LETTERS

Role of breathing exercises in hyperventilating subjects

Thomas and colleagues reported breathing training leading to improvements in asthma-specific health status and other patient-centred measures.[1] These included Asthma Quality of Life Questionnaire (AQLQ) scores, Hospital Anxiety and Depression (HAD) anxiety, HAD depression, Nijmegen scores and Asthma Control Questionnaire (ACQ) scores. The significant improvement in all the above stated scores except the last one at 6 months after the intervention could be due to a few inherent biases. This was discussed in our weekly journal club.

First, most of the population studied were hyperventilating subjects, as evidenced by the mean Nijmegen scores in both groups of >23. Breathing training might therefore have helped these hyperventilating subjects. Second, as stated in the article, most subjects with chronic disease would like to try alternative forms of treatment.[2] If this "alternative form" was mentioned during the invitation to take part in the study (which is not stated in the article), then all the subjects could have been self-motivated, which is not representative of the general population and hence the results cannot be generalised. Last, the subjects who underwent breathing training were encouraged to do the breathing exercises throughout the 6-month period whereas the control group had three sessions of asthma education with no such ongoing "controlling effect".

A significant improvement in forced expiratory volume in 1 s and a significant fall in exhaled nitric oxide 1 month after the intervention in the control group shows the beneficial effect of patient education. Hence, effective pharmacotherapy with asthma education continues to be the core of asthma treatment. The role of breathing training is possibly present in subjects who have a tendency to hyperventilate, which need not be due just to asthma but to any cause.

A B Palamarthy

Correspondence to: Dr A B Palamarthy, All Wales Adult Cystic Fibrosis Centre, University Hospital, Llandough, Vale of Glamorgan CF64 2HR, UK; anandom999@yahoo.com

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Authors' reply

We thank Dr Palamarthy for the interest shown in our study.[1] Three points are made. First, that most of the population studied were hyperventilating, as evidenced by the mean Nijmegen scores. This is incorrect. Our previous work has shown that one-fifth of men and one-third of women with asthma treated in the community in the UK have Nijmegen scores indicative of possible hyperventilation,[2] and that those with such high scores had a high probability of responding to breathing therapy.[3] In the current study, one of the research questions we addressed was whether patients with asthma with a low Nijmegen questionnaire score responded as well as those with a high score—that is, whether those with possible hyperventilation as well as asthma responded better than those with asthma but without symptoms of hyperventilation. Each randomisation group therefore had 50% of subjects with high Nijmegen scores (≥23) and 50% with low scores; this is stated in the Methods section and in the statistical analysis section where we state: "We also assessed whether the Nijmegen Questionnaire (a screening tool for symptomatic hyperventilation) score (<23 or ≥23) or physiological evidence of hyperventilation influenced response to breathing retraining". As reported in the Results section under the heading "Influence of hyperventilation markers on response to breathing training", no difference in response to breathing training was found between high and low scorers of the Nijmegen questionnaire, nor between those with low and higher carbon dioxide tensions at baseline. The results imply that this intervention can help many patients with impaired asthma-related health status, regardless of symptomatic or physiological evidence of hyperventilation.

The second point concerns the generalisability of the findings. As detailed in the Consort diagram, 516 subjects out of 3139 invitation letters (outlining the study protocol) responded with interest—a response rate of roughly 1 in 6—and 183 subjects were randomised. Recent work has shown that typical asthma clinical trials recruit a far lower proportion of potentially eligible subjects than ours,[4] usually in the order of 2%, and we know of no community-based controlled trials in asthma that have achieved a better recruitment rate. The point on generalisability applies to all randomised controlled trials but we feel that our study, because of the recruitment strategy, is likely to have better external validity than the trials on which current guidelines are based.

Finally, it is noted that the control group receiving asthma education achieved within-group benefits and a significant reduction in exhaled nitric oxide concentration. We agree with these observations, and also with the suggestion that pharmacotherapy and asthma education are vital aspects of asthma management. However, the within-group improvements from baseline and the greater improvements in patient-centred end points noted at 6 months in the breathing therapy group compared with the education group point to the possibility that this intervention may be an effective one for patients with impaired quality of life despite pharmacotherapy, and one that may benefit many patients with asthma. Future studies should investigate whether breathing exercises have additional benefits to effective education.

M Thomas,[1] I D Pavord[2]

[1] Department of General Practice and Primary Care, University of Aberdeen, Aberdeen, UK; [2] Institute for Lung Health, Glenfield Hospital, Leicester, UK

Correspondence to: Dr M Thomas, Centre of Academic Primary Care, School of Medicine and Dentistry, University of Aberdeen, Foresterhill Health Centre, Westburn Road, Aberdeen AB25 2AY, UK; mikethomas@doctors.org.uk

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Caesarean section and asthma

Roduit *et al* observed an association between caesarean section and asthma at the age of 8 years in a large group of Dutch children, and attribute the development of asthma partly to the mode of delivery, possibly through a different and delayed pattern of intestinal colonisation of micro-organisms.[1] Although this hypothesis is most interesting, in their discussion the differential reasons for caesarean sections were not addressed. As they state themselves, the prevalence of caesarean section in the Netherlands is low and elective caesarean section is rare. Because of this, the Dutch population of children born by caesarean section might be a highly selected group. One of the main reasons a caesarean section is conducted is a disproportion between the pelvic aperture and the fetal head circumference, and a large neonatal head circumference has been reported as a risk factor for asthma,[2] for any atopic disorder when corrected for neonatal body weight,[3] for

hay fever[4] and for raised IgE for common inhalation allergens at age 11.[5] It would therefore be most informative if the authors could provide additional data on the differences between the neonatal anthropometric data of the children with and without ceasarean section, and on the indications for caesarean sections themselves. This issue is of significant importance and of clinical relevance because, if indeed a causal relationship exists between mode of delivery and development of asthma, this would certainly make an argument against elective caesarean section for non-medical reasons. It would seem that there is currently insufficient evidence to infer a causal relationship, but it certainly seems worthwhile sorting this out.

P Merkus

Correspondence to: Dr P Merkus, P O Box 9101, Nijmegen 6500 HB, The Netherlands; p.merkus@cukz.umcn.nl

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Caesarean section and asthma: alternative explanations?

In their detailed analysis of almost 3000 children followed from birth until the age of 8 years, Roduit *et al*[1] showed that children born by caesarean section have a higher risk of asthma than those born by vaginal delivery. Surprisingly, the authors offer only one explanation for this finding—namely, delayed microbial colonisation—whereas we believe other mechanisms cannot be excluded.

As an alternative hypothesis we propose to investigate the possibility of confounding by factors already present at/before birth. This hypothesis is supported by studies showing that immunological parameters in cord blood are different between children born by vaginal delivery and those born by caesarean section.[2] One such factor could be head circumference which has been repeatedly found to be related to increased IgE and the development of asthma and related disorders,[3–5] and babies born by caesarean section probably have relatively high values.

J C van der Wouden,[1] R M D Bernsen[2]

[1] Department of General Practice, Erasmus MC, University Medical Center, Rotterdam, The Netherlands; [2] Department of Community Medicine, United Arab Emirates University, Al Ain, United Arab Emirates

Correspondence to: Dr J C van der Wouden, P O Box 2040, Rotterdam 3000 CA, The Netherlands; j.vanderwouden@erasmusmc.nl

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Presence of MBL in airways: is it a disease severity marker or an additional host defence mechanism?

We welcome the paper by Fidler and colleagues reporting the presence of mannose-binding lectin (MBL) in infected airways.[1] MBL is an important acute phase protein with pro- and anti-inflammatory immunomodulatory functions.[2] The collectin family comprises surfactant protein (SP)-A, SP-D and MBL, of which the latter is mostly present in peripheral blood while the other two are mostly located in the lung.[3] We agree with Fidler *et al* that MBL might contribute to lung host defence by acting locally at the airway surface because of its similar structure to lung collectins and its presence at a physiological level in the lung. It is possible, however, that the presence of MBL in the bronchoalveolar lavage (BAL) fluid of infected children might just be a marker of lung infection or disease severity. The data of Fidler *et al* clearly show a trend suggesting that MBL was more consistently detectable in acute than in chronic diseases; this may simply be a correlate of alveolar epithelial permeability. A similar study performed by our group on HIV-infected adults showed that the levels of MBL in BAL fluid were undetectable even when present in serum. The levels of SP-D in the same study were not significantly different in lung fluid from HIV-uninfected and HIV-infected individuals with a high CD4 count (>200), but were raised in HIV-infected individuals with a low CD4 count.[4] We tested the hypothesis that levels of SP-D or MBL in HIV-infected individuals would be lower than in HIV-uninfected individuals, but this was not the case. The phenomenon that levels of defence factors are poorly associated with protection has also been shown with other defence factors such as antibodies.[5]

In conclusion, we totally agree with Fidler *et al* that future studies should focus on measuring the functional aspect of collectins. Functional assays will help to determine whether the presence of MBL in the lung acts as an additional host defence or whether it is just a marker of disease severity.

K C Jambo, S B Gordon

Pulmonary Immunology Group, Liverpool School of Tropical Medicine, Liverpool, UK

Correspondence to: K C Jambo, Pulmonary Immunology Group, Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK; kjambo@liverpool.ac.uk

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Thoracic ultrasound: an important skill for respiratory physicians

We read with interest the article by Qureshi and colleagues describing thoracic ultrasound (TUS) characteristics for the detection of malignant pleural effusions.[1] This relatively simple bedside technique has been routinely performed by the respiratory physicians in our department in a busy general hospital for the last 4 years, resulting