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References

Asthma guidelines
We read with interest the new British guideline on the management of asthma recently published as a supplement to Thorax.1 The evidence review groups and guideline authors are to be congratulated on the production of a document of exceptional clarity and ease of use. There is no doubt that adherence to the guideline could contribute substantially to the better management of asthma in the majority of adults, including most elderly patients. However, we feel moved to point out the lack of reference to the difficulties of diagnosis and treatment in patients with abnormalities of cognition, praxis, dexterity and executive function, most of whom are elderly with varying degrees of dementia and/or cerebrovascular disease. This is a retrograde step as earlier versions of the asthma guideline referred to some of these issues. We see this as a missed opportunity to improve the detection and management of asthma in this group of patients who are known to have a high level of morbidity from that condition (class 2 evidence), and in whom the asthma mortality curve is not falling (class 1 evidence). Some of the most recent published work in this domain will not have been included in the evidence travels for the guideline. Nevertheless, there is ample evidence in the literature relating to the quality of clinical information (class 2+ and 3) in such patients, including spirometry (class 2+) and on the issues of inhaler device competence (class 2+ and 3), selected training (class 2+). There is also class 2+ evidence that elderly subjects are less able than younger subjects to detect changes in airflow resistance, which has implications for reliever therapy at steps 1 and 2 of the guideline. We strongly advocate that future revisions of the guideline should take account of this evidence, probably as a grade C recommendation or good practice point.

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Self-management of asthma in general practice
We welcome the paper by Thoonen and colleagues on self-management of asthma in general practice1 as we firmly believe that self-management of chronic diseases is a promising area for innovation in general practice. We wondered whether differential withdrawals—specifically, the difference between groups in the number of patients for whom successfully treated weeks could be calculated—may have affected the outcomes. As reported, 92% of the patients in the usual care (UC) group provided data for this primary outcome compared with 85% of the self-management (SM) group. The mean percentage of successfully treated weeks was 72%
Bronchoscopy in patients with suspected PCP: supine or sitting?

We read with interest the case report by Newton et al describing platypnoea and orthodeoxia in a patient with pneumocystis pneumonia (PCP; Cpcr) . We have had occasion to add our own experience and question whether the observation has an important clinical implication.

A 29 year old patient who had been born and raised in Zambia presented 10 months after arrival in the UK with a history of cough, breathlessness, and progressive dyspnoea. A chest radiograph showed bilateral diffuse lung shadowing, maximal in the perihilar regions and lower zones, but with relative sparing of the lung bases. Feno, breathing air, the patient had an SaO2 of 98%.

Our patient showed orthodeoxia similar to that described by Newton et al. This became clinically important when bronchoscopy was performed in the sitting position. Predominant mid and lower zone shadowing is a common radiographic feature in PCP and we wonder whether the phenomenon of platypnoea and orthodeoxia may be widespread in such patients. A study to identify the frequency of this finding would be worthwhile and easy to do. We suggest that supine and sitting SaO2 measurement should be routine before bronchoscopic investigations in such patients and that bronchoscopy in the supine position might be the approach of choice for patients with suspected PCP.

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References

Atopic cough: little evidence to support a new clinical entity

A cough is by far the most common reason for a patient to seek medical advice. Most of these consultations are for acute cough caused by a myriad of respiratory viruses. In practice, we do not attempt to differentiate rhinovirus infection from adenovirus because no important consequence arises from this diagnostic precision. In chronic cough numerous studies have shown that the overwhelming majority of patients are suffering from one of three conditions—form asthma, gastro-oesophageal reflux, or rhinitis. The question is whether any further subdivision is justified or merely muddies the water.

In their recent paper published in Thorax Fujiwara and colleagues, by inventing a diagnosis of “atopic cough”, have succeeded in adding further unnecessary complexity to the area. There are many aspects of this paper which cannot be left unchallenged and we have outlined these below.

The evidence to support a new clinical entity “atopic cough” is tenuous and is further hampered by the extremely vague term “probable atopic cough”. This point is well illustrated in their table 2 which outlines a variety of diagnostic permutations possible after interpretation of a series of observations. We suspect the authors have, in a significant number of these “probable” patients, merely described atopic individuals with cough predominant asthma. Certainly most patients with cough predominant asthma, (merely) described and cannot be used to support the proposition that atopic cough is unique.

The statement “the defining physiological feature is increased cough sensitivity without BHR...” to differentiate atopic from non-atopic asthma is tenuous. Cough sensitivity can be measured in several ways, but the authors do not provide sufficient information in the paper to conclude that these patients failed to respond to steroids, and have indicated that response to bronchodilators was tested in neither a randomised nor controlled way.

The authors have relied heavily on the “absence of transformation to typical asthma...” to differentiate asthma phenotype from asthma, and this is misleading. Such a conclusion can hardly be supported by a telephone follow up which relies on patient recall and subjective assessment of symptoms. In some series up to one third of patients with PCP have cough and that bronchoscopic examination, while supine and breathing air, the patient had an SaO2 of 98%.

Our patient showed orthodeoxia similar to that described by Newton et al. This became clinically important when bronchoscopy was performed in the sitting position. Predominant mid and lower zone shadowing is a common radiographic feature in PCP and we wonder whether the phenomenon of platypnoea and orthodeoxia may be widespread in such patients. A study to identify the frequency of this finding would be worthwhile and easy to do. We suggest that supine and sitting SaO2 measurement should be routine before bronchoscopic investigations in such patients and that bronchoscopy in the supine position might be the approach of choice for patients with suspected PCP.

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References
or bronchial responsiveness are entirely
tations of eosinophilic airway inflammation,
nise that there are different clinical manifes-
tations runs counter to the progress of
matic” We continue to suggest that such
all of these should be categorised as “asth-
corticosteroids are effective in atopic cough as
criticise the concept. They insist that, because
arguments as McGarvey and Morice to
aetiological entity in 1992, a number of Japa-
 disclose the mechanism, thereby contributing to both our understanding of the pathophys-
ology of atopic cough and to better specific treatment.
In our study patients with a definite diagnosis of atopic cough did not go on to develop typical asthma, indicating that atopic cough is not a precursor to asthma. Only one of 56 patients with a probable diagnosis of atopic cough developed typical asthma 11.5 years after her first visit. Although the patient's bronchial responsiveness was increased, her bronchial reversibility and diurnal variation in peak expiratory flow rate were within normal limits; broncho-
dilator treatment was not effective, leading to a probable diagnosis of atopic cough. Although some investigators believe that BHR is the key criterion for a diagnosis of cough variant asthma, this is incorrect. The most important feature of cough variant asthma is isolated chronic cough responsive to bronchodilators. Furthermore, it has been clearly shown that measurement of bronchial responsiveness cannot predict the efficacy of bronchodilators in the treatment of cough.
Figure 1 shows a consensus opinion of the Japanese Cough Research Society concerning the diagnosis of cough variant asthma and atopic cough based on bronchial responsive-
ness and efficacy of bronchodilators. Only area C represents definite cough variant asthma for the purposes of selecting clinical research subjects; areas A + D represent prob-
able cough variant asthma for general clinical practice, area A + B represent definite atopic cough, and areas A + C represent probable atopic cough. In addition, areas B + D represent eosinophilic bronchitis without asthma.

Worldwide problems regarding the diagno-
sis of cough variant asthma, atopic cough, and eosinophilic bronchitis without asthma are as follows: (1) many researchers have recognised areas A + C as cough variant asthma regard-
less of responsiveness to bronchodilators; and (2) because inhaled corticosteroids are be-
lieved to be the definitive asthma treatment, the diagnosis of asthma is based on the responsiveness of the cough to corticosteroid therapy despite the presence of non-
asthmatic eosinophilic airway disorders. We emphasise again that bronchodilators, which have no effect on cough sensitivity,17 are efficac-
ious against coughing only in cough variant asthma. Thus, as the efficacy of bronchodil-
ators is a key criterion for the diagnosis of cough variant asthma, many Japanese investi-
gators use information regarding broncho-
dilator responsiveness of a cough to diagnose cough variant asthma, recognising the pres-
ence of non-asthmatic eosinophilic airway disorders.

While it is impossible to assess the efficacy of bronchodilator therapy in individual

patients in a randomised and placebo controlled manner, the assessment we used is not
difficult.1 Although the placebo effect may lead to an incorrect diagnosis of cough variant asthma, no effect is meaningful enough to exclude cough variant asthma or cough predominant asthma.

One fundamental feature of cough variant asthma is mildly increased bronchial responsi-
siveness unrelated to cough sensitivity. It is well known that cough sensitivity is in-
creased in some patients with cough variant asthma and asthma (asthmatic patients), as
pointed out by McGarvey and Morice. Our opinion is that increased cough sensitivity is a complication in asthmatic patients but it is not a fundamental aspect of the asthmatic
airway. We label such patients as having “cough variant asthma” or “asthma complica-
ted with cough hypersensitivity”15. In these patients bronchodilator therapy proves effec-
tive; histamine H1-antagonists are useful, as shown by Shioya et al. Corticosteroids do, of
course, relieve the cough because they improve both cough hypersensitivity and BHR which are caused by eosinophilic airway inflammation via possibly different mechani-
isms.

The data presented in our paper showing that six of 20 patients with cough variant asthma not taking long term inhaled cortico-
steroid therapy developed typical asthma are consistent with previous reports. Although Orejas et al. reported that typical asthma occurred in only 6% of 63 patients with cough variant asthma during a 3 year follow up period, there exists an important problem in the diagnostic criteria for cough variant asthma. Orejas et al and many other investiga-
tors have diagnosed cough variant asthma based on BHR without assessing the efficacy of bronchodilator therapy or measuring cough sensitivity, resulting in the inclusion of non-asthmatic patients who, in fact, have atopic cough.

McGarvey and Morice and other investiga-
tors feel that subdividing eosinophilic airway inflammation disorder causes unwieldy complexity in the diagnosis of chronic cough. We hold that delineating the pathophysiology of specific subdivisions such as atopic cough and cough variant asthma will allow more effective and specific treatments to be used rather than relying solely on inhaled corticosteroids which are non-specific to the actual cough mechanism. We continue to suggest that atopic cough should be considered as an entity separate from cough variant asthma, with a unique pathophysiology and its own 

rate of asthma onset.

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References
1 Gibson PG, Fujimura M, Nimi A.

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Transoesophageal echocardiography and lung cancer staging

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