tained during administration by nasal catheter are believed to be sufficient to cause these alterations. These capillary changes are believed to be responsible for the development of "oxygen dependence" and for the variations in pulmonary function which occurred during and after inhalation of high concentrations of oxygen. Presumably, they are reversible at least until the time fibrosis supervenes. Because of the high frequency of oxygen administration, the lesion has been so commonplace as to be considered a normal variation in pulmonary structure. Emphasis is placed upon the importance of appreciating the potential effect of oxygen on the pulmonary parenchyma and of differentiating these lesions from those of various obscure pulmonary diseases. (*Pratt, P. C.: Pulmonary Capillary Proliferation Induced by Oxygen Inhalation, Am. J. Path. 34: 1033 (Nov.–Dec.) 1958.*)

CARDIOTOXIC VASOPRESSORS Rats were injected subcutaneously on two consecutive days with various doses of four sympathomimetic amines. The animals were sacrificed at 48 hours and myocardial lesions were graded. Isoproterenol caused more severe infarct-like myocardial necrosis than did *l*-epinephrine, *l*-arterenol, or ephedrine. It was concluded that the myocardial lesions produced by these drugs are related to their cardiac stimulant properties. (*Chappel, C. I., and others: Comparison of Cardiotoxic Actions of Certain Sympathomimetic Amines, Canad. J. Biochem. & Physiol. 37: 35 (Jan.) 1959.*)

NOREPINEPHRINE Pulmonary congestion and pulmonary capillary hydrostatic pressure are increased while cardiac output is decreased in patients with aortic insufficiency who are infused with solutions of norepinephrine. These changes do not occur if the limbs are excluded by tourniquets even though arterial pressure rises. It is thought that blood is squeezed from the periphery to the pulmonary circulation where it tends to remain since the heart is not able to handle the increased load especially as the aortic-ventricular diastolic pressure gradient is decreased by the elevated arterial pressures. (*Regan, T. J., and others: Mechanism of Norepinephrine-Induced Pulmonary Congestion in Aortic Insufficiency, J. Lab. & Clin. Med. 52: 938 (Dec.) 1958.*)

LEVARTERENOL EXTRAVASATION Experiments on rabbits have demonstrated the value of Regitine and Benodaine in preventing necrosis from levarterenol extravasation. Procaine (1 or 2 per cent) failed to prevent ischemia or necrosis in rabbits. Animal experiments have shown that heparin prevents necrosis. Addition of 10 mg. of heparin to the norepinephrine solution for patients in cardiogenic shock is suggested. Even this small amount of heparin might increase probability of bleeding in postoperative shock and therefore should not be used. In their experiments, the mixture of norepinephrine, meticortelone and heparin caused decidedly less necrosis than norepinephrine with meticortelone. (*Pelner, L., Waldman, S., and Rhoades, M. C.: The Problem of Levarterenol Extravasation–An Experimental Study, Am. J. M. Sc. 236: 755 (Dec.) 1958.*)

RESERPINE Forty-four experiments on the heart-lung preparation of the dog revealed that 100 micrograms per kilogram of reserpine depleted the heart of norepinephrine within 24 hours without causing apparent side effects. Norepinephrine was not detectable after this for 6 days, but within 10 to 20 days afterward, normal stores of norepinephrine were found. (*Waud, D. R., Kottegoda, S. R., and Krayer, O.: Threshold Dose and Time Course of Norepinephrine Depletion of Mammalian Heart by Reserpine, J. Pharmacol. & Exper. Therap. 124: 340 (Dec.) 1958.*)

FLUOTHANE The effects of Fluothane upon uterine contractility was studied by means of the external tocograph in 12 patients at delivery. In each patient, Fluothane clearly inhibited uterine contractility and obliterated both spontaneous and oxytocin induced contractions. This effect was rapidly produced at a relatively light plane of anesthesia and quickly disappeared when consciousness was recovered. (*Embrey, M. P., Garrett, W. J., and Pryer, D. L.: Inhibitory Action of Halothane on Contractility of Human Pregnant Uterus, Lancet 2: 1093 (Nov. 22) 1958.*)