THE SURVIVAL FACTOR IN CANCER AND VIRAL INFECTIONS

AN INTRODUCTION TO CARBONYL AND FREE RADICAL THERAPY

BY

WILLIAM FREDERICK KOCH, PH.D., M.D.

Detroit, Michigan, U.S.A.
Rio de Janeiro, Brazil

Instructor, Histology and Embryology, University of Michigan, 1910-1914
Professor, Physiology, Detroit College of Medicine (Wayne State University), 1914-1919
Pathologist, Women's Hospital, Detroit, Active and Honorary, 1915-1919

NATURAL IMMUNITY SERIES

Printed By
THE MANNEY COMPANY
1041 Isbell Road *Fort Worth 14, Texas*

TABLE OF CONTENTS

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.

AUTHOR'S FOREWORD

We started our inquiry into the basic chemistry of neoplastic and virus parasitisms in 1916 when facts were few and laboratory methods crude—long before Warburg's classical work was reported. The clinical approach therefore promised to be more comprehensive and reliable as the major source of information. Thus it was evident that neither cancer nor virus infection took place unless function was first impeded, impaired, or exhausted. We concluded therefore that the "front line" energy producing oxidations were protective and burned fuel substrates and toxins in much the same manner. The appropriate process thereto is entirely overlooked in the laboratory attack which still recognizes the Krebs system of oxidations and hydrolytic glycolysis as the only sources of energy for function and growth. On the other hand, both of these systems support virus and neoplastic parasitisms. Thus restrictions in the laboratory approach deprived it of its chance to even make "first base" in the search for the survival factor.

We aimed to identify and duplicate the details of this protective process and found it could be initiated by activated dehydrogenator carbonyl groups and carried along by free radicals and peroxide free radicals. The state of activation of the carbonyl groups determined the range and efficacy of the protection. The process could be blocked by groups that abolish the Pasteur Effect, such as sulphydryl, guanidines and amidines extractable from spermatogenic organs, extracts of tissues of animals that died in great fear, cancer extracts, but not by normal tissue extracts. The process could be terminated at will by reagents that block polymerizations, such as inert free radicals and electrophilic double bonds. Thus its chain reaction character is identified. But the laboratory has no suspicion of its existence as yet. In the experimental production of cancer, applications of complex tars stimulated tissue functions, reproductions, glycolysis, and Krebs cycle oxidation, (Deotto, 1936) while pure synthetic carcinogens depressed the oxidations of

normal and neoplastic tissues (Maisin, Warburg, etc., 1933-4) Both poisons bring an anaplastic depletion of the mitochondria and their oxidative enzymes and oxidative capacity with special loss of cytochrome and its oxidase, succinic dehydrogenase, etc. Both poisons block the initiation and propagation of chain reactions, and mask the protective oxidations of the survival factor; but leave Krebs exchanges, and especially glycolysis, uninjured to support neoplastic and virus parasitism. The laboratory approach thus blinds itself again to the survival factor facts.

Without clinical guidance the error of seeking primary agencies of causation of cancer in general tissue growth provisions as the citrovorum factor, or for a cure in its antagonists, is an expected laboratory fault. The attendant positions of such agencies should be evident at the first clinical inquiries. Coordination of clinical with laboratory data has been our practice throughout, and it should be exceedingly interesting to both research branches to observe their mutual clarifying abilities.

While laboratory research gets lost too often in pathological tangles, the clinician must approach each problem as a matter of physiology in which the degree of departure from the normal must be estimated. He therefore enjoys a more balanced outlook as far as he can go. Even at that in none of the authoritative medical literature does one encounter a description or even mention of the features of a basic recovery process. This means that the cures obtained by orthodox medicine are not true fundamental cures that not only remove the cause but make its existence no longer possible. Apparently medical observers since Hippocrates, have been on a long sleep. The laboratory besides, should have offered mathematical data on each feature so its chemistry can be appreciated. But nothing is observed or reported. The big handicap to progress in cancer research has not only been the lack of frank discussion, but the fixed idea that the farther one gets away from the normal, the closer one gets to the facts about disease. Cramped attitudes have spelled failure for much too long.

From the first clinical inquiries it was evident that cancer cells were normal cells that suffered change through injury, and experimental data supports this fact. The surprise is that

orthodox research and therapies are directed toward killing them instead of finding and correcting the fault so as to return them to their normal harmless status. Indeed, 25,000,000 American dollars are collected and spent annually in the United States alone to push this effort at their destruction, even in spite of the absence of any indicative truly differential basis. Besides, in cases of cancer the tissues in general require the activation of the SURVIVAL FACTOR, instead of its suppression as is brought about by all destructive therapies.

In the pathogenesis of the 100% fatal viral diseases like in the pathogenesis of cancer one encounters the mechanism for its correction built right in as a basic reversible feature. A reciprocity is thus indicated.

This reciprocity between pathogenesis and its reversal is observed in the early phase of neoplasia when one or two periods of improvement gives way to a steady decline, thus showing that the basis for reversal of the pathogenesis exists but can not compete with the pathogenic force and that true spontaneous cure can not take place. It is more evident in the 100% fatal virus diseases as Rabies when the energy taken from the host cell by the virus for purposes of building its progeny, is returned to the host cell as the virus proprogeny undergoes oxidative disintegration. This energy is then used for host cell construction. Pathogenesis and its correction or cure must be considered together to gain a useful idea of parasitism, neoplastic and viral. We hope the sketch given here will answer the salient questions, and leave no doubt as to the mechanism of converting the forces of the pathogenesis into those of the cure process.

In the text the word "cure" is used. Since this term has received so many different meanings by the surgeons, and means anything from survival from the operation to freedom from recurrence manifestations for differing periods, we must define what we wish to convey by the term in this text. By cure we mean the disappearance of the physical and functional changes that constitute the trouble for which the patient came for treatment. In cancer cases it means the disappearance of all tumors, and the healing of the destroyed areas with the reconstruction of the organs affected to such a degree that they function well.

Wherever the provocative change can be identified, its disappearance is also included in the term "cure." The length of time normalcy is thus restored in the cases used in this text is the conventional five years or more, unless otherwise specified. Nevertheless, one reports here only on what has already happened, and no predictions as to the extent of good health are meant by the word "cure." It may be assumed that once the cause of the disease has been removed to the extent that normalcy is re-established, that the natural tendency to keep in balance will provide for the continuance of good health. Still so many variables enter the complex, many of which can not be identified or controlled, that predictions must always be provisional.

In our search for a least common denominator in pathogenesis, it was obviously impossible to treat every disease known to mankind, or even every variation of any known disease. However, we have attempted to cover as large a field as possible, and we base our opinions and the conclusions offered here upon that experience. The selected case histories that are presented in this text are given for the purpose of demonstrating the applicable theoretical principles in order that a better understanding might be obtained as to the true nature of the survival factor.

In such complicated systems as the tissue oxidations, nothing much has been really established. It was so twenty years ago when we were condemned for making this claim, and it is so today still. Every new change in technique brings a new answer or nullifies an old one. In our work we attempted to hurdle the difficulties to reach a practicable working hypothesis. How much is fact and how much is fancy is not easy to establish today, and then after it is "established" it may need to be changed tomorrow. What are facts that stand are the results in both controlling and elimnating the pathogenic state. These necessarily verify some of the hypothetical positions, and maybe all of them.

CHAPTER I

SURVIVAL FACTOR PROPERTIES

THE NEED FOR THIS BOOK, AND ITS PURPOSE

Every alert surgeon of wide experience, after due reflection, should admit that his years of surgery per se have not influenced the mortality rate of cancer one bit. He will explain further, that the prolonged recoveries he has occasionally observed in far advanced cases where he had attempted some palliation, could not be credited to his operation. In other words, the survival factor is something different from the completeness of removal of the tumor tissue. This is the concensus of opinion of the advanced surgeons of today, as it was of the leaders of the past.

Our text deals with the chemistry of this survival factor, its place in the tissue metabolism, its chemical structure and action, and how to use it clinically in true far advanced lethal cancer. But to make sure that our first point is really appreciated, we give a few quotations from a recent article in the Lancet based on the reports of the world's leading surgeons. This article has to do with real, infiltrative, metastatic, lethal cancer only, that has ever taxed the energies and ingenuity of the surgeon, but has never yielded. The hope was that early operation would solve the problem, but now after decades of wide observation in the earliest cases, the survival factor is recognized as depending upon a very different matter than early surgery. 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. Disappointment was complete. The following quotations cover the subject quite well and serve brevity. They are authoritative.

"Despite a long and intensive educational programme 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 show metastasis," and with regard to lung cancer—"If recent experience is typical, however, by the time definite abnormality appears on the radiograph, most cases of pulmonary cancer have progressed too far for successful resection." "In cancer of the breast a patient may survive a long time even without treatment, and individual instances of long survival after operation are therefore no proof of the operation's efficacy." "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 show 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 an editorial reporting the experiences of the world's most advanced surgeons and these are agreed to by many more. They state over again after the advantage of observing the earliest possible surgery in the easiest detectable form of cancer, breast cancer, in large numbers, the same conclusions Sir James Paget published a century ago. He concluded that cancer was not a local disease, and the trend now is to interpret surgical statistics as proving the same.

Irradiation has proven to be a failure. In fact an aggravation of the disease when used preoperatively or postoperatively, was proved at the University of Pennsylvania in 1925 and this has been confirmed. Indeed cancer causing viruses are thousands of times more resistant to irradiation than are normal or cancer cells, and where deep therapy is poured through a

neoplasm of one type a more malignant form or a bone sarcoma is created underneath only too often. The literature abounds in such effects, and it would be interesting to make a percentage compilation. The survival factor certainly is no effect of irradiation, and every competent radiologist will admit it. Indeed the survival factor is destroyed by irradiation as 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 those of physicians not exposed to irradiation. Eight to ten times more radiologists die of leukemia, than general practitioners. (American Roentgen Ray Society convention September 1954, Transactions). Nothing more need be said.

Many factors are now recognized as entering into the etiology of cancer. We will explain each of them later. But one factor is essential, and that is an infectious organism of very unusual properties. Glover, in the "Canada Lancet and Practitioner", 74, (March, 1930) demonstrated that this organism was pleomorphic in nature, with a virus, a filament, a bacillus and a coccus phase, and that it could be proven to be the etiological factor in cancer in accord with the four laws of Robert Koch. He also developed an antibody to combat it successfully and thereby also to identify it specifically as different from all other organisms.

At the same time and independently von Brehmer of Berlin discovered exactly the same organism and proved the same facts as Glover. He also demonstrated important matters with regard to the blood chemistry in connection with the cyclic changes shown by the organism. A committee of German bacteriologists appointed to scrutinize his reports, duplicated them and ended up in general agreement. They confirmed the identification of the organism as different from all others except Glover's, and also the fact that the germ existed in the blood for various periods before a neoplasm developed. They concluded, too, that this organism was the actual etiological agent in cancer.

Later Dr. Jacob Engle and Dr. Geo. Clark, using the facilities of the United States Public Health Service, confirmed Glover's historical and original research, but were prohibited from publishing their confirmation. However, in September, 1953,

at the Sixth International Congress of Microbiology, Rome, Italy, Dr. Geo. Clark reported this confirmation of Glover's work; so it is now in the scientific literature at last. They too showed that the organism existed in the blood for long periods before a neoplasm showed up. Other well recognized bacteriologists have confirmed these findings as they dove-tailed with their own studies, such as Wuerthel-Caspe, and Irene Diller. And of course von Brehmer's work is confirmed thereby also. The time has come, indeed is long overdue, for a change in view point regarding the etiology of cancer.

Progress also demands that the surgeon take a little interest in a few chemical phenomena, and he will find satisfaction in directing his genius and acumen in the mastery of the chemical data fundamental to therapeutic success which we have assembled here in a simplified practical form.

These data carry some pleasant surprises, and show that the recovery or survival factor may arise in more than one source, and indeed may be a contribution of the environment on all sides. Its chemistry is exact and simple, and expresses itself with meticulous accuracy even to the smallest fraction of a microgram. On all sides, nature has offered facilities for the cure of cancer, which can be worked up into the exact chemical structure that is efficient. Yet science has been blind, and noncomprehensive, and equally unappreciative. It is not excusable.

The recovery factor exists in the tissues, and particularly the brain and heart, where it is an intermediary in the "smokeless" oxidation of sugar. We have produced it in various ways by the careful oxidation of several sugars. The recovery factor can be found in the activated charcoal after it is used to bleach cane sugar syrup preparatory to crystalization of the sugar. Here some activated carbonyl groups with definite dehydrogenating power are present and able to produce free radicals that have curative chain carrying oxidative powers. Likewise similar groups, as liberated in an anaesthetic, may initiate a recovery oxidation reaction chain, which will progress, or quench itself before it gets started or goes very far, depending upon the amount present. Hence the far advanced case, or even a "very early favorable case" deteriorates quite rapidly or shows only a short period of improvement after a prolonged exposure

to the anaesthetic. Various cases of cancer that improved after an operation of short duration, were really in fact helped by the free radicals contributed by the anaesthetic, while those that were made worse, received too many—enough to interrupt certain vital chain reactions. The curative powers of certain plants and of the famous Queen Bee Jelly, even in cancer, we attribute to their activated carbonyl groups.

Likewise small amounts of certain reagents that can alter the progress of neoplastic processes, are an ever present danger in the interpretation of experiments done with estrogens and androgens, because of the retained traces of impurities from the alcohol, ether, and other solvents, used in their extractions. Moreover, experiments to learn the effects of resection of the adrenal cortex, the hypophysis, and the sex glands, cannot escape the effects of the impurities in the anaesthetic, nor the free radicals produced in the tissues from them, for these markedly influence the tissue oxidations. All oxides of nitrogen present permanent free radicals, and those generated within chloroform and ether cannot be eliminated from the experiment. All past work of this kind requires re-examination and scrutiny, and re-evaluation from this neglected angle. It will then be seen why so much skilled effort and vast expenditure has led only to contradictory results in the recent past. Clinical adventures based upon such experimentation naturally lead to grief. The most obvious chemical reactions of the ketosteroids and estrogens have not even been considered, namely the slight oxidative power of the former, due to its conjugated system of carbonyl and ethylene groups, and the other reactivities we describe later. On the other hand the estrogens show reductive powers which have definite importance in the chemistry of neoplasia. Neither of these substances represents the survival factor, in fact.

Our first isolation of this substance was from heart muscle, and we reported its curative properties in "recurrent," and advanced cancer, in the Medical Record of New York, October 30, 1920. However, all sources of the survival factor do not offer it reliably or in controllable form. They provide it at times in an altered form that reverses the recovery trend into a rapid decline. It was therefore necessary to work out its actual chem-

istry and produce it synthetically with boosted activity for best clinical use.

In spite of the recent splendid advances in precision chemistry and their fruitful application to biological problems, systematic correlation of the enormous amount of data, now at hand, has failed. The central theme is missing. This we have long assumed to be a fundamental defect in vital processes that provides for parasitism in general, be it viral, bacterial, neoplastic or animal. Through our research we have been able to show that the change is reversible, and is basically the same defect, for the same harmless reagent in a single dose, has made the pathogen disappear, be it a deadly virus or bacterium, a carcinoma or a sarcoma, a malaria or amoebic parasite, the heart worms of dogs or their mange insects. The latter two multicellular types require much more observation, though the data gained so far is supportive. The defect that provides for parasitism, and its correction, are the subjects of this booklet. Though the atomic arrangements involved are somewhat theoretical, they have provided the practical successful avenue of attack for the correction of the parasitism in each instance. The parasite then becomes harmless and disappears, while the host cell with which it was integrated or from which it took energy and nutrition, is left free to reconstruct and function normally again. It will be seen too that on the same basis, many of the enigmas that confront the biologist and the clinician, are clarified.

Since 1912 we have been assembling data on several atomic structures that we decided could support virus and neoplastic parasitisms, and provide for three types of parasite-host-cell integrations, able to bring about functional failure with tissue atrophy, allergic hyperfunction, or neoplasia. The discussion given here is rather brief and introductory to a more detailed and complete report which will soon be offered with practical demonstrations in the technical and clinical phases of the subject. Because of its multiple connections, our theme will require some repetition to simplify each phase of the subject.

We do not think that our presentation here is the best that could be made, but since it gives an account of our own personal

investigations, it is our chore to offer it, and we do so with the belief that the time is opportune for such a discussion. Our aim was to identify the ultimate harmony in a number of apparently conflicting observations, about which factual information was very meagre indeed. **Our subject is the Natural Immunity that is provided by the normal instantaneous and "smokeless" tissue oxidations that leave no trace of their course through isolatable intermediaries, as are formed during such hindered procedures as the Krebs cycle, and in cancer metabolism.** It also deals with the factors that weaken this immunity, so that successful inoculations become possible in certain diseases practically always, and in others, during periods of exhaustion of the protective factors. Obviously, facts gleanable from such rapid and intangible processes must be difficult to establish, and few indeed, so that each had to be given its fullest hypothetical value in order to arrive at the basic principles that govern this high efficiency metabolism. Then too it was evident that these principles required "stepping-up" by synthetic procedures to provide the atomic unions and electronic displacements needed to wipe out the pathogenesis of deep seated disease where the immunity offered by the natural processes proved insufficient to give protection.

Later on we will show why we concluded that this immunity is a procedure of converting toxin into antitoxin as a chain process, or free radical affair. (Koch, Cancer Journal, October, 1924, (Philadelphia) and Koch Investigations, p. 34, 1924.) **But we first ascertained that the protective principles could actually be demonstrated to exist in the tissues, and be extracted therefrom, for use in the successful treatment of cancer.** We also learned the significant atomic groups concerned in this protection, which since 1918 we have been reproducing synthetically. The extracts were made from beef heart. (Koch, New York Medical Record, Oct. 30, 1920.)

We named this extract "Tissue Thrombin" because of its coagulating action as initiatory to the digestion and removal of the neoplastic cells, just as milk or blood is first coagulated with calcium addition as the first step in their digestion. Here too the cells showed calcium reaction as the first change. This

extract could be preserved temporarily as a white powder and may have been the same product recently discovered by Dr. Durovic, the efficacy of which was proven by Prof. Ivy of the University of Illinois, (1947-1953). However the occurrence of the substance was not constant, and it seemed that the cows that came up for slaughter at the tail end of the line did not present it like those that were killed first and did not have to go through the agony of smelling and hearing and observing the fates of hundreds of their grass-mates that came to the hammer first. This fact is confirmatory to our hypothesis too, as very likely the adrenalin and amines of decarboxylated amino acids inactivated the protective carbonyl function we will discuss later on. At any rate at times a cancer stimulating substance was encountered. While the curative principle proved to present high carbonyl activity, and the hindering factor demonstrated high amine toxic effects, they fell in with the observations we had published in 1913 on the toxic situation in animals following parathyroidectomy. (J. Biol. Chem. 12; 313, 1912, and 15; 43-63, 1913, Koch.) Some years later vitamin K was discovered and its coagulation function was proven. It also presents a carbonyl group activated by conjugation with the double bonds of an ethylene linkage, and inactivated by sulphides like some of the curative substances extractable from heart muscle. Vitamin K like these substances also shows some detoxication effects.

Our method of extraction aimed to stop metabolism quickly enough to freeze incompletely combusted metabolites "right in their traces" while still held absorbed in the lipoid and particulate division of the functional elements. To do this was very difficult and it became necessary to reproduce the carbonyl activity synthetically for the highest potency possible. This is the phase of the subject discussed in this book.

We investigated the interaction of carbonyl and amine groups in the progress and hindrance of the tissue oxidations, the values of their activating double bonds and the position alpha thereto as a favored point for dehydrogenation to start oxidation chains that serve immunity and function when oxygen is abundant, and which yield to pathogenic polymerizations or additions when oxygen is lacking. In consequence we concluded

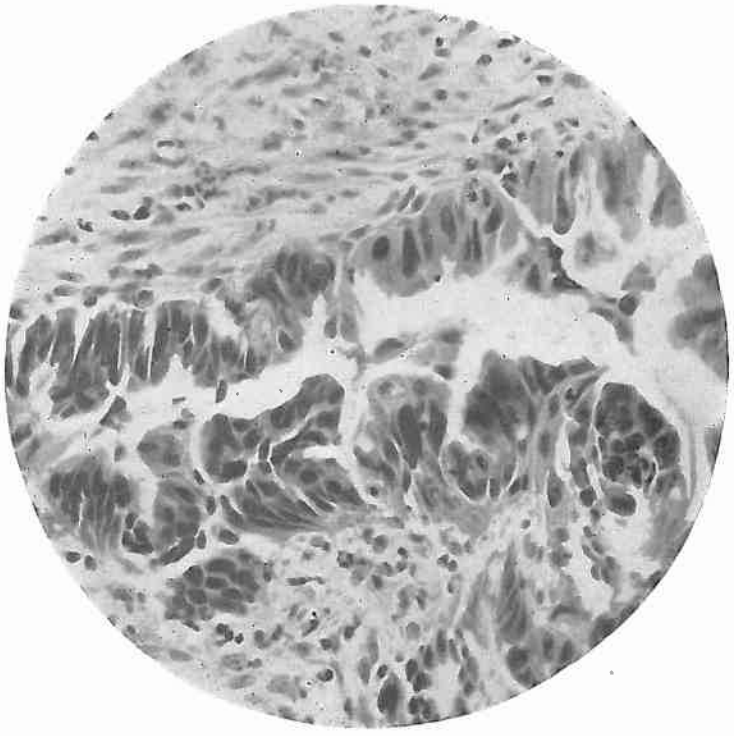
what the atomic bondings must be that constitute the integration of host cells with virus, bacterial filterable toxins, and neoplastic agents, that provide the energy transfers for parasitism, and allergy, or block its production in the anaplasias, atrophies and anergies. It was very evident too that activated carbonyl groups, and activated amine groups offered opposite avenues of antibacterial attack. We decided that the carbonyl group served on a constructive physiologically corrective basis to secure complete and lasting cures in cancer, allergy and infection, while the amine group could only serve through its toxicity, which, as we observed with trimethylmelamine, had to meet the refractory and resistance provisions of the tissues. (Koch, J. Lab. and Clinical Med. 1; 299, 1916). Such resistance phenomena we concluded were general protoplasmic properties that offered the basis for survival mutations that ultimately must make toxic amine therapy more or less a failure. Today this is found to be true as we see that hydrazide and antibiotic drugs breed new resistant types of germs that no longer weaken before them, but thrive on them instead. Today infection, resistant to all antibiotics, is calling for the old fashioned surgical incision and drainage to give the patient any chance at all. Thus trying to annihilate the germ by blocking its carbonyl function, or otherwise poisoning it with activated amine groups is definitely turning out to be the failure we had feared. Besides, the activated amine therapies stimulate neoplastic activity, possibly through activating the ever present fungus phase of the causative organism, which we concluded has to block oxygen transport to give the neoplastic pathogenesis good support. And also the amine therapies do not correct virus infections, but may make them worse.

We decided that constructive therapy that restored the germ to an autonomy where it need not longer be parasitic but could conduct its own oxidations to serve its function in the Great Biological Economy, would rescue it from the pathogenic class and let it serve beneficially as the Creator had no doubt intended. We interpret the increase in the number of staphylococci aureus during the healing of gangrenous mastitis in dairy cattle while the toxic status is rapidly improving, and the increase in the elimination of tubercle bacilli during the healing of

huge cavitations after the toxic symptoms have gone, as evidences of this phenomenon. The clinical demonstrations mentioned later show the tissue cells restored to their unhindered efficiency in conducting their functions whether these have been injured by integrations with parasitic virus or the action of incompletely combusted germ or tissue cell metabolites directly, or indirectly through the fibrosis set up as a defense against these poisons. Completing the combustion in each instance appears to make the pathogen harmless and possibly useful in cleaning up the debris they formerly caused.

Our experience requires the differentiation of two grades of anaplasia. One occurs in non-reproductive cells as those of the central nervous system and retina. Both grades may be shown by neoplastic cells following the preliminary hyperplasia induced by the carcinogen. These are distinguished clinically in the biopsy which reveals their mode of response to our treatment. In Grade A reconstruction of the functional mechanism can take place after the carcinogen is removed, so that normalcy is restored; while in Grade B, the neoplastic cell undergoes a coagulation necrosis with calcification as occurs in the digestion and removal of a blood clot, or in the digestion of casein. (Koch, Medical Record of New York, October 30, 1920.) In Grade A, we conclude, the genes that pattern the reconstruction of cell functional mechanisms are combined with and inactivated by the carcinogen. But though the cell appears anaplastic and gives rise to an apparently anaplastic progeny, some combined and hence inactivated functional mechanism gene material still remains, though in decreasing amount from generation to generation. The gene material may be liberated from the carcinogen by highly efficient carbonyl group activity, and again pattern the reconstruction of functional mechanism material or the functional structure may not be interiorly lost. Plate I. However, after the inactivated genes have all been dealt out without replenishment in the progeny, the time has come when no genes are available for functional mechanism reconstruction. Thereafter the progeny are permanently anaplastic, and may be classified as of Grade B. Here neoplastic cells can no longer function or reproduce after severance of the carcinogen. So they

PLATE I



A. — Adenocarcinoma of the colon, before treatment. (300X)

behave as just so much tissue debris that is removed as stated above. This is shown in Plate II. (From Koch, the Medical Record of New York, 1920.)

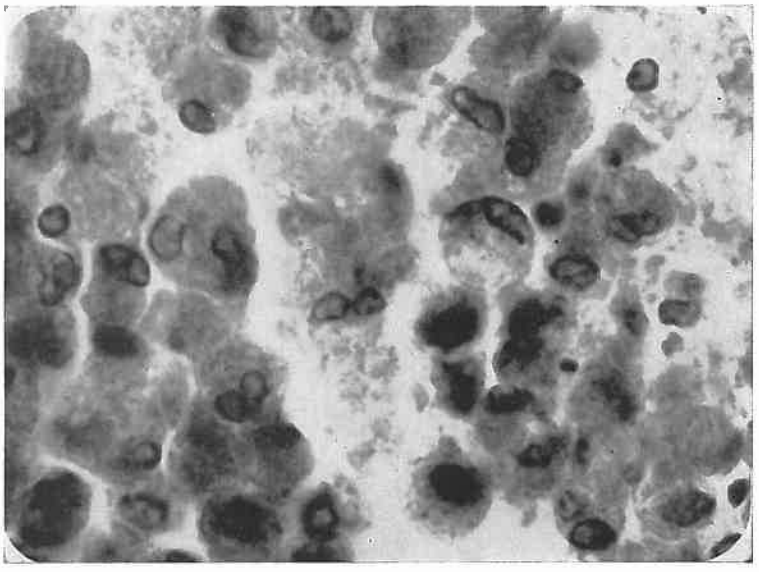
Our hypothesis also covers the pathogenesis of the fibrotic changes in the heart, brain and kidney resulting in the usual fatal diseases. The recovery process here, after our treatment, follows the very unique law of reversal of the symptomatology of the pathogenesis so that the first symptoms to come in the development of the disease are the last to show up transiently and disappear with the cure process. The part played by anoxia in the pathogenesis will be emphasized, and the mechanism of initiating the curative oxidations will be explained. It will then be seen that they are propagated in chain fashion whereby substrate or toxin is converted into carrier or antitoxin

as the essence of the Natural Immunity. (Koch Investigations, p. 34, 1924, and Koch, Cancer Journal, October, 1924, Phil.)

UTILITY

In view of the failure of any amount of antitoxic serum or vaccine to rescue a cell from penetrated or integrated virus or filterable germ toxin, the philosophy we offer here should warrant diligent experimental and clinical scrutiny. This is true since the feat is accomplished by definite atomic structures synthetized with various grades of efficacy at will. The host cell is restored to normal, and stays so through its successive progeny for a third of a century as observations show so far, and the offspring hold the immunity in high degree. The action is immediate as is seen in the acute paralytic stage of Anterior Poliomyelitis, when the paralysis and general toxicity left within hours after the treatment. The curative effect is seen more brilliantly in the chronic symbiotic types of Anterior Poliomyelitis where extensive atrophy and paralysis has existed for as long as twenty years, and within six months after the treatment, the paralysis and atrophy are largely corrected, and in a year they are over 90% normalized, with good function restored. Our Federal Court cases have proven this fact beyond factual contradiction, as the following case histories show. We classify anaplasia of this type as grade A and include with it, the anaplasia of retina and optic nerve atrophy so far as our contact with it has gone, for here too restoration has been complete following the treatment. The same holds for the paralytic type of distemper in dogs, and for the cardiac muscle injury in the fatal types of Hoof and Mouth Disease in cattle where in some series so high a percentage of cures as 100% have been obtained on one injection, while the untreated controls all died of heart failure. Here however we deal with cells that can repair by reproduction, and the proofs are not so undebatable as where non-reproductive cells as the anterior motor cells of the cord, and the neurones of the optic nerve and retina are integrated with the virus or toxin. In the latter case the restoration cannot be accomplished by cell reproduction, but only by destruction of the virus and the freedom of the host cell from all hindrances. Where cell reproduction does not offer the necessary

PLATE I



B. — Shows the operation of the functional genes giving the apperance of Mocoid Cancer. (1500X)

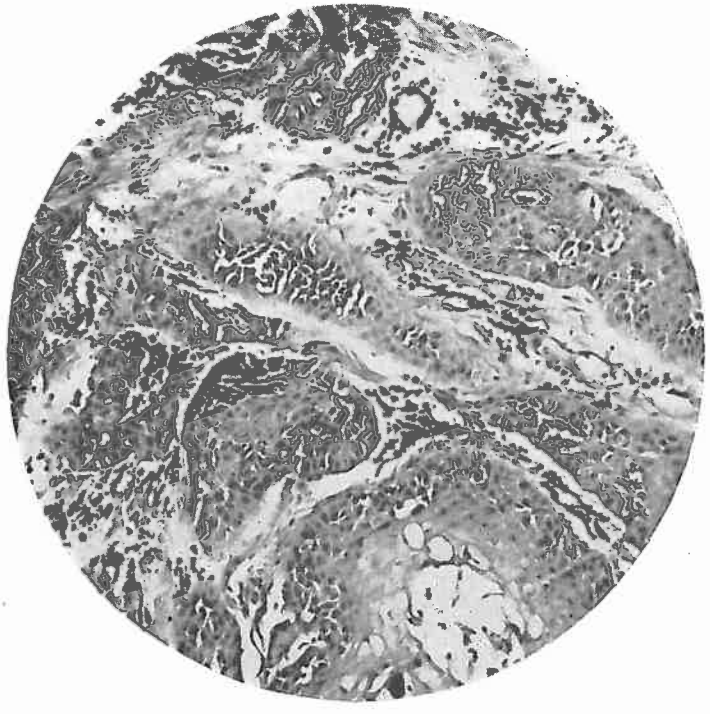
advantages, restoration is made possible, we believe, through the Nissl Substance, a most wonderful survival provision granted by the allwise Creator. Moreover, since the detoxication or deviralization is accomplished by highly active carbonyl catalysts of oxidation, we must conclude that the essential pathogenicity rests with the suboxidized state of the interfering agent, and had this been adequately oxidized at the start, it could never be pathogenic. Complete combustion prevents the pathogenesis, and complete combustion must be induced to correct it.

STATISTICAL PROOFS OF UTILITY

Cases Still Under Treatment When Count Was Made

In a survey covering 19,532 cases of various disease conditions treated with the serially arranged carbonyl groups by 88 American physicians, 51% of the cases treated were reported to have recovered, 35% of the cases showed improvement and 14% of the cases showed no beneficial results. Among the disease conditions reported on were tuberculosis, paralytic in-

PLATE II



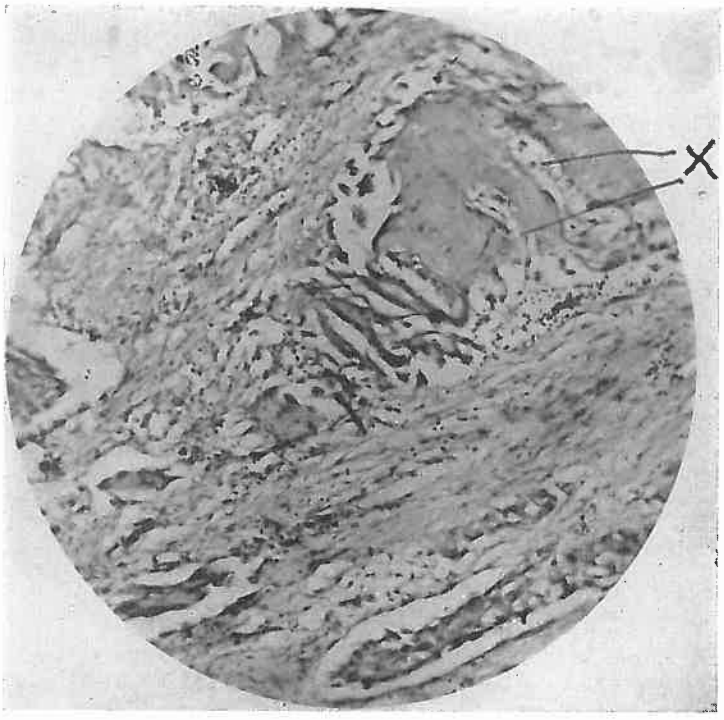
A. — Squamous cell carcinoma of the neck biopsy before treatment. (150X)

fantile paralysis, cancer (including far advanced terminal cases), arthritis, cornary thrombosis, undulant fever and allergies. A second group of 7,317 cases of various disease conditions treated with a quinone structure showed a recovery in 64% of the cases treated, improvement in 23% of the cases and no beneficial results in 13%. Among the disease conditions treated were arthritis, asthma, rheumatic fever, allergies and infections.

Statistics Collected Three Or More Years After Treatment. Not Cancer Cases

To show the average results obtained in a well conducted practice, the following statistics are taken from a report given before a group of collaborating physicians in 1949. Dr. W. H. reports as follows—Up to June 1949 he had treated 2,448 pa-

PLATE II



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.

tients using a total of 4,160 doses of the carbonyl catalyst given by injection, and of these he selected 1,926 cases that had been treated more than a year previously to calculate his statistics. Of these, 937 were allergy problems, and 767 acute and chronic infections. They averaged one and one half injections each. For the statistics on some of these cases see the table on page 26.

It will be seen in this table in some categories the failure percent runs as high as 22%, and in one disease it reached 50%. Thus obstacles hindered the correction of the basic pathology. For such obstacles to interfere with the induction and the evolution of the antitoxic state of structure of the pathogen, it is

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 qrs.	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
Syphilis 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

necessary for them to prevent the production of the free radical by dehydrogenation of the pathogen, or if the free radical has been formed and oxygen is present, the interference must block the production of the peroxide free radical. The agencies that can accomplish this interference are discussed as we go along, and the advantage of being able to supply a peroxide free radical to act on the pathogen will soon become obvious. During the last decades of this research the attempt was made to produce a peroxide free radical bearing molecule that would serve to initiate the oxidation chain, but only recently have we been able to succeed. Explosions and instability in aqueous solution

prevented progress. The material used now is also too unstable for commercial service and must be used soon after it is built up. So far at least the efficiency is promising and the percent of failures can be reduced considerably. More time is needed to know the percent gain in permanent cures over those of the past.

Official Test On Cancer Cases

Our one and only official test was conducted by the American Medical Association's Wayne County Medical Society in 1919, at Detroit, on seven officially selected cases of far advanced widely metastasized, cachectic victims of cancer of the vital organs. The patients were brought from distant cities and another state, although seven cases could have been easily picked up at any Detroit hospital charity clinic. Three weeks after the treatment was given when the first real signs of improvement showed, the official committee suffered a panic, closed the investigation, and sent the patients back to their distant homes, denying them further treatment. They then hastily reported "NO RESULTS" and broadcast world-wide, that the treatment was a "fake", just as they did with Glover's treatment six years later, and with Durovic in 1950.

Of the seven officially chosen cases, at least three, and possibly a fourth, were cured, but as the fourth lived so far away, it was not possible to follow his progress very long. Of the three, one lived fifteen years in perfect health, died from an accident that caused a cerebral hemorrhage, and was cancer free as proven by the coroner's autopsy. She was brought in for the test from Toledo, Ohio, on a stretcher with an enormous adeno-carcinoma of the uterus, that had spread throughout the abdomen and caused severe gastric hemorrhages. Another equally advanced case with abdominal metastases and gastric hemorrhages, brought in on a stretcher from western Michigan, was found to be free of cancer five years later, and in perfect health. Another equally advanced cancer of the cervix with extensive abdominal involvement and terrific pain, was met on the street in Toledo, Ohio, her home, several years later and she stated she was perfectly well, without any tumor or bladder compression, no pain, or bleeding or any trouble what ever. No ex-

amination was made by us, as she appeared perfectly well, and other physicians found her so as she reported. At the time of treatment her life expectancy was less than a year.

In 1923 and 1924 we asked the American Medical Association and its Wayne County Medical Society to change their false report to agree with the facts, and to permit another official test. But they refused. No other official test has ever been made to date.

Court Testimony Cases of Common Diseases

In this book we use many cases that were used in the United States Federal Courts and before the Federal Trade Commission. The cured cases include far advanced widely metastasized cancer of the vital organs, far advanced cavitary tuberculosis, fulminating infections that refused to yield to modern methods, intractable allergies and arthritides, acute rheumatic fever, Anterior Poliomyelitis in the acute, subacute and long standing paralytic phases with atrophies, and the ordinary diseases of dairy cattle.

Many of the diagnoses were made by the leading experts of our nation, at our highest rated institutions, where the patients were held at residence under observation so that all necessary facts could be determined to make a firm diagnosis. These institutions include the Mayo Clinic, The Henry Ford Hospital of Detroit, The University of Michigan Hospital, The Detroit Tuberculosis Sanitarium, The Herman Kiefer Hospital, and others of equally high standing. In fact there are no better authorities in America from which to select expert diagnoses. The cured patients and their physicians gave full uncontradictable factual testimony. These case records are also exceedingly instructive, and have therefore been preferred for illustrative use.

CONTROLLED ANIMAL EXPERIMENTS

Statistical and other information is afforded by the following tumor transplantation experiment done by Dr. Stanley Bandeen, of Louisville, Kentucky, and reported to a group of collaborating physicians. The observations started with inoculations of transplantable C 57 Breast cancer into mice at the

Jackson Memorial Laboratory of Bar Harbor, Maine, on May 7, and the inoculation of transplantable Sarcoma 37 on June 30, and terminated by a frost that killed most of the survivors on November 13, 1950. Some of the mice were killed by fighting. These are excluded from the cured statistics. The reagent used was serially arranged carbonyl groups in the 12x homeopathic dilution.

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: 134 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 cured on the 110th day and other on the 125th day.

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 18th day to the 23rd day. One mouse died cured the 83rd day. One died cured 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 cancer cures 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 minims, 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 for 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.

CHAPTER II

THE SCIENTIFIC BASIS

In 1910 I made observations on the toxins produced in the tissues of dogs after complete parathyroidectomy, and reported the findings in the Journal of Biological Chemistry in 1912, vol. 12, p. 313, and in 1913, vol. 15, pp. 43-63. The work was then followed at the University of Glasgow by Paton and his staff for three years and reported in the Quarterly Journal of Physiology, in 1917, vol. 10, Nos. 3 and 4. For the excellence in which they confirmed my work they were awarded the Triennial Prize in Medicine by Harvard University. This confirmation is important since the deductions which I was impelled to make on these findings resulted in the practical values I will report here as case histories, in the cure of atrophic paralytic diseases, both motor and sensory, and in the true cure of neoplasia in many of its various forms.

The principle findings after parathyroidectomy were excessive production of several guanidin bases and their elimination in toxic quantities in the urine, together with excessive amounts of other amines, creatine, creatinine, lactic acid, phosphate and calcium. The autopsy findings were ante mortem coagulation of the blood in the large veins, hemorrhagic hepatitis, and hemorrhagic glomerulitis, ending up with anuria and death in convulsions. The extensiveness of the convulsions and their severity was proportional to the amount of guanidin and of lactic acid eliminated even when the lungs were well ventilated. The symptoms were the same as one could produce with injections of guanidin in toxic amounts, so we held that guanidin was a key instrument in producing the functional disturbances and the pathological changes.

It seemed reasonable to conclude that the excessive amounts of the normal product of metabolism, *creatine,* that were eliminated in the urine, were displaced from their position in the tissue molecular set-up by the guanidin. We still hold this view. This is important since the type of creatine union with the cell,

and its function as displaced and blocked by guanidin is a key consideration in the whole pathogenesis that we have to discuss here. We decided that creatine was held combined with a highly active carbonyl group of the energy producing mechanism by an azomethine condensation in the performance of its normal function, and that guanidin displaced it permanently destroying that function. As a result phosphoric acid and calcium were not used and were eliminated as waste materials too. The ante mortem clots were to be interpreted as colloidal gellations due to reduction of surface charges from the colloidal particles in the sense of MacDonagh of London. Such gellations occur in cancer and syphilitic infection and reduce oxygen transport between and within tissue cells and cause a local anoxia basic to pathogenic change. It is a result of the guanidin intoxication after parathyroidectomy and can be reproduced by guanidin injections. Lactic acid production, in proportion to the convulsions and guanidin output, indicated that the tissue energy was supplied by hydrolytic glycolysis to produce lactic acid even when the lungs were forcefully ventilated. Lactic acid also accounted for much of the calcium that was eliminated. As the guanidin output increased, every function took on allergic qualities. **As a result of these and further observations we arrived at the conclusion that the energy production mechanism of each aerobic cell has to include a highly activated carbonyl group conjugated with the double bonds of an ethylene linkage from which it received a quota of electrons which made it highly basophilic while its activating conjugated double bond became more electrophilic. This conjugated system of carbonyl and ethylene groups with a carbon atom carrying a highly mobile hydrogen alpha to the double bond, constitutes the fundamental vital unit, or the simplest atomic arrangement that supports life, and plays a part in the fundamental changes that constitute all disease so far as our experience has gone.**

We assume that the carbonyl group dehydrogenates fuel substrates or toxins that come into the field to form in them free radicals which combine molecular oxygen to form peroxide free radicals, and these peroxide free radicals serve as carriers of a chain oxidation which combusts the fuel ultimate-

ly to carbon dioxide and water, and converts each molecule of toxin to the same peroxide free radical antitoxic type of structure, until every molecule of toxin is oxidized out of the way. This was our idea of the Natural Immunity Process wherein toxin is changed to antitoxin as a chain process. This we suggested in Koch Investigations 1924, p. 34, and in the Cancer Journal, October, 1924. Evidences are now accumulating to support this early conclusion.

Creatine phosphate was unknown when we first found the guanidin poisoning that followed parathyroidectomy. Here the phosphoric acid is condensed with the amine group instead of with a hydroxyl as in the other phosphorylated energy storing and releasing compounds. This is a high energy bond carrying some 13,000 kilocalories, and the lability is largely due to the double bond of the imide group with which it is conjugated. By the action of a transphosphorylase the phosphoric acid and energy of equal amount is transferred to adenosine diphosphate (ADP) to form the triphosphate, (ATP). The ATP combines with actomyocin, releasing energy to it and liberating a molecule of phosphoric acid. The energy taken up by the actomyocin changes its viscosity and the shape of its particles, it is assumed, to produce motion.

Every cell has its energy pool, just as it has its nutritional pool. The energy pool is carried in the esters and condensates of ADP, ATP, Creatin Phosphoric acid, and dozens of other compounds no doubt. We take it that the virus can make its combinations here as well as with the energy generating mechanism as we describe it in this text, and hence the atomic units concerned are much the same in both. Thus the Nissl substance of the nerve cell may be so considered, and when the polio virus attacks, it combines with the Nissl material first. If this holds out until all virus is inactivated, the rest of the cell is not attacked, and a non-atrophic and very transiently paralysed or weakened muscle may result. But where the Nissl substance does not hold out, as is true after too much activity with its nerve exhaustion, then the virus integrates with the energy producing mechanism and destructive disease has thereby been established. What holds for a virus holds also in general for

metabolic products and bacterial or synthetic products that are not fully combusted and invite dehydrogenation as via an alpha placed hydrogen atom with reference to a double bond, or one that is "peri" placed. Thus the combining free radicals are produced. The potentials of energy storage and release are a big chapter also.

The phosphoric acid liberated shows how much energy has been taken out of storage, and that its carrier is free to take on more. So we assume it goes to the azomethine bond and causes its separation, which is indeed very easy, and then the carbonyl group is ready for another dehydrogenation to set another oxidation chain going, so more energy is produced to make the condensate between creatine and phosphoric acid; and while this is handing over its load to ADP, the hydrogen is being taken from the carbonyl group by some oxidase and the creatine and carbonyl group can again condense with some energy storage, waiting for the next hydrolysis via phosphoric acid. In this cleavage the energy released helps fix the phosphoric acid to the creatin carboxyl group until energy evolved by the oxidation chain can form a high energy bond between it and the amino group. The phosphoric acid released by the transphosphorylase has the calcium action to keep it from breaking the N-P bond until it can react on the azomethine creatin bond. Such matters are hypothetical applications of known activities. These we must extend to picture both the antagonistic and conjugate actions of amine and carbonyl groups, and the factors that control both. Even now very little of what is needed to be known of the nature of the participants and how they react is well understood. Forty years ago practically all was blank. All we had available was the discovery of the free radical by Gomberg, my chemistry professor, and no one applied it to biochemical processes for many a decade. Today we are in a different position. In line with our hypothesis we may assert with confidence that the ketosteroids function through their carbonyl group as activated by the double bonds with which it is conjugated in ring A. This is proven by the fact that the spent products of their action (dehydrogenation), present hydroxyl instead of carbonyl attached to ring A, and the double bond is saturated. Corticosterone

would be expected to condense, not with creatin but with adrenaline*, and Testosterone with spermin, to regulate energy supply for muscle and sperm fibril contractions as a chain process. As the steroid is bound to the F. M. at C17, only very minute traces are required to co-function with more material doses of the amine in rapid turn-over (see diagram VI). We consider this type of mechanism to be used quite generally also by the lowest forms of plant and animal life for energy liberation for motion, such as the mechanism whereby crocin activates motion in algae sex gametes, and Echinochrome (A) activates motion in the sperm of the Sea Urchin. These substances were demonstrated to act in dilutions higher than one to a billion by Kuhn and Moewus, and Kuhn and Wallenfels (1938-1940), as well as by Prof. Gilbert Smith in 1947. Such is *physiology*. For further discussion see the appendix.

Inspection shows Crocin to be a Carotenoid presenting two carbonyl groups conjugated with seven ethylene linkages each of which receives electrons from a methyl substituent that mesomerically migrate to the carbonyl groups, thus activating them. Echinochrome (A) is a naphthoquinone with two activated carbonyl groups and is quite similar to vitamin K in structure. In line with our hypothesis they may serve with specific amines for energy production and utilization as described above. Vitamin K combines the structures of both the quinone and the carotenoid, the animal type and the plant type, described above, and we propose, in like manner supplies energy with the aid of other carbonyl compounds and amines for motion in the leucocyte and to provide surface energy to preserve the dispersion of the blood plasma colloids, so that when this is inactivated by an amine of strong azomethine bonding, as guanidin in our parathyroid work, or when the energy is removed by a foreign surface, the plasma colloids gellate. The latter situation creates anoxia so that the free radicals formed can no longer serve the oxidative detoxication, but are free to copolymerize with the

*The structure of adrenaline offers opportunity for removal of the methyl and hydroxyl groups right in the tissue where it is destined to function and thus create a double bond that should activate its amine group and facilitate the azomethine condensation. The specificity for involuntary muscle fibers of the blood vessels should be examined from this standpoint.

collagenous units that form the fibrin, and thus enter the clot, in storage as it were. The detoxication function of vitamin K is demonstrated by various observers and we assign this property to the conjugated system of carbonyl and ethylene double bonds it contains, but as their oxidation reduction potential is weak, they are not very effective. Still they serve by inducing oxidations in the incompletely burned germ and tissue metabolites in line with our hypothesis. The cure of coronary, renal, and cerebral deficiencies due to the fibrosis of hypoxia and its resulting occlusions, can be accomplished as we showed decades ago by the timely use of highly activated carbonyl groups. Our hypothesis, clinically proven, is each day receiving new support from each new examination of a pertinent physiological phenomenon. Case histories will demonstrate the reversal of the pathogenesis in vascular occlusions of the heart, kidney and brain, as well as the quick detoxication of partly burned products of metabolism of tissue cells and germs, as well as those produced by steam and fire burns, with quick relief and healing. This will be given thorough discussion in the completed text, where it will be shown that the potential of action of the carbonyl and amine groups determine health when oxygen is present.

The Virus Situation

Well established facts in the demonstration of the infectious agent in cancer, perfectly following the four rules of Robert Koch are reported with conflicting features which appear to be readily harmonized by our working hypothesis. The splendid work of San Felice two thirds of a century ago, and of von Brehmer and of Glover a third of a century ago, and recently of Gerlach, Irene Diller and also Wuerthel-Caspe, using modern facilities of technique, confirm the pleomorphic nature of the infectious agent, reported as being coccus, bacillus, filamentous and filterable virus, in its various cycles of life. Gerona besides showed it to be a coccus that withstood boiling at 120° C. Thus with a filterable phase as a sure transmitter one may assume that an adapted virus, living in symbiosis with the bacillus, the fungus filament, the coccus or spore of any of the foregoing, is the active agent, and since it has a predilection to liv-

ing in the symbiotic state as presented in our diagram IV a, it is part and parcel to the cancer cell and not filterable from it until it dies, having also the identical progeny yield with the cancer cell when infecting epithelia. When integrated with a spore it shares its resistance to heat, irradiation, and chemicals. The type of union with the host cell will determine if a tissue atrophy or a neoplastic response will follow. This will be diagrammed farther on. When the reactive groups of the virus are free, its delicacy is understood, but when it is bound by absorption to some protein surface, or a union takes place by condensation of the virus amine group with a carbonyl via an azomethine condensation, it is inactivated and protected until the azomethine bond is split, and that is easy by hydrolysis often via weak acidity. But it may be separated and inactivated by oxidation of the carbon atom alpha to the azomethine double bond, with comparative ease because of the high mobility of the hydrogen atom there attached. Probably the first reaction observed in virus is the inactivating condensation with formaldehyde to produce a vaccine. Probably the most reactive carbonyl group in the tissues is that which initiates oxidation chains for function, and this would be the first for amine attack. This amine condensation of the virus with the host's carbonyl group must block its activity so as to not only throw the energy producing mechanism off the oxidation path and onto the hydrolytic glycolysis path, as we diagram later on in Diagram II, for guanidin or other activated amines, but also serve as hook up whereby the virus received vital energy. When the functional mechanism is crippled so as to not be able to accept the energy produced by the glycolysis, this energy has no other place to go than to the attached virus and the mitotic mechanism, as explained farther on, to support the neoplastic virus symbiosis. This type of integration to cause neoplasia could take place during the presence of oxygen, if anoxia existed only during the moment the azomethine bond was being formed, that is, during the initiation phase, only. Obviously this type of integration is easily broken by highly active carbonyl groups, and while the virus is protected and nourished during the neoplastic response. After it is oxidized away, it is no longer pathogenic as we will show. The

filtration of virus from epithelial tumors is therefore not so easy, and the progeny yield probably equals that of the daughter neoplastic cells. But when the virus is liberated from a cancer cell because of its death, the pathogenic properties remain. However when the integrations are outlined according to their most likely provisions, the virus is separable by oxidation through a vigorous dehydrogenation, free radical production, peroxide free radical formation and molecular cleavage with carbonyl terminals being formed. So provided, the virus is no longer toxic or pathogenic and its amine group is deactivated.

Possible Origin of Virus

In line with our thesis we hold that no comprehensive biological response can take place without involving the phenomena of the free radical and the double bond. This should be especially true where oxidations provide the energy for the response. We must therefore see how these phenomena could possibly play a part in the origin of the virus with its deficiency that calls for parasitism to secure survival. The deficiency indeed may also involve the protein structure and amino acid content which will produce a specific serology.

We hold that bacteria and fungi, like the matured virus, are polymeres of nucleic acid protein complexes, analogous to the nucleic acid protein complexes of tissue cells which can be depolymerized to similar structures of lesser molecular weight. When during their autonomous metabolism, conditions of anoxia and nutritional difficulties prevent the carbonyl group that accepted the hydrogen from being freed of its accepted hydrogen, and the free radical formed by the dehydrogenations meet no oxygen to combine with, then these free radicals are open to addition to the most active double bond at hand, and thus set up the polymerization chains resulting in complex conglomerates which are chemically and thermally resistent to activity since their reactive groups are already occupied and further reaction is excluded, until a depolymerase of specific nature can act under favorable conditions. Then the non-reactive affair which we may call a spore is split down to its ultimate living units, each of which is a semi-solid and filterable autonomous particle of much less molecular weight. Each is provided with a

carbonyl set-up so it can produce energy for its maturation, and it develops into a body similar to its parent. This may be a bacillus, a fungus, a coccus, rickettsia, or matured virus that is not parasitic, but serves the great biological economy. Such a creature has never been identified since we know virus only by the disease it causes. Since many ultimate units are formed by the desporization, the progeny is large. Oxygen is necessary for the process, as it is for the maturation of each unit. The procedure facilitates adaptive mutation against the conditions that caused spore formation, and the situation is physiological and what one would expect.

However, when such a depolymerized unit gets into a tissue cell where anoxia prevents the peroxidation of the free radicals formed by the host cell, **in it,** or formed by its own carbonyl group acting **on it,** this free radical cannot form a peroxide free radical, and it is open to additions to the closest highly reactive double bond at hand. This position is the host's carbonyl activating double bond. Since its carbonyl group is inactivated by the anoxia, or if it is condensed with an amidine as guanidin, it becomes parasitic automatically on the host, through the union with one pole of this double bond, the other pole of which attaches to the hydrolytic glycolysis system we assume. Then it may follow one of the two courses outlined and diagrammed below, to produce either total host cell lysis with rich progeny yield, or, divide the energy equally with the host cell to produce equal progeny with it numerically, as a neoplastic process. On the other hand the virus may unite with the two poles of the activating double bond so that the hydrolytic glycolysis system is excluded and no energy goes to the virus or to the mitotic mechanism or to the functional mechanism. There is neither neoplasia nor allergy of any function then, but an inactivation that may lead to death without virus progeny, or to a pseudo-mort symbiosis with resultant paralytic and atrophic sequelae, that is curable. Pathogenesis then depends upon the coincidence of several factors, among which anoxia or hypoxia is important and determinative.

One great characteristic of pathogenic viruses is their ability to mutate in a new environment so as to survive with

the incorporation of the adapting qualities newly acquired as permanent hereditary characteristics. They thus can acquire a new specific serology just by being grown in a medium with an unusually higher added content of one or two amino acids. Or they can become fixed in their adaption to one tissue if transplanted over and over again in that tissue. In view of our simple hypothesis of origin, we may assume that when the sprouting unit finds itself through a series of generations growing in the same host and under the same conditions, it can become fixed in its nature and thus become more or less highly specific chemically, biologically and structurally. So far as knowledge of virus goes, there is nothing to exclude the existance of many more normal useful nonpathogenic types of virus than the pathogens we know about. Our idea that virus is synonomous with the acme of parasitism will have to change some day since the steps that could make an autonomous virus parasitic are rather simple, as well as subject to reversal by introducing efficient carbonyl catalysis. This simple idea of the sprouting units of a spore giving origin to a virus, which may later become fixed in its deficiency —through parasitism favoring conditions, may be "all off." However, it may explain a natural behavior too, as the pleomorphic germ of Glover and of von Brehmer and others that are well confirmed now, and at least it offers a simplification, as to the origin of the virus phase. It may explain the association of virus with a germ as in hog colera, tuberculosis, etc.

Accessory carcinogens as the polymerizing acrylic aldehydes offer free radicals that may add to a host double bond in its energy producing mechanism and lay it open to union with a carcinogenic virus or synthetic structure, and the free radicals formed from chloroform and carbon tetrachloride may do the same. But for any such pathogenic act, oxygen deficiency is essential, which is easily secured by circulatory injury, blows, rough physical examinations, etc., and by injury to the oxygen carrying power of the blood produced by coal tar medications as the salycilates, barbiturates, aspirin, etc. Cardiac insufficiency, and obliterative endarteritis are obvious accessories, but smoke laden air with the mucous generated in the lungs by way of protection, block the entrance of oxygen to the blood stream.

Hypoxia is the basic factor in the initiation phase. What follows depends upon the free radicals left uncombined with oxygen to **not form peroxide free radicals,** that carry the oxidation chain forward, but make pathogenic additions instead. Molecular oxygen is concerned, not ozone or atomic oxygen. It is only when the normal function of oxygen is prevented that the interaction of free radicals of one party and the double bonds of another follows to initiate pathogenic states. One sees that molecular oxygen by forming peroxide free radicals is the buffer against disease. Here too is an indication that the Krebs' Cycle is not the procedure of first choice in the tissue metabolism.

The Rupture Of The Symbiosis

The test for the rupture of the symbiosis of the virus with the host cell must be made on a tissue that is not able to undergo compensatory hyperplasia. This is the situation in nerve parenchyma. So we made our tests in both the sensory and the motor paralytic anaplasias or atrophies as they are called. It will be seen that what holds for virus also holds for the polymerizing free radicals of incompletely combusted germ and tissue metabolites produced under hypoxic conditions, and for their co-polymerizing free radicals, also, which can combine the carbonyl group's activating double bond in the same manner as the virus, to produce a neoplasia or an anaplasia or some disturbance of function as an allergy or an anergy. Clinical observations from several angles call for such an explanation. So the separation of the virus or of the toxin of germ or tissue cell origin from the nerve cell is demonstrated where function returns and atrophies give way to muscle reconstruction, after an established paralysis with atrophy, be it motor or sensory. Besides in the retina, the reconstruction can be directly observed. After a long standing paralysis from "Polio" with profound atrophy, the restoration of function and good muscle is demonstration enough, but we have observed the same in dogs with paralytic distemper, in one horse and in one man afflicted with paralytic rabies secured through coyote bite, and in cows with the cardiotropic form of hoof and mouth disease, where the blocked functions returned to normal. The case of optic nerve atrophy like others of the same type given below may be studied

with interest. The interactions will be diagrammed a little later. The inactivation of the functional carbonyl group by condensation with an amine group of a virus, or toxin of germ or other origin must also be considered. The Pasteur Effect is thus annuled and clinical fever produced. It will be seen that this carbonyl group controls energy production, and the oxidations of the natural immunity. We conclude it controls and is the key to the Pasteur Effect.

Hyperplasia and Energy Transfer

The mitotic mechanism exists to supply cells by reproduction that will provide functional mechanism adequate to the demands for survival. Thus when a functioning cell is worn out trying to produce the work required which is above its capacity, the functional mechanism is broken down, and energy cannot flow into its chemical processes, as it has none. So the only other direction the energy can go is to the chemical processes of the mitotic mechanism, setting them to act. The result is cell division, and compensatory hyperplasia.

We also consider the functional mechanism as a specially differentiated structure in which genes pattern the working parts. Apparently, these genes are not worn out by work, and can shed their broken down functional debris and enter the mitotic act as a pattern basis for new functional mechanism construction. Thus where excessive work has called for additional working mechanism, it is shown by functional mechanism destruction that can only be repaired by new cell production. In contrast, fatigue from severe exercise of the function provides for its cessation through oxygen want and the lactic acid production of hydrolytic glycolysis which inactivates the ferments necessary to work. In this way fatigue has an automatic block to excessive tissue destruction, and the pain caused by incompletely burned irritant products of metabolism, puts an end to the injurious overwork too. Still too much forced or repeated effort is destructive enough to call for more functional material so the adaptation is actually provided by the course the energy takes toward the mitotic mechanism after the functional mechanism is not in condition to receive it. We may diagram the procedure as in Diagram I (a), p. 65. Here, the normal relations

of Functional Mechanism, (F. M.), Mitotic Mechanism (M. M.), and the energy producing mechanism (E. M.), also includes a phosphorylation provision, (P. P.) operating with a labile amine, somewhat as we have described. Arrows show direction of energy flow. In (b) the F. M. is broken down and requires restoration with sufficient increase to serve requirements. In Grade B, the cancer cell F. M. loss includes its genes so it cannot be reconstructed during cell multiplication. This is the difference between the anaplasia of the cancer cell and the dissolution of the functional mechanism of a normal cell caused by overwork. When complete, the anaplasia of the former is not correctable through cell multiplication, while the defect caused by exhaustion of overwork under normal conditions is correctable by cell reproduction until the demands for functional material are satisfied. A different provision for survival is offered by non-reproductive nerve cells as we will show. This loss of F. M. gene activity in cancer cells is a fundamental characteristic, as we see it. This may simply be a loss through union with the carcinogen, and thus be correctable by adequate oxidations that burn the latter off. Or after many cell divisions it may be a true loss of the genes themselves, which are shed because of their uselessness when so combined. Such cells are not reparable.

The Restoration of Function

Before accepting our criterion as to the decisiveness of the test proposed to determine the curative separation of the symbiosis of nerve cell and virus, we must examine the situation in the nerve cell in detail. Tissue cells can reproduce to perpetuate their structure and preserve the adaptation and survival of the organism. Nerve cells cannot reproduce, so they possess a different type of survival mechanism. This we hold is the Nissl substance. We hold that the Nissl substance is a nucleoprotein much like the energy producing and functional material of the cell, but that it is not a reserve of food material held in position to enter the mechanism when the latter is worn down and needs replacement of parts. It is a substitute for the replacement of functional material by reproduction. Therefore it carries the conjugated system of carbonyl and ethylene double bonds, and when the virus enters the cell it can make its additions here.

The virus is thus neutralized so long as Nissl material is at hand, but when this material is exhausted, the excess of virus can join up and integrate with the functional material which is actually the living substance of the cell. Here we have the explanation of the influence of fatigue in producing the serious degenerative and symbiotic types of Poliomyelitis, and other virus infections of the central nervous system. The exhaustion opens the functional structure to attack. The non-paralytic cases of "Polio" are those in which Nissl substance neutralized the virus completely or practically all, so that if any functional structure was attacked it was in a minor degree. Reconstruction in such cases does not involve the replacement of nerve cells, but of restoring the Nissl content, and of a minor part of the functional mechanism by using Nissl material. This is accomplished in less than a month as a rule. Therefore any reconstruction and restoration of function coming after a month, must be had by an active curative process which must first separate the integrated virus from the living cell's functional material. This may take place in the symbiotic type of case months or even as long as three or twenty years after the paralysis and atrophy were established. For the symbiotic state may destroy function and result in atrophy of nerve and muscle without actually killing the motor nerve cell, and this condition may be drawn out for many years before the host cell finally dies. Scheme (b) of diagram V, represents the situation as we see it. But the lytic type of integration as represented in (a) of diagram V, generally shunts out all vital energy from the host cell vital processes so it dies quite early and of course is not replaceable. The atrophy and paralysis consequent thereto is incurable.

The rates of restoration of the Nissl involvement and the early phase of the lytic involvement requires only hours, as the virus is subject to quick oxidation by the therapy employed as it is hooked up with the host cell. Thus the paralysis that started twenty-four hours previously leaves in twenty-four hours or even less, as the case histories demonstrate. But the symbiotic type of integration may take twelve weeks to be completely corrected with restoration of muscle and of function in

a case of three years of paralytic atrophy; and it may take from six months to a year for correction to 90% of normal after 20 years of extensive paralysis and atrophy of both legs from hips to toes. This is illustrated here also. Where anterior horn cells were killed before treatment was given, no amount of treatment will restore them. However, the fact that paralysis of longer than a month's duration with the atrophy that was consequent to it is corrected as we show here, demonstrates that the virus is actually removed from the host cell and the latter is free again to reconstruct and function again. This is an accomplishment of which we are humbly proud, as it is done along physiological lines and no other system has ever been able to accomplish this feat. What has become of the virus? It gives us no information. Perhaps it too receives a carbonyl group conjugated with a double bond as a result of the peroxide free radical production at the position alpha to this double bond, and thus it may be restored so it can initiate its own oxidation chains. At any rate it is no longer pathogenic like the staphylococci and streptococci that produced the mastitis in the cows, reported later, and which either decreased in number during the healing, or even increased in number, if much tissue debris was to be removed while the healing and general detoxication of the animal was progressing rapidly.

Where the virus becomes part and parcel to the host cell with its units distributed throughout, and maybe even displacing host cell molecules, the integration is such that it must share the reactivities and properties of the host cell, be the latter tissue cell or bacterial in type. It will thus gain resistance to heat and chemicals when integrated with a spore of a fungus or germ. It will be divided during the mitosis or fission of its host cancer cell as part of that cell, and be liberatable only on the death of the host cancer cell. This type of integration will contribute the protein serological properties to the host cell which the particular breed of virus possesses. On the other hand a carcinogen of chemical origin as the phenanthrene series, or the free radicals of trichlormethane, dichlormethane, acrylic aldehyde polymeres, and incompletely combusted tissue cell and germ metabolites that polymerize in hypoxic foci, have no

protein content to serve serological identification. So two divisions at least can be made which will fit our diagram. The state of union shown here also explains the difficulty in obtaining infective filtrates from epithelial neoplasms, as well as some other puzzling features.

Energy Transfer

The carotenoids, Crocin, Vitamin A, Rhodopsin, and some others with multiple serially arranged conjugated systems of ethylenic double bonds, each enriched in electrons contributed by methyl substitution, offer splendid structure for energy absorption and transfer via fluorescence. This was our idea of the action of the visual purple where radiant energy was absorbed by the mobile electrons of the double bonds and passed on to the chemical processes of nerve cell excitation in the retina. We attributed the energy transfer for carcinogenesis to similar fluorescent processes of the carcinogen where the energy was absorbed from exergonic reactions going on in the field, hydrolytic glycolysis for example, and passed on into the chemical reactions of mitosis and cell division. The intimate hook-up of the carcinogen with the hydrolytic glycolysis system affords the opportunity for the energy transfer. (Koch, Natural Immunity 1936). Crocin and Echinocrome (A) offer the same facilities for energy transfer to the algae motion mechanism and to the Sea Urchin sperm taken from the oxidation process described above, where phosphorylation may also serve as intermediary. This provision seems to be a general affair since virus appears to take up a position between the energy pool and the functional mechanism which it excludes. The rate at which it can take up energy as compared with the rate the host mitotic mechanism can absorb it will determine the survival and progeny rates of both. Some of the enigmas of carcinogenesis can be answered by our fluorescence theory as they can also be explained on the basis of the diagrams given here. Thus when two differently constructed carcinogens of different polarities and different dilutions are given at the same time, instead of boosting their activities mutually, they may interfere with them by combining, each with a pole of the carbonyl group's activating ethylenic linkage, and thus exclude the hydrolytic gly-

colysis system from supplying the energy required for mitosis. It will be recalled too that saturation of this double bond also inactivates the carbonyl group so as to reduce its powers of dehydrogenation and oxidation chain initiation. Of two types of chemical carcinogen exhibiting different polarities, each will select the double bond pole to which it has most attraction, and hence even small amounts of one carcinogen can inactivate a part of a large amount of the other in this way. Likewise, too large a dose of a carcinogen of one type may hinder its own carcinogenic activity. Since fluorescent substances diminish in efficiency as they are concentrated, and increase in efficiency as they are diluted beyond the point where inactivating collisions can occur, too large a dose may postpone activity a bit.

While the schemes we offer here can never represent the situation in all its complexity, they serve as guides in the therapy, and answer many questions besides for the biochemist which otherwise are pure enigmas. They explain the Pasteur Effect as a function of the carbonyl group of a definite position. They show how the (K) region of the carcinogen may operate to make the union that provides for continuous energy transfer into the mitotic mechanism, and they show the position of anoxia in the pathogenesis and what atomic activity is required to liberate the host cell from the carcinogen, be it chemical, virus or both. They show the relation between neoplasia and atrophy and how both are a symbiotic affair that can be broken by the same atomic activity.

CHAPTER III

THE PATHOGENIC MECHANISM

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 cannot 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 dis-

pose 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 made 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.

SYMPTOMATOLOGY OF CANCER

The Pregrowth Phase

The constitutional nature of cancer was well demonstrated by Glover in 1926, when he showed that the pleomorphic organism that was responsible for the disease could be isolated from the blood long before a growth made its appearance. He published in the Canadian Lancet and Practitioner of that year. He also demonstrated the germ in all cancer tissues that were examined, and proved the etiological position of this germ in fulfillment of the four laws of Robert Koch. Of course he further proved his point by developing a curative serum, and that brought the wrath of the profiteers on human suffering down on him. His persecutions are too distasteful to mention here, and too disgraceful to Canadian Medical and American Medical Association leaders to think of. At any rate his great service was lost to the world all this time, and only now at the International Congress of Microbiology held at Rome in Sept., 1953, was Glover's work presented by Dr. Geo. Clark, who with Dr. Engle, repeated the work with full success under the auspices of the United States Public Health Service. However the government did not allow them to publish their results. The exposition at Rome therefore is a great advance for truth. Contemporaneously von Brehmer discovered and developed the same facts

in Germany, using a different technique. But his announcements were not made until a few years after Glover made his. Von Brehmer has the support of the bacteriological faculty of Germany who made a critical investigation to throw it over if possible, but found that the organism was what von Brehmer claimed and what Glover found too, a pleomorphic germ showing varying morphology, bacillus, filiment (fungus), coccus and filterable virus. The cocci are spores that develop in the bacilli. Glover showed that all morphological phases give rise to the virus and bacilli. Besides many other bacteriologists have confirmed these researches in so far as their own investigations have dove-tailed with them. Both Glover and von Brehmer have confirmed the etiological position of this organism serologically by developing an antitoxic curative serum that showed gratifying efficiency. As the German commission reported, it is therefore a bacteriological entity independent of other germs serologically.

This of course means that cancer is an infectious disease, as we have always claimed also, and the existence of the infection in the system should give rise to symptoms. We made our investigations on that point from 1914 on and published our findings in the Medical Record of New York in October, 1920, and in the Cancer Journal of 1924, as well as other papers. Our observations showed that the Flu epidemics of the first world war gave cancer a boost, and the symptomatology was much more intense 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 off-spring 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 absorbing 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.)

We have interpreted this affair as a protective action of the growth through which in time it may not only learn to absorb the poison and take it out of the blood stream and tissues, but also learn to destroy it. The increase in the neoplastic activity generally follows the rate of toxin production, and so we argued that if the toxin (virus) could be done away with, the growth would become obsolete and disappear. Our experience shows that this is exactly the case. We diagram the integration of virus and toxin with tissue functional structures later on.

To illustrate this situation, it can be done no better than to paraphrase a letter received last week from Pasadena, California. It runs as follows—"In 1932 I brought my mother to you from La Cross, 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. Since your treatment was given my mother and she got well and lived fifteen years longer and died of a heart attack, I have confidence in your advice. 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 de-

veloped 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.

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.

Tissue burns from steam or fire produce similar incompletely burned products which are irritating, and the incompletely combusted products of heart muscle activity under coronary insufficiency cause the pain here too. Completion of the combustion in cases of burns, or during a coronary attack, has brought grateful relief. And it should be evident that efficient oxidations would have prevented the pathogenesis as well. The aging of tissues that goes with this toxogenic fibrosis is easy to understand, and the toxins that cause cancer must certainly add to the rate of aging by their stimulation of fibroblastic tissue. Blood plasma of patients with cancer or developing it should show this effect. Case histories that follow show how the fibrosis can be disposed of by supplying highly activated carbonyl groups. We have observed how burns stop paining and heal quickly with minimum scar formation after the catalysts are given, and how the fibrosis of the sclerotic liver, kidney, heart, and brain, disappear after many years of accumulation and hindrance to function. Case histories will demonstrate this. It is a sociological matter of the highest importance.

After the treatment is given the symptomatology of the

years of the pathogenesis is repeated in reverse order to its production. (Koch, Cancer and Allied Diseases, 1926). That is, the last set of symptoms or tissue change to come are the first to flare up and disappear, and the first to come are the last to be repeated transiently and then go for good. Such exhibitions come at cyclic periods that are multiples of three weeks, and follow a definite characteristic course which is informative as to the status of the recovery process. Thus finally after a neoplasm has been absorbed, there may be a sudden intense inflamation of an old scar from a wound, or in the tonsil on the same side as a breast from which cancer was absorbed. This may last a few days and then be gone and with it the induration of the lymphatic glands of the region as well. As the fibers of the scar or interstitial deposits undergo depolymerization as a result of the oxidation chains initiated by the treatment the more superficial deposits are liberated and depolymerized first, and so have a chance to show their symptoms as they are being degraded and burned out of the way. Thus the last symptoms to come are the first to go, and that includes the neoplasm, or the coronary fibrosis, etc. Finally, after the acute inflammation has come and gone, one may conclude that the toxic unit as first produced and incompletely burned, is now out of the way, and with it the pathogenic agent. The cure is then complete. The cyclic reactions, and the retracing of the symptoms of the pathogenesis in reverse order is an established characteristic of this treatment which distinguishes it from all allopathic affairs. The cycle phenomena indicate the basic nature of the recovery process.

Evidently fibrogenesis and neoplasia are both detoxication attempts, and where fibrogenesis becomes exhausted, or is unable to get started as in infants, neoplasia asserts itself. Thus they appear to both be imperfect complimentary processes in which fibrogenesis (mesodermic protection) staves off neoplasia. It is evident too that the dehydrogenations by our reagents, start the oxidative destruction of the pathogen right at the point of attachment to the host cell, where the very inception of the disease took place. The pathogenic significance of the fibrosis of "silent" focal infection is also evident.

THE PAIN IN CANCER

Pain in cancer may be divided into two categories. One is the natural pain, and the other, the pain produced by surgical and radiological injuries.

Natural Pain

The **Natural Pain** is due to exposure of the nerve endings to the air and its contaminants, because of the destruction of the superficial protection, as the malignant erosions progress. Pressure of the tumors produce both pain and painful reflexes through pressure and tension affecting the nerves. Then there is the chemical cause of the pain which resembles that caused by the incompletely combusted products of a burn, or caused by the incompletely combusted fuel products of hypoxia in coronary insufficiency. Thus the faulty oxidations of the neoplastic cells yield irritant products that may cause very intense pain.

The weight of laboratory opinion has decided that cancer cells are much of the same order as normal cells in the utilization of oxygen. However the clinical facts show that the capacity of tumor cells to use oxygen is vastly reduced, and their utilization of oxygen yields irritant by-products rather than carbon dioxide, and does so at a lower potential that is unable to provide energy for the building of the highly differentiated cell ultrastructure and gene material required by a surviving tissue reciprocity and coordination, or by the racial economy. The neoplastic products also belong to the grade liberated in the Krebs' cycle of oxidations, and these are irritant acids, which further reduce the tissue alkalinity to favor amino acid decarboxylations resulting in toxic amine products that interfere with oxygen utilization in the several ways described in this text. Moreover the energy that is produced by hydrolytic glycolysis, is not only able to support tumor mitosis but is yielded at the expense of more acid production which furthers the neoplasia as just described, and also by the pain these highly irritant acids cause. Study of the natural pain of cancer observed clinically may thus help to eliminate many of the laboratory uncertainties. The natural pain proves the quantitive and the qualitative deficiency in the oxidations of tumor cells.

We prove it also by supplying oxidation catalysts that repairs the deficiency. When this is done as described in our thesis, the irritant by-products are oxidized out of the way, and pain disappears completely or to a bearable extent, and in a very short time as a rule. Pain, due to nerve compression from fragments of erroded and collapsed bone is a different matter and always requires the relief from the pressure by proper splinting. Such catastrophes should be anticipated and proper splinting done to prevent them. Likewise pain of sphincter spasm and ballooning of some viscus wall through nerve inhibition, has its specific type of care, as by lavage, proper diet, and the correct homeopathic remedy.

Therapeutic Pain

Therapeutically produced pain is much worse, and more difficult to treat than the natural pain. The necessary stretching of tissues incident to radical surgery cannot be avoided. But the destruction of the blood supply favors more rapid recurrence with necrosis and infection and increased production of incompletely burned metabolites and increase in the pain in a way that soon catches up with the status had it not been operated. Ewing's summary is conclusive—pp. 597-8 of the 1942 Text on Cancer—"In estimating the economic importance of the surgical treatment of mammary cancer there must be charged up the cost of acquiring surgical skill, and the deplorable conditions following local recurrence. There can be no doubt that operation shortens life and *aggravates the terminal suffering* in the great majority of recurrent cases." (Italics are ours.)

Yet the chief cause of therapeutic pain is irradiation. Oxidation deficiency throughout the whole irradiated area is added to that naturally produced, and incompletely combusted irritant products increase with the extensiveness of the irradiation to add to the pre-existing misery. Even though mild irradiation may show no pain effects, the products of the incomplete combustion it causes, may announce their presence in other ways. This situation requires illustration to be understood. In 1941 while visiting in Rio de Janeiro, a leading internist offered a case for treatment, that had baffled all the experts far and wide. This was a young woman of high social position and a remark-

able beauty. She wished to be married, but this could not be done until a mortifying skin condition could be made to disappear. Her whole body was covered with small black pigmented spots from three to ten millimeters in diameter. They started to appear two years previously and increased in intensity steadily month after month. She carefully avoided the sun light, but it did not reduce the increase in the intensity of the pigmentation. A chain type catalysis was therefore evident, and I suspected two factors that would compliment each other in producing the condition. One is some form of irradiation as the X-rays, and another a special sensitiveness lying dormant in a suppressed tendency to pigmentation. Thus a trace of negro blood which is common in Brazil would provide the sensitivity. Inquiry also showed that she had received some X-ray therapy six months before the spots began to appear. The therapy was light, and for a very mild skin complaint that would have cleared up on diet alone. On the theory that the irradiation produced incompletely combusted products that acted like those of a burn from the sun, but in self perpetuating progression as the effects of irradiation are, I figured that completion of their oxidation would destroy their pigmentation exciting effects, and the hereditary tendency to make the response would lose its stimulus, thus the spots may disappear. The treatment was given and they were gone for the most part in seven months, and those that still remained were scarcely noticeable. The girl was married and is reported to have lived happily ever afterward.

Other effects of the suppressed oxidations may extend to the offspring and result in physical defects, or they may suppress function and cause tissue atrophy in the subject himself. In such instances the cell ultra-structure and gene material are injured so they cannot conduct their oxidations. Consequently dependent function fails. The irradiation pain is comparatively a crude effect in contrast. Likewise suppression of the "ultra-oxidations" removes the protection against leukemia, and are hindrances to endocrine development and function. The whole gamut of irradiation has now been used and the turn from the X-rays to isotopes is about the same as leaping from the frying-pan into the fire. Intra-atomic defects are produced both ways

and such defects cannot correct the inter-atomic defects that make up the various diseases. So how can they cure? Suppression of reaction to disease agencies may give symptomatic relief for a time. But the disease only gets a better chance to imbed itself and spring forth when least wanted. We hope that a frank estimate will be made of this subject some day and that when accounts are balanced, the physiological view will be considered and advanced with the same vigor as was given to irradiation therapy.

The high cost of narcotics that must be used but finally fail to quench the pain fire, and the general injury to the tissues, and especially the brain injury by the irradiation narcotic combination are to be considered too. Edema of the brain brought about in this way, with its terrible hallucinations, and the horror of distorted ideas, ravings, etc., offers about the worst way any one can pass on into eternity. Thus a religious problem is concerned. Irradiation is given to reduce pain. The first few applications have a numbing effect on the nerves so that pre-existing pain disappears, but as the nerve injury develops the numbing effect disappears and the pain returns with steadily increasing severity until opiates fail to relieve and it becomes necessary to use some form of intravenous anaesthesia, until death comes as a mercy.

THE KREB'S CYCLE

Decades ago Prof. Roger Williams taught that the injury a surviving tissue preparation suffered, would partly or totally hinder its performance of oxidation of sugar, and that the intermediaries so often isolated and credited with what was later designated the "Kreb's Cycle" are not to be found where the tissue oxidations progress unhindered. Besides this injury to the tissue, which makes it anaplastic or deficient as does anaplasia in cancer cells, the circumstances of oxygen transport and waste product elimination in the tissue slice in the respiratory chamber, set up reverse equilibria that lead to carbon dioxide fixations, that are observed to increase the number of carbon atoms in the sugar chain to more than its six when it is supposed to be broken down to shorter chains. It is not surprising that the Krebs Cycle pictures an anaplastic hypoxic metab-

olism much like the cancer cell exhibits, and that therefore, the cancer cell is not of the order of the normal in its oxidations. Instead the bit of tissue studied in the apparatus does not represent the normal at all. The error of the bio-chemist on this question is very serious, and is one of the great hindrances to the progress the conquest of cancer should have enjoyed for decades.

Waters called attention to the fact that the dehydrogenation of succinate to fumarate could not take place in an aerobic medium, since the free radical formed would add molecular oxygen and form a peroxide free radical that would start other oxidation chains than those of the Krebs' cycle or break the molecule down to shorter bits. Only in an anaerobic medium could fumarate be formed, and the Krebs' cycle is supposed to not deal with an anaerobic medium. Water's criticism is in agreement with our own, since we figured that the fumarate formed under anaerobic dehydrogenations, would lay over as a preserver of aerobic chain oxidations when oxygen again was admitted to the field or when true oxidation catalysts could be supplied. Thus the anaerobic fumarate product preserved the aerobic status of the tissues through its carbonyl groups as activated by conjugation with ethylene linkages, and therefore as dehydrogenators, that are able to initiate chain oxidations in the presence of molecular oxygen.

Tissue cells appear to be well supplied with dehydrogenases in a wide range of oxidation reduction potentials for specific action on the residues encountered under hindered conditions of sugar oxidation. They doubtless keep the field clear for the high efficiency "smokeless" process of energy production that leaves no trace of its action by way of isolatable intermediaries. We have concluded that they serve as an alternate pathway for oxidation when the master "smokeless" system is hindered by hypoxia, or integration with a virus or certain amines or carcinogens. Biochemists do not admit the existence of this master process since they cannot trap its intermediaries. But we have practical proof of both its service in metabolism and the mechanism by which it operates. These cannot be denied any more than the existence of citric acid and the di- and tricarboxylic

acid oxidases so neatly assembled in the Krebs scheme, which we hold to be a secondary pathway still available to such hindered cells as cancer cells.

Oxidations on the Krebs cycle level offer no protection against virus or neoplastic parasitisms, but instead are necessary for their support. The Natural Immunity, universally observed, requires a higher order of dehydrogenation to start oxidation chains in the pathogens of neoplasia and virus diseases, and when this is hindered so as to permit disease, it can be restored by use of catalytic doses of highly activated carbonyl and free radical dehydrogenators that operate at much higher potentials than those of the Krebs cycle. The existence and nature of the **Master Smokeless System** is thus identified.

The dehydrogenases of the Krebs cycle operate under physiological conditions (pH 7 and 30° C.) with E_0' at from + 0.38 to —0.293 volts, and at normal electrode potentials (pH 0 and 25° C.) with E_0 at from 0 volts to + 0.464 volts. Moreover the reversible succinate-fumarate system is calculated at + 0.437 volts at pH 0, and 25°C., but is found by use of indicators in bacterial systems to equal + 0.005 volts. The normal potential of the lactic acid-pyruvic acid system is placed at E_0 equals —0.2 volts while the E_0' equals —0.18 volts at pH 7, and the E_0 of ascorbic acid is placed at + 0.390 volts while the E_0' value is stated to be + 0.058 volts. These may be compared with the E_0 of benzoquinone-hydroquinone which is placed at + 0.699 volts, parabenzoquinone being 0.715 volts, as usually stated.

One would look for oxidation-reduction potentials of a higher range than those that operate during the Krebs cycle to burn adrenaline or DOPA during function, with their E_0 at + 0.80, and E_0' at + 0.38 volts. Other toxic amines and pathogenic viruses call for much higher oxidation potentials, the lowest of which serves fairly well at E_0 + 0.715, while others operate at much higher levels. Since these can be used to substitute for the Master Smokeless system dehydrogenators when the latter have been inhibited from action, we conclude that the main course of the oxidations may provide for oxidation-reduction potentials much higher than those involved in the Krebs cycle. At this high potential not only a greater range of fuel, but also toxins

and virus can be burned, and the system of energy production that supports life also protects itself against injury. However when hypoxia supervenes or anoxia exists this protection may be soon exhausted.

In comparison to this system the Krebs cycle holds second place and is set in motion when lactic acid oxidase converts the product of hydrolytic glycolysis into pyruvic acid, under conditions that suspend the Pasteur Effect, we conclude, and these conditions are anoxia, virus and neoplastic parasitism, and various toxic amines, as a rule. But anaplasia or destruction of the particulate cell structure by manipulation can also annul the Pasteur Effect, proportionately.

From what has been stated here one must not conclude that the therapy is simply the use of the highest possible oxidation-reduction agent. No, indeed, each disease picture has its appropriate dehydrogenator that fits somewhat as a master key fits a lock. The disease picture, however, has variations so as to present a class of syndromes that overlap with other classes. The degrees of activity of a given reagent therefore vary within each class and among the several classes that apply. Steric hindrance is determinative here as is explained farther along.

When we consider that the neoplasm or the interstitial fibrosis started out as a repair process to correct a chemical or a mechanical injury in the presence of a toxic factor under varying degrees of hypoxia or anoxia, and the new cells attempting to accomplish the repair built into themselves the toxic elements, they could not burn, as a copolymerization process, each clinical picture presents its bound toxin in a variety of forms and in a variety of ways. Therefore the dehydrogenator in addition to possessing the correct potential of action, must certainly possess other qualities that permit it to fit the toxin molecule it is to attack in its variety of combination. For example, benzoquinone has established its value in the treatment of a variety of diseases including certain cases of cancer. In some cases its action approached the miraculous, in others it scarcely showed any effect and had to be replaced by the serial system of carbonyl groups, with free radical terminals.

The various molecular structures presented in Plate III (page 88) show different arrangements of the carbonyl group, and others as diquinone and ortho-quinone might be added. The latter offer no advantages that we have seen so far. Yet a larger experience might show such an advantage. In all, the electron contributions from ethylene and carbonyl linkages to a certain carbonyl group are different, as are their molecular weights and spacial arrangements which inspection will show. One should also consider that even the same substance in different states will show activity variation of maximum or zero value. Thus Maleic anhydride, in which all double bonds are coplanar can enter polymerization reactions readily while the esters or the plain acid is non-reactive. The same holds for its curative action in certain forms of glandular tuberculosis that we have ourselves observed. In the esters and acid only one carbonyl group is coplanar with the ethylene linkage at the same time, showing steric hindrance to meet a perpendicularly attacking radical. Even at this time our selections of active molecules in various conditions is still crude and requires much more classification of the clinical data at hand. More will be stated on this subject in the completed text. Here we see that we have a choice between carbonyl activated in various ways by electronic migrations from ethylene groups in different degrees, and from other carbonyl groups in different set-ups, and also of free radicals terminal to carbonyl group chains. Then there is the peroxide free radical we have finally succeeded in producing safely and effectively for clinical use. It is stable in solution only a few hours, but in this time it was able to initiate curative oxidation chains in the tissues that none of the other molecules have yet succeeded in producing, and our stride toward the best cure rate in neoplasia has been advanced greatly and it is more effective in other virus diseases also.

Since the natural protection can be duplicated and even greatly augmented by free radicals and peroxide free radicals of appropriate structure, we are convinced that the master oxidation process is not only initiated by carbonyl groups of high potential, but is carried by free radicals and peroxide free radicals as a chain process. Such facts place the tissue oxidations in

an entirely different light from that of current notions, and show that the free radical and the chain process it initiates is a real feature of the most efficient grade of metabolism.

PARASITE HOST CELL INTEGRATIONS

Diagramatic Presentations

We have long looked upon the **integration of virus and host cell to be an azomethine condensation between the virus amine group of highest activation, and the host cell carbonyl group of highest activation, the group that starts the oxidation chains in fuel and toxin substrates by dehydrogenation.** Let oxygen lack intervene and the virus is no longer attackable by an oxidation chain started at a carbon atom alpha to a double bond. It is free to form a condensation with the host cell carbonyl group and draw energy from the host at this point, since inactivation of oxidation initiation destroys the Pasteur effect and lets the hydrolytic glycolysis system in to produce energy in uncontrolled fashion. Thus the virus can shunt off the energy from the host cell. **Moreover the free radical formed in the virus during hypoxia, by the host cell's last act of dehydrogenation preceding complete anoxia, is able to attach to one pole of a double bond, let loose when the hydrolytic glycolysis system steps in,** for at this time the virus cannot condense with a host carbonyl group as it is not freed of the hydrogen atom it removed from the virus. **Thus two types of union can take place, one in which hypoxia is not essential but is favorable and one where it is essential.** These are shown in diagrams II and III. To the degree that oxygen gains entrance to the host cell, and under the conditions stated, the Krebs cycle can come to the rescue of the hydrolytic glycolysis system to aid energy production, but it cannot serve function under these conditions either, and so boosts virus synthesis and the neoplastic multiplication.

Abbreviations, F. M. — functional mechanism, E. M. — energy producing mechanism, M. M. — mitotic mechanism, H. G. S. — hydrolytic glycolysis system, P. P. — phosphate energy storage system.

(A)

Showing Functional Mechanism Incompacitation Illustrated by Diagram I

In (a) the energy passes into the F. M. normally. But after the Functional Mechanism is worn out from over work and can

DIAGRAM I

(a)

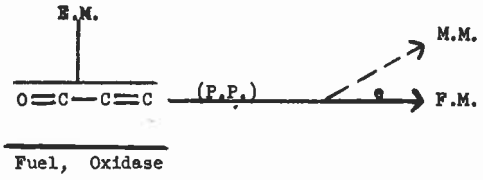
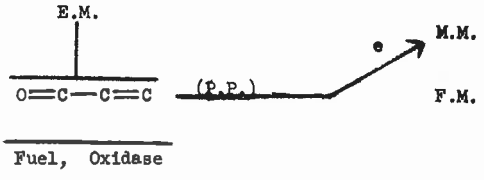


DIAGRAM I

(b)



not accept the energy, as in (b) it passes on to the mitotic, or cell division Mechanism which accepts it into its chemical processes of cell division and daughter cell construction, causing cell multiplication, with adequate functional structural provision.

(B)

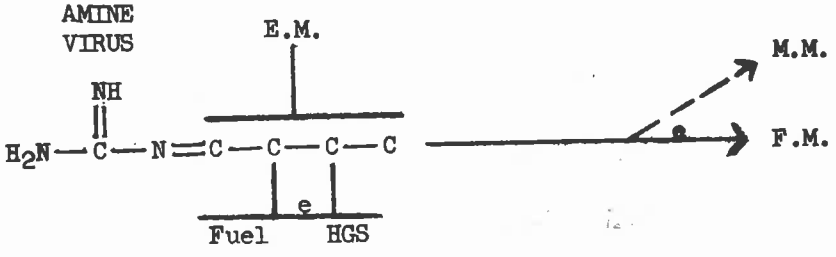
Effect of Condensation of M.F. Carbonyl Group with Guanidin, or Virus

Illustrated by Diagram II

Here the hydrolytic glycolysis system is to be remembered as acidic in nature. And the basic nature of the double bond that activates the carbonyl group that is now condensed with an amidine may be assumed to call for the association of the two. Thus the means of pouring energy from hydrolytic glycolysis into the functional mechanism is provided for even though the atomic groups individually concerned are not to be differentiated. But where a good supply of oxidase is at hand the carbonyl group will dehydrogenate and form free radicals in fuel and HGS so they can join up with the ethylenic linkage, as pictured above. This diagram also pictures the integration of virus via its amine group with the energy mechanism carbonyl group, to cause neoplasia. Blocking host cell oxidation throws the hydrolytic glycolysis system into perpetual action, supplying energy for neoplasia and for the attached virus. And the Krebs system aids with whatever oxygen is available.

DIAGRAM II

Energy Mechanism



(C)

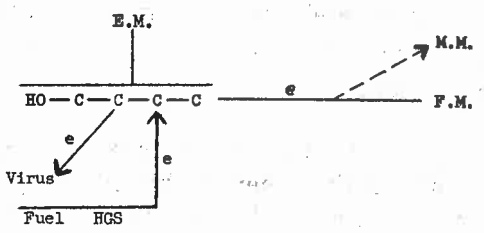
Hypoxia From Over Exertion Illustrated by Diagram III

Here the lack of oxygen prevents the free radicals last

formed by dehydrogenation from adding molecular oxygen so they add to the double bonds of the ethylenic group. The car-

DIAGRAM III

Energy Mechanism



bonyl group is not stripped of its accepted hydrogen atom either after the oxidase serving the purpose is worn out by oxygen lack. Hence the virus amine group cannot condense with the host carbonyl group, but as the hydrolytic glycolysis system is shunted in at a double bond of the host energy producing mechanism, the pole liberated here as a free radical can condense with the free radical formed in the virus, and the energy can pass from the glycolysis system to the parasite by their close proximity of attachment. If there were no virus attachment at the pole of the double bond, of which the other holds the hydrolytic glycolysis system, admission of oxygen to the field would restore the carbonyl group and free the hydrolytic glycolysis system. But where the two poles of this double bond are taken, the bond no longer activates the carbonyl group, and it makes little difference if it is freed of its accepted hydrogen atom or not. However, if a highly active carbonyl reagent is admitted to burn off the virus, complete restoration is again possible in the presence of oxygen.

(D)

The Hook-up In Neoplastic Disease

Illustrated by Diagram IV

During good oxygen supply and carbonyl function, chemi-

cal carcinogens or virus are dehydrogenated and peroxidized and burned out of the way by a chain process. But where the carbonyl group is inactivated by condensation with an amine and cannot dehydrogenate further, and anoxia prevents the last dehydrogenated virus or fuel substrate from forming peroxide free radicals, the virus free radical will join one pole of the ethylenic double bond and the HGS will join the other by its double bond and the free radical left at the other pole in the HGS will join fuel substrate.

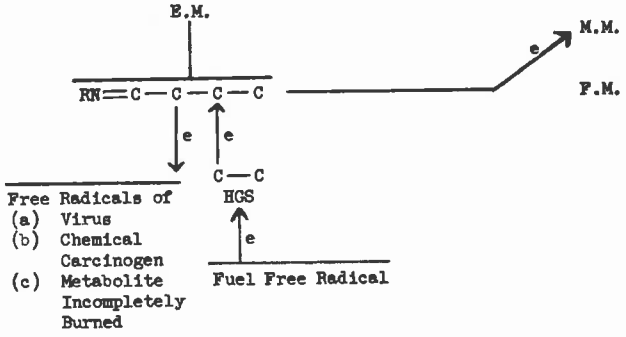
Thus energy is passed to the virus on one hand and to the mitotic mechanism on the other, each having equal opportunity to receive energy. The amount of oxidase at hand does not influence the procedure after the azomethine bond is formed, but before the functional carbonyl group is inactivated in this way the amount of oxidase will determine the number of free radicals formed in any carcinogen or fuel molecules.

The FM is destroyed or inactivated with its gene pattern by the initiating carcinogen that causes the anaplasia so all energy goes into the mitotic mechanism and the virus, and cell division does not produce new functional mechanism in the absence of functioning genes on which such a mechanism is built and patterned. Anoxia will serve much like the toxic amine since the oxidase will not be able to dehydrogenate the FM Carbonyl group much longer than when oxygen is absent. However here return of oxygen supply can accomplish a freeing of the carbonyl group and of the HGS with it.

Inspection of the diagrams given here will show why fatigue should predispose toward virus infection, and why exhaustion can prepare the way for cancer. They show too why infectious filtrates are not readily obtained from carcinomata, and why the mitotic showers occur in cycles, when one considers the hypothesis of the sprouting spore as an origin for "virus," —or maybe pseudo-virus. They indicate why dietary and hygienic measures may aid both in the prevention and in the cure of cancer.

DIAGRAM IV

(a)

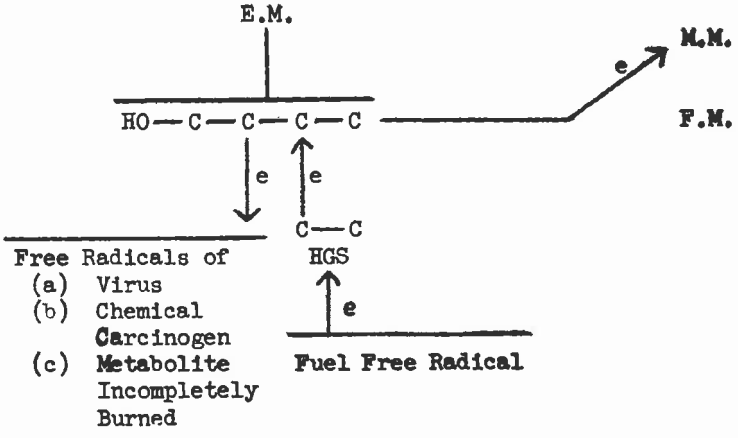


The F.M. does not exist here because of anaplasia, { embryonic / toxic / traumatic }

In set-up (a) the functional carbonyl group is inactivated

DIAGRAM IV

(b)



by condensation with an amidine of the virus, or some tight bond inseparable under ordinary conditions.

In set-up (b) the functional carbonyl group is inactivated by anoxia because of which the oxidase cannot remove the hydrogen atom it extracted from carcinogen, virus fuel or metabolite. The number of free radicals formed in this last process will depend upon the amount of oxidase that was present after the anoxia prevented peroxidations of the free radicals formed.

(E)

Lytic Virus Parasitism

Illustrated by Diagram V (a)

Here anoxia prevents the virus that was dehydrogenated from forming a peroxide free radical, so the bare free radical adds to the ethylenic linkage beta to the carbonyl group where it receives the energy formed by the HGS which has joined the other pole of the ethylenic group by a double bond. The free radical thus formed in the HGS joins the fuel substrate. The host functional and mitotic mechanisms thus are cut off from the energy yielded through hydrolytic glycolysis; and it passes into the virus.

DIAGRAM V

(a)

M.M. Inactive in Nerve Cell

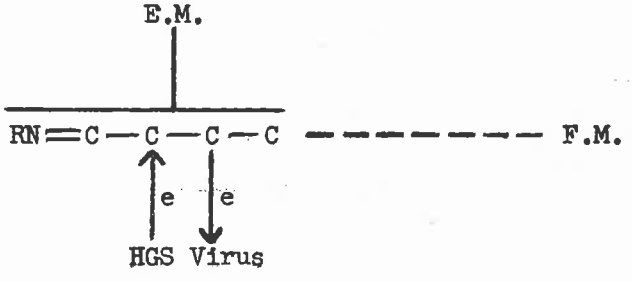
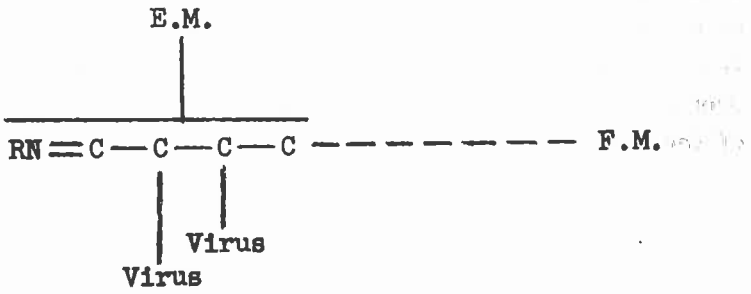


DIAGRAM V

(b)

M.M. Inactive in Nerve Cell



Supportive energy not produced

(F)

Symbiotic Virus Parasitism

Illustrated by Diagram V (b)

Here two dehydrogenated virus units join the two poles of the ethylenic linkage. Thus no HGS or fuel is hooked up with the energy production system and no energy is supplied for the vital processes except as it leaks in through fluorescent transfer from some system as the Krebs cycle which is only sufficient to keep the host and virus alive, but in a nearly moribund state of atrophy where neither can carry on any vital process of function. The Functional Carbonyl group is inactivated by amidine condensation (or oxidase failure), the amidine belonging to the virus, or some toxin (R).

This may be the state of affairs in the anaplasias illustrated by undescended testes, thyroid hypoplasia, etc. A virus or tissue or bacterial metabolic unit not fully oxidized and polymerized under anoxic conditions may offer the free radical that joins up to both poles of the activating ethylenic double bond. The amine group that inactivates the host carbonyl group may belong to the virus and need not be supplied by other toxic agents. It could be the only point of attachment of the virus which would inactivate oxidation initiation and give a path for energy from hydrolytic glycolysis to the virus and away from the vital processes of the host cell.

(G)

Carbonyl-Amine Functional Relation

Illustrated by Diagram VI

As we explained for the carbonyl-creatin interaction of the normal voluntary muscle set-up, here we picture the ketosteroid special amine relations.

Fundamental relations of this type should not escape notice for too long, since a rational explanation of function as based upon chemical structure demands scrutiny along this line in the whole realm of biology.

DIAGRAM VI

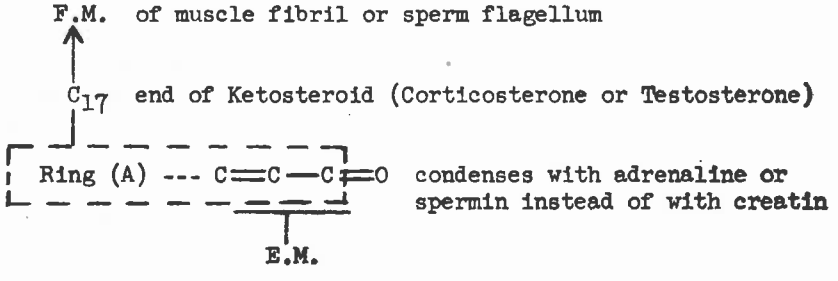
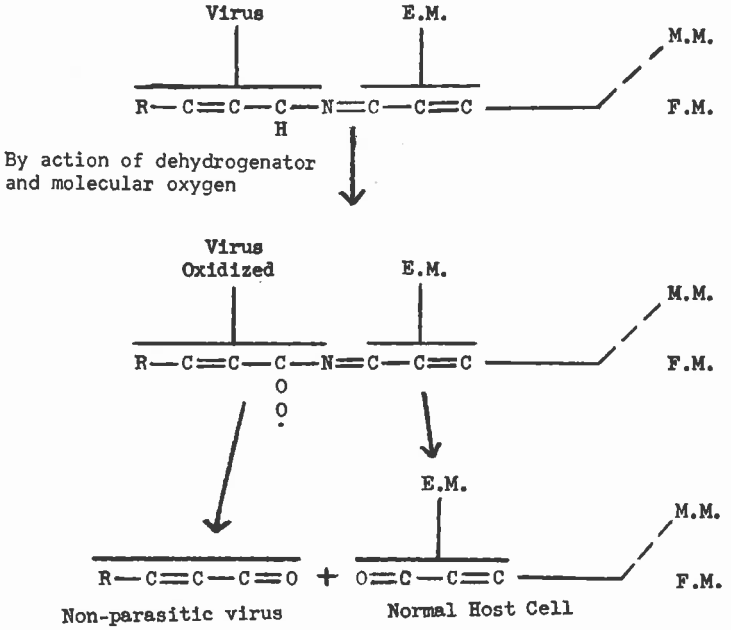


DIAGRAM VII



(H)

Oxidative Rupture of the Symbiosis with Cure

Illustrated by Diagram VII

When the virus is attached to the energy producing mechanism through condensation of its amine with the functional carbonyl group, the azomethine double bond activates the hydrogen attached to the carbon atom alpha thereto, and dehydrogenation by the remedy carbonyl group places the free radical there. Oxidation produces the peroxide free radical that splits the virus off without an amine group, but rather a carbonyl group terminal. This carbonyl group we assume is activated by a double bond in conjugation and then can conduct oxidation chains itself and no longer need be parasitic. Indeed the loss of its critical amine group excludes this possibility.

The host energy producing mechanism is left with a nitroxide free radical type condensate through a double bond which readily hydrolyses off, leaving the host cell in normal condition.

(I)

Mechanism of Fatal Vaccine Production and its Avoidance

Since the virus amine group is its means of inactivating the host cell protective functional carbonyl group, virus collected from living tissue cells may separate off with the amine group condensed with a carbonyl group from which it is readily hydrolyzable. Then when it is exposed to formaldehyde to be **"killed"** the amine group is protected from the aldehyde. Then when it is injected into a patient the protective azomethine bond may readily be split setting the active virus free. To avoid such a disaster the virus on collection should be exposed to a lower pH reaction which will accomplish the freeing of the amine group from protective condensation, and the carbonyl compound should be removed, before the formaldehyde is administered at a higher pH to assist the formaldehyde amine condensation that inactivates the virus. No diagram is required to illustrate this event. Phenol red, used as indicator to show presence of living virus, changes color at pH 7.3, and hence is cause of error as explained here. An indicator that changes at pH 3 is needed, with the medium so acidified to make sure virus amine groups are free.

Besides the conventionally recognized lytic and symbiotic types of virus-host cell integrations, clinical observations show a third type that produces no specific functional symptoms, but only shows hindrance to the tissue oxidations, weakness and the putting on of fat during the integration phase, and congestive symptoms with some fever during the oxidation of the virus off from the host cell during the cure process. This may be the type of integration that dominates during the grippe and characterizes the pre-growth phase of cancer as well as the type of union held by vaccines that block integrations of host cells with the virulent phases of the same viruses. Indeed the preparation of the host cell for the action of some chemical or metabolite of

germ or tissue cell origin that activates the neoplastic process may just be this type III form of virus integration. The "flu" epidemics of the world wars after which cancer incidence took such big strides is rather suggestive of such a situation.

It seems to us that this type of integration yields no functional symptoms as the virus concerned has no functional chemical process that can accept energy from the host cell. For to accept the energy, it must have some place to go, as for example some chemical process it can enter and activate, and which draws energy off from some specific chemical process of the host cell. It seems to us that symptoms of disease are produced when energy is thus withdrawn, or when it is passed back to the host cell, when the virus is being burned off. This holds true also for carcinogens or other toxins integrated with host cells, and so during the recovery process when the host cell is being freed of its pathogen, the symptoms that were produced during the pathogenesis are again being repeated in reverse order. Thus too, the dissolution of fibrotic tissue that walled off infection yields symptoms of the toxin as it is being broken down, and in reverse order to those of its polymerization during the pathogenesis.

The bridge over which this energy passes is the non-specific azomethine bond, or the free radical-double bond addition we have described for the integration with the host cell of virus or toxin. We also held that this bridge for energy passage that is so important to virus vegetation, and so fatal to the host cell, also provided for the cure, as it offers the double bond, or is adjacent to one which activates the alpha placed hydrogen atom, and thus serves the dehydrogenation that initiates the cure process. It is not specific for anything as any energy can pass that has somewhere to go. When the type III integrated virus is undergoing oxidative disintegration or separation, the energy yielded is not of the quality the host cell can accept into any of its chemical systems so it does not produce any symptoms of a specific function but goes off as heat and causes fever. It is also characteristic that when this fever is high, the patient feels the best ever, eats better and is much stronger. It is a different fever than that coming from the dissolution of host tissue during the pathogenesis. Many patients describe this event

with wonder during the reactions of the recovery from cancer.

One question is to be answered relating to this type III integration, where the parasite cannot use the specific energies yielded by the host cell. Does it pass this energy on from the functional mechanism with which it is ligated, to the mitotic mechanism in some such way as we diagrammed earlier? Thus if the virus were condensed with the mitotic mechanism via a free radical-double bond addition, and to the energy producing mechanism of the cell functional mechanism by an azomethine double bond, weak energy production would be assured and the path for its entrance into the processes of cell division could be visualized. The cases of cancer that recover without repetition of symptoms of specific functional interference may be explained by this assumption provisionally at least, especially when the oxidation processes are still hindered.

One has to think of the specific protein of the virus which when integrated with the host cell confers its specific serology thereto. It may be the pattern of this specific protein that accommodates the adherence of the virus to the host cell at the first collision. One sees such an event when boiling cream for its preservation, the protein material tends to accumulate after standing and cooling, as a ball, which increases to a certain point with time. Here molecules of a definite pattern fit and hang on to each other as they make contact. Vaccines may thus also work via their occupancy of the receptor spots of the host cell, and thus exclude the attachment of virulant virus covered with the same type of protein. Besides a specific digestive ability of the host serum for such protein will remove its mechanism of attachment to the host cell surface. Thus the bridge mechanism for true integration, that provides for the parasitism and its energy transfers, is not able to find entrance and gain hold in the host cell, and the specific protein antibody immunity is reasonable protection to a certain point. Thus all immunity known today must be established before the challenging infection is given. The host cell must be protected from penetration by the parasite. The cure process however, in line with our thesis and actual practice, can take place via the oxidations induced any time after the integration has happened, so long as the host cell is not injured

too badly to survive after the parasite is oxidized off. This system then is the only means of protection and cure after the virus has penetrated.

SUMMARY ON CARCINOGENESIS

It will be seen from the foregoing explanations, that carcinogenesis resides not only in a specific quality of the K region of the carcinogen that makes it liable to free radical production but also in an extrinsic event in the field, anoxia, which leaves this free radical free to join a pole of a double bond of the energy producing mechanism associated with mitosis, instead of forming a peroxide free radical and either undergoing fragmentation, or serving as the carrier of an oxidation chain that disposes of many more molecules of the carcinogen. This free radical mechanism is supported by a number of facts as for example, the interference with polymerizations shown by minute traces of carcinogens, and also the marked inhibition of carcinogenesis by azo-dyes, produced by traces of methyl-cholanthrene, while substituted amine types mutually accelerate their activities. (E. M. carbonyl inactivation).

In addition the carcinogenic act is one that causes a pouring of energy into the mitotic mechanism of the host cell, as we have tried to explain, and the electronic density of the K region is not sufficient to account for that. If the energy is transferred via fluorescence as we proposed decades ago, the electronic picture of the molecule as a whole is concerned, for the energy handed over through fluorescence from hydrolytic glycolysis (Koch, Natural Immunity, 1935), to the reactions of mitosis must be of the quality mitotic processes can accept specifically. This thesis must not be confused with the chemiluminescent theory of Anderson (1947) which as we understand him, claims that the energy liberated by oxidation of the carcinogen changed the cell protein from the benign to the malignant type as do the X-Rays. However experimental scrutiny would show that when the carcinogen is oxidized it is no longer active as a carcinogen, and X-rays break the atom down within, with a much greater energy yield than the inter-atomic action of visible and ultra-violet wave lengths that act on physiological and pathological levels. Our old theory was one of photo-sensitization in

which small amounts of carcinogen could mediate a continuous energy transfer without exhaustion.

Another matter to consider is that when the carcinogen free radical adds to one pole of the most reactive double bond of the host cell, it becomes a "substituent" with a rich conjugated system of double bonds, benzene rings, etc., which must alter very greatly the electronic configuration of the vital molecule of the host cell. In consequence to this distortion, energy production and distribution would necessarily be muddled up too, so that instead of going on to the processes of some useful function, it might be diverted to the mitotic reactions.

We are held to an explanation of carcinogenesis which will also clarify the chemistry of virus parasitism, since a broad front of carcinogenesis like a broad front of virus infection is corrected by a single reagent—the activated carbonyl group, or its trailers, the free radical in the presence of oxygen or the peroxide free radical. Hypoxia prepares the way. The clinical cure of cancer or virus infections must take the latter into consideration, as well as the "functional" hypoxia by which one may characterize the paucity in oxidation enzymes and co-enzymes and unhindered action of the energy producing mechanism's carbonyl group. The diet and its riboflavin content, as well as the freedom from oxidation inhibitors as selenium requires comprehensive attention. Trace metals that serve the oxidations command their position too, and here copper is important for phenol and amine oxidase activity.

Oxidation hindrance is seen long before a neoplasm appears. The general toxemia with its pre-growth symptoms that generally affect the nervous system and skin more or less than a decade before the growth comes, and the general loss of oxidation power as seen in the sudden increase in taking on fat, the general weakness and easy fatigue, and the hemolytic changes are characteristic. Besides there is the deterioration of the family from generation to generation with a shortening of the pre-growth toxic period and an earlier advent of the growth until it comes before puberty, and the line is extinguished. Steadily increasing inactivation of functional carbonyl groups throughout the body paves the way for the local weakness that yields to the

carcinogen, and the multiplicity of factors that promote and are favored by carbonyl deficiency cannot escape the careful notations of the case record. (Koch, Cancer Journal, Oct., 1924.)

The problem was to invent carbonyl compounds of highest dehydrogenating power, and that was easy. But to secure active free radicals in a state that would serve transportation was not so easy; and to prepare a peroxide free radical of useful type without exploding, and which would stand up for a useful period in aqueous solution was most difficult of all. Facilities in Brazil made the last achievement possible. So it is now demonstrable that each type of reagent has its place in the chain of events required for explanation of the normal procedure and for the correction of the abnormal.

Carbonyl groups are able to prepare their own field of action. Since the value of dehydrogenation depends upon the use of molecular oxygen to form the peroxide free radicals that continue the curative catalysis, carbonyl groups had to first get rid of the intercellular and intracellular colloidal gelation, and even vascular colloid gelation that blocked oxygen transport and caused the essential anoxia. This gelation is due to the action of the amines produced by the fungi always present in the neoplastic lesion, and carbonyl of high activity does inactivate such amines. Where its activity is unhindered no such fungi can take root either, so cancer cannot exist there either.

The symptoms of the recovery process that show a repetition of the symptoms of the pathogenesis is in reverse order, suggest a fragmentation of the pathogen as via a depolymerization, with oxidation of the fragments and energy passing to the host cell at levels from which energy was taken from it during the pathogenesis. Thus it appears from the clinical side that the specific symptoms of the disease and of the cure are due to energy passage from host cell to pathogen during its production and energy passage from the pathogen to the host cell during the cure, and for each symptom complex the energy passed has a special or specific level or quality.

That viruses may polymerize to larger units is suggested not only by the variety of measurements reported, but by the

type of addition they make with the host cell, a free radical double bond affair during anoxia. Indeed they may add to each other under similar conditions and be responsible for a series of disease pictures as stated before. Energy taken from the host cell may build up the pathogen's vital processes and cause death to the host cell, but the pathogen may pass the energy on to the host cell mitotic mechanism and thus cause uncontrolled cell division or neoplasia. To be taken from the host cell at all, it must have some place to go, that is it must pass into some acceptor, as a chemical process that supports some specific function or polymerization; and it is taken and used at specific levels. This accounts for the specific symptoms of the pathogenesis. The return of the energy to the host cell because of oxidation of the pathogen, will affect its functions so far as it has mechanisms to accept the energy. Otherwise it is lost as heat, and that is exactly what we observe during the recovery process. The energy transfer can easily be pictured as conveyed by fluorescent photosensitization, and we look upon this as a normal process in biology generally.

CHAPTER IV

THE REAGENTS

THE TYPE OF REAGENT

It is evident then from the foregoing that two types of attack may be made. One is to destroy whatever carbonyl respiration a pathogen may have, by condensing its carbonyl group with a highly activated amine as a destructive pharmacological measure. The other is to restore the carbonyl function to its highest physiological value in both the pathogen and in the tissue cell. We found that the first procedure not only injured the host cell, but brought about a resistant mutation in the pathogen. Further, as no carbonyl function was evident in viruses, or of note in cancer cells, such therapy must fail there. Observations with trimethyl melamine (Koch, J. Lab. Clin. Med. 1; 299, 1916) demonstrated a refractoriness to a second dose which we interpreted as the basis for the resistance mutation. We therefore prefer the physiological procedure of establishing high carbonyl function which proved its ability to secure separation of a virus parasite or a toxin after integration with the host cell, and also of making it harmless, and possibly biologically useful as well.

In the atrophic types of integration the results include the full freeing of the host cell of integrated toxin or virus. On the sensory side, this was observed in the cure of optic nerve and retina atrophy. On the motor side, by the cure of long standing paralysis and atrophy of Anterior Poliomyelitis, the restoration of function in the cure of the cardiotropic type of aftosa, and the nervous paralysis of rabies and of well established dog and cat distemper. In the neoplastic cases, the cure of cancer of the usual varieties affecting the vital organs with widespread infiltration and metastasis, the reconstruction of destroyed areas for function as speech, the ability to give birth to children, and the return of good stomach, liver, bladder and intestinal action. The recovery process follows a definite pat-

tern with digestion of the neoplasm into the same food elements taken from the blood to build it, so that nutrition is quickly improved. Just as after the absorption of a tumor, absorption of interstitial fibrosis in impeded organs may be revealed by an exhibition of symptoms and tissue changes transiently that occurred before the neoplasm or functional insufficiency developed, and in a sequence showing a reversal of the order in which such symptoms presented themselves during the pathogenesis. Hence we conclude that the very basis of the pathogenesis was corrected.

Highly active carbonyl groups, that can substitute for those the tissues have lost in the pathogenic set-up, can be produced synthetically by reinforcing the electron concentration at its carbon atom by migrations of electrons from double bonds conjugated with this group. Such double bonds can be those of other carbonyl groups arranged in open or closed chains. They can also be the double bonds of ethylenic groups or the triple bonds of acetylenic groups with which the carbonyl group is conjugated.

The quinones are the simplest examples of the effective ethylenic carbonyl conjugate, although we have used such simple molecules as maleic anhydride effectively. The quinones should not have substituents that displace hydrogen atoms unless these offer double bond systems as conjugates with the carbonyl group. The hydrogen atoms are necessary to restoration of the double bond after free radical addition. Moreover it is seen that the oxidation-reduction potential (ORP) of the quinone increases with the number of double bonds carrying free hydrogen atoms with which it is conjugated. Thus it is seen that anthraquinone with no quinone double bonds carrying hydrogen atoms and with two carbonyl groups, has an ORP, of 0.154 v. Alpha naphthaquinone with one double bond carrying two hydrogen atoms and two carbonyl groups has an ORP of 0.48 v. Betanaphtaquinone, with one double bond carrying two hydrogen atoms, and the double bond of a carbonyl group conjugated directly with the other carbonyl group has an ORP of 0.576 v. Parabenzoquinone presents two double bonds carrying four hy-

drogen atoms and two carbonyl groups, and has an ORP of 0.715 v. Ortho benzoquinone with two sets of double bonds carrying four hydrogen atoms and one of the carbonyl groups conjugated directly with the other has an ORP of 0.792 v. and diphenoquinone with five sets of double bonds with which the two carbonyl groups are conjugated and carrying eight hydrogen atoms, has an ORP of 0.954 v. While the dehydrogenating power increases with the concentration of mobile electrons at the carbonyl group, one will see to it that no substitutions are made in the molecules that withdraw such electrons. As the ethylenic linkages give up their electrons to the carbonyl group so as to make it more basophilic, they become more electrophilic, and hence with the increase of dehydrogenating power of the carbonyl group, the ethylenic linkage also increases in ability to add free radicals.

This ethylenic linkage shows a triple significance. One is important to the detoxicating power of the quinone when acting under anoxic or hypoxic conditions. Thus when the carbonyl group dehydrogenates a pathogen to form a free radical and oxygen is not at hand to peroxidize it, it can add to and injure the host double bonds or free radicals and cause disease. But in the presence of the quinone's highly active double bonds the additions will be made there instead, and the toxic molecule or virus is thus inactivated.

On the other hand the quinone double bonds can absorb and inactivate the free radicals formed in the toxin in the presence of oxygen when the former are in excessive amount, and thus the curative oxidation chain is blocked. Too large a dose of the quinone, especially of the diphenoquinone can block its own good work. Likewise when the recovery process is progressing well, a second dose can stop the favorable progress and even reverse it at times. Then one should not repeat the treatment unless necessary and never use a concentration higher than one to a million parts of solvent. The higher dilutions as the 12X generally work best. They carry millions of molecules per cubic centimeter, and this number may exceed that of the oxygen molecules available where the tissue colloids are gelled or where the circulation is impeded. The best situation for efficient action

is where each molecule of reagent is surrounded by a capsule of toxin molecules, so the oxidation chain progresses from one molecule to another until all are converted. Thus the quinones are good to start the recovery process and detoxicate when oxygen transport is impeded, as it usually is. And then when the inter and intracellular plasma gelling is cleared away a dose of the serial systems of carbonyl groups can be used. These can be prepared in open and closed chains of varying molecular weights.

However, the third aspect of the activity of the quinone's double bonds is not favorable. The greater the number of crossed conjugated systems in the molecule as in dipheno-quinone, the more difficult the situation that may arise. The clinical events qualify a therapeutic reagent better than any other test, and for a complete picture time is required to settle out the ultimate effects. Thus it has become evident after several decades of observation with correctly prepared reagents that where one type of group represents the molecule, as the carbonyl group in multiplied series, the clinical progress after its use is of one type. Recoveries carry through that last. They are cyclic with positive and negative phases, but the progress is forward. Gratifying improvement is appreciated between the negative phases. But when the carbonyl group is activated by electrons obtained from conjugated systems of ethylenic or acetylenic multiple bonds, a tendency to relapse is introduced, and this is greater the larger the number of such groups that are present in the molecule. This property is common to all crossed conjugated systems, and is observed mostly in patients with poor oxygen supply to the tissues. We have observed this situation to be somewhat annoying after using diphenoquinone. Our explanation is that the unsaturated systems present, after the quinone is reduced, activate the reducing power of the molecule, and hinder the recovery process. The sicker the patient, the greater is the anemia, and the more inefficient are the respiratory and vascular systems. Oxygen transport suffers and the exhaustion of oxidation catalysts tend to lessen the release of the hydrogen atom taken up by the quinone. The tendency is for a reduction type of chemistry to prevail or at least a slowing of the

oxidation of the toxin products liberated as the pathogen undergoes destruction. The symptoms of the different stages of its structure in its evolution are then far more annoying than if the serial system of carbonyl groups is used, for here the oxidation of such products may be so fast one scarcely identifies their transient presence. Therefore, also the intervals between the negative phases are never really comfortable.

Even such mild interferences with the oxidative advantage as come to tobacco smokers may show their adverse effects when a quinone is used, and here too the diphenoquinone may serve worst of all. Thus in 1941 a young diplomat who smoked too much and who was physically quite inert received a dose of the 6x benzoquinone solution with a trace of diphenoquinone for a chronic sinusitis with hay fever. He did very well for a few weeks, after which a negative phase set in, that tended to persist, so another dose was given. But it aggravated the symptoms still more. Even a 12x dilution of the diphenoquinone may show this effect. The serial system of carbonyl groups was then used with satisfactory results and the reports coming at times since then, have indicated no more trouble.

It is therefore well to be acquainted with each feature of the reagent structure, and the patient should be studied with these details in mind. This is important not simply at the start of the treatment, but one and a half and two years later when reactions pop up long after one thinks the cure is complete, and these reactions hang on continuously until the dose is repeated, preferably with one of the serial systems of carbonyl groups.

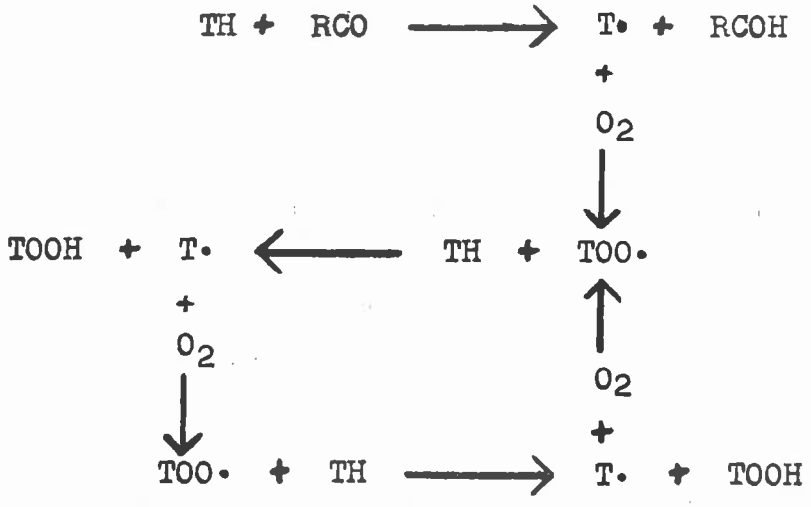
The serial systems of carbonyl groups appear to contribute their own electrons to the one engaged in the dehydrogenation, and as seen in Beta naphthaquinone and in orthobenzoquinone greater reactivity is had than where the carbonyl group is activated by ethylene conjugates only. In the separation of a virus from a host cell during the nearly always presence of hypoxia the free radical formed by the dehydrogenation could add to a double bond of the reagent and thus join it to the host cell, and it would not be freed of anything if a quinone were

the dehydrogenating reagent. But this would not be the case where serial systems of carbonyl groups are used. The virus or combined toxin would separate as a carbonyl compound and not carry a chain reaction, so the dose would be in the 6x range or the 9x range. Or if a quinone is used the dose would be more dilute. Of course as soon as the cell is freed so its carbonyl function is re-established it initiates its own oxidation chains.

Another advantage of the serial systems of carbonyl groups is that they can be prepared with free radical terminals, and when during anoxia they accomplish a dehydrogenation the free radical formed is not peroxidized and can add to the free radical of the carbonyl chain. This gives the toxin or virus plenty of highly reactive carbonyl groups from which to accomplish separation from the host vital structure, and to do so as a compound with a carbonyl terminal, which removes its toxicity and may even give it an autonomous energy producing existence with which it can serve the Great Biological Economy instead of being able to exist only as a parasite. Thus whether the homeopathicity is the matching of a free radical of the corrective reagent with that of the pathogen during hypoxia or anoxia, or whether the corrective free radical is formed via a carbonyl group of the reagent within the pathogen that can carry thereafter antitoxic and curative oxidation chains, in both the free radical mechanism and chain process is envolved. During the pathogenesis hypoxia or anoxia is the promoting factor, and during recovery oxygen is the ultimate requirement. To create the best field for action of the recovery mechanism, dietary and hygienic factors must be considered. They will be given due consideration later in this book. (Koch, Cancer Journal, October 1924; Carneire and Souza, Veterinaria, March 1950, p. 26.) This type of reaction may be outlined as follows:

Let T represent the toxin or integrated virus and H its most mobile hydrogen atom; TH would then be the structure to be attacked. Let RCO represent the remedy reagent with its activated carbonyl group which dehydrogenates the toxin or virus. The free radical T. is thus produced and this free radical adds molecular oxygen to form a peroxide free radical which

can dehydrogenate another molecule of toxin and thus keep the reaction chain going. It serves as the carrier or antitoxin state of structure of the pathogen. The toxin is thus converted into an antitoxin.

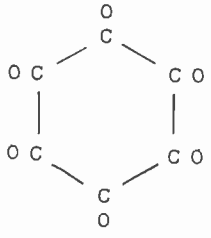


In summary one may say that full range dehydrogenating power is desired as the initial step of the therapy, so that toxins of all grades may be attacked for peroxidation. Then too, when oxygen supply is not sufficient to follow through, the molecular structures with highly electrophilic double bonds as the various quinones we have introduced to medicine may be used, and these are chosen to suit the features evident in each case. Finally the free radical terminals of long chains of carbonyl groups may be required, or even the free radicals exhibited mesomerically by resonance hybrids contributing to certain structures. These structures have activities of each type that is required by our hypothesis, and the case observations that follow demonstrate this.

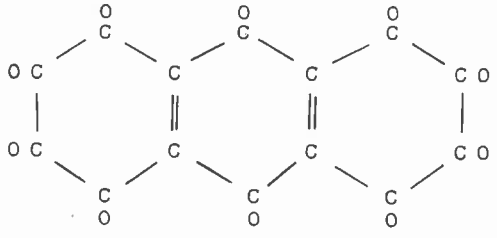
PLATE III

Structural Formulae of Reagents

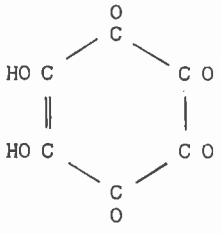
Structural Formulae of Reagents



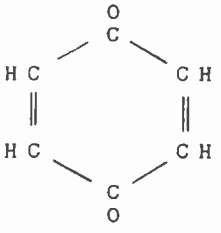
Triquinoyl



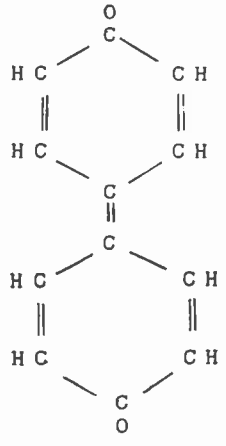
Compound (C)



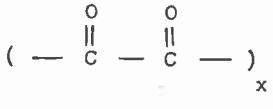
Rhodozonic Acid



Benzoquinone



Diphenoquinone



Serial System of
Carbonyl groups with
free radical terminals.

THE DILUTIONS OF THE SOLUTIONS USED

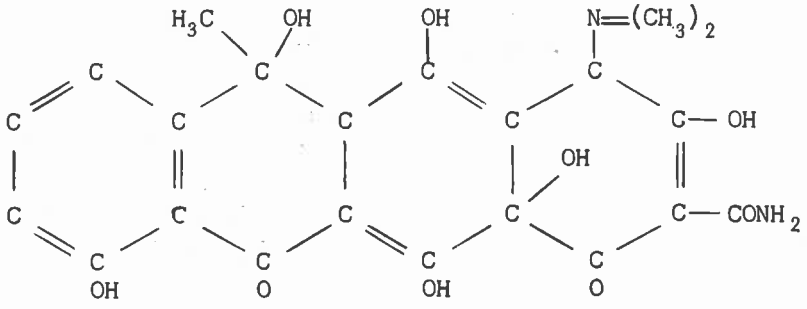
This subject has been a stumbling block to many physicians who do not understand the principle of high dilutions. Some remedies must be used as concentratedly as possible, while others serve the body chemistry in very high dilutions, as catalysts. A dilution of one part to a trillion of solvent, is very high, yet each cubic centimeter carries many millions of molecules, and in many physiological reactions this is the dose that is found to work best. As we pointed out formerly, Kuhn and others showed that the dilutions of crocin worked vigorously in that range, and that one molecule per algae cell was sufficient to activate its sex functions. Other vital agents require high dilutions. Therefore when a cubic centimeter carries millions of molecules there are more than the one required to accomplish a dehydrogenation and start an oxidation chain which will grow with geometric speed.

One should therefore turn his attention to the other phase of the problem, namely, **how concentrated dare the solution be for the best service?** This will depend upon the molecular structure, and in general, the more the carbonyl groups as compared with the number of ethylene linkages in the molecule, the more concentrated the solution may be. For the serial systems of carbonyl groups, the reagent may be diluted to one to a million. But where high double bond content predominates as in diphenoquinone, the dilution should be one to a trillion, lest the double bonds be too close at hand and absorb the free radical produced by the lonely carbonyl groups. It is easy to see that higher concentration here would give no results at all and the excess of double bonds could even inactivate the free radicals of normal oxidation processes and make the condition worse than it was. This principle also governs the repetition of the dose. A second dose may defeat the whole program of recovery. Other factors that determine the dilution and amount are, the extent of the disease, the amount of oxygen present, the circulatory advantage, the development of the musculature, the time the disease is established. What other therapies have been previously employed, the amount of narcotic consumed, the adequacy of kidney and gastro-intestinal function, and the ability of the red blood

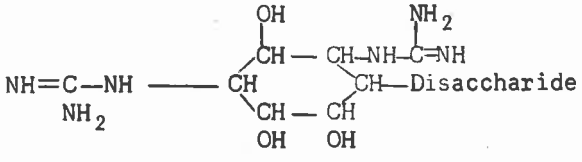
cells to crenate in a one percent salt solution must also be considered.

PLATE IV

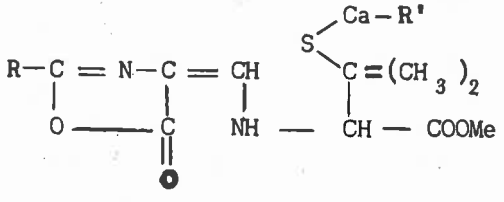
Structures Of Most Efficient Antibiotics And Ketosteroidss



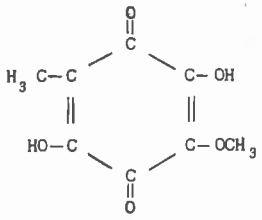
Terramycin



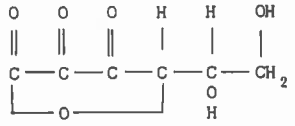
Streptomycin with its abundant activated amine groups



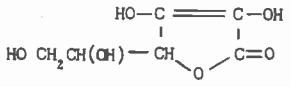
Penicillin (active salt)



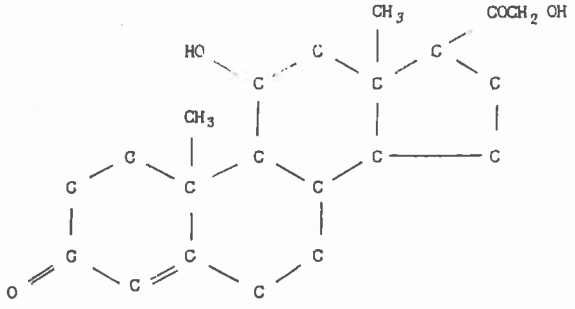
Spinulosin



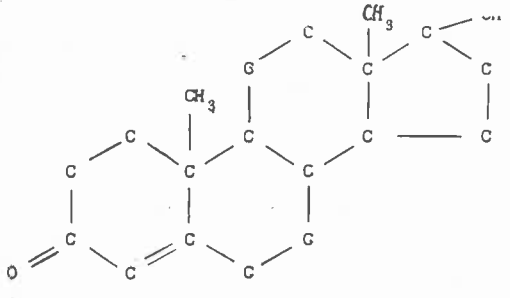
Dehydroascorbic Acid



Ascorbic Acid



Corticosterone



Testosterone

COMPARISON WITH ANTIBIOTICS

Inspection of the formula of the best known antibiotics reveals that there are two sets of groups which are active. These are the activated carbonyl group and the activated amine group, both of which we studied a quarter of a century before antibiotics were discovered. There are two dominant types as well as combination of both as shown in the utilization of these groups. Spinulosin, Phoenicin, Citrinin and Clavicin which present the carbonyl group activated by conjugation with an ethylenic linkage are of one type. Streptomycin presenting the activated amine group only, in two guanidin groups as substituents of cyclohexane represents the other extreme. Then in Penicillin, and Terramycin we have the both combined. However the amine groups are protected from activity by methyl substitution and by carbonyl proximity in Terramycin, while in Penicillin a bond cleavage is required for its liberation. They are then activated by conjugations with ethylenic double bonds. Streptomycin's amine groups are activated by conjugation with the double bonds of imide groups. Penicillin and Terramycin both present carbonyl groups. These are well activated by double bond conjugations in Terramycin, and become activated in Penicillin on forming the salt. Thus as the amine group acts on a molecule for molecule condensation basis, its value depends on the presence of a sufficient number of molecules to inactivate all germ respiration. It must be used in fairly high concentration. The carbonyl group activity being catalytic in line with our hypothesis should act best in high dilution, and this is proven to be the case since high dilutions are constructive and stimulate tissue development. Thus one can suppress the amine action by dilution while at the same time increasing the catalytic oxidative growth promoting action. These facts in addition to the phenomena of the ketosteroids give splendid support to our old hypothesis.

The recent hydrazide drugs belong to this discussion, being related to streptomycin in their activation of amine activity. While they are a great improvement on the confusions of the past in tuberculosis therapy, they still carry the weakness of the toxic amine group as their active agent. The conse-

quences of its use is not yet fully seen, but when compared with the carbonyl group to secure real harmless recoveries, one sees that constructive therapy is far superior. The few case histories given later which were used in the Federal Court establishes this point very nicely. No germ can mutate against being cured of its defects either, while they do mutate against further injury as by the hydrazide and amine therapies. When the clinical material is studied as given later, it will be seen that after the carbonyl treatment the germ becomes harmless, be it a most virulent tubercule bacillus, a leprosy bacillus, a pus germ, or some deadly virus. Since germs that caused the gangrenous lesions may increase in number during the healing, and the detoxication of the patient, when there is much tissue debris to be removed, and as they rapidly decrease in number where the lesion is mostly inflammatory with little tissue destruction, we have concluded that after the treatment they are able to aid in the cleaning up process and the detoxication. This is seen in dairy cattle with gangrenous mastitis, and during the recovery from tuberculosis with huge cavitations. One sees tubercule bacilli undergoing digestion and fragmentation in the large mononuclears and mast cells which themselves appear healthy and uninjured. When the clearing out process is finished the germs disappear even though the radiographs show large cavities with smooth walls that are undergoing absorption. The radiographs that follow show this process with the replacement of the lesion by normal lung parenchyma.

To observe the amino group toxicities stepped up to the highest pitch of action by different electron contributing atomic arrangements, and specific more or less for two different tissue cell mechanisms, mitotic and contractile, one may compare the structures of acetylaminofluorene, and trimethyl melamine. Of all the carcinogens, acetylaminofluorene offers the most active of carbonyl inhibitors and of all the heart poisons trimethyl melamine is the most active in my experience. The amine groups in both are protected by substituents that can be rapidly removed leaving the nascent amine group alive to make its condensations with carbonyl, the most active of which in the tissue cell are those just mentioned. Tissue specificity probably de-

pends upon the ability to liberate the amine group of its substituents right in the cell where it can then act. Of course the first action of the carcinogen should be to paralyse normal mitosis in some such way as we have diagrammed, and when the virus or some incompletely combusted germ or cell metabolite steps in the neoplastic process is put into action.

Amines produced in the neoplastic lesion by molds show two antibiotic effects favoring neoplasia. One is the inactivation of the protective functional carbonyl group, and the other, the gelation of the inter and intracellular colloids that interferes with oxygen transport. This double effect is observed in the lesser use of oxygen by the neoplastic lesion and the greater amount of oxygen in the venous blood issuing from it than is found in the venous blood coming from normal tissue.

The importance of carbonyl of high activation in the use of oxygen in living tissues is also seen in the boost to the use of oxygen by surviving tissue slices when catalytic dilutions of the reagents described in this text are supplied. Thus the oxygen use has been boosted in normal and neoplastic tissues, in yeasts and molds, and some bacteria, as much as 40% at times. This effect on tissues may not take place to such a great extent in the unhindered circulatory set-up, where amines can be washed out of the way so as not to hinder the functional carbonyl activity in initiating oxidation chains. While such experimental data are indicative, they are not quantitatively transposable from the events in an improvised flask to those in the wonderously balanced organism. Such considerations should be applied to the Krebs cycle interpretations also.

The great increase in cancer, even in children, since the sulpha and mold antibiotic amines have been given such wide therapeutic use is significant since (amine producing) molds are now recognized as present in cancer lesions, and the degenerative effects of antibiotics on tissues is beginning to be known.

DIAMINE OXIDASE

In our search for reagents provided by Nature that resemble our synthetic products we find among plants a number of highly active carbonyl compounds, especially in Brazil.

One of these is a plant that shows reflex motion. If one end of a stem is touched the whole section closes up. It is called the "Dormideira" plant. An infusion is used by the natives to cure gonorrhea and other infections of the uterus. The well known Camomile plant offers an active carbonyl group too. So does the dandelion, the yellow colored material of butterfly wings and many other expressions of Nature.

The most pertinent example however is the Diamine Oxidase, which is an oxidation catalyst found in the intestinal mucosa, the placenta, the lactating mammary gland, and in high content in the blood of pregnant women so long as the fetus is developing. When the content drops, abortion threatens. Our knowledge of this substance was developed principally by Zeller, and confirmed by Best, McHenry, Werle, and others. Zeller showed that this enzyme serves as a dehydrogenator by virtue of its carbonyl groups, producing imides that split off as ammonia similarly to the oxidative deamination of amino acids. The intermediate hydrogen acceptor is Flavine-adenine dinucleotide with oxygen the ultimate hydrogen acceptor yielding hydrogen peroxide. (See, "The Enzymes", edited by Sumner and Myrbaek, Vol. II, Part I, page 554, 1951.) No doubt placenta extracts owe their protective action in large part to this enzyme. Diamine Oxidase destroys histamine and has been named histaminase by Best. Its importance in the combat against allergies as a Natural Immunity reagent is well recognized. We believe the virtues of Queen Bee Jelly reside in similar substances of high unsaturation.

Zeller's demonstration that diamine Oxidase works through its carbonyl group as a dehydrogenator (Advances in Enzymology, vol. 2, page 93, 1942) to detoxicate against toxic diamines, brings forth a proof from Nature of the validity of the process we accomplished synthetically and put into use over twenty years earlier. His work was reported at about the time the Federal Trade Commission brought injunction proceedings against us. It is their contention that our advertisement was "false and misleading" and that it constituted "false advertisements." Among the statements that they said were "false representations of material facts" is: "THE BASIS OF IMMUNITY is,

after all, the vital principle, the OXIDATION MECHANISM. When its catalysis ceases, death is the result. When its activity wanes, the toxins that support pathogenic germ activity, that produce allergy, or that cause cancer, are not destroyed in the body, and can execute their effects. All of these toxins depend upon their free valencies between carbon atoms, between carbon and oxygen, and between carbon and nitrogen for their pathogenic photochemic action." The Commission in effect denies that the oxidation mechanism and oxidation catalysis have a real important place in the maintenance of health and in the protection against disease. In the same breath they "ordain" that one cannot die of asphyxia.

Thus in Nature the carbonyl group has played a detoxication role against amines, and as we pointed out decades ago the amine group serves to block the carbonyl activity toward the oxidative destruction of amino acids in the preservation of the protein structure. Thus the amino acid is made up with the carbonyl group flanked by an amine group, both of which combine to constitute the unions of the protein structure. Our thesis deals with other highly important antagonistic and complimentary activities of these two groups. But it was not until thirty-five years ago that we could use synthetic carbonyl structures, and build them up as we wished, to meet any pathogen of importance be they amine compounds or polarity reagents depending upon free radical phenomena. These matters will be demonstrated as we go along.

THE ROLE OF FOCAL INFECTION

The Buried Tonsil

Many persons going beyond forty years of age develop a variety of symptoms and tissue changes that are puzzles to all the specialists they consult. These affairs range from debilitating coronary deficiencies with occasional occlusion crises, to painful varicosities, with or without invaliding necrotic ulcers. Indeed, cancer of the breast may be a sequel to the basic defect.

In such cases the right tonsil generally is found enlarged and rolled out of its hammock with the crypts open and drain-

ing. The left tonsil, most often, or it may be the right or both, are deeply fibrosed and calcified perhaps too and buried in their hammocks and firmly fixed. The fibrosis may be quite extensive, or only enough to hold the tonsil deep in its recess. Some cervical gland hardening may also be observed, but generally the complaint is that the "thyroid" bothers them, and they never are bothered with sore throat on the affected side. Pains however may be severe enough to extend to the back of the neck and call for complaint. The infection is well walled in and calcified generally, and surgical removal would run serious hemorrhage dangers, and would have to be too radical and deforming to be complete. The circulation to the inside that holds the infection is of course much reduced and the area is quite hypoxic so that the toxins evolved by the germ have a good chance to polymerize. Slowly the toxins diffuse into the lymph and blood circulations and may follow the lymphatics against the stream as it were into the glandular tissue of the breast. The toxins circulate quite generally too and as they polymerize to increasing molecular weights they produce a variety of symptoms, that affect each and every tissue of the body. The variety of complaints is too great to be dealt with here. The present day remedy used so often is cortisone for the arthritis, for the skin lesions, for the headaches and for anything else, and gratifying relief is had in many instances. However the pathology keeps on progressing until invalidism and inability to buy drugs calls for a change. The effects of the Cortisone may have prohibited its further use too.

When we see such a case, the examination of the throat tells the program of illness that has been suffered and after a few questions the rest can be given quite accurately without further inquiry. The treatment is satisfactorily one of the quinones, the diphenoquinone serving best. The reactions are rather severe after its use in the 6x dilution, so the 12x may be used best with frail people whose heredity has predisposed them. However where the reactions are too troublesome the serial system of carbonyl groups can be used with even better results and less reaction. The value of the reactions is to indicate the position of the patient in the recovery course and to show that the cure is

not complete before it is safe to leave the regime for the old habits that contributed to the illness.

Generally in three or four weeks the buried tonsil is loose in its hammock and falls forward under favorable conditions. The neck fibrosis is gone and the "thyroid" causes no more trouble. The heart action or high blood pressure may be much improved and the gall bladder is forgotten. In the course of a few months normalcy is established, but there may be some heavy febrile reactions at the third, sixth, ninth, or twelfth weeks or even later. We have seen large cancers of the ovary biopsied to show very malignant adenocarcinoma clear completely while the tonsil was normalizing. The last reaction to come is generally a severe sore throat on the side of the buried tonsil, and where neoplastic disease is established, this reaction comes after the growth is absorbed totally or for the most part. The local inflammation resembles one of years earlier that most often can be remembered. Many people lose much of the pleasure of life because of such buried infection. It is "silent," but not inactive. Its cure should prevent such disasters as cancer in many instances, and arthritis, varicose ulcers, etc., in others.

CHAPTER V

THE RECOVERY MECHANISM

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 week, 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 in detail in the completed text. The case histories that follow demonstrate the return of function. What needs to be illustrated here is the adaption of the mole-

cular structure of the reagent to the status of the patient. We will outline a failure caused by error in judgment, contrast it with a success where good selection accomplished exactly what was required. The selection of the molecular structure is not made to fit the disease, but rather to fit the patient whatever the disease may be, for in all, the pathogenic basis is attacked.

The pathogenic basis includes the change in the steric configuration of the host cell functional mechanism caused by its combination with the pathogen. For each disease the difference is sufficient to require a different steric configuration in the structure of the remedial reagent to give optimum dehydrogenating action on the pathogen, as it is positioned in the functional mechanism. The details will be given later.

Since hypoxia and anoxia are essential features of the pathogenesis, one might look upon different patients as composites of oxic, hypoxic and anoxic centers, the latter associated with the disease lesions. The metabolism in such centers may be compared with that going on in tissue slices during their occupancy of a respiratory chamber. In spite of the agitation aids to oxygen distribution, hypoxic centers permit the dehydrogenation of succinate to fumarate which diffuses out to oxic areas where the carbonyl group it offers, as activated by conjugation with ethylene linkages, can initiate oxidation chains through dehydrogenation. Before we ever suspected fumarate to carry such a function of preserving the aerobic fate of tissue metabolism when the field could again receive oxygen, we used benzoquinone to accomplish this effect in the treatment of disease. Also maleic anhydride with its double bonds all in the same plane and attackable at right angles by the colliding atomic group, showed some good carbonyl effects. Here we looked for the diffusion of the reagent into the suboxic and hypoxic centers of the lesions where the dehydrogenations of the pathogen would inactivate it as previously described, and where access of oxygen at the periphery produced the peroxide free radicals that carried the oxidation chains forward. The colloidal interference to oxygen distribution would then disappear and oxygen enter deeper into the lesion until oxidation chains were readily formed

throughout. The delay of from one to three days in initiating the febrile reactions of the recovery process no doubt depend to some extent upon this procedure. But in tissue slices the inhibitant is the mechanical block to oxygen transport as well as colloidal changes as the acidity develops as the seconds go by. Fumarate would therefore increase with the duration of the experiment within limits, even though it would tend to stimulate oxidation as we suggested. By the therapeutic use of the activated carbonyl group one would expect peroxide free radicals to show ever increasing effects in the hastening of the recovery procedures, simply because the improvement in the circulation of blood with oxygen and other curative agents of the tissue economy gave the lesion a chance to normalize while the tissue slice kept on dying with its reversed enzymatic equilibria, and approached rigor mortis changes. Fumarate could be interpreted as an anaerobic product or hypoxic product of metabolism that tended to preserve the aerobic status of metabolism when oxygen was again available. Tissue slice data must be interpreted in the light of the conditions that determine their production, which are abnormal to start with, and increasingly more so as the observation proceeds. They cannot determine the events of a recovery from cancer.

CLINICAL SECTION

Clinical Cases

Most of the case histories given in this section were used in the Federal Court trials and presented before the Federal Trade Commission. Some of this testimony has been paraphrased and pertinent parts of some of the documents have been copied or reproduced from the records to supplement the case histories. A few other cases that contain pertinent data, but were not used in the courts, are also given. Only the useful data is submitted here to illustrate or prove some point in our philosophy. This data should be studied.

They show the common basis of tissue atrophy and of neoplasia and indicate that both are based on the same atomic disturbance, and how this is expressed in quite opposite ways. High

mobility of a hydrogen atom alpha placed to double bonds, dehydrogenation and peroxidation, give the pathogen a chance to separate off from the host cell, with a terminal carbonyl group and be nonparasitic. When the induced oxidation rate is high, the toxic units are burned out of the way so fast they barely give indication of their presence or may be missed altogether. But where the oxidations are hindered, these symptoms may not only show up in reversed order so as to be recognized, but they may linger too long, and show that the recovery progress needs a boost, and that the dose should be repeated. This time, however, the dose must be in a very much higher dilution, millions of times more dilute than the first dose in some cases and be given only if the **crenation test** of the blood shows **that it is needed.**

In these case histories that follow, the therapeutic reagent is not identified by a drug trade name as was done in the court record, but is simply referred to as a treatment or injection of serially arranged carbonyl groups, carbonyl oxidation initiators, serial system of carbonyl groups, carbonyl catalyst, carbonyls, activated carbonyl structures, etc. These terms refer primarily to a mixture of closed chain and open chain polymeres of the basic hypothetical structure of (O=C=C=O)x and of (O=C=C=C=O)x of various molecular weights and containing activated carbonyl groups and free radical structures.

When a quinone is used, it is so designated and the type of quinone is identified by its name.

Cases that were used in the Federal Courts will be identified by a *, and cases that were used in the Federal Trade Commission hearings will be identified by a **.

CHAPTER VI

ANAPLASIA, HYPERPLASIA, NEOPLASIA, DYSFUNCTION

Their Common Basis and Correction

PRIMARY ATROPHY OF THE OPTIC NERVE AND RETINA* **

Sequel to Scarlet Fever

Treated in collaboration with
Dr. Frank Richards

Anaplasia As In Diagram V, (b)

R. J., age 14, family history negative to cancer and tuberculosis. The pretreatment control period and diagnosis are well described in the correspondence between the Henry Ford Hospital and Jennings Hospital of Detroit, as follows:

HENRY FORD HOSPITAL
DETROIT, MICHIGAN

Henry A. Du . . ., M. D. September 10, 1946
7815 E. Jefferson Avenue
Detroit, Michigan

Re: R. J.
Case No. 453242

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 of the right eye. Vision without correction of both eyes revealed a normal lense. In the macular area there was a chorioretinitis which fits the description given by you in your letter. The left eye was normal to fundoscopic examination. Tangent screen examination was done which showed an absolute scotoma

in the supra-central region. The periphery normal red field 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 of OT; 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 in both antra and probably the ethmoids and frontals as well, and were suggestive of a pansinusitis. There was only a moderate increase in the bronchovascular markings in the base.

Blood count showed on 4-16-46 a hemoglobin of 13.5, white blood cells 10,000, 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 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

At my examination, the absolute scotoma reported in this case was an area of retina and optic nerve atrophy confirmed by the blindness to include the central visual field and much of the upper part of the retina, leaving a ring of periferal tissue

that showed some function. However, the eye was blind for practical purposes, since he could not read or make out objects with it, nor could he get much help from the periferal vision. This condition had been progressing rapidly.

The injection of two cc. of the 12X dilution of carbonyl catalyst, the serially arranged carbonyl groups, was made on April 26, 1946. The headaches attributed to sinus infection, and the drainage from the sinuses soon stopped. The usually frequent sore throats returned only twice. This was during two different reaction periods. The recovery was characterized by the usual reactons for the infection foci in the tonsils and sinuses at the twelfth, twenty-fourth, thirty-sixth, and sixtieth weeks. During the sixtieth week reaction, the fever rose to 103 degrees and there was the usual sore throat of the past, but a rash developed similar to that of scarlet fever. It lasted twelve hours and disappeared. His health seemed to be perfect thereafter. This return of a scarlet fever rash in a person who had the infection only slightly in spite of exposure is not unique. We have observed such occurrences after treatment in similar cases where they had never developed scarlet fever, in spite of exposure, and where the optic nerve atrophy also recovered under a dose of this treatment. We may attribute the nerve atrophy to chronic scarlet fever whose toxins attacked the optic nerve and did not leave the patient's tissues until they were burned down from the polymeric sequelae-producing form, through the erythema-producing monomeric phase, and then to complete harmlessness.

Examinations of the eye made by Dr. Du . . . found that the vision in the right eye was 20/400 in August 1946, 20/100 in June 1948 and 20/40 on October 27, 1948. This shows that improvement was continuing. This, we believe, demonstrates the progressive chain reaction type of recovery. Diagram V, (G), possibly pictures the pathology here.

RETINOBLASTOMA (GLIOMA OF THE EYES)* **

Treated in collaboration with
Dr. Garret Warnshuis

Neoplasia As In Diagram IV

Rita 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 . . . of Wichita, Kansas. He removed the eye. The pathological report is given below.

Within a year the right eye showed the same changes, and the same surgeon observed the same disease here that he had found in the left eye. Operation was not advised. The patient was taken to a renowned ophthalmologist who made the diagnosis of retinoblastoma and referred her to me. The result of my examination and of Dr. H . . . was likewise retinoblastoma and we found that about one-third of the retina was involved as well as some of the optic disc. The eyeball was bulging and somewhat 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 of light showing that a tumor was present within. The area where the left eye was removed was not healthy, but showed neoplastic degeneration in a minor degree. Both foci disturbed her. At this time, November 25, 1935, Rita was over two years old and we gave her one cubic centimeter of the 12X dilution of the serially arranged carbonyl groups by intramuscular injection. The irritation of the affected eye and of the area where the left eye was removed from the socket quickly disappeared after the injection. Every third week she suffered a reaction with general aching of her muscles, some slight fever and considerably more irritation of the affected eye and the operation area. Each lasted a few days only and was followed by improvement. On Aug. 18, 1936, we could not see any bad effects. The iris contracted to light as it normally should do. There was no reflex behind the pupil, no gray curtain, as it were, behind the eye to shut off vision, as far as we could see. Evidently she was able to see at that time all right. She appeared to have recovered, but to reinforce the recovery process she was given a second treatment. We con-

Wichita Hospital

5/5/'34

PATHOLOGICAL REPORT File No. 4120

Name L , Rita Coleen Room 111 . Case No. 1-2668

Age 23 mo. Sex F. Race W.

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

Form 20—Hospital & Physicians Record Co., Wichita, Kansas

sidered her cured after recognized five year period. Her vision has been fully restored in that eye. She went through school at the head of her classes regularly and is a beautiful normal young lady now. Follow-up report of June, 1950, records perfect health and vision and graduation at the head of her class in Junior College. She was married in 1953, and remains in good health. The same type of carbonyl reagent that cured the optic atrophy case cured the retinoblastoma case. Our thesis is confirmed. Scarlet fever or an associated virus cannot be proved here, but virus integrating with embryonic retinal cells cannot be denied.

MALIGNANT SYMPATHOGONIOMA
In A Baby Boy

Treated in collaboration with
Dr. Baldor

Neoplasia As In Diagram IV

John L., age 13 months, developed a tumor in the abdomen

that required an exploratory operation on September 25, 1951. It was found to be retroperitoneal and had infiltrated the surrounding tissues too widely to permit its removal. A biopsy was taken. The report is reproduced below.

On October 6 examination revealed a visible bulging of the abdomen at the umbilical region which palpation revealed to be the size of an ordinary Florida grapefruit, about 15 to 18 cms.

SAINT ANTHONY'S HOSPITAL, INC.
SAINT PETERSBURG 6, FLORIDA

PATHOLOGY REPORT

Name L , Baby John
Room No. 0-1598-51
Date September 25, 1951 Age Sex Male S. M. W. D.
Physician F. H. Langley, M. D.
Clinical Resume: TO
ABDOMINAL MASS OF RECENT DURATION, PROVED NOT BE THE BLADDER. TESTICLES PRESENT IN THE SCROTUM AND APPARENTLY NORMAL.

SPECIMEN: BIOPSY OF RETROPERITONEAL TUMOR

GROSS: The specimen measures 5 x 3 x 4 cm. 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 separates 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 emirregular 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.

LED/fa

F. N. Carmichael
Pathologist

Physicians' Record Co., Chicago 5 - 5813-1249 10M

in diameter, firmly fixed to the surrounding structures. The stools were bloody, and the blood count agreed with his pallor, showing a red cell count of 2,300,000, and a hemoglobin of 52 per cent. The next day, October 7, he received from Dr. Julian Baldor an injection of the linear arrangement of carbonyl groups, 12X dilution, with free radical terminals. Recovery began promptly and by the end of the first year no more growth could be palpated.

On May 5, while in good health, he was run over by an automobile and sustained a broken leg and abdominal injuries. While in the hospital for repair of the injuries, he was thoroughly examined by the surgeons who made the exploratory laparatomy and found free from any trace of palpable growth. He made a nice recovery and is in excellent health. On April 5, 1953, the red blood count was 4,750,000, and the hemoglobin 87.5 per cent.

The facts of this case were given by the surgeons under oath in court testimony at Tampa, Florida, with the operative findings of a malignant neoplasm that could not be removed. They removed a biopsy to identify the cellular type of neoplasm and origin. They also testified to the cured state as found in May, 1953. It shows that other factors than trauma are required to cause cancer. He continues to enjoy good health. (1956).

EUNUCHOIDISM**

Treated in collaboration with
Dr. Catherine S.

Anaplasia As In Diagram II

J. S., at age 14, subject to infections of the respiratory tract and skin, presented marked obesity, female shape, very dull mentality and infantile penis with undescended testicles. He had been under thyroid and pituitary hormone therapy from the age of 10, in 1931, to 1935, without improving his development physically or mentally or increasing his resistance to disease. Examination at the age of 14, by the writer, and testified to by Dr. S . . ., showed that the testicles had not even entered

the canals and so, from all standpoints and authorities, the disease was of a type that is not helped by modern therapy. He was given an injection of the carbonyl catalyst, and in three months definite improvement had already been established. Dr. S . . . 's testimony shows that within three months he lost weight and the left testicle was observed to be entering the scrotum. Within six months after treatment both had successfully passed through the canals into the scrotum and were normalizing in size, while at the same time the penis began to develop. He grew taller, his hips reduced and his shoulders broadened out and his jaw developed a normal prominence. The pubic hair appeared in normal distribution with a stream toward the umbilicus. His mentality changed entirely so that he became alert and did very well in his school work, soon making up for his backward position. By the age of 21 he was a good athlete and student, entered the army and was quickly promoted to a corporal. At the time the testimony was given he was in the Army Law School, making fine progress, married, living a normal, happy sex life and raising children of his own. He has two, a boy and a girl. Both normally developed physically. The inhibiting factor was therefore removed completely.

Here we have the inhibition of development probably based upon an infection focus in the respiratory tract. The history provides no unquestioned relation to scarlet fever infection, although a suspicious rash showed up after the twelfth week reaction and was discounted as a food affair. However, the continued colds from infancy point to respiratory infection and may be a suppressed scarlet fever. At any rate, the toxin that inhibted the development of this section of the urogenital ridge at a late date did clear up, as did other signs of infection, and the inhibition ceased as soon as an effective carbonyl catalysis was established.

The following case of cancer of the testis, in contrast, shows the neoplastic expression of carbonyl deficiency.

CANCER OF THE TESTIS

Neoplasia as In Diagram IV

Treated in collaboration with
Dr. Alpheus Hoyt

Mr. T. was 38 in June, 1925, when treated with carbonyl catalysts. The testis had become neoplastic six months previous to 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 biopsy report read again "medullary carcinoma of the testis." Recurrence was not long in becoming evident and with much more rapid spread of the disease. The abdomen was again opened, but was found so inoperably involved that the opening was closed after only removing a biopsy. This biopsy report read, "Carcinoma, probably secondary to previous carcinoma of testis, as the cells are histologically similar." At this time he was rather emaciated and exhausted and a general cachexia called for a hopeless prognosis. Through the intervention of Dr. Alpheus Hoyt of New York City this patient was referred to us as a test case by the late Dr. James Ewing, the well-known cancerologist. Mr. T. was still well in 1946 when he offered to testify for us in the Federal Court. This was twenty-one years after he received the treatment during the terminal stage of the diseases.

ECLAMPSIA**

Treated in collaboration with
Dr. Baldor

Function Suppression As In Diagram V (b)

Mrs. D. was married seven years and had never been able to go through a pregnancy. Three previous pregnancies had to be terminated because of eclampsia before the end of the second month. In each instance she vomited profusely, formed less and less urine until only blood came from the kidneys.

There was much salivation. The vomiting increased until nothing but blood was thrown out and she developed convulsions and coma. She did everything to keep the baby, as it was her object in life to have one. Of course, abortion was necessary in each instance. It is well known too, that each such experience makes the case more severe. The kidney function is greatly reduced each time and the next pregnancy is more certain to call for earlier abortion. This is what happened in the fourth pregnancy, now under discussion. The symptoms reached the coma stage after going through the others as described. It was then that the attempt was made to overcome the toxemia with the serially arranged carbonyl group reagent. In twenty-four hours the vomiting decreased from twenty times a day to two times. After seventy-two hours she began passing a half liter of urine a day. This urine still carried blood and albumen. In four days she passed a full quart of urine a day. The vomiting had ceased entirely within two weeks, but she still salivated a large quantity a day. During the third week vomiting again started, so another dose was given. She cleared up completely thereafter and carried her baby without adverse symptoms into the seventh month, when she had an accident and miscarriage threatened. Eclampsia symptoms, however did not return. She was delivered of a perfectly healthy premature baby of five and one-half pounds. She retained good health and was able to make a trip to Georgia to visit her relatives soon after the delivery care was over.

This case represents our usual experience in this deficiency. It shows not only the return of function that could be considered normal for the patient, but an added increase able to take care of the products of metabolism excreted by the fetus into the patient's blood stream.

TOXIC AND STRUCTURAL TISSUE INJURIES AND THEIR CORRECTION

Postpneumonia Glomerular Nephritis**

Treated in collaboration with
Dr. Evans, the father.

Functional Aggravation As In Diagram II

The case of T. E., age 4 years, the son of Dr. Evans.

He had enjoyed good health until he had a severe bilateral confluent broncho-pneumonia in 1938 and with good care made a fair recovery. Immediately following the major pneumonia state, an acute glomerular nephritis set in rather suddenly and progressed rapidly to its terminal stage. His father, a physician of very good judgment and wide experience, had left the boy Sunday night in good condition. Early Monday morning the nurse called him to the hospital for his young son Tom, was in convulsions. They lasted several hours and were followed by visual symptoms, delirium, oedema that rapidly increased so that in two days the contours of the chin and neck were obscured and other malformations appeared generally. At the time of the first convulsion the blood non-protein nitrogen was found to be 74.6, and the blood pressure 146/68. In the afternoon of the same day the blood pressure was found to have increased to 160/100, and two days later, when the second convulsion occurred, it was 180/130. The leucocyte count was 23,000. The second convulsion occurred Wednesday morning and was more severe than the first; delirium had preceded it, the oedema had increased, and doubtless had also affected the brain. Coma followed this convulsion and at this time the carbonyl oxidation initiators were administered, one cc. of one part to a trillion of water. Since no medical treatment was known at the time to be of help, he was not bothered much by any other measure before or after the treatment was given. The urinary output was lessening as compared to the intake, less than half being excreted, the other accounted for the oedema which was rather serious.

The injection was followed by the subsidence of the pathological findings. The mental symptoms quickly normalized, the oedema left, the blood pressure dropped to normal and the non-protein nitrogen came to 25 mgms. per cent, so that in a few days he was normal and so he remained. It took a few weeks to regain his strength and be on the go again; but the pressor substance that was rapidly developing without hindrance was quickly oxidized away with other oxidation inhibitors that the boy could not defend himself against, and so his life was saved. The toxins were converted into their own anti-toxins and kidney functions restored by the same oxidation process.

PSEUDO-NEOPLASTIC TISSUE INTOXICATION

Toxic Nodular Goitre**

Treated in collaboration with
Dr. Baldor

Function Aggravation As In Diagram II

This disease reveals the tissue hyperplasia and the perverted secretion of an essential endocrine gland which is incurable surgically, and which does not respond to iodine therapy. The defect is here demonstrated to be a defective oxidation catalysis. This patient was in extremus when given the treatment and the recovery is just what one should expect, if, in fact, an insufficiency in the tissue oxidations resulted from the action of a toxic inhibitor.

Mrs. M. J. was 35 in July, 1943, when examined by Dr. Julian Baldor. She had lost considerable weight recently, suffered from excessive nervousness and tremor, pains in the legs, terrific heart palpitation, shortness of breath and such extreme weakness that she had to be carried into the car to be brought to the doctor. The blood pressure had risen to 190/110, indicating toxically dangerous hypertension. There was throat constriction, and muscle twitching.

Examination revealed a woman with well-developed tremor and muscle twitching, exophthalmus, tachycardia, loss of weight

and strength and nodular thyroid enlargement. The basal metabolism rate was plus 104, July 8, 1943.

The heart was failing. The systolic pressure in the next few weeks dropped to 170, while the heart beat became weaker and the diastolic pressure stayed up to 110, showing that the fundamental toxic state was not abating, and the myocardial injury was increasing. She rapidly became very thin and weak, so that her muscles were too atrophied and incompetent to carry her and her husband had to carry her to the automobile and into the doctor's office.

From July 8 to November 10, 1943, iodine was tried with absolute rest and ice packs to the thyroid gland, but the condition steadily grew worse. The iodine was not succeeding in preparing her for operation and the heart had to be protected from further toxic injury. The nodules on the thyroid kept increasing.

Therefore two cc. of the 12X dilution serial system of carbonyl groups were given intramuscularly, and in a few days one could see that the trend of the disease was reversed.

The tremor improved, the heart beat was stronger, she could soon get around on her own strength; the whole picture steadily changed so that by the end of the eighteenth week she was back to work, a normal woman. The tremor was gone, the exophthalmus was gone, the nervousness was gone, the blood pressure was normal, 145/97 and later 140/80. The heart rate was 80 to 90. The thyroid was normal in size and the nodules had been absorbed. Instead of being carried and helped, she was out working carrying a heavy suitcase of goods. Her basal metabolism, March 16, 1944, four months after treatment was plus 6, a very fine normal.

TOXIC GOITRE WITH CANCER OF THE STOMACH

Impeded Function, Hyperplasia and Neoplasia Based on One Factor, Yielding to Same Atomic Arrangement in the Same Patient

Exemplifying Diagram II, IV, and V (b).

Mrs. W. comes from the goitre belt of Ohio. Cancer is

very frequently met in this locality. Although she was only 58 years old at the time of examination and treatment in September, 1929, she appeared to be at least seventy. It is worth notice that after her recovery she became "younger" in every way as the years passed. The photographs show the condition of the face, neck and eyes before and after the treatment.

Cancer was not recognized in the ancestry, but her husband died of cancer eight years previously, and her daughter, age 28, had a brain tumor that progressed as a malignant neoplasm subject to reversal by the same treatment that corrected the mother's defect.

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 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 exopthhalmus as shown in the photographs (reproduced below); 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.

One dose of two cc. of the 12x dilution of the 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.

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.

GRADE 4 SQUAMOUS CELL CANCER OF THE CERVIX UTERI* **

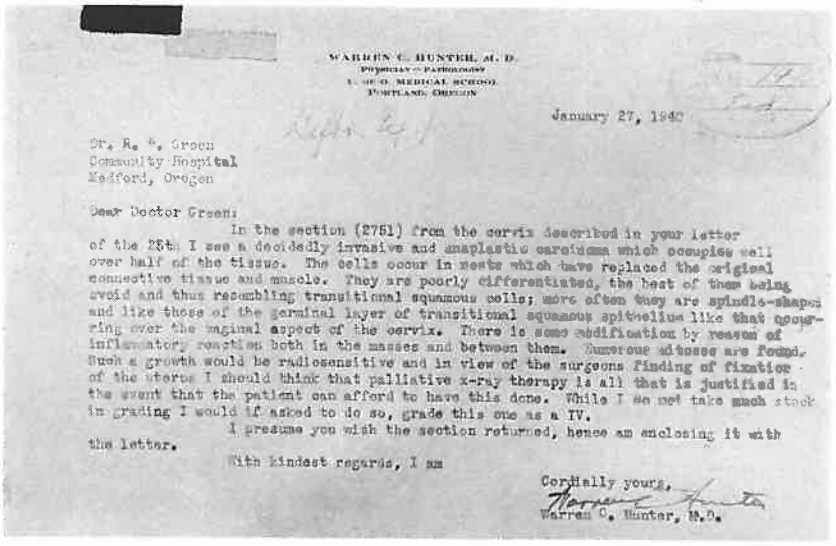
Treated in collaboration with
Dr. L . . . .

Neoplaia As In Diagram IV

Mrs. M. W. came under the care of Dr. L . . . . of Medford, Oregon, on January 12, 1940. Dr. L . . . . stated: "I examined her at that time 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. On January 19, I took her to the hospital and removed a section of tissue from the cervix, which was examined by Dr.

R. E. Green, following which the tissue was sent to Dr. W. C. Hunter, University of Oregon Medical School."

This slide was very carefully prepared by Dr. Green, the hospital pathologist and diagnosed by him as squamous cell cancer of the cervix uteri. Dr. Hunter testified that grading of tissue is done for the purpose of telling someone else how malignant the tumor is, that is, how rapidly growing. The degree of malignancy is indicated by the highest number given, which is IV. It runs from I to IV. So grade IV would be the highest degree of malignancy that we recognize. In his letter to Dr. Green he 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."



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

January 27, 1940

Dr. R. E. 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 carcinoma 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 oval 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 calcification 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.

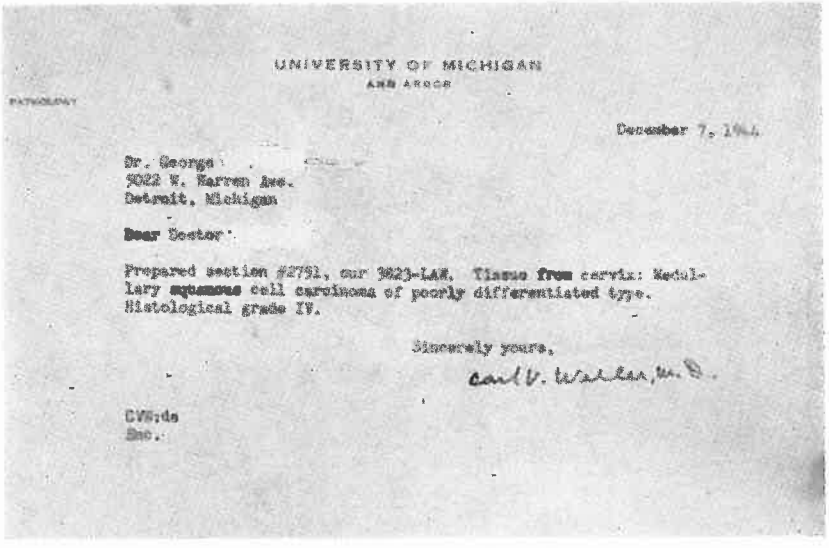
With kindest regards, I am

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

Dr. L.... testified that her cancerous condition would, if untreated, end her life within a year. 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 to be too extensive. It was too late for that sort of thing.

The case had already entered the cachexia stage as his

testimony reveals: "She had lost 30 pounds in six weeks complaining of general weakness and rather a poor color at the time." As Ewing states: "Characteristic cachexia in uterine cancer develops in the terminal stage of the generalized disease,



UNIVERSITY OF MICHIGAN
ANN ARBOR

PATHOLOGY

December 7, 1944

Dr. George
7022 W. Warren Ave.
Detroit, Michigan

Dear Doctor:

Prepared section #2791, our 3823-LAM. Tissue from cervix: Medullary squamous cell carcinoma of poorly differentiated type. Histological grade IV.

Sincerely yours,

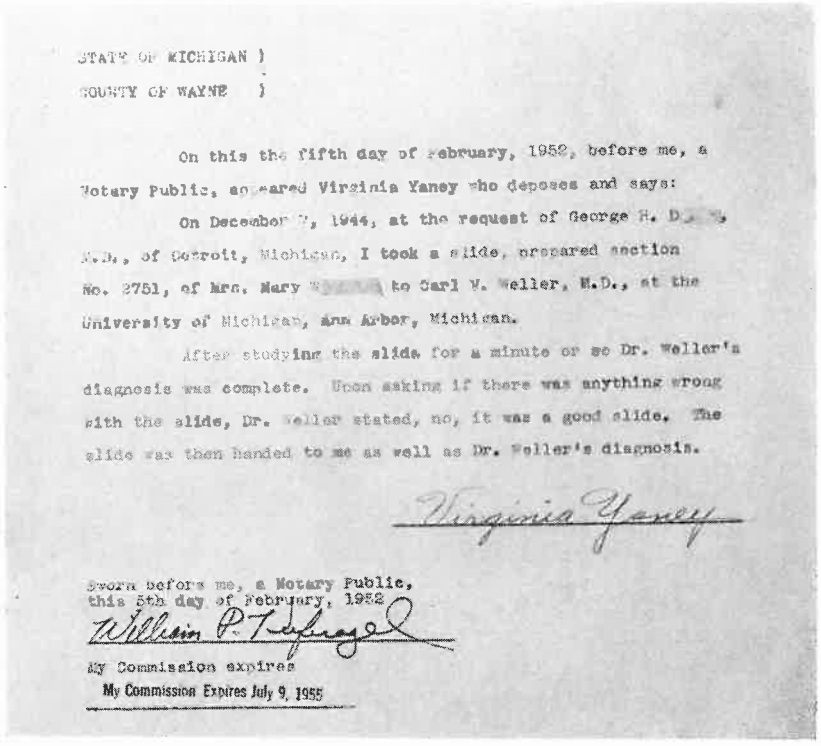
Carl V. Weller, M.D.

CVW:da
Enc.

but when the lesion is localized in the pelvis, cachexia is missing."

While under Dr. L . . . .'s care Mrs. W. M. received 3 injections of the serial system of carbonyl groups. These were given on March 20, 1940, December 30, 1940 and in October 1941. (It should be noted that this patient was on the Welfare. She

had pernicious anaemia and her diet was modified to permit her to eat liver. She also was given liver extract.)



STATE OF MICHIGAN)
COUNTY OF WAYNE)

On this the fifth day of February, 1952, before me, a Notary Public, appeared Virginia Yaney who deposes and says:

On December 7, 1944, at the request of George H. D[illegible], M.D., of Detroit, Michigan, I took a slide, prepared section No. 2751, of Mrs. Mary W[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, 1952
William P. Tefft

My Commission expires
My Commission Expires July 9, 1955

Mrs. M. W. remained under the medical care of Dr. L.... for approximately two and one-half years. At the time of the last vaginal examination by Dr. L.... which was made in the

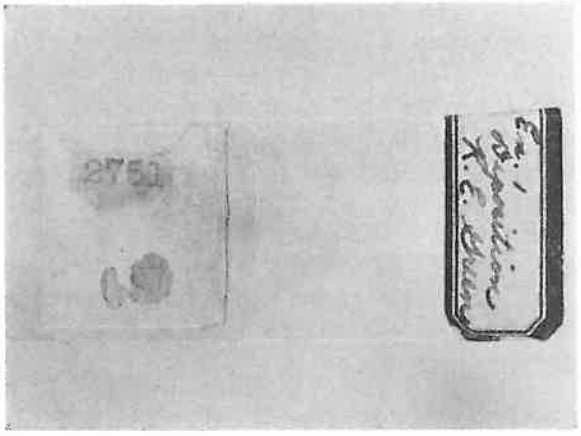
late summer of 1942, sometime before entering the military service, he noted that the appearance of the cervix by an examination with a speculum appeared normal. The mass in her abdomen had subsided to the extent that he could no longer palpate it. She had gained weight, gained in color and improved in her appearance. Though there was marked improvement, she was not classified as cured.

The confirmatory diagnosis made by Dr. Weller, Prof. of Pathology at the University of Michigan, was secured later to complete our records. He found the specimen well prepared and diagnosed it instantly.

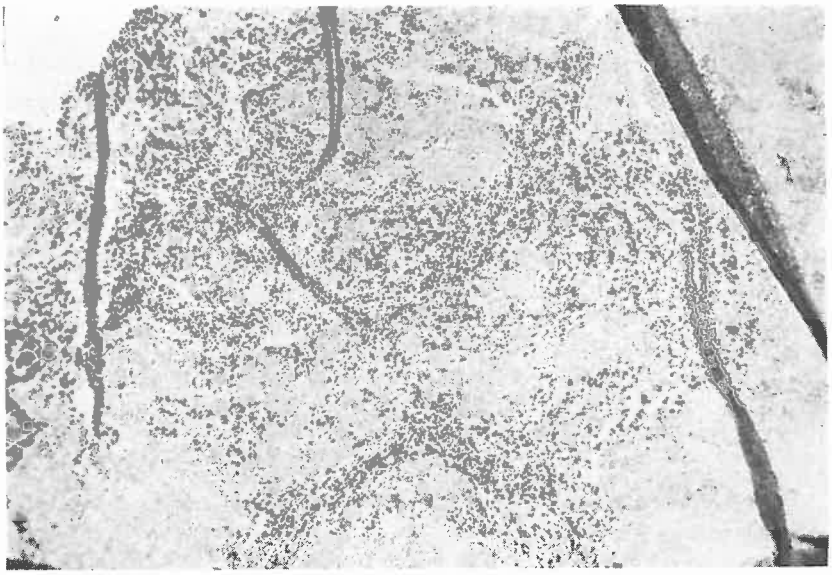
At the second Federal Court Trial in Detroit, Michigan, on May 29, 1946, Dr. Haines testified that he had seen Mrs. W. in June of 1944. That on physical examination he found only a fibroid tumor. He removed the tumor from the left side of the uterus and at the same time did a sub-total hysterectomy. The post operative diagnosis was a "simple fibroid." He stated: "I observed no malignancy, no—nothing except a fibroid tumor." The cervix was not removed, even though this had given rise to the malignant neoplasm. Hence we see that they left it in place in the body since it was normal. Dr. Inskeep, the pathologist, testified that he examined the tissue from this tumor and that his pathological finding was "Fibromata of the Uterus" and that this is a benign tumor.

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 health 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.

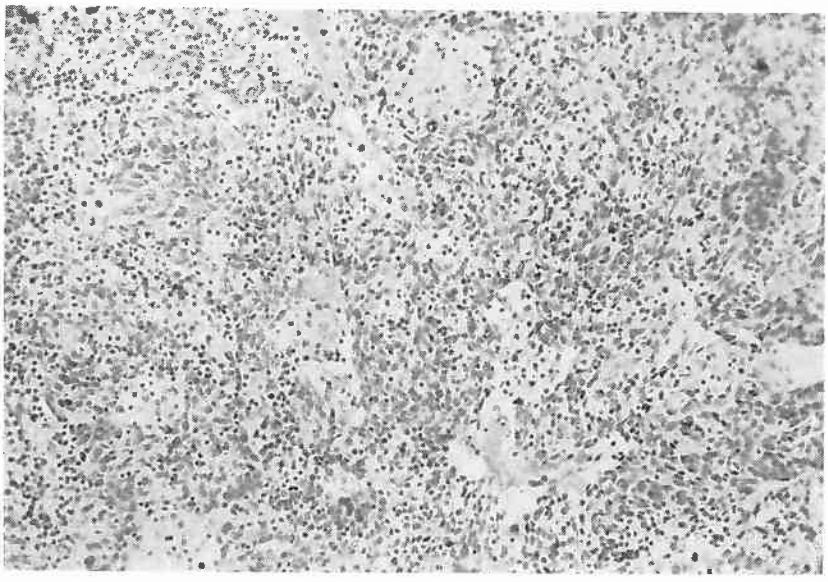
A photograph of the slide carrying the biopsy specimen well placed under the cover slip is given, and three microphotographs made by the Harper Hospital expert are also submitted for your study.



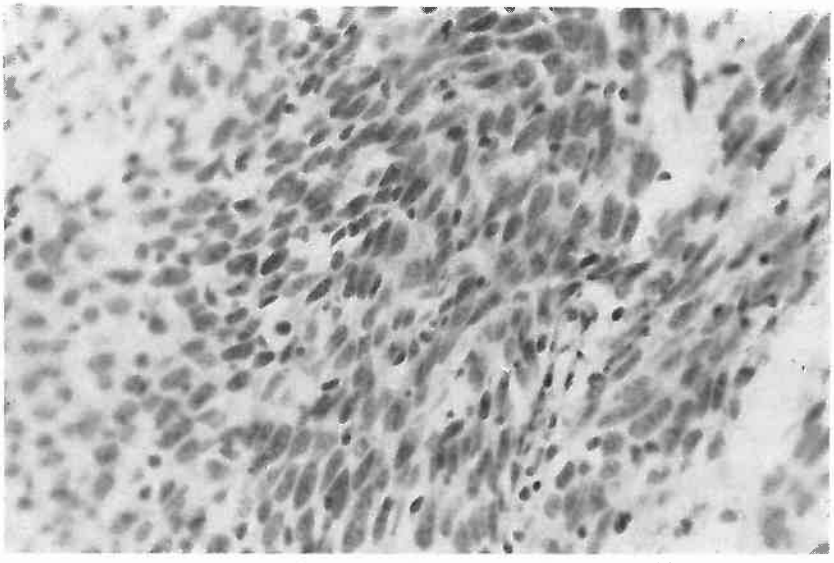
Photograph of the Biopsy slide showing its perfect condition.



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



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



High Power Microphotograph by Mr. Rinelander (675X)

Mrs. M. W. came under Dr. L....'s care 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 occurred within the year period, had there been a connection between this pathological condition and her death. It is very possible that the survival factor was destroyed sometime between June 1944 and her death in 1950 and that a subsequent malignancy developed in the lower end of the large bowel where Dr. L.... observed the foul-smelling mass during the autopsy.

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 therefore 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 occures.

LYMPHOCYTIC LYMPHOSARCOMA*

Treated in collaboration with
Dr. Frank Richards

Neoplasia As In Diagram IV

Mrs. S., 38 years of age, 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 helped. These appeared in April, 1943, and per-

DOWNTOWN CLINICAL LABORATORY
MICHIGAN STATE REGISTRATION NO 726
711 STROH BUILDING
DETROIT 26, MICH
Randolph 4552

CLARENCE I. OWEN M D DIRECTOR

NO. E-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

sisted 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. It did not respond to diligent treatment of any kind. Instead swelling and stiffness was noticed in the region, advancing deeply into the pharynx. Many doctors attempted to help in vain. 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 became evident that involved the neck structures of the right side. Biopsy done October 14, 1944, and examined by several good pathologists gave the diagnosis of *lymphosarcoma,* lymphocytic cell type, as reproduced below.

Two weeks later my examination revealed a marked cachexia, and the lymphatic glandular system quite widely involved with tumefactions. The gland in the neck that was biopsied was about five centimeters by seven in its diameters and involved all the structures of the region including the tonsil area.

The glands of the groin, and axillae were also involved and the largest of all was the size of a grapefruit encountered in the upper abdomen. It bulged, was hard and fixed. One injection was given, the serial system of carbonyl groups, at 11 o'clock at night. The next day at two in the afternoon, just fifteen hours later, a marked reaction developed, fever, chills, general achiness and a relaxation in the neck. Improvement in all of the neoplasms went hand in hand with these feverish chilly spells for about three weeks. This improvement was definite each day, and the stiffness and swallowing difficulty was gone by the end of the third week. In three months no tumors could be found. However, reactions came the twenty-fourth, thirty-sixth, seventy-second, eighty-fourth, and ninety-sixth weeks, and even later, one or more reactions were observed. But her health was better in some way after each, in spite of the fact that her health had already become much better than usual, or what was normal for her. This was noted in her energy, her ability to take care of a large house and of her family, and work in a clothing shop besides. Her prognosis at the time of treatment was probably two or three months to live. Following the absorption of the neoplasm there was no focal reaction in the foot

or the axilla where the infections were so persistent before the growth came. We expected some evidence of reaction as occurs in the tonsil area of the same side as the breast from which a malignant neoplasm had just been absorbed, at one of these points and watched for it, but she remained in good health without any such reaction for ten years, when in August 1955, a keloid condition developed in the scar of the biopsy incision.

On October 10, 1955, this patient received an injection of diphenoquinone. No significant reaction or improvement followed until the seventy-second week as happens in benign tumors. (See the case of Miss G.) The lymphosarcoma and the keloid had independent origins. A more detailed discussion is given in Chapter XVI. Following her seventy-second week reaction the keloid continues to undergo absorption and she feels very good.

CANCER OF THE COLON* **

Treated in collaboration with
Dr. F. L. Richards

Neoplasia As In Diagram IV

Mr. J. K. was 42 years, when X-ray studies and exploratory operation showed widely metastasized carcinoma of the splenic flexure of the colon, which caused complete obstruction. A colostomy was performed at the Henry Ford Hospital, Detroit, Michigan. The neoplasm perforated the abdominal wall in three different places, forming large cauliflower masses that discharged very foul necrotic and fecal material. As this condition developed he suffered a great loss of weight. His normal weight which was 180 had dropped to below 113 pounds. The biopsy report: Metastatic carcinoma of colon. The Henry Ford Hospital doctors had diagnosed this case as Fungating Carcinoma of Colon with a terminal prognosis.

The Surgical Pathological Memo from Ford Hospital is reproduced:

HENRY FORD HOSPITAL
Surgical Pathological Memo

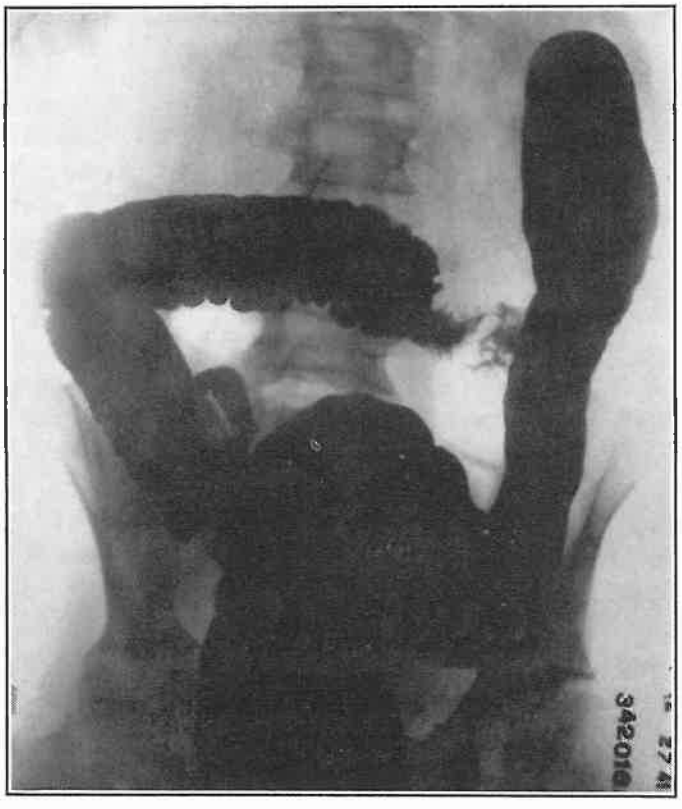
Photo:
Name: K. J.
Sex: Age: 42

Path. No. 66933
Case No. 342016
Date: 2/27/42

Pathological Diagnosis 101.62 CUTANEOUS SYSTEM: SKIN OF ABDOMINAL WALL: METASTAC 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 speciment are cauterized.



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

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/

Our examination revealed a very cachectic condition, and extreme emaciation. The abdomen was bulging from the tumor material within, and a colostomy opening on the right side was found which performed well. The rest of the abdomen presented large and small cauliflower growths ranging from 4 to 8 cm. in diameter, three in number. They were very necrotic in places and discharged fecal material. He was given one dose of two cc. of the serially arranged carbonyl groups on April 3, 1942. He improved some each day. On Sunday, during the third week, he hemorrhaged. This occured again on the following Saturday. He was given a second injection and some Calcarea for the bleeding. The hemorrhage stopped and he started to im-

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. Wallis

ANESTHESIA: Ether and Nitrous Oxide by Miss Bilyea

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. Copious dressings are applied and the patient is returned to the floor in good condition.

This is entirely a hopeless case

prove very rapidly. His weight became around 113 pounds by the end of June and by the time he returned to work the latter part of August it was over 160 pounds. After the second treatment the obstruction left and the bowels moved normally within nine weeks. In September, 1942, the fistulae had completely healed and his health had returned so we sent him back to the Henry Ford Hospital to have the colostomy closed. This was done and examination at that time could find no trace of the neoplasm. He made a splendid recovery.

The radiograph shows the obstructive neoplasm and its rupture through the bowel. The operative findings are decisive.

In a case of this type, Benzoquinone would not be sufficiently active as an initiator of oxidations to serve well. The serial systems of carbonyl groups is more efficient. Here we observe that the recovery rate is a function of the rapidity with which the disease develops. Also we see that normalization is possible when the basic oxidation defect is corrected.

LYMPHOSARCOMA*

Treated in collaboration with
Dr. Garret Warnishuis

Neoplasia As In Diagram IV

This is the case of Mrs. G. G., age 40, at the time of treatment with carbonyl catalyst on May 17, 1937. The disease first showed its malignant nature eight weeks previously when a small cervical lymph gland began to grow rapidly. A biopsy was made but at the same time several other glands enlarged in the posteriolateral cervical region. The biopsy was performed three weeks before her appearance at the Koch Clinic. In the meantime the remnants of the gland that was biopsied grew rapidly so that it became as large as the ball of a man's thumb in the intervening three weeks.

Besides this, several other glands began to show more rapid developments but were still small.

The biopsy report taken from the hospital record reads as follows:

c o p y

5M--5-37

DIAGNOSTIC LABORATORIES
MIAMI VALLEY HOSPITAL
DAYTON, OHIO

SURGICAL PATHOLOGY

Name C , George Mrs. (Last Name / First Name / Initial) Path. No. 95(-F

Station O.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 histological findings in the lymphnodes may or may not be associated with leukemia).

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

The carbonyl catalysts were given and a month later no trace of the growth could be found. It had been fully absorbed. The other glands returned to normal size. Her health remained good without recurrence of the disease. She met with an accident several years later that proved fatal. An autopsy was made which showed that no trace of malignancy could be found. Hence her recovery was complete. If this were not true there would have been extensive evidence of the recurrence of the disease and death long before the fatal accident and autopsy took place.

ENDOTHELIAL SARCOMA OF BONE*

Treated in collaboration with
Dr. Garrett Warnshuis

Neoplasia As In Diagram IV

A much slower growing but regularly fatal type of sarcoma is presented in the case of Harold B., age 41, when trouble de-

veloped in his right arm. Pain in the arm so that it could not be used and a swelling over the scapula brought him to the University of Michigan Hospital. X-ray films, blood chemistry exploratory operation and biopsies established the diagnosis as sarcoma of endothelial cell origin and involving the shaft and head of the humerus, the shoulder joint and scapula as well as the muscles posterior to it. A hopeless prognosis was given but he was offered an operation to remove the arm and shoulder girdle which he refused. He was then sent to me. The biopsy reads as follows:

Record R.

UNIVERSITY OF MICHIGAN
UNIVERSITY HOSPITAL
PATHOLOGICAL SPECIMEN

Name Harold B No. 554166 Date 9-1-34

Service Surg. SE Age Sex Pathological No. 1716-AM

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

XL

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

XL

UNIVERSITY OF MICHIGAN

UNIVERSITY HOSPITAL

SURGERY

HISTORY SHEET

Name B , Harold -2- Case No. 592162 Date 9-21-34

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.

AS

db Dr. Smith

Form M-J041305-J-34-10ZM-193361

46

My first examination was made September 17, 1934. The arm was found to be painful on motion. There was swelling of the shoulder and a tumor mass firmly fixed to the scapula as large as a man's fist laying over the spine of the scapula. Nearer the dorsal spine was a mass of similar nature about the size of an English walnut. There was some cachexia, but no abdominal tumor masses could be found. The treatment of 2 cc. of the 12X dilution of the serial system of carbonyl groups was given. Recovery was steady and slow. In a few months he could use his arm without trouble. In a year the tumor masses were entirely absorbed, and X-rays showed a nearly complete recovery in the bone.

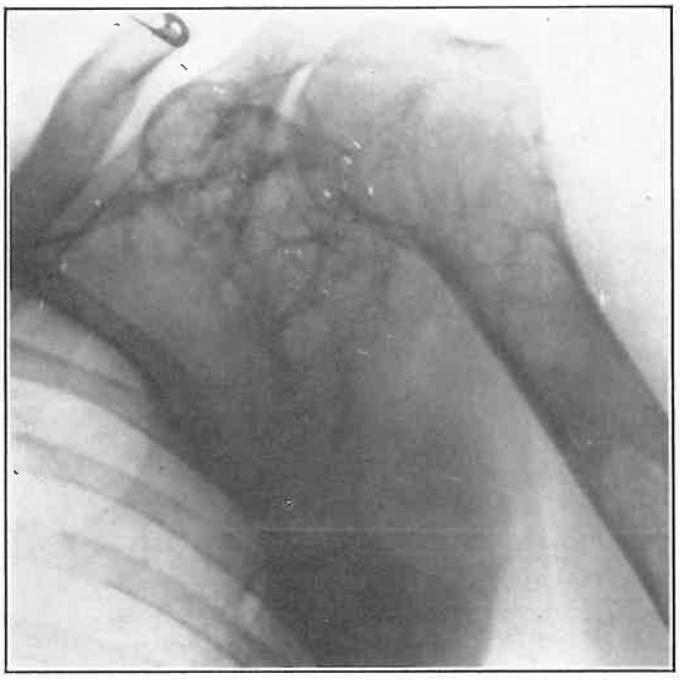
His major reactions were at the twenty-fourth and thirty-sixth weeks. He continued to work and made a full recovery. We did not have opportunity to make another radiograph before 1942. This showed complete recovery. Comparison of the pre-treatment and post-treatment radiographs show a considerable thickening and strengthening of the bone where the disease tissue had to be absorbed. This is seen in other cases of bone destruction by sarcomas or carcinomas, and appears to be a general compensatory repair reaction, and not dependent upon the type of disease that had to be removed. His health remains good and there has been no further trouble of this kind. Radiograph II shows the condition after full recovery.

In this case we observe a slower development in the neoplasm, and no metastases to the vital organs in the year it had been progressing and producing symptoms. The nature of this disease as described by Ewing, our leading authority, p. 361, Text Book on Cancer, 1942, is:

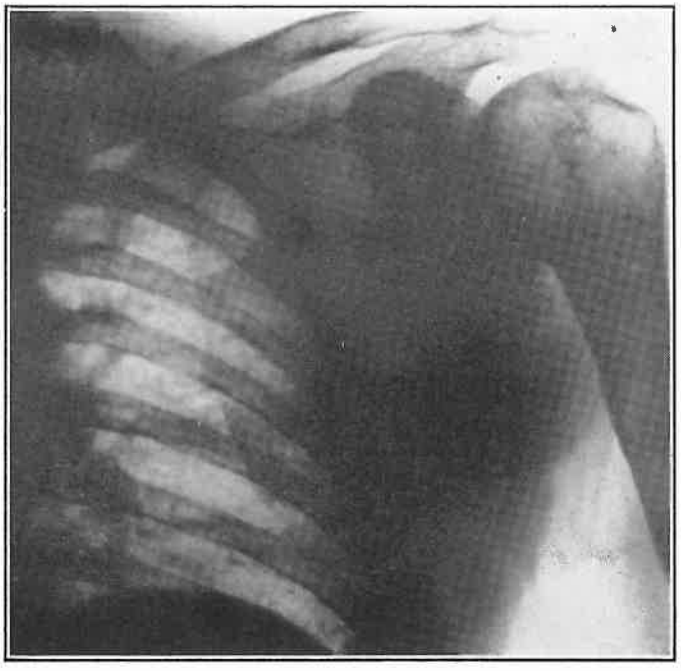
"Angio endothelioma, multiple endothelioma, diffuse endothelioma, or endothelial myeloma, the entire group, is characterized by predilection for the bone shaft, a tendency to multiplicity, a cellular and vascular structure, marked osteolytic properties, failure to produce tumor bone, and a relatively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

The recovery was complete and perfect health was reported in 1950 when he was last heard from.

Thus a slow growing malignant neoplasm, makes a slow recovery. This is our consistent experience.



Radiograph I, showing condition before treatment.



Radiograph II, showing condition after full recovery.

The recovery process must therefore not be forced beyond its natural course by unnecessary repetition of the dose.

CANCER OF THE LIVER

All primary tumors of the liver are malignant whether or not they are associated with cirrhosis. Biopsy in such tumors is not practiced and indeed is not done except as an experimental or academic procedure. The autopsy material and biopsy material together with the clinical history have established this fact. Ewing may be consulted in support of this statement.

Cancer of the liver is interesting to us however much like the anaplasias or atrophies of nerve cells consequent to integration with a toxin or virus, where function is abolished, and yet the cell may not be dead as yet though affected for many months or years, and gradually passing on the way to death. Such cells we show are able to reconstruct themselves and function again after the toxin or virus has been separated away in the correct manner. Live cells, parenchyma or duct cells, may go malignant and completely anaplastic so as to never function again and with the recovery process are digested away as so much debris, but the deficiency left in the organ by such anaplastic loss of the neoplasia can be compensated for by reproduction of the Grade A cells that still remain. A primary tumor of the liver will therefore be able to be absorbed and leave a large defect in functioning tissue. This can be repaired with good return of function of the organ, in time to meet the requirements for function. This is not the case with secondary cancer of the liver always. If a large amount of liver substance has been destroyed by secondary invasion, there may not be enough reconstruction of liver tissue with the absorption of the tumors to support the liver function required by the body in general and to take care of the metabolism of the tumor material absorbed, and death may follow because of hepatic insufficiency. The far advanced cancers of the liver of primary origin are therefore more favorable to recovery than the secondary neoplasms that invade the organ by infiltration and metastases because of the presence of a certain amount of restorable anaplastic cell that we previously described as Grade A.

CANCER OF THE LIVER*

Treated in collaboration with
Dr. David Arnott

As Diagramed in IV

Dr. Arnott was called in to treat Mrs. M. G. by the patient's husband. Earlier, Dr. Arnott had been consulted by Dr. E. W., the surgeon.

From the hospital records and speaking with the surgeon, it was evident that an operation was performed upon Mrs. G. on June 29, 1931, in which the surgeon opened the abdominal wall over the gall bladder area. The gall bladder was brought up to the surface of the incision by forceps and held there while a tube was inserted to drain off the blood and then the gall stones were found and removed. Then a permanent drainage for treatment was left there after the operation and it was still in position when Dr. Arnott saw her over three weeks later.

At the operation an examination was made and no gall stones found to obstruct the flow of bile into the bowel. It was found at this time that there were tumors on the liver. An obstruction of the gall bladder was thought responsible for Mrs. G.'s yellowish looking skin. If nothing else happened, her skin would become normal as the condition was corrected. However, Mrs. G.'s liver had numerous tiny firm nodules which would obstruct the free normal functioning of the liver so that the bile would be absorbed into the blood and would also result in the yellowing of the skin. It appears from the hospital record of July 7th that the patient is intensely jaundiced and is feeling very miserable.

This would suggest that the liver condition was responsible for her failure to recover after the gall stones were removed. Upon this same day the hospital record says that her condition is poor and the prognosis is poor; that is, hope for her recovery is poor. Reports on July 13th and July 22nd, 1931, of the hospital record, indicate that the patient still feels very sick, nauseated, and that the intense jaundice persists with prognosis still poor.

On July 24th, the hospital record indicated that the patient

is still very jaundiced, has no relief from gastric pain and nausea, and that prognosis is bad; in other words, no hope of recovery. It was about that time that Dr. Arnott first visited the patient. Dr. Arnott stated:

"I gave Mrs. G. the treatment in the morning as I remember, and when I saw her in the afternoon her jaundice seemed distinctly relieved to me and by the next morning anybody could see it. From July 26th until August 10, I was out of the country. When I returned to Canada I went to see her at Victoria Hospital. Her condition was much improved. She had lots of stomach distress, but the jaundice was much relieved and her vitality was distinctly better. I saw her at the hospital and two or three visits afterwards. She was making distinct improvement. It is my opinion that Mrs. G. would have died within a few weeks if she had not had the benefit of this treatment." . . . no x-ray treatment, or operation could have cured the patient. The opinion of the hospital itself stands on the record as no hope for recovery. She was comatose; the gravity of this serious state was increasing."

Mrs. M. G. made a complete recovery within six months after receiving one injection of the carbonyl catalyst in July 1931. She has remained in perfect health to date (1943).

SARCOMA OF THE SPLEEN*

Treated in collaboration with
J. W. Kannel, M.D.

As Diagramed in IV

B. G., a young girl, age 6. She had pain in the stomach and chest with some fever. There was an enlargement of the spleen and the axillary and inguinal lymphglands. She was taken to the hospital. Her blood count the first day was 7,200 white cells. On June 23, 1943, it was 16,700 and on the twenty-fourth just before the operation it went up to 22,400. Dr. Kannel knew at the time she had a very virulent disease and he thought it was an abscess.

On June 24, 1943 he did an exploratory laparotomy and

closed her up. Nothing was removed nor did he change the condition of any of the organs. No abscess was found. He did find an enlarged spleen extending 2 inches below the ribs, and compressing about two-thirds of the lower part of the left lung. The upper third was patent. The ribs caused pressing or indentations in the spleen. There was a nodular condition of the spleen. It was irregular and very hard. A normal spleen would have been more soft. It would be so small that it would not have projected down below the ribs nor would it have compressed the lung.

She was sick only five days before Dr. Kannel put her in the hospital. He operated on her because the progress of this case was so rapid. She was sent home on the seventh or eighth day after the operation.

Dr. Kannel's first diagnosis, before the operation, was an abscess under the ribs in the left hypochondriac region. His post operative diagnosis was enlarged spleen, possibly malignant. No biopsy was taken because he felt that the removal of tissue would result in her death. His final diagnosis was sarcoma of the spleen. This diagnosis was made June 24, 1943, and was based upon the clinical findings and the child's case history.

On July 2, 1943, she received an injection of the serially arranged carbonyl groups. Her improvement was gradual and she made a complete recovery. Dr. Kannel ascribed her recovery to this treatment.

Recent reports confirm the good health of this patient.

The hard nodular spleen of rapid growth and great increase in size, showing the elevations where it could grow more rapidly forward between the ribs, is pathognomonic of sarcoma of the spleen.

SARCOMA OF THE UTERUS

Neoplasia As In Diagram IV

Mrs. Mc A., age 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

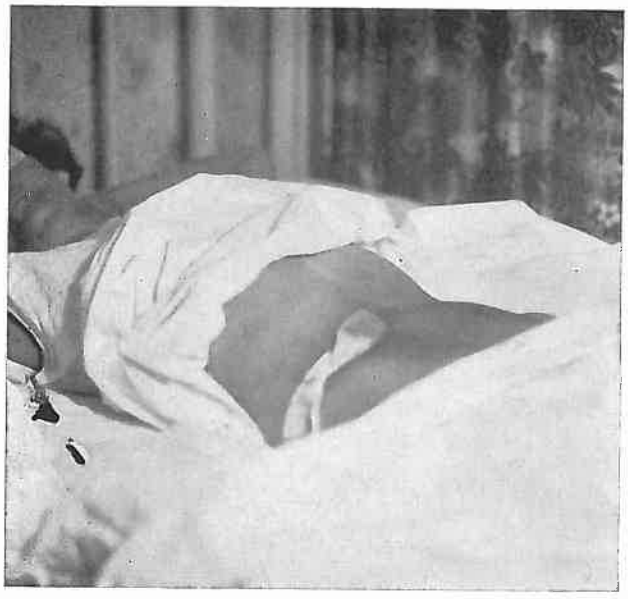
crises of vomiting and pain in the gall bladder region. The abdomen was found widely involved with neoplastic development from deep in the pelvis to the diaphragm, with the stomach and liver and intervening structures heavily invaded. This was identified as the cause of the pain. A biopsy specimen was 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 the treatment of serially arranged carbonyl groups given.

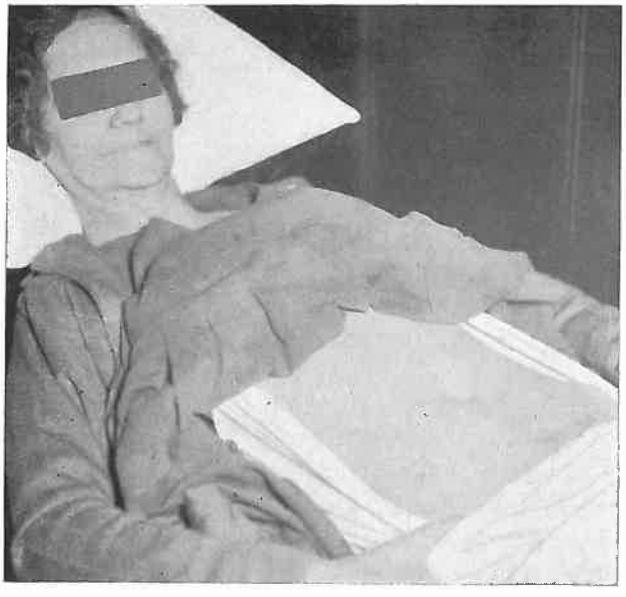
She responded well up to the sixth week, gaining in strength and becoming rapidly free of the pain and vomiting attacks. However, at the beginning of the sixth week she started a reaction that continued to the middle of the ninth week.

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, 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.



Photograph I, taken at time of treatment.



Photograph II, taken after neoplasms were absorbed

RECURRENT METASTASIZED CANCER OF THE PALATE FOLLOWING SURGERY* **

Neoplasia As In Diagram IV

It is well established that unless cancer is fully and completely removed by surgical operation, so that not even one trace of it remains, it will usually come back with much increased malignancy and destructiveness and a more rapid tendency to spread. The case of A. J., here given, shows that even in a simple, readily accessible case of low grade malignancy, surgery can fail and be followed by a rapid recurrence as a wide-spread dissemination of the disease. When the disease is made inoperable, the restoration of the natural immunity can prove curative. This is demonstrated in this case.

Mr. J., age 60, was first seen by Dr. Koch on December 1, 1932. His condition was cancer of the palate and of the glands in the neck. Examination showed the palate, hard and soft, to be covered with a large growth and about a dozen smaller ones. Some of the glands under the jaw and in the neck close by were enlarged and hard, showing fixation.

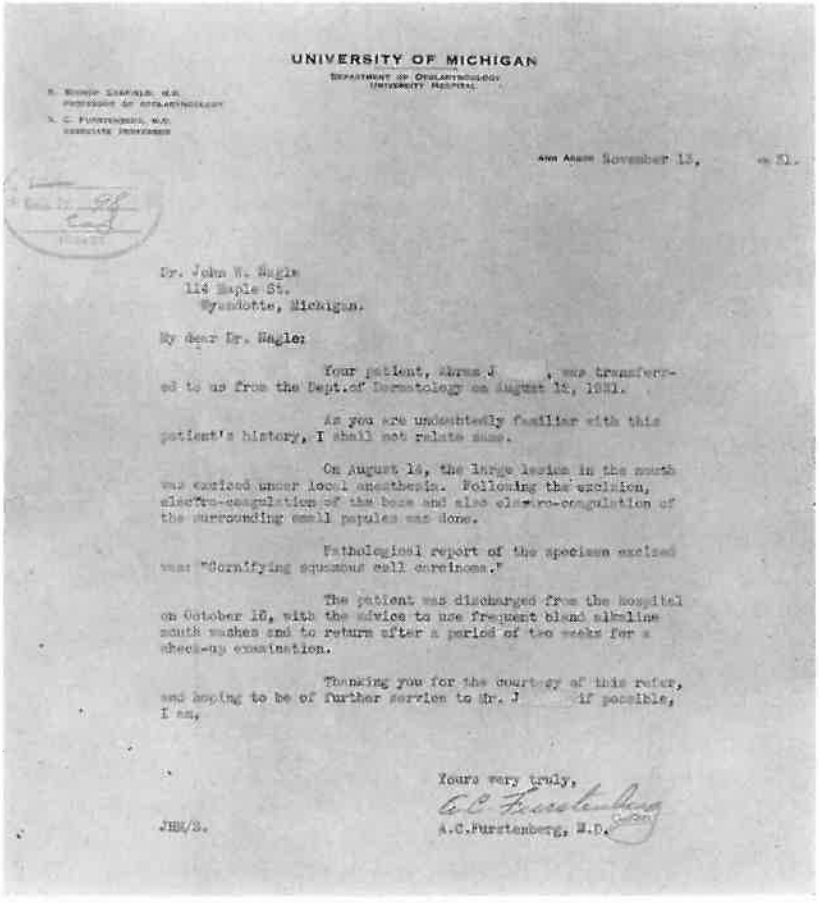
He gave a history of having been at the Hospital in Ann Arbor, Michigan, October 15, 1931, where an operation and touch up with electric cautery was done. The areas healed and all was well for a number of months. Then the same type of growths returned, but they were more widespread and in larger number locally, with extension to the chin and neck glands.

The report of the University Hospital on the biopsy is given as, "cornifying squamous cell carcinoma."

After the carbonyl catalysts were administered in December, 1932, he had a series of reactions with grippiness, chills, and fever at intervals and there was appreciable improvement even in a week. The recovery progressed steadily so that in less than a year it was complete, all tumors had been absorbed and healing was perfect. He also gained good body weight, became much stronger and enjoyed better health than he had experienced for many years and still remains well without recurrence of the trouble. The recovery reactions showed very little focal disturbance, whereas the constitutional symptoms were severe, thus

indicating some distant unidentified focus of infection as the source of the carcinogen. To illustrate this situation, we give the patient's own description of this first recovery reaction following the treatment.

"About the third day I felt pretty badly. I became cold. I thought I was going to freeze. The 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, for, I would say, six months, I believe, every three weeks, but they kept getting lighter."



UNIVERSITY OF MICHIGAN

DEPARTMENT OF OPHTHALMOLOGY
UNIVERSITY HOSPITAL

Ann Arbor November 13, 1931.

Dr. John W. Eagle
114 Eagle St.
Wyandotte, Michigan.

My dear Dr. Eagle:

Your patient, Abram J , was transferred to us from the Dept. of Dermatology on August 14, 1931.

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 12, 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

JHH/B. A.C.Furstenberg, M.D.

In this case there appears to have been a systemic intoxication that was supporting the malignant change in the epithelial cells. The constitutional symptoms of reaction were so severe that they indicate this to be the fact, and the rapid recurrence after the surgery indicates that the toxic stimulus was abundant as well.

CANCER OF THE STOMACH*

Treated in collaboration with
Dr. Harrison

Neoplasia As In Diagram IV

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.

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

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. _____

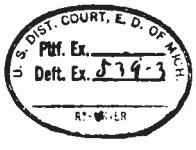
GROSS EXAMINATION

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 bw 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

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 and he was in good health, twenty three years after treatment.

CANCER OF THE STOMACH* **

Treated in collaboration with
Dr. H. E. Mantor

Neoplasia Diagramed in IV

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 supra-clavicular 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

Register B
-13/41 V2

SURGICAL CARD
MAYO CLINIC — ROCHESTER, MINNESOTA

No. 1-15-560 Age 58 Sex M Sec. LOGAN Date of Ex. 5-24-41

Name Mr. J. J. Address HANOVER, KANSAS

Name of Dr. ..... Dr's. Address .....
Not Returned 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 C. W. BROWN Surgeon WALTERS

Preoperative Diagnosis ULCER. CARCINOMA STOMACH.

Operation Indicated EXPLORE.

Considerations affecting risk RISK 114 LEFT RECTUS INCISION.

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

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

Date oprs 5-26-41 Opr. Room 14 Surg Walters 1st Giffin 2nd Strom Recorder MCM

Antist J.M.C. Donald Antic. $C_2H_4 + O_2 + C_6 + T$. Time of Ancs. 1:10 – 1:55 Op. 1:20 2:00

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

Oper.: Abdominal exploration.

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).

J-92-b—Revised 2-10-41.

red blood cells in a one per cent NaCl 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.

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—cm

Philip W Brown

Philip W. Brown, M.D.

Radiograph I, taken at the Tyler Clinic before treatment.

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

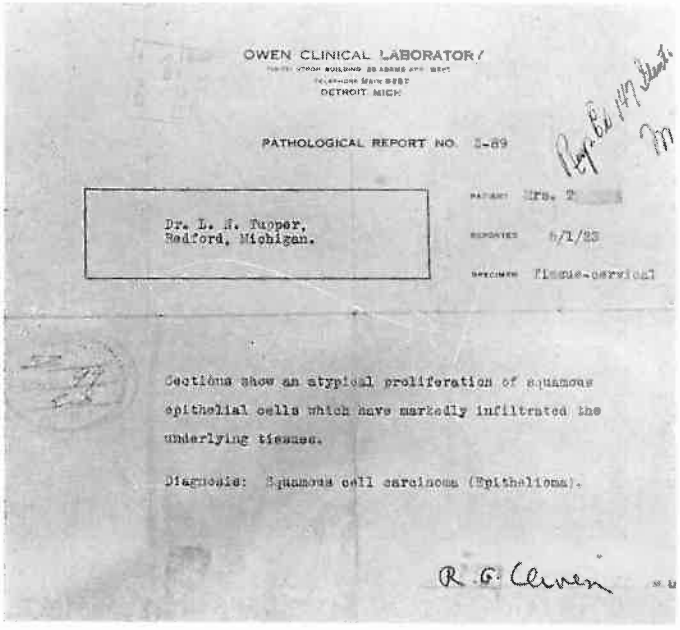
Radiograph III, taken June 15, 1944, after recovery.
These radiographs are court exhibits

CARCINOMA OF CERVIX UTERI* **

Treated in collaboration with
Dr. McCosh

Neoplasia As In Diagram IV

In August 1923, Mrs. T. was 31 years old. For over a year previously she had profuse irregular bleeding, muco-purulent discharge, and increasing pain with progressive reduction in the capacity of the urinary bladder. She consulted a surgeon who made a biopsy. A very responsible laboratory in Detroit made the diagnosis of squamous cell carcinoma of the cervix as follows:



OWEN CLINICAL LABORATORY
DETROIT, MICH.

PATHOLOGICAL REPORT NO. 2-89

Dr. L. J. Hooper,
Redford, Michigan.

Mrs. T.

6/1/23

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

My examination was made a few weeks later and the findings were a typical cancer of the cervix that had spread to involve the corpus uteri and adnexia but mostly on the right side. The mass was fixed and involved the other structures so as to obliterate all normal contours. The bladder wall was also affect-

ed. This was a far advanced hopeless case from the standpoint of the surgeon or the radiologist. The bleeding was profuse and the condition advanced following the biopsy.

We gave her two injections of the oxidation catalyst, 12X dilution, one on August 7, and the other on August 21, 1923. The bleeding and discharge soon improved, and her yellowish color cleared with the return of blood to the circulation. Pain disappeared and the growth was found to soften and recede more every few weeks. At the thirty-sixth week no more evidence of the disease was to be found. The cervix was somewhat deficient on the right side. This evened out several months lat-

er. Her reactions were most marked during the twelfth and twenty-fourth weeks. She later became pregnant and gave birth normally to a normal boy after a normal gestation period. Each two years thereafter another normal child was born after normal gestations and with normal deliveries, four children in all. There has been no return of the disease, but instead extra good health has been her lot ever since. She still remains well more than thirty years after treatment.

The photograph shows Mrs. T. with the first three children born after her recovery.

This case illustrates the destruction of the toxic cause, return of function, and tissue restoration following the administration of a single agent, the oxidation catalyst in high dilution. We have many such cases in our records. Full restoration of function was part of the cure. There have been no miscarriages since treatment as had happened in February 1922.

The uterus is perfectly normal with full reconstruction and the trauma of four parturitions did not cause a return of the disease. We see here that more than injury is required to cause cancer, and that factor no longer existed in this patient following the treatment.

CANCER OF THE LIVER

Treated in collaboration with
Prof. Dr. R. S. Lopes

Neoplasia As In Diagram IV

Mr. G. A., Brazilian, early in March, 1941, in Barbacena, State of Minas Gerais, where he lived, suddenly showed inappetency (lack of desire for food—no appetite), progressive decrease of weight, and general weakness. Dr. Omar Araujo Lima, of that place, upon examination of the patient, noticed a large abdominal tumor. The patient was conveyed to Rio de Janeiro, was interned in the Hospital of the Beneficencia Portuguese, where he was operated by Professors Doctors Thomas Rocha Lagoa and Jorge Morais Grey, helped by the assistant physician. Laparotomy disclosed an extensive neoplasm of the liver and large infiltration of the colon. The tumor had so largely spread about and so extensive were the infiltrations that the operators

decided not to go on with the surgical intervention, due to the danger which the patient would be exposed to and the uncertainty of favorable results. By decision of physicians an operators biopsy was not made, it being supposed unnecessary, so evident was the case. In the opinion of operators such studies were superfluous. He examined the patient about seven days after the surgical intervention and thus noticed the extension of the tumor under the abdominal wall and the extreme weakness of the patient. On the 15th of July of the same year an injection of 6X dilution Benzoquinone was administered. The patient, who, up to that moment showed only light pyrexia, shivered intensely in the afternoon of the 18th, and his temperature raised up to 39 degrees. On the 25th he belched out and had intensive gastric pain. Both symptoms disappeared after a short time.

Proportionally to the gradual decrease of the tumoral volume, the recovery progressed, he regained color, and his body weight increased 10 kilos; he went back to Barbacena where his health gradually returned. On the 3rd of October another chill took him and his temperature was 39 degrees; also a general oedema was shown; everything yielded spontaneously and the patient, gaining then over 20 kilos, was completely recovered and devoted to his daily work, having not been submitted to any other treatment besides that of Dr. Koch. Report in April 1953 shows this patient to be in perfect health still (12 years).

SCIRRHUS CARCINOMA OF BREAST

Treated in collaboration with
Prof. Dr. R. S. Lopes.

Neoplasia As In Diagram IV

Miss C. F., 50 years, Brazilian, was brought to Dr. Lopes by Professor Artidonio Pamplona, of the Escola Fluminense de Medicina, a well known clinical physician. The patient was suffering, for about six months, from a large tumor in her breast; the nipple was completely retracted, as it generally happens in such cases. For instance the patient felt local pain; she was frightened by several surgeons, upon consultation. No improvement was shown after her submitting to 12 applications of deep radiotherapy.

On November 17, 1941, one injection of 2 cc. 6X dilution of Benzoquinone was administered. A second injection was given six months later. She began to recover generally, the tumor was disappearing and the nipple resumed the normal aspect. In 1942, one year after the first injection, no tumor was noticed and the patient was considered clinically cured.

CANCER OF THE BREAST

Treated in collaboration with
Prof. Dr. R. S. Lopes.

Neoplasia As In Diagram IV

Mrs. M. S., age 42, Portuguese, married, late in 1938, in Lisbon, Portugal, where she lived, complained of a ganglion in her breast, which was then operated.

The anatomo-pathological examination revealed a cancer. Sometime in April, 1941, a large tumor appeared in the very same place of the operation and the patient refused the extirpation of the breast.

On October 14th, 1941, we administered to her an injection 2 cc. of Benzoquinone 6X. We examined the patient seven months later and found, in that place, a small isolated scar not larger than a grape. Her general condition was excellent.

DUODENAL ULCER

Treated in collaboration with
Prof. Dr. R. S. Lopes.

Mr. F. G., age 38, Brazilian, chemist. Some 5 years ago he began feeling disturbance and "seasickness"; later on he felt hunger pain, which ceased upon eating. Frequent heartburn.

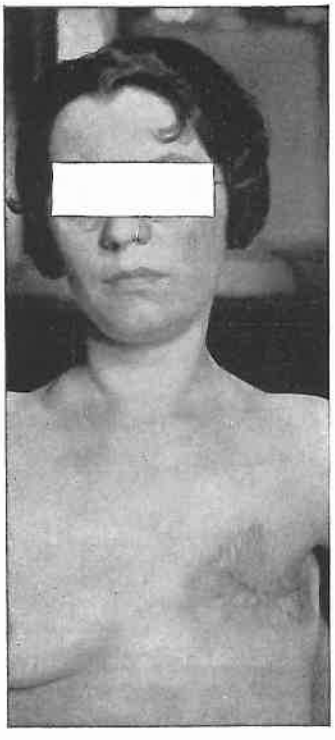
Good appetite. The liver had grown beyond the costal border more than 3 fingers transversely. The radiographic examination, made in November 11th, 1941, confirmed the clinical diagnosis—"duodenal ulcer"—and disclosed deformation of the bulb, with the aspect of a chronic ulcer where one could see the central niche. One injection of 2 cc Benzoquinone 6X was administered on November 9, 1941, and the patient was submitted to the diet

recommended by Dr. Koch, that is to say, deprived of any albumin of animal origin.

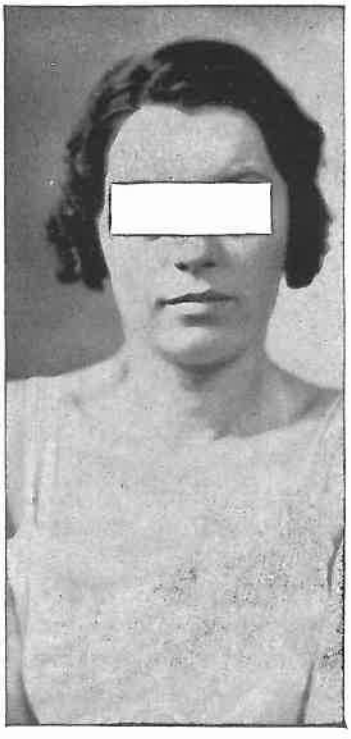
Considerable improvement was shown and he is cured at present. The last radiographic examination, made on September 29, 1942, shows a normal anatomic aspect of the duodenal bulb.

METASTATIC CANCER OF THE BREAST

This case is given in brief by photograph. It shows cancer of the breast removed radically gave rise to deep metastases within the mediastinum which spread up into the right supraclavicular space as a hard fixed metastasis. At the time of treatment the lung envolvement was evident in her dyspnoea,



Miss H. P.
Before Treatment.

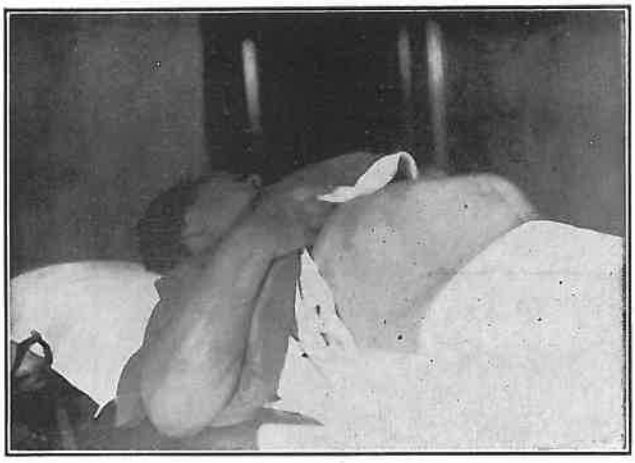


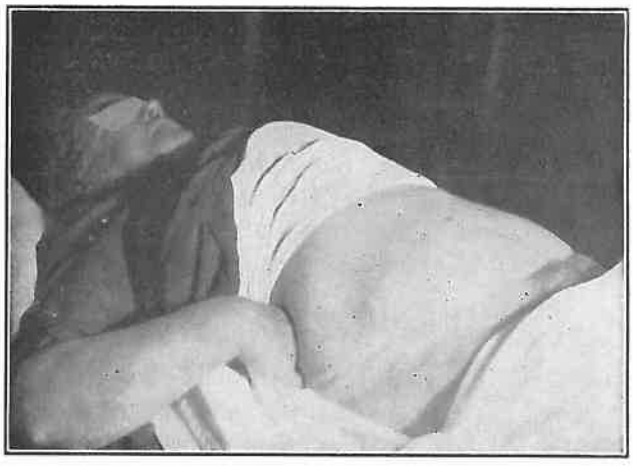
Miss H. P.
After Treatment

and was observed by the breath sounds and by percussion. Miss P. was treated in 1927 with carbonyl catalyst and in six months was free of visible or palpable evidence of cancer and has so remained up to the present time. We take pleasure in presenting cases that remain cured for long periods demonstrating the permanency of the survival factor instituted by this treatment, which did not exist when the disease was spreading at the time the treatment was given. She was reported to be in very good health in March 1955 when we last heard about her case.

RECURRENT CANCER OF THE BOWEL

Mrs. S. before treatment. The photograph shows the exploratory incision, with the cancer mass grown high above it. Operative attempts were not successful.

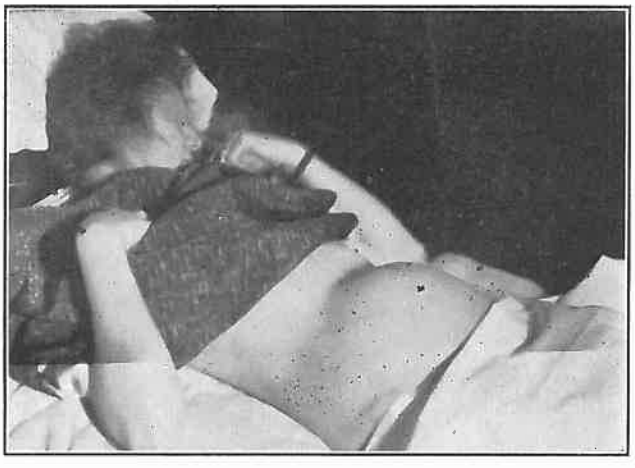




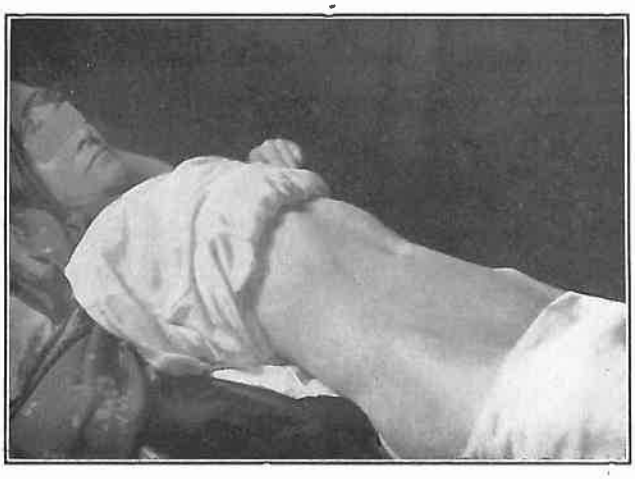
Mrs. S., age 60, was treated May 2, 1932, with 2 cc. of the carbonyl catalyst. This photograph shows the disappearance of the neoplasm. The line of incision is still visible. Palpation revealed no trace of the neoplasm.

FIBROMA OF UTERUS

Miss G., age 45. Two photographs are shown of this patient, one taken before treatment and the second taken after



Photograph I, before treatment.



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

treatment for an enormous fibroid that possibly underwent malignant change. She received three injections of serially arranged carbonyl groups: one on December 2, 1930, May 31, 1931 and May 9, 1932. Her recovery was complete before the end of 1932.

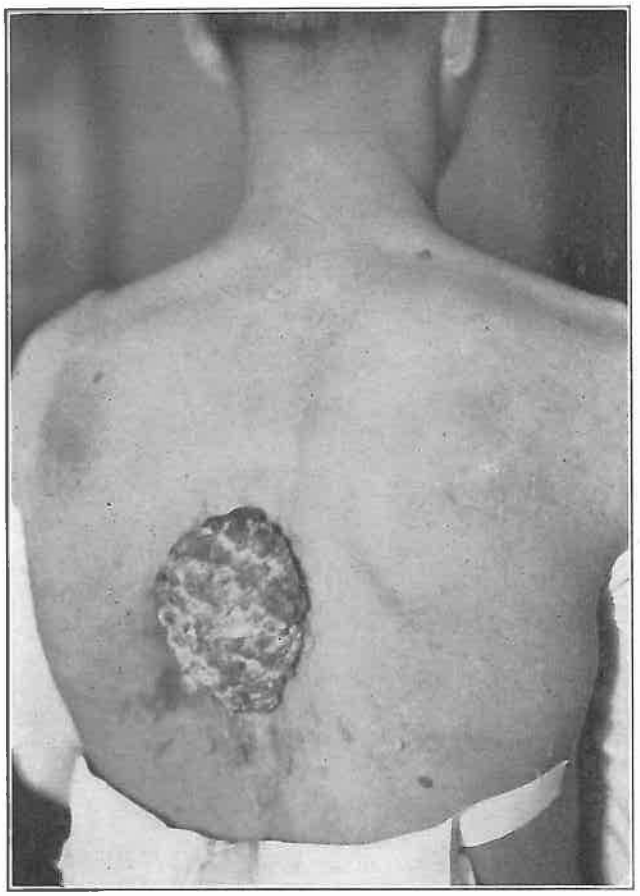
TERMINATION OF MALIGNANT STATE

Demonstrating the Survival Factor

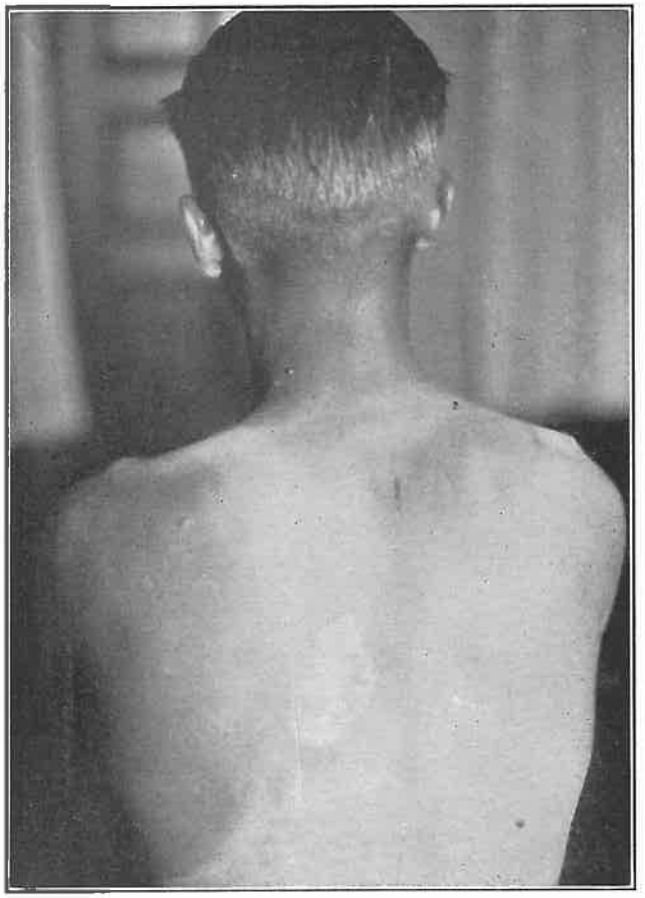
The time of termination of the uncontrolled cell division, is readily demonstrated when the tumor as a whole swells to undergo digestion and then rapidly disappears by absorption. This can be observed en mass. Microscopically it is seen when the tumor cells undergo a coagulation change with calcification, as the microphotographs show. However no one can be sure from such observation that each and every cancer cell has taken part in the destructive change. One must apply a more certain test. It is true that the cancer cells become foreign material or tissue debris requiring digestion and removal as soon as they can no longer use energy for function or reproduction and hence must die. This is early after the treatment is given when the whole trend is set in reverse. The better test is to wait till the metastases and infiltrations have been absorbed and then to re-

move the rest of the growth that is being absorbed. Non-recurrence is final proof.

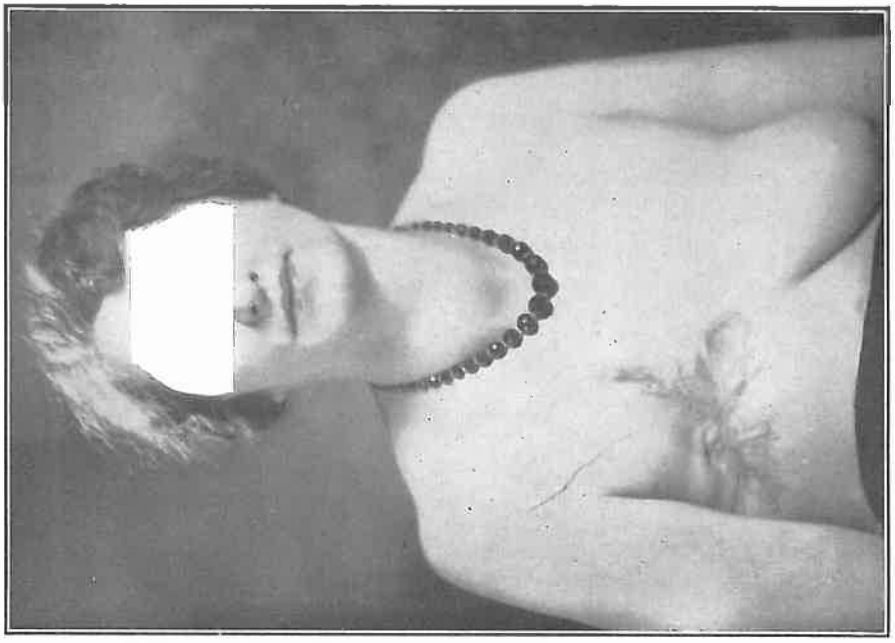
If this portion were taken away without treatment, the disease would be stimulated, recurrence would soon occur, and death would come quicker. But after the carbonyl catalyst treatment, the absorption of the last extensions shows that the causative factor is removed and an involution of the progression is now taking place. Then with the cause removed surgical stimulation by pinching and cutting has no factor at hand to give rise to a spread of the disease, or to hinder its complete absorption and healing, provided no chemical stimulus is used of a carcinogenic nature. In the following cases where it was easy to see when the metastases and infiltrations were well absorbed, and the growth proper was undergoing digestion and was virtually dead, we did crude removals, and watched the much quicker return to health than if the patient had to absorb all the toxic debris present in the infected necrotic mass. The pictures show the state before removal after the absorption of the extensions, and after the removals after healing was completed. Both patients were permanently cured. The breast case came to testify about ten years later at one of the Federal Court trials. But valuable as the case was we could not use her in view of the large number of cured patients who were waiting to testify. Both cases were found malignant at biopsy. The breast case was an adenocarcinoma of duct origin, the skin case was a squamous cell carcinoma with melanotic changes. The photographs show the conditions before and after receiving this treatment followed by the surgical tests. It is to be noted in the Mr. L. case that the areas of malignant pigmented infiltration were not touched surgically but cleared up entirely because of the treatment thus showing that the carbonyl catalyst served as the survival factor. The two non-malignant pigmented moles, however, remained unchanged.



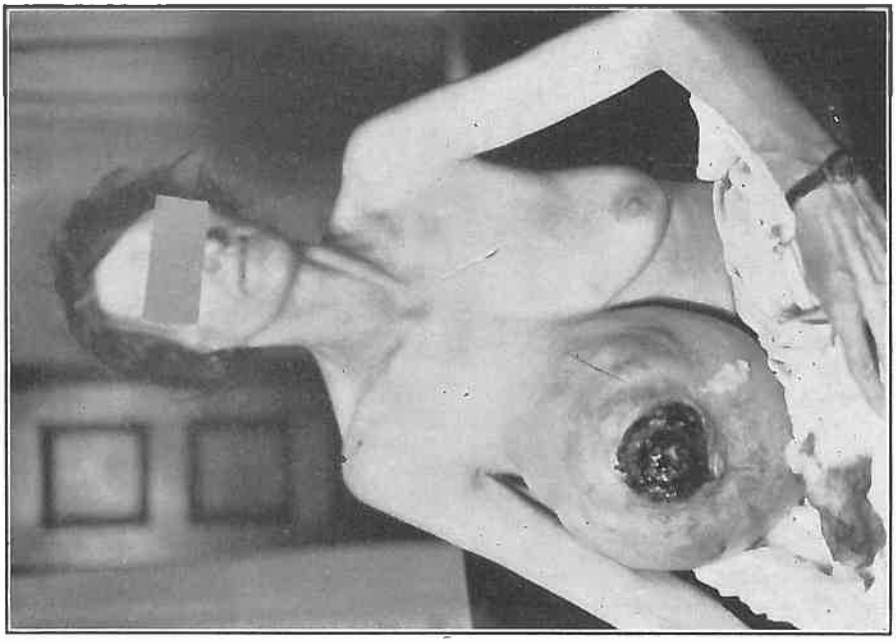
Mr. L. after treatment when the growths were undergoing digestion just before crude removal.



Mr. L. after complete recovery



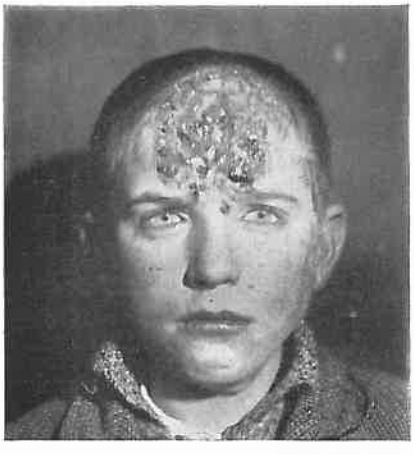
Miss N. after complete recovery.



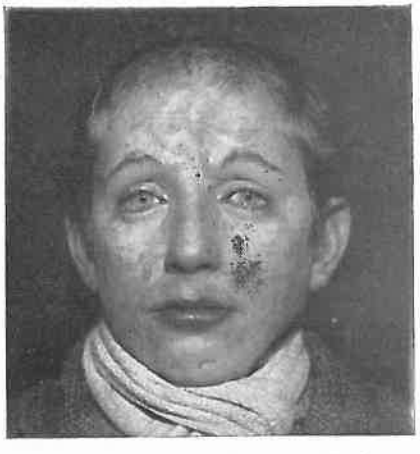
Miss N. after the growth started undergoing digestion after the carbonyl catalysts were given, and before crude removal was done.

SYPHILIS

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. 1. 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.

BLOOD RECONSTRUCTION IN CASES OF HEMATOPOIETIC EXHAUSTION

These cases are cited to show the similarity in the destructive action of irradiation and of a biological pathogen on the bone marrow, as well as their correction. The cases were diagnosed as lymphatic leukemia by the use of sternal marrow biopsies at the institutions where the patients had gone. In each case, blood transfusions failed to stop the hematopoietic failure which steadily progressed in a malignant fashion. In the first case, the leukopenia was of the terminal type. In the second case, it was produced by irradiation. The third case was an acute case of leukemia. In all three cases there was correction in both red and white blood cell production, thus showing the physiological position of the reagent used.

LYMPHATIC LEUKEMIA
With Terminal Hematopoietic Exhaustion In A Boy

Treated in collaboration with
Dr. Baldor.

Neoplasia as In Diagram IV

Teddy S., 14 years, diagnosed as lymphatic leukemia six months earlier, was extremely depressed, unable to walk, suffered with pain and fright, had offensive odor from the mouth, profuse gingival hemorrhages, and evening fever of 102°. The onset was acute with fever, chills, pains in legs, and finally severe anemia with lymphocytosis. He had 57 blood transfusions in three months without help. Blood count when admitted, February 25, showed red cells 2,150,000, hemoglobin 40%, leucocytes 15,000. He showed enlarged tender spleen and liver and enlarged cervical and groin glands and bloody patches over the entire body. Increased tubular respirations in the right pulmonary base and moist rales over the whole lung field. Two cc. of carbonyl catalysts were given after two days of fasting and colonic lavage. In one week he was sent home with a normal temperature, the spleen was much reduced in size and the bloody patches had absorbed very substantially in this time turning from their dark blue to light green, to yellow and then finally disappeared. Nine weeks later he returned to Tampa for a

check-up. He was then able to walk, had gained 12 pounds in weight, and the blood picture was as follows: red cells 3,350,000, hemoglobin 52%, leucocytes 8,000. He gave a twelfth week reaction with slight pains in the extremities and a little epistaxis. The red cells numbered 4,000,000, hemoglobin 72%, leucocytes 6,500. He had gained 25 pounds weight and felt perfectly normal. It is to be noted, Dr. Baldor points out, that without even one blood transfusion after the catalysts were given, the blood no longer underwent destruction but began to pick up without any aid but a vegetarian diet. Thus the infection was eliminated very early after the catalysts were given. He remains well and had gone without blood transfusions since 1949. He was last seen in June 1957 at which time his blood count was normal.

The exhaustion of the blood forming organs in this case was seen in the peripheral blood stream and in the sternal marrow biopsy. Dr. O. Z. Culler's report, at the time of referring this case, stated: "Chronic Leukemia (proved by bone marrow biopsy) with hemhorrhagic diathesis." Here the immature forms accounted for the inability to produce white blood cells for the general circulation and thus showed where the exhaustion lay. The exhaustion was therefore extreme and the blood picture in consequence was atypical as occurs in terminal phases of disease generally when scrutinized carefully. It had run its course.

This case with the few others of the leucemia classification are offered to show that the structural pathology as the blood or marrow picture is really a result of the integration of the pathogen with the host cell so as to make the latter nonfunctional. In this case an actual atrophy of blood cells or rather an anaplasia results just as a hyperplasia resulted from the first or stimulating action of the pathogen. The cases also demonstrate that the oxidative removal of the pathogen from its combination with the host cell leaves the latter in a good functional status. We showed earlier that the pathogen can not be removed hydrolytically and that an oxidative cleavage is required. Here it is demonstrated that normalcy does result as a result of this oxidative cleavage and the pathogen is no longer to be found.

MYELOGENOUS LEUKEMIA
With Irradiation Leukopenia
In An Adult

(Contributed by Dr. Baldor of
Tampa, Florida)

Neoplasia as In Diagram IV

A similar case in an older person should be described.

Mrs. J. W. L., age 47 years, came in 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 an enlarged liver and spleen and cervical lymphatic enlargement. The breath was offensive with gingival bleeding. Dental abscesses were present. Her blood picture showed red cells, 3,160,000 hemoglobin 57%, leucocyte count 14,800, with poly. 88%, lympocytes 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 six weeks. This did not improve her condition. The bleeding went on and the weakness, fever and pains continued on their course. Bone marrow slides showed definite abnormalities suggestive of Myelogenous Leukemia. Two doses of the carbonyl catalysts were given because of the irradiation, one on December 13, 1948, and the other five days later. Improvement showed up soon. The fever was gone in five days. The painful enlargement of the liver subsided slowly. The oral bleeding and infection likewise cleared up. The blood count March 15, 1949, showed red cells 3,850,000, hemoglobin 69%, leucocytes 8,500. Up to August 5, 1949, she remains well, does her housework normally, and has gained ten pounds in weight. On June 16, 1949 the red cells were 4,000,000, hemoglobin 72%, leucocytes 7,000. She received no blood transfusions after the catalysts and the blood improved naturally. The chest signs in this patient, like the former, improved slowly. By the end of 1950 her enormous spleen, which formerly had reached to the left hip area, had receeded to a normal position under the left hemithorax. The last blood count which

was taken in May 1955 showed red cells, 4,150,000, hemoglobin 70%, leucocytes 3,500. She remains well and has not had a blood transfusion since the time of treatment in 1948.

ACUTE LYMPHATIC LEUKEMIA

In A Boy

Contributed by Dr. Arturo Guzman
of Montevideo, Uruguay

Neoplasia as in Diagram D

F. H.—No history of leukemia in the family.

P. H.—P. F. age 12 years, treated January 8, 1956, in Paris France. The onset was rather rapid after a period of ill health. The symptoms were classical with the petechial hemorrhages under the skin and in the mouth, cough, and symptoms of anaemia with great weakness. The red cell count was 1,500,000, the whites were 232,000, lymphocytes in very great predominence, large mononuclears and immature forms.

P. E.—Blanched-out appearance with hemorrhagic spots of various sizes under the skin generally, especially legs, arms and body. Foul breath, gingival bleeding. Some mediastinal dullness increase observed on right side, spleen and lymph glands only moderately enlarged . Very weak, slight fever, cough.

The treatment. Two cc. of carbonyl catalyst and supportive measures were instituted. The recovery was steady with occasional reactions of achiness, periodically.

Results. In August, 1956, all signs of the disease had left. His strength had normalized, and the blood counts were platelets, 350,000, red cells 5,100,000, white cells, 7200, Polymorphs, 76%. No spleen or lymph gland enlargements were found. Blood coagulation normal. All bleeding and its effects had disappeared. The increases in red cells will possibly give way to a count slightly less than normal and then increase to normal again. No reaction for a focus of infection as a starter was identified except the sore throat which is difficult to interpret in this respect. This case should be followed for the accepted 5 year period before establishing it as a cure. The results that were obtained are gratifying and for this reason the case is reported at this time.

CHAPTER VII

ANTERIOR POLIOMYELITIS[1]

The group of cases offered here illustrate the two well recognized types of host cell virus integration, the lytic and the symbiotic types. The third type, which we have to assume on the basis of clinical data, receives no contribution from the cases to be discussed here as it receives from the cases of virus infections in animals we have to report. This is because the "Polio" cases given below were never vaccinated against "Polio." Nor were any serological tests reported showing a response to some "Polio" virus. They are instructive in other directions however, and have been proven factually uncontradictable in the United States Federal Court. They show that in the acute lytic and symbiotic types of integration the virus can be separated from the host cell leaving the latter normal as to functional ability, and the virus is no longer found, or at least is harmless. Moreover, in the long standing symbiotic types of integration, after years of extensive paralysis and muscle atrophy and even developmental failure, 90% of the defects have been restored to normal even after twenty years of complete invalidism. These cases therefore are of intense professional and public interest under ordinary sociological circumstances.

In the first place viruses are recognized because of the diseases they produce, and because they fall in the filterable category. To produce disease they must draw energy off from the host cell's vital processes, and the energy stored in vital host cell structures during their synthesis. Such energy must have a place to go to be withdrawn. That is, there must be an appropriate synthetic process into which the energy can pass and be used for its activation. To do this there must be a full set of structural units to enter a viral synthesis. Incomplete structural material cannot engage in the synthesis and so no host

[1]To save space here other virus diseases in man will be discussed in the complete text. Paralytic diseases in animals as distemper and rabies will be given in the section on animal infections.

cell energy is withdrawn, and no disease is produced. We have concluded that since real immunity to viral infection as in measles, chicken pox and the like is dependent on overcoming an active infection and is quite permanent, that the defeated virus is present in the host cell in a form that is incomplete and cannot vegetate until all the structural units are present for complete virus synthesis. Hence the basis for a third type of virus host cell integration that confers immunity is conceivable, as the part present occupies the position a complete pathogenic virus would take if it were open. This point could take much more discussion.

The third type of integration is a theory assumed by the writer to account for a vaccination accident that occurred during the first year of his practice. There were rumors of a mild small pox scare in Detroit, and two people in apparently good health applied for protection. The same vaccine was given to both within the same half hour. One showed a mild vaccination reaction, and the other came down with rapidly fatal Small Pox. No one else showed the disease in this way at the time. The writer could only assume that something in the patient, added to what was in the vaccine, together formed a rapidly fatal virus. One assumed that since the vaccine tested out "innocent" in the other patient, obviously the victim was carrying the part that made the innocent vaccine fatal—the patient carried the dangerous part even though it was not injurious alone. But also it appeared that viruses were made up of units, like a deck of cards that could shuffle out parts which could contribute or receive fatal units from another virus, and since at the time the cases of small pox were all mild and few in number, we assumed finally that the patient carried a mild infection that did not amount to anything alone, but which received fatal units from the vaccine that permitted the assembly of a complete set as constituted the parent fatal type. This is still our opinion. This case then could represent type III integration. In fact the victim was already protected by the incomplete virus he carried integrated with his tissues. This is our answer to the fatal effects of the two polio vaccines. Broken down products of virus of appropriate structure could diffuse into

host cells carrying type III integration, and build up a fatal breed.

The type of ligation with the host cell would be the same that happened in the fatal infection. The same position is occupied and this would according to our hypothesis be via an azomethine bond or a free radical double bond addition. But since the vanquished part is unable to vegetate it causes no symptoms except a hindrance to the oxidation so as to possibly cause easy fatigue, loss of energy, and the taking on of fat. Then when vaccination comes along, be it Swine-Pest, Aftosa, cinemosa, or some other type, an apparently healthy animal or herd will come down with a violent fatal infection. Farmers, having had this experience, refuse to have their animals vaccinated. However the logical plan would seem to be to make serological tests and if any show positive to not give a vaccine against that breed.

Of course vaccines of the Salk and Fleury types can carry live active fatal viruses because of failure of members present to lose units belonging to parent fatal types, these on reassembly to form the progeny even in the egg embryo culture, shuffle out as fatal forms. There is also the protection against formaldehyde addition by condensation with aldehydes that should have been split off before exposure to the formaldehyde. This could be done at a pH of 3 or 4. But this is not practiced. However such protective aldehyde groups do split off in the patient when the tissues get too acid from exercise, and acid diet, to liberate the fatal forms with active integrating amine groups. Thus, we believe, it is important to the public health that in preparing these vaccines, these protective aldehyde groups be first removed before the virus is treated with formaldehyde to inactivate the virus. Here we have indication that vaccine and killer viruses do not make the same atomic union with host cells. The true disease virus joins via its amines group in the way described. The vaccine has no free amine group if it is a well prepared Salk, or more properly, Cumming type. For Dr. Cumming developed the formaldehyde method of inactivating viruses. Since the killed virus vaccine has no amine group to form the azomethine bond, its union with the host tissues is different; and that accounts for the brief period of protection they afford,

if they in fact afford any at all. It is probably mostly superficial, while the live virus makes a real integration.

One must take present statistics on Polio infection with a grain of salt, since they are so contradictory Commerce seems to have polluted statistics. An error is possible however, since most people show positive reaction to one or other Polio virus, and when such persons take down with flu or an intestinal intoxication, the serological test is interpreted as meaning the sickness in non-paralytic Anterior Poliomyelitis. Thus the vaccine statistics have an ally to any way commerce wishes to interpret them. Our cases are not Polio unless paralysis is firmly established. No one can call a case polio on any other basis.

RECOVERY FROM CHRONIC SYMBIOTIC TYPE PARALYSIS AND ATROPHY ESTABLISHED THREE YEARS**

Treated in collaboration with
Dr. Julian Baldor

Function Suppression as in Diagram V, (b) Symbiotic Type

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 oxidation catalysts were 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 born 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* **

Treated in collaboration with
Dr. Wendell Hendricks.

Suppressed Function as in Diagram V, (b) Symbiotic Type

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 cm.). The left leg was slightly better, but useless. She could not stand without help. She also suffered with migraine headaches. On April 7, the oxidation catalyst 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 catalysts 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*

Treated in collaboration with
D. H. Arnott, M. D.

Diagrams V, (a) Lytic Type

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 catalysts were 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 the 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

Treated in collaboration with
Julian Baldor, M. D.

Diagram V, (a) and V, (b)
Lytic and Symbiotic Types

Patient: Sandra F., age 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 patelar 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 carbonyl catalysts were 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 negative 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 percent 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**

Treated in collaboration with
Dr. George Franklin Smith

Diagram V, (a) Lytic Type

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).

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*

Treated in collaboration with
Michael R . . ., M. D.

Diagram V, (a) Lytic Type

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 September 13th and it was 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.

ACUTE ANTERIOR POLIOMYELITIS WITH PARALYSIS* **

Treated in collaboration with
Dr. Wendell Hendricks

Diagram V, (b) Symbiotic Type

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 initiatores.

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 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 oxidation catalysts. 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.

Diagram V, (a) Lytic Type

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 carbonyls 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 or 1932."

This may have been a simple Nissl neutralization case. Yet the speed of the recovery shows that the reagent accomplished the cure, as well as the prevention of sequelae.

However the steady progress of the paralysis with one leg paralysed only when the mother noticed it first, and two legs

paralysed when my examination was made, would indicate a steady progressive infection where the virus was not produced in overwhelming amounts all at once.

What percentage of cases of atrophy and paralysis are to be found that are still more or less curable cannot be answered now. A sweeping test should be made and then the benefits can be calculated, and a guess can be made as to the percentage of the remaining victims that can be restored from invalidism to practically normal activity. Instead of opportunity, obstruction has steadily been confronted, and largely because the surgeon looks upon the sequelae to anterior poliomyelitis infection as coming within his province of service. The surgeon tries to create some mechanical advantage to aid the use of braces or other special equipment, or to immobilize joints and change the limb into some sort of a peg. Such an attempt as this should only be tried as the final attempt to adapt the victim to his environment. However, in the case of acute poliomyelitis or where the chronic symbiotic form is found, would it not be better for the patient to receive the benefit of a constructive form of treatment that has proven its value, than to disregard this therapy at the expense of the unfortunate patient? Where possible, the acute and chronic infection should be removed by treatment and then let nature be given the opportunity to restore herself to as near normal as possible. Nature is so constructive that she can do this and she should be given the preference over the attempts of the surgeon. For after all, is not the future health of the patient of primary concern?

Influenza, Measles, Mumps, Chicken Pox, etc.

Diseases that are self limiting and have no fatality record of note have been denied attention in this investigation except as was necessary to classify the viruses with respect to the type of reagent to which they responded best. It is found that Measles does ideally on the quinone structure, often having recovered practically, that is for the most part in twelve hours after one dose. Mumps has done well on the serial system of carbonyl groups, as has Influenza. The reactions in the latter show an aggravation of all pains and secretions in six hours or

so with marked improvement in 12 hours. The watery secretions cease in that time and the pus germ complications are absent. No heavy green or yellow pus is found except in cases where influenza complicated a bad bronchitis or bronchiectasis, which had to undergo a "housecleaning" to recover also. In our completed text these matters will receive due attention.

CHAPTER VIII

TUBERCULOSIS

Tuberculosis is assuming more importance every day since the recent antibiotics have become so widely used and bred so many surviving mutants of such high malignancy. Indeed experts have had to return to the old surgical procedures they had discarded in the hope that the new antibiotics would prove sufficient.

The ability of a germ to destroy or assimilate a toxin and even use it to an advantage as a food, does more than simply emphasize its power towards survival. The antibiotics used in practice are amine compounds, and in the hydrazide drugs the amine group's toxicity is raised to the highest pitch. Yet the germ is able to conquer it when it gets a good chance. This recalls that the virus pathogenicity is a function of an amine group in our opinion, and the possibility of a surviving assimilation of a virus and symbiosis therewith is a perfect possibility. The finding of a virus in the tubercle bacillus by Dr. Oswaldo Cruz and Dr. Fontes decades ago is a serious matter. Since the loss of pathogenicity of the Tubercle bacillus follows the action of appropriate carbonyl groups, as does the loss of pathogenicity of the most malignant of viruses, the decision has to be settled if or not the pathogenicity of the tubercle bacillus is due to Oswaldo Cruz's virus.

PULMONARY AND BONE TUBERCULOSIS

The cases presented in this section were used either in the Federal Court trials or before the Federal Trade Commission. The documentation of some of these cases consist of the hospital records from the Herman Kiefer Hospital and the Detroit Tuberculosis Sanitarium. Most of the patients had the best of sanitarium care possible and yet they steadily deteriorated until they were considered hopeless by the authorities. They were given our treatment with curative results. Prolonged bed rest

was not needed and the elimination of the germs followed, leaving sterile areas to be healed. This took place while the patients were able to be active and in some cases they returned to work. The proofs indicate that the germs lose their pathogenicity and the tissues were then able to heal and be replaced by normal functioning tissue with a very minimum of scar tissue remaining. Several cases are shown here with their diagnostic and prognostic documentations supplied by the leading tuberculosis institutions of the United States and made by leading experts in tuberculosis.

These cases should be studied and contrasted with those cases that are treated with the new Hydrazide drugs. As time passes, we will see whether resistant mutations in the tubercle bacillus to this hydrazide therapy will occur, as has been the case in the use of the antibiotics.

BILATERAL PULMONARY TUBERCULOSIS
With Large Retention Cavity* **

Treated in collaboration with
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

Re: 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 tubercu-

losis 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 infraclavicular region. Left lung: Considerable mottling throughcut 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 conformatory. 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 cc. of a 12X dilution of the serially arranged carbonyl groups. 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 or became fatigued. 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 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

The testimony of Dr. Omer Hague from the Federal Court trial in 1946 has been paraphrased and reproduced here. It should be studied with the recovery data on this patient.

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 here. 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 peribronchical 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 re-

lated 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 16th, 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 18th, 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.

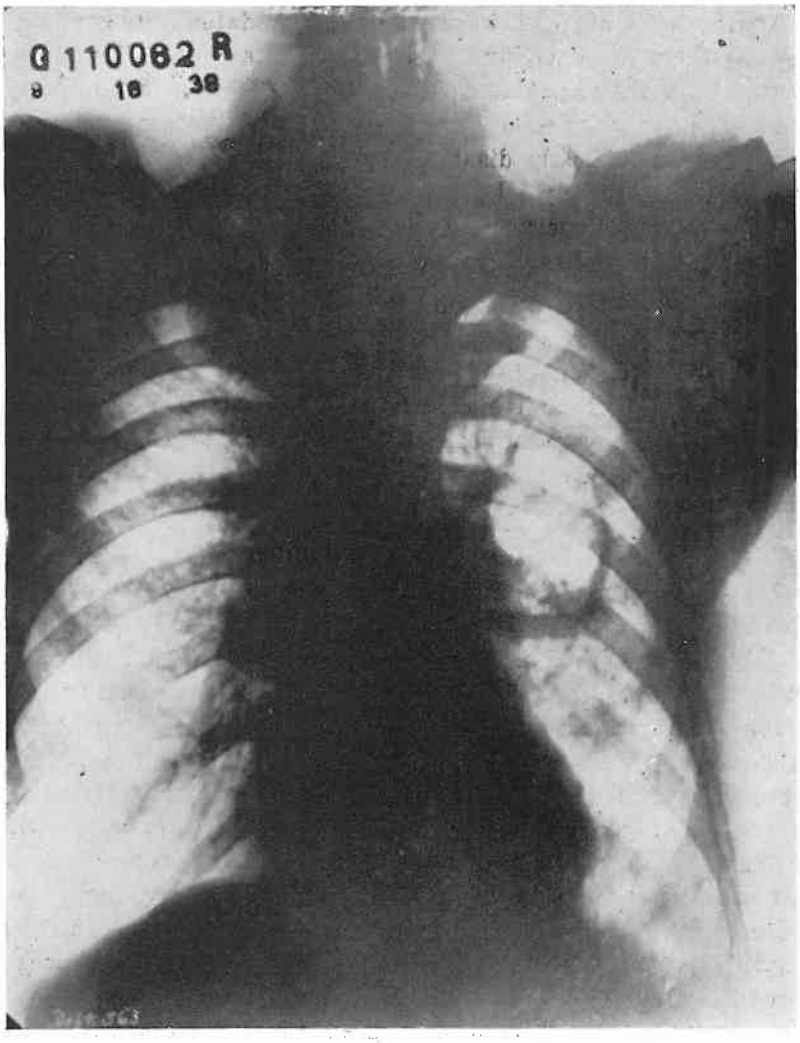
The cavity in the film dated September 16th, 1938, I think is about as large a one as I have ever seen and I would say that that patient's condition would not be a good risk at all.

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 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, with-

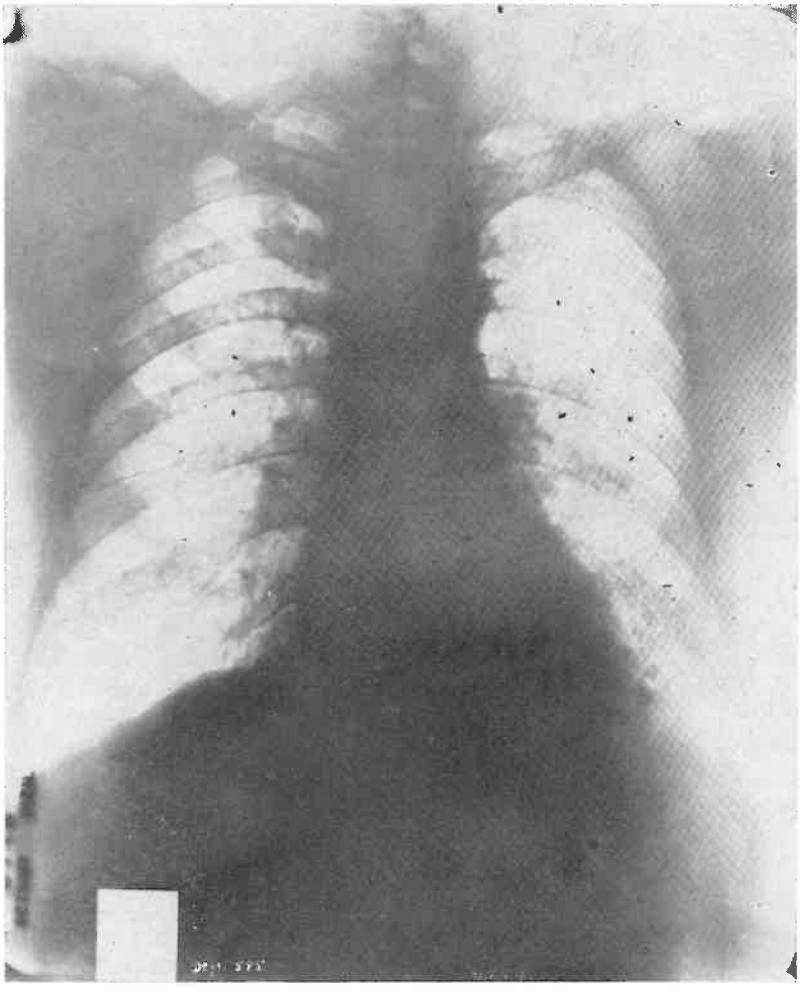


Radiograph I, taken at Herman Kiefer Hospital, September 16, 1938.

out 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 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 work. His sputum has been negative since his recovery. He has continued working and testified in the Federal Court trial in 1946.

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



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**

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 respirations, 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 tuberculous process. She was near death with a tubercular meningitis besides. Two cc. of the carbonyl groups set up in long open chain series was given in the 6X dilution. 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 around 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).

FAR ADVANCED TUBERCULAR PNEUMONIA

Or Acute Miliary Invasion, in Extremus At Time Of Treatment*

Another case of pulmonary tuberculosis in extremus at the time of treatment with carbonyl catalyst 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 2cc injection of carbonyl catalyst was given immediately. She had progressed to this stage from an early subclavicular and apical envolvement 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 hospital doctors wanted 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 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 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, when she appeared to testify at the Federal Court in the writer's defense.

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 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.

HERMAN KIEFER HOSPITAL

X-RAY REPORT

NAME Mary CASE NO.

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.

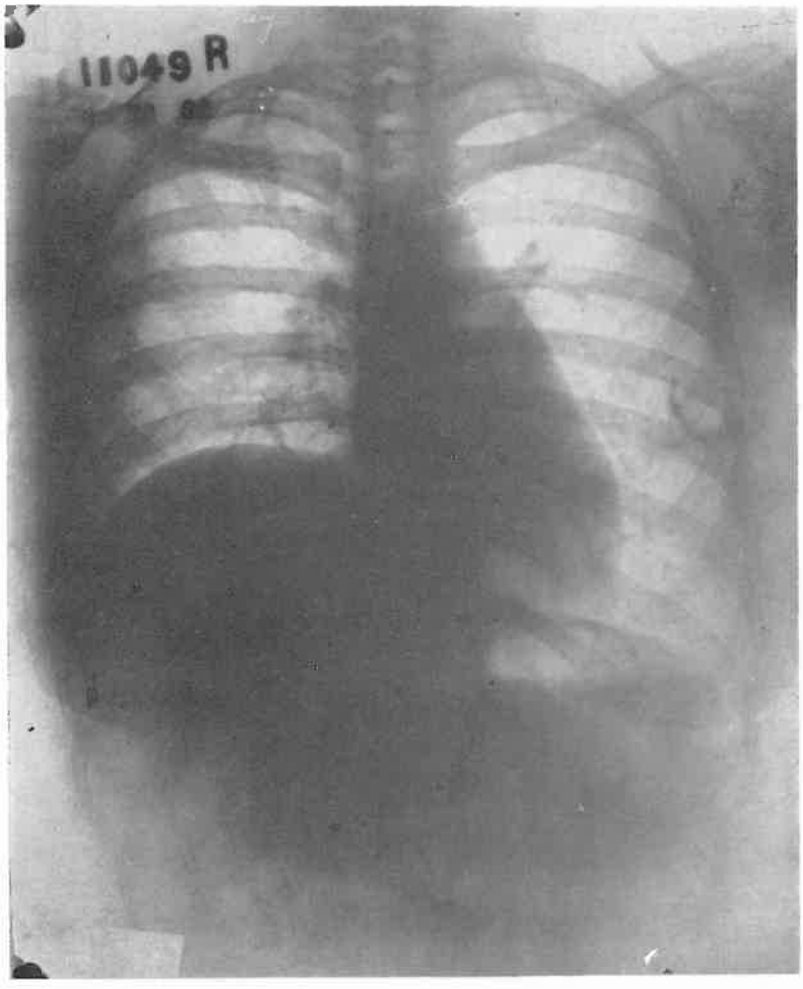
Conclusion: Far advanced tuberculous process of the mixed type, involving the upper third of both lungs. There is a large cavity on the right as described.

Dr. Birkelo - T

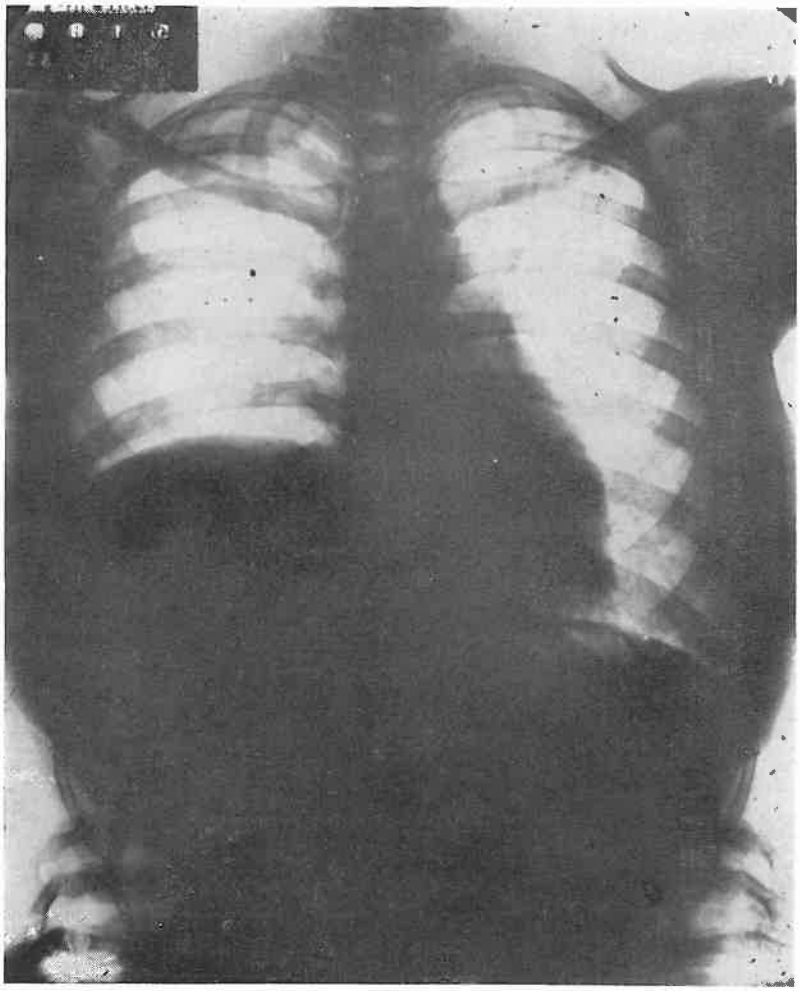
INTERPRETATION

SIGNED

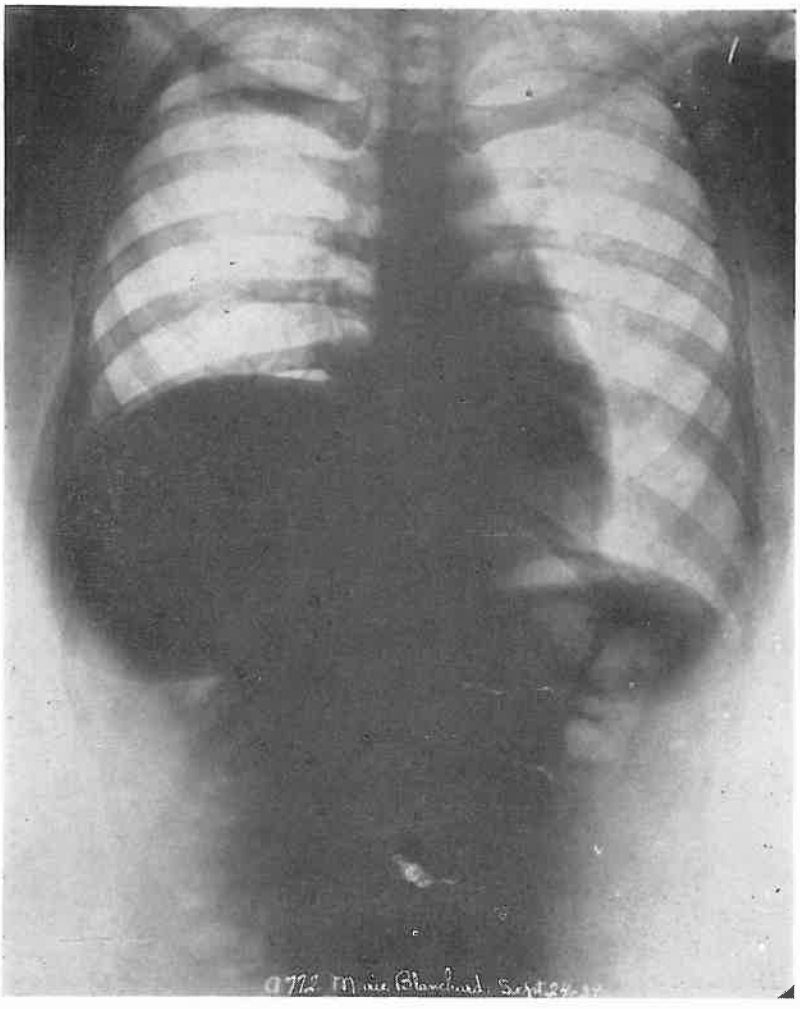
This report shows the condition of Mary B. when she entered the hospital.



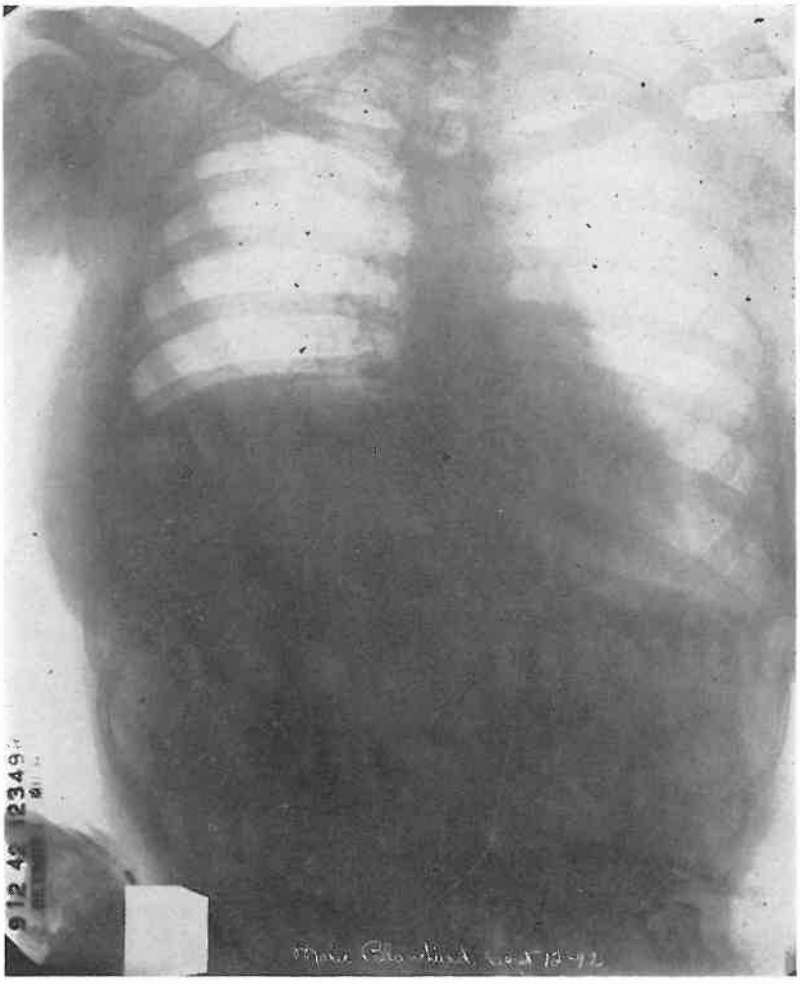
Radiograph I, made in April, 1932 shows advance of disease from August 1931 in the hospital.



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



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*

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 Cora CASE NO. 11884

DATE Mar. 10, 1932 PAVILION E - 1 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 regica, 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, involvingthe upper upper half of the left lung and the upper third of the right. There are cavities on both sides as described.

Dr. Birkle - 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. of the serially arranged 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 testimony on this point taken from the court records 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 breast 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 preceeding, 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."

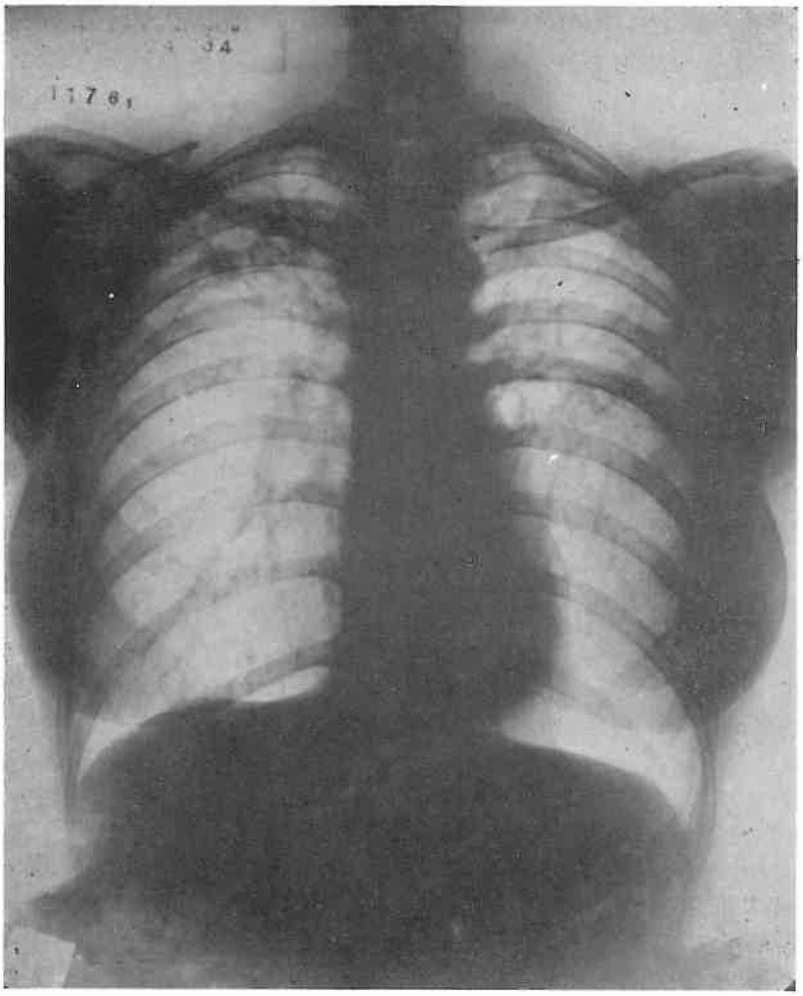
Where the patient is still showing positive sputum, the prognosis is serious. [It should be noted that this last X-ray (5/26/34) was taken over 3 months before the patient was treated by the author.]

Dr. Hague was shown an x-ray taken about seven and one half years after the patient was first treated in Dr. Koch 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."

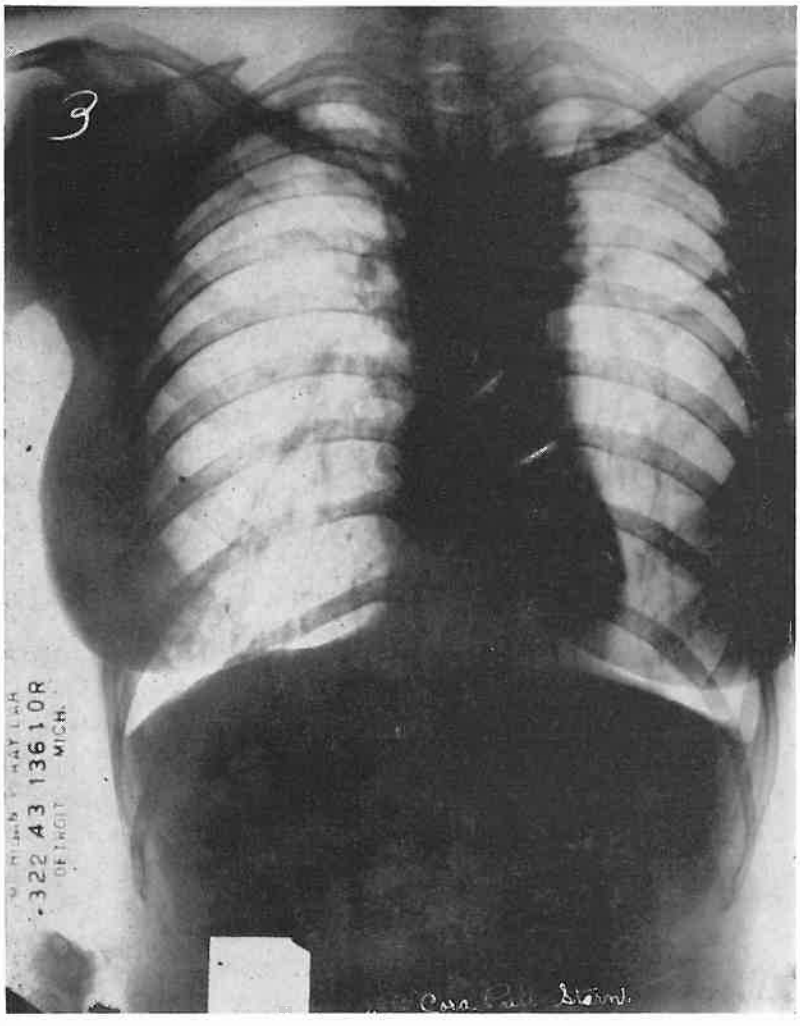
The last film, that had been taken on March 22, 1943, is reproduced here and is radiograph II. Dr. Hague stated: "That it does not show very much change from the one immediately preceding (2/19/42). I would say it is about stationary."

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 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."

The basis for disease in its various exhibitions in this case is demonstrated to be a failure in tissue carbonyl activity which was repaired by the catalytic dose of activated carbonyl groups, given in the form of long chains of carbonyl groups, with free radical terminals.



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



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

TUBERCULOSIS OF LUMBAR SPINE**

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.

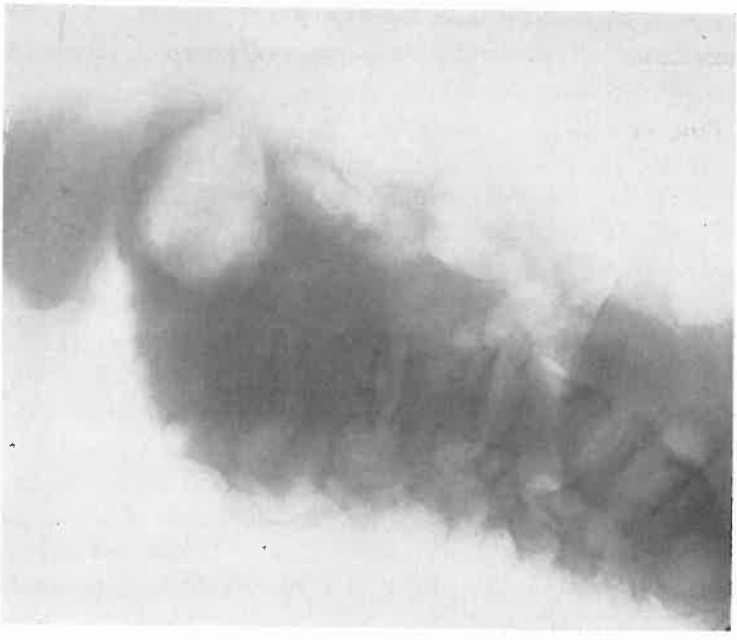
The Family History showed his older sister had far advanced pulmonary tuberculosis.

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.

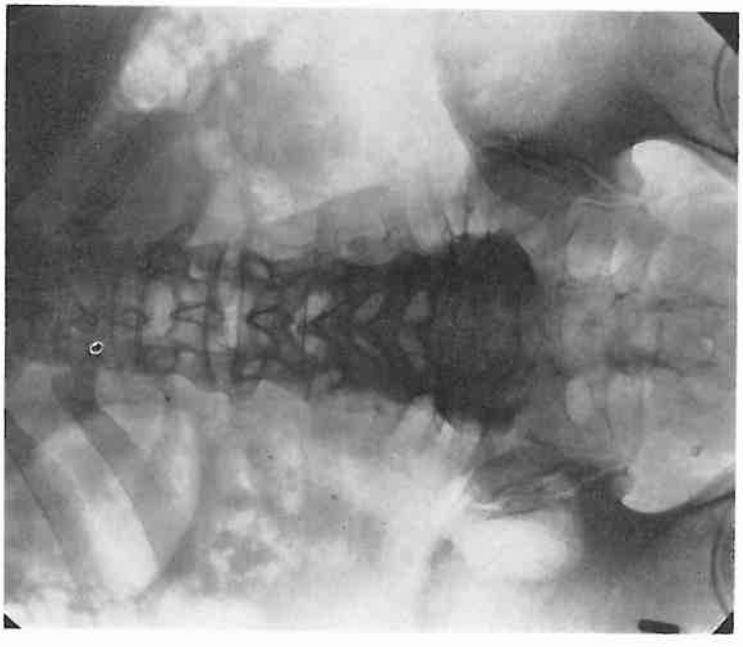
Examination showed a typical tuberculosis kyphosis in the lower lumbar spine. The two lower vertebrae showed a sharp angulation that protruded with collapse of the bodies of the vertebrae. There was considerable swelling of the soft tissues and muscle spasm about the region which limited motion.

We gave him one cubic centimeter, 12x dilution, of the 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



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.

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 in a rigid shell for many years. Where there is present a metastatic lesion 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 in large numbers 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 IX

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 Osteomylitis In A Boy

Treated in collaboration with
Dr. L. Andrews

N. R., age 5 yrs., 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 immedately 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 mumification 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 Aug. 2, 1940. Neither this nor other measures as sulphathiazole made any impression on the advance of the disease. The patient was given up as hope-

less and taken home on August 2, 1940 and present treatment was continued for a few more days, but with no improvement.

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 was 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**

Treated in collaboration with
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 carbonyl catalyst.

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 mumifying process set it. 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 group 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.

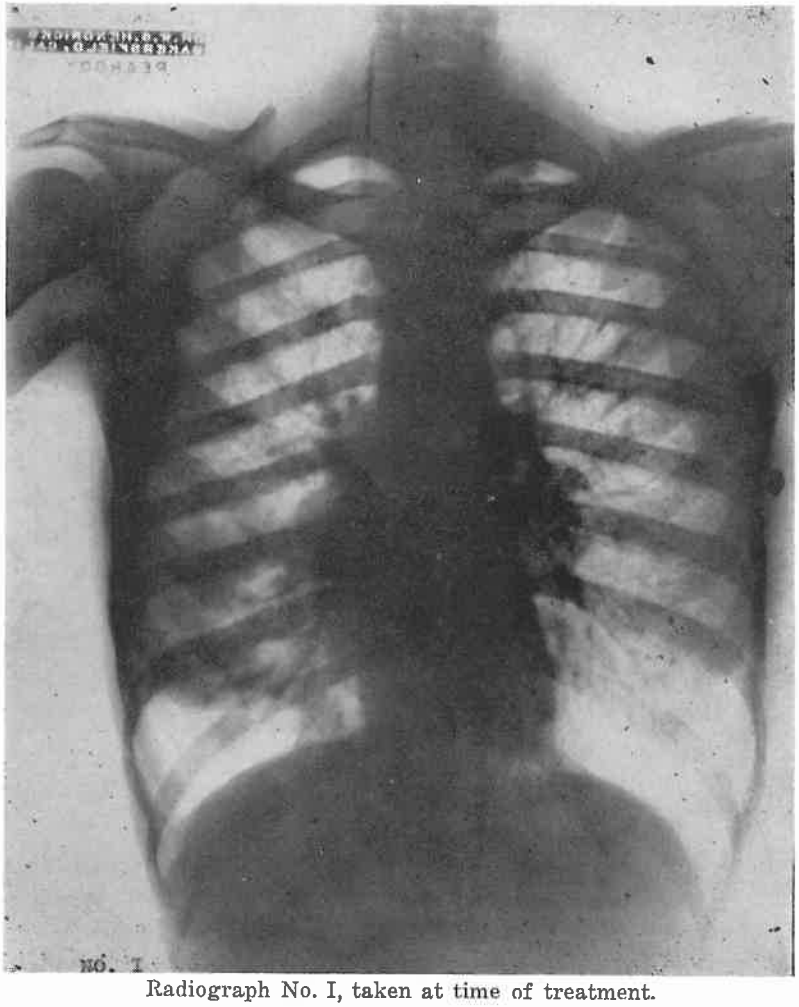
The metabolic fault in the tissues during chronic infection is well represented in bronchiectasis, for here not only is there an excavation of the lung substance starting in a bronchus, but the bronchial walls carry the infection forward so that lobectomy is the only hopeful procedure, from the orthodox 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 consequencial to the restoration of an efficient oxidation catalysis.

ADVANCED BRONCHIECTASIS
With Asthma*

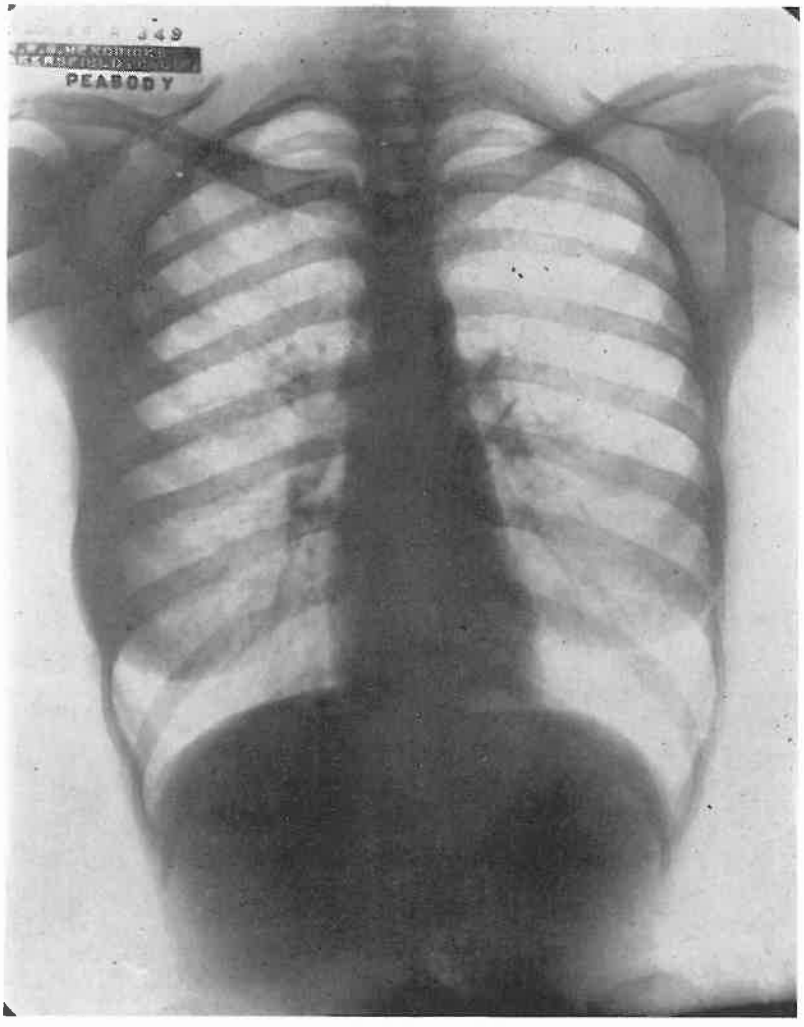
Treated with collaboration of
Dr. Wendell Hendricks

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.



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

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

Carbonyl catalyst 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 nor-

mal (98°) pulse 72, and the blood pressure 110/80. In another month 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.

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 fibrosis to cause functional deficiency or aging.

This type of fibrosis is different from the fibrous connective tissue of normal sheets 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.

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 envolved. 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.

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 carbonyl catalysts 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 control, 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 X

SEQUELAE TO INFECTION

FAR ADVANCED ARTERIOSCLEROSIS

With Senile Dementia

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 catalysts 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

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 for 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. Wt., as regularly, is 157 lbs. Ht. 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 cc. of the 12x solution of the serial 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.

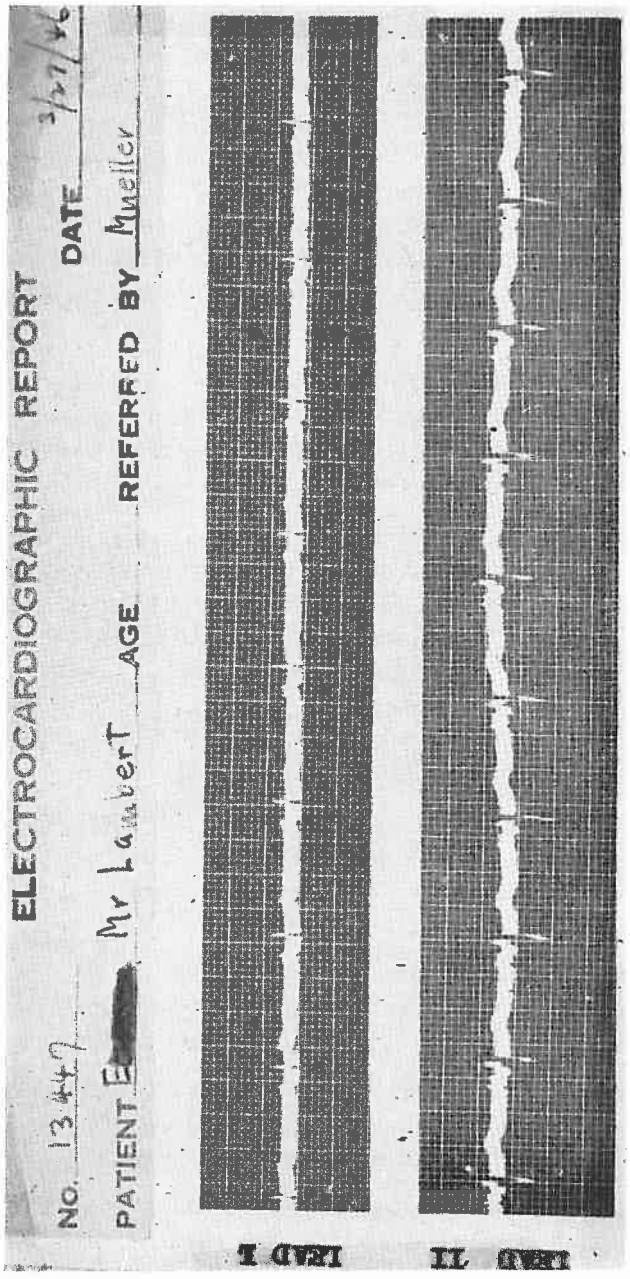
Electrocardiogram on this date showed Rhythm regular at rate 85, P and PR intervals are normal throughout, T_1 shows a late sharp dip. T_2 and T_3 are upright and normal. T_4 is deeply inverted. QRS complexes show low amplitude. QRS_1,— + ½ —1; QRS_2— —3; QRS_3— —5. There is absence of the R wave in 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.

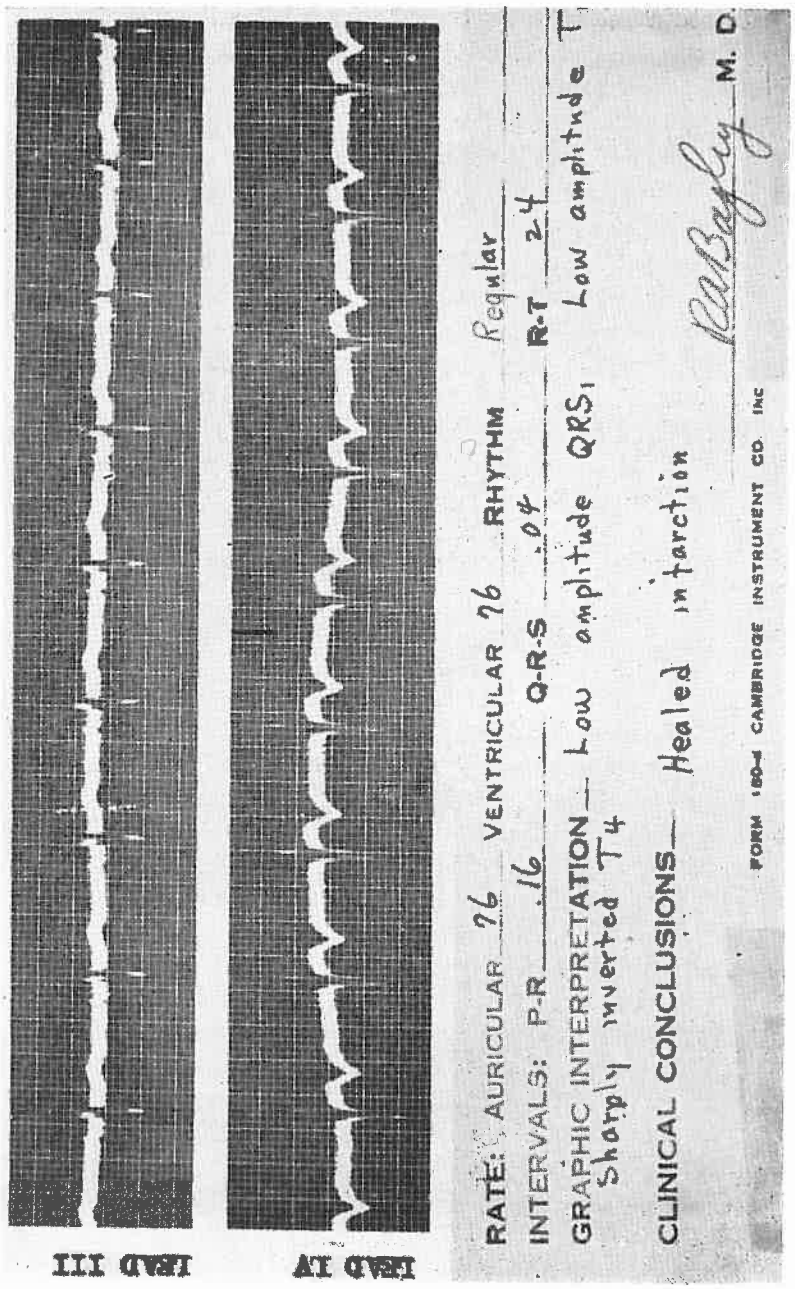
Electrocardiogram taken two years later, March 27, 1946 by the same expert reads as follows and is submitted. Regular rhythm at rate 75, P, and PR intervals normal. Amplitude of QRS 1—1½. Low T_1 ,Inversion of T_4.

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


ELECTROCARDIOGRAPHIC REPORT
DATE 3/7/46
No. 13447
PATIENT E[redacted] Mr Lambert AGE
REFERRED BY Mueller
LEAD I
LEAD II


LEAD III
LEAD IV
RATE: AURICULAR 76 VENTRICULAR 76 RHYTHM Regular
INTERVALS: P-R .16 Q-R-S .04 R-T .24
GRAPHIC INTERPRETATION— Low amplitude QRS; Low amplitude T
Sharply inverted T4
CLINICAL CONCLUSIONS— Healed infarction
M. D.
FORM 180-4 CAMBRIDGE INSTRUMENT CO. INC

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.

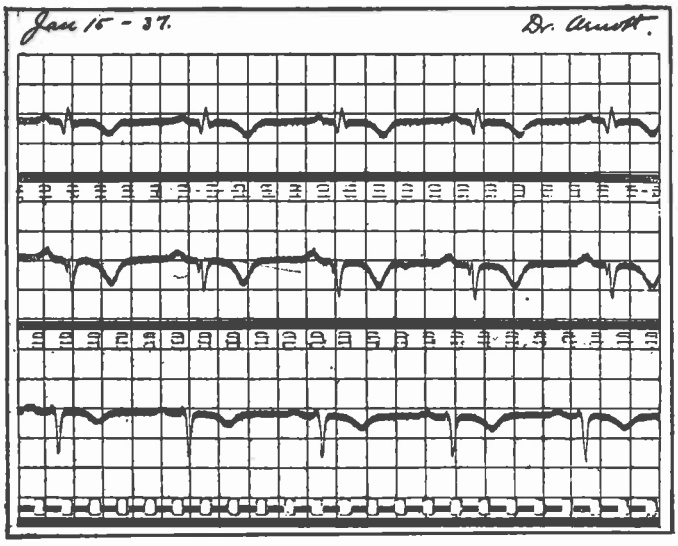
We attribute this subsequent attack of coronary thrombosis to a return to the living conditions that were etiologic in producing the disease in the first place. It took over eleven years to restore sufficient pathology to cause a coronary infarction again while no further treatments were given. Had this 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. See Chapter XIV.

CORONARY OCCLUSION*

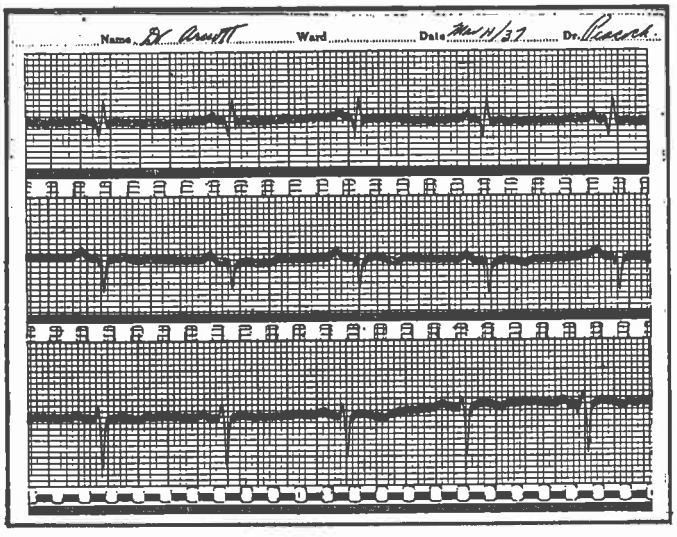
Treated in collaboration with
Dr. David Arnott

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, the carbonyl catalysts were 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 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



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.

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.

CORONARY OCCLUSION

Mr. L. K. in August 1946 had a heart attack of a different type. Though the attack caused some pain, he attempted to continue with his work. A cyanosis told of the failure of the heart to function properly. The blood pressure was found to be 35 mms. of Hg. over zero; that is three and a half centimeters of mercury for the systolic pressure and no diastolic pressure could be observed. He was moving about in bewilderment with slight pain in the back of his head and neck, but not the terrible pain that would stop him in his tracks as in the previous case. His situation was not so dramatic as in the former case though he kept the failing heart turning out more toxic sub-oxidized metabolites which aggravated the condition. A **dose of the carbonyl catalysts was given and in two hours the blood pressure rose to 80 mms. of Hg. over 40 mms. of Hg.** In two more hours the blood pressure was 100 mms. of Hg. over 60. The next day it gained to 120 over 80. However, he had to rest quite completely from 10 to 12 weeks before work was resumed.

He did well for four years, when another milder attack with practically no pain took place. Another injection of the carbonyl catalyst was given. During this four years he was very active for his condition and did more than one man's work. This called for excessive cardiac function. Had the disease response presented pain as a prominent feature, a better check on his regime could be held and the condition could be kept from recurring. However, the second dose of catalysts and a few weeks' rest and then easier work and a pleasure trip brought him back into good health. While coronary insufficiency and

sclerosis was present, jelling of the blood rather than true clotting or embolic occlusion was likely the actual condition. Had the treatment been delayed true thrombosis and infarction would be expected. In such instances, careful dietary control must be followed and good bowel elimination must be had. The cardiac activity must be held down so that no incompletely burned metabolites are formed to maintain or increase the interstitial fibrosis.

BRIGHT'S DISEASE*

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 the carbonyl catalysts 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.

PITYRIASIS RUBRA UNIVERSALIS

Treated in collaboration with
Dr. E. Klaveness

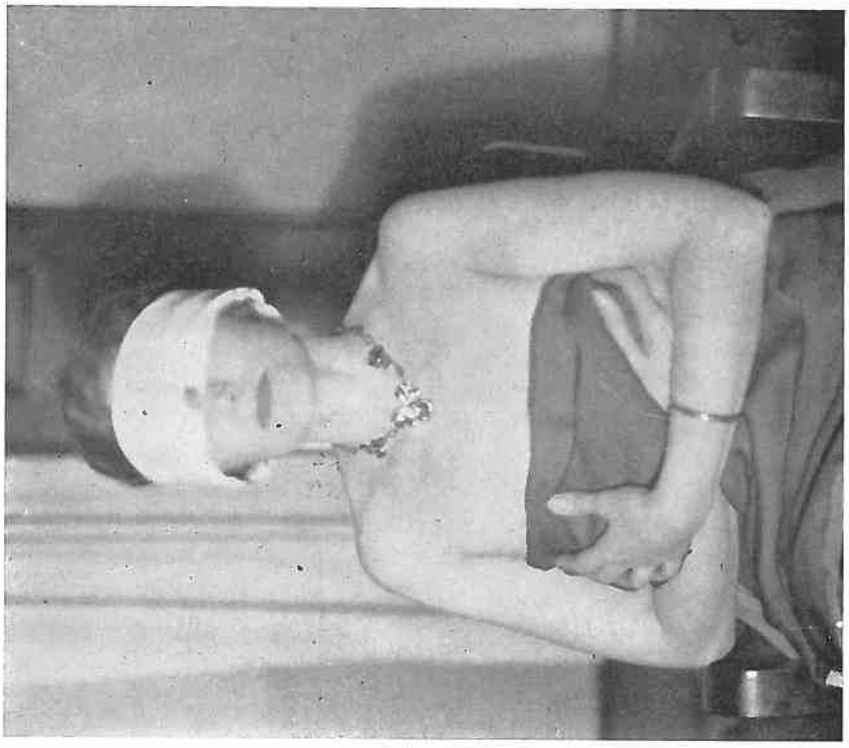
This "incurable" disease recovered after 2 doses of the carbonyl catalysts. This case in a man of 58 years, treated February 25, and in April 1950, had been suffering since October 1948 from a skin disease that answered the von Hebra description perfectly and was diagnosticated as Pityriasis Rubra by 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 wt. 193 pounds, February 25/50, 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 albumin, 2.5 grams per liter.

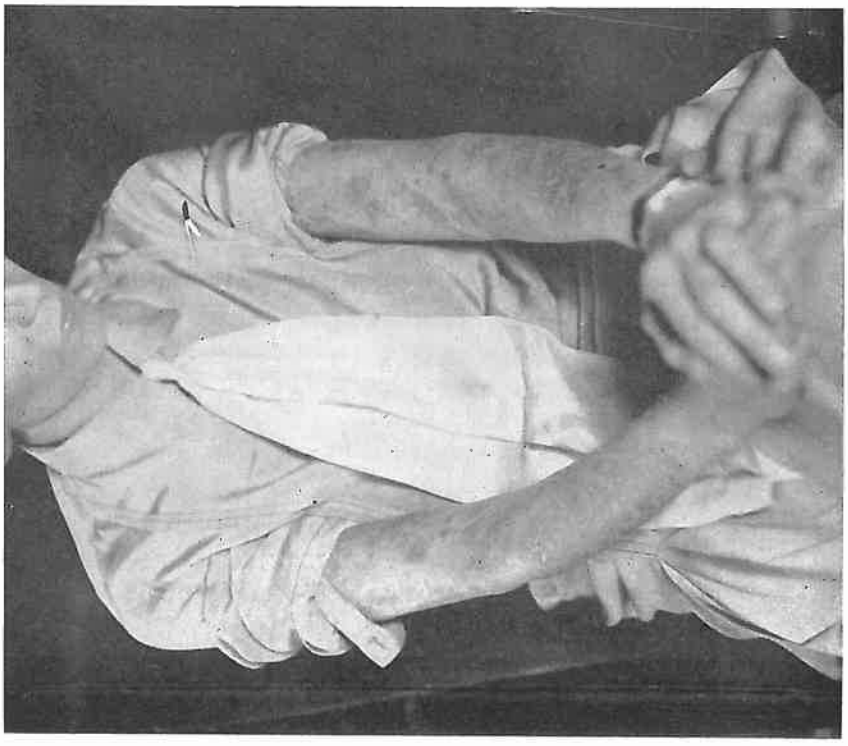
Recovery set in promptly after the carbonyl catalyst injection with no loss of weight. The scaling stopped, first on scalp then the trunk following the first treatment and scaling stopped on the extremities after the second, with change to normal color, gain in strength and weight and clearing of the 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

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 tonsillitis 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 twelfth week only a few very small spots were observed and these were absent at the fourteenth week. She remained well, thereafter. The tachy-



Photograph No. II, showing patient after recovery.



Photograph No. I, taken at time of treatment.

cardia 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 example 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.

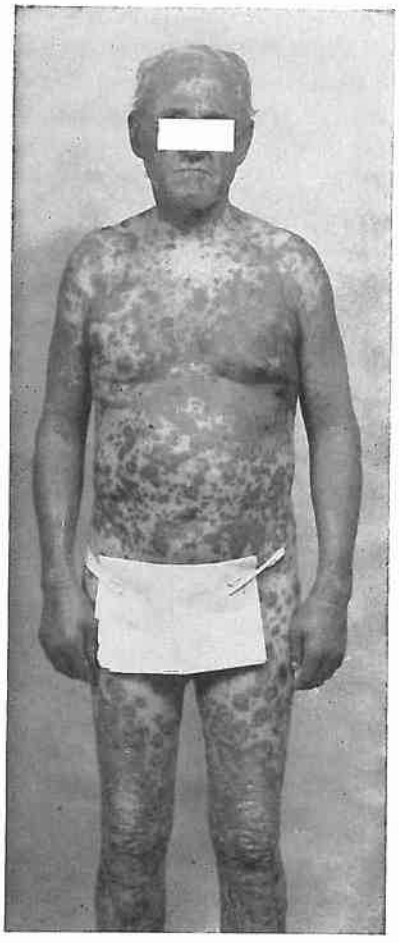
PSORIASIS UNIVERSALIS

Treated in collaboration with
Dr. Chester Dove

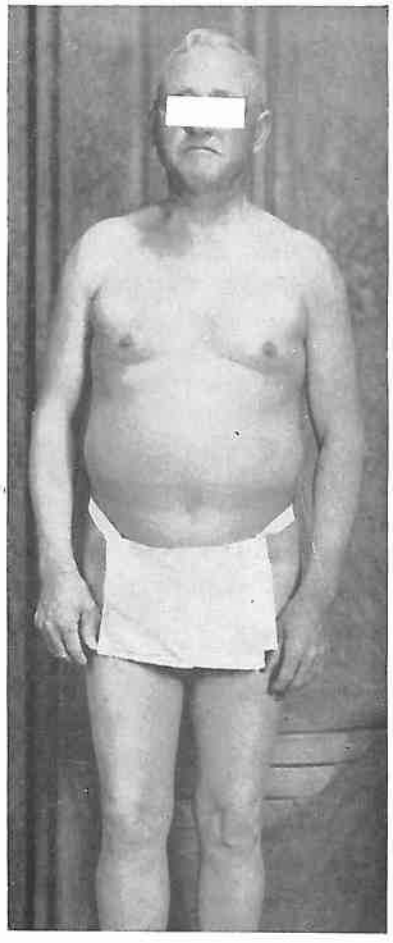
In this case of Mr. C., age 64 when treated in July 1934, showed universal redness from top of head to soles of feet with terrific amount of silvery scaling large and small, leaving a bleeding base with loss of hair, eye lashes, and finger and toe nails. The soles of his feet separated off as foul gangrenous 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.

He received two injections of the carbonyl catalyst, one on July 17, 1934, and the other in October, 1934. He made a com-



Photograph No. 1, taken at time of treatment.



Photograph II, taken after recovery.

plete recovery. In Februry 1957 he told Dr. Dove that there had been no recurrence of the psoriasis since his recovery in 1935.

MULTIPLE ALLERGY

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 University of Michigan

Hospital to be sensitive to 60 different things including fur, feathers, 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.

In May, 1934, we gave her a 2 cc. dose of the 12X dilution of the 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 chain reaction 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 fundemantal pathology in all is the block in the oxidation process which has to be corrected.

ALLERGY OF CEREBRAL CENTERS

On Infectious Basis

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. R., 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

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

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 renouned 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 XI

OBSERVATIONS IN ANIMAL DISEASES

DAIRY CATTLE PLUS PUS INFECTIONS

We are indebted to Dr. David Arnott for development of the use of the carbonyl catalysts in the diseases of Diary 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 virulance 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 gangre-

nous 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.

It was Dr. Arnott's contribution to the court defense of our work that went a long way toward preserving this therapy for humanity. The United States government claimed that the cases we cured, never were sick, and if they were, they did not get well, and if they got well, some other therapy cured them, and if no other therapy was used, they were cured by psychology. The cure of dairy cattle of infections and metabolic disturbances from which no other treatment could free them, would have had to be done by psychology if our treatment did not do it, and as the experts from British Columbia under Dr, Arnott's leadership demonstrated, the cures could only have come from our treatment, as the government witnesses had to admit, psychology could not enter the picture at all.

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 bacterio-

logically 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.

VIRUS INFECTIONS IN ANIMALS

RABIES[1]

Lytic Integration As In Diagram V, (a)

The Virus

There are three varieties of Rabies virus at present, the original "Street" variety, and the two adapted types that are grown on chick embryos and that are fixed to nerve tissue

culture by brain innoculations. Both adapted types tend to revert back to the parent "Street" type under conditions that offer structural units possessed by the latter, but lost in the cultural adaptations to only brain and chick embryo vegetation. Virulence is largely lost by the two types of adaptation; and this fact forms the basis that encourages the production of the Fleury type live virus vaccine.

Immunity to virus diseases has not responded well to the killed virus vaccines, and as it is recognized that the only immunity of value to virus caused disease follows the recovery from the active infection, the effort to produce the disease in a mild readily vanquished form seems logical. However, the most diligent efforts to master the details that must be encountered have shown that real success has not been attained as yet, and as we are combating the most basic laws of Nature, the hope for success is not too promising either. Cow pox is a natural live virus vaccine against Small Pox. Yet it must be an attenuated form of the latter, since under favorable conditions it becomes a most virulant killer small pox virus.

This has happened under my observation with Aftosa vaccine several times, and could be the cause of the scandal with the "Polio" vaccines. Practical experience convinces one that mild or defective virus of "Polio," Small Pox, and other diseases, infects the public as widespread as our Type III integration with host cells on a large scale, and that such infection for example gives the serological reactions for the "Polio" sensitivity or "immunity" that is diagnosed as Infantile Paralysis when the serological tests are given those with the "flu" or an intestinal indigestion, and classified as non-paralytic Poliomyelitis. However, an innocent Polio vaccine may supply the units which when added to those of the silent infection (neither one of which could supply the required units alone for a provirus that could mature to engage in actual vegetation), would together make up the material required for a parent killer type, and thus precipitate a highly fatal epidemic in any large city. This is true because so many people without "Polio" are serologically sensitive, and are protected from the virulent infection coming

[1]This is a report on the first cases of Rabies ever cured. The report has been delayed one year to make sure of the permanency.

from without by this natural vaccine type infection. If it is let alone it protects, but when one tries to improve upon it by any type of virus vaccination, the disaster can be created within the system of the victim. What applies to one person applies to most of the community. Many virus infections including cancer may be represented by this Type III silent integration.

However, in Rabies the situation is quite different, and if not vaccinated previously no such catastrophe would be expected. The epidemic to be described therefore must be accounted for by the presence of active live, fully virulent Rabies virus in the vaccine, since it attacked calves that are not exposed to any type of Rabies virus at all, and hence have nothing to add to the vaccine to make it malignant.

There is another consideration that deserves thought, namely, that the epidemic we will describe took place pre-eminently in bull calves several months old. Here the activated amines and amidines produced by the testis may possibly offer the attenuated vaccine virus the amine groups it needs to make pathogenic azomethine bondings with the host cell, and which amine groups might conceivably be taken up into the virus structure as happens when an excess of an amino acid supplied to the host culture, is built into the virus's protein in such proportions as to change its serology in a transmissable way. If such a thing happens, bull calves would tend to change an innocent attenuated virus to a killer type. Still females were also affected. So this explanation is weak and can be discarded for a more practical one that follows.

In this epidemic short needles were used to make the injections of the vaccine. Since the Zebu possesses a skin structure with a non-elastic peniculus that could hold the injected material a long time and prevent its absorption, the short needle would only deliver the material into the muscle in the calves and not into adult animals. Such protection may account for the unusual resistance of the Zebu to snake-bite poisoning. In the present epidemic it probably protected the larger animals. Our suspicion is that the vaccine contained virulent Rabies virus that had taken advantage of some encouragement to revert back to the parent un-attenuated type, before it was injected.

One must remember the tendency to revert to the parent type is not lost even by prolonged passage through attenuating culture as it is a basic protective response.

The Integrated State

The briefest review of virus parasitism demonstrates a common pattern of action to which the facts we have to contribute here give confirmation. It is now agreed without exception that once a pathogenic virus has penetrated its host cell and become integrated therewith, that is, about a minute and a half after penetration, no amount of immune serum or vaccine can rescue the host cell from destruction. Evidently the union between the host cell and the integrated virus is so very intimate and fixed that displacement by other combining substances is entirely out of the question. Immune sera only partially neutralize the virus anyway, and the latter is easily separated therefrom. The host cell-virus bonding is much stronger. That is why we found it necessary to work out a system of oxidizing the virus off at the azomethine bonding so as to restore the host functional carbonyl group and destroy the virus' amine group it must need for further vegetative integrations. The virus is left harmless then, and there are other indications that it is oxidatively fragmented and completely destroyed as well. It appears too from our aftosa experiments that after recovery following the treatment, the animal is not subject to further infection, as the annually recurring epidemics of aftosa do not affect the cured animals, but do attack the new animals brought into the herd, if they are not treated. Our hypothetical azomethine bonding is again indicated in the cure of Rabies since here too host cell function is returned while the virus is made harmless, as in the cures of Anterior Poliomyelitis with extensive paralysis and atrophy, in cinomose, in the final phases of Swine-Pest, and in Newcastle's disease in chickens, in all of which non-reproducing nerve cells are invaded by the virus. And so function can only return when the virus is eliminated and the host cell groups it combines are restored. (Diagram VII and Chapter VII). Among reproducing cells the lactogenic cells and myocardium of aftosa affected cows are restored to function.

Rabies offers a better demonstration, however, since the

disease is one hundred percent fatal and kills in about four or five days after the onset of symptoms. Its course is definite and cannot be confused with any other disease.

Vaccine Intoxication

One must carefully distinguish between the toxic effects of the vaccine and the inactivation of nerve cells due to actual viral integration with the host cell so as to establish a vegetative parasitism. The intoxication by products of virus disintegration probably has an immunizing effect, and is due to products of virus breakdown that have not been identified as yet. They attack the liver, and in proportion to the damage done there, the recovery will be more or less rapid or transient. Many humans who have received anti-rabic vaccine show these toxic effects. They develop rapidly once they are started and then subside. The nervous system is poisoned. There is dizziness. Incoordination of movements may even occur, and other symtoms of a "Drunk," with nervous excitement. The animal may attack, but humans feel "flighty", etc. There is no true reflex block however, and no real paralysis has taken place. Thus the animal will drink if thirsty. One must remember the old adage that "you may take a horse to water but you cannot always make him drink." The fact the animal will not try to drink means he is not thirsty unless he has had too much pain on former attempts. But under such circumstances if one puts water in his mouth and he cannot swallow, there is true reflex block and nerve cell injury, as occurs in Rabies. The rabid animals, after they develop this inability to swallow due to the nerve cell injury, become dehydrated because they can no longer take in fluids. Dehydrated animals that do not drink and are not able to swallow when water is put in the mouth, can be taken as suffering nerve cell paralysis in such situations as we are discussing here. The intoxicated animal will swallow when the water is put into his mouth as veterinarians know how to do to cause it to be swallowed. The rabid animal will not. The water will run out or into his trachea to set up respiratory spasms.

Once the true nerve cell invasion has taken place the changes are progressive and end up with death in typical fashion. The

toxic effects pass off. Swallowing and chewing centers are first to show reflex block and then the spinal centers. Finally the vital respiratory and heart centers are paralysed and death results. The muscle spasms, torticollis, convulsions and paralyses show up in definite order therefore. According to Rivers and other leading authorities, once true nerve cell invasion by the virus has taken place and real paralytic or reflex block symptoms have developed, the subject is doomed; and never has a case been known to ever have recovered. This is also our experience in the present epidemic, except for the cases we have treated.

Experiment

In April 1955 the Fleury live Rabies virus vaccine, made from the street variety of dog rabies, was used for the protection of a large herd of cattle. Of the 650 animals vaccinated at the Fazenda Indiana near Rio de Janeiro, 600 showed no symptoms of intoxication or rabies. In May however typical rabies developed in 23 animals and they followed the typical course and died in four or five days according to the classical symptomatology and course. We arrived on May 19th and treated such animals as had just come down with the disease, except two, Oceana and Tranca, that were under heavy "Hexa-Meth" treatment. We treated them later when in the final hours of agony. It was too late to help them, and they died. All of the other animals that we treated with 6X dilution carbonyl catalyst showed true dehydration and deglutory reflex paralysis at time of treatment.

There were six far advanced or rather terminal cases in our series, and the rest were well established with deglutory paralysis, an inability to chew, in addition to the toxic excitement. Some showed torticollis, and incoordination of movements, and would fall if pushed and then be unable to get up if not picked up, after which they fell again. This phase was noted after the excitement stage passed and was part of the depressed stage even though spastic paralysis and short convulsions were noted. Any disturbance set off a convulsive attack that accentuated the spasticity. The torticollis persisted throughout the final agony in all untreated animals. But in those we gave the

GX carbonyl treatment, it disappeared before the swallowing reflexes were restored. Here we have another confirmation of actual parasitism, for the sequence of recovery events in our experience in such states is a reversal of the order of the symptoms of the pathogenesis. The first to come are the last to go and the last to come are the first to go. Naturally the longer the pathogenic agent acted, the greater the destruction done and the longer it takes to correct it. There is a disadvantage here since the ability to swallow is so badly needed. Generally death took place in the untreated animals in four or five days after the symptoms started. Our animals treated on the fourth or fifth day were quite dehydrated then, and in the four or five days needed for the restoration of swallowing the dehydration was extreme. This with the starvation of vitamins and trace elements as copper and cobalt so much needed for the recovery process gave the animal a bad deal. Moreover, since animals that had fallen to the ground and showed the deglutory paralysis and torticollis were condemned by the Health Officials for sacrifice and autopsy; they did not receive the nursing care that would have been so welcome. Their normal fate was death.

The rest of the cases were in the second or third day of the infection when treated. The symptoms showed well advanced and progressive integrations of rabies virus with the nervous tissue. Yet they could all walk, but not swallow.

One animal, the calf Iberia, was in the earlier phase of deglutory paralysis when treated, but was sufficiently dehydrated to need subcutaneous infusions of physiological salt solution at the time of treatment and a few days afterward. In this case the deglutory muscles were not all paralysed completely therefore when the injection was given. This case represents a very early phase of viral integration with nerve cells. It is very important since it shows that the drinking power was not fully restored before 84 hours after treatment, since infusions of salt solution were still needed a few days to combat the dehydration. As soon as swallowing showed efficiency such infusions were always stopped. Thus the restoration process takes as long in an early phase of advancement as in the later phases. This will appear in the recoveries in the farthest ad-

vanced cases also. In contrast to this case, one calf showed marked toxic symptom but no deglutory paralysis, and recovered spontaneously as all of the merely toxic cases do. In that case there was no true viral integration with nerve cells therefore. In another neighboring fazenda several highly toxic cases were observed. One such case was excited enough to attack. But no reflex block was observed in any. They recovered spontaneously. The veterinary surgeon of the Fazenda Indiana informed me that no animal that showed deglutory difficulty ever recovered except those that we treated. Thus the status of the cases reported here is established as true rabid infections. The control cases that were autopsied showed Negri bodies, and the macerated brains when injected into mice gave true rabies infections. The sections of their brains showed the Negri bodies also, according to the laboratory reports given us.

Fortunately we were able to treat animals in every stage of advancement of the disease, and have controls in each stage, as well as the 23 animals that took sick and died up to the time of our visit. We arranged several types of collateral controls as well; and in this we were aided by circumstances.

The total recoveries of treated animals with definite nerve cell-virus integrations were eleven out of thirteen that received the one to a million dilution of the carbonyls, and could be counted as legitimate material for the test. This gives a close to 85% cure rate. The names of the cured animals are, Iberia, Paina, Idioma, Maravilha, Apa, Oferta, Trama, Docura, Vila, Jupuboa and Tiara. The two cases that died on the same dosage of the same reagent are Oculista and Tella.

This epidemic also gave us an opportunity to confirm and extend previous work with Rabies. Several decades ago one of our colaborators, Dr. Buchen of a small town in Montana was bitten by his horse while rabid from a bite from a rabid coyote. The horse was treated with an injection of a 12X dilution of the serial system of carbonyl groups. The horse recovered, though treated in the terminal phase. After Dr. Buchen developed well defined paralysis and convulsions he received the same dosage and recovered completely, too. Our next experience was in Rio where a few mad dogs were treated with favorable results.

Many questions arose as to how to classify this highly fatal virus with regard to molecular configuration and for ability to make the parasitic azomethine double bond. Thus we desired to compare killer viruses with regard to tissues specificity, and the type of electronic activation the dehydrogenator carbonyl group must receive to be effective, aside from its oxidation-reduction potential.

We set up one experiment with eight animals. They were divided into two groups.

In the first group, we treated three average advanced cases of well established rabies with 7 cc. of a 6X dilution of diphenoquinone. This was done to confirm the lack of effect of the quinone structure in rabies. Sometime ago, we had found that the quinone had no effect in dogs. These three cases died as if not treated at all, just like other controls of a similar classification.

The second group consisted of five animals divided in two sub groups (a) and (b).

Sub-group (a): A dose limit was tried on, two calves down in the terminal stage of rabies. They were gvien 10 cc. of a 15X dilution of the serial system of carbonyl groups. They both died. One died eight minutes after treatment and the other one-half hour later. Thus there was no effect from the high dilution and they can be taken as controls.

Sub-group (b): Two calves of the terminal stage, that appeared about the same as the two that died, were given 10 cc. of a 6X dilution of the serial system of carbonyl groups. They recovered fully, the symptoms leaving in the reverse order to their coming. Swallowing paralysis disappeared last. The last calf in the terminal phase was found on the ground unable to get up or swallow, had torticollis and the spastic effects. It was also blind at the time of treatment, and had been sick for maybe four days. After treatment it slowly improved so it could get up and stagger about and drink well. Thus it was improving with the dislocation of the parasite from the host cells, when it got caught in a barb wire fence and cut its neck very badly. Liberal applications of methylene blue were poured into the wound. Methylene blue happens to be an antidote to our treatment. One day later, the condition retrogressed, and on the tenth day

after treatment the animal died. This case cannot be counted in with the test for efficacy, as the antidote wiped out the progress started by the carbonyl treatment. The free radicals on which the recovery process depends are absorbed by the Methylene Blue. Thus we also have an indication that the recovery process in rabies is of the same character as in other diseases.

Therefore, of the far advanced or terminal cases, five in number, two made full recoveries on the one to a million dilution of the reagent, the serial system of carbonyl groups with free radical terminals. The other three may be counted as controls of different types, two of the ultrahigh dilution type and one of the antidote type. The three that were moderately advanced and received the quinone may be taken as controls too as in this instance that particular type of molecule is without effect. We thus also confirmed our previous experience with quinones in rabies. The serial system of carbonyl groups, 6X dilution, cured two out of three, including the fifth calf which was subsequently treated with Methylene blue; while controls only lived minutes or hours after their placebo treatments, and all five were in the same terminal condition at the time of treatment so far as anyone could tell.

What The Experiment Taught

The recovery course in Rabies comprises two principle periods. One of about seventy two hours in which the patient is apparently stationary, and not passing on further toward death. Most of our animals would have deteriorated rapidly and died before this had they not been treated with an effective reagent. So the stationary phase can be taken as a sign that recovery is going on. This is followed by the second stage of the recovery. The symptoms are aggravated, and one not experienced in the matter would conclude the animals are getting worse. We interpret the stationary phase as that in which the virus is undergoing dehydrogenation, and the short aggravation phase as that wherein the virus is undergoing oxidative fragmentation with energy returning over the azomethine bond or bridge into the reconstructive processes of the host cell—just the reverse of what took place when it passed on to the virus. Reconstruction is thus going on as energy is pouring back into the functional

mechanism so the reflexes concerned are exaggerated and the situation looks worse. One cannot blame the Health officer for wanting the animals to be sacrificed and relieved of their misery. However, this phase is so short that when taking place over night, one simply finds the calf up and about the next day eating and drinking diligently, or slowly recuperating when the nerve cell injury had been very far advanced. After the passage of energy from the combustion of the virus into the host cell functional mechanism is finished, the azomethine bridge is cut leaving a carbonyl group in the host cell functional mechanism with which it conducts its functions as before the infection took place and the virus has lost its amine group as illustrated by Diagram VII, according to our analysis. The disease and its cause are removed therefore. The cure is complete.

This experiment showed also that the quinone type of structure does not serve in Rabies. In the three cases treated with quinone, the dipheno-quinone was used in the one to a million dilution. Perhaps some other dilution may have given results, but we have no proofs as yet. The effect of dilution in creating strain in the more mobile bonds deserves consideration. However, the fact that the carbonyl group of dipheno-quinone with its high oxidation reduction potential is not effective as a dehydrogenator in Rabies begs for explanation. Our simplest explanation, though others may be better, is that the carbonyl reagent must integrate with the host cell energy producing mechanism, as the virus has already done, that all three occupy the same field of action within the mitochondrial particle. Further, the configuration of the virus structure will necessarily change the pattern of the host cell configuration. The change thus set up will facilitate the admission of one reagent or other preferentially, especially in the plane that permits the carbonyl group to attack the virus perpendicularly to the plane of the double bond and the carbon atom conjugated alpha thereto that carries the hydrogen atom that must be removed to start the oxidation chain.

Further, for union with the host cell functional mechanism, the reagent must present a free radical to add to one of its double bonds or free radicals. Long chains of carbonyl groups with

free radical terminals fit the requirements for oxidation of the integrated Rabies virus. The quinone structure serves well in Swine Pest, one of its more polarized double bonds offering the means of ligation with the host cell functional mechanism, as well as the offer of a free radical while expressing its rich resonance. Here the dipheno-quinone is structurally more proficient, and hence here too it serves best therapeutically. One may therefore classify virus diseases with reference to their response to carbonyl groups activated by electrons received from ethylene linkages, or from serial systems of carbonyl groups, and as these are possessed by different molecules that offer host cell ligation facilities in various manners.

The same factors no doubt control the specificities of the same reagents in the various types of cancer. For example, we have found the quinone structures "specifically" curative in primary diffuse adenocarcinoma of the liver, and in cancer of the pancreas, that widely invades the stomach, the liver, and the lymphatic system. On the other hand the quinone structure does not show a good record in skin and brain or visceral cancer in general. The serial systems of carbonyl groups were found more satisfactory here. It must be recalled too that the separated ring structures serve as carcinogens preeminently in liver cancer, as acetyl aminofluorene, and the methyl-amino-azobenzenes. So the liver seems to admit the separated ring structures into integration more readily than other tissues do. This seems true for the curative quinone rings too. The steric facilities need much more investigation in neoplastic and viral diseases, one may see. Nothing has been done on such matters as yet, except for our own efforts.

Complicating Factors

There were a few complicating factors that adversely influenced the recovery percentage and these will have to be considered.

(a) **Nutrition**—Aside from the first animal treated in the earliest stage where swallowing paralysis was only starting, and which was restored to normal in three days, the rest could not swallow or masticate and so they rapidly dehydrated, and the malnutrition with its absence of trace elements as copper co-

balt and molybdenum that are absent from the soil hindered the recovery process. The cold rains with the animals lying on the cold wet ground, one of them in a deep ditch for days nearly drowning and unable to make a controlled movement, added to the hindrance to recovery. No one could suggest that the good nursing cured them, even though the veterinary of the fazenda did all that kindness could accomplish.

(b) **Short needles**—Not knowing that the Zebu has a thick subcutaneous tissue that droops and the skin itself is not too elastic, half the needles we used were No. 16 bore and ¾ or one inch long. The other half were two inches long. The short needles evidently did not deliver the reagent into the muscle in all cases and these when injected with a second dose using the longer needle responded well. We may have lost one of our cases by use of the short needle as one died quickly as if not treated.

(c) **Confusion**—The veterinarian who served as technician in preparing the vaccine was not a specialized virologist, but was no doubt expert in the routine of producing the vaccine. He was also placed in charge of the epidemic as the Health Officer for Rio. This was unfortunate as he took the affair too much to heart as if he were responsible for the deaths of the animals instead of chalking it up to the whims of the virus as any veteran seasoned virologist would do. He understood too that legal procedures would be started to recover damages for the loss, and took to the defense of his vaccine by claiming that it contained no rabies virus, although autopsies of the animals that died, including my seven control cases all showed the Negri bodies, and their brain macerates injected into mice produced rabies in all the mice, and the brain sections of the mice all demonstrated the Negri bodies. This work was done by the pathologists of the Rural University, the government Agriculture College. Our effort to explain the recovery reactions to the health officer fell on deaf ears, and as he had the right to condemn any animals he wished for sacrifice and autopsy, this was a little dangerous as the following experience shows. It may have reduced our cure percentage some.

The incident was the following, and depended on the fact that Rabies is accepted as 100% fatal. So when the animals

reach the terminal agony they are sacrificed to shorten their misery and to make better biopsy facilities available. This was practiced on the first group as they reached the terminal phase after the first number had died in typical rabies fashion and established the course. It was used on some of our earliest cases that did not recover right away. As the terminal cases take longer to recover, those treated when they were found in the terminal phase after being condemned for sacrifice by the Health Officer, naturally must require at least four days to undo the damage done by the virus. This seemed too long for one who did not understand the therapy. So one such calf that had been condemned for slaughter by the Health Officer, and was treated when it was lying on the ground and paralysed, unable to chew or swallow, and unable to make coordinated voluntary movements, with torticollis, and in about the same condition as the two that died within an hour after being treated, was still lying on the ground 84 hours after treatment, and apparently not making any headway so the Health officer had it dragged to the truck to be taken away for sacrifice and autopsy. But as it was in a position to be lifted into the truck, it kicked up a fuss and tried to get away. The veterinarian of the Fazenda happened by, observed good coordination in its movements, and made other tests for recovery. He found it was able to swallow and chew, so he led it back to the pasture and let it live. It quickly ate its way into good health, and is cured. Thereafter the rest of the treated animals were allowed to make recoveries without interference, or as in the case of the wounded cow to die. Autopsies and Inoculation tests proved the disease to be rabies. Negri bodies were demonstrated in the inoculated mice.

Here are two 100% fatal virus diseases, Rabies that is neurotropic, and Swine Pest that is viscerotropic, that kill regularly in 4 or 5 days, which we have found from 60 to 100% curable even in the terminal stages. In this far advanced state, the virus is not only integrated with the host cell, but has built up its pro-progeny at the expense of the host cell, which now is far along in its destructive dissolution, when the oxidation catalyst is administered. Since the host cell is undergoing full reconstruction while the virus is undergoing oxidative dissolu-

tion and perhaps depolymerization concomitantly, the pathological process is fully reversed, and the energy exchanges are specific between the two parties. The reciprocity is specific, with the treatment reagent entering the process as the specific reversing factor. The relationships are therefore most fundamental, and hence must also be simple, as we have presented them. Such virus parasitism is thus curable on a logical basis, when it was never curable before.

The basic chemistry of the vital unit mentioned earlier is supportive. Thus the phenomena of the double bond will depend upon the nature of its substituents. With an amine group as a terminal substituent the electron drift over the ethylene linkage is away from the amine group and toward the other end where we assume the virus resides. Virus support is thus favored. If a carbonyl group is the substituent, on the other hand, the electrons tend to concentrate at the carbonyl group, making of it an active dehydrogenator, or oxidation initiator. The parasite is devoid thereof, while the host cell requires it in its energy production. The condensation of the parasite's amine group with the host cell's carbonyl group to form the azomethine double bond, will permit energy to pass either way with equal facility, depending on the position of the energy source and the position of its utilization. Thus when the virus progeny is being built up at the expense of the host cell the energy passes to the parasite, but when the parasite is undergoing oxidative destruction, energy returns to the host cell where it provides for the latter's reconstruction. The azomethine bridge is thus neutral and serves not only the energy transfers, but also the disligation of the virus with destruction of its amine group, while the host cell carbonyl group is being restored. Provisions for rapid recovery thus lay in the basic chemistry of the vital unit as we picture it.

AFTOSA

(Hoof and Mouth Disease)

This disease occurs in such great variety of virulence that only by treating many herds under different circumstances can efficiency be tested. But a carefully controlled experiment as

summarized below, using a known virus, bred to the peak of deadliness of the cardiotropic variety will show how the virus can be separated from the cell even weeks after it has produced a deadly myocarditis.

This same deadly type of virus, occurring naturally, was encountered by us several times in epimedics that threatened to wipe out herds of very costly cattle. On October 14, 1949, at the Instituto Quinze de Novembro, a veterinarian college with 59 head of cattle and 200 pigs, the professor in charge was afraid that his herd would be wiped out by this virus. When we arrived, five animals had already died. This was a few hours after the epidemic had started. Others were lying on the ground, unable to get up and soon would be dead. We treated the 54 animals that were still alive. Of these, 7 were newborn, 15 were calves, 17 were young bulls and 15 were cows. Two-thirds were very favorable hosts for the most destructive action of the virus.

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

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

Results—4 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 struck the institute again and ruined the new cows only, that were brought in the herd. The animals we had treated the year before did not develop the disease except a few cases that took it mildly and soon cleared up. So the protection was practically 100% and has so remained for the several years that followed.

In another observation with a milder infection that always killed some members of the herd, but not over ten percent, was at the Rural University, the government Agriculture school, in June 1951, and reported in Veterinaria, no. 1, p. 75, 1951, by Dr. Adalberto da Silva Carneiro, the virus type was identified as "C" with 68 cows in the herd and several not showing symptoms. All recovered as inspection showed four days later and

the lactation which was suppressed had returned to full amount within 24 hours, following one dose of the remedy. But one calf required two doses. No new infections developed and none died. This is the usual type of virus encountered and the results are the same in all other experiments.

The controlled experiment was done on thirty young bull calves, all the same weight and age. Ten were inoculated with the virus and held for controls; ten were given preliminary "protective" vaccine treatment before the virus was inoculated, and ten were treated with the carbonyl compounds three days after the virus inoculation. The virus used was one that regularly killed 80% of animals by heart damage in from 4 to 9 days. The rest that did not die right away, did so sometime later of chronic myocarditis. The experts at the Institution who prepared the virus and the vaccine they made from it, knew their virus to be a standard product and what it universally did in the dose used. We treated the first ten with a dilution of one to a trillion to establish a dosage of the highest limit of dilution; and had one death the fourth day with nine cures. The vaccine protected animals showed four deaths the same day as one treated calf died. The rest were exceedingly sick with myocarditis and the controls were judged to die quickly at this time, and as there were no facilities for cremating so many animals at one time we were asked by the director to treat the controls as they would die otherwise. We gave a dilution of one to a million this time and the curative response in all was immediate and complete so that here, although the myocarditis was advanced to a near fatality point the disligation of the virus was fully established and the heart muscle restored to normal action in a matter of hours or days. Later one vaccinated animal that survived was very ill with myocarditis and had an enormous aftosa abscess that covered his right side. Such animals always die, but we treated this one and obtained a cure. The results,—90% on the 12x dilution, and 100% on the 6x dilution were cured using the serial systems of carbonyl groups.

Many small and large herds were treated with animals that could no longer rise from the ground all of which recovered, if the animals were left in the field. **However, where ammonia**

saturated the air that was breathed in the barn yard or stable the results were disappointing. The same holds true for any infection including brucellosis in the animals we have observed. **Cresols also interfere.**

CINOMOSE

The first official test done in the army Hospital for Small Animals, at Rio de Janeiro yielded a cure percentage of the animals seen through to the end of the experiment, of 80% including the first 17 which died while trying to arrive at a proper dosage, (Carneiro, and da Souza, Veterinaria, Ano IV, Num. I, p. 21, 1950). Thus with these trial animals left out, the cure percentage was 95% at least when the correct dosage was used. And this has been substantiated by further observations by a number of veterinarians in private practice. It includes the serious nervous system infections which otherwise are always fatal. Closely observed cases of this class show a cure percentage of 80 in private practice even when paralysis was established for weeks and was widespread. Altogether several hundred dogs have been treated. The separation of the integrated virus is thus established in other deadly virus infections besides Infantile Paralysis. Such a feat has never been accomplished to date by any other therapy. While the serial systems of carbonyl groups as used in the trials just described have yielded the best results in Cinomose, diphenoquinone has done well consistently here, but the recovery course is much slower and does not complete itself at times unless a dose of the former reagent is used. This is especially true in the paralysed cases. But in Swine Pest it has served well right along, and benzoquinone has served well here too. This would indicate that in Swine Pest there is much more toxin circulating not combined with the tissue cells than in cinomose, and hence the facilities for adding toxic free radicals offered by the many double bonds gives the advantage. Our first discouragement with diphenoquinone came with repeating the dose where a block to the recovery was had instead of the boost that was desired. We learned thereby that the second dose must be very diluted if given at all.

The separation of the virus from nerve cells, and its

separation from heart muscle cells to which it had acquired a fatal affinity in aftosa, places the value of the oxidation mechanism in curative as well as protective therapy as very high. We can not escape the fact that living processes in all aerobic life depend upon the chemistry of oxygen in its molecular form. The secretion of milk too, by animals as they recover from aftosa after this treatment is completely back to normal and takes place immediately, that is within twenty-four hours, or sooner. This is established in many herds about Rio. (Carneiro da Silva, Veterinaria, Ano V, No. I, p. 76, 1951). The return of function is thus prompt with separation of the virus whereas without the carbonyl catalyst treatment it scarcely ever returns and if it does, it is only slight. The manner of union of virus and host cell substance in any instance then seems to be in conformity with that shown in our diagrams, and takes place with the energy producing mechanism. Since the virus on separation is no longer infectious, its chemistry may well be changed as we suggested by acquisition of a carbonyl group which indeed may serve it in initiating oxidation chains for its own energy production and function so that it need not and can not again become parasitic. The same conclusions seem justified from the results of the cure of mastitis in animals where the most virulent streptococci and staphylococci may increase in number during the healing of gangrenous areas, and while the toxic state of the animal is rapidly disappearing. Here the increase appears to follow the amount of tissue debris that must be removed.

The recovery percentages in animals suffers the same handicaps as in humans except that animals are under more thorough dietary control and can be held on a vegetarian diet. We find some dogs after taking vegetables only and some milk for a few weeks, refuse to eat meat any more, and they get along very well indeed. Habitual use of narcotics and depressants, or "calming" drugs are eliminated and so when the veterinary has not overdone with sulpha drugs, ACTH and other modern remedies, the recovery rates should be and are better than in humans where they will cheat on the diet, or on smoking, and call for aspirin and barbiturates in all the different

forms that are offered. Besides, the animal has not been ruining his power to carry oxygen by the blood, or the ability of the tissues to use oxygen, by the use of such medications.

SWINE PEST

In the treatment of Swine Pest (Hog Cholera) the same difficulties were met. Cresol antiseptics and other affairs have interfered as much as 100%, as in two experiments in the pens of the Ministry of Agriculture Maracana station. But in the Ministry's outer stations as at Deodora, and in private farms where such interferences were avoided and only lime was used to clean the pens, the results have run from 60% to 100% cures repeatedly even in the most severe epidemics. Usually one dose proved sufficient but a second dose hastened recovery so that it took three to five days to obtain true and lasting cures. Many of the cured animals could not rise from the ground or eat or drink when treated. That is, they were in the terminal stage when they received the first dose. A second dose given 84 hours later met them on their feet able to take nutrition, but still showing some cramps and traces of hemorrhages into the abdomenal skin. The second dose boosted the recovery rate very remarkably, and should always be employed in cases of this type. The second dose (12x) is always more dilute than the first, (6x) on the decimal homeopathic scale. Most of the herds were small. Since the virus is exceedingly important and *vicious*, a complete discussion is planned for the complete text.

Our observation in animal diseases convince us also that all pathology follows the same pattern. (See Chapter VII). In Swine Pest all tissues of the body are attacked with rapid fatality, an apparently lytic type of virus-host cell integration, but only the pig is attacked. In cinomose the virus may be specific for either the respiratory, the intestinal or the nervous systems, or for all three, and only a few species are attacked, as the cat and dog. In Poliomyelitis, many species are attacked, but only certain cells of the nervous system. In both cinomose and Poliomyelitis both the lytic and symbiotic types of integration are usual, and this is true for aftosa which while usually

symbiotic, may be violently lytic to the myocardium before hoof and mouth symptoms can develop.

Since in these and all other virus infections we have encountered, the survival factor can be replaced by a synthetic atomic arrangement, the carbonyl group, one feels justified in concluding that the survival factor is the same mechanism for all tissues. Specificity for species or tissue is an entirely different matter. The antagonists to the operation of the survival factor have no influence on specificity in any, while they block survival factor activity in all. They are carbonyl inactivators and double bond additives. Specificity of the virus depends upon its protein structure, while specificity of the reagent depend upon steric advantages or hindrances with reference to its union with the host functional mechanism in the plane that permits dehydrogenation of the virus.

Thus in swine pest the serial system of carbonyl groups proved inactive entirely, and the quinone type from 90% to 100% active (diphenoquinone), or 60% active (benzoquinone) in various epidemics. Though the disease is always fatal in 4 or 5 days, in no two epidemics were conditions exactly alike. Tabulation of the various experiments would therefore not mean much. The time of instituting the treatment with regard to the progress of the epidemic, whether early or late, is of no importance. Thus it might be argued that though the infection is one hundred percent fatal in 4 or 5 days, those that died first were less resistant, and those showing symptoms last are the most resistent and would give the 100% cure rate more consistently. This is not our experience, for we have treated the pig that survived in a herd the day after seventeen had just died. Though this pig was the last to come down with the infection, the disease developed at the same rapid rate as in the others, and one injection brought full recovery in the same time as other pigs treated in neighboring farms the same day, when the epidemics were just starting in these farms. One such farm had four pigs in about the third day of infection, and were all equally advanced. One was a very small pig (a runt) that was nearly dead, it had not eaten or taken water all this time and died in a few hours after receiving the injection. The

others all recovered. Another neighboring farm had four with the infection, three in the second day perhaps. We treated the three that appeard worse, and left the other for the control. On returning three days later the treated pigs were about cured and eating well and without fever. The control pig was quite advanced and would probably live another day. We treated it and two other neighbors' pigs that were the first to come down with the disease in their herds. All recovered in due time.

In this group there were nine pigs including the pig that was treated after its seventeen mates had died and it was far advanced in the disease. Of these eight recovered, giving an 88% cure rate. This happened at Santa Cruz in the first week of July 1956. A year earlier we treated five pigs with advanced Swine-Pest in Rio. They were part of a herd of ten, five of which died the day before. The diagnosis here as in the other epidemic mentioned was made by the characteristic symptoms, clinical courses and autopsy findings. Only one of these five could walk. The others were down in the last stage. All were given the dipheno-quinone. Two injections were given; the first injection was a 6x dilution, and the second injection which was given 84 hours later was a 12x dilution of dipheno-quinone. All five of the pigs recovered, thus giving us a recovery rate of 100% in this group.

The age of the animal makes no difference, but cleanliness does. The diagnoses were made by the veterinary surgeons of the Department of Agriculture or of the city veterinary services where the animals were treated. No case was treated without all the cardinal signs and symptoms being present, such as the scleral and cutaneous hemorrhages, the pneumonia, the intestinal changes, the very high fever, or the specific test, and autopsy findings of deaths close by and at the same time. We always arrived late enough for all the data required for a firm diagnosis, after the animals had refused food or drink for several days. The recovery course comprised a period of 72 hours where improvement is only slight, but drink and food is again taken. The pneumonia is still present and the cutaneous hemorrhages are improved some. Then twelve hours of aggravation, is followed by rapid improvement. This is the same course seen in

Rabies recoveries, and its interpretation is given in that section. The cure takes about five days.

THE FATE OF THE VIRUS

The fate of the virus has not been fully established. Clinical evidence has indicated three possibilities. The first possibility is that a part of the virus ligated with the host cell may be burned off leaving a considerable portion still occupying the functional carbonyl group. To compensate for this the host cell constructs another functional carbonyl group. This is illustrated by the type III integration we have suggested, which so changes the steric setup of the host cell that another virus can not ligate with it. A second possibility is the complete oxidation of the virus with restoration of the host cell. This would be an involution process that returned the energy, taken from the host cell during the virus vegetation, back to the host cell and would be used in the latters reconstruction. A restoration of the host functional carbonyl group would be accomplished and the virus or rather provirus would be destroyed. The host immunity would possibly envolve a change in steric setup as the reconstruction came about in a different direction than the usual. The third possibility is the oxidation of the virus off at or adjacent to the azomethine bond. As the virus is separated from the host cell or burned off all at once, the virus would be left with a carbonyl terminal instead of its amine group, and thus it could make no more ligations with the host cell. It is possible that the virus could now conduct its own oxidations for its survival. The host cell is left with its functional carbonyl group restored and is no worse for the experience. Immunity is gained probably through steric change, though other probabilities exist for this also. After treatment oxygen use is increased and area production is diminished in neoplasia. This confirms the azomethine ligation with the pathogen.

CHAPTER XII

INCOMPLETE COMBUSTION BURNS

Products of incomplete combustion caused by suddenly elevated temperatures, from steam, or fire or hot metals demonstrate much the same tissue changes as do the incompletely combusted products of tissue and of germ metabolisms. They offer the toxicities that prove fatal, through gelling of the blood and tissue colloids as well as by direct embolism. This gelling of the blood that prevents its flow through the smallest vessels to cause infarctions, resembles also the situation in coronary occlusion and in cerebral apoplexy. When the oxidation catalysts are used before the vascular wall is completely starved of oxygen and fragments to permit hemorrhage, the dispersion of the colloids is restored and the blood flows on to supply the oxygen and other things the tissues need so that apoplexy and occlusions are prevented. And even after such changes have taken place the treatment has restored the non-toxic normally dispersed state of the colloids so that lesions that exist do not get worse and besides Nature has every opportunity to correct the damage done. Some severe cases of established apoplexy have been restored to a close normal in this way instead of suffering profound invalidism moving on toward death through repetition of the pathology. We submit two cases of burns to illustrate that the incompletely combusted products can be burned out of the way so the usual destructive changes are avoided.

C. B., Nov. 7, 1929, while repairing an automobile was severely burned by a gasoline flame that caused extreme erythema, superficial desquamation and terrific pain, but no immediate visible charring of the tissues. An injection of the serial system of carbonyl groups with application of a high dilution over the burned surface brought immediate relief and rapid healing without tissue destruction or scar.

Mrs. L. T., Sept. 16, 1948, opened a pressure cooker under pressure. The hot soup struck her abdomen covered by a thin cotton cloth only, and caused severe pain and shock. Benzo-

quinone 6x solution was applied immediately and a cubic centimeter of the solution given intramuscularly. She went on with her work in ten minutes forgetting the incident apparently. No blisters formed and the redness soon disappeared. Other similar untreated instances have resulted in prolonged suffering and tissue injury.

It is thus evident that the completion of the combustion of the partly burned products removed the pathogenic activity.

Incompletely combusted germ and tissue cell metabolites, and synthetic carcinogens, unless adequately oxidized in the body, are disposed of by entering into the fibrosis they induce. To the extent that fibrogenesis does not dispose of them, they may induce neoplasia and enter into the substance of the tumor cells where they transfer energy to the mitotic mechanism. Potent, induced oxidation disposes of them and their evil effects, whether fibrogenesis or neoplasia is the main activity. Although the ability to respond with fibroblastic protection is worn out early in some persons, or may have never even started as in infancy, Miss J. McW.; it may be phenomenal as in the case of Mr. P; and it may dispose some organ to a cancer favoring hypoxia. These phenomena may offer the basis for a fairly practical pathological classification.

PRACTICAL SECTION

CHAPTER XIII

CASE MANAGEMENT

REPETITION OF THE DOSE

This is a subject that requires detailed study and cannot be treated fully in an introductory treatise. We will present a few principles that determine the repetition, and the details will be demonstrated in case studies in the complete text.

The purpose of repeating the dose is to restore a recovery process that has been wiped out fully, that is, the reaction chain that was started by the curative reagent and was broken. The conversion of toxin to antitoxic peroxide free radicals was stopped. Thus the recovery process was halted at its very basis. One should find out how such a situation has arisen, since if the recovery reactions were started so as to have progressed far enough to be observed, they should continue unless a definite inhibitory agent entered the system. There are several means of stopping the process, and we will return to them in a minute. However, after the dose was given, it could be that no recovery reaction ever started, and for that reason the dose must be repeated. Interfering factors that could prevent the recovery from getting started could also prevent the recovery from progressing if they came along after recovery had started. They may be considered together therefore. In either event at least one of several interferences should be sought, though all may exist in the same person. The most common are the following.

Highly polar double bonds, as exist in some **terpenes, perfumes, fruit flavors, lemons and orange skin oils, mangoes** and **even tomatoes,** may be at fault. One of the most powerful "innocent" substances that is supposed to be "good medicine" are the **carotenes.** These in cancer cases may prove serious since the cancer patient is not always able to oxidize carotene to vitamin A, and as carotene it is useless. Its rich content

of double bonds even though recipient of electrons from their methyl substituents still show a polarity that can attract the free radicals of oxidation chains in process of formation Clinically we have found that too much carotene in the vitamin mixtures have interfered with the recovery mechanism. The **terpenes of paints, floor waxes,** and **similar affairs are even more serious hindrances to the recovery process. Perfumes** have in certain cases been tagged as the obstacle and therefore **soaps that are highly scented** should be avoided as well as scented powders and the like. **Many medicines and Kerosene oil** as well as **gasoline** are carriers of unsaturated oils that **interfere seriously with the conduct of the free radicals of the** recovery oxidation chains. The physician must force the patient to analyse the situation to avoid interference from the ordinary household affairs. Triple bonds as of **acetylene and garlic are** to be avoided.

Free radicals as of the common anaesthetics are serious obstacles which may reverse the recovery process even after it is under way with splendid progress. **Chloroform** offers a free radical on the same basis as **carbon tetrachloride,** and of all anaesthetics **nitrous oxide is the worst.** All oxides of nitrogen carry permanent free radicals that are ever ready to add to those that carry the oxidation chains, They thus are wide open to swallow up the mediators of recovery. **If an anaesthetic** is to be used **it should not be nitrous oxide,** but rather a local use of cocaine or one of its derivatives should be tried. It is best to look forward before treatment is instituted and have all dental or surgical work attended to first. On the same basis after an anesthetic has been used the treatment should be postponed a week or so to allow the evil products to be eliminated, when it is possible. But in emergency cases the treatment may be given and repeated in three and a half days once or twice to make sure the recovery process has really started. In greater emergency one may repeat every twenty-four hours or thirty-six hours until the recovery process shows it has started by either a positive or negative phase of a reaction.

Steric hindrance resulting from effects of amine medication must also be considered. Clinical experience has convinced

us that when the host cell's energy producing mechanism is integrated with a virus or toxin pathogen, its molecular configuration is so altered that it will accept one or other type of the carbonyl reagents as a free radical or double bond addition. However to be therapeutically efficient, the integration with the remedy must allow the *steric advantage* necessary to the dehydrogenation of the pathogen's carbon atom located alpha to the double bond formed by the condensation of the pathogen's amine group with the host cell carbonyl group that initiates the functional oxidation chains. The remedy must attack in a plane perpendicular to the coplanar arrangement of the system just mentioned.

However when this host cell pathogen integrate is exposed to an antibiotic carrying activated amine groups as in streptomycin, condensation with the latter may also take place and hold rather permanently to change the molecular configuration of the functional mechanism in a way that offers steric hindrance so that the remedy carbonyl group is not able to attack in the preferential plane.

Therefore before giving the carbonyl catalysts one must allow some time for the elimination of the interfering agent and a recovery from the distortion they cause. If an emergency exists, the dose may have to be repeated every 72 or 84 hours until an effect is had.

Remedies of similar structure to the carbonyl catalysts can also interpose steric hindrance and hence mixtures of the remedies are not desired for clinical use.

The treatment itself may be the most effective inhibitant, when used in too heavy a dose or when repeated too often. Both the double bonds of benzoquinone or of diphenoquinone, and the free radicals of the polymerizing chains of carbonyl groups with free radical terminals interfere as described above. Dose repetition is therefore as serious as the excessive amount of reagent in the first dose. The best dosage is that which presents the carbonyl groups in such dilution that in the tissues each is surrounded by a blanket of toxin molecules, and the conversion can go from one to another until all are converted to their antitoxic structure and serve as centers for new oxidation chain initiation.

The process will then go to completion. The highly electrophylic nature of the double bonds of the two quinones and of orthoquinone too, but especially of diphenoquinone make the dosage a matter for expert decision, and this is especially true of the repetition. **When a recovery process is once started and going well,** the dose should **never** be repeated, no matter how good the patient wants to feel. One goes by the fact that the recovery is in progress and by the evidence given by the **crenation test** of the red blood cells. This will be discussed later. When the patient has been properly prepared for treatment, and the avoidance of gas engine exhausts, and other interferences has been followed, the dose does not need repetition as a rule. So the diligence should not be toward finding a time to repeat the dose, but to eliminate factors that call for it.

Direct and indirect interference with oxygen supply and usage is a fairly complicated matter, and comes under two heads.

Interference with oxygen transport, is caused by the usual pain-killers of our day with salicylate origin, as aspirin, sodium salicylate etc. The barbituric acid preparations may even be worse. They cause, like other coal tar products, a change in the hemoglobin so it does not carry oxygen any more and a state of more or less asphyxia is set up. The free radicals of toxins and virus that have been opened up by the treatment have no oxygen to add to so they add to whatever else they can find, host cell, the reagent, or even to themselves and in a sense polymerize to form spores possibly. The fate of an aspirin addict is the worst and requires the greater care. The methemoglobins formed by these poisons may allow the blood to look rich, but they are of no use to the patient and block the production of serviceable hemoglobin besides. All other materials of similar action are to be avoided too, hemolytic materials included. The advanced case of cancer after prolonged use of coal tar products probably has a fairly complete exhaustion of the blood forming organs, and transfusions do no good either as experience has shown us, but may make the situation worse by overwhelming the reticulo-endothelial system, so that the effete and worthless blood cells are not removed from the circu-

lation and converted into blood forming materials any more. In cancer the reticulo-endothelial system is already exhausted before the neoplastic process is very far advanced, and throwing a load of foreign red cells at it to break down, is adding more work than it can carry and making the situation worse. At times one transfusion may get by with benefit, but as a rule not.

Interference with use of oxygen is offered by the toxic amines of intestinal origin or in some medicines. A meal of animal protein taken by a patient with an intestinal flora rich in amino-acid decarboxylases, or even the taking of avocadoes raw when the digestive juices are weak, may set up the production of enough toxic amines to inactivate the tissue functional carbonyl groups and also the carbonyl groups of the treatment reagent, and thus block recovery. One therefore inquires into the diet when a recovery process is blocked. Such amines also tend to gellate the tissue colloids so oxygen transport to and within the cell is blocked. Then the oxidases that take care of the hydrogen atom the reagent carbonyl group removed have no oxygen to work with, and the recovery process is broken. MacDonagh of London has identified several substances that cause this colloidal gellation in the blood and the same action holds for the intracellular proteins, as well. His work requires study and gratitude. We hold that the fungi that infect cancer tissue accomplish the same gellation changes. Amidines are the worst of all.

Metals like arsenic act like the toxic amines and block the phosphorylations we described previously. All such "uncouplers" are to be watched for and avoided. Many a fine recovery process has been cut short by the use of arsenic to speed the blood production. In our cases it never works, but has always interfered, even in highest homeopathic potencies. The action is not a simple uncoupling affair therefore. Gold and Lead used in the past caused hemorrhagic degeneration of the liver and kidneys, and did no good. Still one must be on the lookout for their use even today, and to avoid them.

The diet is discussed soon, and articles that carry the violations mentioned above can be identified and eliminated.

The identification of an interfering agent may be just provocation for the repetition of the dose, yet one must distinguish

between a true block to recovery with cessation of its advance or even its reversal, and a negative phase of the recovery reactions. This is not always easy. Besides even the latter situation may call for a repetition of the dose.

Prolonged negative phases with aggravations of the symtoms, when the crenation test of the blood shows no progress may call for the repetition of the dose, and so the crenation test is important.

The Crenation test should rule the decision. If it shows improvement the dose should not be repeated, no matter what the patient desires, or what the symptoms may indicate, provided the test is made correctly. It is performed like making a blood count; a properly exposed drop of blood is taken into the pipette and quickly **a one percent solution of sodium chloride** is drawn up with it into the mixing chamber and mixed. It is placed on the slide and the number of crenating cells and the non-crenating cells are approximated. I say approximated, since one must not spend too much time recounting as the task should be accomplished in from one half to one minute, to get a true picture. Therefore everything should be in order for performing the test. Alcohol used to clean the finger must be fully removed before making the puncture. Clean glass and pipette are essential and the sodium chloride solution must be accurately made up. In order to make up this one percent salt solution properly, the salt should be dried first before weighing. The test should be made before the treatment is given and then every week or two in order to obtain a true picture of the changes. It is important to remember that the patient's bowels should be well washed out with a mild sodium bicarbonate enema and the diet be followed carefully for a few days before making the initial test.

The change that constitutes crenation is a shriveling due to the withdrawal of water from the inside of the red cell to the slightly hypertonic salt solution on the outside. It should take place immediately. Some cells will not crenate but appear the same while others swell up. The latter are effete, and should have been removed. Those that stay the same are intermediate in their status. All should be approximated or

counted and recorded quickly. The water is drawn into the cells that swell as the number of molecules within has increased as the cell undergoes protein lysis and death. All should crenate in healthy blood and so the salt solution should be tested on a few healthy persons first. As the patient improves the number of cells that crenate will increase and the number that swell will diminish, until normal is reached. Under such circumstances the dose is not repeated. But when the symptoms call for repetition and the crenation test confirms it, it should be done.

The choice of the repeated dose. As a rule it should not be a quinone type compound after a serial system of carbonyl groups is used, unless the quinone has a higher oxidation reduction potential than the dose that was given. This holds for the repetition of the serial systems of carbonyl groups, the repeated dose when given must show higher oxidation reduction potential value. These matters are explained in the completed text in detail so the correct molecule can be chosen for progress instead of reversal of the recovery procedure.

The time of the repeated dose is also important. It should never be given during a period of improvement, but during an aggravation phase of a reaction when the adverse symptoms are on the increase, or when the disease is advancing. The cycles should have been watched and the time for an aggravation to show could then be predicted. This is the time for the repetition, when the progress is more or less stationary but the crenation test shows a failure to advance, or a reversal. Ordinarily, the 24th day after the first dose is a good time to repeat, if it has not been done three and a half days after the first dose. Otherwise the last days of the ninth or twelfth week or beginning of the tenth or thirteenth week, the end of the twenty-fourth or thirty-sixth week or even of the sixtieth or seventy-second week or at some other multiple of twelve. When reactions run on over 100 weeks a repetition may be in order and be followed by most satisfactory results. The patient may have been cured so far as one can tell from a very virulent lymphosarcoma, but may exhibit slight febrile reactions at three week intervals. These promptly have ceased after the dose was repeated, even as late as 108 weeks following

the original treatment. The health quickly boomed also thereafter. However, it should be remembered that the recovery process is a chain affair, that is theoretically at least, and in many practical instances, has featured health improvements for decades after the dose was given. These improvements include better circulation of the blood, better liver and bowel function and even brain and eye function. Many such patients who have been getting stronger glasses each year now reverse the change and require weaker glasses instead. Some have gone back into business and made a fine success at it, where formerly they had failed, and were rated as having a nervous breakdown. Other functions have improved in the same manner.

MEDICATION AND FOOD POISONS

Our reagents do not contain the phenolic group, and indeed may be hindered in their action by that group. Therefore all medication containing it is eliminated from the regime. This includes the common analgesics and narcotics. They produce effects contrary to those for which we labor. As a rule however the severe pain of the cancer patient as he comes in from the ordinary hospital care, can be controlled by good intestinal lavage, and the use of proper diet, and certain homeopathic remedies, until the treatment has been given, and then the pain soon ceases as a rule. This applies to cases taking heavy doses of morphia and other narcotics where irradiation was not applied. In the latter cases a special situation arises that needs careful discussion. The salicylates and barbiturates are forbidden, and morphine if actually needed is used in decreasing amounts until it can be given up entirely.

Medication is used as a physiological complement, such as the gastric or pancreatic juices and a small amount of dilute hydrochloric acid when required. Soda or chalk may be required to keep the intestinal tract alkaline and is given one or several hours after the meal. For catharsis as may be required at times Rochelle salts, magnesia, appear least harmful, but the daily use of plenty of molasses of the cruder varieties, is the best procedure. It can be taken in warm water to substitute for coffee. One soon develops a taste for it and

its benefits should be sought. The enema with dilute soda solution, a teaspoonful to a quart of water may be the daily need. Cod liver oil may be the best regulator of all.

The vitamins, except too much carotene and vitamin E, are required, and of these Thiamine, Riboflavine, and vitamin A from fish liver oil must not be neglected. Likewise the trace elements and some potassium are required. Copper and cobalt should be emphasized.

Pure water and fresh air are essential—free from all contaminants.

The diet is vegetarian entirely, with fruit and freshly ground cereals. Rye is most important and can be cooked into a porridge or baked into bread, pies or cakes, avoiding the use of eggs or milk. Fresh pure butter and honey and pure rich cream are the only animal foods allowed.

Greases are not used and foods are not fried or baked in them. Thus the acrylic aldehydes are avoided. Nothing is over heated or burned and the tars are thus avoided too. Fats are used if unexposed to air, and olive oil should be used exclusively if possible, as its unsaturated fatty acids are protective somewhat against cancer. Corn oil is not used as it tends to stimulate cancer, possibly due to its selenium contaminant, taken from the soil.

Corn, peas and lentiles seem to pick selenium out of the soil and the risk in using them must not be taken, unless one has a garden of known soil purity. This applies to arsenic also for many an old orchard is so badly saturated with arsenic that the fruit is actually toxic.

The sugars should be as natural as possible, honey, brown sugar, and molasses are preferred. White sugar may not be harmful in itself, but it displaces the use of the natural sugars that are very valuable in nutrition. No other details will be given here, except to recommend the raw, well washed fruit or vegetable.

SELENIUM POISONING

Selenium poisoning of cattle was first encountered in the western plains, and was thought to be due to the high alkalinity of the water that was encountered in these regions. Later

it was found that the vegetation carried toxic quantities of selenium, **that is more than four parts per million.** Some plants as the Astragalus of several varieties appears to thrive on it while others die under the same supply from the soil. These plants serve to mark the area as selenium rich as they run as high as several thousand parts to a million. The selenium is distributed in patches and is found in shallow wells and ponds, but not in deep wells as a rule. Farmers find that wheat or other grains grown on the patches are toxic to their cattle or chickens and sell it to buy better food for them.

The toxic effects are had when the food contains four parts per million of this element. An acute condition, which may be quickly fatal, can occur when food containing twenty parts per million is taken in sufficient amounts. The chronic condition which develops, produces such effects as loss of hair, hoofs or finger nails, a general malnutrition, impotency and infertility. In chickens, the eggs do not hatch and in some cases the chickens may not even lay eggs.

Tissue studies in the Warburg chamber show that after being exposed to toxic amounts of selenium the oxidation of glucose succinate, lactate or citrate is blocked, but the oxidation of para-phenylenediamine is not hindered. This holds for all tissues examined as muscle, brain, kidney, liver or tumor slices. Thus from our standpoint it is a serious inhibitant of recovery.

It is possible that selenium may upset favorable steric advantages . Its action as a dehydrogenating agent can be concluded from the unusual oxidative behaviors of its oxide. Instead of oxidizing the aldehyde group of acetaldehyde to the carboxyl, as other oxidation agents do, it leaves the aldehyde group untouched and acts on the beta carbon atom oxidizing it to the aldehyde, thus producing glyoxal. The influence of selenium on the steric advantage offered by the host cell-pathogen combination is thought likewise to be irregular, so that distortion suffered does not permit the addition of the carbonyl catalyst in a position favorable to attack the pathogen for optimum dehydrogenation. Patients who have started splendid recoveries after a well selected dose of the treatment reagent, on returning to selenium rich districts, have suffered reverses, and

have finally landed in some hospital where the staff is entirely uninstructed in the situation at hand and know no better than to follow the routine of care that yields their usual high percent of failures in the treatment of real cancer. The patients, themselves, seem addicted to the toxic situation and long to return from a place where they are making splendid recoveries, to the unfavorable selenium laden district. Once they land there, they can not be persuaded to leave. Selenium requires competent psychiatric investigation, as well as its carcinogenic influences. For this reason, patients from selenium laden districts should not be placed under treatment unless complete arrangements are first made for their continuance in a correct environment until their recoveries are completed and well seasoned. The United States is spotted with such selenium areas, as for example, those located in the state of Ohio and Michigan.

Selenium exists in two forms, the inorganic and the organic. It is a widely distributed substance, so that we are always exposed to small nontoxic amounts. These may even be necessary to good nutrition, such as one or two parts per billion. The organic form is most toxic as it clings to the tissues longer after ingestion while the inorganic form finds its way into the urine more quickly. Our patients, because of the part played by the tissue oxidations in the recovery process, must be protected from any amount more than the normal trace amounts, and the urines should be regularly examined, as should the food on sufficient occasions. Sanitarium care properly conducted will prevent many mysterious failures we have encountered in the past. It is the duty of the Food and Drug administration to protect the public from the interstate commerce in foods poisoned by selenium. But although they know full well the havoc it causes, the public lacks adequate protection.

Selenium interferes with recovery much like the sulphides do. It is not found in sandy soil. Hence rye is free from it while wheat which is grown on heavy soil often carries appreciable quantities.

CHAPTER XIV

FURTHER APPLICATIONS OF THE FOREGOING PRINCIPLES

THE PREVENTION OF CANCER

We have considered the elements that enter into the initiation and propagation of the carcinogenic process and of virus parasitism, and also the means of terminating both in line with the same hypothesis. The practical application of the principles discussed is now in order. Two sources of toxin that play parts in the initiation and conduction of the pathogenesis need review.

Toxic amines produced by decarboxylation of amino acids in the acid reacting colon, or in tissue foci by bacteria, block the action of the tissue cell functional carbonyl groups, and thus wipe out the oxidative protection. They cause block in oxygen transport inter, and intracellularly besides, by gelling the tissue colloids. They cause weakness and clinical fever, as do other amidines. Their elimination is dealt with later.

Toxins brewed in old foci of infection where hypoxia is maintained through scar formation and vascular obliteration. Under hypoxic conditions, the free radicals formed by dehydrogenations are not peroxidized, and react with each other to polymerize with increase of molecular weight over long periods. Such old foci of infection flare up with acute inflammation, and then disappear with absorption of the old scar at the end of the recovery process from cancer. The acute inflammatory phase of the beginning of the whole pathogenesis is thus reproduced, but this time with the cure of the lesion, and the elimination of the disease from the system. Such toxins reaching the blood stream can copolymerize with tissue metabolic products and the collagenous material that forms the interstitial fibrosis, where hypoxia is more or less a factor. They play a part in the aging and insufficiencies that follow sclerotic degenerations of the heart, kidneys and brain, as well as other organs. This fibrogenic status of the cancer patient even before a

growth shows up should be easily recognized in the blood and tissue juices and be a diagnostic aid. It disappears after our treatment just as the concentrated scar does, and the interstitial fibrosis goes away with it, and arteriosclerosis as well. Good parenchymatous function is then returned and the victim so to speak becomes younger again. This is demonstrated in a few case histories given here, but a volume of considerable size would be required to give a good share of our experience in this matter.

The polymerizing toxins and their copolymeres with incompletely combusted tissue metabolites, and fibrogenic material and with other germ poisons while increasing in molecular weight exhibit differing symptoms from the lesser polymeres, and as the last deposits represent the largest molecular weights, the symptoms they produce disappear first with the digestion and removal of the toxin and its fibrotic deposits. As the depolymerization continues the same toxic arrangements are traversed transiently until the monomeric form of the toxin as produced by the germ is liberated. When first formed by the germ it was not oxidized, but now it is, so the whole pathogenesis is thrown out from its very inception. This is why we say the disease is **cured.** What applies to germ toxins applies also to the incompletely combusted tissue cell metabolites resulting from anoxia and inhibited oxidation catalysis.

One will see to it that his oxygen supply is adequate, and that he does not exert himself beyond the ability of the heart to perform well on the oxygen supply available to the heart muscle cells. In fact no function will be overdone. Pain is the warning symptom, but the damage is already done in part when it appears. One must learn his capacity and adjust himself to it. This applies to kidney function and digestion and bowel elimination as well. Thus one will combat the pathogenic factors as follows. First they must be recognized for the part they play in blocking normal function. Then they must be met with steps to prevent their action or their effects.

While these procedures have to do with the prevention of cancer, they must be employed also in the treatment of the disease. To some we can only call attention, for who can avoid the petroleum engine exhausts from flooding one on the

city streets? And how can the tar road dust be avoided by one who must use these roads. The proposal was made at the International Cancer Congress held in London nine or ten years ago to advise the public about the nature of the many industrial cancer causing poisons with which the workmen had to have contact, but some of the members of the group objected with sufficient vigor to prevent this very necessary act, I was informed.

Arsenic has long been recognized for its carcinogenic powers, and its use as an insecticide on fruits and especially to fight the tobacco mosaic virus places those who do not wash their fruits well in danger. The tobacco users cannot remove the arsenic from their cigars and cigarettes so they inhale the arsenic as a volatile compound which probably has greatly accelerated powers in that state. This we think is the cause of the increase in lung cancer rather than the products of the tobacco alone. One should think it over and employ different insecticides. These are extrinsic factors over which we have no direct control. However there are sufficient personal factors also that have to be sought out and removed both for the prevention and for the cure of cancer.

Factor No. I—The intestinal poisons or amines. They cannot exist (a) without the bacteria that produce the decarboxylases. (b) The decarboxylases cannot work without the amino acids to work on. (c) They can only work in an acid medium. So there are three ways of attacking this problem. Since the killing of the germs is not practicable, rendering them harmless is the best means of handling the situation. **Therefore maintaining an alkaline colon,** and not supplying the germs with substrate from which to produce toxins is the practical way. Since the intestinal tract is normally alkaline, it is a constructive way.

To produce and keep an alkaline colon which is its normal status, one does not eat too much food and thus keeps the bulk down, so the center of the bolus is well mixed with the alkaline secretions of the intestinal wall. This secretion runs around a pH. of 8.5 and no decarboxylation of amino acids takes place at that alkalinity. So if the bolus on reaching the colon is not too great it will not dilute the alkalinity to a point where it be-

comes acid enough (5.5 or even 3.5 in pH) whereby decarboxylation of amino acids is serious. Further, by holding down the food quantity the amount of amino acids taken are reduced so there is less material to be changed to poison. But there is the other factor of reduced amount of work to tire the bowel musculature and slow down the elimination. The less the bulk the easier the bowel can pass it on and out and the less the time for toxin absorption. It must be recalled that the colon must terminate the digestion of any left-over material, or get rid of it before it can be turned into a poison. So the alkalinity of the colon contents is the first thing to consider and bulk is the main factor here. Too much water must not be taken with meals or for an hour or two after a meal for the same reason.

To not provide germs with material for their prosperity, one should eat just what is needed for tissue building and energy requirements. One needs the salts and vitamins with the trace elements and the food substrates. Any excess provides the hazard we are trying to avoid, and accomplishes no useful purpose.

Factor No. II—is the cause of the hypoxia. Old scars if removable, and the chronically infected appendix should be removed as should tonsils that have been long infected, even though they cause no bother. This is especially true for women, for cancer of the breast is seen too often associated with such infection, and the tonsil flares up with reaction after the breast neoplasm is absorbed, thus demonstrating the etiological position of its scarred-in infection.

THE DIET

The Quantity of Food

We will take up dietary details in a minute, but first let us consider the balance between the digestive power as supplied by the stomach, liver, pancreas, and intestinal wall enzymes, and the amount of food that is taken. This ratio is so variable that no one can predict the state of affairs until it is carefully examined. Some persons have an awe inspiring proteolytic digestion. They can devour large amounts of fish, eggs, meat and chicken and prosper on it, that is for a while at least. Others cannot even take a gram of animal pro-

tein into their stomachs without suffering the effects of toxic amines a day or two later, and then a cathartic is needed to clear the system. This latter group have often sprung from the first group after they became host to some infernal fecal germ of which there seems to be an endless variety. They then are on the way to cancer. But even those who think they are safely set in the first group can give rise to the most malignant forms of lymphsarcoma and be killed thereby in a few weeks or months. The second group, on the other hand, are subject to breast and liver or gastro-intestinal cancer.

When it is found that the food does not digest in adequate amounts as shown by fecal analysis and the symptomatology, one reduces the amount to a little below the amount that can be handled readily. The food quantity is thus determined by the capacity that is actually found to be operative. As a rule persons eat much too much and a drastic cut as by a fast of a week or two on fruit or vegetable juices and vitamins will take the irritation and nonsense out of the appetite so that the normal warning that enough has been taken at a meal will be heard and understood and then obeyed. This warning is a sense of satiety that soon says stop after the meal gets going. It is almost never heeded in ordinary practice, and especially when a hostess has taken pride in the meal "she alone can prepare," and must be flattered by a "Portus" appetite. The danger is the greatest when meats and eggs are served, and the appetite is whipped up by relishes and a nice wine. This dainty sense of sufficiency, if abused, soon leaves, and one then only knows that he cannot fill up any more when the stomach is full to the brim. Then he goes from the table with a sense of satisfaction he misinterprets. Wives have the duty to warn their husbands against over-eating when Nature's warning fails, as this safety warning is easily lost after abuse. Yet after a fast it is likely to be restored. Then one knows what his needs are, and he takes the amount of food that Nature judges is correct and only so much.

The spacing of the meals must accommodate the speed of digestion too. In some the speed is great and the food is ready for absorption as nutritional units in only a few hours after being taken. In others it does not even get out of the

stomach into the intestine where the main job of digestion is done for six or seven hours. Two meals a day will then provide the correct spacing so the stomach gets a little rest before being put into another exhaustive task. And of course when it does not survive, ulcers, diverticuli and cancer come, and besides, arthritis and other affairs are invited.

Many decades ago the school children were shown pictures of stomachs of men that drank liquor. These were ugly imaginary productions for sale to the board of education. But while they taught us what kind of stomachs we never wanted to have, they did not teach us the truth about their production, Since the little camera was invented that can be swallowed and then used to take pictures of the stomach within and of all sides, it is found that the "hobos" that eat very little and survive on bibling liquor even of the cheapest kind, indeed have the nicest stomachs. These stomachs are never overworked with food that can rot inside and injure. But instead, they receive their daily antiseptic lavage with beer, whiskey, gin, moon-shine and the like. The fates of the kidneys and liver are a different matter. These go to destruction only too soon so the hobo ends up in a terrible fix that is as bad as the man who never drank but ate to capacity on every occasion.

Caloric and protein requirements as set by dieticians a few decades ago have been in error as practical experience shows. They have been much too high. The man who shovels coal steadily all day on a light breakfast; a sandwich at noon and a light dinner at night has baffled the doctor, but it should not, for the energy of the food was efficiently turned into work and the mechanism was not hindered by block to the oxidation catalysis through the enormous amounts of toxic amines into which the large meal could be converted. The small food ration gives the whole system a chance to practice an efficiency to which the biochemist too often denies existence, because he has been deceived by faulty experiments.

If the protein requirements as set for an average sized man are cut to one half, they are probably enough and maybe too much for ordinary needs. The waste materials are so cut and the toxins formed so little that the fibrosis that separates the working machinery of muscle and gland cells from their

vascular nutrition as age advances gets little chance to form, and so the same functional efficiency is seen in the frequent fast, or the meager eater, that one sees in the child. That clarifies the amount of work a laborer can perform on a low protein ration. Our patients lose their fibrosis and often gain the same circulatory advantage. Since the proteins are the sources of the dangerous poisons, they need the most careful watching instead of the starches and fats that we hear so much about as causing the deadly lesions of vascular disease. In fact with a correct protein intake, the natural, unmolested starches, sugars and fats are burned efficiently, yield energy, and leave no residues to cause the lesions they are imagined to cause. One need not worry about cholesterol on a sensibly low protein diet, or so much about sugar or starch, even in some serious cases of diabetes mellitus, for I have seen such instances where the famous experts claimed no help could be given and where generous increase of insulin failed to stop the onward progress of the disease, that a protein free diet so called, as of fruit sugar, honey, vegetables and fruits brought on a steady satisfactory recovery while the insulin was being rapidly withdrawn.

The unwillingness of the "routine specialists" to view the functional insufficiency of an endocrine gland, even when it uses carbohydrate as the substrate for its activity, as possibly due to an error in the ultilization of proteins places him at a great disadvantage for he is then held to the routine of aggravating the pathogenesis by a high protein diet. This point should be taken seriously and the real cause of the diabetes in each case should be determined. In some it is a focal infection, in others a diverticulosis in the colon, and in others it can be cancer or syphilis and even tuberculosis. In others it is an anergy where a toxin diverts the energy that should produce insulin into a wasteful channel, or blocks its production all together. When the nature of the toxin is realized, it might be possible to do away with it.

Every diabetic can not throw away the conventional regime and indeed it has saved many a life when rigorously followed. So the pioneers in its development deserve the everlasting gratitude of humanity for their achievement, but that does not mean

that the disease was conquered. For indeed the regime that is approved has never cured a case as yet and every diabetic has died with diabetes under such care.

The best diet is one which in least amount is able to support the energy and growth or repair needs of the body under the circumstances that exist at the time. There are several ways of going about its determination. The so-called scientific procedures require much apparatus and invite serious error. The most reasonable procedure to find the least amount of food needed to keep the weight and strength in good order, is by trial, guided somewhat by the calculated caloric requirements per kilo body weight, and the amount of work done or heat required to hold the body temperature right. Setting-up exercises and weight estimates may be needed and the diet should be protected from contamination by oxidation inhibitants such as the fruit acids that carry calcium and other cations off into the urine. *Too much citric, tartaric and lactic acids should therefore be avoided.* The diet should contain no toxic materials nor should it produce them. It should stimulate digestive juice flow and gastro-intestinal movements, but not overdo them. As we have seen, too much bulk, aside from carrying useless and potentially dangerous protein excess, can also bring about the toxic amine production that inhibits secretion of digestive enzymes and paralyzes intestinal motion.

Too much bulk not only tires the muscles of the digestive tract so they cannot move and expel the contents they should and thus allows for the support of bacteria and the generation of toxins, but the great bulk stretches the walls of the viscera so they are thinned out and the blood supply for any area is reduced. Likewise the blood vessels are drawn out in length and thus reduced in caliber. The result is reduced blood supply again right when the tissues need the best oxygen supply to carry on their work. This relative hypoxia is another factor in producing cancer and gastric ulcer. So while too much bulk provides the conditions and material for toxic amine production, the fact that it occurs and is not withstood by the patient shows that he is sick and also what the pathology is chemically. One must therefore search for defects in bowel structure that favor it mechanically as via a kinked appendix or a diverticulo-

sis. It is here that the toxic amines are produced for the most part.

Structural Defects

There are invalids who are poisoned by the least amount of protein of animal origin. As time progresses this sensitivity increases until they are forced to live on the raw fruits and vegetables, to escape migraines, urticarias and other allergic affairs. The intestinal radiographs showed enlarged full gall bladder and a diverticulitis without visualizing the appendix. The sphincter ani and pylori muscles as well as the gall duct muscles tend to be spastic and there are other signs of vagotonia. The sensitivity increases with the increase in the structural changes as time progresses. Obstruction with fecal retention and toxin production is the anatomical cause of the invalidism and with time the resistance to the toxic amines and other products is broken down while the facilities for their production increase. Each diverticulum is an area where the wall has become paralyzed by the action of the amines as we explained before and a local vicious circle is established.

The appendix may by previous inflammation become adherent to the caecal wall posteriorly so it is not visualized on the radiograph except at a late period after the cecum is emptied and as this visualization occurs so rarely one may never find it. This fact alone means that a pocket of obstruction probably exists until proven otherwise. It probably started the diverticulosis, and the latter will not be cured until the appendix is removed. A course of terramycin may clear the symptomatology and permit the victim to go on an animal food spree so long as the colon and its recesses are kept sterile with the antibiotic. But as the liver will not take such treatment for too long, the test carries its dangers. These should be profited by with an immediate exploration and appendectomy while the colon is held fairly clean. This is the logical procedure.

One may while following an animal protein free diet, take the oxidation catalysts of our text and thus get the intestinal wall to win against the diverticulosis, and maybe even get rid of the fibrosis that has bound the appendix in its vicious position, and thus condition it to empty normally. But the adhe-

sions may be too firm to yield, so that surgical removal is required after all.

With removal of the appendix and following a careful diet and use of the carbonyl catalysts one may gradually overcome the trouble and the vagotonia will disappear, the gall bladder will empty and the bile get into the intestine where it can do its work instead of accumulating in the gall bladder and blocking bile production and elimination. The yellow color in the eyes and skin may thus be removed and this easy observation serves as an index to the progress being made during the recovery.

The Quality of the Food

Foods are selected so as to avoid their potential toxin production as caused by bacterial action, favored in the manner discussed above, or to avoid an actual toxic contaminant. The latter may be contributed from the soil as in the case of selenium, or it may be an insecticide residue.

Meats not only supply too much protein for ordinary use, the excess offering substrate for toxic amine production, but they carry other elements essential to bacterial activity not found in fruits and vegetables until the latter became putrefied. Their exact nature we do not know. They are not the vitamins of course, except that we suspected vitamin B_6—Pyridoxal phosphate. This is the co-enzyme essential to the amino acid decarboxylases which produce the toxic amines. D-alanine, pyridoxamine, and pyridoxine serve bacteria in different degrees selectively for production of the energy carrying phosphate even though they are used by higher animals with equal ease. And while the streptococcus fecalis can not decarboxylate without the vitamin B_6 complex as coenzyme, this is not the stimulant that is most important to bacterial prosperity to which we refer. Even the lactic acid bacillus requires B_6 as the phosphate itself, which it alone can use as a growth factor. Of course the energy stored in the phosphate requires work to accomplish this phosphorylation, and ATP plus an enzyme are required for the transfer. Therefore the accessory affair may be a dynamic situation of this kind, one factor being supplied by the meat in greater amounts than it is offered by vegetables and fruits,

while the enzyme may be offered by some germ in the intestinal tract. The value of the lactic acid bacillus may be, not the acid it produces which would favor decarboxylation and the production of the toxic amines by the acidity of the bolus it necessarily produces, but the fact that it can not use the pyridoxal or its amine, and when in great enough numbers will monopolize all of the pyridoxal phosphate the toxic germs can produce through their enzymes and thus starve them out. Bacteria need the phosphate for many of their vital reactions as for tryptophan synthesis from indole and serine, and for its conversion to nicotinic acid, for certain transaminations as well as the decarboxylation of amino acid that interests us. Hence the use of such foods as buttermilk or its products may do good or harm depending on the balance that exists with other factors in the intestine. And not all persons are like. This is indeed the point we are working toward, the analysis of each case as it comes with regard to the animal protein back-ground. So while lactobacillus products may serve in the prevention of cancer in some persons, they may prove very harmful and even rapidly fatal to cases of bowel cancer where obstruction has piled up a predominance of decarboxylating bacteria before such poison producers can be starved out of the way via pyridoxine phosphate deficiency. I have actually seen this injury done by kumyss and sour milk recommended by their enthusiasts.

The best plan is to leave meat or animal products out of the picture except for butter and cod liver oil and honey, and use the better forms of nutrition provided by fruits, vegetables and cereals.

The diet of best quality then will not encourage lactic acid producing germs in the intestinal tract, except the lacto-bacillus in certain favorable instances. The lactic acid producing streptococcus fecalis not only is a great lactic acid producer, but it does so to favor its decarboxylating enzymes, so this is one germ we would do well to get rid of. There are others of much the same significance, and *to block them all we search for bacteriophages* whose natural habitat is the food. We are now examining a certain cheese which shows the desired action at an

early period in its maturation. Such phages are transient in their useful phase of existence. The Lincoln phage used in the respiratory tract may serve as an intestinal purifier, too. At present, it is too early to say for sure.

The intestine is supplied with a great variety of bacteriophages that are active in killing toxic germs. Especially valuable are those that kill the toxic amine producers. Thus the paralytic effects of infections within the colon can be prevented and the energy of the tissues in general is not reduced by the oxidation inhibitants evolved by decarboxylating bacteria. One might say that his intestinal function is as good as his bacteriophage, other things being equal. When the cultures that carry these minute friends of life are depleted, they should be renewed. In the United States, therapy of this type is not generally practiced. However, when one sees the wide and successful use of the bacteriophages, as in Brasil, one can see how they can reduce the demand for cathartics and headache medicines and possibly contribute to the resistance against cancer. Thus the food has a chance to be digested properly and be absorbed as nutritional elements and play its part in the tissue economy as nature intended, instead of becoming serious poisons.

Among the bacteria destroyed by the bacteriophages, the Escherichia coli, Salmonella typhii, Shigella dysentariae, and the Streptococcus faecalis are included. Thus a wide range of protection is offered by the normal intestinal bacteriophages. They can be supplied therapeuatically by mouth or enema, to re-establish the normal protection. We believe that bacteriophage intestinal cleansing can be included in our detoxication regime, with the idea in mind that it may be safe for some patients to eat a little meat and drink clean milk in addition to following the vegetarian diet.

Practical observation shows that antiphage changes in the bacteria of the intestine where elimination is constantly going on, is not such a problem as in infected sinuses and focal infection where drainage is impeded.

Any group of vegetables and fruits or cereals that form a pleasing variety offer all the food elements needed if they are prepared correctly, and come from good soil. Good soil is

one with all the necessary trace and gross elements, and without the toxic materials as arsenic and selenium, or the sulphides that may enter as contaminants. *Aluminum of the clay soils as compared with the aluminum-free sandy soils* offers disadvantages too, and for this reason also the whole rye which is generally grown on sand is a better source of nutrition than wheat grown on clay. Orchards sprayed with arsenical insecticides offer a definite hazard, and naturally occurring selenium may so poison the food as to favor the development of cancer. And as we know from experience, it actually blocks the recovery process as instituted by our treatment. Peas, and especially canned peas grown in the middle western states have often been so poisoned and have blocked nicely progressing recoveries many times. We therefore forbid all peas from the diet unless the soil is analyzed and found acceptable. Even at that the protein content is too high to serve people that are not working hard physically, and the same holds for lentils. Our patients require a diet much poorer in protein than peas contain.

The newer insecticides are also poisonous as cattle have died after eating the "cake" left after pressing the oil out of corn, and people have been made seriously sick from the use of the oil. The need of good cleansing is seen.

Corn likewise finds its biggest production in the Middle Western States where selenium abounds in patches as in Ohio and Michigan and it can prove somewhat carcinogenic. Corn oil is found to be so in experimental animals, by E. C. and A. J. Miller, and while no reason is assigned suspicion should be directed here, as well as in the peroxidation of the unsaturated fatty acids it contains. All soils should be held under suspicion until proven selenium free, so serious is this matter.

There are regions where the cancer incidence is markedly less than in others and where good teeth are the rule. Portugal is found to have a low cancer incidence in the rural districts. The diet values are sharply contrasted in such countries as Belgium where the peasants eat plenty of whole rye bread, and the city people eat the white bread and canned foods. Both groups eat meat and cheese, the peasants less, however. The peasants rarely have to see a doctor and cancer is much less

frequent than in the city dwellers whose diets and ills run much like in other cities. The peasants are mostly gardeners and their crouching position at work aids bowel elimination too. They are hard workers and obey the blessing to earn their living at the sweat of their brows.

Protein Food Selection

A word about the protein content of vegetables and cereals is in order, since the popular conception is that they contain such meager amounts that they do not serve the nutrition adequately. The error of this conclusion is evident from the fact that the cow can make enough milk by eating grass or hay to supply the village children with protein and calcium for building bone and muscle, skin and hair, brain and viscera, and still have enough left over to feed her calf. If all this nutrition did not come from the grass or other vegetables, where did she get it? The fleet deer, the strong ox, and the tireless horse and donkey, the POWERFUL elephant lack nothing on the vegetable diet, and neither does man. Compare the protein content of raw beef cuts with rye grain. One pound of raw boneless beef contains 84.5 grams of protein. Beef with bone offers 73.5 grams of protein, and beef ground into "hamburger" offers 73 grams of protein. Rye flour, one cup or 80 grams contains 7.5 grams of protein or roughly 43 grams per pound. Thus it carries half the protein content of an average meat. Nuts carry from 9 to 10% protein and milk contains only 3.5%, while liver carries the usual 20%. Lentils dry offer 25% protein. Lettuce has 1.5% and cabbage 1.4%. So one can choose more protein in his vegetarian diet than is good for him. The fate of the amino acids determines the nutritional value of the food, for if they are decarboxylated and converted into poisons that weaken the muscles and all other functions, not only is their nutritional value lost, but they hinder the functions that are in progress as well. The foods should be well digested and protected from vicious bacterial action so their products of digestion serve as the nutrition they are intended to be.

Proteins should be selected with reference to their freedom from such amino acids as arginin which on decarboxylation yields the poisonous agmatine in an acid reacting colon and in

foci of walled in infection. Arginin is proven by Irons and others to increase cancer growth greatly. Its elimination from the diet they showed discouraged cancer development. We think the agmatine product is the factor at fault. However, nuts, fish-eggs, seeds, should be avoided with other foods of high arginin content. Peanuts are one of these which seem to favor the development of pathogenic germs in the colon and appendix. When roasted they offer a double injury through their burned fat—acrolein content. Foods must be studied therefore in line with the principles set forth in this outline.

Compositions of Ordinary Foods in Proteins

In order to soften the fear that one may starve to death for want of proteins when eating only vegetables, fruits, and cereals a comparison will show that there is no basis for the fear. These figures are taken from tables supplied by the U. S. Dept. of Agriculture and are authoritative. If one compares the protein supplied with the requirement of only 0.3 gms. per day per kilo body weight one sees how stupid it is to overload the metabolism with the habitual amounts of nitrogenous material that are pathogenic. Two big buckwheat pancakes or three wheat cakes at breakfast will supply it for the whole day. One hundred grams of peanut butter supplies 26.1 grams of protein, 100 grams of beef roast pretty well baked dry or roast lamb would do as well, and so would 150 grams of liver, or three quarters of a liter of milk, or 150 grams of rye flour cooked into a porridge or bread. This would be about three good slices of whole dark rye bread, or one fair sized lamb chop, a bowl of pea soup, or a big piece of fish, or a hand full of nuts. On such a diet the protein excretion by the bowel would also be a minimum and in equilibrium with the intake, and the intracellular nutritional pool would be well enough supplied for metabolic purposes while not so great as to invite parasitic invasion.

When one eats apples, pears or peaches, plums, etc., where the protein content is from three tenths to seven tenths percent, one is supplied with so much energy producing carbohydrate that the amount required for a cleansing fast need not be disagreeably large, and could be carried on for many days

before the protein reserve stored in the tissues would be depleted dangerously. Thus fruits offer the best simple means of cleaning house. Bananas give over one percent protein and 30% carbohydrates as well as much calcium and phosphate and ascorbic acid, and only 0.4% of fat. They are an ideal energy producing diet. The egg is about the richest all round food there is outside of carbohydrate and ascorbic acid, running about 13% protein and 12% fat. It is the ideal diet for the support of tissue growth, including, blood, bones, skin, hair or feathers, muscle, viscera, brains, nerves, and even endocrine glands, and their secretions. It gives for each 100 gms., 54 mgms. calcium, 210 mgms. phosphorus, 1,140 I. U. vitamin A, 0.10 mgms. thiamine, 0.29 of riboflavin, 0.1 mgms. niacin, and 2.7 mgms. of iron. Evidently ascorbic acid is not so essential to early tissue growth but has to do with energy production, as its structure also indicates.

Bran of wheat on the other hand offers, 12.0% protein, 3.4% fat, 4.2% carbohydrate, 7.8% ash, and per each 100 grams, 94 mgms. calcium, 1,312 mgms. of phosphorus, 10.3 mgms. of iron, 0.37 mgms. thiamine, 0.39 mgms. riboflavin, and no ascorbic acid. Thus for an all round diet a mixed choice is necessary and must include fruits and vegetables eaten raw so as to have the benefit of the vitamin C as fully as possible. Apples carry 5 mgms. ascorbic acid per 100 grams, raw asparagus 33 mgms. per 100 gms. and cooked asparagus 23 mgms. per 100 grams. Raw Broccoli offers 118 mgms. and cooked Broccoli 7 mgms. per 100 grams, while cabbage offers about half that amount and lemon juice about the same as cabbage. Meat runs low in carbohydrate and vitamins A and C, zero in fact. There are many tables prepared by different analysts that do not agree very well. Probably the best is Agriculture Handbook No. 8, issued by the U. S. Agriculture Department on the composition of foods.

Relativity and Balance in Diet

In nutrition as in mathematics, everything is relative. We have demonstrated the case for the vegetarian diet that all our patients must start with until their oxidation capacity has been restored to a better grade than was "normal" with them

for a decade or two. Then the animal proteins may be taken, but until accurate measurements of the blood pH values and the Oxidation Reduction potential and oxygen concentration are established, the tests to see if meats may be added by a trial meat meal or two are not to be made. It may be only a month after the treatment that the patient is found in condition to take a little boiled meat or fish, but it may be much longer. The care with which he cooperates from the start will be one determining factor at least.

It must be remembered that when patients come to us, they are in the terminal stage of a deficiency disease. In other words they are in "the red" or far on the negative side of the metabolic balance. **Many cannot even digest a simple article of food and must be given peptic and pancreatic extracts to gain a little start.** Many have to start farther down the ladder than that. They cannot even handle the digested food products after these are absorbed into the blood stream and reach the liver. **The only thing to do with them is to give them a fast,** in so doing a little ability that is not used up will be at hand to make a little nutrition useful. One such case, Mr. John K., a case of cancer of the colon received from the Henry Ford Hospital with his abdomen completely filled with cancer that ate through as large fistulous cauliflower masses all over its surface, who lost from 200 lbs. to less than 113 lbs. in weight, and what he had left was mostly skeleton. After the recovery process was well under way, he started to gain weight so fast that the Henry Ford Hospital in its check up could not believe it. His weight went from less than 113 lbs. at the end of June to over 160 lbs. by the end of August when he returned to work. Thus, though we had to starve him a little at the start, the increase in his nutritional ability went forward in geometric proportions. It should also be remembered, that a patient with "fungating carcinoma of the colon" is not able to consume the large quantities of food a normal person would to maintain body weight and health. That this Cancer condition must be corrected before sufficient food can be taken by the patient to maintain his present weight and build it back to the normal. In such matters one must first fence for an advantage, and then use it,

but not break it down by overdoing it. The Mrs McA. case is another where for a few weeks she gained two and a half pounds a day. These gains were made on vegetables.

Some patients become vegetarians by choice. Later they may eat meat, but it did not appeal to them. They wondered why they had worried so much about it. They soon forget it and enjoy well cooked cabbage, potatoes, and the other vegetables, and not only like them, but feel better, lighter, quicker, cooler, and their perspiration does not have a bad odor either, nor does their breath. They stay free from sickness too and rarely if ever catch a cold and when they do it is of little consequence and soon passes off. Man was made to live on fruit and vegetables, and the sooner he returns to them the better off he will be.

Selection of Fats, Sugars and Cereals

The best fats are those rich in unsaturated fatty acids that have not been processed by reduction to prevent them from becoming rancid, and that have not been exposed to air so as to become rancid. They are toxic when rancid, since the molar peroxides formed and their decomposition products are irritant and can give rise to aldehyde polymeres with free radical terminals that serve as co-carcinogens, much like the overheated fats in which glycerol is converted to acrylic aldehyde polymeres with free radical terminals. Such fats are indeed stimulants to cancer growth as 40 years of experience in medical practice has convinced the writer. Overheated fats are not allowed and slowly oxidized fats are likewise not allowed in the diet we recommend for the reasons just stated.

The value of the fat as a protective agent lies in its unsaturation and this must be protected by not being exposed to air. Corn and peanut oils are naturally of this type but they do not always get the protection of being pressed out with air exclusion and during packaging as olive oil does. Yet they should as corn oil has been shown by the Millers to be slightly carcinogenic while olive oil is somewhat protective. The selenium content of corn oil may require consideration too. Therefore if fats are eaten they should be cooked by boiling in water, and olive oil containers must be well stoppered and

not too large so that exposure to admitted air is the minimum possible. Lard and all rendered fats are overheated and contain the co-carcinogenic acrylic aldehyde polymeres. And eggs fried in bacon, sadly enough, must be looked upon askance, unless one is on a hunting or fishing trip where physical exertion is plentiful. The Portuguese who use olive oil freely rarely have cancer and generally have good teeth. They protect the oil from injury by air or other exposures.

The saturated fats as produced by reduction may not be a bit carcinogenic. But they do not protect from cancer and germ toxins either as do the unsaturated fats. The process of oxidation starts coupled oxidation chains in the toxin and fuel materials that carry their combustion. Therefore it has long been observed in our practice, that members of the family that ate fat meat escaped the tuberculosis that those who only ate the lean at the same table were subject to. The Eskimos also escape cancer, it is supposed for the same reason.

The selection of sugars brings up a few questions too. We hear how white sugar is the root of all evils. I have never been intelligent enough to appreciate this, and except for the fact that if any bleach material is retained to do harm, the sugar is otherwise harmless. The fact that it is purified and the best part of the cane or beet juice is thrown away, is a big point against it. The brown sugar or molasses is therefore a superior food. But they carry also the ability to stimulate bowel activity and perhaps also some antibiotic values that protect the intestinal contents from harmful bacterial changes.

Cereals are all good. Rye is maybe the best as it is grown on sandy soil and its silicon sun activated products in the bran are oxidation aids of value and maybe for this reason the black rye bread eating peoples are far more efficient and free from cancer and live to a greater age than the white bread eaters. Right in the colon where the vanguard of disease is brewed, they can manage their detoxications and protect all the tissues from damage from intestinal products that kill. Sand soil does not contain selenium as does clay where wheat is grown.

Food Preparation

Other factors are to be considered with regard to cereal

selection, and preparation. One that is most important and always neglected maybe, is the grinding into flour fresh just before using. Among other reasons, such as protecting the fats from oxidation outside the body, the oxidation of vitamin E can prove a danger, and as vitamin E is a poor quinone when oxidized it can displace a live quinone which can do service in the tissues which the oxidized vitamin E cannot do. Medicines with this material we believe are a potential danger too, and one should get his vitamins fresh from the grain like he should its other products as he would if he chewed it to swallow. But not being able to do that he can use a grinding machine and immediately bake the product or cook it into a porridge. The finer it is ground and the shorter the time of heating the better. It is likewise better to use freshly ground bran.

Such precautions are logical in view of the chemistry of vitamin E. It is one quinone whose physiological service does NOT follow its oxidation powers. Thus its physiological activity is proportional to the methyl substitution in the quinone ring, while this substitution lessens the dehydrogenating activity of the carbonyl group even though it contributes electrons to the double bond. Thus the physiological activity is greatest when the oxidation powers are least. This holds for the oxidized form which may then substitute for an actively oxidizing quinone and so exclude valuable quinone action from the metabolism. Its function is of course to protect mature rats from placental atrophy and fetal absorption, and to prevent degeneration of the spermatogenic tissues in the male. In certain animals its deficiency causes a condition resembling multiple sclerosis in man, but the vitamin has no curative action in man, afflicted with this disease.

The cooking should always be done with as low a flame as serves efficiently. Thus volatile flavors are not lost so rapidly. While some foods cooked in aluminum ware taste better than when cooked otherwise, still others have a better flavor when cooked in stainless steel or pyrex. The latter is the best as no chemical reaction takes place between the vessel and the material. A good brand of stainless steel has much the same advantage. The solubility of aluminum in water, diluted acids and alkalies, make it become a part of the food. It

may readily react with the food in more than one way, and some students of the subject have condemned it, bringing forth many convincing examples of its harmfulness even to healthy persons. It does disturb the calcium and phosphate balance, and it may be harmful in paralytic diseases. Whether or not it plays a part in the increasing incidence of Infantile Paralysis I do not know. Some say that the use of aluminum has to do with the greater prevalence of cancer. We do forbid its use by our patients however as a safety measure. This may be one reason patients under strict observation and care do best. To preserve the wonderful cooking advantages of aluminum, it should be made into an alloy that is insoluble.

The Fast

One amusing means of classifying one's patients is their reaction to a sudden suggestion to fast for a while. The resentment may be exhibited in some as a sort of "shooting in self defense" response, while a critical laugh, of "do you think I am crazy" may be the response of another. However when the purposes and advantages are understood, the task is undertaken quite eagerly, and more than one big league ball player reported better efficiency following it. The fast need not be absolute or prolonged. In fact it should be only a change in the process of nutrition and the word "fast" should not be used at all as it is not accurate unless actually an abstinence from all food except water is undertaken. Only in rare instances and for a few days is such a drastic affair required. The word however offers the "victim" a flattering sense of self control. Some essentials for nutrition must be provided, as the trace elements and vitamins. One may add some soda bicarbonate, and an intestinal disinfectant as camomile tea or one of the Brazilian herbs which do such wonders. Another plan is to just eat watermelon as much as one can eat with comfort to sweep the tract clear while avoiding other foods. This too is a little diuretic. When fruits come in season and are at their best they may be eaten exclusively with benefit, provided they cause no special harmful symptoms This holds also for a big meal of strawberries for those who find them agreeable. Alpine Preizel berries are famous for some protective action, and so a selec-

tion of food when its season is at its best, while abstaining from known insecticide poisoning, will bring the turn for the better by elimination of toxin production and perhaps even changing the intestinal flora. The fast may be made on apple juice. The apples should be well washed to remove all insecticide deposits, and hence soap and brush and water should be employed generously. One may drink all he wishes unless he is a severe diabetic and then he can increase the amount as the oxidation inhibitors are washed out from the intestinal tract. The enema with a teaspoonful of soda to a liter of water may be helpful here too, and for the same purpose. Molasses is somewhat antibiotic and a stimulant to peristalsis and could be used in the enema too. Honey serves both effects as well. After a day of slight headache hunger leaves and one feels rewarded in his lightness and speed, and his clearer thinking. Food should be taken gradually when the fast is broken, and the sensation to quit eating when enough was had will come again. It should be heeded and developed.

Dietary Regimes and Remedies

Various dietary regimes have been put into use to overcome the intestinal intoxication that breaks the way for disease. The grape cure, the cabbage cure—all have had their adherents. Tartaric acid is not burned in the body and it combines the important tissue cations and takes them out into the urine. The loss is serious. All the acids which lower the pH of the intestinal contents are also augmentors of decarboxylation of amino acids and its toxic amine production. Grape and sour milk regimes are therefore dangerous when overdone.

The fast or the comparative fast, using enough powdered chalk to cut any excessive acidity from the fruit, is reported to give the best results. A more rational development of such procedures is the William Howard Hay diet. The Hay idea was to not mix the proteins, fats, and carbohydrates during any one meal. Take starches at one meal, proteins at another, etc. Thus he reduced the dilution of the enzyme that was to do the main work, and kept the bulk down. The Arturo Guzman diet is much the same; but Guzman emphasizes the need to not dilute the food with too much water, and to suspend drinking till an

hour before the next meal. The selection of dietary articles that go well together was also emphasized.

The Kellogg diet was well propagandized and supported by sanitarium facilities. But I could not see the advantage of the sour milk products, in view of the harm that has definitely been done by such a regime. So my own idea was the simplest possible. Follow the Hay system of the monodiet in a sense—but as it is provided by nature, vegetables at one meal, and fruits at another and cereals at another. The latter carry the high protein fraction and beans could be taken then with the black rye bread, too. Then the seasonal spree on whatever is in season and at its best—as a few days on watermelon, a few days on peaches, and when apples come in give them and pears a whirl, and the same for the berries. If one thus follows what nature provides as the wild man would get it in the woods, he would not miss anything either so far as fun or benefits are concerned. One should eat generously for the first day or two, which would serve as a replacement exercise. Raw food including raw potatoes, radishes and apples with their oxidases, give the intestine a purification that it occasionally requires to prevent intestinal poisoning from popping up or staying chronically. Probably a retained typhoid infection is at the bottom of many such cases. The antibiotics that are intended to serve as sterilizers of the intestinal tract have enjoyed their best success in the acute case, but in chronic cases they are temporarily successful only and the aftermath is the persistence of a more resistant and toxic parasite than before the antibiotic was used. I congratulate anyone who has seen better results. Besides, liver damage is often too great, or there may be a mental injury that does not pass off too easy. Simple household remedies as sulphur and molasses should be studied bacteriologically to learn the reason for the prolonged duration of the good they accomplish. It may be traditional, but some old women with progressive arthritides have enjoyed their sulphur better when mixed with a little gin, and think what one may, the benefits they enjoyed have stretched out for decades. The intestinal flora seems to lose its viciousness when confronted with sulphur flour dealt out in that way. *Calomel,* the most denounced of all remedies, and maybe justly at times, still was

in use popularly when cancer was a rare disease. But now with much cancer and no *calomel* one wonders if after all it was not one of the best intestinal antiseptics, and hence a preventive measure against cancer.

Then there is the difficult but miraculous system of Homeopathy. Think of a doctor differentiating between thousands of remedies to find the exact one that is needed to secure a real cure in a particular case. It often took days in the chronic cases. Yet Homeopathy must be revived if we are going to send better physical specimens to the army, and in much higher percentage than we can now. Science has not yet reached the state of development to understand it. Still it offers the best service sufferers from liver disease can receive. When the correct remedy is chosen the response is often miraculous. The writer has never received homeopathic training except what was self-imposed after becoming envious of the results Homeopathists obtained in cases of liver disturbance where no allopathic approach was even to be dreamed of. Fine shades of distinction in these complaints made it possible to adapt the treatment with the same accuracy that a key fits a lock. Allopaths cannot understand this.

With good digestion and elimination and a non-toxic regime, one has little chance to get sick at all, even the common cold finds no soil to take hold. The vascular diseases stay away, and remarkable efficiency stays with the subject even as the years go by. When the digestive powers fail, *pepsin—pancreatin* will help. Since cancer is a secondary development, its prevention follows the cure in the true sense of pre-existing diseases by the proper Homeopathic remedy. Cancer was much less prevalent when the diseases of youth were cured by Homeopathy instead of suppressed and allowed to go chronic through faulty treatment. Homeopathy eliminated the basis for the disease and so the cure could be complete instead of hanging on to brew the toxins that cause tissue atrophies and neoplasias as we have described them. The future is bright with the recent revival of this wonderful science. Now that the mystery is taken out of the high dilutions it employs by the recent advances in isotope chemistry and their investigation as used in

Homeopathic potencies, many vague phenomena receive a physical basis which invites an intense therapeutic interest.

Decades ago we observed that with the reappearance and reversal of the symptoms of the pathogenesis as the cure process advanced, that the final symptoms that returned as the cure was just completed, gave a drug picture of a remedy which could have been used to really cure an acute infection. But under allopathic care it was not given. The pathogen therefore remained to evolve a long pathogenesis that ended up in cancer. The reversal of the affair by our remedy cleared the whole procedure from the system and showed in the final symptomatology of the cure what Homeopathic remedy would have cured it years ago in the first place when it started had it only been given then.

During the highly fatal epidemics of Influenza of the First World War the Allopathic treatment was used mostly. The death rate was about 46% under aspirin and whiskey, according to the U. S. Public Health statistics. These also showed that the Christian Scientists who used no medicine, not even a cathartic, only had a death rate of 3.5% while the homeopaths had a death rate of from 1% to 1.5%. These statistics were collected on many thousands of cases and covered the whole country.

DIGESTIVE AIDS

Bowel Cleansing and Catharsis

All foods are a mixture of protein, carbohydrate and fat in varying proportions, with one or two of the three, fading out to zero proportions. Thus bananas for example, carry much carbohydrate, small amounts of protein and no fat. Meat carries much protein, some fat and no carbohydrate. But man eats by habit in such a way that the heavy protein foods are taken first, and if a carbohydrate is desired it is taken last. It appears that the Creator arranged the digestive process to accommodate this habit, as the shape of the stomach and position of the acid pepsin enzymes for protein digestion provide. Thus when protein food is taken first it is deposited in a layer against the wall of the sac or fundus portion where it quietly lays undergoing the action of acid and pepsin required for the first

steps in protein digestion. When this change is accomplished the liquified product is allowed to flow onward into the duodenum where the digestion is continued in an alkaline medium.

The food is not churned about as in a mixing machine as was taught for so long. The carbohydrates which require an alkaline medium to start their digestion by the ptyalin of the saliva are taken last and deposit on top of the protein layer where digestion progresses without being hindered by being mixed with the acid secretions of the stomach wall. Since ordinary eating habits also include eating meat and potatoes at the same time and an antagonism is set up which is mutually hindering to both protein and to carbohydrate digestion, Dr. Hay recommended that each meal should take but one variety of food. One meal should consist of proteins, another of carbohydrate and fats, and thus hindrance is reduced to a minimum. The results of his system have been very satisfactory.

However even on a most rational diet the problem of elimination of waste products may be difficult as the result of some old typhoid fever infection or the obstructive tendencies of adhesions following an old appendicitis. The subject requires deep study in many an invalid therefore.

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 an invalid. 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 those 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 envolvement 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 paralyse 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 putrefaction 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 it 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.

Avoidance of coffee, chocolate, meats and even teas with high tannic acid content are rational adjuncts. The use of fruit juices freshly prepared and a solution of *calcium carbonate, potassium and sodium carbonates,* in small amount afterward to neutralize the excess acidity of the fruit is rational. After amines have poisoned the bowel wall, the musculature may be depleted chemically so as to not produce the energy required for the work of moving the bolus along or forming secretions. In this situation the trace elements may be a distinct help, as the *homeopathic 12x, of copper sulphate,* cobalt, tin, zinc, and manganese. At times the 30x tablets work better. They dynamize the oxidation catalysis in many directions and help the energy production required for muscle activity and secretion by the intestinal tract. Such electron transfer agents serve oxidations.

Constipation is in large measure due to liver insufficiency, and this is recognized in one of the favored treatments with bile salts, Taurocholic acid and Glycocholic acid salts. After liver shock by various chemicals one meets in some laboratories, or by virus infections, the support of liver function is needed, and the extract of its detoxicating principle is of value. Several are on the market that contain some of the bile salts and these help the intestinal function when the liver fails to respond to ordinary catharsis.

The carbonyl catalysts improve liver function immediately in most instances. This is seen in the efficiency of bowel emptying, the improved color of the skin and sclera. The intestinal musculature no doubt shares the improvement in function, as do other tissues of the body.

Finally muscular work and lack of worry and plenty of sleep, and even the after dinner relaxation and nap may be essential. The exercises given in army training are all good, and especially the crouching and the hopping butterfly affair do good when all other measures are not sufficient alone. Gen-

eral directions can only be given here as each case must be studied on its own merits and needs. Practically every case in this category requires pepsin-pancreatin remedies with each meal.

Fads

Fads in diet have their value. They teach us the meaning of error on a large scale. Surgery has its same fads. Appendectomies were a bonanza a few decades ago. The escape from gastro-intestinal and other diseases followed in grateful numbers. This fad should never have been discouraged. Ovaries are easy to remove. Healthy ovaries were taken out by the bushel, and race propagation was injured in geometric proportions. The victims were converted into chronic invalids, predisposed to cancer, and made fat in an ugly way. Their lives were ruined and most of the surgeons knew it. Yet it taught much in metabolism that is useful today. Then the draining of the gall bladder was presented to the public. What was done was to collect the bile that had already entered the intestine. No catheter could possibly enter the common bile duct when introduced through the patient's mouth. Still it gave useful information as to the alkalinity of the duodenal secretion with which the bile had mixed.

From this fad much was learned about the emptying habits of the gallbladder. We even thereby changed our old notions as to how this organ behaved. It was learned that the contractions of the caecal end of the colon influenced the gall-bladder emptying and the "muscle sense" which served the digestive needs, could proceed without intervention of nerves. The whole interaction is so finely adjusted and incomprehensible even today when we consider the digestive system as a unit, one must marvel at the wisdom which created the adaptability that performs so wonderfully minute after minute whether we respect it or not, and while we have no inkling of what is being done for us. Therefore when the physician requests a little attention to diet as a situation may demand, the patient should not revolt, but be grateful for the knowledge he is putting into use.

CARCINOGENIC INJURIES

Physical and Chemical

Almost twenty years ago the International Cancer Congress held at London received the proposal from certain leaders in cancer research, that the industrial carcinogens should be described and exposed so that the death toll from that source could be stopped. However certain industrial interests were represented in the membership whose fiscal policies demanded that the subject should not be even touched. Since the death rate from cancer is becoming so very serious, leading clinicians like Pullen and Ochsner are adding to the warnings of Ewing regarding the practical carcinogenic influences, as the rough physical examination, the biopsy, and cigarette smoking. To take a biopsy is very much easier than studying the clinical facts at hand and correlating them with the physical findings so as to arrive at a surer diagnosis by harmless and instructive means. "Instructive means" will reveal the facts of the pathogenesis which show how the disease developed and what its initiating factors were, and hence what should be done to combat the very foundation of the trouble. One knows then too, how to estimate the recovery progress under treatment from the basic facts.

For a long time the biopsy in the diagnosis of cancer was claimed to be harmless and not likely to disseminate the disease. At the 1954 International Cancer Congress held at Rio de Janeiro, it was finally admitted that the biopsy was harmful and dangerous in spreading cancer. When a physician knows his gross pathology he can regularly diagnose cancer without the aid of the microscope in any case that is established, for the infiltrations and metastases are evident as well as the characteristics of the tumor. The greatest diagnostic authority, James Ewing stated that "the resort to the biopsy was an admission of lack of knowledge." This of course could be due to insufficient information in any particular case, or it could be the inability to interpret the information that stared one right in the face. At any rate modern diagnosticians with courage are breaking loose from the old surgical dictum that the pathologist must make the diagnosis for the physician,

This is fine progress of course since the *pathologist* is an *autopsy* expert and the physician should be an expert on the manifestations of life, both normal and abnormal.

In the very early accessible forms of cancer as in the breast or a lymph node in the neck, groin or axilla, the whole growth that is visible can be removed and sectioned for the microscope. There is an advantage in that the cancer cell groups that are cut are the long strings or sheets that are invisible and so much of the neoplastic cells are not shoved into the lymph spaces as when the incision is made into the tumor itself. Such early growths may appear easy to remove fully, but in a matter of weeks one knows differently, and the microscopic study predicts the events that are on the way. Complete removal of the breast may indeed get the rest, but one cannot count on it as from 15% to 30% of even the smallest breast tumors, as one centimeter in diameter, are found to have spread to the inside of the chest before the operation was done, so the skill and diligence of the surgeon is spent with the odds against success.

After the characteristics of malignancy are established, the greatest care should be used to not injure the tumor or its surrounding tissue. Pressure will shoot malignant cells into the lymph and blood streams and make the disease general, so as to weaken bones till they fracture and fill the lungs so the oxygen supply is cut down. This is indeed a most unfortunate event, as the recovery depends primarily on the chemistry of oxygen as we have demonstrated above. After a growth has so developed as to distort the shape of the breast or of the viscus it invades, one can make a diagnosis from its changes in shape with change of the position of the arms and the way the breast hangs on bending forward or to the side as the result of the attachments made by the infiltrations. To make the diagnosis sure even without touching the patient additional information from the history may prove sufficient. But the texture revealed by gentle palpation will disclose the nature of the infiltrations and of the metastases, making the diagnosis firm without a biopsy. From these data in the gross pathology, the microscopic

characteristics can be described by one who is expert by experience.

The initiation of neoplastic change in a tissue as we showed, depends not only upon the toxin or virus that enters the neoplastic act, but upon the injury to the deeper circulation that supplies the tissue. Thus a fairly wide source of blood supply should receive obstruction so that there is a central area of anoxia established where the malignant change can get going. We called attention to this fact in "Cancer and Its Allied Diseases" in 1926, and the fact is now beginning to receive confirmation. Hence the blow with the broom handle or the heavy object that injures the breast causes the anoxic area where virus and toxin can go to work. But when the tumor is already started the blow need not be more than a moderate touch or squeeze or pull. The anoxic area is already there and rupture of the very fragile capillaries causes an infarction which gives the neoplastic agent the balance of power so to speak and it can then run away without any hope of natural control. I have seen young women with lumps no larger than an English walnut, that an expert would diagnose as absolutely malignant, make the rounds of a few surgeons for their opinions and after each had squeezed and tugged on it to his satisfaction the patient in each instance became afraid to return for the operation and so in eight weeks instead of a walnut she had the breast eaten off and the whole chest including the other breast involved with cancer and the lungs so full, she could not recite the A.B.C.'s without fighting for breath between every letter that was spoken. These surgeons were in the game for decades and should have known better, and they did. The danger one faces in looking for advice is another phase of the cancer problem—a personal one however.

A woman must protect her breasts from tight corsets, and from any injury or pressure that interferes with the circulation. If a blow is sustained the part should be immediately very gently massaged, and an arnica tincture or Witch-Hazel applied. Warm packs, not hot however, may be applied but not so as to produce pressure. The bowels should get a good cathartic of some salt and not some mixture of aloes, cascara or other

irritant—"Milk of Magnesia" in generous amount so a complete evacuation is had that keeps up until the bowel is empty. At the same time the magnesia gives an alkaline reaction that stops the toxic amine production. There should be a pure fruit and vegetable diet for a month or two, and no peas, lentiles or any form of animal food admitted, no tomatoes, perhaps. Vitamin C, as in berries, apples, etc., or taken in crystaline form with riboflavin, niacin and thiamin. Thus the factors that would settle into a malignancy have been given a battle against which they will very likely not win. The woman should never smoke, and if an injury should come, any carcinogenic agent as *smoke, coffee, burned fats,* etc. are to be eliminated.

It is also necessary to protect the body chemistry against the sulphides, as the sulphidryl group readily adds to the double bonds of conjugated systems of carbonyl and ethylene, and thus inactivates the tissue oxidations somewhat similarly to the cyanides. Sulphide injures the oxidation catalysts, and hemoglobin as well, and thus blocks the transport of oxygen. Sulphides in drinking water, or developed in the intestine by bacterial action on proteins of animal origin antagonize the defenses against cancer and all other diseases. The importance of pure water and correctly selected foods should be plain. Coffee owes its nice flavor to the sulphidryl group in an aromatic complex. It causes arteriole spasms and necrotic degenerations in the stomach wall. One can drink warm water flavored with molasses and fresh cream, and find this substitute to serve very nicely.

Hot drinks cook the inside of the stomach and oesophagus cause scar tissue formation that ruins the vascular supply. This gives the neoplastic agent its most favorable chance. So first test what is eaten or drunk with the outside of the lips. Here the heat and cold fibers are present, whereas in the mouth they are almost absent. Trial will show. If the outside of the lips find the temperature good, one can go ahead and swallow.

THE SELECTION OF THE CASE

It is evident that our clinical tests or demonstrations have been made on cases that are classified as incurable. It is logical that the most basic catalysis in vital chemistry should

be investigated in such cases. However there are secondary changes in advanced cases of any disease that will determine the outcome, even though the basic fault has been corrected by the treatment reagent. So an important part of the analysis of the case should be to determine the presence of such factors that could bring defeat, even though the fundamental pathology has been corrected. Such questions as the following have arisen. A patient of 55 years of age has a history of 15 years of cardiac insufficiency, with several serious infarctions. The aorta is dilated and elongated so as to cause considerable cough, and some difficulty in swallowing. The effects of the anurismal pressure on the trachea and oesophagus are pictured in the radiograph, and the presence of considerable thrombosis is also indicated within the aorta. Areas of calcification of the wall and its weakening are evident. The patient is under sedation to control the cough. The auricular beat is too frequent for counting running well over 200 per minute, and the ventricular beat is 97, irregular and weak. There is some air-hunger. There are other evidences of a depleted survival factor, a destructive lesion on the foot, diagnosed as cancer for example, and excessive adiposity. The question in such a case is would the improvement in the oxidations that is necessary to dispose of the toxins that brought on the vascular lesions give the myocardium such a boost that the systole would be strong enough to rupture the aorta, or loosen up some thrombotic material and cause a fatal embolism? We have seen it happen. The danger was recognized and explained to the family, but death was inevitable and expected very soon, and the desire was to have the treatment even though it was evident that several months would be required to heal the aortic lesion enough to withstand a good ventricular beat, and the indications were that the accident must come before that.

Another matter important to the selection of the case and the guess at a prognosis is the patient's ability to use enough oxygen not only for his ordinary needs but also for the increased demands for oxygen required by the recovery process. When a patient is in an oxygen tent before the treatment is given, the chances are that the vital processes are already suffering

from hypoxia, and the causes must be investigated. The lungs may not be able to take in enough air for ordinary use. This may be due to pleuritic effusion, pulmonary infarction, bronchial obstruction, presence of tumor tissue, hemorrhage into the air space, etc. Then there may be obstruction to the circulation that should carry oxygen laden blood to the tissues, as by thrombosis, tumor pressure, etc. The blood itself may not be able to carry oxygen in sufficient amount, due to hemoglobin failure, weak heart action, etc., or the tissues may not be able to accept the oxygen as when their colloids are gelated. Then even if oxygen is received in good amount there may be an absence of oxidation catalysts in the amounts required. Any of these defects could prevent the recovery process from getting a sufficient start to reach "first base." The prognosis is bad in all such cases. If the hypoxia is due to great displacement or by pressure by a tumor that has been irradiated, and the mediastinum is involved, myocardial injury is expected, and the chances for success are about zero. Cases of that type could be cited. It simply needs to be remembered that since the success of the treatment depends upon the improved use of oxygen, its accessibility should be assured at the very start. Patients have done well though the hemoglobin was less than thirty percent, but these were uterus cases where the tumor mass was not enormous and a good deal of the anemia was due to hemorrhage, and as soon as the treatment was given the bleeding stopped. Cases with enormous chest involvement that require the oxygen tent offer a poor prognosis.

Irradiation, if heavy, will never permit a real recovery. It is given because it blocks the traces of oxidizing power a cancer cell still has, so the cell dies or is badly inactivated for a while. The cells that survive rally in time however and are more malignant and quite resistant to the irradiations. In the meantime the irradiation reduces or destroys the oxidizing power of the normal cells and reduces them to the cancer class in this respect. They lose their protective carbonyl group activity and are open to integration with any carcinogen that comes along. Besides the irradiation has a general toxic effect that injures the bone marrow and reduces red cell production, inac-

tivates the ultra cell structure that conducts the highest type of oxidations that determine cell differentiation, and hereditary transmissions that provide for the coordinations of function for the tissue economy, and for race advancement and survival. Thus the offspring of radiologists are defectives ten to a hundred times as often as the children of general practitioners. And the radiologist himself shows the injurious effects of his frequent but minute exposures in a decade or two.

Necrotic bone produced by the irradiations is a secondary center for further irradiation that destroys more bone and induces malignant change in the soft tissues near-by. As the bone dies it becomes radioactive too and carries the destructive process forward without termination. When bones of the head are so affected the brain receives fatal doses of irradiations and edema is induced that may end life very suddenly, or with a prolonged mental torture that should be mentioned because of its religious significance. Occasionally we have had cases of cancer of the lower jaw with the adjacent soft tissues of the face and neck envolved, where we have observed after careful attention, that the necrotic bone sequestrated and eliminated itself in several pieces, leaving the soft tissues apparently healthy and healed. No further bone necrosis took place in the period of observation (5 to 8 years), but the ultimate outcome could have been a disaster in each such case. Careful watching was necessary and the carbonyl catalysts were repeated at properly chosen intervals. This is rather difficult too since the irradiation effects mask the reactions, and one scarcely knows where he is at in the recovery process, so that the most careful conduct of the case includes a lot of careful guessing. Many histories and photographs could be presented to illustrate the different phases of this chapter, but nothing more would be achieved than this summary presents.

There is a phase to irradiation that is never mentioned. The radiologist of the past decade or two used to report in the authoritative Radiological Journals his cure of high percentage of cases. He reported on the first shock effect of the irradiation which looked like improvement. The later effects, the real results were in these very cases very different from what he

reported. He probably never saw the cases again and did not know their fates. But in addition to the situations that are mentioned above, another is to be observed which the radiologist watched with alarm, but such cases are not mentioned either so far as I know. They should be reported as part of a general biological effect much like the adaptation of a germ or virus to an antibiotic agent. The effect must be distinguished from the production of a new neoplasm of high resistance to irradiation right in the area treated. Most such new growths are bone sarcomas with rapid fatality. But the change we have in mind is the conversion of a neoplasm itself from one, that seemed to disappear under the shock of irradiation, into a tumor that thrived on further doses of irradiations. The energy of the treatment went into the process of neoplasia. This is one clinical observation that argued for the interpretation of the energy transfer into the mitotic chemical reactions as a photochemical affair. Normally we suggested that the energy of exothermic reaction going on in the field were picked up by the carcinogen and transferred to the chemical processes of mitosis at a rate optimum for its acceptance and stimulation of the neoplastic chemistry (Natural Immunity, Koch, 1936). This is however, an interatomic affair. It seemed to the writer that the intra-atomic damage of irradiation resulted in secondary changes at the inter-atomic level where energy was let loose that the carcinogen could make use of in this way, that is by its fluorescence. Later, the British and French cancerologists measured the fluorescence they identified as responsible for the carcinogenic property in coal tar and synthetic products. The writer was criticized by the A.M.A. Journal for stating that the cause was an organism, invisible, and carrying many of the properties of the syphilis germ. Even though there was no microscope of anything like the electronic type to identify the germ, yet the clinical facts proved the infectious nature of cancer, and we hold that the organism was responsible through a ligation with the host cell that permitted the transfer of energy from exergonic processes aroused there, into the mitotic processes as a continuing change. Today it is demonstrated that a virus that can cause cancer is a few thousand times more re-

sistant to irradiation than normal or cancer cells. So at least it has a chance to carry the process from dying cancer cells to living normal cells, or to cells already changed to cancer cells. The disintegration of highly built structures of the cancer cell whether the result of irradiation or of necrobiotic changes caused by a carcinogen of synthetic type, yields energy and this is what we hold the carcinogen transferred at a rate optimum to the neoplastic job. The disintegrations of the tissues in syphilis could easily lend themselves thereto, and it is hard to tell where the syphilitic change left off and the carcinogenic change started. The Journal of the A.M.A. should have been a little more lenient, and thus would have avoided the disrepute their hasty criticisms have merited. Further they criticized thirty years ago when there was no biochemical advice to direct them, and no biochemical theory to support or oppose. Cancer matters are far too serious for such extravagances.

As experience grows with this therapy other interferences that could prevent success will be learned, and the status of the patient before treatment of interfering types with that afterward will be a guide of some value.

CHAPTER XV

THE SOCIOLOGICAL SITUATION

The high incidence of cancer among babies is a subject for concern. In our book, Cancer and Its Allied Diseases of 1926, we predicted the time when cancer would predominate among those who had not reached puberty in a way that would threaten race extinction. Today we see cancer as one of the most frequent causes of death in children, whereas forty years ago one saw a case of leukemia or of hypernephroma only rarely and it was the sole neoplastic disease seen in children so far as I can recall aside from a rare retinoblastoma. Today children and young adults are the victims of rapidly fatal neoplastic disease in many of its forms. Since the oxidation mechanism has demonstrated its protective and curative features, the cause for the breakdown before the rapidly advancing cancer must be of such nature that it inactivates the oxidations. Smoking, gasoline and Diesel engine exhausts, denatured foods, and not enough physical work, plus the exhaustions of worry and the social fatigues, wholesale injections with every sort of germ poison or drugs, some of which are able to increase the incidence of Infantile Paralysis four-fold, the inability to obtain Homeopathic medication, and the practice of aspirin dosing for every little complaint, constipation, too much animal food and white bread, plus the increase in viral disease, create an environment in the home which favors cancer. Add to this the industrial contacts with carcinogens, the tar dust and motor exhausts of the roads, the tobacco smoke from arsenic disinfected tobacco, and one lives constantly in a carcinogenic environment. Any one of the above mentioned affairs could prepare the tissue cells for a carcinogenic virus, and even in the wide open country a new born baby of robust parents may not find it possible to escape. The following case illustrates. It is given in the form of the mother's affidavit since it is subject also for analysis of the professional situation as we have experienced it. The baby was cured by one injection of our carbonyl catalysts.

The steady and rapid increase in the incidence and mortality rates of cancer are concomitant with two other catastrophies, the general physical and the general moral degradation of the races. These are also interdependent. They concern our subject because the physical degeneration is largely due to improper foods, and the denatured state of those that are eaten. Interstate commerce in foods poisoned by *selenium,* which lowers the resistance to cancer is not stopped by the Food and Drug Administration, but seems to be desired like the poisoning of the water of the nation with *fluoride.* These poisons not only diminish the physical ability to withstand cancer, but they also cancel out the reproductive capacities, and so tend toward race extinction. They destroy the *Survival Factor* in two different types of attack. When either poison becomes a part of the daily ration, it cannot be antidoted, but accumulates in its deadly effects.

The attempt to meet disease by prophylactic vaccination to the exclusion of curative measures is more than a matter of poor judgment. It too is a moral issue, especially when there is no vaccine known to be efficient against the diseases in question. The two professional instances mentioned below, show that the curative treatment of cancer and of infantile paralysis are opposed. The doctor, who cured the infant of cancer discussed below, was viciously persecuted by the local medical politicians, who should have honored him for his victory. Under the leadership of the head of the State Board of Medical Registration, they even threatened to take away his license to practice, if he continued to use the treatment that cured the little victim. Their threats failed. The reports on the Infantile Paralysis case that follows needs no further comment. Both are instances of the failure of the natural instinct to protect the fatally sick dependants. Yet they are committed and tolerated by our supposedly superior class of Americans.

Case 1

CANCER OF THE LIVER

The Affidavit

"To whom it may concern:

In order to put on record the facts we know concerning the illness, treatment and recovery of our daughter, Judnita McWhorter, here in after referred to as just Judy, we make the following statement of our own will and accord, without promise of or hope of any remuneration, and having previously received no remuneration of any kind.

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 who was a doctor in good standing, a member of the American Medical Association, and a man whom we trusted and still hold in high esteem, 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 that 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 made. 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 that 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.

For some days prior to this time Mr. Joseph O. Noah, a

*Not doubt Roentgenologist is meant here.

neighbor and old friend of Mrs. McWhorter and her family, had been advocating the use of the treatment offered by Dr. William Fredrick Koch . . . None of us had much confidence in his treatment.

When our doctor was consulted he assured us it was useless. He said he would not give it to his own child under the same circumstances, and that it would be an unnecessary and useless infliction of pain on the patient. He also made the statement that he would believe in the treatment if he could see one case recover from the use of it where a biopsy showed positive malignancy.

It was while we were considering this treatment that Time (magazine) published its defamatory article about Doctor Koch in the issue of September 6, 1948. This article was brought to our attention by both our physician and Mr. Noah. We found it very hard to take Dr. Koch's treatment seriously in the face of such criticism. Nevertheless when we had no other hope and since Mr. Noah made it possible for us to take the treatment without immediate cost to us, we decided to try it.

Dr. Koch's therapy was given by Dr. N. T. Mulloy 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. Noah took a series of color pictures showing her progress. Previously he had taken two pictures at six weeks of age and before diagnosis of cancer. This 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. Mulloy expressed no hope of securing a recovery as he thought the case was too far advanced.

Within ten days after 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, some-

times 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.

During the early days of the recovery process Mrs. McWhorter reported to the doctor who had previously cared for Judy and who had advised against the Koch treatment, that she was apparently getting better. He admonished the mother not to entertain false hopes. He said that it was impossible for a dose of any chemical to "destroy" such a large growth.

On the other hand, Mr. Noah stated that the doctor's remarks showed that he had no conception of how the treatment was to work. He said one might as well say that a small match could not start a large fire and destroy a forest.

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 trace of the growth remained.

Later, about May 12, 1949 I had her examined by a doctor in Paris, Texas. (Mrs. McWhorter told the doctor to make a thorough examination for trouble of any kind.) 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.

Mrs. McWhorter states that a more surprised group of doctors would be hard to find, when they first saw a rosy

healthy child rolled out before them after having read a clinical summary of her case.

An account of this meeting with a picture of Judy and her mother was published in the Fort Worth *Star-Telegram.* The piece was headed: *"Doctors Convinced That Little Judy Overcame Cancer Ailment Herself."*

This, in spite of the fact that all concerned knew the Dr. Koch treatment had been given and that we gave it full credit for bringing about the baby's recovery. The only excuse we can offer for this is that undue excitement might have been raised by a publication of the true facts.

On February 18, 1950 both parents and Judy attended a meeting of physicians and others at Tampa, Florida. Here Judy was again shown to a 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 Dr. Koch treatment and recovering from cancer.

Witness our signatures.

Mr. O. McWhorter Jr., Father

Mrs. Otis McWhorter Jr., Mother

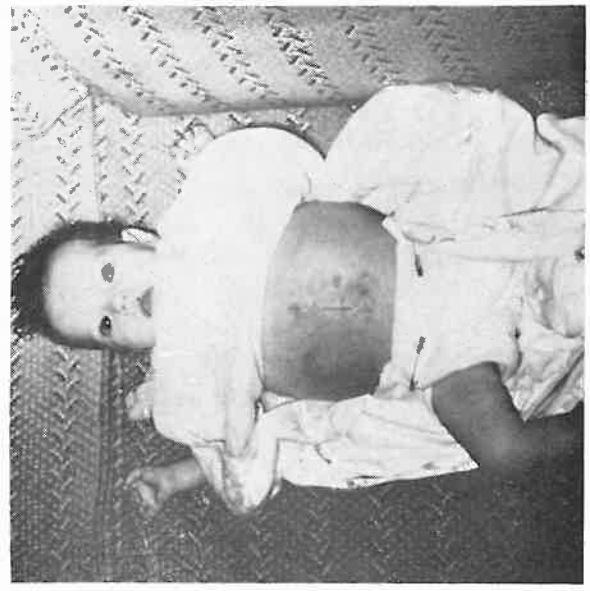
State of Texas . . . County of Parker . . . Sworn and subscribed to before me this 28th day of June, 1950.

Jim Bob Nation

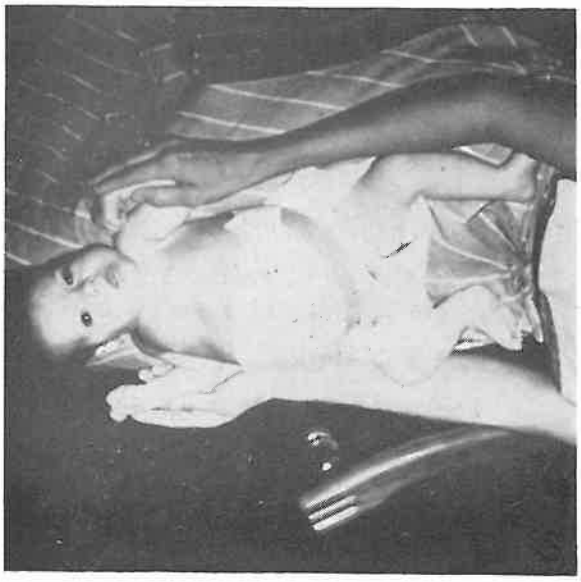
Parker County, Texas
Notary.

The Photographs

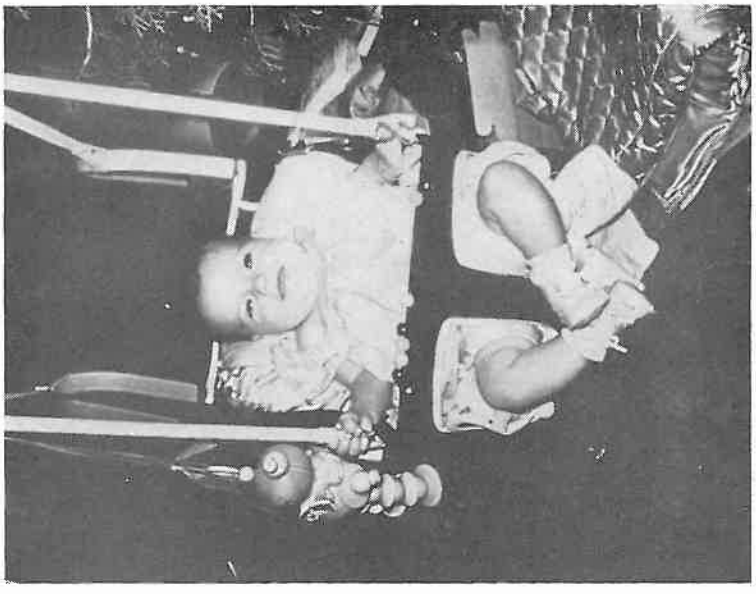
The history of the cure of this case is pretty well shown by the following series of photographs. Latest reports indicate that her health remains good seven years after treatment.



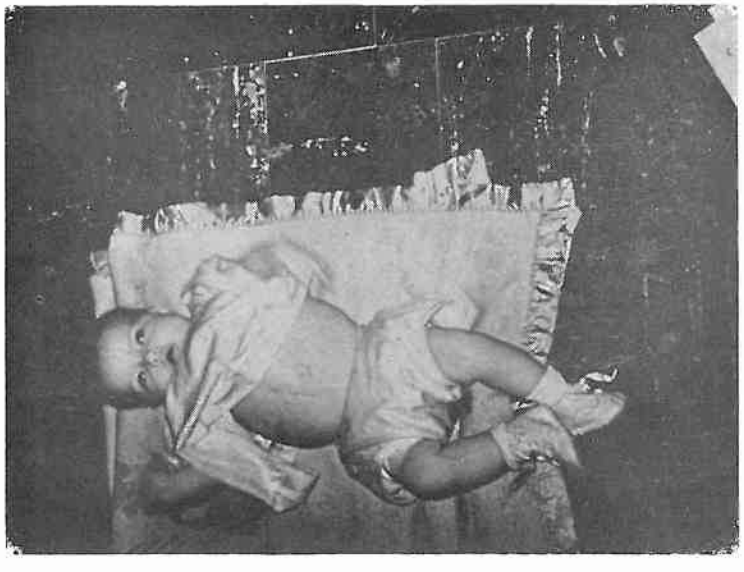
No. II. Taken at the time of the treatment, September 18, 1948.



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

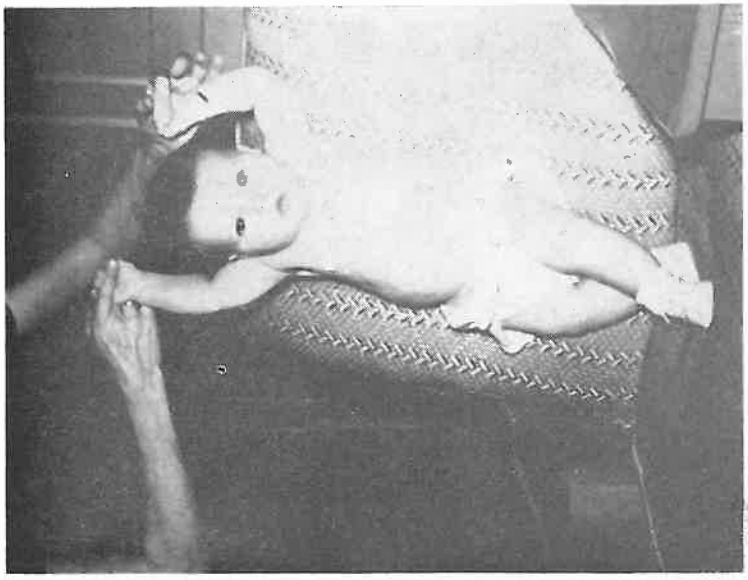


No. IV, Taken a few weeks later.



No. III, Taken several weeks after treatment.

No. VI, Taken a year later.



No. V, Six months after treatment.

No. VII, Taken a few years later.

No. VIII, taken September 1956, eight years after treatment.

When the International Cancer Conference, held at Lake Mohonk, N. Y. in 1926, succeeded in excluding the discussion of new progressive treatments for cancer from the program, and when they negotiated the "division of the business" between the surgeon and the x-ray and radium specialists, they in reality conspired against the family doctor so far as his chances to serve and to be remunerated were concerned. Further they imposed the obligation of referring the suspected cancer cases immdiately to the surgeon and the radiologist, even though they well knew that neither had accomplished more than a minor help to cancer victims. The great boasts about the successes of radiology and surgery that flooded the literature and the press then and ever since are seen from our perspective to have been fraudulent, and heartless. That the conspiracy to hold the cancer business in the hands of the three specialties was well organized and diligently prosecuted is seen in the instance mentioned here, for if the public had been allowed to learn the truth about Judy's cure by an accurate harmless therapy, the family doctor would have been forced to carry out his obligation of taking care of his clientel himself. Further in order to block such a "financial catastrophe" the conspirators had the State Board of Medical Registration lined up to take Dr. Mulloy's license away for accomplishing this service. The instance that follows shows how the same conspiracy against the family doctor and his service applies to the profiteering upon and blocking of real progress in the cure of paralytic Infantile Paralysis.

This simply means that if the family doctor is desirous of carrying out his obligation of using his best judgement in the care of his patient, he must organize. Just as the specialists have their societies where they concoct their business procedures, so the family doctor will have to do the same to hold and protect his rights as a physician. Indeed he must organize to fight to regain them, and also to hold the right to use the hospitals from which the specialists are now attempting to exclude him. Unless he organizes, he will be forced into the degraded position of a referring agency for the specialist. If he organizes, he can then practice medicine in line with its time honored precepts, and he will be able to encourage progress in the conquest

of cancer and Infantile paralysis, and enjoy the satisfaction of being a physician and be loved and respected in his community in the way to which he is entitled. The leaders of such important affairs as the American Cancer Society and the National Institute for Cancer, will also have to be men of honesty, training and ability to fulfill their important obligations, instead of agents of deception that perpetuate the 1926 conspiracy.

Case 2.

POLIOMYELITIS

CO-ED'S SWIFT RECOVERY MYSTERY EVEN TO DOCTOR

Reprint
Toronto Daily Star: Mon., Aug. 22, 1949

By HAROLD GREER
Star Staff Correspondent

Columbus, O., Aug. 22—Golden-haired Mary Lou B . . ., a 19-year-old, Ohio State university student here, has become the bewildered recipient of a nation's scrutiny. Four days after she was stricken by what her doctor said was poliomyelitis, she was out of bed; 10 days later she was back at her classes.

The physician who used . . . (carbonyl catalyst) in his treatment and who said the girl's recovery was miraculously swift" after he made one injection, has left on vacation, a worried and confused man. The drug he discovered later, is blacklisted by the American Medical Association and was developed by a doctor who has fought the association in court and out for many years.

The manufacturers of the drug . . . . have declared emphatically they have never made any claims for (this drug), they have not advertised it, and that the league is a non-profit organization composed of ministers and laymen, numbering many doctors.

Have Authentic Cases

The doctor said he had the drug on hand because the Detroit organization told him it had on file "a few authenticated cases where (the drug) had been used successfully." He con-

tacted the league originally because he had lost two herds of cattle on his suburban farm from Bang's disease—the same disease which is called undulant fever in humans—and had heard the drug was successful in fighting it. It saved a third herd.

(This drug) is described as an oxidation catalyst—an agent which increases the oxidation process of the body, one of the prime processes of life. It was developed by Dr. William F. Koch, who has been connected with its manufacture for more than thirty years.

These are the principals on whom the curtain went up a few days ago, when a newspaper here made front page news of Miss B . . .'s recovery. Its story was carried for the first edition only. A wave of protest from the medical profession —to the effect that the drug does not have the approval of the A.M.A., that it was not a cure for polio, and would therefore raise the hopes of victims of the dread disease and their relatives without justification—induced the editors to kill the story in later editions.

Carried on Wire Services

But it was too late to stop its spread across the country via the press wire services. Miss B . . . and the doctor, despite nondisclosure of his name, have been flooded with inquiries from a polio-conscious nation which has not yet forgotten the controversy which waged over Sister Elizabeth Kenny's method for alleviating the effects of the crippling paralysis which always accompanies polio in some degree.

For Mary Lou B . . ., as she goes back to her classes without any discernible ill effect . . . (it) is simply a name for something she believes saved her from being a cripple and, perhaps, saved her life. She was dismayed to learn the drug is unknown by the medical profession's rank and file and is frowned upon by its leaders.

She answers: "It worked for me."

Mrs. Laura B . . ., her mother, a business woman who owns her own fashion shop, agrees. She said: "It was a miracle."

For her doctor, a successful 45-year-old general practition-

er, who has been the family physician for many years, the use of (this drug) may mean banishment to medical Siberia. Although his identity is generally known among his associates, the thing he fears most is publication of his name. He wants to avoid any suggestion he is advocating the drug as a "cure-all" for polio.

Delayed Reporting

He admitted he delayed reporting he had a patient with a communicable disease to the city health authorities until it was necessary to wait over the week-end until the following Monday. He did so, he said, in order to try (this drug), and has since squared himself with the board of health.

He described his attitude toward the apparent success of the drug as similar to that of a man turning on a light switch for the first time. "If the light goes on, all I can say is simply that I turned the switch and the light went on," he said. "If I can turn the switch dozens of times and every time the light goes on, then I am safe in saying that when one turns the switch, the light may be expected to go on."

So he is a long way from believing (the drug) is a cure for polio, he emphasized. But he does believe it should be given further trial. For this reason, when news of Miss B . . .'s recovery leaked out, he asked newspapers to withhold the story for 24 hours while he endeavored to get permission from City Isolation hospital authorities to try the drug on more polio cases. Permission was denied, he said. It was then he learned the drug simply did not have the stated approval of the A.M.A. but was actually blacklisted.

Wife Sick With Anxiety

"I don't want to be a martyr," he said. "My wife, who is a nurse and familiar with the way the powers-that-be operate, is almost sick with anxiety. She thinks I stand a good chance of having my reputation ruined and our means of livelihood destroyed."

At the same time, he added, he was so impressed by the results of the drug in this one case that he would not hesitate to try it again should one of his own children be stricken by polio. He has one son and two daughters.

"I saw something happen which I believe to be cause and effect," he explained. "Mary Lou B . . . and her mother are good friends of mine. I have watched her grow up and have seen her through all the childhood ailments.

"To my knowledge, the symptoms she had could have indicated nothing else but polio. It began with a headache and a sore throat and coryza. Her right leg gave way under her. She described it as a "queer feeling" in her leg and said there was a tingling in her right hand.

Leg Was Paralyzed

"I saw her first in the morning; by evening the leg was completely paralyzed; she couldn't move a toe. There was considerable pain. She still had a weakness in the wrist, but it was not developing. I then administered two c.c.'s of (this drug) intra-muscularly. Later that evening, the paralysis of the leg was complete.

"But in the morning I found I could move the leg myself although she had no voluntary control. Some of the stiffness had gone. She complained of a severe headache.

"In the afternoon she had a chill with a flare-up of fever. This was the result of the headache building up. The manufacturers of the drug told me both the headache and the chill could be expected.

"A short time later, I telephoned Mrs. B . . . . She said Mary Lou's leg was beginning to move and sensation was returning. I got over as soon as I could and found it to be true. She began to improve very rapidly after that and was able to go down to dinner the next day. That was a week ago yesterday."

These two cases illustrate the extent to which "Official Medicine" has gone to deny and oppose basic truth. The Congress of the United States, which has spent so much time and money investigating crime, labor racketeering, un-American activities and the like, should follow Senator Tobey's precedent and carry on a full investigation of the field of American medicine.

CHAPTER XVI

SUMMARY AND CONCLUSION

The dominant idea in medicine thirty years ago when we first presented our thesis was *specificity,* and any remedy that claimed utility in more than one disease was shunned as unscientific quackery, unheard of, and impossible. Our offer of a least common denominator in pathogenesis and treatment with curative results in a wide field of virus and bacterial infection, in cancer, and allergy, was beyond the comprehension of the best trained physician of that time.

Today the antibiotics and ketosteroids are opening the path that we tried to open years ago, but now it is done with the aid of the drug industry, whereas we tried it with the opposition of that power lined up with the state of mind of that day. Specificity belonged to the acquired immunity of Ehrlich, and Pasteur. Our field of the Natural Immunity, a metabolic affair was given no place in the presence of such theories as amboceptors, haptophores, etc. But today the antibiotic is rated according to the breadth of field it is able to conquer, and the idea of the least common denominator will finally be received.

Our basis it must be recalled is the conclusion that Nature was made as a harmonious complex of reciprocals, and that this status could be somewhat restored when the basic fault in metabolism was recognized. That has been our research, and the progress made can be judged by the results reported here. The restoration to normal offers nothing to mutate against. It can only win. We conclude it is from molecular oxygen that all life we are concerned with takes its existence directly, and certain forms of anaerobic germ life depend upon indirectly. The opportunity for molecular oxygen to function was definitely the free radical brought forth by dehydrogenation, or by double bond cleavage and one pole taken by the attracted atom while the other lay open as a free radical. Then when molecular oxygen was present the energy production of vital pro-

cesses could go on, and if it was not present, then the abnormal additions and polymerizations that constitute the basis for disease, as we explained here, were necessary events. The nature of the dehydrogenator, the carbonyl group as required for free radical production, and as the ultimate and intermediate product of free radical production, indicated a very significant reciprocity in metabolism, when oxygen is present. Next, the amine group which when activated in a series of gradations could serve to aid the carbonyl function or block it, had to be given due consideration, and the very convenient values of the azomethine double bond for both events had to be calculated into the living mechanism as they are the elements ever present in the field of cellular life. Thus from the simplest and most basic facts in biology the basic function of the carbonyl group in aerobic life is evident, and where its function is deficient it may be reestablished so far as our observations go, when catalytic amounts of highly active carbonyl groups are supplied.

Since virus is unable to perform this carbonyl function, it cannot initiate oxidation chains to supply its own energy for its vital processes and must integrate with the energy producing mechanism of an appropriate host cell to have its vital processes activated. Other more complex parasites possess carbonyl function in varying degrees of deficiency, and must rely on products of host cell metabolism that their carbonyl deficiency prevents them from producing. They thus accept soluble products from the medium into which these diffuse from the host cell, or within the host cell. No definite ligation is required with the host cell, other than proximity. On the other hand when virus enters a host cell, it breaks up into its component units (which are nucleoprotein monomeres so to speak) similarly to a depolymerization process, and the free radicals exposed make the unions with the host cell units, free radicals or double bonds. But where these are already occupied the virus amine group may condense with the host cell functional carbonyl groups. Separation would take place (See Diag. VII, Ch. III) leaving a carbonyl group terminal in the virus which has thereby lost its pathogenic amine group. Likewise high efficiency carbonyl therapy should separate the virus at a point

alpha to the double bond closest to any point of attachment, leaving a carbonyl group here also. The virus is no longer found, but it is suspected that it is able to initiate its own oxidation chains for energy production and be self supporting because of its newly acquired carbonyl group.

In the case of the more complex parasites as bacteria, we have some strong evidence that the reagents not only remove the cause of their parasitism, but contribute an autonomy whereby they become useful members of the great biological economy. Part of page 17 from the report of the Minister of Agriculture of British Columbia, Canada, to the Parliament in 1944 is reproduced here. It gives the bacterial counts on twenty-seven cows that were part of a total group of seventy-one cows that were reported on a year later. These seventy-one cows treated for mastitis presented 263 quarters affected with the disease out of a total of 284 quarters. No saleable milk was being obtained from these 263 quarters affected with mastitis at the time of treatment. Ten months later it was revealed that production of market milk had been restored to 256 quarters.

This table gives the bacterial count just before treatment and one week after treatment, duplicate samples being sent to two different laboratories. Seven of the cows show an increase in bacterial count and four of these showed an improvement in milk and udder condition, while the lesions were healing and the toxic effects in general were disappearing. Twenty of the cows showed improvement according to the bacterial count.

The 1945 reports stated: "A consistent result was a definite softening of the udder after treatment. The disappearance of fibrous tissue was noticed in a considerable number of cases. In no case was any other treatment used in conjunction with the Koch treatment."

Other experiments show that the hemolysins of Staphylococcus aureus disappeared and the bacteria produced toxins no longer. The calcium balance was restored, etc.

The remarkable reduction in bacteria is of interest, particularly in view of the fact that bacterial reduction was not claimed for the treatment which was simply that **dairy cows could be brought back by means of an injection to the condition that would permit them to produce market milk.**

The following table shows a favourable reduction in bacterial count in the majority of cases as between the first test and the second test made seven days later:—

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. 10	57,000	26,000
No. 11	110,000	1,000
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. The trade name of the drug used has been deleted.)

The increase in the bacteria after treatment instead of a sharp drop, as occurred in so many others shown in the table, appears to be parallel with the amount of tissue debris that has to be removed; and thus we conclude that the bacteria are now able to work through their newly attained carbonyl activity.

The physiological objectives of this research appear to have been attained therefore, against which no mutation is possible as no harm is done. Thus both host and pathogen are set back on a healthy, useful course by the physiological approach. The return of function is illustrated in the Poliomyelitis cases, the cancer and allergy cases, the bacterial infections in man, and in the animal experiments, especially those that have to do with infertility.

Infectious Basis of Neoplasia

The case histories were selected moreover to offer a wide enough range of data in order that one might draw a reasonable conclusion as to the mode of origin of a neoplasm. The case of Mrs. S . . ., who had lymphocytic lymphosarcoma of a very acute type, for example presents a recovery process that is very informing, especially in view of the case of Miss G. with the large myofibroma of the uterus. It is well known that malignant neoplasms originate in benign growths at times. Thus a benign epithelial papilloma or polyp may change to a malignant neoplasm of the same epithelia. On the other hand as in the case of Mrs. S . . . a highly malignant growth of one type of cell (lymphoblastic cells) may be the seat of a benign growth of connective tissue origin, a keloid. Here two types of neoplasm are observed which have different origins, yet the causative agent, the pathogen may be the same for both tumors. The data given by these cases is at least suggestive. A few experiments would be required to make it conclusive.

We may review the following in the Mrs. S . . . Case. There were several sets of pus infection, but right after the malignant neoplasms were absorbed, there was no reaction such as is observed to terminate a recovery course from cancer of the breast. For example, there were no acute flare-ups of an old focus of infection with fever, chills, pain and intense inflamation which subsides in a few days and with it the focus disappears. In the Mrs. S. case the malignant growth was gone for ten years before the terminal reaction took place. It showed up as a keloid that originated at the point of the biopsy and progressed slowly until an injection of diphenoquinone was given. Thereafter no significant reaction took place until the seventy second

week. The keloid swelled during this period and was acutely inflamed, and then subsided so that it became smaller and soft with an improvement in color and vascular circulation. From her seventy-second week, she has shown continuous improvement. More reactions may be required to accomplish its complete absorption.

In the Mrs. S . . . case, while the highly malignant neoplasm disappeared rapidly, this late appearing keloid is behaving like other fibroid tumors. The question is: Can the keloid be interpreted as an initial phase in the malignant disease, as is seen where a papilloma undergoes a malignant change, or are the two growths causatively unrelated? Our answer may be found in a taint of tuberculosis; for it is known that the tuberculosis poison sets up keloid responses in some people, and this poison also stimulates lymphomatous changes in the parenchyma of the lymph glands in some people as well.

The fact that the keloid appeared so late after the recovery from the malignant neoplasm does not discredit its simultaneous or earlier origin with reference to the latter. The connective tissues could have been saturated with the poison at the same time and as lymphocytic tissue is so highly reactive a quick response is natural, while the fibrous connective tissue with its chondroitin compounds would naturally be very slow to give a response even though an injury as the biopsy intervened to give it a boost. It was still over ten years after the tissues were cut before the keloid showed up. All cases of keloid that we have seen were remarkably slow and required not only the initiatory action of the tubercle poison, but also some injury as a burn or cut to set it into motion. Thus while different tissue elements were affected, each could have been acted upon by the pathogen at the same time and each responded according to its natural capacities. Then we may conclude that it is reasonable to have one and the same pathogen causing the two different effects without any genetic relationship between them.

As to the nature of the pathogen, one fact indicates that it is a virus which plays a role in the pathogenesity of the tubercle

bacillus. This virus was first identified by Oswaldo Cruz and Fontis of Brazil some decades ago. It is well known that when a tubercle germ goes non-pathogenic, it grows in filament form with branches like certain molds. In this stage, the rapid reproduction mediated by the virus is missing and thus the pathogenesity may be assumed to be due to the virus. In our review of the Mrs. S . . . case, we recall that the malignant phase of her disease responded in hours to the therapeutic reagent, while the keloid phase took months. The treatment given at first probably was used up quite completely by the lymphocytic cells since they are so highly reactive. The treatment given in 1955, many years after recovery, had no lymphocytic tissue to compete with the keloid tissue for its effect. The response of the keloid was in line with that of other benign fibroid tumors and other keloids we have treated in the past. The indication that the pathogen was a virus is further strengthened by the fact that when a virus integrates with a host cell it takes over the characteristics of the host cell. Thus in the lymphocytic tissue where oxidative exchanges are rapid when unhindered, this therapeutic reagent was followed by a rapid response; while in the connective tissue tumor with its chondroitin sulphuric acid fraction that blocks oxidation, the virus is protected from oxidation assault and the recovery response is slow indeed. Just as the pathogen may be the same in both phases of the disease, the break in the survival factor that permitted its integration with the two tissue elements was also the same and the correction of this break turned out to be the same attack on the virus; a favored dehydrogenation in the malignant phase and a hindered slow effect in the keloid phase. Both responses depended on the reactivity of the tissue elements concerned, though the pathogen was the same. Clinical observation may identify the nature of a pathogen therefore without the aid of the laboratory, or it may give support to the laboratory observation after it is made.

Suboxidation in Vascular Disease

A more detailed study of the role of fats in vascular diseases brings the conclusion that the more saturated fatty acids of the solid fats share etiological responsibility equal to that of

Cholesterol. In as much as the oxidation facility of the unsaturated more fluid fats makes them more readily disposed of, they of course do not remain to produce deposits in vascular walls. Here we have another support to our theory of the place of the free radical in the oxidation mechanism.

The unsaturated fatty acids offer a mobilized hydrogen atom alpha to each double bond, ethylenic or carbonyl, which prepares for dehydrogenation at these points. Free radical production and peroxide free radical formation where there is no interference with oxygen function results in cleavage into units presenting terminal carbonyl groups. These units undergo further oxidation by the same process. Of course the commercially saturated hydrogenated fats are least oxidizable. They should be studied with reference to the increase in their use as compared with the increase in vascular diseases to see how close a parallel there is.

Besides the fatty acids concerned and the short carbon chains that are built up to form cholesterol, all of which normally are fuels, the utilization of oxygen is the determining factor in the prevention of vascular disease. Here we have shown that the improved dehydrogenations of fuels and toxins, as produced by the reagents described, not only reverse the pathogenesis sharply but also secure protection for a time even though the patient slides back into the regime that contributed to the disease in the first place. Recurrence took place under such conditions five to ten years after treatment.

The prevention of coronary disease is then not only a matter of reducing nervous strain with its adrenalin increase in the blood and the consequential blocking of the oxygen supplies as well as contra-oxidative chemistry of the amine group, or even a matter of diet, but is a matter of maintaining high oxidation catalysis. The cardiologist should be able to measure the percentage influence of this factor with detailed attention to this point. Our own conclusion is that it is the chief factor and where the oxidation catalysis is adequate, no vascular disease can take place.

TOXIN INTEGRATIONS IN RHEUMATIC FEVER AND ARTHRITIS

The integration of toxin and host cell can be seen in the arthritides and in the acute rheumatic fever cases. In acute rheumatic fever the articulations may clear up, but the endocardium may carry the toxin for a lifetime as a chronic contracting fibrosis. In rheumatic endocarditis the fibrosis may be looked upon as a copolymerization of the toxin with the fibroblastic material. The same holds true for the articular changes in the chronic forms of arthritis. In both (A) the proliferative adhesive rheumatoid and (B) the degenerative atrophic osteo-arthritis, there are cartilagenous degenerations. In (A) there is granulomatous type fibrovascular overgrowth with fibrotic and oseous adhesions causing ankylosis. In (B) there are the cartilagenous degenerations and calcification. While the overgrowths cause an interlocking of the joint surfaces, there is no bone or fibrous adhesion to cause ankylosis as one finds in (A). However, there are cases that present both types of change. Thus while the degenerative changes due to the action of the toxin and the hypertrophic changes that inactivate the toxins are both present, there is enough clinical as well as pathological difference in the mode of toxic attack to show the etiological agents are different in both. The age factor, 50 years or over, in osteo-arthritis (B) points to a metabolic influence on the pathogen. Both (A) and (B) are progressively polyarthritic. The pathological changes are also similar in the Spondylitis of Marie-Strumpel.

The integration of toxin, whether of germ or metabolic origin, with the fibrogenic elements of the connective tissue, bone or cartilage and the consequent degenerations and hyperplasias soon comes to an end after the pathogen is oxidized away from its combination. The fibrotic portion undergoes a hydrolytic digestion and is removed. Hand in hand with this cleaning process the joint tissues undergo reconstruction to normal or a very close to normal structure. During this healing phase the joint is very tender and immobilized by swelling and muscle contraction. As soon as the reconstruction is completed, the joint is fit for good function. In severe poker spine where large

plates of calcification have infiltrated the muscle and fascia of the back and shoulders, these plates have been observed to dissolve away. Such cases have been seen to change from complete ankylosis of all joints to be able to climb stairs and go hunting after treatment.

The arthritis of acute rheumatic fever is purely toxic and inflamatory. It may be compared with the inflamatory changes in a soft tissue as in encephalitis, and in the quickness of recovery and return of function. Where tissue changes are degenerative and hyperplastic, the normalization process takes more time.

ACUTE RHEUMATIC FEVER**

Treated in collaboration with Dr. Wendell Hendricks

E. N., female, age 11 years. The childs symptoms had progressed for five days before examination. Her knees were flexed and contracted with acute painful swelling. Other joints were hot, painful, swollen and flexed. Her finger joints, elbow joints, knee joints and hip joints could be straightened out, but would "pop" right back to the flexed position. It was very painful for the child to even try to move them. She had a heart murmur, pulse rate of 120, temperature of 102°, inflamed throat, swollen tonsils, and severe adenoiditis. On July 3, 1942, after making the diagnosis of acute rheumatic fever, the child was placed on a fruit and vegetable juice diet and given 2 cc. of 6X benzoquinone solution.

On July 4, 1942 the throat and knees were better, the temperature dropped to 100°, and the pulse was 108. On July 6, 1942 all joints were better, the throat clear, the temperature was 99.2°, and the pulse was 100. On July 9, 1942 all joints were normal with normal function, no pain or swelling, the temperature was 98.6°, and the pulse was 78. The throat was normal and the heart murmur and adenoiditis had disappeared. By August 20, 1942 there had been no recurrence of any of her symptoms. A tonsillectomy was subsequently performed.

ADVANCED RHEUMATOID ARTHRITIS**

Treated in collaboration with
Dr. Wendell Hendricks

Mrs. L. J., age 60 years. She fell and injured her right knee 6 years before. Arthritis developed in the knee and progressively became worse. It spread to other joints. About a year later she developed nodules of the popliteal fossae and subcutaneous nodules on the hand. Examination on February 6, 1941 was negative for blood pressure and pus. There was an arthritic process of the metacarpal phalangeal joints with atrophy of the human snuff box. There were swollen, subcutaneous nodules, one in the tissue of the dorsal surface of the right hand and one in the left popliteal space. Hypertrophic arthritis of both knees was very marked. There was a general inflammation and disability of practically all of her joints. Her ankles were swollen and she could not wear shoes, she could not raise her arms, turn her head, or hold anything in her hands, nor could she get up alone. Thus the function of the joints were about nil.

On February 9, 1941, after 3 days of preliminary clearing of the bowels and a liquid diet, she received 2 cc. of the carbonyls. On the first three days of May 1941 she received 100,000 units of Vitamin D. per day. On June 7, 1941 (at the end of her 17th week) she received a second injection of the carbonyls. She had a local reaction a week after this injection. There was a definite improvement, a limbering up of the joints. On August 4, 1941 (the 9th week from the second injection) there was reaction. On September 20, 1941 (during the 15th week) 150,000 units of Vitamin D was prescribed for 3 days because of a local flare-up of the joints. It seemed to aggravate the condition and so on October 3, 1941 she received 2 cc. of benzoquinone solution. (The benzoquinone may have set her progress back or reversed it for the reason explained in the text.)

On January 7, 1942 a small nodule was noted forming in the left hand on the dorsal surface. At this time the nodules on the right hand and the back of her knees had all disappeared

and her joints were practically limbered up. However, after a temporary enlargement of the joints and another reaction of fever, she received a third injection of the carbonyls on February 20, 1942. The pathology was corrected or reversed and she made a fine recovery. She received two more injections of the carbonyls, one in February 1943 and one in February 1944 even though she was out and around doing her normal occupation.

ANKYLOSIS OF ALL OF THE JOINTS IN ATROPHIC ARTHRITIS

Major O. M. N., age 49 years, Physician in the Brasilian Army. Major N. condition started 3 years previously with pain and stiffness of the neck and the right shoulder. This progressed to envolve all of the joints with complete ankylosis. By October 1941 the muscles were markedly and characteristically 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 as demonstrated by palpation and by the radiographs. The visual fields were becoming restricted due to progressive restriction of the optic foramen. He could not use his jaws as they were completely ankylosed and thus he had to be fed with a tube inserted into his mouth. Coronary sclerosis was identified as part of the pathology.

Diagnosis: Marie-Strumpel syndrome with universal atrophic adhesive ankylosed polyarthritis.

Treatment: (a) The patient had received the classical treatment without any improvement. His natural resistance decreased and he was developing a very dangerous anemic condition. The fever was constant and the pain excessive.

(b) In October 1941 he received 2 mcgms of benzoquinone in 2 cc. of distilled water.

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 set up in bed by

himself. 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 he felt quite normal. He returned to active Army duty and remained well until his death in 1947 in the highland wilderness of Parana from pneumonia.

Discussion

The integration of toxins with tissue structures, be they supportive or parenchymatous, produce their obvious structural and functional changes. The destruction of the toxin by a chain oxidation initiated by dehydrogenation leaves undestroyed structures in condition for reconstruction on physiological levels so that function is restored. We hold that this circumstance is evidence that the normal protective oxidations are chain oxidations, carried by free radicals, and peroxide free radicals as we explained before. That these chain oxidations serve the tissue reconstruction as well as tissue function. This broad opinion seems justified since the destruction of the pathogen goes hand in hand with the tissue reconstruction and its returned activity.

CONCLUSION

A least common denominator in pathogenesis has been demonstrated and consequently a least common denominator in correction was to be expected. All tissues are concerned and all primary functions are envolved — contraction, conduction, secretion, reproduction. The least common denominator in pathogenesis affects the lowest living forms and is the cause of viral and bacterial parasitisms, and protozoal as well. Here we only mentioned syphilis and malaria though a fairly large experience in these diseases and in some trypanosome diseases could be detailed. The parasitism of the highest animal forms as expressed in cancer is seen to follow the same pattern and yield to the same corrective system. The parasite itself is a deficient form and therefore sick in comparison to the ideal that must surely have been a part of the original perfect creation. Some experiments here, that could be greatly extended, show that

the pathogen goes harmless and maybe useful after the corrective system is employed. Both parasite and host are thus cleansed of the same defect and returned to a normal level of function by one and the same catalytic acquisition, the first step in the vital process, dehydrogenation with free radical production in the presence of ample molecular oxygen. Pathogenesis then must start with a deficiency of any of these factors or their activities, and concerns the allergies and anergies as well.

The clinical problem then takes in the environment of the patient as well as his tissue defects, and the directions given here should be adequate to serve most cases.

APPENDIX

Amplifying Comments

Sugar Metabolism.

The highly efficient "smokeless" oxidation of sugar that leaves no traces of its existence by way of isolatable intermediaries, has been claimed by a number of brilliant biochemists. We believe it is the normal process and the others that are impeded enough to yield intermediaries are alternative paths provided to accommodate the need for vital energy production under the hindered circumstances that exist, such as anoxia, and injury. Isolation for study and the hindrances of the Warburg Chamber exemplify. In cancer a different hindrance exists. One resultant pathway has been ingeniously outlined by Krebs, but it has been erroneously accepted as the major process.

There are phenomena that suggest the "smokeless" process is one that is carried by a peroxide free radical, and the set-up may be as follows, but there is no real proof that the process is such. There is a strong confirmatory suggestion however in the competition offered by the amino groups of the oft used antibiotics, *which cause memory lapses,* periods of *suspended consciousness and the like,* that give rise to so many serious accidents. We therefore suggest that the F.M. especially of nervous tissue is provided with an activated amine group that condenses with the carbonyl group of fructose to form an azomethine bond, and that by an Amadori rearrangement this double bond passes to between C 2 and C 3. This of course mobilizes the hydrogen atom of C 4, and makes it subject to removal with free radical production, and in the presence of adequate oxygen, to peroxide free radical formation that carries the burning of the sugar to carbon dioxide and water. In the *absence of adequate oxygen* the sugar would split at this point to form lactic acid, or pyruvic acid. Catalysts that are required to facilitate this procedure are at hand, including the phosphorylases. Since we are unable to identify the "smokeless" process, we cannot tell how many such procedures there really are, and though we know much about the hydrolytic glycolysis system, for which we suggested a "hook-

up" in the text, we do not know the connections whereby the energy carriers give up their energy to the functional mechanism. Several processes including the fluorescence we suggested may operate here. In view of the vast deficit of fact we must consider all explanations as provisional. Since fructose is oxidized so much easier than glucose we offer the above suggestion as a possible course consistent with established chemistry. (See Chapter II and III).

References to the Literature

References to supporting contributions well known to biolcgists are not listed with their publications. The time only of the publication is given. This is because it is better for anyone not informed on such developments to consult one of the many fine text books on biochemistry and obtain a workable view of the subject. Our own references are given because they did not follow the beaten paths and are not included in such reviews. Fact is always preferred to hypothesis, but where facts are inadequate a given goal has no other path than by hypothesis, and this was our predicament. However our hypotheses are founded on fact and established chemical theory, and we did reach our goal, as the clinical achievements demonstrate.

Alkalinization of the bowel to normal pH for patients under treatment.

Since we look upon the amines developed in the colon by bacterial decarboxylation of amino acids as the vanguard of disease in an important degree, we have to suggest a means of normalizing the reaction of the colon contents to a normal of pH 8 where decarboxylation does not take place. To accomplish this without injuring the acid secreting cells of the gastric mucosa, we *suggest the use of several mild alkalies* during the meals, *three times a day.*

Notice is taken of the fact that iron in the ferrous state inhibits some of the more important decarboxylases in only a one percent solution, while hydrazine inhibits the four "co-decarboxylase enzymes" practically completely in a dilution of but one to a thousand. The ferrous iron excites no contrary

mutation, while the hydrazine does. However the early improvement secured would prove helpful were it not that in cancer *this reagent must excite a more vigorous neoplasia.* No doubt in the recent tuberculosis therapy this inhibition of decarboxylation is a most important factor in the early improvement, but with adverse germ mutations that are inevitable, it is better to not use the reagent. A simple procedure of securing a normal intestinal alkalinity is all that is needed, so we have prepared a capsule which can be dissolved in water and taken during the course of the meal. Several alkalies are taken as the citrates and carbonates. These are, besides a small dose of iron, and minute traces of copper and cobalt, tin and zinc, material doses of sodium, potassium, calcium, magnesium, and manganese. The stools should be examined as to reaction, which should be held at pH 8 to pH 8.5. The amounts of alkali may then be increased or reduced as needed.

Prolonged Silent Symbiosis

After vaccination against small pox the virus may maintain a prolonged absorption into the tissues or an integration with cutaneous tissue material either as a living virus or as a toxin which is able to produce tissue changes of a pox nature generally over the surface of the body when this toxin or virus is subjected to the immunological changes consequent to the carbonyl therapy of our text. Thus at the twenty-eighth week after the treatment has been given to certain cases of cancer, and the neoplasms have about completed their absorption, a general eruption of "small-pox" or vaccinia may show up, and then clear in three weeks, as the terminal phase of the recovery process. We have not had enough experience to master the details of this reaction, but it appears to fall in line with the rule we proposed that during the recovery the first symptoms to show up are the last symptoms of the pathogenesis, and the last symptoms to appear are the first of the pathogenesis. Thus we would place *vaccination* or a mild small pox infection as a causative agent in the development of the neoplasm, or one of the causative agents. It is probably held all this time absorbed or copolymerized with the collagenous material of the protective fibrosis, and being first to be laid down in this tissue, it is

the last to be broken down in the structure of the protective fiber, and is the one to show the monomeric or original form of the toxin before polymerizing through the successive phases that produce differing symptoms as the pathogenesis goes on. The last change in the toxin when we meet it, is its neoplastic property, and hence in the reversal of structure back toward the monomeric original form, the carcinogenic form is first to be lost and the cause of the cancer is thus removed, so it undergoes dissolution first. Any intermediate symptoms of the pathogenesis would show up in reverse order to their coming and disappear before the monomeric pox forming structure is reached. Thus while this is being set free from the absorbing or copolymerizing fibrosis it may produce the original symptoms of the infection before it is fully burned out of the way. We hold that it is fully burned out of the way since there are no cicatricial sequelae to the affair. Where we have given the carbonyl treatment to deep scars as from burns and in keloids, or following pox, this fibrosis has also cleared up to a remarkable degree, so we conclude that the scar is what we say, a protective copolymere or structure of collagenous material that adds the free radicals or highly polar unoxidized double bonds of the toxin of germ origin, or some incompletely burned tissue cell metabolite, or of a heat product, or an integrated virus.

The toxic fibrosis may predominate in a given tissue as the vascular walls and nerve nodes to produce a malignant hypertension with a systolic pressure holding stubbornly at 280 mms. Hg. Then twelve weeks after treatment a few days of reaction may duplicate the symptoms of an old malaria of several decades previously. The blood pressure is then found to be 140/80m ms. Hg. and remains so while the other symptoms are forgotten. A "silent" malaria was a factor in the fibrosis. It was the cause of the "essential" hypertension.

While vaccinia appears to form only the symbiotic type of integration, it may go lytic as would other less stabilized attenuated mutants, used as vaccines, when entering a favorable host. This may require hours or days. However certain viruses as Swine Pest virus are lytic only, and kill 100% of the pigs regularly in from 2 to 5 days. The state of union with the

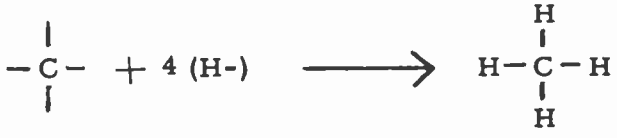
host cell however envolves the same atomic groups, as we have cured at times 100% of the treated pigs with one dose in from three to four days, even in the most virulent epidemics where the victims could not get up from the ground or eat or drink, and all untreated animals died in due course. The atomic unions are quite the same then no matter what form is taken or the state of the virulence. A twin or dimere status of the symbiotic virus holding unions as of Diagram V, (b), should be considered, as cleavage would favor change to the lytic type of action.

A Review of the Double Bond and the Free Radical

(1) Chemical Bonds:

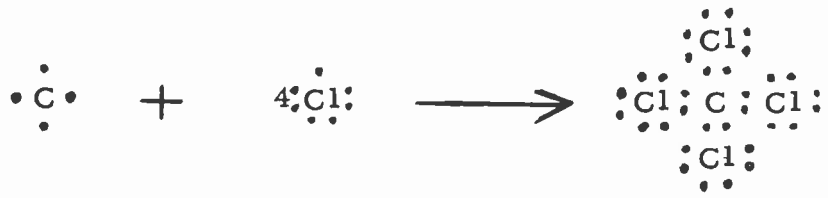
The forces that hold atoms together to form molecules are called bonds. Each comprises a component or valence contributed by each atom. Up to 1859, when Kekule the renowned German chemist clarified the atom bonding system, progress was slow in establishing the structure of molecules. He proposed that the atom carbon holds four valences in all molecules, simple or complex, in which it takes part. Each valence he represented by a short line drawn out from the atom initial and joined to one from the other atom to represent the bond between them. This bond is termed the covalent bond for the reason just stated. It is made up of a pair of electrons shared by each atom. The following diagram illustrates (1) the Kekule formulae and (2) the electron formulae.

(1)



(2)

One will note that each atom donates and also accepts an electron to make up the bond. In such a molecule, all of the electrons and nuclear charges are in the same atomic field and hence the molecule is non-polar, non-ionized and stable in structure. Carbon atoms have relatively little tendancy to give up or accept electrons and hence tend not to form ionic bonds. Carbon does more readily combine with other atoms by electron sharing, which envolves much less energy expenditure than ionization, and hence is most favorable for biological structures and processes. It is also preferable to silicon for purposes of organic life. Carbon also tends to form a stable octet with electron accepting atoms such as chlorine, thus:



Carbon also gains stability through its four electrons in the outer shell with little tendancy to gain or contribute electrons, standing as it does midway between the stable electron states of Neon and Helium and being very different in type.

The Covalent Double Bond envolves the sharing of four electrons by two atoms, two electrons being contributed by each atom. The bond is shorter and the bond energy values are greater than for the covalent single bond. However, the mobile electrons are not entirely satisfied and tend to make additions. This may be surmised from the fact that the C-C bond energy is 58.6 kcal. while in the C=C double bond it is 100 kcal. per molecule, thus leaving about 17 kcal. looking for "satisfaction". In symetrical molecules the electrons are symetrically disposed. Otherwise one terminal of the double bond would present more than the other and be partially polarized. The substituents determine the direction in which electrons are asymmetrically displaced. Thus carbonyl and nitro substituents tend to polarize the electrons in the opposite direction, and like halogen tend

to decrease the availability of electrons in the bond. On the other hand, alkyl, aryl, alkoxy and ester groups increase the density of the electrons in the bond and tend to make it more negative. The tendency and position of addition of radicals carrying relatively positive or negative charges is thus determined. While methyl substituents tend to displace the mobile electrons in the opposite direction to make the closer pole of the double bond more positive and more able to attract a negatively charged atom or group, the situation may be reversed in the presence of peroxide catalysts. This is worth noting since peroxide catalysis is quite universal in tissue chemistry.

Some covalent double bonds are polarized by the charges the constituent atoms normally carry. Thus in the carbonyl group the carbon atom has an electro-negativity of 2.5 in comparison with oxygen's 3.5 and will tend to attract electrons from a conjugated ethylenic linkage, as well as a negatively charged atom, while the oxygen would tend to add the more positively charged unit. In the azomethine double bond the carbon electronegativity is less than that of the nitrogen, being 2.5 and 3.0 respectively, so the carbon would tend to attract the more negative oxygen first in the oxidative cleavage of the bond. A carbonyl group as a terminal is thus assured. The nitrogen would also attract oxygen and separate as an oxide of that nitrogen.

Double bonds tend also to mobilize the hydrogen atom attached to the carbon atom in the alpha position thereto. This hydrogen atom tends to separate as a proton. Continuous oxidative breakdown is thus facilitated with production of a peroxide free radical as a chain carrier, or of a terminal carbonyl group with each step of cleavage when an efficient dehydrogenator is supplied.

(2) Free Radicals:

The double bond may make an addition at one pole so as to break one bond with the other pole. This produces an unpaired electron or free radical at the other pole which wants to satisfy its stable octet by adding another electron. Whether this free radical is neutrophilic or electrophilic will depend on

the electronic concentration at that position. Free radicals so formed will add to one pole of a double bond of the same type molecule or another and thus form a dimer which presents a free radical at the opposite pole. These continue with the additions until the polymeres or copolymeres formed are so large in molecular weight and so inert that they fail to make further additions. Thus the polymerization process starts out with an initiator addition and continues as a propagation phase until it terminates itself by the inert nature of the final product. The propagation phase may be halted by addition of an inert free radical in like manner and at any stage of its progress.

In free radical production, the fission of a covalent bond is "homolytic". That is, each resultant fragment possesses an odd electron. One may contrast this situation with "heterolytic" fission in which the pair of electrons go with one fragment leaving the other with an exposed nucleus (ionization). The former fragment is negative and is nucleophilic (an anion), while the latter is positively charged and is electrophilic (a cation). Such rupture of a covalent bond results when the attacking atom has an affinity for an electron pair or for an atomic nucleus. A cation attacks at the point of highest electron availability while the anion where the greatest paucity of electrons is offered. The energy envolved in ionic reactions is much greater than where free radicals are produced, since two electrons are moved instead of one. So the latter are adapted to biochemical processes pre-eminently.

It is easy to see that when one dehydrogenator molecule can start a chain reaction that will increase in velocity with great speed and convert an enormous amount of substrate in short order, the concentration of the initiator need not be great. When acting on substances in solution, a minimum concentration as 1×10^{-12} will suffice. Such a solution still carries many millions of molecules where but one is required theoretically. When the reagent must initiate the reaction separately in a number of tissue cells, even though one molecule per tissue cell may suffice, the number of tissue cells must be considered and so the solution must be adequately concentrated or dilute as you please. Physiological reactions are of this order and that

speaks for their free radical and chain characters. Thus acetylcholine is found to renew contractions in surviving guinea pig intestine muscle in dilutions of one part to a thousand million of water. (Paul Karrer, Organic Chemistry, 3rd edition, 1947, p. 239). In this instance the hormone itself becomes the free radical. The quinones may offer free radical resonance hybrids which initiate reactions in equally high dilutions, and besides, the quinone carbonyl group may serve as a dehydrogenator and produce a free radical in the substrate from which the reaction is propagated. For example; Echinochrome A, the pentahydroxyl quinone of beta-ethylnaphthalene, produced in the ovary and egg of the Sea Urchin is able to motilize sperm cells in dilutions as high as one part to 2×10^9 parts of water. (Kuhn and Wallenfels 1939—Fieser & Fieser, Organic Chemistry, 1944 edition, pages 752-753). Likewise Crosin, whose dehydrogenator carbonyl group is activated by conjugation with an ethylene linkage, was shown by Kuhn and Moewus (1938-1940) to activate the gametes of certain algae in like high dilutions where but one molecule of the reagent served each sex cell. Benzoquinone has shown therapeutic effect in high dilution.

Diphenoquinone offers resonance hybrid free radicals in solution. When going into solution under correct conditions, an apple greenish yellow color is at first produced by diphenoquinone. This color lasts a few hours before turning to yellow and then becoming colorless as its polymeres separate out. The color may last as long as seven hours during which period it is therapeutically active, but after it becomes colorless it is inactive. The rich quota of resonance hybrids serve as chain initiating free radicals.

For this reason and because its highly activated carbonyl groups are such efficient dehydrogenators, its ability to initiate oxidation chains in high dilutions would be anticipated. Observations have shown that doses of a fraction of a microgram are sufficient to cure well established Hog Cholera in four days. To accomplish this, the amount of virus and toxin that had to be converted into peroxide free radical (Antitoxin State of Structure) to progagate the reaction was quite large. It was evident in the clinical symptoms that the rate of turnover in-

creased rapidly as the hours passed. In the treatment of Rabies, a small fraction of a microgram of a compound presenting serially arranged carbonyl groups with free radical terminals demonstrated the same conversion power. Molecular oxygen is necessary to the production of the "antitoxin" state of structure we referred to as early as 1924. It is the carrier of the oxidation chain so long as toxin is present, and thereafter persists as the "immune body" we referred to in "Cancer and its Allied Diseases" 1926. How this carrier is stabilized until new substrate arrives and how it is activated to again convert substrate is a matter of conjecture. Clinically and in animals we find it behaves that way, but the mechanisms are obscure still. A dimerization as when triphenylmethyl is converted to hexaphenylethane could be the case, or adsorption of the free radical into some colloidal structure, or perhaps the resonance hybrids discussed below. The subject is worth investigation.

The greenish yellow to pure yellow color of diphenoquinone, when under going solution in water, recalls the yellow color of triphenylmethyl which the writer observed while enjoying the courtesy of working in Prof. Gomberg's personal laboratory, just seven years after that wonderful gentleman and chemist demonstrated the free radical beyond any doubt. He found that his free radical gave the same absorption in the visible spectrum that quinoid structures show, as benzoquinone for example. This fact helped him work out the resonance hybrids, nine in number, that added to the stability of the structure. Because of this resonance triphenylmethyl as a free radical tends to show more stability than when dimerized to form hexaphenylethane. That is its resonance tends to weaken the C - C bond (70-80 kcal) which compensates for the energy needed to break the bond. The increase in entropy gained by the wandering of the lone electron of a free radical in this way should prove very instructive in considering the phenomena of the long persisting immunity. The lone electron in this capacity also determines the color characteristics and makes the molecule paramagnetic, both properties serving in their detection and measurements.

(3) Photoactivity:

Nitric oxide permanently presents a free radical stabilized as a covalent bond carrying three electrons. It can block chain reactions carried by free radicals. All oxides of nitrogen offer the same predicament; thus when nitrous oxide is used as an anaesthetic, it has sharply reversed many a splendid recovery course. This fact throws light on the nature of the recovery mechanism, indicating that it is a chain process carried by free radicals. Another experience is the blocking of the recovery course by substances carrying highly polarized double bonds. Substances in coffee, tea, in motor exhausts and paint solvents are of that nature. Substances that release hydrogen easily may reduce the free radical chain carrier and inactivate it so as to bring an end to the propagation of the reaction. If, however, the hydrogen donor is itself converted to a free radical and oxygen is at hand the oxidation chain may progress. Hindering substances are those with sulphidryl groups. The hydrogen is released to inactivate the carrier free radical and two dehydrogenated sulphur radicals will unite as a dimer, terminating the process. Free radicals act like cations preferring to attack positions of high electron density. Halogen anions and halogen free radicals thus act differently as the anion is nucleophilic (basophilic). This difference should be remembered in the interpretation of biochemical events and in the preparation of therapeutic agents.

Covalent bonds are split by light waves quite specifically. It takes more energy for this cleavage than when produced chemically or by heat since the atoms are activated by light in addition to the cleavage of the bond. The activation may be translational and result in more effective reactivity through increased collisions or it may be internal with transition of a valency electron from one quantum orbit to another, thus a new binding energy is obtained. In this excited state the molecule vibrates, and if the energy is sufficient, a dissociation into fragments may occur. When the energy is not sufficient, a rearrangement of atoms within the molecule may happen or a new electronic configuration may result. The reactivity is thus altered.

A molecule that has absorbed radiant energy is capable of fluorescence, that is the emission of this energy (somewhat degraded) as radiation almost immediately (10^{-8} sec.) after its reception, unless the energy has been used in some other way as by collision with a particle that can use the exact energy quantum that the activated molecule is carrying. This specificity is important to our photosensitization theory of carcinogenesis (Koch, Natural Immunity, 1934, 1936, 1939). The gas phase is ideal for emission as irradiation, as the closer the molecules are together and the denser the medium, the greater the opportunity for inactivating collisions. Such inactivation by collision is a very specific affair and only molecules of a particular molecular species can accept the energy available and become inactivated thereby. Particles that can quench fluorescence in this way gain the energy as internal energy and not as translational energy. This type of activation is termed photosensitization. Such energy may bring about dissociation of covalent bonds and produce free radicals that bring about activation of reactions that mediate the cycle of nucleoprotein construction and cleavage of viral and neoplastic propagation. Once the free radical is formed, whether by light energy or in some chemical way discussed earlier, its deportment follows the known behaviors of free radicals. The initiation of carcinogenesis need not be by irradiation, though the processes of photosensitization depending on the energy passed internally through collisions can produce free radicals and the ultimate effect is the same as if from irradiation.