Editorial: Airway pressure and xenon anaesthesia

Since the rediscovery of xenon as an anaesthetic in the 1990's, there has been some concern about the effects of xenon's high density and viscosity as compared to air, oxygen and nitrous oxide. Several investigators observed higher driving pressures at their ventilators, necessary to generate standard ventilation patterns (1). In animal studies it was shown that the physical properties of xenon indeed could explain these elevated pressures and that the greatest pressure loss within the system occurred over the endotracheal tube (2,3). Katz and colleagues present a combined study of simulation and experimental measurements to demonstrate the effects of ventilation with xenon-oxygen mixtures on external airway and intrapulmonary pressure distribution under adult human conditions. They have developed an interesting model and – again – clearly demonstrate that the most important pressure drop is found over the external tubing, including the endotracheal tube. Within the lung, xenon-containing gas will most likely not result in significantly higher pressures than any air-oxygen mixtures. Although their modelling is very sophisticated, there may be an easier way to look at the problem: When substituting the law of Hagen and Poiseuille for laminar flow with some of the human airway dimensions as provided by Nunn's Applied Respiratory Physiology (7^{th} edition, 2010 (4)), it becomes evident that already at the level of the small bronchi (generation 12) gas density becomes negligible for pressure loss:

$$P_1 - P_2 = (64/Re) \times (l/d) \times (\rho/2) \times v^2,$$

with Re as the Reynolds number, l as length and d as diameter, ρ as density, v as the mean velocity. When d is increased from 18 mm (trachea) to about 2000 mm (2000 small bronchi of 1 mm diameter each), the pressure difference becomes less than 1/400 because v^2 will decrease to about 1/10, l to 1/40 (3 mm small bronchi length as opposed to 120 mm trachea length), and Re to around 1/100, according to data from Lumb and Tsuda (4,5). Thus, the denominator of the equation will not change while the numerator is decreased by a factor of about 400. It is obvious that this change will virtually eliminate the influence of density.

Although Re will be up to 4 times higher for a high xenon concentration as compared to air, as it also depends on density, this will not be important for pressure distribution, with Re in a range of less than 1 in small bronchi. Accordingly, a hypothetical pressure loss across the trachea of as high as 30 hPa would translate to less than 0.1 in the small bronchi, regardless of the gas mixture insufflated. Keeping this in mind, it is not surprising that the simulation yields a pressure of less than 1 hPa from bronchial generation 5 on, regardless of flow pattern and xenon concentration, as demonstrated in figures 1 to 3 of the Katz paper.

The authors probably correctly state that there is really no more pressure difference from generation 15 on. This means no pressure-driven mass flow but only convection and diffusion which depend on viscosity and diffusion properties. As xenon's kinematic viscosity is less than 30% of that of air and oxygen (which are almost identical), and its diffusion coefficient in air is around 40% of that of oxygen, a difference in mass transport between xenon-containing gas and oxygen in air is likely to occur within the terminal airways. However, at that point the picture becomes very complex and the forces and patterns directing flow are not completely understood yet (5-7). In any case, pressure obviously does not determine mass transport in the small airways anymore, and thus pressure distribution is only relevant within larger airways.

Much more than inspiration, it would have been interesting to model expiration ac-

cordingly because this is where a difference between a high-density gas and air/oxygen may become clinically relevant: as the authors show in their figure 7C, the pressure – which is probably distributed uniformly throughout the lung at the beginning of expiration – would be about 6 instead of 3 hPa when a xenon-containing mixture is used. Still, it would be interesting if uniformity of pressure distribution will hold true also for xenon as the authors suggest in their figure. In any case, a pressure of 6 hPa is in the range of therapeutically administered PEEP and could be causing gas trapping and eventually over-insufflation due to extended expiration time. On the other hand, just like deliberately applied PEEP this may have favourable effects on alveolar gas exchange by preventing airway closure during anaesthesia.

Moreover, gas distribution in the pulmonary acinus which does not depend on pressure as stated above, may be influenced by adding a compound with different diffusivity and viscosity by changing the small local concentration gradients driving gas exchange. Another noble gas which has in fact been investigated extensively in this regard is helium, with much lower density and intermediate diffusivity when compared to xenon and air. However the clinical effects aside from lowered large airway pressure in high-frequency ventilation appear quite limited (8,9).

References

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