between the clavicle and first rib utilizing a 6 inch 15 gauge Rochester needle is described. The plastic catheter is advanced into the innominate vein or superior vena cava over a Seldinger guide wire which is then removed and an infusion begun with a pressure monometer attached for monitoring central venous pressures (CVP). Although isolated absolute CVP measurements are of value only if they are exceptionally high or low, continuous monitoring of CVP for several days, if necessary, and correlation with associated fluid balance and therapy is useful in assessing and treating abnormal circulatory blood volume (ineffective venous return). Complications of the method have an incidence of 4–10 per cent and include pneumothorax, hemothorax, hydrothorax, brachial plexus injury, subclavian artery puncture, thrombophlebitis and breaking of the catheter with embolization. Increasing experience and proper technique minimize these. (*Longerbeam, J. K., and others: Central Venous Pressure Moniotring. A Useful Guide to Fluid Therapy During Shock and Other Forms of Cardiovascular Stress, Amer. J. Surg. 110: 220 (Aug.) 1965.*)

PULMONARY FLOW Pressures were measured in various segments of the pulmonary vascular bed in 20 closed-chest preparations in which airway hypoxia was acutely induced. Total pulmonary blood flow was simultaneously recorded by means of an electromagnetic flowmeter. In every experiment acute hypoxia resulted in increased pulmonary blood flow, pulmonary arterial pressure, and pulmonary arterial wedge pressure while left arterial pressure remained unchanged. A marked rise was noted in postcapillary pulmonary vascular resistance, while precapillary resistance fell. The net result was an insignificant change in total pulmonary vascular resistance. If the pulmonary artery wedge pressure accurately reflects pulmonary venous pressure, the constriction would appear to occur in the pulmonary veins. (*Berry, W. C., and others: Effects of Acute Hypoxia on Pressure, Flow, and Resistance in the Pulmonary Vascular Bed, Surgery 58: 404 (Aug.) 1965.*)

ACCELERATION Six dogs under morphine-pentobarbital anesthesia were exposed to forward accelerations of 2, 4 and 6 G for one minute and 6 G for three minutes while in the horizontal, 15 degree head-up, and 15 degree head-down positions breathing room air. Femoral artery oxygen saturation decreased at 6 G, averaging 11 per cent at the end of 60 seconds with return to normal 50 seconds after exposure. The average increase in pulmonary arterial-venous shunt when air was breathed during 1 G was 17 per cent. The oxygen saturation of mixed venous blood decreased during exposures to 2, 4 and 6 G. Changes in blood oxygen saturation were not systematically affected by the presumed differences in intrathoracic blood volume and position of the diaphragm associated with the 15 degree head-up or head-down positions. (*Banchero, N., and others: Blood Oxygen Changes Induced by Forward (+G) Acceleration, Aerospace Med. 36: 608 (July) 1965.*)

CITRATE TOXICITY Toxic effects of citrate relate only to the production of a reduced serum ionized calcium concentration which causes cardiac arrhythmia, reduced output, arrest and death. There is no evidence that coagulation difficulties result from citrate-induced hypocalcemia. If an animal is supported by cardiac massage or parallel pumping during the brief period of cardiac difficulty, ionized calcium is quickly mobilized and cardiac function restored. Metabolism of citrate appears unimportant as mobilization of calcium occurs much before the citrate is removed. These effects can be prevented by the use of ionized calcium. Open heart surgery with total cardiopulmonary bypass has been performed on 27 patients with acid-citrate blood modified with 2 mg. of heparin and 0.5 g. of calcium chloride per unit. No difficulties have been encountered on this regimen. (*Jennings, E. R., and others: Citrate Toxicity and the Use of Anticoagulant Acid-Citrate Dextrose Blood for Extracorporeal Circulation, Surg. Gynec. Obstet. 120: 997 (May) 1965.*)