body temperature and avoidance of use of cold blood when large quantities are needed are prophylactic measures against ventricular fibrillation. (*MacLean, L. D., and van Tyn, R. A.: Ventricular Defibrillation, J. A. M. A. 175: 471 (Feb. 11) 1961.*)

DEFIBRILLATOR The shock of the human heart should not exceed 250 volts (12 amp.) for 0.2 second. Seldom does one find it necessary to exceed 5 amp. for 0.2 second. Excessive stimulation will also produce an atrioventricular block or asystole rather than a normal sinus rhythm. The use of saline in the pericardium to moisten the electrodes caused severe abnormalities of electrical conduction after the shock. These abnormalities could be reversed by the application of mammalian Ringer's solution. (*Shephard, R. J.: Design of Cardiac Defibrillator, Brit. Heart J. 23: 7 (Jan.) 1961.*)

HEART SOUNDS The intensity of cardiac sounds is frequently at or near the lower level of hearing, therefore, one must pay close attention to a particular sound or event in the cardiac sound cycle in order to obtain a good evaluation. The first and second heart sounds are relatively high pitched and result from valve closure. The interval between the first and second sound approximates mechanical systole of the ventricles. The third and fourth sounds are low pitched ventricular filling sounds that may occur during diastole. Opening snaps of the mitral and tricuspid valves are frequently associated with atrioventricular valve pathology. (*Schwartz, M. L., and Little, R. C.: Physiologic Basis for Heart Sounds and Their Clinical Significance. New Engl. J. Med. 264: 280 (Feb. 9) 1961.*)

BALLISTOCARDIOGRAPHY Quantitative ballistocardiography proved to be a practical and clinically useful research tool. In 7 out of 12 patients myocardial depression was demonstrated during Fluothane anesthesia. Ganglionic blockade and peripheral vasodilatation are insufficient to explain the hypotension during Fluothane anesthesia. (*Eger, W., and Hügin, W.: Ballistographic Investigations During Narcosis, Especially Concerning Hypotension Under Fluothane, Der Anaesthesist 10: 38 (Feb.) 1961.*)

VASOPRESSORS Different vasopressors were given during Fluothane-induced hypotension. All of them elevated peripheral resistance but caused a marked reduction of stroke and minute volumes as shown by ballistography. Anticholinergic drugs prevented hypotension to some extent without interfering with peripheral blood flow. (*Hügin, W., and Eger, W.: Ballistographic Investigations Concerning Effect of Vasopressors in Halothane Anesthesia, Der Anaesthesist 10: 44 (Feb.) 1961.*)

PERIPHERAL RESISTANCE Quantitative ballistocardiography showed that thiopental caused a marked reduction of stroke and minute volume with moderately lowered blood pressure. There was in every case a marked and sudden increase of peripheral resistance which subsides in about ten minutes after a single sleeping dose. Third stage cyclopropane anesthesia also caused increased peripheral resistance but with elevated blood pressure. These effects are prevented or abolished by *d*-tubocurare or ganglionic blockers. (*Hügin, W., and Eger, W.: Ballistographic Investigations Concerning Changes in Peripheral Resistance due to Thiopental or Cyclopropane Narcosis, Der Anaesthesist 10: 46 (Feb.) 1961.*)

CORONARY FLOW Changes in the hemodynamics of coronary blood flow were revealed in dogs by producing varying degrees of cardiac failure, with the aid of graded constriction of the pulmonary artery. The coronary blood flow may be considered the critical factor in determining cardiac performance and diastolic size. (*Bacaner, M., and others: Coronary Blood Flow as Critical Determinant of Cardiac Performance and Cardiac Size, Amer. J. Med. 30: 392 (Mar.) 1961.*)

BLOOD BRAIN BARRIER There may be no morphological evidence of a blood-brain barrier. The relationships for various agents may be explicable in terms of central nervous system metabolism and it is unwise to assume

an impediment for any one molecule by analogy with any other. Most evidence from dye studies has been discredited. (*Dobbing, J.: Blood Brain Barrier, Physiol. Rev. 41: 130 (Jan.) 1961.*)

PULMONARY CIRCULATION In normal pulmonary circulation vasomotor activity is slight when compared to mechanical influences. The adjustment of alveolar perfusion to alveolar ventilation is good when the patient is supine; but in the lateral or standing position, the upper lung becomes hyperventilated with respect to perfuson and the lower lung becomes overperfused. This is manifested by higher respiratory exchange ratios and by lower oxygen uptakes in the upper lobes. (*Fishman, A. P.: Respiratory Gases in Regulation of Pulmonary Circulation, Physiol. Rev. 41: 214 (Jan.) 1961.*)

PULMONARY ANATOMY Three distinct subgross lung types are recognized: type I is represented by the cow, pig, and lamb; type II by the dog, cat, and monkey; type III by the horse and man. Great caution should be exercised in the choice of an experimental animal for pulmonary studies if the results are to be applied to man. Known interspecies anatomical differences, which at times can be severe, and known interspecies differences in susceptibility to disease not only reinforce this concept but could cause the failure of any experiment which neglects them. (*McLaughlin, R. F., and others: Subgross Pulmonary Anatomy in Various Mammals and Man, J. A. M. A. 175: 694 (Feb. 25) 1961.*)

VENTILATION CONTROL A large volume of evidence demonstrates conclusively that carbon dioxide is a powerful respiratory stimulant, and there is indisputable proof that the arterial tension of oxygen and the pH of the arterial blood do have some effect on respiration. The role of these three classical stimuli in the control of pulmonary ventilation has been investigated, and it has been found that they are not an adequate explanation either for the hyperpnea of muscular exercise in normal and abnormal subjects, or for the hyperventilation observed in patients suffering from cardiopulmonary disease. They apparently play a minor role, if any, in the control of pulmonary ventilation under normal conditions. Studies demonstrate that the arterial carbon dioxide tension is determined by the activity of the respiratory center. There is no correlation between carbon dioxide tension and pulmonary ventilation at various levels of physical exercise. Patients who hyperventilate have a low carbon dioxide tension, and these subjects are less sensitive to inspired carbon dioxide than is the normal, despite the low tension. Both normal and abnormal subjects show a decreasing sensitivity to carbon dioxide as the exercise stimulus is increased. These observations are not compatible with the hypothesis that carbon dioxide is an effective regulator of the respiratory response to muscular exercise. A possible change in the pH of the arterial blood cannot be invoked to explain the inadequacy of carbon dioxide. The tension of arterial oxygen can be dismissed as a factor in normal subjects, and investigation suggests that it is of little importance as a cause of hyperventilation in patients with cardiopulmonary disease. (*Sinnott, J.: Control of Pulmonary Ventilation in Physiological Hyperpnea, Canad. Med. Ass. J. 84: 471 (Mar. 4) 1961.*)

PARADOXICAL RESPIRATION From a discussion of the theory of paradoxical respiration following thoracoplasty ("pendelluft") and from experimental studies of ventilation, P_{CO_2} measurements in both main bronchi and intrapleural pressures, it is demonstrated that this condition does not exist. True "pendelluft" can exist in the presence of open hemithorax and an anesthesia bag distended to a pressure greater than atmospheric in a patient who is breathing spontaneously. The concept of "pendelluft" in the presence of a closed chest should be abandoned. (*Maloney, J. V., and others: Paradoxical Respiration and "Pendelluft," J. Thor. Cardiov. Surg. 41: 291 (Mar.) 1961.*)

NITROGEN NARCOSIS The effect of increased air pressure was studied on trained