Introduction. There have been interests in using electroencephalogram (EEG) to determine anesthetic depth. The recent power spectrum analysis of the EEG has facilitated accurate quantification. Nevertheless sensory evoked response might be a better index of anesthetic depth than the EEG since the suppression of responses to stimulation is characteristic of anesthesia. Uhl et al recorded the visual evoked response (VER) during halothane anesthesia and concluded it was useful. Although both the power spectrum analysis and the VER are effective, they will require expensive computerized instruments.

The eye existing near body surface could be the appropriate model to monitor the cerebral function or circulation. That is, the light-evoked electrical response in the retina or electroretinogram (ERG) might be valuable as an index of anesthetic depth. The typical gross ERG in the rabbit (Fig. 1) is composed of early receptor potential (ERP), a-wave and b-wave. The oscillatory potentials (OPs) are also the component of the ERG and superimposed on the b-wave. The a-wave is thought to be generated in a distal layer and the b-wave generated in Müller cells. The OPs reflect possible feed-back synaptic circuit initiated by amacrines in the retina where GABA and glycine act as neurotransmitters.

The present study was aimed to investigate the effect of volatile anesthetics on the ERG, particularly on the OPs.

Methods. Twelve rabbits were used. Under pentobarbital anesthesia, the animals were prepared with tracheostomy and femoral arterial cannulation. They were paralyzed with continuous infusion of succinylcholine and artificially ventilated throughout the experiment. The ERG was recorded between an active electrode mounted in a scleral contact lens and a reference electrode inserted in the forehead, using the oscilloscope equipped stroboscopic light (Heiwa electric Co. RX-3T, Tokyo, Japan). 2 hr after intravenous injection of pentobarbital, control ERG was obtained. Subsequently, either methoxyflurane, halothane or enflurane was inhaled in each rabbit. Inspired concentrations of anesthetics were given in the following sequence; 0.5, 1, 0.5 and 0 % for methoxyflurane, 1, 2, 1 and 0 % for halothane, or 1, 2, 3, 2, 1 and 0 % for enflurane. Each concentration was maintained for at least 30 min before the ERG recording. Increased concentrations of inhaled anesthetics lowered arterial pressure. In order to discriminate the effects of hypertension and anesthetics, induced hypotension with Na nitroprusside was performed in unanesthetized animals.

Results. Volatile anesthetics examined had no effect on both the a-wave and the b-wave. Methoxyflurane and halothane decreased the amplitude of the OPs concentration-dependently, and also did 1 % of enflurane. Higher concentrations of enflurane significantly increased the second OP (Fig. 2). Induced hypotension below 30 mm of mean arterial pressure had no influence on the pattern of control ERG at all.

Discussion. Our data clearly indicate that the reversible alteration of the OPs' amplitude does occur following volatile anesthetic inhalation. The OPs' amplitude could provide a useful index of anesthetic depth. Wachtmeister & Dowling reported that all the OPs were selectively abolished by GABA. It is interesting that volatile anesthetics possess the same action on the ERG as does GABA. On the contrary, higher concentrations of enflurane caused the increase in the second OP. This phenomenon might be related to its convulsive activity. These results suggest that the ERG could also provide a useful model to study anesthetic action on characteristic brain receptors for various neurotransmitters.