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Drugs and Their Actions

CARDIAC OUTPUT AND LIDOCAINE METABOLISM A high incidence of toxic reactions to intravenously-administered lidocaine is apparent in patients with markedly depressed circulatory function. To evaluate this situation, the relationship between cardiac output, hepatic blood flow, and lidocaine metabolism was studied in 17 patients at time of cardiac catheterization during the constant infusion of lidocaine at a rate of 40 μg/kg/min. Hepatic blood flow was estimated by a dye-dilution technique. The mean arterial lidocaine concentration (MALC) was significantly higher in patients whose cardiac indexes were below 2.5 l/min/m². The relationship can be expressed by the equation: MALC (μg/ml) = 0.13 C.I. + 0.207. A similar relationship was found between the steady-state arterial lidocaine concentration and estimated hepatic blood flow (EHBF), with a significant rise in lidocaine levels when EHBF fell below 750 ml/min/m². Since a direct linear correlation was found between cardiac index and EHBF, the authors suggest that the rate of metabolic breakdown of lidocaine depends on cardiac output, and intravenous dosage must take this factor into account in order to prevent the possible production of high blood levels and clinical toxicity. (*Stenson, R. E., et al.: Interrelationship of Hepatic Blood Flow, Cardiac Output and Blood Levels of Lidocaine in Man, Circulation 43: 205–211, 1971.*) EDITOR'S COMMENT: It is surprising how little information about the relationship between cardiac output and drug metabolism is available in the anesthesia literature. Considering the rigidity of the drug dosages we employ (*i.e.*, units of drug/unit body weight), these findings open a new vista of potential clinical investigation.