Misconceptions About Emergency and Critical Care: Metabolic Disease and Intensive Care Medicine*

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ABSTRACT: As knowledge and clinical experience in emergency and critical care increase, previously held beliefs are being found to be inaccurate or outdated. Diagnostic and therapeutic techniques that were previously considered commonplace are no longer recommended based on current knowledge and research. This article dispels misconceptions pertaining to metabolic disease and intensive care medicine in emergency and critical care and discusses evidence of and reasoning for implementing new techniques and standards.

This article dispels outdated beliefs regarding sodium bicarbonate (NaHCO₃) and dextrose therapy, the diagnosis of diabetic ketoacidosis, and evaluation of calcium status in critically ill patients. In addition, this article discusses why it is no longer recommended to use pulse oximetry as the only monitoring tool in critically ill patients, to use heparin as an anticoagulant for blood gas analysis, to administer anticholinergics with medetomidine, or to use ice-water baths to cool heatstroke patients. Up-to-date alternatives are provided.

Misconception: NaHCO₃ administration is appropriate for all animals suspected of being acidic, such as ketotic patients.

Reality: Although NaHCO₃ administration is a suggested therapy for patients with acidosis, there are numerous circumstances in which NaHCO₃ therapy is contraindicated or has deleterious effects. In patients with respiratory acidosis secondary to hypoventilation, NaHCO₃ therapy should be avoided because it inevitably decreases respiratory drive, thereby worsening acidosis and hypoxemia.¹,² NaHCO₃ administration in these patients may also cause transient hypercarbia because NaHCO₃ is converted to water and carbon dioxide. Alkalemia may then develop as hypoventilation resolves. Likewise, animals with normochloremic metabolic acidosis (elevated anion-gap acidosis; e.g., caused by ketoacids or lactic acid) are also less likely to benefit from NaHCO₃ therapy.¹ In these patients, as organic acids are metabolized, they form bicarbonate.

* A companion article on misconceptions about cardiopulmonary cerebral resuscitation, fluid therapy, shock, and trauma starts on p. 420.
anions (HCO$_3^-$), resulting in rebound alkalosis if NaHCO$_3$ has been administered concurrently. Therefore, treatment of the underlying disease, in addition to intravenous fluid therapy, is preferred in these patients and is enough to correct the acidosis in most cases.

Other detrimental effects of NaHCO$_3$ administration include volume overload due to hypernatremia, tetany due to reduced ionized calcium (iCa$^{2+}$) levels, and hypokalemia as potassium ions enter cells in response to alkalinization of extracellular fluid. If NaHCO$_3$ administration seems inevitable based on a documented pH of less than 7.1 and HCO$_3^-$ concentration of less than 10 mEq/L despite measures to correct acidosis, NaHCO$_3$ may be replaced using the following formula:

$$\text{Bicarbonate deficit} = \frac{(\text{Base deficit} - \text{Measured bicarbonate}) \times 0.3 \times \text{Body weight}}{\text{kg}}$$

This formula is designed to avoid excessive NaHCO$_3$ replacement because it aims to correct bicarbonate to 12 mEq/L rather than to the normal value of 24 mEq/L. Complete correction of acidosis should always be avoided to reduce the risk for iatrogenic alkalosis.

**Misconception:** Administering dextrose to patients with seizures is recommended in case they are hypoglycemic.

**Reality:** Veterinarians often administer intravenous dextrose to patients with seizures in case these patients are or will become hypoglycemic. Although hypoglycemia should be the first metabolic abnormality to be ruled out in a puppy with seizures, it is less common in adult animals with seizures. In the early stages of status epilepticus, hyperglycemia and lactic acidosis are common. It is only during the very late stages of status epilepticus that energy substrates are exhausted and hypoglycemia occurs. Elevated blood glucose concentrations provide a substrate for anaerobic glycolysis in the brain, especially during episodes of ischemia. This results in accumulation of lactic acid, which causes cellular destruction and permanent neuronal damage. In hyperglycemic rats, neuronal cell damage occurs more rapidly during ischemia. Similarly, elevated blood glucose concentrations are associated with proconvulsant effects due to increased neuronal excitability. Thus, dextrose administration in animals with seizures may exacerbate existing hyperglycemia, precipitate further seizures, and lead to neuronal damage.

Therefore, because many intensive care patients are already hyperglycemic due to excessive circulating glucocorticoids, catecholamines, and cytokines, evaluation of glucose levels before dextrose administration is important. Interestingly, recent studies show that in critically ill humans, hyperglycemia is associated with increased mortality rates; therefore, strict glucose control has been strongly advocated by some authors. Clearly, dextrose administration should be carefully considered before administration in any critically ill patient with seizures. Because of the widespread availability of rapid, accurate glucometers, empiric administration of dextrose should be avoided unless hypoglycemia can be documented.

**Misconception:** Urine reagent test strips are always an accurate way to ascertain the presence or absence of ketonuria in animals with diabetic ketoacidosis (DKA).

**Reality:** A diagnosis of DKA is based on concurrent evidence of hyperglycemia, glucosuria, and ketonemia. The presence of ketonuria implies ketonemia; therefore, urine reagent test strips are widely used in animals to confirm a diagnosis of DKA. However, these color-change urine strips rely on the reaction between sodium nitroprusside and a minimum of 5 to 10 mg/dl of acetoacetic acid in the urine and do not detect the

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**Figure 1. Diagnosing ketonemia.** Plasma from a heparinized microhematocrit tube may be placed on a urine reagent test strip to confirm the presence of acetoacetic acid in the blood.
presence of β-hydroxybutyric acid or acetone. In humans, β-hydroxybutyric acid is the predominant ketone body produced during DKA. In severely dehydrated states, the β-hydroxybutyric acid:acetoacetic acid ratio may reach 20:1 if lactic acidosis is severe enough to shift production of ketone bodies toward β-hydroxybutyric acid. Therefore, a negative ketone strip does not definitively rule out the presence of ketonemia because the strip may appear negative in the presence of ketosis in a severely dehydrated patient.

To avoid misdiagnosing a patient with DKA, other tests are available and should be considered. Duarte et al found that measuring serum β-hydroxybutyric acid levels was an accurate method of diagnosing and monitoring DKA in diabetic dogs. Unfortunately, this diagnostic test is not readily available to most clinicians. However, it was recently demonstrated that plasma from a heparinized hematocrit tube placed on a urine reagent test strip (Figure 1) accurately reflects urine ketone results in diabetic dogs and cats. Another option to consider is the addition of one drop of hydrogen peroxide to the urine of patients suspected of having DKA. This facilitates a chemical reaction that increases the amount of acetoacetic acid present to subsequently react with the nitroprusside reagent strip. Hydrogen peroxide converts β-hydroxybutyric acid to acetoacetic acid and water, thus increasing the likelihood of a positive ketone reaction (Figure 2). This increases the sensitivity of the urine reagent test strip and enables a prompt diagnosis of DKA.

Administering analgesics to critically ill patients hastens recovery and improves patient outcome.

Intensive Care Medicine

Misconception: Culture of urinary catheter tips following removal of indwelling urinary catheters is a reliable way to diagnose the development of catheter-associated urinary tract infections (UTIs).

Reality: To test for acquired UTIs, many veterinarians culture the tips of indwelling urinary catheters after removal. However, recent studies in both the human and veterinary literature have revealed that this technique is not entirely accurate. Smarick et al compared urinary catheter tip and urine culture results in 19 dogs and found that catheter tip cultures tested positive in eight dogs (i.e., two with UTIs, and six without) and negative in the other 11 dogs without UTIs. Thus the sensitivity of culturing catheter tips in this study was 100%, but the positive predictive value was only 25% because of the number of false-positive results. The authors suggested that bacterial culture of urinary catheter tips should be used only as an initial screening technique and that urine should also be cultured to verify positive results. Others have suggested collecting urine samples a few days following catheter removal because of the potential for bacteria to be cleared on their own after the catheter has been removed.

A recent study compared cultures of urinary catheters and urine in humans with indwelling catheters and also found a disparity between urinary catheter and urine culture results. Matsukawa et al showed that the overall rate of positive catheter culture results was significantly greater than that of positive urine culture results (53.5% versus 30.2%, respectively). However, the authors concluded that depending on the method of catheter culture (i.e., extraluminal or intraluminal), positive catheter culture results may be due to contamination from the urethra or external surfaces after catheter removal, resulting in false-positive urinary catheter culture results. In addition, adherence of bacteria to urinary catheters varies with the type of catheter material. Thus certain species of bacteria cling more readily to certain types of catheters and are more likely to culture positive. Therefore, because of the lack of correlation between catheter tip and urine bacterial cultures following removal of indwelling urinary catheters, both urine and catheter tip samples should be cultured to determine whether patients have developed catheter-associated UTIs.
Pulse oximetry accurately estimates \( O_2 \) by an increasing margin of error. Pulse oximeters are well tolerated by most patients, thus enabling continuous monitoring when necessary (Figure 3). However, the limitations of pulse oximetry must be recognized, especially in the absence of arterial blood gases, because certain circumstances yield erroneous readings and may result in misguided and inappropriate clinical assessments.

Pulse oximeters generally provide accurate estimates of \( SaO_2 \) during normal oxygen hemoglobin saturation and minor desaturation. However, as \( SaO_2 \) values drop below 70%, pulse oximeters overestimate the \( SaO_2 \) by an increasing margin of error. One study showed that SpO\(_2\) readings were as much as 29% different from \( SaO_2 \) values. Multiple factors interfere with the ability of pulse oximeters to provide an accurate reading, including ambient light interference, dark pigmentation, hypotension and hypertension resulting in poor peripheral perfusion and vasoconstriction, and the site of probe placement. Various studies have determined that the tongue and lip provide the best readings, followed by reliable estimates of \( SaO_2 \) via the ear, toe, and tail; however, the Achilles tendon and flank sites do not provide accurate readings. Reflective pulse oximetry probes produced reliable estimations of \( SaO_2 \) via the rectum. Certain pulse oximeters have been found to be inaccurate in cats, especially when \( SaO_2 \) drops below 90%.

Diseases and conditions that induce methemoglobinemia (e.g., acetaminophen toxicosis in cats, benzocaine toxicosis in dogs) and smoke inhalation leading to carboxyhemoglobinemia may cause pulse oximeters to overestimate \( SaO_2 \). This is because pulse oximetry cannot differentiate oxygenated hemoglobin from carboxyhemoglobin or methemoglobin; thus it counts all saturated hemoglobin molecules as oxyhemoglobin.

Particular attention should be paid to animals receiving supplemental oxygen. In patients breathing supplemental oxygen, changes in oxygen saturation are not detectable by pulse oximetry until the \( PaO_2 \) value is less than 100 mm Hg (Figure 4). For example, at sea level, an animal receiving nasal oxygen (inspired oxygen of approximately 40%) should have a \( PaO_2 \) value near 200 mm Hg and an animal receiving 100% oxygen should have a \( PaO_2 \) value near 500 mm Hg. Therefore, it is possible for dramatic decreases in arterial oxygenation to occur before being detected by changes in \( SpO_2 \). Serial arterial blood gas monitoring would be a more accurate way to follow changes in arterial oxygenation in these animals. Clearly, the pulse oximeter may be least accurate in animals that are the most critically ill and in need of accurate assessments of oxygen delivery. In these patients, \( SpO_2 \) measurement should not be considered a reliable replacement for arterial blood gas analysis, which provides \( PaO_2 \) and \( SaO_2 \) values and enables accurate assessments of arterial oxygenation and alveolar ventilation.

**Misconception:** Heparin can be used as an anticoagulant when obtaining samples for blood gas analysis without interfering with the accuracy of the values obtained.

**Reality:** It is common for both veterinarians and technicians to heparinize syringes before obtaining an arterial sample for blood gas and \( iCa^2+ \) analysis with a portable
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A clinical analyzer or other blood gas and electrolyte analyzer. This process usually involves aspirating 1,000 IU/ml of sodium heparin into a syringe and then pushing the plunger to the end of the syringe. This leaves only a small amount of sodium heparin in the hub (dead space) of the syringe. This small amount is adequate to provide anticoagulant effects; therefore, the sample that is obtained may be saved for later use if there are problems in analyzing it. Unfortunately, this small amount of heparin, although seemingly harmless, is enough to interfere with many electrolyte and blood gas values.

Excess sodium heparin in a syringe can interfere with multiple values provided by a clinical analyzer, including false decreases in potassium, glucose, iCa$^{2+}$, pH, and PCO$_2$ levels. False increases can also occur in sodium and chloride levels. A recent study by Hopper et al found that heparin interferes with partial pressure of oxygen (PO$_2$), partial pressure of carbon dioxide (PCO$_2$), base deficit, bicarbonate, potassium, sodium, chloride, lactate, and iCa$^{2+}$ levels. In most instances, the altered laboratory values are a result of dilutional effects. Because the volume of blood needed to run the tests is so small and because many patients have suffered blood loss or are anemic, attempts are made to draw as little blood as possible. Unfortunately, with the amount of heparin in the syringe and the small volume of blood drawn, the sample becomes diluted. Ionized calcium values are also lowered because of chelation of divalent, positive calcium ions with the strongly negative heparin molecules.

Sodium heparin concentrations in blood samples greater than 15 IU/ml form complexes with calcium ions, resulting in false decreases in measured iCa$^{2+}$. The dead space in a 3-ml syringe is 60 to 100 µl. This space contains 60 to 100 IU of heparin if a 1,000-IU/ml sodium heparin solution is used to heparinize the syringe. Even after filling the syringe with a 3-ml blood sample, the concentration of sodium heparin to blood is 20 to 33 IU to 1 ml, which is more than necessary to chelate calcium. Depending on the concentration, this may produce an error of up to 0.15 mmol/L of iCa$^{2+}$, which is significant, considering the narrow reference range for iCa$^{2+}$. Therefore, if there is concern specifically regarding iCa$^{2+}$ levels, it is best not to use an anticoagulant. Alternatively, special syringes (e.g., Gas-Lyte, Micro ABG, and Aspirator ABG; Marquest Medical Products) are available that contain only 2 to 3 IU/ml of heparin in a “puff” of inert filler material that dissolves rapidly in the sample. This small amount of heparin is sufficient to provide anticoagulation but is too small to complex with iCa$^{2+}$ or dilute the sample. Similarly, calcium-neutralized lithium zinc heparin has been developed and virtually eliminates the chelation effects on iCa$^{2+}$. Air bubbles or spaces should be avoided when drawing the sample because they can cause loss of CO$_2$, thereby falsely lowering the pH, PCO$_2$, PO$_2$, and iCa$^{2+}$ levels.

Misconception: Critically ill patients are not suitable candidates for analgesia because it inevitably makes them more unstable and hinders proper patient monitoring.

Reality: Critically ill animals often experience pain due to trauma, systemic disease, or surgical intervention. In addition, life-prolonging procedures cause further pain in these patients. Many patients are too sick, debilitated, or compromised to display behaviors indicative of pain. Consequently, analgesic administration is often forgotten because of failure to recognize pain or is purposely avoided for fear of worsening a patient’s status. Catecholamine release associated with persistent pain inevitably decreases oxygen delivery to tissues and increases oxygen demand, causing detrimental effects that outweigh the adverse effects of analgesia administration. Energy is subsequently diverted to coping with
pain rather than allowing rest for recovery. Studies have shown that appropriate analgesics are necessary in critically ill humans and animals to reduce stress, enable sleep and healing, and hasten recovery time. Appropriately treating patients in pain also increases immune function, reduces morbidity, and decreases catabolic demands. Not treating patients in pain not only is inhumane and unethical but also results in a poorer patient outcome.

Contrary to common thought, analgesic administration does not mask physiologic indicators of patient deterioration. It has been shown in both humans and animals that analgesics do not conceal signs of worsening patient status; therefore, analgesics should not be withheld for this reason. Even when large doses of opioids are used to treat pain, the heart rate remains high in response to hypotension, hypoxia, hypovolemia, and hypercarbia. Another benefit of analgesic administration in critically ill patients is that after pain is reduced, the potential for mistaking tachycardia for a response to pain, rather than other deteriorating physical parameters, is eliminated. Thus a clinician can be alerted to a patient’s declining status faster without questioning the decline. Analgesic administration should be avoided in a few situations (e.g., in obtunded or neurologic patients). Animals with neurologic disease should be assessed carefully and analgesics administered only if a patient is painful. Administering opioids to patients with neurologic disorders that already have altered mentation or are hypventilating is not recommended. However, because pain can increase intracranial pressure, adequate analgesia must be provided to these patients. Opioids are frequently preferred in most critically ill patients because these drugs are safe and can be dosed to effect and the potential for reversal with naloxone is available in case adverse effects occur.

Similarly, in 1989, Flanders et al demonstrated that only 18% of the variability of the serum total calcium concentration in cats could be attributed to serum albumin. A strong correlation also could not be found by Bienzle et al in dogs, cats, horses, or cattle when the relationship between serum total calcium and albumin was evaluated. Thus the association between serum total calcium concentration and serum albumin was weak, rendering correction formulas highly inaccurate. Recently, Schenck and Chew confirmed this by determining that total calcium levels, even after the correction formula was applied, did not correlate with iCa levels in dogs, especially those with chronic renal failure. This study evaluated the application of the calcium correction formula in 1,633 dogs and demonstrated that the corrected calcium values incorrectly predicted iCa levels in 37% of all dogs and in 53% of dogs with chronic renal failure. Similar findings have been noted in humans. Several studies in critically ill humans have demonstrated a

**REALITY:** Correction formulas to evaluate the calcium status in animals were designed decades ago but are still widely used. These formulas are meant to correct low serum total calcium levels in dogs and cats with low albumin with the belief that concurrent hypoalbuminemia decreases the protein-bound fraction of calcium, thereby falsely lowering the total serum calcium value. Thus if the corrected total calcium value falls within the normal range, it gives the false impression that the serum total calcium level is actually normal. An example of this formula was designed by Meuten et al in 1982:

\[
\text{Ca}_{\text{serum total corrected}} = \frac{\text{Ca}_{\text{serum total measured}} - \text{Albumin}_{\text{serum measured}} + 3.5}{100}
\]

Unfortunately, because this formula was derived using an analytic method different than that used by modern autoanalyzers, the normal range for serum albumin used in the report was considerably lower. Although a positive correlation existed between the serum total calcium and serum albumin, only 33% of the variability in serum total calcium could be attributed to serum albumin.

**REALITY** (Continued): Application of calcium correction formulas to serum total calcium levels in dogs and cats with hypoalbuminemia is an accurate method of predicting the true serum total calcium level.

**Pulse oximetry may be least accurate in the most critically ill animals or those receiving oxygen therapy.**
poor correlation between corrected serum calcium and iCa\textsuperscript{2+} levels. In one study\textsuperscript{63} evaluating 1,040 surgical patients requiring intensive care treatment, corrected serum calcium values failed to accurately classify iCa\textsuperscript{2+} status in 38% of the cases. Thus use of the correction formula failed to detect hypocalcemia in a significant number of intensive care patients, leading to underestimation of the prevalence of hypocalcemia and overestimation of normocalcemia. It is suggested that because serum total calcium levels are affected by changes not only in albumin but also in free fatty acids, globulins, acid–base status, and lactate, phosphate, citrate, and hydroxybutyrate levels, it is inappropriate to correct solely for a low albumin level, especially in critically ill patients.\textsuperscript{67,68} Therefore, because the ionized fraction of calcium is the physiologically active form and iCa\textsuperscript{2+} analyzers are widely available, it is recommended that albumin correction formulas be abandoned in favor of measuring iCa\textsuperscript{2+} levels in dogs and cats when evaluating calcium status.

**Misconception:** Ice-water baths are effective and necessary for cooling heatstroke patients.

**Reality:** The most important component of treating heatstroke patients is quick and efficient core cooling to prevent thermal injury to vital organs. A retrospective study\textsuperscript{69} of dogs with heatstroke showed a mortality rate of 42% in dogs that were not cooled by their owners before presentation compared with 19% in dogs whose owners began cooling methods in advance. This suggests that early initiation of cooling is essential to a successful outcome. However, care must be taken to avoid excessive cooling, which may instead induce heat-provoking mechanisms, iatrogenic hypothermia, or even more serious complications.

Ice-water baths should be avoided because they can induce peripheral vasoconstriction, thereby shunting warm blood to the core and inhibiting heat loss. Shivering also occurs in response to extreme cold, thereby producing more heat and counteracting efforts to cool patients. Ice-water baths may also cause capillary sludging and induce disseminated intravascular coagulation.\textsuperscript{70,71} Instead, recommended cooling methods include cool-water rinses, fans, administration of cool intravenous fluids and cool-water enemas, and cool gastric or peritoneal lavage.\textsuperscript{70–73} Regardless of the method chosen, the goal should be to reduce the patient’s temperature to 103°F (39.4°C).\textsuperscript{70,71} It is important not to reduce the temperature below this level because iatrogenic hypothermia could occur, resulting in a poorer prognosis.\textsuperscript{69}

**Misconception:** Anticholinergics are indicated to treat bradycardia caused by medetomidine use.

**Reality:** α\textsubscript{2}-Agonists are useful in providing sedation, analgesia, anxiolysis, and muscle relaxation in dogs and cats.\textsuperscript{74–76} Medetomidine is the most specific α\textsubscript{2}-agonist licensed for clinical use in veterinary medicine and centrally stimulates receptors to produce dose-dependent sedation and analgesia. The typical negative cardiovascular effects produced by other α\textsubscript{2}-agonists (i.e., bradycardia, bradyarrhythmias, reduced cardiac output, hypertension with or without hypotension) also occur with medetomidine use, warranting precautions when it is used and necessitating appropriate patient selection (i.e., young to middle-aged healthy animals). Although hypotension may occur, sedative doses of medetomidine (dogs: 10 to 20 µg/kg IV and 20 to 40 µg/kg IM; cats: 10 to 40 µg/kg IV and 40 to 80 µg/kg IM) typically raise the blood pressure because of effects on peripheral α\textsubscript{2}-adrenoreceptors.\textsuperscript{76} Anticholinergic (i.e., atropine or glycopyrrolate) premedication has been recommended with medetomidine use to prevent bradyarrhythmias and reduced cardiac output.\textsuperscript{77} This recommendation has been widely accepted by most veterinary practices. However, current research does not demonstrate a clear improvement in cardiovascular function. Overall, the anticholinergic-induced increase in heart rate potentiates α\textsubscript{2}-agonist–mediated hypertension\textsuperscript{78–80} and may increase myocardial oxygen tension, demand, and workload.\textsuperscript{79} A simplified illustration of this phenomenon is a garden hose with a nozzle that is closed off so that it can release only a small amount of water. If the water supply is increased, the pressure in the hose increases, but more water does not necessarily come out of the nozzle (i.e., systemic vascular resistance is increased, but not cardiac output, resulting in increased myocardial oxygen demand and consumption).

During evaluation of calcium status, albumin correction formulas should be abandoned in favor of measuring ionized calcium levels in dogs and cats.
Typically, preemptive administration of an anticholinergic prevents reduction in heart rate and related bradyarrhythmias associated with medetomidine use but may cause initial tachycardia and even induce certain dysrhythmias.78,79 Dysrhythmias characterized by heart block, premature ventricular contractions, and tachycardia have been noted with the use of anticholinergic and α2-agonist combinations, especially if the anticholinergic is administered concurrently rather than before the α2-agonist.79,80

Atropine or glycopyrrolate administered before, with, or after medetomidine (30 to 60 µg/kg) also resulted in heart block, premature ventricular contractions, and tachycardia.81 Other studies78 have shown that preemptive administration of atropine in medetomidine-sedated dogs induced hypertension and pulsus alternans—an alternating strong and weak pulse suggesting cardiovascular compromise in humans.

At this time, routine use of anticholinergics with sedative doses of medetomidine does not appear to be beneficial and may even be detrimental. Concurrent administration of anticholinergics and medetomidine cannot be recommended. Decreased doses of medetomidine compared with the recommended label dose should be considered in combination with other sedatives to enhance sedation and analgesia and lower the duration and potential severity of negative cardiovascular side effects. A primary difference in how α2-agonists are used in animals versus humans is the dose. Human doses are much lower than labeled doses in animals.76 Although there may be obvious species differences, lower doses of α2-agonists are usually adequate in veterinary species when used in combination with other sedatives. Decreased doses of medetomidine ranging from 2 to 10 µg/kg have been combined with various preanesthetics (e.g., butorphanol, oxymorphone, hydromorphone, midazolam) to enhance sedation and analgesia while reducing the duration of adverse cardiovascular effects associated with the drug’s use.76 In general, in any emergency involving the use of medetomidine alone or as premedication to general anesthesia, reversal of negative cardiovascular effects with an α2-antagonist (e.g., atipamezole) is the most appropriate treatment rather than administration of an anticholinergic.76

CONCLUSION

Advances in veterinary and human medicine are constantly challenging practitioners’ knowledge and understanding of emergency and critical care medicine. It is imperative that previously held beliefs be reevaluated to avoid perpetuating outdated and disproved medical practices. New research has refuted many beliefs and procedures in emergency and critical care in intensive care units. Veterinarians should constantly strive to keep pace with the ever-changing realm of emergency and critical care to provide the highest quality of patient care.

REFERENCES


ARTICLE #2 CE TEST

This article qualifies for 2 contact hours of continuing education credit from the Auburn University College of Veterinary Medicine. Paid subscribers may purchase individual CE tests or sign up for our annual CE program. Those who wish to apply this credit to fulfill state relicensure requirements should consult their respective state authorities regarding the applicability of this program. To participate, fill out the test form inserted at the end of this issue or take CE tests online and get real-time scores at CompendiumVet.com. Test answers are available online free to paid subscribers as well.

1. NaHCO$_3$ administration is indicated for patients with
   a. ketoacidosis.
   b. lactic acidosis.
   c. inorganic acidosis.
   d. respiratory acidosis.

2. Which statement regarding dextrose administration to patients experiencing seizures is incorrect?
   a. Hypoglycemia is more common in puppies than adults with seizures.
   b. Hyperglycemia occurs only during the very late stages of status epilepticus.
   c. Hyperglycemia is associated with proconvulsant effects due to increased neuronal hyperexcitability.
   d. Hyperglycemia is associated with increased mortality rates in critically ill humans.

3. With which ketone body in the urine does the sodium nitroprusside pad on the urine reagent test strip react?
   a. acetoacetic acid
   b. acetone
   c. β-hydroxybutyric acid
   d. lactic acid

4. What is the best way to determine whether an animal has developed a catheter-associated UTI?
   a. Culture the catheter tip following removal of the indwelling urinary catheter.
   b. Obtain a urine sample using the catheter, and submit it for culture before removing the urinary indwelling catheter.
   c. Initially culture the urinary catheter tip, but also culture a urine sample a few days following catheter removal.
   d. Obtain a free-catch sample for culture following removal of the indwelling urinary catheter.

5. Which factor does not interfere with pulse-oximeter readings?
   a. poor peripheral perfusion
   b. ambient light
   c. probe placement
   d. lack of skin pigmentation

6. What effect occurs with sodium heparin anticoagulant use in blood gas analysis?
   a. false increases in iCa$^{2+}$ levels
   b. false increases in pH
   c. false decreases in P$_{CO_2}$
   d. false decreases in sodium

7. Which statement regarding analgesic administration in critically ill patients is incorrect?
   a. Analgesics may mask physiologic indicators of patient deterioration.
   b. Appropriate analgesics are necessary to reduce stress and enable sleep and healing.
   c. Analgesics should be avoided in obtunded patients.
   d. Patients with neurologic disorders should be treated appropriately with analgesics because pain can increase intracranial pressure.

8. Which alter(s) serum total calcium levels?
   a. free fatty acids
   b. acid–base status
   c. albumin
   d. all of the above

9. Which statement regarding the management of heatstroke patients is correct?
   a. Ice-water baths are the most effective method of cooling these patients.
   b. It is essential to cool heatstroke patients to 101˚F (38.3˚C) or less.
   c. Early initiation of cooling leads to better patient outcome.
   d. Ice-water baths draw warm blood to the periphery, thereby enabling rapid heat loss.

10. Which treatment is not indicated in preventing or treating medetomidine-induced bradycardia?
    a. Decrease the dose of medetomidine, and administer it concurrently with an opioid.
    b. Decrease the dose of medetomidine, and administer it concurrently with midazolam.
    c. Administer atropine or glycopyrrolate before or with medetomidine.
    d. Reverse the effects of medetomidine with atipamezole.