communications to the editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

PEEP Titration Using the PaCO₂-PetCO₂ Gradient
Fact or Fallacy?
To the Editor:

In a recent report (Chest 1984, 85:100-04), Murray et al and an accompanying editorial describe use of the difference between endtidal and arterial carbon dioxide tension as a useful way to titrate PEEP. This is a view which has been previously expressed and one with which I concur. However, their study purporting to demonstrate this raises several questions which require response before proceeding from laboratory to bedside with this technique.

First, since the main purpose of their publication is to demonstrate the value of the PaCO₂-PetCO₂ gradient in adjusting PEEP levels, it seems necessary for the authors to show that PEEP significantly affects the gradient in the oleic acid damaged lung. In their study, did the gradient change significantly from its peak value after PEEP was applied? If not, I question whether the authors have shown that their technique is useful. Second, if the PaCO₂-PetCO₂ gradient is minimal at very high peak inflation pressures, what considerations should guide the clinician in deciding whether to choose lower inflation pressures instead of the optimal gradient so that risks of pneumothorax may be lessened? What inflation pressures were seen in the study as PEEP was varied? Finally, I question whether this study was adequately controlled.

In order for the authors’ conclusions to be substantiated by their findings, I must assume that pulmonary and hemodynamic stability were maintained from 90 to 165 minutes after oleic acid infusion. If not, many of the changes which were observed may have been due to time-related deterioration of pulmonary function and not due to PEEP as they supposed. The authors claim stability, citing an abstract of a previous study, presumably from the same laboratory several years ago. Yet, they made no comparisons with any of the data from that abstract. In fact, the present and the past groups are scarcely comparable with a twofold difference in calculated intrapulmonary shunt. No mention is made about similarities or difference in anesthetic management fluid maintenance or size of dogs in the previous study. Hence, it is hard to know whether stability implied by a few measurements in an earlier study implies stability in this later effort.

In conclusion, though Murray et al recommend their technique on reasonably sound physiologic principles, I do not believe that their conclusion is warranted without inclusion of additional data to substantiate their claims.

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REFERENCES

To the Editor:

We are pleased that Dr. Geer agrees that the arterial minus end-tidal carbon dioxide tension gradient (PaCO₂-PetCO₂) is a useful way to titrate PEEP. We appreciate his taking time to comment on our study.

The first question posed in his letter is, “In their study, did the gradient change significantly from its peak value before PEEP was applied? We refer Dr. Geer to Figure 1 of our article where we showed that the PaCO₂-PetCO₂ increased from a pre-oleic acid level (control) of 7.3 ± 2.3 mm Hg to 18.3 ± 8 mm Hg after oleic acid injection (p < .05). With the institution of 10 to 20 mm Hg of PEEP, the PaCO₂-PetCO₂ gradient returned to a level that did not differ significantly from control but did differ significantly from the value attained after oleic acid injection without PEEP (p < .05). As PEEP was increased further to 25 mm Hg, the PaCO₂-PetCO₂ increased (p < .05), which suggests an adverse effect of PEEP at this level.

Dr. Geer also asked what considerations should guide the clinician in deciding whether to choose a lower inflation pressure instead of the optimal gradient so that risks of pneumothorax may be lessened. The PaCO₂-PetCO₂ reflects the effect of PEEP on dead space and is not directly related to peak inflation pressures, which are secondary to the determinants of mechanical ventilatory support and were not studied specifically in our report.

We believe that the PaCO₂-PetCO₂ can be very helpful in permitting the clinician to seek a lower level of PEEP when the shunt remains fairly constant, regardless of the amount of PEEP applied. A small PaCO₂-PetCO₂ gradient may indicate the ability to lower PEEP and if a significant increase in gradient is not seen, this will permit the patient to receive ventilatory support at a decreased inflation pressure.

Finally, Dr. Geer questioned whether this study was adequately controlled. He suggests that we compare our data with those from an earlier study performed by two of us (TJJG and MJB). Dr. Geer is specifically concerned about the fact that these two studies report almost a twofold difference in calculated intrapulmonary shunt. It is important to point out that the control pre-oleic acid shunt in that earlier series was 12.3 ± 4.7 percent, whereas the control shunt in our latter group of ten animals was 2.3 ± 1.6 percent. Therefore, we would expect a much larger absolute change in shunt to occur after oleic acid injection in those dogs that had a higher shunt to begin with. The earlier study that he refers to did indicate that a stable intrapulmonary shunt was maintained between 90 and 180 minutes after the administration of oleic acid. Dr. Geer also questions whether the anesthetic management, fluid maintenance, and size of the dogs in the two studies are comparable. These aspects are comparable between the two studies. It is our belief that the models are comparable and, therefore, we feel that the experiment is properly controlled and is valid.

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