CARDIAC DEFIBRILLATION The late mortality in dogs following successful defibrillation is considered to be due to cardiac burns. Padding of the electrodes is suggested as a method for preventing this difficulty. (*Kortz, A. B., and Swan, H.: Electrical Ventricular Defibrillation, A. M. A. Arch. Surg. 74: 911 (June) 1957.*)

EKG IN CHEST SURGERY In 444 patients, a six-month postoperative EKG was compared with the preoperative one. Operations were thoracoplasty and pulmonary resection for tuberculosis. Ninety-four patients showed a change in the position of the heart. In 113 patients followed in first few weeks postoperatively, 35 showed extensive S-T and T wave changes probably due to pericardial irritation. (*Laros, C. D., and van der Slikke, L. B.: Influence of Intra- and Extra-Thoracic Operations on Electrocardiograms in Patients with Pulmonary Tuberculosis, J. Thoracic Surg. 34: 11 (July) 1957.*)

ATRIAL RHYTHM In dog experiments, it was shown that adrenalin and noradrenalin increase the duration of the atrial action potential, thus inhibiting atrial fibrillation due to electrical stimulation. Acetylcholine has the opposite effect. (*Burn, J. H., Gunning, A. J., and Walker, J. M.: Effects of Noradrenalin and Adrenalin on Atrial Rhythm in Heart-Lung Preparation, J. Physiol. 137: 141 (June 18) 1957.*)

HUMORAL VASODILATATION Hyperventilation, causing a fall in carbon dioxide tension, is demonstrated in man to produce in the forearm a vasodilatation confined to muscle vessels. Arterial blood pressure falls slightly in response to this vasodilatation, which is apparently due to a humoral mechanism, since it occurs in the presence of brachial plexus block. (*Roddie, I. C., Shepherd, J. T., and Whelan, R. F.: Humoral Vasodilatation in Forearm during Voluntary Hyperventilation, J. Physiol. 137: 80 (June 18) 1957.*)

VASODEPRESSOR SYNCOPE This can be produced by 60 degrees head-up tilt with the aid of sodium nitrite. The main feature is a widespread loss of peripheral resistance in the face of the inability of the heart to increase its output. This is probably due to a limited inflow. (*Weissler, A. M., and others: Vasodepressor Syncope: Factors Influencing Cardiac Output, Circulation 15: 875 (June) 1957.*)

ABNORMAL BLEEDING For extensive surgery, fresh blood less than six hours old, collected in plastic bags, is available. After each 1,500 ml. of older blood, 500 cc. of fresh blood is given plus 50 to 100 mg. of oil-soluble vitamin K_1 (intravenously). For each 2,000 ml. of blood, 1 gram of calcium chloride is given over a 15-minute period. Hydrocortisone (100 mg.) is given intravenously. (*Crehan, J. P.: Abnormal Bleeding in Surgical Patient, S. Clin. North America 37: 803 (June) 1957.*)

HEMORRHAGE In 8 of 12 dogs with sinoaortic denervation there was respiratory stimulation following hemorrhage. This respiratory stimulation began with the fall in blood pressure and reached a peak with the lowest blood pressure. The rapid fall in blood pressure may be the stimulus to respiration rather than a decreased blood flow through the respiratory center. (*Schopp, R. T., and others: Mechanisms of Respiratory Stimulation During Hemorrhage, Am. J. Physiol. 189: 117 (April) 1957.*)

COLD BLOOD Using constant perfusion rates of chilled blood in carotid artery, coronary artery, and femoral artery, changes were recorded in perfusion pressures and possible causes for changes in blood flow in these organs were discussed. (*Senning, A., and Olsson, P. I.: Changes in Vascular Tonus During Cerebral and Regional Hypothermia, Acta chir. scandinav. 112: 209 (March) 1957*).

HEMORRHAGE Profound and prolonged hemorrhage for 4 and 5 days was reported in two patients in whom dental extractions were performed during Dicumarol therapy. Discontinuation of Dicumarol therapy two days prior to surgery is recommended. Dicumarol therapy should be resumed following surgery as soon as hemostasis is certain in view of the high incidence of recurrent thromboembolism

following cessation of Dicumarol. (*Ziffer, 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-*