atrial volume, rather than the total blood volume, by appropriate volume infusion.

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In Reply—The comments and the hypothesis expressed by Dr. Urzua are pertinent and exciting. At present, the exact mechanical parameter responsible for ANF release is not known. Theoretically, it could be intratricular pressure or volume, stretching tension of the atrial wall, or intramyocardial tissue pressure. In this respect, the effect of PEEP is interesting but complex. In terms of transmural right atrial pressure (RAP TM), there are conflicting experimental and clinical data. Following cardiac surgery, we have measured intraluminal RAP and plasma concentration of ANF (pANF) before, after 15 min of PEEP 15 cm H2O, and after discontinuing PEEP. We found a linear relationship between the percent change of RAP and the percent change in pANF (fig. 1). When RAP was greatly increased, pANF was increased, and when RAP did not change, decreased, or only slightly increased, pANF was decreased. We conclude that ANF secretion during PEEP is largely dependent on the initial filling pressure and volume status. Furthermore, the finding that ANF release was only seen with a large increase in RAP suggests that ANF stimulation is more related to the transmural pressure than the intraluminal pressure.

It is clear that there is a blood volume redistribution during PEEP. Using a G Suit with moderate inflation, we have corrected the peripheral blood pooling induced by PEEP. Unfortunately, the correction of cardiopulmonary blood volume, cardiac output, systemic arterial blood pressure, and renal blood flow associated with a large increase in RAP failed to increase diuresis and natriuresis.

In addition, while stimulation of ANF was shown to occur in numerous disease states, such as congestive heart failure, cirrhosis, or paroxysmal atrial tachycardia, an inhibition of ANF release was only demonstrated during lumbar epidural anesthesia. At present, there is no evidence that an inhibition in pANF release could induce an antidiuresis. In conclusion, we think that renal function alteration during PEEP is probably not under the control of a single mechanism, but results from the interaction between cardiovascular reflexes and stretch reflexes from the lung.

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REFERENCES