suffer from greater underascertainment than we estimated the incidence of childhood diabetes would be correspondingly increased throughout the British Isles, indicating an even greater difference between the incidence in 1988 compared with that reported by Bloom *et al* for 1973-4.[2] The cause for public concern remains: urgency is added to the case for further national studies such as ours and multinational studies such as EURODIAB.

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1 Patterson CC, Smith PG, Webb J, Heasman MA, Mann JI. Geographical variation in the incidence of diabetes mellitus in Scottish children during the period 1977-1983. *Diabetic Med* 1988;5:160-5.
2 Bloom A, Hayes TM, Gamble DR. Register of newly diagnosed diabetic children. *BMJ* 1975;iii:580-3.

Implantable venous access devices

SIR,—The report by Mr M J Davis and colleagues[1] is consistent with our experience using implantable long term venous catheters. It is, however, important to note that such devices are not entirely without problems.

We recently implanted a Porta-Cath (Pharmacia) into the left subclavian vein of a 14 year old girl with cystic fibrosis. During the second course of intravenous antibiotics given with this device she presented with a three day history of a painful swollen left arm with dilated collateral veins over the left shoulder. The catheter site was immediately visualised by injecting 2 ml water soluble radiographic contrast medium and showed probable occlusion of the subclavian vein with perivascular leaking of contrast media. The catheter was removed and the patient given anticoagulation treatment initially with heparin and subsequently with warfarin. Adequate anticoagulation could not be achieved despite 24 mg warfarin daily, the international normalisation ratio remaining consistently less than 2·0, so she was started on subcutaneous heparin for three months. Five days after finishing heparin treatment she presented again with a swollen left arm; a venogram confirmed a left subclavian vein thrombosis, and heparin treatment was started again.

We agree with Mr Davis and colleagues that implantable venous catheters are to be recommended in patients with chronic lung disease in whom peripheral venous access has become difficult. It must be remembered, however, that any indwelling venous cannula system may lead to thrombosis or sepsis. Patients, parents, and nursing staff need to be aware of the potential complications of such devices and seek advice early when they arise.

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1 Davis MJ, Wilson RG, Keston M, Nixon SJ. An implantable device for long term venous access in chronic lung conditions. *BMJ* 1991;302:570-1. (9 March.)

SIR,—I dispute the contention of Mr M J Davies and colleagues that external catheters necessarily have a high rate of septic complications.[1] With appropriate catheter care the incidence is low; in our experience of prolonged parenteral nutrition it is around 0·3 episodes of infection per year of treatment.[2] Furthermore, without proper care the Intraport may become infected. I agree, however, that in comparison with external catheters there are significant benefits in lifestyle, and it is for this reason that we have used the Intraport in three young patients who require home parenteral nutrition. The experience of one of these patients highlights a potential hazard of this system.

A 27 year old man had received home parenteral nutrition for seven years on account of Crohn's disease and a short bowel. The external catheter that had been in place for three years occluded, and at his request an Intraport was inserted. He was trained to use the system, discharged after one week, and received nutrient infusions over four nights a week at home. During this time his health and nutritional state improved. Three months later he awoke at 3 am, after one litre of nutrient solution had been infused, with severe swelling of one side of his chest and face. The infusion was discontinued, and when he was assessed in hospital the swelling was subsiding; a catheter angiogram showed that the Intraport was intact, and no leaks were found. It was concluded that the Huber needle had not been properly located in the port, or that it had been adequately secured and had become displaced. The patient made an uneventful recovery and has continued his treatment at home with the same Intraport and no further problems.

One reason why our patient failed to insert the needle properly on this occasion may have been the improvement in his nutritional state, which meant that the port was covered with more adipose tissue and therefore more difficult to locate. Weight gain may also be expected in such patients with adequate control of infection. The extravasation of some antimicrobial and particularly cytotoxic solutions could have serious consequences. Nevertheless, I endorse the views expressed by Mr Davies and colleagues that this system provides convenient venous access for a variety of indications. The importance of adequate patient training for the proper insertion and anchorage of the needle merits emphasis.

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1 Davies MJ, Wilson RG, Keston M, Nixon SJ. An implantable device for long term venous access in chronic lung conditions. *BMJ* 1991;302:570-1. (9 March.)
2 Pennington CR. Towards safer parenteral nutrition. *Alimentary Pharmacology and Therapeutics* 1990;4:427-41.

SIR,—Mr M J Davies and colleagues report one episode of occlusion and one episode of catheter displacement in the use of 13 Intraport implanted venous access devices in patients with chronic lung disease for the delivery of intermittent courses of antibiotics.[1] They correctly point out a previously reported high incidence of problems with a metal port in a similar group of patients.[2]

At East Birmingham Hospital we have inserted 14 metal ports (two Portacath and 12 Vascuport) in 13 adult patients with cystic fibrosis over the past four and a half years (mean time with port in situ 12·3 months, range 0·5-56 months), 12 sited in the pectoral area and one in the groin. Over this period we have experienced only one episode of catheter occlusion; this occurred during treatments at home and required replacement of the system. We have had no episodes of catheter related sepsis or catheter displacement. We maintain system patency by monthly flushing with heparinised saline (100 U/ml); when the system is used to give antibiotics needles are replaced weekly.

Most patients complete courses of antibiotics at home, returning to hospital for removal of their Huber needle at the end of treatment. Several patients access their own ports for flushing at home. Both patients and staff in our unit find these devices highly acceptable. Our experience suggests that metal ports are no more prone to failure or complications than their plastic equivalents when they have been inserted by an experienced team and managed by well trained patients and staff.

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1 Davies MJ, Wilson RG, Keston M, Nixon SJ. An implantable device for long term venous access in chronic lung conditions. *BMJ* 1991;302:570-1. (9 March.)
2 Ball ABS, Duncan FR, Foster FJ, Davidson TI, Watkins RM, Hodson ME. Long term venous access using a totally implantable drug delivery system in patients with cystic fibrosis and bronchiectasis. *Respiratory Medicine* 1989;83:429-31.

Jarman index

SIR,—I was not being unduly modest, as Dr Roy Carr-Hill and Mr Trevor Sheldon suggest,[1] when I said that "The underprivileged area score attempts to develop a measure of general practitioners' opinions of factors that increase their workload or pressure on their services."[2] This is exactly why it was developed. I have given the history of the different methods that we considered using—a predetermined combination of appropriate social variables, measures of consultation rates, and measures of morbidity and mortality—and of why we eventually adopted the underprivileged area methodology.[3] I am sure that we would not have obtained the agreement of the General Medical Services Committee's underprivileged area subcommittee and successive annual conferences of local medical committees had we not used a national survey of general practitioners to choose and weight the variables and had we not found good agreement with independent local assessments of workload or pressure on services.[4]

It is true that the score has other correlates, which were not our main concern. Firstly, when indices of social deprivation were developed by combining selected social variables[5-7] they were found to correlate strongly with the underprivileged area score. Secondly, Curtis showed a linear increase in general practitioner consultation rates with underprivileged area score. I was surprised that the relation held for high scores as I would have expected consultation rates to decrease slightly in areas with very high scores because general practitioners are overwhelmed by inner city pressures and consequently unable to respond to demand. Thirdly, the underprivileged area score was found to have the highest or second highest correlation coefficient (for the district health authorities of England) of more than 100 social and health service provision variables with several internationally accepted measures of health status—infant, neonatal, and postneonatal mortality; death rates in childhood (age 0-5); and proportions of low birthweight babies.[3] Standardised mortality ratios, on the other hand, had higher correlations with individual social variables (such as the proportion in socioeconomic group 10 (semiskilled workers) than with any of the composite social variables.[4-7]

I was closely concerned with the work of Chase and Davies[8] to which Dr Carr-Hill and Mr Sheldon refer.[1] As Chase and Davies pointed out, the underprivileged area score of patients joining a list in one year could not be expected to be the same as the score of a whole practice. I have suggested that the 1991 census could be used to calculate a practice's underprivileged area score from the individuals' census data for comparison with the conventional method of calculation.[10]

I agree with Drs Joy and Paul Main that there can be anomalies, as, for example, with their practice, which is in a deprived part (Hartcliffe) of a large ward (Bishopsworth, population 25 000) which has an underprivileged area score under 30.[11] The enumeration district scores for the Hartcliffe part of Bishopsworth ward are much higher

than those for the rest of the ward and would qualify for deprivation payments if enumeration district scores were used. These difficulties could be reduced if deprivation payments were phased in more gradually, starting at a cut off score somewhere between 10 and 20.

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1 Carr-Hill R, Sheldon T. Jarman index. *BMJ* 1991;**302**:850. (6 April.)
2 Jarman B. Jarman index. *BMJ* 1991;**302**:527. (2 March.)
3 Jarman B. *Social deprivation and health service funding*. London: Imperial College, 1990. (Papers in science, technology, and medicine No 22.)
4 Jarman B. Underprivileged areas: validation and distribution of scores. *BMJ* 1984;**289**:1587-92.
5 Department of the Environment. *Urban deprivation*. London: DoE, 1983. (Information note No 2.)
6 Townsend P, Phillimore P, Beattie A. *Inequalities in health in the Northern region*. Newcastle: Northern Regional Health Authority and University of Bristol, 1986.
7 Carstairs V, Russell M. Deprivation: explaining differences in mortality between Scotland and England. *BMJ* 1989;**299**: 886-9.
8 Curtis SE. Use of survey data and small area statistics to assess the link between individual morbidity and neighbourhood deprivation. *J Epidemiol Community Health* 1990;**44**:62-8.
9 Chase HD, Davies PRT. Calculation of the underprivileged area score for a practice in inner London. *British Journal of General Practice* 1991;**41**:63-6.
10 Jarman B. General practice, the NHS review and social deprivation. *British Journal of General Practice* 1991:**41**:76-9.
11 Main J, Main P. Jarman index. *BMJ* 1991;**302**:850. (6 April.)

We cannot afford an AIDS epidemic

SIR,—Dr Mike Bailey states that the origins of the HIV epidemic in sub-Saharan African countries "lie in poor living conditions, education, nutrition, and access to health care and treatment mixed with the extremes of inequality found in developing countries."[1] He must realise, however, that by far the commonest method of transmitting HIV infection is through sexual intercourse and that this spread is facilitated by the presence of concomitant sexually transmitted infections, especially genital ulceration. The only way to control HIV infection in these countries is through health care programmes aimed at controlling the spread of all sexually transmitted diseases, preferably through the World Health Organisation's initiatives.

Dr Bailey concludes that the HIV epidemic is no different from any other disease of poverty and that its remedies must be seen to lie in the development of government health and social welfare sectors. HIV infection, however, is more common among the more affluent urban populations than the rural populations. The high cost of treating people positive for HIV is a drain on resources in developed countries and makes it unlikely that the same standard of treatment will be available to most of those infected in developing countries in the near future. Poverty is an important issue but must be kept separate from the issue of HIV infection, which places its own, separate demands on limited Third World resources.

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1 Bailey M. We cannot afford an AIDS epidemic. *BMJ* 1991;**302**: 726. (23 March.)

Changing disease patterns in AIDS

SIR,—Dr Barry S Peters and colleagues describe changing patterns in AIDS and attribute an increase in short term survival from 1986 primarily to a decrease in deaths from *Pneumocystis carinii* pneumonia.[1] Therapeutic advances have no doubt had a major role, but equally important may be a spectrum in the clinical course of HIV infection itself and the effect on this of changes in the sexual behaviour of infected men.

There is good evidence that the majority of gay men in the United Kingdom were infected between 1982 and 1984, when the prevalence among those attending clinics rose from 4% to 20%[2]: those who died earlier are likely to have been those most profoundly affected by the virus. Weber *et al* showed that unmodified sexual behaviour after acquiring infection potentiates morbidity.[3] Until 1984, when testing became available, the risk of superinfection as well as other sexually transmitted diseases would have been considerable. From 1984, there was a highly significant downward trend in the sexual behaviour of gay men.[4]

The improving prognosis of AIDS may not be entirely attributable to better management.

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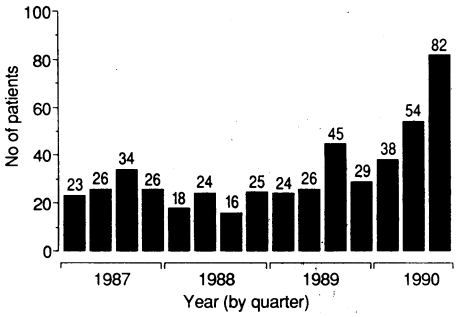
1 Peters BS, Coleman D, Beck EJ, *et al*. Changing disease pattern in AIDS. *BMJ* 1991;**302**:726. (23 March.)
2 Carne CA, Weller JVD, Sutherland S, *et al*. Rising prevalence of human T-lymphotropic virus type III (HTLV-III) infection in homosexual men in London. *Lancet* 1985;i:1261-2.
3 Weber JN, McCreaner A, Berrie E, *et al*. Factors affecting seropositivity to human T cell lymphotropic virus type III (HTLV-III) or lymphadenopathy associated virus (LAV) and progression of disease in sexual partners of patients with AIDS. *Genitourin Med* 1986;**62**:177-80.
4 Evans BA, McLean KA, Dawson SG, *et al*. Trends in sexual behaviour and risk factors for HIV infection among homosexual men, 1984-7. *BMJ* 1989;**298**:215-8.

Preventing the spread of HIV infection

SIR,—Dr A E Singaratnam and others reported an increase in the incidence of gonorrhoea among homosexual men in London.[1] We too have noted this increase, both at the Middlesex Hospital (figure) and at St Mary's Hospital, London.[2]



Number of men with urethral or rectal gonorrhoea, or both, at Middlesex Hospital, London, 1987-90

Though this may indicate continuing unprotected sexual intercourse in this population, Dr Singaratnam and colleagues do not indicate the sexual behaviour that led to gonorrhoea. We performed a collaborative prospective study at our centres from August to December 1990 inclusive, which included a detailed history of sexual behaviour during the four weeks before the onset of symptoms in 121 homosexual men who presented with gonorrhoea. Four weeks is long enough to include the sexual contact leading to infection in most men with urethral gonorrhoea.[3] Nine men had pharyngeal gonorrhoea alone and five men had both pharyngeal and urethral infection. Of the 68 men with urethral gonorrhoea alone, 31 gave insertive oral intercourse as their only risk factor for infection in the preceding four weeks. We have previously reported that rectal gonorrhoea can be acquired in the absence of penetrative anal intercourse.[4]

Knowledge of the mode of transmission of gonorrhoea was variable, whereas knowledge of transmission of HIV was good, with 120 men stating that unprotected receptive anal intercourse conferred a high risk of acquiring HIV. Of note is the fact that 70 men considered oral sexual intercourse to have a low a risk and 13 considered it to have no risk of transmitting HIV.

When counselling about safer sexual practices it is important to emphasise that, despite the low risk of transmission of HIV,[5,6] sexually transmitted diseases can be acquired by orogenital contact.

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1 Singaratnam AE, Boag F, Barton SE, Hawkins DA, Lawrence AG. Preventing the spread of HIV infection. *BMJ* 1991;**302**: 469. (23 February.)
2 French PD, Davis J, Beck E, Goldmeier D. Trends in gonococcal infections: no room for complacency. *Genitourin Med* 1990;**66**: 302.
3 Harrison WO, Hooper RR, Wiesner PJ, *et al*. A trial of minocycline given after exposure to prevent gonorrhoea. *N Engl J Med* 1979;**300**:1074.
4 Tomlinson DR, French PD, Harris JRW, Mercey DE. Does rectal gonorrhoea reflect unsafe sex? *Lancet* 1991;**337**:501.
5 Lifson AR, O'Malley PM, Hessol NA, Buchbinder SP, Cannon L, Rutherford GW. HIV serconversion in two men after receptive oral intercourse with ejaculation: implication for counselling concerning safe sex practices. *Am J Public Health* 1990;**80**:1509-11.
6 Rozenbaum W, Gharakhanian S, Conlan B, Duval E, Coupland JP. HIV transmission by oral sex. *Lancet* 1988;i:1395.

Preventing needlestick injuries

SIR,—In conjunction with other guidelines,[1] my two rules designed to prevent needlestick injury, particularly in general practice, are:

(1) Never hurry. If you are rushed do not attempt venepuncture, simply postpone it to a more convenient time.

(2)Ensure on every occasion that a sharps container is adjacent to or preferably below the patient, such that the needle is removed and swiftly but smoothly placed in the box. No sharps box, no venepuncture.

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1 Anderson DC, Blower AL, Packer JMV, Ganguli LA. Preventing needlestick injuries. *BMJ* 1991;**302**:769-70.

Organophosphorus poisoning

SIR,—Dr T J Hodgetts has given a management strategy for organophosphorus poisoning.[1] Consideration of the mechanisms of organophosphorus toxicity leads us to believe that pralidoxime treatment should be continued for as long as the organophosphorus compound or its active metabolite is present in the body. It is generally accepted that plasma pralidoxime concentrations of 4 mg/l are necessary to achieve a satisfactory therapeutic effect in severe cases of organophosphorus intoxication. This is based on experiments in anaesthetised cats given lethal doses of intravenous sarin[2]; plasma pralidoxime concentrations of 4 mg/l were required to counteract neuromuscular block, bradycardia, hypotension, and respiratory failure. More recent studies have also supported this conclusion.[3,4] To achieve plasma pralidoxime concentrations of 4 mg/l it is necessary to administer a pralidoxime salt (in the United Kingdom *N*-methylpyridinium-2-aldoxime