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### Surgery

**HALOTHANE AND LIVER DAMAGE** Numerous factors may be responsible for postoperative liver damage with or without halothane as the anesthetic agent: (1) surgical trauma to the biliary or vascular system of the liver, (2) prolonged anesthesia, (3) prolonged hypotension or shock with secondary ischemia of the liver, (4) hepatotoxic medication (antibiotics, tranquilizers), (5) infections, particularly peritonitis, (6) incubation stage of coincidental virus hepatitis, and (7) hypersensitivity reaction to halothane. Autopsy reports of 219 surgical patients over a 2-year period were studied, of these, 160 had received halothane and 59 some other form of anesthesia. In 38 patients, liver damage of varying degree was encountered. Of these 38 patients, 27 had been anesthetized with halothane, 6 of them more than once. Duration of anesthesia or the number of repeat exposures to halothane seemed to be unrelated to the degree of hepatic damage. On the other hand, associated infection (peritonitis, intra-abdominal abscesses, pancreatitis and/or cholecystitis) and prolonged circulatory collapse contributed significantly to the incidence and degree of liver pathology. In the group with severe liver damage (12 cases), there were 4 patients with acute hepatic necrosis of "unexplained" etiology. All 4 had received halothane. Two of the patients had previous exposures to halothane, 7 and 15 days earlier. Death occurred on day 4, 4, 5, and 12, respectively. Two patients who had received no halothane and died in acute liver failure showed autopsy findings which were undistinguishable from "halothane induced" hepatic necrosis. Although proof to incriminate halothane does not exist, but a causative relationship between halothane and liver damage cannot be excluded with certainty. (Jensen, H. H., and others: *Acute Postoperative Liver Damage with Special Reference to Halothane*, *Langenbeck Arch. Klin. Chir.* 317: 96 (March) 1967.) **ABSTRACTOR'S NOTE:** The most valuable aspect of this paper is the large bibliography reviewing the typical, but by no means specific findings of liver damage after exposure to halothane. The authors stress the entity of "halothane sensitization" after multiple exposures, but do not ascribe enough significance to factors responsible for ischemia of the liver, particularly iatrogenic, such as infusions of norepinephrine. Two of the "unexplained" cases could be explained on this basis. One, after an intraoperative cardiac arrest secondary to blood loss of 6,000 ml., received norepinephrine for over 36 hours and the other patient was markedly anemic before surgery. The other two "unexplained" cases had more than one exposure to halothane within two weeks.