Risks and Benefits of Peritoneal Dialysis

To the Editor:

Epstein et al.1 recently reported that two liters of dialysis fluid had little or no deleterious effect on ventilatory function in the patients they studied with mild COPD. In an accompanying Editorial2 it was suggested that, during peritoneal dialysis, the diaphragm became more curved when fluid was introduced into the peritoneum because of the increased peritoneal pressure. The muscle might therefore be capable of generating more force as a result of its altered length/tension relationship.

Singh et al.3 have extended these observations in their prospective serial study of pulmonary function in patients undergoing CAPD, finding that the decrease in pulmonary volume that occurs immediately after CAPD is not associated with any defect in gas exchange or flow rates in the lung. As lung volumes returned to baseline values within two weeks in all patients, including those with mild chronic obstructive pulmonary disease, they concluded that CAPD caused no significant compromise in lung function, even in those with mild COPD. They did, however, caution that baseline studies of pulmonary function should be obtained before insertion of the abdominal catheter. In an accompanying Editorial,4 Winchester appropriately drew attention to the complications of CAPD and the high incidence of pulmonary complications in acutely ill patients with renal failure. We agree with Winchester that higher volumes of dialysis fluid (3 or 4 L) are not tolerated by some patients.

Winchester concluded that “it remains to be seen whether peritoneal dialysis can be used in patients with moderate-to-severe respiratory diseases”, with which we agree. To our surprise however, he proceeded to say that, based on present evidence, most would choose hemodialysis for renal failure patients with severe impendiment of pulmonary function. Obstructive lung disease has been found to be one of the risk factors that influence the survival of patients on hemodialysis, along with old age, diabetes and arteriosclerosis.5 We believe that present evidence does not exclude peritoneal dialysis from these patients, especially if performed with less than 2 L of peritoneal fluid, and that more concrete evidence should be available before excluding this form of therapy, especially as these patients are also known to be at risk on hemodialysis.5

Drs. Oreopoulos and Rebuck have taken issue with my statement, “Based on present evidence, most clinicians would chose hemodialysis for patients with renal failure in the face of major impairment of pulmonary function.” I think the sentence still stands since peritoneal dialysis may be associated with severe reductions in arterial Po2. Indeed, Khanna and Oreopoulos2 have recently drawn attention to falls in Po2 of 3 to 26 mm Hg when the abdomen is distended with 2 L of dialysate during intermittent peritoneal dialysis in acutely ill patients. Certainly, it could be argued that a fall of 3 to 26 mm Hg could be quite hazardous to someone with moderate-to-severe respiratory disease. I also acknowledge that chronic obstructive pulmonary disease is a significant risk factor for survival of patients on hemodialysis, as pointed out recently by Shapiro and Umen.6 However, careful perusal of the manuscript of Shapiro and Umen does not give any indication as to the degree of impairment of pulmonary function. Additionally, in the accompanying figures of probability of survival versus time, the effect of chronic obstructive pulmonary disease (COPD) alone on survival is not clear, since in all of the survival diagrams the combined risk of arteriosclerotic heart disease and chronic obstructive pulmonary disease is portrayed. In the tables, however, COPD unquestionably is a significant risk factor. In addition, the deaths from pneumonia totaled four and pulmonary embolism three, giving a total of 123 PubMed-related deaths (5 percent) out of 141 total deaths. Therefore, one is brought to the conclusion that pulmonary-related deaths in hemodialysis patients over or under the age of 61 years is a relatively infrequent event compared to cardiovascular deaths (48 percent), and one is also led to the conclusion that chronic obstructive pulmonary disease as a major risk factor in the absence of arteriosclerotic heart disease may be a lesser prognostic risk factor in such patients. I would, however, agree with Drs. Oreopoulos and Rebuck that “more concrete evidence should be available before excluding” peritoneal dialysis in chronic obstructive pulmonary disease patients, and expect that this would require increased frequency of dialysis exchanges if using lower dialysis fluid volumes, for both intermittent and continuous ambulatory peritoneal dialysis.

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RECOMMENDATIONS FOR THE READER


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,1,2 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 (Ppmin) becomes less. Thus, if the descent of the diaphragm is close to the maximum (TLC), the Ppmin will only reach the maximal static recoil pressure. In contrast, the stability of FRC, or its decrease, would permit it to reach a Ppmin 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,2 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 before1,4 or after1 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 pressure2 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.

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To the Editor:

We thank Masa-Jimenez et al for their interest in our article. We did not wish to imply that changes in lung volume were the only possible mechanism at work here. Since we did not measure lung volume at FRC, we can only speculate that this might have been increased. The fact that the diaphragms were not visibly flattened does not rule out the possibility that lung volume at FRC was increased, since this increase could have occurred by increasing rib cage volume rather than shortening and descent of the diaphragm.

Regarding changes in pleural pressure swings, although it is true that maximum pleural pressure generated by performing a maximal inspiratory maneuver decreases as one approaches TLC, this does not imply that the negative swings in pleural pressure do not increase even with increasing FRC. During attacks of asthma in humans, maximum pleural pressure during inspiration decreases considerably, even though FRC increases.1,4 We are unaware of any data that refute or confirm the hypothesis that there is a tendency for edema formation during asthma, and suggest that this article be a subject for future investigation.

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Isoxsuprine and Pulmonary Edema

To the Editor:

We have read with great interest the article by Gozo and Yebes (Chest 1984; 86:736-40). This report shows that, following administration of a 10 mg IV bolus of isoxsuprine 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 isoxsuprine may be a valuable adjunct in the therapy of acute cardiac failure.

Isoxsuprine possesses several pharmacologic characteristics besides its vasodilatative properties. It is a potent uterine muscle relaxant and has been prescribed as a tocolytic agent for pregnant women in premature labor.

Several beta2 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 isoxsuprine over a seven year interval.2 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 isoxsuprine. 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. Isosuxprone also reduces plasma colloid osmotic pressure and has been reported, in high doses, to induce experimental myocardial necrosis.4

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

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