Acid-Base Balance and Respiration
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The subject of acid-base balance has become one of increasing complexity over the past two decades. Along with better understanding of the processes involved has come an array of confusing terms and ideas that leaves most physicians completely bewildered. For example, in a recent article on acid-base balance, the author listed six commonly used chemical definitions of the words "base" and "acid." For the past few years there has been a running international controversy carried on in the pages of Lancet and The New England Journal of Medicine, which has been dubbed The Great Trans-Atlantic Acid-Base Debate. This debate has mostly dealt with methods and terminology of acid-base chemistry. I hope you will, therefore, understand my reluctance in becoming too involved with semantics, various techniques, and complicated chemistry, and permit me to limit this discussion to basic principles. However, I would admonish you to learn as much as you can about acid-base balance, since we in anesthesia are exposed to these problems more often than those in most other fields of medicine.

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THE CHEMISTRY

The electrolytic dissociation theory of Ostwald and Arrhenius states that when acids and bases are placed in watery solution, they tend to dissociate and form their respective ions.

\[
\begin{align*}
\text{HCl} & \rightarrow \text{H}^+ + \text{Cl}^- \\
\text{NaOH} & \rightarrow \text{OH}^- + \text{Na}^+ \\
& \downarrow \downarrow \\
\text{H}_2\text{O} & + \text{NaCl}
\end{align*}
\]

When an acid and a base are present in the same solution, they tend to neutralize each other, forming a neutral salt and water.

The strength of an acid is determined by its degree of dissociation, e.g., the concentration of its hydrogen ions in watery solution. Likewise, the strength of a base is dependent on the concentration of hydroxyl ions in the solution. Thus, a base came to be identified as a substance containing a hydroxyl group. In the chemical reactions occurring in the body this is not usually true, since most of these bases do not contain the OH\(^-\) radical.

Bronsted defines an acid as a hydrogen ion (proton) donor, and a base as a hydrogen ion (proton) acceptor.\(^3\)
A (acid) \[ \rightleftharpoons \] B (base) + H⁺ (proton)

Acid (Hydrogen ion or proton donor) \[ \rightarrow \]

B (base) (Hydrogen ion or proton acceptor)

\begin{align*}
H_2CO_3 & \rightarrow H^+ + HCO_3^- \\
H_3PO_4 & \rightarrow H^+ + H_2PO_4^- \\
HCl & \rightarrow H^+ + Cl^- \\
NH_4^+ & \rightarrow H^+ + NH_3
\end{align*}

This theory offers a better explanation of the action of chemical buffers in the body, and is the one most generally accepted today. Note, that in this instance the ammonium ion \( (NH_4^+) \) — which ordinarily would be considered basic — behaves like an acid, in that it can lend a hydrogen ion.

The concentration of hydrogen ions in body fluids is quite small. The symbol \( pH \) was introduced by Sorensen in 1909 as a convenient way to express minute concentrations of hydrogen ions, thus avoiding the use of decimals or fractions. \( pH \) is defined as the negative logarithm of the hydrogen ion concentration, or \( pH = -\log (H^+) \). Water dissociates into \( H^+ \) and \( OH^- \) ions, and the \( H^+ \) ion concentration of pure water has been calculated to be \( 10^{-7} \). The negative logarithm of this figure is 7. Thus, the \( pH \) of water is seven. Acids have a \( pH \) lower than 7, ranging from one to seven, the lower numbers denoting the stronger acids. Bases have a \( pH \) above 7, ranging up to 14. Similarly, the stronger bases are expressed by the higher numbers. Normal \( pH \) of blood ranges from 7.35 to 7.45, which means that blood normally is slightly alkaline. When we speak of acidosis or alkalosis in the body, we mean that the \( pH \) has deviated from this normal range. Thus, a \( pH \) of 7.2 is definitely acidotic, even though chemically speaking this figure would indicate a mildly basic substance. A \( pH \) below 6.8 or above 7.8 is incompatible with life, so one can note that the margin of safety is indeed narrow.

There are three principal means by which the body strives to maintain a normal \( pH \):

1. The buffer systems. These serve as the first line of defense against strong acids or bases formed in or entering the body.
2. The respiratory system. While we are well versed in the chemical control of respiration, we must also understand that the opposite is also true, e.g., that the lungs help to control the chemistry of the body.
3. The kidneys. Because of their ability to selectively retain or excrete acids and bases, they are one of the most important compensatory mechanisms. It must be remembered that it takes a few hours before the kidneys can influence the situation; so, they are of little value in very acute acid-base disorders.

BUFFER SYSTEMS OF THE BODY

Let us consider the buffer system in a little greater detail. A "buffer" is defined as a substance which tends
to maintain a solution at a relatively constant \( p\ell H \) when an acid or an alkali is added to it. There are four main buffer systems which help maintain a stable \( p\ell H \) in the body:

1. The bicarbonate—carbonic acid system operates primarily in the plasma. This is quantitatively the largest buffer system: (Fig. 1)

\[
\text{NaHCO}_3 + \text{HCl} \rightarrow \text{NaCl} + \text{H}_2\text{CO}_3
\]

\[
\text{H}_2\text{CO}_3 \rightarrow \text{H}_2\text{O} + \text{CO}_2
\]

\[
\text{NaOH} + \text{H}_2\text{CO}_3 \rightarrow \text{NaHCO}_3 + \text{H}_2\text{O}
\]

In these reactions, a strong acid entering the body is converted to a neutral salt and a weak acid. Carbonic acid is quite unstable, and the \( \text{CO}_2 \) formed by further dissociation is readily excreted via the lungs. A strong base is converted to water and a weak base (sodium bicarbonate) which itself is one of the buffers in the system.

2. The phosphate buffer system operates in the cells, including the red cells and the cells of the kidney tubules. (Fig. 2)

\[
\text{Na}_2\text{HPO}_4 + \text{HCl} \rightarrow \text{NaOH} + \text{NaH}_2\text{PO}_4
\]

\[
\text{NaOH} + \text{NaH}_2\text{PO}_4 \rightarrow \text{Na}_2\text{HPO}_4 + \text{H}_2\text{O}
\]

In these reactions, a strong acid is converted to a neutral salt (\( \text{NaCl} \)) and a strong base is converted to water (by a phosphate buffer salt). The buffers themselves change from a mildly basic substance to a mildly acid one, and vice versa.

3. The protein buffer system predominates in the tissue cells. Proteins act as anions, accepting either hydrogen or basic cations such as sodium or potassium:

\[
\frac{\text{H}^+ \text{ protein}}{\text{B}^+ \text{ proteinate}}
\]

4. The hemoglobin buffer system. Hemoglobin has the ability to accept hydrogen or basic cations. Reduced hemoglobin is less acid than oxyhemoglobin and has greater ability to accept hydrogen ions. The hemoglobin is oxygenated when it reaches the lungs to become \( \text{HHbO}_2 \), which then reacts with bicarbonate salts to form carbonic acid.\(^4\) (Fig. 3)

As you may recall, this is one of the reactions taking place in the chloride shift.

It has already been mentioned that the bicarbonate-carbonic acid buffer system is quantitatively the largest of the four. This is also the most often measured, since fundamental
laboratory data are most readily obtained in this system. The Henderson-Hasselbalch equation best exemplifies how this buffer system functions to maintain a normal body pH:

\[ \text{pH} = \text{pK}_1 + \log \frac{\text{HCO}_3^-}{\text{H}_2\text{CO}_3} \]

In the equation, \( \text{pK}_1 \) represents a dissociation constant for carbonic acid. Carbonic acid ionizes according to the equation:

\[ \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^- \]

The product of the concentrations of the substances on the right divided by the concentration of the substance on the left is equal to a constant:

\[ \frac{(\text{H}^+) \times (\text{HCO}_3^-)}{\text{H}_2\text{CO}_3} = K \]

In the case of carbonic acid, at a pH of 6.1, the concentrations of bicarbonate and carbonic acid in the body are equal. Thus, \( \text{pK} \) has the value of 6.1.

\( \text{H}_2\text{CO}_3 \) represents the CO\(_2\) physically dissolved in the plasma plus hydrated CO\(_2\) or carbonic acid. Actually, about 99 percent of the carbon dioxide is physically dissolved. Since the normal value of bicarbonate in the plasma is 27 mEq/L and the concentration of CO\(_2\) + H\(_2\)CO\(_3\) is 1.35 mEq/L our equation becomes:

\[ \text{pH} = \text{pK}_1 + \log \frac{\text{HCO}_3^-}{\text{H}_2\text{CO}_3} \]

\[ \text{pH} = 6.1 + \log \frac{27}{1.35} \]

\[ \text{pH} = 6.1 + \log 20 \]

\[ \text{pH} = 6.1 + 1.3 \]

\[ \text{pH} = 7.4 \]

Since \( \text{pK}_1 \) remains constant, so long as the ratio of \( \text{HCO}_3^-/\text{H}_2\text{CO}_3 \) remains at 20/1, pH will remain normal.

Consider how this works in a hypothetical case. A lazy anesthetist gives a cyclopropane anesthetic to a patient for a lengthy procedure without assisting respiration. Carbon dioxide tension becomes elevated, and in time doubles its normal value, increasing to 2.7 milliequivalents per liter. As this occurs, bases in the form of sodium and potassium cations are released from the hemoglobin and plasma protein to form more bicarbonate, so that its value is also doubled. Thus our equation would change to:

\[ \text{pH} = \text{pK}_1 + \log \frac{\text{HCO}_3^-}{\text{H}_2\text{CO}_3} \]

\[ \text{pH} = 6.1 + \log \frac{54}{2.7} \]

\[ \text{pH} = 6.1 + \log 20 \]

\[ \text{pH} = 7.4 \]

This theoretical patient is in a state of respiratory acidosis, but since the pH has not changed, he is said to be compensated. His alkali reserve has been depleted however, and further insults will eventually cause the pH to be decreased toward the acid side.

**TYPES OF ACID-BASE DISORDERS**

In the language of the laboratory, “acidosis” and “alkalosis” would be defined strictly in terms of deviation of blood pH. These nouns would then become synonymous with low and high blood pH, respectively. In physiological language, the term “acidosis” is an abnormal condition that tends to produce a downward devi-
ation of blood pH, although this change may be prevented or reduced by secondary or compensatory mechanisms. We have already seen an example of respiratory acidosis with an unchanged pH.

"Alkalosis" would similarly be defined as an abnormal state that tends to elevate blood pH from its normal. Through common usage it would appear that the adjectives "respiratory" and "metabolic" will continue to be applied to denote the primary cause of the acid-base disturbance. Thus, we continue to speak of respiratory and metabolic acidosis, or respiratory and metabolic alkalosis. The term "compensation" is defined as the restoration of the pH to normal in the presence of the initial acid-base disturbance. Compensation in an acid-base problem may be complete, partial, or absent, depending on the extent to which the blood pH is deviated from the normal, and whether or not compensatory effects are evident.

It is essential that at least three parameters be known before one can assess the acid-base status of an individual with reasonable accuracy; that is,

1. The pH
2. The carbon dioxide tension
3. The bicarbonate level

Most laboratories will measure two of these, for example, pH and bicarbonate, and by means of the Henderson-Hasselbalch equation, calculate the third. Undoubtedly, in many instances a complete electrolyte panel will add a great deal of information. The following table depicts the changes which are most often seen in primary acid-base disorders with partial compensation. (Table 1)

Respiratory acidosis is due to an elevated level of carbon dioxide in the blood. Laboratory data would usually show a lowered pH and an elevated bicarbonate along with the elevated pCO2. The condition is almost always due to hypoventilation, although in some instances (e.g., during anesthesia) it can be produced by breathing an atmosphere with an increased CO2 content. Respiratory acidosis may be chronic, as occurs in emphysema or obstructive lung disease. It may also be acute, as is sometimes seen during surgery or the period following it. Since chronic respiratory acidosis tends to develop slowly, the renal buffer system of the body has more time to participate in compensating for the elevated carbon dioxide tension. With moderate increases of pCO2, the pH is often in the normal range. With high levels, above 80 mm Hg, some lowering of pH is uniformly present.

A respiratory acidosis is usually compensated by the development of a metabolic alkalosis. As this occurs,

<table>
<thead>
<tr>
<th>pH</th>
<th>pCO2</th>
<th>Bicarbonate</th>
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<tbody>
<tr>
<td>↓</td>
<td>↑</td>
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<td>↑</td>
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</tbody>
</table>

TABLE 1
the pH tends to return toward normal, bicarbonate becomes more elevated, and the pCO\textsubscript{2}, of course, remains unchanged. One must keep in mind that, when the respiratory acidosis has an acute onset, pH changes may occur within a few minutes, and coma may ensue when the pCO\textsubscript{2} reaches 70 or 80 mm Hg.

Respiratory alkalosis is defined as a primary reduction in the partial pressure of carbon dioxide in the blood. Accompanying the low pCO\textsubscript{2} is an elevation in pH, and a decrease in bicarbonate. This disorder is due to hyperventilation, causing excessive amounts of CO\textsubscript{2} to be blown off. It may be seen in certain disease states such as hypoxia, anemia, bacteremia, and heart failure, as well as during anesthesia with assisted or controlled respiration. Respiratory alkalosis is compensated by a fall in bicarbonate, thus causing a lowering of pH to more normal values. It is of interest to note that all the clinical conditions which bring about respiratory alkalosis are also associated with elevation of blood lactate, or lactate and pyruvate. These fixed acids produce a metabolic acidosis of severe degree. If in time it becomes evident that lactic acidosis is an end stage of respiratory alkalosis, we may again have to revise our concepts of ventilatory control during anesthesia.

Metabolic acidosis may be defined as an excess of hydrogen ions in the blood which result from an accumulation of acids other than carbonic. It can occur from base loss as well as acid gain in the body. As one would expect, the pH and bicarbonate level are both decreased. Increased hydrogen ion concentration stimulates the respiratory center and the chemoreceptors, resulting in hyperventilation and lowered pCO\textsubscript{2}, e.g., Kuss-Maul respiration in diabetes. Thus, a respiratory alkalosis tends to compensate for a metabolic acidosis. The compensation is never complete, however, for as CO\textsubscript{2} continues to be blown off, a point will be reached where the lowered carbon dioxide tension will depress ventilation. It remains for the kidneys to correct the disturbance permanently. Metabolic acidosis is the most common of the serious acid-base derangements. It is seen in a wide variety of conditions, among which are diabetes, uremia, diarrhea, high fever, shock, and anesthesia.

Metabolic alkalosis results from an excess of base or a deficit of hydrogen ions in the body. Laboratory data usually show an elevated pH and bicarbonate, with a normal or slightly elevated pCO\textsubscript{2}. Theoretically, hypoventilation with its concomitant increase in carbon dioxide tension would be the logical means of compensating for a metabolic alkalosis. In this situation, however, hypoventilation would eventually cause a lowered oxygen saturation, which in turn would tend to stimulate respiration. Metabolic alkalosis is seen most often in patients having hydrogen ion loss from vomiting or gastric suction, and in individuals given excessive doses of sodium bicarbonate or other alkaline salts. Unless present preoperatively, it is seldom a problem during anesthesia.

It must be understood that acid-base disturbances are not always as simple as the foregoing descriptions would seem to indicate. There are frequently abrupt swings in acid-base chemistry, or mixed patterns, such as respiratory acidosis superimposed on a metabolic acidosis, which add
to the confusion. Perhaps the following case reports will serve as an example of more complicated problems:

CASE NO. 1

Mrs. B. L., a healthy 32-year-old white female, para 1, gravida 2, was admitted to the hospital for delivery. Since she gave a history of having recently ingested food, soda water was given to induce vomiting which, according to the nurse’s notes, proved quite successful. The patient was medicated with demerol, scopolamine, and intravenous pentobarbital (Nembutal). Two hours after admission, she was taken to the delivery room, and anesthesia was induced with nitrous oxide-oxygen. Five minutes later, the patient suddenly vomited copious amounts of semi-solid material. Intravenous succinylcholine was given and a cuffed endotracheal tube inserted. It was obvious that aspiration had occurred. The trachea was repeatedly suctioned and lavaged with normal saline. The patient was delivered, and since she continued to have bronchospasm and bilateral chest rales, suction bronchoscopy with further lavage was performed.

She was taken to the intensive care unit, placed in a high humidity oxygen tent, and intermittent positive pressure therapy was given for 10 minutes every hour. Her condition deteriorated during the next eight hours, and she appeared slightly cyanotic in spite of oxygen administration. A tracheotomy was performed and oxygen was continued through a tracheostomy adapter. One hour later, blood was drawn for an acid-base study and oxygen saturation. This study was repeated in 12 hours and again in 24 hours. Table 2 shows the results of these studies as compared with the normal values for our laboratory.

<table>
<thead>
<tr>
<th>Sample 1</th>
<th>pH</th>
<th>pCO₂ mm Hg</th>
<th>HCO₃⁻ mEq/L</th>
<th>O₂ Sat.%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>7.36-7.42</td>
<td>33-44</td>
<td>23-33</td>
<td>94-99</td>
</tr>
<tr>
<td>Sample 1</td>
<td>6.33</td>
<td>26.5</td>
<td>16.8</td>
<td>94</td>
</tr>
<tr>
<td>Sample 2</td>
<td>7.16</td>
<td>85</td>
<td>31.9</td>
<td>86</td>
</tr>
<tr>
<td>Sample 3</td>
<td>7.47</td>
<td>36</td>
<td>26.4</td>
<td>98.5</td>
</tr>
</tbody>
</table>

Comparing the results of this table with the expected changes shown in Table 1 gives us the following analysis:

Sample 1: The levels of pH, pCO₂, and HCO₃⁻ are all below normal, indicating metabolic acidosis. There is almost complete compensation by virtue of hyperventilation and blowing off of carbon dioxide from the lungs. The metabolic acidosis is undoubtedly due to tissue hypoxia, which causes an accumulation of acid waste products due to incomplete metabolism.

Sample 2: The picture has changed from a metabolic acidosis to a severe respiratory acidosis. Compensation is minimal, since the bicarbonate level has not risen significantly. The patient at this time was comatose. Since there was obviously an interference with carbon dioxide elimination, the patient was placed on a ventilator with 40 percent oxygen, and respiration was assisted.

Sample 3: The ventilator has done its work well. There exists a very mild respiratory alkalosis, and oxygen saturation has returned to normal. The patient at this time was awake and asking for food. Fortunately, she continued to improve and was discharged one week later.

CASE NO. 2

Mr. J. L. W., a 31-year-old white male, was admitted to the hospital for an elective gastric resection because of chronic intractable duodenal ulcer. Except for his present illness, he was quite normal and healthy. Laboratory work was all within normal limits. The patient was premedicated with 75 mgm of demerol and 0.4 mgm of atropine administered one hour prior to surgery. He was induced with a slow dose of thiopental and a 0.1 percent succinylcholine drip was started. As apnea ensued, the patient was hyperventilated with oxygen, and endotracheal intubation was performed. Maintenance was begun with a five-liter flow of nitrous oxide-oxygen in equal amounts plus 0.5 percent halothane, and a slow drip of succinylcholine.
December, 1966

the patient was carried on nitrous oxide-oxygen, supplemented by small (10-20 mgm) intermittent doses of intravenous demerol. Two liters of Ringer's lactate had been given in the interim, and the blood pressure temporarily rose to 100/70 with a pulse rate of 100. Thirty minutes later the pressure again dropped to 60/40 and the pulse rose to 120. Several doses of methoxamine intravenously had no effect. Five hundred milligrams of hydrocortisone followed by 25 mgm of ephedrine failed to elevate the blood pressure. At this point it was noted that the patient appeared flushed and felt warm, but was not perspiring. Nasopharyngeal temperature was taken and recorded at 103°F.

One hour had passed since surgery had commenced, and the surgeon had already divided the stomach and was closing the duodenal stump. There was no alternative except to continue the operative procedure. The patient was placed on a hypothermic blanket, ice water enemas were begun, and the peritoneal cavity was lavaged with literally gallons of cold normal saline. Three liters of Ringer's lactate were rapidly infused. In spite of these measures, the temperature continued to rise to 108°F. The patient was hyperventilated with 100 percent oxygen. An ECG oscilloscope had been hooked up which showed a sinus tachycardia of 140, and high peaked T waves, indicative of hyperpotassemia. One gram of calcium chloride was given, and 50 mgm of chlorpromazine (Thorazine) were administered intravenously in an attempt to make the patient pellagroid. Two ampules (88 milliequivalents) of sodium bicarbonate were also administered intravenously, and five minutes later blood was drawn for an acid-base and electrolyte study. Blood pressure at this time was still 60/40.

The cooling measures were continued, and following the administration of the Thorazine, the temperature fell quite precipitously to 103°F. Cardiac arrest occurred, and closed chest massage was instituted immediately. Epinephrine (0.25 mgm) injected into the left ventricle was effective in restoring a pulse. One gram of calcium chloride and 176 milliequivalents of sodium bicarbonate were given intravenously. The operative procedure had now been completed, and the patient was moving, swallowing, and hyperventilating. Since we knew from the ECG tracing that the patient was severely hyperkalemic, peritoneal dialysis was instituted in an effort to reduce the potassium level. Another blood sample was drawn for an acid-base study. Shortly thereafter, cardiac arrest recurred, and all resuscitative efforts were in vain. The electrolyte and acid-base panels are shown in Table 3.

Analysis of these studies indicates:

Sample 1: A severe acidosis exists. The high carbon dioxide tension is evidence of a marked respiratory acidosis, and the lowered bicarbonate signifies a metabolic component.

Sample 2: The acidotic state has progressed in spite of the administration of six ampules (264 mEq) of sodium bicarbonate, and hyperventilation with high flows of oxygen.

An electrolyte panel done at the same time as the first acid-base study showed the "rest acids" to be 22 mEq/L compared to our normal of 5.6-10.4. This refractory type of metabolic acidosis is typical of lactacidemia due to excessive production of lactate secondary to anaerobic metabolism. Certainly, an individual with such an elevated metabolic rate in the presence of hypotension cannot possibly have adequate tissue oxygenation. Potassium determination revealed an elevation to 8.9 mEq/L which in itself is sufficient reason for cardiac arrest.

The Department of Anesthesia at the University of California recently reported two cases of hyperthermia during anesthesia.9 The laboratory data presented in their first case were very similar to ours. They, too, found that the acidotic patient fails to respond to the action of vasopressors and adrenocortical steroids. Their

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<td>92</td>
</tr>
<tr>
<td>Sample 2</td>
<td>6.74</td>
<td>105</td>
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</table>

TABLE 3
conclusion is clear: "Hyperthermia during anesthesia with its associated acidosis is a life-threatening condition that requires both rapid and vigorous therapy if the patient is to survive."

DISCUSSION

According to most authorities, changes in the carbon dioxide or hydrogen ion concentration acting on the normally sensitive respiratory center essentially govern the chemical control of respiration. In the healthy, awake individual, a rise of as little as 0.2 volumes percent over the normal 5.5 volumes percent alveolar carbon dioxide concentration will double respiratory minute volume, while a similar reduction produces apnea. The respiratory center is depressed by oxygen lack, narcotics, and all anesthetic agents. The carotid and aortic body chemoreceptors are relatively insensitive, being stimulated by rather marked reductions in arterial oxygen tension or elevations in hydrogen ion concentration.

It is little wonder that respiratory derangements producing acid-base disorders are common in anesthetized patients. We give narcotics and/or barbiturates preoperatively, then administer a potent anesthetic agent along with a high oxygen concentration. This has the effect of depressing both the respiratory center and the chemoreceptors. There is usually some depression of renal function, and the patient must depend entirely on the anesthetist to maintain respiratory equilibrium.

Acute respiratory acidosis has long been recognized as one of the major acid-base disorders which may occur during anesthesia. Although it is generally agreed that impaired alveolar ventilation is responsible for CO₂ retention and the ensuing respiratory acidosis, there are many factors which may produce this situation. While some of these are beyond our control, e.g., diffuse pulmonary disease, in most instances we are able to prevent respiratory acidosis during surgery by assisted or controlled respiration. Hypercapnia renders the patient more vulnerable to the effects of hemorrhage, decreases the normal vascular response to sympathomimetic agents, increases the susceptibility of the heart to vagal stimulation, and causes elevation of serum potassium levels. The last two factors predispose to ventricular irritability and cardiac arrest. Further, sudden correction of a respiratory acidosis at the conclusion of surgery may be associated with hypotension and cardiovascular collapse. This is the etiology of so-called "cyclopropane shock."

Hypoventilation continues to be a problem in the early postoperative period. The patient's respiratory center often remains depressed and respiratory muscles may be partially paralyzed from relaxant drugs. Splinting of the diaphragm or chest wall from pain, abdominal distension, tight binders, and pre-existing pulmonary disease all tend to increase respiratory acidosis.

Since decreased alveolar ventilation is the cause of respiratory acidosis, the obvious corrective measure is to increase inspiratory depth. If for some reason, this cannot be accomplished, effective alveolar ventilation can be relatively increased by reducing anatomic dead space by means of tracheotomy or endotracheal intubation. Since some of the patients with chronic lung disease depend on
“hypoxic drive” from the chemoreceptors to stimulate respiration, the administration of oxygen without mechanical assistance may result in a decrease in minute volume and further rise in pCO₂. Sodium bicarbonate should be given with caution since this, too, may precipitate a further rise in carbon dioxide tension as the body strives to maintain bicarbonate-carbonic acid equilibrium.

So much has been said in the past about the dangers of hypoventilation during anesthesia, we now tend to overcompensate and carry our patients in some degree of respiratory alkalosis. While this practice does not appear to produce ill effects clinically, hyperventilation does produce fall in blood pressure and reduced cerebral blood flow due to marked cerebral vasoconstriction. We should also remember that sustained hypocapnia has been shown to lead to metabolic acidosis. Although bicarbonate drops along with the fall in pCO₂, the true bicarbonate deficit is thought to be due to the rise in pyruvic and lactic acids. These and other acid waste products accumulate rapidly during tissue hypoxia from any cause as a result of anaerobic metabolism. Accordingly, when there is poor tissue perfusion from shock, cardiac failure, hypothermia, or cardiopulmonary bypass, metabolic acidosis can be expected. These acid metabolites first build up locally, and as tissue perfusion later improves, the circulation is flooded by excess hydrogen ions.

At various times in the past, ether, cyclopropane, multiple blood transfusions and various anesthetic techniques were thought to precipitate metabolic acidosis during surgery. According to more recent work, this can no longer be accepted. All agree that metabolic acidosis occurs with any agent or technique in the presence of shock.

The first step in the treatment of metabolic acidosis is the removal of the precipitating factor. Many times this is all that is necessary. The body does the rest. More severe cases can be aided by the administration of sodium bicarbonate or tromethamine (THAM). Tham acts as a hydrogen ion acceptor, and after combining with the proton, is excreted via the kidneys. Whichever drug is used, one must be careful not to correct the disturbance too quickly.

Whether the acidotic state is due to respiratory or metabolic components, it is usually associated with an elevated serum potassium level. As hydrogen ion concentration increases in the extracellular space, potassium moves out of the cell as a compensatory mechanism. Conversely, if the acidosis is suddenly corrected, potassium moves from the plasma back into the cells, and hypokalemia may occur. This emphasizes the importance of a complete electrolyte panel when dealing with a severe acid-base disturbance.

SUMMARY

I have attempted to present some of the more basic concepts of acid-base balance, particularly as they apply to the anesthetized patient. Acidosis in both its forms gives us more problems than alkalotic states, although we must be aware that marked respiratory alkalosis may present its own hazard. There is no doubt that we should take advantage of the tremendous growth of knowl-
edge in this field, and obtain frequent electrolyte and acid-base panels on problem cases. As with any other patho-physiologic disturbance, correction of existing disorders should be made before surgery.

BIBLIOGRAPHY

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12 op. cit., Eichenholz, p. 702.