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Variations in Visual Evoked Potentials under Anesthesia

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Even though the brain is the target organ of anesthetics, relatively little is known about brain function under anesthesia, and the means of monitoring it are meager. In contrast, abundant research on the heart and lungs, which are secondarily affected by anesthetics, has provided the basis for aggressive, invasive monitoring of circulation and ventilation.

The EEG and power spectrum analysis of the EEG have been used to monitor the effects of anesthetics, anesthetic concentration and the patient's response to surgical stimulation (Uhl, 1977), however, the EEG has not been a rewarding tool in these applications since it represents the overall activity of the brain and lacks specificity. Sensory evoked responses may provide a better index of brain function under anesthesia since the ultimate aim of anesthesia is to prevent CNS registration of sensory stimulation.

We have been using visual evoked responses (VERs) to monitor visual system function during neurosurgery and have noted unexplained changes under halothane anesthesia which had no specific relations to the surgical procedure. In view of the fact that qualitative changes in the VER have been reported with halothane anesthesia (Domino, 1967), the following systematic study of the effects of halothane concentration on the VER was performed.

Eight female patients between the ages of 23 and 57 years consented to be studied. Six were undergoing vaginal hysterectomies, one an abdominal hysterectomy, and one an orthopedic procedure on the hand. VERs were recorded between O_z and C_z to monoptic presentations of a visual stimulus. The visual stimulation was provided by an array of 10 red LEDs positioned in two rows on an opaque eyepatch. The eyepatch was placed over the closed eyelid and the LEDs were illuminated for 30 msec at a rate of 3/sec. The result was a very diffuse, low intensity red light flash to the retina.

VERs were obtained in an awake condition prior to medication and at varying concentrations of halothane in oxygen during surgery. All patients received thiopental induction, followed by anesthetic maintenance with halothane in oxygen, paralysis and endotracheal intubation. The alveolar halothane concentration, measured in end-tidal gas samples, was gradually increased to 1.13% (v/v), and surgery proceeded. Standard ventilation procedures were followed and esophageal temperature was maintained between 35 and 36°C.

VERs were obtained at halothane concentrations of 1.13, 0.90 and 0.75%. At least 30 min after induction were allowed before VER recording was begun, and each anesthetic

level was held constant for at least 10 min prior to VER recording. At least 4 VER waveforms were obtained at each level. When time permitted, each level was re-established and additional VERs were recorded.

The VERs obtained for one patient are shown in Fig. 1. The reproducibility of the data as well as the principal results are illustrated in the figure. With increasing alveolar halothane concentration, there was a progressive increase in the VER latency. The most reliable VER peak was the large positive peak with a latency of approximately 100 msec (P1). In the awake condition, the mean P1 latency was 113 msec (S.D. 10 msec), increasing to 134 msec (S.D. 8 msec) at a halothane concentration of 1.13% (a one-way ANOVA for repeated measures yielded, $F_{3,21} = 12.8$, $P < 0.005$).

No systematic changes in VER amplitude as a function of halothane concentration were noted.

In order to further correlate the VER latencies with anesthetic depth, the halothane concentrations were converted to *m*inimum *a*lveolar *c*oncentration (MAC) levels. At a MAC level of 1.0 (a halothane concentration of 0.76% (v/v) for patients over 30 and 0.85% for patients under 30), 50% of patients do not move in response to a skin incision, at a MAC level of 1.2, 95% of patients will not move, and a MAC level of 1.5 is considered to be deep anesthesia (Eger, 1977). Dose-response curves for P1 latency versus MAC level are shown for all subjects in Fig. 2. Linear correlation coefficients (ρ)
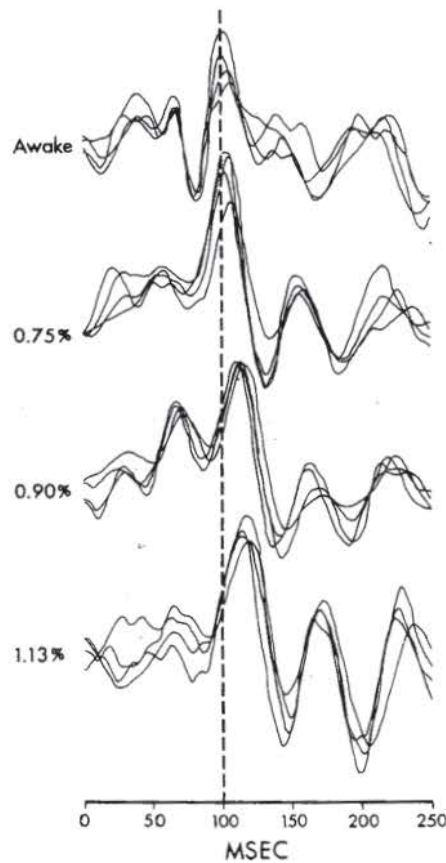

Fig. 1. VER waveforms for one subject at varying alveolar concentrations of halothane in oxygen.
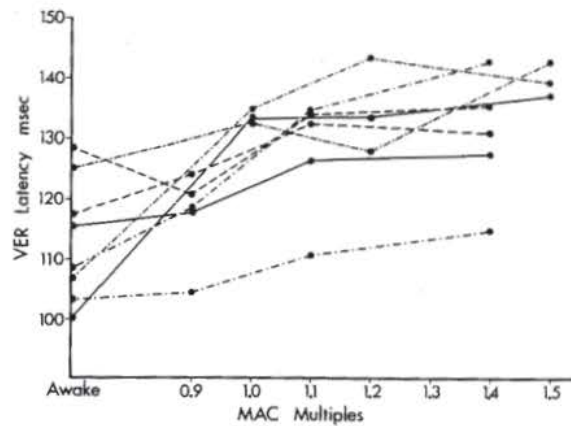
Fig. 2. Latencies of the P1 peak of the VER as a function of halothane concentration. Each point represents the mean latency over 4 replications for 8 subjects.

TABLE I

CORRELATIONS BETWEEN THE VER P1 LATENCY AND ANESTHETIC DEPTH (MAC) FOR INDIVIDUAL SUBJECTS

Subject	Correlation coefficient (ρ)
M.B.	0.925
J.K.	0.911
O.G.	0.953
J.D.	0.871
H.L.	0.767
S.H.	0.516
S.D.	0.836
D.H.	0.387
Mean	0.771

for the individual subjects ranged from 0.387 to 0.953, with a mean of 0.771, as shown in Table I.

Halothane anesthesia over time periods comparable to those studied here reportedly has no effect on pupil diameter and eyeball movement (Cullen et al., 1972). Control studies with awake subjects, however, were conducted to insure that artifactual changes in retinal sensitivity or direction of gaze did not account for the results. Upward and downward gaze had no influence on the VER waveform and neither did the time elapsed from placement of the eyepatch on the subject in a dimly lit room.

These data indicate that while VERs recorded under surgical concentrations of halothane are qualitatively similar to those recorded in an awake condition, variations in anesthetic concentration may significantly affect the P1 latency. Thus, when VERs are used to monitor sensory function during neurosurgical procedures, it is very important that anesthetic concentration be closely monitored. In view of the highly reproducible latency of the P1 peak normally obtained in a clinical diagnostic laboratory with

reversing checkerboard stimulation (cf., Halliday, 1978), latency shifts of the magnitude reported here (7–37 msec) may be interpreted mistakenly as due to surgical manipulation unless cognizance is taken of anesthetic depth, which may change substantially over the course of a long surgical procedure.

The close correlation found here for the P1 latency, a neurophysiologic variable, and MAC level, a behavioral index of anesthetic potency, further suggests that the VER may be used as a direct measure of the effectiveness of anesthesia in producing unconsciousness. The current procedures for monitoring anesthetic depth rely on secondary effects on respiration and circulation. Further studies will be necessary to optimize the procedures for such monitoring, as will studies of other sensory modalities and anesthetics.

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