Response to: Bacterial co-infection and the interpretation of immunological data from BAL fluid specimens in severe RSV bronchiolitis (Thorax 2006;61:1098)

Eisenhut expresses a valid consideration that pertains to many of the studies investigating inflammatory mediators in respiratory syncytial virus (RSV) bronchiolitis. Many RSV immunopathogenesis studies presume a pure RSV aetiology. Co-infection (viral and/or bacterial) in severe bronchiolitis is not uncommon.

A phagocytic (neutrophils and macrophages) and natural killer cell response is common to both viral and bacterial lower respiratory infections.

Analysis of the blood differential leucocyte counts in our study group failed to discriminate between the RSV-only and bacterial co-infection groups (Mann–Whitney–Wilcoxon test: neutrophils p = 0.2; lymphocytes p = 0.2; monocytes p = 0.9). Did the C-reactive protein (p = 0.9)? However, we only evaluated total number of leucocytes/white cells, bacterial growth density and microorganism species in the broncho-alveolar lavages, and not white cell type. Therefore we are unable to comment on potential differences in concentrations of lower respiratory tract neutrophils and alveolar macrophages between the groups.

The interaction between the phagocytic effectors cells, the pathogen and endogenous immune molecules orchestrate the inflammatory response. Cytokine and chemokine reactions can therefore be customised by the triggering pathway(s). Consequently it is vital to identify the correct perpetrator when assigning responsibility for these inflammatory effects.

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References

Assessing fitness to fly in young infants and children

Commercial aircrafts cruise between 9150 and 13000 m above sea level, with a cabin pressure equivalent to 1530–2440 m at which passengers breathe the equivalent of 15–17% of fractional inspired oxygen (FiO2) at sea level. British Thoracic Society recommendations for passengers with chronic respiratory disorders planning air travel suggest that infants and young children unable to perform spirometry, with a history of neonatal respiratory disease consult a paediatrician and a hypoxia test be considered. The recommended hypoxia test method in young children is to place the child in a body plethysmograph while seated on the parent’s lap and introduce nitrogen until FiO2 equals 13%. This method relies on equipment not readily available outside the tertiary hospital setting. We have reviewed a report of hypoxia testing in infants and young children with a history of neonatal lung disease.

In our study, the hypoxia test was performed by exposing the children to high flow (15 l/min) 14% oxygen in nitrogen (Air Liquide, Perth, Western Australia, Australia), via a mask, for 20 min. Here we report the validation of this hypoxia test method in a group of infants and young children.

Seven children aged 2–51 months (3 boys) underwent a hypoxia test as described earlier. In addition, a gas sampling line was fixed under the nares of the child, permitting real-time recording of respired gas concentrations (Sensormedics Spectra V.12.3; Viasys, Yorba Linda, California, USA). A two-point calibration procedure encompassing the respirable range was performed before testing and accepted at an accuracy of 0.05%. Once the hypoxia test commenced, two separate periods of 30 s of quiet breathing were recorded and results saved for retrospective analysis. The median FiO2 for each inspiration was obtained and median FiO2 of FiO2 similar to that encountered during flight.

In summary, we present a simple, easily-applied hypoxia test method for use in infants and young children, which produces a median FiO2 similar to that encountered during flight.

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