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The Biopathic Diathesis (Part II)

By ROBERT A. DEW, M.D.*

Introduction

With the present paper, I will begin the discussion of specific biopathies in detail; I have chosen to start with the autoimmune *inflammatory biopathies.* This choice is not entirely arbitrary. In this area, the triumph and failure of mechanistic biology is vividly illustrated.

The idea of immune mechanisms in disease is currently enjoying a tremendous vogue. The literature is vast, complex, and imposing. Those acquainted with classical methodology, when thrust into the milieu of functional thinking, are understandably uneasy with the burden of putting such intricate, detailed information into proper perspective. Conversely, functional thought is so simple and straightforward, by contrast, that one may even experience a certain self-consciousness in attempting to relate such a maze of facts to the clear observations of the living with which we are familiar. Consider, for example, the following excerpt from *Diagnosis News* (1) on the possible pathogenesis of rheumatoid arthritis:

> Infection or some other antigenic substance reacts with IgG (immunoglobulin G), a product of lymphocytic synthesis, to form an antigen antibody complex. Such complexes usually contain altered or antigenic IgG.
>
> The altered IgG stimulates the plasmocytes in the synovial lymphoid collections and lymph nodes to form the rheumatoid factor, the antibody, which complexes with the altered IgG. The complex fixes complement, which renders it chemotactic; it is phagocytized by polymorphonuclear leukocytes, forming the rheumatoid arthritis cells.
>
> In this process, lysosomal enzymes, which are damaging to tissue, are released into the synovial fluid and by irritation probably stimulate further

*Medical Orgonomist and Internist.

> lymphocyte activity. The proteolytic enzyme, cathepsin, from these ruptured lysosomes also may partially denature or split IgG molecules, rendering them antigenic and thus continuing the vicious cycle.

Whether or not we accept this hypothesis at face value, it behooves us to relate it conceptually to the biopathic mechanism.

From the immunologic point of view, the mechanism postulated above may ultimately prove to represent a successful application of cytochemical and other techniques in the elucidation of a pathologic process. However, it is clear that the events described account for what may appear in "susceptible" individuals after the fact of the functional disturbance. Thus, the hypothesis is *treatment*-oriented and not *prophylaxis*-oriented. To us, the word "susceptible" implies a good deal that we can touch in examining the patient on the couch. The great failing of classical science in this case is that it can only vaguely characterize such patients. For example, the finding of autoantibodies in normal or "control" individuals without autoimmune disease creates considerable confusion theoretically and practically. It is presumed, therefore, that the answer to the severe disturbances in this group of illnesses involves "other factors." It would seem that, to some extent, the difference between the mere presence of autoantibodies and the actual manifestation of autoimmune disease is a matter of degree. As is so often the case, a normal or physiologic or protective phenomenon has become destructive and pathophysiologic in those affected. It is our purpose to clarify the reasons for this transformation and, if possible, suggest methods for its prevention. As will be seen, the invocation of "infection or some other antigenic substance" will not suffice as an explanation.

Classical Mechanisms of Immune Disease

The literature reveals a wide variety of immune mechanisms in disease, some examples of which are presented below to convey the line of thought currently holding sway in classical immunopathology:

I. Direct Sensitivity to Foreign Antigens

   A. Antigen (Ag) gains entry parenterally, orally, via the respiratory tract, etc.

   B. Antibody (Ab) is elaborated by the lymphatic system in response to the foreign antigen.

C. On repeat exposure to Ag, an Ag-Ab reaction occurs.

Examples:

1. Serum sickness
2. Allergic rhinitis, poison ivy
3. Hemolytic anemia, thrombocytopenia, leukopenia

II. Indirect Sensitivity to Foreign Antigens

A. A bacterial or other Ag stimulates an Ab response in the host.

B. The Ag, if it bears some structural similarity to a normal component of the host's tissues, will thus elicit production of an antibody with the potential of reacting with body-own tissues.

Example:

1. Streptococcal antigen streptolysin "O" elicits production of antistreptolysin O gamma globulin[1] in the patient. However, streptolysin O shares structural similarities with components of human connective tissue; when represented to the immune apparatus, it induces rapid production of an Ab which not only complexes with streptolysin O but with the connective tissue itself. A destructive inflammatory reaction (*e.g.* carditis) results. Thus the body "mistakes" its own connective tissue for a "foreign" material.
   This mechanism is thought to account for the pathogenesis of acute rheumatic fever and glomerulonephritis (post-streptococcal) and is the basis of the hemolytic disease of the newborn.

III. Sensitivity to Autologous (Body-own) Antigens (*i.e.*, Tissues)

A. Tissue breakdown (*e.g.*, secondary to inflammation, trauma, or necrosis) causes release of tissue components or cell products into the general circulation.

B. Ab develops which attaches itself to and reacts with the corresponding normal tissue or cell *in situ.*

Recent work indicates that normal proteins (*e.g.*, gamma globulins) readily become antigenic, with but slight alterations in structure. As we have seen, the mechanism advanced above for rheumatoid arthritis involves the idea that the cycle of events is initiated by an alteration of IgG, presumably through the action of some infectious agent. Other

[1] ASLO.

diseases in which similar mechanisms are involved are:

1. Hashimoto's Thyroiditis: Ab against thyroid cells + thyroglobulin
2. Hemolytic Anemia of Viral Pneumonia: RBC agglutinins
3. Cold Agglutinin Syndrome: anemia, jaundice + hemoglobinuria
4. Idiopathic Thrombocytopenic Purpura (ITP): platelet agglutinins
5. Lupus Erythematosis: antinuclear antibodies (*e.g.,* anti-DNA Ab)
6. Bacterial Endocarditis: rheumatoid factor
7. Sjogrens Syndrome: autoimmune reaction in salivary glands
8. Pernicious Anemia: Ab to intrinsic factor, and gastric parietal cells
9. Myasthenia Gravis: Ab against muscle constituents
10. Ulcerative Colitis: Ab against colon
11. Pancreatitis: Ab against pancreas
12. Addison's Disease: anticortical Ab

In addition to the direct effect of Ag-Ab interaction, serious secondary effects have been attributed to the presence of high levels of abnormal gamma globulins in the plasma. The peripheral vascular disease in systemic lupus erythematosis has been related to the presence of cryoglobulins (*i.e.,* gamma globulin insoluble at lower temperature). Likewise, the malignant arteritides (*e.g.,* periarteritis nodosa) may have similar mechanisms. The presence of such proteins has been correlated with the occurrence of the subcutaneous and pulmonary nodules seen in rheumatoid arthritis.

In regard to autoantibodies, it is arguable that the Ab is the result and not the cause of the disease. There is in fact evidence for both situations. In rheumatoid arthritis, for instance, these proteins usually appear *after* the emergence of frank clinical disease, whereas, in lupus erythematosis, the reverse is often the case. However, if we accept the idea that *some* functional disturbance always antedates either the abnormal Ab or the clinical disease, the exact order of events with regard to the latter two takes on a different significance. Orgonomically speaking, it is the functional disturbance and the characteristic mode of reaction against contraction which interests us.

The Significance of Autoimmune Mechanisms in Orgonomy

The considerations of immunopathology outlined above raise some important questions:

1. Are the divers mechanisms functionally significant, or are they simply variations on a single theme? In other words, need we concern ourselves with the mechanical immunologic distinctions between,

say, allergic rhinitis and lupus erythematosis?
2. Can orgone therapeutic technique be utilized to recognize "susceptible" individuals?
3. Can treatment prevent the emergence of these diseases or ameliorate or cure them once they become apparent?

Firstly, I think orgonomy, for the present, can take little specific action in the matter of which particular type of immunologic pattern underlies any given disease. In a sense, we are in the same position as the internist who, when faced with either intractable "allergic" asthma or exacerbating lupus erythematosis, must resort to corticosteroid therapy. That is to say, in our present state of knowledge, the specificity of mechanisms is essentially an academic question. Our obligation is to pursue, patiently and cautiously, the same goal in every patient; namely, to restore natural pulsation whenever possible. What *is* of extreme importance is that we learn to characterize and identify a patient's most likely mode of reaction long before abnormal globulins can be found in his blood plasma. This, of course, leads us to the second question.

As I emphasized elsewhere (2), the basic energy level of the patient, his reaction to contraction, and his focal armoring are, at least theoretically, the primary determinants in biopathic differentiation. The accuracy and precision with which we can predict his medical future is obviously no better than our capacity to evaluate him in each of these areas. At present, this capacity is quite limited. While a thorough medical history may give us the flavor of a patient's "reactivity," it is hardly precise prognostically. The biophysical examination is more helpful in that it may characterize the focal armoring, give us a general (but, again, imprecise) idea of energy level, and, hopefully, disclose the presence or absence of biophysical resignation. The Reich Blood Test is a step in the direction of more objective analysis, but can hardly be relied upon to make the truly fine distinctions we would demand of it. Variations and refinements may ultimately make it a more discriminating test. It would be of immense value if the Reich Blood Test or some modification of it could be used to distinguish the potential bearers of inflammatory biopathies from, say, those likely to develop the cardiovascular biopathy. At any rate, the laboratory potential of orgonomy in diagnosis and prognosis has been largely unexplored for years; it is evident, from the accomplishments of conventional research alone, that functional thinking in a diagnostic laboratory could yield inestimable benefits, as well as provide vital correlations for clinical

work. Today, however, the exact quantification and qualification of parameters in biopathic differentiation are beyond our means. Such pioneering work as Reich's in measuring skin potentials in various emotional states (3) and Burr's measurement of field potentials around living objects (4) offer perhaps the most accessible footholds in this territory. Consider, for instance, the possible value of characterizing the behavior of the human orgone energy field in health and disease. Does the focal armoring alter the configuration of the field? Could such studies head off a biophysical disaster by *anticipating* the behavior of the armoring before it precipitates diabetes, strokes, ulcer attacks, coronaries, etc.?

As to the third and last question, we should keep in mind the frequent and often misguided tendency to attribute miraculous efficacy to orgonomy. Nevertheless, one must inevitably feel that therapy should thwart the development of biopathies in many. This, of course, requires that the patient's structure be accessible to treatment and his organism not too severely damaged. The experiences of Reich (5), Baker (6), Hoppe (7), Sobey (8), Herskowitz (9), and others are encouraging. On the whole, I should expect the autoimmune biopathies to respond favorably. However, as Baker (6) points out, there are sometimes explosive eruptions of biopathies in the end stages of therapy. While these may be predictable and do not necessarily preclude the successful completion of treatment, they should make us cautious in our optimism. Just as certain structures may not be able to tolerate therapy, it is likewise probable that some of the biopathies may have "hook-like" characteristics. In other words, for some patients, dissolution of armoring could precipitate an intolerable or prohibitive medical condition. This tendency may, of course, vary in different diseases and in different patients with the same disease. It is obvious that the clinical and research tasks before us are staggering in their immensity.

Specific Disease Entities

In the following consideration of individual immune inflammatory diseases, certain ideas will be seen to recur repeatedly. This is not by design. In going from one disorder to another, I was invariably confronted by the same basic theoretical concepts. I regard this as an affirmation of the validity of my classification of these biopathies under the same functional heading. For example:

1. Inflammation with antibody production (as part of lumination) seems to be a primary pathogenic mechanism in this group.

2. These illnesses, by and large, have their peak incidence in the young.

3. The skin seems to be frequently and directly involved, particularly in the acute stages of these illnesses. Likewise, the GI tract mucosa and submucosa are similarly afflicted, *e.g.,* ulcerative colitis and regional enteritis. This makes sense functionally, since the lining of the GI tract develops embryonically from the invagination of the outer cell layer of the blastocele, producing the primitive archenteron or gut. Therefore, simultaneous pathologic breakthroughs in both these areas in ulcerative colitis, periarteritis, etc., may not be so bizarre when seen in this light. Furthermore, since frustration of core expansion by armoring leads to rage, which, in turn, is forbidden,[2] should we not expect pathological eruption in the skin? The skin, after all, represents the outer limits of that which can be blocked. Thus, to my mind, this aspect of the natural history further substantiates the idea that a high energy system is involved in this group of disorders. The core expands, *come what may.* What happens in the inflammatory biopathies in general corresponds to the ignition of gunpowder (core expansion) inside a closed rigid container (the armored biosystem). The result is an *explosion,* which understandably *destroys the container.* Obviously, the force of the explosion is related to the amount of gunpowder and the rigidity of the container. As a corollary, the disease seems to discharge energy in a way which is acceptable to the patient's structure. It punishes guilt and guarantees dependency gratification in the form of medical care and/or parental concern (*i.e.,* the old idea of "secondary gain").

4. These biopathies often appear to represent high-energy analogues to other biopathies which occur later in life or in lower-energy organisms. We contrast, for example:
   Rheumatic carditis versus arteriosclerotic heart disease
   Rheumatoid arthritis versus osteoarthritis
   Glomerulonephritis versus arteriolonephrosclerosis

5. My theoretical considerations of treatment generally led to the conclusions that:

   A. *Avoidance* of active orgone therapy is warranted during the

[2]Various authors agree that severe repression of rage is a prime feature of these diseases. See *The Theory and Practice of Psychiatry* by F. C. Redlich and Daniel X. Freedman (New York: Basic Books, 1966) pp. 412, 422, 425-7, 442.

acute stages of diseases with *severe, systemic* manifestations.

B. Orgone therapy in patients with a history of the more severe forms of autoimmune inflammatory biopathies must be carried out with extreme caution even when they appear fully recovered.

Hay Fever (Allergic Rhinitis)

Baker refers to the biopathic mechanism of this disorder in *Man in the Trap* (6). It would appear that the symptoms seen in "head colds" are almost identical to those in hay fever and allergic rhinitis. A patient without knowledge of his allergy will complain of a "head cold" lasting for weeks. The ocular block is important here; mobilization of the periocular, frontalis and upper facial musculature should prove beneficial. It occurs to me that treatment of the suboccipital musculature might also help. While the functional relationship of this musculature to the vascular events in the nasal mucosa may seem remote, I am reminded that for years nose bleeds have been treated by allowing the head to tilt back (thus partially relaxing tension on these muscles) and applying ice to the back of the neck. I have observed that the simple expedient of grasping the nose between thumb and forefinger at the roots of the nostrils, exerting a gentle rotary motion in the sagittal plane, has provided partial or temporary relief from nasal congestions in some patients.

Just why the emotional state should influence the result of Ag-Ab interaction is unexplained in classical medicine. In view of the existence of nonseasonal, nonallergic, vasomotor rhinitis whose symptoms are identical to those of the allergic form, I am inclined to believe that the ups and downs of allergic rhinitis in part reflect the state of contraction in the eye segment armoring.

Clinical experience suggests to me that symptoms of allergic rhinitis are a parasympathetic overreaction to the armoring, consisting of vasodilation, hyperemia, and hypersecretion by the nasal mucosa and lacrymal glands. The subsequent development of "allergic" asthma in some thirty percent of untreated hay fever patients is of extreme interest. Classical medicine regards this as a further manifestation of "susceptibility," as it does the frequent concurrence of skin atopy. From the orgonomic point of view, hay fever, asthma, urticaria, and skin atopies are segmental manifestations of the same biopathy, namely, *lumination against contraction by specific armorings*. The various allergens which are implicated simply trigger the process in the segments rendered "susceptible" by the blocks. This would explain

most of the epidemiologic features of the disease:

1. Removal of allergens, or from an allergenic environment, is beneficial in many cases but *not all.*
2. The patients will often "outgrow" these allergies. I have seen many patients whose childhood or adolescent allergies disappeared in time *without* alterations in environment.
3. The number of allergens to which the patient is "sensitive" on skin-testing are often staggering. They are sometimes so numerous as to preclude escaping them by change of domicile or habits; yet the symptoms come and go or abate with or without medication. On skin-testing, the patient, when told he is allergic to this or that material, will often tell you he has used it for years without developing symptoms. Skin-testing, in effect, merely reflects the reactivity of the patient, *i.e.,* it simply indicates that he luminates with the slightest provocation. This is borne out by the finding that many of these patients will develop urticaria on exposure to extremes of temperature, *e.g.,* hot and cold water, or pressure, *e.g.,* dermatographia.
4. The value of desensitization injections (graded exposure to the allergenic substance) is highly controversial. Many physicians regard them as worthless. There is no question in my mind that they *do* work in certain cases. However, the fact of their success in no way compromises our viewpoint. No one questions that allergens *are* involved.

Wegener's Granulomatosis

I include this rare but lethal variant of polyarteritis mainly for the sake of completeness. Almost invariably, these patients will first come to the attention of specialists in ear, nose, and throat disease. The disorder is characterized by necrotizing giant cell granulomatosis of the upper respiratory tract and lungs, widespread necrotizing vasculitides of small arteries and veins, and focal glomerulitis. The majority of cases present with intractable purulent rhinorrhea, nasal antral pain, and epistaxis; and eventually the disease causes relentless and cosmetically horrendous destruction of bone and cartilage of the nose, palate, and orbit. It would be fascinating to discover what facial expressions are being held back in these patients.

Temporal Arteritis (Cranial Arteritis)

This is another very uncommon disorder characterized by granulomatous inflammation and panarteritis. Its main distinction is its rela-

tive confinement to the arteries of the carotid system. This disease would appear to be a *segmental* version of polyarteritis. Its possible relationship to the latter will be discussed in a later paper, under polyarteritis.

Rheumatic Fever

One cannot help but suspect an underlying biopathic mechanism in this illness. If the immune mechanism suggested by classical medicine is corrrect (and we have little reason to think otherwise), rheumatic fever would appear to be an exception to the other biopathies, whose pathogeneses are relatively obscure. Nevertheless, its occurrence raises some difficulties which the mechanics of autoimmunity does not clarify.

The disease has a predilection for children from five to fifteen years of age. It is rare in infants and grows increasingly uncommon with advancing age. This predilection has been attributed to the increased incidence of group A beta-hemolytic streptococcal infections in childhood. It seems odd that this fact in itself satisfies the medical epidemiologist. If the streptococcus can gain access to the respiratory tract of a ten-year-old, one should except it to lodge with equal ease in those of older age.

Furthermore, since numerous serologic group A subtypes have been implicated in rheumatic fever, the odds *against* their contaminating a sixty-year-old would appear slim. Of course, it may be argued that with advancing age one develops a more complete spectrum of immunity against the multifarious strains of streptococci, but this would imply that a previous infection with these bacteria had occurred. We might well ask why no rheumatic fever resulted *then.* Obviously, a great number of people of *all* ages have been exposed repeatedly to one or more serologic strains of group A streptococci. Yet the childhood predilection for rheumatic fever remains an unassailabe and unexplained fact. Classical medicine, faced with this dilemma, acknowledges the probable existence of "host factors"—a view which we heartily endorse.

Rheumatic fever occurs more commonly in lower socio-economic groups. While an increased likelihood of strep infection in such a group might lie in poor sanitation, it should be recalled that the emotonal misery engendered by poverty may be a factor here. The disease also has familial tendencies. This has been commented on previously. However, I would agree that this aspect of its epidemiology makes sense on bacteriologic grounds. A hereditary predisposition to streptococcal infections, however, has never been demonstrated (10).

A second difficulty not entirely clarified by immunopathology is the focal nature of the lesions of rheumatic fever. There is a special predilection for mesenchymal tissue (derived from the middle or mesodermal layer of the embryo) with extensive involvement of small blood vessels, which accounts for the heterogeneous signs and symptoms. However, just why the disease "licks the joints and bites the heart" has never been understood. If we accept certain theoretical premises advanced earlier, some of these facts fall into place. Firstly, the age of peak incidence is at a time when the human organism has a particularly high energy level, a time when the stage of phallic narcissism is fully developed and genitality is struggling to emerge. In our culture, this is necessarily a time of great frustration and misery, when sexual abstinence and inhibition are not only institutionally supported but reinforced by even the most liberal elements in society. I believe that rheumatic fever, and carditis in particular, represents the childhood analogue of the cardiovascular-hypertensive biopathy seen in adults. Bioenergetically speaking, it may be the result of truly *heartfelt* longing in the presence of a pelvic block, with a resultant overloading in the thoracic segment. As in all the biopathies, core expansion in the form of sexual excitation and longing, when thwarted by the armoring, becomes rage. It is the manner of handling this rage which not only leads to the particular picture of rheumatic fever but, to my view, also serves as the basic mechanism for most of the diseases to be considered under the presently discussed group of biopathies.

The patient's anger is just as intolerable to him as the original core impulse. Consequently, energy must be bound to preserve him from overwhelming anxiety. It is my contention that, in the autoimmune inflammatory biopathies in general, the patient's armoring cannot bind sufficient energy in itself to prevent disaster. However, it does block the explosive drive to the periphery. This stasis produces lumination, *i.e.,* the plasmatic system strives to overcome the stasis. The lumination corresponds to the disease state in its acute phase. The frequent appearance of skin lesions with these illnesses, to my mind, signifies a chaotic breakthrough at the periphery. To shift to the psychoanalytic realm, I would call it a somatic representation of unconscious guilt—the patient's "scarlet letter." Orgonomic analysis, then, should reveal under the illness first guilt, then rage, and, finally, longing.

The inflammatory biopathies, therefore, may be viewed as a mechanism for establishing a new energy equilibrium, *i.e.,* orgasm anxiety is thwarted, and the energetic drive is dissipated in the disease syndrome. In psychiatric terms, we might say that the illness assuages

guilt feelings by "punishing" secondary layer impulses. In the case of rheumatic fever, carditis is more "acceptable" to the patient than his underlying rage. In the autoimmune inflammatory biopathies, destruction of tissue is often dramatic: the concept of biopathic self-castigation is certainly compatible with the natural history of these disorders. It would be of enormous value if exacerbations could be shown to coincide with flare-ups of deep rage. Also of interest is the fact that the mesenchymal structures, derived from the embryonic mesoderm which gives rise to bone, muscle, and blood vessels—the structural apparatus for the expression of rage—are so regularly involved. As will be seen, this has possible implications phylogenetically in other inflammatory illnesses, *e.g.,* rheumatoid arthritis.

On immunologic grounds, one is at a loss to account for the relapses seen in rheumatic fever (or any of the other autoimmune biopathies, for that matter). It has been suggested that reexposure to streptococcal antigens may play a role. Indeed, prophylactic penicillin seems to reduce the incidence of relapses. The need for such prophylaxis is felt by most authorities to exist throughout childhood and adolescence. However, the insistence on this regimen is abandoned by many after the fourth decade of life. This is based on the observed rarity of relapses in untreated patients beyond this period.

Clinical Manifestations of Special Interest in Rheumatic Fever

Joint Involvement

Classic migratory polyarthritis is much more common in older children and young adults than in younger children. This is perhaps related to the general observation that such patients are more "mature" and "better behaved." A fifteen-year-old is better able to contain his rage than is a five-year-old, *i.e., he is better armored.* Consequently, there is more likely to be a pathologic breakthrough in the organs of aggression.

Cardiac Involvement

This aspect of the disease was felt by Reich to be due to armoring of the thoracic segment. There is certainly fairly direct evidence for the armoring of the heart itself. Consideration of this bears on cardiac malfunction in other diseases, as well. It should be remembered that the heart develops from a pulsatory muscular tube whose axis parallels the long axis of the body. Functionally, the heart in adulthood consists of a tube folded back upon itself. Since it is morphologically similar in many respects to striated (voluntary) muscle, the possibility of armor-

ing in the heart itself does not seem remote. I would postulate, further, that the heart would be armored in the same fashion as the rest of the body, *i.e.,* transverse annular rings. Normally, a wave of excitation traverses the heart in an orderly fashion, as depicted on the ECG. The excitation is characteristically delayed in its propagation at certain points which correspond to its coursing through specific anatomic structures; namely, the A-V node and bundle branch systems. There are also annular structures in the heart—the valve rings. Thus the presence of conduction disturbances (*e.g.,* prolonged P-R and Q-T intervals) and valvulitis makes complete sense. Damming up of energy behind the valve annulus could account for the subsequent development of stenosis after the acute inflammation subsides. The advantage of this hypothesis is that it provides a rational explanation for the *specific effects* of rheumatic fever on the heart. It also suggests a way of understanding functional disturbances of the heart in the absence of morphologic disease, *e.g.,* the ectopic beats, arrhythmia, and tachycardia which are so frequently encountered in anxious patients. The intimate involvement of the heart in the expression of the living organism and its verbal language needs no amplification here.

Cutaneous Lesions; Subcutaneous Nodules

These manifestations of rheumatic fever clearly point to a blocking of expansion at the periphery. Curiously, the nodules are frequently symmetrical in distribution and appear over bony prominences on the dorsum of the body. The reason for this is obscure.

Sydenham's Chorea

Interestingly, chorea most often appears alone rather than in conjunction with other manifestations of acute rheumatic fever. It may be that this is the pathway of the biopathy in patients with strong eye blocks, such as schizophrenics. The schizophrenic would be less likely to have explosive breakthroughs in the heart, skin, and joints by virtue of his weak armoring in these areas. With the decreasing incidence of acute rheumatic fever and relative rarity of chorea, we may never be able to test this hypothesis.

Treatment of Rheumatic Fever

It goes without saying, that current standards of practice regarding aspirin, penicillin, etc., must be observed. Orgone therapy for the present is impractical, and possibly even dangerous, in the patient with acute rheumatic fever. Treatment in the remission stage would require utmost caution, with careful avoidance of "push" which the patient

cannot easily tolerate. It is of interest that steroids and salicylates quiet the overt disease but have little, if any, effect on the underlying process. However, this is not surprising considering the underlying biopathic mechanism.

Subsequent articles will deal with:

Thyroiditis
Glomerulonephritis and the nephrotic syndrome
Ulcerative colitis
Rheumatoid Arthritis
Lupus Erythematosis
Polyarteritis
Dermatomyositis
Scleroderma

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