that there was no genetic heterogeneity,[3] but a few families have now been identified in whom there is no linkage to chromosome 16 markers; this is type 2 adult polycystic kidney disease.[4] Even in these cases, however, the use of recently developed flanking gene markers has made it possible to predict the presence of the condition with greater than 95% confidence in families with an appropriate pedigree structure.

In young patients without symptoms the main clinical problem is to detect the onset of hypertension, which often occurs well before the development of renal impairment. The risk of renal failure usually becomes important only in older patients. Patients and their families may—understandably—find some difficulty in integrating all the information about genetics, asymptomatic hypertension, renal failure, and other complications of the condition. Indeed, a survey of the knowledge and attitudes of affected patients in Edinburgh showed much confusion and misunderstanding.[5]

Counselling needs to be improved if the patients are to be able to understand their condition and make informed decisions. The advice can be integrated with improved assessment and follow up of patients and their families.

In our own unit most patients aged over 18 undergo ultrasound scanning as part of an initial assessment, but preferably only after they have received education and counselling. The first step is an interview during which the background to the condition is explained. Patients are then shown a locally made video that explains the inheritance of the condition and the possible use of genetic markers for early diagnosis, as well as other aspects. Particular attention is given to ensuring that patients understand both the advantages and disadvantages of an early diagnosis. For example, a positive diagnosis may well create difficulty for those seeking employment—on the mistaken assumption that high morbidity is inevitable. Life assurance premiums will also be loaded, increasing the financial pressure on a group of patients who are already financially disadvantaged.[6] Some may not wish to suffer the anxiety over the future that may be associated with a positive diagnosis. On the other hand, awareness of the diagnosis makes regular follow up possible and this facilitates the prevention of complications.

The long natural course of the disorder and the number of patients affected (around one in 1000 of the population[7]) make effective follow up depend on close cooperation among the genetic counsellors, the medical renal unit, and the primary care services. For example, annual checks on blood pressure and renal function may be undertaken by the patients' general practitioners. Unaffected relatives under the age of 30 should be followed up in the same way, though at less frequent intervals, because ultrasound scanning may fail to detect the disease in 15% or more of gene carriers in this age group. Over 400 patients are now being followed in this way in Edinburgh.

A positive diagnosis is technically feasible early in life, but the potential disadvantages are such that only in very rare instances need the diagnosis be confirmed in those aged under 18. Parental curiosity to know the diagnosis may not be in the best long term interests of the child, and monitoring blood pressure and renal function may be all that is required until the age of 18 or more for those "at risk." In our view routine genetic screening is therefore unnecessary.

The availability of a method for antenatal diagnosis raises other ethical problems. Though many patients are keen to know whether their fetus is affected, few would consider termination of pregnancy on the basis of such information. Prenatal diagnosis seems likely to be limited to those families with particularly severe difficulties, and even then should be undertaken only after extensive counselling about the problem.

The availability of DNA markers and high quality ultrasonography for the diagnosis of adult polycystic kidney disease have not, therefore, made any great difference to clinical practice, but they have certainly sharpened awareness of potential ethical problems. The main hope for the future must be that identification of the gene or genes responsible may lead to an understanding of the pathogenesis of the condition and so to the development of more specific treatment. The emphasis at present must remain on diagnosis using ultrasound scanning at an appropriate age, careful counselling of families, and detailed follow up; genetic studies should be limited to those with clear indications.

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HIV infection and tuberculosis

Consider tuberculosis in patients with AIDS

In the United States the epidemic of HIV infection has been blamed for the recent increase in tuberculosis from some areas because these increases have occurred among groups in which AIDS is also concentrated.[1,2] In Africa too, increases in tuberculosis have been reported from areas with high prevalences of both tuberculosis and HIV infection.[3] What is the connection between HIV infection and tuberculosis? Do the American findings have implications for Britain?

Studies in New York city showed that among patients with both tuberculosis and AIDS almost two thirds had developed tuberculosis within six months of their diagnosis of AIDS (E Laroone *et al*, International Conference on AIDS, Montreal, 1989).[4] Tuberculosis preceded the conditions that make up AIDS[5] by a median of two months, and similar findings have been reported from other studies.[6-8] HIV infection is a cofactor with one of the highest risk ratios for the development of tuberculosis in people already infected with *Mycobacterium tuberculosis*.[9]

Strong evidence that in people infected with HIV tuberculosis develops from the reactivation of a latent tuberculous infection comes from a follow up study of a cohort of injecting drug users whose tuberculin and HIV state was known.[10] Some 14% of those seropositive for HIV and with prior tuberculin sensitivity developed tuberculosis compared with only 0·3% of those seropositive for HIV but without tuberculin sensitivity. Rates of tuberculin conversion during follow up

were similar in both those who were seropositive for HIV and those who were seronegative.

The Advisory Committee for the Eliminaton of Tuberculosis in the United States has issued recommendations for managing people who have, or may have, infection with both HIV and *M tuberculosis* and their contacts.[11] It recommends tuberculin testing of all people infected with HIV so that preventive treatment may be offered to those who react to tuberculin, recognising that in some cases HIV related anergy will cause the tuberculin test to be falsely negative. It also recommends that all patients in whom tuberculosis is diagnosed should be offered HIV testing and that all people sensitive to tuberculin should be questioned about any risk of HIV infection. How appropriate are these recommendations in Britain?

Over the past 20-30 years the number of notifications of tuberculosis in England and Wales has continued to decline.[12] The crude total for 1988 was, however, 1·5% higher than the total for the previous year (A McCormick, personal communication), and this increase will be scrutinised when more detailed information is available. Nevertheless, no increases occurred in the rates of tuberculosis notifications up to 1988 in men aged 25-44 in south east England—the sex, age group, and region from which most cases of AIDS have been reported.[13] Nor has any association been observed between increases in tuberculosis notifications and cases of AIDS in London health districts.[13]

The likely incidence of tuberculosis in patients with HIV infection in Britain will depend on any overlap between the population infected with HIV and the population with previous tuberculous infection. This overlap is likely to be small. Almost two thirds of the 2649 patients with AIDS reported by the end of September 1989 were white men aged 25-44, whereas only 9% of patients with tuberculosis in the 1983 Medical Research Council survey were in this group (J Darbyshire, personal communication). The highest rates of tuberculosis in England and Wales are found among people whose families originated in the Indian subcontinent,[12] but only 1% of AIDS cases have been reported in Asian or Oriental ethnic groups. In the United States injecting drug users were at increased risk of developing tuberculosis even before the HIV epidemic,[14] but this has not been reported in Britain, and only 109 (4%) British patients with AIDS are injecting drug users. Increases in cases of tuberculosis associated with HIV infection have also been reported in prisoners in the United States[15] but have not been observed in Britain.[16]

Nevertheless, tuberculosis in patients with AIDS in Britain is well recognised.[17,18] Some 2% of the patients reported in the voluntary confidential reporting system to the Communicable Diseases Surveillance Centre and the Communicable Diseases (Scotland) Unit had tuberculosis at the time of reporting. More complete information comes from case series such as that reported by Helbert *et al* from St Mary's Hospital, London, where 13 (6%) of 207 patients with AIDS followed up between 1983 and 1988 had tuberculosis and another 12 (6%) had disseminated infections with other mycobacteria.[19] This proportion is similar to that seen in New York from 1985 to 1988, where 2·5-6·3% of registered patients with AIDS were also on the tuberculosis register (E Laroone *et al*, International Conference on AIDS, Montreal, 1989). So although the overlap between the population previously infected with tuberculosis in Britain and the population with HIV infection is unlikely to be large enough in the near future to have a major impact on the incidence of tuberculosis, clearly *M tuberculosis* is an important pathogen in patients with AIDS.

Therefore a diagnosis of tuberculosis should be considered in all patients with AIDS in Britain. As pulmonary disease due to tuberculosis cannot be distinguished from disease associated with the opportunist mycobacteria all patients with acid fast bacilli in their sputum should be given multiple drug chemotherapy effective against tuberculosis until the results of mycobacterial culture are known. Tuberculosis in HIV infection is more often extrapulmonary and disseminated than in the absence of HIV infection, and chest radiographs usually show diffuse or miliary infiltrates rather than focal lesions or cavitation.[20,21] Nevertheless, people with pulmonary tuberculosis and HIV infection may be as likely to spread tuberculosis to their close contacts (some of whom may be immunosuppressed) as their counterparts without HIV infection (S B Manoff *et al*, Epidemic Intelligence Service Conference, Atlanta, 1988). Contacts of people infected with HIV with smear positive pulmonary disease should therefore be examined and followed up according to the guidelines suggested for other contacts of tuberculosis even while awaiting the results of sputum culture.[22] Health workers caring for patients with HIV infection may also be at increased risk of tuberculosis[23] and should be offered protection.[22] As the diagnosis of tuberculosis may precede the development of conditions that indicate AIDS a history of HIV risk behaviour[24] should be taken from patients presenting with tuberculosis, particularly from sexually active adults, and HIV testing with counselling[25] should be offered.

The effect of previous vaccination with BCG in preventing the development of tuberculosis in patients infected with HIV is unknown, but disseminated BCG infection has occurred in those given BCG after contracting HIV infection.[26] It is not recommended in Britain that BCG should be given to people with HIV infection.[27] It would be prudent to give a tuberculin test to people infected with HIV who have not had BCG vaccination and offer prophylaxis to those sensitive to tuberculin. The correct action in those who have had BCG vaccination (usually denoted by the presence of a small vaccination scar) is less clear, and prospective studies of cohorts of people infected with HIV who may have had BCG vaccination as adolescents or infants are needed. In the meantime surveillance of HIV infection in Britain should be strengthened through the use of unlinked anonymous surveys, and the incidence of tuberculosis in different sections of the population, including ethnic groups, should be closely monitored.

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Medical audit

Closing the feedback loop is vital

Good clinicians have always organised some kind of systematic review of their daily work, recording and assessing the accuracy of their diagnosis and the outcome of their treatment. We have learnt to call this kind of activity audit.

Everyone now has to become an auditor; the NHS Bill requires general practitioners and hospital staff to engage in regular audit by 1991, the royal colleges and faculties require evidence of audit before accrediting posts for specialist training, and hospital managers seem to believe that audit will be the key to achieving their prime challenge—a high quality service at the lowest possible cost.

A lot of thinking, talking, and writing about audit is still, however, ill focused and vague. The word audit is not some sort of magic talisman that will change practice simply by its repetition. There are some basic principles, now generally agreed.[1] This week (p 85) the *BMJ* begins publication of a new regular series, Audit in Practice, which will, we hope, help readers to understand those principles and see how other groups of clinicians have been achieving audit. The section will include some submitted articles accepted after the normal editorial process of assessment and peer review; but there will also be commissioned articles explaining practical aspects of audit and a news and diary section prepared by the King's Fund Centre.

In deciding whether or not to publish an article describing medical audit in a hospital or general practice setting we shall look for specific features. As with any research study we shall need clear statements of why the project was started, what was done, what was found, and how the data were analysed. But the essence of audit is that it should be designed to achieve change. This is true whether the audit is of process (examining records and other data to find out how patients are being treated) or of outcome (looking at the results of treatment). The first stage in the audit is defining the standard that should be achieved (the proportion of children vaccinated or of adults having their blood pressure recorded, for example) or the pattern of investigation and treatment to be followed for patients with a defined condition (such as haematemesis and melaena in a patient with no previous episodes of bleeding). Next, the auditing group assesses how their performance has been measuring up to the agreed standard and the circumstances of any omissions or oversights.

From that assessment should come practical conclusions—how performance can be brought closer to the agreed standard or how the standard can be modified to improve outcome further. Next—and this is the crucial step so often omitted in reports submitted for publication—the conclusions should be agreed with the clinicians and put into practice. Finally, the audit must be repeated to ensure that change has occurred in the right direction. Without this "closing of the feedback loop" audit may be little more than a pious exercise in self congratulation.

Where do the standards come from? Sometimes they will be consensus statements, sometimes guidelines agreed by expert bodies such as colleges and faculties. In many cases the primary source will be the conclusions of formal prospective clinical trials. Sometimes a group of clinicians may find that the audit they had in mind cannot be started because there is no agreed protocol of management and their first task may then be to set up an appropriate trial. And what should be the priority topics for clinical audit? Here each group of doctors will make its own decisions, but data collected by community physicians will often provide a basis for identifying targets—in terms of patients to be screened or treated, mortality and morbidity, and so on.

All concerned have a lot to learn—and that includes the editorial team responsible for the new section. The format of audit articles seems likely to evolve, but at this stage we believe that most such articles should have a structured abstract setting out the purpose and design of the study, the conclusions reached, the action taken, and—ideally—the results of that action. The catchment population or number of participating hospitals or centres should also be given. Statements for revision of regional or district guidelines generally have less impact than recommendations to specified regional or national bodies. In addition, the first of this series includes a review of a clinical audit kit, and we shall be pleased to consider other similar material for future review in the section.

The whole process should be exciting and stimulating, and standards of care should improve simply by the process of being examined and questioned. Of course most clinical audits will not warrant publication as they will repeat work already done and reported elsewhere; but we should like to hear about any experiences—successes or failures—that may have practical lessons for others.

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Britain bans oral snuff

Government's action is tough and commendable

Just before Christmas the Department of Health announced a ban on oral snuff to come into effect in March 1990, under consumer protection legislation. The move will prohibit the supply of oral snuff (the best known brand is Skoal Bandits) and will mean the closure of the factory in Scotland originally built with the aid of a government grant. In announcing the ban the Secretary of State for Health, Kenneth Clarke,