Electrolytes, their physiological action and interaction: A review

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Knowledge concerning the physiological action and interaction of electrolytes has greatly increased during the last few years. This article provides a review of the management of electrolyte disturbances, encompassing first the normal values of electrolytes, then their physiological actions and interactions, as well as the effects of their abnormal values.

In the 1980 edition of the Council on Accreditation of Nurse Anesthesia Educational Programs/Schools’ Standards and Guidelines for Accreditation of Nurse Anesthesia Educational Programs/Schools, the 7th “minimum competency required of new graduates” is the management of fluid therapy within the medical plan of care, to include management of electrolyte and metabolic disturbances.

If nurse anesthetists are to be responsible for managing electrolyte disturbances, they need to know what electrolytes are, including their normal values, their physiological actions and interactions and the effects of abnormal values. This article will review electrolytes and their management.

Electrolytes defined

Electrolytes are “charged particles” that carry electrical currents, hence their name. In most scientific disciplines, the term ion is used to indicate an electrolyte. An anion is a negatively charged ion, and a cation is a positively charged ion.

When evaluating the electrolyte content of our patients, we should keep in mind three characteristics:

1. Electrolytes tend to be balanced. That is to say, an equal number of negative ions and positive ions are usually found on each side of a membrane. This condition does not always exist, but physical nature encourages this equilibrium of charges.

2. Unlike charges attract. Just as opposite poles of a magnet attract, positive charges attract negative charges and vice versa. It requires energy to keep unlike charges separated.

3. Like charges repel. Energy is needed to keep like charges together. An anion repels an anion, a cation repels a cation.

Keeping these three characteristics in mind, it becomes obvious that we cannot consider any one electrolyte alone, since the presence of an electrolyte affects surrounding electrolytes.

Extracellular amounts of electrolytes are measured clinically because there is a constant interchange of ions between the intracellular fluid (ICF) and extracellular fluid (ECF). The normal values of the electrolytes to be considered in this article are summarized in Table I.

Hydrogen ion

The hydrogen ion (H+) is often considered only when trying to determine the acid base balance condition. However, because of the three electrolyte characteristics mentioned above, it must be considered along with other ions. The negative logarithm of the normal (H+) hydrogen ion content in equivalents per liter is 7.4. A real number
measurement that is now coming into clinical use is 40 nanoequivalents (or $10^9$ equivalent) per liter of ECF.

Because of the relationship of the H$^+$ content, bicarbonate ion (HCO$_3^-$) content and carbonic acid (H$_2$CO$_3$) content as depicted by the Henderson-Hasselbalch equation ($\text{pH} = 6.1 + \log \text{HCO}_3^-$), we cannot consider the H$^+$ content without considering the HCO$_3^-$ content and the amount of H$_2$CO$_3$.

An equal value of H$_2$CO$_3$ is PCO$_2 \times 0.3$. The values needed to keep the ratio of HCO$_3^-$ to H$_2$CO$_3$ (the metabolic component of the acid base balance) 20 times as great as the ratio of carbonic acid, (the respiratory component of the acid base balance) make up the “normal” value of HCO$_3^-$ (Thus, its normal value was given in quotation marks in Table I). With a 20:1 ratio of HCO$_3^-$ to H$_2$CO$_3$, the pH is 7.4 and the H$^+$ content is 40 nEq/L ECF. The HCO$_3^-$ value is 24 mEq/L if the PCO$_2$ is 40 mmHg and the pH is 7.4.

A pH less than 7.4 is acidotic, indicating a greater than normal number of H$^+$. A pH greater than 7.4 is alkalotic, indicating a less than normal number of H$^+$. When there is a greater than normal amount of hydrogen ions, many physiological changes occur that attempt to keep a 20:1 ratio between HCO$_3^-$ and H$_2$CO$_3$. Another result is that the hemoglobin will release oxygen molecules more readily. This is a compensatory mechanism since an increase in hydrogen ions is usually a result of increased carbon dioxide (CO$_2$).

Taking this equation: H$_2$O and CO$_2 \rightleftharpoons$ H$^+$ + HCO$_3^-$, an increased amount of CO$_2$ usually reflects an increased amount of metabolism and usage of oxygen. An exercising muscle has a local increase in CO$_2$ and H$^+$. Therefore, an increased release of oxygen where there is an increased H$^+$ content is of great value in replacing the already utilized O$_2$.

A decrease in H$^+$ can be a response to hyperventilation. It is very hard for some to comprehend that you can, in essence, “have too much of a good thing,” ventilation. As the amount of CO$_2$ decreases, the amount of H$^+$ also decreases (see equation above). When the amounts of H$^+$ and CO$_2$ decrease, vascular changes occur. For example, the cerebral arteries constrict. This can be used to advantage in neurosurgery, but it should be remembered that cerebral hypoxia may result from cerebral vaso-constriction. A PCO$_2$ of less than 25 mmHg is considered to be in the danger zone.

A decrease of H$^+$ because of a decreased PCO$_2$ in the extracellular fluid can also cause temporary hypokalemia in the ECF. In an effort to maintain the H$^+$ at its important normal value, the potassium ion (K$^+$) will move intracellularly, as H$^+$ moves out of the cell into the ECF. It is important to remember the propensity of electrolytes to having “equal charges on each side of the membrane.”

### Table I: Electrolytes and their normal values

<table>
<thead>
<tr>
<th>Name</th>
<th>Symbol</th>
<th>Normal value in extracellular fluid</th>
<th>Normal value in intracellular fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydrogen ion</td>
<td>H$^+$</td>
<td>pH 7.4; 40 nEq/L*</td>
<td>pH 7.0; 100 nEq/L*</td>
</tr>
<tr>
<td>Bicarbonate ion</td>
<td>HCO$_3^-$</td>
<td>“24” mEq/L</td>
<td>“12” mEq/L</td>
</tr>
<tr>
<td>Potassium ion</td>
<td>K$^+$</td>
<td>4.5 mEq/L</td>
<td>141 mEq/L</td>
</tr>
<tr>
<td>Sodium ion</td>
<td>Na$^+$</td>
<td>142 mEq/L</td>
<td>10 mEq/L</td>
</tr>
<tr>
<td>Chloride ion</td>
<td>Cl$^-$</td>
<td>103 mEq/L</td>
<td>4 mEq/L</td>
</tr>
<tr>
<td>Calcium ion</td>
<td>Ca$^{++}$</td>
<td>4.5 mEq/L 9 mg/dL</td>
<td>1 mEq/L</td>
</tr>
<tr>
<td>Magnesium ion</td>
<td>Mg$^{++}$</td>
<td>2.3 mEq/L</td>
<td>60 mEq/L</td>
</tr>
</tbody>
</table>

*Nanoequivalents per liter
K+ content intraoperatively simply because it existed preoperatively. Cardiac arrhythmias may result from hyperventilation even though the patient had a normal K+ content preoperatively. The K+ content in the ECF will decrease due to respiratory alkalosis and can become apparent in several hours.

Changes in the K+ content of the ECF can result from the treatment of the diabetic patient. When 1 gram of glucose is changed to glycogen, 3.6 mEq/L of K+ are retained. If a diabetic is given insulin for a high blood sugar level, an appreciable amount of K+ may be removed from the circulating fluid as the “sugar” is changed to glycogen and stored. Therefore, whenever a diabetic is given insulin, his heart should be monitored. It should be kept in mind that a patient on digitalis is also extremely susceptible to cardiac arrhythmias from hypokalemia. Many diabetics are also on digitalis.

Nurse anesthetists are all aware of the substitution of K+ for H+ that occurs in urine when the patient is alkalotic. We know that certain diuretics cause depletion of K+. Acetazolamide and chlorothiazide are particularly known for causing K+ depletion. No elective surgery should be started without knowledge of the ECF K+ for a diabetic or a patient on diuretics.

Hyperkalemia, excess K+ in the ECF, can also have ill effects. Chronic hyperkalemia is for the most part confined to patients with renal failure or adrenal insufficiency (Addison’s disease). It can also occur because of too rapid and/or too great a replacement of K+ in treatment of hypokalemia.

The administration of succinylcholine causes a temporary increase in K+ in ECF, probably due to muscular fibrillation and contractions. In lower motor lesions caused by burns or trauma, or upper motor lesions due to strokes, tumor or spinal cord injury, the potassium release from succinylcholine may be sufficient to cause hyperkalemia five minutes after intravenous administration. This K+ is absorbed intracellularly in 10 minutes. Cardiac problems, however, may be precipitated before that time. Hyperkalemia causes elevated T waves, widening of the QRS complex with a flattening of the P wave (see Figure 2).

The increase in K+ released after a succinylcholine injection does not occur immediately post-upper or -lower motor injuries. One week post-injury seems to be the minimum time for this increase, and 9 weeks seems to be the maximum depending on the type of injury.

Sodium ion

The normal values of the sodium ion (Na+) are 142 mEq/L ECF and 10 mEq/L ICF. It should be pointed out that Na+ is seldom at abnormal values. The conscientious person will make every effort to consume sufficient amounts of salt (NaCl) necessary to maintain normal values of Na+ and
Cl-. The renal system responds to hormonal demands for conservation of both.

When actively reabsorbed into the ECF from the renal tubules, Na+ is followed by Cl- because of the second characteristic of electrolytes which is that unlike charges attract. Both ions increase the osmolarity so water molecules are also reabsorbed into the ECF. Thus, Na+ plays an important role in contributing to the osmolarity of the ECF and consequently, its volume.

Another example of why we cannot consider electrolytes separately is the effect of aldosterone, excreted by the adrenal glands in response to increased K+ and/or decreased Na+. The secretion of this hormone causes the excretion of K+, Ca++, and Mg++ while causing the reabsorption of Na+ and Cl-.

Chloride ion

The chloride ion (Cl-) has a normal value of 107 mEq/L ECF and 10 mEq/L ICF. Besides contributing to the osmotic pressure of the ECF, it also functions to maintain the "equal charge" on both sides of the membrane. In the red blood cell, in the presence of the enzyme carbonic anhydrase, the equilibrium $\text{H}_2\text{CO}_3 \rightleftharpoons \text{H}_2\text{CO}_3 + \text{H}^+ + \text{HCO}_3^-$ occurs very rapidly.

The vast majority of the H+ is gathered up by the hemoglobin in the red blood cells and thus does not contribute to the H+ content in the ECF. The vast majority of the HCO$_3^-$ moves into the ECF by simple diffusion. The Cl$^-$ diffuses into the red blood cell from the ECF to maintain the charge balance in what is called the "chloride shift."1

Calcium ion

The normal values of the calcium ion (Ca++) are 4.5 mEq/L ECF and 1 mEq/L ICF. Ca++ plays an important role in coagulation. It also plays an important part in all muscle contractility. It is believed that four calcium ions are utilized per vesicle of acetylcholine released at the neuromuscular junction. In addition, Ca++ must be present for the interdigitation of muscular molecules that results in muscle contraction.7

The calcium ion is of greater importance to cardiac excitability because of the heart's smooth muscle characteristics. It is now believed that the action potential, the depolarization of cardiac neurotransmission, may be initiated by the influx of Ca++ as well as Na+, or by Ca++ alone.1 We know that a decrease in Ca++ in ECF, hypocalcemia, will cause a prolonged QT interval on the ECG, reflecting a decreased contractility (see Figure 3). Hypercalcemia, an increase in Ca++ in ECF, will cause a shortened QT interval reflecting an increased contractility, which may progress to spastic contractions (see Figure 4).4,8

These muscular contractions are affected by Ca++ on specific receptor sites. It is believed now that the other cations (K+, Na+, and Mg++) compete with Ca++ for these receptor sites, just as the curare or pancuronium we administer competes with acetylcholine for acetylcholine receptor sites.
At normal ion contents in the ECF, the "right" proportion of the receptor sites are occupied by Ca++ and the "right" proportion of the receptor sites are activated to cause contraction. The other cations Na+, K+ and Mg++ occupy Ca++ receptor sites but cause no effect.

If the proportions are changed, the proportion of receptor sites occupied by Ca++ will change. Remember, the other ions do not cause a contraction, just as an acetylcholine receptor site occupied by pancuronium or curare does not cause a contraction.¹

Hyperkalemia causes weakness of contractility. A greater proportion of Ca++ receptor sites may be occupied by K+. The resultant ECG reading has a wide flat P wave, a wide QRS, and a peaked T wave (see Figure 2). One treatment for hyperkalemia is the administration of a Ca++ dosage to increase the proportion of Ca++ in the ECF. Hypernatremia (a greater than normal Na+ content) causes the same effect, a flaccid heart muscle.

**Magnesium ion**

The normal magnesium ion (Mg++) content is 2-3 mEq/L ECF and 60 mEq/L ICF. Magnesium sulfate is given in toxic pregnancies because of the fear of too much muscle contractility. The effect of an increased proportion of Mg++ competing with the Ca++ is a decrease in muscular contractility believed to be caused by a greater proportion of Mg++ occupying Ca++ receptor sites. This decrease in muscular contractility in smooth muscles explains the vasodilatation resulting from excess Mg++ and K+.

Mg++ is also necessary for the parathyroid hormone secretion and the calcitonin secretion needed to change the calcium present into its active Ca++ form.¹ Only in severe malnutrition is there
a deficiency of Mg++. In man, this deficiency can be manifested by hyperexcitability with tremors and convulsions. In chronic alcoholics, or with patients on hyperalimentation where Mg++ has not been added, there can be a deficit. Some authorities believe delirium tremens are caused or contributed to by a Mg++ ion deficit.2

Since the ECF has such a small amount of Mg++ compared to the intracellular content, the latter might be deficient with normal or near normal values in the ECF. It takes five days to replace a deficiency of Mg++ in the intracellular fluid, so patience is required. Mg++ is a very important co-factor or co-enzyme of vital intracellular activities.

Summary
The following points are important to bear in mind:

- Hyperventilation causes a decrease in H+ which will cause a decrease in K+ in ECF. The reason for this is that the K+ is substituted when a positive ion is needed intracellularly.
- An increase in H+ causes a greater number of oxygen molecules to be released from hemoglobin to the tissue.
- A decrease in K+ can result from the treatment of a diabetic with insulin, since 3.6 mEq/L of K+ are withdrawn from the circulating ECF fluid with every gram of glucose changed to glycogen.
- A patient on digitalis is very susceptible to a decrease in K+ with resulting cardiac arrhythmias.
- The reabsorption of Na+ and the excretion of K+ and Mg++ are simultaneous results of aldosterone secretion.
- Cl- follows Na+ as a rule because unlike charges attract. Cl- also substitutes for HCO₃⁻ in the "chloride shift."
- Na+ and K+ move in opposite directions through a neural membrane to initiate an action potential.
- A decrease in K+ will interfere with neuromuscular transmission to the point of paralysis, if severe enough. The T wave becomes flat or inverted. The U wave appears and becomes more prominent as the T wave is lowered.
- Ca++ may substitute for Na+ in cardiac and smooth muscle in initiating an action potential. Ca++ is vital for skeletal muscle contractility and for cardiac muscle contractility and conductivity.
- Na+, K+, and Mg++ all compete with Ca++ for its muscle receptor sites, and once they are actually on the receptor sites have no effect, that is, no contraction results.

- Mg++ deficiency is to be considered when it seems impossible to maintain normal Ca++ content, and in prolonged malnutrition. Near normal ECF values of Mg++ may occur while there is a large intracellular deficiency.
- We usually only measure ECF and there is a constant movement of electrolytes between ECF and ICF.

As nurse anesthetists, we must continually update and review our knowledge of electrolytes— their normal values, their physiological actions and interactions and the effects of their abnormal values. This article afforded a brief review of this subject. For a more in-depth understanding of electrolytes, further reading is encouraged.

REFERENCES

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