The Weight of the Heart and Its Chambers in Hypertensive Cardiovascular Disease with and without Failure

By Russell S. Jones, M.D.

The myocardial weights of the different cardiac chambers were determined in 130 cases of hypertensive cardiovascular disease, both with and without failure. The left ventricle hypertrophies before and continues to hypertrophy after the advent of congestive failure. The right ventricle and both auricles hypertrophy only after failure develops. In correlating the ventricular work with its muscle mass, the left ventricle apparently performs twice as much work per gram as the right ventricle. Several possible explanations for this apparent greater efficiency of the left ventricle are presented. The changes in chamber weights are discussed in relation to concepts of congestive failure.

More is known of the physiology of the heart than of any other organ, primarily because of the knowledge of mechanical principles and the development of the sphygmomanometer, roentgenography, electrocardiography, and cardiac catheterization. It is, therefore, surprising that a further means of understanding cardiac function has been neglected, that is, the determination of the different chamber weights. From such information the degree and sequence of hypertrophy of the different chambers might be established. Accurate methods for determining the weights of the myocardium of the different chambers were devised by Müller in 1883, Wideröe in 1911, and Lewis in 1912. Lewis, basing his study upon 23 controls, 46 cases of valvular disease and 27 of "nephritis," observed that (a) elevated blood pressure was the most potent factor in producing preponderance of the left ventricle, (b) general and uniform hypertrophy of both ventricles is frequent in renal disease and (c) the ventricles are heavier in mild nephritis than with marked contraction of the kidneys. He advised that "before we can consider our knowledge of hypertrophy in relation to valvular or renal disease to be complete, the number of hypertrophied hearts studied must be multiplied." This has not been done, although there are correlations of electrocardiographic axis-deviation with ventricular weights and of pulmonary diseases with weights of the chambers.

It is the purpose of this paper to: (a) present a simple and reliable technic for determining myocardial hypertrophy of the different chambers, (b) indicate the degree and sequence of hypertrophy of the different cardiac chambers in compensated and decompensated hypertensive cardiovascular disease, (c) correlate these changes with concepts of congestive failure and (d) compare the work per gram of myocardium of the two ventricles.

Methods and Materials

Technic of Dissecting the Heart

After routine weighing of fresh or formalin-fixed hearts, the myocardium is freed of epicardial fat, extramyocardial coronary arteries, cardiac valves and the attached portions of aorta, pulmonary artery, pulmonary veins, venae cavae and any tissue or mass of any appreciable weight which does not represent myocardium. This leaves only the myocardium of the cardiac chambers. The chambers are then divided into four segments: the left ventricle, the interventricular septum, the right ventricle, and the two auricles. The illustrations (fig. 1) demonstrate how this dissection may be performed most easily. The right ventricle is detached from the interventricular septum by cutting through the ventricular wall close to the septum and in a plane parallel to the septum. One follows closely along the interventricular wall at the pulmonary valve and finally, the right ventricular wall is completely freed.

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by detaching it from the tricuspid ring. The left ventricular wall is removed in a similar manner:

incisions are made close to and parallel to the septum and as perpendicular as possible to the overlying epicardial surface (so that the inner endocardialized portion of the septum equals the thickness of its outer sectioned portion); the sectioning is extended across the base of aortic valve and then the lateral wall of left ventricle is completely detached by removal from the left auricle at the mitral ring. The interventricular septum is the only remaining myocardium attached to the auricles, aorta and pulmonary artery. The septum is freed by cutting through its membranous portion at the base of aortic and pulmonary valves. This leaves the combined auricles to be detached from aorta and pulmonary artery, their valves, portions of parietal pericardium, epicardial fat and as much as possible of the fat in the outer part of the interauricular septum. The four segments of myocardium are now accurately weighed, the sum of their weights representing the "true" myocardial weight.

Discussion of the technic. It is believed that the above technic affords a favorable balance between simplicity and accuracy. Since this technic, like previous ones, differs in various aspects, some explanation for the selection of the above methods of dividing each of the different myocardial segments should be given. After initial studies it was observed that the auricles were difficult to divide accurately at the interauricular septum and that they are usually of equal weight. Therefore, in subsequent dissections the two auricles were left attached and weighed as a single segment. Division of the septum into its left and right ventricular components cannot be carried out with ease or accuracy. Initial studies of dividing the septum by serial transverse sections, then separating each of the sections at the juncture of "scroll" muscles indicated that the ratio of the right to left ventricular septal components equalled that of the nonseptal components, that is, 1 to 2. Accurate separation of the left ventricular wall from the septum is obviously difficult, since the septal wall of the left ventricular wall is concave. Nevertheless, there was an unexpected uniformity and equality of the interventricular septum to the left ventricular lateral wall weights.

The lateral wall of the right ventricle can be most accurately determined by this method. Its detachment from the relatively flat interventricular septum and adjoining structures is readily accomplished, a few portions of trabeculae carneae representing the only points of uncertainty in sectioning. The right ventricle is also usually free of infarction or fibrosis but may show some infiltration of adipose tissue.

Besides the above technical errors there are other sources of inaccuracy. These are related to the presence in the myocardium of collagen, amyloid, fat, edema fluid, "swelling of ground substance," and leukocytes. Considerable fluid drains from a sectioned heart over a period of several hours. Although the reduction in weight through fluid loss is probably of the same degree in all parts of the myocardium, a 20 per cent loss can occur over a 12-hour period. Considerable increase in weight is possible from
thrombi or blood clots clinging to the heart or from fluid clinging to the numerous crevices and surfaces in a recently washed heart.

The weights of the ventricular walls are of prime importance in this study and are accurate within the technical limitation of this method.

Classification of Material

Six hundred eight unselected hearts of children and adults were studied. Of this total group 110 patients were free of cardiovascular disease, emaciation, significant anemia, chronic pulmonary disease, and renal disease and were utilized as controls. Eighty-five of these were adults. One hundred thirty hearts were from patients with hypertensive cardiovascular disease and form the basis of this report. Eight cases, grouped separately for comparative purposes, had either fluctuating hypertension or a history of hypertension but none recently. Two hundred ninety cases were grouped in various other categories such as thoracic disease, congenital anomalies, valvular lesions, coronary thromboses and mixed. The remaining 70 cases had inadequate clinical records and were discarded.

For comparative purposes cases of fluctuating hypertension and cases with previous hypertension but none prior to death are listed, while the cases with hypertensive cardiovascular disease are subdivided as follows:

1. Compensated: (a) Death from noncardiovascular causes. (b) Death from cerebrovascular accidents. (c) Previous cerebrovascular accidents then death from noncardiovascular caused. (d) Death in uremia.

2. Decompensated: (a) For a few weeks. (b) Over a few weeks and up to one year. (c) From one to three years. (d) Over three years.

The diagnosis of hypertension was based upon a "fixed" diastolic blood pressure over 100 mm. and a systolic pressure over 150 mm. Hg prior to failure in patients without significant cardiac valvular lesions, anemia, chronic pulmonary changes, or other diseases which might give rise to cardiac hypertrophy. The diagnosis of cardiac failure was based upon the symptoms of orthopnea, paroxysmal dyspnea, and exertional dyspnea, and the signs of pulmonary edema, hepatomegaly, ascites, and peripheral edema.

It was not possible accurately to distinguish left ventricle failure alone from that combined with right ventricular failure. Therefore, "decompensation" was used to denote irrefutable evidences of failure as indicated by combinations of the above clinical findings. The onset of hypertension usually was uncertain; therefore, no classification in regard to the duration of hypertension was deemed valid. The existence of essential hypertension was substantiated by microscopic evidence of visceral arteriosclerosis.

Observations

Control (Normal) Values

In the adult the myocardial weight after removal of fat, great vessels and clots is approximately 200 Gm. The relative weights of the different chambers are quite uniform and closely approximate the easily recalled ratios of 2:2:1:1 for the left ventricular lateral wall (L.V.), the interventricular septum (Sept.), the right ventricular lateral wall (R.V.), and both auricles (Aur.), respectively. The relationships of the chamber weights in the adult may be presented by comparing the actual weights, the percentages, and the numerical ratios as follows:

<table>
<thead>
<tr>
<th>Myocardial Wt</th>
<th>L. V.</th>
<th>Sept.</th>
<th>R. V.</th>
<th>Aur.</th>
<th>L/R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grams</td>
<td>200</td>
<td>66</td>
<td>33</td>
<td>34</td>
<td>2.0</td>
</tr>
<tr>
<td>Per cent</td>
<td>33.3</td>
<td>33.3</td>
<td>16</td>
<td>17</td>
<td>2.0</td>
</tr>
<tr>
<td>Ratio</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
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</tbody>
</table>

Early interest in concentric and eccentric hypertrophy of the heart was reflected in the emphasis placed upon the left to right ventricular (L/R) ratio. In Lewis's controls this ratio ranged from 1.57 to 2.18, averaging 1.8. Lewis's dissection resulted in a relatively small septal bulk which was not considered in the calculation of the ratio. Müller divided the septal weight by calculation into right and left portions and added these to the respective right and left ventricular weights to arrive at a right to left (R/L) ventricular ratio of .552. Lewis's L/R and Müller's R/L ratios are almost identical reciprocals of one another. The L/R ratio obtained by Hermann and Wilson in 16 controls averaged 1.74. They divided the formalin-fixed heart at the juncture of the scroll muscles in the septum and thus avoided any separate septal weight.

In the present study the estimation of the ventricular ratio is based, like Müller's, upon the calculation of the weight of the left and right ventricular portions of the septum. In a preliminary group of 15 hearts it was found that the ratio of the right to left septal portions was approximately the same as the ratio of the left and right lateral walls. Estimation of the
L/R value could be based upon the ratio of the sums of the septal plus lateral wall portions of the respective ventricles:

\[
\frac{L}{R} = \frac{\frac{L.V.}{R.V.} + \frac{L.V.}{L.V.} \times \text{Sept.}}{\frac{R.V.}{R.V.} + \frac{L.V.}{L.V.} \times \text{Sept.}}
\]

The resulting L/R value actually would be the same as if the lateral wall values alone had been used. Since the weight of right lateral wall value is determined with the greatest accuracy and the septum and left lateral ventricular wall, in contrast, are difficult to divide, it was decided to average the weights of the septum and left lateral wall and then divide by the right lateral ventricular weight:

\[
\frac{L}{R} = \frac{\text{Sept.} + L.V.}{2R.V.}
\]

This calculation, however, fails to consider the changing ratio of right and left septal portions in hypertrophy. For example, a twofold right ventricular hypertrophy should give an L/R value of 1.0 but by the last formula the L/R would be 1.17. A twofold increase in the left ventricular weight should have an L/R of 4.0 but by the formula yields 3.67. By graphically charting a number of such relationships the following corrected value, \(\left(\frac{L}{R}\right)_c\), is derived.

\[
\left(\frac{L}{R}\right)_c = 1.19 \frac{L}{R} - 0.37
\]

The L/R of 2.0 used in this report is a convenient approximation, the actual average for the adult controls being 1.92 with a range of 1.36 to 2.43. These extreme ranges are unusual but are utilized for completeness since control series in previous reports included cases with similar diseases. The few low L/R ratios occurred in the patients with metastatic carcinoma and a low "true" myocardial weight but without marked weight loss. The high ratio of 2.43 occurred in a patient dying from traumatic injuries but also having portal cirrhosis. The need for further study of the cardiac function in different diseases is suggested by this variation of the L/R ratio and total myocardial weight.

The L/R values alone cannot indicate the entire relationship of ventricular weights. If both ventricles were hypertrophied or atrophied to the same degree the ratio would be the same as before. To give a more complete picture the various chamber weights may be tabulated, and graphically charted or the L/R value may be supplemented by the ratio of observed to expected myocardial weights. In the latter method, comparison to the expected weights would give a ratio of 4:2 instead of the usual 2:1 (or 2.0). In this paper these data have been tabulated (table 1) as well as graphically charted (fig. 2).

**Values in Hypertensive Cardiovascular Disease**

A complete analysis of the sequence and degrees of hypertrophy in the different cardiac chambers has not been possible for two major reasons: (a) the onset of hypertension is rarely known, hence, the effect of duration of hypertension upon degree of left ventricular hypertrophy cannot be evaluated; (b) clear and unequivocal evidences of left ventricular failure without right ventricular failure are usually not obtainable from the clinical or hospital records, hence, "decompensation" in this paper is applied to all cases with definite evidences of failure. However, the data presented in table 1 and figure 2 do indicate the effect of failure as well as the duration of failure upon the relative chamber weights.

In the compensated phase the left ventricle hypertrophies while the right ventricular and combined auricular weights remain unchanged. This has been termed "eccentric" hypertrophy in the older literature. With the onset of failure the left ventricular weights continue to increase but now the right ventricle hypertrophies along with a parallel increase in the combined auricular weights. Finally, the hypertension becomes "concentric." Expressed as ventricular ratios, the L/R is high in compensated hypertrophy, but approaches normal in protracted failure. The average left ventricular and septal weights in the seven cases with failure for over three years (group X, fig. 2) was less than that of group IX with failure.
from 1 to 3 years. The reason for this is uncertain and the small number of cases makes one hesitate to consider as significant such factors as the older average age, the degree of hypertension, the severity of failure and the effects of digitalis and diuretics. The problem of partial atrophy following a marked that local myocardial hypoxia resulting from coronary arteriosclerosis may induce hypertrophy of the involved area. It is probable, however, that coronary arteriosclerosis may influence the myocardial weights in many ways such as (a) by limiting the degree of myocardial hypertrophy in severe hypertension, (b) by

<table>
<thead>
<tr>
<th>Table 1.—Myocardial Weights in Hypertensive Cases with and without Failure</th>
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<tbody>
<tr>
<td>Number of Cases</td>
</tr>
<tr>
<td>-------------------</td>
</tr>
<tr>
<td><strong>Cases Without Failure</strong></td>
</tr>
<tr>
<td>I. History of hypertension but none recently</td>
</tr>
<tr>
<td>II. Fluctuating hypertension</td>
</tr>
<tr>
<td>III. Hypertensives without failure dying of noncardiovascular and nonrenal lesions</td>
</tr>
<tr>
<td>IV. Death from cerebrovascular lesions</td>
</tr>
<tr>
<td>V. Previous cerebrovascular accidents, then death from noncardiovascular causes</td>
</tr>
<tr>
<td>VI. Death in uremia without significant failure</td>
</tr>
<tr>
<td><strong>Cases With Failure</strong></td>
</tr>
<tr>
<td>VII. Failure for a few weeks only</td>
</tr>
<tr>
<td>VIII. Failure over a few weeks and up to one year</td>
</tr>
<tr>
<td>IX. Failure from 1 to 3 years</td>
</tr>
<tr>
<td>X. Failure 3 years or more</td>
</tr>
<tr>
<td>L. V.—Left Ventricle, lateral wall.</td>
</tr>
<tr>
<td>Sept.—Interventricular septum.</td>
</tr>
<tr>
<td>R. V.—Right Ventricle, lateral wall.</td>
</tr>
</tbody>
</table>

hypertrophy of the heart awaits further investigation.

In the present study, careful examination of the coronary arteries was performed. It was interesting to note that marked myocardial hypertrophy did not occur when the coronary arteries were markedly narrowed by atherosclerosis. This is contrary to the belief of some7,13 leading to progressive ischemic effects on an overtaxed, hypertrophied myocardium, and (c) by shortening the patient’s life and hence interval of time in which hypertrophy could occur. In the “compensated” group, the cases dying in uremia but free of significant failure showed the most marked hypertrophy of the septum
and left ventricle with only a mild increase in the auricular and right ventricular weights. The hypertension was not necessarily more severe in these cases but the fluid and electrolyte disturbances may have led to hypervolemia and hemodynamic alterations.

**Fig. 2**. The Effect of Hypertension and of Hypertension with Failure upon the Mass of the Different Cardiac Chambers. The data and groups of cases in table 1 are graphically presented. Groups I through VI are not arranged as to duration of hypertension, while groups VII to X are arranged as to duration of failure. The smooth curves, however, suggest that the left ventricular and septal weights increase before and after failure, while the right ventricular and combined auricular weights increase only after failure ensues. The general equality of septal with lateral left ventricular wall weights and of both auricles with lateral right ventricular wall weights is seen.

**Discussion**

**Relation of Myocardial Bulk to Work Performed**

In the absence of local infiltrations or of disease, the myocardial bulk of any chamber should be proportional to work performed by that chamber. This view finds support in the following observations: (a) the relatively constant ratio of heart weight to body weight with growth and maturity, (b) the fairly constant L/R values for all ages after infancy, (c) similarity of L/R values in different species of mammals, (d) hypertrophy from increased work of a chamber, as from valvular lesions and hypertension, (e) hypertrophy of other forms of muscle from increased work, as skeletal muscle in exercise and smooth muscle in obstructed hollow organs. It would appear reasonable to conclude that increased work leads either directly or indirectly to hypertrophy. This is disputed. An even more uncertain aspect is the means through which such mechanical work may lead to growth of the myocardium, the following factors having been suggested: intracardiac neurologic reflexes, temporary stretch causing nutritional effects, injury or "toxic" products therefrom, impaired blood flow as well as increased blood flow. Some of these have been proposed as operating, per se, and not necessarily from mechanical factors. It is obvious that the cause and mechanism of myocardial hypertrophy are poorly understood, and that the various suggested biochemical or physiologic factors have not clarified the problem. Some idea of the relationship of work performed per weight of the myocardium may be gained from a discussion of the factors involved in the work of the heart and from the data presented in this paper.

The heart performs work in two ways, first by ejecting a certain volume of blood against resistance or pressure (static factor) and secondly by imparting a certain velocity to this ejected blood (kinetic factor). The work (W) of a ventricle is the sum of these two components and may be expressed by the following formula:

\[ W = QR + \frac{MV^2}{2g} \quad Kg. \, M. \, per \, second \]

in which \( W \) = work in kilogram meters per second; \( Q \) = liters of blood expelled per second; \( R \) = mean blood pressure in terms of meters of blood; \( M \) = mass of blood ejected in kilograms per second; \( V \) = velocity at which blood is ejected in meters per second; and \( g \) = gravity constant (9.81 meters per second²).

The static component of ventricular work (work against resistance, \( QR \)) is the product of
cardiac output and mean systolic ejection pressure. This use of mean pressure may yield a 10 to 20 per cent error since blood is ejected against a variable and not a constant pressure. The kinetic component, \( MV^2 \), is not easily assessed in man although efforts have been made to do so.\(^7\) \(^{11}\) \(^{12}\) \(^{13}\)

**Relation of Left Ventricular Weight to Work Performed**

Right ventricular weights could not be correlated with work of this chamber since cardiac catheterization was not performed in any of these patients. In figure 3 an attempt has been made to correlate the weight of the left ventricle with the work performed by this chamber. The total left ventricular weights (lateral wall and septal portions) per square meter of body surface are plotted against the mean arterial pressures. Only those cases with repeated blood pressure recordings were utilized; the seventy-eight plotted cases are equally divided between those without failure (circles) and those with failure (crosses). The dotted line drawn near the mid-area has the approximate value of \( W = 1.06P - 76 \), in which \( W \) represents the left ventricle in grams per square meter body surface and \( P \), the mean arterial pressure. The failure cases are equally distributed on either side of the dotted line while the greater proportion of the compensated cases occur below the line. The relation of weight to work performed as presented in figure 3 should be regarded more as a guide for future investigations than as an accurate analysis since the work cannot be expressed by the blood pressure alone without knowledge of the output. Also, the blood pressure in cases with failure will be lower than in compensation, and the blood pressure in cerebrovascular accidents may have been augmented by increased intracranial pressure. The probable straight-line relationship in figure 3 does suggest, however, that within wide variations the increasing bulk of the left ventricular myocardium is associated with increased work as manifested by increased blood pressure.

There were two cases (only one of which is plotted in figure 3) of irrefutable hypertension with ensuing failure for over 7 and 10 years in which the total left ventricular bulk was of less than normal value. The right ventricle weighed more than the left. This small left ventricular weight is not easily explained, but several possibilities may be mentioned: atrophy following previous hypertrophy, adaptation of body to diminished blood supply, assumption of left ventricular work (such as increased filling pressure) by the right ventricle. One of these patients was 68, the other 72 years of age, but plotting all cases in figure 3 against age did not show a more significant correlation. Separate plotting of all cases of uremia, cerebrovascular accidents, and marked coronary atherosclerosis also failed to show a more significant relationship.

**The Apparent Greater Efficiency of the Left as Compared with the Right Ventricle**

Comparison of the two ventricles discloses difference in their work per gram of myocardium, the right ventricle appearing less efficient than the left. Since the difference in the work of the two ventricles is almost entirely proportional to the differences in their pressures, it is usually considered that the right ventricle performs only one-sixth of the work.
of the left ventricle\textsuperscript{11} although this may be only a rough approximation.\textsuperscript{13} Actual measurement of the work of the two ventricles suggests a less marked difference. Dexter\textsuperscript{14} found the work of the right ventricle in eight normal resting controls ranged from 0.64 to 1.10 Kg.M. per minute per square meter, with an average of 0.89, but considered Riley’s\textsuperscript{15} somewhat lower values for three normal patients as a more accurate basal figure. Left ventricular work is approximately 4.5 to 6.0 Kg.M. per minute\textsuperscript{19} or 2.5 to 3.5 Kg.M. per minute per square meter. Thus, the work of the left ventricle is at least three and maybe six times that of the right ventricle while the left ventricle only weighs twice as much as the right.

When using the more conservative values for the comparison of the work of the ventricles we obtain:

\[ \frac{L.V. \text{ Work}}{R.V. \text{ Work}} = \frac{3.0}{10} = 0.30 \]
\[ \frac{L.V. \text{ Weight}}{R.V. \text{ Weight}} = \frac{0.7}{0.64} = 1.10 \]

The left ventricular myocardium performs at least twice as much work per gram as the right ventricular myocardium. There are several possible explanations for the seeming greater relative efficiency of the left ventricle: errors in estimation of ventricular work, errors in dividing the myocardium between right and left ventricles, the relation of wall thickness and ventricular volume to force of contracture.

The greater efficiency of the left ventricle cannot be explained by the kinetic component of work. The kinetic factor is negligible at rest and is generally disregarded in the estimation of cardiac work. Only when there is a marked increase in the cardiac output and hence, the velocity of blood, does the kinetic factor reach significant proportions necessitating its calculation in the work of the heart. In strenuous exercise the kinetic factor may be in excess of 10 per cent of the total work performed and in violent activity it may exceed the static factor.\textsuperscript{10} This disproportion is to be expected since the kinetic factor increases as the cube of the output while the static factor increases in direct proportion to the output. The kinetic factor on the two sides is about equal since the velocity of blood in the pulmonary artery and aorta is the same, the latter being determined by similar cross-sections of the two arteries and equal volume output of the two ventricles.

The division of the septum into right and left portions may have been erroneous in all reported methods since Müller.\textsuperscript{1} If the entire septum is considered as left ventricle the above ratio of work per gram of myocardium is approximately 1.0.

\[ \frac{L.V. \text{ Work}}{R.V. \text{ Work}} = \frac{3.0}{1.10} = 2.73 \]

Finally, there is the basic problem of muscle function with such well-known aspects as the stretch of the muscle fibers, the diastolic and systolic volumes, the contracture distance, and the wall thickness. The observations that the energy of contraction of voluntary muscle is proportional to its stretch\textsuperscript{16, 17} were applied to the heart by Starling to explain the wide adaptability of this organ to increased load. The “law of the heart” (cardiac output as a function of diastolic volume) was subsequently amplified\textsuperscript{18, 19, 20} and related to the work of the heart, the energy of contraction being proportional to the diastolic ventricular volume, hence, to the length of the muscle fibers. It is generally interpreted that the myocardial fibers do more work by contracting the entire distance over which they have been stretched by the greater diastolic volume, but the actual contracture distance was not regarded by Starling and his associates\textsuperscript{19, 20} as a significant factor. They contended that systolic volume was unrelated to oxygen consumption of the heart and that the unimpaired heart responded to increased work by greater diastolic as well as greater systolic volume. This was true whether the work increase resulted from elevated arterial resistance or increased output. While these conditions occurred in the heart in “good condition” in the heart-lung preparation, it was further contended that “as the heart tires it has to dilate continuously in order to maintain its mechanical performance constant.” Thus, both the intact and “tiring” heart are aided in work performance through dilatation.

Gesell\textsuperscript{21} believed these observations by Starling and associates required broader interpretation, especially in regard to the surface-volume factor. If the ventricle is considered
roughly spherical then the volume of the dilating chamber increases more rapidly than the surface (surface = $4\pi r^2$; volume = $\frac{4}{3}\pi r^3$).

The greater the ventricular volume, the greater will be the output per unit length of muscle shortening. Or, a constant output may be maintained by ventricular dilatation and a shorter contracture-distance of the muscle fibers. Applied to the observations of Starling as cited above, we will expect a difference in the contracture distance depending upon which component of static work is augmented; increase in work-against-resistance is accompanied by a shortened equivalent distance, while work of the same magnitude due to increased output will be associated with a relatively greater contracture distance. And the “tiring” heart of the heart-lung preparation performs the same work by chamber dilatation and by reduction of contracture distance.

The observations of subsequent investigators have indicated that the heart expends more energy in performing increased work due to augmented arterial resistance than it does in performing equivalent work from greater output. Also, dilatation of the heart is not accompanied by increased mechanical efficiency, which remains about the same with increased venous inflow and is diminished with augmented resistance-load. This correlates well with the changes in contracture-distance of the muscle fibers. It is also possible that increased resistance load or afterload requires more energy in raising the interventricular pressure, the mechanical efficiency thereby being reduced. Further elucidation of the significance of the contracture distance, the initial tension and initial length of muscle fibers is needed. The isolated analysis of a physiologic phenomenon, such as by the heart-lung preparation, does not reconstruct the normal function; the innervated heart, for example, does not show the same diminution of mechanical efficiency for increased resistance-load as the isolated heart.

The apparent greater efficiency of the left as compared to the right ventricle may have some relationship to a difference in their surface-volume or contracture-distance factor. Since the output of the two ventricles is the same, a difference in their contracture distances must be related to a difference in systolic volume, but the estimated diastolic volume of the two ventricles is approximately the same. If, however, the left ventricle has the smaller volume, as one might interpret from the gross appearance at autopsy, then the average contracture distance of its fibers would be greater, and presumably more effective in performance of work. At the same time this would imply a greater “reserve” of the right ventricle not only as a work per gram basis but also since its contracture distance will change less with increased work or with failure.

There is a further aspect which might have some bearing on the relative efficiencies of the two ventricles. In a thin-walled spherical viscous, the true wall thickness varies inversely with the square of the radius, while the “effective wall thickness” of Brody and Quigley, based upon the number of elastic fibrils in 1 cm. of unstretched wall, is proportional to the first power of the radius. If this concept may be applied to a thick-walled, nonspherical contractile ventricle, the ratio of “effective wall thickness” of the ventricles at equal diastolic volumes would be the same as the ratio of the true wall thickness at the “unstretched” systolic values. If the ratio of “effective thickness” to actual thickness were to be increased 2.14 times, thereby accounting for the greater relative efficiency of the left ventricle, the chamber of this ventricle would have to be empty at systole in comparison to the 80 cc. systolic volume of the right ventricle.*

* $d_s = \text{thickness of wall before stretch begins (as at systole)}$

$\frac{d_{eff}}{d_{at}} = \text{effective wall thickness when stretched (as at diastole)}$

$d_{at} = \text{actual wall thickness when stretched (as at diastole)}$

$r_s = \text{radius at which stretch begins (as at systole)}$

$r = \text{radius (as at diastole)}$

$d_{eff} = \left(\frac{r_s}{r}\right)^3 d_0$ Brody and Quigley

$d_{at} = \left(\frac{r_s}{r}\right)^2 d_0$

Frank

These two formulas have the relationship,

$$\frac{d_{eff}}{d_{at}} = \frac{r}{r_s}$$

(continued on following page)
Relative Chamber Weights and Concepts of Failure

The correlation of the changing myocardial weights of the two ventricles with the concepts of failure is as difficult as the explanation of their relative efficiencies. Cardiac failure is the inability of the heart or a ventricle to supply the tissues with required blood. The phenomena of venous congestion, edema and increased blood volume with accompanying symptoms constitute the syndrome of congestive failure. The exact mechanism of congestive failure is controversial, there being two types of failure and two concepts for the mechanism of failure. The types of failure are low output and high output; the two concepts of congestive failure are the “backward” and the “forward.” According to the “backward-failure” theory, the left ventricle fails to pump sufficient blood which then accumulates in this chamber and is dammed back into the pulmonary bed, right side of heart, and venous system. The “Lungenstarre” or pulmonary rigidity due to congestion from left heart failure described by von Basch in the latter part of the nineteenth century has been fully elaborated by Harrison. According to the current “forward-failure” theory the diminished cardiac output leads to a proportionally greater decrease in splanchnic flow. The renal tissue is thought to be very sensitive to these circulatory changes, and although normal filtration of sodium is maintained by constriction of the efferent glomerular arterioles, the tubular reabsorption of sodium is accelerated. The resulting retention of sodium or salt leads to fluid retention, increased blood volume, venous congestion and edema. The increased venous pressure may aid the failing heart by increasing the filling pressure and diastolic volume.

In all types of failure, acute or chronic, high- or low-output, there is a relative decrease in renal and splanchnic flow, a retention of sodium and water, a rise in blood volume and an increase in vasomotor tone. But the degree of failure of the myocardium to meet the tissue needs does not parallel the degree of congestion, and the evidences of congestion, such as increased blood volume edema, decreased circulatory rate, increased venous pressure, may vary independently of one another over wide ranges. Further investigations are necessary before the syndrome of congestive heart failure can be adequately explained.

From the data presented in table 1, and figure 2, it is apparent that: (a) only the left ventricle hypertrophies in hypertensive individuals without congestive failure; (b) with the advent of congestive failure, the right ventricle hypertrophies progressively with the duration of failure, both auricles hypertrophy and the left ventricle also continues to increase in weight; (c) after failure has existed for over three years the two chambers diminish in weight; the reduction in weight may be more marked in the left ventricle.

The observation under (a) above suggests that the left ventricle acts as an isolated pump responding to an increased peripheral resistance, hence to increased work, with hypertrophy. Since there is an eccentric hypertrophy we may conclude that the right ventricle does not hypertrophy through a generalized stimulus of the scroll muscles encompassing both right and left ventricles. In this study it was not possible to determine the presence of left
ventricular failure unaccompanied by right ventricular failure, but the right ventricular weight increases only after the advent of congestive failure. This is to be expected if the myocardial bulk of a chamber is proportional to work performed since the pulmonary arterial and right ventricular pressures are elevated in left ventricular failure.\(^{39}\)

According to the “backward-failure” theory, blood would be dammed back into the lungs from the failing left ventricle. Comparable to the response of the left ventricle to the increased peripheral resistance of hypertension, the right ventricle responds to the impaired flow of blood through the lungs into the left ventricle by increased pressure and work. “Forward-failure” might lead to an increase in blood volume, venous pressure, and filling pressure of the right heart with dilatation and, if Starling’s postulate is correct, with increased work. Since the output cannot be increased above that of the weak left ventricle, pressure becomes the elevated component of work.

The apparent continued hypertrophy of the left ventricle after the onset of failure is not easily explained by either “forward” or “backward” failure theories. It is possible that the cases with cardiac failure represent a group separated from the others on the basis of a more marked hypertrophy which has “outstripped the capillary bed.”\(^{40, 41, 42}\)

As discussed above, one possible explanation for the greater efficiency of left as compared with right ventricle may be a smaller residual systolic volume of the former. Since an equal volume is ejected by the two chambers, those fibers encompassing the smaller volume contract over a greater distance. Through the completeness of contracture this may increase the work performed per gram of myocardium. When the ventricle fails, the accompanying dilatation stretches out each muscle fiber but also requires less contracture of each fiber to eject the original volume of blood. Increasing increments of chamber volume require progressively less contracture distance of their fibers. The most marked changes will occur in the chamber of smaller initial volume, such as has been suggested for the left ventricle. If this is true, the right ventricle with its greater initial volume would have more “reserve” in failure since the increasing systolic residual volume would affect it less. If the stretched fibers of the dilated chambers now become adjusted to their new length (thus acquiring a new “initial length”) for instance through hypertrophy, the myocardium will again be able to perform more work but may be less efficient per gram of myocardium than before. The decreased efficiency of the dilated ventricle may explain the continued hypertrophy of the left ventricle after the advent of congestive failure.

This brings us to the role of dilatation in the stimulation and mechanism of hypertrophy. On one hand, dilatation is regarded as a means of increasing work performance and on the other hand, “beyond a certain optimum,” it is considered as an evidence of myocardial weakness. Some consider that dilatation always precedes hypertrophy but roentgenographic studies in man indicate that hypertrophy advances without dilatation or that the two processes occur gradually and concomitantly. Whether or not a dilated, “failing” ventricle can continue to hypertrophy without undergoing compensation has not been established.

Other factors which may be operative but are difficult to assess should be mentioned: (a) intermittent periods of compensation brought about by rest, digitalis, and diuretics, (b) alterations in cardiac rhythm, (c) hypoxia during failure as a possible direct stimulus to myocardial hypertrophy or as an indirect influence through altered blood volume, and blood viscosity.

It would appear that the changes in the weights of the cardiac chambers presented in this paper cannot be entirely explained by the present concepts of cardiodynamics and cardiac failure.

**Summary**

1. By the use of a comparatively simple technic the weights of the different myocardial chambers have been studied in 130 cases of hypertension both with and without failure.

2. In the absence of failure only the left ventricle hypertrophies, but with the advent
of congestive failure the right ventricle hypertrophies along with a continued increase in the left ventricle. The right ventricle continues to hypertrophy with the duration of failure up to three years; after this time there is an apparent decline in the right and especially the left ventricular bulk.

3. An attempt has been made to correlate the work of the left ventricle with the weight of left ventricular myocardium in grams per square meter of body surface.

4. The left ventricle performs at least twice as much work per gram of myocardium as does the right ventricle. Several possible explanations for the apparent greater efficiency of the left ventricle are presented. The possible influences of the surface-volume or contraction-distance factor as well as the “effective wall thickness” are considered.

5. The changes in chamber weight before and after failure are discussed in relation to the concepts of congestive heart failure.

**SUMARIO ESPAÑOL**

El peso del miocardio de las diferentes cavidades del corazón fue determinado en 130 casos de enfermedad cardiovascular hipertensiva con y sin descompensación. El ventrículo izquierdo se hipertrofia antes y prosigue hipertrofiándose después que la descompensación aparece. El ventrículo derecho y ambas aurículas se hipertrofian solamente después que la descompensación aparece. Correlacionando el trabajo ventricular con la masa muscular, el ventrículo izquierdo aparentemente produce dos veces mas trabajo por gramo de músculo que el ventrículo derecho. Varias posibles explicaciones para esta aparente mayor eficiencia del ventrículo izquierdo se presentan. Los cambios en el peso de las cavidades cardíacas se discuten en relación al concepto de la descompensación cardíaca.

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The Weight of the Heart and Its Chambers in Hypertensive Cardiovascular Disease with and without Failure

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