Title: PLEURAL EFFUSIONS CAN BE CAUSED BY SUPERIOR VENA CAVAL HYPERTENSION

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Introduction. The occurrence of pleural effusions (PE) is relatively frequent in the critically ill population, especially those patients with congestive heart failure. Because these patients often have both increased central venous and pulmonary venous pressures, the exact role of either in the formation of PE is not clear. Increased central venous (superior vena caval) pressure could cause PE by slowing lymphatic drainage from the pleural cavity and by obstructing lung lymph flow sufficiently to result in leakage of lymph out of the lymph nodes and into the pleural space. The objective of the present study was to determine the effect of long term elevation of SVCP on the formation of PE.

Methods. Eleven sheep were chronically instrumented with catheters for monitoring of SVCP, pulmonary artery pressure and arterial blood pressure. Through a right thoracotomy, a silastic cuff was placed around the superior vena cava and the tube to inflate the cuff was brought out through the incision. After recovery for at least one week, baseline pressures were measured and cardiac output determined by dye-dilution. SVCP was elevated with the silastic cuff to a constant level between 3 and 45 cm H_2O and maintained for 24 hours. Cardiac output was remeasured 1-2 hours after cuff inflation and again at 24 hours. At the end of the experiment, the chest was opened, pleural fluid removed and the volume measured. Plasma protein (Cp) and pleural protein concentrations (Cpl) were measured and the ratio (Cp/Cpl) determined.

Results. The effect of elevated SVCP on the volume of PE is displayed in the Figure. We found no significant change in PAP, blood pressure or cardiac output following SVCP elevation. The Cp/Cpl was 0.54 ± 0.1 in those sheep where SVCP was $\geq$ 20 cm H_2O versus $0.27 \pm .05$ when SVCP was $<$ 20 cm H_2O.

Conclusions. Our data show that elevation of SVCP above 20-25 cm H_2O for 24 hours causes PE, the volume of which is related to the magnitude of the SVCP elevation. Lymphatics that drain the lung and the pleural cavity empty into the superior vena cava. When pressure in the superior vena cava is elevated, the rate of lymph flow is decreased[1]. Obstructing lymph flow could enhance the formation of PE by two mechanisms. First, by impeding lymph drainage of the pleural cavity, fluid could collect that would normally empty into the superior vena cava. Second, impairment of lymph flow from the lung could result in lymph to leak out of the lymph nodes and pass into the pleural space.

In a previous study we did not find any PE when SVCP was elevated for only three hours. As patients often have elevated venous pressures for more than a few hours, this study underscores the need for further long term studies in the investigation of PE.

Reference.
1. JAP 61:1634-1638, 1986

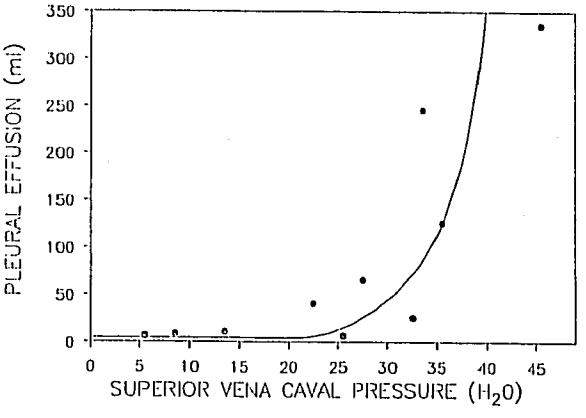

Figure. Amount of pleural effusion formed after 24 hours of superior vena caval pressure elevation.

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