Potassium - Some Practical Aspects of the Physiology of its Electrolyte Balance

Frances E. Noe
POTASSIUM — SOME PRACTICAL ASPECTS OF THE PHYSIOLOGY OF ITS ELECTROLYTE BALANCE

FRANCES E. NOE, M.D.*

There is a general tendency in the field of clinical medicine to reduce electrolyte replacement therapy to a few simple principles, the application of which is usually based on a minimum of routine laboratory procedures. Such practice is a matter of practical necessity, not of convenience or neglect, and one could not expect the majority of clinicians to be familiar with the maze of academic and often hypothetical material published in this field. However, there are noteworthy clinical applications which should be culled from the literature and added to one's stock of such knowledge from time to time.

A new entity, and by no means a homogeneous one, must be added to our concepts of electrolyte balance — the intracellular space. This has made it more important than ever to treat the patient, not the flame photometer results. We are more and more into an era which makes it possible by the application of a few fundamental principles to prevent the electrolyte imbalance and not have to treat it, as the pathophysiology of electrolyte disturbance becomes an essential part of general medical knowledge.

The phenomenon of potassium transfer to extracellular fluid with concurrent increased intracellular sodium content and relative acidity is by now familiar to most clinicians but what are the clinical implications of such an exchange? How do existing electrolyte disturbances such as chronic respiratory acidosis affect body response to newly imposed stress such as surgery? This paper will attempt to sketch briefly material from recent literature which may help to answer such questions.

POTASSIUM DEFICIENCY

The signs of cellular potassium deficiency as defined and described by means of experimental studies present a specific clinical syndrome complete with electrocardiographic changes. However, such a condition seen in the post-surgical patient or accompanying a refractory case of chronic congestive failure may not be readily apparent. Usually the first complaints of the patient with early potassium deficiency are weakness and lassitude. With severe hypokalemia muscular weakness may advance to a state of flaccid paralysis, usually of the lower extremities, but involvement of the respiratory muscles may mask the basic disturbance. Smooth muscle dysfunction results in gastric and intestinal distention, nausea and vomiting, and occasionally paralytic ileus. With prolonged deficiency renal functional changes may occur which are not easily reversed and McAllen and others have reported myocardial pathology including necrosis which may present the clinical picture of myocardial infarction.¹

CELLULAR TRANSFER OF POTASSIUM

Potassium leaves the cells when metabolism ceases, and likely does so to a lesser extent when reversible metabolic disturbances such as anoxia or insulin deficiency occur. It has been postulated that the cellular cation deficit is replaced by sodium to the extent of 2/3 of the loss and by hydrogen ion for the remaining third, the hydrogen ion shift promoting a relative extracellular fluid alkalosis. Experimentally

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and clinically it appears that the concomitant lowered sodium plasma level which may occur cannot be raised to normal levels when potassium deficiency prevents replacement of intracellular potassium. Though there are mechanisms for the retention of sodium when depletion of that ion is threatened, there seems to be little evidence for significant support of cellular stores of potassium and Berliner and others have shown that even in dire deficiency states experimental animals and humans may continue to lose large amounts of that ion in the urine.\(^2\)

**EFFECT OF pH**

An increase in plasma pH tends to enhance movement of potassium into the cells; acidosis or decreased pH promotes potassium transfer from cells to extracellular fluid. In potassium-depleted animals the same relationship between serum potassium and pH is evident but serum levels tend to be somewhat lower. However, any significant rise in serum potassium is accompanied by transfer of potassium into the cells and the opposite transfer occurs with serum level decrease. Darrow develops the clinical implications of these experimental findings as follows.\(^3\)

In alkalotic states one would expect the serum potassium to be low even if cellular stores are normal, and especially low if cellular deficiency is present. Serum levels in alkalosis are not likely to be high unless renal function is impaired or unless disturbances of cellular metabolism cause intracellular loss. The increased tendency for potassium transfer into the cells when the pH of extracellular fluid is high has a bearing on therapeutic measures in alkalotic states with potassium depletion. Sudden decrease in the serum potassium level may accompany potassium therapy due to intracellular transfer when cellular stores are depleted, much as insulin may bring on low serum levels symptoms in the diabetic patient.

In acidosis the intracellular transfer of potassium is retarded by the decreased pH and serum levels are likely to be normal or high in spite of considerable cellular deficit. Low serum levels in acidic states are not likely to be high unless renal function is impaired or unless disturbances of cellular metabolism cause intracellular loss. However, renal functional impairment or significant changes in cellular metabolism must be considered here also. It must be remembered too that sodium bicarbonate used to correct acidotic imbalance will tend to accelerate cellular transfer of potassium and may cause rapid changes in serum potassium levels in spite of acidosis.

**ACIDOSIS**

Acidosis with potassium deficiency is not an uncommon clinical association. Many conditions leading to acidosis also result in potassium deficit; the most frequently seen are probably severe diarrheal states, starvation and base-depleting forms of renal impairment. More often, however, potassium deficiency is superimposed on an unrelated acidosis or conversely, the patient with chronic potassium deficiency becomes acidotic.

Bland states that though moderate potassium depletion in itself does not cause severe symptoms, it may and usually does so when associated with metabolic acidosis. The potassium depletion of alkalosis does not result in nearly so ill a patient as the same degree of depletion in the presence of metabolic acidosis. Muscular paralysis occurs more readily and earlier in the latter instance also.\(^4\)
Respiratory acidosis in itself does not seem to promote disturbances of potassium balance. The kidney compensates for the increased CO₂ tension of the blood by retaining bicarbonate and sodium ion and excreting hydrogen and chloride ions. However, it is most important to differentiate between respiratory acidosis and metabolic alkalosis. Both are characterized by a high serum CO₂ content and low serum chloride and the urine in both states may be acid in reaction and contain increased amounts of ammonia and chloride. Usually the clinical history and physical examination are sufficient to allow diagnosis; if not, a pH determination is the only reliable test unless measurement of CO₂ tension is available. The pH of the urine may help — an alkaline pH especially if associated with shallow respiration in the absence of pulmonary disease suggests metabolic alkalosis.

Bland notes that potassium depletion may be associated with an over-all acidosis of body fluids even in the presence of a raised serum bicarbonate. As the result of such findings he suggests treating clinical potassium depletion with potassium in combination with anions, such as citrate or lactate, which can be oxidized by the body.

In metabolic acidosis the renal demand for base for acid excretion depletes potassium as well as sodium. In addition the compensatory excretion of bicarbonate requires cations and accounts for more potassium loss. In the process of repair, experimental work reveals that the low bicarbonate level due to acidosis will not return to normal level if deficits of sodium and potassium have developed, since renal excretion of hydrogen ion and retention of bicarbonate can take place only if an adequate supply of those cations is present.

Sodium replacement in acidosis cannot be estimated from the bicarbonate level, as has been assumed in the past. Cellular cation deficit should be replaced by potassium. Some of the replaced sodium will enter the cells due to the increased plasma level but the extent of such transfer of sodium is not predictable. Therefore, Darrow recommends fluid replacement in acidosis consist of solutions resembling extracellular fluid, such as Hartmann’s Ringer lactate. He suggests potassium in sufficient amount to cover daily loss as well as some of the deficit be started simultaneously or as soon as restitution of renal function and cellular metabolism are adequate.

It is worth remembering that intestinal losses as well as renal losses can be a major factor in potassium depletion. Colonic loss of potassium as high as 70 mEq. per liter of stool has been reported in severe diarrhea. Schwartz and Relman have described severe potassium deficiency resulting from excessive use of cathartics over long periods of time with no apparent alkalosis. Decreased intake, dehydration and rapid therapeutic infusion of sodium solutions often contribute to the potassium deficit of diarrheal illness.

Potassium replacement therapy in diabetic acidosis has been generally accepted. If adequate potassium is available, the not uncommon practice of overcompensating for sodium loss by lactate or bicarbonate administration need not result in serious potassium deficit; however, there is still danger of making the patient hypernatremic.

The mechanism of the drop in potassium and phosphate stores in developing diabetic acidosis has not been clearly defined. That is intimately connected with

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cellular balance and the impaired carbohydrate oxidation seems evident from the rapid sequence occurring with therapy but the serum levels do not reflect the changes, according to Bland, and may even rise as renal function declines with increasing acidosis. Therefore, with very severe acidosis the serum levels are frequently high rather than low.

ALKALOSIS

A great deal of clinical and experimental evidence indicates that metabolic alkalosis will not develop if potassium intake is sufficient and that it cannot be corrected if potassium deficit exists. The renal compensation for metabolic alkalosis is essentially retention of hydrogen ions. This is accomplished by excretion of bicarbonate or organic acids with sodium and potassium. Since, as Darrow points out, there is usually a relative or absolute excess of sodium in the body in alkalosis, renal compensation is likely to produce only deficits of potassium.

Apparently potassium excretion is necessary to prevent alkalosis and to maintain plasma chloride levels. Either potassium chloride or potassium bicarbonate will bring about normal balance in potassium deficiency associated with alkalosis. Sodium from the cells is excreted with bicarbonate or with chloride if an excess of either ion is present.

Medical conditions prone to develop metabolic alkalosis include those tending to potassium deficiency. Vomiting, diarrhea, or continuous tube aspiration and prolonged administration of ACTH or cortisone-like substances are probably the most commonly encountered in medical practice. Excessive use of alkaline preparations, diuretic agents or the impaired hormonal response in certain renal or cardiac edematous states may be etiological factors.

Chronic respiratory alkalosis has seldom been the subject of physiological study. Hyperventilation experiments of relatively short duration have revealed no sustained impairment of sodium or potassium balance secondary to pH or pCO₂ changes but marked increase in excretion of base, particularly potassium, and retention of phosphorus have been noted. Recent studies with respirator patients who have been over-ventilated indicate that renal compensation in such cases may not be complete for as long as 5 months. In general, evidence seems to indicate that potassium deficit should be suspected in patients with central nervous system disease or psychic disturbances causing hyperventilation for long periods of time.

Acute respiratory alkalosis is seldom a problem for treatment. Most physicians are aware of the danger of mistaking the acute respiratory alkalosis of salicylate intoxication not uncommonly seen in children for diabetic acidosis.

POTASSIUM AND CALCIUM BALANCE

The relationship between potassium and calcium remains obscure though clinical observations seem to indicate some reciprocal function. Though improvement in the hyperkalemic abnormalities of the electrocardiograph hase been shown to follow the administration of intravenous calcium gluconate, the protective action of calcium against the toxic effects of hyperkalemia seems controversial. Krainin, et al. using dogs could demonstrate no significant evidence to support this hypothesis.
However, the not infrequently observed onset of tetanic symptoms due to low calcium serum levels soon after therapy is started in potassium depletion has been the subject of some conjecture. Bland quotes Rapoport, et al. to describe a postacidotic state following diarrhea therapy in infants. Restoration of water and electrolyte balance may be succeeded by reduction of calcium and phosphorus serum levels severe enough to cause tetany and by concurrent decreases in potassium and phosphate levels. This syndrome is more prone to occur if acidosis has been severe and therapy has been rapid with excessive amounts of sodium bicarbonate. Bland advocates supplementary calcium in spite of normal serum levels in diarrheal acidosis. Calcium should be given separately since it precipitates any phosphate in electrolyte mixtures. Darrow feels that the hypocalcemia in such conditions is often iatrogenic. He claims that he has seldom seen hypocalcemia in such infants when potassium was plentiful. Experiments with normal rats indicate that sodium solutions will produce hypocalcemia only when there is previous potassium depletion. He believes that tetany may be the result of too much sodium bicarbonate administered to patients deficient in potassium.

CONGESTIVE HEART FAILURE

Lown and Levine summarize the electrolyte changes in congestive failure as follows. There is an increase in body sodium though serum levels are moderately lowered due to dilution and intracellular transfer. Chloride concentration is slightly increased, tending to acidosis. Though serum potassium is normal, radioactive isotope technique has proved significant reduction of body stores of that ion. Heart muscle increases in weight with reduction in cellular potassium, phosphorus and creatine. Fasting, anoxia, acidosis, edema and prolonged exercise may induce additional potassium loss.

There is general agreement that significant electrolyte disturbances in congestive failure stem from therapy — the major factors being the salt-restricted diet and diuretic agents, almost all of which can induce potassium deficit. Potassium losses do not occur to as great an extent or as frequently as do those of sodium and chloride, and Bland is of the opinion that sodium and chloride diuretic losses will lead to symptoms before clinically evidenced potassium losses have occurred in most cases. But potassium deficiency should be anticipated in the patient with advanced failure, those who are having repeated predominantly chloride diureses or in patients receiving prolonged ammonium chloride therapy.

The patient suffering heavy losses of chloride may be in a state of hypochloremic alkalosis and on the verge of hypokalemia. Experimental studies with animals show that withholding chloride results in the same electrolyte imbalance as potassium deprivation. Giving ammonium chloride to such a patient causes further potassium loss because the kidney will excrete excess chloride with sodium and potassium the first 2 days of therapy until ammonium ion production begins to increase. Caution is also advised when using ammonium chloride in patients with metabolic alkalosis because of the possibility of misjudging what is actually respiratory acidosis. Even in the absence of respiratory impairment or renal disease, ammonium chloride may produce severe systemic acidosis in the patient with advanced failure, as well as hepatically induced ammonium intoxication in patients with liver damage.
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Seldin advocates the use of exchange resins for the therapy of the patient in failure but admits that repeated use of even the newer modifications intended to conserve potassium do result in some depletion of that ion as well as allow the risk of hyperchloremic acidosis due to the ion exchange.1

Lown and Levine feel that the major factor in the rising incidence of digitalis toxicity is the increasing dependence of treatment on electrolyte manipulation. Their work indicating that potassium deficiency is related to the threshold of clinical symptoms due to digitalis excess has been universally accepted. They state that in patients with chronic potassium depletion either through the renal or GI route with depression of serum levels, small amounts of digitalis can be fatal, though serum potassium is usually normal during digitalis intoxication.

They believe that postmercurial redigitalization is a state of increased myocardial sensitivity to toxic properties of digitalis resulting from the negative potassium balance precipitated by the diuretic therapy. They claim this can be prevented by giving potassium with the diuretic.

The clinical combination of congestive failure and hepatic cirrhosis is particularly likely to result in potassium depletion because such patients show an inexplicable tendency to lose potassium rather than sodium.4,12 Excessive aldosterone activity can cause a secondary potassium depletion and alkalosis. Potassium and serum bicarbonate levels should be guarded in those patients with cardiac, nephrotic or cirrhotic edema whose urine shows increased amounts of the adrenal cortical steroid that appears to be responsible for sodium retention.12

RENAL DISEASE

Schwartz claims in the course of an extensive review of the effect of potassium on the kidney that renal wasting is not seen in azotemic patients with primarily glomerular damage, but in those with tubular involvement predominant. This may include the diuretic phase of any acute renal insufficiency, however.12 Furthermore, both Berliner and Schwartz note that an occasional case of hyposthenuric renal insufficiency may show symptoms of potassium depletion due to renal losses.

Functional renal changes which improve with potassium replacement have been reported as resulting from the excessive use of cathartics, chronic diarrhea, ulcerative colitis and in other instances of severe gastrointestinal losses.6,12 Such renal disturbances included reduced urea clearance, polyuria, lowered glomerular filtration rate and renal plasma flow, and isosthenuria. These changes are reversible, but may require as long as four or five months.

THE SURGICAL PATIENT

The normal pattern of electrolyte response to surgery is a loss of potassium for 2-5 days with little or no change in potassium plasma levels according to Moore and Ball.13 During this time there is decreased urinary excretion of sodium but plasma sodium levels often fall, especially where trauma is extreme. They state emphatically that though the potassium loss is well tolerated by the average patient for three days,
the period of negative balance should not be allowed to continue over that time. The postoperative period of insufficient caloric intake is at least partially responsible for the electrolyte imbalance and also should not be extended more than 3-5 days postoperatively.

Such a program is not adequate for the previously depleted patient or the complicated surgical case. Harned and Cooke's therapeutic principles for preoperative and postoperative care of infants and children begin with the recommendation that preoperative chemical analysis of plasma or serum and urinalysis including specific gravity precede long or complicated surgery and that any alteration or imbalance be corrected where possible. They recommend repeated studies 12 hours after surgery and at 24-48 hour intervals thereafter as the clinical condition of the patient may warrant.

The surgical care of the patient who has been on a rigid cardiac routine or whose surgery is preceded by a period of starvation or severe vomiting or diarrhea demands that his electrolyte status be investigated and alterations recognized if not corrected prior to operation. Postoperative losses of potassium in such persons must be replaced as they occur. The usual urinary loss during the day of operation will be 50-90 mEq. depending on the nature of the operative procedure and for the three days following surgery will average 20-50 mEq. per day. When possible such losses should be replaced orally — otherwise potassium salt solutions may be added to intravenous fluids at a concentration not exceeding 40 mEq. per liter of fluid. Such infusion must be given slowly over a period of several hours and only when the patient's urinary output is known to be adequate, at least 500 cc. per day, and his plasma potassium is known.

Chloride deficiency and the adrenal response to trauma may quickly produce alkalosis in the patient with chronic potassium deficiency subjected to surgery. Such a state usually results in low plasma potassium levels and low chloride with a high plasma CO₂; these may be accompanied by a high blood urea nitrogen or non-protein nitrogen. Sodium may be normal or low. The patient with a long period of postoperative starvation is especially prone to such a hypokalemic change. Plasma pH and potassium levels should be investigated in the surgical case which suddenly shows a profound prostration and weakness, fever and distention.

Withholding of sodium from the postoperative patient except for replacement of extrarenal losses is generally accepted. Where sodium infusion in reasonable amounts does not elevate low plasma levels, it is advisable to check potassium, CO₂ and plasma protein levels and to add potassium chloride, at least 40 mEq. per day, to infusion fluids. Sodium in amounts concomitant with replacement should be given as chloride, bicarbonate or lactate as indicated by the individual case. Whole blood, protein and caloric replacement must be an integral part of the therapeutic program also. Evans remarks that the restriction of gastric and intestinal drainage to the shortest possible time, adequate daily replacement of sodium and potassium chloride above daily losses before and after operation, and the restriction of intravenous fluid therapy to what is actually required after intelligent appraisal of the individual's need make the surgeon's problem much easier.
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There are, of course, cases with refractory low sodium levels which are not due to potassium deficit. Among such is the postoperative hyponatremia in children requiring use of hypertonic saline described by Harned and Cooke and credited to antidiuretic hormone imbalance. Similar syndromes may be seen in intracranial diseases in children and in hypoadrenal conditions.

PYLORIC STENOSIS

The alkalosis associated with pyloric stenosis secondary to peptic ulcer or cancer has been commonly attributed to loss of chloride in excess of base. However, recent findings indicate that this is not the basis for the rise in pH in such patients. Black and Jepson compared balance studies from postgastrectomy recovery records of patients with and without clinical pyloric stenosis and found postoperative retention of potassium as well as sodium and chloride only in the cases with pyloric obstruction. Lans, Stein and Meyer also found potassium deficiency common in pyloric stenosis. Oral administration of potassium salts has been found to diminish the alkalosis even before operative correction of the obstruction. The etiology of potassium loss is not clear in these cases.

Clinically the implications of these findings are two-fold. Heavy administration of sodium and chloride or of other solutions without potassium will add to potassium depletion. The use of ammonium chloride in these patients will enhance potassium loss as well as cause base depletion in general.

Patients with fecal fistula are reported to have potassium deficit reflected by low serum levels, but with normal sodium and chloride balance and no alkalosis. In these cases oral therapy proved fruitless until the fistulae were closed surgically.

POTASSIUM ADMINISTRATION

When all fluids are parenteral Darrow advises that the potassium requirement is from 1-3 mEq. per 100 calories metabolized or 20-30 mEq. per square meter of body surface for twenty-four hours. Total fluid requirements for infants allow 25 mEq. per liter at the most and 60 mEq. per liter for adults. As a general practice it is best to limit the amount per liter of fluid for adults to 40 mEq. and greater concentrations should be given only with constant monitoring of the patient during administration. Deficits should be replaced during at least a 3-4 day period and a longer time if severe. One day’s requirement should not be given in less than 4 hours.

We are inclined to think of oral administration as a safe route without question. However, Schwartz warns that in the normal individual 2.5 mEq. per Kg. in a single dose causes a sharp rise in serum level and may cause toxic symptoms if given rapidly. In normal subjects peak serum and excretion levels occur 2-3 hours after ingestion and blood and urine are at initial levels in 4-5 hours. In patients with renal insufficiency as little as 1 mEq. per Kg. in a single oral dose can cause a sudden serum rise and prolonged elevated levels for many hours longer than in control subjects. However, in small doses given at intervals throughout the day, normal persons have been given as much as 600 mEq. daily with no ill effects.
HEMOGLOBIN — AN IMPORTANT BUFFER

Hemoglobin as a buffer is often overlooked as an important factor in electrolyte therapy. In anemia, transfusions provide not only volume replacement but also a medium for taking up hydrogen ions and reducing acidosis. This is especially important in the diabetic case. Bland states that hemoglobin is fully as important a buffer as bicarbonate and accounts for 60% of the CO₂ capacity of whole blood. Occasionally one sees a patient, often with renal insufficiency, whose electrolyte imbalance can be treated only after transfusions have corrected severe anemia. Darrow notes that although restoration of extracellular water and electrolytes is the most important means of correcting the circulatory failure of dehydration and deficit of sodium and chloride, transfusions make this therapy definitely more effective in both experimental animals and patients.

### TABLE ONE

<table>
<thead>
<tr>
<th>Conditions contributing to potassium depletion:</th>
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<tbody>
<tr>
<td>1. Metabolic alkalosis</td>
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<tr>
<td>2. Metabolic acidosis</td>
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<td>3. Periods of starvation</td>
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<td>4. Dehydration</td>
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<tr>
<td>5. Anoxia</td>
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<td>6. Trauma, stress, or adrenal hormone administration</td>
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<td>7. Renal insufficiency</td>
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<td>8. Repetitive diuresis — mercurial, acetazolamide, etc.</td>
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<td>9. Severe diarrhea or prolonged overuse of cathartics or enemas</td>
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<td>10. Insulin deficiency</td>
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<td>11. Pyloric stenosis or obstruction</td>
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<td>12. Postoperative states:</td>
</tr>
<tr>
<td>a. Loss due to surgery — 3-5 days</td>
</tr>
<tr>
<td>b. Upper GI losses over 2 liters per day</td>
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<td>c. Lower GI fluid losses</td>
</tr>
<tr>
<td>d. Excessive sodium administration</td>
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<tr>
<td>e. Heavy purulent discharge</td>
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### TABLE TWO

<table>
<thead>
<tr>
<th>Respiratory Alkalosis</th>
<th>Respiratory Acidosis</th>
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<tbody>
<tr>
<td>CO₂ tension decreased. Renal excretion of bicarbonate and sodium proceeds with retention of hydrogen and chloride ions. Muscle cell potassium level — decreased.</td>
<td>CO₂ tension increased. Renal retention of bicarbonate and sodium proceeds with excretion of hydrogen and chloride ions. Muscle cell potassium level maintained.</td>
</tr>
<tr>
<td>Metabolic Acidosis</td>
<td>Metabolic Alkalosis</td>
</tr>
<tr>
<td>CO₂ tension decreased. Renal excretion of metabolic acid proceeds at the expense of sodium and potassium loss. If sodium and potassium deficit develops, bicarbonate concentration cannot be restored since the cations are necessary for hydrogen ion excretion and bicarbonate retention.</td>
<td>CO₂ tension increased. Renal retention of hydrogen ions accomplished by sodium and potassium excretion with bicarbonate. Usually excess sodium is present so only potassium deficit is likely. If potassium is deficient there will be no correction despite abundant sodium excretion.</td>
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### TABLE THREE

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<thead>
<tr>
<th>Extrarenal Potassium Losses</th>
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<tbody>
<tr>
<td>Gastric juice</td>
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<tr>
<td>High free acid</td>
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<tr>
<td>Low free acid</td>
</tr>
<tr>
<td>Bile</td>
</tr>
<tr>
<td>Miller-Abbott tube drainage</td>
</tr>
<tr>
<td>Ileostomy</td>
</tr>
<tr>
<td>(more with ileostomy diarrhea)</td>
</tr>
<tr>
<td>Diarrheal stools</td>
</tr>
<tr>
<td>Formed feces</td>
</tr>
<tr>
<td>Purulent discharge</td>
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</table>
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BIBLIOGRAPHY


