THE SURVIVAL FACTOR IN NEOPLASTIC AND VIRAL DISEASES

KOCH

A STUDY OF THE PHENOMENA OF THE FREE RADICAL, THE DOUBLE BOND, AND ITS ALPHA PLACED HYDROGEN ATOM IN THE PATHOGENESIS AND CORRECTION OF NEOPLASTIC, VIRAL AND BACTERIAL DISEASES.

ABOUT THE AUTHOR

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Articles and publications writ-

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ten by Dr. Koch include: "Toxic Bases in the Urine of Parathyroidectomized Dogs", Journal of Biological Chemistry, Vol. XV, No. 1, July 1913; "A New and Successful Treatment of Cancer" (Preliminary report), Detroit Medical Journal, July 1919; "A New and Successful Diagnosis and Treatment of Cancer", Medical Record of New York, October 30, 1920; "The Function of Cancer", Cancer, October 1924; "The Causes and Prevention of Cancer", 1926; and "Clinical Demonstration of the Laws of Chemical Structure that Determine Immunity to Disease, and their Application in the Treatment of Patients", 1939. Other books in the Natural Immunity Series written by Dr. Koch include "Cancer and Its Allied Diseases", 1929, revised in 1933; "Natural Immunity", 1936; "The Chemistry of Natural Immunity", 1939; and "The Survival Factor in Cancer and Viral Infections", 1958.

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NEOPLASTIC AND VIRAL
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An Introduction to
Carbonyl and Free Radical Therapy

A Study of the Phenomena of the Free Radical, the Double Bond, and its Alpha Placed Hydrogen Atom in the Pathogenesis and Correction of Neoplastic, Viral and Bacterial Diseases

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DEDICATION

This book is dedicated to the memories of two leaders in American Science and Industry, Dr. Willard H. Dow, and Dr. William J. Hale. Their humanitarian genius was great enough to build the vast Dow Chemical Company to its present proportions and service, and also take interest in other humanitarian efforts, such as our own, which they investigated fully, evaluated carefully, and then supported effectively in our court battle.

ACKNOWLEDGEMENTS

Gratitude is due Professor Joseph Maisin, of Louvain University, for his many experiments in small animals with and without the writer, from which conclusions of fact could be drawn.

Likewise gratitude is due Dr. Willard Dow, Dr. William Hale, Dr. Drake, Dr. Rubens and other Dow scientists for every help in every need, especially for their winning defense against United States Government attacks instigated by competitive drug interests.

FOREWORD

The world's leading surgical journal, the London Lancet, gave an editorial review of the present status of surgery in the treatment of cancer. It gave the same conclusions as did Sir James Paget a century ago, when he stated in his text on Cancer that this is not a surgical disease, that the condition was profoundly constitutional, and that operated cases did not live as long on an average as those that were left untouched. From 1910 to 1950, the American Cancer Control Society created an energetic propaganda that 85% of breast cancer could be cured surgically or by irradiation, and that early diagnosis was a prime advantage. Now after the statistics are analyzed the Lancet quotes the world's leading surgeons on the results of early operation with the same discouraging conclusions as Sir James Paget stated a hundred years ago. In the meantime life insurance statistics and others established the fact that operated cases, the early cases, lived less by two and a half months than the inoperable, far advanced cases that were not operated. Add to this two and a half months the year or so it took the early case to become inoperable and advanced, one sees that surgery done with all its courage, sacrifice and dexterity is not the attack that is required to win against this disease. The Lancet states, "The intensive campaigns to awaken the public to keep on the watch for tumors and report for the earliest possible diagnosis and treatment has met with good response, but the anticipated drop in the mortality rate did not follow." "Despite a long and intensive educational program for the early detection and treatment of cancer, the death rate from cancer of the breast *shows no downward trend.*" In fact, "The comparative mortality index, which allows for changes in the age structure of the population, shows for men a rise of 6% in cancer mortality between 1938 and 1950." "The size of the primary tumor is no guide to curability; two-thirds of patients reporting with tumors of the breast which were smaller than a hazel-nut already showed metastases," and with regard to lung cancer, "If recent experience is typical, however, by the time definite abnormality appears in the radiograph, most cases of pulmonary cancer have progressed too far for successful resection." "Survival rates after simple excision, radical mastectomy, and irradiation, are depressingly uniform." "Our basic approach may be wrong; the attempt to treat cancer as a local disease rather than a general disease, may be as irrational as treating syphilis by excising the primary chancre." "In most if not all lethal breast cancer, remote spread takes place by the blood stream before interference is practicable." "The survival rates after different periods of delay before seeking medical advice often shows a curious paradox. Thus Swynnerton and Truelove reviewing 395 cases of gastric carcinoma, showed that the greater the delay and the longer the history of symptoms the greater was the survival rate." Here we find in the Lancet of April 3, 1954, p. 714, with other statements of similar import, the

conclusions of the world's most advanced surgeons. A year later Dr. George Crile of the famous Crile Clinic in Cleveland gave thorough information to the profession and the public on this subject and was in exact agreement. Now comes the report of the 12th annual scientific meeting of the Detroit Institute of Cancer Research. The concensus was the same, Dr. Harden B. Jones, professor of medical physics at the University of California, gave the ultimatum, "The odds for or against the recovery from cancer are set long before the patient sees a physician." and "There is no evidence that treatment by surgery or radiation, the *only recognized methods of therapy* affect the course of the really malignant forms of cancer." and "Early treatment is a nice theory, but there is no evidence that it benefits the patient." "Some drastic cancer therapies not only do not help but are harmful." "The tumor easily could have a billion cells before it is large enough to be recognized as cancer. Some of these cells are already in the blood stream."

Unfortunately radiation does not answer the needs of the patient, but adds to the basic pathology. The convention of the American Roengten Ray Society of September 1954, added to the report of the Roengtenologist of the University of Pennsylvania in 1925 when he stated that irradiation before and after surgery opened the vascular and lymph spaces and helped the spread of the disease instead of retarding it. His report was so unpopular that it was suppressed. But today the statistics are so disheartening that even the radiation therapists are bold in reporting that where deep therapy is poured into a neoplasm of one type, a more malignant form or a bone sarcoma is created underneath only too often. That the Survival factor is destroyed by irradiation is seen in the hereditary defects in the offspring of radiologists. These show in some 10,000 children, of radiologists, twice the incidence of cancer and more defects in eyes, heart and blood, than in children of physicians not exposed to irradiation. Eight to ten times more radiologists die of leukemia than general practitioners. When one recalls that viruses are thousands of times more resistant to irradiation than tissue or cancer cells, the situation is logical.

Fifty years ago nothing was known about cancer except the diagnosis, which was about all there was to become expert in. The gross and microscopic pathology was so well learned that the resort to the biopsy was regarded as a sign of poor training (Ewing) (Warthin). Our professor of pathology insisted that we make 100% correct diagnoses and give the microscopic description from the gross findings alone. Every surgeon on the University staff did it regularly.

Today, however, high specialization makes the biopsy an essential for many. For many years ahead of my day, all that was known beyond diagnosis was that cancer was caused by "irritation." But no one knew exactly what "irritation" meant, or how it operated to cause cancer. Further, there was no information to serve as a starter to investigate the problem. But still the walks through the hospital wards fervently cried for the solution. The surgeon was doing his untiring best and the radiologist hoped and hoped that his approach might some day prove fruitful. And yet no facts stepped forth to

show how to even make a start, — nothing from within the cancer properties themselves.

So the writer decided it might be helpful to get the basic facts on any of the deepest injuries to the body chemistry that could be produced, observe their effects on every tissue quality possible, and then figure out how any of these changes might take part in the pathogenesis of cancer. The effects of complete parathyroidectomy were chosen for this purpose, largely because the great experts of that day on this very subject seemed to have overlooked the main factors in parathyroid insufficiency, and because a subject as important as that should be at least reasonably explored.

As the writer's investigations progressed in accumulating more data it began to appear that he made the correct start. The findings were carefully evaluated, the conclusions drawn, and from these a postulate was formulated and tested out in the broad field of disease. It was hoped that if the venture would be propicious, a century of ignorance would be hurdled, and a basis for investigating the cancer problem itself would be reached. A landing in barren territory simply called for a fresh start and another trial. However, the first attempt proved fortunate. Our postulate had been drawn up with every effort at precision, and the conclusions were fruitful. Under the circumstances this was even more important than if our interpretations were correct or "true." For the aim was to reach a position of utility.

The utility has two leading aspects. One lies in the proof that the four primary cell functions — contraction, secretion, conduction, and cell division — are provided with energy that is produced and received by each functional unit in accord with one and the same pattern, and when interrupted so as to produce disease, the fault is the same in pattern and subject to the same type of correction by one and the same atomic structure. The other phase of utility is the explanation of both viral and neoplastic parasitisms, the atomic bondings and electronic displacements that constitute the integration of the pathogen with the host cell, which not only accomplish the pathogenesis, but actually provide for and invite the oxidative cleavage that leaves the host cell in normal functional status, perfectly reconstructed, and the virus no longer to be found. The text demonstrates this as well as the fate of the neoplastic cell and the process by which it is disposed of. These matters are based on firm chemical laws as the text will show.

So whether the cell contractile fibrillae as in asthma, or the secreting fibrillae as in hay fever, or the conductile fibrillae as in a compulsory neurosis, or some other phase of insanity, or the mitotic fibrillar system as in neoplasia, happens to be attacked, the basic pathology is the same and its correction is necessarily the same, too. This is the subject we will demonstrate in this text. We have no thought that our presentation is the best that could be made. However, since we have opened the door and uncovered the mysteries it enclosed, it is our chore to make the disclosure. This door stands open for endless investigation and for collaboration as well. It should be inviting for our proofs of the cure of the many forms of cancer offered in this text stand

firm, firstly in their diagnoses made by America's leading surgeons, with the patients housed in our proudest institutions where every facility for a firm diagnosis was at hand. Then, too, the clinical diagnoses were confirmed by our foremost pathologists. Secondly, the cures were demonstrated to be permanent with reconstruction of tissue so good function was restored, and accomplished by a definite process without leaving even a microscopic trace of cancer cells.

It will be seen that whether the correction happens to be in far advanced cancer of the vital organs, widely metastasized, and the patient in extremis, or the correction happens to be in the terminal phase of rabies, hog cholera, or some other 100% fatal viral disease, the reversal of the pathogenesis follows the same definite order. This physiological aspect of the correction, we will attempt to show, depends upon well proven laws in chemistry that are basic to tissue cell energy production and energy use, and primarily basic to all vital processes. Thus a least common denominator in pathogenesis and its correction has been reached. It serves as a key to the interpretation of disease production and also to its correction in the whole field we have investigated so far.

CHAPTER I

THE POSTULATE

The initial work that spearheaded the Survival Factor Investigations was a research into the cause of the convulsions and deaths that always followed complete parathyroidectomy. The findings were published in "The Journal of Biological Chemistry"—12; 313, 1912, Koch, and 15; 43-63, 1913, Koch. This work was confirmed by Prof. Patton and his staff at the University of Glasgow, and published in the "Quarterly Journal of Physiology" in 1917 using two of the four numbers. For the care and excellency with which the confirmation was made, Patton was awarded the Triennial Prize in Medicine by Harvard University. This confirmation is of great importance because of the broad field of applicability of the facts brought forth and also the depth of their interpretations of disease processes.

The cardinal facts were just three:

(1) guanidin, methyl guanidin, and some other toxic bases were produced in the tissues and eliminated in the urines in fatal amounts that increased until the dog died in convulsions;

(2) calcium, lactate, and phosphate were eliminated in excess;

(3) the post-mortem findings showed antemortem coagulation of the blood in the large veins, and hemorrhagic degenerations of the liver and kidneys.

From these findings, several important conclusions were made, based upon:

(1) the chemistry of guanidin and its derivatives showing the activation of its amine group by its conjugation with an imide group, and also the tendency to deactivate this amine group by such substitutions as acetic acid as in creatine and amino-valeric acid in arginine;

(2) the fact that guanidin and methyl guanidin are highly toxic while creatine and arginine are not;

(3) the large elimination of lactic acid even while the lungs were well ventilated showing that fuel was not burned via an oxidation process, but was fermented hydrolytically to produce lactic acid, and, thus, the oxidation mechanism was blocked at its very inception;

(4) the block to oxygen transport in the blood and inter and intracellular fluids by the antemortem coagulation or gellation of tissue colloids that depended on the lack of energy production via oxidation to keep the colloidal particles charged on their surfaces and hence a failure in their dispersion; the resultant failure in oxygen transport further blocked the oxidation mechanism in a vicious circle;

(5) the fact that the oxidations were blocked by an amine group of guanidin, that dehydrogenation is the first step in oxidation, that the

carbonyl group is a good dehydrogenator, and that it can be inactivated by condensing firmly with an amine group as in guanidin, but functions normally by condensing with a weakly activated amine group as in creatine to form an azomethine double bond.

The conclusions are the following:

(1) after Parathyroidectomy, the activated amine group of guanidin and methyl guanidin condensed with the carbonyl group of the cell's energy producing mechanism for function (FCG) to form a firm azomethine double bond, and thus prevented it from initiating oxidations in fuels or toxins that came into the field;

(2) the failure to oxidize made it impossible to charge the tissue colloids, and so they precipitated as a gel and did not flow through the capillaries and tissue spaces or even through the large vessels to carry oxygen to the intracellular mechanisms, so an anoxia or hypoxia was secondary to the inability to start oxidation chains;

(3) that the oxidations of highest quality are chain reactions started by dehydrogenations, free radical production, addition of molecular oxygen to the free radicals to form peroxide free radicals, that carried the oxidation chain, or caused molecular cleavage into parts with terminal carbonyl groups that promoted further oxidation;

(4) that the Pasteur effect was a function of this tissue cell functional carbonyl group (FCG) and was suspended or destroyed by its condensation with tight binding amines as guanidin, but its function was supported by condensing with the weakly binding amine group of creatine after the hydrogen atom the FCG removed from fuel was passed on to an appropriate electron acceptor;

(5) that the Creatine-FCG azomethine bond is held until the energy developed as the oxidations progress caused phosphoric acid to enter this azomethine bond, combine the creatine and liberate it as a high energy carrying phosphate, and that the burning of fuel is regulated by the factors concerned, — the FCG, creatine phosphoric acid and stored energy;

(6) that toxic amines of various metabolic, bacterial, viral or of fungal agents (present day antibiotics may be included) are able to make the same crippling condensations with the FCG that no metabolic measure is able to break, and thus disturb the physiology in various ways;

(7) that to dislodge such toxins it is necessary to oxidize them away as no adequate hydrolytic provisions are available;

(8) that the double bond of the azomethine condensation activates the hydrogen atom of the carbon placed alpha thereto to provide for its easy dehydrogenation, and thus start an oxidation progression via free radical, peroxide free radical, and cleavage that burns away

the amine group of the toxin or other pathogen, and restores the host cell functional carbonyl group;

(9) that the FCG is activated to be the preferred dehydrogenator through conjugation with the double bond of an ethylene linkage, that contributes electrons to it; additions to this linkage must destroy its activating powers.

We shall see how these conclusions fit into the pathogenesis of cancer and viral infections, and determine the controlling therapeutic measures. It will be seen also that they explain the long unsolved Pasteur Effect, which we hold to be a splendid demonstration of the presence and action of the Functional Carbonyl Group, the FCG, as we designate it for short, and a solid proof of the correctness of our working hypothesis and postulate.

CHAPTER II

VIRUS AND CANCER CELLS

Cancer cells and viruses are both parasites; that is they have to depend upon sources of energy and material that belong to other usages to conduct their characteristic activities. The virus cannot produce the energy it needs for its vital processes, so it gives signs of life only so long as it is integrated with a living source of energy and food which it diverts to its own ends. The cell it preys upon, the Host Cell, is killed thereby. The cancer cell is not able to perform the functions it was created to do for itself or any of the other cells of the body to which it belongs and is responsible. It has lost its capacity to conduct oxidations, and also the mechanisms that use the energy of oxidation for useful work. Instead, a low grade process, wasteful fermentation, is used to produce energy. This energy is transferred to the mitotic mechanism, where it forces cell division, as it has no functional mechanism to use it where its production is normally controlled by the demand for the function. The mitotic mechanism thus becomes parasitic upon the rest of the cell and the body as a whole.

Viruses may cause normal cells to go neoplastic: maybe all cancers require them. Several hundred synthetic substances are known to cause cancer. It has not yet been decided if or not viruses play a part here, too, but many cancerologists think so. It will be seen how the synthetic carcinogen may prepare the way for the virus to integrate with the mitotic mechanism and complete the neoplastic change.

The institution of parasitism within a cell, be it viral or neoplastic is a complex affair that depends upon a disposing cause besides the particular virtues of the pathogen to show its specific action. Of these anoxia or hypoxia is a leading factor. With plenty of oxygen available as normal structure determines, there would be no pathology if the oxidation catalysis were adequate, and logically enough, it happens that an adequate oxidation catalysis prevents oxygen deficiency in any tissue. This fact will become apparent as we go along. So the key to the correction will be seen to be the restoration or provision of an adequate oxidation catalysis. The initiating act in the oxidation process, namely, dehydrogenation is a main subject of this book. Then there is the subject of the environmental factors that have contributed to the block in the oxidation catalysis. These are discussed also in the hope that a good working picture of the matter is at hand.

NATURE OF VIRUSES

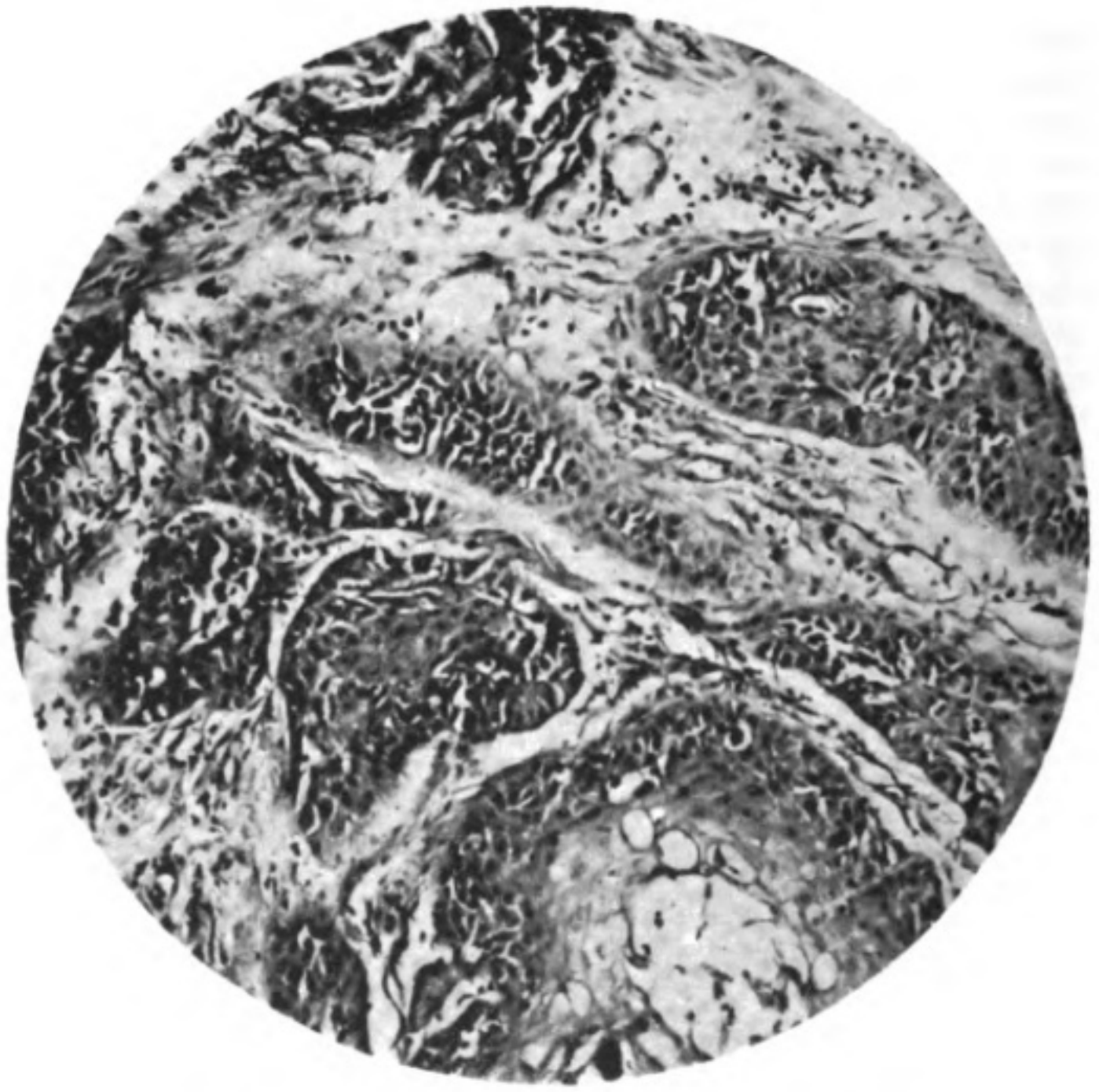
The electronic microscope and bacteriophage studies have yielded rich information on this score. Their sizes vary from 8 millimicra, the size of a protein molecule, to 175 millimicra, the size of the smallest bacteria. Each type has its own shape and these vary from rod to spherule in form. The essential structure is a protein capsule within which lies a nucleoprotein mass.

They are specifically cytotropic obligative parasites and always pathogenic. The protein capsule has specific antigenic powers that yield specific immunological responses, and serological reactions. The latter, for example, serve the differential diagnosis of non-paralytic Polio from La Grippe and other viral infections. This is the part that is convertible into a vaccine used to excite immunological reactions in the patient. There is no immunological response to the nucleoprotein part though this is the part that causes the pathology. The protein capsule protects the inner part and carries it to the cell where it attaches to its outer surface and injects the nucleoprotein content into its victim. An acquired immunity inactivates the capsule and prevents the attachment to and hence the penetration of the host cell. As soon as the material is injected, it breaks up into a myriad of similar units of nucleoprotein as by a depolymerization process, and each particle unites chemically with a nucleoprotein particle of the host cell, and thus integrates or becomes one with it as by a co-polymerization act. The host cell particles with which they integrate are the "grana" that perform the vital functions of the cell and produce the necessary energy thereto by oxidation. As the virus shunts this energy into itself and uses it with host cell material for its vegetation, the host cell grana break down and are used up to form the viral colony which is soon a mass of provirus ready to mature and break forth from the dead host cell as infectious parasites.

It is the concensus among experts that once the nucleoprotein of the virus has penetrated, integration with the host cell is complete within a minute and a half, and no amount of vaccine, antitoxin or other serological effort can separate the virus and rescue the host cell. It is doomed. On the other hand, we will show how the bondings we postulate as forming the integration actually invite oxidative cleavage in a way that leaves the host cell in good functional status while the virus is no longer to be found. In this process the energy taken from the host cell to serve viral vegetation is returned to it to support its reconstruction, while the virus undergoes a stepwise oxidation. This explanation is based on the repeatedly observed fact in the cure of Rabies, that when the reagent is given to promote the oxidative destruction of the virus, in an animal that has only maybe 12 to 24 hours to live, a period of 72 to 84 hours are required for the paralyzed victim to be free of paralysis and the affected nerves to be functioning normally. We have proved this restoration not only in terminal rabies, but in paralytic dog distemper, paralytic hog cholera, and paralytic Anterior Poliomyelitis. The same chemical corrective measure was used in all and fit the pathology in all. This is taken to mean that the state of integration of the host cell and the virus presents the same atomic bondings in all instances. The same corrective attack was employed in the sensory nerve atrophies with loss of function, and in neoplastic disease with success, so it is concluded that the state of integration of the host cell and the pathogen is of the same order in all, and a least common denominator in pathogenesis and its correction has been established in a broad field.

Though the atomic bondings that constitute the integrations are of the same order, the states of integration vary. Thus in "Polio" an acute lytic type

PLATE I

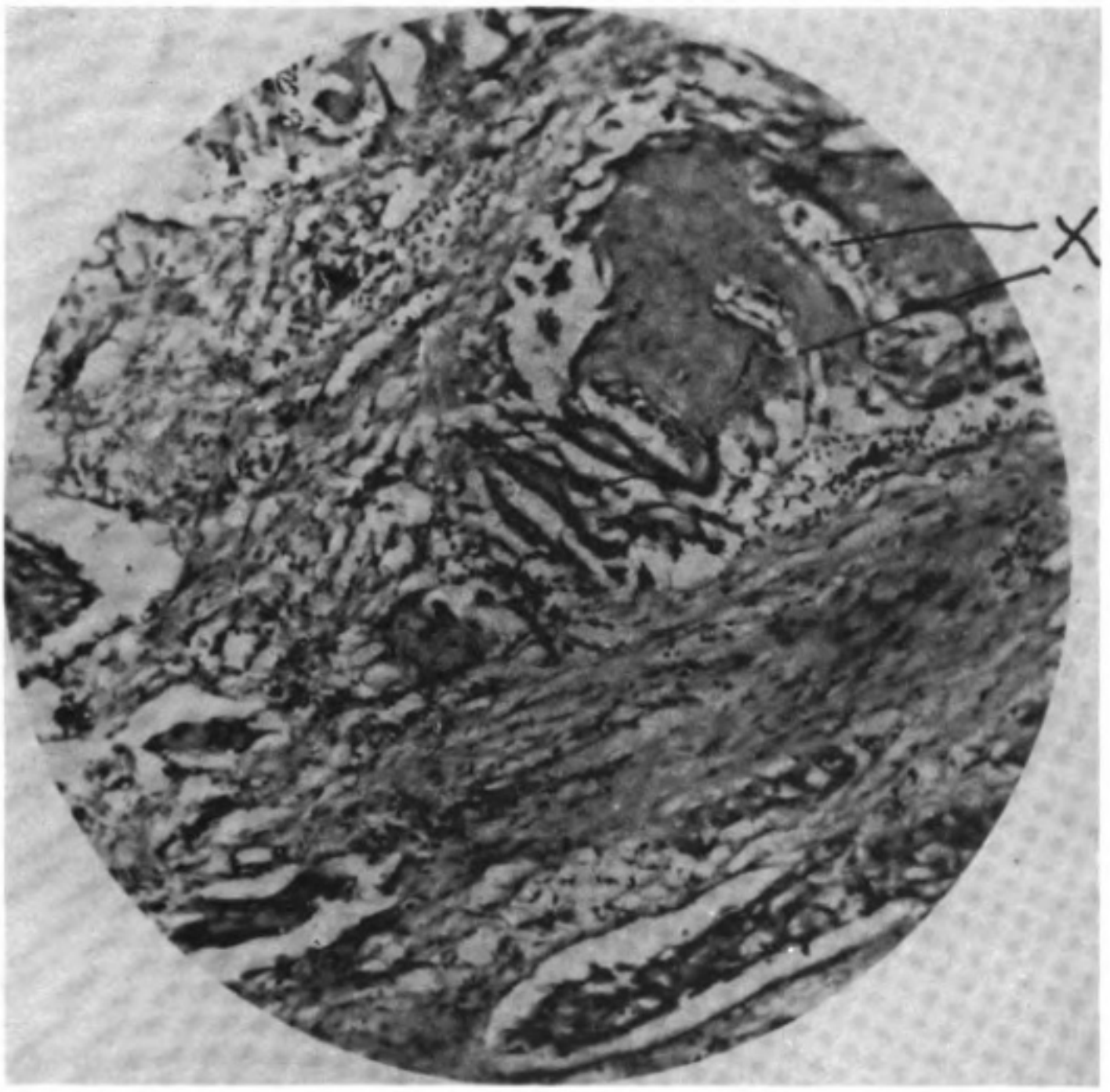


A.—Squamous cell carcinoma of the neck biopsy before treatment. (150X)

causes the death of the host cell in hours or days as a rule, while a prolonged symbiotic type may cause extensive paralysis and atrophy that invalids the patient for many years or until death. And yet the host cell is not dead, but as its energy producing mechanism is paralyzed by the integration with the virus, it cannot function any more than a dead cell and the results appear the same. We have found by trial, however, that such cases can be freed of their offending virus and the host nerve cell returns to normal in function and so the atrophied muscles again get impulses to contract and rebuild themselves. Cases that were extensively paralyzed and atrophied for three years have required three months to be restored to 95% or more of normal in function and muscle reconstruction, while a case extensively paralyzed and atrophied for over twenty years has required two years to be restored to 95% of normal, functionally and structurally. Since nerve cells do not reproduce, the cleavage of the integration is proven by these tests.

Since the lytic type of infection only takes days or only hours to kill the host cell, the quicker the patient is treated the better the chance to have living cells

PLATE I



B. — Taken from the same tumor several weeks after treatment, showing the calcified coagulated hyalinized debris, into which angioblastic tissue is growing with an area of liquification preceding each ingrowing bud of angioblastic tissue.

freed from their viruses, and get complete recoveries. The case records illustrate these situations.

In neoplasia the integration of the virus or synthetic pathogen or bacterial toxin with the host cell is originally with the functional grana energy producing mechanism, until the latter is destroyed in building up the virus and nuclear material integrated with it and undergoing mitosis. This is the only mechanism left to accept energy produced by an uncontrolled process of fermentation, and so neoplasia is the expected result. The details will be discussed later, where it will be seen that the carbonyl groups that institute energy production and storage as ATP, and creatine phosphoric acid, are like those that mediate the transfer of this energy to the working mechanism and both are paralyzed by condensation with firmly binding amines, of virus or other carcinogens. The virus bound in the atrophic muscle of "Polio" we picture as similarly integrated. The integrations may exist long periods before actual destruction of

the paralyzed functional mechanism is accomplished and, during this period, liberation of the cell from the pathogen can restore it to normal. For a period the pathogenesis is reversible therefore in viral infections and the same holds for cancer. This situation is exemplified in the microphotographs (A) and (B). (B) shows the destruction of the cancer cell after the pathogen is removed and the cell residues can function no longer. They then undergo calcification and digestion like a blood clot. Plate I (Medical Record of New York, Koch, October 30, 1920).

VACCINE PROBLEMS

From what was stated so far it is seen that vaccines for a specific virus do not immunize against the nucleoprotein that is the actual pathogen, especially after it has penetrated and integrated with the host cell, so to talk about curing cancer with vaccines or immune sera is a waste of time. Even the prevention of viral infection by vaccines is meeting the strongest statistical opposition since large scale small-pox and Salk vaccinations have been recorded. In line with what is known about vaccine structure, statistics appear logical when they show that paralytic "Polio" is increased both in incidence and fatality by use of the vaccine. One may compare various regions of different climatic conditions for the data. In all of these the Salk vaccine was enthusiastically applied, in greater number each year, and the incidence increase was tremendous each year, whereas, if the vaccine were effective there should have been at least a little statistical improvement. In Montreal, generally cool, they reported on August 27, 1959, 521 cases with 27 deaths, just while the "Polio" season was getting well under way, as compared with less than one hundred in 1958. In Ottawa, generally cool, 455 cases with 41 deaths were reported on August 22, 1959, as compared with 64 cases with 7 deaths in 1958. In all of Canada, even before the epidemic started to decline, there were 7 times more paralytic cases in 1959 than in 1958, with a greater death rate. In Detroit, much warmer, where vaccination was thorough, the number of cases in 1958 was 697, against 226 in 1957. In the District of Columbia, still warmer, the Health Department reported 7 times as many cases in 1958 as in 1957. In New Jersey, in 1958, the Health Department reported twice as great an incidence as in 1957. The United States Public Health Service reported an increase of 15½% of paralytic cases in 1958 over those in 1957 (49% against 33.5%). In Hawaii (tropical) there were 65 victims including 32 paralytic cases in 1958; half of these paralyzed cases (16) had received three Salk shots, in an island where 60% had been vaccinated. In 1957 only 25 and 8/10ths% were paralytic instead of 49 and 9/10ths% in 1958. If the vaccine were effective there should have been a 60% decrease in the incidence in the whole island of the paralytic infections, instead of an increase of nearly 100%.

Nationwide statistics issued January 4, 1960, by the United States Public Health Service, show that for the year 1959, up to December 26th (51 weeks), the increase in the incidence of Polio rose 85% over that of the same period of 1958. There were 8,531 cases listed for 1959, of which 5,661 were paralytic, as compared to 5,987 in 1958, of which 3,090 were paralytic. We just showed

the great increase in 1958 over the incidence of the total and the paralytic cases of 1957. Where compulsory vaccination was practiced as in North Carolina and Tennessee, Bealle's investigations report a 400% increase in paralytic and non-paralytic Polio during 1959 over 1958. So it seems that the more vaccine that is used the more the actual infection that comes about. The statistical analysis teaches much about the nature of the virus.

Of course, this is comprehensible when one considers that the virus breaks up into its component units on penetrating the host cell, as if by a depolymerization process, and it grows by acquiring new units to add to each, as by a copolymerization process. Some investigators compare the viral structure to a deck of cards. The complete deck or complete virus with all its units is the parent pathogenic killer type. The vaccines may be regarded as incomplete decks, with not all the units required to make up the full killer type. Now, if a person carried vaccine units of, let us say, half or less than the killer type requires and another vaccination or infection by a crippled non-fatal virus comes along that presents the units missing in the protective infection or vaccination of a previous period either one of which alone can not produce the disease, the units all added up could constitute the complete killer type, and it has been shown that they are "shuffled" in at random to make up the full virus, vaccination may add to the incidence of serious or fatal infection, and the more the vaccination the more the chance for building fatal viruses.

This happened in the writer's early practice (1920). Two cases were vaccinated against small-pox from the same vaccine lot. One had no effect. The other came down with a rapidly fatal small-pox. There was no epidemic at hand in Detroit at the time, so it was concluded that the fatal case's innoculation carried units required by a previous silent infection to make it fatal.

SMALL-POX

Statistics on vaccination against Small-Pox in the Philippines when the United States took over are instructive. Reports run thus: In 1918, the Army forced the vaccination of 3,285,376 natives when no epidemic was brewing, only the sporadic cases of the usual mild nature. Of the vaccinated persons, 47,369 came down with small-pox, and of these 16,477 died. In 1919 the experiment was doubled. 7,670,252 natives were vaccinated. Of these 65,180 cases came down with small-pox, and 44,408 died. One sees here that the fatality rate increased in the twice vaccinated cases. In the first experiment, one-third died, and in the second, two-thirds of the infected ones. This speaks for the retention of viral units from the previous vaccinations, and indicates that, in the vaccine the shuffling in of units varies in different specimens of vaccine. It should be stated also that every epidemic of viral disease treated by the writer followed vaccination within a few months, when protection should have been had instead of an epidemic. This was so in Brazil, in Aftosa, Cinemosa, Hog Cholera and Rabies, and in Cuba in Hog Cholera.

The question arises then as to how one accounts for the decrease in the incidence of small-pox, since vaccination was instituted. The question is not

easy to settle, since the hygienic improvements in sewage disposal has wiped out the means of spread of intestine carried viral infections. In the great small-pox days, excreta were thrown out of the window into the streets, then the outhouse was invented with its flies, etc. Today modern sewage is an obvious advantage, and soap and water are available even for washing the fingers of cooks and waiters in restaurants, and inspections by Public Health Officers help greatly in keeping down the spread of infection. It must be recalled that viruses integrate with bacteria and when these form spores, the integrated virus shares the protection of the spore against sterilization by chemicals and heat. They can thus survive for many months or years with full virulence. The intestinal tract is known to be a favorable habitat for such integrated viruses, so the hygienic measures of today would wipe out small-pox anyway without the benefit of vaccination, if there is any when carried on commercially. The present-day kitchen garbage disposal sink apparatus has cut down the incidence of the house fly so much that its universal adoption should become the greatest health booster of the century. In the writer's experience, vaccination is a laboratory success when the technique is correct all the way through. Commercially the statistics do not look so favorable when other variables are encountered.

CHAPTER III

CANCER

Glover showed in 1923 that the cancer virus existed in a pleomorphic germ that was bacillus in one phase and coccus in another, and virus in the third phase. He also showed it could exist in a fungus or micelium phase. The latter form has been identified lately by Irene Diller, and some others, and the whole chain of forms was independently proved by von Brehmer, in the last few decades as well. The work was thoroughly repeated and proved by my friend Jacob Engel and George Clark, at the U.S. P.H.S. laboratories, but, for reasons we will not discuss, they were not allowed to publish their findings. The infectious nature of natural cancer was thus proven beyond any doubt by carefully following the four laws of Robert Koch. Doctor Clark was able to get a paper read on this confirmation in 1953, at Rome, Italy, at the Sixth International Congress of Microbiology. So at last the facts are recorded in the archives of orthodox scientific literature.

In the usual viral infections, the host cell material and energy are used to build the viral colony with terrific multiplication of new viruses. In cancer, both nutrition and energy go into the building of new cancer cells and perhaps only an equal number of integrated viruses. For this reason it has been difficult to demonstrate the virus in certain cancer growths. Synthetic carcinogens numbering over two hundred have been tried out. One sees that the same two atomic units required for viral integration with the host cell are to be found. These are:

(a) the activated amine group, and

(b) the highly mobile hydrogen atom alpha to a double bond in the most exposed area, the "K region", as it is now named.

When dehydrogenated during anoxia it adds to the FCG activating unit. Carcinogens carrying the amine group that integrates with the host cell FCG to start the pathogenesis, as in acetyl amino-fluorene, and in Butter Yellow, and its analogues, hold the amine group in a protected state until the agent enters the body and hydrolysis or oxidation frees it of its protection, so it can make the azomethine condensation. Some experts think that the synthetic carcinogen prepares the cell for the virus carcinogen, but give no explanation of how this is done. Our postulate, on the other hand, shows that the amine group of synthetic carcinogens or of the fungus always found in cancer can play a part by inactivating the cell's FCG. The blocking of the oxidations that results brings about the colloidal gelling that causes the anoxia necessary for addition of the free radical (brought about in the virus by dehydrogenation) to the double bond that activates the FCG. Our thesis also shows how the carcinogen can produce cancer without the aid of a virus by addition of either pathogenic atomic unit. Some animal experiments with neoplastic transplants, and some with carcinogenic agents are reported here for comparison to show that the

same reagent gave protection and cured in high percentage in each set. The parathyroidectomy experiments should be recalled in connection with the pathogenesis and the anoxia involved. This is a main pillar of our thesis as based upon our earliest findings which, of course, took considerable thought to be appreciated. They are not even appreciated today in the orthodox circles, although Warburg, the Nestor of the biochemical profession, has championed the fact that anoxia is the cause of cancer for decades. He is the pioneer who developed methods of study of the oxidations in tissues. However, he has not yet appreciated the place of the free radical in the process. It will be seen that it is this position of orthodoxy that has limited progress in the explanation of the mechanism of anoxia in causing cancer, and the true nature of the carcinogenic change. Nevertheless, his contributions that won for him the Nobel Prize in Medicine on this subject are a monument of support to our postulate. Hence we give some quotations from his most recent summary in "Naturwissenschaften," Vol. 42 — p. 401, 1955:

If one examines them in the light of the data we have presented in the preceding pages, it will be evident that they confirm our own thesis on the pathogenesis of cancer and disease in general. They point out the essential status of anoxia, which we have claimed is necessary for the pathogen to be changed by dehydrogenation (of the tissue metabolism) to a free radical which instead of being burned and disposed of as fuel in the presence of oxygen, is not burned in its absence but is able to add to the cell Grana and do so at the very point where the activation of the oxidations is generated, namely the double bonds that activate the carbonyl group which we credit with initiating the tissue oxidations by serving as a dehydrogenator of fuels and pathogens. Thus anoxia is essential to the pathogenesis as it disposes the pathogen, carcinogen, virus or what not, to be able to integrate with the host cell and block grana function. The following quotations support our thesis as far as they go.

"One method for the destruction of the respiration of the body cells is removal of oxygen. If, for example. embryonal tissue is exposed to an oxygen deficiency for some hours and then is placed in oxygen again, 50 percent or more of the respiration is usually destroyed. The cause of this destruction of respiration is lack of energy. As a matter of fact, the cells need their respiration energy to preserve their structure, and if respiration is inhibited, both structure and respiration disappear." If one estimates the amount of normal function, one sees how the pathogenesis we have described will accomplish what Warburg reports here. Again, "If an injury to respiration is to produce cancer, this injury must, as already mentioned, be irreversible. We understand by this not only that the inhibition of respiration remains after the removal of the respiratory poison but, even more, that the inhibition of respiration also continues through all the following cell divisions, for measurements of metabolism in transplanted tumors have shown that cancer cells can not regain normal respiration, even in the course of many decades, once they have lost it."

"But why are the body cells dedifferentiated when their respiration energy is replaced by fermentation energy? At first, one would think that it is

immaterial to the cells whether they obtain their energy from respiration or from fermentation, since the energy of both reactions is transformed into the energy of adenosine triphosphate, and yet adenosine triphosphate = adenosine triphosphate. This equation is certainly correct chemically and energetically, but it is incorrect morphologically, because, although respiration takes place for the most part in the structure of the grana, the fermentation enzymes are found for a greater part in the fluid of the protoplasm. The adenosine triphosphate synthesized by respiration therefore involves more structure than the adenosine triphosphate synthesized by fermentation." Since the enzymes and intermediaries of fermentation, which biochemists accept as playing a big part also in the cell respiration, bathe the grana and the grana do not use them, as the quotation shows, then the conventionally accepted process is not correct, and a different process and different set of enzymes and intermediaries are involved. This process must be one which inactivates the grana when oxygen is missing, hence the logical deduction is that the free radical is an essential part of an early intermediary as we have hypothesized for so long.

Further, "The first notable experimental induction of cancer by oxygen deficiency was described by Goldblatt and Cameron, who exposed heart fibroblasts in tissue culture to intermittent oxygen deficiency for long periods and finally obtained transplantable cancer cells, whereas in control cultures that were maintained without oxygen deficiency, no cancer cells resulted. Clinical experiences along these lines are innumerable." Warburg emphasizes, ". . . but there is only one common cause into which all other causes of cancer merge, the irreversible injuring of respiration." He states, "In recent years it has been recognized that subnarcotic doses of urethane cause lung cancer in mice in 100 percent of treatments. Urethane is particularly suitable as a carcinogen, because, in contrast to alcohol, it is not itself burned up on the respiring surfaces and, unlike ether and chloroform, it does not cytolyze the cells. Any narcotic that has these properties may cause cancer upon chronic administration in small doses." So Warburg recognizes that a carcinogen must be *not* destructible by the cell's oxidative mechanism or otherwise. It can then become integrated with the cell and become a part of it, and can become accumulative as the disposing anoxia provides occasion. Warburg states in this connection, "Any respiratory injury due to lack of energy, however, whether it is produced by oxygen deficiency or by respiratory poisons, must be cumulative since it is irreversible."

The essential nature of the process of fermentation subjects it only to control by the circumstances that control any enzyme action. These are temperature, the pH reaction of the medium, the concentration of the ferment and of the substrate. Besides such accessories as the magnesium ion or when needed, a co-enzyme will determine the speed and extent of the reaction. Fermentation progresses as well in a test tube as in a living cell. No physiological control of these qualities for the specific service of the cell economy are known, except one, and that is the presence of the oxidation process. Normally this is the presence or absence of oxygen. Pasteur was first to observe this relationship, and the great Warburg named it after this supreme observer, — the Pasteur

Effect. This phenomenon was first described by him when observed in yeast cultures, but it is a common property to all cells that are obligatively aerobic. He reported that if a culture of yeast is deprived of oxygen, fermentation comes to the aid of the cell to supply the energy for vital processes, and if oxygen is again admitted to the culture, the fermentation ceases and oxidation takes its place. Fermentation is very wasteful and less than one-fifteenth as efficient as oxidation in the use of fuel material. The mechanism of the Pasteur Effect has never been explained by orthodox biochemistry. However, one will see that our postulate incorporates its explanation as a function of the Functional Carbonyl Group.

Oxidation has several positions of control in its process in line with our postulate. The first is the potency of the FCG which must start the process by dehydrogenating the fuel. When this carbonyl group is not free, as when the hydrogen it removes from the fuel is not taken away by some electron acceptor system, then oxidation is blocked. And for this, oxygen is essential as the ultimate electron acceptor in aerobic organisms. So lack of oxygen has two steps in blocking oxidation or hindering it. Another position of control is the inactivation of the FCG by additions to the double bonds that activate it. This would happen as we explained when anoxia prevents the free radical formed by FCG action from becoming a peroxide free radical so it must add to some position as that of the activating double bond. When this happens the FCG is inactivated and the starting of another oxidation progression is blocked. Fuels so added may easily be burned away by the process we outlined for removal of pathogens, but normally it is not accomplished by FCG action when oxygen is again admitted unless the FCG belongs to a different unhindered structure. If a pathogen has been added the SSR is needed to free it.* However, when oxygen is lacking the FCG as a rule will not be relieved of its hydrogen atom and can not form a free radical in the fuel until oxygen is admitted, and this is a protection to the mechanism.

Physiologically, however, the control that is designed to serve the economy of the tissue itself and the organism as a whole is regulated by the need for energy production. Oxidation is regulated so quantatively but fermentation is not. The need for energy is determined by the work to be done by a functional unit, and this is regulated by nerve or some hormone action which releases the phosphate stored energy to become work energy. This creates a deficit of stored phosphate energy, and oxidation must get going to replenish the supply. We explained elsewhere how the FCG accomplishes this act. So physiologically oxidation is controlled by the need of energy for work; and the substances concerned in the process in addition to oxygen and the fuel are the FCG, creatine and phosphoric acid. Therefore free creatine and free phosphoric acid and possibly some calcium balancing factor will give the free FCG a chance to start oxidation progressions that will yield the energy to form energy carrying creatine-phosphoric acid, as we explained before. Dehydrogenation and FCG action are concerned in oxidation only, and not in fermentation. Therefore,

*The SSR is a synthetically produced carbonyl compound of high Oxidation-Reduction Potential, used as a therapeutic agent.

fermentation is probably blocked by inactivating its initiating enzyme by the act of dehydrogenation to form an active free radical in it, so that it makes an inactivating addition that is split by phosphoric acid set free when it is liberated from an energy carrying ester. The ferment would then act through its restored hydroxyl group when energy fails, — a possibility only, as the need for energy production would be indicated by an accumulation of phosphoric acid, the first indicator for the need of fermentation, that is also able to liberate the ferment's bound hydroxyl group, that was inactivated by oxidation (dehydrogenation) during anoxia. Aside from such automatic control, there is the nerve and hormone control of the FCG and its azomethine double bond with the amine group of creatine where the nerve impulse determines its rupture to form the phosphate energy carrier, or its formation to discharge the energy load into the working mechanism. The phosphate energy bonds of fermentation are not formed or split in that way. Their energy must enter the functional mechanism through a different door. Mass action and energy transfered by photosensitization may determine such activity. There is no data on which to base a decision.

Fermentation need not be a general affair, but may be localized in some particular tissue where the FCG function is hindered. Function forced by fermentation energy beyond physiological control is the characteristic of all allergies including cancer. The type of allergy is determined by the functional unit involved, secreting fibrillae in hay fever, contractible fibrillae in asthma, conducting-synapse fibrillae in compulsion neuroses, mitotic fibrillae in cancer, etc. However, FCG block is necessary as described before. And the permanent block is done by a condensation with a firmly bonding amine as guanidin, or of some virus or an amine produced by decarboxylase action on some amino acid. When Victor C. Vaughan demonstrated in 1910 that the alkaline hydrolysis of various proteins gave rise to a toxic fraction that caused anaphylaxis, the writer was privileged to work in this kindly scientist's laboratory and isolated several toxic amines from his alkaline hydrolysate. They produced the allergic changes of fatal anaphylaxis. Later on it dawned on the writer that if his postulated FCG were blocked by toxic amines produced in a tissue by their decarboxylases that operate best in an alkaline medium, fermentation would be the result that could force the allergic responses that occur in anaphylaxis, and the type of response would depend upon the functional unit acted upon, while the amount of amine could be very small.

To check up on this a large number of allergy cases were treated with the SSR by the writer and several hundred collaborators. The results reported were about 85% recoveries obtained on one or two doses. These included the intractable asthmas and hay fever cases as well as the infantile eczemas that fail to respond to known methods. Some guinea pigs sensitized to egg white by the Vaughan method were also treated with success, but such experiments are not determinative unless all variables can be excluded, and they can not. At any rate, one sees here the practical meaning of the Pasteur Effect as modified by a pathogen. Physiologically, fermentation stops in an anoxic tissue when oxygen is admitted, but here, pathologically, the use of oxygen was blocked until the hindrance to the FCG was removed by use of a "super" carbonyl group, the

SSR. Though in orthodox biochemistry the explanation of the Pasteur Effect has not yet been made, we see that the carbonyl group is the key to the situation both normally and in pathological processes. Our postulate is thus strengthened by its broad utility.

That the narcotic action promotes neoplastic behavior is only too well known to the clinician. Especially is this true of the oxides of nitrogen that present permanent free radicals that can block respiration and promote cancer growth most disasterously. Their essential action is to hinder cell respirations. Other confirmatory quotations in support of our postulate could be given, but this is enough.

While it was important to recognize the essential role of oxygen lack in carcinogenesis, the observation is of no practical use until one understands the mechanism whereby the anoxia disposes to neoplasia. It is too bad that Warburg was not aware of our findings after parathyroidectomy, the action of high-energy amino groups in attacking the oxidation initiating carbonyl group of the grana as we postulated, and that thereby the oxidation mechanism was blocked. It is too bad he gave no thought to the position of the free radical formed by these dehydrogenations and the absolute need of molecular oxygen to carry the oxidation progression forward, and that when the substrate acted upon could find no oxygen to combine, it must combine an appropriate double bond, and thus integrate with the cell's energy producing mechanism. It is too bad that he did not make these steps and then the third step to recognize that the very integration of the pathogen with the host cell invited the oxidative separation of both leaving the host cell in good functional status, while the pathogen was destroyed. He therefore missed the essence of the reversibility of carcinogenesis, and the means of bringing it about. We will give the details as we go along and the proofs for each step. However, what Warburg did establish was a great advance in cancerology and his prestige as a biochemist makes this support to our thesis, a most valuable one.

CHAPTER IV

PROOFS OF REVERSIBILITY

Official Test

The first official cures of cancer of the vital organs in the far advanced stages were had in 1919 under the auspices of the Wayne County Medical Society branch of the American Medical Association. Five cases were selected by an officially appointed committee of surgeons and one pathologist numbering five in all. The cases were officially selected as fit for the test, and the test was officially closed three weeks after the patients were treated and improvements began to show. Five years later it was seen that three of the cases were cured and possibly a fourth who lived too far away to be examined, but through the results in his case, new patients were sent to the writer, five years later. One of the test patients with cancer of the uterus, proven at laporotomy as extending throughout the abdomen and perforating the stomach so as to cause severe bleeding, lived fifteen years in good health after the treatment, died from an accident, and the coroner's autopsy showed no cancer was present, and the cause of death had been a brain injury. Yet the Official A.M.A.-W.C.M.S. committee reported no results, and the W.C.M.S. Cancer Committee that was appealed to five years later to change the false report, refused to do so and summarily denied all the diagnoses made by the official committee. The cures, however, could not be denied. They refused any further tests. No other official test was ever made. Every request for one has been refused. But the false report is still being circulated.

NATIONAL STATISTICS

Cancer mortality statistics during the decade, 1920 through 1929 inclusive, for the six largest cities of the United States, reported by Hoffman, in the "Spectator", and elsewhere while we specialized in cancer treatment at Detroit, are given below. Since the only variable that entered the picture was our own therapy, we take the credit for the drop in the death rate in Detroit and its lesser increase in cities that sent us patients, as compared to the high increase in death rate in all largest cities that did not send us patients. While Philadelphia and Los Angeles showed a 30% increase in this decade, Detroit

showed a drop of over 20%, and it was the only large city that showed any fall whatever in the death rate from cancer.

National Statistics

SIX LARGEST CITIES IN 1930	CANCER DEATH RATES (per 100,000 pop.)		
	1920	1923	1929
New York City	107.9	106.0	115.4
Chicago	104.9	103.7	107.7
Philadelphia	103.9	116.3	135.8
Detroit	96.7	67.4	74.5
Los Angeles	96.8	130.4	128.1
Cleveland	93.8	89.9	104.2

Individual case studies that support this thesis are taken from the testimony of collaborating physicians in the U.S. Federal Court where they were proven factually incontestable. They demonstrate the different features of the *reversibility*.

UTILITY IN GENERAL PRACTICE

What is most important to the physician is the field of utility of synthetic Survival Factor therapy. The following table is contributed by Dr. Wendell Hendricks as part of a paper given before a convention of collaborators who were making observations with this therapy. The best showing is in the viral diseases which gave a 100% recovery rate, measles, mumps, infantile paralysis, and the acute infections as gonorrhea, rheumatic fever, sinusitis, etc., and influenza. The poorest showing was in Arthritis Deformans which gave only a 50% cure rate. This disease is 100% incurable otherwise, however, and perhaps the patients did not stay with the treatment long enough to give it a chance. In the 100% incurable hypertrophic type of arthritis a cure rate of 82% of 144 cases is certainly a good service with observations showing the permanency of the cures over a period of 4 years of check-up.

NAME OF SICKNESS	Number of cases	Age of patients	Average weeks between reactions	Time required for cure	% of cures	Years since cured
Allergies nasal	282	1-70 yrs.	9	18 w.	82	6
Acute Tonsillitis	61	2m-54 yrs.	0	3 d.	100	4
Chronic Tonsillitis	20	2-84 yrs.	3	6 w.	80	3
Vincent's Angina	35	2-57 yrs.	0	6 d.	89	4
Arthritis Deformans	16	22-57 yrs.	9	36 w.	50	5
Arthritis Hypertrophic	144	24-82 yrs.	9	27 w.	82	4
Gonorrhea	15	22-55 yrs.	0	3 w.	100	3
Bronchitis acute	64	2m-56 yrs.	0	2 d.	94	4
Bronchitis asthmatic	460	1-69 yrs.	9	27 w.	80	6
Bronchitis chronic	35	3-67 yrs.	3	18 w.	80	4
Brucellosis	35	29-58 yrs.	9	18 w.	93	3
Coccidioidomycosis	70	7-63 yrs.	0	3 w.	95	3
Cholecystitis	44	26-58 yrs.	3	18 w.	84	2
Coryza acute	100	6m-74 yrs.	0	2 d.	100	5
Eczema	120	1-68 yrs.	9	27 w.	80	6
Rheumatic Fever	20	4-11 yrs.	3	6 w.	100	3
Gout	10	30-55 yrs.	0	12 w.	90	6
Influenza	51	8-65 yrs.	0	8 d.	100	3
Nephritis acute	22	6-66 yrs.	3	6 w.	90	3
Nephritis Chronic	20	22-68 yrs.	6	18 w.	85	3
Neuritis	67	17-84 yrs.	0	3 w.	85	4
Pneumonia	22	1-69 yrs.	0	6 d.	82	3
Poliomyelitis acute	10	3-16 yrs.	0	3 d.	100	4
Poliomyelitis chronic	2	23-33 yrs.	9	18 m.	100	3
Syphillis chronic	10	24-47 yrs.	9	36 w.	80	4
Sinusitis acute	38	12-63 yrs.	0	3 d.	92	3
Sinusitis Chronic	27	2-66 yrs.	3	18 w.	78	3
Urticaria	75	1-72 yrs.	9	18 w.	88	4
Combined total of measles, whooping cough, mumps and scarlet fever	66	6m-36 yrs.	0	3 d.	100	3

CHAPTER V

ANIMAL EXPERIMENTS

When presenting a thesis such as this, it is good policy to eliminate superstitions and misinterpretations. For example, the idea that the cures we will report were secured by psychology or suggestion, or that after all the disease was not Cancer, Infantile Paralysis, Tuberculosis, or what it is represented as being. That the diagnoses were uncontradictable is established, and the pretreatment control period showing the downward course of the patient was also proven uncontradictable. The recoveries were established for many years, even several decades in some cases. It was also proven that no other remedy was used, and the sharp contrast between the pretreatment control period and the characteristic cyclic recovery that characterises the recovery process after the Survival factor reagent was used settles the credit according to modern scientific procedures in drug testing. Because of the many shortcomings in the collateral control system when humans are under treatment, it has been discarded long ago, and the ACTH and Cortisone experiments show the clinical procedure of using an adequate pretreatment observation period to compare with the post-treatment period is the only reliable procedure known; and it is the procedure used by all highest rating clinicians for clinical tests. This is the system we use throughout, and each case should be studied with this in mind.

However, to remove any doubts about etiological and psychic factors we offer a few animal experiments, in which transplantable C 57 Breast Cancer was inoculated into mice, and also transplantable Sarcoma 37 was inoculated into mice, all at the Jackson Memorial Laboratory at Bar Harbor, Maine. The inoculated animals were then sent to Dr. Stanley Bandeen, of Louisville, Kentucky for treatment and observation. The C 57 inoculations were made on May 7th and the Sarcoma 37 on June 30, 1950. The experiment was terminated by a frost on November 13, 1950, that killed most of the cured animals and those undergoing recovery. The details are given below. Since the etiological factors in these experiments are uncontradictable, the diagnoses of the conditions treated are also uncontradictable. Some mice were killed by fighting. They are excluded from the statistics, even though they appeared cured. The treatment used was the 10^{-12} concentration of the serial system of carbonyl groups with free radical terminals, which we designate as the Synthetic Survival Reagent (SSR). We sometimes call it the Survival Factor reagent or remedy. The chemical formula is stated a little farther along. The dose is that equal to the smaller dosage of Vitamin B_{12} proven clinically active.

EXPERIMENT I

Twenty-five mice C 57 breast tumor transplantation, May 7, 1950. Five were held as controls, and the rest were divided into two sections: (a) 8 mice

each receiving 4 minims of the reagent by injection, and (b) 12 mice each receiving 6 minims of the reagent. Treatment was given three days after tumor transplantation.

RESULTS

Controls: Five mice. All of the controls died of cancer from the 12th to the 24th day following tumor inoculation. Average length of life: 17 days after inoculation.

Section (a): Eight mice, 4 minims each, the third day after inoculation. All tumors had ruptured through on the 11th day and started to heal on the 12th day. They were completely healed on the 15th day. One of the mice had recurrence which proved fatal on the 44th day. On the 64th day one mouse gave birth to 3 young which lived until killed by frost on the 126th day after birth. Three mice died on the 64th and 66th days tumor free. One was killed fighting on the 32nd day. Three lived cured until killed by frost on the 190th day.

Death from cancer: 1
Death from fighting: 1
Recoveries: 6
Average length of life of those which recovered: 127 days.

Section (b): Twelve mice, 6 minims each, third day after inoculation. All tumors ruptured through from the 9th to the 10th day. All tumors were healed between the 13th and 14th days. Three mice died fighting, one on the 4th day, one on the 32nd day, and one on the 36th day. One died of cancer on the 38th day via recurrence. (On the 62nd day one mouse gave birth to 4 young. On the 112th day she gave birth to 3 young, her 2nd set). The rest, 8 cured mice lived to the 190th day when killed by frost, cancer free.

Death from cancer: 1
Death from fighting: 3
Recoveries: 8
Average length of life of those which recovered: 190 days.

EXPERIMENT II

Twenty-five mice were inoculated with C 57 Breast Cancer by transplantation on May 26, 1950. Five were used as controls. Four were treated with 2 minims, 8 were treated with 4 minims and 8 were treated with 6 minims of reagent.

RESULTS

Controls: One died fighting the third day. The other four died from cancer between the 12th and 18th days. Average length of life: 15½ days.

Section (a): Four mice treated with 2 minims of reagent. One died of cancer on the 24th day. Another died of cancer on the 26th day. On the 30th day and the 32nd day the other two tumors healed. One died cancer free on the 104th day and the other died cancer free on the 128th day.

Death from cancer: 2
Death from fighting: 0
Recoveries: 2
Average length of life of those which recovered: 116 days.

Section (b): Eight mice were treated with 4 minims of reagent. Three tumors healed on the 13th day, the others on the 11th, 12th, and 16th days. Three mice with healed tumors were killed fighting. One died on the 44th

day, one on the 135th day, one on the 139th day, and two were killed by frost on the 177th day, cancer free.

Death from cancer: 0

Death from fighting: 3

Recoveries: 5

Average length of life of those which recovered: 135 2/5 days.

Section (c): Eight mice treated with 6 minims of reagent. On the 12th day 4 tumors were healed, and on the 15th day the other 4 tumors were healed. Two mice were killed fighting on the 16th day. One was killed on the 24th day, tumor recurrent. One died with cancer on the 34th day, tumor recurrent. One died on the 128th day and three were killed by frost on the 177th day, all four being cancer free.

Death from cancer: 2 (including the one killed fighting on the 24th day).

Death from fighting: 3

Recoveries: 4

Average length of life of those which recovered: 164¾ days.

EXPERIMENT III

Sixteen mice received by transplantation Sarcoma 37 on June 30, 1950. Four were used as controls, and the rest were divided into three sections: (a) 4 mice received 4 minims each of the reagent, (b) 4 mice received 6 minims, and (c) 4 mice received 8 minims.

RESULTS

Controls: Four mice. All of the controls died of cancer between the 12th and the 20th day. Average length of life: 16½ days.

Section (a): Four mice, 4 minims each. Two died fighting on the 10th day and the 14th day before tumors were healed. The tumors healed on the other two on the 16th and 17th days; one died on the 110th day and the other on the 125th day, both cancer-free.

Death from cancer: 0

Death from fighting: 2

Recoveries: 2

Average length of life of those which recovered: 117½ days.

Section (b): Four mice 6 minims each. One died from cancer on the 18th day. Three tumors healed on the 35th day. They lived cured until killed by frost on the 136th day.

Death from cancer: 1

Death from fighting: 0

Recoveries: 3

Average length of life of those which recovered: 136 days.

Section (c): Four mice, 8 minims each. All 4 tumors healed on the 30th day. On the 84th day one mouse died fighting, this animal being cured. The other 3 remained cured until killed by frost on the 136th day.

Death from cancer: 0

Death from fighting: 1

Recoveries: 3 (4 if one includes the mouse killed fighting on the 84th day).

Average length of life of those which recovered: 136 days.

EXPERIMENT IV

Twenty-four mice were inoculated with Sarcoma 37 on July 28, 1950 and were treated with the reagent 5 days later. Four mice were held for controls, and the rest were divided into three sections: (a) 8 mice receiving 4 minims each, (b) 8 mice receiving 6 minims each, and (c) 4 mice receiving 8 minims each.

RESULTS

Controls: Four mice. Two mice died from cancer on the 31st day, and two died from cancer on the 36th day. Average length of life: 33½ days.

Section (a): Eight mice, 4 minims each. Two died fighting on the 11th day and on the 28th day after inoculation. Two died on the 84th day, cancer free. Two were killed by frost on the 108th day, cancer free.

Death from cancer: 0

Death from fighting: 4

Recoveries: 4

Average length of life of those which recovered: 96 days.

Section (b): Eight mice, 6 minims each. All tumors healed from the 18th day to the 23rd day. Two mice died cancer-free, one on the 83rd day, the other on the 101st day. Five of the others lived until the 108th day and were killed from frost. One survived the frost and lived to the 411th day.

Death from cancer: 0

Death from fighting: 0

Recoveries: 8

Average length of life of those which recovered: 142 days.

Section (c): Four mice, 8 minims each. Two tumors were healed on the 10th day, and two were healed on the 12th day. All continued in good health, cured, until the 108th day when 3 were killed by frost. One survived the frost and lived to the 412th day.

Death from cancer: 0

Death from fighting: 0

Recoveries: 4

Average length of life of those which recovered: 184 days.

It should be noted that the two mice that survived the frost, lived for an average of 411½ days and died free of cancer. This is equivalent to 41 years after cure on the human scale. One of the mice received 6 minims of the reagent and the other received 8 minims.

Discussion

The average length of life of the untreated controls was 20½ days, that of the treated animals that survived the frost was 411½ days, and those that lived up to the frost and were killed by it was 190, 177, 136 and 108 days for the different groups. We see that the frost reduced the possible life of the recoveries on an average from 411½ days to 153 days. When considering the average of Groups II, III, and IV, it must be remembered that the animals killed by freezing were all killed by the same freeze that killed those in Group I, and that those mice in the last three groups were treated 21, 56, and 84 days after those in Group I were treated. Therefore, the average length of life, while it appears to be shortened in the last 3 groups, actually was not shortened.

However, the frost experience makes this experiment valuable in that it showed the effect of dosage, for the only frost survivors were those that received 6 and 8 minums, and those that lived to the frost were those that received the heavier dosage for the largest part.

Two minims showed very poor results as compared with the 6 and 8 minim dosages, but even the 2 minims gave cures, while the controls all died of cancer within three weeks. Hence a minim of a solution of one part to a trillion of water is a great deal of material when one considers the effects. It is just a few millions of molecules, that is all, and only one molecule should be able to start a chain reaction under ideal conditions.

As a comparison of cure rate, with the controls showing 100% deaths from cancer and 0 cures, in spite of the frost, the experiment is decisive, if any such experiments mean anything and it shows the effect of higher oxidation catalysis from the heavier dosage in fighting the cold.

In other experiments we took accounting on the 100th day or the 200th day instead of letting the frost set the limit as in this experiment. The end result runs about the same as the others.

A matter of interest here is the recurrences of the tumor after it healed pre-eminently in 3 cases that received more than 4 minims each. There were two such that received 6 minims in one group, and one in another. The explanation can be found in the text. In these animal experiments the word "cure" is used to indicate the complete absorption of all tumors, visibly and palpably, the healing of the lesion, and the return of health to the animal.

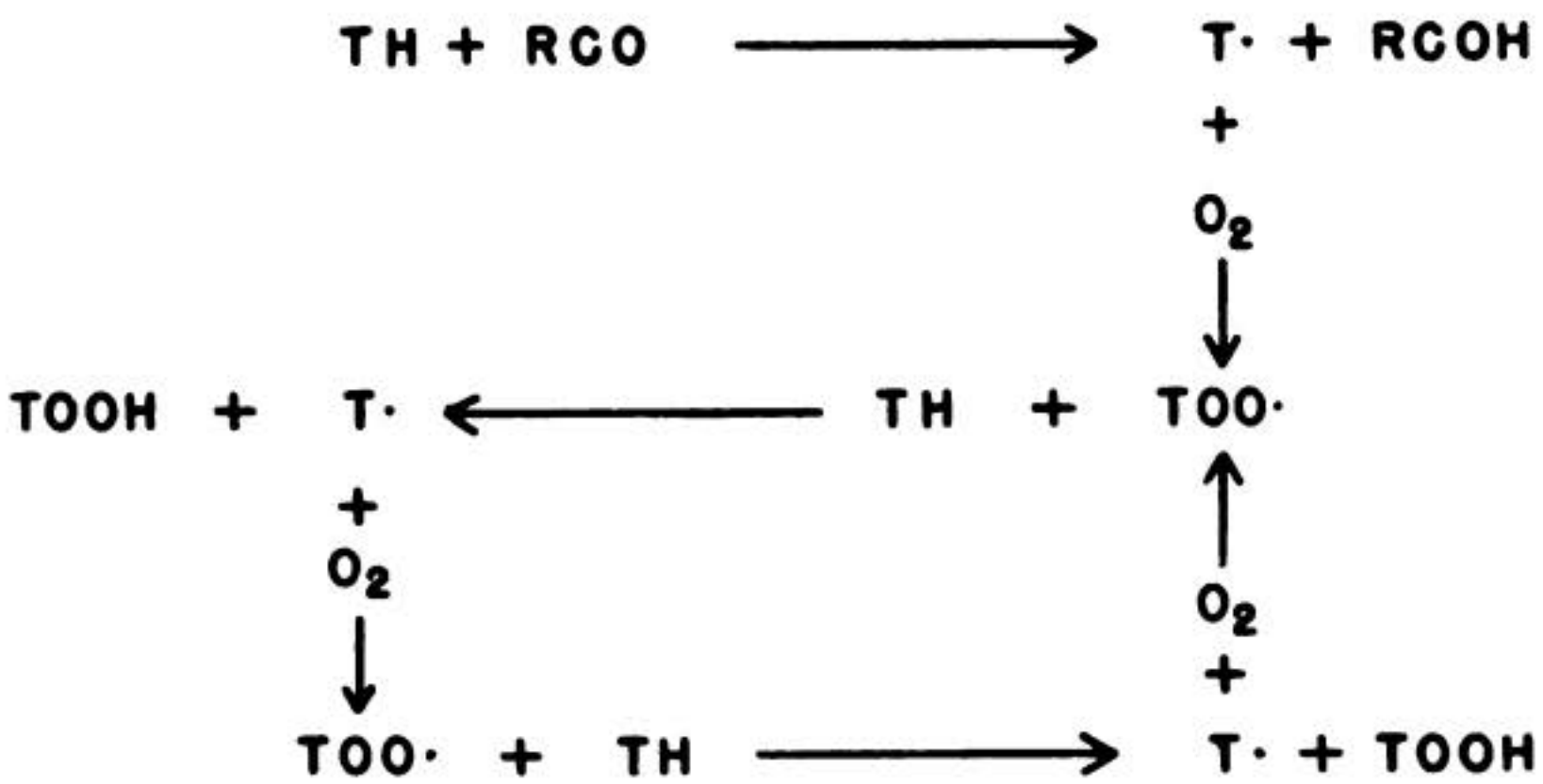
CHAPTER VI

ENERGY PRODUCTION

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

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

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



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

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

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

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

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

In case the normal range of energy production can not cleave the bond, FCG function is destroyed and disease results. Then a superiorly efficient dehydrogenator carbonyl group must be supplied to burn off the pathogen as

previously stated. This restores the FCG for normal function. This super dehydrogenator is our therapeutic Synthetic Survival factor. (SSR).

The adequate oxygen supply plays two parts:

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

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

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

Since creatine did not interfere with FCG function as did guanidin, and since it is the only amine possibly that forms high energy phosphate bonds, it was easy to assume that it played a role in the transfer of energy produced from the oxidation of fuel to high energy phosphate bonds as of ATP. It would accommodate this transfer by condensing with the FCG to form an azomethine bond until enough energy had been generated to admit phosphoric acid into the bond and unite it with the amine group of creatine sending the creatine phosphate off as a high energy carrier. The FCG is thus free to start further dehydrogenations physiologically. However, if the amine condensed with the FCG forms a tight bond not separable under normal ranges of energy production as did guanidin in the parathyroid experiments, the whole train of pathological events must follow. For this reason it is well to inquire into the sources of such pathogenic amines. One is the production of toxic amines in the acid colon by various bacteria that decarboxylate amino-acids. In many people, the intestinal flora are firmly entrenched and convert the food into one's poisons, that serve as the vanguard of disease. Animal proteins are the main sources of these toxic amines, and sulphides, while vegetables, cereals, and fruits supply plenty of protein and at the same time do not support decarboxylating germs. The intestine must be kept at a range of pH above 7, since the

decarboxylations progress best at a pH of 3.5 to 6 when mediated by the streptococcus fecalis and so many others.

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

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

The atomic set-up of the reagent itself that carries the Super-high-efficiency carbonyl group must offer a steric advantage in each disease where it is applied. For example: in Hog Cholera, diphenoquinone proved 100% efficient in several epidemics, while it proved 100% worthless in Rabies, and the serial system of carbonyl groups used in Hog Cholera proved 100% worthless, while it was 86% efficient in Rabies in terminal cases. Both diseases kill 100%, within 3 to 5 days. Rabies is neurotropic always and Hog Cholera rarely before the terminal hours. Our search has been for a molecule carrier of the Survival Efficient Carbonyl group that is equally applicable in all diseases where drug interference has not modified the steric set-up. This will be discussed later on. It will be seen, however, that to identify the high efficiency oxidation system, we consider normal to the cell, as of the same order as the therapeutic substitute used to rescue the FCG and restore normal function and structure, a few comparisons will have to be made. For example: that they are of the same order is seen in being blocked by the same agencies as anoxia, sulphydryl, and that their processes are blocked by permanent free radicals, highly polar double bonds, etc. Likewise, the restitution program, that follows the freeing of the FCG system of its pathogen by the Synthetic reagent, is the normal process. So the Synthetic reagent fits into the mechanism with equal

grace as did the FCG before it was attacked by the pathogen. Likewise, since recoveries from viral and neoplastic diseases that could never be combatted successfully before are accomplished by the natural resources of the body after the Synthetic Survival Reagent is used, they both fit the cell chemistry, but each in its respective capacity for survival. Since the recovery mechanism includes cyclic reactions at definite periods never seen in medicine before, just as the cure of the pathologies involved were never seen before, a deeper grasp on tissue physiology is made possible and a wider range of expertness can be acquired. Any successful clinician will recognize that expertness in this therapy depends upon study and experience, and some new viewpoints must be adopted. To illustrate let us review a few toxic cases.

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

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

A case of toxic nodular goitre illustrates some of the main features of the oxidation mechanism of our postulate.

CHAPTER VII

CLINICAL PROOFS OF HIGH EFFICIENCY AND SSR OXIDATIONS

TOXIC NODULAR GOITRE

CASE No. 1 — Dr. Baldor

Mrs. M. J. was 35 years of age in July, 1943, when she came to Dr. Julian Baldor for treatment of a rapidly developing weakness, tremor, sweating, a big change in her appearance, loss of weight, twitching of her muscles, continual excitement, excessive nervousness, considerable loss of weight, pains in her legs, terrific heart palpitation, and shortness of breath. She noticed her eyes were "popping out", and that her neck had become enlarged by a number of hard nodular tumors. Although the situation had only started a few months previously it had advanced rapidly until she was almost helpless. Examination by Doctor Baldor showed a very rapid pulse, a blood pressure of 190/110 and the other symptoms mentioned, and the Basal Metabolism Rate was found to be plus 104, instead of plus 4 or 6 which would be normal. He noted the nodular development of the goitre which meant that iodine therapy would not help as it does in other cases without this pathognomonic change. The exophthalmia was excessive, and meant an advanced stage of toxicity. Operation could not be done under the circumstances, without first reducing the symptoms to the limit and, in the only way this is attempted, he gave her the iodine therapy with ice bags to the neck and quiet, on July 8, 1943. This treatment was continued until October 30th when it was discontinued as a failure. Indeed her whole condition had become so much worse, she was about wild with excitement and nervousness, she experienced things that made her think she was losing her mind, the muscle tremors increased and jerked uncontrollably besides. She had become so weak she had to be carried and had lost control of her hands and feet. The exophthalmus and the tumors of the neck had increased exceedingly, the heart showed signs of failing in the weakness of the ever rapid pulse, and the drop in the systolic pressure from 190 to 170, while the diastolic stayed at 110 showing that the toxic cause of the high blood pressure was still as bad as ever. Operation was out of the question, so Dr. Baldor decided to use the reagent discussed in this book. The iodine therapy was stopped two weeks, so she would be ready for the SSR treatment. In these two weeks, she became worse at the same speed as previously. Thus the pretreatment control period showed a steady advance of the disease.

She was so weak by Nov. 10th that she had to be carried into the car and into Dr. Baldor's office to be given the treatment, an injection of 2 micro micrograms of the Synthetic Survival Reagent (SSR). Every physician knows the value of the patient's own description of her symptoms and status, so we will let

LA BENEFICA ESPAÑOLA

LABORATORY REPORT

Patient Monetta B. J.

Address 916 E. Hamilton

Dr. Baldor

Date of Specimen 7/8/43.

Date of Report

Basal Metabolic Rate: Plus 104%

Pulse during test: 114

Body Temperature: 98.6 F.

Signed M. Faibe

LABORATORY OF CLINICAL PATHOLOGY

911 CITIZENS BUILDING

TAMPA, FLORIDA

HERBERT S. MILLS, M. D.
FELLOW OF THE AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS

SEROLOGY AND METABOLISM

HOURS: 8:30 A. M. TO 5:00 P. M.
PHONE 4003

PATIENT Mrs. Albert C. J DR. Baldor

BLOOD

Kolmer

Kolmer Dilute Serum

Kahn

COMPLEMENT FIXATION:

For Tuberculosis

For Gonorrhea

For Echinococcus

BASAL METABOLISM

BMR plus 6

Pulse 100

Temperature 98.6

SPINAL FLUID

Kolmer Rept 35

Colloidal Curve

Cell Count

Differential Count

Smear for Bacteria

Sugar

Chlorides

Protein

Culture

Pulse was taken after violent coughing spell.

March 16, 1944 (Date)

H. S. Mills M. D. (Pathologist)

Author's Note: M. B. J. and Mrs. A. C. J. are one and the same person.

a few words from her personal report emphasize some of the points we wish to establish. She stated:

> "First my trouble started in my finger tips with throbbing. It seemed as if the blood circulation was half stopped. My hands began to swell and I could not wear my rings any more. I had terrific pains and then I began having trouble with my legs. I began to have contraction of the muscles, my toes would draw up into knots. I went to Dr. Baldor about it. He gave me one thing after another, but I did not improve any. He sent me to the clinic for the metabolism test, and after that he started a different treatment. It was some drops. All the time I was taking on like crazy. I could not sleep at night. My husband had to lift me up in bed. My hands and legs got steadily worse. Finally I got so bad, my husband had to pick me up and put me in the car. I could not get in. My legs would just turn to water. Dr. Baldor gave me the Koch treatment. About two or three weeks later, I felt like a new woman. My strength came back, my legs and hands cleared up, and I can use them again. I now have a job demonstrating. I carry a suit case weighing fifty pounds in and out of homes."

The Basal Metabolism Rate was taken three months after the treatment, and found to be perfectly normal, namely plus 6, and physical examination showed her normal in all other respects, no sweating, no jerking, no tremor, no muscle twitches. The exophthalmus had completely disappeared, and so had the thyroid tumors. The thyroid gland was normal on palpation, inspection and function. The pulse was normal 80 to 90, and so was the blood pressure, 140/80. She was strong, slept well, and without any trace of the former disease.

An analysis and interpretation of this case notes two toxic states — one that is the result of the forced secretion of the Thyroid cells, the *thyrotoxicosis* that nearly killed the patient. The other is the toxin that blocked the regulated energy production and the regulated energy acceptance and utilization in both the thyroid cells and the tissues in general. This is the *pathogen toxin* which the postulate identifies as an amine of higher O/R potential than the functional carbonyl (FCG) group of the tissue cells could dehydrogenate and thus burn out of the way. The pathogen toxin therefore had the upper hand and as it was being increased in amount, its effects were also increasing as the block to energy production and energy acceptance by the tissue functional carbonyl groups. These normally initiated oxidations that produce energy efficiently, and received energy in a regulated way to perform work.

As a result the Krebs Cycle energy production took over, and had already largely replaced the high efficiency oxidations of the FCG-s, when the patient came under observation of Dr. Baldor. If the toxic amine pathogen had been subject to dehydrogenation at the hands of the FCG like the usual run of pathogens and fuel substrates, it would have been burned out of the way, and could not condense with the FCG of either the high efficiency energy producing

system or the FCG of the energy accepting system of the cell and block their functions.

It was evident clinically that energy was not reaching the working mechanisms. This was seen in the steadily increasing weakness of the skeletal and heart muscles and nervous system. It was also clinically evident that energy production was going on at the highest rate shown by the BMR of 104%. It was also seen that the thyroid gland was forced to the limit in producing its secretion to push cell activity to evolve more energy. But no matter how much was produced even to the exhaustion of the patient, none was used by the energy starved cells. The patient lost weight rapidly and to the extreme to supply material for energy production, but it could not get into the working mechanism via the blocked carbonyl groups of the energy accepting system of the postulate. *The basic pathology then was the block to energy acceptance by vital working units.* Since one toxin, an amine of high activation was the pathogen, it is also evident that the FCG's of energy production and energy acceptance are similar atomic groups and since these are dehydrogenators, the postulate identifies them as highly activated carbonyl groups. This conclusion is supported by the type of response to oxidation the integrated toxin gives, after it is condensed with the tissue cell FCG's. That is, the type of cleavage observed is that of an azomethine double bond when its alpha positioned hydrogen atom actually invites dehydrogenation and is removed so a free radical can be formed and add molecular oxygen to become a peroxide free radical which accomplishes the oxidative separation with restoration of the functional carbonyl groups of the tissue cell, and the toxic amine group is burned away. The facts of the case history support this explanation. (See Appendix).

It is seen here that the pathologic state actually invites correction, and any clinician would suggest correctly how it could be done. He would say, since the FCG can not dehydrogenate the toxin and start its combustion, because its O/R potential is too low, then the thing to do is to offer a carbonyl group of higher activation with a potential equal to the job. This is what was done in this case. A molecule of correct steric advantage carrying a carbonyl group of high O/R potential was used. The results were the rapid reversal of the pathogenesis. As soon as the integrated toxin was burned out of the way, energy could enter the cell working units, and the urgent call for more energy stopped. The thyroid was not called upon to whip up the tissues to do more oxidizing, and the nodules it had developed to aid its work subsided and disappeared. The BMR dropped to a normal of plus 6%, and all of the symptoms of the thyrotoxicosis, and of the basic pathogen disappeared. The woman was normal in 3 months after one dose of a highly activated carbonyl compound.

This case proved a few things in the Koch postulate, and it also shows that the thyroid secretion takes no part in the oxidation process, any more than the poisonous nitrophenol series that some have classified as accelerators of the oxidations. As we pointed out here, thyroid function is to whip up the cells

to put their oxidation apparatus to work to supply the energy needed for the occasion. It itself does not enter the oxidation process. Nitrophenol blocks various esterifications with phosphoric acid which normally form high energy carrying phosphate bonds. Thus it starves the cells of energy and the tissues are whipped up to produce more energy for survival, just as in the case at hand. Nitrophenol thus works as an "uncoupler" and is so classified. It prevents the energy accepting mechanisms from receiving the energy. In the case at hand the energy came to the doors of the energy accepting mechanism, the FCG of energy acceptance, but the door was closed, — blocked by the condensation with the amine compound. Thus the carbonyl group of energy acceptance was already occupied and could not condense with the amine of the ATP that carried the energy that would be liberated by ATP-ase with the help of calcium. Our postulate goes on to explain that with the liberation of energy by the hydrolysis of the ATP to ADP, the phosphoric acid set free can split the azomethine bond setting the ADP free to again do another cycle of energy transport with the acid.

One sees that there is no similarity in the actions of the thyroid secretion, the nitrophenols and the highly activated dehydrogenator carbonyl compound (SSR) used to oxidize the pathogen out of the way. The SSR actually took the leading part in the oxidation mechanism and did the work the normal oxidation initiator would have done if it had an adequate O/R potential. It is not possible to compare a reagent that prevents energy storage for use in work, with an agent that produces energy for use in work, and besides, actually starts the oxidation process in the cell by burning away the pathogen that was blocking energy production.

Further, the nitrophenols are pathogens whose action can wear out if not forced too long. But if they are forced too long, they are subject to reduction to aminophenols, which would then act much like the pathogen in this case, and block the initiation of the oxidation progression, and bring about a dangerous situation much like in the case at hand. The nitrophenols proved to be pathogens in the attempt to beautify obese women. The reduction in weight took place but in too many the destructive action continued because of the situation that existed in the case we are discussing, and these victims went on to their deaths. They need their FCG's freed from the obstructive amine as was accomplished in this case. However, the experts are still at sea with regard to the true action that caused the fatalities.

The block in the use of the energy of oxidation by dinitrophenol is seen also in its inhibition of mitosis in Sea Urchin eggs reported by Clowos (1951 Ann. N. Y. Acad. of Science). Even though the dinitrophenol in doses of .01 mM concentration caused a fourfold increase in the consumption of oxygen, the mitosis and phosphorylation was cut in half, and further increase in the concentration of the poison completely blocked mitosis and phosphorylation. So whether the oxidation process is blocked in producing energy, in transfering and carrying energy in phosphate bonds, or in receiving this energy, the reactive response is to produce more energy to make up for the energy starvation in the tissues whose working mechanisms do not receive

the energy. Thus an analysis of effects of toxic amines and nitrophenols shows they do not give impetus to the oxidation mechanism, but block its ultimate purpose—the supply of energy to the vital mechanisms of the tissues. Here we find in 1951, a nice confirmation of our postulate measured with microscopic accuracy.

The thyroid secretion is a hormone whose intimate action is still unknown. However, it does not take any part in the oxidation process itself. Comparing its action with that of the SSR, one sees that the latter took the leading part in the oxidation mechanism. Further the action of nitrophenol and of thyroid extract are of different orders and challenge comparison. The former always has a toxic action, the latter is physiological, but the action of both, as explained before, is very different from that of the SSR. The high BMR in the case at hand has a pathologic cause depending on the pathogen that blocked energy acceptance by the cell's working mechanism.

The statement of some biochemists that the oxidation process has no immunological significance is based on the fact that the Krebs Cycle has none. We gave the reasons before. The O/R potentials of the participants are too low. Then these biochemists also hold that the Krebs Cycle is the only mechanism concerned in the tissue oxidations, and is all sufficient. They do not consider that the Krebs Cycle is a hang-over from the process used by primitive forms as bacteria and though it is retained by the higher forms as animals and man, it is only used by such as an alternative pathway when the High Efficiency System already explained is inactivated for a time. That it offers no protection is seen. Moreover, it gives no clues to the explanation of the Pasteur Effect. The early chapters of "The Survival Factor in Neoplastic and Viral Diseases" show how both depend on the action of the FCG. While the carbonyl group that initiates the oxidations of the High Efficiency Smokeless System, lacks the high O/R potential carbonyl dehydrogenator that some pathogens require for their destruction, yet its range of O/R potentials is twice as high (0.7 v) as that of the Krebs Cycle participants (0.3 v). So the opportunity to give protection by the High Efficiency System is considerable, — enough to maintain good health under the usual circumstances. The use of a Super-high carbonyl dehydrogenator of correct steric advantage is proven in this case to offer protection by way of an oxidation process that imitates that postulated for the High Efficiency System, and a close analysis of this case is all that is needed to prove the existence of the High Efficiency System. However, two more cases will be submitted to show that the toxic basis for malfunction can be removed, and the pathology corrected by the processes of adequate dehydrogenating efficiency, started by the Super-high dehydrogenator, and continued by the natural dehydrogenator (FCG) system.

POST-PNEUMONIA NEPHRITIS

CASE No. 2 Dr. Evans

Tom E., 4 years old, was recovering from bilateral bronchopneumonia, when he suddenly took a convulsion of considerable severity. Oedema rapidly

developed with blurred vision, headache, dizziness, delirium, etc. The urine secretion diminished as the oedema rapidly increased. The blood pressure was found to be 146/68, and the blood non-protein nitrogen 74.6 mgms.%. Twelve hours later the pressure rose to 160/100, and two days later it was 180/130 showing a rapid development of the pressor substance that blocked the kidney elimination. The oedema had developed by then to the point where the contours of the chin and neck were obliterated, and very little urine was passed. Then the second convulsion took place. It was severe and the boy passed into coma. It was in this condition that he received the Synthetic Survival Reagent. A few hours later, the mental symptoms had improved, he came out of the coma; soon the headache, blurred vision, delirium, etc., gave way to rational mental comfort, the blood pressure steadily dropped and the urine increased as the oedema disappeared. The blood pressure was found normal in a few days with a normal non-protein blood nitrogen of 25 mgms.%. The correction was completed by rescuing the FCG so it would go back to work again. The pressor substance is well known now to be a toxic amine, so our thesis is supported nicely by this case also.

ECLAMPSIA

CASE No. 3 Dr. Baldor

Mrs. D. was married seven years and could never carry a baby to term. Abortion was required before the end of the second month of pregnancy each time, and the period was shorter each time. This was the 4th pregnancy, and they all followed the same course and symptoms but with increasing severity. In each instance she vomited profusely, with much salivation constantly; the urine was progressively decreased until only blood came, just as in the last hours of the parathyroidectomy intoxication. Convulsions followed by coma called for immediate abortion, if life was to be saved. This time, however, Dr. Baldor tried the Synthetic Survival Reagent as her big ambition in life was to have a baby. Twenty hours after the injection was given, vomiting had decreased from 20 times a day to twice per day. The urine increased and, in 72 hours, she was passing half a liter a day. This urine still carried blood and albumen. In four days, she passed a full quart of urine per day. The vomiting disappeared entirely within two weeks, but the salivation had continued and, during the third week, vomiting started again. She was given another dose of the Survival Reagent, and all cleared up quickly thereafter. No more symptoms of eclampsia returned. She carried her baby comfortably into the seventh month, when she had an automobile accident, and spontaneous abortion threatened, so she was delivered of a 5½ pound baby that thrived well. She had no return of eclampsia symptoms and gained full health quickly.

Here we see again that the toxin that blocked the oxidations of function and the regulated energy acceptance by the working mechanisms, could be removed by an atomic group similar in kind but of higher O/R potential. The allergic uncontrolled spasms of the small blood vessels, and the anoxia caused by colloidal gellation, had to yield to restored efficient FCG function. The basic

pathology was met and corrected, at its very inception. Still, some of America's greatest biochemists and clinical experts claim that "the oxidation mechanism has no significant action or position in the maintenance of health or in the combat against disease." They are limited, of course, by the performances of the Krebs cycle, which to them is the whole oxidation mechanism. But, if one were to accept such a dictum, one would have to add *"it is impossible to die of asphyxia."* The predicament is rather contrary to progress.

TOXIC GOITRE AND CANCER OF THE STOMACH

CASE No. 4

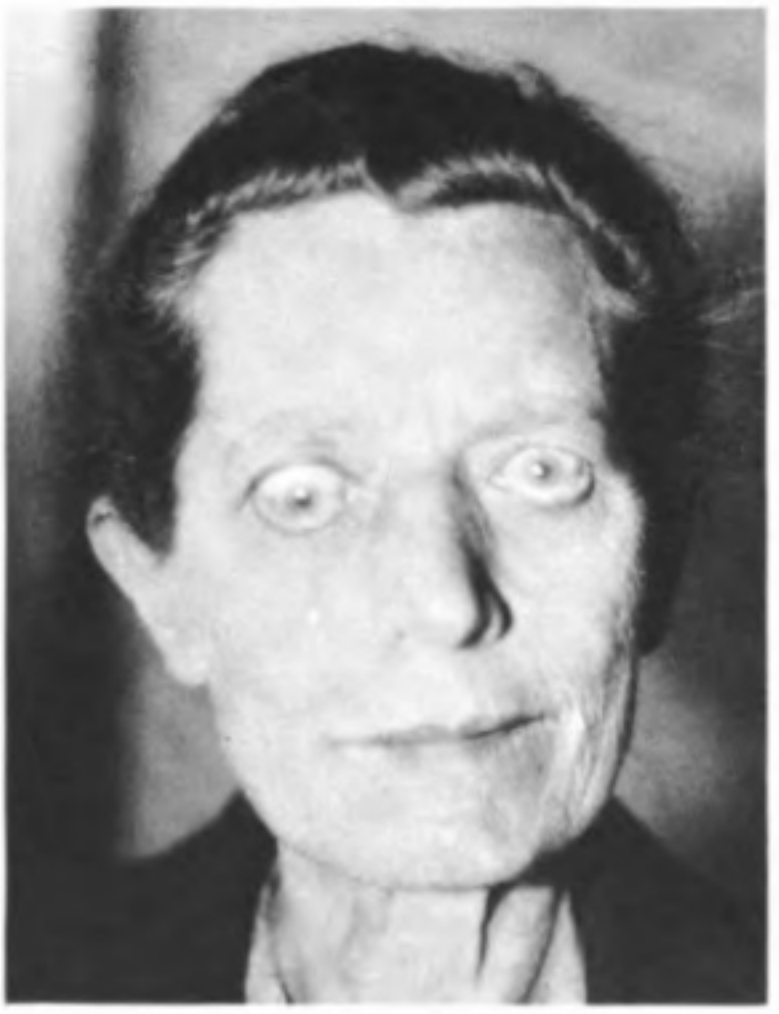
To show that one toxic agent (removable by one corrective attack) can cause a toxic hyperfunction as in Case I, and also cause a very high grade malignant neoplasm of the stomach in the same person and at the same time, the case of Mrs. W. is offered.

At the time this patient was treated, the Geiger Counter had not yet been invented, so it was impossible to estimate the earth's irradiations in her environment. However, it is noted that she lived in what is known as the goitre belt, a region of iodine deficiency and also of high cancer mortality rate. Her daughter had been treated for a rapidly developing brain tumor. Many other patients came from this region for treatment. However, one thing this study lacks is a systematic correlation of the terrestrial radiations with cancer incidence and also the number of conditions allied to cancer to be met; and most of all, how the terrestrial rays affect the recovery rates both of the neoplasms and of the allied diseases.

There was no history of cancer in the ancestry, but her husband died of cancer 8 years previously, and her daughter, with a very malignant tumor, was only 28 years of age as compared with the patient's age of 58 years, at her first visit. One recognizes here the vigor of the carcinogenic flux of this region. Both the mother and daughter made typical recoveries under the treatment. There was nothing in the geophysical environment that interfered with the cyclic reactions and the steady progress of the recovery process. One feature to be noted is that as cancer is associated with aging processes, this patient, at only 58 years of age, looked like a person twenty to thirty years older. The skin and tissues in general were senile, though the hair was not grey. During the recovery process the senility changes disappeared. The main features were as follows:

The disease started two and one-half years previously as a steadily-increasing nervousness, progressive cardiac weakness, tachycardia, increasing ease of perspiration, loose bowels, and tremor of characteristic hyperthyroid type. Radiographs showed considerable enlargement of the heart and mediastinal shadows early in 1927. There was dyspnoea on slight exertion or lying down. Exophthalmus developed rapidly, the skin was bronzed, and gastric distress and inefficiency set in. The feet and ankles swelled considerably, yet she lost weight, falling from 150 pounds to 108 pounds in less than nine months. The

physical examination revealed the exophthalmus as shown in the photograph before treatment. There was also a greatly enlarged lymph gland (walnut size) in the left supraclavicular space; the veins of the head and neck engorged with blood when she laid down, and percussion showed a marked increase in the mediastinal dullness. Examination showed the epigastrium and the whole area below the costal border down to two centimeters below the umbilicus on the right side to be occupied by a huge, bulging, solid, fixed, irregular tumor. The stools showed decomposed and occult blood. There was vomiting and great weakness and considerable pain throughout the abdomen. Thus the stomach, the liver, and probably the suprarenal glands were involved by the neoplasm. At the time of this examination she was very weak.



Mrs. W. before treatment showing the exophthalmus from toxic goitre excited by the carcinogenic toxin.

Mrs. W. after treatment and recovery from cancer of the stomach, and toxic goitre as secured from one chemical reagent. The exophthalmus is gone for good.

One dose of two cc. of a 10^{-12} solution of the (SSR) serial carbonyl system was used on September 28, 1929. The recovery process exhibited the usual cyclic three week reactions, with chills, fever, and general aching; and with improvement following each reaction until the recovery became complete. At last report, ten years later, she was in good health. We lost track of her thereafter.

Regular FCG function, both for producing and using ATP energy, was blocked and this showed for the thyroid cells, the stomach growth mitotic mechanisms and the general tissue oxidations as demonstrated by the senility changes. Still the Krebs Cycle oxidations went on, and fermentation supported

the neoplastic cells. Had we supplied a carbonyl group of FCG oxidation potential, we probably would have gained nothing. However, a carbonyl group of boosted O/R potential cleared the inactivator of FCG functions away so normal FCG metabolism (in contrast with the Krebs metabolism) was restored, and senility, toxic goitre, and cancer, all faded away permanently.

TOXIC GOITRE AND ETIOLOGICAL TOXIC FOCI

CASE No. 5 Dr. Jayme Treiger

In this case the pretreatment control or observation period lasted from September 12, 1953 until March 13, 1958. The development of the etiological factors with the progress of the disease itself was well noted.

Mrs. D. S., F. 27 years old, married, a thin brunette woman, very nervous, complained of dyspnoea, cold sweating, pharyngial spasm (sensation of an egg in her throat), able to bear heavy duties but not simple ones, urine sometimes fetid and strongly colored, acne, leucorrhea, sometimes bloody, and painful nodules in the right breast. These breast symptoms arrived after a second electro-coagulation of an ulcer on the cervix uteri, produced after the second childbirth. These nodules were helped by hormone treatment for a while but had returned, with further toxic symptoms as a tachycardia of 106 per minute, and slight thyroid enlargement, that started nine years previously. She had pertussis, measles and vericella during childhood.

During and since childhood, she had periodic crises of angina with high temperature and pus from the tonsils. Homeopathic treatment helped the tachycardia and the throat spasms and made her feel much better, but the basic pathology was not retarded, and she went to a gland specialist who treated her from November 1954 to January 1956. From him she received Dexamyl, Somniphene, Prometron, Ovocycline, Diiodotyrosine, Apliotil, Thiouracil, and Nodular on different occasions. She did not improve on this series of modified benzene rings, though enough were tried. This shows that the therapeutic conception was not based on physiological considerations, but was the fruit of modern pharmacology.

Feeling worse, she returned to Petropolis. The B.M.R. by Dr. T. showed a plus 45 and a Cholesterol of 122 mgms. percent on 3/12/58. She was now exhausted, extraordinarily excited, always tired, difficult to sleep, with frequent nightmares, pulse 106 per minute, and her blood pressure in a low range. She was given 2 millimicrograms of the SSR intramuscularly on March 13, 1958, and the reactions that followed are indicative of the sources of her toxins.

Reactions: Tonsilitis that was suppressed from activity while under the phenolic treatments mentioned above, started to be active with high fever, pus discharge and pain in violent periodic crises. The bloody drainage from the cervix uteri that was suppressed by the cautery started up again. However, one week after the treatment in spite of the strong angina crises, she was feeling very well, as if with renewed vitality. A few weeks later she reported again. The pulse was normal, 82 per minute, the blood pressure normal 120/90, and as

her good health was being restored, old symptoms of years of little difficulties returned briefly and disappeared. She felt good enough to not need a doctor. The throat had normalized and the cervix uteri had healed, and she did not return for more observation. She had received two injections of the SSR, the second one a year after the first, for while the cervix showed no abnormality on examination, there were symptoms suggesting reaction in the deep scars within. The BMR in February, 1960 showed 6% over normal, the breasts, tonsils, uterus, nerve responses were normal, temperature 36.7°C, pulse 60, the B.P. 110/70, and she enjoying the best health she had ever experienced.

In this case the etiological lesions that brewed the toxins that attacked the breast tissues and the thyroid gland were respectively the cervix infection and the tonsil infection. The cautery sealed up the drainage facilities, and made the scar tissue that was infected more anoxic. The reactivity of the reticuloendothelial cells of the tonsils to their contained infections was suppressed by the phenolic derivatives, so the thyroid was poisoned all the more. Further, the poisons from the cervix and those from the tonsils while showing some specificity to the thyroid and breast tissues, were also generai poisons and affected all of the tissues making her nervous and weak aside from a special thyroxin effect. Here the relation of the reactions (following the treatment with the SSR reagent, which were severe) to her improvement in tissue function showed that these reactions were not of a vaccination nature, but were actual reticle-endothelial battles against the disease agents going on in conjunction with the chain oxidation of these agents. Then, too, as the various FCG units were liberated from combined toxins and went back to work, she started to feel normal and her various functions behaved normally again. It is to be recalled that after the SSR was given the tonsils became acutely inflamed, and the cervix lesion broke loose with a strong inflamatory process. Thereby both lesions were cleared of their imprisoned germs and fibrosis integrated pathogens, and as the induced oxidations burned the pathogens away, the fibrosis disappeared also. The anoxic centers were wiped out, so the disease was cured right at its very inception. She has no more sore throats nor cervix troubles, and no more secondary effects, as thyroid enlargement or abnormal function. The breasts have no more nodules either. Her general health is normal. Her nervous system is steady. She sleeps normally and does not sweat as she formerly did. In other words, the pathology was fully reversed and discarded. This same course will be seen in the other cases reported here, and in all others when one takes the trouble to thoroughly check the recovery course.

CHAPTER VIII

ATROPHY, ANAPLASIA, AND NEOPLASIA

One may compare the correction of nerve atrophy and neoplasia very nicely in retinal cases where cell reconstruction can go on, but not cell reproduction. We may also compare the corrective responses in reproductive cells when anaplastic and neoplastic. In all four situations the correction is had by restoring normal FCG function, and thus a least common denominator is established in these supposedly widely varying conditions. So the basic pathology is the same. There is a suggestion that the requirement for neoplasia is the loss of the functional mechanism. In the Glioma, there is no development of functional mechanism, because the cells are too embryonic and immature or undifferentiated. In the Sympathicogonioma case one sees how the nucleus is separated from the functional mechanism, the fibrillar structure. The latter lays separated as a fibrillar syncitium supported by cytoplasm, while the reproductive portion is separated off as individual entities, — cells. Thus the functional mechanism is discarded. The return to normal which we are not able to see, must be like the maintenance of the normal, a correct balance and interaction of the reproductive and functional mechanisms. Thus the energy production in nerve cells must serve their work of repair and Nissle substance synthesis only to keep them normal. But when these systems are blocked the energy has only one place to go, and that is mitosis. When the working part is shed as in Sympathicogonioma, of course, there is the visible separation. The very high grade malignancy of such neoplasms may therefore be understood.

In this case of Neuroblastoma, which will be detailed a few pages farther on, visible separation of reproductive and functional elements of the cell is demonstrated, even though the child is born with the neoplasm latent in his system. In the Retinoblastoma case, the functional mechanism did not exist in the cells that went malignant, so the mitotic mechanism had no other course than to use the energy produced in the nucleus for reproduction. There was no other path for it to follow. It appears from these two cases alone that energy production is primarily evolved in the nucleus even though the nucleo-protein grana are found throughout the cytoplasm. In the glioma case these granules did not exist while in the Neuroblastoma case their connections with the nucleus were severed.

In the optic atrophy case to follow, complete atrophy was not possible as yet as a result of the retino-choroiditis, for regeneration of the affected cells took place with restoration of function. A toxic injury of bacterial origin is doubtless concerned here.

There is another situation due to toxic injury, that of chronic alcoholism where this weakness is transmitted from father to son for several generations, and the normally buried concepts of man's earliest days on Earth flash forth. A case given on another page should interest the geneticist in view of the foregoing remarks, for indeed hereditary transmission is concerned in all of these conditions. Even in this case of Delerium Tremens, where the suppression of

the balmy picture of the tropical garden with its flowers and snakes is broken by the effect of a toxin on the cerebral cells, the connection between the genes and the functioning elements is concerned, — the nucleus and the mitochondria. Evidently this is the toxin that caused the cancer in this case, for the delirium tremens returned during the 12th week reaction in his recovery from cancer of the face. The patient himself had never used alcoholic drinks but his father, and other predecessors did drink themselves to death. Still, the patient gave the D.T. symptoms during the correction of the neoplastic defect. Both defects were corrected by the reagent that corrected the hereditary neuroblastoma defect in the case to follow very shortly.

This case with its high malignancy characteristics teaches another very important fact, namely that trauma with its circulatory injury and consequent anoxia alone is not sufficient to cause cancer. Also it teaches that the recovery from this disease removed the carcinogenic factor that requires anoxia to become effective as our postulate outlines. For as will be seen this boy, after being cured by restoration of his FCG Survival Factor, did not again get cancer after being severely injured by the automobile accident that bruised him at the site of the former tumor. This case repeats the lesson learned from case No. 11 to follow.

Recovery Reactions

Cyclic reactions often characterized by the symptoms of the pathogenesis showing up in the reverse sequence to their coming, and accompanied by a local congestion at the lesion, as well as constitutional symptoms of grippiness as occurs in so many virus infections are to be expected periodically while recovery is in progress. The cycles run in a definite periodicity in which a three hour unit, or more often, a twelve or twenty-four, thirty-six, seventy-two or eighty-four hour interval is usually observed. Thus a reaction of chills and fever and achiness can show up twenty-four, seventy-two or eighty-four hours after the treatment. Or it may come the third week, the sixth week, the ninth week, or the twelfth, twenty-fourth, thirty-sixth, sixtieth, seventy-second, eighty-fourth, ninety-sixth, hundred and eighth, or later multiple of twelve weeks.

The local features in the lesion are congestion, swelling, hyperaesthesia, hyperreflexia, more or less pain, local heat, and maybe some bleeding. But this passes off in three hours or a multiple thereof, and improvement is then noted. When biopsies are taken of the lesion undergoing recovery in this way, it will be found to first undergo a coagulation necrosis and then a calcification much like accompanies or mediates the digestion of a blood clot, or of milk. Vascular ingrowth is observed, first angioblastic and fibroblastic tissue and mast cells or other white blood cells to help carry off the debris. (Koch, New York, Medical Record, October 30, 1920) Angioblastic tissue finally replaces the growth, and then functionating parenchyma grows in to reform the organ on physiological lines. The cure is therefore complete, as it could not start without preliminary elimination of the pathogen, and its functional status is returned. The sequence of events during reconstruction is exceedingly interesting and will be discussed.

PRIMARY ATROPHY OF THE OPTIC NERVE AND RETINA SEQUEL TO SCARLET FEVER

CASE No. 6

R. J., 14 years old, gave a family history negative to tuberculosis and cancer. The pretreatment control period and diagnosis are well described in the correspondence between the Henry Ford Hospital and Jennings Hospital experts as follows:

HENRY FORD HOSPITAL
DETROIT, MICHIGAN

Henry A. Du-----, M.D.
7815 E. Jefferson Ave.,
Detroit, Michigan

September 10, 1946

Case No. 453242 Re: R. J.

Dear Dr. Du-----:

Our first contact with the above named child, according to our records, was a precamp examination done by Dr. J. A. Jo---, of the Division of Pediatrics, in July, 1945. At this time his vision was recorded as being 20/20 bilaterally. He was seen by us April 15, 1946, at which time he had developed a scotoma in the right eye. Vision without correction on the right was 8/200, left 20/20. External examination of both eyes revealed them to be normal. Funduscopic examination of the right eye revealed a normal lens. In the macular area there was a chorioretinitis which fits the description given by you in your letter. The left eye was normal to funduscopic. Tangent screen examination was done which showed an absolute scotoma in the supracentral region. The periphery was normal; red field was reduced.

He returned to Pediatrics for a general physical examination by Dr. Jo---- April 17. Dr. Jo---- noted that the child had been seen by Dr. Ca--- who had applied .1 mgm. of 1/10,000 O.T. He had been negative to 0.01 mgm. when seen by Dr. Jo---- the previous year.

Sinus and chest X-rays were made. Dr. Do--- reported them as showing chronic pathology of both antra and probably the ethmoids and frontals as well, and were suggestive of a pan sinusitis. There was only a moderate increase in the broncho-vascular markings in the bases.

Blood count showed on 4-16-46 a hemoglobin of 13.5, white blood cells 10,100, red blood cells 4.66, polymorphonuclears 46, small lymphocytes 52, monocytes 2.

He was last seen April 20 by Dr. Di---- for ear, nose, and throat consultation. No foci of infection were found in the ears, nose or throat to account for his eye condition. Dr. Di---- reviewed the sinus X-rays and washed out the left antrum. The return flow was clear.

He did not return for follow-up eye appointment, and we have not seen him since. Trusting this information will aid you in your studies, we are,

Sincerely yours,
Henry Ford Hospital
/s/ E. L. W.----, M.D.
Surgeon-in-Charge
Division of Ophthalmology.

Per;
M. W. S-----, M.D.
rms.

Our History

Our examination made April 26, 1946, gave a history of headaches and chronic sinus drainage with acute exaccerbations, sore throats often, and a history of exposure to scarlet fever that did not take on him. He was slightly sick, but had no rash and did not peel. He noticed the eye condition when at a shooting gallery he aimed a gun using the right eye and found he could not see with it. Thus in one year exactly, a 20/20 vision went to zero so far as practical use was concerned. He could not make out objects with it nor read any size print. The area of "absolute scotoma" noted by the experts was an area of retinal and optic nerve "atrophy" that involved the upper half of the retina and the central field. There was some peripheral vision but it did not help much.

We gave him a 2 cc. injection of the 10^{-12} solution of the serial system of carbonyl groups (SSR), the Synthetic Survival Reagent, on April 26th. The sinus drainage and headaches soon ceased. The sore throats returned only twice during two reaction periods. But the other symptoms were aggravated during the 12th, 24th, 36th, and 60th weeks. During this last reaction the throat was most sore, and he had a fever of 103° F. as well as a typical scarlet fever rash that lasted only 12 hours. His health was perfect after that.

Examinations of the eye by the same experts found that the vision in the right eye was 20/400 in August, 1946, that is four months after treatment. Thus a definite improvement had taken place. In June, 1948, it was 20/100, and in October 27th, 1948, it was 20/40. Thus the improvement was continuing. Further reports, but without examination, claim his eye is perfect so far as reading fine print is concerned or for any other use. Here we see the polymerized scarlet fever toxin causing a degenerative change, and after it was broken down to the monomeric phase, it gave rise to the symptoms of the acute infection transiently. This was burned away in only twelve hours, and the recovery was complete. Other similar occurrences could be reported.

ACUTE CHORIORETINITIS SEQUEL TO PELVIC AND TONSIL INFECTION

CASE No. 6A — Dr. Jayme Treiger

Mrs. L. P. S., age 35 years. April 9, 1957 when first examined.

Her Pretreatment Control Period of observation extended until December 5th, 1958, during which time she received all of the homeopathic remedies, and a course of Cortisone and its derivatives with the most recent and efficient antibiotics. There was a steady downward course in her general health and her two chief areas of infection, the pelvis and the tonsils, and steady deterioration of the function and pathology of the retina of the left eye, that developed during the last six months of this period.

Physical Examination on entrance showed, fever of 38.6° C. with a bilateral tonsilitis that had persisted for the past three years, worse at the end of the menses. She was very exhausted because of excessive use of antibiotics and antipyretics. Also there was an inflamatory process in upper and lower abdo-

men, sequel to violent gynecological infection sustained shortly after marriage. The tonsils—red, hypertrophied; tonsils full of pus, Heart—O.K., Gall Bladder sensitive XXXX, Chaufford Zone XXX, and Ovaries inflamation. The menses were prolonged. There was no benefit from medication followed during the first six months. Indeed headaches, fetid leucorrhea and varicose veins were aggravated. Three months later oedemas of feet and hands on waking, and of the upper eye lid with severe sacro-ovarian pains were added to the symptoms, with severe chest pains, and blurred vision, also cracking of the skin on the palms of her hands. Fundascopic examination by the ophthalmologist showed no retinal pathology at this time, 7/18/58. The examination on 10/28/58 showed the left eyelid congested, vision blurred, photophobia. Fundascopic examination on 11/5/58 showed left eye vision 0.67 fundas turbid with exudate, and a focus of Chorioretinitis in evolution, juxta-papillary. Right eye normal. V--1, Signed Dr. A. H.

Prognosis—Healing with scar causing corresponding scotema.

This Ophthalmologist placed her on Cortisone and its derivatives, antibiotics and topical applications. One month later the eye was strongly congested, much worse than a month previously, and she could see nothing at all with the left eye, much worse than at the beginning when she still had some vision. Her weight was 80 Kilograms. Two millimicrograms of the SSR were given intramuscularly, 12/5/58, by Dr. J. Treiger.

Post treatment period, 12/6/58, swelling of peritonsilar lymph nodes, dizziness, uneasiness, violent pain in the pelvis recalling the time of her acute initial pelvic infection, with exquisite tenderness. Vision more blurred for 2 days. Copious flow of pus from genetiles for the past three weeks, 12/31/58. On January 12, 1959, she had severe tonsilitis that lasted for 3 days at the end of her menses, fever 39.5° C., great hypertrophy of the tonsils, weight down to 74 kilos. with terrible vaginal itching, that soon improved. By the middle of 6th week, 1/14/59, much better, temp. 36.7° C. On 2/17/59 her weight was 72 kilos, feels better, and shows cracking in the skin of the palms of hands as of ten years earlier, and skin desquamation. Temporary improvement of left eye vision. On 3/18/59 she reported she spent a wonderful week, including her eye. On 5/5/59 she reported she had two weeks of normal vision and was in good health generally. On 4/9/60 she had three days of reaction with slight swelling of the left tonsil, pain in the left eye, slight blur in vision, and then quickly normalized and has stayed well ever since. The pelvic and throat pathology completely disappeared. This was the 69th week reaction.

Fundascopic examination by Dr. B., reads as follows:

Dr. A. De S. B.
Petropolis
Estado de Rio de Janeiro

December 21, 1959

Dear Dr. Treiger:

The "Fundus Oculi" examination which was done on your recommended patient, Mrs. L. P. S. accused on the left eye an inferior nasal focus, entirely

healed of chorioretinitis all the way well into the periphery. There is *no abnormality concerning the vascular supply*. The visual field through comparison showed no abnormality. Therefore I did not try the instrumental perimetry.

Yours truly,
Dr. B.

Discussion. No scar was produced as always happens with corresponding blindness. The retina was restored. If or not a scarlet fever infection played a part in the etiology no one can say. However, the cracking of the skin of the palms of the hands and the desquamation that showed up as she finished her recovery may have some indicative value. Even though it may not be scarlet fever, it is a toxic effect of infection antecedent to the retinal lesion. In contrast to the former case which showed no inflamatory change reportable, but only the atrophic changes here the inflamatory changes predominated and the degenerative effects were also in evidence. This woman was also too heavy at first. She lost weight down to 70 kilos and then got thinner without losing more weight, showing a building of solid tissue with functional efficiency that went with her return of happy good health. Both foci of pus infection cleared up, and the non-pusy inflamatory and degenerative changes likewise disappeared. Thus an integration of the toxins of bacterial infection that excite no leucocytosis, as they are bound to and become part of a different tissue, are separated from their host cells by the SSR as we will show viruses are separated and carcinogens are separated, leaving the host cell in good functional status while the pathogen is destroyed. This accompanies destruction of unbound toxins that excite leucocytosis in other parts of the body.

Her reactions occured typically, twenty-four hours after the treatment was given and at multiples of three week intervals thereafter for a few times only, the sixth and the 69th weeks. The last reaction occured after the retina function had returned to normal and examination showed it was structurally normal. Thus the reactions are not to the lesion, but to the cause of the particular lesion, a matter much deeper than the toxic factor of the etiology. One case alone demonstrating this phenomenon, would be good to remember, but many such cases showing reactions for example coming five years after a large cancer of the stomach has been absorbed and health follows a normal course, indicate that a phenomenon is observed that clears up the predilection to the pathological response to the etiological factor rather than an attack on the pathogenic factor which is long out of the system. The correction is more basic than getting rid of the etiological agent and healing the lesions.

Confirming this, one recalls the case of cancer of the face of Dr. M. of a small town in Texas in 1927. He was 81 years old and blind with bilateral retine-choroiditis of several years standing. He was treated with the SSR. for the cancer and while that lesion was healing up, the vision returned, so that before the growth had entirely healed, his vision had so perfectly improved that he could see the string that was used to tie up packages in the country store and hung from the ceiling. This was visible to him as he entered the store. He

had reactions which he reported long after he considered himself cured. There was high fever, 106.5° F. three years after full recovery. During this fever, which was different from any he had seen in his long practice of medicine, he was surprised by a good appetite and the desire to work and expend effort. Here is food for thought. He reported the best health for years afterward.

Here are three different etiological factors, Scarlet fever, a mixed infection of the gonococcus with strep and staph resistant to all latest antibiotics, and the etiological factors in cancer. In the latter case, an infiltrating type of basal cell, solar rays were one big factor and high winds, frost bite, etc. that caused a circulatory injury, anoxia, etc. This circulatory injury was present in the mixed infection case too as scar formation was in progress with the vascular injury that goes with the inflammation, and neuron injury. In the first case (No. 6) the inflammatory changes were a minimum but the degenerative changes were neuron loss and scar were most prominent. This is rather strange as there was no inflammatory history, but a very rapid development of the degenerative change. The other cases of this disease we may review add nothing to the present meager information. This is sad since the presence of degenerative change away from the pus forming areas that supported the infection and toxin production gives one a chance to investigate the cause of degenerative disease. If we had sufficient data we could demonstrate its different steps. This much is brought to mind anyway, it is the job of an Ophthalmologist.

The old doctor had plenty of contact with scarlet fever, and he no doubt had an attack that gave lasting immunity of high degree, but maybe not enough to prevent or clear out a suppressed infection he had carried for some years. Did his immunity lay the foundation for the degenerative change in the retina? If so does vaccination which brings about comparable condition not play a big role in the great dominence of degenerative diseases of today? We say the virus or the germ toxin must integrate with the cell functional structure to bring about the loss of function and its sequel of tissue degeneration and visa versa. Does not vaccination do the same? If vaccination altered the steric set-up of the cell so fresh entrance of the pathogen could not take hold, then the vaccine must persist and do its damage to function. This would lead to degenerative changes with gene effects.

Let us compare this type of immunity with the oxidative protection used in Dr. Treiger's case (6A) and in the others. The scars did not form or they went away as they were replaced by functional parenchyma and the distant site of the infection that brewed the toxin, be it scarlet fever, ordinary pus infection, or some other factor that enters the etiology of cancer, also was wiped out. The cow experiments, a few of which are reported here, show that the most vicious staphylococci, etc., lose their pathogenicity and maybe become faithful members of the great Biological Economy after they are "cured" by oxidation restoration facilities. Inferior oxidations it will appear as we go along, are the basis of disease.

RETINOBLASTOMA OF BOTH EYES (GLIOMA)

CASE No. 7

R. L. was one year of age when the disease first made its appearance. In less than a year the left eye was filled with a tumor mass, irritated, swollen, and blind. The diagnosis was made clinically by Dr. C-----, a noted opthalmologist, of Wichita, Kansas. He removed the eye. The pathological report confirmed his diagnosis of *retinoblastoma.* The accompanying document from the court records gives the gross and microscopic pathology. This was in May, 1934.

Wichita Hospital

5/5/'34 **PATHOLOGICAL REPORT** File No. 4120

Name L .Rita Coleen Room 111 c. Case No. 2468

Age 23 mo. Sex F. Race W.

Surgeon Dr. Cheney Examined by Dr. Harold M. Palmer

Pre-Operative Diagnosis: glioma of retina - left eye

Post-Operative Diagnosis: Same

Gross Pathology Eyeball having a normal external apearance. On section, the posterior chamber is practically filled with a greyish friable tumor mass which seems to be attached to the region of the nerve head.

Microscopic Pathology Section of tumor shows rounded dark staining nuclei of cells practically devoid of cytoplasm set in a thin connective tissue stroma having no characteristic arrangement. Marked necrosis is present in some areas and round cell infiltration may be seen in some areas. Section of nerve head shows no tumor tissue.

Pathological Diagnosis Glioma of retina.

Signed Harold M Palmer

Form [illegible] Hospital & Physicians Record Co., Wichita, Kansas

Within a year the right eye showed the same changes. The same surgeon observed the same disease here and so stated that it was the same disease that had attacked the left eye. This is a well known characteristic of this disease. Prof. Frohlich, of the University of Michigan, testifying as an expert, stated that there is nothing known to Scientific medicine that can combat this disease. Even when operated early it returns and kills. She was taken to a renowned ophthalmologist in Niles, Michigan, who made the same diagnosis and referred her to us.

We examined her with Dr. H-----, a good specialist, and made the same diagnosis. He found that one-third of the retina was replaced by the neoplasm, including the optic disc. The eyeball was bulging and distorted, the iris

dilated and fixed. There was no ability of the iris to move from its infiltrated attachments. Behind the pupil was a yellowish, pinkish reflection to light, showing that a tumor was present within. The area where the left eye was removed was not healthy, but showed neoplastic degeneration, though only in a minor degree. Both foci disturbed her. At this time, November 25, 1935, she was over two years old, and we gave her one cubic centimeter of the 10^{-12} solution of the SSR serially arranged carbonyl groups with free radical terminals by intramusucular injection. The irritation of both foci soon left. Every third week she exhibited a reaction, with general achiness, slight fever, and an aggravation of the irritation of both foci. Each reaction lasted a few days and was followed by improvement.

Examination on August 18, 1936, showed that all visible pathology had been removed. Dr. H---- and Dr. W------ made fundascopic examinations and found the retina normal, fully restored. Since she lived so far away, we gave her another dose for assurance, and no reaction thereto ever developed. She has remained well to date with perfect vision in the right eye. She went through her school courses and college at the head of her classes, is now married and a healthy, happy woman. Reports made in June, 1950, and June, 1953, and September, 1960 confirm her full recovery.

This case with those obtained in Canada by using the same treatment, are the only cases of Retinoblastoma ever cured. And as Prof. Frohlick, of the University of Michigan Department of Ophthalmology stated, "there is nothing known to scientific medicine that can cope with this disease," there is no information available as to the changes in the retinoblastoma cells during the recovery process. This is unfortunate for here we have a tumor of a nerve tissue that is supposed not to be able to reproduce in its normal state, even for a reconstructive or compensatory purpose, that does reproduce wildly with great stubbornness, as a malignant tissue. In this case and the others, the retina was restored with good vision performance. In this case it was 20/20. The question is, where did the new neurones come from, or were they some of the tumor cells that were able to reconstruct functional mechanisms as soon as the FCG's were liberated by the SSR? Thus, an undifferentiated tissue probably because of FCG inactivation, went malignant, and then underwent normal differentiation so as to be able to function and take its place in the organism's economy, when its FCG's were liberated. This sequence of events calls for deep thought. It shows full reversal of the malignant state.

EUNUCHOIDISM

CASE No. 8

J. S., at age 14, was subject to infections of the respiratory tract and skin, presented marked obesity, female type shape, was very dull mentally, and was found to have an infantile penis and undescended testicles. In fact they had not even descended to the canals. He had been under thyroid and pituitary hormone therapy from the age of 10 in 1931, to age 14, in 1935, without any improvement mentally, physically, or in his resistance to his infections. He

was closely watched by Dr. S., a relative, who reported that in this time the testicles had not even entered the canals. Thus the disease was classified as the type that is not helped by modern therapy and is permanent.

He received his dose of the SSR serial system of carbonyl groups in November, 1935, and in three months he lost weight, his infections were gone, the left testicle had traversed the canal and was entering the scrotum. After the sixth month both testicles were in the scrotum, the penis was developing and the pubic hair distribution became masculine. He grew taller, his hips reduced and his shoulders and jaw developed to good proportions. He became very bright in his school work, soon making up for his backward position. He became a good athlete. He entered the army and was soon promoted to a corporal. He is married, and is raising a nice family of children of his own. At the time this testimony was given he was in the Army Law School where he was making splendid progress. His boy and girl are physically normal and mentally excellent. Thus his genes were re-established in normal line. One may contrast 14 years of FCG insufficiency with a few months of rescued FCG efficiency, placing him in the normal progression of full health.

CANCER OF THE TESTIS

CASE No. 9

Mr. T., age 38 in June, 1925, when treated with SSR serial system of carbonyl groups with free radical terminals, his testis had become malignant six months previous to its removal. At this operation, no metastases were noted. The biopsy report was "Medullary Carcinoma of the Testis." Recurrence showed in the groin and scrotum within six months, and another operation revealed that the neoplasm had invaded the abdomen. Removal was attempted and the microscopic report on the material removed read again "Medullary Carcinoma of the Testis." Recurrence was not long in coming with more rapid spread of the disease. The abdomen was again opened, but was found so inoperably involved and a biopsy is all that was done. This biopsy was reported: "Carcinoma, probably secondary to previous carcinoma of the testis, as the cells are histologically similar."

At this time he was rather emaciated, and exhausted, and a general cachexia called for an early termination, — a hopeless prognosis. Through the intervention of Dr. Alpheus Hoyt, this patient was given the SSR reagent in June, 1925. He regained his health and all tumorous tissues disappeared. He remained well thereafter and in 1946 he offered his report stating he was perfectly well. This was 21 years after he received his treatment in the terminal phase of the disease.

The preceding case of anaplasia and this case of neoplasia were corrected

by the same reagent which restored good FCG function in both. Here is food for thought.

MALIGNANT SYMPATHICOGONIOMA

CASE No. 10 Dr. Julian Baldor

John L., age 13 months, developed a tumor in the abdomen that required an exploration on September 25th, 1951. A retroperitoneal growth had infiltrated the region too thoroughly to permit removal of any more than a biopsy. On October 6th, examination revealed a visible bulging of the abdomen in the umbilical region which we found on palpation to be the size of an ordinary Florida grapefruit, about 10 to 15 cms. in its diameters, firmly fixed to the surrounding structures. The stools were bloody showing the intestine was invaded, and the blood count agreed with his pallor, — 2,300,000 red cells with a hemoglobin of 52%. The next day he received from Dr. Julian Baldor an injection of the SSR reagent, the serially arranged system of carbonyl groups with free radical ends, 2 cc's 10^{-12} solution. Recovery began promptly and at the end of a year no more tumor could be palpated on careful examination.

On May 5th, while in good health he was run over by an automobile and sustained a broken leg and abdominal injuries. While in the hospital for repairs, he was carefully examined by the same surgeons who had removed the biopsy. They found him free of any trace of palpable growth. He made a nice recovery and is in excellent health still. His blood count on April 5, 1953, was 4,750,000 red cells, hemoglobin 87.5%. All the documentation and facts of this case were put in the Florida State Court records by his surgeons in May, 1953. His last report was good health in 1958.

Discussion

The case of Sympathicogonioma is of interest not only because it is so very highly malignant, but that the mitotic mechanisms, which are now the malignant cells, are separated from the nerve fibrillae, the functional mechanism, as entities. The functional mechanism is entirely shed or discarded and, of course, as an immature and unorganized fibrillar syncitium. The malignant cells are practically only made of nuclear material. It is unfortunate that periodic sections could not have been taken of the tumor as it normalized, to see if the neoplastic cells made any efforts to form cytoplasm and fibrillae, as a recovery response. In the retinoblastoma case, the new neurones that restored the retina to normal structure, probably had their origins in the neoplastic cells, as new neurones are supposed never to be formed. In the case of optic atrophy this assumption would also have to be made with the conclusion that the new functional retinal neurones were developed from remnants of atrophied cells that probably were integrated with toxin as in symbiotic viral infections, and the atrophy was never complete enough to prevent reconstruction after the FCG was relieved of its impediment. That the FCG is the position that is stopped

SAINT ANTHONY'S HOSPITAL, INC.

Saint Petersburg 6, Florida

PATHOLOGY REPORT

Name L Baby John

Tissue No. O-1558-51

Age **Sex** Male S.M.W.D.

Date September 25, 1951

Physician F. H. Langley, M. D.

Clinical Resume: ABDOMINAL MASS OF RECENT DURATION, PROVED NOT TO BE THE BLADDER. TESTICLES PRESENT IN SCROTUM AND APPARENTLY NORMAL.

SPECIMEN: BIOPSY OF RETROPERITONEAL TUMOR

GROSS: The specimen measures 5 x 3 x 4 mm. in size. It appears to be partially encapsulated by a relatively thick, fibrous membrane immediately beneath which is a brownish discolored thin area 1 mm. in width that separate the underlying, somewhat lobulated, gray, friable tumor from the capsule.

FROZEN SECTION DIAGNOSIS: UNDIFFERENTIATED CARCINOMA.

MICROSCOPIC: Sections reveal a trabeculated, fibrous, thick capsule which merges with the underlying tumor. Immediately beneath the capsule tumor cells are arranged in small clusters surrounded by thin strands of fibrous tissue. Large dilated vascular spaces are seen in this area. In the deeper portions, the tumor consists of strands of small lymphoid like nucleated cells which are separated by intervening masses of a fine eosinophilic sieve-like network of tissue. The nuclei vary from a round to an irregular shape and the chromatin from a fine granular to a heavy dark staining clumped variety. Cytoplasm is scant. Near the capsular surface, the nuclei assume in some instances a spindle shape. Scattered throughout the tumor are large relatively giant sized nuclei having a hyperchromatic appearance. There are areas of hemorrhage. Tumor cells are found in the vascular channels. Some sections show a tendency toward the formation of rosettes although these are very poorly defined.

DIAGNOSIS: IMMATURE TYPE TUMOR OF NEUROGENIC ORIGIN, SYMPATHOGONIOMA.

LHD/fa

L. H. DOMEIN, M.D.
Pathologist

PHYSICIANS' RECORD CO., CHICAGO 5 · 5812-1249-10M

from functioning to cause the pathology in each condition is quite evident.

Often a biopsy may reveal a tumor to be of grade one or two malignancy, and after the tumor is removed the sections show it is definitely a grade four, thus being changed by the manipulations of the biopsy, the effect of the anaesthetic, etc. The case to be reviewed now is one that was exceedingly anaplastic and a grade 4 malignancy at biopsy, as the examinations of the specimen by several leading pathologists and many well trained practitioners of medicine found. Some microphotographs are submitted besides.

This case shows that the Survival chemistry, as established by the high efficiency synthetic carbonyl activity, may fade out in ten years when the environmental conditions are adverse as they were in this case, and the data should be compared with that of the case that follows it, where a similar neoplasm of malignancy was cured after the Synthetic Reagent was given and still remains cured 36 years afterward. Here the environmental conditions were satisfactory, even though she was submitted to the trauma of four natural perfect childbirths, and no return of the neoplasm ever threatened. As in the neuroblastoma case just reviewed, trauma alone is not sufficient to cause cancer or its return. The anoxia may thus be established for a sufficient time for the co-factor, the pathogen, to make the integration. However, with the improved carbonyl function established by oxidizing the pathogen away from the FCG system, so that the FCG is activated by a carbonyl group, a sufficiently high O/R potential is established to burn the pathogen, instead of condensing with it. Still the survival chemistry is a comparative matter, as any other phase of life, and as this case demonstrates.

It will be seen as we go along, as in the above case, that anoxia alone is not sufficient to cause cancer, A CO-FACTOR, be it a virus or other carcinogen, is required. Where trauma is not concerned in causing the anoxia, toxic amines of colon origin may prepare the soil, and the response may not be neoplastic, but a necrosis instead.

GRADE 4 SQUAMOUS CELL CANCER OF THE CERVIX UTERI

CASE No. 11

Mrs. M. W. came under the care of Dr. L----, Dr. H----, and Dr. D----, of Medford, Oregon, in January, 1940. Dr. L---- examined her and found that she had an enlarged and fixed uterus. She had a cervix that protruded into the vagina and could be seen by a vaginal examination with a speculum; a cervix which was indurated and showed islands of apparent new growth. A diagnosis of far advanced cancer of the uterus was made clinically. Dr. L---- reported that her cancerous condition would probably, if untreated, end her life within a year. That because of the fixation of the uterus and the involvement of the adnexia, it was his opinion, that it was not a surgical case, as surgery would have had to be too extensive. It was too late for that sort of thing. The case had already entered the cachexia stage as Doctor L----'s testimony reveals: "She had lost 30 pounds in six weeks, was complaining of general weakness and

had a rather poor color at the time." As Ewing states: "Characteristic cachexia in uterine cancer develops in the terminal stage of the generalized disease, but when the lesion is localized in the pelvis, cachexia is missing."

Dr. L---- gave the SSR treatment, 2 cc.'s of the 10^{-12} solution on March 20, 1940, December 30, 1940, and October, 1941. His examination made a year and a half later, in the summer of 1942, found that: "She had gained weight, she had gained color, and improved in general appearance. The mass in the abdomen had subsided to the extent that he could no longer palpate it. The appearance of the cervix by examination with the speculum appeared normal."

Before giving the SSR injection she was placed in the Medford Hospital, and there Dr. L---- removed a specimen from the cervix for microscopic study. The slide was very carefully prepared by Dr. Green, the hospital pathologist, and diagnosed by him as squamous cell cancer of the cervix uteri. But the case was so interesting that he sent the slide to Professor Hunter, the Pathologist at the University, who established the grade of malignancy. Dr. Hunter, in his letter to Dr. Green on January 27, 1940, wrote: "I see a decidedly invasive and anaplastic carcinoma which occupied well over half of the tissue . . . I would if asked to do so, grade this one as a IV."

[redacted]

WARREN C. HUNTER, M. D.
PHYSICIAN — PATHOLOGIST
U. OF O. MEDICAL SCHOOL
PORTLAND, OREGON

Depts. 241

January 27, 1940

Dr. R. A. Green
Community Hospital
Medford, Oregon

Dear Doctor Green:

In the section (2751) from the cervix described in your letter of the 25th I see a decidedly invasive and anaplastic carsinoma which occupies well over half of the tissue. The cells occur in nests which have replaced the original connective tissue and muscle. They are poorly differentiated, the best of them being ovoid and thus resembling transitional squamous cells; more often they are spindle-shaped and like those of the germinal layer of transitional squamous epithelium like that occurring over the vaginal aspect of the cervix. There is some modification by reason of inflammatory reaction both in the masses and between them. Numerous mitoses are found. Such a growth would be radiosensitive and in view of the surgeons finding of fixation of the uterus I should think that palliative x-ray therapy is all that is justified in the event that the patient can afford to have this done. While I do not take much stock in grading I would if asked to do so, grade this one as a IV.

I presume you wish the section returned, hence am enclosing it with the letter.

With kindest regards, I am

Cordially yours,
Warren C. Hunter
Warren C. Hunter, M.D.

The confirmatory diagnosis made by Dr. Weller, professor of pathology at the University of Michigan, was secured to complete our records. He found the specimen well prepared, and diagnosed it instantly as follows:

UNIVERSITY OF MICHIGAN
ANN ARBOR

PATHOLOGY

December 7, 1944

Dr. George
5022 W. Warren Ave.
Detroit, Michigan

Dear Doctor

Prepared section #2751, our 3823-LAM. Tissue from cervix: Medullary squamous cell carcinoma of poorly differentiated type. Histological grade IV.

Sincerely yours,

Carl V. Weller, M.D.

CVW:de
Enc.

Dr. Weller's statement that the slide was well prepared is incorporated in an affidavit which reads:

STATE OF MICHIGAN)
COUNTY OF WAYNE)

On this the fifth day of February, 1945, before me, a Notary Public, appeared Virginia Yaney who deposes and says:

On December 7, 1944, at the request of George M. D[illegible], M.D., of Detroit, Michigan, I took a slide, prepared section No. 2751, of Mrs. Mary [illegible], to Carl V. Weller, M.D., at the University of Michigan, Ann Arbor, Michigan.

After studying the slide for a minute or so Dr. Weller's diagnosis was complete. Upon asking if there was anything wrong with the slide, Dr. Weller stated, no, it was a good slide. The slide was then handed to me as well as Dr. Weller's diagnosis.

Virginia Yaney

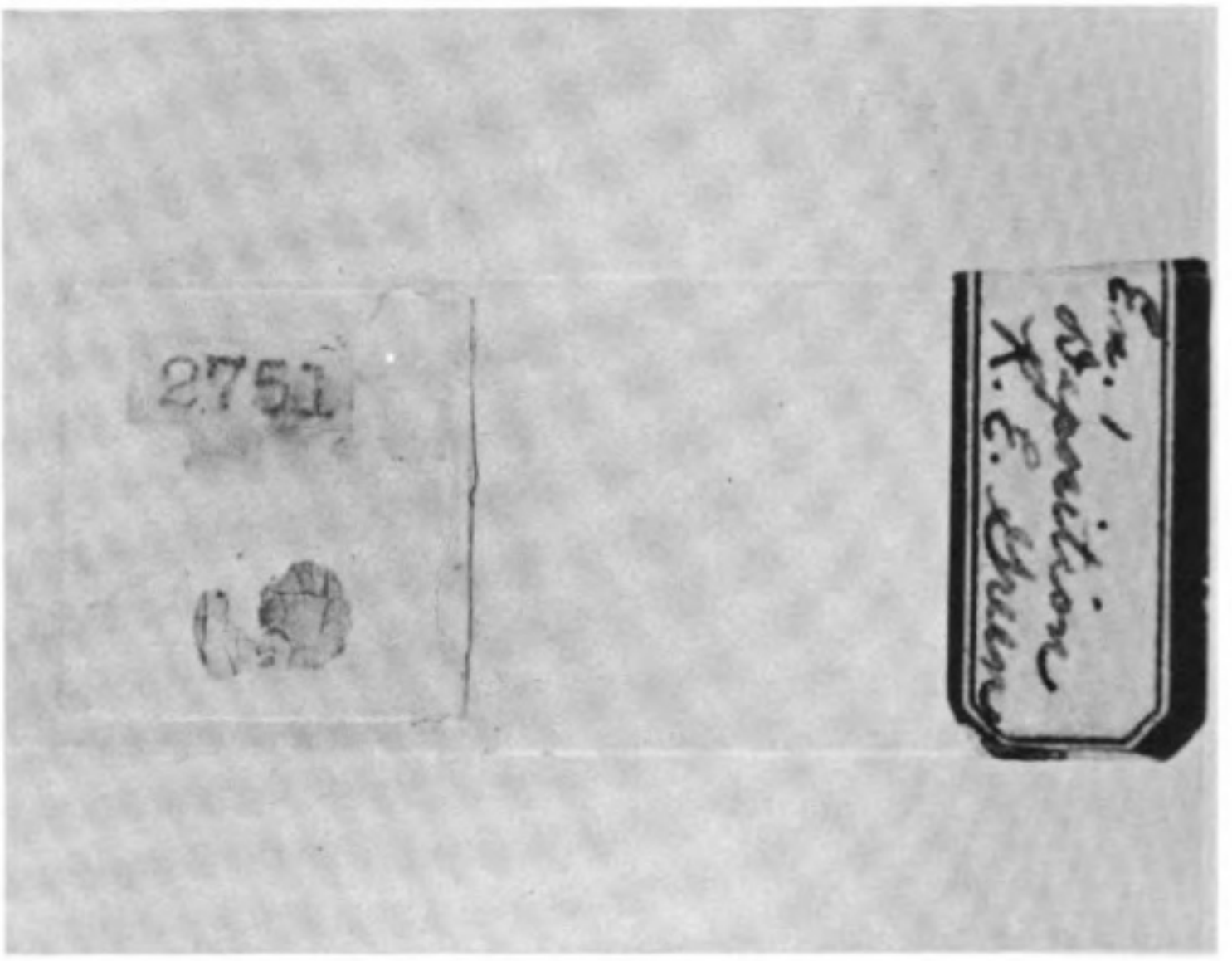
Sworn before me, a Notary Public,
this 5th day of February, 1945.
William P. Tefft

My Commission expires
My Commission Expires July 9, 1945

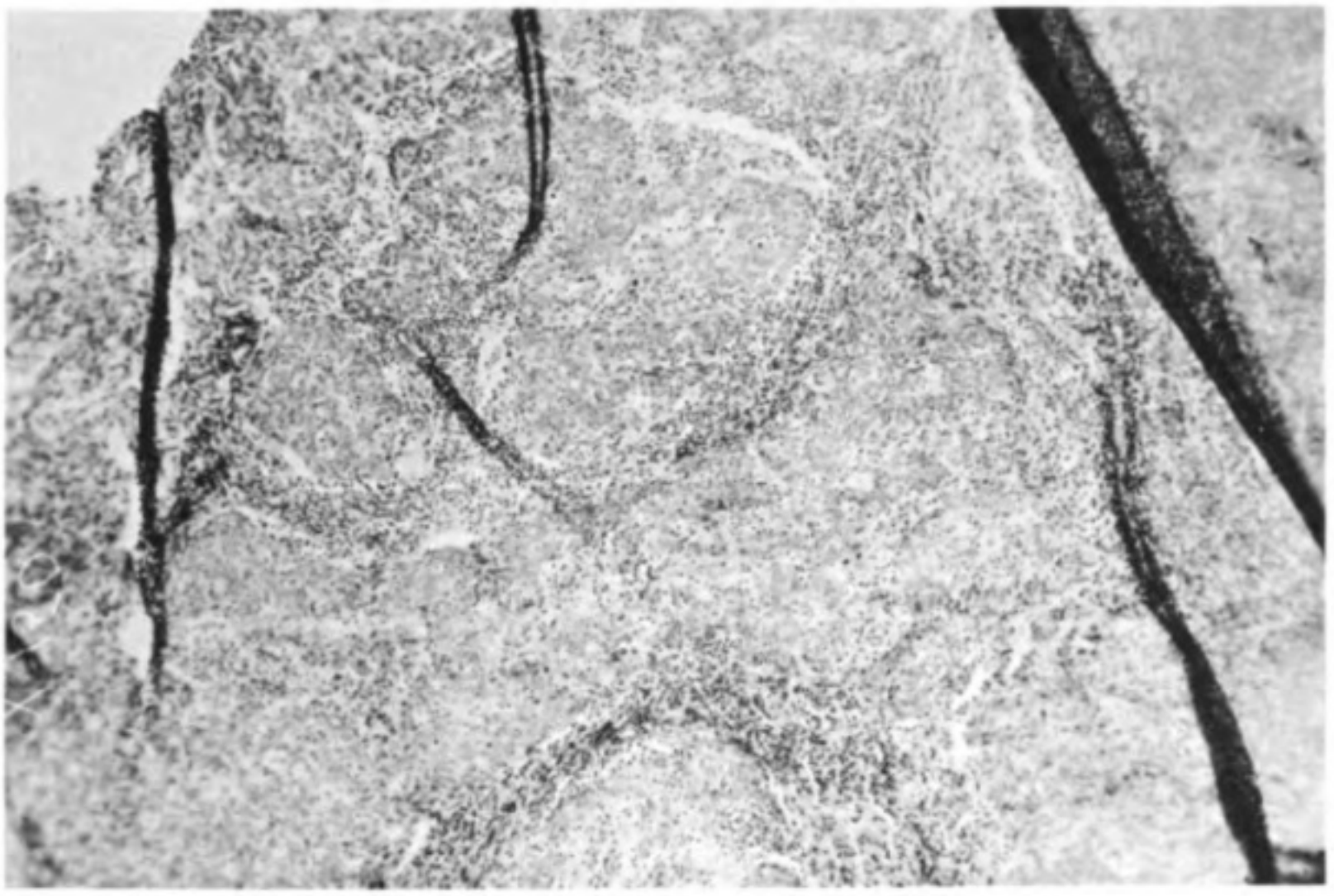
During her fifth year after treatment it was desired to ascertain her exact status so an exploratory laparotomy was done by Dr. Haines in June 1944. He found a normal cervix and uterus to which a small fibroid was attached. He removed the body of the uterus and the fibroid, but left the cervix in place as he found it perfectly normal. On gross examination the uterus and fibroid were normal, that is no signs of malignancy were observed but they were both submitted to careful serial section for a thorough microscope search for malignant cells. None were found. Dr. Inskeep, the pathologist, found no malignant cells and testified that the fibroid was benign.

Here we see, that at the time Dr. Haines operated on Mrs. M. W., there was no evidence of this Grade IV carcinoma present. Dr. Inskeep's report shows that no cancer cells were found. This indicates that, except for the benign fibroid tumor like so many healthy women carry ordinarily, the uterus was perfectly normal. Thus, we believe, she was found cured several years after being treated in the terminal stage of Grade IV cancer of the cervix when her life expectancy was less than a year.

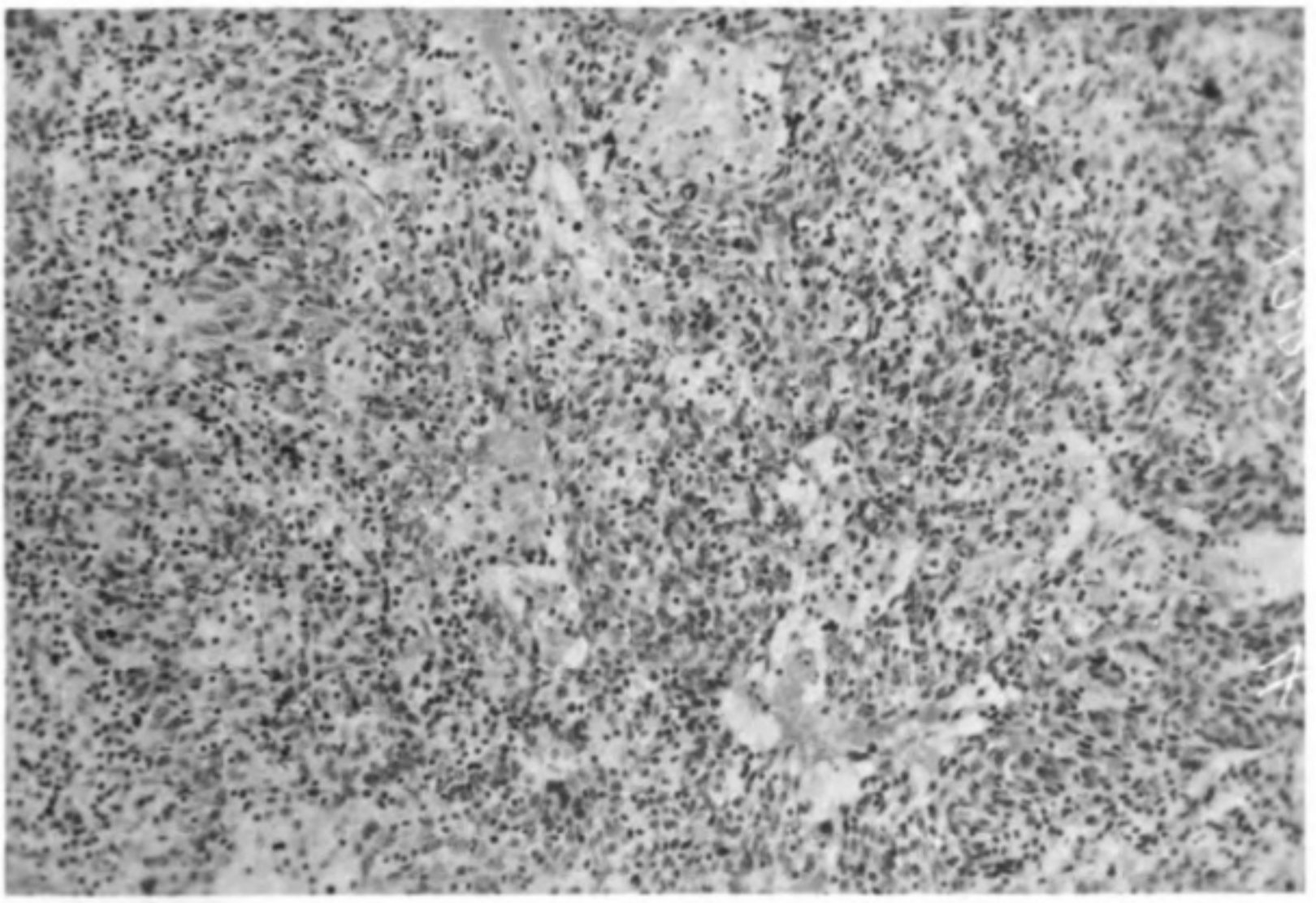
A photograph of the slide carrying the biopsy specimen well placed under the cover slip is given, and three microphotographs made by the Harper Hospital expert are also submitted for your study.



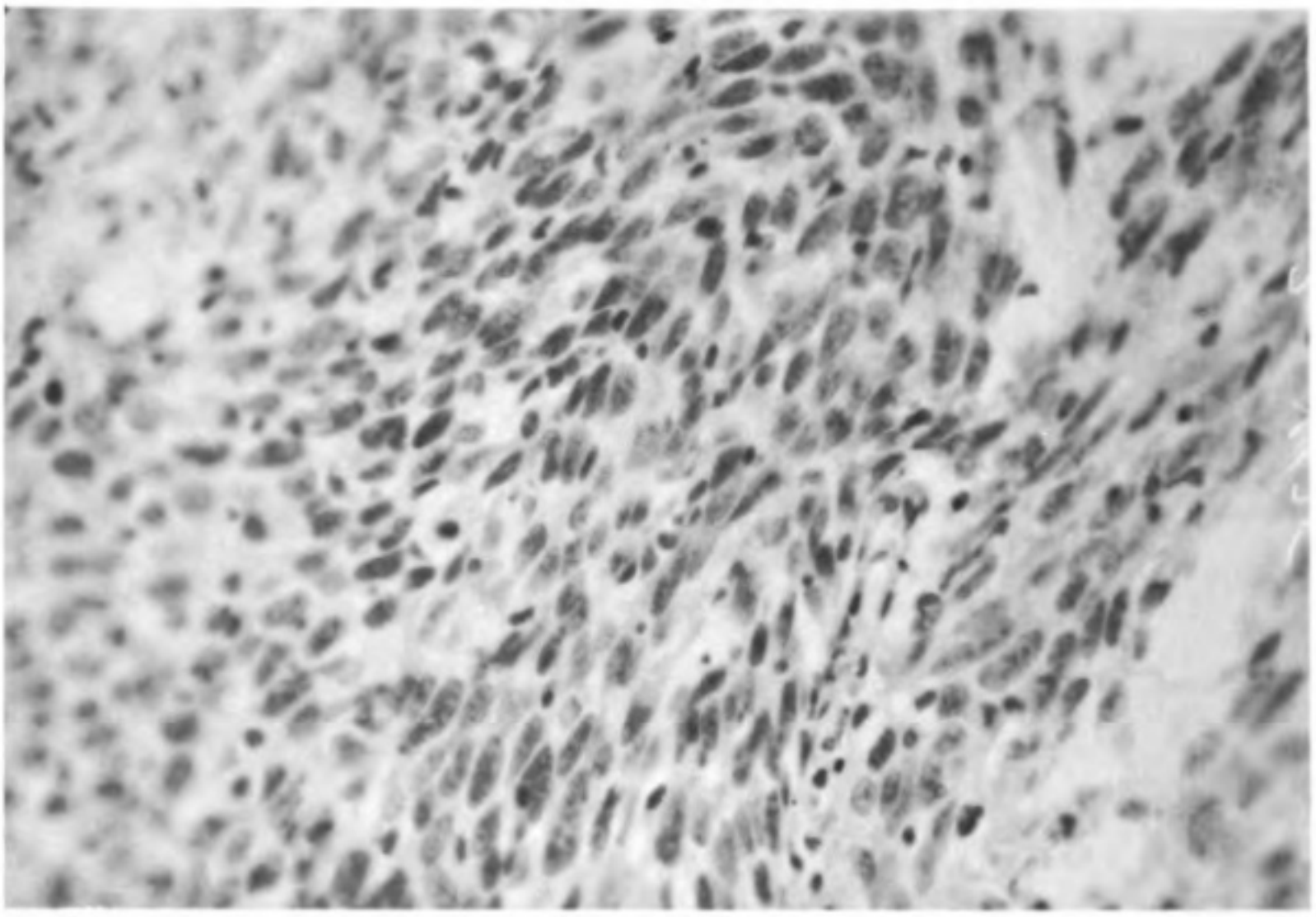
Photograph of the Biopsy slide showing its perfect condition.



Low Power Magnification of part of specimen Microphotograph by Mr. Rinelander of Harper Hospital. (150X)



Medium Magnification Microphotograph by Mr. Rinelander, of Harper Hospital. (200X)



High Power Microphotograph by Mr. Rinelander (675X)

Mrs. M. W. was seen by Dr. L----. again just a few months before her death in December, 1950. At the time of her death he was not aware of the sub-total hysterectomy. Dr. L---- found a very foul-smelling friable mass with the gross appearance of a disintegrating carcinoma in the left side of the pelvis and involving the lower end of the large bowel. A complete autopsy was not performed and no biopsy was taken post mortem. He reported that the condition leading to her death was cancer of the uterus.

In light of the whole case history, it is questionable that the original Grade IV squamous cell carcinoma of the cervix uteri in 1940 was the cause of her death in 1950. Her death should have occured within the year period, had there been a connection between this pathological condition, and her death.

Even though the exact cause of death is not established, this case serves our purpose here, namely to show that the survival chemistry can be restored to curative efficiency and that it persists for a period of years without further treatment. That when ill health threatens again, its repetition must be required to restore the desired survival chemistry again as in the first instance.

This case also illustrates the importance of continued medical observation of a patient even after the accepted five year period. That the survival factor after being restored synthetically can be subsequently destroyed by inhibiting factors.

This case should be compared with the case of Mrs. T., who also had advanced squamous cell carcinoma of the cervix uteri. Mrs. T. is enjoying best

health over thirty years after treatment. One sees that the survival chemistry is subjected to environmental influences. In Mrs. W.'s case, they were the worst possible. She was on the Welfare for many years prior to her death and the type of care she could receive was limited. In the case of Mrs. T., they were ideal even with the traumata of four childbirths. Thus the physician must carefully measure all influences that may determine health in each case and see that the ideal is maintained, both while the treatment is followed and after recovery occurs.

SQUAMOUS CELL CANCER OF THE CERVIX UTERI

CASE No. 12

In August, 1923, Mrs. T. was 31 years old. She could never carry a baby to term and spontaneous abortion took place. For over a year before seeing a physician she had irregular bleeding, mucopurulent discharge and increasing pain with progressive reduction in the capacity of the urinary bladder. She consulted Dr. T. who took a biopsy. A very responsible laboratory in Detroit made the diagnosis of squamous cell cancer of the cervix as follows. This diagnosis was also confirmed by several visiting pathologists, and a high grade of malignancy was observed.

OWEN CLINICAL LABORATORY

TELEPHONE MAIN 9297
DETROIT, MICH.

PATHOLOGICAL REPORT NO. S-89

Dr. L. S. Tupper,
Redford, Michigan.

PATIENT Mrs. T[illegible]

REPORTED 8/1/23

SPECIMEN Tissue-cervical

Sections show an atypical proliferation of squamous epithelial cells which have markedly infiltrated the underlying tissues.

Diagnosis: Squamous cell carcinoma (Epithelioma).

R. G. Owen M.D.

My examination was made two weeks later and the findings were those typical of a far advanced widely invading squamous cell type of cancer. It had invaded all the structures of the pelvis, but was more extensive on the right side. All the normal contours were obliterated and the structures hardened by the firm infiltrations. The bladder wall was extremely involved and the mass could be easily palpated above the pubes half way to the umbilicus. Her surgeon did not offer an operation, or recommend any treatment, but watched her progress with interest and took care of her four pregnancies later on. This case was not so far advanced as the case previously outlined. She bled profusely, especially since the biopsy was made, and she gave a yellow color that one would attribute to the prolonged excessive bleeding aside from the natural cachectic effects of a neoplasm that had broken through into the abdomen.

She received two injections of 2 micromicrograms of the Synthetic Survival Reagent (SSR) on August 7th and 21st, 1923. She had the typical reactions of

general achiness as one observes in most viral infections. The bleeding soon stopped, the pussy drainage soon stopped, and her color took a change for the better. The pain steadily let up and the growth on examination became softer, more elastic and movable and the normal contours of the pelvic structures returned. After the 36th week no more evidence of the disease was observable, but the right side of the cervix was a little deficient. This did not fill in fully to make a symmetrical cervix until a few more months had passed.

Her reactions came most vigorously on the twelfth and twenty-fourth weeks. They exhibited chills, fever, achiness, pains in the back and increased urinary frequency. But after each reaction her health gained very rapidly, and the bladder capacity normalized. She became pregnant and after a normal term of gestation gave birth to a fine healthy boy, with a normal easy delivery, managed by her surgeon, a man of exceeding expertness. Three more children were subsequently born at two-year intervals. All are fine physical specimens, and the births were easy and normal in every way. There was never any return of a neoplastic condition or any other trouble, nor even any serious sickness. She has been remarkably resistant to colds besides, and is still alive in perfect health 36 years after being treated for a terminal stage squamous cell cancer of the cervix that was widespread into the abdomen.

The important lesson from this case is the fact that she had plenty of exercise, taking care of a big strong husband, and four vigorous children. She did the family washing with tub and rubbing board and had plenty of exercise of the abdominal muscles that massaged the intestinal wall, aiding its movements and its circulation. Her diet was well chosen and well prepared, and her life was happy and inspiring. In contrast to the previous case one sees that the environment was very favorable for the maintenance of a good Survival Chemistry.

This case is also interesting in that her husband developed a cancer of the prostate gland 33 years after his wife was cured. The hospital put him through surgical and radiological procedures without offering a diagnosis. Radioactive Cobalt was new then and used vigorously. He quickly suffered widespread bone metastases and died in great agony. The infectious nature of the provocation of neoplasia may be inferred from this history, as well as its conjugal nature. It would have been a helpful observation to remove the cause of the husband's cancer with the same reagent that cured his wife. But the hospital doctors had no guiding information.

Trauma of four parturitions did not excite cancer in her, and neither did the exposure to the viral agent that infected her husband ever since her infection had been contributed to him over thirty years earlier. Here is food for thought.

CHAPTER IX

SURVIVAL FACTOR CHEMISTRY

The Antibiotic Problem

There is no evidence that a high efficiency system of oxidation exists in bacteria, especially in disease germs. However, they do depend upon an unrecognized carbonyl function that is activated by electrons received from the double bonds of an ethylene linkage, even in the performance of the Krebs Cycle. This is seen where it is necessary to keep the cycle going by supplying the unsaturated dicarboxylic acids, whereas the saturated dicarboxylic acids, as succinic, are of no help. Thus, even in the low level Krebs Cycle, upon which bacteria depend, activated carbonyl function of this particular order is required. Here, antibiotic activated amine groups have a position in which to produce their toxic effects, and either kill, asphyxiate, or suppress vital activity. The modern antibiotic in medical use all contain the toxic amine group, and the toxic effects are seen both on the germ and on the host, when they are used therapeutically. Suspensions of consciousness, injured liver function, and other metabolic injuries, are frequently reported, and sometimes instantaneous death.

Further, the injurious effects on bacteria excite well proven mutations against the amine poisons. The survival factor in the germ has reached enough success in this combat to excite scientific envy. When the writer balanced up his observations on the reciprocal and antagonistic actions of carbonyl and amine groups and the reactions of the tissues to them, one experiment which he published in the "Journal of Laboratory and Clinical Medicine," in 1916, exposed the existence of a basis for the mutation against toxic amines. The poison observed was trimethyl melamine. It is probably one of the most toxic substances in existence, and produces *instantaneous* death when injected in fatal amounts into the blood stream. When subfatal doses are injected and the blood pressure recorded on the revolving drum, it is seen that a rapid and sustained fall in blood pressure occurs, but if more of the poison is injected during this period, in amounts that add up to more than the lethal dose, there is no fatal event, but only a minimal decrease in the blood pressure during this period of sustained depression. After the pressure returns to normal for ten minutes, a fatal dose will kill immediately. However, the occurrence of this refractory period indicated to the writer that a basis for building a resistance to amine toxins and their derivatives existed, and, to attack germs via toxic amine therapy, would ultimately turn out unsuccessful. He, therefore, decided that for the correction of the basic fault in the host, the boosting of its carbonyl function would be a preferable means of attack. To the surprise of all observers, it was found that after animals suffering with severe mastitis were given the Synthetic Survival Reagents, the most deadly

Staphylococcus Aureus lost its toxic action, and its hemolysins were no longer formed. Later on, the same was found true for the streptococcus causing dairy cattle mastitis. It is seen, in the following table, that where gangrenous conditions were caused by the infection after injury had taken place, the bacteria actually *increased in numbers while the toxic symptoms disappeared, and rapid healing was going on.* But, where no gangrenous condition was present, the infection and its changes subsided together, with rapid decrease in the number of bacteria. Thus, it is evident that the bacteria lost their pathogenicity after the host was treated with Survival Carbonyl groups, and became useful in clearing up the debris they had formerly caused. Evidently they gained also from the Survival Carbonyl chemistry and were apparently enabled to serve constructively in the Great Biological Economy again. There were special studies by the University of British Columbia Veterinary Department on the loss of hemolytic properties of bacteria after the host was treated, as well as restoration of the calcium balance showing that the tissue colloids were again in good dispersion, in other words, the tissue oxidations were restored. The following table of bacterial counts showings the reductions of bacterial counts and no change or the increase in these counts while healing and constitutional recovery is progressing is taken from the Annual Report (1944) of the Minister of Agriculture to the Parliament of British Columbia.

Name of Cow	First Bacterial Count	Second Bacterial Count
Diane	472,000	165,000
Pearl	25,000,000	95,000
Edna	2,580,000	6,000
Lily	88,000	4,000
Molly	18,000,000	25,000
No. 11	110,000	1,000
No. 10	57,000	26,000
Nancy	10,000	1,000
Polly	215,000	172,000
6351	414,000	6,000
Beauty	24,000,000	18,600,000
Flossie	5,000	4,000
Mary	2,000,000	700,000
icl-2v	23,000	2,400
Star	483,000	82,800
7601	203,000	78,000
x33140	2,000	1,100
5051	228,000	5,500
Hole	45,000	10,000
Mona	3,300,000	16,700
Marjory	10,000	35,000
No. 13	3,000	13,000
No. 14	4,000	14,000

ely-4p	20,000	12,500,000
Nigger	1,000	5,000
Jeannette	8,000	15,000
Vera	3,000	16,000

(Note — These counts were verified by a second laboratory).

It may be noted that cow No. 13 shown above is reported on as drying up with mastitis and ely-4p is shown as improving in udder condition in spite of the high bacterial content. The last-mentioned cow, Vera, had a severe accident to the udder between treatments which doubtless accounts for the increase in bacteria. However, she was making satisfactory recovery when last reported on.

Other experiments with cultures of the bacteria that caused the lesions, and their high toxicity in comparison to the loss of toxicity of cultures of the same germs planted at different periods after treatment was given to the host, confirm the observation. This work needs further chemical and clinical investigation, aside from what is reported here. This is useful in showing how antibiotic resistant gonococci and pneumonococci became nonpathogenic after contact with the synthetic survival reagent.

In line with our postulate the toxic amine groups of intestinal poisons, those present in antibiotics and those produced by fungi, tend to diminish the oxygen supply to the tissues, and thus hinder the combat against viral diseases. The toxic amine group also inactivates the FCG and blocks its function so it fails to oxidize pathogens destructively. Altogether the toxic amine tends to favor the integration of the virus with the host cell's energy producing mechanism. This can be observed practically in the following case of measles.

MEASLES

CASE No. 13 Dr. Jayme Treiger

Baby R., 18 months of age, weight 11,500 grams, was first seen by Dr. J. Treiger 12/3/59. She took sick November 17, 1959, with a terribly itchy rash that was diagnosed an allergy, for which liver extract was given until November 22, when it was seen that it did no good and the condition became worse. Then until December 1, aminophilin, streptomycin, and penicillin were given because of the high fever and aggravation of the rash, acetyl salicylic acid was also given because the pulmonary congestion was increasing. There was no improvement. The rash became worse and so did the other symptoms during these nine days. Further, the child refused all food and drink but allowed sugar water to be put on the tongue. Prostration developed and constipation also as no food was taken.

At his examination the fever was 39.5°C., the rash of measles fully fulminated, there was a bad tonsilitis, pulmonary congestion, and great prostration. It was not unconscious, but noticed nothing, and refused food and drink. Homeopathic remedies were given that improved the chest and throat symptoms,

but the rash, fever and prostration persisted with no improvement in the mental state. The prognosis was very grave. Dr. Treiger gave one microgram of one of the Survival Factor remedies, parabenzoquinone, in one cc. of water, hypodermically, on December 5th to avoid encephalitis.

Results: In a few hours there was improvement in the fever and rash. In 12 hours, the temperature was a normal 36°C. with the rash and itching about gone. The child asked for food and ate with appetite. During the rest of the day the recovery was completed, with good restoration of bowel function as well. Measles regularly recover under this treatment in 12 hours. So even here where every toxic amine group available assaulted the body chemistry, the tissues were liberated by the oxidation potential and advantage of benzoquinone, the weakest of the Survival Factor Reagents. One may contrast the therapeutic effects of toxic amines and constructive carbonyl in this typical case.

ANTIMITOTIC AGENTS

The approach offered by antimitotic agents to hit the very center of the cancer problem has been most illuminating. This work was started in the past decade, and has been increasing world wide ever since. An excellent review is given by Biesele of the Sloan Kettering Institution ("Mitotic Poisons and the Cancer Problem," Biesele — 1958 — Elsevier). Very interestingly it will be seen that the antimitotic agents present carbonyl and amine groups of the same order that we have been studying for the last 49 years. Biesele, while paying no attention to these groups or their action individually, classifies all antimitotics as poisons. It must also be noted that since the antibiotic products of moulds and soil bacteria have been studied, inspection of their structural formulae reveals similar active amine and carbonyl groups. In view of our experience the classification as poisons should be reconsidered to group them as amine poisons and carbonyl restorative or survival agents. Indeed the survival capacity is small because of the structures of the molecules, but for the service of the mould that would protect itself, these groups might have corrected the evil effects of the germ's toxins, or hindered them when being constantly produced by the mould in fair amounts. Certainly the mould did not attempt to kill the germ by the carbonyl activity. It might even have tried to correct the germ's fault and thus showed some therapeutic discretion. Such agents could really be mould hormones designed to serve within its own level of oxidations and reductions — constructively. Representative of carbonyl containing antibiotics are the substituted quinones, Phoenicin, Citrinin, Clavicin, and Spinulosin. They all offer carbonyl activated by the double bonds of two ethylene linkages, but this activation is reduced to a low ebb, by the substitutes for the hydrogen atoms that would have allowed splendid antibiotic activity. The representatives of antibiotics carrying highly toxic amine groups are the sulpha drugs, streptomycin with its two guanidin groups and all of the other synthetic products. Some of these, like terramycin and penicillin, carry carbonyl activated by conjugation with an ethylene linkage, as well as the toxic amine groups, and, in some, such groups are protected by a substituent as methyl,

that can be readily removed leaving the nascent amine group. The amine groups are likewise activated by conjugation with double bonds.

The same active groups are seen in the antimitotic agents, amine and carbonyl. Maleic acid and benzoquinone carry the carbonyl groups, and their activity is measured accurately even in such infinitesimal concentrations as one part to a million, and one part to a billion of water. It would be unreasonable to speak of such high dilutions as being toxic. So, from their own observations, these agents can not be classified as poisons to the mitotic mechanism.

In 1943, before antimitotic agents were ever studied, we testified in the Federal Court in Detroit on the corrective action of high dilutions of both substances which we had used therapeutically with corrective results years earlier. Since no evidence could be found of their injuring any of the participants or any of the processes concerned with mitosis, Biesele gives them a premitotic position, and loses the real action which is to reduce the necessity for mitosis by their protective action against injurious agents, and their rejuvenating vitality or survival boosts that lengthen the life span and thus reduce the need for reproduction or mitosis to assure the cultured tissue cell survival. The tissue culture effect is exactly what we demonstrated in animals and man even under the most adverse circumstances. That the action is attributable to the carbonyl group as activated by double bonds in conjugation, one sees in the greater activity of maleic anhydride over that of fumarate which we proved. This is a steric virtue, since in the anhydride both carbonyl groups are in the same plane with the activating double bonds, whereas in the acid only one carbonyl group has that position. This action is, of course, curative and reconstructive, in any reasonable dilution up to one part to a billion of water. As to the carbonyl group working on a physiological and corrective basis that removes the functional and mitotic failure of survival, so well illustrated by cancer, we may refer to the same court testimony, which proves the complete permanent cure of far advanced terminal cancer of the liver, accomplished by one dose of two cc. of a dilution of one part of benzoquinone to a million parts of water. The diagnosis was made by a half dozen leading surgeons by laparotomy, and the cure had in 1941 is still standing as perfect, 18 years later.

A few quotations from Biesele will be helpful, along with some explanatory discussion that we must give. This whole book of Biesele supports our thesis, in spite of its coming from outside and non-cooperative interests, that see nothing in their findings except cytolytic effects, and destructive action. Their observations are well made and accurately recorded, but they lack the physiological viewpoint that gives these interpretations scientific and therapeutic values.

On page 31 it is reported that "The threshold of mitotic inhibition of chick fibroblast cultures by benzoquinone was 10^{-9} (Meier and Schar, 1947), or 0.001 ug/ml, the same as for Colchicine." "The mitosis inhibition caused by some quinones and by maleic acid has paralleled their uptake of sulphydryl." (P. 33). This shows, in line with our thesis, that the action depends upon the activation

of the carbonyl group as recipient of electrons from its conjugated double bonds and that a physiological rather than a toxic action is to be expected, a dehydrogenating power that can initiate chain oxidation. Our diet has accordingly always eliminated sulphides and sulphydryl. Lettre's suggestions, that "quinone blocked sulphydryl compounds to inhibit mitosis," Biesele finds "inadequate as other antimitotics are effective without blocking sulphydryl action." *However, the oxidation catalysis of activated carbonyl is overlooked by all students of the problem.*

On page 30, Meyerhof and Randall (1948) and Burrough (1955) are quoted on the inhibition of epidermal mitosis by adrenaline and adrenochrome in dilutions of one part to a million. They found that "this inhibition occurred not only with glucose as an energy source, but with fructose, lactate and pyruvate . . . Further experiments indicated that the critical influence on metabolism imposed by epinephrine or adrenochrome was not on glycolysis, the tricarboxylic acid cycle, or the cytochrome system." Thus, in line with our thesis, the energy source that is cut off by toxic amines is the unidentified system which we named the preferred smokeless process initiated by activated carbonyl groups, which burns all fuels and even the inhibitors that may be present, but which can be inactivated by highly potent amine groups as of phenylenediamine and epinephrine, which we point out, show reduction potentials of E'o= +0.38v. and Eo= +0.80v. Other supportive data deserve attention.

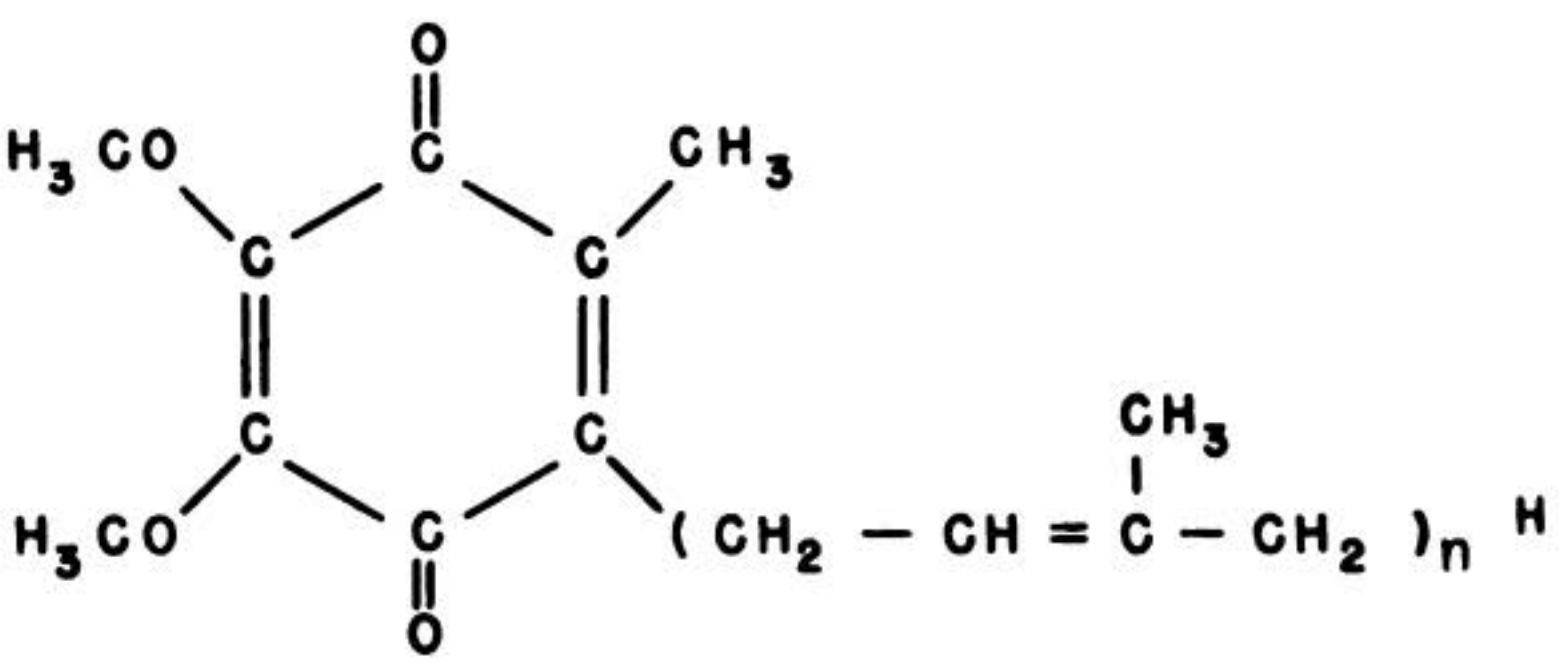
Thus on page 50, Biesele reviews the contributions of Gellhorn, Hirschberg and Kream (1952) demonstrating that "the differential susceptibility of various tumors to inhibition by 8-azaguanine was inversely related to their ability to deaminate 8-azaguanine to the non-inhibitory 8-azaxanthine. In tissue culture inhibition by 8-azaguanine and the non-inhibitory effect of 8-azaxanthine on Brown-Pearce tumor were confirmed by Flint, Hirschberg and Murray (1953)." Here, again, the inhibitory effect is due to an activated amine group while the protection against the action is a matter of oxidation that removes the amine group and substitutes a carbonyl group in its place as we claim happens to viral and other amine poisons during the reversal of carcinogenesis. A further example of this inactivation of toxic amines is found in the observation of Woodside (1953) in "tissues of the mouse in which 8-azaguanine restricted mitotic rates in carcinomas 755 and EO771 but not in tumors C954, C1300, S180 and S91, nor in ileum, jejunum or testis." It must be recalled that the ileum, jejunum and testis are rich in diamine oxidase, and here a carbonyl action is also involved in removing the toxic amine group and replacing it with a carbonyl group in line with our thesis. Here is another indication that free radicals and peroxide free radicals are involved.

On Page 50, Biesele suggests that the "inhibitory effect of 8-azaguanine may be related to its incorporation into ribonucleic acid, as suggested by Kidder, Dewey, Parks, and Woodside (1949) and demonstrated by Mitchell, Skipper, and Bennett (1950). The latter authors found, however, that the amount of 8-azaguanine incorporated into visceral RNA exceeded the amount incorporated into tumor RNA. This posed a dilemma for which Parks (1955)

saw no solution. However, 8-azaquinine-2-^{14}C was incorporated into RNA of the susceptible mouse leukemia L1210 at a level 100 times that of its incorporation into the derivative azaguanine-dependent leukemia (Bennett, Skipper and Law, 1953)." "The incorporation of 8-azaguanine into tumor RNA probably produces defective RNA. It is at this polynucleotide stage that Mandel (1955) envisioned the action of 8-azaguanine as an antimetabolite, rather than as an antagonist of free purines." Here we see in line with our thesis that normal tissues are armed with many more active carbonyl groups than are the susceptible tumor tissues and these carbonyl groups can condense with the toxic amine structures and hold them in combination instead of their oxidative destruction. So our interpretation of the induced antimitotic activities and their prevention is that the same survival factor that is dealt with in this book is concerned, and that the antimitotic data gathered so far, gives further support to our thesis with measurable proof of the efficacy of high dilutions. Though not recognized by the investigators, their data again show one phase of the interaction of amine and carbonyl groups dealt with in this book. Their data prove the protective action is an oxidation, though the whole process has not been investigated by them.

THE QUINONE STRUCTURE AS AN ELECTRON ACCEPTOR IN NORMAL TISSUE METABOLISM

Recently a fat soluble moderately substituted benzoquinone has been identified and given intensive study. It is found in all animal and some plant tissues in several modifications and has been named because of its structure and function, Coenzyme Q_{10}. It serves in the electron transport during the oxidation of succinate, and precedes cytochrome C as an obligatory electron carrier in the antimycin A sensitive oxidation of succinate by heart mitochondria. (Ziegler, 1959). Ordinarily about one gama is eliminated in the urine per hour. When a tissue on extraction with acetone and other solvents is made inactive for want of this coenzyme the addition of coenzyme Q_{10} restores the oxidative activity. (Crane, 1959). Its structure is, — where n is from 6 to 9. This isoprenoid residue gives it fat soluble properties, so it works in fatty fractions.



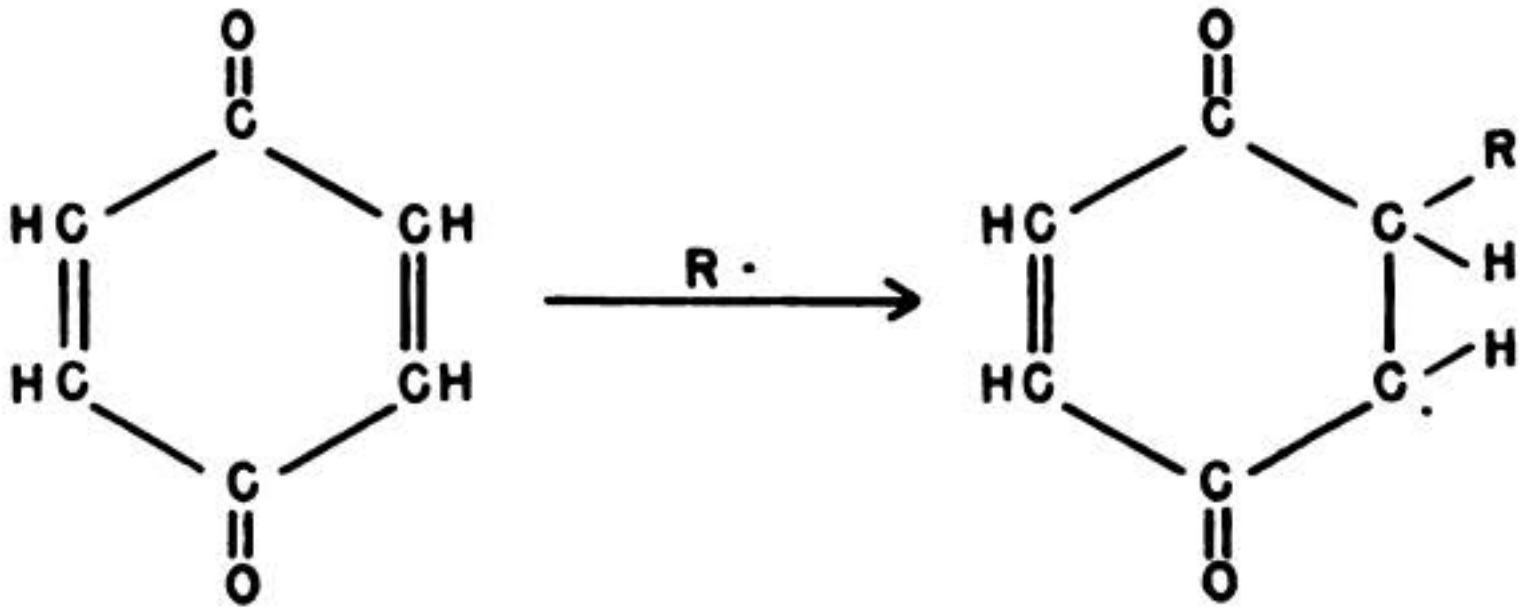
The kinetics of Coenzyme Q have been worked out by Britton Chance, and the mechanism of its reduction in mitochondria has been investigated by Ziegler, while the specificity of Coenzyme Q homologues in electron transport restorations has been studied by Crane. Thus the physiological position of the quinone structure in metabolism is amply assured, and measured during the past year. This recent work confirms our postulate, the chemistry of which we put into practical action some decades ago.

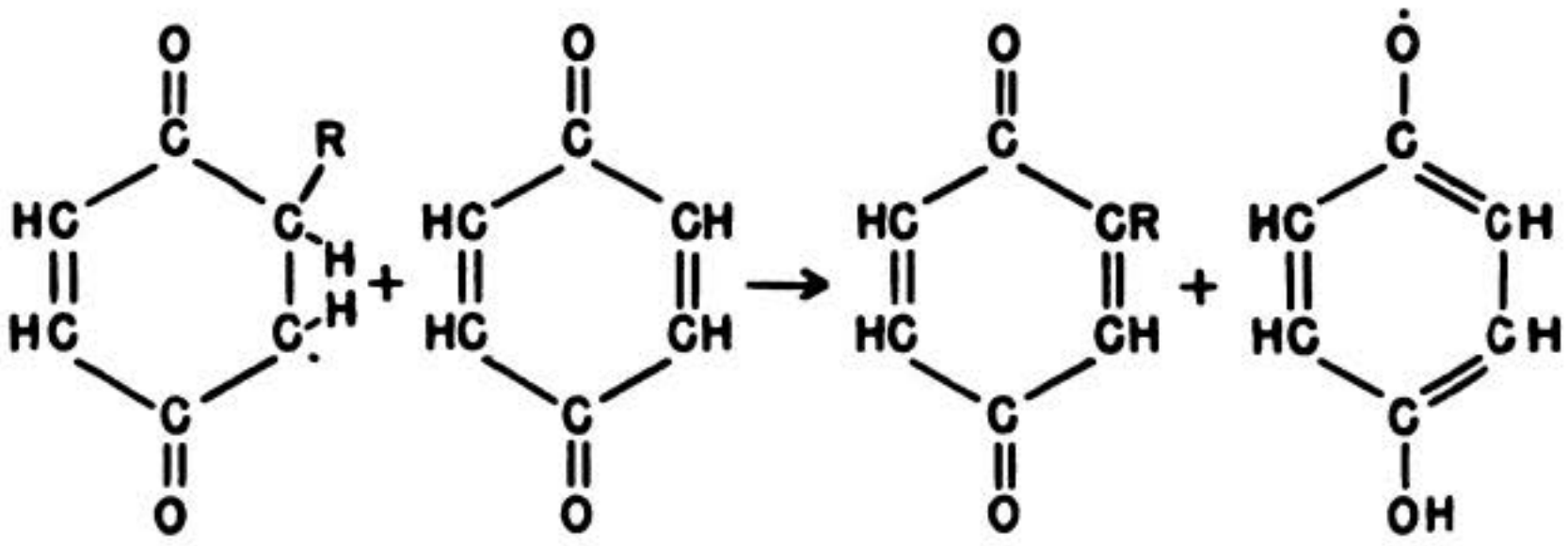
CHAPTER X

RECENT PHARMACEUTICAL STRIDES

Very recently, in the last few years, some highly substituted benzoquinones have been introduced into medicine, as cytolytic agents in the treatment of cancer. The high substitution defeats any action, inherent to the quinone structure, that might be curative. Such action depends upon high activation of the carbonyl group, and upon the wealth of resonance hybrids that would be possible if the hydrogen atoms, held by the ethylenic linkage, were present, instead of displaced by complex groups that not only prevent electron migrations to the carbonyl group, but prevent additions of free radicals to the carbon atoms of the ethylene groups. In certain instances, the benefit of benzoquinone is due to its ability to absorb antagonistic free radicals, and the high substitutions annul this effect. The following well recognized reactions may be offered as a demonstration of this detoxicating quality, which is lost in the admitted non-curative commercial drugs.

Well established free radical absorbing
power of benzoquinone . . .





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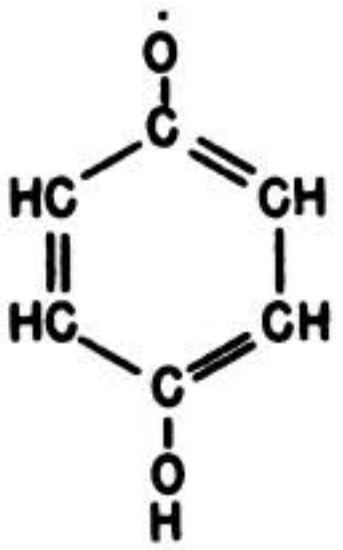
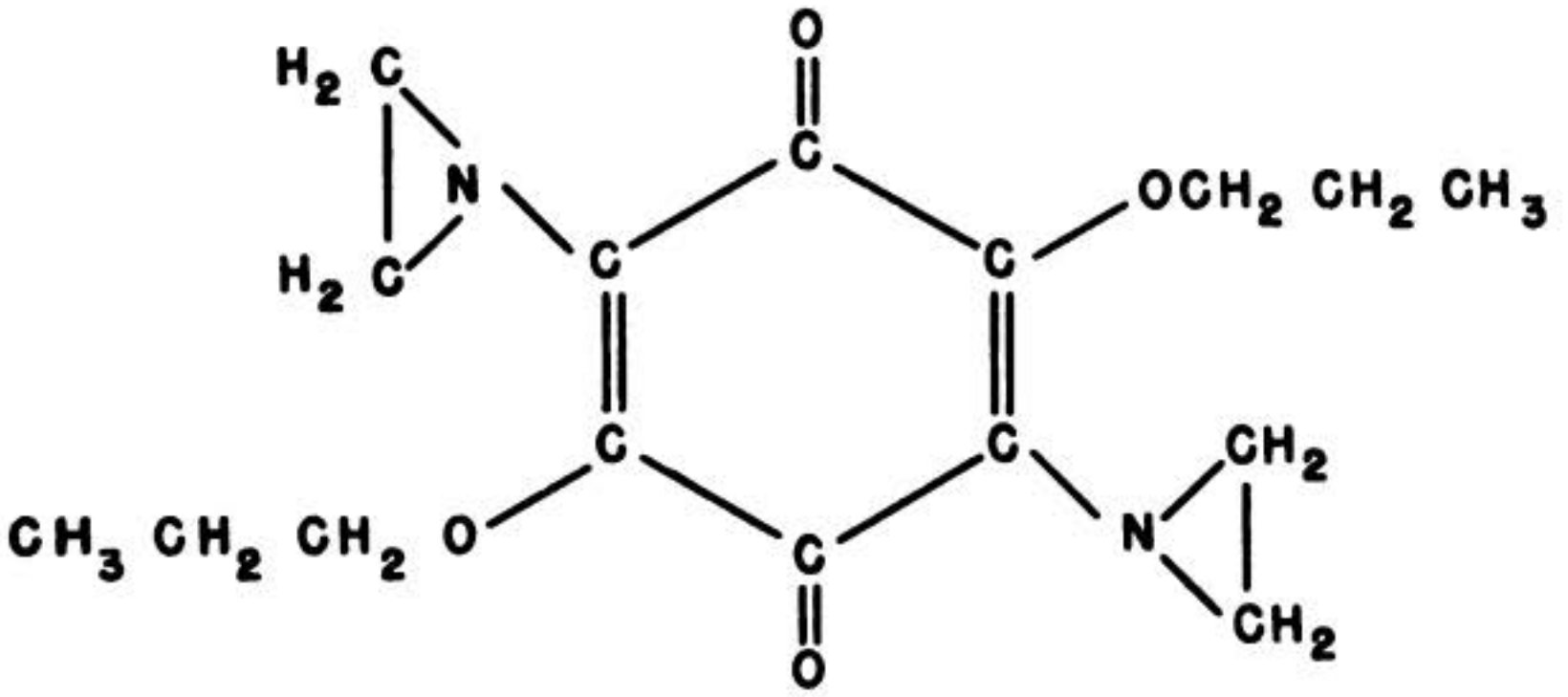


. . . yields the quinhydrone, which disproportionates to hydroquinone and benzoquinone. Thus: free radicals are inactivated by benzoquinone and the therapeutic action is hindered as clinical tests have proven. We therefore conclude that the therapeutic activity of these reagents is partly at least to be attributed to the free radicals they contained, and which were responsible for the oxidation and polymerizations in progress in these systems. There is nothing cytolytic about this structure. It is constructive in action.

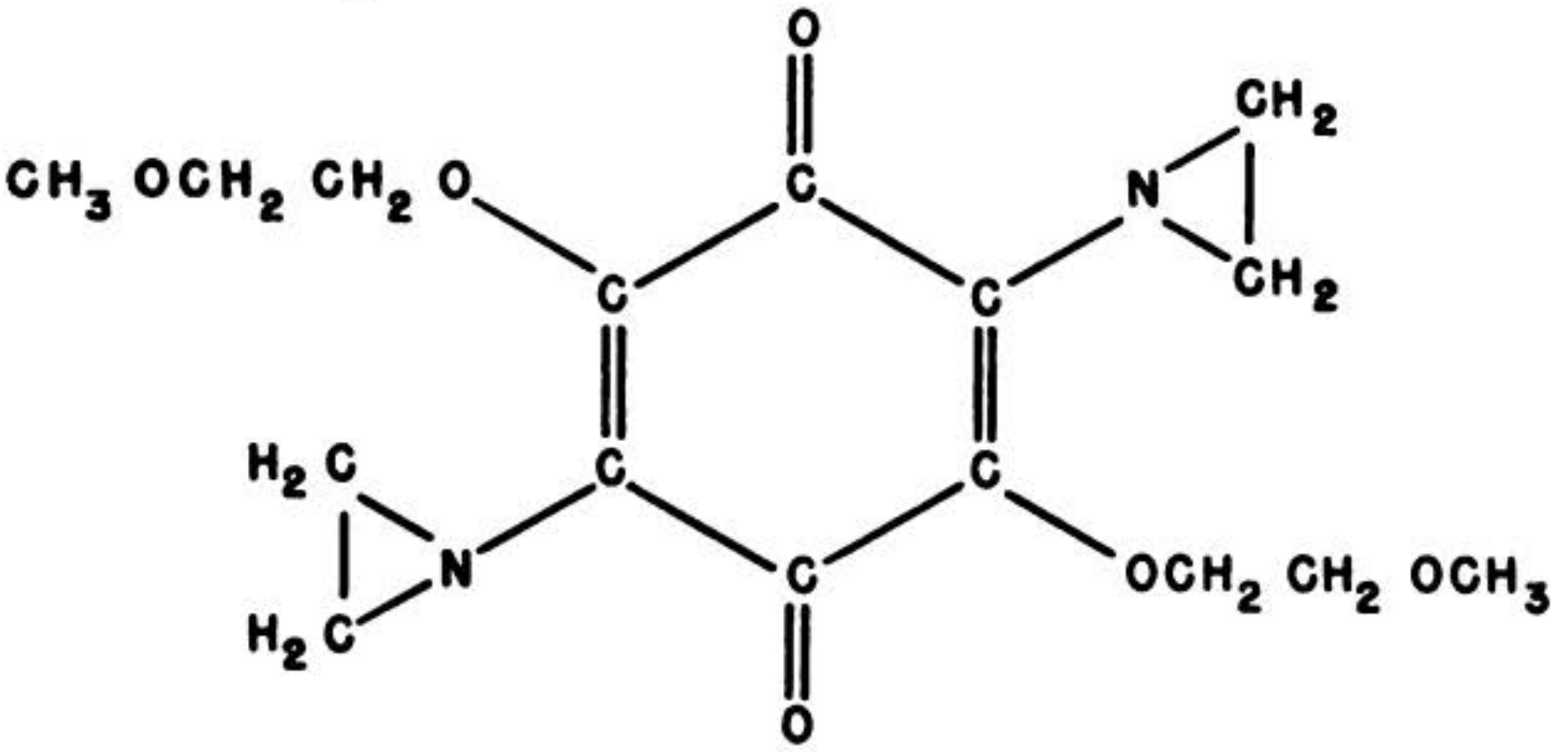
Pharmaceutical Substituted Quinones

As examples of these cytolytic agents that are admittedly unable to protect or to cure, the following of Schenley Laboratory may be taken as an example:



It carries toxic nitrogen, -2,5-di-n-propxy-3,6-bis-ethylenimine-1,4-benzo-quinone.

And the following A-139 is of the same order:



Actinomycin is a natural nitrogen free antibiotic that has had some trial but without any curative results. Whatever they are, they might be a little better than that of the preceding or Bayer's E39, which is built on the same order as Schenley's and A-139. In Actinomycin, carbonyl activity is limited by the substituents, and by the failure to have even one hydrogen atom attached to an ethylene linkage. This again is for the benefit of the organism that produces it and not to serve as a therapeutic agent.

Certainly the analytical and synthetic work of the antibiotic chemist deserves the highest praise. The search for virtue in complexity of structure, however, has no basis aside from the needs of the organism that produces the antibiotic. These are not identical with the requirements of higher animals in the situations we have discussed. Rather the greatest simplicity possible wins the laurels here.

CHAPTER XI

THE AZOMETHINE DOUBLE BOND

The azomethine condensation, like the free radical and the chain oxidation to which the latter is related, have not found much utility in past biochemistry except as we have employed them in our postulate. If the criticism is offered that we are giving them too much speculation, the answer comes right back, that we are postulating processes in matters that were not understood before, nor mastered until these hypothess were applied. The azomethine double bond is present in many vitally important molecules. Thiamine has it three times, adenine three times, flavine twice, pyridoxal once, cytochrome twice, porphine twice, cystosine twice, vitamin B_1 four times, and it is found in many others and their derivatives. In view of its wide occurrence and great versatility it warrants thorough study. We have used it as a first step in the process of energy transfer and its block in reactions that belong to the high efficiency system oxidations. Recently, however, it is being written in the preliminary steps to certain hydrolytic reactions as where the carbonyl group of Pyridoxal phosphate condenses with an amine group to co-factor certain transaminations and the decarboxylations of amino acids. (Snell and Metzler, 1952). Here the azomethine double bond is hydrolized to reproduce an amine group and a carbonyl group, whereas we use it in preparation for the oxidative destruction of the amine group and the restoration of carbonyl. We regard it as a bridge for energy transfer and as an activator of dehydrogenations that lead to peroxide free radical formation and oxidative amine destruction. We also use it in preparation for the Amadori reaction as a first step in sugar oxidation. It impressed one as significant that guanidin and methyl guanidin contained it and so we supposed it played a part in the oxidative cleavage that yielded urea and in the synthetic reactions as where guanidin was built into guanin. So we noted the possibility that the parathyroid glands might have to do with the formation and destruction of this bond. Whether this supposition is correct or not further investigation will open up a big field of information.

Glucose is more difficult to oxidize than fructose, and the consensus is now beginning to shift to the idea that all glucose is converted into fructose before it is burned. Let us assume that the functional mechanism where sugar is attached to be burned, presents an activated amine group as in creatine, adenine, etc., much the same way as it has the highly active FCG. This amine would condense with the carbonyl group of fructose to form an azomethine double bond, which would undergo an Amadori rearrangement, placing the double bond between carbon atoms C_2 and C_3. This would mobilize the hydrogen atom attached to C_4 and subject it to easy removal with production of a free radical, that would add molecular oxygen to become a peroxide free radical. This would cause cleavage of the molecule into ketoglyceric acid and

glyceric acid, with reversal of the Amadori reaction and regeneration of the functional mechanism's amine group. If plenty of oxygen were at hand, there would be no interruption, simultaneous further dehydrogenations would produce more peroxide free radicals that would break the molecules down into carbon dioxide and water. If oxygen failed, the reduction chemistry in dominance would change the glyceric acids to lactic acid. These would remain until oxygen came along. This, of course, is not the fermentation process, and in the presence of oxygen no intermediaries would be trapped. Oxygen would be the electron acceptor right on the job. One finding regarding the effects of parathyroidectomy gave clues that led to a successful attack on the cancer and virus problems, with ramifications touching on the most pressing questions in the basic medicine of today. These orient us with regard to a least common denominator in disease production.

The recent support to our postulate offered by the structures and actions of antimitotic and antibiotic agents is strengthened by the phenomena and structure of the prosthetic group of Diamine Oxidase, as revealed by Zeller in 1951 ("The Enzymes" — edited by Sumner and Myrback — Vol. II, Part I, page 554 — 1951 Edition). Here Zeller shows that Diamine Oxidase removes one amine group from the toxic diamines by an oxidation process started with the dehydrogenating action of a proven carbonyl group. The action is thus of the order of our postulated FCG, and its range of action is similar, since it acts on loosely combining amines, but is inactivated by the firmly combining guanidins. Thus, it detoxicates putrescine, cadaverine, agmatin, etc. . . . but is inactivated by condensing with guanidin, just as the postulated FCG is inactivated. This condensation blocks the oxidations and causes failure of function and then death, in line with our postulate. For this reason, Diamine Oxidase is produced in greatest quantities in the placenta, the intestinal mucosa, and the liver. It is found in the blood of pregnant women, and, when its content falls, death of the foetus threatens. Its production in the intestinal wall protects the muscle and secreting glands of the mucous membrane from block in energy production for function. This is particularly necessary because the diamines are produced within the intestine by the decarboxylation of amino acids by bacteria, especially in an acid reacting (pH 3.5 - 6) colon. It is produced especially in the liver, too, to protect the tissues in general from toxic diamines entering the portal circulation. The action of the diamines is of the same order as those of virus, carcinogenic amines, guanidin, and other toxic amines of our postulate. Their detoxication is likewise initiated clearly by the dehydrogenating action of the carbonyl group, — oxidation. However, Zeller does not go far enough to identify free radical and peroxide free radical action, which would be the normal chemical sequel to dehydrogenation. He rather lets the enzyme conception carry the burden of explanation.

He also assumes the enzymatic activation of the two hydrogen atoms attached to the carbon atoms alpha and beta to an amine group, and that these are the hydrogen atoms that are removed by the enzymatic carbonyl group. He assumes that a double bond is thus produced between these carbon atoms, and that this double bond is shifted to the amine group to form an imide group

which is then hydrolyzed off as ammonia. Thus a carbonyl group takes the place of the amine group, and the detoxication is accomplished. Prof. Zeller gives no thought to a condensation between the carbonyl group of the enzyme and the amine group of the toxin to form an azomethine double bond. Nor does he refer to the oxidation we have postulated that replaces the amine group of the toxin with a carbonyl group. However, the products of the reaction are ammonia and hydrogen peroxide, as would be expected from a hydrolysis in an enzyme system that operates in solution instead of on the grana surfaces, where oxidations would be expected, as in the case of the FCG system. This difference in position determines the reaction mechanism.

Diamine oxidase is inactivated by various guanidines and diguanidines, imidazole, such dyes as pyocyanine, methylene blue, streptomycin, pyridoxamine, by cyanide, and by the carbonyl reagents, semi-carbazide, hydroxylamine, phenylhydrazine, etc. The enzymatic carbonyl group does form an azomethine bond with the latter, the carbonyl reagents, and with guanidin in agreement with our postulate, so it can also form the same azomethine bond with the other amines except as the direction of the reaction is determined enzymatically. Such enzymatic forces, however, are not able to prevent an inactivating condensation of the carbonyl group with guanidin, phenylhydrazine, hydroxylamine, etc., which take place outside the physiological range of control, just as they can inactivate the FCG. Both inhibited systems, that of the grana FCG, and of the enzymatic Diamine Oxidase carbonyl group, require the oxidative rescue via a high O/R potential Survival reagent, which indicates that they are both inhibited in the same way by a firm azomethine condensation. This will be demonstrated in the case histories.

CHAPTER XII

GENERAL ASPECTS OF THE REAGENTS

The structure of the Survival Reagents is designed to carry out the chemical acts that are desired, and these are certain specific dehydrogenations to initiate oxidation progressions in pathogens integrated with host cell functional mechanisms. The atomic groups of these functional mechanisms are their functional carbonyl groups and supporting ethylenic linkages that activate them and with which they are conjugated. Two types of pathogen host cell unions were described, one via an azomethine condensation of the FCG and a tightly binding amine group, and the other via a free radical addition to one pole of the activating double bond. Anoxia or hypoxia was a deciding factor toward pathogenesis and molecular oxygen plus an adequate dehydrogenator were the essentials for the restoration of normal.

Necessarily the reagents are highly reactive, with energy content reaching the "bursting point" so to speak. Nevertheless their chemistry is not crude mass action chemistry, but instead, catalytic in the sense that they initiate reactions that convert amounts of material far out of proportion to the amount of reagent used. High dilutions are necessary for their best action. Activation also depends upon the high dilution, wide molecular and group dispersion in addition to the activation of the specific groups that do the work. High dilution probably aids the electron migrations toward these groups that endow them with high activity. It is also characteristic that in nature the activating molecules or structures similar to the reagents we offer, also work best in high dilution. This is true of Echinochrome A, Crocin and many other substances. (See Chapter XIV).

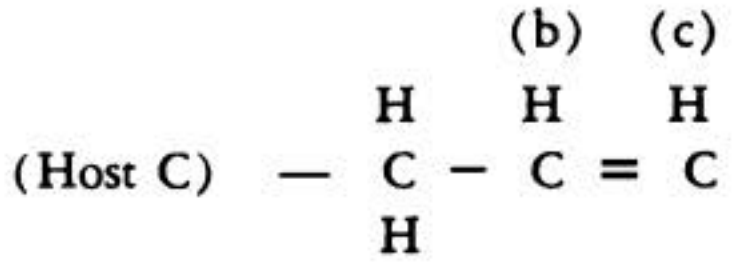
Highly reactive substances used in high dilution, require the greatest care in production and the higher the dilution, the greater is the care needed. Likewise in the management of any case that is treated, precautions against inactivation by contaminating influences or injuries from exposing the reagent to strong light or even mild heat must be avoided. Contact of the reagent with foreign substances may cause the expenditure of its energy so that it is inactivated when it reaches the blood stream. Clean needles and syringes, and clean air are essential, and these must be FREE from even the slightest traces of contamination.

The physician's eyes must be "wide open," too, so he knows for sure that the regime that is selected for the patient to follow is entirely harmonious with its chemistry. This means the diet must be chosen scientifically, and not by guesswork, and other hygienic methods and measures must be helpful. Every patient is a complex of many factors in this complicated world, and the environment must be studied so the patient is protected from contrary influences both mental and chemical. The principle of the therapy is simple, but its management is a job requiring expertness, intuition and patience plus a large measure of devotion.

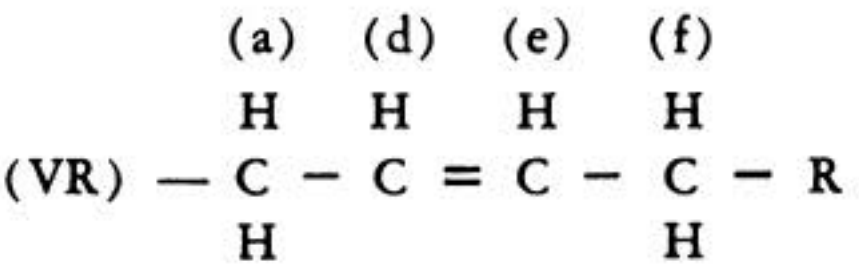
CHAPTER XIII

CLEAVAGE OF THE HOST-CELL PATHOGEN INTEGRATION

Let the host cell and its functional carbonyl group — FCG — and the activating ethylene linkage be represented by



and the virus by



or by a chain of its polymeres.

Whether dehydrogenation takes place at the point of integration of the pathogen and the FCG system, or at the farthest end to begin the destructive oxidation of the pathogen, depends on its structure. At any rate, the most exposed and most active hydrogen atom would be attacked. In the virus pathogen this would be a terminal unit of the virus, since viruses are built up as by a polymerization process, that is: by the addition of a free radical of each unit to a pole of a double bond or free radical of the other unit already laid down. Therefore, the last added unit would be most exposed and dehydrogenation would take place at the carbon atom alpha to a double bond in this outer unit as at (f) or (a). A free radical so produced would add molecular oxygen to make a peroxide free radical, and this would cleave the molecule into two parts, each of which would carry a carbonyl terminal. The double bonds of this carbonyl group would activate the hydrogen on the carbon atom alpha thereto and dehydrogenation would lead to another cleavage producing two more carbonyl groups, etc. Thus, step by step, the virus would be burned off until the ethylene linkage to which it was added was reached, as at (c), and here a carbonyl group would be made, thus activating the FCG in place of the ethylene linkage. This would prove an immunizing help as the carbonyl group

is a much better electron donor than the ethylene linkage, and thus a higher O/R potential would be gained by the FCG. Further, carbonyl groups do not add free radicals as readily as do ethylene linkages, and the chances for integration with a pathogen during anoxia would be correspondingly reduced. The long lasting protection observed in our cured patients may thus be explained. The energy liberated in this combustion of the virus would pass on to the host cell and support its reconstruction. See Appendix.

The virus attached via an azomethine double bond by condensation of its amine group with the FCG could undergo the same stepwise oxidation with restoration of the original carbonyl group of the FCG System, and leaving its ethylene linkage undisturbed as the donor of electrons to the FCG. However, alpha to the azomethine double bond a hydrogen atom could be removed and the oxidation at this point would burn off the amine group and restore the FCG. The virus would thus lose its pathogenic amine group and receive a carbonyl group to change its whole attitude toward the world, as it was separated off as a whole. Likewise the virus attached at the ethylene linkage could be burned off at the point of attachment and leave a similarly restored FCG activated by electrons from a carbonyl group. The virus would also acquire a new carbonyl group to change its behaviors. It is even possible that the acquiring of an active carbonyl group would give it autonomous properties so it need no longer be parasitic to obtain its energy, but may be able to produce it itself.

Should the pathogen be a synthetic carcinogen or a polymerized toxin produced by some germ trapped in a scar where oxygen supply is low, the burning would start at the most active hydrogen atom exposed: and that would be one that is alpha to a double bond located in the "K region", as is now identified by cancerologists. At any rate, the pathogen is no longer to be found. It, therefore, is no longer a disturber of physiological processes. Normalcy is thus established.

Toxins attached to fibroblastic tissue where healing is going on, and held in the scar as an integrate, no doubt make the addition as a free radical or via an amine group as described above. It is a clinical fact that scars disappear after the Survival Factor dehydrogenator starts to work on them. The toxin is thus burned out of the way and the fibrosis has no more incentive to exist. It becomes obsolete, and is absorbed. Extreme arterial sclerosis in very old people has been observed to disappear and senile dementia to clear up in a major way following one dose of the Survival Reagent. Under such circumstances one is justified in assuming that the pathogen was burned out of the sclerotic tissue and the fibrosis could then be absorbed, without any reason for hindrance.

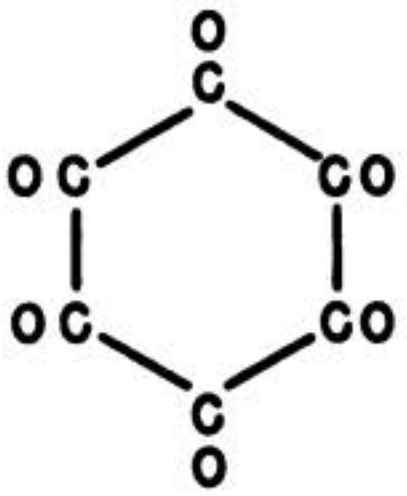
The ideal therapeutic reagent must possess an adequate O/R potential in a molecule free from steric hindrance, and of the simplest possible structure, so that one action and only one predominates. It is necessary to gain as broad a field of steric advantage as possible since the steric qualities of the host cell functional mechanism pathogen integrate changes some with each different

pathogen. Simplicity in structure is therefore an advantage. The Survival Reagent's carbonyl group as so often stated is activated by conjugation with the double bonds of an ethylene linkage, or of another carbonyl group, or with the triple bonds of an acetylene linkage. The greater the number of ethylenic groups that carry free hydrogen atoms the greater is the O/R potential of the carbonyl group. Substitution of these hydrogen atoms must not be permitted. This is seen in the following quinone structures:

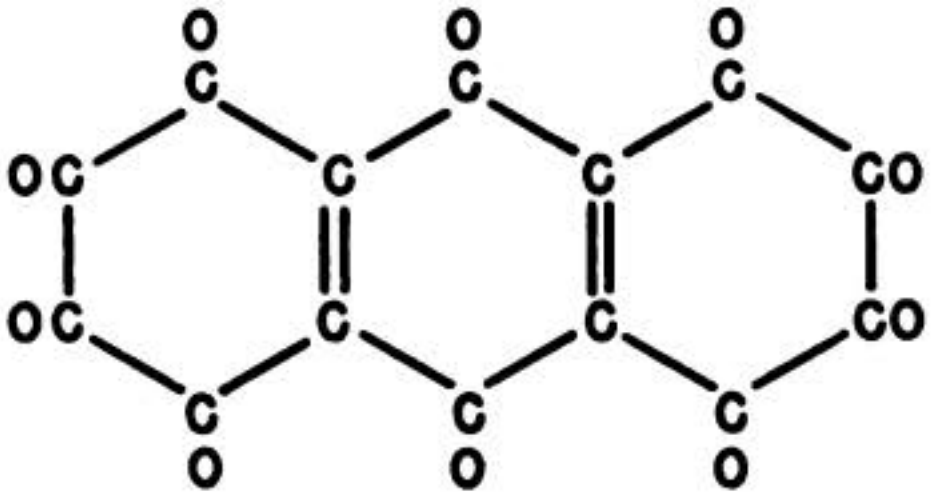
Anthraquinone with no quinone double bonds carrying hydrogen atoms and with two carbonyl groups shows an O/R potential of 0.154 v. Alpha-naphtha-quinone with one double bond carrying two hydrogen atoms and with two carbonyls has an O/R potential of 0.484 v. Beta-Naphtha-quinone with one double bond carrying two hydrogen atoms, and the double bond of a carbonyl group conjugated directly with another carbonyl group of the quinone structure has an O/R potential of 0.576 v. Parabenzoquinone carries two ethylene linkages presenting four hydrogen atoms and two carbonyl groups. It has an O/R potential of 0.715 v. Orthobenzoquinone with two sets of double bonds carrying four hydrogen atoms and two carbonyl groups directly conjugated, offers an O/R potential of 0.792 v. Diphenoquinone with its four ethylene linkages presenting eight hydrogen atoms in two quinone groups united by a double bond, and offering two carbonyl groups, shows an O/R potential of 0.954 v. As the dehydrogenating power increases with the O/R potential and likewise the energy content of the molecule, one will see to it that no substitutions are allowed unless the substituent offers a series of additional ethylene linkages in conjugation. Yet that may interpose some steric disadvantage. As the migration of electrons to the carbonyl group make the ethylenic linkages more electrophylic they will tend to add free radicals of toxins more readily, and when hypoxia hinders peroxide free radical formation in the dehydrogenated toxin, this property of the ethylenic linkage is an advantage as it will protect the Functional Carbonyl group activator unsaturated bonds from paralyzing additions. On the other hand, the quinone ethylenic linkages when present in excess, can add to and inactivate the free radicals produced in the pathogen that is undergoing destructive oxidation, and thus block further progress in its destruction and also block the liberation of the host cell FCG. Too large a dose of quinone, especially of diphenoquinone, or its repetition when recovery is going on, competes with molecular oxygen and can block the recovery process or even reverse it. This is especially true when hypoxia reduces the opportunity to change the free radicals formed in the toxin into peroxide free radicals. On the other hand, some advantage may be had in protecting host cell unsaturated bonds from adding free radicals of the pathogen. The structure of the reagent, therefore, must be understood for best use.

Another valuable consequence of not allowing the quinone structure to carry substituents of the hydrogen atoms, is that these substituents cut down the activity and formation of the resonance hybrid free radicals that have great value in starting oxidation chains, and continue the dehydrogenations after the Carbonyl group has accepted a hydrogen atom.

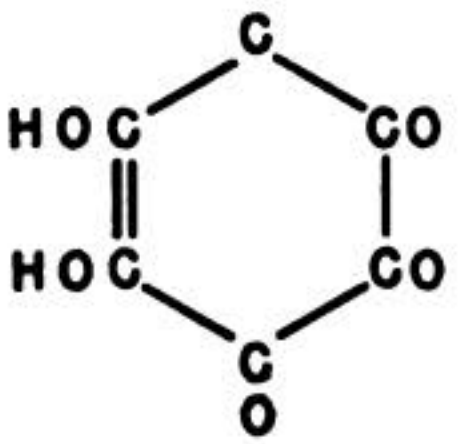
Structural Formulae of Reagents



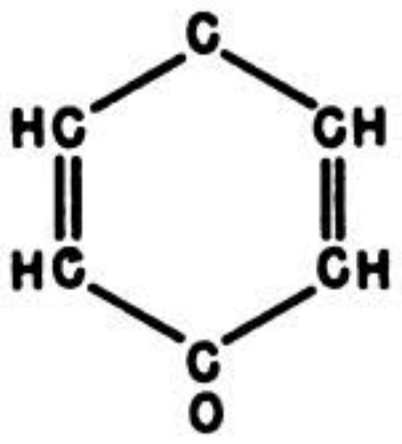
Triquinoyl



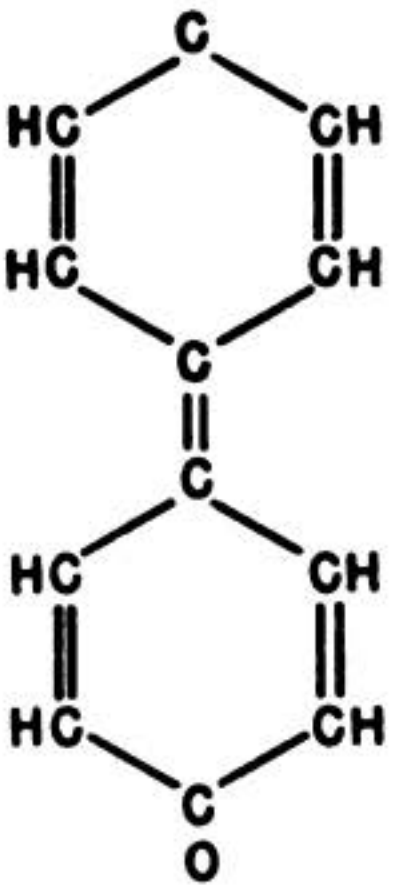
Compound (C)



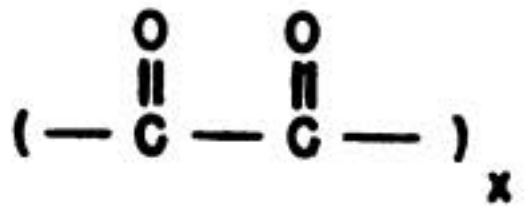
Rhodozonic Acid



Benzoquinone



Diphenoquinone



Serial System of Carbonyl groups
with free radical terminals

The disadvantages of even the best quinone structures called for molecular set-up where crippling conditions were eliminated and so the chains of carbonyl groups had to be developed. Among them, the following structures were compared with the straight chains of carbonyl groups represented by the formula: (— C — C —)x, developed in various molecular weights.
‖ ‖
O O

The last mentioned structure offers great versatility because of different molecular weights that can be produced for fairly specific selection.

CHAPTER XIV

CATALYTIC DILUTIONS

The philosophy of the dominant therapy has been, up to now, to saturate the patient with as much destructive agent as possible, so as to destroy the enemy invader without too much injury to the patient. Quinine and atabrine in Malaria, and the present-day antibiotics are examples. So, safe concentration is the logical procedure under this system. This is pharmacology. On the other hand, the physiological approach has different aims. It is corrective and restorative, and induces the changes it wants. Then, the question becomes what are the physiological ranges of concentration in which the remedy shows best action by actual measurements and careful observation. The question not only is how dilute must the solution of the reagent be, but also how concentrated dare it be and still retain physiological activity. A look at the physiological natural reagents that carry activated carbonyl groups will show how high nature dilutes them for best action. As a starter, no better guide could be chosen. Here, again, we see that our postulate of nearly half a century of application is just recently receiving support from the physiological processes of Nature herself.

A dilution of one part of a reagent to a trillion of water carries thousands of billions of molecules in each cubic centimeter. Since only one molecule is required to start a catalytic action that can grow with geometric progression, nature is not stingy when she supplies vitamin B_{12} in quantities so small that they can act normally in concentrations of 1×10^{-9} or one part to a billion. Only three micrograms is an effective therapeutic dose. Only 0.01 millimicrogram gives definite growth response to bacteria. (U.S. Pharmacopeia Vitamine B_{12} Study Panel). Echinochrome A, is a naphthaquinone without one double bond carrying a free hydrogen atom. Its O/R potential is only 0.080 v. This is because the molecule has five hydroxyl substituents where hydrogen instead would have raised the potential 0.3 v. higher. Still it is active in inducing motion in Sea Urchin eggs in dilutions of $1:2 \times 10^{-9}$ (Fieser, 1944 Edition, page 753). Crocin belongs to the carotenoid family and presents two terminal carbonyl groups conjugated with ethylene linkages, a chain of seven, with four methyl substituents. The carbonyl groups are part of the terminal carboxyl groups that have each been esterified with gentiobiose. It is active in the highest dilutions as demonstrated by Kuhn and Kuhn and Moewus — (1938-1940). Only one molecule is required to determine sex activity in the C. augamentos F. simplex and Chlamydomonas genus of algae. Thus the dilutions may be higher than one part to a trillion of water, and Prof. Gilbert Smith, in 1947, reported the production of motility in certain plant sex cells by use of only one part of this reagent to 250,000,000,000 parts of water. Heparin

is active in suspending blood coagulation in amounts so minute that there is no method to detect its presence (Fieser — p. 487 — 1944 Edition), and acetyl choline (Karrer — p. 239 — 1947 Edition), is able to renew suspended contractions in the intestinal muscle of the surviving guinea pigs in a dilution of one part to 1,000,000,000 parts of water. Since biological reactions are so much more refined than laboratory manipulations, one can not judge physiological activity by chemical detection or measurements. Likewise, clinical results are not to be predicted by the swing of a chemical balance. One thing must be observed, however, and that is when high dilutions are used, freedom from interfering reagents or measures are imperative, and the work must be more careful and scientific than when crude work is being done on a mass-action scale. The chemistry of the free radical and mesomeric electron displacements will give biochemistry a different hue from the conceptions of the pharmacology laboratory that have controlled up to this time. The dosage and dilution are matters of expert decision, based on experience and enlightenment, not upon prejudice or superstition.

Following the 1955 and 1958 editions of this book, some eminent biochemists such as Szent Gyorgyi, have expressed opinions supportive to our basic philosophy with regard to the position of the free radical (lone electron) in carcinogenesis and other biochemical fields. This is indeed a gratifying advance over the dominant approach, and its correctness has already been established for decades as our text demonstrates.

See Appendix III, for further confirmation of the double bond and free radical phenomena concerned in pathogenesis and its correction.

CHAPTER XV

TERMINATION OF THE MALIGNANT PHASE RESTORATION OF THE FUNCTIONAL CARBONYL GROUP

We have already explained the two activated carbonyl groups of our postulate: one that initiates the oxidation progression and transfers the energy produced to carrier phosphate bonds. The other receives the energy from these bonds, and passes it on to the working mechanism of the cell, the contractile, the conductile, the secreting, or the mitotic fibrillae and units. They can both be blocked in their functions, by inactivation of their activating ethylenic linkages, or by a firm condensation with an amine group of some pathogen. When this takes place energy does not pass to the functional unit by the normal controlled route and only fermentation or Krebs Cycle energy is produced to pass directly into the working unit. Thus excessive contraction as the spasms of bronchial asthma, or excessive secretion as the secretion of hay fever, or excessive synapse closure as in some phase of insanity, a compulsion neurosis, etc., or impulse generation as of tic doulereau, or excessive uncontrolled mitosis as of neoplasia will have to be the result in the tissue that is affected. The functional fault is demonstrated in the uncontrolled nature of the act, and all may be classified as allergies (Koch, Natural Immunity, 1934, and Cancer and Its Allied Diseases, 1929, where we attributed the abnormal energy transfer to a photosensitization process).

The correction is, of course, the restoration of the normal FCG activity in both capacities. That this can be accomplished no matter what the allergy happens to be is illustrated further in this section. Many more cases could be given in all categories, especially in the common allergies where the cure rate ran over 80% in the court testimony, but what is given is enough to illustrate.

The speed of the recovery is shown very well in the allergies of the respiratory system, and in the cure of the compulsion neurosis case, but it is also seen in the neoplastic category, too, as is illustrated immediately by biopsy tests or in the long run by the surgical tests outlined farther on. It is to be emphasized that the cure or correction of the disease in any instance is the restoration of the normal FCG function and the cleaning out of the debris and restoration of the normal structure and function, as the case requires. Then the normal being established, it can be broken down perhaps again when circumstances force this change. There may be some immunity or increased resistance due to a carbonyl substitution for the ethylenic activation of the FCG, at any rate, though many cases stay well after being cured of cancer for several decades, there is no absolutely permanent and impregnable protection, and an

understanding of the pathogenesis and what food diet means, is the best assurance of maintaining health after recovery.

There are some forms of primary cancer of the liver that are grossly pathognomonic. In such cases the expert surgeon can make a firm diagnosis without the biopsy, and he does so to protect his patient from embolism and hemorrhage incident thereto. This is not a matter of neglect, but of good judgment, and usually practiced. The type of greatest interest is that of diffuse distribution of miriads of small lesions over the surface and between the somewhat larger nodules that press up from underneath. The "feel" is also characteristic. In such cases there is deep jaundice because the smaller bile ducts are compressed or blocked by tumor tissue. The biopsy is characteristic also so that one can tell what the microscopic picture is from the gross features. In the diffuse type the small surface nodules are much of the same size as if originating simultaneously, and thus speaking for a multiple origin of the carcinosis, a generalized equal distribution of the pathogen. As these nodules are smaller than many inside the liver, it is evident that the inner ones had an earlier start. Likewise, often, one lobe of the liver is more affected, especially the right lobe. Thus, in the beginning of the disease, the amount of pathogen is not so great that it makes an attack throughout all at one time. It is used up in one locality. However, at the terminal stage the miriads of small nodules equally and independently distributed speak for a swamping of the system with the carcinogen as if there were more toxin present than liver cells to combine with. Such a case will be described, and also a baby with a massive primary cancer of the liver proven by biopsy. This case is well described in the mother's affidavit from which several points are reproduced. The series of photographs are also instructive. This information was sent us by Dr. M----, our collaborator, who had charge of the case, with his notes. This patient was not seen by the writer:

PRIMARY CANCER OF THE LIVER

CASE No. 14

Judy McW., three months old. "After a normal birth, Judy, before the age of six weeks, showed signs of illness. Her abdomen was enlarged, she was restless, and her face did not show the repose of a healthy baby. Her physician . . . could not find anything wrong with her until his check-up and examination at the end of her eighth week. At that time the doctor found her abdomen hard and much distended. During the period from August 20, 1948, to August 27, 1948, a tentative diagnosis of cancer was made and X-rays were given although the X-ray technician stated it was hopeless to expect a recovery."

"By the time Judy was three months old the attending physician and another surgeon made an exploratory operation on Judy's abdomen at which time a biopsy was taken. The physicians reported to us that the biopsy showed a high degree of malignancy which involved 85% of the child's liver. They told

us that there was nothing that could be done to save Judy's life; that we should take her home and make her as comfortable as possible for the few days she could live."

"Her life expectancy was placed at 21 days. We were told not to remove the bandage from her abdomen lest the stitches burst out. It was the doctor's opinion that the incision in her abdomen would not heal."

"Dr. Koch's therapy was given by Dr. N. T. M-----, of Cisco, Texas. The dose was injected into Judy's hip on September 18, 1948. At this time and during the course of Judy's recovery, Mr. N----- took a series of color pictures showing her progress. Previously he had taken two pictures at six weeks of age and before the diagnosis of cancer. The series of pictures gives a good idea of her case.

"At the time the injection was given, Judy's abdomen was so much enlarged that she could hardly breathe due to upward pressure on her lungs. The circulation on the surface had greatly increased and she had a bluish cast from a diffusion of blood in and just under the skin. Veins under the skin of the abdomen were plainly visible. The abdomen was very firm, even hard. At the time the Koch's treatment was given, Dr. M----- expressed no hope of securing a recovery as he thought the case was too far advanced.

"Within ten days after the treatment Judy showed definite reactions which raised our hopes. Shortly, she began to pass large quantities of mucous with bowel movements. She also passed a large amount of water in the normal manner, sometimes requiring as many as twenty diaper changes per day. No medication was used after the injection of the Koch treatment, and only minor changes were made in the baby's diet. Apple juice was substituted for orange juice, and Judy liked it. After treatment was given and until recovery was practically complete, only one doctor saw Judy. That was a doctor residing at Azle, Texas, who removed the stitches from the healed incision about the middle of October, 1948.

"Soon Judy began to gain weight and her abdomen rapidly reduced in size and became more soft and pliant so that she could breathe better. The hard growth receded toward the lower right side. By December 25, 1948, she had a healthy and normal appearance as the pictures mentioned before show, but some traces of the growth remained.

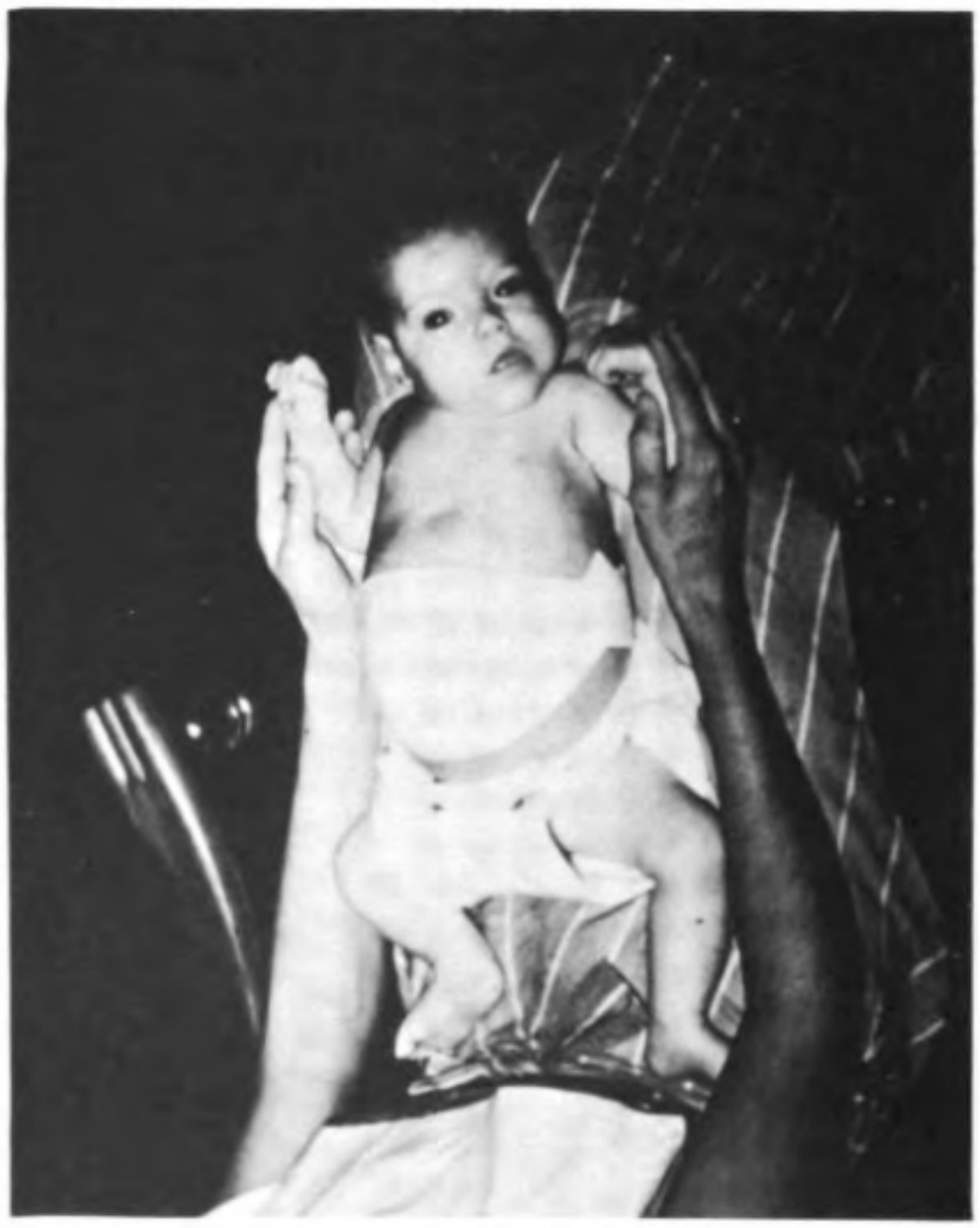
"Later, about May 12, 1949, I had her examined by a doctor in Paris, Texas. He could find nothing, after which he was told of the baby's former illness and he could still find no trouble.

"On November 11, 1949, Judy and her mother appeared before a group of physicians and surgeons especially interested in cancer who met at the Blackstone Hotel in Fort Worth, Texas. While before this group, more than one doctor examined Judy and nothing was found wrong with her.

"On February 18, 1950, both parents and Judy attended a meeting of physicians at Tampa, Florida. Here Judy was again shown to the group of

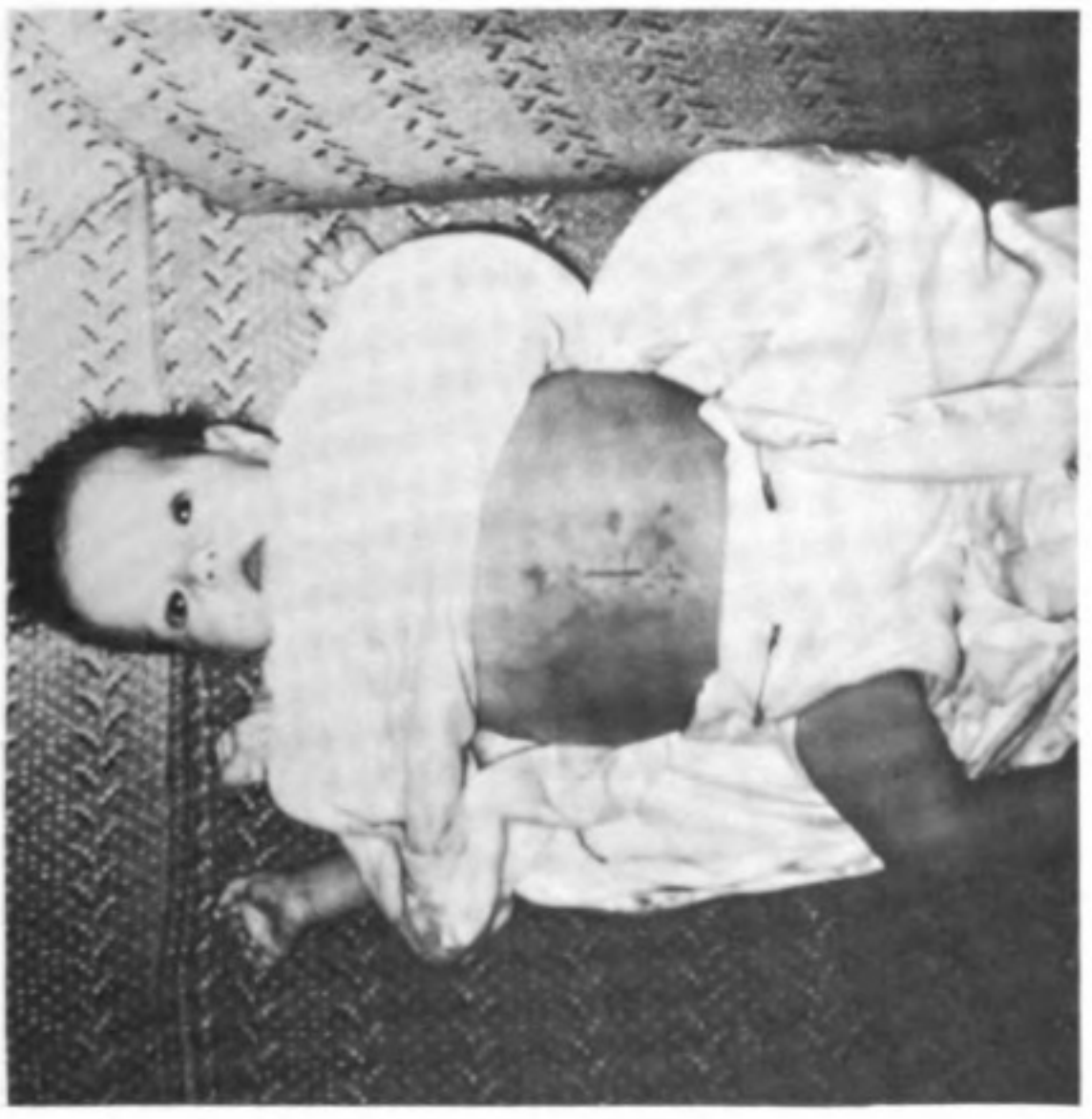
doctors. These were most friendly to the Koch treatment. Judy is now past two years old. She has shown a normal growth and development, normal mental development, and absolutely no abnormalities that we are aware of. She is very active, mischievous, and friendly. She has had practically no illness after taking the Koch treatment and recovering from cancer." These statements by Judy's parents are signed and notarized.

Dr. M---- sent a more technical account which adds nothing to the facts given by the mother. This mother's report should be studied. The observations are well made, and any physician who is experienced, can get a great deal of information out of them. The steady progress of the pretreatment period is plainly established. The manner of absorption of the neoplasm shows it was not of the diffuse type as the next case demonstrates, but was a massive cancer starting in the right lobe of the liver, and of very high (grade IV) maglignancy no doubt. The block in the portal circulation was not due to destruction of the vein, but to simple pressure, and the quick relief had, shows also in the better bowel and kidney action as the massive growth underwent absorption. This case should be compared with Case No. 15 which begins on page 101.

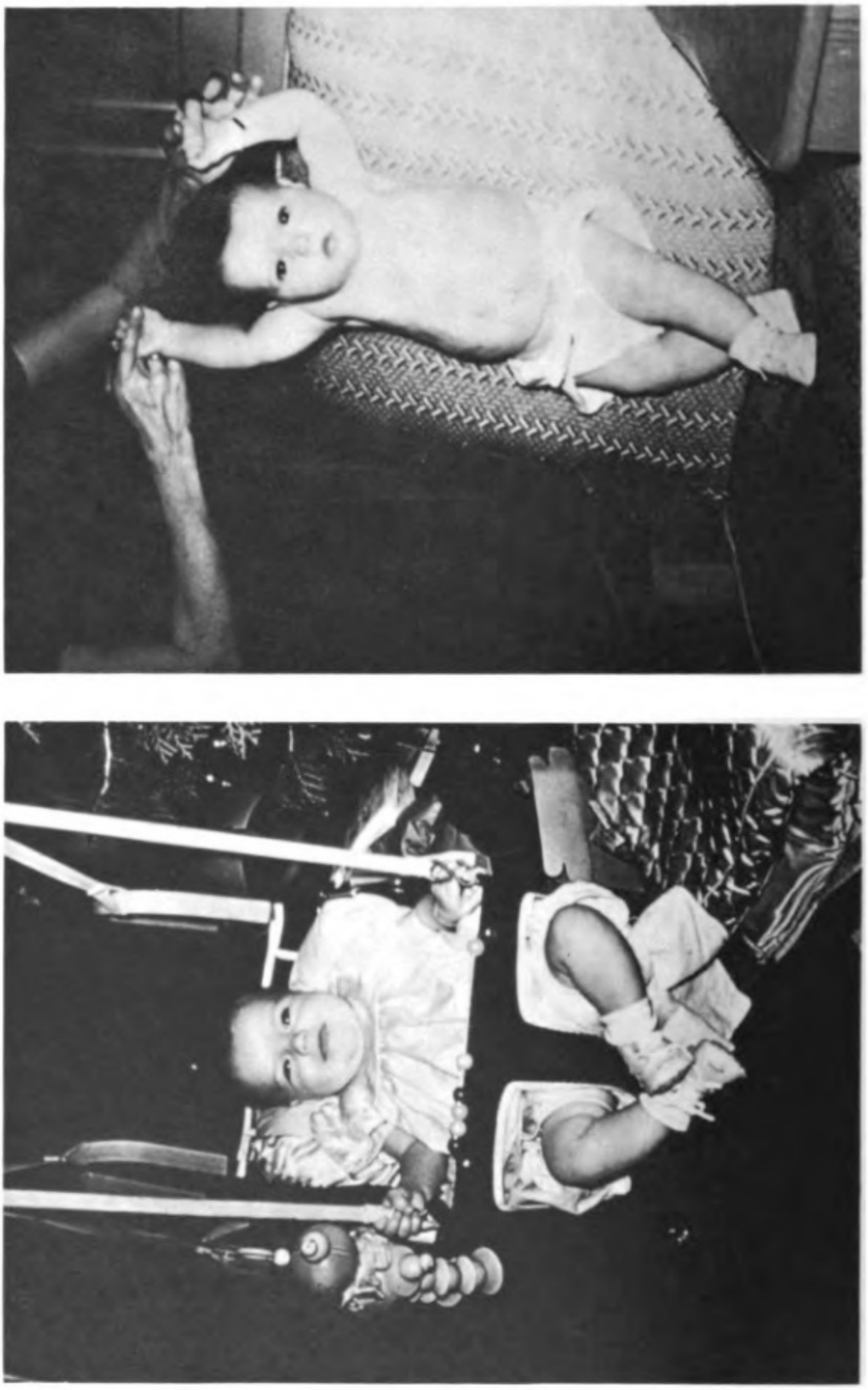


No. I, Taken before the injection,
September 18, 1948.

No. III, Taken several weeks after treatment.



No. II, Taken at the time of the treatment, September 18, 1948.



No. V, Six months after treatment.

No. IV, Taken a few weeks later.

No. VII, Taken a few years later.

No. VI, Taken a year later.

**No. VIII, Taken September 1960,
twelve years after treatment.**

DIFFUSE TYPE PRIMARY CANCER OF THE LIVER

CASE No. 15 Prof. R. S. L.

Mr. Geraldo A., early in March, 1941, showed loss of appetite, progressive loss in weight and weakness. Dr. O. A. L. examined him and found a large tumor in the abdomen. The patient was conveyed to Rio de Janeiro and placed in the hospital of the Beneficiencia Portuguese where he was operated by Professors R. L. and J. M. G. and helped by the assistant physician. Laparotomy disclosed an extensive neoplasm of the liver that had infiltrated the colon extensively. So extensive were the infiltrations and so widely had it spread, the operators decided it was impossible to give any surgical aid. Further, the type of liver cancer was so definite it was not necessary to remove a biopsy to add to the information already provided by the laparotomy findings.

The writer examined the patient together with his surgeons and Prof. R. S. L. seven days later. The patient could not turn in bed, was vomiting black bile or blood continuously, could not eat, and was of a dark jaundice color, very thin, in fact so emaciated that one could feel the diffuse nodules over the whole liver surface as it bulged forward and extended down into the pelvis. His doctors said he might live a week or two but certainly not longer.

We gave him an injection of a parabenzoquinone solution, 2cc. containing two micrograms in solution, on July 15, 1941. The patient who, up to that time, had been showing only a light fever, had a severe chill three days later, and a fever of 39°C. On July 25th, he belched much gas, and had intense gastric pain for only a short time. The tumor steadily absorbed, and concomitantly his weight and strength returned. The jaundice faded and he gained 20 pounds in weight, was able to walk, and returned to his home in Barbacena, where his health gradually returned. On October 3rd, he had another reaction with a chill and fever of 39°C., and a general oedema was shown. Everything yielded spontaneously, and the patient gaining then over 40 pounds had completely recovered and was back to work. The report in April, 1953, and again in 1955 shows he is still in excellent health and cured.

CANCER OF THE STOMACH

CASE No. 16

Mr. Wesley R., had a past history of severe gastric ulcer from 35 years of age until he reached 50. When he was 69 he had a more serious stomach trouble — pain and vomiting, with rapid emaciation, and loss of strength. Pyloric obstruction became complete. Several physicians made examination and found a tumor at the pyloric region. He was operated by Dr. Demling, June 28, 1926. A gastroenterostomy was done and a part of the tumor removed. The pathological report follows:

PATHOLOGICAL LABORATORY

3

Patient R (Last Name) W. S. (First Name) Ft.Wayne (Room) Steele (Doctor)

Date 8/7/26 Clinical Diagnosis (Stomach tissue)

Slide No. 268 Gross No. _____ Museum No. _____

GROSS EXAMINATION

Tissue of stomach.

MICROSCOPIC EXAMINATION

Small alveoli combined with a diffuse growth of atypical proliferating epithelium form the structural picture of this neoplasm. The epithelial cells are generally polyhedral or round in shape, with large hyperchromatic nuclei. One portion is necrotic - a superficial ulceration. This may be classified as the diffuse type of gastric carcinoma. I am unable to determine this point exactly as it is necessary to know something of the gross appearance. If there were extensive involvement of the wall, this would be the correct interpretation. If the growth were sharply defined, rounded and ulcerating, it would be placed with the circumscribed types of carcinoma simplex.

This type is always infiltrating and early invades the lymph nodes with wide-spread metastases.

DIAGNOSIS Carcinoma of the stomach. (Type dependent upon the gross pathological anatomy.)

Andrew Waltham —

It is evident from this pathological report that the whole neoplasm was not removed and this is confirmed by the early recurrence of obstruction by the neoplasm.

Improvement was noted for only a few weeks and then the trouble recurred with more pain than ever and constant vomiting, rapid emaciation and cachexia. He was brought to me by Dr. Harrison on August 20, 1926. My examination revealed a large fixed tumor mass filling the epigastrium and extending below the level of the umbilicus. It was fixed to the liver, and bulged outward so as to be plainly visible and caused practically complete obstruction of the gastric outlet. The supraclavicular space on the left side showed a fixed lymphatic tumor as large as a walnut. There was considerable hemolysis. One dose of the serial systems of carbonyl groups was given and recovery set in so that its effects were observed in a few weeks. The obstruction soon disappeared and he regained about twenty pounds to reach his normal weight in five months. Examination after the twenty-fourth week revealed no tumefaction. Radiographs show no tumefaction, but a stomach about one-third normal size, motility good. Only at the third, twelfth and twenty-fourth week periods were there reactions of note. Fever, tenderness in the stomach, and loss of appetite and a general achiness lasted about three days and then a much more pronounced improvement set in after each reaction. This improvement continued until full recovery was established. He has not had any stomach trouble since and enjoys vigorous health, works every day and walks to town in all sorts of weather as well as he did at fifty years of age. We heard from him last when he was 92 years of age and in good health, 23 years after treatment. Certainly the restoration of the Survival Chemistry was satisfactory in this case.

CANCER OF THE STOMACH

CASE No. 17 Dr. W. Mantor

This was a case of cancer of the stomach equally far advanced as the former. His trouble started as indigestion in 1940. Radiographs revealed no pathology then. It soon changed to a progressive stomach complaint with constant pain and frequent vomiting, rapid loss of strength, and a weight loss from 150 to 120 pounds in less than a year. Several well reputed clinics were tried in this year but the disease progressed. Radiographs made May 12, 1941, at the Tyler Clinic at Omaha gave a firm diagnosis of cancer of the stomach. At least two-thirds of the stomach wall was involved as the plates show. Exploratory operation at the Mayo Clinic within a week revealed massive involvement of the stomach wall, the pancreas, the glands about the aorta and the liver. The supraclavicular glands of the left side were also involved. They gave a diagnosis of far advanced cancer, primary in the stomach, entirely inoperable, and hopeless, and sent him home.

On June 16 he was carried into Dr. Mantor's office for treatment. Dr. Mantor's description includes the following, "extreme exhaustion, anemia, hemolysis, cachexia. No crenation of red blood cells in a one per cent NaCl

Master B 12/14/42

SURGICAL CARD
MAYO CLINIC — ROCHESTER, MINNESOTA

No. 1-152-560 Age 58 Sex M Sec LOGAN Date of Ex 5-24-41

Name [illegible] Address HANOVER, KANSAS

Name of Dr ..... Dr's. Address .....
Not Referred Accompanied Patient Sends Letter to CLAGETT Referred Only Wishes to be notified date of operation

Name of Relative ..... Patient accompanied by .....

Operation advised by Consultant [illegible] BROWN Surgeon WALTERS

Preoperative Diagnosis ULCER vs CARCINOMA STOMACH.

Operation Indicated EXPLORE.

Considerations affecting risk RISK III LEFT RECTUS INCISION.

Former operation here or elsewhere Date ..... No former operation here or elsewhere

A2-R(2-)- B B2 Col.

Date oprn 5-26-41 Oper. Room I-4 Dr Walters 1st Giffin 2nd Strom Recorder MKM

H McDonald

Antist Antic. $C_2H_4 + O_2 + C_{O_2} + \Sigma$. Time of { Anes. 1:10 – 1:55 / Op. 1:20 2:00

Oper. Diag.: Carcinoma of the stomach (inoperable).

Oper.: Abdominal exploration.

MKM

Drainage

Add. cond. to index:
Detail

Primary upper left rectus muscle splitting incision. There was a carcinoma forming a mass about 12 cm. in diameter on the posterior wall in the fundic end of the stomach with invasion of the pancreas. There were several enlarged apparently involved nodes along the aorta. The condition was inoperable and the wound was closed as an exploration, using five buried silk sutures in the fascia. Closure by (first).

A-82-b—Revised 2-10-41.

MAYO CLINIC
ROCHESTER, MINNESOTA

CLINICAL SECTION
OF
DR. ARCH H. LOGAN
DR. PHILIP W. BROWN
DR. J. ARNOLD BARGEN
DR. E. G. WAKEFIELD

October 13, 1941.

A-1-158-500

Dr. H. E. Mantor,
Sidney, Nebraska.

Dear Dr. Mantor:

Mr. William J. S of Hanover, Kansas, asked that we send you a report, and the following is a copy of the letter which Dr. Walters wrote to Dr. Hurtig on May 28, 1941. As you will see by this report, the situation certainly looked none too favorable and it is gratifying to know that Mr. S says he is feeling fairly well at this time. Both Dr. Walters and I would appreciate hearing from you if there are any additional findings in his case.

"I operated on Mr. S on the twenty-sixth. There was a carcinoma forming a mass about 10 cm. in diameter on the posterior wall in the fundic end of the stomach with invasion of the pancreas. There were several enlarged, apparently involved, nodes along the aorta. The condition was inoperable and the wound was closed as an exploration.

"Mr. S withstood the exploration satisfactorily. We are very sorry, indeed, that the lesion proved to be inoperable. While we fully realized the seriousness of the patient's condition, we are disappointed that we were not able to accomplish something that would afford him at least a measure of relief."

Yours very truly,

pwb-on

Philip W Brown

Philip W. Brown, M.D.

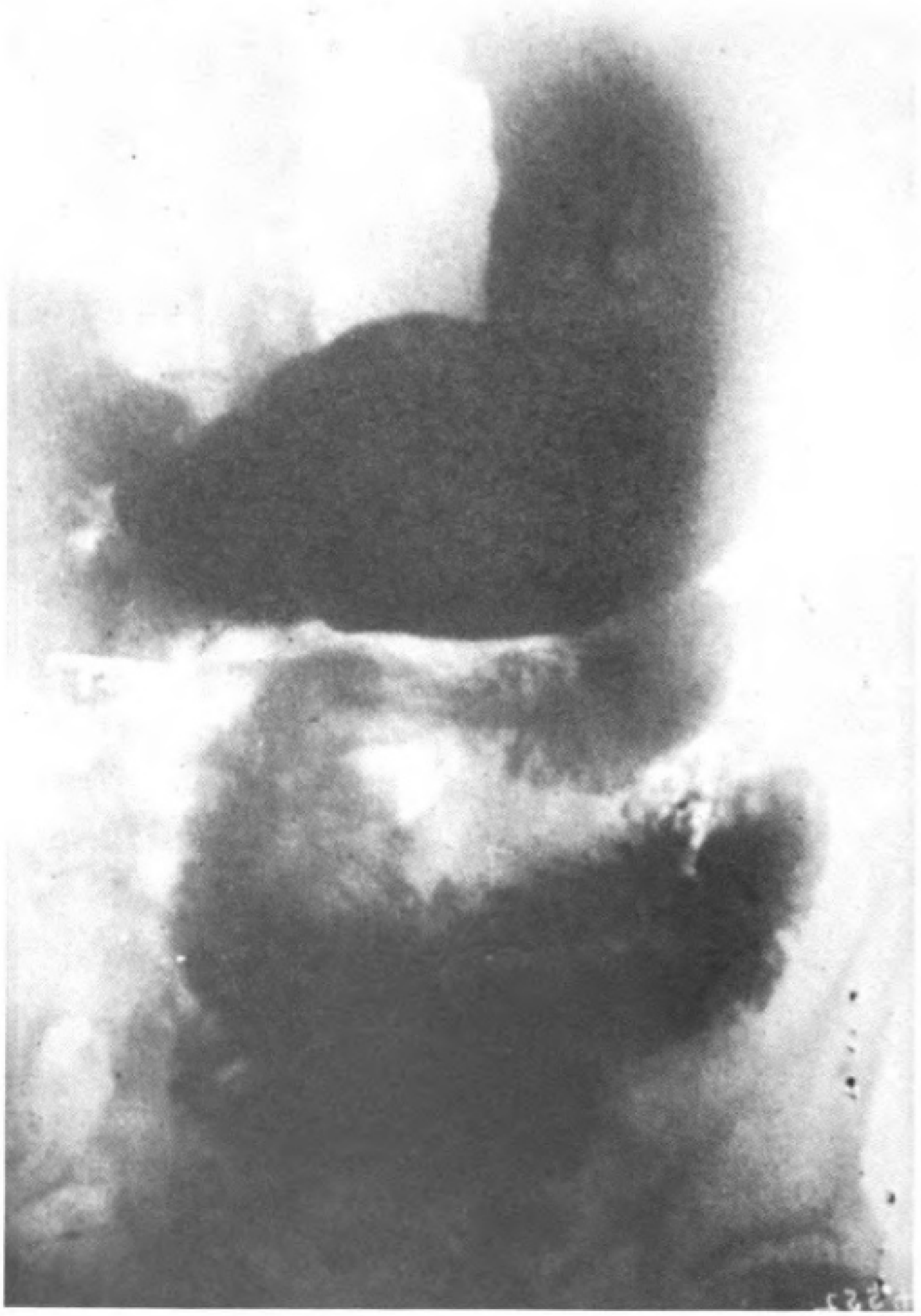
solution (all should crenate). Linear scar from exploratory operation, massive induration of the epigastrium, readily palpable and bulging forward so one could see it easily as he lay down. Since one year previously an X-ray of the stomach showed no pathology whatsoever, this neoplasm was very malignant and rapidly progressive and destructive."

One injection of 2 cc. of the 12X dilution of the serial system of carbonyl groups was given June 16, 1941. In a few days he started to feel better and soon took up the farm he had to leave because of the sickness. Nine weeks after the treatment, examination could reveal no tumor mass whatever. He had gained weight, color improved and was more active. By the 12th week he could walk down the street rapidly without losing his breath and reported he was eating well and was feeling fine. By that time he had been working on his farm. There was no more cachexia. Dr. Mantor gave him a second injection on September 8, 1941, during his 12th week. He continued towards complete recovery. A third injection was given two years later, September, 1943.

Radiograph III, taken on June 14, 1944, shows his stomach after complete recovery. He was still well in 1947 when we last heard.

Radiograph I, taken at the Tyler Clinic before treatment.

Radiograph II, taken at the Tyler Clinic a few weeks after treatment showing marked improvement.



Radiograph III, taken June 14, 1944, after recovery.
These radiographs are court exhibits.

The Survival Chemistry of course is concerned with the oxidation of burnable substances for energy production. Fuels and disease producing toxins that come within its O/R potential ranges are destroyed. The energy is used for doing work in the performance of function, and for the growth and repair. One can estimate the thoroughness with which the FCG system is restored by the rate of tissue reconstruction after the most devastating of cancers. The following two cases will illustrate.

METASTATIC CANCER OF THE BOWEL

CASE No. 18 Dr. F. Richards

Mr. J. K. was 42 years old when X-ray studies and exploratory operation showed widely metastasizing cancer of the splenic flexure of the colon, which caused complete obstruction. A colostomy was made at the Henry Ford Hospital at Detroit. The neoplasm spread rapidly throughout the abdomen and perforated the abdominal wall in several places as cauliflower growths with central fistulous openings that discharged feces and foul necrotic material. His general health deteriorated rapidly under the toxic strain, and the neoplasms grew proportionately rapidly under the same impulse. He had been a strong muscular man of 180 pounds swinging a heavy hammer all day when he took sick, and after the surgical aid he lost to 135 pounds, and kept on losing. The necrotic tissue in the abdominal wall had such a terrible odor it was necessary to slice away the most gangrenous part, and cauterize the borders to block the bleeding. The cauliflower fistulae took on greater speed thereafter. He was sent home to die, his case being entirely hopeless. On the way home he was examined at our clinic. Dr. Richards gave him an injection of the SSR reagent on April 3, 1942. At this time the cauliflower growths were from 4 to 8 centimeters in diameter and equally high. During the following three weeks he did not show much improvement. Then he took a heavy reaction with such great congestion of the exposed cancer masses that another injection had to be given. The second treatment was given at the beginning of the fourth week. Thereafter the bleeding quickly stopped and his whole condition rapidly improved. The cauliflower masses melted away and the abdomen was healed without leaving a visible scar. Palpation could reveal no more tumor masses after four more months. His bowels started to move normally through the rectum, and the colostomy stopped functioning. He gained weight to 113 pounds in June, and to 180 pounds in September when we sent him back to the Ford Hospital for repair of the colostomy, after he had returned to work a few weeks.

Henry Ford Hospital

NAME K JOHN I 2 DATE 2-27-42 CASE NO. 342016

GENERAL MEMO

DIAGNOSIS: Fungating Carcinoma of Colon

OPERATION: Electro Coagulation of Tumor Mass.

OPERATOR: Dr. Fallis

ANESTHESIA: Ether and Nitrous Oxide by Miss Silyce

PREPARATION: Hexylchloro-M-Cresol

OPERATION: As we commenced the operation, we do consider the possibility of resection of the left half of the transverse colon. With the radio knife, we therefore cut around well beyond the margin of the fungating mass and strip back the skin flap. As we encounter the left rectus muscle, we find that the tumor mass is infiltrating throughout, so that resection is out of the question.

With the electric cautery, we therefore cut off the bulk of the tumor and coagulate all the protruding mass. The skin flaps are then undermined and brought together with interrupted wire sutures. Sopi us dressings are applied and the patient is returned to the floor in good condition.

This is entirely a hopeless case

HENRY FORD HOSPITAL
Surgical Pathological Memo

Photo:
Name: K. J.
Sex: Age: 42
Path. No. 66933
Case No. 342016
Date: 2/27/42
Pathological Diagnosis 101.62 CUTANEOUS SYSTEM: SKIN OF ABDOMINAL WALL: METASTATIC ADENOCARCINOMA
Location of Lesion Abdominal wall
Clinical Diagnosis
Operation
Operator Dr. Fallis
Gross Pathology

The specimen consists of a piece of skin measuring 14x14.5 cm. The central portion is destroyed and partially filled by a friable gray 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.

IMPRESSION: Secondary carcinoma of abdominal wall.

MICROSCOPIC: Section shows a tumor mass invading the subcutaneous tissues. 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. A massive necrosis affects large areas of the tumor.

ea/

This X-ray was taken of Mr. J. K. on
December 27, 1941 before treatment

His liver and other FCG's that had been blocked by the carcinogen certainly went back to work for him, for he not only could digest food efficiently to return to work, but to build up his tissue at the same time. His gain is shown in part in the Ford Hospital records as noted by Dr. Bohr. For the month of July the record notes that he gained from 113 pounds at the first of the month to 175 pounds at the end of the month, that is a gain of 62 pounds in a month, or just two pounds or one kilo per day. This gain, we must add, was made on our vegetable, fruits and cereal diet, without any meat or animal proteins. The Ford Hospital documents that were part of the court record are worth studying. Some are appended. This gain in good solid flesh and blood on a vegetarian diet is not just an incident in this case. It is our experience, and meat eaters who can not curb their appetites for animal food, should give these facts some consideration.

The following significant statements are taken from the Henry Ford Hospital record, the Interval History taken by Dr. Bohr on August 28, 1942. "Patient left hospital April 1 of this year with a diagnosis of fungating Cancer of 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. His weight was 175 pounds at the end of July and he has maintained this weight ever since then. He enters the hospital now, after being back to work for three weeks, for first stage in a colostomy closure."

This patient was examined at the Ford Hospital every year for many years and always found well. When last heard from he was still well. The radiographs show the obstruction of the bowel during the first weeks of his illness. The radiographs made after recovery are the same as any normal person's. No adhesions or other sequelae showing that disease existed formerly, are found.

What the Case Teaches

Firstly, the Ford Hospital "General Memo" tells that they could not enter the abdomen to attempt a resection of the growth as it had infiltrated throughout and invaded the abdominal wall throughout. When one considers the position of the splenic flexure of the colon, back against the posterior abdominal wall and diaphragm the distance to the umbilicus is twelve or more inches and as it had finally ruptured through the belly wall in three different places as large cauliflower fistulous growths the amount of involvement was about total. It is important to realize this and also that the amount of infection throughout the whole neoplastic envolvement was also tremendous. Fecal fistulae always heal with a great amount of scarring and distortion and epidermal change consequent thereto. But in this case there was no scar left, nor epidermal change left to mark the areas of neoplastic or infectious envolvement. The abdomen wall healed right through the skin without leaving

a mark. The abdomen looked as clear as any normal baby's that had never been sick. Just as the bowel function was restored so the colostomy became obsolete. So too, the only sequelae to the neoplasm were completely restored structure and function.

To the regular physician this fact is not comprehensible, and indeed if the other patients to be demonstrated here did not do likewise, one would scarcely have the courage to make this report. There is an explanation, however, and the case is offered with others as a demonstration of this explanatory observation. It is this. The recoveries in these cases after the SSR is used are different from those that take place under the flux of regular ordinary healing. This is due to the fact that after the SSR does its work no toxin either carcinogenic nor from disease germs is present, integrated with the protective fibrosis or the cancer cells. Before the SSR was used the toxin content was tremendous, if the odor, cachexia and rapid tissue destruction mean anything. The state of integration as we explain here and in the Appendix actually invited oxidative destruction of the toxins of all types, so that the fibrosis and cancer cells became obsolete and were absorbed by the ordinary autolytic procedures so that nothing was left to interfere with normal tissue reconstruction. So normal reconstruction was not prevented and the normal rectifying tendency had full sway. It is such an observation, so oft repeated, that makes one think that the normal state of man was such that he did at one time in his perfect state really possess an FCG of the order of the SSR or even more efficient.

SARCOMA OF THE UTERUS

CASE No. 19

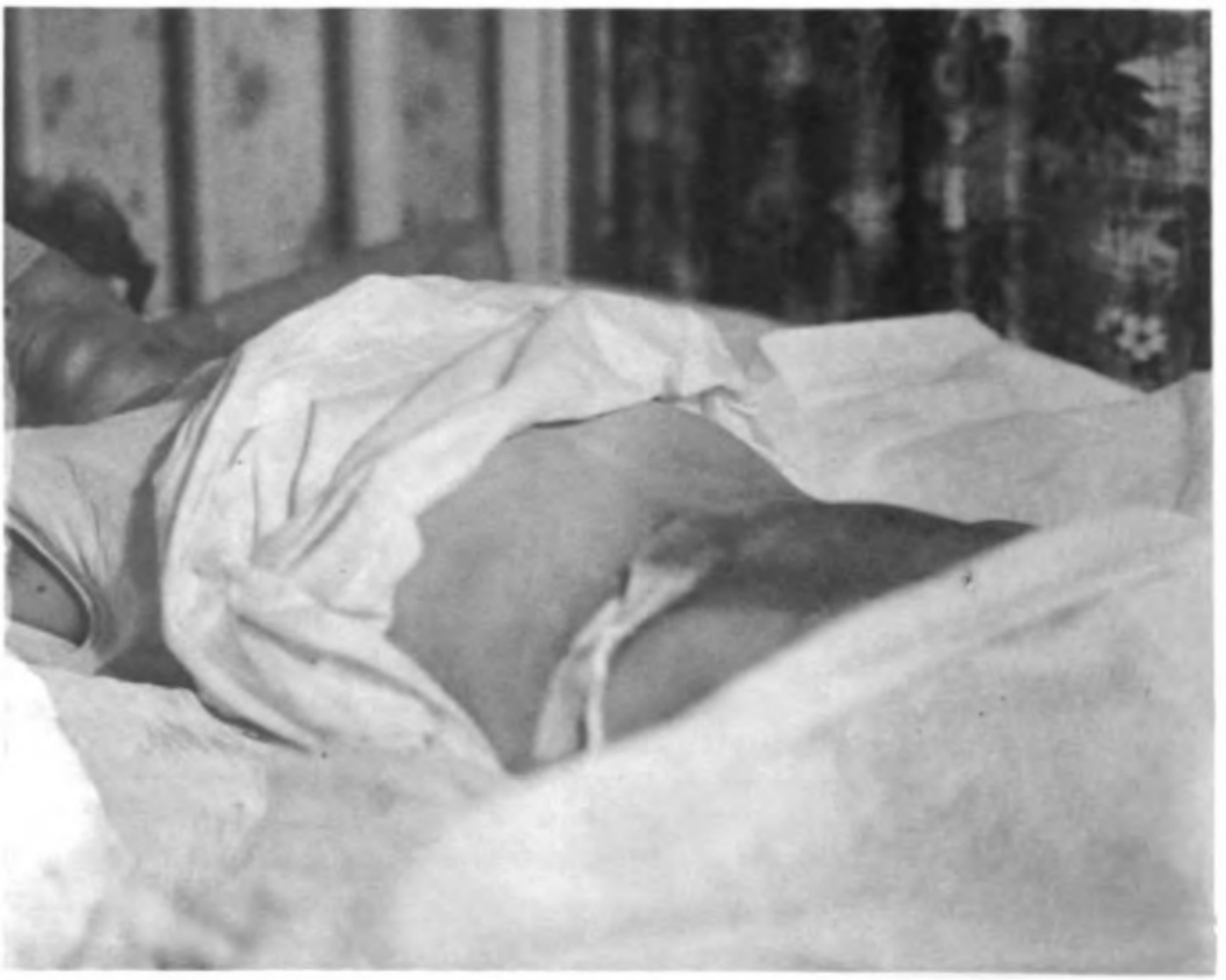
Mrs. McA., aged 43, was first seen on July 29, 1929. She was bedfast, emaciated, and exhausted. She had not rallied well from an abdominal exploration done by a very good surgeon two weeks previously to ascertain the cause of severe and frequent crises of vomiting and pain in the gall bladder region. The abdomen was found widely involved with neoplastic development from deep in the pelvis to the diaphragm, with the stomach and liver and intervening structures heavily invaded. This was identified as the cause of the pain. A biopsy specimen was removed. The abdomen was closed as inoperable. A biopsy report was given to us personally by the surgeon, Dr. Trimby, when he referred the patient. It showed a small round cell sarcoma of high grade malignancy.

Our examination revealed a patient in bed, exhausted with weak pulse, sighing respiration, vascular shock, cyanosis, and an abdomen bulging with

tumor masses, particularly on the lower left side. The liver and epigastric involvement could be readily palpated besides. The incision was not healed and appeared to be infiltrated with neoplastic extensions. This incision was made over the largest tumefaction within and it seemed that the abdominal wall had been invaded by the neoplastic tissue underneath. A photograph was taken and 2 micromicrograms of the Synthetic Survival Reagent were given.

She responded well up to the sixth week, gaining in strength and becoming rapidly free of the pain and vomiting attacks. However, at the beginning of the sixth week she started a reaction that continued to the middle of the ninth week.

It featured vomiting of a quite continuous nature whether she took food or not. No food was held. The pain feature that was so severe before treatment was a minor matter, however. She lost weight and strength, and the dehydration was difficult to overcome. However, with the closing of the reaction at the middle of the ninth week she became very hungry and took on weight rapidly. For a few weeks she gained at the rate of five pounds a week,



Photograph I, taken at time of treatment.

Photograph II, taken after neoplasms were absorbed.

then at the rate of two pounds a week until she had reached 180 pounds. A slow gain followed to 200 pounds and then a slow loss to 180 pounds. Her health was fully established. All the tumefactions had disappeared before the end of the twelfth week after the treatment. She is still in perfect health, according to our last report.

Photograph I shows the condition at the time of treatment. Photograph II shows the condition after the neoplasms were absorbed.

Another way to estimate the restoration of FCG activity is in the cure of Leukemia. Here in the terminal phases, the exhaustion of the blood forming organs can be so complete that leucopenia instead of the former leucocytosis is observed. The FCGs required for cell construction are blocked, no doubt, by the carcinogen, and both the red cell count and the white cell count are not able to be restored until the FCG's are liberated and can go back to work again. The following cases are illustrative. Here the bad effects of irradiation are to be observed as well as those of the natural carcinogen. One might compare the response in a natural case of acute lymphatic leukemia with other cases that have been irradiated, or have reached the point of exhaustion and impending death. These latter states are about the same, showing how irradiation works on the blood forming organs. It kills them as does the natural pathogen. Red and white blood cell restoration demonstrates FCG restoration.

ACUTE LYMPHATIC LEUKEMIA IN A BOY

CASE No. 20 Dr. A. Guzman

P. F., age 12 years, treated January 8, 1956, by Dr. Guzman. His family history denied leukemia. The onset in the boy was rather rapid after a period of ill health. The symptoms were classical with petechial hemorrhages under the skin and in the mouth, cough, symptoms of anemia with great weakness. The red cell count was 1,500,000, the white were 232,000, lymphocytes in very great predominance, large mononuclears and immature forms. The physical examination showed a blanched out boy with hemorrhagic spots of various sizes under the skin generally, especially the legs and arms and body. The gums bled, and the breath was foul. The mediastinal dullness was increased mostly to the right. The spleen and lymph glands were only moderately enlarged. He had a cough, was very weak and showed fever.

The injection of the Synthetic Survival Reagent was given, 2cc. of the 10^{-12} solution. He was put on a supportive vegetarian diet. The recovery was steady with periodic reactions of chills and fever, general achiness, etc., as characterizes the recoveries under this treatment.

The results showed in an improvement after each reaction until in August, 1956, he was completely well. There were no signs of the disease left. The hemorrhagic spots had disappeared, the mouth was clear and the breath clean. The spleen and lymph glands were no longer enlarged. The spleen could not be palpated, and he was strong, had gained a normal weight and nutrition, etc. His platelets count was then 350,000, the red cells 5,100,000, white cells 7,200, polymorphs 76%. The coagulation time was normal. The only reaction of a focal nature was a sore throat, and that in a boy is difficult to interpret. However, it might mean that this was the site of the pathogenic toxin or virus production. Especially is this suspicious since the mediastinal glands were enlarged and the mediastinal dullness returned to normal with the recovery.

LYMPHATIC LEUKEMIA WITH TERMINAL EXHAUSTION

CASE No. 21 Dr. Julian Baldor

Teddy S., age 14 years, in February, 1949, when treated by Dr. Baldor. He was referred by Dr. C----- with the report, "Chronic leukemia (proved by bone marrow biopsy) with hemorrhagic diathesis." The exhaustion of the blood forming organs was seen in the fall in the blood count of whites to only 15,000, while the hemoglobin was 40% and the red cell count 2,150,000. His first diagnosis of leukemia was made six months previously from the high lymphocyte count, the hemorrhages under the skin and the gums, the weakness, enlarged spleen and lymph glands, the fever, pains in his legs and arms and the anemia. He was given 57 blood transfusions. These pepped him up a little at the start, but soon were found to be less effective and finally to be of no

help at all. In fact, they were wasted on him. It was then he was sent to Dr. Baldor for treatment.

Dr. Baldor's examination showed an exhausted bleached-out boy, suffering with pain and fright, depressed, unable to walk, with an offensive odor from the mouth, profuse gingival bleeding, generalized hemorrhagic spots under the skin. The mediastinal dullness was definitely enlarged and also were the lymph glands generally, increased tubular breathing in the right lung base, and moist rales over the entire field. The liver and spleen were enlarged and tender. He was put on fruit juices and intestinal lavage for two days and then given 2 cc. of the SSR serial system of carbonyl groups. The fever at the time was 102°F.

In a week he was sent home with a normal temperature. The spleen and liver were reduced in size, but not yet normal, the bloody patches had changed from a dark purple to a greenish color and no new ones had formed. They then turned to yellow and finally disappeared. Nine weeks later he returned to Dr. Baldor for a check-up. He was able to walk, had gained 12 pounds in weight. The blood picture was red cells, 3,350,000, hemoglobin 52%, and leucocytes 8,000. At the twelfth week, he gave a reaction showing slight pains in the extremities for a few days, and a little epistaxis. The blood count then showed 4,000,000 red cells, white cells 6,500, hemoglobin 72%. He had gained 25 pounds in weight, and felt perfectly normal in all respects in feeling, behaviour, and in his physical findings.

It was reported to Dr. Baldor in 1957 that Teddy S. at the age of 21 was examined for military service and classified 1-A. This was seven years after being treated for what normally is a 100% fatal disease, in its terminal stage. Upon knowledge of his full previous medical history he was re-classified. Dr. Baldor reported at that time that Teddy S. had held the best of health since his recovery, is married and is the father of a robust healthy child.

It is to be noted that after being seen by Dr. Baldor he did not receive even one blood transfusion, but gained in all respects on a vegetarian diet. It might appeal to one that the extreme exhaustion is not only a matter of lack of nutritional elements to support cell function, but even more a lack of energy to perform the functions of work and nutrition. The impediment to the mechanisms concerned blocked all activities including the burning of all sorts of toxins absorbed from the bowel, the mouth, tonsils, etc. Then, with the liberation of the tissue FCG systems every impediment was burned out of the way, and was kept out of the way thereafter.

MYELOGENOUS LEUKEMIA WITH IRRADIATION LEUCOPENIA IN AN ADULT

CASE No. 22 Dr. Julian Baldor

Mrs. J. W. L., age 47 years, came on December 7th, 1948. She gives a history of an acute process with chills, fever, nausea and perspiration, six months previously following an influenza attack. Examination showed enlarged liver, enlarged spleen and enlarged cervical glands. The breath was offensive and

the gums were bleeding. Some dental abcesses were present. Her blood picture showed red cells, 3,160,000, hemoglobin 57% and white count, 14,800, ploys. 88%, lymphocytes 10%, monocytes 2%. Both myelocytes and premyelocytes were present.

She had received two courses of X-rays over the spleen and long bones, each of 600 R at an interval of 6 weeks. This did not improve her condition. The bleeding, weakness, fever and pains continued getting worse. Bone marrow slides showed definite abnormalities suggestive of Myelogenous Leukemia.

Because of the irradiation two doses of the SSR reagent were given, one on December 13, 1948, and the other five days later. The improvement was prompt. The fever had left in five days. The enormous spleen which reached to the left illiac region, and the enlarged liver showed improvement and were less painful. The oral bleeding and infection likewise cleared up. The blood count March 15, 1949, showed red cells 3,850,000, hemoglobin 69% and leucocytes 8,500. Up to August 5, 1949, she gained ten pounds in weight. On June 16, 1949, the red cell count was 4,000,000, hemoglobin 72% and white cells 7,000. The chest signs improved slowly. By the end of 1950 her enormous spleen had receded to its normal position under the left ribs. The last blood count taken May, 1955, showed red cells 4,150,000, hemoglobin 70% and leucocytes 3,500. She remains well.

This patient, like the others, was not given one blood transfusion after the SSR treatment was started, and she improved on a strict vegetarian diet. Here the destructive effects of the X-rays on the blood forming organs is easily seen, and the recovery of blood production could be better. The hemoglobin was only 70% when it should have been 80% or better and the white cell count could have been a thousand or two more. Thus the injury to the blood forming organs is not entirely corrected, and never will be, in line with our experience with the effects of irradiation. However, the gains made under this handicap when the FCG function is restored are well shown in this series of cases.

One should contrast these cases with the recovery from a slowly developing fatal form of bone sarcoma. Here it will appear that the rate of recovery is a function of the rate of development of the disease. It will be seen also that in the healing of the bone, the tissue is made much more dense and stronger than before the disease attacked it. Blood reconstruction is of this order, too.

ENDOTHELIAL SARCOMA OF THE BONE

CASE No. 23

Mr. Harold B. was age 41 in September, 1934, when he appeared for treatment. He first noticed trouble with the right arm when he threw a ball some weeks previously. His family physician took some X-rays and noted the sarcomatous status and sent him to the University of Michigan Hospital for thorough attention. The pain in the arm was sufficient to prevent its use, and motion was limited because of a hard swelling over the scapula. The X-ray

studies, blood studies and biopsies of soft and bone tissues led to the final diagnosis of Endothelial Cell Sarcoma of the bone. To reach this diagnosis, Paget's disease and all other bone tumors were definitely eliminated. They gave him a hopeless prognosis since this type of sarcoma is always progressive and fatal, no matter how it is treated, but they offered to remove the whole shoulder girdle if he wished. He refused and presented himself for our attention instead.

Record R.

UNIVERSITY OF MICHIGAN
UNIVERSITY HOSPITAL
PATHOLOGICAL SPECIMEN

Name Harold B No. 554166 Date 9-1-54

Service Surg. SE Age Sex Pathological No. 1716-A[M]

Address Occupation

History of Case Tumor mass over right scapula posteriorly. ? myeloma.

Operated by Dr. Iglesias. Nature of Operation Biopsy. Incision.

Question

Gross Description I. Numerous bits of cancellous bone.
II. Soft tissue from right shoulder: Bits of soft brown tissue, some pieces apparently blood clot. (I bits decal, II bits ns).

Pathological Diagnosis II. This is a malignant neoplasm, the final classification of which is in doubt. It is composed of round cells, only a small proportion of which show the eccentric nucleus and basophilic cytoplasm usually seen in the plasmocytoblastomas. The arrangement of the cells is suggestive of an endotheliosarcoma, probably hemangiosarcoma. Further report after decalcification.

Pathologist

H. Gordon

Pathological Diagnosis After decalcification: Bone is in large part replaced by neoplasm showing the same general histological characteristics as in the soft material. This is a spindle cell hemangiosarcoma.

Pathologist

H. Gordon

Our examination, made September 17, 1934, showed a lame right arm, a fist size mass over the spine of the scapula firmly fixed, and a walnut size mass closer to the dorsal spine and of the same texture as the other mass. Both were fixed to the underlying structures. There was some cachexia, but no tumor could be found in the abdomen or anywhere else. The X-rays showed bone destruction extensively of the humerus and scapula, and neoplastic development between the two. The progress of the disease was slow and steady. He was being poisoned by the etiological factor as well as the tumor products, following a down-grade course.

UNIVERSITY OF MICHIGAN

UNIVERSITY HOSPITAL

SURGERY

HISTORY SHEET

Name B , Harold -2- Case No 359162 Date 8-31-34

PHYSICAL EXAMINATION

GENERAL: The patient is a well developed male of 41 years of age appearing not to be acutely ill. Color good. Nutrition good.

HEAD: Scalp:Hair is normal in amount and distribution. No exotoses or areas of tenderness present.

EYES: The conjunctivae are of normal color. Sclerae are not icteric. The pupils are equal in size and round. They react normally to light.

EARS: No discharge. Hearing seems to be normal.

MOUTH: Mucous membranes are of normal color. The throat is not injected. The tonsils are present, not enlarged. The tongue papillae show no atrophy. The teeth are in fair condition.

EARS: No discharge.

NOSE: No discharge.

NECK: Neck symmetrical showing no abnormal masses or pulsations. The veins are not abnormally engorged. The thyroid is not enlarged or nodular.

THORAX: Percussion reveals no abnormal areas of dullness. The breath sounds are normal. No rales heard. The thorax is symmetrical. Considerable pain was complained of in the right upper chest posteriorly in the region of the scapula. Heart: LBCD 7 cm. Rate and rhythm normal. No murmurs. Blood pressure:100/80. No edema of the ankles.

ABDOMEN: Symmetrical. No areas of tenderness present. No masses could be felt. The liver and spleen are not enlarged. No signs of inguinal hernia.

GENITALIA: Testicles are normal. No nodularities of the vas. Shows normal development.

LYMPHATICS: No enlargement of the cervical, axillary, epitrochlear or inguinal glands.

EXTREMITIES: Biceps, triceps, reflexes are present, equal and active. The fingers are clubbed. Examination of the right scapula revealed a lemon sized mass below the spine of the scapula. This mass was firm, and rubbery in consistency; much tenderness complained of; the overlying skin was not discolored. The mass moved with the scapula. Pain was complained of on passive movement of the shoulder joint, however muscular motion seemed normal. There appeared to be no noticeable disturbances of the right arm. Pain was complained of in the upper dorsal region of the spine. This, he states, has been present for a considerable time. Lowers: Patellar reflexes are present, equal and active. The Achilles reflexes could not be obtained even with reenforcement. Babinski negative.

IMPRESSION: Spindle sarcoma of the scapula, with questionable metastases to the dorsal spine. Infiltrative involvement of the right shoulder joint.

db Dr. Smith

Form H-704201-7-34-100M-19538

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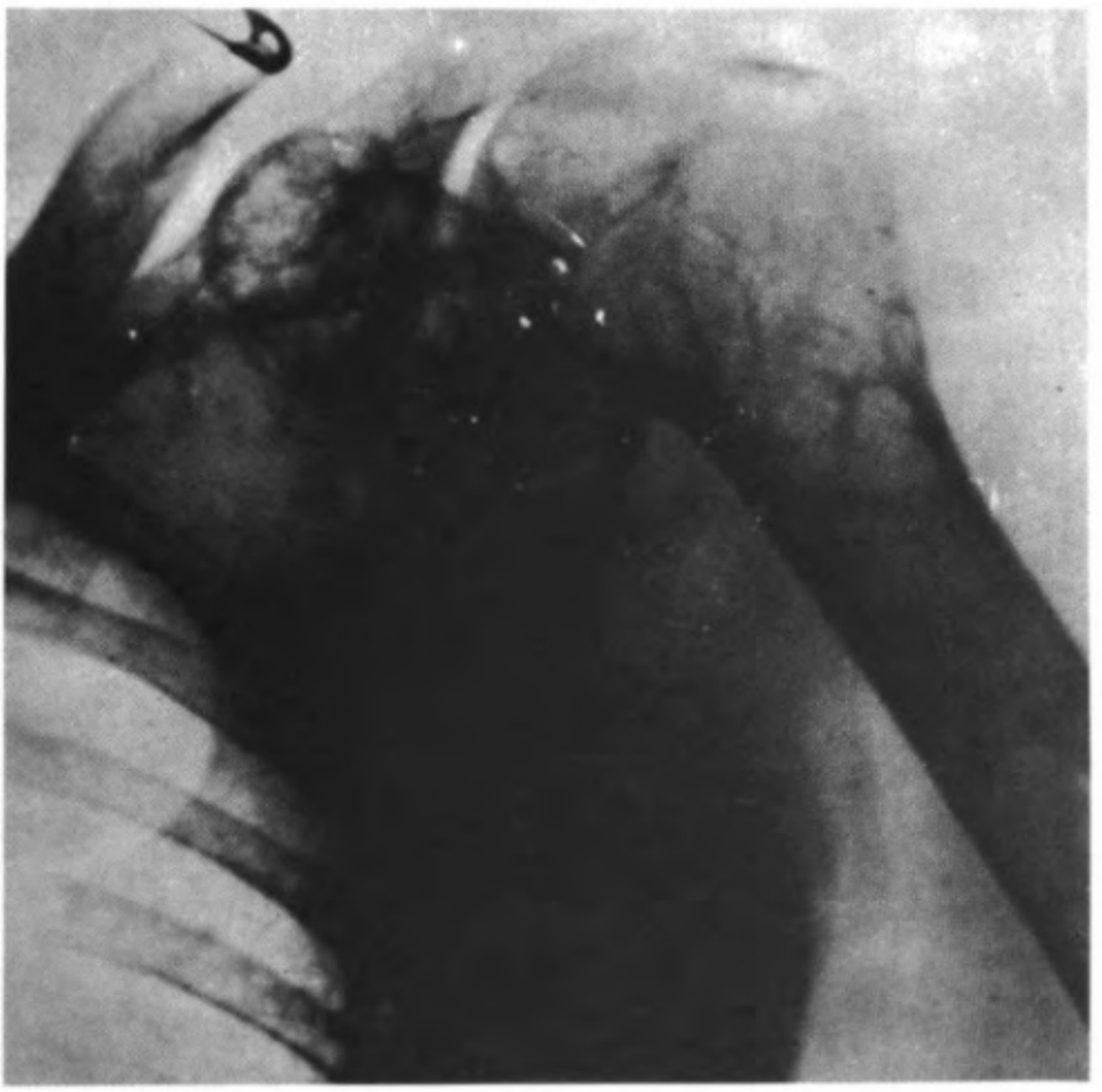
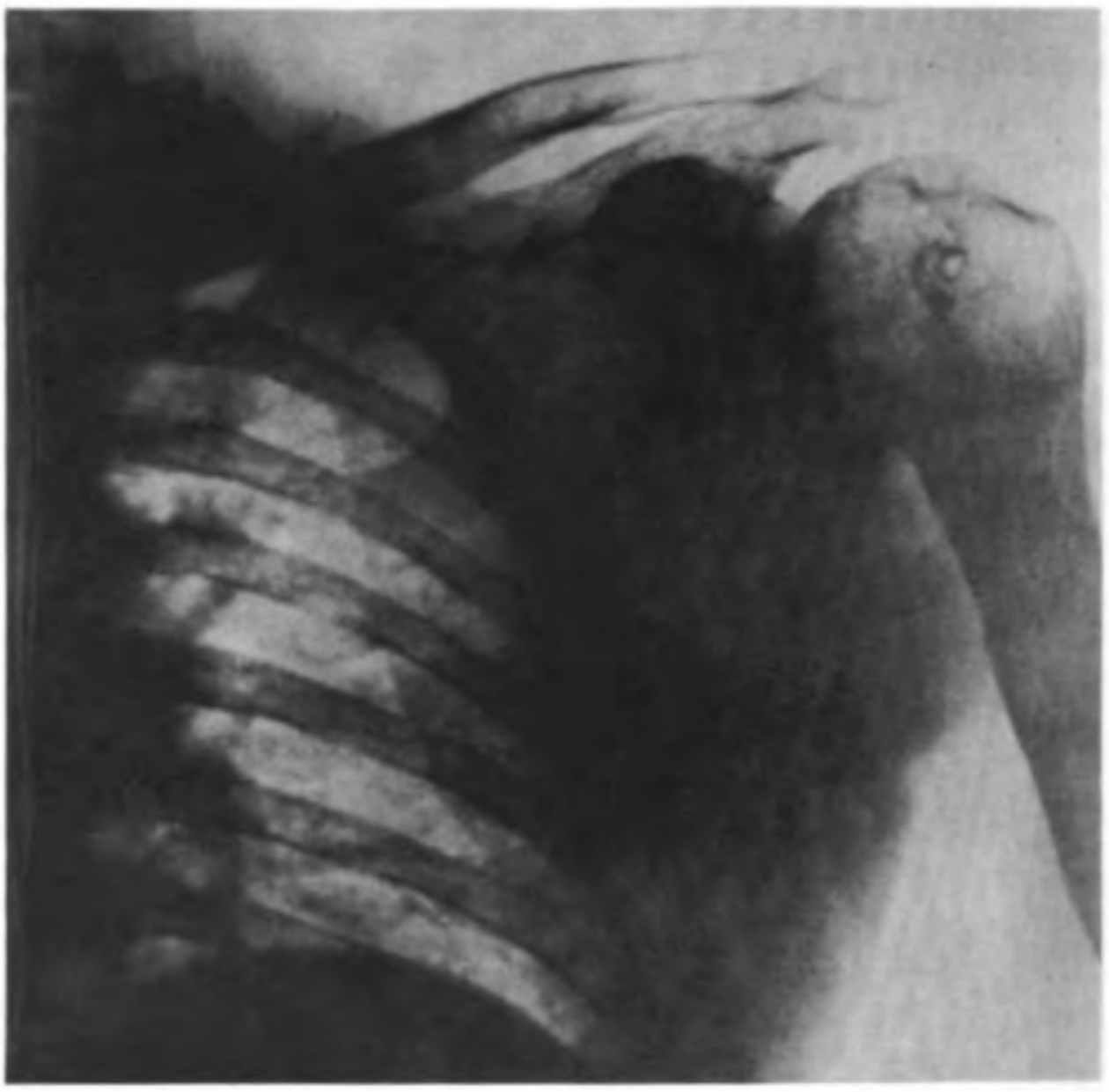
The Synthetic Survival Reagent (SSR) was given in a dose of 2 cc. of the 10^{-12} solution. There was no immediate sharp reaction, that is, in the next few days. The major reactions were at the 24th and 36th weeks. But he continued to improve slowly from the start. It took a few months to be able to use the arm without pain. In a year the tumors were completely absorbed, and an X-ray showed nearly complete recovery in the bone, with considerably more denseness in bone structure. He was able to return to work, and made a full recovery. Our next chance to make a radiograph was when he appeared in Detroit to testify for us in the Federal Court. The X-ray made then showed full recovery and is reproduced here. He has remained well. Our last report was received in 1950. Ewing's estimate of this disease is as follows, page 361, 1942 edition, Text Book on Cancer:

"Angio endothelioma, multiple endothelioma, diffuse endothelioma, or endothelial myeloma, the entire group, is characterized by predilection for the bone shaft, a tendency to multiplicity, a cellular and vascular structure, marked osteolytic properties, failure to produce tumor bone, and a relatively slow but fatal course."



Radiograph I, showing condition before treatment.



Radiograph II, showing condition after full recovery.

This relatively slow course is seen both in the progress of the disease and in its cure, and should be contrasted with the J. K. and Mrs. MacA. cases and the recoveries from the leukemia cases just given.

The University of Michigan records selected from the court records give the details sufficiently for a working idea. This record, like those of the other cases cited is voluminous. All other details are available to anyone who desires them. The radiographs should be studied also. The fact that the tumor is not confined to the bone but has grown between the bones and out over the shoulder shows it is not Paget's disease. One should note this in the radiographs.

RECURRENT METASTASIZED CANCER OF THE PALATE FOLLOWING SURGERY

CASE No. 24

Mr. A. J., age 60 years, was first seen by us on December 1, 1932. He gave a history of an attempt to remove one large and a few small growths from the hard and soft palates, by excision and touch-up cautery at the University of Michigan Hospital on October 15, 1931. The areas healed and all was well for a few months. Then the same type of growth reappeared over a wider

area of the palate and enlargements formed in the neck and under the jaw. These were deeply fixed. The palate was well covered at the time of our examination. The large growth had returned with a dozen smaller ones around about it. The biopsy was squamous cell cancer.

We gave him the Survival reagent injection and three days later he experienced a typical reaction. Generally a highly malignant cancer will give a recovery reaction twelve or twenty-four hours after the treatment is given and in the less malignant types as this one, the actions come as late as 72 or 84 hours after treatment. But the symptoms are the same, namely a general achiness, chills and fever. All patients describe their reactions much alike so we will give Mr. J's description as an example. His testimony stated, "About

UNIVERSITY OF MICHIGAN

DEPARTMENT OF OTOLARYNGOLOGY
UNIVERSITY HOSPITAL

R. BISHOP CANFIELD, M.D.
PROFESSOR OF OTOLARYNGOLOGY

A. C. FURSTENBERG, M.D.
ASSOCIATE PROFESSOR

ANN ARBOR November 13, 1951.

Dr. John W. Nagle
114 Maple St.
Wyandotte, Michigan.

My dear Dr. Nagle:

Your patient, Abram J , was transferred to us from the Dept.of Dermatology on August 12, 1951.

As you are undoubtedly familiar with this patient's history, I shall not relate same.

On August 14, the large lesion in the mouth was excised under local anesthesia. Following the excision, electro-coagulation of the base and also electro-coagulation of the surrounding small papules was done.

Pathological report of the specimen excised was: "Cornifying squamous cell carcinoma."

The patient was discharged from the hospital on October 16, with the advice to use frequent bland alkaline mouth washes and to return after a period of two weeks for a check-up examination.

Thanking you for the courtesy of this refer, and hoping to be of further service to Mr. J if possible, I am,

Yours very truly,
A.C. Furstenberg
A.C.Furstenberg, M.D.

JHM/S.

the third day I felt pretty badly. I became cold. I thought I was going to freeze. My wife put me in bed. We had the hot water bottles and about all the blankets we had to cover the bed with on me. It lasted possibly an hour. About three weeks from that time I had another cold spell, and for about six months, I believe, every three weeks, but they kept getting lighter." The physical examination and careful questioning did not reveal any focus of infection, that contributed the toxin that excited the neoplastic change, as is so common in other cases. This case was probably a general viral infection.

After six months no trace of any growth in the mouth or in the neck or under the jaw could be palpated. He remained well until a few years ago. It was reported he died of a stroke. He was then over 80 years old. There was no examination of the body to get the facts, however, only rumor.

In this case, as in so many that are biopsied or operated, the recurrent growths are of higher grade malignancy than when first touched with a knife and that appears to be so here since the recurrence was rapid, and the reactions so intense, though they came three days after the treatment.

SARCOMA OF THE SPLEEN

CASE No. 25 Dr. J. W. Kannel

B. G. was 6 years old when she was attacked with pain in the stomach and some fever. She had been experiencing an increasing difficulty in breathing for some weeks previously. Examination by Dr. J. W. Kannel revealed an enlarged spleen, enlarged axillary and inguinal lymph nodes, and a white cell count of 7,200. A few days later, June 23, 1943, the white cell count rose to 16,700, and on June 24th, it was 22,400. The aspect of the child was that of one very seriously sick. He immediately did an exploratory laparotomy, and found the spleen so greatly enlarged that it had compressed two-thirds of the left lung and had grown out against the intercostal muscles in ridges, so as to leave impressions of the ribs in between. The gross pathology was pathognomonic of sarcoma of the spleen. It would have served as a good museum piece. It was besides very hard and nodular, which is the case with spleen sarcoma. If it were an infection, it would have been soft, and pliable. So the high white cell count was due to reticulo-endothelial disease, comparable to the high lymphocyte count in leukemia. Moreover, no abscess was found to account for the leucocytosis or the fever, which after all are characteristic of acute forms of cancer. Dr. Kannel did not do a biopsy as he felt there was too much risk of causing uncontrollable hemorrhage or embolism, because of the vascular structure of the organ, and because the gross findings were absolutely diagnostic without microscopic aid. Thus, his knowledge saved the patient from possible death resulting from embolism or hemorrhage.

On July 2, 1943, she was given 2 micromicrograms of the SSR and her recovery was gradual and steady until it was complete. The breathing

became normal and so did the breath sounds. The projection of the spleen down into the abdomen 2 inches gradually subsided until it was of normal size. Her health became normal and so remains so far as we know. The last report was in 1956.

One sees that the disease was corrected, and the straying tissues were normalized. The cause was removed right at its point of attack on the vital structure.

LYMPHOSARCOMA CASES

The call for lymphocytes to fight the toxins of chronic infection is standard experience. Though, the neoplastic hyperplasia no doubt had a protective purpose to start with (the reticulo-endothelial system always leads the combat against cancer) this type of hyperplasia does not accomplish any protection. It injures the patient just like the excessive production of poorly evolved thyroid secretion in Case No. I, the toxic nodular goitre case. No doubt the interference with function was a matter of carbonyl group block, as it was in the goitre cases, and so if this is true, the use of the survival synthetic carbonyl remedy should restore the normal functional efficiency of protection, and the normal mitotic process. A few cases of different types of lymphocytic cell lymphosarcoma are given to illustrate the disappearance of neoplastic mitosis, and the restoration of protection. This is seen in the return of the regular health, and disappearance of the pregrowth signs and symptoms similarly in lymphosarcoma, as in the other forms of cancer.

It is the consensus that lymphosarcoma is a generalized disease, and indeed most cases when seen first by us showed generalized tumifaction of all palpable lymph nodes, and increased dullness of the mediastinum and great enlargement of the mesenteric glands. However, there is a type that grows up rapidly in one region without showing great involvement of other areas. This is the type that is most rapidly fatal, and may indeed kill in a few months after the onset. Such a case is the following:

LYMPHOSARCOMA

CASE No. 26 Dr. J. W. Kannel

Miss L. M., age 31, came under Dr. Kannel's care in July, 1925. Physical examination showed a tumor as large as a large orange involving the upper outer quadrant of the right breast and the axilla, which was completely and deeply involved. He removed most of the breast portion, but found the axillary development too deep to extirpate and left it in situs. The microscopic examination showed it to be a malignant lymphosarcoma. This tissue is not derived from the breast tissue itself, but seems to have its origin in lymphoid tissue. Characteristic of this type of lymphosarcoma, cachexia was developing rapidly, so Dr. Kannel prepared her for the Survival Reagent treatment, and gave the

injection on July 16, 1925. The presence of cachexia shows the extremely toxic status of the patient, both from the pathogenic toxin and from the products of the neoplasm intended to be protective, but which were toxic as the thyroid secretion in Case I.

Following the reagent, there was rapid absorption of the axillary and other extensions of the neoplasm, with a simultaneously quick recovery from the cachectic state. She remained well until 1931, when a lump appeared in the left breast the size of one's thumb. It grew rapidly also, as a heavily infiltrating mass. It was removed by Dr. Kannel and diagnosed as a lymphosarcoma from the gross pathological features. She was given another dose of the same reagent, recovered, and remained well thereafter, and on last examination in April, 1946, twenty-one years after treatment, was still found well.

This experience plus the one that follows, shows that the cause of the neoplastic effort may return in the course of years, or maybe was removed by the treatment sufficiently to reverse the neoplastic effort, and still a seed of the cause, — an old infection in an anoxic scar, might have still escaped complete removal. Later on the cause grew to pathogenic proportions, and started trouble again. This means that the therapy should have been repeated a year or so after, or even three years after the first neoplasm was cured to make sure the cause was fully removed. In this case the infected scar was not identified. If it were it could have been removed, and cultured for further information. At any rate, the dose should have been repeated before any further trouble could start. The case shows that the second treatment was just as effective as the first, and no resistance is built up to it.

LYMPHOSARCOMA, LYMPHOCYTE CELL TYPE

CASE No. 27

Mrs. M. S., 38 years old, came under my observation on October 27, 1944. She gave a history of a persistent crop of axillary boils that cleared up on an autogenous vaccine, but no other remedy as antibiotics helped. These appeared in April, 1943, and persisted for nearly a year. During the latter part of this period the right side of her neck became stiff and painful. She could not stand a draft of air on it. Every diligent effort at treatment failed. Instead, swelling and stiffness developed and advanced deeply into the pharynx. In this bewildering condition she stepped on a nail, and sustained a severe infection of the foot. The condition in the neck became much worse then. A mass as big as an apple developed, involving the neck structures on the right side. Biopsy done October 14, 1944, and examined by several good pathologists confirmed the diagnosis of lymphocytic cell type lymphosarcoma.

Two weeks later my examination revealed a marked cachexia, and wide involvement of the palpable lymphatic system, axillary, groin and a large mass behind the umbilicus. It bulged, was hard and fixed, and could be observed

DOWNTOWN CLINICAL LABORATORY

711 STROH BUILDING
DETROIT 26, MICH

CLARENCE I OWEN M D DIRECTOR

NO. B-1589 Oct. 18, 1944

PATIENT Mrs. M S DOCTOR J.M. Jones

SOURCE OF SPECIMEN Neck gland

Gross examination:

The specimen consists of a nodular mass of tissue which measures approximately 2½ cm in size.

Microscopic examination:

The specimen is a lymph node with complete loss of architecture. The lymphoid follicles and germ centers no longer exist. There is a widespread lymphoid hyperplasia with a considerable amount of variation in size of cells although most of them are small. An occasional one is multi-nucleated. The cells have large hyperchromatic nuclei and very little cytoplasm. In some areas there is considerable amount of hyalinization.

Diagnosis:

Lymphosarcoma.

EXAMINED BY C. I. Owen

on inspection. The mass in the neck that was biopsied was 5x7 cms. in its diameters, and had infiltrated the surrounding structures even into the inside of the throat, and bulged greatly on the outside.

She received 2 cc. of the Synthetic Survival Reagent at 11 p.m. that night, and the recovery reactions started at 2 p.m. the next afternoon. Chills, fever and a general achiness as from the grippe accompanied by a relaxing of the stiffness in the neck were evident through the following three weeks. Her cachexia disappeared and her well being was being re-established. All of the neoplastic masses improved in the same way. By the end of the third week the stiffness and swallowing difficulty was fully overcome. In three months no more tumors could be palpated. However, her reactions were repeated at the regular periods of three months, that is the 24th, the 36th, the 72nd, the 84th, and 96th weeks and even later, and her health improved after each, in spite of the fact that her health had become much better than was normal for her, even as early as the twelfth week. Besides taking care of a large house and her family, she was able to work in a clothing shop, and carry on her social affairs. Following

the absorption of the growths there was no sudden reaction in the old site of the boils or the foot infection as would be expected had they contributed the etiological agent. Instead, she showed a reaction in the tonsil area that presented the characteristics of a keloid. This came ten years after the cure of the lymphosarcoma. She was given a different remedy at this time, diphenoquinone, but its action was too slow. Within a year a pain in the dorsal spine developed as she had had for many years off and on since an automobile accident some twenty years earlier. The radiographic studies by a well respected expert gave a diagnosis of an old lesion of maybe thirty years' standing. There was a history of suspected tuberculosis in early life, so, as a keloid is a response to tubercular toxins, and the old bone lesion that resembled a tubercular affair became evident as a reaction, our conclusion was that the lymphosarcoma was caused by the toxins of an old tuberculosis, that had been suppressed.

The writer's services were not available to her at the time, and she yielded to the persuasions of a radiologist, and took intensive X-ray over the spine and the neck. In August 1959 she died.

In this case a therapeutic fact was learned as in the former case of lymphosarcoma. Since lymphomas are primary protective in their intentions, one should give repeated doses every few years a few times until there is no vestige of the exciting cause left in the system. Had this been done, this patient probably would have had no more trouble.

LYMPHOSARCOMA, LYMPHOCYTIC CELL TYPE

CASE No. 28

Mrs. G. G., age 40, at the time of treatment with the Survival Reagent on May 17, 1937, came for treatment of a rapidly developing mass on the back of her neck, toward the right side. A biopsy had been made of an enlarging gland in that location three weeks previously, when it started to grow rapidly. The microscopic diagnosis was lymphocytic cell lymphosarcoma. At the time of our examination, the scar region had become tumorous and several smaller tumors had also developed in the area. The largest was the size of the ball of one's thumb, or an English walnut, but was deeply infiltrated. She was ill and toxic, even in spite of the cleansing regime she had followed for a week before the treatment was given. Two cc. of the 10^{-12} concentration of the Survival reagent was injected in the upper arm. At her visit four weeks later the whole area had normalized. No vestige of any neoplasms could be found. Her normal health had also returned and she remained well until she was killed in an automobile accident seven years later. An autopsy established that there was no trace of any neoplasm to be found, so we assume she was cured even without a repetition of the dose. The exciting cause was not identified in this case and no one knows if or not some interesting manifestation would have showed up later had she not had the accident.

c o p y

DIAGNOSTIC LABORATORIES
MIAMI VALLEY HOSPITAL
DAYTON, OHIO

SURGICAL PATHOLOGY

Name G , George Mrs. (Last Name / First Name / Initial) Path. No. 950-K

Station C.P. Room Age 45? Public / Private

Clinical Diagnosis
(Must be stated by surgeon before operation)
Gland from neck

Surgeon's Pathology
(Must be described by surgeon following operation)

Surgeon P. Shank

Date of Operation 4-27-37

PATHOLOGIST'S REPORT

Gross pathology

Cherry size mass of firm grayish-white tissue

Microscopic Examination

The normal lymphnode architecture is largely replaced by diffuse hyperplasia, including localized areas containing large pale lymphoblasts. The microscopic appearances are those of early lymphoblastoma of the lymphosarcoma type. (Does the peripheral blood show evidence of an excessive number of abnormal immature white cells? Such histologic findings in the lymphnodes may or may not be associated with leukemia).

Walter [illegible] Simpson, M.D. *Pathologist.*
(The original sheet is to be placed on the patient's chart)

CANCER OF THE BREAST

The resting breast is especially prone to take on malignant change. It has plenty of FCG structures, but they are inactive and not carrying on the oxidative functions of a working tissue. The circulation has no aid as occurs in muscle containing organs where contractions pump the blood along and move the lymph, thus aiding the metabolic exchanges and extending the equilibrium point of enzymatic reactions. A blow on the breast therefore produces a dangerous injury that can interrupt the circulation and create an anoxic area much easier than in a tissue with an active circulation. Blows need not be very hard to bring about the unfavorable change, and indeed after the tumor has developed the trauma incident to vigorous physical examination by palpation may step up the malignancy of the lesion most dangerously. For this reason America's great diagnostician, Cullen, recommends very clever means of inspection for demonstrating the malignant infiltrations, and thus avoids the need of adverse amount of palpation. Great pressure tugging on the growth, etc., are vigorously condemned. Even the needle biopsy is considered dangerous by

Cullen. Great care, then, is used in making the examination in breast cases. The various types are so characteristic that in most cases inspection alone should settle the diagnosis, when the growth is well established.

To return the breast cancer cell to a normal functional status when it is not at work because of the normal lactogenic stimuli, seems a little paradoxical. However, the restoration of the FCG does not necessarily mean it has to produce milk, but only that its impediment that blocks its dehydrogenating power is removed. When the energy producing and energy receiving carbonyl groups are freed and able to function normally, no energy is shunted vicariously into the mitotic mechanisms to produce neoplasia.

In breast cases the great problem is early diagnosis to give the surgeon a chance, but as the vast majority of cases are well metastasized before the lump is discovered, its early total removal by simple resection is the logical procedure to give the patient every possible chance, and then correct the Survival chemistry and teach the victim how to live.

When the breast case is under treatment the affected area must be kept warm. Internal cancer has a better chance to recover because of the natural normal temperature that is sustained. Breast cancer must be well covered, and must also be protected from strong sunlight, perfumes and powders women usually use. Then the examinations must be made with care as the fine blood vessels that grow up into the coagulated and digesting mass are indeed very delicate, and are easily injured. Hemorrhage at the point of vascular rupture complicates the situation as the circulation is cut off and an excellent medium for infection is created. The continuance of the digestive process is blocked also, and one has a bad situation that may limit the recovery process at this point of injury, and indeed may prove to be the starting point for a reversal of the recovery. The patient's responsibility for protecting the breast is a matter that must be emphasized. Of course, the physicians of experience will know what type of patient needs this advice most and will illustrate the need of care by his own manner of examination with care.

CARCINOMA SIMPLEX

CASE No. 29 Prof. R. S. L.

Miss C. F., age 50 years, Brazilian, referred by Prof. A. P. on November 17, 1941, was suffering with a painful tumor of the right breast for six months. It was considered inoperable, and 12 applications of deep X-ray therapy were given. It continued to grow and become more painful. The nipple was already retracted, and the skin hard and infiltrated, and the whole mass was fixed to the chest wall, making it immovable to touch, or by change of position as on stooping or leaning to the side. It had become the size of a large fist. Two micrograms of parabenzoquinone were given on November 17, 1941, intramus-

cularly. She recovered gradually. But six months later the dose was repeated. One year after treatment no tumor could be found, and she was considered clinically cured.

SCIRRHUS CANCER OF BREAST

CASE No. 30 Prof. R. S. L.

Mrs. M. S. was 42 years of age, Portugese, and married. She found a lump in the breast in 1938, when living in Lisbon, Portugal. It was removed surgically and diagnosed cancer microscopically. Recurrence was well advanced in 1941 as a large tumor in the same location had developed as early as April, but she was not treated until on October 14, 1941. She was given two micrograms of benzoquinone solution intramuscularly. Examination seven months later showed that the tumor had entirely disappeared leaving a scar isolated and no larger than a grape. Her general condition was excellent.

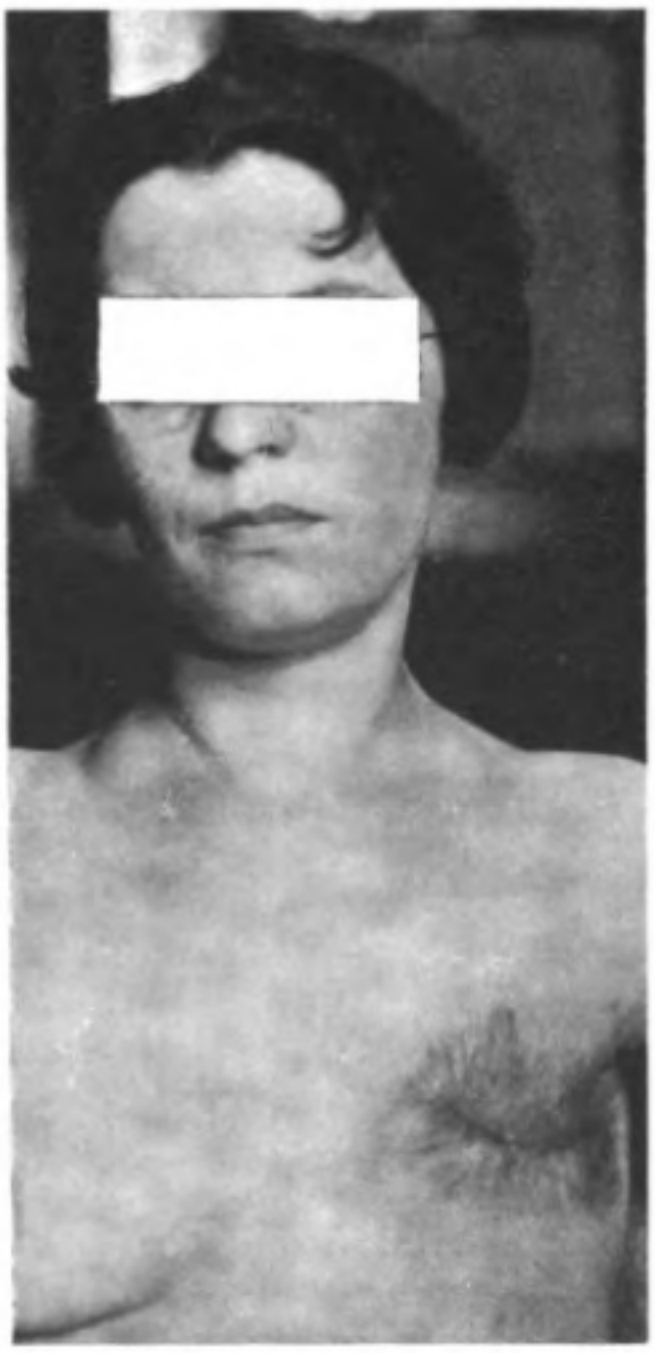
In the writer's experience, the hard calcified residues of digestion of the tumor may be removed for microscopic study. They are found to be composed of calcareous material and some dense fibrous connective tissue without epithelial structure. Right after the malignant status is corrected and calcification starts they are subjects for removal, vascular ingrowth and may be compared with a blood clot, or of the casein in milk, that must undergo calcification as the first step in its digestion. This is shown in Plate No. I. Then after the digestion and absorption have become complete stones of the type described in this case may still remain. We have never seen them go malignant again, but their absorption is very slow. Whether they give any protection against cancer is not established though we thought for a long time that they did.

METASTATIC CANCER OF THE BREAST

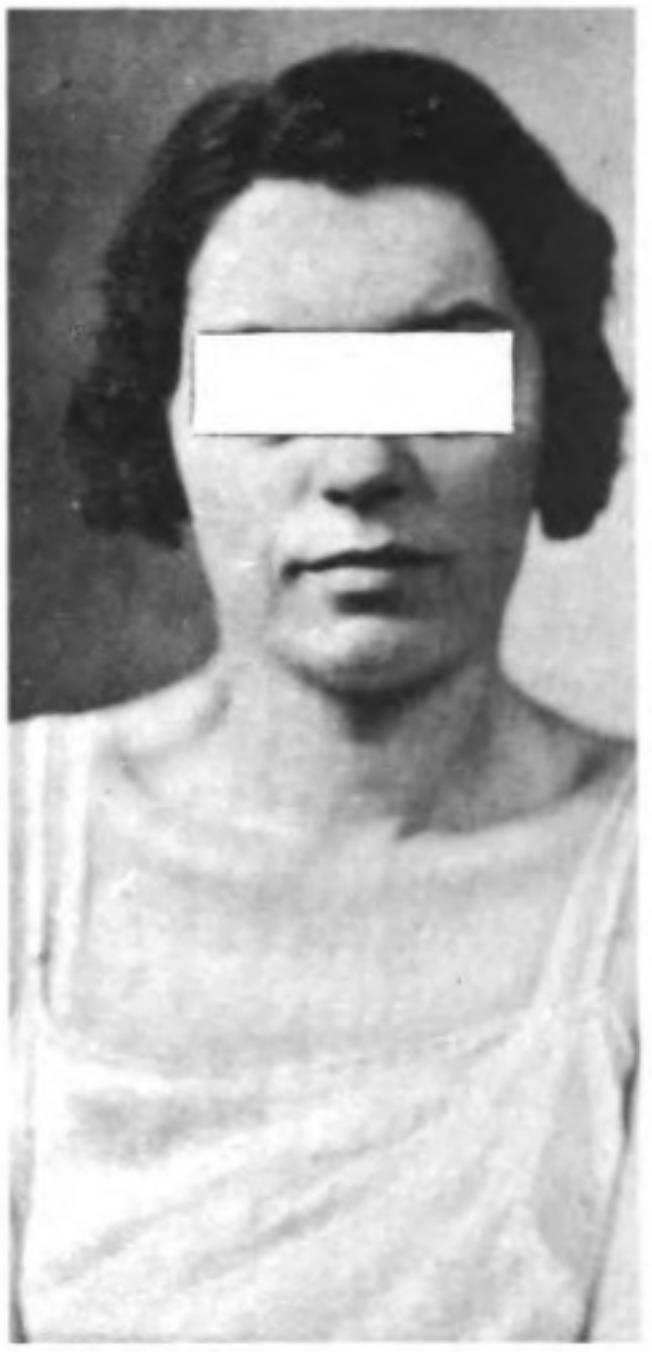
CASE No. 31

Miss H. P., age 32, Canadian, was treated with the Serial System of Carbonyl groups with free radical terminals in 1927. She had been operated and the microscopic study showed a grade 4 malignancy of the carcinoma simplex type. It had infiltrated the axilla and quickly showed renewed activity there and it had also metastasized to the lungs and over to the right supraclavicular space as a good sized (half egg) fixed tumor. The dyspnoea and chest signs showed heavy involvement of the mediastinum and cough was incessant.

Six months after the treatment there were no more symptoms or signs of the disease left. The supraclavicular space was clear, and the chest examination indicated the metastases had been absorbed. The photographs made at the time of treatment and after recovery. In 1955 we heard from her again, that she is in perfect health and no signs of recurrence have appeared.



Miss H. P.
Before Treatment.

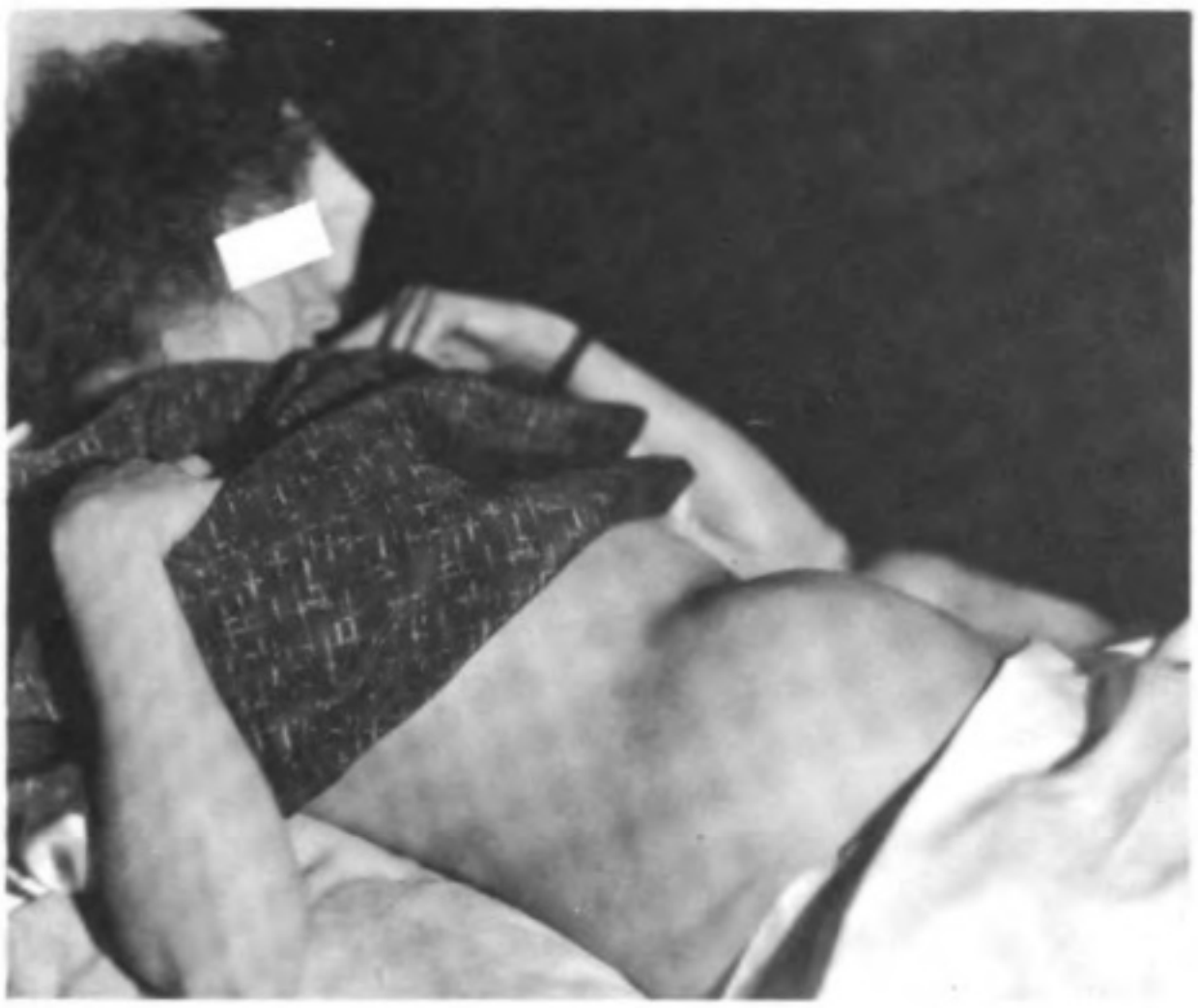


Miss H. P.
After Treatment.

FIBROMA OF UTERUS

CASE No. 32

Miss G., age 45 in December, 1930, when first treated. These are interesting cases, exceedingly slow to recover, often requiring a year or two to be free of the growth. The most intriguing feature in such cases is that the toxin that causes cancer is fibrogenic, that is, it stimulates fibrosis as we explain later on, and when the fibroblastic response peters out neoplasia is due to start. The protection offered by fibrogenesis probably is due to the integration of the pathogen with the fibroblastic tissue and its neutralization in that way, so that when fibrogenesis fails, the toxin must add to some other cell structure and this seems to be the mitotic mechanism. In this connection it is significant that neoplasms came later on in such cases. The photographs tell the story in this case. The second picture was taken after the absorption was complete. It is 30 years since her treatment was given, and she has not developed any cancer as yet. The photographs were part of the evidence in the U. S. Federal Court.



Photograph I, before treatment.

Photograph II, after treatment.
The recovery was complete, no vestige of the growth can be found.

CHAPTER XVI

THE TERMINATION OF THE MALIGNANT PHASE, THE CONSTITUTIONAL NATURE OF CANCER AND OF THE SURVIVAL FACTOR

We have considerable data on the speed with which the malignant state turns to normal, or is corrected, and the cancer cells are digested and disappear. The biopsy photo in Plate I shows the calcification and organization of a squamous cell cancer of the skin within a month after treatment. However, complete microscopic disappearance may take place in less than two weeks as is revealed in the report of Dr. E., a prominent internist, whose daughter was found to have a grade III squamous cell cancer of the cervix uteri that was deemed inoperable from its state of advancement. She was given a dose of the SSR on June 2, 1957. On June 14th examination showed that all of the evidences of the disease had disappeared. There were no extensions into the adnexia and the cervix appeared normal and showed a normal texture. A series of biopsies were then taken from different parts of the cervix to see if any cancer cells could be found on serial sectioning. Every biopsy proved negative. She has remained free of any sign or symptom to date. This is the second case of cancer cured in this physician's family, both cancers confirmed by biopsy, and the cure fully established.

Twelve days is a short period when one thinks of the long time it takes for a cancer to reach recognizable features, and when one thinks of the years of the pregrowth toxic period that is characteristic. However, we have often observed that the last expression of the recovery process is an acute inflammation of some old focus of infection, as the tonsil on the same side as the breast under treatment for cancer, After the growth disappears from the axilla and the breast, the tonsil or a scar located where an infection once existed, lights up with an acute inflammation much like the inflammation that existed there years ago, long before the breast growth came. In about a week the sore throat or other inflammation normalizes and then the tonsil that was deeply scarred or calcified and fixed becomes loose, and pliable. We take this event to mean that the infection that gave rise to the carcinogen was still present in a partly asphyxiated state elaborating its toxins which polymerized until they reached a carcinogenic structure, and during the recovery process, stepwise oxidation of the polymer tore it down through the various stages by which it had been poisoning the patient until it could produce cancer. Now, however, it had reached the monomeric form as produced by the captive germ, and while it was being burned out of the way with its imprisoning scar, this inflammation was going on. This event is the cleaning out of the pathogen of the very inception of the disease. It must be also recalled that each act of the correction process is going on independently in the different cells and regions concerned,

and these may occur in parts of the body that are far apart. This is not only because the metastases are spread all about, but because of the distance of the "primary" lesion, the focus of infection that was able to brew the carcinogen because of its hypoxia. One would have to say then that the case is not constitutionally cured until this primary focus was eliminated. However, locally at the site of the cancer growths, wherever they occur, the cure may be complete as shown by the biopsies taken as in the case just mentioned, even in less than two weeks, and is confirmable in five years as in the case of Mrs. M. W., Case No. 11, by radical biopsy procedures.

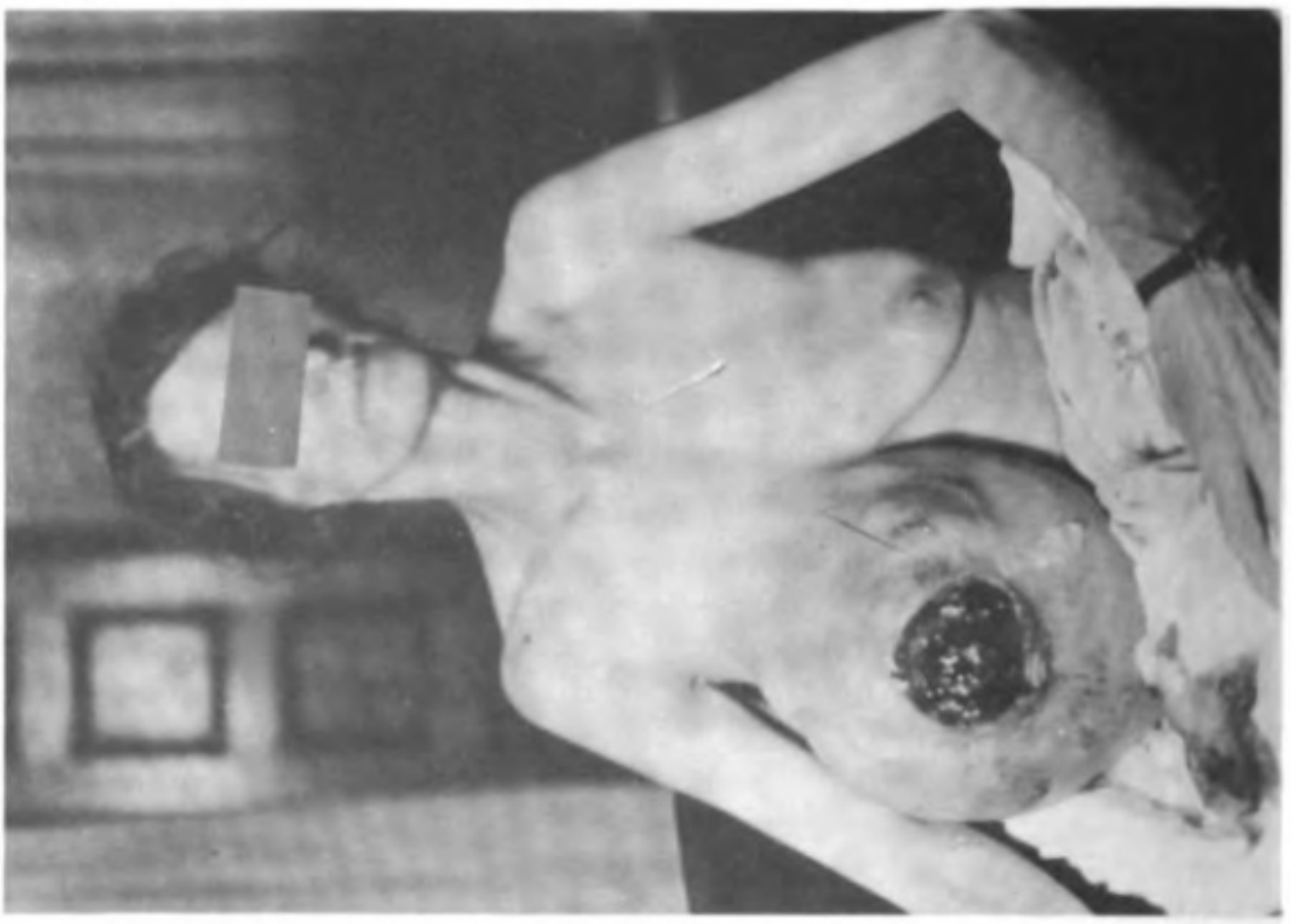
However, getting the pathogen out of the way does not mean that some day another may not come along and start trouble again, that is, a new cancer develops or some other fatal condition comes about as in case No. 11, five years after being cured.

The speed of recovery has an additional significance. It is the last feature of the disease that regularly disappears first in the reversal of the disease process and this is the malignant change. But as each region that plays a part in the disease is also undergoing recovery at the hands of the SSR independently, the events in the last tumors to come have no direct influence on the events of the first tumors, or of the primary lesion, the scar where the pathogen is brewed. To test out this proposition we waited for some cases to show the need of getting rid of the necrotic material because of its toxicity and bad odor. The cases shown in the photographs demonstrate that after the SSR starts its work and the absorption of the corrected cells is well under way, manipulation which would regularly stimulate an untreated cancer to metastasize and grow faster, and kill quicker, has no such effect. So the recovery process continues on in the extensions as well as in the primary growth and even in the focus of infection that gave rise to the carcinogen. Both of these patients were permanently cured. The breast case visited in Detroit ten years later and was examined as thoroughly as possible physically. She was found completely well so far as one could tell. In the other case, the melanotic extensions of the growth continued to be absorbed and disappear, while the non-malignant pigmented moles remained unaltered. The independent correction course in each affected cell is thus demonstrated. The time element also speaks for the correctness of polymerization process we assign to the pathogen.

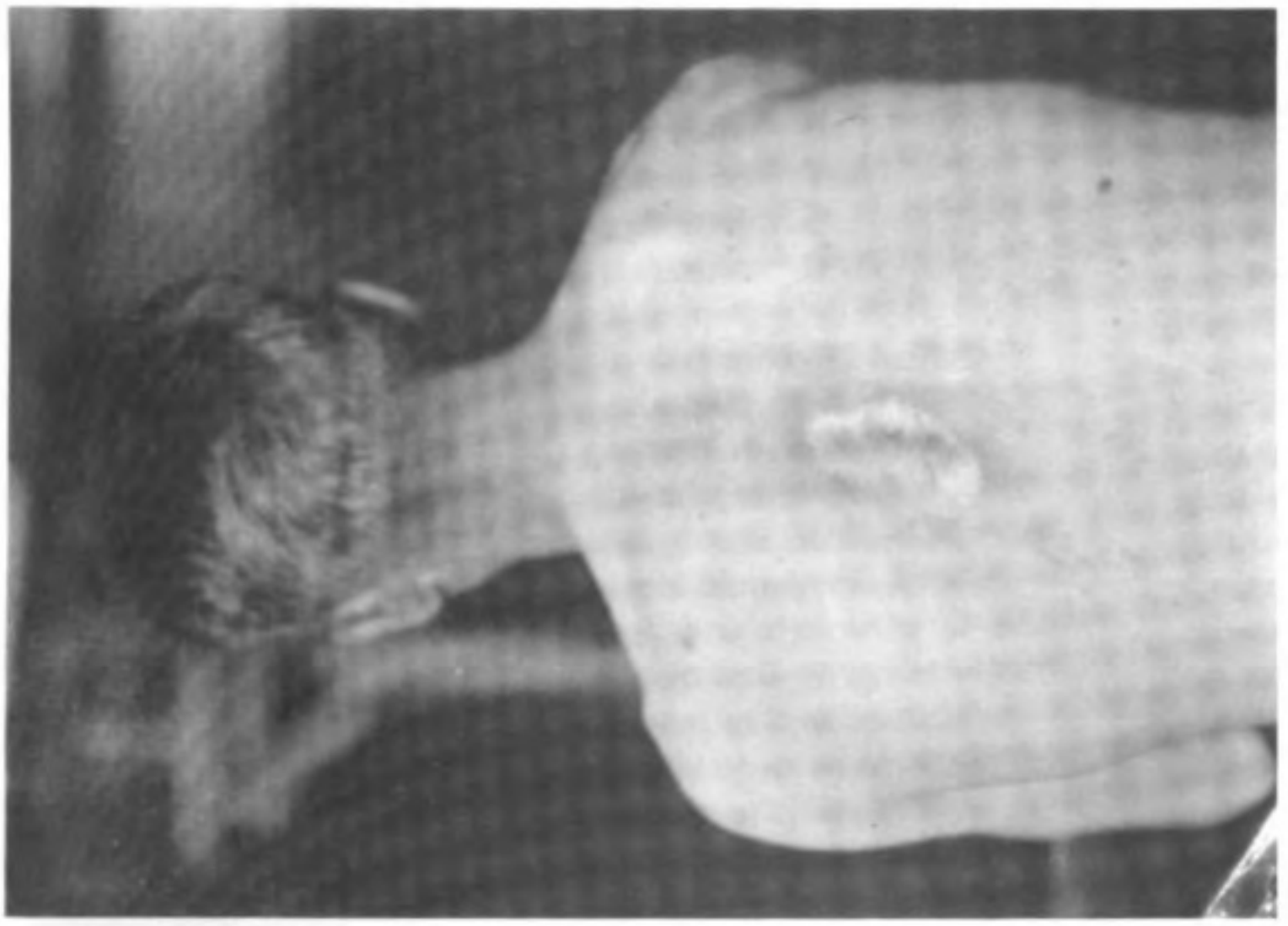
In considering the constitutional nature of cancer and of its correction, one must recall that when cancer is induced by applying chemical carcinogens, the lesion does not show malignancy until the applied pathogen is no longer detectable in the cells undergoing malignant change. It can not even be detected by spectography, according to Peacock. Neither can it be detected in the blood stream or in the other tissues of the body. It is our opinion that it is integrated with the cancer cell under the alteration we assign to it. Hence, it would show an entirely different spectrographic character, if it showed any at all when combined with the cell grana.

This situation should be compared with the clinical fact that while the recovery from cancer is progressing ideally, no trace of the injected SSR can be

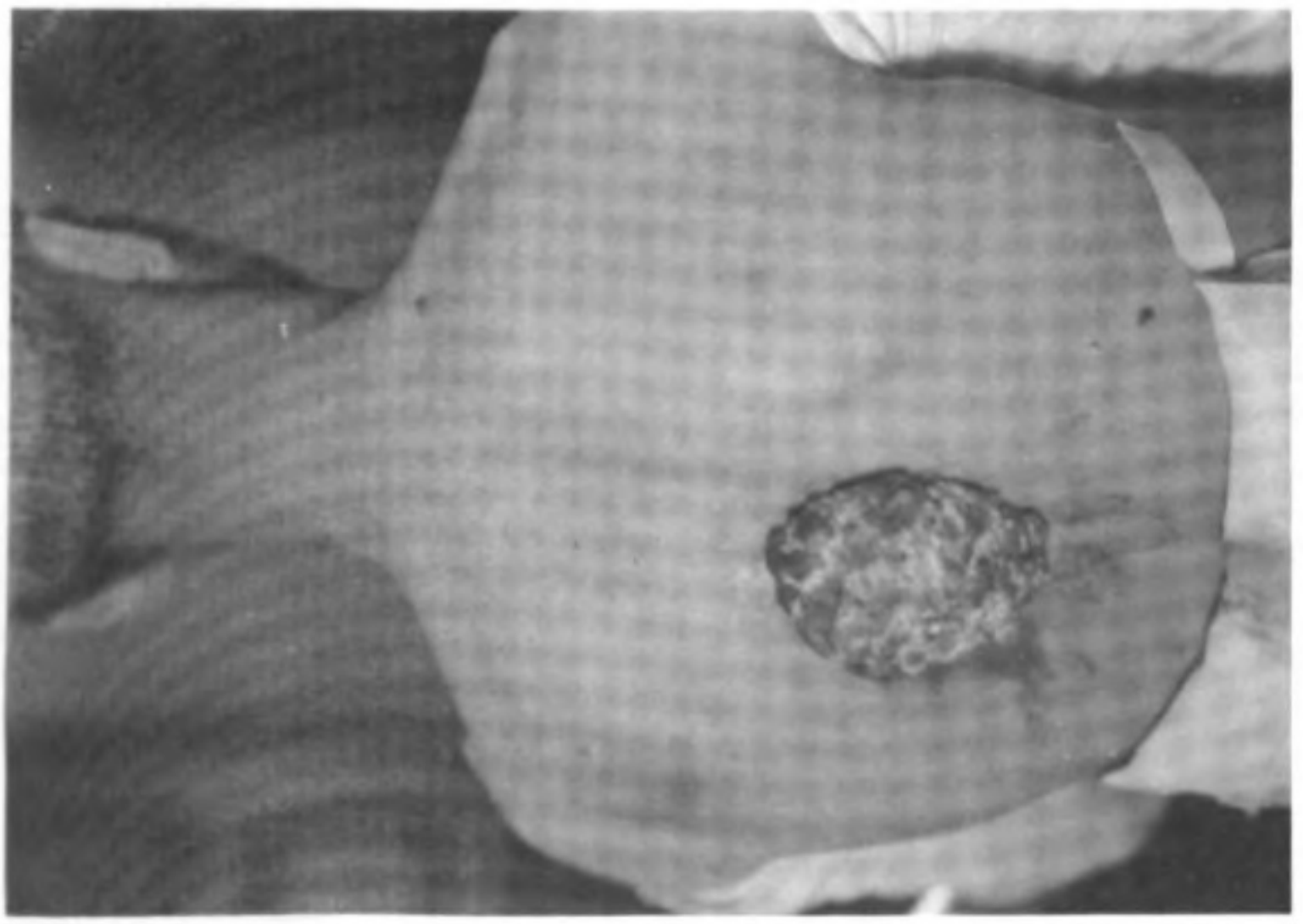
Miss N. after complete recovery.



Miss N. after the growth started undergoing digestion after the carbonyl catalysts were given, and before crude removal was done.



Mr. L. after complete recovery.



Mr. L. after treatment when the growths were undergoing digestion just before crude removal.

identified in the blood stream or other tissues than those where the curative process is going on. And here it is identified only by the curative change and not by any spectrographic or other tests. To illustrate a case under treatment by Dr. Treiger is cited. This patient, age 38 years, developed a carcinoma simplex of the left breast. It was considered inoperable by the referring surgeon as the whole breast was involved including the axillary glands, and the chest wall was invaded so as to cause pleural pain. The nipple was completely retracted and the skin invaded so as to show the "pig-skin" appearance, indicating the high grade malignancy, mucinous grade III adenocarcinoma, on biopsy. Seven weeks after one injection of the SSR, and after several strong reactions, the tumor had reduced to the size of a small egg or big plum, was loose, the axilla was clear, the skin normal and the nipple out in normal fashion. There was no more pain and her health had returned very satisfactorily. One would think that she held an excess of the synthetic survival factor reagent circulating in her blood, just as one would think that the progress of the cancer growth depended upon new pathogen attacking non-malignant cells. But this is not the case. It is likely that the pathogen-malignant-cell-integrate is established at the start and is a localized affair, and the reproductions carry the pathogen with the new generations of cells forward as metastases. The initial foci may be multiple, however, and the virus may multiply within the cancer cells and infect normal cells nearby when the parent host cell disintegrates in its death. In the untreated case the death of each cancer cell would then supply virus for the infection of many other cells and cause them to go malignant. One occasionally sees an "independent" adenocarcinoma of gastric mucosa origin spring up in a case of squamous cell cancer of the cervix, and both cancers recover under the same treatment.

The case mentioned shows that there are no excess molecules of the SSR in the blood stream seven weeks after the treatment is given. The family of this patient all came down with mumps at the same time. She did also and it ran the regular course of eight days. Had there been excess SSR in the blood, it would not have developed, or would have cured up in hours, as mumps regularly does if treated early. The recovery from the cancer, however, went on uninterrupted. We consider this situation important to the management of the case and to the understanding of the recovery process. Whatever SSR was left in the system after the recovery is well under way is in the diseased tissue, and the infectious lesion that gave rise to the toxin that started the trouble, where it mediates the burning of the toxin polymere down through the initial monomeric form until no more is left as a chain process. That is what the clinical data shows. The constitutional quality rests in the fact that the lesion that sends the pathogen into the blood stream is generally at some distance from the injured area of anoxia or hypoxia where the pathogen has the chance to integrate with the cells injured by the anoxia, and in the early widely spreading metastases. The rest of the body and particularly the reticulo-endothelial system, may be injured by the pathogen in many ways for years as it is polymerizing.

CHAPTER XVII

VIRAL INFECTIONS

It will be recalled that within a minute and a half after a virus has penetrated the host cell, the integration is so firmly set that no amount of vaccine, immune serum or antitoxin can dislodge the virus and rescue the host cell. It is doomed. On the other hand, it is our chore to show that any time after the virus has penetrated and integrated with the host cell, so long as the latter is still alive, it is possible to accomplish the separation in a way that leaves the host cell in good functional status, while the virus is no longer to be found. In fact, the atomic bondings that constitute the integration, according to our postulate, actually invite and provide for the oxidative separation.

To prove this the best field of observation is the paralytic viral diseases. When a nerve cell is integrated with a virus its functional mechanism is paralyzed, and it has to die. This will be soon if the integration is lytic, but death can be postponed if the integration is symbiotic. However, as soon as the integration has taken place, function is stopped. Therefore, after paralysis is demonstrated, its restoration means two things. It means that the virus is no longer integrated with the cell, and it means the destructive changes leading toward the death of the cell, and which supported the viral vegetation, have been repaired. It also means, since the integration prevents host cell function and vital activities, that the energy required for the reconstruction came from some other source than its grana and enzyme activities. The only other source of energy during the integration must be the oxidation of the virus. So while it is possible to burn off the virus at its point of attachment, in line with our postulate, it is indicated that the oxidation of the virus is stepwise, starting in the last units laid down during its vegetation, and ending up at the point of attachment. Thus the whole energy taken from the host cell to accomplish the vegetation, is returned for host cell reconstruction. This will appear especially in the cure of rabies, because the time relations are so clean cut.

We chose a variety of paralytic viral infections that are 100% fatal to the host cell, and three of them, 100% fatal to the host as well. Thus the data presented should be substantial proof of the correctness and certainly of the utility of our postulate. Somatic cell infection by virus as the 100% fatal hog cholera, and the picturesque epidemic hepatitis are also used.

ANTERIOR POLIOMYELITIS

Infantile Paralysis (Polio)

In the cases to be presented there are the two types, the lytic and the symbiotic. However, no case is purely of one type; they are mixed with one form or other predominating. Why a virus makes a lytic type destruction in

one case and a symbiotic type of integration in another, or why a virus having made a symbiotic type integration suddenly goes lytic is not explained so far as I know. However, since the same virus type is concerned in any epidemic, the variant is probably the patient or some change in the patient's host cell. The effect of the symbiotic integration is paralysis and atrophy of dependent nerves and muscles so that the invalidism can not be distinguished as to whether a symbiosis exists or if the nerve cells are dead and not restorable. We will show that the symbiotic type can cause extensive paralysis and atrophy for many years, and still both the host cell and the virus are still alive and so firmly integrated that neither shows any sign of life. To all intents and purposes they are dead. Yet the virus can be oxidized off and the energy evolved during the process will still support the host cell reconstruction so it can function again. Naturally in any case where a symbiotic integration has existed for 20 years, and produced extensive paralysis and atrophy, some of the host nerve cells have had a chance to die, and the restoration will not be 100%, but we can show a good 90% or 95% restoration of function and muscle reconstruction which means host nerve cell reconstruction after 20 years of invalidism of this kind. To solve the puzzle as to why the integration is lytic in one case and symbiotic in the other the situation in the host cell can be postulated or assumed, but since no practical aid is had by this type of discussion, it is best to defer it for future experimentation.

Each case to be presented shows a determinative pretreatment control period showing that the virus actually has integrated with the host cell and has started the latter's destruction to support its own vegetation. It will be seen that the time required for restoration is proportional to the time required for the amount of destruction done. So in all of the cases nerve cell destruction was started, and in all, the reversal of the pathogenesis was accomplished. The time ratio is not the same as in the rabies cases or distemper cases cured by the same reagent. Thus a difference in species of host cell or of the virus is a factor to be studied.

RECOVERY FROM CHRONIC SYMBIOTIC TYPE PARALYSIS AND ATROPHY ESTABLISHED THREE YEARS

CASE No. 33 Dr. Julian Baldor

Myrna R., age 10, presented an atrophy of three years' standing with complete paralysis of the left leg from the waist to the toes. The leg was too weak and atrophied to wear a brace. There was also a contracture of one toe of the paralyzed foot. The calf measured four inches (10 cm.). The rest of the body was normal. She had been at Warm Springs Foundation in Georgia but they decided they could not help her and sent her home without improvement.

The Synthetic Reagent (SSR) was injected February 11, 1944. Following this treatment there were two reactions with pain spreading from the head,

down the back to involve the left leg. These came the third and sixth weeks. Following each, there was noticeable improvement. The muscles began to regenerate and motion began to be restored. By the end of the twelfth week she could walk with the leg very well and play with other children. The muscle reconstruction was practically complete, for one could not tell by observation which was the affected leg. She later took up toe dancing. The third and sixth week reactions are characteristic of recovery from chronic infection.

The history in this case showed a sudden overwhelming infection with instantaneous full paralysis of the whole leg. This is characteristic of the symbiotic type of infection, and is borne out by the recovery reactions that came the third and sixth weeks. The following case showed the same characteristics.

Therefore, where the paralysis is sudden and complete in the affected area, one may suspect the symbiotic type of integration and also a hope of securing improvement years after the acute stage has passed.

RECOVERY FROM CHRONIC POLIOMYELITIS WITH PARALYSIS AND ATROPHY ESTABLISHED OVER TWENTY YEARS

CASE No. 34 Dr. Wendell Hendricks

Mrs. V. N., age 23, was first observed April 5, 1943. She was carried into the office by her husband as she was not able to walk because of poliomyelitis since the age of one and one-half years. All efforts were made with braces, casts, operations to fix the joints and shorten tendons, but to no avail. Both legs were atrophied and paralyzed completely, from the waist to the toes. The right leg was 4 cms. shorter than the left. The circumference of the calf of the right leg was 4 inches (10 cms). The left calf was 10 inches in circumference, but equally useless. She stayed in bed most of the time or was in a wheel chair, but with the aid of crutches and steel braces from hips to toes she could swing herself about inside the house and stand as per 3 point suspension. During the last two years of the invalidism the contraction of the fibrous tissue where muscles should be, caused the legs to cramp up stiffly so the braces were not comfortable and she did without them, staying in bed most of the time or swinging about with the crutches and sitting in the wheel chair. She never had voluntary control of any of the muscles, and never walked. There were several operations aimed at stiffening the legs but they gave no help. She had continuous migraines, besides. Such was the pretreatment control period, paralysis atrophy and finally the contractures the ultimate result of the paralysis and atrophy. There were some vestiges of muscle fibers present even though in the atrophied state and paralyzed for want of nerve impulses. No one knew at the time of treatment if or not the nerve cell bodies in the cord were dead or blocked and also atrophied by the integration with the virus. The results of treatment proved that the latter situation prevailed. On April 7, the Synthetic Reagent

(SSR) was given and by June 8, that is in about nine weeks, there was motion and visible development of muscles in the right leg. She could also stand by herself without help.

On August 13, 1943, she had a reaction of chills, fever, and headache. Following this reaction complete control of both legs developed steadily. There was much muscle restoration. She walked about unaided without crutches or braces. The improvement continued in all respects. She could do her housework and adopted a baby. On June 12, 1944, she had another reaction with pain in her right foot and thigh and some fever. The migraine still persisted so another injection of the SSR was given on June 23, and again November 14, 1944. At that time the migraine was still present at times; however, the right leg grew so as to be only one-half inch shorter than the left. The circumference of the calf of the right leg was then 10 inches, or 25 cm., and the left calf, 11½ inches, or 28 cm. She was able to run up and down stairs and walk about or drive her automobile like any other person. She clerks in a store, is on her feet all day and requires no aid whatever. The migraine disappeared in 1944, and her health seems fully restored. This case illustrates the principles of our working hypothesis throughout.

The recovery reactions noted in this case are similar to those observed during the recovery from cancer and tuberculosis and others with seriously damaged tissues. In this case they took place where the state of paralyzing symbiosis was established over twenty years.

ADVANCED BULBAR POLIOMYELITIS WITH RESPIRATORY PARALYSIS

CASE No. 35 D. H. Arnott, M.D.

John K., a boy of 16, was in the acute stage of Landry's ascending type of paralysis. It started in the right leg and within a week involved the other leg, the arms, torso, neck, swallowing muscles, and respiratory muscles; and oculomotor nerves were paralyzed on the right side also and he was unconscious when he was first seen at a cottage near Port Huron, Michigan, during the polio epidemic in August, 1934. He was cyanotic and appeared to be dead except for a faint heart beat. There were no respiratory excursions nor signs of breathing that could be distinguished. The abdomen was blown up. The abdominal muscles were relaxed in a flaccid paralysis. Later we learned that the bowel and urinary bladder were also paralyzed. The (SSR) was given and within a few minutes there was some respiratory movement, the flaccid abdominal muscles contracted some and the cyanosis started to leave. Within twelve hours he could move his arms and respiration was well established. The neck and eye and swallowing paralysis had left and speech returned. The left leg showed improvement also and some of the back muscles demonstrated a return of tonicity. So far as we could learn the pathogenesis was reversed in the order in which it developed. Two accidental matters intercepted a fine recovery. The cook brought him a hot cup of tea the next morning. *Within*

an hour a reversal of the recovery set in with great rapidity, and it took several days for him to regain his status before the tea was taken. After a week of improvement he was taken in an ambulance some eighty miles. The trip was too much for him, for after being removed from the ambulance he had a general convulsion in which all of his muscles took part. This showed that the nerve cells had regained their function. Yet it proved disastrous, for they seemed to be burned out and the relapse took twelve weeks for function to return to the affected muscles. Satisfactory restoration of muscle development and control required about two years. He is well now except for some 30 per cent atrophy of his right transversalis muscle, and the quadraceps extensor of the right leg shows a 50 per cent atrophy. This does not impede walking, but it weakens his ability to climb stairs.

It is evident from this case that the recovery results will be determined by the care received, as well as by the length of time the disease is established. It also shows that the oxidation catalysis reverses the disease process immediately but that the recovery process can be upset by being thrown out of balance by physical and chemical means. Yet once it has started it will reassert itself and partly at least overcome the impeding factor. Thus the recovery process behaves like a chain reaction.

One observes the dominance of the lytic type of integration of the virus with the host cell here in the steady spread of the disease, in the face of exhaustion of Nissl substance consequent to exposure and intense effort fighting a storm on Lake Huron in a small sail boat. The nerve cells were all open to infection directly and as fast as virus was produced it found a host in each successive layer of motor nerve cells. Here the culture predominated over the virus production and each cell took a minimum of infective agent, in contrast to the symbiotic type where the infected cells were each flooded with an abundance of virus all at once, and the hydrolytic as well as the oxidative glycolysis systems were excluded from material energy production.

ACUTE BULBAR POLIOMYELITIS

CASE No. 36 Dr. Julian Baldor

Patient: Sandra F., aged 9 years, female, student, admitted September 19, 1951, 7:30 p.m.

Exposure: Brother seven years old, died of acute bulbar poliomyelitis proven by autopsy (two days before), September 17, 1951, State Board of Health Certificate No. 3920. He presented the same symptomatology as Sandra, and died shortly after reaching the state in which Sandra was when she received the treatment by the oxidation catalysts.

Personal History: Measles, Chicken Pox, at five years of age.

Present Ailment: Temperature 102°F. for the past four days, with nausea, severe occipital headaches, and extreme difficulty in breathing. She was required to raise her shoulders at each attempt to breathe, to get some chest motion.

This suggests a diaphragmatic paralysis was present as well as the intercostal muscle paralysis. Abdominal excursions not palpable.

Physical Examination: Both patellar reflexes were practically abolished, although extremely weak motion was still elicitable. B.P. 100/75. Because of the respiratory failure, no further time was given to the physical examination, and the Synthetic Reagent was injected immediately in a dilution of one to a trillion, of water, two cubic centimeters by volume, into the gluteal muscles.

Progress: Twelve hours later a reaction occurred in a n.gative phase with more intensive headaches and nausea. The temperature remained the same. There was no change in leg function, voluntary or reflex.

Seventy-two hours later she went into a semi-coma which lasted three days. In this period the temperature and respiration became normal, and she emerged from the stupor with normal cerebral functions, also. However, there was paralysis of the muscles of the back and of the left arm and both legs, which cleared up within one week, except that some weakness remained in the legs and left arm. This difficulty became nearly well within three months so that a slight drag in the left leg remained, which did not hinder her walking without the aid of braces, etc. Speech, vision, hearing, and alertness tests, showed 100 per cent recovery. *Memory* and other cerebral functions were found to be normal.

During the past two years the weakness of the left leg has not changed very much for the better but persists as a slight incoordination, according to Dr. Baldor.

Remarks: A residual, very slight atrophy and corresponding slight flaccid paralysis of the right rectus anterior with very slight atrophy in muscles of left hand are still retained in spite of a third dose given in July, 1952. This shows that the injury is spinal and more or less permanent. In checking accounts in this case, the full cerebral recovery plus the timely cure of the respiratory paralysis without the use of an "iron lung," and the retention of the minor spinal injury in such a virulent case of bulbar paralysis, speak well for the method of treatment. Had the patient had time for a thorough intestinal lavage and the elimination of interfering drugs like aspirin, even the spinal injury may have been avoided.

POLIO WITH PARALYSIS

CASE No. 37 Dr. George Franklin Smith

This case is given since differentiation between anterior poliomyelitis and encephalitis is not always easy in babies without waiting for the symptoms to develop further defects. The treatment was given to avoid further injury and possible death, and the recovery shows that the choice was good practice no matter which diagnosis was applicable. The fact that the paralysis became flaccid without voluntary motion or reflexes point to anterior poliomyelitis of

the most dangerous type, where integration with the nerve cell functional material was well established, and sequelae would be expected to be extensive had not the treatment been given as early as possible.

Robert L. was eight months of age when affected with this paralysis.

When Dr. Smith first saw Robert L., the baby was having mild convulsions, that is his eyes were twitching and maybe some little part of the muscles of the face were also twitching mildly. He was limp and he had been vomiting just before Dr. Smith arrived. His temperature was 99°. His mother told Dr. Smith that he had been having convulsions one right after another, that he would draw his right hand up to his shoulder.

Dr. Smith recognized the case as being that of Infantile Paralysis. The following day there was paralysis of the right arm and leg. He continued to have the convulsions. His foot began to draw, and his eye turned toward the side of his head so that you could not see the iris. It was two days before the carbonyl catalysts could be given. At that time there was complete paralysis of the right side of the body. He was perfectly limp. There was no motion or reflexes in either the right arm or leg.

Dr. Smith gave him two-thirds of one treatment (1½ cc.) of the SSR. The following day there was some improvement, and the improvement continued until the child was perfectly well. There was no muscle impairment as a sequel.

SUBACUTE POLIO WITH PARALYSIS

CASE No. 38 Michael R------, M.D.

The I. child, age 10, came to the office on September 10, 1941. The patient complained that for three days he had a headache and pain in the legs. His pulse was 90, temperature 98.6°, and the urine examination showed no pathology. The next day the symptoms of Infantile Paralysis were more evident; the Kernig sign was positive, he had a tenderness along the spine, he had neck stiffness, he could not lift his legs and the fingers of his hand were so weak that he could not turn on the radio. He had to lift himself with the elbows.

The Health Commissioner was called in the next day and he confirmed the diagnosis of Infantile Paralysis.

The Benzoquinone arrived on Sept. 13 and 2 micrograms were injected intramuscularly. The next morning the patient said that he felt some better, but his symptoms were about the same. A second injection was given on September 14th. About two days later he had less pain, he could bend his legs better and he had less neck stiffness. By September 20th, he had less lameness and he felt well.

On September 22nd, his temperature was 99.4°, and a third injection was given. Two days later the adductors, that is the muscles that bring the legs together, were less lame. He could bring his legs together. On October 8th

his temperature was normal and from that day on he did not have any more symptoms. The Health Commissioner was called on October 15th and he was shown how the boy could walk through the room. He had no more symptoms. In 1943 he was active playing basketball and football. This case shows that benzoquinone offers inferior survival reagent qualities.

ACUTE ANTERIOR POLIOMYELITIS WITH PARALYSIS

CASE No. 39 Dr. Wendell Hendricks

The following case is typical of many we have treated and illustrates the early reversal of the disease process following the administration of the oxidation initiators, the SSR.

Loman A., age 10 years, came down with the prodromal symptoms of headache, pains in his back and legs, stiffness of the neck and back muscles, vomiting, and fever of 104 degrees on February 3, 1944. The condition did not abate but became steadily worse through the night and the next morning. Our examination found the symptoms mentioned and flaccid paralysis of both legs from the waist to the toes. There were no knee jerks, or other reflexes to be induced in the legs or feet. The spinal fluid was taken and found to be 4 cms. on the manometer. We waited one-half hour so as to give plenty of time to see if the withdrawal of the few cc. of spinal fluid would alter the symptoms. He continued to get worse, the fever mounted to 104½ degrees and his pains increased. The pulse was 128. We then gave the SSR solution. Recovery started to make itself evident within two hours in a reduction of pain and headache and the vomiting ceased. Seven hours after the injection he could move his legs. His neck was limber and his temperature normal. He ate a light supper. The next morning he got up and walked to the bathroom unaided. His recovery was complete in a day or so and no sign of return has been observed. However. during the third week following the treatment he had a reaction of chills and fever that lasted three hours, after which he felt very well. There was no development of atrophy whatsoever. The third week reaction is of interest. It showed that a destructive or symbiotic integration was established.

The rapid establishment of widespread paralysis indicates the symbiotic type, but as the back muscles were becoming involved a lytic type extension was also probable.

POLIOMYELITIS

Acute Polio with Extensive Progressing Paralysis
The Case of Walter N.

CASE No. 40

This child was two and a half years old when carried into our office September 19, 1931. Both legs were paralyzed from the waist down and there was foot drop. He had fever and suffered pain. No reflexes could be elicited

from the legs. The paralysis was of the flaccid type and progressing. The spine was rigid. He had been vomiting. One dose of the Synthetic Reagent was given subcutaneously. He was held by his father about an hour longer, while being observed; then he slid off his father's lap and stood on the floor, making a few steps, but was quickly raised from the floor and prevented from further action. The paralysis had existed from eighteen to twenty-four hours previous to the treatment, so the release was rapid. The testimony of the mother has been paraphrased and is given for your consideration.

"I took my son, Walter, to Dr. Koch when he was two and one-half years old. He had been playing the night before but during the night he did not sleep, and cried; and when he got up he could not walk. I did not take his temperature, but felt his forehead and thought that he had a temperature. His leg was still sore, so the next day we took him to Dr. Koch's office. My husband had to carry him because he could not stand.

"Dr. Koch gave him an injection in the leg. We were in the office about an hour and soon after he had the shot, he wanted to get off my husband's lap on to the floor. He could stand a little, but we carried him to the car and kept him in bed that day. The next day he was up and playing. That was the summer of 1931." The recovery was complete and has remained.

ATROPHIC ANTERIOR POLIOMYELITIS COMPLICATED BY ANTIVARIOLIC VACCINATION

CASE No. 41 Dr. J. Treiger

Miss P., age 7, seen first by Dr. Treiger March 8, 1957. Her weight was 20 kilos. She had had measles, varicela, whooping cough, and at 3 years of age a nasal diphtheria, following which she has always been underweight in spite of a good appetite and sleep. Examination showed slight dyspnoea and constant cough with a hypertrophied left tonsil, much flatulence, fearful, crying at nothing, without appetite, very thin and weak, with paralysis and muscle atrophy of the right leg. The history showed that she received smallpox vaccine in March, 1957, and had a very strong general and local reaction. Her ill health continued with the added disadvantage of the paralyses and atrophies of Anterior Poliomyelitis which struck her in May, 1957. She caught colds very easily and every contusion produced a swelling that became purulent with ease. Good homeopathic prescribing helped her sustain herself at this status but something more fundamental had to be removed. From March, 1957, to March, 1958, she gained only one kilo in body weight. So, before the polio struck her, she was practically a metabolic invalid. The vaccination mark, however, developed a large mushrooming Keloid raised high above the surrounding skin. The atrophies prevented her from raising the right foot when she was lying on her abdomen on the table, and from bending the right knee when standing. The right leg gave no support when standing as the knee and the ankle

were both wobbly and without strength or control. The foot hung loosely from the ankle joint. She needed braces to support both.

On April 15, 1958, she received 2 millimicrograms of the SSR from Dr. Treiger, intramuscularly. The results were: four weeks later she had gained two kilos, and had better muscle tone. In two more weeks she gained two more kilos. In the middle of the twelfth week, after treatment, she had a characteristic reaction (July 7th). There was pain in the right leg, general achiness and fever, and itching of the Keloid. Three weeks later another reaction occured with fever of 108°F., much cough and the elimination of much clear mucous. This lasted only two days, but in these three weeks the Keloid reduced remarkably. One year later, in August, 1959, she weighed 29 kilos, and showed another reaction with pain in the leg, and the return of an itchy rash on the eyelid plus a general itching that she had six years earlier. In this period the keloid reduced so as to be at the same level as the skin and shrunk otherwise. At the time of treatment it was 10 to 12 mms. in diameter, and raised 6 to 8 mms. above the level of the skin. How deep it went is not known, but its present depth is not great as it is flexible. Still, there is maybe 20% to be absorbed to become an ordinary scar. Her appearance is healthy, she is vigorous and has a stable nervous system, and she walks without any bracing apparatus with a practically normal gait. She can now raise the right foot when lying on her abdomen, and can raise and bend the right knee when standing. The right leg supports her when standing and the ankle has regained fair function useful in walking without braces and with ordinary shoes. The circumference measurements showed an increase in the right thigh from 28½ to 34½ centimeters while the left thigh stayed at 38½ cms. and the right calf gained from 19 to 20½ cms. and the left stayed at 25 cms., between April, 1958 and Nov. 16, 1959. It would be interesting to decide if or not the smallpox vaccination gave the polio infection. Since the Keloid reacts concomitantly with the Polio reactions this might be suspected. However the eczematous rash also reacts at the same time and points to a fungus infection received at an earlier date. The Keloid is not yet fully absorbed and the atrophies are not yet fully corrected. More reaction is still expected. These will solve the etiological position of the vaccination. On Nov. 4, 1960, the right calf measured 22½ cms.

EPIDEMIC HEPATITIS

It will be seen in this group of Infectious Hepatitis cases that the virus host cell integrations follow the same pattern as in Anterior Poliomyelitis and the time relations of the recovery process measure up similarly to those of the recovery from paralytic polio, rabies, and dog distemper. Both the acute lytic and the chronic symbiotic types are observed, indeed the virus appears to exist in some cases as if integrated with foci of long suppressed infection, either in symbiosis with some germ or with the scar tissue that imprisons it. The relation of the vaccination scar to polio, and to cancer as revealed in their recoveries might be compared with the fecal contaminated scar of the fourth case given here. In all such stored viral infection, a drop in the resistance of

the patient allows the symbiosis to go rapidly lytic with rapid multiplication, and then the burst through as a general infection that attacks the most susceptible tissue dominantly.

The examples offered here present the four types of classification. The second case represents both the protracted type with remissions and relapses, and also the fulminating type when the infection suddenly fulminated and quickly went into the terminal phase with wild delirium, hallucinations and then a state bordering on coma when the Survival Reagent was given, and quickly reversed the pathogenesis. The first case represents the common type with jaundice and sickness that hangs on regularly for a month or two before it starts to yield, but here the reversal was evident in hours, and was completed in a week or two. Interesting is the thyroid disturbance like the brain disturbance in the second case showing that the virus can integrate with more tissues than the liver cells. The third case directs attention to the rate of recovery of function by the liver cells, as do the others also though they were more complicated. This is regularly about 48 hours in the acute lytic type of integration. In this time the bilirubin drops to a near normal, as from nearly 6 mgms. per cent to less than 0.5 mgms. per cent, while the bile absorbed into the skin and other structures takes longer to be liberated and eliminated. The patient, however, is well with good liver function, and no toxicity whatsoever. The following cases are reported by Dr. Treiger. The last one was observed by the writer.

INFECTIOUS HEPATITIS

CASE No. 42

M. C. S., a girl of 12½ years, 41 kilos, student, with a good school record, appeared Nov. 12, 1959, and gave the following findings. She was changing color from the healthy hue to a greenish yellow, great urgency to move the bowels after eating, slight pain in the muscles, pain in the bowels with marked nausea, that was violent in the days preceding school examinations, which were at hand then. The pulse was 120 per minute, temperature 37.6°C. and a sub-icteric tone of the sclera. When examined, complained of pain over the liver and epigastrium, with an accentuated tympanism, and terrific itching. There was excessive sweating of the hands and feet, and together with the tachicardia the thyroid gland appeared to be attacked. An epidemic was running at the time in her school.

Blood analysis on 11/16/59: Bilirubin, 1.43 mgms.% instead of 0.2% to 0.4%; Van den Bergh, postitive, but weak and delayed; Hemogram normal; Crenation test, only 20% crenated, 80% remained round.

Treatment: 2 micrograms of Benzoquinone solution was given on November 16, 1959, with homeopathic doses of iodine and chelidonium. Improvement showed the next day, was very good in 48 hours, and continued until on the tenth day, when she was practically well. The pulse had dropped from 120 to 86 per minute, the jaundice had faded away, no more pain or itching, and no nausea or sweating. The blood showed Bilirubin 0.41 mgms.% which is normal, the Van der Bergh was negative, the Crenation test was 90% normal.

She took her exams without any nervousness and with good results between Nov. 25 and Dec. 12. The correction was permanent.

INFECTIOUS HEPATITIS PROTRACTED SYMBIOTIC TYPE WITH SUDDEN LYTIC CHANGE FULMINATING TO TERMINAL STATUS

CASE No. 43 Dr. J. Treiger

S. M. L., age 31 years, gave a history of pyelitis in early childhood, frequent colds and in April, 1958, a broken leg, weight 45 kilos., when she consulted Dr. Treiger on 5/4/59. She had a fever of 38.8°C., complained of grippiness, muscular pains, tonsilitis, worse on the right side, halitosis, dirty tongue and facial neuralgia on the right side. This attack came after a season at the seashore on a heavy crab and shrimp diet. She was given homeopathic medication without result and she continued to get worse. On May 8, 1959, the fever was gone, but she was prostrated with nausea, repugnance to all food, feeling like a drunkard, and a terrible taste on her tongue. The muscular pains were gone, but there was a severe pain over the gall-bladder. On May 11, 1959, the blood picture was: Bilirubin 5.95 mgms.%; Van den Berg strongly positive, immediately, three plus; Cefalin-Cholesterol (Hanger) three plus; Thimol turbidity 7.5 units; Thimol Flocculation (MacLaglan), positive three plus.

That night she was much worse, again with a high temperature of 39.5°C, extremely agitated, afraid of dying and of being alone, hallucinations and delirium She had received a new homeopathic prescription that did not work. Dr. Treiger gave her two millimicrograms SSR when the agitation gave place to a new phase of prostration bordering on coma. However, the nausea did not develop though it had been strong and constant. After 48 hours the improvement was evident, with lowering of the temperature and the return of appetite and bowel functions. She steadily improved and on June 17, 1959, the blood showed Bilirubin, 1.02 mgms.%; Van den Bergh, delayed and weakly positive; Cefalin-Cholesterol negative; Thimol turbidity 5.5 units; and Thimol flocculation, negative.

July 10th, during the ninth week after the treatment she had a reaction with violent nausea and dizziness. She was given another dose of the SSR. The improvement was observable in three hours. Two weeks later the blood test showed a normal Bilirubin 0.41 mgms.%, and all other tests negative.

During the 12-15 week reaction period she showed an intestinal upset and this cleared up quickly aided by Ipecac and Terramycin. In the 27th week reaction period there was a transient light pain in the left lobe of the liver that cleared up quickly, but there were no other symptoms. The correction was complete. No doubt the intestinal tract was the first to become infected and was therefore the last to become normal, in line with the characteristic behaviour of the recovery process after the FCG is rescued and restored to action.

AN ACUTE LYTIC TYPE CASE OF EPIDEMIC HEPATITIS

CASE No. 44 Dr. J. Treiger

J. H. C., age 13 years, student, appeared with a history of having drunk water that was under suspicion of being filthy 15 days previously. He had abdominal pain that was helped by intestinal lavage. The urine was loaded with bile. There was deep jaundice and profound drowsiness. His state was subfebrile, and the blood examination on February 5, 1959, showed Bilirubin 5.83 mgms.% (Malory and Evilin). The Van den Bergh was immediately directly positive, the cefalin-Cholesterol positive, the Thimol Turbidity 5 units, and the Thimol Flocculation positive. Two millimicrograms of the SSR were given on February 5th. On February 7th the jaundice had faded considerably and he felt very much better, no more pain or other disturbance. The bile stained tissues had not completely unloaded until three weeks later when the Bilirubin test was almost to normal, namely 0.86 mgms.% and the Van den Bergh and other tests were normal. On March 10th he was fully recovered and back to school. Here again we see the restoration of liver cell function, which means the rupture of the viral host cell integration, and the restoration of the host cell to functional structure, required 48 hours. In rabies it required 72 to 84 hours, and in polio the restoration was proportional to the time the paralysis had lasted, and the host cell injury had been going on. Thus, this disease supports our postulate very well. While this virus is not killed by chlorine oxidation, it is destroyed by induced oxidation after it is integrated with the host cell.

CHRONIC INFECTIOUS HEPATITIS WITH MALNUTRITION

CASE No. 45

John S., age 28, when appearing for diagnosis and treatment, height 6 feet 3½ inches, weight 137 pounds, on August 4, 1957. When he took sick in 1952, he was in the air force. He had been in good health until an attack of appendicitis called for an operation. This resulted in severe peritonitis, and the use of 70 injections of antibiotics, mostly of streptomycin that favors virus infection so markedly. Ever since this attack, he was an invalid steadily getting worse, losing weight and strength, becoming more and more anaemic with less ability to digest his food until he had lost his appetite completely. The use of digestive ferments medication failed to help. Moreover, what little he did digest was not metabolized by the liver. This deterioration progressed until August 4, 1957, when he appeared for observation.

Examination showed slight jaundice, profound anaemia, enlarged tender liver, loss of elasticity of the skin, great weakness, and gastro-intestinal failure with its consequences. There was severe prolonged headache at times and difficulty to perform his obligations. The operation scar was hard and extensive.

Two cubic centimeters of the SSR solution containing two millimicrograms of reagent were given on August 4, 1957. There was no sharp turn for the better as usually follows, but he did not continue to get worse, and a slight

improvement could be reported on the 14th week. This trend gained a little momentum up to the 36th week when a sharp reaction took place, with acute inflammation of his enlarged liver and the abdominal wall scar, severe jaundice, high fever, abdominal pain, vomiting and increased headache. This lasted only a week and cleared up spontaneously with fair rapidity when one considers his greatly depleted condition. The blood examinations were interpreted by his physicians as indicating Infectious Hepatitis. But as the course of the disease was so different from the regular course and the improvement so rapid, we interpret it as a reaction to a chronic infectious hepatitis in which the virus was integrated with the fibrotic tissue of the extensive abdominal scars as well as his liver tissue. The oxidation of the toxic virus factor off from the liver cells gave rise to the inflammatory changes, and jaundice, but as soon as the liver cells were free, they were able to function again, and normal status returned.

He gained a good digestion, strength and weight so that when seen in December, 1959, he was perfectly well, the liver was normal, the scars soft and pliable, and with good vigor, and ambition to work.

RABIES

In April, 1955, the Fleury type live rabies vaccine made from street dog virus, was injected into 650 thoroughbred Zebo cows at the Fazenda Indiana, near Rio de Janeiro. The vaccine was prepared by the Health Department of Rio, and given by expert veterinarians. Six hundred cows showed no adverse effects, neither intoxication nor rabies infection. However, 23 younger animals did come down with virulent rabies. They all progressed in their symptomatology, and died in from 3 to 5 days in the classical course. The brains showed negri bodies, and the mouse innoculations were positive for rabies.

The writer arrived on May 19, 1955, while some of the animals were still alive but showing the disease in well advanced stages. Only one calf was in the early stage of deglutory paralysis, but she had to be given saline transfusions to combat the dehydration consequent to impeded swallowing. Two cases were under heavy hexamethylene tetramine treatment, and were not offered to us until they were in the terminal stage, and were not fit for statistical observation — only for experimentation, because of the saturation with the drug, which is an inhibitor to the Survival remedy. Six cows were in the terminal stage, about the fourth day, with typical torticollis, convulsions, deglutory paralysis, and were either paralyzed so as not to be able to walk, or if able to stand, would fall if pushed and not be able to get up again. Six were moderately advanced, and could stagger about, showed torticollis, and short convulsions, but could not swallow. They were in the third day or at the end of the second day in some instances. So there were thirteen cases treated for statistical observations, including the calf Iberia that had only beginning deglutory paralysis for a day or so. In all the symptom course was the same as in those that died, and

started with deglutory paralysis. There were seven other cases used as testimonials — collateral controls, but as the opportunity was a rare one we used them for experimental observations in a way that did not prevent them from going the regular course to death as the cows that died before them. We had learned in dogs that the quinone structure does not affect rabies favorably, so we treated some of these with diphenoquinone one part to a million, and some we treated with high dilutions, that is, a billionth of a microgram of the serial system of carbonyl groups, used in a dose of ten micrograms in the treated cases. Of the thirteen treated cases, eleven made complete recoveries, and two died.

All treated cases required 72 to 84 hours for restoration of controlled movements including deglutition. Its paralysis was the first symptom to come and the last to clear up. Animals treated during the fourth day of their downward course, were then widely paralyzed and had gone through a heavy convulsion period. They lay paralyzed for four days longer, before they showed restoration of function. An interesting case will illustrate. The government veterinarian who took charge of this epidemic had no faith in the cure of rabies, which was always 100% fatal, and as fast as any diseased animal showed it was following the classical course of the disease, he took it away for sacrifice, to save it useless suffering and to have better autopsy material. Our treated animals were allowed to follow along without interference except one cow that was treated at the end of the third day or the beginning of the fourth day, when it was already badly dehydrated. It lay paralyzed for four more days when the Government veterinarian ordered it dragged to the truck to be taken away, but on reaching the truck when they tried to hoist it in, it kicked up a fuss and tried to run away. The Fazenda veterinarian was passing by and as he saw the cow show coordinated movements he ordered a halt and led it to water, where it drank greedily. He then let it loose in the pasture to eat its way back to health. Here we see that at the end of the quiet period which is as long as the symptomatology period the reconstruction is complete. In other words the type of integration is such that it affords a reciprocity between the virus and the host cell. During viral vegetation the energy and material passes over to the viral colony, but during the viral oxidation a stepwise burning of the virus returns the energy to the host cell that was taken from it, and the food which diffuses into the cell supports its reconstruction. This explanation is the only one we can think of since a full reversal of the process takes place even after the host cell is at the point of full destruction and the viral colony ready to burst forth as miriads of mature parasites. This is especially significant since so long as the host cell and virus are integrated, the former can not carry on constructive or functional processes, but must yield to complete dissolution. So the oxidative destruction of the virus supplies the energy for grana reconstruction so that the grana can then control enzymatic activities for function. One should contrast this evidence with the fact that a minute and a half after a virus penetrates a host cell the integration is complete and no amount of vaccine or serological effort is able to cause the separation and rescue it. It is doomed. Serological aids are merely substitutional. The SSR rescues and restores the host cell and burns up the pathogen.

DISTEMPER IN DOGS

(Cinamose)

The hospital of the Army's veterinary service for small animals, Col. Columbo and his staff with Dr. Adelberto Carneiro, and Dr. Cantuaria Guimaraes, arranged for the treatment of dog distemper as brought in from the street, and homes. For the first 17 cases treated we had to guess at the dosage and get it regulated for a systematic treatment of this disease. These cases were lost, and although they are included with all treated cases in the statistics, they bring the cure rate down from 90-95% to 80%. The detailed report is published in Veterinaria, the official journal of the University Veterinary Department of Brazil. Columbo and Carneiro, Veterinaria Ano IV, Num. 1, p. 21, 1950.

In private practice in the nervous form of distemper the recovery percent ran 80% or over, but only small groups of dogs were treated. Many of these animals were paralyzed for several weeks, even longer than a month with involvement of the respiratory and cardiac centers beginning to show. Thus the chances to live as long to accomplish the restitution, as the nervous system was involved were certainly reduced. However, the time required for restoration of the affected cells, like in those of rabies, is about equal to that which accommodated the destructive process. We have the documentation of some private veterinarians, professors in the university on short series of cases where 80% of well established distemper of the nervous system were cured. However, a case of polio in a dog which acted just like distemper of the nervous system was the subject of the testimony before the Federal Trade Commission in 1946 by Dr. B. J. Myers, of Oklahoma City, a veterinarian who attended the dog through its recovery. It was a valuable Pekinese, that had been playing with a little girl neighbor when she came down with paralytic poliomyelitis during a severe epidemic. The paralysis started in the hind legs and tail and then affected the front legs and body, in the course of a week the neck and "bark" were paralyzed and death threatened from respiratory failure when the SSR was administered. It took a week to ten days to undo the paralytic degeneration. First starting with the "bark" and neck and reversing the paralysis until last of all the hind legs and tail regained their functions. Cinamose does not attack human beings, polio does attack humans and other animals. So far as the symptoms were concerned it could have been either disease that affected the dog, but because of the polio epidemic that was raging at the time and the contact the dog had with the sick child, the diagnosis of polio was made. No serological tests were perfected at the time to make a differentiation. The case is of value in that it shows the identical nature of the attack of a virus of different types on the same functional mechanism even in different animal species. The similarity of the pathogenesis and of the corrective process regardless of species is demonstrated. Also that the recovery course is not a matter of imagination, but a definite procedure, without psychotherapy.

HOG CHOLERA

Hog cholera attacks the whole body generally. The intestinal and respiratory tracts, the skin and vascular system, and the nervous system. At the end of the disease, just before death, the whole body is involved. However, in a small percentage of cases the nervous system infection dominates the symtomatology very early. There is ophosthotonus, torticollis, and convulsions. The animal behaves as if it had rabies. So far as this writer has seen this type, there was no attempt to swallow as the head could not be bent to reach the trough. Less than 20% show this type of infection in Cuba, and in Brazil it is even more rare. The disease is 100% fatal in three to five days after symptoms begin.

The following report is taken from an official record at the Veterinary Department of the Cuban Army under Fidel Castro:

A. "On May 15, 1959, Dr. Rodriques, Director of the Veterinary Center of the Estado Major of the Army, in Ciudad Liberdade, facilitated and arranged for the treatment of animals involved in an epidemic of Hog Cholera at Quartel 10, of San Antonio, that had already caused the deaths of 15 vaccinated pigs. The remaining pigs of the colony comprised a total of 74 infected pigs, 35 small and 39 large.

B. *"Prominent Symptoms:* Among the prominent symptoms that established the diagnosis were:

1. The deaths of the 15 pigs before our arrival.
2. All the pigs that were still alive showed,
   a. prostration,
   b. Respiratory difficulty, stentorous in type, characteristic of bronchiogenic pulmonary lesions,
   c. Hemorrhagic plaques, small and large in the skin of the abdomen of all animals, extending in some cases under the neck and jaw.
   d. Excretion of solid and liquid material with rectal congestion in some cases.
   e. Teats inflamed and hemorrhagic, very severe in one case,
   f. Nervous manifestations with torticollis, very severe in one case.
   g. Fevers running between 39.5° and 41°C. in various animals, the small animals with 40°C. The sickest animals showed a drop in temperature to 39.5°C. characteristic of the advanced stage.

C. *"Pathology:* The conditions of the material was the poorest as a result of the observed facts and changes. The animals had already carried the incubation period of 10 days with a prognosis of 100% fatality, according the virological standards, for after the virus is integrated with the host cell, no serological measure can set the host cell free and rescue it.

D. *"Treatment and Material Used:* The 74 animals were treated in two sessions, the first on May 16, 1959, and the second on May 19, 1959. The first group treated were 10 small pigs and 12 large pigs. The second group treated were 25 small pigs and 27 large pigs.

"A solution of diphenoquinone, freshly prepared, containing 1 microgram per cubic centimeter, was used. Small pigs were given from one to two cc. of the solution, and the larger pigs received from five to ten cubic centimeters by injection.

E. *"Results:*

"First Group. This group was observed on May 23, 1959:

a. No deaths,
b. Temperatures normal in checked pigs with the exception of pigs No. 3 and No. 4; on these the injection was then repeated,
c. The red hemorrhagic spots on the abdominal zone had disappeared in all animals. The congestion of the cornea had also left,
d. The rectal areas appeared normal,
e. The respiration was normal in all animals,
f. The female's teats that were formerly inflamed and hemorrhagic were normal in all animals,
g. One male of the group segregated as the most debilitated, attempted one week after treatment to accomplish reproductive relations with the females, successfully.

"Second Group. This group was observed on May 23, 1959, five days after treatment. The following results were observed:

a. No deaths,
b. Normal temperatures in all that were checked,
c. The red hemorrhage areas had disappeared,
d. The rectal areas were normal,
e. The respirations were normal in all animals,
f. The teats of the females appeared normal.

"This group was examined again May 26, 1959, one week after treatment when Group No. 1 was also examined. All pigs were found normal and attested to by the officials in control of the test."

In Brazil, the epidemics were small. At the Ministry of Agriculture Maracana Station, where cresol was used liberally, and the pigs were exposed to other inhibitants, the results were 100% failure in a group of seven pigs. But at the Deodora Station where no interference was met, the results were 60% cures, that is two cures out of three treated. These pigs were all in extremis, at the time of treatment. That is, unable to stand or eat, symptomatology typical. In another epidemic at a private farm 5 far advanced large pigs were treated with diphenoquinone, each receiving ten micrograms each at two different sessions 3 days apart; of the five only one could walk. They all recovered in 5 or 6 days, and were able to eat again, completely clear of chest and skin changes. In another epidemic at Santa Cruz seventeen of eighteen pigs in the herd had just died of Swine Pest. One still was alive and sick, it did not eat or drink, but could walk. The skin and chest symptoms were characteristic. It recovered also.

At the same time at a neighbor's farm several herds were affected. One group of four pigs equally advanced at about the third day, except a very small

"runt" that was near death. It had not taken water or food all this time and died within a few hours after being treated. The others all recovered. Another neighbor's farm had four pigs with symptoms in the second to third day of the disease. We treated the three that were worse, and held the other as a control. The third day later these were found cured and eating, having fully normalized. The control pig was quite advanced and not able to live more than another day, perhaps. We treated it, and two pigs in another neighbor's farm that were characteristically affected. They all recovered within a week when the next visit was made. Thus in the Santa Cruz group there were nine treated, including the far advanced "runt", and eight recovered and the "runt" died. This is a fair percentage and gives an idea of how the infection gets around. In another epidemic of ten pigs we used a different atomic arrangement, — the serial system of carbonyl groups. The results were 100% failure. This serial system of carbonyl groups gave 80% cures in rabies, and the quinone structure gave 100% failure. Thus there are two different 100% fatal viral diseases, each of which takes the same time to kill, that do not respond to the same carbonyl group when activated by different sources of electrons. The steric advantage and hindrance are different in each integrate evidently.

HOOF AND MOUTH DISEASE
(Aftosa)

There are three types of Aftosa virus, and their virulency varies. There is the deadly cardiotropic type that can wipe out a herd in short order. Some of the infected animals drop dead from heart failure before they can develop any lesions in the mouth or on the hoofs. It was such an epidemic that started at the Agriculture College, Institute Quinze de Novembro on October 14, 1949. There were 59 head of cattle and 200 pigs. The professor in charge was afraid his herds would be wiped out by this virus as five cows had already died a few hours after the epidemic had started. Others were laying on the ground unable to get up. They appeared doomed. Others showed lesions in the mouth and feet but were able to walk. We treated all 54 animals that were still alive, of these seven were newborn, 15 were calves, 17 were young bulls, and 15 were cows. Thus two-thirds were most favorable hosts for the destructive action of the virus.

Results:

Two cows and one calf died of the disease. All others recovered and no new infections developed.

Of the 200 pigs, 35 were adults, and 165 were young. 167 were well established cases of aftosa, while 33 were symptom free. All were treated.

Four pigs died of the infection giving a cure percentage of 98% and as no new cases developed, a prevention percentage of 100%.

The following year aftosa again struck the Institute. The cows we treated did not come down with the disease except a few showed mild symptoms for

a short time. The new cows brought into the herd took sick and died before we could be notified to come. A year later the treated cows were still immune, and again a year later we had the same report.

At the Rural University, the government Agriculture school, an epidemic broke out that was reported by Dr. Adelberto da Silva Carneiro, in Veterinaria No. 1, page 75, 1951. The virus was found to be type C. There were 68 cows in the herd. This virus was endemic and was active every year at the same time. It never killed over 10% of the cows, so it was not too fatal. However, the affected cows lost their milk production, and that was the big point of worry. Practically all of the cows showed symptoms including drying up the milk secretion. But a few were symptom free. All were treated. There were no deaths. Twenty-four hours after the treatment was given, the dried-up mammary glands started producing milk at the normal rate. Thus there was 100% cure, 100% prevention and 100% restoration of function. Only one cow needed a second injection, and then cleared up normally.

CONTROLLED AFTOSA EXPERIMENT

At the Rubino Institute at Montevideo, Uruguay, a controlled experiment was arranged in which 30 calves, all bulls, of the same size, age and "texture," were inoculated with a standard cardiotropic virus that killed 80% in from 4 to 9 days. The other 20% became chronic heart cases that died somewhat later. But the virus was 100% fatal in the dose used. The experts prepared a vaccine from this virus, and protected 10 of the cows therewith at an optimum interval before innoculation with the virus. Ten cows were held untreated as controls, and ten were given the SSR reagent. Since we were promised plenty of leeway and material for a good period of experimentation, we gave these cows the usual human dose used in cancer, that is a concentration of 10^{-12}, that is one part to a trillion of water. They were treated three days after the innoculation. The cows we treated and the untreated controls were held together in a small room with a bare cement floor in the coldest month of the year, July. They were grouped together with their noses all touching and the untreated cows were smearing the treated animals with their dripping infected saliva. The vaccinated controls were held in a warm barn with plenty of straw and given medical aid, transfusions and all the care possible for success.

Results: On the fourth day we had one death, and the vaccine protected animals had four deaths. The rest of the control animals were very sick with myocarditis, but our animals, the nine still alive were not, and they recovered fully. Since the control animals that had not yet died were getting ready to do so as the myocarditis progressed, the director of the institute asked us to treat them since they did not have cremation facilities for so many animals, and the law required cremation. We treated the rest this time as well as our test cases with the one to a million dilution, and the results were rapid. All of our cows recovered and so did our treated controls. The cure percentage on the one to a trillion dilution was not over 90%, but the cure percentage of the one

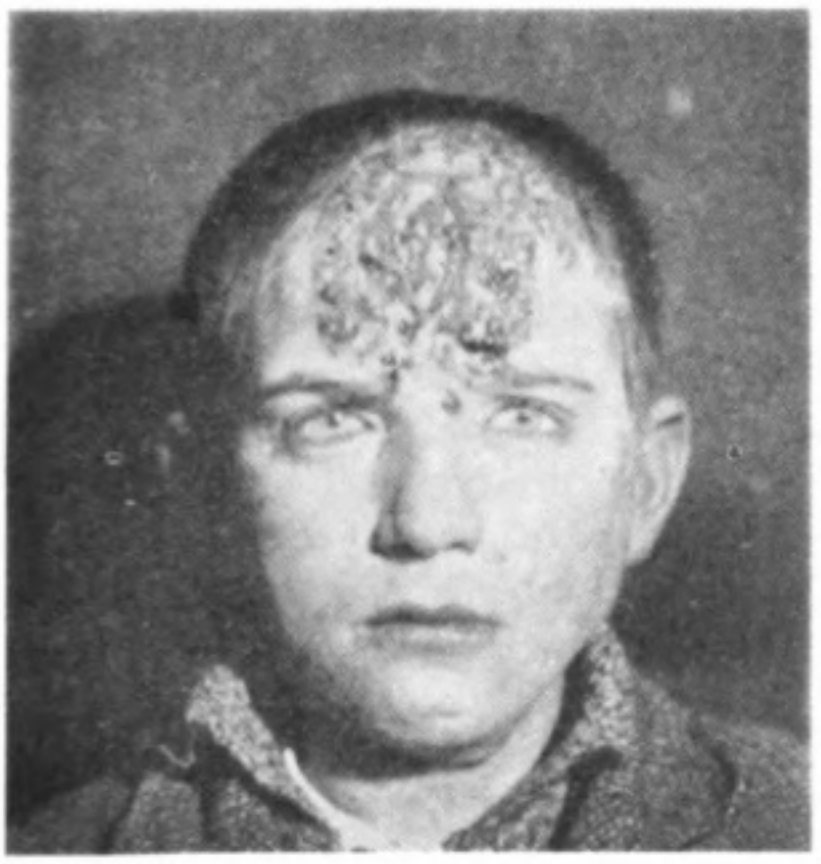
to a million dilution was 100%. The experiments were stopped because of political pressures that will not be discussed here. Another volume is devoted to curiosities.

In aftosa we see one virus type that paralyzes function of heart muscle and another that paralyzes milk production, both cured by the same reagent as cured the nerve cell paralysis in rabies, cinamose and poliomyelitis. The structure of the virus-host-cell integrate must be atomically of the same order then, in them all, since the end results of action of one reagent is the same — functional restoration of the host cell and disappearance of the virus.

SYPHILIS

CASE No. 46

This subject will be amply aired in the completed text, we here just give a case in photographs, to touch on the subject. This boy was sent into the hospital at Louvain University, Belgium, in 1934, with a diagnosis of cancer of the skull since he did not respond to antisyphilitic treatment though given expertly and with vigor. We found on biopsy and serological tests that he had syphilis. However, the neoplastic taint was not ruled out thereby, and his resistance to antisyphilitic treatment may be due to a neoplastic agent that may have been present. He was given a dose of the carbonyl catalysts and at that time his photograph showed as in No. I. Six months after treatment he was serologically cured and the photograph shows good healing of the area. The necrosis had gone into the middle layer of the bone, and the healing involved bone reconstruction as well as skin healing. One year later the fibrosis shown in the second photograph was replaced by perfectly normal tissue and could not be detected.



B. W. — Photograph No. I, taken before treatment.

B. W. — Photograph No. II, after treatment.

CHAPTER XVIII

TUBERCULOSIS

Several bacterial infections will be given a little attention to show that the bacterial toxin integrates with the host cell according to the same pattern as the virus or carcinogen. This is our conclusion since the same reagent causes the destruction of the toxin with liberation of the host cell. In tuberculosis, staphylococcus, streptococcus and corynebacterium infections, the walling off process is often extensive. Here the fibroblastic tissue evidently integrates with or copolymerizes with the toxin since after the high efficiency carbonyl groups are admitted to the field, the toxic fraction is burned off and the fibrous tissue has no more function, as there is nothing to protect against. The fibrosis disappears. It will be seen that the shaggy walls of the tubercular cavities first become smooth, and the cavity enlarges as the wall and debris are removed, then nothing more is to be seen of it as it becomes replaced with efficient breathing lung tissue. In the dairy cattle the extensive fibrosis of the udders is seen to disappear completely in over 80% of the cows after only one treatment, as part of the recovery process. Besides the hemolysins of the Staph. Aureus likewise disappear, and none of the bacteria are toxic any longer. So wherever the toxins are, freely circulating, integrated with the tissue cell, or as part of the germ, they appear to invite dehydrogenation by the activated carbonyl group, and in the presence of molecular oxygen they are burned away. The fibrosis is then replaced by functioning tissue as milk production increases to normal or better, and respiratory efficiency is restored.

As bacteria mutate against the best of antibiotics, all infections including tuberculosis become surgical diseases again. The old fashioned drainage is required and lobectomies or pulmonectomies in such cases as are favorable. It will be observed, too, that cases of lung infection that are not good surgical material as the bilateral cavitations and those with heavy walls that involve the mediastinum may respond favorably under the therapy described here, and thus give the surgeon the aid he needs until the case becomes a good surgical risk. Many factors enter into the management of such cases and the expert will have to make the decision as to procedure. It is observed in cases with large cavities, that while the patient's health is improving, the heavy toxic symptoms as fever, sweating, flush and weakness are disappearing, and the germs are thrown out in great numbers until the healing is complete. These bacilli are often seen to be phagocytized, and undergoing fragmentation and dissolution in the phagocytes, instead of the phagocyte being destroyed. We conclude that after the toxic fraction is burned off of the germ substance, it is readily digested, like tissue debris.

Prolonged bed rest is not needed in the care of these patients, even though far advanced. In fact, as soon as the fever disappeared, which is rather

early, they are expected to move about as they please and rest enough so as not to get tired. Long before the cavities have disappeared they do light work, and it helps them physically and mentally.

BILATERAL PULMONARY TUBERCULOSIS
With Large Retention Cavity

CASE No. 47 Dr. G. Warnshuis

The data on the first case, Mr. S. M., was taken in part from the records of the Herman Kiefer Hospital, Detroit, Michigan. He was sent to Herman Kiefer Hospital for X-rays and sputum test in September, 1938, but received no treatment there. Hospitalization was recommended at that time. Dr. Derby's letter describes his condition at the time of this examination.

March 10, 1939

Chrysler Industrial Association
7900 Jos. Campau Avenue
Detroit, Michigan

R: Stanley M---- G 110062
17207 Conley

Attention — G. A. B----

Gentlemen:

The above named was examined at this clinic on September 16, 1938, when a diagnosis of active pulmonary tuberculosis was made. Hospitalization was recommended. Our X-ray showed as follows:

Diaphragm: Costophrenic angle on the left is obliterated.

Right lung: Small amount of fibrosis visible in the infra-clavicular region. Left lung: Considerable mottling throughout the lower two-thirds of the lung with a large excavation near the root, measuring about 7 cm. in diameter and showing a definite fluid level.

Sputum examinations made in September and October, 1938, were all positive for tubercle bacilli.

It was understood at that time that the patient was cared for by Dr. Koch's Clinic. He has not been examined at this clinic since then.

Very truly yours,
Arthur P. Derby, M.D.
Director of Out Patient Department

Z.

Mr. S. M. came to our Clinic on October 20, 1938, for treatment by Dr. G. Warnshuis. His case history revealed that his father had died of intestinal obstruction. His previous illnesses were pleurisy in the left chest which was

followed by pneumonia in 1935. In September, 1938, he began feeling badly, had cough raised sputum and had night sweats. We had his sputum examined by the Public Health laboratory. It was reported positive for tuberculosis. Our own sputum examination was confirmatory. His weight was 153½ pounds, normal weight being 182 pounds.

Radiograph I was taken at the Herman Kiefer Hospital on September 16, 1938. Radiograph II was taken at the time he came to our Clinic. A definite increase in the extension of the large cavity and tuberculosis infiltration is seen in this short time. His condition also retrograded constitutionally and with regard to the cough, night sweats, fever, etc.

He received 2 micromicrograms of the Synthetic Survival Reagent. At that time he spent several weeks at our rest home, but was not put on the strict bed rest so rigidly enforced for patients in his condition. He was then sent home and kept on a vegetarian diet. He was allowed to be up and about, but told not to exert himself so that he got tired whatsoever. He kept a record of his temperature and other symptoms. He did his own cooking, shopping and drove his car from his country home, about 40 miles away, to our clinic every two weeks for a checkup. His improvement was slow at first, but steady. In a year he could do a little work. Radiograph III was taken on July 8, 1939. It shows healing of the large cavity during the recovery process.

On July 19, 1939, Mr. S. M. was examined by Dr. Douglas. He states in his letter of July 21, 1939, that "there has been some clinical improvement since last September," but that it was his opinion, "that this man is totally and permanently disabled because of pulmonary tuberculosis." Thus we see that in spite of Mr. S. M.'s condition when he came to us, he did make some definite improvement during this nine month period. Dr. Douglas still considered Mr. S. M. "totally and permanently disabled," and by this he meant, ". . . that the chances of recovery to the degree that this patient might be able to work are so poor that it is proper to say that he is totally and permanently disabled."

July 21, 1939

Dr. Peter Ivkovich
14128 East Jefferson
Detroit, Michigan

Dear Dr. Ivkovich:

In re: Stanley M----:

Stanley M---- was examined here on July 19th and I have procured the films from Dr. West for comparison.

This man has a far advanced pulmonary tuberculosis and while there has been some clinical improvement since last September, still there is evidence of quite extensive disease of both lungs and sputum tests run last month in the laboratory here showed the sputum to be strongly positive for tubercle bacilli.

With a disease of this extent existing for this length of time it would be in my opinion that this man is totally and permanently disabled because of pulmonary tuberculosis.

Very truly yours,
Bruce H. Douglas, M.D.
Tuberculosis Controller.

BHD
M

Mr. S. M. continued under our care. In February, 1940, we had an X-ray taken at St. Francis Hospital. At that time there was no evidence of a tuberculous process in either lung. The report of the Roentgenologist is reproduced here.

SAINT FRANCIS HOSPITAL
HAMTRAMCK, MICHIGAN

X-RAY ROOM PERMANENT RECORD

Patient's name: Stanley M---- Age: 45 Date 2-2-40
X-ray ordered by: Dr. Wm. Koch X-ray No.: 17339
Region: Chest Address: 269 River Road
A flat roentgenorgram was made of the chest.
Diaphragm: The leaves are smoothly rounded and normal in position. The costophrenic and cardio-phrenic angles are clear.
Heart: Is normal in size, shape and position.
Right lung: There is some increase in the lung markings toward the base. The lung field is otherwise clear.
Left lung: Here also there is some mottling at the base. The upper portion of the lung field is clear.
Conclusions: The findings are those of a low grade respiratory infection. There is no evidence of a tuberculous process in either lung at this time. The patient should be re-examined in from two to four weeks.
S. FORD, M.D.
Roentgenologist.

A summary of his progress as seen in the radiographs is given by Dr. Hague, a noted expert, as made from the films themselves.

DR. OMER GRENVILLE HAGUE

"The radiograph of September 16, 1938, is that of a male chest with the bony cage and ribs and collarbones and heart cavity in the middle and diaphragm down there. There are some infiltration shadows in parenchyma, or the active portion of the lung in these areas, in the fourth, fifth, and sixth and seventh interspaces anteriorly and a large cavitation shadow in the mid-lung

zone. I am measuring the left lung. That cavity measures 2½ inches by 3¼ inches, a little better than 3¼ inches. The outside measurement of the capsule of the cavity. By a little better than 3¼ inches I mean about ⅛ of an inch more. The reason I am not saying that with certitude is that the upper border of that cavity is very, very thin and very, very faint, but we can see that line that it follows and I would say it would be 3¼ inches at least. That is being very conservative. There is a small fluid level at the bottom of that cavity. There are, also, some heavy hilar shadows, and some thickening of the peribronchial trunks; that is, the lymphatics that follow the bronchi and smaller bronchioles. Those shadows indicate repeated infections that have resulted in inflammation and the inflammation has gone on to scarring.

The film dated July 8th, 1939, appears to be a film of the same chest; the ribs strip with the previous film. The lung tissues on both sides show soft infiltrated shadows throughout the lower two-thirds of both lungs. There is an interlobar line, indicating a thickening of the pleura between the middle and lower lobes, on the right side. There is a shadow in this area. It is smaller than the cavity on the left side previously referred to. It is in the same interspace level, so that I conclude it is related to the previous cavity. It measures 1½ inches by 1 inch. The wall of this cavity is less distinct. That is why it is a little harder to see. The shadows in the lung are of a soft consistency which would suggest an activity of disease in the lung structure itself.

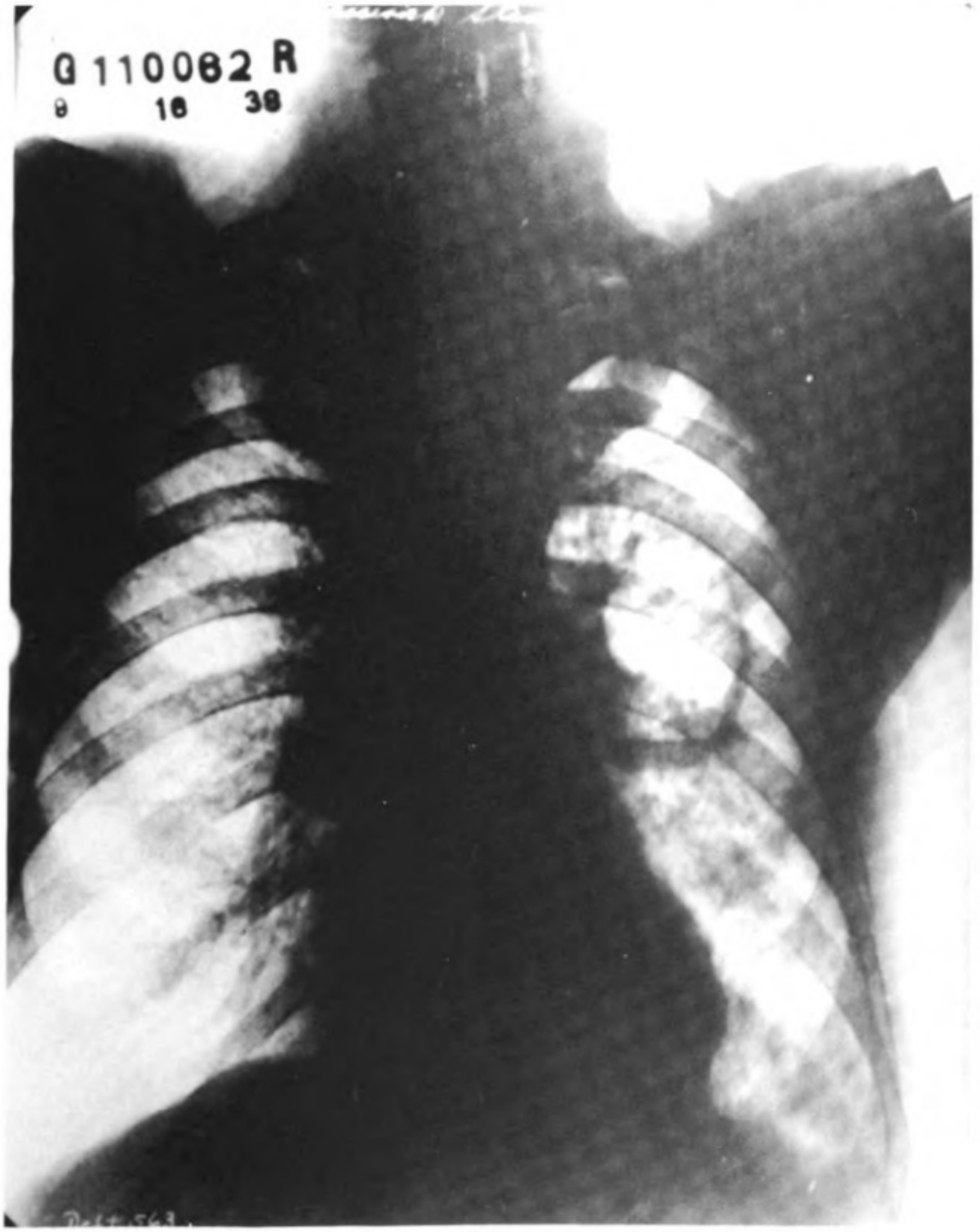
In the film dated September 16, 1938, the linear markings are fairly well fibrosed, hard. In the film dated July 8th, 1939, we see them softened and in an active state of inflammatory change. In the film dated June 18, 1940, this inflammatory reaction has disappeared and the outline of the cavity is very, very faint, practically disappeared. It would be very hard to measure it accurately. It would be about 1 inch by an inch and a quarter. The general appearance of this chest is much better than in the films taken September 16th, 1938, and July 8th, 1939.

Cavities almost of any size are a poor prognosis type of tuberculosis cases. The tendency usually is for individuals that have cavitation, that they get more cavitations rather than less. Cavities usually tend to get large and unless they are treated successfully by a pneumothorax, or some other compression therapy, and are held down for a long time, they usually get worse and the patient's outlook is serious.

The cavity in the film dated September 16th, 1938, I think is about as large a one as I have ever seen and I would say that patient's condition would not be a good risk at all.

The two succeeding pictures dated July 8th, 1939, and July 18th, 1940, show that there has been an extensive constitutional change taking place; that is, the soft tissue of the lung has undergone a remarkable exudative change; that is, there is a softening of the structure all through and in an instance like this that patient would have more cough and more sputum and it might be in the healing phase following this type of chest. For instance, the tubercle from this cavity may have been coughed up and spread out throughout the whole

lung and that might be a cause for the infection from here to become broadspread in that chest almost like a tuberculous pneumonic condition and then in this view, this pneumonic process has disappeared and the shadows in the lung



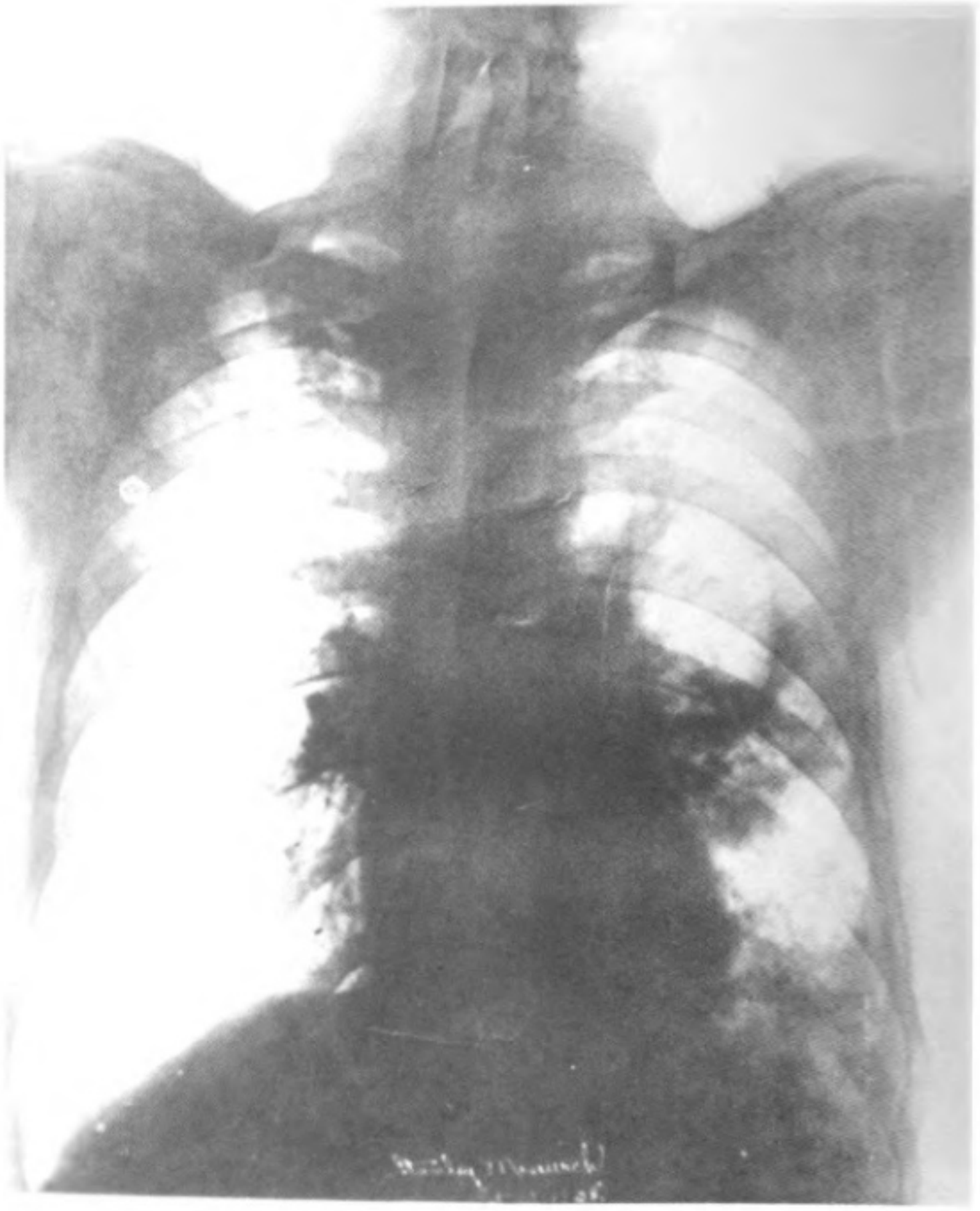
Radiograph I, taken at Herman Kiefer Hospital, September 16, 1938.

are back to what you would expect of an individual of this age and following conditions of a tuberculous recovery.

The prognosis on the first film dated September 16th, 1938, would indicate a very serious situation.

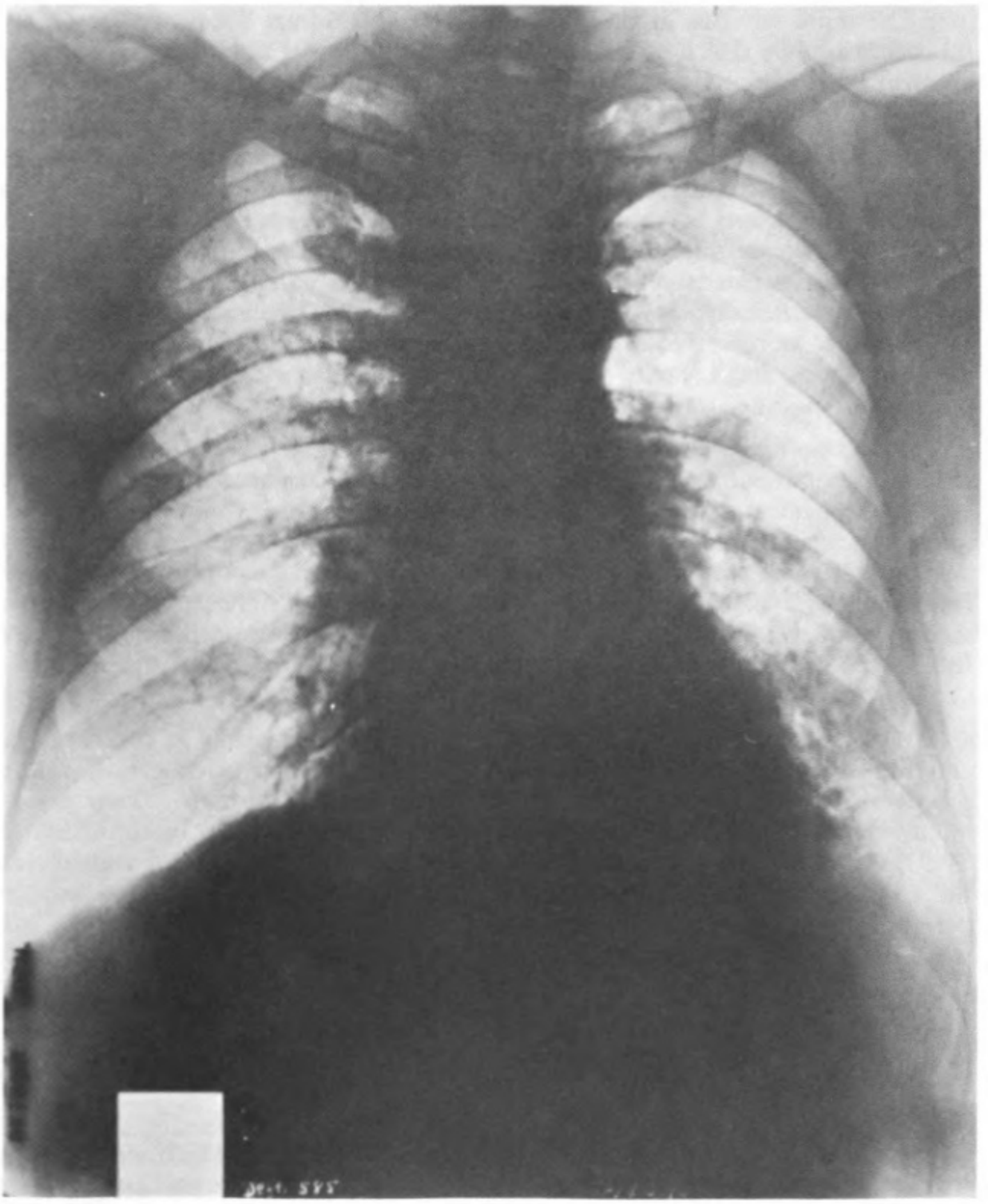
The prognosis on the third film dated June 18th, 1940, without knowing anything about the other two, would be very good."

In the Spring of 1942, Mr. S. M. went to work for Fisher Body, a division of General Motors Corporation. He did hard manual labor, worked long hours



Radiograph II, taken at our clinic on October 20, 1938 and showing extension of the tubercular process.

and overtime as well. During his employment he was given examinations and X-rays were taken. They established his good health and he was permitted to continue to work. His sputum has been negative since his recovery. He has continued working and testified in the Federal Court trial in 1946.



Radiograph III, taken on July 8, 1939, about nine months after treatment and showing healing of the cavity and the lung tissue undergoing reconstruction

EXTENSIVE MILIARY TUBERCULOSIS
With Tubercular Meningitis and Tubercular Nephritis and Splenitis

CASE No. 48

The rapid advance of the disease in Miss N. A., age 14, when treated by the writer in July, 1922, was reversed from the terminal stage by one injection. She had been in the Detroit Tuberculosis Sanitarium from the end of January until April. The radiographic findings of January 30, 1922, were:

"Both diaphragm leaves are clear. The trachea shows no compression or deviation. The heart shadow is normally placed. Increased deposit at the hilum on both sides. Some accentuation of the linear markings throughout the right lung field. No definite evidence of a parenchymal lesion in the right lung field. There is extensive parenchymal infiltration throughout the left lung field, with greatest changes in the upper half. Several small annular shadows at the apex and at the level of the first and second ribs anteriorly. We believe these represent small cavity formations. Diagnosis: Advanced parenchymal tuberculosis confined to the left lung field."

It will be noted that the lesions are located subclavicularly as in the most rapidly disseminating type. Six months later I examined her. She was emaciated, bedfast, comatose, with frequent projectile vomiting for three weeks, cyanotic, with rapid shallow respiration, very rapid, thready, practically uncountable pulse, with the head drawn back in continuous opisthotonus. The fever was 105.5°F. The heart was drawn over into the right side of the chest; the left chest was empty of viscera so far as could be determined, but contained fluid that splashed on shaking her. This lung had spontaneously ruptured.

The right lung showed huge cavitations and consolidations, and the splenic area presented a hard, fixed tumefaction as large as her head. It could not be determined then if this was kidney, spleen or both involved in the tuberculosis process. She was also near death with a tubercular meningitis. Two micro-micrograms of the Synthetic Survival Reagent were given intramuscularly. The recovery process was slow at first, but this was to be expected considering the condition she was in at the time I examined her. It was some months before she got out of bed. A year later the heart was still on the right side of the chest, but the left chest was not empty any more, it was full of apparently a fluid and fibrotic tissue. At the time she was walking about, her heart rate was still exceedingly rapid and weak, though it had dropped from around 150 to 130 per minute. Her temperature was normal. After that she gradually got better and the heart went gradually over into the left chest where it belonged. The heart rate slowed down.

An X-ray taken July 24, 1944, shows that the left chest, which was so badly involved, is not exactly normal yet in all these twenty-two years because there is less lung tissue there. That is because part of the chest is replaced by the structures of the mediastinum. The lung tissue shows markings in chests that one would interpret as healed tuberculosis. These marks are very, very

small, about a millimeter or two in diameter, dense, fibrous, showing calcification. It shows that the healing process has been very complete, and the scar tissue present from the large lesions healed and are very, very minute.

In this case we have a recovery with reconstruction in both lungs. The other pathological conditions cleared up during the recovery process. Her weight has increased to 145 pounds. She is married and has given birth to twins, pretty husky children. She has remained well to date (January, 1957).

The large mass in the splenic area disappeared completely, but the first improvement was noted in the nervous system, the disappearance of the opisthotonus and comatose state. This showed the SSR had taken effect, but still it was doubted that so much lung destruction could ever be repaired. Time showed that the advantage given here by the SSR not only permitted full lung reconstruction and return of the abdominal lesion to normal, but a general excellent health was gained that proved resistant to the usual infections. Her twins showed a remarkable immunity to the usual school children infections that none other exhibited, indicating a possible gene change.

FAR ADVANCED TUBERCULAR PNEUMONIA
Or Acute Miliary Invasion, in Extremus at Time of Treatment

CASE No. 49

Another case of pulmonary tuberculosis in extremus at the time of treatment with a dose of SSR is that of Mrs. M. B. H. and is of the type that has always advanced to fatality, namely originating in subclavicular lesions in both lungs. At the time of our treatment April 2, 1934, her condition was too critical to permit a thorough differentiation as to whether she was dying of an acute tubercular pneumonia, or of an acute widespread miliary tuberculosis. She was "sunk in bed," fever hovering about 104°F., rapid, thready pulse, cyanosis, with flush from the fever, yet the skin showed yellow hemolytic color after compressing the flushed capillaries. The breathing was very shallow and rapid, and very little but bloody sputum was raised. The physical findings scarcely revealed the cavity, only rales and solidification. No radiograph was taken at the time. A 2 cc. injection of the SSR, 10^{-12}, was given immediately. She had progressed to this stage from an early subclavicular and apical involvement thought to be a "cold" in August, 1931, when she entered the Herman Kiefer Hospital of Detroit. The condition advanced to that shown in their film of April 23, 1932, Radiograph I. She remained in the two Detroit Public tuberculosis hospitals up to late in March, 1934, when she was brought to our clinic. Films of January 18, 1934, and March 8, 1934, show the advance of the disease under their care with the development of the large, shaggy cavity with slight fluid level behind the right clavicle. The latter film being reproduced here, Radiograph II. The experts offered to perform a Thoracoplasty operation upon her at that time. She left because she did not want this operation. She felt too sick for such a drastic operation, with her high fever and exhaustion. Her sister brought her to our clinic on March 31, 1934. The sputum was

HERMAN KIEFER HOSPITAL

X-RAY REPORT

NAME B , Mary CASE NO 11049

DATE Aug. 31, 1931 PAVILION 2 - A BOX NO

PART X-RAYED Chest - Single film

REPORT OF X-RAY

Thorax, diaphragm and heart reveal no pathology.

Right lung: There are scattered infiltrations of the mixed type, throughout the upper third of the lung. There are areas of rarefaction near the clavicle, the largest measures about 3 cm. in diameter and shows a small fluid level. The remainder of the lung is clear.

Left lung: There are exudative infiltrations visible at the 1st interspace. We see no cavities. There are less dense infiltrations in the apical region and at the 2nd interspace.

Conclusion: Far advanced tuberculous process of the mixed type, involving the upper third of both lungs. There is a large cavity on the right as described.

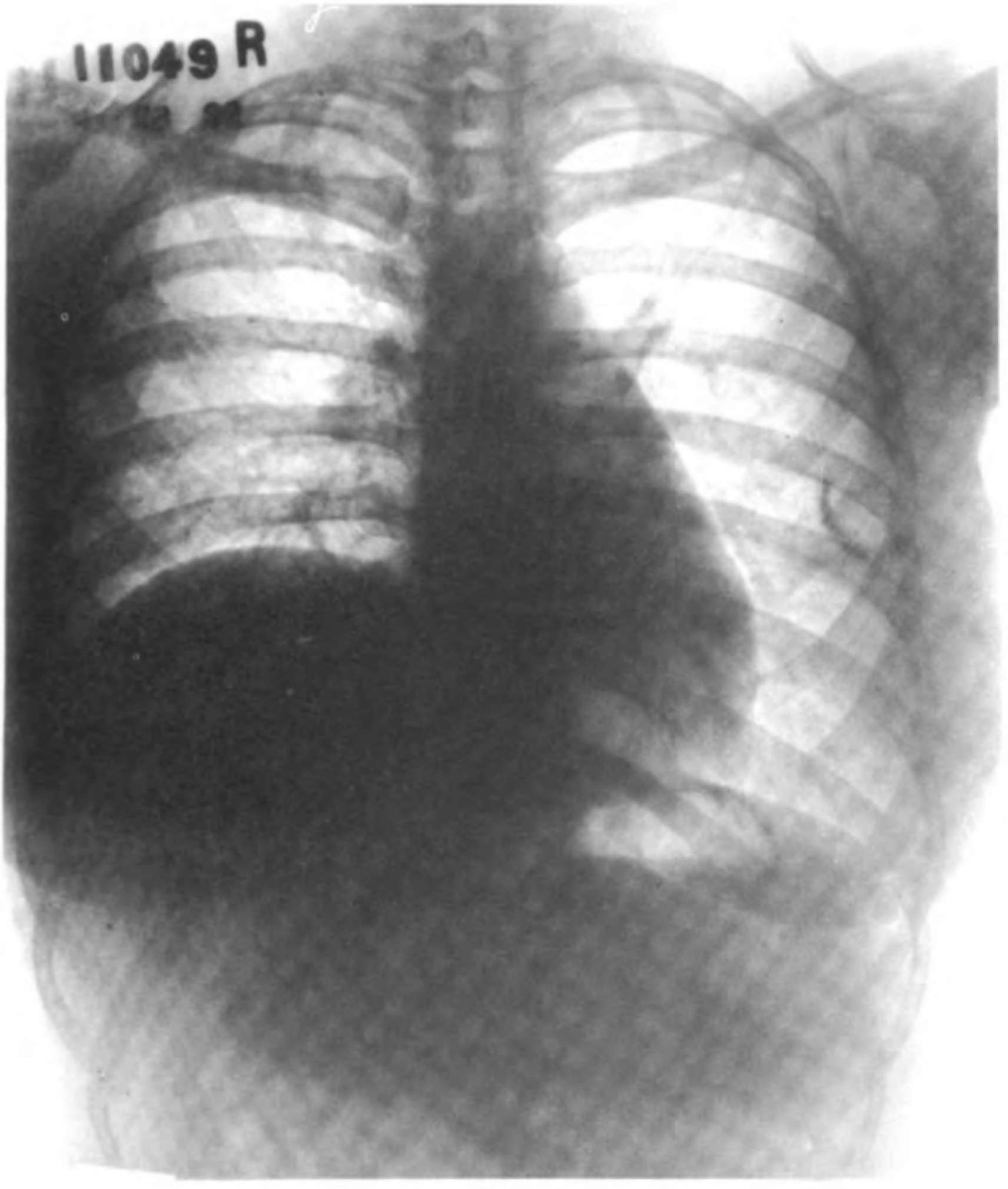
Dr. Birkelo - T

INTERPRETATION

SIGNED

This report shows the condition of Mary B. when she entered the hospital.

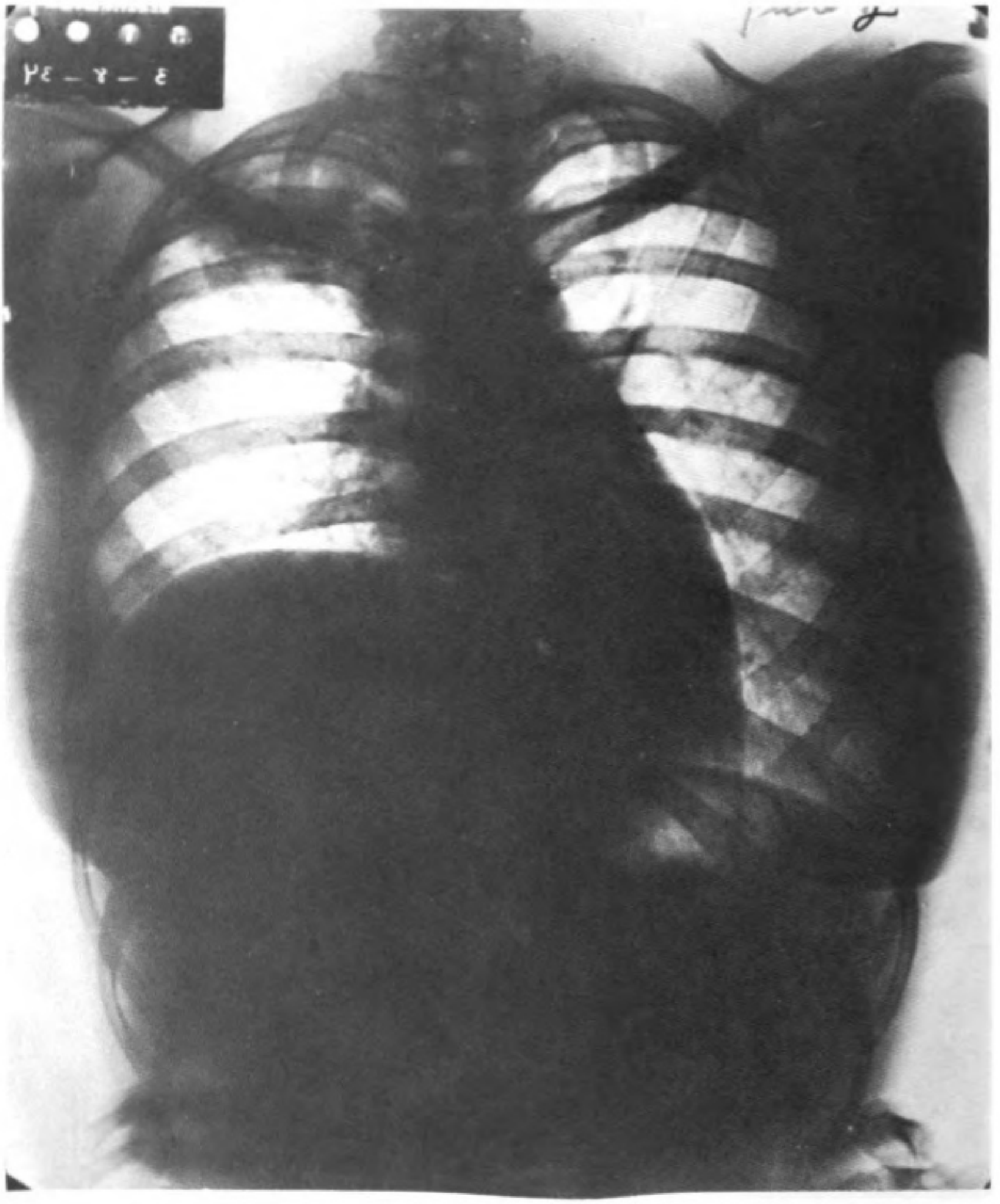
loaded with tubercle bacilli. The films show the advance of the disease in both lungs and the infiltrative development of the cavity wall. We took no radiographs until six months later when she was up and about and doing light work. Radiograph III, September 24, 1934, shows a smooth wall cavity. At this time there were no tubercle bacilli found in the sputum at daily tests for two



Radiograph I, made in April, 1932, shows advance of disease from August, 1931, in the hospital.

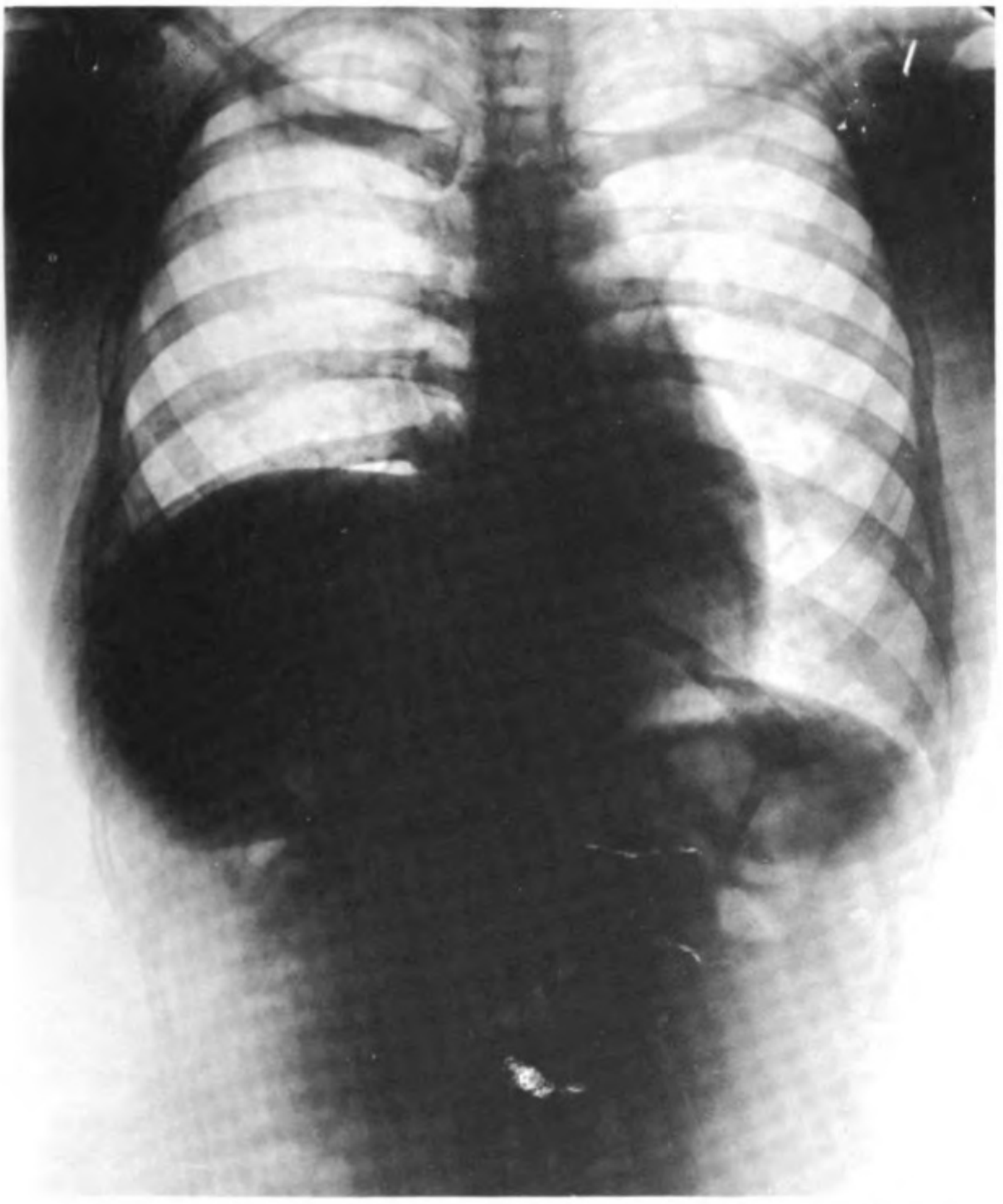
weeks. This cavity represents the area where the infection had taken place, and where lung tissue was again healing in after the disease tissue was removed, and the completed process is shown in Radiograph IV, September 12, 1942.

She left the city after leaving our clinic and within a year went to work, at which she has remained ever since besides being married and living a normal

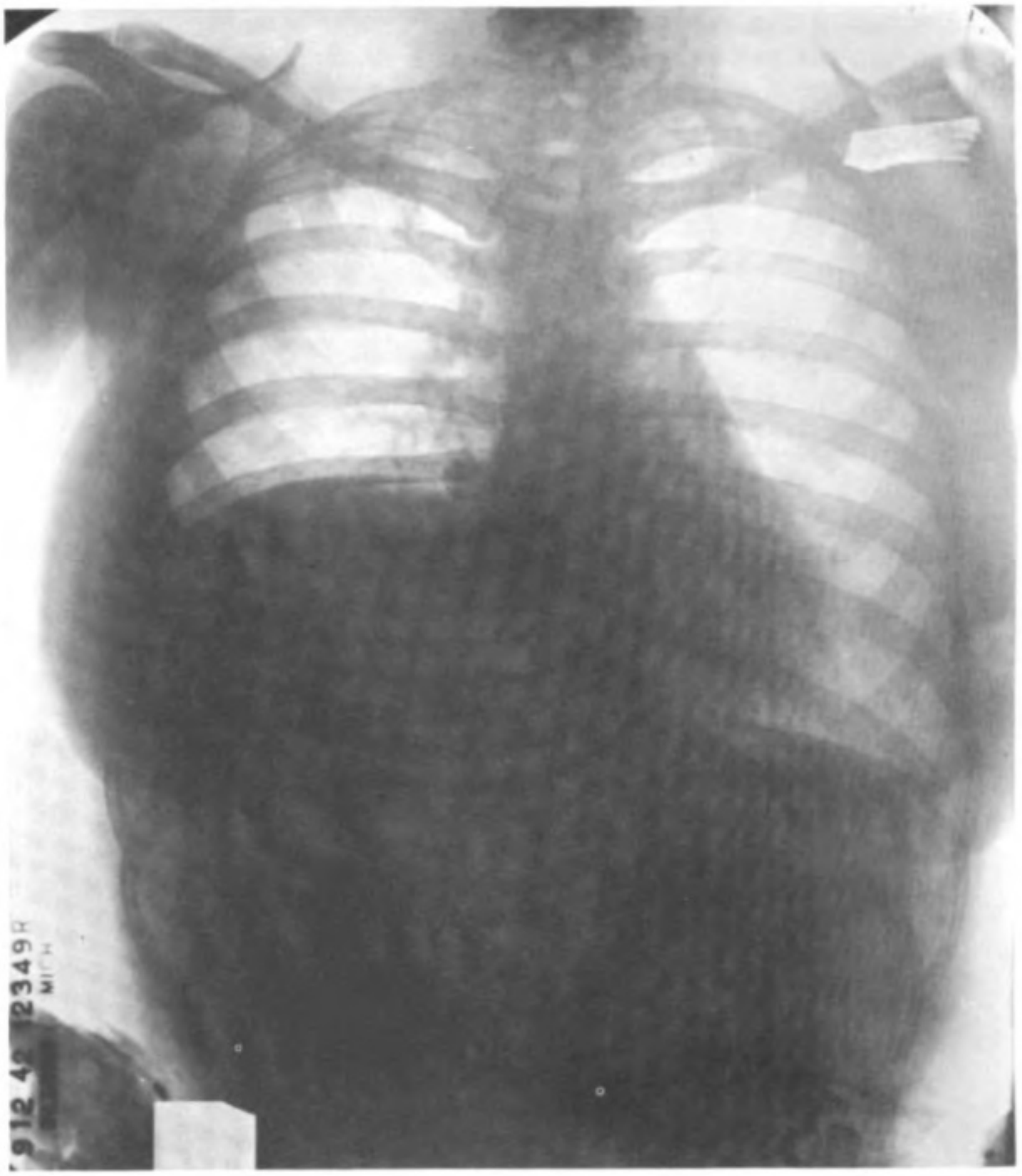


Radiograph No. II, shows advance of disease in hospital up to March 8, 1934, with cavitations.

life. Frequent sputum tests showed no more germs of tuberculosis and her gain in weight and perfect health mark her cure as complete. Physical findings are normal. Her X-ray report at the time of entering the Herman Kiefer Hospital is reproduced in photostat, from the court documentations. Reports in June 1960 show persisting recovery, good health, and working regularly. (See appendix).



Radiograph No. III, shows the cleaning out of the tubercular infection and a sterile cavity undergoing healing. This radiograph was taken on September 24, 1934, about 5½ months after treatment.



Radiograph No. IV, showing the cured state in 1942,
eight years after treatment.

ADVANCED TUBERCULOSIS
With Cavitations in Both Lungs

CASE No. 50

This is a case of bilateral cavitation with subclavicular lesions and two large thin walled apical cavitations on the right side. She steadily deteriorated under the best Sanitarium care in Cleveland, Ohio (Sunny Acres), and in

Detroit, Michigan, from 1931, when her thyroid gland was removed at the Crile Clinic of Cleveland, until September 2nd, 1934, when she received treatment in our clinic at Detroit

HERMAN KIEFER HOSPITAL

X-RAY REPORT

NAME R Cora CASE NO. 11584

DATE Mar. 10, 1932 PAVILION 2 - A BOX NO.

PART X-RAYED Chest - Single film

REPORT OF X-RAY

Thorax, diaphragm and heart reveal no pathology.

Right lung: There are dense infiltrations of the mixed type, in the upper third of the lung. There are small areas of rarefaction in the infraclavicular region, the largest measures about 2 cm. in diameter.

Left lung: There are rather dense infiltrations in the upper half of the lung. There are several areas of rarefaction near the clavicle, the largest measures about 2- /2 cm. in diameter.

Conclusion: Far advanced tuberculous process mixed in type, involving the upper upper half of the left lung and the upper third of the right. There are cavities on both sides as described.

Dr. Birkelo - T

INTERPRETATION

SIGNED

Miss C. P. was examined at Herman Kiefer Hospital, Detroit, Michigan, in March, 1932. A photostat of the hospital X-ray report is reproduced here. This is done because the radiograph does not show all of the lesions well.

Radiograph I shows the extent of the disease on February 24, 1934, about six months before she came to us for treatment. Phrenectomy and pneumothorax had been unsuccessful in curing this patient. At the time we first saw her, the prognosis was serious. Bloody sputum loaded with tubercle bacilli was expelled up to the time of our examinations in September, 1934. We found the upper half of both lungs invaded and a highly toxic state that

resulted in unusual muscular weakness. On September 2, 1934, she was given 2 cc's of the serially arranged SSR carbonyl groups. This toxic state quickly left so that two weeks later she went to work instead of being confined to bed rest. She received a second treatment on November 24, 1935. Since then she has had three more treatments over the years, one in 1937, 1939, and 1942. She has been working, is married and in good health, according to our last report. Radiograph II, taken March 22, 1943, shows the cured state, with minimum of scar tissue and return of normal lung parenchyma.

This case shows how the basic toxic state that caused hyperthyroidism requiring thyroidectomy, also removed her resistance against tuberculosis infection, and caused other disturbances resulting in muscular weakness. This latter effect persisted until the time of receiving the carbonyl therapy, and then very quickly disappeared. *It was no doubt due to suprarenal cortex inhibition.* Her statement on her weakened condition includes this: "When I first visited Dr. Koch, I was very short of breath and was able to walk a short distance only," and "I felt better almost immediately after the treatment, and went back to work fourteen days later, September 16th."

The testimony of Dr. Hague on this patient's X-rays has been paraphrased and reproduced here.

After examination of the X-rays from Sunny Acres Sanitorium, Dr. Hague stated in regard to the X-ray taken 8/6/32 that: "This is a radiograph of a thorax of a female patient, showing the chest cavities with a tuberculous process in the upper half of both lungs, of an advanced degree, with cavitations in the left apex, about three of them contiguous with one another, so I shall measure them all together. They measure two inches by one inch.

"There are also some shadows in the opposite side that suggest smaller cavitations behind the second rib anteriorly on the right side. This area of whiteness is an extensive tuberculosis process in the upper lobe of the right lung. A similar condition exists on the left side, but it doesn't show so much density as on the right, because there is a cavitation process which has taken away some of the fibrosis and that has been spat out as sputum.

"The descending bronchi are thickened because of repeated drainage from the upper areas of infection that have passed down into the lower trunks on both sides — lower bronchial trunks on both sides."

X-ray film dated 11/5/32: "This film shows the same patient with an aggravation of the disease, in which there is a shadow in the first interspace anteriorly, suggesting cavitation; and an enlargement of the shadows on the left side, indicating enlargement of the previous cavities. And, I believe an angular shadow that is on the left side in the previous film now shows more clearly that it is becoming a cavity, too. So that you now have an area of potential multiple cavitations measuring three inches by two inches. I would say that this patient is worse on this (11/5/32) film than on that one (8/6/32)."

X-ray film dated 2/11/33: "The tuberculosis process in the right lung has increased. The cavitation is large; measures an inch and a quarter outside

measurements in both diameters. The total area of cavitation in the left upper lung is slightly more, but there is a concurrent factor of a pneumothorax in which there is air in the base and up over the upper lobe of that lung."

X-ray film dated 5/17/33: "The only significant change in these two sets of film is that there is a little better compression over the apex of this lung, and one fairly strong adhesion band at a level of the third rib anteriorly is holding lung structure from complete collapse in that area."

X-ray film dated 8/16/33: "The same conditions exist in this film, with the exception that the outline of cavity in the right upper lobe, measuring two by one inches, is more clearly seen. There are still adhesions on the left side."

X-ray film dated 11/29/33: "I would say the left lung doesn't show any significant change from the left lung in the immediately preceding, but the changes in the right upper lobe indicate the cavitation a little more sharply outlined, and a little heavier in cavitation wall thickening, which would suggest to me that there is more activity (the tuberculous process would be more active, creating more inflammation), and the response to the inflammation is characterized by a deposit of fibrous tissue surrounding that cavity."

X-ray film dated 2/24/34: "This is a little lighter film. The right apical cavity is clearly seen. The fibrous tissue surrounding it is a little less in density, but it is still present."

X-ray film dated 5/26/34 was then shown to Dr. Hague and he stated his opinion as to the general picture of the pathological condition of this patient at the time of this picture. "The three last views show no appreciable improvement under pneumothorax therapy. The diseased area of the left lung, with its cavitations, has not completely collapsed, because there are adhesions remaining, and the cavitation in the right apex with the associated fibrosis still exists, and I would say that that is a very serious case of active tuberculosis."

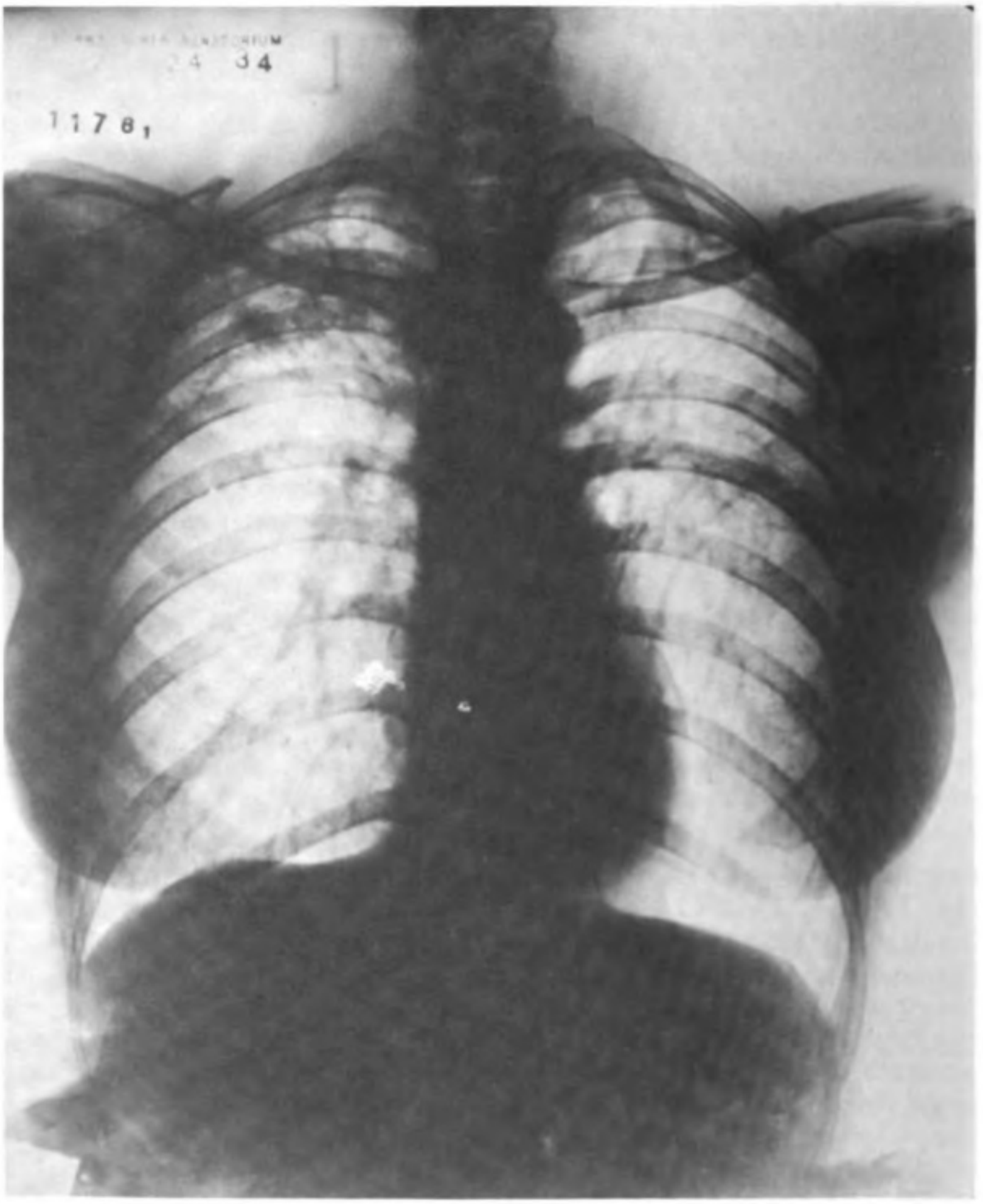
Where the patient is still showing positive sputum, the prognosis is serious. [It should be noted that this last X-ray (5/26/34) was taken over 3 months before the patient was treated by the author.]

Dr. Hague was shown an X-ray taken about seven and one-half years after the patient was first treated in Dr. Koch's Clinic. He stated that the X-ray taken February 19, 1942 "indicates a very marked improvement of this patient. The pneumothorax previously seen has now disappeared, the gas has been absorbed, and the lung has re-expanded to fill the chest cavity. The areas of former large cavities in this side, in the left upper first and second interspaces, have practically gone, and the annular shadow on the right side in the first interspace also is gone, but there still remains a mild fibrosis in the first and second interspace at the site of the previous infection. There are fairly heavy hilar shadows in the left upper mediastinal area which have come from the inflammatory reaction of the large area of cavitation previously seen."

The last film that had been taken, on March 22, 1943, is reproduced here and is radiograph II. Dr. Hague stated: "That it does not show very much

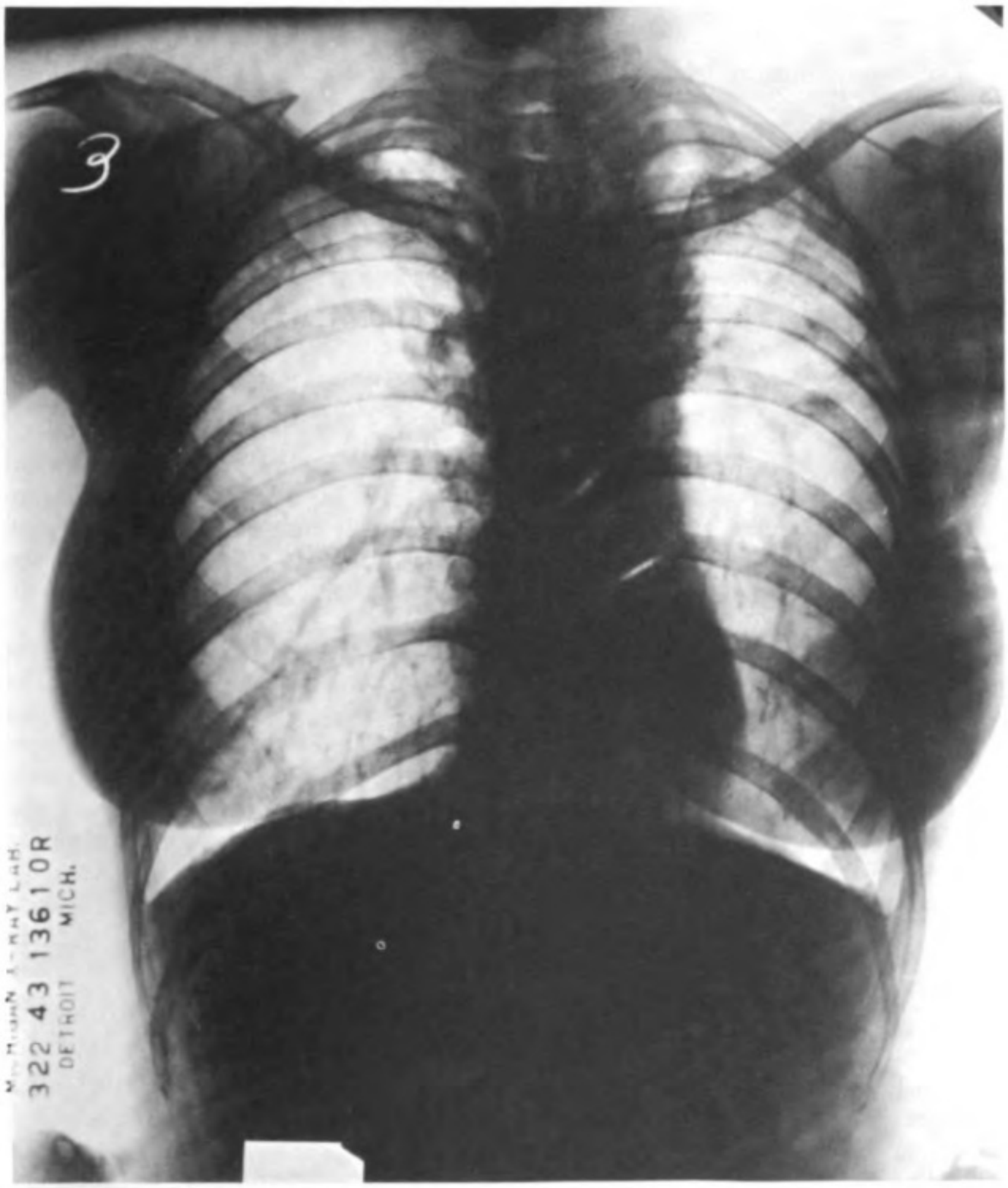
change from the one immediately preceding (2/19/42). I would say it is about stationary."

To the question: "Assuming at the time of the last two films that the general health of the patient was good, and there was no sputum or no blood, what would you say as regards improvement or recovery process in these films?" Dr. Hague answered: "Well, having seen all the cavitations of the left side and the large one on the right — these two show a remarkable removal



Radiograph No. I, taken February 24, 1934, about six months before treatment, showing cavitations in the right apex.

of disease process. It would be considered an excellent recovery if it were in the ordinary course of observation in a sanitarium. We would consider that a cure, under sanitarium conditions."



Radiograph No. II, taken March 22, 1943, showing recovery.
It was taken for court purposes.

TUBERCULOSIS OF LUMBAR SPINE

CASE No. 51

J. A., age four, came to us with a diagnosis of tuberculosis of the spine in August, 1924. He had been diagnosed by Detroit's leading orthopedist, Dr. La F----, Sr., who advised an Ablee Splint operation. He was supported by a brace that limited motion and reduced the pain only partially.

The family history showed his older sister had far advanced pulmonary tuberculosis.

For about a year he had increasing pain in the lower back and legs, found it difficult to get up after falling and would cry out with pain when he relaxed in bed on going to sleep. We fitted him with a brace, too. It did not limit his motion too much, but it did aid him during his sleep.

Examination showed a typical tuberculosis kyphosis in the lower lumbar spine. The two lower vertebrae showed a sharp angulation that protruded with collapse of the bodies of the vertebrae. There was considerable swelling of the soft tissues and muscle spasm about the region which limited motion.

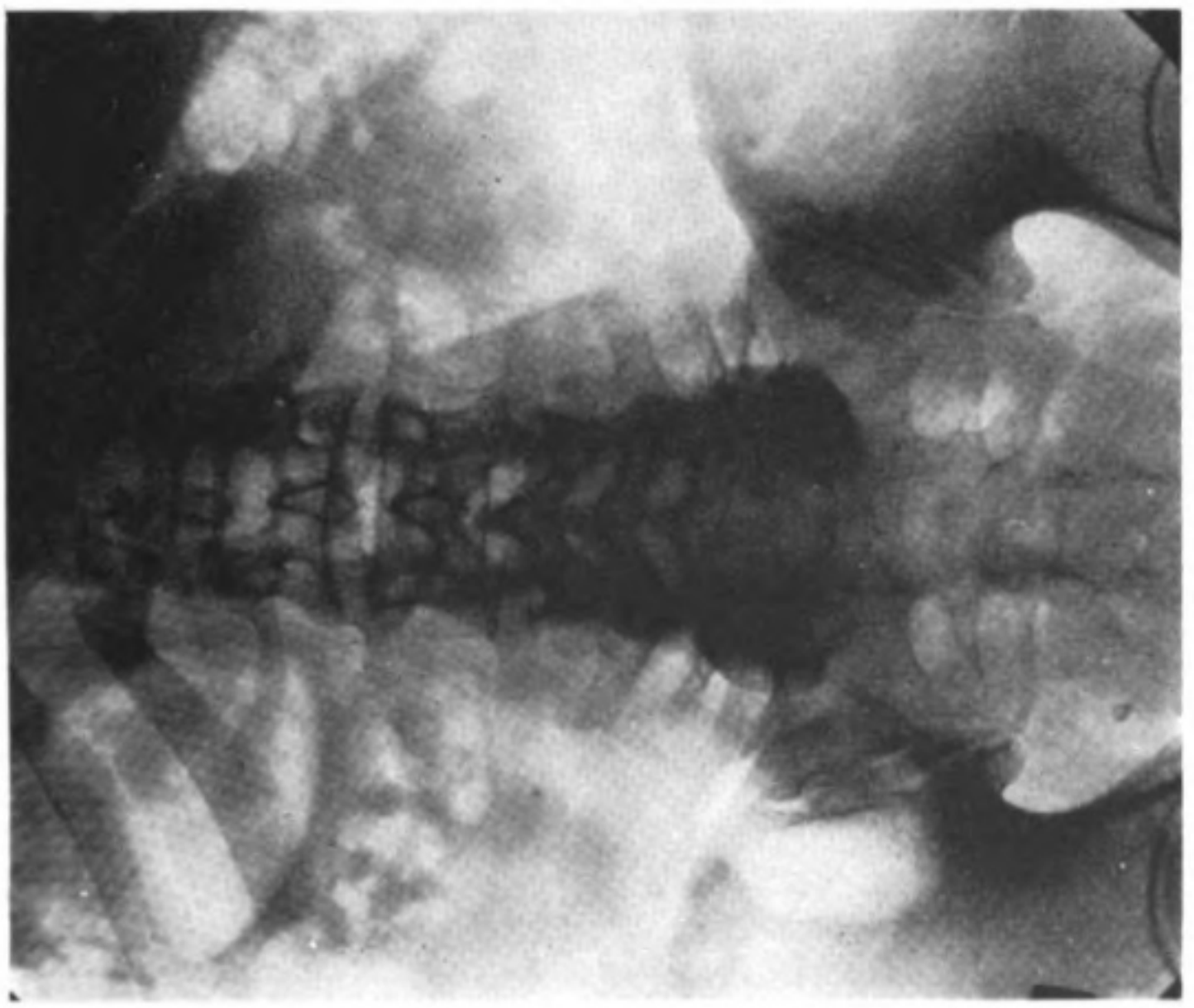
We gave him 1 micro microgram of the SSR serial system of carbonyl groups. There was a steady improvement so that he could discard the brace entirely in November, 1925. The kyphosis gradually disappeared and the pain and limitation of motion disappeared with it. Within a year the spine was straight.

The radiographs made by the orthopedist at the time showed the collapse of the bodies of the fourth and fifth lumbars and the angulated deformity. The radiographs taken after he was cured more than eighteen years showed the two vertebrae fused in perfect alignment so as to form one vertebra. In the upper thoracic spine an area or rarefication the size of a large pea was to be seen. This showed in the healed spine as an area of dense bone where the disease was cured before it could do any damage. The radiographs showing the healed state were submitted as exhibits to the Federal Trade Commission, but we did not receive them when the exhibits were returned and thus we are unable to reproduce them here. In April, 1957, two radiographs were taken of the lower spine; one an anterior-posterior view and the other a lateral view. The perfect alignment in all directions is seen. The spine is found now to be one vertebra short.

In high school he participated in sports and received a letter in track. He attended the University of Michigan. Since his graduation, he has continued to live an active life and earn his own living. In April, 1957, he reported that he is in excellent health and that his back never bothers him.

Normally, tuberculosis of the thoracic vertebrae usually results in severe deformity under conventional methods of treatment, i.e., by holding the patient

Radiograph II, was taken in April, 1957, of Mr. J. A. and shows the lateral view of the lower spine.



Radiograph I, was taken in April, 1957, of Mr. J. A. and shows the anterior-posterior view of the lower spine.

in a rigid shell for many years. Where there is present a metastatic lesion as in this case, the chances of survival are only slight.

Extensive discussion is due this disease since it supports our working hypothesis so nicely. After the dilute dose of the reagent, the most virulent tubercle bacilli, even as found in exceedingly destructive skin tuberculosis in Brazil seem to lose their pathogenicity, and the most resistant and progressive lesions have healed with complete cure, just as we have seen in leprosy. The disappearance of the fibrosis with its captured germs and the replacement with angioblastic tissue and then with normal lung parenchyma, the increase in strength and general health of the patient, even while tubercle bacilli are thrown out during recovery has been our observation for nearly 40 years. It suggests the curing of the germ as well as the patient, and implies that the germ was pathogenic only because it was sick itself — defective in its own oxidation catalysis.

CHAPTER XIX

PUS INFECTIONS

The etiological position of chronic infection in the causation of cancer requires that any treatment that will cure cancer must very efficiently cure infection. We will show by a few of the many cases cured how fulminating staphylococcus septicemia of the most virulent kind, that failed to yield to the other methods at hand, did reverse and become cured after a dose of one of the reagents reported here. Since these cases are decisive more need not be given.

ACUTE FULMINATING STAPHYLOCOCCUS AUREUS PYEMIA

With Double Pneumonia and Pylonephritis Complicating Osteomyelitis in a Boy

CASE No. 52 Dr. L. Andrews

N. R., age 5 years, took sick with 105° fever and pain in the left tibia. This was opened at the Victoria Hospital and yielded 300 cc. of pus which proved to be a pure culture of the staphylococcus Aureus. In a few days the symptoms pointed to infection of both kidneys and the urine showed the same organism in large amounts. Lobar pneumonia in both lungs appeared immediately and the blood culture showed a pure rich infection of the same organism. He rapidly and steadily declined and soon it was difficult to get him to take food and water. The fever remaining high, led to progressive weakness and a sort of mummification in spite of all of the best hospital care. His brother was immunized with a vaccine made from a pure culture of the staphylococcus aureus. Then the brother's blood serum was given to the patient. He had nine blood transfusions while in the hospital between July 13 and August 2, 1940. Neither this nor other measures as sulphathiazole made any impression on the advance of the disease. The patient was given up as hopeless and taken home on August 2, 1940, and present treatment was continued for a few more days, but with no improvement. He refused all food and even water.

On August 9, 1940, he was given an injection of the serially arranged carbonyl groups. In twenty-four hours the patient was better. In forty-eight hours, the patient was taking food. He was hungry and ate in quantity for the first time of his own choice since he took sick. He made a rapid improvement from that hour on. By September 10, 1940, the urine and blood were cleared and the lungs had considerably improved. The X-ray of the tibia bone showed a "moth eaten" appearance. On September 12, 1940, Dr. Andrews operated on

the leg to clean out the dead bone and any infection that was present. It was packed with iodoform gauze. A second injection was given after the operation.

By November 4, 1940, the effects of the pneumonia had practically disappeared. The patient was discharged. The child is strong and vigorous. He made a complete recovery.

SUBACUTE STAPHYLOCOCCUS AUREUS INFECTION OF THE PROSTATE GLAND

With Septicemia Following the Incision and Drainage of a Boil

CASE No. 53 Dr. J. M. K----

The boy, aged 18 years, while at camp in July, 1940, developed symptoms of appendicitis and was operated for it but the appendix was found to be normal. Soon afterward the pains concentrated in the kidney region and the urine showed the infection to be the staphylococcus aureus which was also found to be the cause of a superficial boil that was incised a month previously and after which all his troubles began. The pain, however, soon showed the major location of the infection to be the prostate gland. Sulpha drugs were used with other of the best hospital care that was guided by ample laboratory data. But they gave no help. The condition steadily became worse with high fever, and steady loss of strength and nutrition. Since incision for drainage of the prostate has uniformly turned out fatal, this course was eliminated and when hope was abandoned, Dr. K---- phoned the writer for an ampoule. It arrived in Los Angeles, by air mail, and was given without delay. Here again an infection that was steadily winning for months quickly reversed after receiving the SSR injection, 2 millimicrograms.

The change may be reported from Dr. K---'s testimony which has been paraphrased and reproduced here. It shows the intense interest of a father finding favorable facts. "The treatment was administered. The next day, from my observation the boy was better. He was definitely improved. There was a definite change. The change was one such as you see at a sick bed. I watched him from the next day on. The boy improved. He had an appetite then. He complained less, was less nervous and had less pain. His general condition was definitely improving until the seventh day after treatment when the abscess broke (as Dr. Koch advised it would). The pus discharged through the penis. The pus was cultured and it showed staphylococcus aureus. On the day it broke he had quite some pain and after this he was, to all appearances, well. I kept him in bed and kept him under close observation, but he was perfectly all right. His temperature was normal. He commenced to eat better. Of course, he showed the effects of the sickness, but he had no more of the septic condition. The fever never came back. This was followed, of course, by a definite and lasting and complete recovery."

In these cases two things are to be recalled. The advancing infection poisoned the nutrition, food could not be taken, not even water, and the fevers

persisted and mounted until they became chronic when the protective reactivity broke down and the mummifying process set in. The hopeless prognosis was then evident, too, and the sulpha drugs gave no help, but seemed to injure the patient as did the toxins of the infection. *It should be recalled that the activated amine groups plays* a part in both poisons. The infection steadily went forward also. Then after the carbonyl catalyst treatment, the change was for the better. The appearances changed. Appetite returned, the fever stopped, the resistance showed up by sequestration of the infection and its discharge as in the prostate case. Examination of the discharged pus showed it to be the same staphylococcus both before and after treatment. But after the treatment the infection became suddenly harmless and was quickly thrown out. This is the experience with cows with infectious mastitis, as reported by the Ministry of Agriculture of British Columbia in five years of observation in cattle infections. In a few days the gangrenous infections that lay the cows low subsides and the cows are up and about, even as the germ count increases the wounds heal.

This is an important observation as is also the *disappearance of the fibrosis* of the chronic mastitis infections in the dairy cattle. Evidently a change has taken place in the germ as well as in the patient. The metabolism of both have become normal and no toxins are produced. Thus the physiological approach which does not aim to injure or kill the germ, makes it no longer pathogenic, and the patient, too, burns his accumulated poisons out of the way so he is again hungry, even after weeks and months of inability to take food, as he should.

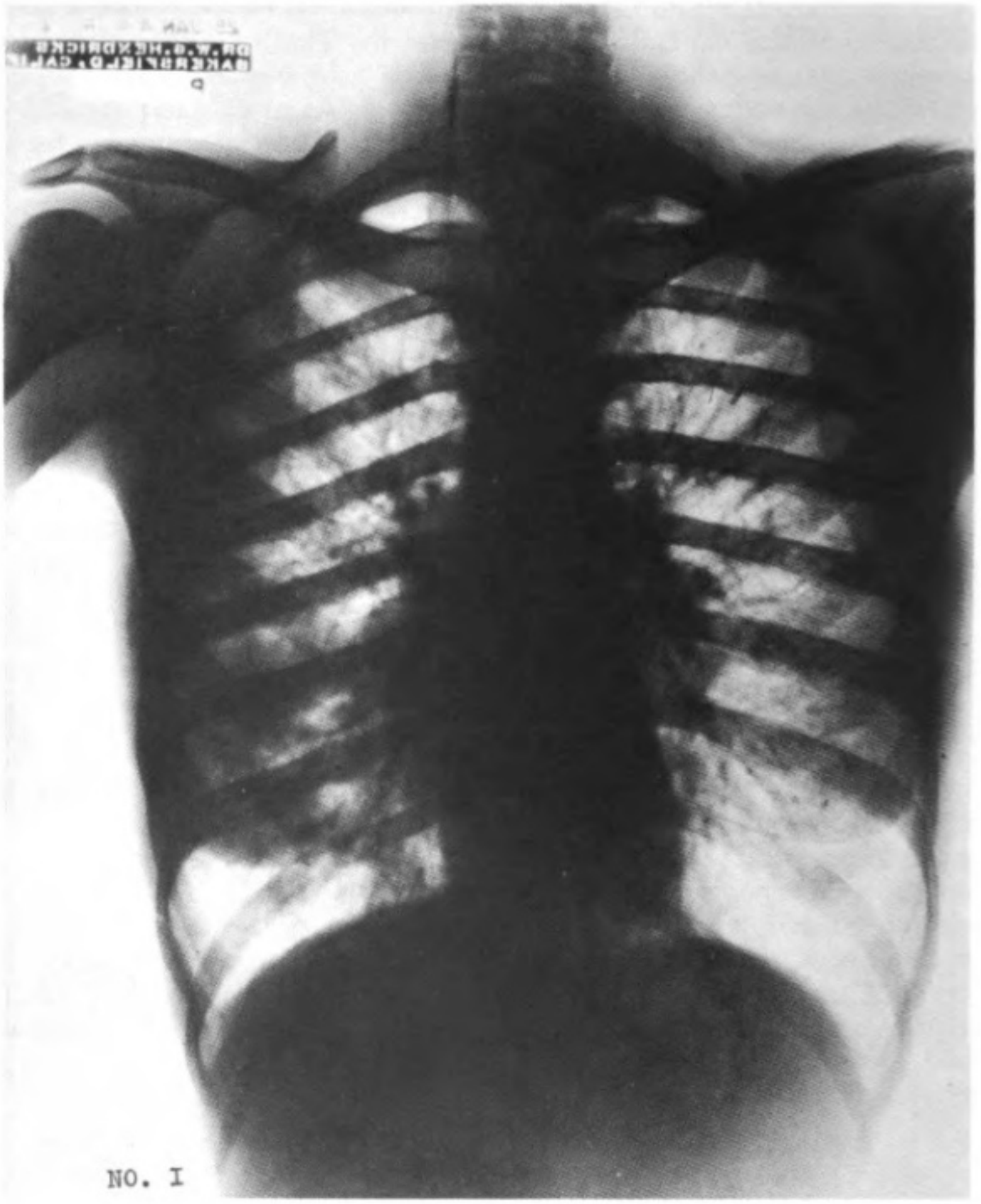
The metabolic fault in the tissues during chronic infection is well represented in bronchiectasis, for here not only is there an excavation of the lung substance starting in a bronchus, but the bronchial walls carry the infection forward so that lobectomy is the only hopeful procedure, from the orthodox standpoint. The following case like the others in our experience shows the normalization of the tissues resulting in the cure of the disease locally, and systemically, for this patient showed a terrific allergy to her bronchial infection through a most severe asthma. The correction of the fault made it impossible for the infection and its sequel the asthma to find soil, and the cure was therefore consequential to the restoration of an efficient oxidation catalysis.

ADVANCED BRONCHIECTASIS

With Asthma

CASE No. 54 Dr. Wendell Hendricks

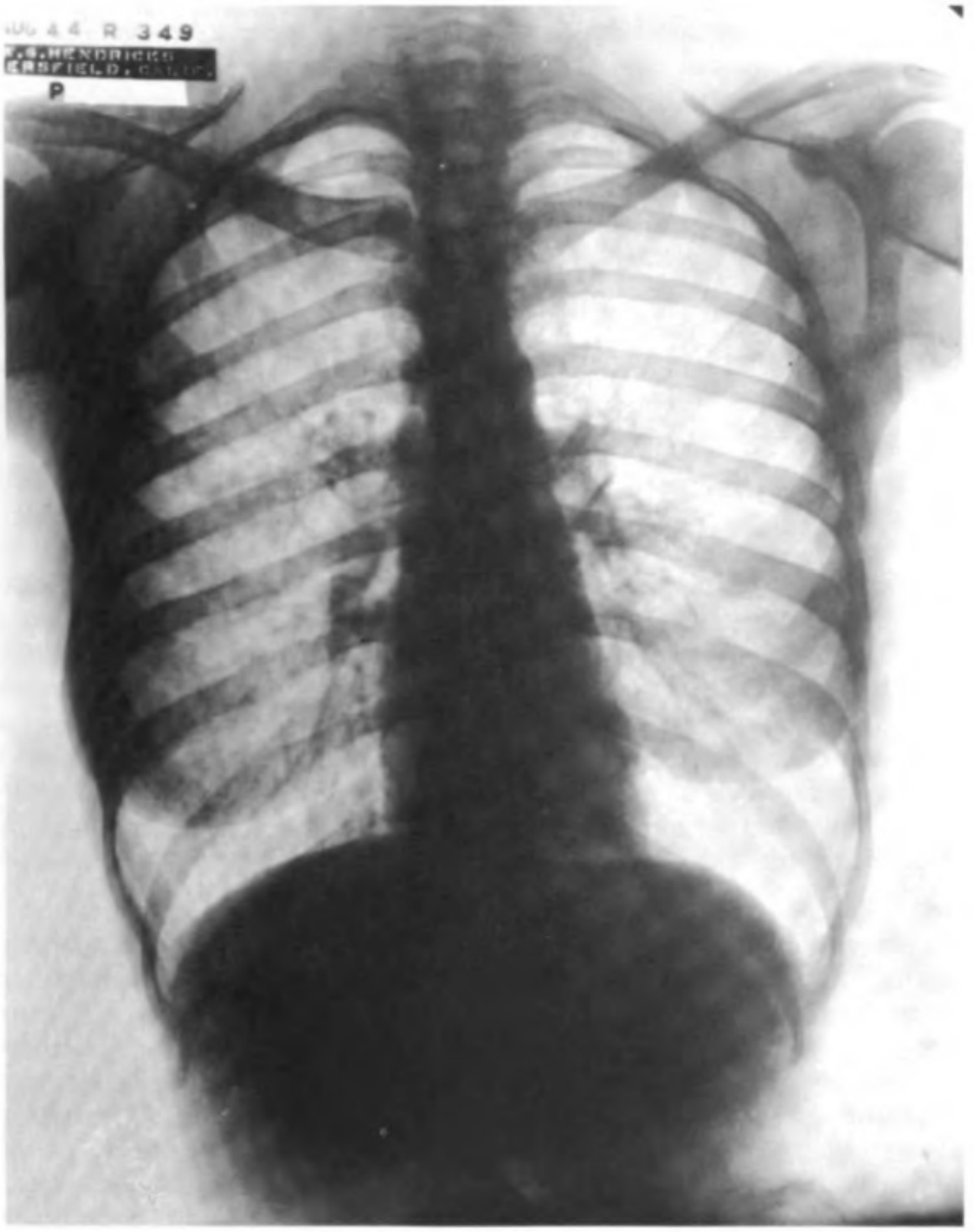
On January 26, 1944, Mrs. P., a woman of 31 years of age, was helped into Dr. Hendricks' office. She weighed 82 pounds and was in a severe state of asthma and coughing. The condition had persisted for many days and prevented sleep and correct nutrition, and brought her to the point of collapse. She raised enormous amounts of pus laden sputum for many years, but the asthma had persisted only for the past three years. A severe sinus infection was probably the initiatory factor. Her pulse was 130 beats per minute and temperature 100°. The blood pressure was 100/80. It was necessary to be propped up



Radiograph No. I, taken at time of treatment.

in bed to secure any sleep. The red blood count was 4,000,000, the hemoglobin 80%.

One dose SSR was given on January 28, 1944. Three weeks later she exhibited a reaction with chills and high fever. Following this reaction the asthma ceased, the blood pressure rose to 112/80, she gained 10 pounds in weight, and the fever dropped to 99.2°. By the 12th week the temperature was normal (98°) pulse 72, and the blood pressure 110/80. In another month



Radiograph No. II, taken August 4, 1944, showing recovery.

the blood pressure was 120/80, and she gained to 93 pounds, but real marked relief from the cough and the excessive sputum did not come until the 24th week had passed. During the 27th week she was given a second injection. She made a complete recovery. There were no more symptoms of the disease, no more pus to be expelled and no more asthma. She slept normally and lived normally again, tending to her card parties and home duties as usual, and lived a brisk life thereafter. We believe that only through the proper restoration of

the oxidation mechanism and its catalyst, can tissue vitality be restored and a permanent recovery obtained. X-ray films, taken before and after treatment are submitted in demonstration.

Occasionally during the observation visits she was given a lavage of the nasal sinuses to aid their elimination. Colon lavage was part of the general care, together with vitamins and the vegetarian diet upon which we insist in all of the cases under treatment. No honest physician or expert in this dire disease will claim that an occasional nasal lavage would cure a deeply established bronchiectasis when indeed they admit the only cure is removal of the lung or the lobe that is affected. Even such cases relapse, it is found, as the fundamental tissue weakness still remains. Only the proper restoration of the oxidation catalysis can restore the tissue vitality.

ANTIBIOTIC RESISTANT GONORRHEA

The response of the antibiotic resistant gonococcus infection to the Carbonyl Therapy depends partly on the general health of the patient, and partly on the chronicity of the case when one is comparing the time required to make a full recovery. One may compare the progress of the two young men of nearly the same age each with about the same envolvement, and both antibiotic resistant. One had the infection two years and the other four years. The former was in fair health giving a test of the red blood cells of 100% no crenation. The other was very depleted and showed a very poor crenation of his red cells in a one percent salt solution.

Case 54 A, was a boy of 22 years of age. He sustained his infection two years earlier and had received all the antibiotics on the list without stopping the specific infection. This germ had acquired immunity to antibiotics before it infected him. The urethral discharge of gonococci continued. The urine sediment was also positive to Gram negative "coffee bean" intracellular diplococci, on January 15, 1960, when he received two micrograms of benzoquinone (2 cc. of the 6x homeopathic solution) intramuscularly. His prostate was seriously envolved so that it interfered with the passage of urine and feces. Two weeks later there was still the same secretion of pus and gonococci. But the crenation test of the blood had improved to 90% crenation two weeks later and only a few extra-cellular diplococci could be found in the urinary sediment. Two weeks later the urinary sediment gave only a few gram negative diplococci. But they were Pleomorphic and extracellular. No intracellular "coffee-beans" were found. Ten days later and ever since until now, there were no symptoms either prostatic or urethral, and the urinary sediment was free of gram negative diplococci. Only a few leucocytes could be found. Thus he made a full recovery on one dose of 2 cc. of the 6x homeopathic solution of benzoquinone given intramuscularly, though the germ in this case was persistently resistant to all the best known antibiotics right from the very start.

Case 54 B, was 25 years old when he became infected in March, 1956. Besides the urethral involvement there was a venereal papyloma and a

prepuce lesion, but the blood tested negative to the lues. He received 1,600,000 units of Penicillin. There was a slight improvement and then an acute recurrence with prostatic involvement that interfered with both the passage of feces and urine. On October 23, 1959, when he received his dose of 2 micrograms of benzoquinone the urethral discharge showed the typical "coffee bean" Gram negative intracellular diplococci of Gonorrhea. There was a reaction with aggravation 84 hours later (this is typical of the recovery process). There was some pain on passing urine, but the pus discharge had become only slight, and in a few days he felt much better in many ways. The feces passed better with less prostatic interference. But on February 19, 1960, there was abundant secretion, though he had gained 7 kilos in body weight as he was overcoming his great depletion of ill health. This was the beginning of his 18th week after the treatment and is a usual reaction week in a chronic infection. But it was not a reaction that won recovery. The benzoquinone was not adequate. On March 16, the dose of SSR was given. A reaction followed in two days with headache and burning in the stomach. The secretion started to diminish the day following the treatment, and then disappeared. On May 30, he was free of every symptom and of gonococci in the urinary sediment. He had gained 10 kilos, and the urethra and prostate were normal. The urinary sediment showed only a few extra-cellular pleomorphic diplococci. He was found cured again on June 15, and has remained well ever since. It took six months for his health to reach the good level where he could react with a full cure. The SSR gave an immediate response while the benzoquinone was helpful to his general health and improved the infection considerably but did not produce a cure. For this the more highly efficient carbonyl groups of the SSR were needed and then the response was immediate and complete.

In both cases it is seen that the resistance gained by the germ to the toxic amine group of the antibiotic did not influence the response to the oxidation initiating carbonyl group. Indeed, resistance to the injury is along the same line as welcoming improvement in the vital chemistry. Evidently the germ was made non-parasitic and non-pathogenic for surely it was not injured by an agent that gave its oxidations a boost. And the patient was improved in his whole vital chemistry at the same time. Thus both patient and germ were not injured, but benefited. These observations, like so many others, show that the physiological attack on infection has a rational basis, while the pharmacological attack which rests on a destructive principle may invite failure. The data at hand again emphasize the paradox of trying to correct by way of destruction. Here success is had within certain limitations or excluded entirely where nature has full sway. The constructive philosophy on the other hand is not hedged in by limitations, and nature aids the corrective process from every side and angle. It appears here also that the sicker the patient, the sicker the germ, and the longer it takes to establish the correction. The correction of the fault in the patient and in the germ run parallel—"hand in hand." Both faults are of the same order, as they respond to the same reagent.

CHAPTER XX

FIBROGENESIS

Fibrogenesis may be general or at least at points quite distant from the source of the fibrogenic agent. Thus an infection that has become scarred-in and silent may still exist in a comparatively anoxic focus so as to evolve products of its metabolism that do not have a chance to be oxidized away, but form free radicals that can polymerize at the focus, or may enter the circulation as such. Hypoxia in any parenchyma subject to exhaustion from over work will also cause incompletely combusted metabolic products to be formed and exist as free radicals that may copolymerize with the product coming from the infectious focus. Thus there will be located at this distant point a copolymere of incompletely burned metabolites of germ and tissue cell origins which, being irritant, call for the production of a protective fibrosis as occurred at the point of infection in the first place. The toxic polymere is taken up by the collagenous material of the fibroblast and enters into the structure of the fibroblast as a free radical addition to a double bond that invites oxidative removal just like the pathogens added to the tissue cell FCG invites oxidative separation and destruction. But the fibrosis increases with toxic increase and progressively the fibrosis shuts off the circulation more and more causing a progressive vicious circle. However, when the toxic factor is burned away through the induced oxidation, the rest of the fibrosis has no reason to exist. It is obsolete and undergoes autolysis. This is our conclusion after seeing such recoveries as in Case No. 55 (page 196).

This type of fibrosis is different from the fibrous connective tissue of normal sheaths and tendons for it contains the incompletely burned metabolites which still invite oxidation, and thereby dissolution. On the other hand the normal collagenous tissues contain chondroitin sulphate and hyaluronic acid and other polysaccharides which are insured against combustion by the cyclization of the carbonyl group which is flanked by an amine group besides. Thus the skeletal structures are quite "fireproof" while the scar tissue that combats infection and toxic states is open to disintegration via oxidation. The pathogenesis thus again provides for its correction. While our oxidation catalysts may clear up a fibrosis of coronary insufficiency or renal and cerebral insufficiency, they do not touch the normal structural fibrous connective tissues. Oxidative destruction of germ toxins makes the germ nonpathogenic, in our experience, and this of course prevents pathological fibrosis.

The pathogenesis depends upon suboxidation and toxins of various kinds. Cartilages are subject to dissolution, however, not because they contain built in oxidizable units, but because of their colloidal adsorption characteristics and poor oxygen supply. Here toxins coming from a distance can be ADSORBED

and held to call forth a fibrogenic invasion which will destroy a joint cartilage, for example. Such invasion is accompanied by angioblastic tissue. Together they destroy much of the joint structure and cause ankylosis. There is enough cartilage left to initiate joint reconstruction after the invading fibrosis is reversed by the oxidation of the toxic units built into its fibers. The pathogenesis thus again provides for the restoration of normal functioning tissues, following the institution of an efficient oxidation catalysis.

To keep the tissues young, elastic, oxic, and efficient, one eliminates all infectious foci, no matter where they reside, and one avoids the fatigue of any functioning tissue, or activity beyond what can be well supported by oxygen and the catalysts involved. The ordinary industrial and road dust and smoke poisons, smoke from arsenic laden tobacco, as we pointed out many years ago when the tobacco mosaic became a menace, are common fibrogenic and carcinogenic factors. The dual nature of substances of this class is seen in the dissolution of a scar following the disappearance of a neoplasm that developed at a distance, and the transient repetition of the symptoms associated with the infection before its encapsulation by the scar. The joints may also be protected, and the vocal cords of singers may be helped to hold their very fine elastic and sensitive muscle qualities. All vital organs may be protected. See the chapter on diet also.

CHAPTER XXI

THE PATHOGENIC MECHANISM IN CANCER AND CONNECTIVE TISSUE DISEASES

When one compares the tireless activity of the child with the aches and restriction on motion as age advances, it is evident that the exchanges between the tissue parenchyma and blood stream of the child have no impediment so that waste products get out as fast as formed, and oxygen fuel and food enter as freely as is required for full combustion and tissue construction. There is no separating fibrosis between the parenchyma and the blood supply. In old age the separation reaches the limit where the exchanges are practically abolished, and finally, where life is no longer supported. One organ or other as habits and heredity have determined has suffered most at the hands of the fibrogenic agent, and cerebral paralysis or heart or kidney failure may be the immediate cause of death.

We have identified the initiating factor as an activated amine which causes a gellation of the plasma and cellular colloids and this gellation blocks the transport of oxygen causing a local hypoxia or anoxia. In addition to producing the gellation as we have observed results from the guanidin poisoning in our parathyroidectomy work, the amine condenses firmly with the tissue function carbonyl groups so as to block its initiation of energy producing oxidation chains. Incompletely burned metabolites of tissue cell origin or of germs caught in the anoxic area, then may be dehydrogenated so as to become free radicals but under the anoxic state can not be burned. Since the amount of dehydrogenation accomplished under such circumstances is small at any time, a slow polymerization of the metabolite can proceed. This is especially true where scarring has reduced the oxygen transportation. The free radicals, be they of tissue cell or germ origin, may copolymerize with each other, too, or with the collagenous material that is produced by fibroblasts in response to the irritant effects of the incompletely combusted state. Thus they are incorporated into the fibroblastic tissue intended to dispose of the toxicity. Cancer cells likewise we hold take up the toxic products into their structure in the same way and thus serve as detoxication agents. (Koch, Cancer and Its Allied Disease, 1926, Koch, Cancer Journal, October, 1924). As the fibers increase by added deposition of collagenous and toxin co-polymeres the last depositions must present the highest molecular weight, and as each such group produces a characteristic set of disease symptoms, each fiber carries a history of the intoxication from the inception of the anoxia. The same appears to be the case for each cancer cell as well. If the cancer cell could complete the combustion of the toxin, it would offer protection and serve the immunity mechanism, which we

feel is its intended goal (Koch, Cancer Journal, October, 1924) in antitoxic hormone evolution.

Our observations showed that the flu epidemics of the first world war gave cancer a boost, and the symptomatology was much more uniform after this event. As a rule the pregrowth symptoms were found to exist five to twenty years before the growth came and the longer they existed in the parent with cancer, the shorter their appearance in the offspring that developed it. Thus the pregrowth toxic period became shorter and the growth appeared earlier in life besides, by some five to ten years as a rule, in each successive generation that showed it, — quite a virus characteristic.

The symptoms are generally a neuritis which may be rather violent, and exist in the arm or shoulder in cases that develop gastro-intestinal neoplasms. It may show up as a sciatica, or a dizziness, or an epilepsy, headaches, visual disturbances, etc. The nervous tissues seem to be the favorite site for the toxic action, but there may be a psoriasis, some skin allergy, or a compulsory neurosis, while the lymphatic reticulo-endothelial system undergoes some atrophy. Before the growth comes, for some six years or so there may be a gain in useless fat, of a watery variety and weakness develop concomitantly. After the growth comes the symptoms disappear wholly or in part, only to return again if the growth is removed, and again disappear with the recurrence of the growth. Depending upon the rate at which the toxin is produced as compared with the rate of growth of the neoplasm and its adsorbing and copolymerizing with the toxin, the latter is stored out of the way more or less and the symptoms will disappear proportionately. (Koch, Cancer Journal, October, 1924; Koch, Cancer and Its Allied Diseases, 1926).

To illustrate this situation, it can be done no better than to paraphrase a letter received in Brazil from Pasadena, California. It runs as follows: "In 1932 I brought my mother to you from La Crosse, Wisconsin, with cancer of the stomach. She was an advanced case, and the symptoms she suffered for years before the tumor was discovered are being repeated in me. I am asking your advice about an exploratory operation to see if I have what my mother had, and the doctor's examinations here indicate that I have, and only time will give the full proofs . . . your treatment was given my mother and she got well and lived fifteen years longer and died of a heart attack . . . I started out with a terrible neuritis in my left arm, and could not raise it above my head. It increased in severity until I had to carry it in a sling for months. It gradually left there and went to my sciatic nerve, I also fell and hurt my knee. Tetanus antitoxic serum was given me and I developed a terrible urticaria that nothing helped except Cortisone. Then my body became covered with boils and I am taking an antibiotic for that, but have to keep it up. Before the trouble located in my abdomen, I began to take on weight and dieting has not helped a bit. Obstructive symptoms in the abdomen are the trouble now, and the neuritis has improved a great deal. This is the way my mother's cancer developed." Such reports are the rule, with slight variations. But there are

exceptions also. In this case the pregrowth toxic symptoms were easily contrasted with the symptoms of the neoplasm.

The fact that the toxin causing the symptoms can add to the structure of a developing fibrosis or of developing cancer cells, shows that it presents either highly polar double bonds or free radicals as we explain later, and hence exists as a sub-oxidized residue of some sort of metabolism, tissue cell, germ or virus in the sense offered below, and operates as we diagram later. This incompletely combusted state, and the anoxia that governs it are the important factors we must consider to secure a correction of the pathology.

So long as fibrogenesis can absorb the pathogen there will be no neoplastic response. But when this defense of the mesoderm fails, and the reticuloendothelial cells of the spleen and lymph glands become exhausted, the way is open for the pathogen to attack epithelia and cause cancer. This defense of the fibroblastic mechanism is a physiological hyperplasia to combine and wall off irritants and germ products. On the other hand the neoplastic hyperplasia as seen in spindle cell sarcoma lacks the FCG to keep the activity under control. In the malignant situation the FCG is inactivated and requires liberation, to resume correct behaviour. In the protective hyperplasia situation the SSR is needed to burn up all combined and circulating toxin including that in the germ that is its producer. Then the fibrogenesis is obsolete and involutes. So in both situations the SSR is required to remove the cause.

VASCULAR DISEASE

Arterial sclerosis and thickening of the interstitial connective tissues are protective attempts to dispose of a toxin, an imperfectly burned metabolic product of the tissues, or of some germ hidden in an old scarred-in focus of infection where anoxic conditions prevent the full combustion of its products. They then have the chance to polymerize, passing through stages that are pathogenic in different ways that cause various diseases preliminary to the production of cancer. The fact that the cancer patient gives a history of a series of disease states before the growth comes, which after the growth is made to disappear, repeat their appearance briefly in reverse order to their coming is observed very regularly, and it indicates a depolymerization of the toxin from the cancer producing stage, down through each disease producing form until the monomeric form is reproduced that was first produced by the acute infection. Indeed in cancer of the breast after the tumor is absorbed one observes a sudden acute swelling of the tonsil area of the same side, and a sore throat that lasts a week or so, and then clears up with the disappearance of any scar tissue or hardened lymph nodes in the area. A breast case may get a final reaction in an old scar in some other part of the body, after the growth is absorbed. Thus, we link cancer and its allied conditions with germ toxins or viruses developed in anoxic areas.

One of the pregrowth symptoms or changes is vascular sclerosis, general, or in some organ as the brain, kidney, or heart, accompanied with an interstitial fibrosis. The toxin can here integrate with the developing fibroblasts

that come to give protection against the poison, or its co-polymer with an incompletely combusted tissue metabolite. It may be taken as a rule that fibrosis protects against cancer so long as the tissues show the ability to produce the necessary fibroblasts, and when this ability fades away, the toxin can keep on polymerizing until it can produce cancer, or let us say the fibrotic protection is lost before it achieves that property, and for this a hypoxic focus is necessary, a scarred-in tonsilar crypt or focus of infection. Then one must agree with our first experiences that the carcinogenic agent was primarily fibrogenic.

In Case No. 55, the fibrosis with all its tissue accompaniments of depressed oxidations, such as cholesteral deposits, calcified plaques, parenchymatous hypoxia and malnutrition and blocked elimination, had reached the limit. Still, the restoration of FCG function cleared up the whole pathology in a progressive undoing of the pathogenesis. The "disease building" was torn down starting at the "roof," and new tissue was healed in where the repair was needed. In the coronary cases, however, besides the diseased arterial walls and interstitial hyperplasia, there was the element of blood gellation that caused the acute attack. The circulation was blocked by the gelled blood as in the parathyroidectomy situation, but true coagulation would not have taken place for a while anyway, though it was inevitable, had not the Survival Oxidations been restored. Therefore, the recovery was just as fast as the restored oxidations could charge the blood colloids and restore their correct dispersion. In acute apoplexy, this has only taken a few hours at times. The coronary cases thus picture the acute action of the toxin along side the chronic action, and the Senile Sclerotic case and the Bright's disease case expose the chronic action, with the reversal of the pathology.

Here we see in the polymerization tendency, the action of the free radical and the double bond. This free radical, when it gets a chance, can add to a double bond of fibroblasts, and cause the sclerosis, or to the FCG system of the parenchyma, and cause various allergies or other disease pictures, like piteriasis rosea, which has been used as an example, or of psoriasis. And when it polymerizes to the point where it has reached the correct steric advantage it can add to the FCG of the mitotic mechanism of cells to cause cancer. It is our opinion that wherever it has integrated with a tissue FCG system and interfered with function it stays there undisturbed. But it undergoes destructive oxidations as fast as exposure permits at the same rate no matter where it is lodged. Thus the orderly reversal of the pregrowth symptomatology is possible.

CHAPTER XXII

SEQUELAE TO INFECTION

FAR ADVANCED ARTERIOSCLEROSIS

With Senile Dementia

CASE No. 55

Mr. P., age 93. He was treated in April, 1933, with an injection of the serially arranged carbonyl groups. He was a painter by trade and for some years was experiencing the effects of advancing arteriosclerosis. I had personally observed this change as I saw him at long intervals when I checked up on his wife, who was one of my first cured cancer patients. This woman had had a complete obstruction of the pylorus to involve the liver and other organs. She made her recovery on two injections of the carbonyl catalysts in 1918, and remained well thereafter. It was at a call to check her condition that she showed me her husband lying in bed on his right side with knees bent some and unable to move at all. All the muscles were spastic. He could not speak and had to be fed and cared for like a baby. The heart was dilated and palpation of the radial artery showed a high blood pressure.

He presented a marked arcus senilis and heavy tortuous nodulated pipe stem blood vessels. In the previous year he had had several "strokes" and passed into senile dementia. The whole condition was an extreme senile change. The skin elasticity was completely lost.

A dose of the carbonyl SSR was given and in a month a definite improvement took place. In seven months he was able to dress himself and walk about. He was rational and discussed political matters expertly. In a year he was able to lay a small cement sidewalk in front of his home and do other work. At that time the blood vessels had lost 80% of their sclerosis and all nodulations and the extreme tortuosity. The skin had lost its cyanosis and had regained its elasticity. The blood pressure had fallen to a high normal range for his age, 180 over 110. He remained well for three years longer and then died suddenly.

The reversal of the sclerosis in this case depended upon the oxidation chains that converted toxin into its antitoxic type of structure and so the recovery was progressive to the limit after it was well started. Years of accumulated incompletely oxidized metabolites of tissue cell and germ origin were gotten out of the way and the sclerosis they supported and which had absorbed them to inactivate them, had no more utility and left also, as the toxic factors were burned to completion.

CORONARY THROMBOSIS IN EXTREMUS

CASE No. 56 Dr. H. B. Mueller

Mr. L. E. had been under the care of Dr. H. B. Mueller for some two years before the present complaint developed, on January 16, 1944, and for minor complaints only. After a short, easy walk on January 16, 1944, numbness accompanied by pain occurred in the left arm from elbow to wrist. Almost immediately afterward pain developed in the epigastrium and extended to the throat. He never had suffered such severe pain before in his life. It was in the arm and precordial area. He was frightened and did not care what happened. It passed off in a few minutes and he went home. In twenty minutes the pain recurred with full severity, and did not respond to nitroglycerin, nor until two hypos of morphia were given (½ grain).

Family history shows that his mother died of senility at 89, and his father was still in good health at 86 years of age.

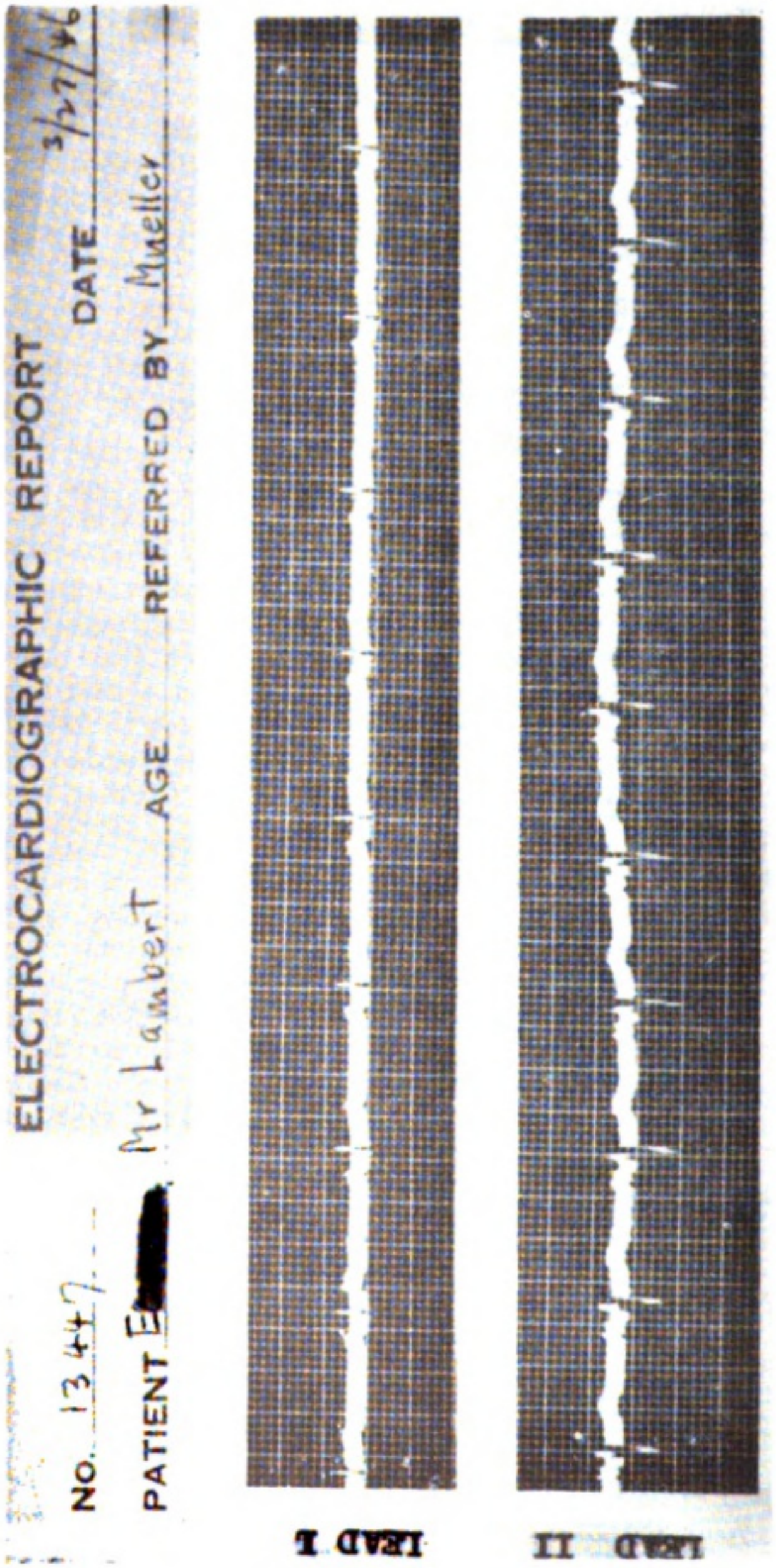
The past history showed three or four years of occasional attacks of "palpitation." They were transient and forgotten quickly, after he was quiet a few minutes. Bowel action regular, no nocturia, except once during the past month. Sleeps well more than 8 hours out of 24, has a mild cough slightly productive, smoked a package of cigarettes per day, recently reduced to three per day. Can do considerable work ordinarily without undue fatigue. No dyspnea. Weight as regularly, is 157 pounds. Height five feet, ten and a half inches. Age 47 years.

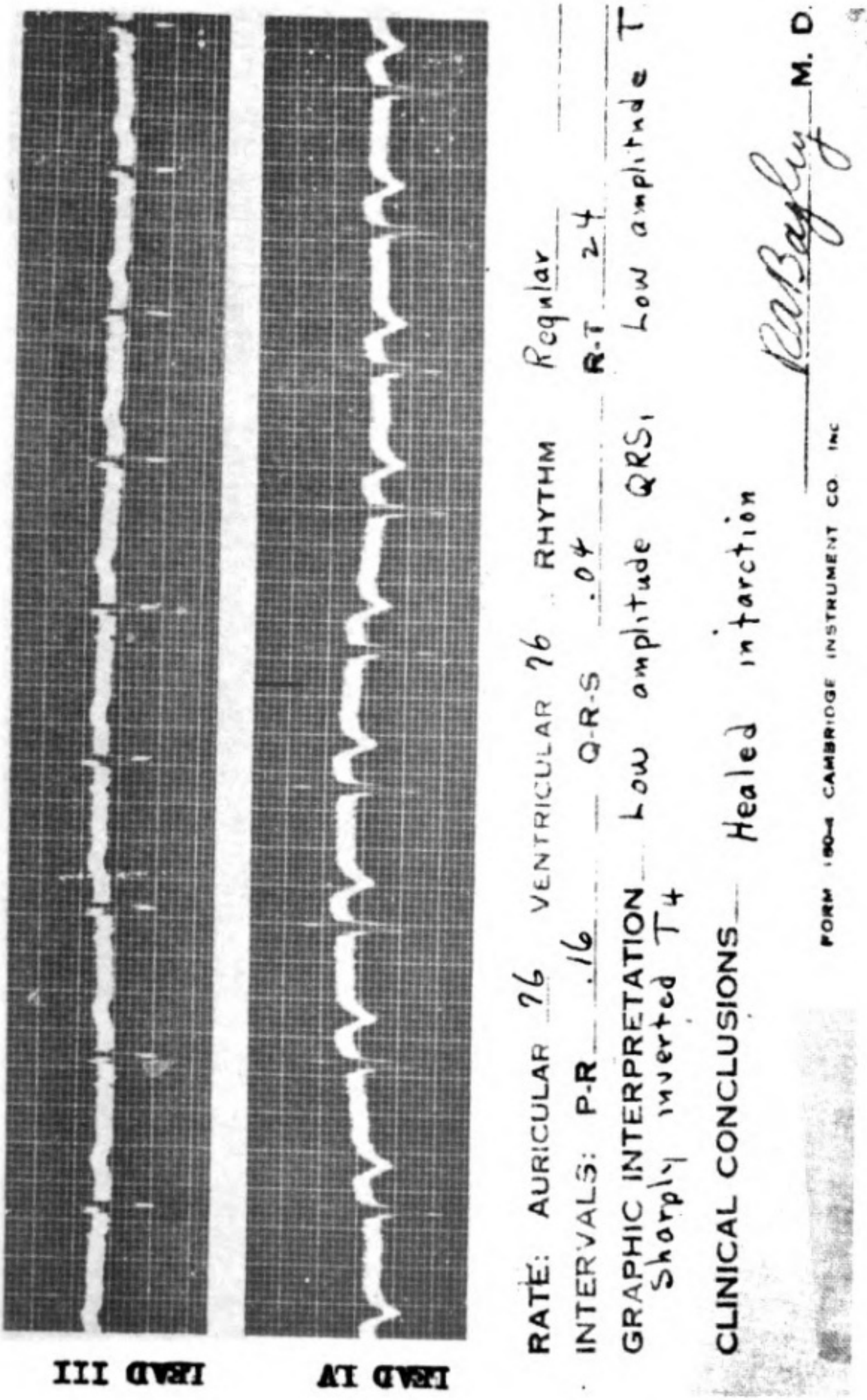
Physical examination at the time of the attack showed a man in complete collapse, snow white, heavy cold perspiration, almost pulseless, shallow gasping breathing — in a dying condition. He was given two micromicrograms of the SSR system of carbonyl groups on January 20, 1944. He responded dramatically within the next half hour, but was held at complete bed rest on a light vegetable diet and without any medication whatsoever. He was kept in bed for eight weeks, and was well enough in another month so that he could climb the three flights of stairs to his apartment daily without discomfort, and was again fully active back to work within six months.

On April 15, 1944, his physical examination showed the heart apex to be in the 5th interspace one inch inside the M.C.L., the sounds were not well heard, no murmurs, pulse Mod. vol., regular in force and rhythm, 85 per minute. Blood pressure 96/68, right, reclining after rest.

Electrocardiogram on this date showed rhythm regular at rate 85, P and PR intervals are normal throughout, T_1 shows a late sharp dip. T_2 and T_3 are upright and normal. T_4 is deeply inverted. QRS complexes show low amplitude. QRS_1 — +½—1; QRS_2— —3; QRS_3— —5. There is absence of the R wave in lead IV. *Conclusions:* Low amplitude of QRS complexes, inversion of T_1, absence of R_4, suggest *healed infarction at apex of left ventricle.*

(signed) R. A. Bagley.





Electrocardiogram taken two years later, March 27, 1946 by the same expert reads as follows and is submitted. Regular rhythm at rate 75, P, and PR intervals normal. Amplitude of QRS_1 — 1½. Low T_1, Inversion of T_4.

Clinical interpretation: Low amplitude of QRS complexes and inversion of T_4 indicate healed lesion — probably posterior infarction. There is improvement over previous tracing.

Remarks: On this date, patient appears well clinically. Blood pressure is 110/70. The pulse is regular at 76. The heart shows no enlargement, and there are no murmurs.

(signed) R. A. Bagley

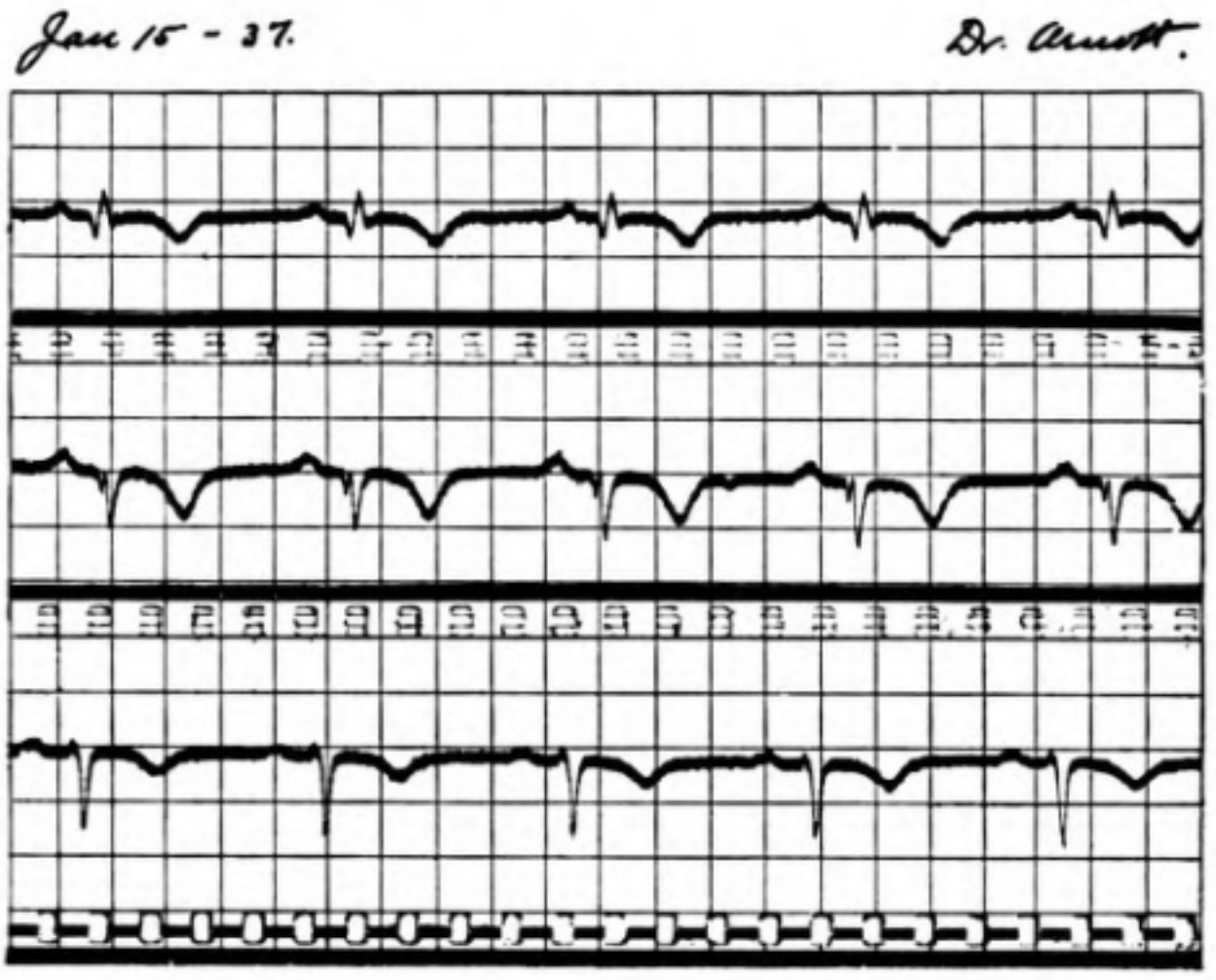
This patient was last seen by Dr. Mueller on June 27, 1951. The patient felt so good at the time that he thought medical observation was no longer needed. This was over seven years after treatment. He continued in good health until the fall of 1955 when he had what was reported as a subsequent attack of coronary thrombosis and died. This was over eleven years after treatment and a long period of good health.

We attribute this subsequent attack of coronary thrombosis to a return to the living conditions that were etiologic in producing the disease in the first place. It took over eleven years to restore sufficient pathology to cause a coronary infarction again while no further treatments were given. Had this patient remained under medical observation by his physician, followed the recommended dietary living and received subsequent treatments, if and when advisable, we feel that he would be living today. Thus this case also illustrates the importance to the patient of continuing under proper medical observation and healthful living.

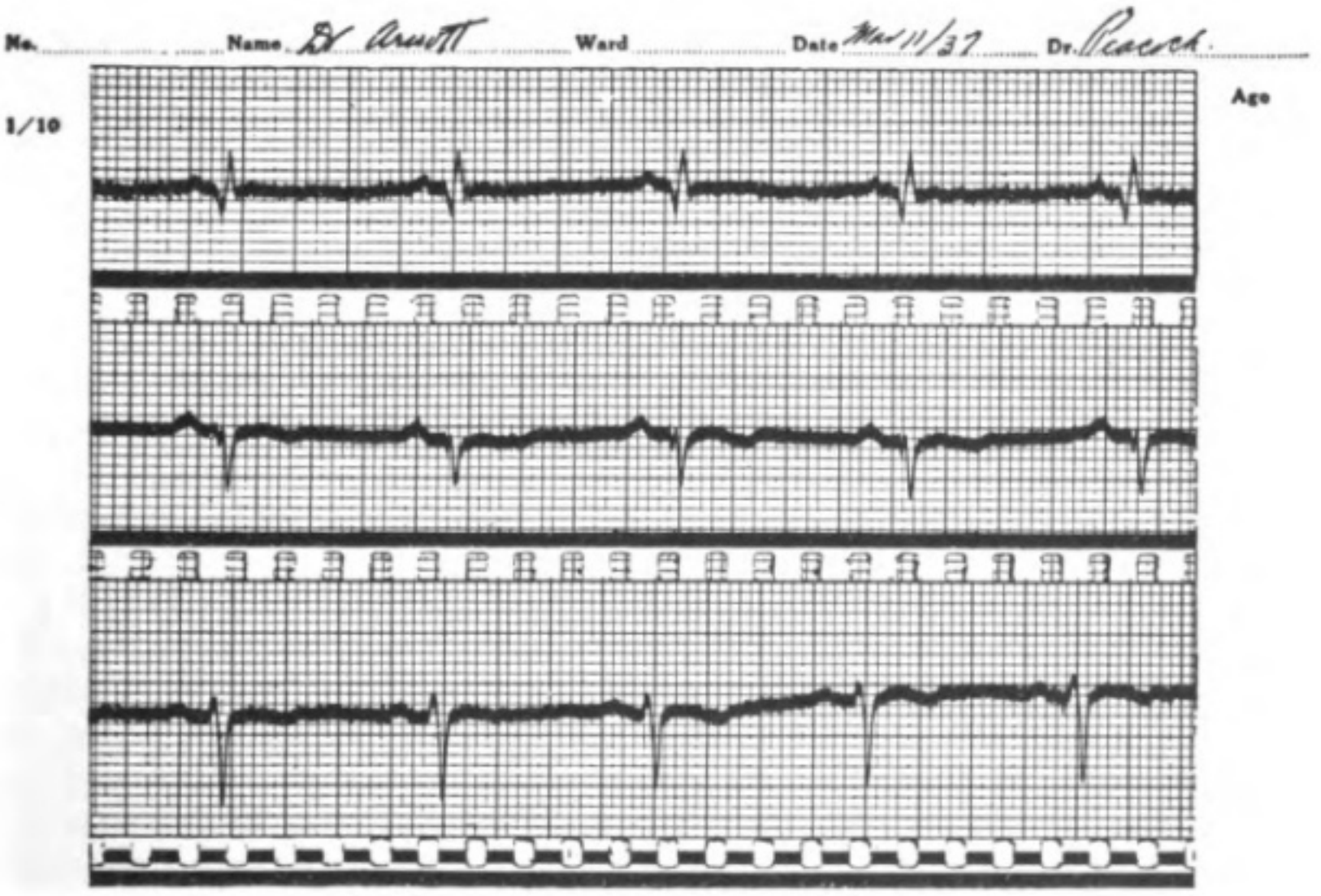
CORONARY OCCLUSION

CASE No. 57 Dr. David Arnott

Dr. A., age 64, brisk and active habits, was taken with a mild attack while walking on December 2nd, 1936. This passed off in a few minutes after resting. Two days later an extremely severe attack followed while he was resting. Repeated heavy doses of morphia hypodermically influenced the pain only when sufficient to stupify him profoundly. The slightest lengthening of the intervals between injections was followed by severe pain. On December 8th, 1936, the SSR was given subcutaneously in a dose of two cc. of the 12X dilution. Considerable relief was had in one hour. Eighty-four hours later another dose was given after which the pain soon disappeared entirely and has not returned. The opiate was discontinued after the first injection of the catalysts and none has been required since. A careful convalescence was followed with strict observation of the diet and of good bowel hygiene. Effort



Electrocardiogram taken as soon as possible after treatment, shows profound pathology.



Electrocardiogram II, taken eight weeks after the first shows good recovery. This was made three months after treatment.

was reduced to a minimum until the repair of the lesion was satisfactory for ordinary activity.

The electrocardiograph could not be made during the attack and the first one which is reproduced here was made five weeks later. It still shows a profound pathology. But the tracing taken eight weeks later shows a good return to normal. He remained active and well for nearly fifteen years and died at the age of 79 years from a prostate operation sequel.

BRIGHT'S DISEASE

CASE No. 58

Mr. C. L., lawyer, age 40, let his insurance payments lapse and to be readmitted was required to pass a physical examination. The urinary findings showed advanced chronic Bright's disease in harmony with his symptoms of elevated blood pressure and severe migraine headaches, that lasted three days to a week at a time. He was given 2 cc's of SSR in March, 1925, and made a steady recovery so that the headaches ceased after the sixth week. One year later the urinary findings were normal so he applied for readmission to his life insurance. The company physicians examined him on surprise occasions and secured urine specimens by catheterization. After a year of such tests they concluded that he was cured, and accepted him on the usual basis of a healthy man of his age. He lived in good health, free from nephritis and from migraines, for twenty-three years and died from an abdominal injury.

CHAPTER XXIII

ALLERGY

PITYRIASIS RUBRA UNIVERSALIS

CASE No. 59 — Dr. E. Klaveness

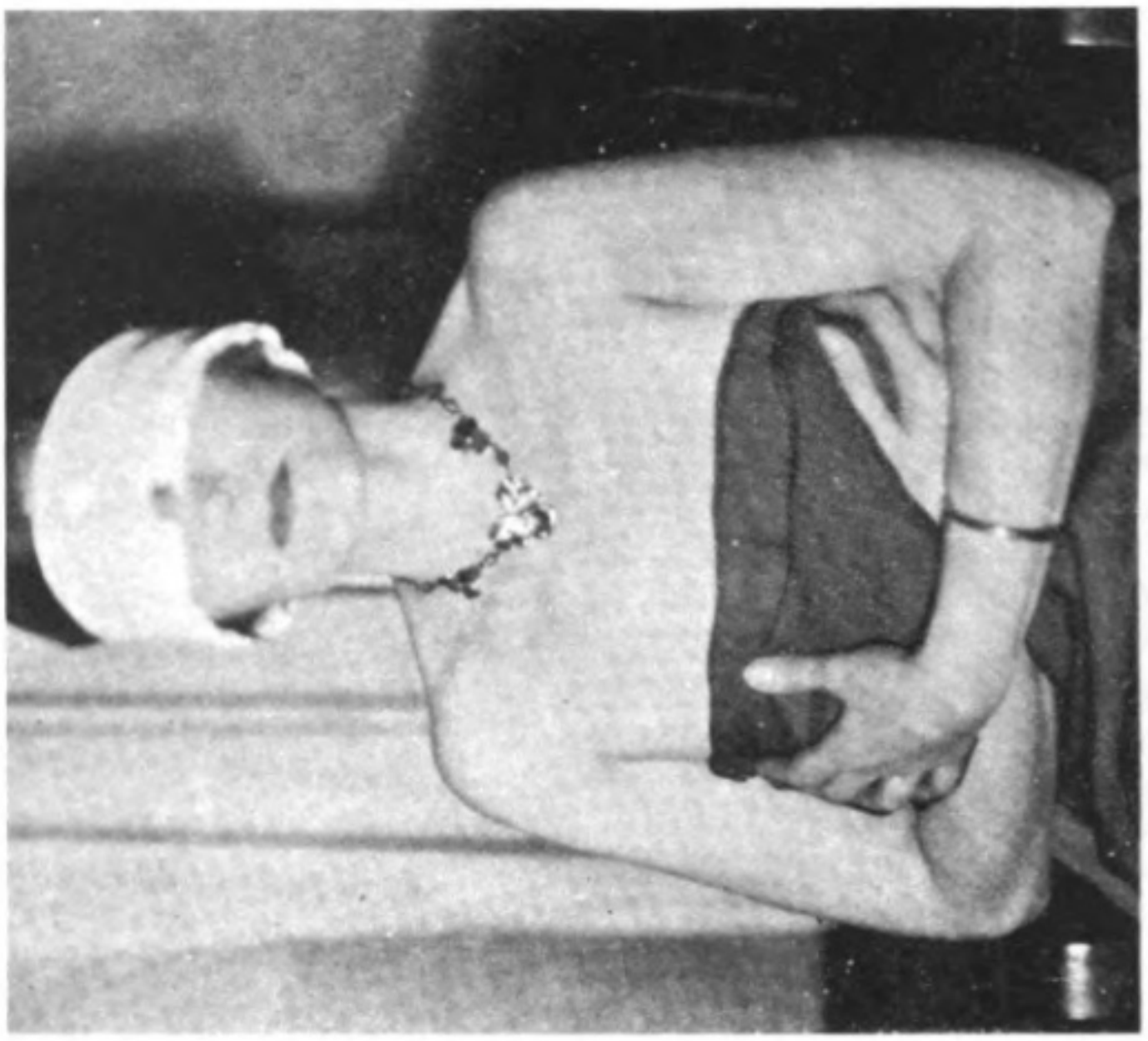
This "incurable" disease recovered after two doses of the carbonyl catalysts. This case in a man of 58 years, treated February 25, and in April, 1950, had been suffering since October, 1948, from a skin disease that answered the von Hebra description perfectly and was diagnosticated as Pityriasis Rubra by all available skin specialists, and also by Dr. Klaveness. While at St. Joseph's Hospital from December 16 to February 23 he lost 20 pounds, with terrific loss of scales daily, and steady deterioration in all respects. Normal weight, 193 pounds; Feb. 25, 1950, 172 pounds, showing pronounced erythema of skin from top of scalp to soles of feet, no papules, no blebs, no marked infiltration of skin which was richly covered by small thin scales, curled up from the periphery on itself, moderate itching. Inguinal glands enormously enlarged, felt chilly even when heavily clothed. The urine showed albumen 2.5 grains per liter.

Recovery set in promptly after the SSR injection with no loss of weight. The scaling stopped, first on scalp then the trunk following the first treatment and scaling stopped on the extremities after the second, with change to normal color, gain in strength and weight and clearing of the urine of albumen, so that by July 22 he was discharged as cured. Found in good recovery also August 26, and September 9th. Examination then showed the whole situation normalized with the inguinal glands very much smaller, possibly normal.

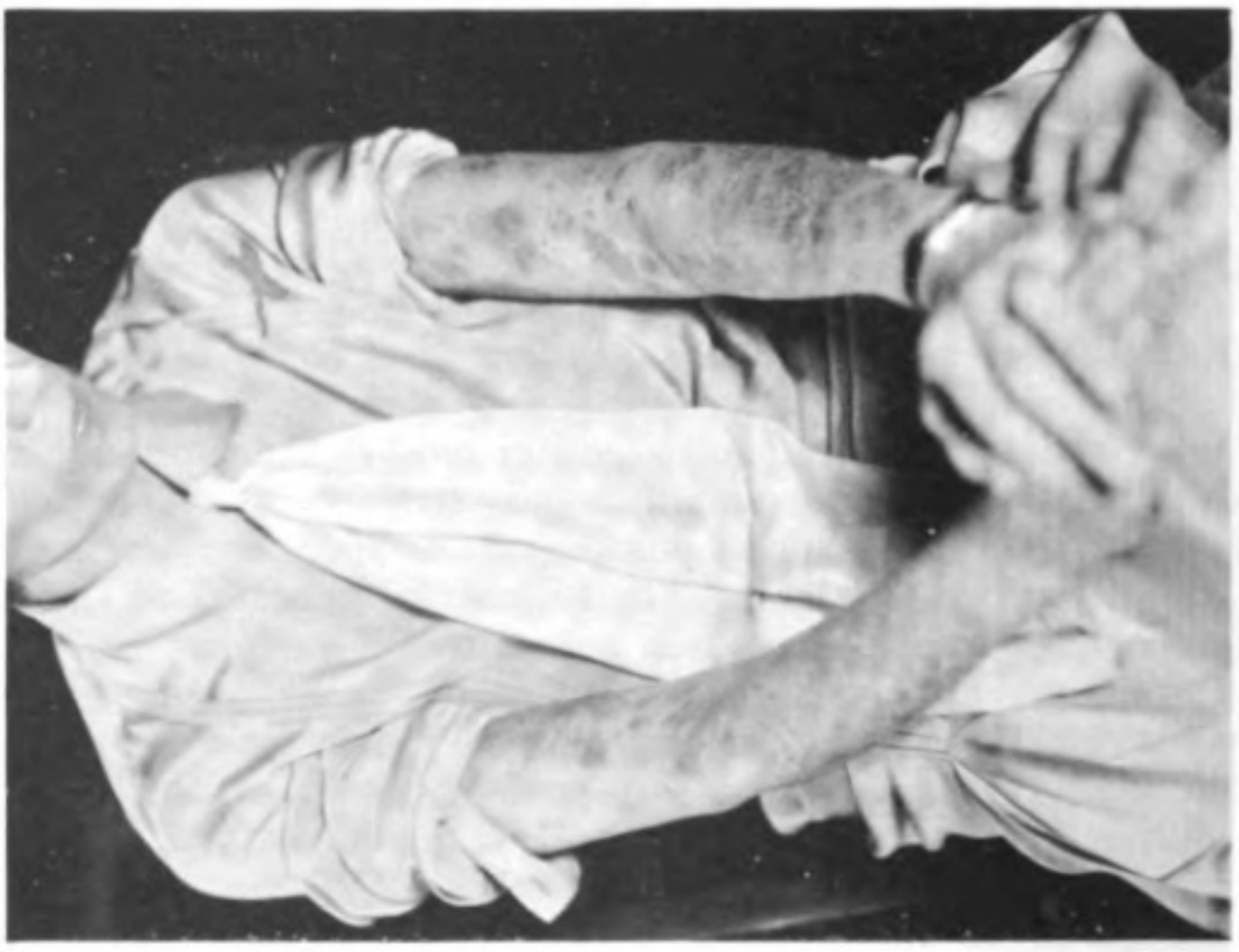
ACUTE FULMINATING PSORIASIS

CASE No. 60

While psoriasis is generally slow to recover and often disappointing, some cases recover rapidly and permanently. This is especially true of the acute type. An example is Miss N., age 32. (Her brother had psoriasis chronically and severely). The psoriasis came a month after an attack of tonsilitis with an acute tachycardia on changing posture as a sequel. She was much sicker than a case of psoriasis usually is. The lesions first appeared on the left thigh and spread rapidly in spite of the best attention of the experts until it covered the entire body affecting the hair and nails in usual fashion. Some of the lesions were deeper than we generally see and especially those between the head and ears. She was given the carbonyl catalysts by the writer on April 2, 1926, and a reaction followed on the fourth day with chills and fever and general achiness with an inflammatory reaction in the tonsils. Thereafter, improvement began to show in the last lesions to come. This improvement continued with slight aggravations in the congestions of the lesions during the third, sixth, and ninth weeks. In between the whole condition improved rapidly so that by the



Photograph No. II, showing patient after recovery.



Photograph No. I, taken at time of treatment.

twelfth week only a few very small spots were observed and these were absent at the fourteenth week. She remained well, thereafter. The tachycardia recovered right with the psoriasis. The photographs were taken at the time of treatment and at the fourteenth week. She remains well to last report in 1946 when she offered to testify at the court trial.

As in this case, we have offered enough examples of toxin-host cell integration where both structural and functional changes resulted in different tissues. The psoriasis cellular defect and the neuromuscular control of cardiac rhythm both responded to correction at the same time with the withdrawal of the toxin through the agency of the same molecule. Here again is an indication that the type of toxin ligation with the host cell in both instances is the same. And after all the structural change in the psoriasis may be considered a functional matter, for epithelial cells of the dermis function by reproduction to afford protection. Both phenomena present hyperfunction beyond physiological control, and thus conform to our old definition of allergy. (Koch, Cancer and Its Allied Diseases, 1927) (The Chemistry of Natural Immunity, Koch, 1936). Neoplasia falls in the same classification, so the clinical evidences in humans on a wide front elucidate a simple pathogenic process to be met clinically, simply, and with complete success, in correctly managed cases. If pathology were always interpreted physiologically there would be very few serious defeats for the clinician. We will see that this is true for animal diseases as well.

PSORIASIS UNIVERSALIS

CASE No. 61 Dr. Chester Dove

In this case of Mr. C., age 64, when treated in July, 1934, showed universal redness from top of head to soles of feet with terrific amount of silvery scaling large and small, leaving a bleeding base with loss of hair, eye lashes, and finger and toe nails. The soles of his feet separated off as foul gangrenous sloughs. He suffered day and night without the least relief from the best medical attention available.

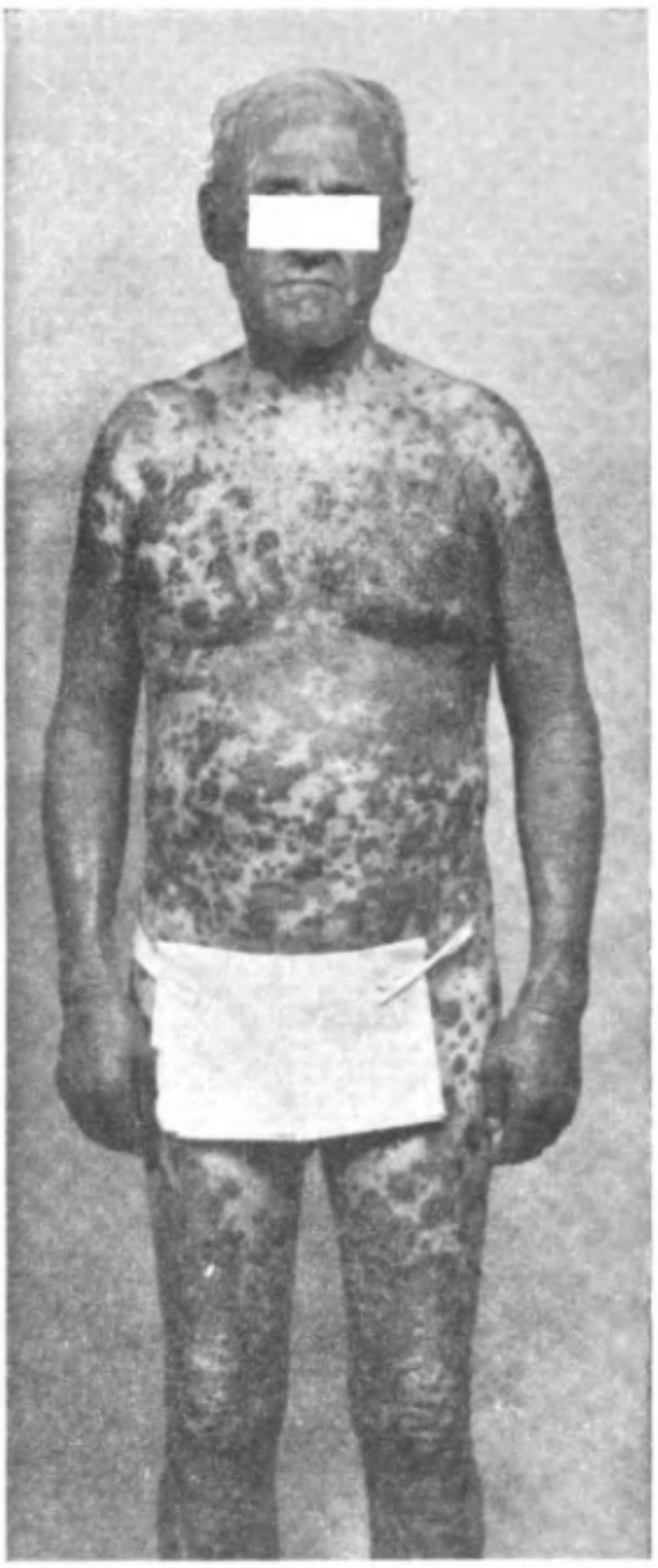
The photos show the situation before and after treatment (P. 206).

He received two injections of two micromgms. SSR, one on July 17, 1934, and the other in October, 1934. He made a complete recovery. In February, 1957 he told Dr. Dove that there had been no recurrence of the psoriasis since his recovery in 1935.

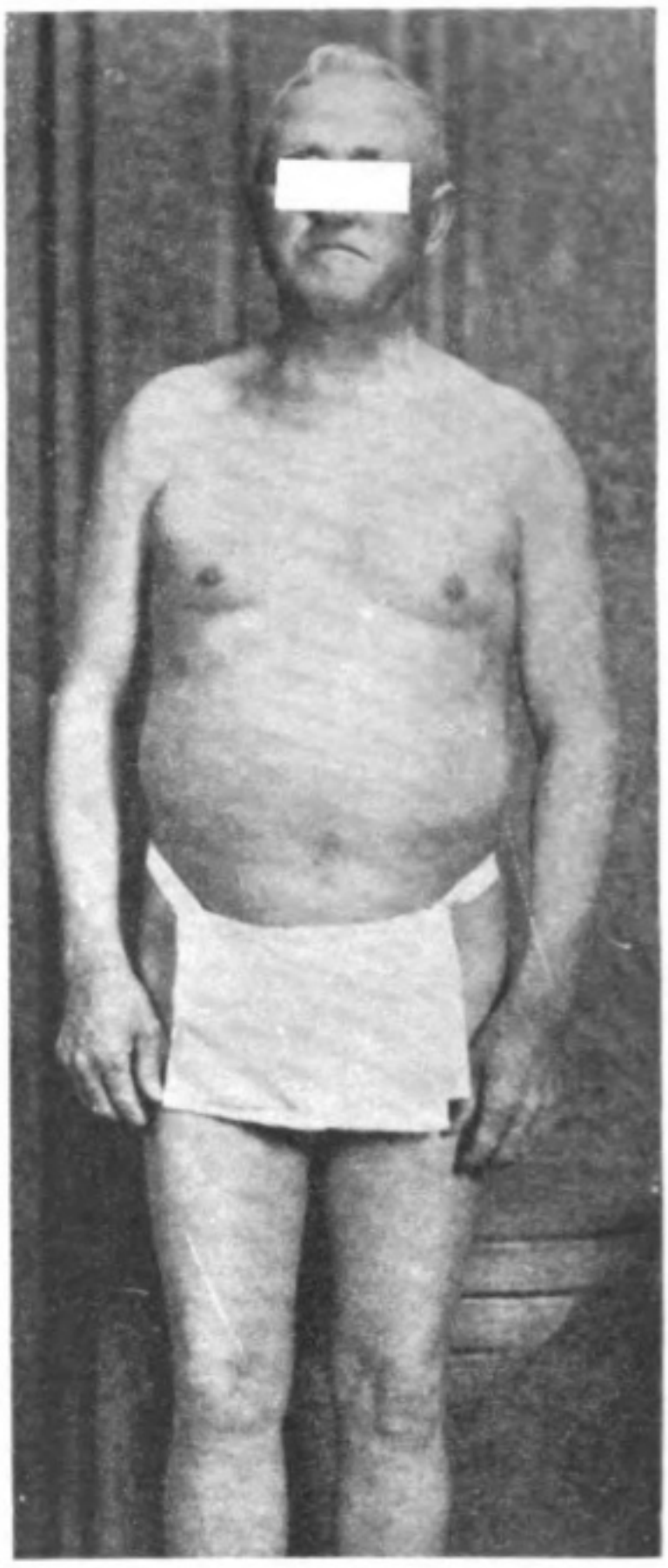
MULTIPLE ALLERGY

CASE No. 62

Mrs. R., was 45 years old at the time of examination and treatment in May, 1934. For a year and a half she had continuous, intensive hay fever and asthma, burning, watering eyes, and running nose. She was found at the



Photograph of Mr. C. taken at time of treatment



Photograph of Mr. C. taken after recovery.

University of Michigan Hospital to be sensitive to 60 different things including fur, feather, and most foods. These she avoided most carefully but still suffered as badly. Continuous nasal sinusitis for years.

There was a continuous urticaria of the hemorrhagic type that added to her misery. There was also a disturbance of the bowel that expanded with gas at every eating, and an incontinence of urine. This was seriously troublesome.

We gave her two micro micrograms of the SSR serial arrangements of carbonyl groups with free radical terminals. In three weeks she was much improved in all symptoms, was able to sleep on a feather pillow, keep a

dog and eat what she pleased. The recovery was complete in nine weeks and has so remained. The sinus infection became well also but was not fully cured until the twelfth week had passed. Here, too, the first condition to come, the sinus infection that supplied the allergenic toxin, was the last to completely heal. However, the toxins produced there were in large part immediately induced to undergo oxidation and were made harmless.

While the allergy affected the secreting and involuntary muscle contractil fibrillae in this case, it often affects the nerve impulse generating mechanism or the conductile fibrillae in other cases. The following two cases show that the toxemia may be expressed by a cerebral allergy, in various ways. Psychic suggestion may have an instigating effect in nervous cases like a pollen does in the hay fever type, but we believe the fundamental pathology in all is the block in the oxidation process which has to be corrected.

ALLERGY OF CEREBRAL CENTERS

On Infectious Basis

CASE No. 63

This patient is representative of the most serious cases of the common allergies. A less frequent type which illustrates the response of the psychic section of the nervous system is, also submitted.

The patient was the Rev. A., age 52. He came in December, 1938, for a serious sinusitis that involved the left maxillary sinus most severely. He had this infection for over five years and nothing used helped at all. He reported that for the past few years he suffered from a compulsion neurosis that did not yield to treatment of any kind. When hearing a train whistle, he was forced to let out a yell at the top of his voice. We did not go into a psychoanalysis or ask whether or not he had a shock or fright associated with the sounds of a train. No matter what such shock might be, the toxin at the base of the trouble made the synapses hyperactive so contact was extraordinarily easy and impulse transmission easier than normal. Hence, removing the toxin should restore normalcy of function. Or we may say the nerve cell bodies were under higher energy evolvement because of the energy poured into their mechanism toxically. Thus only a slight impulse received would set off a maximum response that could travel a whole neurone system with a force much stronger than the inhibiting impulses could manage. He was given but one dose of Benzoquinone, 2 cc. of the 6X homeopathic solution and the sinus infection and the compulsory neurosis both cleared up in three months. He has reported no trouble since.

It is noteworthy that the nervous symptoms stopped immediately even before the infection was all cleared away. Thus the toxic state was first corrected and as in the other cases reported here, the bacteria can be considered non-toxic after the oxidation catalysis has accomplished its work.

ALLERGY OF CEREBRAL CENTERS
On Neoplastic Basis
Dual Personality

CASE No. 64

Another case of cerebral allergy is that of a woman of sixty with a massive carcinoma of the stomach. For two years she suffered a delusion that the air was full of needles and pins that she was breathing. Any drink brought her was full of the same sharp objects and her husband put them there. She recovered both from the cancer of the stomach and from the delusions. Even though the delusion of seeing the air full of needles was excited by hearing a phonograph play in the neighborhood, the delusions existed on a toxic basis as was demonstrated by the complete cure of both conditions at the same time as the result of the use of the oxidation catalysts. One dose was given of the serial system of carbonyl groups with free radical terminals, on July 20th, 1924. She remained well until 1943 when last seen. At this time she explained that the "crazy notions" she had she knew were not true, yet she could not help but believe them. In this case a dual personality existed in conflict with each other. Thus one section of the brain was affected by the toxin while others were not.

Therefore the basis for Freud's hypothesis of abnormal psychic states falls flat, since he does not consider the oedematous effects of toxins on the synapses that take part in any concept. Nor does he consider the transfer of energy to the nerve impulse generating mechanisms in the brain cells that result from fluorescent toxins, as we are dealing with in this case and the previous one. His whole system must be altered to meet this newer information on the subject.

ALLERGY OF MOTOR CENTERS

CASE No. 65

The allergy may involve motor coordinating centers, and manifest itself by continuous repetition of the same motion without ceasing, as a phase of a highly toxic insanity that failed to respond to all known therapies, even to 1500 doses of metrozole and cardiozole, and the course going steadily toward death. Such a case was given to Prof. Renato Souza Lopes and the writer in 1941 by Prof. Roxo, the renowned professor of nervous diseases of the University of Brazil. The prognosis was fatality within two weeks so that if after the benzoquinone injection we offered to give, he would live three weeks, this would be a sign of favorable action, according to Prof. Roxo.

The patient was a young man of about 23 years of age. He had been ill for months without significant remissions, but each forward step of the disease was more severe than its preceding state. The man was raving, swinging both arms in the same order of motion with constant repetition, just as the secretory cells of the mucous membranes keep on acting in hay fever, or the bronchial musculature keeps on contracting in asthma. We gave him two cubic centimeters of the 6X homeopathic dilution of benzoquinone. He started to improve within twelve hours and was sent home on the fifth day as cured.

CHAPTER XXIV

PERCENTAGES AND CAUSES OF FAILURE

In the acute infections, especially the severe type, there is no diet problem, as the patient is not able to take food and has generally vomited what he had. Intestinal lavage tends to the rest and the injection is given in greater concentration. The recoveries have been quick with clean tissue repair. Even after the best antibiotics have failed and the nurse advises the doctor his patient will not live till morning, an ampoule of the Survival Reagent has changed the trend immediately to recovery and in a few days the double pneumonia is a thing of the past. The etiological factor is out of the way before the patient is tempted to violate the regime.

In all chronic cases it is different. Where the eating and drinking habits of the past rule the mind, recovery may go on so long as the patient is under controi, but when he is well enough to go free, he falls into the way of life that led to his illness. In cancer cases the etiological factor is not gotten out of the way entirely, until the growth is completely absorbed and the focus of infection that gave rise to the toxin is cleaned out and absorbed by a late reaction. Even then some old scars as from an early syphilis may still hold malignant cells that happened to drop in that way, and a still later reaction may be needed to clean these foci out, although they generally clear up before the original focus of infection has been cleared away.

Breaking the regime before one is fully cured, and the cure is "seasoned," permits carbonyl group antagonists to develop and possibly wipe out the defense. Amines produced from meat in the colon, the harmful nitrogenous derivative in coffee or tea, the tars of smoke and coffee, sulphides in coffee or sulphides developed in the intestinal tract by bacterial action on eggs and meat, and sulphides in the drinking water, these all hinder or wipe out carbonyl activity and block the activating power of the conjugated double bond systems. Patients are usually grateful that there is a regime worked out that helps them get well, but all are not, and perhaps 30% will desert the regime as soon as they think they are well, which is always too early, and then there may be a slow reversal from the recovery status. Perhaps thirty percent of our patients waste their chance to get well because of gluttony. Others have been ruined by irradiation and while they may improve so they think they are well even for as long as ten years, they are not truly cured and never can be. Ultimately an irradiation anemia will conquer the corrected chemistry. In other cases, where extensive explorations or exposure of the abdomen to seeding of the malignant cells during a corrective operation, the healing following absorption of the neoplastic tissue may cause widespread adhesions which on contraction compress the viscera and prevent their function. Gall bladder and intestinal obstruction may thus take place, or the pylorus may heal shut. At times the adhesions are so

dense it is impossible to correct the situation surgically. Embolism is an occasional cause of defeat in rapidly recovering cases. Thus, in some series of cases of far advanced type only 46% are reported cured by experts with this treatment. In some series where most cases are not in the terminal stage, and one would look for a high percent of recovery, only 72% have recovered. And among the failures, some were not caused by giving up the regime too soon. Something in the system destroyed the reagent, so it had no effect.

CHAPTER XXV

OBSERVATIONS IN ANIMAL DISEASES

We are indebted to Dr. David Arnott for development of the use of the carbonyl catalysts in the diseases of dairy cattle. The enormous amount of data meticulously built up by the scientists of the Ministry of Agriculture and the University of British Columbia, Canada, showed the basic place of this therapy in the tissue oxidation processes that use sugar. Thus some 95 to 100% of cases of acetonemia are quickly cured by a single injection per animal in both the acute and the chronic cases. The hundreds of cases treated for infectious mastitis show a rapid cure in the acute cases with hemolytic streptococcus and staphylococcus cases approaching 90% while the chronic cases, with much fibrosis, showed something like an 80% cure rate with restoration of function, and replacement of the fibrosis with normal gland tissue. This is the only therapy that has ever demonstrated this result. In Brazil we showed that the fibrosis that completely invaded the udder and closed the teats after local treatments with various antibiotics, could be completely cured by this therapy, too. The fibrosis and the infection it contained were eliminated by replacement with normal functioning gland tissue.

However, the reports of the Minister of Agriculture of British Columbia to the Parliament in the official bulletins of years 1944 through 1949 inclusive not only verify our working hypothesis, in general, but also supply bacteriological counts before and after treatment which indicate the pathogenic germs of highest virulence before treatment may rapidly drop in numbers a few days after treatment when the udders are undergoing healing and also that where the injury was very severe, the bacteria at times increased in number during healing, and while the toxicity of the animal was rapidly disappearing. Our interpretation of this oft observed affair, especially in gangrenous mastitis, is that the germ became no longer toxic, as we see in huge tuberculous cavitations in man, and indeed appear to help in the clean-up process. As soon as the tissue debris is eliminated, they rapidly disappear, even before the cavity or lesion is healed by tissue reconstruction. They thus appear to have shared in the benefits from the carbonyl catalysts and become normal useful members of the biological economy, and help clean up the mess they formerly caused.

Brucellosis

It was the cure of dairy farmers suffering with Brucellosis that gave start to the treatment of this disease in cattle in Canada by Dr. Arnott. The cure percentage ran somewhat over 80% in dairy cows, and this is what was recorded in the Michigan experiments, as well as those conducted in a small number of cows in Brazil. In the latter cases which I treated for the Ministry of Agriculture the cases were far advanced and of the broken down type. One had a severe infection of the udder with the diphtheria bacillus which completely

involved three quarters and half of the other quarter. It was resistant to all forms of treatment and pronounced entirely hopeless by the University Pathologist who had supervised this case. They were all cachectic with "moth-eaten" fur, or with an arthritis, ulceration, infertility or some other complication. Of five* such cases four gave birth to normal calves at normal term, and the placenta in each case was found to be structurally and bacteriologically normal, and free from the Brucella germ. The other case aborted within three months of receiving the treatment, but no follow-up was had to determine if the cure came after the third month which is usually the case. The cow with both brucellosis and Corynebacterium mastitis was fully cured of both infections — a surprise to all observers. She gave birth to a normal calf and the placenta was proven normal and free from infection. No Brucella germs were found, and the udder normalized completely without fibrosis, with return of full lactation.

Absorption of the fetus no longer occurred after the treatment and thus the normal reproductive physiology was restored whether the interference was a matter of dietary insufficiency, or from selenium in the plants, soil, or water, or if the injury came from the Brucellosis germ. High potency oxidation catalysis removed the interference and restored tissue function energy production so normal behaviour could be resumed. The subject of infertility in cattle is treated by Dr. Bruce Richardson in his graduation thesis from the University of British Columbia, and by Dr. Wood the professor of Pathology. Their recovery percentages were about 72% while those in Michigan ran much higher. Thus the environmental features deserve consideration, and these are vastly different in the two places.

Infertility in cattle as in man may have a complex origin and many factors may be determinative. Thus imperfection in food, toxins of various origins, and those as selenium coming from the soil are definite causes. Yet the poison of Brucellosis is most important. Correction of the feeding may be somewhat helpful, but in the confirmed cases a basic boost to the metabolism able to burn the hindering toxin out of the way is needed.

* This experiment started on 10 cows, of which observations on 5 were not carried through.

CHAPTER XXVI

DISEASES OF THE ARTICULATIONS

For accuracy's sake a few words of review on Arthritis should be welcome. Several types of arthritis have been classified. Anderson's Pathology, page 1255, 3rd edition, and Karsner, 7th edition, page 809, give the main characteristics in a simple practical way that disposes of any uncertainty.

Rheumatoid Arthritis goes by the following synonyms: Atrophic and Proliferative Arthritis in adults. In children it is known as Still's Disease. If dominant in the spine, the sacroiliac and hip areas, it is called Strumpell-Marie Spondilitis. It is a systemic disease of unknown etiology and is characterized by *chronic* and *progressive inflammatory* involvement of the articulations, and by atrophy and rarefaction of the bones and muscles. Eighty per cent of cases are between 20 and 50 years of age. It may show an insidious or violent onset, pain, swelling, stiffness, redness, early in the disease; warmth and thickening of the soft tissues about the joints. This swelling goes with muscle atrophy, causing spindle shaped digits. A striking feature is exacerbation and remission irregularly for months or years. There is early inflammatory cell infiltration, but no suppuration. As it progresses granulation tissue at the perichondrial margins grow in and cover the articular surface. Concomitantly cartilage is invaded and replaced by well vascularized connective tissue showing moderate inflammation on both sides causing fibrous adhesion (fibrous ankylosis). Articular cartilage is destroyed causing permanently stiffened joints. There is twisting and bone atrophy from disuse, muscle and skin atrophy, and nodules somewhat resembling the gumma are common changes. Others than Anderson state that the fibrous ankylosis may ossify to make the ankylosis permanent. This is regarded as certainly irreversible. There is a general systemic degeneration present that shows a toxic or infectious basis. The tissue degenerations do not improve but get worse with each inflammatory aggravation.

Osteo-Arthritis, also called Degenerative Arthritis, or Hypertrophic Arthritis and Chronic Senescent Arthritis, comes mostly after the third or fourth decade. The primary pathology is in the cartilage. The large weight bearing joints are affected first. Heberden's nodes appear on the finger joints.

The gross and microscopic changes in all stages indicate *regressive cartilage changes, that progress continuously, but at various rates, throughout the life of the individual* (Anderson, page 1263). The cartilages undergo reduction, fragmentation, splitting in the vertical plane, become softened and mossy in appearance, and break loose, and disappear in smaller or larger areas, leaving denuded bone, that may become polished and grooved. Marginal perichondrial

cartilage forms and breaks down causing "lipping." It is associated with stress and nutritional handicaps. This description from Anderson will help understand the pathogenesis and the corrective process or reversal that takes place during the recovery reactions. This disease, by the way, as Anderson emphasizes, has *NO REMISSIONS, but is continuously progressive* at varying rates of speed *throughout life,* but there are no remissions as in Rheumatoid Arthritis.

It is well known that in this disease acute aggravation of pain and acute difficulty in using a joint is due to a piece of cartilage breaking loose and locking the joint causing pain, etc. This goes with the advance of the disease, as progress of the structural degeneration. This is not an inflammatory affair. Should an exacerbation subside it means that other pieces of cartilage have broken loose and take positions that relieve the impediment, or that the pieces are pulverized. This is also a part of the progressive degenerative change in the joint structure as the disease progresses. This is not an inflammatory disease, but a progressive degenerative disease leading to thickening of the joints and their ankylosis. There is no remission in the advance of the structural degenerative changes, and the progress of its effects — ankylosis. No treatment in scientific medicine is known that will halt the continuing advance of its structural degenerative changes, much less reverse them.

From the Federal Court records and Federal Trade Commission testimony, a case of Osteo-Arthritis will serve as a factually uncontradictable demonstration of the reversal of this disease when the FCG is put back in commission. We will also give one cure of Rheumatoid Arthritis of the most advanced type — one showing boney ankylosis where remission never takes place. The reactions in such cases might be compared with those in acute Rheumatic fever, an example of which will be submitted.

OSTEO-ARTHRITIS

CASE No. 66 Dr. Mantor

Mrs. M. M. was 52 years of age when her trouble started in 1938. She went to the Mayo Clinic, where nothing was done but make the diagnosis. They gave her no medicine or treatment. She was steadily becoming worse. In June 1943, she went to Dr. Mantor for treatment. The trouble was pain, enlargement and stiffening of the joints, mostly of the right knee. She testified, "The joints kept getting worse and worse until I was not able to walk without a chair or something." She had to discontinue work and hire help to run her rooming house, from which she had her support. There were no remissions, but steady "worsening" in all respects. Her arms were somewhat affected, but mostly her legs and feet — five years of continuous increasing misery. The pretreatment period was one of steady progress of the disease, and steady loss in her health.

Treatment and Post-Treatment Progress

After Dr. Mantor's examination of Mrs. M.M., he gave her two micro-micrograms of the Synthetic Survival Reagent (SSR) on June 15, 1943.

There was no change visible until the ninth week. The joints were enlarged, hard, and nodular, with increase in the boney structure. This all remained stationary after the treatment until the ninth week, so he repeated the dose on August the 14th, nine weeks from the first dose. Her reactions were severe. She testified to the swelling of her feet beyond the usual enlargement, soreness of her flesh, and "every bone in my body ached; chills, right knee swollen, stiff, pains like lumbago, pains in right leg and muscles, pain under right shoulder blade, dizziness." This lasted pretty well from August to December, when she began to get better. She continued to improve and on April 8, 1944, she took a job working at the local country club. The improvement was steady after December, and included the decrease in the boney enlargements of the joints, especially the right knee. Thus, the structural pathology was normalized, and with it, function returned to what may be considered normal. When she gave her testimony three years later she walked up the high court house steps as easily as any normal person, could stoop and pick up things from the floor like a normal person. The restoration of normal structure of the right knee was demonstrated so any one could see it.

She kept a record of her symptoms and reactions which, on study, are typical of the recovery course followed after this treatment, thus proving the mechanism by which the recovery was accomplished. She has remained well, the last report having been received in 1949.

RHEUMATOID ARTHRITIS TERMINAL STAGE

Pretreatment Control Period

CASE No. 67 Prof. R. S. L.

Major O. M. N. was age 49 years, physician in the Brazilian Army. His condition started three years previously with pain and stiffness of the neck and right shoulder. This progressed until it had involved all the joints of the body and each progressed to complete ankylosis including the jaws and there was a narrowing of the optic foramena, causing restriction in vision.

When seen in October, 1941, the muscles were markedly atrophied. He had been bedfast for a year without the ability to move his arms, legs or head more than a half inch. The joints were atrophied and deformed and the articulations fixed by boney unions demonstrated by the X-ray and by simple palpation. He had to be fed through a tube as he could not move his jaws. Coronary sclerosis was identified by his experts as part of the pathology. At this stage remissions never take place spontaneously. The damage is done. He also suffered with constant migraine.

Diagnosis — Marie-Strumpel Syndrome, with universal atrophic, ankylosed Poly-arthritis.

Treatment — He had received the classical efforts without any improvement. His natural resistance was diminishing and he was developing a

dangerous anemia. The fever was constant and the pain severe. On October, 1941, he received two micrograms of parabenzoquinone in two cc. of water.

Post-Treatment Progress

In thirty days there was improvement. The temperature became normal, the headache disappeared, the appetite improved and he felt stronger. In six months he could sit up in bed by his own efforts. In nine months he was able to stand up a few minutes and walk a little. At the end of twelve months he left the hospital. His diuresis returned to normal. His articulations returned to about 90% of normal. He could get about freely and felt fairly well. He returned to active army duty and remained well until 1947 when he contracted pneumonia in the wilds of the highlands of Parana during a campaign with severe exposure, and died as a result. In this case the reversal was complete, even of the osseous ankylosis, and it was permanent.

Several other extreme cases of Rheumatoid Arthritis have been encountered and the recovery course and results were the same, thus establishing a pattern.

Discussion

In all of the cases of arthritis treated so far, whether rheumatoid or hypertrophic arthrosis, the recovery reactions are characteristic for the type of disease. In the rheumatoid form the reactions are cyclic and repeat the former symptoms he exhibited from the very inception of the trouble. The last reaction is generally a sudden red inflammation of the joints with considerable pain as if he were attacked with acute rheumatic fever. This happens even though the onset in such cases is insidious. One has the impression that if the patient had a frank, full attack of acute rheumatic fever, the condition would have ended there and not dragged along as a chronic progression of degenerative changes. In Osteo-arthrosis (hypertrophic), with some large joints mostly affected, the reaction after once starting, stays right with the pathology, and the joint is ankylosed until the whole pathology is corrected. Then it is ready to function normally. In one case the right hip was enlarged and hard like a medium-sized pumpkin. He was bedfast for some time before treatment — many months — but after treatment the reaction took hold and in six months the hip was normal. He rapidly gained his strength back so that by the ninth month he could climb a mountain with ease, and was back to steady work. Thus, the pattern of recovery is characteristic for each type. The etiology is different as demonstrated by the different recovery course in each of the two types.

Nevertheless, in both types the pathogen, whether of bacterial origin or some unoxidized metabolic product or virus, had the means of integrating with the fibrogenic tissues of the joint and changing its properties so the specific pathology for each condition was carried out. All that was necessary to restore the normal was to burn away the integrated pathogen. The excessive fibrous tissue in the form of cartilage or bone or highly vascular fibrosis was then in

the way and obsolete and subject to digestive autolysis, and removal, and the deficient tissues reconstructed to normal. Nothing was left to hinder such correction for the pathogen was removed. It is the same story as with neoplasia. The results are the same when the pathogen is oxidatively removed.

In the acute toxic stage of rheumatic fever, the arthritis is entirely inflammatory, but can lead to structural changes. Quick restoration of the normal follows the oxidative removal of the pathogen whatever it is. The following case from the court records illustrates:

ACUTE RHEUMATIC FEVER

CASE No. 68 Dr. Wendell Hendricks

E. N., female, age 11 years, showed a pretreatment observation period of five days. During this period her knees became fixed, flexed and contracted so she could not move them; greatly swollen and acutely painful. Other joints were hot, swollen, painful and flexed. Her finger joints, elbows and hip joints could be straightened out, but would "pop" right back to the flexed position. This was painful as was her effort to move them. The heart showed a murmur. The pulse rate was 120, temperature 102°F. There was a severely inflamed throat with swollen tonsils and adenoiditis.

Recovery Course

On July 3, 1942, she was given two micrograms of parabenzoquinone dissolved in water by injection. On July 4th the throat and knees were better, the temperature 100°, and the pulse 108. On July 6th all joints were better, temperature 99.2°, pulse 100, and the throat clear. On July 9th all joints were normal with normal function, no pain or swelling, temperature 98.6°, pulse 78. The throat was normal and the adenoiditis and the heart murmur had disappeared. On August 20, there had been no recurrence of any of the symptoms. Here again the FCGs had to be rescued by using a superior dehydrogenator carbonyl group with correct steric advantage. Two micrograms of benzoquinone were sufficient. The serial systems of carbonyl groups have shown better action, however.

CHAPTER XXVII

CASE MANAGEMENT

Elimination

One of the greatest problems the invalid has to face is to get rid of the debris left over from the digestive procedure before germs are able to convert it into food material for their own prosperity while at the same time converting it into the very poisons that are making the person sick. The amount and condition of the debris will depend upon the thoroughness of the digestion process and this will depend upon how it was started in the first place. Therefore, thorough mastication of the food is required, and the time allowed for eating should be generous in proportion to the amount taken. This we have shown must be a minimum amount and not the maximum as is the usual habit. When the work of the stomach and small intestine have been the best, the amount of debris to support bacteria will not be greater than the bowel can get rid of in good order. If under these circumstances there is hindrance to the elimination, one must find out what it is and correct it.

Worries, nervous tension, and the habit of not heeding the call of the bowels to evacuate, may set up a stubborn constipation. The obvious cure anyone can figure out. But to make sure, let us say that attention to the call must be practiced liberally even if a cathartic is needed to get things going again. The enema may be used to the best advantage here as is required until the habit is re-established, and to do this the scheduled trips to the toilet are required as were taken before the neglect was permitted. The enema must not displace the effort to move the bowels naturally, but should supplement it.

Intestinal spasms, especially at the several sphincter muscles tend to block the onward progress of the bolus. At the same time a paralytic sort of relaxation of the musculature of the body of the viscus makes matters worse. Both features unite to constitute a reflex which is normally designed to keep the organ quiet to accommodate healing as when our cancer cases with bowel involvement are undergoing healing. The situation is troublesome until the healing is finished, and the enema at this time may be a daily necessity for a while at least.

So when the patient blows up with gas after a meal, there is some reason for the bowel to relax. It may be the need for a rest to survive from too hard a task, that is, too much has been eaten day after day. It may be that an obstruction has made the bowel tired in trying to force the material through.

It may be that the undigested food material is offering the germs the chance to produce so much toxic amines that these act on the muscle cells to paralyze them by blocking their oxidations as we described above. The sensible thing to do is give the bowel a rest, by a fast or by eating very lightly or just going on fresh vegetable or fruit juices for a few days and washing the bowel out with a mild soda solution. One must also look for an obstruction if the condition persists. It may be due to adhesions causing a kink, or due to a tumor. Sometimes an ulcer will cause the reflex to give the part a rest to accommodate healing. But when the bowels do not empty completely and the retained material accumulates until a cathartic must be taken, gluttony is the general cause for the constipation. A radiograph may show what is wrong and if the poisoning comes from material retained in a diverticulosis or a kinked appendix.

The choice of a cathartic is a serious matter as is its use. When necessity demands it there is no sense in putting it off. But after the evacuation is obtained, the cause must be corrected. Milk of magnesia is an easily available and good remedy, as is sodium citrate or sodium sulphate. In cases of heart disease the magnesium ion may be a disadvantage and sodium citrate or the sulphate should be used. The amount taken should be large enough to do a thorough job of clearing out the debris and the cathartic as well. Castor oil of the olden days was as injurious as the taste threatened it must be. It blistered the bowel inside after it reached an alkaline intestinal medium where it was split into its blistering components. All the other irritant affairs like cascara, aloes, and senna act the same way. They ruin the bowel.

The enema taken with patience is the best procedure. The water should be warm as one has during a high fever, about 42° Centigrade or 108° Fahrenheit. This temperature relaxes the spasms that may otherwise make the entrance of the fluid difficult. The pressure must not be too high either as it can excite a reflex to expell it. Therefore the position of the body, the height of the water bag, and the most favorable temperature need to be ascertained and adapted to each patient for the most comfortable and easy passage of the water into the colon and through it over into the caecum, where the worst putrifaction generally is found. To aid this process and provide more comfort and less spasm one may use a tampon about the tube that is inserted into the anus. This tampon should surround the tube about four centimeters below its tip so it can be pressed against the external sphincter in a way that prevents the loss of fluid. Thus, the muscle does not have to be contracted to retain the water, and the effect of this relaxation is felt throughout the whole bowel, and lessens the tendency to other spasms throughout its length. The tampon can be made from a cork. A hole is bored through it to accommodate the tube, and it can be rounded off so as to not prove painful. A solid small rubber ball could be used if one can secure a cork bore to cut the passage through it. Before inserting the tube into the anus, it should be lubricated with some oil as olive oil, or one of the vegetable fats used for cooking. Vaseline should not be used as mineral oil products are

sometimes carcinogenic. This has even been demonstrated for mineral oil sold as a laxative.

Repetition of the Dose

In chronic disease where exhaustion or cachexia stand in the way of the work that must be done to absorb a malignant growth and restore the broken down tissues in general, one does not always have the data to know how big a load a patient can carry in his fight for recovery. In early cases this difficulty is not encountered, and one runs no chance of overloading the patient — that is within reason. But in patients for example who can not even handle a few blood transfusions, and are not able to destroy and digest the blood received and turn it into living blood, the exhaustion must be handled with expertness. One cannot give a bigger dose or repeat it beyond the *ability of the patient to use it.*

In most advanced cases of cancer, the reticulo-endothelial system is exhausted. It was so before the cancer took hold, in fact, when fibrogenesis started to fail. And it is more so as the disease advances. Likewise the oxygen carrying power of the blood may be very poor. The hemoglobin may have been changed to a methemoglobin that is worthless and gives a false color index, that is, an index that does not tell the amount of oxygen carrying hemoglobin. The recovery depends on the use of oxygen, so a limitation is imposed by blood injury of this type. Aspirin and other coal-tar drugs, and any situation that causes cyanosis will hinder. Thus with a poor or deceptive blood count, there may be the greatest need for speed in changing the situation that exists, yet the supply of oxygen to the tissues may be restricted so one must go slowly. It takes material to build up the blood capillaries that absorb the growth. That means the metabolism of the food by all the important organs must be sufficient to meet the needs of the case. So many things must be considered before one can decide on how often a patient must be treated, and how big a dose to give. The dose is never repeated so long as improvement is observed.

Endocrine deficiency adds other difficulties. And in spite of medicinal support, a lowered metabolism rate, in more respects than the conventional meaning of the word, may hinder progress greatly in response to a boost. One must not overload. The homely analogy to the mule, that was overloaded and sunk to the ground and was never able to get up, may give the picture accurately. Hence, the doctor must never overdo his job. Since endocrine and general tissue cell deficiency results from radioactive materials, treatments and environment, food and water, these interferences must be removed. It is even necessary at times to take the patient to a different district. At any rate, all interferences must be gotten out of the way, and then all the aids given that are required. After that an adequate dose is given, and the situation held favorable to its action. If this is done ideally, no repetition of the dose will be necessary. It often does happen so. But more often interferences within the patient,

or coming from without, block the progress of recovery. We mention them under diet and management.

The correct dose of benzoquinone is the one to a million solution, given in two cubic centimeters under the skin or in the muscle. The correct dose of the serial system of carbonyl groups, cyclic or linear, is either, as for benzoquinone, two micrograms in two cc. of water, or a dose of two millimicrograms in two cc. of water which, by the way, is the active dose for vitamin B_{12}, or it should be just one thousandth as much, one part to a trillion of water. Higher dilutions are active but are as a rule not required to meet the state of depletion of any patient, though at times they may be needed. Repeat-doses are of higher dilution. It is our good fortune to have a blood test that tells much about the state of depletion. It is the crenation test, and is very simple, but must be done expertly and accurately.

The Crenation Test

If one makes up an accurate one per cent solution of pure sodium chloride and keeps it well stoppered in stock and then draws off a small bottle for daily use, thus protecting the stock solution from being opened too often, he has all that is needed, plus a hemocytometer pipette, and glass slide, cover slip and microscope. One draws the measuring tube one-half full or less than half full, of blood from a fresh cut on CLEAN, DRY skin, quickly draws up the salt solution to fill the chamber, mix, and puts a drop of the mixture on the slide and makes an estimate of the cells that crenate (shrivel up) within the first minute, and the number that do not, and those that not only stay round but swell up instead. Speed is essential, as in time they may all crenate, and by being slow one gets a false count. The theory is this: the red cell offers a semipermeable membrane. The salt solution is correctly hypertonic for the test. When water is drawn into the cell it swells up. When it is drawn out by the 1% salt solution, it shrivels. Normally it should shrivel as the 1% salt solution is hypertonic to the 0.85% normal osmotic pressure of the blood and inside of the red cell. Therefore, when the cell refuses to shrivel, and more so when it swells up, the osmotic pressure inside is too high. This means that the large protein molecules are split to many smaller molecules. It also means that food molecules are present that are not built up into the normal structures. *The picture of the gross error is presented.*

Lytic changes manifested by failure to crenate and their counterpart, the failure to build up the correct cell structure, point to a lack of energy production of the high efficiency type by the FCG. These changes mean that the FCG is blocked, and hence the Super FCG (the SSR) is needed to release it. This is a straight indication that either the Survival Super FCG (the SSR) administered has not finished its work to a safe point or that there is none working. One must decide between the two, however. If there is reason to believe the Synthetic Survival Reagent was blocked right at the start the treatment can be repeated right away three days to two weeks after the first dose. Also, after the third week reaction is passed failure of an improvement in

the crenation test and in other changes in the patient indicate the need to repeat the dose. If the curative chemistry is started it must be allowed to go as far as it can before the dose is repeated. It may even be good policy to give a second dose at the end of the second week as a routine if no heavy febrile reaction was had 24 hours, 36, 72 or 84 hours after the treatment. Repeating on the 14th or 15th day is good policy even if the crenation test shows only slight change. In that length of time it will not be much and there is much room for more. This is a different matter from repeating the dose at the sixth week or later since the improvement in the crenation test may be definite, but not 100%. *But it may be the maximum for the patient at the time.* So repeating the dose would then overload him. Never repeat if he is improving, therefore.

Repeating the dose in the first few days or at the end of the second week is more like giving a double dose at the start. It is better than that, as in these two weeks interferences may have neutralized or destroyed a big part of the remedy, and repeating at the end of the second week would restore the lost part so the second dose would have cleaner ground to work on. Nevertheless, one must balance up all the factors, the crenation test, the reduction in the size of the growth, the color change in the patient, that is, the loss of the hemolytic color, the gain in red cells and hemoglobin, improvement in the sedimentation test, etc. The appetite and gain in strength may be the best indicators as they speak for many specific changes such as those just mentioned. The quality of the pulse and respirations are also most valuable as is the ability to have a refreshing sleep. The improvement in taste and smell and even of hearing have the greatest significance. The old-fashioned doctor who knew how to observe his patient as a physiological complex knows what to look for. When such data is balanced with the crenation of the red cells, a correct decision on the need for dose-repetition can be reached. It is better to take one's time and think.

Diet, Medication, Hygienic Aids

Diet, medication and environment are factors that influence the success or failure of the therapy, for any influence that will hinder the recovery reaction cycles, or block them, can bring defeat. The following agencies must be considered. They have to do with the soil and the food that comes from it.

Radiological Hindrance

There are radiological influences of the soil in certain districts that are revealed by the Geiger Counter that should be watched. Some areas do not have enough irradiation to interfere seriously with the recovery program, or with the survival chemistry. In such regions the death rate from cancer is less and here the recoveries from cancer are much more satisfactory in their course and percentages. Detroit and the shores of the Great Lakes have proved gratifying, while farther into Michigan as west of Ypsilanti, patients do not do so well and the Geiger Counter registers higher rates. The difference is perceptible to patients travelling from western Michigan to Detroit. So many

have reported feeling better after they came east of Ypsilanti, that the report had to be recorded. Those who leave the high irradiation districts to come to Detroit for care soon became homesick and had to return. At home they were retarded in their recoveries and even when food and water was shipped to them from Detroit, they still lost out. It seems that they are habituated to the irradiations much like a drug, alcohol and tobacco addiction. The evil effects of the irradiations are quite understandable when one considers the harm done by even slight exposures to the X-ray or radium. It is now the concensus of the experts that no amount of irradiation is too small to have a measurable bad effect on the individual's longevity, health, and well being, and the development of his offspring. For this reason, frequent unnecessary radiographs are to be avoided, and irradiation by radium, X-rays, and Cobalt are scorned as overdone and extremely dangerous experiments, that have never shown a favorable record in true cancer, but on the contrary, they stimulate the neoplastic state, and even give rise to new cancers deep below the lesion that is being treated. That the survival chemistry is destroyed by irradiation is seen in the hereditary defects in the offspring of radiologists. This shows in 10,000 children of radiologists, twice the incidence of cancer, and more defects in eyes, heart and blood, than in children of physicians not exposed to irradiation. Eight to ten times more radiologists die of leukemia than general practitioners. (Am. Roentgen Ray Soc. Trans. 1954). Certainly this sacrifice of the radiologist in the conquest of cancer has never been appreciated in full.

The areas of high radiations are not always large but occur in patches and 40 miles away from the worst of districts, the very best can be found, at times. This is especially true about Columbus, Ohio. Those who were exposed to therapeutic irradiation do not recover as do those who were not exposed. All depends on how much was received. For some years now it is observed that the serious radiologist gives very little exposure, and often, since he considers the therapy a dangerous placebo, he gives none at all, but rolls the patient under the machine for a while and then away again without even turning it on. This certainly is an act of mercy. It can be appreciated in patients who were said to have been irradiated but were not, by the course of their recovery after this Survival chemistry treatment. In such cases the reactions follow the same course as in non-irradiated persons, and the healing powers are not reduced. Dead bone is not encountered with its secondary irradiations and poisonous products sent into the blood stream. Further, the neoplasms are convertible into a normal type of chemistry for digestion and absorption, instead of being stimulated to grow by an uncontrollable agency. It must be recalled that the Survival chemistry operates in the outer electron shells of the atom, the field of physiological reactions and their pathological variations as provided by nature. The irradiations that operate on the nucleus of the atom produce a type of change the Survival Reagent is not built to combat. For the same reason X-rays and radium and the isotopes have no chance to ever cure cancer.

The destructive action of irradiation can be concentrated to kill the superficial layers of a tissue, and for that reason the basal cell cancers of the

skin that do not penetrate deeper than a few millimeters can be destroyed by irradiation in a way that is comparable to the action of the Percy cautery with the hot iron or the escoratic action of Zinc Chloride and other destructive chemicals. The destructive action is not limited to the superficial layer only but extends deep enough to only partially kill the tissue underneath and thus conforms to Warburg's postulate on carcinogenesis. The fibrosis set up is also a cause of anoxia that falls in line with the Warburg conclusions on the cause of cancer. The highly malignant cancers that spring up under the cancers being irradiated therapeutically are an example of this destructive effect. In like manner strands of cancer cells that have penetrated deeply below the surface are stimulated to reproduce more vigorously and cause death earlier in line with the statistics that are now being carefully assembled.

Highly Polar Double Bonds

Highly polar double bonds can add free radicals of the high efficiency oxidation process, especially those developed during the Survival oxidations, and block the recovery process before it even gets a good start. Illuminating gas, exhaust gases from combustion engines, chimney smoke from oil burners, paint solvents, and floor wax solvents, many terpenoid substances, as those in the skins of citrus fruits, and even too much carotene in the food are to be avoided. The cancer patient, for example, is somewhat night-blind, simply because he cannot oxidize carotene to vitamin A, as efficiently as he should. He should receive his vitamin A, in the form of fish liver oil, and not as the previtamin sold as vitamin A. Perfumes belong to the excluded class, natural or synthetic.

Free Radicals

Chloroform, carbon tetrachloride, and the oxides of nitrogen, offer free radicals that can add to and inactivate the free radicals of the Survival oxidations. The most dangerous are those from nitrous oxide used as an anaesthetic. If any surgery is to be done to a patient contemplating treatment, it should be done first. All tooth extractions or repairs or other attentions should be anticipated, searched for and taken care of before the treatment is given. If a heavy general anaesthetic has been used, time should be given for its waste products to get out of the system before the treatment is given. If, during the course of treatment an emergency operation must be done, preferably cocaine or novocaine as a local anaesthetic should be used if at all possible. Years of experience has proven this the best course. Arsenic is an antagonist under this heading also.

Steric Hindrance

The steric setup of the normal cell is one thing. Addition of each different pathogen to the FCG system changes this set-up differently in accord with the structure of the pathogen. Some pathogens cannot add to the cell because

of steric hindrance, the cell is immune for this reason. The addition of an antibiotic and even of the toxic amines developed in the colon can so change the steric setup of a host cell pathogen integrate that the Survival remedy is hindered in its attack. It will be recalled that the Survival Reagent must attack perpendicularly to the plane of conjugation of the double bond and the carbon atom that carries the hydrogen atom to be removed. The additions of altering reagents are to be avoided. Hence, we give no antibiotics, we maintain a clean colon, and follow a careful diet with careful selection of medications during this treatment.

Acrolein, and the polymerized acrylic aldehydes produced in the frying pan or roasting ovens interfere in every possible way. They offer highly polar double bonds, and free radicals during their polymerizations. These not only inactivate the free radicals of the oxidation service, but can add to the double bonds that activate the FCG. Fried or roasted foods where fats are used, must be avoided.

Interferences With Oxygen Transport and Use

Amines gel the tissue colloids and prevent their flow, and reduce their surface carrying power of oxygen and other materials. Salicylates and aspirin and their analogues change the hemoglobin so it does not carry oxygen. They produce cyanosis and asphyxia, and in a fight where molecular oxygen is the first consideration, one sees that all coal tar products are to be avoided. This includes the creosols and other bug-killers. The subject of insecticides is most important; and the selection of foods to avoid contamination by them is a critical problem. For example, the corn is sprayed to kill the bore. The corn is pressed to extract the oil. The cake is fed to cattle. The cattle are killed and the people who eat the meat get sick and sometimes fatally. Spraying airplanes with the passengers aboard accomplishes nothing except to poison them uselessly. This custom must be stopped, for the insect can stand a far bigger dose than the passenger. Much of the lung cancer gets its boost that way. Aramite, a recently used insecticide was found to be strongly carcinogenic even in the most minute traces. It was used in spraying fruits and vegetables, but is now stopped by recommendation of its manufacturers, the U.S. Rubber Co. Colorings and other agents used to beautify preserved foods have been proven carcinogenic by direct action on the cell. Nevertheless, they all act adversely in other ways, as in their effects on the blood forming organs, and in blocking the use of iron by the reticulo-endothelial system. Therefore, the protection of foods which should be taken care of by the Food and Drug Administration is not done efficiently, instead, that outfit is trying to tell physicians how to practice medicine to twiddle their time away.

There are some special poisons against which the public has no protection but are not added artificially, but are contributed by the soil itself. One of these is SELENIUM. It is picked out of the soil by corn, peas and lentils, and even by wheat. Corn is grown in the middle west where selenium carrying soil abounds, likewise peas are grown there and canned and shipped nation wide.

More than one of our patients was made fatally sick by can-peas without any infection being present. Analysis showed it was the high content of selenium.

SELENIUM poisoning of cattle was first encountered in the western plains and was thought to be due to the high alkalinity of the water encountered in these regions. Of course, the surface water was poisoned by it. But later it was found that the vegetation carried toxic quantities — over 4 parts to a million. Some plants, notably the Astragalus thrive on it while others are killed by it. The astragalus plant is taken as a signal of danger therefore, and animals that eat its leaves die, as it sometimes carries several thousand parts per million. Farmers find that wheat grown on selenium-rich soils is toxic to their cattle and chickens, so they sell this wheat and buy grain grown on a healthy soil. Toxic effects are shown when the food contains four parts per million. There are two types, the chronic and the acute. The latter is quickly fatal and comes when the food carries 20 parts per million. The chronic effects are loss of hair, and hoofs or finger nails, a general malnutrition, impotency and infertility. Eggs do not hatch, or the chickens may not even lay eggs.

Tissue studies in the Warburg chamber show that after being exposed to toxic amounts, the oxidations of glucose, of succinate, and of lactate and citrate are blocked, but the oxidation of para-phenylene diamine is not hindered. This holds for all tissue examined as muscle, brain, kidney, liver, and tumor slices. Thus, from our standpoint, it is a serious inhibitant to recovery and it supports the pathogenesis of cancer and all other diseases for that matter. At times our patients would eat canned peas and become fatally ill. We therefore forbid canned peas in the diet, or even the dried peas unless one knows the soil on which they were raised is pure.

The selenium exists in two forms, inorganic and in organic combination. The former is more easily eliminated from the system, but it is the amount that kills, not the particular form in which it is taken. Fortunately it is known that selenium exists in the heavy clay-like soil, while sandy soil and coral reef soils do not contain it. Therefore, foods like rye, that are grown on sand, are free from this poison. This is one reason rye should be eaten instead of wheat, which is grown mostly on the heavy soils where selenium abounds.

Sulphides in the drinking water, or produced in the intestine by putrifying bacteria, are also highly toxic. This is another reason for avoiding animal foods, for as we explained, sulphides and sulphydryl in many forms can add to the double bonds that activate the FCG, and thus paralyze the tissue oxidations. Their action on the bowel wall is like that of the toxic amines. They paralyze the musculature and the secretion of ferments and mucous and cause diverticulosis and ultimately a gangrenous degeneration with prolonged exposure.

The parathyroid experiments showed that the guanidin bases caused a gellation of the tissue colloids that hindered oxygen transport even in the large

veins. The other amines produced in the intestine by the bacteria that decarboxylate amino acids, have the same action. For this action which is the vanguard of disease of all kinds, three factors are needed. The bacteria must be there, the amino acids must be there in excess, and the reaction of the medium must be acid. The streptococcus fecalis, for example, decarboxylates arginin to become agmatin, an amine that is oxidized by diamine oxidase only in dilute solution, but it inactivates diamine oxydase in more concentrated solution. The reaction of the medium where it is most active is from pH 3.5 to pH 5. In the same way lysine is changed to cadaverine, histidine to histamine, ornithin to putrescine, tryosine to tyramine, by such bacteria as B cadaveris, E. Coli, Cl Welchi, S. fecalis, etc. Some bacteria like S. fecalis require an exogenous source of vitamin B_6 in order to form their decarboxylase. Lactic acid bacilli consume pyridoxal phosphate greedily and thus may prevent the S. fecalis and the others, that require this vitamin, from producing their enzymes. Only histidine decarboxylase appears to not require B_6 as a coenzyme. Thus, there are four ways of trying to control the production of the toxic amines in the colon. One is to keep the bowels moving, and not eat an excess of protein food, eat no animal food to supply the B_6 for too much bacterial action, keep the colon alkaline in reaction, as it should be NORMALLY. Lactic acid bacilli, however, create a favorable medium for amine production and this more than counterbalances their action in using up pyridoxal phosphate. The S. fecalis even produces its own lactic acid to activate its decarboxylases, and make a sure job of it. The food must be well chewed, not too much liquid taken at the meal, and as small a volume of food as is serviceable for nutrition. Thus the natural secretion of the intestinal wall which is pH 8 or so will have a chance to dominate the field. When the bolus is solid and supports germ action, the bolus is found to be alkaline on the outside only, and acid inside, where the toxins are being brewed. The vegetarian diet tends to avoid this distribution, and to hold the whole bolus alkaline as it should be.

The other dangerous source of toxic amines is the antibiotic therapy which, of course, must be controlled by the physician to be safe.

Food Preparation

The food should be eaten raw as far as possible. The cooking should be gentle, and not overdone, and steaming in a well covered kettle is best. The utensils should be stainless steel, or Pyrex glass so far as possible, and a good, granite or porcelain is maybe best of all. Copper kettles are good for some uses, especially for preparing fruits for preservation. It has some antiseptic action. Aluminum has its advantages and disadvantages. Among the latter are the easy solubility in acids and alkalies and even in distilled water.

Some observers have classified Aluminium as a trace element essential to the body chemistry, but in such minute doses that no worry need be had that the amount taken in needs help from such sources as the cooking utensils. The Government issued a comprehensive report on aluminium poisoning. This

book gave a splendid review of laboratory and clinical fatality records caused by this metal when improperly used in the kitchen.

The fruits and vegetables must be cleaned before using. This is evident from what was said before. Soap and water and a brush to really clean what is to be eaten need little elaboration. The pits where the stems are placed and at the reverse end of the fruit should also be cut out to remove the insecticide that accumulates there. It may be healthier to buy wormy fruit than that which is so perfect, as the soil of orchards that have been long in use is saturated with arsenic and the fruit, therefore, carries this poison, too. It is one of the very worst. If copper would be used as an insecticide we would all be better off. Arsenic, of course, is an uncoupler. It prevents the energy of oxidation being stored in high phosphate bonds from being usable. It is carcinogenic, too.

There should be no foods fried or baked with fat, because of the acrolein produced by dehydration of glycerine of the fat. This aids carcinogenesis and other diseases. The polymerizing acrylic aldehydes so produced boost carcinogenic action a million fold. I have seen the results in practice many times.

Spoiled foods should not be eaten. They should be ripe, however.

Food Quantity and Quality

Common sense here is all that is needed. Not too much bulk so as to not dilute the digestive juices too much, not too much weight to tire the intestinal muscles and permit easy passage of the refuse away from the body without any accumulations to form diverticulous sacs along the wall of the colon.

Dietary Recommendations

Tea and coffee are drugs and have no place on our diet. The Brazilian Mate is also excluded, because of its caffein or theobromine content. Warm water and honey make a good healthy drink, and the readily available fruit and vegetable juices offer all one can desire. If orange or lemon juices are to be allowed, the skins should be trimmed off first and they should be washed before being crushed to extract the juices. This is to get rid of the terpenoid oils of the skin.

The foods—fruits and vegetables—nature offers have enough sodium and potassium without additions, for the best service, so salting need not be done for nutritional needs. Sometimes high potassium diets are helpful, and lists are available. Beans and peas are rich in potassium as well as all fruits and vegetables. They are superior to meats, therefore. Attention should be given to the special concoctions, the contents of which are not known, as the artificial ice creams and soft drinks. These should be avoided, as well as the poorly brewed beers and caffeinated soft drinks on sale. For our patients, however, no artificial drinks are to be used. Arsenic poisoning has been produced en mass by bad brews more than once. Since vinegar is a completed fermentation product, if it is made from clean apples, it is a valuable article in the diet. A

little on the salad helps digestion, and offers ferments that are also very useful. On the other hand, lactic acid products are to be avoided as they give support to the decarboxylating germs in the colon. In this connection the food could also be examined with reference to the amino acids they offer that might be good only in small quantity. Peanuts are such a food. They should be eaten raw only, as roasting produces acrolein. The rich supply of arginine they carry is easily changed in agmatine, the very toxic guanidin derivative, through decarboxylation by bacteria in the colon.

Iron in organic form as contained in rye, the whole grain ground fine, or in wheat germ, and not as a salt, should be used. Iodine is generally deficient and some potassium iodide or better still some kelp every day is a great help, in such communities where the soil is iodine deficient. The unsaturated fats and oils promote oxidation of themselves and the more saturated fats, and should be selected. Olive oil is a healthy fat, lard is carcinogenic. Carefully kept peanut oil and other unsaturated oils require exclusion of air to be serviceable. Oxidation destroys their unsaturation and makes them rancid, unfit for use. But taken unoxidized into the body, they promote oxidation within.

Cholesterol is produced from short chain unoxidized fatty residues. These come from poorly combusted saturated fatty acids. To prevent their presence one must not eat too much fat, and select the fats that are unsaturated, since the double bond activates the hydrogen of the carbon atom alpha thereto, and aids its removal so oxygen can make its addition. This probably is a free radical affair as we have described before and results in cleavage into short chains of two carbon atoms each. Peroxide free radical formation by molecular oxygen addition to the free radical would result in cleavage with carbonyl group terminals to the chains produced, and these would activate further dehydrogenations, and tend to continue the process. Thus, the unsaturated fatty acids promote their own oxidations and tend to induce the oxidation of the saturated fatty acids. This explanation is a chip off of our postulate, and is beginning to be adopted now.

The snow white fats bought in cans that never develop a bad taste by going rancid have been reduced by nickel catalysts to become fully saturated and hence they are the very fats that are difficult to burn and would tend to support the production of cholesterol. The conclusions are obvious. *Fresh olive oil is best* for all purposes, and can be used instead of butter on one's bread.

Natural foods, not prettified or poisoned by additives, should be chosen. Rye is the best grain. It can be bought whole and washed well and dried, and then ground at home by a small mill, and cooked into a porridge or baked muffins or bread. The finer it is ground the better. One should know the soil on which his food is grown, but that is not practical, certainly not for most people. But if one knows the soil is selenium free and without radioactivity, anything that grows on it should be good to eat in line with what was said previously.

The question then arises as to how to eat it. This is determined by the nature of the digestion process. The stomach does not churn the food like a

cement mixer, as was taught for so long. The food arrives and forms in layers and as the pepsin and HCl are formed in the glands of the fundus and prepyloris proteins should be eaten first, carbohydrates last, but better still the meals should be either carbohydrate or protein, so the digestion will be done by one enzyme system at a time, though provision is made for a mixed digestion. These simple statements cover the choice of foods, the best fat, the best cereal, and the fruits and vegetables that supply the rest. How to eat it, — the mono-diet being best, and to avoid overloading the digestive tract, the best way to eat is as nature provides. The baby, for example, does not want its three square meals a day for the convenience of the kitchen. Children fall into the habit of snacking. If one adds up the quantity taken in snacks, and compares what would be eaten on the three square meal system, the economy on the food and metablolism will readily become apparent, and the better digestion and sleep will soon be realized, too. It is far better to abolish the dining room table, and go on the "pick-a-bit" system to avoid heart lesions, big bellies, and big doctor bills. This is the solution to the cholesterol danger. One should discuss the metabolism of fats to show how carefully these substances are broken down and then built up to suit the species architecture, and how cholesterol is really a by-product of value in certain directions and amounts. It, however, is no problem when the oxidations are efficient and the diet sensible. In all our observations high values drop soon after the FCG gets back to work in normal fashion, the disease in question makes no difference, and the erratic character of the curve plotted out from its estimates may be the worst, as is seen in cancer. Yet, it steadies to a good normal when the oxidations are re-established to normal — and one of the easiest ways to avoid over-eating is to develop the snack habit and stay away from the table. This paragraph covers the problem in a "nut-shell."

Food need not be hot to be eaten. Better cool, in fact, though not cold so as to insult the stomach, and the snack conforms here, too. One thinks vegetables are supposed to be eaten hot. But this is not always true. A whole rye bread-lettuce-tomato, or bean sandwich, or asparagus or what-not sandwich can be made and be found delicious even cold, and be very satisfying. A slice of melon, an apple, peach, or other fruit, and especially the banana, a raw vegetable, carrot, onion, or chunk of cabbage, makes a good snack, and so does a raw vegetable salad. There is no need to worry that there is nothing to eat on this system. Its value lies in the opportunity it offers to not eat too much. At the same time the tyranny of the kitchen is abolished, and there are very few dishes to wash.

Sufficiency in the meatless diet is readily understood when one observes what the cow can get out of grass. Most people would let themselves starve to death if marooned in a rich pasture, but not the cow. She would manufacture enough milk to supply herself, her calf and the neighborhood children with protein, fat and carbohydrates and all the tissue salts, and have enough left over to make some cheese besides. The grass, like every other vegetable and fruit, has a perfect proportion of protein. Some as peas, lentils and beans, have too much. In the bean-eating countries like Brazil, the beans are mixed with rice

and mandioca powder to raise the carbohydrate content. This is an adopted custom, not built on the chemical analysis of the bean, but on the survival analysis basis, and is found wise by trial in a natural instinctive way. A few analyses of foods will prove helpful.

Protein Food Selection

Let us compare the protein contents of meats and 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. 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 gms., protein Breads run about 2% protein, Brussels sprouts about 4%, broccoli 4%, potatoes 2.4%, peas about 23%, beans 21.4% and nuts 9 to 10%.

Thus, since the daily requirements of an average sized 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, a good bowl of pea or bean soup and a slice or two of bread and a few greens cooked or in salad, would supply all he needs. But one needs the salts, vitamins, the unsaturated fats, and carbohydrates. Bran or wheat offers 12.4% protein, 3.4% fat, 4.2% carbohydrates and 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.% and bananas 10 mgms.%, cabbage 50 mgms.%. Thus a mixed diet, according to taste without any animal product, will give all the nutrition one cares for or needs, and some articles as beans and peas and nuts should be eaten sparingly, especially peanuts with its 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 half a hundred blood transfusions could not keep the blood up to normal or even half normal, but without even one transfusion each of these cases gained a normal blood count only on the vegetables, fruits and cereals, without any medications whatever. There 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 the same diet monstrous women have reduced to most attractive proportions and curves after the treatment gave the oxidations their needed help. 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.

CHAPTER XXVIII

PREVENTION OF CANCER, ALLERGY AND INFECTION

This whole text has developed the thesis of prevention and cure of these conditions and some others not classified with exactness. One might give a brief summary as a reminder, however.

The role of suppressed or buried infection in causing chronic diseases has been exposed in detail. It is only logical that such foci should be removed if at all possible. Most of them can, if they are discovered in time. They are dead teeth, infected teeth roots, the scarred-in buried tonsil and associated hardened lymph glands, the infected appendix that gave an acute attack years earlier, the infected gall bladder. Most of these sources of suffering and death can be removed by expert surgery without real inconvenience to the patient, and a good surgeon can diagnose their presence without error.

The gastrointestinal tract as a source of the toxic amines and of viruses living in symbiosis with various germs, requires all the attention spoken of under diet, and intestinal hygiene. To this end the enema should be used freely and intelligently to strengthen the bowel wall by washing away the toxins that paralyze it and cause diverticulosis. Special lavage systems are available most everywhere, but when this is not convenient, one can learn to use the enema at home with good results as well. It is discussed in the chapter on the care of the patient. Correct eating, as to quality and quantity are discussed sufficiently in the text. But some factors need emphasis.

The choice of foods calls for the avoidance of selenium containing wheat, peas, corn, etc. and the avoidance of foods poisoned with insecticides. It is within the power of the Food and Drug Administration to correct the whole food poisoning situation, and just now public worry over the insecticide and carcinogenic contaminations of the food is calling for some protective action. But this action is even now as stingy as it can be, and no protection against the selenium contaminations are offered as yet. If such neglect continues, it will be necessary for those who are able to organize their own farming services and see to it that the ground is fertilized scientifically by the organic procedure so the plants are correctly nourished and healthy and resistant to insect attack. In fact, it is demonstrated that insects attack the sickly plants rather than good healthy stock. Selenium-free soil will be used, and crops rotated wisely.

Whole rye is the superior grain, and the whole grain should be bought, washed, dried and ground fine for baking the bread and muffins at home, and for making a porridge. Freshly ground grain is preferred.

Whenever possible, honey should be used instead of sugar, and olive oil in preference to other fats. Foods should not be fried or roasted with fat and, of course, no animal proteins should be taken at all.

Sufficient exercise to meet the personal needs must be taken and especially such exercises as massage the abdominal contents. Floor scrubbing and wash tub

exercises are the best and if there is no garden or house work to do, golf, tennis, swimming, or polo will serve with the added advantage of stimulating the spirits. Each day the muscles need to be fatigued a little.

Plenty of sleep, free from worries or fears, a clear conscience, forgetting oneself with a healthy interest in the welfare of others so that constructive attitudes dominate, have their good effects on the circulation, and prevent the harmful mental influences from dominating the body chemistry. This is particularly true with reference to the harmful secretion of adrenaline, adrenochrome, etc., that are thrown into the circulation by anguish and anxiety, fear and insecurity. Many amines possess high reduction potentials, and can inactivate the FCG of the tissue cells, laying them open to other pathogens, just as the toxic amines absorbed from the toxic intestine can do. In this respect toxic amines were found in recent psychiatric research to disturb brain function in line with our thesis. The stabilizing influence of religion is seen in our ancestors. They had courage, were not subject to jitters, and never took calming dopes and had much less cancer than our generation. Rudderless politics were unknown, and treason in high places was not tolerated. Men had a right to confidence in their government, and there was no income tax to rob them of their earnings. In fact, they kept the devil out of important affairs instead of letting him run things as is happening today world-wide. The bureaucracies tried to serve the national welfare, instead of betraying it, as is happening to the knowledge of every thinking citizen today. In those days the vote counted and there was "something one could do about it" to keep one calm. Today, it is just the opposite and people are worried over their own fates and the future of their dependents. Terror is actually gripping the world, and with it the two great killers, heart disease and cancer, take increasing toll.

The mental influence over the reflexes that govern digestion and intestinal movements is to be considered here. Spastic bowels with dry walls, the failure to empty the rapidly increasing bacterial poisons, add to the mental instability that brings these conditions about. Calm joy serves otherwise.

In the prevention of cancer it is important to consider the structure and behavior of the lower end of the large bowel, namely the descending colon, the sigmoid sphyncter and the sigmoid cavity that ends with the inner anal sphyncter, the short rectal canal and the external anal sphyncter muscle. Much cancer of the bowel is located in this region, and much intoxication is developed in the sigmoid cavity that poisons the body generally and gives rise to cancer in other parts that are disposed to anoxic effects. One must give attention then to the causes of anoxia in any particular tissue. Much information is given on this matter that is unreliable. It is stated that one blow on the breast will not cause cancer. Many times this is true, but anyone with long experience in medical practice knows better and can recall specific instances where the broom handle was to blame. It depends upon the deep injury to blood vessels and resultant block to the circulation. Then the anoxia plus the intestinal diamines, plus the virus or some other carcinogen from the food or from an old scar with a buried infection are the other elements that are needed. The blow may

not be avoided, the scar may be identified and removed, and the bowel can be trained to not produce the diamines that serve as vanguard to the neoplastic change.

Cancer of the bowel, the sigmoid flexure and cavity particularly, come in people who neglect the emptying of the bowel once or more times a day, and particularly the emptying of the sigmoid cavity with completeness at each movement. The victims are generally nervous people who suppress the act of defecation to meet some other activity. Generally, they are travelling, and must catch the plane or be on time to work, etc. When they are travelling in Europe on gluttony trips, the accumulation of material in the sigmoid area can prove most disastrous, as real cleanliness is often not encountered in the restaurants, and the heavy meat diet and too much alcohol and bad coffee or tea make the situation most difficult for the poor sigmoid. After such trips the sigmoid is paralyzed and will not even discharge the full load it is carrying. It has lost its parastaltic wave, that normally strips the contents out through the anus without leaving any residue. In many only the "overflow" gets out, and a big portion is retained. Here the bacteria have their chance to continually reinfect all material that is admitted and keep the intoxication going at its highest rate. The bowel wall gets poisoned so it cannot produce the energy necessary to make the muscle fibers contract. The pressure developed inside by accumulation and gas production causes the wall to dilate and form sacs or diverticulae that hold the feces indefinitely, poisoning the wall more and making it dilate still more, in a vicious circle.

Obviously, the obligation is to wash out the accumulated feces and keep on washing them out twice a day or oftener depending on the case, but at least to keep the cavity clean and free of the paralyzing poisons. Then the system that has been poisoned generally from the fecal toxins needs a boost in its oxidation advantage so as to defend any poisoned tissues and particularly the bowel wall itself. It should be understood, too, that not only the sigmoid is injured by the constipation, but the whole colon and particularly the ceacum at the beginning of the colon. A constitutional aid is required to give the wall enough freedom from FCG inactivation so it can start to have muscle contractions and abundant secretions again. However, the daily lavage as often as required will give the bowel a rest as well as remove the source of poisoning. The diet will also help in removing the substances that produce poisons at the hands of the bacteria.

When this procedure is carried on long enough the bowel will regain its normal parastaltic habits and the parastaltic wave will progress forward right through the two sphyncters of the anus and to the outside. It is an error to state that intestinal lavage carried out scientifically causes a habit that one must depend upon ever after. Indeed, the best possible aid to cure is to be had via the lavage system. This will result in the complete emptying of the bowel as one observes in healthy children. The heart, bowel, and all other tissues will then benefit and have the advantage of remaining healthy.

SUMMARY

This book has carried a few new things worth knowing to the profession:

(A) The oxidative separation of the host cell pathogen integration, when the pathogen is a virus, a carcinogen, a bacterial toxin, an incompletely burned tissue metabolite. The host cell is left reconstructed in good functional status *in the first instance by* the return of energy to the host cell as the virus is burned away stepwise as a reversal of its synthesis. In the other instances any reconstruction is compensatory and not hindered by the presence of scar tissue or toxins.

(B) The basic cyclic recovery process that reverses the whole evolution of the disease as a continuing progression, cleaning out the last manifestations first and the first manifestations last.

(C) The importance of suppressed infection buried in an old scar where hypoxia favors polymerization of germ toxins into toxins of increasing molecular weight with differing pathogenic properties, causing different disease states.

(D) The position of the activated amine group, the free radical, the double bond and the carbonyl group in pathogenesis and in its correction.

(E) The place of block in energy production and its acceptance by functional units in the cause and the cure of disease.

(F) Proofs that the Citric Acid Cycle is not the main process of sugar oxidation, but that a high efficiency dehydrogenator system exists.

(G) The explanation of the Pasteur Effect, antimitotic action, and some other Biochemical Puzzles as functions of the activated carbonyl group.

(H) The reversability of carcinogenesis.

(I) The conversion of pathogenic germs into harmless and possibly useful members of the Great Biological Economy, and the cure of antibiotic resistance germ diseases.

(J) Identification and synthetic reproduction of the Survival Factor with potency boosted at will in accord with the known laws of chemistry in the cure of viral and neoplastic diseases.

(K) The use of the foregoing principles in case management for best success in difficult clinical problems, such as far advanced widely metastasized cancer of the vital organs resulting in complete lasting cures on but one or two doses of the reagent.

APPENDIX I

SUGAR OXIDATION

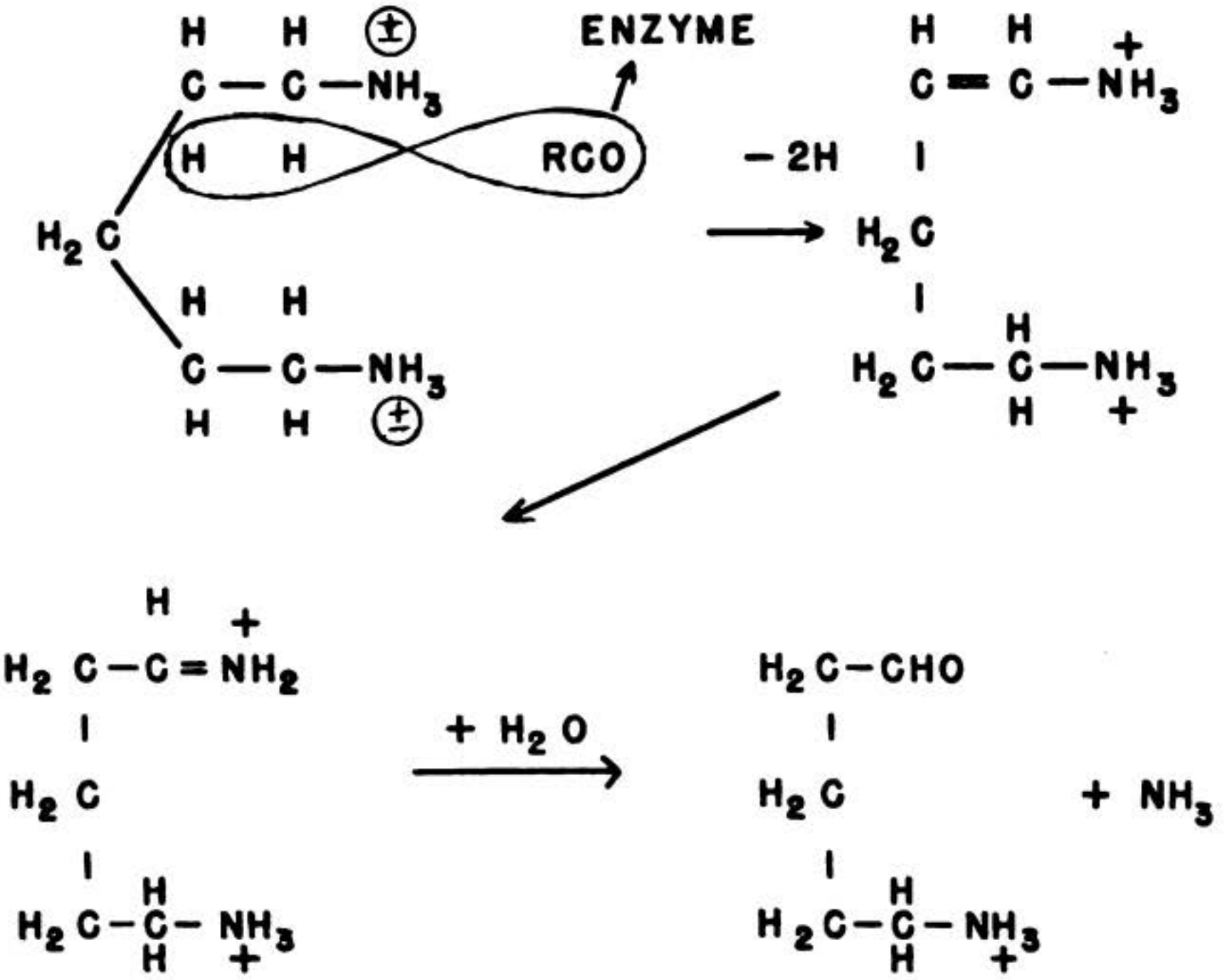
Although specific enzymes play a part in all metabolic reactions, it is also possible that non-enzymatic reactions play an equally important role in certain positions. There is no harm in visualizing a non-enzymatic factor in sugar oxidation. Fructose is much more easily oxidized than glucose so the enzymatic conversion of glucose to fructose is assumed. The non-enzymatic condensation of the carbonyl group of fructose with the amine group of the cell's oxidation mechanism to form an azomethine double bond offers the advantage of holding the fuel and receiving the energy produced by the first oxidative steps. The diagrams picture the process. When cleavage takes place and the Amadori rearrangement is reversed, an acetyl free radical is formed which adds to the phosphoric acid residue of the cell and further oxidation steps pass their energy on to the cell via this union. This happens so long as molecular oxygen is at hand. When it is not, the end products are lactic acid under the reduction flux of the medium, and the fermentation system takes over. The free radical and peroxide free radical intermediaries in the presence of adequate oxygen lead to such unstable, oxidation-inviting fragments, that they can never be trapped.

It must also be recalled that though the carbon hydrogen bond strength equals 58.6 Kg-calories per molecule and the O-H bond 110.2 Kg-calories, the set-up in sugar gives the hydroxyl hydrogen great freedom, so that when sugar is placed in heavy water the deuterium replaces the hydrogen atoms at random and here the bond strength is so greatly reduced that dehydrogenation of the hydroxyl group virtually leaves an oxygen free radical. The very structure of fructose invites dehydrogenations of the carbon atoms and the hydroxyl groups, that gives specific and non-specific dehydrogenators the preference over any agencies envolved in the Krebs's cycle. If the progress would follow the scheme outlined above no free degradation products would be at hand to be isolated. However, if the above process is crippled the last steps would require known dehydrases, and the latter products would be identifiable. The removal of hydroxyl hydrogen would take place very early however, even before the carbon chain is broken and thus the process requires more facts for clarification. One therefore sees how futile it is to outline such mechanism as the Citric Acid Cycle or any other mechanism, in view of the facts stated above, and in view of the lack of further directive data.

APPENDIX II

DIAMINE OXIDASE ACTION

Zeller's idea of the action of diamine Oxidase, as published in "The Enzymes" edited by Sumner and Myrbaeck, 1951, p 554, may be condensed as follows, using putrescine as substrate.



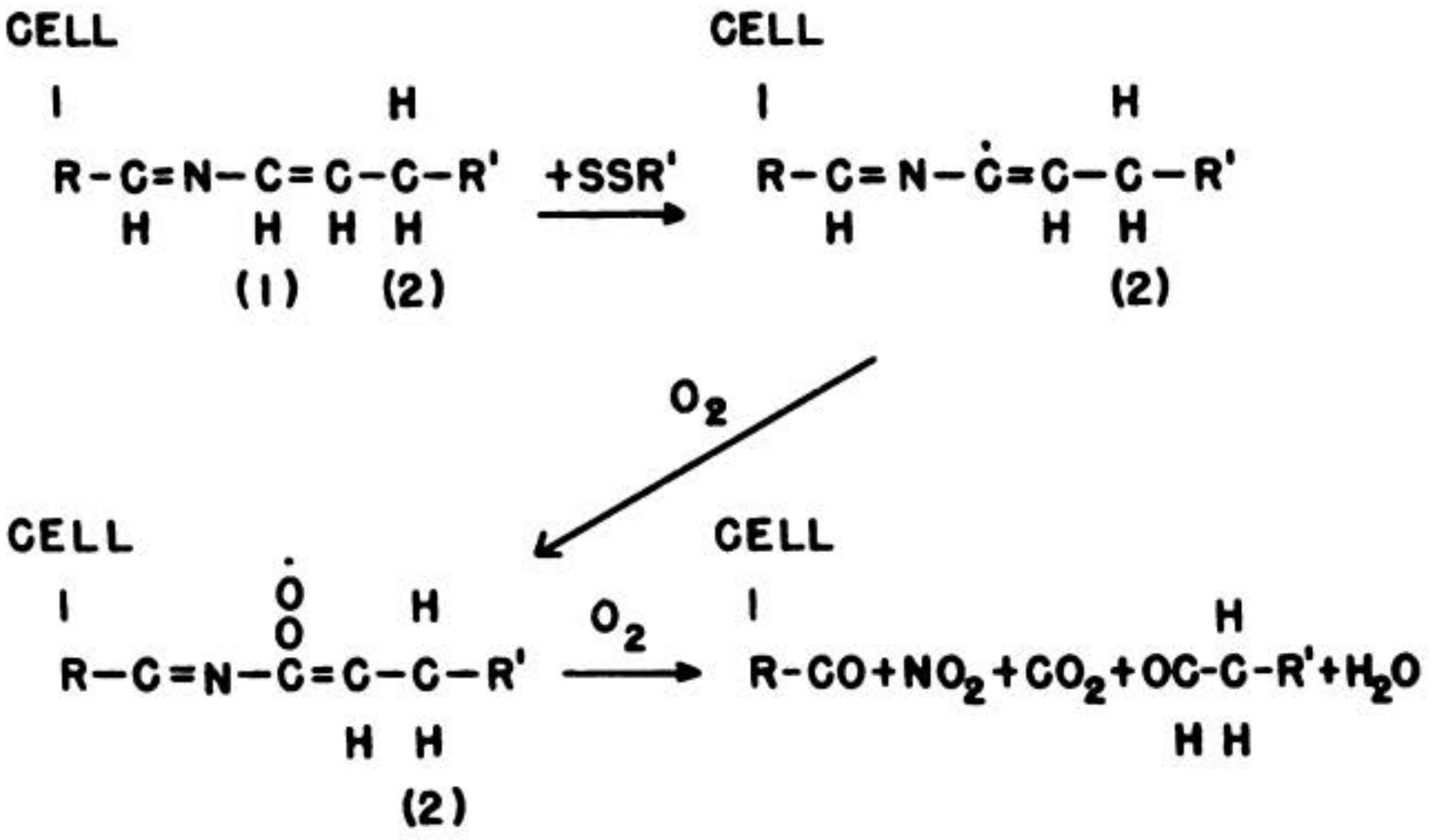
The products are hydrogen peroxide and ammonia, oxygen being the ultimate electron acceptor, and Flavin-Adenine dinucleotide, the electron carrier. The carbonyl group of the Diamine Oxidase (DO) serves as a dehydrogenator removing the hydrogen atoms from the carbon atoms alpha and beta to the amine group that is to be removed. A double bond is thus produced. The other amine group is not attacked. When the hydrogen atoms so removed are taken up by the dinucleotide, the DO carbonyl group is ready to start another cycle of detoxication. The steps are oxidation, a reversed Amadori shift, so to speak,

and then hydrolysis removing the amine group as ammonia, as shown in the diagrams.

Oxidative Separation of the Integrated Pathogen

Our postulate proposes oxidation all the way through. Dehydrogenation alpha to a double bond of the pathogen that is integrated with the FCG by an azomethine condensation. This may take place close to or far from the point of integration. The free radical produced adds molecular oxygen, becomes a peroxide free radical, and splits, producing two terminal carbonyl groups. The double bonds of the carbonyl groups activate the alpha placed hydrogen atoms facilitating further dehydrogenations by the SSR carbonyl group, and thus whether the azomethine double bond yields an oxide of nitrogen in the pathogen, and a carbonyl group as was originally present in the host cell as its FCG, right at the start, or as the end reaction of a stepwise oxidative progression starting at a distance, the results are the same, restoration of the host cell FCG and destruction of the pathogen with the production of energy. The diagrams illustrate.

If oxidation starts at a distant position, more energy is produced, and this may be the way viruses are separated with return of the energy to the host cell that was taken up by the viral colony during its vegetation. The same holds for the separation as in (D) to follow.

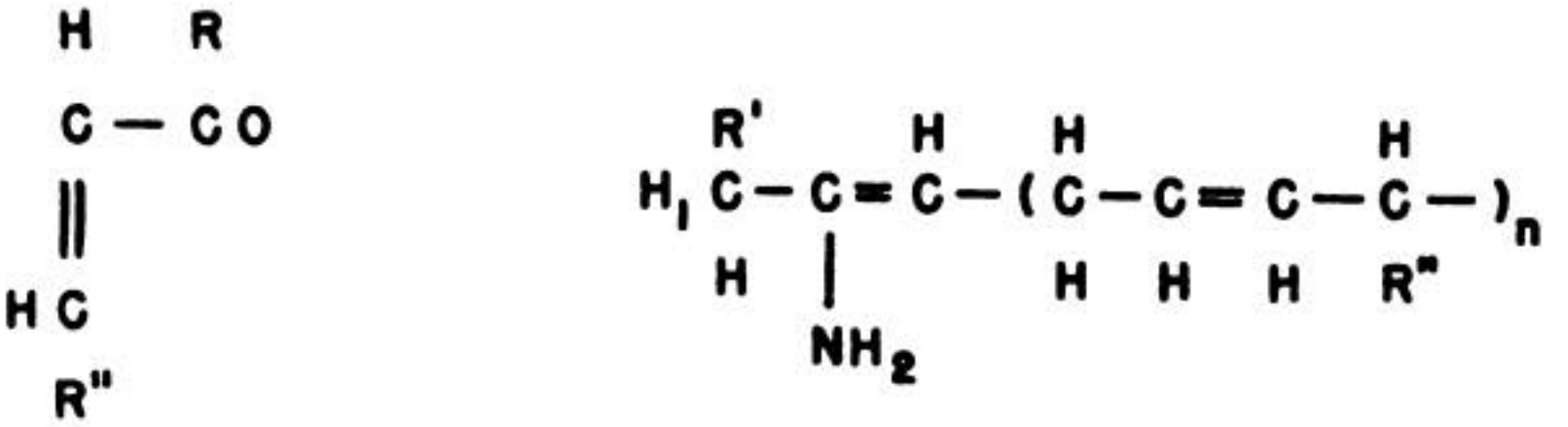


Integration of the Pathogen with the Host Cell Energy Producing Mechanism by a Free Radical Addition to the Double Bonds that Activate the F.C.G.

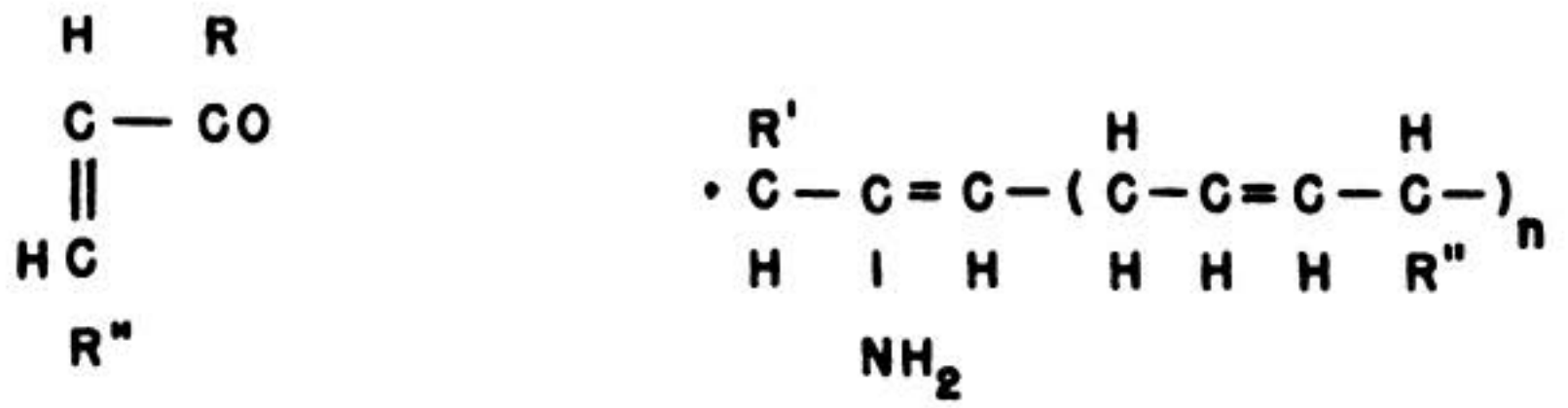
D

Host Cell Energy Producing System FCG and Double Bonds.

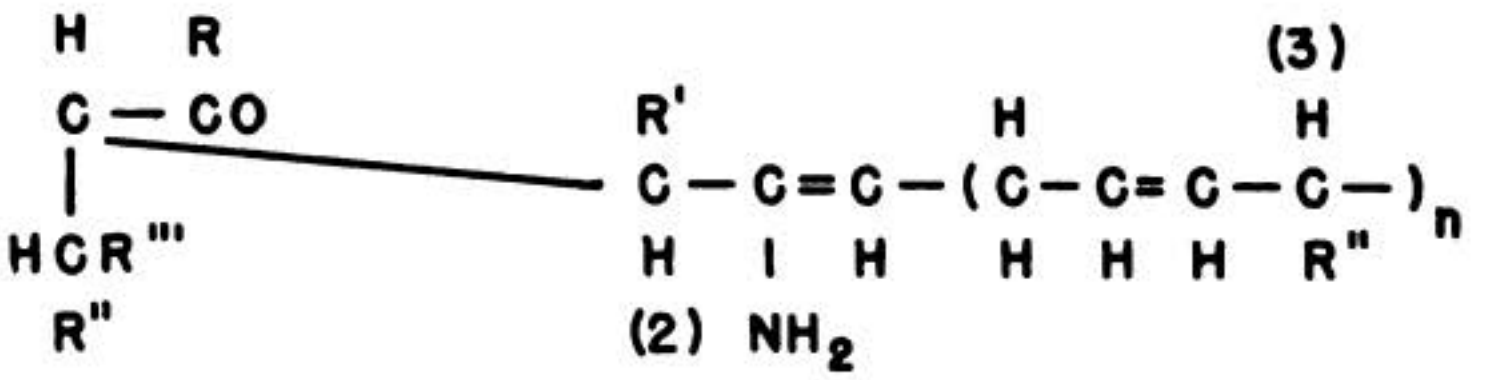
Pathogen (P)

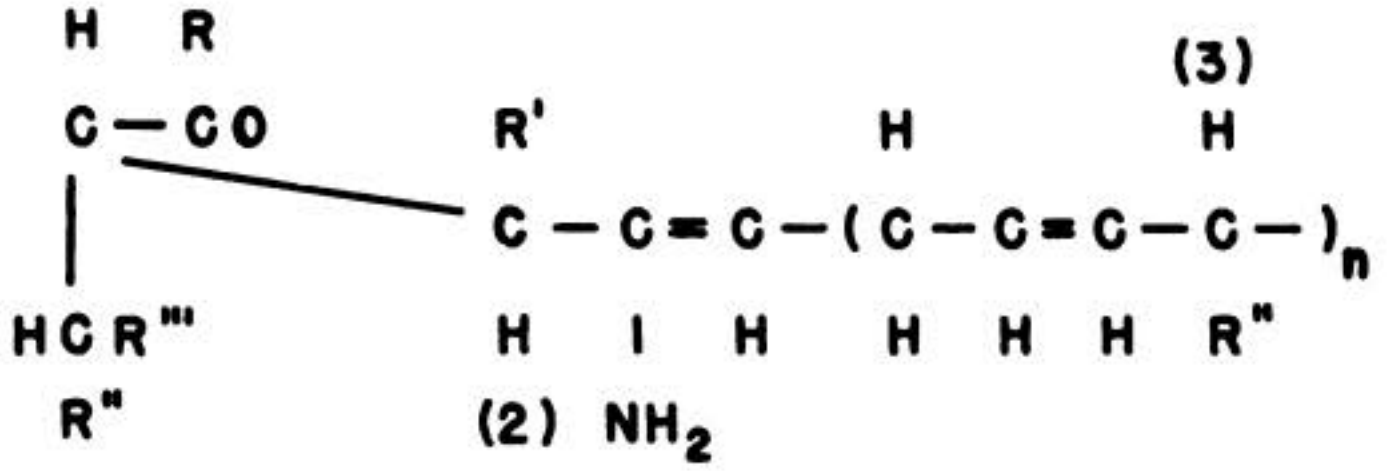


When the Pathogen (P) is dehydrogenated during anoxia a free radical is formed at position (H_1) which adds to one pole of the Host Cell ethylene linkage that activates its FCG, thus,—

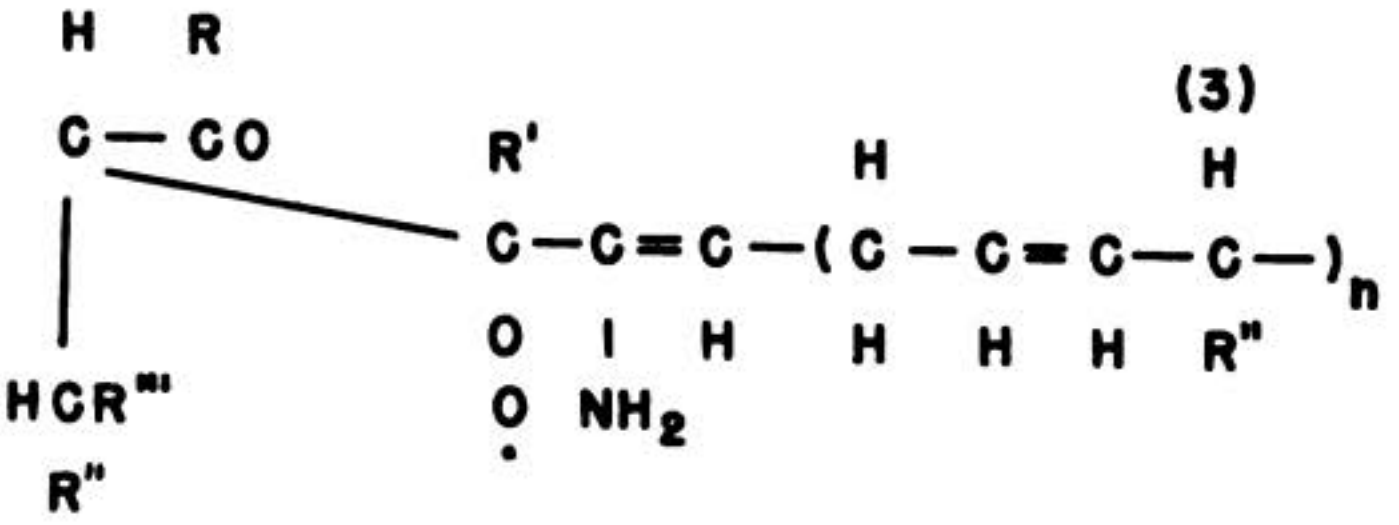


As the pathogen adds to one pole of the activating double bond of the FCG, the other pole of this double bond becomes a free radical which makes an addition to whatever group it has the most attraction for which is represented by R‴, thus,—

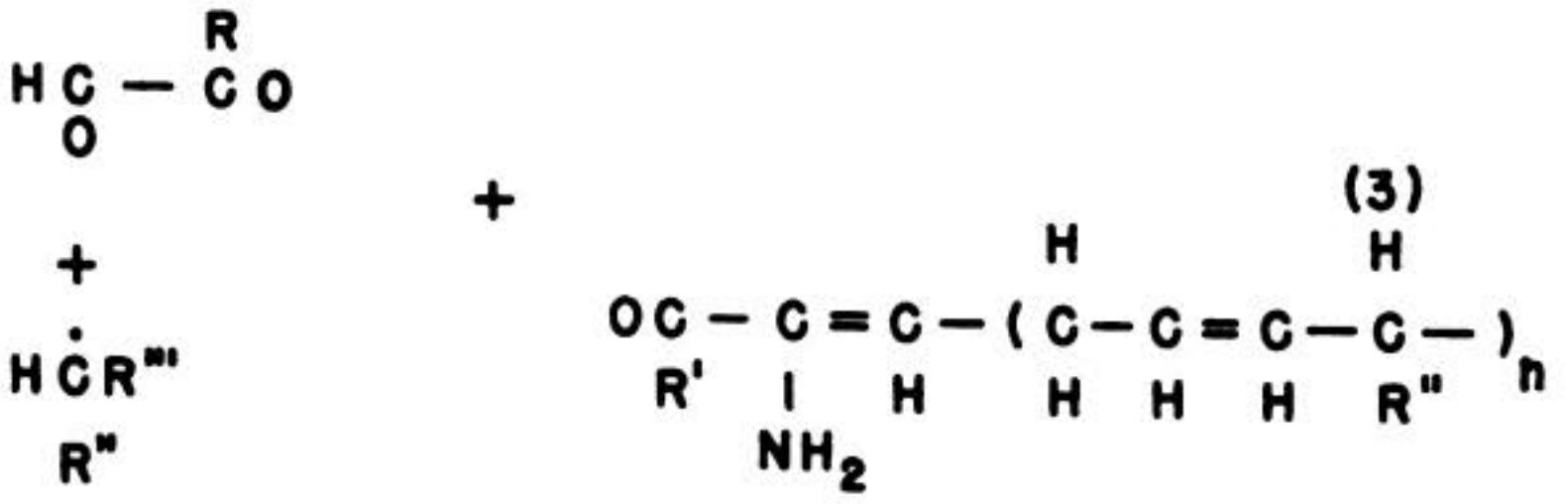




+SSR and OXYGEN to form a free radical and then a peroxide free radical at position (2)



Cleavage then takes place leaving the host cell FCG conjugated with a carbonyl group replacing the former ethylene linkage that activated it.



The residue left by the cleavage at the activating position is a free radical which adds molecular 0_2 to undergo further oxidation.

If the H (3) is the most exposed and activated hydrogen atom, it will invite removal by the SSR, and the free radical formed there in the presence of molecular oxygen becomes a peroxide free radical and splits the molecule with the production of two terminal carbonyl groups as in the former instance. Each carbonyl group double bond serves as activator of the hydrogen atom in the alpha position to it, and the process is repeated step by step until the FCG is reached where a carbonyl group is formed, and the electrons it can contribute, go to the FCG to activate it as the ethylenic linkage had done previously. The FCG is activated to a higher O/R potential than formerly as the carbonyl group is a better electron doner than the ethylene linkage. Also, the carbonyl group does not add free radicals so readily as does the ethylenic linkage and hence the FCG system is protected from inactivating additions; and a higher degree of immunity is gained than existed before. This hypothesis seems to answer the fact that our cured patients are more resistant to disease than they formerly were, and in fact more resistant than others usually are. Since viruses are built up of similar units as a copolymerization process, the last monomere added is most exposed, and oxidation would start at this position. The energy liberated would then pass on to the host cell and support its reconstruction, so that when the paralysis as in Rabies and Polio has disappeared one knows that not only the virus is separated out of the way, but the host cell functional mechanism has been restored. Since so long as the virus is integrated with the host cell, function is blocked, and the functional mechanism is progressively destroyed to support viral vegetation; the reversal of the process with reconstruction of the host cell so function has returned means that the energy for this reconstruction must have come from the oxidative destruction of the virus as there is no other source for such energy while the FCG is blocked. The facts have been demonstrated. The explanation, of course, is a matter of choice as limited by the facts.

IMPORTANCE OF DIVALENT AND MONOVALENT CATION BALANCE

The parathyroidectomy experiments emphasized another matter of the greatest clinical importance. It is the relation of the balance of divalent and monovalent cations to cell irritability. Ordinarily the normal cell presents a disposition of the cations, adsorbed and unadsorbed by the cell colloids, so that a water in lipoid phase is maintained. The lipoids thus diffuse to the perifery of the cell forming a concentration of lipoid there that serves as a limiting membrane, and tends to shut out water soluble materials, while water and its soluble materials tend to concentrate in the center. The effect is reversed by an excess of monovalent cations and accentuated by divalent cations. The monovalent cations thus tend to cause a lipoid in water phase with the lipoids concentrated at the center and the water soluble materials and water at the perifery. This tends to increase the entrance of water soluble substances into the cell. Sodium and Potassium thus tend to increase the cell's exchanges, and the entrance of water soluble toxins. Calcium, magnesium and strontium tend to reverse this

situation. Calcium is important with magnesium in lessening the cell irritability, while sodium and potassium increase it. Variations play their roles in functional activities of all cells and will be apparent also in the functions of heart muscle.

After parathyroidectomy, the increase in irritability of the nervous system followed the loss of calcium from the tissues. The entrance of guanidin toxins gave convulsions of greater severity the more the calcium that was lost. Calcium transfusions reduced the convulsions for a time, that is so long as the kidney function was maintained. Other solutions as of potassium and sodium and even distilled water did the same for a time, so diluting the blood and washing the toxic element out via the kidneys, showed that calcium metabolism was not the whole problem after parathyroidectomy. However calcium, magnesium and strontium had a greater depressing effect than the monovalent cation solutions, and the suppression was greater in the order of the divalent cations named. Indeed strontium put the cells to "sleep" as it were by its excessive effect. It was such facts as these that convinced the writer that the function of the parathyroids was not just a matter of calcium metabolism as Carlson and his school claimed. As soon as the hemorrhagic glomerular nephritis after parathyroidectomy prevented the washing of the guanidins out of the blood, no amount of calcium or any other solution could prevent or hinder the convulsions and deaths of the animals. Thus it was evident that a toxic element existed, and the writer set out to find it.

A complicating factor is the normal place of calcium in activating ATP-ase for the transfer of energy into the working mechanism of the cell. Here is a chance for energy transfer also to the surfaces of the tissue colloids that aids their dispersion and oxygen transport, to get rid of toxic materials and preserve a better colloidal structure in the cell throughout its contents. Normal cell excitability and response to stimulus is thus maintained by this other function of calcium.

When one sees exaggerated reflexes, or persistent excitability of a tissue one therefore thinks of the dispersions of the lipoids to or from the cell surface and also the disposition of the monovalent and divalent cations. In cases of high irritability, one would not give transfusions of solutions that would increase the monovalent salt content. Isotonic salt solutions made with sodium chloride would not be used unless the sodium is balanced by calcium and some sort of a Ringer's solution would be used. Even the sera of the blood banks could be taken from individuals with high sodium chloride blood content. These matters deserve consideration.

Our diet calls for calcium in plentiful amount, as crude calcium carbonate. We also give the potentized calcium as carbonate to cancer patients. This is to diminish the nutrition of the sodium rich cancer cells, and to reduce the pain caused by the ever present incompletely combusted metabolites that enter the nerve endings in the affected areas. Calcium and good intestinal lavage and a well chosen diet go a long ways in abolishing the pain in cancer. Likewise the carbonyl reagents tend to burn the incompletely combusted metabolites out of the way so they cause no more pain. The cancer cells also under good

calcium supply do not tend to swell up so much and cause so much pressure, for the reasons just mentioned. All of these factors enter the treatment of the neuroses and psychotic states.

An illustration of the parallel run of toxins with failure of the use of calcium is seen in the treatment of dairy cattle that were badly diseased by hemolytic staph. aureus infections of the mammary glands. After the carbonyl (SSR) therapy was used, both the hemolysins disappeared, and the calcium content of the blood and milk increased. The lactiferous cells were able to use the calcium for cell building and for milk production, and the germ no longer produced the hemolysins. This work was done at the University of British Columbia and by the scientists of the Ministery of Agriculture. Thus the toxic inhibition of the use of calcium must be removed to obtain its full biological effect.

STERIC ADVANTAGE AND HINDRANCE

There is a problem bound up in the activation of atomic groups by electron shifts as determined by the character of a substituent for hydrogen at a carbon terminal of a double bond. This is the steric arrangement of the groups concerned. It is observed that the groups taking part must lay in the same plane. We took data on this matter in our earliest observations. Comparison of the action of fumaric acid, maleic acid and maleic anhydride were made on the course of glandular tuberculosis where the enlargements were easily measurable — the cervical and supraclavicular glands. It was found that fumaric acid had no action, maleic acid showed some, and maleic anhydride gave a satisfactory response. This response was not continuous longer than a few months and the dose had to be repeated. This is not what we were looking for. We desired a continuous curative action that kept up until the patient was fully cured. Never the less the observations showed the effect of steric influence. Reference to these early experiments was made in our Court trial in 1943, where comparison was made with benzoquinone.

In benzoquinone one finds the carbonyl groups activated by conjugation with two ethylenic linkages, where no hindering substitutions of the hydrogens are had. They all lay in the same plane. High grade continuous curative action was had in a wide field. In fumaric acid, which is the trans-isomere of maleic acid, only one carbonyl at a time is coplanar with the ethylenic linkage, while in Maleic acid which is the cis-isomere all double bonds, carbonyl and ethylenic lay in the same plane. Here however hydroxyl in the carboxyl group acts as a substituent of a dampening nature against carbonyl activity. In Maleic anhydride the two hydroxyls are removed, and this offers an advantage, but not one that would equal the advantage given by the presence of hydrogen. Indeed in such an ideal molecule the energy content is too high to let it exist as such. So as such it was not practical.

The energy content of maleic acid is 7 large calories higher than that of fumaric acid, and hence it is more reactive. The ionizing power of Maleic acid

is increased by electron shift from the ethylenic linkage and also from the other unsaturated (carbonyl) group, tending to liberate one hydrogen as a proton. Other properties show this increase in energy content due to all double bonds laying in the same plane so far as the hydroxyl groups allow. This example of steric influence on reactivity must be helpful in understanding the change in reactivity of atomic groups concerned in the mitotic act — that is, the shift of energy that forces cell division. For example observations on fumaric acid by Friedman et al. show that fumaric acid has no influence on mitotic rates while maleic acid and its two methyl derivatives all showed strong antimitotic effects. Friedman, Marrian, and Simon-Reuss, (Brit. J. Pharmacol., 3 (1948a) 263, attempted to learn if the antimitotic action was due to sulfhydryl addition, (Biesele p. 34. Mitotic Poisons and the Cancer Problem, Elsevier, 1958). But it was found that the addition products were not active. The chlormaleic acid and chlormaleimide added sulfhydryl in the same way as if non-chlorinated, but were inactive as mitotic inhibitors. Activation of carbonyl by electron drift from the other double bonds was not considered by these gentlemen, even though the quality of the influence of the chlorine substituent on the distribution of electrons to the carbonyl groups both in the cis-acid and anhydride forms was to withdraw them and decrease the all around electron density. This shows that the antimitotic effect is due to carbonyl action, as this writer has interpreted it to depend upon electronic activation, throughout his whole postulate. Due to the presence of a conjugated ethylenic linkage, sulfhydryl can be added where such an ethylenic linkage can contribute electrons to the carbonyl group, but that the addition of sulfhydryl has nothing to do with the antimitotic act is right in line with this postulate. This is just another of the antimitotic agent puzzles this postulate has solved long before antimitotic work was ever undertaken. The unheeded data in the hands of the investigators showed that the antimitotic effect was due to carbonyl activity enhanced by electron contributions from the other conjugated systems of double bonds laying in the same plane. Thus the steric effect of a virus or carcinogenic chemical that becomes integrated with the host cell must be viewed in the light of its effect on energy distribution and reactivity. The Postulate of this thesis that calls for an oxidative separation of the pathogen from the host cell where a hydrolytic effort could never bring its release, is in line with this postulate. Indeed, it is a practical application of the idea.

APPENDIX III

A variety of cases with fuller discussion of Case I, the exophthalmic goitre case, showed how the basic pathology was the block in energy production and utilization in the functional mechanism. The carbonyl group of function was combined with the pathogen or its activating double bonds were so combined. It could neither dehydrogenate nor form an azomethine bridge for energy passage, in frank infection, allergy or neoplasia. Many conditions that were observed to support our thesis, were not touched upon. So to make the report more complete we will show how the two obvious metabolic disturbances, epilepsy and sugar diabetes, conform to the same pattern also.

DIABETES

In this disorder which is a symptom of a number of diseases, there is the suppression of function of the Islets of Langerhans, even though microscopically the cells may appear normal in number and structure. They may be diseases that destroy the islets and thus cause failure of insulin production. But in the vast majority of cases the trouble is functional and correctable in line with our postulate. Enough clinical proofs were given before the Federal Court and the Federal Trade Commission to establish this fact. Two recent cases are given here besides to illustrate two facts which explain two different forms of response. In one, the high blood sugar was reduced to below normal indicating that new islets of Langerhans were formed to compensate for the loss of insulin. But even these were blocked by the inhibitor toxin, and when they were all liberated, there was an excess of insulin poured into the blood. The other extreme is the reduction of the high blood sugar slowly and gradually, showing that the islets were deficient in number and had to be repaired or increased to accommodate the demand for their product. In both the general health was restored and thus the basic or constitutional cause was removed. In both the high protein diet was changed for an unrestricted carbohydrate diet of fruits, vegetables, cereals, bread, fats, honey and molasses. No proteins of animal origin were allowed and coffee. tea, alcohol and tobacco were forbidden. No insulin or other antidiabetic drugs are given; the patient is left under the influence of a returning islet function as its cells are liberated from the paralyzing toxin. The blood sugar then comes toward normal as fast as the islet cells are relieved. Sometimes the blood sugar goes below normal as the compensatory hyperplasia of islet cells are also liberated from the toxin and go to work. Where the toxin has been integrated with the islet cells longer than the life cycle period of any of these cells, they naturally die off and are not replaced and so a deficit of islet tissue is observed in the blood sugar level that is above normal after all of the toxin is removed. To replace these islet cells takes time as we will show by cases examined at this particular period. One must remember that the islet tissue produces insulin and hence no great deficit of this material is ever present in normal islet cells to stimulate a compensatory hyperplasia as occurs in muscle cells that are exhausted by work and call for more tissue reconstruction.

Therefore compensatory hyperplasia in islet tissue is comparatively slow and the time to reach the usual normal level varies with the length of time the diabetogenic poison had been active. The recovery of the islets from the integrated toxin that had simply inhibited their function is rather rapid, and is accompanied by systemic reaction in which the tissues are generally cleared of toxic effects. The overweight and weakness is soon overcome and other disturbances leave, — allergies, etc. The recovery of the destroyed and deficient islets to a normal quota is slow as was just explained. However, the whole picture of diabetes changes with this exposition. These cases again show that the work of the SSR is to start an oxidation progression in the toxin that is integrated with the tissue cell and blocking its function. The difference between such a toxin integration and the viral integration will be seen by comparing the recoveries of diabetics with paralytic nerve infections in which paralytic dog distemper serves as a good example.

The cases submitted here are only to show that the SSR is not a substitute for insulin in the oxidation of sugar but is given to remove the pathogen that blocks the production of insulin.

Case of Sr. L. S., 53 years old — Dr. Jayme Treiger

He had a rich venereal past, malaria at 21 years of age, and was operated for varices in 1941. He complained of vertigo, edema of the lower extremities, grade 2, small varicosities. Aorta palpable, Fundus oculi, — veins with second grade manifestations (Wagner), Blood pressure *24/13, Pulse 96, Glicemia 112 mgm %, was placed on hyposodic diet, did not tolerate CIK, received clortiazamide because of the edema. This reduced his blood pressure to 20/10, 18.5/11, and 21/11 with vertigo.

On January 19, 1960, there was dyspnoea and B.P. 20/11. On February 14, B.P. 22/12, pulse 84. Rauwolfia, clortiazamide, Naturetin K, Mecanil, were given, April 10. On May 16, vertigo, tachacardia, and dyspnoea, after lying down, B.P. 25/13, pulse 90/m received more energetic hypotensive drugs and Alcachofre tincture, B.P. 22/11, pulse 84. On August 18, epistaxis, B.P. 26/12, dyspnoea, — Reserpine, Alcachofra, weight 85 kgms. Constrictive feeling in the neck, *Blood sugar 320 mgms. %*, urea normal.

Treatment was given on September 24th, 1960, only a few drops intramuscularly of the serial system of carbonyl groups, one-tenth of a microgram. He had a reaction on the next day; the edema and constrictive feeling in the neck disappeared quickly and three weeks after the treatment he felt very well, weight 82 kilos, blood pressure 17/10. *Blood sugar 75 mgms. %*. He has remained in good health. The clearing up of the basic cause is evident in the normalization of the other evidences of disease, to which the diabetes was part and parcel. *"All anti-diabetic drugs including insulin were stopped before the SSR was given."*

Case of Sra. M.P. 51 years of age, was given the same dose of the SSR within the same hour as the preceding case, so as to facilitate comparisons

* The blood pressure is given in centimeters.

chronologically in their recovery processes — the depth of the pathogenesis with the recovery rate.

She had been diabetic since 1955, but was first seen by Dr. Treiger on August 24, 1959. The first complaints were articular pains, thirst and excess weight, 95.6 kgms., height 1.58 meters, blood pressure 17.5/9, edema grade 2 in both feet. Glycemia Aug. 1959, 240 mgms. %. Folin-Wu. Urine S.G. 1.036, glucose 4x. As she did not accept very well the new anti-glicemiant, Diabenase, she started to receive insulin and Protamin during the whole of 1959 and 1960. But the blood sugar stayed always at higher than normal levels even though the insulin was taken always with 40 U PZI, running generally around 220 and 240. In June 1960, the *blood sugar was 340 mgms. %*, while receiving 60 U PZI, and by September 15, it rose to *398 mgms. %* while receiving 60 U PZI. Before being given the SSR, she was taken off of insulin completely and all medication was stopped.

Treatment—On September 24th, 1960, her weight was 94.5 kilos. She was given a few drops of the same solution as the other case. In five days the sugar dropped to *210 mgms. %*. In two months her weight dropped to 89.5 kilos even though she was taken off of proteins and given a liberal vegetable, fruit, cereal diet. The edema of the legs was leaving within a week of the treatment, and she had a splendid reaction of a few days of fever and aching in her legs and bones generally. Her whole health aspect changed for the better. The blood sugar on November 30, 1960 was 160 mgms. %, and she is taking no insulin or other drugs. Her appetite is good and she feels the best she has ever felt. The blood sugar is not expected to reach normal until islet tissue is reconstructed to normal. It is expected that this will require from three to six months.

One would ordinarily think that the rich carbohydrate diet after the treatment would precipitate a sugar crisis. However, the relief of the islet cells was fast enough to prevent that as these cases show. Further our diet regime that cuts out animal proteins also reduces the production of the pathogen in the bowel. It will be seen that the whole aspect of diabetes comes up in a new light, as these and so many other cases show. Reduction of the blood sugar as observed in these cases of diabetes while on a non-protein, high carbohydrate liberal diet without taking insulin, has but one interpretation, and that is the islets have again resumed their function. In other words, the inhibitant has been removed. How rapidly injured islets can be restored depends upon the situation at hand, and the management of the case is directed accordingly, including the withdrawal of insulin. Epilepsy is definitely a metabolic disorder that may be subject to the same corrective therapy as the following case shows.

It must be emphasized sufficiently that while severe diabetes can recover after the SSR is given, this recovery is part and parcel to the removal of the pathogen and its effects generally. Moreover, while in an early mild case the islet cells can be liberated from their integrated toxin so as to fully restore islet function, in the long standing heavily intoxicated cases, the islet cells that remain as functionable structures may be reduced in number and the blood sugar will not return to normal until more islet cells are reconstructed. The

amount of reconstruction required in such cases will be proportionate to the length and severity of the toxic period of injury. A few more cases should be scanned to observe these points in their varying aspects.

The toxic injury may be dominantly cardiovascular.

J.M., age 61 years in June, 1960. She complained of pains in the limbs, dyspnoea after effort, constant cough, precordial pains intermittently, edema of the right ankle, and cholecystectomatized because of lithiasis. Physical examination revealed a blood pressure of 170/90; pulse 90; systolic murmur at A2; fundus oculi, hypertensive angiosclerotic retinopathia, grade 2; KWV, urine with traces of sugar. She was first given homeopathic remedies with improvement in the blood pressure. On 8/16/60 her B.P. was 140/80, but there was no improvement in the pains in the chest or limbs. Salicyates were given. On 9/14/60, glycemia was 190 mgms %. She was given a dose of the SSR and no other medication was given. She was given a rich carbohydrate diet and all animal foods denied. On 11/14/60, glycemia was 145 mgms %. On 12/2/60, glycemia 100 mgms % and she was entirely symptom free. Her blood pressure was normal. All the conditions cleared up at the same time on the unrestricted fruit, vegetable and cereal diet.

The toxic injury may be dominantly those of an old focal infection with allergic symptoms and lack of tolerance to antidiabetic drugs and a failure of large doses of insulin to reduce the blood sugar notably with loss of resistance to infection.

In these cases the disappearance of all toxic effects comes quickly and the blood sugar drops to a level (without medication and on an unrestricted carbohydrate diet, without animal foods) to a point that indicates how much of the islet tissue still remains undestroyed by the pathogen. Then the reconstruction of islet tissue goes on slowly as was shown above, and the rate will depend on the length of time the pathogen was active. In this way one illustrates that the SSR is a liberator of the functional mechanism, restoring it to normal action on a broad or maybe on all functional planes, and not simply an agent to oxidize sugar. It demonstrates itself to be an oxidizer of all integrated toxins we have investigated so far with the widest possible symptomatology. The following two cases are selected to illustrate a variety of tissue injuries a diabetes producing toxin can cause and the degree to which the islet tissue may be paralyzed and also destroyed.

Mrs. P. S., age 50, in August, 1956, started with diabetic symptoms after a hysterectomy. Her blood sugar was found to be 430 mgms %. With insulin it came down to 173 mgms %. After August, 1956, she did fairly well, using Insulin 20 U, and Nadisan, 3 tablets daily. After May, 1958, she started taking Diabenese in varying amounts. On September 2, 1959, her blood sugar was 195 mgms %. In November, 1959, it was 240 mgms %. In January, 1960, the blood sugar was 300 mgms % with Rastonin, 2 daily. It stayed about the same until August when Rastonin had become intolerable due to toxic effects and was stopped on September 29, 1960. Her blood sugar was 325 mgms %. On October 4, 1960, she was given the SSR. She had a

strong reaction on the third day that lasted a week with general achiness, fever, chills, etc. The reaction focalized as a heavy inflammation about an old tooth root which opened and cleaned out with a good hemorrhage and then cleared up. Her weight dropped from 75 to 66 kilos by the middle of December, 1960, her blood sugar was 260 mgms % and she felt very well without any antidiabetic medication, and on a diet of unlimited carbohydrates and fats and without animal proteins. This improvement is expected to follow the pattern of other cases with decrease in the blood sugar as fast as islet tissue is restored. At this stage the case differentiates between inactivation and destruction of islet cells.

Mrs. G. S., age 38, in October, 1958, showed a different type of toxicity in which she could not use insulin without toxic injury and she was unable to heal any wound or furuncle. This all cleared up concomitant with the correction of the injury to the pancreas. In this case as in the former case a strong systemic reaction ushered in the improvement. The insulin poisoning showed as an echimoses and general itchiness and rheumatic symptoms.

She was diagnosed as diabetic in 1957 since the wound from a cholecystectomy done 2 years earlier would not heal and the blood sugar was 248 mgms %. She was given classical treatment and diet like the rest, first 40 U PZI, then 60 U, then 70 U, and then 80 U on November 6, 1959, as the blood sugar increased to 440 mgms %. On 80 U daily, it dropped to 300 mgms %, but echymosis and itching resulted. Two months later she presented an eruption similar to the luetic roseola, but the blood tests were all negative for syphilis. The insulin was reduced to 50 U, PZI, and was fortified with Rastinon, 2 tablets daily. The blood sugar was 380 mgms % on August 18, 1960, and she was put on Diabinese, 2 tablets and then 1 tablet per day when the blood sugar rose to 394 mgms %, September 17, 1960. She was given the SSR on October 19, 1960, with one Diabinese tablet and the blood sugar jumped to 360 mgms %, as part of a strong reaction. The Diabinese was stopped and a half dose of the SSR was given on November 18, 1960. On December 13, 1960, she was feeling very well, her furuncle healed without help, and the blood sugar was 265 mgms % on an unrestricted carbohydrate diet without any medication or animal proteins. The break in the survival chemistry showed in more than one direction in this case, and showed not only a paralysis of the islet cells, but their actual destruction which is not yet fully repaired. The job of repairing the islets is not a function of the SSR, but is made possible by the removal of the toxin that was integrated with the islet cells and destroyed them. With this toxin out of the way reconstruction can take place as in other cases.

That the pattern of integration of the inhibiting toxin with the FCG is the same in islet cell block and in hindrance of other cell functions is well demonstrated already. Moreover, no matter how symptomatology of nerve cell hindrance may vary, the architecture of the block is the same. The severity of the symptoms does not determine the length of time required for recovery, but the length of time the tissue has been occupied by the pathogen does determine the time required for restoration to normal. Another case must be sketched in brief to illustrate this point and to show that sleep is an active

function involving energy generation and use. This case could not use the energy because of FCG block. There was also an islet block of moderate degree, the blood sugar being only 161 mgms %.

Mrs. M. S., age 42 years, came to Dr. Treiger in December, 1960, in a highly nervous state. She was without thyroid symptoms, complained of utter inability to sleep for over six months and had been placed on the whole list of depressant drugs by different specialists. These drugs did not help her in the least, but even as their dosage was increased to the limit of her tolerance, she became steadily worse. She developed into a melancholia with a compulsion that she wished to die which became ever constant. She was in terror and could stand it no longer. Dr. Treiger removed all drugs and the condition remained the same, fat and all, presenting a perfect homeopathic picture of treatment with Alum. He gave her Alum in carefully selected potencies, but it had no effect. He then gave her 2 millimicrograms of the SSR intramuscularly. At the time of treatment, the blood sugar was 161 mgms %. The change for the better was rapid so that her whole physical status improved, the high excitement state left and she calmed down so that in less than a month she was sleeping soundly three nights a week, and the other nights were improving steadily. The desire to die faded away and the melancholia changed to a habitual cheerfulness. The blood sugar dropped to 100 mgms % within the month while on a meatless, high carbohydrate, honey, molasses diet. Here the clinical facts teach something that laboratory research has not fully settled about the physiology of sleep and its functional mechanism, namely that energy production and utilization are essential features and that the carbonyl group is concerned in mediating both features of the function just as in any other tissue function, islet cells, thyroid cells and all the rest.

It is pretty well demonstrated now that the SSR does not do the work of the insulin, but removes the disease. This is seen in Varicella, a viral disease with a natural collateral control. Two brothers are concerned. M. M. P., age 11 years, had fever and Varicella. The SSR was given to him on 12/3/60. The next day the fever increased to 39°C. On 12/5/60, the second day after treatment the pustules stopped coming and all of the lesions started to improve. He was feeling well and in a few days was all healed. His brother, age 6 years, refused the injection when he developed Varicella on Dec. 15, 1960. Four days later, 12/19/60, the Varicella showed the classical development with new pustules coming in waves. He ultimately got well, but the involution did not start within 48 hours as in the SSR treated case, but was still developing in the classical fashion as is usual for days afterwards.

To further define the position of the Functional Carbonyl Group and its activating double bonds of the Koch Postulate a review of a case of antibiotic resistant gonococcus infection will help. Mr. J. V., age 25 years, presented an annual venereal infection. Finally the antibiotics were not effective and he did not do well on homeopathic drugs either. On August 28, 1959, after an additional exposure, he presented an acute Blennorrhagia confirmed by bacteriological examinations. He was energetically treated with Penicillin, Terramycin and Tetracyclines without the least effect. On October

23, 1959, he was given a dose of Benzoquinone, 6X dilution, when the bacteriological examination showed the infection was the same, a rich flow of intracellular and extracellular Gonococci laden pus. Examination one week later showed no improvement so he was given a dose of the SSR, 9X dilution on October 31, 1959. One week later, November 7th, he was feeling much better, including the prostate pain which had disturbed the passage of feces through the rectum. On the 9th day he had a reaction and on the 16th the urinary sediment still showed some intracellular diplococci. He was given a dose of the SSR, 10X, one-tenth as much as the former dose, on the 16th of December, 1959. Three days later no more intracellular diplococci were found in the urinary sediment, but some extracellular germs were still present. Three weeks later he had a general systemic reaction of grippiness. He felt well thereafter, but there were still extracellular diplococci until the middle of March, 1960, when the dose was again repeated. Ten days later a systemic reaction occurred and no more secretion was to be found. His health improved in every way. On May 30, 1960, the urinary sediment still showed some extracellular pleomorphic germs. On June 7, 1960, he showed a gain of 10 kilos in weight, the best of health, no longer any signs or symptoms of the old or lasting infections, the prostate was normal and the urinary sediment entirely negative. He has so remained.

Here we see the hang-over of the earlier gonococci infection as extracellular diplococci that required three injections of the SSR and six months to be cleared away after the acute infection was gone. This shows also that the SSR is not an antibiotic affair at all, but works on an entirely different principle. This point must be emphasized here. This final review shows besides that viral infections which do not respond to antibiotics either, are correctable immediately by the high potency dehydrogenations secured by the SSR, and the free radical and peroxide free radical carriers of the oxidations that continue the separation of the pathogen from the host cell functional mechanism.

In the diabetes cases it was seen that the longer the pathogen is integrated with the islet cells — not only is the inhibition of function proportional, but the amount of destruction of islet cells is also proportional. This is not a similar destruction to that which accompanies viral host cell integration, but one that blocks the formation of new islet cells after the integration is established a long time, longer than the life of the islet cell would be. Then no reproduction would take place in such cells and the amount of islet tissue would decrease. In viral integration there is the active destruction of the host cell as its energy is drawn off by the parasite and its structural material is also removed to support the viral vegetation. There is no tissue cell reconstruction of the islet cells when the toxin is removed as occurs reciprocally when the virus undergoes stepwise removal by the oxidation process we outlined and leaves a restored tissue cell when the virus is fully burned away as we have demonstrated in the paralytic viral infections. Reconstruction in diabetes recovery must be carried forward by the liberated cells and as there is no utilization or depletion of insulin in the islet cells themselves, the shock for a compensatory hypertrophy is very weak as compared with that taking place in

PLATE 1.

PLATE 2.

PLATE 3.

PLATE 4.

PLATE 5.

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.

muscle when put under increased activity. Here the muscle fibrillae that do the hypertrophying are directly stimulated by work, while the stimulus to the islet cells is mostly a lowered partial pressure of the insulin at the cell's periphery and possibly some hormonal influence if such exists. Therefore the restoration of islet cells is a slow process that succeeds only with time.

The contrast can be seen in the case of paralytic Distemper in a dog. In such a case there is no chance to error by way of hysteria. In the dog case to follow in two weeks full restoration of nerve function was re-established, whereas in diabetes the islet reconstruction goes on for months before it is completed. Only the toxic inhibition of function is removed in weeks or rather a few months.

Singo, a 10-year-old pointer dog, one month before receiving the SSR treatment, became sad, inaperent and trembling. He ran away and returned with bowel and urinary bladder paralysis. He was lavaged and received Nujol that helped the bowel movements. He also received Penicillin, Alcachofra, Terramycin, etc., which was prescribed by the veterinarian who made the diagnosis of distemper of the nervous system since there were chills, fever, then a lowering of the temperature with paralysis of the muscles of the left costal region causing a curve of the spine and difficult breathing. These muscles also showed an atrophy. There was also a paralysis of the left posterior quarter so that the leg would give no support and he could not use the paw, raise or lower it. The photographs show his condition before and after treatment. Photographs 1-3 show this paralysis of the left posterior quarter when the dog is supported by his upper half and the right foot posterior is allowed to take some pressure for his support. The left posterior quarter was useless. The muscles of this limb were also atrophied. As none of the medication helped, he was given two ampoules of the cinomose serum. It did not help him either. The integration of the virus with the nerve cells was thus established in a symbiotic way.

The SSR was given October 14, 1960, to save the dog, as the veterinarian decided he must be sacrificed. The recovery took two weeks to be completed. The atrophied muscles soon became redeveloped and full normalcy was established.

EPILEPSY

Under the heading of Allergies Of The Nervous System, we gave a case of Psychomotor Epilepsy. This patient had taken thousands of dollars worth of suppressing drugs without suppression and steadily went on toward fatality. At the time that the experts agreed he could not live more than a few weeks, he was given two micrograms of parabenzoquinone in two milliliters of water intramuscularly, and was sent home as cured in five days. The experts who decided he was cured are the same men who gave the fatal prognosis after months of unsuccessful drugging. This was in 1941, at the famous government mental disease hospital of Rio de Janeiro. The reagent that

freed the tissues of their energy producing and energy utilizing impediment was a carbonyl group activated by conjugation with the double bonds of an ethylene linkage. In the case to be given here the reagent carried a series of carbonyl groups that contributed electrons to the carbonyl group that did the dehydrogenating, and thus started the oxidative removal of the pathogen. The following case also shows the constitutional nature of the disease.

E. A., 17 years old, first seen on August 13, 1958, had diphtheria in childhood. Started periods of petit mal with loss of consciousness in crises of from 5 to 10 minutes, getting more frequent, once or twice monthly, without convulsions. He suffered severe headaches since childhood. Blood pressure 14/5 cms. of mercury, Systolic murmur, grade 3 at apex, 2 cms., outside of the left midclavicular line 5th interspace also a diastolic murmur grade 1, at the same place. Pulse 86 per minute. There was a diagnosis also of persistence of the Ductus Arteriosus.

The Electro-encephalogram on August 22, 1958, showed:

"The basic sequence is several times disturbed by the interference of abrupt and irregular slow waves with 6-7, 5 c/s. of high voltage, diffuse and bilateral, predominating in the frontal-temporal areas. The hyperphase did not reveal any new fact.

"Conclusion: Abnormal electro-encephalogram, paroxistic cerebral Dysrythmia, difuse and bilateral, predominating in the fronto-temporal areas.

"signed, Dr. A. A. G."

Thereafter the boy was kept from work because of the losses of consciousness that occurred while under the regular treatment receiving constant doses of Trilafen and Mezantoine. The blood pressure came to normal levels, but recurred at times with the crises of loss of consciousness. Since December 1959, the epileptic attacks became more frequent in spite of regular doses of Mezantoine, Equanil and Promazionon.

In June, 1960, this treatment was interrupted so that the SSR could be given.

Results—After three weeks there were no more attacks, sleeps well, eats well, is no longer tired and without headaches. The electro-encephalogram was repeated by the same expert and the report is, September 30, 1960:

"Electro-encephalogram presents no evidence of abnormality."

The patient is ready to go back to work but this will be deferred until the EEG is repeated after a couple more months. However, it is established that normal function has returned, and the energy production and use is not interfered with any more. In these cases two different means of activating the dehydrogenator carbonyl group were used. Hence the correction of the pathology depends upon adequate dehydrogenation in the presence of molecular oxygen. The correction is constitutional evidently here, too.

LATE REPORT ON MRS. M. H., Case No. 49

To demonstrate the permanency of the cure of tuberculosis by restoring the cell functional mechanism, we give Dr. Paul V. O'Rourke's description of the status of both lungs which was made on August 1, 1960 from the latest X-Rays. This leading Roentgenologist states:

"Mrs. M. H.---- was seen by me on July 1, 1960. Her chest X-ray at that time showed the bony thorax to be intact, with the heart in normal position and contour. The left hemidiaphragm is normal in position and contour. The right hemidiaphragm is elevated because of a phrenic nerve paralysis done some 30 years ago. The left lung showed some increased markings, most of which are vascular, and a small amount of calcific deposits. The right lung is greatly reduced in volume because of the extreme elevation of the right hemi-diaphragm to the level of the third anterior rib. At the extreme apex, there are numerous small calcified areas, which are the result, undoubtedly, of her previous tuberculosis of some thirty years ago. No active lesion of tuberculosis or rarefactions resembling cavities can be seen."

Mrs. M. H. was treated in the terminal stage of this disease and at a time when her state of exhaustion was so desperate that even taking a radiograph would not be risked.

This recovery was obtained while at work after the active attack was subdued by the SSR and a few weeks of bed rest until the fever abated. It speaks for the curing of the tuberculosis germ and the ability of her metabolism to keep tuberculosis germs cured, and harmless, even perhaps useful.

EXERCISE AND REST

Enough exercise to fatigue the muscles a little each day is wise, but the heart must not be pushed too hard during such diversion. Walking on level ground serves well and there are plenty of games that stimulate the spirits as well as the muscles that are helpful. Common sense is the guide.

To rest well at night the bed must be wide enough to support the blankets. Narrow beds do not and the weight of the covers are supported by the patient. Further, the warmth desired is not conserved in a narrow bed as it is where the blankets can lay on the bed instead of hanging over the side towards the floor.

The blankets must be of a good quality, warm and light. They must NOT be tucked in about the feet so as to restrict motion or confine the feet. A neat bed with the patient laid out like a mummy is an atrocity. There must be freedom so that relaxation is possible. Stupid bed provisions call for narcotics, and narcotics defeat recovery. Such care must be avoided with our patients.

APPENDIX IV

As the work progressed it was deemed advisable to obtain data on the curative action of molar peroxides and their unsaturated precoursers. For an investigation of this importance the best talent available was necessary. The American research institutions were examined carefully and found wanting. Europe was likewise scanned until Professor Joseph Maisin was selected as the best prepared for a task of this type. Cancer was induced in many hundreds of mice and rats by the usual carcinogens and tumor transplants and treated with the peroxides of formaldehyde and formic acid and with unsaturated substances prepared at the time of their use. These latter substances were preperoxides by virtue of their high double bond content. The tables appended show the results of their use. These observations were made at Louvain in 1934-35 with many others.

The protective action was found on further investigation to be due to the free radicals formed by the dehydrogenating action of the carbonyl groups present in the dehydrated unsaturated bodies and the free radicals formed in the peroxides which gave rise to peroxide free radicals. A comparison with the highly efficient curative action of the reagent used in the Bandeen experiments, (pages 30-35) bears this conclusion out since in the latter experiments the very highest carbonyl ability to dehydrogenate was provided whereas in the Louvain experiments no attempt was made to boost any activity at all. Nevertheless the latter experiments show that it is impossible to prepare molar peroxides sufficiently clear of free radicals to serve for pure biological tests. The speed with which free radicals are formed by light and by simple rubbing of the dry crystals also shows this trend. This is one of the basic provisions of nature for the continuance of life on our planet.

Protective Action of Diformaldehyde Peroxide

The mice received applications of benzene solution; 1/200 benzopyrene three times each week. The peroxide was injected, 1 cc. solution 1/20,000, on the 35th and 65th days.

Groups	Mice At Start	Mice Surviving To First Cancer	To 147th day	Day Of Observation	Relative Percent Tumors	Relative Percent Cancers	Absolute Percent Tumors	Absolute Percent Cancers
				CONTROLS				
1	40	28	21	147th day	53	48	55	48
				INJECTED				
2	50	40	27	147th day	4	—	5	2.5

Peroxide of Diformaldehyde

1/20th of a mgm. as one dose was given at the end of the first month of benzopyrene application.

Groups	Mice Surviving To First Cancer	To	Percentage Absolute Of Cancer
Controls	28	120th day	18
		210th day	57
Injected	40	120th day	5
		210th day	7.5

1/10 mgm. of the peroxide of diformaldehyde was given as one dose intraperitoneally before the first benzopyrene application.

Controls	78	120th day	30
Injected	75	120th day	10

Mice receiving three drops a week for 6 to 7 weeks of a benzene solution of benzopyrene, of a strength of one to two hundred. For treatment each animal received one dose of ½ cc. of a 1/5000 solution of the peroxide of formic acid.

Groups	Mice Surviving To First Cancer	To	Percentage Absolute Of Cancer
Controls	68	120th day	45
		210th day	69
Injected	52	120th day	23
		210th day	31

For treatment each mouse received 1/10 mgm. as one dose of diformaldehyde peroxide every 14 days after the first application of benzopyrene.

Controls	78	120th day	30
Injected	30	120th day	9

Unsaturated Substances

All mice received twenty applications of a benzene solution of benzopyrene 1/200 three times a week for twenty doses, and then one dose of the unsaturated substances.

EXPERIMENT A

	Controls			*Injected*		
	Number Of Mice Alive	Percent Of Tumors	Percent Of Cancer	Number Of Mice Alive	Percent Of Tumors	Percent Of Cancer
Day 60	75	36	1	62	10	0
Day 80	72	50	8	61	30	1.5
Day 100	71	65	16	60	38	4
Day 120	66	67	22	56	38	10

EXPERIMENT B

Day 60	71	28	0	75	22	0
Day 70	70	40	5	69	27	1

One finds that the phenomena that control polymerization are active here in the molar peroxides and the unsaturated substances in bringing about free radical activities.

THYMUS GLAND DEFICIENCY AND MUSCULAR DYSTROPHIES

The patterns of endocrine deficiency vary in some instances from that exposed in the exophthalmic goitre case, Mrs. M.J., on pages 40-45. 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 cofactor, 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, the thymus and muscle deficiencies are complete until the block is removed.

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 nontoxic amounts of manganese to the tissues in general Josephson 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

high potential carbonyl groups of the text are the logical procedures. We will give a special discussion to this subject in the near future with photographs.

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 π electrons are mobilized toward the carbonyl group, and such substituents as CH_3, CH_2CH_3, $CH(CH_3)_2$ and $C(CH_3)_3$ 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 electronegativity 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 electronegativity 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 groups so that the hydrogen atoms tend to be liberated with ease as protons. This mobilization is seen when glucose or fructose are dissolved in heavy water. Here it is found that the hydrogen atoms trade places freely 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 for a covalent bond; 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. It is a force too long neglected in tissue chemistry, and even in chemistry in general, but it is basic to our thesis and has made our contribution possible.

Pathogens, like unsaturated fats, also invite dehydrogenations in various degrees. Here we postulate that a methylenic group positioned alpha to a double bond of an ethylenic linkage offers two activated hydrogen atoms; one is important for the integration with the FCG system during the anoxia and the other invites removal from the integrated pathogen by the carbonyl group of the curative reagent. (This dehydrogenation can also be accomplished by an appropriate free radical.) The activation of the pathogen's hydrogen atom 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 sulphydryl as well as 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-hydrogen or oxygen-hydrogen bond weakens it to facilitate dehydrogenation. The stage is thus set intrinsically in the pathogenesis for its oxidative reversal. The pathology actually provides for its correction. The philosophic implications deserve thought.

The basic pathology in endocrine, viral and neoplastic disease is, therefore, of the same pattern and depends upon the electron passing powers of an ethylenic linkage to activate the position alpha to it. The carbonyl group is an electron accumulator, but also an activator through its orbital mechanics.

THE IMPORTANCE OF THE FREE RADICAL

Recently Prof. Cheves Walling of Columbia University stated: "Twenty-five years ago free radical chemistry interested only a few gas-phase kineticists . . . It is remarkable that one set of simple principles is basic to such a diverse group of processes and products." As our investigations show, the service of free radical products and processes made it possible to understand and reverse the pathogenesis of cancer and liberate the host cell from fatal viral integration decades earlier than 25 years ago, and in fact, concomitant with the development of the plastic industry. Thus the free radical occupies an important field in biological processes also.

It made possible an explanation of the Pasteur Effect, and its reverse, the Crabtree Effect, which in our opinion is a means of measuring the contents of respiring elements that conduct cell functions. Crabtree (1929) showed that when glucose was added to cancer slices, the use of oxygen was inhibited in favor of glycolysis, which was not the case with normal tissues as liver and kidney cortex. Here addition of glucose stimulated respiration. It was also shown that such metabolically inert tissues as cartilage and kidney medulla exhibited the Crabtree Effect much like cancer tissue. Natal retina did also, until it matured and could function as an oxidation mechanism. Thus, where and to the extent that respiration is possible, addition of glucose will stimulate oxygen use, and where this function is limited, glycolysis is used to dispose of the added glucose, and the use of oxygen is depressed to the extent that common factors engage in both respiration and glycolysis. Thus cancer tissue has a limited oxidation rate or capacity which it cannot increase under stimulation, and is using to the limit all the time, thus revealing the inability to supply further oxidative facilities. What is the cause of this deficiency?

Aisenberg (1961) offered the two explanations that are supported by data given in this text, (a) the diminished amount of mitochondria in tumor cells, as Warburg also suggested; and (b) the statement forwarded by Chance and Hess (1959) that the respiratory elements are still present in normal amount, but are under a restraining influence which blocks the oxidation ability proportionately. This is exactly what our text has demonstrated together with an explanation of both the hindering mechanism's chemistry and that of the liberation of the oxidation mechanism.

Not only the FCG's of the various specific functions may be concerned, but also the carbonyl groups of the accessory factors to the oxidation process,

the electron or hydrogen acceptors as the quinones so recently discovered to exist in all living cells. Their structures are essentially carbonyl groups activated by conjugation with double bonds of ethylenic linkages. Similarly we find activated carbonyl groups in the keto-steroids, and the spent products of all such structures are hydrogenated, reduced to hydroxyl. In the case of the keto-steroids the double bonds are also inactivated, and so the molecule can not be reversed to function again, but in the case of the quinones the double bonds are not altered and reversal with return of function is possible. Thus co-enzyme Q can function as a co-enzyme over and over again as an electron transfer agent.

The quinone structure is also admirably adapted to such function so as to meet the requirements of specificity in oxidation-reduction potential and for selecting the specific materials it will react with in each particular cell activity. The substituents placed about the quinone's double bonds give the steric advantages and hindrances required for specific reactions and for elevated or depressed negativity and oxidation-reduction potentials of the carbonyl groups. Twenty years ago the most celebrated biochemists testified that the quinone structure had no place in biological processes in opposition to our thesis. Today, we know otherwise and the position of the free radical which can only be proven now by the application of postulates, cannot escape being demonstrated as fundamental to all living processes. The present writer's Professor of Chemistry was the discoverer of the Free Radical, Moses Gomberg. He developed his thesis as a pure piece of philosophy, but he lived long enough to see it become the basis of the plastic industry, and even learned of the first adventures of the writer in the interpretation and correction of pathologic states. We join Prof. Walling in saluting the glorious work of Gomberg.

In our early work we found that polymerizing unsaturated free radicals of low molecular weight stimulated cancer development decidedly, whereas large inert polymers blocked cancer growth and involution soon followed. Our conclusion was that the carcinogen integrated with the host cell nucleus by a copolymerization process of free radical double bond additions, whether the carcinogen be a polymerizing bacterial toxin or a vegetating provirus whose units were largely host cell nuclear products and this symbiosis held through the mitotic act. The small polymers offered more double bonds and free radicals for hastening the mitosis point, whereas the large inert polymer blocked further additions as a terminator of the chain. Moreover, the energy for the mitotic act was provided by the polymerization and was not dependent on usual sources as glycolysis or oxidation. Hence, the small free radical additions provided more energetic cell division. Dehydrogenated synthetic carcinogens initiate the integration of either pathogen with the mitotic mechanism's FCG activating double bond when during anoxia the free radical formed adds to one pole of this double bond. The free radical formed thus at the other pole adds to the critical ethylenic linkage of the pathogen producing a free radical at the other pole that continues the polymerization process that supplies the energy for uncontrolled mitosis. This is why the synthetic carcinogen is lost track of when the malignant change sets in.

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. In the respiratory allergies, the secreting mechanism and contractile mechanism energy producing and receiving FCG's and their activating double bonds are concerned and in the neurological allergies as epilepsy, compulsory neuroses and fixed ideas the conductile energy producing and receiving FCG systems of activating double bonds are attacked. We have classified cancer as an allergy of the cell's mitotic mechanism decades ago (Natural Immunity, 1934, Christopher Publ. Co. - Koch).

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 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 proximal 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 distal 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 sulfhydryl 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 distal pole of the FCG activating double bond, the free radical formed at the other pole can copolymerize with the sustaining pathogen as just stated whose energy liberated by polymerization forces the excessive uncontrolled mitosis or other functions 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 be it an allergic affair or a neoplasm.

The initiating toxin could be one of the sulfhydryl products of a certain bacteria trapped in occluded tonsilar crypts, the apical infection of teeth, or some occluded scarred sinus of long standing. Sulfhydryl readily forms free radicals on dehydrogenation by the FCG, and it also has the ability to add to double bonds of ethylenic linkages conjugated with carbonyl groups. It can thus interfere with oxidations in several ways for it can inactivate the quinone type co-enzymes as Co-enzyme Q-10, which is an electron carrier or transfer agent. 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 add to the FCG activating system to initiate the 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 envolved by biopsy proven cancer of the uterus, and 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. At the end of the twenty-fourth week the absorption of all growths was complete and an acute violent inflammation of the tonsil and lymphatics of the neck on the right side set in. She could neither swallow nor speak for about a week, when 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 so good afterward. During that attack she could not speak nor swallow and both symptomatologies were identical, except that the 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 depolymerization 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 cicitrization. Both inflammatory reactions to the monomeric form of the toxin were identical except that the recovery reaction induced by the corrective reagent burned the infection away completely with its toxins as adsorbed in the protective scar tissue. These were also burned away, so 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 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 clinical experience and the established facts of physiology and chemistry, and they are a guide to successful treatment, which after all was the goal of 50 years of investigation.

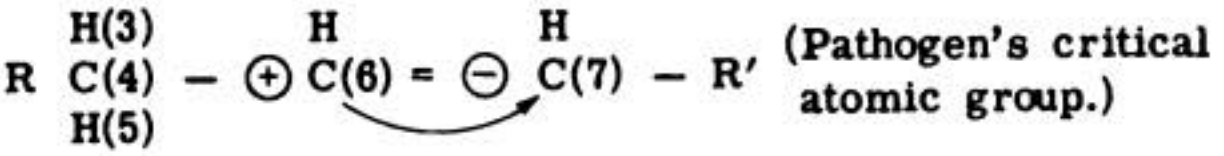
A little more discussion should be given to the easy rotation of the single covalent bond, and also its ability to be fixed in one plane by mutual polar attractions and repulsions of component atomic groups in both the host cell and the integrated pathogen. This rigidity exhibited by each species in 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 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 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 and 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 in more 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 critical atomic group from the pathogen which undergoes a stepwise oxidation. There are, however, more than one question that are 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 hinderance 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

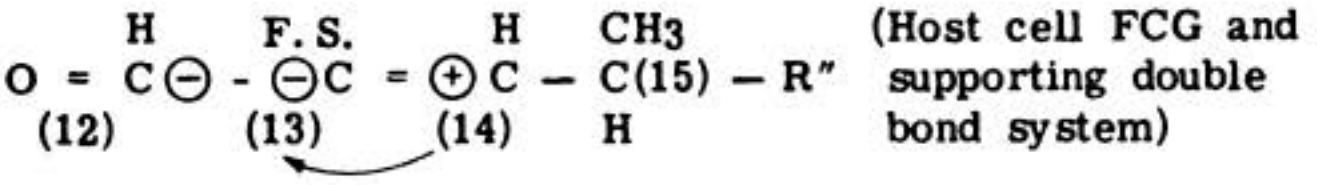
The pathogen may integrate with the host cell FCG by the condensation via its amine group, and block FCG function or pour polymerization energy into it to force an allergy or a neoplasia. This need not be diagramed as only one pathogen is required. Blocked functions as in diabetes or mental suspensions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



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.

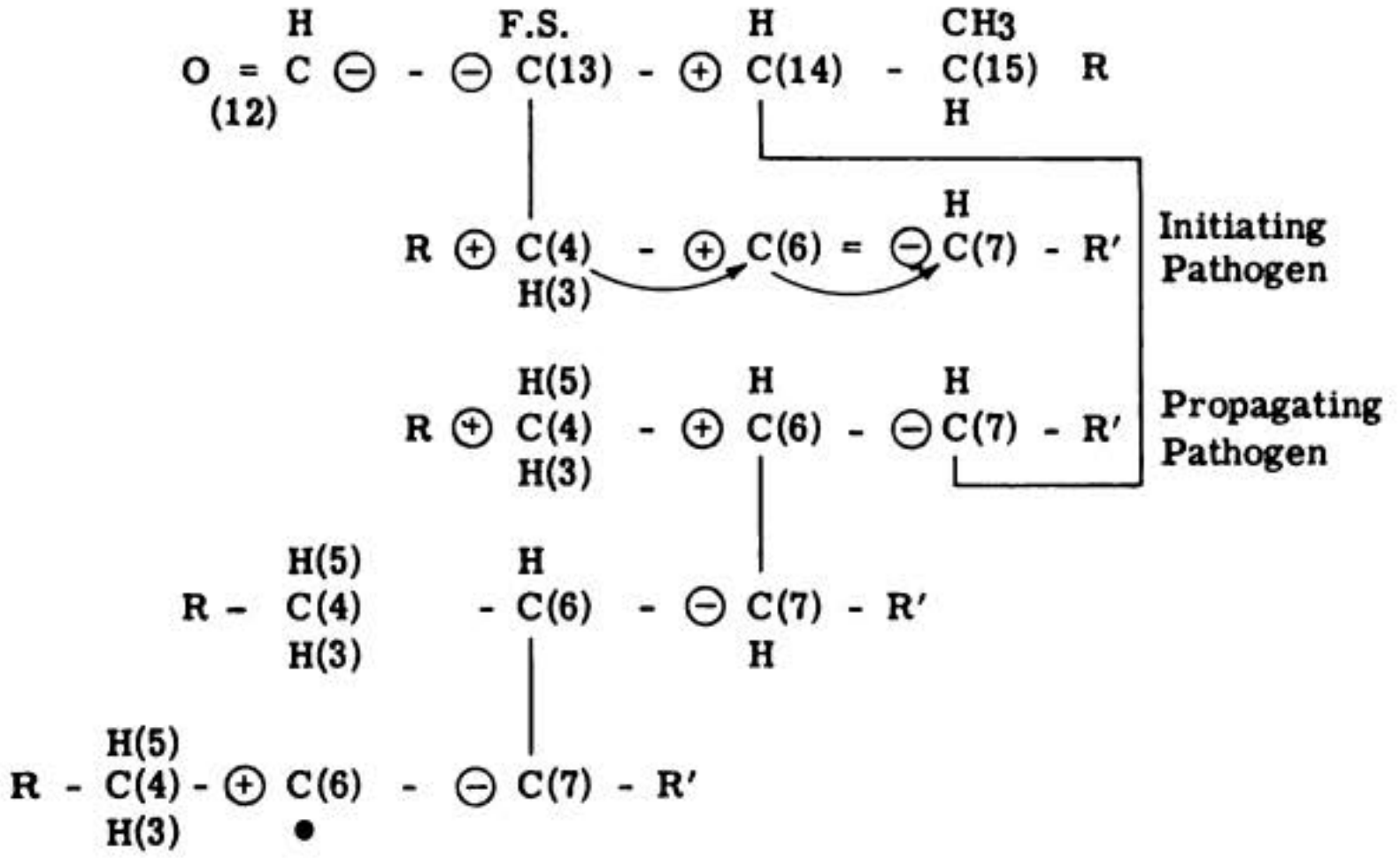
ESSENCE OF AUTONOMY



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)



The polymerization continues at C(6) free radical.

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 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 is bathed 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 the 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 polio case, the 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.

APPENDIX V

CORROBORATIVE SUPPORT PREDICTABILITY

One recalls that the conjugated double bond of a carbonyl group or of an ethylenic linkage activates many physiological processes pointed out in the text. These activations demonstrate its value as an electron donor, even in the high energy carrying phosphate esters and especially the enol-phosphate esters. Activation of the function of the carbonyl group in various capacities, as in the deaminations and decarboxylations co-enzymed by pyridoxal phosphate are demonstrated in their structural formulae. However, no attention has been given to this mesomeric function of the double bond. Our own thesis made use of this action long before such agencies were discovered. Thus one recalls that the mobile electrons of an ethylenic linkage activate both the dehydrogenating and condensation processes of the FCG's of energy production and energy acceptance. In the pathogen it activates the position alpha thereto and determines its ability to integrate with the host cell FCG's and with their activating double bonds. And after integration has taken place it invites the oxidative separation of the pathogen from the host cell. We saw that the various unions were accomplished by azomethine condensations and free radical additions as mentioned in the text. In Pyridoxal phosphate's coenzyme services as in some other recently discovered activities the azomethine condensation is now identifiable. All such activations depend on the ability of the ethylenic double bond to pass its mobile electrons on to the position alpha to it, of course as influenced by the substituents present in the molecule. The double bond is therefore identified by the fact that the condensations and cleavages take place at the point alpha to it and it holds a critical value physiologically, pathogenically and therapeutically. We can also demonstrate the presence of this double bond in the pathogen after integration has taken place, by causing cleavage of the double bond itself, when we make use of its identifying characteristics. The cleavage results in the liberation of the host cell FCG from the pathogen. This is demonstrated biologically by the cure of the disease.

The identifying characteristics are: (a) the greater rigidity of the double bond against steric rotation as compared to the greater ease of rotation of the single covalent bond in response to environmental influences; (b) the ability to add a radical or atom at one pole or at both poles in response to the number of atoms or radicals supplied; and (c) when an addition is made at one pole a free radical is formed at the other pole. In the presence of molecular oxygen, peroxide free radical formation and cleavage of the bond follows with separation of the pathogen from the host cell FCG system by forming two terminal carbonyl groups. The biological evidence is the rescue of the host cell with return of its normal functional status. The pathogen is no longer found.

The double bond is further identified since its characteristic rigidity against rotation provides that the distal pole will hold the same steric

relation to the plane of the double bond of the functional carbonyl group (FCG) in any particular disease or pathogenic integration. Whereas if the bond were single, easy rotation in response to various influences would place this pole in positions where attacking atoms or radicals could not always reach it. Therefore, the number of successful attacks would not be predictable because of steric hinderance. The number of successful attacks, on the other hand, is predictable where the bond is double because the steric advantage holds throughout any particular pathogenic category. In other words, where the double bond is concerned, if a successful attack is made once in any disease category, it can be expected to be successful in all other instances of the same disease, other things being equal.

Different pathogens would be judged to present the distal pole in the same steric relation to the FCG double bond, if a predictable response is had in each of a series of diseases caused by different pathogens. This fact has been realized. So we conclude that the least common denominator both in the pathogenesis and in its correction depends upon this ethylenic linkage of the pathogen, and *nature provides for the cure right in the pathogenesis.* It is easy to conclude on this basis that animal life was originally free from disease. Only one protective atomic value was needed. We have shown in the text what it is, the SSR. Now we will show by an alternate procedure what the curative process is and thus corroborate our postulate, and also the standard status of the pathogen's activating double bond.

For this demonstration we used the following procedure, which differs from the procedure used throughout the text. The latter, one will recall was the use of a highly activated DEHYDROGENATOR CARBONYL GROUP, the SSR, which brought about a free radical alpha to this double bond of activation of the pathogen after integration. The sequence was peroxide free radical production and cleavage alpha to the double bond. Now instead of an oxidizing agent, a HYDROGENATOR, a reducing agent of high activity is used to add hydrogen to the distal pole of this double bond by preference when given in sufficiently high dilution, and which adds to both poles when given in adequate or saturating concentration. For steric reasons Hydrogen is a better carrier of the free radical that makes the additions than any complex group of atoms.

When a dilution of one part to a billion or higher is given, only one pole, the distal pole, is reduced, and a free radical is formed at the other pole. This free radical goes through the regular sequence of adding molecular oxygen to become a peroxide free radical that causes cleavage of the double bond itself with production of two carbonyl groups, one belonging to the FCG and the other being terminal in the pathogen as an activator of further oxidation. Biologically the results are restored FCG function and cure.

Further as the double bond can add two atoms of hydrogen, one at each pole, and thus become resistant to oxidative attack when sufficient reducing reagent is given, the integration with the host cell is made permanent,

and the disease remains. In serious viral diseases in animals as hog cholera, fatality resulted in 100% of cases treated with concentrations of one part to a million. The same was true in aftosa, dog distemper, some bacterial infections and after poisoning with a tetanus toxin. In animals and man with the high dilution of one part to a trillion and higher, rapid corrections were had in all diseases treated, predictably. These include anterior poliomyelitis, malaria, cancer, the acute crises of coronary occlusion with infarction, chicken pox and in some hereditary degenerations of serious degree.

Thus by steric and bipole evidences, the double bond of activation of the pathogen is demonstrated after integration has taken place, and its part played in both the pathogenesis and in the restoration of normalcy are also seen.

It is helpful to contrast this action with that of the SSR of the text, since the latter is a dehydrogenator and directly an oxidizing agent, while the action of the reducing agent is indirectly an oxidation, but only when used in very high dilution. The SSR shows corrective effects in every dilution from one to a million up to one to a trillion and higher. Here only one action can take place and that is the removal of a hydrogen atom alpha to the activating double bond. This dehydrogenation is followed by the usual sequence of peroxide free radical formation and cleavage alpha to the double bond, and liberation of the host cell. Thus only a curative action can be had. When the concentration is needlessly high, as one part per million, the recovery reactions are uselessly too uncomfortably vigorous, without anything being gained over the recovery had by a reasonably dilute dose. The recovery gained by the reducing agent requires an appreciation of the basic chemistry involved. When correctly conducted it is just the same as when the oxidizing agent, the SSR, is used. A few cases will show this. They will be greatly condensed to give the main facts.

ANTERIOR POLIOMYELITIS PARALYTIC

Miss N. L., trained nurse, age 33, took ill August 16, 1947 with the prodromal symptoms, headache, nausea, terrible leg and back pains and leg stiffness. On August 19, at 4 p.m., both legs from hips to toes were paralysed, could not support her, and the pains were worse. I saw her at 11:30 that night. There were no tendon reflexes in the legs, no knee jerk. When a pencil point was jabbed forcefully into the soles of her feet, there was no effort at withdrawal, and no muscle contractions could be felt. This is the best test of all. The fever was only 102.5°F. The spine was getting stiffer and the neck also, so the disease was spreading. The injection of 2 cc. of the one to a trillion dilution of the Reducing Agent was given in the right triceps muscle. Results: On the next morning at 8, the mother phoned that the girl could move her legs, slept some and ate a little. At midnight I saw her again. She could walk, felt pretty well, and was hungry. In two more days she was ready to work. All reflexes were normal.

MALARIA, CHRONIC MALIGNANT TYPE

Mr. L.M., age 36, gas station attendant, World War II veteran, contracted malaria in the Pacific area, treated in Veteran's hospitals for many months, but without results. Attacks still came. After one on June 13, 1947 he was given an injection of 2 cc. of the one to a trillion solution of the Reducing agent. Result: No more attacks in the two years that followed under our observation. His health returned very nicely. The history states this was a falciform infection.

CANCER

Mrs. F., age 58. The history claimed full diagnosis of cancer at the Henry Ford Hospital, with hopeless prognosis. Examination showed a weak cachectic woman with an abdomen greatly enlarged with hard irregular tumification and a pelvis so envolved the landwarks were completely obliterated. Hemorrhage and drainage from vagina and rectum and the enormous envolvement, with the great anemia, indicated she could not survive many days. We did not wait for the biopsy report as we considered the situation hopeless and made a clinical diagnosis only. The Reducing agent was given 2 cc. of the one to a trillion dilution on August 17, 1947. In a month's time drainage, pain, bleeding and functions had improved. She gradually recovered according to reports. She moved to California. The last report came in August 1961, stating that she fully regained her health and is still well.

CORONARY OCCLUSION

Mrs. S., age 74, with a long history of arterial sclerosis and aortic insufficiency, usually carrying a blood pressure of 200/100, had a severe coronary attack in June 1960. The SSR was given before true infarction could happen, and the recovery was immediate. The ECG showed no infarction. The following year on June 27, 1961 she had a very severe attack. She was hospitalized and given every possible aid while under the oxygen tent, but without favorable response. The situation rapidly deteriorated, B.P. 190/100, great pulmonary edema, thin weak pulse at 130 P/M, great dyspnoea, general cyanosis, chest pain, and she was at the point of collapse when the reagent was injected in the triceps muscle, 2 cc. of the one to a trillion dilution. The response was immediate. The house physician took the pulse while the injection was being given to check the change that followed. The needle was a No. 12 bore, one inch long, to accommodate the rapid injection. Just after the needle was withdrawn, a part of a minute after the injection was made, the pulse immediately changed to 60 per minute, the dyspnoea ceased, the cyanosis faded away, and as fast as the blood pressure could be taken it was found to be 140/80. She was comfortable in a minute. The house physician was "shocked". The next morning the ECG was taken, and showed nodal fibrilation, extensive infarction of the

Septum, and ischemia extending over the lateral wall of the left ventricle. Another ECG taken a week later showed marked improvement with diminution of the area of infarction. The day following the crisis the Blood Pressure was back to its normal for her, 180/100. Her health remains what is considered good for her.

During the first attack the blood was extensively jelled so that the ischemia of the heart muscle gave the same symptoms as a thrombosis since the blood was not flowing. But on receiving the SSR the blood colloids were quickly charged and good dispersion restored the fluidity for normal movement. Infarction was prevented. During the second attack the jelling of the colloids went on long enough to injure the vessel intimae and true clotting took place over a wide area, and besides a still wider area was ischemic from extensive blood jelling. Wherever the true clot had not interrupted the circulation, the dispersion was restored and a good flow assured. The perifery of the infarcted area showed so much improvement within a week that one could conclude that much integrated toxin was really removed. The speed of the restoration to normal in both instances was the same and in each instance depended on the separation of the pathogen from the FCG of all of the cells concerned. She was attended by Dr. Jayme Treiger in both attacks.

CHICKEN POX

Dr. Treiger reports on 14 cases of Chicken Pox treated during an epidemic in 1961. Twelve cases gave immediate responses so that the lesions did not progress any further after treatment with the reducing agent, but started to involute right away. In from 24 to 48 hours the recoveries were complete. The other two cases recovered in from 5 days to a week. Thus the virus was instantaneously removed from the tissue cell FCG-s, in the first 12 cases. The results in the two cases that failed to respond to the reagent were also predictable since they did not follow the regime and ate such antagonistic foods as smoked ham. As in measles where the recoveries require about 12 hours after the patient has been treated, the response is predictable and the scientific basis of the therapy is thereby established.

HEREDITARY GENERAL ATROPHY-IDIOCY

A girl of 11 years of age with the intelligence of a child of one year, could walk before she was three years old, but not afterward. Her arms, legs and body were atrophied, but not her head although her brain failed to develop. She had convulsions every day of her life until July 1, 1961 when the Reducing Agent was given. On that day she had convulsions and was unconscious all day until the remedy was injected by Dr. J. Treiger. After the injection the convulsions stopped and she had no more until during the sixth week reaction when one mild convulsion took place. There has been a steady improvement in her health besides during the 2 months that followed the treatment up to this date (9/1/61).

DIABETES WITH GANGRENE

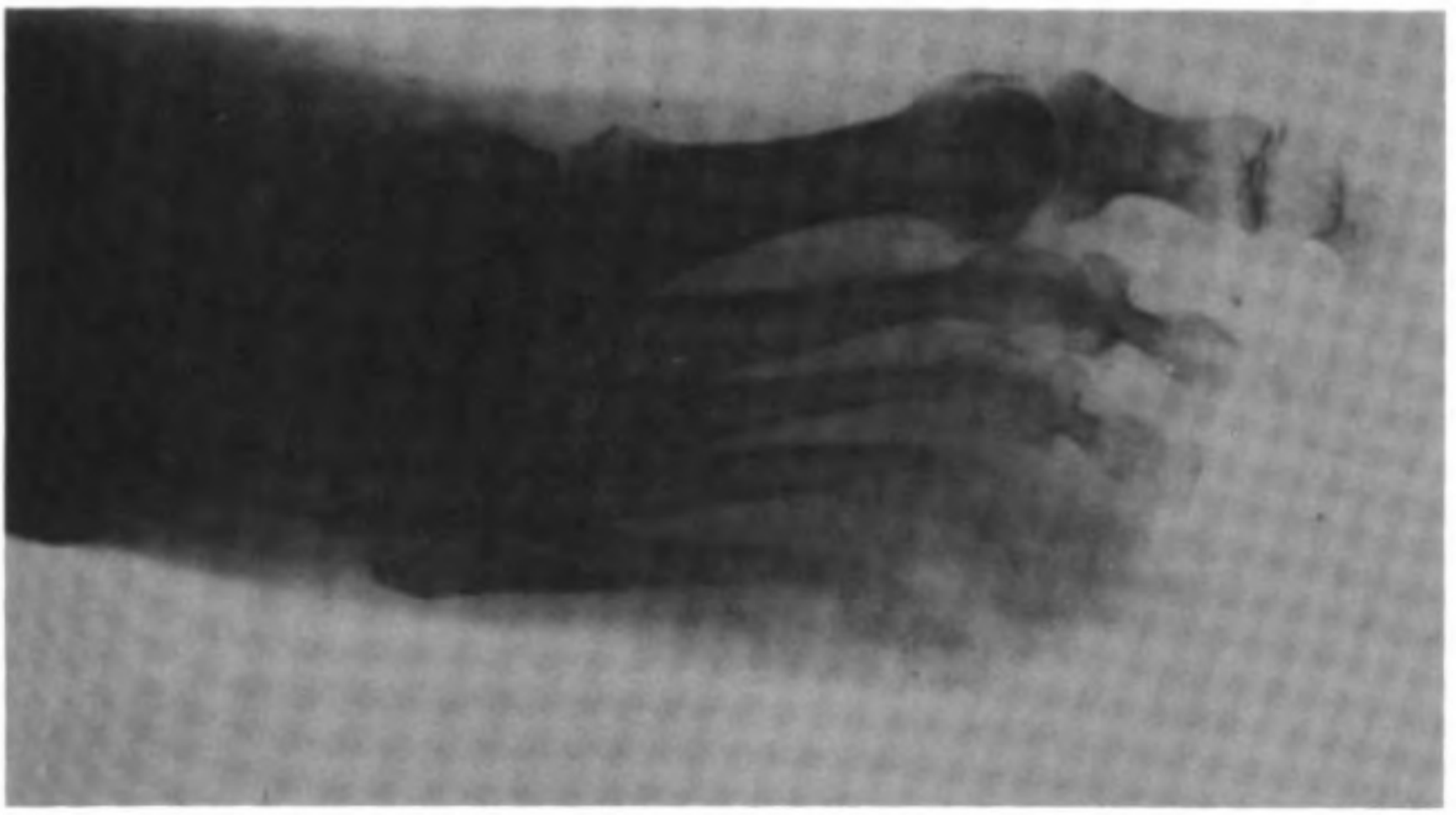
Dr. Julian Baldor reports the following case of diabetes with gangrene. Mr. A. C., age 71, had been diabetic for five years. He had had many high blood sugar analyses and insulin injections, but a steady decline in health. On January 5, 1961, he had a severe crisis; blood sugar 375 mgm.%, high fever, much pain in the right foot and was approaching coma. Gangrene of the fifth toe set in. Following toe amputation on February 12, 1961, fever continued and there was a rapid spread of the gangrene to envolve the entire foot with numerous fictulae on the dorsum and plantar surface. Further amputation was considered, but because of the virulence of the gangrene, it was considered useless, even though done above the knee.

The Reagent was given on March 18, 1961. Two days later, on March 20, 1961, Radiograph I of the right foot was taken showing bone destruction from diabetic gangrene. After treatment, improvement followed. On March 30, 1961, without insulin his blood sugar was 124 mgm.%. No medication was required. In eight weeks the lesions were healed, the edema was gone and healing of the destroyed bone tissue in exact normal detail was observed in the radiographs. Radiograph II taken on June 3, 1961, shows bone reconstruction where the gangrene had formerly destroyed the boney structures. Only where amputation removed the bone was no reconstruction possible. His foot became normal, all fistule were healed, function was good and he is able to walk normally. On August 25, 1961, his blood sugar was 80 mgm.% and his health remains good. Here is a good demonstration of the reversal of the pathogenesis.

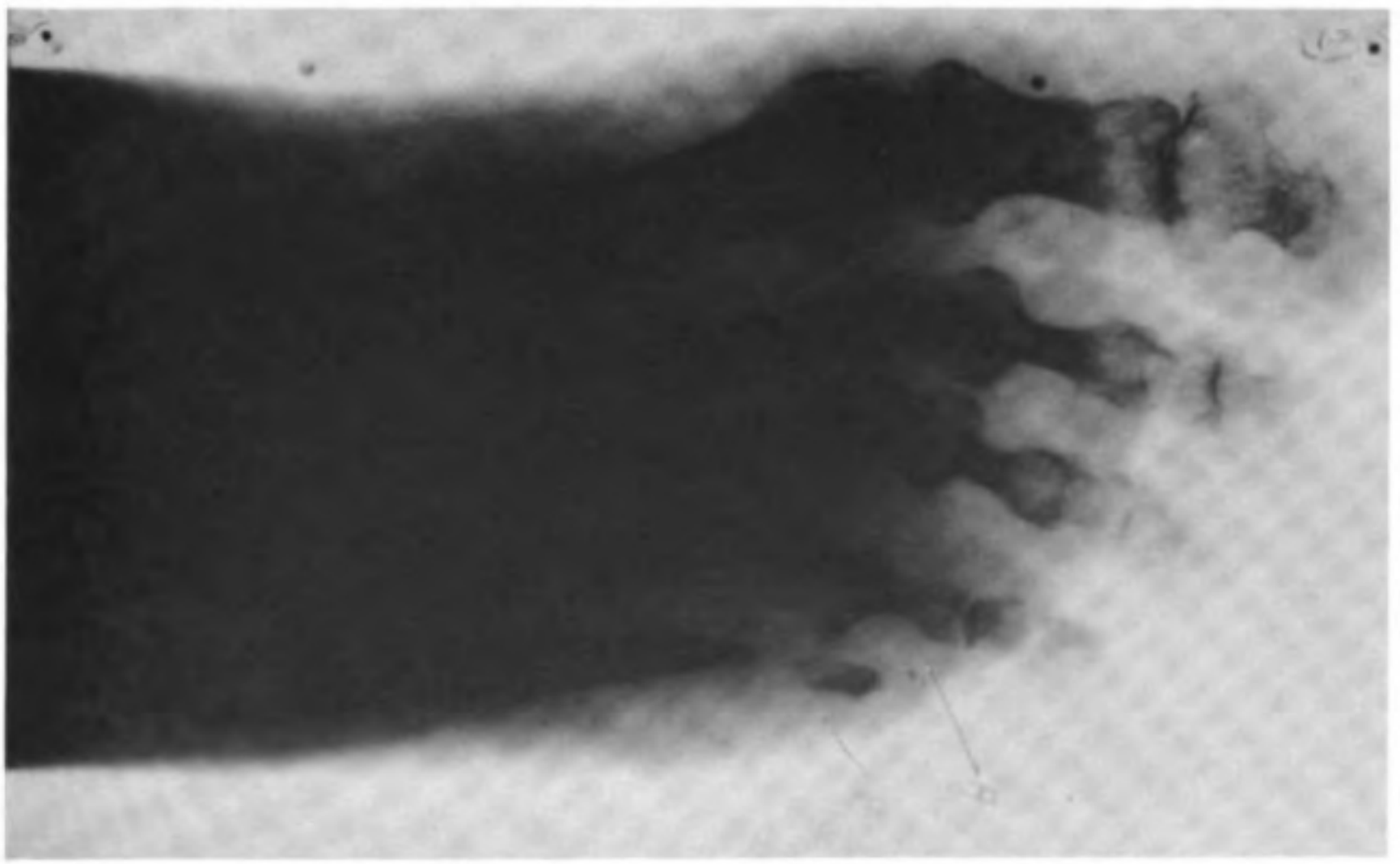
We have fully reviewed the established theoretical chemistry basic to our postulate, and have seen how it is exemplified in Nature's biochemical systems. We have seen how the double bond is postulated to serve in the production and correction of ALLERGY, AND ESTABLISHES A LEAST COMMON DENOMINATOR IN DISEASE CONDITIONS.

The reversal of the pathogenesis has been observed many times in the reconstruction of bone that was destroyed by cancer. In this process soft bone is formed first and after its shape and dimensions conform to the normal with the fragments drawn to their normal positions, the soft bone calcifies and is hardened so it can function normally. The original normal structure is thus restored as seen in the radiographs. Soft tissues as the vocal cords, the uterus and the stomach wall have also been observed to be reconstructed to good functioning-normal Thus the pathogenesis was reversed in its totality. So it is not unreasonable to suppose that if the FCG of the tissue cells and of the reticuloendothelial system possesses an O/R potential equal to or superior than that of the SSR, disease changes as we know them today could never be initiated nor propogated. Was man so equipped when the Creator pronounced him perfect in the beginning? And what has unnaturally produced food and other features of civilization to do with our present status?

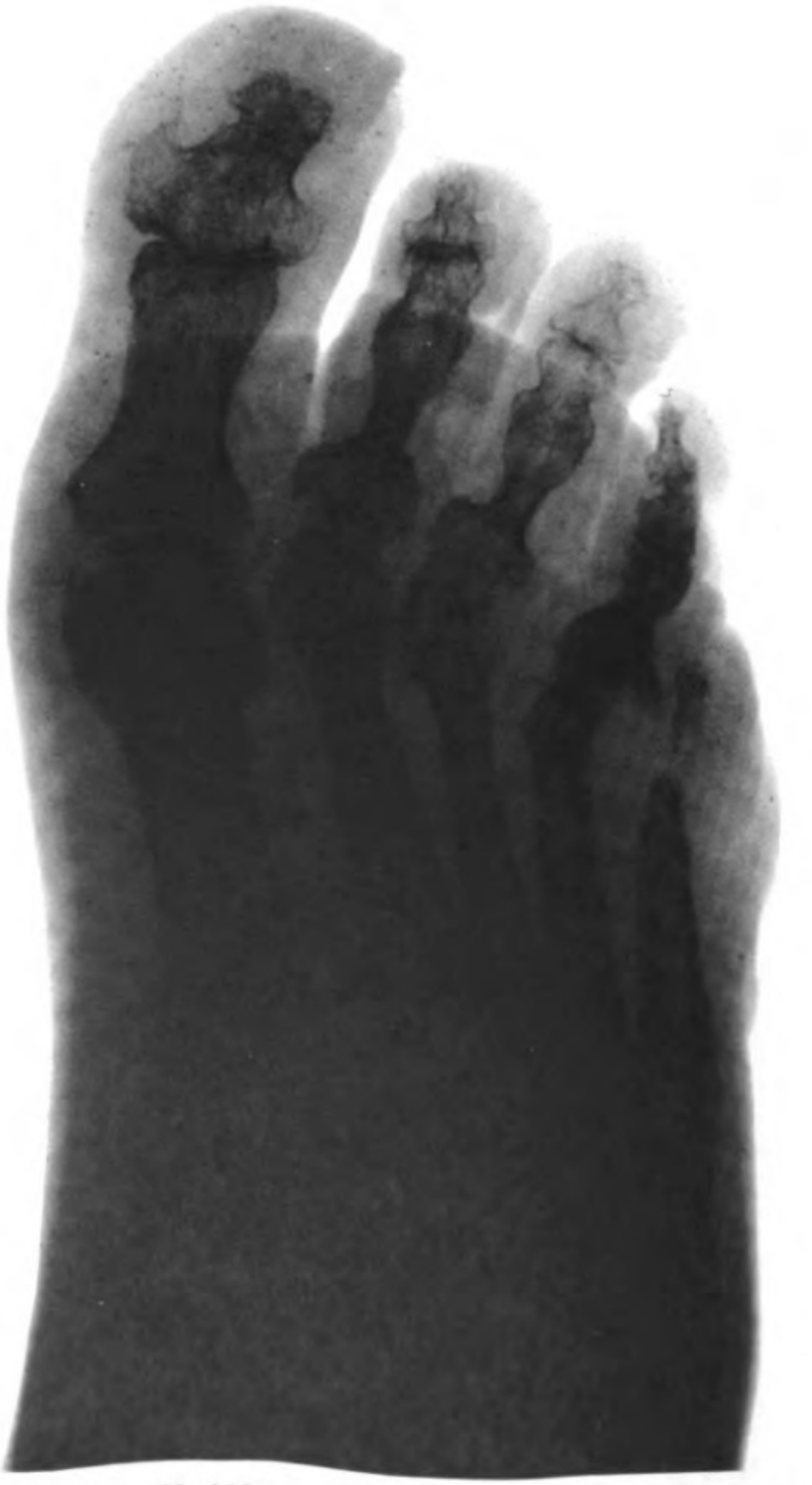
VISIBLE DEMONSTRATION OF REVERSAL OF THE PATHOGENESIS



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 boney structures.



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

PRACTICAL APPLICATION OF THE FORMER CHAPTERS AND OTHER ESSENTIAL INFORMATION

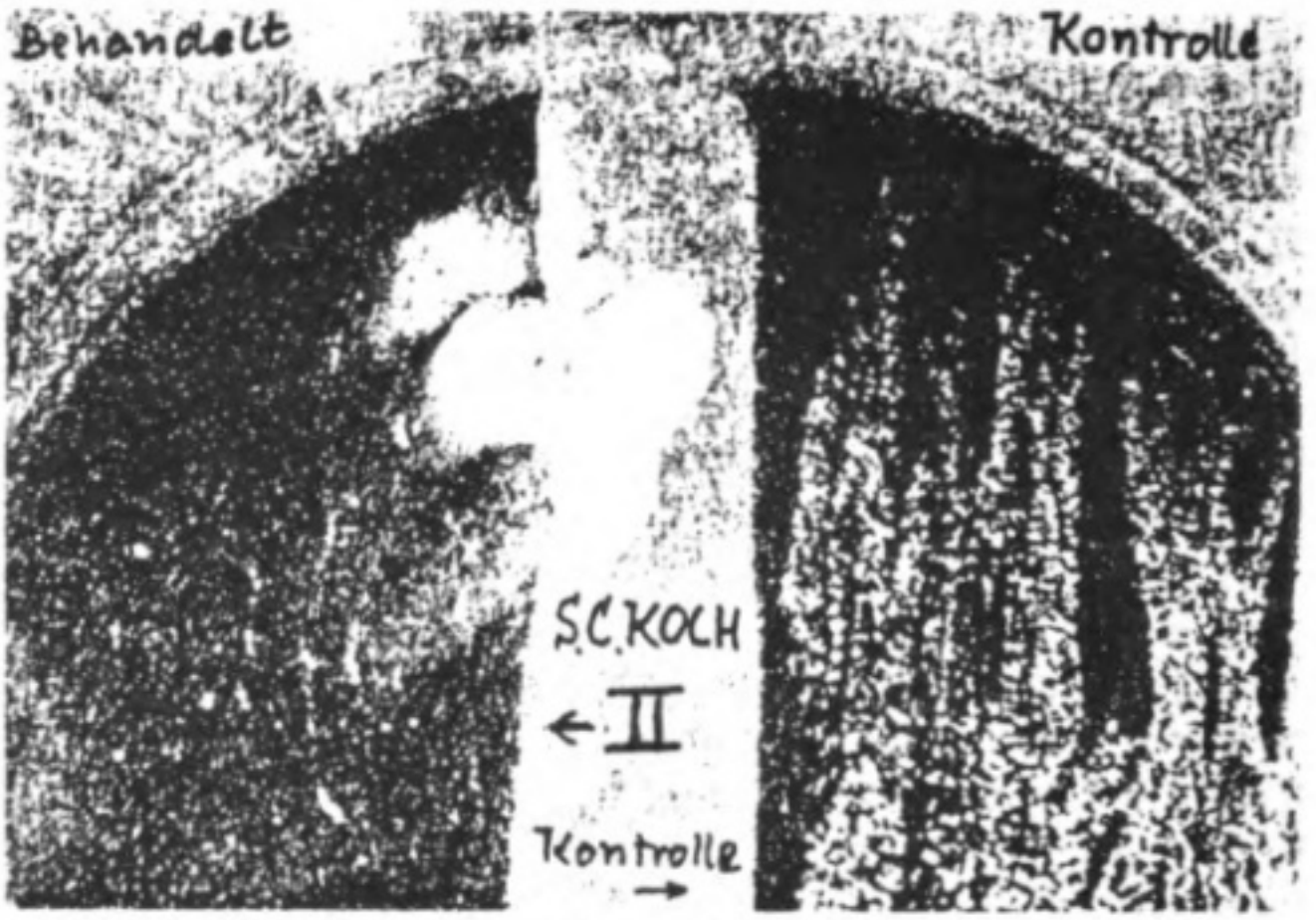
One has seen that the reversal of pathogenesis of micro-organisms follows much the same pattern as the reversal of neoplasia in tissue cells. Virulence is proportional to pathogenicity in both; and their parasitism is a necessary property depending on the degree of deficiency against which survival is attempted. It is also an economic provision that normal tissue survival arises in the successful performance of the specific function of any form of life in its contribution to the Great Biological Economy. Under this circumstance, parasitism, pathogenicity and their virulences are utterly impossible, as the pattern of activity is determined by functional procedure; and restoration of function eliminates all three.

One has also seen that normal function depends on normal structure, the key to which is the unhindered carbonyl group in the presence of molecular oxygen. Energy production in any form of aerobic life can then proceed by dehydrogenation, free radical production, peroxide free radical production and the rest of the oxidative progression.

It is axiomatic therefore that where any one of the three features, virulence, pathogenicity or parasitism is eliminated by this basic corrective provision, all three are eliminated as well. Clinical demonstrations were given in the testimony of many physicians and veterinarians in the 1942 and 1946 Federal Court trials, the veterinarian department of the University of British Columbia of Canada, and by the Ministry of Agriculture of that province. Their testimony showed that highly virulent hemolytic staphylococci lost their toxicity and hemolytic properties together with the restoration of the normal calcium balance and the restorative healing of the parenchyma of seriously infected mamary gland structures instead of by scar tissue. At the same time a rapid return of health followed after the therapy of the text was given, and most important of all, the rate of recuperation was proportional to the rate of increase in the germs that had formerly produced the disease (see page 74 and case 18 of the text for further examples). One concludes that the normal function of the bacteria of cleaning out dead tissue debris had been restored by the action of the activated carbonyl group of the text. The same effects were observed in the healing of huge tubercular cavities, the healing of fulminating and chronic terminal osteomyelitis and other infections. Toxicity tests of before and after treatment, of gangrenous mastitis in dairy cows, showed that filtrates from the bacterial cultures taken from untreated animals were fatally toxic, while those taken from treated animal lesions were non-toxic.

To test the above principles Dr. Dieter Reinstorff ran various bacterial cultures of which the photographic plate pictures an example. It served as a test for virulence. The innoculum was a highly virulent hemolytic streptococcus taken from a child. At two weeks after the innoculation of the test and the control cultures, the latter showed rich growth while the test plate showed no growth at all. The test plate was treated with 2 milliliters of the 10^{-12} concentration of the reagent. This test for virulency is in accord with

REVERSAL OF VIRULENCE OF HEMOLYTIC STREPTOCOCCUS BY CONTACT WITH GLYOXYLIDE SOLUTION, 10^{-12} CONCENTRATION



Treated culture at left, Control culture at right after two weeks incubation.

the loss of pathogenicity after treatment as reported above.

It is also evident that the data show that the pathogenicity was not removed by a destructive reagent, but rather by the corrective function of the activated carbonyl group. There was no longer any necessity for parasitism to win survival and no forced effort at reproduction was necessary to try to overcome a deficiency, for the correction was already made by supplying the normal source of energy for its created function, and hence pathogenicity could not result. Similar patterns of recuperation are observed clinically.

The delay in the occurrence of the first reaction to treatment, be it 24 hours, 36, 72 or more hours, or even days and weeks or months (as in the case on page 278) after the remedy is injected, presents the characteristics of the induction period to a chain reaction, a free radical affair, and hence shows that fundamentally the result of the activated carbonyl to be not an equa-molecular affair, but an inductive affair. It resembles the refractory period following a toxic drug when subfatal, but when the tissues fail to respond to a second or third dose all of which together are more than fatal in amount if given at the start at once. The culture plate is also an example, for the adaptation of the parasite to the medium took two weeks to reach full bloom while the test plate showed no growth. So the parasitic survival effort is likewise a chain affair, another instance of the universality of free radical phenomena in life processes. One should therefore observe the response to the treatment with the carbonyl groups of the text with careful attention before deciding that a dose is inactive when visible changes of recovery do not show up immediately.

MAGNETIC ASPECTS OF CARCINOGENESIS AND ITS REVERSAL

Ever since 1920 treated cancer patients in transit from Western parts of the country to Detroit reported that they felt much better after passing East of Ypsilanti, Michigan. The reports were too numerous to ignore, so we investigated the district between Detroit, Ypsilanti, Wayne and Redford. After eliminating alpha, beta, gamma, and cosmic rays, we settled on the magnetic flux of the district as responsible. The dip-needle did not vary as much as 20 degrees from the horizontal and the flux was stronger than that of unfavorable places.

We found many residents of the district who made rapid easy recoveries from widespread neoplastic invasions of the vital organs, and the reversals were permanent and complete. So the recovery process was favored as well as the feeling of well being of the patients in transit. The soil was a rich sandy black shiny loam with rich vegetation and many earth worms and grubs, so we figured that the magnetic flux favored soil building by the flora and fauna of that function. Centries of fertilization by animal excretia and decaying organic matter, that supplied iron-porphyrin compounds from bile and blood and flesh, with paramagnetic oxygen profited by the magnetic flux to accelerate its soil building reactions. Foods raised here were therefore the very best.

The cancer incidence was just as high and the neoplasms just as malignant as one found anywhere else. But the recoveries from cancer by the therapy of this text were exceptionally rapid and easy. Both carcinogenesis and its reversal are free radical affairs. The former is anoxic while the reversal stages paramagnetic oxygen and peroxide free radicals as its principle actors. So, since oxygen is 4,000 times more magnetically susceptible, like iron with which it coordinates, than most other elements in the tissues, it concentrates with other paramagnetic materials as calcium and iron in the activated spots of the mitochondria to boost the reaction rates of function and detoxicative oxidations.

Confirmatory are the boosting effects of magnetic storms that occur with the sun's rotation every 27 days, on both the rates of neoplastic activity and the reversals to normal. Here again the recoveries are boosted far beyond the pathogenic rates, and no doubt for the same reasons, for in addition to the paramagnetic oxygen and trace elements the mitochondria hold the activated carbonyls and quinones and the necessary calcium in their activated

spots. Our postulated claims on this matter were opposed decades ago, but are now being confirmed by modern microbiologists even including the blocking effects of guanidin. We hold that the electron mechanics of the double bond as of carbonyl and its activating ethylenic linkage are also magnetitc susceptible whereby their dehydrogenations are also hastened. Antagonistic are mercuric salts, amines, and sulphides, against which cyanide protects, though antagonizing the cytochrome system, and still tending to prevent neoplastic change. This indicates that the primary pathology of cancer follows proton withdrawal and blocks electron transport for it depends on hypoxia. The curative progression depends on vigorous proton withdrawal in the presence of oxygen and is aided by the magnetic activation of free radicals.

SIGNIFICANCE OF THE ETHYLENE BRIDGE

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 electrons are mobilized toward the carbonyl group, and such substituents as CH_3, CH_2CH_3, $CH(CH_3)_2$ and $C(CH_3)_3$ 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 electronegativity 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 electronegativity 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 alters the activity. When the carbonyl group is free, however, it attracts the electrons away from the hydroxyl groups so that the hydrogen atoms tend to be liberated with ease as protons. This mobilization is seen when glucose or fructose are dissolved in heavy water. Here it is found that the hydrogen atoms trade places freely 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 for a covalent bond; 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. The same applies to the C-H linkage

Pathogens, like unsaturated fats, also invite dehydrogenations in various degrees. Here we postulate that a methylenic group positioned alpha to a double bond of an ethylenic linkage offers two activated hydrogen atoms; one is important for the integration with the FCG system during the anoxia and the other invites removal from the integrated pathogen by the carbonyl group of the curative reagent. (This dehydrogenation can also be accomplished by an appropriate free radical). The activation of the pathogen's hydrogen atom 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 sulphydryl as well as imide 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-hydrogen or oxygen-hydrogen bond weakens it to facilitate dehydrogenation. The stage is thus set intrinsically in the pathogenesis for its oxidative reversal. The pathology actually provides for its correction. The philosophic implications deserve thought.

The basic pathology in endocrine, viral and neoplastic disease is, therefore, of the same pattern and depends upon the electron passing powers of an ethylenic linkage to activate the position alpha to it. The carbonyl group is an electron accumulator and the alpha methylene group an electron donor.

THE IMPORTANCE OF THE FREE RADICAL

Recently Prof. Cheves Walling of Columbia University stated: "Twenty-five years ago free radical chemistry interested only a few gas-phase kineticists . . . It is remarkable that one set of simple principles is basic to such a diverse group of processes and products." As our investigations show, the service of free radical products and processes made it possible to understand and reverse the pathogenesis of cancer and liberate the host cell from fatal viral integration decades earlier than 25 years ago, and in fact, concomitant with the development of the plastic industry. Thus the free radical occupies an important field in biological processes also.

It made possible an explanation of the Pasteur Effect, and its reverse, the Crabtree Effect, which in our opinion is a means of measuring the contents of respiring elements that conduct cell functions. Crabtree (1929) showed that when glucose was added to cancer slices, the use of oxygen was inhibited in favor of glycolysis, which was not the case with normal tissues as liver and kidney cortex. Here addition of glucose stimulated respiration. It was also shown that such metabolically inert tissues as cartilage and kidney medulla exhibited the Crabtree Effect much like cancer tissue. Natal retina did also, until it matured and could function as an oxidation mechanism. Thus, where and to what extent that respiration is possible, addition of glucose will stimulate

oxygen use, and where this function is limited, glycolysis is used to dispose of the added glucose, and the use of oxygen is depressed to the extent that common factors engage in both respiration and glycolysis. Thus cancer tissue has a limited oxidation rate or capacity which it cannot increase under stimulation, and is using to the limit all the time, thus revealing the inability to supply further oxidative facilities. What is the cause of this deficiency?

Aisenberg (1961) offered the two explanations that are supported by data given in this text, (a) the diminished amount of mitochondria in tumor cells, as Warburg also suggested; and (b) the statement forwarded by Chance and Hess (1959) that the respiratory elements are still present in normal amount, but are under a restraining influence which blocks the oxidation ability proportionately. This is exactly what our text has demonstrated together wtih an explanation of both the hindering mechanism's chemistry and that of the liberation of the oxidation mechanism. Thus our thesis is supported again.

Not only the FCG's of the various specific functions may be concerned, but also the carbonyl groups of the accessory factors to the oxidation process, the electron or hydrogen acceptors as the quinones so recently discovered to exist in all living cells. Their structures are essentially carbonyl groups activated by conjugation with double bonds of ethylenic linkages. Similarly we find activated carbonyl groups in the keto-steroids, and the spent products of all such structures are hydrogenated, reduced to hydroxyl. In the case of the keto-steroids the double bonds are also inactivated, and so the molecule can not be reversed to function again, but in the case of the quinones the double bonds are not altered and reversal with return of function is possible. Thus coenzyme Q can function as a co-enzyme over and over again as an electron transfer agent.

The quinone structure is also admirably adapted to such function so as to meet the requirements of specificity in oxidation-reduction potential and for selecting the specific materials it will react with in each particular cell activity. The substituents placed about the quinone's double bonds give the steric advantages and hindrances required for specific reactions and for elevated or depressed negativity and oxidation-reduction potentials of the carbonyl groups. Twenty years ago the most celebrated biochemists testified that the quinone structure had no place in biological processes in opposition to our thesis. Today, we know otherwise and the position of the free radical which can now be proven by electron spin resonance techniques is demonstrated as fundamental to all living processes. The present writer's Professor of Chemistry was the discoverer of the Free Radical, Moses Gomberg He developed his thesis as a pure piece of philosophy, but, he lived long enough to see it become the basis of the plastic industry, and even learned of the first adventures of the writer in the interpretation and correction of pathologic states. We join Prof. Walling in saluting the glorious work of Gomberg.

In our early work we found that polymerizing unsaturated free radicals of low molecular weight stimulated cancer development decidedly, whereas large

inert polymers blocked cancer growth and involution soon followed. Our conclusion was that the carcinogen integrated with the host cell nucleus by a copolymerization process of free radical double bond additions, whether the carcinogen be a polymerizing bacterial toxin or a vegetating provirus whose units were largely host cell nuclear products and this symbiosis held through the mitotic act. The small polymers offered more double bonds and free radicals for hastening the mitosis point, whereas the large inert polymer blocked further additions as a terminator of the chain. Moreover, the energy for the mitotic act was provided by the polymerization and was not dependent on usual sources as glycolysis or oxidation. Hence, the small free radical additions provided more energetic cell division. Dehydrogenated synthetic carcinogens initiate the integration of either pathogen with the mitotic mechanism's FCG activating double bond when during anoxia the free radical formed adds to one pole of this double bond. The free radical formed thus at the other pole adds to the critical ethylenic linkage of the pathogen producing a free radical at the other pole that continues to polymerization process that supplies the energy for uncontrolled mitosis . This is why the synthetic carcinogen is lost track of when the malignant change sets in. Such is our explanation.

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 principal 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. In the respiratory allergies, the secreting mechanism and contractile mechanism energy producing and receiving FCG's and their activating double bonds are concerned and in the neurological allergies as epilepsy, compulsory neuroses and fixed ideas the conductile energy producing and receiving FCG systems of activating double bonds are attacked. We have classified cancer as an allergy of the cell's mitotic mechanism decades ago (Natural Immunity, 1934, Christopher Publ. Co. - Koch).

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 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 sensitiz-

ing 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 distal pole of the FCG activating double bond, the free radical formed at the other pole can copolymerize with the sustaining pathogen as just stated whose energy liberated by polymerization forces the excessive uncontrolled mitosis or other functions 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 be it an allergic affair or a neoplasm. Also the smaller the molecule, the more the energy, shown by the free radical wandering in its domain, to force the polymerizations.

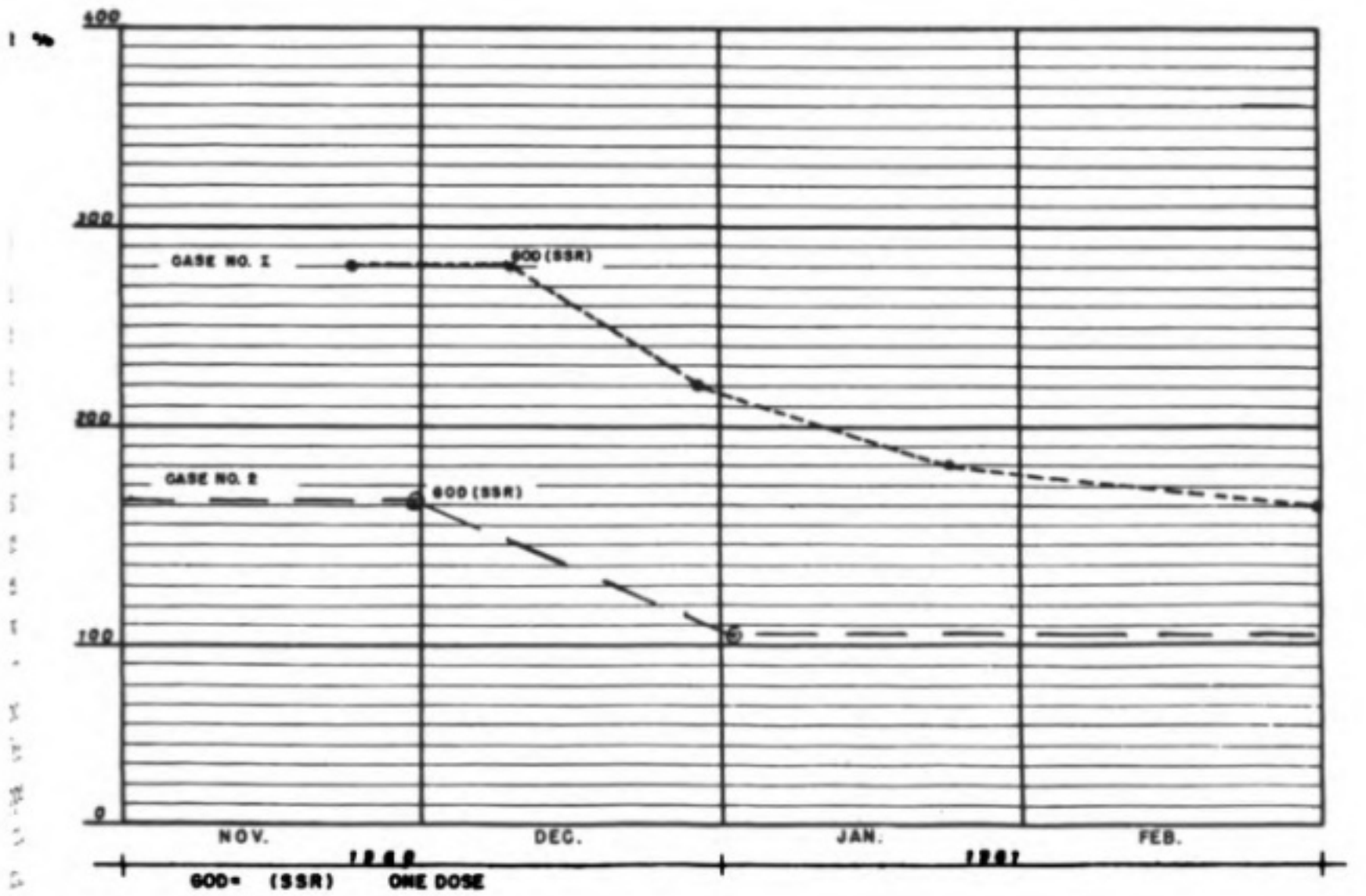
The initiating toxin could be one of the sulfhydryl products of certain bacteria trapped in occluded tonsilar crypts, the apical infection of teeth, or some occluded scarred sinus of long standing. Sulphydryl readily forms free radicals on dehydrogenation by the FCG, and it also has the ability to add to double bonds of ethylenic linkages conjugated with carbonyl groups. It can thus interfere with oxidations in several ways for it can inactivate the quinone type co-enzymes as Co-enzyme Q-10, which is an electron carrier or transfer agent. 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 add to the FCG activating system to initiate the pathogenesis.

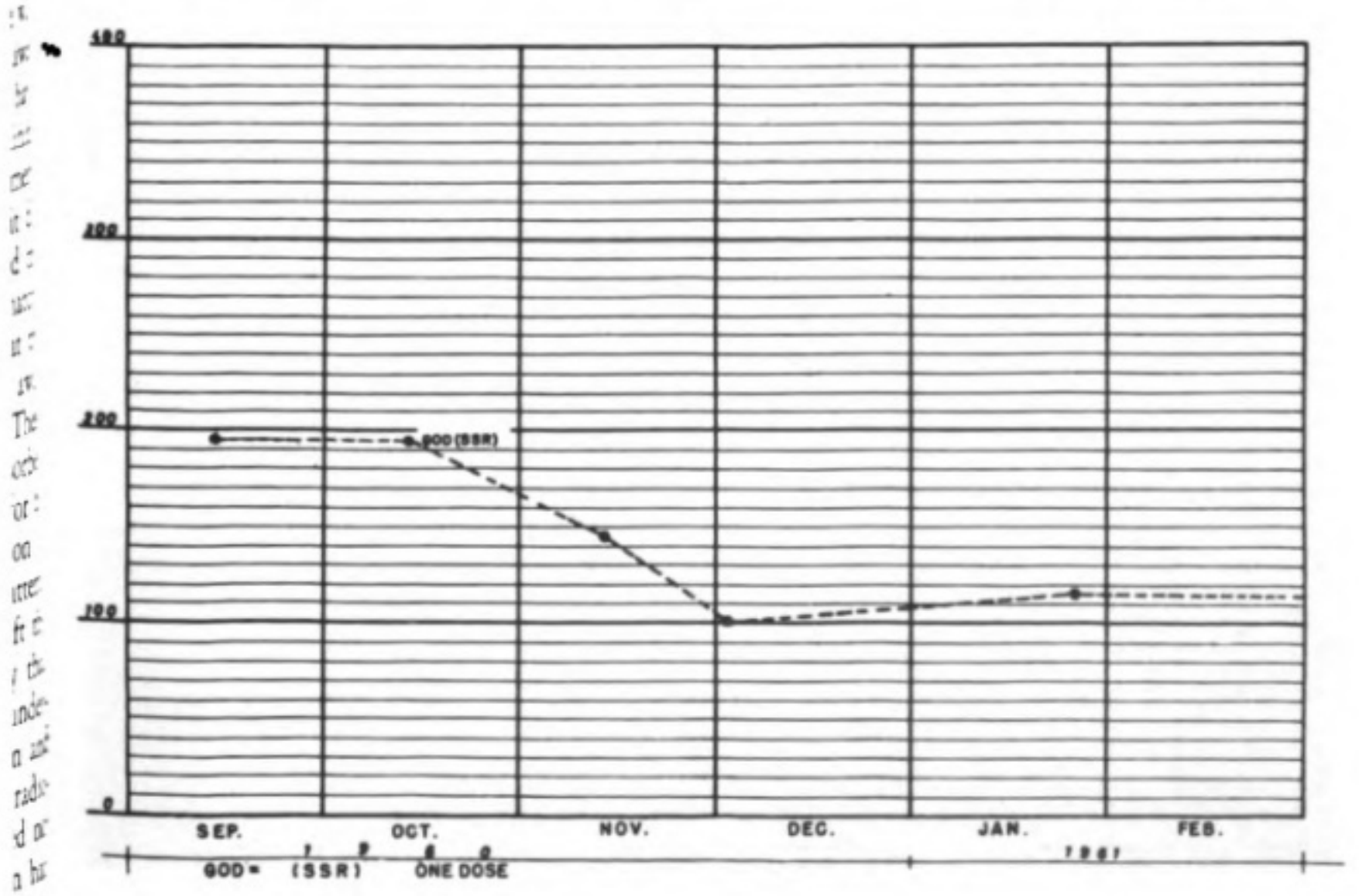
To show that the focal infection of long standing is a factor in carcinogenesis a typical case history will suffice. This woman was then 56 years of age and her case history was included in the testimony before the Federal Trade Commission in 1943, as a demonstration of the nature of the recovery process after the Koch reagent was given. The uterus and most of the pelvis and lower abdomen were envolved by biopsy proven cancer of the uterus, and 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. At the end of the twenty-fourth week the absorption of all growths was complete and an acute violent inflammation of the tonsil and lymphatics of the neck on the right side set in. She could neither swallow nor speak for about a week, when 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 so good afterward. During that attack she could not speak nor swallow and both symptomatologies were identical, except that the 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 depolymerization 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 cicitrization. Both inflammatory reactions to the monomeric form of the toxin were identical except that the recovery reaction induced by the corrective reagent burned the infection away completely with its toxins as adsorbed in the protective scar tissue. These were also burned away, so 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 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 clinical experience and the established facts of physiology and chemistry, and they are a guide to successful treatment, which after all was the goal of 50 years of investigation.

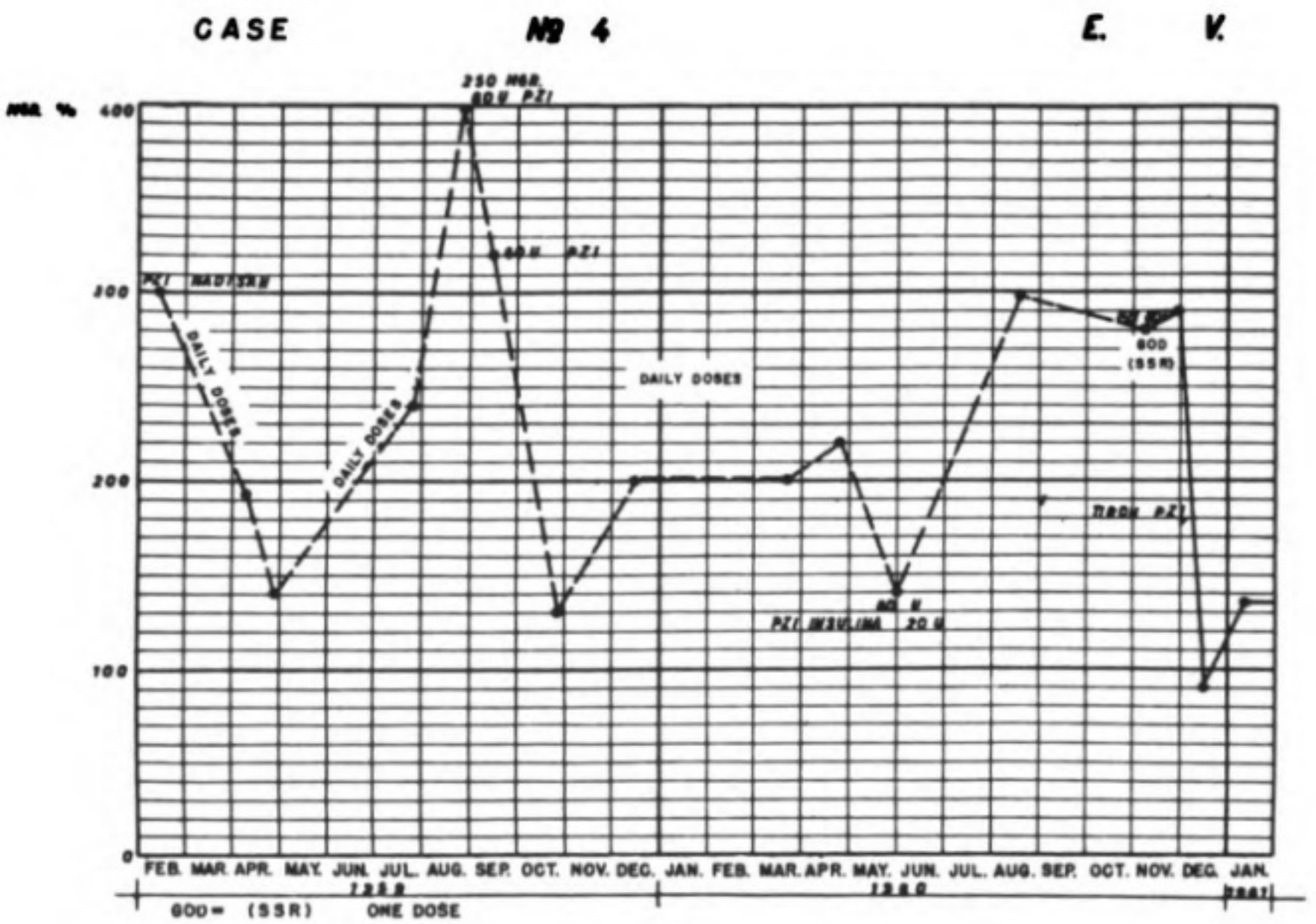
GRAPHS SHOWING BLOOD SUGAR BEFORE AND AFTER TREATMENT

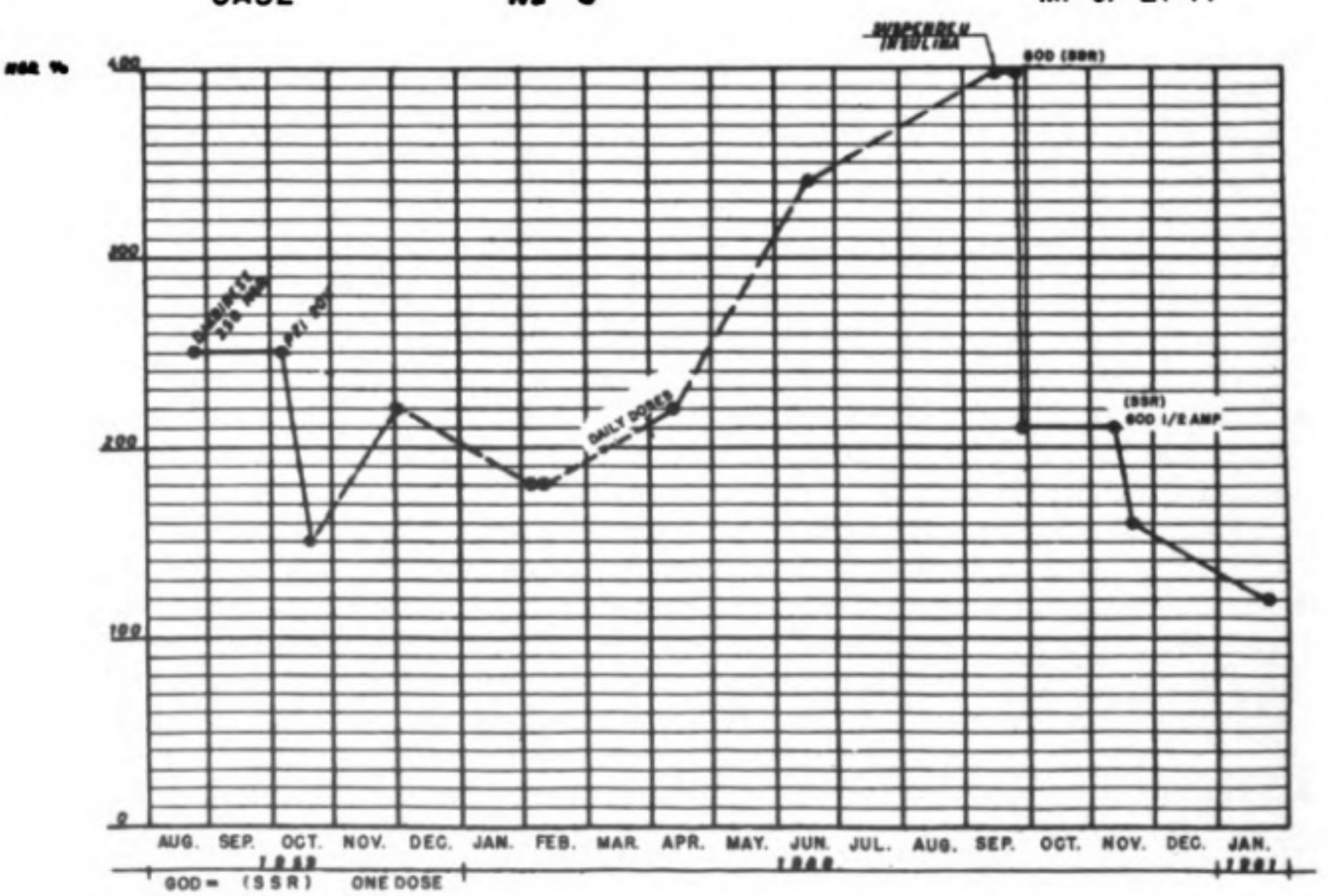




1, 2, 3, and 6 are cases that had no previous insulin or other treatment. The rest are cases of longer standing that had insulin and other anti-diabetic remedies, but could not tolerate them and were not helped by them except transiently.

These cases show the immediate return of function of the islet

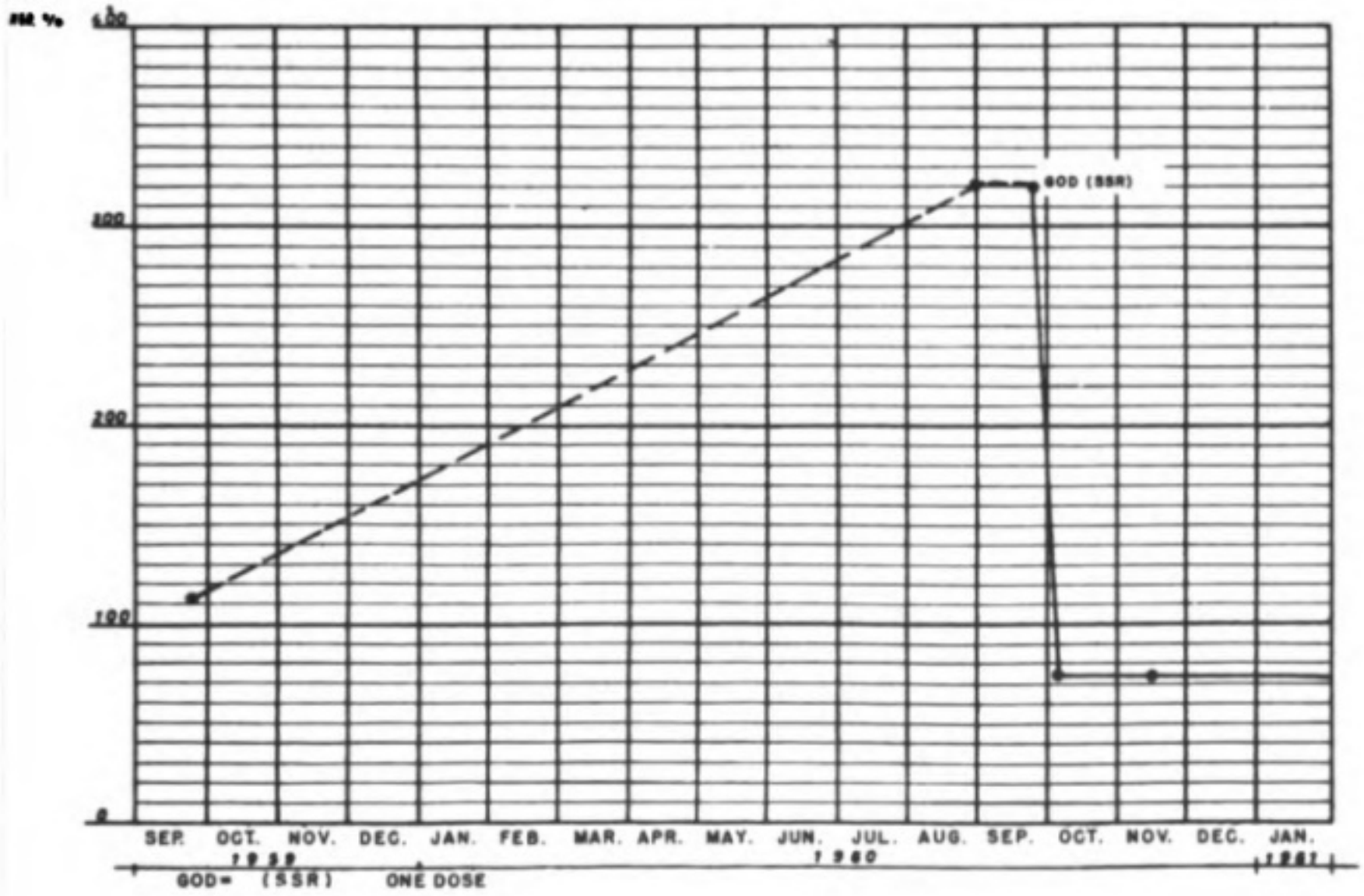




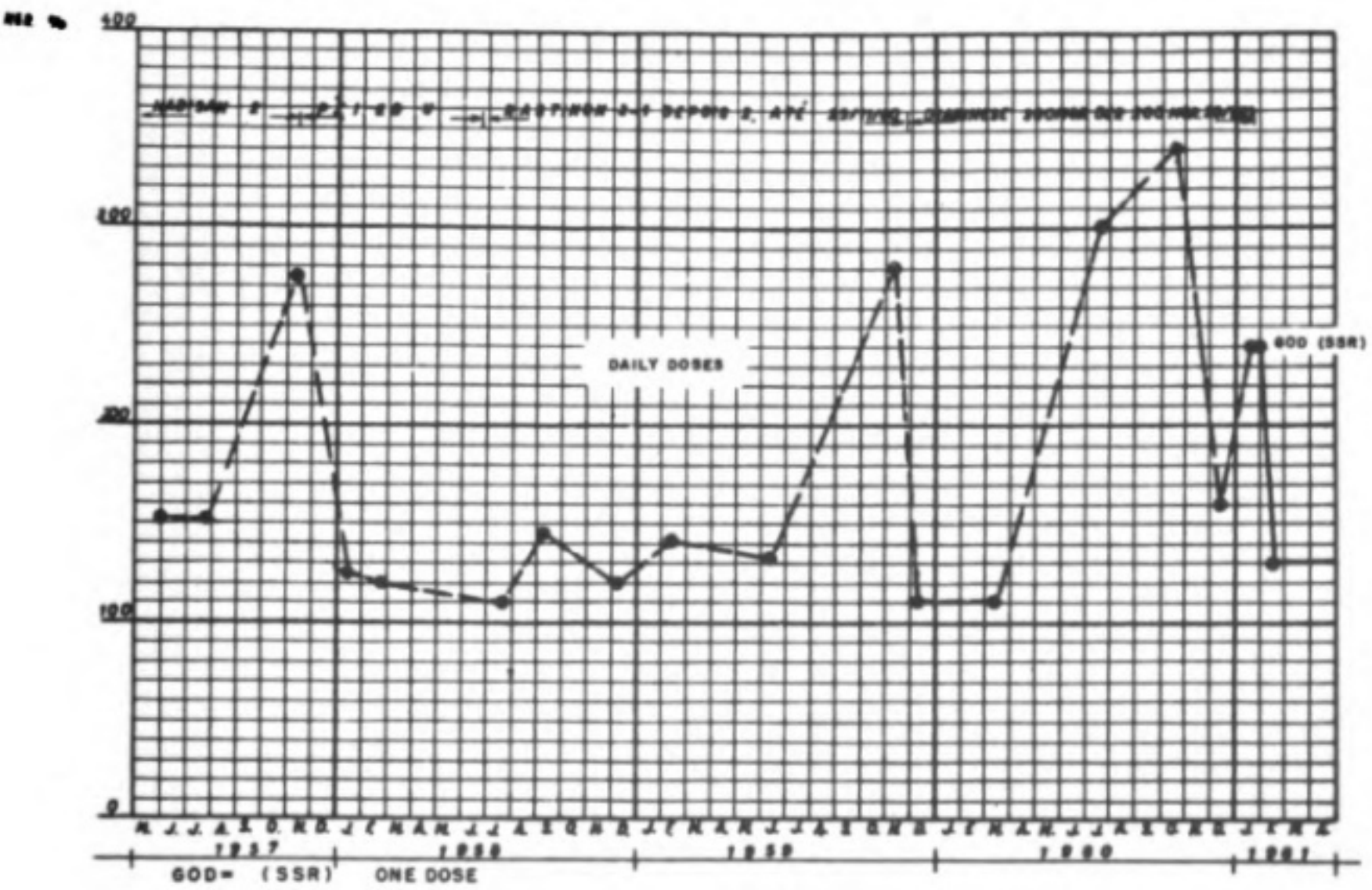
cells that still remain after the inhibiting toxin is removed, and a slower return as reconstruction of islet cells are being rebuilt as in Case No. 5 and 9.

Some cases await further blood tests to show this reconstruction effect, such as Cases No. 8 and 4.

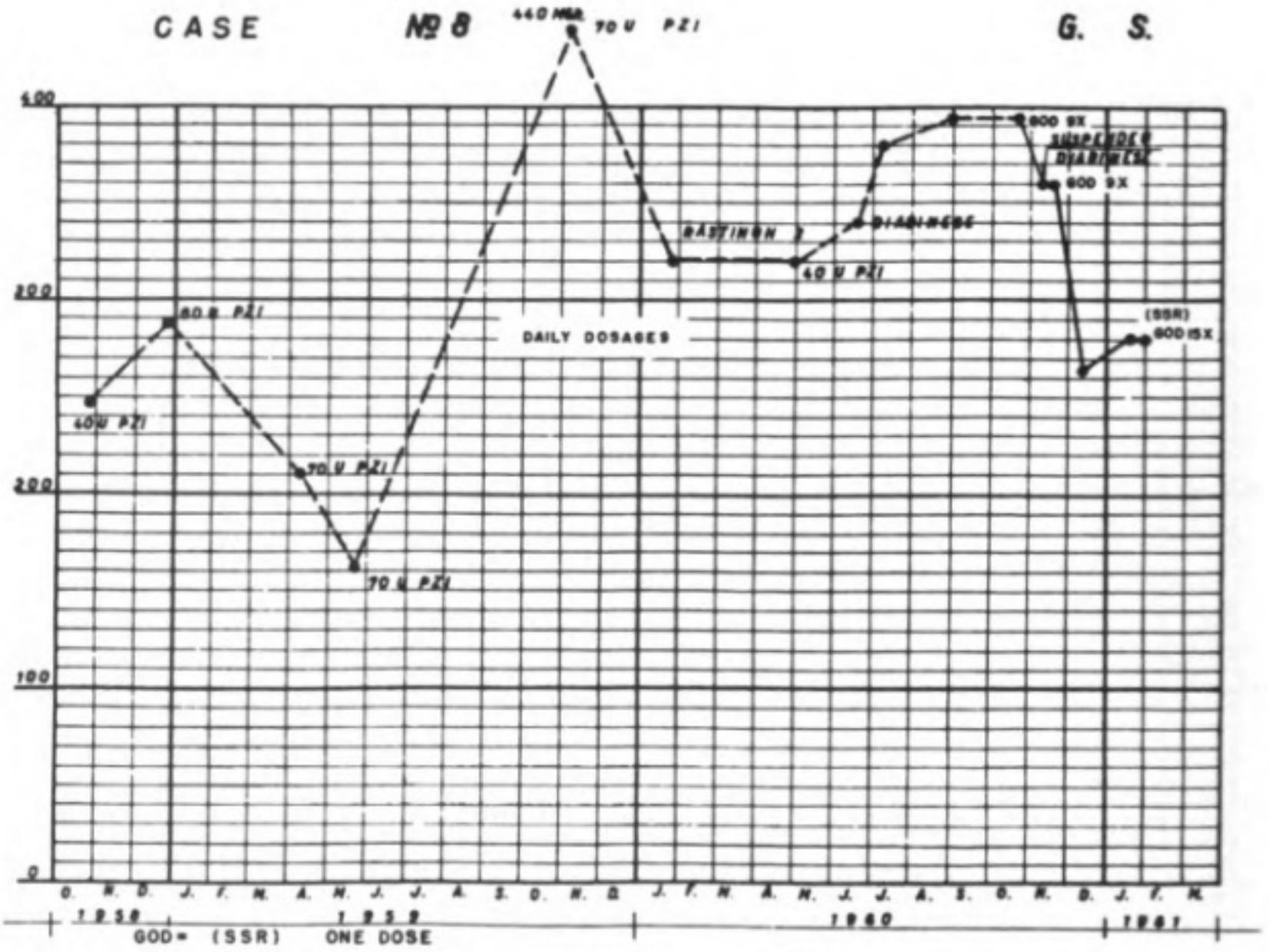
CASE NO 6 L. S.

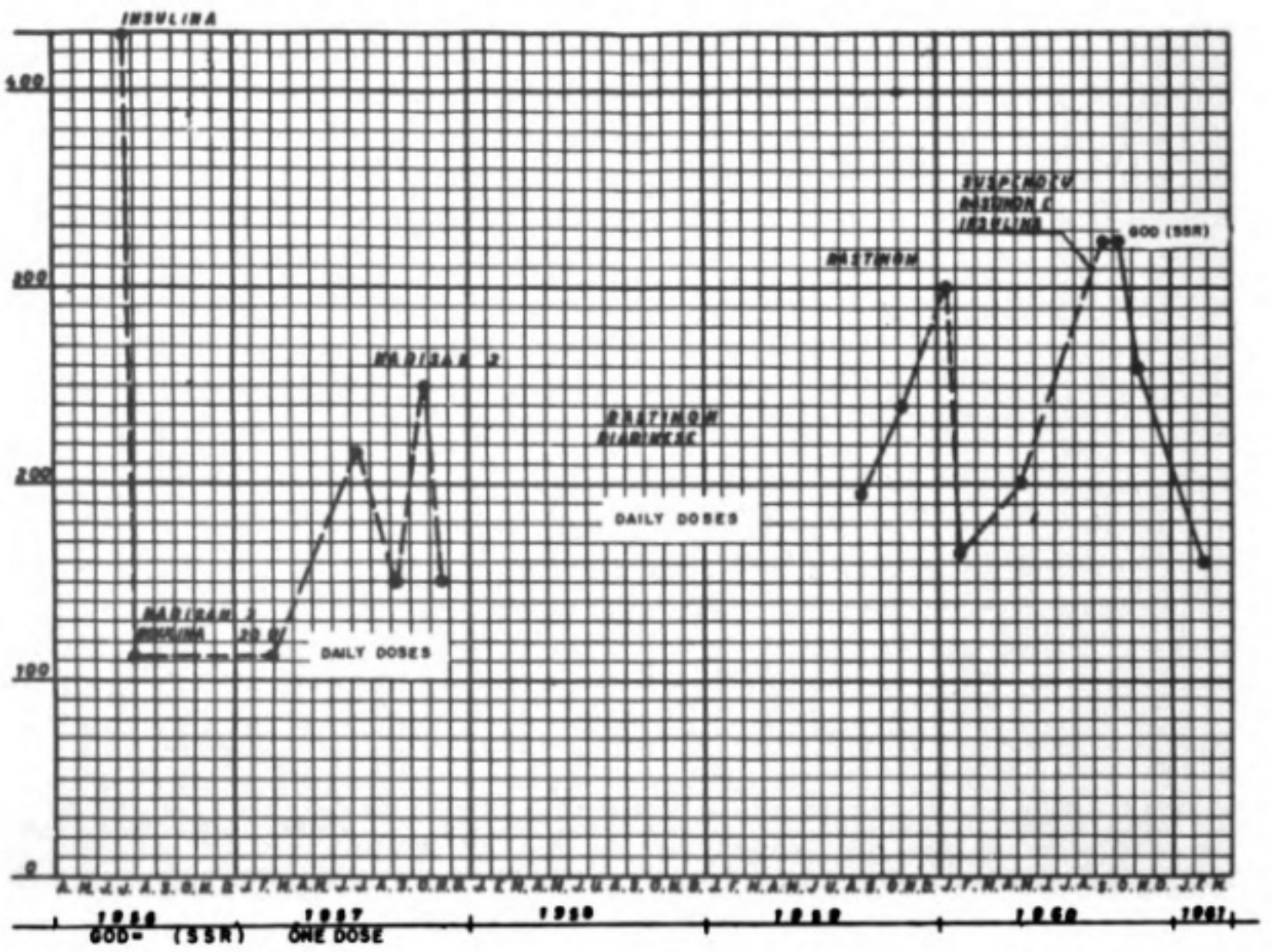


CASE NO 7 A. P. C.



Before giving the SSR the diet was changed to unlimited carbohydrate fruits, vegetables, cereals and bread with all the honey and molasses desired, and the animal proteins were all removed as well as all medications and insulin. Tea, coffee, tobacco and alcohol were forbidden.





These patients were all sick in many different serious ways, and their health returned fully after the treatment, and in the late cases the return of full health took place before the sugar came to its low point or the normal.

DYNAMIC PROPERTIES OF DOUBLE BOND CLINICAL DEMONSTRATION PREDICTABILITY

One recalls that the conjugated double bond of a carbonyl group or of an ethylenic linkage activates many psysiological processes pointed out in the text. These activations demonstrate its value as an electron bridge, even in the high energy carrying phosphate esters and especially the enol-phosphate esters. Activation of the function of the carbonyl group in various capacities, as in the deaminations and decarboxylations co-enzymed by pyridoxal phosphate are demonstrated in their structural formulae. However, no attention has been given to this dynamic function of the double bond. Our own thesis made use of this action long before such ferments were discovered. Thus one recalls that the mobile electrons of an ethylenic linkage activate both the dehydrogenating and condensation processes of the FCG's of energy production and energy acceptance. In the pathogen it activites the position alpha thereto and determines its ability to integrate with the host cell FCG's and with their activating double bonds. And after integration has taken place it invites the oxidative separation of the pathogen from the host cell. We saw that the various unions were accomplished by azomethine condensations and free radical additions as mentioned in the text. In Pyridoxal phosphate's coenzyme services as in some other recently discovered activities the azomethine condensation is now identifiable. All such activations depend on the ability of the ethylenic double bond to pass its mobile electrons on to the position alpha to it, of course, as influenced by the substituents present in the molecule. The double bond is therefore identified by the fact that the condensations and cleavages take place at the point alpha to it and it holds a critical value physiologically, pathogenically and therapeutically. We can also demonstrate the presence of this double bond in the pathogen after integration has taken place, by causing cleavage of the double bond itself, when we make use of its identifying characteristics. The cleavage results in the liberation of the host cell FCG from the pathogen. This is demonstrated biologically by the cure of the disease.

The identifying characteristics are: (a) the greater rigidity of the double bond against steric rotation as compared to the greater ease of rotation of the single covalent bond in response to environmental influences; (b) the ability to add a radical or atom at one pole or at both poles in response to the number of atoms or radicals supplied; and (c) when an addition is made at one pole a free radical is formed at the other pole. In the presence of molecular oxygen, peroxide free radical formation and cleavage of the bond follows with separation of the pathogen from the host cell FCG system by forming two terminal carbonyl groups. The biological evidence is the rescue of the host cell with return of its normal functional status. The pathogen is no longer found.

The double bond is further identified since its characteristic rigidity against rotation provides that the distal pole will hold the same steric relation to the plane of the double bond of the functional carbonyl group (FCG) in any particular disease or pathogenic integration. Whereas if the bond were single, easy rotation in response to various influences would place this pole in

positions where attacking atoms or radicals could not always reach it. Therefore, the number of successful attacks would not be predictable because of steric hinderance. The number of successful attacks on the other hand, is predictable where the bond is double because the steric advantage holds throughout any particular pathogenic category. In other words, where the double bond is concerned, if a successful attack is made once in any disease category, it can be expected to be successful in all other instances of the same disease, other things being equal.

Different pathogens would be judged to present the distal pole in the same steric relation to the FCG double bond, if a predictable response is had in each of a series of diseases caused by different pathogens. This fact has been realized. So we conclude that the least common denominator both in the pathogenesis and in its correction depends upon this ethylenic linkage of the pathogen, and *nature provides for the cure right in the pathogenesis.* It is easy to conclude on this basis that animal life was originally free from disease. Only one protective atomic value was needed. We have shown in the text what it is, the SSR. Now we will show by an alternate procedure what the curative process is and thus corroborate our postulate, and also the standard status of the pathogen's activating double bond.

For this demonstration we used the following procedure, which differs from the procedure used throughout the text. The latter, one will recall was the use of a highly activated DEHYDROGENATOR CARBONYL GROUP, the SSR, which brought about a free radical alpha to this double bond of activation of the pathogen after integration. The sequence was peroxide free radical production and cleavage alpha to the double bond. Now instead of an oxidizing agent, a HYDROGENATOR, a reducing agent of high activity is used to add hydrogen to the distal pole of this double bond by preference when given in sufficiently high dilution, and which adds to both poles when given in adequate or saturating concentration. For steric reasons Hydrogen is a better carrier of the free radical that makes the additions than any complex group of atoms.

When a dilution of one part to a billion or higher is given, only one pole, the distal pole, is reduced, and a free radical is formed at the other pole. This free radical goes through the regular sequence of adding molecular oxygen to become a peroxide free radical that causes cleavage of the double bond itself with production of two carbonyl groups, one belonging to the FCG and the other being terminal in the pathogen as an activator of further oxidation. Biologically the results are restored FCG function and cure.

Further as the double bond can add two atoms of hydrogen, one at each pole, and thus become resistant to oxidative attack when sufficient reducing reagent is given, the integration with the host cell is made permanent, and the disease remains. In serious viral diseases in animals as hog cholera, fatality resulted in 100% of cases treated with concentrations of one part to a million. The same was true in aftosa, dog distemper, some bacterial infections and after poisoning with a tetanus toxin. In animals and man with

the high dilution of one part to a trillion and higher, rapid corrections were had in all diseases treated, predictably. These include anterior poliomyelitis, malaria, cancer, the acute crises of coronary occlusion with infarction, chicken pox and in some hereditary degenerations of serious degree.

Thus by steric and bipole evidences, the double bond of activation of the pathogen is demonstrated after integration has taken place, and its part played in both the pathogenesis and in the restoration of normalcy are also seen.

It is helpful to contrast this action with that of the SSR of the text, since the latter is a dehydrogenator and directly an oxidizing agent, while the action of the reducing agent is indirectly an oxidation, but only when used in very high dilution. The SSR shows corrective effects in every dilution from one to a million up to one to a trillion and higher. Here only one action can take place and that is the removal of a hydrogen atom alpha to the activating double bond. This dehydrogenation is followed by the usual sequence of peroxide free radical formation and cleavage alpha to the double bond, and liberation of the host cell. Thus only a curative action can be had. When the concentration is needlessly high, as one part per million, the recovery reactions are uselessly too uncomfortably vigorous, without anything being gained over the recovery had by a reasonably dilute dose. The recovery gained by the reducing agent requires an appreciation of the basic chemistry involved When correctly conducted it is just the same as when the oxidizing agent, the SSR, is used. A few cases will show this. They will be greatly condensed to give the main facts.

ANTERIOR POLIOMYELITIS PARALYTIC

Miss N. L., trained nurse, age 33, took ill August 16, 1947 with the prodromal symptoms, headache, nausea, terrible leg and back pains and leg stiffness. On August 19, at 4 p.m., both legs from hips to toes were paralyzed, could not support her, and the pains were worse. I saw her at 11:30 that night. There were no tendon reflexes in the legs, no knee jerk. When a pencil point was jabbed forcefully into the soles of her feet, there was no effort at withdrawal, and no muscle contractions could be felt. This is the best test of all. The fever was only 102.5°F. The spine was getting stiffer and the neck also, so the disease was spreading. The injection of 2 cc. of the one to a trillion dilution of the Reducing Agent was given in the right triceps muscle. Results: On the next morning at 8, the mother phoned that the girl could move her legs, slept some and ate a little. At midnight I saw her again. She could walk, felt pretty well, and was hungry. In two more days she was ready to work. All reflexes were normal.

MALARIA, CHRONIC MALIGNANT TYPE

Mr. L.M., age 36, gas station attendant, World War II veteran, contracted malaria in the Pacific area, treated in Veteran's hospitals for many months,

but without results. Attacks still came. After one on June 13, 1947 he was given an injection of 2 cc. of the one to a trillion solution of the Reducing agent. Result: No more attacks in the two years that followed under our observation. His health returned very nicely. The history states this was a falciform infection.

CANCER

Mrs. F., age 58. The history claimed full diagnosis of cancer at the Henry Ford Hospital, with hopeless prognosis. Examination showed a weak cachectic woman with an abdomen greatly enlarged with hard irregular tumification and a pelvis so envolved the landwarks were completely obliterated. Hemorrhage and drainage from vagina and rectum and the enormous envolvement, with the great anemia, indicated she could not survive many days. We did not wait for the biopsy report as we considered the situation hopeless and made a clinical diagnosis only. The Reducing agent was given 2 cc of the one to a trillion dilution on August 17, 1947. In a month's time drainage, pain, bleeding and functions had improved. She gradually recovered according to reports. She moved to California. The last report came in August 1961, stating that she fully regained her health and is still well.

CORONARY OCCLUSION

Mrs. S., age 74, with a long history of arterial sclerosis and aortic insufficiency, usually carrying a blood pressure of 200/100, had a severe coronary attack in June 1960. The SSR was given before true infarction could happen, and the recovery was immediate. The ECG showed no infarction. The following year on June 27, 1961 she had a very severe attack. She was hospitalized and given every possible aid while under the oxygen tent, but without favorable response. The situation rapidly deteriorated, B.P. 190/100, great pulmonary edema, thin weak pulse at 130 P/M, great dyspnoea, general cyanosis, chest pain, and she was at the point of collapse when the reagent was injected in the triceps muscle, 2 cc. of the one to a trillion dilution. The response was immediate. The house physician took the pulse while the injection was being given to check the change that followed. The needle was a No. 12 bore, one inch long, to accommodate the rapid injection. Just after the needle was withdrawn, a part of a minute after the injection was made, the pulse immediately changed to 60 per minute, the dyspnoea ceased, the cyanosis faded away, and as fast as the blood pressure could be taken it was found to be 140/80. She was comfortable in a minute. The house physician was "shocked." The next morning the ECG was taken, and showed nodal fibrilation, extensive infarction of the Septum, and ischemia extending over the lateral wall of the left ventricle. Another ECG taken a week later showed marked improvement with diminution of the area of infarction. The day

following the crisis the Blood Pressure was back to its normal for her, 180/100. Her health remains what is considered good for her.

During the first attack the blood was extremely jelled so that the ischemia of the heart muscle gave the same symptoms as a thrombosis since the blood was not flowing. But on receiving the SSR the blood colloids were quickly charged and good dispersion restored the fluidity for normal movement. Infarction was prevented. During the second attack the jelling of the colloids went on long enough to injure the vessel intimae and true clotting took place over a wide area, and besides a still wider area was ischemic from extensive blood jelling. Wherever the true clot had not interrupted the circulation, the dispersion was restored and a good flow assured. The perifery of the infarcted area showed so much improvement within a week that one could conclude that much integrated toxin was really removed. The speed of the restoration to normal in both instances was the same and in each instance depended on the separation of the pathogen from the FCG of all the cells concerned. She was attended by Dr. Jayme Treiger in both attacks.

CHICKEN POX

Dr. Treiger reports on 14 cases of Chicken Pox treated during an epidemic in 1961. Twelve cases gave immediate responses so that the lesions did not progress any further after treatment with the reducing agent, but started to involute right away. In from 24 to 48 hours the recoveries were complete. The other two cases recovered in from 5 days to a week. Thus the virus was instantaneously removed from the tissue cell FCG's, in the first 12 cases. The results in the two cases that failed to respond to the reagent were also predictable since they did not follow the regime and ate such antagonistic foods as smoked ham. As in measles where the recoveries require about 12 hours after the patient has been treated, the response is predictable and the scientific basis of the therapy is thereby established.

HEREDITARY GENERAL ATROPHY-IDIOCY

A girl of 11 years of age with the intelligence of a child of one year, could walk before she was three years old, but not afterward. Her arms, legs and body were atrophied, but not her head although her brain failed to develop. She had convulsions every day of her life until July 1, 1961 when the Reducing Agent was given. On that day she had convulsions and was unconscious all day until the remedy was injected by Dr. J. Treiger. After the injection the convulsions stopped and she had no more until during the sixth week reaction when one mild convulsion took place. There has been a steady improvement in her health besides during the 2 months that followed the treatment up to this date (9/1/61).

DIABETES WITH GANGRENE

Dr. Julian Baldor reports the following case of diabetes with gangrene. Mr. A. C., age 71, had been diabetic for five years. He had had many high blood sugar analyses and insulin injections, but a steady decline in health. On January 5, 1961, he had a severe crisis; blood sugar 375 mgm.%, high fever, much pain in the right foot and was approaching coma. Gangrene of the fifth toe set in. Following toe amputation on February 12, 1961, fever continued and there was a rapid spread of the gangrene to envolve the entire foot with numerous fictulae on the dorsum and plantar surface. Further amputation was considered, but because of the virulence of the gangrene, it was considered useless, even though done above the knee.

The Reagent was given on March 18, 1961. Two days later, on March 20, 1961, Radiograph I of the right foot was taken showing bone destruction from diaebtic gangrene. After treatment, improvement followed. On March 30, 1961, without insulin his blood sugar was 124 mgm.%. No medication was required. In eight weeks the lesions were healed, the edema was gone and healing of the destroyed bone tissue in exact normal detail was observed in the radiographs. Radiograph II taken on June 3, 1961, shows bone reconstruction where the gangrene had formerly destroyed the boney structures. Only where amputation removed the bone was no reconstruction possible. His foot became normal, all fistulae were healed, function was good and he is able to walk normally. On August 25, 1961, his blood sugar was 80 mgm.% and his health remains good. He is a good demonstration of the reversal of the pathogenesis.

We have fully reviewed the established theoretical chemistry basic to our postulate, and have seen how it is exemplified in Nature's biochemical systems. We have seen how the double bond is postulated to serve in the production and correction of ALLERGY, AND ESTABLISHES A LEAST COMMON DENOMINATOR IN DISEASE CONDITIONS.

The reversal of the pathogenesis has been observed many times in the reconstruction of bone that was destroyed by cancer. In this process soft bone is formed first and after its shape and dimensions conform to the normal with the fragments drawn to their normal positions, the soft bone calcifies and is hardened so it can function normally. The original normal structure is thus restored as seen in the radiographs. Soft tissues as the vocal cords, the uterus and the stomach wall have also been observed to be reconstructed to good functioning-normal. Thus the pathogenesis was reversed in its totality. So it is not unreasonable to suppose that if the FCG of the tissue cells and of the reticuloendothelial system possesses an O/R potential equal to or superior than that of the SSR, disease changes as we know them today could never be initiated nor propogated. Was man so equipped when the Creator pronounced him perfect in the beginning? And what has unnaturally produced food and other features of civilization to do with our present status?

Without doubt it has to do with the anoxia that makes possible the integration of foreign material with the host cell functional mechanism, for the eating of meat was never prescribed by the Creator, and wherever dead animal tissue is found, be it in the ground or in the colon, the germs that putrify it into fertilizer for plants are there, and the toxic amines are one of the products. These cause the jelling of cellular and blood and lymph colloids that block the circulation, and also combine with and inactivate the carbonyl groups that should initiate the oxidation progressions and serve electron transport from substrate to oxygen. Sulphidryl compounds add to quinones and interfere similarly.

Then how often can the functional mechanism, as we have outlined it take on a pathogenic free radical and accommodate the oxidative cleavage to get rid of the pathogen and function normally again? In the case at hand, the diabetes has not returned and the leg and foot tissues remain normal, as he lives on a clean vegetable-fruit-cereal diet. Moreover, there are other sequelae to diabetes that offer data that answer this question, for example, diabetic retinitis. Here the basic cellular pathology is the same, which can be estimated by the delicate sense of vision. Also in myelogenous and demyelinating diseases of the nervous system the pathology can be estimated as it comes and also as it is caused to leave under treatment of this text.

In all cases the toxic amines, sulphides, etc., produced in the putrid colon strike the endothelial cells and intima of the small blood vessels first to cause them to swell and multiply while the tissue colloids are jelled to a hyalin substance, all of which tend to shut off the circulation and oxygen to the functional elements and allow the pathogenic integrations that constitute the specific diseases. This can be seen in daily practice; and two cases that recently appeared for consultation illustrate very well. One is a case of diabetic retinitis where the pathogen stepped in as supplied by a toxic colon, and was separated out when the oxidation therapy of the text was employed, and again returned with neglect of colon hygiene and again responded under treatment. The other is a case of multiple sclerosis which shows the true course of the pathogenesis as responsive to a putrid colon.

DIABETIC RETINITIS

A few pertinent facts will suffice. Mrs. J. C. C., age 39 years, was under good insulin management for 12 years for diabetes discovered after a still birth. The vision did not start to deteriorate until a year later when the glycemia was found to be 350 mgm.'%. Put on protamine zinc insuline and restinon, the glycemia dropped to 280 mgms.%. A good cardiologist found the circulation to be grossly normal, the B.P. 14/8, pulse 90 b.p.m.

Blood examination on 9/10/64, showed a glycemia of 216 mgms.% and at this time the vision had so seriously deteriorated that she could not make out forms but could distinguish light from dark faintly. She was given

a dose of the 6x dilution of parabenzoquinone, and 18 days later the dose was repeated. In a few days a reaction set in with vision entirely absent for a day. Thereafter the vision improved steadily so that at the end of 3 months she could read large newspaper print and got around very well. She then deserted her vegetarian regime and indulged freely in the animal proteins she enjoyed so well, and the retinitis returned, but vision was still practical enough to get around. Treatment was again instituted as before and the response was satisfactory to her, she reports. However, she did not return for vision tests.

MULTIPLE SCLEROSIS

Demyelization of the pyramidal tracts is the visible pathology, with gliosis and axone degeneration in advanced cases. The case history facts of the following case under treatment at present shows that the chemical pathology lays primarily in the functional elements, and the demyelization is secondary or concomitant. The course of the recovery process demonstrates this.

Mrs. R. P., age 40, first started to show paralysis of the lower limbs late in the fall of 1963 following an intestinal infection that was diagnosed as typhoid fever, but probably was a different infection, as she had typhoid at the age of eleven followed by falling of the hair during convalescence. This latter "typhoid" attacked her in October, 1963, and its terrible headaches kept repeating every day thereafter between 11 a.m. and 11 p.m. with a fever of 37.5 to 38, until she was placed on our colon cleansing diet and lavages, and given the parabenzoquinone injection on August 3, 1965. It was during this two years of poisoning that the paralysis developed, and then reversed toward normalcy under our "get rid of the pathogen" system.

The diagnosis was made of multiple sclerosis by the experts of the Lahey clinic and of the Massachusetts General Hospital of Boston, with the usual prognosis summed up in a letter by Dr. Chas. Fager, and Dr. Norcross as follows: "Spastic type of paraparesis affecting the lower limbs quite symetrically, worse in the distal segments than proximally, associated with pathological reflexes in the toes and absent abdominal reflexes. For the most part the long sensory tracts testing was normal except for slight impairment of vibrational sensation in the feet. In the upper extremities and mandible, the reflexes were also hyperactive, but from a neurological standpoint there were no other abnormalities, no evidence of any cranial nerve, optic nerve, cerebellar or upper extremity disfunction. I am sorry we have nothing further to offer but this seems clearly to be a case of demyelinating disease of the spinal cord for which unfortunately there is no specific treatment." The cerebrospinal fluid showed a gama globulin of 12.25 mgms.% and there was an eosinophilia of 17%. They recommended heavy animal protein diet for strength.

The recovery course showed that after the two weeks of colon cleansing on a fruit-vegetable juice diet and the injection of the two cc of a one to a

million dilution of benzoquinone and then a regular vegetarian diet, reversal of the pathogenesis followed the rule that the first to come was the last to go, and vice versa. The headaches and fevers left immediately only to return during the reaction periods the third day, the third week and the sixth week for an hour or so. But the point for which we recite this case is that within one-half hour after the injection of the reagent she had a reaction of fibrillation of the muscles that control the toes of the left foot. This lasted for 20 minutes and returned for similar periods several times that day, and continued to come for a few days when it also started in the same way in the muscles of the right foot. These fibrillations traveled from the feet to the calves, and then after the sixth week to the thighs and slightly to the abdominal muscles, and as they left the feet and calves, the use of these muscles lost most of their spasticity, gained much more control, so she could walk to the chair and sit down and get up again easily without help as was required by two attendants previously, and her balance became about normal. After the sixth week reaction her improvement slowed down so that by the ninth week it was stationary. This meant, that necessary elements to the tissue reconstruction were exhausted, and one had to wait until the deficiencies could be overcome. Such recuperation takes time, and in one case had to wait for one and a half years after treatment in a man whose multiple sclerosis was developing for 15 years and was completely hemoplagic for 4 years before he received his dose of GLYOXYLIDE. Then after a year and a half restoration set in and in the course of 4 months he could walk across the room with a little support. In this case during the recuperation his legs jerked every third week for a few days and then improvement set in, resembling somewhat the fibrillations referred to above. So in the case of this woman time must be given to accomplish whatever changes are necessary for further progress. Time factors cannot be predicted!

It will be recalled that at the beginning of treatment the woman was running high fever with intense migrains for many months until the two weeks of comparative starvation with colon washings cleansed her system of the load of the debris from the forced meat diet, that had stuffed the mitochondrial spaces and covered their surfaces so that the necessary electric potentials and diffusion facilities were obliterated and normal function was prevented. Then after the treatment within some hours she was freed of headache, the fever disappeared and the toe muscles twitched, etc. To me this meant that the mitochondria were unloading their metabolic debris and in time they were able to function with improvement until the period of exhaustion set in. Another fast was recommended with colon cleansing to prepare her for the further progress she required. There can be no doubt about the mitochondria being injured by her prolonged toxic condition as favored by viral and toxic invasions, the extent of which one is not able to estimate.

However, the SALUTARY effect of fasting in all chronic diseases must be emphasized and a brilliant case in mind will illustrate. It is a woman of about fifty years of age who had an enormous cancer of the right breast that had metastasized to all quarters of the lungs, causing difficult painful breathing, and also metastasized to the brain so that she was paralyzed quite generally, blind, unable to take nutrition, and then went into coma for about ten days

before the cardiologist was called to strengthen her heart. On seeing the full situation he gave the treatment of the text, giving a dose of parabenzoquinone in one arm and a combined solution of glyoxal and methyl glyoxal (one of the original forms of Glyoxylide) into the other arm. The heart responded immediately, and continued to become normal, the breast and lung and brain neoplastic invasions absorbed so that in eleven weeks, when I saw her she was perfectly normal and up and about, and free of all traces of the disease, and took a trip to Sao Paulo as any normal person. Subsequent X-ray investigations showed she was free of all traces of the disease.

Now then, what was the secret of her brilliant response? It was the prolonged fast she had been forced to follow, when entirely helpless. This unloaded the mitochondria of their metabolic debris so that the oxidations instituted in the pathogens integrated with mitochondria could be burned away without impediment. Never force feed, but fast the patient!

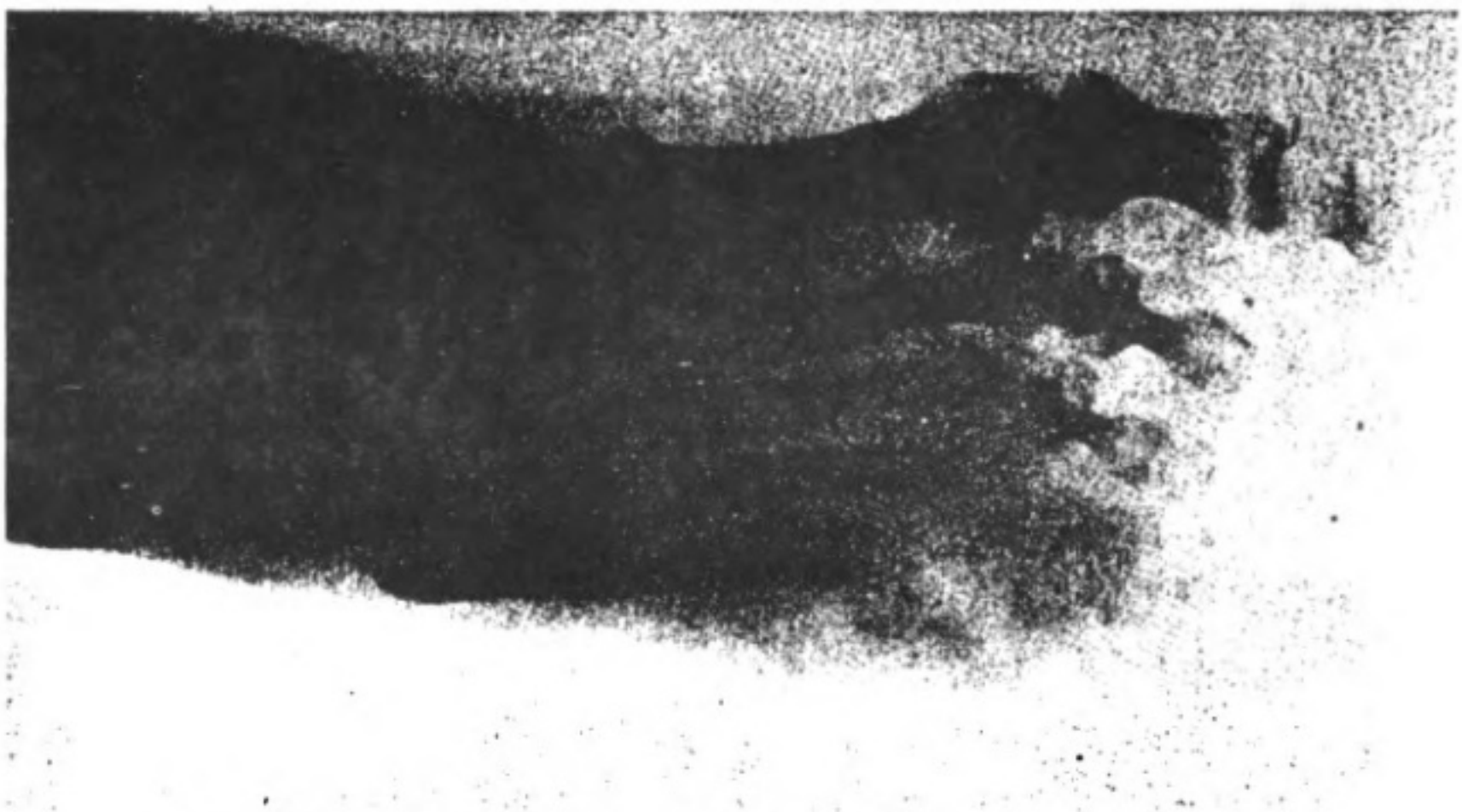
INSOMNIA

Whether insomnia is an allergy of the waking centers in the mesonceph-alon or an inhibition of the sleeping centers, has not been decided so far as I know. But it makes little difference. For wherever the interfering pathogen is integrated, it proved subject to oxidative removal and freeing of the center in a matter of a few days, so that normal sleep became the habit. One case of insomnia lasted twelve years without sleep in spite of all of the best efforts of the therapists. He recovered permanently in less than a week after one dose of benzoquinone. Another case was only of two years standing, and also recovered in a few days following a dose of the same reagent. One must conclude therefore that sleep is a function in which energy is used, and the oxidation mechanism supplies it.

Interference with this energy production as by toxic amines and sulphides lead to schizophrenic and paranoid states hallucinations and compulsions mentioned only a few times in the text. But there are many others who responded more quickly with perfect sanity. One therefore concludes that the integration of the pathogen in nerve tissue is weak and readily ruptured by the high oxidation potential of the reagents of the text, and the pathogens would then be products of bacterial action generated in the colon, as for example by the tryptophane changes leading to rearrangements of the pyrrol ring to produce lysergenic and reserpine types of products as well as plenty of others.

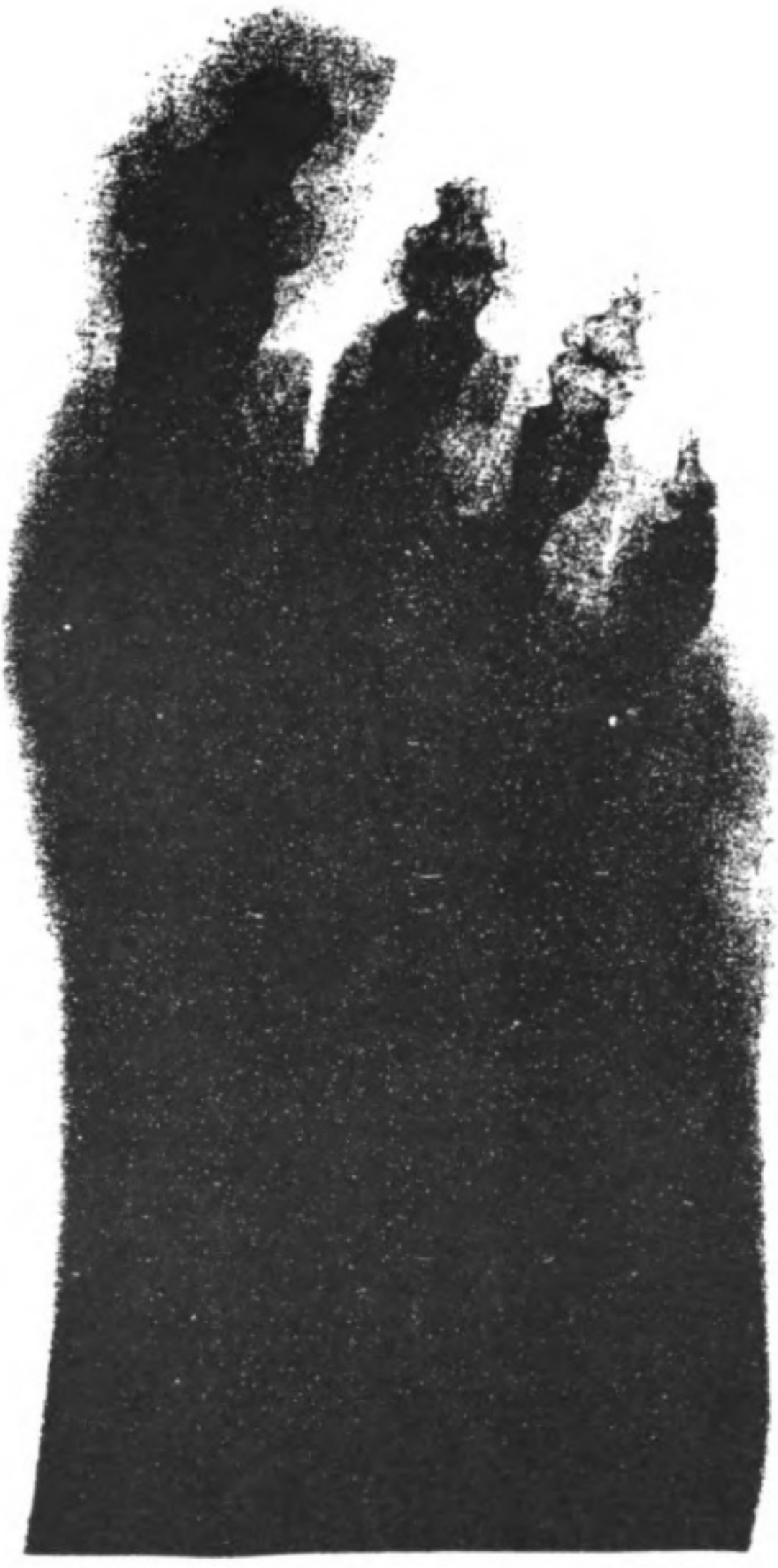
Heart muscle has not entirely lost its nerve characteristics in its differentiation and so its higher tendency to survive oxidation defects influenced the decision to test out heart muscle as a carrier of the glyoxal derivatives used in the 1919 official A.M.A. investigation so successfully (60% to 80% of cures) in terminal cancer, and falsely reported by the A.M.A. ever since. The report in the Medical Record of October 30, 1920, demonstrates a similar curative action in heart muscle. And on this fact was built the larger serial systems of carbonyl groups mentioned on page 90 of the text. Further activation of carbonyl by conjugation with ethylenic double bonds confirmed the truth that highly negatively charged carbonyl groups in correctly constructed molecules are the guardians of perfect tissue function.

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 boney structures.



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

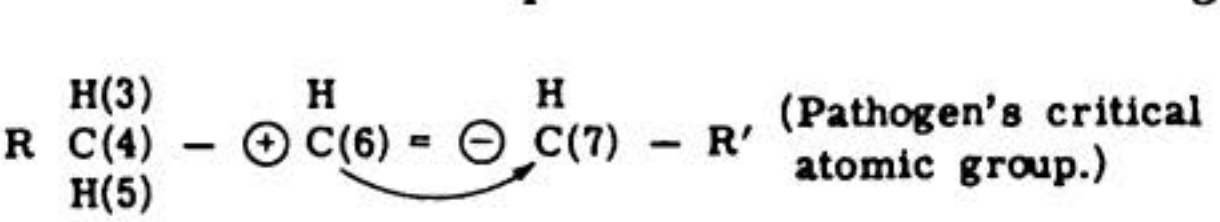
A little more discussion should be given to the easy rotation of the single covalent bond, and also its ability to be fixed in one plane by mutual polar attractions and repulsions of component atomic groups in both the host cell and the integrated pathogen. This rigidity exhibited by each species in 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 FCG's 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 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 and 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 in more than one epidemic. So the species pathogen integrate for each disease is set. A diagram in one plane only can be given on paper 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 critical atomic group from the pathogen which undergoes a stepwise oxidation. There are, however, more than one question that are 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. This fixation is, of course, electrostatic.

CRITICAL ATOMIC GROUP OF PATHOGEN, ESSENCE OF PARASITISM

The pathogen may integrate with the host cell 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 diacetyl aminofluorene require a supporting carcinogen to supply the energy for mitosis. Where the amine condenses with the FCG to form an azomethine double bond and initiate neoplasia, a nitrogen free radical would be expected to mediate the carcinogenesis.

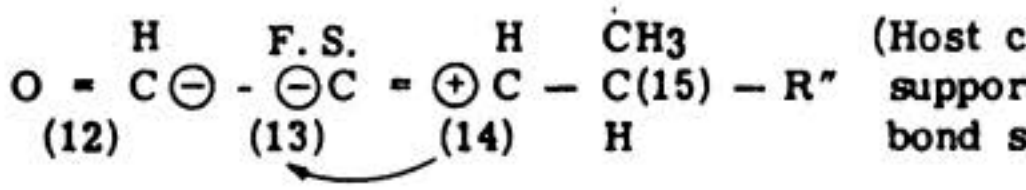


(Pathogen's critical atomic group.)

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

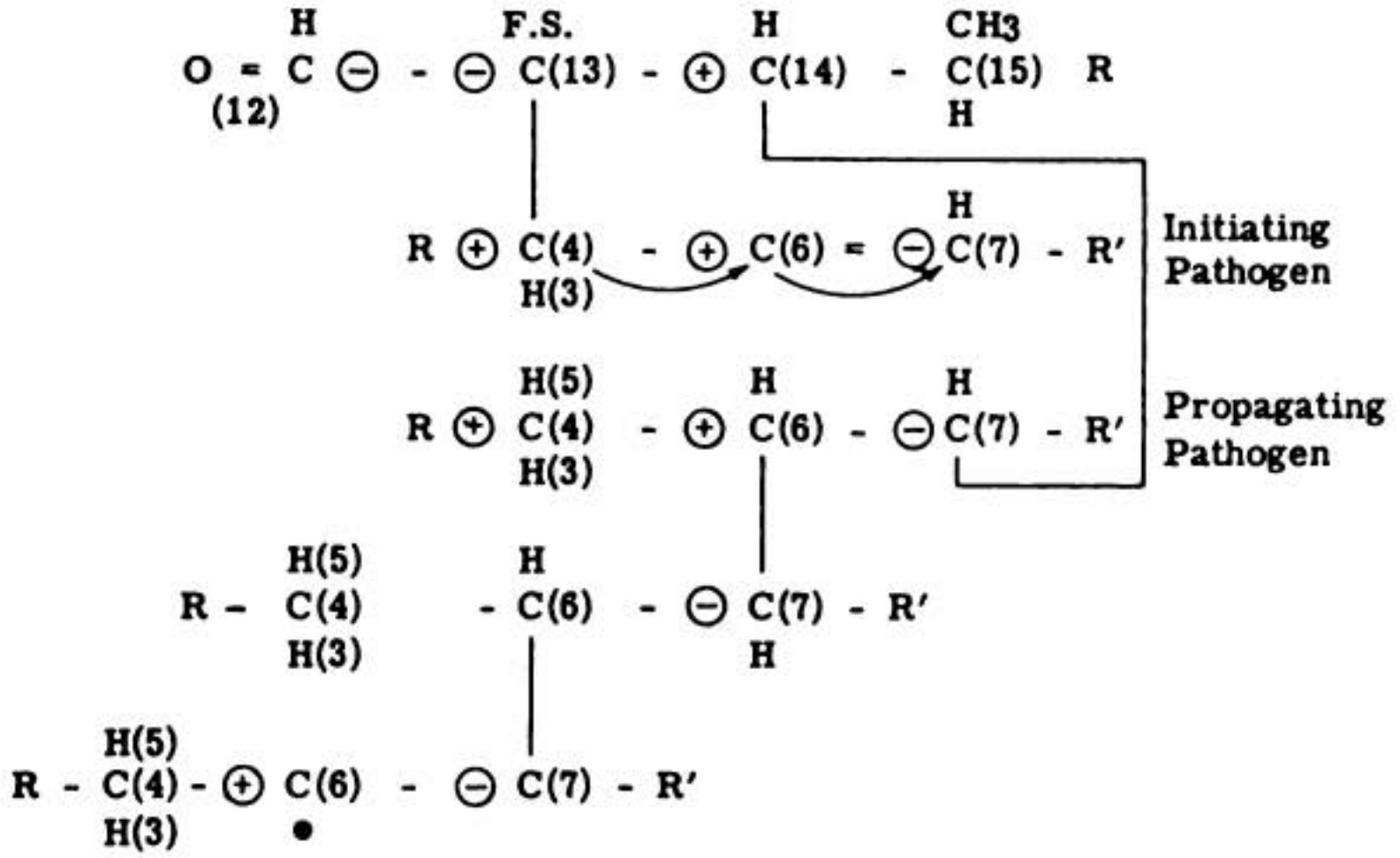


(Host cell FCG and supporting double bond system)

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)



The polymerization continues at C(6) free radical.

To rupture the integration oxidatively, the therapy dehydrogenator remove 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 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 is bathed to form a peroxide free radical that splits the double bond to form a carbonyl group at C(6). The 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 the 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 polio case, the 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.

MITOCHONDRIA AND THEIR CLINICAL ASPECTS

Complicated as the mitochondria are now shown to be by modern methods, the patterns detailed are far too simple to account for their complex performances. And while our observations are made on the intact patient via case history and physical examinations we were able thereby to outline decades ago what the microbiologists are demonstrating today. The clues gained from our parathyroidectomy experiments were indeed fortunate not only in teaching us the effects of activated amine groups in bringing about anoxia, but also the mechanism whereby they blocked the use of oxygen, and the operation of the Pasteur Effect: and step by step, even to the conclusion that the direction of flow of electrons over the double bond bridge whether to or from an alpha placed carbonyl, amine or methylene group determined a normal, a pathogenic or a corrective process. Other guides for determining most likely reaction sites and courses in oxidation reduction substitution affairs were the loosening up of hydrogen-carbon, hydrogen-oxygen, and hydrogen-nitrogen bonds by the electron mechanics of neighboring groups, as in the hydroxyl of the phosphate, the exposed amine group of the adenine and the methylene (2) group conjugated with the ring oxygen of the ribose fraction of the ATP. The same earmarks of energy transfer agency admit creatine phosphate and guanosine triphosphate and the whole series of substituted quinones to the energy transmission belt, each holding its specific position in each particular function, and the kestosteroids may also be included. Each must be identified with its clinical signs of deficiency as the basis of a rational therapy.

Another simple principle in chemistry recalled by the clinical events is that the energy liberated by exergonic reactions must be disposed of or the reaction will be blocked. It usually passes into and energizes another process or is lost as heat. Intrinsic in the mitochondria is the function-trophic balance whereby the gene pattern of its architecture is mainatined. So it appears that the energy the working mechanism is not adequate to use, is shunted into trophic processes that build up the mechanism until it is able to use supplied energy efficiently. In a sense the need for the function determines the amount of energy offered, and the mechanism either uses it or is built up by it, or it passes into the mitotic mechanism in cells that can reproduce. None reproducing cells as the anterior horn cells, undergo mitochondrial reconstruction and Nissle substance rebuilding to meet the functional demands after the obstructing virus or carcinogen is oxidatively removed. Thereby also the trophic neurones are enabled to again resume the development of tissues that were destroyed by cancer, or were stopped from developing during a symbiotic Polio infection as the case histories show. Retarded growth of a limb may thus be corrected 20 years after the polio infection took place, even to nearly the normal.

PROVEN REVERSIBILITY OF CANCER

And this leads to the question posed by Warburg as to the irreversibility of cancer which we show exists only so long as the carcinogen is integrated

with the cell's energy producing and receiving mechanisms for function and mitosis, and which is reversed by oxidatively removing the integrated carcinogen, virus, etc. The case histories of the text are good examples.

The diagnoses were made by America's most noted expert surgeons and pathologists, at our proudest institutions with all the facilities for making a firm diagnosis in the regular course of business. Only a few examples are used here, but they are enough. Besides, the American Medical Association and its Wayne County Medical Society officially proved that cancer is fully and permanently reversible away back in 1919 when they investigated this therapy on "five undoubted cases of cancer" treated in the terminal stages as is fitting for such a test. Three weeks after the treatments were given, the several patients started to improve so rapidly that the official committee became panicky, and closed the investigation, sending the patients back to their distant homes with the warning that further treatments would not be allowed. However, three of the five patients continued to recover until they were again normal with full reversal of the pathogenesis, and permanently so. A fourth patient also made such a rapid recovery from a generalized von Recklinghausen neurosarcoma that had invaded his whole body, and the uncountable tumors dissolved away so rapidly that the committee warned the patient that if he received another treatment he would melt away just like the tumors and he had better return home for safety's sake. He lost no time in doing so. It was not possible to follow him personally thereafter as he lived over 300 miles away. So I cannot personally state I found him cured like the others. However, five years later, patients continued to come from his home town, because of the good results they observed in his case, and unless he were cured he certainly could not have survived over a few months from the time of treatment. Certainly a recovery rate of 60% or 80% of cases treated in the terminal stage is not, "nothing came of it" as was officially reported. Moreover, five years later when I reported the curative results to the Medical Society and asked them to change their misleading report, they persistently refused.

The full permanent reversibility of far advanced cancer was also firmly and factually uncontradictably proven in two Federal Court trials of daily sessions of about five months each in 1942 and 1946, whereby the Food and Drug administration was forced to withdraw their false charge that the remedy of this text was ineffective . The answer to the Warburg question is established "Cancer is fully reversible, and permanently and completely so."

ELECTRON TRANSPORT, SUPPLEMENTARY STATEMENT 1964

Continuing the discussion of page 79 of the text on Coenzyme Q_{10}, a supplementary statement is required. In 1953 when the first edition was written nothing was known by the author about the Ubiquinones, and in 1959, when the present text was written, all that was known to him was stated on pages 78 and 79. But up to the present day an enormous literature has developed, contributed by biochemists in the leading centers of the world. Since

they give practical information yielded by precision methods and instruments not even dreamed of when this postulate was formulated and confirm this thesis throughout, the student will profit by reviewing some of the facts. This is not a review of the subject, but only some of the outstanding facts and our interpretations, helpful to the conduct of the therapy.

Because of the intriguing nucleophilic properties of parabenzoquinone, and the facts stated in the early pages of this text regarding the parathyroidectomy experiments it was chosen with other activated carbonyl groups as in the alpha-keto-aldehydes, triquinoyl, and other polymeres of O=C=C=O as a key for investigating, and for demonstrating the principles whereby pathogens could be dehydrogenated and thus detoxicated before and after they had integrated with the host cell energy producing and receiving mechanisms, and of course to open up the electron transport where a bottle-neck blocked the metabolism. It is of special interest here that the benzoquinone nucleus now offers a historical background that supports the demonstrations we have made over the past half of a century. For it is the nucleus of the Coenzyme Q series, the Ubiquinones, that are so named because of their universal distribution in all aerobic cells, animal, plant, and microbal. Their intensive investigations over the past few years show that the chemical properties we relied on to develop our postulate are actually used biologically by all aerobic cells. This is in contrast to the sworn testimony of leading biochemists in 1942-1946 who claimed that on the basis of their broad general knowledge and great education they could state that the quinone structure as well as calcium and oxygen and its catalysis had no significant place in health or in the treatment of disease, of course, in opposition to our thesis. In the preface to the 1963 edition of the ANNUAL REVIEW OF BIOCHEMISTRY, Szent Gyorgyi gives biochemists some moral advice they may weigh profitably.

The specific place in metabolism held by the Ubiquinones depends upon the way in which the benzoquinone nucleus is dressed up by its substituents so as to fit some specific function as a key in a lock. What we showed was that the properties so used in a particular position, can be used generally when the dressing is removed, that is when it is stripped of its steric hindrances. In other words, when the substituents are replaced by hydrogen atoms as in Benzoquinone.

The structural formula of Coenzyme Q_{10} on page 78 shows a methyl substituent at position 2 of the quinone nucleas, an isoprenoid sidechain at position 3 and methoxy groups at positions 5 and 6. The isoprenoid sidechains of different Ubiquinones have different lengths. Those in man and higher animals have ten carbon atoms and are named CoQ_{10}. Lower forms have from 6 to 9, as the suffix in each case indicates. This sidechain gives the quinone greater lipoid solubility and plays a part in the chromanol change made possible by the position of its first double bond. It does not alter the redox potential of 98.8 milivolts, which is to be attributed to electrons received by the carbonyl group from the other substituents. The coenzyme is therefore a specific dehydrogenator and electron carrier, and is placed between the

flavoproteins and cytochromes in the electron transport chain on the way to oxygen. It is also agreed that it does not serve on the main transport path, but on a different undetermined pathway. One might suggest also that it acts on a very special substrate as well. It therefore serves the general immunity only in a very restricted way. Other highly activated carbonyl groups present with it in heart muscle lipids are far more important as we have seen. Since the average human cadavre contains about one gram of CoQ_{10} and it is present in the urine in health and disease in amounts far greater than catalytic doses, its high substitution is in agreement with its restricted protective value, in contrast with the general high protection offered by the naked benzoquinone structure. The same facts hold for the serial systems of carbonyl groups.

As Coenzyme Q occurs in 5 times the quantity as other members of the electron transporting chain in some tissues, it must have other functions than simple electron transport though it is present in proportion to both the capacity and continuity of oxidation. So with its neat Redux potential one would assume that it serves as a dehydrogenator of especial grade. Since it is shown to undergo cromanol change as does both vitamine K and vitamine E, to serve as a phosphorylator, this latter function is also tied up with the carbonyl group. All three substances are known to enhance sperm motility, to serve in blood coagulation, and to serve in the phosphorylation of ADP to ATP.

However, long before these functions were known, the writer used the naked benzoquinone nucleas which does not undergo chromanol change, to not only enhance sperm activity in bulls, but to restore their total fertility, also, not only to stop hemorrhage immediately, but to restore the colloidal dispersion of the blood so it flowed freely after such gelling as was caused by the guanadin bases and by other toxic amines. Thus it prevented coronary thrombosis, cerebral apoplexy, etc. See example on page 258 which illustrates the inexplicable speeed with which the blood flow is restored by the more active of the carbonyl structures, and the reducing substance. Likewise, the naked unsubstituted quinone can serve as a phosphorylating agent in a very neat efficient way as when it is reduced by its dehydrogenating function, it may add phosphoric acid with withdrawal of hydrogen, and by losing another electron is reoxidized to the quinone structure as it hands over its phosphate group to ADP. Thus it prepares to go through another cycle of dehydrogenating and phosphorylating as an example of highest efficiency with which nothing in the Krebs cycle can compare.

Echinochrome-A, a quinone secreted by the Sea Urchin egg to mobilize and attract the sperm for fertilization is proven active by Kuhn and Wallenfels in dilutions as high as one part for two billion parts of water, and Kuhn and Moewus showed at about the same time (1940), that the female gamete of certain algae mobilized the male gamete by secreting Crocin, a carotenoid which presents a carbonyl group activated by conjugation with a series of ethylenic double bonds, and in dilutions equally high. Thus Nature presents a pattern, whereby carbonyl serves as initiator and transferer of energy oxidatively to serve many functions, the unrestricted "core" of which forms

the basis of the therapy of this text. The Ubiquinones are just one class, and the ketosteroids are another. Coenzyme Q is part of the liver oxidase system that catalyses the burning of certain aliphatic and aromatic aldehydes. It is also present in succinic oxidase, and is found in richest amount in the lipid fraction of heart muscle, — all since 1955 while its structure was definitely determined before 1960.

However this writer had experience with it and other carbonyl groups as early as 1917 on finding a lipoid soluble agent in the cephalin fraction of heart muscle which could be inactivated by adding guanidin. So on the basis that cancer cells were lacking in functional capacity, it was used to treat far advanced cases of cancer, to learn if it would correct the deficiency. Biopsies from the tumors after parenteral treatment was given however showed no return to normal cell structure, but a calcification followed by coagulation in the tumor cells only, which were then invaded by capillaries and absorbed like a blood clot. I therefore named it "Tissue thrombin" in a paper published by the Medical Record of New York on October 30, 1920. At the request of the Journal of the American Medical Association, the Medical Record refused to publish follow-up papers. So we decided that the profession was not free to receive further information because of the monopolistic impass, imposed by the American Medical Association.

In the 1964 Annual Review of Biochemistry, Warburg states in his Preface that he found quinone to show slight curative value in mice with Ehrlich ascites cell cancer, but it proved too toxic for practical use. He depended on large doses to produce hydrogen peroxide as the destructive agent, instead of catalytic doses to initiate dehydrogenations and free radical progressions as we demonstrate in the text, and our court testimony established to be entirely harmless and efficient in the true cure of cancer and other serious affairs. He also reports the successful use of L and D- glyceraldehyde in the cure of Erlich ascites cell cancer, both with equal effect in spite of the fact that the L form is more efficient as an inhibitor of fermentation in cancer cells, thus indicating that the effect is not enzymatic, and claiming the mode of action is unknown. In both instances he overlooks the properties of the carbonyl group which we have demonstrated in our literature to be highly curative when activated by conjugation with other carbonyl groups as in pyruvic aldehyde. Here again we see the block to the advance of cancer research for decades imposed by the American Medical Association and the U.S. government, which has prevented such valuable scientists as Warburg from learning about our findings.

A most significant clinical fact is the presence of CoQ_{10} in the lipoid fraction of the mitochondrial membranes together with cholesterol, lecithin and cephalin. Here, with variations, cholesterol occurs in about equal amount to the sum of the other two. In liver the lecithin and cephalin are each about 40% of the total, while in succinate-Co-Q- Reductase, the cephalin fraction is only half as much as the lecithin fraction, 24% and 48%, respectively. Since it is also known that the reduction of CoQ accounts for

only about one third of the oxidized substrate, other dehyrogenators as our FCG must carry the major part of this function, within as well as outside of the mitochondrial membranes, one concludes.

The most intriguing fact however is that the amine groups of lecithin choline each carry two substituent methyl groups, while the hydrogen atoms of the Cephalin ethanol amines are free and unsubstituted so they can condense with the carbonyl groups under discussion to form labile azomethine bases which are protected thereby from forming permanently inactivated condensations with guanidin and other toxic amines. The other value of the cephalin Schiff bases is their ability to carry the coenzyme right to the substrate, as for example when the phosphate of cephalin is transferred to ADP followed by ready liberation of the carbonyl structure, free to start another cycle. Lecithin in this respect would serve in contrast as a vehicle. Here we find the explanation of some clinical facts that assign cephalin a role in the immunity mechanism.

In many years of medical practice one has seen among members of families with tuberculosis, that those who eat the fat of the meat did not have nor acquire the infection, but those who had the infection, never eat the fat. No doubt the cephalin of the fat protected the functional carbonyl groups all the way from the intestinal lumen to the mitochondrial membranes as stated above, so they could initiate oxidations in the fatty capsules of the tubercle germs, if that be necessary, or at least to correct their faulty metabolism as described on pages 160-182. The other two observations are the reduction in weight of obese patients following a dose of the carbonyl structures of the text, and the restoration to normal the very erratic cholesterol counts in the blood of cancer patients, by restoring adequate dehydrogenation potentials right in the mitochondrial membranes where fats and cholesterol are burned. Further the efficient burning of acetate chains prevents the excessive production of cholesterol. It appears that not the eating of fats, but paralysis of the oxidation facilities here mentioned is the cause of arterial disease, as the case on page 196 illustrates.

All aerobic germs possess Ubiquinones, that is they have carbonyl activated by conjugation with ethylenic linkages that serve the dehydrogenation function, and can be inactivated as described. We postulated that such activated carbonyl groups produce free radicals followed by peroxide free radicals in the substrate to be oxidized. That they actually do produce free radicals is now proven by electron spin resonance spectroscopy. Our postulate is thus supported in this respect, so that when a germ's dehydrogenating power is crippled it cannot obtain energy for survival in the normal way by making harmless oxidation products, but instead produces such toxic products as toxic amines and hemolysins that cause disease. It is not surprising therefore that after contact with the reagents of this text, the dehydrogenating power should be restored, and it is again able to oxidize to harmless materials, the toxic products it had formerly produced. See the examples on pages 73-75 where gangrenous mastitis of high toxicity in dairy cattle recovered rapidly

after a dose of the reagent, with rapid healing, loss of toxicity, and concomitantly a rapid increase in the number of germs. The same holds true for the rapid cure of terminal cases of fulminating tubercular pneumonia and cases with huge cavitations. See pages 160-182 for examples. The time element gives the clue, and culture studies confirm it.

In line with this explanation a recent report from a physician in the frozen North, where an epidemic of fulminating pneumonia was raging, sometimes complicated with measles or scarlet fever, states the following,— "I had to use five doses to save the lives of five small children with fulminating pneumonia, who certainly would have died without it. The results in these cases were spectacular (about five hours in each case cured them)". Such are the usual experiences. Carbonyl, free radicals, and molecular oxygen are the principles. Proton relaxation times, and especially electron spin resonance techniques show that all aerobic tissues contain free radicals so long as they are alive. (Schoffa 1964) Cancer cells are shown to contain less free radicals than normal tissues. This is of course in proportion to their anaplasia and inability to function oxidatively . Any increase in free radical content above that of normal tissue must be attributed to the polymerizing of the carcinogen in proportion to its malignancy, and as activated by magnetic influences, magnetic storms, etc.

These techniques show also that gamma rays are able to destroy carbonyl groups, and thus tend to make the tissue metabolism of the malignant order, quite as Warburg's Oxygen starvation techniques, and with complete reversibility for the addition of quinones or other carbonyl structures of the text restore the normal oxidative progression, when oxygen is also admitted. This is another confirmation of our thesis that the Pasteur Effect is a function of the carbonyl group, and unless the functional carbonyl group is present to dehydrogenate, the Pasteur Effect is not observed when oxygen is admitted. Inactivation of bacteria, reduction of inflammation by gamma rays and evil consequences of the use of modern toxic amine antibiotics may be considered also under this heading.

Intimately connected with the function of carbonyl groups as activated by conjugation with other carbonyl groups or ethylenic double bonds stands copper, zinc and the divalent paramagnetic cations, calcium, magnesium and manganese and also iron. Copper is mainly diamagnetic, its oxide being weakly paramagnetic. Still its presence is essential to the action of polyphenol oxidase, vitamine C, the ubiquinones, and for the utilization of iron in hemoglobin and other activities. We found copper as supplied by the waters of the Great Lakes as essential to the best success of our reagents in the treatment of cancer for nearly half a century. Where the tissues held a good quota such successes as illustrated on page 284 were the rule. Here we have given a few recent confirmations of our thesis from leading research centers that certify our theoretical basis, long established by the clinical results.

THE O=P GROUPS COMPLEMENTS CARBONYL AIDS IN RECOVERY

Before the historic work of the Cori-s, Lipmann, and some others had elucidated the energy carrying phosphate esters during the 1930s, we took interest in the hydrogen attracting powers of the O=P group of phosphoric acid, to compare this double bond with the O=C group chemically and clinically. We had observed the dampening effects of hydroxyl and amine groups on carbonyl action, and by analogy figured that the three hydroxyl groups of phosphoric acid must reduce the dehydrogenating powers of the O=P group. At that time the electronegative units of neither the carbonyl nor the oxygen phosphorous double bond had been estimated. But the dehydrogenating facility as hindered by hydroxyl and amine groups were very evident. Today one can measure their effects mathematically, and it is seen that while the carbonyl group offers 6 electronegative units, the O=P group runs a close second, with 5.6 such units. The position of the O=P group is therefore to be reckoned in the oxidation process as something more than a passive unit in the phosphorylation energy passing function.

One may appreciate the repelling effect of hydroxyl on the electron attraction power of the O=P group by analogy when one recalls its effect in maleic acid, as compared with the anhydride on the attracting power of the carbonyl groups. Similar effects are seen where chlorine replaces hydroxyl as in acetyl chloride. A similar effect should be considered in the increase of energy carrying power of pyrophosphate by condensing two molecules of orthophosphate. Likewise the increase in the attracting power of the carbonyl group produced by conjugation with unsaturated groups as in maleic acid, as compared with malic acid and by the increase of O/R power of diphenoquinone as compared with paraquinone. The effect of unsaturation and anhydride formation, all of which are dehydration affairs, influence carbonyl and phosphoryl in affecting their electronegatativity.

To test this proposal one combines rich hydroxyl carrying molecules as glucose or fructose with phosphoric acid under forceful dehydration and unsaturating conditions to produce their polymeric unions. The increase in oxidation-reduction potential did not reach that where carbonyl is involved, still the energy carrying power was tremendously increased in a way that was useful in the reconstruction of injured tissues and in restoring bacteria from their pathogenic states. Bacteria lost their pathogenicity and virulence as is demonstrated by bacterial plate cultures, similar to that shown on page 257, and by clinical observations sketched on pages 141, 161, 169 and comparable to those mentioned on page 292 in the text and many others.

One concludes that bacteria and tissue cells regained the energy utilization powers required for their normal metabolism and function, and thus their parasitism and pathogenicity was lost. Since the recuperation of tissue cells and of bacteria is accomplished by the carbonyl structures of the text, as ob-

served in other case reports of the text, it is concluded that energy was produced and transferred into the same functional structures by both the carbonyl and the phosphoryl double bond structures under similar means of activation. That both groups serve complementary to each other with carbonyl holding the initial position is seen in cases where both types were administered to the patient and followed by an increase in the recovery rate. It is also to be noted that the reagents were given in such high dilution that the amounts represented only 10^{-12} grams per dose which obviously is not an injurious dose, but instead could only be a catalytic constructive dose. The conclusion therefore follows that the bacteria and tissue cells are restored to their normal places in the biological economy by these two atomic groups, and that disease germs in active and suppressed focal infection loose their pathogenicity whether they are arsenic fast, antibiotic resistant, or otherwise unresponsive to medication like many trypanosomes, thus showing that the position of carbonyl and phosphoryl action is different from that of the current antigerm agents and their high position in biology is assured.

USEFUL REMEDIES FOR CLEANSING THE MITOCHONDRIA

As was seen, the origin of tissue intoxication via the decarboxylated amino acids and sulphides as found in the putrid colon and chronic focal infections, as infected tooth roots, infected sinuses and diverticulae. The colon can be cleansed by the use of pure linseed oil, taken every day at amounts of a soup spoonful once or twice a day or oftener. The oil must be unoxidized for otherwise it forms irritant peroxides. It offers three sets of double bonds which probably present free radicals that activate molecular oxygen which detoxicate bacteria and their products. It makes a good salad oil, but must be protected from exposure to air.

Catalase combines with peroxides to form a peroxide complex which reacts with the toxin substrates and possibly also with unburned food residues to produce oxidation products as for example ethyl alcohol is oxidized to aldehyde. If large amounts of peroxide are at hand it dehydrogenates hydrogen peroxide to form molecular oxygen, that is when more H_2O_2 than any toxic substrate is present to be burned.

It should be used first in small doses in such cases as cancer victims, for the catalase content is reduced in such persons, and the amount of peroxide used should be increased as the ability to produce more catalase increases, as for example during the recovery from cancer under the treatment of the text. Ordinarily one drop of the 10% solution of H_2O_2 per 50 kilos of body weight used but once a day is recommended as a safe procedure. However, one has observed angiospasms of a dangerous type in persons where a dose of ten times as much had been used.

PHYCHIC EFFECTS ON PATHOGENESIS

Psychic influences on the recovery process are of utmost importance since the ischemia and its anoxia consequent to sympathetic-adrenal action on blood vessels of neoplastic and infected lesions can block the recovery process. This is because oxygen is essential to it. Therefore inquiry must be made in every case under treatment to learn if or not some subconscious conflict must be identified, analyzed and harmonized and eliminated, so that no psychosomatic effect can emerge to hinder the blood supply to any lesion. This is important.

Conscious fear-induced protective vasomoter reflexes are coordinated and controlled to meet the need. Subconscious fear-induced sympathetic-adrenal produced ischemia is not adapted to protection, is uncontrolled and may strike any area of irritation, injury, or an area associated with a guilt complex. It may produce fatal ischemic crises through anoxia that favors carcinogenic or viral integrations with the tissue cells. Aside from viral agents the pathogens are generally produced in old scarred-in focal infections, as stated in the text. Being free radical products in anoxic foci they polymerize through the neurotoxic state and then on to the carcinogenic state after which the neurotoxic symptoms disappear only to return transiently in the reversal from the neoplastic state back to the original form of the toxin as produced during the acute state of the infection. Then as the oxidation continues through the action of the therapy agent, the germ, its toxin, and its protective scar are eliminated. The disease is thus cured completely from its very inception when the blood supply is good. However, there are the ischemic psychosomatic effects on existing lesions referred to above as caused by *submerged concepts of guilt.* The vigor of the ischemia so produced and its persistence are proportional to the depth of the conflict between the right and the wrong involved and the energy put into the evil act. Persons of high moral standards show greater depths of the conflict and proportionately greater psychosomatic sequelae. Whereas those who find an evil deed not reprehensible to want to forget it and flee from it, will not submerge it to the subconscious and set up a conflict of sufficient violence to bring on the psychomatic effect, or if they do the sequel will be delayed and not as violent as in the highly moral victim.

A third situation may also result in a highly moral man guilty of a crime done under complsion against his will and tastes as was done by American and British airmen during the Dresden Massacre of a half million of old women and children and the sick segregated there to be protected from war casualties in an unprotected city. Their acts were pure unadulterated murder as there were no enemy military activities or defenses there, and American and British soldiers suffered psychosomatically for the crimes done there. They did not in all instances show the results, but the sequelae were evident in their spematogenic tissues as witnessed in the defective offspring of all types including the high mortality rates of children from cancer — a practically unheard of catastrophy in children before the two world wars.

The practical significance of the psychosomatic apparatus is seen in the

prompt deaths from cancer, insanity or something worse of all the leaders who attempted to destroy the serviceability of the therapy of this text. The casualties included the three surgeons of the official 1919 A.M.A. investigation of this therapy, who lead the destructive maneuvers and false reports, the leaders of the Food and Drug Administration, Federal Court attacks and the leaders of the Federal Trade Commission attacks. Only one had a delayed death who, in accord with the rule, was of too low a moral status to submerge to the subconscious his crime as regrettable. All passive opponents escaped. This is a controlled observation of psychosomatic justice.

NATURALLY OCCURING ANTICARCINOGEN QUINONES

While searching in Nature for plants that present chromophore groups of the order we have proven to be anticarcinogenic, namely carbonyl as reenforced by conjugation with ethylenic linkages or by adjacent carbonyl groups, as stated in the text, three were found in Australia and Africa and two in Brazil. All are naphthoquinones of para and ortho structure existing isomerically and interchangeably. Energy is gained by the change from para to ortho structure and probably the sun and soil provide the agencies for it.

None of the African or Australian pigments have been identified with medicinal activity. And still we may do so simply on the basis of the carbonyl arrangements in each, as they all conform including the Brazilian product to the laws we have identified with anticarcinogenic activity. The Brazilian pigment has not been identified as to structure except by ourselves since we find it to be both the para and ortho forms of a naphthoquinone in isomerism in two different trees. Here they can be extracted from the inner bark, and are named the Pau Arco Amarilo and Roxo for the paraquinone and ortho quinone forms respectively. Both are proven to be curative in cancer and other diseases, the ortho form being most active in conformance to our thesis. Both are of lower oxidation reduction potentials than the synthetic agents we offer in the text, and run from 0.3 to 0.9 volts lower than our reagents including glyoxal, methyl glyoxal, rhodozonic acid, triquinoyl, compound C and the long straight chains of carbonyl groups of the text, simple parabenzoquinone and diphenoquinone. They hence have a much limited field of activity and are adapted to continued use over long periods by mouth, in which form they meet the needs of primitive people. However, as civilization has changed disease systems, the synthetic products we have used for the past fifty years that were kept from the sufferer by bureaucratic and commercial interests must be awaited by present and future generations. It will be of interest also that Rhodizonic acid, being stabilized, and though reduced in activity by the two hydroxyl groups, is of use when taken by mouth and will thus serve those who cannot afford professional assistance. The structural patterns of the others can be represented by that of the Australian pigment Lamatol or the African Lapachol with but slight changes in the sidechains.

The Brazilian pigments run very similar and all must have about equal medicinal values as the others will be found to have.