have been due to direct stimulation of arterial muscle by oxygen. Although no conclusion about mortality can be drawn from this study, the changes observed appeared to be beneficial. (*Kenmure, A. C. F., and others: Circulatory and Metabolic Effects of Oxygen in Myocardial Infarction, Brit. Med. J. 4: 360 (Nov.) 1968.*)

BARORECEPTORS IN SLEEP Baroreflex function, as measured by blood pressure and pulse rate responses to intravenous angiotension, was determined in human subjects awake and during sleep. During wakefulness and sleep, a linear relationship was found between the increase in arterial pressure and the increase in pulse interval from beat to beat. However, the reflex slowing did seem to be greater during sleep, and may maintain a lower arterial blood pressure during sleep. (*Smyth, H. S., and others: Reflex Regulation of Arterial Pressure during Sleep in Man, Circ. Res. 24: 109 (Jan.) 1969.*)

CAROTID SINUS The carotid sinus, a fusiform enlargement of the internal carotid artery at the level of the carotid bifurcation, provides an important physiologic mechanism for reflex regulation of cardiovascular function. Specialized nervous receptors embedded in the sinus wall constantly monitor systemic arterial blood pressure and act to maintain it within relatively narrow limits. These receptors constitute the sensing element in a reflex arc whose afferent limb is formed by the glossopharyngeal, and probably the vagus, nerves and whose efferent limb is the cardiovascular autonomic outflow. The principal reflex mechanisms arising from the carotid sinus and other baroreceptor areas affect circulation, heart rate, and respiration. The undisputed changes elicited by an increase in intrasinusal tension are a reduction in systemic arterial pressure, slowing of heart rate, and depression of respiration. Conversely, a decrease in tension within the sinus increases the blood pressure, accelerates the heart, and augments respiration. (*Thomas, J. E.: Hyperactive Carotid Sinus Reflex and Carotid Sinus Syncope, Mayo Clin. Proc. 44: 127 (Feb.) 1969.*)

PULMONARY HYPERTENSION Pulmonary artery pressure (PAP) was found to be elevated in eight patients in acute respiratory failure. All of these patients were hypoxic (mean $Pa_{O_2} = 38.7$ torr) and hypercarbic (mean $PA_{CO_2} = 64$ torr). Treatment consisting of antibiotics, bronchodilators, IPPB and postural drainage resulted in a decrease in PAP synchronous with an increase in Pa_{O_2} and a reduction in Pa_{CO_2}. Acute early changes in PAP could be induced by breathing 24 and 28 per cent oxygen or by infusion of acetylcholine into the pulmonary circulation. It is suggested that acute pulmonary hypertension seen in patients with acute respiratory failure is due primarily to pulmonary vasoconstriction induced by hypoxia. (*Abraham, A. S., and others: Factors Contributing to the Reversible Pulmonary Hypertension of Patients with Acute Respiratory Failure by Serial Observations during Recovery, Circ. Res. 24: 51 (Jan.) 1969.*)

SHOCK Obstruction to the inferior vena cava was demonstrated as the cause of hypertension and subsequent shock in acute gastric dilatation in 18 dogs. Neurogenic reflex mechanisms were not demonstrated. (*Passi, R. B., and others: Pathophysiologic Mechanisms of Shock in Acute Gastric Dilatation, Surgery 65: 298 (Feb.) 1969.*)

LYTIC COCKTAIL The lytic cocktail mixture of meperidine, promethazine and chlorpromazine, popular several years ago for premedication, is still used frequently for sedation in patients undergoing cardiac catheterization. The pulmonary and systemic vascular effects of the individual agents and of the lytic cocktail mixture were studied in dogs. Meperidine, 2 mg/kg, decreased cardiac output (CO), increased systemic vascular resistance (SVR), and increased pulmonary vascular resistance (PVR). Promethazine (1 mg/kg) increased CO, SVR, and PVR. Chlorpromazine (1 mg/kg) increased CO, decreased SVR, but increased PVR. The increased PVR with meperidine passively reflected the decreased cardiac output. However, the increased PVR with promethazine and chlorpromazine probably represented active pulmonary vasocon-