following cessation of Dicumarol. (*Zifer, A. M., Scopp, I. M., Beck, J., Baum, J., and Berger, A. R.: Profound Bleeding after Dental Extractions during Dicumarol Therapy, New England J. Med. 256: 351 (Feb. 21) 1957.*)

PULMONARY CIRCULATION Inhalation of 5 per cent oxygen in dogs caused a fall of 45 per cent in oxygen saturation of arterial blood and a 25 per cent rise in pulmonary arterial pressure. The immediate pressure rise due to anoxia was delayed and less intense when the carotid and aortic bodies were denervated. Other evidence indicated a vasoconstriction of pulmonary vessels with anoxia. (*Aviado, D. M., and others: Effects of Anoxia on Pulmonary Circulation: Reflex Pulmonary Vasoconstriction, Am. J. Physiol. 189: 253 (May) 1957.*)

PULMONARY ARTERY PRESSURE Increases in intratracheal pressure are quickly reflected in the pressure within the pulmonary artery. Block of the vagus nerve increases respiratory excursion but has no effect on pulmonary artery pressure in the normal subject. Vagus block in bronchospastic patients results in a decrease of systolic and diastolic pulmonary artery pressures. (*Abbott, O. A., and others: Comparative Studies of Function of Human Vagus and Sympathetic Nerves in Relation to Pulmonary Bed, Surgery 42: 170 (July) 1957.*)

PULMONARY RESISTANCE In patients with congestive heart failure the assumption of the supine position results in a marked increase in viscous (air flow) resistance. The work of breathing in these patients is increased 25 per cent. This dyspnea of recumbency is possibly due to reduced patency of the air passages at low levels of lung inflation. (*Cherniak, R. M., and others: Significance of Pulmonary Elastic and Viscous Resistance in Orthopnea, Circulation 15: 859 (June) 1957.*)

PULMONARY VASCULAR RESISTANCE The injection of 1.0 to 1.5 mg. of acetylcholine directly into the pulmonary artery reduced pulmonary arterial systolic and diastolic pressures and calculated pulmonary vascular resistance in three patients with primary pulmonary hypertension, and in 7 of 9 patients with mitral stenosis. Systemic effects were minimal. It was concluded that there was a functional vasoconstriction in the pulmonary vascular bed, capable of being reversed by acetylcholine, in both mitral stenosis and in primary pulmonary hypertension. (*Wood, P., Besterman, E. M., Towers, M. K., and McIlroy, M. B.: Effect of Acetylcholine on Pulmonary Vascular Resistance and Left Atrial Pressure in Mitral Stenosis, Brit. Heart J. 19: 279 (April) 1957.*)

COLLATERAL CIRCULATION The collateral circulation of the dog's lung, that portion of the bronchial flow that drains into the pulmonary veins, normally amounts to 0.5 to 1 per cent of the total flow. (*Salisbury, P. F., Weil, P., and State, D.: Factors Influencing Collateral Blood Flow to Dog's Lung, Circulation Res. 5: 303 (May) 1957.*)

RESPIRATION RESISTANCE Measurements of the elastic and nonelastic resistance to breathing in normal patients and those with diseased cardiorespiratory systems was determined before, during and after general anesthesia and surgical operations. The work of breathing increased 200 to 500 per cent during general anesthesia and interference of the chest wall movements by surgical assistance, such as retractors. (*Brownlee, W. E., and Allbritton, F. F., Jr.: Work of Breathing During Surgical Operations, A. M. A. Arch. Surg. 74: 846 (June) 1957.*)

HYPOXIA AND HYPERCAPNIA Circulatory performance in hypoxia and hypercapnia represents the resultant of direct cardiac depressant effects and opposing reflexly mediated alterations in contractility and vascular tone. In dogs cardiac contractility increased on 10 per cent carbon dioxide but decreased steadily when reflex compensation was prevented by spinal transection, cardiac depression occurred on return to 100 per cent carbon dioxide. Though mild hypercapnia increased amplitude in both dog and man, severe hypercapnia markedly depressed the heart until reflex epinephrine discharge restored contractility toward normal. (*Honig, C. R., and Tenney, S. M.: Determinants of Circulatory Response to Hy-*