fusion (V/Q) ratio in the ventilated lung would almost double. Relationship of alveolar ventilation and $PA_{CO_2}$ is $\dot{V}_A = \frac{\dot{V}_{CO_2}}{PA_{CO_2}} \times (PB - 47)$. At a constant $CO_2$ production, the equation can be simplified as $V_A = K/PA_{CO_2}$.[2] Thus alveolar $P_{CO_2}$ and alveolar ventilation have an inverse relationship. Doubling the alveolar ventilation of one lung, as would happen with endobronchial intubation at least initially, will lead to a marked decrease in alveolar $P_{CO_2}$ in the ventilated lung, increase in $PA_{CO_2}$ in the nonventilated lung with a significant drop in arterial $P_{O_2}$, and minimal increase in $PA_{CO_2}$.[3] As mass spectrometry during accidental endobronchial intubation measures the alveolar $CO_2$ from the ventilated lung only, initially, it would show a marked *fall* in end-tidal $CO_2$.

What the authors have reported is a case of hypoventilation of a ventilated lung. Their suggestion that an acute elevation of $PET_{CO_2}$ may result from an increase in dead space is not true. The immediate effect of increased physiologic dead space by endobronchial intubation, would be to lower the alveolar and expired $CO_2$ in that lung.[4]

Could reduced tidal volume to the ventilated lung be due to partial obstruction in an uncuffed tube? There is no mention in the report whether the authors used a volume-cycled or a pressure-cycled ventilator and what expired tidal volume was during one-lung ventilation. A use of a pressure-cycled ventilator with partial obstruction in an uncuffed tube could explain the reduced tidal volume in a ventilated lung and increased end-tidal $CO_2$.

We would like to emphasize that while using an end-tidal $CO_2$ monitor, an early warning sign of an accidental endobronchial intubation is a sudden drop in $PA_{CO_2}$* and not a sudden rise in $PA_{CO_2}$.

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*In reply:*—We agree that the first explanation offered in our report was an unlikely possibility. That is, unimpeded ventilation of one lung is generally associated with an initial fall in $PE_{CO_2}$ (this may be missed when using a time-shared spectrometry system), a minimal rise in $Pa_{CO_2}$, and markedly increased airway pressure.

The incident we reported describes a case of hypoventilation of an intubated lung. We postulated that the position of the endotracheal tube in relation to the bronchial wall of the ventilated lung was such that resistance of the intubated lung was markedly greater than that of the alternative pathways for flow; namely, the upper airway, contralateral lung, and pressure-limiting valve of the ventilator. Because breath sounds could not be heard over the contralateral lung, we assumed gas was escaping to the atmosphere around the endotracheal tube in sufficient quantity to result in hypoventilation. However, since expired tidal volume was not measured during the course of the anesthetic, this explanation must remain speculative.

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