comparable. PLV-2 therapy results in significantly higher (1 per cent level-chi sq.) survival. *Conclusion*: The findings that RES activity correlates as both a diagnostic and therapeutic parameter of host responses at a tissue level suggest the potential therapeutic value of exogenous stimulation of the RES in shock. (Study aided in part by U.S.P.H.S. Grant HE-09042.)

Dead Space During Controlled Ventilation. Myron B. Laver, M.D., Bertil Löf-STRÖM, M.D., HARTMUT HEITMANN, M.D., and HENNING PONTOPPIDAN, M.D., Anaesthesia Laboratory of the Harvard Medical School and the Respiratory Unit at the Massachusetts General Hospital, Boston. The present study was undertaken to define the relation between physiological shunting and dead space during controlled ventilation. Method: Data were collected from 23 anesthetized and paralyzed mongrel dogs (9-25 kg.) ventilated with pure oxygen. Physiological dead space in the presence of physiological shunting was calculated from the Bohr equation, using a mean alveolar P<sub>CO2</sub> obtained from the in vivo CO<sub>2</sub> dissociation curve and solution of the shunt equation with the CO<sub>2</sub> content data. Various degrees of venous admixture were produced by suction of air from the airway. Measurements were made after the animals had been maintained on constant volume ventilation at a particular tidal volume, for a period of 30 minutes. Changes in the state of expansion of the lungs were produced by either increasing the tidal volume or by repeating the deflation maneuver. The functional residual capacity was determined with a whole body plethysmograph. The cardiac output was calculated by the Fick principle using the CO2 data. There was a consistently small arterial-alveolar PCO. difference ranging from zero to 2.5 mm. of mercury when the Q<sub>S</sub>/Q<sub>T</sub> fraction was less than 10 per cent of the cardiac output. The P<sub>CO2</sub> gradient increased significantly (P < 0.01) to a maximum value of 10 mm. of mercury as the  $Q_s/Q_T$ rose to 60 per cent of the cardiac output. The calculated regression equation for the a-AD $_{
m CO_2}$ on  $Q_S/Q_T$  was y = 0.72 + 0.08x. Results: The physiological dead space, determined either with the Bohr or Enghoff equation, showed no change over a range of tidal ventilation that

resulted in venous admixture which varied from 10 to 60 per cent of the cardiac output. Although the absolute value of the physiological dead space (Bohr):  $23.6 \pm 9.9$  ml./kg. body weight was insignificantly higher with a low  $Q_s/Q_T \times 100 \ (7.5 \pm 1.7 \ per \ cent)$  than at higher degrees of venous admixture [Q<sub>s</sub>/  $Q_T \times 100 = 14.3 \pm 2.4$  per cent and physiological dead space (Bohr) =  $9.4 \pm 3.7$  ml./kg. body weight] there was no significant alteration in the dead space to tidal volume ratio  $(V_D/V_T)$ . There were no significant changes in the cardiac index, functional residual capacity, and CO2 output as the tidal volume varied from  $8.5 \pm 0.72$  to  $52.3 \pm 8.54$  ml./kg. body weight. The large tidal volumes were associated with the lowest shunts  $(Q_s/Q_T \times 100)$ and the highest arterial oxygen tensions (Pa<sub>O2</sub>). Use of the term dead space does not seem appropriate when describing the effects of large tidal volumes in the normal lung. Changes in arterial to alveolar carbon dioxide tension do eccur, but there is no evidence to suggest a reduction in the effectiveness of ventilation. The  $Q_{s}/Q_{T} \times 100$  was maintained below 10 per cent of cardiac output with tidal volumes above  $12.8 \pm 2.95$  ml./kg. body weight. Conclusion: Clearly, during controlled ventilation large tidal volumes are required in order to maintain maximal oxygenation. (Supported by NIH grant number HE 09340-01.)

Observations on Fetal Electrocardiographic Responses to Hemodynamic Influences. Elia Lipton, M.B., Ch.B., Francis W. SENNOTT, M.D., and BERNARD BATT, M.D., St. Margaret's Hospital, Boston. This study was prompted by clinical experiences involving possible influences on fetal heart rate of maternal hypotension and/or maternally administered vasopressors. Methods: The effects on the fetal heart rate of intravenously injected single doses of methoxamine (4 mg.), phenylephrine (washings) and mephentermine (15 mg.) were studied in both normotensive and hypotensive parturients. Fetal heart rates and patterns were continuously monitored with a Telemedics RKG 500 remote recorder system. Improved noise-free tracings were obtained with two abdominally applied German silver electrodes. The patients under observation were selected entirely from mothers in the