Circulatory effects of fast ventilator rates in preterm infants

Sir,—Fenton et al have recently described the circulatory effects of fast ventilator rates in critically ill neonates.1 These authors found a decrease in arterial oxygen tension (PaO2) with an increase in ventilator rate and have concluded that this fall in PaO2 was a result of the predicted theoretical fall in mean airway pressure with increasing rate. Mean airway pressure can be calculated from the following formula:

$$\text{IT} = \text{PEEP} + \text{ET} + \text{IT}$$

(where IT=inspiratory time, ET=expiratory time, PEEP=positive end expiratory pressure). It can also be directly measured through a transducer and should essentially reflect the area under the ventilator pressure curve. As can be seen from the above formula, if the inspiratory-expiratory ratios are kept constant and there is no significant change in peak inspiratory pressure or PEEP, then mean airway pressure should remain constant as rate is changed. A critical determinant of whether mean airway pressure will in fact change under these circumstances will be the shape of the ventilator pressure curve, which will be affected by the lung compliance and the gas flow to the ventilator.

We have tested this hypothesis by subjecting an Infant Star (Infrasomatic) and a Scherist Model IV-100B (Scherist Industries) to the changes that were made by Fenton et al for rates of 30 (IT=ET=1), 60 (IT=ET=0.5), and 100 breaths/minute (bpm) (IT=ET=0.3) with a constant inspiratory (20 cm H2O) and expiratory pressure (5 cm H2O).

We measured mean airway pressures using an independent transducer and both ventilators were connected to a test lung with a compliance of 1 ml/cm H2O. There was no change in mean airway pressure for the Infant Star with the ventilator flow varied between 8 and 18 l/min. For the Scherist, the same model used by Fenton et al, the mean airway pressure at 30 and 60 was 12 cm H2O and at 100 bpm was 11 cm H2O at a flow of 10 l/min. If the flow at 100 bpm was increased to 15 l/min, then the mean airway pressure was also 12 cm H2O. Our results also confirm the observations of Greenough and Greennell.2

We believe that the fall in oxygenation associated with the higher ventilatory rates may reflect an inadequate period of time above the opening pressure of the terminal airways in infants with significant respiratory disease, as opposed to any significant change in mean airway pressure.

We believe that the fall in oxygen tensions observed in the current study reflect mechanisms other than those suggested by the authors.

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Antiviral and antibacterial lipids in human milk and infant formula

Sir,—We read with great interest the recent paper by Isaacs et al in which they conclude that the antiviral and antibacterial activities of lipid extracts from gastric aspires in infants probably result from intragastric lipolysis by lingual lipase to monoglycerides and fatty acids from the triglycerides of the original feeds.1 Lingual lipases hydrolyse medium and short chain triglycerides at a higher rate than longer chain fats.2 In the study by Isaacs et al, at least 50% of the fat content of both formulas was medium chain triglyceride if the additional coconut oil in the infant formula was removed. Analysis of milk fat by lingual lipase produces a large amount of monolauryl glycerol, which has been shown to have appreciable antibacterial and antifungal activities.3

We have recently shown a similar effect in mice, where by modifying the mother’s dietary fat intake we were able to change the fatty acid composition of their milk. The offspring were inoculated orally with EEMD rotavirus. In those young mice fed on milk naturally enriched with lauric acid (C12) and other medium chain fatty acids, the onset of diarrhoea was delayed and the stool output of rotavirus was appreciably reduced by comparison with mice fed milk on different fatty acid composition (unpublished observations).

At least in mice, therefore, and contrary to the assertion of Isaacs et al, the protective effects of milk fatty acids appear to operate against non-enveloped viruses as well as enveloped viruses. Therefore, of course, rule out the possibility that other mechanisms such as secondary variation in trypsin secretion could be responsible for our observations,3 but we think that this is inherently unlikely.

It is becoming increasingly clear that milk fat, whether human or of animal origin, has important protective value against intestinal infection. Skimmed milk should not be used as a food source for young children unless there is a strong medical reason for the withholding of fat, and this policy should be followed particularly in countries with a high incidence and severity of gastroenteritis.

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3 Jessen RG, Clark RM, Dejong FA, et al. The lipolytic triad: human lingual, breast milk and pancreatic lipases: physiological implications of...