uneventful, with a normal oral dietary intake and urinary output (table 1). Following the last two operations the patient was treated with penicillin and chloromycetin. Gantrisin was prescribed for her after discharge from the hospital.

Discussion

This report suggests that (unlike hepatic necrosis) renal damage following methoxyflurane does not contraindicate the subsequent use of halothane in the same patient. To our knowledge, this is the only reported case in which the patient received halothane before and after methoxyflurane-induced nephrotoxicity. If cross-sensitization existed with respect to nephrotoxicity it would be reasonable to expect an exacerbation or recurrence of the renal syndrome when halothane was administered to a patient previously sensitized with methoxyflurane. Most of the symptoms following methoxyflurane-induced renal damage suggest a primary tubular defect with less impairment of glomerular function, which is characteristic of true nephrotoxins rather than sensitizing drugs.[1] Yet, methoxyflurane lacks important characteristics of a true nephrotoxin[1, 6] in that it fails to produce injury in every patient, there is no proven relationship between dose and severity of renal damage, and the syndrome has not been produced in experimental animals. Therefore, the mechanism of methoxyflurane-induced renal damage remains unclear. Obesity with possible abnormal metabolic pathways and resulting abnormal-methoxyflurane metabolism remains an unknown variable in the pathogenesis of the methoxyflurane-induced nephrotoxicity in our patient.

References

1. Elkington SG, Goffinet JA, Conn HO: Renal and hepatic injury associated with methoxyflurane anesthesia. Ann Intern Med 69:1229, 1968
2. Klein NC, Jeffries GH: Hepatotoxicity after methoxyflurane administration. JAMA 197: 1037, 1966
3. Lindenbaum J, Leifer E: Hepatic necrosis associated with halothane anesthesia. New Eng J Med 268:525, 1963
4. Klatskin G: Recurrent hepatitis attributable to halothane sensitization in an anesthetist. New Eng J Med 280:515, 1969
5. Crandell WB, Pappas SG, Macdonald A: Nephrotoxicity associated with methoxyflurane anesthesia. Anesthesiology 27:591, 1966
6. Klatskin G: Toxicity of Anesthetics. Edited by RB Fink. Baltimore, Williams and Wilkins, 1968, pp 159–172

Drugs

METABOLISM OF PENTAZOCINE A spectrophotofluorometric method was used for determining urinary levels of pentazocine. Pentazocine was found to be extensively metabolized; less than 13 per cent of the dose appeared in the urine unchanged. Between 12 and 30 per cent was excreted as a glucuronide conjugate, and at least one other unidentified polar metabolite was detected. The major portion of pentazocine and metabolites was excreted in the first 12 hours. (*Berkowitz, B., and Way, E. L.: Metabolism and Excretion of Pentazocine in Man, Clin. Pharmacol. Ther. 10: 681 (Sept.) 1969.*)

ATROPINE AND PRALIDOXIME The interaction of atropine and pralidoxime, when given intramuscularly to volunteers, was studied in a crossover random design. Subjects received atropine, 2 mg, and pralidoxime, 600 mg, alone or in combination or simultaneously at two injection sites. The increase in the heart rate after atropine was significantly delayed if atropine and pralidoxime were injected as a mixture. This delay did not occur if the two drugs were injected simultaneously but at different injection sites. (*Sidell, F. R., Magness, J. S., and Bollen, T. E.: Modification of the Effects of Atropine on Human Heart Rate by Pralidoxime, Clin. Pharmacol. Ther. 11: 68 (Jan.) 1970.*)