CHAPTER 15

ACID/ALKALINE BALANCE EXPLAINED

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) Electrolyte/Water Imbalance
2) Anaerobic/Dysaerobic Imbalance
3) Glucogenic/Ketogenic Imbalance
4) Sympathetic/Parasympathetic Imbalance
5) Endocrine Insufficiencies
    a) Kidney
    b) Adrenal Insufficiency or Stress
    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 emergency status; 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.

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 upon Nutri-Spec Metabolic Balance Analysis 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 your Unified Acid/Alkaline Imbalance Analysis, 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 Vital 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.

Actually, your greatest influence on Acid/Alkaline Imbalance is achieved with your BALANCING PROCEDURE. That procedure, with which you begin care of the majority of your Nutri-Spec patients, directly affects pH at the tissue level of biological organization, and secondarily at the cellular and systemic level. The extraordinary efficacy of your BALANCING PROCEDURE results as not only is tissue pH balanced, but it is achieved simultaneously with restoring selective membrane permeability, and with enhancing cellular energetics. (Keep in mind also that all the dualistic symptoms such as pain, vertigo, itchiness, histamine reactions, upper GI function, lower GI function, high cholesterol or low cholesterol, and allergies are manifest at the tissue level.)

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 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 ventilatory 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 ventilatory 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 Electrolyte/Water Imbalance

Acid/Alkaline and Electrolyte/Water Imbalance are related to the extent that any type of Acidosis involves a tendency to dehydration, and any type of Alkalosis may involve either a decrease or increase in extracellular fluid volume. Any Imbalance, therefore, requires either an increase or decrease 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 (glycolysis). 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 resulting in excess conjugated Fatty Acids, 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 energetics 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 vasoconstriction. Both 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.

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.

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 A**cidosis** 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 **Nutri-Spec test result 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 be discussed below, giving a brief summary of the physiology associated with each.

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 easily understood 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?

   Ventilatory 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 Breath 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 Breath 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 ventilatory 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.

Your Nutri-Spec Metabolic Imbalance Analysis puts a major emphasis on the contribution of Metabolically produced carbon dioxide, with the Breath Rate response either increasing or decreasing to control CO_2 as a major factor.

Acid/Alkaline Imbalance and Respiratory Control of Carbon Dioxide and Bicarbonate

Day and night there is continual production of acid by the body's metabolic processes. To maintain balance, these acids need to be excreted or metabolized. The various acids produced by the body are classified as respiratory (or volatile) acids and as metabolic (or fixed) acids. The respiratory acid is more correctly called carbonic acid (H2CO3), but the term "respiratory acid" is conventionally termed carbon dioxide. (But CO2 itself is not an acid in the Bronstead-Lowrey system as it does not contain hydrogens, so it cannot be a proton donor. However, CO2 can instead be thought of as representing a potential to create an equivalent amount of carbonic acid.)

Carbon dioxide is the end product of complete oxidation of carbohydrates and fatty acids via the CAC or the phosphogluconate pathways of energy production. The amount of CO2 (respiratory acid) produced each day is huge compared to the production of fixed acids. Basal CO2 production is typically quoted at 12,000-13,000 mmols/day.

Venous pH is determined almost entirely by the amount of CO2 in the blood --- in accord with the Henderson-Hessalbalch equation:

- pH = HCO3-/CO2

The sole source of carbon dioxide is the CO2 formed in the Krebs Cycle or the phosphogluconate pathway (none of the other energetic pathways produce CO2 --- neither glycolysis, nor beta-oxidation, nor the Cori Cycle).

Alkaline Imbalance = Insufficient Metabolic CO2 production = increased HCO3-/CO2 = increased pH via decreased CO2 production? Ketogenic Imbalance? Anaerobic Imbalance. [Likely Ketogenic, Anaerobic or Glucogenic Imbalance, or, hypothyroid]

--- Or, Alkaline Imbalance = Respiratory Alkalosis = increased CO2 loss via Sympathetic or Respiratory Alkalosis Imbalances with hyperventilation.

According to the law of electroneutrality, if HCO3- is low, then either some other anion(s) must be high, or some cation(s) must be low. For example: in a hyperchloremic acidosis, HCO3- is low, and Cl- is high.

----- If HCO3- is high, then either some other anion(s) must be low, or some cation(s) must be high. For example: in a hypochloremic alkalosis, Cl- will be low relative to HCO3-.

In a Potassium Depletion Alkalosis, the cation K+ will be low, but that imbalance caused by the low K+ and high HCO3- will be corrected by high Na+ and low Cl-.

If [HCO3-] is high, then there is either a Metabolic Alkalosis, or, a Respiratory Acidosis with compensating Metabolic Alkalosis.

If [HCO3-] is low, then there is either a Metabolic Acidosis, or, a Respiratory Alkalosis with a renal compensatory Metabolic Acidosis.

Carbon dioxide is produced in huge quantities in cell mitochondria, and an efficient system exists for its removal. Arterial pCO2 is of critical importance for intracellular acid-base balance because of both its potential to change rapidly and its effectiveness in altering intracellular [H+].

Carbon dioxide crosses cell membranes easily. A change in respiratory ventilation affects the arterial pCO2 level and the intracellular pCO2 throughout the body simultaneously.

Changes in pCO2 will alter [H+] in all fluid compartments rapidly, and equally.

Changes in pCO2 cannot be used to produce differences in [H+] in fluids on opposite sides of a membrane.

So --- hyperventilation that induces a Respiratory Alkalosis alkalizes the blood, the interstitial fluid, and the cytoplasm simultaneously; hypoventilation that causes a Respiratory Acidosis lowers the pH of the blood, tissues, and cells at once.

$$CO2 + H2O < > H2CO3 < > HCO3- + H+$$

Considering the above reaction, it is seen that CO2 is produced metabolically and expired by the lungs. The amount of C02 gas dissolved in the blood is expressed as the partial pressure of CO2 (pCO2). [This reaction occurs predominantly in RBCs where there is an abundance of carbonic anhydrase enzyme.]

The Henderson-Hasselbalch Equation expresses blood pH in terms of pCO2 and [HCO3-] :

$$pH = 6.1 + \log HCO3-/pCO2$$

A simplification of the Henderson-Hasselbalch Equation serving as a convenient model for clinical use is:

$H+ = pCO2/[HCO3-]$ or, $pH = [HCO3-]/pCO2$

Normal values at sea level:

Blood pH = 7.35 – 7.45

[HCO3-] = 24-28 (women one lower than men)

pCO2 = 35-48

It is the **ratio** of [HCO3-]/pCO2 that determines blood pH, NOT their concentrations. At normal pH of 7.4, the ratio is 20:1. In other words, the HCO3- concentration is 20 times the CO2 concentration. The CO2 concentration is 1,000 times the H2CO3 concentration. So, the HCO3- concentration is 20,000 times the H2CO3 concentration.

An increase or decrease in metabolic rate results in a parallel increase or decrease in pCO2, then of course, an increase or decrease in H+. But --- an increase in metabolic rate increases CO2 (and H+) more than a decrease in metabolic rate decreases CO2 (and H+).

An increase or decrease in blood flow rate to a tissue results in a parallel increase or decrease in pCO2 of the interstitial fluid and venous blood, with a corresponding increase or decrease in H+.

Any increase in respiratory ventilation (increased respiratory rate and/or depth) results in a decrease in pCO2, and a corresponding increase in pH. Any decrease in respiratory ventilation (decreased respiratory rate and/or depth) results in an increased pCO2 and a corresponding decrease in pH.

Increased respiratory ventilation causes a decrease in pCO2 = the primary cause of a Respiratory Alkalosis.

An increased respiratory ventilation causes a decrease in pCO2 = the respiratory compensation for a Metabolic Acidosis.

A decrease in respiratory ventilation causes an increase in pCO2 = the primary cause of a Respiratory Acidosis (respiratory rate in particular, will be increased because the volume/depth will be decreased due to pulmonary pathology).

A decrease in respiratory ventilation causes an increase in pCO2 = the respiratory compensation for a Metabolic Alkalosis.

Clinical note:

Breath Hold (voluntary hypoventilation): the breath can be held until CO2 increases and H+ increases enough to decrease the venous blood pH to 7.0.

Both hypercapnia and hypoxia, independently and together, increase breathing frequency more than they increase tidal volume.

Voluntary hyperventilation: hyperventilation can achieve a decreased CO2 and H+ to a venous blood pH of 7.8.

Respiratory regulation of Acid/Alkaline Balance: Respiratory ventilatory compensation has 1 to 2 times the buffering capacity of all the chemical buffer systems combined.

An increase in metabolically produced CO2 (or upon Breath Hold) = increased H+ (Acidosis) = increased ventilation = increased CO2 expiration = decreases pH toward normal.

A decrease in metabolically produced CO2 = decreased H+ (Alkalosis) = decreased ventilation = CO2 retained = increases pH toward normal.

Increased Breath Rate for any reason = decreased CO2 and an increase in pH.

Decreased Breath Rate for any reason = increased CO2 and a decrease in pH.

Any increase in pH (decreased H+) for any reason = a decrease in Breath Rate and an increase in CO2 as compensation.

Any decrease in pH (increased H+) for any reason = an increase in Breath Rate and a decrease in CO2 as compensation.

Respiratory ventilation can be decreased to 0 (holding the breath) or increased by a factor of 15 (hyperventilating to the maximum).

Decreasing pH from 7.4 to 7.1 triples respiratory ventilation.

Increasing pH from 7.4 to 7.6 decreases respiratory ventilation to 70% of normal.

The respiratory response involves an adjustment of the denominator of the H-H equation, pCO2, by alterations in ventilation. Adjustment of pH by respiratory means is relatively rapid (minutes to hours).

An increased CO2 excretion due to increased ventilation will result in 1 of 3 Acid/Alkaline outcomes:

a) correction of a Respiratory Acidosis
b) creation of a Respiratory Alkalosis
c) compensation for a Metabolic Acidosis

Which of these 3 circumstances is present cannot be deduced merely from observation of the presence of hyperventilation in a patient.

This respiratory response (Breath Rate) is particularly useful physiologically because of its effect on intracellular pH as well as extracellular pH. CO2 crosses cell membranes easily, so changes in pCO2 affect intracellular pH rapidly and in a predictable direction.

This system must be able to respond quickly and to have a high capacity because of the huge amounts of respiratory acid to be excreted.

Respiratory rate and depth are stimulated by epinephrine (Sympathetic) = causes a blow off of CO2 = Respiratory Alkalosis and decreased H+.

Decreased metabolic rate = small decrease in CO2 = small decrease in H+ and decreased respiratory rate and depth.

If an increased metabolic rate is accompanied by increased epinephrine (Sympathetic) or hyperthyroid, then there is an extreme increase in Breath Rate and depth = to such an extent that H+ and CO2 will be normal, since all the excess metabolically produced CO2 will be blown off.

If a decreased metabolic rate is accompanied by increased epinephrine (Sympathetic) (in compensation for Electrolyte Insufficiency &/or Anaerobic &/or Glucogenic Imbalances, or, in association with a Respiratory Alkalosis), then the respiratory rate and depth may be normal, but there will be decreased CO2 and H+. (= Hypocapnic Alkalosis)

A Sympathetic Imbalance accompanied by decreased metabolically produced CO2 = Breath Rate normal with Pulse Rate either increased or decreased, depending on Electrolyte Stress Insufficiency, or Glucogenic/Ketogenic, or Anaerobic/Dysaerobic Imbalances.

Normal and at rest: inspiration = 2 seconds, and expiration = 3 seconds = 5 seconds = 12/minute. An inspiration time greater than or equal to the exhalation time indicates Sympathetic Imbalance. That Sympathetic tendency is easily detected when there is no delay at the end of exhalation before the next inhalation.

Respiratory Acidosis due to lung pathology = pathology such as COPD is the only time O2 drives respiratory rate and depth as much as pCO2 and H+. --- because CO2 and H+ accumulate in the blood (since CO2 cannot be blown off).

Hyperventilation Syndrome = triggered by anxiety, nausea and vomiting, fever. Symptoms = dyspnea and numbness --- HVS patients report frequent sighing and air hunger when in remission.

Respiratory rate is driven by both elevated H+ and elevated CO2 (and by emotion, epinephrine, elevated body temperature, and thyroid).

Chemical Regulation of Ventilation has 2 control centers:

- The Respiratory Center in the medulla oblongata of the brain

- Chemoreceptors in the Aortic Arch and in the Carotid Bodies

- CO2 is sensed in central and peripheral neurons. Changes in CO2 and H+ are sensed in chemosensitive neurons in the carotid body and in the hind brain. Whether CO2 or the pH are preferentially sensed is unclear, but the ventilatory response to hypercapnic acidosis exceeds that of an equivalent degree of Metabolic Acidosis, suggesting specific CO2 sensing.

The oxygen content of the blood (pO2) is the least important factor affecting the respiratory control centers.

The pH, as defined by the Henderson-Hesselbach equation, has a large effect on respiratory ventilation. The pH ($H^+ = CO_2/HCO_3^-$) has a huge impact on the Respiratory Center of the brain.

Any decrease in pH (increase in H^+, increase in CO_2, or decrease in HCO_3^-) has an immediate strong influence on increasing Breath Rate.

Any increase in pH (decrease in H^+, decrease in CO_2, or increase in HCO_3^-) has an immediate and strong influence on decreasing respiratory ventilation.

However, when the increase or decrease in H^+ is due to acid or alkali other than the relationship between CO_2 and HCO_3^-, then the respiratory ventilation changes are delayed by the cerebrospinal fluid response to the change in H^+ concentration.

The body can tolerate higher pCO_2 during Breath Holds performed at larger lung volumes. In other words, the deeper the inspiration, the higher the level of pCO_2 can be achieved before breath hold must be terminated. That is why Nutri-Spec uses the Exhalation Breath Hold to get a more accurate assessment of CO_2.

HCO_3^- is controlled by the kidneys long-term, and by the liver short-term:

While the pCO_2 component of the Henderson-Hesselbach equation is controlled by a balance between metabolic formation of CO_2 and pulmonary expiration of CO_2, the HCO_3^- component of acid/alkaline balance is controlled by the kidneys, and the liver.

Obviously, for Acid/Alkaline Balance, the amount of acid excreted per day must equal the amount produced per day.

The routes of excretion are the lungs for CO_2 and the kidneys for the fixed acids.

Each molecule of metabolically produced CO_2 excreted via the lungs results from the RBC reaction of 1 molecule of bicarbonate with 1 molecule of H^+. The H^+ remains in the body as H_2O.

$$HCO_3^- + H^+ \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O$$

Under non-physiological conditions, when acids such as ketone or lactic acids are added to the blood, their H^+ goes 2 ways: some is buffered by combining with the blood buffers (see below) and the rest combines with HCO_3^- to form H_2CO_3. The H_2CO_3 is immediately dehydrated to form CO_2. The CO_2 is expired by the lungs.

Thus, CO_2 is produced by HCO_3^- buffering fixed acids --- which adds CO_2 to CO_2 produced metabolically.

Whether due to the addition of acids as in diabetes or starvation, or to the loss of alkali from the gut, the addition of Fixed Acid causes H_2CO_3 to be removed (to maintain pH), which takes HCO_3^- out of the blood by the same number of millimoles as acid added.

The kidneys eliminate excess H+ by secreting H+ in exchange for NaHCO3 = increased [HCO3-] = increased pH.

The kidneys retain H+ in compensation for an Alkalosis by decreasing H+ secretion. There is no exchange of H+ for HCO3- $\Rightarrow$ HCO3- is excreted in an alkaline urine = decreased [HCO3-] = decreased pH.

The major buffer system in the blood and interstitial fluid is the CO2-HCO3- buffer system. It is responsible for about 80% of extracellular buffering. It is the most important ECF buffer for metabolic acids, but it cannot buffer respiratory acid-base disorders.

The components are easily measured and related to each other by the H-H equation: pH = HCO3-/pCO2.

Clinical Note: Low Body Temperature tends to be associated with an Alkalosis; high Body Temperature is often associated with an Acidosis.

The Krebs Cycle produces CO_2 , but – the Krebs Cycle is limited by the unavailability of CO_2 during stress/fatigue.

Production of CO2 requires adequate thyroid function.

CO_2 is low in Ketogenic & Anaerobic Imbalance & Respiratory Alkalosis

Ketogenic = insufficient production

Anaerobic = insufficient production

Respiratory Alkalosis = CO_2 blown off

Respiratory Alkalosis Test Pattern & Hypothyroidism give similar patterns, except Breath Rate is generally high but sometimes low in a Respiratory Alkalosis, but always low in Hypothyroidism.

Anaerobic Test pattern with increased SpH is often associated with Anaerobic + Hypothyroidism.

Anaerobic SpH is determined by: Low CO_2 = increased SpH, and High Lactic Acid = decreased SpH.

Another consideration in Acid/Alkaline Balance is the Urea Cycle:

CO_2 is the main physiological factor in elimination of ammonia (NH3), since CO_2 + NH3 combine to form urea in the Urea Cycle.

Anaerobic urine = increased NH3 (since there is insufficient CO_2 to convert NH3 to urea), while balanced urine = mostly urea, with very little NH3 or uric acid

- in a balanced urine nitrogen is eliminated mainly as urea
- in an Anaerobic urine nitrogen is eliminated predominantly as ammonia
- in a Dysaerobic urine nitrogen is eliminated mainly as uric acid

Hypocapnia (as in Respiratory Alkalosis or deficient metabolically-produced CO_2) --- increases Pulse Rate by 4 beats per minute without changing blood pressure. Hypocapnia causes frequent sigh breaths.

Hyperventilation = decreased CO2 = orthostatic increased Pulse Rate and decreased pulse pressure, both of which are reversible by CO2 rebreathing.

Hypocapnia is 7 times more common in Chronic Fatigue Syndrome than in normals, but, the hypocapnia is <u>not</u> necessarily accompanied by orthostatic tachycardia nor orthostatic hypotension.

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 (H_2CO_3), 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 excretion 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 (and to a certain extent, cortisol) 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.

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.

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 CO_2) 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, 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 magnesium) 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 sodium. Perhaps the most common cause here is Cortisol Stress. The most extreme Cortisol-associated Potassium Depletion Alkalosis is Cushing's Syndrome. But many individuals respond to physical or mental overload with Cortisol Stress and will show some degree of Alkalosis.

   The mechanism of cortisol-induced Potassium Depletion Alkalosis results from the high cortisol over-stimulating mineralocorticoid activation in the kidneys. Cortisol activates mineralocorticoid receptors just as strongly as does aldosterone. But normally, a renal enzyme, HSD2, converts cortisol into inactive cortisone to prevent mineralocorticoid receptor activation.

   But when the cortisol level is high, the HSD2 enzyme is overwhelmed. Cortisol then binds to mineralocorticoid receptors, causing the kidneys to reabsorb sodium and water, while excreting potassium and hydrogen ions. The loss of hydrogen creates a Metabolic Alkalosis, and the resulting low serum potassium drives up the blood bicarbonate, compounding the Metabolic Alkalosis. The cortisol-driven Potassium Depletion Alkalosis can cause fluid retention and/or hypertension.

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 may be (temporarily?) 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 always parallel the systemic pH.

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.

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 CO_2 as possible, thus lowering carbonic acid. Decreasing the CO_2 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 H20

2) Deep vomiting = loss of bicarbonate, sodium and other alkali, and H20

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 (Dysaerobic)

    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 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 (although, these are also symptoms of Sympathetic Imbalance). The urine output is decreased.

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 CO_2) 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.

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 CO_2. (CO_2 can **only** be decreased by hyperventilation, i.e., CO_2 being blown off faster than it is metabolically produced.) The loss of CO_2, 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 breath rate faster than it increases the pulse rate)

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; Metabolic-Associated Fatty Liver Disease

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 CO_2. 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 CO_2, 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.

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 CO_2 through the lungs. (Hypoventilation, i.e., CO_2 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 COPD patient. The inability to blow off CO_2 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 CO_2 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 CO_2 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 COPD

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 CO_2 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.

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 intracellularly, 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 a person's protein buffering system begins to lose much of its punch. The person, therefore, is much more susceptible to Acid/Alkaline Imbalances.

The other major buffering system of the body is the ventilatory control of CO_2. We can use lactic acid to illustrate how fixed acids such as lactic acid can be buffered by control of CO_2.

At rest, [Lactate] is approximately the same in fit, sedentary, and CVD subjects, but, during exercise, the increase in [Lactate] is inversely related to fitness.

During incremental work, [Lactate] increases very little until a distinct metabolic rate (VO2 Anaerobic Threshold (AT)) at which [Lactate] begins to increase sharply. At that point there is accelerated GLYCOLYSIS, producing muscle lactic acidosis. The muscle lactic acid appears to be buffered by bicarbonate (HCO3-). There is increased CO_2 output relative to O_2 uptake, as O_2 uptake (VO_2) increases linearly with work rate.

VCO_2 increases linearly at first, then increases steeply after VO_2 AT, as HCO3- buffering of lactic acid begins.

Ventilation parallels CO_2 increase.

CO2 has a much greater effect on Breath Rate than on Pulse Rate.

Buffering the H^+ of lactic acid with HCO_3^- does not increase CO_2 production in muscles (--- * There is no non-metabolic CO_2 produced in tissues. *).

The dramatic increase in VCO2 above V(TH) is due to, a) hyperventilation (not the reverse, that hyperventilation is due to an increase in CO_2), and b) the low plasma pH. Both a) and b) reduce the pool of bicarbonate in the body.

Note that CO2 elevates Breath Rate, but not Pulse Rate = decreases PR/BR Ratio.

Other examples of buffering:

Both **NaCit** and **NaCl** = improve performance of exercise of 120-240 seconds duration, but not 10-30 seconds duration. There is increased time to exhaustion, decreased Pulse Rate increase, decreased Breath Rate increase, decreased O_2 debt, increased VCO_2, decreased blood pH decrease, increased [lactate].

Exercise with **Sodium Citrate** (alkalosis) vs NaCl control = decreased [H^+] and increased [HCO_3-], but no difference in exercise time to exhaustion, Breath Rate, Pulse Rate, VO_2, VCO_2, [Free Fatty Acids], [Lactate] --- but [glycerol] is lower. The sodium citrate may have prevented lipolysis that occurs in exercise, and, prevented the glycolysis observed in bicarbonate-induced Alkalosis.

Exercise with $CaCO_3$ (control), ammonia chloride **NH_4Cl** (acidosis), or sodium bicarbonate **$NaHCO_3$** (alkalosis):

- NH_4Cl = decreased endurance (160 seconds compared to control 270)

- $NaHCO_3$ = increased endurance (438 seconds)

- No differences in Pulse Rate or $[H^+]$

- NH_4Cl = increased Breath Rate (decreased PR/BR Ratio), decreased pH, decreased lactate, decreased plasma glycerol and FFA => inhibition of glycolysis and decreased lactate efflux from muscles (= Dysaerobic, anti-Anaerobic effect).

- $NaHCO_3$ = increased [Lactate], no change in H^+, decreased Breath Rate increase (= increased PR/BR Ratio)

POINT OF EMPHASIS:

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.