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The Biopathic Diathesis

(Part IV: Arteriosclerosis and Coronary Artery Disease)

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Introduction

Much effort has been expended on the problem of coronary artery disease. It is the most common cause of death in the United States and is the most frequently encountered lethal form of arteriosclerosis. Particularly alarming is that relatively young people appear to be afflicted in increasing numbers.

To review in depth what is known about arteriosclerosis and coronary artery disease is beyond the scope of this paper. However, it is necessary to lay the facts before us to discern the functional relationship in seemingly unrelated bits of information.

The overwhelming majority of cases of coronary artery disease, whether manifest as occlusion with infarction, with or without antecedent angina, occur in a setting of atheromatosis or arteriosclerosis. We will exclude from our discussion coronary insufficiency and/or occlusion related to aortic stenosis, dissecting aneurysm, syphilitic aortitis, congenital malformations, etc.

Atherosclerosis

It would appear that to a large extent the problem of coronary artery disease (CAD) is the problem of atherogenesis. The literature on this subject alone could fill a library; however, most of it boils down to a relatively few recurring themes:

1. *Atherogenesis is the result of abnormalities in lipid synthesis and/or metabolism due to:*

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A. *Genetic factors:* There are forms of familial hyperlipemia associated with elevated blood lipids, xanthomata, and precocious atherosclerosis with cerebrovascular and cardiovascular disease, etc. This is felt to represent one end of a spectrum, and the evidence for direct genetic transmission is quite strong. At the other end is the overwhelming majority of patients with CAD in whom no clear pattern of inheritance is seen. The hypothesis of genetic transmission is retained, however, and the concept of variability of genetic penetrance is advanced to explain these *sporadic* cases.

B. *Dietary constitution:* The controversy regarding the significance of high cholesterol and saturated animal fat intake has raged for years. However, there seems to be very little doubt in the minds of most clinicians and researchers that the quality and quantity of dietary fats have a definite bearing on atherogenesis. This idea is obviously not at variance with the concept of a genetic defect. One can see that if a metabolic defect exists in the handling of lipids or their structural components, then an increased loading of these metabolic pathways might saturate an already defective system. A good example of the amalgamation of genetic and dietary factors can be seen in the work of Frederickson and co-workers who with electrophoretic and other techniques were able to distinguish at least five different groups of hyperlipemia, some of which are *carbohydrate-* rather than fat-induced (1). These have been shown to be more common in younger persons with CAD.

It should be emphasized here that the lipid abnormalities found in the various hyperlipemias are of a *quantitative* nature. That is, it is the *total amount* and *ratio of one to the other, not* the chemical constitution which is believed to be at fault.

C. *Cigarette smoking:* The very interesting suggestion has recently been made that the inhalation of cigarette smoke has a bearing on the constitution of plasma lipids and hence atherogenesis (2), a mechanism quite apart from the effects of cigarettes on cardiac function itself (see below). This raises the whole question of a possible relationship between planetary atmospheric pollution and the increasing incidence of atherosclerosis.

D. *Obesity, diabetes, myxedema and physical activity:* These factors need no elaboration here. Suffice it to say that they have more or less definite effects on the lipid constitution of the blood and the walls of vessels. Hyperlipemia and hypercholesterolemia are frequently associated with diabetes, hypothyroidism and obesity. A sedentary life is said to carry with it a greater risk of CAD.

2. *Atherogenesis is the result of injury to the blood vessel wall.*

This is a highly controversial area in classical medicine, but one which I believe should be extremely fruitful if a functional approach is made to the discoveries therein. Essentially, what is implied is that a series of traumatic effects on the arterial intima *allows* the vessel wall to be infiltrated by the plasma lipids, producing initially the "soft" collection of fat referred to as an atheroma or atheromatous plaque. Due to the continuous accumulation of plaque, the endothelium is destroyed and calcium salts are deposited. Calcification completes the picture of arteriosclerosis. The disruption of the smooth vascular endothelium promotes focal agglutination of platelets, which activates the enzymatic "cascade" or blood-clotting, culminating in the deposition of fibrin. Occlusion of the vessel presumably may result from the continued deposition of fibrin and/or the undermining of a plaque by the stream of blood, causing it to lift free of the wall and block the lumen acutely.

What is most interesting here are some of the mechanisms involved in initiating trauma.

A. *Hypoxia:* A lowered oxygen tension in the blood produces decreased vitality of the endothelial cells, causing degeneration. The difficulty with this hypothesis is that certain conditions associated with chronic hypoxia (*e.g.,* chronic pulmonary disease) are not characterized by an unusual incidence of arteriosclerosis. As a matter of fact, clinicians have long noted a somewhat lower incidence of coronary artery disease in patients with chronic obstructive pulmonary disease.

B. *The "hypercoagulable" state:* In recent years, attention has been drawn to the increased readiness of the blood to clot in patients with established arteriosclerosis. It has been shown that there are increased levels of blood-clotting factors and increased "stickiness" of the platelets in these patients. Spontaneous platelet agglutination may occur, further enhancing the clotting mechanism and liberating proteolytic enzymes (*e.g.,* thrombin) which attack the vascular endothelium. It is of interest that increased sympathetic tone is associated with a rise of fibrinogen and other clotting factors in the plasma.

C. *Hypertension:* It has been known for a long time that hypertension accelerates the process of arteriosclerosis, particularly at the branching points of vessels. Hypertension directly traumatizes the vascular endothelium by producing turbulence which has an erosive effect and leads to microscopic endothelial leakages, which enhance the influx of lipid and the adherence of platelets. Hypertension would appear not to be a primary factor in the production of CAD, since a good many pa-

tients have no evidence of elevated blood pressure, past or present.

D. *Turbulent flow:* Atherosclerotic changes, even in the absence of hypertension, tend to occur at branching points in blood vessels. It is believed that the vulnerability of branching points is based on the disruption of the smooth or *laminar* flow of blood, observed in straight segments of arteries. Thus, the eddying of the plasma and the formed elements therein collide forcefully with the vascular endothelium at these points. In short, there is more wear and tear on the arterial lining where it branches. Without belaboring the obvious, this phenomenon simply serves to indicate that trauma, albeit physiologic and inevitable, does play a role in atherogenesis. Its *practical* significance is that, since atheromata in themselves generate turbulence, they are perforce self-perpetuating. Consequently, one observes that atheromata tend to form *proximally*—usually at branching points—and spread *distally.*

Non-Arteriosclerotic Factors in Coronary Artery Disease

A quick review of anatomy reveals that the blood supply of the heart is peculiarly "marginal" compared to, say, the gut, brain, liver, or lungs. Normally, the heart has a poorly developed collateral circulation. Furthermore, of all our organs, only the heart squeezes itself pallid in the natural exercise of its function. Coronary blood flow is free only in diastole. Thus, the circulation of the heart is easily compromised.

Secondly, like the brain, the heart muscle cannot regenerate. Destroyed fibers are replaced by scar tissue and the remaining muscle must *hypertrophy* to maintain functional capacity. Since the physiologic requirements of hypertrophy may readily outstrip an often fixed blood supply (*e.g.,* supervening arteriosclerosis), there can be but one outcome—further necrosis and fibrosis.

Thirdly, the heart has a protean involvement in the physiological processes of living. It is rarely exempt from some role in any of the innumerable homeostatic mechanisms which sustain life.

Cigarette smoking, air and chemical pollutants, etc.: Reference to the role of cigarettes in atherogenesis has already been made. In addition, nicotine effects on the vascular tree and the effect of carbon monoxide on the oxygen-carrying capacity of the blood increase the work of the heart. Cigarettes are known to precipitate angina pectoris (3). Certainly air pollution, a good deal of which is in the form of carbon monoxide, must produce similar effects, as well as those induced indirectly by impairing pulmonary function.

Coronary Insufficiency

Whether a patient has angina pectoris or a myocardial infarction, we may say he has coronary insufficiency. In the first case, the insufficiency is of lesser consequence or so transient that, while ischemic pain is experienced, clinical and laboratory evidence of heart muscle destruction is absent.[1] In the second case, the insufficiency is so gross and/or prolonged that the myocardium suffers acute asphyxia and dies. Since the necrotic tissue is replaced by fibrosis, the region may no longer give rise to ischemic pain. Thus, the survivor of an infarct who previously had suffered from angina may be "cured" of the angina as a result of the infarction.

That coronary insufficiency occurs when the oxygen requirements of the heart muscle may no longer be met by the blood supply can be readily appreciated. Therefore, coronary insufficiency with angina and/or infarction may supervene when either the coronary flow is *decreased* (*e.g.,* arteriosclerosis, vasospasm, shock) or when the demands of the myocardium are acutely or severely *increased* (*e.g.,* physical or emotional stress, hyperthyroidism and other "high output" states).

The heart and its nutrient vessels receive both sympathetic and parasympathetic (vagal) innervation by which their function is integrated with the general vasomotor tone. Discrete sensory bodies representing the afferent limb of the cardiac and vasomotor regulatory system are present in the central nervous system, the neck (carotid sinus), and the arch of the aorta. These bodies are sensitive to either changes in blood pressure or oxygen or carbon dioxide tensions in the blood. The sympathetic (adrenergic) impulses:

(1) increase the heart rate and the force of contraction,
(2) *dilate* the coronary arteries, and
(3) shunt blood *out* of the gut, skin and voluntary muscle into the heart and brain by constriction of the peripheral blood vessels.

The parasympathetic (cholinergic) exerts essentially opposite effects.

Now if we examine these vasomotor activities, we seem to encounter a paradox. If the sympathetic system *dilates* the coronaries and shunts blood preferentially to the heart and brain, and the parasympathetic does the reverse, the parasympathetic function would appear to be more likely to contribute to insufficiency. But clearly, this is *not* so. Clinical pharmacology and physiotherapeutics demonstrate beyond any doubt

[1]Actually, clinical and autopsy studies have shown that longstanding, recurrent angina may lead to myocardial fibrosis so that in essence these patients are suffering over a long period what the infarction victim experiences within a relatively brief span.

that interference with the *sympathetic* impulses by, for instance, beta-adrenergic blocking drugs (propanolol) or carotid sinus stimulation *alleviates* angina pectoris (4, 5).

The explanation of this paradox is quite simple. While vagal stimulation induces coronary vasoconstriction and otherwise indirectly reduces coronary blood flow, its overriding effect is to decrease peripheral vascular resistance by dilating the splanchnic vessels, the vessels of the skin, etc. This decreases the head of pressure against which the heart must pump. Furthermore, by slowing the heart rate, the work of the heart and hence its metabolic demands are additionally reduced. These benefits offset the decrease in coronary flow. Support for this explanation is found in the modern elucidation of the action of nitroglycerin. For a long time, nitroglycerin was thought to alleviate angina by inducing coronary vasodilation. While this does occur, careful study has shown this effect to be insufficient to explain the drug's benefits.

Nitroglycerin causes a universal vasodilation; that is, peripheral dilation of arteries and veins. This produces flushing of the skin and "pounding" headaches—side effects well known to nitroglycerin users; but at the same time, it results in peripheral pooling of blood with a decreased return to the heart, thus reducing myocardial work.

The Functional Overview

Essentially, my task here is (a) to establish a theoretical framework for the causation of CAD in terms of the functional concepts of orgonomy and (b) see how, if at all, the great body of knowledge gathered by medical science fits and lends support to this hypothesis.

The Antithesis of Autonomic Life Functions

This concept of Reich's, advanced by him even before the discovery of the orgone, is the foundation of our comprehension of the biopathies. Let us now review it in connection with the cardiovascular system.

The functions of expansion and contraction in the human organism are mediated through the parasympathetic and sympathetic autonomic nerves. Healthy functioning requires free pulsation in both directions. Conversely, chronic contraction (sympatheticotonia) or hyperexpansion (parasympatheticotonia) constitutes *stasis* (*i.e.,* decreased or absent pulsation).

In the biopathies thus far discussed in the present series of articles, the apparent mechanism of the pathologic physiology has appeared to be a violent, reactive parasympatheticotonia (expansion against con-

traction). In the cardiovascular biopathy, the opposite seems to be the case—*i.e.,* contraction against expansion, and hence a marked reactive sympatheticotonia.

The Role of the Thoracic Block

Those of us who have done orgone therapy have repeatedly witnessed what may be the anlage of arteriosclerotic heart disease (ASHD). Patients free of the stigma of coronary artery disease complain of a feeling of tightness or constriction in the chest. One of my patients who had a recurrent myocarditis of undetermined origin (presumably viral) even complained of chest *pain. In every case, deep sadness and longing came to the surface when the chest segment was mobilized.* Suppressed anger is frequently present; however, it always seems to be superficial to the longing and, in many cases, these patients are manifestly capable of expressing aggression. Many are phallic types who might be best described as "feisty" or "bristling." On the couch they often seemed so angry that one was inclined to let them punch and "yell it out." In some, this would lead to sadness. Others would not get anywhere; they would even appear to be enjoying themselves, raging session after session. Only after changing one's tack biophysically and characterologically would a breakthrough occur. Inevitably, what came out was sadness and longing. In fact, in these latter cases the stalemate might be broken by having them stop their raging because it had become a *defense* against the deeper feeling. Therefore, while these patients undoubtedly had in them genuine rage, it would appear as a *substitute contact* phenomenon. In this case, the core emotion is longing which takes the form of rage in the secondary layer.

An understanding of the biophysical meaning of *longing* is essential to our thesis. Generally, we experience longing as a keenly felt urge to reach out. Reaching or "moving out" represents an energetic flow from the core to the periphery, and consequently we identify it with the activity of *expansion.* Expressions in our language leave no doubt that mankind has spontaneously tied the heart to the feeling of longing. We "long with our hearts." We are "broken-hearted" when our longings are not gratified.

Expansion in the cardiovascular realm corresponds to dilatation of the peripheral vasculature with the resultant increased flow of the highly charged blood to the skin, etc. Since this is a parasympathetic activity, we may say that longing, expansion, and increased parasympathetic tone are functionally related. It is clear from our clinical experiences that this function in patients is constrained by the thoracic

armoring. We observe the chest to be held high in an *inspiratory attitude.* The intercostal muscles are contracted and are "ticklish" or painful. The accessory muscles of respiration—anatomically originating in the cervical segment—are tense. These observations are of particular significance when one considers that the peripheral autonomic motor fibers to the heart and its vessels originate in or pass through the neck.[2] The diaphragm is *depressed.* Diaphragmatic contraction may play a role in the not infrequent association of gallbladder disease and coronary insufficiency. The thoracic armoring is functionally identical to the increased sympathetic motor tone of thoracic sympathetic fibers to the heart.

In any case, elimination of the chronic inspiratory attitude, by orgone therapeutic techniques, allows the chest to fall in expiration. Accomplishment of full, spontaneous expiration in respiration is accompanied by the appearance of sadness and longing. This suggests then that the abnormal inspiratory attitude of the thorax corresponds to *contraction* or sympatheticotonia. Its dissolution invariably produces *anxiety.* It is evident that, as usual, the armoring functions to preserve the patient from unbearable anxiety. Consequently, the keener the longing (expansion), the more intense the anxiety (contraction against the expansion) will be and the more severe the armoring. We have established in this situation a setting of chronic sympatheticotonia. But how does this produce heart disease?

As has been repeatedly asserted, the victim of the cardiovascular biopathy is emotionally *alive.* Unlike the cancer patient, he does not yield easily to the inexorable process of contraction. This "not yielding," "fighting back" biophysical characteristic involves sharp efforts to break through; that is, to expand against the contraction. The stasis occurs then at a high energy level. The chest becomes a battleground, as it were, in which powerful forces are literally locked in combat. It is obvious that in most cases the patient *does* control himself. There are outbursts of anger which might be viewed as minor "leakages" of energy through the armor in secondary drive form, but the great bulk, the fundamental longing, is not fully expressed. We might then infer that the forces of contraction "have the edge." In this physiologic, emotional tumult, the heart must function. It must answer the needs of a lively organism attempting to master its own anxiety. We would postulate that the sympatheticotonia, with its rapid and vigorous myocardial contraction (increased effort), is accompanied by a parasym-

[2]Vagus nerve—parasympathetic.
Stellate ganglion—sympathetic.

pathetically-induced coronary vasoconstriction (decreased blood supply). This fits well with the observation that "emotional upsets" frequently precipitate angina. Furthermore, the chronic recurring bouts of coronary vasoconstriction may render the coronary endothelium susceptible to the hypoxia and other insults elucidated earlier, resulting in arteriosclerotic changes, irreversible insufficiency, and irreparable organic myocardial disease. This is the typical pattern of the biopathic disease proposed earlier: that is, *functional changes precede organic changes* (6). Such events might explain the occasional cases in which clinically clear-cut infarction occurs, but the postmortem shows *no* arteriosclerosis of the coronaries.

The reader might ask at this point why, if simultaneous powerful parasympathetic and sympathetic discharges are occurring, the coronary arteries are selectively influenced by the parasympathetic, while the peripheral vessels seem to be clearly dominated by sympathetic influences? One may recall in answering this important question the features of these antithetical functions. *Contraction starts at the periphery and proceeds toward the core, while expansion stems from the core and spreads toward the periphery.* The heart, anatomically and physiologically, is literally the "core" of the vascular system.[3] Consequently, *primary* autonomic expansile impulses (parasympathetic) are initated, and might reasonably be stronger, at the level of the heart than at the periphery. The word "primary" is important here since we have said that the heart is working harder in this biopathy (under sympathetic influences). It must be kept in mind, however, that the increased effort of the heart is *secondary* to contractile changes in the periphery. That is, its response to increased peripheral resistance is increased vigor of contraction (Starling's Law). This concept may be of value in understanding peripheral vascular arteriosclerotic occlusive disease. Here contractile forces would naturally predominate and hence little physiologic evidence of parasympathetic counteractivity is seen. The sympathetic action in this case would be *primary.*

The Role of Atherogenic Factors

Earlier in this paper, we enumerated some of the classical views on atherogenesis. A few words are in order to place these factors in perspective in our functional survey. While traditional medicine has many clues as to the cause of atherogenesis, a fundamental understanding of

[3]The Latin word *cor* means "heart," though it has other meanings, too, such as "body" or "horn."

its origin remains lacking. Although genetic and dietary factors in one's lipid "constitution" are undeniably relevant, I am inclined to believe that they bear the same relationship to atherosclerosis as cigarettes to lung cancer. If there is fertile soil (on biophysical grounds) for atherosclerosis, then the factor of one's plasma lipids, perhaps ordinarily of less significance, assumes greater importance in the presence of chronic sympatheticotonia. Furthermore, even plasma lipid is highly variable and is undoubtedly affected by our emotional lives. Stress is known to stimulate a rise in the serum cholesterol (7). In epidemiological studies of arteriosclerotic cardiovascular disease, it was the ambitious, driving, "executive" types with more responsibility who as a group had higher serum cholesterol and incidence of CAD (8).

It is the concept of intravascular trauma which intrigues us most since it lends itself so well to our view of the situation. Let us deal first with the idea of vascular endothelial and intramural hypoxia. The large and medium-sized vessels receive their oxygen and nutrition directly from the blood coursing through them and via the vasa vasorum, a fine network of arterioles infiltrating the layers of the arterial wall. This network must traverse and supply the smooth muscle coat immediately beneath the adventitia. It is obvious that spasm in this muscular layer would compromise circulation to the deeper structures (*e.g.,* the intima). In fact, in diabetes mellitus, the precocious arteriosclerosis is attributed to the blockage of the vasa vasorum by glycoprotein deposition. Thus, we have reason to believe that this circulation is not only vital, but is in some way connected to the phenomenon of arteriosclerosis. It is tempting to postulate that spasm of the smooth muscle layer produces hypoxia and hyporgonia of the intimal and medial layers, creating the extraordinary susceptibility to wear and tear normally repaired by regenerative activity in the endothelium, etc. Thus the circumstances for atheromata formation are produced.

Before accepting this hypothesis, we must answer one important objection. If vascular hypoxia is the underlying pathophysiologic event in atherogenesis, why is it we don't see more atheromatosis in patients with, say, chronic pulmonary disease in which long-standing and severe hypoxia may be an outstanding feature? For one thing, in these patients there may be varying degrees of hypercapnia; that is, elevated partial pressure of CO_2 in the blood. Hypercapnia tends to produce vasodilatation, which is partially responsible for the clinical appearance of these patients, that is, the flushed color of the skin (wherefore they are often called "pink puffers"), which is a distinct contrast to the pale, cool skin seen in an ischemic anginal attack or peripheral vascular in-

sufficiency. It would appear that the vasomotor dynamics in chronic obstructive pulmonary disease are quite different from those of ASHD. Is this strictly on the basis of CO_2 retention? We know that O_2 transport from the alveoli into the blood is much more susceptible to disturbances in ventilation-perfusion relationships[4] than is CO_2 transport *out* of the blood into the alveoli. Hence, the earliest blood gas abnormality to be manifest would be hypoxia. Hypercapnia would tend to occur later when: 1) obstruction to airflow is diffuse and more severe and 2) there is more alveolocapillary membrane disease to interfere with the usually facile transport of CO_2. In any case, hypercapnia would tend to be accompanied by a simultaneous and more marked hypoxia. Nevertheless, this does not appear to be associated with an unusual degree of ASHD or peripheral vascular occlusive disease. Clearly then, the blood gas constitution is not a primary factor in atherogenesis.[5] Furthermore, significant blood gas abnormalities are rarely seen in uncomplicated coronary artery disease. Interestingly, chronic obstructive pulmonary disease frequently leads to *pulmonary vascular hypertension* which, with prolonged survival, produces arteriosclerosis in the pulmonary arteries. This is a further indication of the role of vasospastic injury in arteriosclerosis.

In summary, then, the arteriosclerotic peripheral vascular and CAD patients have a predominantly sympathetic tone in the periphery, while in chronic lung disease it is mainly parasympathetic. This fundamental difference may account for their often divergent fates with regard to atherogenesis. Even more important, and really the main point of this digression, is that vasoconstriction is the main factor in atherogenesis, whether it be in the peripheral vascular or pulmonary vascular beds. The degree of arterial hypoxia due to vasospasm must constitute a much more serious insult to the arterial wall than hypoxia inflicted by low O_2 tension in the blood itself.

I believe it would be helpful at this point to summarize our picture of the events leading to atherogenesis in general and coronary atheromatosis and insufficiency in particular.

(1) The human organism is blocked in the free discharge of its sexual energy by virtue of its armoring; *e.g.,* orgastic impotence.

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[4]The modern theory regarding chronic obstructive pulmonary disease is that hypoxia and hypercapnia are partly the result of portions of the lung being perfused by the blood, but simultaneously lacking adequate ventilation (*e.g.,* due to obstruction of the airways).

[5]Carbon monoxide may indeed be a primary factor in atherogenesis (2).

(2) As a result of this block, charge builds up leading to a state of heightened orgone energy tension.
(3) The increased tension produces anxiety (expansion against armoring—fear of breaking through).
(4) The organism strives to control the anxiety by additional contraction (sympatheticotonia).
(5) In the cardiovascular realm, the sympatheticotonia is manifest as chronic vasospasm in the periphery.
(6) Chronic vasospasm results in hypoxic (ischemic) injury to the arterial wall by compressing the vasa vasorum.
(7) Injury to the arterial wall in the face of impaired regenerative capacity permits infiltration of lipid, thus initiating atheromata formation.
(8) In the case of the CAD biopathy, the increased charge due to orgastic impotence produces intense longing and sadness which is a form of expansion in the chest.
(9) The site of increased sympatheticotonia is predominantly the chest, which is armored, held high, and fixed in inspiration.
(10) At the core of the vascular system, which in this case is very lively, renewed attempts to break through the stasis appear in the form of increased parasympathetic tone producing coronary vasoconstriction, while simultaneously at the periphery the contraction increases the work of the heart.
(11) The unfavorable combination of increased demands with decreased blood supply produces coronary insufficiency.
(12) The chronic coronary vasospasm allows atherogenesis to occur as described above, producing angina and/or infarction.

From the above analysis, we may understand how a high energy level would promote the biopathic process; *i.e.,* there is an ever increasing push to expand, against which there is a rising need for armoring.

Orgone Therapy in CAD

It is clear from the foregoing considerations that orgone therapy must have as its goal relieving the patient of the spasms of the chest, neck, and diaphragm to permit the expression of his sadness and longing. Ultimately, where possible, a more ambitious therapeutic goal requires the establishment of orgastic potency. However, there are particular risks involved in the management of this biopathy. Reich recognized how dangerous is the treatment of such patients.[6] Furthermore, since

[6]Personal communication with Albert I. Duvall, M.D., who stated that Reich said orgone therapy was contraindicated for angina patients.

a rapid resolution of the pertinent blocks cannot always be assured, one must not ignore proper medical therapy in the interim.

It is not my purpose here to review the medical treatment of CAD, but to indicate the importance of implementing what is known. It is obvious that there is particular urgency in eliminating hyperlipemia, obesity, and hypertension. Diabetes, cigarette smoking, and other aggravating conditions should be controlled. Current treatment of these factors often leaves much to be desired, but I think sufficient evidence of its benefits has been accrued to warrant a conservative attitude.

One should not forget that it may befall the orgonomist to make the primary diagnosis of CAD. Some care should be taken to elicit a history of CAD even when it is not volunteered. It is particularly vital to rule out the diagnosis, and, in doubtful cases, appropriate studies should be done under the supervision of a medical specialist. The electrocardiogram (ECG) is an excellent screening test and should be done on all patients with a positive or questionable history. It should be routine in all men over the age of thirty-five and all post-menopausal females. A convincing history of angina, however, takes precedence over all other modalities of diagnosis, even in these days of coronary cineangiography. Fortunately, an ECG, especially in combination with an exercise tolerance test (*e.g.,* "Double Master Test"), will settle the question in the vast majority of cases.

Candidates for Therapy

In general, patients with angina at rest, fresh myocardial infarctions, or frank congestive heart failure should not be subjected to the rigors of full-blown orgone therapy.[7]

In patients with infarctions, it would probably be wise to wait six months to a year before resuming or beginning biophysical work,[8] although cautious character analytic therapy may be permissible (see below). In cases of congestive heart failure not due to infarction, normal therapy may be pursued only after the underlying cause has been determined and treated (*e.g.,* supervening hypertension, infection, hyperthyroidism), and the failure has been controlled. Asymptomatic patients with abnormal cardiograms should be investigated medically. Provided that they have not had a recent "silent" infarction, I can see

[7]Dr. Elsworth F. Baker has treated one patient with angina at rest who insisted on continuing therapy despite the risk. Considerable improvement (*e.g.,* increased exercise tolerance and decreased frequency of attacks) resulted.

[8]This is the standard medical recommendation regarding elective surgery.

no reason for denying them the benefits of orgone therapy if certain precautions are observed.

Special Precautions in the Therapy of Patients with CAD

The following recommendations are based on experiences in standard medical practice with respect to stress in CAD and are therefore not specific for orgone therapy.

1. In patients with documented or suspected CAD, the *apical* cardiac impulse should be ausculted or palpated periodically during the session (*e.g.,* q. 15 minutes and after stressful experiences). All activity should be suspended if there is:

a. sustained tachycardia (rate > 100/min. for longer than 5 minutes),
b. paroxysmal tachycardia (frequent bursts of rapid heart action in excess of 120/min. for 1 minute or longer),
c. gross irregularity in the heart rhythm unrelated to respiration,
d. unusually slow heart rates (< 50/min.), or
e. persistent heart rate in excess of 90/min. in a resting, calm patient with a history of heart failure.

2. The appearance of chest pain with exertion or excitement during therapy probably, but not necessarily, indicates ischemia. Stopping the work is probably the safest course to follow. One should be reminded that cardiac ischemia may present in an aberrant form in which actual chest pain is absent. A choking sensation, pain in the neck, jaw, shoulder, or upper arm, or merely paresthesias in either or both hands may be the only manifestation. The patient may complain of nausea or "indigestion." All these symptoms are commonly seen in therapy with patients having no heart disease whatsoever. It would be a dangerous error to ignore their significance in the patient with CAD.

One is also reminded that characterologic work may have a jolting effect on patients. There obviously may be great physiologic upheavals during discussions in "sensitive areas," despite an outward calm. This could be of great danger to the patient with a recent history of myocardial infarction or severe angina.

3. Dyspnea, orthopnea, shortness of breath out of proportion to effort, and coughing are symptoms of great significance in this context. They all may indicate failure of the heart. Indeed, heart failure rather than chest pain may announce the presence of an acute myocardial infarction.

The DOR Effect in CAD

In concluding our discussion on CAD, I should like to mention briefly the possible role of DOR in this biopathy. A complete description of DOR sickness is available elsewhere (9). Suffice it to say that some of its manifestations, such as a feeling of oppression in the chest and edema, are commonly found in persons with CAD. One cannot help but feel that there is a functional identity in this situation.

The other important implication of DOR saturation in CAD is its possible correlation with the rising incidence of CAD, as well as other forms of arteriosclerosis. It may well be the rising concentration of DOR in our atmosphere is a major factor in the increasing frequency of CAD. A DOR-laden atmosphere, whether it be the result of air pollution or the crowding together of vast numbers of armored people, must make expansion very difficult for the human organism. This may be part of what we are talking about when we speak of the "rat race" to which we attribute the death, illness, and insanity of so many of our number.

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