The hemodynamic response of the right ventricle following acute and chronic partial obstruction of the main pulmonary artery in dogs

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Summary: In eight adult dogs the main pulmonary artery was constricted to elevate the right ventricular peak systolic pressure to 50% of the peak aortic pressure at rest. The response of the right ventricle was assessed immediately, at 30 minutes and at six months. The right ventricle responded to acute systolic loading by complete compensation. After 30 minutes there was a reduction in the right ventricular outflow tract resistance. The cardiac output, heart rate and aortic pressure were maintained. The right ventricular systolic ejection period, end-diastolic pressure, peak pressure time, mean systolic pressure, right ventricular–main pulmonary artery mean systolic gradient, right ventricular work index, systolic work and outflow tract resistance were all increased.

The right ventricle in the dog was shown to have an immediate capacity to compensate for systolic loading and retains this capacity for long periods of time. The ability to increase work is accomplished by adaptations in right ventricular physiology which increase right ventricular mean systolic pressures and prolong the right ventricular ejection period.

The response of the right ventricle to acute increase in pressure load (sudden partial obstruction of the main pulmonary artery) may be summarized as one of three responses dependent upon the characteristics of the ventricle and the severity of the imposed pressure load.1-7

1. The response of complete compensation

Following an elevation of end-diastolic right ventricular pressure, the peak right ventricular pressure is elevated and the cardiac output is maintained with the preservation of normal systemic arterial pressure and slightly elevated or normal atrial pressures.

2. The response of partial compensation

The right ventricular end-diastolic pressure is increased. The peak right ventricular pressure is elevated and the cardiac output is slightly diminished. The right atrial pressure is elevated and the systemic arterial pressures are normal or slightly reduced.

3. The response of decompensation

The right ventricular end-diastolic pressure is increased and the peak right ventricular pressure is elevated. The cardiac output is sharply reduced, the right atrial pressures are elevated and the systemic arterial pressures are markedly reduced.

The creation of an acute systolic load for the right ventricle by partial occlusion of the main pulmonary artery occurs in the clinical situation of banding of the main pulmonary artery, a procedure which is gaining wide acceptance in the surgical treatment of selected forms of heart disease in infants to reduce the high pulmonary flow and pressure in patients with large left-to-right shunts.6-9

The purpose of this study is to define the characteristics of the response of the right ventricle to acute and sustained pressure load of a degree sufficient to allow the right ventricle to adapt by complete compensation.

Methods

Eight adult dogs weighing 16.7 to 22.7 kg. were given morphine ⅛ grain and atropine 1/150 grain as sedation and were then anesthetized with intravenous Nembutal (24 mg. per kg.).10 The trachea was intubated, and respiration was maintained with a positive-pressure respirator using 100% oxygen.

The heart was exposed by an incision in the fourth left intercostal space. After incising the pericardium the main pulmonary artery was carefully dissected and a soft cotton tape was secured loosely about it immediately distal to the pulmonary valve. Cannulæ (Intracath No. 14 gauge) were inserted into the mid right ventricle, into the main pulmonary artery distal to the band and into the ascending aorta immediately distal to the aortic valve; these were secured by purse-string sutures. The right ventricular cannula was inserted through the myocardium at the apex of the ventricle.
The cannulae were flushed with heparinized saline and connected to P23 Db Statham pressure transducers. All tracings were photographically recorded using an Electronics for Medicine DR8 Recorder. Cardiogreen was used as an indicator medium, and all dilutions and injections were accomplished with calibrated syringes. The injection sites were the main pulmonary artery and the mid left atrium. The sampling site was the ascending aorta, immediately distal to the aortic valve. Dye concentration was calculated using a Waters Corporation Blood Densitometer model X-300A. The arterial sample was withdrawn with a reciprocal infusion pump at 32.2 ml. per minute. All blood was reinfused into the ascending aorta after the recording of the curves.

The main pulmonary artery was constricted to elevate the right ventricular peak systolic pressure to 50% of the peak aortic pressure at rest, and the constricting tape was sewn securely and tied. A series of preliminary experiments defined that adjustment to this degree of systolic loading in similar experimental animals was by the response of complete compensation. Pressure determinations and cardiac output determinations were recorded before and immediately after the banding of the pulmonary artery. These measurements were repeated 30 minutes after constriction of the main pulmonary artery was completed.

Penicillin and streptomycin were placed in the thoracic cavity, the thoracotomy was closed and the animals were permitted to survive. Procaine penicillin G 400,000 units and dihydrostreptomycin 0.5 g. were given parenterally daily for three days.

After six months the same animals were restudied using identical techniques.

From the dye dilution curves and the recorded pressure data the following parameters were calculated:

Cardiac output
Right ventricular stroke volume
Cardiac index
Right ventricular work index

Characteristics of:

a) right ventricular pulse curves

b) pulmonary artery pulse curves

c) aortic pressure curves

Right ventricular stroke work
Right ventricular outflow trace resistance
Right ventricular work index

Results

Complete experiments were conducted on eight dogs. The immediate response was characterized by the maintenance of cardiac output (Fig. 1, Table I), heart rate and aortic pressure. The right ventricular systolic ejection period (Fig. 2, Table II), right ventricular end-diastolic pressure, right ventricular peak pressure, right

ventricular mean systolic pressure (Fig. 3, Table III), right ventricular—main pulmonary artery mean systolic gradient, right ventricular work index, right ventricular systolic work and right ventricular outflow tract resistance were

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FIG. 2—Right ventricular ejection systolic period in eight experiments measured in seconds.

FIG. 1—Cardiac output determinations in eight experiments measured in litres per minute.

FIG. 3—Right ventricular mean systolic pressure in eight experiments measured in mm. of mercury.
all increased. The immediate response was not maintained, and after 30 minutes the right ventricular mean systolic pressure, right ventricular—main pulmonary artery mean systolic gradient, right ventricular—main pulmonary artery index, right ventricular systolic work and right ventricular outflow tract resistance were reduced partially; however, the other parameters measured remained unchanged.

When the dogs were restudied six months later, no significant alterations had occurred when responses were compared with those at 30 minutes.

**Comment**

The right ventricle responded to the acute systolic loading by complete compensation. After 30 minutes there was an alteration in the acute response, with a reduction in the right ventricular outflow tract resistance (Table IV). This could have been due to a slackening of the band. An alternative explanation is that there is a transient factor in the acute response, such as infundibular constriction contributing to the total right ventricular outflow tract resistance. Our experiment was limited by the use of open-chest anesthetized animals; this technique interferes with the normal atrial physiology. Arterial oxygen, carbon dioxide and pH were not measured. One hundred per cent oxygen was used to ventilate the animals to avoid the effects of hypoxemia on the pulmonary vascular resistance. The animals were clinically well oxygenated during the experiments and the respirations were maintained during the control and experimental periods.

The present experimental model differs from the clinical situation in which pulmonary artery banding is used in that the ventricular septum is intact. There has been no left-to-right shunt before the banding and there can be no right-to-left shunt after the banding should the right ventricle fail. In the clinical situation the right ventricular pressure can be elevated to 100% of the peak aortic pressure.8, 17 It is not known if the response of the human right ventricle is that of complete compensation in this situation. It is possible that it is not and a right-to-left shunt develops at the ventricular septal defect, allowing the pulmonary flow to decrease below the systemic venous return. Thus the right ventricular outflow resistance can be further increased without the development of right ventricular decompensation. Another obvious factor to be considered is the difference in the ventricular physiology of the adult dog and the child.

In the clinical situation of pulmonary artery banding the hemodynamic objective is complete compensation of the right ventricle with minimal right-to-left or left-to-right shunting or none at all. This is essentially the model we have used.

**Conclusions**

The right ventricle has the immediate capacity to compensate for systolic loading and retains this capacity for long periods of time. The ability to perform increased work is the result of adaptations in right ventricular physiology which increase right ventricular mean systolic pressures and prolong the right ventricular ejection period. The right ventricular output is not altered by acute or sustained increases in total pulmonary vascular resistance within the physiological range which allows it to adapt to the response of complete compensation.

The mechanisms whereby the right ventricular output is regulated are not clarified by this experiment, but presumably it is by a mechanism responsive to volume alterations in the venous return to the right ventricle rather than by one responsive to changes in pulmonary vascular resistance.

**References**