THIOPENTAL The pharmacologically active, un-ionized molecules of thiopental pass readily through lipoid membranes such as the blood/brain and blood/tissue barriers. After the intravenous injection of thiopental-C^{14} high radiodensity (indicating high concentration of the drug) has been observed within one minute, in the visual, auditory and somatosensory areas of the cerebral cortex, the geniculate bodies and the inferior colliculus. Within the first 30 minutes of injection, thiopental is present in the lean body mass and to a lesser extent in fat depots. From 30 minutes to 12 hours, distribution is to fat alone, after which time, further uptake by fat is of minor significance. The rate of disappearance of thiopental from plasma, after tissue distribution is completed, is 9 to 15 per cent per hour. Biotransformation by liver microsomes accounts for disposition of thiopental, except for 0.3 per cent, which is excreted unchanged by the kidneys. (*Mark, L. C., and Brand, L.: Where Does the Pentothal Go?, Bull. N. Y. Acad. Med. 40: 476 (June) 1964.*)

HALOGENATED HYDROCARBONS C-14 and Cl-36 methoxyflurane and Cl-36 labeled halothane have been studied in rats and were found to undergo biotransformations with the exhalation of $C^{14}O_2$ and the excretion of urinary chloride-36. Rat liver slices and tris buffer (*p*H 7.4) were incubated for 2 hours in sealed Warburg vessels. Total activity of the $C^{14}O_2$ or chloride-36 produced during incubation was determined. Viable liver slices are required for the biotransformation of methoxyflurane indicating that the reaction is presumably enzymatic. In the case of halothane, chlorine is removed to some extent by heated protein and to a greater extent by viable liver slices. Thus, this reaction is partially enzymatic. (*Van Dyke, R. A., and Chenoweth, M. B.: Biotransformation of Methoxyflurane and Halothane in Liver Slices, Fed. Proc. 23: 179 (March–April) 1964.*)

CHLOROFORM Chloroform, even if used only once in subnarcotic concentrations, caused delayed death in white mice, while halothane given daily for 4 weeks did not cause any permanent liver damage. (*Siess, M., and others: Halothane-Chloroform, Der Anaesthesist 13: 106 (April) 1964.*)

HALOTHANE Hepatotoxicity ascribed to halothane consists of centrizonal hepatic necrosis, minimal fatty change and a severe inflammatory (mononuclear) reaction predominantly in the portal zones, a picture indistinguishable from acute viral hepatitis. Hepatotoxicity is more frequent after multiple exposures to halothane. Pyrexia develops post-operatively and usually is maximal at about five days. The liver is tender. Jaundice develops usually one to two weeks after the anesthetic but sometimes within only a few days. Serum transaminase values are greatly elevated. After variable periods of excitement and vomiting the patient passes either into coma and dies or recovers. Postnecrotic cirrhosis may be a sequel. Association between hepatotoxicity and halothane is so rare that abandonment of the anesthetic at the present time is not merited. With regard to prevention, more than one exposure to the agent should be avoided, particularly if the first one was followed by unexplained postoperative fever. There is no evidence that patients with underlying liver or biliary disease are more susceptible than others to ill effects from halothane. (*Sherlock, S., and others: Hepatotoxic Effects of Anaesthetic Drugs, Proc. Roy. Soc. Med. 57: 305 (Apr.) 1964.*)

CURARE ANTAGONISTS The anti-tubocurarine effects of physostigmine, neostigmine and edrophonium were studied in the intact rat over large dose ranges of the antagonists. With increasing doses of the three drugs the average paralyzing dose of tubocurarine increased, reached a plateau, and then decreased. The antitubocurarine effect is directly related to the anti-cholinesterase activity of these agents. The loss of anticurare effect after large doses of edrophonium is related to the neuromuscular blocking effects of this drug. The loss of anticurare effects after large doses of neostigmine and physostigmine may be attributed to a decrease of acetylcholine released by the nerve endings per stimulus. (*Maanen, E. F.: Anti-Tubocurarine Effects of Physostigmine, Neostigmine and Edrophonium in the*