To the Editor:

Ibáñez and Raurich had several questions regarding our study of head-injured patients. It is true that patients with head injury often have other injuries, but we believe that reductions in functional residual capacity (FRC) and elevated shunt fractions (Qs/Qt) are common findings even in those patients with isolated head injury. Of the ten patients we studied whose chest roentgenograms were normal, six had no evidence of other major trauma. Mean FRC for these six was 74 percent of predicted; mean FRC was 71 percent of predicted for the four with other trauma (p = NS). FRC percent predicted and Qs/Qt tended to be linearly related for these six patients (r = 0.72), but this relationship was not statistically significant due to the small sample size.

The predicting formulae we selected are widely used and recommended and the values given for the three female patients are in fact correct. Drs. Ibáñez and Raurich have a valid point that the measurements used to generate the predicting formula for men were made in a semirecumbent position. While we were technically in error to refer to this formula as generating a "predicted value for the upright position," FRC is larger in a sitting or standing position than it is in the fully recumbent position. FRC in a semirecumbent position must lie between these two extremes. The mean FRC of our male head-injured patients was 71 percent of the value predicted for the semirecumbent position. Our observed FRC would therefore have been even less than 71 percent of a value predicted for the fully upright position.

The mean FRC of 11 head-injured patients studied by Ibáñez and Raurich (1.84 liters) was virtually identical to the mean FRC we measured in our 24 patients (1.83 liters). Although it may be within normal limits for an individual's FRC to be 81 percent of predicted, it is distinctly abnormal for a group of 11 patients to have a mean FRC which is 81 percent of predicted. FRC for their 11 patients (1.84 ± 0.40) is significantly smaller than 2.27 ± 0.56 (p < .005 by t test). For this comparison, 2.27 is the mean predicted FRC of their 11 patients, and 0.56 is the standard deviation of FRC of their published normal male reference population.

The reduction in FRC which we found was only part of the evidence for the role of reduced lung volume in the production of hypoxemia. We also found a highly significant linear correlation between FRC and Qs/Qt. If reduced FRC did not contribute to hypoxemia there should be no such relationship. Lastly, we agree that the high Qs/Qt we observed is predominantly due to a mismatch of ventilation and perfusion. However, this is a physiologic mechanism for hypoxemia and not a pathophysiologic explanation of why the mismatch exists. Reduction of FRC below closing capacity is thought to play a major role in the production of hypoxemia in obese patients and older patients with mild airways disease, particularly in a supine position. We did not measure closing capacity in our patients, but thought it helpful to point out that our patients' measured FRC was reduced enough to expect that it would be smaller than closing capacity.

We did not wish to imply that any of the mechanisms we described were unique to patients with head injury. On the contrary, we hypothesized that well known pathophysiologic processes producing mismatch of ventilation and perfusion can explain the hypoxemia which is commonly found in these patients and we presented the data to support this hypothesis.

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Trendelenburg Positioning to Correct Hypoxemia from Chest Trauma

To the Editor:

We recently treated a patient with bilateral pulmonary contusions where the use of physiologic principles governing ventilation-perfusion (V/Q) relationships resulted in reversal of life-threatening hypoxemia refractory to conventional methods of ventilation. A 22-year-old man was admitted with flail sternum and bilateral pulmonary contusions confined to the lower lung fields secondary to blunt trauma. Arterial blood gas levels showed a pH of 7.30, PaO_2 of 53 mm Hg, and PaCO_2 of 46 mm Hg on a 30 liter per minute oxygen mask. Following intubation, we attempted to improve oxygenation with positive end-expiratory pressure (PEEP), but this caused a dramatic increase in PaCO_2 to 70 mm Hg. There was no mechanical reason for this increase in dead space, such as pneumothorax or partially occluded endotracheal tube. The fraction of inspired oxygen (FIO_2) was temporarily increased to 90 percent, but the PaO_2 remained 50 mm Hg on 18 cm H_2O PEEP. Decreasing PEEP caused worsening of his hypoxemia, yet increasing the level caused a severe respiratory acidosis.

Cardiac abnormalities did not contribute to his hypoxemia as evidenced by a cardiac output of 12 liters per minute and a normal mixed venous oxygen tension and saturation.

A portable perfusion lung scan obtained to rule out massive pulmonary embolus showed normal perfusion to both upper lung fields with bilateral defects present only in the areas of contusion seen on chest roentgenogram. Because of these findings we placed the patient in a 60° Trendelenburg position to increase perfusion by gravity to the normal alveoli of the upper lobes. This resulted in an increased PaO_2 from 50 to 63 mm Hg. The FIO_2 was decreased over the next four hours to 50 percent while PEEP was gradually increased to maintain arterial oxygen saturation above 90 percent. We left the patient in this position for 12 hours as his PaO_2 gradually increased to greater than 120 mm Hg on 40 percent FIO_2. The patient was extubated five days later and developed no further problems.

We postulate that the dramatic increase in dead space as shown by the immediate rise in PaCO_2 was due to overdistension of normal alveoli in the upper lung fields. Positional therapy for unilateral lung disease has been previously described. Placing our patient in a steep Trendelenburg position may have caused just enough alteration in V/Q matching to improve gas exchange at a safer FIO_2. This position may also have improved alveolar, interstitial and microvascular pressure relationships enhancing alveolar recruitment with higher levels of PEEP.

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