and nausea that sometimes follows the initial dose of morphine." 5 references.

J. C. M. C.

BEYER, K. H., AND LATVEN, A. R.: *An Evaluation of the Influence of Succinate and Malonate on Barbiturate Hypnosis.* J. Pharmacol. & Exper. Therap. 81: 203–208 (June) 1944.

"The ability of the brain to oxidize glucose, lactate and pyruvate in vitro has been shown to be inhibited markedly by certain barbituric acid derivatives. . . . It seems well established . . . that barbiturates do inhibit respiration of the brain cortex to an extent more or less consistent with their potency as hypnotics. . . . It remained for Soskin and Taubenhaus to reason, since barbiturates did not inhibit the oxidization of succinate, that 'by supplying sufficient of the latter substrate (succinate) one might adequately maintain the metabolism of the brain of a poisoned animal until the barbiturate had been destroyed or excreted.' The results of their experimentation substantiated their hypothesis. This correlation of information concerning the effects of compounds on intermediary cellular metabolism with the observed pharmacodynamic action of barbiturates apparently served at once to demonstrate the practicality of this approach to such a problem and to stimulate interest in sodium succinate as an antidote for barbiturate poisoning. The purpose of the work presented in this paper was to evaluate in another laboratory the effectiveness of succinate therapy in shortening the duration of barbiturate hypnosis. . . . Sodium malonate and glutamate, as administered in these experiments, were without effect on the duration of pentobarbital hypnosis in mice or rats. We were able to confirm the observation that pentobarbital did not inhibit the in vitro oxidation of succinate by the succinoxidase system. Though it was found that the intramuscular administration of sodium succinate to mice and rats moderately diminished the duration of pentobarbital hypnosis this succinate-barbiturate antagonism was not nearly as great on a dosage basis as has been reported previously." 6 references.

J. C. M. C.

ORTH, O. S.; STUTZMAN, J. W., AND MEEK, W. J.: *Relationship of Chemical Structure of Sympathomimetic Amines to Ventricular Tachycardia during Cyclopropane Anesthesia.* J. Pharmacol. & Exper. Therap. 81: 197–202 (June) 1944.

"In previous studies with comparable pressor dosages of eleven sympathomimetic amines, the five primary and secondary amines with a catechol nucleus caused ventricular tachycardia during cyclopropane anesthesia, and the one tertiary amine with a catechol nucleus did not. Further studies have been made as 15 additional amines have become available. Tests of the present drugs have confirmed the conclusion that a catechol nucleus and also a primary or secondary amine group are necessary for the production of ventricular tachycardia by drugs in the dog during cyclopropane anesthesia. . . . Dogs served as the test animal. . . . In a dosage producing a blood pressure rise equal to that caused by 0.01 mg. of adrenalin per kilogram, the six primary and secondary amines with a catechol nucleus consistently elicited ventricular tachycardia. Tertiary amines with a catechol nucleus and the other amines used did not cause this irregularity." 7 references.

J. C. M. C.

SIEVER, JAMES M.: *Continuous Caudal Anesthesia: Analysis of 1,200 Cases with Comparison of Methods.* J.A.M.A. 125: 327–332 (June 3) 1944.