

INTRODUCTION

The history and physical examination is an important part of the 'equation' when assessing the acid-base status of the patient. Many conditions are associated with alterations in the metabolic status of a patient, for example, diabetic ketoacidosis may result in profound acidosis, while upper gastrointestinal vomiting is frequently associated with alkalosis. Assessment of the acid-base status of a patient in the emergent and critical care setting is one of the most valuable tools a clinician can use to evaluate the severity of illness or injury, the pre- and post-operative condition of the patient, the success of therapy, to assist in diagnosis, to select and formulate the 'prescription' for fluid therapy, and to influence many other therapies, especially the delivery of electrolytes. As you review the remainder of this manual you will see how frequently the acid-base status of the patient is considered when directing therapy.

The *non-respiratory (metabolic)* status of patients can be derived from information that all clinics are able to obtain on a biochemical profile as a blood gas machine is not required for a 'ballpark' assessment. Abnormalities relating to the *non-respiratory* component are more common than the *respiratory* component of acid-base disorders in veterinary practice. To assess the *respiratory* component contributing to the acid-base status, and a more thorough evaluation of the *non-respiratory* component, PaCO₂ or PvCO₂ is required and therefore, a blood gas machine is necessary. In addition to a primary abnormality in either the *respiratory* or *non-respiratory* component, a secondary effect occurs as the body attempts to maintain a neutral pH so the *respiratory* and *non-respiratory* components will respond (change) to an abnormality in the other compartment to approach this neutrality. In addition, electrical neutrality has to be maintained and it is on this basis that one can use electrolytes and weak anions (plasma proteins, HCO₃⁻, phosphate) to assess the acid-base disorder. Only the essentials will be presented here. The elements of the equations will be used to illustrate various aspects rather than the lengthy, but fascinating, physiological reasons for these alterations. As you can imagine, the topic is a very complex one and you are referred to the suggested reading for more details. The values given here will vary from laboratory to laboratory and with different analyzers, as do published normals, but the information will be able to give a 'ballpark' assessment. As a day-to-day tool for assessing the patient's condition, a very simplified version is also presented that does not require a blood gas analyzer. However, even if you do not possess a blood gas analyzer, the first part of the discussion will help in understanding the 'biochemical profile-only' approach.

The terms acidosis and alkalosis refer to the pathophysiologic process that causes a net accumulation of an acid or alkali in the body. The terms acidemia and alkalemia refer to the pH of the blood. The patient would be acidemic if the underlying problem resulted in acidosis due to H⁺ gain or HCO₃⁻ loss.

Assessment of acid-base status using blood gas information

When assessing the acid-base status of the patient, venous blood gases are more commonly used as venous blood is easier to collect than arterial blood, and is a better reflection of the metabolic status of the body. As much of the earlier research was performed on arterial blood, normal values may differ by a small number, except PO₂ which is dramatically different between the two samples. Arterial blood gases are discussed in *Oxygen Supplementation* p. 577. First, identify if the patient is normal and if not, what is the primary disturbance? Is the patient acidemic (decreased pH of blood) or alkalemic (increased pH of blood), normal values ~ 7.33 – 7.38? If acidemic or alkalemic look at the PvCO₂ to see if it is within normal range, ~38 – 46 depending on sampling site. If pH is high and PCO₂ low, there is a respiratory alkalosis, if pH is low and PCO₂ high, there is a respiratory acidosis. If the base excess (BE) or adjusted base excess (ABE) is less than normal (0±4) i.e., has a negative value, or otherwise stated, a base deficit exists, and the pH is less than normal, there is a *non-respiratory acidosis*; if the BE or ABE has a positive value, or a base excess, with a higher than normal pH, there is a *non-respiratory alkalosis*. The normal [HCO₃⁻] is 20±4, if higher with an increased pH then there is a *non-respiratory alkalosis*, if lower with a decreased pH, a *non-respiratory acidosis* is present. Where a high PCO₂ exists, the [HCO₃⁻] will be increased as CO₂ is carried as H₂CO₃ (i.e., increased CO₂ shifts the equilibrium to yield more HCO₃⁻ and H⁺). In this setting the pH is usually low with *respiratory* acidosis. A normal pH does not imply the patient is normal. As an example, a mixed acid-base picture may exist where a primary *respiratory alkalosis* will raise the pH of a patient with a *non-respiratory acidosis*, the two abnormal pH's average out to a normal pH, therefore, a pH within normal range requires further investigation to rule out a mixed acid-base disorder. When the abnormality is identified as *respiratory* or *non-respiratory*, identify whether the secondary response is appropriate. In this situation the pH is not normal but approaching normal. An example would be a patient with diabetic ketoacidosis [HCO₃⁻] is low due to an increase in unmeasured anions (ketoacids) and the PCO₂ is decreased via a hyperpneic respiratory pattern

(Kuhsmal) in an attempt to raise the blood pH. This secondary, or compensatory, response is a change in the PCO_2 in the same direction as the HCO_3^- is shifting i.e., when the pH is decreased in *non-respiratory acidosis* there is a 0.7 mmHg decrease in PCO_2 for each 1.0 mEq/L decrease in plasma $[\text{HCO}_3^-]$; or the converse when the pH is increased in *non-respiratory alkalosis*. During *respiratory acidosis*, the pH is decreased but the PCO_2 is increased; the HCO_3^- also increases, but by 0.15 mEq/L within 24h and 0.35 mEq/L >48h. In respiratory alkalosis the pH is increased but the PCO_2 is decreased and an appropriate response is a decrease in HCO_3^- by 0.25 mEq/L for each mmHg PCO_2 decrease. However, the HCO_3^- shift decreases by 0.55 mEq/L after 48 hours. The reason for the delayed response is that HCO_3^- shift requires renal excretion which commences within hours of the alteration in PCO_2 but may take up to 5 days (potentially longer) to be complete. Where the secondary response is appropriate, this is referred to as a *simple disorder* as it is limited to the primary abnormality. However, more than one *primary* acid-base disorder may coexist and the pH may be normal or abnormal; this is a *mixed disorder*.

A common situation is a sick animal with a *non-respiratory acidosis* but is anxious and panting resulting in a *respiratory alkalosis*. Here the pH may be normal, or increased if the anxiety and panting are severe. As an example of the effect hyperventilation may have on acid-base, this author recalls a healthy dog with a pH 7.55 due to a PvCO_2 of 19 mmHg and was obviously very stressed based on behaviour. Administration of 0.3 mg/kg acepromazine eased the anxiety resulting in a return towards a normal respiratory pattern and a subsequent rise in the PvCO_2 to 30 mmHg thus lowering the pH. A similar respiratory effect may be seen when a hypovolemic (*non-respiratory acidosis*) animal is tachypneic (*respiratory alkalosis*) as a normal response to low volume state. The pH may be normal but this is due to the low CO_2 (*respiratory alkalosis*) 'masking' the hypovolemic lactic acidosis. In this instance the *mixed disorder* is detected because the expected compensatory response to *non-respiratory acidosis* exceeds the magnitude of change expected for just compensation. If the CO_2 had been normal or increased, then the magnitude of change for compensation would fall short of that expected indicating the presence of a *primary respiratory acidosis*. Respiratory depression (e.g., head injury) or lung injury (e.g., pulmonary contusions) should then be considered. It is important to identify the primary disorder as this influences treatment. Again, history and physical examination should be considered in the evaluation. Where *non-respiratory acidosis* is detected, a balanced electrolyte (BES) alkalizing solution should be selected. If this is associated with a *primary respiratory alkalosis*, as could be present in the anxious patient, and the pH is normal or slightly increased, therapy to correct this primary disorder should be given (i.e., analgesia for pain, sedative for anxiety). Do not treat with 0.9% sodium chloride as an acidifying solution. Likewise, a *non-respiratory alkalosis* due to loss of chloride as a primary problem, with a *primary respiratory acidosis* i.e., drug-induced bradypnea, or over-zealous furosemide administration and associated hyponatremic neurological disorder lowering the pH, should not be given an alkalizing solution as the sodium chloride deficit must be corrected to correct the *primary non-respiratory disorder*.

While it has been said that a PO_2 value in a venous sample cannot be used to assess oxygenation, this is not entirely so. A SpO_2 value <70 mmHg from a central vein catheter suggests low oxygenation (i.e., inadequate oxygenation due to lung pathology or oxygen availability), or increased extraction (e.g., sepsis, inflammation). A jugular venous blood sample with a PO_2 value <30 mmHg, and saturation <50%, suggests a significant lack of oxygen. The history and physical examination will identify the primary problem causing this. In addition, one would expect to see a *non-respiratory acidosis* as the reduced oxygen delivery to tissues, or increased requirement, would cause a lactic acidosis. A primary or secondary (compensatory) *respiratory alkalosis* would accompany this finding if there was no lung pathology.

Blood gases are useful in identifying the acid-base disturbance as that resulting from a *respiratory* or *metabolic (non-respiratory)* problem, the secondary or compensatory response, and whether a mixed disturbance is present. The *traditional approach* to acid-base evaluation focuses on the relationship between pH, PCO_2 and HCO_3^- as described by Henderson-Hasselbalch equation where pH is shown to be a function of $[\text{HCO}_3^-]$ and PCO_2 . The PCO_2 being the respiratory component determined by alveolar ventilation, and the $[\text{HCO}_3^-]$ being the non-respiratory component controlled by the kidneys. The implication is that both are independent variables, that is, their changes are not influenced by other aspects considered in acid-base assessment. In fact $[\text{HCO}_3^-]$ is dependent on many other aspects of acid-base balance i.e., a primary increase in PCO_2 results in H_2CO_3 (carbonic acid) which dissociates into H^+ and HCO_3^- . Here, HCO_3^- is dependent on PCO_2 . Serum electrolytes and plasma proteins also contribute to alterations in acid-base status as their concentrations influence the $[\text{HCO}_3^-]$. The *non-traditional approach (Stewart's Theory)* where consideration is given to the maintenance of electrical neutrality (cations must equal anions), conservation of mass, and satisfaction of dissociation equilibria for incompletely dissociated solutes, also considers the role of electrolytes, and the total concentration of weak acids (i.e., proteins, especially albumin and phosphates). With this information assessment of the *non-respiratory* acid-base status can be better differentiated with information from the biochemical profile. It should be understood that there is variation in data obtained from the different analyzers used, and the various normal values published so a definitive, or totally correct, answer may not be obtained in some cases. However, in most instances assessing the acid-base status of your patient will be helpful in case management.

Assessment of acid-base status using biochemical profile information

From a clinical standpoint it is important to ask: Is the patient acidemic or alkalemic? Is the pH within normal range? To answer these questions one needs to know the pH. However, this is not given specifically on a biochemical profile but can be elucidated. The question: 'Is the acid-base status normal?' can be answered using information from the biochemical profile. The anion gap [AG] equations (1) and (2) and the strong ion difference [SID] equations (3) and (4) will give us this information. Using this information and equation (5), the base excess can also be estimated using equation (6). Starting with the AG it is important to note, there is no 'gap'. This term represents anions that can (albumin- and inorganic phosphate-), and cannot (e.g., uremic toxins, ketoacids, ethylene glycol) be measured in the most part in pathologic states. As electroneutrality and mass have to be conserved, the 'gap' represents unmeasured anion(s), and in humans has been estimated at approximately 2 x total protein (g/dL). However this is a crude estimate as the gap is greater in dogs due to the higher protein charge. From the biochemical profile, the total CO₂ (tCO₂ = 13 – 24 mmol/L) is a good estimate of the [HCO₃⁻] for use in the formula below, while the Na⁺, K⁺, Cl⁻ are easily obtained. The low value here may be due to handling (*see Technical Aspects below*).

Equations 1 & 2 define a *non-respiratory* disorder due to an **unmeasured anion imbalance**.

1. **The AG = (Na⁺ + K⁺) – (Cl⁻ + HCO₃⁻) = 12 – 24 mEq/L in dogs and 13 – 27 mEq/L in cats.**

As there are many other unmeasured anions, especially during illness or injury, the equation is better utilized by manipulation to:

(Na⁺ + K⁺ + Unmeasured Cations) = (Cl⁻ + HCO₃⁻ + Unmeasured Anions). As the change in UC (magnesium, calcium) would have to be small to be compatible with life, the equation is essentially:

Na⁺ + K⁺ = Cl⁻ + HCO₃⁻ + UA⁻, which is:

2. **UA⁻ = (Na⁺ + K⁺) – (Cl⁻ + HCO₃⁻).**

When the patient's values (these are values for dogs, extrapolated to cats, as values are not available for cats) are plugged into this equation, the UA⁻ (or gap) will be lower, higher or the same as the values given in equation (1) above. A low anion gap is very rare and is commonly associated with hypoalbuminemia (each decrease of 10 g/L [1 g/dL] in albumin is associated with a decrease of 4.1 mEq/L in the AG, whereas each decrease of 1 g/dL total protein is associated with a decrease of 2.5 mEq/L in AG which will be discussed later). The AG differentiates between hyperchloremic and high-AG metabolic acidosis (HCO₃⁻ is decreased in both situations). To maintain electrical neutrality, the decrease in HCO₃⁻ may be associated with an increase in Cl⁻, which results in a normal AG, or an increase in UA⁻, which results in a high AG. The increased UA⁻ contributes to acidosis as does the high Cl⁻. Plasma [Cl⁻] and [HCO₃⁻] move in opposite directions in non-respiratory acidosis and alkalosis where UA⁻ are unchanged. For example, the mechanism for alkalosis following administration of furosemide or gastric vomiting, where Cl⁻ is lost, results in an increase in [HCO₃⁻]. Sodium concentration tends to stay within normal limits in acid-base disorders, unless the primary problem also affects sodium. The cations rarely contribute to changes unless sodium is lost or gained in excess of Cl⁻. This is where the strong ion difference (SID) comes in.

Equation 3 calculates the SID which defines a **non-respiratory disorder due to an electrolyte imbalance**.

3. **SID = (Na⁺ + K⁺ + Ca²⁺ + Mg²⁺) – Cl⁻** is referred to as the *apparent* SID. For clinical purposes this SID_{app} = Na⁺ – Cl⁻ = 32 – 40 mEq/L (36 mEq/L frequently used).

As the main components are Na⁺ and Cl⁻ the difference (Na⁺ – Cl⁻) can be used as a clue to the problem.

Obtaining the difference between the [Na⁺] and [Cl⁻] is useful in assessment of mixed disorders, or metabolic disturbances not associated with an increase in UA⁻. Changes in [Na⁺] occur with changes in water balance, contraction or dilution of the plasma volume. However, Cl⁻ can change for reasons independent of water balance. To identify this and remove the water component, the correction for [Cl⁻] must be made in conjunction with measurement of [Na⁺].

4. **Dogs: [Cl⁻]_{corrected} = [Cl⁻]_{patient} x 146/[Na⁺]_{patient} and for cats: [Cl⁻]_{corrected} = [Cl⁻]_{patient} x 156/[Na⁺]_{patient}**

Normal [Na⁺] for dogs is 146 mEq/L and for cats [Na⁺] is 156 mEq/L. [Cl⁻]_{corrected} for dogs is 107 – 113 mEq/L and for cats 117 – 123 mEq/L. Where [Na⁺] is normal and [Cl⁻] is low, the SID is increased above 36 mEq/L representing a non-respiratory alkalosis; if the SID is decreased due to an increased [Cl⁻], a non-respiratory acidosis exists. If the SID is normal, with normal [Na⁺] and [Cl⁻] and the AG is normal, the metabolic status

of the patient is normal. However, if the AG is increased, a non-respiratory acidosis is present due to UA⁻. The UA⁻ are likely lactate, ketoacids, sulfate, phosphate, or very rarely increased protein. Sometimes the SID can be normal because the [Na⁺] and [Cl⁻] are both low but the difference is still close to 36 mEq/L. This can occur in large volume, high gastric vomiting with loss of chloride and the effect of anti-diuretic hormone conserving water (volume) with dilution of sodium, or the patient drinking a lot of water. So looking at the SID alone may not help with assessing the acid base status if sodium is abnormal. However, when one looks at the AG equation, the Cl⁻ will be low, and the HCO₃⁻ may be reciprocally high (normal AG) (in the early stages) indicating a *non-respiratory alkalosis*. In this case 0.9% sodium chloride would be the fluid of choice to increase both Na⁺ and Cl⁻. The Cl⁻ in this solution is in excess of Na⁺ which would then replace the Cl⁻ debt, reduce the [HCO₃⁻] and correct the alkalosis.

While this is simplified into two categories either a problem with the SID or a problem with the AG, both may be used to identify a *mixed non-respiratory disorder*. Using our vomiting patient above, if the HCO₃⁻ is lower than expected, the AG will be increased indicating an excess of UA⁻ which would suggest lactic acidosis if the patient was hypovolemic/hypotensive due to fluid loss through vomitus (later stage). In this case 0.9% sodium chloride would still be indicated to replace the losses, but a larger volume of fluid would be required as there is indication of hypovolemia with perfusion deficits in this instance whereas the patient in the previous example may not require such a large volume of fluid as it is in the earlier stages of fluid losses.

If the SID is low the [Na⁺] may be low due to losses in excess of Cl⁻ such as in diarrhea. If the [Cl⁻] is normal and volume loss is minimal the AG may be normal as a minimal drop in [HCO₃⁻] may offset the low [Na⁺] in the calculation. With large volume small bowel diarrhea, Na⁺ and HCO₃⁻ losses may be severe resulting in a hyperchloremic acidosis. If volume loss is enough to reduce perfusion, then the AG may be high due to lactic acidosis increasing the UA⁻. This combination results in a severe non-respiratory acidosis. This situation may be managed by fluid selection and adding 5% sodium chloride at 0.5 – 1.0 mEq/kg/h. The SID may also be decreased due to an increase in [Cl⁻] with normal [Na⁺]. This may occur when 0.9% sodium chloride is used as the [Na⁺] and [Cl⁻] are equal, raising the serum [Cl⁻] relative to [Na⁺] resulting in a non-respiratory, normal AG, acidosis. The addition of potassium chloride (KCl) to intravenous fluids is routine. If a high [KCl] is required to maintain K⁺ within normal range, the [Cl⁻] can increase enough to cause a non-respiratory acidosis. Lowering the administered Cl⁻ is necessary to correct the acidosis and this can be achieved by using potassium phosphate (this will have only a minimal effect on the anion gap) instead of potassium chloride (see #7 in *Selection of Fluid* p. 410).

Other changes in SID

Aside from changes in our typical calculated SID due to electrolyte abnormalities ([Na] + [K] – [Cl]), free water abnormalities also change the true SID. A deficit of free water will proportionally increase all strong cations and anions and therefore, SID (the so-called concentration alkalosis). SID increases 1 mEq/L for each 4 mEq/L (4.4 mEq/L in cats) increase in Na⁺ concentration. Dilutional acidosis occurs during hyponatremia where a decrease in 4 mEq/L in sodium concentration is associated with a 1 mEq/L decrease in SID.

Plasma Proteins⁻ and Phosphate⁻ [A_{TOT}]

Plasma proteins⁻ and phosphate⁻ also influence the acid-base status as they are weak anions and are included in the UA⁻ part of the equation where they are referred to as ATOT. Proteins contribute a charge of approximately 12 (cats) or 16 (dogs) to the UA⁻ part of the equation in normal individuals. As one can see from equations (1) and (2) as plasma proteins decrease, such as in hypoalbuminemia, which is quite common in severely ill animals, the anions in the equation have to increase to maintain electrical neutrality. The [HCO₃⁻] frequently increases causing a *non-respiratory alkalosis*. If hypoalbuminemia is accompanied by a situation that results in generation of UA⁻ (i.e., lactate due to poor perfusion), then the AG could be unchanged, or increased (although not to the extent typically seen in situations of lactic acidosis where albumin is normal) if the UA⁻ caused a reduction in [HCO₃⁻] as it is used as a buffer. Hypoalbuminemia, therefore, can mask the severity of an acidosis. As other buffers, such as hemoglobin, can also buffer the UA⁻, a reduction in [HCO₃⁻] may not occur early in the disease process. As phosphate is considered in the [A_{TOT}], severe hyperphosphatemia can cause a *non-respiratory acidosis* due to decrease in [HCO₃⁻].

To identify the *true base excess or deficit* and the existence of unmeasured anions, the changes in SID influence [A_{TOT}] must be considered. To calculate the change in [A_{TOT}], the following calculations can be used:

5. Other changes in [A_{TOT}]

Changes in the components of A_{TOT} will impact the AG. A decrease of 10 g/L [1 g/dL] in albumin will decrease AG roughly 4 mEq/L, whereas a change of 10 g/L [1 g/dL] in TP will change the AG, in the same direction, by 2.5 mEq/L. For example, if the TP is decreased by 20 g/L [2 g/dL], the AG is expected to be 5 mEq/L less than normal. If the AG is normal, this may represent an increase in UA⁻ of 5 mEq/L. An increase in phosphate can also increase the AG (e.g., toxicity following the use of a hypertonic sodium phosphate enema in cats).

Since many patients with increased unmeasured strong anions also have hypoalbuminemia (typical of critical illness), the AG may be artificially normal because of the decrease in $[UA^-]$ resulting from hypoalbuminemia. The AG can be corrected for changes in protein concentration in dogs by using the following formulas.

$$AG_{\text{Alb-adjusted}} = AG + 4.2 \times [(3.77 - [\text{alb}])]$$

OR

$$AG_{\text{TP-adjusted}} = AG + 2.5 \times [(6.37 - [\text{TP}])]$$

where $[\text{alb}]$ is albumin concentration in g/dL and $[\text{TP}]$ is total protein concentration in g/dL.

Although contribution of phosphate concentration to the AG is negligible in normal dogs and cats, hyperphosphatemia can also increase the AG in the absence of an increase in strong unmeasured anions. The AG can be adjusted to increase in phosphate concentration by expressing phosphate in mEq/L and assuming plasma pH to be 7.4 as:

$$AG_{\text{alb-phosph-adjusted}} = AG + 4.2 \times [(3.77 - [\text{alb}])] + (2.52 - 0.58 \times [\text{Phosph}])$$

OR

$$AG_{\text{TP-phosph-adjusted}} = AG + 0.25 \times [(63.7 - [\text{TP}])] + (2.52 - 0.58 \times [\text{Phosph}])$$

where $[\text{Phosph}]$ is the concentration of phosphorus in mg/dL.

6. **Base excess (BE)** can be crudely estimated by considering all the changes in the metabolic state discussed above using the equation:

BE = change in SID due to electrolyte abnormalities – change in AG (since a rise in AG is acidosis) + change in SID from free water abnormalities. Note that the change in AG accounts for protein abnormalities.

Selection of fluids to correct acid-base disturbances (see *Fluid Therapy* p. 347)

Crystalloids available are:

1. Balanced electrolyte solutions (BES), which are sodium based with a similar composition of electrolytes to that of plasma. Most have acetate + gluconate, or lactate as bicarbonate precursors and are therefore, alkalinizing solutions. These solutions are recommended for treating *non-respiratory acidosis*.
2. Sodium chloride (NaCl) 0.9%, which has 154 mEq/L Na^+ and 154 mEq/L Cl^- . This is an acidifying solution as the $\text{SID} = 0$ and an increase in Cl^- will require the loss of another anion, which is usually HCO_3^- . The administration of 0.9% sodium chloride has been reported to be a common reason for acidosis in humans as it is used as a standard treatment rather than specifically for treating *non-respiratory alkalosis*. The addition of KCl to fluids also increases the $[\text{Cl}^-]$, contributing to acidosis. 0.9% NaCl is recommended for treating *non-respiratory alkalosis* as both Na^+ and Cl^- may be low, or the SID is increased due to hypochloremia.
3. Maintenance alkalinizing solutions contain 40 mEq/L Na^+ and Cl^- and acetate + gluconate, with or without 5% dextrose. These may be used if a reduction in Cl^- is desired and an alkalinizing solution is indicated.
4. Half-strength of the BES solution (1:1 sterile water BES) may be administered as a substitute for (3) above if this is not available, or if 5% dextrose is not wanted.
5. Half-strength (0.45%) NaCl may be indicated in hypernatremia and water loss (see *Hyper/Hyponatremia* p. 381/386), or as a maintenance solution.
6. Most synthetic colloid solutions have a 0.9% NaCl base and are, therefore acidifying solutions.
7. Where a reduction in $[\text{Cl}^-]$ is required but supplementation with K^+ is necessary, potassium phosphate may be used as a substitute for KCl. Volumes and rate of administration of the various fluids should also be considered in the fluid plan, as well as alterations in electrolytes, especially potassium, as acid-base status is improved (see *Fluid Therapy* p. 347, *Hypokalemia/Hyperkalemia* p. 394/396).

SUGGESTED READING

1. Constable PD, Stämpfli HR, Experimental determination of net protein charge and $A(\text{tot})$ and $K(\text{a})$ of nonvolatile buffers in canine plasma. *J Vet Intern Med.* 2005;19(4):507-14.
2. DiBartola SP, de Moraes HA. Section on Acid-Base Disorders. In *Fluid, electrolyte and Acid-Base Disorders in Small Animal Practice* 3rd ed. Elsevier, Philadelphia 2006. In Press.