Abstracts

Work in Progress

John Adriani, M.D., Editor

Effects of Cyclopropane Anesthesia on Cerebral Blood Flow and Carbohydrate Metabolism of Man. S. CRAIGHEAD ALEXANDER. M.D., F. M. JAMES, M.D., E. T. COLTON, M.D., H. R. GLEATON, M.D., and HARRY WOLLMAN, M.D., School of Medicine, Universitu of Pennsulvania, Philadelphia, Penna. We have investigated the effects on cerebral circulation and metabolism of four levels of inspired cyclopropane during normocarbia. In addition the response of the cerebral circulation to changes in arterial Bco. (Paco.) over the range of 20 to 50 torr was studied while the inhaled cyclopropane concentration was Method: Cerebral circulatory held constant. and metabolic measurements were made in 18 normal adult male volunteers, while the subiects were conscious or during cyclopropane An endotracheal tube was inanesthesia. serted during anesthesia and pulmonary ventilation was controlled with a Bird respirator in a non-rebreathing circuit. End-tidal Pco: was monitored with an infrared CO2 analyzer and Pco2 was controlled during anesthesia by varying ventilation. d-Tubocurarine was administered intravenously as necessary to facilitate the control of ventilation. Cerebral blood flow (CBF) was measured by an inert gas uptake technique utilizing inhaled KR-85. Cerebral utilization or production of oxygen, glucose, lactate, and pyruvate was calculated from the CBF and the appropriate arteriovenous difference. The arteriovenous difference for cyclopropane was less than 0.1 volume per cent at the time measurements were made, indicating that the brain was in equilibrium with arterial blood. Studies were conducted in twelve men during the inhalation of 0, 5, 13, 20, and 37 per cent cyclopropane in oxygen while arterial PCO2 (PaCO2) was maintained at normal levels. Each subject was studied in the conscious state and while anesthetized at two different depths of cyclopropane anesthesia. Results: Cerebral blood

flow declined 42 per cent during the inhalation of 5 per cent cyclopropane and 24 per cent during the inhalation of 13 per cent cyclopropane. In contrast, the inhalation of 20 and 37 per cent cyclopropane caused 35 and 86 per cent increases, respectively, in cerebral blood flow. Thus, cerebral vessels appeared to constrict during light cyclopropane anesthesia but dilated when deeper levels were achieved.

Cyclopropane had a diphasic action on brain energy utilization as well. Cerebral oxygen consumption was reduced to approximately 70 per cent of normal during the inhalation of 5 per cent cyclopropane and there was no further metabolic depression during the inhalation of 13 and 37 per cent cyclopropane. In contrast, brain oxygen consumption was not significantly different from normal during the inhalation of 21 per cent cyclopropane. No alterations in the pathways of brain carbohydrate metabolism were noted.

A second group of six volunteers was anesthetized with 21 per cent cyclopropane in oxygen and measurements were made at Pa_{CO_2} levels of approximately 20, 35, and 50 torr. CBF increased as Pa_{CO_2} was elevated and a regression line having the equation:

$$CBF = 1.95 Pa_{CO_7} - 9.2$$

was calculated to fit the data. The slope of this line is significantly greater than that obtained in this laboratory during the inhalation of either 1.2 per cent halothane or 70 per cent nitrous oxide. Summary: These studies demonstrate biphasic circulatory and metabolic responses to increasing concentrations of cyclopropane and increased sensitivity of brain blood vessels to alterations of Paco₂ during the inhalation of 21 per cent cyclopropane. Cerebral vessels appear to constrict during light cyclopropane anesthesia. The biphasic action occurs with energy utilization as well.