

FIG. 2. Note medicine cap taped (by patient) over bolus button to discourage overuse of bolus feature during daytime hours.

his confusion and initiated a spiraling pattern of medication excess, confusion, and sedation.

Solution. The patient was followed closely by the pain management team, but these events occurred in the space of just a few days and were not reported. Had these events been described, efforts would have been made to prevent their repetition by altering the prescribed lockout interval and dose. Instead, the patient again undertook independent problem solving with good results. He carefully taped a cap from one of his medication vials over the bolus button, reasoning that the effort and forethought required to remove the cap would serve as a deterrent to unintended overuse of the device's bolus feature (fig. 2). No further incidents of overuse followed this intervention.

DISCUSSION

This case illustrates the potential role for innovative pain relief modalities (neuroaxial administration of opioids, patient-controlled analgesia) and their adaptability to the environment of the home. These measures were indicated in this case because analgesia was incomplete despite efforts at control using more conservative means, and because iv opioids seemed to be responsible for impaired mental status and gastrointestinal dysfunction.

Both of these problems probably could have been resolved by alterations in medication orders, but in this case they were reconciled by novel solutions proposed by the patient. The details of this case were reported to the equipment manufacturer and interestingly, they have announced that the new model of this pump features a textured bolus button that can be readily identified in the dark.

This case also serves as a reminder that patients may have valuable insight into the nature of their problems, and that on occasion they may make important contributions to problem solving.

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Oral Midazolam for a Mentally Retarded Patient

To the Editor:—We were recently asked to anesthetize a muscular 18-yr-old mentally retarded (I.Q. of 60) male who presented to our outpatient facility for proctoscopy for diagnosis of rectal bleeding. Upon presentation to the preoperative area he became frightened to the point of panic. He could not be approached by anyone except his mother. Because of his emotional state, size, and strength, attempts at intramuscular sedation were impossible. His mother attempted to get him to accept intranasal sufentanil but he would not allow this either.

Therefore, based on the studies cited, ^{1,2} we elected to administer oral midazolam (0.6 mg/kg). Although the dose used in the above studies was 0.2 mg/kg, we chose the higher dose because of his extreme state of anxiety, the possibility of paradoxical effect with lower doses, and "first pass" hepatic metabolism of about 50–60% when given by the oral route.⁵

Other than his rectal bleeding and mental retardation, he had no other known medical problems and was A.S.A. physical status 1. His daily medications included pemoline 50 mg bid and thioridazine 25 mg bid.

The medication was prepared by mixing 30 mg of midazolam in 25 ml of a carbonated cola beverage. The pH of the resultant mixture was 3.8; this acid pH allowed the midazolam to remain in solution. The cocktail was then administered by his mother. The patient complied by ingesting the entire amount (31 ml) without incident. Continuous observation by the anesthesia staff was begun following ingestion.

Within 10 min he was sedate enough to allow physical examination and application of a pulse oximeter. Within 20 min he was somnolent but responsive to verbal stimulus. At this point an iv catheter was inserted and blood for preoperative lab work was obtained without objection. Forty minutes after ingestion, general anesthesia was induced with 250 mg of sodium thiopental and maintained with 1% isoflurane by mask for 1 h 20 min. The volume of the cocktail was small and the risk of aspiration was therefore felt to be quite low. Also, because of the relatively noninvasive procedure, tracheal intubation was not felt to be warranted. A recovery period of approximately $3\frac{1}{2}$ h was required before the patient was "fully awake." He was discharged home shortly thereafter under the care of his mother.

We feel that some caution must be used with this regimen because of the possibility of paradoxical reactions in aggressive patients, as well as the risk of oversedation. Keeping these possibilities in mind, however, oral midazolam might be considered for anxiolysis and sedation for children and mentally retarded or otherwise uncooperative individuals.

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REFERENCES

 Raybould D, Bradshaw EG: Premedication for day case surgery. A study of oral midazolam. Anaesthesia 42:591-595, 1987

- Forrest P, Galletly DC, Yee P: Placebo controlled comparison of midazolam, triazolam, and diazepam as oral premedicants for outpatient anaesthesia. Anaesth Intensive Care 15:296-304, 1987
- Reves JG, Fragen RJ, Vinik HR, Greenblatt DJ: Midazolam: Pharmacology and Uses. ANESTHESIOLOGY 62:310–324, 1985

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What is the Etiology of "Reactions" to Vascular Graft Material?

To the Editor:—The report by Roizen et al. is a well-documented examination of a legendary phenomenon in the history of vascular surgery, one often hidden under the rubric "loss of preclot." One can only conclude that this phenomenon is related to the particular choice of a highly porous nonvelour material for the initial graft. In recent years, either velour grafts, which preclot more readily and which are more likely to maintain a laminated coating of fibrin and thrombus, or polytef grafts have become the more common choices for these reconstructions, and this pattern of loss of preclot and apparent disseminated coagulopathy seems to have become rare. The exact biocompatability problem, which is nevertheless very unusual for the nonvelour grafts, has not been elucidated to my knowledge, and it has received no recent attention in the vascular surgery literature.

Case 5 as presented may have a different etiology than that suggested, and crucial information to assess the validity of this case is not presented in the manuscript. The patient had a secondary aneurysm at or just distal to the renal arteries. The authors do not note whether suprarenal or supraceliac cross clamping was necessary. I must presume that this was performed. If so, then a different mechanism for disseminated coagulopathy and severe vasodilitation would have occurred. With the reconstruction of the thoracoabdominal aortic segment, where all visceral artery ostia may be occluded for periods up to 1 h, declamping hypotension and a coagulopathy have been commonly noted. The exact mechanism for this phenomenon is yet unclear, but it presumably occurs with intense vasodilatation of the early ischemic intestine and with

initial washout of vasoactive substances into the systemic circulation, perhaps exacerbated by the systemic acidosis which may occur during these procedures. The treatment is anticipatory: early bicarbonate replacement, use of cell-saving devices, volume replacement with fresh frozen plasma, preparation for the administration of aminocaproic acid, and vigorous attempts to maintain the highest possible body temperature during the procedure.

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REFERENCE

 Roizen MF, Rodgers GM, Valone FH, Lampe GH, Benefiel DJ, Gelman S, Rapp J, Weiler JM, Ota M, Shuman A, Goetzl EJ: Anaphylactoid reactions to vascular graft material presenting with vasodilation and subsequent disseminated intravascular coagulation. ANESTHESIOLOGY 71:331-338, 1989

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In Reply:—I believe the phenomenon we described is not related to the loss of preclot or to the choice of a highly porous nonvelour material for the initial graft. As Dr. Kaufman correctly points out, we found this phenomenon both in woven and in nonwoven grafts; it occurred with three different types of grafts. An anaphylactoid reaction is a particularly difficult reaction to prove because one doesn't want to expose the patient to the graft material in vivo. The rechallenge, which was associated with the reaction, was performed in the patient whom we could challenge, and it was a rechallenge done in vitro with the graft material. Thus this reaction clearly is not related to loss of preclot in the patient whose blood reacted with abnormal generation of kallikrein when rechallenged with the graft. I hope Dr. Kaufman and other readers appreciate this point.

Figure 1 of the manuscript shows that the plasma of patient 3 generated abnormal amounts of kallikrein activity when exposed to the graft in vitro. In addition, in comparison with controls, there were

abnormal amounts of C3a generated *in vivo* in both patients 1 and 2, the two patients whose blood was available for study.

Our group of three physicians was privileged to give anesthesia for more than 200 abdominal aortic reconstructions per yr for this group of vascular surgeons and we never saw this problem other than the times reported. This phenomenon was rare—not the usual phenomenon slower or less technically competent surgeons experience. Yes, the treatment of hypotension upon opening of a newly revascularized limb is anticipatory, but these surgeons work so fast and are so competent that bicarbonate is virtually never necessary and volume replacement suffices. Clearly, the reason we were able to discover this phenomenon is that something atypical of the normal course of expected events occurred in these patients. Volume replacement and maintenance of normal left ventricular end-diastolic volumes as assessed by echocardiography, or maintenance of left ventricular end-diastolic pressures as measured by pulmonary capillary occlusion pressure, had