Pulmonary Edema and Upper Airway Obstruction

To the Editor:

Recently, Dr. Sofer et al (Chest 1984; 86:401-03) suggested that the pulmonary edema of patients with upper airway obstruction (UAO) was roentgenologically invisible because of the increase in lung volume before the relief of the obstruction. In Dr. Sofer’s article, as well as in others,1-3 objective dates of increase in lung volumes are not reported. Research in patients with chronic UAO showed different results in their volumes. However, in studies of healthy volunteers with experimental stenosis,4 a maintenance or a decrease of their dynamic and static lung volumes was observed. If we were to admit an increase of FRC with or without increase in TLC following UAO, a descent of the diaphragm would occur. Consequently, the minimal pleural pressure (Pplmin) becomes less. Thus, if the descent of the diaphragm is close to the maximum (TLC), the Pplmin will only reach the maximal static recoil pressure. In contrast, the stability of FRC, or its decrease, would permit it to reach a Pplmin close to the maximal inspiratory muscular pressure. A similar but inverse argument can be applied to the expiratory pressure.

It has been suggested that a severe attack of asthma could favor the formation of pulmonary edema,5 but wide clinical experience contradicts this hypothesis. Therefore, it is probable that the mechanical alterations following hyperinflation could explain partially this contradiction. In summary, we believe that there are neither objective nor theoretic data to support the notion that these patients experienced an increase in lung volume. If this increase were enough to mask roentgenologically the pulmonary edema, it would be necessary to call in question the effect of the highly negative pressures in the origin of the pulmonary edema.

On the other hand, we suggest that the appearance of the pulmonary edema before or after the relief of UAO would have to be related to its fixed or variable behavior. The fixed behavior, on increasing the expiratory pressure, would counteract the effect of the inspiratory pressure and the edema could be developed with the relief of the obstruction. In the variable behavior there would exist a clear prevalence of inspiratory pressures and the edema could be developed before the relief of the obstruction.


REFERENCES

REFERENCEs
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Isoxsuprline and Pulmonary Edema

To the Editor:

We have read with great interest the article by Gozo and Yebe (Chest 1984; 86:736-40). This report shows that, following administration of a 10 mg IV bolus of isoxsuprline to twelve patients with acute pulmonary edema, there is an increase in cardiac index up to 31 percent and a drop in pulmonary artery wedge pressure by as much as 33 percent. The authors go on to suggest that isoxsuprline may be a valuable adjunct in the therapy of acute cardiac failure. Isoxsuprline possesses several pharmacologic characteristics besides its vasoactive properties. It is a potent uterine muscle relaxant and has been prescribed as a tocolytic agent for pregnant women in premature labor.

Several beta agonists have been implicated as a cause of pulmonary edema during premature labor.1,4 We recently reported a 0.5 percent incidence of pulmonary edema among 1,407 pregnant women treated with parenteral isoxsuprline over a seven year interval.5 The infusion rate ranged from 0.1 mg/min to 0.55 mg/min over a 24 to 72 hour period. The seven patients who developed pulmonary edema all had normal cardiac function otherwise, and the pulmonary edema responded promptly to oxygen, diuretics and withdrawal of isoxsuprline. For tocolysis, the drug necessitates infusion of extra fluids to counteract systemic hypotension. It increases hemodilution and, as a peripheral vasodilator, it eventually results in retention of fluid and an increase in both pulmonary blood volume and ventricular dilatation. Isoxsuprline also reduces plasma colloid osmotic pressure and has been reported, in high doses, to induce experimental myocardial necrosis.1,4

In their study, Gozo and Yebe did not prime their patients with parenteral fluids and, as a consequence, there occurred a significant

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