NEOPLASTIC AND VIRAL PARASITISM THEIR BASIC CHEMISTRY AND ITS CLINICAL REVERSAL

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The research that is being offered now was started 50 years ago with the writer's discovery that complete parathyroidectomy resulted in the production of fatally toxic quantities of guanidine and methyl guanidine in the tissues and their excretion in the urine. **(1)** Three years later Paton of Glasgow fully confirmed this finding. **(2)** We wish to show now that the data gathered from this study led to a clinically useful concept about energy production in the tissue cell and its transfer to the functioning elements to support contraction, secretion, conduction and cell division. The atomic exchanges and the electronic displacements that take part appear to follow a pattern of construction and activity which one identifies in the normal functioning mechanism and also in the natural pathogens and which accounts for the properties of viral parasitism. **This pattern also guides the construction of the agent used to reverse the pathogenesis and correct the parasitism in viral and neoplastic diseases -- a sort of a Least Common Denominator.** The facts and conclusions were assembled at first as a Postulate but, now that they are confirmed by their clinical utility, they form the basis for a new Therapy. **This research did not follow the usual lines of thought that govern current therapeutics, for it is based on the properties of the free radical, the double bond and the Carbonyl and azomethine groups.** Such matters have not interested medicine heretofore, though they are the fundamental chemistry of the great industries including the plastic industry. But now (June, 1961) **that Szent Gyorgyi suggested that future cancer research might do well to learn if the "lone electron" (free radical) played a part, a general interest in the science that is basic to our Thesis has been created and we are, therefore, obligated to make this disclosure.** This report should stimulate or interest biologists and physicians to broaden their approach when meeting their unsolved problems.

First let us consider what complete parathyroidectomy revealed as a guide to this study. In addition to the guanidine bases, excessive amounts of lactic add, phosphate and calcium were eliminated in the urine in spite of forced ventilation of the lungs. It was, therefore, apparent that fermentation supplanted oxidation as a source of energy for function. The autopsies showed extensive ante-mortem dotting of the blood in the large veins and in the tissues and a hemorrhagic glomerulitis and hepatitis. After kidney and liver functions were destroyed, the convulsions were more severe and soon proved fatal.

We attribute the high toxicity of the guanidine structure to its activated amine group as conjugated with the imide group by which it was relieved of a quota of inactivating electrons. The reaction we are most interested in here is the ability of an activated amine group to condense with a Carbonyl group to form an azomethine double bond, which physiologically serves as an "avenue of electrons" or bridge for energy transmission, in our opinion, and is readily formed or split by hydrolysis at physiological

levels. Pathologically, the bond is stable against hydrolysis at physiological levels and, hence, interferes with important Carbonyl functions. Though it has attracted little attention in biology it is present, once or more often, in most of the vital molecules in tissue chemistry and recently is assigned a place by Braunstein and Kritzman in the co-factoring of transaminations by pyridoxal phosphate. **Its remarkable properties have fulfilled the requirements of our Concept, long before it was considered in biochemical processes. It is now known that guanidine destroys the Pasteur Effect.** This is what happened in the parathyroidectomized dogs and **our Thesis claims that the *Pasteur Effect is governed by the Carbonyl group that initiates oxidations for energy production.* (3) *This group we designate the Functional Carbonyl Group of energy production, the FCG, which is inactivated by condensing with guanidine, whereby energy production is blocked.***

On the other hand, the amine group of creatine, methyl guanidine, acetic acid, is also activated by conjugation with the imide group of the guanidine fraction, but because of the influence of such substituents as methyl and acetic acid, its condensations with Carbonyl are very liable to hydrolysis, so it is not toxic and can serve important physiological functions. We assign it an accessory place in carrying out the Pasteur Effect.

The ante-mortem clots in the large veins showed that fermentation and the Krebs Cycle oxidations progressing in the tissue fluids were not sufficient to charge the surfaces of the blood and tissue colloids for good dispersion, so the blood jelled in consequence. It must be recalled that an ante-mortem clot is not a true fibrinous clot, **but a jelly-like change due to lack of electric charges on the colloidal surfaces and indicates a defect in the energy production by the oxidation mechanism, aside from energy carried by ATP.** Jelling of the blood in the coronary or cerebral vessels blocks the circulation in the early part of an apoplectic stroke or coronary occlusion attack not caused by direct embolism. In such instances, the oxidations within the functional elements that provide the energy for good dispersion of the colloids are blocked. In the parathyroid studies, guanidine was the blocking toxin and its injection in toxic doses produced all the changes that follow parathyroidectomy, including ante-mortem clots. ***Because of the properties of the activated amine group and the evidence that the tissue oxidations were blocked, we concluded, that the functional oxidations are initiated by dehydrogenation accomplished by highly activated Carbonyl groups of the functional mechanism -- the Functional Carbonyl Group (FCG).***The activation, we concluded from additional observations, was **due to its conjugation with electron contributing double bonds of an ethylenic linkage,** which would lose this power by making additions with free atoms or radicals.

Because of clinical encounters, it was necessary to assume that a Functional Carbonyl Group received the energy produced by the oxidations initiated by the FCG of energy production, for if this FCG of energy acceptance was blocked bycondensing with a firmly binding amine, the functional elements would be under energy starvation and function would be blocked just as much as if energy were not produced. So we **Postulated that if the energy evolved at the FCG of energy production could pass via an azomethine bridge into the mobile electrons of an amine rich in double bonds and this amine could split off from the FCG at physiological levels, it could carry its energy to the FCG of energy acceptance and release it for function.** For clinical purposes this is all we needed to assume to explain the ***two possible types of functional block*** -- that is due to ***failure to produce energy,*** and that is due to ***failure to pass the energy into the functional units.*** The blocking would be by firm azomethine condensation, or by an addition of a free radical to the activating double bonds of either FCG, as previously stared.

After 1930 when Lohmann exposed the function of creatine phosphate **(4)** and Lundsgaard showed its relation to muscle contraction, it was easy to fill in our Thesis as follows, especially after Englehardt **(5)** demonstrated in 1939 that ATP (adenosin triphosphate) gave up its energy to support the contraction of muscle fibrillae. ***We assumed that the FCG of energy production dehydrogenated fuel or pathogens that entered its field. As soon as the acquired hydrogen***

atom was removed by some cofactor, the FCG condensed with the amine group of creatine to pass energy into its mobile electrons, until it was sufficiently activated to split the azomethine bond by adding to phosphoric acid and thus liberate the FCG to start another cycle of oxidation. It is now accepted theory that creatine phosphate passes its energy on to ADP (adenosin diphosphate) to form ATP and the latter hands it over to the functional elements for work and growth. ***We then sharpened our Thesis by considering the exposed amine group of the adenine unit of ATP (which is activated by conjugation with a series of 4 sets of conjugated double bonds) as the group that condenses with the FCG of energy acceptance to pass the energy into the functioning elements.***

One and the same FCG could perform both functions in rapidly alternating succession **and thus account for the undulations estimated to run at some 30,000 per second when an impulse is conducted along a nerve fiber.** We will discuss them as separate entities, however.

FCG blockage explained the ante-mortem coagulation of the blood in the parathyroid experiments. It **explains the jelling of the blood** in the early phase of an apoplectic stroke or coronary attack, **for in both the administration of a Carbonyl group of high oxidation potential corrects the jelling of the blood** so it flows freely and thus prevents the degeneration of the vessel wall that would lead to true thrombosis and infarction. Case histories will be given as examples, both of the block to energy production, as in coronary occlusion, and of the blockage in energy acceptance, as in exophthalmic goiter, even after the pathogen has integrated with the functional mechanisms concerned, the FCG systems of energy production, and the mechanisms of energy acceptance. **When the normal tissue's FCG has failed to dehydrogenate an amine pathogen, as when its O/R potential was too low, the firmly binding amine group must block FCG function. If because of anoxia the free radical formed in the pathogen, by its dehydrogenation at the hands of the FCG, has no oxygen to combine with it then adds to the activating double bond conjugated with the FCG and blocks its function.**

Therefore, the absence of adequate molecular oxygen is able to prevent the free radical, so formed, from becoming a peroxide free radical to be further burned. It would add to the activating ethylenic linkage of the FCG and thus block FCG function. Therefore, ***two circumstances are basic to the pathogenesis (a) the presence of a firmly binding amine structure and (b) the presence of anoxia; either, can block the FCG function to produce disease, or prevent the use of ATP for tissue cell development.*** **The restoration of the growth process in hindered children of many classes and the reconstruction of tissues destroyed by cancer or by diabetic gangrene,** ***demonstrate that restored FCG function can again use ATP not only for the primary cell functions, but for growth as well.*** Case histories will illustrate.

The relation of the FCG to gene structure and function is thus opened for study. **The destructive effects of irradiation on function and structure are another study in which we have collected important data, which must be reported some day in the interest of radiologists who have sustained injury, and of atom fission chemists who have been diseased through professional exposures.**

INHIBITION OF FCG FUNCTION

Anoxia is necessary to the blocking of the activating function of the ethylenic linkage. Warburg's Thesis **(6)** that anoxia is the cause of cancer, is supported here and one may add that it is necessary to certain types of viral integration with the host cell's functional mechanisms to produce paralysis. ***When the FCG dehydrogenates a pathogen, viral or chemical, that enters its field during hypoxia, the free radical formed cannot add molecular oxygen to become a peroxide free radical and be combusted, so it adds to the attracting pole of a double bond with which it has contact. That must be the proximal pole of the ethylenic linkage that activated the FCG that removed the hydrogen atom. Addition here will block all electron migrations to the FCG and the oxidation initiating mechanism cannot function any more. The deprival of the functional mechanism (grana) of***

oxygen does away with them structurally and functionally as Warburg's Thesis claims, however, we find that they are NOT eliminated from the cell. It is their identity that is lost and the change is clinically reversible by removing the pathogen. Warburg spoke only of cancer. **We include cancer and extend the observation to all other diseases studied to date.**

We will show that together, with the anoxia, *a co-factor is required. In cancer, it is a carcinogen (viral or chemical) and clinically, a polymerized product of bacterial action in a hypoxic focus of fibrosis carrying a silent infection.* The integration formed by the pathogen with the activating double bond of the FCG, or with the FCG by free radical addition or by firm condensation with an amine, respectively, is provided for by the activation of the position alpha to a double bond. This double bond is the electron withdrawer and the alpha activation also provides for the dehydrogenation of this position after integration of the pathogen takes place, so that cleavage of the pathogen from the FCG system with restoration of its Carbonyl group and of its activating double bond are had. *The host cell is thus separated from the pathogen in good functional status, while the pathogen is no longer to be found.* It undergoes a progressive oxidation favored by activation alpha to the terminal Carbonyl groups produced at each fragmentation. There are two means of securing this separation and they confirm our Thesis as to the nature of the pathologic integration. It will be seen also that ***one Corrective Reagent used is constructed on the same pattern and is essentially a highly activated Carbonyl group of a potential of one volt more or less according to the carrying structure which is built up to secure the greatest steric advantage for its particular attack, which is, of course, high potential dehydrogenation.***

Reversal of the pathogenesis is closely followed by tissue reconstruction. Thus, when Ehrlich ascites cells are transplanted into the peritoneal cavity of mice, the liver and spleen reticuloendothelial cells immediately atrophy and both organs shrink. The neoplastic cells infiltrate and produce tumefaction with ascites. Treatment can be given at various periods after inoculation and the animals sacrificed for observation. It is then seen that, as the neoplastic invasions are removed and the peritoneum becomes absolutely clear and glistening, the spleen and liver and especially their Kupfer cells regenerate rapidly. **If any traces of neoplastic invasions are found, they are undergoing nucleolysis, calcification, and coagulation as an early phase of digestion.** Such changes are similar to what we observed in the biopsy material taken from skin squamous cell cancer during recovery. *(Medical Record of New York,* October 1920). ***The initiating factors are, of course, the activated Carbonyl group of the Reagent and the activated position alpha to an ethylenic linkage in the integrated pathogen tending to release its hydrogen atom unrestricted.***

In reality, the ethylenic linkage is not an electron donor, but a weak withdrawer of electrons. When conjugated with a Carbonyl group, which is an active electron attractor, the ethylenic (pi) electrons are mobilized toward the Carbonyl group, and such substituents as CH3, CH2CH3, and C(CH3)3, which are active releasers of electrons will, when located at the opposite end of the double bond, supply their quota for attraction to the Carbonyl group of the FCG system. In addition, the Carbonyl group is negatively polarized with an oxygen atom rating 3.5 electronegative units and a carbon atom of 2.5 electronegative units. Only fluorine exceeds the electro-negativity of oxygen. **Therefore, the Carbonyl group of the FCG system as conjugated with an ethylenic linkage serves as an active dehydrogenator of fuels and pathogens that enter its field, and the ethylenic linkage serves as the bridge for the electronic migrations toward the Carbonyl group. *Where two or more Carbonyl group double bonds are conjugated in series, the orbital mechanics determine so heavy a concentration of electrons and electro-negativity at one of the groups that it becomes a most active dehydrogenator, and as in Triquinoyl, the strain becomes so great that one group even becomes expellable to form the more stable five member ring.***

In addition, fuels and pathogens are especially equipped to mobilize their critical hydrogen atoms. In glycogen and the polysaccharides, the Carbonyl groups are inactivated and in the monosaccharides, the lactone structure makes the molecule inert. When the Carbonyl group is free, however, it attracts the electrons away

from the hydroxyl group so that its hydrogen atom tends to be liberated unrestricted. This mobilization is seen when glucose or fructose is dissolved in heavy water. Here it is found that the hydrogen atoms trade places freely, and at random with the deuterium of the heavy water. Such mobility is surprising in view of the fact that the bond energy of the O-H group is one of the highest of the covalent bonds; namely, 110.2 Kilo-Calories and the bond length is one of the shortest; namely, 0.95 A units. ***Thus one sees the power of mesomeric induction to bring about reactivity without causing ionization.***

Pathogens and unsaturated fats also invite dehydrogenations in various degrees. Here we **Postulate that a of an *ethylenic methylene group positioned alpha to a double bond linkage offers two activated hydrogen atoms; one is important for the integration with the FCG system during the anoxia and the other invites its removal from the integrated pathogen by the Carbonyl group of the curative reagent continuously when oxygen is present.*** (This dehydrogenation can also be accomplished by an appropriate free radical). The activation of the pathogen's hydrogen atoms is secured by withdrawing electrons from the alpha placed methylene group by the substituents placed at the other end of the double bond. Those that withdraw electrons are halogens, methoxyl, hydroxyl, aldehyde, Carbonyl, vinyl, phenyl, cyano, and sulfhydryl, but not by amino groups. **Here one sees the possible place of iodine in activating the initiation of physiological oxidation.** The withdrawal of electrons from the alpha positioned carbon atoms weakens the bond to hydrogen and facilitates dehydrogenation. **The stage is thus set intrinsically for the oxidative reversal of the pathogenesis. *The pathology actually provides for its correction. The philosophic implications deserve thought.***

TWO SEPARATION PROCEDURES

To cause the cleavage of the pathogen from the host cell's FCG system at the position alpha to the double bond of the pathogen that activated the integration, be it by an azomethine condensation or a free radical addition, one uses a highly activated Carbonyl group dehydrogenator. **The dehydrogenation thus brought about leaves a free radical, which when oxygen is present will add a molecule of molecular oxygen to become a peroxide free radical. This will cause the cleavage leaving a Carbonyl group to restore the FCG, or a Carbonyl group to replace its activating ethylenic linkage that formed the integration. *This exchange is to the advantage of a long lasting immunity or resistance of a higher order than the ethylenic linkage had offered formerly, because the Carbonyl group is a richer assembler of electrons than the ethylenic linkage and gives the FCG a higher dehydrogenating power that will start combustion in a wider field of fuel or pathogens that enter the field.*** This is seen in the Triquinoyl molecule we use as a therapeutic agent. The orbital mechanics of the six Carbonyl groups united in cycle determine such a heavy concentration of electrons at one of the groups that it is actually expellable from the group to make a five-member ring, leuconic acid. **It is the electronic saturation of this Carbonyl group that makes it a splendid dehydrogenating agent to serve so satisfactorily as a therapeutic Reagent.**

Warburg credited **anoxia with the power to produce cancer,** and ***while anoxia is essentially provocative, we have found that in addition, two carcinogenic agents are also necessary. They are the initiating and sustaining carcinogens.*** One and the same chemical structure may serve in both capacities, as will be seen in the diagrams that follow. However***, the initiating carcinogen or allergen may be: a virus, a product of germ activity in the intestine, a mercaptan or other structure carrying a sulfydryl group, or a free radical produced by the sun's rays on a ripening pollen, or a present in some plastic material.*** **The *sustaining allergen or carcinogen* is in our experience *a product of germ activity.* It is brewed in an old scarred in focus of silent infection where enough oxygen is not admitted to burn the free radicals produced in its slow metabolism, thus giving them a chance to polymerize. Both are joined to the energy receiving mechanism of the host cell by free radical addition, and hence, by a single covalent bond. The integration of a virus is with the energy producing mechanism and likewise by a single covalent bond, and *so the phenomena associated with viral infection and allergy, including cancer production as well as their reversals, are of the same pattern and may be*

discussed together. Though the single covalent bond is well known for its easy rotation under environmental influences, it appears fixed for each disease entity, a fact that gives a good clue to the nature of the critical atomic groups, both of the pathogen's and of the host cell's energy producing and receiving mechanisms.

A little more discussion should be given to the ***easy rotation of the single covalent bond***, and also **its ability to be fixed in one plane by mutual polar attractions and repulsions of component atomic groups in both the host cell and the integrated pathogen.** This rigidity exhibited by each species in each of its viral infections has been observed as a constant feature and would be the only explanation available, if we assume that the pathogenic integration takes place by an addition at one position in the host cell's FCG, and its activating double bond.

The additions of the two pathogens, the initiating and the supporting pathogens, cannot be formulated with exactness as the chemical structures are not known with exactness and we have arrived as far as we have by Postulates and check-up of each Postulate, all of which were based on sound chemical principles. **With this reservation in mind, we may also formulate the integration of both pathogens with the critical atomic group of the host cell's energy producing and receiving mechanisms, as directed by the polarity forces exhibited by the double bond and its substituents. This cannot be claimed to be absolute for we do not know the atomic groupings sufficiently for an absolute diagram.** However, any utility in a conclusion reached by postulate is just as good a utility as that reached by cold fact, for it is the utility we need now to face the cancer and viral plagues we fret about or are not willing to tackle. The utility of an explanation is some reward.

We have observed that hog Cholera fails in 100% of cases to respond to the serial system of Carbonyl groups that hog Aftosa, cow Aftosa, and rabies respond to very satisfactorily. Many epidemics of Aftosa in cattle have responded 100% to this Reagent. On the other hand, Aftosa does not respond to Benzoquinone nor does rabies respond to diphenoquinone to which 100% of hog Cholera responds inmore than one epidemic. ***So the species pathogen-integrate for each disease is set.*** A diagram in one plane can be given on paper only, and will have to be interpreted by the reader with reference to other planes. The substituent groups R, R' R" cannot be given in detail for they are not known. However, the signs will have to be understood to carry the polarity values that cause the fixation of the single covalent bond that joins the two parties, as we outlined before. **What we can show is how the polarity values of the critical atomic groups of the autonomous host and of the parasitic pathogen favor the pathogenesis and also the separation of the host's critical atomic group from the pathogen, which undergoes a stepwise oxidation.** There is, however, more than one question that is not answered by the diagram. Further data must first be won. ***The main question answered is how and why the Reducing Agent is successful in all of the pathogenic integrations, regardless of species or viral type.***This, one can see, is due to the **firmness of the double bond against rotation**, since the cleavage is had between its two terminals and **they remain fixed with reference to each other.** The diagram also indicates **the fixation of the single covalent bond that combines the pathogen and host cell, in each specific disease integration, so as to offer steric hindrance to successful attack by certain Reagents, and steric advantage to others, and this is confirmed by clinical experience.**

CRITICAL ATOMIC GROUP OF PATHOGEN, ESSENCE OF PARASITISM

The pathogen may integrate with the host cell's FCG by the condensation via its amine group and block FCG function, or pour polymerization energy into it to force an allergy or a neoplasia. This need not be diagramed, as only one pathogen is required. Blocked functions as in diabetes or mental suspensions following the toxic amine carrying antibiotics are examples. But neoplasms caused by butter-yellow and diaceryl aminofluorene require a supporting carcinogen to supply the energy for mitosis.

H(3) H H
R C(4) ---+ C (6) = - C(7) â€" R' (Pathogen's critical atomic group.)

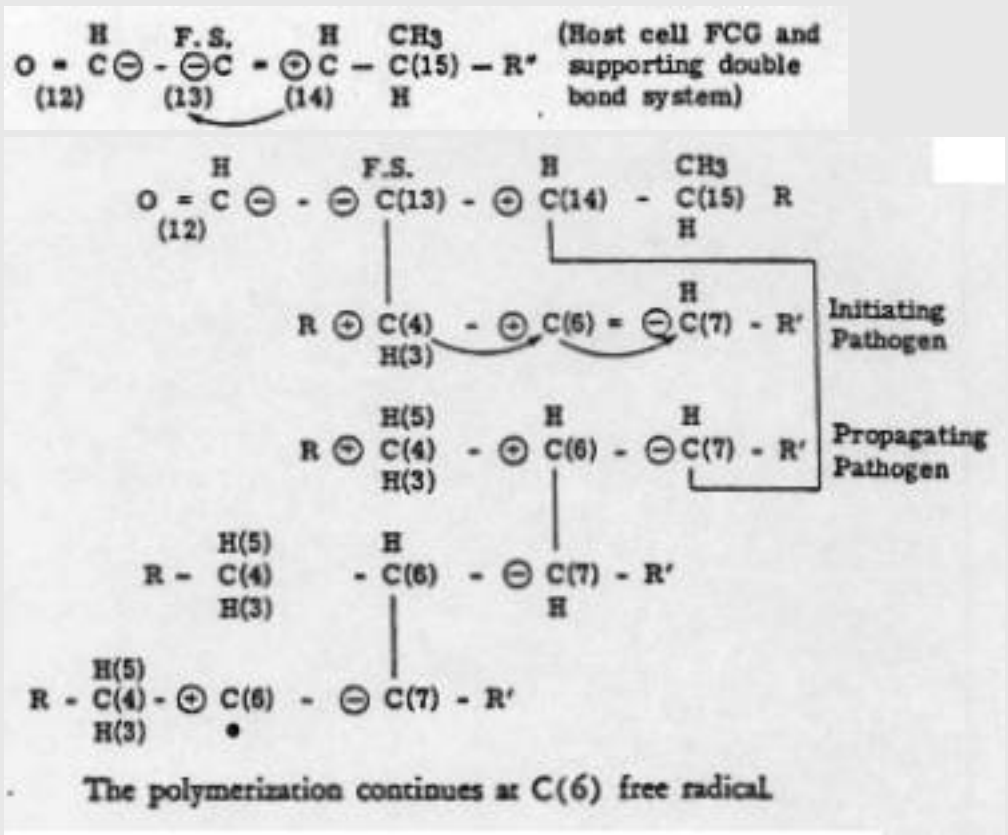
H(5)

The polarity of C(4) is positive like C(6) through withdrawal of electrons by R’ and C(7), which thus become negative. R’ could contain halogens, nitrile, etc.

CRITICAL ATOMIC GROUP OF THE FUNCTIONAL ENERGY PRODUCTION AND ENERGY ACCEPTING SYSTEMS. ESSENCE OF AUTONOMY

The polarity of the Carbonyl group (12) is strongly negative through the electrons it has withdrawn from the double bond, and C(13) is also negative because of laying in the orbit the Carbonyl electrons, which polarizes the electrons to the pole nearest to it and removing them from the distal pole, which makes C(14) positive comparatively. The methyl functions at C(15) contribute electrons via the double bond to the Carbonyl group. R” carries groups like R and R’ of the pathogen that determine the line-up of the two when they integrate, and the polarities of the critical atomic groups’ atoms determine, which make the unions or additions to the double bonds. C(4) being positive tends to expel H(5) for easy removal by the Carbonyl group (12) forming the free radical that makes the addition of C(4) to the negative pole of the FCG’s activating double bond at C(13). Thereby, a free radical is produced at C(14) which adds to the negative pole C(7) of a fresh molecule of the pathogen to start the polymerization chain, which continues as an end to end addition yielding the energy that supports the allergy or the neoplasia.

THE INTEGRATION OF PATHOGEN AND HOST CELL CRITICAL ATOMIC GROUPS AND THEIR SEPARATIONS
(Schematic)



To rupture the integration oxidatively, the Therapy dehydrogenator removes H(3) of the initiating pathogen producing a free radical that adds molecular oxygen to become a peroxide free radical that cleaves C(4) from C(6) producing a Carbonyl group at the latter. C(4) also becomes a Carbonyl group which being positive remains attached to the negative C(13). **By gaining a Carbonyl group, the pathogen looses its parasitism and becomes autonomous.**

The polymerization bond between C(14) of the host’s FCG activating double bond and C(7) of the pathogen’s invites cleavage as C(14), is positive in polarity and tends to release its H atom to the action of a dehydrogenator of appropriate qualities, as offered in the Therapy Reagent. A free radical is formed there and a peroxide free radical results in the presence of oxygen that cleaves C(14) from C(7) of the pathogen, forming two terminal Carbonyl groups. ***The Functional System of the host cell thus now holds a cluster of three Carbonyl groups to serve its dehydrogenating function as activators and as dehydrogenators.***This is a quite formidable array, via its orbital-mechanics. ***The Carbonyl group won by the pathogen attracts electrons from the methylene group alpha to it and thus releases its hydrogen atom to any dehydrogenator at hand, as the cytochrome or ferrous-ferric electron acceptor systems, and so a new Carbonyl group is formed at each***

terminal again; a process that can be repeated until the pathogen is burned out of the way.

SEPARATION OF THE INTEGRATION VIA THE REDUCING AGENT

The Reducing Agent is constructed to yield a hydrogen free atom, which C(7) of the pathogen being of high negative polarity immediately combines. A free radical is thus formed at the C(6) pole, which being of positive polarity immediately combines the molecular oxygen in which it isbathed to form a peroxide free radical that splits the double bond to form a Carbonyl group at C(6). This Carbonyl group withdraws electrons from C(4), which is already positive and makes it release H(3) to any ordinary dehydrogenator, as before mentioned. **The initiating pathogen is thus removed and the FCG system gains a Carbonyl group, joined to its functional mechanism.** Another Carbonyl group is gained at C(14) by the progressive oxidation of the integrated supporting pathogen starting at the closest C(4) to the newly formed Carbonyl group, which now reinforces the FCG so it is amply able to remove the H(5), which is already repelled by the positive polarity of C(4). C(4) thus becomes a Carbonyl group as a result of the usual sequence of free radical action. Likewise, so does C(6) that draws off the electrons from C(7) so that it tends to release its hydrogen atom to the ordinary hydrogen acceptors and become a Carbonyl group that, in like manner, causes C(14) to release its hydrogen and become a Carbonyl group. **Now the FCG is a triple Carbonyl group affair with properties, as just described, as resulting from the action of the oxidation process instituted through the Therapy dehydrogenator.**

Whatever toxin debris is present in the FCG is readily burned away by the high power of triple Carbonyl system of the FCG as a dehydrogenator. The rapid action of the recovery process in cases where the Reducing Agent was used in dilutions of one part per trillion, may be explained on the basis of the procedure just outlined. The following polio case, coronary case, and the diabetes case, being typical examples.

The processes, just outlined, must be considered in any investigation of cancer, allergy, and infection, as they use the most basic of chemical phenomena, as we understand chemistry today. Whether the outlines given are the actual processes that take place is not easy to prove without much work. However, they lay out the paths to be followed in any basic investigations of the subject, and they were fruitful to us in our limited approach. The results cannot be overlooked, as such results have never been known before in the whole history of medicine unless, of course, we are scientific enough to factually examine the superior results of Divine Miracle Healing, as reported by Nobel Laureate Alexis Carrell, which he compared with his tissue culture data and, which yielded some enlightening conclusions that cannot be scientifically brushed aside, though they follow laws of Nature we are not as yet able to understand. **The cases we present follow basic cycles and laws that we have observed before, whether interpretable or not.**

THE RECOVERY PROCESS

This is a cyclic affair with periods of 12, 24, 36, 60, 72, and 84 hours, weeks, and other multiples of 12 hours, 3 week, and 3 months until recovery is completed through the reverse repetition of the symptoms of the pre-growth toxic period in which functional block suppresses oxidation-favoring development of fermentation with its displacement of oxidation. *For this, one dose or two of either the Oxidation or Reducing Reagent is all that is generally used. During this recovery period, the symptoms of action of the pathogen's toxin during the development of the disease are repeated in reverse order to their coming.* These the writer showed, as early as 1927 (7) to be neurological, vascular, or digestive disturbances, and represent different phases of the polymerization of the toxin from a monomeric form of the acute infection on through various molecular weights, until it has reached the structure able to produce a neoplastic response. *During the recovery, the process is reversed as the polymer is oxidatively broken down peeling off its accumulated monomer units and passing through stages where it produced the various symptoms of the pre-growth period.* Headaches, dizziness, epilepsy, diabetes, psoriasis, arthritic changes, etc. may be the changes

that reappear for a short time and then disappear. ***Usually the last reaction is an acute inflammatory process at a point where the patient had experienced a severe infection many years earlier.*** In breast cases, it is usually the tonsil and associated lymphatics on the same side as the affected breast, but it may be a scar somewhere else where a severe infection was present. This reaction generally comes right after the growths are absorbed, or when only the supporting fibrous tissue for the neoplastic cells remains. This absorbs much slower or may become calcified and absorb still slower. Many biopsies have demonstrated this fact.

This reaction may come in the appendix even where the pathogenesis concerned the breast, and was not a tumor, but instead a neuritis. For example in my early experience, a woman of about fifty had her left breast removed because the celebrated surgeon diagnosed cancer behind the nipple. The symptoms were sharp shooting pains. After the breast was healed and she returned for her first inspection, she had the same sharp pains in the right breast nipple and the same surgeon insisted on removing that breast that very day, but because of social engagements, he consented to let her wait till after a large dinner party, scheduled for the following day in Detroit. Instead, her husband brought her to me for examination, and I could find no tumor, whatsoever, but the pains were the exact pictures of the Homeopathic Berberis symptomatology. She said the left breast felt exactly the same, and was now frightened into having the right breast removed. I gave her Berberis and the pains were gone in a few days, never to return. The breast was never removed either. A recent different "Berberis case" came to Rio for diagnosis, because her Arizona surgeon wanted to remove the left breast for the same type of sharp pains. My examination found no tumor, whatsoever, and as she was so emaciated with the skin stretched over the ribs, so that any tumor would easily have been observed. The axillary glands were palpable as they would be in such a state of emaciation. Examination revealed a chronic appendicitis over to the left of the midline and tenderness below the umbilicus. **She was advised that her great reaction would occur in that position.** She was so weak it was difficult to walk and carry her purse. **It took a year after the SSR injection for the pains to completely disappear and to gain strength, when suddenly on the 60th week she came down with an "acute appendix attack."** On removal the appendix, it was found to be attached firmly to the left ovary and kinked. Her health quickly returned. Had the appendix been cultured, the germs would have possibly been found to have been non-pathogenic at that time, as was observed in other instances.

This recurrence of the reaction cleaning out the lesion that harbored the original infection (as a last feature of the recovery process) also occurs in the treatment of rheumatoid arthritis, and many neurological and psychiatric problems, and in diabetes. It teaches much that the profession is eager to know. The complete text gives examples such as fill the insane asylums, but would be simple problems to clear up, if this etiological factor and the pathogenesis described here were common knowledge.

THYMUS GLAND DEFICIENC AND MUSCULAR DYSTROPHIES

The patterns of endocrine deficiency vary in some instances from that exposed in the exophthalmic goiter case, Mrs. M. J. Here, the deficiency was not in the thyroid gland that attracted so much attention, but in some other tissue that could not accept the energy of ATP into its functional units and hence, was starving for energy. So some nerve or hormonal factor acted on the thyroid to produce thyroxin to whip up the tissues to produce energy carried as ATP to supply the starving tissue. However, the block to the FCG of energy acceptance for function prevented this energy from being used and a vicious circle was established that was leading to fatal exhaustion. After the FCG was freed so it could accept the energy, the whole mischief was normalized.

In the muscular dystrophies, the thymus is the essential deficient tissue upon which the muscle deficiency depends. In both the thyroid and in the complete thymus deficiencies, the inability to accept energy into the functional mechanisms is evident in the hyperplasia of the gland, and the increased use of oxygen and higher basal metabolism rate. In the thyroid case, the BMR was as high as 104%, but in complete thymus deficiency cases it is very much less elevated, though enough to indicate the inability to use the energy of ATP. It is also evident that the thymus defect may not be complete, but may depend on the inability to use its

specific trace element, manganese, as a thyroid case may not use iodine, or an anemia case may not use cobalt. So one must provide a concept of how the thymus gland works as we have for diabetes, especially, because orthodoxy has no solution.

There are a few facts that can be organized for a practical pattern of its function. Alpha tocopherol is essential to its function as well as manganese. The spent product of tocopherol appearing in the urine is in the form of the hydroquinone of tocopherol Therefore, our Thesis is simply that tocopherol is oxidized to the quinone, which on performing its task, is reduced to the hydroquinone. In other words, the manganese is used by the thymus Hassall's cells as a co-factor, possibly as the trioxide, to oxidize tocopherol to its quinone and the quinone serves as an oxidizing agent (hydrogen or electron acceptor) in the further function of the gland, as in the production of a substance for the development and function of the muscles, and of the reproductive system. In this latter function, the use of ATP is required and when the FCG of energy acceptance of the Hassall's cells cannot accept this energy because of a block via an integrated pathogen; thus the thymus and muscle deficiencies are complete until the block is removed. **Many years ago we used the serial system of Carbonyl groups and those as activated in Benzoquinone, to accomplish the liberation of the FCG.**

But the deficiency in the thymus may not be complete and may involve the simple oxidation of manganese to its trioxide. The supply of fair but non-toxic amounts of manganese to the tissues in general Josephson **(8)** found would correct such cases. Evidently the chain of subsequent processes was unimpeded and the body cells in general oxidized the manganese. But when the FCG of energy acceptance is integrated with a pathogen, the use of the Reagent given to the thyroid cases, is also required to reverse the disease picture in Parkinson's disease, myasthenia gravis, and some other forms of muscular dystrophy, as reported by our collaborators. We will give a special discussion to this subject with photographs demonstrating muscle reconstruction and return of function.

CASE HISTORIES TO ILLUSTRATE CORONARY OCCLUSION AND INFARCTION SHOWING JELLING AND RECOVERY OF BLOOD COLLOIDS

Mrs. S., age 74, with long history of arteriosclerosis and aortic insufficiency, usual blood pressure 200/100, had a severe coronary attack in June 1960. The Oxidation Reagent was given before true infarction could take place and the recovery was immediate. The electrocardiogram showed no infarction. The following year on June 27, 1961, she had a severe attack and was immediately hospitalized and every possible aid given while under the oxygen tent provided no favorable response. Her condition deteriorated rapidly, blood pressure 190/100, great pulmonary edema, thin weak pulse at 130 per minute, great dyspnoea, general cyanosis, chest pain, and she was at the point of collapse when the Reducing Reagent was given, 2 cc. of the one to a trillion dilution injected into the triceps muscle by Dr. Jayme Treiger. The response was immediate. Right after the needle was withdrawn, the blood pressure was found to be 140/80, the pulse 60 P.M., the dyspnoea ceased, and the cyanosis faded away. She was comfortable within a "minute." The next morning the electrocardiogram was taken and showed extensive infarction of the septum extending over the lateral wall of the left ventricle. Another electrocardiogram taken a week later, showed much improvement with diminution of the size of the area of infarction. The day after the crisis, the blood pressure was back to her usual normal of 180/100. At the first attack, the occlusion caused by the jelling of blood in the coronary vessels was quickly changed to good dispersion before infarction could happen after the Treatment was given. In the second attack, this also happened so that the infraction process was halted from extending to include the area where the jellification had occurred. The pathology started to reverse visibly within a week and immediate functional improvement, is to be noted.

EXOPHTHALMIC GOITER, NODULAR TYPE

Mrs. M. J., age 35, in July, 1943, showed rapidly developing weakness, tremor, sweating, great changes in appearance, extreme exophthalmus, rapid loss of weight and strength, excitement, excessive nervousness, tremor, jerking of the muscles, spasms with toes bending inward, and use of the fingers became difficult. The thyroid region was enlarged by a number of hard nodular tumors, rapidly increasing, pulse thready and too rapid to count accurately, blood pressure

190/110, and B.M.R. plus 104%. She was too weak to walk and had to be carried into Dr. Julian Baldor's office on November 10th, 1943, after being under iodine therapy, ice bags to the neck, and absolute quiet from July to November 10th, while steadily deteriorating. She thought she was losing her mind, had hallucinations. She was given 2 micro micrograms of the serial system of Carbonyl groups in the triceps muscle. In two weeks, a remarkable change for the better was evident. She was stronger, could sleep, gained weight, etc. In twelve weeks, she was completely normal, physically and symptomatically, B.M.R. plus 6%, pulse 80, blood pressure 140/80, back to normal weight and strength, working hard by carrying a suitcase weighing 50 pounds in and out of houses as a demonstrator, playing in an orchestra, etc. Eyes, thyroid, and nervous system completely normal and have remained so to date.

Comment: In this case, somewhere in the body, because of block of FCG function, important cells could not obtain the energy of ATP; they were starved of energy. So the hormone message was sent to the thyroid to whip up all the tissues to produce more ATP to overcome the energy starvation of that group of cells where ATP energy was blocked from entering the working mechanism. Thereby, the thyroid gland was stimulated to the limit and the tissues were depleted to utter exhaustion. The Carbonyl compound of high oxidation potential caused removal of the obstructing toxin, as we have explained. As soon as the FCG of energy reception was freed and could go back to work, the tissue that was starved received all the energy it could use, so the call on the thyroid ceased and its nodular hyperplasia and activity subsided to normal. The depleted tissues were no longer forced to produce ATP and all was well.

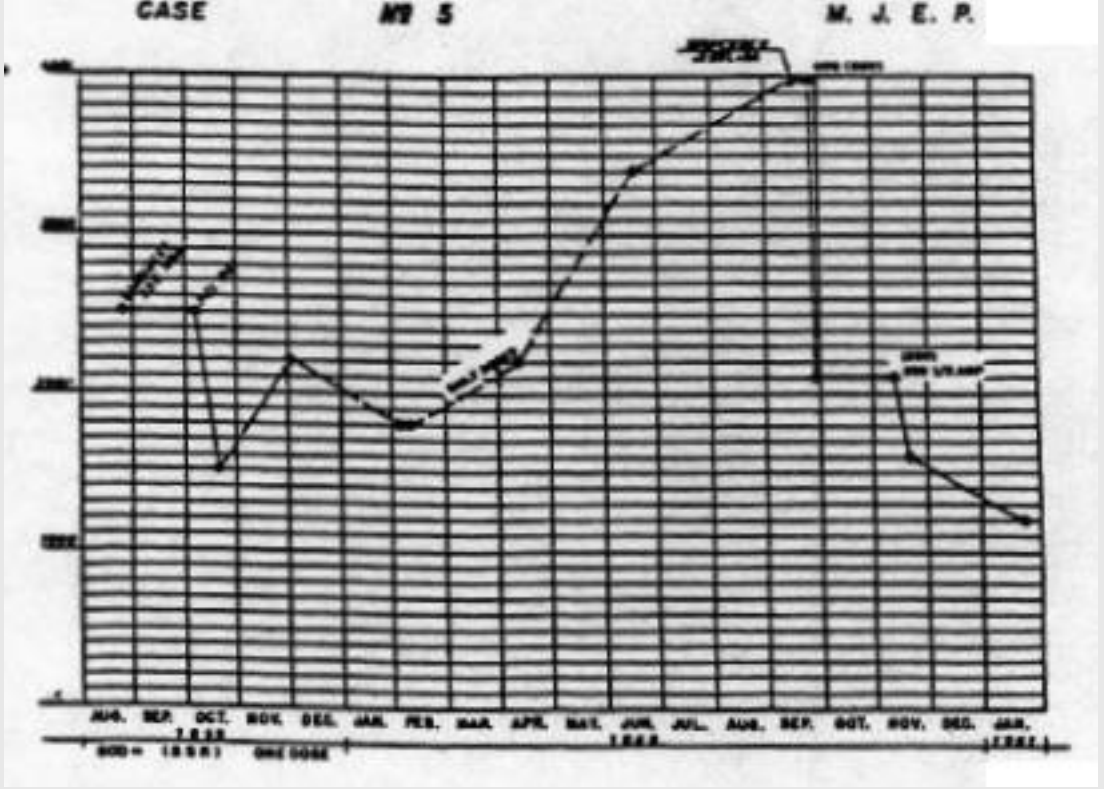
EXOPHTHALMIC GOITER

Mrs. D. F., age 27 years, thin, very nervous with dyspnoea, cold sweat, pharyngeal spasm, acne, leukorrhea, sometimes bloody fetid urine, painful nodules in the right breast that persisted and followed a cautery of an ulcer on the cervix uteri, tachycardia of 106 per minute, and a slight thyroid enlargement. Since childhood, she had periodic crises of angina, along with high fever, and pus coming from the tonsils. Treated by a gland specialist, she received a dozen modern drugs without benefit. Feeling worse, she consulted Dr. Jayme Treiger on March 12, 1958. The B.M.B. was plus 45%, blood pressure extremely low, nightmares, pulse 106, and weak. She was given two-millionths of a microgram of the Reducing Agent on March 13, 1958, by Dr. Treiger.

Recovery Course: Every three and a half days, the tonsils became inflamed and swollen with a strong pus discharge until they were normal in texture and clear of pus. The cervix uteri, likewise, became inflamed periodically and drained freely until it became normal. In spite of these crises, she was feeling better with renewed vitality that began to show within the first week. Three weeks later, she reported with a normal pulse of 82, and a blood pressure of 120/90. The cervix ulcers were healed; the B.M.R. was 6% above normal. Two years later, she was perfectly normal, pulse 60, blood pressure 110/70, temperature 36.7, with the best health she claimed she had ever experienced. **In this case, twenty years of pathogenesis steadily retrograding turned to a near normal in a few weeks and was perfectly normal in two years. The reversal of the pathogenesis in this case is like that of the former case, even though the Reducing Agent was used.**

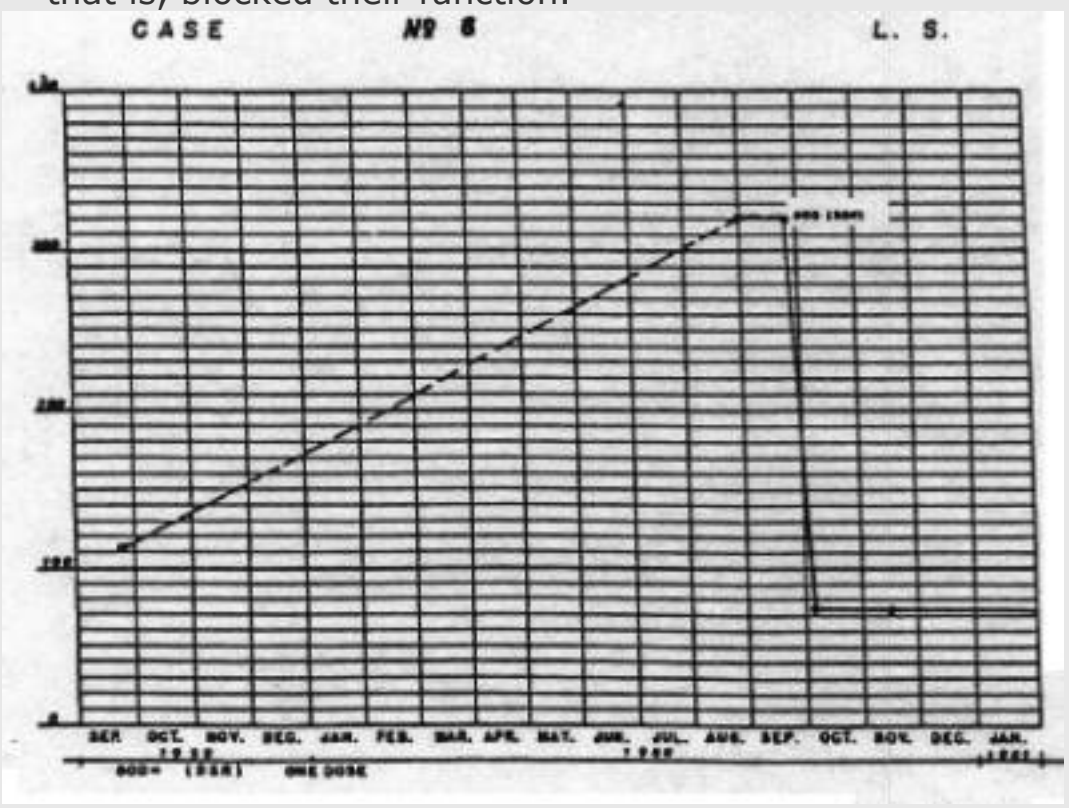
DIABETES

It will be seen that this disease as currently met in the great majority of cases is an intoxication that blocks islet cell function. The energy of ATP does not enter the fibrillae that synthesize insulin in the affected cell. If the situation lasts long enough, some of the cells die off and are only slowly replaced after the toxin is removed. The case of Mrs. M. J. E. P. will illustrate. On the other hand in fairly early cases, as Mr. L. S. of only one year's duration, even with a very high blood sugar 320 mgm.%, there may be a hyperplasia of islet cells, which are also paralyzed by integration with the poison so when this is renewed, the effect of the hyperplasia is seen in that the blood sugar stays at a very low normal level or less, 75 mgm.%



Mr. L S., age 53, had a rich venereal past, malaria at 21, and operated for varices in 1941. He complained of vertigo, edema of the legs, grade 2 small varicosities, aorta palpaple, fundus oculi showed veins with second grade manifestation (Wagner), blood pressure 240/130, pulse 96, glycemia 112 mgm.%. Clortiazamide reduced the blood pressure to 210/110 with vertigo. On January 19, 1960, there was dyspnoea, and blood pressure 200/110; on February 14, blood pressure 220/120, pulse 84; on May 16, vertigo, tachycardia, dyspnoea and after lying down, blood pressure 250/130, pulse 90. On August 18 epistaxis, blood pressure 260/120, dyspnoea, constrictive feeling in neck, blood sugar 320 mgm.%, urea normal. Thus, there was a steady rapid deterioration during the pre-treatment control period. One-tenth of a microgram of the serial system of Carbonyl groups was given on September 24, 1960. He had a reaction on the following day. The edema and constrictive feeling in the neck disappeared quickly and in three weeks he felt very well, weight 82 kilos, blood pressure 170/100, blood sugar 75 mgm.%. He has remained in good health. One sees that the diabetes was but one feature of a multiple symptom poisoning. Before the Reagent was given, all insulin and other drugs were stopped. He was taken off of animal proteins and placed on an unrestricted cereal, vegetable, and fruit diet, with sugar, honey, and molasses. This diet is our usual procedure followed in diabetes. He has remained well with high efficiency islet function as the low blood sugar persistently shows. **(9)**

The reaction in this case was severe with chills, fever, and general muscular pains, especially in the legs. When one compares this reaction with the mild one of the following case, one sees that the etiological factors were different and caused different general effects, though they both affected the islet cells in the same way -- that is, blocked their function.



The structures of the etiological toxin then were different, but they still had the one feature in common; namely, ***the ability to integrate with the islet cell's***

functional mechanisms and this common feature we identify as the activated position alpha to a double bond, which provided for the integration and also invited the oxidative separation. Evidently there are a number of toxins that have different general effects, but are able to attack and integrate with the islet cells by the same mechanism--a mechanism that invites separation from the host cell by using the same Reagent, in exactly the same way.

Another case, given the oxidation serial Carbonyl group Reagent is that of Mrs. M. J. E. P., age 51. **(10)** A few minutes after the previous case was treated, she was given the same dose of the same Remedy from the same ampoule as the preceding case, 1/10th of a microgram in a one to a million dilution. This was to facilitate comparison.

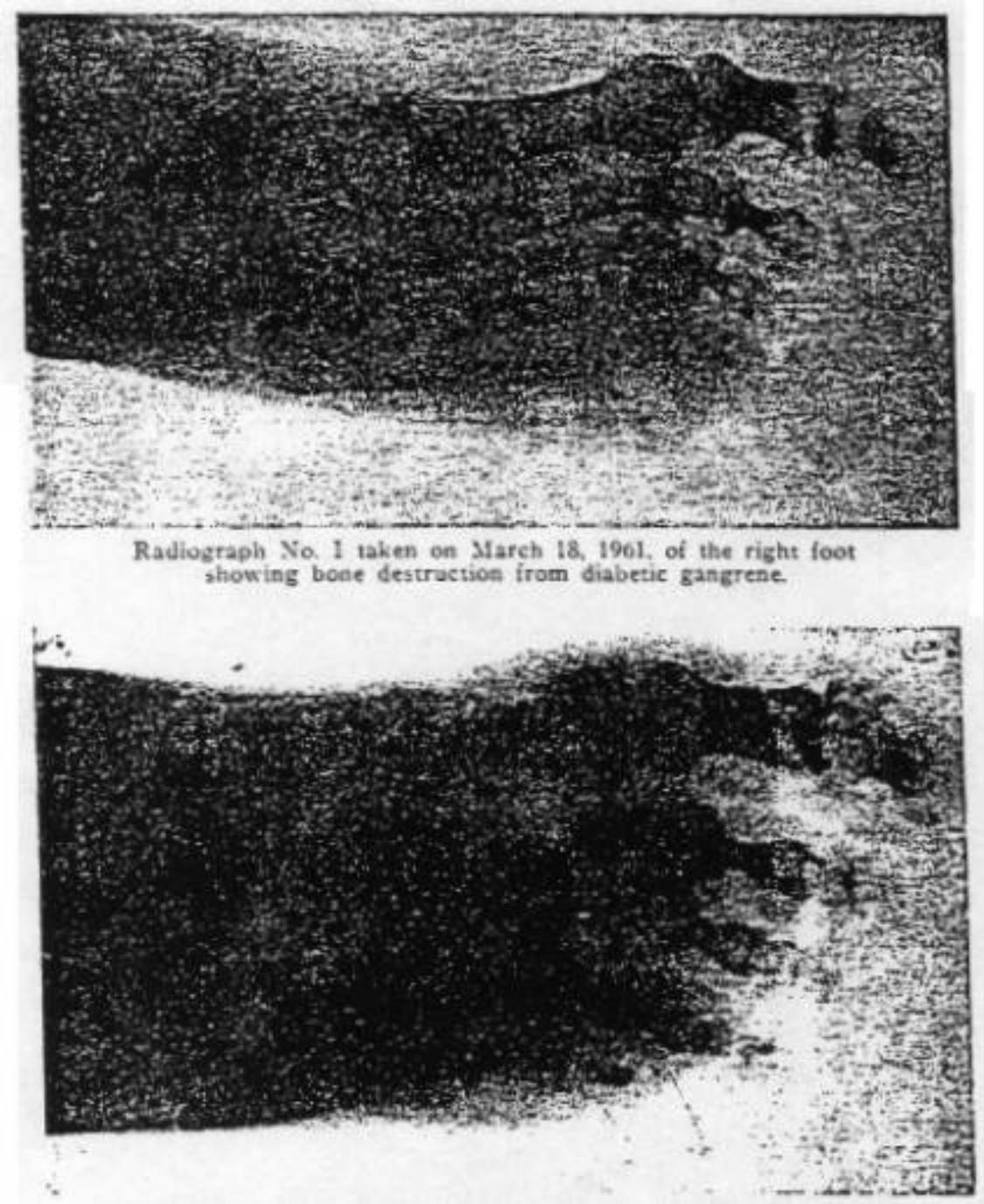
Her pre-treatment control period showed that she had been diabetic for five years, but was first seen by Dr. Treiger on August 24, 1959. Her first complaint was articular pains, thirst, excess weight (95.6 kilos), height 158 in., blood pressure 175/90, edema grade 2 in both feet, glycemia, 240 mgm.%, Folin Wu, urine S.G. 1.036 glucose 4x. She could not tolerate diabenase and was put on protamine-zinc insulin during the whole of 1959 and 1960, but the blood sugar generally ran about 200 to 240 mgm.% on 40 units of PZI. In June 1960, the glycemia was 340 mgm.%, while on 60 units of PZI, and by September, it rose to 398 mgm.% while on 60 units of PZI daily. She was then taken off of insulin and all other medication, taken off of animal proteins and on September 24th, she was given the serial system of Carbonyl groups, one-tenth of a microgram, as in the previous case and placed on the same unrestricted carbohydrate diet.

In five days, the glycemia fell to 210 mgm.%. In two months, her weight dropped to 89.5 kilos. The edema left the legs within a week after Treatment; there was a slight grippy reaction about that time, also. Her whole health changed for the better. Hemglycemia on November 30, 1960, was 160 mgm.% and six weeks later, it fell to 120 mgms. and has so remained.

USING THE REDUCING AGENT

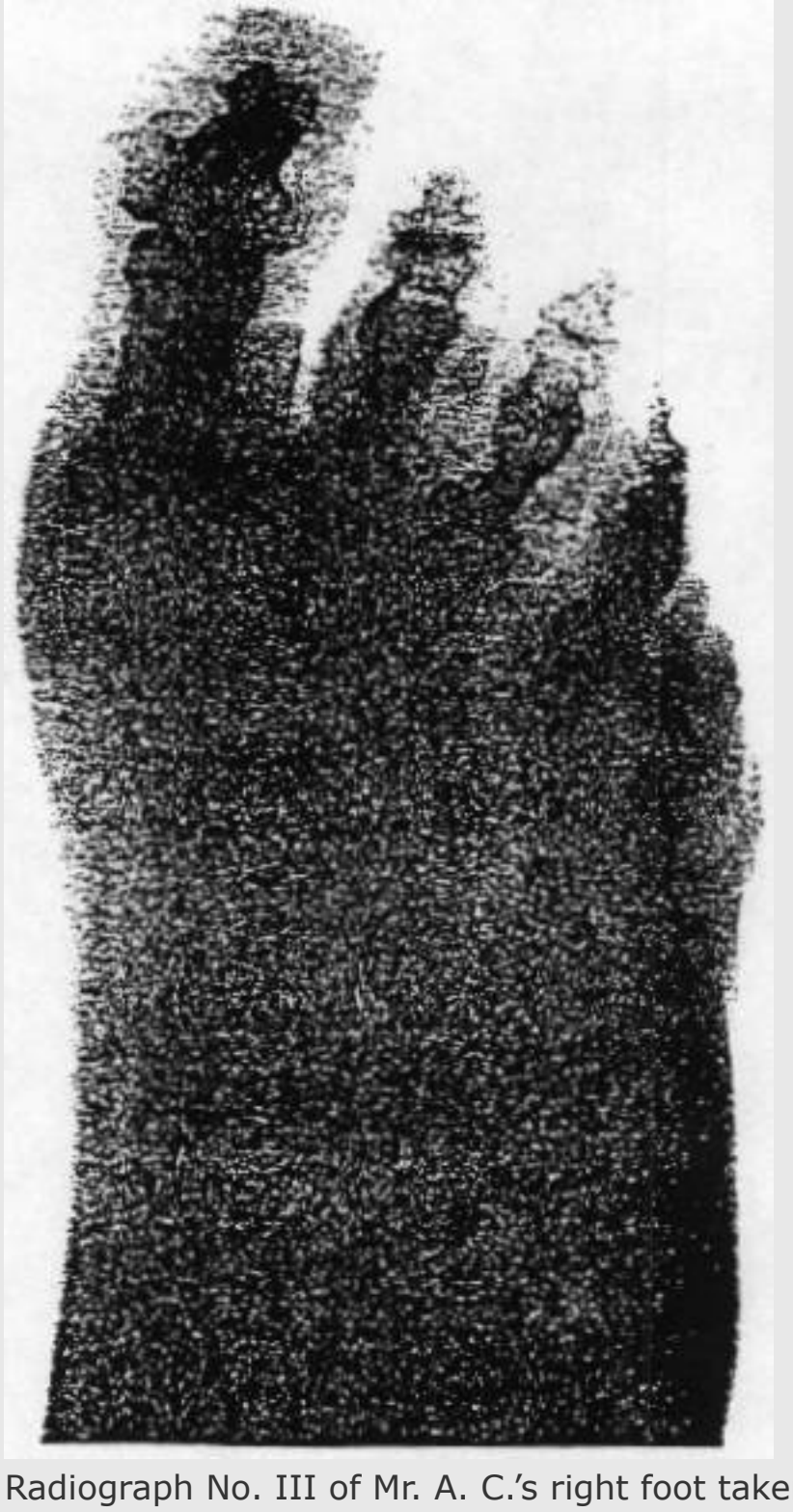
A case of diabetic gangrene was given the Reducing Agent by Dr. Julian F. Baldor while in a state bordering on coma, Mr. A. C., aged 71. He had been diabetic for five years and was on insulin, but gradually his condition deteriorated. On January 5, 1961, he had a severe crisis, glycemia 375 mgm.%, high fever, much pain in the right foot, and was approaching coma. Gangrene of the 5th toe had set in; the toe was amputated on February 12, 1961.

VISIBLE DEMONSTRATION OF REVERSAL OF THE PATHOGENESIS



Radiograph No. I taken on March 18, 1961, of the right foot showing bone destruction from diabetic gangrene.

Radiograph No. II taken on June 3, 1961, of the same foot showing bone reconstruction where the gangrene had formerly destroyed the bony structures.



Radiograph No. III of Mr. A. C.’s right foot taken in December 1961, about 9 months after Treatment.

AN INTRODUCTION TO FREE RADICAL THERAPY

The fever continued and the gangrene spread rapidly to involve the foot. Gangrenous fistulae developed on all aspects of the foot and discharged dead bone. Amputation above the knee was considered, but then abandoned because of the violence of the gangrene.

The Reducing Agent was given in a dose of one cc. of one millionth of a microgram to the cc. on March 18, 1961, after being taken off of all medications and animal proteins. Two days later, March 20, the radiograph of the foot was taken. It showed the destruction of the fourth tarsal and metatarsal bones and part of the third and part of the fifth metatarsal bone. On March 30, 1961, without insulin, the glycemia was 124 mgms.%. In eight weeks, the lesions were all healed and the destroyed bones were reconstructed in normal minute detail, and he was able to walk normally. On August 25, 1961, the glycemia was 80 mgms.% and his health perfectly normal. The radiographs demonstrate.

Here we see that the pathogenesis was reversed completely and our Concept is verified. (11) Other diabetic cases of great severity and long standing, when placed on the same regime, reduced their glycemia from over 400 mgms.% to 135 mgms.% thus, showing that islet tissue was somewhat deficient and had to be restored. However, their diets were unrestricted cereals, fruits, vegetables, honey, etc., and without any medication whatsoever. Consult complete text for other observations.

VIRAL INFECTIONS

The nerve cell gives the best chance to observe viral integration and its separation. The restored function being the criterion, for anterior horn

cells do not reproduce.

Two states of viral integration with the host cell's functional mechanism are known, the lytic in which the host cell is quickly and completely destroyed and its energy and substance are used up for viral vegetation. This is the only type encountered in rabies and the full destruction takes about four days. In dog distemper, **the other type of integration where the virus lives in symbiosis with the host cell is that usually encountered, but the rapid lytic type may intervene at any time.** Eventually the symbiosis yields to a lytic destruction with possible fatality. Here energy production and acceptance is completely blocked and the host cell and virus appear to be dead. The host cell withers and the axis cylinder of the nerve fiber appears to be lost. There is no function and the dependent tissue atrophies and the growth of the affected limb are hindered more or less. The invalidism caused is equal to that of the lytic type. ***Still the virus can be removed and the host cell will recuperate so that the dependent tissues and growth of the limb will be restored, even twenty years after the infection took place.*** This is the best type to demonstrate our Thesis**. During the acute phase, it is not always possible to differentiate between the two types, except that the rapid spread of paralysis and the violence of the toxic attack, speaks for the lytic type. Its reversal is also the quickest when treated early.**

CHRONIC SYMBIOTIC ANTERIOR POLIOMYELITIS WITH PARALYSIS AND ATROPHY OF THE WHOLE LEFT LEG

Myrna R., age 10, presented paralysis and atrophy of the left leg from hip to the toes, for three years. She was at the famous Warm Springs Foundation, but was turned away as entirely hopeless. The leg was too flabby to support a brace and she had to be carried. She received the serial system of Carbonyl groups in a two cc. dose containing one millionth of a microgram from Dr. Julian Baldor on February 11, 1944. Two reactions showing pain that spread down the back into the left leg occurred on the third and sixth weeks. Thereafter the recovery was steady, until after the twelfth week when she could walk and play with other children. **The muscle reconstruction was so nearly perfect and also the function, that it was difficult to tell which leg was affected.** She took up toe dancing.

CHRONIC SYMBIOTIC ANTERIOR POLIOMYELITIS OF TWENTY YEARS STANDING WITH COMPLETE PARALYSIS WITH ATROPHY FROM HIPS TO TOES

Mrs. V. N., age 23, when first observed on April 5, 1943, by Dr. Wendell Hendricks. She could not walk, so she was carried into Dr. Hendricks' office and placed in the chair; picked up and carried our again. She received the Oxidation Reagent on April 7, 1943, and a steady recovery followed. The dose was repeated on June 23, 1944, and on November 14, 1944, because the case was complicated by terrific migraine that was related to the polio infection. The calf measurements were right leg 4 inches, left leg 10 inches in circumference. The right leg was 2 inches shorter than the left. Both were completely useless, except as serving with steel braces from hips to toes for three point suspension, so she could swing about the house with crutches; otherwise, she was confined to the bed or the wheel chair. There was no voluntary motion, nor reflex motion, nor strength. The attack came at the age of one and a half years. Toward the end of the nineteenth year, contractures set in which required remaining in bed for the most part, as she could no longer wear the braces with comfort. During her 18-week and 63-week -- after the first Treatment, she had reactions of chills and fevers with more rapid improvement thereafter. The migraines disappeared completely in 1944. She was able to walk in about six months and in twenty-one months she was practically normal, able to run stairs and work all day standing on her feet without the use of braces or any other contraptions. The right leg had grown to be only Â½ inch shorter than the left, right calf 10 inches, left calf 11Â½ inches, function perfect and health excellent.

ACUTE POLIOMYELITIS WITH PARALYSIS LYTIC TYPE INTEGRATION

Loman A., age 10 years, started with violent headache, pains in back and legs, vomiting, high fever 104, and pulse 128 on February 3, 1944, in the afternoon.

The next morning his legs were paralyzed and the pains were worse. He shrieked with pain. Our examination showed the legs paralyzed from hips to toes, perfectly flaccid and without any tendon reflexes, or voluntary motion. When a sharp point was used to prick the soles of the feet, he made no response. The spinal fluid was taken and agreed with the standard findings for anterior poliomyelitis. There was no time wasted in withholding the Treatment, as the back was becoming paralyzed too, and his screaming was getting worse. Two cc of the same Reagent and dilution was used here too, at 11 a.m. By 2 p.m., he was more comfortable, the vomiting ceased, and the headache and pains were yielding. At 7 p.m., he could move his legs, the fever had left, and he ate a light supper.

The next morning he could walk to the bathroom. The recovery was rapid after that. He suffered a reaction during the third week, with chills and fever for three hours, and with pains in the spine and legs. This showed there was some symbiotic integration, as always occurs more or less in the lytic type. But here the violence of the spread of the infection, the very unusual fever and pulse, indicate a predominantly lytic infection and this is confirmed by the rapid recovery.

ACUTE POLIOMYELITIS WITH COMPLETE PARALYSIS OF BOTH LEGS TREATED WITH THE REDUCTION AGENT

Miss N.L., nurse, age 33, took sick on August 16, 1947, with the usual prodromal symptoms of terrific pains in legs and back, nausea, headache, and stiffness in both legs. On August 19 at 4 p.m., both legs from hips to toes went flaccid and failed to support her; she could no longer move them. The other symptoms either stayed the same or became worse. The writer saw her at 11:30 p.m. that day and found a flaccid paralysis of both legs from hips to toes and noted the other symptoms. There were no tendon reflexes and a sharp instrument used to forcibly prick the soles of both feet brought no movement, whatsoever. She was given one millionth of a microgram of the Reducing Agent in 2 cc. of water intramuscularly in the upper arm. The next day at 8 a.m., the mother phoned that she could move her legs and felt better in all ways; had slept some, and eaten a little. That midnight, I saw her again. She could stand and even walk, felt well, and was hungry. The recovery was complete in two days and all reflexes were normal.

A GENERALIZED SYMBIOTIC TYPE INCLUDING RESPIRATORY INVOLVEMENT

Florizinha, a girl of 6 years, showed a flaccid paralysis from head to toes. She could not move a finger or a toe, or speak even at a whisper. The respirations were too shallow to see. There was no voluntary muscle action and no muscle action was observed in all attempts at eliciting reflexes. She was in the Hospital Jesus of Rio de Janeiro, for four weeks before she was brought to the ward as a "sequel case" to receive this Reagent. The Reducing Agent was given in the usual dose of one millionth of a microgram, dissolved in 2 cc. of water. Two weeks later, when making the next visit, she was seen sitting on the edge of the table swinging her legs and waving her arms as she laughed and joked with the other children. Her recovery was complete and rapid. This was a totally symbiotic infection that involved the whole spine without anylytic complications, whatsoever. Many sad cases of invalidism depend on the same type of infection.

INFECTIOUS HEPATITIS, ACUTE LYTIC TYPE INTEGRATION

This is generally a lytic type of integration with occasional chronic symbiotic cases. Since the liver parenchyma is able to reproduce its cells and compensate for those injured, the disease is not fatal except in rare cases. The signs of rupture of the integration lay with the speed of recovery after the Reagent is administered, as compared with the usual course. It will be seen that the disease progress is stopped immediately, though the elimination of bile adsorbed into the tissues, takes a few days. Two cases will suffice. Every case treated so far responded ideally.

J.H.C., age 13, student, consulted Dr. J. Treiger giving a history of having drank water suspected to have been contaminated fifteen days earlier. He developed abdominal pain and the urine was loaded with bile. There was deep jaundice and profound drowsiness. He was subfebrile. Blood examination on February 5, 1959, showed Bilirubin 5.83 mgms.% (Malory and Evilin), the Van den Bergh was immediately directly positive, the cephalin-cholesterol positive, and the thymol

flocculation positive. He was given the Oxidation Reagent on February 5th, two millimicrograms in 2 cc. of water intramuscularly. In two days, he felt much better and the jaundice had faded considerably and there was no pain or other disturbance, but for all traces of the bile to disappear, it took over two weeks. The tests were repeated and all were found normal then.

INFECTIOUS HEPATITIS SYMBIOTIC TYPE WITH SUDDEN LYTIC CHANGE FULMINATING TOWARD A TERMINAL STATUS

Miss S.M.L, consulted Dr. J. Treiger on May 4, 1959, prostrated, with nausea, feeling like a drunkard. She had a fever of 38.8Â°C., muscular pains, halitosis, and facial neuralgia following a feast on seafoods. On May 8, 1959, the fever was gone, likewise, the muscular pains, but there was a severe pain in the gall bladder. On May 11, 1959, the blood showed Bilirubin 5.95 mgms. %, Van den Bergh strongly positive immediately 3 plus, cephalin-cholesterol (Hanger) three plus, thymol turbidity 7.5 units, thymol flocculation (MacLaglan) positive 3 plus. That night she was much worse, fever 39.5Â°C., extremely agitated, fear of death, hallucinations, and delirium. She was then given two millimicrograms of the Oxidation Reagent after the agitation gave place to a new phase of prostration bordering on coma. Improvement was evident in 48 hours with lowering of temperature, and return of appetite and bowel functions. Steady improvement followed and on June 17, 1959, the blood showed Bilirubin 1.02 mgms.%, Van den Bergh delayed weakly positive, cephalin-cholesterol negative, thymol turbidity 5.5 units, and thymol flocculation negative. On July 10, 1959, during the ninth week reaction following the Treatment, there was violent nausea and dizziness. She was given another dose of the Oxidation Reagent and improvement was apparent in three hours. Two weeks later, the blood test showed a normal Bilirubin of 0.41 mgms.% and all other tests were negative. During the twelfth-fifteenth week reaction period, there was an intestinal upset that cleared up quickly and during the 27th week reaction period, there was a pain in the left lobe of the liver. After that she remained normal. These reactions show that a symbiotic integration of the virus was present, as well as, the acute lytic type that fulminated to cause serious mental symptoms.

RABIES

This one hundred percent incurable disease has yielded like all other viral infections treated so far. Twenty years ago a rabid coyote caused the infection in a physician and in his horse on the plains of Montana. Both were treated with the serial system of Carbonyl groups after they had reached the convulsion stage. The recoveries took exactly as long as the time of development of the symptoms, three to four days. Some dogs were treated in the Army Hospital for small animals in Rio de Janeiro with encouraging results, also, and finally an epidemic of rabies in 1955 brought on by vaccinating costly zebu cattle gave the best chance for observation. Only the lytic type is known and from the earliest symptoms where swallowing is paralyzed it takes four days or less to terminate. Terminal cases took the same length of time to recover.

In this epidemic, 60 cows were vaccinated with the Fluery type live vaccine. Forty died typically and were proven rabid by autopsy and inoculation tests. Twenty were still alive when the writer arrived at the Fazenda. Of these, thirteen were treated and seven were held as control material, but were used for study in ways that would not interfere with the course of the disease. The controls all died typically and were proven to be rabid. Of the thirteen treated, eleven recovered. The two that died did so within an hour of receiving the Remedy, as they were about moribund when treated. One other case like the two that died was able to make a recovery. **It illustrates the recovery course and demonstrates, like the other ten, that recovery of nerve cell function depends upon cell reconstruction, a reversal of the pathogenesis in which the energy and material taken from the host cell during viral vegetation is returned for host cell reconstruction.** A perfect reversal of a reciprocal nature is, therefore, observed. **This suggests, but does not prove that the virus uses the host cell’s enzymes during the pathogenesis, as well as, by the host cell during its reconstruction.**

All cows treated showed paralysis of swallowing, some had severe torticollis and spastic convulsions and others could stand on their feet, but if pushed would fall and not be able to get up without help. One animal treated at the end of the third

or beginning of the fourth day of symptoms was unable to stand and showed the typical tonic convulsions. It lay paralyzed in the same condition for four more days after Treatment and was badly dehydrated at that time. The government veterinarian thought best to sacrifice the animal to get better autopsy material and save it more suffering. He, therefore, had it dragged to the truck, but when the attempt was made to hoist it into the truck it kicked up a fuss and tried to run away. The Fazenda veterinarian happened by and seeing the animal show coordinated movements, he ordered it let loose, led it to water where it drank greedily and was then chased to pasture where it ate its way back to good health. The point here is that, in the four days of progress of the symptoms and the paralysis before Treatment, the nerve cells had reached a state of advanced destruction, but were not dead as yet. **The energy taken by the virus through its integration with the FCG was able to support its vegetation and the autolysis induced in the host cell supplied the material for the viral vegetation.** After the Treatment, the situation was reversed to a complete reconstruction in the same time required for the host cell destruction and provirus colony formation, for at the end of that period, the cell was able to function normally again. **This is what happened in all of the eleven cases that recovered and the time relations indicate that the Reagent used to reverse the pathogenesis, did not attack at the point of integration of the virus, but at the most exposed unit, which would be the last one laid down in the co-polymerization of the viral nucleic acid units that constitute the essence of its central or pathogenic part.** ***The oxidation would be induced there by the Reagent as described and the separation of the last laid down unit would make it available for host cell reconstruction, into the very place from which it was taken during the host cell lysis. The energy liberated by this oxidation must be able to pass on to the host cell to serve its reconstruction, for so long as the FCG is occupied, it cannot produce energy. Thus, the successive steps of splitting off each unit of the virus provide both energy and material for the host cell's reconstruction.*** **The indication then is that the nucleoprotein part of the viral colony is made up of host cell's nucleic acids, which can excite no serological reaction of immunity, though it is the pathogenic part.** ***The energy is that supplied by the host cell originally, as well as, the enzymes concerned. This clarifies the fact that after integration, no serological effort can rescue the cell.*** One is, therefore, tempted to **look upon the** ***origin of the virus center as of a nonspecific material taken from and common to animal cells and workable by their ferments.***

The protein capsule is made of other material built on by the nucleoprotein center and is specific to each variety of virus. It has immunological, antigenic, properties. Cleavage of the viral nucleoprotein from the FCG results in restored structure and function and thus overcomes the pathology.

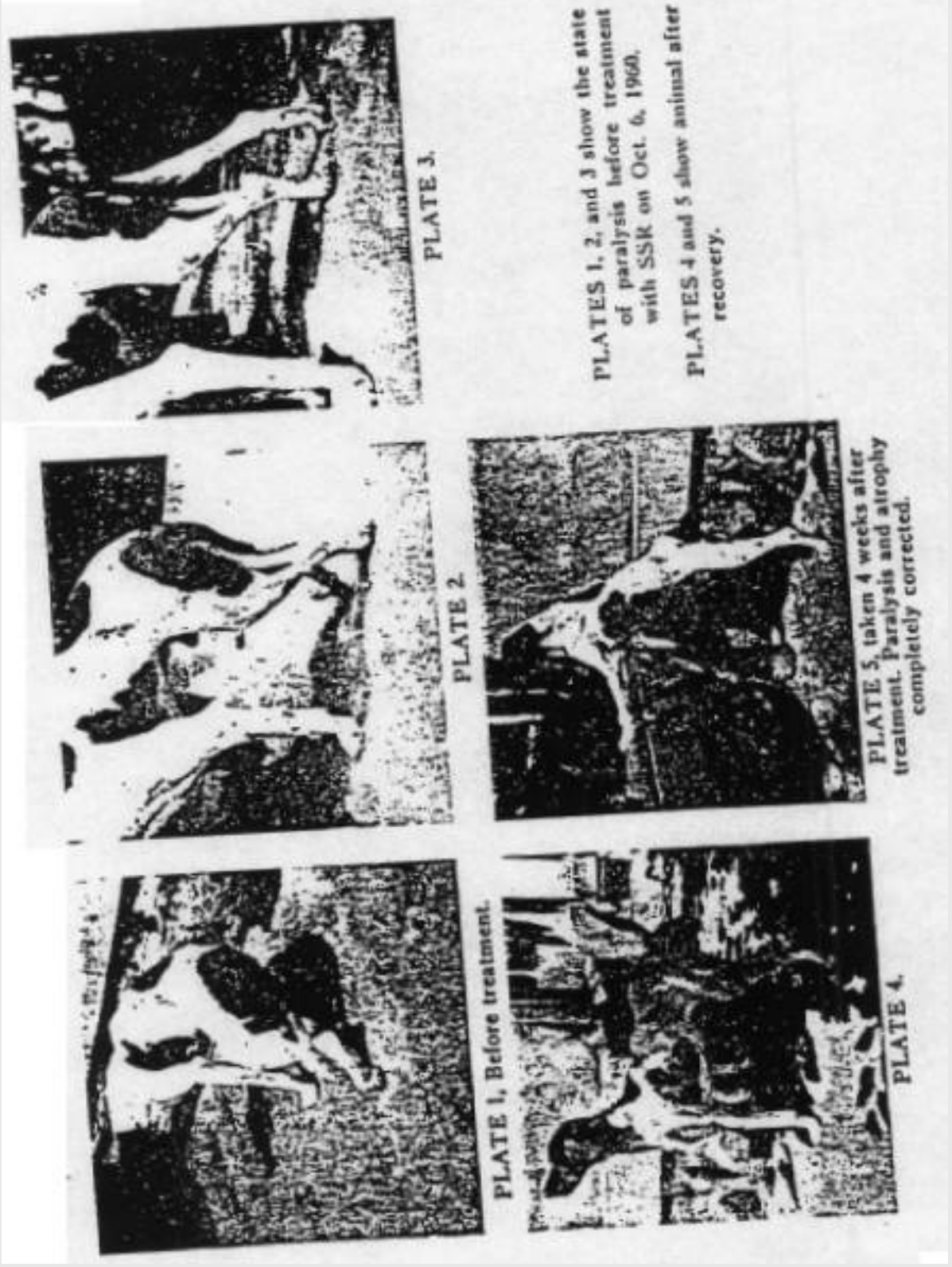


PLATE 3.

PLATES 1, 2, and 3 show the state of paralysis before treatment with SSR on Oct. 6, 1960.

PLATES 4 and 5 show animal after recovery.

PLATE 2

PLATE 5, taken 4 weeks after treatment. Paralysis and atrophy completely corrected.

TE 1, Before treatment.

PLATE 4.

PARALYTIC DOG DISTEMPER

While rabies is a 100% fatal example of the lytic type of viral integration, paralytic distemper is a 100% fatal expression of the symbiotic type. It has responded with 100% restoration of function in 90% of cases treated by the Oxidation and the Reduction procedures, outlined here in private practice. In the Treatment of all cases that came along, with care left to the owner, the Army Hospital for small animals secured an 80% recovery rate in two hundred cases in all types of distemper. (‘“*Veterinaria*,” Vol. IV, No. 1, p. 21, 1950, Colombo and Carneiro.) **(12)**

A typical case is that of the pointer, Singe, age 10 years. He was treated with the Oxidation Reagent on October 14, 1960, after he had become paralyzed in the right half of the torso and the left back quarter as the pictures show. The prodromal symptoms of trembling, loss of appetite, and sadness, lasted two weeks and the paralysis was of two weeks duration, before the Treatment was given. There was visible atrophy of all muscles concerned. The veterinarian offered to sacrifice the dog, as it was a hopeless case. The recovery took two weeks to overcome the paralysis and in two more weeks the atrophy was repaired as well, and the dog perfectly well. This was an early case and the reversal of the pathogenesis took as long as its development. Cases created with the Reduction Reagent showed no difference in their recovery percentages or course. Restoration of function of the paralyzed milk producing cells takes twenty-four hours after either Reagent is used in cows treated for hoof and mouth disease. **(13)**

CANCER

Warburg’s Thesis on tissue oxidation is well known and his report on anoxia as the

etiological factor in cancer **(14)** demonstrates this fact. He, however, does not explain how anoxia produces cancer and concludes that the pathology is irreversible. ***Our Thesis shows the essential place of anoxia in the pathogenesis, as it provides for the integration of a co-factor the pathogen or virus, with the host cell's functional mechanism via free radical additions or azomethine condensations. We have demonstrated for decades that the pathogen can be separated from the host cell by the two mechanisms of cleavage -- one at the position alpha to the activating double bond, and the other by cleavage through the double bond itself. Excess tissue of the tumors is placed in a position for normal function, but being in excess, it is digested and absorbed like a blood clot, and thereby serves as nutrition.*** **(Koch,** ***Cancer Journal,*** **October, 1924).** ***The pathogenesis is thus reversed!***

Warburg suggested that the mitogenic energy comes from glycolysis and it seems reasonable. However, ***our experience allows us to conclude that it arises in the polymerization of incompletely combusted metabolites (produced by germs trapped in an anoxic scar) that have entered the host cell and integrated with the mitotic mechanism, or by the energy arising in the polymerization or a part of a provirus that has integrated with the host cell's mitotic mechanism. We have observed in our earliest experience that when cancer is given small, rapidly polymerizing, unsaturated free radicals, their growth is terrifically stimulated by the energy, so liberated. Whereas, if a large, inert, free radical is given, their growth ceases and involution sets in.*** Here polymerization has been terminated and the source of energy is cut off.

Synthetic carcinogens serve as the initiators of the co-polymerization of the bacterial metabolite or provirus during anoxia, when the free radical formed by its dehydrogenation at the hands of the FCG adds to one pole of the double bond that activates the FCG, and the free radical thus formed at the other pole adds to the unsaturated ethylenic linkage of either pathogen, thereby producing a free radical that continues the polymerization, as an end to end process**.*** Very little energy is required for mitosis, and the energy liberated by slow polymerization should be sufficient, especially as it is liberated in the energy generating mechanism. ***The removal of each pathogen involved requires oxygen and an efficient dehydrogenator, as we explained earlier.***

METASTIC CANCER OF THE BOWEL

Mr. J. K., age 42. The pre-treatment control period extended from September 10, 1941, when the condition was so poorly developed that it was given a diagnosis of diverticulosis of the colon with a ruptured abscess at the Henry Ford Hospital of Detroit. By February 3, 1942, it had developed a bowel obstruction, which the X-ray revealed to be a cancer of the splenic flexure of the colon, which had already obstructed completely and had perforated. A colostomy was done on the ascending colon, as the left half of the abdomen was fully occupied by the neoplasm. By February 24, the extension occupied the whole abdomen and had perforated the belly wall in several places, showing large and small cauliflower growths with central necrotic festulous openings that discharged feces and extremely putrid material. Practically the whole belly wall was thus invaded. The biopsy taken from the fistulous invasions showed--Gross Pathology: Pathological diagnosis 101.62, John K., February 27, 1942.

"The specimen consists of a piece of skin measuring 14 x 14 axis. The central portion is destroyed and partially filled by a friable grey tumor mass which involves the underlying structures and has been cut through upon removal The tumor shows extensive necrosis. The edges of the specimen are cauterized.

"Microscopic: Sections show a tumor mass invading the subcutaneous tissue. The normal epithelium is absent over the mass. The cells of the tumor are large, hyperchromatic and show many mitotic figures. Poorly differentiated tubular glands are formed by these cells. The massive necrosis affects large areas of the tumor.

"Diagnosis: Metastatic carcinoma of the colon." The voluminous Henry Ford Hospital record gives much more data, showing that the invasion of the neoplasm

was so extensive that it was impossible no cut through the abdominal wall. The retrogression from a strong man at work, of 180 pounds of good muscle to less than 135 pounds and bedfast from September 10, 1941, to February 27, 1942, took less than six months. A half dozen new fistulous cauliflower masses formed in and about the area that was operated, discharging even more offensive necrotic pus."

Then the Henry Ford Hospital experts wrote in his case record -- "This is an entirely hopeless case." He retrogressed still more rapidly to April 1, 1942, when he was sent home to die. On the way home, by ambulance, he received 2 cc. of the Oxidation Reagent intramuscularly. One month later, the dose was repeated.

"Post Treatment Progress: The interim report of the Henry Ford Hospital as recorded by Dr. Bohr, August 28, 1942, five months after he left the hospital and was given the Oxidation Reagent states: Case No. 342016, John K.:

"Patient left hospital April 1 of this year with a diagnosis of fungating cancer of the colon and a terminal prognosis. On the way home that day he received one of Dr. Koch's Cancer cure shots. On July 1, he weighed 113 pounds, but from that time on he began feeling stronger and gained weight. By the middle of July, his wound was completely healed. He weighed 175 pounds at the end of July and he has maintained his weight ever since then. He enters the hospital now, after being back to work for three weeks, for first stage of colostomy closure."

The history shows that the bowel functioned normally through the rectum at this time, so the colostomy was successfully closed and at the time, no cancer tissue was found on exploration. His health returned. He gained to his normal weight and strength and annual examinations for over a five-year period showed the recovery was permanent. **The recovery rate is proportional to the pathogenesis rate, which we have found to characterize the 100% fatal viral diseases.** Characteristic of the healing by this recovery process, no scar tissue was required to accomplish the tissue reconstruction, because the infection is cleared away with the cancer cells and healing can take place by first intention, without scar.

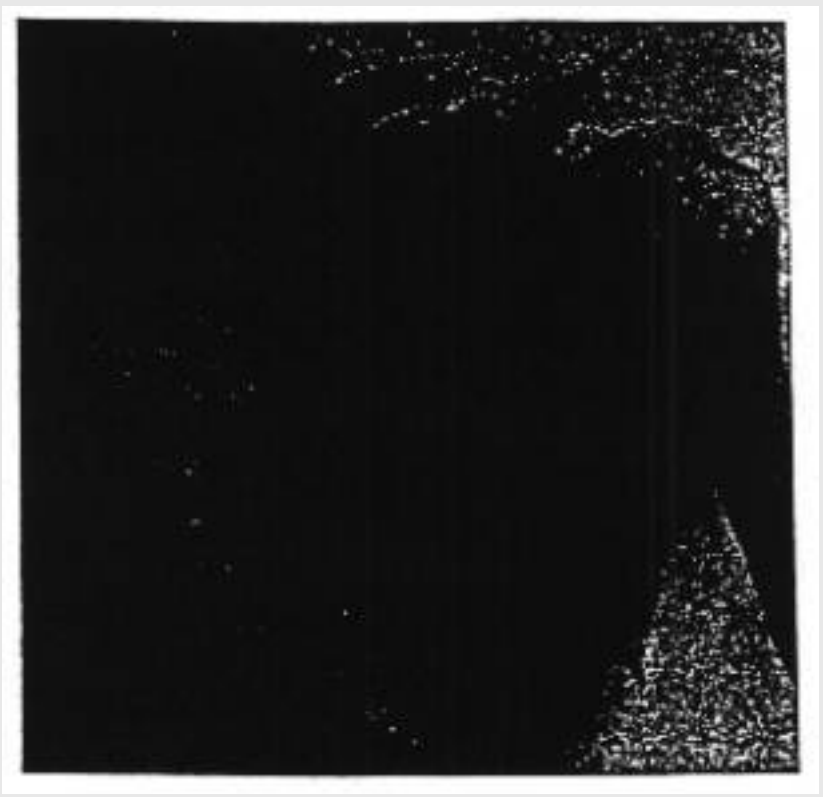
SARCOMA OF THE BONE

Mr. H. B. had pain in his right shoulder in July 1934. In August, he threw a ball and the pain became severe thereafter. He went to the University of Michigan Hospital for care. Radiographs showed a destructive involvement of the upper half of the humerus, the scapula, and clavicle. There was a tumor behind the scapula the size of half an orange and a smaller one of the same consistency near the spine. Biopsy, blood, and urine, analysis ruled out all conditions as giant cell sarcoma, Paget's disease, multiple myeloma, etc., and both the soft tissue tumor and the invaded bone showed it to be an endothelial cell sarcoma, a spindle cell hemangiosarcoma. This is a slow growing neoplasm, but always fatal according to Ewing and other experts. One can contrast the pathogenesis and recovery rates here with those of the former case.

He was given a hopeless prognosis at the University of Michigan Hospital. The findings of the University Hospital experts were confirmed. He was given the Reduction Reagent intramuscularly, two millionths of a microgram on September 17, 1934. The recovery reactions did not appear until the twenty-fourth and thirty-sixth week after the Treatment. These were fever, greater sensitiveness and grippiness, which lasted from three to four days each. The general health returned and the arm became useful again and normal. Radiographs taken in August 1942, showed complete healing of the lesions with denser bone than is normal. It is to be noted in this case, that the disease is one characterized by decalcification of the bone; the reversal of the pathogenesis shows an intense re-calcification of all the reconstructed bone tissue. This situation should be compared with the healing of bone destroyed by gangrenous diabetes where normal bone structure is restored, but the bone tissue itself, is not greatly denser than the normal bone. **It is the actual pathogenesis that is reversed.** He remained cured for over a decade and was lost track of in 1948, when the writer left the United States for work on viral diseases in Brazil. ***It is seen that the rules of recovery hold here with the Reducing Agent as are seen in recoveries obtained by the Oxidation Reagent.***

Radiograph I, showing condition before treatment.

Radiograph II, showing condition after full recovery.



SQUAMOUS CELL CARCINOMA OF THE CERVIX UTERI GRADE W

Mrs. M. W. received the Oxidation Reagent in the same dose as the J. K. colon case. It was also a most malignant invasive growth.

She was examined by three physicians, led by Dr. Loeffler, on January 12, 1940. He reported: "Her cancerous condition would probably, if untreated, end her life within a year. Because of the fixation of the uterus and the involvement of the adnexia, it is my opinion that it was not a surgical case, as surgery would have to be too extensive. It was too late for that sort of thing." It had already entered the cachexia stages, Dr. Loeffler stated, "She had lost 30 pounds in six weeks, complaining of general weakness, and had a rather poor color at the time." Examination showed it had broken through the pelvis and entered the abdomen.

Dr. Loeffler gave the Reagent on March 20, 1940, December 30, 1940, and in October 1941. His examination made a year and a half later in the summer of 1942, recorded: "She had gained weight; she had gained color and improved in appearance. The mass in the abdomen had subsided to the extent that I could no longer palpate it. The appearance of the cervix by examination with the speculum appeared normal," Before Treatment was given, she was taken to the Medford Hospital where a biopsy was taken. The diagnosis by Dr. Green, the Hospital pathologist, was "squamous cell cancer of the cervix uteri." But because of its highly malignant characteristics, it was sent to Dr. Hunter, the Professor of Pathology of the University of Oregon. He gave the following diagnosis: "I see a decidedly invasive and anaplastic carcinoma, which occupies well over half of the

tissues." It was graded four, which would be the highest degree of malignancy that is recognized. The specimen was submitted later to Dr. Weller, the Professor of Pathology of the University of Michigan. He reported as follows: "Prepared section # 2751, our 3823-LAW. Tissue from cervix: Medullary squamous cell carcinoma of poorly differentiated type. Histological Grade IV." December 7, 1944.

There was considerable interest in this case and to make sure of her status she was given a laparotomy on June 1944, by the surgeon, Dr. Haines. He found her pelvis normal with a small fibroid attached to the body of the uterus. The cervix was found to be perfectly normal and was left in its place. The body of the uterus, with the fibroid, was removed and given a careful serial sectioning for a careful search for malignant cells. Dr. Innskeep, the pathologist, reported there were no cancer cells found in the body of the uterus or in the fibroid. Thus, she was found cancer free by the most rigid test, in her fifth year after being treated. Ten years after Treatment, Dr. Loeffler was called to see her just before she died. He attempted to do an autopsy, but found the lower bowel and surrounding area to be a necrotic mass that was too foul to be of use for a specimen. The odor would not permit it. He did not know the uterus had been removed and sectioned and found cancer free, but he signed the death certificate "death from cancer." His report to Dr. Koch was, however, that he did not determine what the necrotic affair was and could not say it was not a gangrenous diverticulosis. **The case teaches that cancer can be reversed to normal in about the same time it took to develop and a malignant or equally serious pathology can start again, in the same position many years later, when the hygienic conditions are very unfavorable, as they were in this case.**

SQUAMOUS CELL CARCINOMA OF THE CERVIX

Mrs. T. was 31 years of age in August 1923, when because of serious bleeding from the uterus, pain in the abdomen, back, and in her legs because of compression of the urinary bladder; she appealed to Dr. Tupper for attention. His examination found an inoperable cancer of the cervix uteri. He took a biopsy. The laboratory report reads: "August 1, 1923; tissue cervical. Sections show an atypical proliferation of squamous epithelial cells, which have markedly infiltrated the underlying tissues. *Diagnosis*: Squamous cell carcinoma (epithelioma). Signed, R. G. Owen, Owen Clinical Laboratory."

Examination by the writer revealed a fixed mass involving the uterus, and adnexia in both the bladder and rectal walls. The normal landmarks were obliterated and the mass extended into the abdomen, one-third the distance to the umbilicus. The pelvis was "frozen" by the extensive infiltration. The history showed that she was unable to carry a child to term and always aborted. She was anemic, weak, with a yellowish tinge, and suffered much pain. The changes developed rapidly. Two doses of the Oxidizing Reagent were given in August 1923. In two weeks, definite improvement in pain, bleeding, and pressures were observed. During the twelfth and twenty-fourth weeks, there were reactions of pain, fever, and general achiness, as in the grippe. Thereafter, she normalized rapidly and after the 36th week she had lost all signs of the neoplasm, except that the cervix though healed was deficient on the right side. This was normalized before the first year had passed. Then she was pregnant and in term had a normal delivery of a normal child. Every two years later, she had another normal delivery of a normal child until four were born. There were no more miscarriages, as she had a normally constructed and normally functioning uterus. The pathology was completely reversed. She is still in perfect health over 39 years after being treated.

One sees that the grade of malignancy can be gauged by the rate of recovery--the speed of the reversal of the pathogenesis. *Our Thesis states that the Least Common Denominator of the pathogenesis is of the same pattern as that of the recovery mechanism; it would, therefore, be anticipated that the recovery process would share the characteristics of the pathogenesis in reverse.*

MALIGNANT SYMPATHOGONIOMA

Baby John L, age 13 months, developed a tumor in the abdomen that required exploration on September 25, 1951. A retroperitoneal growth was found, which could not be removed. The biopsy diagnosis read: "Immature type tumor of neurogenic origin, Sympathogonioma." The tumor developed rapidly, thereafter, so

that it caused a visible bulging of the abdomen in two weeks. A mass as large as a grapefruit could easily be palpated in the umbilical region with diameters of ten to fifteen centimeters. The red count was 2,300,000 and the hemoglobin 52%. On October 7th he received 2 cc. of the Oxidizing Reagent containing 2 millimicrograms of the serial system of Carbonyl groups. The recovery was rapid and in a year he was back to normal in all respects. No growth could be palpated. On May 5th, while in good health, he was run over by an automobile and sustained severe leg and abdominal injuries. While in the hospital, he was examined by the same surgeon, who had made the exploration and biopsy. A most careful examination could reveal no trace of the former tumor. He made a nice recovery and is still well. The blood examination on April 5, 1953, showed red cells 4,750,000, Hemoglobin 87.5%. He still remains in good health; Dr. Julian F. Baldor was his physician throughout.

LEUKEMIA

The more typical the case; the quicker the recovery in this disease. The reversal has only the primary disease to overcome. The terminal exhausted case can recover fully, but the secondary injuries, call for more time for complete reversal. The two following cases illustrate:

P. F., age 12, Treated January 8, 1956, by Dr. A. Guzman. The symptoms were classical with subcutaneous hemorrhages and bleeding from the mouth, etc. The red count was 1,500,000 and the white count 232,000 with lymphocytes in great predominance, both large mononuclear and immature forms. The spleen and lymph glands were enlarged, mediastinal dullness increased, weakness, etc., and a rapid decline in health. The Reducing Agent was given in a dose of one millionth of a microgram. The response was rapid with periodic reactions of chills, fever, and general achiness, as characterizes the recovery from cancer. In August 1956, he was perfectly well in every respect. The platelet count was 350,000, the red cells 5,100,000, and the white cells 7,200. This case should be contrasted with the following case of Teddy S.

LYMPHATIC LEUKEMIA WITH TERMINAL EXHAUSTION

This patient was referred to Dr. Julian Baldor in February 1949. Teddy S., age 14 years, with a diagnosis of lymphatic leukemia chronic established by bone marrow biopsy, by Dr. C. The white count was only 15,000 showing the degree of the exhaustion, the hemoglobin was 40%, and the red cell count was 2,150,000 in spite of blood transfusions, which numbered 57. Hemorrhages in the skin and gums were profound and typical, the liver was enlarged, and the spleen was greatly enlarged as were the lymph glands; the weakness, pains in the legs, and the high lymphocyte count six months previously had established the diagnosis, at that time. Since then, he retrogressed steadily until he was unable to walk, was seized with pain and fright, depressed, bleached out, and very weak, fever 102Â°.

He was placed on the usual vegetarian fruit diet at the start of the Treatment; he was given the Oxidation Reagent by Dr. Baldor, 2 cc. of the one to a trillion dilution and all blood transfusions were stopped. The hemorrhagic spots, which were profuse started to subside and change color within a few days; his disposition improved to one of cheer. In nine weeks he was able to walk, had gained twelve pounds in weight, the red cell count was 3,350,000, hemoglobin 52%, and the white cells were 8,000. At the twelfth week, he had a reaction showing slight pains in the legs and a little epistaxis. The blood count then showed 4,000,000 reds, 6,500 whites, hemoglobin 72%; he had gained twenty-five pounds in weight and his spleen, liver, and lymph glands were again normal without even one blood transfusion after Treatment. Thus, the pathogenesis was reversed in every respect.

At the age of 21, he was examined for the military, and classified as IA. He is married and has a healthy child.

The inhibition to the bone marrow’s use of energy was corrected, evidently. ***Other cases of leukemia have conformed to these two extremes in pattern, both in the pathogenesis and in the recovery, no matter which of the two Reagents was used. The best response is not had simply in early cases, and the location does not determine the outcome either.***

For example, in brain cases that are completely diagnosed by exploration and

biopsy, the recovery rate, after being treated in the advanced stages, is five cures out of a series of seven.

In the cases treated as an "official test in 1919" of the five far-advanced, widely-metastasized cases, three were cured and a fourth case, who lived too far away to be examined, sent new patients to the writer five years after he had received the Treatment. So the percentage runs about 75%. This is a sufficient recovery rate to eliminate any delusion that these recoveries are spontaneous from some unknown cause. They are partly spontaneous, no doubt, for the recovery is brought about by the patients' own resources after the pathogen is burned off of the functional mechanism, by one of the two Reagents described. Therefore, it is a matter of inducing a spontaneous recovery and this mechanism is plainly set forth, whereby, the pathogen is burned off of the host cell's functional structure. Prof. Wm. Boyd **(14)** defines spontaneous regression of tumors as, "occurring without a recognized adequate external cause." In Boyd's sense, then our recoveries are not spontaneous, since they were repeatedly induced by adequate external agents. ***However, the removal of the pathogen was induced and the digestion and absorption of the tumors and the healing with normal functioning tissue was not accomplished by external agents, but by the body's own resources.*** So these are induced spontaneous cures. The parasitism was corrected. The normal physiology is restored.

REVIEW OF CARCINOGENESIS AND ALLERGENESIS

As we have seen, the cause of cancer is a multiple affair in which anoxia and two pathogens are the principle actors, and the same pattern holds for the production of the allergies.

The only difference is that in cancer, the basic functional cell unit attacked is the mitotic mechanism for cell reproduction. **We have classified cancer as an allergy of the cell's mitotic mechanism decades ago (*Natural Immunity*, 1934, Christopher Publ. Co. Koch).**

In the respiratory allergies, the secreting mechanism and contractile mechanism's energy producing and receiving FCG's, and their activating double bonds, are concerned.

In the neurological allergies, as epilepsy, compulsory neuroses, and fixed ideas, the conductile mechanism's energy producing and receiving FCG systems, and their activating double bonds, are attacked.

The energy for excessive action of an allergy or neoplasia is not received from the normal sources of oxidation nor even glycolysis as Warburg suggested, for the FCG of energy production and acceptance is blocked by the pathogenic additions.

We conclude that the energy comes from the polymerization of one of the pathogens integrated with one terminal of the FCG activating double bond as a free radical addition.

In the case of cancer and any other allergy, the pathogen is a virus or a polymerizing toxin produced by bacteria trapped in the scar of an old infection where ischaemia protects it from oxidation. This pathogen is the sustaining toxin, which may be difficult to differentiate from a virus, or a bacteriophage living in symbiosis with the germ and paralyzing its activity, instead of causing its lysis. When it gains entrance into the blood stream and into the host cell, its critical double bond adds to the distal pole of the FCG activating double bond, which has become a free radical through addition of the free radical offered by the exciting or sensitizing pathogen to the proximal pole.

The sensitizing or initiating pathogen may be a synthetic carcinogen that has been dehydrogenated by the FCG during anoxia, or the free radical of an incompletely combusted metabolite, a dehydrogenated sulfydryl bacterial product, or a free radical produced by sun rays in the polymerizing units of a maturing pollen. The latter would be the initiating pathogen in hay fever or asthma.

When it adds to the proximal pole of the FCG's activating double bond, the free radical formed at the other pole can co-polymerize with the sustaining pathogen, as just stated, whose energy liberated by polymerization forces, either an excessive uncontrolled mitosis (cancer), or a function, such as an allergy.

The smaller the molecule, the greater the content of double bonds, the more rapid the polymerization, and the greater the amount of energy produced, and hence, the more intense the pathogenic action, whether it is as an allergic affair, or as a neoplasm.

The initiating toxin could be one of the sulfydryl products of certain bacteria, trapped within occluded tonsilar crypts, the apical infection of teeth, or some occluded scarred sinus of long standing. Sulfydryl readily forms free radicals upon dehydrogenation by the FCG; and it also has the ability to add to the double bonds of ethylenic linkages conjugated with Carbonyl groups. It can therefore interfere with oxidations in several ways, for it can inactivate the quinone type co-enzymes as Co-enzyme Q-l0, which is an electron carrier or transfer agent. As when one closes a culture of such bacteria taken from a focus of infection, just mentioned, it soon shows the development of malodorous mercaptans. In like manner, it may also add to the FCG's activating system to initiate pathogenesis.

To show that the focal infection of long standing is a factor in carcinogenesis, a typical case history will suffice. **This woman was then 56 years of age and her case history was included in the Testimony before the Federal Trade Commission in 1943, as a demonstration of the nature of the recovery process after the Koch Reagent was given.** The uterus and most of the pelvis and lower abdomen were involved by 'a biopsy proven' cancer of the uterus; the right breast also presented a massive cancer of the simplex type, which extended into the axilla. There were numerous metastases to the skin, as well, when she received the Koch Reagent in 1938. Recovery was in evidence within three weeks and continued with reactions at the twelfth and twenty-fourth weeks, and by the end of the twenty-fourth week, the absorption of all growths was complete; but an acute, violent, inflammation of the tonsil and lymphatics on the right side of the neck, set in at this time. She could neither swallow nor speak for about a week, then it quickly subsided and she felt very well in all respects. When describing her symptoms, she stated that she had the very same thing happen some 20 years earlier, and her health was never as good afterwards. During that attack, she could not speak or swallow, otherwise, both symptomatologies were identical, except that this recent attack left rapidly, leaving her in exceptionally good health.

Here we have an example of the reversal of the pathogenesis as the essence of the recovery process. The first symptom in the initiation of the disease was the last symptom to be brought to light and its causative pathology cleared away at the wind-up of the correction process.

The interpretation is what we have offered since 1926; During the recovery the de-polymerization of the sustaining pathogen was going on and finally when the growth was gone, the monomeric form of the toxin only was present to produce the same symptoms, as it did when the germ (and its virus) infected the tonsilar area and produced the original inflammation and its subsequent cicatrization.

Both inflammatory reactions to the monomeric form of the toxin were identical, except that the recovery reaction induced by the corrective Reagent burned away completely the infection with its toxins, once adsorbed in the protective scar tissue. These were also burned away, so that the scar tissue became obsolete and was absorbed like the neoplasms, themselves. The correction was therefore complete for no scar tissue was needed after the toxin was burned away.

The completion of the recovery from diabetes with its gangrene conforms to the same pattern. Here the block to FCG's function of energy production and acceptance left the islet cells unable to produce insulin and the evolution of the pathology that followed included bacterial infection of the ischaemic bones, which then underwent necrosis. The recovery process removed the basis for this infection and the infection

left so the bones could be restored in minute detail. The radiographs demonstrate this. The pathogenesis patterns, as outlined here, need not be rigid and must conform to the attending circumstances. They are in harmony with the clinical experience and the established facts of physiology and chemistry, and therefore, are a guide to successful Treatment, which after all, was the goal of 50 years of investigation.

Healing without infection or scar tissue also permits the parenchyma to be redeveloped, so that the injured and defective organs are reconstructed to their normal architecture and function. Thus, the uterus can bear children normally, the stomach can do its normal digestive work, and the bowel can again function, as it was intended to do. Likewise, the bone is restored to do its supportive skeletal work with increased strength and structural density. The complete text should be consulted for more examples.

THE DIET

The diet is completely devoid of animal proteins as meat, eggs, fish, milk, except in cases where buttermilk (Yogurt) is allowed.

Fresh green leafy vegetables, raw or cooked, are preferred. One eats the whole vegetable, the turnips and also the leaves, and the same with cauliflower, beans, radishes, and all other vegetables. They must be well washed; likewise, all fruits may be eaten but are to be well washed. The citrus fruits must be smelled before they are eaten, so as to avoid the terpenes in the outer shell.

Cancer patients should use no citrus fruit until the growths are absorbed, nor should they eat grapes, until the recovery is completed. This is because tartaric acid is a robber. Instead of being burned in the body like citric acid, it combines with calcium and other important salts to carry them out through the urine. However, bananas are an ideal fruit as are apples and one can eat them to their heart’s desire.

Cereals, as whole grain rye, wheat, and oats made into a porridge or bread, fully ground so as to be more digestible, and these should be used plentifully. Wheat germ should be a daily ration as it supplies blood forming materials, trace elements, and vitamins of the B class. It has good regulatory action on the bowel. Since people are often not instructed as to the protein content of vegetables, cereals, and fruits, this should be examined.

PROTEIN FOOD SELECTION

Let us compare the protein content of meats, vegetables, and fruits. One pound of raw, boneless beef or 453 grams contains 84.5 grams of protein. Beef with bone offers 73.5 grams of protein and beef ground into hamburger contains 73 grams. One cup of rye flour, 80 grams, contains 7.5 grams of protein, or about 43 grams per pound. This is about half the protein content of average meat. Besides, it offers the important tissue salts. Nuts carry 9 to 10% protein; milk contains only 3.5%, liver 20%, and dry lentils 25%, lettuce and cabbage about 1.5%. One hundred grams of peanut butter has 26.1 grams of protein. Breads run about 2% protein, Brussels sprouts about 4%, potatoes 2.4%, peas about 23%, beans 21.4%, and nuts 9 to 10%. The actual protein content of easily accessible edible plants is as high as that of meat often enough.

Thus, since the daily requirements of an average size man doing light work is only 0.3 grams per day per kilo body weight, or for 80 kilos (170 pounds), 24 grams per day; therefore, a good bowl of pea or bean soup, a slice or two of bread, a few greens cooked or as a salad would supply all he needs. But one also needs the salts, vitamins, unsaturated fats and carbohydrates. Bran or wheat offers 12.4% protein, 3.4% fat, 4.2% carbohydrates, 7.8% ash, and for each 100 grams, 94 mgms. calcium, 1.312 mgms. phosphorous, 10.3 mgms. iron, 0.37 mgms. thiamine, 0.39 mgms. riboflavin, and no ascorbic acid. Meat, likewise, has no ascorbic acid, but apples carry 5 mgms., bananas 10 mgms., and cabbage 50 mgms. Thus a mixed diet, according to taste without any animal products, will give all the nutrition one cares for or needs; and some articles as beans, peas, and nuts, should be eaten sparingly, especially peanuts because of their high arginine content. Yeasts, the richest sources of vitamins, are taboo because of their high diamine toxin content.

The practical meaning of the vegetarian diet is seen in the cases of the leukemia blood depletion, where 50 transfusions could not keep the blood up to a normal or even half of a normal level, but without even one transfusion, each of these cases gained to a normal blood count only on vegetables, fruits, and cereals, without any medications whatever. Their gain took a few months, but it was observable within one month after the Treatment was given and the diet put into action. Mr. J. K. gained two pounds a day for a month. Mrs. Mac A. did as well and so have countless others.

On this same diet patients have reduced to a healthier weight after the Treatment gave the oxidation they needed. ***So diet and oxidation capacity determine tissue efficiency and health. Nature is always beautiful when unimpeded. It is joyous and rewards one for dietary care.***

Early in our experience, we noted that patients whose homes were in Mt. Clemens, just 20 miles from Detroit, did not do well under Treatment for cancer after they returned to their homes. The recovery process was reversed. This we found was due to the sulfides in the water, and we noted that asparagus that contains methyl mercaptan was as obstructive to recovery, too. The sulfydryl group adds readily to the double bond that activates the FCG of energy production and of energy acceptance, and not only is its function thus blocked, but the addition serves, as we believe, the synthetic carcinogens act to initiate carcinogenesis, as is explained earlier. Very small amounts of mercaptans are physiologically active. Methyl mercaptan is active in one part per 50 billion, and in more concentrated doses, causes blistering and paralytic effects. It blocks the production of rhodopsin in the retina from vitamin A. **Like other sulfydryls, it can inactivate such essential electron carriers as the quinones now known to be oxidation co-enzymes. And they readily reduce Oxidizing Agents to become disulfides. They easily inactivate iodine and thus, cripple the oxidation mechanism and serve as do its sister compounds containing selenium, to block oxidations of surviving tissue slices in the Warburg Chamber.** Needless to say, the diet and bowel hygiene must be guarded against the sulfydryl group, and groups that inactivate sulfydryl, will prove helpful in combating cancer. **Potassium iodide, and such plants as the dandelion, and the chamomile flowers are thus good intestinal aids.**

PREPARATION OF FOOD

Cleansing is important to get rid of the carcinogenic insecticides. Scrub with soap and brush and rinse very well; peel if necessary. Cook in stainless steel, Pyrex, copper kettles, or in the iron pot. Never cook alkaline or acid materials in aluminum, as they react with it and dissolve it, and it enters the body with the food and interferes with important chemical processes in the tissues. Eat moderately; masticate the food well; eat slowly and joyfully.

Water should be pure and free of poisons such as fluorides, selenium, and sulfides that block the tissue oxidations and ferment actions.

DISEASE PREVENTION AND CORRECTION REQUIRE ADEQUATE OXYGEN SUPPLY

After parathyroidectomy, the guanidine bases that formed caused a gelling of the blood colloids visible in the large veins as ante-mortem clots. In the fine tissue capillaries, the gelling blocked the flow of blood and led to the degeneration of the capillary walls and hemorrhage, -- the hemorrhagic glomerulitis in the kidneys and the hemorrhagic degeneration of the liver and brain. But even before the injury had progressed that far, the colloids about and within the tissue cells were also gelled enough to block oxygen transport. **How does this gelling come about?**

The energy required to charge the tissue and blood colloids so they have a correct dispersion, fluidity, and adsorption power to carry oxygen and electrolytes is not supplied when the functional Carbonyl groups that initiate the oxidations for energy production are blocked by condensing with such tightly binding amines as certain guanidines and other amine bases. The sulfydryl group, indirectly, causes the same effect, as described in the text. ***The diminished carrying power for oxygen and reduced fluidity lead to hypoxia in the tissue cells, and then the free radicals formed by the dehydrogenation action of the Functional Carbonyl***

Groups have no molecular oxygen to combine, so they add to the closest double bond at hand, which of course, is the double bond that activates the Functional Carbonyl Group. In this way, the pathogenic integrations with virus, carcinogen, allergen, or incompletely combusted metabolites, are brought about. Where do these toxic amines originate?

They arise for the most part in the filthy colon, as a result of a diet on animal proteins. Old focal infections in the tonsils, appendix, fallopian tubes, or other areas, especially at the roots of dead teeth, are very often to blame. **To defend the tissues from pathogenic anoxia, one must remove all such foci of infection and wash the colon free of its toxic contents.** The diet must be changed to clean fruits, vegetables, and cereals, without any animal proteins whatsoever, as the Good Creator ordained. ***One great function of bacteria is to convert dead animal tissues into food for plants, whether the dead animal product is in the ground or in the colon. And it is certain that a large part of the ingested meat is not digested and absorbed completely before it reaches the colon. The toxins evolved paralyze the bowel wall and cause diverticulae. These hold putrid material for varying periods among which the toxic amines are transition bodies. Diamine oxidase present in the intestinal wall and liver combats them, more or less successfully. But in the presence of excessive sulfydryl groups also formed during the putrefaction, the diamine oxidase may also be inactivated.***

After the pathogen, be it a virus, carcinogen, or some allergen, has made the pathogenic integration, the need for oxygen in the diseased cells is all the more imperative, and removal of all sources of the pathogenic amines is the prime consideration. And, not until a good dispersion of the tissue colloids is had and a good oxygen supply is present in the diseased cells, should the Oxidative Reagent be given. For if it does not have a molecule of oxygen at hand to combine the free radical formed by each dehydrogenation, there will be no curative progression of oxidation, and the Reagent is given in vain. This also applies to the free radicals produced by the use of the Reducing Agent. So the first thing to do is clean the colon and remove any old focal infection that is practicable. Plenty of water must be used for thorough cleansing. At least two lavages are had per day.

To cleanse the colon a solution of 1% of sodium chloride or of sodium bicarbonate should be used. At least a liter should be held by the colon and it should be so manipulated that the fluid passes over into the cecum where most of the putrid material is held, often in diverticulae. But in old chronic cases of constipation, the crypts of Lieberkuhn are jammed full of fine sand-like deposits that hold the germs that develop the poisons. The bowel should be expanded by the enema to open these crypts and let their contents out.

It may take from 4 days to 2 weeks to get the colon clean when one is taking no solid food whatsoever, but only liquids as watermelon juice, sugar cane juice, prune juice, apple, or pear juices. Grape juice is not used and citrus fruits are not either, for reasons explained earlier. Vegetable juices are easy to prepare now with the modern kitchen appliances. Cabbage juice, carrot, and beet juices are very desirable. The variety is large. However, each object must be scrubbed absolutely clean with soap and water to get rid of all insecticides.

After a few days of this regime the patient feels better, and is happy to continue. Pre-malignant growths in the breast have been seen to disappear in two or three weeks on this regime, and even the pain from deep irradiation burns that scarcely yielded to large doses of opiates, has greatly improved. ***In other words, nothing is lost by following the regime and great gains are made aside from the preparation of the tissues for the action of the Reagent.***

Prevention and cure of disease is not so mysterious as orthodoxy assumes. They depend upon definite chemical processes that are understandable and controllable over a broad range. They involve a new conception of the oxidation mechanism within the tissue cells, as dependent on the phenomena of the Free Radical and the Double Bond without which the pathogenesis of the baffling diseases could not be interpreted, but which as we have shown introduces a new approach in pathology and in therapeutics. Just as the chemistry is most fundamental,

so is the recovery process in its cyclic nature, the perfect healing, and the return of function, as even the most dangerous germs have lost their pathogenicity.

BIBLIOGRAPHY

1. KOCH, W. F., *J. Biol. Chem*. 12, 313 (1912) *J. Biol. Chem*. 15, 43-63 (1913)

KOCH, W. F., *Medical Record of New York*, Oct. 30, 1920.

2. PATON, *Quart. J. Physiol*. 10, Nos. 3 and 4 (1917)

3. KOCH, W. F., "*The Survival Factor in Neoplastic and Viral Diseases*" Grafica Laemmert, 1955, The Manney Co., 1958, The VanderKloot Press, Detroit, 1961

4. LOHMANN, K., *Biochem.* Z., 271, 264 (1934)

5. ENGELHART, *Yale, J. Biol. and Med*., 15, 21 (1942)

6. WARBURG, Otto, *Narurwissenschaften*, Vol. 42, p. 401 (1955)

7. KOCH, W. F., *Cancer Jour*. (Phil.) October, 1924

8. JOSEPHSON, *Emanuel, Myasthenia Gravis, Manganese and the Thymus* (1961) Chedney Press, New York

9. TREIGER, JAYME, *Revista Fluminense de Medicine*, Vol. 25*,* No. 3, 3-24 (1961)

10. TREIGER, JAYME, *Second Annual Brazilian Congress on Cancerology* (1961)

11. TREIGER, JAYME, *A Folia Medica*, Vol. 44, No. 1, 9-21 (1962)

12. COLOMBO and CARNEIRO, *Veterinaria,* Vol. IV, No. 1, 2 1-33 *(1950)*

13. CARNEIRO, *Veterinaria*, Vol. V, No. 1, p. 75(1951)

14. BOYD, WM., *Proceedings of the Second Canadian Cancer Research Conference*, p. 354 (1956)