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. 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. 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. 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. 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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Reference

Asthma guidelines

We read with interest the new British guideline on the management of asthma published recently as a supplement to Thorax. 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 reviews 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 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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Reference

Authors’ reply
In their letter van der Wouden and colleagues question the issue of selective withdrawal of subjects excluded from 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 diagnosis for asthma: 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 limited and, if present, 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 PPC: 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). The bronchoscopy, which was performed with the patient sitting, was complicated by a fall in SaO2, to 85%, which was only partially corrected by supplemental oxygen.

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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 asthma in the United Kingdom, with most patients suffering from asthma because of a continuous form of rhinovirus infection from adenovirus because of a myriad of respiratory viruses. In their recent paper published in Thorax we read with interest the case report by Newton et al. describing platypnoea and orthodeoxia in a patient with pneumocystis pneumonia (PCP). The bronchoscopy, which was performed with the patient sitting, was complicated by a fall in SaO2, to 85%, which was only partially corrected by supplemental oxygen.

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References
and cough sensitivity and bronchomotor tone manifestations of eosinophilic airway inflammation. It is very important to recognise that increased cough and areas B + D as cough variant asthma represent eosinophilic bronchitis without asthma based on bronchial responsiveness of the cough to corticosteroids. 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 responsiveness and efficacy of bronchodilators. Only area C represents definite cough variant asthma for the purposes of selecting clinical research subjects; areas A + D represent probable cough variant asthma for general clinical practice, and areas B + C represent possible cough variant asthma. In addition, areas B + D represent eosinophilic bronchitis without asthma.2

Worldwide problems regarding the diagnosis of cough variant asthma, atopic cough, and eosinophilic bronchitis without asthma are as follows: (1) many researchers have recognised that cough variant asthma is not synonymous with atopic cough, and areas B + C represent possible cough variant asthma. In addition, areas B + D represent eosinophilic bronchitis without asthma.2

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 58 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; bronchodilator treatment was not effective, leading to a probable diagnosis of atopic cough. Although some investigators believe that BHR is the key criterion for the 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.

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