Left Ventricular Volume in Dogs, J. Clin. Invest. 42: 649 (May) 1963.)

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 vasoactive drugs (norepinephrine, epinephrine, trimethaphan), as well as antihypertensive 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-

out appearance of fibrillation. (*Petropoulos, P. C.: Effects on Cardiac Function During Long-Term Perfusion of the Coronary Arteries with Unsaturated Blood or Colloidal Isotonic Solutions, J. Thor. Cardiov. Surg. 45: 460 (Apr.) 1963.*)

AORTIC CLAMPING During cross clamping of the aorta a shunt was established, bridging the aortic clamp with a number 10 catheter, with a 13 gauge needle at each end. This permitted 50 to 75 ml. of blood flow per minute into the distal vessels and prevented or greatly reduced acidosis, hypoxia and vascular pooling. Release of the clamp was not followed by hypotension. (*Engler, H. S., and others: Shock Following Release of Aortic Cross-Clamping, Arch. Surg. 86: 791 (May) 1963.*)

HYPOTENSION Severe hypotension frequently accompanies the restoration of distal blood flow after an abdominal aortic aneurysmectomy. This problem was investigated in ten dogs whose aorta was cross clamped. The influence of systemically administered phenylephrine, and phenylephrine injected directly into the distal aorta prior to the release of the clamp, was observed. In the latter group, there was less disturbance of systemic blood pressure than with any of the other techniques. This concept was applied clinically in 15 patients prior to the release of the aortic clamps. The average fall in blood pressure of these patients was 15 mm. of mercury in contrast to 20 unselected patients not given this therapy whose average fall in blood pressure was 110 mm. of mercury. Hypotension is thought to be due to a reactive hyperemia and intravascular sequestration of a significant proportion of the total circulating blood volume into the lower extremities. (*Fry, W. J., and others: Prevention of Hypotension due to Aortic Release, Surg. Gynec. Obstet. 116: 301 (Mar.) 1963.*)

ARTERIAL HYPOTENSION Hemorrhage of 3 per cent body weight in dogs produced an average decrease in systolic blood pressure and skeletal muscle oxygen tension of 40 per cent. Administration of methoxamine restored blood pressure to control levels, and increased oxygen tension to 27 per cent below prehemorrhage levels. Mephentermine administration could only increase blood pressure to 27 per cent below control values, though it had the same effect on tissue oxygen tension as methoxamine. After hemorrhage, venous muscle lactate was higher than arterial muscle lactate. Administration of methoxamine tended to decrease, while mephentermine administration increased, this difference. (*Greene, N. M., and Willenkin, R. L.: Skeletal Muscle Oxygen Tension and Metabolism During Hemorrhagic Hypotension and Subsequent Vasopressor Administration, Yale J. Biol. Med. 35: 429 (Apr.) 1963.*)

SELECTIVE ISCHEMIA In five normal men studied during breath-holding and immersion of the face in water bradycardia and reduction of blood flow in the periphery (calf of the leg) averaging 80, 68, 35, 30 and 14 per cent, respectively, of control values. This is a protective mechanism (found also in diving animals) to shunt blood to vital organs during apneic diving, in effect producing a "heart-lung-brain" preparation. (*Elsner, R. W., Garcy, W. F., and Scholander, P. F.: Selective Ischemia in Diving Man, Amer. Heart J. 65: 571 (Apr.) 1963.*)

CONTROLLED HYPOTENSION Deliberate hypotension has been induced in 25 patients with nitrous oxide, ganglionic blocking agents, and two new substances, one a potent analgesic and the other a neuroleptic. The narcotic (Phentanyl) is brief in action and 1000 times more potent than meperidine. The neuroleptic is a butyrophenone with potent antiemetic effects and capable of producing cataleptic immobility. The level of hypotension was noted to be easily controlled and tachycardia and tachyphylaxis did not appear. After the operation the blood pressure rapidly returned to preoperative levels and full consciousness was regained in a few minutes. There was no operative pain or restlessness. Nalorphine was required in two patients to reverse respiratory depression. (*Larson, A. G.: New Technique for Inducing Controlled Hypotension, Lancet 1: 128 (Jan. 19) 1963.*)