CHAPTER 8

ACID/ALKALINE BALANCE

Introduction

Acid-/Alkaline Balance is a dualistic model representing the two opposite abnormalities of pH control. Failure to maintain normal pH may be associated with one or more of seven causative factors. They are:

1) Water/Electrolyte Imbalance
2) Anaerobic/Dysaerobic Imbalance
3) Glucogenic/Ketogenic Imbalance
4) Sympathetic/Parasympathetic Imbalance
5) Endocrine Insufficiencies
   a) Kidney
   b) Adrenal
   c) Testosterone, Estrogen, Progesterone
   d) Thyroid
   e) Posterior Pituitary
   f) Parathyroid
6) Chronic dietary imbalance with respect to the acid/alkaline character of foods
7) Respiratory Dysfunction

Acid/Alkaline balance is much unappreciated and abused. Unappreciated by the medical profession which chooses to ignore it altogether as a clinical entity until its severity has reached life and death proportions; and abused by so many in the alternative healing arts who throw around the words acidosis and alkalosis indiscriminately, blaming them for this or that condition, without the vaguest notion of the various mechanisms of pH control. In either case, you are seeing a state of ignorance whose existence is all the more surprising because the biochemistry of Acid/Alkaline balance is described in any elementary physiology text. Until the NUTRI-SPEC system was developed, no one bothered to apply this physiology clinically.
No one with even a rudimentary knowledge of biochemistry could argue against the primary importance of maintaining pH balance as a prerequisite to health. A patient with insufficient reserves of either acid or alkaline buffers is a patient in trouble. Your clinical nutrition practice is incomplete until you have the means to objectively measure Acid/Alkaline Imbalances, and, until you can offer patients the specific nutrition regimen they need to restore control of their pH maintenance system.

Regrettably, however, it is found that there is almost universal confusion over:

1) the factors controlling pH

2) the means to objectively measure Acid/Alkaline Imbalances

3) the nutritional tools to restore pH balance

Much of the misinformation clouding the minds of so many doctors derives from just a few very popular nutrition systems that have been heavily hyped and sold to a substantial number of alternative health care practitioners. These systems, while claiming to offer an analysis and treatment for pH imbalances, do not stand up well under scientific scrutiny.

The intent here is not to take issue with the promoters of the various nutrition techniques. Rather, it is to clear away the confusion regarding Acid/Alkaline Balance, replacing common misconceptions with a scientifically grounded understanding of the basics.

A Common Misconception

The most alarming misconception among nutritionists concerned with pH balance, one that seems to reign supreme in the minds of an appalling majority of doctors, is that Acidosis is ubiquitous among the sick of this world. Acidosis, they have been given to believe, is an accompaniment to, and even the primary cause of, every disease, every pain, every state of ill health to afflict humankind.

Wouldn't it be nice if it were that simple? Pump up your patients' alkaline reserves and cure them of anything?

And an Alkalosis? No such thing. Acid is bad, this theory contends, and alkaline is good. And there is no way one can have too much of a good thing.

In truth, excess alkalinity is just as harmful as excess acidity. To clear the confusion, all physiological systems are maintained in homeostasis by a negative feedback mechanism that operates in a dualistic manner. Dualistic means that for every normal condition (normal pH, normal body temperature, normal gastric secretion, etc.), there are two abnormals — abnormally high and abnormally low. To say that there is only one abnormal with respect to pH balance is to display a total ignorance of the most basic fundamentals of physiology.

This absolute phobia with respect to Acidosis is common enough among your colleagues and your patients that you will want to have a rational, scientific, rebuttal to their frequently emotion-charged fear of acid. Following is a presentation of frequently heard rantings and ravings from these faithful believers in "Acid Fighter" propaganda, along with some simple, yet thoughtfully conceived answers that you can use when challenged by such a believer.
The propaganda of these "acid fighters" implicates the acid forming diet, particularly eating meat, as the primary threat to a long, healthy life. Their argument centers on the fact that eating meat results in phosphoric and sulfuric acids as metabolic end products.

Well, so what?

"So what?!" they shriek in near hysteria. "Don't you know that these acids are metabolic poisons? Don't you also know that the burden of eliminating these toxic substances falls exclusively on the kidneys – Can't you imagine the devastation wrought on your renal excretory mechanism during a lifetime of over-indulging in these poisons?"

"Well, I beg your pardon, great Doctor of Pseudo Science, but the logic of your argument escapes me. After all, is not carbon dioxide another metabolic end product? Is CO2 not also a "metabolic poison?" You bet it is – and far more deadly than your much maligned phosphoric and sulfuric acids. You could triple your plasma concentrations of those acids and your body would adapt without batting an eye. Triple the partial pressure of CO2 in your blood and you are dead.

"So – how does your body save you from this deadly poison, CO2? Are you aware that the "burden" of eliminating this toxic substance falls exclusively on the lungs? Are your lungs being devastated by the continuous unrelenting assault of noxious gas?"

"Of course not.

"Your lungs have about as much trouble with CO2 as your kidneys have eliminating phosphoric and sulfuric acids. All in a day's work – no sweat.

"You may be surprised to learn the ease with which your kidneys handle the acid end products of meat metabolism. The efficiencies of renal function are such that doubling the dietary intake of meat protein only increases the work load of the kidneys by 10%. (1)

"No sweat.

"The point you need to understand is that the kidneys are not burdened to the point of overload in eliminating metabolic acids – they are merely doing precisely what they were designed to do – just as easily and naturally as your lungs eliminate CO2."

So – commit the above factual presentation to memory and use it. Furthermore, the next time some emotion-crazed Doctor of Pseudo Science extols the virtues of complex carbohydrates while demonizing meat and acids – calmly remind him that his beloved complex carbs result in the formation of 45% more carbonic acid than does meat, and that at the same time, the starches cause a build up of lactic acid in the tissues. Let him put that information in his acid-phobic pipe and smoke it. Kind of pulls the plug on his whole argument, doesn't it?

If your protein-basher hasn't yet retreated with his tail between his legs – here's another way to take the wind out of his sails. His claim that eating meat is bad for you because it results in the formation of phosphoric and sulfuric acids makes as much sense as saying that exercise is bad for you because it results in the formation of lactic acid. Right?

Is lactic acid a "metabolic poison?" Sure, so what. Your body is perfectly well equipped to handle it – just as it is perfectly able to handle normal healthy CO2 production, and, just as it is perfectly able to handle a healthy omnivorous diet that includes meat.
You could stuff your face with meat around the clock for days and still not produce the level of acidosis resulting from 15 minutes of hard running. So — if acid-forming meat is at the root of all your patients' health problems, then by all means they should avoid exercise like the plague. Sure.

By now your acid-phobic adversary is left without a pseudo scientific leg to stand on. And, you did him and his patients a big favor.

**Overview of Acid/Alkaline Imbalance**

You will learn as you read this chapter that Acid/Alkaline Balance is reactive as often as causative. In other words, pH imbalances are often secondary to more primary causes — most particularly secondary to other NUTRI-SPEC imbalances and their associated endocrine imbalances.

It is no coincidence that Acid/Alkaline is number 5 out of your Five Metabolic Balance Systems for consideration on each patient. The truth is that you are favorably influencing pH balance with your correction of each of the other four NUTRI-SPEC metabolic balance systems that you evaluate in each patient.

The percentage of your patients who show an Acid or Alkaline Imbalance as per question five of your QRG is not that high. Most patients do have pH abnormalities, but those pH abnormalities are either associated with one or more of the other imbalances, or, those pH imbalances are compensated to some degree such that they are not apparent as a pure Acid or Alkaline Imbalance. When patients test positive on question five of the QRG, you know they are in a rather severely de-compensated state. No matter what other imbalances they have, the Acidosis or Alkalosis needs specific therapeutic attention.

In describing the impact on patients of an Acid or Alkaline Imbalance we like to use the analogy of a person with a 20 pound weight strapped to his right shoulder. The weight may be light enough that the person is not devastated by it, nor even experiencing acute symptoms because of it. But so much of his reserves are preoccupied with trying to compensate for this load that he has difficulty performing even the most routine daily functions. Hour after hour he carries this burden which completely alters his ability to work, to eat, to rest, or to perform any other function.

Such is the metabolic burden associated with Acid/Alkaline Imbalance. Nothing else will work metabolically, nothing else will balance biochemically, as long as the load must be carried. The “weight” itself may be an Anaerobic Imbalance or a Glucogenic Imbalance or whatever, and will eventually have to be removed. But suppose you could remove 10 of those 20 pounds, immediately boosting the patient’s vital reserves. Such is analogous to what you are doing with your Phos Drops, your Proton Plus and so forth. You have not yet removed the weight (the other metabolic imbalances in this patient), but you have relieved the burden enough that the person can begin some semblance of function.

Your NUTRI-SPEC system has identified six types of Acidosis and Alkalosis. Each of these abnormal test patterns affects different parts of the body, and each has a different effect on urine and saliva pH’s. There are those who would try to convince you that the urine and saliva acidity or alkalinity varies directly with the acidity or alkalinity of the entire body. You are about to learn that this is simply not true. The various patterns of Acidosis and Alkalosis may vary either directly or inversely with the urine pH and/or the saliva pH.
To understand how the urine or saliva pH can vary either with or against the pH imbalance of the body requires that you know the simple clinical facts of life with respect to Acidosis and Alkalosis:

- Acid/Alkaline Imbalances always involve respiratory function
- Acid/Alkaline Imbalances always involve renal function

The respiratory and renal involvement in an Acidosis or Alkalosis may be either part of the cause of, or part of the compensation for the Acidosis or Alkalosis.

Consider now the respiratory system in Acid/Alkaline Imbalances. The respiratory system may be the primary cause of the imbalance, or, it may be the primary defense in compensation for the imbalance.

The respiratory system is causative by hyperventilation in a Respiratory Alkalosis. It is causative by hypoventilation in a Respiratory Acidosis.

The respiratory system is compensatory by suppressing respiration to retain carbon dioxide in an Alkalosis. The respiratory system is compensatory by stimulating respiration to blow off carbon dioxide in an Acidosis.

In any of your patients with an Acid or Alkaline Imbalance the respiratory rate will either be increased or decreased, depending on whether CO2 is being blown off or retained; and, the breath holding time will be increased or decreased, depending on whether CO2 is low or high.

Now, consider the kidneys in an Acid/Alkaline Imbalance. The kidneys are causative when they have lost their ability to either excrete or retain acid. The kidneys are compensatory in that they will do their best either to dump or to retain acids as needed.

When we put together all the considerations regarding respiratory system involvement and renal involvement in an Acid/Alkaline imbalance, we come up with what we could call the NUTRI-SPEC rule of Acid/Alkaline evaluation:

The urine pH and saliva pH help you identify the type of Acidosis or Alkalosis, but do not indicate the presence of an Acidosis or Alkalosis – only the respiratory rate and the breath hold do that.

In other words, no matter how outrageously high or low your patient’s pH’s are, you will only treat an Acid/Alkaline Imbalance if these pH’s are accompanied by an abnormal respiratory rate and/or breath hold time. If respiratory parameters are within normal limits, then the high or low pH is due to something other than an Acidosis or an Alkalosis.

You will look at these rules relating to the cause of, and the compensation for pH aberrations several more times throughout this chapter.

**Acid/Alkaline Imbalance Associated With Water/Electrolyte Imbalance**

Acid/Alkaline and Water/Electrolyte Imbalance are related to the extent that any type of Acidosis involves a tendency to dehydration, and any type of Alkalosis involves a decrease in extracellular fluid volume. Any imbalance, therefore, requires an increase in water intake, usually along with certain selected electrolytes.
A major problem in all your Electrolyte Stress patients and your Electrolyte Insufficiency patients is the inability to control fluid and electrolyte movement between the various extracellular and intracellular fluid compartments. This water/electrolyte problem is always associated with some loss of pH control in one or more of the body fluid compartments. Many of the dispersing agents and electrolytes you use for your Electrolyte Stress and Electrolyte Insufficiency patients are the same as the buffers used for your Acid and Alkaline patients.

Understand that anytime you correct a patient’s ES or EI Imbalance you have also corrected a pH imbalance. (In fact, in most cases you will have corrected more than one pH imbalance.)

**Acid/Alkaline Imbalance Associated With Anaerobic/Dysaerobic Imbalance**

There are tissue pH changes associated with Anaerobic/Dysaerobic imbalance. Truthfully, the tissue pH changes associated with Acid/Alkaline imbalance are of much less clinical significance, both in terms of severity and frequency, than are the acid/alkaline changes associated with Anaerobic/Dysaerobic Imbalance. In other words, as a clinician you will most often be influencing tissue pH abnormalities by dealing with Anaerobic/Dysaerobic Imbalance rather than with Acid/Alkaline Imbalance per se.

Anaerobic Imbalance is accompanied by a metabolic alkalosis in conjunction with a tissue acidosis. At the tissue level (and especially in lesioned tissue) there is anaerobic energy metabolism (fermentation). There is thus an accumulation of lactic acid in the interstitial fluid.

Dysaerobic Imbalance is typified by a metabolic acidosis concurrent with a tissue alkalosis. At the tissue level (and especially in lesioned tissue) there is dysaerobic oxygen metabolism which results in excess conjugated FA and increased fixation of chloride ions as they bind the FA double bonds. This excess cellular chloride fixation allows sodium to remain free in the interstitial fluid. The excess sodium combines with carbonate ions, forming alkaline compounds.

Never lose sight of the fact that aberrant oxidative metabolism (i.e., Anaerobic/Dysaerobic Imbalance) is the most important cause of tissue acidosis/alkalosis.

**Acid/Alkaline Imbalance Associated With Glucogenic/Ketogenic Balance**

Both Glucogenic and Ketogenic Imbalances are typified by abnormal carbon dioxide and bicarbonate levels. As you will read later in this chapter, carbon dioxide and bicarbonate levels are at the root cause of many Acid/Alkaline Imbalances. Your patients with Glucogenic/Ketogenic Imbalances are continuously struggling to adapt to their abnormal carbon dioxide and bicarbonate levels, and thus have a tendency to deplete their buffering system reserves. Your Glucogenic patients tend to be relatively acid at the systemic level, while your Ketogenic patients are excessively alkaline. The only way to correct the acid or alkaline conditions of these patients is to normalize their oxidative energy metabolism by reversing their Glucogenic or Ketogenic Imbalance such that normal levels of carbon dioxide are produced.

**Acid/Alkaline Imbalance Associated With Sympathetic/Parasympathetic Imbalance**

A Sympathetic Imbalance can influence Acid/Alkaline Imbalances in two ways. First, the Sympathetic patient will tend to show an over-stimulated respiratory center. Second, the Sympathetic patient is typified by renal vaso constriction. Both of these tendencies can lead to several different types of Acidosis or Alkalosis.
Your Parasympathetic patients can also easily have an acid or alkaline tendency. The parasympathetic patient will be typified by an inhibition of the respiratory center, as well as by bronchial constriction (as is found when there is an asthmatic condition).

**Acid/Alkaline Imbalance Associated With Endocrine Dysfunction**

Acid/Alkaline Imbalances can also be associated with endocrine dysfunction, as the endocrines control the movement of mineral elements. The control of sodium, potassium and chloride levels is associated with kidney, adrenal, sex hormone, and posterior pituitary function. The control of calcium, phosphorous and magnesium is associated with the kidneys, adrenals, thyroid, and parathyroids. (See the Chapters on Endocrine Dysfunction.)

**Acid/Alkaline Imbalance Associated With Dietary Imbalance**

Another prevalent misconception among clinical nutritionists concerns acid forming and alkaline forming diets. Acid ash foods (high in phosphorous, sulfur or chloride, or, low in potassium, magnesium or calcium) and alkaline ash foods (high in potassium, magnesium or calcium, or, low in phosphorous, sulfur or chloride) are widely believed to be the major factor influencing Acid/Alkaline Balance.

The truth, as you have just learned, is that acid forming foods and alkaline forming foods are only one of many factors influencing pH balance. And, in fact, the acid/alkaline character of the diet is one of the least significant of these factors (2) – yet it forms the entire basis of many doctors’ evaluation of pH.

A diet high in "alkaline minerals" such as potassium, as many doctors recommend, will not only not correct most forms of Acidosis – it can actually create a Potassium Excess Acidosis by interfering with the kidney's ability to eliminate acids, thus allowing acids to accumulate in the body.

Furthermore, a diet low in potassium will not only not cause an Acidosis, as many doctors believe – it can actually cause a Potassium Depletion Alkalosis by causing the kidneys to lose excess hydrogen ions, thus leaving the body too alkaline.

A summary of the dietary factors influencing Acid/Alkaline Balance is impossible because each patient responds differently to a particular food based upon what metabolic imbalances exist in that patient. This variability is yet another example of the NUTRI-SPEC key concept of biological individuality. To illustrate: fruit will make an Alkalosis patient more alkaline, yet will make an Acidosis patient more acid. Fruit (in moderation) will make a Dysaerobic patient less acid at the systemic level and less alkaline at the tissue level. An Anaerobic patient responds to fruit by becoming more acid at the tissue level, yet more alkaline at the systemic level.

**Acid/Alkaline Imbalance and Respiratory Dysfunction**

The lungs exert an influence on pH via their control of carbon dioxide, and thus the levels of carbonic acid and bicarbonate in the body.
Types of Acid/Alkaline Imbalance

Much of the confusion regarding Acid/Alkaline Balance stems from the fallacious assumption that Acidosis and Alkalosis are each singular disease entities. In actuality, there are several types of Acidosis and several types of Alkalosis. Each of these Acid/Alkaline Imbalances has a different effect on urine pH and on saliva pH. For example, a person with a Metabolic Acidosis will have an acid urine, but will have an alkaline saliva. And a person with a Potassium Excess type of Acidosis will often have both an alkaline urine and an alkaline saliva. So much for the simplistic notion that the pH of the body varies directly with the pH of the urine and saliva.

Clearly, the terms acidosis and alkalosis really have very little meaning in themselves, because there are actually several different types or patterns of Acidosis, and several different patterns of Alkalosis. For the purposes of the NUTRI-SPEC System, three different patterns of Acid Imbalance have been defined as clinical entities, and three different patterns of Alkalosis have been identified.

Each of the six patterns of Acid/Alkaline Imbalance will now be discussed, giving a brief summary of the physiology associated with each, followed by a description of the commonly associated clinical findings, as well as recommended supplementation.

We could have written a 50-page chapter on Acid/Alkaline Imbalance. We could have gone into great depth on the acid buffering action of the imidazole group of the amino acid histidine as it occurs in the hemoglobin molecule. We could have talked at length about how the electronegativity of the blood associated with the release of oxygen and the uptake of CO2 affects the ability of the blood to buffer acids. We could have discussed the importance of amino groups forming carbamino groups with CO2, and how this function is impeded when hydrogen is fixed by the amino group to form ammonium ions. We could have explored the dissociation of carbonic acid into hydrogen and bicarbonate ions under the influence of carbonic anhydrase enzyme. We could have given you enough biochemistry to keep your head spinning for weeks.

But the beauty – the gloriously simple beauty – of your NUTRI-SPEC system is that all that technical gobbledygook has been distilled down to a few easy to understand clinical facts:

1) Acid/Alkaline Imbalances always involve respiratory function.

2) Acid/alkaline Imbalances always involve renal function.

So, as you will learn below: (3)

1) The respiratory system is always associated with an Acidosis or Alkalosis. The respiratory system may be the primary cause of the imbalance, or, it may be the primary defense in compensation for the imbalance. But whether as cause or effect, the respiratory system is always part of the clinical picture.

The respiratory system is causative in a Respiratory Acidosis or Alkalosis. A Respiratory Alkalosis is caused by hyperventilation (CO2 is blown off faster than it is metabolically produced). The decreased CO2 means decreased carbonic acid – thus the Alkalosis.
A Respiratory Acidosis is caused by hypoventilation (metabolically produced CO2 is produced faster than it can be blown off). The increased CO2 retention means increased carbonic acid — thus the Acidosis.

The respiratory system is an essential part of the compensation for the various types of Metabolic Acidosis or Alkalosis. In an Alkalosis, respiratory activity is suppressed so that CO2 (and thus carbonic acid) can be retained to decrease the Alkalosis. In an Acidosis, respiratory activity is stimulated to blow off CO2 and lower carbonic acid levels.

Do you see how easy it is to spot an Acid/Alkaline Imbalance once you know the pH - respiration connection?

The respiratory rate will either be increased or decreased, depending whether CO2 is being blown off or retained; and, the breath holding time will be increased or decreased, depending on whether CO2 levels are low or high. So, your NUTRI-SPEC test procedures do not even consider Acid/Alkaline Imbalances if the respiratory rate and breath hold are normal.

2) The kidneys will always be doing what they can to compensate — excreting acid urine in an acidosis, or alkaline urine in an alkalosis, unless — they are hampered by either potassium excess or potassium depletion, which impede the ability to excrete or to retain acid, respectively.

Putting facts 1) & 2) together, we see that the urine pH helps you identify the type of Acidosis or Alkalosis, but does not indicate the presence of an Acidosis or Alkalosis — only the respiratory rate and breath hold do that.

Once again, do not go tilting at windmills when your patient has high or low urine or saliva pH. No matter how outrageously high or low your patient's pHs are, you will only treat an Acid/Alkaline Imbalance if these pHs are accompanied by an abnormal respiratory rate and/or breath hold time. If respiratory parameters are within normal limits, then the high or low pH is due to an imbalance other than an Acidosis or Alkalosis.

**Metabolic Alkalosis**

The first pattern for discussion is that of a Metabolic Alkalosis. In a Metabolic Alkalosis we find in the extracellular fluids a low hydrogen ion (H+) concentration and high bicarbonate, which gives us a high, or alkaline, pH. To compensate for this alkaline condition two things will happen. First, there will be a compensatory suppression of the respiratory center in an attempt to retain carbon dioxide, which leads to increased carbonic acid (H₂CO₃), and an acid saliva. The respiratory compensation is not alone capable of restoring normal pH. A 50-75% compensation is achieved, with the kidneys performing the rest of the compensation. (Note that saliva pH depends largely on the relative concentrations of free CO2 and combined CO2. High CO2 means high carbonic acid and an acid saliva. Low CO2, conversely, means an alkaline saliva.)

The kidneys, meanwhile, also compensate. We must pause here for a few words about kidney function. Since the natural diet is a very low sodium diet, our kidneys are designed with the capacity to retain sodium to protect us from sodium depletion. The kidneys, in order to retain sodium, must exchange for each sodium ion retained either a potassium ion or a hydrogen ion. In
a person on a natural diet, therefore, the kidneys will be seen to retain sodium and excrete potassium and/or hydrogen.

This brings us then to the means by which the kidneys compensate for a Metabolic Alkalosis. In a Metabolic Alkalosis there will be a compensatory renal tubal decrease in sodium-hydrogen exchange. There will be an increase in sodium-potassium exchange. In other words, the kidneys will dump potassium, and eventually even dump sodium, as they retain as much hydrogen as they possibly can. Along with the increased retention of acid there is an increased retention of ammonia, mostly in the form of ammonium chloride.

In addition to the retention of acid, the kidneys compensate in a second way. They increase the secretion of bicarbonate. Since the bicarbonate concentration is elevated in a Metabolic Alkalosis, the amount entering the glomerular filtrate is greater than that which can be reabsorbed by the renal tubules. Therefore, bicarbonate passes into the urine and the urine pH increases. In order to maintain electric neutrality, each of the bicarbonate anions is matched by a cation (the potassium and sodium described above) in the urine.

There is a reciprocal relationship between the amount of bicarbonate and the amount of chloride excreted in the urine. Since the compensation for a Metabolic Alkalosis involves an increased bicarbonate excretion, the urine chloride concentration is decreased. This chloride retention allows the concentration of chloride in the body to increase. The net effect of renal compensation is to decrease the body pH toward normal, to decrease the bicarbonate concentration toward normal, a decrease in the body's concentration of potassium and sodium, and an increase in the chloride level in the body.

Causative factors of a Metabolic Alkalosis include the following:

1) Loss of acid (excluding H2CO3)
   a) Loss of gastric juice (vomiting)
   b) Poor urinary retention of H+ with its associated anions (Cl, P, S, N)

2) Excess consumption of bicarbonate, Na, or other alkaline salts, which causes a decreased H+ activity of the extracellular fluid; antacids

3) Use of diuretics, which cause water, cation, and chloride depletion, while bicarbonate is retained
   a) Loss of H+, K+, and Mg ++ and buffers exceeds the loss of Na+ = can result in potassium depletion and magnesium deficiency

4) Cl depletion (See 1) a) and b), and 3), above)

5) Potassium depletion, causing an increased exchange of H+ for Na+ in the kidneys, allowing H+ to be excreted and bicarbonate to be retained.

6) Adrenal aldosterone excess, causing increased sodium retention, and urinary loss of buffers, H+, K+, and Cl-

7) Excess consumption of lactate, citrate, acetate, carboxylate, etc.

8) Decreased extracellular fluid and NaCl
9) Deficient production of acid (excluding carbonic)
   a) Liver insufficiency (e.g., secondary to cirrhosis)

10) May occur post ventilator therapy for Respiratory Acidosis

The effects of a Metabolic Alkalosis include the following:

In an Alkalosis, an increased amount of potassium moves into the cells, while sodium and hydrogen move in the opposite direction. Thus we find an increased potassium in the urine and in the saliva and a lower serum potassium. An Alkalosis is also accompanied by an increased glycolysis, which moves phosphorus into the cells, but is also accompanied by a loss of phosphorus in the urine. This decreases the phosphorus levels in both the saliva and the blood.

A Metabolic Alkalosis also causes increased fat and decreased carbohydrate metabolism. This leads to ketosis. Ketosis, in turn, however, can cause a Metabolic Acidosis, with excretion of acid urine containing ketones.

Another effect of a Metabolic Alkalosis is nervous over-excitability. This state of neurological facilitation can be associated with muscle spasms, and when taken to an extreme, can even be associated with tetany and seizures.

The final effect of Metabolic Alkalosis (when it is prolonged) is a decrease in gastric secretion.

What are the clinical findings in a Metabolic Alkalosis? The saliva pH will tend to be low, or acid. While there are many influences on saliva pH, the biggest contributing factor is carbonic acid which, as we have shown, increases as part of the compensation for Metabolic Alkalosis. Let us pause for a moment and contemplate what we have just learned; a Metabolic Alkalosis is accompanied by an acid saliva. What does this fact reveal about all the methods of alternative therapy that use saliva as a clinical indicator, concluding that acid saliva corresponds to an acidosis and alkaline saliva means an alkalosis? It reveals an ignorance of the physiology involved.

Other clinical findings in a Metabolic Alkalosis include a urine pH that will be increased, or alkaline, due to the decreased kidney excretion of acid. The respiratory rate is decreased, as there is a suppression of the respiratory center. The breath holding time will increase. The pulse will tend to be lower. The skin is often dry. And finally, calcium, which requires a certain H+ concentration to remain in solution, precipitates out of body fluids, potentially causing a number of problems including calcium deposits in the soft tissues; bursitis and rheumatic type pains associated with calcium carbonate crystals precipitating on nerve endings; osteoarthritis; stiffness of muscles and joints; tetany; irritability; neuro-muscular hyperexcitability.

Recommended supplementation for a Metabolic Alkalosis:

1) H20
2) Phosphoric Acid
3) Sodium Glycerophosphate
4) Hydrochloride (use only if associated with chloride loss, (as in vomiting, diuretics, etc.) or, if urine pH is > 6.5)
5) Phosphatase enzyme (raw bone is a good source)
6) Aspartic acid
7) (If diuretics) then magnesium aspartate or magnesium chloride
8) (Reduce any excess intake of bicarbonate, citrate, carboxylate, lactate, acetate, etc.)
9) Sodium chloride (use salt freely if blood pressure is low, as long as there is no problem with fluid retention)
10) Correct Glucogenic/Ketogenic Imbalance
11) Correct Sympathetic/Parasympathetic Imbalance

Potassium Depletion Alkalosis

The next pattern we want to look at is a Potassium Depletion Alkalosis. This is actually a form of Metabolic Alkalosis (in other words low H+, high bicarbonate, high pH and high CO₂) associated with depletion or chronic deficiency of potassium. In the normal kidney there is an on-going attempt to retain sodium; but for every sodium ion the kidney wants to keep it has to exchange either a potassium or hydrogen ion. Normally about equal numbers of potassium and hydrogen ions are exchanged. But suppose this person is low in potassium, so that there is no potassium available to exchange for sodium? Now, the only way for the kidney to perform its job of retaining sodium is to exchange hydrogen for it; there is no alternative. So, as all the body's H+ is being dumped into the urine, what happens to the pH of the body fluids? As the H+ levels drop lower and lower, the extracellular fluids become more and more alkaline; Potassium Depletion Alkalosis.

As you can see, we have here an Alkalosis pattern with a "paradoxical aciduria." The urine ammonia increases with the H+. The urine remains acid only until H+ is depleted, however, then becomes alkaline.

However, chronic potassium depletion impairs the renal acid secretory mechanism, thus increasing urine pH. Decreased H+ in the urine is compensated by increased ammonia excretion in the urine. "Paradoxical aciduria" then occurs during potassium repletion.

Causative factors in a Potassium Depletion Alkalosis include:

1) Chronic dietary potassium deficiency
2) Use of diuretics that cause potassium and chloride depletion.
3) Anterior pituitary stress (ACTH), glucocorticoid stress, or steroid therapy, which result in urinary potassium loss.
4) Mineralocorticoid stress resulting in decreased potassium retention, and increased sodium retention.
5) 3) and 4) above (unlike other forms of Metabolic Alkalosis) are accompanied by increased extracellular fluid and NaCl.
Potassium Depletion Alkalosis is accompanied by a shift of hydrogen from extracellular to the intracellular. This hydrogen shift results in an intracellular acidosis. Therapeutic attempts to acidify this alkaline patient must be monitored very carefully in order to avoid exacerbating the intracellular acidosis.

Clinical findings in a Potassium Depletion Alkalosis are as follows:

First, the urine pH is decreased due to the increased H+. In a chronic case of Potassium Depletion Alkalosis, however, the kidney's ability to secrete acid is impaired and the urine pH will be increased. But in most cases the urine is acid, and it will remain acid as long as there is H+ available to dump in the urine. When H+ is depleted, then the pH starts to increase.

The saliva pH in a Potassium Depletion Alkalosis is decreased for the same reason it was in the Metabolic Alkalosis pattern, i.e., increased carbonic acid. Look at this – an Alkalosis pattern which is generally associated with both an acid saliva and acid urine. Again, do not be misled by those who would have you believe that urine and saliva pH parallel the pH of the body.

In a Potassium Depletion Alkalosis we also find a decreased respiratory rate and an increased breath holding time, and a pulse which is somewhat slow. And again, we have calcium precipitating out of body fluids with its associated symptoms; sclerosis, bursitis, osteoarthritis, cramping, and so on. We also have muscular weakness. This is a peculiar muscular weakness as those patients often appear to have muscular development well above the average. (That is a clinical observation which is conjectured to be related to these patients' inherently strong adrenal function.) Typically, there is impaired gastro-intestinal motility, with constipation a frequent complaint among these patients. Polyuria is another complaint, i.e., increased quantity and frequency of urination.

Recommended supplementation for a Potassium Depletion Alkalosis:

1) H20
2) Di-Potassium Phosphate
3) Potassium chloride
4) Hydrochloride
5) Phosphoric acid
6) Magnesium Chloride
7) Phosphatase enzyme (Bone)
8) (Reduce any excess intake of bicarbonate, carboxylate, lactate, citrate, acetate, etc.)
9) Reduce salt intake
10) Correct Anaerobic/Dysaerobic Imbalance
11) Correct Glucogenic/Ketogenic Imbalance
Metabolic Acidosis

Now consider one of the Acidosis patterns; a Metabolic Acidosis. In a Metabolic Acidosis, the H+ concentration is high and the bicarbonate is low, giving the extracellular fluid a low pH. What compensatory mechanisms would you expect to see here? The lungs and the kidneys are going to swing into action, are they not? You will have a compensatory acceleration of the respiratory rate as the lungs try to blow off as much CO₂ as possible, thus lowering carbonic acid. Decreasing the CO₂ increases the pH toward normal, but only a 50-75% compensation is achieved. The kidneys do the rest.

The kidneys will dump as much H+ as they possibly can, along with ammonia. Ammonia is excreted in direct proportion to the acidity of the urine and to the duration of the Acidosis (thus conserving sodium and maintaining electric neutrality). Because there is a reciprocal relationship between bicarbonate and chloride excretion, urine chloride increases as part of the compensation for a Metabolic Acidosis, thus decreasing the chloride level in the body.

Causative Factors in a Metabolic Acidosis include:

1) Diarrhea = loss of bicarbonate, sodium and other alkali, and H₂O
2) Deep vomiting = loss of bicarbonate, sodium and other alkali, and H₂O
3) Renal loss of bicarbonate, potassium, and sodium
4) Excess endogenous production of organic acids, which may lead to depletion of alkaline cations excreted with the acid anions
   a) Diabetic keto-acidosis
      1) Diabetic = increased fat metabolism = increased liver formation of acetoacetic acid in quantities greater than can be oxidized by other tissues. This acid must be excreted in the urine. Only about 1/4 to 1/3 of the acetoacetic acid can be excreted by the kidney as acid. Therefore, the remaining 2/3 to 3/4 takes cations such as Na⁺ and K⁺ with it. This cation depletion is a significant factor in diabetics.
   b) Lactic acidosis associated with diminished tissue oxygenation
   c) Fatty acids
   d) Carbonic acid
5) Excess chloride intake, if it cannot be combined with ammonia for excretion, will cause an increase in sodium, potassium and bicarbonate lost in the urine
6) Excess dietary intake of organic acids which may cause a depletion of alkaline cations excreted in combination with these acids
7) Carbonic anhydrase inhibitors
8) Failure of renal acid excretion (e.g., renal insufficiency or adrenal insufficiency)
9) Excess dietary potassium, which causes decreased H⁺ secretion and decreased bicarbonate re-absorption
The effects of a Metabolic Acidosis are as follows:

When there is Metabolic Acidosis, potassium moves out of the cells with sodium and hydrogen moving opposite. This increases serum potassium while decreasing potassium levels in the saliva and urine. There is also decreased extracellular fluid volume. Accompanying a Metabolic Acidosis there is dissolution of cellular phosphorous. There is thus a lower phosphorous level in the cells, and saliva, while there is an increased blood phosphorus and phosphorous loss in the urine.

A Metabolic Acidosis is associated with a decreased oxygen carrying capacity of the blood. This oxygen deficit leads to an increase in the pulse rate.

Your clinical findings in a Metabolic Acidosis include an alkaline saliva associated with the low carbonic acid; an acid urine associated with the elevated H+ and ammonia; an increased respiratory rate and a decreased breath holding time; a somewhat rapid pulse, and a dramatic increase in the pulse as the patient stands up from the supine position. There may be a cold sweat to the palms of the hands and a dry mouth. The urine output is decreased.

Recommended supplementation for a Metabolic Acidosis includes:

1) H2O
2) Na or K Bicarbonate
3) Di-K Phosphate and/or Sodium Glycerophosphate
4) Na or K Citrate
5) NaCl + H2O
6) Magnesium Citrate
7) Lysine, Arginine, Histidine, Hydroxyglcine, Glutamine
8) (Reduce any excess intake of organic acids (juices, fruit, acid amino acids, etc.))
9) Correct Glucogenic/Ketogenic Imbalance
10) Correct Sympathetic/Parasympathetic Imbalance
Renal/Potassium Excess Acidosis

Another pattern of Acid Imbalance is a Renal or Potassium Excess Acidosis. This is a type of Metabolic Acidosis (in other words high H+, low bicarbonate, low pH and low CO₂) associated with excess kidney retention of acid. It can be a renal problem, in which case we need to consider once again the exchange in the kidneys of potassium and hydrogen for sodium. If there is excess potassium, more potassium and less hydrogen is exchanged for sodium. This means a decrease in urine H+ which means an increase in urine pH. And as this H+ is being retained instead of excreted, what happens to the body fluids? Obviously, they become progressively more acid.

If the potassium excess is associated with excess intake, then potassium retention is decreased, in other words, urinary potassium is increased. If the potassium excess or the renal insufficiency is associated with adrenal insufficiency, the potassium retention will be increased, i.e., the urine potassium is decreased. In this case, sodium and chloride retention will be decreased.

As in the Metabolic Acidosis pattern, there will be a decreased extracellular fluid volume, and dissolution of cellular phosphorous. If this is a Potassium Excess Acidosis there will be a decreased phosphorous level in the cells, and the saliva; an increased phosphorous in the urine (decreased phosphorous retention) until phosphorous levels have been depleted. If this is a Renal Acidosis there will be excess retention of phosphates, i.e., decreased urinary phosphorous and increased phosphorous in the blood and saliva.

Clinical findings in a Renal/Potassium Excess Acidosis: the saliva pH is variable. The decreased carbonic acid tends to increase the pH of the saliva, but the accumulation of organic acids tends to decrease it. The urine is alkaline due to the decreased H+ excretion. The respiratory rate is increased and breath holding time decreased. The pulse will be somewhat increased unless, as is often the case, there is a degree of adrenal insufficiency. In any case, the pulse will increase dramatically upon standing from the supine position. The mouth will be dry and the urine output decreased. The oxygen carrying capacity of the blood is below normal.

Recommended supplementation for a Renal/Potassium Excess Acidosis:

1) H₂O
2) Na Bicarbonate (may make saliva more acid)
3) Sodium Glycerophosphate
4) Na Citrate
5) Ca and/or Mg Citrate
6) Phenylalanine
7) (Reduce any excess intake of organic acids: juice, fruit, acid amino acids, etc.)
8) Correct Anaerobic/Dysaerobic Imbalance
9) Correct Glucogenic/Ketogenic Imbalance
Respiratory Alkalosis

There is an Alkalosis pattern and an Acidosis pattern in which the lungs play the primary role. These are a Respiratory Alkalosis and a Respiratory Acidosis.

In a Respiratory Alkalosis we have a low H+ concentration or high pH of the extracellular fluid due to a loss of CO₂. (CO₂ can only be decreased by hyperventilation, i.e., CO₂ being blown off faster than it is metabolically produced.) The loss of CO₂, of course, decreases carbonic acid, resulting in an Alkalosis. The kidneys will compensate by excreting bicarbonate, sodium and potassium, and by retaining H+, ammonia and chloride. The respiratory rate is irregular, i.e., may be increased or decreased.

Causative factors in a Respiratory Alkalosis include:

1) Hyperventilation associated with chronic or acute anxiety
   a) (Fear increases the respiratory rate faster than it increases the pulse)

2) Hyperventilation associated with low blood pressure
   a) When the mean blood pressure is less than 85, blood flow through the aortic and carotid bodies decreases. This decreases the aortic and carotid partial pressure of oxygen, thus stimulating chemoreceptors to increase the respiratory rate.
   b) You must consider the cause of the low blood pressure (Electrolyte Insufficiency, Dysaerobic Imbalance, Parasympathetic Imbalance, etc.).

3) Hyperventilation in compensation for a Metabolic Acidosis

4) Hyperventilation associated with salicylates

5) Hypoxemia (due to abnormal pulmonary gas exchange)

6) Fever

7) Gram-negative infection

8) Liver cirrhosis (or hepatic coma)

9) Primary CNS disorders

The effects of a Respiratory Alkalosis include:

Respiratory Alkalosis is accompanied by an increased glycolysis, which moves phosphorous into the cells, thus decreasing phosphorous levels in the urine, saliva, and serum. Since there is a phosphorous excess at the cellular level, there is merely a qualitative deficiency at the blood level. This means that phosphorous supplementation need not be given in large quantities, despite the appearance of low phosphorous levels.

Cerebral tissue also experiences a rise in pH and a fall in CO₂. This results in cerebral vasoconstriction which, in turn, means cerebral hypoxia, and can lead to seizures. Blood lactate and pyruvate increase in a Respiratory Alkalosis, while blood levels of ionized calcium decrease.
A Respiratory Alkalosis decreases gastric secretion. It also causes (just as it can be caused by) nervous over-excitability. This excessive neuro-muscular excitability can be associated with muscle spasms even to the point of tetany. This over-excitability is another factor that can precipitate seizures in the Respiratory Alkalosis patient.

One other effect of a Respiratory Alkalosis is an increase in fat metabolism and a decrease in carbohydrate metabolism. This shift in metabolism can lead to ketosis. Ketosis, in turn, however, causes a Metabolic Acidosis with excretion of acid urine containing ketones. It is not uncommon for patients to vacillate between a Respiratory Alkalosis and a Metabolic Acidosis.

The clinical findings in a Respiratory Alkalosis include an alkaline saliva due to the loss of CO₂, and an alkaline urine due to renal compensation. The respiratory rate may be increased or decreased. The respiratory rate in these patients is often quite irregular. If you observe their breathing you are likely to see rather long intervals during which they do not breathe at all, followed by two or three rapid shallow breaths, then another period of apnea, and so on. These people frequently experience air hunger, or the inability to "catch their breath." The breath holding time may be decreased or increased. As in other patterns of Alkalosis, there is a tendency to tetany and other calcium precipitation symptoms. There are often paresthesias of the extremities. There is often lightheadedness or even syncope.

Recommended supplementation for a Respiratory Alkalosis:

1) H₂O
2) Carbonated water
3) Phosphoric acid
4) Di-K Phosphate and/or Sodium Glycerophosphate
5) Hydrochloride
6) Glutamine, Tyrosine
7) Mg and/or Ca Aspartate (especially if there are ketones in the urine)
8) Correct Anaerobic/Dysaerobic Imbalance
9) Correct Glucogenic/Ketogenic Imbalance
10) Correct Sympathetic/Parasympathetic Imbalance
11) Consider anxiety as a causative factor
12) Consider fever/infection
13) (During a crisis) re-breathe expired CO₂ from a paper bag
Respiratory Acidosis

The last of our patterns of Acid/Alkaline Imbalance is the Respiratory Acidosis pattern. In a Respiratory Acidosis we have a high H+ and a low pH of the extracellular fluid due to a decreased excretion of CO₂ through the lungs. (Hypoventilation, i.e., CO₂ being blown off at a slower rate than it is metabolically produced, is the sole cause of a Respiratory Acidosis.) A classic example of an acute Respiratory Acidosis is the asthma patient. A classic example of a chronic Respiratory Acidosis is the emphysema patient. The inability to blow off CO₂ increases the carbonic acid level, thus the Acidosis. The kidney responds to the Acidosis by decreasing retention of hydrogen, chloride, ammonia, phosphoric acid, and, if the adrenals are weak, sodium. The kidney increases retention of bicarbonate, potassium, and, if the adrenals are strong, sodium.

An interesting phenomenon can now occur. The kidney retention of bicarbonate further increases the CO₂ and carbonic acid levels, which can actually perpetuate the imbalance. The kidney dumps even more acid, and before the thing is finished, the body is depleted of acid and chloride. What has happened is that the patient has traded a Respiratory Acidosis for a Metabolic Alkalosis.

This vacillation between a Respiratory Acidosis and a Metabolic Alkalosis is typical of many patients, and the asthmatic is a classic example. The Metabolic Alkalosis over-stimulates the parasympathetic nervous system (see Sympathetic/Parasympathetic Balance), including the vagus nerve, which causes bronchial spasms, which precipitates the CO₂ retention of the asthma attack, which puts the patient into a Respiratory Acidosis, which stimulates the kidneys to dump acid, which swings the patient back into a Metabolic Alkalosis, and the cycle begins all over again. Treating the Respiratory Acidosis that you observe during the asthma attack frequently aggravates the problem by pushing the patient even more quickly and deeply into a Metabolic Alkalosis, thus stimulating more vagus irritability. Over the long term, the key to effective therapy is to treat the pattern of Parasympathetic Stress, plus whatever other patterns of metabolic imbalance are involved.

Another clinically significant consequence of Respiratory Acidosis is the loss of chloride due to the kidney's compensatory response. The decreased chloride retention can result in a gastric hydrochloric acid insufficiency.

Causative factors in a Respiratory Acidosis include:

1) Hypoventilation (may be increased respiratory rate, but breathing is shallow or congested) associated with the airway obstruction of respiratory infections, or with the bronchial constriction of asthma, or with the decreased pulmonary surface area of emphysema

2) Hypoventilation in compensation for a Metabolic Alkalosis

3) Hypoventilation associated with high blood pressure

   a) When the mean blood pressure is greater than 115, blood flow through the aortic and carotid bodies increases. This increases the aortic and carotid partial pressure of oxygen such that there is stimulation of chemoreceptors to decrease the respiratory rate. This hypoventilation can lead to a Respiratory Acidosis.

   b) You must consider the cause of the elevated blood pressure (Electrolyte Stress, Anaerobic Imbalance, Sympathetic Imbalance, etc.)

4) Hypoventilation associated with brain stem damage, which decreases breathing
The clinical findings in a Respiratory Acidosis include an acid saliva due to the increased levels of CO₂ and carbonic acid, and an acid urine resulting from the high urinary excretion of hydrogen, ammonia, and phosphoric acid, and retention of bicarbonate. The respiratory rate is increased unless high blood pressure is the causative factor, in which case the respiratory rate is decreased. The breath holding time is decreased. The pulse tends to be somewhat rapid, and the orthostatic pulse shows a dramatic increase upon standing. There is, as in other forms of Acidosis, a decreased oxygen carrying capacity of the blood.

Recommended supplementation for a Respiratory Acidosis:

1) H2O

2) Sodium or Potassium Citrate

3) Di-Potassium Phosphate and/or Sodium Glycerophosphate

4) NaCl (Unless blood pressure elevated)

5) Magnesium chloride

6) Correct Anaerobic/Dysaerobic Imbalance

7) Correct Glucogenic/Ketogenic Imbalance

8) Correct Sympathetic/Parasympathetic Imbalance

Buffering Systems

In the above explanation of the several types of Acidosis and Alkalosis we have discussed many of the buffering systems, both the blood buffers and the urinary buffers. We have included in our discussions some mention of bicarbonates, phosphates, citrates, and ammonia. While these buffering systems are important to understand in your evaluation and treatment of your Acid/Alkaline Imbalance patients, there is one other buffering system that dwarfs all these others in its importance.

By far the most powerful buffers, both in the plasma and within the cells, are the protein buffers. At least 75% of all buffering power of body fluids occurs intracellulary, and most of this is via proteins. Here you see one more example of the importance of adequate dietary protein. When protein levels are only marginally adequate, as they are in the currently popular high carbohydrate diet, a person's protein buffering system begins to lose much of its punch. The person, therefore, is much more susceptible to Acid/Alkaline Imbalances.
Another comment about buffering systems is indicated here. You have seen above that many of the same supplements are recommended for both an Acid and an Alkaline Imbalance. Many doctors find it confusing that, for example, phosphates of sodium or potassium can be beneficial for an acid and an alkaline patient. The reason is that the buffering systems of the body (which include bicarbonates, phosphates, citrates, ammonia, hemoglobin, and protein buffers) are equally capable of buffering excess acidity or excess alkalinity. The following is an illustration of how sodium phosphate can neutralize both hydrochloric acid and lye:

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

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

The Acid/Alkaline Page of your Quick Reference Guide

Your Quick Scan for your Acid/Alkaline QRG analysis consists of just three tests – the respiratory rate, the breath hold time and the ratio of respiratory rate to breath hold time as expressed in the empirical formula (RR-(BH/5)). The QRG is set up with six columns, each of which represents a different pattern of Acidosis or Alkalosis.

Simply carry the three numbers of the 3-Point Quick-Scan over to your QRG page and see if they give you a match in one or more of the six columns. For a match, you are looking for two out of three of those tests to be positive. If you do not have at least two out of three positives in one or more columns then you are finished with your analysis of Acid/Alkaline Imbalance in this patient – you are certain there is none.

If you do get two or more matches in one or more columns, then proceed in your analysis with a consideration of the adjusted urine pH and the adjusted saliva pH. Pull the adjusted urine pH and the adjusted saliva pH off the patient’s Test Results Form and bring them over to your QRG to any of the columns which have qualified for further consideration. If you do not have a perfect pH match with both pH’s to one of those columns then you are finished – there is no Acid or Alkaline Imbalance. If you do have a perfect pH match in one or more columns then you must go further to consider the final few tests for confirmation. Look at the remaining two tests in any column that you are considering, and if you find one more positive test then you have confirmation of that pattern. Without one more additional positive, your patient does not have an Acid or Alkaline Imbalance.

One detail to remember considering this analysis regards a Respiratory Acidosis. The respiratory rate for a Respiratory Acidosis is considered positive if it is either 19+ or 13-. But, the 13- parameter only indicates a Respiratory Acidosis in those patients who have blood pressure that is somewhat above normal. In other words, if the patient’s blood pressure is normal to low, then the only positive respiratory rate indicating the possibility of a Respiratory Acidosis is a respiratory rate that is 19+.
As stated earlier in this chapter:

IF YOUR PATIENT'S RESPIRATORY RATE AND BREATH HOLD TIME ARE NORMAL, YOUR PATIENT EITHER HAS NO ACID/ALKALINE IMBALANCE, OR, AN ACID/ALKALINE IMBALANCE EXISTS, BUT IS BEING HIDDEN BY ANOTHER IMBALANCE WHICH IS PRIMARY AND MUST BE CORRECTED FIRST, BEFORE THE ACID/ALKALINE IMBALANCE IS CLINICALLY SIGNIFICANT.

Read that last paragraph again and memorize it. Do not chase abnormal urine or saliva pH in patients when their respiratory rate and breath hold time are normal. To do so only makes a mess of that patient's biochemistry. No matter how extreme a patient's urine and saliva pH's may be, those abnormal pH's are not associated with an Acid/Alkaline Imbalance unless the respiratory rate and/or the breath hold are outside normal limits.

With the NUTRI-SPEC system you have perhaps the first and only means of evaluating and treating the important Acid/Alkaline Balance.