The cerebrospinal fluid chloride and blood inorganic phosphate levels showed a slight decrease but remained within limits observed during normothermia. The potassium level decreased significantly both in blood and cerebrospinal fluid under the influence of hyperventilation. (Blauenstein, U. W.: The Influence of Moderate Hypothermia on Acid Base and Electrolyte Balance in the Spinal Fluid and Arterial Blood, Der Anaesthesist 14: 361 (Dec.) 1965.)

HEPATIC DAMAGE In a prospective study designed to detect evidence of subclinieal cases of hepatic damage following halothane anesthesia, serum isocitric dehydrogenase (ICD) activity was measured in 233 patients. Serum ICD activity has generally been found to be elevated in association with hepatocel-Although some investigators lular necrosis. regard it as a slightly less sensitive reflector of hepatocellular damage than serum glutamic oxalacetic transaminase (SGOT) and glutamic pyruvic transaminase (SGPT), ICD activity was selected for this study because of the decreased likelihood of its reflecting other possible postoperative complications such a myocardial infarction. Sporadic elevations of serum ICD activity were found preoperatively and postoperatively and there seemed to be some trend for serum values to be higher postoperatively. However, this trend was not significantly greater in the group receiving halothane than in the group receiving other anesthetic agents. This study employing ICD activity thus provided no evidence that subclinical, anieterie liver damage occurs more frequently following halothane anesthesia than following other inhalation anesthetic agents. (DeBacker, L. J., and Longnecker, D. S.: Prospective and Retrospective Search for Liver Necrosis Following Halothane Anesthesia. J.A.M.A. 195: 157 (Jan.) 1966.)

ACETYLCHOLINE Spontaneous release of acetylcholine from the rat-diaphragm motor-nerve terminals was greatly reduced below the resting rate by prolonged exposure to calcium-free solutions. The presence of a chelating agent made the decline in rate greater; after six hours, miniature end-plate potentials were often abolished. Local application of calcium

induced return of activity as did restoration of calcium to the bath. In muscle exposed to the chelating agent, caffeine or ouabain caused a rapid further decline in miniature end-plate exposential frequency. Without the chelating agent, these agents induced a rate increase even though calcium ion was absent. Barium chloride and strontium chloride were able to restore and maintain activity completely abolished by prolonged calcium deprivation with the chelating agent. It is concluded that calcium is a necessary cofactor for the spontaneous release of acetylcholine at the motor-nerve terminals. Intracellular strongly bound stores are capable of supplying sufficient calcium to maintain the activity. (Elmquist, D., and Feldman, D. S.: Calcium Dependence of Spontaneous Acetylcholine Release at Mammalian Motor Nerve Terminals, J. Physiol. 181: 487 (Dec.) 1965.)

no methemoglobin-forming action on human P hemoglobin solution and erythrocyte suspen-However, o-toluidine, a possible intermediate metabolite of L-67, exhibited the action in a concentration of 40 mg./100 ml. In cats, an intramuscular injection of L-67 and N o-toluidine produced moderate methemoglobinemia. The average ratio ("mole ratio") of total moles of methemoglobin produced to total moles of the drug administered was cal-culated as about 1.0 for both the drugs. On 25 the other hand, lidocaine and its possible intermediate metabolite, 2:6-xylidine, did not affect the concentration of methemoglobin in the animal. In clinical experiments on 50 patients who received L-67 (200-1,600 mg.) epidurally, an increase of methemoglobin accounting for over 10 per cent of the total hemoglobin was observed occasionally when a the dose of the drug exceeded 10 mg./kg. of S body weight. The concentration of methemoglobin increased roughly in parallel with the $\overline{\mathbb{S}}$ dose of L-67 administered, although marked o individual differences were noted in the susceptibility to methemoglobin. The highest > methemoglobin concentration among all the cases was 2.79 g./100 ml. which was found in \(\sigma \) a patient given 1,200 mg. of the drug. The E average "mole ratio" calculated in 39 cases who received L-67 in a single dose was 0.9.