per cent–k value 0.347–0.180; phenylephrine HCl, a drop of 54.3 per cent–k value 0.383–0.175 and levarterenol, a drop of 61.8 per cent –k value 0.196–0.113. The cardiac output was moderately reduced. In this preliminary report, the effect of vasopressors on the blood flow through the liver was measured in normotensive adult patients. In all instances, there was a relative reduction in rate of blood flow with increased vascular resistance. This effect was pronounced with methoxamine HCl, phenylephrine HCl, and levarterenol. Nevertheless, the effect may be different in conditions of peripheral vascular dilation and hypotension of neurogenic, cardiogenic or hypovolemic origin. It is interesting to note that with epinephrine and ephedrine, the rate of blood flow values were close to resting level values. This may be due to the fact that the cardiac output was markedly increased which compensated, in part, for the increased vascular resistance.

Central Nervous System Effects of Hyperventilation During Anesthesia. GERALD D. ALLEN, M.B., and LUCIEN E. MORRIS, M.D., *Department of Anesthesiology, University of Washington School of Medicine, Seattle, Washington.* An attempt has been made to assess the effect upon the cerebral cortex of hyperventilation under anesthesia, utilizing the critical flicker fusion value as an index of cerebral function. The Evipal test as described by Berg was used to detect latent cerebral damage (*Berg, O.: Acta Psychiat. Neurol., Suppl. 58: 1949*). The instrument used for determination of the critical flicker fusion value was the Strobotac, modified for use under clinical conditions. Confirmation of the efficacy of hyperventilation was by means of Plesch alveolar CO_2 samples, and/or arterial carbon dioxide content and arterial *p*H. Positive Evipal critical flicker fusion tests were found in 17 cases, all of which were shown to have had hyperventilation of longer than two hours throughout anesthesia. The type of ventilation at operation in these cases was considered to be as follows: 1 assisted, 2 controlled, and 14 artificially ventilated. The degree of cerebral damage was not great as indicated by the depth of the depression together with its length and duration; the maximal depth being 7 flashes and length being 18 minutes whilst the longest duration noted was 3 days. The results in 51 satisfactorily completed cases indicate that hyperventilation under anesthesia caused measurable change in the critical flicker fusion value. These changes in critical flicker fusion value were correlated in this series as well as in other series with cerebral damage and cerebral anoxia.

Effect of Variations in Lung Volume and Alveolar Gas Composition on the Rate of Fall of Oxygen Saturation During Apnea. NORMAN A. BERGMAN, M.D., *Department of Anesthesiology, Veterans Administration Hospital, University of Utah, College of Medicine, Salt Lake City, Utah.* This study extends observations by other investigators which showed that when the respired gas is changed from air to oxygen the rate of arterial desaturation during a subsequent apnea is markedly slowed. Anesthetized, paralyzed dogs hyperventilated with room air through a nonrebreathing circuit were made apneic by disconnecting the endotracheal tube from the respirator at the end of an exhalation. Lung volume during apnea was varied by either leaving the tube open to atmospheric pressure or by rapidly inflating the lung with either 400, 800, or 1200 cc. of air using a 2,000 cc. "super syringe" and holding the lung at these respective volumes during the apnea. In some dogs lung volume was varied in the above manner both before and after thoracotomy. In other experiments the lung was held inflated with 400 cc. of either air, oxygen, or helium during apnea. Arterial oxygen saturation was measured with an oximeter on femoral arterial blood. Initial saturations were obtained during hyperventilation with air and the interval between the onset of apnea and the attainment of an oxygen saturation of 75 per cent was measured with a stopwatch. In 7 dogs the time required for saturation to fall from its initial value to 75 per cent was directly proportional to the volume of air in the lung during apnea at all volumes measured. As lung volume during apnea increased, time required for saturation to fall to 75 per cent increased in a linear manner. In 4 dogs this linear relationship was observed at the three lower volumes measured, but following infla-