HIV infection in the pathogenesis of respiratory function alterations in IHA is still unknown. Since HIV has been isolated in lung tissue of patients with AIDS, it is possible to hypothesize that the Dco reduction in IHA could be caused by an alveolitis due to the direct localization of HIV in lung tissue or to an immune-mediated response to HIV antigens. In order to evaluate the role of HIV infection in alterations of respiratory function in IHA, we prospectively studied a group of 33 IHA (23 men and ten women), 12 HIV-negative and 21 HIV-positive. Six belonged to group II CDC classification system, 11 to group III and four to group IV. For all patients the following tests were taken: clinical examination, hemoglobin concentrations, CD4 and CD8 lymphocyte counts, anti-HIV antibodies assay (ELISA and Western blot), chest x-ray film, respiratory function tests (TLC, FEV1, FVC, FEV1/FVC and Dco expressed as a percentage of predicted values) and blood gas analysis (pH, PaO2, PaCO2, SaO2). None of the subjects presented with respiratory symptoms and/or infiltrates on chest x-ray film. In both groups and for each smoker, cigarette consumption in pack/years was calculated as the number of packs (20 cigarettes/pack) smoked per day multiplied by the number of years the subject smoked. Data were analyzed by the Student's t-test. A "p" value less than 0.05 was defined as significant. All values are expressed as means ± SD. HIV-positive subjects did not show statistically significant differences compared with HIV-negative subjects for age (26.3 ± 5.1 years, ranging from 17 to 43, vs 29 ± 4.1 years, ranging from 24 to 35); length of intravenous drug use (7.3 ± 3.6 years, ranging from one to 15, vs 7.2 ± 4.8 years, ranging from 1 to 18); number of cigarettes smoked (13.8 ± 7.7 pack/years, ranging from 0 to 30, vs 16.8 ± 7.4 pack/years, ranging from 0 to 26); hemoglobin concentrations (14.3 ± 1.2 g/dl, ranging from 12.5 to 16, vs 14.7 ± 1.6 g/dl, ranging from 12.1 to 17.1); and CD8 lymphocyte counts (1.088 ± 0.444 x 109/L, ranging from 0.296 x 109 to 2.043 x 109, vs 0.922 ± 0.514 x 109/L, ranging from 0.222 x 109 to 1.866 x 109/L). HIV-positive subjects showed a statistically significant reduction in Dco compared with HIV-negative subjects (74.6 ± 14.1 percent, ranging from 50 to 107, vs 86.9 ± 6.6 percent, ranging from 79 to 100, p < 0.01) and CD4 lymphocyte counts (0.785 ± 0.428 x 109/L, ranging from 0.145 x 109 to 1.715 x 109, vs 1.236 ± 0.464 x 109/L, ranging from 0.848 x 109 to 2.051 x 109, p < 0.01). Significant correlations between Dco and CD4 lymphocyte counts were not found. Dco was < 80 percent in 15 of 19 (79 percent) HIV-positive subjects and in one of 11 (9 percent) HIV-negative subjects. The four HIV-positive subjects with normal Dco (> 80 percent) were in group III and IV of the CDC classification.

These data show that HIV-positive IHA present more frequently than HIV-negative IHA alterations in respiratory function, suggesting interstitial pulmonary disease. The absence of respiratory symptoms and/or chest x-ray abnormalities suggests subclinical pulmonary disease or an interstitial pneumopathy due to HIV. An alveolitis composed of cytotoxic T-lymphocytes directed against HIV-infected alveolar macrophages has been effectively demonstrated in patients with HIV infection. Since the Dco reduction is prevalent in HIV-positive subjects, since it is already present in the asymptomatic stage of infection and is not related to CD4 lymphocytes reduction, we suggest that pulmonary involvement could be due to a direct action of HIV rather than a subclinical opportunistic infection.

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Pulmonary Edema Due to Upper Airway Obstruction

To the Editor:

We have read carefully the report, "Pulmonary Edema due to Upper Airway Obstruction in Adults" by Willms et al (Chest 1988; 94:1090-92). We treated six adult patients with noncardiogenic pulmonary edema. This edema was attributed to upper airway obstruction (UAO). Two of these were due to laryngospasm, another to acute laryngeal obstruction with food, and the other three appeared in the context of a myxedema coma with severe obstructive apneic episodes which have been reported as a cause of pulmonary edema.

In agreement with Willms et al, the evolution was benign in the early three cases with resolution of the pulmonary edema before 72 h. Nevertheless, the development of myxedema comas was fatal due to respiratory distress, with progressive increase in intrapulmonary shunt and dead space, marked decrease of static compliancy and pulmonary hypertension with normal pulmonary capillary pressure. We don't know if inherent alterations in myxedema (such as inadequate lymphatic drainage of proteins) might have played a role in that evolution.

In his report, Dr. Willms comments on the frequent existence of pulmonary edema when UAO is released due to the fact that an "auto-PEEP" effect would protect the edema against transudation of fluid while the acute obstruction remains. However, pulmonary edema has been found before the release of the obstruction. We have pointed out that the presence of pulmonary edema before or after the release of the obstruction can depend on the fixed or variable behavior of the UAO. The fixed behavior, perhaps more habitual, might produce "auto-PEEP" with a protective effect and the edema would raise on releasing the obstruction. By contrast the variable behavior would not produce "auto-PEEP" and edema might develop before the relief of UAO.

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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 Oswald'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 myzempeda comas 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."1 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 disregard 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 conform 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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