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 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 effective grounds. But as simple advice from a GP is one of the most cost effective interventions in health care, this is unlikely.

One is therefore left to consider the incremental cost per quitter achieved by superimposing screening and advice on a program 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 additional 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 preventative 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 the one in the population (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 develop monies.

Lieberman offers a model for measuring cost effectiveness of colorectal cancer screening programmes. The sensitivity analyses considered 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.

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, 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 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 they are forced to stop smoking when and if they wish. The economic benefits of treating all smokers (30% 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 certainty, particularly as very small 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 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.

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 population showed that FEV1 was a stronger predictor of premature death than serum cholesterol measurements and as a strong 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 among 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 vane spirometer costing around £300 which states only FEV1, and FVC values, and a more accurate device costing £1400. This produces a full range of results, interpreted by quality checks. The guidelines state that a volume/time plot is mandatory, thus condemning cheaper varieties.

Both of these devices have their uses. 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.

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

Although 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 of administration required for acute pulmonary embolism.

ANDREW MILLER
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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 Steersholm et al rigorously tested the hypothesis proposed by 1 British Thoracic Society, Standards of Care Committee. Suspected acute pulmonary embolism: a practical approach. Thorax 1997;52(Suppl 3):53.
BOOK REVIEW


At the title suggests, this volume covers the care of inpatients with cystic fibrosis, but it is not restricted to this alone. It provides a clear account of the management of all aspects of cystic fibrosis, from initial diagnosis to terminal care, with the emphasis on the practical approach. The book consists of a collection of chapters by different cystic fibrosis physicians, mostly paediatric. All the common complications of cystic fibrosis are discussed in depth and from the perspective of each author. Optimal treatment regimens are suggested in the text, supported by objective data where possible. Some of the topics overlap, such as the management of pneumothorax which is discussed by several authors including cystic fibrosis physicians and a thoracic surgeon. The text is therefore more a source of reference to be dipped into than to be read from cover to cover. The book is aimed at physicians caring for patients with cystic fibrosis and does have a paediatric slant. Certain sections would also be of interest to nurses, dieticians, pharmacists, social workers, and other health care professionals working with patients with cystic fibrosis.

The volume is almost pocket sized and contains many handy investigation and management tables to aid the busy physician. Highlights include an excellent section on pharmacokinetics which clearly explains the principles of treatment in cystic fibrosis and attempts to tease out evidence-based medicine from cystic fibrosis folklore in dosing and drug monitoring. The chapter on cystic fibrosis related diabetes mellitus (CFRDM) is also very good, explaining how the management of CFRDM contrasts with classical treatments for diabetes mellitus.

The book is American in style and content, most evident in the transplant chapter where current practice differs from that in the UK. The volume also includes a chapter presenting a personal view of one physician to the hospitalised patient with cystic fibrosis which seems out of keeping with the rest of the text. 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; email info@rms.org.uk; web page http://www.rms.org.uk

INTERASMA 98

A joint meeting of Interasma 98 and the Vth 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.
Malignant neoplasms in pulmonary sarcoidosis

JEROME M REICH

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