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The Dynamics of congestive heart failure

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THE DYNAMICS OF CONGESTIVE HEART FAILURE

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INTRODUCTION

It is impossible thoughtfully to survey, in the light of daily experience, the field of medical work covering diseases of the heart, varied as the manifestations may be, without realizing the central problem to be failure of the heart to accomplish its work in lesser or greater degree. This work consists in the propulsion of blood through the circle of vessels in adequate quantity to meet the needs of the body in the ordinary and varied circumstances of life. The very essence of cardiovascular practice is recognition of early heart failure and discrimination between the different grades of failure. This simple truth is not stated here for the first time; in theory it receives occasional homage from many. It emerges into view for a fleeting moment, to retreat and lie concealed beneath a mass of technical, and by comparison trivial, detail; it does not dominate cardiac practice as it should. When a patient seeks advice and heart disease is suspected, or is known to be present, two questions are of chief importance. Firstly, has the heart the capacity to do the work demanded of it when the body is at rest? Secondly, what is the condition of the heart's reserves? These questions can

be answered, and correctly answered, in almost all cases by simple interrogations and by bedside signs; and the answers force all other considerations into the background in most cases of chronic heart disease; they are essential to sound prognosis and treatment.

Sir Thomas Lewis (47).

HISTORY

The early history of cardiology is one of logic and sound reasoning but very little experimental investigation. The concepts of the physiology of the heart and blood vessels were based almost entirely on anatomy, but the key to the anatomic solution of the circulation, the capillary system, was yet undiscovered. Until Harvey conceived of the blood traveling in a circle, several rather elaborate theories were attempted to explain the facts then known.

The practice of eviceration in embalming their dead (31) at least exposed the ancient Egyptians to the human anatomy, and they did recognize the heart as the center of the vascular system. Though they had no inkling of the circulation, they realized the relation of the heart beat and the pulse, and stated that the motion of the heart can be detected by placing the fingers on the head, the hands, the arms, or the legs. (17)

The objective investigation of nature, led by Hippocrates and Aristotle, flourished in the intellectual atmosphere of ancient Greece. Aristotle's conception of the laws of energy and physiology were correct in many respects, and his reasoning entirely

logical. The modern technique of measurement has shown up his errors, but the facts available he systematized most logically. The liquid blood is contained within vessels which extend to every organ in the body. In one organ only is the blood found outside the vessels. That is the heart, whose cavities open directly into the vessels themselves. Therefore the heart must be the "source and fountain of the blood, and its prime receptacle." The dominant idea in the physiology and physics of Aristotle was that heat was the source of all energy, being the immediate cause of all chemical and organic activity. This "fire" was not always manifested as warmth, but could convert matter from solid to liquid to vaporous form, etc. This idea of a single energy manifesting itself in various forms is still the foundation of our physical sciences. Aristotle believed that the products of digestion were absorbed from the intestine into the blood vessels to be mingled with the blood; and, that after passing into the vascular system, they received their complete transformation or sanguification in the cavity of the heart and thence passed to the tissues, but there is no evidence that he imagined anything like a physical movement of the blood. To Aristotle the lungs

were a cooling system which regulated the temperature of the blood, but the "channels" reaching both the right and left cavities of the heart (the pulmonary artery and the pulmonary vein) served to receive from the lungs the "spiritus" which was incorporated into the blood. Seeing the expansion and pressure of boiling liquids, he attributed the pulsation of the heart and vessels to the expansion of the heated blood. (16a)

Parxagoras, the anatomist of greatest repute in the period immediately following Aristotle, was probably the first to differentiate between the arteries and veins; not only was pulsation confined to the arteries, but these vessels contained air and not blood, and he accordingly named them arteries or air tubes. (16b)

Accepting the findings of Parxagoras and the theory of Aristotle, Erasistratus and his contemporary, Herophilus, elaborated them from anatomical discoveries of their own. Sensing for the first time the importance of the brain and nerves, Erasistratus proposed a second pneuma or spirit. The spiritus vitalis, derived from the inspired air and transmitted by the arteries, gave the immediate stimulus to the functions of organic life, but the spiritus-animalis was

elaborated in the brain and transmitted through the nerves, thereby giving rise to the perceptions of sight, smell, hearing, taste, and touch; and by influx into the muscles, it caused their contraction by expanding them laterally and shortening their longitudinal dimension.

For five hundred years the arteries were considered bloodless, the spurting of a cut artery being considered leakage into it from the veins after the aeriform spirits were discharged from the wounded vessel. First to doubt and disprove this theory was Galen, the leading physician of Rome. By a variety of experiments he conclusively proved the true nature of the arterial contents. In Galen's system of physiology the blood was manufactured in the liver from the products of digestion carried in by the portal veins. The blood was then distributed throughout the body via the vena cava and to the lungs by way of the right heart. A second circulation contained the thinner, brighter colored blood which derived the vital spirits from the lungs. The tissues were, then, supplied with two systems and nourished by two kinds of blood. The heart's movement was for suction...."Thus the heart dilates, to attract the necessary materials, remains fixed while using what it has drawn into it,

and contracts when discharging its superfluities."(16c) The pulsation of the arteries was for the same purpose, but the dissemination of the nutritive material was accomplished mainly by a physiological force by which each tissue attracted, like a magnet, those ingredients which it needed. This material could travel in both directions in the arteries and veins as it was needed. The doctrine of anastomosis advanced by Erasistratus he accepted because of its experimental proof...."If you take an animal like the ox, ass, horse, sheep, etc., in which the veins and arteries are large and easily recognizable, and open several of the principal arteries, you will evacuate from them the whole of the blood. We have often performed this experiment, and always finding the veins empty as well as the arteries, we are convinced that the dogma of their mutual anastomosis is true." Galen's belief in the anastomosis in the lung and perforations in the septum of the ventricles was based on indirect evidence which has now been proven erroneous, although his idea of anastomosis was correct.

The next name of importance in connection with the subject of circulation did not appear for over a thousand years. In the fourteenth century Mondini Da Luzzi of the University of Bologna explained the

heavy wall of the left ventricle was to balance the heavier blood of the right. Two centuries later Berengario Da Carpi, also of Bologna, published a treatise on anatomy. This work, like that of Mondini, was mainly a manual for dissections which were occasionally made to demonstrate the writings of Galen.

Using the strictly unorthodox method of the human body as his text and Galen as his supplement, Vesalius, in the sixteenth century, published his own anatomy. Beautifully and correctly illustrated, well indexed, and systematized, it often disagreed with Galen who dissected mainly the higher animals. Vesalius altered the theory of the circulation but little. From his dissections he found no direct evidence of communication through the ventricular septum, and so doubted the passage of blood through such perforations. He believed all the arterial blood came from the right heart but could not account for its passage.

This was accounted for by Servetus, who lived at the same time as Vesalius. Servetus occupied himself mainly with questions of controversial theology, but his book expressing his views was denounced by both Protestant and Catholic authorities;

so he withdrew from this field and studied medicine in Paris under the assumed name of Michel Villeneuve. His views on the circulation were like those of his contemporaries, but he believed the arteries were filled by way of the lungs. "This communication does not take place through the median wall of the heart, as commonly believed, but, by a grand device, the refined blood is driven from the right ventricle of the heart, in a long course, through the lungs. By the lungs it is prepared, assuming a bright color, and from the vena arteriosa is transferred into the arteria venosa." As evidence of this fact he offers the varied conjunction and communication of the vena arteriosa with the arteria venosa in the lungs, the remarkable size of the vena arteriosa unnecessarily large for simply nourishing the lungs, and the foramen ovale, which Galen showed did not close until the hour of birth, and "consequently, it is for another purpose that the blood is poured from the heart into the lungs at the hour of birth and in such abundance." Servetus' doctrine had little opportunity to gain much fame as it was given an obscure page in a secretly published book which was destroyed, along with its author, as soon as it was discovered.

Though the doctrine of Servetus produced no

comment, the theory of the circulation was importantly affected during these years by the work done in Italy. Colombia, the successor to the chair of Vesalius, was convinced that the left heart was supplied with blood by way of the pulmonary artery and vein and not through the septum. He believed the vitalization of the blood occurred mainly in the lungs, the left ventricle adding only a 'last touch'. The main evidence for this new thought was the absence of any pulsation in the pulmonary vein. The pulsation of the arteries was acknowledged to proceed from the heart, and to accompany the dissemination of the vital spirits. If this one vessel alone did not pulsate, the most natural explanation was that the blood in it was flowing to the heart, not out of the heart.

Did the lungs add a vital substance to the blood or only cool it? How were the left ventricle and arteries supplied with blood? These two problems produced considerable speculation and many answers.

Leonardo Botalli, finding several cases of persistent foramen ovale, thought he had discovered the true route of the blood. Varolius believed the material for the generation of the arterial blood was taken from the intestine directly to the heart through the mesentric arteries. Umeau conceived the spleen as the

filter for the passage of venous blood into the arteries. Among the earliest to admit the doctrine of the transit of blood through the lungs was Andreas Caesalpinus. He still thought only a part went through the lungs for the purpose of refrigeration, and the rest was transmitted through the septum. (16d)

The physiology of the veins was added to by Hieronymus Fabricus by his discovery of the valves of the venous system in 1574. He correctly conceived how they strengthen the veins, and enable them to bear a pressure which might otherwise cause their dilitation. "For in the case of varices, where the ostiola are either relaxed or ruptured, we always see the veins more or less dilated."

The flow of blood was believed to be against the valves, but the blood seeped through as it did at the cardiac valves. The importance of this new anatomical fact was the link it offered Fabricus' pupil Harvey in his chain of evidence for the true circulation of the blood.

In the year 1628 William Harvey published a small quarto of seventy-two pages in which he proposed an entirely new theory of the physiology of the cardio vascular system. In the introduction and first chapter he reviews the current opinions and points out

the inconsistencies of the various theories together with the reasons which induced him to write this work. The second chapter is on the character of the heart's motion, as shown by vivisections. In this chapter he advances his first great argument, with the experimental proof, that the heart expels its blood during systole, and receives it at the time of its collapse or relaxation. The next chapter treats of the character of the arterial motion, as shown by vivisection. He shows that on arteriotomy the blood is ejected during ventricular systole and arterial diastole. Hence the pulsation of the arteries is from the movement of the blood, just the reverse of the accepted theories. The next chapter deals with the auricles. Their function he proved by cutting off the tips of the ventricles and observing the escape of blood with each auricular pulsation. He then describes the heart cycle, and how the blood is expelled by the ventricles into the arteries and from the right heart to the left. The next chapter is concerned with the proposition that the blood is carried from the right heart to the left by transudation through the parenchyma of the lungs. Since he could find no anatomical evidence of anastomosis in the lungs, he thought it

passed through the porosities.

The final arc in the circuit became inevitable from the calculations on the amount of blood passing through the heart from the veins to the arteries. The argument till now was over the details of how the veins supplied the arteries, but now an entirely new idea had to be coped with, for even by the most conservative experimental measurement, the heart output far exceeded the amount used up. Such a rapid flow would, in a short time, inevitably empty the veins and overwhelm the arteries with blood. There was only one condition in which it could be possible, that is, in case the blood were to pass again from the arteries to the veins, and so return to the right side of the heart. (12)

With the knowledge that the blood traveled in a circle and returned to the heart to be expelled again, the dynamics of the circulation in congestive heart failure could be investigated. The first to speculate on this problem was Corvisart, Napoleon's physician. He realized that venous back pressure played an important role in the manifestations of cardiac failure when he wrote, "It is possible that, in the diseases of the heart, the difficulty of breathing proceeds entirely from the mechanical compression

of the lungs, by the enlargement of the heart, or the evolution of an aneurismal tumor; this is true in some cases, but in a greater number, the difficulty of respiration appears to belong solely to the accumulation of the blood in the vascular system of the lungs, from the embarrassment which it suffers on returning into the cavities of the heart, deranged wholly or partly in their natural organization." (36a)

This conception was developed into an organized theory by James Hope. His views are so in accord with the modern work described in this paper that they could well be used as a conclusion rather than an historical introduction.

So long as the left ventricle expelled its contents the auricle escaped distension. When the ventricle ceased to expell its contents, the inflow was obstructed, and the auricle labored against increased pressure and so distended. The obstruction might be propagated backwards through the lungs to the right heart, and the same phenomena would ensue. He stressed the valvular lesions as a cause of obstruction, but he also noted the impediment could be elsewhere, as in the lungs. From the following quotations it can be seen that Hope realized that a weak heart muscle was a cause of obstruction as well as faulty valves.

"So long as the left ventricle is capable of propelling its contents, the corresponding auricle, being protected by its valve, remains secure. Hence, in a large proportion of cases, the auricle is perfectly exempt from disease, while the ventricle is even enormously thickened and dilated. But when the distending pressure of the blood preponderates over the power of the ventricle, its contents, from not being duly expelled, constitute an obstacle to the transmission of the auricular blood. Hence, the auricle becomes over-distended, and the obstruction may be propagated backwards through the lungs to the right side of the heart, and there occasion the same series of phenomena. When the obstruction thus becomes universal, as is frequently the case, it may either happen that all the cavities are thickened, or those only which, from their conformation, have the greatest predisposition to it.

"When the mitral orifice is contracted especially if the aperture be very small, the left ventricle, being insufficiently supplied with blood, is not stimulated to its ordinary contractile action, and consequently becomes emaciated and occasionally flaccid or softened. Meanwhile, the left auricle, having to struggle against the contracted valve in front, and

also to sustain the distending pressure of blood flowing in from the lungs, invariably becomes thickened and dilated. The engorgement, extending backwards through the lungs to the right ventricle, often occasions its hypertrophy and dilation; under which circumstances, namely hypertrophy of the right ventricle and contraction of the mitral valve, the lungs suffer in a preeminent degree; for, being exposed to the augmented impulsive power of the right ventricle behind, and incapable of unloading themselves on account of the straightened orifice in front, their delicate and ill-supported vessels are strained beyond the power of resistance. If, therefore, they cannot disgorge themselves sufficiently by a copious secretion of water and mucus, they effuse blood by transudation into the air-vesicles and tubes, and form the disease denominated as pulmonary apoplexy. I have found this affection to occur more frequently under the circumstances described, namely great contraction of the mitral valve, with, or even without, hypertrophy and dilation of the right ventricle, than under any other.

"When the mitral orifice is permanently patescent, so that, at each ventricular contraction, blood regurgitates into the auricle, this cavity

suffers in a remarkable degree, for it is not only gorged with the blood which it cannot transmit, but, in addition, sustains the pressure of the ventricular contraction. Permanent patescence of the mitral orifice constitutes an obstruction on the left side of the heart; and the effect of this, as of contraction of the orifice, may be propagated backwards to the right side. The regurgitation is always considerable when it renders the pulse small and weak.

"When the impediment to the circulation is primitively seated in the lungs, the right ventricle, situated immediately behind them, is the first to experience its influence; and when the cavity is so far overpowered by the distending pressure of the blood as to be incapable of adequately expelling its contents, the obstruction extends to the auricle--the process being exactly the same as that which I have described above, in reference to the left ventricle and auricle." (36b)

The importance of the power of the cardiac muscle, rather than the valvular resistance or regurgitation, was stressed by William Stokes shortly after the publications of Hope.

The emphasis was then shifted from increased

pressure in the venous system to a decreased cardiac output. This idea was developed by Sir James Mackenzie. He explained the symptoms of heart failure on the deficient blood supply to the various organs and on deficient pressure at the terminal vessels which allowed stagnation of the blood. (36c)

The experimental investigations of recent years have done much to clear this controversy and bring about agreement. Such writers as Parsons-Smith, who previously rejected the back pressure idea (53), are now reconsidering its worth. (52)

DEFINITION OF TERMS

Heart Failure

Since not all men are in agreement as to what the heart fails to do in heart failure, their definitions of the term do not always agree.

Sir James Mackenzie (48) wrote, "Heart failure may be defined as the condition in which the heart is unable to maintain an efficient circulation when called upon to meet the efforts necessary to the daily life of the individual."

Similarly, Sir Thomas Lewis (47) said, "There is but one meaning to the term cardiac failure - it signifies inability of the heart to discharge its contents adequately."

Meakins and Long (36d) were specific as to what the inefficiency of the circulation was and in what way the heart's discharge was inadequate when they wrote, "Circulatory failure may be defined as a state in which the volume of blood circulated per unit of time is not adequate for the physical needs of the moment."

Compensation and Decompensation

The terms "compensation" and "decompensation"

are apt to be more confusing than enlightening. Mackenzie (48) mentioned them only to denounce their use. Harrison (36e) used decompensation as a rather unsatisfactory synonym for congestive heart failure, and compensation, likewise, for the absence of the congestive phenomenon. Sir Thomas Lewis (47), tracing the term compensation back for a hundred years, found it was first used to indicate the recovery by hypertrophy of that power which the heart lost by way of dilatation. Since it has been shown that dilatation of the heart increases its capacity for work, this meaning must be discarded. Compensation has come to mean the disappearance of venous congestion, while decompensation or broken compensation is used synonymously with venous congestion. Lewis denounces the use of the word in this sense as being pernicious to precise thinking.

Wiggers (60a) uses the term compensation for the increased power of contraction by the heart in response to increased venous pressure or increased aortic resistance. By decompensation he means congestive failure and a decrease in the power of contraction in response to increased venous pressure or increased aortic resistance as indicated by a lower pressure summit and a decrease in the length of systole.

If the term decompensation means venous congestion and also inability of the heart to respond by increased power, how does the term apply to those cases in which there is evidence of venous congestion and yet the heart is capable of still further response? It seems to me that the word is really being used in three separate senses. It has come to mean congestive cardiac failure, a decrease in cardiac reserve, and finally it has been used to describe complete exhaustion of the cardiac reserve, a condition in which the heart responds to increased venous pressure and increased aortic resistance by not only a failure to contract more powerfully but by an actual decrease in its force of contraction.

MECHANISM OF ADAPTATION TO VENOUS RETURN, ARTERIAL PRESSURE, AND HEART RATE

When the minute output of the heart equals the minute volume returned, the circulation is in a state of balance. To maintain this balance the heart must adjust itself to alterations in venous return, arterial pressure, and heart rate. In a closed circulation a state of unbalance for any length of time is not conceivable, and such an unbalance must result in death.

A consideration of the physical ways in which such adaptations of the heart obtains is of first importance since they constitute, in short, the mechanism of normal compensation, quantitatively known as the heart's reserve. Any reduction in the heart's ability to respond to changes in venous return, arterial pressure, and heart rate would be a degree of heart failure. A consideration of the mechanism of heart failure can most logically be approached, therefore, by studying first the normal heart's response to the three conditions named.

Effects of Increased Venous Return

It is generally accepted as demonstrated that a decreased return of blood to the heart results in a

reduction of ventricular discharge.(42, 39, 63) There has been no such agreement as to whether increased venous return automatically produces a larger discharge. Y. Henderson and his associates believed that no such increase takes place when the auricular pressure exceeds a critical level of about 50 mm. of saline, and as normally the arterial pressure is maintained at or near this level, an increased ventricular output was an impossibility. He explained the circulatory reserve on a basis of hemoglobin reserve and oxygen debt, denying a cardiac reserve.(42, 39, 41, 43) The 30 per cent utilization of blood oxygen at rest rises to about 80 per cent in severe exercise; (32a) so it does constitute at least a part of the circulatory reserve. In favor of a cardiac reserve is a great deal of work showing that the minute volume output is increased as the venous pressure is raised, the heart responding to venous pressures up to maximums of 250 to 300 mm. of saline. (59a)

The mechanisms through which a greater discharge becomes possible have been clearly established. This has been done by a careful study of the volume and pressure curves in the auricle, ventricle, and aorta of both the right and left heart through the cardiac $\overset{OUT}{\wedge}$

cycle. Knowing the pressure and volume at each instant the phases of the cardiac cycle which give the blood its increased velocity can be found. Most of the work has been in agreement except for the question of initial pressure in the ventricles. Patterson, Piper, and Starling (54) published tracings which indicated that the initial pressure does not rise consistently but often falls. Straub (62a) agreed that there was no increase in initial tension but never observed an actual fall. Wiggers and Katz, using very sensitive manometers, carefully reinvestigated the work under better controlled conditions and found, without exception, an increase in initial tension.(62b)

Diastole. A study of the separate phases of filling in diastole shows that each attains an increased velocity, but its time is never prolonged. The greater rate of inflow during the early diastolic inflow phase is due to the greater venous pressure occuring at the moment of opening of the A-V valves; the distension of the auricle causes a more forceful auricular systole with a more rapid inflow, and in slowly beating hearts having a diastasis phase of some duration, there is an additional increase in the rate of filling during diastasis as well. (59b) A high intra-auricular pressure, though it does not

prolong the period of diastole, does prolong the interval of diastolic inflow, since the higher the pressure level in the auricle, the earlier it exceeds the pressure in the ventricle, and the sooner the period of inflow begins. This is especially effective if the gradient of ventricular relaxation is steep. (62c)

Systole. The pressure and volume curves of the systolic phase as recorded by Patterson, Piper, and Starling, as well as those of Straub, were not the same as the optical records obtained by Wiggers and Katz. Since the latter's work seems superior, it will be discussed. They showed that the increased systolic discharge was accomplished in part through an increase in velocity of ejection and in part by a prolongation of systole. In both the right and left heart the gradient of primary rise of pressure was steeper, the pressure maximum higher, the phase of isometric contraction abbreviated, and the ejection phase prolonged. (62a, 61)

Initial Tension. The disagreement over the initial tension of the ventricles on increased venous return has caused Wiggers and Starling to disagree about the fundamental cause of the heart's increased reaction to distension. Starling, finding

no increased tension, but greater length of the muscular fibres of the heart wall, believed that the energy of contraction is a function of the length of the muscle fibres. This generalization is commonly called Starling's 'Law of the Heart'.(55) Wiggers, observing an increased initial tension, concluded that either the initial tension per se is responsible for the increased response or that it secondarily produces changes in initial length which, in turn, are responsible for the greater reaction. Wiggers is inclined to believe that changes in initial length, rather than initial tension, fundamentally determine the vigor of response. A critical experiment in which initial tension could be increased and initial length decreased or visa versa would decide the question, but this has never been satisfactorily done. Whichever is fundamentally responsible, it still remains that under normal conditions alterations in initial length are chiefly, if not solely, produced by previous changes in initial tension. (62d)

The conception of the heart thus far is that it is in itself unable to modify its output, being entirely dependent on its diastolic size and initial tension. X-ray studies on intact animals, however,

have demonstrated that the systolic output of the heart is not always proportional to the diastolic size, but the completeness with which it empties may vary. Nicolai and Zuntz were the first to demonstrate this increased systolic emptying. They could not account for the increased output during exercise by the measurable increase in diastolic size, and so decided it must come from a more complete emptying during systole. They have pointed out that normally the heart does not empty itself completely, and its ability to do so is a part of the cardiac reserve. (32b) Bardeen has calculated from X-ray measurements that during rest only about 60 per cent of the ventricular blood is ejected per beat. (3) Meek and Eyster found that after exercise the diastolic size of the heart in some cases was increased but in others decreased, the latter maintaining output by more thorough emptying. (51) Burns and Roemer (32c) found that the diastolic size was consistently decreased in some during exercise, and consistently increased in others, while in a third group the size varied from time to time. These changes were in no way correlated with the accompanying changes in pulse and blood pressure, and so they

concluded that factors other than mechanical conditions in the circulatory system determine the degree of relaxation and contraction of the heart muscle.(49)

The Effects of Increased Aortic Resistance

If the heart is in good condition the arterial pressure may be raised to the highest limits possible in the body, and still the systolic discharge will not be reduced, but may even increase slightly. (54, 65, 62e) Studies of volume and pressure curves have shown that as the aortic resistance is raised, the discharge is lessened for a few beats. This residual blood added to the normal inflow causes an increase in the diastolic volume and an elevation of initial tension. The heart now responds in accordance to the principle of Starling, and the discharge is maintained.

The next question is: Does the increased systemic aortic resistance affect the pulmonary arterial pressure, or do both ventricles work under an increased burden? Those working with heart-lung preparations find the pulmonary pressure raised (23, 62a), but most investigators using intact animals have failed to show that the diastolic pulmonary pressure is altered significantly. (62e) These last results seem

the most accurate.

The greater response and lengthening of systole have been entirely attributed by Straub to a secondary increase in initial tension, (62e) by Patterson, Piper, and Starling as the primary effects of afterloading, but Wiggers attributes them directly to the increased initial tension. (62e)

Pressure changes in the left auricle have been carefully studied and increases are very slight. This means that backing up of the blood or stasis is very minimal. (62f)

The reactions of the right ventricle have been variously interpreted. As shown, the pulmonary resistance is not raised so alterations in this ventricle must be due entirely to changes in the return of venous blood. Here again the results from heart-lung preparations do not agree with those of intact circulation which Katz and Wiggers believe superior. (62g) In these experiments systolic output is not directly reflected in increased venous return because it is believed that after sudden compression of the aorta or pronounced constriction of the peripheral vessels, a certain volume of blood is probably captured in the splanchnic vessels and withdrawn from active circulation. Since there is a

slightly increased auricular pressure, some immediate compensation for this must take place. Though no measurable increase in initial tension accompanies the small increase in right auricular pressure, there is undoubtedly a slight augmentation of the systolic discharge which accounts for the slight elevation of systolic pressure observed in the pulmonary artery.

The Effects of Changes in Heart Rate

The systolic discharge is easily adapted to changes in heart rate. When the heart slows, the diastolic period of filling is prolonged, giving a greater systolic discharge (62h, 42, 39, 54, 65). When the heart accelerates, the reverse is true. The mechanics of this system were first clearly analysed by Y. Henderson (42, 39, 41, 43). Since most of the filling occurs early in diastole, the filling and subsequent emptying of the heart is not reduced as long as this early period is not shortened. But when the heart beats faster than 60 or 80 per minute, this period of rapid inflow is abridged, diastolic filling is incomplete, and the systolic discharge is proportionally reduced.

However, this is not the only mechanism involved, for the smaller volume curves of the rapid rates are

not superimposable on the larger ones of slower rates, though Y. Henderson has maintained that they are (42, 39, 41, 43). Wiggers, Katz, and others (59c, 54, 65, 62h) have found that the velocity, amplitude, and duration of ventricular contraction are modified by the changes in diastolic size, initial tension, diastolic load, and in some cases by alterations in cardiac nutrition as well. With moderate slowing, the two ventricles react alike, though one ventricle to a greater degree. The greater filling of the two hearts gives a greater discharge and pulse pressure; this tends to persist in the right heart where pulmonary systolic pressure is maintained, but the systolic pressure of the left heart is soon lowered by a decrease in peripheral resistance. Systolic and diastolic systemic pressures both decline progressively until a stabilized level is reached. The effects of greater initial tension resulting from an increased filling of the heart still maintain, however, so that the period of systole is still prolonged, and the gradient of the isometric pressure rise remains steep. Too great a decline in arterial diastolic pressure would impair the heart's nutrition so that systole would be impaired no matter what other conditions prevailed. The decline of venous return

from too great slowing would impair the heart's output as described earlier.

An increase in heart rate above 50 to 150 per minute does not increase aortic and pulmonary pressures, but they begin to decrease. One of the reasons for this is the abridgment of the effective filling phases, and the small diastolic size means a less vigorous contraction even though the initial tension is increased. At very rapid rates the isometric phase of systole is prolonged, the amplitude of the ejection phase is less, and the time of ejection is shortened. This reduced discharge gives a lower systolic pressure and pulse pressure. (62h)

Although an increased pulse rate may be essential for a great increase in cardiac output and also essential for optimal work by the heart for more moderate increases in cardiac output, marked changes in pulse rate may occur without appreciable changes in output. "Such acceleration of the heart rate is to be considered as a call via nervous impulses for an accelerated cardiac output which is unanswered by the heart because other adjustments (dilatation of the arterioles or capillaries, increase in venous return, etc.) are not forthcoming. An accelerated pulse rate may thus be either an indication of an

actual increase in the cardiac output or of a demand for such an increase. And a diminished pulse rate indicates that the cardiac output is decreasing or that stimuli compatible with such a decrease are activating the pulse regulating mechanism." Grollman(32a)

SUMMARY OF CONSECUTIVE EVENTS IN BOTH VENTRICLES*

ON INCREASED VENOUS RETURN

Greater auricular distension causes--
- (a) Larger auricular contraction--higher pressure summit, and
- (b) Greater filling pressure at moment when A-V valves open.

Increased ventricular filling takes place--
- (a) During phases of rapid inflow, diastasis, and auricular systole.
- (b) Due to increased velocity of inflow, without increase in diastolic filling time.

Increased diastolic size and higher initial tension responsible--

1. In pressure curves, for--
   - (a) Steeper gradient of contraction.
   - (b) Higher pressure maximum.
   - (c) Prolonged duration of systolic ejection and total systole, and
   - (d) Slight abbreviation of isometric contraction phase.
2. In systolic discharge, for--
   - (a) Increased volume of discharge.
   - (b) Increased velocity of discharge, and
   - (c) Increased duration of discharge.

INCREASED ARTERIAL RESISTANCE

Left Ventricle

First Stage

Systolic discharge, reduced.
Pressure maximum, increased.
Systolic residual volume, slightly greater.
Systolic ejection and systole, abbreviated.

Second Stage

Diastolic size and initial tension, increased.
Isometric rise, steeper.
Pressure maximum, higher.
Systolic ejection and systole, lengthened again to or beyond normal.
Systolic discharge, equal to or exceeding normal.

*summary from (62i)

Left Auricle

First Stage. -- No change.
Second Stage. -- Pressure, slightly increased.

Pulmonary Artery

Diastolic pressure, unaltered.
Systolic pressure, slightly increased.
Pulse pressure, slightly larger.

Right Auricle

1. Inflow, reduced due to diminished return via inferior vena cava.
2. Inflow, increased due to--
   (a) augmented flow through collateral circuits, and
   (b) reduced vascular capacity.
3. Net result, auricular pressure, slightly increased.

Right Ventricle

Diastolic size, greater.
Initial tension, very slightly increased.
Isometric rise, slightly steeper.
Pressure maximum, higher.
Systolic discharge, slightly increased.

CARDIAC SLOWING

A. When Diastolic Pressure Is Kept Reasonably Constant And Nutrition of Heart Is Ample

Auricles and Ventricles

1. Diastolic pause, lengthened and filling pressure, raised.
2. Ventricular filling, more complete.
3. Initial tension and diastolic size, increased.
4. Isometric contraction, steeper and briefer.
5. Pressure maximum, higher.
6. Ejection phase, prolonged.
7. Systolic discharge, greater.
8. Velocity of discharge, increased.

Pulmonary and Systemic Arteries

Diastolic pressure, decreased.
Systolic pressure, increased.
Pulse pressure, larger.

B. When Diastolic Pressures Fall Naturally, But Nutrition Is Not Impaired

Auricles and Ventricles

Changes as above, except pressure-maximum may become lower than normal in left ventricle.

Pulmonary and Systemic Arteries

Changes as above, except systolic and diastolic pressures, decreased in aorta.
Pulse pressure, increased.

C. When Nutrition of Heart Is Impaired

Auricles and Ventricles

1. Diastolic size and initial tension, markedly increased.
2. Isometric contraction, more gradual and longer.
3. Pressure maximum, lower.
4. Systolic ejection phase, shortened.
5. Systolic discharge, smaller.

Pulmonary and Systemic Arteries

Systolic and diastolic pressures, greatly lowered.
Pulse pressures, decreased.

THEORIES OF THE DYNAMICS OF CONGESTIVE HEART FAILURE

Disease of the heart muscle and heart valves, progressive coronary sclerosis, excessive overwork as chronic hypertension, etc., is adjusted for by the natural compensatory mechanisms just described. An adequate peripheral circulation is maintained and no symptoms are complained of at rest, although the capacity for exercise is diminished. This stage of circulation is called compensation with diminished cardiac reserve. When the evidence of venous engorgement, cyanosis, dyspnea at rest, and edema appear, the patient is said to be suffering from congestive heart failure. The dynamics of the circulation have changed to such an extent that they produce these symptoms. The dynamic factors involved in the circulation are the output of the heart per minute in relation to the metabolic needs, the pressure in the arteries in relation to the pressure necessary to force the blood to and through the capillaries, the velocity of the blood flow, and the venous pressure.

Until recent years satisfactory methods for measuring these phenomena had not been found. And even much of the recent work has been shown to be inaccurate. Without these necessary facts at hand the theories of

congestive heart failure were based on assumptions which modern research has in some respects failed to confirm.

THE "FORWARD-FAILURE" HYPOTHESIS OF INADEQUATE CARDIAC OUTPUT

This hypothesis has been brought forward and developed mainly by the clinicians and physiologists of the English speaking countries. Harrison (36d) has selected quotations from these men which suggest they believed that inadequate cardiac output was at least the principal cause of the clinical phenomena of congestive failure. Sir James Mackenzie, who was one of the chief proponents of this hypothesis, stated: "The symptoms of heart failure from deficient output of blood might be found in almost any organ did we possess the means of observing them. It so happens that one system which suffers early from an impaired blood supply is one which readily gives rise to distress. This is the respiratory system, and it is the distress in breathing on reponse to effort which is usually the earliest sign of heart failure. As the heart failure proceeds the distress in breathing becomes more easily provoked until a stage is reached when it is present even when the body is at rest."

Sir Thomas Lewis is quoted: "Breathlessness is to be ascribed to a deficiency in the flow of aerated

blood to the head and neck; at first the deficiency is confined to those exercises in which normally the cardiac output is much above resting values; at last there is a deficiency in the physiological quantity of aerated blood expelled by the heart while the body is at rest.... It is when the output at rest declines that blood begins to collect on the venous side and the patient begins to manifest signs of congested veins and, associated with these, enlargement of the liver, cyanosis, a high-colored and scanty urine, ascites, dropsy of the lower members, and congestion and oedema of the lungs." Meakins and Davies likewise said, "We are pursuing further work on this question, but we would suggest that cardiac failure of this character is due to an incomplete ventricular systole as a consequence of which the circulation rate is greatly and progressively diminished until the amount of circulating blood is grossly insufficient to carry on the functions of the heart, kidneys, nervous system, and other important organs." Similarly, Meakins and Long said, "Circulatory failure may be defined as a state in which the volume of blood circulated per unit of time is not adequate for the physical needs of the moment." Means stated, "The fundamental fault responsible for cardiac dyspnea

is obviously to be found not in the nature of the blood but the rate at which it is pumped in the heart itself.... The important point is that the heart either because of increased work, fatigue, or degeneration is unable to maintain an adequate rate of blood flow." Stewart and Cohn stated, "The volume output of blood per minute from the heart which is in failure is diminished and its size larger than when it is in a state of decompensation."

THE "BACKWARD-FAILURE" THEORY

The backward-failure theory has been supported mostly by the clinicians of France and Germany.(36f) Briefly, it is summarized as follows: Damage and overwork lead to dilatation of the heart, which reduces the cardiac reserve and necessitates the reduced activity of a symptomatic early heart failure. If dilatation becomes extreme, the venous pressure is raised in order to dilate the heart during diastole. The increased venous pressure leads to congestion of the organs drained by these veins. Thus, disease primarily of the left ventricle causes an increased pressure in the pulmonary veins. Because pulmonary arterioles have little or no "tone," the resistance in the pulmonary arteries is increased. The right heart then dilates to the point where the venous pressure in the systemic veins is raised with venous congestion, engorgement of the abdominal viscera, edema, and, in some cases, ascites and hydrothorax.

The two theories are built on these basic differences. According to the "back-pressure" idea the essential phenomenon is an alteration in intracapillary pressure; whereas the "forward-failure" thesis postulates a change in volume flow as the

outstanding feature.

Wiggers (60b) has interpreted the work of Mackenzie, Lewis, Meakins, Means, Cohn, Starling, Y. Henderson, etc., somewhat differently than Harrison. In the opinion of Wiggers, the Anglo-American clinicians and physiologists did not deny that venous pressure and congestion were important in accounting for the clinical symptoms, but merely stressed the importance of decreased cardiac output.

Wiggers stresses the importance of the disproportion between arterial pressure and peripheral resistance brought about by the venous congestion of congestive failure. The peripheral resistance is increased by the elevated pressures beyond the arterioles, and therefore a greater pressure in the arterioles is required to maintain a rate and volume of flow equivalent to normal. Ordinary left ventricular contractions with the ejection of ordinary or normal volume outputs will not maintain this greater pressure; supernormal systolic volumes are needed, and though the normal minute volumes are ejected, the systolic and diastolic pressures are inevitably reduced if the stroke volumes are reduced. In short, venous congestion necessitates a super circulation with an abnormally high blood pressure to prevent

slowing of the capillary circulation. If this is true, it seems to me that increased venous pressure is still the most important factor, although some of its effects, as in this regard, may be indirect rather than directly local mechanical effects in the capillaries.

THE DYNAMIC FACTORS

CARDIAC OUTPUT

Methods

The great importance of cardiac output determinations in studies on circulation has led to an extensive study of this subject by many methods, but, as Grollman has shown (32d), most of the methods have been quite inaccurate in normal subjects and even more inaccurate in cardiac cases. Harrison (36g), comparing the data on patients with congestive failure studied by these older methods, found that the cardiac output per minute was less than that of normal persons by the same method in about one-half of the cases, was within normal limits in approximately one-fourth of the patients, and was greater than normal in the remainder.

The more recent methods, especially the acetylene method, have proven quite accurate, but even here special measures in technique must be used to prevent serious errors (32d, 34).

The direct Fick method has not been used extensively on human patients because of the dangers involved in puncturing the heart. Using this method, Lauter (36g) found diminished cardiac outputs

in three patients with congestive failure, but found a similarly low value in a patient with complete heart block and without any complaints referable to the heart.

Hamilton, Moore, Kinsman, and Spurling (35) injected vital red into a vein and then plotted the concentration curve of successive samples of arterial blood. From this curve they were able to calculate the cardiac output, the volume of blood in the heart and lungs, and other circulatory functions. Their method checked satisfactorily with the direct Fick procedure and with the known values in artificial systems. Studying a large number of patients with cardiac disease, they concluded that the most characteristic change in patients with congestive failure was a small cardiac output per beat in relation to the amount of intrathoracic blood. Little correlation was found between the clinical condition and the cardiac output per minute. Patients with severe congestive failure sometimes had larger outputs per minute than persons with well-compensated cardiac disease.

Knowing the oxygen content of the blood as it leaves the lungs (arterial blood) and the oxygen content of the blood as it enters the lungs (mixed

venous blood), plus the total amount of oxygen taken in by the lungs per minute, the amount of oxygen absorbed by the body per minute, and the amount of oxygen taken up by each unit of blood as it passes through the lungs may be determined. Knowing these two facts, the cardiac output may be calculated according to Fick's principle. Fick's principle may be applied as well if the corresponding figures for carbon dioxide are known. (11) Using a procedure first described by Plesch, Burwell, and Robinson were able to obtain the "mixed venous blood" from a peripheral artery. This was done by rebreathing a gas mixture until it was in equilibrium with the venous blood. This method has been used on patients and proven accurate even in lungs containing fluid, but is difficult and uncomfortable to use. (27)

The most generally useful method for determining the cardiac output of healthy persons is Grollman's acetylene procedure; with the proper precautions, it has been proven accurate in congestive failure cases also (34). Further proof of its accuracy is the agreement of values by this method with values obtained by the venous plateau method described above, the two being entirely different

in principle.

Cardiac Output in Heart Failure

Harrison, Friedman, Clark, and Resnik (37) studied the cardiac output of twenty-seven patients with heart disease without symptoms of congestive failure while at rest and nineteen cases with frank congestive failure; eighteen patients without cardiac disease were studied as controls. The cases studied represented three common etiologic groups--vascular, rheumatic, and syphilitic. Wide fluctuations in output were found within each group, but there was no evidence of any correlation between this function and the underlying processes. The eight patients of the compensated group who had auricular fibrillation had somewhat smaller outputs than the other compensated cases, but this difference was not striking. But those cases of auricular fibrillation in the decompensated group had outputs equal to the decompensated cases without fibrillation.

Comparison of the cardiac output per minute per square meter was not of consistent significance. The controls as a rule had higher values than those with heart disease, but there was considerable overlapping,

four of the controls having lower outputs than the average output per unit of body surface of the decompensated patients. Of this series seven of the compensated cases and seven of the decompensated cases had outputs per square meter above the lower normal limit set by Grollman at 1.9 liters.

No significant changes in cardiac output or arteriovenous difference was observed following clinical improvement. These functions sometimes increased and sometimes decreased. However, in more than one-half of the patients, clinical improvement was associated with an increase of ten per cent or more in cardiac output per beat and with a decrease in the metabolic rate.

Cardiac Output Following Various Therapeutic Measures

Friedman, Resnik, Clark, and Harrison studied cardiac output and its related functions on patients before and after the use of such therapeutic measures as rest, digitalis, diuretic drugs as salyrgan and theophyline, and after venesection. (28, 30) They found that clinical improvement caused by rest alone was usually accompanied by diminished minute output, while no consistent change in oxygen utilization was noted. Output following digitalis increased, decreased

and remained the same among the various patients. Diuretics acted similarly. Venesection diminished the output for several hours as a rule. (29)

THE VELOCITY OF BLOOD FLOW

The velocity of the blood flow has been of concern to physiologists since the first conception of the circulation. Harvey himself attempted to calculate the heart output, but he made no calculation as to the velocity of blood flow. Stephen Hales in 1733, by timing the capillary circulation and from measurement of the heart volume and cross section area of the aorta, computed the velocity of the blood mathematically. (4) Many instruments have been designed to measure the velocity, but the method which best meets all the requirements is the one elaborated by Blumgart and Yens (5) whereby radium C, the active deposit of radium, is injected into the antecubital vein and the time of arrival in the other arm is determined by a suitable detecting device.

Using this method, Blumgart and Weiss (8) have studied the circulation time of a series of patients with cardio-vascular disease. Venous pressure and vital capacity measurements were also made on these patients, which makes the study particularly valuable. They found the pulmonary circulation time ranged from 5 to 17 seconds with an average of 10.8 seconds. This time is not affected by the respiration, systemic blood pressure, venous pressure, or vital

capacity of the lungs. A small but definite increase in velocity was noted with an increased ventricular rate, although this relation was not found in every instance. (7)

The patients with rheumatic valvular heart disease who were well compensated and of regular rhythm had generally normal velocities of blood flow, vital capacities, and venous pressures. With early symptoms of cardiac insufficiency, the vital capacity was reduced and the velocity of blood flow was definitely retarded, but the venous pressure was frequently still within normal limits. With the appearance of congestive failure, all three were still further decreased from normal. Those patients with auricular fibrillation had a still slower velocity of blood flow.

The patients with syphilitic heart disease still maintained relatively rapid peripheral blood flow even after the pulmonary circulation began to slow. This has been taken as evidence of early failure of the left ventricle in aortic insufficiency while the right heart is still capable of receiving all the systemic venous blood. With the lesion at the mitral orifice the right heart is strained, and the stasis in the pulmonary circulation is soon felt in the

general circulation.

The decrease in vital capacity and the slowing in blood flow occur at about the same time in the development of circulatory insufficiency, but definitely precede the rise in peripheral venous pressure. (6) Drinker, Peabody, and Blumgart (18) found that the lungs act as a slightly elastic sponge, and are able to take up a vast amount of blood without significant change in pressure. The ease of distension of the veins allow them to be filled with blood to the limit of their capacity with but very small increases in venous pressure. Once they are filled to their limit, however, the relative inelasticity of these tubes prevent further expansion, and the rise of venous pressure will be rapid. (6) This, in all probability, is what occurs in the lungs, and it explains why slowing of the pulmonary blood flow occurs so early in circulatory failure, even before the clinical signs appear.

The functional elasticity of veins is subject to great individual variations, but it markedly decreases with age. Though the veins of older subjects show decreased extensibility, this is probably not due to a change in the stretchability of the vein wall, but is due in part, at least, to the more rapid escape

of fluid from the veins when the pressure within them is increased. This, in turn, has been accounted for by smaller tissue fluid volume and lower tissue fluid pressure in old age. (13)

Other similar methods of measuring the circulation time depend on the color, vasodilator effect, neuromuscular stimulation, or taste for the detection of the substance injected. By the use of sodium dehydrocholate, Tarr, et al. (24, 56) found the circulation time of cardiac patients without evidence of congestive failure only two seconds above the average for normal adults, while the general average for decompensated patients was twenty-six seconds—twice the average for normal adults.

Like Blumgart and Weiss (4) they found no constant relationship between the blood pressure, venous pressure, pulse rates, or weight of the patient and the circulation time. Anemia tended to increase the velocity of the blood, while polycythemia tended to slow it.

By the use of the arm to tongue circulation time studied by decholin, and systemic venous blood pressures measured by the direct method, Wood (66) investigated a series of 93 cases of heart disease and decided that if pulmonary congestion could be

assumed from a slowed pulmonary circulation time and systemic venous congestion from a raised venous pressure, then right and left ventricular failure may occur separately, the former being seen most strikingly when secondary to emphysema and the latter in hypertension and aortic incompetence.

VENOUS PRESSURE

The pressure exerted by the heart upon the columns of blood in the arterial system progressively decreases due partly to the degradation of pressure energy into heat, and partly to its conversion into kinetic energy of flow. In the smaller vessels the first is a dominant factor; in the larger vessels the latter predominates, the loss of energy due to friction being small. (60c) The drop in pressure in the arteries is slow, the diastolic pressure being maintained with little loss up to the division of the smaller arteries into arterioles. A pronounced decrease in gradient occurs in the arterioles(68>32 mm. Hg), with a second abrupt decrease in the capillaries (32>12 mm. Hg). There is a more gradual decline in the venules and veins until a negative pressure is actually reached in the central veins.

A second factor of great significance has been

proposed by Y. Henderson (44, 40) and his associates. The arterioles, capillaries, and venules are enmeshed by the muscle fibres of the muscular organs. Because of their tonus, these fibres, like the fibres of a stretched rope, will constrict these vessels. Sufficient blood pressure must be maintained to overcome this resistance until the vessel emerges from the muscle. This pressure is then available as the pressure found in the collecting veins. This is not the same action as that produced by the contraction of a muscle which squeezes or pumps the blood toward the right heart. The intra muscular tension for the biceps is about 47 mm. of water in the lying position and 74 mm. of water in the standing position. It is considerably increased by the inhalation of CO2 which acts in part directly upon the muscles themselves and in part upon the spinal centers of the motor nerves. They cite numerous observations in support of this theory, and repeated Riml's experiment as important evidence. By means of this experiment it can be shown that after all heart action has ceased the tonic elasticity of the tissues is sufficient to squeeze a considerable part of the blood in the body out through the veins under a pressure corresponding to a column of blood of 10 or 12 cm.

In addition to this extra vascular support by tonic tissue pressure, there is evidence that the venules and veins are under nervous control.(25, 26, 45, 50) Although not proven, this nervous mechanism may be of some importance in preventing a drop in venous pressure following generalized vasodilation.

Henderson also emphasizes the importance of the negative pressure in the thorax in determining venous return, but this has been strongly questioned. (21)

The control of blood flow through the muscle during exercise is determined chiefly by the direct action of its metabolites. Contraction of the muscle arrests the flow of blood, but on relaxation the flow increases considerably above normal and only gradually returns to the resting level. This has been definitely attributed to the liberation of a vasodilator substance which may prove to be histamine.(1, 2)

It is generally agreed that the capillary pressure is transmitted on to the veins, and is a considerable if not the principal vis-a-tergo to the venous blood. It does not follow, however, that changes in the capillary pressure will necessarily be reflected in the venous pressure. This independence of the venous pressure from the capillary pressure is indicated by a number of observations. Hooker (46) found the

venous pressure in the hand uninfluenced by local changes in vascular tone or by changes in pulse rate. White (58) found considerable change in the capillary pressure by warming and cooling the skin, but this did not alter the local venous pressure. No correlation between the capillary pressure and the venous pressure was found in patients showing the "Raynoud Phenomenon" as exhibited by high capillary pressures and local stagnation of the peripheral circulation. In extreme cases the veins were much reduced in calibre, but the pressure was unchanged. (10) Low peripheral vasomotor tone seen in acute febrile conditions and manifested by low diastolic blood pressure, high pulse pressure, macroscopic capillary pulse, auscultory sounds over the peripheral arteries, low capillary loops in the nail bed as observed directly through the microscope. (23, 20a) It is my belief that these observations demonstrate that increased capillary pressure or increased blood transmitted to the collecting veins of a local area is compensated for by an increased rate of drainage, thereby preventing a rise in pressure. This increased rate would be necessary only to the nearest large calibre vein which, being easily distended by very small pressure, could easily

accommodate for it. In other words, the venous system acts like a reservoir in which the pressure of the inlet stream is dissipated as long as the outlet offers no resistance. If the low peripheral vasomotor tone of the acute febrile conditions is a generalized manifestation, then the venous return of all the organs will be increased, and venous pressure will rise if the drainage of the larger veins is not speeded up. The mechanism for expelling such an increase in venous return without an increased venous pressure is more than adequate in the normal heart as explained in an earlier chapter. A generalized vaso dilatation by the use of vaso dilators such as the nitrites does cause an increased cardiac output, but the prolonged administration of the drug causes excessive vaso dilatation which, like shock, prevents a further increase in output. (32e) On the other hand, vasoconstriction with adrenalin and vasodilatation with amyl nitrite does alter the venous pressure. (22)

Under the basal conditions of rest for at least fifteen minutes and in the recumbent or semi-recumbent position, the venous pressure in normal individuals, as measured in a superficial vein, is usually found

to be between 40 and 60 mms. of water. (20b) The "critical level" of venous pressure in decompensation has been established by Clark (14) at 200 mms. of water; a rise above this level indicating further failure, while a drop is of favorable prognostic significance. There is usually a direct relationship between the venous pressure and the clinical course, though not in the actual heighth of the pressure so much as its trend. (20c) It is becoming quite well established that no condition but heart failure and mechanical obstruction of the veins will cause a rise in the general venous pressure. Chronic heart disease, paroxysmal tachycardia and fibrillation, endocarditis, arterial hypertension, and pneumonia show no rise in pressure until the heart begins to fail. (20d, 57, 38, 19) Little correlation can be made between venous pressure and age, respiratory rate, holding of the breath at intervals, systolic arterial tension, arterial pulse pressure, surface area, electrocardiographic findings, or X-ray cardiac findings. (15)

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