Performance of the Right Ventricle under Stress: Relation to Right Coronary Flow

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ABSTRACT Right ventricular performance was studied relative to right coronary artery flow in the chloralose-anesthetized, open chest dog. The right coronary artery was cannulated for measurement and control of flow and pressure. Under control conditions, right coronary artery occlusion caused no change in cardiac output, or right and left ventricular pressures, although right ventricular contractile force fell markedly. With right coronary artery flow intact, incremental pulmonary artery obstruction caused a corresponding decline in cardiac output and elevation of right ventricular end-diastolic pressure with eventual total right ventricular failure and systemic shock. With right coronary artery occlusion, identical degrees of pulmonary artery obstruction resulted in more pronounced changes in cardiac output and right ventricular end-diastolic pressure with right ventricular failure occurring at a much lower level of right ventricular stress.

However, with right coronary artery flow intact, the right ventricular decompensation induced by pulmonary artery obstruction, could be reversed by raising right coronary artery perfusion to levels above normal, thus increasing right ventricular performance and restoring cardiac output.

We conclude that right ventricular failure and resultant systemic hypotension due to severe pulmonary artery obstruction can be reversed simply by right coronary artery hyperperfusion, and that, although a normally contractile right ventricular free wall is not essential to maintain cardiac performance at rest, during right ventricular systolic stress, over-all cardiac performance becomes increasingly dependent on the right ventricle. The data further imply that increased myocardial impingement on right coronary artery flow during systole in right ventricular hypertension may be an important factor leading to right ventricular failure.

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INTRODUCTION

Fineberg and Wiggers (1) postulated that "circulatory failure following obstruction of the pulmonary circuit had no other cause than fatigue of the right ventricle." However, the mechanism of this fatigue has yet to be clearly defined. This takes on added significance relative to the clinical problem of massive pulmonary embolization with acute obstruction to right ventricular outflow. Early studies in which the right ventricle was rendered nonfunctional by coagulation (2–4) implied that this chamber was merely a passive conduit that played a very minor contractile role in maintenance of cardiac function. More recent studies have provided indirect evidence that right coronary flow becomes increasingly important as the right ventricle is stressed in systole (5–7). The present work was designed to study more directly the relationship between performance of the right ventricle and its supply of blood from the right coronary artery under normal conditions as well as those of systolic overloads leading to acute right ventricular failure.

METHODS

Experiments were performed on healthy mongrel dogs of both sexes, ranging in weight from 21 to 30 kg. After induction of anesthesia with a small intravenous injection of thiopental, animals were anesthetized with an intravenous infusion of a warm solution of alpha chloralose (80 mg/kg). Supplementary doses of chloralose were given during the study to maintain a relatively uniform state of anesthesia. Respiration was maintained at a steady rate by a pressure-cycled respiratory pump connected to a cuffed endotracheal tube. Pure oxygen was administered to maintain optimal arterial oxygen saturation throughout the experiment.

A right thoracotomy was performed, and the heart was suspended in a pericardial cradle. Right and left ventricular

pressures were measured at the midpoint of the right atrium through short polyethylene catheters inserted transmurally via the ventricular apices and connected directly to Statham P23Db pressure transducers. With the exception of two of the studies in which the indicator-dilution technique was used, cardiac output was determined by an electromagnetic flow meter with the flow probe on the proximal aorta. The flow meter technique was chosen in the majority of studies because of the advantage of beat-to-beat analysis of cardiac output as well as greater accuracy during the low flow states. Right ventricular contractile force was measured by an isometric strain gauge arch[1] sutured to the central area of the right ventricular free wall. The gauge was oriented in series with the epicardial fibers, attached at 40% stretch so that forces recorded approximated the active contractile property of the subjacent area of myocardium (8). The primary purpose of the strain gauge arch data was to assess contractile changes occurring in the right ventricular free wall and produced by right coronary artery occlusion only in the resting state. No attempt was made to interpret their data during subsequent pulmonary obstruction because of the obvious marked right ventricular dilatation and consequent, inevitable changes in anchoring sutures. Balloon catheters were positioned fluoroscopically in the proximal segments of the right and left pulmonary arteries. The series of experiments comprised the following two groups.

Group 1 (five dogs). In these studies, the right coronary artery was isolated and dissected free of epicardial fat at its point of origin from the aorta. A silk snare was placed around the artery with special care being taken to place it proximal to the ventricular branches but distal to the sinus node branch. In many animals this was a critical step in the procedure due to very high origins of the sinus node and conus branches. In most of these animals, significant and persistent atrial ectopic arrhythmias, sinus bradycardia, or atrioventricular conduction disturbances occurred during the dissection and necessitated discarding these animals from the study. Thus, in none of the 10 animals reported in whom a successful study was carried out was there a perceptible alteration in the control heart rate caused by occlusion of the artery.

The right ventricle was subjected to graded degrees of systolic stress by partial inflation of two balloons in the proximal right and left pulmonary arteries. In this manner, increments in right ventricular systolic pressure of 10 ± 2 mm Hg were imposed to a point where acute right ventricular failure occurred characterized by progressively rising right ventricular end-diastolic pressure, a falling cardiac output, and marked systemic hypotension. Right and left ventricular pressures, right ventricular contractile force, and aortic flow were measured with unmodified right coronary artery flow and during temporary total occlusion of the right coronary artery.

Group 2 (five dogs). The model employed in this group of experiments is shown diagrammatically in Fig. 1. The right coronary artery was directly cannulated by means of a specially designed metal cannula which was passed from the right carotid artery through the ascending aorta into the right coronary ostium, where it was tied in place with a proximal suture. The cannula was perfused from a peripheral artery. Flow through the system was measured by an extracorporeal flow transducer,[2] and perfusion pressure was measured by a Statham P23Db transducer, both of which transducers were attached to the perfusing circuit. A peristaltic pump was also connected to the circuit to allow control of flow and perfusing pressure so that with activation of the pump, spontaneous flow was immediately converted to controlled flow. The area of right coronary arterial supply was evaluated after the experiments by postmortem perfusion of the right coronary cannula with carmine blue. In each case, the right coronary artery was shown to supply only the free wall of the right ventricle.

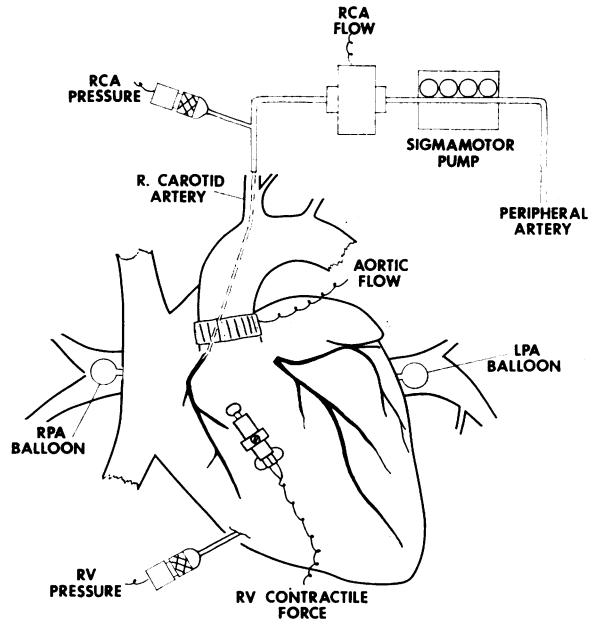

FIGURE 1 Schema for right coronary artery cannulation experiments. The cannula is perfused from a peripheral artery via an external circuit. Right coronary artery flow and perfusion pressure can thus be measured and controlled by the attached peristaltic pump.

The right ventricle was subjected to systolic stress as in group 1. In both groups of experiments, intravenous infusions of dextran were used to correct any degree of hypovolemia as judged by low right ventricular diastolic pressures, and thus to insure optimal ventricular performance.

RESULTS

Group 1. As illustrated by the representative experiment shown in Fig. 2, total occlusion of the right coronary artery caused a marked drop in right ventricular contractile force. Nevertheless, there was essentially no change in right or left ventricular pressures or aortic flow (Fig. 2). This effect was reproducible in the same animal as well as in the entire group of five animals. Occlusions lasting as long as 90 sec caused no change in heart rate. In contrast, with a moderate elevation of right ventricular systolic pressure (40 mm Hg), total occlusion of the right coronary artery resulted in a drop of systolic and rise in end-diastolic pressure in the right

[1] Walton-Brodie type, from John Warren, Department of Pharmacology, University of South Carolina, Charleston, S. C.

[2] Biotronex Lab, Inc., Silver Spring, Md.

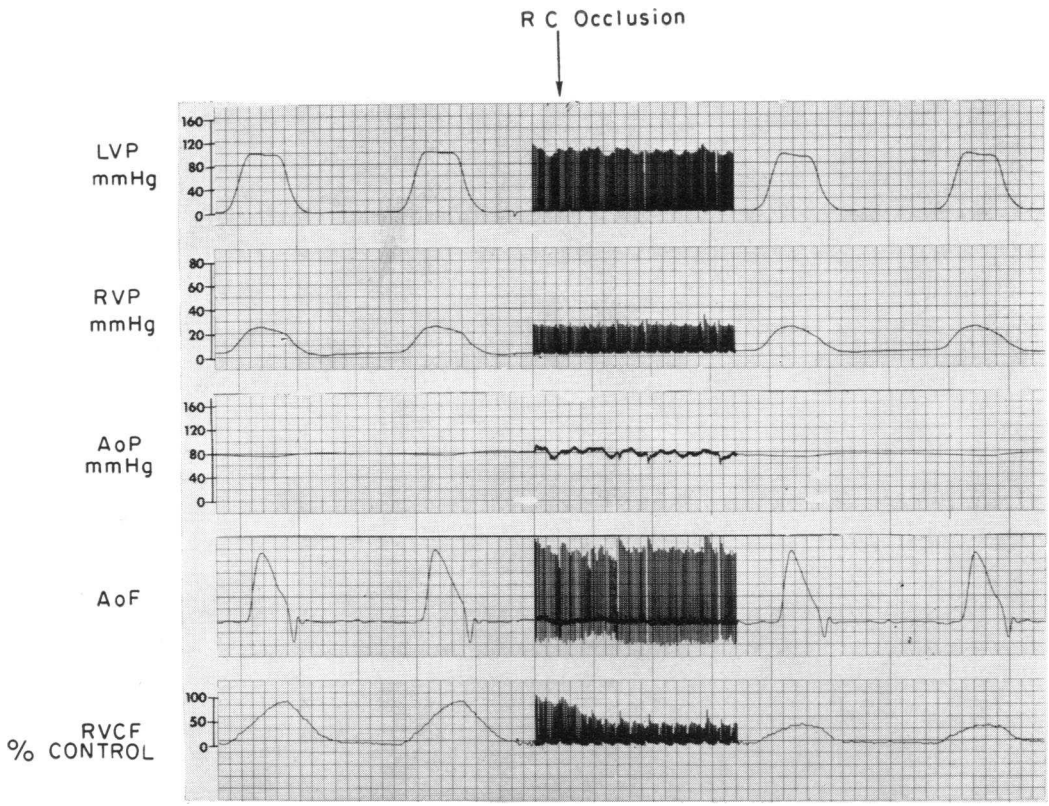

FIGURE 2 Effect of temporary right coronary (RC) artery occlusion on right and left ventricular hemodynamics and right ventricular contractile force (RVCF), under normal, resting conditions. LVP = left ventricular pressure; RVP = right ventricular pressure; AoP = mean aortic pressure; AoF = aortic flow. Although there was a marked drop in contractile force, there was essentially no change in right or left ventricular pressures or flow.

ventricle (Fig. 3). Delivery of flow to the left ventricle was decreased as evidenced by a drop in left ventricular end-diastolic pressure as well as aortic flow and pressure. Concomitant with these changes, there was visible dilatation of the right ventricle.

In Fig. 4, the results from the experiment illustrated in Fig. 3 are presented graphically, showing the entire range of systolic pressures from control to that just before total decompensation. In the presence of intact right coronary flow (solid lines), incremental increases in pulmonary arterial obstruction caused an increase in right ventricular systolic and end-diastolic pressures. Simultaneously, aortic flow and pressure and left ventricular end-diastolic pressure fell. The changes continued sequentially until right ventricular systolic pressure was between 60 and 80 mm Hg beyond which total right ventricular failure and systemic shock occurred. With the right coronary artery occluded (broken lines), total decompensation occurred at much lower right ventricular systolic pressures despite an absence of change at normal right ventricular pressures. These relationships are tabulated and presented with levels of statistical significance in Table I. It can be seen that, with the right ventricle moderately overloaded (right ventricular systolic pressures 26–40 mm Hg), occlusion of the right coronary artery resulted in statistically significant lowering of only aortic flow and peak right ventricular pressure, while above this level, significant changes occurred in all parameters.

Group 2. In the resting state, normal right coronary arterial flow ranged from 12 to 17 ml/min in these five dogs. Perfusion of the right coronary artery at higher than normal pressures, without a high systolic load imposed on the right ventricle, caused no change in peak right ventricular pressure, end-diastolic pressure, left ventricular pressure, or aortic flow.

The initial protocol was the same as for group 1. Right ventricular systolic pressure was gradually increased in increments of 10 ±2 mm Hg, and similar alterations in right and left ventricular pressures and aortic pressure and flow resulted, indicating no alteration in performance due to the cannulation itself. In addition,

right coronary arterial flow rose concomitantly with increasing right ventricular systolic stress. That these intrinsic levels of coronary flow were the maximum obtainable by autoregulation was suggested by the fact that at higher loads there was no further response to large doses (40 μg) of intracoronary nitroglycerine. Critical levels of severe pulmonary obstruction were thus attained where a slight further increase led to progressive right ventricular decompensation.

In Fig. 5, the continuous recording from a representative experiment is shown. The beginning of the record represents a stable state under maximal systolic stress and critical levels of pulmonary obstruction in this preparation. Here, right coronary flow has reached maximal levels at 38 ml/min, right ventricular systolic pressure is 77 mm Hg, and mean aortic and right coronary perfusion pressures are slightly lowered presumably due to reduced delivery of blood to the left heart. An attempt to increase pulmonary obstruction slightly at this point results in the onset of total right ventricular decompensation and severe systemic hypotension with a consequent fall in right coronary artery flow and perfusion pressures to near zero levels. At this time (arrow, Fig. 5), without any adjustment in pulmonary artery obstruction, the coronary perfusion pump is turned on, which restores coronary flow to a rate such that right coronary perfusion pressure and flow are higher than the level just before decompensation. With this intervention, right ventricular pressures return to maximal stress levels, followed in 15–20 sec by a reversal of right ventricular decompensation and a restoration of systemic blood pressure. A transient episode of hypertension in both ventricles occurs subsequently, followed by a stable state with right ventricular and aortic pressures at the levels just before decompensation.

The onset of right ventricular failure and consequent cardiac decompensation reached by incremental pulmonary obstruction was a definite end point, easily recognized once it had begun. On the other hand, it was never possible to predict in a given experiment the exact critical point of stress beyond which decompensation would occur and thus with certainty to prove whether prophylactic coronary perfusion before failure could be preventative. Indeed, the experimental design was the converse: that is, to reach the threshold of peak systolic stress and right ventricular failure in small increments (10 mm Hg right ventricular systolic pressure) and, once decompensation had begun, attempt to reverse it

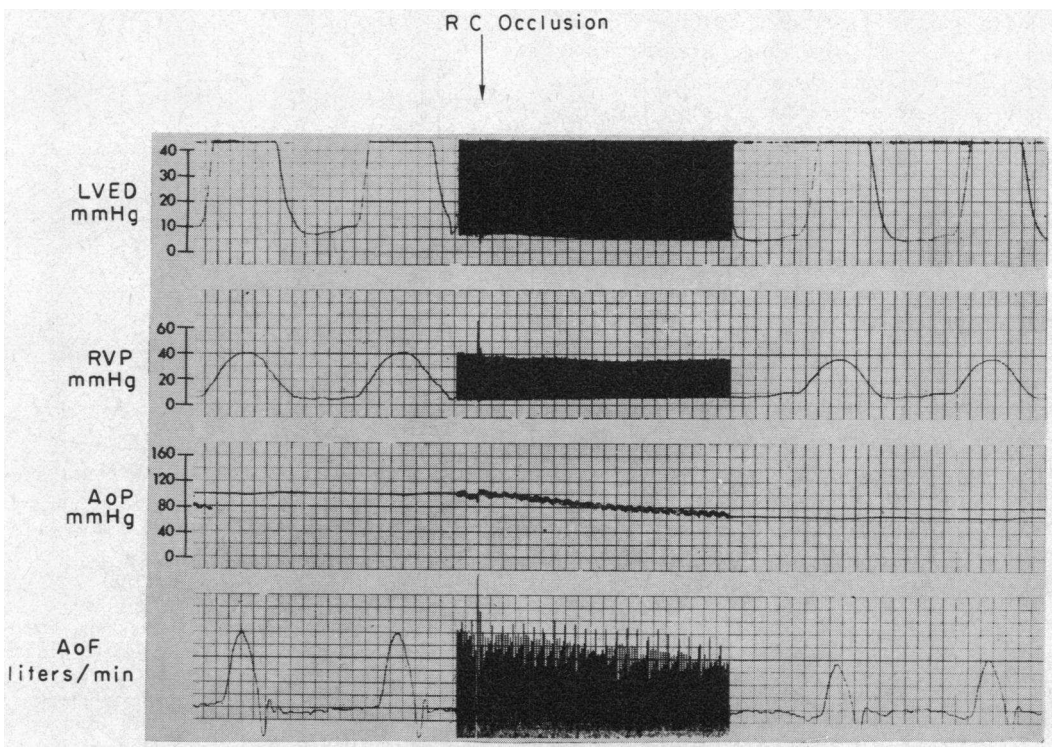

FIGURE 3 Effect of temporary right coronary (RC) artery occlusion on right and left ventricular hemodynamics under conditions of a moderate added systolic load. LVED = left ventricular end-diastolic pressure. Other abbreviations as in Fig. 2. In contrast with the control resting state, right coronary occlusion results in significant alterations in both right and left ventricular hemodynamics.

TABLE I

Hemodynamic Responses with Increasing Levels of Right Ventricular Systolic Stress, Induced by Incremental Pulmonary Artery Obstruction, before (A) and after (B) Right Coronary Artery Occlusion. Results Are Expressed as Mean Values ±SEM.

Level of right ventricular systolic stress	n	RVSP		RVEDP		AoF		AoP		LVEDP	
		A	B	A	B	A	B	A	B	A	B
		mm Hg				*liters/min*		*mm Hg*			
15–25 mm Hg (control)	5	20 ±1	20 ±2	3 ±0.5	3 ±0.5	1.9 ±0.2	1.8 ±0.2	104 ±6	103 ±7	6 ±1	6 ±1
26–40 mm Hg	5	35 ±2	31 ±2*	3 ±0.5	4 ±0.5	1.9 ±0.1	1.4 ±0.2‡	102 ±6	88 ±11	6 ±1	5 ±1
41–54 mm Hg	5	45 ±1	40 ±1‡	4 ±0.5	6 ±0.5*	1.6 ±0.1	1.1 ±0.1*	106 ±6	84 ±12‡	5 ±1	4 ±1‡
55–70 mm Hg	5	60 ±3	49 ±3*	8 ±1	10 ±2*	1.2 ±0.1	0.5 ±0.1*	92 ±10	52 ±9*	4 ±1	2 ±0.5‡

Abbreviations: RVSP, right ventricular systolic pressure; RVEDP, right ventricular end-diastolic pressure; AoF, aortic flow; AoP, mean aortic pressure; LVEDP, left ventricular end-diastolic pressure.

* $P < 0.01$ paired t test, comparing A with B.

‡ $P < 0.05$ paired t test (9), comparing A with B.

simply by perfusing the right coronary artery at pressures equal to or higher than those under control conditions. In this regard it was never possible to reverse failure by restoring perfusion pressure to levels just before decompensation. It was always necessary to perfuse at pressures substantially above control, ranging from 108 to 160 mm Hg. Furthermore, although right ventricular performance was extended by this maneuver —was, indeed, sufficient to bring about reversal of right ventricular failure—the additional range of performance was not great. The additional increment in right ventricular systolic pressure ranged from 6.8 to 11.8 mm Hg, and in aortic flow from 0.2 to 0.4 liter/min.

Phasic characteristics of right coronary flow were studied in the presence of normal and elevated right ventricular systolic pressures. Fig. 6 shows a recording of right coronary inflow and right ventricular pressure at control levels and during severe pulmonary obstruction. The portion of flow during systole is indicated by shading during two consecutive beats, allowing for appropriate time delays due to the external perfusing circuit. Elevation of right ventricular pressure from 28 to 68 mm Hg caused an increase in right coronary arterial flow from 13 to 21 ml/min. Systolic flow under control conditions is approximately equal to diastolic flow. At high right ventricular pressures, right coronary arterial flow increases primarily in diastole, indicating that the coronary vasodilation is ineffective in augmenting systolic flow.

DISCUSSION

These studies have demonstrated that right ventricular performance is not impaired after total occlusion of the right coronary artery as long as pulmonary arterial pressure is not increased.

In the presence of high levels of pulmonary arterial resistance and an occluded right coronary artery, failure of the right ventricle ensues with a reduction of venous return to the left ventricle, and a fall in cardiac output and arterial pressure. Furthermore, a return of right ventricular function and ultimate reversal of cardiac decompensation were brought about by mechanical restoration of right coronary flow to levels above those before decompensation. Thus under these conditions of

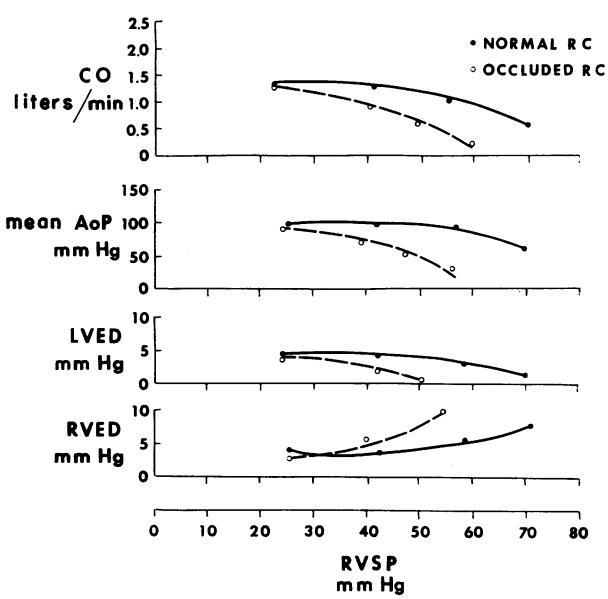

FIGURE 4 Composite graph of data taken from a representative experiment of group 1, relating cardiac output (CO), mean aortic pressure (mean AoP), left ventricular end-diastolic pressure (LVED), and right ventricular end-diastolic pressure (RVED), to right ventricular systolic pressure (RVSP), over the entire range of systolic stress, from control to the state just before total decompensation. Solid lines join the points in which the right coronary flow is intact; dotted lines join those points at identical degrees of pulmonary artery obstruction, but with the right coronary artery temporarily occluded.

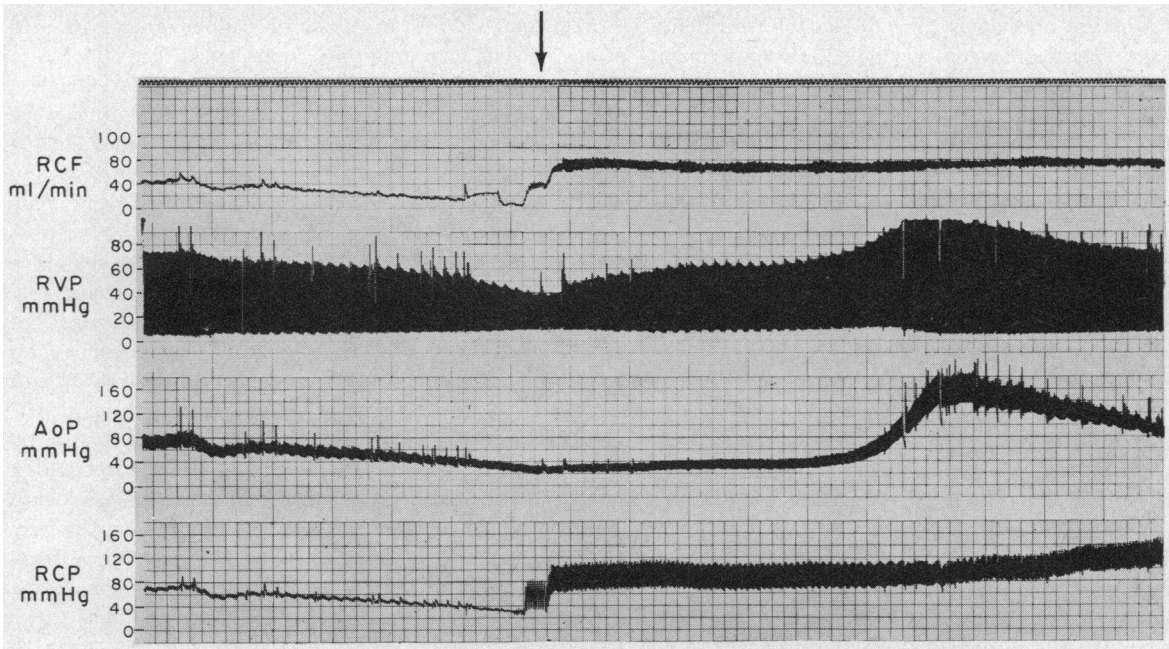

FIGURE 5 Continuous recording taken from a representative experiment showing responses to supercritical pulmonary obstruction which results in total right ventricular decompensation and severe systemic hypotension with consequent fall in right coronary artery flow and perfusion pressure to near zero levels. At this moment (arrow), without any adjustment in pulmonary artery obstruction, the coronary perfusion pump is turned on which restores right coronary flow. Subsequently right ventricular pressures and ultimately total cardiac decompensation are reversed.

extreme right ventricular hypertension, it appears that the maximal coronary vasodilating capability is not quite equal to the peak function capacity of the myocardium so that blood flow to the ventricular free wall is the primary factor which first limits response to further systolic stress.

These data help to elucidate the sequence of events leading to right heart failure after acute pulmonary obstruction, termed "muscle fatigue" by Fineberg and Wiggers (1). The point at which right ventricular decompensation occurs may be altered by several factors which together place higher demands on the free wall on the myocardium and augment oxygen demand and a need for higher blood flow. These include higher systolic and end-diastolic pressures, and right ventricular enlargement with greater tension development for a given pressure.

Concomitant with these increased demands for oxygen, other factors place limitations on right coronary arterial blood supply. First, systemic hypotension occurs due to a decreased return of blood to the left ventricle and decreased cardiac output. At critical levels of diminished coronary perfusion pressure, beyond the point of maximum coronary autoregulatory capabilities, coronary flow diminishes rapidly as systemic arterial pressure falls. This circumstance is in contrast with comparable pathologic events involving the left ventricle, such as acute malignant, systemic hypertension in which the increased demands for coronary flow are accompanied by increased coronary perfusion pressures. A second factor which may lead to right heart failure under these conditions is impingement of the myocardium upon coronary flow during systole. The phasic coronary flow recordings observed in these experiments showed that in the right coronary artery during the normally low resting intraventricular pressures, systolic flow was approximately equal to diastolic flow. This is in contrast with the phasic distribution of coronary flow in the left coronary artery where flow is primarily diastolic. These findings are similar to the right coronary inflow studies of Gregg (10), who also showed in other studies (11) that systolic contraction of the myocardium impedes coronary flow, particularly in the left ventricle where intraventricular systolic pressures are high. Moreover, distribution of flow to different layers of the myocardium may be altered by acute changes in intraventricular pressures (12–14). Whether these same phenomena may contribute to precipitation of failure of the right ventricle during acute hypertensive states, such as massive pulmonary embolism, remains to be proven by other methods. However, the observed re-

versal of the normal systolic to diastolic ratio in right coronary phasic flow observed in these experiments indicates that this may be an important factor leading to an impairment of adequate blood supply during acute right ventricular hypertension. This seems particularly important when increased right ventricular systolic pressure is not accompanied by increased perfusion pressures in the right coronary artery.

The present investigation lends support to the view that a normally contractile right ventricular free wall is not necessary for maintenance of cardiac performance at normal pulmonary artery pressures. Starn, Jeffers, and Meade (2) and others (3, 4) have already demonstrated that little change occurs in venous or systemic pressure after extensive electrochemical destruction of the free wall of the right ventricle. There are several possible explanations for this interesting phenomenon. At low resting pressures, septal contraction as well as contraction at the extreme periphery of the free wall, both of which are supplied by branches of the left coronary in the canine heart (15), may be sufficient to maintain adequate contractile responses. Venous return per se may be an important factor as first suggested by the experimental studies of Rodbard and Wagner (16). Indeed, the vena cavae may be connected directly to the pulmonary artery to bypass totally the right ventricle. They showed that venous pressures of 12 cm saline were adequate to maintain flow through the pulmonary artery without the contraction of the right ventricle. In these present studies, however, with normal right ventricles and relatively low end-diastolic pressures after right coronary artery occlusion, the factor of venous return alone could not adequately explain maintenance of right heart function. There are abundant anastomoses between some branches of the right and left coronary arteries (14). These, as well as a direct absorption of nutrients from right ventricular blood, might help maintain some element of free wall function at a normal afterload. However, during conditions of right ventricular systolic overload, the data demonstrate that a contractile right ventricular free wall becomes increasingly important in the maintenance of cardiovascular performance.

In relating these experimental data to any comparable clinical setting, it should be pointed out that the canine right coronary artery differs considerably from that of the human, supplying only approximately 15% of the

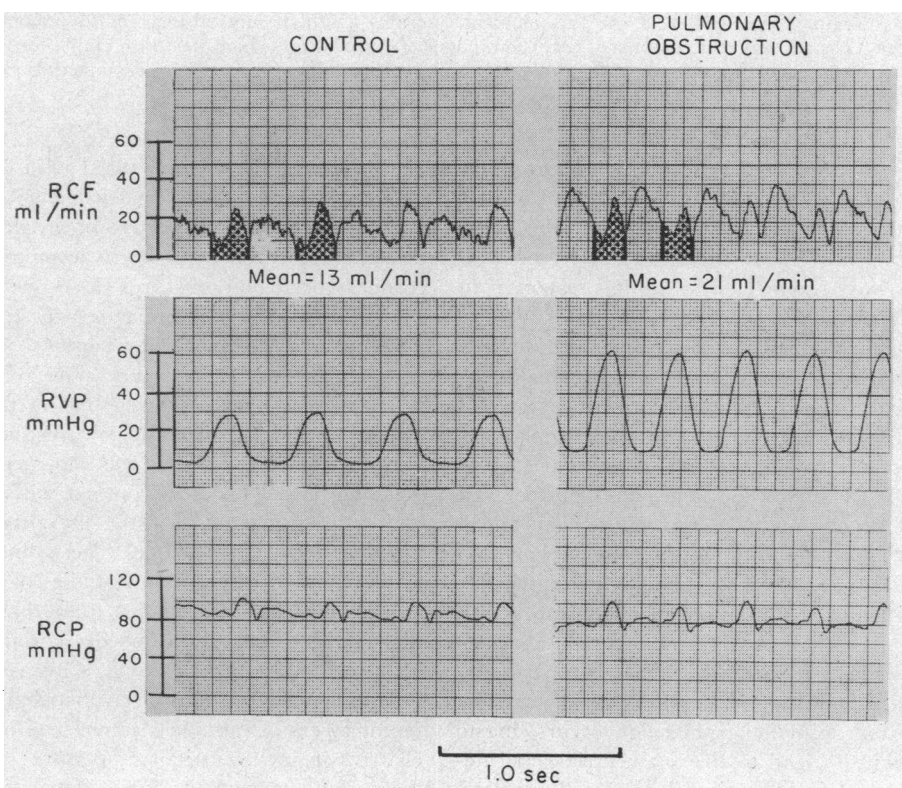

FIGURE 6 Effects of acute elevation of right ventricular pressure (RVP) on systolic and diastolic right coronary in flow (RCF). RCP = right coronary perfusion pressure. The systolic intervals for two consecutive beats are shaded in. The augmentation in right coronary flow occurs primarily during diastole.

total myocardial flow, in contrast with approximately half the total flow in man (17). The fact remains that in both species the right coronary artery is the primary source of blood supply to the right ventricular free wall, and it is this portion of the myocardium which is under greatest pressure stress during acute pulmonary obstruction. The present results may therefore have important clinical implications when considering the management of massive pulmonary embolism in its early stages with acute right heart failure and systemic shock. Restoration of an adequate perfusion pressure for the right coronary artery appears to be a prime prerequisite in increasing contractility of the right ventricle so as to restore cardiac output. While direct manipulation of right coronary artery flow may not be feasible in the clinical setting, the immediate institution of measures to increase right coronary artery perfusion pressure as suggested by the studies of Salisbury (6) and Berman, Spotnitz, and Epstein (7) may be specifically helpful. Thus systemic pressor agents such as norepinephrine or methoxamine appear to be the measure of first choice. In contrast, the use of a beta adrenergic agonist such as isoproterenol, which reduces peripheral arterial resistance, may be detrimental by further decreasing right coronary perfusion pressure and at the same time increasing right ventricular oxygen demands.

ACKNOWLEDGMENTS

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