HISTORIC AND FUTURE DEVELOPMENT OF HIGH-FREQUENCY VENTILATION

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High-frequency ventilation (HFV) has not one but several histories. Proceeding along largely independent pathways are techniques using frequencies of 1–5 Hz and techniques using 10–40 Hz, the former being introduced by Sjostrand in 1971, the latter by Lunkenheimer in 1972. It was nearly ten years before it was recognized that these techniques must radically alter our concepts of gas transport within the lung. There has also been an unfortunate series of clinical studies purporting to show that HFV is superior to conventional ventilation in patients with lung disease. There is no doubt that nearly all reports show that HFV controls PaCO₂ very easily, and that the mechanisms by which this is achieved is the primary topic of the symposium. What has not been proved is that HFV is better than conventional ventilation in increasing PaO₂. The mechanisms of oxygen exchange when the lung has extensive shunts are quite different from those for CO₂ exchange, and this problem has not been rigorously addressed.

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INTRODUCTION TO HIGH-FREQUENCY VENTILATION

The equation

\[ V_A = (V_T - V_D)f \]  

where
- \( V_A \) = alveolar ventilation
- \( V_T \) = tidal volume,
- \( V_D \) = volume of the dead space,
- \( f \) = respiratory frequency

is embedded in textbooks of physiology and serves well enough when a reasonable \( V_T \) and \( f \) are employed. But the chimerical nature of the “dead space” has been recognized for many years. Henderson et al. (4) in 1915 speculated that “there may easily be gaseous exchange sufficient to support life, even when the tidal volume is considerably less than the dead space.” He supported this with an admirably simple experiment. He blew tobacco smoke...
down a tube and found that it formed a long thin spike and that "the quicker the puff, the thinner and sharper the spike." When the puff was stopped, "the spike breaks instantly, everywhere, and the tube is seen to be filled from side to side with a mixture of smoke and air." This phenomenon was more rigorously investigated by Briscoe and his colleagues (2) who showed, in man, that inspired volumes less than the dead space volume could reach the alveolus. They speculated that this might explain why "some patients can live despite the fact that they are breathing very small tidal volumes." Thus the dead space is not a true volume, it is a volume of convenience.

The first convincing confirmation of these predictions was a remarkable paper by Lunkenheimer et al. (6), which is another example of serendipity in science. They were oscillating the airway with a loudspeaker to estimate mediastinal impedance. To make their measurements they had to paralyze the animals and, presumably to their astonishment, there was no rise in arterial CO₂. Perhaps these experiments were not taken as seriously as they might have been, as although the dogs maintained excellent gas exchange, they developed a progressive metabolic acidosis, oliguria, and falling blood pressure. These rather discouraging findings, in retrospect, had nothing to do with the high-frequency ventilation (HFV) and were almost certainly due to the use of a high mean airway pressure, impeding cardiac output.

Our own group also backed into this field by accident. We were interested in cardiogenic mixing and were astonished to find, in normal subjects, that a loudspeaker connected to the airway "shook" CO₂ out of the mouth during breathholding. Unfortunately, the results were not reproducible. We did not appreciate that it is difficult to breathhold with the glottis open for any length of time. When we eventually bypassed the glottis with an endotracheal tube, we reproduced Lunkenheimer's results and, using low mean airway pressures, never saw a metabolic acidosis or a fall in cardiac output. We incorrectly concluded that 15 Hz was the "optimum frequency," but this was in fact a circuit artifact (1). Oscillations do not need to be applied at the airway opening; Zidulka et al. (7) showed that high-frequency chest wall compression is capable of "shaking" CO₂ out of normal dogs. This may aid in the elimination of CO₂ without the need for intubation. However, its efficacy will be a function of chest wall compliance, which is lower in man than in the dog. Further, as the net pressure of the lung is positive, one can predict difficulties with oxygenation.

While we were doing this work we were fortunate enough to have the collaboration of Jeff Fredberg, and he subsequently published a landmark paper (3), which was the first systematic attempt to explain how HFV might work. Variations and alternatives have subsequently appeared, but it is not my function to review them, as this is the task of the other speakers at this symposium.

I have perhaps a more difficult task: attempting to predict the future of HFV, and that future depends on whether HFV can achieve good oxygen exchange in a lung with diffuse disease. It has sometimes been naively assumed that if HFV can "shake" CO₂ out, it will "shake" O₂ in. This is true in the normal lung and in moderately diseased lungs. But in diffuse lung disease with hypoxia such as Infant Respiratory Distress Syndrome (IRDS) or Adult