75 Deaths in asthmatics prescribed home nebulisers

SIR,—Like Dr M R Sears and coworkers (21 February, p 477), we are concerned about the possible toxicity of high doses of bronchodilators delivered by nebulisers in patients with asthma who are also hypoxic and may be hypokalaemic.

We studied 10 patients with asthma, mean age 38.5 years, who were admitted to hospital with an acute exacerbation of their asthma. They were treated conventionally with intravenous and oral corticosteroids, nebulised salbutamol and ipratropium bromide, oxygen, and intravenous and oral theophylline if required. They were studied during the recovery period and on two consecutive days at 9 am, when salbutamol and ipratropium bromide nebuliser solutions were administered in random order using a Cirrus nebuliser driven by air at a flow rate of 8 l/min. Salbutamol was given in a total dose of 10 mg diluted in normal saline and ipratropium bromide in a total dose of 500 µg diluted in normal saline. Oxygen saturation was measured continuously using a Biox IIA ear oximeter, plasma potassium concentration was sampled, and continuous electrocardiographic recording was performed.

There was no evidence of a carryover effect, and we therefore compared the individual day effect, using Student's *t* test, to see if there was any significant difference from baseline. There was a non-significant maximum fall in the mean (SD) plasma potassium concentration of 0.08 (0.29) mmol/l with salbutamol from a baseline of 3.74 (0.35) mmol/l and a non-significant rise in plasma potassium concentration of 0.15 (0.30) mmol/l with ipratropium bromide from a baseline of 3.64 (0.17) mmol/l. There was a significant fall in oxygen saturation with salbutamol ($p < 0.002$) of 2.30 (1.64)% from a baseline of 94.7 (2.45)% and a fall with ipratropium bromide ($p < 0.005$) in oxygen saturation of 1.80 (1.55)% from a baseline of 94.1 (2.25)%. None of these changes in potassium or oxygen saturation were likely to be clinically important. The only cardiac arrhythmias during the four hour period either side of the nebulisation were ventricular ectopic beats. For all the patients there was a total of 24 ventricular ectopic beats before salbutamol and 14 after and with ipratropium bromide 16 before and six after.

Our study suggests that 10 mg of salbutamol or 500 µg of ipratropium bromide, delivered with an air driven nebuliser, has no clinically important effect on oxygen saturation, plasma potassium, or cardiac rhythm in patients recovering from asthmatic attacks.

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Asthma mortality: comparison between New Zealand and England

SIR,—In my letter of 3 January (p 53) I expressed doubts on the validity of a comparison of asthma mortality¹ in a New Zealand study² and an English one,³ which Dr M R Sears and Dr H H Rea have not convincingly resolved (7 March, p 646). I also postulated that annual mortality figures for all races in the 5-34 age group, such as those presented in an earlier article,⁴ might have exaggerated the severity of the recent "epidemic" of asthma deaths among caucasian subjects, since these figures included non-caucasians, in whom the death rate was known to be much higher; no fewer than 48%

of all deaths in the 5-34 age group in 1981-3 were of non-caucasians,² although they constituted only 10.9% of its population.

I used an incorrect figure of 15% for the non-caucasian population and also wrongly assumed that the proportion of non-caucasian deaths in the 5-34 age group had been similar each year from 1975 onwards to what it was in 1981-3. Correction of the first error actually strengthened my case, but the statement by Drs Sears and Rea that the non-caucasian death rate fell from 48% to 16% of the total death rate between 1981-3 and 1985 unexpectedly undermined my second assumption. The only possible explanation of this sudden change, seemingly statistically significant, is either that the treatment of non-caucasians improved dramatically over two to three years or that their asthma miraculously diminished in severity.

I agree unreservedly with Dr Sears and Dr Rea that inappropriate treatment and delays in seeking and receiving medical help are the most important contributory factors in deaths from asthma. That conclusion has no doubt been valid ever since the disease was first recognised, and there must be a more specific reason for the "epidemics" of asthma deaths in the United Kingdom and several other countries in the 1960s and in New Zealand in the 1980s. Such epidemics have not been recorded previously in the centuries old history of the disease, yet they have now occurred twice in only 20 years. Dr Sears and Dr Rea warn against assuming that the second New Zealand epidemic was a rerun of the 1960s epidemic, but the onus is on them to prove it was not. The time scales of the two epidemics were so similar that any other hypothesis is scarcely credible.

The 1960s epidemic in the United Kingdom followed the introduction of a symptomatically effective, patient controlled form of treatment (with pressurised isoprenaline inhalers), which is not intrinsically dangerous but which may have altered the pattern of medical supervision so that both patients and general practitioners underestimated the risk of a fatal outcome. That epidemic subsided almost certainly not because of restrictions placed on the supply of isoprenaline inhalers but because the alarm sparked off by a government warning on their supposed dangers resulted in a vast increase in prescriptions for oral corticosteroids and in the number of patients with acute asthma admitted to hospital.

In 1982, when the second epidemic in New Zealand was at its height, it was estimated that 6000 nebulisers for the delivery of high doses of bronchodilator aerosols in the home had been procured in the previous two years by private purchase or as gifts from charitable bodies.⁵ Bronchodilator solutions were available either without prescription or because general practitioners were reluctant to withhold them from patients with nebulisers. After nationwide publicity on the dangers and restrictions on the supply of bronchodilator solutions in late 1982 mortality from asthma began to fall and is now almost back to the pre-epidemic level, although still higher than that in other countries.

In their article on home nebulisers (21 February, p 477) Dr Sears and colleagues claim again that overreliance on a home nebuliser was a factor in, at most, 8% of fatal cases. An equally reassuring claim could probably have been made about pressurised isoprenaline inhalers in the 1960s. Although the temporarily uncontrolled availability of both forms of treatment may have been directly responsible for only a relatively small number of deaths, their very introduction and perceived efficacy could ipso facto have led to a diminished awareness of the risk of death from asthma and thus of delays in starting systemic corticosteroid therapy or admitting patients to hospital.

High dose β_2 agonist aerosols delivered by nebulisers are both effective and safe provided that treatment is carefully controlled and monitored and is not allowed to tempt patients to delay seeking medical help or doctors to delay in starting corticosteroid treatment.

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An uncompromising report on health visiting for the elderly

SIR,—It is refreshing to see the Health Visitors' Association confirming its responsibilities to the elderly as well as to children, and its recent report in conjunction with the British Geriatrics Society is to be welcomed.¹ Certain conclusions in this report, however, and Dr Simon Barley's comments on them (7 March, p 595) give cause for concern.

The benefit of health visitors' case finding or screening of the elderly population remains unproved.² A reduction in mortality has been claimed, but results from controlled trials are inconclusive. Vetter *et al* found a difference in the number of deaths between a screened group and only one of two control groups.³ Tulloch and Moore, on the other hand, found significantly more patients in their screened population who were free of medical disorders or suffering from only one condition at the end of a two year study.⁴ There were more patients with two disorders, however, in the study group, and this pattern was repeated for those with three or more conditions, but the differences were not significant. No significant improvement in functional state has been observed.

Though most investigators believe that their patients benefit from regular contact with a health visitor, such subjective impressions have not been confirmed by more sophisticated measurements.⁵ Screening does, however, seem to reduce the number of elderly patients admitted to institutions, and two studies have shown a reduction in the number of admissions to hospital and the number of hospital bed days utilised.^{4,6} Nevertheless, referrals for hospital clinic attendance may increase, and some uncontrolled screening programmes in the United States have reported increased inpatient referral rates. Prevention of admission to hospital seems to depend on the skills of each health worker in acting as a gatekeeper for inpatient resources.

Caution should be exercised before large scale funding is provided for an approach to health care whose benefit has not been proved. Opportunistic case finding may offer an equally effective but more economical means of health maintenance in the elderly in Britain, where most have at least yearly contact with the primary care team.

In the current restrictive economic climate it is insufficient even to prove that a health maintenance strategy is effective. Its value, as judged by the health workers and the population concerned, must be compared with alternative strategies. Many of the needs of the elderly revolve around surveillance, personal care tasks, and companionship. Instead of using highly trained staff to meet only one of these needs, would it not be better to meet all the needs of the elderly by providing more contact with a less comprehensively trained care attendant?

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SIR,—Dr Simon Barley's article illustrates several common misconceptions about health care of the elderly.

Firstly, he hints that it is unthinkable that health visitors should stop doing things that they already undertake. In the same edition of the *BMJ*, however, Ms Emily Grundy (p 626) shows that health visitors have already altered their practice. Between 1976 and 1984 they reduced their visits to the over 75s by 15%, thus opting to neglect the group with the most severe health problems. In the light of this shameful fact it is essential that they should reconsider their current practice and how they will allocate their time in the future.

Secondly, the concept of employing 1900 additional staff seems unrealistic to Dr Barley. On the other hand, Mrs Cumberlege's report advocated that practice nurses should be abolished, pointing out that continuing the current arrangements could necessitate the employment of an additional 20 000 nurses, mainly at the expense of central funds. General practitioners have argued for the retention of this facility, and the government seems willing to continue to foot the bill. So money and manpower can be made available for some actions but not for others.

The care of the elderly, both in the community and in hospital, is expensive in time, money, and manpower. If we are to treat elderly people as they deserve and need we must face the financial consequences. There will be serious humanitarian consequences if we fail to fulfil our duties. The degree of civilisation of a society (or profession) may be judged by its attitudes to the care of the elderly.

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Orchiectomy versus oestrogen for prostatic cancer: cardiovascular effects

SIR,—With regard to Dr T W Meade's comments (11 October 1986, p 953), the last 20 patients in the randomised study were evaluated with regard to activators and inhibitors of blood coagulation and fibrinolysis.¹ Before treatment the patients with prostatic carcinoma already had increased concentrations of fibrinogen and factor VIIIc compared with healthy controls of the same age. During oestrogen treatment factor VII concentration rose significantly ($p<0.001$) and the antithrombin III concentration decreased significantly ($p<0.001$). With regard to fibrinolysis, plasminogen concentration increased and that of urokinase inhibitor fell ($p<0.001$) after treatment with oestrogen was started.

Thus patients with prostatic cancer receiving oestrogen treatment have increased concentrations of factor VII, factor VIIIc, and fibrinogen. All these changes are identical with the findings that predicted death in patients with ischaemic heart disease in the prospective Northwick Park heart

study.² In addition, the patients' concentrations of antithrombin III decrease and the fibrinolytic capacity seems to increase during oestrogen treatment. Fibrinolysis was also activated in many of the patients who received oestrogen, as judged by increased concentrations of a fibrinogen degradation end product, the so called D dimer, during treatment.

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Barrett's oesophagus

SIR,—Dr R C Heading (21 February, p 461) cites many references that fail to explain the true nature of this condition. He and other authors regard the anomaly as being acquired and refer to such things as short oesophagus, gastric lined lower oesophagus, Barrett metaplasia, and heterotopic gastric cells.

The title Barrett's oesophagus is a misnomer, and Mr Barrett laid no claim to the eponym but described a chronic ulcer in the lower gullet, which is lined with columnar epithelium.¹ A clear distinction between this and superficial ulceration associated with reflux oesophagitis has now been established.

Before accepting a meaningless eponym we should find a name that relates to development and anatomy and thus explains the true nature of the anomaly and its origin. The development of the oesophagus is complex but has two remarkable features²: firstly, rapid elongation from a length of 1 mm in the 5 mm (35 day) embryo to reach its full length at birth; and, secondly, the unique pattern of epithelial formation. Differentiation of the gut epithelium occurs in a regular pattern, which starts at the 15 mm stage and is well advanced in the 17 mm embryo. Vacuolation of the primitive endodermal tube starts around the 5th week (14 mm embryo), and the columnar lining cells are gradually replaced by squamous cells to become mature squamous cell epithelium. This is a gradual change, however, which progresses from the cranial to the caudal end of the primitive gullet, so that at any given time a squamous lined proximal segment of variable length merges with a distal part lined by cells in various stages of metaplasia.

Usually, elongation and metaplasia are complete at birth, but if they are not a lower segment of variable length will retain a columnar lining into advanced pregnancy or until full term, and in some cases into adult life. If oesophagogastric function is competent there may be no symptoms, and as there is no need for endoscopy to be performed the condition may never be diagnosed. But incompetence often occurs in patients with this anomaly, and reflux oesophagitis occurs either in the very young or in later life, when the incompetence may be aggravated by hiatal herniation. Ulceration, with fibrosis and stricture formation, may occur in the squamous epithelium near the columnar lined segment and, depending on the length of this, may be near the cardia or as a high stricture at around 28 or 30 cm.

Large numbers of histological reports will continue to be written and the confusion over the terms used will persist unless the complex developmental processes are studied. Acquired changes do occur as a result of gastro-oesophageal reflux over the multipotential cells of the columnar lining, which is in itself a congenital abnormality.

It has been suggested that there is no sound support for the view that this condition is acquired and that many terms used to describe it should be discarded. Congenital columnar lined lower oesophagus would be a suitable name.

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AUTHOR'S REPLY—Is Barrett's oesophagus congenital or acquired? The debate has been going on for some years, with most of the reports of the 1950s and 1960s supporting a congenital origin for various reasons, including the embryology described by Mr Hutchison. Since then, however, the view that Barrett's oesophagus is an acquired lesion has gained favour, with the recognition that the columnar epithelium has features that indicate that it is metaplastic (that is, it is an epithelium that has replaced a different, earlier type) and that operative treatment that suppresses gastro-oesophageal reflux may allow reversion to a normal squamous oesophageal lining. One weakness of this theory is that there is no good explanation of why some patients with gastro-oesophageal reflux develop Barrett's oesophagus while others do not. There is perhaps an analogy in the gastric mucosa: it is not clear why some patients with gastritis develop extensive intestinal metaplasia, which does not occur in others.

The proposal that Barrett's oesophagus should be renamed congenital columnar lined lower oesophagus is logical if we are confident that the condition is indeed congenital. I believe, however, that most gastroenterologists do not share Mr Hutchinson's conviction and that the balance of evidence favours the view that Barrett's oesophagus is an acquired lesion.

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AIDS and intravenous drug use

SIR,—Dr J R Robertson and Carol Skidmore (28 February, p 571) produce a table relating the year of starting heroin addiction to the presence of antibody to the human immunodeficiency virus (HIV). They interpret the high prevalence among those who became addicted in 1982-3 as being due to some other factor or factors operating in these years and suggest that "a minor epidemic of heroin use resulted in extreme and damaging behaviour, giving rise to ideal conditions for transmission of virus."

We would interpret their data differently. In our study relating sexual lifestyle to HIV antibody state in homosexual men we found that those who had practised a gay lifestyle for five years or less had a significantly lower prevalence of HIV antibody than those who had practised for more than five years.¹ Our figures showed that of the 98 who were HIV seropositive, 91 (93%) had been homosexually active for more than five years, compared with 153 of 206 (74%) of those who were sero-