Introduction: Despite increasing clinical interest in the use of high frequency ventilation (HFV) and general agreement that this ventilatory mode is at least as effective in promoting expiratory gas exchange as conventional techniques, there is a noticeable lack of experimental data bearing on its physiological properties. One of the reasons for this lack of data has probably been the problems involved in the accurate measurement of respiratory gas flows and concentrations in an animal which is being ventilated at rates of 1 Hz (60 bpm). Using a specially designed ventilator and the experimental setup shown in Fig. 1, we have carried out a series of studies in rabbits with the aim of investigating the physiology of HFV in this species.

Methods: Adult New Zealand White rabbits weighing between 3.0 and 4.5 kg were anesthetized by means of IM ketamine and promazine and prepared for study with the insertion ofuffed endotracheal tubes and arterial and venous cannulae. Baseline measurements of arterial PO2, PCO2, & pH and of mixed expired CO2 concentration (PFeCO2) and minute ventilation (VE) were made during spontaneous breathing. The rabbits were then attached to the ventilator and given IV pancuronium to prevent further spontaneous respiratory attempts. Supplemental doses of the anesthetic agents and the relaxant were given as required during the course of the experiment. Blood gas determinations and measurements of VE and PFeCO2 were repeated at different ventilatory frequencies after adjustment of VE to produce steady-state PAO2s within the normal range. The results of these determinations were used to calculate VD/VT at each frequency. At the end of each experiment the relaxant was reversed and the animal was allowed to recover. Further arterial blood gas determinations and measurements of PFeCO2 and VE were made with spontaneous ventilation during the recovery period to confirm that the measured respiratory parameters returned to control values.

In subsequent series of experiments:
1) Nitrogen washouts were performed at various frequencies after the achievement of CO2 equilibrium in order to determine end-expiratory lung volume.
2) The effect of changes in the mean airway pressure on the relationship between VD/VT and frequency was investigated.

Results:
1) With our experimental system, physiological dead space is virtually independent of ventilatory frequency in the range of 1 to 20 Hz (Fig. 2) and remains within a range (1.5 - 2.5 mL/kg) consistent with the assumption that it is accounted for almost entirely by the anatomical dead space.
2) At constant PAO2, PAO2 increases with increasing frequency (from a mean of 67 mm Hg at 1 Hz to a mean of 84 mm Hg at 10 - 14 Hz (7 studies in 4 rabbits, ventilated with air).
3) Lung volume at end-expiration increases with increasing frequency, suggesting that PEEP is present at the alveolar level, as predicted by our mathematical model (1).
4) Lowering the mean airway pressure to zero does not materially affect the constancy of physiological dead space with varying frequency.
5) Animals studied on more than one occasion show no apparent cumulative or long-term effects on pulmonary function.

Discussion: These studies suggest that HFV need not be accompanied by any dramatic change in the behavior of the respiratory system, at least from the viewpoint of CO2 exchange. The frequency-related improvement in PaO2 which was noted has been previously reported and may be associated with changes in FRC resulting from "alveolar PEEP". We cannot confirm the frequency-dependent fall in physiological dead space reported by others (2), even when mean airway pressure is reduced to simulate the to-and-fro ventilation which some have used.

References:

Fig. 1

Fig. 2