Systemic Blood Pressure Response to Changes in Right Ventricular Function

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Previous investigations have described areas in the circulation which, when appropriately stimulated, induce changes in total peripheral vascular resistance. Such receptor areas have been demonstrated in vessels of the lesser and greater circulation and three of the heart chambers. Whether similar vascular reflexes can originate in the right ventricle has been questioned. Although increasing pressure in the vascularly isolated left ventricle results in a decrease in total peripheral vascular resistance, a similar response is not elicited from the isolated right ventricle. A reflex increase in pulmonary blood flow without changes in systemic vascular resistance has been demonstrated following elevation of the systolic pressure in the intact right ventricle.

The present report describes a pressor reflex which originates in the intact functioning right ventricle. An increase in systemic blood pressure and vascular resistance is shown after a rise in right ventricular systolic pressure.

Experiments were performed in open-chest anesthetized dogs. The systemic vascular response was measured 1) with the circulation intact, 2) during total perfusion of the systemic circulation with a mechanical pump, 3) after the administration of atropine, and 4) following bilateral cervical vagotomy.

Methods

Fifteen mongrel dogs weighing 10 to 20 kg were anesthetized with sodium pentobarbital (25 mg/kg). A left thoracotomy was performed under positive pressure ventilation maintained with a Starling Ideal pump. A cotton tie was passed around the main pulmonary artery. Intravascular pressures were measured in the inferior vena cava, right atrium, right ventricle, pulmonary artery, and abdominal aorta via polyethylene catheters and recorded on a direct-writing, multichannel oscillograph.

Right ventricular systolic pressure was increased by gradual constriction of the main pulmonary artery at its bifurcation or at the level of the pulmonic valve by gradual tightening of the cotton ligature. After initial studies in dogs with an intact circulation, a mechanical pump, which could be preset to deliver a constant stroke volume at a fixed rate, was connected between the left atrium and descending thoracic aorta (Fig. 1). When blood draining from the left atrium into the reservoir was pumped through the T tube into the aorta, aortic diastolic pressure remained higher than left ventricular systolic pressure. The aortic valves therefore remained closed and the systemic cir-

![FIGURE 1](http://circres.ahajournals.org/)

Schematic diagram of the technique used to bypass the left ventricle. Tygon tube is tied into the left atrium and the pulmonary venous return drained out of the atrium into the open reservoir. Blood is pumped into the aorta through the T tube. Arrows show the direction of blood flow.
culation, including the coronary arteries, was perfused only by blood from the pump. Complete diversion of blood from the left atrium to the pump reservoir was determined by inspection of the aortic pulse contour as previously described.7

The effect of gradual and total occlusion of the main pulmonary artery was then recorded with the vagi intact, following the administration of atropine (0.4 mg/kg) and after bilateral carotid vagotomy.

Results

In each instance as the main pulmonary artery was gradually constricted, right ventricular systolic pressure progressively increased above baseline values. Right ventricular end diastolic, right atrial, and venous pressures remained constant until the main pulmonary artery was markedly constricted (figs. 2 and 3). Mean pulmonary artery pressure showed no consistent changes during the early stages of stenosis. When right ventricular end diastolic pressure began to rise, pulmonary artery pressure and the pump reservoir level

![FIGURE 2](image)

*Effects of increasing degrees of main pulmonary artery constriction in dogs with an intact circulation. \( P_{PA} \) = pulmonary artery pressure. \( P_{RA} \) = right atrial pressure. \( P_{VEN} \) = venous pressure. \( P_{RV} \) = right ventricular pressure. \( P_{Ao} \) = aortic pressure. Straight lines before the instantaneous pressure recordings indicate mean pressures. Panel C represents the immediate preconstriction control. Panels 1, 2, and 3 show responses to graded increases in main pulmonary artery constriction. Fine lines represent 1-sec intervals.*

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declined rapidly. Similar pressure responses following main pulmonary artery stenosis have been reported previously.\textsuperscript{4,8}

Prior to vagotomy, both before and after left ventricular bypass, aortic pressure consistently increased with the elevation of right ventricular systolic pressure (figs. 2 and 3). The graphs in figure 4 show this relationship in ten dogs before there was any rise in right ventricular end diastolic pressure. In table 1, under "Vagi intact," are listed the maximum aortic pressure responses to partial pulmonary artery occlusion in eight dogs with controlled perfusion of the systemic circulation. As also shown in table 1, the increases in aortic pressure in four dogs were similar after the administration of atropine.

Following bilateral cervical vagotomy, aortic blood pressure showed no increase although right ventricular systolic pressure still increased and the ventricular end diastolic pressure remained unchanged (fig. 3 and table 1).

With the left ventricle bypassed, sudden complete occlusion of the main pulmonary artery with total interruption of pulmonary blood flow was associated with an increase in mean aortic pressure of 14 ± 9 mm Hg in seven of nine animals (fig. 5). Following release of the occlusion there was a sudden further rise in aortic pressure. The aortic and right ventricular systolic pressures then returned gradually to baseline levels. After division of both cervical vagi, the systemic responses associated with total main pulmonary artery block were markedly diminished or absent (fig. 5).

**Discussion**

The experimental results show that appropriate stimulation of receptors in the right ventricle influences peripheral vasomotor activity through autonomic nervous pathways. This interaction between the heart and systemic circulation was demonstrated by gradually increasing right ventricular systolic pres-
pressure. The systemic vascular responses were dependent on the presence of intact vagal fibers.

Similar increases in aortic pressure following partial occlusion of the main pulmonary artery have been previously noted in animals with intact circulation. The possible explanations for the systemic pressure rise include increased blood flow, increased activity of the left ventricle, or increased total systemic resistance. In the present experiments, with use of a mechanical left ventricle, flow to the systemic circulation was controlled. The increased aortic pressure while venous and right atrial pressure remained constant represented an increase in total systemic vascular resistance. An increased vascular resistance in the isolated dog hind limb has been described after occlusion of the main pulmonary artery. However in these experiments, total systemic perfusion was not controlled and the increased resistance was attributed to decreased carotid sinus pressure.

Although the increases in right ventricular systolic pressure were similar, the rise in aortic pressure was blocked only by vagotomy and not by the administration of atropine. This difference in systemic response clearly shows that the changes in vascular resistance were dependent on impulses transmitted through vagal afferent fibers from the heart. In addition, the increase in aortic pressure prior to vagotomy could not have been due to release of vasoactive elements directly from the right ventricle into the general circulation or incomplete bypass of the left ventricle.

The possibility that occlusion of the main pulmonary artery could stimulate other receptors on the right side of the circulation in addition to those in the right ventricle has been discussed previously. Since central venous, right atrial, and right ventricular end diastolic pressures showed no change, and pulmonary artery pressure responses were variable, the important stimulus must have been in the right ventricle. A slight decrease in pulmonary artery pressure distal to the site of occlusion has been suggested as a stimulus for the increased aortic pressure. In many instances in the present study, however,
Effects of sudden complete occlusion of the main pulmonary artery in dogs with controlled perfusion of the systemic circulation. Tracings on the left are before, and on the right, after bilateral cervical vagotomy. Brackets at the bottom of each panel show the total time from onset to release of the occlusion. Symbols and time lines are the same as in figure 1.

Pulmonary artery pressure increased with the rise in systemic pressure.

An increase in systemic vascular resistance has also been described following acute over-distention of the main pulmonary artery. The pressures used to distend the pulmonary artery were from 80 to 200 mm Hg. In the present experiments the changes in systemic pressure were obtained while main pulmonary artery pressures were lower. Stenosis near the pulmonic valve or distally near the bifurcation also did not alter the systemic response.

During acute total occlusion of the main pulmonary artery, there was still a direct relationship noted between the elevation in right ventricular systolic and aortic pressures in the pump dog. Under these extreme circumstances there was an associated elevation of right ventricular end diastolic, right atrial, and venous pressures which probably influenced the systemic response. The further increase in pressure which was seen immediately following release of the main pulmonary artery constriction in the pump dog was interpreted as release of vasoinhibitory impulses probably originating in the right atrium while vasoconstrictor impulses from the right ventricle and possibly the veins were still active. Incomplete drainage of the sudden increased blood volume in the left atrium could also have contributed to the post release rise in aortic pressure. This latter possibility, however, would not explain the marked decrease in response noted after vagotomy.

It would appear that in the normal animal as well as during certain pathological states the vasomotor centers in the central nervous system are influenced by nerve impulses transmitted through vagal afferent fibers from the right ventricle. Nervous end organs have been described in histologic preparations of the right ventricle in the endocardium, myocardium, and epicardium. The various nerve endings which were seen are similar to sensory receptors in other areas of the body. Electroneurograms of the vagus nerves also have shown bursts of impulse activity with each ventricular systole.
Summary

The relationship between right ventricular and aortic pressure was studied in open-chest anesthetized dogs. A simultaneous increase in both pressures was observed in animals with an intact circulation and animals with the systemic circulation perfused at a constant rate and stroke volume by an extracorporeal pump. With gradual partial constriction of the main pulmonary artery, the increase in right ventricular systolic and aortic pressures occurred without consistent change in other measured parameters. Bilateral cervical vagotomy abolished the systemic vascular response.

These observations are consistent with a pressor reflex which is mediated over vagal afferent fibers from the right ventricle. It is suggested that in animals under varying physiologic stimuli an increase in right ventricular pressure is associated with a reflex increase in systemic vascular resistance. In abnormal circumstances with increased resistance to right ventricular emptying, the increased total peripheral vascular resistance would be important in maintaining systemic blood pressure and blood flow.

References

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