Title: EFFECTS OF SYSTEMIC ARTERIAL HYPOTENSION ON PULMONARY VASCULAR RESISTANCE IN CONSCIOUS DOGS

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Introduction. Although it is well known that systemic arterial hypotension is associated with an active compensatory increase in systemic vascular resistance (SVR) which is mediated by reflex sympathetic adrenergic activation, concomitant changes in pulmonary vascular resistance (PVR) have not been clearly delineated. Utilizing a chronically-instrumented, conscious animal model to avoid potentially confounding influences of anesthesia and surgical trauma on the pulmonary circulation, our objectives were to determine: 1) whether PVR increases during hypotension, 2) whether changes in PVR during hypotension are mediated by sympathetic adrenergic activation, and 3) whether the choice of "downstream" pressure (i.e. left atrial pressure or pulmonary capillary wedge pressure) affects apparent changes in PVR in response to hypotension.

Methods. Experiments were performed on 7 conscious dogs chronically instrumented with catheters in the thoracic aorta, main pulmonary artery and left atrium to measure pressures. Cardiac output (thermal dilution) and pulmonary capillary wedge pressure (PCWP) were measured with a Swan-Ganz catheter acutely positioned in the pulmonary artery via percutaneous jugular puncture on the day of the experiment. SVR and PVR were measured during normotension (100±4 mmHg) and systemic arterial hypotension (53±3 mmHg) induced by partial constriction of the thoracic inferior vena cava. Measurements were made with nerves intact and following sympathetic adrenergic block with iv propranolol (1 mg/kg) and phentolamine (2 mg/kg). Data were analyzed by two-way analysis of variance and are presented as Mean ± 1 SEM.

Results. As summarized in Figure 1, systemic arterial hypotension elicited a significant increase ($p<0.01$) in SVR, which was abolished by adrenergic block. When left atrial pressure (LAP) was used as the "downstream" pressure to calculate PVR, hypotension was associated with a significant increase ($p<0.01$) in PVR both with nerves intact and following adrenergic block (Figure 2: top panel). However, stepwise constriction of the thoracic inferior vena cava (Figure 3) demonstrates that when cardiac output decreases below approximately 2.5 lit/min during hypotension, LAP decreases to a negative value, whereas PCWP decreases toward the level of mean alveolar pressure (i.e. approximately 0 mmHg during spontaneous respiration). Under these circumstances, PCWP and not LAP is the effective "downstream" pressure for the pulmonary circulation. When PCWP is used as the "downstream" pressure to calculate PVR, PVR is not increased during hypotension (Figure 2: bottom panel).

Conclusion. Thus, unlike the systemic circulation, systemic arterial hypotension does not result in reflex constriction of the pulmonary circulation in conscious dogs. During hypotension, calculated changes in PVR are critically dependent on the use of PCWP, and not LAP, as the effective "downstream" pressure. Importantly, the use of LAP as the "downstream" pressure can lead to erroneous conclusions concerning the effects of physiological and pharmacological stimuli on pulmonary vascular resistance.

Fig. 1

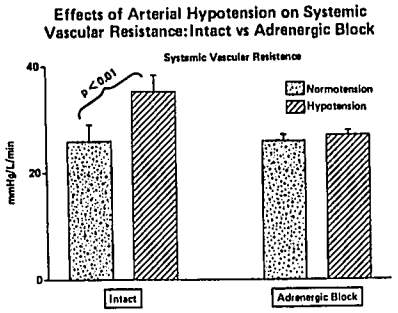

Fig. 2

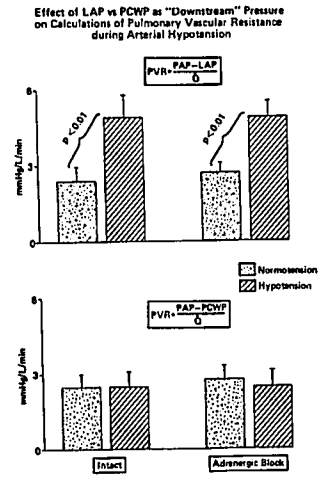

Fig. 3