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.