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 LÖFSTRÖM, 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_{CO_2} obtained from the *in vivo* CO_2 dissociation curve and solution of the shunt equation with the CO_2 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 CO_2 data. There was a consistently small arterial-alveolar P_{CO_2} difference ranging from zero to 2.5 mm. of mercury when the Q_S/Q_T fraction was less than 10 per cent of the cardiac output. The P_{CO_2} 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_{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 ± 9.9 ml./kg. body weight was insignificantly higher with a low $Q_S/Q_T \times 100$ (7.5 ± 1.7 per cent) than at higher degrees of venous admixture [$Q_S/Q_T \times 100 = 14.3 \pm 2.4$ 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 CO_2 output as the tidal volume varied from 8.5 ± 0.72 to 52.3 ± 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_{O_2}). 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 occur, 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 ± 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

prodromal stage of labor (normotensive group) or from parturients about to undergo either elective cesarean section under spinal anesthesia (hypotensive group) or a Shirodkar procedure for incompetent cervical os (early fetal development). All pregnancies were clinically normal and all premedication was withheld. *Results:* Varying stages of fetal development between the twenty-first and fortieth weeks of gestation were encountered. Thus far 26 patients have been studied, consisting of a control group of 10 nonanesthetized patients and 16 receiving spinal anesthesia with consequent hypotension for periods of 2 to 33 minutes. Blood pressure levels ranged between 90/50 and 40/20 mm. of mercury. Prior to the injection of the vasopressors, moderate fetal tachycardia (7–10.5 per cent) was actually observed in 7 out of the 16 hypotensive cases as compared to only one instance of fetal bradycardia (supine hypotension syndrome). With methoxamine 11 of 13 developed fetal bradycardia; with phenylephrine 5 of 9; and with mephentermine 0 of 4. There was no correlation between the incidence of bradycardia and the normotensive or hypotensive status of the mother. In other respects the responses of the two groups were the same, except with methoxamine, where fetal bradycardia in the spinal patient was of later onset (4–9 minutes) than the normotensive group (1–2½ minutes) and of shorter duration (1–5½ minutes as compared to 6–14 minutes). In two Shirodkar procedures selected specifically for early fetal development (21 and 23 weeks, respectively), rapid and prolonged slowing responses were readily elicited with methoxamine. At delivery all babies but one demonstrated no evidence of adverse effects from the previously administered pressor drugs. The exception occurred with phenylephrine in the hypotensive series where a profound and persistent (23 minutes) fetal bradycardia ensued. Hysterotomy revealed placental separation and the neonatal course was characterized by severe depression necessitating vigorous resuscitation. *Conclusion:* The anticipated increased fetal bradycardia from the combined effects of vasopressor drugs and the reduced uterine blood flow with spinal anesthesia was not apparent. On the contrary the effects on the fetus in the spinal anesthesia group were less sustained than in the control group. It appears that certain (methoxamine and phenylephrine) but not all (mephentermine) maternally administered vasopressors produce fetal bradycardia at all stages of fetal development. This may be a manifestation of direct effects of the vasopressors on a responsive fetal circulation.

Corticosteroids in Hypovolemic Shock. PHILLIP S. MARCUS, M.D., JOHN C. SHEEHAN, M.D., and FRANCIS L. COMUNALE, M.D., *Department of Anesthesiology, Boston City Hospital, Boston.* Emphasis on the microcirculation has directed much attention toward the use of vasodilators in the treatment of hemorrhagic and endotoxic shock. The two most common types of drugs used have been the adrenergic blocking drugs and the corticosteroids. The purpose of this study was to explore the possible benefits of corticosteroids in a clinically controlled study. *Method:* A double blind randomized study was performed to compare two groups of 48 surgical patients each. Patients selected had one or more of the following: hypotension, tachycardia, decreased total blood volume, a history of recent massive blood loss or multiple preoperative blood transfusions. These patients were given one dose of a coded drug in an ampule, intravenously, immediately before induction of anesthesia. The treated group received 125 mg. of methylprednisolone (Solu-Medrol) while the control group received a placebo. Operation, blood and fluid replacement, and postoperative care were in the manner indicated by the patients' disease and clinical state. *Results:* Homogeneity of the two groups was found to exist as to age, anesthetic agents for induction and maintenance, operative procedure and mortality. Although the overall survival rate in both groups was identical, a breakdown of the deaths occurring during the first twenty-four hours following operation showed a statistically significant decrease in the treated group. The results of this preliminary study illustrated a greater survival rate for the treated group during the first twenty-four hours.

Conclusion: The survival rate of surgical patients in hypovolemic shock during the immediate postoperative period can be significantly increased by the use of a single dose of