that index patients are in later stages of the disease is not taken into account.

The observed interaction between sex related efficiency of transmission of HIV and the stage of disease of the index patient was based on a rough classification of disease stages (presence of clinical symptoms and T4 counts over or below $0.2 \times 10^9/l$). It is thus possible that men and women considered at an early stage, even with no difference in mean T4 counts ($0.643 \times 10^9/l$ v $0.625 \times 10^9/l$ respectively, $p=0.71$), were not strictly comparable. Unfortunately, more precise data such as duration of infection were not available.

I agree that the exact knowledge of periods of exposure to the potential risk factors studied, related to the level of immunodeficiency of the index patients, would be of great interest. But dates of HIV infection and dates of appreciable immunological deterioration (for example, T4 count below $0.2 \times 10^9/l$) are needed to define the periods of exposure, and these dates are rarely available. Furthermore, the study was implemented in early 1987, when little information was available regarding heterosexual transmission except that it existed. Thus, ideal data to be recorded were difficult to define. The follow up data will be more precise. Because of intensive counselling, however, we do not expect many seroconversions and thus the power of the statistical analysis will probably be low.

In research on this subject, in which recruitment is difficult, ideal data cannot be expected so results should be interpreted with caution. Such cross sectional studies of partners, however, are one of the main sources of information on the effectiveness of, and risk factors for, heterosexual transmission.

ISABELLE DE VINCENZI

European Centre for the
Epidemiological Monitoring of AIDS,
Hôpital National de Saint-Maurice,
94410 Saint-Maurice,
France

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Incidents involving exposure to blood

EDITOR,—Katie Oakley and colleagues¹ provide excellent practical guidance on implementing the BMA's code of practice for the safe use and disposal of sharps.² Staff are often unsure about risk assessments to be made after accidental exposure to blood or sharps injury. Knowledge about national and demographic differences in the prevalence of infection may be important as any infection control policy may need to cater for local situations.^{3,4}

To gauge health care workers' knowledge about prevalence of infection in the United Kingdom as a whole I used a self assessment questionnaire with three groups of health care staff, including doctors, nurses, laboratory staff, and clinical medical students from various areas of the United Kingdom. Respondents were asked to estimate the number of confirmed HIV positive people, the prevalence of hepatitis B virus (carrier state) in the United Kingdom, and the annual number of occupationally acquired hepatitis B infections in British health care workers. All respondents were at risk of infection from their work by virtue of regular exposure to patients' blood or used sharps. Responses were judged against the current data published by the Public Health Laboratory Service—that is, 50 000 carriers of hepatitis B virus, 15 500 HIV seropositive people, and an annual rate of 35 cases of hepatitis B virus infection in health care workers from occupational exposure.

Of 116 respondents, 98 provided answers for the number of HIV positive people, 91 for carriage rate

Estimated population prevalence of HIV, hepatitis B virus, and occupational infections with hepatitis B virus in health care staff in United Kingdom. (Bars show actual prevalence)

of hepatitis B virus, and only 81 estimated the number of occupational infections with hepatitis B virus. The results varied considerably (figure). Only seven knew the correct number of HIV seropositive people, 14 gave a correct estimate for carriage rate of hepatitis B virus, but no one estimated exactly the number of occupational cases of HBV infection. The range of answers provided by respondents suggests that staff, even after many years of experience in health care work, may have little accurate knowledge about the prevalence of infection in the United Kingdom as a whole and, in the case of occupational infection with hepatitis B virus, tended to overestimate considerably the infection rates. This was not reflected in their individual practices for handling needles or their hepatitis B virus immunisation status; of the sample, only about a half had been immunised and many often resheathed used needles.

Gerberding and colleagues found that surgical staff in San Francisco General Hospital have a lower risk of intraoperative exposure to infections because they recognise the high prevalence of bloodborne pathogens in their surgical patients.⁵ As a result they practise a high standard of infection control with safer handling of needles and other sharp instruments and use protective clothing. Clearly if the principles of infection control are to be practised effectively health authorities must ensure that staff have an adequate knowledge of background information, such as data on the prevalence of infection; are fully trained in infection control procedures²; and receive regular refresher courses to update them as the AIDS epidemic progresses.

DAVID R MORGAN

Head of Scientific Affairs,
British Medical Association,
London WC1H 9JP

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EDITOR,—Proper management of sharps injuries requires that they are reported promptly by the staff who sustain them. In their review of the management of incidents involving exposure to blood Katie Oakley and colleagues allude to the importance of reporting injuries, though they give

no indication of the magnitude of the problem.¹ In a recent cross sectional survey of doctors employed by South Glamorgan Health Authority we found that many continue to pay scant regard to the reporting of needlestick injuries despite the risk of hepatitis B and HIV infection.

An earlier postal survey of all 684 doctors employed by the authority indicated that 145 had sustained a sharps injury (undefined) during the six months August 1990 to February 1991. We sent a follow up questionnaire to these 145 doctors to find out more. This concentrated on needlestick injuries and asked for details on the number of incidents, the mechanism of injury, and the action subsequently taken. A needlestick injury was defined as "a penetrating injury with a hollow needle which had been contaminated with a patient's blood."

We received replies from 94 of the 115 doctors who could be traced (response rate 82%). Twenty five respondents reported having sustained 42 needlestick injuries during the six months of the survey. Twelve had had more than one injury, including one doctor who had sustained four. Only one respondent had reported the injury to the occupational health service, suggesting a rate of underreporting of over 95%. The reasons given for non-reporting among the 25 respondents who had sustained a needlestick injury were: low risk patient (22 respondents), not important (18), too busy (17), unsure of procedure (14), could not be bothered (nine), felt stupid about injury (five), and injury occurred out of hours (four).

Reporting of sharps injuries serves three important functions. Firstly, it ensures speedy and appropriate management of the injury. Secondly, it provides documentation of the incident should the question of compensation for occupational injury arise in the future. Thirdly, it is a means of epidemiological surveillance, allowing estimates of the size of the problem and targeting and evaluation of preventive measures.

The low reporting rate of under 5% is similar to that found in the United States.² Many doctors seem complacent, while others are misguided. Doctors' perceptions of low risk patients have been shown to be inaccurate.³ Doctors clearly need to be better educated about the importance and desirability of reporting all sharps injuries. Without this, prophylaxis after exposure, counselling, and follow up will be inadequate.

PETER D DONNELLY
MEIRION R EVANS

Department of Public Health Medicine,
South Glamorgan Health Authority,
Cardiff CF1 3NW

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Row over Maori cot death rate

EDITOR,—The report by Charlotte Paul entitled "Row over Maori cot death rate"¹ warrants comment. Unfortunately the title and first paragraph deflect attention away from the success of the national cot death prevention programme in New Zealand,² which has seen the national total post-neonatal mortality fall from 5.9/1000 in 1989 to 4.0/1000 in 1991 and the cot death rate from 4.0/1000 to 2.2-2.4/1000 (there are 13 deaths awaiting the coroner's findings).

The programme was based on the results of the first year of the three year case-control study.³ Three risk factors were targeted—namely, maternal smoking, prone sleeping position, and not

breast feeding. A fourth modifiable risk factor, infants sharing the bed with another person, was identified and incorporated into the programme.¹

The recent debate concerning cot death in New Zealand relates to the possible reasons for the higher cot death rate in Maoris compared with non-Maoris; the possible reasons why the Maori cot death rate has not decreased substantially, as has occurred in non-Maoris; and what can be done to improve the Maori rate. The Maori cot death rate has been known to be two to three times that of non-Maoris, but has not previously been explained. At no time in the present debate has anyone suggested that the Maori rate is 20 to 40 times higher than the non-Maori rate.

The higher Maori rate can be explained. The prevalence of maternal smoking in pregnancy is appreciably higher in Maoris than in non-Maoris (62.4% and 22.7% respectively). Relative attributable risk calculations suggest that increased smoking by Maori mothers explains about half (57%) of the excess cot death risk among Maoris compared with non-Maoris. At the second international conference on the sudden infant death syndrome in Sydney (12-16 February 1992) we presented data showing that the risk factors for cot death in Maoris were similar to those in non-Maoris and that the higher cot death rate could be largely explained by a higher prevalence of many of the known risk factors in Maoris.

Despite the overall success of the prevention programme it has been of little benefit to Maoris. This has been debated both by Maori and non-Maori communities. The consensus view is that to reach Maoris there must be a system which will send a "Maori message" (culturally appropriate information and education) via a "Maori messenger" (Maori community health workers). We hope funding will be forthcoming to extend the programme.

E A MITCHELL

Department of Paediatrics,
School of Medicine,
University of Auckland,
Auckland, New Zealand

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What counts as cot death?

EDITOR,—Sylvia R Limerick and Angela Gardner suggest¹ that when the incidence of cot death in England and Wales is being monitored the figures issued by the Office of Population Censuses and Surveys for "sudden infant death syndrome—any mention"² should be used. Though I agree that we should all try to use the same figures, I cannot admire their choice. The table shows the possibilities in a slightly different way.

Definition A is their choice, but it includes some

explained deaths (such as tracheobronchitis) and does not have an ICD number, and the figures appear at two year intervals and, as Limerick and Gardner say, are not yet available for regions and districts.

Definition B gives the figures for ICD code 798.0.³ This represents what remains after the explained deaths have been reallocated to their correct category. But so does definition C, and the numbers are the same. There is no real need for code 798.0.

Definition C—ICD code 798 is called "sudden infant death—cause unknown."⁴ This is an excellent description of the phenomenon we are trying to measure (syndrome it never was). If we add to it the words "cot death" this gives a shorter and more popular version. Two names for the same condition always confuse. One difficulty with definition C is that these figures, though available for regions, are not available for districts.

Definition D—ICD codes 790-799⁴ are unfortunately named "signs, symptoms, and ill defined conditions" because they apply to all ages.⁴ The youngest group is 28 days to 1 year—the postneonatal period. This is fortunate as this is the best age for the study of cot death, for the neonatal variety is much less common and, on general principles, may have different causes. Total numbers for definitions C and D are slightly different, but the rates are identical. This is because ICD code 798 constitutes 99% of codes 780-799. On average, only 14 deaths a year in definition D are not coded as 798, and they have such vague diagnoses that they could easily be cot deaths. So for practical purposes, definitions C and D are the same. Definition D has the advantage of having been available at district level since 1981.

My suggestion is that we should no longer use the word syndrome in relation to sudden infant death and should use only cot death. We should no longer use definitions A and B for monitoring but should use C and D interchangeably as cot death; once they are identical in every way we should omit definition D.

M R GORDON

Halstead,
Essex CO9 1SF

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Cot death and sleeping position

EDITOR,—When I was a baby I was nursed, like most of my generation, on my back; my mother knew that this was the best way to nurse a baby. My babies were all nursed on their tummies: having been a senior house officer in special care baby units I knew that this was the best thing to do

and wasn't going to listen to any advice from my mother on the subject. As one of my babies died of a cot death I now wish that I had done. Doubtless the 500 or so other families who, it would seem (from population attributable risk estimates), have lost babies for this reason every year for the past two or three decades wish the same.

In the increasing literature that has been written on cot death and sleeping position I have yet to see an analysis of how this remarkable shift in knowledge and behaviour was achieved. Nobody seems to know how it happened. Surely the phenomenon deserves to be studied. There must be lessons we could learn that would help prevent such a tragedy happening again. There must also be lessons we could learn about how to effect change in other behaviours that have been well researched and shown to be beneficial to health.

I have my theories on the matter but would welcome other people's perceptions. It seems to me that it all started in the late 1960s and 1970s, when special care baby units appeared and paediatricians discovered that very small babies in incubators had fewer apnoeic attacks when nursed on their tummies. They assumed that this must be true for all babies, and their nursing colleagues accepted their superior knowledge. Gradually, as a result, midwives started putting normal babies on their tummies and mothers in delivery units learnt from them that this was the best thing to do.

If this was the case a golden rule was broken and the medical profession should accept collective responsibility for what happened. The golden rule states that it is wrong to extrapolate the results of studies on small sick subsets of the population to formulate policy for improving health in the population as a whole. It is a monument to the enormous power that doctors wield that they could in such a short time and on such slender evidence influence the care of normal babies so profoundly and to such devastating effect.

SARAH STEWART-BROWN

Department of Public Health Medicine,
Worcester and District Health Authority,
Worcester WR4 9RW

Cerebral palsy and intrapartum events

EDITOR,—Clare Dyer makes the astonishing claim that 10% of cases of cerebral palsy are due to obstetric mismanagement.¹ Though most studies seem to suggest that 90% of cases are related to events other than labour and birth,^{2,3} it is quite another thing to assume that the remaining 10% are due to obstetric mismanagement. Though profound and prolonged hypoxia is probably a central factor contributing to the 10% of cases related to intrapartum events, whether there are important contributing cofactors remains to be seen.

Severe intrapartum hypoxia can occur well before admission to hospital and regardless of obstetric management. Causes of this include placental abruption and cord prolapse.

As a profession we have made progress in our understanding of the relatively small proportion of cases of cerebral palsy that are related to intrapartum events. It is unfortunate that Dyer does not differentiate between severe intrapartum hypoxia and obstetric mismanagement.

J L REYNOLDS

Department of Family Medicine,
University of Western Ontario,
London,
Ontario,
Canada

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*Not yet available.