asked her to touch his penis. The sweets were a bribe to keep the secret. I was shocked. Was this child sexual abuse in our house? Only a few weeks earlier I had attended a day's workshop on child sexual abuse and learnt that it usually occurred within the household—perpetrated by an older member of the family who often used bribes.

I decided that I must challenge my son straight away, and the same evening I questioned him in the most non-accusatory voice I could muster. He didn't say much and denied the part involving my daughter touching him. I wasn't too surprised so did not press him further but concluded our talk with the advice that it was not appropriate to do this with his sister, who was much younger than him. From then on I made sure the two of them were not left alone together. I was alone at the time as my husband was working abroad. I shared my concern with a close friend (a paediatrician and mother), who supported my action. When I did tell my husband on his return I found him unhelpful. He would make no comment, nor would he talk further to our son. However, he has never talked to our son about sexual concerns, and when our son was 17 and met a girl it was I who watched the relationship develop into a caring and sexual one, I who advised on birth control, and I who insisted that they did not sleep in the same bedroom in our house.

We have all undergone our sexual development and have all had sexual experiences good and bad. We are talking about it more, especially in schools, but sadly not enough—not enough to little children when they are receptive to discussion about their bodies, not enough to growing children so that they know they can talk about what is happening to them, and not enough so that when they are adult they can confide in others if they are having problems. Perhaps if more families could be more open about sexuality there would be fewer problems of sexual and related abuse. These problems could and should be prevented.

1 Stop, look, listen. *BMJ* 1992;305:838-9. (3 October.)

Site of injection for vaccination

EDITOR,—Mark Henley correctly states that the anterolateral aspect of the thigh is an acceptable alternative to the upper arm for injecting hepatitis B vaccine.¹ But his assertion that the only indications for administering the vaccine in the upper arm are convention and convenience is misleading. The reason why public health bodies recommend that hepatitis B vaccination should be given in the upper arm is to prevent vaccine being administered into the buttock.

There are over 100 reports of unexpectedly low antibody seroconversion rates after hepatitis B vaccination by injection into the buttock, confirmed by studies carried out at the Royal Free Hospital.² In one centre in the United States a low antibody response was noted in 54% of healthy adult health care staff. Many studies have since shown the antibody response rate to be significantly higher in centres using deltoid injection than centres using the buttock. On the basis of antibody tests after vaccination, the advisory committee on immunisation practices of the Centers for Disease Control recommended that the arm be used as the site for hepatitis B vaccination in adults,³ as have the departments of health in the United Kingdom.⁴

A more recent comprehensive study by Shaw *et al* showed that participants who received the vaccine in the deltoid had antibody titres that were up to 17 times higher than those of subjects who received the injections into the buttock.⁵ Furthermore, those who were injected in the buttock were two to four times more likely to fail to reach a minimum antibody level of 10 mIU/ml after vaccination. Recent reports have also implicated

buttock injection as a possible factor in a failure of rabies postexposure prophylaxis using human diploid cells rabies vaccine.⁶

The injection of vaccine into deep fat in the buttocks is thought likely with needles shorter than 5 cm, and there is a lack of phagocytic or antigen presenting cells in layers of fat. Another factor may involve the rapidity with which antigen becomes available to the circulation from deposition in fat, leading to delay in processing by macrophages and eventually presentation to T and B cells. An additional factor may be denaturation by enzymes of antigen which has remained in fat for hours or days. The importance of these factors is supported by the finding both at the Royal Free Hospital and by Shaw *et al*⁵ that thicker skinfold was associated with a lowered antibody response.

These observations have important public health implications, well illustrated by the estimate that about 20% of subjects immunised against hepatitis B via the buttock in the United States by March 1985 (about 60 000 people) failed to attain a minimum level of antibody of 10 mIU/ml and were therefore not protected.

We strongly urge that hepatitis B surface antibody titres should be measured in all people who have been immunised against hepatitis B by injection into the buttocks, and when this is not possible a complete course of three injections of vaccine should be administered into the deltoid muscle or the anterolateral aspect of the thigh, the only acceptable sites for hepatitis B immunisation.^{7,8}

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Dyspnoea in palliative care

EDITOR,—Opioid drugs used to control both pain and dyspnoea in palliative care do not, by any means, always hasten death. A recent review of the treatment of intractable dyspnoea is both informative and interesting¹ but, as Minerva points out, makes "rather depressing" reading.² The paper is not, in fact, a comprehensive review of the literature.

Though there is good evidence that single doses of morphine cause a deterioration in blood gas concentrations, the same is not true for long term treatment. Gray *et al* reported no difference in opiate consumption between patients with chronic lung disease who developed respiratory failure and those who did not.³ Furthermore, in a study of 20 terminally ill patients (including 12 with chronic airflow limitation) receiving regular oral morphine in doses exceeding 100 mg morphine sulphate/24 h Walsh found a raised arterial carbon dioxide concentration in only one.⁴ New routes of administration have been investigated, and anecdotal

evidence and one published report indicate the efficacy of low dose nebulised morphine in breathlessness.⁵

In palliative care dyspnoea is often regarded as the poor relation to pain. The results of current research, however, are encouraging and should lead to therapeutic advances and improvements in patients' quality of life.

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Faecal incontinence

EDITOR,—E S Kiff's article on faecal incontinence rightly says that impaction of faeces in elderly people is a cause of faecal incontinence and diarrhoea.¹ A diagram indicates that impaction is caused by hard faeces in the rectum. This is often so, but in my long experience of geriatrics I have found "soft" impaction—in which the rectum and colon are distended by soft faeces—to be probably more common. Presumably peristalsis is ineffective in this condition. Beside overflow, distention, vomiting, general deterioration, and excessive gas shadows can occur. Stimulant laxatives aggravate the symptoms and should be avoided until the bowel is cleaned from below. Small volume enemas are less effective than soap and water. The bowel may take over a week to clean, even with enemas on alternate days.

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Neuroleptic sensitivity in dementia with cortical Lewy bodies

EDITOR,—Ian McKeith and colleagues' paper¹ adds to the published clinical data on dementia with cortical Lewy bodies.² The authors make no comment on the severity and frequency of parkinsonian symptoms in dementia with cortical Lewy bodies found by other groups,^{3,4} although four of their 20 patients presented with Parkinson's disease—a proportion similar to the 14-40% reported previously.^{3,4} In the later stages of the disease parkinsonian symptoms are reported in most cases (70-87%).^{3,4} Parkinsonian symptoms responsive to levodopa have also been reported in young patients with the disease.^{6,7} We recently confirmed the severity of such symptoms and their responses to levodopa.⁸

With such a high frequency of important parkinsonian symptoms it is perhaps not surprising that the patients reported on by McKeith and colleagues were sensitive to neuroleptic drugs. Most of the patients were elderly and were receiving fairly high doses of neuroleptics. We have not observed sensitivity to such drugs in our patients—either those in whom dementia with cortical Lewy bodies was diagnosed after death³ or living patients in whom the diagnosis was made according to

unvalidated operational clinical criteria, including parkinsonian symptoms (S Williams *et al*, unpublished study).

We suggest that sensitivity to neuroleptics in patients with dementia with cortical Lewy bodies is further evidence of the importance of the parkinsonian component of the condition and a reminder that the dosages in neuroleptic regimens for elderly people are largely determined empirically. We advocate much lower doses of such drugs in routine psychogeriatric practice.

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Risk of HIV infection from blood transfusion

EDITOR,—Dominic Savarit and colleagues show that, in an area of west Africa having a high HIV prevalence, a random first time blood donor has a probability of 1·8/1000 to 3·8/1000 of being in the initial phase of HIV infection. This “window period” is not detected by standard HIV tests as there is no detectable antibody and so remains a serious hazard. We have confirmed these results using an alternate methodology and have also developed a simple equation linking the probability that an HIV carrier is in the “window” (p_w) with the rate of spread of HIV. We treat the incubation period of untreated HIV as having four successive phases, giving rise to a 10·8 year median incubation period. These phases correspond to initial infection, CD4 counts > 500, CD4 counts 200-500, and CD4 counts < 200. The associated infectivities are bimodal, being raised during the first and fourth phases. The mean durations of the phases are 1·5 months, 2 years, 10 years, and 1·5 years.²⁻⁴

This leads to the simple differential equation $dN_1/dt = \alpha N - N_1/\beta$ where N_1 is the number of HIV carriers in the “window”; N is the total number of carriers; α is the ratio between the monthly number of new infections and the number of carriers; αN is the number of new infections per month; and β is the duration of the “window” (1·5 months). In a mature epidemic ($dN_1/Ndt \approx 0$), yielding the deceptively simple relation $p_w = N_1/N = \alpha \times \beta = 1 \cdot 5 \alpha$. In western Africa the estimated doubling time of the epidemic is of the order of four years, which is consistent with a 2·5% seroconversion rate among repeat donors and a nominal prevalence rate of 10%.¹ This translates into $\alpha \approx 0 \cdot 015$ infections per carrier per month, so that $p_w \approx 0 \cdot 023$. The fraction of random blood donors who are infected with HIV and who are in the window is given by the equation $f_x = p_w f_{hiv}$, where f_{hiv} is the fraction of donors testing positive for HIV.

In Abidjan, the fraction of first time donors who

test HIV positive is 0·11, giving an estimate that 2·5/1000 random blood donors are in the “window period” of HIV infection, which agrees with Savarit's estimates. In the United States, in 1991, $\alpha \approx 0 \cdot 01$ ^{2,3} and $f_{hiv} = 1/5000$ so that 1/300 000 blood donors would be in the initial phase of infection (the window). In 1987 this number was about 1/100 000.

Not only do we corroborate Savarit's analysis but in addition we show the linkages between the fraction of carriers who are antibody negative and the monthly rate of new HIV infections. It is theoretically feasible to compute the instantaneous growth rate of the epidemic by estimating the fraction of carriers in the window. This could be done by testing serum samples negative for antibodies to HIV with the polymerase chain reaction to detect the presence of the virus. The probability that a carrier is in the window (p_w) is simply the ratio of the number who are positive with the polymerase chain reaction though negative for HIV to the number who are either positive with the polymerase chain reaction or positive for HIV antibodies. This approach could provide a method of determining the effectiveness of interventional strategies.

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AIDS, ethics, and clinical trials

EDITOR,—The statement of the Institute of Medical Ethics working party on the ethical implications of AIDS¹ provides a helpful and constructive overview of the challenges to clinical research conventions posed by the new circumstances of the HIV epidemic. However, it may be incorrect in its assessment of the part played by the parallel track mechanism which provided early access to didanosine and, more recently, to d4T (stavudine).²

The statement argues that expanded access to didanosine contributed to the lack of firm evidence of clinical benefit from this drug, implying that the development of and accrual to formal clinical studies of didanosine were hindered by its release for compassionate use. This is not the case. Daniel Hoth, director of the Division of AIDS at the National Institute of Allergy and Infectious Diseases, reported that “the trials’ entry rates are roughly equivalent to those seen in AZT trials.”³ The only patients eligible to receive didanosine under the compassionate release programme were those who did not meet the rather restrictive entry criteria for the formal clinical trials. If those trials had been better designed more patients would have been eligible and more data on the clinical efficacy of didanosine might now be available. In any event, the expanded access programme provided important safety data and contributed to the approval of didanosine “earlier in the drug approval process than any AIDS drug in history” (D Barr *et al*, VIII international conference on AIDS, Amsterdam, 1992).

This is not to say that the parallel track system was without faults. Patient advocates have suggested that expanded access programmes could be improved by including an element of randomisation

and dose comparison and by collecting data on clinical events. Activists accept that “the key to achieving faster drug development in AIDS lies not in an exclusive focus on expanded access or parallel tracks, but rather on their integration into an enlightened program of rapid, flexible, humane and attractive clinical trials.”⁴

In addition, I am not aware that the lack of patient interest in the optional placebo arm of the MRC's alpha trial of didanosine has led to increased demand from people with HIV and their advocates for more traditional placebo controlled studies. Rather, it indicates that many people with HIV participate in clinical trials as a means of access to experimental therapies, rather than for the interest of science and society. The challenge facing investigators is to design trials which offer valid treatment options in every arm while retaining the ability to produce reliable efficacy data. “Unless every arm of a clinical trial is a viable treatment option, that trial is fundamentally impractical and unethical.”⁵

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Learning from “the South”

EDITOR,—The editorial¹ marking the 50th anniversary of Oxfam emphasised the need for health workers in countries like Britain to learn from the experiences of those in developing countries. As Garner states, throughout the Third World paramedics such as medical assistants and clinical officers are responsible for most of the hands on medical care. His few examples of their capabilities give a falsely rosy impression of general reality.

In over 25 years of working in India, Africa, and the Middle East I have seen these reasonably well trained and initially enthusiastic people packed off into what is often a foreign part of their country and starved—of drugs and other supplies, facilities, literature, refresher training, and, most important of all, hope of advancement. A visit to a school of health sciences in central Africa a few months ago confirmed that my impressions are not outdated. Primary health care has been emphasised to the neglect of secondary care.

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Persistent vegetative state

EDITOR,—The opinion of the medical ethics committee of the BMA that artificial feeding of patients in the persistent vegetative state is a medical treatment¹ deserves further scrutiny.

The purpose of medical treatment is to cure, remedy, or palliate clinically diagnosable conditions. Nutrition and fluids do not cure any clinically diagnosable condition; they meet the need of the body for basic resources. Although hyper-alimentation is a form of assisted feeding that may be regarded as an ordinary form of medical treatment, other forms can be maintained in most