

The Journal of Ergonomics

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Virginia L. Whitener, Ph.D.

An Overview of the Biopathic Diathesis: Update, Application and Analysis

Peter A. Crist, M.D.

General Introduction

In the three and a half decades since the publication of Dr. Robert Dew's first article on the somatic biopathies (1), modern science and medicine have amassed a wealth of facts about biochemistry and the molecular structure of enzymes, hormones, neurotransmitters and receptors, and other biologically active proteins and peptides. The extraordinary complexity of the cellular and molecular basis and mechanism of immunity, inflammation, endocrine function, neurochemistry and genetics has been investigated and described.

A functional energetic perspective of medical illness, however, provides a framework within which to better comprehend many of these details and to develop functional approaches to the treatment of specific diseases. As such, this article serves as a companion piece to the republication of Dew's first article. It will review some of the vast amount of information made available by modern science and place it in a functional perspective. It will also review some of the advances in organomic understanding of the somatic biopathies since the article was first published. The focus will be on the general topic as outlined in that article, applying some of the principles Dew laid out and analyzing afresh some of his formulations.¹

Triumphs and Limitations of the Mechanistic Approach to Medicine

Most of the triumphs of modern medicine have been attained by the application of mechanistic thinking to those medical illnesses where a specific cause or a definitive structural change can be identified.²

¹Each subsequent article that accompanies the republication of one of Dew's "Biopathic Diathesis" articles will go into more detail with updated information on specific diseases.

²Mechanistic thinking reflects the view that nature is made up only of material substances and functions like a machine.

Examples include nonbiopathic physical trauma and infections,³ as well as conditions, whether biopathic or nonbiopathic, in which there is organ damage, joint destruction, vascular or valvular heart and large blood vessel pathology. These can be corrected with modern surgical technology, e.g., organ transplantation, joint replacement, open-heart surgery, and vascular grafts. Contemporary medicine has been less successful with disorders that manifest primarily in disturbances of physiological function, and has had even less success with disturbances of emotional function, despite media reports to the contrary.

Thus, despite advances in scientific knowledge and technology, the illnesses we refer to as the somatic biopathies—cancer, atherosclerosis, coronary artery disease, hypertension, diabetes, and a wide range of immune illnesses, among others—continue to be major causes of morbidity and premature death in the Western world. Nowhere, aside from psychiatry, are the limitations of the mechanistic approach more evident than with this disease group.

Disease Causation and the Differentiation of Biopathic from Nonbiopathic Disorders

Dew introduced a systematic approach to distinguishing biopathic from nonbiopathic disorders. He identified five specific characteristics of the somatic biopathies: 1. These diseases are of unknown or obscure etiology. 2. They have an emotional or psychosomatic component. 3. There is generally a disturbance in function which precedes gross morphologic abnormalities. 4. Most have a prolonged course punctuated by exacerbations and remissions, ultimately with irreversible morphological and functional changes. 5. Often the disease involves the entire organism without a single anatomic focus. While Dew was clear he did not regard these as true “criteria,” these features remain helpful in evaluating the biopathic character of an illness (1:156).

³Some of modern medicine’s triumphs over infectious disease in the 20th century began to erode in the last decade when the serious problem of antibiotic-resistant microorganisms appeared and concerns were raised about the possible long-term effects of vaccines.

In discussing his first “criterion,” Dew states, “I can think of no biopathic syndrome for which classical medicine has a *clearly proven explanation*. They are diseases of ‘obscure origin.’” [italics in the original, 1:156] He clearly states that this is not intended as a criticism but as a factual observation. At first it seems odd to define something by the criterion that we do not understand it.⁴ However, when viewed in light of the basic features of mechanistic thinking, this criterion takes on a deeper significance. In other words, the very character of biopathic diseases, i.e., those that result from a primary disturbance of orgone energy pulsation, dictates that they will not have an identifiable external etiology and a clearly proven mechanistic explanation because they are outside the realm of mechanical functions.⁵ In using Dew’s first criterion, the emphasis must be on “clearly proven,” because mechanistic thinking tends to look so assiduously for cause and effect, that cause may be attributed when there is in fact no more than an association between phenomena. This particular group of diseases especially provokes the tendency to premature attribution of causation, which interferes with further exploration and impedes a true understanding of the disease.⁶

For example, peptic ulcer disease was long understood to have significant emotional factors in its pathogenesis, described as being due to “Hurry, worry and curry.” In the 1980s an association was found between ulcers and the bacterium, *Helicobacter pylori*. This bacterium was then considered to be “the cause” of ulcers and all other factors such as emotional stress were essentially dismissed. The standard treatment became a course of antibiotics. Subsequent research has shown that perhaps half the world’s population has this bacterium. The obvious question arises: Why do some people with the bacteria develop the disease and others not? Continuing with the mechanistic approach, scientists have identified a disturbance in the connection

⁴This criterion becomes increasingly obsolete the more a disease is understood, i.e., has a “clearly proven explanation,” in functional energetic terms.

⁵It might even be said that the very idea of disease etiology, in its common usage, comes from the mechanistic view.

⁶To suspend premature explanation of a phenomenon requires the researcher, scientist, or clinician to tolerate the anxiety associated with uncertainty and not knowing.

between the cells that line the stomach in those who develop the disease. This observation could be helpful in understanding the disease process if viewed from a functional perspective, specifically, the capacity of the tissues to hold their energetic charge and remain cohesive. On the other hand, the mechanistic approach will likely lead the investigation down another blind biochemical alley looking for the protein that accounts for the disturbed connection between cells (2).

Another notable example of the adverse effect of premature conclusion about disease causation is found in the history of diabetes mellitus research. Charles H. Best was one of the principal investigators in the original research that led to the discovery of insulin in the early 1920s.⁷ This discovery and the commercial production of insulin and its use has allowed countless people to extend their lifespan. Best went on to prominence as the preeminent physiologist in the field of diabetes research and was president of the American Diabetes Association for many years.⁸ Meanwhile, because diabetics lived longer, previously unknown features of long-term diabetes were revealed, such as significant cardiovascular, renal and ophthalmologic complications, even in the face of good blood sugar control with insulin. Some thirty years after the Nobel Prize was awarded for the discovery of insulin, Best, now recognizing that diabetes was much more than a disease of insulin deficiency as had originally been concluded, was reported to have said that the discovery of insulin had set back understanding of the disease by at least thirty years (3).

In the absence of a clear understanding of the cause of disease, it has become commonplace in medicine to identify risk factors for developing an illness, especially those for which a clear cause has not

⁷An indication of Best's stature in the field is found in an interesting footnote to the history of medicine. Best, as the junior member of the research team, was not named by the Nobel Prize committee when it awarded the 1923 Nobel Prize in Medicine and Physiology for this discovery. The principal researcher, Fredrick G. Banting, protested to the committee to no avail and decided to share his prize money equally with Best. This prompted the co-recipient of the prize, John J. R. McCloud, the biochemist who had determined the chemical structure of the molecule, to share his prize money with his co-worker, James B. Collip.

⁸Tragically, Banting's career ended with his death in a plane crash during World War II.

been found, i.e., biopathies such as cancer, heart disease, hypertension. Risk factors are nothing more than associations. As with all associations, they may be helpful in understanding a disease but may also confuse the issue and prevent true understanding when they are misinterpreted as causative. For example, cigarette smoking is widely regarded as “the cause” of lung cancer and statements to this effect are even required by Federal law on packs of cigarettes. But cigarette smoking is also considered a risk factor for atherosclerotic heart disease. Why does one smoker develop cancer, another heart disease, and why does still another remain disease-free?

Modern medicine has a number of terms to describe those illnesses for which a specific cause has not been identified. Besides being described simply as “diseases of unknown (or obscure) origin,” they are often identified as “essential,” as in essential hypertension, essential epilepsy, etc., or described with the modifying term “idiopathic,” as in idiopathic thrombocytopenic purpura, idiopathic cardiomyopathy, idiopathic pulmonary fibrosis, etc. Both the word “essential” from “essence” or something inherent, and the word “idiopathic” from the Greek “idios” meaning “self,”⁹ convey the sense of internal, inherent, or individual factors that dispose the person to the disease.

These so-called “host factors” are being increasingly acknowledged in many areas of medicine. Such factors account for one person having an increased risk of cancer from smoking while another is at greater risk for heart disease.

There is no absolute, mechanical distinction between biopathic and nonbiopathic, however. For example, not everyone exposed to an infectious agent will become infected. Such individual susceptibility represents the biopathic component of infection. In many cases even accidents and their resulting trauma have a biopathic component because contactlessness may cause an accident by interfering with the individual’s ability to respond appropriately to an emergency situation or cause the injured person to fail to seek appropriate treatment. If

⁹A medical professor once said the origin of the word “idiopathic” was “idio- for ‘I don’t know,’ -pathic, ‘anything about it.’”

chronic, contactlessness may even cause the individual to be “accident prone.”

Biopathic Differentiation¹⁰

In addition to identifying “criteria” which differentiate biopathic from nonbiopathic conditions, Dew identified five factors important in determining the development of a specific somatic biopathy (1:168). He does not ascribe any significance to their particular order. To clarify the factors and their functional relationships, minor wording changes have been made and the list reorganized to put them in order from the most general to the most specific.

1. Basic level of orgonotic charge, as determined by genetic factors.
2. Intrauterine environment as it affects vitality, e.g., total body charge, capacity for lumination.
3. Character structure; somatic armor, location and severity.
4. Basic manner of reaction to contraction, i.e., lumination or shrinking.¹¹
5. Differences in susceptibility of tissues to hyporgonia and hypoxia as a result of armor (1:168).

In addition to placing these factors in order from the general to the specific, they are also organized along an approximate time line from earliest disturbance on. The first is a basic factor that refers to the individual’s innate, natural, energetic capacity. (This may be determined at the moment of conception by energetic processes involving the superimposition of egg and sperm as well as what are called “genetic” factors.) Second, are the effects of the intrauterine environment on energetic vitality and its vicissitudes. Third, are factors that influence the energetic function of a particular somatic component, i.e., the segment or organ system contained therein.

¹⁰The differentiation of psychic biopathies has been extensively presented in the orgonomic literature (4). Dew’s articles address the differentiation of somatic biopathies.

¹¹The word “shrinking,” a somatic realm term, replaces Dew’s “resignation,” a psychic realm attitude.

Fourth, is the factor that determines the specific type of reaction within that organ system, i.e., lumination or shrinking. Fifth, are the particular armor-determined susceptibilities of specific tissues within that organ system.

Advances in Orgonomic Knowledge

The Psychosomatic Relationship

Since the original publication of Dew's article there have been significant clarifications of the functional basis of psyche and soma and their relationship (5). Konia's work on orgonotic contact, in particular, has simplified comprehension of psyche and soma: the psyche is related to functions of the whole organism, whereas the soma is related to component functions of the organism (6). Every biopathy has both psychic and somatic aspects. The psychic aspect of a biopathy affects the person *as a whole* while the somatic aspect affects a *specific* segment, organ or part. The psychic biopathies are identified by the various character types. Also, an individual has only one character diagnosis, i.e., one specific, primary psychic biopathy that lasts for life, but can have many somatic biopathies. Psychic biopathies are determined by armoring in the segments with erogenous zones, whereas somatic biopathies result from armoring in any of the segments (7:224). This makes sense since the erogenous zones, although anatomically localized to particular segments, have an effect of exciting the whole organism. Every psychic biopathy has specific patterns of somatic armoring, while every somatic biopathy has an underlying psychic biopathy.

Many biopathies that show clear physical manifestations are primarily psychic biopathies. Cancer is a psychic biopathy as it relates to the attitude of resignation of the whole organism, with the malignant tumor being the somatic aspect of the biopathy with relation to the specific component part, i.e., the organ or organ system. This view of cancer is supported by Reich's observations that if solely physical, somatic interventions were used to treat the tumor in

cancer patients, the overall disease and the outcome was not improved unless the attitude of resignation was addressed (8). This view is also supported by the work of LeShan (9) who successfully treated cancer patients with psychological, character interventions alone. Similar reconsideration of other biopathies that have long been considered somatic should be undertaken. For example, diabetes, as a peripheral shrinking biopathy of the whole organism that relates to being “too sweet” (10:184) and unable to mobilize anger, is psychic, but the specific localized effect on the pancreas, especially in Type I, is the somatic aspect. Obesity affecting the whole organism is likely essentially a psychic biopathy.¹²

Orgonometry

There have been many advances in the use of orgonometry to clarify the relationship of functions. This has been particularly helpful in understanding orgonotic (i.e., energetic) contact and the functions differentiated from it (6,12). This work has great potential to further clarify the determinants of biopathic differentiation because it should eventually lead to identification of a specific dysfunction and its realm at the basis of a particular disorder. It is now known, for example, that pulsation is in a more superficial realm than excitation and perception. Such orgonometric analysis suggests that the basic disturbance in some biopathies may be in a realm deeper than pulsation, such as that of excitation, perception or even orgonotic streaming. Should this prove to be so, the definition of biopathy as a disturbance of pulsation will have to change.

Clarification of the Plasmatic System

Reich used the term “biopathy” to refer to those diseases resulting from a primary disturbance in orgone energy functioning in the plasmatic system of the organism. He identified the plasmatic system as the “autonomic life apparatus,” which included the autonomic

¹²Harman has even considered the possibility of obesity as a “social biopathy,” i.e., one that is a primary disturbance in the social realm of personal interactions (11).

nervous system and the vascular system (1,4b,8). A major advance in the understanding of the somatic biopathies was made with the identification of the immune and endocrine systems as components of the plasmatic system (7:224,13).

Contact in the Somatic Realm

Using the tool of orgonometry, Konia's investigation of the immune and endocrine systems showed that understanding the functions that comprise contact can be applied to the somatic realm (13). Orgonotic contact, based on the integration of excitation and perception, is dependent on orgonotic streaming (the natural, spontaneous movement of primary orgone energy), the common functioning principle of these paired functions (6). This provides a bioenergetic basis for understanding the biology of "receptors" and biologically active substances. The mechanistic approach is to measure quantity, either excess or insufficiency. In contrast, a functional approach allows us to begin to assess problems of loss of contact on a *cellular* level. Disturbances in receptor function, i.e., somatic contact, must result from disturbances in excitation, perception, or orgonotic streaming itself. The implications of this understanding are profound for all of biology and medicine, but particularly for neurology, immunology, and endocrinology.¹³ The subject of somatic contact will be revisited and application of these formulations made in discussion of autoimmune disorders and diabetes below.

The Role of Lumination as a Determinant in Biopathic Differentiation

The role of lumination is important in all biopathies, both psychic and somatic. Lumination, in fact, has a specific role in the formation of repressed and unsatisfied character reactions (14). Energy excitation above the lumination point is perceived as sexual excitation. A repressed or an unsatisfied block occurs when a particular impulse during its natural stage of development is consistently frustrated when

¹³The endocrine system is well known. The paracrine and apocrine systems, while not as well known, are just as profoundly important.

its level of excitation is below (in the case of repressed) or above (in the case of unsatisfied) the lumination point. Also, this formulation focuses on a point in the four-beat formula (tension → charge → discharge → relaxation) at which the functional disturbance occurs.¹⁴ In other words, some biopathies result from an intolerance of expansion (the individual is said to be “stuck in contraction”), while others result from an intolerance of contraction (the individual is said to be “stuck in expansion”). In the former, the capacity for tension and charge is disturbed. In the latter, energetic excitation and charge is tolerated but there is typically a block that prevents discharge and relaxation.

Dew described the role of lumination in somatic biopathic differentiation noting the basic difference between cancer at the low energy end and inflammatory biopathies at the high energy end. We can postulate luminatory and shrinking (resignation) biopathies within each organ system based on the tendency toward either lumination or shrinking (resignation)¹⁵ (15). There are many examples well known to traditional medicine but not understood in bioenergetic terms. Among the pulmonary biopathies, individuals with emphysema who fight hard to breathe to maintain normal blood gas levels are referred to as “pink puffers,” while “blue bloaters” are those who apparently resign themselves to living with elevated carbon dioxide in their blood.¹⁶ Among the bowel disorders there is ulcerative colitis with a stormy, inflammatory course and Crohn’s disease with a chronic, smoldering course. In cardiac disease there are patients with a tendency toward acute myocardial infarction (MI) and those with “silent” MI’s and chronic congestive heart failure. In neurology there are two courses of illness in multiple sclerosis: one characterized by acute flare-ups and another chronic, smoldering type

¹⁴Reich originally used the four-beat “orgasm formula” to describe the orgasm function but subsequently saw that it had applicability to a wide range of life functions and named it the “life formula” (17:255).

¹⁵These are the somatic equivalent of specific unsatisfied and repressed types in the psychic realm.

¹⁶This example among the pulmonary biopathies is the only one of these pairs for which traditional medicine recognizes an emotional attitude related to the different conditions.

(16). In thyroid disease there is Graves disease with acute thyrotoxicosis and Hashimoto's thyroiditis with resultant hypothyroidism. Among the arthritides there is acute, inflammatory rheumatoid arthritis and chronic, smoldering osteoarthritis.¹⁷ Thus, the same formulation of "stuck in contraction" (the chronic, smoldering types) or "stuck in expansion" (the inflammatory types) apparently applies to the somatic biopathies as it does to the psychic biopathies mentioned above.

An understanding of the specific disturbance in the four-beat cycle has therapeutic implications. The therapist must address the particular block in order to restore pulsation. For example, those with inflammatory biopathies are in a state of over-expansion. They can tolerate energetic excitation and charge but typically have a block to discharge and/or relaxation. Therefore, discharge is the function that must be addressed. Those with shrinking-type biopathies cannot tolerate tension (the tissues break down on swelling) or charge (intolerance of charge). In such cases these functions, rather than discharge or relaxation, must be addressed.

Illustration of a Case with Several Physical Illnesses

The clinical case of a man who had mononucleosis, Hashimoto's thyroiditis, and a myocardial infarction during the course of medical orgone therapy illustrates many of the principles described in Dew's original article, and supports some new hypotheses about specific aspects of the somatic biopathies. A discussion of the case leads into a more general discussion of new knowledge gained in both traditional medicine and organomic research over the past thirty-five years.

Case Summary

The patient is a fifty-seven-year-old, single, male, free lance writer who originally began therapy with a complaint of "not enough energy." He had heard that medical orgone therapy dealt with energy and said, "That's for me."

¹⁷Type I and Type II diabetes also appear to fit this pattern, as will be discussed below.

On the first visit he marched into my office with head erect, as if perched on his neck. He was superficially cooperative and friendly in a way that seemed to hide a subtle wariness and a judgmental attitude. I had the impression he was covertly scanning in detail me, my office and everything in it with a subtly critical eye. He said nothing overtly critical and seemed cautious with a restrained bravery.

On biophysical examination, he was thin with decent muscular development more prominent in the legs than in the upper body. His eyes had a sharp, subtly piercing quality, and there was significant tension in the occiput. His jaw was tight and the cervical segment was also armored. His voice was harsh, as if coming through a restriction. His chest moved very little and was held mostly in expiration, although it could move when he was asked to breathe. With fuller respiration, color came to his face but did not extend down into the body—there was a clear demarcation at the top of the cervical segment. The diaphragm showed resistance to pressure below the xyphoid and this segment also showed little evidence of movement. His pelvis was held stiffly and his legs had significant tension in both calves and thighs.

In summary, on initial biophysical examination, he breathed shallowly with his chest held in expiration. There was major holding in the ocular, cervical and diaphragmatic segments. There was also armor in the oral and pelvic segments.

Over the course of his therapy several character reactions were identified and addressed: his critical eye was expressed as “the inspector general” and he tended to react to everything in a subtle way, “the subtle man.” For example, he subtly pushed himself, overriding sensations of tiredness or awareness of any form of emotional contraction. (In this way he avoided experiencing fear and sadness.) Also, there were no dramatic breakthroughs in his therapy but rather slow, steady progress with subtle changes in attitude, behavior and feeling.

Another character attitude, being “the brave soldier,” was associated with a particularly vivid, poignant childhood memory. He

recalled being sent off to summer camp at a very young age. After learning camp songs, whenever he felt afraid, sad or lonely, he sang to himself, "Pack up your troubles in your old kit bag and smile, smile, smile." These character attitudes reflected and constituted the psychic aspect of his pulsatory disturbance. Manifesting on the surface primarily as an intolerance of contraction, on a deeper level there was a significant intolerance of expansion.

In 1994 he had a bout of mononucleosis, following which his complaints of low energy and exhaustion were even more pronounced. This illness was understood as very likely related to his tendency to push himself. He made contact with how he just did not want to accept the reality of his limited supply of energy.

In the late 1990s, because of his continued complaints of "low energy," he was urged to ask his medical doctor to include a thyroid function panel (TFP) on routine physical screening blood tests. (The table on p.82 and 83 summarizes pertinent laboratory test results.) In October 1998 a TFP showed a mild elevation of thyroid stimulating hormone (TSH), with other thyroid values normal. Repeat TFP a few weeks later showed all values in the normal range. A Reich Blood Test (RBT) around this time showed a predominant T-reaction.¹⁸ (These findings will be discussed below.) Follow up TFP one year later showed significantly elevated TSH, with other values normal. Several months later when his internist recommended thyroid hormone replacement, Bill was urged to have this physician pursue further work-up to determine the cause of the hypothyroidism. A presumptive diagnosis of Hashimoto's thyroiditis was made in January 2001 when a test for anti-thyroid antibodies (Ab) proved positive for thyroid peroxidase Ab and thyroid ultrasound showed nodularity throughout both lobes of the gland consistent with thyroiditis.

¹⁸Reich described two antithetical reactions in bionous breakdown. The healthy, life-positive one he called "B" with large, highly-charged bions; the other unhealthy, life-negative reaction he called "T" for the small, energy-starved T-bacilli he discovered in his research on cancer. "T" is derived from "Tod," the German word for death (8).

In addition, routine blood testing in November 1999 showed a mild elevation of blood cholesterol (210), which had been normal (178) in May 1995.

He suffered a “mild” myocardial infarction in February 2003. He was hospitalized following 8 hours of chest and general “discomfort” (“not pain”). While there were no EKG changes, blood tests were positive for elevated cardiac enzymes. Coronary catheterization showed 100% occlusion of the right coronary artery and narrowing of two branches of the left anterior descending artery.

Through all of this, he had significant improvement in his work life and social functioning, but in his love life he remained largely unsatisfied. Currently, he continues to complain of “low energy.”

Case Discussion

This case provides a good opportunity to discuss the differentiation of biopathic from nonbiopathic disorders and illustrates principles of somatic biopathies in general.

Mononucleosis Postulated as an Acute Biopathic Condition

Mononucleosis is a good example to illustrate the difficulties in differentiating a biopathic disorder from a nonbiopathic disorder. How does it fit the various “criteria” listed by Dew?

1. Is the disease of obscure origin? The answer to this question depends entirely on the perspective with which we view mononucleosis. *Harrison's Textbook of Medicine*, one of the standards of internal medicine, unequivocally asserts that:

Epstein-Barr virus (EBV) is the cause of heterophile-positive infectious mononucleosis (IM), which is characterized by fever, sore throat, lymphadenopathy, and atypical lymphocytosis. (18:1109)

Several problems arise in evaluating whether EBV is “the cause” of mononucleosis. First, the clinical picture of mononucleosis is also found with other viruses most notably cytomegalovirus (CMV). It is

difficult to ascertain what is cause and what is association because as *Harrison's* continues:

EBV is also associated with several human tumors, including nasopharyngeal carcinoma, Burkitt's lymphoma, Hodgkin's disease, and—in patients with immunodeficiencies (including AIDS)—B cell lymphoma. (18:1109)

EBV is also associated with chronic fatigue syndrome. Further raising questions about EBV as a specific cause of mononucleosis is the fact that epidemiologically:

EBV infections occur worldwide. These infections are most common in early childhood, with a second peak during late adolescence. By adulthood, more than 90% of individuals have been infected and have antibodies to the virus. IM is usually a disease of young adults. (18:1109)

This is attributed in a vague and unexplained way to socioeconomic status and hygiene:

In lower socioeconomic groups and in areas of the world with lower standards of hygiene (e.g., developing countries), EBV tends to infect children at an early age, and symptomatic IM is uncommon. In areas with higher standards of hygiene (e.g., the United States), infection with EBV is often delayed until adulthood, and IM is more prevalent." (18:1109)

It is apparent from the above discussion that the cause of mononucleosis is not "clearly proven."

2. There is much to suggest that the emotional life of this patient was a significant factor. Those who have contact with individuals with mononucleosis consistently have the sense that there are emotional factors involved, especially since it often occurs during periods of increased emotional stress. The fact that it frequently occurs in the adolescent population is also suggestive. A hypothesis that adolescence and its emotional turbulence is a factor in the etiology of the illness is just as plausible as the hypothesis of socioeconomic and hygienic factors.

Table of Test Results (Part 1)

Date of Test	5/27/95	11/14/98
<u>Reich Blood Test</u>		
<i>Saline</i>		
breakdown rate	very slow (2%@50 min)	normal (25%@39min)
appearance of bions	medium (in frame)	small (throughout cell)
T spikes	none	widespread T spikes
<i>Autoclave</i>		
cohesivness of clot	fair	poor
fragment size	medium	small to fine
fluid	clear	clear
microscopic		
T vs B reaction	strong B	low B strong T

Date of Test	10/19/98	11/2/98
<u>Thyroid</u>		
TSH (0.40-4.20 MU/L)	4.6	3.3
T3 Uptake (27-41%)	32.6	36.5
T4 Thyroxine (4.5-12 MCG/dL)		6.7
T4, Free (1.75-3.8 units)	2.2	2.47
Thyroid peroxidase Ab (0-2 IU/mL)		
Thyroglobulin (0-2 IU/mL)		
<u>Lipids</u>		
Cholesterol (120-199mg/dL)	178	
HDL Chol. (35-59 mg/dL)	50	
Chol/HDL ratio (3.9-6.6)	3.6	
LDL Chol. (75-129mg/dL)	100	
Triglycerides (40-199 mg/dL)	142	

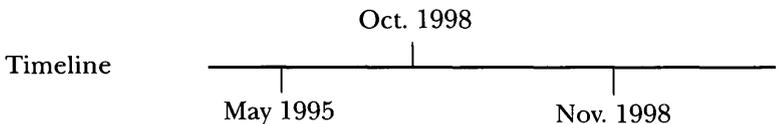


Table of Test Results (Part 2)

11/28/98

4/7/01

slow (19%@44min)
normal (in frame)
none

normal (33%@45min)
small (throughout cell)
T spikes present

fair
medium to small
turbid

indistinct clot
small
slightly turbid

low B reduced liveliness

moderate B

11/3/99 1/18/01 1/30/01 2/26/01 5/9/01 8/17/01

8.1	8.4		6.5	3.44	8.6
32	30.3		31.2	32.2	29.5
6.5	6.8		8	5.4	5.8
2.1	2.1		2.5	1.74	

>70

<2

210	225
49	49
4.3	4.6
122	150
194	132

Nov. 1998

Jan. 2001

Apr. 2001

Aug. 2001

Nov. 1999

Feb. 2001

May. 2001

3. It is not clear what the functional process is that might precede the gross morphologic abnormalities.

4. Mononucleosis generally presents as an acute or subacute illness without clear long-term sequelae, although there are reported cases of chronic mononucleosis related to chronic fatigue syndrome.

5. Mononucleosis, although primarily localized to the lymphatic system, affects the entire body through the inflammatory process.

I postulate that mononucleosis is a biopathy affecting the lymphatic system, an extension of the vascular system. It is associated with viruses but since many people with these viruses do not develop the disease, there must be a biopathic component that determines the development of the mononucleosis syndrome. It differs from many other biopathies in its relatively acute and self-limited character. Generally, there are no remissions and exacerbations—one of the “criteria” Dew described as present in most biopathic disorders. In this aspect, IM resembles an infectious disease.

The factors that seem to be pertinent are that it is a generalized inflammatory reaction affecting the organism overall but frequently showing localization in the throat (cervical segment) and organs of the diaphragmatic segment (liver and spleen). These two segments were areas of particularly prominent armor in this patient. It would be valuable to assess if these segments are particularly armored in those who develop mononucleosis.¹⁹

A Functional Postulate about Autoimmune Disease: Hashimoto's Thyroiditis

The second disease the patient developed, Hashimoto's thyroiditis, is understood classically as a localized, smoldering, autoimmune inflammation of the thyroid. Over the past three decades there has been an explosion of information about the specific mechanisms of immunity and their relationship to autoimmune disorders. Antibodies to “self” have clearly been shown and are the basis for laboratory tests

¹⁹Another patient who developed mononucleosis during the second year of a very stressful graduate school experience also had chronic armoring especially prominent in the cervical and diaphragmatic segments.

used in the diagnosis of these disorders. The mechanism by which the individual develops these is not clearly proven in traditional medicine.

Various hypothesis have been proposed. One is that the individual is exposed to a foreign antigen such as a virus which has elements similar enough to self antigens causing “confusion” in the immune system which produces antibodies that attack self. There is little doubt that such situations occur. As described in *Harrison's*:

One of the best examples of autoreactivity and autoimmune disease resulting from molecular mimicry is rheumatic fever, in which antibodies to the M protein of streptococci cross-react with myosin, laminin, and other matrix proteins. Deposition of these autoantibodies in the heart initiates an inflammatory response. (18:1840)

This process, however, begs the question why only some people exposed to the antigen develop autoantibodies while others do not.

In all of these hypotheses, a functional understanding of the underlying process has largely been lacking. Some years back it was hypothesized that in order for antibodies to form to intracellular components those components must become “extracellular” (19). In other words, the cells must break down and release intracellular contents into the vascular system where lymphocytes can interact with them. Recent research shows an association of cell injury and death with stimulation of the immune system. The discovery of innate factors that may be significant in the autoimmune disorders supports an organomic hypothesis of cell destruction in autoimmunity (20). The hypothesized “chemical messenger” mechanism whereby this occurs, however, leaves unanswered the question of why one person with tissue injury develops autoimmune disease while another develops cancer and still another no disease at all.

Reich's discovery of bionous disintegration of cells provides a basis for understanding how tissue damage may occur spontaneously, beyond direct damage from a mechanical source. If cells are energy-starved due to chronic armoring, the resulting hyporgonia will reduce the energy charge to a level insufficient to maintain the cell's natural

cohesiveness and structure. Objective evidence for this functional hypothesis in autoimmune disorders has been lacking until the laboratory evaluations and RBTs of this patient.

The RBT performed on 11/14/98 showed a poor B-reaction and a strong T-reaction with T spikes. The literature has described this pattern of the RBT as that associated with cancer (8,21). The rest of the clinical picture of this patient, however, did not suggest cancer. A repeat RBT was done on 11/28/98 to evaluate the possibility of laboratory error on 11/14. This repeat test showed a decreased B-reaction, with only fair clot cohesion of the autoclaved specimen and turbidity of the supernatant fluid. While not as severe, it nonetheless indicated an overall reaction in the T direction. At the time, it was not clear what to conclude from these tests. In retrospect, however, all of the laboratory findings were consistent with tissue-specific bionous disintegration localized to the thyroid: First, there was evidence of variable thyroid function, decreased on 10/19/98 (elevated TSH) and normal on 11/2/98; second, RBTs around this time demonstrated significant but variable T-reactions; and third, on 1/30/01, a significant level of thyroid peroxidase antibodies was discovered, showing evidence of antibodies to intracellular thyroid elements after there was evidence of bionous disintegration in the blood. The further decrease in thyroid function on 2/26/01, as demonstrated by a significant increase in TSH, suggests further destruction of thyroid tissue and loss of glandular function.

Taken as a whole, these test results suggest that the tendency toward bionous disintegration or lumination is segment, organ, or tissue specific.²⁰ They suggest that the susceptibility of specific tissues to hyporgonia determines the tendency for bionous breakdown, while the capacity for lumination determines whether the breakdown leads toward an autoimmune disorder or to cancer. These are valuable observations that need follow-up to see if the RBT results can be

²⁰This is a point of differentiation from cancer which is a disorder of generalized shrinking and resignation, with tumor formation as the localized phenomenon.

understood, not just in terms of overall organismic organotic charge, but with greater specificity as suggested in this case.

There is tremendous variability among autoimmune phenomena. Many individuals develop antibodies to components of “self,” especially with advancing age but these do not become involved in actual immunological “attack” on the tissues. Traditional medicine makes the distinction between “autoimmunity,” i.e., the presence of antibodies to self, and “autoimmune disease” in which there is evidence that these antibodies are involved in the active immunological disease. The level of somatic contact as mentioned above is undoubtedly a crucial factor in whether the autoantibodies become involved in a pathological response, or not.

Atherosclerosis and Coronary Artery Disease are an Inflammatory Condition

We know from previous investigation that the cardiac biopathy is associated with a contraction against a primary parasympathetic expansion (22). Traditional medical thinking now recognizes atherosclerosis and acute myocardial infarction as involving much more than structural anatomic pathology. They are now widely recognized as manifestations of both a chronic and an acute inflammatory disorder (23).

The role of cholesterol is an interesting example of the difference between mechanistic and functional thinking. Elevated cholesterol has been implicated as a risk factor of coronary artery disease. There is controversy within the medical community about the significance of this association. The more traditional view of the past several decades has been that elevated cholesterol is a central issue and a prime risk factor. A less prominent minority hold that the original research is flawed and that the role of cholesterol as a risk factor has been overstated (24). The mechanistic reasoning apparently went along the following lines: arteries are like pipes; atherosclerosis blocks the artery like mineral deposits in a pipe; atherosclerotic plaques are made up largely of cholesterol; therefore an excess of cholesterol in the blood will lead to a buildup of cholesterol in the walls of the arteries, just as

an excess of minerals in water flowing through a pipe will lead to a buildup of mineral deposits therein. It was already well established that there is an increased incidence of atherosclerosis and myocardial infarction among a group of patients with a familial disorder of lipid metabolism in which cholesterol is markedly elevated. Major studies were undertaken to evaluate risk factors for atherosclerosis and heart disease. Blood cholesterol was one among many factors included in the research. Most of the studies failed to show any significant association between blood cholesterol and cardiac risk (25). One major study, however, did show an association of mild to moderate significance. As a result of this study, blood cholesterol became the major focus of cardiology for several decades.

A premature conclusion of causation was made and a moralistic attitude toward cholesterol as “bad” and the “culprit” in heart disease became prominent. The problem became finding the “cause” of the elevated (excess) cholesterol. Many investigations were directed toward external sources of the compound and it was reasoned that the obvious solution is to reduce dietary intake of cholesterol. Subsequent research showed that the majority of the cholesterol in the blood is made within the liver, i.e., it is endogenous. In addition, some researchers whose work did not gain prominence showed that the blood level was influenced by emotional stress (26). Subsequent research further divided the biochemical components associated with cholesterol into High Density Lipoproteins (HDL) and Low Density Lipoproteins (LDL). The moralistic terms prevailed here as well with HDL referred to as “good cholesterol” and LDL as “bad cholesterol.” How can we find our way out of this mechanistic maze?

In a functional evaluation of cholesterol, its natural function in the body must be considered. As a major component of cell membranes, cholesterol is essential to life. Therefore, it cannot be “bad” in and of itself. Blake proposed that the elevation of cholesterol in many disorders including heart disease may be a natural response of the organism to stabilize unstable membranes in the face of inflammation or any other process which tends toward cell membrane

destruction, i.e., bionous disintegration (27,28). It may be significant that this patient did not show an elevated blood cholesterol until after the evidence of tissue breakdown, as discussed above in relation to Hashimoto's thyroiditis. The fact that the majority of the cholesterol circulating in the blood is produced in the liver may be a significant factor in evaluating whether there is an abnormal production or imbalance of cholesterol resulting from a block in the diaphragmatic segment, which includes the liver.²¹

Types of Inflammatory Responses

All of the disorders suffered by this patient have in common an inflammatory component, but they are not of the same type. These and other clinical experiences, as well as various theoretical formulations, suggest three different types of inflammatory response:

- Healthy, natural, inflammatory response secondary to external threat; e.g., in nonbiopathic illnesses such as a normal response to infection.
- Primary biopathic inflammatory reaction resulting from the over-excitation of chronic stasis; e.g., coronary artery disease, probably ulcerative colitis.
- Secondary inflammatory response to bionous disintegration of "self," as in autoimmune disorders; e.g., autoimmune disorders such as Hashimoto's thyroiditis.

Diabetes Update

In his first article Dew used diabetes mellitus as an example to illustrate various aspects of the somatic biopathies and discussed several original hypotheses and thought-provoking functional formulations. He hypothesized that the basic mechanism of "transport" of nutrients into cells results from their movement from

²¹The subjects of atherosclerosis and cholesterol will be discussed more fully when Dew's article on coronary artery disease (CAD) is republished (22).

the blood toward the highly charged peripheral tissues according to the orgonotic potential.²² He postulated that this normal movement is disturbed in diabetes mellitus because of an energetic shrinking of the periphery of the organism with maintenance of relatively high intravascular charge. Lacking the normal “pull” into the peripheral tissues, glucose and other nutrients build up in the blood, leading to and requiring increased production of insulin to move them into the cells.

Unfortunately, Dew never returned to diabetes as the subject of one of his subsequent comprehensive articles and a thorough functional analysis of the disease has never been made. His only further discussion of the disease was a letter to the editor in which he briefly modified some of his formulations, particularly reemphasizing the role of the diaphragmatic block (30). Subsequently, Dew’s basic functional formulations were confirmed in a clinical case reported by Konia (10). In this light, his formulations will be reviewed here with a brief update.

Advancing orgonomic understanding of diabetes continues to be of great value in advancing understanding of other biopathies, but is also timely in the current context of an epidemic of the disease, especially among young people in this country.²³ Although much has been learned in the past thirty-five years about diabetes and insulin, the function of insulin and its role in a number of diseases has recently received increased attention. This is a good time to reevaluate the state of knowledge of the illness.

Not mentioned by Dew, but confirming his view of diabetes as a peripheral shrinking biopathy, is the fact that exercise is well known to reduce the insulin requirement of diabetics. This is consistent with the view that expansion, increased parasympathetic tone and movement of

²²Reich discovered the orgonotic potential in which orgone energy moves from areas of low energy concentration and charge to areas of high concentration and charge in contradistinction to mechanical energy which flows “downhill” from areas of high to areas of low energy (29).

²³This increased incidence is primarily of Type II diabetes. (Clearly, the old designation “adult onset type” no longer applies.)

orgone energy outward into the periphery of the muscles, will provide the “pull” needed to move nutrients into the tissues, even in the absence of insulin.

At the time Dew’s first article was published, diabetes was divided into two types based on the age when the disease is usually diagnosed: juvenile onset and adult onset. Then the two designations “insulin dependent” and “non-insulin dependent” evolved, based on the typical treatment approach (roughly parallel to juvenile and adult onset). This distinction was limited because many so-called non-insulin dependent (adult onset) diabetics could be treated with insulin with beneficial results. The currently accepted terms Type I and Type II evolved to classify two different functional classes related to the physiology of the disorder rather than the time of onset or its method of treatment. Type I diabetics (usually the juvenile onset variety) have an absolute lack of endogenous insulin, while Type II diabetics have endogenous insulin production, often in excess, but are insulin resistant.

Type I diabetes is now understood to be an autoimmune disorder in which the individual develops antibodies to components of the insulin-producing Islet cells of the pancreas. The eventual destruction of the Islet cells leads to a total and permanent eradication of insulin production (18:2112). Diabetes fits the theoretical formulation outlined above that autoimmune disorders result from localized, tissue-specific, bionous disintegration. This view supports the formulation that the Islet cells may be particularly susceptible to hyporgonia and resultant cellular breakdown.

It is generally accepted that Type II diabetes is associated with obesity, an absolute excess of insulin production, but resistance of the tissues to the effects of insulin. To date, there has not been a discussion of Type II diabetes in the organomic literature in light of Dew’s formulations.

What then is the disturbance in Type II diabetes? The current epidemic of Type II diabetes is associated with an epidemic of obesity.

Why is this so? Fat tissue contains a charge but is associated with a reduction of excitability.²⁴ Thus, both biopathies appear to share the same formulation: a shrinking from the periphery with a lively intravascular charge. In obesity, energetic shrinking of the organism manifests in the deadening effect of the fat. That this formulation applies to Type II diabetes, where there is an excess of insulin but resistance to its effect, supports the view that the primary disturbance in diabetes is not simply a lack of insulin.

As noted above, this fact was clear some time ago to major researchers such as Dr. Best. It can be said that insulin treatment has allowed diabetics a longer life span so that underlying disease processes have become more evident. Treated diabetics now live long enough to develop kidney failure from diabetic nephropathy while renal dialysis prolongs many of these lives still further.

This was the status of medical technology at the time Dew's first article was published. Since that time, many of the problems associated with dialysis have been eliminated by kidney transplantation. Now commonplace for diabetics, this technological advance provides the fascinating observation that a healthy kidney transplanted into a diabetic patient develops classic diabetic changes over time. Again, it is clear that the disease is a systemic disorder not just a problem of insulin deficiency.

What is the Functional Basis of the Mechanism of Action of Insulin?

A functional understanding of insulin is important beyond the understanding of diabetes. Dew does not specifically indicate what he believes the bioenergetic action of insulin is but suggests it affects cell membrane charge. "Apparently the successful treatment of the biochemical disturbances in diabetes with exogenous insulin occurs as a result of an overwhelming effect on the tissue cell membrane charge." (1:165) It has been hypothesized that insulin works by an excitatory effect on cells (31).²⁵ This hypothesis is of particular

²⁴There is a charge contained in the fat cells but a diminished capacity for excitation. This is suggestive that the factor in the organotic potential is excitation rather than charge.

²⁵Insulin is known to have a parasympathetic effect (10:183, 18:450).

interest in light of significant research indicating that insulin or hyperinsulinemia is associated with a number of disorders such as atherosclerosis and polycystic ovary syndrome, themselves associated with an increased tendency toward inflammation, i.e., a state of over-excitation (18:483,1385)(32).

The Status of Treatment of the Somatic Biopathies

As described above, modern mechanistic medicine has made major gains in the treatment of nonbiopathic conditions and the anatomical changes resulting from biopathic disorders. Dew's first article focused entirely on a functional, theoretical understanding of the somatic biopathies, with minimal discussion of their treatment. This was also largely true of his subsequent articles on specific biopathies. Where then do things currently stand regarding a functional approach to treatment? The organomic literature, both before Dew's original article and after, reports a number of patients with somatic biopathies successfully treated with medical orgone therapy. Discussion with practicing organomists, however, suggest significant variability in response to treatment.

The history of organomy is marked by continuing efforts to integrate theory and practice.²⁶ At the present time we have a good theoretical functional energetic understanding of the somatic biopathies. The next step must be to develop the ability and expertise to achieve more consistent success in the treatment of individual patients.²⁷

²⁶It has been said, "There is nothing more practical than a good theory." This can only occur when theory and practice are integrated.

²⁷The current state of the organomic treatment of somatic biopathies is analogous in many ways to the treatment of the psychoneuroses in the 1920s. During this period Freud became concerned about the lack of a clear relationship between theory and technique in psychoanalysis. The elegant psychoanalytic theory was of little help to the practicing analyst who lacked sufficient practical knowledge to define a systematic technique. Discussions of technique were little more than isolated anecdotes. At the urging of several of the younger analysts including Reich, Freud instituted a technical seminar. Within this seminar the systematic technique of characteranalysis was developed by Reich.

Trends in the Functional Understanding of Somatic Biopathies

Orgonometry has great potential as a tool to define the determinants of somatic biopathic differentiation much more specifically and precisely than has been available to date. The investigation of the orgonometric relationship between the various functions related to orgonotic contact has already provided important insights (6,12). The very definition of the biopathy as a primary disturbance of pulsation may need to be reevaluated because there are important relevant functions in domains deeper than that of pulsation. In fact, one day, biopathies may come to be understood as those disorders that result from a primary disturbance in any function related to orgonotic contact. For example, there is evidence to suggest that cancer arises from a primary disturbance of the very deepest biological function orgonotic streaming, which in turn affects the more superficial realms of excitation, lumination, attraction, and pulsation. Other biopathies may primarily arise from disturbance of such functions as excitation or lumination. These are preliminary considerations that need further investigation. Aside from understanding the specific functions that are affected in particular diseases, a better understanding of changes in the relationships between particular orgonometric functions may better define specific pathological conditions.

A better understanding of genetic factors in somatic biopathies will come from a functional energetic approach to genetics which is already being investigated (33).

In sum, a clear understanding of energetic functions and their relationship in biopathic disease is a prerequisite to developing a systematic approach to treatment.

References

1. Dew, R. "Biopathic Diathesis (Part I) General Principles," *Journal of Orgonomy* 2(2): 155-170, 1968. (This article is also on the American College of Orgonomy website at www.orgonomy.org.)
2. Seppa, N. "Ulcer Clue? Molecule Could be Key to Stomach Ailment," *Science News* 163(10): 148-149, 2003.
3. Endocrinology Lecture series, University of California Davis School of Medicine, Fall 1974.
4. a. Reich, W. *Character Analysis*. New York: Orgone Institute Press,

- 1949.
- b. Baker, E. *Man in the Trap*. New York: The Macmillan Co., 1967.
 - c. Konia, C. "Functional Diagnostics: Criteria for a Functional Medical Nosology," *Journal of Orgonomy* 25(2): 241-254, 1991.
 5. a. Konia, C. "Orgone Therapy: Part VI: Part and Whole Functions," *Journal of Orgonomy* 22(1): 76-88, 1988.
b. Konia, C. "Orgone Therapy: Part IX: The Application of Functional Thinking in Medical Practice," *Journal of Orgonomy* 23(2): 237-247, 1989.
 6. Konia, C. "Orgonotic Contact," *Journal of Orgonomy* 32(1): 61-81, 1998.
 7. Konia, C. "Somatic Biopathies (Part I)," *Journal of Orgonomy* 23(2): 224-236, 1989.
 8. Reich, W. *The Discovery of the Orgone, Volume Two: The Cancer Biopathy*. New York: Orgone Institute Press, 1948.
 9. LeShan, L. *You Can Fight for Your Life*. New York: M. Evans and Company, Inc., 1980.
 10. Konia, C. "Somatic Biopathies (Part II)," *Journal of Orgonomy* 24(2): 181-195, 1990.
 11. Harman, R. Orgonometry Course, American College of Orgonomy, July, 2003.
 12. a. Konia, C. "Orgonotic Contact, Part II," *Journal of Orgonomy* 34(2): 50-59, 2001.
b. Konia, C. "Orgonotic Contact, Part III," *Journal of Orgonomy* 36(1): 50-54, 2002.
c. Harman, R. Orgonometry Course, American College of Orgonomy, 2002-2003.
 13. a. Konia, C. "The Plasmatic System (Part I): The Immune Function," *Journal of Orgonomy* 27(1): 23-47, 1993.
b. Konia, C. "The Plasmatic System (Part II): The Endocrine System," *Journal of Orgonomy* 28(1): 4-22, 1994.
 14. Crist, P. Didactic Course (determinants of character), American College of Orgonomy Medical Orgone Therapy Training Program, 1987.
 15. Ibid. (somatic biopathies).
 16. Livingstone, I. Personal communication interpreting references re: the natural history of multiple sclerosis.
a. Matthews, W. "Clinical Aspects" in McAlpine's, *Multiple Sclerosis. Part 2*. New York: Churchill Livingstone, 1985: 49-278.

- b. Weinschenker, B. et al. "The Natural History of Multiple Sclerosis: a Geographically Based Study. I. Clinical course and disability," *Brain* 112(Pt.1): 133-146, 1989.
17. Reich, W. *The Function of the Orgasm*. New York: Orgone Institute Press, 1942.
18. Editors, Braunwald, E. et al. *Harrison's Principles of Internal Medicine*. New York: McGraw Hill, 2001.
19. Crist, P. MDB/ORAC Research Sub-Committee Meeting, American College of Orgonomy, May 31, 2001.
20. Shi, Y. et al. "Molecular Identification of a Danger Signal that Alerts the Immune System to Dying Cells," *Nature* 425: 516-521, October 2, 2003.
21. a. Raphael, C., McDonald, H. "The Orgonomic Diagnosis of Cancer," *Orgone Energy Bulletin* 4(2): 65-128, 1952.
b. Baker, C, Dew, R, Ganz, M., Lance, L. "The Reich Blood Test," *Journal of Orgonomy* 15(2): 184-218, 1981.
22. Dew, R. "Biopathic Diathesis (Part IV: Arteriosclerosis and Coronary Artery Disease)," *Journal of Orgonomy* 4(2): 192-206, 1970.
23. Winslow, R. "Coronary Culprit: Heart Disease Sleuths Identify Prime Suspect: Inflammation of Artery," *Wall Street Journal*, p.1, October 7, 1999.
24. Ravnskov, U. The Cholesterol Myths, www.ravnskov.nu/cholesterol.htm.
25. Ravnskov, U., www.ravnskov.nu/myth2.htm.
26. Friedman, M. *Type A Behavior and Your Heart*. New York: Knopf, 1974.
27. Blake, J. Personal communication.
28. Ravnskov, U. "High Cholesterol May Protect Against Infections and Atherosclerosis," *Quarterly Journal of Medicine* 96: 927-934, 2003.
29. Reich, W. "Ether, God and Devil," *Annals of the Orgone Institute, Number 2*. New York: Orgone Institute Press, 1949. (Republished and retranslated, *Ether, God and Devil/Cosmic Superimposition*. New York: Farrar, Straus and Giroux, 1973).
30. Dew, R. Letter: "Further Comments on the Biopathic Diathesis," *Journal of Orgonomy* 6(1): 131-133, 1972.
31. Crist, P. Research Presentation, Advanced Laboratory Course in Orgone Biophysics, American College of Orgonomy, June 24, 1981.
32. Sears, B. *The Zone*. New York: Regan Books, 1995.
33. Konia, C. "The Transgenerational Induction of Armor," *Journal of Orgonomy* 36(1): 43-49, 2002.