

LETTERS TO THE EDITOR

Pseudo-steroid resistant asthma

The report by Thomas *et al* of patients whose symptoms masquerade as asthma is to be welcomed, although it is not clear how the authors selected their patients from all those referred to their clinic with "true" steroid resistant asthma. In addition, the opportunity has not been taken to determine what proportion of these patients would have fulfilled the criteria for type 1 brittle asthma. In a recent review of brittle asthma² I discussed, along with one of the authors of this report, factors such as gastro-oesophageal reflux, psychosocial aspects, sleep apnoea, and hyperventilation/vocal cord adduction as contributory or coincident factors, and in that review we put in a plea for future studies to try to characterise phenotypically patients at the severe end of the spectrum compared with other phenotypes or genotypes.

In our patients with brittle asthma hyperventilation is common and I agree that it can be difficult to discern between pure hyperventilation and hyperventilation on a background of pre-existing asthma. The authors describe a hyperventilation test but it is perhaps surprising that they have not reported which test they used nor what objective measures were made. The tendency to hyperventilate commonly overlaps with the difficult area of psychosocial aspects in severe asthma.^{3,4} The authors misquote our paper³ in that we did not, in that study, compare severe asthma with another chronic disease group. Equally, the authors have not read our second paper⁴ where the psychosocial impact was greater. This study also provided evidence of unusual self management strategies in this group which, in many cases, led to delay in starting or increasing oral steroids. The authors of this report have not attempted to correlate the well established indices of psychosocial burden—including assessment of panic/fear/anxiety—with hyperventilation, and this would have been both helpful and interesting. Vocal cord adduction is also difficult to verify as it is a diagnosis usually made, as here, on clinical grounds, although video recording the vocal cords during an episode is generally regarded as the gold standard.

In our experience we have seen a number of patients similar to those described in whom oral steroids can, in the short term, be reduced. However, there is often a tendency for oral steroid doses to increase insidiously once the patient is back in the community, reinforcing the need for clinical psychological input in these patients.

Patients with intractable breathlessness as seen in brittle asthma and in hyperventilation are difficult to sort out, but it is crucial to be aware that both can co-exist; it is not just a matter of "pseudo-steroid resistant asthma".

JON G AYRES
Department of Respiratory Medicine,
Birmingham Heartlands Hospital,
Bordesley Green East,
Birmingham B9 5SS,
UK

- 1 Thomas PS, Geddes DM, Barnes PJ. Pseudo-steroid resistant asthma. *Thorax* 1999;54:352-6.
- 2 Ayres JG, Miles JR, Barnes PJ. Brittle asthma. *Thorax* 1998;53:315-21.
- 3 Garden GMF, Ayres JG. Psychiatric and social aspects of brittle asthma. *Thorax* 1993;48:501-5.
- 4 Miles JF, Garden G, Ayres JG. Psychological factors in brittle asthma: a case control study. *Clin Exp Allergy* 1997;27:1151-9.

We would like to thank Dr Thomas and coworkers for their detailed report of a number of cases of apparent severe asthma resulting in prescribed oral steroid therapy for which an alternative diagnosis, particularly of a functional nature, appeared to be the major problem.¹ Those physicians who run difficult asthma clinics around the country will be familiar with the practical issues raised by these patients, and the authors' advice with an investigation protocol, psychological and functional management is helpful.

However, we feel that there are a number of important issues that should have been addressed by the authors. Firstly, the flow-volume loops presented are not altogether characteristic of vocal cord dysfunction in that a number of them show good reproducibility and none show the typical features of large airway obstruction. Secondly, six of the 14 cases detailed have had episodes of ventilation. Superficially this might imply the presence of severe asthma, but no information regarding these episodes is presented. Critical information can be gleaned from anaesthetic records and this should be part of the routine assessment of these patients. The inflation pressures (if accurately recorded) and duration of required ventilation can be used to assess the degree of airway resistance and the reversibility of it at the time of ventilation. Low pressures and a short period of ventilation are indicators that the episode was not primarily severe bronchoconstriction (as presumably clinically perceived at the time), and is a major clue to the underlying functional aetiology of the problem (either vocal cord dysfunction or pseudo-severe asthma).² High inflation pressure or a prolonged period of ventilation suggest that there is severe airways obstruction and any associated functional disease is superimposed.

This leads on to the next point—namely, that severe asthma and vocal cord dysfunction/hyperventilation may, and frequently do, co-exist. Bronchoscopic examination may help to identify those patients who have significant underlying inflammatory disease of the airways, but the sensitivity and specificity of lavage and biopsy material has not been evaluated in patients with severe asthma on steroid therapy. In addition, the identification of vocal cord dysfunction and/or hyperventilation does not necessarily result in a successful withdrawal of steroids and, indeed, inappropriately rapid or unsupervised withdrawal of treatment may have catastrophic results if severe asthma and vocal cord dysfunction are co-existent.

The readers of this article need to be aware of the practical dangers involved in the complex management of these patients. Rapid withdrawal of steroids in a patient who has been shown to have either vocal cord dysfunction or hyperventilation is hazardous as the latter conditions may be a sequel to or co-exist with severe asthma. We would recommend a staged steroid withdrawal during hospital supervision even in patients with

convincing evidence of alternative pathology to explain their apparent "pseudo-steroid resistant asthma".

R NIVEN
F KELLETT
North West Lung Research Centre,
Wythenshawe Hospital,
Southmoor Road,
Wythenshawe,
Manchester M23 9LT,
UK

- 1 Thomas PS, Geddes DM, Barnes PJ. Pseudo-steroid resistant asthma. *Thorax* 1999;54:352-6.
- 2 Niven RMcL, Roberts T, Pickering CAC, *et al*. Functional upper airways obstruction presenting as asthma. *Respir Med* 1992;86:513-6.

AUTHORS' REPLY Professor Ayres rather misses the point of our paper and has ignored the date of submission.

The article was a retrospective review of those labelled as having asthma which was unresponsive to corticosteroids. Our main finding was that virtually all of these cases had no evidence of asthma at all. Detailed discussion of brittle asthma was therefore inappropriate.

Furthermore, the submission antedates two of his articles to which he feels we should have referred. Publication was delayed by circumstances beyond our control at *Thorax*.

He feels that we have misquoted his article on brittle asthma but this contains references to psychological disorders in other disease states. To define a denominator for the problem of pseudo-steroid resistant asthma is rather difficult in the decidedly skewed population as found at the Brompton Hospital, but it would be useful in a study of atypical or "difficult" asthma performed in a prospective manner.

The purpose of the article was not to address the separate issue of brittle asthma, as we would assume that there is clear documentation that those subjects do indeed have asthma. The purpose of the article was to draw attention to the fact that some individuals with breathlessness are inappropriately given corticosteroids and inappropriately labelled as having steroid resistant asthma.

Drs Niven and Kellett raise the question of airway obstruction and its measurement. The flow-volume loops shown were the two most similar of a number of attempts, which is why they may appear to be reproducible. There is, however, unusual oscillation on the expiratory curve suggestive of variable obstruction. This may represent repetitive vocal cord movements causing variable obstruction, and it would be unlikely that the flow-volume loops of these subjects would show classic features of a large fixed obstruction.

We would agree that inflation pressures in ventilated subjects are important but unfortunately we were unable to address this problem as the patients were ventilated at the referring hospitals.

We would concur that any of the confounding conditions mentioned such as vocal cord dysfunction could co-exist in asthma and these need to be considered as contributory factors.

With regard to withdrawal of corticosteroids, oral steroids were withdrawn while monitoring the subjects as inpatients. We did not advocate abrupt steroid withdrawal after prolonged treatment.

PAUL S THOMAS
Department of Respiratory Medicine,
Prince of Wales Hospital,
Randwick,
Sydney, NSW 2031,
Australia