Ward based patients may have even more variable glucose levels due to the intermittent nature of their caloric intake, in contrast to the continuous feeding that occurs commonly in critical care. The compounding effects of glucocorticoid treatment, frequently used in patients with COPD, have already been highlighted. Insulin infusions carry a risk of inadvertent hypoglycaemia which increases as blood glucose targets become more stringent. Indeed, in a major study of insulin therapy which aimed for blood glucose levels between 4.4 and 6.0 mmol/l, a 4.2% increase in the incidence of blood glucose levels of <2.2 mmol/l was identified, although these episodes apparently had no adverse sequelae.⁴ Finally, the implications for nursing workload are considerable; the development of computerised infusion control systems may help.

If the benefits of tight glycaemic control on mortality in patients with acute exacerbations of COPD are proved, the potential impact on the health care of patients with such a common disease is enormous. It will be revealing if the outcome is significantly influenced by what many would consider to be routine medical care that should be undertaken in all patients.

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REFERENCES

- 1 **Modan B**, Schor S, Shani M. Acute myocardial infarction. Prognostic value of white blood cell count and blood glucose level. *JAMA* 1975;**233**:266–7.
- 2 **Capes SE**, Hunt D, Malmberg K, *et al*. Stress hyperglycemia and prognosis of stroke in nondiabetic and diabetic patients: a systematic overview. *Stroke* 2001;**32**:2426–32.
- 3 **Yendamuri S**, Fulda GJ, Tinkoff GH. Admission hyperglycemia as a prognostic indicator in trauma. *J Trauma* 2003;**55**:3–8.
- 4 **Van den Berghe G**, Wouters P, Weekers F, *et al*. Intensive insulin therapy in the critically ill patients. *N Engl J Med* 2001;**345**:1359–67.
- 5 **McAlister FA**, Majumdar SR, Blitz S, *et al*. The relation between hyperglycemia and outcomes in 2,471 patients admitted to the hospital with community-acquired pneumonia. *Diabetes Care* 2005;**28**:810–5.
- 6 **Baker EH**, Janaway CH, Philips BJ, *et al*. Hyperglycaemia is associated with poor outcomes in people admitted to hospital with acute exacerbations of chronic obstructive pulmonary disease. *Thorax* 2006;**61**:284–9.
- 7 **NHS Connecting for Health**. Clinical coding: sample audits, 2006. Available at <http://www.connectingforhealth.nhs.uk/>
- 8 **Finney SJ**, Zekveld C, Elia A, *et al*. Glucose control and mortality in critically ill patients. *JAMA* 2003;**290**:2041–7.
- 9 **Plant PK**, Owen JL, Elliott MW. Non-invasive ventilation in acute exacerbations of chronic obstructive pulmonary disease: long term survival and predictors of in-hospital outcome. *Thorax* 2001;**56**:708–12.
- 10 **Malmberg K**, Ryden L, Efendic S, *et al*. Randomized trial of insulin-glucose infusion followed by subcutaneous insulin treatment in diabetic patients with acute myocardial infarction (DIGAMI study): effects on mortality at 1 year. *J Am Coll Cardiol* 1995;**26**:57–65.
- 11 **Dandona P**, Mohanty P, Chaudhuri A, *et al*. Insulin infusion in acute illness. *J Clin Invest* 2005;**115**:2069–72.
- 12 **Mesotten D**, Wouters PJ, Peeters RP, *et al*. Regulation of the somatotrophic axis by intensive insulin therapy during protracted critical illness. *J Clin Endocrinol Metab* 2004;**89**:3105–13.
- 13 **Van den Berghe G**, Wouters PJ, Bouillon R, *et al*. Outcome benefit of intensive insulin therapy in the critically ill: insulin dose versus glycemic control. *Crit Care Med* 2003;**31**:359–66.
- 14 **Mesotten D**, Swinnen JV, Vanderhoydonc F, *et al*. Contribution of circulating lipids to the improved outcome of critical illness by glycemic control with intensive insulin therapy. *J Clin Endocrinol Metab* 2004;**89**:219–26.
- 15 **Mohanty P**, Hamouda W, Garg R, *et al*. Glucose challenge stimulates reactive oxygen species (ROS) generation by leucocytes. *J Clin Endocrinol Metab* 2000;**85**:2970–3.
- 16 **Philips BJ**, Redman J, Brennan A, *et al*. Glucose in bronchial aspirates increases the risk of respiratory MRSA in intubated patients. *Thorax* 2005;**60**:761–4.
- 17 **Krinsley JS**. Effect of an intensive glucose management protocol on the mortality of critically ill adult patients. *Mayo Clin Proc* 2004;**79**:992–1000.

Childhood asthma

The childhood asthma epidemic

G Russell

A case of delayed rather than mistaken diagnosis

Although asthma has been recognised for millennia,^{1–2} early writers made no attempt to estimate its prevalence. Salter³ declared that asthma “cannot, in this country, be said to be by any means rare, and I believe that all who direct their attention to it will find it to be much commoner than is imagined”. He did not, however, venture any estimate of its prevalence, and it was another 60 years before epidemiological studies began to appear.⁴

These early studies were bedevilled by the lack of an agreed definition of asthma, reluctance to diagnose a chronic untreatable condition that in the Oslerian tradition was widely

regarded as psychoneurotic,⁵ and failure to appreciate that upper respiratory tract infection was a major trigger of asthma attacks, so that wheeze triggered by infection was usually diagnosed as bronchitis. Thus, when Collins⁶ described morbidity patterns in 9000 American families in 1935, he reported that, in children aged 5–9 years, bronchitis and chest colds affected 5.6% whereas asthma affected only 0.5%. A similar numerical relationship between asthma and bronchitis was still being reported from English general practices over 20 years later.⁷ How many of these children with bronchitis and chest colds would now be offered a diagnosis of asthma is a moot point, but there can be

little doubt that the prevalence of asthma was underestimated on both sides of the Atlantic.

In the 1950s several papers on the epidemiology of childhood asthma appeared from Scandinavia, reporting prevalences of 0.6–1.4%.^{8–11} These studies relied on identifying cases that were known to school doctors, nurses or other professional personnel, and the prevalence data are likely to have been underestimates, partly because of the method of ascertainment and partly because of the widespread diagnosis of bronchitis.⁸

By 1967 the prevalence of asthma in children on the Isle of Wight was reported to be 2.3%¹² and in Aberdeen in 1969 the figure was 4.8%,¹³ a figure that excluded 6.7% of children who wheezed only in the presence of infection.

Taking these figures at face value, it appeared that the prevalence of asthma had climbed steadily from 0.53%⁴ to 4.8%¹³ over a period of 40 years. Thus, although these data must be viewed with extreme caution, there is nothing new about the idea that childhood asthma is becoming more prevalent.

THE IMPORTANCE OF DIAGNOSING ASTHMA

Whether or not the prevalence of asthma was increasing was interesting but of little practical importance in the 1960s when the therapeutic options were limited and only more severely affected children would tolerate the side effects associated with isoprenaline (isoproterenol) inhalers, oral theophylline, and systemic corticosteroids or ACTH. All this was to change with the development over the next few years of effective anti-asthmatic drugs that were relatively free of side effects.^{14–16}

In 1983 a study from North Tyneside showed that the prevalence of wheeze in 7 year old children was 9.3%,¹⁷ little different from the 11.5% that had been reported from Aberdeen in 1969.¹³ The importance of this study was not that wheeze was so common (although this surprised some readers), but that all the children with episodic wheeze responded to asthma treatment,¹⁷ suggesting that all—or virtually all—episodic wheeze in 7 year old children was asthmatic in nature. The recognition of the underdiagnosis and consequent undertreatment of asthma¹⁸ then became a burning issue and was followed by a massive educational initiative, much of it sponsored by the manufacturers of the then new asthma treatments, resulting in a gradual change in diagnostic and therapeutic practice. Bronchitis and chest infection gradually disappeared from hospital discharge statistics to be replaced by asthma.

EARLY INDICATORS OF AN ASTHMA "EPIDEMIC"

It was therefore not surprising that recorded hospital admissions for childhood asthma increased, but there was also strong evidence to suggest that this was not entirely explained by diagnostic relabelling.¹⁹ The upward trend in admissions continued throughout the world in the 1980s,²⁰ and the possibility that asthma was becoming more prevalent was widely entertained. Numerous repeated cross-sectional studies, the first of which was a series of studies from Birmingham,²¹ suggested that the prevalence of asthma was indeed increasing.

Although these studies were all too easily criticised on methodological grounds such as performance in different populations, inconsistent questionnaires, method of application or sampling frame, acceptance of the diagnosis of asthma without examination of symptoms, and lack of objective testing,²² it was notable that none showed that the prevalence of asthma was declining. By the end of the 1980s it is

fair to say that only one study fulfilled all the criteria for reliable repeated cross-sectional studies on asthma. Using identical methods in two studies performed at an interval of 15 years and including a test for exercise induced bronchospasm, Burr *et al*²³ showed that the prevalence of asthma, its cardinal symptom wheeze, the atopic conditions eczema and hay fever commonly associated with it, and its pathophysiological hallmark bronchial hyperreactivity had all increased. Whether it is appropriate to describe these increases as an epidemic is debatable, but the term does provide a convenient shorthand description of a phenomenon that became the centre of attention for numerous research workers in the latter part of the 20th century.

THE REASONS FOR THE ASTHMA EPIDEMIC

From 1989 onwards we could therefore be confident that there had been a genuine increase in the prevalence of childhood asthma and focus on finding possible explanations. The rest—to coin a phrase—is history. Strachan's hygiene hypothesis²⁴ explained many earlier findings such as the protective effects of overcrowding,¹⁰ birth in the Indian subcontinent,²¹ and residence in rural areas,^{10, 25} and has been supported by numerous subsequent studies.²⁶ Whether improved hygiene is the only explanation for the emergence of childhood asthma as a major scourge in westernised societies in the latter part of the 20th century remains to be seen. Numerous other factors have been incriminated, beyond the scope of this review, and research workers continue to probe the complex genetic-immunological-environmental interactions that lead to the expression of asthma.

THE COURSE OF THE ASTHMA EPIDEMIC

It soon became apparent that wheezing and atopic disorders were increasing at a rate that could not be sustained. In Aberdeen between 1989 and 1994 we observed a 92% increase in the prevalence of diagnosed asthma, most of which was explained by increased diagnosis in symptomatic children. Nevertheless, there was also a 28% increase in the prevalence of wheeze,²⁷ a rate of increase which if maintained would have resulted in universal childhood wheeze within a generation. It was therefore no surprise to find that, by 1999, although the diagnosis of asthma was still increasing, there was no significant increase in the prevalence of wheeze.²⁸ Similar findings indicating that the increase in asthma and asthma related symptoms is moderating or even

declining have been reported from around the world,²⁹ and it is interesting to note that, in the most recent study by Burr *et al*³⁰ published in this issue of *Thorax*, although current asthma had increased by 69% during the previous 15 years, there was a more modest increase of 30% in current wheeze, confirming earlier suggestions that part of the asthma epidemic is more apparent than real and reflects changes in diagnostic labelling.³¹

BENEFICIAL EFFECTS OF THE ASTHMA EPIDEMIC

The asthma epidemic has not, however, been an unmitigated disaster, and the increased public and professional awareness of asthma that has occurred in the past 20 years has ensured great improvements in the diagnosis and therefore the treatment of asthma. Undiagnosed untreated severe asthma is now vanishingly rare, and more attention is paid to improving the quality of life enjoyed by asthmatic children and to mitigating the psychosocioeconomic burden borne by them and their families.³² Burr and his colleagues deserve much of the credit for finally convincing the medical profession that childhood asthma was becoming more common, the fruits of which are apparent in their latest study, which shows that there has been a substantial increase in the proportion of children on both bronchodilator and prophylactic treatment. The implication of the decline in exercise induced bronchoconstriction is that, with appropriate diagnosis and treatment, children with asthma should now be better able to participate in normal childhood activities. To coin another phrase, it's an ill wind ...

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REFERENCES

- 1 Sakula A. A history of asthma. The FitzPatrick lecture 1987. *J R Coll Physicians Lond* 1988;**22**:36–44.
- 2 Ellul-Micallef R. Asthma: a look at the past. *Br J Dis Chest* 1976;**70**:112–6.
- 3 Salter HH. *On asthma: its pathology and treatment*. London: Churchill, 1868:1.
- 4 Sydenstricker E. The incidence of various diseases according to age; Hagerstown morbidity studies. *Public Health Rep* 1928;**43**:1124–56.
- 5 Osler W. *The Principles and practice of medicine*. New York: Appleton, 1901:628.
- 6 Collins SD. Age incidence of specific causes of illness based on records of 9,000 families in 18 states visited periodically for 12 months, 1928–31. *Public Health Rep* 1935;**50**:1404–27.

- 7 Logan WPD, Cushion AA. *General Register Office Studies on Medical and Population Subjects: Report No 14: Morbidity Statistics from General Practice*. London: HMSO, 1958.
- 8 Kraepelien S. The frequency of bronchial asthma in Swedish school children. *Acta Paediatr* 1954;**43**(Suppl 100):149–53.
- 9 Eilertsen E. Astmahyppighet et skolebarnsmateriale fra Bergen. *Tidsskr Norske Laegefor* 1954;**73**:322–8.
- 10 Peltonen M-L, Kasanen A, Peltonen TE. Occurrence of allergic conditions in schoolchildren. *Ann Paediatr Fenn* 1955;**1**:119–29.
- 11 Eriksson-Lihr Z. Special features of allergy in children. *Acta Allergol* 1955;**8**:289–313.
- 12 Graham PJ, Rutter ML, Yule W, et al. Childhood asthma: a psychosomatic disorder? Some epidemiological considerations. *Br J Prev Soc Med* 1967;**21**:78–85.
- 13 Dawson B, Illsley R, Horobin G, et al. A survey of childhood asthma in Aberdeen. *Lancet* 1969;**1**:827–30.
- 14 Hartley D, Jack D, Lunts LHC, et al. New class of selective stimulators of β -adrenergic receptor. *Nature* 1968;**219**:861–2.
- 15 Gregg I. Disodium cromoglycate in bronchial asthma. *Lancet* 1968;**2**:350–1.
- 16 Brown HM, Storey G, George WH. Beclomethasone dipropionate: a new steroid aerosol for the treatment of allergic asthma. *BMJ* 1972;**1**:585–90.
- 17 Lee DA, Winslow NR, Speight AN, et al. Prevalence and spectrum of asthma in childhood. *BMJ (Clin Res Ed)* 1983;**286**:1256–8.
- 18 Speight AN, Lee DA, Hey EN. Underdiagnosis and undertreatment of asthma in childhood. *BMJ (Clin Res Ed)* 1983;**286**:1253–6.
- 19 Anderson HR. Increase in hospitalisation for childhood asthma. *Arch Dis Child* 1978;**53**:295–300.
- 20 Mitchell EA. International trends in hospital admission rates for asthma. *Arch Dis Child* 1985;**60**:376–8.
- 21 Smith JM. The prevalence of asthma and wheezing in children. *Br J Dis Chest* 1976;**70**:73–7.
- 22 Magnus P, Jaakkola JJ. Secular trend in the occurrence of asthma among children and young adults: critical appraisal of repeated cross sectional surveys. *BMJ* 1997;**314**:1795–9.
- 23 Burr ML, Butland BK, King S, et al. Changes in asthma prevalence: two surveys 15 years apart. *Arch Dis Child* 1989;**64**:1452–6.
- 24 Strachan DP. Hay fever, hygiene, and household size. *BMJ* 1989;**299**:1259–60.
- 25 Van Niekirk CH, Weinberg EG, Shore SC, et al. Prevalence of asthma: a comparative study of urban and rural Xhosa children. *Clin Allergy* 1979;**9**:319–4.
- 26 Strachan DP. Family size, infection and atopy: the first decade of the 'hygiene hypothesis'. *Thorax* 2000;**55**(Suppl 1):S2–10.
- 27 Omran M, Russell G. Continuing increase in respiratory symptoms and atopy in Aberdeen schoolchildren. *BMJ* 1996;**312**:34.
- 28 Devenny AM, Wassall HJ, Ninan TK, et al. Respiratory symptoms and atopy in children in Aberdeen: questionnaire studies of a defined school population repeated over 35 years. *BMJ* 2004;**329**:489–90.
- 29 Toelle BG, Marks GB. The ebb and flow of asthma. *Thorax* 2005;**60**:87–8.
- 30 Burr ML, Wat D, Evans C, et al. Asthma prevalence in 1973, 1988 and 2003. *Thorax* 2006;**61**:296–9.
- 31 Hill R, Williams J, Tattersfield A, et al. Change in use of asthma as a diagnostic label for wheezing illness in schoolchildren. *BMJ* 1989;**299**:898.
- 32 Lenney W. The burden of pediatric asthma. *Pediatr Pulmonol (Suppl)* 1997;**15**:13–6.

Surgical training

Training in the operating theatre: is it safe?

R Aggarwal, A Darzi

The importance of a systems approach to surgical training

Recent years have witnessed a number of drivers for change in the delivery of health care.¹ Working time restrictions, quality assurance targets, the introduction of new technologies and star ratings for hospitals have served to create antagonism between service and training priorities. The provision of a high quality service necessitates the employment of proficient practitioners, using tools to the highest of their abilities. This is in discord with the apprenticeship model of training whereby trainees undergo graded practice on patients, leading to the development of proficiency.² Furthermore, a number of high profile cases have highlighted the need for regular audit of outcomes to ensure patient care is not compromised.³

It is well known that achievement of proficiency to perform a procedure entails a learning curve during which morbidity and mortality gradually decline.^{4,5} However, some of the complications which occur during the learning curve are avoidable through appropriate case selection and adequate supervision during the procedure. In the example offered by Chaudhuri *et al*⁶ in this issue of *Thorax*, trainee thoracic surgeons led over one

third of the cases of lung resection. A comparison of outcomes revealed similar rates of complications and survival at 1 year. In terms of case selection, consultants operated on greater numbers of stage III tumours and trainees on a greater number of stage I tumours.

This study echoes the main features of the apprenticeship model which has been a cornerstone of skills acquisition in surgical disciplines. The key principle is to ensure patient safety whilst enabling the trainee surgeon to acquire and hone skills to perform the procedure. The notion of a graded approach allows trainees to acquire skills in a stepwise manner, through close supervision by a senior surgeon. It is the responsibility of the senior surgeon to ensure the patient does not come to any undue harm. Upon achievement of proficiency, the trainee can proceed onto performing more complex cases, eventually gaining the ability to operate independently.

Although the apprenticeship model has stood the test of time, the incorporation of new technologies for technical skills training outside the operating room can further improve patient safety.⁷ In analogy with the airline industry, it is

now possible for trainees to acquire basic skills which transfer to improved performance in the operating suite.^{8–11} It is no longer appropriate (nor acceptable) to have a surgeon dithering in theatre. The trainee must know the basic skills and be able to undertake complex manoeuvres by the time he comes to the operating theatre. With the incorporation of simulation based training earlier in the curriculum, it may also be possible to reduce the length of the learning curve for the achievement of proficiency on real cases.

Upon achievement of proficiency in the skills laboratory, training must continue in a structured manner in the operating room. Graded exposure along with appropriate support when necessary is most effective in transferring skills from tutor to student. This should also not be limited to the operating theatre, but augmented by discussions and feedback before and after each case. In addition, the postoperative dialogue of each procedure can be supported by video footage of the operation. Thus, the model is still recognisable as graded exposure in the operating suite, but amplified by a number of other factors.

For each interventional speciality, outcomes from a key procedure are traditionally used as markers of an individual surgeon's technical performance. However, this approach is too simplistic and fails to take account of the numerous factors which can affect patient outcomes.¹² Patient characteristics can decrease or increase the risk of complications, especially during major surgical procedures. This can be accounted for through appropriate case selection, ensuring that the sickest or most complex patients are operated upon by the most experienced surgeons. However, it