Arterial Blood Gases in Older Patients during N₂O-O₂ Anesthesia with Unassisted Respiration and with Controlled Hyperventilation. H. H. Hunt, Jr., M.D., T. D. Graff, M.D., and D. W. Benson, M.D., Department of Anesthesiology, Johns Hopkins University School of Medicine, Baltimore, Md. Hypoxia has been reported as frequent during anesthesia when less than 50 per cent oxygen was inspired. This prompted a study of blood gases in ten patients more than 60 years of age, undergoing prostatectomy in the lithotomy position. Methods: All patients were studied twice preoperatively as well as during anesthesia and within 90 minutes after operation. Blood samples were drawn during breathing of 20-21 per cent oxygen and again during breathing of 99-100 per cent oxygen to estimate true shunt. Samples were analyzed for Pₐ, pH, P CO₂, standard bicarbonate, and hematocrit. Anesthesia incorporated endotracheal intubation, semiclosed-circle system, and a gas inflow of 8 1./min. N₂O, 2 1./min. O₂ for 45 minutes. A blood sample was drawn and 99-100 per cent oxygen given with up to 1 per cent halothane or with thiocyanate for an additional 45 minutes before drawing a second sample. Five patients were allowed to breathe spontaneously, and the remaining patients were ventilated at two to three times their resting ventilation using a mechanical ventilator. Results: Average arterial Pₐ decreased 11 mm. during anesthesia with spontaneous respiration. Co-incident hypercarpnia occurred with an average increase in P CO₂ of 11 mm. and calculated oxygen saturations decreased from an average 96 per cent to 89 per cent. In the ventilated group, average Pₐ increased 11 mm., average P CO₂ decreased 14 mm., and oxygen saturation increased 2 per cent. Standard bicarbonate fell an average of 3 mEq/l. in patients with spontaneous respiration, and was unchanged in the controlled-ventilation patients. "True shunt," calculated with an assumed 4.5 vol. per cent arteriovenous oxygen difference during oxygen breathing, revealed an average increase of 7 per cent in the patients with spontaneous ventilation and a 1 per cent increase in those mechanically ventilated. In the spontaneous respiration group, standard bicarbonate increased an average of 1 mEq/l. between anesthesia and recovery, but in the mechanically-ventilated group there was a decrease in bicarbonate averaging 2 mEq/l. between anesthesia and recovery. All studies returned to approximately control values with this exception. Conclusions: The studies to date confirm the previous suggestions that hypoxia was likely during anesthesia with 20 per cent inspired oxygen and unassisted respiration. Mechanical hyperventilation with adequate gas inflows was associated with normal blood gas values. The findings of Prs-Roberts and Kelman (Brit. J. Anaesth. 38: 681, 1966) regarding the positive correlation of cardiac output and arterial oxygen tension during anesthesia suggest that the actual increase in true shunt in our patients during anesthesia with spontaneous respiration was greater than that calculated using an assumed 4.5 vol. per cent arteriovenous O₂ difference. Conversely, it seems likely that true shunt probably decreased during anesthesia with controlled hyperventilation. Simultaneous hypoventilation and increase in true shunt in the subjects with spontaneous respiration appeared adequate to explain the degree of hypoxemia found. This suggests that primary ventilation perfusion alteration was not severe. Most changes in standard bicarbonate were consistent with in vivo versus in vitro CO₂ dissociation curve differences. Hypoxia may have contributed to the more marked drop in standard bicarbonate noted during anesthesia with spontaneous ventilation.