The close anatomical relationship between the two ventricles of the heart implies some kind of functional effect upon each other. This effect could be manifested in at least two different ways. An indirect "hemodynamic" effect could be exercised by one ventricle upon the other, because differences in the output of the one ventricle may affect the input to the other ventricle. A direct or "mechanical" effect could also be expected because of anatomical connections. Common muscular bundles encircle both ventricles and it is difficult to believe, for example, that a change in the strength of contraction could be limited strictly to the part of these bundles that extends over one of the ventricles. The degree of distension of the wall of one ventricle may be expected also to affect the function of the other ventricle.

A limited number of studies has been published, referring to the effect of by-passing or distending the right ventricle on the dynamics of the circulation. Grodins et al. replaced the right ventricle by a Sigmamotor pump in order to study the response of systemic pressure to variations of right heart output. They found that the arterial systemic pressure was a positive function of the pump output. Guyton et al. used a partial right heart by-pass preparation and pointed out the dependence of cardiac output upon several heart parameters. On the other hand, Taquini et al. constricted the lumen of the pulmonary artery by over 70% and noted a reduction of cardiac output and a decrease of systolic pressure but an increase of the diastolic pressure in the left ventricle. In general, the work previously done on the subject refers mainly to hemodynamic relationships between the two ventricles of the heart and the systemic or pulmonary circulations.

In the first part of this study, a right heart by-pass preparation was used. In the second, the effect of increasing the pressure head in the pulmonary artery was studied. In both groups, the input to the left ventricle was kept constant. Thus, an indirect hemodynamic effect upon the left ventricular function could be excluded, while the right ventricle was being either emptied or distended.

**Methods**

Data were obtained on 20 mongrel dogs with a weight range of 17 to 25 kg. The animals were anesthetized with chloralose, 40 mg/kg, and urethane, 400 mg/kg, given intravenously 30 minutes after the intramuscular injection of morphine, 4 mg/kg. Coagulation was prevented with heparin, 5 mg/kg, given initially and followed by 10 mg each hour thereafter. The chest was opened by a left thoracotomy in the third intercostal space under intermittent positive pressure breathing.

Figure 1 is a diagram of the preparation used. Ascending aortic and left ventricular pressures were recorded continuously. Left atrial and venous pressures were also read. The azygos vein was ligated and loose ligatures were placed around the inferior and superior venae cavae. Both venae cavae were cannulated through the right jugular and left femoral veins by means of catheters leading to a venous reservoir. The right atrium was also cannulated with a tube leading to the same reservoir. All these tubes were clamped until the next stage of the experiment.

The main pulmonary artery was clamped at two different points, for a period not exceeding two minutes. During this time the pulmonary artery was divided between the two clamps and both ends were cannulated. The tube from the proximal end was connected to the venous reservoir. From the reservoir the blood was pumped to the distal part of the pulmonary artery by a Sigmmotor pump.

In this way, by tightening the ligatures around the veins cavae and unclamping the tubes coming from them and the right atrium, a total right heart by-pass could be established. The by-pass circuit could be interchanged with "control" circulation, when the reverse manipulations were applied. During both stages, the blood flow to the lungs could be maintained constant or it could be changed at will, by regulating the pump output. After a short period of equilibration, the blood flow to the lungs was considered as indicating the input to the left ventricle.

The venous catheters were large enough to make the resistance introduced by them to the venous return relatively unimportant. The venous pressure during the by-pass was not increased greatly. The superior caval catheter had necessarily a small flow probably from arterial anastomoses because the arterial supply to the upper part of the body was obstructed by ligating the brachiocephalic and subclavian arteries. The ligation of these arteries may have interfered, by reason of cerebral ischemia, with the innervation of the heart and possible pressoreceptor reflexes via the vagi from the right heart and the pulmonary artery. As indicated below it was necessary to sympathectomize a group of animals, in order to be sure that any reflexes through this system were abolished.

The mean pressure head of the blood flowing into the proximal part of the pulmonary artery was kept at 17 cm of water by a constant pressure reservoir (fig. 1). In 10 dogs the level of this reservoir was raised in steps up to 50 cm and acute distension of the right ventricle was produced. In all experiments the output of the pump and therefore the input to the lungs was kept constant.

The thoracic aorta was opened between two clamps and both parts of this artery were cannulated. The tube from the proximal part led to a second constant pressure reservoir, and the blood from this reservoir flowed through another tube to the distal part of the aorta. The flow in the tube leading to the distal part of the aorta was measured by a rotameter. The level in the pressure reservoir was maintained constant by means of a mechanical feed-back system (fig. 1). The brachiocephalic trunk and the left subclavian artery were ligated. Under these conditions the blood flow indicated by the rotameter represented the left ventricular output, minus coronary artery flow. The flow from the tube in the right atrium indicated the coronary sinus flow.

The tubes of the extracorporal circuit were filled with dog blood. The temperature in the circuit was kept at approximately 38°C by dipping the reservoirs into a water bath with regulated temperature. A heat exchanger was also used. The temperature was checked by an electrical thermometer. Two electrodes of a pacemaker were attached to the right atrium. A constant rate was used during the experiment, with an impulse frequency at or above the spontaneous heart rate.

A four-channel Sanborn recorder was used to record aortic, left and right ventricular pressures, together with the electrocardiogram, which served to make certain that the heart was driven steadily by the electronic pacemaker. Sanborn inductance type pressure transducers were used. Artificial respiration was stopped during recordings, to avoid even transient changes in left ven-
tricular input or any interference arising from distension of the lungs. The recordings were repeated during right ventricular by-pass or during distension of the same ventricle. The left ventricular external work was calculated and plotted against left ventricular end diastolic pressure.

In 15 animals the lower cervical and upper thoracic sympathetic ganglia were excised, including the stellate ganglia on both sides. The vagi were cut also. Five animals were used for control observations in which the sympathetic chains were not excised. Both procedures, i.e., by-pass together with distension of the right ventricle, were studied in the five control animals and in five animals of the sympathectomized group. The right heart by-pass and the right ventricular distension were applied separately in five animals each, of the sympathectomized group.

Results

A. LEFT VENTRICULAR FUNCTION DURING RIGHT VENTRICULAR BY-PASS

One hundred fifty stroke work curves were obtained in ten sympathectomized animals and five animals of the control group, before and during right ventricular by-pass. The function curves were obtained by changing the left ventricular input with the Sigma-motor pump. During the by-pass the left ventricular end diastolic (LVED) pressure was increased, in comparison to the heart without by-pass and producing the same left ventricular stroke work.

The effect of right ventricular by-pass on left ventricular contractility, was determined in two ways: First, the left ventricular function curves, obtained before and during right ventricular by-pass, were compared. Second, the changes in the ratio between the maximal pressure attained during the isometric systole and the left ventricular isometric systolic time were followed, as they were affected by the right heart by-pass.

When the heart rate was kept constant, the left ventricular function curves were displaced to the right during the by-pass (Fig. 2), indicating a decrease in contractility. Figure 3 presents the averaged curves obtained before and during the by-pass for the control experiments and the experiments done after excision of the sympathetic chains. The displacement to the right during the by-pass was present in all curves obtained. The excision of the sympathetic chains did not alter the results significantly.

Figure 4 shows the ratio between the maximal isometric pressure and the isometric systolic time plotted against the stroke work of the left ventricle, before and during the right ventricular by-pass. For the same stroke work, the rate of development of the isometric tension is reduced during the by-pass. The curves obtained from the other experiments indicate a constant change in the same direction. No significant difference was noted between the control group and the sympathectomized one.

B. LEFT VENTRICULAR FUNCTION DURING RIGHT VENTRICULAR DISTENSION

Ten sympathectomized dogs and five animals of the control group were observed for this part of the study. When the right ventricular end diastolic (RVED) pressure was increased above 10 mm Hg, the left ventricular end diastolic (LVED) pressure began to in-
CONTROL EXPERIMENTS

FOLLOWING SYMPATHECTOMY

\[ \text{LVED PRESSURE mmHg} \]

Average left ventricular function curves obtained during right ventricular by-pass and during normal circulation. The control group represents average values from five dogs (20 curves) while the post-sympathectomy curves were obtained from ten dogs (130 curves).

Rate of development of left ventricular isometric tension (dP/dt) plotted against left ventricular stroke work, during normal circulation (dots) and right ventricular by-pass (circles), in one sympathectomized animal.

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Discussion

In the present series of experiments, an attempt was made to evaluate the changes in performance of the left ventricle, while the input to it and the peripheral resistances were kept constant. The sympathetic ganglia were excised. Control experiments with an intact sympathetic system were also done, although the ischemic brain might have interfered with normal nervous reflexes. Under these conditions the right ventricle was either

FIGURE 5
Relationship between right and left ventricular end diastolic pressures during gradual distension of the right ventricle in one sympathectomized dog. The output of the pump to the peripheral part of the pulmonary artery was kept constant.

FIGURE 6
Rate of development of left ventricular isometric tension (dP/dt) plotted against the right ventricular end diastolic pressure, during gradual distension of the right ventricle in one sympathectomized animal. Output of the pump to the peripheral part of the pulmonary artery was kept constant. The direction of change was constant in all curves obtained under the same conditions from ten sympathectomized and five "control" animals.
The performance of the left ventricle of a normal intact dog's heart is known to be affected by biochemical and hemodynamic factors. As to the hemodynamic factors, the input to the left ventricle, as well as the peripheral resistances, exert important effects upon the performance of this ventricle.

There is some evidence that the functional condition of the right ventricle may also affect left ventricular function. Taquini et al. observed a rise in left ventricular diastolic pressure, when they constricted the pulmonary artery. It is noteworthy that the LVED pressure rose while the input to the left ventricle was presumably reduced by the constriction of the pulmonary artery. Unless reduced coronary flow is involved, this observation may indicate direct effect of right ventricular distension on the LVED pressure.

In the present study, the data indicate that left ventricular performance is affected by both the by-pass and also the distension of the right ventricle. In both conditions, left ventricular function curves indicate decreased myocardial contractility.

For a constant left ventricular input, the relationship of isometric time to maximal LVED pressure is given in Figure 8.

Left ventricular function curves obtained during right ventricular distension in one sympathectomized animal. Height of the blood level in the pulmonary artery reservoir was 17 cm (dots), 27 cm (squares) 37 cm (triangles).

"emptied" (by using the by-pass) or distended (by increasing the pressure in the pulmonary artery). The question investigated was whether the degree of filling of the right ventricle could change the left ventricular performance, not by changing the flow to the left ventricle but by directly or mechanically affecting the contraction of this ventricle.

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isometric pressure was affected by both the distension and the by-pass of the right ventricle. The reduction of the ratio dP/dt (Hill\(^n\)) observed during the by-pass, as well as while the right ventricular distension progressed, may also be considered as an indication of a decreasing myocardial contractility (Gilmore et al.\(^7\)). The reasons for using the isometric time-tension relationship as an index of myocardial contractility have been analyzed by Siegel et al.\(^8\)

As should probably be expected, the left ventricle seems to work more efficiently when the right ventricle maintains its normal function. Normal right ventricular blood volume may provide the left ventricle with the mechanical support that is necessary for the development of an efficient left ventricular contraction. If this volume is either increased or decreased, the left ventricular stroke work is accomplished with higher LVED pressures. A number of factors were controlled, namely, constant left ventricular input and outflow resistance, constant coronary perfusion pressure, constant heart rate, and absence of sympathetic system interference. Hence the findings described here may be due to the impaired activity of the muscular bundles encircling both ventricles, i.e., the deep sinospiral and superficial bulbospiral muscles. If this assumption is correct, the results probably indicate the existence of a direct mechanical relationship between the two ventricles of the heart. The functional significance of this relationship may have been underestimated, and needs further study. No significant difference was observed between curves obtained from control experiments and those from animals after excision of the sympathetic chain. This observation also favors a mechanical effect.

**Summary**

A dog heart preparation was used to in-
investigate the direct or mechanical effects of a right ventricular by-pass and of right ventricular distension on the performance of the left ventricle. Several other factors known to affect left ventricular performance were eliminated.

The data obtained indicate impairment of left ventricular function either by right ventricular by-pass or by right ventricular distension. It is suggested that this impairment may be due to a direct effect of the volume of blood contained in the right ventricle and of right ventricular function on the common muscle bundles that encircle both ventricles.

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Circ Res. 1965;17:484-491
doi: 10.1161/01.RES.17.6.484

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1965 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/17/6/484

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