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### Jaundice Following Fluorene Anesthesia

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There have been no published reports of hepatic damage secondary to fluorene anesthesia in healthy human beings since its introduction into clinical anesthesia in 1953, and we are aware of only one report of such damage in a chronically ill and elderly patient, who had abnormal liver function tests and blood transfusions prior to operation.<sup>1</sup>

#### REPORT OF A CASE

The patient, a 39-year-old Caucasian man in perfect health, was scheduled for elective thoracotomy for biopsy of an asymptomatic mediastinal mass discovered on routine chest x-ray. He had no history of prior surgical operations, anesthesia, blood transfusion, allergies, recent tattoos, foreign travel, exposure to hepatitis, or use of parenteral materials possibly contaminated with hepatitis virus. On admission, all blood studies (including CBC, electrolytes, bilirubin, alkaline phosphatase, CPK, and LDH) and urinalysis were normal.

Anesthesia was induced with thiopental prior to intubation facilitated by succinylcholine and maintained with N<sub>2</sub>O-O<sub>2</sub> (3 and 2 l/min) and concentrations of fluorene (approximately 2 per cent) necessary to keep the systolic blood pressure between 110 and 140 torr. During the 1½-hour procedure, a benign neurofibroma was easily excised. Blood transfusion was not necessary. The immediate postoperative course was uneventful except for a slight temperature elevation (100.2-100.4 F) until the fourth postoperative day, when

serosanguineous wound drainage was noted. Prosthaphlin therapy was instituted at this time. On the fifth day a pulmonary infiltrate had developed in the right lower lobe, and the patient had a spiking fever, with temperatures of 101-103 F, and was visibly jaundiced. The next day he was afebrile, but laboratory studies revealed the following abnormal values: bilirubin 4 mg/100 ml, alkaline phosphatase 260 mU/ml, CPK 425 mU/ml, LDH 250 mU/ml, SGOT 445 mU/ml. The surgical wound was clearly infected, but responded to routine care and continuation of prosthaphlin therapy. Prosthaphlin was discontinued on the tenth postoperative day, but was reinstated for a four-day period two weeks later without further jaundice. By the twelfth day, bilirubin, CPK, LDH, and SGOT had returned to normal. The remainder of the postoperative course was unremarkable. The patient refused to submit to a fluorene challenge.

#### DISCUSSION

One advantage proposed for fluorene is its lack of hepatotoxicity, and there is no evidence that it alters liver function tests, even in the presence of severe hypercarbia and prolonged or severe hypotension.

It is presently accepted that most inhalation agents are metabolized in the body.<sup>2-5</sup> Currently the question has been raised as to whether the agents themselves or their metabolites cause tissue reactions and damage. Trifluoroacetic acid and trifluoroethanol are compounds demonstrated in the metabolism of both fluorene<sup>3,6</sup> and halothane.<sup>3,7,8</sup> Although the toxicity of these metabolites in man remains to be determined, there is evidence of hepatotoxicity in experimental ani-

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The opinions expressed in this article are those of the authors and do not necessarily reflect those of the Department of the Navy.

mals following administration of both of these compounds.<sup>7, 9, 10</sup> Since these agents share similar biodegradation products, it would not be surprising for fluroxene to cause hepatic damage similar to that described for halothane. While we cannot conclude that there was a definite cause-effect relationship between fluroxene and hepatic dysfunction in the case presented here, we feel that the likelihood of such a relationship is strong. As clinical use of fluroxene increases, more case reports such as this may be anticipated.

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### Neonatology

**PREDICTABILITY OF RDS** This study presents data relating the respiratory distress syndrome to gestational age and to mode of delivery. Incidence of and mortality from RDS were determined in 10,335 infants delivered vaginally and 1,457 delivered by cesarean section. In the vaginally-delivered group, as gestational age decreased from 39–40 weeks to 29–30 weeks, the incidence of RDS increased from 0.05 to 64 per cent and mortality from RDS rose from zero to 37 per cent. In babies delivered by cesarean section, the incidence of RDS increased from 0.8 to 54 per cent and mortality from zero to 43 per cent as gestational age decreased from 39–40 weeks to 31–32 weeks. The authors also present data indicating that RDS is unrelated to the presence of maternal diabetes or to artificially-induced labor. The syndrome seldom was seen after 38 weeks of gestation in any group analyzed, but the degree of prematurity necessary for onset of the disease is slight, since 37–38-week-old newborns may be affected. The reason for the greater incidence of RDS in infants delivered by cesarean section is unexplained. (*Usher, R. H., Allen, A. C., and McLean, F. H.: Risk of Respiratory Distress Syndrome Related to Gestational Age, Route of Delivery, and Maternal Diabetes, Am. J. Obstet. Gynecol.* 3: 826–832, 1971.) **ABSTRACTER'S COMMENT:** The value of these data is limited only by the fact that the population under consideration was 98 per cent Caucasian. The authors, however, present data from Boston's Joslin Clinic corroborating their findings regarding lack of a relationship between maternal diabetes and RDS.