- **Publications 1912-1939**

On the Occurrence of Methyl Guanidine in the Urine of Parathyroidectomized Animals, 1912

Chemical Consequences of the Removal of the Parathyroid Glands, 1913

Toxic Bases in the Urine of Parathyroidectomized Dogs, 1913

The Physiology of the Parathyroid Glands, 1916

Tetany and the Parathyroid Glands, 1918

A New and Successful Diagnosis and Treatment of Cancer, 1920

CANCER Its Function and Cure, The Evolution of the Immunity Process, 1925

The Prevention of CANCER, 1926

Cancer Supplementary Points, 1926

The Koch Cancer Treatment and its Investigation , 1927

Blood Chemistry in Malignancy

The Function of Cancer

Journal of the American College or Proctology

Pathogenesis and Immunity as Conveyed

Natural Immunity via Aerobic Glycolysis, 1938

- **Publications 1940-1949**
- **Publications 1950-1967**

BLOOD CHEMISTRY IN MALIGNANCY

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It is the current teaching that the cardinal inflammatory phenomena are compensatory, protective, combative, physiological events. In our January paper, we demonstrated conclusively that these changes are the direct result of chemical action of the invading toxin upon the proteins of the area attacked; an action that results in dehydration of the protein with liberation of surface energy in the form of heat, vascular degeneration, extravasations of blood and lymph, and the well known symptoms. These are evidently pathological changes. But the lymphocytic infiltration and subsequent fibrosis are repair processes, and though they cannot replace parenchyma, they are physiological events.

Cancer tissue is generally thought to be a pathological tissue. In our January paper, we demonstrated conclusively that this is not the case, but that it performs, in an imperfect way, a protective function; that as soon as the etiological toxin has been removed, it reverts to a normal type of tissue and is immediately digested, absorbed and becomes nutrition material for the rest of the body. The injury done to a paranchematous tissue through inflammation is permanent and normally is not regenerated. The injury done by cancer tissue is corrected with restoration of lost parenchyma sufficient to meet functional requirements. Thus, cancer tissue differs from a pathological tissue and performs a new function in its imperfected way. It thereby resembles the physiological instead of the pathological.

To discover the pathology in malignancy, we must look beyond the cancer growth. Surgery and other attempts at destruction of the cancer have not succeeded in removing its pathology, in any instance, even though employed with skill and on a large scale. However, an inquiry into the chemistry of the blood not only locates the etiological factor, but it traces the injury done, and discloses the means of arrival at the cancer state. Just as a correct valuation of the changes of inflammation was gained from an inquiry, so to a useful comprehensive conception of cancer will be gained from an inquiry into its chemistry.

The pathogenesis and pathology of all diseases have so much in common that the discussion on blood chemistry to which this paper is devoted will be found enlightening in many conditions that might appear to be only remotely related to malignancy.

All diseases are caused by toxins, whether the toxin arises through the action of a bacterium, a protozoan, a snakebite, a plant, or a chemical laboratory. It is not the morphology of a germ that is poisonous; it is the toxin it produces. Moreover, the toxin of each germ is essential to the germ's existence, and since it is impossible to destroy germs directly through an antiseptic within the body, the only feasible means of destroying a germ is to remove its essential toxin, better yet, change the structure of this toxin so that it is no longer useful to the germ but incompatible with its life. We demonstrated in an earlier Bulletin that chemotherapeutic agents, like salvarsan, do not slaughter germs in the body, but act entirely through their dispersion effects on the proteins of the host, and that their use in too large dosage may destroy the host while the germs are stimulated into activity. A measure, therefore, whose action is entirely upon the toxin of the germ and thus removes the true etiological factor in the first instance and necessarily, thereby, the germ that depends upon the toxin, must be a matter or profound interest.

The primary action of a toxin, as we demonstrated in the January paper in our discussion of inflammation, is one of chemical combination with the protein of the host. The result is an alteration in the colloidal structure of this protein, mainly the circulating blood protein that picks up and carries the toxin. The changes in the physical structure of the protein determine the fate of nutritional elements that should be carried and metabolized, and besides the passage of blood through capillaries may be partly or wholly impeded. As a result, intestinal changes or parenchymatous degenerations result from changes occurring in the physical state of the blood protein. Lytic toxins favor vascular injury and intestinal changes, while hydrators injure the parenchyma preferably.

In health, the blood protein presents the following physical properties. Besides the well-known characteristics of the red and white cells, the tendency of these cells to remain suspended uniformly in the plasma must be emphasized. Normally, the suspension stability of the red cells is such that they tend not to settle more than two-tenths of a c.c. from a citrated blood in one hour. They tend to remain in suspension by virtue of the surface charges they carry, just like the big bodies of the solar system remain suspended by virtue of such charges. In disease where these charges are lost, the cells tend to settle out to an extent and with a rapidity that follows the rate of loss of the surface charges.

When observed with the dark field microscope, the plasma is seen to be made up of many small, evenly dispersed, moving particles of about equal size. These particles are seen by the light they diffract, and are much smaller than they appear. When albuminous they appear dull gray; when their lipoid content has increased they become gloubinous and appear whiter and more highly refractile, with increase of fat content. There is a normal extent to which the albumin and globulin characteristics may vary, but beyond certain limits an increase in fat content is pathological, and determines increased viscosity, diminished surface tension with resulting functional injury.

The dissolved crystalloid constituents are present in two forms. A definite equilibrium is maintained between the crystalloids present in the dispersion medium in ionized solution, and those absorbed into the protein particles in a colloid state. Those in ionized solution present ionic activity, and those in the colloid state present electronic activity contributing to the dispersion charges carried by the particles. Losses in the absorbed colloidal constituents, therefore, diminish the surface charges and detract from the extent of dispersion of the protein particles. The state of dispersion of the particles is thus dependent upon the surface tension, which determines ease of absorption of crystalloids by the colloids. The balance between monovalent and divalent cations control whether or not a lipoid in water, or a water in lipoid phase shall predominate and thereby cause a rise or a fall in the surface tension and absorption powers. Thus, the balance in crystalloids of various characteristics will help determine their concentration in ionized and electronized state. Since deviation from the normal state of dispersion contributes so much to pathogenesis, the amount and distribution of crystalloids is of utmost importance, and the data revealed by blood analysis have a far greater significance than is awarded them at present.

The electric charges contributed to the particle as its absorbed constituents contribute much to the state of dispersion and capacity to functionate. There is another very important but heretofore unrecognized source of surface charges for the proteins of living tissues, namely, electromagnetic waves arising through chemical activity going on in functionating nerve, muscle and gland tissue, and perhaps also in the contents of the digestive apparatus. It is no longer possible to deny (even by the most ignorant) that each chemical action has its electromagnetic counterpart. So the chemistry of thought possesses, the chemistry of each nerve impulse, and of each muscle contraction has its wave of change in electric potential and electronic manifestation, depending thereon. The nerve impulses to an injured or inflamed area are increased reflexly and pain is registered in consciousness. Muscle spasm about the injured area cannot help but contribute electromagnetic waves and electronic charges to the region. Nerve impulses, with their accompanying waves of increased electric potential dissipating along the capillaries and to their thin layers of contained blood, cannot help but serve to add electric charges to the circulating protein particles. These are physiological means of aborting the beginning dehydration of inflammation. An osteopathic treatment causing a flood of nerve impulses to be sent out to an inflamed area cannot help but produce an increase in this physiological electronic

bombardment of the protein particles of the area, at the expense of the chemistry of the nervous system. But it has been amply demonstrated that such a flood of charges contributed to extremely dehydrated protein, as occurs in pneumonia, and can bring about the restoration of a satisfactory state of dispersion. Perhaps the osteopath does not realize the minutia of the changes initiated through his treatment, except that he is playing upon a natural mechanism with limits of responsiveness and limits toward exhaustion, and requiring intervals of rest for nerve chemistry regeneration. We must appreciate the effectiveness of the mechanism. The favorable influence of sunlight and some therapeutic light measure upon the state of dispersion of protein particles is indirectly recognized. The only favorable influence of radiotherapy depends upon the very early and comparatively brief tendency to increase dispersion of proteins. It is too bad that therapists do not care to remember how transient this phase of radioactivity really is and that its dominant phase is a persistent induction of non-dispersion, e.g., hydration. Thus too great exposure to sunlight, or any exposure to X-ray or radium tends to permanently increase the development of cancer. An optimum amount of sunlight, exercise and nerve tension tends to favor and preserve normal dispersion, and hinder the influence of disease, and cancer causing toxins. (Fifty years ago, Dr. Koch advocated fresh air, sunlight, exercise, and low stress as factors in disease prevention!)

With progress in the evolution of the nervous system, the glands of internal secretion and the much-misunderstood hormones have come into existence. We discussed their means of action from the basis of their chemical and electronic structure, in a former paper. The selective physiological action they display goes hand in hand with their differentiation from the various germ layers. Those of entodermal origin protect against injury to the colloidal state of the proteins by substances having their origin in the digestive tract. So the pancreas, the parathyroid and the thyroid tend to maintain normal dispersion of the proteins, in the face of the lytic and hydrator influences of materials absorbed from the intestinal tract. The cells of internal secretion of the testis, ovary and the cortex of the adrenal, protects the state of dispersion in tissues originating in the mesoderm.

The functioning derivative of the ectoderm and the nervous system is aided by the posterior lobe of the hypophysis, the pineal and the various chromofine tissues, including the medulla of the adrenal that develop from the ectoderm. These chemical substances are distributed throughout the body by the bloodstream and serve complimentary to the impulses sent out by the nervous system. In the long run, they all determine, to a great extent, the distribution of crystalloids in the blood, and accordingly, the blood analysis must be interpreted with their function in view.

When in spite of all normal regulatory influences, a disease agency disturbs the chemistry of the blood, its physical manifestations are: a loss of surface charge carried by the particles with accompanying disintegration of the particles, increases in number, decreases in size, and a lessening of the Brownian Movement. The particles may subdivide so greatly as to become invisible and pass into true solution, or they may form a gel. This change is called lysis or dehydration. Loss of the Brownian Movement and the surface charges permits them to come together and agglutinate, precipitate, or form large clumps.

Thus, they can physically plug up blood vessels. The anti-mortem clots that occur in the veins in parathyroid deficiency, lytic fevers and all of the white and striated clots, have their origin through gelation of the particles. This great loss in surface charge, moreover, reduces the suspension stability of the red cells, so they settle out and leave large white clots and striations of red and white. This type of coagulation differs from true coagulation in important respects. Whether the settling out of agglutinated particles, or the gelation disturbs the circulation in an organ, clinical manifestations occur that can be classified. They are discussed below:

In consequence to prolong lysis and following the action of toxins of protozoal or gram negative bacterial origin, the particles upon loss of electronic charges clump very markedly with only a short, barely noticeable lytic or dehydration phase. This process is called hydration. The enlarged particles, thus formed, increase in lipoid content. As time goes on, their surface tension decreases and they take up or absorb practically any colloid or crystalloid that comes in contact with them. Thus,

they enlarge and increase their electronic charges with each addition of crystalloid absorbed. But their absorption takes place not only throughout their surface, but also throughout the body of the particle, even to the extent that an inner surface is formed to better accommodate the charges gained. Thus, the particles become hollow spheres and rings. Together with this change, a drop in the content of ionized crystalloids takes place. Since the usual blood estimations register only the ionized form of a substance, they must be interpreted in accord with the physical state of the blood colloids in order to have full significance.

When the blood protein is in a normal state of dispersion, the equilibrium between ionized and electronized, or absorbed sugar is reached with approximately one-tenth of a gram in solution in one hundred c.c. of blood. Sugar is one of the substances absorbed with moderate tenacity by the protein particle, the salts are held in less firm colloidal combination, and the amino acids and urea are held more firmly than the salts or sugar. Hence with mild lysis a change in ionized salts is estimated, with more effective lysis, the sugar is set free showing an increase in solution and with very deep lysis, the urea and certain nitrogenous rings are liberated. When we observe a high increase in urea, therefore, we know that an advanced state of protein disintegration has taken place. Normally, there is a fairly wide divergence in urea estimations, which depends upon deaminization going on in the liver after meals. It will vary normally from twenty to forty milligrams per one hundred c.c. of blood. Manipulations during the analysis set free some of the substances to be estimated from the colloidal form, and no estimation can be counted as absolutely correct, but with careful technique a valuable insight into the proportioning of materials in the blood is obtainable.

Let us illustrate the above relationships with common observations in diabetes. Sugar diabetes is not a disease in itself, but a symptom of a constant state of lysis of the protein particles at the hands of a persistent poison. The change in the blood picture, the ultra-microscopic lysis, the loss of suspension stability of the red cells, the increase in salts, sugar, and finally of urea, above the normal dextrose to nitrogen ratio allowed by amino acid metabolism, simply traces a chronic or acute increase in lysis of the blood protein. The interstitial changes in the tissues and finally the parenchymatous changes that follow the blood injury are its ultimate and fatal results.

The first effect of the poison is a mild dehydration of the protein particles, they lose their Brownian Movement and split up into smaller, sluggishly moving particles and some of these particles come together and coalesce. An increase in salt is estimated in the blood and also in the urine. If the lysis is very rapid, sugar and urea may show immediate increase and there may be some filtration of the very finely divided protein into the urine without any nephritis being, as yet, produced. But as a rule, the condition is mild and goes on without notice, except perhaps for some increase in blood pressure and such symptoms as dizziness, headache and vasodilation that accompany the rise in blood pressure. The production of the etiological toxin increases and the blood injury increases with it. Sugar appears constantly in the urine and is found to also increase in the blood. The symptoms may not be marked, but the state of lysis in the blood sensitizes it to further lysis by other dehydrators, such as the coccogenic germs and resistance to infection is thereby reduced. Boils occur. Any physiological increase in dehydration, as menstruation or pregnancy, will increase the lysis and the amount of sugar set free from the blood protein will increase. Likewise, lytic substances, as anesthetics, aggravate the loss of sugar from the blood protein, or may increase the lysis to the point of gelation and result in impeding the passage of blood through the capillaries and thus cause coma. On the other hand, an old gonorrhea infection, an old lues, tuberculosis, cancer, or malaria will moderate the lysis and the diabetic state will improve because of the hydration induced by the toxins of these diseases. The injection of insulin, which is really a hydrator, likewise brings together and collects the disintegrated protein particles, lowers the surface tension and increases the absorption powers, so that the free ionized sugar is absorbed by the particles and held in the electronized state, in which condition it can functionate. Each molecule thus absorbed, increases the particle's charge and its state of dispersion. If too large a dose of insulin is given, the hydration may be so extensive that the collected particles form such large clumps as to block the cerebral vessels and cause shock, mild or fatal. Hydration increases the absorption power for all blood constituents, such as oxygen, salts, amino acids and urea. Under the influence of a hydrator, they are taken up by the

colloids and are usable because after absorption; they exist in the electronized state. For the same reason they do not exist to so great an extent in the ionized estimable condition, nor are they filtered out through the urine. An interesting feature that reflects upon the chemistry of the hydrated particle, distinguishing it from the hydrated particle, is the behavior of blood sugar in early diabetes, as compared with that in late diabetes when lipaemia arises. Early in the disease, when dehydration is prominent, the blood sugar estimates are high. Late in the disease when hydration has gone on sufficiently to produce a high degree of globulin and lipogobulin, the blood sugar estimates drop. This is because the surface tension of the lipogenous hydrated particles has become sufficiently low to raise their absorption powers and most of the sugar is taken up into the colloidal form, even though the etiological toxin is still as active as ever. The change from the lytic phase to the hydration phase of reduction has not received enlightening discussion from physical chemists, but we may hazard the explanation that the prolonged lysis has brought about so great a loss of monovalent over divalent cation, like calcium, that the colloids assume the water in lipoid phase of structure, the fat diffuses to the surface of the particle, and oxidation is impeded. The failure toward oxidation of Carbonyl groups leads to production of further fatty acids and the general lipoid characteristic increases still more. The production of Carbonyl groups adds to the surface charges and independence of the particle, but when a reversal in the balance of monovalent and divalent cations comes about, the whole particle undergoes disruption with an extreme degree of lysis, gelation taking places in the cerebral capillaries, and a coma results. It is at this stage that the urea nitrogen rises high, and creatine and other nitrogenous substances are found in the urine in great quantity. If sugar were fed when it is found to drop in late diabetes, it would tend to prevent this evil form of hydration, likewise calcium and parathyroid extracts, both of which are dispersers of conductors of electrons, would help prevent the rapid fatal lysis.

Insulin is not a specific for diabetes. If carefully handled it will manage a useful degree of hydration over dehydration so that sugar will be absorbed and exist in a useful electronic state in colloidal combination with the protein. Insulin does not remove the etiological toxin that started and continues the mischief, but patches up to some extent the bad effect of the toxin on the protein particles. The changes in the sugar behavior of diabetes are only secondary to the protein injury. It would be more logical to remove the cause of the trouble, stop the production of the toxin, or better yet, change it to its anti-toxin and have a fairly permanent protection against the etiological factor.

A certain amount of lysis is physiological in women at menstruation and still more during pregnancy. After menopause, hydration is the tendency, yet after the age of forty, hydration is the tendency in many people of modern habits. The states of lysis that are pathological are usually due to focal infection by streptococci and staphylococci, and autointoxication from the colon. With high blood pressure, a mild degree of hydration that goes with the lysis at the start increases as time advances until hydration dominates, as described above. However, the lysis endures long enough to bring about marked interstitial damage before hydration sets in with its tendency to parenchymatous degenerations. We may look upon the state of lysis going on as a universal state of inflammation of mild degree. The blood changes are like those occurring in diabetes in its earliest stages, lysis of the protein particles, diminished suspension stability of the red cells, lowered viscosity, vasodilation of the capillaries and increased surface tension with loss of electric charges carried by the particles and increased systolic and diastolic pressure. The dehydration of the intima of the smaller vessels increases the permeability of the walls so that rupture is entertained; perilymphatic and perivascular infiltration by lymphocytes, and a subsequent replacement with fibrous tissue bring about interstitial damage. If such important capillaries as the glomerulus of the kidneys are concerned, the early changes may be shown by bloody and albuminous urine. The final fibrosis means their obliteration and a permanently high blood pressure till heart failure supervenes. If the liver is the seat of rapid dehydration, red atrophy is the result; if dehydration and hydration go on together rapidly, acute yellow atrophy is the result. Rapid lysis, such that has reached the degree of gelation, means an abrupt loss of supply of oxygen and nutritional elements to the tissues, and the clogging of the capillaries by the gelated protein. If the central nervous system is involved, coma results. If the cord is the site of the change, a transverse myelitis results; if the heart is concerned, an interstitial myocarditis results. Dehydration is a matter of tissue

hardening or deaquafication, as indicated by Perdue. It follows an increase in amino above carboxyl groups, (the state of so-called hyper-alkalinity), irritability and sleeplessness are among its symptoms.

Hydration, on the other hand, is a softening process due to the development of fat in the particle, (the so-called acidaemia state), as previously explained. Syphilitic arteritis (hydration) is characterized by areas of softening, whereas, the arteritis due to dehydration agents, is characterized by hardening and calcareous deposits. The soft leutic arterial wall is subject to aneurismal dilation; the hard area of arteriosclerosis does not do so. Yet, they may be found side by side in an old leutic. With hydration, parenchymatous degeneration of liver and convoluted tubules marked with fatty change is the rule, and other things being equal, low blood pressure, tendency to be chilly, disinclination to be active, amyloid changes and the cachexia of malnutrition and anemia characterize this state. Hydration with its parenchymatous degeneration is the sequel to prolonged lysis, and this late change with the interstitial injury that belongs to the early damage done leaves the patient in a sad state for efficient kidney, heart and liver function. Yet this represents the condition of so many cancer patients, for cancer is most often a sequel to a prolonged intestinal intoxication that has exerted full dehydration changes and a good quota of degeneration attending hydration.

The blood of most cancer cases, as we meet them, presents a fairly marked anemia, a low suspension stability of the red cells. They may drop three or four cm. instead of two-tenths of a cm. in one hour. The ultramicroscopic picture shows both lysis and hydration of marked degree in that only very few diminutive, sluggish particles with proportionally many large, highly refractile, hydrated particles are seen. The blood sugar and urinary sugar show increase, and there is albumin, perhaps simply filtered into the urine because of the high degree of lysis, but often of a fatty globulin nature due to tubular degeneration. The behavior of the red cells to hypo and hypertonic salt solution is characteristic in demonstrating hydration. Either lysis with its hypertension or hydration with its hypotension may predominate. At any rate, both conditions have progressed beyond any hope of control through physiological means. The necessity for a reaction on the part of the body, in the way of a protective response, has become inoperative and the cells most handicapped by the difficulty attempt the conversion of the primarily lytic toxin, into a substance with inductive dispersion properties. They fail with the result that their product is a hydrator, fatally toxic even to the cancer cells that induce the change, as well as, a general producer of cachexia. Removal of the cancer growth does not materially alter the blood picture, demonstrating that the etiological toxin is still at work. But the longer the cancer activity is permitted, the greater the tendency towards muscular softening and the greater the likelihood for hemorrhage, because of fatty change. The very pronounced and fundamental general pathological changes that lead to and exist with cancer are so great, that it is impossible to claim good vitality or a good prognosis for any cancer patient, simply because fatal complications of the disease are apt to be imminent.

The return to health that we observe a year or so following the cure of a severe cancer case involves regeneration of much tissue and the renovating of the whole body, as it were. It is not surprising when the extensive organic degeneration of so many of these patients is considered, that they should come to termination aside from events taking place in the cancer growth. The surprising experience that with recovery from the cancer state, regeneration of a normal state with loss of both the interstitial changes of the early dehydration and the late degenerations of hydration, takes place so frequently.

These pathological states may be variously named, but usually an interstitial, or a glomerulo-tubular nephritis, a myocarditis, and hepatitis with a certain amount of fatty degeneration, is present. There may also be myelitis and the vascular changes consequent to hydration, such as military aneurisms. Functional glandular expressions of dehydration of exophthalmic goiter, so-called diabetes and of Addison's disease may be present. But, the removal of the etiological toxin can, in time, result in complete removal of all pathology. The cure of an advanced cancer case, therefore, involves a host of changes and presents an interesting study.