primary care trust have a named person to take a lead for children and every strategic health authority already has such a person. The latter will have the responsibility for monitoring the performance of primary care and provider trusts.

Who then should be driving these important changes? They must be locally and professionally driven. There is a real commitment by the professions to improve services. The Royal College of Paediatrics and Child Health can help by providing leadership and disseminating examples of good practice.[6] Its recent document, *Old Problems, New Solutions*, is proving helpful in thinking about new ways of working.

Paediatricians must work closely with the physicians over transitional arrangements, with general practitioners to smooth the interface between primary and secondary care, and perhaps most importantly with nursing.

It is 25 years since the last major report on the care of children. The Court report, *Fit for the Future*,[7] proved effective in slowly bringing up the standard of services, and most of its recommendations were eventually implemented. Even the concept of the specialist general practitioner paediatrician, ridiculed at the time, may be about to have its day. There is little that is new in the current reports, but if the best ideas and services could become universal then Cinderella would truly be ready for the ball. However, she can't wait another 25 years to fill her dance card.

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Managing Barrett's oesophagus

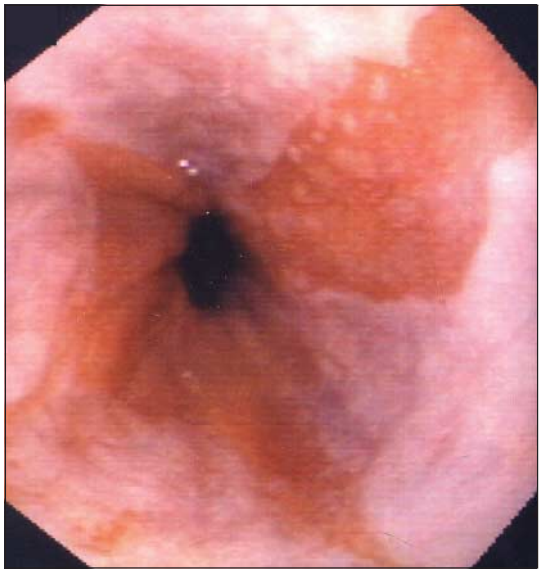
Decisions have to be based on indecisive data

In Barrett's oesophagus the stratified squamous epithelium that normally lines the distal oesophagus is replaced by an abnormal columnar epithelium that has intestinal features.[1] The abnormal epithelium (called specialised intestinal metaplasia) usually shows evidence of DNA damage that predisposes to malignancy,[2] and most oesophageal adenocarcinomas seem to arise from this metaplastic tissue.[3] Barrett's oesophagus affects mainly white men, among whom the incidence of oesophageal adenocarcinoma has more than quadrupled over the past few decades.[4] The quandary is to know what to do to prevent Barrett's oesophagus from turning into oesophageal cancer.

Barrett's oesophagus develops as a consequence of chronic gastro-oesophageal reflux disease (GORD), and is usually discovered during endoscopy performed to evaluate the symptoms of reflux disease. Endoscopists recognise Barrett's oesophagus because the dull red of the metaplastic columnar epithelium contrasts sharply with the pale glossy normal squamous lining (figure).

Barrett's oesophagus is classified as long segment or short segment, depending on whether or not the specialised intestinal metaplasia extends 3 cm or more above the gastro-oesophageal junction.[5] Among patients who have endoscopy for symptoms of reflux, long segment Barrett's oesophagus is found in 3-5% and short segment disease in 10-15%.[1] Although it is not clear whether long and short segment Barrett's oesophagus have the same pathogenesis and risk for malignancy, the two conditions are managed similarly.

Barrett's oesophagus is a strong risk factor for oesophageal adenocarcinoma, a lethal malignancy; yet several studies have found that survival for patients with Barrett's oesophagus does not differ significantly from that for matched individuals in the general population.[6] This seeming paradox may be explained with the low absolute (rather than the high relative) incidence of cancer in Barrett's oesophagus. Modern data indicate that patients with Barrett's oesophagus



Long segment Barrett's oesophagus. The dull red of the metaplastic columnar epithelium contrasts with the pale, glossy appearance of the normal squamous lining

develop oesophageal adenocarcinomas at the rate of 0.5% per year, a rate that is more than 30-fold higher than that of the general population but low in absolute terms.[7] Studies of survival in patients with Barrett's oesophagus have been done predominantly in older men, for whom the risk of death from common lethal disorders (myocardial infarction, stroke) far exceeds their 0.5% annual risk of oesophageal adenocarcinoma.[8] A long term study of young patients with Barrett's oesophagus might show that the condition shortens life, but no such study has been published.

Several management strategies have been proposed to reduce mortality from cancer in Barrett's oesophagus. These include *(a)* normalisation (rather than mere reduction) of oesophageal acid exposure with antisecretory drugs, often in doses and combinations beyond those required to heal the symptoms and signs of reflux disease; *(b)* antireflux surgery; *(c)* endoscopic ablation of the metaplastic epithelium; *(d)* non-steroidal anti-inflammatory drugs that inhibit cyclo-oxygenase and its effects on cellular proliferation; and *(e)* regular endoscopic surveillance.[1 9 10] Although each strategy has a plausible rationale and some indirect evidence to support it, none has proved to reduce deaths from cancer in Barrett's oesophagus. Furthermore, each entails expense, inconvenience, and variable risk. Among the preventive strategies for cancer, only regular endoscopic surveillance has been recommended for routine clinical use by several medical societies, including the American College of Gastroenterology.[1 10]

When assessing cancer prevention strategies for Barrett's oesophagus, doctors should consider the implications of the low absolute risk of developing oesophageal cancer. Assume that there is a highly effective treatment for Barrett's oesophagus that will reduce the risk of cancer development by half—from 0.50% to 0.25% per year. This represents an absolute risk reduction (ARR) of 0.25%. Thus the number needed to treat (NNT) to prevent one case of cancer in one year is 400 (NNT=1/ARR; 1/0.0025=400). Thus, even if there were a highly effective cancer preventive treatment for Barrett's oesophagus, 400 patients would need to be treated to prevent one case of cancer in one year. Such a large number can be acceptable if the treatment is reasonably inexpensive, convenient, and safe.

Regular endoscopic surveillance is recommended for patients with Barrett's oesophagus despite the high cost and inconvenience and the lack of proof that it prolongs survival. A randomised trial to establish the efficacy of surveillance would require dauntingly large numbers of patients and length of follow up, and the results of such a study are unlikely to be available in the near future. Indirect evidence that surveillance is beneficial comes from observational studies, showing that cancers discovered during surveillance are less advanced and associated with longer survivals than those detected during endoscopies performed for evaluating cancer symptoms.[11] Such studies are not definitive because they are highly susceptible to biases that can inflate the benefits of surveillance by including biases of selection, healthy volunteers, lead time, and length time.

Computer models too have implied that endoscopic surveillance can be beneficial,[12 13] but such models also are not definitive. Models provide a range of possible outcomes that vary with changes in baseline assumptions and with estimates of what a healthcare provider is willing to pay for a good result. In one Markov model that assumed an annual cancer incidence rate of 0.4%, endoscopic surveillance performed every five years was the preferred strategy for patients with Barrett's oesophagus, costing $98 000 (£62 000; €92 000) per quality adjusted life year gained.[12]

Thus both observational studies and computer models indicate that surveillance can reduce mortality from cancer in Barrett's oesophagus, but at considerable expense. Surveillance is clearly associated with risks, including complications from both endoscopy and the invasive procedures used to treat lesions found by endoscopy, but no study has shown an overall survival disadvantage for patients in surveillance programmes.

In this murky situation, where most of the indirect evidence implies that surveillance is beneficial, I prefer to err by performing unnecessary surveillance rather than missing curable oesophageal neoplasms. Therefore, I support the strategy recommended by the American College of Gastroenterology as follows[10]:

- Patients with Barrett's oesophagus should have regular surveillance endoscopy. Gastro-oesophageal reflux disease should be treated before surveillance to minimise confusion caused by inflammation in the interpretation of dysplasia.
- Patients who have had two consecutive endoscopies that show no dysplasia should undergo surveillance by endoscopy every three years.
- If dysplasia is noted the finding should be verified by consultation with another expert pathologist.
- Patients with verified low grade dysplasia after extensive biopsy sampling should have yearly surveillance endoscopy
- For patients found to have high grade dysplasia another endoscopy should be performed with extensive biopsy sampling (especially from areas with mucosal irregularity) to look for invasive cancer, and the histology slides should be interpreted by an expert pathologist. If there is verified, multifocal high grade dysplasia, intervention (oesophagectomy) may be considered.

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Screening for cancer with computed tomography

Advising patients is difficult given the lack of evidence

Whole body screening with computed tomography is the focus of a major advertising campaign in the United States. Enticing testimonials on billboards and radio spots urge the public to use this technology, implying that there is much to gain and little to lose. How should primary care doctors advise their patients?

In one sense screening with computed tomography has much to offer. As part of a study conducted by the National Institutes of Health, our centre has used computed tomography to screen for lung cancer for the past four years and has identified 56 lung cancers. Fully 62% of the non-small cell cancers were stage IA.[1] In the absence of screening, only 15-20% of lung cancers present at stage IA. Five year survival for stage I lung cancers, which is about 60-70%, is higher than for cancers diagnosed at more advanced stages. There is little doubt that computed tomography is more sensitive than chest *x* ray in detecting small, early stage lung cancers. We found two cancers measuring only 3 mm in diameter.

Recognising that we found 56 patients with lung cancer, one could ask why screening should not be advocated. Why wait until patients develop symptoms and later stage disease? Screening could potentially save hundreds of thousands of lives in just a few years. Several uncertainties, however, make it premature to advocate screening on a large scale with computed tomography.

Some lung cancers may progress too rapidly. Although computed tomography certainly achieves earlier detection, biological destiny may render this value moot. Angiogenesis occurs at 1-2 mm for many tumours,[2–5] and we do not know how early metastasis occurs.

Other lung cancers may progress too slowly. Over diagnosis of cancers that pose little or no clinical threat to the patient (pseudo disease) may be a confounding factor. We are finding more early stage lung cancers, but the more pivotal question is whether we will change the incidence of advanced stage tumours. If, for example, screening detects cancer in the same proportions among smokers and never smokers, it may be detecting lesions that patients would die with rather than from.[6]

The false positive rate of screening may be too high. In our series, over 70% of participants had a false positive finding for lung cancer. Fully 98% of uncalcified lung nodules were benign. There are more than 90 million current and past smokers in the United States. Extrapolating our findings to this high risk population indicates that screening would identify more than 180 million uncalcified, radiologically indeterminate nodules.

Investigating lesions detected at screening may be harmful. The mortality associated with surgery for benign nodules may offset the gains in disease specific mortality achieved by screening. Multicentre studies in the United States and Europe show that about 50% of lung nodules removed at surgery are benign,[7 8] but wedge resections of lung nodules (benign or malignant) carry a mortality of 3.8% at community hospitals in the United States.[9] Radiation exposure associated with follow up examinations might induce more deaths due to cancer than are prevented. The first duty of medicine is to do no harm.

The cost of screening may be too high. By some estimates, screening would cost $116 300 (£74 456; €107 002) to $2.3m per quality adjusted life year gained.[10]

High risk patients, the cohort most likely to benefit from screening, are at risk for comorbid illness. The benefits of early detection may be lost in smokers, who are arguably more likely to die from stroke, heart disease, or obstructive lung disease.

Whole body screening with computed tomography engages the same issues on a larger scale. In our cohort we found over 700 ancillary findings, including four renal cell carcinomas, three breast cancers, two lymphomas, two gastric tumours, one pheochromocytoma, and 114 abdominal aortic aneurysms.[1] However, most of these ancillary findings were falsely positive, the investigation of which adversely affected quality of life and resulted in unnecessary diagnostic and interventional procedures.

Although important scientific questions must be answered to know whether screening of the lung or the whole body with computed tomography results in more good than harm, it is unclear whether either the public or the marketers are willing to wait. A search of the internet will show hundreds of facilities offering screening with computed tomography from coast to coast.

Some of the best doctors in the world have sincere differences of opinion about the merits of such screening. This balance in opinion, which ethicists call equipoise, provides the ideal context for conducting a

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