formation that treatment should be available to all on the basis of need and free at the time of use.)

The second shift requires an acknowledgment that the outcomes in question belong first and foremost to patients. Marianne Rigge of the College of Health showed just how alien this concept is in Britain by quoting from the publicity leaflet for the conference. "It should be obvious that many different groups have a stake in debates over outcomes—certainly doctors (as providers of medical care), but also pharmaceutical companies, managers, insurance companies and governments," it read. Patients were missing.

Greater use of outcomes research in Britain will be impeded by the poor quality of data—the stumbling block affecting the introduction of so many other good ideas. Brendan Devlin, consultant surgeon at North Tees General Hospital and secretary of the national confidential enquiry into perioperative deaths, decried the deterioration of information provided by the Department of Health and the NHS since the NHS reforms. But even when good quality data are collected confounding (by differences in case severity), bias, small sample sizes, and self serving data manipulation can make interpretations difficult, explained Martin McKee, senior lecturer at the London School of Hygiene and Tropical Medicine. Anyone hoping to compile or use hospital league tables for deaths from surgery needs to be aware of these problems (as neither the *Times* nor the Department of Health seemed to have been last year when the newspaper published league tables supplied by the department[3]).

Despite the attractions of using death rates as a measure of outcome too few deaths occur to make them sufficiently sensitive. For example, Brendan Devlin has estimated that only 0·6% of surgical operations end in death. Similarly, Nottinghamshire general practitioner Mike Pringle said that in general practice death was too infrequent to use to monitor changes in health of or the quality of care received by his patients. Instead, every month his practice discussed "significant events" in the life of his practice—that is, every stroke, myocardial infarction, new diagnosis of cancer, suicide attempt, unplanned pregnancy, patient complaint, patient leaving the practice without changing address, and so on. "All are outcomes of the clinical and administrative commitment of the practice. We ask: was everything possible done that should have been done?"

Although we undoubtedly need more randomised controlled trials, we cannot afford to squander the new knowledge that can be gleaned from disciplined, systematic aggregation of collective clinical experience, said Mulley. He singled out surgery for coronary artery disease and for breast cancer as prime candidates for this sort of research. "But our need for better information about what works and what is valued, and about how to do it right, is a general one," he said. "Few conditions would not be better treated if we developed a system of outcomes research that could be brought into clinical practice."

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Asthma in children: epidemiology

Changing prevalence, patterns, and treatment

Asthma is one of the commonest childhood illnesses, and its prevalence and severity may be increasing. Determining whether this is so has been difficult, however, because of the lack of a widely accepted epidemiological definition of asthma and an objective measure of asthma that is applicable to children and is reasonably sensitive and specific. Furthermore, many studies purporting to show an increase in prevalence have used different measures and studied far from comparable populations over the period of the suggested increase.

Several valid studies have now reported a substantial increase in the prevalence of asthma in some countries. In 1991 Robertson and colleagues in Melbourne showed a doubling over 26 years, to 46%, in the prevalence of children with wheeze on one or more occasions before the age of 7; 21-23% had wheezed in the previous 12 months.[1] These findings have been confirmed by Peat and colleagues, who studied children aged 8-10 in two country towns in New South Wales (p 1591).[2] They found about a doubling over 10 years, to 23-27%, in the prevalence of children reporting wheeze in the previous 12 months. This is not solely an Australian phenomenon—Anderson *et al* report a more modest increase over 13 years, to 13%, in the prevalence of children aged 7·5 to 8·5 who reported wheeze in the previous 12 months (p 1600).[3]

The study by Peat and colleagues is particularly important as it reports objective measurements whereas the other studies were based on responses to a questionnaire. It showed that airway hyperresponsiveness increased 1·4-fold to twofold. Although airway hyperresponsiveness is not equivalent to asthma (8-15% of children who have never wheezed will show bronchial hyperresponsiveness and up to 30% with typical asthma will fail to show it on several occasions), its increase supports the validity of the questionnaire completed by parents. Interestingly, this study found that airway hyperresponsiveness increased mainly in children with other evidence of atopy, although the prevalence of atopy was unchanged.

Even when differences in methodology are allowed for, there is considerable international variation in the prevalence of childhood asthma, defined as wheeze, within the past 12 months. Similar high rates to those reported in Australia have been noted when almost identical questionnaires were used in Fiji[4] and in La Serena, a non-polluted town in northern Chile.[5] In Switzerland the prevalence is much lower at about 7%[5] and Germany has rates of 4% to 6%.[6]

The reasons for this increase are not known. As genetic change seems highly unlikely some environmental factors must be responsible. The increase has occurred during a time when general atmospheric pollution has fallen, and high prevalences were seen in Fiji and northern Chile—where pollution is not a problem. The prevalence was lower in Leipzig (formerly in East Germany) than in Munich.[6] Bronchodilating drugs have been relatively little used in Fiji so attributing the high prevalence to some effect from them is

difficult. Peat and colleagues suggest, with some supporting data, that the increased responsiveness in atopic children may be due to a much greater allergen load in a child's environment.[2] Compatible with such a mechanism is the lower frequency of wheeze in the Swiss mountains.

The studies of Peat *et al* and Anderson *et al* indicated that the number of episodes of wheeze in affected children had increased,[2 3] but Anderson *et al*'s study suggested, however, that the morbidity from asthma as indicated by days lost from school and interference with normal activities had fallen substantially over the 13 years of study. Hyndman and colleagues in a study from East Anglia found that the increase in admission to hospital of children with asthma, which has been reported from many countries, may be reaching a plateau or even falling (p 1596).[7] These are reassuring findings and could well be due to more effective treatment in those with recognised asthma. The greater willingness of doctors to diagnose asthma in children with recurrent wheeze is probably the single most important factor in improving treatment.[8]

The increase in the prevalence of asthma in children may well have serious implications for adults as 40% of children with infrequent trivial wheeze and 70-90% of those with more troublesome asthma continue to have symptoms in mid-adult life.[9] As yet studies have not shown an increase in asthma in elderly people, although it may be increasing in young adults.[10] If the increase in the prevalence in children continues into adult life this will create substantial health problems and may well have important economic consequences through time off work and the cost of treatment.

Prophylactic drugs, particularly inhaled corticosteroids, might be expected to increase the likelihood of childhood asthma resolving, yet there is no evidence to support this. The long term use of inhaled corticosteroids in children with more troublesome asthma has failed to alter permanently the long term course of the disease.[11] When inhaled steroids were stopped after 24-36 months of treatment the pattern of asthma rapidly returned to the pattern that had been present before they were started. Furthermore, increasing evidence exists that even low dose inhaled corticosteroids may have a prolonged growth suppressant effect in children with mild asthma.[12] The more widespread use of the currently available inhaled corticosteroids in the hope of modifying the natural course of asthma cannot be recommended. Their use should be limited to those children in whom asthma substantially interferes with the enjoyment of a normal lifestyle or in whom there is persisting airways obstruction. Whether the newer inhaled corticosteroids will have fewer side effects, particularly effects on growth, remains to be determined.

Ways to limit the increase in the prevalence of asthma and if possible to reduce it are clearly vitally important. Until the reasons for the observed increase are determined, however, it is not possible to suggest intervention strategies. The hypothesis of Peat and colleagues that the likely explanation is a change in the degree of exposure to allergens is interesting and is further discussed by Cullinan and Newman Taylor below (p 1585).[13] Reducing exposure to allergens, particularly to house dust mites, is expensive and substantially alters lifestyles. It is too early to recommend interventions in the absence of proof of efficacy and benefit.

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Asthma in children: environmental factors

Increased sensitisation to inhaled allergens seems the most likely explanation for asthma's increased prevalence

Two studies of asthma in children, using identical survey methods and objective assessments separated by a period of 10-15 years, have now been published; each shows an increase in asthmatic symptoms and airway hyper-responsiveness and in seasonal rhinitis.[1] (p1591).[2] The increased prevalence of asthma has been matched by, and is probably a manifestation of, an increase in sensitisation among children to inhaled allergens, such as those present in house dust, cat fur, and grass pollen.[3]

Although genetic factors are important in determining both the propensity to atopic disease and the specificity of the response to protein epitopes, the short period during which the increases in asthma and other allergic diseases have occurred suggests that environmental influences have been mainly responsible. For example, secular trends in the Finnish armed forces show a 20-fold increase in asthma among 18 year old recruits during the past 30 years.[4]

Further evidence of important environmental influences comes from the recent observation that sensitisation to common aeroallergens is about twice as common among children in Munich as among genetically similar children in two cities that were formerly in East Germany, Leipzig and Halle.[5] Against this background it is valuable to consider what environmental changes have been common to Western countries in the past 30 to 40 years.

Childhood asthma is predominantly an allergic disease; changes may have occurred in exposure to aeroallergen (quantitatively or qualitatively) or in concurrent exposure to factors that modify the response to allergens. These factors include various respiratory irritants, such as tobacco smoke