Behavior of the Right Ventricle Following Acute Constriction of the Pulmonary Artery

By A. C. Taquini, M.D., J. D. Ferroso, M.D., and P. Aramendia, M.D.

Numerous investigations have been undertaken to elucidate the factors concerned in the production of acute cor pulmonale secondary to embolism. Acute failure of the right ventricle has been studied from the point of view of the factors responsible for the increase in pulmonary resistance, direct mechanical obstruction and/or reflex vasoconstriction of the pulmonary vascular bed. On the other hand, the capacity of the right ventricle to overcome the increased pulmonary resistance has been studied only sporadically. In 1936, Fineberg and Wiggers reported that the dog's right ventricle is able to sustain progressive reduction of the lumen of the pulmonary artery, first by a simple increase in the initial tension of its fibers with a concomitant increase in the end diastolic filling pressure. Further constriction diminishes the passage of blood to the left ventricle and the heart rapidly fails.

Since the experiments of Fineberg and Wiggers are based exclusively on pressure changes, we have reinvestigated the effects of acute constriction of the pulmonary artery on cardiac output as well as intracardiac pressures.

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Fifteen dogs under thiopental anesthesia (25 mg./Kg. intravenously) were used. After tracheal intubation and commencement of artificial respiration, the thorax was opened by means of a wide incision in the fourth left intercostal space. The pericardium was opened and either a plastic or silk thread ligature was passed around the pulmonary trunk. A catheter was introduced into the right jugular vein and threaded into the right ventricle, and a second catheter was inserted into the left ventricle via the right carotid. Pressures in both ventricles were recorded using Statham transducers. Oxygen consumption was estimated in the expired air collected for a period of 3 minutes in Douglas bags by means of an apparatus consisting of valves and a double action, suction and expulsion, respiration pump. Blood samples from the femoral and pulmonary arteries were analyzed in a Van Slyke apparatus and the cardiac output calculated by the direct Fick method.

In all dogs, determinations were made with the pulmonary artery free and with the ligature tightened to the limit at which the right ventricle could compensate for the resistance by maintaining the pressures in the right and left ventricles essentially unchanged. In 7 of them, a third determination was made during the period when the left ventricular pressure showed a more or less sustained fall. A fourth determination was obtained in 2 dogs a few minutes after the development of failure. In 3 dogs, determinations were made after the administration of strophanthus (0.03 mg./Kg.).

Results

Table 1 shows the results obtained in 15 experiments. Prior to ligation of the pulmonary artery, the right ventricular pressure was within normal limits in most of the dogs. In 4, the systolic pressure was slightly high (40 mm.Hg). Diastolic pressure was normal in all cases. The corresponding cardiac output values for all dogs ranged between 0.79 and 2.76 L./min. Oxygen consumption varied between 41 and 176 ml./min., and arteriovenous oxygen difference between 5.20 and 9.38.

Partial constriction of the pulmonary artery caused an immediate rise in right ventricular systolic pressure. The limits of pressure attained by the right ventricle, without the appearance of left ventricular pressure changes or signs of right ventricular failure (alternation), lay between 50 and 90 mm.Hg, mean 62.3± 3.72 mm.Hg. The corresponding diastolic pressure remained unchanged in 8 of the dogs, rose by 2 mm. Hg in 3, by 3 mm. in 1 and by 1 mm. in 1. In 1 dog, there was a fall of 2 mm. Hg in the end diastolic filling pressure.

Arteriovenous oxygen difference rose in 4 dogs, fell in 6 and remained virtually un-
Summary of Results of Constriction of Pulmonary Artery

The results confirm those of earlier workers1-3 in the sense that a constriction of approximately two-thirds of the pulmonary trunk is required to cause significant changes in right ventricular pressure at rest. They also confirm the findings of Fineberg and Wiggers1 who noted a limited ability of the right ventricle to withstand increases of pulmonary resistance. Dog No. 4 responded to ligation of the pulmonary artery by an increase in the right ventricular systolic pressure from 20 to 60 mm. Hg without variation in its diastolic pressure, nor in the pressures of the left ventricle (fig. 1). Simultaneously there occurred a moderate increase in minute volume. Minimal tightening of the ligature produced first a further rise in right ventricular systolic pressure, and then a rise in the diastolic. At the same time, arrhythmia appeared and the cardiac output began to fall from 1.08 to .97 L./min. Several minutes later, while the ligature remained unchanged, the cardiac output was reduced to 0.66, right ventricular pres-

Table 1

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*G.: Control without ligature. L.: With ligature. w. dig.: With digitalis.
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PULMONARY ARTERIAL CONSTRICTION

VI. B.O.V.D.

Figure 1

Upper pair: The upper tracings represent pressure curves from the left ventricle and the lower ones those of the right ventricle. On the left can be seen the record obtained before constriction of the pulmonary artery. The middle tracings were obtained after a moderate degree of restriction. A slight rise of right ventricular systolic pressure can be observed, with no significant change in that of the left ventricle. At the same time, there is a moderate increase in cardiac output. The tracings at the right were taken after a further constriction of the pulmonary artery, and show only minor variations from the previous ones, while the cardiac output has not shown significant variation.

Lower pair: As the degree of constriction is increased, the systolic pressure in the right ventricle rises, with a concomitant fall in that of the left ventricle. During this period the cardiac output drops considerably. Time markings at the lower edge of the chart represents 1-second interval.

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Figure 2

These tracings were obtained at a paper speed of 25 mm./sec. The upper curves represent left ventricular pressures and the lower ones right ventricular pressures, registered after constriction of the pulmonary artery. Systolic pressure of the right ventricle rises with no change in the diastolic pressure, even in the stage at which the systolic pressure of the left ventricle begins to fall. Beyond this level, an increase in resistance produces a second phase of adaptation: a greater increase in systolic pressure, a rise in diastolic pressure of the right ventricle with preservation of the cardiac output. The second phase of adaptation was observed to be transitory in 5 out of 15 dogs. In most experiments, the increase of resistance beyond the first phase was followed by a fall in output and a secondary fall in left ventricular pressure, and later by a fall in right ventricular pressure and progressive cardiac failure (fig. 2).

The above experiments demonstrate the poor ability of the normal right ventricle to compensate for acute increases in pulmonary vascular resistance. This fact contrasts with the behavior of the right ventricle undergoing chronic increases in resistance, in which circumstances it can endure pressures equal or superior to those of the left ventricle. This poor ability of the right ventricle to respond to acute increases in resistance may be explained by its thin muscular coat.

The fact that the right ventricle can increase its systolic capacity without alteration of its output, appears to be about 60 mm. Hg. Up to this level, the first phase of adaptation, the increase of systolic force occurs without significant or apparent changes in diastolic pressure of volume of the right ventricle. Beyond this level, an increase in resistance produces a second phase of adaptation: a greater increase in systolic pressure, a rise in diastolic pressure of the right ventricle with preservation of the cardiac output. The second phase of adaptation was observed to be transitory in 5 out of 15 dogs. In most experiments, the increase of resistance beyond the first phase was followed by a fall in output and a secondary fall in left ventricular pressure, and later by a fall in right ventricular pressure and progressive cardiac failure (fig. 2).

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Constriction of the pulmonary artery is followed by a simultaneous rise in right ventricular systolic pressure and fall in left ventricular pressure. Diastolic pressure variations are not important. Time markings represent 1-second intervals.

Figure 3

Reduction in coronary flow may play a part in the parallel increase in end diastolic pressure in both ventricles during failure, but this requires additional investigation.

Summary

In 15 anesthetized dogs, constriction of the lumen of the pulmonary artery by less than 50 per cent produced no significant change in intraventricular pressure or cardiac output. Additional constriction caused a progressive increase of the right ventricular pressure; up to a pressure of 62.3±3./min. Hg, there was no change in cardiac output. With higher right ventricular systolic pressure there was a reduction in cardiac output, gradual decrease in systolic pressure but increase in diastolic pressure of both ventricles, and ultimately dilatation of the heart and ventricular arrest or fibrillation.

Interlingua translation will be found on page 346.

References

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