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To the Editor:

We appreciate the interest of Masa-Jimenez et al in our article. The development of a prolonged course of adult respiratory distress syndrome following upper airway obstruction (UAO) was noted as early as Oswalt's initial recognition of the syndrome.1 Presumably, this evolution occurs because of concomitant prolonged hypoxia, shock, or perhaps other associated physiologic derangements, as in the myxedema coma cases reported by Masa-Jimenez. Fortunately, this more severe course after UAO appears to be exceedingly uncommon and, as pointed out in our review, the vast majority of patients with pulmonary edema after UAO will recover promptly.

Whether or not "auto-PEEP" plays a role in delaying the onset of pulmonary edema associated with UAO in some cases remains to be seen. Dr. Masa-Jimenez' postulate that the fixed or variable nature of UAO may determine time of onset of pulmonary edema seems reasonable. However, this remains a difficult area to study due to the dramatic and urgent nature of acute UAO.

Since publication of the article, I have seen two additional cases of self-limited pulmonary edema in the setting of post-extubation laryngospasm, underscoring the notion that laryngospasm appears to be the most common culprit in upper airway obstruction-associated pulmonary edema.

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Assessment of Right Ventricular Function

To the Editor:

I read with interest Dr. Albert's editorial in Chest entitled "Assessment of Right Ventricular Function." His editorial was largely a criticism of our paper, published in the same issue of the journal.1 The major criticism was related to our method of measuring right ventricular ejection fraction using a technique we have previously reported.2 In this previous paper, we presented a detailed critique of the use and reproducibility of gated equilibrium radionuclide ventriculography to measure right ventricular ejection fraction (RVEF). Dr. Albert criticises our method on two counts.

First, he criticizes the contribution of the right atrium to the right ventricular region of interest using this technique. He quotes an experiment from our previous paper using a fixed human heart filled with radio-isotope which was imaged in a 20° LAO position.3 The experiment indicated that that, in this projection, although separation between the right and left ventricles was good, the right atrium contribution to the right ventricular counts was 30 percent. He neglected to mention that, for this reason, in our study we elected to discard the fixed region of interest for the right ventricular outline which would result in a significant under-estimation of the RVEF. Instead, we drew regions of interest around the right ventricle—both at end-diastole, when the atrium largely will have emptied, and at end-systole, when the atrium will have filled—but will not be included in the right ventricular end-systolic region of interest.

We do agree that Figure 3 in our previous paper4 appears to indicate that there is a slightly greater difference between the RVEF measured by the first pass and equilibrium techniques at lower values of RVEF. However, closer inspection of the correlation suggests that the deviation of the points from the line of identity is due to an over-estimation of RVEF using the first pass technique.

Finally, Dr. Albert also criticises our study on right ventricular function in patients with COPD,5 principally because we measured only one point on the right ventricular pressure/volume relationship. This problem is also dealt with in our paper. The major thrust of presenting the end-systolic pressure/volume relationship in Figure 2 is to relate end-systolic volume in our patients to the calculated end-systolic volume in normal subjects. The figure clearly suggests that the presence of pulmonary hypertension (which was present in 72 percent of our patients) does not correlate with the right ventricular end-systolic volume. Furthermore, many patients with pulmonary hypertension had normal right ventricular end-systolic volumes, suggesting relatively normal right ventricular contractility even in the presence of pulmonary hypertension.

In order to overcome the difficulty of measuring the slope of the pressure/volume relationship (necessitating the use of pure pulmonary vasodilators, none of which are very satisfactory), we extended our measurements by examining the change in the relationship during exercise. We measured both the end-systolic pressure/volume relationship and—in order to confirm our observations—the relationship between stroke work index/stroke volume index and right ventricular end-diastolic pressure. These latter measurements are independent of our assessment of the right ventricular volume derived from the RVEF. Both sets of data suggest relatively normal right ventricular contractility, despite increasing pulmonary arterial hypertension.

We believe that these data add to our knowledge of right ventricular function in patients with hypoxic pulmonary hypertension and support our findings of a lack of correlation between pulmonary arterial pressure and right ventricular ejection fraction, as well as indicating relatively normal right ventricular function in such patients. We do not agree with Dr Albert's implication that our study should be "viewed with scepticism".

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