LETTERS TO THE EDITOR

Improvement of respiratory failure with NIV

In their recent paper Nickol et al studied the possible mechanisms by which non-invasive ventilation (NIV) improves ventilatory failure in patients with a restrictive defect due to either neuromuscular disease or kyphoscoliosis. They investigated three possible hypotheses for reduction in daytime hypercapnia—namely, increased ventilatory sensitivity to CO₂, improved respiratory muscle function, and increased respiratory system compliance. They showed that the reduction in diurnal PaCO₂ after treatment was accompanied by an increase in hypercapnic ventilatory response (HCVR), with no changes in non-volitional tests of respiratory muscle strength or respiratory mechanics. They conclude that an increased ventilatory response to CO₂ is the principal mechanism underlying the long term improvement in gas exchange associated with NIV.

Interpretation of HCVR in patients with lung disease is often difficult and, as the authors point out, the measurement is highly variable. In attempting to minimise this variability they report the mean of two tests, but the finding of no significant difference between the first and second test is insufficient evidence to assess repeatability. Furthermore, acknowledging that an association between HCVR and PaCO₂ has been demonstrated, there is a danger of over-interpreting this as cause and effect (increased HCVR resulting in lower PaCO₂), and I would suggest that reverse causality (lower PaCO₂ resulting in higher HCVR) is at least equally (and probably more) likely.

Studies over many years 1,2 have shown that the ventilatory response to CO₂ is dependent on the prevailing PaO₂ and bicarbonate concentration. The law of mass action dictates that, in patients with chronic hypercapnia and raised bicarbonate and CSF bicarbonate levels, a given change in PaCO₂ during stimulated breathing will result in a smaller than normal increase in hydrogen ion concentration (the fundamental stimulus to the respiratory centres) and consequently a smaller increase in ventilation. When a chronically raised PaCO₂ is lowered (as occurs with NIV), the bicarbonate concentration also falls (as clearly shown in this study) and an increase in the ventilatory response to CO₂ would be expected.

I therefore question the conclusion of Nickol et al that the ventilatory control mechanism is “fundamental” in determining the improvement in ventilatory failure accompanying NIV. As they explain, gas exchange improves as a result of optimising the “load/capacity/drive balance of the respiratory system” but, in my view, the “drive” is likely to be of secondary importance. The authors produce good evidence, as have others, that changes in load are probably not relevant. As they acknowledge, however, they have examined only one aspect of “drive”—that is, by relieving the load for several hours per day, some aspect of respiratory muscle function is improved, allowing PaCO₂ to be maintained closer to normal for the remainder of the 24 hour period. Whether this improvement relates to better endurance, less fatigue, or an aspect of strength which is incompletely assessed by the tests used remains to be determined. I submit that, when considering the mechanisms of improved gas exchange with NIV, the focus should remain on the respiratory muscles rather than on the ventilatory control mechanism.

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References

Consideration of palivizumab not justified

Broughton and colleagues’ state that consideration should be given to the use of prophylactic palivizumab to infants born at less than 32 weeks in the case of maternal smoking or even if they have siblings. However, the authors present no data from their own or other studies to indicate that this would be in any way cost effective or justified. Certainly the word “consider” is fortunate, given the stated funding provided to one author by the manufacturer.

The study demonstrated a relationship between lower respiratory morbidity from respiratory syncytial virus (RSV) and smoking which has been widely shown elsewhere. The numbers of smokers were in fact very small—surprisingly so at 18 per 126 babies, given both their prematurity and the catchment population for this hospital, although 28 experienced smoking in the home. One wonders whether N1 non-consenters and non-attenders may have comprised a higher proportion.
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