cent, $P < 0.05$), suggesting greater intra-pulmonary shunting and physiological dead space. Despite an increased correction of metabolic acidosis, this resulted in a consistently lower post-bypass arterial *p*H in the animals given Arfonad. The quantitative effects of this acidosis on cardiac index, peripheral resistance and blood flow has not been delineated. The increased efficiency of the lower cardiac index in perfusing tissue beds resulted in a significantly greater ($P < 0.001$) A-V_{O_2} difference in the treated animals. *Summary:* These experiments demonstrate the value of both monitoring oxygen consumption as a parameter of tissue perfusion, and active vasodilatation to aid this perfusion. The changes in pulmonary ventilation-perfusion ratios in the animals given Arfonad necessitate further evaluation. (Supported by U. S. Army Contract DA-49-193-MD-2160.)

Influence of Tidal Exchange During IPPB on Pulmonary Mechanics and Blood Gases in Anesthetized Humans. MARTIN I. GOLD, M.D., and MARTIN HELRICH, M.D., *Department of Anesthesiology, University of Maryland, Hospital and School of Medicine, Baltimore, Maryland.* The purpose of this study was to relate degree of change in blood gases to degree of increase in tidal and minute volume (V_T and V_E) and to correlate gas and volume parameters to changes in pulmonary mechanics. *Methods:* Esophageal balloon and pneumotachographic techniques were used to measure pulmonary compliance (C_L) and resistance (R_L) in 5 supine, anesthetized preoperative patients. Arterial gases were measured simultaneously during awake inhalation of 50 per cent O_2 in N_2 after denitrogenation and then 30 per cent O_2 in N_2O—halothane after intubation. Anesthetic depth was maintained at a steady level defined by venous and arterial halothane levels measured chromatographically. A pressure regulated ventilator at a rate of 20 per minute increased V_T stepwise during 5 minute periods from 250 through 500, 750 and 1,000 ml. Each IPPB period was interrupted by 10 minutes of spontaneous respiration (S.R.). There were, therefore, 8 series of data, 4 during S.R., each followed by one of 4 periods of IPPB. *Results:* During IPPB the P_{CO_2} rose during lower V_T's and fell during the 750 and 1,000 ml. trials. P_{O_2} was decreased during the 250 and 500 ml. V_T, but then rose. During the 1,000 ml. trial it attained the awake, control level ($V_T = 350$ ml.). During S.R. P_{CO_2} was increased, while P_{O_2} was depressed after each period of IPPB. The P_{O_2} at lower V_T's and V_E's during S.R. prior to the 250 and 500 ml. IPPB trials was higher than during the trial. The R_L during IPPB was consistently above that during S.R. The C_L was low during small V_T's but rose above the S.R. level at higher V_T's. Changes in V_T and C_L were positively correlated. During S.R. the C_L did not fall but remained at the control level, each period of IPPB possibly acting as a buffering "sigh." *Discussion and Conclusions:* With increasing V_T and V_E during IPPB, P_{CO_2} fell and P_{O_2} rose. However, 750 and 1,000 ml. V_T's elevated P_{O_2} to the awake, spontaneously breathing level, where V_T averaged only 350 ml. On the other hand, P_{CO_2} became succeedingly depressed as V_T increased stepwise. During IPPB, R_L consistently increased. C_L increased only as V_T passed 500 ml., this increase paralleling the V_T increase. Correlation between changes in pulmonary mechanics and blood gases was demonstrated. At low IPPB, V_T, the decreased C_L corresponded to a depressed P_{O_2} and increased P_{CO_2} while at high V_T the opposite occurred. However, this correlation was nonexistent during S.R. when each level of P_{O_2} after IPPB showed a stepwise decline, while C_L increased. Such changes during IPPB are interpreted as a departure from normal pulmonary physiology. In order to oxygenate comparatively, greater transpulmonary pressures and higher V_T's were necessary during IPPB than during S.R. At higher V_T the C_L increased indicating a more efficient pressure volume relationship than during lower V_T with its decreased C_L. However, R_L during IPPB at all V_T's was consistently elevated. These changes in mechanics and blood gases during IPPB may be related to changes in distribution and the development of increased physiologic shunting. In unfit patients and in those ventilated with mixtures of oxygen approaching those of air, abnormally low P_{O_2} levels may occur.