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The Biopathic Diathesis

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Editor's Note: *From its beginnings in character analysis in the 1920s, orgonomy addressed primarily psychic disturbances. In the early 1930s the nascent science extended into the somatic realm with the understanding of the relationship between emotional life and the autonomic nervous system in what Dr. Wilhelm Reich called "the basic antithesis of vegetative life." (1) In his investigation of this antithesis and attempts to understand exactly what flows out toward the world with pleasure and withdraws away from it with anxiety, Reich studied the simple, one-celled ameba. In the course of this laboratory research during the 1930s, he discovered bionous disintegration—the breakdown of organic and inorganic substances into membrane-bound, fluid-filled, energy-charged vesicles. He called these "bions," a term indicating the elemental particle of life on the border between living and nonliving nature.*

Concurrent with his revolutionary work on bions and bionous disintegration, Reich's discovery of muscular armor as the somatic functional equivalent of character armor allowed emotional therapy to be put on a solid biological foundation—emotional disturbances result from a tangible, physical armor in the body.

In 1939 Reich discovered orgone energy in his work with bion preparations. His discovery of orgone as the specific life energy provided the natural scientific basis for the psychoanalytic concept of libido and the understanding that emotions are the perceived movement of a real energy. The function of the orgasm is to regulate the economy of this energy and armoring was understood as the organism's attempt to contain painful, intolerable feelings. Armor, when chronic, interferes with the natural flow and discharge of this energy, thus creating an energetic stasis, an excess of energy, and a self-perpetuating cycle of neurosis.

Reich's continuing work with bions led him to investigate cancer. He observed and conceptualized that cancer results from the bionous disintegration of the body's tissues due to energetic shrinking of the organism with

concomitant, deep emotional resignation. With the publication of his groundbreaking The Cancer Biopathy *in 1948 (2), Reich addressed the vast realm of physical medical illness. He coined the term "biopathy" to refer to those diseases resulting from a* primary *disturbance in orgone energy functioning in the plasmatic system of the organism,*[1] *distinguishing them from nonbiopathic conditions, such as certain infections or trauma.*

Among the biopathic conditions, we can distinguish psychic biopathies, such as the character disorders, from somatic biopathies, the wide range of diseases well known and described (but not fully understood) by classical medicine such as cancer, coronary artery disease, high blood pressure, asthma, diabetes, autoimmune disorders, and so on. Reich made preliminary formulations of the determinants of particular illnesses, however the details were left to be elaborated by future scientists.

Dr. Robert Dew, a trained internist, made the first attempt to systematically address Reich's pioneering work in this field. "Biopathic Diathesis, Part I," (4) published in 1968 in the Journal of Orgonomy, *was the first of eleven seminal articles on the somatic biopathies that were published over the next ten years. The word "diathesis," from the Greek meaning "disposition," modified by "biopathic," appropriately and succinctly captured the essence of the daunting task of systematically organizing an understanding of the bioenergetic factors that dispose an individual to particular medical illnesses.*

Dew's title paper, being republished here, addressed general principles, reviewed the definition of somatic biopathies, and enumerated their general phenomenological features. He identified specific points about the somatic biopathies that distinguish them from nonbiopathic conditions. While Dew was clear he did not regard these as true "criteria," they remain helpful in evaluating the biopathic character of an illness.

Dew also surveyed and identified specific factors in the differentiation of somatic biopathies. For example, the intrauterine environment is discussed as an important factor that has life-long effects on the basic bioenergetic qualities of the organism. Thus, Dew drew an important distinction between the familial pattern of a disease and its genetic cause.

[1] *Reich identified the plasmatic system as the "autonomic life apparatus" which includes the autonomic nervous system and the vascular system (2). Konia also includes the immune system, as an extension of the vascular and the endocrine systems (3).*

Based on these factors, he systematically organized various diseases according to the bioenergetic qualities of energy level, capacity for lumination, armor, and bioenergetic behavior. On a spectrum of energy levels, he placed cancer at the low end and the inflammatory biopathies at the high end. He used diabetes mellitus to illustrate basic bioenergetic concepts in understanding the somatic biopathies in general and, in a masterpiece of functional thinking in medicine, proposed several quite original hypotheses about this particular disease.

In this and upcoming issues of the Journal of Orgonomy *a number of Dew's articles will be republished reflecting recognition of the groundbreaking nature of his work. Each of these articles from our archives will be presented with a companion article including updates from both the fields of orgonomy and traditional medicine to put the particular subject matter in a historical and contemporary perspective.* [Peter A. Crist, M.D.]

Introduction

Reich left an astounding legacy to medical science. The following partial list of his accomplishments is to orient us functionally and historically with regard to the biopathies. To those acquainted with his work, the overwhelming logic of his discoveries speaks for itself:

1. Character analysis
2. The principles of vegetative functioning; the function of the orgasm
3. Muscular armoring
4. Bions: the link between the non-living and the living
5. The discovery of orgone energy
6. The cancer biopathy: the organization of cancer cells from bions; the Reich blood test; the T-reaction
7. The DOR function; the DOR-buster

In *The Discovery of the Orgone,* Volume Two: *The Cancer Biopathy,* Reich discussed the variability of biopathic expression and laid the theoretical groundwork for the functional differentiation of the biopathies. His conclusions were based partly on his experiences in characteranalytic technique and orgone therapy. In other words, the

disease entities and syndromes which he included under the heading of "biopathy" were chosen because clinical work with patients having these disorders revealed their biopathic nature.

As far as I know, Reich did not attempt a comprehensive classification of the welter of disease states into biopathic and nonbiopathic categories. It is evident that he had not come to definite conclusions regarding certain conditions, e.g., tuberculosis and rheumatic heart disease. At any rate, I certainly have not yet had the opportunity to examine a sufficient number of patients with these diverse illnesses to come to firm conclusions on the basis of orgone-therapeutic and character-analytic techniques. It is obvious to me that this would take several lifetimes. Consequently, some of my inclusions in the list of biopathies are of necessity partially intuitive.

There are, however, certain general features (apart from energetic considerations) of the biopathies that tend to distinguish them from nonbiopathic conditions:

1. I can think of no biopathic syndrome for which classical medicine has a *clearly proven explanation.* They are diseases of "obscure origin." This is not intended as criticism, but simply a factual observation. Classical science has garnered a vast amount of information on the biochemical and biophysical concomitants of the biopathies, but it has been more or less continually frustrated in its efforts to find the agents responsible for their initiation.
2. It has become increasingly evident to the clinicians of traditional medicine that the emotional life of the patient is deeply involved in the behavior of what we call "the biopathy." In other words, it is generally agreed that there is a "psychosomatic component."
3. The biopathies most frequently present with a functional disturbance which precedes the gross morphologic abnormalities.
4. Biopathic processes are often characterized by prolonged courses punctuated by exacerbations and remissions for which

there is no apparent explanation. Ultimately, they lead to a state in which there are irreversible morphologic and functional changes.

5. The biopathies, even from a purely pathologic morphologic point of view, may involve the entire organism. That is to say, one frequently cannot point to a single anatomic nidus for the disease. This is particularly evident in such disorders as arteriosclerosis, hypertension, diabetes, and the collagen diseases. There are, however, notable exceptions: cholelithiasis, duodenal ulcer, uterine fibroids, etc.

On the above grounds, one's intuition takes on more concrete form. As was indicated, not every biopathy will satisfy each "criterion" listed; however, these points may be helpful in considering individual entities for inclusion in a theoretical compilation of biopathic disorders.

This paper, since it is an introductory survey, will not treat individual diseases in great detail except to illustrate certain principles. The main purpose here is to review our inventory of theoretical and practical tools for dealing with the problem of biopathic differentiation. It is hoped that the syndrome-specific mechanisms suggested below will serve as some sort of guide in approaching the biopathies. In addition, certain gaps in our understanding of biopathic specificity will emerge.

General Principles

Reich defines the biopathies as "all those disease processes which take place in the autonomic life apparatus, or simply, in the plasmatic system . . . that which all of these diseases have in common [is] a disturbance of the biological function of plasmatic pulsation in the total organism" (1: p. 129). He further defines the central mechanism of the biopathy as a disturbance in the discharge of biosexual excitation which has as its root stasis and chronic contraction of the autonomic apparatus (1: p. 132).

With these definitions in mind, we address ourselves to the problem of what determines the development of the biopathic process into a specific syndrome complex.

The Mechanisms of Biopathic Differentiation

It is quite clear that Reich related the sites of specific malfunctions, tumors, metastases, etc., to the location of chronic powerful muscle spasms, i.e., armorings. He also emphasized the role of total body charge in determining the fate of the organism. A third and most important determinant is how the organism reacts to chronic sexual stasis:

> The clinical comparison of the cancer biopathy and cardiovascular hypertension necessitated the assumption of a basically different energy process in the two:
>
> *In the cardiovascular biopathy* (stasis neurosis due to abstinence) *the sexual excitation remains alive,* biologically, physiologically, and emotionally. That is, the biological core of the organism, the autonomic vital apparatus, continues to produce energy to the fullest extent. The organism, in its state of contraction, reacts to this with outbreaks of anxiety or anger and with somatic symptoms such as hyperthyroidism, diarrhea, tachycardia, etc.
>
> *In cancer, on the other hand, the biological core reduces its energy production.* Thus as times goes on, the excitations and emotions become weaker and weaker (1: pp. 177-8).

The Role of Lumination in the Biopathies

It is evident that the capacity of lumination, whether we are dealing with unicellular or multicellular organisms, is related to the organism's total charge. Ontogenetically speaking, total body charge (and hence lumination) is one of the earliest and most fundamental determinants in biopathic differentiation. It is important here to review the reasons for this assertion.

We know that the human organism gains energy from the atmosphere by direct radiation, by respiration, and from food intake. Energy is discharged in childbirth, growth, mental and physical work,

the expression of emotion, the orgasm reflex, the maintenance of numerous metabolic processes, in excreta, and in combating disease. The lumination reaction in disease is perhaps second only to the orgasm reflex in its impact on the organism. Consider the events attending orgonotic lumination in illness.

1. Fever, tachycardia, diarrhea.
2. Leukocytosis, erythrocytosis, thrombocytosis (pancytosis).
3. "Mobilization" of the cells of the reticuloendothelial and lymphatic systems.
4. Increased antibody production.
   a) Against foreign antigens.
   b) Against body-own antigens, i.e., autoantibodies.
5. The elaboration of "chemotaxic factors" causing diapedesis and phagocytosis by the cellular elements of the blood.
6. Increased production of trophic hormones and the hormones of their target organs, e.g., adrenal gland.
7. Expansion of the blood plasma volume.

This incomplete list of course brings to mind an enormous number of disease states, as well as physiologic states such as pregnancy. One may well ask how these phenomena of lumination relate to the specificity of the biopathic syndromes.

To repeat, the capacity for lumination depends primarily on the energetic state of the plasmatic system and the biological core. As we have seen, in the cancer biopathy, the plasmatic system is contracted, its charge is low, and the core is inexorably depleted by the defense mechanism of bionous disintegration. The capacity for lumination is manifestly impaired, biophysically and emotionally.

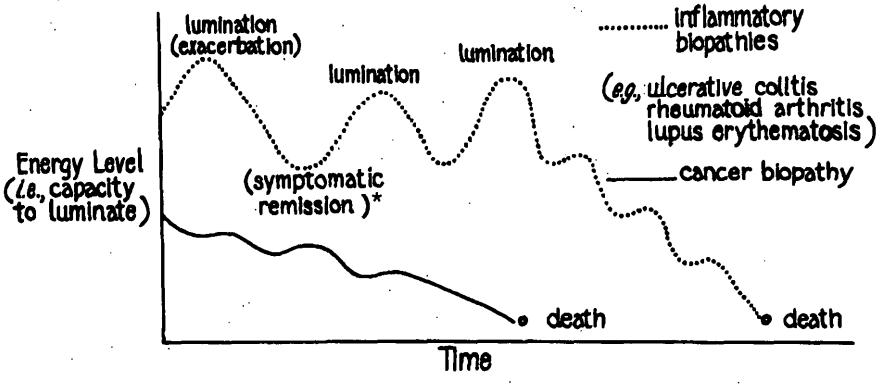
In ulcerative colitis, by contrast, the course is one of exacerbations and remissions which must correspond to lumination and contraction. The core expands violently against the contraction, the total body charge is high, intense lumination and parasympatheticotonia ensues, with fever, inflammation and ulceration of the colon, diarrhea, blood loss, and dehydration. The pathophysiologic events have as their

concomitant rage with anxiety. While bionous disintegration doubtless occurs, classical morphology reveals the bowel submucosa to be engorged with inflammatory cells. This is clearly a proliferation and infiltration by the body's own cells.

It is not my intention here simply to reiterate Reich's assumption that two different energy processes are at work (although this is certainly germane) but to emphasize the importance of luminatory capacity in explaining differences in the natural history, pathophysiology, and morphology of two distinct biopathies.

It seems reasonable, therefore, to propose the following scheme to depict the gross energy charges which distinguish the behavior of certain groups of biopathic syndromes.

It is obvious that, while the total energy trend is the same, the course is different. It is interesting, and quite pertinent, that the inflammatory biopathies affect younger (i.e., more highly charged) people. The cancer syndrome becomes manifest more commonly in older people.



The careful student of clinical medicine and pathology will raise serious objections at this juncture. He will correctly point out that

*As indicated here, the remission coincides with a decreased capacity for lumination. This, of course, depends upon: 1) the emotional push behind the lumination, i.e., the cause of the emotional upset behind the exacerbation may have been removed; 2) discharge of energy through the diseased mechanism itself, *e.g.*, fluid, electrolyte, blood loss, fever, etc.; and 3) a normal cyclic change in energetic status.

inflammatory phenomena, fevers and auto-immune manifestations occur in cancer patients. He will point out, conversely, that our so-called inflammatory biopathies occasionally undergo malignant degeneration or develop *incidental* tumors of various kinds, e.g., colonic cancer in colitis; gastrointestinal tract and renal tumors in dermatomyositis.

First of all, Reich never stated that cancer patients are *incapable* of lumination—quite the contrary:

> It should not be assumed that the organism accepts the gradual extinction without a fight. At a time when the orgonotic excitation of the total system decreases, the excitation may still be intense in individual cells, or cell systems, just as a suffocating organism defends itself against the final relaxation by clonismus (1: p. 178).

As long as there is some energy left in the biosystem, lumination will occur. This is implied in the left end of our scheme above. Nevertheless, the red thread of the biopathy remains biophysical resignation with bionous disintegration.

Secondly, while the red thread of the inflammatory biopathy is lumination against contraction, if the organism survives long enough, it may, energetically speaking, reach the same state as the shrinking biopathy. The occurrence of colonic malignancy in ulcerative colitis is rare and is usually a late development, i.e., when the case is "burnt-out"; in other words, when the capacity for lumination reaches the far right side of our graph. Clinicians are well aware of the observation that very old or chronically ill people often fail to become febrile in the presence of clear-cut infection. In fact, they may even exhibit subnormal body temperatures on these occasions. This is quite rightly regarded as an ominous sign. It reveals the total incapacity for lumination and hence signifies the energetic collapse of the organism. What is surprising, at the same time bearing out the integrity of Reich's assumption, is that malignant transformations do not occur more often than they do.

The Uterus and Heredity: The Biopathic Process and Intrauterine Development

Classical medical science has amassed a wealth of statistical information on genetic factors in the incidence of various diseases. It would be a grave error to brush this aside as so much mechanistic wishful thinking. To be sure, to some degree, the preoccupation with genetics in the biopathies (particularly cancer) reveals a certain deep feeling of futility, and certainly has been responsible for carrying modern research up blind biochemical alleys. Nevertheless, there are, clearly, enough examples of genetic diseases in the literature, e.g., phenylketonuria and various other metabolic and hematologic disorders, to warrant this line of approach even if only for purposes of exclusion. To my knowledge, however, none of the biopathies has been conclusively demonstrated to follow the pattern of Mendelian genetics. There is no dearth of documentation for the *familial* incidence of certain of the biopathies, e.g., thyrotoxicosis, diabetes mellitus, and rheumatoid arthritis. From the literature, one gets the distinct impression that the word "familial" is more or less intended to convey the supposition that a genetic mechanism does exist, but it simply has escaped elucidation until now. The concept of genetic "penetrance" was evolved to explain the discrepancies observed. I prefer to use "familial" in its strictest sense; it at least puts us on factual grounds.

Character analysis has clearly related the emotional atmosphere of the family to the structure and function of the individual. In *The Function of the Orgasm* and the *Mass Psychology of Fascism*, we see how character attitudes are anchored in each succeeding generation. Since character attitudes are *biophysical* the theoretical basis for the familial incidence of biopathic disease would seem to be on solid ground.

Reich also touched upon the intrauterine factor in the biophysical fate of human beings (1: pp. 339-40). We can only guess at the consequences to the fetus of spending nine months in a chronically spastic, characterologically "disgusted" uterus. It is reasonable to

assume that the vitality of the placenta and fetus will suffer or prosper along with any other organ of the mother's genital system. Inasmuch as embryonic development must involve lumination, streaming, and superimposition, it is entirely possible that congenital malformations are in part uterine in origin. The deleterious effects of drugs, viruses, and certain bacteria on embryogenesis would seem to bear this out. Likewise, we may conclude that, since character structure is the result of environment and the raw material (biosystem) upon which it works, the foundation for a particular biopathy or group of biopathies is laid in the uterus.

Here, again, I should like to return to the problem of genetics. There is probably as much evidence for the existence and behavior of genes as there is for subatomic particles, e.g., nuclear physics and animal husbandry both *work.* Furthermore, there is a mass of cytochemical and morphologic data which irrefutably links genes to reproduction and development. Yet, since all structure in nature is ultimately derived from orgone energy, so must the genes. It is apparent from cytologic observations that chromosomal division and segregation in mitosis and meiosis are subject to a more fundamental order of regulation than is inherent in the genes themselves. We would point out, in this connection, the similarities between the orientation of iron filings in a magnetic field and the chromosomal disposition in the metaphase of mitosis.

Thus, viewing the organism's environment as beginning in the uterus, we can understand the familial epidemiology of the biopathies. It is interesting that, in this light, the previously abandoned idea of Lamarck, i.e., the inheritance of acquired characteristics, appears to have some basis in fact.

The Theoretical Problem of the Metabolic Biopathy—Diabetes[1]

It may seem premature to introduce a specific disease entity at this point in a general survey of the biopathies. However, diabetes presents

[1]We refer here to "idiopathic" diabetes mellitus—not that secondary to surgical extirpation or inflammatory disease of the pancreas, or hemochromatosis, all of which account for very few cases.

us with some difficult theoretical problems which are relevant to the problem of biopathic differentiation.

Reich expressed the belief that diabetes mellitus was a biopathy based on a disturbance of pancreatic function due to a diaphragmatic block. There is reason to believe, however, that the problem is somewhat more complex than that. For one thing, the incidence of diabetes in the general population is roughly two percent, rising to as high as ten percent in the sixty-and-over age group. Even these high figures indicate that there are undoubtedly fewer cases of diabetes than there are severe diaphragmatic blocks. For the moment, though, let us hold this possible objection in abeyance. Does the disease have the general features of the biopathies which we outlined above?

1. *The cause of diabetes is as yet unclear.* The earlier assumption that it is due simply to a failure of the pancreas to produce adequate insulin has not withstood the scrutiny of more recent investigations. At the present time, as one authority observes, "The biochemical and physiologic explanation of the mechanism of diabetes is undergoing intensive reexamination" (2: p. 1175).
2. There is a "psychosomatic component." The factor of emotional stress in modifying the course of the disease is well known and needs no further documentation here.
3. The disease may be characterized by a long course with exacerbations and remissions leading to irreversible morphologic changes.
4. Diabetes is a generalized disorder with no recognizable local nidus of origin.[2]

Except for one final criterion, then, diabetes mellitus seems to fit the picture generally associated with the biopathies. The difficulty arises in identifying the specific functional disturbance preceding the gross morphologic changes.

At one time, the disease could be divided into a "latent" phase in which no biochemical abnormality could be demonstrated; a "preclinical" phase when a biochemical abnormality, e.g., glucose

[2]The idea that the pancreas is the anatomic "nidus" is no longer certain.

tolerance test, could be shown, but no clinically apparent derangement of carbohydrate metabolism is present; and, lastly, the "clinical" phase when polyuria, polydypsia, glucosuria, etc., are there for all to see. It would have been a fairly simple matter to regard the abnormality in carbohydrate metabolism as the functional disturbance antedating the morphologic changes of early and clinical diabetes. Recent work has shown, however, that changes in the basement membrane of the capillaries in muscle and renal glomerulus may occur long before the most sensitive tests of carbohydrate metabolism can detect any abnormality. A similar angiopathy has been demonstrated in women with so-called idiopathic edema of the extremities. There is a high incidence of diabetes in the families of these women and a significant number go on to develop diabetes. One currently proposed explanation for these capillary changes is that they are due to the deposition of a glycoprotein derived from glucose which has been shunted away from a normal metabolic pathway because of a specific pyruvate kinase deficiency.

These considerations may create some anxiety; we feel ourselves on the brink of a mechanistic biochemical morass. Recalling some of the features of diabetes may help guide us past these muddy waters.

First of all, the description of diabetes (by Artaeus in the 1st century A.D.) as "a melting down of the flesh and limbs into urine" is, to my view, a functionally accurate one. In essence, the plasmatic system is literally "melting away" and being excreted through the kidneys. The kidneys, beside their functions of maintaining acid-base and water balance, etc., normally excrete the "waste products" of metabolism, e.g., the products of tissue breakdown: *catabolism.* Might we not then regard the polyuria, ketonuria, hyperkaluria, and negative nitrogen balance (i.e., *catabolism*) of diabetes to some extent as a pathologic exaggeration of a normal process? If, for the moment, we accept this premise, we must go on to explain just what prompts this unique "exaggeration."

The basis of all the biopathies is more or less chronic sympatheticotonia. In the shrinking cancer biopathy, there is a gradual

loss of energy through the bionous disintegration of the red blood cells. I would suggest that diabetes mellitus is another form of shrinking biopathy in which the tissues themselves undergo disintegration, i.e., a massive catabolic "melting down." The red blood cell system is not directly involved, hence anemia is not a characteristic concomitant of the disease. The features of shrinking are certainly there. Autonomic and peripheral neuropathy (which is recently receiving much attention in clinical cancer disease) is a long-recognized complication of diabetes. I believe it is identical functionally to what is seen in cancer. The predilection for furunculosis, "premature" atherosclerosis, and indolent ulcerations of the lower extremities, all indicate *low tissue charge*, shrinkage of the plasmatic system and bioenergetic core. The poor handling of infection suggests impaired luminating capacity, as well. The extreme frequency of maturity-onset diabetes further supports the idea that this is closely related to the shrinking biopathies.

Let us return, now, to the biochemical aspects of this disease. We know that there is not necessarily a primary *underproduction* of insulin. It has been shown that in "latent" and even clinical phase diabetics there is increased insulin-like activity (ILA) in the serum. Radio-immuno assays (RIA) of insulin are also elevated. It is generally agreed that there is, at least initially, an *overproduction* of insulin, very probably in response to an increased *requirement*, and that the histologic morphologic exhaustion of the islet cells seen later on is a result of this. Why the increased insulin requirement?

One of the earliest observations in bion research was that the stronger orgonotic system attracted the weaker. The importance of this phenomenon in the process of absorption and assimilation is obvious. The removal of nutrients, e.g., amino acids, fats, carbohydrates, etc., from the blood stream by the tissue cells, is a further manifestation of this. Insulin is presumed to work at the cell membrane aiding the transport of glucose across to the cell interior. Insulin also promotes fat and glycogen synthesis and growth, and it

effects the *movement of potassium into the cells.* In short, insulin exerts a parasympathetic effect on the plasmatic system: it has a *life-positive action.*[3] If the assumption that tissue cell charge is low is correct, then the primary energetic drive (or pull) in glucose transport would be blocked. As a consequence, glucose piles up in the blood plasma and the stimulus for increased insulin production and release would result.

This mechanism would also explain other metabolic and morphologic derangements seen in diabetes, hypertriglyceridemia and hypercholesterolemia, for example. These substances are known to be implicated in atherogenesis. The failure of undercharged tissue cells to take up these materials from the blood plasma could account for the increased concentration and eventual deposition in the walls of the vessels.

Our hypothesis carries us further. Water and orgone energy mutually attract one another. If the tissue cells are losing charge, then water would tend to leave the tissues and enter the capillaries (carrying with it potassium and other electrolytes) where there is a relatively higher charge owing to the presence of erythrocytes and abnormally increased concentrations of unassimilated nutrients.[4] This would account for the poor tissue turgor, the "hypokalemia" evidenced on the ECG, and the normal or near-normal *serum* potassium not infrequently encountered in diabetic acidosis.

For the present, our discussion of the biochemical derangements of diabetes need go no further. If our theoretical assumptions are correct, then the chemical sequelae of the disease as described in the textbooks would make complete sense once the low tissue charge set them in motion. The functional disturbance in diabetes, in my view, is an imbalance in the relative charge of tissue cells and blood plasma which interferes with nutrient transport. This is "interpreted" by the endocrine system as a "shortage" of insulin. Apparently the successful treatment of the biochemical disturbance in diabetes with exogenous insulin occurs as a result of an overwhelming effect on the tissue cell

[3]We recall in this connection the insulin-antagonistic effects of epinephrine.
[4]Passive osmotic functions are, of course, also at work here.

membrane charge. The fact that the underlying biopathic process is untouched is borne out by the relentless advance of vascular, neurological, and other complications in the face of "good control" of blood glucose and glucosuria. Our hypothesis, however, gives rise to two difficult questions:

1. If the basic disturbance in diabetes is bioenergetic shrinking, should we not see a much higher incidence of cancer in diabetics?
2. Or, conversely, why don't cancer patients routinely develop diabetes?

There are no data, to my knowledge, which suggest an unusual correlation of cancer and diabetes. In fact, in the period 1914 to 1963, cancer as a cause of death in diabetes rose only from 4 to 9.5 percent, a rise which is easily attributable to the decrease in deaths due to coma and infection and the well-known increase in the incidence and diagnosis[5] of cancer itself. Deaths due to cardiorenal, i.e., non-neoplastic, causes in diabetes have more than tripled. There are isolated syndromes such as cancer of the uterine body and diabetes in which the two occur together with a higher than expected incidence; however, the frequency of this is not sufficiently high to provide much support for our thesis. How, then, can we account for the fact that what we call a shrinking biopathy does *not* especially enhance the *cancer* process?

I cannot give a clear answer to this question any more than I can explain why biophysical resignation and shrinking give rise to a cancer tumor in one patient and leukemia in another. The pathological behavior of these diseases suggests that they may occur at different energy levels. That is, we may postulate a (quantitatively) descending order of energy states, as follows:

[5]Cancer is more assiduously sought and more easily diagnosed today than it was fifty years ago.

BASAL ENERGY LEVEL
Total charge
Capacity to hold charge
Capacity to luminate

Likewise, we may postulate a descending hierarchy of conditions (with the latter corresponding to quantitative energy levels and qualitative energy functions), as follows:

CONDITION
Health
Inflammatory biopathies
Hypertensive cardiovascular biopathies
Diabetes
Leukemia
Cancer

The capacity for lumination seems less impaired in leukemia than in cancer. The closer epidemiological alignment of diabetes with the cardiovascular biopathies may indicate that the general energy level at which diabetes appears may lie somewhere between the leukemias and cardiovascular hypertensive disease.

The preceding schema is not an answer to the problem of differentiation of the shrinking biopathies; future experiences will doubtless reveal its crudeness, but it may at least provide a theoretical foothold in very difficult terrain.

Regarding the failure of cancer patients routinely to develop diabetes: We recall that our hypothesis implicates an energy imbalance between tissue and plasma to explain the metabolic defect. In the cancer biopathy, *both* tissue and blood plasma with blood cellular elements have a poor charge. This is evident from tumor cell formation and the T-reaction of the erythrocytes. Thus, we would not expect an imbalance between tissue and plasma comparable to that in diabetes. In other words, both orgonotic systems are decaying at the same rate, and the primary metabolic lesion of diabetes does not routinely develop. It should be remembered, however, that the occurrence of diabetes in cancer patients is *not uncommon.* Certainly it

is no less infrequent than the concurrence of arteriosclerotic heart disease and cancer. As a matter of fact, cancer patients not infrequently will die of some complication of arteriosclerosis rather than their neoplastic disease. This is, of course, particularly true in the older age groups. The overlapping of biopathies in general should remind us that patients may enjoy or suffer (as the case may be) a vertical mobility in their energy status as well as a certain "pleomorphism" in their response to contraction.

Let us now reconsider the role of a diaphragmatic block in diabetes. If we can no longer regard diabetes as primarily a defect in insulin production, then what role would the block play in its development? Obviously, as long as the pancreas can respond to the increased demands upon it, the appearance of clinical diabetes may be forestalled, i.e., increased endogenous insulin production can compensate for the failing energetic "pull" of the tissue cells. A severe diaphragmatic block, then, in disturbing pancreatic function, would hasten the emergence of signs of insulin lack.[6] Thus, the diaphragmatic armoring, like a glucose tolerance test or obesity, would bring out the much deeper disorder.

Yet, this raises an interesting problem. Why does the diaphragmatic block selectively affect the Islet cells' function and leave the acinar cells intact? This, again, might cast some doubt on the primacy of the diaphragm armoring in the development of diabetes. But, more important are its much broader implications with regard to the selective behavior of the biopathies in general. To clarify this, one would require a detailed knowledge of the discrete differences in susceptibility of various tissues to changes in oxygen tension and orgonotic charge. In this case, what are the relative capacities of acinar and Islet cells to "do without"? In this connection, we think of the relatively high incidence of cancerous change in rapidly dividing and regenerating epithelial tissues, e.g., gastric, bronchial, and the low incidence in supportive and connective tissue (smooth muscle and cartilage). Clearly, rate of cell growth and division are related to the

[6]The initial abnormal increases in insulin production seen in the early phases of the disease may in part reflect an over-response of the Islet cells to the suffocating effects of the diaphragmatic armoring.

proclivity for bionous disintegration in the face of hypoxia and hyporgonia. Thus, *the needs of the various tissues may be a further factor in the differentiation of the biopathies.* This will be discussed more fully elsewhere.

To summarize, the differentiation of the biopathies depends upon:

1. The intrauterine environment, particularly as it affects the vitality of the offspring, e.g., total body charge, capacity for lumination.
2. Character structure; character armor, location and severity.
3. Basic level of orgonotic charge.
4. The basic manner in which the organism reacts against contraction, i.e., lumination versus resignation.
5. The discrete differences in susceptibility of the various tissues to hyporgonia and hypoxia brought about by the armoring, etc.

Classification of the Biopathies on a Bioenergetic Basis

As we have gleaned from the previous material, the lack of accumulated clinical data of a character-analytic and orgone-therapeutic nature in the biopathies has created immense gaps in understanding their differentiation. Except in very general terms, it is impossible, at this point, to relate structure to individual diseases.

We do know that, although the fundamental disorder underlying all the biopathies is the same, the energy processes which result from this disturbance may vary. We believe, in addition, that total body charge and capacity for lumination undeniably affect the course of the biopathic process. On these premises, then, we shall construct a classification of the biopathies, including as much relevant factual information as possible. Speculative statements will be followed by a question mark in parentheses.

Because of the lack of detailed knowledge in each case, the number of conditions listed is limited. Also, because of the welter of states involved, no attempt at comprehensiveness is made. We strive

Type of Disease Process	*Energy Level*	*Capacity for Lumination*	*Armor*	*Bioenergetic Behavior*
Health	High	Vigorous, appropriate	Absent or temporary	1. Emotions vividly felt; expressed in appropriate *somatic* motor activity. 2. Energy discharged.
Non-biopathic	High	Vigorous, appropriate	Temporary; may account for "susceptibility"	1. Emotions vividly felt; expressed in appropriate *somatic* motor activity. 2. Energy discharged.
Inflammatory Biopathy Examples: ulcerative colitis and collagen diseases	High	Vigorous, inappropriate: *e.g.*, unexplained fever, auto-immune phenomena	May dictate locale of reaction, *e.g.*, ulcerative colitis: energy trapped in abdominal segment (?)	1. Stasis: sexual excitation alive. 2. Rage felt, but with anxiety. Rage expressed in *visceral* motor activity, *e.g.*, causes diarrhea in ulcerative colitis.
Cardiovascular— Hypertensive Biopathy Renovascular Biopathy Cerebrovascular Biopathy	High	Virgorous, generally appropriate	Dictates locale of reaction, *e.g.*, pelvic block in hypertensives traps energy in upper segments (chest)	As above. Rage felt, but with anxiety. Increased vasculomotor activity, *e.g.*, vasospasm and finally arteriosclerosis.
Metabolic biopathy: *e.g.*, diabetes	1. Decreased 2. Tissues cannot hold charge (?) 3. Disproportionate loss of charge between blood plasma and tissues (?)	Impaired, but appropriate	*e.g.*, Diaphragmatic block may precipitate the clinical appearance of the disease by disturbing pancreatic function (?) Other blocks may influence complications, *e.g.*, renal, cardiovascular, eye	1. Rage reaction (?) 2. Resignation (?) Shrinking biopathy(?)
Leukemia Lymphomas (?)	1. Markedly decreased	Intense and inappropriate Directed against patient's own RBCs which are abnormal (T-reation)	Role of specific armorings not understood	1. Resignation 2. T-reation 3. Cells of reacting system abnormal
Cancer	1. Severely decreased	Poor and sometimes inappropriate. Directed against tumor cells. RBCs lost as defense mechanism	Dictates locale of tumors and metastases	1. Resignation: "chronic emotional calm" 2. Sexual excitation dead 3. T-reaction 4. Cancer cells

here only to indicate some of the lines along which the diseases seem to classify themselves.

Conclusion

It is said that the fruitfulness of research depends mainly on asking the right questions. In this general survey, I have alluded to some basic principles, elucidated by Reich in *The Cancer Biopathy,* in an effort to set in relief possible mechanisms of biopathic differentiation. In the discussion of the metabolic biopathy, I attempted to utilize those principles to explain more fully some of the features of diabetes. Whether or not my handling of these concepts is valid remains to be seen. However, questions have emerged which underscore tremendous gaps in our knowledge of the effects of disturbed orgonotic pulsation on the human organism.

We cannot, as yet, fully account for the *selectivity of reaction* in the different biopathies, e.g., why, if adrenal tumors are due to abdominal blocks, do they occur either in the medulla or the cortex in a given patient but rarely in both layers simultaneously? Why does one patient get regional enteritis, and another get ulcerative colitis, when the small bowel and colon originate in the same segment? Why does biophysical resignation lead to cancer in one and leukemia in another?

Related to this problem is the question of why certain illnesses involve many segments (e.g., lupus) and others affect only one (e.g., cholelithiasis).

The answers to these and other enigmas will doubtless carry us deep into involvement in cellular physiology, embryology, and biochemistry.

The prospect, quite properly, makes us uneasy. Mechanistic science has sailed these same waters and has run aground. Hopefully, the fundamental tool of orgonomic research, *functional thinking,* will rescue future investigators from this fate.

Editor's Note References

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