most likely explanation is that the vaccinees were incubating the virus at the time of vaccination; and this was probably true for most patients in this study, in whom HBsAg was detectable for only a short while. One child developed severe, chronic disease, however, and this child was investigated in detail. The child was born to a carrier mother and had been given hepatitis B immunoglobulin at birth and one month later. Vaccination was performed at 3, 4, and 9 months of age. After this regimen, and despite adequate serum concentrations of antibodies to HBsAg, the child developed a severe infection with persistent antigenaemia with both surface and e antigens. Studies on the virus isolated from the mother and from the child at two time points four years apart showed that a stable mutation had occurred in the isolates from the child. This took the form of a single amino acid substitution in the HBsAg which had caused a configurational change in an external loop. The change was such that the antibodies provoked by the vaccine could not bind to the modified virus antigen, so allowing the virus to replicate in the face of a normal antibody response induced by vaccination.

During the replication of any virus mutant forms are continually being produced; their survival depends, firstly, on their ability to maintain a high replication efficiency and, secondly, on the selective pressure engendered by the immune response. In the influenza virus system—the best studied example of this phenomenon—mutations giving rise to antigenic drift regularly lead to the emergence of variants, which can then infect the susceptible population. The mutant hepatitis B virus described by Carman *et al* probably arose from the maternal strain during replication in the child under selection pressure from the vaccine induced immune response.[1] The delay of three months before administration of the vaccine to the child must have allowed replication of hepatitis B virus in the liver to proceed, with the mutant emerging possibly even before vaccination. The antibody response to vaccination would then have imposed the selection pressure necessary to allow the "escape" mutant to replicate unrestricted. It is perhaps surprising that a single antibody binding structure (epitope) should be so dominant, and remarkable that this epitope should be essential in inducing neutralising antibody, as apparently it plays no part in essential virus functions such as cellular binding and entry—the mutant has clearly maintained these functions. What remains to be seen is whether this is an isolated case or whether mutations at this site will be commonplace.

A few reports have now appeared of patients maintaining viral replication after vaccination[2] and of mutant[3,4] and variant[2,5] hepatitis B viruses arising in chronically infected patients. We will have to await detailed analysis of the DNA genomes in such cases before passing judgment on the clinical relevance of the present finding. If, however, this mechanism does emerge as a common cause of vaccination failure, alternative strategies will have to be considered such as the inclusion in the vaccine preparation of other epitopes to stimulate B and T cells. Clearly, as the scope of vaccination widens to include other chronic, persistent, and latent virus infections more such problems may emerge. But they will certainly not be a reason for doubting that vaccination is the method of choice for preventing many of the infectious diseases that still devastate the developing countries.

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BCG vaccination in children

Routine vaccination of schoolchildren is not cost effective and could be stopped

For the past 40 years one of the main planks of the public health strategy against tuberculosis in Britain has been BCG (bacille Calmette-Guerin) vaccination for tuberculin negative schoolchildren aged 10-14. Uptake rates of 75% have been consistently achieved over the past decade. BCG is still recommended for this age group and for children at any age at high risk of infection, such as neonates of Indian ethnic origin and children with a family history of tuberculosis. Vaccination is also recommended for tuberculin negative immigrants from the Indian subcontinent as soon as possible after their arrival in Britain and for contacts of patients with active respiratory tuberculosis.[1-5]

BCG vaccination provides effective protection against active tuberculous infection for a minimum of 20 years.[6] It is also safe. Early and late local cutaneous and regional complications are rare when a defined, age dependent dose of freeze dried vaccine is given by staff well trained in proper techniques.[3,7-9] Hypertrophic scarring and keloid formation may be minimised by injection at or below the insertion of the deltoid, or by using the less accessible sites—the inner aspect of the arm, the thigh, or the buttock.[10] Serious hypersensitivity reaction, osteitis, and intrathoracic and intra-abdominal lesions have been reported only rarely and often without full bacteriological or histological confirmation. Disseminated BCG infection is rare and occurs only in patients with serious defects in cell mediated immunity.[7,8]

Since the introduction of mass BCG immunisation of schoolchildren the incidence of tuberculosis in all ethnic groups—in tuberculin positive and tuberculin negative, vaccinated and unvaccinated groups—has steadily fallen.[2,11] The annual decrease in notifications of tuberculosis attributed to the protective effect of BCG, at a maximum 4·1% from 1965-71, fell to 1% in 1978-83.[12] Thus, despite its safety and efficacy BCG is no longer uniformly offered to all British schoolchildren, and district health authorities have inconsistent vaccination policies.[13] The present low risk of infection for young white adults in England and Wales[14] depends more on higher living standards, effective chemoprophylaxis, and chemotherapy than on vaccination.[15,16] The BCG school vaccination programme has not been cost effective since the mid-1970s,[17] and if it were to be stopped altogether there would be no disaster, only a temporary slowing in the rate of decline in new notifications.[14] These assertions are based on clear evidence from countries where routine BCG vaccination has been stopped.[18]

If, then, the arguments favour abandoning the continued

mass vaccination of schoolchildren what are the implications for public health policy? Whatever decisions are taken locally or nationally about the school BCG programme the vaccine must continue to be offered to all children in high risk categories. This should include neonates born in urban priority areas, poverty perhaps being a better indication of risk than race.[19] It must not, however, be given to patients who are immunocompromised, including children with HIV infection, or to those with malignancy, fever, or generalised skin sepsis.[1,20]

Next, medical vigilance against tuberculosis must continue. A new threat to the steady decline in notification rates is posed by patients with immunosuppression caused by HIV infection. These patients are at increased risk of developing active tuberculosis and acting as a new source for household and other contacts.[21] Not only immigrants, alcoholics, and old people are at risk[12]: outbreaks affecting children will still occur[22] with potential fatal and long term sequelae.[2]

It is for society and the medical profession to answer the major question. When does the effectiveness of chemoprophylaxis and chemotherapy, coupled with the low risk of infection for the individual, make it acceptable to have a few extra cases of tuberculosis because the school vaccination programme has been stopped?

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Retreat from general practice

Dislike of the contract has turned away trainees

One of the most encouraging features of British medicine over the past 25 years has been the development of planned postgraduate education for general practice. After the earliest initiatives in Inverness and the pioneer work of George Swift in Wessex the BMA, the Royal College of General Practitioners, and the departments of health displayed remarkable unanimity in supporting the creation of training schemes comprising two years in hospital and one year in general practice. The Royal Commission on Medical Education in 1966 pointed the way to legislation introduced a decade ago that required all aspiring principals to complete this three year training period. This structured programme resulted in general practice becoming the most popular career choice of medical graduates, attracting some of the most able young doctors. Its attraction often perplexed their hospital consultants, who nevertheless generously supported the programmes themselves. Vocational training schemes were developed throughout Britain and were oversubscribed some seven or eight times, so that those sitting on appointments committees had a wide choice of the most highly qualified and motivated doctors to appoint to these schemes.

Suddenly, this position has changed, and changed dramatically, since the publication of the general practice contract. Organisers of vocational training schemes throughout Britain have reported a precipitous fall in applicants—by a factor of about five. Whereas last year the appointments committees had a wide range of choice, this year, for the first time in 20 years, many vocational training schemes could not be filled on first advertisement.

Fear is the key to understanding this fall in applications. Undoubtedly young doctors are anxious that the government's intention of encouraging increased list sizes will result in fewer opportunities to obtain partnerships. An expansion of the senior house officer grade in hospital has also provided an opportunity for potential trainees to widen their experience in hospital while waiting for the impact of the new contract to become clearer.

But by far the greatest fear expressed by young doctors is their perception of a changing ethos. They are concerned that a career in general practice will not now fulfil the sense of vocation that drew them to medicine. The idea of serving patients in the community, with extensive clinical freedom, is seen as under threat, with values confused with costs. Even more important, however, is what they see as the sinister intrusion of state direction into clinical relations: general practitioners are being asked to undertake clinical tasks that defy their clinical teaching.

Formal vocational training schemes provide only about half the total entrants to general practice in any year, with the remainder planning their own rotations through hospital posts and general practice. Inevitably the sheer numbers of medical graduates will ensure that there are enough entrants to general practice, but to draw satisfaction from that fact is to fail to comprehend the damage done to the morale of these young doctors. By their reactions they have provided the first audit of the contract in practice. Measurements of fear and lowered morale do not translate easily to the accountant's balance sheet. They are, however, central to the success of any enterprise.

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