CHAPTER 12

ELECTROLYTE/WATER BALANCE EXPLAINED

Introduction (This Chapter is based largely on models proposed by Riddick (4) and Pollack (29).

General Principle: Over 200 years ago, chemists discovered that if a ball of clay is placed underwater in a glass jar, and if 2 glass tubes are extended from above the water surface into the ball, then a DC voltage through electrodes placed in the tubes would cause the water surface to be elevated in one tube and depressed in the other. This phenomenon illustrates the electric charge differential typical of colloids, such as clay. **But another colloidal suspension more relevant to our interest, is human blood, and indeed, all body fluids. Our body fluids are aqueous electronegative colloidal suspensions.**

These chemists had discovered what would later be called **Zeta Potential**. What is critical about Zeta Potential --- a Natural Law of Physical Chemistry --- is that just as it applies to inanimate matter, the Law of Physical Chemistry demonstrated by Zeta Potential applies equally well to biological fluids. Zeta Potential deals with the stability of liquid-solid systems. An unstable body fluid is "electrified" with vitality.

To illustrate the principle of Zeta Potential as it applies to human blood, interstitial fluid, and intracellular fluid, snatch a handful of the clay ball mentioned above and immerse it in water, but this time in water containing mineral salts, with concentrations of anion and cation electrolytes similar to body fluids. Will that handful of clay form an adhesive mass, or will it disperse into billions of tiny particles, each remaining separated by the repulsion of their negative charges? It depends entirely on the Zeta Potential of the system.

If the mineral salts in the water are dominated by multivalent cations (such as Calcium and Magnesium, but particularly toxic cations such as …

- aluminum, or,
- multivalent cations produced by microbial pathogens, or,
- endotoxins from unhealthy microbiota, or,
- environmental toxins inhaled or ingested, or,
- the end-products of unhealthy metabolism (Sympathetic/Parasympathetic, Anaerobic/
- Dysaerobic, or Glucogenic/Ketogenic Imbalances), or,
- the end-products of Endogenous Inflam-Aging or Exogenous Inflam-Aging …

the handful of clay will agglomerate (--- the equivalent of body fluids forming a sludge, or of RBCs coalescing into Rouleaux formation).

In contrast, if the water receiving your handful of clay is dominated by multivalent anionic electrolytes such as Sulfates, Phosphates, and Citrates, the system will reach maximum dispersion, with the billions of clay particles appearing as a colloidal cloud filling the water. This is equivalent to a healthy colloidal system as maintained in high-vitality body fluids.

There is one additional environmental stressor that devastates the health of body fluids measurably, even though it has no clearly observable effects on inanimate clay. That environmental "pollutant" is distortion of the Earth's geomagnetic field, plus the presence of distorted

electromagnetic fields derived from electronic devices, or the blue spectrum light emitted from video or phone screens and from some interior lighting.

The term "blood sludge" was coined by Melvin Knisely in the early 1960s. Knisely was published extensively in the medical literature on the topic of Intravascular Coagulation. His work linked blood sludge to Cardiovascular Disease and many of his studies were published in medical journals focusing on CVD. Knisely confirmed quite nicely the work of Riddick, showing that the formation of blood sludge relates to a lowering of Zeta Potential brought about by:

1. Ingestion of a relatively large amount of 1:1 electrolytes (Sodium Chloride), a much smaller amount of 2:1 electrolytes (Magnesium or Calcium electrolytes), but only requiring a small amount of 3:1 electrolytes (such as any Aluminum compounds).

2. Acute or chronic microbial activity. Multivalent cations are produced by all bacterial, fungal, and viral infectious microorganisms. The loss of Zeta Potential can be extreme at the peak of the infection, and only gradually subsides as the victim recovers.

3. Extreme combinations of microbial secreted cations (from infection or from unhealthy gut microbiota) + ingestion of foods, supplements, or drugs with multivalent cations, to cause extreme Zeta Potential, resulting in sludging to the point of sedimentation of both Red and White Blood Cells and other blood proteins and lipids.

4. Extreme failure of colloidal electronegativity, as Zeta Potential is decreased by extreme infection, compounded by administration of cationic drugs. It is worth noting that the Spike Protein of COVID and the Aluminum used in all vaccines and the Mercury used in some, have an extreme sludging effect. Platelets are activated, Thrombin combines with Fibrinogen, and actual Thrombosis occurs. Micro-thrombi can thoroughly block the circulation through capillaries, thus affecting virtually any part of the body. In extreme cases, more significant Thrombocytosis occurs, and blood clots can cause life-threatening pathology.

Recall that RBCs can only pass through smaller capillaries single file, and even then, they must alter their shape to progress to the target tissue. That partially explains the elevated blood pressure that can ultimately result from the loss of Zeta Potential, as the heart is forced to pump harder and harder to maintain circulation.

These same forces that devastate Zeta Potential also reduce Liquid Crystalline Water (LCW), the term given by Pollack and others describing the zone ("The Exclusion Zone") just within cellular membranes where high biologically active electromagnetic polarity is maintained.

So now you have tissue membranes with no protective electronegative glycocalyx, and no protective Exclusion Zone within the cell and organelle walls.

Vascular endothelial cells are particularly vulnerable. As the endothelium is stressed, inflammatory reactions ensue. In particular, activated macrophages exacerbate the inflammatory process by producing inflammatory cytokines, especially Interleukin-6. Typically, C-Reactive Protein will be elevated as a marker of this process.

As Seneff has shown, there is a dualistic balance system in both the endothelium and in the Red Blood Cells between Sulfation and Nitric Oxide. When the Nitric Oxide aspect of the balance mechanism is deranged there is the production, not of the normal vasodilatory Nitric Oxide, but inducible (stress-related) Nitric Oxide synthase that results in tissue-damaging Peroxynitrite.

The lack of **tissue sulfation** (as achieved by your Nutri-Spec BALANCING PROCEDURE) makes it impossible to preserve both the electromagnetic voltage differential between blood colloids in the bulk water of the blood, and to also maintain the intracellular LCW.

The good news is that Riddick showed how a rigid, inelastic, sclerotic endothelium can be rejuvenated to a certain extent by restoring Zeta Potential and the activation of LCW. Riddick (in cooperation with medical physicians with whom he worked) saw dramatic reductions in cardiac arrhythmias, including PVCs and Paroxysmal Tachycardia when individuals were properly treated to increase Zeta Potential.

The dispersion of particles is essential to the function of healthy fluid suspensions, which is to serve as the body's transport medium. As long as the colloid remains dispersed it effectively transports nutrients, waste products, enzymes, hormones, antibodies, to, into, and out of, cells. Furthermore, if dispersed, the various substances in transport can pass through membranes in appropriate quantities upon demand.

Two of the essential functions you will restore with NUTRI-SPEC metabolic balancing are the abilities to:

a. Maintain ideal pH at each level of biological organization.

b. Maintain ideal concentrations of biologically active (electronegative colloidal) Liquid Crystalline Water at the cellular level of biological organization.

What do we mean by the various levels of biological organization? Very briefly, we mean the hierarchy of organizational levels from the systemic level down to the tissue level, down to the cellular level, down to the nuclear level, down to the sub-nuclear level, and so on. Each of those levels of biological organization has its own fluid compartment. The systemic level has as its fluid compartment the plasma; the tissue level is associated with interstitial fluid and the lymph; the cellular level is associated with the cytoplasm.

The best way to gain an understanding of the significance of your Electrolyte Stress and Electrolyte Insufficiency Imbalances is to talk about the movement of water and electrolytes throughout these fluid compartments. Your NUTRI-SPEC protocol will normalize fluid dynamics and pH control within and between the various levels of biological organization.

Keep in mind, as you read what follows, that if your patient has lost some of the electromagnetic properties of body fluid and membrane interfaces, then that patient's battery is running down. When we call water/electrolyte balance one of the **fundamental** metabolic control systems, we mean all that is implicit in the word fundamental. When your patient's battery is low:

- **no** metabolic pathways work optimally, and
- **any** condition or disease can result.

This chapter is largely about water and electrolyte dynamics. You will want to refer to Figure 1 through much of this discussion. Look at Figure 1 and think of the human body as a series of well-mixed fluid compartments. (1) The **intra**cellular fluid is divided into three compartments:

- the red blood cells
- the capillary endothelial cells
- the parenchymal cells of the various organs

The **extra**cellular fluid compartments consist of:

- the plasma
- the interstitium
- the lymph

Under normal conditions extracellular fluid can move freely between the interstitium and the plasma, but these must be considered separate compartments since the plasma contains a higher protein (principally albumin) concentration. (2)

Keep in mind that we are presenting a model that applies to the dynamics of the whole body fluid and solutes, but the actual exchanges of water and solutes occur exclusively in the micro circulation, i.e., the capillaries and post-capillary venules of each organ.

All the membranes separating these compartments are permeable to water but have different permeability for other substances. (3) Cellular membranes are semipermeable, allowing only water and no solute to pass through them. The capillary walls that divide the plasma from the interstitium consist of two identical endothelial membranes, but which have intercellular pores. The pores are the only pathway for solute transport between the plasma and the interstitium. These pores can be visualized as an open membrane that freely passes water and sodium chloride and many other electrolytes, but almost completely restricts albumin and complex carbohydrates and most other macromolecules.

The flows of water and solute across membranes are a function of three driving forces:

- the gradient of hydrostatic pressure
- the gradient of osmotic pressure
- the concentration gradient

The positive direction of fluid and solute flow is shown by the arrows in the diagram.

There are fluid shifts into the plasma volume from the GI tract, and from RBCs, that occur across membranes. Fluid is also returned to the plasma via the lymphatic system. Fluid is eliminated from the plasma volume by the kidneys (and also by hemorrhage).

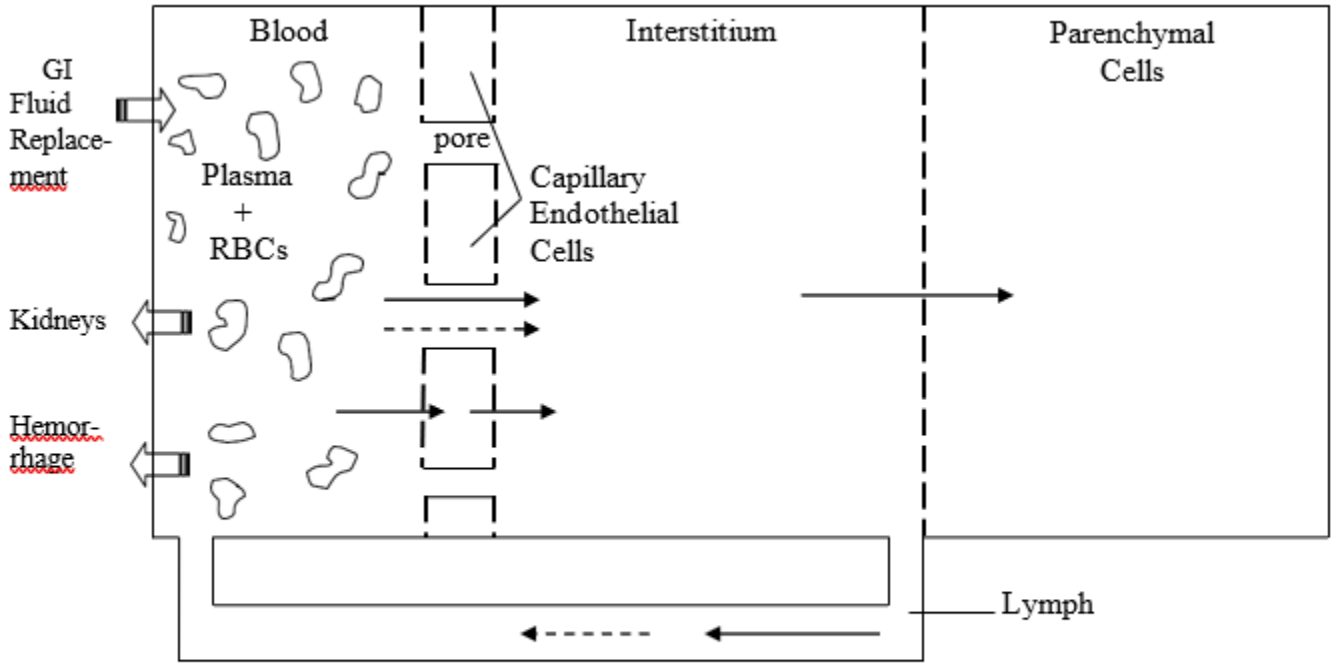
The parenchymal cell membrane does not support a difference in hydrostatic pressure, so intracellular and adjacent interstitial hydrostatic pressures are equal and water is driven only by an osmotic gradient. The exception to this is the albuminal endothelial membrane of the capillary wall, which supports a hydrostatic pressure difference between the interstitium and the plasma.

The compliance of the interstitium --- the change in interstitial volume for each unit change in interstitial pressure --- limits how much water the interstitium can take in or give up.

The oncotic pressure in the plasma, i.e., the osmotic pressure exerted by the impermeable protein (albumin) molecules, is a function of the total protein concentration. Because albumin does leak slowly across the capillary membrane, interstitial changes in osmotic pressure are directly dependent on interstitial albumin concentration.

Parenchymal Cells	= 20 liters (intracellular; cellular)
Interstitium + Lymph	= 10 liters (extracellular;
tissue) Capillary Endothelial Cells	= 1.5 liters (intracellular;
cellular) Blood = Plasma + RBCs	= 5 liters
Plasma	= (1-Hct) x 5 liters (extracellular; systemic)
RBCs	= 0.8 x Hct x 5 liters (intracellular; cellular)

water flow
solute flow



(Figure 1)

WATER/ELECTROLYTE DYNAMICS

The lymphatic system is a single flow passage that transports fluid and solute from the interstitium to the plasma, with the flow dependent only on the hydrostatic pressure in the interstitium. The lymph has the same composition as the interstitial fluid.

To maximize stability, with the colloid retaining its complete discreteness, with no agglomeration, there are two mechanisms operating in biological fluids:

a. Adsorption of an anionic electrolyte (Sulfate, Phosphate, or Citrate) on a colloid to create a strong mutual repulsion.

b. Adsorption of a strong hydrophilic protective colloid (such as Albumin or Gelatin) on the larger hydrophobic (water-resisting) colloids (blood proteins).

In a healthy colloidal suspension of body fluids, dispersion is achieved largely through the blood protein adsorption of Albumin. In turn, the stability of Albumin is controlled by the inorganic blood electrolytes (multivalent cations vs. multivalent anions). Ingesting uncooked egg whites adds to the Albumin activity in body fluids, much of the ovalbumin is absorbed intact rather than being digested into its constituent amino acids.

There are consequences to low serum Albumin. Low Albumin impedes the capacity to maintain colloidal dispersion. But additionally, we must consider the Law of Electric Neutrality; if the anion Albumin is low, then automatically some other anion must be elevated.

If that other anion is Bicarbonate, then the person, along with being deficient in Zeta Potential and LCW, will suffer from a high Bicarbonate Metabolic Alkalosis. For these individuals, electronegativity should be restored with a combination of Phosphoric Acid along with Thiosulfate. In contrast, if the anion is elevated in compensation for low Albumin is Chloride, then we have a relative Hyperchloremic Metabolic Acidosis. These individuals need supplementation with Thiosulfate accompanied by Potassium Citrate.

In a healthy body chemistry, colloidal dispersion is maintained by two criteria: type and concentration. Type relates to the valence of the cations and anions, which constitute any nutrient or toxin added to the system. These range from a ratio of 3:1 for agglutinating agents to 1:3 for dispersing agents.

But concentration is also important. To illustrate, Sodium Chloride, a 1:1 electrolyte initially disperses, but then at a somewhat higher concentration agglutinates the fluid system. Even a powerful dispersing agent such as Potassium Citrate, will actually agglomerate the system if it exists in high enough concentration.

Body fluids are poised on a delicate stability balance, just on the side of dispersion (= slight “instability”). The reason is to protect against excess blood loss through injury. The system only needs to change slightly to precipitate a clot, to prevent further blood loss. Thrombin is a powerful multivalent cation, released in response to injury, which facilitates the agglomeration/clotting mechanism. Heparin is a powerful multivalent anion employed to disperse the blood colloid and relieve intravascular agglomeration/coagulation.

Imbalances in electrolyte (salt) concentration normally do not exist in the body, since they are quickly compensated by water movement. In the presence of many of the NUTRI-SPEC Fundamental Metabolic Imbalances, however, there exist non-physiological states with severely altered plasma osmolarity that can generate enormous osmotic pressure gradients as well as non-physiological concentration gradients.

One example of a water/electrolyte imbalance is your patients who are **Hypo**volemic. These would include all your Electrolyte Insufficiency patients and many patients with other imbalances as well. These patients have low plasma volume; many of them have low interstitial fluid volume and low cellular hydration as well. Many of them, in contrast, have an excessive interstitial fluid volume, but decreased intracellular volume. Another variation on **Hypo**volemic patients are those who have low cellular volume in parenchymal cells but actually have an increased volume of the capillary endothelial cells.

How do you as a NUTRI-SPEC practitioner deal with all these many variations that can occur with a single condition – **Hypo**volemia? The answer is simple. Your Nutri-Sped testing and analysis protocol automatically sorts through all these many variations of the **Hypo**volemic theme. You will automatically be giving your patients just the supplements they need to supply electrolytes that flow, along with biologically activated water, to exactly where they need to be, while at the same time pulling excess fluid out of where it should not be.

Consider now patients who are **Hyper**volemic. These include all your Electrolyte Stress patients. Some of these patients will have low interstitial volume and low cellular volume. Many, however, will have elevated interstitial fluid volume and extreme edema. Some of these **Hyper**volemic patients will have elevated serum pH and elevated tissue pH. Others will have elevated serum pH and acid tissue pH. Still others will have the reverse – acid plasma and alkaline tissues.

How do you get just the right electrolytes in just the right quantity into the proper fluid compartment? How do you ensure that pH is normalized in each fluid compartment? How do you move the excess fluid out of the plasma and into the parenchymal cells that are starved for biologically active water? Again, your Nutri-Spec Testing sets your course to maximize your patient's functional capacity. (Or, you can prescribe a diuretic to decrease the excess plasma volume – which is about as logical and beneficial as the blood letting of days gone by.)

Consider another example. You have a large portion of your patients who are suffering the effects of the abnormal fatty acids with conjugated double bonds (the ones that most accelerate free radical oxidation damage and associated catabolic and Exogenous Inflam-Aging processes). (See Chapter 13 on ANAEROBIC/DYSAEROBIC BALANCE for a more in-depth discussion of pathological fatty acids.) These abnormal fatty acids in the parenchymal cells pull excess chlorides from the interstitium into the cytoplasm, binding the chlorides irreversibly.

The excess binding of chlorides allows excess sodium and carbonate (and eventually potassium in many cases) to accumulate at the tissue level. These sodium and potassium carbonate compounds, (particularly in the absence of sufficient chloride) leave the tissues extremely alkaline. This tissue alkalinity is associated with an exaggerated sensitivity to all the dualistic symptoms such as pain sensitivity, allergic sensitivity, vertigo, itching, etc.

Patients suffering from this biochemical abnormality include many of your Electrolyte Insufficiency patients, some of your Glucogenic patients, and almost all of your Dysaerobic

patients. Nothing other than Electro Tonic or Oxygenic D+ can reverse this destructive catabolic aging process. No one but you, as a NUTRI-SPEC practitioner, can even touch this problem.

Your primary means to address membrane permeability inadequacies, non-physiological pressure gradients, and fluid compartment pH imbalances is the NUTRI-SPEC BALANCING PROCEDURE. Beyond that clinical protocol, you have objective test procedures guiding you to the needs of each individual patient.

Let us look more closely now at your Electrolyte Stress, then your Electrolyte Insufficiency patients – the two imbalances that are most directly associated with water and electrolyte abnormalities. You will learn that your ES and EI patients are divided into sub-categories based upon their hormonal stress reaction and their pH aberrations. How do you make the differential analysis between these sub-categories within each imbalance? As you might suspect, your Nutri-Spec Testing does it for you. Read on to learn how and why.

The ES section of this chapter is largely about declining cardiovascular/renal health. You will find that the vast majority of your cardiovascular disease patients have a NUTRI-SPEC Electrolyte Stress Imbalance. Many of them will also have a concurrent Anaerobic Imbalance and/or a Sympathetic Imbalance or a Glucogenic/or Ketogenic metabolic energetics insufficiency.

Here are some amazing facts. Nearly half of all Americans die of heart attacks. Yet until about a century ago no such pathology was recognized. The first reported heart attack occurred in 1896. The condition did not even have a name. Several years later, the first write-up on heart attacks appeared in the New England Journal of Medicine. The article was devoted to this curious new and apparently rare condition that it named “myocardial infarct.” (5)

"Imagine that," the authors were saying, "We have seen several patients suffer ischemic damage to the heart, and several have actually died." They were studying a previously unheard-of disease and really did not know what to make of it.

Paul Dudley White, M.D., a famous heart specialist in the mid twentieth century said, "When I graduated from medical school in 1911, I had never heard of coronary thrombosis." (5)

We have come a long way in a hundred years. Half the population is dying from cardiovascular disease, a condition that was scarcely recognized a century ago. Just as significant (and an accurate commentary on how thoroughly the medical establishment has pulled the wool over the public's eyes), few even realize that this condition is preventable by simply deleting from our lifestyle the pernicious practices that have become the conventional way of life in the Western world in the last century.

What are these pernicious practices? Is it eating too much cholesterol and saturated fat? Is it too much "stress" in our lives? The common wisdom of our day would have us believe that these are prime culprits. But really, these are relatively insignificant factors. An honest look at epidemiological studies shows that cholesterol and saturated fat intake have not increased, but have actually decreased in the last one hundred years. And what about stress? A little more than a century ago many people had to work like slaves just to provide themselves with minimal food, clothing, shelter, and sanitation. Our standard of living has increased so dramatically since then that nothing we face remotely resembles the real stress that our ancestors faced. (Most of what we call modern "stress" is more accurately described as stress deprivation. Most people simply do not have enough constructive, satisfying work to please and ease their mind and heart.)

There are 7 changes that have occurred in the United States in the last one hundred years that are direct causative factors in the deplorable incidence of cardiovascular disease. They are:

1. The chlorination and fluoridation of our drinking water.
2. The demineralization of our drinking water.
3. The dramatic increase in consumption of sugar and other refined carbohydrates.
4. The increased consumption of polyunsaturated and hydrogenated oils.
5. The homogenization of milk.
6. The trace mineral depletion of our soil and thus our food.
7. The disturbance of the Earth's geomagnetic field and electromagnetic pollution.

These six pernicious factors are easily reversed by you and your patients. Cardiovascular disease could be eliminated in one generation if these destructive practices were avoided.

Electrolyte Load and Blood Flocculation

When under excessive electrolyte load, body fluids begin to flocculate, i.e., form a thickened sludge, with the following consequences:

A) Blood sludge is an inefficient carrier of oxygen, nutrients, waste products, enzymes, hormones, antibodies, etc.

B) Blood sludge circulates through arterioles only with great difficulty, and through capillaries not at all (RBCs must move single file through capillaries).

1) Flocculating blood increases the load on the heart.

a) Blood pressure and pulse rate must increase to meet stress demand.

2) Sludge precipitates = formation of deposits in arterioles.

3) Circulatory stasis = poor circulation.

a) Trophic changes occur in virtually all organs and tissues, resulting in a slow steady decline of health (with many and varied symptoms).

4) Thrombus and embolism formation.

a) Myocardial infarction, stroke, pulmonary embolism, etc.

5) More than half of all Americans die of cardiovascular disease associated with blood sludge accompanied by tissue pH and energetics imbalances

C) Blood sludge does not allow for efficient and timely transport through cell membranes.

Blood sludge can result from excess electrolyte load. Chronic excess electrolyte load **impairs kidney function** and leads to gradual renal debilitation. What is the major offender in excess electrolyte load? Quantitatively speaking, it is sodium chloride (NaCl), table salt.

Following are some interesting facts regarding NaCl. A natural diet free of processed foods provides a total electrolyte intake of 5-10 grams daily. A typical American diet, including a high

percentage of refined food to which salt is added, contains an electrolyte intake of 20-25 grams. The sodium/potassium (Na:K) ratio changes from less than 1:6 on a natural diet to more than 3:1 with refined food. That represents an increase in the Na:K ratio to eighteen times as high.

Electrolyte Load On The Kidneys

How does this drastic increase in electrolyte load affect the kidneys? Healthy kidneys can handle a natural electrolyte load in fifteen hours a day. Under natural conditions, therefore, the blood remains dispersed, and the kidneys get to rest many hours each day. To handle a typical excess electrolyte load, however, the kidneys must work twenty-four hours and still cannot clear the excess salts. This gradually reduces kidney efficiency. Riddick found that impaired kidney function occurs in the majority of people who have reached middle age, and many younger.

Under natural conditions of low electrolyte intake, the kidneys discharge urine with little or no concentration required. Their principal function is not to eliminate excess salts, but rather to retain minerals to prevent depletion.

As a side note on water intake: Monkeys (with essentially the same renal physiology as humans) on their natural diet, have a daily urine excretion of 80 ml/kg of body weight, while humans excrete less than 20 ml/kg of body weight. To equal the monkey's output, a 70 Kg man needs to excrete over 5 liters of urine daily. This would necessitate a more than two-fold increase in water intake.

Under heavy electrolyte load, and with average water intake, the kidneys have difficulty keeping blood, interstitial, and intracellular minerals down to within normal limits, and to do so requires extreme concentration of blood electrolytes to form urine.

The high energy gradient against which the kidneys must work under these conditions (concentrating blood electrolytes to urine electrolytes in a ratio of 1:2) causes gradual debilitation of the kidneys. The only way to compensate for this blood:urine electrolyte concentration is with a doubling of water intake. This quantity of water would effectively remove the entire increased kidney load. In other words, if we have more than doubled our salt intake by processing foods, we need to more than double our water intake to maintain the same concentration gradient for the kidneys. Have we doubled our water intake? Many of your patients have not.

The early morning urine, when there is chronic Electrolyte Stress, will show a somewhat elevated electrolyte load, but not excessively high. With each cup of water consumed before breakfast, the urine volume increases, allowing a greater excretion of electrolytes. The electrolyte load in grams/liter decreases, thus yielding a low specific gravity. It is absolutely essential that your Electrolyte Stress patients drink 2 cups of water **with dispersing agents** between arising in the morning and taking their first food or other drink.

Here is a side note, relating to the ever more popular consumption of so-called sports drinks. Studies have shown conclusively that these drinks not only do not improve sports performance, they do not even quench thirst. In truth, they contribute to dehydration. In other words, the electrolyte concentration is so high in these drinks that they cause blood to sludge, thereby decreasing oxygen delivery to the tissues, not to mention putting a strain on the cardiovascular and renal systems. To prevent the damaging effect from Electrolyte Stress, some sport drinks need to be diluted with five times their volume of water; the most popular sport drink needs to be diluted with 2 times its volume of water. Even fruit juices need to be diluted with equal quantities of water, as do carbonated soft drinks. (We have considered here only the deleterious effects of the electrolytes in these drinks. We have made no mention of the harm done by the concentrated sugars they contain.) (6)

Electrolyte Type

What else besides NaCl contributes to blood sludge? Any mineral salt increases electrolyte load to some extent. But the electrolyte sludging vs. dispersing effect depends not only on electrolyte concentration, but on electrolyte type as well. What is meant by electrolyte type? Type refers to the valences of the cation and the anion in a particular salt.

Type is defined as the ratio of the valences, cation to anion, in a particular electrolyte molecule.

Examples: $Na^+ Cl^-$ = 1:1
$Ca^{++} (Cl^-)_2$ = 2:1
$(K^+)_2 SO_4^{--}$ = 1:2
$Al^{+++} (Cl^-)_3$ = 3:1

Rules of Sludging vs. Dispersing Effect:

a) Excess quantity (Electrolyte Stress) of any electrolyte contributes to sludge.

b) Cations have a sludging effect.

c) The higher the cation valence, the greater its sludging effect.

d) Anions have a dispersing effect.

e) The higher the anion valence, the greater its dispersing effect.

f) Electrolytes with the worst sludging effect are those combining a multivalent cation with a monovalent anion, e.g., aluminum chloride, Al(Cl)3 is a 3:1 electrolyte = extreme sludging effect.

g) Electrolytes with the best dispersing properties are those combining a monovalent cation with a multivalent anion; e.g., potassium citrate, K3 (C6 H5 07) x H2O is a 1:3 electrolyte = good dispersing (anti-sludge) qualities.

It is interesting to note the relation between the colloidal state of the blood and the clotting mechanism. The blood colloid is naturally maintained in a dispersed state, but just on the verge of beginning to flocculate. This is so that if blood is shed through injury, not much change in colloid is required to make it clot. Clotting is associated with release and activation of prothrombin-thrombin (produced in the liver). Thrombin is simply a **cationic** polyelectrolyte. Heparin (produced in the liver), on the other hand, is simply an **anionic** polyelectrolyte. It is used medically to relieve intravascular coagulation. (An excessive dosage of heparin will actually cause hemorrhage.)

When dispersing agents such as 1:2 or 1:3 electrolytes are consumed in the diet, the kidneys tend to retain these and excrete an equivalent amount of 1:1 electrolytes. This exchange is the actual mechanism for naturally maintaining electronegativity of the blood colloid and relieving intravascular coagulation. The kidneys are able to selectively utilize the 1:2 and 1:3 electrolytes if they are available. Typically, however, as we have already indicated, dietary intake is unnaturally low in these, and high in 1:1 and 2:1 electrolytes.

Water treatment facilities further aggravate this problem. Electrolytes containing multivalent cations are used to flocculate impurities. Residues of these electrolytes appear in tap water and have the same flocculating (sludging) effect on blood.

Aluminum is a trivalent cation. It thus has a devastating effect on body fluids. Dangerous quantities of aluminum may be consumed from:

- public water supplies
- aluminum cookware
- canned fruits and vegetables
- antacids
- baking powder
- aluminum foil and aluminum containers
- deodorants
- commercially refined salt may contain alumino-calcium silicate and sodium alumino-silicate.

Other Factors Influencing Electrolyte Stress

A) Alcohol aggravates blood sludge.

B) Elevated blood lipids, generally derived from dietary sugar and polyunsaturated oils, are a complicating factor in the formation and precipitation of blood sludge when they exceed their critical micelle concentration and begin to precipitate. (Elevated blood lipids are not the result of excess dietary fat.) (See Chapters on Anaerobic/ Dysaerobic Balance, Glucogenic/ Ketogenic Balance, and the NUTRI-SPEC Fundamental Diet.)

Lipid-related sludge involves the release of a cationic polyelectrolyte by the platelets. This combines with fibrinogen to form a fibrinous sludge which accumulates on the vascular walls.

Excess fatty acids cause RBC sludge formation via their effect on cell membranes. (See Dysaerobic Imbalance Chapter).

C) Sludging vs. Dispersion is also pH-dependent.

The ideal pH for blood is 7.4, which is very close to the point of maximum

Either increased acidity or alkalinity will aggravate blood sludge.

D) Intensive and prolonged microbial activity has the same blood sludging effect as excessive electrolytes.

A cationic polyelectrolyte is produced by all infectious and many non-infectious microorganisms, including viruses.

The sludge diminishes as the infection subsides.

E) Urinary Surface Tension as an indicator of blood sludge: (See the Chapter on Anaerobic/Dysaerobic Balance).

Elevated Surface Tension = deficiency of surface-active substances that disperse.

Sludge associated with electrolyte load and/or cholesterol.

Low Surface Tension = RBC sludge (rouleaux formation) due to excess polyunsaturated fatty acids

Protective Factors

Fortunately, nature gives us some degree of protection from excess electrolyte load and from the sludging effects of multivalent cations. First, there is the blood protein **albumin**, which acts as a dispersing agent or anti-sludge agent. The blood colloid remains dispersed by adsorption of albumin. Albumin acts as a strongly hydrated hydrophilic colloid adsorbed on a larger hydrophobic colloid. The affinity for water exceeds the mutual attraction of adjacent colloid particles.

The protective action of albumin gives the blood colloid tremendous resistance to the sludging effect of excessive electrolyte load. This is the only reason why ingestion of large quantities of particularly harmful electrolytes (e.g., aluminum hydroxide antacids) is not instantly fatal. This may be the principal function of serum albumin. (4)

Glycosaminoglycans (GAGs) have many important functions in human physiology. Among the most important is the protection of the cardiovascular system. Glycosaminoglycans help maintain arterial elasticity; they also possess lipid-clearing activity. Most important, however, is their effect on the electronegative colloidal property of body fluids. Photomicrographs taken within arteries show that glycosaminoglycans work by inducing electronegative charges on platelets. Electronegativity prevents blood flocculation as the sticky adhesion associated with thrombus formation is prevented. Chondroitin sulfate is an effective GAG.

There are other natural dispersing agents besides albumin and GAGs. These are the multivalent **anions**. The higher the negative valence of the anion the greater its anti-sludge effect. A good example is the citrate ion with a valence of -3. Unprocessed foods (especially vegetables) and natural water are abundant in the multivalent anions.

Electrolyte Stress and the Heart

Having read this far in Chapter 12, you can easily see the progression of events that lead to heart disease, and also place a tremendous load on the kidneys, which leads to renal debilitation over a period of years.

If the patient has excess renal renin production associated with a sympathetic tendency, there will also be vasoconstriction, which further compromises the circulation. Thrombus and embolism formation is steadily increasing in likelihood, and heart attack, stroke or pulmonary embolism are inevitable. It is now a matter of when, not if.

If you consider the unhealthy drinking water, the sugar and oil consumption, the use of pasteurized-homogenized milk, and the relentless assault from electromagnetic fields, you understand the factors accelerating cardiovascular aging/degeneration. They all do damage to the vasculature and to the myocardium. The communication lines to the baroreceptors of the aorta and carotids and chemoreceptors in the brain stem are disrupted. A new "abnormal normal" set point for blood pressure, pulse rate, and breath rate is established. Oxidative and reductive stress to the vascular intima, to the platelets, to the blood lipids, to homocysteine, and to the myocardial cells ensues, which completes the clinical picture of cardiovascular disease (CVD).

Over the years the blood pressure and pulse rate must steadily increase in ES patients to meet the additional stress demand placed on the heart. When you see patients with high blood pressure, that is your first clue that the Electrolyte Stress Imbalance is firmly established. With NUTRI-SPEC you can have a tremendous impact on the health span of these patients. You can increase the vitality of all the tissues suffering trophic changes; you can add quality years to their life. The key to helping these patients is to correct the Electrolyte Stress by supplementing with dispersing agents, plus supplements to feed cardiovascular energetics.

It is also vitally important that you correct the other NUTRI-SPEC imbalances occurring concurrently with the Electrolyte Stress. For example, in addition to the dispersing agents, an Electrolyte Stress patient with an Anaerobic Imbalance also desperately needs the magnesium, the negative valence sulfur, the chondroitin, and the trace minerals in Oxygenic A and Oxy Tonic. The Electrolyte Stress patient who also has a Sympathetic Imbalance must have, in addition to the dispersing agents, the magnesium and potassium orotate the arginine, and the niacin in Complex S. The Electrolyte Stress patients who have either a Dysaerobic or a Glucogenic Imbalance will likely have elevated serum cholesterol, yet paradoxically need to increase their dietary intake of foods high in cholesterol such as meat and eggs in order to improve their body's ability to metabolize cholesterol.

Without the slightest bit of exaggeration or sensationalism, we can unhesitatingly claim that you will achieve **LIVE STRONGER LONGER** with your NUTRI-SPEC protocol for Electrolyte Stress Imbalance. As you have just learned, CVD kills 50% of all Americans. And as you also know, nearly every person who suffers from CVD has a NUTRI-SPEC Electrolyte Stress Imbalance. Since half the people you know – half your patients, half your friends, and half your family – are going to die of CVD, do you not fully appreciate how significant it is that many hundreds of NUTRI-SPEC patients across the country have had this Electrolyte Stress/CVD complex slowed at the very least?

How many people do you know who have had heart attacks, who are plagued with high blood pressure, who have high triglycerides and low HDL cholesterol, or who suffer from angina – who desperately need your help? Remember, despite the "best" of medical science, the mortality from cardiovascular disease still lingers near the 50% mark.

Since virtually every person who suffers from CVD has a NUTRI-SPEC Electrolyte Stress Imbalance, ask yourself, "How can I find and fix this fundamental imbalance that is insidiously draining the very life out of so many people I care about?"

Clinical Findings in Electrolyte Stress

Your Nutri-Spec analysis will tell you **in seconds** to what extent a patient is suffering the cardiovascular ramifications of fluid and electrolyte dynamics that are out of control.

It is by addressing the **causes** of CVD that your ES supplements work wonders on the health of the heart and vascular system. Each ES patient's individualized supplement regimen:

- inhibits oxidative and reductive stress (aging processes) within the arterial wall
- enhances hepatic metabolism of both cholesterol and triglycerides
- supports arterial elasticity
- improves the colloidal dispersion of the blood and interstitium
- facilitates normal myocardial metabolism
- increases RNA and DNA synthesis in the heart
- facilitates myocardial strength and dilates coronary arteries
- maintains platelet dispersion, protecting from platelet aggregation
- improves exercise tolerance
- assures healthier cardiac rhythm

Please appreciate that when you are working with a CVD patient with an Electrolyte Stress Imbalance you are effecting these changes **not** by drugging and blocking physiological activity as is done by the pharmacological approach to CVD. Rather, you are restoring a healthy blood colloid and enhancing cellular energetics of the endothelium and myocardium.

Electrolyte Stress Treatment Protocol

Never forget – this imbalance involves the destruction of the electronegative colloidal properties of the body fluids. Once the polarity of these body fluids begins to drop, you get a vicious cycle. The loss of electronegativity accelerates the rate of oxidative and reductive tissue destruction – and the tissue destruction further decreases the electronegativity.

Above, the concept was introduced of normal vs. abnormal extracellular and intracellular fluid composition and movement. Much of what goes wrong in ES/CVD patients involves either abnormal solutes and abnormal pH of one of the body fluid compartments, or, involves the inability to move biologically active water and/or electrolytes **into** the proper body fluid compartment.

Another ES/CVD problem is the flocculation of the body fluids. Red blood cells begin to clump and platelets begin to aggregate. These changes are largely associated with the loss of normal electronegative charge on the platelets and the RBCs. In restoring this electronegative colloid we are lowering the tendency to develop thrombosis.

Our ability to lower cholesterol and triglycerides is also tied in with our ability to maintain water and electrolyte dynamics. It is only when damage to the arterial intima creates a loss of tissue membrane polarity that cholesterol, calcium and the other components of atherosclerotic plaquing are pulled into the lesion.

With your Nutri-Spec Testing protocol, you can not only identify the existence of ES/CVD in a matter of seconds, but you can also identify the specific causative factors in each individual patient.

Look at the Table on page 12-22. The Table breaks down your ES patients into two broad sub-categories. Then, you see some variations of the main theme within each of the two columns.

The principal sub-categorization of ES patients separates them into those with either excess renin activity or low renin activity. Renin is the hormone associated with the kidneys relating to sodium excretion and retention as well as arterial constriction and dilation. The column in the Table for the high renin activity patients is labeled "ES/R+"; the column for the low renin patients is headed "ES/R-."

The first distinctions made between these two sub-categories of ES patients involve the primary stress hormones that are found to be excessive in these patients, as well as the NUTRI-SPEC metabolic imbalances that are typical of these patients.

Next, you see the plasma pH typical of each category. That is followed by a description of the fluid distribution aberrations, plus the sodium and chloride status of each category.

Then, the major mechanisms by which this category of Electrolyte Stress is effected are described. Essentially, what we have here is one category of ES patients that is extremely chloride sensitive and one category that is extremely sodium sensitive. (7,8)

Consider your ES (R+) patients. Renin is the renal hormone mediating extracellular fluid volume (plasma + lymph + interstitial), and vasoconstriction. Renin is released in response to 3 stimuli:

- decreasing nephron sodium level
- low blood volume or low blood pressure (particularly low pulse pressure) (via baroreceptors)
- Sympathetic beta-adrenergic activity

Your ES (R+) patients will have high blood pressure of the type that is generally categorized as renovascular hypertension, or malignant hypertension. If you look at the three stimuli of renin output listed above, which do you think are typical of your ES (R+) patients? Obviously, since they are hypertensive and **Hyper**volemic, the second stimulus, low blood volume and low blood pressure does not apply. The first stimulus, low nephron sodium, rarely applies. The third stimulus to renal renin output, increased beta-adrenergic activity, is present to some degree in all your ES (R+) patients. That beta adrenergic activity can be associated with a Sympathetic Imbalance; it can be secondary to a Glucogenic or Dysaerobic Imbalance; it may be a <u>localized</u> sympathetic response due to pathology in the kidney, or, decreased blood flow to the kidney as in renal artery stenosis. High renin output is also found in toxemia of pregnancy, Metabolic-Associated Fatty Liver Disease (Anaerobic/Dysaerobic or Glucogenic/Ketogenic), and in congestive heart failure.

Renin's activity is on angiotensinogen, produced in the liver, converting it into Angiotensin I. The Angiotensin I is then converted (mainly in lung capillaries by angiotensin converting enzyme (ACE)) into Angiotensin II. It is the Angiotensin II, then, that causes the ES (R+) Electrolyte Stress Imbalance. What exactly are the actions of Angiotensin II?

The most immediate effect of Angiotensin II is the stimulation of the sympathetic nervous system. This sympathetic activation is the killer. Killer? Yes, as you see, we have here a positive feedback loop – a devastating disaster in any metabolic pathway. Excess sympathetic activity causes the stimulation of renin, which in turn causes an increase in sympathetic activity, which further pumps up the renin – in a vicious cycle.

The renin-stimulated Angiotensin II creates your ES (R+) Electrolyte Stress Imbalance by several other mechanisms in addition to sympathetic stimulation. Here is a list of all the ramifications of excess Angiotensin II:

- Sympathetic Nervous System stimulation (dangerous positive feedback loop)

- Vasoconstriction. This is vasoconstriction in addition to the vasoconstriction that occurs secondary to the sympathetic stimulation. This vasoconstriction increases both the systolic and diastolic blood pressures.

- The hypothalamus is activated to increase thirst and increase the appetite for sodium (salt).

- The posterior pituitary is stimulated to release ADH/vasopressin. ADH causes the kidney to retain water and dump sodium. The water retention contributes to the **Hyper**volemia of your ES (R+) Electrolyte Stress patient.

- The sodium excretion stimulated by ADH is achieved largely by sodium-hydrogen exchange in the kidney. The loss of sodium as hydrogen ions and water are retained decreases the acidity and increases the specific gravity of the urine, while the plasma becomes more acid.

- Aldosterone (and some cortisol) is released from the adrenal cortex. The aldosterone has two effects on the kidney, one of which works in parallel with the ADH, and one of which opposes it. The aldosterone increases the retention of water, thus contributing further to the **Hyper**volemia and high blood pressure in parallel with the ADH. However, the aldosterone works against the ADH by stimulating the kidney to retain sodium (while it dumps potassium). This aldosteronism secondary to high renin is why there is often not a clear distinction in your NUTRI-SPEC analysis between ES (R+) and ES (R-) Electrolyte Stress patients. As you see on your ES (R+) and ES (R-) chart, high aldosterone and glucocorticoids is typical of your ES (R-) Electrolyte Stress patients.

Here is an important clinical note. You see that renin output from the kidney is stimulated by low sodium. This is why it is important for ES (R+) patients not to go on a severely salt-restricted diet. Long-term restriction of salt in these patients can actually produce a paradoxical rise in blood pressure, and increase vascular damage secondary to the increased stimulation of already elevated renin output.

Another clinical note: renin production is stimulated by caffeine. It is essential that your ES (R+) Electrolyte Stress patients severely limit, if not totally eliminate coffee, tea, and cola. (Related topic: Caffeine consumption also causes elevated homocysteine, an independent risk factor for CVD.)

There is another mechanism at work in your ES (R+) Electrolyte Stress patients in addition to the excess renin. Many of these patients have a chloride-sensitive hypertension. The chloride sensitivity in these patients causes vasoconstriction, increases the blood pressure, and increases the incidence of strokes. Chloride ingestion in these patients will elevate the blood levels of chloride relative to bicarbonate, contributing to Metabolic Acidosis. Supplementing these patients with potassium chloride has a paradoxical effect. Potassium generally is effective at lowering blood pressure, yet in these ES (R+) chloride-sensitive patients the pressor effects of chloride override the anti-pressor effects of potassium.

The clinical implications here can be huge. Many of your hypertensive patients have been put on diuretics that cause the loss of potassium. To compensate, they have also been given a prescription for potassium chloride to replace the lost potassium. That potassium chloride exacerbates their Acidosis, while it actually negates any effect of the diuretic on lowering the blood pressure. And, the diuretic counterproductivity exacerbates the sodium loss.

Potassium supplementation is absolutely critical for your ES (R+) Electrolyte Stress patients. As you might expect, one effective form of potassium is potassium citrate. Studies have shown that supplementation with potassium citrate will decrease the blood pressure even on a normal sodium chloride diet. The proper potassium supplementation will increase the plasma pH, increase the plasma bicarbonate, and decrease the partial pressure of CO2 – all contributing to minimizing the Acidosis, while lowering the blood pressure.

Let us turn our attention now to your ES (R-) category of Electrolyte Stress Imbalance.

How is it that these hypertensive patients are low in renin, and how is it that they are hypertensive despite the low renin? This category of Electrolyte Stress Imbalance is characterized by elevated adrenal corticoid output and sensitivity. There are excess glucocorticoids and most often also a primary excess of mineralocorticoids as well. (The aldosteronism found in many of your ES (R+) patients described above is a secondary aldosteronism.) The excess adrenal corticoids cause retention of sodium and chloride and water, along with renal loss of potassium. The excess retention of water and salt increases the blood volume and increases the blood pressure.

Recall that the renal release of renin is triggered by decreasing nephron sodium. In these patients with elevated sodium retention, there is negative feedback on renin production, which is why these hypertensive patients can have renin levels as low as 1/3 normal. Renin production is switched off by both elevated aldosterone and elevated cortisol, along with the high sodium.

Your ES (R-) patients are those with a salt-sensitive hypertension. Even the slightest salt intake exacerbates the hypertension. This high blood pressure is the type that initially responds well to diuretics. The problem is, however, that the diuretics will quickly cause a Potassium Depletion Alkalosis since these patients were low in potassium to start with, and the diuretics cause an Alkalosis as they deplete the body of potassium (and magnesium). While these patients are on a diuretic (– and we recommend getting them off the diuretic as soon as possible) they must supplement with potassium (and magnesium --- and the magnesium should be as magnesium chloride).

An important aspect of the mechanism behind the development of ES (R-) Electrolyte Stress Imbalance is a deficiency of **natriuretic peptide**. The low natriuretic peptide decreases vasodilation (increases vasoconstriction) and elevates sodium in all body fluid compartments. Natriuretic peptides are supposed to be produced in the heart, and in their absence, hearts exhibit marked hypertrophy with fibrosis. The receptors for natriuretic peptides that initiate natriuresis, diuresis, and vasodilation, are found in the kidney, the vascular tissue, and the adrenal glands. With inadequate natriuretic peptide, the heart progresses through pathological stages, including hypertrophy, left ventricular dilatation, and ultimately an increased incidence of myocardial infarct.

Relative to your ES (R+) patients, your ES (R-) Electrolyte Stress patients will show relatively higher systolic blood pressure and comparatively normal diastolic blood pressure. With the elevated systolic and relatively normal diastolic blood pressures, the pulse pressure in your ES (R-) patients tends to be much higher than in the ES (R+) patients whose systolic and diastolic blood pressures are elevated by about an equal percentage. Also, the resting pulse rate will tend to be somewhat lower (or at least less elevated) in your ES (R-) patients.

The deficiency of natriuretic peptide and the elevated adrenal corticoids do not explain the entire mechanism behind your ES (R-) Electrolyte Stress Imbalance. The third and final component of the pathological process here is an **elevated insulin** (Glucogenic/Ketogenic &/or Anaerobic &/or Parasympathetic Imbalances). Elevated insulin levels have been shown to have an anti-natriuretic action. Particularly in cases of insulin resistance, there is exacerbation of the elevated blood pressure, the renal sodium and water retention, and vascular smooth muscle cell hyperplasia. The damaging effects of high insulin are mediated via upregulation of protein kinase C. The protein kinase C directly causes vasoconstriction along with atherogenesis. A second damaging ramification of elevated insulin is that the high insulin increases the sensitivity to cortisol.

Note where restricted salt intake fits into our ES model. We have six research studies showing the complete inability to elevate hypertensive test animals' blood pressure with sodium **unless** it is accompanied by chloride. (9,10,11,12,13,14)

Other studies on water/electrolyte dynamics show that inadequate sodium intake for ES (R+) hypertensives will actually further increase renin activity and exacerbate the high blood pressure. (15)

You have only a minority of your Electrolyte Stress patients who are harmed by sodium and a substantial percentage who actually **need** sodium as long as it is not accompanied by chloride (and **is** accompanied by adequate water). About 50% of hypertensives are salt-sensitive. In other words, salt restriction will yield a 10% or more decrease in blood pressure. About 50% of hypertensives are not sodium chloride sensitive. How does this 50-50 relationship of salt-sensitive to non salt-sensitive hypertensives relate to your ES (R+) and ES (R-) categories of Electrolyte Stress Imbalance? About 1/3 of all hypertensives are low in renin – your ES (R-) category. These all need salt restriction. 50% - 33% = 17% additional hypertensives who are salt-sensitive. Where do these 17% come from? These are your ES (R+) patients who have the secondary aldosteronism.

Putting the numbers together, we see that about 2/3 of your Electrolyte Stress patients are ES (R+) category and about ¼ of those have a secondary aldosteronism that creates a clinical picture that overlaps with your ES (R-) category. From a Nutri-Spec clinical point of view, it is encouraging to know that there are studies showing that even in salt-sensitive hypertensives, ingestion of sodium chloride does not increase blood pressure when calcium, magnesium, and potassium reserves are restored. So, we have at least 50% of our Electrolyte Stress patients who never require sodium restriction and only a small minority who require salt restriction.

In terms of danger from cardiovascular disease mortality, which of your two categories, ES (R+) or ES (R-), is the more severe? Once these two pathologies have reached the point of creating moderate hypertension, they are equally deadly. However, among mild hypertensives (those with blood pressure less than or equal to 150/100), the high renin category is by far the more dangerous. In patients with elevated renin, the incidence of myocardial infarct is 2½ times as high as when renin is normal, and 5 times as high as when renin is low. Furthermore, the incidence of myocardial infarcts is absolutely zero among low-renin mild hypertensives if there are no other risk factors such as smoking.

As you might expect, not too many of your patients are going to be considerate enough to fall neatly into one of these two ES sub-categories. To be honest, most of your ES/CVD patients will have elements from both columns in their clinical picture. In other words, they will have excess catecholamines **and** excess cortisol. Or, they will be both Dysaerobic **and** Parasympathetic, for instance, or perhaps both Glucogenic **and** Anaerobic.

Your Nutri-Spec Testing protocol does a very nice job of focusing on the most pressing needs of your patient at the moment. It also does a nice job of adapting to the changes in your patient's body chemistry as your NUTRI-SPEC regimen begins to show its benefits.

The often quick response to NUTRI-SPEC is why we cannot emphasize strongly enough the absolute essentiality of the first follow-up test being within a week of your initial testing. Significant changes are likely to be required in the supplement regimen within a week. You **want** changes to happen quickly. The supplements you recommend based upon the patient's initial testing are to be thought of in two ways – both as therapeutic and as a **clinical challenge.** Seeing how the patient responds to the initial supplementation tells you as much or more than the initial testing did.

Electrolyte Insufficiency

A natural diet provides 5 grams or more of salt daily. Yet there are people, usually so-called "health fanatics," who so severely restrict salt intake that they do not receive even this small amount. The point to realize is that, despite the harm of excess salt intake to a small percentage of your patients (with an ES (R-) imbalance), Na and Cl are essential nutrients. Furthermore, there are certain patterns of metabolic imbalance that reduce the retention of Na and Cl, as excess quantities are lost in the urine. Excessive losses of these essential nutrients must be replaced daily.

Clinical findings reflecting poor retention, not just of NaCl but of other electrolytes as well, are reflected in your Nutri-Spec Testing analysis for Electrolyte Insufficiency (EI) Imbalance, and include:

- Orthostatic Pulse Rate increased
- Blood Pressure decreased
- Orthostatic Blood Pressure failure

Poor retention of electrolytes, worn out, stressed out, and too weak to face any challenge – that describes your typical Electrolyte Insufficiency patient.

What ails these patients, anyway? Quite simply, they are depleted in electrolytes and drained of plasma volume (**Hypo**volemic) in association with some combination of Fundamental Metabolic Imbalances and the accompanying hormone insufficiencies.

A decrease in extracellular fluid volume by 5% causes an extreme orthostatic tachycardia, as well as extreme orthostatic hypotension (with the systolic blood pressure decreasing by 10 or more). Even lesser decreases in extracellular fluid volume still cause a significant orthostatic failure of the cardiovascular system, and a decrease in physical performance. These decreases in extracellular fluid volume are revealed by your Nutri-Spec Testing analysis of Electrolyte Insufficiency.

Total body sodium content is the chief determinant of extracellular fluid volume because sodium is essentially confined to the extracellular fluid, with negligible cellular infiltration. Sodium, the cation present in the highest concentration by far, maintains the osmotic pressure of the serum. The extracellular fluid volume is monitored by baroreceptors that send a signal to the kidneys to increase or decrease sodium excretion as needed. Thus, any decrease (or increase) of ECF volume indicates some form of sodium imbalance. The mechanism by which the kidneys retain and excrete sodium and water as needed is under the control of glucocorticoids, mineralocorticoids, angiotensin II, sympathetic and parasympathetic input to the kidney, and natriuretic factors.

The clinical signs of sodium depletion are those of low extracellular fluid volume, and are picked up by your Nutri-Spec tests for EI Imbalance. Most notable are orthostatic blood pressure failure and orthostatic tachycardia.

Sodium loss from the body is always combined with water loss, resulting in ECF volume depletion. Whether the ECF volume depletion is hypotonic, isotonic, or hypertonic depends upon the mechanism of sodium and water loss, plus the amount of fluid ingested. Among the mechanisms of sodium and water loss, the following result in combined sodium and water deficits, and thus isotonic body fluids:

- Diarrhea; Vomiting
- Sweating
- Renal loss (Anaerobic Imbalance = renal loss of sodium and chloride and water, with a decreased urine specific gravity)
- Adrenal insufficiency (Dysaerobic Imbalance = renal loss of sodium, but with water retention and increased urine specific gravity)
- Diabetes mellitus
- Diuretic therapy = loss of sodium and water and potassium and magnesium, and a Metabolic Alkalosis
- Primary Metabolic Alkalosis = sodium excreted but chloride retained

Hyponatremia can also exist in conditions that cause sodium loss greater than water loss:

- Excess water intake in response to ECF depletion
- Thiazide diuretics = salt + water loss = hypovolemia = posterior pituitary ADH secretion = increases volume a bit, but causes further sodium loss
- Thiazide diuretics = salt and water loss = hyponatremia + hypokalemia + Metabolic Alkalosis = increased intracellular sodium uptake and no decreased ECF volume, but an increase in posterior pituitary ADH = further sodium loss
- Adrenal (pituitary) insufficiency associated with a Dysaerobic Imbalance and low glucocorticoids and mineralocorticoids
- Excess posterior pituitary ADH caused by drugs, including NSAIDs, barbiturates, opioids, etc., and by stress
- Cycling pre-menopausal women = cerebral edema associated with hyponatremia from excess estrogen and resultant decreased solute extrusion from brain cells due to the inhibition of sodium-potassium ATPase = diabetes insipidus can result.

Hyponatremia + hyperkalemia + hypotension = generally indicates some form of adrenal corticoid insufficiency. Hyponatremia + hypotension + ECF depletion, but potassium and adrenal function reasonably normal, will respond to a 0.9% sodium chloride solution, which will correct both the hyponatremia and the hypotension. However, a 3% or even a 5% sodium chloride infusion must be used in severe cases. These deficits of sodium chloride and other minerals cannot be corrected with hypertonic solutions ingested orally, since drinking a hypertonic solution does not cause the electrolytes to be absorbed into the blood, but rather causes water to be pulled out of the blood and into the GI tract. (That is the mechanism by which saline laxatives work.) Oral replacement of electrolytes in your EI patients must be achieved gradually as the renal capacity for mineral retention is improved by your recommended NUTRI-SPEC supplement regimen.

To help you clearly envision where the body chemistry is breaking down in your EI patients, page 12-23 shows a Table paralleling the Table categorizing the mechanisms of Electrolyte Stress Imbalance. This EI chart is presented in the same manner, such that you can see the two principal sub-categories of EI patients.

To prepare for the discussion of your EI patients that follows, take just a minute and review the water/electrolyte dynamics illustration. Recall that the major body fluid compartments are plasma, interstitial fluid plus lymph, and intracellular fluid. (The intracellular fluid can be further broken down into the parenchymal cells, the RBCs, and the endothelial cells.)

	ES (R+)	ES (R-)
Renin Activity	+	-
Primary Stress Hormones	Catecholamines ADH/Vasopressin (Secondary Aldosterone)	Glucocorticoids (Mineralcorticoids Hi or Normal) (Insulin or Insulin Resistance)
Metabolic Imbalances	DYSAEROBIC GLUCOGENIC SYMPATHETIC P'SYMP INSUFFICIENCY (K-DEPLETION ALK) (RESP ACID)	ANAEROBIC KETOGENIC PARASYMPATHETIC K-DEPLETION ALK (RESP ACID)
Fluid Distribution	Plasma Volume Hi Interstitial Volume ? Intracellular Volume Hi	Plasma Volume Hi Interstitial Volume Hi Intracellular Volume Hi or Lo
Fluid pH	Plasma Hi or Lo Urine SpGr Hi	Plasma Hi or Lo
Na+ Status	Must not severely restrict intake; dilutional hyponatremia	Excess retention: must restrict intake (salt-sensitive hypertension)
Cl- Status	Cl- -sensitive hypertension	Some decreased retention of Cl- (loss of K+ and H+)
Mechanisms	CL- excess causes vasoconstriction of the afferent renal tubule arteriole = Hi renin = vasoconstriction systemically Hi renin = Hi ADH (H2O retention & Na+ loss) & Hi angiotensin = H2O retention & Hi catecholamines (dangerous positive feedback loop), & secondary Hi aldosterone	Natriuretic peptide deficiency = decreased vasodilation & Hi Na+ (& renin Lo = 1/3 Normal) Hi adrenal corticoids = Hi retention of Na+ & H2O Insulin Resistance & Hi protein kinase C = vaso-constriction & atherogenesis & sodium retention

	EI (A-)	EI (R-)
Hormonal Involvement	Aldosterone Lo (Glucocorticoids Lo) Catecholamines Hi (Norepinephrine) (Renin Hi in compensation)	Renin Lo Catecholamines Lo (Alpha Adrenergic Receptors)
Metabolic Imbalances	DYSAEROBIC GLUCOGENIC SYMPATHETIC RENAL/K EXCESS ACIDOSIS (RESP ALKALOSIS)	ANAEROBIC KETOGENIC PARASYMPATHETIC RESP ALKALOSIS (RENAL/K EXCESS ACIDOSIS)
Fluid Distribution	Plasma Volume Lo Interstitial Volume Hi or Lo Intracellular Volume (Hi)	Plasma Volume Lo Interstitial Volume Lo Intracellular Volume (Lo or Hi)
Fluid pH	Serum Acid Interstitium Alkaline	Serum Alkaline Interstitium Acid or Alkaline
Na+ Status	Must increase intake significantly since renal retention Lo (But Cl- not lost w/Na+)	Moderate increased intake of NaCl needed
Mechanisms	Aldosterone Lo = renal loss of Na+ (& Ca+2) & excess retention of K+ & H2O = Lo BP Plasma Albumen Lo = Lo oncotic pressure = Interstitial edema & alkalosis = (Hi K+ & HCO3- & CO3--) Plasma hypotonic = H2O may move thru ISF into cells Na+ needed to assimilate glucose & decrease excess catecholamines Na+ removes excess Ca+2 from cells = anti-DYS & anti-SYMP	Renin Lo = renal loss of Na+ (&Ca+2) & extreme hypovolemia ISF Volume drops even more than Plasma Volume since oncotic pressure OK, &, since ISF translocates to plasma to compensate. Plasma isotonic Fatigued or over-stimulated cell absorbs excess Na+ & H2O. Na+ at cell level is anti-adrenergic.

Recall also that each body fluid compartment corresponds to a different level of biological organization. The plasma is part of the blood, which corresponds to the systemic level of biological organization. The interstitium relates to the tissue level of biological organization, and the intracellular fluid compartment relates to the cellular level of biological organization.

The interstitium is the one that seems to create some confusion. When we say, for instance, that an Anaerobic patient is acidic at the tissue level of biological organization, or that a Dysaerobic patient has alkaline tissues, we are referring to the abnormal pH of the interstitial fluid. It is the abnormal biochemistry at the tissue level of biological organization (i.e., at the interface between cells) that is responsible for many of the symptoms that bring patients to us – including pain sensitivity, allergies, emotional symptoms, digestive disorders – not to mention vertigo, itching, and the various other dualistic symptoms and conditions.

Note in Figure 1, page 12-5, that the pores between the endothelial cells allow solute to pass between the plasma and the interstitial fluid, but allows the passage of almost no albumin (nor globulin, nor other macromolecules). This macromolecule impermeability is important because it is the oncotic pressure of the albumin in the plasma that helps hold water, and thus maintain normal plasma volume. Understanding the importance of oncotic pressure due to albumin is essential when we make the distinction between the two sub-categories of EI patients.

Let us now examine the EI Imbalance Table. You will note the sub-category headings for your Electrolyte Insufficiency patients are "EI (A-)" (Low Aldosterone), and "EI (R-)" (Low Renin). The first section of each column deals with the hormonal factors associated with each EI sub-category. Next, you will find the NUTRI-SPEC fundamental metabolic imbalances that are typical of each EI sub-category. Note how this Table parallels the analogous Table for the ES sub-categories.

In the metabolic imbalances section of the chart you will see a Respiratory Alkalosis listed under EI (R-), and a Renal/Potassium Excess Acidosis in the EI (A-) column. Let us elaborate a little on these Acid/Alkaline shifts.

A Respiratory Alkalosis will always result from low blood pressure or pulse pressure (which you find in all your EI patients), since low blood pressure induces **hyper-ventilation** (via baroreceptors), which blows off carbon dioxide (decreasing carbonic acid). This tendency to a Respiratory Alkalosis in all people with low blood pressure is particularly pronounced in your EI (R-) EI patients since they have a tendency to be systemically alkaline anyway.

Now consider the Potassium Excess Acidosis tendency in your EI (A-) patients. This Acidosis is not due to excess potassium intake as much as with decrease adrenal corticoids. That corticoid insufficiency causes **excess renal retention** of potassium, as sodium and other minerals are lost via the urine. The sodium loss is critical, and NaGP supplementation is often needed.

Here we have a patient with an Acidosis tendency due to the weak adrenals, while at the same time an Alkalosis tendency due to the low blood pressure. What does this do to your test results? The patient will rarely show a clear-cut Acidosis pattern. But the beauty of your Nutri-Spec Testing protocol is that the analysis and treatment of this Acidosis tendency is built right into the EI Nutri-Spec Testing page. Likewise, the tendency toward an Alkalosis in your EI (R-) EI patients is picked up and handled automatically with your Nutri-Spec Testing analysis. In other words, though virtually all your EI patients are vacillating continuously between Acidosis and Alkalosis, you will be able to manage those stressful swings in body chemistry with the proper NUTRI-SPEC regimen.

The next section on your Table describes the fluid distribution pattern typical of each sub-category of EI patients. The universal finding among all EI patients is, of course, that they are

Hypovolemic. But though the plasma volume is low, the fluid distribution in the interstitium and the intracellular fluid varies depending on which metabolic imbalances predominate.

The pH abnormalities of the body fluid compartments are covered next in the Table. Note that they are opposite between one sub-category and the other. But note even more importantly that they are opposite within each sub-category between the systemic and tissue levels of biological organization. When you have done NUTRI-SPEC for a short while, you will realize that there is almost no such thing as calling a patient either Acid or Alkaline. Most often, patients with pH aberrations are Acidic in one fluid compartment and Alkaline in another. Only with NUTRI-SPEC can you thoroughly analyze the dynamics of fluid movement and composition and the associated membrane dysfunctions.

Sodium is an essential consideration for all your EI patients. The sodium status of each EI sub-category is covered next in the Table.

Now, let us look a little deeper into the mechanisms underlying the two major sub-categories of EI imbalance. First, look at the EI (A-) column. Due either to excess protein hydrolysis in the blood, (9) or to insufficient dietary protein, there is low albumin activity in the serum. As a result, fluid leaks from the albumen-deficient plasma into the interstitial fluid, since colloids are designed to prevent interstitial fluid edema (as their oncotic pressure keeps water in the plasma). (16) So – in this sub-category of EI patients, you will typically have interstitial edema and interstitial Alkalosis. (This interstitial edema applies to all tissues except the skin, as water accumulates in the upper GI tract at the expense of the skin. The skin can be very dry.)

The excess interstitial fluid may also pass into the cells (especially if there is a strong Dysaerobic component to the patient's body chemistry) due to altered membrane function, as well as to the low plasma and interstitial oncotic pressure.

Sodium is important in these patients because it is needed to absorb glucose, thereby decreasing excess stress hormones norepinephrine and epinephrine (and increasing ATP). Sodium is also important because it removes excess calcium from the cells, thus having a good anti-Dysaerobic and anti-Sympathetic effect in these patients. (17,18)

Now, look over the EI (R-) column. In these patients the interstitial fluid volume (and often the intracellular fluid volume) will decrease even more than the plasma volume. This occurs since there is generally adequate albumin in the plasma to maintain enough oncotic pressure to hold water in the plasma at the expense of the interstitium. The low interstitial fluid volume also occurs to some degree since the interstitial fluid translocates to the plasma as an adaptative response to the hypovolemia in an attempt to maintain somewhat normal plasma volume. (19)

In many of these EI (R-) patients, there is so little ability to hold water at any level of biological organization that their hydration status is incredibly low. The more water they drink, the more they urinate – but none of that water can be held within the body.

Even though these patients desperately need sodium at the systemic level and at the tissue level, they may have excess sodium at the cellular level. The reason is that fatigued or over-stimulated cells absorb excess sodium and water. (20) The sodium entering the cells is anti-adrenergic – which paradoxically exacerbates the EI (R-) imbalance. Only by correcting the fatigue and metabolic stress with the proper NUTRI-SPEC regimen (which includes judicious sodium supplementation) will the cellular vitality be increased enough to release the excess sodium.

One thing you must understand regarding the sub-categories of EI Imbalance is that many of your EI patients will not fall strictly within one column of the Table. They will have elements of both sub-categories.

In other words, they will be somewhat low in both aldosterone **and** renin. They may have, for example, both a Glucogenic tendency **and** an Anaerobic tendency. More than one mechanism is at work in these patients, depleting them of electrolytes and destroying their control of fluid dynamics and pH.

The best feature of your Nutri-Spec Testing protocol for EI patients is that it sorts through each patient's test results, enabling you to choose exactly the electrolytes and adaptogens to restore strength to each individual.

Clinical Significance of Electrolyte Insufficiency

Your practice is full of hypotonic, **Hypo**volemic weaklings. Patients by the dozens drag themselves into your office every day with the following complaints:

- chronic fatigue
- muscular weakness
- anemia
- postural dizziness
- decreased libido
- poor circulation
- deficient self-assurance
- osteoporosis
- accelerated aging

You realize now that many of these patients have an EI Imbalance. You know that this imbalance is all about two things:

- poor mineral retention
- fluid dynamics out of control
- loss of zeta potential and liquid crystalline water

What must you do to put the wind back in the sails of these flaccid patients? You have several NUTRI-SPEC supplements at your disposal to help restore normal fluid dynamics. But just as importantly, you must restore the lost minerals. This includes most particularly sodium chloride and/or sodium glycerophosphate.

Countless studies are reported in the research literature highlighting the essentiality of salt. Probably the most fascinating of these is the study done at Johns Hopkins University and published in the September 1995 Journal of the American Medical Association. (24) This study demonstrates a treatment for **chronic fatigue syndrome** that resulted in improved energy levels in 76% of the patients. We have never seen another study that achieved such dramatic symptomatic improvement in this devastating clinical condition. What was the keystone of this phenomenally successful treatment for chronic fatigue syndrome? Sodium chloride. These patients virtually all had low blood pressure accompanying their chronic fatigue and responded dramatically to an increased intake of common table salt. In NUTRI-SPEC terms, of course, these patients had an Electrolyte Insufficiency Imbalance.

Research done at Hartford Hospital in Connecticut and reported at the American College of Cardiology Conference in Anaheim in March of 1997 also studied chronic fatigue syndrome. It was found that more than half the patients achieved symptomatic improvement by increasing their salt and water intake. (22)

In the March 14, 1998 issue of The Lancet, a study was published showing the harmful effects of restricting salt intake. The data for this study was collected from over 11,000 subjects over a period of over 25 years, beginning in 1971. There was an inverse relationship discovered between salt intake and mortality. In other words, the length of your life is directly proportional to how much salt you eat. (23)

Never lose sight of the clinical importance of sodium chloride. There is a small segment of your patient population who have an Electrolyte Stress Imbalance and fall into the low renin category (refer to the ES (R-) column of the Electrolyte Stress Chart) who must restrict sodium intake. Some other Electrolyte Stress patients need to be careful about salt intake because of its chloride (not its sodium) content. All the rest of your patients must be careful to obtain adequate salt. This is not a problem for most, because our need for salt is accompanied by an appetite that is almost certain to meet our needs. In your EI patients, however, the taste for salt – even a craving for salt – may not provide adequate intake, at least until you have corrected the fundamental metabolic imbalance.

The problem with your Electrolyte Insufficiency patients is not just with sodium chloride but with poor mineral retention in general. Once EI patients have lost the ability to retain electrolytes, they are left without the strength to handle most any kind of stress load – either physical or emotional. These patients are a deflated balloon begging for you to pump them up.

Why have these Electrolyte Insufficiency patients lost their ability to retain minerals, and why are they likely to suffer from one or more of the conditions listed at the top of this section? The short answer is, they have "weak kidneys." The complete answer is that they have lost kidney control of electrolyte balance because of a combination of metabolic and endocrine imbalances.

As illustrated in the Electrolyte Insufficiency Table, there are two sub-categories of endocrine dysfunctions. One represents a **renin** insufficiency and one represents an **aldosterone** insufficiency. These represent the two major sub-categories of EI patients – EI (R-) and EI (A-).

What is the association between renin and "weak kidneys? Via renin, the kidney works with the liver and lung to form angiotensin, which has much to do with vascular tone and with the retention of mineral salts.

You also see on the chart that EI (R-) patients typically have one or more of Anaerobic, Ketogenic, Parasympathetic, or Respiratory Alkalosis Imbalances. (24)

Your other basic category of EI patients consists of those whose adrenal aldosterone output is generally inadequate. This deficiency results in further loss of sodium and chloride, along with citrates, bicarbonates, and calcium. The low aldosterone output is also reflected in the decreased pulse pressure and an increased urinary specific gravity. Your EI (A-) patients are typically found to be either Dysaerobic, Glucogenic, or Sympathetic and will likely have Renal or Potassium Excess Acidosis.

What else goes wrong with the kidneys of your EI patients? Kidney dysfunction can be associated with anemia. A normal kidney splits erythropoietin from plasma protein, thus stimulating bone marrow RBC production.

The kidney also produces the active form of vitamin D. Renal endocrine insufficiency thus results in calcium deficiency, osteomalacia, and osteoporosis.

The protein anabolism of testosterone and DHEA is partially mediated by the kidney. There is a reciprocal relationship between androgens and the kidney, as androgens are renotropic. They increase the size and weight of the kidneys. Androgens are extremely anabolic. This anabolic

property is partly under renal control. Your EI patient will often be emaciated in association with poor protein metabolism.

Your Nutri-Spec Testing protocol for this imbalance has given you the first comprehensive and effective approach to patients suffering the debilitating fatigue and stress, and hormonal imbalances associated with EI Imbalance.

The ability to retain electrolytes and the ability to control electrolyte and water composition in the three body fluid compartments are such a critical part of maintaining health. A failure of this metabolic balance system pulls the plug on a patient's personal power.

Sodium Supplementation and Fluid Retention

The amount of sodium you give many of these EI patients may concern you, particularly in those patients (usually high estrogen or high cortisol women) who have a tendency to retain fluid.

Our experience has been that the sodium salts not only do not increase fluid retention, they actually decrease fluid retention by strengthening the adrenal and pituitary control of fluid dynamics. However, there is the possibility of running into problems with fluid retention if one or both of the following problems occur:

The first of these problems is that you do not monitor your patients' hydration, and so their water intake is inadequate. They will not have adequate water to keep the electrolytes moving into the cells and/or out through the kidneys. In such cases, you can get an interstitial edema until the patients consume enough water.

The other common problem in people who retain fluid on the indicated electrolytes is in those who are eating too many carbohydrates. This carbohydrate-induced fluid retention is common to people of all metabolic tendencies, not just those with an Electrolyte Insufficiency Imbalance. There are many people who can eat two pieces of bread and gain more weight than the weight of the bread. It is as if the bread were a sponge that diffuses throughout their interstitium, sucking up enough water (and salt) to make them puffy and squishy.

We wish we could say the exact mechanism by which this carbohydrate-induced fluid retention occurs – but we cannot. The reason we cannot get a good handle on it, despite having studied hundreds of patients who suffer with the problem, is because the condition is probably largely hormone-related and thus can cross the boundaries of the metabolic balances. In other words, since excess estrogen is always a factor, as is excess insulin or excess cortisol in some cases – you will see this fluid retention reaction in patients who are Ketogenic as well as those who are Glucogenic, and in Anaerobic patients as well as Dysaerobic patients.

Another way to say this is that excess carbohydrate hits patients at their weakest link, exacerbating whatever metabolic imbalances they tend to have. And – virtually all the metabolic imbalances can be associated with abnormal fluid and electrolyte dynamics which, in turn, can be associated with fluid retention.

One other major factor in this carbohydrate-induced edema relates not so much to the direct effect of the excess carbohydrate, but rather because excess carbohydrate means, almost by definition, insufficient protein. The colloidal effect of adequate protein in the body fluids prevents interstitial edema since the oncotic pressure caused by the proteins keeps fluid in the plasma.

For the purposes of this discussion on Electrolyte Insufficiency Imbalance, the main thing you need to know is that the sodium salts will give your patients a tremendous surge in vitality if normal hydration is maintained, and, if their carbohydrate intake is appropriate.

Other Considerations Regarding Drinking Water

Public water supplies contain large quantities of chlorine. Chlorination is essential to the distribution of sanitary water for household use. For household uses **other than** drinking that is. (Although recent evidence suggests that bathing in chlorinated water may play some role in increased incidence of Melanoma skin cancer.)

Chlorinated water is a significant contributor to arteriosclerosis. Chlorine sets off a free radical chain reaction via the enzyme xanthine oxidase, damaging smooth muscle cells in arterial walls. This oxidative damage is followed by mutation and proliferation of the damaged cells, leading to atheromatous plaque formation. Clinically, this arterial damage results in atherosclerotic senile degeneration, coronary, and stroke.

Chlorinated water also results in the formation of chlorinated hydrocarbons in water, which are carcinogenic. Trihalomethane, the byproduct of chlorination, also increases cancer risk. (26) A study by the California health department shows that chlorinated drinking water can increase the risk of miscarriage by 65%.

Chlorinated water is a killer. It is not fit for human consumption. You should routinely recommend to all your patients that they find a natural source of drinking water.

What about fluoridation? Every country outside the U.S. that has tried fluoridating water to promote dental health has ultimately outlawed the practice due to its being ineffective and dangerous. Fluoridated water does not improve dental health; in fact, it actually makes teeth more brittle.

The quantities of fluoride found in many public water systems are toxic and inhibit cellular DNA repair. It is thus carcinogenic. It is also a causative factor in Down Syndrome. All your patients should be advised to avoid fluoridated water.

The case against drinking public water is enhanced by pointing out the residues of aluminum salts and other flocculating agents that it contains. We have shown that these have a potent sludging effect on body fluids.

Finally, the case against public water is completed by noting the clinically significant quantities of pharmaceuticals found in water that has been through water treatment plants. Billions of pounds of drugs are eliminated annually in people's urine and feces. Waste treatment plants do not even touch much of this drug load, which is then "recycled" to you in your "drinking" water. Giving one example – the concentration of antibiotics is 1000 times higher in
U.S. water than German water. The Center For Adaptative Genetics And Drug Resistance at Tufts University speculates that this may contribute to the antibiotic resistance developed by so many bacteria. Antibiotics are only one of hundreds of drugs found in public water.

There is a tendency in your quest to provide the most scientific, most sophisticated and most efficacious clinical nutrition to your patients, to neglect the most basic nutrient of all -- and that is natural water.

There are actually three reasons why, long term, natural water may be the most important nutrition consideration for all your patients.

First: A good source of natural water is essential in preventing strokes, heart attacks, and kidney degeneration that may be derived from toxins in municipal water.

Second: Natural water is the best source of certain very valuable mineral nutrients.

Third: Natural water has electromagnetic properties that give it powerful biological activity.

Adequate natural water intake can take the load off the kidneys and help prevent the most devastating pathology of our modern society.

An important benefit of natural water is as a source of nutrient minerals. The ionic minerals in water **are assimilated more efficiently than the minerals in food.**

The minerals in natural water occur both as ions, which is exactly how they exist in body fluids. These minerals are assimilated and utilized more effectively than the minerals in foods, thus making a significant contribution to mineral nutrient intake. Assimilable multivalent forms of minerals exist in water that are not found in food. Even low valence cations like Ca++, K+, and Mg++ tend to be combined in natural water with multivalent anions, and are thus assimilated. Multivalent anions in natural water are important as dispersing agents. Many natural waters are also high in silica, one of the most common nutritional deficiencies.

You can see, therefore, that it is not just the quantity of natural water that has a protective effect on the cardiovascular system, but the quality of natural water, with its natural content of dispersing agents and other minerals.

Hard water, i.e., with high Total Dissolved Solids, is directly correlated to health and longevity, and inversely related to heart disease, cancer, and aging. (26) A study in Sweden confirmed the benefits of water hardness on the coronary death rate and the incidence of strokes. They found a 41% difference with respect to these pathologies between those who consumed hard water vs those who consumed soft water. (27) Another study demonstrated the effect of mineral nutrients on reducing blood lipids in those with risk factors of cardiovascular disease. After only six weeks, those with a diet supplemented with calcium, magnesium, potassium, copper, selenium, and chromium compared to those on an unsupplemented diet showed a significant fall in triglycerides, total serum cholesterol and LDL cholesterol, while HDL cholesterol remained unchanged. (28)

Considering the above, we recommend that drinking water be from a natural source such as a spring or a well. We further recommend that it be relatively hard water with a hardness factor of 170 or a total dissolved solids of 300 ppm. The water pH should ideally be 7 or above.

What about filters or water purifiers for your public water? These are beneficial only in the rare instance that your public water has a pH above 7 and the desirable hardness. Then and only then will a good filter produce good drinking water. Otherwise, you are wasting your time and money. You are better off finding a source of spring water.

Water softeners can be deadly. They function by replacing the essential minerals Ca and Mg with Na. A lifetime of drinking this Na-filled water may inevitably increase an ES patient's susceptibility to Electrolyte Stress and blood sludge. All your patients who demonstrate Electrolyte Stress should be asked if their drinking water comes through a water softener. If so, they should be instructed to immediately find a source of spring or well water. The softened water is ideal for other household uses, but should under no circumstances be used as drinking water.

The third important consideration regarding drinking water is the biological activity of natural water. This biological activity is associated with the complex grouping of the water molecules (see Anaerobic/Dysaerobic Balance). This grouping of molecules results from natural water's electromagnetic properties. By biological activity is meant the ability to enter into biochemical reactions in the body.

Distilled water molecules are not grouped and thus have **no biological activity.** Ungrouped water molecules are the waste water of the body. In fact, the kidneys **only** eliminate ungrouped water. Water molecules are ungrouped in freshly distilled water, and ungrouped in heated water. Water molecules are grouped in natural water, and most grouped in melted ice. (15)

Having completed this chapter on the first of the Five Fundamental Balances, you should begin to appreciate the **prophetic** capacity of the NUTRI-SPEC system. In other words, health problems can be detected **before** they become pathological, and pathology can be detected **before** it creates a crisis. With Nutri-Spec, you are enhancing health rather than treating disease.

Half of all Americans die of cardiovascular disease. Think about it. Think about how that relates to what you just learned in this chapter. Half the people you know - family, friends and patients, are going to die partly due to fading health associated with Electrolyte Stress Imbalance. If you get nothing more out of NUTRI-SPEC than the knowledge you just acquired, plus the resolve to apply that knowledge, you will produce more value as a clinician than you have with the sum total of all the therapies you have used in the past.

Then, think about how many of your patients suffer from chronic fatigue and the other debilitating symptoms of Electrolyte Insufficiency Imbalance. Only with your NUTRI-SPEC system can these patients be empowered with the vitality to take active control of their lives.

How do you begin to apply your knowledge of Water/Electrolyte balance? Immediately find a source of healthful drinking water from a spring or well for yourself and your family. Immediately begin recommending a healthful source of drinking water for your patients. Recognize that most people need to increase their water intake, and that approximately one-third of the day's water intake should be consumed upon arising in the morning, before taking any food. Employ your knowledge about the importance of salt, looking for both excesses and insufficiencies. Begin using your high biological activity NUTRI-SPEC supplements according to your Nutri-Spec Testing protocol.

You will be amazed at the health-restoring impact of achieving Electrolyte/Water balance. Electrolyte/Water balance is perhaps no more important than any of the other Fundamental Balances, but it **is primary.** In other words, nothing else you do therapeutically will have any lasting benefit until Electrolyte/Water balance is achieved.

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