# THE CURE OF DIABETES

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The period of observation includes scattered cases treated since 1922 and recent systematic studies. The cases treated cover about every type known, including a few of diabetes insipidus.

The treatment material consists of catalytic dilutions of the carriers of aerobic oxidation which we have described in the past elsewhere.* These substances are 1:4 Benzoquinone and its transition products Glyoxylide, (O=C=C=O), and Malonide, (O=C=C=C=O), and also Ketene. Their chemical structures conform to the rules we have laid down as requisite to the production of immunity against disease; namely, they possess the smallest molecular weight possible: they possess carbonyl groups that share ethylene linkages or carbonyl groups that are conjugated with ethylene linkages in molecules that can yield carbonyl groups sharing ethylene linkages.**

Substances of this type can be extracted from the heart and brain. Such extracts demonstrate the same curative results as the synthetic products, and like them correct a definite blood coagulation deficiency. Because of this physiological property, I named them "Tissue Thrombin" and showed that they are able to cure cancer, in a paper published in the New York Medical Record in October, 1920. The synthetic products can be used with good control while the tissue extracts are very unreliable and have only academic interests. Vitamin K offers very weak protective properties in addition to its influence on blood coagulation and in its little way confirms what we have been teaching for years with reference to carbonyl and ethylene groups in the oxidations. It is also interesting to note that researchers in the United States Public Health Service now proclaim they have accepted the view that cancer is a disease based upon oxidation deficiency. The Rockefeller Institute is reported to be doing actual confirmatory work. So in time, opposition from such quarters may be expected to vanish. This is important since it is our mission to demonstrate that the common basis of disease of all kinds, including

diabetes mellitus and diabetes insipidus, is a specific defect in the oxidation catalysis.

The clinical data indicate that the cause of diabetes is the prolonged poisoning of the tissues by bacterial products liberated in scars that have scanty circulation. These products are intended to serve the nutrition of the germs that produce them and are originally of fairly small molecular weight, diffusible, and fully oxidizable by the oxidation mechanism of healthy tissues. However, when secreted under the anaerobic conditions prevailing in scars they are not burned, but instead some of their free valencies yield to polymerizations by which the molecular weight increases progressively while the photochemic properties vary also with the different stages in the polymerizations. The different photochemic values have different pathogenic powers; and so, as the polymerizations progress, the patient passes through a series of different pathogenic influences that produce different symptomatologies and physical changes.

Diabetes is one of these effects, with a predilection to express itself in certain persons and races. Neuritis, various degenerative diseases, psoriasis and other changes may be exhibited, but the final change in all, if the patient lives long enough to develop it, is cancer. Here too the hereditary factor plays a part for we have observed that successive generations tend to develop malignant growths earlier and earlier in life and the longer the disease has expressed itself in the ancestry the shorter is the pregrowth toxic period that exhibits diabetes or the other changes.***

The oxidation catalysts we use therapeutically are depolymerization agents and while the toxin is being depolymerized to its simple burnable structure, it passes through the various phases that were active during the pathogenesis, and so fleeting accentuation of these changes come and disappear in the reversed order while recovery is going on. Thus when the patient is treated in the cancer stage of the poisoning and retraces the symptomatology of the pathogenesis, the last toxic expression to come is the first to go and the first to come is the last to go. Therefore, an acute inflammation in some old cicatrix that imprisoned the causative infection is the last change to take place. After the germ's nutritional agents have been oxidized the germ dies and the scar which imprisoned it becomes obsolete and is absorbed. Thus with the destruction of the etiological factor, the disease is completely cured. When some form of diabetes is

one of the pregrowth symptoms it is overcome before the scar is cleaned out and since this happens soon after the cancer growth is absorbed we conclude that the molecular weight of the toxin at this stage is quite great. Cases of diabetes cured by this treatment, before they can develop cancer, may pass through other expressions of the intoxication producible by greater or lesser molecular weights, thus the neuritis will get well before the diabetes and the obliterative endarteritis will start to heal before the blood sugar is normal. But a psoriasis or other change may show up transiently after the blood sugar has become normal. Therefore, diabetes must be studied as a phase of a systemic intoxication and cure cannot be fully established until the focus of infection has been wiped out. Thus the recovery is not measured alone by the return of the blood sugar to normal.

Obliterative endarteritis, which is a specific effect of the poison that causes diabetes, we regard as the lesion that specifically indicates a depression of the oxidation catalysis of the tissues, general or local. Therefore, this lesion is a definite indication that the oxidation catalysts we have contributed to medicine should be employed to remove the basic cause of many diseases, including Buerger's disease, leprosy, tuberculosis, syphilis, cancer, and so many more. There can be little doubt that the endothelial hyperplasia is a compensatory attempt to increase the surface for filtration of oxygen from blood to the tissues, even though it defeats its own purpose. The demand for activated oxygen on the part of the tissues is also expressed by poor sugar oxidation as observed in diabetes mellitus, cancer, the thyroid diseases, and others. By removing the basic pathology, that is, by restoring the oxidation catalysis to normal or better, all expressions of the intoxication are removed, and the metabolism, the blood pressure, and the tissue functions are again able to run along as they should. The focus of infection is wiped out and the disease is cured in its totality.

After an intramuscular injection of our Benzoquinone solution or one of the other catalysts mentioned is given in diabetes, recovery begins very quickly and its progress can be measured by the decrease in the blood sugar. The amount of sugar eaten will affect this reading, but not the recovery mechanism. We feed all the sugar and starches the patient desires and as he recovers he is more and more able to use them. Where the sugar intake is controlled, however, it is

usual to observe a drop of twenty mgms. per cent every week until normal is reached. But we have seen both slower and more rapid restoration to normal. Sometimes a severe case is encountered in which the blood sugar is over four hundred mgms. per cent and the vital organs have undergone fatty degeneration to a fatal extent. In such cases the blood sugar may come to normal and the gangrene heal, but the patient may die from heart failure after excessive exertion, either very early or very late after recovery from diabetes.

In one series of cases, where the patients could be watched closely, the four percent of failures belonged to this group only, but in each instance the blood sugar came to normal before death. Acidosis is not a serious factor either, because with the restoration of the oxidation of sugar, the fatty acids are also burned. The recovery is not simply the reduction of the blood sugar to normal. It involves the correction of the whole pathology and the removal of its cause. Therefore, the use of insulin is not essential. Indeed it is better as a rule to do without it from the commencement of treatment. The few cases of diabetes insipidus treated so far have also recovered.

The treatment procedure is to stop all medication and cleanse the bowels for a few days. Then one dose of one of the oxidation catalysts is injected intramuscularly. The dietary regime is vegetarian entirely. No animal proteins whatever are permitted. Thus the production of nitrogenous negative oxidation catalysts in the intestine is retarded. Colon lavage is helpful. The diet should be reasonable and include plenty of vitamins and tissue salts. Foods containing quinones and terpenes that serve as negative oxidation catalysts must also be eliminated from the diet. Therefore, coffee, tea, mangoes, and citrus fruits are not used, and exposure to paint solvents, perfumes and automobile or furnace gases is avoided. Food should not be cooked in aluminum. Alcohol, tobacco, and spices are forbidden. One dose of the remedy is usually sufficient where cure is possible.

After the remedy is injected one should watch for periodic reactions which play their part in the recovery process. These have already been described.**** They generally come at three and a half day or three week intervals until recovery is complete. If an interfering factor prevents recovery it should be identified and removed and the dose repeated.

The whole profession is invited to make ob-

servations with the four remedies we have been using most effectively. We prepare each dose as carefully as possible for every patient that is chosen for treatment and at the most reasonable cost possible. Case discussion is welcomed, and advice is offered whenever desired; and all progress reports are of the utmost interest to us.

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