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Transoesophageal echocardiography and lung cancer staging

Cardiac involvement by tumour is found in 25% of patients who die from lung cancer and, in the majority of these patients, the involvement is asymptomatic.[1] With a significant proportion of patients subjected to lung cancer surgery dying from disease progression within 6 months of surgery, the British Thoracic Society guidelines on the selection of patients with lung cancer for surgery may help to reduce this number.[2 3] These guidelines acknowledge the excellent staging potential of PET scanning but only mention echocardiography in the context of valvular or ischaemic heart disease. Echocardiography is an excellent modality for detecting cardiac malignancy.[4 5] An increasing number of anaesthetists are joining the already large number of cardiologists able to perform transoesophageal echocardiography (TOE). We suggest that patients presenting for lung cancer surgery who have either ECG abnormalities or poorly differentiated paracardiac tumours should undergo TOE before thoracotomy. The TOE examination would add little to the total operative time but would add contemporary information to existing older information regarding disease stage. Moreover, there would be a small number of patients who would be spared thoracotomy.

A recent case in which left atrial extension of a lung cancer was demonstrated by intraoperative TOE but missed by preoperative PET scanning and the standard staging methods lends anecdotal weight to our argument. Not only did our patient suffer an unnecessary thoracotomy, but also left popliteal artery tumour embolism. This is not the first time a patient has had a thoracotomy abandoned because of tumour involvement of the left atrium revealed only by intraoperative TOE.[6] In both cases TOE in the anaesthetic room would have prevented thoracotomy and, in our case, distant tumour embolisation may not have occurred.

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Asthma guidelines

We read with interest the new British guideline on the management of asthma published recently 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 trawl 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), selection and 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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Authors' reply

We thank Dr Allen and colleagues for raising these issues. As they point out, the difficulties of diagnosis and treatment are mainly features of the co-morbidity which increases with age, not age itself, and we had not felt it desirable or possible to cover all the changes to routine practice which might be required because of the presence of other diseases. We cannot comment on the items of evidence included in their letter since references are not given, except to say that their points seem correct in principle but some of the evidence levels look unrealistic—for example, one of the flaws of the current grading system is that, however good the evidence on something like asthma mortality, it cannot be level 1 evidence since that is possible for randomised controlled trials only. Nonetheless, we agree that general reference to the potential problems in this patient group might be appropriate and we will consider this in the next version of the guideline.

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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 practice[1] 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%

and 78% for the UC and SM groups, respectively, a difference reported to be statistically significant. As we do not expect that these specific outcomes were analysed on an intention to treat basis, non-random withdrawals might have biased the findings—for example, when most patients who withdrew from the SM group did so because they were not able to comply with the SM programme *and* their asthma was badly controlled, this would affect the outcomes, inflating the differences in favour of the SM group.

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Authors' reply

In their letter van der Wouden and colleagues question the issue of selective withdrawal of subjects from the two study groups which may have had consequences for the validity of our conclusions. As a possible source of bias in calculating successfully treated weeks this may be an important issue which was not discussed in depth in our original paper.

To address this question we performed an additional analysis of the study records of all subjects excluded from the calculation of the successfully treated weeks. We divided all these subjects into two categories: (1) those excluded from the calculation because of side effects to inhaled steroids, poor asthma control, referral to a pulmonary physician, or non-compliance with the study protocol (categorised as "unable to comply"), and (2) subjects excluded because of a change of address or GP, pregnancy, or unknown reasons (categorised as "other reasons"). The number of subjects in the "unable to comply" subgroup was four out of 15 in the self-management group (4% of all self-management subjects) and five out of eight in the usual care group (5% of all usual care subjects). The number of withdrawals for asthma related reasons (including poor asthma control) was therefore relatively small in both study groups and was, in fact, relatively higher in the usual care group. We therefore believe that the impact on the number of successfully treated weeks must have been very limited and, if present at all, was in favour of the usual care group rather than the self-management group.

We conclude that, even if there were differences in the withdrawals between the two groups, this does not change our conclusion that self-management of asthma is at least equally as effective as the asthma treatment usually provided in Dutch primary care. This conclusion supports the view of van der Wouden and colleagues that self-management of asthma is a promising innovation in general practice.

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Bronchoscopy in patients with suspected PCP: supine or sitting?

We read with interest the case report by Newton *et al*[1] describing platypnoea and orthodeoxia in a patient with pneumocystis pneumonia (PCP). We would like 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 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 apices. Pao_2 breathing air was 9 kPa. PCP was suspected and fibreoptic bronchoscopy and BAL performed. Sedation was with 2 mg midazolam. The bronchoscopy, which was performed with the patient sitting, was complicated by a fall in Sao_2 to 85% which was only partially corrected by supplemental oxygen. Pneumocystis was identified in the BAL fluid sample and treatment commenced with high dose co-trimoxazole. The day after the bronchoscopic examination, while supine and breathing air, the patient had an Sao_2 of 98%. Rising to the sitting position led to a steady fall in Sao_2 to 94%, which rapidly corrected to 99% when the patient returned to the supine position.

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[2] 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 Sao_2 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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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.[1] 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—a form of asthma, gastro-oesophageal reflux, or rhinitis.[2] The question is whether any further subdivision is justified or merely muddies the water.

In their recent paper published in *Thorax* Fujimura and colleagues, by inventing a diag-

nosis of "atopic cough", have succeeded in adding further unnecessary complexity to the area.[3] 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 investigations. We suspect the authors have, in a significant number of these "probable" patients, merely described atopic individuals with cough predominant asthma. Certainly the demonstration of bronchial hyperreactivity (BHR) in 15 such patients (median PC_{20} 2.5 mg/ml, range 1.25–5.0), many of whom had airway eosinophilia and all of whom appear atopic, is suggestive. Such diagnostic imprecision may yet have therapeutic consequences, 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 a controlled way.

The authors have relied heavily on the "absence of transformation to typical asthma" to differentiate atopic cough from asthmatic cough. 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 cough variant asthma may develop typical asthma symptoms,[4] although in a 3 year follow up of 63 patients with cough variant asthma typical wheezing occurred in only 6%.[5] Thus, lack of progression to typical asthma is well 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 in atopic cough, and BHR without cough hypersensitivity in cough variant asthma" is simply untrue. Capsaicin hypersensitivity in patients with asthmatic cough has been reported by many researchers.[6–9] This heightened cough sensitivity is, of course, not directly related to bronchomotor tone, but most probably reflects a different pattern of airway inflammation.[10]

With reference to the authors' interpretation of the study by McGarvey *et al*,[11] we strongly contest the suggestion that patients identified as having cough variant asthma may have been misclassified. In this study steroid efficacy was not a sole criterion for the diagnosis of cough variant asthma as all 10 patients had BHR (mean PC_{20} 3.44 mg/ml, range 0.04–8). Only two of these patients were atopic.

We believe our clinical understanding of asthmatic cough is enhanced by the recognition that an individual patient may show different features of the disease process. The unnecessary subdivision into arbitrarily defined "diseases" such as atopic cough (or, indeed, eosinophilic bronchitis) is not helpful either diagnostically or therapeutically.

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Authors' reply

Since we proposed atopic cough as an aetiological entity in 1992, a number of Japanese chest specialists have used the same arguments as McGarvey and Morice to criticise the concept. They insist that, because corticosteroids are effective in atopic cough as well as in asthma and cough variant asthma, all of these should be categorised as "asthmatic". We continue to suggest that such insistence runs counter to the progress of medical science. It is very important to recognise that there are different clinical manifestations of eosinophilic airway inflammation,[1] and cough sensitivity and bronchomotor tone or bronchial responsiveness are entirely independent.[2 3] Two mechanisms of non-productive cough are generally recognised: cough triggered by increased bronchomotor tone and cough based on increased cough sensitivity. Both may result from eosinophilic airway inflammation, although via different mechanisms. We do not yet fully understand why eosinophilic airway inflammation increases cough sensitivity in atopic cough while both asthma and cough variant asthma exhibit mild bronchial hyperresponsiveness (BHR) without increased cough sensitivity. We are hopeful that future studies will disclose the mechanism, thereby contributing to both our understanding of the pathophysiology of atopic cough and to better specific treatment.

In our study[4] 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 58 patients with a probable d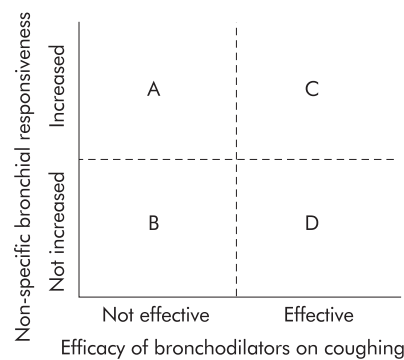iagnosis of atopic cough developed typical asthma 11.5 years after her first visit. Although the patient's bronchial responsiveness was increased, her bronchial reversibility and diurnal variation in peak expiratory flow rate were within normal limits; bronchodilator treatment was not effective, leading 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.[5] Furthermore, it has been clearly shown that measurement of bronchial responsiveness cannot predict the efficacy of bronchodilators in the treatment of cough.[6]

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 responsiveness and efficacy of bronchodilators. Only area C represents definite cough variant asthma for the purposes of selecting clinical research subjects; areas C + D represent probable cough variant asthma for general clinical practice, area B represents definite atopic cough, and areas A + B represent probable atopic cough. In addition, areas B + D represent eosinophilic bronchitis without asthma.[7]

Worldwide problems regarding the diagnosis 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 regardless of responsiveness to bronchodilators; and (2) because inhaled corticosteroids are believed 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,[2] are efficacious against coughing only in cough variant asthma. Thus, as the efficacy of bronchodilators is a key criterion for the diagnosis of cough variant asthma, many Japanese investigators use information regarding bronchodilator responsiveness of a cough to diagnose cough variant asthma, recognising the presence 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.[4] 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 responsiveness unrelated to cough sensitivity. It is well known that cough sensitivity is increased 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 complicated with cough hypersensitivity". In these patients bronchodilator therapy is not sufficient; histamine H_1-antagonists are useful, as shown by Shioya *et al*.[8] 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 mechanisms.

The data presented in our paper[4] showing that six of 20 patients with cough variant asthma not taking long term inhaled corticosteroid therapy developed typical asthma are consistent with previous reports. Although Orejas *et al*[9] 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 investigators 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 investigators feel that subdividing eosinophilic airway 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.

Figure 1 Recognition of cough variant asthma, atopic cough, and eosinophilic bronchitis without asthma based on bronchial responsiveness and bronchodilator response in chronic coughers with eosinophilic airway inflammation.

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