The Effects of Anesthetics on the Release of Norepinephrine from Sympathetic Nerves. S. H. NGAI, M.D., SIDNEY OZERNITSKY, M.D., and P. M. DIAZ, M.D., *Department of Anesthesiology, College of Physicians and Surgeons, Columbia University, New York, N. Y.* Biosynthesis, uptake and release of norepinephrine in the peripheral sympathetic nervous tissue play important roles in circulatory homeostasis. Differences among anesthetics in their action on these mechansims could well explain their varied effects on the circulation. The present study deals with the release of norepinephrine as affected by anesthetics, particularly cyclopropane. *Method:* One series of experiments was carried out in dogs anesthetized with chloralose (100 mg./kg., intravenously). The chest was opened through a midline sternotomy. The coronary sinus outflow was diverted with a modified Morawitz cannula, measured with an electromagnetic flowmeter and returned to the right atrium. The arterial pressure was measured with a Statham transducer. A small catheter (PE 50) was inserted retrograde through a branch of the anterior descending coronary artery with its tip placed near the left main coronary artery. The myocardial norepinephrine store was labelled by intraarterial infusion of *dl*-H ^{3}-norepinephrine (100 μC.). Arterial and coronary sinus blood samples were obtained at intervals. After appropriate treatment radioactivity in the plasma water was measured with a liquid scintillating counter. The body temperature was maintained at 38–39° C. with heating blankets. A Frumin-Lee respirator provided constant-volume ventilation. Cyclopropane (15 per cent) or halothane (1 per cent) was administered with a non-rebreathing system after a period of control observation. *Results:* With cyclopropane the pattern of norepinephrine release was not altered in seven of ten animals. In the remaining three radioactivity in the coronary sinus blood increased during inhalation of cyclopropane. Electrical stimulation of the left stellate ganglion raised the arterial pressure and the coronary sinus outflow with a concomitant increase in norepinephrine release. Cyclopropane did not appear to influence these effects of stimulation either. In four animals no change in release of norepinephrine was observed during inhalation of halothane. In another series of experiments using decerebrate cats, the norephinephrine store in the iris was labelled by intracarotid infusion of *dl*-H^3-norepinephrine. The anterior chamber was perfused with Ringer's solution and the radioactivity of effluent perfusate measured. Electrical stimulation of the cervical sympathetic trunk caused a papillary dilation and a release of norepinephrine into the anterior chamber. The magnitude of release became constant after five to seven bouts of stimulation. Cyclopropane caused no change in the response in five of eight animals, a decrease in release in two and an increase in one. *Conclusions:* It may be concluded that under these experimental conditions cyclopropane anesthesia did not increase the release of norepinephrine from the peripheral sympathetic nerves. Studies are being carried out to elucidate further the adrenergic transmitter mechanisms in the periphery. (Supported by USPHS Grants 5T1-GM-00056 and GM-09069.)

A Possible Ventilatory Effect of Carbonic Anhydrase Inhibition Following Topical Sulfamylon in Burned Patients. PAUL J. SCHANER, CAPT., MC; JERRY M. SHUCK, CAPT., MC; CHARLES R. RITCHEY, MAJ., MSC, *US Army Surgical Research Unit, Brooke Army Medical Center, Fort Sam Houston, Texas.* Hyperpnea has been noted frequently in patients with 30 per cent or greater total body burns who are treated with Sulfamylon®. Arterial blood gas studies in these patients disclosed normal to subnormal P_{O_2}, hypocapnia, some degree of base deficit, and normal to alkaline *p*H. Sulfamylon therapy causes carbonic anhydrase inhibition (CAI). The study was undertaken to determine the possible ventilatory effect of prolonged inhibition of carbonic anhydrase (CA) in the burned patient. *Methods:* Patients sustaining a 30 per cent or greater total body burn were studied for as long as 60 hours after the initial application of Sulfamylon. Parameters studied included: inhibition of renal CA as judged by urinary titratable acidity, bicarbonate and ammonium excretion, degree of red blood cell CAI, blood levels of Sulfamylon, arterial *p*H and blood gases, respiratory minute volume, with end-tidal P_{CO_2}. *Results:* Renal CAI