The Question of the Function of the Right Ventricular Myocardium: An Experimental Study

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A laboratory study designed to investigate the independent pump-like function of the right ventricle of the dog heart is presented. Systolic and diastolic pulmonary arterial pressure fluctuations were used as the definitive criterion for pumping efficiency. Control pressure levels are compared with those recorded after complete damage, by electrosurgical coagulation, of the free right ventricular myocardium. The degree of damage so produced was ascertained from an analysis of electrocardiographic, physiologic and histologic observations. The results are discussed and explained in terms of a postulated mechanism based upon the architecture of the individual ventricular muscle bands.

The musculature of the right ventricle of the normal mammalian heart is generally considered to function as an efficient pump, alternately contracting and relaxing and thus transferring blood from the venous side of the systemic circulation to the pulmonary arterial side. This, supposedly, lowers pressures in the systemic veins and raises them in the pulmonary arterial circulation and assures an adequate pressure gradient for normal blood flow in both systems.1-4 On the basis of this concept it is reasonable to assume that a rise in the upstream (peripheral venous) and a fall in the downstream (pulmonary arterial) pressures will occur as a result of a decrease in the pumping efficiency of this chamber. The usual elevation in the peripheral venous pressure in clinical cases of dissociated right-sided heart failure is generally interpreted as a manifestation of such a unilateral deficiency.5-11 Much clinical12-14 and experimental15-18 evidence has accumulated in the past, however, which conflicts with this concept. Starr19 demonstrated that experimentally produced severe damage of the right ventricle of the dog heart did not effect more than a minimal rise in the upstream systemic venous pressure. This latter investigation gave impetus to the present studies which are concerned with the effect of similarly produced damage of the right ventricle on the downstream systolic and diastolic pressures in the pulmonary artery. These experiments demonstrate a maintenance of the control level of both these pressures as well as the systemic venous pressure after the production of complete coagulation and necrosis of the myocardium of this chamber. This seems to indicate that active contractions of the right ventricular myocardium of the dog heart are not absolutely necessary for efficient force-pump function of the right ventricle. An explanatory anatomic and physiologic correlative study is discussed.

Methods

Acute experiments were performed on healthy mongrel dogs anesthetized by an initial injection of 35 mg. per kilogram of body weight of sodium pentobarbital (Nembutal) and then placed in a supine position. With the attainment of surgical anesthesia the thorax was opened by a longitudinal, midsternal incision; thereafter the animals were sustained by intermittent positive pressure from an artificial respirator. A hammock-like sling was devised from the pericardial sac as a means of preventing positional changes of the heart during the course of the experiments. Mean systemic arterial pressure was recorded by a mercury manometer from a direct cannulation of the left carotid artery. A membrane manometer of the Hürthle type was employed for the determination of the systolic and diastolic pressure fluctuations in the pulmonary arterial system. This was accomplished by the in-
sition of a glass cannula into a stem branch of the left pulmonary artery. Heparin was used as the anticoagulant. Peripheral venous pressures were obtained by periodic observations of the meniscus of a saline hydrostatic manometer connected to the femoral vein. The arterial pressures were recorded continuously on smoked paper. The “zero” pressure of all systems was referred to the horizontal level at which the superior vena cava entered the right atrium.

After the allowance of a sufficient period of time for the animals to recuperate from the preliminary operative procedures, cautering instruments were periodically applied to the right ventricular musculature. Short intervals of time were permitted between applications for cardiovascular readjustments. In early experiments a soldering iron was used as the cautering agent and many repeated applications were necessary to produce extensive damage. In subsequent studies a high-frequency electrosurgical coagulator was resorted to in order to produce more severe and practically complete anatomic damage of the musculature. With the use of this latter method, which required fewer applications, it was necessary to exercise great care in order to prevent immediate and fatal ventricular fibrillation. This was accomplished by removing the electrosurgical cautery at the first indication of any irregularities in rhythm. Cauterization was produced in all experiments by a direct application of the cautery to the whole epicardial surface of the right ventricle; charring was restricted to the surface landmarks of the ventricle as determined by preliminary anatomic studies of normal dog hearts. Damage was inflicted to the maximum degree that could be obtained by the particular method used. With the use of the soldering iron this was determined by the seepage of luminal blood through small fissures occurring in the charred muscle; with the electrosurgical cautery a definite period of time, as determined by preliminary studies, was allowed for each cauteration in order to produce destruction of the entire thickness of the wall. It was necessary to raise the heart momentarily in order to reach its posterior portions with the cautery instruments.

In several experiments the right coronary artery was dissected free from its supporting tissues and ligated near its aortic origin. Damage was then inflicted to the right ventricle of the 2 animals that survived this procedure. In two other experiments superficial damage to the left ventricular musculature was induced subsequent to the production of maximum damage to the right side.

Unipolar electrocardiographic tracings, utilizing a nonpolarizing electrode, were recorded directly from several selected points representing the whole area of the right ventricular epicardium. These were taken initially as controls and later, from the same selected points, in 7 animals after myocardial damage was completed by electrosurgical coagulation.

The degree of damage so produced in these studies was ascertained at the termination of each experiment by a gross and microscopic post-mortem examination. The development of any peripheral or pulmonary venous congestion was also noted at this time.

**Observations**

The following results are based on observations taken from the 21 experimental animals which survived the drastic procedures employed. In 9 animals the soldering iron was used, in 10 the electrosurgical coagulator, and in 2 a ligation of the right coronary artery followed by electrosurgical coagulation. In all these right ventricular damage was carried out to the maximum degree.

In all twenty-one experiments the systolic and diastolic pressures in the pulmonary artery remained surprisingly constant from the initial control period to the time following the completion of the damage. As can be seen in figure 1, which is a tracing from a representative experiment, these pressures fluctuated between approximately 40 mm. Hg systolic and approximately 10 mm. Hg diastolic initially as well as after the final charring period. These values are closely similar to those obtained in all animals studied and are consistent with the results reported by others. In some instances the animals were allowed to survive as long as six hours after maximum right ventricular damage without any recorded change in these pressures. Following this extensive damage the peripheral venous pressure did not show more than a 1 or 2 cm. of water rise or fall in any experiments. This corroborated Starr's previous observations. No significant changes were observed to have occurred in the level of the mean systemic arterial pressures. A small drop in these latter pressures usually occurred following the cannulations and the chest surgery; however, a steady state was reached prior to the infliction of damage to the heart and from then on no change was noted.

It was further noted that following this extensive destruction of the musculature of the right ventricle the free wall of this chamber failed to show any signs of active contractions. This occurred without any evidence of altera-
tion in the systolic or diastolic pulmonary arterial pressures.

In these instances, and after a few cauteriza-

The only significant changes in pressures were observed in the two experiments in which the left ventricle was moderately damaged sub-

tions of the epicardium of the left ventricle the pressures in both the pulmonary and systemic arteries began to fall simultaneously.
and finally, with the death of the animal a few minutes later, they reached the static level.

Electrocardiographic tracings recorded directly from the epicardial surface of the normal right ventricle by means of a nonpolarizing unipolar exploring electrode displayed ventricular complexes of the type reported by Wilson and his associates. These showed the same variations in form as was to be expected from points from the various regions of the epicardial surface of the right ventricle from which they were obtained. In general they all showed characteristic R- and S-wave deflec-
outline but which closely resembled each other regardless of the position of the exploring electrode (fig. 2, lower series). These were characterized by deep QS deflections, representing the potential variations of the cavity of the right ventricle, and upwardly convex and upwardly displaced RS-T segments. This latter change, in all probability, had its origin from the zone lying between the destroyed muscle of the wall of the right ventricle and the normal undamaged left ventricle.

Gross and microscopic postmortem examinations showed that over 75 per cent necrosis of the wall of the right ventricle was produced in experiments in which the soldering iron was used as the cautering agent (fig. 3). With the use of the electrosurgical coagulator these studies demonstrated coagulation necrosis of the entire area and thickness of the wall of the right ventricle including the subendocardial myocardium so that the free wall of the right ventricle showed complete anatomic damage. No evidence of peripheral venous or pulmonary congestion or edema was noted at this time in any of the experiments including the ones in which the left ventricle was damaged subsequent to the right. In these latter experiments postmortem observations revealed small circumscribed areas of necrosis of the free wall of the left ventricle due to the few applications of the cautery prior to death of the animal.

**DISCUSSION**

It can be stated without equivocation that as a result of the cautery procedures employed in these acute experiments the right ventricular myocardium was thoroughly damaged anatomically, and no longer functioned as an actively contracting organ. This is apparent from the postmortem gross and microscopic findings of coagulation necrosis of the entire free wall of the right ventricle and the conspicuous absence of action potentials (electrocardiographie R waves) which normally originate from activity of the right ventricular myocardium.

Before discussing the results of these experiments in terms of function of the right ventricle the value of systolic and diastolic pulmonary arterial pressure changes as an index of the efficiency of the right ventricular pump must be considered. It is well known that the hemodynamics of a pulsating circulation system depends upon the discharge volume of the pump (stroke output), and upon the elasticity, and peripheral resistance of the receiving conduits and upon the viscosity and volume of the circulating fluid. Changes in any of these factors can alter the size and configuration of the pulse pressure curves recorded. The stroke output of the pump, of course, is directly dependent upon the amount of fluid it receives and the efficiency of its forcepump-like contractions. The manner in which these factors affect the systolic and diastolic arterial pressure fluctuations is adequately discussed in textbooks of physiology. It is important to state here, however, that with a decrease in pump stroke output (other factors remaining constant) there would result a greater drop in the systolic pressure than in the diastolic pressure. The net effect would be a decrease in the pulse pressure. The reason for this less pronounced effect on the diastolic pressure is that as a result of the lower pressure at the end of the systolic ejection period (owing to the decreased stroke output) the pressure gradient is less throughout diastole and therefore less blood will “run off” through the peripheral arterioles. If the heart, which is the source of the energy driving the fluid through the system, should entirely cease its function as a contracting pump organ, its stroke output will be reduced to zero and the pressures in the receiving arterial system will then be dependent upon but less than the head of pressure in the upstream peripheral veins.

This line of reasoning can now be applied, along with a consideration of the other hydrodynamic factors, to the right ventricular pump and the pressure fluctuations in the pulmonary arterial system. Normally, since the right atrial pressures are lower than those recorded in the pulmonary arteries, it is obvious that the vis a tergo of the left ventricular pump, along with the intrapleural respiratory pressure changes and the skeletal muscle “squeeze,” cannot be responsible for the higher pulmonary arterial pressures. Because of the decreasing pressure gradient from the right ventricle to
the left atrium, the pulmonary arterial pressures must therefore be dependent upon pressure changes occurring in the cavity of the right ventricle. According to our classic concepts the independent active pumping contractions of the right ventricle are entirely responsible for these intracavity pressure changes which thus in turn maintain the stroke output into the pulmonary arterial system. The pressure fluctuations in this system vary as the product of the stroke output of the right ventricle and the peripheral resistance offered by the pulmonary arterioles. Increases in the latter can no doubt compensate for minor decreases in the stroke output and still maintain an adequate mean pressure level. Under these conditions, however, the pulse pressure will decrease and with eventual reductions of the right ventricular stroke output to zero, the pulsatile flow will cease altogether and the pulmonary arterial pressures will then be dependent upon the level of the filling pressure in the upstream peripheral veins. Likewise, regardless of the high degree of elasticity possessed by the pulmonary arterial conduits (which thus affords relatively large stroke volume changes with minor pressure changes) the ultimate pulse pressure will be directly dependent upon the stroke volume of the right ventricle.

In so far as no recordable changes were observed to occur in the systolic, diastolic, or pulse pressures in the pulmonary arterial system following the production of this extreme degree of right ventricular damage, the question is raised as to the source of energy responsible for the output of the right ventricle and the maintenance of these pressures. The artificial respirator used in these studies was not the source, since the pulmonary arterial pressure fluctuations were not synchronous with the rhythm of the respirator. It was further observed that when the respiratory pump was momentarily turned off the pressure fluctuations in the pulmonary arteries continued. The relatively constant systemic venous pressure, which was well below the recorded pulmonary arterial pressure in all experiments, excludes the possibility that the latter pressures were maintained by the vis a tergo of the left ventricular myocardium and the other factors which tend to maintain the systemic venous pressure.

The results observed in the two experiments in which the left ventricle was superficially cauterized subsequent to maximum damage of the right ventricle seem to indicate that the energy expenditure of the contracting left ventricle is directly responsible for the maintenance of these pulmonary arterial pressures. The basis for a probable explanation of the manner in which this occurs can be seen from a study of the architecture and function of the four main, distinct, and independently contracting ventricular muscle bands comprising musculature common to both ventricles. The structural relationships between these muscle bands are similar in both human and canine hearts. These include the superficial sinospiral and bulbospiral muscles (fig. 4) and the deep bulbospiral and sinospiral muscles (fig. 5). Each of the superficial muscles partially encircles both ventricles, but considered together they completely envelop, in a supplementary manner, the whole surface of the heart. The deep sinospiral completely encircles both ventricles but splits, at the posterior interventricular groove, in such a manner that the greater mass of its fibers, though continuous with the rest of the muscle, projects deeply and enters into the formation of the interventricular septum. The lesser mass of this muscle passes more superficially and fuses with the previously described superficial muscles. The deep bulbospiral is the only muscle band which is confined to one ventricle, namely, the left. It has been postulated by Robb and Robb that this muscle band is important, owing to its sphincteric arrangement around the base of the left ventricle, in completing ventricular ejection and maintaining systolic pressures in the aorta by supporting the column of blood ejected. These same investigators state that “the deep sinospiral muscle forms the main mass of the right ventricle and must be responsible for the maintenance of the pulmonary circulation.”

The importance of these muscle masses as a probable explanation for the results of these experiments is based upon (1) their encircling
arrangement—enveloping both ventricles in one continuous sweep—and (2) the fact that the portion of these muscles making up the

developed during contraction of the undamaged, thicker, portion is mechanically transmitted, through this anatomic continuity, to the damaged, nonfunctioning, thinner, right ventricular portion. Since the mass of the left

left ventricle is larger, thicker, and more powerful in its contraction than the portion making up the thinner right ventricle. Because of this encircling arrangement the tension de-

Fig. 4.—Posterior view of the superficial bulbospiral (A, top) and the superficial sinospiral (B, bottom) muscles. Note how the two muscles, considered together, completely envelop the heart in a supplementary manner. M represents the mitral valve; A, the aorta; T, the tricuspid; L is the left and R the right ventricle; P, AP, and PPM are the papillary muscles. (This figure and figure 5 were obtained through the courtesy of Dr. J. S. Robb, from Robb, Hiss, and Robb: Am. Heart J. 10: 289, 1935.)

Fig. 5.—Posterior view of the deep bulbospiral (A, top) and the deep sinospiral (B, bottom) muscles. Note that the deep bulbospiral muscle is limited to the base of the left ventricle and that the deep sinospiral completely encircles both ventricles and also forms the muscular interventricular septum. Labels same as in figure 4.

ventricular portion is far greater than that of the right ventricular portion it is assumed that the contribution of each towards the total tension developed during contraction of the muscles as a whole is also of this proportion. Thus, according to this postulation, removal of the thinner right ventricular contribution
does not materially subtract from the total tension developed during systole. Therefore, contraction of the left ventricular portion transmits tension to the right ventricular portion with a resultant increase in the cavity pressure of this latter chamber and consequent ejection of blood into the pulmonary arterial system. When the thicker, more powerfully contracting, left ventricular portion is damaged, subsequent to maximum damage of the right ventricular portion, the ejection of blood from both chambers decreases with a resultant simultaneous drop in the pressures in both arterial systems.

**Summary and Conclusions**

In acute experiments performed on 21 dogs the musculature of the right ventricle was directly and completely damaged by cauterizing instruments. No changes were observed to occur in the peripheral venous and systolic and diastolic pulmonary arterial pressures following this degree of damage as compared to the control level of these pressures recorded prior to the cauterizations. The degree of damage produced was ascertained from direct unipolar electrocardiographic tracings recorded during the study and later by gross and microscopic postmortem observations.

Despite this complete inactivation of the right ventricle the myocardium of the left ventricle apparently mechanically transmitted its energy, through the continuity of the circumscribing individual ventricular muscle bands, to the right ventricle so that this latter chamber passively functioned as an efficient force pump.

An actively functioning right ventricle is therefore not absolutely necessary for the maintenance of a normal pressure gradient in the pulmonary arterial tree. It is also apparent that the function of the two ventricles cannot be dissociated in an independent manner since the architecture of the distinct myocardial bands makes it mandatory for an integrated and unified function of both chambers.

The relationship of these experiments to the dynamics of congestive heart failure is yet to be considered. Perhaps chronic experiments performed along similar lines may offer interesting observations towards the solution of this perplexing problem. Recent observations during clinical investigative studies seem to parallel the conclusions from the observations in these present experiments.

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**References**


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