found for 35 drugs. Contrary to hospital advice, 59 drugs were issued in childproof containers.

We had spent time counselling the patients on their treatment before discharge. We had attempted to improve communication by sending a copy of the discharge prescription to the general practitioner by first class post and alerting the practice surgery by telephone to the patient's discharge.

Closer communication between hospital and community care professionals is clearly required. Hospitals should endeavour to inform general practitioners of patients' discharge on the day of discharge or the next working day, and a subsequent home visit by either the doctor or a liaison health visitor would facilitate continuity of treatment. We agree that a more active role for community pharmacists would help iron out potential sources of confusion in nomenclature, timing of doses, and containers and would benefit this vulnerable group of patients.

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Human insulin and awareness of hypoglycaemia

EDITOR,—Mr R Kiln dismisses our study on insulin species and awareness of hypoglycaemia¹ as worthless.² Kiln's comments reflect a less than careful reading of our paper. Contrary to the assertion that "nobody . . . seems to have taken any account of what diabetic patients are saying," this was in fact the main selection criterion for entry into our study.

Kiln finds serious flaws in the design of our study. The first is that one month is not long enough for patients to detect a difference in insulin species. This comment shows a lack of understanding of the design of our study. The randomisation schedule resulted in 28 of 50 patients being treated for two consecutive months with porcine insulin. Not one of these patients was able to identify the species of insulin. Furthermore, other studies in which patients were treated for up to three months also failed to show a difference (A Stocks, personal communication).¹ Kiln suggests that improvement in control may have accounted for our finding of no significant differences between human and porcine insulins. There was no suggestion, however, that glycaemic control improved or changed during our study.

The second concern relates to the method by which patients were asked to identify the insulin species. Kiln's reference to "the questionnaires" again highlights a lack of understanding of our study methods. No questionnaire was used. Instead, patients were invited to identify the sequence of insulin species by whatever means they wished, which invariably required the help of relatives and friends. Participation in a study may alter behaviour. Since patients were aware that one of the main outcomes of our study was their ability to distinguish the insulin species, however, it is likely that any bias would have favoured the patient being able to pick a difference rather than the contrary. The fact that only two of 50 patients could do so is convincing evidence against a species effect on hypoglycaemic awareness.

We appreciate Kiln's disappointment at the accumulating evidence that overwhelmingly fails to support anecdotal impressions about human insulin. Anecdotal evidence is important in

medicine to alert scientists to possible associations. It must then, however, be tested by well designed, controlled studies. Our study tested this anecdotal evidence and did not show an association between human insulin and hypoglycaemic awareness in the very patients who thought that they were experiencing a problem.

Our study is one of five double blind crossover comparisons of human and porcine insulin in patients selected because they complained about hypoglycaemic unawareness developing after their transfer to human insulin (A Stocks, personal communication).^{1,4,5} Not one of these studies has found a significant difference between human and porcine insulin. The continued focus on insulin species diverts attention from the serious problem of hypoglycaemic unawareness in people with insulin treated diabetes.

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Unexplained death in fit young people

EDITOR,—In the editorial on unexplained death in fit young people M J Davies states that there is an analogy with the sudden infant death syndrome.¹ Davies observes that in both instances necropsy does not yield a definitive answer, but the author focuses mainly on necropsy after sudden death. No mention is made of the fact that the National Institutes of Health in Bethesda, Maryland, updated its definition of the sudden infant death syndrome in 1989; this requires an investigation of the scene of death in addition to necropsy. A strong emphasis on such investigation may increase diagnostic reliability when the cause of death is unexplained by necropsy. My experience confirms that more thorough investigation of the death scene in sudden infant death confers a much greater chance of uncovering a cause of death other than the sudden infant death syndrome.²

In sudden unexplained death of a fit young person prompt investigation of the death scene by trained staff from the medical examiner's office, in addition to police investigation, may provide valuable information on the cause and manner of death not detected at the necropsy or by the police investigation. Police are trained and equipped to search for criminal activity, but public health problems, especially elusive environmental hazards, may escape their attention at the death scene.^{1,3}

Forensic pathologists might find it helpful to compile a checklist of basic procedures to use during an investigation of the scene of a sudden unexplained death of an infant or fit young person. Difficult diagnostic cases may require more than one visit to the scene, at different times of the day. Given adequate resources and professional collaboration, forensic pathologists who have restricted their practice to necropsies can easily

gain skill and conduct adequate investigations of the death scene. With an organised, systematic investigation of the death scene greater advances will be made in unravelling the mystery of sudden unexplained death.

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Severity of imported falciparum malaria

EDITOR,—Stephen J Lewis and colleagues retrospectively investigated factors affecting the severity of imported falciparum malaria.¹ They found that prior antimalarial prophylaxis was associated with reduced severity of disease. In addition they concluded that ethnic origin (African versus white) was also associated with a favourable outcome: Africans (whether living in Africa or in Britain) were at significantly lower risk of developing severe malaria. The authors argue that this effect is due to acquired immunity against falciparum malaria. Immunity against subsequent infections with *Plasmodium falciparum* is, however, largely ineffectual, mainly because of the changing antigenic nature of the merozoites but also because most persisting anti-gametocyte IgG fails to offer immunological protection.²

The authors overlooked an important variable by failing to investigate the sickle cell status of their patients. In sub-Saharan Africa the rate of carriage of haemoglobin S is 10-30%.³ Heterozygotes for haemoglobin S are protected against falciparum malaria because the malarial parasites cause sickling of the cells containing the haemoglobin S and the affected cells are removed by the spleen and the reticuloendothelial system. The severity of falciparum malaria in those with sickle cell trait is therefore reduced.¹

Thus the observed effect of ethnic origin on the severity of falciparum malaria is probably due not to acquired immunity but to confounding variables, particularly sickle cell status.

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Controlling deaths from volatile substance abuse

EDITOR,—A Esmail and colleagues suggest that butane gas lighter refills should be withdrawn from the market as abuse leads to many deaths, especially in the under 18s.¹ They acknowledge that withdrawal of these refills might "force young people into abusing other gaseous products." History suggests that this fear is well justified.

McBride, writing in 1910, devoted 17 pages of