

17082. By-passing the Right Ventricle.*

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A comparative analysis of the physiology of pulmonary and systemic pressures of the several classes of vertebrates indicated that the vascular tree of respiratory organs is a circuit of very low resistance.¹ The pulmonary arterial pressure was found to be of the order of 25/10 mm Hg in fish, amphibiae, reptiles, mammals and birds, while the systemic pressures varied according to class and body temperature.^{1,2} Consideration of these data suggested that the *vis a tergo* for pumping the venous blood through the lungs might be supplied adequately by the venous pressure. Further, in the presence of a congenital or acquired stenosis of the tricuspid or pulmonary orifice, an atrio-pulmonary shunt by-passing the right ventricle might provide a means for complete oxygenation of the entire venous return. Experiments were done on acute and chronic preparations to demonstrate the feasibility of this procedure.

Methods and Results. Acute experiments were carried out on 5 dogs anesthetized with pentobarbital. A cannula was placed in the left atrial appendage and connected with a modified Ludwig Stromuhr. The outflow from the stromuhr was via a cannula tied into the pulmonary artery so that the blood would flow in the direction of the lungs. The pulmonary artery was then tied at its origin. In this way blood was shunted from the right auricle directly to the pulmonary artery without passing through the right ventricle. Pontamine-fast Pink BL (150 mg/kilo) or heparin were used as anticoagulants.

On connecting the stromuhr, blood was seen to flow directly through the glass system. The venous pressure, measured in the right auricle,

was found to be 9 to 14 cm of saline. On tying the origin of the pulmonary artery, the right ventricle ballooned out but the dilation regressed as the experiment continued. The preparations lasted up to one hour.

Chronic experiments were carried out in 5 dogs. Under pentobarbital anesthesia and using artificial respiration via an untracheal catheter, the thorax was opened at the third intercostal space. The pericardium was opened via a longitudinal incision, taking care to avoid the phrenic nerve. The right atrial appendix was clamped by a nontraumatic, specially designed C-shaped forceps.‡ A second C-forceps was used to occlude about 3 cm of the right aspect of the main pulmonary artery parallel to its trunk. Using 5.0 silk on atraumatic needles, the atrial appendage was brought into approximation and anastomosed to the clamped portion of the pulmonary artery. Minimal bleeding was seen on removal of the C-clamps. The pericardium was closed only partially to prevent pericardial tamponade. The thorax was closed with linen ligatures.

The animals showed prompt recovery from the operative procedure. A loud blowing systolic murmur was heard over the entire thorax, loudest at the apex. Venous pressures were found to be about 7 cm of saline. Fluoroscopy indicated a heart size within normal limits. Dogs with these atrio-pulmonary artery anastomoses have been observed for periods up to two months. They show no notable disability. Systemic arterial blood pressures recorded with the Hamilton manometer were about 180/100 mm Hg.

In one dog, after the anastomosis as described above was completed, the origin of the pulmonary artery was tied off almost completely. This procedure tended to kink the anastomo-

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¹ Rodbard, S., Brown, F., and Katz, L. N., *Am. Heart J.*, in press.

² Rodbard, S., *Science*, 1948, **108**, 413.

‡ Machined by Mr. Emil Tiger of the Michael Reese Hospital Medical Research Institute Workshop.

sis. The right ventricle dilated and irregular heart action was seen for several minutes. A loud systolic murmur was heard post-operatively. The dog was well for 12 hours, but was found dead the next morning. Post mortem examination revealed a patent functioning anastomosis of 1 cm diameter.

Discussion. The acute experiments described above demonstrate that the venous pressure may operate as *vis a tergo* to pump at least some of the venous return through the lungs without benefit of right ventricular action. This illustrates anew the remarkably low resistance of the pulmonary circuit. Obstruction to outflow through the pulmonary artery results in a momentary damming of blood in the great veins until the venous pressure rises to levels of about 12 cm saline. A transmission to the venous pressure of some of the pulse from the distended right ventricle continues because of the resulting equivalent of a functional tricuspid regurgitation. However, it is unlikely that this participates in the development of an increased pressure in the right auricle because of the large distensible venous reservoir in free communication with the atrium.

In the chronic experiments with production of the atrio-pulmonary shunt, a functional tricuspid regurgitation is produced because of

the flow of some of the output of the right ventricle via the pulmonary artery anastomosis to the right atrium. The amount of this regurgitation has not been determined. Under normal circumstances the anastomosis results in a flow from ventricle to pulmonary artery to atrium, especially during the heightened pressure of ventricular systole. The possibility must be considered that some blood passes from atrium to pulmonary artery during diastole when the pulmonary diastolic pressure is low, and especially during atrial systole before ventricular contraction occurs. Production of a stenosis or complete occlusion of the pulmonary artery central to the anastomosis forces the flow of blood from atrium directly to the pulmonary artery.

Summary. 1. An anastomosis was made between the right atrial appendage and the main pulmonary artery.

2. In acute experiments, the venous pressure was demonstrated to drive venous return through the lungs without benefit of the pumping action of the right ventricle.

3. In chronic experiments, the anastomosis acts similarly to a tricuspid insufficiency. No deleterious effects were seen in the course of two months observations on such dogs.

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17083 P. A New Imidazoline Derivative with Marked Adrenolytic Properties.

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Intense interest in efforts to combat the adrenergic predominance associated with various vascular dyscrasias has resulted in the development of numerous agents of diverse chemical structure, including the hydrochloride of 2- [N-p'-tolyl-N-(m'-hydroxyphenyl) - aminomethyl] - imidazoline (C-7337).¹⁻³

Toxicity. The acute LD₅₀ values (20 rats per group) are as follows: Intravenous, 75 mg,* subcutaneous, 275 mg, oral, 1250 mg. A brief report of chronic toxicity studies in dogs has been made.⁴

Specific Sympathetic Blocking Effects 1.

¹ Meier, R., *Farmacoterapia Actual*, 1948, 44, 84.

* All doses mg or µg/kg intravenously unless stipulated otherwise.

⁴ Warren, M. R., Woodbury, R. A., and Trapold, J. H., *Fed. Proc.*, 1949, 8, 343.

¹ Marxer, A., and Miescher, K., to be published.

² Meier, R., *New York Academy of Sciences*, 1947, in press.