and position leading to the animal's death. The second animal was hypoventilated and apparently received too large a concentration of methoxyflurane. Both conditions were successfully corrected. The longest survival was 24 hours. This animal responded well in the postoperative period and was neurologically intact and allowed to breathe spontaneously postoperatively for two hours. Death of this animal was due to a short circuit in the power supply. *Conclusion:* This experiment shed much light upon the problems one may encounter in the placement of a cardiac prosthesis with pulsatile flow characteristics. After these problems are overcome, such an experimental preparation will permit the anesthesiologist to investigate the effects of anesthetic agents and drugs on cardiovascular dynamics under conditions of constant cardiac output and rate.

Deadspace Requirement for Normal Alveolar Ventilation During Artificial Respiration in Man. KUNIO SUWA, M.D., BENNIE GEFFIN, M.D., and HENNING PONTOPPIDAN, M.D., *Anesthesia Laboratory of the Harvard Medical School and the Respiratory Unit at the Massachusetts General Hospital, Boston.* During the management of patients receiving prolonged artificial ventilation, the need to overcome the sensation of dyspnea, and the tendency towards airspace collapse, frequently require the use of large tidal volumes. Patients usually resist a reduction in respiratory frequency of a degree sufficient to reduce the alveolar ventilation to normal. This results in both weaning difficulties and an undesirable and unnecessary respiratory alkalosis with compensatory changes in renal function and fluid balance. A normal Pa_{CO_2} can be achieved, even with a large minute ventilation, if mechanical deadspace of a suitable size is added. A quantitative analysis of this problem has not been done previously and therefore the size of the additional mechanical deadspace required to raise the Pa_{CO_2} to a normal level has not been known. A theoretical analysis of the relationship between anatomical, alveolar and physiological deadspaces in a two compartment lung model showed that the size of the mechanical deadspace (V_{DM}), necessary to raise the Pa_{CO_2} by certain amount, can be calculated from the following formula:

$$V_{DM} = \frac{Pa2_{CO_2} - Pa1_{CO_2}}{Pa2_{CO_2} - Pa1_{CO_2} + Pa1_{CO_2}} (V_T - V_{Dan}),$$

where V_T is the tidal volume, V_{Dan} is the anatomical deadspace of the patient plus that of the ventilator, and Pa_{CO_2} is the P_{CO_2} of the alveoolar component of the expired gas. Subscripts 1 and 2 refer to the conditions before and after V_{DM} was added. *Experimental Method:* Data were collected from 6 patients placed on constant volume, time-cycled ventilators. After obtaining the control values for CO_2 production, tidal volume, respiratory frequency and Pa_{CO_2} mechanical deadspaces of various sizes were added. Tidal volume, frequency and CO_2 production were constant. The observed Pa_{CO_2} was compared to that calculated from the equation. The anatomical deadspace was estimated from body weight. Pa_{CO_2} was calculated from the Bohr equation. *Results:* The observed Pa_{CO_2} agreed to within 2 mm. of mercury with that calculated in nineteen out of twenty determinations, confirming the validity of the equation. *Nomogram:* In order to make this information easily available clinically, a nomogram was constructed on the basis of a modification of equation 1, giving $Pa2_{CO_2}$ the value of 40 mm. of mercury. V_{DM} required to raise Pa_{CO_2} to 40 mm. of mercury can be obtained by measuring tidal volume, body weight and Pa_{CO_2} without the mechanical deadspace. A minor modification of the nomogram has been incorporated for patients with lung diseases. *Conclusion:* The change in Pa_{CO_2} produced by an added mechanical deadspace was analyzed both theoretically and experimentally. A nomogram has been designed to predict mechanical deadspace requirements for patients receiving artificial ventilation in order to prevent respiratory alkalosis.

Comparison of the Effects of Some Inhalation Anesthetic Agents on Forearm Blood Flow. JAMES S. TAYLOR, M.D., and J. S. DENSON, M.D., *Los Angeles County General Hospital and University of Southern California School of Medicine, Los Angeles.* In a preliminary attempt to define the effects of cyclopropane and halothane on the forearm blood flow, 15 clinically healthy unpremedicated