LETTERS TO THE EDITOR

COPD Guidelines

The BTS guidelines on COPD™ 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 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 abound 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-effectiveness 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 stop 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 the different threat of COPD makes to smoker quit rates.

If the costs of screening are distributed solely to the increment of true positives who then fail to stop smoking. One is therefore left to consider the incremental stop 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 the different threat of COPD makes to smoker quit rates.

The costs of screening are distributed solely to those who stop and are therefore reducing 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.² 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 rates 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.

Spiriometry 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,³ 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 of smokers, 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 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.

Most patients only present to the health service with moderate to severe disease at a stage when it is either too late, or 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,⁴ 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). The extra costs of treating all smokers (perhaps half of all smokers) 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 the 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 if the 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 benign pre-test probability will 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 intensive help. Their anxiety levels are unlikely to be raised by this any more than by the frequent publicity about smoking in the media.

Spiriometry 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

REFERENCES

COPD Guidelines

The British Thoracic Society is to be congratulated on the new COPD guidelines1 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 spirometer costing around £300 which states only FEV1, and a more accurate device costing £1400. This produces a full range of results, including flow rates, and quality checks. The guidelines state that a volume/time plot is mandatory, thus condemning cheaper varieties.

Both of these devices have their place. The cheap one 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 let alone 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 9.3 kPa should be considered as a relative contraindication to air travel. A PaO2 of 6.7 kPa at sea level will equate to a PaO2 of 4.6 kPa at an altitude of 2400 metres using Gong’s nomogram, and this is unacceptable low.

I wonder if you would agree with me that this recommendation should specify that a predicted PaO2 of less than 9.3 kPa (not at sea level) of less than 6.7 kPa should be considered as a contraindication to travel by air. Using Gong’s nomogram a PaO2 of 6.7 kPa at an altitude of 2400 metres is equal to a PaO2 of 9.3 kPa at sea level. Therefore, a PaO2 of less than 9.3 kPa, not less than 6.7 kPa, measured at sea level should be regarded as a contraindication to air travel unless supplemental in-flight oxygen is provided.

In practice, if the PaO2 at sea level is less than 9.3 kPa, the flow rate of oxygen that would increase the PaO2 to 9.3 kPa or above should be determined and recommended for administration during flight.

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Authors’ reply

The problem of knowing whether it is safe for any individual to travel by air is difficult. Breathing air with a reduced oxygen content should produce an additional degree of hypoxia, which might be expected to carry a risk of either cardiac or respiratory difficulty. However, there are few reports of patients with COPD encountering specific difficulties other than the well documented (but still uncommon) risk of pneumothorax. The prediction nomogram described by Gong was derived from experiments in a laboratory on the ground, maintaining 15.1% oxygen and may not compare with actuality under hypobaric conditions. Although the American guidelines1 do recommend pre-flight assessment, they specifically do not recommend the Gong nomogram and avoid stating any specific levels of hypoxia as of concern. With marked hypoxia (6.7 kPa) there must be concern that supplemental oxygen is likely to be of benefit, hence the recommendation—albeit one for which there is no strong evidence. With mild hypoxia there is suspicion but no evidence. This is an area where further research would be helpful to clarify the benefits and risks of travel with and without supplementary oxygen.

Many patients with COPD can and do travel apparently safely by air. Until there are more substantive data to the contrary, we must be careful not to place any additional constraints on COPD patients over and above those already present due to their limited exercise tolerance.

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.1 Indeed, only 0.3% of patients in ISIS-3 had allergic reactions causing “persistent symptoms”.2

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, predict patients with IgG antibodies to streptokinase who are at risk of delayed reactions and may not have effective thrombolysis due to IgG neutralising antibodies.

Aspirin 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 agents if there is genuine concern regarding allergic reactions in individual patients.

CHRISTOPHER W H DAVIES
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Authors’ reply

Although indeed unusual, an allergic reaction to streptokinase in a patient with massive pulmonary embolism who, by definition, is already critically ill and hypotensive (unlike most myocardial infarction patients) could be disastrous, which is our reason for advocating the routine administration of hydrocortisone. This is not a problem with other thrombolytics; some hospitals, including mine, have already agreed that this advantage of alteplase in massive pulmonary embolism justifies its much greater cost. Our third suggestion, urokinase, is both safe and cheap, but although many pharmacies stock it for unblocking central venous catheters, few have the much higher dose formulation required for acute pulmonary embolism.

ANDREW MILLER
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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

MICRO 98, the International Microscopy Conference and Exhibition organised by the Royal Microscopical Society, will be held on 7–9 July 1998 at the Novotel, Hammersmith, London. For further details please contact Allison Winton, RMS, 37/38 St Clements, Oxford OX4 1AJ, UK. Telephone: +44 (0)1865 248768; Fax: (0)1865 791237; emailinfo@rms.org.uk; web page http://www.rms.org.uk

INTERASMA 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 Oulad Hadda, Casablanca, Morocco. Fax (212 2) 722355/296850.

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.
Malignant neoplasms in pulmonary sarcoidosis

JEROME M REICH

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