

$\pm 1.2$  per cent). Oxygen consumption and cardiac output changed proportionately at each phase of measurement and, therefore, arteriovenous oxygen content remained in the normal range (3.5–5.5 ml/100 ml). This was confirmed by direct measurement of arterial and mixed venous oxygen contents. *Summary:* The rapid onset, offset and lack of progression of the increased venous admixture observed in this study suggested a pharmacologic action of the anesthetic agents on the pulmonary circulation rather than progressive atelectasis. Furthermore, the increased  $Q_v/Q_t$  found during the early postoperative period was sufficient to account for much of the postoperative hypoxemia that has been demonstrated in similar patients breathing air after extremity surgery. (Supported in part by USPHS Grants 5-T01-GM-215-10, 1-P01-GM-15430-01 and 5-P01-GM-09070-06.)

**Cardiovascular Effects of Halothane and Halothane-Nitrous Oxide Anesthesia during Controlled Ventilation.** W. E. MARTIN, M.D., F. G. FREUND, M.D., T. F. HORNBEIN, M.D., and J. J. BONICA, M.D., *Department of Anesthesiology, University of Washington School of Medicine, Seattle, Wash.* *Methods:* Eight healthy unmedicated male volunteers 21 and 28 years old were studied. Arterial and venous catheters were placed under local anesthesia, the subjects rested for 30 minutes and control measurements obtained. Anesthesia was induced with halothane and nitrous oxide, the trachea intubated under topical anesthesia, and respiration controlled in a constant pattern thereafter. Following one hour of constant 1 per cent end-tidal halothane anesthesia, the level was shifted in a random sequence to 0.3 per cent halothane in 70 per cent nitrous oxide, 0.8 per cent halothane in 100 per cent oxygen, and 0.8 per cent halothane in 70 per cent nitrous oxide; and measurements were made at  $P_{aCO_2}$  values of approximately 20, 40, and 60 torr. *Results:* Three primary observations emerge. First, as the  $CO_2$  rises from 20 to 40 to 60 torr, cardiac performance improves, since minute work, stroke work, mean arterial pressure, cardiac output, stroke volume, central venous pressure, and the first derivative of arterial pressure all increase; heart rate is rela-

tively stable, and total peripheral resistance decreases. Second, cardiac output is not dependent on the anesthetic level, since at any given  $P_{aCO_2}$  cardiac output is the same with all three anesthetic levels. Third, the data support but do not prove the concept that nitrous oxide has a sympathetic stimulating effect, since the addition of 70 per cent nitrous oxide to 0.8 per cent halothane results in an increase in total peripheral resistance and venous pressure and a greater rate of rise of the first derivative of arterial pressure as  $P_{aCO_2}$  increases. *Summary:* The increased cardiac output resulting from a higher  $P_{aCO_2}$  coupled with a lower total peripheral resistance and a higher mean arterial pressure suggests that a modest increase in  $P_{aCO_2}$  (up to 60 torr) might be a desirable clinical goal during halothane or halothane-nitrous oxide anesthesia.

**Mycardial Metabolism and Hemodynamics in the Halothane-depressed Canine Heart.** R. G. MERIN, M.D., *University of Rochester School of Medicine and Dentistry, Rochester, N. Y.* Limited information concerning the mechanism and significance of the myocardial depression produced by inhalation anesthetics is available. This study has looked at one phase of energy kinetics in the heart by assessing the effect of halothane on the manner in which the dog heart handles its fuels and oxygen. *Methods:* Fasting, nonmedicated dogs were intubated and artificially ventilated with halothane and oxygen. With fluoroscopic guidance catheters were placed in the femoral artery, left ventricle, right atrium and coronary sinus. Temperature, arterial blood gases and blood volume were maintained constant. A very low halothane concentration was used as the control (0.6 per cent mixed expired) and 1.6 per cent served as the test, each animal being his own control. At each concentration, heart and arterial pressures, left ventricular  $dp/dt$ , ECG, cardiac output, myocardial blood flow (MBF) (by a radioisotope technique) and arterial and coronary venous levels of  $O_2$ , glucose, nonesterified fatty acids (NEFA), lactate and pyruvate were determined. Myocardial uptake was calculated from MBF and coronary A-V differences. *Results:* Significant myocardial depression was