

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. (Kennure, 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 intrasinus 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 P_{aO_2} = 38.7 torr) and hypercarbic (mean P_{aCO_2} = 64 torr). Treatment consisting of antibiotics, bronchodilators, IPPB and postural drainage resulted in a decrease in PAP synchronous with an increase in P_{aO_2} and a reduction in P_{aCO_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-

striction. The net effect of the mixture was no significant change in CO, but a 60 per cent increase in PVR and a 20 per cent decrease in SVR. An increased PVR and decreased SVR could alter the magnitude of left-to-right shunts, leading to misinterpretation of cardiac catheterization data. (Goldberg, S. J., and others: *The Effects of Meperidine, Promethazine, and Chlorpromazine on Pulmonary and Systemic Circulation*, *Amer. Heart J.* 77: 214 (Feb.) 1969.)

HYPERVOLEMIA Deliberate hypervolemia is effective in restoring cardiac output and renal blood flow to normal during maintained hypotension. The accompanying acidosis is also mitigated and the diminished clot-tensile strength is restored. (Shaftan, G. W., and others: *Hypovolemia during Ganglionic Blockade Hypotension*, *Surgery* 65: 321 (Feb.) 1969.)

BLOOD CLOTTING The clotting time of human plasma and blood could be accelerated by contact with human skin, the greatest activity being associated with those areas rich in sebaceous secretions. Cleansing with alcohol reduced this clot-promoting ability and rubbing cleansed skin or areas rich in sebaceous secretion restored it. (Ogston, D., and others: *Studies on the Clot-promoting Effect of Skin*, *J. Lab. Clin. Med.* 73: 70 (Jan.) 1969.)

Respiration

INTERCOSTAL MUSCLE ACTIVITY No evidence of reciprocal electrical activity in internal and external intercostal muscles of the rabbit could be shown during quiet breathing by use of clip electrodes. The greater part of the expiratory phase was characterized by slight activity of both internal and external intercostals. Inspiration was accompanied by continuous activity in both sets of muscles until the last 20 to 40 msec of this phase. (Boyd, W. H.: *Electrical Activity of Intercostal Muscles during Quiet Breathing in Rabbits*, *Canad. J. Physiol. Pharmacol.* 46: 749 (Sept.) 1968.)

PULMONARY FUNCTION Pulmonary function of infants and children was studied by measurement of alveolar ventilation and alveolar-arterial gas tension differences for O₂, CO₂, and N₂. The increased ventilation in these subjects was effective in eliminating CO₂, but there was evidence of considerable unevenness of distribution of ventilation-perfusion ratio (V_A/Q). A measure of V_A/Q unevenness was obtained by the use of the urine-alveolar nitrogen tension difference. It is likely that under the prevailing conditions of hyperventilation and hypoperfusion, maldistribution of perfusion is a major abnormality. Uneven distribution of perfusion is probably due to the effects of gravity enhanced by low pulmonary arterial pressure and blood flow. This represents an exaggeration of the normal physiologic overperfusion of the dependent parts of the lungs. (Lees, M. H., and others: *Ventilation Perfusion Relationships in Children with Heart Disease and Diminished Pulmonary Blood Flow*, *Pediatrics* 42: 778 (Nov.) 1968.)

ABTRACTER'S COMMENT: This study presents excellent scientific confirmation that patients with congenital heart disease may also have significant respiratory limitations. Significant derangements in either of these two vital systems cannot be managed without careful attention to the other.

ATMOSPHERIC OXYGEN The outline of the evolution of oxygen now seems clear, although argument and study will continue for many years to establish the details and the precise dating of the various stages. We see a remarkable chain of events, many of them irreversible, and most depending critically on the physical and chemical properties of the basic chemical substances of which the earth is composed. Each stage in the development of life is based on previous changes and often, the attainment of the new stage makes continuation of the previous changes impossible. Thus, we can never go back. If life were to be destroyed today, it could not be restarted unless the condition of the earth were to revert to that of at least 3,000 million years ago. We sit at the apex of a pyramid of development lasting 4,500 million years and man is