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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