often. If the results of a single work up are inconclusive and microscopic haematuria persists then the diagnostic tests should be repeated in due course.

FRITZ H SCHRÖDER
Professor

Department of Urology,
Erasmus University and Academic Hospital,
3015 GD Rotterdam,
Netherlands

1 Mariani AJ, Mariani MC, Macchioni C, Stams UK, Hariharan A, Moriera A. The significance of adult haematuria: 1000 haematuria evaluations including a risk-benefit and cost-effectiveness analysis. *J Urol* 1989;**141**:350-5.
2 Messing EM, Young TB, Hunt VB, Wehbie JM, Rust P. Urinary tract cancers found by homescreening with haematuria dipstick in healthy men over 50 years of age. *Cancer* 1989;**64**:2361-7.
3 Daum GS, Krolikowski FJ, Reuter KL, Colby JM, Silva WM. Dipstick evaluation of hematuria in abdominal trauma. *Am J Clin Pathol* 1988;**89**:538-42.
4 Laville M, Roy P, Pellet H, Fabry J, Zech P. Hématurie microscopique de l'adulte. Prévalence et facteurs associés. *Presse Medicale* 1991;**20**:545-50.
5 Corwin HL, Silverstein MD. The diagnosis of neoplasia in patients with asymptomatic microscopic haematuria: a decision analysis. *J Urol* 1988;**139**:1002-6.
6 Fassett RG, Horgan BA, Mathew TH. Detection of glomerular bleeding by phase contrast microscopy. *Lancet* 1982;i:1432-4.

Growing up with asthma

The two thirds with milder symptoms should grow out of asthma

See papers on p 90 and p 95

Childhood asthma has been regarded as relatively benign and a condition that children often "grow out of." Recently, however, investigators have reported higher prevalence of asthma in children[1 2] and more frequent persistence of childhood asthma into adult life.[3] Hence further studies on factors predicting remission or persistence of childhood asthma, and the development of adult asthma, are welcomed.

Two papers in this week's journal describe outcomes of asthma in cohorts of Australian children. Both longitudinal studies began in the 1960s and both followed up children recruited at age 7 until their 30s. One might therefore expect the findings of the two studies to be similar. Nevertheless, Jenkins *et al* conclude that "children with asthma reported by their parents were more likely than not to be free of symptoms as adults" (p 90)[4] while Oswald *et al* conclude that "many children do not grow out of asthma" (p 95).[5] How could similar studies in similar populations come to seemingly opposite conclusions?

Jenkins *et al* followed up a birth cohort of 8700 7 year old Tasmanian children whose parents had originally completed a questionnaire inquiring about a history of wheezing and asthma, recent wheeze, frequency of episodes, age at onset, presence of hayfever, and smoking. All children were assessed by spirometry. A follow up at 29-32 years, 1000 of those with parent reported asthma or wheezing were selected, together with 1000 others; 87% were located, and 75% provided information on their current respiratory status.

Four major conclusions can be drawn from this study. Firstly, a high proportion of asthmatic children become symptom free. The symptoms of fewer than one in three asthmatic children persisted into the fourth decade. Secondly, asthma in adults can be partially predicted from childhood asthma, especially from those features reflecting severity of the childhood disease. Those with childhood asthma were 2·5 times more likely to have adult asthma than those with no such history. In those with persistent disease, risk factors included female sex, onset after age 2, more than 10 attacks throughout childhood, lower peak flow rates in childhood, and parental asthma.

Thirdly, atopy is a major determinant of the outcome, with personal and parental histories of atopy being more commonly associated with frequent, current asthma in adulthood. Fourthly, new asthma in adulthood is not uncommon. One in nine children not reported as having asthma at age 7 developed asthma by age 30. Female sex, maternal hay fever, and lower lung function at age 7 were important risk factors for this. The most impressive abnormality of lung function in children who developed asthma in adulthood was a lower mid-expiratory flow, suggesting that small airway disease is predictive of adult asthma.

In contrast, the emphasis of Oswald *et al*'s paper is that most childhood asthma persists. This report comes from Melbourne children who were recruited in the 1960s and reviewed at age 35. Methodological differences explain the different findings. The cohort was selected from the population of 7 year olds, but when reviewed at age 10 the cohort was "enriched" with children with more severe asthma (asthma with an age of onset below 3 years, with persistent symptoms, and either chest wall deformity or low lung function at age 10). These characteristics might be expected to predict more persistent asthma, and the finding that 70% of those with severe asthma at age 10 had persistent asthma at 35 is therefore not surprising. Others have reported similarly higher levels of symptoms persisting in people who had attended asthma clinics when children.[6] But even among the Melbourne subjects with severe childhood asthma, 10% were fully symptom free as adults and 15% had only minimal symptoms. Jonsson and Boe similarly reported that up to a half of children admitted to hospital with asthma were symptom free as adults.[7]

If the Melbourne sample with wheezing, wheezy bronchitis, or asthma at age 7 is examined before the enriched subsample was added it is apparent that only 20% of subjects had persistent adult asthma likely to be clinically important. Of those with "wheezy bronchitis," only a third had any symptoms at 35. This is consistent with "growing out of" mild asthma or wheezing, and the frequent persistence of more severe asthma. Interestingly, among Melbourne children with mild asthma, atopy was not a prognostic factor. This finding differs not only from that of the Tasmanian study, but also from other studies in Australia,[8] New Zealand,[9] Scandinavia,[10] and Britain.[11] Atopy was very common in the Melbourne cohort (99% in those with more severe disease), which makes the effect of atopy more difficult to determine. Prognosis of childhood asthma in the Netherlands, however, was likewise apparently not influenced by the presence of atopic disease.[12 13]

Other risk factors for persistent asthma in adults include airway hyperresponsiveness in childhood,[12] but the present reports do not address this. Most studies show that persistent asthma is more likely in females. The likelihood of a low forced expiratory volume at one second in adulthood is related to the degree of abnormality of forced expiratory volume in childhood.[14 15]

Neither of these Australian papers addresses the impact of treatment on "natural" course. The favourable outcome

for over two thirds of asthmatic children should influence long term studies of treatment. It is logical to study the efficacy of treatment in those at greater risk of persistent disease rather than in those who will probably grow out of their disease. Jenkins *et al* argue the benefits of treating children at higher risk, but the absence of substantiating evidence highlights the need for well controlled, long term studies of intervention in childhood asthma.

These two longitudinal studies therefore yield essentially consistent outcomes: about two thirds of children will out grow their disease (especially those with mild asthma), while the third with more severe asthma will have persistent symptoms in adult life. Risk factors for persistence have been identified: the challenge now is to determine whether managing these "at risk" children more intensively alters the outcome.

MALCOLM R SEARS
Professor of medicine

Asthma Research Group,
McMaster University,
Hamilton,
Ontario L8N 4A6,
Canada

1 Peat JK, Van den Berg RH, Green WF, Mellis CM, Leeder SR, Woolcock AJ. Changing prevalence of asthma in Australian children. *BMJ* 1994;**308**:1591-6.
2 Anderson HR, Butland BK, Strachan DP. Trends in prevalence and severity of childhood asthma. *BMJ* 1994;**308**:1600-4.
3 O'Brien KP, Fischer TJ. The Dutch hypothesis revisited: recent evidence that children do not outgrow asthma. *Pediatr Asthma Allergy Immunol* 1993;7:89-97.
4 Jenkins MA, Hopper JL, Bowes G, Carlin JB, Flander LB, Giles GG. Factors in childhood as predictors of asthma in adult life. *BMJ* 1994;**309**:90-3.
5 Oswald H, Phelan PD, Lanigan A, Hibbert M, Bowes G, Olinsky A. Outcome of childhood asthma in mid-adult life. *BMJ* 1994;**309**:95-6.
6 Roorda RJ, Gerritsen J, van Aalderen WMC, Schouten JP, Veltman JC, Weiss ST, *et al.* Risk factors for the persistence of respiratory symptoms in childhood asthma. *Am Rev Respir Dis* 1993;**148**:1490-5.
7 Jonsson JA, Boe J. Asthma as a child. Symptom-free as an adult? *Ann Allergy* 1992;**69**:300-2.
8 Peat JK, Salome CM, Woolcock AJ. Longitudinal changes in atopy during a 4 year period: relation to bronchial hyperresponsiveness and respiratory symptoms in a population of Australian schoolchildren. *J Allergy Clin Immunol* 1990;**85**:65-74.
9 Sears MR, Burrows B. Changes in airway responsiveness from age 9 to 15: relationship with atopy, symptoms and lung function. *Am J Respir Crit Care Med* 1994;**149**:A912.
10 Kokkonen J, Linna O. The state of childhood asthma in young adulthood. *Eur Respir J* 1993;**6**:657-61.
11 Anderson HR, Pottier AC, Strachan DP. Asthma from birth age 23: incidence and relation to prior and concurrent atopic disease. *Thorax* 1992;**47**:537-42.
12 Roorda RJ, Gerritsen J, van Aalderen VHC, Knol K. Influence of a positive family history and associated allergic diseases on the natural course of asthma. *Clin Exp Allergy* 1992;**22**: 627-34.
13 Gerritsen J, Koeter GH, de Monchy JGR, Knol K. Allergy in subjects with asthma from childhood to adulthood. *J Allergy Clin Immunol* 1990;**85**:116-25.
14 Godden DJ, Ross S, Abdalla M, Mc-Murray D, Douglas A, Oldman D, *et al.* Outcome of wheeze in childhood. Symptoms and pulmonary function 25 years later. *Am J Respir Crit Care Med* 1994;**149**:106-12.
15 Roorda RJ, Gerritsen J, van Aalderen WMC, Schouten JP, Veltman JC, Weiss ST, *et al.* Follow-up of asthma from chil hood to adulthood: influence of potential childhood risk factors on the outcome of pulmonary function and bronchial responsiveness in adulthood. *J Allergy Clin Immunol* 1994;**93**:575-84.

Hepatitis B and exposure prone procedures

New guidelines should be implemented this month

See papers on p 94

By this month all of Britain's surgeons undertaking exposure prone procedures should be immune to hepatitis B—either through natural immunity or after a course of immunisation.[1-3] Government guidelines make exceptions for staff who fail to respond to the vaccine and those who are positive for hepatitis B surface antigen—provided that they are negative for hepatitis B e antigen. The guidelines' aim is to identify and exclude any health care worker who is positive for hepatitis B e antigen from performing procedures in which injury to the worker could result in blood contaminating the patient's open tissue.

Two short papers in this week's journal suggest that implementing these guidelines has not been without problems. Poole and colleagues report that four surgeons needed six or nine doses of vaccine (instead of the usual three) to develop adequate immunity; two consultant surgeons refused either to be vaccinated or to provide evidence of immunity (p 94).[4] In another report Hassan and Oldham describe one health care worker who developed Reiter's syndrome after the second dose of vaccine and another who developed arthritis after the first (p 94).[5] Revaccination was not attempted as more severe adverse effects may follow.

The guidelines take account of these and similar problems. People without evidence of previous infection who do not respond to the vaccine may continue to perform exposure prone procedures provided that inoculation incidents are followed up according to the guidelines; so may "health care workers...in whom completion of the course is deemed inadvisable because of a severe reaction to vaccine." On the other hand, those who refuse to provide evidence of immunity face the same restrictions as those imposed on staff who are positive for hepatitis B e antigen—that is, not being allowed to perform exposure prone procedures (and the offer of alternative work).

Before refusal doctors should be aware that they will be treading a lonely path. Some employers might regard refusal to supply information as failure to comply with a reasonable managerial request and consider disciplinary action. The NHS Executive recommends that employers should take legal advice regarding their responsibility for retraining or redeploying staff whose work is restricted because of refusal to comply with the guidelines.

Defence bodies can defend a doctor only if they can identify experts to support the doctor's case. So far no convincing arguments have emerged to support doctors who put their "rights" to remain ignorant of their hepatitis B status above their responsibilities to patients. Both the Central Consultants and Specialists Committee of the BMA and the Royal College of Surgeons suggest compliance with the guidelines.

The college has, however, emphasised two further points.[6] The first is that surgeons found to be positive for hepatitis B e antigen should insist on further virological assessment of their antigen status and not rely on a single assay from one department. This conforms with recent advice given by the chairman of the British health departments' advisory group on hepatitis that tests of infectivity should be repeated, preferably by various laboratories using different techniques.[7]

The college's second point is that surgeons who are concerned about the test's outcome should, before the test, seek a written undertaking from their employer that it will follow the recommendations of the advisory group on hepatitis. "In particular, they should agree to the individual injury benefits which would be available to them under the conditions laid down in the NHS Injury Benefits Scheme, were they to be found to be e antigen positive."

But strict compliance with the terms of the injury benefits scheme may not be enough: doctors must show that they were infected during work—which may be impossible. And for those having to give up work the scheme