Observations on IRV

To the Editor:

In the February, 1986, issue of Chest, Dr. Gurevitch et al attribute improvement in two patients with ARDS to reverse I/E or inverse ratio ventilation (IRV) after changing the mode of ventilation from IMV with one ventilator to IRV with another ventilator (1986; 89:211-13). At the same time they paralyzed the patients and sedated them with diazepam and morphine. Thus, at least four significant factors were changed concurrently, and only one of them was given credit for improvement.

Their conclusion, "that IRV leads to improved oxygenation with decreased peak airway pressure," is not at all supported by the way they managed the mechanical ventilation; paralysis alone certainly can lower peak airway pressures with mechanical ventilators. They state that "our data support . . . that it (mean airway pressure) . . . is not the major determinant of oxygenation," yet they did not report mean airway pressure in either patient. They quote a PEEP vs CPAP study in ketamine-anesthetized pigs in which respiratory pressures are likely to be significantly different than those in a patient whose pressures depended "on the patient's level of agitation." There was only one period of IMV with paralysis and morphine in either patient; "a premature switch back to IMV" on the ninth day of the first case hardly qualifies as any sort of control. In fact, there does not appear to have been an attempt, before the IRV, to optimize ventilatory support with "conventional" IMV.

After instituting the new regimen, "the (second) patient's metabolic abnormalities were corrected"; another variable altered after starting IRV. Along with paralysis, "anesthesia" was continued for 14 and five days respectively for the two patients; was this morphine? Does morphine administration equal anesthesia? IRV may prove to be a promising addition to ventilator care, but gee-whiz look at this case studies do nothing to advance the scientific approach we expect of Chest.


To the Editor:

Dr. Misuraca summarizes several important criticisms with regard to our report of two patients with severe ARDS treated with inverse ratio ventilation, and he asks many of the same questions that we are currently attempting to answer by means of an ongoing prospective evaluation of this modality.

Certainly, ventilator dependant patients who are sedated and paralyzed have lower peak airway pressures (PAP), and the fact that this was imposed upon both these patients at the time of starting IRV is an important shortfall. We cannot, however, ignore the fact that when restarting conventional ventilation in the first patient (day 9), or 20 cm H2O increase in PAP occurred despite continued sedation with paralysis and at a time when the patient appeared to be clinically improved. Therefore, even if the patient's initial drop in PAP from 70 to 50 cm H2O was due to sedation and paralysis, another 20 cm H2O drop from 50 to 30 cm H2O was likely due to the modality. This trend has been consistent in subsequent cases we have studied. Whether switching ventilators alone would result in the magnitude of change we report (as implied by Dr. Misuraca) is probably unlikely as, again on day 9, the first patient's pressure changes occurred on the same ventilator.

The relationship of mean airway pressure (MAP) to oxygenation is a difficult one to understand. Although there is a trend to increased PaO2 with increased MAP it clearly is not consistent. MAPs were not reported due to the constraints imposed on the reporting of cases; however, in the first patient, days 3 to 6 MAP ranged from 20 to 24 cm H2O (I/E 1.5 to 2.1), yet while at a ratio of 4:1 (days 7 to 9) MAPs ranged from 27 to 30 cm H2O. Except for an initial jump in PaO2 when changing modes, the majority of PaO2 levels recorded during these widely different MAPs were quite similar. We continue to observe this trend and note that while "clamping" MAP (as can be done with a pressure-controlled, time cycled ventilator), changing the rate, I/E ratio and PEEP can independently alter PaO2. The mechanisms are not clear. The CPAP vs PEEP study by Bowe et al., to which Dr. Misuraca makes reference, supports the lack of correlation between PaO2 and MAP and, in many ways, is very similar to our paralizedsedated patients as the "level of agitation" plays no role in our data once the patients were started on IRV therapy.

In the second patient, correction of the metabolic abnormalities does introduce an added variable; however, the rapid improvement in oxygenation after starting IRV therapy (within 12 hrs) occurred well before significant metabolic improvements, which lagged by 48 to 72 hrs.

Observations described in these cases were not reported casually, and our impetus to present them stems from the fact that these same trends have been observed in our own subsequent patients and in those of others. A formal prospective evaluation is underway, as it is fully appreciated that case reports in and of themselves prove nothing. In reviewing the history of science, and of medicine in particular, it can be noted that many important discoveries were based only on initial observations/correlations; more formal investigation may not have occurred until some time later. Dr. Misuraca's reprimand of Chest is unjustified as we feel that, by providing a forum for the presentation of interesting correlations and observations, the journal serves an important educational service. The controversy, scientific thought and curiosity which are generated result in a more detailed and thorough investigation which ultimately does result in the advancement of science.

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