Ventricular Response to Increased Outflow Resistance in Absence of Elevated Intraventricular End-Diastolic Pressure

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With the assistance of Geraldine Nelson

Classically, the adaptation of the ventricle to increased outflow resistance is considered to be associated with increased end-diastolic pressure and volume. Recent studies by Rosenblueth and his co-workers have shown, however, that when the right ventricle is forced to contract against an augmented outflow pressure, after an initial dilatation, the right ventricle gradually resumes its original volume while maintaining its full degree of compensation. Similarly, Sarnoff and his associates have demonstrated that initially, the end-diastolic pressure in the left ventricle increases when outflow resistance is suddenly augmented; however, the end-diastolic pressure shortly returns to or toward the control level with no detectable reduction in the degree of cardiac adaptation to the maintained increase of resistance. Many years earlier, Anrep had observed a slight, similar response to increased arterial resistance; with the addition of epinephrine to the blood, however, this reaction was much more fully developed.

The experiments to be described were designed to determine whether the initial increase in end-diastolic volume and pressure is an integral component in the cardiac adaptation to increased outflow resistance. A canine, isolated heart preparation was designed in which it was possible to prevent increased ventricular volume in response to augmented resistance, and to compare the magnitude of the response so obtained to that in which the heart was permitted to dilate when subjected to the same increase of resistance.

Methods

Thirteen experiments were performed upon modified, isolated, supported, canine heart preparations, as described by Sarnoff and his collaborators. The experimental dogs weighed 17.6 ± 3.38 kg (mean ± SD), and the supporting dogs weighed 16.8 ± 4.47 kg. All dogs were anesthetized with sodium pentobarbital, 30 mg/kg, iv. A small incision was made in the fourth right intercostal space, and intermittent positive-pressure respiration was instituted. The azygos vein was ligated, and loose ligatures were placed about the superior and inferior venae cavae and the hilus of the right lung. An incision was made in the fourth left intercostal space. The arch of the aorta was dissected free from the pulmonary artery and the posterior thoracic wall to the level of the first intercostal artery. A heavy ligature was looped loosely around the aorta, between the origins of the brachiocephalic and subclavian arteries. Heparin (donated by Lilly Research Laboratories), 3.4 mg/kg, was administered intravenously to prevent blood coagulation. A catheter was passed into the left atrium (LA, fig. 1) via a pulmonary lobular vein for pressure recording. A cannula was inserted into the left branch of the pulmonary artery (PA), and a loose ligature was placed about the right branch of the pulmonary artery close to the bifurcation. A large-bore cannula was inserted into the left auricular appendage. This cannula was connected to a reservoir (RES 2), which also served as a bubble trap. Loose ligatures were placed about the hilus of the left lung and the origin of the brachiocephalic artery.

After all of these surgical procedures had been completed, a large-bore cannula was inserted into the descending aorta, and it was advanced in the upstream direction until its tip was located just proximal to the origin of the brachiocephalic artery. It was secured in this position by means of the ligature placed about the aortic arch at the
level between the origins of the brachiocephalic and left subclavian arteries. As soon as the aortic cannula was inserted, the left ventricular outflow was diverted through the external circuit shown in figure 1. Initially, stopcocks SC₁, SC₄, SC₅, and SC₆ were open, and SC₁ and SC₂ were closed, so that blood was pumped by the left ventricle through the circuit up to the open reservoir (RES₁), and thence through SC₁ and RES₁ back to the left atrium (LA). This circuit was filled with donor blood prior to aortic cannulation. A heat exchange unit (HEU) was included in the venous return line to maintain blood temperature constant at about 37°C, as sensed by a thermistor inserted into the aortic cannula.

As soon as flow was established through this external circuit, the brachiocephalic artery, venae cavae, right pulmonary artery, and the hili of both lungs were ligated in rapid succession. Since stopcock SC₆ was opened and SC₇ was closed, the coronary venous outflow was diverted to an external jugular vein of a supporting dog (fig. 1, DONOR). When solenoid valve SV₁ was opened, blood from a carotid artery of the supporting dog was directed into reservoir RES₁. This solenoid valve was governed by a level control (LC) in order to maintain the volume of blood in RES₁ virtually constant.

The methods of recording phasic pressure and flow, and of the derived variables, phasic ventricular power, stroke work, and stroke volume, were identical with those described previously. Just as in this preceding study, the short, compliant segment of ascending aorta proximal to the more rigid external circuit was responsible for the forward flow registered by the extracorporeal flowmeter probe during diastole. As a corollary, the instantaneous power computed at the level of the flowmeter probe was not equal to the simultaneous power being generated by the ventricle. Under the conditions of these experiments, however, the peak computed power was a direct function of the left ventricular power. Furthermore, this small compliance may have altered slightly the values for stroke volume and stroke work during the first few beats after changes in outflow resistance, but not after the steady state levels had been attained.

Outflow resistance was varied in a manner similar to that described in the preceding report. A variable resistance (VR, fig. 1) was preset before any experimental observations were made. A sudden, reproducible increase in resistance could be imposed by closing solenoid valve SV₁ in a channel parallel to the variable resistance.

The external circuit described above, in which the left ventricular outflow passes through the aortic cannula, solenoid valve SV₁, electromagnetic flowmeter (FM) probe, peripheral resistance, and SC₁ to the reservoir RES₁, and thence through SC₁ and RES₁ back to the left atrium, is designated as the “open system.” It is analogous in certain respects to the external circuits of the classical heart-lung preparations and other types of isolated heart preparations, in that atrial inflow is somewhat independent of ventricular outflow, by virtue of the constant level of blood in RES₁, which is exposed to atmospheric pressure. Under these conditions, there is a progressive diminution of the oxygen saturation of the blood. This was made to occur at a very gradual rate by interposing a suitably large reservoir (RES₂) between the point of return of coronary venous blood and the left atrium. In this way, the closed system could be maintained for several minutes with only a relatively small reduction in the oxygen saturation of the blood. Immediately after switching from the open to the closed system, the blood volume in the closed circuit was rapidly adjusted so that the atrial pressures approximated those in the open system at the control level of resistance.

Results

The effects of increased outflow resistance upon left ventricular performance in the “open system” are displayed in figure 2 for a typical experiment. The increased resistance was imposed late in diastole during the fifth beat shown on the record. Although the
ventricle began its next contraction from an identical end-diastolic intraventricular pressure and volume (as verified by the left atrial pressure tracing), there was a 6-mm increase in systolic pressure manifest in the next beat, confirming the findings of a previous study. The augmented resistance was maintained for 20 cardiac cycles, and during this time, aortic pressure rose progressively, reaching a plateau by the twelfth beat. The imposition of increased resistance produced an immediate diminution of aortic flow (i.e., cardiac output minus coronary blood flow). The control flow varied from a peak value of 31 ml/sec during systole to a minimum of 18 ml/sec during diastole (ascribable to the compliant segment of ascending aorta proximal to the extracorporeal flowmeter probe). During the beat immediately following the increase of resistance, phasic flow varied from 13 to 20 ml/sec, and gradually rose toward, but not to, the control rate during the subsequent 20 beats. Similar changes are also evident in terms of the stroke volume (minus coronary blood flow). During the period of increased resistance, it is likely that coronary blood flow was enhanced, and this was confirmed in some of the experiments by direct measurement of right ventricular output. Therefore, the fraction of the total left ventricular outflow not passing through the flowmeter was undoubtedly greater during the phase of augmented resistance.

Peak left ventricular power fell appreciably immediately after the augmentation of resistance, but progressively rose until it attained a value slightly in excess of the control level. The stroke work also was reduced immediately after the resistance was increased, but it progressively rose to levels above control. All of these changes were accompanied by a progressive rise in the left atrial pressure. The
end-diastolic pressure during the control period was 2 mm Hg; this increased to 9 mm Hg 20 beats after resistance was augmented. The heart rate remained constant during these changes in resistance.

In the same experiment, the external circuit was then switched to the "closed system," and the blood volume was quickly adjusted so that the left atrial pressure at the control resistance was almost identical with that in the open system. As figure 3 shows, control systolic aortic pressure was approximately the same as that in the open system (fig. 2), but diastolic pressure was somewhat lower and aortic flow somewhat greater. After a control period of approximately one minute (the last four beats of the control period appear as the first four beats in fig. 3), an increase in resistance was imposed which was the same magnitude as in the preceding study upon the open system. Resistance was increased late in diastole, and this resulted in an immediate elevation of aortic systolic pressure and reduction in phasic aortic flow and stroke volume, peak left ventricular power, and stroke work, similar to those changes observed under analogous circumstances in the open system. Also, progressive changes in these variables were observed over the next several beats. These changes were directionally the same, but of lesser magnitude than those observed in the open system during the equivalent augmentation of resistance. Although peak ventricular power and stroke work rose above the levels attained during the first beat after increasing resistance, they did not exceed the control levels, as they did in the open system. Also, in contrast to the observations in the open system, the end-diastolic left atrial pressure remained virtually constant during the period of augmented resistance in the closed system.

To determine to what extent left atrial pressure...
INCREASED OUTFLOW RESISTANCE

TABLE 1
Changes in Projected Left Ventricular Dimensions After Increasing Outflow Resistance

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<tr>
<th>Expt. No.</th>
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pressure reflected ventricular dimensions in this study, motion pictures of the isolated heart were taken at 38 frames per second in two of these experiments. Fine sutures were placed in the ventricular wall at several locations to serve as reference points. An electric lamp, wired in parallel with the solenoid valve (fig. 1, SV₁), was located close to the heart to identify the moments of application and release of the augmented resistance. Frames from these films were projected sequentially on a screen, and the distances between reference points in the longitudinal and transverse axes of the images of the left ventricle were carefully measured. The onset of ventricular ejection was identified as the frame in which the first definite reduction in dimensions was detected. The preceding frame represents the end of diastole (ED). The dimensions from this frame, from the preceding frame (ED₁), and from the one before this (ED₂) were measured carefully on the enlarged image and are presented in table 1 as indices of left ventricular end-diastolic volume.

In the first experiment (no. 10), in the open system, the distance between the longitudinal reference points in the projected image was 98 to 99 mm at the end of diastole during the control beat (C₁) just prior to increasing the resistance; between the transverse reference points, the distance was 70 to 71 mm. During the diastole of the seventh experimental beat (E₇) after resistance was augmented, these dimensions had increased to 103 to 104 mm for the longitudinal, and to 78 mm for the transverse dimensions. In the closed system, the control (C₁) dimensions were essentially the same as for the open system. However, after resistance was increased, these dimensions did not change detectably by the seventh experimental beat (E₇).

Very similar results were obtained in the other experiment (no. 11) in which motion pictures were taken. In the open system, the
Figure 4

Ventricular response to the same increase of outflow resistance in the open and closed systems in the experiment in which the greatest compensatory response was observed in the closed system. Recording speed: 5 mm (distance between heavy vertical lines) per 2 seconds.

Longitudinal dimensions increased from a control value of 61 to 62 mm to a length of 66 to 68 mm during the 20th beat ($E_{20}$) after resistance was augmented. The transverse dimensions increased only very slightly. In the closed system, on the other hand, both longitudinal and transverse dimensions remained constant. In both of these experiments, observations were made repeatedly at different degrees of increased outflow resistance. Consistently, increases in diastolic dimensions were manifested when resistance was augmented in the open system, but significant differences were never detectable in the closed system for equivalent changes in resistance.

In addition to observations made at the recording speeds employed in figures 1 and 2 (25 mm/sec), similar tracings were obtained in each experiment at slower recording speeds (2.5 mm/sec) for longer periods of increased resistance. In figure 4 are reproduced tracings made at this speed in that experiment in which the maximum degree of adaptation was observed in the closed system. The changes which occurred in the open system (left half of figure) were quite typical of those which were observed in all experiments under similar circumstances. Aortic and left atrial pressures rose, but all other variables fell immediately after imposing the increased resistance. However, in the course of the next several beats, aortic flow and stroke volume progressively rose toward, but did not attain, control levels, while peak ventricular power and stroke work actually increased to levels in excess of control. Left atrial pressure increased progressively to attain a maximum value in 12 to 14 sec, and then gradually diminished. By the end of the 58th sec during which the resistance was augmented, left atrial pressure had decreased to a level only slightly above control. In a few experiments,
Composite data for all experiments in this study. Lengths of the bars represent the mean value of each variable for any given beat, expressed as the per cent of the value for that variable during the beat (C₁, not shown) just prior to augmenting the resistance. The vertical lines in each bar represent the magnitudes of the standard error. The unshaded bars represent the data for the open system; the hatched bars, the closed system. Beats C₁ and C₅ are the third and fifth control beats, respectively, prior to augmenting resistance. Beats E₁ to E₅ are the first, third, fifth, tenth, and terminal (mean duration, 32 seconds) experimental beats during the period of increased resistance. Beats R₁ and R₁₀ are the first and tenth recovery beats, after returning to the control level of resistance.

Mean left atrial pressure returned to the control level, usually within 30 seconds.

In the closed system in the same experiment (right half of fig. 4), most of the changes were quite similar. During the 37 sec period of augmented resistance, progressive compensatory changes were evident in aortic pressure and flow, and in ventricular power and work. Peak ventricular power attained control levels and ventricular work exceeded control levels by the end of this interval of time. Left atrial pressure, however, decreased slightly.
as resistance was increased, and remained constant during the entire period of augmented resistance.

The composite data for all 13 experiments are shown in figure 5. The heights of the bars represent the magnitude of any given value in terms of per cent of the corresponding value for the beat just prior to increasing the resistance; the vertical lines in each bar represent the standard errors of the means. The open bars depict the changes which occurred in the open system; the hatched bars, those in the closed system. The values for the third (C₃) and fifth (C₅) control beats prior to increasing resistance are shown to indicate the extent of variability with time. The increased resistance was maintained for a mean duration of 32 seconds in both the open and closed systems. It is evident from the figure that the changes observed during the first experimental beat (E₁) after increasing resistance were not significantly different in the two systems. In the subsequent beats, however, the differences became progressively more evident. Peak aortic pressures increased to a considerably greater extent in the open than in the closed system. Peak aortic flow and stroke volume increased toward control in the open system, but changed only slightly in the closed system. Peak ventricular power attained the control level by the 10th beat (E₁₀) in the open system, and exceeded control at the termination of the period of increased resistance (E₅). In the closed system, on the other hand, peak ventricular power did increase progressively, but did not attain control values. Similarly, stroke work exceeded control levels after the fifth beat in the open system, but was still slightly below control by the terminal beat during augmented resistance in the closed system. Finally, left atrial pressures were elevated in the open system when resistance was augmented, but in the closed system, left atrial pressures were slightly reduced. In the open system, left atrial pressure typically increased progressively to attain a maximum value at some point in time between the tenth and terminal experimental beats (as exemplified by the experiment depicted in figure 4). After having reached a peak value, the left atrial pressure then usually declined toward a mean value which was still significantly above the control level, as shown by figure 5, beat E₅. In all experiments, the heart rate was not affected by the change of resistance in either the open or closed system.

Discussion

In almost all of the isolated heart preparations which have been studied previously, the experimental conditions were similar to the "open system" of the present study, in that a reservoir of blood, in which the surface was exposed to atmospheric pressure, was connected to the atrium. In such a circuit, the venous return to the atrium is equal to \((P_h - P_a)/R\), where \(P_h\) is the hydrostatic pressure exerted by a static column of blood from the atrium up to the blood surface in the reservoir, \(P_a\) is the atrial pressure, and \(R\) is the resistance to flow in the circuit from the reservoir to the atrium. It has been recognized since the classical experiments with heart-lung preparations that when outflow resistance is suddenly increased, stroke volume is initially diminished. The atrial pressure is virtually unchanged at first, so that venous return to the ventricle is not appreciably reduced. The combination of reduced outflow and constant inflow immediately after augmenting peripheral resistance leads to the progressive increase in diastolic ventricular volume which has so frequently been described. These changes in ventricular dimensions are confirmed by the data in table 1 relative to the open system, and are accompanied by concordant alterations in atrial (and ventricular diastolic) pressure, as shown in figures 2, 4, and 5. Atrial pressure rises progressively, and hence venous return diminishes, in accordance with the above equation. Simultaneously, cardiac output rises progressively, largely as a result of the increasing end-diastolic ventricular volume. The atrial pressure reaches a plateau or peak when inflow equals outflow.

In the "closed system," on the other hand,
the venous return is not dependent upon the height of any reservoir of blood. It is equal to the cardiac output minus the rate of storage of blood in any distensible elements on the arterial side of the circuit. In this study, essentially the only compliances in the closed system are the ascending aorta and left atrium, other than the left ventricle itself.

When the peripheral resistance is increased, a slightly greater fraction of the circulating blood volume is retained on the arterial side of the circuit, largely in the short segment of ascending aorta. This is reflected as the increased aortic pressure in figures 3 to 5. Since the total volume of circulating blood is constant, the retention of some blood in distending the ascending aorta results in an equivalent reduction in left atrial and left ventricular diastolic volumes. This is reflected as a very small diminution in left atrial pressure in figures 3 to 5 (closed system). Analogous changes have been described previously in studies upon a closed-circuit heart-lung preparation. The change in left ventricular diastolic volume is small in the present study is indicated by the virtual constancy of the dimensions included in table 1, before and after augmenting resistance in the closed system. It is very unlikely that there is any appreciable alteration in the volume of blood circulating from the left ventricle around the closed external circuit and back to the left atrium which is related to the parallel coronary circulation, which involves the right ventricle and pulmonary artery. Since the return of coronary venous blood is into the venous side of the closed external circuit, and since the venous pressure diminishes by only a trivial amount when resistance is increased, augmenting resistance to left ventricular outflow should cause no appreciable change in the volume of blood contained in the right ventricle or pulmonary artery segment.

When outflow resistance is increased in the open system, there is initially a progressive rise in left atrial pressure (figures 2, 4, and 5), and a peak value is usually attained in from 5 to 15 seconds (approximately 10 to 30 beats). Accompanying this change in left atrial pressure, there is undoubtedly an increase in left ventricular diastolic volume as well (table 1). In accordance with the Frank-Starling formulations, progressive elevations of aortic pressure, ventricular outflow, stroke work, and ventricular power are observed. This type of compensation has recently been designated "heterometric autoregulation" by Sarnoff and his collaborators.

Soon afterwards, however, the left atrial pressure begins to decline. Most often, over the usual period of observation employed in this study, the left atrial pressure remains somewhat above control (fig. 4), in conformity with the observations of Müller. Occasionally, the left atrial pressure returns to the level which prevailed at the lower, control outflow resistance, similar to many of the observations of Sarnoff and his co-workers. Very probably, these changes in atrial pressure are paralleled by variations in ventricular volume, as described by Anrep, by Müller, and by Rosenbluth and his colleagues. Despite the reduction of left atrial pressure (and presumably of left ventricular end-diastolic volume) toward or actually to the control level, the compensatory changes in power and work are maintained or continue to progress for a brief period while atrial pressure is actually falling (fig. 4). This maintenance of compensation despite the return of atrial pressure (and presumably of left ventricular end-diastolic volume) to or toward control has been termed homeometric autoregulation by Sarnoff and his colleagues.

In the closed system, on the other hand, even though the control left atrial pressures were adjusted to the same level as in the open system, and even though the ventricle was subjected to the same degree of augmentation of outflow resistance, the degree of compensation was usually minimal in comparison to that observed in the open system.

As the composite data in figure 5 show, there is a slight, progressive increase in peak ventricular power and stroke work. Whereas power and work are well above control by the end of the increased resistance period
(E,.) in the open system, power is still significantly less and work slightly less than control in the closed system.

Although the compensatory changes in response to augmented resistance are considerably less marked in the closed system, they are still definite. The most prominent compensatory response observed in this study is displayed in figure 4. Peak ventricular power returned approximately to the control value, and stroke work was significantly enhanced, despite a maintained, slight diminution of left atrial pressure. These adaptive changes may be attributable in part to the increase in coronary perfusion pressure associated with the elevated aortic pressure. Gradual diminution of ventricular dilatation was often observed during exposure to increased arterial resistance in the studies of Patterson, Piper, and Starling. They postulated that this phenomenon was related to enhancement of the myocardial blood supply associated with the elevated arterial pressure. Recent work by Salisbury and his associates has demonstrated that coronary perfusion pressure exerts a profound influence on the response of the ventricle to augmented arterial resistance. The studies of Rosenblueth et al. and of Sarnoff et al. have revealed, however, that homeometric autoregulation may occur even in the absence of appreciable changes in coronary blood flow. Increased activity per se improves ventricular contractility appreciably. It has been suggested by Sarnoff and Mitchell that "an increase in the amount of tension developed by the myocardium per unit of time may be the hemodynamic factor which elicits homeometric autoregulation."

In this regard, the degree of compensation exhibited by the left ventricle in response to augmented resistance is considerably greater in the open system than in the closed system. This was true in the present study even in those experiments in which the left atrial pressure returned completely to the control level in the open system during the period of augmented resistance. This would suggest that a preliminary period of increased left atrial pressure (and of increased left ven.

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stances in more intact preparations, it would be extremely difficult to assess the fraction of the total compensatory response which should be attributed to these two distinctly different intrinsic mechanisms.

Summary

In the isolated, supported, canine heart preparation, the response of the left ventricle to augmented outflow resistance was compared under conditions in which ventricular end-diastolic pressure and volume were increased and in which the pressure and volume remained virtually constant. The compensatory changes were much greater when end-diastolic pressure and volume were increased, even if these alterations in pressure and volume subsequently returned to or toward control levels. Immediately after resistance was augmented, peak ventricular power and stroke work diminished to less than control values. Within a very few beats, however, both of these parameters rose to exceed control levels. However, definite adaptive changes were also observed under conditions in which ventricular dimensions and pressures did not change appreciably, even temporarily. After a similar initial reduction in peak power and work after resistance was increased, peak power rose progressively to a value still significantly below control, while stroke work attained control levels by the end of a mean interval of 32 seconds of increased resistance. The intrinsic adaptation of the ventricle to increased resistance per se undoubtedly contributes to the total compensatory response, even under conditions in which diastolic stretch of the ventricle plays a major role.

References

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