Chapter 21

THE PATHOGENIC MECHANISM IN CANCER AND CONNECTIVE TISSUE DISEASES

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

We have identified the initiating factor as an activated amine, which causes a gellation of the plasma and cellular colloids and this gellation blocks the transport of oxygen causing a local hypoxia or anoxia. In addition to producing the gellation as we have observed results from the guanidin poisoning in our parathyroidectomy work, the amine condenses firmly with the tissue function Carbonyl groups so as to block its initiation of energy producing oxidation chains. Incompletely burned metabolites of tissue cell origin or of germs caught in the anoxic area, then may be dehydrogenated so as to become free radicals but under the anoxic state 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 also co-polymerize with each other or with the collagenous material that is produced by fibroblasts, in response to the irritant effects of the incompletely combusted state. Thus, they are incorporated into the fibroblastic tissue intended to dispose of the toxicity. Cancer cells likewise, we hold, take up the toxic products into their structure in the same way and thus serve as detoxication agents. (Koch, Cancer and Its Allied Disease, 1926, Koch, Cancer Journal, October, 1924). As the fibers increase by added deposition of collagenous and toxin co-polymers, the last depositions must present the highest molecular weight and as such, each group produces a characteristic set of disease symptoms because each fiber carries a history of the intoxication from the inception of the anoxia. The same appears to be the case for each cancer cell, as well. If the cancer cell could complete the combustion of the toxin, it would offer protection and serve the immunity mechanism, which we feel is its intended goal (Koch, Cancer Journal, October, 1924) in antitoxic hormone evolution.

Our observations showed that the flu epidemics of the first world war gave cancer a boost, and its symptomatology was much more uniform after this event. As a rule, the pre-growth symptoms were found to exist five to twenty years before the growth came and the longer they existed in the parent with cancer, the shorter their appearance in the offspring that developed the disease. Thus, the pre-growth toxic period became shorter and the growth appeared earlier in life, 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 sciatica, dizziness, epilepsy, headaches or visual disturbances, etc. The nervous tissues seem to be the favorite site for the toxic action but there may be psoriasis, some other form of skin allergy, or a compulsory neurosis, while the lymphatic reticulo-endothelial system undergoes some atrophy. Before the growth appears, 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 appears, the symptoms disappear, wholly or in part, only to return again if the growth is removed and then again disappear with the recurrence of the growth. Depending upon the rate at which the toxin is produced, as compared with the rate of growth of the neoplasm and its adsorbing and co-polymerizing with the toxin, the latter is stored, out of the way more or less, and the symptoms will disappear proportionately. (Koch, *Cancer Journal*, October, 1924; Koch, *Cancer and Its Allied Diseases*, 1926).

To illustrate this situation, included is a letter received in Brazil from Pasadena, California. It runs as follows:

"In 1932, I brought my mother to you from La Crosse, Wisconsin, with cancer of the stomach. She was an advanced case, and the symptoms that she had suffered for years before the tumor was discovered are being repeated through me. I am asking your advice about an exploratory operation to see if I have what my mother had, and the doctor's examinations here indicate that I have, and only time will give the full proofs - your Treatment was given my mother and she got well and lived fifteen years longer and died of a heart attack. I started out with a terrible neuritis in my left arm, and could not raise it above my head. It increased in severity until I had to carry it in a sling for months. It gradually left there and went to my sciatic nerve, I also fell and hurt my knee. Tetanus antitoxic serum was given me and I developed a terrible urticaria that nothing helped except Cortisone. Then my body became covered with boils and I am taking an antibiotic for that, but have to keep it up. Before the trouble located in my abdomen, I began to take on weight and dieting has not helped a bit. Obstructive symptoms in the abdomen are the trouble now, and the neuritis has improved a great deal. This is the way my mother's cancer developed."

Such reports are the rule, with slight variations. But there are exceptions also. In this case the pre-growth toxic symptoms were easily contrasted with the symptoms of the neoplasm.

The fact that the toxin causing the symptoms can add to the structure of a developing fibrosis or of developing cancer cells, shows that it presents either highly polar double bonds or free radicals 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 in order to secure a correction of the pathology.

So long as fibrogenesis can absorb the pathogen, there will be no neoplastic response. But when this defense of the mesoderm fails, and the reticuloendothelial cells of the spleen and lymph glands become exhausted, the way is open for the pathogen to attack epithelia and cause cancer. This defense of the fibroblastic mechanism is a physiological hyperplasia to combine and wall off irritants and germ products. On the other hand, the neoplastic hyperplasia, as seen in spindle cell sarcoma, lacks the FCG needed to keep the activity under control. In the malignant

situation, the FCG is inactivated and requires liberation in order to resume its correct behavior. In the protective hyperplasia situation, the SSR is needed to burn up all combined and circulating toxin, including that in the germ that is its producer. Then the fibrogenesis is obsolete and involutes. So in both situations, the SSR is required to remove the cause.

VASCULAR DISEASE

Arterial sclerosis and the thickening of the interstitial connective tissues are all protective attempts to: dispose of a toxin, an imperfectly burned metabolic product of the tissues or of some germ hidden in an old scarred-in focus of infection, where anoxic conditions prevent the full combustion of its products. They then have the chance to polymerize thereby passing through stages that are different pathogenically, temporarily resulting in various diseases, in route to the production of cancer. The fact that the cancer patient gives a history of a series of disease states before the growth appears, which after the growth is made to disappear, repeat their appearance briefly in the reverse order to their coming, is observed very regularly and indicates a de-polymerization of the toxin from the cancer producing stage, down through each disease producing form until finally the monomeric form is reproduced, which was the initial disease form produced by the acute infection. Indeed, in cancer of the breast, after the tumor is absorbed, one observes a sudden acute swelling of the tonsil area of the same side, and a sore throat that lasts a week or so and then clears up with the disappearance of any scar tissue or hardened lymph nodes in the area. A breast case may get a final reaction in an old scar in some other part of the body, after the growth is absorbed. Thus, we link cancer and its allied conditions with germ toxins or viruses developed in anoxic areas.

One of the pre-growth symptoms is vascular sclerosis in general, or in some organ as the brain, kidney or heart and is accompanied by an interstitial fibrosis. The toxin integrates with the developing fibroblasts that come to give protection against the poison or its co-polymer with an incompletely combusted tissue metabolite. It may be taken as a rule that fibrosis protects against cancer so long as the tissues show the ability to produce the necessary fibroblasts, but when this ability fades away, the toxin can keep on polymerizing until it either produces cancer or, let us say, the fibrotic protection is lost before it achieves that property, and for this to occur, an hypoxic focus is necessary as in a scarred-in tonsilar crypt or focus of infection. Then one must agree, with our first experiences, that the carcinogenic agent was primarily fibrogenic.

In **Case No. 55**, the fibrosis with all its tissue accompaniments of depressed oxidations, such as cholesterol deposits, calcified plaques, parenchymatous hypoxia, malnutrition and blocked elimination, had reached the limit. Still, the restoration of FCG function cleared up the whole pathology in a progressive undoing of the pathogenesis. The “disease building” was torn down starting at the “roof,” and new tissue was healed in where the repair was needed. In the Coronary Cases, however, besides the diseased arterial walls and interstitial hyperplasia, there was the element of blood gellation that caused the acute attack. The gelled blood, as in the parathyroidectomy situation, blocked the circulation, but true coagulation would not have taken place for a while anyway, though it was inevitable, had not the Survival Oxidations been restored. Therefore, the recovery was just as fast as the restored oxidations could charge the blood colloids and restore their correct dispersion. In acute apoplexy, this has only taken a few hours at times. The Coronary Cases thus picture the acute action of the toxin along side the

chronic action and the Senile Sclerotic Case and the Bright's Disease Case expose the chronic action, with the reversal of the pathology.

Here we see in the polymerization tendency, the action of the free radical and the double bond. This free radical, when it gets a chance, can add to a double bond of fibroblasts and cause the sclerosis or the free radical can add to the FCG system of the parenchyma and cause various allergies or other disease pictures, like pityriasis rosea, or psoriasis. And when it polymerizes to the point where it has reached the correct steric advantage, it can add to the FCG of the mitotic mechanism of cells to cause cancer. It is our opinion that wherever it has integrated with a tissue FCG system and interfered with function, it stays there undisturbed. But it undergoes destructive oxidations as fast as exposure permits at the same rate no matter where it is lodged. Thus, the orderly reversal of the pre-growth symptomatology is possible.