
VENOMOTOR CONTROL In anesthetized open-chest dogs on cardiopulmonary bypass with a fixed cardiac output, venomotor function was observed by measuring venous return to the oxygenator, a large return indicating reduction in the systemic venous bed (venoconstriction) and a reduced return indicating an enlarged bed (venodilatation). Neurohumeral stimuli affected the capacity of the systemic venous bed profoundly. Pressure changes in the carotid sinus and the cardiac chambers, variations in arterial oxygen and carbon dioxide, and vasodepressor drugs (noretiphen, epinephrine, trimethaphan), as well as anithypertensive drugs (reserpine, guanethidine), all significantly altered venomotor tone under these experimental conditions. Further, in the intact human, additional studies with oral guanethidine and reserpine indicated these agents block reflex venoconstriction. Such alterations of venous return undoubtedly play an important role in regulating cardiac output and arterial pressure. (Braunwald, E., and others: Reflex Control of the Systemic Venous Bed. Effects on Venous Tone of Vasoactive Drugs, and of Baroreceptor and Chemoreceptor Stimulation, Circulat. Res. 12: 539 (May) 1963.)

RESPIRATION AND AORTIC PRESSURE In pentobarbital anesthetized dogs, inspiration resulted in a drop in aortic blood pressure. This drop was found to be dependent upon both a reduction in the left ventricular stroke output and the transmitted fall of intrathoracic pressure. During cardiac tamponade and airway obstruction the fall was accentuated, in the first instance by a greater fall in left ventricular output, and in the second by a greater fall of intrathoracic pressure. (Shabetai, J., Fowler, N. O., and Gueron, M.: Effects of Respiration on Aortic Pressure and Flow, Amer. Heart J. 65: 525 (Apr.) 1963.)

OXYGEN REFLEXES Whereas acetylcholine increases pulmonary blood volume by actively dilating regions within the pulmonary circulation, inhalation of 100 per cent oxygen decreases pulmonary blood volume. Since, at the same time as the volume decreases, the pulmonary artery and left atrial mean pressures and, hence, pulmonary vascular distending pressure tend to fall, a passive mechanism of action for 100 per cent oxygen must be invoked. Possibly oxygen reflexes produce systemic venodilation with a consequent redistribution of blood from the pulmonary to the systemic compartment. Inhalation of 100 per cent oxygen may improve the status of patients with pulmonary edema, not only by increasing arterial oxygen content, but also by decreasing the amount of blood in the lungs, thereby relieving pulmonary congestion. (Glick, G., and others: Effects of Inhalation of 100 Per Cent Oxygen on Pulmonary Blood Volume in Patients with Organic Heart Disease, Circulation 27: 554 (Apr.) 1963.)

HYPOXIA Samples of blood were drawn from brachial arteries and the renal veins of patients being investigated for disorders of kidney function. Following control determinations, 8.5 per cent oxygen in nitrogen was administered for 25 minutes. The oxygen saturation of arterial blood fell to between 74 per cent and 87 per cent, that of renal vein blood to between 72 per cent and 80 per cent. Urine flow decreased to one-half or less during hypoxia. The PAH-clearance and inulin clearance also were reduced during hypoxia. (Duner, H., and Granberg, P.: Effect of Induced Hypoxia on Renal Function in Man, Acta Chir. Scand. 125: 253 (Mar.) 1963.)

CORONARY PERFUSION Cardiac function was studied after regional perfusion of the coronary arteries with oxygen unsaturated blood or isotonic colloidal solutions (dextran) at a pressure level equal to that in systemic arteries. Perfusion of 4 ml/minute during 30 minutes into the anterior descending coronary artery did not alter the rate of heartbeat, the aortic or left atrial pressures. Rapid perfusions of 20 ml/minute during or after the above perfusions, also did not alter heartbeat or pressures. The retropressure and backflow increased during these perfusions. Gradual exsanguination of the animals during dextran perfusion permitted total exsanguination with-