Anesthesiology
70:881, 1989

Safety of PCA Devices

To the Editor:—In his letter concerning the safety of patient-controlled analgesia (PCA) devices, McKenzie[1] stated that "there are no reports of severe respiratory depression with catastrophic outcome following PCA." We would like to bring to your attention a case where such an outcome *did* result. Grey and Sweeney,[2] of the State of Utah Office of the Medical Examiner, reported a patient who was utilizing PCA therapy for postoperative pain control and who received an accidental massive overdose that resulted in respiratory arrest. The patient was discovered apneic and pulseless approximately 20 min after beginning PCA therapy, with 49.5 ml of the original 60 ml of meperidine solution (10 mg/ml) missing from the PCA device. The patient was resuscitated but remained comatose until death 5 days later. Analysis of blood samples drawn at the time of resuscitation revealed a meperidine level of 4.2 μg/ml, well within the fatal range of 1–8 μg/ml.[3] The PCA device was reportedly set to deliver a dose volume of 1.0–1.5 ml, with a 15-min lockout interval. The PCA device was not turned over to the Office of the Medical Examiner where it could have been tested. Although this incident appears to have been due to a malfunction of a PCA device, this assumption was never substantiated.

This report[2] raises the importance of proper testing of PCA devices to insure that mechanical failures do not occur. Although the pumps are tested by the manufacturer prior to delivery, in our hospital, each pump is further tested by our Biomedical Engineering division. Their lengthy check ensures that volume infusion rates and bolus doses are accurate at each setting. It must also be realized that, although these pumps have self-check mechanisms, they are mechanical devices and may break down. C. R. Bard, Inc., lists a detailed checkout procedure that should be performed at least every 6 months to insure accuracy of their PCA 1 device.* The latter includes a check of flow rates and delivery volumes using either a Mini-Infuser Calibrator (C. R. Bard, Inc.) or a burette and timer.*

McKenzie[1] also mentions that the danger of opioid overdose is present when the *continuous* mode of administration is used. We are aware of anecdotal reports of respiratory depression following activation of the PCA device by a patient's visitors. This unauthorized activation bypasses the inherent safety feature of PCA by which, as patients become more sedated, they will not give themselves a bolus dose. We, therefore, consider that the preoperative teaching of patient-controlled analgesia must emphasize that the *patient* is the *only* person who should activate the pump. If this is adhered to, the PCA device becomes a much safer therapeutic instrument.

JOEL M. KREITZER, M.D.
Fellow, Pain Management Service

LAWRENCE P. KIRSCHENBAUM, M.D.
Assistant Professor of Anesthesiology
Director, Pain Management Service

JAMES B. EISENKRAFT, M.D.
Associate Professor of Anesthesiology
Director, Anesthesia Research

The Mount Sinai School of Medicine
New York, New York 10029-6574

REFERENCES

1. McKenzie R: Patient-controlled analgesia (PCA). ANESTHESIOLOGY 69:1027, 1988
2. Grey TC, Sweeney ES: Patient-controlled analgesia. JAMA 259: 2240, 1988
3. Baselt RC: Disposition of Toxic Drugs and Chemicals in Man, 2nd edition. Davis, Biomedical Publications, 1982, pp 458–461

(Accepted for publication February 1, 1989.)

* Bard® PCA 1 Pump. Operator's Manual. C. R. Bard, Inc., North Reading, MA. July 1988, pp 21–22

Anesthesiology
70:881, 1989

Serum Samples from Patients with Hepatic Dysfunction following Enflurane

To the Editor:—We have recently demonstrated that the metabolism of enflurane produces covalently bound liver protein adducts that are recognized by hapten selective antibodies, as well as antibodies found in the sera of patients with halothane-induced hepatitis.[1,2] To further investigate the possibility that a syndrome of idiosyncratic, immune-mediated "enflurane hepatitis" exists, we are soliciting serum samples (and liver biopsies) from patients who have a provisional diagnosis of enflurane-induced hepatotoxicity. Patients with unexplained postoperative elevations of serum transaminases, or other clinical indications of possible hepatic dysfunction, should be included. Post mortem tissue samples would also be useful.

JACKIE L. MARTIN, M.D.
LANCE R. POHL, PHARM.D., PH.D.
Laboratory of Chemical Pharmacology (JLM, LRP)
NHLBI, Building 10, Room 8N115
NIH, Bethesda, Maryland 20892

Department of Anesthesiology and Critical Care Medicine (JLM)
The Johns Hopkins Medical Institutions
Baltimore, Maryland 21205

REFERENCES

1. Christ DD, Satoh H, Kenna JG, Pohl LR: Potential metabolic basis for enflurane hepatitis and the apparent cross-sensitization between enflurane and halothane. Drug Metab Dispos 16:135–140, 1988
2. Christ DD, Kenna JG, Kammerer W, Satoh H, Pohl LR: Enflurane metabolism produces covalently bound liver adducts recognized by antibodies from patients with halothane hepatitis. ANESTHESIOLOGY 69:833–838, 1988

(Accepted for publication February 1, 1989.)