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The Biopathic Diathesis

(Part VI: Hyperthyroidism)

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Introduction

The hormones of the thyroid gland are essential for normal growth and development and adult functioning in all but the simplest vertebrate life. It is likely that every tissue in the body is influenced by these hormones in some way. While their mechanism of action remains unclear, under ordinary circumstances they appear to enhance protein synthesis, producing as a consequence an increased oxygen consumption in the total organism. Electron microscopy reveals that the hormones induce a swelling of the mitochondria, the intra-cytoplasmic organelles believed to be a major site of chemical energy production in the cell.

The gland itself is superficially located in the anterior neck, straddling the trachea just beneath the larynx. It is a soft, flat, roughly shield-shaped structure consisting of right and left lobes and a central connecting "isthmus." It is not easily palpable in the normal state. Two hormones—triiodothyromine (T_3) and tetraiodothyromine (T_4)—are produced. Of these, the concentration of unbound[1] serum T_4 is most relevant metabolically. Disorders of the thyroid are noteworthy for their capacity to induce vivid and dramatic changes in physical appearance and behavior. Most prominent among these illnesses is hyperthyroidism —signifying a pathologic over-production of thyroid hormone with its attendant clinical picture.

Since there is very little objection from any quarter to the idea that hyperthyroidism is a psychosomatic disease, we shall not argue for its inclusion in the present series of articles. Reich also regarded it as a biopathy (1:p.177). However, the exact relationship of emotional and biophysical factors to its cause is not well understood.

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[1]Most of the circulating hormone is bound to carrier proteins such as albumin and thyroid-binding globulin (TBG).

Clinical Picture—Effects of Excess Thyroid Hormone[2]

1. *Connective Tissue*

Increased ground substance and hyaluronic acid production produces swelling in connective tissue. This is the presumed mechanism of *exophthalmos* and *pretibial edema.*

2. *Cardiovascular Effects*

Thyroid hormone has three main effects on the circulatory system:

a) A direct adrenergic (epinephrine-like) influence on heart muscle.

b) It enhances sensitivity to the effect of the adrenergic neurotransmitters (epinephrine, norepinephrine).

c) Increased blood volume and cutaneous vasodilation, presumably due to the opening up of arteriovenous shunts in the periphery and increased cardiac output.

These three are the mechanisms underlying the *warm, erythematous sweating skin, tachycardia, arrhythmias* and *palpitations, functional heart murmurs, angina pectoris* and *congestive heart failure* not unfrequently associated with thyrotoxicosis.

3. *Skeletal Muscle*

Thyroid hormone has a profound effect on the contractile apparatus of the voluntary muscle fiber. While the speed of contraction is increased, its duration is reduced, the net result being *weakness.* This is the central mechanism behind the *myopathies* of *hyperthyroidism.* Among these are: a) Chronic toxic—associated with generalized *weakness,* b) ophthalmoplegic—*strabismus, double vision,* c) *myasthenia gravis* with diffuse toxic goiter.

4. *Nervous System*

Presumably because of its metabolic and catecholamine effects,[3] thyroid hormone induces *nervousness, irritability,* and *tremor. Severe* thyrotoxicosis may produce an *encephalopathy* characterized by delirium, stupor, coma, and convulsions. The electroencephalogram in hyperthyroidism exhibits an increased alpha rhythm.

5. *Gastrointestinal Tract*

In the hyperthyroid state, there is a decreased volume of saliva, and decreased secretion of hydrochloric acid in the stomach. Some au-

[2]An exhaustive presentation may be found in reference 2, Chapters 32 through 42 and 44.

[3]Increased O_2 utilization, cardiovascular blood flow, and sensitivity to epinephrine.

thorities have linked the latter effect to the finding that, in 30% of hyperthyroid subjects, auto-antibodies against the gastric parietal (acid secreting) cells are produced. The effect on gastric motility is unclear; however, intestinal motility is definitely increased, accounting for the symptom of *diarrhea* in some patients. Jejunal secretions are increased in volume and enzymatic content and mucosal absorption appears to be facilitated. *Abnormal glucose tolerance* in hyperthyroidism has been attributed to the suppression of insulin release by an overexcited sympathetic nervous system. There is a decreased absorption of fat.

6. *General Metabolic Changes*

There is a heightened expenditure of calories with *weight loss* in the face of an *augmented appetite.* Enhanced fat turnover and oxidation —a catecholamine effect—occurs. Since there is no evidence for an increased synthesis of sympathetic neurotransmitters, the presumed mechanism is the enhanced sensitivity to those agents noted above. Gluconeogenesis is fostered at the expense of glycogen from liver and muscle. Protein catabolism may be a consequence of the relatively inadequate carbohydrate intake. Vitamin deficiencies develop because of the increased metabolic demands. Interestingly, administration of certain vitamins such as A, thiamine, B_{12} and C has been shown to mitigate the effects of experimental hyperthyroidism. Thiamine may work by inhibiting the thyroid gland directly.

Hyperthyroidism may be complicated by *hypercalcemia, hypercalciuria, rarefaction of bone,* increased bone turnover, and fecal excretion of calcium and phosphorous. In brief, there is a negative calcium balance at the expense of bone. *Kidney stones* occur with greater than usual frequency, except in Japan, where a peculiar combination of thyrotoxicosis, lowered serum potassium, and periodic paralysis of the muscles is found and abnormalities in potassium are rare.

Extracellular fluid and plasma volumes are expanded. There is increased renal blood flow and loss of solute-free water in the urine.

7. *Emotional and Mentational Disturbances*

The range of symptoms once hyperthyroidism develops varies from *restlessness* to *severe agitation,* but whether this is cause or effect is not always discernible. It may be significant in this connection that epinephrine administration is known to cause thyroidal swelling (despite concurrent vasoconstriction of the gland's blood supply). A *loosening of associational* trends and *fear of impending insanity* have been reported. Psychosis has occurred after excessive ingestion of thyroid hormone, and also with exacerbations of Graves' diseases (diffuse toxic

goiter). Many patients remain emotionally unstable even after becoming euthyroid with medical and/or surgical therapy.

Toxic delirium appears to result from the heightened metabolic rate.

In summary, the symptoms and signs of hyperthyroidism are mainly those of sympathetic overactivity. It seems clear that catecholamines activate thyroid function and that thyroid hormone(s) in turn potentiate the sympathetic effects by sensitizing the organism to epinephrine.

The Pathogenesis of Hyperthyroidism—Classical Investigations

So far, the cause of hyperthyroidism remains obscure. Early in the investigation of this disorder, it was believed the underlying mechanism was simply a hypersecretion of the thyroid-stimulating hormone (TSH) by the pituitary gland. However, studies of patients have consistently shown subnormal levels of TSH. Consequently, opinion shifted to the view that the disease was the result of an intrinsic disorder of the thyroid gland itself due perhaps to a genetically determined predilection. In 1956, an abnormal thyrotropic-like substance was discovered in the serum of patients with Graves' disease. It has been termed *long-acting thyroid stimulator* or LATS. Found in 80% of hyperthyroid subjects—predominantly in those with exophthalmos—it was initially believed to be the cause of the disease. Certain antibody-like properties of LATS further suggested that hyperthyroidism might be an autoimmune disease in which the over-production of hormone, exophthalmos, etc., were the result of an antigen antibody reaction. This theory ran into a number of difficulties, however. LATS is absent in many patients with active disease including some with exophthalmos. Conversely, in the few families investigated, some siblings of hyperthyroid patients have been found to have LATS in the absence of disease.

Autoimmune mechanisms, furthermore, do not explain the cyclic pattern of exacerbations and remissions or the increased incidence in puberty and shortly after the menopause. While LATS as a primary etiologic agent has many firm adherents, current opinion has largely returned to the idea that hyperthyroidism is most likely due to an intrinsic disturbance of the gland. Some go so far as to suggest that there may be two disease populations, *i.e.,* those with an intrinsic pathogenesis and those with an autoimmune etiology.

Of extreme interest is the opinion of some experts that LATS titers are closely linked to the emotions. While this point is debated in the literature, there is little argument that hyperthyroid patients have a remarkably consistent psychologic profile, that exacerbations of their

disease are commonly associated with emotional traumata, and that a history of chronic emotional stress predating the appearance of symptoms is found with striking regularity.

1. *The Psychiatric Background—Hyperthyroidism*

Psychiatric case histories of patients with hyperthyroidism reveal the following characteristics:

a) Loss: separation from or rejection by a figure for whom the patient has strong dependency feelings.
b) Terror of loss or separation and anger at the object of the dependency. Defenses against these feelings are constantly threatened by a breakthrough, so anxiety is considerable. As a consequence, there are secondary defenses against the anxiety.
c) Male patients tend to have a passive-feminine orientation with fear of desertion and isolation by and hatred for the mother.

In brief, these patients have had to contend with fear of isolation and rejection since childhood. Their pattern of existence evolves to avoid not only the isolation but their terror of it. The hyperthyroidism is felt to represent in an as yet obscure way the outcome of these developmental problems compounded by the additional endocrine stresses of puberty, pregnancy, or menopause.

These features are remarkably similar to those found in asthma (3:p. 188), peptic ulcer, ulcerative colitis, and other disorders, raising some question as to their bearing on the specificity of these biopathies. However, a number of important points emerge which are relevant to our discussion:

1. These patients are emotionally and hence energetically *alive.*

2. The repressed anxiety and terror is eventually manifest via an endocrine mechanism in the disease picture itself.

3. The elaborate defense against dependency feelings suggests the presence of a pregenital *oral repressed block* which may be crucial in the specific expression of thyroid disease.

A Functional Theory of the Pathogenesis of Hyperthyroidism

In reviewing some of the classical literature on hyperthyroidism, it is evident that a tremendous amount of data has been collected. I think the classical investigators would agree that the greatest difficulty in this body of information is a lack of cohesiveness. New discoveries seemed

to point the way to an understanding of the disorder; yet, as in the case of LATS, for example, these breakthroughs more often than not have created more problems than they've solved. We cannot pretend in our theoretical survey to rectify this state of affairs, but it is hoped that certain links between the patient and his disease process might be clarified.

It may be helpful to start out by considering the place of the thyroid in plasmatic functioning.

1. *The Role of the Thyroid*

It is apparent that the thyroid hormones potentiate the effects of the sympathetic amines. The catecholamines are the chemical neurotransmitters by which the function of *contraction* is conveyed throughout the mass of the more complex organisms (such as man). Selye proposed the thesis that the adrenergic compounds readied the organism for "fight or flight." Needless to say, these compounds constitute an evolutionary advantage in this respect. They are clearly part of the organism's response to stress. Thus the thyroid would seem to be an adaptation which further enhances the capacity to withstand stress, first by sensitizing or "priming" the organism to the contractile process, and secondarily by enhancing metabolic activities which play a role in mechanically carrying out that process.

It is also evident that in so far as *contraction* and *expansion* are but member functions in the antithetical pair which constitute *pulsation,* each must engender the other. In other words, contraction and expansion are *interdependent.* This point is clearly illustrated in Reich's description of the plasmatic movements of amoebae (1:p.50). Since organismic integrity is a function of pulsation, one can envision that a chronic predominance of contraction *or* expansion would radically alter the behavior of the organism in toto and of its constituent organs. Hyperthyroidism obviously constitutes a massive swing in the direction of contraction or sympatheticotonia. Our task is to understand how a gland which normally plays a role in contraction and yet yields to the subsequent expansion can suddenly become unresponsive to the pulsatile process.

2. *The Mechanism of Thyroid Glandular Hyperfunction*

Our understanding of thyroid hyperfunction rests on two assumptions:

a) that it is somehow a result of specific muscular armorings and

b) that it reflects a generalized sympatheticotonia in the body.

Let us now consider these assumptions in detail.

a. *The Role of Segmental Armor*

The mechanisms by which the armor exerts its effects on organs have been discussed in previous articles in this series. It would be helpful, however, to review certain points with respect to hyperthyroidism.

First, the soft tissues of the body—organs, nerves, vessels, muscle, etc.,—are pulsatile both by virtue of their having a blood supply and due to an inherent quality in their plasmatic substance. Any chronic impingement upon the organs themselves or their blood and nervous supplies will be reflected in a disturbance in pulsation and hence, *function.* The thyroid, by virtue of its anatomic location, is not subject to direct compression by overlying muscle. Its vulnerability lies in its blood supply; consequently, the sympatheticotonia in the cervical segment would be expected to influence thyroidal arteries, veins, and lymphatics. The connection with a *throat block* thus seems direct enough. There is, however, reason to suspect that there is at least one additional *specific* segmental armoring at work here.

We alluded earlier to the psychiatric evidence for an *oral block* in hyperthyroidism. Such an armoring would provide a partial explanation for a certain manic coloration sometimes seen in the irrational picture of the disease, *e.g.,* garrulousness, rapid speech, wittiness. This would appear to be a breaking through of the repression at the oral level. We must keep in mind that this behavior constitutes a substitute contact in that it is not only inappropriate but also *defends* against the anxiety and secondary drives (such as hatred), which lurk beneath the surface. The proposed relevance of such a block, however, poses a problem, namely, how if at all this particular armoring might influence thyroid glandular function. A clue may lie in the embryonic development of the gland. The thyroid begins life as an invagination in the floor of the oropharynx at the base of the tongue; later, it migrates downwards into the neck. Its passage forms the *thyroglossal duct* which normally has no lumen.[4] The point of origin is indicated by a dimple (foramen cecum) in the tongue root. The oropharyngeal region constitutes the terminus of the oral segment and the most rostral portion of the cervical segment. The question arises as to whether or not an armoring in the segment of embryonic origin can have an effect on an organ which comes to lie in a subjacent region and whether such an armoring in the jaw might potentiate the effect of that in the neck.

This question introduces a possible additional mechanism by which

[4]Thyroglossal duct cysts are a common developmental anomaly and consist of a persistent duct lined with pharyngeal epithelium.

the segmental armoring may effect organismic integrity. In *Cosmic Superimposition* (3:p.48); Reich schematized the flow of excitation in the orgonome, indicating the mutual influences of surrounding membrane and the internal streaming. Energy flows up the back of the orgonome from the tail, to curve forward and outward at the head. It is reflected then backward and downward toward the tail, impinging on the anterior aspect of the membrane in its course (see *Figure 1*). This drawing of Reich's corresponds closely to the shape of the human embryo. We draw specific attention to the role of the orgonotic movements in the embryologic development of the thyroid gland (numeral 2). If

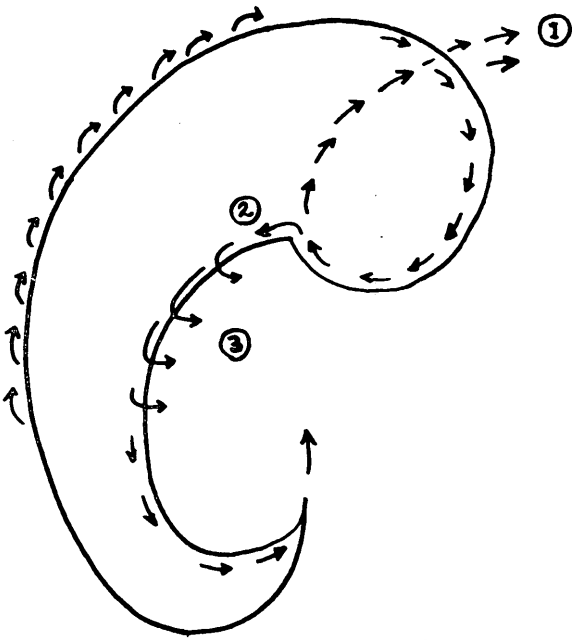

Figure 1.
The Living Orgonome
(As modified from Reich (3))

1) Direction of *forward movement.*
Breaking out of the form of eyes, nose, mouth; origin, and upward migration of pituitary anlage from ceiling of oropharynx.
2) *Downward movement* (in the neck).
Origin and downward movement of thyroid anlage from floor of oropharynx.
3) "Intermediary breakouts," *e.g.,* arms, breasts, legs.

Reich is correct, the origin and migratory course of the thyroid anlage become entirely comprehensible.[5]

It is evident from Reich's discussion of the living orgonome that the orgonotic dynamics function not only in its development and growth but throughout its life, *e.g.* they are the basis for its genitality. It would seem reasonable to expect that the healthy functioning of the organs within should stem from these same energetic movements, *i.e.,* the same currents that give rise to an organ sustain it in adult life.

With this idea in mind, one can see how the oral block could be strategic. There would not only be spasm in the region of the thyroid (cervical block), but little energy coming into the region from above, creating a locus of depressed orgonotic charge. In this way, the terror and anger blocked from expression by these armorings might set the stage for the thyroid disorder.

It would be extremely desirable at this juncture to point to a collection of cases solidly confirming the proposals we have made. Unfortunately, my personal experiences with hyperthyroidism have been principally outside the realm of orgonomy. Precise biophysical data on sufficient numbers of patients are lacking. However, there is some clinical material available which may have an indirect bearing on the present case.

I have had the opportunity to examine in some detail four patients with diverse types of thyroid disease:

a) A 28-year-old female paranoid schizophrenic with a history of hypothyroidism, who became euthyroid in the course of therapy.[6]

b) A 40-year-old female catatonic schizophrenic with a history of hypothyroidism, who requires 2 grains of thyroid extract supplement daily.

c) A 25-year-old female hysteric with a history of papillary adenocarcinoma of the thyroid treated with surgery and radiation therapy, who requires total replacement therapy—75 micrograms of triiodothyromine daily. At no time prior to diagnosis and treatment for the neoplasm did she show any evidence of hypothyroidism. She'd had a thyroid nodule detected at age 15 and was operated on when she was 23. Her mother has a history of goiter with hypothyroidism.

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[5]As does the pituitary anlage.

[6]No claim for her cure under the influence of orgone therapy is made here. A significant number of patients with hypothyroidism often related to *thyroiditis* will spontaneously revert to normal.

d) A 35-year-old male phallic with a "cold"[7] adenoma of the thyroid excised surgically. He was euthyroid before and after treatment for the tumor.

Aside from having thyroid disease, these four patients have the following features in common:

a) Throat (cervical segment) block
At the start of orgone therapy, all four patients had harsh, nasal, high pitched or otherwise affected voices. Patient *a* is subject to headaches that are regularly brought on when she is agitated and relieved by screaming angrily. Patient *b* has a "cervical radiculitis" that formerly required the continual use of a cervical collar, but it has responded favorably to orgone therapy. Patients *a*, *b*, and *c* choke and gag readily and frighteningly when aroused emotionally. In all four, intense crying impulses are blocked in the throat, but a remarkable deepening in pitch and softening in the quality of the voice becomes evident when the inhibition is overcome. The male patient's (benign adenoma) throat block appears to be the most accessible therapeutically.

b) Oral block
All four have oral repressed blocks characterologically, although patient *a* exhibits some oral unsatisfied features. There is marked masseter, temporalis and digastricus muscle spasm (here, again, the male patient seems the easiest to manage). The jaws are like steel traps exhibiting a jackknife rigidity, holding back intense rage and sadness. Protrusion of the tongue will precipitate gagging.

b. *Miscellaneous Observations*

All four patients are heavily armored; the ocular segment is particularly dead, although the hysteric shows some overt anxiety in the eyes. A most striking feature of these patients is a quality of explosiveness. Even the patient with carcinoma, while usually either docile or stonily resistant, at times—particularly when the jaw and neck are probed—turns literally purple with rage. Despite the heavy armoring, one feels a savage violence just beneath the surface—deep hatred held rigidly in check.

The reader should be cautioned at this point regarding the significance of these cases. The reason for presenting them is to demonstrate a possible connection between certain armorings and thyroid *disease*—

[7]No endocrine activity in the tumor.

any kind of thyroid disease. These cases cannot be construed as prototypical examples in hyperthyroidism.

c. *The Effect of the Generalized Sympatheticotonia*

From our observations of hyperthyroid patients, it is obvious that there is a great deal of feeling in them which cannot be discharged. They shrink from their impulses as one would from any noxious or fearful stimulus. Even the resultant anxiety is buried, leading to frequent denials that anything is "wrong."[8] The patient is thereby subject to continual stress and contraction and has to contain both primary (core) and secondary drives. The sympatheticotonia is thus a response to the threat of an intolerable expansion. The thyroid reacts as it would to any stress, *i.e.,* with swelling and release of thyroid hormone. In the absence of stasis, the plasmatic system would normally reexpand through emotional and/or physical expression of stress, thereby discharging the tension. In hyperthyroidism, the emotion is blocked (stasis) by the armoring, and the only recourse is further contraction and its attendant hormonal release. Add to this the very considerable emotional rigors of puberty or pregnancy. The thyroid is literally "caught in the middle" between focal spasms; unrelieved catecholamine bombardment on the one hand and a high energy level, a high degree of excitability, on the other. The organism tends continually to break the sympatheticotonia; this produces incessant anxiety and a perpetual need to control it. The thyroid is therefore enmeshed in an ongoing defense against chronic terror, in which the excess hormone acts to fortify the contractile process. The disease's specificity is related to the armoring in the neck and possibly the jaw; this distinguishes it from the hypertensive and cardiovascular biopathies in which a similar contraction against expansion prevails, but in which the predominant armoring appears to be in the chest.

3. *The Autonomy of the Thyroid in Hyperthyroidism*

We have touched upon the mechanism by which excess hormonal secretion is perpetuated. There is one last point to be made in connection with the clinical observation that the gland is *not suppressible.* In a number of endocrine disorders, it is apparent that a chronic stimulus induces hypersecretion which may continue even after the stimulus is removed. For example, the *parathyroid* glands which regulate the plasma concentration of calcium, normally respond to a fall in calcium by an increased

[8]Many patients are brought to physicians at the urgings of friends and relatives.

production of parathyroid hormone. In certain intestinal diseases of fat malabsorption, great quantities of calcium are lost in the stool, leading to a state of chronic hypersecretion by the parathyroid. In certain cases, even after the diarrhea has been corrected and the serum calcium restored to normal, this hypersecretion continues. Tests which overload the blood with calcium fail to suppress the gland. The glands themselves may be considerably enlarged or hypertrophied. Sometimes one or more of the four glands will develop functioning (hormone secreting) adenomas. And in still rarer instances, secreting parathyroid malignancies have been found.

Apparently chronic hyperstimulation and hypersecretion have an effect on the gland's responsiveness to regulatory stimuli. Not only is the thyroid unsuppressible in hyperthyroidism, but often it will fail to respond to administered TSH with an expected rise in hormone output. The usual explanation for this is that the gland is already "maximally" stimulated. This seems unlikely as an explanation for every case of hyperthyroidism, particularly the milder ones in which hormone levels may be only modestly elevated or on the rise.

I believe the trouble lies in the gland itself and that it is due to the continually low or falling charge in the glandular tissue. Anyone who has witnessed or felt the terror or panic of an anorgonotic attack might understand the behavior of the follicular cell in these circumstances. The normal regulatory mechanisms would have about the same impact as telling a man about to be guillotined to "relax."

In short, the hyporgonia of the gland countermands the normal regulatory mechanisms of hormonal secretion. Therefore there is no *suppression* of secretion by exogenous administration of hormone (*e.g.*, T_3) or *rise* in secretion after giving synthetic TSH.

4. *The Significance of LATS, Anti-Microsomal and Anti-Thyroglobulin Antibodies*

This particular aspect of hyperthyroidism might best be introduced with a comment by Bondy in his review of some recent work on the role of autoantibodies in this disease:

> The present article shows that LATS is not a particularly specific agent, and the antibodies to M and TG[9] are so nonspecific . . . that no conclusions seem warranted to me about their roles, either. I feel uncomfortably as

[9]M=microsomes of thyroid follicular cells.
TG=thyroglobulin—the form in which thyroid hormone is found within the follicular lumen.

> though we had set off on the wrong path in our search for an explanation of this disease. Ordinary controls are inactive; antibodies are nonspecific. Surely there must be some explanation that does not involve either of these and which would be intelligible to a sensible man (that means me, of course). What we need now is a new look at the problem! (4:p.530).

From our earlier discussion, it is evident that Dr. Bondy is not alone in his discomfort with the autoimmune explanation for hyperthyroidism. It appears to us that LATS, anti-M, and anti-TG are the *results* of the disorder in some cases and not the cause, since the endocrine disturbance seems at times to manifest itself in their absence. Still, these antibodies have a significance which should be pursued.

In previous papers on autoimmune diseases, we have expressed the opinion that the order of events begins with a sympatheticotonia in the tissues due to the armor. We would speculate that this contraction develops rapidly in a fairly vigorous organism (indeed, the rapidity may be a function of the vigor). We have further proposed that bionous changes (perhaps reversible) occur which confer on the affected cells an organism-alien quality.[10] In response, the body, if capable of energetic lumination, produces antibodies against alien proteins. There are certainly well-known instances of this observation: For example, antibodies to cancer tumor cell antigens, hemolyzing antibodies to red cells in lymphatic leukemia, etc. In short, the organism tends to isolate and destroy cells that behave in a wild or disorganized fashion. The presence or titer of antibody not only must depend on the vigor of the organism but also on the amount of abnormal tissue present and probably on how far advanced the abnormality is in the orgonotic sense (*i.e.,* on whether more toward a B- or T-reaction).

We believe that this idea for the pathogenesis of LATS or other antibodies in hyperthyroidism may solve some difficulties. Classical immunologists are in agreement that the stimulus for autoantibody formation may be normal cells rendered antigenic by some injury. The presumption in many instances, *e.g.,* rheumatoid arthritis, is that the injury is infectious in origin and usually viral. In other instances, toxic influences—chemical and physical—have been implicated. It has always been assumed that the force altering the normal tissue has come from *outside* the body. The one mechanism which has been seriously neglected is that

[10]Bionous disintegration is a normal process. At one end of the spectrum is the normal breakdown of cells; at the other, there is the T-reaction of cancer. It may be that the degeneration of thyroidal cells in hyperthyroidism represents an intermediate between these two extremes. All autoimmune reactions may well be found to fall in this intermediate category.

of auto-injury.[11] Let us examine the advantages of this concept.

a) Variations in the incidence of thyrotoxicosis would become comprehensive in emotional terms. For example, the well-known stresses of puberty, pregnancy, and menopause.
b) The rise in LATS titers with emotional upheavals would be explained, as would the persistence of these titers after the total removal of all thyroid tissue.[12]
c) Exacerbations and remissions could occur with or without corresponding fluctuations in antibodies, since these antibodies are the *result* of a deeper underlying process. That is, the disturbed thyroid function is primarily a result of the sympatheticotonia and the antibody results only if the clamping down is of sufficient severity and chronicity to induce a subsequent bionous change as well. This might also explain the absence of LATS in so many cases of active disease.[13]

In summary, the antibodies of thyrotoxicosis in our view are the products and not the cause of the disease and furthermore do not appear to be an inevitable consequence of the disorder. In addition, the presence of LATS, anti-M, anti-TG, etc. may reflect more the state of "alien" change or "auto-injury" to the thyroidal cell rather than its endocrinologic recalcitrance. At the same time both the cell disintegration and hypersecretion stem from a common source (hyporgonia).

5. *The Relationship of Thyroid and Thymic Disorders*

While this relationship between the thyroid and thymus glands is fraught with vagaries, certain common functions are discernible. The thymus is involved in the attainment of immunologic potency in the small lymphocyte. Thymic hyperplasia and hyperthyroidism have been found to occur together, and remission of myasthenia and hyperthyroidism has been described following thymectomy. Myasthenia gravis has been associated with thymic tumors (benign and malignant). Thus, thymus and thyroid diseases may occur together and are linked by the clinical feature of muscular weakness, auto antibody production, and an anatomic proximity to the neck.

[11]It should be mentioned that *genetic factors* have always been considered, the implication being that some external stress brings out a genetic weakness which had been heretofore unexpressed. However, this avoids the psychosomatic question entirely.

[12]Stress causes a rise in immunoglobulin production.

[13]It would not explain so easily the rare presence of LATS in the *absence* of disease, or the presence of antiparietal cell antibodies.

The Orgonomic Treatment of Hyperthyroidism

I. The Role of Conventional Medical and Surgical Therapy

Since the job of breaking through the characterologic and muscular armor is arduous and time consuming and is likely at first to accentuate the patient's symptoms, it may be essential to give top priority to the control of the potentially serious physiological effects of excess circulating thyroid hormone. Keeping in mind the possible benefits of orgone therapy, we would perhaps be inclined toward even greater conservatism than the classical endocrinologist. In conventional therapy, relapse is a greater therapeutic problem than is initial control. Some fifty percent of patients treated with antithyroid drugs (*e.g.,* propylthiouracil) have a recurrence of their disease after withdrawing medication. Although second and even third courses of the drugs are tried in many cases, recourse to destructive forms of treatment such as I^{131} becomes necessary. I would anticipate that orgone therapy will prove itself primarily in the prevention of relapses with conservative drug therapy. At present, however, no such guarantees can be made, lacking, as we do, an adequate "clinical trial." Such drugs as the adrenergic blocking agents and sympathetic antagonists may be of value in allowing us sufficient time to work with the patient's functional problems so that more toxic and destructive therapies may be avoided. The conventional methods for the endocrinologic evaluation of the efficacy of treatment are applicable in both medical and orgone therapy.

II. Orgone Therapy

From our working hypothesis on the biopathogenesis of hyperthyroidism, certain points in therapy would seem to require special emphasis.

1. The patient's anxiety must be brought to the surface, *i.e.,* he must be encouraged to reveal it *within the context of his conflicts.* This is important, since he may already have "free floating" anxiety as an hormonal effect. The eye segment deserves particular attention because of the great likelihood of contactlessness and the possible value in preventing exophthalmos.

2. The oral and cervical blocks are of crucial importance. Nevertheless, extreme caution seems warranted. One doesn't know what effect a sudden flooding of the thyroid might have. It is conceivable that a thyroid "storm" could be precipitated (much in the same way that occurs in surgery for hyperthyroidism), particularly if the gland is enlarged and highly vascular. Therefore, the structures of the anterior neck must be handled gingerly.

3. The appearance of psychotic symptoms must be considered a strong

indication for definitive medical therapy. Psychosis in hyperthyroidism may prove quite resistant to orgone therapy alone.

4. Exacerbations of hyperthyroidism may be expected to occur as therapy begins to reach the hatred in the deeper layers of the patient's structure. The upper three segments must therefore be continually checked for unrevealed anxiety and rearmoring.

Summary

1. A brief description of the metabolic and clinical effects of excess thyroid hormone has been presented with an abbreviated review of the classical pathogenesis of hyperthyroidism.

2. A functional hypothesis regarding the biopathogenesis of hyperthyroidism views the disease as the result of a chronic terror of expansion. The associated sympatheticotonia triggers the swelling and hypersecretion of the thyroid as a response to stress. The excess hormone acts to fortify the process of contraction. The organism reacts with vigorous attempts to break through the sympatheticotonia, which leads to severe anxiety, further contraction, and further hormonal release. The thyroid is thus enmeshed in a desperate defense maneuver at the endocrine level.

3. The specific involvement of the thyroid is believed to be mediated by severe armorings of the jaw and throat. These also result in autonomous hypersecretion through the mechanism of local hyporgonia—cellular "panic."

4. Antithyroid hormones in hyperthyroidism appear to be the result and not the cause of the disease and perhaps reflect degrees of bionous deterioration in the thyroid gland. The variations in antibody levels are discussed.

5. The treatment of hyperthyroidism first requires control of the runaway gland by traditional conservative drug therapy. Prevention of relapse and ultimate control can then be attempted through orgonomic work on the underlying stasis.

REFERENCES

1. Reich, W.: *The Discovery of the Orgone, Vol. II: The Cancer Biopathy.* New York: Orgone Institute Press, 1948.
2. Werner, S. C., and Ingbar, S. H.: *The Thyroid,* 3rd Edition. New York: Harper and Row, 1973.
3. Dew, R. A.: "The Biopathic Diathesis (Part V: The Pulmonary Biopathies)," *Journal of Orgonomy* 6:187-200, 1972.
4. Reich, W.: *Cosmic Superimposition.* Rangeley, Me.: Orgone Institute Press, 1951.
5. Bondy, P., et al.: *Yearbook of Internal Medicine.* Chicago, Ill.: Yearbook Medical Publishers, 1972.