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NEUROPHYSIOLOGICAL MECHANISMS INDUCING APNOEA DURING HFO

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The occurrence of apnoea during high-frequency oscillatory ventilation (HFO) depends on the instant ratio of all inhibitory and excitatory mechanisms which are involved in the control of breathing during HFO. We attempted to define HFO conditions leading to apnoea and to identify the underlying neurophysiological mechanisms.

In rabbits anaesthetized with urethane we applied short periods (10–15 s) of HFO, the HFO parameters (pump frequency, stroke volume, mean airway pressure, amplitude of pressure changes) being varied systematically. We analysed the ventilatory responses to HFO in intact rabbits, in rabbits having both vagal nerves cooled gradually from 37 °C to 0 °C, and in vagotomized rabbits. In our experimental procedure, neither the mean airway P_{CO_2} nor arterial P_{CO_2} were changed during HFO and therefore were not involved in the responses observed.

As all effects of HFO on spontaneous breathing were abolished both by bilateral vagotomy and by vagal cooling to less than 5 °C, we assume that extravagal pathways did not play a role in our experimental conditions. Depending on the HFO variables, three types of ventilatory response to HFO were observed: decrease in breathing frequency (mostly at low pump frequencies and low stroke volumes); increase in breathing frequency (at high pump frequencies and high pressure amplitudes; its incidence was increased by cooling the vagi to about 10 °C); apnoea accompanied by a tonic diaphragmatic activity (incidence increased with increasing mean airway pressure and pressure amplitude, the diaphragmatic activity being reinforced by cooling the vagi to less than 20 °C).

In one part of the experiments we analysed the changes in activity in single afferent vagal fibres during HFO. The majority of pulmonary slowly adapting stretch receptor (PSR) fibres increased the discharge rates in proportion to both airway pressure and pump frequency. Rapidly adapting stretch receptors (RAR) were generally less affected by HFO; the most frequent response was an increase in activity, but a decrease in discharge rate was observed also. The majority of RAR reached the maximum discharge rate at mean airway pressure zero and pump frequencies between 15 Hz and 20 Hz.

We conclude that, in the experimental conditions described, apnoea during HFO is mediated solely by vagal nerves; that apnoea during HFO is caused mainly by stimulation of PSR, whereas the accompanying tonic diaphragmatic activity results from stimulation of RAR; and that, depending on the combination of HFO parameters used, different groups of vagal receptors are stimulated, giving rise to different ventilatory responses to HFO.

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