Title : LEFT VENTRICULAR MECHANORECEPTOR REFLEX FUNCTION

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Introduction. In the neural control of the circulation, the sino-aortic mechano- or baroreflexes are considered the sole regulators maintaining the systemic arterial blood pressure constant by feedback mechanisms. Of the myriad of intrathoracic receptors (pulmonary, atrial, etc.) sending impulses centrally via the vagi, the reflex systemic hypotension initiated by stimulation of the left ventricular (LV) mechanoreceptors, found in many species including man and which overrides the sino-aortic mechanoreflexes (1, 2), was considered by us important in circulatory control. Hypotension from sudden LV balloon distention in pneumonectomized dogs on cardiac bypass (1), suggested that this reflex, in addition to altering the peripheral resistance, also acts on the heart itself to regulate myocardial contractility. Our purpose was to determine the physiological stimulus of the LV mechanoreceptor reflex to determine its role in the body's homeostatic mechanisms.

Methods. The coronary and systemic circulations of pneumonectomized dogs (chloralose-Flaxedil®) on total cardiac bypass were separated by inflation of the terminal balloon of a closed-ended aortic balloon catheter (6-8 mm ID) just above the coronary ostia. A left atrial catheter permitted infusion of blood into the beating LV for ejection into the coronary circulation (Fig.). Large holes in the LV portion of the aortic balloon catheter permitted the LV to eject its contents alternatively via a Starling resistor connected to the catheter's apical end, affording control of the coronary artery perfusion pressure (LV afterload) (insert, Fig.). A working LV preparation resulted in which coronary flow and pressure were controllable while in the separately perfused systemic circulation flow was kept constant so that changes in pressure reflected changes in resistance. Aortic, aortic mean, and LV pressures were measured by strain gauges referred to midchest and recorded along with LV dP/dt and an ECG. Positive or negative inotropic agents were injected into the isolated coronary circulation to determine whether, analogous to the sino-aortic mechanoreflexes, changes in myocardial contractility are able to initiate reflex changes in both the hypo- and hypertensive directions.

Results. Intracoronary injection of norepinephrine (0.001-0.4 µg) in 9 dogs (N=22), control mean aortic pressure 73±3 mm Hg (SEM) and heart rate 147±9, increased the LV peak dP/dt by 278±40% which resulted in a decrease of 25±3% (P<0.001) in the mean systemic arterial pressure at 18 sec. All positive inotropic agents (ouabain (N=4), veratridine (N=15)) also resulted in systemic hypotension which was abolished by bilateral vagotomy, evidence of its reflex nature. Contrariwise, intracoronary injection of 40-1000 mg EDTA (N=7), control mean aortic pressure 83±4 mm Hg, produced a decrease in LV contractility (peak dP/dt) to 46±6% of control in 5 and asystole in 2 dogs, which resulted in a rise in the systemic pressure averaging 11±3% (P<0.05) at 26 sec and which was abolished by vagotomy. All negative inotropic agents (verapamil (N=3), tetrodotoxin (N=4)), produced similar rises in systemic pressure. Thus, changes in LV contractility reflexly initiate directionally opposite changes in systemic resistance via LV mechanoreceptors which can override the sino-aortic mechanoreflexes.

Discussion. From these results, the most important function of the LV mechanoreceptors is the reduction of the peripheral resistance in order to prepare the systemic circulation to receive the LV stroke volume with minimal alteration in the systemic pressure. Thus, in exercise the LV mechanoreceptor reflex permits the systemic vascular bed to receive the increased cardiac output without a deleterious rise in systemic pressure. Their location in the LV myocardium also permits the LV mechanoreceptors to sense the force of myocardial contraction immediately at the time that this force is generated and thus by means of neural feedback mechanisms to the heart to actively control the development of the pressure by the LV for perfusing the systemic circulation.

References.

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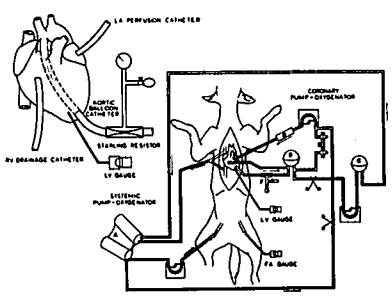

Separate Coronary & Systemic Circuit Perfusion