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Hypotension During Anesthesia in Dogs and Cats: Recognition, Causes, and Treatment

KEY FACTS

- Many injectable and inhalant anesthetic agents depress blood pressure.

- Hypotension is the most common perianesthetic complication in small animal patients.

- Pulse pressure alone does not accurately reflect systemic arterial blood pressure in many small animals.

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ABSTRACT: Perianesthetic hypotension is common in veterinary patients. The pathophysiology of hypotension is multifactorial but includes effects of injectable and inhalant anesthetic agents on the cardiovascular system. Hypotension can result in decreased perfusion to vital organs such as the brain, heart, and kidneys, leading to dysfunction. Direct and indirect methods of arterial blood pressure monitoring are available to veterinary practitioners. Continuous or frequent perianesthetic blood pressure monitoring allows quick recognition of hypotension and treatment of its underlying cause before negative consequences can occur. Treatment of hypotension may include decreasing anesthetic depth and administering intravenous crystalloid and colloid fluids, anticholinergic agents (as positive chronotropes), and positive inotropic agents to improve contractility. All these help to improve cardiac output and tissue perfusion, thereby preventing organ damage.

General anesthesia requires a balance of providing sensory deprivation to noxious surgical stimuli and muscle relaxation while simultaneously maintaining adequate tissue perfusion and oxygen delivery throughout the body. Many injectable and inhalant anesthetic agents provide the former but have the unfortunate side effect(s) of decreasing cardiac output (CO), systemic vascular resistance, or both, leading to hypotension or low blood pressure (Table 1). Systemic blood pressure is a product of CO (i.e., stroke volume, heart rate) and systemic vascular resistance (Figure 1). Stroke volume is influenced by preload (i.e., blood volume), afterload, and cardiac contractility. Therefore, factors that affect these variables can result in changes in systemic blood pressure. Additionally, age, underlying disease(s), duration of anesthesia, and number of surgical procedures may further predispose individual patients to the development of perianesthetic hypotension.[1]

Hypotension is the most common perianesthetic complication observed in veterinary patients.[2] Although blood pressure alone is not an exact indicator of tissue perfusion, it provides the most reliable and least invasive means of assess-

TABLE 1
Effects of Drugs on Cardiovascular Function[3]

Drug	Heart Rate	Cardiac Output	Contractility	Systemic Vascular Resistance	Blood Pressure
Anticholinergic	Increase	Increase	Increase	NC	NC or increase
Phenothiazine	± Increase	± Increase	± Decrease	Decrease	Decrease
Benzodiazepine	NC	NC	NC	NC	NC
α_2 Agonist	Decrease	NC or decrease	Decrease	Increase	Increase then decrease
Opioid	Decrease	Decrease	NC or decrease	NC or decrease	NC or decrease
Barbiturate	Increase	Decrease	Decrease	Increase	Decrease
Propofol	NC or increase	Decrease	Decrease	Decrease	Decrease
Inhalant	NC	Decrease	Decrease	Decrease	Decrease
Etomidate	NC or increase	NC or decrease	NC or decrease	NC	NC or decrease

NC = no change; ± indicates that the change may or may not occur.

ing cardiovascular well-being.[3] Normal systolic, diastolic, and mean arterial blood pressure (MAP) values for conscious, nonanesthetized small animals are 100 to 160, 60 to 100, and 80 to 120 mm Hg, respectively.[4] It is widely accepted that a MAP of 60 to 70 mm Hg is necessary to maintain adequate blood flow to vital organs.[5–6] Hypotension, by definition, occurs when systolic pressure and MAP are less than 80 and 60 mm Hg, respectively.[7] Early recognition of hypotension is paramount because it may prevent the negative consequences of inadequate tissue perfusion such as renal, cerebral, and myocardial ischemia. Routine blood pressure monitoring during anesthesia is therefore advocated in all veterinary patients,[8] especially those that are very old,[9] very young,[10] or critically ill[6] and may not have functional reserve to tolerate inadequate perfusion to vital organs. This article describes the methods of measuring arterial blood pressure (ABP) in veterinary patients, reviews the pathogenesis of hypotension in anesthetized patients, and proposes an algorithm for treatment of hypotension.

MONITORING ARTERIAL BLOOD PRESSURE
Direct Monitoring

Arterial blood pressure can be measured by direct and indirect methods. The gold standard involves direct catheterization of an artery (e.g., dorsal pedal, femoral, auricular, coccygeal).[6] The catheter is then connected to an aneroid manometer, which indicates mean pressure, or to a pressure transducer that displays systolic, diastolic, and MAP waveforms and values. Although this direct arterial catheterization is the most accurate method, it is not without slight risks (e.g., bleeding, hematoma formation, infection, arterial thrombosis) and requires manual skill and frequent flushing to prevent clotting.[6–7] Additionally, arterial

catheters may provide erroneous information if the catheter or line becomes clogged or kinked or if air bubbles are present.[7] Familiarization with normal pressure waveforms is necessary to recognize that erroneous information is being displayed. Therefore, arterial lines are advocated primarily in the most critically ill patients in which minute-to-minute changes in ABP will dictate immediate changes in treatment and anesthetic regimens.

Indirect/Noninvasive Monitoring

Before the advent of Doppler and oscillometric monitoring equipment, palpation of pulse pressure was the only means of noninvasively assessing blood pressure; however, this technique is unreliable and fairly inaccurate.[6] Pulse pressure is associated with the blood pressure difference between systole and diastole. Factors that decrease the magnitude of difference between these values (e.g., vasoconstriction) may artifactually cause a decreased pulse quality even in conjunction with normal to supranormal blood pressure. Therefore, palpation of pulse should be used merely for assurance that
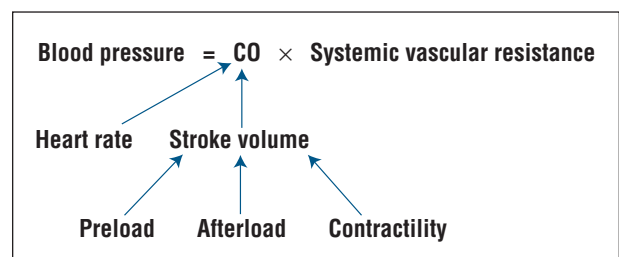

Figure 1—Systemic blood pressure is a product of cardiac output and systemic vascular resistance. Many variables (e.g., heart rate, preload, afterload, contractility) may be affected by anesthesia and surgery and, therefore, can contribute to perianesthetic hypotension.

the heart is, indeed, providing forward flow. Because palpation of pulse pressure does not provide an accurate assessment of tissue perfusion, it should be used only as an adjunctive physical parameter assessment with other noninvasive blood pressure monitoring methods.

Numerous methods for noninvasive ABP monitoring are now readily available to veterinary practitioners.[7] The most commonly used are the Doppler and oscillometric methods. **Doppler ultrasonic flow detectors** are one of the most common and most versatile methods of blood pressure monitoring in small animal medicine. A Doppler ultrasonic crystal is placed over a peripheral artery (e.g., digital, dorsal pedal, coccygeal). The probe transmits ultrasonic energy to the artery; the energy is reflected back by the flow of erythrocytes within the vessel. The reflected energy is then transmitted to a receiving crystal within the probe and converted to electrical energy in an audible transmitter.[11] An inflatable cuff with a width that is approximately 40% of the circumference of the extremity is placed proximal to the Doppler crystal.[4] Once inflated, the cuff occludes erythrocyte flow into the artery. As the pressure within the cuff is deflated, the Doppler flow probe detects erythrocyte movement within the artery; and an audible signal is produced, indicating systolic ABP. A sphygmomanometer attached to the pressure cuff allows measurement of systolic ABP (mm Hg) at the first detectable sound.[12]

Doppler flow detectors have been validated for use in small animal patients. Although systolic measurements are fairly accurate and congruent with direct ABP measurements in dogs,[13] the use of the Doppler in cats may underestimate systolic blood pressure by approximately 15 mm Hg and may more accurately reflect MAP.[12,14–16] Because hypertension is rarely a problem in anesthetized animals, the tendency toward lower pressure readings with a Doppler in cats serves to alert the anesthetist to take preemptive measures to treat potential hypotension.

A second method of indirect ABP monitoring is oscillometric sphygmomanometry.[17–19] During systole and diastole, the blood flow through an artery causes a pressure wave. The pressure wave is detected as oscillations within a pressure cuff placed over the peripheral artery. The **oscillometric sphygmomanometry method** provides a digital reading of systolic, diastolic, and MAPs as well as pulse rate. This method has been validated for accuracy in dogs at a wide range of blood pressures. However, its use may be unreliable and limited in cats[16] and other small animal species (e.g., ferrets).[20] Oscillometric blood pressure accuracy is also reduced in canine patients with extreme vasoconstriction or bradycardia.[17]

Despite the potential disadvantage of limited use in species other than dogs, the oscillometric method has several advantages over direct arterial catheterization in that it has no potential for complication to the animal, requires little technical skill, is not labor intensive, and is inexpensive.

The Doppler method, too, requires little technical skill but is more labor intensive than is the oscillometric method. Whereas the oscillometric method is automated, the Doppler method requires frequent manual monitoring to obtain blood pressure values. The Doppler method can be used across a large range of species, unlike the oscillometric method, which is valid primarily for use in dogs. Although Doppler measurements tend to provide artifactually low measurements in cats, it is still useful to measure trends of change in ABP during anesthesia. Private practitioners must weigh the expense of each type of equipment with its versatility, information obtained, ease of use, and accuracy to determine which method will be most useful for their individual practices. With the wide availability and minimal expense of the noninvasive blood pressure monitors, their use should be a routine part of all anesthetic procedures in small animals.

PATHOGENESIS OF PERIANESTHETIC HYPOTENSION
Cardiovascular Effects of Anesthetic Agents

The pathogenesis of perianesthetic hypotension can be multifactorial. Hypotension is often an adverse pharmacologic consequence of drugs, including many anesthetic agents. Various injectable and inhalant anesthetic agents directly affect heart rate, preload, afterload, myocardial contractility, or systemic vascular resistance. These variables are intimately associated with blood pressure; therefore, a change in any of the variables (alone or in combination) can decrease CO and/or blood pressure.

Various preanesthetic agents interfere with cardiovascular reflexes and the sympathetic nervous system, thereby affecting ABP. Injectable α_2 agonists (e.g., medetomidine, xylazine) used alone or in combination with other induction agents (e.g., propofol) may depress myocardial contractility.[21–25] α_2 Agonists cause a dose-dependent decrease in MAP by eliciting profound bradycardia and decreased CO.[26–31] Phenothiazine premedications (e.g., acepromazine) decrease ABP by numerous mechanisms, including depression of hypothalamic and brain stem vasomotor reflexes, peripheral α-adrenergic blockade, vascular smooth muscle relaxation causing vasodilation, and direct cardiac depression.[32] In contrast, benzodiazepine and opioid premedicants cause little cardiovascular depression and rarely

cause a decrease in blood pressure. In rare cases, morphine can cause hypotension by evoking histamine release (if administered intravenously).

Anesthetic induction agents can result in profound hypotension by various mechanisms. At induction doses, thiobarbiturates cause a central and peripheral cardiovascular depression, thereby reducing blood pressure.[32] Propofol also causes hypotension by promoting peripheral vasodilation. The hypotensive effects of both drugs can be truncated by administration of a preinduction crystalloid fluid bolus (5 to 10 ml/kg). In contrast, the dissociative anesthetic agent ketamine promotes cardiovascular stimulation following its use as an induction agent.[33] Although ketamine may appear to benefit patients by increasing CO and ABP, controversy exists whether the increased oxygen consumption and cardiac work are detrimental to the myocardium. Additionally, increased blood pressure may not be observed in patients premedicated with benzodiazepines or α_2 agonists.[33] Finally, although use of ketamine has been advocated for critically ill patients, some patients with critical illness may experience profound hypotension following ketamine administration due to exhaustion of adrenergic stimuli.[33,34]

The gas anesthetic agents most commonly available to veterinary practitioners today cause dose-dependent cardiovascular depression.[35,36] Depressed myocardial function with decreased contractility and reduced CO is observed in conjunction with vasodilation and decreased peripheral vascular resistance. The combination of decreased CO and vasodilation can result in hypotension, leading to decreased organ perfusion.[35–39] The situation may be exacerbated further by depression of the body's normal sympathetic reflexes, preventing normal physiologic maintenance of blood pressure and organ perfusion.

A perianesthetic, perioperative complication that can cause hypotension is hemorrhage. Severe hemorrhage decreases circulating blood volume, reducing venous return. In normal awake animals, baroreceptors and stretch receptors located in the carotid body and aortic arch sense decreased wall tension associated with a decrease in circulating blood volume. In response to decreased arterial wall tension, sympathetic afferents to the myocardium and peripheral vasculature transmit signals to cause an increase in heart rate and contractility, both of which serve to maintain CO. Catecholamine stimulation of α receptors in the peripheral vasculature causes peripheral vasoconstriction, which increases systemic peripheral vascular resistance, maintaining perfusion to vital organs (e.g., heart, brain, kidneys).[40] Additionally, β-adrenergic stimulation increases heart rate and force of contraction.[41] These normal reflex responses are obtunded during anesthesia, rendering normal adaptive mechanisms unable to preserve MAP and tissue perfusion during hemorrhage.

Normally during hemorrhage, release of catecholamines causes vasoconstriction and a reflex increase in stroke volume. However, α-receptor antagonists (e.g., acepromazine) can cause arteriolar vasodilation, leading to decreased preload, which may prevent an increase in stroke volume.[37,38,42–44] Additionally, α_2 agonists and gas anesthetic agents can also depress the normal sympathetic catecholamine response to hemorrhage, thereby exacerbating hypotension.

PREVENTION AND TREATMENT OF HYPOTENSION

In animals at risk for hypotension, the use of premedicants or induction agents known to exacerbate hypotension should be avoided. Instead, combinations of benzodiazepine drugs such as diazepam (0.2 to 0.5 mg/kg IV), midazolam (0.2 to 0.5 mg/kg IV) with the opioid fentanyl (5 to 10 µg/kg IV), or etomidate (0.5 to 1.0 mg/kg IV) can provide rapid, smooth anesthetic induction without causing significant cardiovascular compromise. If no known preexisting cardiac disease is present, ketamine in combination with diazepam may also be considered.

Treatment of perianesthetic hypotension should be directed at the primary cause. Many gas anesthetic agents depress myocardial function and decrease vascular resistance, causing an apparent decrease in circulating volume (apparent hypovolemia). The combination of decreased CO and vasodilation results in hypotension. Decreasing the depth of anesthesia can improve CO and systemic vascular resistance, resulting in increased blood pressure. The use of balanced anesthesia, combining opioid analgesic agents (e.g., fentanyl bolus 2 µg/kg or constant-rate infusion [CRI] 5 to 45 µg/kg/hr) with decreased doses of inhaled anesthetics, can aid in maintaining adequate anesthetic depth without further depressing cardiovascular function. The use of opioids, however, can result in profound respiratory depression,[45] necessitating assisted or controlled mechanical ventilation to prevent hypercapnia. Intermittent positive-pressure ventilation can contribute to hypotension by decreasing venous return to the heart, ultimately decreasing CO. Therefore, although opioids are beneficial in providing balanced anesthesia to hypotensive patients, careful monitoring of ventilation, heart rate, and blood pressure is necessary. The analgesic actions of ketamine (0.5 to 1 mg/kg IV) may allow dosages of inhalant and opioid to be decreased.

Intravenous crystalloid fluid boluses (5 to 10 ml/kg) can increase circulating fluid volume in apparent hypo-

volemia secondary to peripheral vasodilation. Additionally, hemorrhage or acute loss of circulating volume is a frequent perianesthetic complication.[46,47] Treatment of acute blood loss due to hemorrhage includes crystalloid fluid replacement at a dose of three times the blood volume lost.[48] In severe hemorrhage, combination colloid therapy in the form of whole blood, packed erythrocytes, plasma, or synthetic colloids such as hetastarch (5 ml/kg boluses) can also be used to expand intravascular fluid volume with fewer adverse consequences of hemodilution or decreased colloid oncotic pressure.

In some cases, decreasing anesthetic depth and expanding fluid volume are not enough to restore adequate blood pressure, which may require the use of various anticholinergic or inotropic agents.[48,49] Vagally mediated bradycardia caused by drugs or certain surgical manipulations may contribute to decreased CO in some patients. The use of parasympatholytic agents such as atropine (0.01 to 0.04 mg/kg IV or SQ) or glycopyrrolate (0.005 to 0.02 mg/kg IV or SQ) can increase heart rate and help restore CO.

Inotrope Therapy

Ephedrine is a noncatecholamine sympathomimetic drug that directly stimulates α- and β-adrenergic receptors and indirectly stimulates norepinephrine release.[50] Bolus ephedrine administration (0.1 to 0.25 mg/kg IV) to isoflurane-anesthetized dogs causes an immediate increase in MAP and cardiac index.[49] A second beneficial effect of ephedrine is improved oxygen delivery to tissues via increased hemoglobin concentrations by an unknown mechanism.[49] The advantage of ephedrine over dopamine, dobutamine, and epinephrine is that it can be administered as a bolus and has a longer duration of action, thus eliminating the need for CRI. Additionally, ephedrine is less expensive than dobutamine and apparently does not predispose animals to arrhythmias.[49]

Dobutamine, a synthetic balanced catecholamine, stimulates cardiac contractility, CO, and coronary blood flow without causing a significant change in peripheral vascular resistance.[51,52] The combined effect is that dobutamine provides a more reliable increase in MAP when compared with dopamine. The therapeutic dose ranges from 2 to 20 µg/kg/min.[52] However, doses above 10 µg/kg/min are rarely used. At the lower end of the dose range, improved cardiac contractility via stimulation of cardiac β receptors is observed. Doses higher than 20 µg/kg/min may result in unmasking of the drug's α effects, resulting in decreased coronary perfusion.[53]

Dopamine, a norepinephrine precursor, exerts its effects through stimulation of dopaminergic and α- and β-adrenergic receptors. The receptor effects of exogenous dopamine are dose-dependent.[54] Low-dose dopamine (1 to 2 µg/kg/min) preferentially affects dopaminergic receptors on renal, splanchnic, coronary, and cerebral vasculature, resulting in vasodilation and apparent improved perfusion of these organs. At a dose range of 2 to 10 µg/kg/min, the effects of dopamine are primarily at β-adrenergic receptors, causing increased cardiac contractility.[54] Stimulation of cardiac α_1 receptors can also cause an increase in heart rate and tachycardia, particularly at doses greater than 10 µg/kg/min. This can cause coronary vasoconstriction and myocardial excitability as well as increase the presence of tachyarrhythmias.[55,56] Some authors[6] report that dopamine, particularly at lower doses, is unreliable at increasing MAP because of the combined effects of peripheral vasodilation with increased CO. The benefits of dopamine therapy may be difficult to perceive. Additionally, dopaminergic receptors have not been identified in cats; therefore, the use of this drug to improve splanchnic, coronary, and cerebral perfusion in this species remains questionable and controversial. Dobutamine, therefore, may be the preferred drug to use to improve MAP. The effects of both dopamine hydrochloride and dobutamine are short-lived, and thus CRI administration is required.

Norepinephrine acts at α, β_1, and β_2 receptors, causing increases in MAP and peripheral vascular resistance, particularly in capillary beds preferentially dilated, as in sepsis. Coronary blood flow is also increased. Norepinephrine (0.05 to 0.4 µg/kg/min) may be beneficial in increasing MAP in septic patients or patients currently on β-blocking drugs (e.g., propranolol, atenolol) that do not respond to dobutamine. Potential negative effects of norepinephrine therapy include tissue ischemia secondary to intense vasoconstriction and tachyarrhythmia.[57]

Epinephrine causes an increase in MAP by three mechanisms: positive inotropic and chronotropic actions at cardiac β_1 receptors and peripheral vasoconstriction at vascular α receptors.[50] The increased heart rate and contractility may cause increased cardiac work and increased myocardial oxygen consumption and may predispose the animal to arrhythmia(s).[50,58] Epinephrine has been shown to increase arrhythmogenic potential in anesthetized animals, particularly those under the influence of halothane.[55] Concurrently, intense vasoconstriction may decrease tissue perfusion and oxygen delivery to some organs. The use of epinephrine (0.05 to 0.4 µg/kg/min IV CRI) should be reserved for the most hypotensive patients that do not respond to other inotropic support.

The use of **doxapram** as a respiratory stimulant (1 to

5 mg/kg IV) is well known in veterinary medicine. Limited reports of improved CO and ABP have been documented. Although improved CO is beneficial in hypotensive patients, other effects of doxapram (e.g., central nervous system stimulation) may be undesirable in anesthetized patients.[6] Its use, therefore, may be limited in clinical cases of perianesthetic hypotension.

Calcium is an important factor in maintaining cardiovascular stability. Calcium plays an integral role in contraction of cardiac and vascular smooth muscle cells.[59] Perianesthetic ionized hypocalcemia as well as severe alkalosis can occur secondary to administration of blood products. The anticoagulants used in blood administration systems can chelate ionized calcium, making it unavailable. Adverse effects of hypocalcemia include depressed myocardial contractility and hypotension. With multiple transfusions or transfusion in very small patients, hypocalcemia should be considered as a differential diagnosis for refractory hypotension and corrected when it occurs. Calcium can be administered as calcium chloride (10 mg/kg) or calcium gluconate (23 mg/kg) via slow IV.[60] During calcium administration, careful attention should be paid to heart rate and electrocardiography. If bradycardia develops or electrocardiographic changes (e.g., ST-segment depression, QT-interval shortening) occur, the infusion should be discontinued temporarily and then restarted at a slower rate.[60]

CONCLUSION

Hypotension is a common occurrence in anesthetized veterinary patients. Because the equipment for perianesthetic blood pressure monitoring is relatively inexpensive and readily available to all veterinary practitioners, its use is strongly advocated for all anesthetic episodes. Close monitoring can assist in early recognition and treatment of hypotension and prevent its negative consequences (e.g., organ dysfunction). With careful anesthetic planning and use of balanced anesthesia, hypotension may be avoided in high-risk patients. If hypotension does occur, quick action should be directed at treating the cause. Decreasing gas anesthetic depth, providing fluid support with crystalloids or colloids (natural or synthetic), and careful use of inotropes can correct hypotension in many cases and improve anesthetic outcome.

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ARTICLE #3 CE TEST

The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. *Choose the best answer* to each of the following questions; then mark your answers on the postage-paid envelope inserted in *Compendium.*

1. Pulse pressure is the difference between _____ pressures.
   a. arterial and venous
   b. systolic and diastolic arterial
   c. central venous and pulmonary
   d. capillary and interstitial hydrostatic
   e. none of the above

2. Which of the following components affects ABP?
   a. preload   d. heart rate
   b. afterload   e. all of the above
   c. cardiac contractility

3. What is the most common perianesthetic complication observed in small animal patients?
   a. hemorrhage   d. hypotension
   b. arrhythmia   e. vomiting
   c. hypertension

4. Normal values for systolic, diastolic, and MAP in awake, conscious animals are _____, respectively.
   a. 100 to 160, 60 to 100, 80 to 120 mm Hg
   b. 80 to 120, 60 to 80, 100 to 120 mm Hg
   c. 160 to 180, 60 to 80, 40 to 60 mm Hg
   d. 120 to 140, 40 to 80, 50 to 75 mm Hg
   e. 150 to 200, 50 to 100, 100 to 150 mm Hg

5. During anesthesia, it is generally accepted that the minimum MAP to maintain adequate tissue perfusion is _____ mm Hg.
   a. 50   d. 120
   b. 100   e. 80
   c. 60

6. When used on cats, Doppler ultrasonic crystal blood pressure monitoring
   a. is completely inaccurate.
   b. is more accurate at low heart rates.
   c. more accurately reflects MAP rather than systolic ABP.
   d. accurately reflects diastolic arterial pressure.
   e. none of the above

7. A blood pressure cuff with a width that is _____ % of the circumference of the extremity is most appropriate for use on dogs and cats.
   a. 80   d. 40
   b. 100   e. 20
   c. 60

8. Which of the following can cause hypotension?
   a. acepromazine   d. medetomidine
   b. isoflurane   e. all of the above
   c. propofol

9. A safe drug combination for anesthetic induction that causes minimal cardiovascular compromise is
   a. propofol and thiopental.
   b. fentanyl and diazepam.
   c. etomidate and diazepam.
   d. propofol and diazepam.
   e. b and c

10. Which of the following can be used to help improve blood pressure during anesthesia of a hypotensive patient?
   a. decreasing inhalant anesthetic depth
   b. intravenous crystalloid bolus
   c. positive inotropes (e.g., dobutamine, ephedrine)
   d. natural and synthetic colloids
   e. all of the above