common signs and symptoms were: spasm of the neck muscles and opisthotonus; trismus, sardonic grin and facial spasm; protrusion or uncontrolled movement of the tongue and jaw, dysarthria, spasm of the extremities; and dysphagia. Most of the patients received phenothiazine for nausea and vomiting and only five were under management for psychiatric disorders. Prochlorperazine (Compazine) and perphenazine (Trilafon) had been administered to 87 per cent of the patients. The longest duration of phenothiazine administration before onset of reactions was six days and the shortest was four to eight hours. (*Cain, H. D., and Malcolm, M.: Phenothiazine Ataraxics, California Med. 93: 24 (July) 1960.*)

**DIGITALIS** The administration of acetylstrophanthidin in dogs with intact portal circulation resulted in a decline in venous return and a consequent intravascular pooling of blood. When pooling in the splanchnic bed is prevented, an increase in venous return and a decline in intravascular volume occurs. Small elevations of inferior vena caval pressure are observed. Similar increases occur in superior vena caval pressure. These findings are consistent with the hypothesis that in the dog, digitalis induces a generalized venoconstriction, most striking in the hepatic area. (*Ross, J., Jr., and others: Studies on Digitalis; Extracardiac Effects on Venous Return and on Capacity of Peripheral Vascular Bed, J. Clin. Invest. 39: 937 (June) 1960.*)

**DIGITALIS** Effects of acetylstrophanthidin, ouabain, and lanatoside C on peripheral vascular resistance was studied in dogs with the use of cardiopulmonary bypass to exclude cardiac action. With constant perfusion rates significant elevation of arterial pressure occurs in each instance and reflects an augmentation of peripheral vascular resistance. The elevation is not abolished by ganglionic blockade or adrenalectomy and not modified by splanchnic arterial ligation. Injection of acetylstrophanthidin into the constantly perfused lower aorta results in a pressor response limited to this portion of the vascular bed. Injection into the upper segment of the aorta produces a reflex fall in pressure in the lower segment. A direct action of digitalis on arteriolar smooth muscle is probably responsible for the digitalis induced increase in peripheral vascular resistance. (*Ross, J., Jr., and others: Studies on Digitalis; Direct Effects on Peripheral Vascular Resistance, J. Clin. Invest. 39: 930 (June) 1960.*)

**DIGITALIS ANTAGONISM** Objective evidence of digitalis intoxication is revealed by characteristic electrocardiographic changes, but gives no information of its degree. At low levels of intoxication cardiac arrhythmias may revert to normal without the need for antidotal measures. Various degrees of digitalis toxicity are antagonized by potassium salts, but dosage determination is difficult because of fluctuations in the distribution of total body potassium stores in both extra- and intra-cellular compartments. "Acute" and "subacute" categories were established in dogs poisoned with K-strophanthidin, on the basis of the distribution of intravenously administered potassium chloride. L-glutamate and A-ketogluterate potassium salts reversed electrocardiographic evidence of intoxication where the potassium salts of chloride, acetate, aspartate, succinate, lactate, and citrate were ineffective. Clinical trial is suggested. Monopotassium glutarate has been found to be clinically effective in reversing arrhythmias due to digitalis intoxication, and possesses a margin of safety which makes it useful in the presence of altered renal function. (*Keyl, A. C., and others: Digitalis Antagonism, A.M.A. Arch. Int. Med. 105: 709 (May) 1960.*)

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