5-1-1932

Mechanisms in cardiac failure

Ellis E. Baker

University of Nebraska Medical Center

Let us know how access to this document benefits you
http://unmc.libwizard.com/DCFeedback

Follow this and additional works at: https://digitalcommons.unmc.edu/mdtheses

Part of the Medical Education Commons

Recommended Citation
https://digitalcommons.unmc.edu/mdtheses/190

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.
MECHANISMS IN CARDIAC FAILURE

Nebraska University
College of Medicine

Ellis E. Baker
Mechanisms in Cardiac Failure

For the year 1929 "Diseases of the Heart" holds first place in the principal causes of death in the continental United States (53). One in every six deaths was due to diseases of the heart.

It is the purpose of this paper to emphasize a few questions in regard to the adequacy or inadequacy of the more commonly accepted concepts of the mechanism occurring in a group of these heart diseases which are variously known and described under such terms as "Cardiac failure", "cardiac insufficiency", "cardiac weakness", "cardiac asthenia", and "cardiac exhaustion".

It is conceded, in the beginning, that the human body is not built for the same modus operandi as was the Deacon's Wonderful One-Hoss Shay; that is to say, the admission is made that the probability of breakdown of one part of the individual make-up, more quickly than other parts, is always present. The question would seem to arise, then, as to why the heart is the one part to be the site of this breakdown in so large a number of cases as to make it outstanding. Perhaps this question would scarcely need this emphasis if all individuals were living under uniform conditions which are apparently conducive to special strain or to any and all other elements which might lead to pathology and death of the heart. The heart has a function to fill as a muscle, specialized though it be, and questioned though it be as to the degree of its vital role. Does it fail to perform its function? Does it fail more often than do other muscles, composing other organs and other structures, of the body?

The possibility that the heart is more liable to infection than other muscles of the body, because of its contact with large blood mass,
might even be conceded and still have remaining the large class of hearts which have been judged as being perfectly normal but which also have been said to have failed. Other muscles perhaps play as vital a role in life as does the heart and it is hard to conceive of Mother Nature's placing the heart muscle in such a precarious position without placing also compensatory protections and safeguards. The heart muscle might be thought of as being intermediate in its properties between the smooth musculature as found in the gastro-intestinal tract, and the striated skeletal muscles. It thus possesses qualities which result in greater efficiency in function than do either of the other two classes of muscles when considered singly. The heart of lower animals when used for experimentation has been found to be very resistant to trauma and to environmental changes which would appear to bear out this comparison.

There are many other questions which arise and which will be discussed in the course of the paper.

The term "dropsy" as a cause of death is objectionable to the census bureau for the very obvious reason that it is a symptom and not the disease. Somewhat similarly the terms "heart disease", "heart trouble" or "organic heart trouble", "heart failure", "cardiac weakness", "cardiac exhaustion" are unsatisfactory to clinicians who would seek always to get to the basic cause of a pathological condition and so correct it. They are also unsatisfactory to the doctor who believes that the last word has not yet been said in therapeutics and who is looking towards an ultimate ideal treatment in cases classed under such terms as the above ones.

Because of long use and their connotations, however, many such, loosely-applied terms must necessarily be used here.
An attempt is made to present views of present-day research workers, varying from pure scientists to those who are obliged to draw conclusions from a purely clinical point of view. Past views will be mentioned also and the reasons for all set forth briefly.

A history of cardiac failure is included in the more general history of cardiology itself (7), the former being the central point of interest in any study made of the latter subject.

Cardiology is the youngest child of medical evolution and has not quite as yet come into its own as a distinct specialty. Though the cardiologist likes to believe that a complete and elaborate knowledge of all the facts of nature in relation to the heart are advantageous, yet in his practice he remains practically as empirical as did his ancestor in ancient times. Complete knowledge is not necessary for correct practice, but it is an insurance that practice shall be correct. It does not make any difference to the patient whether a particular measure is carried out because of tradition or because of the application of a multiplicity of methods and a highly elaborate theory.

Before the fifth century B.C. the various phenomena of life were considered as evidences of supernatural power rather than as anatomical or physiological manifestations. In ancient Egypt, for example, the heart was thought of as having something to do with the future life of an individual and after death was carefully preserved outside of the body but in the vicinity of the mummy.

Plato (427-347 B.C.) considered the heart as the seat of the higher emotions. "It may be remarked that in some vague way Plato hinted that the heart was the ruler of the arterial circulation"(7).

Aristotle (384-332 B.C.) believed the heart was the source of the blood and that the blood was the general nutritive fluid of the body. "He thought that the beating of the heart and the pulsations of the
vessels were due to the expansion of the blood within these cavities rather than to any active motion of the fluid itself. Strangely enough he did not consider the pulmonary arteries and veins as part of the circulatory system but believed them to be entirely separate. "(7) He realized that the heart contained blood and not air as was believed by some of his followers and he described three cavities in the heart, omitting the right auricle.

Praxagoras (300-400 B.C.) distinguished between arteries and veins, by the pulsations in the former, but believed that the arteries contained merely a mysterious vapor.

Erasistratus and Herophilus (Alexandrian period, 331 B.C.) originated dissection. Erasistratus named the valves and the chorda tendenae and thought there must be some communication between the arteries and veins. The anastomosis between the arteries and veins was not thought to be a permanent one but was only thought to be brought into play during disease or undue activity. The doubtful, vital spirit, generally designated as "pneuma" was believed to be the content of the arteries.

Galen (131-201 A.D.) discovered blood in the arteries. He recognized the importance of the muscles in the heart and blood vessels and drew the conclusion that the impulse for the heart's contraction originated within the organ itself.

In the fourteenth chapter of the book of Luke and the second verse, an account is given of the healing of the man with dropsy.

During the next period, the dawn of modern cardiology, processes in the body were considered as some power of nature more or less controlled, if not completely understood, by definite laws.

Mondino de'Luzzi (1250-1325) was doubtful of Galen's views of the permeability of the interventricular septum.

Leonardo da Vinci (1452-1519) believed that systole was the impor-
tant phase in the cardiac cycle and not the diastole.

Sylvius (1478-1555) first described a foramen ovale of the fetal heart. He also knew that valves existed in veins.

John Winter (1487-1574) described muscular functions of the heart.

Vesalius (1514-1564) dissipated the error of interventricular pores.

Servetus (1509-1553) described the pulmonary circulation as separate from the systemic.

Erissot (1478-1522) reformed the practice of venesection as a therapeutic aid in an embarrassed circulation. This mode of treatment is used today and consists of free blood letting on the same side and near to the lesion.

Fabricius (1537-1619) described the valves of the veins.

Cesalpino (1524-1603) is considered by the Italians the discoverer of the circulation of the blood before Harvey.

Harvey (1578-1657) put the discovery of the circulation in concrete form and popularized it and made a mechanical science of physiology.

Malphighi (1628-1694) made existence of capillaries a histological certainty.

Stenson (1638-1686) described the true muscular tissue of the heart as being the factor concerned in contraction.

Francis Glisson (1597-1677) showed that when a muscle contracts its volume does not increase and discussed at some length the muscular contraction of the heart.

Giovanni Maria Lancisi (1655-1720) noted hypertrophy and dilatation of the heart and that the latter sometimes caused sudden death. He attempted a classification of heart disease.

Morgagni (1682-1771) described pathology of valvular lesions and is said to have recorded the first case of heart block.

Hales (1677-1761) was the first to accurately measure blood pressure.
Jean Baptiste Senac (1693-1770) published a work on the structure of the heart, its action and diseases. He also studied the irregularities of the heart to some extent and discussed such symptoms as orthopnea, dropsy, and so forth in its relation to cardiac decompensation. Quite modern is his observation that you cannot always connect up the symptoms and signs found in a living person with the pathological findings in a cadaver.

The third period is that of scientific application of principles. Auenbrugger (1722-1809) discussed percussion and fremitus. Jean Nicolas Corvisart (1755-1821) who was physician to Napoleon, and a teacher, said, "Upon the muscular efficiency of the heart depends life itself," and so made a remark that is strikingly modern and of the greatest importance. He believed hypertrophy and dilatation to be the chief causes of abnormal cardiac function and that valvular disease was purely secondary to this.

William Withering (1741-1799) did his monumental work with digitalis and stated the proper dosage as being up to the point of nausea in the patient and that its chief indication was dropsy, — making no distinction yet between cardiac and renal dropsy.

William Stokes (1804-1878) thought that both muscle and valvular defects are important in the appraisal of a diseased heart. He judged cases by clinical rather than by speculative means and his name is familiar to the profession in the "Stokes-Adams" and Cheyne-Stokes combinations, — both having to do with circulatory phenomena.

Laennec (1781-1826) by using his stethoscope gave distinctions between hypertrophy which he said produced a rumbling sound, and dilatation which gave a short, sharp tone.

Jean Poiseuille (1799-1869) invented the mercury manometer for determining blood pressure.

James Hope (1804-1841) said the second heart sound was due to
the closure of the pulmonary valves.

Marey (1830-1904) perfected the sphygmograph.

Three rather recent cardiologists are Theodor Schott, J.M. Groedel and Sir James McKenzie. Theodor Schott had a marvelous command of psycho-therapy and an empirical knowledge of the heart sufferer and reasons for his remarkable cures are disputed. J.M. Groedel systematized examination of heart sufferers, using the orthodiagram and later electrocardiogram studies together with routine examinations of blood, urine and blood pressures. Sir James McKenzie laid stress on the subjective feelings of the patient. He believed that cardiac failure was the most important thing to consider in appraising the cardio-vascular system of a patient, and he also soon observed that failure was due, as he says, rather to muscular exhaustion of the heart than to valvular incompetence. His outstanding contribution was his appreciation of the true significance of auricular fibrillation and its specific treatment by digitalis. He detected by polygraph the evident paralysis of the auricle in cases of total irregularity of the pulse. He showed that digitalis slows the heart. He also noted that infectious diseases often lead to cardiac complications. He believed that high blood pressure acted as a compensatory mechanism and so treated it by good elimination and support of the circulation,-prescribing castor oil, exercise, low protein, salt with nitro-glycerin for the cardiac pain.

Einthoven may be considered in this group of men because of his work with the x-ray, fluoroscope and electrocardiogram.

In general, we may speak of two forms of circulatory failure, namely the central or cardiac and the peripheral forms(20). "If the heart becomes unable to maintain an efficient head of blood for the purpose, circulatory failure of cardiac origin will result"(20). As a matter of distinction these same authors have the following to say of peripheral circulatory failure:-"There is evidence for supposing that organs which
are in a state of activity can augment the quantity of blood they receive by the action of their metabolites in dilating the arterioles which supply them. At the same time, the parts of the body which are inactive have their blood supply curtailed by means of the vasomotor nerves. When these mechanisms break down, as they do in shock, in such a way that the terminal vessels become dilated over most of the body at the same time, a failure of the circulation takes place at the periphery. The blood stagnates in the various organs; there is a diminishing return through the veins to the heart; and death results from a lack of a sufficient quantity of blood in circulation.

"Cardiac failure and peripheral failure are usually quite distinct. They occur under different circumstances; cardiac failure is the more common." (20)

Clendening (14) gives a very satisfactory definition of cardiac failure which offers an approach to its study from a physiological point of view. He defines it as the failure of one of the functions of the heart muscle, i.e., tonicity, rhythmicity, conductivity, and contractility. Tone, he defines, as that property of heart muscle which prevents complete relaxation during diastole; rhythmicity as regularity; conductivity, as transmission of impulses along muscle bundles; and contractility, as cell shortening during systole.

Barringer (3) states that the danger to which children, with cardiac disease, are exposed, is more damage to the heart from infection. He says further, "There is, however, a large group of heart patients who suffer from attacks of what used to be called "decompensation" and are now called "heart failure" or cardiac "insufficiency" in which the cause of the breakdown is not considered to be an infection. It is thought to be, rather, a physical or mental strain, or the very nature of the mechanical defect in the valves is supposed to lead ultimately to "decompensation."
Another brief definition and the immediate problem is well stated by Kerr(34); "Heart failure, the inability of the heart muscle to maintain the circulation for the ordinary needs of the individual." There is no parallel between the pathology of the heart, as ascertained by physical examination, and its functions. The functions of the myocardium form the ground-work of the present knowledge of the heart. In one of the largest clinical groups, the cardiac disease associated with hypertension, nephritis and arteriosclerosis, the evidence is strong that the initial pathology arises elsewhere in the vascular tree than in the myocardium, probably in the smallest arteries, the cardiac and renal changes being secondary processes. The term "circulatory failure" in this group is more accurate than "heart failure".(34).

Allbutt (1) has placed this line of thought in a near classic from which the following excerpts are taken.

"In my youth it was a common jest to ask what N or M died of, the witty answer being "shortness of breath of course." the jest has vanished, but the platitude remains, with some alteration of the formula, which, instead of "shortness of breath", now runs "of heart failure". Now, do we owe this new fashion to the physician or to the pathologist? Does the phrase mean that a snap of the heart stopped the whole machine, otherwise capable of working on, as a snap of a connecting rod may stop the engines of the Olympic? or does it mean that the heart was the ultimum moriens, and that, so long as this central organ held to it, death was kept at bay? In this case to say that one died of "heart failure" is surely as empty a truism as to say that he died of "shortness of breath".

"Kirkes, Bence Jones, Wilks, Fagge, T.H.Green, and many others began to demur at formerly accepted reasons for heart deaths. By what system of compensations was it that the same heart could be at once
fairly competent clinically and yet pathologically the seat of old and intimate decay? By what system of compensations could a heart go on fairly well, or indeed without suspicion, while its structure was being undermined? Surely there ought to have been some stage of incompetency or at least of falter before the sudden collapse."

Allbutt(1) then takes up in detailed discussion the common clinical signs and symptoms commonly associated with impending heart failure as they are presented to the doctor and concludes that more or less sudden heart failure is frequent without any of these features. Next he turns his attention to the pathologist and his common findings in the heart at autopsy. He not only discusses findings which he himself has experienced but refers to other authorities with similar discussions and concludes,"In this heart he (the pathologist) found too little to account for death, in that too much—the patient had no business to have lived so long." He observes that cardiac fibrosis seems to have some conservative value and the riddle of frequent death in one who scarcely having known illness, expires under no extraordinary effort, remains unsolved. Allbutt does, however, offer one loophole of possibility for satisfactory explanation, perhaps to his own mind in the following," In all living structures there is a factor of safety, that potential which in the heart we call reserve, but in none, perhaps, is it so capacious as in the heart. Yet of this reserve, of what the heart can do in case of need, we have no scientific measure. We begin to learn in the heart, as we learn in less striking ways in other forms of organized matter, that complexity of structure and condition mean not less but more stability; that equilibrium endangered in one direction, or in more than one, may be supported by readaptations of other factors of the concert."
From Floyd (24) we get a bird's-eye-view of the changing attitude toward disease with its application to heart failure:

"Half a century ago, the symptoms of disease were commonly regarded as the result of pathological changes in the body's structure. During the last century attention was focussed chiefly on valve lesions and the term "organic heart disease" was then almost synonymous with "valvular disease". In recent decades more and more emphasis has been laid on the importance of primary failure of the heart muscle. In this country where there are so few autopsies to control our thinking, a widespread tendency has arisen to interpret disease in terms of function alone, and to disregard the lesions. Disease is coming to be looked on as the reaction of the living body to irritating stimuli, a reaction which finds expression both in symptoms and in lesions. This view is well exemplified in the modern view of heart failure. If all the cases of heart failure which show no valvular lesions at autopsy are taken together, they form a group about twice as large as all those in the group which includes valve lesions. Below forty years of age, valve lesions preponderate, but beyond forty, heart failures without valve lesions are far the more numerous. Heart failure in its ultimate analysis is a functional disaster."

Soma Weiss (48) sets forth the following concept: "A majority of the symptoms and signs of the patient with cardiovascular disease depend rather on the state of the circulation than on the heart. Whenever the circulation fails to accomplish its task of efficiently supplying the tissues with nourishment and promptly eliminating the waste products of cell metabolism during the normal activities of life, we speak of circulatory failure. Heart disease may coexist with a circulation normal in every respect for years. However, failure of the circulation may be independent of heart disease." The two are not inter-changeable in use. "Circulatory failure often results from primary
derangement of the vasomotor system, as shock following surgery or trauma, in certain diseases of the nervous system, infectious diseases and toxemias. Certain investigations have suggested that primary changes in the peripheral circulation are also responsible for the failure of the circulation in heart disease. Thus it has been claimed that primary disturbances in the lactic acid metabolism cause important changes in the peripheral vascular system and precipitate the sequence of events that results in circulatory failure. A recent investigation in the Boston City Hospital fails to support this claim. No primary disturbances of the lactic acid formation before the appearance of an adequate blood flow could be discovered. All the evidence obtained by us thus far supports the logical concept suggested by clinical manifestations and post mortem findings, that local disturbance of the heart is primarily responsible for the circulatory changes (49).

There are certain clinical signs which are commonly accepted as indicating cardiac failure when such failure comes gradually. Of course, it is questioned as to whether cardiac failure is ever sudden. Cardiac failure indicates a condition showing either a sudden failure of the heart, as in pulmonary edema following coronary sclerosis, or a gradual failure, evidenced by a progressive decrease in the heart's reserve power, i.e., declining exercise tolerance, and the signs of venous congestion (3).

So-called early symptoms include dyspnea, fatigue, precordial pain and palpitation during amounts of physical exertion which formerly did not cause these symptoms. Later signs are those of venous congestion and are cyanosis, engorged cervical veins, enlarged liver, and edema of subcutaneous tissues, or the lungs, and of serous cavities (3).

"They (the early symptoms) are the same symptoms that any person with a normal heart experiences when he performs work beyond his
heart's capacity." (3) These are symptoms of overtaxing the heart. When heart failure begins, each day, smaller and smaller amounts of work call forth these symptoms of cardiac overtaxing until finally he is confined to bed and the signs of venous congestion appear or increase. "To express the sequence of events in different words, his cardiac reserve power, as shown by the exercise tolerance, has steadily decreased until the heart is unable to carry on circulatory demands, even with the body at rest.

"The symptoms of overtaxing the heart are frequently called symptoms of cardiac failure. Strictly speaking, this is incorrect". They may be present with no sign of heart failure. Their characteristic is their progressive increase with smaller and smaller amounts of physical exertion. (3).

Based on the assumption that heart failure cannot occur without an impairment of one or more of the properties of the heart muscle, Bohan (10) classed cases for study, all of which showed clinical signs of disturbed myocardial function:—First, impaired tonicity, including valvular lesions, hypertension, aneurysm, dilated aorta, myocardial degeneration and influenza; second, disturbances of rhythm, showing auricular fibrillation, paroxysmal tachycardia and auricular flutter; third, depression of contractility, with hypertension, toxic goitre and coronary disease cases; and last, lessened conductivity, in two classes,—syphilis and cause unknown.

Formerly, universal assent was freely accorded the time-honoured theory of back-pressure with its sequence of events. (40). As so well described by Osborne (39) this theory is as follows: First there is the phenomenon of dilatation which is due to disease, degeneration or increased work. The left ventricle perhaps is the first to dilate causing enlargement of the mitral valve opening. The left auricle then, unable to cope with the regurgitated blood, allows
congestion to occur in the lungs. The right ventricle then finds increased pressure to overcome ahead in the lungs and it becomes dilated because it cannot evacuate its contents perfectly and must of necessity accommodate an ever-increasing volume of blood. The right auricle, likewise is soon unable to force its venous blood into the right ventricle with the result that a damming back occurs and the circulation becomes sluggish in the superior and inferior vena cavae. There results congestion of the lungs and dypnea, passive congestion of the various organs with headache, venous congestion of the eyes and throat, cerebral irritability, sleeplessness, inability to do good mental work. The enlarged liver produces imperfect bile secretion, imperfect antidotal liver action and venous blood is dammed back in the portal vein giving a congested stomach, congestion of the mucous membranes of the bowels, imperfect secretion of digestive fluids, congestion of the spleen. Likewise, the renal congestion produces a concentrated urine and perhaps an albuminurea. The sluggish venous flow together with the imperfect arterial tone results in imperfect circulation to the feet and legs with the pendant edemas which disappear at night.

However, according to one school of thought, (40) back-pressure does not necessarily exist in cardiac failure and we have no definite evidence of etiological relationship between the back-pressure state and subsequent heart failure. Parsons-Smith believes that both depend on a common etiological factor,—myocardial incompetence.

The mechanics of cardiac failure have been much obscured in the past by the "vis a tergo" and the "vis a fronte" controversy (20). The importance of the old or "vis a fronte" view lay in the fact that a pulmonary congestion was ascribed to a defect in the ventricle, while venous engorgement and edema were due to a failing right ventricle. It became untenable in its entirety when it was realized
that cardiac failure could supervene in hearts in which there were no valvular lesions, nor were there any murmurs to suggest a functional incompetence of the valves. The view of a "primary myocardial deficiency" or the lack of the "vis a tergo", was therefore substituted.

The "vis a tergo" or myocardial deficiency view considered the heart muscle as the important thing and the valve lesions of secondary account since they could be compensated while the myocardium was still adequate. The heart muscle failed as a whole and resulted in edema and venous congestion due to the inefficient propulsive power.

The mechanism of normal heart response occurs as follows: First, a demand occurs for an increased output. The demand is the cause for a change in the heart rate through change in the vagal and accelerator balance of impulses. The volume and energy output of the ventricles is changed, that is, the ventricles are filled and the initial tension in them is increased due to the increased venous pressure. That cardiac stimulation at a rate exceeding so-called critical, is associated with functional failure of the organ, deficient output per beat, loss of muscular tone, dilatation of the chambers and venous engorgement without a compensatory rise in initial tension, may conveniently suffice as a preliminary introduction to the modern theory of cardiac failure and its etiology in contradistinction with the alternative mechanism of backpressure (40).

Two basic concepts are essential to bear in mind in studying heart failure (24). The first is that failure of the heart's power is a functional event. That is, the methods of investigation now available cannot demonstrate any structural change in cardiac muscle constantly associated with it. In other words, if fifty hearts were submitted without clinical history to the laboratory for anatomical study, the pathologist could not tell, unless the heart wall were actually ruptured.
or unless extreme dilatation were present, which of them had given evidence of heart failure during life and which had not. On this point of fundamental importance practical unanimity of opinion is finally being reached.

"A second fact that must be grasped in order to understand these cases is that hearts vary enormously in their ability to withstand injury, so that the same damage, either toxic, nervous, or anatomical, may be affect one heart that death results, which, in another heart, would cause only a very moderate and transitory disturbance of action."

Laws governing the heart contraction in health to which no theory of mechanism of cardiac failure must run counter are: (20)

Starling's law:-The force of the cardiac contraction is determined by the amount that the individual muscle fibers are stretched at the beginning of systole. If the optimum stretch is exceeded, the contractions weaken and the chambers become permanently dilated.

The force of contraction of each ventricle must be in a constant proportion to the resistance it has to overcome. Each ventricle must expel equal quantities of blood. The force of the left ventricle contraction is normally controlled largely by that of the right, since the left ventricle has only to deal with that amount of blood which reaches it from the right side through the pulmonary system. (Henderson and Prince). Harrison's law states that when the right ventricle output is diminished, that of the left falls to the same level. (27).

During exercise the heart accelerates. Increased muscle work leads to excess carbon dioxide in the circulation which constricts the veins causing increased venous pressure. The Bainbridge reflex occurs as follows:-Increased muscle work produces vaso-dilatation and the muscles drive the blood along in the veins causing a rise in the
venous pressure with diminished vagal tone and increased sympathetic tone.

In normal subjects severe exercise causes a breakdown of the circulatory and respiratory functions with a complete failure to eliminate the carbon dioxide and lactic acid so that exercise ceases and protects the heart from damage. (Clark-Kennedy and Owen). According to the same men, exercise in diseased hearts causes the limits to be exceeded and cardiac failure to result.

Cardiac failure decreases velocity of blood flow (§) in proportion to the amount of dyspnea and edema. Edema may be expected when the velocity of the returning venous blood is less than half the normal rate.

In cardiac failure, the minute volume of blood flow was reduced to about half the normal. (Davies and Gilchrist) (Kininmonth).

Rise of venous pressure means that the right heart fails to pass all the blood that returns to it on to the left heart. "There is no doubt that the venous pressure is often raised in cardiac failure" (Blumgart & Weis) (Eyster & Middleton) (Danzer19).

The principle of Fick (21) states that the output of the heart may be greatly increased when heart failure sets in (43).

In may be well to state some conclusions of research workers along these lines. The reduction of heart output favors relief of heart failure symptoms. Heart output is less in the sitting position than in lying down. (22). Digitalis reduces output approximately 25% after administration of the full therapeutic dose (28).

The orthodox conception of mechanisms involved in the ordinary signs of cardiac failure are about as follows:

First, there is dyspnea which is concerned with carbon dioxide in the blood and the respiratory center. The latter is equally sensitive
to any acid radicles (14). Dyspnea can be caused by metabolism increase which results in an increase of acid bodies in the blood. Cases of cardiac failure have some increase in metabolism (41), but not a sufficient increase to account for the marked degree of dyspnea that is usual. In regard to acidosis, cardiac failure cases collect carbon dioxide in the blood and the tension becomes high, while that of the alveolar air is low. This discrepancy tends to disappear as compensation is regained. Blood composition in cardiac failure is unchanged as a whole (14).

The depth of respiration is decreased in cardiac failure. This has been variously attributed to pleural effusion, exudate, and lessened lung elasticity due to stasis, all of which are inadequate.

Cyanosis in heart failure is attributed to the lack of oxygenation of the blood due to the decreased mass-volume output and consequent accumulation of carbon dioxide in the blood. (14).

According to Florence Sabin, the tissue spaces are closed spaces, lined with lymphatic endothelium and intimately connected with the veins. Edema is thereby accounted for as being due to obstruction of the venous return, to vital secretory activity of the endothelial cells (Haidenhain's postulate), or when acute in the lungs, to left ventricular failure and right ventricular preponderance (Welch).

The irregular pulse of heart failure may be classed as to the extent of the myocardial damage (14). First are those where no change of the myocardium is indicated as sinus arrhythmia, whose rates increase on inspiration; extrasystole, and sinoauricular block with dropped beats and irregularity after exercise. Second are those which may indicate cardiac failure or not as the tachycardias. Third, those which indicate heart failure but are remedial, as auricular fibrillation, which is said to be the most common cause of failure; auricular Flutter; and nodal rhythm in which the impulse arises in the node (White). And fourth are those indicating grave damage, as pulsus al-
ternans and heart block.

One other sign of heart failure, not always mentioned, is orthopnea. The rate of flow of blood is diminished by a quarter in the sitting position and by a half in the standing position, so that the venous return to the heart is smaller. Therefore the output from the right ventricle would decrease and the left ventricle would be better able to deal with the pulmonary congestion (23). Also, the vital capacity of the lungs is less in recumbency than in the upright position (12).

Circulatory efficiency may be considered as an index between efficient capillary blood supply and tissue metabolism. It is disturbed by abnormal changes of the circulation or by an increase in metabolism. Clinical symptoms and signs are often the expressions of an altered balance rather than absolute deviations from the normal (48).

Underlying causes of cardiac failure as given by Hay (29) are:
1. Myocardial inadequacy resulting from the effect of toxins, as in acute rheumatism, diphtheria, pneumonia, and influenza.
3. A steady persistent rise in blood pressure.
4. In cases of valvular disease, an increase in the degree of mechanical defect.
5. Persistent increase in rate: (a) paroxysmal tachycardia; (b) rapid irregular action in auricular fibrillation.
6. Vascular or lack of vasomotor tone.

It is to be noted that in the above list of underlying causes, as in practically all the leading writers on the subject, the heart muscle is placed in the leading role. This is further emphasized by East and Bain (20) as follows, "This new advance was two-fold. MacKenzie was the
leader. It depended on an idea and a method. The idea was the conception of the importance of the heart muscle, first and last. All our modern views of the nature, causation and treatment of heart failure depend on this. The method was the graphic method. "And again, "The two main lines of advance have resulted in the recognition of the importance of the myocardium, and the clarification and classification of the arrhythmias. Following upon the recognition of the importance of the heart muscle, the significance of its failure has assumed full proportions. But now attention is turning from the central pump to the periphery, or rather to the circulation as a whole. The point of view is shifting back to Harvey, and we are more and more inclined to include failure of the heart in the wider conception of failure of the circulation. The recent work on the variations in the circulation rate, and the effects produced by them at the periphery, is very important."

Starling's conclusions regarding heart failure (54) are somewhat different than the majority of workers. He observes that blood pressure does not fall in heart failure, that there are very often in heart failure, cyanosis, distended veins, and signs of inadequate circulation. "Direct measurement has shown that there is in fact a defective circulation, that the heart at each beat and in each unit of time is putting out less blood than normally. The essential factor, therefore, in heart disease is diminished output of the heart." Heart disease is considered here only insofar as it is thought to influence heart failure. Starling's observations on the action of the isolated heart and their application to the heart in heart failure seem rather inconclusive:—"If either the inflow or the arterial resistance is increased too much, the blood may accumulate in the heart cavities, the diastolic pressure rises, and the output falls off." Maximum output is increased by opening the pericardium and the heart distends. The greater the dilatation of the heart—the stronger is the contraction.
With a constant inflow the heart's contractile power, or its physiological condition, is measured by its diastolic size. As its contractile power deteriorates, or as its physiological condition deteriorates, its fibers have to lengthen to exert the same force during contraction. It therefore dilates and has considerable residual blood. It has bad tone. The heart grows larger as it becomes tired, though there may be no signs of failure.

Fatigue, or diminution of the heart's physiological efficiency, reduces the heart output because the pericardium and the fibrous tissue of the myocardium in disease, does not allow the heart to dilate more during diastole.

"In the belief that venostasis unloads the heart sufficiently for recuperation, its effect was tried on clinical cases of heart failure requiring large doses of digitalis. Only far advanced cases were studied. Digitalis was not given and no other treatment besides the restriction of salt and fluid intake was applied. Venostasis was produced for ten to twelve minutes every two hours. Marked improvement in the dyspnoea resulted. Orthopnea disappeared. The edema lessened and the water excretion increased—in some cases exceeded the fluid intake." (19)

The conclusions are that the venostasis artificially applied, depletes the heart, and reduces diastolic filling; that digitalis increases the heart tonus, and reduces diastolic filling; that venostasis is an effective procedure in cardiac asthma and heart failure. It can frequently replace digitalis in the treatment of cardiac decompensation. A convenient apparatus called the "Venostat" is presented in the printed article. (19).

Harrison and Leonard (28) found that digitalis acts by reducing the cardiac output. From their work it appears that digitalis is a cardiac sedative rather than a stimulant. The keynote of therapy of cardiovascular failure may be cardio-sedative measures. The principle of the
venostat method is that of inflation of manometers simultaneously on each of the extremities up to the systolic pressure. (19)

To create an empirical phrase and apply it as "functional event" has been applied to heart failure, would seem to help but very little, if any, towards a practical understanding of heart failure mechanism.

In his discussion of congestive heart failure or myocardial insufficiency under which he places the two functional disorders of the heart designated as congestive failure due to insufficiency of the myocardium and angina pectoris due to insufficiency of the coronary circulation, White (52) states that a perfectly normal heart may fail under sufficient strain. "It is a question in such cases purely of muscle fatigue with the abnormal chemical state that exists in an exhausted muscle. Much has been written about abnormality and limited reserve of the myocardium as the primary causes of heart failure, while such factors as valvular disease have been considered more or less incidental; this point of view is incorrect. In fact, the older views that certain organic lesions were of prime importance were more nearly correct than the recent teaching that the heart muscle is everything and that little else matters; as usual the truth rests between the extreme views. --It is of great importance to realize that a heart muscle, strong and even massive and healthy, may fail simply from severe strain in its effort to overcome some defect, with never a sign of any degeneration or inflammation, as is frequently illustrated by hypertrophied healthy left ventricular muscle in a heart that has failed from essential hypertension (hyperpiesia) and by the hypertrophied healthy right ventricular muscle in a heart that has failed from marked mitral stenosis."

A post mortem study of 102 cases of myocardial failure by Clawson (13) resulted in the following figures:

Coronary sclerosis of serious degree present in 22.5%
Myocardial fibrosis of marked or moderate degree in 20.5%
Myocardial fibrosis of slight degree present in 30.0%

"There is usually a close correspondence between the situation and the extent of myocardial fibrosis and the distribution and degree of coronary sclerosis. Myocardial fibrosis is usually due to coronary disease, but occasionally rheumatic infections may give rise to a slight degree of fibrosis. Myocardial strain (hypertensive or non-hypertensive) is not a cause of myocardial fibrosis. Myocardial failure is rarely due to anatomical changes in the myocardium. It may be explained as an exhaustion of the cardiac muscle. True chronic inflammation of the myocardium is very rare. What is commonly called "chronic myocarditis" is usually myocardial fatigue resulting from the various conditions mentioned above. Approximately half of the cases of myocardial failure show no anatomical changes in the heart muscle. The anatomical changes in the heart muscle are seldom sufficient in themselves to cause death." (52)

White then goes ahead to explain the signs and symptoms of this type of failure on the basis of back-pressure resulting from the inadequacy of the heart function.

In a concept of circulatory failure, Soma Weiss (48) divides the mechanisms involved according to the stage of the failure. The following findings are noted:

Heart disease causes dyspnea and weakness with smaller amount of muscular activity than it does in the normal individual. In the beginning, the blood supply of the organs is adequate and normal at rest; the blood pressures are unaltered; the cardiac output per minute is normal or slightly reduced (50) & (51). The velocity of blood flow in capillaries and veins is frequently normal; and the arterio-venous oxygen differences of arms and legs and lactic acid content of arterial
and venous blood are normal (49).

When the patient becomes dyspneic after mild exercise, the peripheral supply of blood to the tissues may still be adequate and the lactic acid formation in the exercising muscle may not be disturbed.

Factors that do show a change at this early stage are first, reduction of the vital capacity of the lungs (32) & (41). Second, blood flow through the lungs is retarded. Third, the total lung volume is normal or increased (6) & (36). Lastly, the residual air is increased and the reserve air decreases (36).

Conclusions to be drawn from the above findings are:—(a) In early stages of circulatory failure changes occur in the pulmonary circulation; (b) Changes in the pulmonary circulation may be independent of the larger circulation.

"In rheumatic, syphilitic, hypertensive and arterio-sclerotic heart disease it is in the pulmonary venous system that the first effect of the heart disease manifests itself. The back-pressure effect in cases of mitral disease and in cases of failure of the left ventricle is obvious. Its occurrence was claimed, long before the application of physiological concepts to clinical medicine, by morphologists of the seventeenth century. But the significance of this back pressure effect has not been examined in detail. If the left ventricle is not normally efficient and a certain amount of blood accumulates in the pulmonary vein, one or a combination of two possibilities may occur: (a) there being no essential alteration in the cross-sectional area of the vascular portion which offers the resistance to the right ventricle, the increase in the pulmonary venous pressure will result in a proportional increase in the pulmonary capillary pressure, and later, in the pulmonary arterial pressure; (b) a considerable increase in the cross section and volume of the vascular bed of the pulmonary circuit
may result without necessitating much alteration in pulmonary arterial pressure. Teleological and experimental evidence strongly supports the presence of the second factor in early stages of circulatory failure.

There are reserve capillaries in the lungs and all organs (15). Increase in the venous pressure is one of the most effective means by which reserve capillaries can be opened. In progressive heart failure Weiss (48) believes "the pulmonary arterioles widen and a reserve capillary bed opens in the lungs; and the mechanism of this process is governed by the same physiological laws as have been demonstrated in the peripheral circulation," i.e., increased venous pressure (55). "A progressive increase of the capillaries makes it possible to maintain a normal or approximately normal volume of blood flow through the lungs per unit of time without much added burden to the right side of the heart. It is probably this mechanism which makes it possible for the patient with heart disease to live fairly comfortably for many years."

The shape and consistency of the alveoli change due to the above and the alveoli stiffen (3½). This explains the increase of residual air and this is responsible for early decrease in vital capacity,—a functional emphysema.

"The increased blood volume in the lungs does not decrease the total air space but rather causes the diaphragm and bony thorax to assume a slightly inspiratory position, thus allowing space for the increased total blood volume." (48).

Loss of alveolar wall elasticity makes expiration difficult and thus adds to the subjective sensation of dyspnea,—a nervous communication between pulmonary system and the medulla.

The following mechanisms are visualized in the later stages of circulatory failure:

In congestive failure
1. Vital capacity and velocity of blood flow are diminished (9).
2. There is a decreased cardiac output.
3. The peripheral venous pressure rises.
4. Greater amounts of oxygen are utilized in the capillary blood.
5. Oxygen saturation of the arterial blood is reduced.
6. The physico-chemical changes occur in the blood,---increase in lactic acid, altered buffer capacity, changes in hydrogen-ion.

"A normal or decreased amount of blood may flow through the lungs under entirely different pressure relations within the pulmonary circuit. Thus the same minute volume of blood flow may represent different burdens for the right ventricle in different states of the circulation." (46)

"The slowing of the blood flow in the arterial system, if present, must be less than in the venous system."

(a) Although in a given time the total amount of blood flowing through a cross-sectional area of the larger arteries must be the same as that in the large veins; nevertheless, because of the independence and variety of the changes in the diameter of the cross-sectional areas in the venous and arterial systems, an abnormal deviation of the velocity of blood in the venous system may develop quite independently of any changes in the velocity of the arterial system;

(b) Changes in pressure in the arterial and venous systems and velocity are to a considerable extent independent. Therefore, with engorged veins, normal, lowered, or slightly elevated arterial pressure may exist.

The opening of new capillaries and increased oxygen utilization are perhaps consequences of the changes that follow venous engorgement. The capillaries of the patient with congestive heart failure
are always under abnormal pressure wherever the venous pressure is elevated. The velocity of the capillary blood flow is independent of the capillary pressure, and is to a certain extent determined by the functional capacity of the left ventricle and both have a fundamental relationship to cellular nutrition and function. High capillary pressure and a blood flow low in volume and velocity, as in numerous instances of congestive failure, are a combination seriously damaging to tissue functioning. It produces not only a "passive congestion" and an edema per se— but as a result of disturbances of the internal respiration between the capillary blood and tissue cells, specific substances, depending on the organs involved, appear in abnormal quantity in the blood stream. Ischemia and anoxemia, histamine, lactic and other acids play important roles. Circulatory damage to the liver will completely upset the water metabolism. Circulatory changes in the kidney and other organs will break the mechanism that maintains a constant acid-base equilibrium in the blood and tissues.

Weiss (48) applies this concept to the types of heart disease and concludes that it should aid diagnosis, prognosis and therapy.

The following cases are submitted as being rather typical ones from both the viewpoints of the clinician and the pathologist. Only such cases that have come to autopsy are used in order to illustrate points which have been made in the paper thus far.
Case I.

Case of G. J. R., hospital No. 35782, a white American janitor, entered the University of Nebraska hospital on the urology service July 29, 1931 and died at noon August 23, 1931. His complaints on entrance were: 1. Nocturia, 3 times; 2. Dribbling; 3. Frequency; 4. Painful urination.

Pre-entry findings are noted by referring physician: General health good; teeth poor, tonsile small; no murmurs in heart. Not only was his past history negative in regard to the heart but the positive notes in this regard are interesting:—Childhood diseases, measles and mumps; does not tire easily. No edema. This man's age is eighty.

Physical examination revealed patient in good physical condition. The left border of the heart was one finger outside the nipple line, no enlargement to the right demonstrable. Sounds clear, slight systolic murmur at apex, with the second sound accentuated. Blood pressure 165/95. Liver at costal margin.

Enlarged prostate, smooth.

On August 7, 1931 medical consultation was requested as the patient was coughing, was hoarse and was breathing rapidly. The next day the condition was worse with rapid and shallow respiration. Moist rales could be heard throughout the entire chest. Temperature is up some. Prognosis appears very bad,—is getting digitalis, force fluids and elimination. Does not complain of pain. Cough not bad. On August 9, the patient appeared to be improved and he felt better; but on the eleventh he was becoming irrational and the condition on the twenty-second is noted as growing steadily worse with the patient comatose. Digitalis was discontinued as the pulse was down and was fairly regular. He died suddenly at noon on the twenty-second.

Post-mortem findings in heart were:

Comment on Case I.

In the first series of three cases in which the heart has been implicated by the pathologist and not particularly by the clinician, a selection is made in order to theorize upon the probable mechanism of onset and progress which led to a cardiac death in the case.

Although the subject of this first case was not admitted to the hospital with any apparent signs or symptoms of cardiac involvement, it is suggested because of the poor teeth that he was the victim of endocardial changes from focus of infection. The patient may come under the class which Christian (11) points out "has neither the valve lesions of rheumatic heart disease nor the aortic lesions of syphilis". He states that the usual findings are an enlarged heart, enlarged valve orifices secondary to the dilatation of cardiac cavities and heart muscle well nourished, normal in color. Furthermore, narrowing of coronaries and diffuse fibrosis are very unusual findings; and in the clinical study of this class of patients nothing is found to fasten etiology on infections, no endocrinopathies. He states that the cases occur after the forty year age level.

The pulse pressure of the patient was high on entrance, showing that the left ventricle is raising a column of mercury 70 mm at each beat. The first sign of the cardiac failure probably was the coughing accompanied by the rapid breathing and indicates somewhat the dilated heart(39). There occurs venous congestion in the pulmonary circuit which may have been precipitated by the position of the patient on his back, but which is continued and made worse by the weakness of the cardiac engine to perform its work adequately. The immediate cause of death is not discernible.

Morison (38) believes back pressure begins in the capillaries but is hastened by cardiac failure which fails in the face of the peripheral obstruction to propulsion.
Case II.


Onset:—In March 1931 the patient began to notice that one-half to three-quarters of an hour after meals he had an aching gnawing pain in his stomach. Two months later it got so bad that he had to take sodium bicarbonate for relief. The pain continued gradually more severe. Noted black stools about two months ago and sour stomach. For three months he has noticed blurring and black spots before the eyes. He noticed that he became weaker, then nocturia and frequency began. The last four or five months he has had bad headache. He has had no diseases of significance. His teeth are poor and he has frequent colds but no sore throats or tonsillitis.


Family History:—Father died at 76 of heart trouble. Otherwise negative.
Case II continued.

Examination of the heart showed a loud diastolic murmur heard best at Erb's point. There were also murmurs heard at the apex and over the aortic area. Murmur was not transmitted to axilla. There is no thrill and no friction rub. A slight irregularity of rhythm was noticed. No pulse deficit present.

This patient gradually grew weaker and died November 18, 1931.

Postmortem findings:

The heart weighed 580 grams. There was a uniform hypertrophy of the left side and there was no evidence of scarring in the musculature. The coronary arteries were tortuous with small plaques of atheroma frequently. No evidence of gross insufficiency was noted.

The right lung weighed 1000 grams and the left lung weighed 920 grams. There was marked congestion of the bronchi with a bloody exudate.

Gross diagnoses were:

Cardiac hypertrophy and dilatation
Chronical passive congestion of the liver and lungs.
Arteriosclerosis generalized.
Chronic nephritis.
Ulcers of the stomach, healed.
Comment on Case II.

This man came into the hospital because of gastric ulcer and died a cardiac death. His is not such a well defined case as was Case I in lacking clinical signs because this man had blurring of vision black spots before the eyes, weakness and headache which might be attributed to a failing circulation or might have been attributed to kidney disease. The major signs of heart insufficiency are not present clinically, however. His teeth were poor and he has frequent colds, but these are found quite commonly in people who are free of heart complaints. The precordial pain takes on none of the significance of an angina pectoris. However, there is a loud diastolic murmur heard best at Erb's point, upon which the clinician would be likely to attach some significance. The patient also has pains in both knees at times which is worse during damp weather. These, of course, would point to a rheumatic fever, or at least to a rheumatoid condition which connotes an endocarditis.

Curiously enough, the autopsy findings did not support the concept of an endocarditis. The heart failure which was the cause of death would rather be explained as being due to exhaustion secondary to arterial hypertension and sclerosis. Hypertrophy is undoubtedly the result and not the cause of pathology, although the dilatation plays its part in a vicious cycle. This case would seem to be an example of the typical functional failure.
Case III

Q.R., male aged twenty-one, hospital No. 36322, entered the University hospital on September 20, 1931 with complaints of attacks of nausea and vomiting dating from an auto accident six months previously in which he had suffered some internal injuries.

Physical examination revealed the following:

Blood pressure 100/90. Tachycardia. Tenderness on palpation in the epigastrium on the right side. The edge of the liver extended below the costal margin. Patient also had a lid lag.

There is no swelling of the ankles but the patient states that he has shortness of breath during his attacks. Patient also says that when he gets an attack of the severe pain and he gets up and walks steadily, his pain leaves him.

The second sound over the heart cannot always be elicited. A systolic sound or murmur at the apex is transmitted to the pulmonic area.

Tentative diagnosis:-1. Mitral regurgitation with passive congestion of the liver. 2. Tachycardia.

Progress:

9-22-31. Patient had a severe attack of shortness of breath and vomiting. Caffeine sodio-benzoate was given and was followed by digitol. He was placed on tincture of digitalis minimums XXX t.i.d., hypodermically due to the vomiting.

10-5-31. Had attack similar to the above and after one-half hour the breathing became of the Cheyne-Stokes type. The vomitus was streaked with blood and the patient became unconscious.


The summary of the post-mortem findings may be stated briefly in regard to the heart:
Case III concluded.

Broken down mural thrombi found in each ventricle chamber. There were massive infarcts in the lung and a small one in one kidney. No point of origin of the infection was found.

The heart weight was 520 grams. There was a walled off purulent mass of matter in the tip of the right ventricle and a similar one in the tip of the left ventricle.

The diagnosis was made of cardiac decompensation and multiple infarcts.

Comment on Case III.

The case is a peculiar one throughout. The history points to the auto accident as the beginning of the pathology in the body. A mechanism such as follows may be visualized:—A ruptured pulmonary capillary, or even a larger vessel, allowed infection to pass into the blood stream from the lung tissues. The infection was almost overwhelming to the heart and although it was partially walled off in the tips of the ventricles, infected thrombi passed into the blood stream to the kidney. Massive infarcts in the lung may have been the site of the original injury or injuries.

Without any other damage to the heart muscle than the purulent mass in each ventricle, the case is said to be one of cardiac decompensation. Classification would be neither upon a structural or a functional basis but probably each had its part. The case is atypical and was selected because the symptoms seemed to point to a definite structural basis of pathology. The symptoms in all probability were on a structural basis, but the cause of death cannot be so easily placed.
Case Ia

Mrs. M.M., housewife age 43, hospital No. 33953, was admitted to the University hospital on January 3, 1931 with a diagnosis of Cardiac decompensation.

Pre-admission notes:-Best weight up until 1928 was 190 lbs. Present weight at admission was 250 lbs and patient was gaining. She had been ill since June 1929. Three years previously she had been in this hospital for ruptured ectopic pregnancy. No other previous illnesses. Present complaint:-Shortness of breath, swelling of legs. Present condition:-Resp. 22. Pulse 98. Edema legs. Urine scanty, cloudy, trace alb., sp. g. l.020, fair condition. Tentative diagnosis:-cardio-renal insufficiency.

Admission note(1-3-31):-Shortness of breath one year; swelling of feet, one year; cough, two weeks; dizziness on exertion; pain over heart; patient much worse the last two weeks. History of rheumatism and high blood pressure. Nocturia for last year.

Physical Examination:-Tremendous edema of legs (skin broken); edema seen in abdomen, arms and eyelids. Eyes react slowly to light. Slight discharge from nose. Throat injected. Chest:-fluid in both pleural cavities (flatness). Cardiac:-size not determined; regular. Abdomen markedly distended with fluid. Diagnosis:-cardiac decompensation, probably cardio-renal basis.

Progress notes:
1-3-'31:-Examination of patient fails to reveal any gross endo-cardiac pathology which in itself might account for the tremendous edema. Put on digitalis to slow pulse and because of diuretic action. Smith cardiac diet ordered. 1-5-'31:-Patient gives history of rheumatism infection two years ago which she states moved into various joints and kept her in bed for several weeks. She states further she has hypertension. Blood pressure now with decompensation 175/110. On account of tremen-
dous weight and edema, size of heart cannot be made out. There is a slight apical systolic blow and the tones are not strong. Urine is markedly diminished in amount and contains albumen and casts. Numerous moist rales both lung bases. Respiratory murmurs can be heard, particularly over left chest. More anteriorly, small moist rales with piping which with history of upper respiratory infection two weeks ago precipitating this illness would indicate low grade broncho-pneumonia process left. Diagnosis:-1. Hypertensive cardiac disease (possibility rheumatic infection) 2. Myocardial degeneration. 3. Extreme cardiac decompensation with general anasarca. 4. Albumin and casts in urine indicate a likely renal lesion probably also on a vascular basis. 5. Low grade broncho-pneumonia. 1-5-31:-Edema of extremities not quite so marked as yesterday but dyspnoea seems worse-Patient is losing. 1-6-31 (12:55 P.M.):-Found patient gasping for breath. No response to questioning. Died at 1:00 P.M.

Laboratory findings:-1-5-31:

Blood: Hb 90%, rbc 5,200,000, wbc 12,500, polymorphs 88%. Wassermann negative. Non-protein-nitrogen 61 mg per 100 cc.

Heart Autopsy findings:-

Weight 800 grams; heavy fat deposit in heart. Left ventricle greatly hypertrophied, wall 2½ cm. thick. Left ventricular cavity relatively smaller, being smaller than the right ventricular chamber. Some atheromatous degeneration in right descending coronary artery.

Summary of findings:

Comment on Case Ia

This second group of three cases is chosen to illustrate what may be expected at the autopsy table in patients who have typical cardiac failure signs and symptoms.

The first case is in that of a middle-aged woman who is very much overweight and gives a history of rheumatism and high blood pressure.

In the first place then, an enlarged heart would be expected in order to account for her ability to live with so high a blood pressure over a long period of time. The pulse pressure is not so very high, being only 65. The enlarged heart was found at autopsy and the left ventricle was hypertrophied.

In view of the rheumatic history, deformed valves would have been expected. No mention is made of them in the autopsy report. No anatomical changes were found to account for death from cardiac source.

Hypertrophy is never a cause of heart failure (24). Dilatation was not found here. The huge heart such as found here is to be expected usually in Bright's disease. It is huge due to the increased load. "The failure of these large hearts without lesions is perhaps the most striking proof that heart failure is a functional event" (24).
Case IIa

H. H., a trucking transfer man, age 53, hospital No. 35459, was admitted to the University hospital June 25, 1931 with the diagnosis of chronic myocarditis.

Previous history:—Two years previously patient began to have cough and dyspnea. Precordial pain began one year ago. He has not worked for two years. No previous illness except diphtheria at age 7.

Entrance notes:—Heart has gallop rhythm. Pushed to right, faint but regular. Blood pressure 110/60. Definite evidence fluid in left pleural cavity. Has bad nights. Controlled only by morphine gr $\frac{1}{4}$ last four nights. Has been on tr. digitalis gttae XX t.i.d. since July 6, 1931. Urine is red and loaded with wbc and casts.

Progress notes:

7-17-31:—x-ray and physical examination demonstrated fluid in the left chest. Chest tapped and 135 cc. of pale reddish clear fluid of sp.g. 1.018 was removed with immediate relief to patient. Heart has definite bundle lesion and is fibrillating. Tr. digitalis reduced to gttae L per day. Morphin sulphate q nite gr. $\frac{1}{4}$ to 1/6. Patient much better subjectively. Blood culture negative.

7-21-31:—Patient has had rigors with profuse cold clammy perspiration accompanied by pain in L.U.Q. the last two days. One very severe attack precipitated by climbing on a bed pan this morning. Only partly relieved by morphin gr $\frac{1}{4}$ hyp. Nitro-glycerin gr 1/100 seemed to give but slight subjective relief.

Blood count:—wbc 25,000 with 90% polys.
Pain suggests a new coronary infarct or probably a splenic infarct with peritoneal involvement.

7-22-31:—Patient was given digalen loc. hypo. at 11:00 P.M. and morph sulp. gr 3/8 hypo. at 8:30 last night. He was very cold with profuse sweating at 11:30. Respiration rapid about 60 and shallow suggesting
terminal pneumonia. Temperature about normal.

At 4:35 A.M. patient apparently conscious with rapid shallow respiration. Pulse now palpable. No sounds heard over the precordium. Caffeine Sod. Benz. gr ½ given hypo. with no response. At 5:25 A.M. loc. adrenalin given "extra cardiac"—four or five strong pulsations felt at wrist. Respiration became gasping. Patient more cyanotic and lividity occurred at 5 A.M.

Autopsy:—Very markedly enlarged heart and a definite coronary sclerosis. No infarcts in spleen and liver. Mitral valve and aortic somewhat adherent and showed signs of previous chronic inflammatory condition. Passive congestion of lungs and other visera.

Admission diagnosis, chronic myocarditis; Final diagnosis, coronary disease. Contributing, myocardial degeneration.

Comment on Case IIa

Symptoms and signs are fairly typical of those which have been described in heart patients who have a badly involved organ.

The heart pushed to the right in a patient receiving digitalis, and having a gallop rhythm and fibrillating would indicate a very badly infected heart.

The heart was very markedly enlarged at autopsy findings, but the basis for such enlargement is not clear from the history given. It might be thought to be compensatory to the evidences of chronic inflammation and coronary sclerosis which were found.

Because of the disturbed rhythm and the anatomical changes found, the venous congestion evidenced by the pulmonary edema and dyspnea may be ascribed to the failure of the cardiac pump to adequately propel the blood through the pulmonary circulation. Death may have been on a myocardial degeneration basis with arrhythmia and failure.
Case IIIa

Case of J.N., hospital No. 35746, farm laborer, age 63, entered the University hospital July 25, 1931 complaining of: 1. Shortness of breath. 2. General edema. 3. Difficulty in walking.

Onset and Development:—Five months ago the patient fell from a horse and bruised the testicles. One week later swelling of the ankles and wrists was noted. At first the swelling was worse at nights and would disappear in the mornings. Swelling became progressively worse and one month later the legs became edematous and stiff; edema continued upward into the abdomen and thorax. The shortness of breath has been worse the past two weeks. Puffiness of the eye lids and face has been noticed the past two months. This is worse in the mornings.

The patient was under the doctor's care and was given diuretics and digitalis but he would not stay in bed. The condition is much worse the past three weeks. The past four days the patient has had palpitation. There is no past history of any disease.

Heart examination:—Sounds are faint and indistinct. Rate 100. Rhythm irregular. Blood pressure 150/90. No murmurs heard. No other findings.

Diagnosis:—Myocarditis with possible secondary kidney involvement.

Progress:—On July 28, laboratory tests showed no albumen in the urine with specific gravity of 1.023 and 10 to 15 red blood cells per high power field. Non-protein nitrogen was 21.3 mg per cent. The patient seemed improved although the sounds were faint and the pulse was irregular and rapid. He was put on the Karrel diet with digitalis, magnesium sulphate and restricted fluids.

On July 30, rales were noted in both lung bases and he died on this date.

The post-mortem findings in the heart were:—150 cc of amber-colored fluid in the pericardium; heart weight, 230 grams; scarred myocardium;
Case IIIa concluded.

enlarged orifices; absence of muscular hypertrophy; coronary orifices show slight decrease in size; evidence of hypertensive nephritis in both kidneys. Other findings are judged to have been negative.

Cause of death:—Chronic myocarditis with fibrosis of the liver contributing.

Discussion of the case:

The case would be placed in an intermediate position between the ones illustrating typical findings at autopsy without any previous indications of cardiac involvement, and those with typical symptoms and signs of heart failure but with no indications of heart pathology at autopsy. The complaints point to the heart for the most part with the puffy eyelids which bothered mostly in the mornings may be due to the kidney inefficiency. There is apparently no basis for an endocarditis here and the etiology for a myocarditis is just as obscure. The heart found at autopsy is very small at 230 grams and may be partially explained by the short duration of the symptoms and signs. There were no murmurs but there were enlarged orifices. The presence of these two things together is not the rule. There was no muscle hypertrophy of the heart so that, with the small weight, the heart walls must have been very thin. The slight decrease in size of the coronary orifices is probably a senile change associated with some hypertensive atherosclerotic in the other blood vessels of the body.

The diagnosis of a chronic myocarditis was presumably made upon the findings noted, "myocardium shows scarring". The findings are certainly not those to have been expected in a seriously affected heart being the cause of death.
Summary and Conclusions:

In reviewing the literature along the lines of the mechanisms involved in cardiac failure, one is easily impressed by the large amount of writing and thought that has been expended upon the questions. The amount of actual research work done affording proofs of theories of mechanisms seems to be rather scanty and inadequate, however. By the review of means by which symptoms of cardiac failure occur, it is hoped that points suggestive of the possible mechanisms in heart failure are set forth. Many contradictions have been stated and indicated, and many have been stated but not indicated.

According to the personal experiences of the present writer in regard to cardiac malfunction, it seems probable that muscular tone plays a big part. It is my belief that the cardiac muscle depends as much for its proper functioning upon the optimum tone of its fibers as do the skeletal muscles. In this regard Allbutt (1) states that dilatation is not significant if it merely means loss of tone and not textural dissolution, but tonicity may maintain an extremely decayed ventricle in position. Parsons-Smith (40) also refers to this possibility. On the same basis too, more interest is aroused to Sir James MacKenzie's attitude in emphasizing the subjective feelings of the patient. Clendening (14) mentions this too, in the use of digitalis.

It would be interesting to know more about the part played by the respiratory cycle in relation to the circulation. Practically everything inside the body and outside in nature, moves in cycles; oddly enough, little or no progress is made unless cycles are broken.

The modern view of cardiac failure appears to be somewhat as follows:

Neither the back pressure nor the "via a tergo" theories are entirely satisfactory. (20) "The primary factor in cardiac failure is the heart muscle". The myocardial view would be correct altogether
if heart failure were always a gradual process in which the contraction of the heart became weaker and weaker, uninfluenced by any extraneous factor. Usually heart failure is precipitated by such a factor and the heart suddenly becomes inefficient, and fails to deal with the blood that is returned to it. Muscle fibers stretch beyond optimum point, heart dilates, pressure rises behind the heart. The back-pressure theory, though wrong in its essentials, was right in ascribing venous engorgement to a failure of the right ventricle to pass on the blood, and pulmonary congestion to a failure of the left. When the cardiac failure affects one side more than the other, it will be the circulation immediately behind the side which is primarily affected that will first show signs of engorgement. (20)

In cardiac failure circulation is slowed. The slowing may precede signs. Edema appears when the slowing has reached a certain point. The venous pressure is often high, i.e., the heart is unable to cope with venous blood returning to it. The capillary stream is slowed. (20)

"The essential disturbance in the heart is a lack of perfect balance between the two sides of the heart, especially between the right and left ventricles." (43 & 35).

The above conclusions are to be drawn from the most recent literature on the subject of cardiac failure.

Finis
Bibliography

15. Black, H. L., and Neilson, J. Jr., History of Cardiology.
17. Black, H. L., and Neilson, J. Jr., History of Cardiology.
23. Black, H. L., and Neilson, J. Jr., History of Cardiology.
25. Black, H. L., and Neilson, J. Jr., History of Cardiology.
27. Black, H. L., and Neilson, J. Jr., History of Cardiology.
29. Black, H. L., and Neilson, J. Jr., History of Cardiology.
30. Black, H. L., and Neilson, J. Jr., History of Cardiology.
31. Black, H. L., and Neilson, J. Jr., History of Cardiology.
46. Singer, Chas., The Discovery of the Circulation of the Blood.
49. Weiss, Soma, and Ellis, L. B., Heart Disease and Peripheral Circulation, Jr. Clinical Invest.
52. White, Heart Disease, Chap. XXIX, Congestive Heart Failure.