here, muscle response to nerve stimulation was used as the criterion for relaxation. This criterion has the advantage of being little influenced by blood pH or $F_{CO_2}$, depth of anesthesia, or external stimulation. Method: One hundred adult patients receiving general anesthesia were included in this study. The majority of patients received nitrous oxide, oxygen and halothane anesthesia, but other kinds of nonflammable agents were used. Electrodes from a "Block-Aid" monitor were inserted subcutaneously over the ulnar nerve at the wrist. Supramaximal stimulation was applied at a rate of 34 per minute. A Grass force displacement transducer was secured in the hand, and the signal resulting from thumb adduction was recorded on a Sanborn recorder. The patients were divided into 7 groups in order to study varying doses and routes of administration of the drug. Commercially obtained succinylcholine was used. In most groups the concentration of drug was 20 mg./ml. In each case, the drug was administered as a single injection. The latent period was defined as the time from injection to the time of maximum twitch abolition. The duration of action was defined as onset of block until 50 per cent twitch recovery (T-50). After the twitch height reached a relative plateau, the patients were tested for post-tetanic facilitation. If this phenomenon was evident, it was assumed that some degree of dual block existed. Those patients exhibiting dual block were not used in the determinations of the duration of the block. Results: Four groups of 15 patients receiving intravenous succinylcholine, 0.5, 1.0, 2.0, and 4.0 mg./kg., had mean latent periods varying from 25 to 45 seconds. The mean durations of action (T-50) for the groups were, respectively, 5 minutes 50 seconds, 10 minutes, 13 minutes, and 17 minutes 10 seconds. Post-tetanic facilitation was seen in 2 patients receiving 2 mg./kg. and in 1 patient receiving 4 mg./kg. of the drug. Two groups of 15 patients receiving 1 and 2 mg./kg. of drug intramuscularly had mean latent periods of 4 minutes 25 seconds, and 4 minutes 10 seconds. Mean duration of action was 17 minutes 50 seconds and 27 minutes 10 seconds, respectively. Post-tetanic facilitation was seen in 6 patients receiving the higher dose. One group of 10 patients was given succinylcholine, 4 mg./kg., intra-
muscularly. The mean latent period was 1 minute 50 seconds. Seven of these patients developed post-tetanic facilitation. Because of the limited number of remaining cases, no assessment of duration was made. Comment: Partial paralysis from succinylcholine was determined to be of longer duration, when this technique was used, than when recovery from apnea was used as an end point. Although ventilation may commence early, the adequacy of early ventilation can be questioned. Certainly, respiratory reserve would be limited. The fact that the intramuscular route of administration caused a greater number of patients to develop dual block suggests that this is time dependent. This is further substantiated by other work showing that 3 mg./kg. of succinylcholine, when given by prolonged intravenous infusion, produces dual block in 100 per cent of patients. (Katz, R. L., Wolf, C. E., and Papper, E. M.: Anesthesiology 24: 784, 1963).

Hyperventilation, Brain Damage and Flicker. J. G. Whitwam, M.B., Ch.B., M.R.C.P., F.F.A.R.C.S., Robert B. Boettner, M.D., Anita P. Gilger, M.D., and Arthur S. Littell, Sc.D., Anesthesia and Ophthalmology Services, Department of Surgery and Division of Biometry, Western Reserve University, Cleveland, Ohio. Passive hyperventilation during operation has recently been condemned as an anesthetic technique because of alleged adverse neurological sequelae. Allen and Morris (Brit. J. Anaesth. 34: 298, 1962) detected latent cerebral damage by applying Berg's critical flicker fusion test (Acta Psychiat. (KBH) Suppl. 58, 1949) postoperatively to 24 patients who had been hyperventilated during operation. Because of the many variables present in measuring critical flicker fusion we undertook to study the effect of hyperventilation per se on critical flicker fusion in a carefully controlled environment. Method: Six health male volunteers between 22 and 26 years were passively hyperventilated by a Technician Huxley cuissar-type ventilator to 17-25 liters per minute for two hours. Critical flicker fusion was measured: (1) before hyperventilation, (2) after two hours of hyperventilation and (3) two and four minutes after one tenth of a hypnotic dose of hexobarbital. The same sub-
jects were studied two weeks later in the same manner with the period of hyperventilation replaced by two hours of rest, thus serving as their own controls. Results: The data showed no significant difference between the means of the initial critical flicker fusion after an interval of two weeks, indicating the reproducibility of the experimental situation. The data showed a highly significant fall in the critical flicker fusion two minutes after hexobarbital in the control group indicating consistence with Berg’s studies on normal subjects. The data did not show any significant changes as determined by differences of the means for critical flicker fusion obtained during two hours rest and two hours hyperventilation and no significant differences in the two groups following hexabarbital.

Anesthetic Level and Electrical Impedance of the Brain Stem. Robert L. Willenkin, M.D., Department of Anesthesiology, Yale University School of Medicine, New Haven, Connecticut. The mechanism of action of anesthetic agents is a subject for considerable speculation for years. The most recent statements, those of Pauling (Science 134: 15, 1961) and Miller (Proc. Nat. Acad. Sci. 47: 1515, 1961), propose that anesthetics produce their effects by making intracellular water more structured, interfering with movement of ions and thus decreasing conductivity through neural pathways. The purpose of this study was to determine whether there is, in fact, a change in conductivity associated with general anesthesia and to locate grossly the areas in which this occurs. Method: Cats were anesthetized with methoxyfluorane after a small induction dose of pentobarbital. Tracheotomy was performed and each animal was ventilated with a nonrebreathing, controlled-volume respirator. End-expired Pco2 and blood pressure were monitored. Silver electrodes were placed on the lateral surfaces of the brainstem from the level of the spinal accessory nerve to the level of the suprachiasmatic. The electrodes were connected to a General Radio Co. type 1605-A impedance bridge, the output of which was connected to a Grass polygraph. Measurements of magnitude and phase angle of impedance were made at a frequency of 100 kc./sec. Inspired gas concentrations of methoxyfluorane varied from 0.1 to 1.0 per cent. Results: The results are expressed in terms of conductance, susceptance and admittance because (1) the common model of cells and cell membranes is that of resistances and capacitances in parallel and (2) the quantitative relations between elements of parallel circuits are more meaningfully expressed in this manner. Conductance is the reciprocal of resistance, susceptance, the reciprocal of capacitive reactance, and admittance, the reciprocal of impedance. Admittance is the vector sum of conductance and susceptance as impedance is the vector sum of resistance and reactance. In other words, an increase in any of the three parameters is an increase in conductivity, a decrease is a decrease in conductivity. At the caudal end of the medulla there were no changes in these parameters associated with a change in anesthetic level. At the level of the vagus-nerve, changing the inspired gas concentration of methoxyfluorane from 0.1 to 0.6 per cent decreased conductance from 1570 to 1860 microhms, decreased admittance from 1,590 to 1,580 microhms but increased susceptance from 289 to 295 microhms. At the level just caudal to the inferior colliculus, increasing the concentration from 0.1 to 1.0 per cent decreased conductance from 1,600 to 1,550 microhms, decreased admittance from 1,620 to 1,570 microhms but increased susceptance from 245 to 251 microhms. With electrodes at a level between the superior and inferior colliculus, increasing concentrations from 0.1 to 0.9 per cent decreased conductance from 2,320 to 2,200 microhms, decreased admittance from 2,330 to 2,220 microhms but again increased susceptance from 259 to 314 microhms. Conclusions: During methoxyfluorane anesthesia there was a decrease in conductivity in that portion of the brain stem where the major portion of the reticular activating system is located. This decrease is due to a change in resistive and not to capacitive factors. The amount of decrease in conductivity is a function of the level of anesthesia. (Supported by USPHS Grant NB 05495 and the Josiah Macy Foundation.)

Disturbances of the Oxidative-Phosphorylation Mechanism as a Possible Etiological Factor in Sudden Unexplained Hyperthermia