LETTERS TO THE EDITOR

COPD Guidelines

The BTS guidelines on COPD1 exhort GPs to diagnose COPD early, preferably in the pre-symptomatic stage. In effect what is being promoted is “secondary prevention” (screening) through the use of spirometry. In most early cases this would involve two readings at an interval of four to five years. The intended outcome is that the patient stops smoking.

At first sight this appears a sensible proposal (although not one included in the ATS’ or ERS’ guidelines). However, the same outcome can be achieved without the screening process, so before GPs adopt this recommendation wholesale it is worth noting that there is no direct evidence to support screening.

Literature abounds on the costs of persuading smokers to stop smoking. If by screening for COPD one could achieve a lower cost per quitter amongst susceptible smokers, then an argument could be made on cost-efficiency grounds. But as simple advice from a GP is one of the most cost effective interventions in health care, this is unlikely.1

One is therefore left to consider the incremental cost rate achieved by superimposing screening and advice on a programme of advice alone. Most smokers are already aware of the risk of lung cancer and ischaemic heart disease. It is not known what difference the distant threat of COPD makes to smoker quit rates.

If the costs of screening are distributed solely to the increment of true positives who stop with the enhanced programme one arrives at a true marginal cost of this preventive measure. The costs to be considered should include the direct health service costs of equipment and training, and the opportunity cost to society of the time spent by GPs and nurses.

Screening is intended to improve well being (the ethic of “maximising public welfare”), yet there is the paradox of identifying conditions (in order to reduce perceived well being) in pre-symptomatic individuals when the majority derive no benefit. Intangible costs become relevant as do indirect costs (with well people often having to miss work for screening). Intangibles include the anxiety created by screening and the even greater anxiety in the true positives who then fail to stop smoking. One also has to decide what to tell the true negatives (those shown not to be at risk of COPD).

A broader cost-utility analysis would permit comparisons with the benefits of other screening interventions in primary care. Cervical cytology, mammography, and newer technologies such as colorectal cancer screening compete for decision monies. Lieberman offers a model for measuring cost effectiveness of colorectal cancer screening programmes.1 The sensitivity analyses considered are patient compliance, varying costs of procedures, frequency of surveillance, costs of downstream care, cancer detection rate, and cancer prevention rate. The parallels for COPD screening might include patient response rate to invitation, varying costs of equipment and staff time, frequency of surveillance, costs of treating diagnosed COPD, COPD detection rate, and smoker quit rates. A full analysis might also consider the discount rate for costs and benefits over time. In a programme that detects disease 10 years before it becomes symptomatic, an accepted discount rate of 6% per annum compounds considerably.

Spirometry is invaluable in the diagnosis and management of COPD. GPs should welcome the guidelines but must consider the opportunity costs to their activities before embarking on screening pre-symptomatic patients.

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AUTHORS’ REPLY

With regard to the points raised by Dr Perry we would comment that there may be little direct evidence about screening for early COPD, but the indirect evidence was sufficient to persuade the guidelines committee. The natural history of COPD, elegantly described by Fletcher and Peto,1 is that a proportion of cigarette smokers (15–20%) have an annual loss of FEV1 that is larger than for non-smokers. Initially such losses, while measurable in large groups, are too small to be detected in the individual. After 20–40 years the cumulative excess loss of FEV1 is large enough for an individual’s FEV1 to be below the lower limit of the predicted normal value and thus be detectable. Thereafter, with continuing smoking, the subject will progress from mild impairment through moderate to severe impairment. The only proven treatment that slows this inevitable decline in function is stopping smoking.2

Most patients only present to the health service with moderate to severe disease at a stage when cure is not possible, often lost, and lifestyle limited. Since the damage is irreparable, it is logical to consider prevention at an earlier stage and preferably before symptoms develop.

Dr Perry is correct to point out that cost effectiveness studies should consider the wider picture, but it is important not to extrapolate from studies based on different principles. Simple advice from a GP is effective in a very small proportion of subjects (2–5%) and is cost effective only because it is so cheap. The health benefit of adding nicotine patches and nurse counselor advice,3 which increases the quit rate significantly (21%), are both clear and desirable, but the extra treatment costs have left doubt about the cost effectiveness of such additional work. However, studies in unselected smokers will include those not susceptible to COPD, cardiac disease, or lung cancer (perhaps half of all smokers).4 The extra costs of treating all patients (20% of the population) when only some can benefit makes the cost effectiveness equation less favourable. It also presupposes that non-symptomatic patients will agree to come and see their GP. Targeting smokers in their 50s with a reduced FEV1 (who amount to less than 1% of the population) would result in a very different calculation. Now only 15% of smokers in a defined age range are being targeted for non-smoking advice, which reduces the cost implication of the more effective treatment and, moreover, the health benefit is greater because every person who gives up smoking is being prevented from developing symptomatic COPD, with its costs to both the individual and the health service. A formal study should be done to confirm such estimates, but progress cannot always wait for absolute confirmation, particularly when the health benefits to this defined subgroup are so clear cut.

Dr Perry is concerned at the adverse effects of screening when many will derive no benefit. Those with a normal FEV1 should still be advised to quit on the grounds of the risk from heart disease and lung cancer, but on cost effectiveness grounds they may not qualify for more intense help. Their anxiety levels are unlikely to be raised by this any more than by the frequent publicity about smoking in the media.

Spirometry is not an expensive procedure, costing less than a chest radiograph and probably less than a fasting lipid measurement. Radiographs are of limited value in COPD yet are often requested, whereas measurement of the FEV1 informs diagnosis, treatment and prognosis and has been grossly underused in both hospital and general practice. Finally, it should be pointed out that a recent paper in the BMJ describing the prospective Renfrewshire population5 showed that FEV1 was a stronger predictor of premature death than serum cholesterol measurements and as strong a predictor as cholesterol when only heart disease was considered. It concluded with the recommendation that anti-smoking activity be targeted on those with a low FEV1.

M G PEARSON
P M A CALVERLEY
COPD Guidelines Committee

COPD Guidelines

The British Thoracic Society is to be congratulated on the new COPD guidelines which are clear and will prove a major boon to patients and their carers in primary care.

Spirometry lies at the heart of these guidelines, perhaps most importantly in screening for asymptomatic cases amongst smokers. After all, there is no cure. In our practice we perform such screening on smokers over 40 years old. To be effective, screening must be largely opportunistic. We own both a simple hand vane spirometer costing around £300 which is largely opportunistic. We own both a simple and cheap one and it is ideal for screening by general practitioners and respiratory nurses; it is quick and simple to use. The recommended expensive variety is wholly unsuitable for this purpose as it takes at least 10 minutes to set up and calibrate then to print out the result. We use it only to check those with abnormal results on screening, as well as in “asthma/COPD” clinics where it is set up and used repeatedly.

It is unrealistic to expect most practices to purchase two types of spirometer and our experience would suggest that the cheap spirometers are preferable for routine primary care use with abnormal results being checked by an open access spirometry service. Furthermore, such a service is no substitute for performing spirometric measurements in-house. If cheap vane spirometers are condemned, opportunities for preventing this devastating disorder will be lost.

RUPERT JONES
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Travel by air for patients with COPD

Publication of the long awaited BTS guidelines on the management of COPD has been welcomed by all concerned and will go a long way towards providing a practical guidance on management of patients with COPD.

However, I would like to comment on the section on travel (page S14). This informs us that aircraft are pressurised to the equivalent of 900–2400 metres above sea level and that a Pao2 of less than 6.7 kPa should be considered as a contraindication to air travel.

This is clearly welcome to help all clinicians dealing with such patients. I would, however, like to take issue with the statement advising the administration of hydrocortisone in conjunction with streptokinase to reduce the risk of allergic complications.

M G PEARSON
P M A CALVERLEY
COPD Guidelines Committee


Guidelines on pulmonary embolism

The new guidelines for the practical approach to management of pulmonary embolism (PE) are clearly welcome to help all clinicians dealing with such patients. I would, however, like to take issue with the statement advising the administration of hydrocortisone in conjunction with streptokinase to reduce the risk of allergic complications.

Allergic reactions are a well recognised adverse effect of intravenous streptokinase treatment and are probably due to immediate hypersensitivity reactions mediated by IgE antibodies to streptokinase. The incidence of allergic reactions is low (1.7–18%) and was only seen in 3.6% of patients entered into the Third International Study of Infarct Survival (ISIS-3) trial. Indeed, only 0.3% of patients in ISIS-3 had allergic reactions causing “persistent symptoms”.

Patients at risk of allergic and anaphylactic reactions can be identified rapidly by intradermal streptokinase skin testing, which correlates with elevated levels of IgE to streptokinase. This test will give results in approximately 15 minutes but is not widely utilised. At risk patients include those who have received prior streptokinase treatment, including those who have had previous intradermal streptokinase skin tests. The intradermal skin test will not, however, detect patients with IgG antibodies to streptokinase who are at risk of delayed reactions and may not have effective thrombolysis due to IgG neutralising antibodies.

Antihistamines and hydrocortisone may help to reduce the effects of immediate hypersensitivity reactions, but steroids are not routinely administered in the major cardiovascular thrombolytic trials or in most UK coronary care units. As patients with pulmonary embolism are at no greater risk for allergic reactions to streptokinase, there does not seem to be good evidence for the statement in the guidelines. It may be more appropriate to consider alternative thrombolysis if there is genuine concern regarding allergic reactions in individual patients.

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M G PEARSON
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COPD Guidelines Committee


Malignant neoplasms in pulmonary sarcoidosis

As its authors intended—by limiting their analysis of the association to instances in which recognition of malignancy succeeded a diagnosis of sarcoidosis by more than one year—the study by Seersholm et al rigorously tested the hypothesis proposed by
BOOK REVIEW


At $165.00 the book is more expensive than its contemporaries, but it does cover a wider range of topics in greater depth. It also includes numerous relevant illustrations. Overall, this short text is an excellent source of reference and will benefit all health professionals who care for patients with cystic fibrosis.—HCR

NOTICES

MICRO 98

A joint meeting of Interasma 98 and the VIth Congress of the Moroccan Society of Allergology and Clinical Immunology will take place in Marrakech on 8–11 October 1998. Further information can be obtained from Professor M Bartal, SMAIC, B.P. 1754, Derb Ghallef, Casablanca, Morocco. Fax (212 2) 222355/296850.

Pharmacology of Asthma

A course on the pharmacology of asthma will be held at the Imperial College School of Medicine at the National Heart & Lung Institute in collaboration with the Royal Brompton Hospital on 23–26 November 1998. For further information please contact the Postgraduate Education Centre, National Heart & Lung Institute, Dovehouse Street, London SW3 6LY, UK. Tel: 0171 331 8172. Fax: 0171 376 3442.

AUTHORS’ REPLY

Dr Reich raises an interesting hypothesis that previous malignancy and, in particular, treatment with chemotherapy may cause sarcoidosis. In our study, however, we found no cases of malignancy prior to the diagnosis of sarcoidosis, which would have been the case if the hypothesis is correct. A larger study of the risk of sarcoidosis following chemotherapy may reveal an association but requires national sets of sarcoidosis rates in order to calculate the expected number of cases. Unfortunately a national registry of the incidence of sarcoidosis is not available.

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