ARRHYTHMIAS AND RESPIRATORY FAILURE Seventy patients with acute respiratory failure superimposed on chronic obstructive pulmonary disease were carefully monitored for arrhythmias. Fifty-nine of these patients developed some kind of dysrhythmia during their stay in the intensive care unit. Benign supraventricular arrhythmia developed in 26 patients. Eight out of the 26 died. Supraventricular tachycardia occurred in 13 patients, and six of them died. Twenty patients developed ventricular arrhythmias, and none of them survived. Only 11 patients had no arrhythmia, and all 11 survived. Hypoxia, hypercarbia, acidosis, hypokalemia, and increased blood levels of catecholamines and digitalis are thought to cause these arrhythmias. *(Brammell, H. L.: Arrhythmias in Acute Respiratory Failure Associated with Chronic Airway Obstruction. Heart and Lung 2(6):888, 1973.)*

Circulation

ENDOTOXEMIA Circulatory effects of *E. Coli* endotoxin were studied in awake, intact baboons. Early endotoxemia (2–4 hours) produced tachycardia, hypotension, hypocarbia, and base deficit. There was no significant change in cardiac output, peripheral resistance, or myocardial contractility (defined as the slope of the relationship between left ventricular dP/dt and developed isovolemic pressure at constant preload). *(Geocaris, T. V., and others: Effects of Gram Negative Endotoxemia on Myocardial Contractility in the Awake Primate. Ann Surg 178:715–720, 1973.)*

DIGITALIS GLYCOSIDES One measure of inotropic action is the shortening of electromechanical systole (QS_2I). Several glycosides were given intravenously to normal volunteers to determine the time course and magnitude of the inotropic response. The onset of shortening of QS_2I was exponential, with time constants of 5.8 minutes for ouabain, 7.2 minutes for deslanoside, 23 minutes for digoxin, and 56 minutes for digitoxin. Acetyl strophanthidin had a shorter time constant than ouabain, but the data were insufficient for statistical analysis. No significant differences in the maximum effects (ΔQS_2I/mole), calculated by an optimization technique, could be shown. *(Forester, W., and others: The Onset and Magnitude of the Contractile Response to Commonly Used Digitalis Glycosides in Normal Subjects. Circulation 49:517–521, 1974.)*

VOLUME EXPANDERS Short-term effects of infusion of volume expanders upon plasma volume and cardiac output were measured in eight healthy volunteers (10 per cent dextran 40) and in 36 critically ill patients (10 per cent dextran 40, 5 per cent dextran 40, dextran 70, plasma, and whole blood). Following control measurements, 500 ml of volume expander were infused over 60 minutes. Plasma volume (RISA, rate of radioactivity determined before and after infusion) and cardiac output (Stewart-Hamilton, cardio-green dye injected into right atrium or superior vena cava) were measured after each 100-ml infusion and 15, 30, and 60 minutes following the end of infusion. Infusion of 10 per cent dextran 40 produced greater increases in plasma volume (1,000 *vs.* 650 ml) and cardiac output (70 *vs.* 30 per cent) in critically ill patients than in normal volunteers. Whole blood, plasma and 5 per cent dextran 40 produced less increase in cardiac output than did 10 per cent dextran 40 and dextran 70; however, this may have been secondary to higher preinfusion cardiac outputs in the former groups. Effects of dextran infusion upon plasma volume appeared to be more prolonged when urinary output was depressed. Preinfusion cardiac output was not markedly depressed in any group. No statistical analysis is given, but the data do not appear to indicate any difference in short-term effect of the volume expanders tested upon either cardiac output or plasma volume. No information regarding blood gases or arterial or venous pressure is given. *(Shoemaker, W. C., and Monson, D. O.: The Effect of Whole Blood and Plasma Expanders on Volume–Flow Relationships in Critically Ill Patients. Surg Gynecol Obstet 137:453–457, 1973.)*

(Additional literature briefs may be found on pages 244, 311, 324, 351, and 370.)