higher hemoglobin levels. The only factor which consistently lowered the hemoglobin values of women was confinement to the house. Any reduction in hemoglobin in the elderly must be attributed to an abnormality of health or environment and not to age alone. (*Saunders, C. R., and Chalmers, D. G.: The Haemoglobin Level of Fit Elderly People, Lancet 2: 261 (Aug.) 1968.*)

SHOCK Blood volume was measured and central venous pressure was monitored in 16 patients in shock, to assess the relative merits of these two measurements for use in small hospitals where detailed studies cannot be made routinely. Blood volume greater than the predicted "normal volume" was often required to establish normal circulation in these patients. Central venous pressure did not always reveal alterations in blood volume, but was useful for assessing the ability of the right heart to accept a fluid load. (*Prout, W. G.: Relative Value of Central Venous Pressure Monitoring and Blood Volume Measurement in the Management of Shock, Lancet 1: 1108 (May) 1968.*) ABSTRACTER'S COMMENT: Blood volume values in this study may have been unduly high because the RISA used for measuring blood volume was injected into intravenous tubing rather than directly into a vein. Thus, theoretically, some RISA particles may have remained in the tubing.

SHOCK Portal blood flow, intestinal oxygen consumption and total oxygen consumption were studied in dogs subjected to hemorrhagic shock. The changes in control dogs were compared with those in a group in which metabolic acidosis was corrected with THAM. Portal blood flow and intestinal oxygen consumption decreased during shock in both groups. However, an initial marked increase in intestinal oxygen consumption occurred following retransfusion in the THAM-treated animals, but not in the control group, suggesting preservation of either the patency of the capillary circulation or cellular viability by THAM during shock. Total oxygen consumption was greater in the THAM-treated animals, both during and after shock. Isoproterenol caused significant increases in portal blood flow and intestinal oxygen consumption before the onset of shock. The response to isoproterenol was greatly reduced during the shock period and after retransfusion. Correction of the metabolic acidosis with THAM did not improve the response to isoproterenol. (*Myers, K. A., and others: Responses to Isoproterenol and THAM During Experimental Hemorrhagic Shock, Surgery 64: 653 (Sept.) 1968.*)

DEFIBRILLATION Sequential ventricular defibrillation was accomplished by monophasic trapezoid-shaped double pulses of low electrical energy. The uniformity of ventricular fibers needed to allow sinus rhythm to recur was achieved in two stages: the first pulse depolarized fibers excitable at the time of pulse passage; the second pulse depolarized fibers refractory to the first pulse. A critical separation of the two pulses was found to be 100 msec, an interval which allowed minimal voltages to be effective in defibrillation. The smaller electrical energy required for defibrillation with this method (average 2.4 Joules), compared with alternating current (50 to 400 Joules) or direct current (50 to 100 Joules), decreases the possibility of myocardial injuries incident to electrical defibrillation. (*Resnekov, L., and others: Ventricular Defibrillation by Monophasic Trapezoidal-shaped Double-pulse of Low Electrical Energy, Cardiov. Res. 31: 261 (July) 1968.*)

THYROID AND HEART RATE The effects of propranolol on the heart rates of patients with hyper- and hypothyroidism were compared in an attempt to delineate more precisely the relation between the sympathetic nervous system and the thyroid gland. Propranolol reduced heart rates in ten patients with hyperthyroidism by 17.5 beats/min and in five patients with hypothyroidism by 11.4 beats/min. Mean heart rates after propranolol were 74.2 beats/min in hyperthyroid patients and 47 beats/min in hypothyroid patients. This difference was due mainly to thyroxine acting directly on the heart and not to its potentiation of the action of catecholamines. (*McDevitt, D. G., and others: Role of the Thyroid in the Control of Heart Rate, Lancet 1: 998 (May) 1968.*)