general anesthesia and muscle relaxant. All patients had 0.5 mg. of ergometrine maleate intravenously. A marked rise in both systolic and diastolic pressures occurred within three minutes in patients suffering from hypertensive toxemia while no response was seen in normotensive patients and little change in those with chronic hypertension. Anesthetics causing blood pressure falls minimize the effects of ergometrine, while those causing blood pressure elevation and sympathomimetic amine release exaggerate its effects. (*Baille, T. W.: Vasopressor Activity of Ergometrine Maleate in Anesthetized Parturient Women, Brit. Med. J. 1: 585 (Mar. 2) 1963.*)

CURARE ANTAGONIST Panthenol is the alcohol of pantothenic acid, which is an essential part of the co-enzyme of acetylization (co-enzyme A). Numerous substances are acetylated, amongst them choline to acetylcholine. This might explain curare antagonism of pantothenic acid. Panthenol was used as a curare antagonist in 120 patients. It was highly effective; and since it did not cause bronchospasm or bronchial secretions, no atropine had to be given. Recurarization was not observed. No untoward circulatory effects occurred. It is believed superior to all other presently available curare antagonists. (*Weber, T. M.: Position of Panthenol Amongst the Curare Antagonists, Der Anaesthesist 12: 1 (Jan.) 1963.*)

RESPIRATORY DEPRESSION Arterial oxygen and carbon dioxide tensions were measured to compare respiratory depression following administration of racemorphan with that following a mixture of meperidine and levallorphan. In doses producing equal analgesic effects, meperidine, despite the addition of levallorphan, caused a significantly higher elevation of carbon dioxide tension than racemorphan without addition of an antagonist. (*Schmidt, K.: Blood Gas Analysis Concerning the Respiratory Depression of Two Commonly Used Analgesics in View of Intracranial Hypertension, Der Anaesthesist 12: 6 (Jan.) 1963.*)

DIGITALIS The only differences in action of cardiac glycosides are in onset of action, maximal effective dose, rate of absorption and daily loss of activity. Cumulation may better be expressed as "remaining active dose per day." (*Gillman, H., and Grosse-Brock-Hoff, F.: Therapy With Cardiac Glycosides, Deutsch. Med. Wschr. 1: 1 (Jan.) 1963.*)

SODIUM SALICYLATE Sodium salicylate decreases the time necessary to effect respiratory arrest from ether and chloroform anesthesia in rats. In addition it decreases the time to respiratory arrest due to hypoxia from hypoxic mixtures of nitrous oxide and oxygen. A dosage response relationship seemed to exist for ether anesthesia but not pentobarbital. The effect of salicylate on inhalation anesthetics was interpreted as being primarily caused by the increased respiration seen in these animals after sodium salicylate. (*Bowman, D. C., Damgaard, E. D., and Doemling, D. B.: Sodium Salicylate Effect on General Anesthesia in Rats, J. Pharmacol. Exp. Ther. 138: 236 (Nov.) 1962.*)

PHENOTHIAZINE POISONING Stimulating drugs are contraindicated in the treatment of phenothiazine poisoning. While chlorpromazine may protect against convulsant poisons, it can occasionally potentiate convulsant effects of drugs. A 60 mg. dose of megimide caused severe convulsions in a patient with mild chlorpromazine poisoning. (*Lendle, L.: Phenothiazine Poisoning, Muenchener Med. Wschr. 105: 152 (Jan.) 1963.*)

ASSESSMENT OF DRUG SAFETY Before drugs are released to the public, their testing should include: (1) information on the rate and degree of absorption, the excretion of the drug and the duration of its action; (2) results of tests for screening for toxicity included and uterine-fetal tests on at least two or more species; (3) means of assessing the degree and rate of recovery after symptoms have appeared and something on antidotal tests; (4) the nature of severe reactions that might be anticipated. (*Noel, P. R.: Toxicity Tests in the Assessment of Drug Safety, Lancet 2: 824 (Oct. 20) 1962.*)