deficiency syndrome (AIDS) and others concerned in the AIDS industry.

There seems, however, to be some muddled thinking about this matter. A life insurance policy is not another aspect of state funded social security but a straightforward financial transaction. In the past it was understood that an application for an insurance policy, even for a few hundred pounds, would necessitate a full medical examination. If some life shortening condition was found it was equally understood that cover might be refused or accepted only on increased terms. I assume that a detailed medical examination has been found to be uneconomical for smaller policies, but, presumably, this does not imply the right to defraud the company.

In discussing the test for antibodies to the human immunodeficiency virus (HIV), some counsellors have been suggesting that the subject should seek (perhaps additional) life insurance before the blood test is performed. This is surely no more ethical than advising an overweight 40 year old man with polydipsia to increase his life insurance before his fasting blood glucose concentration is investigated.

Life insurance companies owe it to their shareholders and policyholders (particularly those holding "with profit" policies) to take all possible care to exclude poor risk lives—for whatever reason. We keep hearing of the "rights" of various groups in our society. A carrier of HIV antibodies has every right to advice, civilised handling at all times, and, when necessary, full medical treatment. He or she does not have the right to cheat insurance companies.

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Infantile apnoea and home monitoring

SIR,—Dr Peter G F Swift's letter (11 July, p 118) about infantile apnoea and home monitoring clearly reflects his belief in the value of alarms. We agree that alarms may be useful in selected patients (and parents), but some points need clarification. Children have died while being monitored despite the alarm responding,¹ and, as Dr Swift says, this must be explained to the parents. Though Dr Swift has encountered no deaths during home monitoring so far, Emery *et al* have already pointed out that it will still take over 100 years to prove scientifically whether apnoea alarms save lives.²

Dr D M Reid (p 119) correctly reminds us that for parents scientific proof is not the only issue. Nevertheless, Dr Swift's statement that monitors "helped many parents to sleep, reduced overt anxiety, and improved parent-child interaction" must be challenged, particularly as five families subsequently declined a second monitor. No monitor is trouble free, and comprehensive support arrangements must be arranged. Our own experience of 70 monitored children (32 siblings and 38 infants who had experienced severe "apparent life threatening events") showed that almost half of the parents needed to contact the hospital at some time during monitoring for mechanical or medical problems.³ This was despite frequent outpatient and telephone contact, especially during the first weeks of monitoring.

The importance of the National Institutes of Health consensus report is, as Professor Hamish Simpson (30 May, p 1367) states, that for the first time the use of apnoea alarms is considered for other conditions than sudden infant death syndrome.⁴ We welcome this as we believe that it is necessary to analyse the reasons why an alarm is being used. Attacks of lifelessness requiring vigorous or mouth to mouth resuscitation in infants who have suffered severe apparent life threatening

events have a high risk of recurrence (40%) and probably of causing sudden death.^{5,6} All such infants should thus receive a home apnoea alarm.

In our experience, however, most parents of siblings of victims of the sudden infant death syndrome do not need a home alarm. The risks to a sibling have probably been exaggerated in the past.⁷ The syndrome and the use of monitors are fully explained and any parental worries dealt with during pregnancy and before discharge. The child is reviewed regularly, especially around the time of the previous infant's death, and parents of such infants may contact the hospital if they are anxious.

If the parents cannot cope or sleep because of their fear of the sudden infant death syndrome despite our reassurance they are given a monitor. The lack of scientific proof should never deny such parents an alarm, for their sake as much as for the child's. Home monitors are not, however, the perfect answer, and yet many parents now feel guilty if they do not use an alarm "just in case something happens." Dr Swift, in stating that "it is surprising that many paediatricians are reluctant to provide respiratory monitors for use at home," puts similar pressure on paediatricians. Clinical judgment and the lack of local finances for monitors and follow up may mean that the correct decision is not to provide an alarm. The clearest message from the National Institutes of Health is that, despite the difficulties, it is time for scientific evaluation of the monitors and psychological problems related to them so that the correct decisions may be made, based on fact not emotion.

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SIR,—I agree with many of the points raised by Dr Peter G F Swift (11 July, p 118) regarding the possible benefits of home apnoea monitoring in siblings of victims of the sudden infant death syndrome and infants who have suffered previous apnoeic attacks.

Since 1980 we have monitored 288 infants (244 siblings of victims and 44 infants with apnoea) using the monitor described by Dr Swift. Like him, we have found that most families have found monitoring helpful: 28% of the families in which one child has died as a result of the syndrome have requested a second monitor and six families have used a third. Only three families to date have declined a second monitor.

Several points regarding the issuing of home monitors merit further emphasis. It must be explained clearly to all parents that there is absolutely no guarantee of infant survival. In our experience, most parents can accept this. Parents should be shown how to use the monitor, and possible advantages and disadvantages should be clearly explained, preferably before a final decision about monitoring is made. If monitoring is to be undertaken all parents should be instructed in basic resuscitation techniques, and there must be an adequate 24 hour back up service to deal with any problems that may arise.

I would, however, disagree with Dr Swift on one point. There is at least one recorded case,¹ and several anecdotal cases, of death occurring while an infant was being monitored. In our experience this has occurred only once to date, in a sibling of a victim of the sudden infant death syndrome. These parents are currently using a monitor for a subsequent infant and do not in any way regard the monitor as a failure because their previous infant died.

Finally, I would agree wholeheartedly with the comments of Dr David M Reid (11 July, p 119) to the effect that some of the benefits of monitoring may not be easy to assess in scientific publications. I suspect that those who have not lost a child in this way cannot fully appreciate the difficulties faced by parents in handling another baby. In the absence of any investigation that might assure parents that a second infant will not suffer the same fate as its sibling, constant observation is deemed necessary. In my view, if such parents think that an apnoea monitor will help them in caring for their baby one should be provided—that is, the decision should eventually be made by the parents in full knowledge of the facts. I would, however, emphasise that no parents should be talked into using a monitor or be made to feel guilty if they decline one, and the continuing use of a monitor should be seriously questioned if the monitor itself seems to be causing problems.

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The myelodysplastic syndromes

SIR,—Drs D M Layton and G J Mufti (25 July, p 227) draw attention to the main features of the myelodysplastic syndromes and the need for further research into new therapeutic strategies. The problem, of course, is that we have only a very limited understanding of the disease processes, which makes our therapeutic choices difficult. It is now generally accepted that the syndromes arise from a premalignant clonal abnormality of a haemopoietic stem cell¹ and that the various clinical manifestations reflect a multistage progression to overt leukaemia.

Treatment may be directed towards suppression or elimination of the abnormal cells or, as Drs Layton and Mufti suggest, to differentiation of the functionally immature preleukaemic cells. It is perhaps worth drawing attention to the potential attractions of tretinoin (retinoic acid) in the management of these conditions. While retinoic acid is well established as a differentiating agent for preleukaemic cell lines in culture,^{2,3} there is an additional rationale for its use. It has been known for some time that retinoids prevent chemically induced skin cancer in mice and other tumours, possibly by interfering with tumour promotion. Retinoic acid can cause regression of experimental premalignant lesions^{4,5} and suppress radiation induced leukaemia in mice, though it is not clear whether this is due to immune stimulation or to a direct effect on the leukaemic cells.⁶ A recent randomised therapeutic trial of isotretinoin (13-*cis* retinoic acid) compared with supportive treatment alone showed that in non-sideroblastic patients with less than 6% blasts in the marrow there was a significant increase in survival at one year in the group who received treatment from 36% to 77%.⁷

This is encouraging, though it remains to be confirmed, but we still have little information about the mode of action or whether there is a longer term benefit in suppression of abnormal

haemopoiesis. Interestingly, improved survival in this trial was not accompanied by any striking change in blood counts, but Kerndrup *et al* and Clark *et al* have shown a reversion of both neutrophil myeloperoxidase activity and abnormal cell surface markers towards normal as a consequence of treatment with tretinoin,^{8,9} and leucocyte function may improve.

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Not all travellers need immunoglobulin for hepatitis A

SIR,—The leading article by Drs Jonathan H Cossar and Daniel Reid (13 June, p 1503), which was commented on by Drs G Kudesia and E A C Follett (11 July, p 118), reopens the argument about the cost benefit of screening for immunity to hepatitis A before foreign travel and before the administration of prophylactic normal human immunoglobulin.

The value of pretesting in regular business travellers is beyond question.^{1,2} Not only have they probably had greater past exposure to infection but they will also need repeated injections of a preparation that, although safe, is costly and painful. Screening would identify immune subjects, reducing the numbers requiring prophylactic treatment. On the other hand, the need for screening holiday travellers, who generate most of the requests for immunoglobulin, depends on age, socioeconomic backgrounds,³ and also the travellers' place of residence in the British Isles.

The figures for hepatitis A immunity in Avon (table) are much lower than those quoted by Drs Cossar and Reid for the west of Scotland, and pretravel screening in our area would not be cost effective below the age of 40. A recent report from Japan shows how rapidly susceptibility to hepatitis A may increase in a community as a result

of rising living standards.⁴ It is important to review figures for local antibody prevalence in the United Kingdom regularly to give the most helpful advice. This should take into account not only the country to be visited but also the duration of stay and the lifestyle of the traveller while abroad.⁵

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Reduction in postprandial energy expenditure during pregnancy

SIR,—Unlike Dr A M Prentice and colleagues (25 July, p 266), who used a method for measuring diet induced thermogenesis that is derived by exclusion with an estimate for physical activity, we actually derived our results by direct measurement of the thermic response to a meal (20 June, p 1573). We found a reduction in this response in all of our pregnant women in the second trimester. We measured the thermic response for only two hours after ingestion of the meal and would agree that this probably represents about two thirds of the total response induced by the meal. If this is the case and we extrapolate using the Cambridge group's own reported calculated intake for pregnant women in the second trimester—namely, 8.1 MJ—then the energy savings amount to 94 kJ, about 1% of total energy expenditure, as Dr Prentice and coworkers point out. If this is compared, however, with the rise in resting metabolic rate during the second trimester of 288 kJ/24 h then it represents an energy saving of one third.

An actual fall in resting metabolic rate would be a much more important energetic adaptation as the resting metabolic rate accounts for over 70% of total energy expenditure in humans. We were, therefore, interested to note that Dr Prentice and colleagues observed such a reduction in four out of seven women in the second trimester, as has been reported by Durnin *et al* in Glasgow women.¹ The direction of change in the resting metabolic rate may ultimately depend on the energy intake in each case. We therefore stand by our conclusion that the pregnant woman shows evidence of enhanced energetic efficiency in the second trimester, some more than others, which would reduce the need to consume as much extra energy as official estimates would have us believe.

Our data, nevertheless, indicate that even if dietary energetic savings are taken into account the

pregnant woman still needs to consume extra energy. In our women the calculated minimum extra intake necessary for energy balance was 310 kJ and 420 kJ a day during the second and third trimesters, respectively. These figures are much lower than the 630 and 1470 kJ officially recommended but, interestingly, somewhat nearer to those reported by Durnin *et al*.¹

Dr Prentice and coworkers also emphasise the various estimates of energy intake observed by others, indicating a possible figure of 1 MJ for the rise in energy intake during pregnancy. This contrasts with their own work, which we quoted in the introduction to our paper, which showed no appreciable rise in intake.² As they recently have shown the unreliability of such measurements in giving a true picture of energy intake, we think that little reliability can be placed on those of others in evaluating a precise figure of energy intake by using similar methods.³ This is best achieved by accurate measurement of energy expenditure.

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Delivery after caesarean section

SIR,—In one of the largest published series Dr B G Molloy and colleagues (27 June, p 1645) report a low incidence of both elective and emergency repeat caesarean section. Their conclusion that these figures represent the lowest incidence of abdominal operative delivery in a group of patients after one caesarean section must be interpreted with caution, however, as the populations in the studies cited were not defined. We believe that these results reflect the patient population rather than their obstetric management.

As an example, we presented a review of 260 such patients at the 1987 meeting of the Bavarian Society for Obstetrics and Gynaecology. These women were delivered in the department of obstetrics, Grosshadern, at the University of Munich, which serves as a referral centre for high risk patients. Consequently, we assume that the patients cannot be compared directly with those described by Dr Molloy and coworkers. Of 260 patients, 26.5% were delivered by elective repeat caesarean section compared with only 18% in Dublin. The difference may be explained by analysing the percentage of infants born weighing less than 2500 g as an indicator of high birth risk. In our population 8.5% of infants fell into this category compared with 4.9% in Dublin. Low birth weight plays an important part in the decision to perform an elective repeat caesarean,¹ particularly when the fetus presents as a breech.²

The low proportion of emergency repeat caesarean sections reported (9%) compares with 22.3% in our study. If the population is analysed 62.3% of the patients described by Dr Molloy and colleagues had already delivered at least once vaginally compared with only 13.5% of our patients. It is well recognised that women who have had previous vaginal deliveries have a better chance of delivering vaginally after a trial of labour.^{3,4}

Proportion of patients immune to hepatitis A, September 1985 to July 1987

	Age (years)						
	1-9	10-19	20-29	30-39	40-49	50-59	≥60
<i>Patients screened for immunity alone</i>							
No examined	0	16	85	103	103	95	57
No (%) ELISA IgG positive		0 (0)	7 (8)	16 (16)	36 (35)	43 (45)	42 (74)
<i>All patients tested except those with hepatitis A</i>							
No examined	38	207	444	378	284	265	112
No (%) ELISA IgG positive	5 (13)	23 (11)	93 (21)	110 (29)	139 (49)	146 (55)	92 (82)

ELISA = Enzyme linked immunosorbent assay.