negra, P., et al.: Effect of Angiotensin on Myocardial Function, Amer. J. Physiol. 218: 1267, 1970.) EDITOR'S COMMENT: An interesting paper on the basic cardiovascular effect of angiotensin in a normal hemodynamic state. The relevancy of these findings to the clinical situation remains to be established.

ETHYL ALCOHOL AND CORONARY VASODILATION Ethyl alcohol has been considered to be an effective therapeutic agent for angina pectoris, although experimental evidence has not necessarily confirmed the clinical impression. In six conscious dogs with implanted electromagnetic flow transducers, ethyl alcohol (1 g/kg intravenously) resulted in significant decreases in coronary (41 per cent) and peripheral (17 per cent) vascular resistance, whereas heart rate and mean aortic pressure were not changed significantly. Both propranolol and intravenous pentobarbital anesthesia diminished the coronary and peripheral vasodilator effects of ethyl alcohol. The experiments demonstrated a coronary vasodilator effect of alcohol in the dog. Whether coronary vasodilation is effected in the presence of myocardial ischemia remains to be demonstrated. (*Pitt, B., Sugishita, Y., Green, H. L., et al.: Coronary Hemodynamic Effects of Ethyl Alcohol in the Conscious Dog, Amer. J. Physiol. 219: 175, 1970.*)

ANGINA AND LEFT VENTRICULAR FAILURE Clinical electrocardiographic and hemodynamic studies were made of six patients with uncomplicated angina pectoris and six normal controls during rest and during supine leg exercise (bicycle and ergometer). Measurements included aortic and left-ventricular pressures obtained during left heart catheterization, cardiac output, electrocardiogram, and arterial blood gases. All six patients studied (age range 35–42 years) had histories of severe breathlessness accompanying the onset of angina, and all developed anginal pain within one to six minutes after onset of exercise. Onset of pain was associated with the appearance of striking dyspnea, S-T segment depression, and a significant increase in left ventricular end-diastolic pressure, as well as increases in mean arterial pressure and systemic vascular resistance. The average oxygen uptake, heart rate, stroke volume, and cardiac output were not different from those of control patients at rest, but average heart rate rose and stroke volume fell significantly with the appearance of anginal pain. Although minute ventilation nearly doubled on exercise in patients with angina compared with the control group, there was no significant alteration in arterial oxygenation. Thus, acute, severe, and rapidly reversible left ventricular failure can accompany the onset of pain in angina pectoris. Inability to adapt the fiber-length relationship for the necessary rise in stroke volume is probably secondary to myocardial ischemia. (*Taylor, B. S. S. H.: Reversible Left-ventricular Failure in Angina Pectoris, Lancet 2: 902–906, 1970.*) EDITOR'S COMMENT: The recent upsurge in surgical relief of intractable angina pectoris and coronary occlusive disease has stimulated active study of cardiovascular changes secondary to disease. The information thus obtained is of critical importance to the anesthetist charged with the care of these patients for surgery other than cardiac. Since the onset of pain is not available as a warning signal during general anesthesia, we must begin a thorough study of factors which will alert us to potential trouble. The old adage about adequate oxygenation during general anesthesia is no longer adequate. Patients with coronary artery disease and angina react in a peculiar manner to autonomic stimuli, including tachycardia or an elevated arterial blood pressure. It is conceivable that the workload imposed on an otherwise ischemic myocardium by anesthetic agents known to activate autonomic mechanisms may not be beneficial and could explain the higher incidence of postoperative myocardial infarction in this group. This paper emphasizes the importance of transient left ventricular failure in the patient with angina and a history of dyspnea on exertion. The stress of illness, anesthesia, and surgery can easily progress to the development of necrosis of muscle. Better criteria for monitoring these patients during and after surgery need to be evolved.