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Chronic heart failure

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CHRONIC HEART FAILURE

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CHRONIC HEART FAILURE

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CHRONIC HEART FAILURE

I. Forward

1. Personal reasons for choosing subject

When it comes time for a senior in medical school to write his senior thesis, he is first confronted with the problem of choosing a subject and next perplexed as to the method of presenting his chosen topic. One or two days spent in the files and archives of the library convince him that he can never completely and thoroughly cover a subject and at the same time remain in medical school the last semester. Consequently, he thinks to himself that perhaps he should pick some subject about which little is known and less written and get the whole thing over with as quickly and painlessly as possible. After more mature reflection, however, he decides that since it is the first and last time he will ever be called upon to perform such a task, he should appreciate the privilege of becoming a graduate in medicine and honestly and wholeheartedly attempt to construct a paper worthy of one so trained. Inspired somewhat by this decision, he sits down and reflects as to what phase of medicine would

repay him most for the energy expended.

Needless to say, no one phase of medicine is more important than another but doubtless there is some particular topic which to him seems most interesting. Perhaps it is a particular organ of the body which confused him in anatomy and physiology and completely confounded him in medicine and surgery later. Or it might be a specific disease whose etiology and therapy intrigued him.

In my case it was a combination of the two. The anatomy of the heart, its peculiar musculature, its intricate valvular system, and its efficient circulatory system all captured my fancy as a crude dissector in the anatomy laboratory. The fascinating study of the extrinsic and intrinsic nervous mechanism of the heart still further interested me in physiology. The action of the various drugs and ions which accelerated or depressed the heart action proved one of the most interesting phases of the basic years.

Then later on in clinical medicine, observing both young and old afflicted with faulty

hearts and realizing keenly how such an affliction would have handicapped me in my life and realizing that as a physician, particularly one whose father and father before him, had died with a coronary, I was quite likely to develop a cardiac lesion, I again became acutely aware of cardiac disease. It is small wonder then that I chose to spend my time writing about the heart.

Determined to write about some phase of heart disease, I thought to myself, what about the heart most perplexes me? In my mind there immediately arose the vague question of decompensation, heart failure, cardiac reserve, the law of the heart, coronary thrombosis, hypertension, rheumatic fever, how are all these related? Consequently, I decided to see if I could untangle this confusion in my own mind. As a result I chose the topic Chronic Heart Failure.

2. Method of handling subject

Now well satisfied with my chosen topic and content to spend the necessary time and energy investigating cardiac decompensation, the problem arose as to how to present the fruits of my reading and organizing. It is obvious that

I can contribute nothing original to the subject. Neither can I speak as an authority on what has already been investigated. The idea of criticizing such authorities as Levine, Warfield, Berger, Harrison, Mackenzie, Lewis, and others, to me seems ludicrous in the extreme. Also, after having spent many hours laboriously sifting a salient fact here and there from a world of articles concerning unheard of formulae, definitions, ballistocardiographs, metabolic phenomena and what not, I am convinced that one as inexperienced as I in the art of medical investigation may easily become lost in a forest of isolated facts.

Therefore, I have attempted to organize in logical sequence the knowledge that I have absorbed in various articles. Throughout this thesis I have kept in mind that, without doubt, and despite the theoretical aim of the paper to prove or disapprove a problem, it is I myself who is going to derive most benefit from this humble endeavor. Consequently I have written the paper so that the pertinent facts will remain in my mind. Whenever I could make a general statement that it is probable I might remember I have used it rather

than tediously explaining specific details in great number which of course would remain only as isolated and unrelated questions. For example, in discussing etiology, it seems so much more logical to remember three classes of disease which are the chief causes of cardiac decompensation, namely valvular, coronary, and hypertensive disease rather than discuss all the various conditions which can cause valvular or coronary lesions or hypertension.

In the style of composition, although the first person is implied and the paper is written in an authoritative manner, I do not wish the inference to be drawn that the author considers himself an authority on chronic heart failure. In each case where I have spoken authoritatively I have given complete credit to the correct author from which the statement was made.

3. Practical reasons for choosing subject

These personal reasons and explanations having been dispensed with, be assured that they are not the only or necessarily the chief reasons for choosing the topic, Chronic Heart Failure. The importance of the disease to the medical profes-

sion alone is enough to warrant its choice as a topic. For example, a slightly less than one third of all deaths in New York State in 1935 were due to heart failure. (32) It is by far the greatest cause of all deaths. Is it any wonder then that a medical student would be interested in this subject from a practical point of view?

Although the medical profession never loses sight of its responsibility to all patients whatever their economic and social standing, a class of patients prone to coronary and hypertensive heart disease, are especially interesting patients. Generally they are those persons with a dynamic personality, interesting if for no other reason. This type of patient is also more keenly aware of his responsibility to help make the world a better place in which to live. He is more stabilized socially and economically, and for this reason too, it seems a physician should derive a great deal of satisfaction from helping him.

Before undertaking the study of medicine, I spent three years as an instructor in mathematics and science and as athletic coach in a pub-

lic high school in Nebraska. The one most definite concrete impression that remains of that three-year period is that all men are not created equal either physically, mentally, or given equal chances in our professed democratic social order. Certain persons retarded by inherent handicaps must be carried by those more fortunately endowed by nature with potential abilities. My experiences in the dispensary have confirmed and strengthened my convictions. Therefore, believing that no matter what the patient's station in life, he deserves the best that the physician can give, it seems to me that a double service is performed if we can lengthen the productive period of those who also feel and bear the load of helping mankind in general.

4. Scope of thesis clearly defined

From the title one can see that the paper is limited to chronic heart failure. In this type of heart disease a slowly progressive history of prodromal symptoms can be elicited. The mention of sudden failure as a result of toxins, trauma, and fulminating precipitating factors is secondary and included because a knowledge of

such heart lesions is necessary to a potential physician in explaining the more underlying causes. Chronic heart failure then is to be discussed and in the discussion I have been most interested in the etiology and theory. Whenever etiology is discussed, although various phases of etiology should be mentioned, the principle analysis must be toward underlying and precipitating factors. My chief interest has been in the underlying causes, those various lesions which whenever recognized, no matter at which age in life, will ultimately cause exodus of the patient in terminal heart failure.

CHRONIC HEART FAILURE

II. Definitions

1. Chronic heart failure

By definition chronic heart failure may be defined as the inability of the heart to maintain adequate circulation of the blood despite utilization of any or of all compensatory mechanisms. Osler indicates much the same when he states: "With lessening of the muscular power of the heart the rapidity with which the blood circulates is diminished and the tissues fail to receive their proper supply of oxygen and food and to be adequately relieved of their waste products--this is cardiac failure." (30)

2. Cardiac decompensation

Cardiac decompensation implies that the heart has already compensated at least once so the term failure is inclusive of the loss of circulatory efficiency before there has been proper compensation.

3. Myocardial insufficiency

A term often used synonymously for heart failure is myocardial insufficiency. However, myocardial insufficiency can be caused by various toxins and injuries, which condition it is

true, will ultimately result in failure of circulation, but such cardiac insult is not to be exhaustingly discussed in this paper.

4. Congestive heart failure

On the other hand, congestive heart failure carries with it the implication that the systemic system of veins is engorged and the heart is embarrassed more by the backward pressure in the right heart. Such a condition is often a manifestation of chronic heart failure, it is true, but the left ventricle can fail due to various causes and the patient expire with few if any of the symptoms of congestive heart failure. (26)

(Over a period of years these above four terms have taken on their distinctive implications. However they are more or less used synonymously in this paper.)

5. Adequate circulation

In an analysis of the definition of chronic heart failure as stated, the phrase "adequate circulation of the blood" has been used. By this is meant that the heart to maintain adequate circulation must discharge as much blood as it receives.

6. Cardiac output

The term cardiac output is used and by definition means the quantity of blood pumped in unit time. The amount of blood delivered by the heart into the aorta (an equal amount is simultaneously pumped into the pulmonary arteries) is designated in terms of liters per minute, or as cubic centimeters per beat, the latter volume is called the stroke volume. (27)

7. Starling's Law

The principle generally known as Starling's (1918) Law of the Heart has been amply confirmed and may be briefly stated as follows: "The work per beat of a normal heart is a function of its size." To this may be added the corollary which is a definition of heart failure: "The weak heart is one which is doing too little work in proportion to its size." (35)

Can the conclusion be drawn then that the failing heart is always enlarged? The answer to this question is no. At first hand, this seems a contradiction of the corollary to Starling's Law of the Heart. However, if the heart is failing to expell as much blood as it receives, even

though it be normal in size, it is certainly failing to do the work required of it. (4)

8. Cardiac compensation

When a heart is failing to maintain adequate circulation, it makes various attempts at compensation and may be so successful that to all practical purposes circulatory efficiency may be restored. For clearness in composition it is necessary to define cardiac compensation. This term means the successful attempt of the heart to maintain adequate circulation of the blood despite either (a) an increase in the load placed upon it or (b) a decrease in its normal functioning ability.

9. Load of the heart

Here again a term has been incorporated in a definition which term so used needs further explanation in its own right, namely, the load of the heart. The term, load of the heart, includes two distinct physiological mechanisms: (a) the resistance against which the heart is working, i.e. the pressure in the aorta and pulmonary artery and (b) the pressure head determining its filling, i.e. the pressure in the ante chambers to the two ventricular pumps, the left

atrium and pulmonary veins on one side and the right atrium and venae cavae on the other. (18)

10. Cardiac reserve

The cardiac reserve therefore is an expression of cardiac compensation. Or, it is the added load that the heart is competent to meet. In other words, cardiac reserve can be measured by the load that can be added over and above that already existing without rendering the heart incompetent.

In the end analysis then, there are only two causes why the heart fails. These two main causes incorporate a variety of reasons which will be discussed at length. They are: (a) too great a load for the essentially normal heart mechanism and (b) weakening of the myocardiam or loss of cardiac reserve.

CHRONIC HEART FAILURE

III. Incidence

The most common cause of death is failure of the heart. (4) In New York State in 1935, 31.3% of all deaths were due to this cause. (32)

A table indicating the age incidence of the patients dying in heart failure in New York State in 1935 shows the following incidence as to be age of life.

55-64 yrs.----------17%

65-74 yrs.----------29%

75-84 yrs.----------28.2% (32)

Hence, cardiac failure is definitely a lesion of advanced years. In this age group when the heart begins to fail the vascular bed in the cerebrum takes the brunt of insult and often, though not necessarily, an apoplectic stroke accompanies the myocardial insufficiency.

Males with congestive heart failure outnumber females two to one. (35) Although females have more hypertensive heart disease, a fact not always appreciated, males have more coronary and luetic heart disease. The incidence of rheumatic fever seems about equally divided between the sexes. (38)

Although girls are much more susceptible, men on the whole show a larger proportion of the cases. The mentioning of these etiological facts will be discussed at greater length later on.

The social incidence of chronic heart failure is particularly interesting. (2) It has been implied that rheumatic fever is an underlying cause of chronic heart failure, also that luetic heart disease can eventually terminate in decompensation. Rheumatic fever is definitely, though not exclusively, a disease of the poorer classes and luetic heart disease is seen predominantly in the negro; but a large percentage of terminal heart failures have as an underlying cause coronary disease and hypertension which conditions are seen in the more privileged classes. Among this latter group there is a particular emotional type that seems predisposed to cardiac failure. Such persons could be described as the "go-getter" type of individual. It is often suggested that the pressure of social and economic life to which such persons are subjected contributes to the incidence of hypertension and degenerative vascular disease. Perhaps this is a misstatement and they may owe their business and professional successes to the cause of the hyperten-

sion rather than that the hypertension has resulted from their mode of life. (2) The classic example of this group are those members of the medical profession who characteristically are subjected to vascular accidents.

Incidence of chronic heart failure will be covered much more thoroughly as the etiology of heart failure is discussed. It is often quite difficult to draw a sharp line of demarcation between etiology per se and incidence of a disease.

CHRONIC HEART FAILURE

IV. Etiology

1. Underlying causes

It is not the purpose of this paper, as has been stated before, to enter into all the etiological factors of chronic heart failure. The underlying and precipitating factors will be considered but in the main the underlying causes are of most interest. According to Boyd, the three great causes of gradual heart failure are valvular, coronary and hypertensive heart disease. (4) An analysis of these underlying causes will show that they incorporate a variety of specific etiology.

For example, any valvular lesion whether stenosis or regurgitation, will have exactly the same result as far as increasing the load on the ventricle proximal to it. The chamber of the heart considered as being proximal to the valve is so considered in relation to the stream of blood. Any or all of the four valves of the heart can be involved and eventually cause termination in heart failure. Certain diseases or conditions have a predeliction for specific valves. Bacterial endo-

carditis, either acute or subacute, may affect any valve but has a predeliction for the mitral and aortic. On the other hand, congenital lesions are more common on the right sided valves particularly the pulmonary valve. Syphilitic aortitis, very commonly surrounding the arch of the aorta with a girdle of dilated vascular tissue, secondarily affects the aortic semilunar valve. Rheumatic fever, although it may involve other valves or the myocardiam, is notorious because of its predeliction for the mitral valve, ultimately causing stenosis. Mitral stenosis is perhaps the most common of the valvular defects with which heart failure is associated. (40) Also valvular defects may be secondary complications of other valve lesions which are primary. In general, the valves of the right side of the heart are more often involved as complications of left sided lesions. And in any valvular disease the inefficient valvular mechanism secondary to dilated myocardial fibers must not be overlooked.

Coronary disease could be summed up as coronary occlussion either acute or chronic. (2) Acute coronary disease is rare and is caused by

emboli usually from vegetations on a valve of the heart. (2) Emboli in the systemic circulation are trapped in the pulmonary capillaries before entering the coronary circulation. Likewise emboli from the intestinal vessels are first stopped by the portal capillary bed and then by the pulmonary. Chronic coronary occlussion is most commonly the result of arteriosclerotic narrowing of the vessels, the arteriosclerosis falling into the atheromatous type. Syphilitic aortitis of the root of the aorta may also seal the mouth of the coronary arteries, the right coronary artery arising from the anterior sinus of Valsalva and the left coronary artery from the left sinus. Coronary thrombosis may also occur clinically but it is less common. (2) Any thrombus forms when two essential requisites are at hand, namely, slowing of the blood stream and injury to the vessel wall. Occasionally coronary sclerosis may be caused by syphilitic arteritis of the coronaries themselves.

Hypertensive heart disease, i.e. chronic hypertension, may be divided into three groups. (40) First, before any rational approach to a

discussion of hypertension, it is necessary to establish some arbitrary level at which it can be said hypertension occurs. As good a rule as any might be when the diastolic reading is at 100 mm. of mercury. In practically all instances the systolic pressure will be above 170 mm. of mercury. (2)

One group of hypertensives consists of those patients with chronic nephritis. The etiology of the condition is obscure but the hypertension is characterized by a high pulse pressure. Clinically the symptoms are, briefly: polyuria, nocturia, albumen and casts in the urine, low specific gravity and edema. Pathologically the kidney is found to be small, red, and contracted. It is interesting to note that these patients are more apt to die in uremia than in congestive heart failure. (40)

Then there are the essential hypertensives in which there seems to be a definite hereditary trend. The hypertension is characterized by an exceedingly high systolic pressure. Clinically the patient is the robust, florid, exhuberantly healthy person. The urine is normal in amount, specific gravity, and color. There is no impair-

ment of kidney function and no edema. Pathologically the kidney is also small, red, and contracted but there are many fine granulations and many destroyed glomeruli with marked arteriolar changes. This patient is more apt to die because of an apoplectic stroke. (40)

A third group of hypertensives are those patients whose high blood pressure is due to arteriosclerotic changes. Such patients are usually more advanced in age and have led strenuous and dissipated lives both physically and mentally. The blood pressure readings are characterized by a high systolic, normal diastolic reading with large pulse pressure. Clinically there is no polyuria, nocturia, or albuminuria. The urine is normal in amount, color, and specific gravity. Kidney function tests are normal and the kidney pathologically is found to be large, firm, dark red with fatty streaks in the cortex. It is in this type of hypertension that death is usually due to cardiac decompensation. (40)

Although with all hypertensive heart disease there is insult to the heart and it makes effort to compensate, the majority of exit because of cardiac decompensation is in the arteriosclerotic

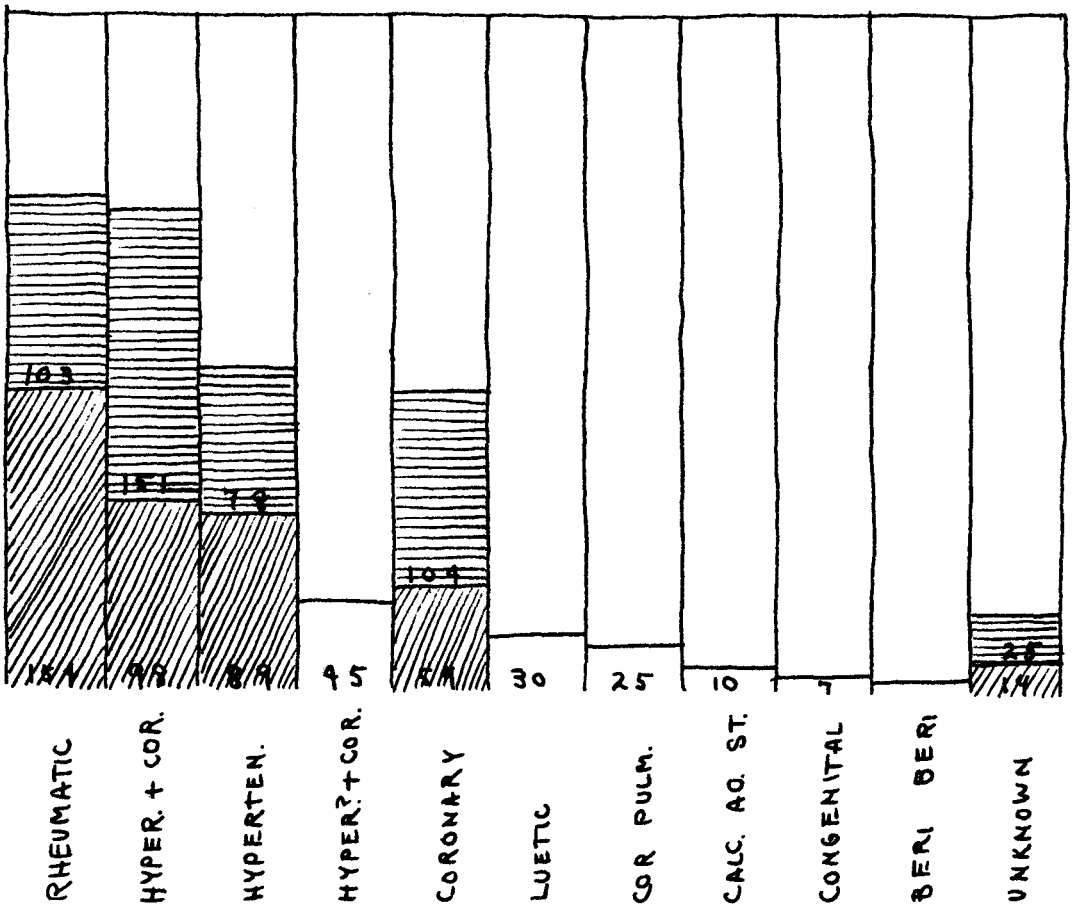
type of hypertension. Perhaps the mentioning of kidney so prominently is not encompassed by this article, yet because of the kidney hormonal theory of hypertension the mentioning of cardiovascular-renal disease is not too far amiss. In this theory it is assumed that there is liberated from the adrenal cortex a lipoid closely related to cholesterine which exerts its effect in one (or both) of two ways; either on the vasomotor center or on the capillary bed or muscular coats of the arterioles. (40)

Harrison in his book, Failure of the Circulation, lists four underlying causes which he states most commonly result in congestive heart failure. They are, hypertension, arteriosclerosis, rheumatic fever, and syphilis, and he further states that two thirds to three fourths of all cardiac disease is due to the first two. (14) These same four etiological types are also mentioned as being the most common underlying causes of failure by various other authors. (36) Since arteriosclerosis in itself can be a factor in causing both hypertension and coronary disease, there is no descrepancy between Boyd's and Harrison's classification. Harrison also

mentions various other less common causes including deficiency and bacterial diseases as well as chronic fibrotic conditions of the lungs which all totaled cause less than 10% of organic cardiac disease. It does not seem too important to list these various less common etiological factors.

An interesting as well as educational graph tabulating the percentage of various underlying causes of heart failure is the one that follows. An attempt has been made to correlate the etiology and the social incidence.

A series of one thousand cases among different types of underlying heart disease is graphically expressed. These cases are further divided into ward and private patients. In general, there is no great difference in the two groups except for a greater percentage of hypertensives and coronaries with less rheumatic heart disease in the private as compared to the ward patients. (2) The importance of poverty and poor hygenic conditions as predisposing factors in rheumatic fever is well recognized. The greater number of patients with coronary and



Legend: The entire column represents the total number of patients both ward and private; the cross-hatched area represents the number in the ward group and the horizontally lined area those cases among the private patients.

hypertensive heart disease in the private group is probably due to a number of factors: (a) the age incidence is somewhat higher and more likely to occur in patients with a stabilized earning capacity, (b) there seems no doubt that the pressure under which the successful, modern business and professional man exists contributes something to the incidence of hypertension and degenerative vascular disease, and (c) a possibility mentioned before, such patients may owe their success to the cause of hypertension rather than that their hypertension has resulted from their mode of life. (2)

The cases classified as unknown include all those in whom none of the recognizable forms of heart disease were present, who had no history of angina pectoris and who either had no electrocardiograph made or in whom the records were inconclusive.

It is obvious that the less common causes of heart disease whether infectious, metabolic, blood, or other disease are rarely underlying causes of failure; they are occasionally exciting factors when some more important underlying cause is present. Incidentally, the table offers a commentary on the uncommonness of congestive failure in congenital heart disease and emphasizes that the dangers to these patients lie in such complications as bacterial endocarditis and thrombosis of cerebral vessels. (2)

Other factors which ultimately may cause death in chronic heart failure are merely mentioned and not discussed. The lesser importance in this instance is simply because these less frequently are found as etiological factors in failing hearts. Certain drugs, allied in action

to the hormones adrenalin and pituitrin, in long continued usage cause failure of the circulation. Authorities differ widely as to the effect of nicotine on the heart as it is absorbed by tobacco users. Most authorities admit that the use of tobacco may precipitate anginal attacks but many clinicians believe that the problem of nicotine absorption is mainly one of individual susceptibility. (17) The question of smoking in the problem of chronic heart failure resolves itself into one of the determination of the extent to which a previously normal cardio vascular system may be made more susceptible to other predisposing causes. Nevertheless, instances will arise when the physician must not be too dogmatic in depriving his patient of such comfort as he may secure from smoking; Mackenzie pointed out that a single cigar might do more good in certain cases than all the drug sedatives at his command. (26) Alcohol used in moderation is not necessarily contraindicated in a person with a failing heart. (17)

A local anemia of the medulla has been offered as an explanation for hypertension. (40)

Bordley and Baker in 1926 examined the medulla at the site of the vasomotor center in 24 cases. In every case of increased blood pressure they found definite changes in the arterioles. Gull amd Sutton had already described exactly the same condition in the small vessels of the pia mater. In spite of all the literature written on the subject it cannot yet be definitely stated as to whether hypertension (and resultant cardiac failure) or the arteriolar changes came first.

Chronic focal infection resulting in chronic toxemia can ultimately destroy the efficiency of the heart. (26) Uterine fibromata is at times mentioned as the underlying cause of cardiac decompensation; so too is the hypertension found developing as a part of the menopausal syndrome. (2) Thyrotoxicosis is definitely a predisposing factor as are certain mental states belonging to the minor psychoses. Overweight or obesity is frequently cited as a predisposing factor in hypertension and ultimate decompensation. However, overweight in itself is not in any sense a cause of hypertension, yet it is

interesting that the overweight persons in any large series show a higher ratio of hypertensive cases. (40)

Pericardial effusion furnishes the classical example of circulatory failure due to interference with the diastolic filling of the heart. (10) However, heart failure is so often absent with even large effusions that the presence of fluid in the pericardial cavity does not seriously embarrass the heart. When the fluid accumulates rapidly as in hemorrhage due to trauma, rupture of aneurysm, scurvy or other hemorrhagic diathesis, fatal tamponade of the heart may occur with relatively small effusions. If the fluid accumulates slowly, as in most instances of rheumatic, tuberculosis or uremic pericarditis, much larger effusions are necessary to produce circulatory failure. (10) Adhesive pericarditis may also seriously embarrass the heart. (10)

There is another underlying cause of heart failure which merits a little more discussion, namely pregnancy. (34) It may be questioned whether or not pregnancy is a chronic affair

and that perhaps it should be included as a precipitating factor. Three types of heart disease are caused by pregnancy in which decompensation is apt to occur. It is easier and more appropriate to discuss these related diseases separately remembering that they are results of a normal pregnancy in a woman who when not pregnant has no heart symptoms.

Gestatory heart disease might be a good name for the purely physiological condition caused by the increased cardiac work. In equation form, cardiac work is equal to the cardiac output times the mean blood pressure. Since there is no appreciable drop in blood pressure during pregnancy and the uteris and fetus require an increased blood supply it is obvious that the cardiac work is increased. (34)

Certain changes dependent upon the toxemia and nephritis of pregnancy cause cardiac failure. As one would expect the cardiac changes which accompany the toxic states vary with their etiology. A certain explosive character is conferred by pregnancy in these individuals, particularly upon their hypertensive state. (34)

Cardiac failure is sometimes seen post partum. Although the heart has been adequate throughout pregnancy, fourteen to twenty-five days following delivery, congestive heart failure develops, first on the left and then on the right side. It has been suggested that this disorder might be due to changes in endocrine function or a nutritional deficiency. However the symptoms occur at the time when the patient resumes full exercise and so places an additional strain upon the heart. (34)

Still another cause of cardiac failure which may develop insiduously or precipitously is ventricular aneurysm. (6) Five etiological varieties have been classified. The arteriosclerotic aneurysm is the type following coronary occlussion and myocardial infarction. Rheumatic aneurysm results from necrosis of the myocardiam by the Aschoff body. In syphilitic aneurysm there is first the development of a myocardial gumma. The mycotic aneurysm is secondary to bacterial endocarditis. And lastly, there is of course the traumatic aneurysm. Steinberg (1914) states that 84.8% of myocardial

aneurysm is due to coronary occlusion. (6)

As stated before it would be foolish to list all the specific causes of cardiac failure. Analysis would show that by far the majority of cases will fall under the three major headings listed in a former paragraph. (Refer to Boyd p. 17)

2. Precipitating causes

The various disease processes which constitute the underlying causes of cardiac disease are usually of progressive nature and in time will produce cardiac failure regardless of therapy. However, the rate of progression of the underlying disease is usually slow and in the majority of patients cardiac failure does not supervene until some added factor precipitates it. Most of such precipitating causes can be avoided or treated with some success. These are therefore of great practical importance and will be considered in some detail. (14)

When precipitating factors are mentioned by various authors it is quite confusing because many factors listed as underlying causes by one author are considered as precipitating

by another. It really makes little difference how they are classified; if they can be prevented their importance in producing heart failure should be recognized and guarded against. For example, pregnancy has been considered as an underlying cause in those patients who ordinarily have no cardiac pathology. Yet in a woman with known valvular disease, pregnancy is undoubtedly a precipitating factor. The same might be said of chronic toxemia and infection.

From Harrison's book, Failure of the Circulation, underlying causes have already been mentioned. He also mentions various causes of heart failure which he considers precipitating. Infection is considered by and large most important of the conditions which superimposed on a potentially weak heart will precipitate failure. (14) This is particularly true of respiratory infections not only because of the specific pathology but also because of the great number of upper respiratory infections. Also, because cough, one of the common symptoms, is in itself a considerable muscular effort. Excessive exertion is another precipitating factor. Potential cardiac patients should be warned against over-

taxing themselves. Only within very recent times has the importance of sexual excitement as a precipitating factor in anginal attacks and resulting heart failure been recognized. (17) Brooks points out that sexual excitement may be divided into two phases: (a) the actual physical exertion entering into the act of coitus and (b) the role played by sex endocrines.

Pregnancy also can be considered a precipitating factor in a patient with potential cardiac disease. (Refer to p. 30) Not only does labor severely tax the already overburdened muscle but during the entire course of pregnancy the output of the heart is increased. Sudden increase in weight may precipitate cardiac failure. The increased amount of fat requires a greater oxygen consumption and at the same time the heavy fatty mesentery pushing the diaphragm higher cuts down on the vital capacity of the lungs. (14) Anemia, although it places no more work on the heart, causes failure by the deficient blood circulating

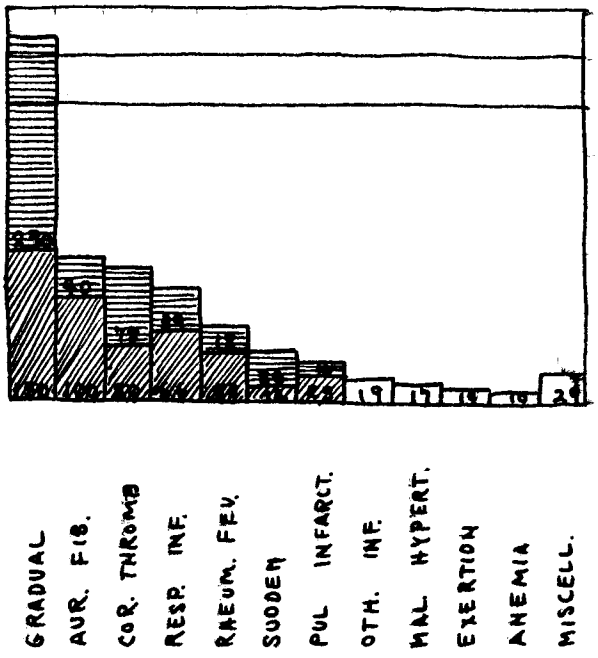
through the myocardial fibers. Emotional upsets and mental strains can raise the blood pressure and since the cardiac output remains the same consequently increase the cardiac load. (i.e. Cardiac load equals cardiac output times mean blood pressure.) (Refer to p. 30)

Changes in rate and rhythm of the heart may be brought on by various factors such as those mentioned. Sometimes an arrhythmia can develop without apparent explanation. The same is true of a paroxysmal tachycardia. The obvious explanation is the upset of the threshold to stimulation of auricular or ventricular musculature or a shortening of the refractory period of the nervous tissue of the intrinsic cardiac mechanism. Tachycardia without arrhythmia can precipitate congestive heart failure. (14) Increase in heart rate when accompanied by diminution in pulse pressure (i.e. when the cardiac output is not increased) tends to reduce coronary flow. Any arrhythmia in which there is a pulse deficit will tend to diminish the coronary flow because during the ineffective systoles

the increased intramural pressure will not be counterbalanced by a rise in aortic pressure. However, when tachycardia is accompanied by increase in pulse pressure the blood flow through the coronary arteries is augmented.

With the same one thousand cases studied before in Massachusetts General Hospital, Bayer, Leach, and White have tabulated the various precipitating factors in those patients exhibiting congestive heart failure. (2) The following graph excellently depicts the various causes responsible. Here again the entire column represents the number for the total series, the horizontal area represents the number in the private group and the cross-hatched area the number in the ward group. In the smaller columns there was no difference and the marking so indicates. The miscellaneous group includes: thyrotoxicosis--seven, operations--five, paroxysmal tachycardia--four, gall bladder colic--three, pregnancy--two, (there is too small a lying in service in the Massachusetts General to consider this number significant), asthma--two, pulmonary malignancy--two, trauma--two,

emotion--one, excess fluid administered--one.



Heading the list of precipitating causes of failure is the group labeled gradual onset. Some of these undoubtedly had a specific precipitating cause which remains hidden because of lack of detailed history. There unquestionably remains a rather large group in whom failure begins insiduously with no assignable cause. These cases are to be attributed simply to the accumulating stress and strain of advancing years. (2)

Because auricular fibrillation proved to be such an important precipitating factor, an effort

was made to discover the cause of the fibrillation. In the series of one thousand cases studied, there were one hundred forty in which the onset of auricular fibrillation seemed to play an important role as the cause of failure. Of these twenty were associated with rheumatic fever, fifteen with respiratory infections, six with coronary thrombosis, four with pulmonary infarction, three with thyrotoxicosis, and one each with operation, trauma, pregnancy, exertion, and acute pyelitis. In eighty-seven instances the auricular fibrillation seemed to rise without definite cause.

There are those who believe that heart failure is the cause of auricular fibrillation, (Luten, 1936) rather than the result. (2) The authors who outlined the above graph, in view of the fact that such an arrhythmia occurs in normal or abnormal hearts without signs of failure, believe that auricular fibrillation can and often does induce heart failure rather than result from it. (In the above graph the cases with auricular fibrillation are listed twice, once under the heading of auricular fibrillation

and also under what other pathology has been considered as the cause of fibrillation.)

The incidence of respiratory infection as a precipitating factor is difficult to evaluate. In this tabulation, cough alone was not considered to be a true respiratory infection unless accompanied by purulent or mucopurulent sputum. Consequently a fair number of true respiratory infections were eliminated so that its importance as a precipitating factor is minimized. It is possible that in a few patients unrecognized rheumatic fever precipitated by an antecedent respiratory infection may have been the more important exciting cause of heart failure. (Refer to underlying causes p. 23)

The remainder of the graph seems self-explanatory. The causes listed as sudden are not included in the discussion in this paper. They would include those various causes such as toxins and trauma which would suddenly increase the load on the heart enough to cause failure without the underlying cardiac pathology in which eventual congestive heart failure would be inevitable.

3. Summary

Before in this paper the idea of the load of the heart has been clearly postulated as being the resistance against which the heart is working as well as the pressure head determining its filling. (Refer to definition p. 12) It is important to clearly understand the definition since depending upon its load the work of the heart may be either increased or decreased and may be either adequate or inadequate for the needs of the body. In fact the contractile power of the heart must be interpreted in terms of the load. Thus, the fact that any condition, mental or physical, or any pathological lesion which forces the heart to do more than its expected amount of work, must be considered a cause, either underlying or precipitating, of heart failure.

The added load that the heart is competent to meet is defined as the cardiac reserve. (Refer back to definition p. 13) Therefore, in cardiac failure when the load on the cardiac muscle is increased the amount of the reserve also determines whether or not the heart will

fail. (18) The question of cardiac failure then is boiled down to the simple statement, that in such a condition as failure, the heart is unable to maintain adequate circulation by means of its reserve energy.

CHRONIC HEART FAILURE

V. Theory

1. Functional theory

Cardiology has long been the battleground of speculative and conflicting doctrines. The terminology based upon some of these doctrines has unfortunately outlived their truth and certain expressions have become meaningless in the sense in which they are still widely used. These descrepancies between theory and fact give rise to much confusion. (33)

Before entering into a lengthy discussion of the various conflicting theories of cardiac failure it appears to be advantageous to state the theory which seems most logical. It should be emphatically stated that in the end analysis failure of the heart is functional in nature. (26) Such a statement becomes more impressive when fatal heart failure is found associated with no perceptible lesions of the structures of the heart. The fact that the heart may fail to do its work when there is an abundance of seemingly healthy muscle forces the conclusion that the failure is really the outcome of the

impairment of the function of the heart muscle itself. (26)

It seems quite obvious that the inability to maintain circulation can occur only because of two fundamental upsets in the circulatory mechanism, namely: (a) an inability to get blood into the circulatory tree or (b) an inability to get blood from it. The first condition is called Forward Failure and hinges upon the cardiac output or minute volume of the heart. The second is termed Backward Failure and revolves about the idea of residual blood in the ventricular chambers of the heart. However, in the discussion there comes a certain point in both theories when the statement must be made that the ventricle fails to put out the normal quota of blood per beat. Why this occurs is the answer to cardiac failure and again it must be repeated that the answer is in functional failure of the myocardiam. (26)

Whether or not a name is given to the functional explanation of heart failure is a matter of small importance. It seems unlikely that the terms backward or forward failure can either one

be all inclusive or necessarily exclusive of the opposite theory. (36) Without doubt symptoms of cardiac insufficiency are due both to a decreased output and to an increase in venous pressure explained on the basis of the backward failure. (27) At any rate the circulatory time is greatly increased thus allowing transudates to form in the tissues. (1) Whether the stasis is caused by decreased arterial pressure or an increased venous pressure makes little difference. Both are present in heart failure.

It has been shown by several investigators that in patients with congestive heart failure there is a very small increase in cardiac output with exercise. (28) These same investigators have proven that in normal subjects (i.e. with no cardiac pathology) there is a considerable increase in cardiac output with exercise and with patients who have compensated for difficulty in maintaining circulation there is a moderate increase in output with exercise. (28) The conclusion must be drawn that the relative inability to increase the cardiac output may

have an important bearing on the pathogenesis of congestive heart failure. Hence the question seems intimately associated with the cardiac reserve. The functional efficiency of the heart therefore depends upon the amount of this reserve force and heart failure invariably starts by exhaustion of the cardiac reserve. (26)

A descrepancy arises when it is proven that there is at times no decrease and in fact may be an increase in cardiac output. (27) From the various observations made it is evident that the majority of patients with congestive heart failure have subnormal cardiac output. however, if it is appreciated that certain factors known to elevate the output of the normal heart are present during decompensation the seeming paradox of normal and even higher than normal values for cardiac output during failure can be understood. Even a tired horse will walk faster if whipped. These factors are: (a) the exercise associated with hyperpnea, (b) elevated venous pressure, (c) increased metabolism, (d) anxiety and apprehension, (e) low grade fever. These conditions prevent the attainment of a

truly basal state by the decompensated individual. (27)

For these reasons it seems logical to believe that disease of the myocardiam diminishing systolic discharge of the ventricles (invariably decreased in relation to the accelerated physiological activities of the decompensated individual) is the initial factor responsible for cardiac failure. (27) Such decrease in cardiac output necessarily produces increased venous pressure and diminished forward flow simultaneously. Consequently a vicious cycle is created and the manifestations of heart failure result.

2. Back Pressure theory

The common theory of chronic heart failure is called the Back Pressure theory. It is the view most commonly held. The term was originally used to convey the idea that regurgitation of blood through the valves of the heart is the cause of the overfilling of the venous side of the circulation and that tricuspid regurgitation is necessary before engorgement can develop. (33) This overfilling (which is implied in the

term congestive heart failure) is now recognized as occurring quite apart from valvular damage or incompetence. (14) (Refer to p. 39)

Certain facts seem to confirm this view. In aortic regurgitation or stenosis the left ventricle early enlarges. In mitral stenosis there is no change in the size of the left ventricle but the right side of the heart is greatly enlarged.

Harrison in his book, Failure of the Circulation, staunchly upholds a modification of this hypothesis which he chooses to call the theory of Backward Failure. (14) Briefly, the etiology is always cardiac in origin and the chief characteristic is an abnormal fullness of certain portions of the vascular bed. There are two main types, failure of the left side of the heart in which there is congestion in the pulmonary capillary bed, and failure of the right side in which there is congestion in the systemic veins. Failure of right and left ventricles occurs together.

The salient features of the backward failure

hypothesis are briefly enumerated and easy to understand. First, there is a rise in pressure back of the failing ventricle of the heart with the resultant symptoms developing in the organs which feed the blood to the failing chamber. The essential phenomenum is a change or alteration in intracapillary pressure. Thus, with overwork the chamber of the heart dialates in turn causing a rise in pressure in the veins which supply the affected side of the heart. Such a condition is evidenced by extreme congestion. (14)

In valvular disease, for example mitral stenosis, there is first a hypertrophy and dilatation of the left auricle followed by pulmonary congestion with a resultant hypertrophy and dilatation of the right ventricle. Even though the tricuspid valve remains competent a relative insufficiency of the orifice occurs and a rise in the pressure of the right auricle and great veins occurs manifested by the symptoms recognized as congestive heart failure. In hypertensive disease the mechanism is similar but not identical because pulmonary congestion does not occur until

the left ventricle fails. Therefore in a left sided atrio-ventricular lesion pulmonary congestion is an early symptom which gradually develops to severe dyspnea. In hypertension, pulmonary congestion appears late in the disease but develops with great suddenness. The dyspnea is not to be explained entirely on the basis of pulmonary congestion but also due to the increased cerebrospinal fluid pressure which occurs simultaneously with an increased venous pressure. (39) (Refer to symptoms p. 76) However the greater part of the dyspnea is due to reflexes arising from the engorged lungs and stimulating the respiratory center. (27)

Other authorities have objected to the Backward Pressure theory on the ground that this hypothesis necessarily involves the assumption of regurgitation through the atrio-ventricular valves. (14) However, it is possible for the auricular pressure to arise as the accumulation of the residual blood in the ventricle even though the atrio-ventricular valves function normally. The collection of residual blood is

explained by a fatigue of the ventricular blood. Physiologically the ventricle will dialate in order to increase its power of contraction and again bring the cardiac output up to par. However the ventricle now contains a certain amount of residual blood.

Nevertheless in order to get the residual blood into the ventricle it is necessary that it should first fail in its normal output. Consequently the precipitating mechanism for increased back pressure is a decrease in cardiac output, for a short time at least.

3. Forward Failure

In contrast to the theory of Backward Failure, Harrison although disapprovingly has stated a theory which he calls Forward Failure. (14) The etiology can either be cardiac or peripheral in origin and the chief characteristic is an abnormal emptiness of the vascular bed. (14) The peripheral etiology of Forward Failure is one of three causes, namely: (a) neurogenic (primary shock), (b) hematogenic (i.e. secondary shock), (c) vasogenic. The etiology that is cardiac in origin is more complex. Ventricular

fibrillation may cause Forward Failure. Cardiac syncope, the second cause of Forward Failure which is due to cardiac causes can be either neurogenic or cardiogenic in origin. Neurogenic cardiac syncope is due to either psychogenic or reflex action, (i.e. carotid sinus, vago vagal or oculo cardiac factors). Cardiogenic cardiac syncope would be either a Stokes-Adams syndrome or an aortic valve lesion. A third factor in the etiology of Forward Failure due to cardiac causes is cardiac collapse produced by marked tachycardia either, auricular fibrillation, auricular flutter, auricular tachycardia, or ventricular tachycardia. Cardiac collapse may also be caused by acute severe myocardial impairment due to either coronary thrombosis or diptheritic myocarditis. Mechanical hindrance to the heart may also cause cardiac collapse. Such hindrance may be brought about by cardiac tamponade, (Refer to p. 29) massive pulmonary embolism or a ball valve thrombus obstructing the mitral orifice.

The Forward Failure theory presupposes a

decreased output of blood from the left ventricle. Exponents of this theory explain cardiac dyspnea in a unique manner. The decreased cardiac output results in a state of relative oxygen lack and carbon dioxide excess in the cells of the medulla. Hence the respiratory center is stimulated resulting in cardiac dyspnea. (26) Mackenzie is one of the staunchest supporters of this hypothesis. He would explain the various symptoms of cardiac failure as being due to the loss of driving power of the left ventricle. Most of the older men including Sir William Osler tend towards the acceptance of this theory. (30) Other recent investigators also have demonstrated that the functional capacity of the heart as a pump is markedly decreased in congestive heart failure. Smith definitely states that: "Failure of the circulation is usually due to failing systolic power and output. (33) Lewis, Blumgart, and others ascribe manifestations of congestive heart failure to inadequate cardiac output in proportion to the metabolic needs of the body. (33)

The opponents of the Forward Failure hypothesis argue that acceptance of such a theory in-

volves the following assumptions: (a) the cardiac output is subnormal in persons with congestive heart failure, (b) improvement in the clinical state is necessarily associated with increase in the cardiac output, (c) procedures which cause diminution in the cardiac output in proportion to the metabolic needs necessarily result in an increase in the severity of the symptoms. Harrison is one of those who disprove this theory. He states, and cites adequate facts that, although it is admitted the blood volume output per minute is usually subnormal in patients with cardiac disease, there are occasionally individuals with no symptoms of cardiac disorder who have minute cardiac output as low or lower than patients with congestive heart failure. (Refer to p. 45) In many patients a clinical improvement with disappearance of congestive manifestations may be associated with an increase or with a decrease in cardiac output or with no change whatsoever. The various theraputic measures which produce benefits in persons with congestive heart failure do not ordinarily cause an increase in cardiac output. (14)

Harrison further concludes that persons with congestive heart failure may and often do have decreased cardiac output but this is not the essential cause of their symptoms. The clinical picture produced by conditions which head to marked decrease in cardiac output is not that of congestive failure but that of shock or collapse. Such individuals have no edema, lie flat in bed without discomfort and suffer from weakness rather than dyspnea.

Again these statements can be objected to upon the grounds that, although it is true in primary or secondary shock the cardiac output is low and no manifestations of congestive failure are noted, in shock, unlike cardiac decompensation, the volume of the circulating blood is low and the venous pressure subnormal. Consequently the conditions are not comparable as in one instance the heart pumps away the blood brought to it and in the other condition it fails to do so. (10)

4. Conclusion

It does not seem necessary to line up definitely on the side of either theory. The real question of cause of failure seems to be in the

fatigue of the ventricular muscle. The question to answer is why does the fatigue of the ventricle with decreased output occur before there is compensatory dilatation and increased contractility. The answer to this question can be explained satisfactorily only by the functional theory of cardiac failure. Exponents of both theories apparently are making a mountain out of a mole hill. In the end analysis both make concessions admitting fundamental principles which are salient points in both explanations. Whether or not the arterial pressure lessens causing stasis in the capillaries or whether or not the venous pressure rises making the driving power of the ventricle inefficient is a matter of small import. After stasis occurs, which is relatively late in failure, backward pressure does modify the circulation and produce definite symptoms. While back pressure is the mechanism principly responsible for the symptoms of congestive heart failure the forward failure and cardiac output cannot be completely separated from the Back Pressure theory. Both develop simultaneously and operate conjointly to produce the clinical

manifestations of cardiac decompensation. (22)

CHRONIC HEART FAILURE

VI. Pathology

1. Gross and microscopic

It is practically a hopeless matter in the present state of medical knowledge to correlate the various forms of heart failure with gross changes found post mortem either with the naked eye or with the microscope. (10) Even when the patient has the well defined symptoms of chronic heart failure such as orthopnea, dyspnea, edema, enlarged pulsating liver, or those shown by attacks of angina pectoris, the pathological changes are so varied that it is impossible to attribute the symptoms with assurance to any given pathological condition. (26)

Of course when failure is due to valvular defects the pathologist can demonstrate heart valves which fail to close properly. In coronary disease, due to any or all of the causes mentioned, the end result is weakening of the myocardiam, i.e. temporary or permanent ischemia with destruction of muscle fibers and replacement by fibrotic tissue. One of three things may happen when a large branch of a coronary

artery is occluded. The patient may die immediately, (coronary occlusion being the only thing which will kill a person instantly) he may linger a few days or he may survive at least for a time. With aneurysm the pathology is obvious. Necrobiotic changes may be widespread in various toxemic conditions of the heart.

The statement has been made before that the failure of the heart in the end analysis is a functional affair. (Refer to p. 42) Many of such hearts present so few pathological features that no distinction can be drawn between a heart with abundance of healthy muscle that just stopped functioning properly and one that continued the task of circulating the blood until some undercurrent infection or disease struck the individual down through another organ.

The assumption is generally made that it is finally anoxemia of the heart muscle which causes fatigue and inability to maintain circulation. (14) There is often observed microscopically a condition of the heart muscle seen when an oxygen deficiency has persisted for some time as in coronary disease and long standing anemia. This

condition is known as "tigering" and it naturally occurs in those portions of the heart where the blood supply is more likely to be interfered with because of higher intramural pressure. Such "tigering", which is really a form of fatty degeneration, is usually more outspoken in the papillary muscles and subendocardial fibers of the left ventricle. (14)

Atrophy of the heart is sometimes considered significant in the origin of myocardial insufficiency that often terminates protracted cachexis. Such a condition, termed brown atrophy, also occurs in old age and is seen in elderly patients at post mortem examination although no evidences of failure were present during life. (10)

Deposition of albuminous granules in the sarcoplasm producing cloudy swelling is often to be seen when bronchopneumonia or other infections or intoxications accompany the last stages of heart failure. (10) However, cloudy swelling is often found in the absence of failure and a heart which has undergone considerable post mortem autolysis often closely simulates

cloudy swelling. When chronic heart failure is present, it is not rare to find vacuoles filled with fluid in the sarcoplasm. Usually they are not abundant and they may be found accompanying inflammatory edema of the heart. (10) Fragmentation (longitudinal splitting) and segmentation (transverse splitting) of the myocardial fibers is sometimes seen at post mortem examination. The phenomenum is rare in the young and common in the aged. The papillary muscles of the left ventricle are the site of predilection and the course of the fracture is generally oblique. The nuclei are not involved. Moenckeberg suggests that fragmentation may be the anatomical expression of terminal ventricular fibrillation. (10)

Cases of extreme softening of the myocardiam have been reported in which the entire muscle mass may become so soft as to fall over the hand like a mushroom when the heart is held by the base with the apex above. (26) Such a heart has been seen when the myocardial fibers were almost entirely replaced by fatty tissues.

Fatty degeneration is a retrogressive condi-

tion in which fat droplets are found in myocardial sarcoplasm. It is usually secondary but has been noted to occur without any demonstrable cause. The condition is common especially in minor degrees. Most authorities do not believe that fatty degeneration produces insufficiency. MacCallum states that fatty degeneration seemed to have very little detrimental effect upon the function of the heart. (25) Herrmann concluded that fatty degeneration occurring during infectious diseases was rarely sufficient to produce failure. (16) However, certain conditions have been reported in which myocardial insufficiency was certainly caused by excessive fatty degeneration of the heart muscle. (12)

2. Physiology

Remembering that the heart fails because of the inadequacy of the compensatory mechanisms to maintain adequate circulation, it would be well to recall the three means of compensation, namely: (a) tachycardia, (b) hypertrophy, (c) dilitation. (2) There is no set rule as to the ratio or order in which these mechanisms occur. Suffice it to say, that in long continued insult

to the heart, all three means of maintaining circulation are utilized in varying degrees.

The question naturally arises, to what extent can cardiac compensation be carried? Obviously there are limits which when reached, compensatory measures fail. With this thought in mind, the term cardiac decompensation becomes an admirable synonym for chronic heart failure. (Refer to definition p. 9)

When the rate of the heart becomes too rapid there is incomplete filling of the ventricles and the stroke volume of the heart is greatly reduced. With each beat of the heart, too small an amount of blood is expelled, and the heart is no more efficient than when beating at its normal rate. (2)

Hypertrophy is defined as the increase in the muscle mass. However the coronary arteries do not increase in anastomasis and extent as does the cardiac muscle. Hence, there is inadequate nutrition to the individual fibers. (2)

By dilitation is meant the increase in length of the heart muscle fibers. When there is too great stretching the fibers become ineffici-

ent in elastic recoil. Tonicity is that term applied to that function of the heart muscle which keeps the heart during diastoli in a state of slight contraction. (15) In dilitation this function is depressed. Such distortion of the size and shape of the heart secondarily involves the valves, superimposing valvular disease upon an already overburdened heart.

In general, cardiac decompensation can be either right or left ventricular or both. The end result is the same and the heart must compensate in some manner in order to maintain circulation. It has been pointed out that the three main underlying causes of heart failure are valvular, coronary, and hypertensive disease. It is unnecessary to enter into a detailed discussion of the pathological physiology of failure valve by valve amd branch by branch of the coronary circulation. Such a discussion would entail anatomy and physiology beyond the scope of this paper. However, a few of the salient facts might be reiterated.

In valvular disease the ventricle proximal to the diseased valve, whether the valve be sten-

otic or regurgitant, is the chamber affected of ultimate importance. (2) In coronary disease the ventricle deprived of its blood supply is the chamber which will undergo hypertrophic and arrhythmic changes. In hypertensive disease, the ventricle which is forced to work against the increased load whether the resistance be in the peripheral or pulmonary circulation is the one which will immediately fail. The word immediately is used in this manner because very often failure of the ventricles follow each other in short order.

In the chapter on etiology of heart failure, the various etiology of decompensation was implied. In the preceding chapter, the Back Pressure theory of chronic heart failure was discussed. Needless to say, this theory has been built entirely upon the physiological explanation. For illustrative purposes, those patients predisposed to mitral valve lesions would be expected to suffer from right ventricular failure before left. On the other hand a person with aortic disease would first suffer from left ventricular failure.

In like manner, since the coronary artery

most often thrombosed is the anterior descending branch of the left coronary and since the artery supplies mainly the left ventricle, left ventricular failure would be expected in those patients predisposed to coronary occlusion. This same class of patients is also prone to hypertension, in which the brunt of the load is thrown on the left ventricle.

Rheumatic fever particularly, as well as pulmonary infections in general, are suffered in the main by a type of patient subjected to improper hygenic conditions caused by poverty. Thus, in a class of patients from the lower social level one would expect more of the right ventricular heart failure. Such a statement must not be taken too literally. The syphilitic aortitis most commonly seen in the negro would cause failure of the left ventricle.

In an attempt to correlate social incidence with right and left ventricular failure a general statement can be made well substantiated by carefully planned statistics in a series of ward and private patients at the Massachusetts General Hospital. In a class of patients from

the lower social levels one would expect more of the right ventricular heart failure. From the higher social levels including the patients with coronary and hypertensive heart disease one would expect left ventricular failure. (2)

3. Chemistry and metabolic

The chemical and metabolic pathology of the failing heart proves of interest because of its prophylactic and theraputic possibilities. As would be expected, fatigued hearts are poorer in those substances which are normally more abundant in the tissue cells and richer in those elements which are normally more abundant in tissue fluids. (26) This general statement could be made of any muscle either smooth or skeletal.

The greater activity of the muscle is probably associated with potassium loss. It has not been conclusively demonstrated as yet that overwork of cardiac muscle causes loss of potassium but this seems likely in view of the greater acidity of the muscle mass. Potassium is ordinarily regarded as a powerful cardiac depressant, along with the other single valency ion sodium, but experimental evidence suggests that potassium

under certain conditions is a powerful recuperative agent. (26)

In persons dying in congestive failure, there is always found a slight increase in the water content of the heart. This increase in intramuscular edema may help to explain the loss of the important ion potassium.

The creatine content of the heart muscle is diminished in subject with cardiac failure. A striking decrease in creatine was found in the infarcted areas of the myocardiam. Since it is well established that creatine compounds play an important role in muscular contraction, the loss of creatine may favor the developement of myocardial fatigue and failure. (26)

This metabolic and chemical physiology and pathology does not offer much as yet to a clinician but the pertinent statement might be made that this is fruitful field for research.

CHRONIC HEART FAILURE

VII. Symptoms

1. Subjective versus objective symptoms

In the discussion of the pathology of cardiac failure it has been pointed out that no one condition can be considered typical. Neither can any symptoms be set up arbitrarily and it be said of them, these represent cardiac failure in its entirety. The statement has been made that in the end analysis cardiac failure is functional in nature. (Refer to theory p. 42) Mackenzie also explains the same idea by saying that cardiac failure invariably begins by exhaustion of the reserve force. (26) Evidences of exhaustion are of two kinds, those due to the organ itself and those due to other organs on account of the inefficient circulation. Thus it is seen that the physician has to consider in every case the efficiency of the heart and this can only be done by testing its functional capacity and the functional capacity can only be appreciated by understanding how the heart responds to effort. Mackenzie further states emphatically: "Here

we get the only method of estimating the significance of any abnormal sign." (26) The significance of symptoms is carefully weighed after watching how the patient meets the exigencies of life.

In a discussion of any symptoms of cardiac failure an axiom should be stated, that: "Any sign which is indicative of heart failure is always accompanied by other symptoms." The converse is equally true, that: "Any sign unaccompanied by abnormal phenomena may be taken to be of little significance so far as the functional efficiency of the heart is concerned."

The meaning of the term symptom embraces all the manifestations due to or exhibited by the organs of circulation. The manifestations which are revealed by physical examination rarely yield the information which it is so essential for the examining physician to acquire. No one sign nor combination of signs is significant of cardiac failure. (26) A heart may be inefficient and yet perhaps nothing can be detected by physical examination except feeble sounds and a low tension pulse. (30) The presence

of murmurs or irregularities or increase in the size of the heart may reveal the nature of some disease process but it does not tell what bearing it may have on the heart's power to maintain an efficient circulation. More important than the signs disclosed by physical exam are those revealed by the patient's sensations, for in the first instance exhaustion of the reserve force is made evident only by subjective sensations. (26)

2. Left and right ventricular failure

The clinical cardiac manifestation occurring with chronic heart failure may be protean, seldom conforming to a constant pattern. For purposes of convenience, symptoms and signs of failure may be grouped into those occurring with left ventricular failure and those occurring with right ventricular failure. (29)

The symptoms of left sided cardiac failure differ from those of the right side and in each may be distinguished a number of types which however merge gradually the one into the other. (30) Failure of the left ventricle is seen in its severest forms in the abrupt death stroke

of angina pectoris, or in the fainting and convulsive attacks of Adams-Stokes disease. The milder degrees show themselves in an inability to do much mental work or an inability to take much exercise without a sense of great fatigue. In severe left ventricular failure, cardiac asthma or paroxysmal dyspnea with edema of the lungs is a frequent sign alone or with Cheyne-Stokes respiration. The cardiac rhythm of this type of failure may be of the gallop type or any of the arrhthmias may be present. Pulmonary congestion is frequent and vital capacity may be reduced to 60% of normal while circulation tests reveal a prolonged decholin time and a normal ether time.

Sudden and slow types are also seen in failure of the right side of the heart. Subjected to a slight strain, great hyperpnea and distress may come on, and one form of cardiac dyspnea which attacks the patient at night is of this nature. Chronically there is an increasing inability to undergo slight extra exertion and there is usually some edema of the feet, especially at night if the patient is on his feet most

of the day. Generally with the edema there is a rapid gain in weight. The liver is as a rule enlarged and may be tender and pulsating. Jaundice is not uncommon. The finding of albumin and erythrocytes in the urine is indicative of renal congestion. Special tests of the circulation reveal an increased venous pressure together with a prolongation of the ether time (prolonged ether and decholine time indicate a failure of both right and left ventricles). (29)

3. Early symptoms

The earliest symptom is shown by that organ which first experiences a deficiency in its blood supply. The variableness of the symptoms of heart failure is due to the fact that the different organs may be rendered inefficient in different individuals by the deficient blood supply. Moreover such evidence is afforded in the early stages only when the heart is forced to exercise itself at the full extent of its power. Later when a full blown congestive failure is evident the same symptoms are present in exaggerated degree even when the heart is at rest.

The first symptoms of inefficiency may arise from the heart itself due to its failure to supply blood to its muscular or to its nervous mechanism. These symptoms are varied in character, the most common being a disagreeable consciousness of the heart's action. In health this is shown usually by violence or rapidity in its action, in disease by sensations due to abnormal action as fluttering sensations produced by its irregular action. When dilatation occurs there are gallop rhythms, shortening of the long pause and a systolic murmur at the apex. (9) Anginal attacks are frequent due to the anoxemia of the heart muscle. As a result of the myocardial anoxemia, disturbances in the metabolism of the muscle occur. Lactic acid and some pain producing substances develop. These in turn irritate afferent fibers within the myocardiam. (24) In later stages of insufficiency the angina pectoris disappears. This fact is difficult to correlate with the theory of pain due to anoxemia of the heart muscle because with late congestive failure the

coronary flow is still further diminished. There is perhaps some correlation of angina with cardiac tone. (24)

Most authorities agree that one of the earliest if not the earliest symptom resulting from failure of circulation that is cardiac in origin is the symptom of breathlessness. Mackensie explains that this sensation is due to oxygen lack in the cells of the medulla about the respiratory center. (26) (Refer to theory p. 52) The conclusion seems logical, it being proven beyond doubt by various physiologists that the respiratory center is the most sensitive tissue to a relative oxygen lack and must consequently compensate by overbreathing. On the other hand, the early dyspnea is explained by those who believe in the Backward Failure of the heart as due to the rise in cerebrospinal fluid pressure. (39) (Refer to theory p. 49) With an increased venous pressure the intrathecal pressure rises rapidly and in many cases when the cerebro-spinal pressure falls to normal levels the dyspnea is much relieved. (39)

Manifestations of cerebral anoxemia are early elicited. Weariness following prolonged mental effort and memory impairment particularly for recent events are early symptoms. Giddiness is a symptom frequently present and usually of a very transient nature. When the ventricle pauses briefly the patient speedily recovers from slight giddiness, but if the ventricular pause is longer a loss of consciousness follows. If the standstill be still more prolonged, convulsive movements of skeletal muscles are produced.

Various forms of dyspnea may arise from an inefficient supply of blood to the brain. Not withstanding that the exact conditions which induce Cheyne-Stokes respiration are still obscure, it is so frequently present with evidence of circulatory enfeeblement that it may be a symptom of inefficient blood supply to the respiratory center. (26) Notably is this the case in advance degenerative changes in the heart muscle, but its frequent sudden occurrence in cases of auricular flutter and complete heart block when the circulation is impaired seems to

suggest that diminution of the heart's output is one of the causes of its onset. (26) Harrison takes exception to this forward theory of heart failure, (Refer to theory p. 49) and would explain dyspnea as due to congestion and stasis in the pulmonary capillaries. Berger (1936) dismisses dyspnea which he called the earliest symptom as being due to anoxemia.

4. Late symptoms

Without attempting to enter into a detailed tabulation of the multitude of symptoms which may arise from the late stages of failure, it seems advisable to mention the various symptoms as grouped under their special systems.

From the cardio-vascular system many variable symptoms are derived. Pain in the cardiac area or radiating down the arm is common. Palpitation is not so prominent as a late sign. When continuous abnormal rhythms develop other effects appear generally associated with the great increase in rate. Therefore the symptoms become confused, some arising from the excited heart action itself and some due to insufficient

heart circulation. (30) The venous stasis (which directly effects the organs of the various systems) causes a stasis of blood in the capillaries impairing their nutrition and allowing transudation to take place. Thus edema is formed in the tissues. Characteristically cardiac edema is a pitting, dependent edema first appearing in the legs. It may ascend to levels as high as and including the thorax. Fluid may also collect in the serous cavities. (30)

Although signs of cardiac failure are early evident in the respiratory system, late symptoms are also very characteristic. Dyspnea becomes prominent even at rest and orthopnea indicates a definite decrease in the vital capacity of the lungs. Rales are heard first over the lung basis and then over the entire pulmonary area. Hemoptysis is common, the blood tinged sputum being derived from infarcted areas in the lungs. Cough and loss of voice from pressure of the dilated left auricle on the left recurrent lyngeral nerve is an almost constant symptom. Terminally the respirations become of the Cheyne-Stokes type and cynanosis is prominent. (30)

The venous stasis in the alimentary system produces definite symptoms. Passive congestion of the stomach and intestines causes indigestion, anorexia, flatulence, vomiting, either constipation or diarrhea, hemorrhoids, abdominal pain and many other symptoms. The portal stasis of congestive heart failure produces an enlarged tender liver which may be pulsating. Portal cirrhosis is common and in severe cases jaundice is an associated symptom. (30)

Renal stasis is evidenced by characteristic symptoms in the excretory system. The urine is scanty, highly colored and contains both casts and albumin. Decreased functional activity is marked as determined by excretion of phenolsulfopthalein. There is considerable nitrogen retention in the blood. (30)

Sometimes there are well marked changes in the lower extremity, namely, edema, cyanosis, pallor and occasionally purpura. In the terminal changes of congestive failure the edema is a prominent symptom produced even when the patient

is lying at rest in bed.

The symptoms referrable to the central nervous system are those of increasing cerebral anemia. Sleeplessness, impaired mental functions, delusions, melancholia and especially toward the end stupor and rowsiness are most prominently mentioned. The patient may even at times become maniacal. (30)

5. Physical signs

It has been emphasized in preceding paragraphs that the evaluation of the symptoms complained of by the patient are of more importance than the physical signs found. The statement may well be repeated that no one sign or combination of signs is significant of cardiac failure. (26) (Refer to p. 70)

Here again the failure must be subdivided into right and left ventricular failure. Remembering the three fundamental methods of compensation, particularly hypertrophy and dilitation, in chronic insult to the heart when these methods have been utilized, that side of the heart will be most enlarged which suffers from direct

insult. It hardly seems effacable at this point to enter into a description of physical findings. A correlation of the anatomy and physiology of the heart along with the mechanism of failure makes such physical findings obvious.

However, physical examination of the heart may reveal an apex beat which is feeble, outside the nipple line, diffuse and whose maximum intensity is not easily localized. (20) The pulsations may be marked on inspection and cover a very wide area; arterial pulsation in the neck in left ventricular failure may be great; in right heart failure the jugular veins may be very dilated. (11) On percussion, the cardiac area may be much increased to the right or to the left or to both. On auscultation the sounds may be difficult to hear, much feebler than normal; murmurs, usually soft, may be present at both apex and base. (37) Gallop rhythm may be present and is usually significant of approaching exodus. The pulse may show great variations, usually it is feeble with diminished

tension, it may be irregular, intermittent, slow or rapid. (8)

The myocardial lesion is not always proportional to the intensity of the symptoms. As stated before, (refer to pathology p. 57) the patient may present enfeebled irregular action and signs of dilitation with shortness of breath and edema and the post mortem show little or no change in the myocardiam.

Fever is common in patients with heart failure severe enough to require bed rest. (10) When high, some such cause as rheumatic fever, bacterial endocarditis, pneumonia, or infarction is usually revealed as the cause. But some times slight or moderate elevation of the temperature may exist and yet careful examination reveals no other cause than circulatory failure. (19) Such a clinical finding is not surprising. The oxygen consumption and heat production is generally increased in heart failure. At the same time, the mechanisms of heat dissipation, particularly the cutaneous circulation of the blood is definitely cut down.

In accordance with this conception Steele has found that the difference between rectal and surface temperature is greater in heart failure than with unimpaired circulation. (7)

The chest lead electrocardiograph is a valuable diagnostic aid in the diagnosis of myocardial anoxemia. Many times the subjective symptoms and the electrocardiographic findings do not seem correlated but such a fact is accounted for by an explanation of delayed electrocardiographic abnormalities in some cases of myocardial infarction. (23)

There is no one typical picture found in the electrocardiograph that can be displayed as a perfect example of heart failure. The heart can fail from one or several of many causes each one or each combination being a distinct entity in itself. The T wave, which is generally considered as representing the left over potential of the ventricles may be an accurate indication of exhaustion of myocardial reserve. Characteristically, with loss of cardiac reserve, whether it be myogenic or neurogenic in origin, there is a

flattening of the T wave in Lead I, absence of the T wave in Lead II and inversion of the T wave in Lead III. The various arrhythmias and nerve blocks, themselves indicative of a failing heart, have their own characteristic graph. Other diagnostic aids such as the roentgenkymograph and the cardiomensuator may be utilized. Each requires a certain technique and experience which field is not to be included in this paper. (37, 11)

Means of determining the circulation time as a method of diagnosing right or left sided failure has been attempted. Certain chemicals are injected intravenously in the arm and the time of circulation compared with the normal. For example, saccharin is injected and the time until it is tasted by the patient would be compared with the normal. More specifically, if ether were injected and the circulatory time were longer than the normal, it would indicate right ventricular failure. Decholin injected, with a resultant prolonged time, would be indicative of left ventricular failure. If both

ether and decholin time were prolonged both ventricles would be failing simultaneously. (33)

Again it should be emphasized that it is the function of the heart that is important and the response of the heart to effort is the paramount question to determine. (26)

CHRONIC HEART FAILURE

VIII. Treatment

1. Aim of treatment

Unlike acute infectious diseases and many surgical conditions in which a complete cure is expected, the treatment of chronic heart failure is concerned with amelioration of symptoms. Its purpose is to diminish suffering, to prolong life, and to increase the usefulness of the patient as long as possible. Although cures are not to be hoped for, because the underlying structural changes in the heart are for the most part irremediable, proper treatment may render individuals more comfortable, may restore some to useful occupation and occasionally real, complete, symptomatic recovery may even be attained. (21)

In the treatment of heart failure, certain important points must be born in mind. A cardinal principle to emphasize is that all cases of cardiac failure cannot be grouped together and a common type of treatment instituted. (14) It should be determined by history, physical examination, and other diagnostic aids the type of failure present. This includes an understanding of

the underlying causes as well as the precipitating factors and just important as the above two, the question of which ventricle, or both, is failing.

"The treatment of heart failure is too broad a subject to be comprehensively discussed in every detail in the brief space of one paper." (15)

With the implication of the preceding paragraph clearly in mind the discussion of treatment of cardiac failure is hesitatingly undertaken. No two clinicians would absolutely agree on the exact methods of therapy. All would have the same principles in mind but each would prefer certain variations in theraputic measures to achieve the same aims. For example, every physician would attempt some means of combating the cardiac edema, but each one would possibly choose a particular diuretic which had proven most efficacious in his experience. The same statement might be said for diet, enema or cardiac stimulant. Therefore, the general considerations are mentioned and only certain more or less specific

theraputic means of relieving the cardiac distress suggested.

2. Prophylactic treatment

Treatment of cardiac failure begins as soon as the potential disease is discovered. (15) The causative or underlying factors are for the most part beyond control but the precipitating factors can often be eliminated. The patient must avoid overexertion both of physical and mental as well as emotional energy. Acute and chronic infections should be removed in order to relieve the strain of any toxemia on the heart. Every attempt should be made to lower the basal metabolic rate particularly by watching the diet and by adequate rest. A questionable prophylactic procedure is the administration of oxygen for the chronic myocardial anoxemia which accounts for chemical abnormality in hearts of patients who have died of heart failure. (Refer to pathology p. 66) This is accomplished by the administration of aminophyllin which acts as a dialator of the coronary arteries without increasing the cardiac output. (The council on

pharmacy and chemistry of the A. M. A. now accepts xanthine derivatives as diuretics and myocardial stimulants but not as vasodialators.) Small daily doses of the amino acids such as alanine, arginine, glycocoll are recommended along with small daily doses of digitalis in order to conserve the creatine and phophocreatinine disturbances of the heart muscle. (Refer to p. 67)

3. Rest

After the appearance of symptoms of myocardial insufficiency the treatment takes on a more active nature. Rest is the prime requisite and both mental and physical rest must be prolonged. It has been found that Fowler's position is excellent for these patients who must be confined to bed for complete physical rest. The back rest should be firm and if pillows are used they should be hard and offer good support. The patient should not exert himself in any way. His position should be shifted by the nurse and he should use a bedpan. If possible a cardiac table is desirable because it facillitates the use of

a bedpan without the straining and lifting which is almost unavoidable otherwise. Absolute rest is imperative and must be maintained by sedatives if no other way. Morphine sulfate may be used, grains one fourth to one half. Dilaudin, one sixty-fourth to one thirty-second grains may be preferred because it is not as constipating and its withdrawal is not as disturbing to the patient. (31) The length of time bed rest should be imposed is debateable but must be determined by the individual case. When minor symptoms have appeared, absolute bed rest for at least three days is imperative, relative rest for a few weeks thereafter and an extra hour of sleep each night for the rest of life. Bed rest should be imposed for at least a short time after symptoms have disappeared and then return to a more active existence should be more gradual. (31)

4. Diet and gastro-intestinal tract

Diet is an important consideration in treatment. Five or six small feedings per day with a total caloric value of twelve hundred to fifteen hundred calories is more desirable than fewer

meals with higher caloric value. Foods which yield an acid ash and are easily digestable, being high in carbohydrate and protein values, should be incorporated, providing of course there is no diabetis. Sodium chloride should be removed from the diet, the sodium ion being substituted by potassium as much as possible. The fluid intake should be limited to fourteen hundred cubic centimeters per day. Ideally no more fluid should be ingested than excreted. (15) (Refer to edema p. 91)

The bowels should be watched and carefully regulated. When the patient is receiving little food it is not absolutely necessary that a daily bowel movement take place. (21) Vigorous cathartics should be avoided as being too exhausting and not much affecting any edema which may be present. If some catharsis is necessary, a small enema is most efficacious. Also mineral oil and agar agar emulsion, with or without cascara, encourage bowel movements and make straining unnecessary.

Flatulence should be relieved by avoidance of those foods prone to the production of gas and by the administration of a simple carminative. (21)

5. Edema

In order to relieve the heart of unnecessary embarrassment, edema must be controlled both by preventative measures and diuretics. The diet should be salt free and the fluid intake low. (Refer to diet p. 90) For active treatment against edema, the Karrell diet is recommended. This consists of four glasses of milk per day at four-hour intervals, which regieme should be utilized for only a few days (a week at the most) and the additional carbohydrate and protein should be added gradually. Raw unpeeled fruits and vegetables, not exceeding fifteen hundred grams in amount, utilizing their juices only for fluid may be a stable portion of the diet for a time. (15) Later skimmed milk, eggs, and cereals may be added. The acid salts ammonium, potassium, or calcium chloride, (grains fifteen to thirty t.i.d.), may promote

withdrawal of fluid from the tissues. If there is no renal impairment nor nitrogen retention, urea ten to thirty grams per day is advisable. Loss of excess fluid by the bowel is encouraged as indicated as in former paragraphs. (Refer to p. 90) Saline catharsis and hypertonic enemas are considered too drastic and have fallen into disrepute. (15)

6. Diuretics

At times a specific diuretic is indicated if there is adequate kidney function. A combination organic mercurial and xanthine derivative has proven excellent. Such a combination of salyrgan and theophylline is Mercupurin (two c.c. of ten per cent salyrgan and five per cent theophylline) being injected intravenously. (15) The mercurials reduce the tubular reabsorption while the addition of the acid theophylline neutralizes the irritating alkalinity and augments the diuresis as well as reducing the toxicity.

The diuresis is useful also in relieving the dyspnea because with an increased venous

pressure there is a corresponding increase in cerebro-spinal fluid pressure. (39) (Refer to symptoms p. 49)

7. Cardiac stimulation and tone

After all, active treatment of myocardial insufficiency must utilize some stimulation to the cardiac muscle itself. As has been so aptly stated so many times, "It is useless to flog a dying horse," similarly it is unwise to over-stimulate a failing myocardiam. In few other diseases is there a more specific drug than in heart failure. Digitalis is of course the drug of choice but utmost discretion must be utilized in its administration. Digitalis is a cardiac tonic that relieves dyspnea and edema. (15) It acts primarily upon the heart muscle and in auricular fibrillation it acts through the atrio-ventricular node depressing the conduction of impulses from the auricle, thus slowing the heart and immediately eliminating any pulse deficit. The drug improves the circulation particularly in the brain. Most patients require one cat unit (approximately 1.5 grains of standard powdered leaf) per ten pounds of body weight for digitali-

zation. By the rapid oral method the patient is given 4 grains per four hours for two doses; 6 grains per six hours for two doses; and 1.5 grains per six hours to nausea, vomiting, diarrhea, xanthophobia or block. The safer, slower oral method is 3 grains per four hours for ten doses followed by 1.5 grains per six hours for digitalization. (15)

In case of actual emergency, a rapid intravenous method of digitalization is used. If digitalis is injected into the tissues a serious slough is produced. A crystalline glucoside from digitalis lanata, namely digoxin is recommended by Hermann. (15) Digitalization is accomplished by 1/60-1/40 grains. The response to the drug is evident in one hour and complete within two hours. The approved method is the injection of .75 milligrams to 1.0 milligrams (1/85-1/64 grains) at the original dose and a maintenance dosage of 1/120 grains per day.

Digitalis has a creatine conserving effect as well as a hypertrophy restraining action on the heart muscle. (15) Consequently many clinicians thus justify the prolonged usage of

the drug. An argument to such therapy is raised by those men who believe that prolonged usage may produce undesirable toxic changes in the myocardiam causing invisible histological myocardial damage. It is also argued that prolonged usage, particularly as prophylactic therapy, is unjustified because only the hypertrophic and failing heart responds to theraputic doses of digitalis in characteristic fashion.

Digitalis is indicated in all cardiac cases in which there is evidence of congestive failure. It is also indicated in cases in cardiac insufficiency associated with renal involvement. (31) It is entirely useless in many of the functional disturbances such as palpitation, extrasystole, tachcardia, and others. There should be no hesitancy in the use of this drug in moderate dosage in any case of decompensated organic heart disease at any time providing that there are no digestive disturbances.

Indications to cease the use of digitalis are: (a) those referable to the heart and circulation (such as a marked grade of sinus arrhythmia, premature contraction particularly

pulsus bigeminus, ventricular tachycardia, partial heart block, and true nodule rhythm), (b) those referrable to its effects on other organs, namely: nausea, vomiting, and diarrhea.

No matter what preparation is employed, it is best to give digitalis following meals, and it is seldom necessary to administer it more than four times a day. When once the physiological effect is produced, it continues, and two doses each day are sufficient.

Under certain conditions digitalis may defeat its own purpose. Those classic clinical signs of digitalis intoxication may not be present and electrocardiographic changes may be the first indication of an insiduously developing toxic condition which accelerates cardiac failure. Cardiac arrythmias may develop which persist in spite of withdrawal of the drug. In such cases quinidine may be useful in again restoring normal rhythm. (13)

Certain arrythmias, one in particular, namely, auricular fibrillation, have been considered so intimately associated with cardiac failure, that there is some question whether or

not failure is caused by auricular fibrillation or whether the failing heart is evidenced by the arrhythmia. A more or less specific drug is used in this condition, quinidine. Quinidine has three specific indications, auricular flutter, auricular fibrillation, and ventricular tachycardia. Contraindications for quinidine are: a longstanding auricular fibrillation, serious impairment of intraventricular or auriculoventricular conduction, or a hypersensitivity to quinine and its derivatives. (13) Quinidine acts directly upon the sino-auricular node of the right atrium whereas digitalis acts primarily upon the heart muscle directly or else upon the atrio-ventricular node of Tawara. The manner of dosage is an individual variable. Levine has suggested the following routine course from which the physician must make individual variations. (21) The first day the patient's susceptibility to the drug must be tested by an injection of .2 grams (3 grains) given twice at four-hour intervals. The quinine toxicity is manifested by a rash, tinnitus, nausea, vomiting, diarrhea and syncope. Providing these symptoms

do not occur following the original ingestion of quinidine, the second day .3 grams (5 grains) is administered three times a day. The third day the dosage is stepped up .1 of a gram, the fourth day another equal step up of dosage, and so continued until fibrillation stops. This oral administration of quinidine is very convenient for those patients who are in no danger of collapse and in whom there is no quinine toxicity. (21)

Another drug used particularly as a heroic measure in cardiac collapse is a new derivative of oubain called strophanthim K. (5) Strophanthim K is slightly less toxic than oubain and 30% less toxic than strophanthim G as well as being more soluble in water. However, physicians in this country hesitate to use it. Hesitancy is based on the opinion that strophanthim is dangerous, that it can be given only by intravenous injection, and that digitalis given orally can produce the beneficial effects claimed for strophanthim without the attendant dangers. It is unquestionable, nevertheless, that strophanthim K has a quicker action and is more rapidly

eliminated. It should not be used in patients who have been previously digitalized and in whom evidences of digitalization persist. Neither should it be used in too large doses. The recommended dosage is .5 milligrams to .75 milligrams in 10 to 20 cc. of 10% glucose. In this combination there is a slower injection thus allowing the heart to absorb a greater amount. Beneficial effects on congestive failure are noted in a few minutes. The maintenance dosage is .3 milligrams per day for as long as 24 days. The action of the drug produces a marked sweating which aids diuresis. Often accessory measures such as sedatives and diuretics are used along with the strophanthim. (5)

The conditions of heart failure precipitated by an upset in the intrinsic nervous mechanism such as heart block, do not respond well to any cardiac stimulation. (12) Metrazol has been attempted and some beneficial results reported. It acts on the vasomotor and respiratory centers and consequently is more useful in circulatory collapse due to peripheral causes. Camphor has

much the same use as metrazol, the latter being preferred because of its water solubility, its ease of sterilization, and the fact that it can act rapidly subcutaneously. (12)

8. Mechanical methods

Various mechanical means of relieving distressing symptoms of cardiac failure, particularly those caused by edema and dyspnea, are attempted. Southey tubes are sometimes inserted subcutaneously in the hands and feet in an attempt to diminish edema and as much as several hundred cubic centimeters of fluid may be removed daily. (21) Often catheterization will remove a considerable amount of residual urine. If a hydrothorax is present in a degree that causes respiratory embarrassment, it is advisable to drain the pleural cavity. If only one or two hundred cubic centimeters of fluid is present, the strain of thorocentesis is greater than the benefits derived. (21) If a marked ascites is observed, in the abdomen, the removal of two thousand cubic centimeters or more of fluid by abdominal paracentesis will often bring relief. Removal of any amount less than two thousand cubic centimeters seems to prove entirely unbeneficial.

In many patients the total blood volume is increased as evidenced by an engorged and tender liver, distended veins in the neck, cyanosis, and pulmonary edema. In these cases a phlebotomy may be tried, attended many times with outstanding success. (21) The operation is contraindicated in any case in which the blood pressure is low indicating symptoms of shock. The phlebotomy should be done rapidly, four hundred to seven hundred cubic centimeters of blood should be withdrawn in ten minutes. The physician should use a large needle and strict aseptic precautions. Instead of blood letting, a less radical means of cutting down the embarrassment on the heart by too much blood may be tried. This procedure is the application of tourniquets on the four extremities just tight enough to shut off the venous return and so pool the blood in the periphery.

9. Hope

All cardiac patients suffering from any of the minor, and particularly any of the major, symptoms of cardiac failure should be given as much hope and encouragement as possible. No matter how bad the case may seem to be, immediate recovery is always possible even though

permanent recovery may be impossible. (31) A positive reassurance must be given repeatedly and it is surprising what a beneficial effect hopeful announcements have on the cardiac patient. (31)

However, in no case should a false prognosis be given; truth of statement is a first essential. The physician is not always expected to volunteer the whole truth but answers must be given to direct questions. The patient who is in grave danger will himself sense his condition so it is seldom necessary that the physician spontaneously need acquaint him with the desirability of settling his estate. (22)

CHRONIC HEART FAILURE

IX. Prognosis

The ability to gain compensation seems to depend upon several factors. On the whole, the underlying type of heart disease does not seem to be of great importance. In general, the four factors which do more or less determine whether the condition can be improved are discussed in the following paragraphs.

1. Environment of the patient and precipitating factors

The first consideration is whether or not the precipitating cause can be removed or controlled. When the exciting cause can be removed, such as auricular fibrillation, respiratory infection, exertion, thyrotoxicosis, etc., the patient roughly has a fifty-fifty chance of regaining some degree of compensation. In order to remove precipitating causes it is necessary that the patient have a comfortable, pleasant, and restful place of recuperation.

2. Economic and mental makeup of the patient

A second consideration is the ability of

the patient to reduce the demands on the heart. In any series of patients it is difficult to evaluate the seriousness of the heart lesion as regards possibilities of compensation because the personal element cannot be eliminated. Those patients have a better chance of compensation who are better equipped socially, economically, and intellectually to change their mode of life. Thus, perhaps one of the reasons why patients with rheumatic fever coming in the main as they do from a lower social class do so poorly once decompensation sets in. (Refer to p. 24) It is because of the fact that they are unable to maintain an existence in the present economic order and reduce the load on the heart. A certain light might be thrown in a similar manner on syphilitic heart disease. It is general that once failure develops following luetic aortitis or aortic regurgitation the patients do poorly. This opinion might be formed from statistical evidence and yet not necessarily be true because most syphilitic heart disease is among negroes who are unable, due to financial reasons, to

reduce the load on the heart. (2)

3. Complications

A third factor in the prognosis of the patient is the presence or absence of complications. Although obesity contributed to an earlier onset of failure, it does not apparently affect the outcome. Pulmonary infarction, cerebral vascular accidents and pneumonia are common to all groups and do much to make impossible a healthy prognosis. Undercurrent infections play a tremendous role on the prognosis.

4. Age

Lastly the age of the patient will undoubtedly affect the prognosis. Quite naturally, as the stress and strain of advancing years is added to the already overtaxed heart, the chances of regaining compemsation are lessened. (26)

5. Specific questions to be answered

The physician does not possess the prophetic powers of determining how long a patient is going to live. Nevertheless, he must be able to answer certain general questions with some degree of certaincy and accuracy. The following are certain

criteria upon which the answer to the various questions are based. (22)

a. Many years expectancy

Young adults with valvular lesions and with little or no signs of enlargement and with good or fair exercise tolerance fall into this class.

b. About ten years expectancy

Early cases of angina of effort, including cases in which a fair tolerance to effort and little enlargement of the heart has taken place can reasonably be promised a number of years expectancy.

c. About three to six years expectancy

Those patients who can walk only a short distance without angina, or who have moderate enlargement complicating valvular or syphilitic heart disease, and those who have decompensated, cannot expect a life of any activity beyond a few years.

d. Live precariously up to one or occasionally two years

Patients with great enlargement, or with

anginal attacks unrelieved by rest or with unrelieved congestion are living on borrowed time.

e. In imminent danger

Those persons with edema of the lungs, a recent coronary or with a Stokes-Adams syndrome are in great danger although they may recover for the immediate future. Patients with a leaking aneurysm or in a severe unrelievable anginal state or in extreme congestive failure, rarely, if ever, recover.

However, it must be remembered in all cases, that it is possible for an immediate recovery to take place in every patient although a permanent recovery might be out of the question. Hence, the prognosis must be guarded at least until a treatment consisting mainly of rest has been tried, and the functional efficiency of the heart thus in some measure determined. (26)

CHRONIC HEART FAILURE

X. Summary

There are a few definite facts which must be emphasized. These points are the ones which should remain in the reader's mind whenever chronic heart failure is discussed. They may be taken almost as axioms and with them as a background, a comprehensive understanding of heart failure, its method of production, its manifestations and its treatment can be grasped.

1. Chronic heart failure can be used as an inclusive term for myocardial insufficiency, cardiac decompensation, and congestive heart failure. These other terms have their peculiar implications built up by years of usage.

2. More persons die as a result of heart failure than from any other cause. It is primarily a disease of old age but affects both sexes and all social orders with equal incidence.

3. The three main causes of cardiac failure are coronary, valvular, and hypertensive heart disease. Arteriosclerosis, particularly of the atheromatus type, is important in the etiology of coronary and hypertensive heart disease. Rheumatic fever and

syphilis are important factors in the development of valvular disease.

4. There can be no rational correlation between the appearance of the heart at autopsy and symptoms during life.

5. The heart has three means of compensating for difficulty in maintaining circulation, namely: (a) increase in rate, (b) hypertrophy, and (c) dilitation. There are limits beyond which these compensatory mechanisms prove useless.

6. The subjective symptoms reported by the patient are to be more carefully weighed in the determination of cardiac pathology than any system of physical signs.

7. In the end analysis it is the functional impairment of the myocardial fibers which causes failure. There is a decreased cardiac output as well as a state of congestion in the veins in most cases of cardiac failure. Forward Failure and Backward Failure cannot be completely separated.

8. Most commonly, both ventricles fail simultaneously, although the left ventricle will fail alone followed by right ventricular failure more times than the primary failure of the right side of the

heart.

9. The aim of the treatment is not to establish a complete cure because such a recovery is impossible. If the underlying causes are present, the patient will eventually suffer heart failure, but all precipitating factors should be removed. No one regieme of treatment can be outlined for all cardiac patients.

10. Digitalis is the drug of choice for myocardial stimulation. There should be no hesitancy in using it when definite organic heart disease is present. More rapid cardiac stimulants may be employed, but their use is not unattended by certain danger.

11. Although it is possible for an immediate recovery in every case of heart failure, a guarded prognosis must be made concerning any permanent cure. No hopeless attitude should be taken until a treatment, consisting mainly of rest, has been adequately tried.

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