PostScript

LETTERS TO THE EDITOR

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The editors will decide as before whether to also publish it in a future paper issue.

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 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 pre-operative 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.5 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 tables 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 practice6 as we firmly believe that self-management of chronic diseases is a promising area for innovation in general practice.7 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%
B Bronchoscopy in patients with suspected PCP: supine or sitting?

We read with interest the case report by Newt


Authors’ reply

In their letter van der Wouden and colleagues question the issue of selective withdrawal of subjects in 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 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, 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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References


Atopic cough: little evidence to support a new clinical entity

A cough is by far the most common reason for consultation in primary care. This consultation is often vexed by the multiplicity of possible causes, many being non-specific. A number of terms have been used to describe these conditions—e.g., atopic, eosinophilic, rhinitis, asthma, gastro-oesophageal reflux, or rhinitis. 1 Why is it that the term atopic cough is so often used?

1 The term atopic is used to define those conditions that are presumed to have a genetic component inherited from parents. This component is then driven by the presence of specific IgE antibodies in patients with atopy. These patients react with specific antigens that are not the cause of the disease process. In such cases the atopy may be defined as a predisposition.

In atopic asthma, the IgE antibodies are directed against aeroallergens such as house dust mite or pollen. A similar situation may be seen in atopic rhinitis, where there may be a reaction to allergens such as pollen or house dust mite. In atopic cough, the antibodies may be directed against a variety of respiratory viruses, such as rhinovirus, parainfluenza virus types 1 and 2, adenovirus, and respiratory syncytial virus. The term atopic is therefore appropriate for atopic asthma and atopic rhinitis, but it is inappropriate for atopic cough.

We believe the term atopic is inappropriate for atopic cough because it is not clear whether the term is used to describe a new clinical entity, or to describe all coughs with a genetic predisposition. The term atopic cough may be defined as a cough that is associated with a genetic predisposition. The term atopic cough is a misnomer and should be replaced by a more specific term.

In atopic asthma, the term allergic is appropriate, as it describes the mechanism by which the condition is caused. In atopic rhinitis, the term allergic is also appropriate, as it describes the mechanism by which the condition is caused.

In atopic cough, the term atopic is inappropriate, as it is not clear whether the term is used to describe a new clinical entity, or to describe all coughs with a genetic predisposition. The term atopic is therefore inappropriate for atopic cough.

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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 “asthma”. We continue to suggest that such insistence runs counter to the progress of asthma research subjects; areas C+D represent probable asthma, area B represents definite atopic cough variant asthma, area A represents definite non-asthmatic cough, and area D represents probable atopic cough variant asthma. Thus, as the efficacy of bronchodilator therapy is not sufficient, 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 different mechanisms.

The data presented in our paper 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. 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 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


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