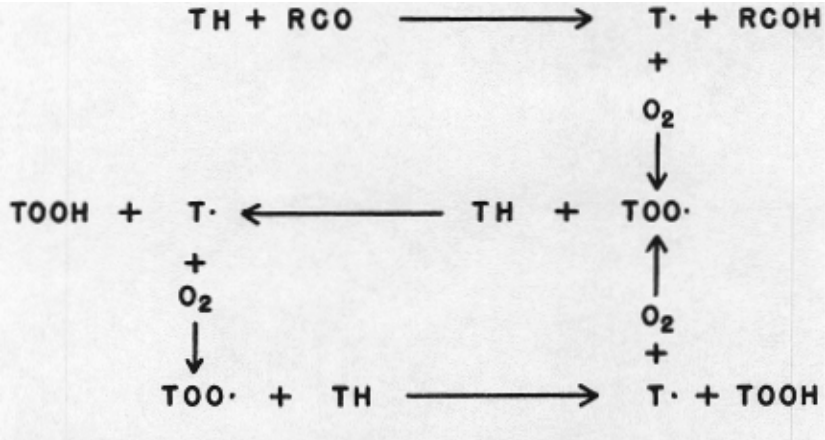
CHAPTER 6

ENERGY PRODUCTION

In order to explain the therapeutic procedure and reasons for certain features controlling the care of the patient, a consideration of the energy producing mechanism will be helpful.

We do not know what energy is, but we can differentiate several forms, and measure them in various ways. It is the consensus that all energy produced in the cell, whether by oxidation — namely the Krebs tricarboxylic acid process—or by fermentation, is the same and is stored as ATP (Adenosin triphosphate) high-energy bonds, before it is transferred to the working elements of the cell to be transformed into the energy of work. No other mechanisms of energy production are recognized, simply because no intermediaries identifiable with any other processes have been encountered. However, there is plenty of room for the operation of a far more efficient process than the Krebs Cycle, which indeed is a decarboxylation process nicely adapted to the lower forms of life. The clinical data show that the Krebs system does not fulfill the requirements of the oxidations that maintain health and that some other process of higher efficiency is present that provides the Survival Factor we have identified and reproduced for four decades for clinical use. The intermediaries of this High Efficiency Process are not to be trapped. They constitute the “smokeless flame” that supplies the energy as a preferred process. This is our Postulate that will be supported by practical proof later.

Ochoa and others have calculated that the combustion of a gram mol. of glucose in the tissues yields 450,000 calories of energy as the total from the various steps of the Krebs Cycle. This gives 36 (38) high-energy phosphate bonds with a P/O ratio of three. The free energy ^ F of glucose is, however,—691,000calories, and the energy of combustion ^ H is —673,000 calories. Thus, 18,100 calories are consumed in the process. The energy calculated as —450,000 calories from the Krebs process of oxidations is therefore —220,000 calories shy of the —673,000 calories available for work, and that are not accounted for in anyway. Therefore, some other process of higher efficiency than the Krebs Cycle has plenty of room to operate. Further, the highly inefficient Krebs process (65%) offers no protection against pathogens as it provides no O/R potentials high enough to start their combustions, but supports viral and neoplastic processes instead. It thus does not account for the survival oxidations that are clinically demonstrated. This process we identify with the FCG dehydrogenations that start a chain of oxidations via the free radical formed and its addition of oxygen to produce a peroxide free radical as carrier of the process. In free circulating toxins the reaction may be pictured thus:



Where TH is the toxin, and RCO the Survival Reagent Carbonyl Group.

In combined or integrated toxins, the most mobile hydrogen atom is alpha to a double bond in the most activated region which would be adjacent to the position of integration with the FCG or its activating double bond. Free radical production where this hydrogen is removed and addition of molecular oxygen to form a peroxide free radical causes cleavage into two parts with Carbonyl terminals. The azomethine double bond is broken, and the nitrogen oxidized away. The double bond of the new Carbonyl groups formed by the cleavage activate alpha positioned hydrogen atoms where further dehydrogenations repeat the process forming more peroxide free radicals that are the best dehydrogenators and carriers of the oxidation chains to new molecules of fuel or toxin that enter the field. All of the peroxide free radicals serve destructively to toxins therefore and constitute what we referred to as the antitoxic state of structure of the toxin after the Synthetic Antitoxic Reagent worked on it. Koch, *"Cancer Journal,"* October 1924; Philadelphia; *"Koch, Investigations,"* 1924. Here the process was described as converting toxin into antitoxin.

Our Postulate required that the FCG is activated by conjugation with the double bonds of an ethylene linkage from which it receives electrons. These two atomic groups are the center of romance in the maintenance of function and in the protection against toxins that enter the field. They are also the centers of attack of pathogens in viral, neoplastic and degenerative diseases. Whether or not a pathogenic response is to be had will depend upon:

(a) the presence or absence of a normal quota of molecular oxygen, and

(b) upon the firmness of condensation of an amine with the FCG, that guarantees or destroys its potency.

In case the normal range of energy production cannot cleave the bond, FCG function is destroyed and disease results. Then a superiorly efficient dehydrogenator Carbonyl group must be supplied to burn off the pathogen as previously stated. This restores the FCG for normal function. This super dehydrogenator is our Therapeutic Synthetic Survival Factor, (SSR).

The adequate oxygen supply plays two parts:

(1) it is the ultimate electron acceptor when the hydrogen atom removed from fuel or toxins by FCG is transferred to some oxidase system so the FCG is free to start new oxidations, and

(2) after the fuel or toxin is dehydrogenated to become a free radical, molecular oxygen must be at hand to convert it into a peroxide free radical to continue the oxidation process.

Otherwise the free radical would under hypoxia add to the closest reactive group that would accept it, and this is the double bond of the ethylene linkage that activates the FCG. The pathogen would thus integrate with the host cell's energy producing mechanism where it would draw off energy for its own vegetation or to transfer ectopically, and produce disease, and at the same time block the activation of the FCG and stop further FCG dehydrogenations. Thus oxidation is blocked and the consequent colloidal degenerations would follow to produce further anoxia. Thus anoxia is essential to the integration of the pathogen with the grana when the FCG is still operating. However, the moment the pathogen is added to its ethylenic activating double bond, electrons are no longer contributed to it, so the FCG is no longer able to dehydrogenate, and the grana appears to be out of commission, destroyed and lost. Ectopic uncontrolled transfer of energy to various secreting and contractile or conducting functional systems referred to above we hold to be the cause of allergy.

Since creatine did not interfere with FCG function as did guanidine, and since it is the only amine possibly that forms high-energy phosphate bonds, it was easy to assume that it played a role in the transfer of energy produced from the oxidation of fuel to high-energy phosphate bonds as of ATP. It would accommodate this transfer by condensing with the FCG to form an azomethine bond until enough energy had been generated to admit phosphoric acid into the bond and unite it with the amine group of creatine sending the creatine phosphate off as a high-energy carrier. The FCG is thus free to start further dehydrogenations physiologically. However, if the amine condensed with the FCG forms

a tight bond not separable under normal ranges of energy production, as did guanidine in the parathyroid experiments, the whole train of pathological events must follow. For this reason it is well to inquire into the sources of such pathogenic amines. One is the production of toxic amines in the acid colon by various bacteria that decarboxylate amino acids. In many people, the intestinal flora is firmly entrenched and converts the food into one's poisons that serve as the vanguard of disease. Animal proteins are the main sources of these toxic amines, and sulfides, while vegetables, cereals, and fruits supply plenty of protein and at the same time do not support decarboxylating germs. The intestine must be kept at a range of pH above 7, since the decarboxylations progress best at a pH of 3.5 to 6 when mediated by the Streptococcus fecalis and so many others.

The fungus found always in cancer is an amine producer that could initiate the pathogenesis as explained above, with its whole train of symptoms. And the modern antibiotic amine poisons, especially those that attack the liver and cause suspensions of consciousness like the sulfa drugs, and any in fact, are to be scrutinized with great suspicion as the cancer death rate his increased so greatly since they have become so widely used. Sulfides and sulfhydryl derived from food add to the double bonds that activate the FCG and thus block its activation powers. The intestinal flora again is to be considered with the diet if one is to maintain a normal function of the FCG as an energy producer and protector against pathogens**.** Especially during the Treatment period, when a dehydrogenator Carbonyl group of highest efficiency has been administered, one must protect the oxidation progression that follows from being blocked, as can take place through permanent free radicals as the oxides of nitrogen. Gas anesthesia should never be used in connection with this Therapy. Highly polar double bonds can also add to and quench the free radicals of the recovery process and block it, so certain terpenoids and even perfumes, and especially acrolein and polymerizing acrylic aldehydes from frying pans, are to be avoided in this regime. The proofs of our Postulate are serious practical facts.

Some medications absorbed into the tissue colloids may alter the steric set-up so that the remedial Carbonyl group which ordinarily could attack the hydrogen atom to be removed perpendicularly to the plane of the conjugation of its carrier carbon atom with the double bond that activates it, now finds a distortion that hinders this line of attack. Opiates and coal tar drugs, and especially aspirin, appear to interfere in this way.

The atomic set-up of the Reagent itself that carries the Super-high-efficiency Carbonyl group must offer a steric advantage in each disease where it is applied. For example: in Hog Cholera, diphenoquinone proved 100% efficient in several epidemics, while it proved 100% worthless in Rabies, and the serial system of Carbonyl groups used in Hog Cholera proved 100% worthless, while it was 86% efficient in Rabies in terminal cases. Both diseases kill 100%, within 3 to 5 days. Rabies is neurotropic always and Hog

Cholera rarely before the terminal hours. Our search has been for a molecule carrier of the Survival Efficient Carbonyl group that is equally applicable in all diseases where drug interference has not modified the steric set-up. This will be discussed later on. It will be seen, however, that to identify the high efficiency oxidation system, we consider normal to the cell, as of the same order as the therapeutic substitute used to rescue the FCG and restore normal function and structure, a few comparisons will have to be made. For example: that they are of the same order is seen in being blocked by the same agencies as anoxia, sulfhydryl, and that their processes are blocked by permanent free radicals, highly polar double bonds, etc. Likewise, the restitution program, that follows the freeing of the FCG system of its pathogen by the Synthetic Reagent, is the normal process. So the Synthetic Reagent fits into the mechanism with equal grace, as did the FCG before it was attacked by the pathogen. Likewise, since recoveries from viral and neoplastic diseases that could never be combated successfully before are accomplished by the natural resources of the body after the Synthetic Survival Reagent is used, they both fit the cell chemistry, but each in its respective capacity for survival. Since the recovery mechanism includes cyclic reactions at definite periods never seen in medicine before, just as the cure of the pathologies involved were never seen before, a deeper grasp on tissue physiology is made possible and a wider range of expertness can be acquired. Any successful clinician will recognize that expertness in this Therapy depends upon study and experience, and some new viewpoints must be adopted. To illustrate let us review a few toxic cases.

These cases show the characteristics of the recovery process, which itself gives evidence as to the nature of the etiological factor. Only the most pertinent data is used. It will be seen that each case presents a long pretreatment control period that definitely established the downward trend of health with the steady and often rapid advance of the diseases. Thus the best possible control for comparison of pretreatment and post-treatment progress was followed, and no confusing variables were permitted, as for example, other medications or treatment measures. Likewise factors that interfere with recovery were eliminated, so that the contest lay plainly between the Therapy, the patient's cooperation, and physical advantage on the one hand, and the disease forces on the other. The Remedy is named the Synthetic Survival Reagent (SSR). There are two forms, the Quinone form which when used is so named, and the Carbonyl group chain form with free radical terminals. This is simply called the Synthetic Survival Reagent (SSR) or given a similar appellation. The quinone dose is two micrograms, and the SSR, two micrograms, millimicrograms and micro micrograms in water, given intramuscularly or under the skin.

With few exceptions, the case records are taken from Federal Court and Federal Trade Commission Testimony, where they were proven factually uncontradictable. Some of the exhibits have been reproduced for use in this book. This policy was adopted to give the student full confidence in the proofs of a Thesis as unusual as this one.

A case of toxic nodular goiter illustrates some of the main features of the oxidation mechanism of our Postulate.