was found over a wide range of clinical concentrations. At a total concentration of 10 μg/ml, 89.3 per cent binding (10.7 per cent unbound) was found by ultrafiltration and 88.5 per cent binding (11.5 per cent unbound) by equilibrium dialysis. Discrepancies between the results of this study, that of Koch-Weser and Sellers,[6] and the ^{14}C results of Dayton *et al.*[5] and the spectrophotometric and spectrofluorometric work of others are not readily explained, but may be due to incomplete extraction of thiopental or interference from proteins and metabolites of thiopental with the spectrophotometric and spectrofluorometric assays.

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Hepatic Function and Anesthesia

HALOTHANE AND HEPATIC FUNCTION Thirty-nine patients with uterine cervical carcinoma underwent repeated radium insertion. They were randomly divided into two groups. One received halothane for all procedures (18 patients); the other received neither halothane nor methoxyflurane (21 patients). SGPT levels were measured the day before each anesthetic; patients received four anesthetics within four weeks. If SGPT was greater than 100 IU/l, further tests of hepatic function were performed. The two groups were comparable prior to the institution of treatment and had no prior clinical evidence of hepatic disease. In no control patient was any SGPT level greater than 100 IU/l recorded. Four of the patients receiving halothane developed SGPT elevations to more than 100 IU/l; three of them had been exposed to halothane on three occasions. No abnormality in any other test of hepatic function was observed. (*Trowell J, Peto R, Smith AG: Controlled Trial of Repeated Halothane Anaesthetics in Patients with Carcinoma of the Uterine Cervix Treated with Radium. Lancet 1:821–824, 1975.*) ABSTRACTER'S COMMENT: Alterations of post-anesthetic hepatic function produced by halothane have been observed even when operation is not performed (Stevens WC, Eger EI, Joas TA, et al, Canad Anaesth Soc J: 20: 357–369, 1973). These authors felt that these resulted from the action of halothane metabolites rather than the drug itself. As yet, there is no convincing evidence that these minor changes of hepatic function bear any relationship to "fulminant halothane hepatitis."