anesthesia. He sampled venous blood from the back of the hand without special aids to vasodilation and concluded that this method was not subject to the gross errors occasionally obtained by estimating Pa_{CO_2} from end-tidal samples or rebreathed gas samples. Neither Harrison nor Cooper studied arteriovenous difference for *p*H or oxygen.

We conclude that during general anesthesia peripheral venous P_{CO_2} and *p*H approach values in arterial blood. Application of tourniquet pressures of as much as 90 torr did not alter this general result. Although the oxygen partial pressure in venous blood was significantly less than that in arterial blood, the difference in saturation was small. Venous blood from the hand, therefore, may provide a reasonable estimate of the presence or absence of hypoxia.

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CNS Function

THIOPENTAL AND INTRACRANIAL PRESSURE. Six patients, three with elevated intracranial pressures (ICP) and three with presumed normal ICP's, were studied prior to and during operations on the head. Central venous pressure, arterial pressure (AP), and lateral ventricular (intracranial) pressure were continuously monitored via appropriate catheters placed under local anesthesia before the start of the procedures. Induction of anesthesia with thiopental and succinylcholine caused no significant change in AP or ICP from awake control values. However, laryngoscopy with tracheal intubation, placement of the head clamp, and passage of a saw guide through the burr holes produced marked rises in ICP, particularly in those patients with an elevated preoperative ICP. During controlled ventilation with Pa_{CO_2} maintained at 25–30 torr, 2 per cent inspired halothane produced a steady increase in ICP and a decrease in AP. Thiopental in 100-mg intravenous doses seemingly reversed the rises in ICP without affecting AP. The authors suggest that thiopental may offer a means of attenuating acute ICP increments during anesthetic and operative manipulation. *(Shapiro, H. M., and others: Acute Intracranial Hypertension during Anesthetic Induction. Partial Control with Thiopental. Europ. Neurol. 8: 118–121, 1972.)*

Respiration

BED REST AND BLOOD-GAS EXCHANGE Pulmonary function was studied in seven healthy male volunteers maintained at supine bed rest for two separate 48-hour periods, and included sequential monitoring of arterial blood gases, $A\text{-}aD_{O_2}$, V_A/Q and Q_S/Q_T. There was no significant alteration in mean *p*H, Pa_{O_2}, or Pa_{CO_2} during the study. The initial and 48-hour mean V_A/Q ratios were 0.51 ± 0.15 and 0.69 ± 0.14, respectively ($P > .005$). This was attributed to high initial cardiac output (mean 6.91 ± 1.45 l/min), which decreased to 5.19 ± 1.07 l/min after 48 hours. There was no significant alteration of alveolar ventilation during the experiment. On breathing ambient air, mean $A\text{-}aD_{O_2}$ was 9.9 ± 6.5 mm Hg at the beginning of the experiment and 10.4 ± 6.8 mm Hg after 48 hours. Initial and 48-hour mean values of $A\text{-}aD_{O_2}$ during breathing of oxygen were 78 ± 35 mm Hg and 69 ± 24 mm Hg, respectively. Mean Q_S/Q_T had not changed significantly at the end of 48 hours, and chest x-rays taken before and after the experiment were normal. This study demonstrates that gravitational redistribution of pulmonary perfusion and ventilation during supine bedrest for 48 hours does not cause a significant alteration in blood-gas exchange in healthy subjects. *(Trimble, C., and others: The Effect of Supine Bedrest upon Alveolar–Arterial Oxygen Gradients and Intrapulmonary Shunting in Normal Man. J. Thorac. Cardiovasc. Surg. 63: 873–879, 1972.)*