tachycardia due to catecholamine administration. In previous studies in dogs of the metabolic effects of catecholamines, it has been shown that the intravenous administration of norepinephrine at 1 μg./kg./minute (or equivalent vasopressor doses of epinephrine and phenylephrine) produces a metabolic ketoacidosis. This is characterized by a blood *p*H of less than 7.1, a standard bicarbonate of less than 8 mEq./liter, and accumulation of acetoacetic and betahydroxybutyric acids which equal m*M* for m*M* the bicarbonate deficiency, hyperglycemia, and suppression of insulin release. Associated with these metabolic changes are a decrease in blood volume of 30–40 per cent with 70 per cent of the loss as plasma, and a progressive myocardial deterioration. Death of the animal occurs within two hours. *Method and Results:* Intravenous administration of an initial dose of propranolol of 10 mg. followed by a constant infusion of 200 μg./minute protected dogs from metabolic acidosis produced by infusion of norepinephrine in doses four times the LD_{50}. In these experiments the blood volume, blood *p*H, standard bicarbonate, and electrolytes remained unchanged from control levels. In further contrast to the usual effects of norepinephrine, there was a hypoglycemia and an apparent increase in glucose utilization. *Summary:* These results suggest that propranolol acts by causing a catecholamine blockade not only at the beta-adrenergic endings, but also at a tissue and cellular level, and that it influences all membrane transport of glucose. The prevention of catecholamine induced acidosis by propanolol indicates a more extensive effect than those related only to the nervous system.

Effects of Diethyl Ether on Ventilation with or without Vagal, Somatic and Sympathetic Blockade in Dogs. MUSA MUALLEM, M.D., and C. PHILIP LARSON, JR., M.D., *Department of Anesthesia, University of California Medical Center, San Francisco.* We have observed that diethyl ether produces a progressive depression of the ventilatory response to inhaled CO_2 at increasing anesthetic concentrations. Despite this, the resting arterial P_{CO_2} (Pa_{CO_2}) remains at (man) or below (dogs) normal up to an alveolar ether concentration of 5 per cent (2.5 MAC) in man (ANESTHESIOLOGY, Work-In Progress Abstract, 1967, Larson) and 4.5 per cent (1.5 MAC) in dogs. MAC is the minimum alveolar anesthetic concentration required to prevent movement in 50 per cent of animals in response to a painful stimulus. From the above it would appear that ether does not depress, and in fact may stimulate, resting ventilation. The suggested explanations for this include: (1) sensitization of pulmonary stretch receptors, (2) irritation of the lower respiratory tract, (3) stimulation of extrapulmonary peripheral sensory receptors, (4) release of catecholamines from the adrenals by central sympathetic stimulation, (5) development of acidosis and (6) direct stimulation of the central respiratory mechanism. *Method:* We tested the first four of these hypotheses by blocking the nerve pathways from or to these sites. In unpremedicated dogs anesthetized with ether to 1, 1.5 and 2 MAC (3, 4.5 and 6 per cent alveolar ether, respectively), we measured Pa_{CO_2} before and after bilateral vagotomy, high spinal anesthesia, vagotomy and spinal anesthesia combined and vagotomy spinal and carotid sinus denervation. *Results:* The data we obtained are given in the following table.

	V	S	V + S	V + S + CSD
No. of Dogs	6	6	6	3
Pre Pa_{CO_2} block 1 MAC	32.6 ±5.7	35 ± 1.6	32.3 ± 3	29.8 27–33
1.5 MAC	33.7 ±6.6	38.4 ± 3.1	34.3 ± 5.5	29.6 24–32
2 MAC	75 ±32	67.9 ± 6.7	55.2 ±11.2	41.3 40–42
Post Pa_{CO_2} block 1 MAC	25.5 ±5.7	36.3 ± 6	30.1 ± 5.7	27.8 25–31
1.5 MAC	30.4 ±7.5	39.8 ±12.9	32.8 ± 5.4	30.6 28–33
2 MAC	51 ±22	76.7 ±15	54.9 ±17.4	43.1 35–51

V. vagotomy, S. spinal, CSD, carotid sinus denervation. Mean values with 1 standard deviation or range.