Whatever the reason for any discrepancies between the calculated APRs in Edinburgh and Belfast, there is clearly a real difference in the number of deaths due to gastric carcinoma, which accounts for 7.1% of all deaths in the Belfast study, compared with only 2.2% in the Edinburgh study. As all patients had vagotomy and drainage in the Belfast study compared with less than 13% in the Edinburgh study, the type of operation may explain the difference, though longer follow up may be required before increases are seen after gastrectomy.

None the less, of greater importance is the confirmation by Watt *et al* of the much larger contribution to excess mortality from disease having close associations with smoking (ischaemic heart disease, lung and oesophageal carcinoma, and chronic bronchitis).² One hundred and forty one of their patients died of these conditions while only 16 died of gastric carcinoma, emphasising the need to influence smoking habits if late mortality is to fall appreciably.

Finally, they confirm our finding of a statistically significant excess mortality from colorectal carcinoma following gastric surgery (APR 1.8, $p=0.01$). Although not a major contributor to excess mortality, the increased incidence of colorectal carcinoma is academically stimulating in the search for factors of aetiological importance in the development of such tumours.

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¹ McLean Ross AH, Smith MA, Anderson JR, Small WP. Late mortality after surgery for peptic ulcer. *N Engl J Med* 1982;307:519-22.

² Doll R, Peto R. Mortality in relation to smoking: 20 years observations on male British doctors. *Br Med J* 1976;iii:1525-36.

Pneumothorax in acute asthma

SIR,—Dr J Rees (26 May, p 1595) states that in acute asthma if there is any doubt as to the clinical diagnosis of a pneumothorax then a chest radiograph should be taken. It is standard teaching, however, that *all* patients with acute asthma presenting to hospital should have a chest radiograph to exclude a clinically unsuspected pneumothorax as a cause of deteriorating asthma. The following case illustrates the dangers of not adhering to such a policy.

A 69 year old man with chronic bronchitis and asthma presented with a one day history of increasing wheeze and dyspnoea. On examination of the chest there was a generalised soft expiratory wheeze but no signs of a pneumothorax. The peak expiratory flow rate was 110 litres/minute. A chest radiograph showed a right sided pneumothorax occupying a quarter of the right lung field. Before a chest drain was inserted the patient suddenly developed signs of a right sided tension pneumothorax and became cyanosed. He needed immediate insertion of a White needle into the right chest, which relieved his symptoms. A second chest radiograph was taken, which was identical to the first, and a chest drain inserted. The patient's dyspnoea progressively improved and he was discharged home a week later with a peak expiratory flow rate of 220 litres/minute.

This patient's pneumothorax was unusual in that for no apparent reason it suddenly developed into a life threatening tension

pneumothorax. Because a pneumothorax was clinically unsuspected in this patient, however, he would not, according to Dr Rees, have merited a chest radiograph. Since a pneumothorax can be difficult to diagnose clinically in patients with acute asthma its presence should *always* be suspected until excluded radiographically, and a chest radiograph taken in all such cases.

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* * Dr Rees replies below.—Ed, *BMJ*.

SIR,—I would certainly recommend a chest radiograph in a 69 year old man presenting with wheezing unless I were well acquainted with his recent condition. As well as the difficulties in diagnosing pneumothoraces, large airway obstruction may mimic asthma. When the patient is well known and has had frequent similar attacks, repeated radiographs on every visit to casualty are unnecessary. A recent study by Gershel *et al* of 371 children with their first attack found 21 abnormal chest radiographs but only one patient where abnormal findings were not predictable from the physical signs.¹ One patient had a pneumomediastinum, and there were no pneumothoraces. Foreign bodies are a possible cause of confusion in children, and an expiratory chest radiograph may then be useful (7 April, p 1074).

My point about chest radiographs in patients with acute asthma is that they are not an immediate priority on arrival at hospital unless there are suspicious findings. Assessment of severity and suitable treatment are the first essentials, and in my experience it is too common to see patients left waiting in radiography rooms before suitable treatment has been given.

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¹ Gershel SC, Goldman HS, Stein REK, Shelar SP, Ziprkowski M. The usefulness of chest radiographs in first asthma attacks. *N Engl J Med* 1983;309:336-9.

Asthma in children: diagnosis

SIR,—Dr John Price (2 June, p 1666) has once again emphasised that one of the important factors responsible for under-diagnosis and undertreatment is doctors' reluctance to use the word asthma. It remains a disease of incompletely elucidated and sometimes quite unknown aetiology. Nevertheless, there is good evidence of well defined allergic mechanisms in asthma,¹ particularly in children.²

That most asthmatic children show increased bronchial lability to non-allergic stimuli does not necessarily mean that the basic mechanism is also non-allergic. The development of bronchial hyperreactivity probably requires both a genetic predisposition and an injury to the bronchi. The injurious agent may be an allergen, pathogen, or otherwise. Bronchial hyperreactivity has been reported in cystic fibrosis,³ laryngotracheobronchitis,⁴ after surgery for tracheo-oesophageal fistula,⁵ in bronchopulmonary dysplasia,⁶ after foreign body aspiration,⁷ and in survivors of near drowning.⁸ Once the bronchi have become hyperreactive they may be triggered into

bronchospasm by specific as well as non-specific stimuli. There is also evidence that bronchial reactivity could be reduced by allergen avoidance^{9,10} and by inhaled sodium cromoglycate and steroids.¹⁰

Would it, therefore, not be reasonable scientifically as well as emotionally to diagnose the condition as "allergic airways disease" instead of asthma in children? This should remove the problem of non-acceptance, and it would help in better allergen avoidance and control.

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¹ Pepys J. Immunological mechanisms in asthma. In: Pepys J, Frankland AW, eds. *Disodium cromoglycate in allergic airways disease*. London: Butterworth, 1970:5-11.

² Morrison-Smith J, Disney ME, Williams JD, *et al*. Clinical significance of skin reactions to mite extracts in children with asthma. *Br Med J* 1969; ii:732-6.

³ Mellis CM, Levison H. Bronchial reactivity in cystic fibrosis. *Pediatrics* 1978;61:446-50.

⁴ Loughlin CM, Taussig LM. Pulmonary function in children with a history of laryngotracheobronchitis. *J Pediatr* 1979;94:365-9.

⁵ Milligan DWA, Levison H. Lung function in children following repair of tracheo-oesophageal fistula. *J Pediatr* 1979;95:24-7.

⁶ Smyth JA, Tabachnick E, Duncan WJ, *et al*. Pulmonary function and bronchial hyperreactivity in long term survivors of bronchopulmonary dysplasia. *Pediatrics* 1981;68:336-40.

⁷ Givan D, Scott P, Jeglum E, *et al*. Lung function and airway reactivity of children three to ten years after foreign body aspiration. *Am Rev Respir Dis* 1981; 123 (suppl):158.

⁸ Laughlin JJ, Eigen H. Pulmonary function abnormalities in survivors of near drowning. *J Pediatr* 1982;100:26-30.

⁹ Kerrebijn KF. Endogenous factors in childhood CNSLD. Methodological aspects in population studies. In: Orie NGM, van der Lende R, eds. *Bronchitis III*. Assen, The Netherlands: Royal Van Nostrand, 1970:38-48.

¹⁰ Platts-Mills TAE, Tovey ER, Mitchel EB, *et al*. Reduction of bronchial hyperreactivity during prolonged allergen avoidance. *Lancet* 1982;ii:675-8.

Nebuhaler versus nebuliser in children with acute asthma

SIR,—Dr M Freeland and Dr P P Van Asperen note that difficulty was experienced by patients in triggering the valve of the Nebuhaler at low airflow rates (23 June, p 1873). We have now changed the valve material so that the valve will open and close at lower airflow rates.

Testing Nebuhalers with the new valve shows that the valve will now open at flow-rates of 20-30 l/min, which compares favourably with the Nebuhalers used in the study—they opened at flowrates of about 40 l/min. We hope that the incorporation of this new valve into the Nebuhaler will avoid this problem in the future.

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Specificity of surfactant deficiency in neonatal respiratory disorders

SIR,—Dr D K James and colleagues (2 June, p 1635) report that phospholipid measurements on lung secretions from newborn infants failed to discriminate between those with hyaline membrane disease, transient tachypnoea, and congenital pneumonia. These observations were made on a total of 77 babies with 20 in the largest control group and between nine and 11 infants in the other groups. They conclude that pulmonary phospholipid measurements are unreliable in