Balance Between Right and Left Ventricular Output

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The right and left ventricles differ greatly in their size, shape, architecture, and function. Even so, these chambers must eject quantities of blood precisely equal, except for the flow from the bronchial arteries into the pulmonary veins, which is probably less than 1 per cent of the systemic flow. This balance in right and left ventricular outputs must be maintained under equilibrium conditions and also must be rapidly restored during or after transitions from one flow rate to another, in spite of many apparently adverse factors. For example, the right ventricle generally begins to empty before the left. Consequently, the time course of ejection must be different in the two chambers. Von Capeller reported that left ventricular output could increase 40 per cent without a change in right ventricular output when the two ventricles were pumping into different systems. Conversely, congenital defects in the partitions of the heart commonly lead to a sustained right ventricular output which is three to five times greater than the output of the left ventricle. On the other hand, the outputs of the two ventricles appear to be equalized even after destruction of much of the right ventricular wall or after extensive damage to the left ventricular wall by myocardial infarction. These observations indicate the extent to which the individual ventricles may adjust their outputs under varying conditions.

One of the oldest controversies in the field of pulmonary circulation concerns the effects of respiration on the blood flow into the pulmonary artery. Some investigators believe that the act of inspiration increases pulmonary resistance and reduces pulmonary flow, while others believe that inspiration increases "venous return" and augments right ventricular output. In either case, the outputs of the two ventricles should be augmented at different phases of the respiratory cycle so that the fluctuations should be out of phase. Similarly, during sudden changes in the circulatory flow (e.g., exercise) or redistribution of blood (e.g., during changes in posture), accelerated right ventricular flow has been postulated to precede the augmented left ventricular output by an interval related to the circulation time through the lungs. Actually, entrance of blood into the pulmonary artery should cause an efflux of blood from the pulmonary veins after an interval equal to the pressure pulse propagation time. The restoration of balance in the right and left ventricular outputs is generally explained in terms of the length-tension relationship emphasized by Starling. According to this hypothesis, increased inflow into the right ventricle would cause that chamber to distend progressively over a few beats as the energy release and stroke volume attained a new higher level. After traversing the pulmonary vascular bed, the additional flow would progressively distend the left ventricle. Thus, during the transition from one flow level to another, the filling pressures and outputs of the two ventricles would be demonstrably out of phase with adjustments in the left ventricular output lagging several cycles behind. Recently, Guz et al. reported that the outputs of the two ventricles were out of phase during respiratory variations when the two flows were measured simultaneously.
This study was designed to test the hypothesis that balance in ventricular output is maintained or restored in accordance with the Starling mechanism and to determine the extent to which changes in the output of the two ventricles are out of phase during spontaneous cardiovascular adjustments.

**EXPERIMENTAL DESIGN**

The balance of ventricular output has usually been studied either by comparing venous or arterial pressure in intact animals or men, or by measuring flow in and out of the ventricles in anesthetized thoracotomized animals. Exposed to an abnormal environment, the hearts of thoracotomized animals are greatly reduced in size and beat rapidly. Furthermore, general anesthesia depresses or distorts neural reflex adjustments and eliminates spontaneous activity.

In order to circumvent these difficulties and limitations, the outputs of both ventricles were recorded simultaneously by flowmeters chronically implanted in intact conscious dogs, and the spontaneous cardiovascular adjustments to respiratory activity, changes in posture, and exercise were studied.

**Methods**

The patterns of right and left ventricular ejection in eight dogs were recorded simultaneously from two pulsed ultrasonic flowmeters positioned on the ascending aorta and on the main pulmonary trunk (fig. 1). The pulsed ultrasonic flowmeter, which has been described previously, is based on the principle that sound waves passing through moving fluid travel at a greater net velocity downstream than upstream. Briefly, two barium titanate crystals are mounted diagonally across a bivalved plastic cylinder. Bursts of ultrasonic waves (3 megacycles) travel along the diagonal path between the crystals, alternating in direction 400 times per second. When the fluid between the crystals is motionless, the transit time of sound is precisely equal in the two directions. If the fluid is moving along the channel, the difference between the transit times upstream and downstream is linearly related to the flow velocity as sampled across the tube. The plastic cylinder prevented expansion of the vessels. Records could be calibrated to indicate volume flow by computation and by perfusion of known volumes through the gauges in situ (see fig. 1).

Additional variables were recorded in some of the experiments. The diameter of the left ventricle was measured continuously by sonocardiometry in two dogs (fig. 1, dogs V34 and V44). Left ventricular pressure was recorded routinely through an indwelling polyethylene cannula extending from the left atrial cavity through the back to the outside (see fig. 1). At times, this pressure was determined by retrograde arterial catheterization. Pressure in the right ventricle was recorded through a catheter inserted into the jugular vein in some instances and through an indwelling atrial cannula in others. In three additional dogs, ultrasonic flowmeters were installed on the inferior vena cava and on the ascending aorta to determine the extent to which the flow into the right ventricle altered before the flow into the left (fig. 5). In all experiments, the heart rate was continuously recorded by a ratemeter triggered by the steep rising phase of the pressure or flow pulses.

Stroke volume was recorded by means of an electronic integrator which was reset after the completion of each cycle, so that the deflection was proportional to the area under the flow curve of each cardiac cycle. For each systole a step was produced, the height of which was proportional to the stroke volume. To facilitate inspection, the deflections indicating the stroke volumes of the two ventricles were adjusted until the amplitudes were approximately equal, even though the aortic flowmeter did not register the flow through the coronary arteries. In some records, the cardiac output was indicated continuously by means of an integrator circuit with a long time constant (figs. 3 and 4). In the experiment illustrated in figure 5, cardiac output was indicated by an integrator that returned to the baseline every 2.5 seconds (stroke flow integrated).

The flowmeters and cannulas were installed under aseptic conditions and the experiments were conducted from 5 to 30 days after the surgery. Five of the eight dogs were active and appeared healthy within a few days after surgery; the other three remained lethargic and displayed rapid and forced breathing. Previous experience had indicated that these signs were characteristic of postoperative atelectasis, usually with hydrothorax. This diagnosis was confirmed by thoracentesis and by postmortem examination in these animals. Experiments were conducted on these three dogs to determine the effect of pulmonary collapse on the functioning of the right and left ventricles. In one, a pressure drop of some 40 mm Hg within the main pulmonary artery was demonstrated by withdrawing a catheter and was ascribed to obstruction of the pulmonary artery by angulation of the flow section. These studies of ventricular function under abnormal thoracic conditions pro-
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duced most illuminating results. In the animals which appeared healthy, the lungs were well inflated, the pleural spaces were free of fluid, and the pulmonary artery and aorta were widely patent.

Results

Typical wave forms of the instantaneous flow velocity through the pulmonary artery and aorta in a healthy dog are illustrated in figure 1 (dog V22). The ejection patterns from the two ventricles had distinctive differences in the healthy dogs. Ejection from the right ventricle generally preceded the onset of outflow from the left by intervals ranging up to 0.02 seconds. In no case did left ventricular ejection begin before right ventricular ejection, although the patterns sometimes appeared to begin simultaneously (fig. 1, dogs V34 and V44).

The right ventricular ejection pattern displayed a gradually rising flow, reaching a rounded peak in midsystole. Left ventricular outflow rose abruptly to a sharp peak in early systole and then declined toward zero flow, slowly at first and then more rapidly. At the end of systole, left ventricular ejection was terminated abruptly by a retrograde surge of blood, apparently associated with a snapping closure of the aortic valve. Right ventricular ejection continued after left ventricular outflow had ceased.

The temporal relations between the outflow pattern and the change in the diameter of the left ventricle are illustrated in figure 1 (dog V34). The ejection velocity reached a peak early in systole when the diameter was diminishing most rapidly. During late systole, the left ventricular diameter decreased more gradually as the ejection slowed. Thus, the ejection patterns are consistent with the changing ventricular diameter to the extent that a single dimension can serve as an indicator of the change in the volume of the ventricle.

The peak in the ventricular systolic pressure rose rapidly during the isovolumetric contraction period and during early systole when the ejection velocity increased most rapidly (fig. 1, dog V44). The pressure then leveled off and descended during the later portions of systole as the ejection slowed. The patterns of right and left ventricular pressure are very similar in wave form but very different in amplitude. Systolic pressure was much lower in the right ventricle than in the left, reflecting the lower hydraulic impedance to flow through the pulmonary circulation as compared with the systemic circulation. On this basis the peak ejection velocity of the right ventricle might be expected to be higher and occur earlier. The slower ejection of the normal right ventricle must, therefore, reflect differences in either the architecture or the effective rate of myocardial shortening of the two ventricles.

The ventricular ejection patterns of the healthy dogs differed from those of the dogs with hydrothorax and atelectasis as illustrated in figure 2. In each animal with pulmonary atelectasis, the right ventricular ejection pattern displayed an abrupt rise in flow velocity, reaching a peak in early systole. The velocity then descended toward the zero level, gradually at first and then more quickly. In addition, systole terminated with a retrograde surge, which was more abrupt than is observed under more nearly normal conditions. Thus, the right ventricular ejection pattern altered to conform more closely to the left ventricular ejection pattern under conditions in which the outflow resistance was increased (i.e., pulmonary atelectasis and pulmonary stenosis).

EFFECTS OF RESPIRATORY ACTIVITY

In the dogs that appeared healthy, the flow velocity and stroke volume (integrated flow) from the pulmonary artery and aorta changed very little during normal respiratory activity. Pulmonary flow reached higher levels during the inspiratory phase. Although these small respiratory fluctuations in the output of the two ventricles appeared to be almost exactly in phase, careful examination indicated that the right ventricular changes preceded the left ventricular fluctuations by one cardiac cycle in most instances. In contrast, flow patterns recorded from the animals with hy-
The instantaneous flow velocity patterns from the pulmonary artery are generally rounded with peak flows in midsystole in a healthy dog (V22). Aortic flow reaches a much higher peak in early systole and has a much sharper retrograde surge at the end of systole. The relations between the flow patterns and left ventricular diameter (V34), and between flow and right and left ventricular pressures (V44) are shown.

A hydrothorax and pulmonary atelectasis consistently revealed greatly accentuated fluctuations in outflow from both the right and left ventricle. These greatly exaggerated fluctuations were nearly 180 degrees out of phase with the right ventricular stroke volume approaching minimal values at times when left ventricular stroke volume approached peak levels (fig. 2B). As the hydrothorax was alleviated by thoracentesis, the respiratory fluctuations in the ventricular stroke volumes became attenuated and were almost in phase (fig. 2C). In three animals, injection of fluid into the thorax produced greatly augmented respiratory fluctuations, which were again out of phase.

CHANGES IN POSTURE
When a dog is induced to stand erect, the long hydrostatic columns of blood should cause distention of the dependent veins, so that "venous return" and stroke volume would be transiently reduced, first in the right ventricle and then in the left. Records obtained as a dog was elevated into an erect position and lowered again to the normal standing position are illustrated at slow paper speed (0.25 mm./sec.) in the left hand column of figure 3. On an expanded time scale (50 mm./sec.), the changes in right and left ventricular output could be followed stroke by stroke. A progressive fall in stroke volume in both ventricles accompanied the increase in heart rate. There was, however, a slight discrepancy in stroke volume between the two ventricles, as designated by arrows 1 and 2 in figure 3. When the animal was permitted to resume the standing position, the sequence of ventricular adjustments was similar, but there were more examples of reciprocal changes in ventricular stroke volume.
onset of increased right ventricular output in advance of the left ventricular adjustment was only occasionally observed in this series of experiments.

**RESPONSES TO EXERCISE**

An abrupt onset of exercise without previous warning might produce a transient imbalance between right and left ventricular outputs. A highly trained dog, standing quietly on a motor-driven treadmill, can be induced to exercise almost instantaneously and unexpectedly by the mere closing of a switch.

The sequence of events at the onset of such an exertion was analyzed, stroke by stroke, in dogs with flowmeters on the pulmonary artery and aorta. The instantaneous flow patterns in the pulmonary artery and aorta, as well as the integrated stroke volume records, are presented for comparison in figure 4. In general, the stroke volumes of the two ventricles increased together at the onset of exercise, but on any record occasional cycles showed ventricular stroke volumes which varied in opposite directions (see numbered arrows) without any consistent pattern.

The changes in stroke volume of the two ventricles were so evenly matched that it
The pulmonary and aortic flow velocity and right and left ventricular stroke volumes are all greatly reduced transiently when the dogs assume an erect position. These changes all occur simultaneously and accompany a transient tachycardia such that the cardiac output remains unchanged. A similar effect of smaller magnitude occurs during the return to normal standing position.

would be difficult to state with confidence that either ventricle responded before the other. In many different exercise responses at treadmill speeds up to 4 m.p.h. on grades up to 12 per cent, there was no obvious tendency for the output at the right ventricle to increase before that of the left.

During the exercise responses illustrated in figure 4, the heart rate reached a high peak abruptly after the treadmill started and then diminished transiently before ascending to a sustained plateau. During the wide excursions in heart rate, the stroke volume varied reciprocally so that the cardiac output rose smoothly and progressively.

When an increased "venous return" was experimentally induced by a rapid intravenous injection of saline, the right ventricular stroke volume increased progressively for a few beats and then diminished. The left ventricular output increased similarly after a lag of at least three cardiac cycles. This observation demonstrates that a phase lead of the right ventricle can be readily detected by the techniques employed here and, in fact, was noted during abnormal respiratory activity (pulmonary atelectasis) and during experimentally accelerated "venous return." The expected phase lead of right ventricular output did not occur during any of the responses to exercise observed in this series.

If increased cardiac output is initiated by increased "venous return," the inflow into the right ventricle through the inferior vena cava should be augmented before outflow from the left ventricle increases. In the experiment illustrated in figure 5, three different exercise responses were recorded at three
different paper speeds. In the record at slow paper speed (0.1 mm./sec.) showing the entire exercise response, the changes in all variables appear to be completed very promptly. In the response recorded at faster paper speed (10 mm./sec.) the heart rate and peak instantaneous flow velocity increased over several cardiac cycles. The increase in cardiac output is indicated by the steeper slope of the integrated stroke flow record. Large fluctuations in right atrial pressure and inferior vena caval flow appeared at the onset of exercise and may have been associated with the vigorous respiratory excursions, which lasted an interval equivalent to two or three cardiac cycles. At the end of that time the
inferior vena caval flow was sustained at a higher mean level, and the slope of the integrated stroke volume record was steeper (see dotted line in middle of record). Thus, right ventricular inflow and left ventricular output appeared to increase almost simultaneously. Faster paper speeds did not permit more accurate timing because of the fluctuations in the vena caval flow. However, the increase in cardiac output at the onset of exercise could not be clearly ascribed to accelerated inflow into the right ventricle, or to increased “venous return.” Furthermore, a phase lead of right ventricular adaptation in advance of left ventricular adjustments could not be established by these observations.

**Limitations of the Experiment**

All but three of the dogs studied in this series of experiments were active and appeared healthy at the time of the experiments (i.e., presented no obvious evidence of malfunction). However, all the dogs had recently undergone thoracic surgery and could not reasonably be regarded as “normal,” even 20 or 30 days after the operation. In the best of these preparations, there was no abnormal collection of fluid in the chest and adhesions of pleura and pericardium were minimal. However, all showed evidence of the previous surgical manipulations within the thorax. In three dogs, frank thoracic pathology was present in the form of rather severe hydrothorax and pulmonary atelectasis. Furthermore, demonstrable obstruction in the pulmonary artery had been produced in one dog by angulation of the plastic flow section. In the other animals, the aortic and pulmonary arteries were widely patent within the flow sections, although the cross-sectional area had been slightly reduced to prevent changes in circumference by the pressure pulses. Coronary
flow was not included in the output of the aortic flowmeter.

The differences in wave form between right and left ventricular ejection patterns cannot be ascribed to deficiencies in the ultrasonic flowmeters, which were identical at the two sites and had frequency responses flat to 30 c.p.s. or above. Right ventricular ejection patterns were distorted to resemble wave forms typical of the left ventricular outflow when the pulmonary resistance was increased (i.e., by pulmonary atelectasis and pulmonary stenosis).

The sudden and unexpected starting of a treadmill is a desirable method of producing an abrupt response to exercise but is likely to cause a startle reaction which could complicate the picture to some extent. For example, the transient increase in the heart rate at the onset of exercise was not characteristically observed in earlier experiments in which the treadmill was accelerated rapidly rather than started at full speed by the switch (see fig. 4). Furthermore, in trained animals, the ventricular performance often changes before the exercise when the slightest clue is given that the treadmill is about to be started.

Extrapolations

The observation that the stroke volumes of the right and left ventricles change almost, if not precisely, in phase under different conditions was unexpected, since it was contrary to popular opinion based on many studies on anesthetized thoracotomized animals. Since pulmonary atelectasis apparently caused the ventricular responses to be greatly accentuated and almost 180 degrees out of phase, it is tempting to assume that this abnormal situation is common to standard open-chest dog preparations.

The precise balance between right and left ventricular outputs suggests that the pulmonary circulation may normally offer so little resistance to flow that even a slight increase in the output of the right ventricle is promptly reflected in flow from the left. Interposing abnormally high resistance between the two ventricles in the form of pulmonary atelectasis or pulmonary obstruction accompanied by forced breathing consistently produced cyclic right and left ventricular responses that were grossly out of phase.

A number of factors could operate to cause the ventricular stroke volumes to change together. Both ventricles beat the same number of times per minute and have corresponding changes in diastolic filling interval. Furthermore, during cardiovascular adjustments both ventricles are likely to be bombarded by discharges along sympathetic nerves to produce changes in the performance of the myocardium.

Summary

The right ventricular ejection pattern is characterized by early onset, gradual rise to peak flow velocities in midsystole and gradual return to baseline. Left ventricular ejection begins very shortly after right ventricular systole, abruptly reaches peak flow, and diminishes during the remainder of the systolic interval, terminating with a brief, sharp, retrograde surge as the aortic valves close. The duration of ejection is shorter in the left ventricle than in the right. In healthy dogs, respiratory activity produces very slight fluctuations in right and left ventricular output that are almost in phase. In dogs with hydrothorax and pulmonary atelectasis, the right and left ventricular outputs fluctuate in greatly exaggerated fashion with each forced respiratory effort. Under such conditions, the changes in right and left ventricular stroke volume are nearly 180 degrees out of phase. Pulmonary outflow resistance is increased and the right ventricular ejection pattern closely resembles those generally characteristic of the left ventricle. Assume that the erect posture causes cardio-acceleration and large changes in right and left ventricular stroke volume, which are essentially synchronous. Treadmill exercise with sudden and unexpected onset produces changes in the outputs of the right and left ventricles without obvious lead or lag of one ventricle over the other. These experiments do not support the concept that changes in "venous return" are dominant mechanisms inducing alterations in cardiac output.
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