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

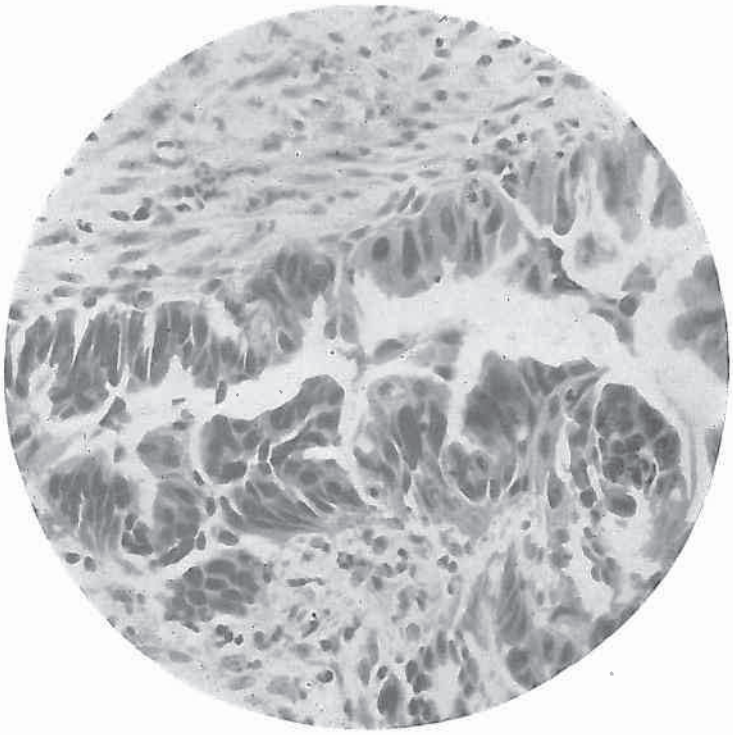
what the atomic bondings must be that constitute the integration of host cells with virus, bacterial filterable toxins, and neoplastic agents, that provide the energy transfers for parasitism, and allergy, or block its production in the anaplasias, atrophies and anergies. It was very evident too that 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.)

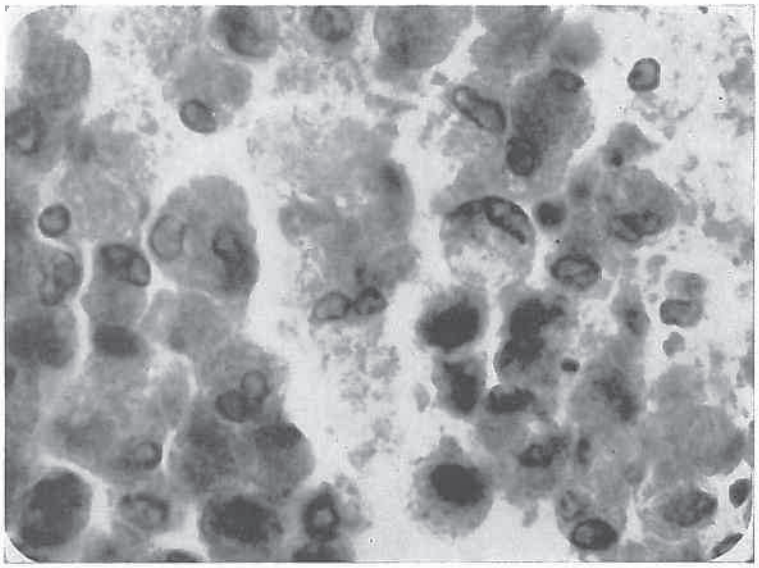
Our hypothesis also covers the pathogenesis of the fibrotic changes in the heart, brain and kidney resulting in the usual fatal diseases. The recovery process here, after our treatment, follows the very unique law of reversal of the symptomatology of the pathogenesis so that the first symptoms to come 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)

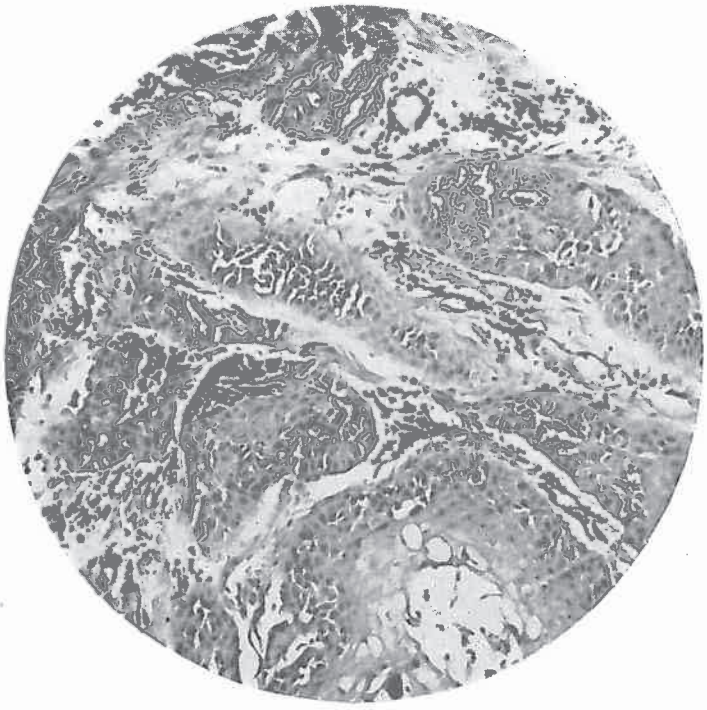
advantages, restoration is made possible, we believe, through the Nissl Substance, a most wonderful survival provision granted by the allwise Creator. Moreover, since the detoxication or deviralization is accomplished by highly active carbonyl catalysts of oxidation, we must conclude that the essential 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



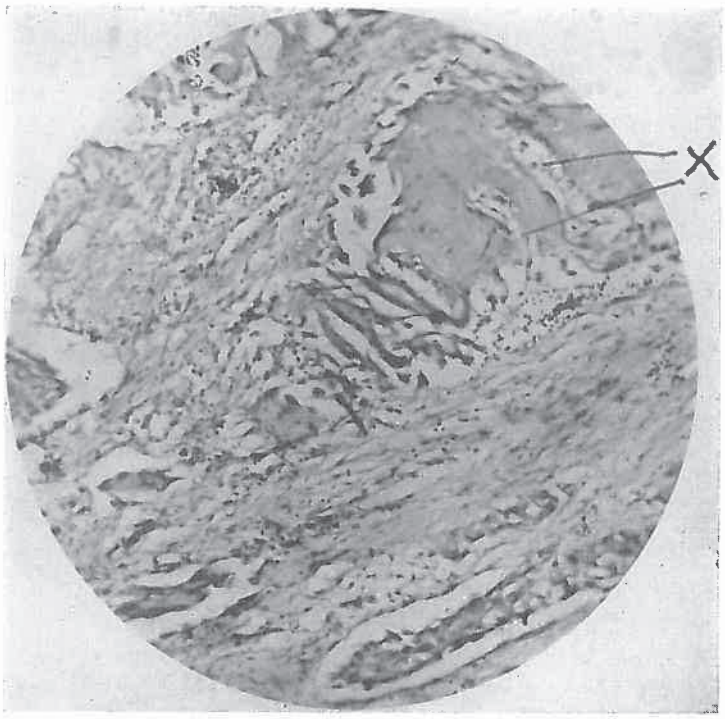
A. — Squamous cell carcinoma of the neck biopsy before treatment. (150X)

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,