produce lower ratios (Trimble et al., 1971). They also adjusted ventilation by changing frequency, whereas all our patients were ventilated at the same frequency. As a last comment on this point, we do not feel that they have given enough consideration to the effects of the prone position in which their patients were studied. Little is known about the effects of this particular position during anaesthesia, but regional distribution of ventilation differs considerably between supine and prone positions in conscious subjects (Cortese et al., 1976). Their conclusions can only be relevant to the prone position.

With regard to any possible change in Vd/Vt on induction of hypotension, we would accept that their policy of fluid loading would prevent any alteration resulting from changes in cardiac output or, perhaps more relevantly, pulmonary artery pressure. However, they do not consider the direct relaxant effect of sodium nitroprusside on tracheobronchial muscle (Kreye et al., 1975), which would lead to an increase in Vd/Vt, assuming that some bronchial tone were present.

Sir,—We would like to thank Drs Wildsmith, Drummond and MacRae for their comments and interest shown in our work. Many investigators in the past, from Eckenhoff and his associates (1963) to Wildsmith, Drummond and MacRae (1975), have concluded that an increase in pulmonary deadspace occurs with deliberate hypotension using a whole gamut of agents from ganglionic blocking drugs to direct acting vasodilators. However, we have demonstrated that deliberate hypotension, per se, does not necessarily cause an increase in pulmonary deadspace (Khambatta, Stone and Matteo, 1982).

We believe that the apparent discrepancy between our results and those of others has a basis in cardiopulmonary pathophysiology. It has been repeatedly shown that, whenever cardiac output and pulmonary artery pressure decrease, pulmonary deadspace increases. In our study, these were maintained by adequate hydration. Thus, we conclude that the maintenance of cardiac output and pulmonary artery pressure during induced hypotension will prevent the increase in pulmonary deadspace.

REFERENCES


