the heartbeat was maintained up to 7 hours. Oxygen tension of the myocardium was maintained at 450 mm. of mercury. (*Camishion, R. C., and others: Retrograde Perfusion of the Coronary Arteries with Gaseous Oxygen during Cardiopulmonary Bypass, Surgery 59: 145 (Jan.) 1966.*)

BETA ADRENERGIC BLOCKADE The effect of beta adrenergic blockade on the circulatory response to maximal and submaximal exercise was studied in 7 normal subjects and 9 patients with various cardiac disorders. Beta adrenergic blockade invariably caused a reduction in heart rate, cardiac output, mean arterial pressure and left ventricular minute work. Arterial-venous oxygen difference increased and there was a small rise in central venous pressure. An apparent consequence of the impaired circulatory response to exercise was the reduction of maximal oxygen consumption and capacity for strenuous exertion. Even after beta adrenergic blockade, cardiac output still rose substantially during exercise. It is concluded that although the sympathetic stimulation of the heart plays a significant role in the circulatory response to exercise, its contribution is not very great quantitatively. It would appear that sympathetic stimulation of the heart is only one of a number of mechanisms by which the cardiac output is augmented during exercise. (*Epstein, S. E., and others: Effects of Beta-Adrenergic Blockade on the Cardiac Response to Maximal and Submaximal Exercise in Man, J. Clin. Invest. 44: 1745 (Nov.) 1965.*)

PREOPERATIVE DIGITALIS In unanesthetized patients with normal hearts, digitalis glycosides increase the rate at which intraventricular pressure rises, shorten the time interval from the onset of contraction to the development of peak tension of the ventricle and improve the force-velocity relationship of the contractile element of the myocardium, findings which indicate that these drugs augment the strength of myocardial contraction in normal man. Digitalis also constricts smooth muscle of peripheral arterial and venous beds, an action which may explain, in part, the failure of digitalis to augment cardiac output in patients without cardiac failure. In patients with congestive heart failure, digitalis increases myocardial contractility but, in contrast to normal subjects, also elevates diminished forearm blood flow and lowers elevated forearm vascular resistance. The use of digitalis preparations would appear to be indicated in cardiac patients without overt heart failure, in whom the development of cardiac decompensation is feared because of a sudden excessive hemodynamic burden resulting from acute infection or surgical procedure. Since digitalis appears to augment rather than depress myocardial contractility of normal hearts, fear of its use prophylactically does not appear to be warranted. (*Mason, D. T.: Cardiovascular Effects of Digitalis in Normal Man, Clin. Pharmacol. Ther. 7: 1 (Jan.) 1966.*)

PREOPERATIVE DIGITALIS A four-year clinical study is reported involving 286 patients undergoing general thoracic and closed-heart procedures and 368 patients having open-heart surgery. Each group was divided into two categories, those who received preoperative digitalization and those who did not. The subsequent course of each group was compared and found to be strikingly dissimilar in favor of those receiving preoperative digitalis. The value of digitalis in certain specific clinical situations was demonstrated, namely, during general anesthesia, hemorrhage, massive transfusion, pulmonary resection, closed valvular commissurotomy and open-heart surgery with extracorporeal circulation. The clinical unimportance of the phenomenon of digitalis "wash out" during cardiopulmonary bypass was documented. The desirability of unhurried, careful preoperative digitalization over "crash" programs in times of stress was emphasized. (*Burman, S.: Digitalis and Thoracic Surgery, J. Thor. Cardiov. Surg. 50: 873 (Dec.) 1965.*)

PULMONARY CIRCULATION Measurements of pressure, flow and volumes of the pulmonary circulation were carried out in dogs in an attempt to localize the site of action of the vasoconstriction produced by hypoxia or the administration of serotonin. During hypoxia, either a rise or fall in pulmonary vascular resistance may occur. The rise is associated with a significant decrease in pulmonary