in severe steroid dependent asthmatic patients (aged 13–65) further supports the possibility that antibody titres indicative of "previous infection" may also indicate persistent chronic infection.[3]

Acute primary (presence of IgM) or secondary (fourfold change in titre without IgM) *C pneumoniae* infection has been reported to initiate asthma in previously non-asthmatic individuals.[4] Since the incidence of asthma in adults is very small (around one per 1000 per year) it is likely that most of the acute exacerbations occurred in patients who had had previous wheezing episodes. It would be interesting to know whether Cook *et al* can retrospectively identify any patients who had their very first wheezing episode; this might be easier in general practice than in a hospital based study.

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1 Cook PJ, Davies P, Tunnicliffe W, *et al. Chlamydia pneumoniae* and asthma. *Thorax* 1998;**53**:254–9.
2 Hahn DL. Intracellular pathogens and their role in asthma: *Chlamydia pneumoniae* in adult patients. *Eur Respir Rev* 1996;**6**:224–30.
3 Hahn D, Bukstein D, Luskin A, *et al*. Evidence for *Chlamydia pneumoniae* infection in steroid-dependent asthma. *Ann Allergy Asthma Immunol* 1998;**80**:45–9.
4 Hahn D. Incident wheezing and prevalent asthma have different serologic patterns of "acute" *Chlamydia pneumoniae* antibodies in adults. *Proceedings of the Third Meeting of the European Society for Chlamydia Research, Vienna, Austria*. Bologna: Società Editrice Esculapio, 1996: 226.

Non-Hodgkin's lymphoma with CFA

We read with interest the case report by Orchard *et al* on non-Hodgkin's lymphoma arising in cryptogenic fibrosing alveolitis (CFA).[1] Although the authors state that this has not been described previously, we recently reported six cases of pulmonary B cell non-Hodgkin's lymphomas arising in patients with autoimmune disorders, three of whom had CFA.[2] As in the case described by Orchard *et al*, prognosis in these three patients was much poorer than that in the patients with high grade pulmonary non-Hodgkin's lymphomas unassociated with CFA, presumably due to the combined effects of the two diseases.

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1 Orchard TR, Eraut CD, Davison AG. Non-Hodgkin's lymphoma arising in cryptogenic fibrosing alveolitis. *Thorax* 1998;**53**:228–9.
2 Nicholson AG, Wotherspoon AC, Jones AL, *et al*. Pulmonary B-cell non-Hodgkin's lymphoma associated with autoimmune disorders: a clinicopathological review of six cases. *Eur Respir J* 1996;**9**:2022–5.

AUTHOR'S REPLY We are grateful to Dr Nicholson and Professor Corrin for pointing out their very interesting report, which was published after the original writing of our case report.

In the patient we reported the association was with cryptogenic fibrosing alveolitis (CFA) alone whereas, interestingly, the three patients they report had CFA associated with other systemic autoimmune disorders. The fact that CFA alone may be associated with B cell lymphomas, and the poorer prognosis seen by Nicholson and Corrin in their patients, as well as ours, supports the hypothesis that chronic local stimulation of the lymphoid system may play an important part in the aetiology and prognosis of these tumours.

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BOOK REVIEWS

Respiratory Measurement. Göran Hedenstierna. (Pp 184, paperback; £19.95 (UK), £22.00 (overseas)). London: BMJ Books, 1998. ISBN 0 7279 1207 0.

A large amount of information has been packed into the 184 pages of this new guidebook in the Principles and Practice Series. This is a comprehensive review of the principles of ventilation and gas exchange with special emphasis on the application of pulmonary function measurement during anaesthesia. The book details physiological principles and gives practical measurement guidance, with common sources of error, in the normal circumstances and during anaesthesia. The content is concise, the style direct and occasionally hard going. The text is clear and the diagrams are worth a special mention for their clarity and simplicity. This is not a textbook for beginners and requires a moderate familiarity with the principles of respiratory physiology, and the rules which govern respiratory mechanics and gas measurement. This guide represents excellent value for money and would be equally at home in the pulmonary function laboratory as well as the anaesthetics department. —SR

Asthma: Basic Mechanisms and Clinical Management. 3rd Edition. Barnes PJ, Rodger IW, Thomson NC, eds. (Pp 942; hardback; $150.00). London: Academic Press, 1998. 0 12 079027 0.

This is the third edition of an established book. Aiming to bring together all the recent information on basic mechanisms of asthma and also cover clinical aspects and therapy in depth, this is achieved successfully. The scope of the book provides accessible reviews of all facets of asthma, from epidemiology and physiology to allergen avoidance, including recent developments in these fields. Modifications to the popular second edition include separate chapters on mediator antagonists and immunomodulators with consideration of the potential therapeutic benefits of intervening in the complex inflammatory and pharmacological pathways systematically covered in previous chapters. A new chapter on the pharmacoeconomics of asthma treatments provides a pertinent reminder that, after the wonders of basic science and the development of beneficial interventions, a wider perspective is required to successfully deliver benefits to those who require them. The addition of colour plates provides a welcome change to the previous black and white prints of the old edition which look a little drab in retrospect.

Well written by authorities in their fields and uniformly edited with an attractive presentation, this is an excellent book which succeeds in linking the rapidly developing body of knowledge on asthma with current treatment, while keeping the future constantly in mind.—AF

CORRECTION

Clinical features of non-smokers with α_1-antitrypsin deficiency

The authors of the paper entitled "Clinical features and prognosis of life time non-smokers with severe α_1-antitrypsin deficiency" by N Seersholm and A Kok-Jensen, which appeared on pages 265–8 of the April issue of *Thorax*, regret that some errors occurred in the text and in table 3. On page 267 the first line of column 1 should have read: ". . . 50 years at entry was **56%** compared with **50%** for the subjects over 50 years . . .". Table 3 is reproduced here with the corrections shown in bold italics.

Table 3 Mean (SD) FEV_1 % predicted and FEV_1/FVC of index and non-index cases stratified by age at entry

	Index cases	*Non-index cases*	*p value (t test)*
All age groups	n = 27	n = 40	
FEV_1 (% predicted)	54 (25)	100 (21)	<0.001
FEV_1/FVC	0.57 (0.18)	0.79 (0.13)	<0.001
N (%) with FEV_1 % pred ⩽70%	20 (74%)	3 (8%)	<0.001
Age at entry <50 years	n = 8	n = 26	
FEV_1 (% predicted)	***56 (37)***	***100 (19)***	<0.001
FEV_1/FVC	***0.53 (0.20)***	0.80 (0.12)	<0.001
N (%) with FEV_1 % pred ⩽70%	4 (50%)	2 (8%)	<0.001
Age at entry ⩾50 years	n = 19	n = 14	
FEV_1 (% predicted)	***50 (20)***	***101 (24)***	<0.001
FEV_1/FVC	0.58 ***(0.17)***	0.78 (0.14)	<0.001
N (%) with FEV_1 % pred ⩽70%	16 (84%)	1 (7%)	<0.001