Title: WHAT IS THE APPROPRIATE THERAPY TO MAINTAIN CARDIAC OUTPUT AS PULMONARY VASCULAR RESISTANCE INCREASES?

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Introduction. In some patients with severe acute respiratory failure and elevated pulmonary vascular resistance (PVR), the increased work load on the right ventricle (RV) may be an important factor limiting survival. Despite this, effects of treatment on right ventricular function in this setting have not been systematically investigated. Traditionally, volume loading has been suggested as the treatment of choice when cardiac output is reduced because of increased RV afterload. Alternatively, volume infusion could increase right ventricular wall stress and cause a deterioration in right ventricular function. Accordingly, this study was designed to compare the effects of gradual volume expansion to continuous inotropic support on RV function as pulmonary vascular resistance was gradually increased.

Method. In 10 anesthetized dogs, ventilated with 40% oxygen, we measured cardiac output (CO), arterial blood gases and relevant hemodynamic parameters. Pulmonary vascular resistance was calculated as PVR = PAP - PCWP. Dogs were randomly assigned to treatment with either volume (5 dogs) or dopamine (DP) (5 dogs). After baseline measurements, PVR was raised gradually by IV injection of glass beads (80-120 μ). This intervention lead to a gradual reduction in CO. Treatment with volume (albumin or whole blood) or dopamine began when CO fell below 90% of baseline. To increase CO or ≥ 90% of baseline, volume was slowly infused in 50 ml aliquots whereas in those dogs receiving DP the infusion rate was adjusted (2-15 μg/kg/min⁻¹) until the desired response was obtained. If the goal of therapy was reached, more beads were given to further increase the RV afterload and when CO fell, additional volume was given or the rate of DP infusion increased.

Results. The effect of treatment on cardiac output at increasing levels of PVR is illustrated in the figure. When cardiac output was less than 80% of baseline or, with one exception, when PVR was greater than 11 mmHg/litre.min⁻¹, volume infusion increased right ventricular end diastolic pressure (RVEDP), reduced RV systolic pressure (RVSP) and either reduced CO or had no effect. The mean RVEDP at which volume infusion caused RV dysfunction was 6 mm Hg. Also, despite a progressive fall in CO with volume, BP remained relatively constant. Conversely, dopamine consistently increased CO and BP while it reduced RVEDP. These results were significant at P < 0.05 by the 2 sample t-test.

Discussion. These results indicate that when RV afterload is significantly elevated volume expansion can lead to deterioration in RV pump function i.e. reduced CO and RVSP despite increased RVEDP. Conceivably, by increasing RV wall stress and RVO requirements, volume expansion results in RV ischemia and a deterioration in ventricular performance. That RVEDP volume increased with volume infusion when RVEDP increased was confirmed using equilibrium scintigraphic techniques. The relatively low RVEDP (6 mmHg) at which volume expansion caused deterioration in RV function indicates that RVEDP may be a poor predictor of the response to volume in patients with high RV afterload. In such patients volume infusion may not be the therapy of choice to increase CO. Alternatively, an inotropic agent may be preferable.

References: