**INVESTIGATION**

Determine if arterial or venous
- Venous: Jugular sample provides the best idea of whole-body status. Peripheral samples may represent local tissue bed and not whole body.
- Arterial: Dorsal metatarsal artery, femoral artery, auricular artery, or caudal artery

**INVESTIGATION**

Arterial sample (to assess respiratory function)
- PaCO₂ assesses ability to ventilate
- PaO₂ assesses ability to oxygenate

**INVESTIGATION**

Venous sample (pulse oximetry can be used with venous blood gas to assess oxygenation)
- PvCO₂ can suggest ventilation (usually about 5 mm Hg higher than PaCO₂)

**INVESTIGATION**

Evaluate pH to determine if acidemia or alkalemia present

**INVESTIGATION**

Evaluate PCO₂ and BE for masked disturbances if pH = 7.35–7.45

**INVESTIGATION**

Acidemia = pH < 7.35
- Determine if metabolic or respiratory in origin

**INVESTIGATION**

Alkalemia = pH > 7.45
- Determine if metabolic or respiratory in origin

**RESULTS**

Respiratory = PaCO₂ > 45 mm Hg

**RESULTS**

Metabolic = BE < -4 mmol/L (or HCO₃⁻ < 21 mEq/L)

**RESULTS**

Respiratory = PaCO₂ < 35 mm Hg

**RESULTS**

Metabolic = BE > 4 mmol/L (or HCO₃⁻ > 27 mEq/L)

**DIFFERENTIAL**

Respiratory Acidosis
- Depressed respiratory center
- Cervical spinal cord disease
- Neuromuscular disease
- Pleural space disease
- Airway obstruction
- Rarely severe pulmonary parenchymal disease
- Respiratory muscle fatigue

**DIFFERENTIAL**

Metabolic Acidosis
- Increased anion gap:
  - Ketones
  - Lactate
  - Uremia
  - Toxicity (ethylene glycol, salicylates)
- Normal anion gap:
  - HCO₃⁻ loss through kidneys or intestinal tract

**DIFFERENTIAL**

Respiratory Alkalosis
- Hypoxemia
- Pulmonary disease
- CNS disease (stimulating respiratory center)
- Exercise, pain, stress

**DIFFERENTIAL**

Metabolic Alkalosis
- GI obstruction with loss of H⁺, K⁺, and especially Cl⁻ in vomitus
- Loop diuretics
- NaHCO₃ administration

**TREATMENT**

- Correct underlying problem
- Relieve airway obstruction/restrictive disease (pleural space)
- Intubate, begin PPV

**TREATMENT**

- Correct underlying cause
- Lactate: Improve oxygen delivery to the tissues
- Ketones: Insulin therapy
- NaHCO₃ (if needed)
  - HCO₃⁻ deficit = BE × body weight (kg) × 0.3
  - Give one-fourth to one-third of dose and recheck blood gas

**TREATMENT**

Correct underlying diseases

**TREATMENT**

- Hypokalemic hypochloremic metabolic alkalosis: Volume expansion with 0.9% NaCl
- Loop diuretic or HCO₃⁻ therapy: No treatment, usually self-limiting

Go to the next page to see a list of key terms.
This algorithm reflects canine normals. For cats, substitute feline normals for pH, BE (or HCO$_3^-$), PCO$_2$, and PO$_2$ values (Table 1).

### Rules of Compensation

1. Change in respiratory or metabolic component of the acid-base status will normally induce opposite, compensatory change in the other to return the pH toward normal.
2. Lungs compensate rapidly by changing minute ventilation (respiratory rate/tidal volume/both) within minutes.
3. Metabolic compensation occurs via the kidneys and is much slower, starting after a few hours and requiring 4 to 5 days for maximum compensation.
4. Absence or presence and degree of compensation for respiratory disturbance can give an idea of chronicity (Table 2).
5. Overcompensation does not occur.
6. If expected compensation is absent, a mixed disturbance is present. For example, if metabolic acidosis is not accompanied by compensatory respiratory alkalosis (the CO$_2$ is normal or increased), a mixed disturbance is occurring with both metabolic acidosis and respiratory acidosis.

### TABLE 1

**NORMAL VALUES FOR BLOOD GASES**

<table>
<thead>
<tr>
<th></th>
<th>Arterial</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CANINE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.35–7.45</td>
<td>7.35–7.45</td>
</tr>
<tr>
<td>PO$_2$ (mm Hg)</td>
<td>90–100</td>
<td>30–42</td>
</tr>
<tr>
<td>PCO$_2$ (mm Hg)</td>
<td>35–45</td>
<td>40–50</td>
</tr>
<tr>
<td>HCO$_3^-$ (mmol/L)</td>
<td>20–24</td>
<td>20–24</td>
</tr>
<tr>
<td>BE (mmol/L)</td>
<td>−4–+4</td>
<td>−4–+4</td>
</tr>
<tr>
<td><strong>FELINE</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.34 ± 0.1</td>
<td>7.30 ± 0.08</td>
</tr>
<tr>
<td>PO$_2$ (mm Hg)</td>
<td>102.9 ± 15</td>
<td>38.6 ± 11</td>
</tr>
<tr>
<td>PCO$_2$ (mm Hg)</td>
<td>33.6 ± 7</td>
<td>41.8 ± 9</td>
</tr>
<tr>
<td>HCO$_3^-$ (mEq/L)</td>
<td>17.5 ± 3</td>
<td>19.4 ± 4</td>
</tr>
<tr>
<td>BE (mmol/L)</td>
<td>−6.4 ± 5</td>
<td>−5.7 ± 5</td>
</tr>
</tbody>
</table>

### TABLE 2

**EXPECTED COMPENSATORY CHANGES**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Primary Change</th>
<th>Compensatory Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>↓ HCO$_3^-$</td>
<td>0.7 mm Hg decrease in PCO$_2$ for each 1 mmol/L decrease in HCO$_3^-$</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑ HCO$_3^-$</td>
<td>0.7 mm Hg increase in PCO$_2$ for each 1 mmol/L increase in HCO$_3^-$</td>
</tr>
<tr>
<td>Acute respiratory acidosis</td>
<td>↑ PCO$_2$</td>
<td>1.5 mmol/L increase in HCO$_3^-$ for each 10 mm Hg increase in PCO$_2$</td>
</tr>
<tr>
<td>Chronic respiratory acidosis</td>
<td>↑ PCO$_2$</td>
<td>3.5 mmol/L increase in HCO$_3^-$ for each 10 mm Hg increase in PCO$_2$</td>
</tr>
<tr>
<td>Acute respiratory alkalosis</td>
<td>↓ PCO$_2$</td>
<td>2.5 mmol/L decrease in HCO$_3^-$ for each 10 mm Hg decrease in PCO$_2$</td>
</tr>
<tr>
<td>Chronic respiratory alkalosis</td>
<td>↓ PCO$_2$</td>
<td>5.5 mmol/L decrease in HCO$_3^-$ for each 10 mm Hg decrease in PCO$_2$</td>
</tr>
</tbody>
</table>
BLOOD GAS ANALYSIS ALTERNATIVES

Author Commentary

If blood gas analysis is not available, some information can be obtained through other tests. Pulse oximetry can be used to assess a patient’s oxygenation. It must be remembered that pulse ox saturation and PaO$_2$ are not directly correlated; a pulse ox of 93% corresponds with a PaO$_2$ of 80 mm Hg and a pulse ox of 90% corresponds with a PaO$_2$ of 60 mm Hg. If the patient is intubated, end tidal CO$_2$ (ETCO$_2$) can be used to assess for hypercarbia or hypocarbia. ETCO$_2$ usually corresponds well to the PaCO$_2$, with ETCO$_2$ approximately 5 mm Hg lower than PaCO$_2$ in normal patients. In medium and large dogs, the end tidal tubing can be placed just inside the nostril of an awake, compliant patient to estimate ETCO$_2$.

If anion gap measurement is available on a chemistry screen, it can be used to detect some causes of metabolic acidosis, including those caused by ketones, lactate, and exogenous acids (eg, toxins, phosphates, sulfates). These will all cause an increase in the anion gap. If the anion gap is normal but the corrected chloride is elevated (see equation below), loss of bicarbonate via the kidneys or the large bowel may be causing a metabolic acidosis.

Additionally, the patient’s ketones may be measured via either ketone strips for use with urine or a ketometer to measure serum ketones. A handheld lactate meter can be used to measure ketones. These are relatively inexpensive (typically cost several hundred United States dollars), small pieces of equipment similar to a glucometer and give results in a few minutes. Although they do not give information about pH, they can help guide fluid therapy and potentially help prognosticate in patients that are presented with a high lactate and do not respond to therapy. To detect a metabolic alkalosis, the patient’s chloride concentration should be evaluated. If the corrected chloride is low, a metabolic alkalosis is likely.

Corrected Cl$^- = (\text{normal Na}^+/\text{measured Na}^+) \times \text{measured Cl}^-$

This equation helps determine if the chloride concentration is abnormal in comparison to the sodium concentration, as normally the 2 ions will change in the same direction and to the same degree. Changes in the corrected chloride can indicate metabolic derangements.