7.6 cases in the month after immunisation in the United Kingdom, based on the typical reported annual incidence of 1/100 000).¹

We questioned 103 patients with Guillain-Barré or Miller Fisher syndrome, 98 household controls, and 93 hospital controls in south east England in 1992-4 (before the measles/rubella immunisation programme).² Thirteen patients, and nine household and five hospital controls reported being immunised within 12 weeks before onset of neuropathy in the index case. The immunisations were for influenza in 10 patients and eight household and three hospital controls, typhoid/cholera in one patient and one household control, typhoid alone in one patient, tetanus toxoid booster in one patient, diphtheria/tetanus/pertussis in one hospital control, and human immune globulin in one hospital control. The median intervals between immunisation and neuropathy onset in the index case were 44 (range 3-64) days in patients, 44 (7-64) days in household controls, and 36 (14-62) days in hospital controls. The differences in proportions immunised between the patients and either of the control groups are not significant but the confidence intervals for the odds ratios are wide. A matched analysis shows that the odds ratio of cases having been immunised was 4.0 (95% confidence interval 0.4 to 197) in comparison to household controls and 2.2 (0.7 to 8.1) in comparison to hospital controls. A similar study in 1983-4 also failed to show an increase in risk since 6/100 patients and 5/100 hospital controls had been immunised in the previous 12 weeks.³ The odds ratio for the two series combined was 1.8 (0.7 to 4.4).

Our case-control studies and the literature do not suggest any increased risk of Guillain-Barré syndrome after vaccines currently used in the United Kingdom. However, obtaining the number of individuals required to detect or exclude a small increase in absolute risk would require an active surveillance programme or classification of Guillain-Barré syndrome as a notifiable disease. At present we rely on diligent reporting to the Committee on Safety of Medicines of cases of the syndrome after vaccination.

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Providing intensive care

Cases of trauma can be managed in general intensive therapy units

EDITOR,—D W Ryan points out that increasing resources is one solution to the current crisis in the provision of intensive care services.¹ The editorial fails, however, to address the problem of the inefficient use and staffing of some units.

About 210 cases of major trauma occur in adults each year in Birmingham. About one third

of the patients are admitted to the major injuries unit at University Hospital (formerly sited at the Birmingham Accident Hospital). Most of the rest are managed at the two other main district general hospitals. There are 24 general beds in intensive therapy units on the three sites, five of which are allocated to the major injuries unit. The major injuries unit is separately staffed by four consultant anaesthetists and junior anaesthetic staff on rotation and, in addition, has eight consultant and 18 junior orthopaedic staff sharing the on call rota as well as its own complement of nursing staff. The general intensive therapy unit at University Hospital, with six beds, is separately staffed by its own anaesthetists and nursing staff. The two other district general hospitals manage their major trauma workload within existing resources and have no intensive therapy unit beds designated for cases of trauma.

A recent report on the major injuries unit found no significant difference in outcome between patients admitted from the accident and emergency unit and those admitted direct to the major injuries unit.² There seems to be no rationale for allocating a fifth of the total complement of intensive therapy unit beds at the three sites to a dedicated trauma intensive therapy unit, particularly when no improvement in outcome can be shown.

While intensive therapy unit services in Birmingham are undoubtedly underresourced, the resources that are currently available are clearly not being used to their maximum efficiency. The amalgamation of the major injuries unit with the existing on site intensive therapy unit would eliminate duplication of staff and services and lead to savings, which could be used to increase the overall number of beds.

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Service can't cope with troughs in demand, let alone peaks

EDITOR,—David Ryan correctly points out that "moving critically ill patients between hospitals has become a far from ideal way of life."¹ Politicians have only recently taken note of this, but in fact this situation has existed for as long as I have been a consultant (11 years) working in an intensive care unit in a large teaching hospital.

Recent deaths of critically ill patients who were being moved between hospitals and the media publicity that followed stimulated the health secretary into a typical knee jerk reaction.² The fact that more use will be made of high dependency beds and that a national database of intensive care unit beds will be set up are two such announcements that Stephen Dorrell has made in the House of Commons. Either Mr Dorrell has been misinformed or, as a typical politician, he chooses to ignore the glaring facts that (a) the high dependency unit beds that he is talking about do not exist (only 15% of hospitals in Britain have such beds), (b) high dependency unit beds are not a substitute for intensive care unit beds, and (c) setting up high dependency unit beds will require resources.

The database of intensive care unit beds is flaunted as the "cure all" for the lack of intensive care unit beds in Britain. We have had such a scheme in the north west for the past six years or so (it was previously simply called the bed bureau and is now named the intensive care bed

information service). This was set up to save time for clinicians trying to find a bed. It did not (and was not meant to) solve the problems with intensive care unit beds that existed then, as they do now. I recently heard a chief executive of a health authority in the north west say that there were troughs and peaks in demand and that it was not possible to plan for all contingencies. He was absolutely correct. The trouble is that, throughout my working life as a senior registrar and as a consultant, we have barely been able to cope with the troughs.

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Centralised paediatric intensive care beds are blocked

EDITOR,—While I echo DW Ryan's call for more resources for intensive care, I take issue with Ryan's choice of words when referring to paediatric intensive care.¹ The inability of major paediatric centres to accept all referrals for intensive care may undermine but in no way negates the British Paediatric Association's recommendation that sick children be provided with specialist nursing and medical care.² The recommendation is sound.

Centralisation of paediatric intensive care is the way forward, as Shann³ and others have urged. But there are other reasons, apart from a lack of resources, for the shortage of acute paediatric intensive care beds. More children are surviving previously fatal illnesses but remain dependent on a ventilator. At present there is no facility for caring for these long stay patients who no longer need the full resources available in intensive care units. Altogether 42% of our acute paediatric intensive care beds are currently blocked by such patients, and these unavailable beds were an important factor in our having to refuse 276 patients referred to our paediatric intensive care unit last year. These patients are ready for discharge, but the health authorities from which they came are unable to arrange the provision of services for them locally.

This is likely to be an increasing problem and needs to be addressed by those people charged with planning intensive care and high dependency services across Britain.

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Disodium pamidronate has beneficial effect in Paget's disease of bone

EDITOR,—We are surprised that the authors of the review on diagnosing and managing Paget's disease of bone did not comment on the efficacy of a single infusion of disodium pamidronate in active Paget's disease.¹ Several studies have shown the beneficial effect of this treatment, with a substantial number of patients remaining in remission for up to 18 months to two years.^{2,3} Our study showed the beneficial effect of even a moderate dose (60 mg) given as a single infusion

in moderate to severe active Paget's disease.² We wonder if the authors had any particular reason for not advocating wider use of this treatment.

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Retention of drugs in venous access port chamber

Cause of drugs' effect is not clear

EDITOR,—Although we agree with the assertion that port systems should be flushed before an anaesthetic is finished, as should all intravenous access devices after any drug is given, we hesitate to agree as to the sole cause of the drugs left in the port system in the case reported by Myriam Ben-Arush and Moshe Berant.¹

Precise calculation of dosage given is difficult as the weight of the patient is not given. Using the widely accepted Oakley chart² we might expect a weight of about 13 kg. This would mean the bolus dose received was only: 0.19 mg/kg of ketamine, the anaesthetic dose being 1.0-4.5 mg/kg³; 0.003 mg/kg atropine, the usual paediatric dose being 0.02 mg/kg; and 0.012 mg/kg of midazolam, the sedative dose being 0.03-0.3 mg/kg.⁴ Although ketamine can cause respiratory depression in overdose or too rapid infusion,⁵ it seems unlikely that this small dose of cocktail was solely to blame. However, the possibility of airway obstruction from residual anaesthetic, possibly worsened by the bolus dose, leading to apnoea and bradycardia would fit the scenario well. It is well recognised that both midazolam and ketamine do not totally preserve airway reflexes.^{3,4} Patients who have had sedation or anaesthesia should be observed in a safe environment, such as a recovery room, until they are conscious, breathing, maintaining a clear airway with protective reflexes present, and cardiovascularly stable.⁵ It is not clear from this report that such criteria were met in this case.

Secondly, the assertion that port systems are "unsuitable for intravenous anaesthetic and sedation drugs" that are titrated to effect is not valid. So long as there is knowledge of the volume of the system, allowances for this can be made by slowly flushing the residual volume before the end of the procedure during continued monitoring of the patient in a secure environment. With drugs of set dose, such as neuromuscular blocking agents, the calculated dose should be given and then the system flushed. The use of portal systems will thus prevent the need for further intravenous access in children who are often afraid of needles and difficult to cannulate.

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Drug did not cause collapse of patient

EDITOR,—Myriam Ben-Arush and Moshe Berant attribute cardiorespiratory collapse in a 2 year old child to flushing of residual anaesthetic drugs in the lumen of an implanted central venous cannula.

Our department administers over 300 anaesthetics a year to small children undergoing radiotherapy, using similar drugs and techniques. I am not convinced by the authors' reasoning and feel that it is an incorrect interpretation of the event.

Ben-Arush and Berant do not tell us the weight of the child or the time interval between anaesthesia and the "collapse." The child probably weighs 13 kg and the time for induction of anaesthesia, subsequent treatment, recovery, and transport back to the ward is unlikely to be less than 45 minutes. The described event implies unconsciousness, apnoea, and modest hypotension (pulse remaining palpable) with bradycardia. The normal intravenous anaesthetic induction dose of ketamine is 1-2 mg/kg. This dose will keep a child still for a radiotherapy treatment lasting four to seven minutes.

Ketamine is specifically used for radiotherapy procedures (where the anaesthetist cannot remain with the patient during treatment) as it maintains airway reflexes and respiration. The maximum potential dose reaching this child is 0.16 mg/kg (maximum dead space of device, according to Ben-Arush and Berant, is 2.1 ml; ketamine concentration 1 mg/ml). This could not produce the clinical response described. The effects of 0.1 mg of midazolam in a 13 kg child and 2 µg of atropine/kg can be discounted.

It is more likely that the temporary loss of cardiac output was caused by either an air embolus at the time of "flush" or through the detachment of a piece of thrombus within the central venous catheter. The only drug which could cause this clinical response in this dose would be the short acting muscle relaxant suxamethonium, which was not part of the anaesthetic technique.

It is a pity that the main lesson of the week, which should be that all intravenously administered drugs should be flushed through the catheter, is lost by this incorrect and highly critical speculation. A further "lesson" is that all who inject drugs through central venous lines should be aware of the potential for delivery of a bolus of air or thrombus and have the necessary resuscitation skills. The comments regarding the unsuitability of ported systems for induction of anaesthesia show a complete lack of insight regarding modern anaesthetic techniques. I wonder whether this article was discussed with the anaesthetist responsible for the case or indeed the department of anaesthesia at Rambam Medical Centre?

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Port systems can safely be used for anaesthesia

EDITOR,—As anaesthetists involved in the care of children receiving anaesthesia repeatedly during chemotherapy, we wish to respond to Myriam Ben-Arush and Moshe Berant on indwelling central venous catheter port systems.¹

We agree entirely that failure to adequately flush any residual drugs through a venous access port device, or indeed any intravenous cannula with appreciable dead space, could result in grave consequences for the patient, but we strongly disagree that such devices should be deemed unsuitable for use with intravenous anaesthesia or sedation. By the nature of their illnesses, these children are subjected to multiple procedures under anaesthesia, which can lead to problems with access to veins and to needle phobia. Indeed, some units advocate the routine insertion of a long term venous cannula to avoid these very problems, and not using this type of venous access when it is present is absurd. The correct approach is to lay more emphasis on the way these port systems can be used more safely.

It is the responsibility of the anaesthetist to ensure that he or she is fully aware of the particular type of port device, including its dead space. It is good practice to flush through each drug with saline to avoid unwanted reactions—for example, between sodium thiopentone and atracurium. In our hospital it is routine practice to flush such cannulas with heparinised saline at the end of the procedure while the patient is still being monitored. Doing this, and making a clear record in the patient's notes, minimises the risk of disaster. This practice should be extended to each time a drug is given by this route. Indeed, when dealing with children and neonates it is good risk management to flush all intravenous cannulas after anaesthesia, especially if muscle relaxants have been used.

The authors also hold the opinion that such devices are unsuitable for use with intravenous anaesthesia and sedation on the basis that such drugs are titrated against effect and a relative surplus would always remain in the port dead space. We disagree: provided that the anaesthetist is aware of the dead space and has a sound knowledge of the drug pharmacokinetics and pharmacodynamics, this does not pose a major problem.

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Authors' reply

EDITOR,—We thank the correspondents for their remarks and for their suggestions of additional possible causes for the dramatic event that occurred in our patient. All three letters agree with the main issue presented in our paper—namely, that the safe use of venous access port systems warrants careful flushing of their dead space after giving drugs, including anaesthetics.

The error of omission in the case we reported suggested that a message carrying a reminder of the importance of meticulous adherence to such "good practice" would be of benefit.

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