Alveolar deadspace for anesthetic agents

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Video Abstract

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Abstract

When patients undergo general anesthesia, there's a shift in the distribution of ventilation and perfusion throughout the lung, with more areas of the lung getting too much air, relative to the amount of blood flow, and others getting too little. This type of scatter is traditionally described by Riley's three-compartment model, in which high-ratio lung regions getting less blood flow produce increases in the alveolar deadspace. But new work published in the journal *Anesthesiology* shows that this model fails to account for different blood solubilities of various anesthetics – and shows how multicompartment models better predict what is happening in the lungs. The researchers extended an earlier study in anesthetized patients that found that partial pressure measurements of inhaled anesthetic in the lungs did not match those made for carbon dioxide – and were inconsistent with the three-compartment theory. Those authors hypothesized that this could be due to the lack of inclusion of diffusion gradients, which would be important for heavy anesthetic molecules. But that explanation overlooked what multicompartment models of lung gas exchange could tell us, which predict that the alveolar-arterial gradients and alveolar deadspace would be different for gases with different blood solubilities. The researchers hypothesized that solubility could be the key factor, and therefore measured partial pressures in 52 patients undergoing cardiac surgery with four different inhaled anesthetics. They found that the alveolar deadspace fraction was inversely related to the anesthetic's blood solubility – with deadspace being significantly larger for each volatile agent than for carbon dioxide. For all agents other than desflurane, there was a significant difference between the ideal alveolar partial pressure and the end capillary partial pressure they calculated from the Riley model, which should be identical. The 50-compartment model showed how the predicted alveolar capillary gas uptake distributions for the agents shifted toward lower ventilation perfusion ratios – with increased deadspace – as anesthetic solubility declined. Interestingly, while this more physiological multicompartment modeling could predict deadspace for the different anesthetics fairly well, it consistently underestimated the values. This suggests that a residual factor other than solubility, such as diffusion limitation, may also contribute to deadspace after all. The results indicate that because anesthetics are less soluble than carbon dioxide, the pulmonary alveolar deadspace fraction for anesthetics is much larger than when calculated from arterial to end-tidal carbon dioxide partial pressure gradients. The authors therefore conclude that the conventional practice of calculating a definitive alveolar deadspace using carbon dioxide measurements is misleading in predicting the uptake of anaesthetic gases in the lung. Further research is needed to identify the potential contribution of diffusion limitation.