Coronary Artery Pressure and Strength of Right Ventricular Contraction

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Experiments show that the right ventricle is weakened to the point of irreversible failure by decreased coronary artery pressure and also demonstrate that augmented coronary artery pressure can cause a remarkable increment of the right ventricular strength. Both ventricles, even when in otherwise irreversible decompensation, can be restored to full functional competence by various maneuvers which augment coronary blood flow. The studies reported appear to have important clinical applications in the treatment of circulatory disturbances of the right ventricle.

A number of previous reports have strongly suggested but not proven that contractile power of the right ventricle can be improved by augmenting its blood supply. An endeavor was made in the presently reported investigation to substantiate this concept with more direct evidence and to determine whether a failing right ventricle would respond favorably to increased coronary flow.

Methods

Mongrel dogs were anesthetized with intravenous injections of pentobarbital sodium, 25 mg. per Kg. Plastic cannulas were inserted into the femoral artery and via the jugular vein into the right ventricle. The anterior descending ramus of the left coronary artery was cannulated in some experiments; in others the metal cannula, described by Agress and co-workers was introduced through the right carotid artery into the ascending aorta. The pressure sensing cannulae were connected with Statham strain gages no. 23A; electric impulses were amplified on Edin amplifier no. SI 10 and registered on Edin direct writing multi-channel oscillograph no. S066.

A hinged ellipsoid polyethylene frame was installed where the pulmonary artery and the aorta were encircled in separate compartments and where distensible bags allowed the graded compression of each vessel individually (fig. 2). Pressures in these "hydraulic clamps" were varied by injecting water into the bags by means of a screw driven, mounted syringe. The bag pressures were observed on manometers. In some experiments, a distensible rubber bag attached to a syringe by a tube was inserted into the left ventricle through a small stab wound at the apex. In other experiments, the animals were given 1 mg. heparin per pound and one of their carotid arteries, or the left subclavian artery, connected by means of one-quarter inch I.D. polyvinyl chloride tubing with a siliconed glass reservoir bottle. The reservoir bottle was suspended at variable elevations above the heart of the animal; it contained 400 cc. heparinized dog blood.

The compression of the pulmonary artery was changed by discrete stepwise increments of the liquid volume in the clamp. In some experiments, the clamp was completely emptied of fluid between changes of the pulmonary artery compression.

Results

Effects of Graded Compression of the Pulmonary Artery and of Its Subsequent Release. Figure 3 shows changes which occur when the pulmonary artery is compressed in the open chest dog and when the femoral or coronary artery pressures are not influenced by any therapeutic maneuvers. It is apparent that increase of the pulmonary compression up to 60 mm. Hg causes a corresponding rise of the right ventricular systolic pressure without changing the end-diastolic pressure. When the pulmonary artery compression is raised above 60 mm. Hg decompensation of the right ventricle occurs, as is evident by its rising end-diastolic pressure, its falling systolic...
pressure and the drastic fall of the peripheral arterial pressure. When the pulmonary artery compression reaches values above 80 to 100 mm Hg, complete incompetence of the heart supervenes. Complete incompetence is characterized by a high end-diastolic pressure of the right ventricle, a low right ventricular pulse pressure and a mean femoral artery pressure of less than 30 mm Hg. Once the heart has reached the condition of complete incompetence, release of the pulmonary artery clamp will not resuscitate it and the incompetent condition persists until ventricular fibrillation or cardiac arrest supervenes. Complete incompetence is not only defined by pressure measurements, but is also noticeable because of the flabby, cyanotic and dilated appearance of the heart, weak, inefficient contractions and the shiny, tense epicardium.

When the pulmonary artery compression is released before the state of complete incompetence is reached, the heart slowly recovers; arterial and right ventricular pressures gradually return to normal during a period of 3 to 5 minutes. When the pulmonary artery is compressed repeatedly in the same dog a normal peripheral artery pressure is observed preceding the first compression, but, after the first compression which produces incompetence of the right ventricle the peripheral arterial pressure becomes markedly elevated. The peripheral arterial pressure returns to the level obtaining before the onset of right ventricular decompensation within 10 to 15 minutes following release of the pulmonary artery compression.

**Resuscitation of Incompetent Heart by Elevation of Aortic Pressure.** Figure 4 shows the improvement of right and left ventricular function as a result of increasing aortic pressure...
in the completely incompetent heart. It is evident that complete incompetence of both ventricles was reversed by clamping the ascending aorta for a period of 33 seconds. During the period of aortic clamping, there was no femoral artery pulse pressure, but, as the coronary artery pressure increased, the systolic pressure of the right ventricle rose and its end-diastolic pressure fell. The pressure measurements indicate return of both ventricles to full competence after release of the aortic constriction. Figure 4 is representative of 10 similar experiments in which the ascending aorta was constricted with a hydraulic clamp and six experiments in which aortic blood flow was directed into the coronary artery by the bag distended in the ascending aorta. In six other experiments, the completely incompetent heart was resuscitated by the sudden opening of an arterial reservoir which was at a level of at least 150 cm. above the heart. In yet another series of 10 experiments, the completely incompetent heart was resuscitated when phenylephrine hydrochloride, 0.1 mg./Kg. (Neoersynephrine), was injected into the right ventricle through the pressure registration cannula. For purposes of resuscitation with this pressor agent, it was usually necessary to massage the heart with 5 to 10 vigorous manual compressions which apparently served to transfer the drug from the right ventricle to its sites of action. It was thus found that complete incompetence of the heart which was caused by compression of the pulmonary artery could be reversed by four different therapeutic maneuvers which increased coronary artery pressure.

Avoidance or Delay of Right Ventricular Incompetence by Elevation of Arterial Pressure. Figure 3 describes pressures in the right heart and in the peripheral arteries as a function of the pressure in the pulmonary artery clamp. It is noted that in this control observation, which makes no attempt to vary coronary artery pressure or coronary flow by experimental maneuvers, the maximum systolic pressure of the right ventricle is observed with a pulmonary artery clamp pressure of 70 mm. Hg or so and that complete incompetence of the heart supervened with a clamp pressure of about 80 mm. Hg.

Figure 5 describes the relationship between mean femoral artery pressure and the pulmonary artery compression necessary to cause complete incompetence of the heart when the pulmonary artery compression is applied suddenly, starting from zero. The systemic arterial pressure in this experiment was varied by means of arterial reservoirs and by pressor agents. It is evident that the production of complete incompetence requires a much higher level of pulmonary artery compression when the systemic pressure is elevated. Not shown in this figure are results of experiments in which the distensible bag in the ascending aorta was filled with liquid to increase the pressure in the root of the aorta to a mean of 200 mm. Hg and yet maintain an average femoral artery pressure of 60 mm. Hg. With the aid of this distensible bag in the ascending aorta, such pressure differentials could be maintained for 15 minutes or more, during which the right ventricle did not show signs of incompetence in spite of 350 mm. Hg pulmonary artery compression. With this maxi-
nal pulmonary artery compression, decrease of the volume of the intraaortic bag caused prompt fall of the aortic root pressure and incompetence of the right ventricle. Redistension of the bag would then increase aortic root pressure and restore full right ventricular competence provided the preceding incompetence was not complete.

Figure 6 shows the relationship between mean femoral artery pressure and the maximal systolic pressure which is developed by the right ventricle when the pulmonary artery is compressed with gradually increasing pressure. It is evident that a direct relationship exists between the contractile strength of the right ventricle and the mean femoral artery pressure. In this experiment, the femoral artery pressure was varied by reservoirs and by pressor agents. Not included in figure 6 are experiments in which the pressure in the root of the aorta was varied with the hydraulic clamp shown in figure 2 or with the distensible bag in the ascending aorta. In these experiments, right ventricular systolic pressures up to 180 mm. Hg were observed when the aortic root pressure reached 250 mm. Hg.

Exclusion of Mechanisms Other Than Coronary Flow. The connection of an elevated pressor reservoir with the arterial system causes blood to flow into the dog whenever the pressure in the arterial tree falls below the elevation of the reservoir. In order to test whether the augmented contractile strength of the right ventricle was caused by the expansion of the blood volume or by the increased arterial pressure, pulmonary artery compression was applied to animals which received intravenous injections of blood at a rate corresponding to the outflow from the arterial reservoir. It was found in six experiments that intravenous injection of blood at a rate of 50 to 100 cc. per minute did not resuscitate hearts in complete incompetence, did not appear to have an appreciable effect in delaying or avoiding cardiac failure which occurred during compression of the pulmonary artery and did not increase the maximum right ventricular systolic pressure.

In order to reproduce the blood loss from the dog to the lowered arterial reservoir, blood was withdrawn from the venous system and the peripheral arterial pressure kept normal.
with pressor agents. It was noted in eight experiments that the competence of the right ventricle was diminished whenever the blood volume decrease amounted to 2 per cent of the weight or more. After such an acute loss of blood, hearts with complete incompetence could be resuscitated only when prolongedmassage was added to the other therapeutic maneuvers and the contractile strength of the right ventricle was markedly decreased. It must be concluded that blood loss has an adverse influence on the contractile strength of the right ventricle, but that infusion of blood in the normovolemic dog does not increase right ventricular contraction.

In order to test the hypothesis that increased initial stretch and tension of the left ventricle such as occurs as a result of increased pressure in the aortic root may influence the contractile strength of the right ventricle, a distensible bag was inserted into the left ventricle. When this bag was injected with 10 to 15 cc. water, the residual volume of the left ventricle was increased by that amount and the left ventricle became noticeably dilated. In this experiment the mean arterial pressure and the right ventricular pressure was not changed as a result of distension of the left ventricular bag per se. It was found in six experiments that distension of a bag in the left ventricle did not noticeably influence the maximum systolic strength of the right ventricle, the degree of pulmonary artery compression necessary to produce complete incompetence of the heart or the resuscitation of the completely incompetent heart.

Six experiments were performed in which the pulmonary artery was compressed and the aortic pressure was varied in dogs which had received hexamethonium, 4 mg./Kg. intravenously, and also at the same time total spinal anesthesia. In this "chemically denervated" animal, the bradycardia and the "overshooting" of the arterial pressure did not occur after release of the pulmonary artery compression. As far as the influence of coronary artery pressure on right ventricular strength is concerned, there was otherwise no difference between those dogs which had received "chemical denervation" and those which had not.

The persistent effect of coronary artery pressure in the "chemically denervated" dog would seem to rule out a reflex mechanism.

**DISCUSSION**

Previous investigators have shown that a reduction of the blood supply in the heart muscle and the ability of the myocardium to perform work are directly related. The present study not only confirms preceding work, but also shows that cardiac competence can be increased when more blood is made to flow through the coronary circuits. When other factors are equal, the coronary flow has been found to be a function of coronary artery pressure. In the experiments described, the coronary artery pressure was raised by pressor agents, by partial occlusion of the ascending aorta with a balloon, by external compression of the aorta and by an arterial blood reservoir. An increment of the pressure in the root of the aorta automatically increases the resistance against which the left ventricle empties itself and increases left ventricular work; therefore the left ventricle is not necessarily helped by an increase of the coronary artery pressure. However, unless the arterial pressure is high enough to cause failure of the left ventricle, the resistance in the pulmonary circuits is not known to be influenced by changes in the peripheral arterial pressure and therefore the resistance in the systemic arterial circuit will usually influence the level of metabolic support of the right ventricle without simultaneously changing its work load.

The opportunity exists therefore of increasing the blood supply to both the right and the left heart, while increasing the work load of the left ventricle only. Pathologic conditions which are characterized by insufficiency of the right ventricle, but in which the left ventricle has a considerable reserve should therefore benefit by therapeutic maneuvers which raise the coronary artery pressure. The influence of pressor agents on incompetence of the right ventricle has apparently not been studied except for one report in which acute cor pulmonale caused by a pulmonary embolism was treated successfully with arterenol.

The data presented here indicate that when
the function of the left ventricle is not impaired, the limits of competence of the right ventricle can be greatly extended by an increase in coronary artery pressure.

SUMMARY AND CONCLUSIONS

In anesthetized open chest dogs, the limits of compensation of the right ventricle were tested by repeated compression of the pulmonary artery with a distensible hydraulic clamp. Coronary artery pressures were changed by means of pressor agents, arterial blood reservoirs, compression of the aorta with a hydraulic clamp and by partial occlusion of the ascending aorta with a distensible bag in its lumen. It was found that the strength of the right ventricle is directly related to aortic root pressure and consequently to coronary pressure; with low coronary artery pressure (irrespective of how it is produced), the right ventricle is incapable of generating much force. When the coronary pressure is normal, the right ventricle is able to overcome pulmonary artery clamp pressures of 80 to 120 mm. Hg. With very high coronary artery pressures produced in different ways, failure of the right ventricle did not occur in our experimental preparation even with maximal pulmonary artery compression. The maximum observed right ventricular systolic pressure rose to 180 mm. Hg. Furthermore, otherwise irreversible acute heart failure could be reversed by an increase of aortic root pressure.

REFERENCES

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