropriate use of analgesic drugs in the preoperative and intraoperative period will decrease pain in recovery;¹⁶ however, breakthrough pain often occurs. Thus, regardless of the use of preemptive or intraoperative analgesia, pain should still be anticipated,

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Table 5: Effects of hypothermia

Some of the problems that can arise when an animal's core body temperature decreases are as follows:*

- >96 F—no adverse effects from hypothermia. Shivering and nonshivering thermogenesis increase in conscious patients.
- 90 to 94 F—cerebral depression with reduced anesthetic requirements, prolonged anesthetic recovery. Shivering is impaired in conscious patients. *Note:* Animals often get this cold during anesthesia. Anesthetic gas delivery should be decreased, but this is often overlooked.
- 82 to 86 F—marked cerebral depression with little or no anesthetic required. Atrial arrhythmias occur; shivering is nonexistent in conscious patients.
- 77 to 80 F—cold-induced ECG changes (prolonged P-R interval, widened QRS) and increased myocardial automaticity occur. Oxygen delivery is usually inadequate (due to extreme vasoconstriction) and lactic acidosis occurs, normal reflexes and pain responses are absent, blood viscosity is increased, and microcirculatory sludging occurs.
- 72 to 74 F—spontaneous ventilation ceases, ventricular fibrillation and coagulation disorders occur.
- <68 F—asystole and ECG silence occur.

* Haskins SC. Operating room emergencies. In: Slatter D, ed. *Small animal surgery*, 3rd edition. Philadelphia: Saunders, 2003;2521-2532.

and all patients should be assessed for pain in recovery.

Hypothermia

Hypothermia develops rapidly in patients under anesthesia, and the cause of the drop in body temperature is multifactorial. Some contributing factors include direct suppression of thermoregulatory activity of the hypothalamus by general anesthesia; anesthesia-induced vasodilation, which redistributes warm blood from the body's core to the skin where heat is released; evaporation of surgical scrub solutions from the body surface; equilibration of core body temperature with ambient temperature through open body cavities; delivery of cold anesthetic gases to the large surface area of the alveoli; and conduction of heat to the metal table.

Hypothermia causes a variety of complications, including clotting dysfunction, increased risk of infection, tissue hypoxia, acidosis, abnormal electrical conduction in the heart, and myocardial ischemia.¹⁷ Hypothermia also causes cerebral effects that decrease the patient's anesthetic needs. Unfortunately, the decreased anesthetic need is not always recognized and anesthesia delivery is not changed, resulting in an anesthetic overdose. Although shivering in recovery may increase the body temperature, the intensive muscle movements associated with shivering cause discom-

fort and increase oxygen consumption by as much as 200%.¹⁸ In fact, in human medicine, one area of research focus is prevention of shivering in the postoperative period. Finally—and importantly—hypothermia is the main cause of prolonged recovery in small animal patients. Listed in *Table 5* are some of the problems that arise as core body temperature decreases.¹⁹

Prevention of hypothermia should be the goal for all patients, and active rewarming should begin immediately once hypothermia has occurred. Forced air blankets have been shown to be the most effective means of rewarming.²⁰ Increasing ambient temperature also contributes significantly to warming. This can involve everything from heating an entire room to creating a warmed area around the patient using hot water bottles or sand bags with a blanket covering the patient and the warming devices. As always, insulation should be placed between heated items and the patients so that the skin is not burned.

Summary

Unfortunately, the importance of the anesthetic recovery period is often overlooked even though most unexpected anesthetic deaths occur during recovery. Common complications include hyperventilation, hypotension, hypothermia, and excessive pain. For a successful anesthetic outcome, appropriate moni-

toring and patient support must occur well into the recovery period.

References

1. Tarrac SE. A description of intraoperative and postanesthesia complication rates. *J Perioperative Nursing* 2006;21(2):88-96.
2. Mayson KV, Beestra JE, Choi PT. The incidence of postoperative complications in the PACU. *Can J Anesth* 2005;52(S1):A62.
3. Gaynor JS, Dunlop CI, Wagner AE, et al. Complications and mortality associated with anesthesia in dogs and cats. *J Am Anim Hosp Assoc* 1999;35:13-17.
4. Dyson DH, Maxie GM, Schnurr D. Morbidity and mortality associated with anesthetic management in small animal veterinary practice in Ontario. *J Am Anim Hosp Assoc* 1998;34:325-335.
5. Clarke KW, Hall LW. A survey of anaesthesia in small animal practice. *J Assoc Vet Anaesthesiol* 1990;17:4-10.
6. Daley MD, Norman PH, Colmenares ME, et al. Hypoxaemia in adults in the post-anaesthesia care unit. *Can J Anaesth* 1991;38(6):740-746.
7. Buhl K, Kersten U, Kramer S, et al. Incidence of post-anaesthetic arrhythmias in dogs. *J Small Anim Pract* 2005;46(3):131-138.
8. Laste NJ. Cardiovascular pharmacotherapy. Hemodynamic drugs and antiarrhythmic agents. *Vet Clin North Am Small Anim Pract* 2001;31(6):1231-1252.
9. Hellyer PW. Treatment of pain in dogs and cats. *J Am Vet Med Assoc* 2002;221(2):212-215.
10. Kehlet H, Dahl JB. Anaesthesia, surgery, and challenges in postoperative recovery. *Lancet* 2003;362(9399):1921-1928.
11. Skinner HB. Multimodal acute pain management. *Am J Orthop* 2004;33(5S):5-9.
12. Middleton C. Understanding the physiological effects of unrelieved pain. *Nurs Times* 2003;99(37):28-31.
13. Vaurio LE, Sands LP, Wang Y, et al. Postoperative delirium: The importance of pain and pain management. *Anesth Analg* 2006;102:1267-1273.
14. Manworren RC, Paulos CL, Pop R. Treating children for acute agitation in the PACU: differentiating pain and emergence delirium. *J Perioperative Nurs* 2004;19(3):183-193.
15. Pandharipande P, Ely EW, Maze M. Alpha-2 agonists: can they modify the outcomes in the Postanesthesia Care Unit? *Curr Drug Targets* 2005;6(7):749-754.
16. Lascelles BD, Cripps PJ, Jones A, et al. Efficacy and kinetics of carprofen, administered preoperatively or postoperatively, for the prevention of pain in dogs undergoing ovariohysterectomy. *Vet Surg* 1998;27(6):568-582.
17. Noble KA. Chill can kill. *J Perioperative Nursing* 2006;21(3):204-207.
18. Sessler DI. Temperature disturbances. In: Gregory GA, ed. *Pediatric anesthesia*, 4th ed. Philadelphia, Pa: Churchill Livingstone, 2002; 53-69.
19. Haskins SC. Operating room emergencies. In: Slatter D, ed. *Small animal surgery*, 3rd edition. Philadelphia: Saunders, 2003;2521-2532.
20. Good KK, Verble JA, Secrest J, et al. Postoperative hypothermia—the chilling consequences. *AORN J* 2006;83(5):1055-1066.