CHAPTER 13

ANAEROBIC/DYSAEROBIC BALANCE EXPAINED

Introduction

We begin now a discussion of the third of your Five Fundamental Balances, Anaerobic/Dysaerobic Balance. Anaerobic/Dysaerobic Imbalance can be thought of in 2 ways. It can be thought of as an imbalance in oxygen metabolism, and, as an imbalance in lipid (fat) metabolism. In this discussion you will learn that the two, oxidative metabolism and lipid metabolism, are inextricably related.

(Due credit must be given to Revici (1) from whose Anabolic/Catabolic paradigm your Anaerobic/Dysaerobic balance is largely derived.)

Anaerobic/Dysaerobic balance represents a dualistic, diphasic model of both oxidative metabolism and of lipid metabolism. This imbalance relates to the metabolic use of different types of fats (fatty acids and sterols) with 2 objectives:

1) to <u>produce energy</u> with oxygen, and,

2) to control <u>membrane permeability</u>

When there is an Anaerobic/Dysaerobic Imbalance, the composition of fats in tissue membranes is out of proportion. There are either excess fatty acids (Dysaerobic), or excess sterols (Anaerobic) in the membrane structure, affecting the membrane's ability to control selective permeability.

Regarding oxidative energetics: Anaerobic Metabolic Imbalance represents an insufficiency of oxidative metabolism. The Anaerobic patient, instead of producing energy with oxygen, is relying on fermentation (anaerobic glycolysis rather than the Krebs Cycle) to produce energy, which is very inefficient. The Anaerobic patient can also be thought of as having insufficient fatty acid activity and excess sterol activity. It is this imbalance in lipid metabolism that causes problems both with energy production and with membrane permeability.

In contrast, Dysaerobic Metabolic Imbalance represents oxidation out of control, i.e., oxidative free radical reactions with peroxide formation. This patient produces energy with oxygen but in a form that the body cannot use. Instead of producing useful energy, the Dysaerobic patient produces catabolically damaging free radicals. The Dysaerobic patient can also be thought of as having excess fatty acid activity and insufficient sterols. This imbalance in lipids results in the abnormal energy production, and in the changes in membrane permeability typical of the Dysaerobic patient.

Are we talking about anything important here? When we talk about oxidative energy production and the selective permeability of biological membranes we are talking about the essence of life itself.

That is why you can consider Anaerobic/Dysaerobic balance as the most pervasive of your five NUTRI-SPEC metabolic control systems. Without strength and balance in both anti-anaerobic and anti-dysaerobic forces, your patient cannot:

- produce oxidative energy in sufficient quantity to power a high vitality life.
- prevent the oxidative free radical damage that accelerates tissue degeneration and what Nutri-Spec terms "Exogenous Inflam-Aging".
- maintain glycemic control.
- maintain normal fluid and electrolyte dynamics in each of the three body fluid compartments.
- maintain normal pH in each of the three body fluid compartments.

In other words, Anaerobic/Dysaerobic forces play a part in virtually everything that happens in the human body. Any symptom or condition you can name is likely to have an Anaerobic or a Dysaerobic component.

Summary of Anaerobic/Dysaerobic Imbalance

	ANAEROBIC	DYSAEROBIC
A) Definition:		
	1) Anaerobic glycolytic energy metabolism; fermentation	1) Dysaerobic oxidative energy metabolism; free radical and peroxide formation
	2) Lipids with positive polar groups predominate (sterols, alcohols, amines)	2) Lipids with negative polar groups predominate (fatty acids, ketones, aldehydes)
	3) Anabolic processes	3) Catabolic processes
B) At the Cellular Level:		
	1) Cellular oxidation decreased	1) Cellular oxidation increased
	2) Membrane permeability decreased	2) Membrane permeability increased
	3) Less differentiated cells (especially connective tissue)	3) More differentiated (functionally specialized cells) (especially connective tissue)
	4) Increased amount of connective tissue	4) Decreased amount of connective tissue
	5) Prolonged cellular anabolism	5) Premature catabolic aging
	6) High potassium (low serum K)	6) Low potassium (high serum K)
	7) Low calcium (poor urinary Ca retention)	7) High Calcium (high urinary Ca retention)
C) At the Tissue Level:		
	1) Decreased water content	1) Increased water content
	2) Poor water retention (increased urination)	2) Excess water retention (decreased urination)
	3) Urinary surface tension high (retention of surface active substances)	3) Urinary surface tension low (excretion of of surface active substances)
	4) Local Acidosis of lesioned tissue	4) Local Alkalosis of lesioned tissue
	5) Systemic Alkalosis	5) Systemic Acidosis
	6) Urine pH high; Saliva pH low	6) Urine pH low; Saliva pH high
	7) Low chloride in lesioned tissue (poor urinary Cl and Na retention)	7) High chloride in lesioned tissue (excess urinary Cl and Na retention)
	8) High urinary retention of phosphorous and sulfur	8) Low urinary retention of phosphorus and sulfur

9) Dermographic red line	9) Dermographic white line
10) Oxido-reduction potential processes decreased	10) Oxido-reduction potential processes increased
11) Increased lymphatic tissue	11) Decreased lymphatic tissue
12) Eosinophils high in tissues	12) Eosinophils absent in tissues
13) Pain; itching, vertigo; dyspnea hearing loss; manic depressive	13) Pain; itching, vertigo; dyspnea; hearing loss; manic depressive

D) At the Organ/Organism Level:

1) Surface Tension high (retention of surface surface active substances)	1) Surface Tension low (excretion of active substances)
2) Polyuria	2) Oliguria
3) Urine Specific Gravity low	3) Urine Specific Gravity high
4) Sedimentation Rate low; RBC isolation	4) Sedimentation Rate high; RBC sludging
5) RBC oxygen fixation persistent = decreased oxygen delivery to cells	5) RBC oxygen fixation lacking persistence = excess cellular oxidation.
6) Absence of oxidizing substances in urine = indican low	6) Presence of oxidizing substances in urine = indican high
7) Hematocrit & Hemoglobin high	7) Hematocrit & Hemoglobin low
8) Eosinophils high	8) Eosinophils absent
9) WBC high	9) WBC low
10) C-Reactive Protein negative	10) C-Reactive Protein elevated
11) Blood Pressure high	11) Blood Pressure low
12) Tachycardia	12) Bradycardia
13) Body Temperature high	13) Body Temperature low
14) Somnolence	14) Insomnia
15) Constipation	15) Diarrhea
16) Exophthalmia	16) Exophthalmia
17) Endogenous Inflam-Aging	17) Exogenous Inflam-Aging
18) Death in coma	18) Death while conscious

Lipids and Biological Membranes

We must look more specifically at lipids. Lipids are the key to all biological function. We say that for two reasons:

1) lipids are what biological **membranes** are all about, as membrane structure and function are dependent upon the unique qualities of lipids.

2) lipids play a functional role in virtually all biochemical processes.

What does a lipid look like? For our purposes let us create in our minds an over-simplified picture. Picture a lipid as a long chain of carbon atoms (non-polar), with one end of the chain attached to a polar group. The polar group can be either a negatively charged fatty acid, or a positively charged sterol.

C-C-C-C-C-C-FA(-)   C-C-C-C-C-C-S(+)

A lipid, then, is a substance made up of polar and non polar groups in which the nonpolar predominates, i.e., the Van der Waals forces of the nonpolar group predominate over the electrostatic forces of the polar group (2). A lipid has greater **solubility** in neutral solvents than in water. Its molecules will remain at the surface of water with only its polar group penetrating and its nonpolar group protruding. (The opposite occurs in a neutral solvent.) Thus, we have at the water surface a molecular layer formation.

The very existence of biological entities depends upon the ability of lipids to build up boundary formations (membranes) separating and thus assuring the individuality of those biological entities. Membranes are made up of lipids in layers, which give the membrane polar faces. These polar faces give the membrane the ability to separate two aqueous media. In other words, for example, to separate one cell from another, or to separate a cell from the interstitial fluid.

Now that we have constructed a membrane, let us ask ourselves, "What is its function?". The function is permeability. But should it let just anything in or out? No. And that brings us to the truly fascinating quality of biological membranes - **selective permeability.** Membranes can alter their permeability, both quantitatively and qualitatively, according to changing circumstances. And how is this selective permeability achieved? It is associated with the <u>antagonistic action of the fatty acids and sterols present in the membrane.</u>

Fatty acids increase permeability (especially to anions) due to the perpendicular orientation to the water surface of their nonpolar group. This orientation is favorable for the passage of metabolites. However, the negative charge of the polar group provides a barrier to cations. Cations are attracted and retained by the acid polar group.

Sterols decrease membrane permeability two ways. First, by bonding with fatty acids they block the passage of other molecules through them. Second, the orientation of the nonpolar group almost parallel to the water surface is unfavorable for passage of metabolites.

Membrane function is largely dependent on proper balance of electro-magnetic forces in the membrane. The positive and negative polar groups on fatty acids and sterols are an important part of maintaining proper membrane polarity. <u>The relative proportions of fatty acids and sterols in a membrane determine its degree of permeability</u>. Changes in permeability resulting from the antagonistic action of the two groups of lipids are a major factor in normal and abnormal physiology.

Fatty Acids and Oxygen Metabolism

Fatty Acids (FA) are defined for the purposes of the NUTRI-SPEC Anaerobic/Dysaerobic model as lipids with negative polar groups (FA, ketones, and aldehydes). FA have three physiological functions: in caloric metabolism, in a constitutional role, and in a functional role.

As part of caloric metabolism, long-chain saturated FA are desaturated to mono-ethenic FA, then split into two shorter chains which undergo beta oxidation (in the liver). A hydroperoxide is formed in the process, and is a normal by-product of efficient energy metabolism.

The constitutional role of FA relates to the formation of membranes (especially by di-, tri-, and tetra-enic FA), as discussed above. The constitutional role of FA also includes the formation of complex lipids such as lecithin, a natural neutral solvent.

The functional role of FA is associated with oxygen metabolism and is related to two factors:

1) The presence of an uncombined carboxyl polar group (-COOH).

2) The intervention of the double bonds of the nonpolar poly-unsaturated carbon chain.

Free FA (FFA) are functionally active while combined FA are not. The carboxyl group gives the FA molecule the capacity to realize new chemical combinations. The carboxyl group also is responsible for an induction effect exerted on the non-polar group. This induction effect charges successive carbons of the non-polar group with alternate signs; odd carbons = positive, even carbons = negative. Oxygen combines with + carbons only.

The double bond of the FA gives it the capacity to become a semipolar center and consequently to bind or lose oxygen radicals without altering the chain of the molecule itself. The + carbon adjacent to the double bond is an extremely active carbon due to the proximity of the semipolar double bond. This activity principally involves reactivity with nonmetallic elements.

This semipolar character of double bonds (rendering the nearby carbon highly reactive), allows FA to participate in several forms of oxidation processes. In the normal oxidation process, oxygen is temporarily bound to the reactive nearby carbon to produce a hydroperoxide. Under proper circumstances the oxygen fixation becomes reversible, and the FA subsequently liberates the oxygen in highly activated (ionized) free radical form. Thus, the physiological role of unsaturated FA in oxidation processes is to change oxygen to a high-energy free radical. It should be emphasized that this is normal, healthy free radical production.

Most polyunsaturated FA have two double bonds in a non-parallel position, which exerts a very strong influence on the special carbon in the intermediary position between them. When this carbon has a + character, it is especially able to fix oxygen. This accounts for the role of what we know as "Essential Fatty Acids".

In a Dysaerobic Metabolic Imbalance (i.e., under abnormal conditions), the fixation of oxygen occurs at the double bond itself, instead of the nearby carbon. Thus are formed epoxides or peroxides (depending upon the ionic or molecular character of the oxygen). This dysaerobic condition is associated with - radiation, certain inflammations (e.g. streptococci), selenium, and excess polyunsaturated FA. Peroxides will appear in the urine, accompanied by indican and glucuronic acid due to abnormally intense oxidation taking place on tryptophan and glucose.

Another possibility in a Dysaerobic Metabolic Imbalance (i.e., under abnormal conditions) is the fixation of chloride at the double bond. This allows sodium to remain free to enter into other combinations, especially with carbonate, thus forming strongly alkaline compounds in the interstitial fluid. This results in a local tissue alkalosis.

The Dysaerobic Metabolic Imbalance described above occurs following the **rancidity** of FA. There are three types of rancidity:

1) Separation of fats into FA and glycerol by intervention of lipolytic enzymes (e.g., produced by molds, microbes, or lipase present in tissues.).

2) Conversion of saturated FA into methyl-ketones by beta-oxidation enzymes (e.g., peroxidase in certain molds). This especially affects 8 -12 carbon FA.

3) a) Unsaturated FA (UFA) changed to hydroperoxides by oxidation (normal) or peroxides (Dysaerobic).

   b) UFA may end up with a displacement of the double bonds. These, Revici called conjugated isomers. Conjugation of polyunsaturated FA (PUFA) occurs when the double bonds are displaced into a parallel position. There now exists a reciprocal induction enhancing the oxidative energetic value.

When there are three conjugated double bonds (conjugated tri-enic FA) you have Revici's "abnormal FA". These are the most important FA in the body in terms of pathogenesis. Abnormal physiology results from the inability of the organism to control the processes involved, i.e., the inability to counteract the harmful effects of "abnormal FA". (See the discussion of sterols, below.) In the absence of control, in vivo changes approach those which occur in vitro where there are **no** controls. Rancidity occurs rapidly and in high quantities. Tissue destruction is the ultimate result. As a side note: radiation acts specifically as an "abnormal FA". (3)

Fatty Acids can be divided into four groups based on their physiological role;

1) Saturated and monounsaturated = caloric metabolic role.

2) Di-, tri-and possibly tetra-unsaturated = constitutional, organizational roles.

3) Tetra and higher polyunsaturated = functional roles.

4) Conjugated = pathogenic

   a) FA from pathological tissues are conjugated more readily than FA from normal tissue

   b) Normal tissues have no free lipids (except brain and RBC) (-Revici)

   c) Lesioned tissue has free lipids (This explains the therapeutic effect of Revici's lipid substances - they bind abnormal lipids.)

Sterols and Oxygen Metabolism

Sterols are defined for the purposes of the NUTRI-SPEC model as lipids with positive polar groups (sterols, alcohols, and amines). They can collectively be referred to as "anti-fatty acids" (AFA). AFA function by determining the fate of the FA to which they bind. Again, this is the dualistic concept of FA vs. AFA. The direct activity of AFA is to control and limit FA activity, each AFA selectively influencing (binding) specific FFA. AFA, like FA, have three physiological functions; in caloric metabolism, in a constitutional role, and in a functional role.

The caloric role of AFA is fulfilled by glycerol and the glycerophosphate ion, as they bind saturated and mono-ethenic FA. Their constitutional role relates to membrane construction and the production of phospholipids as glycerophosphoric acid binds di-, tri-, and tetraenic FA. Sterols perform the functional role in oxidative processes by binding polyunsaturated FA (PUFA).

Sterols are steroids with OH- at Carbon 3 and a long chain at Carbon 17. The OH- gives Carbon 3 a nucleophilic character, i.e., it combines in general with substances having a negative polar group to form esters. For example, cholesterol has selective affinity for FA with polyunsaturated chains. This is a labile combination, but while the bond exists, cholesterol demonstrates its antagonism for FA (decreases FFA).

Corticoids are a type of sterol and in general act as positive lipids. Corticoids with no attached electrophilic OH- or nucleophilic O at Carbon 11 have an influence on electrolyte metabolism (mineralocorticoids). Corticoids with an attached electrophilic OH or a nucleophilic O at Carbon 11 are gluconeogenic. Gluconeogenic corticoids function as a "template", forming members of the "gluco group" (glucose and galactose and their amines and their acids, abscorbic acid, etc.) over and again without affecting the corticoid molecule.

AFA can be divided into three groups based upon their biological role:

1) Glycerol and Glycerophosphate ions control absorption and circulation of mono/di/tri-unsaturated FFA.

2) Sterols counterbalance normal polyunsaturated FFA.

3) Adrenal corticoids (especially gluconeogenic) counteract the toxicity of FA in general, and of the abnormal conjugated FA in particular.

    a) The use of gluconeogenic corticoids in connective tissue diseases is partly associated with the role of amino sugars (e.g., glucosamine) in connective tissue constitution.

The influence of AFA on FA relates to their combination with FA, thus decreasing FFA, thus decreasing the role for which that particular FA is suited. Both when decreasing membrane permeability and when decreasing FA functional activity, AFA exert their influence by altering oxygen metabolism. Therefore, they favor anaerobic in contrast to normal aerobic metabolism.

Four Mechanisms of Lipid Physiological Activity

A) Activity via its **lipid solubility character:**

1) There is a selective influence upon those entities with lipids in an active form in their constitution. The influence exerted is proportional to the richness of the entity in these active lipids.

2) This explains why administration of a lipid affects selectively the abnormal entities rich in free lipids, and to a much lesser degree, the normal ones. (Only lesioned tissues or cells contain free lipids.)

B) Activity via the bond realized through the **charge of the polar groups**:

1) A wide variety of nonpolar groups have similar activity if their polar groups have the same charge (+/-).

   a) sterols, alcohols, amines = similar activity (+)

   b) FA, aldehydes, ketones = similar activity (-)

C) Activity due to the **chemical constitution of the polar group:**

1) Activity differs between varying polar groups of the same charge.

D) Activity related to the **nonpolar group, specifically its energetic formation:**

1) Greatest specificity of action.

E) A) and B) = general, non-specific effects such as membrane permeability

C) = specific action on certain metabolic processes via intervention of metabolites or on the agents (enzymes) governing them.

D) = specific action upon **one** particular metabolic system (This explains the therapeutic effect of Revici's lipid substances on many pathological conditions.)

Physiological Activity of Specific Lipids

I) Lipids with negative polar groups:

A) **Saturated FA:** members with an even number of carbons are caloric metabolites. They are absorbed, circulated and stored as triglyceride

B) **Unsaturated FA:**

1) **Monoethenic** (e.g. oleic) have **little physiological effect.**

2) **Tri-/di-ethenic** (e.g. linolenic and linoleic) have an organizational more than a functional role.

a) Their preferred bond is to glycerophosphoric acid. Their intestinal absorption is as phospholipids.

b) **Mild anti-anaerobic effect**

3) **Arachidonic Acid** (Salmon oil). Its caloric role is nil with relation to its functional role.

a) Absorbed, circulated and stored principally as esterifying sterols.

b) Represents greater than 25% of the FA in the adrenals. Liberation occurs during the stress response (with particular role as part of the immune defense mechanism).

c) The adrenal PUFA intervene in normal physiology, liberation alternating with liberation of sterols and corticosterones. These are the dualistic, diphasic oscillations which characterize physiological dynamic balance.

d) **Mild anti-anaerobic effect at the systemic level; moderate at lower levels.**

4) **PUFA with more than 4 double bonds (Cod liver oil, sardine oil) have a mild anti-anaerobic effect at the systemic level; strong anti-anaerobic effect at lower levels.**

a) PUFA are transported in blood within RBCs. After injecting PUFA, RBCs contain 5-6 times as much PUFA as plasma.

C) **Conjugated PUFA** have a greater effect than their non-conjugated isomers, and a longer lasting effect.

D) **Fading Response** to FA

1) Effects are of short duration

2) Small doses have a potent effect early in treatment, but larger and larger doses are required with time, until even large doses have little effect.

3) This fading response is due to progressively increasing intervention of the **adrenals** in defense against a factor which is able to influence the body balance.

E) **Radiation** has biological effects identical to polyunsaturated FA (anti-anaerobic).

1) PUFA potentiate radiation at all levels of organization.

2) Anti-FA (AFA) antagonize the effects of radiation.

3) Adrenal FA induce shrinking of the thymus, spleen, and lymph nodes, and lymphopenia; and there are similar effects from radiation.

4) Radiation increases FA, and, stimulates the adrenals to release FA, thus potentiating the radiation. The adrenals also intervene with AFA <u>against</u> conjugated FA.

II) Lipids with positive polar groups:

A) **Glycerol:** Inactivates FFA present in abnormal conditions by neutralizing their polar groups, and eliminates FFA by turning them into caloric metabolites.

B) **Glucose**: Same effects as glycerol, probably due to glyceric aldehyde and glyceric acid which appear in glucose metabolism.

C) **Glycerophosphoric** Acid: Binds FA to form phospholipids in membranes.

1) Di- and tri-ethenic FA are preferentially bound when they pass from the intestines into the circulation.

2) Anti-dysaerobic effect, especially when urine pH is paradoxically alkaline in the presence of high specific gravity and low surface tension.

3) Increases thyroid activity, resulting in increased BMR, and increased work capacity.

D) **Sterols**: Absorbed and circulated bound to PUFA. They are found as esters and as free sterols in all tissues (except brain and RBC = free only). They are anti-dysaerobic.

E) **Corticoids:** All corticoids are antagonistic to FA, yet mineralocorticoids are in some ways antagonistic to glucocorticoids.

1) Adrenals are very rich in arachidonic acid (greater than 25% of its FA), which is found in only small quantities in other organs (pent-enes and hex-enes are also greater than in other organs).

2) Adrenal corticosteroid release is especially stimulated by conjugated trienic FA related to trauma and by alpha hydroxy FA related to microbial invasion.

3) Administration of glucocorticoids increases resistance to the noxious effects of FA. Mineralorticoids have less effect; NaCl (though important to the adrenals) has nil.

F) **Peroxidases:** Hydroperoxides are formed under normal physiology from oxidation of UFA, but peroxides appear only under abnormal conditions associated with Dysaerobic dominance (especially when abnormal conjugated FA are present). Peroxidase (pears and horse radish) will exacerbate an Anaerobic Imbalance, but, have no effect on Dysaerobic dominance.

G) **Antioxidants** (e.g. alpha tocopherol): oppose abnormal FA (anti-dysaerobic).

Physiological Activity of the Elements

Each element has its own level of biological organization at which it acts when combined into specific compounds suited to physiologic activity at that level. (The various levels include sub-cellular, cell, tissue, organ/organism.) An element acts at a level other than its own only in other compounds and only when its availability and proper combination is abnormal at its own level.

Thus, two factors determine the activity of an element:

1) Its availability

2) The possibility to enter into its proper combinations

All abnormalities in the activity of an element can be related to these two factors.

A deficiency of an element's function at its proper level of organization is accompanied by an increased concentration at the next higher level. This is a compensatory increase. Excess activity of an element at its proper level of organization is accompanied by a decreased concentration at the next higher level. This is a compensatory decrease. For example, potassium is Anaerobic at the cellular level. An Anaerobic Imbalance is accompanied by a potassium increase at the cellular level, and a decrease at the blood level; while a Dysaerobic Imbalance is accompanied by a decrease at the cellular level, with an increase in the serum. (See the chart, below)

A **qualitative** excess or insufficiency, while representing a biological defense on the part of the higher level to combat abnormality at the lower level, **still represents a problem** in itself for the higher level. A good example of this concept is cholesterol, which is Anaerobic at the cellular level. A qualitative insufficiency of cholesterol at the cellular level will result in a compensatory increase of cholesterol in the blood. You are well aware of the pathologies associated with high blood cholesterol.

An element when acting at a level **lower** than its own has an effect **opposite** to that at the proper level (Anaerobic/Dysaerobic). For example, sodium is anti-dysaerobic at the tissue level, yet anti-anaerobic at the cellular level. Magnesium is anti-anaerobic at the tissue level, and anti-dysaerobic at the cell level.

Analysis of Insufficiency/Excess of an Element:

AMOUNT OF ELEMENT AT PROPER LEVEL	AMOUNT OF ELEMENT AT HIGHER LEVEL	INTERPRETATION
High	High	Quantitative excess
Low	High	Qualitative insufficiency: compensation at higher level for poor utilization due to inability to form active compound at proper level.
High	Low	Qualitative excess utilization: attempt at higher level to control excess utilization by decreasing availability
Low	Low	Quantitative deficiency

Anti-Anaerobic Elements (activity parallels FA)

1) Cellular level

   a) Calcium

   b) Manganese

   c) Selenium

   d) Copper

   e) Cobalt

   f) Germanium

   g) Vanadium

2) Tissue level

   a) Magnesium

   b) Silicon

   c) Sulfur

3) Organism level

a) Oxygen

4) Sub-cellular levels

a) Tin, lead, strontium, silver, gold. These heavier elements have the greatest anti-anaerobic effect, **but** have minimal effect on already existing clinical problems (which are already manifest at higher levels of organization).

Anti-Dysaerobic Elements (activity opposes FA)

1) Three mechanisms of AFA activity:

a) Halogens are AFA via inactivation of the FA double bonds

b) Other AFA elements bind the FA positive polar group

c) Others are AFA indirectly through the metabolic processes in which they take part

2) Cellular level

a) Potassium

b) Zinc

c) Chromium

d) Iron

e) Nickel

f) Bromine

g) Arsenic

3) Tissue level

a) Sodium

b) Chloride

c) Phosphorous

d) Aluminum

e) Oxygen (in large quantities)

4) Organism level

   a) Lithium

   b) Fluorine

   c) Boron

5) Sub-cellular levels

   a) Iodine, mercury, bismuth, molybdenum, rubidium, caecium. These heavier elements have the **greatest** anti-dysaerobic effect, but have minimal effect on already existing clinical problems (which are already manifest at higher levels of organization).

Physiological Activity of Common Therapeutic Agents

When you have patients on medication it is essential to know whether that medication is compatible with their Anaerobic/Dysaerobic Imbalance. If a medication has been prescribed which is exacerbating the Anaerobic/Dysaerobic Imbalance, serious consideration should be given to having that medication changed.

Anti-Anaerobic Therapeutic Agents:

1) Testosterone
2) Epinephrine
3) Atropine; quinine (alkaloids); caffeine & theobromine?
4) Penicillin, streptomycin, aureomycin, sulfas (all antibiotics)
5) Acetaminophen (Tylenol), amenopyrine, antipyren (analgesics and antipyretics)
6) Chlorophorm
7) Benzene
8) Digitalis
9) Radiation
10) Ibuprofen; Naproxen

Anti-Dysaerobic Therapeutic Agents:

1) Estrogen
2) Progesterone
3) Cortisone, prednisone, desoxycorticosterone
4) Insulin
5) Codeine; morphine (alkaloids)
6) Acetyl-salicylic acid/aspirin/ascriptin
7) Phenobarbital; pentobarbital
8) Aminophylline; theophylline; caffeine & theobromine?
9) Procaine; Novocain; Glycerine; Sodium gluconate
10) Benzedrine, dexidrine (amphetamines)
11) Barbiturates
12) Demerol

Medication may be valuable for its ability to control severe symptoms. But it is essential to know what secondary metabolic effect will be produced by the medication. To illustrate, let us look at a common everyday occurrence, the use of Ibuprofen to control pain. Ibuprofen has anti-anaerobic activity. If the person in pain has an Anaerobic Imbalance, Ibuprofen is likely to effectively and harmlessly relieve the pain. However, if the person is Dysaerobic, Ibuprofen may provide minimal pain relief, while exacerbating the Dyserobic Imbalance, which will actually increase pain sensitivity, and will likely create the harmful side effects associated with Ibuprofen such as gastric bleeding.

With the above information you can guide your patients intelligently in their use of medication. Furthermore, NUTRI-SPEC procedures give you a system by which to monitor the metabolic effects of the medications, so that they can be stopped before creating side effects, or, can be safely increased to control symptoms.

Endocrine Relationships

There are several endocrine and exocrine secretions which have a specific effect on Anaerobic/Dysaerobic balance. They are as follows:

A) Anti-Anaerobic:

1) Epinephrine (Adrenal Medulla)

2) Testosterone

3) Bile

4) Pancreatic Enzymes

B) Anti-Dysaerobic

1) Estrogen

2) Progesterone

3) Adrenal cortex (especially glucocorticoids) (hypoglycemics are often Dysaerobic)

4) Anterior Pituitary (ACTH and GH)

5) Pancreas (Insulin) (Type I diabetics are often Dysaerobic, yet just as often test Anaerobic due to their insulin injections)

Clinical Manifestations of Anaerobic/Dysaerobic Imbalance

What does a patient with Anaerobic/Dysaerobic Imbalance look like walking into your office? That person can look like anything and everything you have ever encountered in your clinical experience. As we described earlier, this fundamental balance is associated with phenomena that define life itself, namely, membrane permeability and energy production. There probably is not a single clinical condition that does not involve Anaerobic/Dysaerobic Imbalance to some degree; and in many conditions Anaerobic/Dysaerobic Imbalance is the primary underlying cause.

Clinical manifestations of Anaerobic/Dysaerobic Imbalance are the result of imbalance at the tissue and organism level.

Many clinical symptoms can be the result of **either** Anaerobic or Dysaerobic Imbalance. Some examples of symptoms whose occurrence follows <u>a dualistic pattern</u> are pain, itching, vertigo, hearing loss, dyspnea, and manic depression. These symptoms are associated with the pH changes typical of Anaerobic/Dysaerobic Imbalance at the tissue level. Controlling the underlying imbalance is frequently as simple as controlling tissue pH.

We are now going to discuss pain in depth. Keep in mind, however, during this discussion of pain that the same principles apply to the other symptoms of a dualistic nature listed above.

When a patient is in pain, the pH of the lesion is opposite the pH of the urine (and system) due to metabolic aberrations involved with the lesion. FA and AFA influence pH, not directly, but indirectly by the influence of their non-polar group on local metabolism. Thus, for example, FA have an alkalizing effect on the lesion rather than the acidifying effect of most other organic acids (and they have no effect on the pH of normal tissues).

The pH of painful abnormal tissue is much more labile and extremely sensitive to body pH changes. Induced acid-base variations produce very slight pH changes in normal tissues, but an exaggerated response in pathological tissue.

An Anaerobic pain is an acid pain. There is a systemic alkalosis and alkaline urine. However, at the tissue level (and especially in lesioned tissue) there is Anaerobic energy metabolism (glycolysis), which forms increased lactic acid in the interstitial fluid. This acidity causes a sensitization of nerves, resulting in pain.

A Dysaerobic pain is an alkaline pain. There is a systemic acidosis and acid urine. However, at the tissue level (and especially in lesioned tissue) there is Dysaerobic oxygen metabolism, which forms excess conjugated FA, and increased fixation of chloride ions as they bind the FA double bonds. This excess chloride fixation allows sodium to remain free. Excess sodium combines with carbonate ions and forms alkaline compounds in the interstitial fluid. Excess sodium also enters the cells, causing potassium to be lost into the interstitial fluid to form more alkaline compounds. The alkaline sensitization of nerves results in pain.

The pain of traumatic wounds is always accompanied by an initial period of Dysaerobic/FA/catabolic dominance (usually lasting a few days) resulting in alkaline pain. This includes burns (and sunburn). It also includes post surgical pain. Wound healing, fractures, etc., are therefore retarded by FA/negative polar groups, and enhanced by sterols. (Sterols increase the depth of the scar in epithelial tissue wounds.)

Vertigo, itching, and manic depression are all, as stated above, also tissue level manifestations of Anaerobic/Dysaerobic Imbalance. They, too, require that you control tissue pH.

You will soon understand that virtually any symptom or complaint in any one of your patients can be associated with this fundamental metabolic balance system. High cholesterol and cardiovascular disease almost always have an Anaerobic or Dysaerobic component. Allergies almost always are associated with an Anaerobic or Dysaerobic tendency. Gastrointestinal function depends upon Anaerobic/Dysaerobic balance. Chronic fatigue, arthritis, and emotional problems are all frequent manifestations of Anaerobic/Dysaerobic balance.

If one of your patients is suffering from one of these or any of dozens of other maladies associated with an Anaerobic or a Dysaerobic imbalance – nothing you do for them will have a permanent therapeutic effect until you have corrected the <u>cause</u> of their affliction. Only by restoring normal oxidative energy production and restoring normal membrane function will your patient enjoy any lasting benefits.

Other clinical manifestations of Anaerobic/Dysaerobic Imbalance include:

A) Hearing loss

1) Tissue level dualistic pattern (commonly caused by Acetaminophen, Ibuprofen, Naproxen & Aspirin)

2) Can be helped if sclerosis is not yet complete

B) Dyspnea

1) Reflex control of respiration:

a) Lung parenchyma is stretched = stimulates nerve endings which (via the vagus) stimulate the respiratory center to stop inspiration and begin expiration.

b) The involved nerve endings are stimulated by chemical as well as mechanical changes.

c) Edematous tissue has alkaline pH (Dysaerobic/FA).

d) Fibrotic or lymphoid tissue has acid pH (Anaerobic/AFA).

C) Arthritis

1) Arthritis pains follow a dualistic pattern, but the character of the pain frequently shifts from one pattern to its opposite, thus making pain control a difficult clinical challenge.

2) Rheumatoid Arthritis is most often Dysaerobic/Catabolic.

3) Osteoarthritis is most often Anaerobic/Anabolic.

D) Gall Bladder Colic

1) Usually Dysaerobic = often bilirubin in the urine

E) Connective Tissue

1) Inflammation that responds favorably to corticoids is Dysaerobic.

2) Glucosamine supplementation is effective in Dysaerobic connective tissue conditions.

3) Chondroitin Sulfate is effective in Anaerobic tissue conditions.

F) Psoriasis

1) Usually Dysaerobic

G) Hemorrhage

1) Oozing/Capillary Hemorrhage/Ulcerations/Arteriolar Hemorrhage (non-traumatic)

a) Anaerobic: responds to supplementation with FA plus Ca as aspartate or orotate.

b) Frequently aggravated by B1, B3, glucose, glycerol, and cholesterol. (Never give large doses of Vitamin B1 to an ulcer patient.)

2) Arterial Hemorrhage (Post-traumatic/Surgical/Burn)

a) Dysaerobic

b) Oxygenic D-Plus may be used <u>topically</u> for the first 3 days post trauma.

H) Schizophrenia

1) Usually Dysaerobic

I) Allergies (food allergies, asthma and urticaria)

1) Dualistic pattern with urinary Surface Tension or Specific Gravity as the best indicator of Anaerobic/Dysaerobic. The dermographics line is frequently the most useful indicator.

2) The answer to allergies is **not** avoidance. For patients to have a comprehensive set of allergy tests, receive a list of 20 allergens, then proceed to turn their lives upside-down in an attempt to avoid those 20 substances is an expensive and stressful exercise in futility. How many patients do just that, then upon re-testing at a later date find that the list of allergens has grown from 20 to 40? Avoidance is not the answer if you want to see allergy patients participate in a normal, productive life. The answer is to correct the underlying Metabolic Imbalances that are creating the state of constant **allergic tension.** The truth is, severe allergy cases, on a bad day, are allergic to **everything** – everything they eat, everything they touch, everything they breathe. Avoidance is obviously not the answer.

3) This is not to say that allergy testing is not a valuable clinical tool. If a person's throat is going to swell shut so that he cannot breathe whenever he eats fish, obviously he needs to avoid fish. Allergy testing can be effective in pinpointing the causes of the most severe reactions. But as far as avoiding a list of 20 substances, that is running away from the problem, not solving it. It is also perpetuating the problem by ignoring the ultimate cause.

4) Migraine = Dysaerobic imbalance with an allergic trigger (often food allergies).

J) Multiple Sclerosis

1) Auto immune disease set up by nervous tissue allergy.

2) Usually low Surface Tension: Dysaerobic

K) Viral and Bacterial Infection

1) Anaerobic: stimulated by positive polar groups

2) High levels of positive lipids (anaerobic/anabolic) in a growing child explains the frequency and intensity of childhood infections.

a) A virus increases its virulence by passage through a child. It then easily infects adults of the same family who were not susceptible to the infection before passage through the child.

3) Virus infections increase in hot weather (e.g., polio).

4) Viral and bacterial infections are Anaerobic – their occurrence and virulance is associated with excess sterols and other anti-dysaerobic agents. The body's defensive reaction against infections is dualistic, diphasic in nature, marked by vacillations

between a catabolic/dysaerobic phase and an anabolic/anaerobic phase. These vacillations continue until the pathogen is neutralized. A healthy person, who will only provide himself with sufficient rest when infected, requires no therapeutic intervention. Requires none, and in fact is usually inhibited in his recovery by such intervention. Most of us, however, are either unhealthy, or refuse to rest sufficiently. The result is to "get stuck" in one or the other phases, in which case therapeutic intervention, guided by objective testing to define the patterns of metabolic imbalance, is most helpful.

5) Cancer typically begins as an Anaerobic imbalance below the cellular level.

a) More than one in three people in the U.S. gets cancer.

b) Cancer becomes clinically significant as it progresses to the level of the cell (invasive).

c) By the time a diagnosis is made, the condition has progressed to the tissue level (painful).

d) Organ involvement is pre-terminal.

e) Systemic involvement is terminal.

f) May switch to Dysaerobic, particularly with therapeutic intervention (radiation, chemotherapy).

L) Intestinal Function

1) Large dose of oral (e.g. cod liver oil) or parenteral highly unsaturated FA will cause diarrhea; colitis.

2) Sterols and other AFA cause constipation.

M) Kidney Function

1) The **kidney only passes free water.** (Water molecules ungrouped)

a) Grouping of water molecules increases biological activity.

b) Water molecules are ungrouped in freshly distilled water; ungrouped in heated water; grouped in natural water; most grouping in melted ice.

2) FA (Dysaerobic) cause decreased urine output (associated with excess grouped water).

3) AFA (Anaerobic) cause diuresis (associated with excess free water).

N) Seizures; Convulsions; Coma

1) Usually Anaerobic: aggravated by Vitamin B1, and sterols (butter, cream)

a) Extreme aggravation by lipids with positive polar groups in combination with adrenal corticoids.

b) Mineralocorticoids have the opposite effect of glucocorticoids in this regard.

c) Convulsions are decreased by FA.

O) Cardiac Rhythm

1) Sterols cause tachycardia; extra systoles; auricular fibrillations.

2) FA cause bradycardia; decrease extra systoles; decrease auricular fibrillations.

P) Blood Pressure

1) Sterols induce hypertension

2) FA induce hypotension

Q) Oxygen Carrying Capacity of Blood

1) Increased by cholesterol (Oximeter = (98+ = Anaerobic)

2) Decreased by FA (Oximeter = 97- = Dysaerobic)

3) RBCs passing through the lung have high cholesterol and low FA, which enables them to fix oxygen. Upon circulating to tissues, the cholesterol decreases, (and the FA increases) so that oxygen is liberated.

R) Menstrual Cycle

1) Positive polar groups, especially sterols, suppress the cycle.

S) Age

1) Youth is typified by anaerobic/anabolic processes.

a) There is a higher percentage of positive lipids (especially sterols) (at the cell and tissue levels, **not** in the blood).

b) Anaerobic metabolism is induced by sterols. With the intervention of dehydrogenases, the abundance of H+ ions leads to a predominance of the kind of synthesis favoring anabolism.

c) Persistent anabolic processes after age 23 = mTOR- & Senolytic-mediated Endogenous Inflam-Aging

2) Aging prematurely is dysaerobic/catabolic.

a) Associated with an increase in negative lipids (especially FA) particularly at the cellular level. The opposite occurs at the blood and organism levels.

b) Cholesterol is low in the cells, and increased in the serum (as a compensation for a qualitative cholesterol deficiency at the cellular level due to cellular FA predominance). This explains atherosclerosis (Treat with anti-dysaerobic agents at the cell level, and/or anti-anaerobic agents at the tissue level).

c) Increased urinary surface tension with paradoxical dermographic red line.

d) Oxidative Stress free radical damage = Exogenous Inflam-Aging

3) Summary of Aging:

a) FA to Cholesterol Ratio increases in cells.

b) The ratio of serum cholesterol to cell cholesterol increases.

c) (In other words) aging is a dysaerobic/catabolic imbalance at the cellular level, with anaerobic compensation at the tissue level, with catabolic signs (= high Specific Gravity) at the systemic level

T) Sex

1) Females, in general, have a higher percentage of positive lipids than males.

a) Females are Anaerobic; males are Dysaerobic. (These are very general tendencies only; females often have Dysaerobic imbalances and males Anaerobic.)

2) Sexual intercourse

a) Increases FA activity (Dysaerobic) in males.

b) Increases sterols (Anaerobic) in females.

c) Changes are manifest on the organism level, i.e., in temperature, blood pressure, pulse, etc.

U) Muscular Exercise

1) Catabolic (Dysaerobic) during exercise; followed by a much longer anabolic (Anaerobic) phase.

V) Intensive Mental Exercise

1) Anaerobic

W) Diurnal Cycle

1) Most Anaerobic at 4:00 a.m.

2) Most Dysaerobic at 8:00 - 9:00 p.m.

3) Sleep increases sterols (anaerobic/anabolic).

a) Nocturnal polyuria; urine pH increases; surface tension increases; specific gravity decreases

b) Increase in acid pain (Anytime patients' symptoms worsen during sleeping hours t this is a clue that they have an Anaerobic Imbalance).

X) Environmental Influences

1) Summer: Anaerobic
Winter: Dysaerobic

2) Hyperthermia: increases sterols in the blood (Anaerobic)
Hypothermia: increases FA in the blood (Dysaerobic)

3) Applications of Heat: Anaerobic
Applications of Cold: Dysaerobic

4) Barometer Pressure Low: decreases serum potassium, increases cellular potassium (Anaerobic)
Barometer Pressure High: increases serum potassium, decreases cellular potassium (Dysaerobic)

Y) Hypercholesterolemia

1) Cholesterolemia is one of those conditions for which every empirical nutritionist has a favorite treatment. Some recommend PUFA, some recommend Vitamin B6, some swear by Vitamin B3, and so on and on. The truth is, these empirical approaches demonstrate very little understanding of the true nature of the problem. Cholesterolemia has a dualistic nature. In other words, there are two ways you can get it; either by being Anaerobic, or by being Dysaerobic.

2) Here are the facts: Cholesterol is a lipid of the sterol type, in other words it has anti-dysaerobic or anti-FA activity. Cholesterol is a normal, healthy component of the human body; its physiological activity is at the **cellular** level of organization. An Anaerobic person tends to have excess cholesterol activity at the cellular level. In time, the excess can be so large quantitatively that it exceeds the cellular saturation point and spills over into the higher level of organization, the blood. A Dysaerobic person, on the other hand, tends to have deficient cholesterol activity in the cells. As a compensation for this qualitative deficiency of cholesterol at its proper level of activity, a quantitative excess is maintained at the blood level.

3) Obviously, to effectively treat the high cholesterol patient requires objective testing to determine the underlying pattern of biochemical imbalance. In an Anaerobic patient the common approach of prescribing a low cholesterol diet with an increase in PUFA may be at least minimally effective, particularly if used in conjunction with other anti-anaerobic agents. In the Dysaerobic patient, however, this regimen will produce very little in the way of permanent results. The only long-range benefit will come from treating the patient as a Dysaerobic imbalance. Paradoxically, this will require the dietary recommendation for foods such as butter, cheese and eggs, which are high in cholesterol.

Anaerobic/Dysaerobic Analysis

Two essential tests to consider in analyzing Anaerobic/Dysaerobic Imbalances are the adjusted urine pH and the adjusted saliva pH. Why do these pH's relate specifically to this balance system?

Dramatic pH changes occur within and without the tissues of your Anaerobic and Dysaerobic patients. Anaerobic tissues are typified by excess anaerobic glycolysis. What is one major end product of anaerobic metabolism? **Lactic acid** is produced in excess quantities in anaerobic tissues. Because of the abnormal membrane permeability of the Anaerobic tissues, other acids accumulate as well. Thus, we have a powerful shift toward a tissue acidosis in your anaerobic patients.

At the systemic level, there is a valiant effort put forth to mobilize alkaline reserves (particularly bicarbonate) to protect against the acidosis. As a result, your Anaerobic patients present a paradoxical situation in which the tissues are extremely acid, yet there is a systemic alkalosis. This paradoxical pH imbalance is reflected in the high adjusted urine pH and low adjusted saliva pH.

Your Dysaerobic patient is exactly the opposite of the Anaerobic – there is a tissue alkalosis and a systemic acidosis. How does this occur? Remember, the Dysaerobic patient has excess fatty acids in the tissues. Not only are the fatty acids excessive in quantity, but <u>qualitatively</u> the tissues tend to produce abnormal, pathological fatty acids. The abnormal polarity of the fatty acid molecules causes energy centers which tend to bind chloride ions. The chloride ions bind at the site of the double bonds in the fatty acid molecule. This excess chloride fixation allows sodium to remain free to combine with carbonate ions forming strongly alkaline compounds. As in the Anaerobic patient, there is a compensatory reaction at the systemic level in a futile attempt to balance the excess alkali. This reaction explains the systemic acidosis and the low urine and high saliva pH typical of your Dysaerobic patients.

The next test to consider in your QRG is the specific gravity. Why does an Anaerobic patient tend to have low specific gravity? The answer lies in the effect of Anaerobic and Dysaerobic imbalances on both the kidneys and the GI tract.

An Anaerobic imbalance causes the kidneys to pull excessive quantities of water out of the blood stream for elimination. This explains the low specific gravity and the tendency to polyuria in your Anaerobic patients.

As you might expect, quite the opposite is the case with your Dysaerobic patients. The kidneys tend to reabsorb most water back into the blood stream, eliminating a urine which is extremely concentrated. Your Dysaerobic patients have high urinary specific gravity and a tendency to oliguria.

In the intestines, the Anaerobic cells tend to pull excessive quantities of water out of the intestinal lumen and into the blood stream. While that water heads straight for the kidneys for elimination, it leaves the intestinal environment somewhat dehydrated. This explains the Anaerobic patient's tendency to constipation. The Dysaerobic patient tends to dump excessive quantities of water into the intestinal tract, particularly in the duodenum via the common bile duct. This leaves the body somewhat dehydrated and causes a tendency to diarrhea in your Dysaerobic patients.

Dietary Recommendations

A) Anti-Anaerobic Foods:

The following foods should be **strictly avoided** by Dysaerobic patients:

1) Vegetable oils

   a) Unsaturated Fatty Acids

2) Fish oils

   a) Rich in PUFA (four or more double bonds)

3) Fried foods

   a) FFA are split off from fats cooked at high temperatures

   b) [See Caution in 6) a) below]

4) Preserved/processed meat and fish (e.g., canned tuna)

   a) FFA are split off from fats during high-temperature processing

5) Nuts, seeds, avocados

   a) Unsaturated Fatty Acids

6) Caution: The use of foods rich in fatty acids for their anti-anaerobic effect is efficacious only over a short time span due to the fading response of the body to fatty acids. In other words, the Dysaerobic effect of FA is neutralized as the body puts up an Anaerobic defense against them.

   a) Heating FA causes conversion of the natural cis isomers to unnatural trans isomers. While these trans isomers initially have a Dysaerobic effect, they ultimately stimulate the formation of three times the amount of sterol (anti-FA) esters as do their cis isomers. This means that fried foods are always immediately harmful to Dysaerobic patients even in small quantities, and, that they are ultimately also harmful for Anaerobic patients when eaten frequently, or in a large portion.

The following foods are particularly beneficial to Anaerobic patients:

1) Natural water (spring/well); ice-water

   a) Grouping of water molecules

2) Fish

B) Anti-Dysaerobic Foods:

The following foods should be **strictly avoided** by Anaerobic patients:

1) Fermented foods (yogurt, sauerkraut, soy sauce, alcoholic beverages, vinegar, buttermilk, etc.)

2) Butter, cream, cheese

3) Fruit in large quantities; fruit and vegetable juices

4) Sugar (honey, etc.)

5) Tea (theophylline), coffee (caffeine), chocolate (theobromine)

6) Pears & horseradish (peroxidase)

7) Heated water; freshly distilled water

   a) Free/ungrouped water molecules

8) Eggs should be eaten in moderation only

9) Salt should be eaten in moderation only

10) Vegetable oils (other than olive oil) [See A) 6), above]

The following foods are particularly beneficial to Dysaerobic patients:

1) Eggs (poached or boiled, **not** fried)

2) Cheese

C) Other Foods

1) Meat, fish, fowl

   a) If fried, it must be avoided by the Dysaerobic patient.

   b) If excessively fatty it should be avoided by the Anaerobic patient.

   c) Meat does not pose a problem for either imbalance if the over-all diet is chosen according to A) or B) above.

2) Vegetables

   a) Mildly anti-dysaerobic (unless fried)

3) Grains

    a) May be eaten without affecting either imbalance

4) Dried Beans

    a) Anti-dysaerobic (unless fried)

D) Food Allergies

1) Foods may cause an Anaerobic/Dysaerobic response indirectly via an allergic reaction. It is not uncommon for the response to be opposite the Anaerobic/Dysaerobic imbalance typically associated with that particular food.

    a) For example: Cheese would be expected to cause an Anaerobic reaction; but if a patient is allergic to cheese, the allergic stress reaction may manifest as either an Anaerobic or a Dysaerobic response. In other words, the patient's fundamental imbalance will override any specific effects of the food in question.

Recommended Supplementation

Read through the lists of ingredients on bottles of Oxygenic A and of Oxygenic D. Ask yourself just what it all means. Oxygenic A gives your Anaerobic patient every vitamin, every mineral, every trace mineral and every adaptogen needed to reverse the causes and the effects of insufficient oxidative energy production. Similarly, Oxygenic D provides all the vitamins, all the minerals, all the trace minerals and all the adaptogens essential to reverse the causes and effects of free radical peroxidation.

Now consider your Oxy Tonic and Oxygenic D-Plus. These are the closest thing to magic you will ever find. While the Oxy A and Oxy D provide the nutrient raw materials, it is the Oxy Tonic and the Oxy D-Plus that drive the mechanism. By that we mean two things:

- The membrane permeability is repolarized such that fluid and solutes (including all the juicy ingredients of Oxy A or Oxy D) are able to flow to their site of activity.
- The spark is provided to fire up the metabolic engine – Think of the Oxy A and D as the fuel being sucked into a car's engine, while the Oxy Tonic and D+ are the spark that ignites the fuel.

You cannot correct your Anaerobic/Dysaerobic patients with Oxy A and Oxy D alone any more than you can make your car run by dousing the engine with a gallon of gasoline. The fuel has to be put exactly where it needs to be and must be energized. That is the role of your Oxy Tonic and D+. These are the therapeutic agents that make the typical clinical nutritionist look impotent compared to you and NUTRI-SPEC.

Consider that Revici tested literally hundreds of biologically active substances for their metabolic effects. Out of all those substances he selected the ingredients of your Oxy Tonic and Oxygenic D+ to use for their powerful and immediate impact on body chemistry. He used these two supplements **exclusively** for nearly 40 years as his sole means of controlling metabolic balance at the tissue and systemic levels.

Let us look just a bit closer. Consider Oxygenic D+. Glycerol has powerful AFA/positive polar group activity, thus exerting control over fluid and electrolyte dynamics, tissue pH, and neutralization of free radical peroxidation. Glycerol is just one ingredient of Oxygenic D+. The most powerful AFA/positive polar group effect is provided by its 4-carbon natural alcohol. Multiply the anti-catabolic antioxidant effect of Oxygenic Dit perhaps by 10 and that is what you have with Oxygenic D+. Oxygenic D+ is the ultimate anti-oxidant.

And what about Oxy Tonic? Here you have sodium combined with negative valence sulfur. The negative valence (thiosulfate) sulfur is the key to innumerable biochemical processes. It constitutes the active functional group in such diverse compounds as glutathione, cysteine, taurine, and methionine. Just think of it as promoting normal oxidation, while protecting against the tissue damage that accrues from insufficient oxidation and excess anabolism.

It is no exaggeration to say that you could build an entire nutrition practice on nothing more than the judicious use of Oxy Tonic and Oxygenic D-Plus (plus Activator to provide trace minerals plus Adaptogens and Rejuvenins, along with the ideal Immuno-Synbiotic to build healthy microbiota).