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.
7815 E. Jefferson Avenue
Detroit, Michigan

September 10, 1946

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