A BRAIN FUNCTION MONITOR FOR USE DURING ANAESTHESIA
PRELIMINARY REPORT

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MOST GENERAL ANAESTHETICS produce similar, predictable changes in the electroencephalogram (EEG). Deepening narcosis results in a gradual shift to ever-lower frequencies of the dominant rhythm recorded on the EEG, increasing the relative amplitude of delta frequencies.[1,2]

Impairment of cerebral oxygenation, also, increases delta-wave activity.[3] Therefore, monitoring the EEG during anaesthesia should provide useful information on both the depth of anaesthesia and the adequacy of cerebral oxygenation. Unfortunately, however, changes in the EEG during anaesthesia are too complex for most physicians to interpret and equipment capable of processing such data and presenting the results in simple form is not readily available.

This paper describes an inexpensive, noninvasive analogue device that analyses the EEG signal and provides an instant numerical display. It can be used to monitor anaesthetic depth and cerebral function.

METHODS

The depth of anaesthesia is monitored by measuring the relative amplitude of delta waves and the shift in frequency of the dominant rhythm in the EEG. The ratio of the mean amplitude of "augmented" delta frequencies (MADA) to the mean amplitude of the entire EEG signal (MA) is termed the augmented-delta quotient (ADQ); that is, the proportion of low frequencies in the EEG, which is a standard, dimensionless quantity. The ADQ is displayed on a meter and can be recorded along with other physiological data on a strip-chart recorder. A reading of 0 indicates no low frequency components in the EEG, and 1 indicates no high frequency components. Thus, all possible combinations of frequencies yield a quotient between 0 and 1.

A simple on-line brain-function monitor extracts the information, derives the ADQ, and displays the record. This device is termed an ADQ monitor. EEG electrodes are usually placed in a fronto-occipital configuration, and the ADQ monitor is attached to a standard EEG amplifier.

ADQ Monitor (Figure 1)

The monitor continuously measures the mean amplitude of the EEG in the low (delta) frequency range and divides this by the mean amplitude of the entire EEG signal. Filter A passes frequencies mainly in the delta range, but because of its inherent imperfect selectivity the delta frequencies are "augmented" by higher frequencies (Figure 2). This lack of selectivity is utilized to detect spectral shifts in the range of 3 to 10 Hz. The ADQ is extracted by dividing the rectified and averaged signal from filter A by the similarly processed signal from filter B.

The unit includes circuitry (not illustrated) that minimizes interference by electrosurgery and motion artifacts. Only signals that are within accepted limits of amplitude and rate of change can pass into the averagers; as long as artifacts are present the averagers maintain the last acceptable value.

Performance

In six preliminary experiments, two beagle dogs were anaesthetized by a bolus intravenous

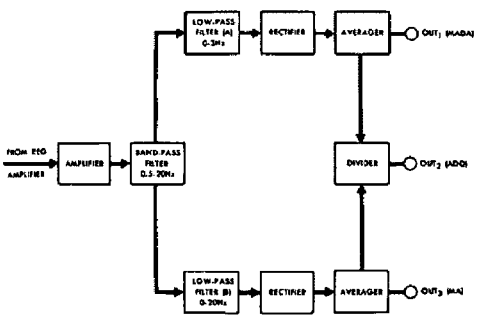

FIGURE 1. Simplified block diagram of the ADQ monitor. Upper third: site of extraction of mean augmented delta amplitude (MADA). Lower third: site of extraction of mean amplitude of the entire EEG (MA). Middle third: left, input; right, output as ADQ (MADA ÷ MA).

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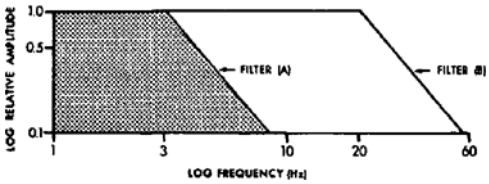

FIGURE 2. Frequency response of the two low-pass filters (A and B). The shaded portion represents frequencies passed in the delta range, augmented by higher frequencies that also are passed but with selectively diminished amplitude. Filter B passes all frequencies in the entire range (shaded and unshaded portions).

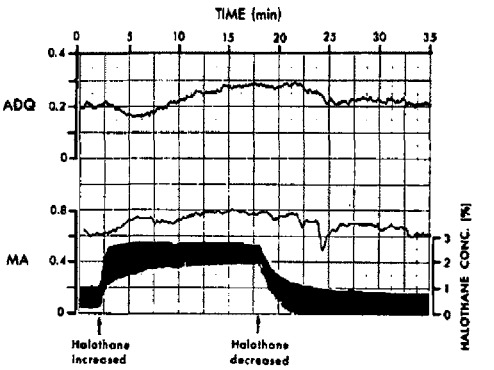

FIGURE 3. The effects of halothane on the ADQ and MA in a dog. The wide trace records the halothane concentration.

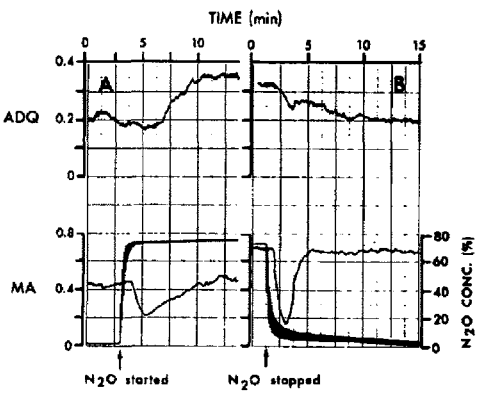

FIGURE 4. The effects of nitrous oxide on the ADQ and mean amplitude in dogs. A. during anesthesia; B. during recovery. The wide trace records the nitrous oxide concentration.

injection of thiopentone sodium (10 mg/kg body weight) and were maintained on 0.5 per cent halothane in various gas mixtures. D-Tubocurarine was injected intravenously at the beginning and at intervals throughout the experiments. The tracheae of the dogs were intubated, and the lungs were ventilated with air throughout to maintain a steady end-tidal carbon dioxide which was monitored continuously with a mass spectrometer.

Fronto-occipital EEG leads were attached and recording of the outputs from the ADQ monitor was begun. The halothane concentration was periodically increased to approximately 3 per cent until a steady-state recording was obtained, then returned to 0.5 per cent. Two hours after induction, 80 per cent nitrous oxide was added; this was continued until the recording stabilized and then was withdrawn. Ten minutes later 10 per cent carbon dioxide in air was added; this was continued until the recording stabilized and then was turned off. Fifteen minutes after the tracing showed recovery from the effects of carbon dioxide, a bolus of ketamine (2 mg/kg) was injected intravenously. When the recording ceased to show an effect of ketamine, the halothane was discontinued. The EEG electrodes were removed and the dogs were allowed to recover consciousness.

For 12 months (July 1976–June 1977) the ADQ was monitored periodically in children undergoing open-heart surgery, with hypothermia in some cases.

RESULTS

Figure 3 shows a representative recording by the ADQ monitor of the response to increasing the concentration of halothane from 0.5 per cent to approximately 3 per cent. After a slight initial decrease, which may indicate the hyperexcitation phase of anaesthesia, the ADQ increased. The effect on MA roughly parallels increases and decreases in the halothane concentration. In both dogs the response to repeated exposure to the same concentrations of halothane was slightly less each time.

The response of the ADQ to administration of 80 per cent nitrous oxide (Figure 4) was faster than with halothane and revealed no excitation phase. Temporary disappearance of the EEG when administration of 80 per cent nitrous oxide was started or stopped caused an abrupt, severe decrease in the MA but had little effect on the ADQ. When the signal recovered, the MA appeared little affected by the nitrous oxide.

When 10 per cent carbon dioxide was introduced during inhalation of air (Figure 5), the ADQ again increased faster than with halothane and

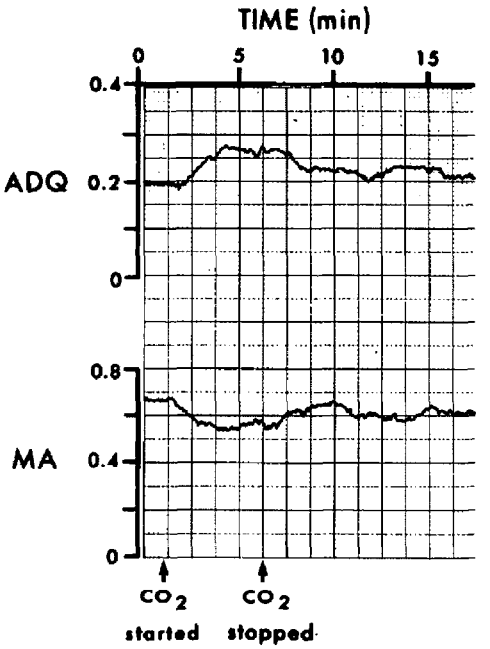

FIGURE 5. The effects of an inspired mixture of 10 per cent carbon dioxide in air, in dogs.

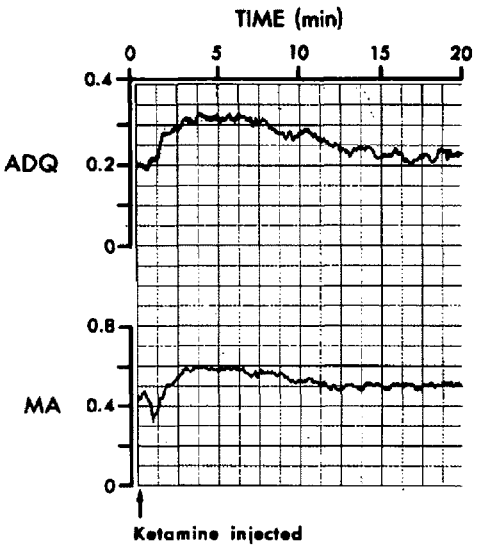

FIGURE 6. The effects of ketamine anaesthesia in dogs.

without an excitation phase. The MA decreased slightly.

The injection of ketamine resulted in a substantial rise in ADQ (Figure 6). After a brief initial fall

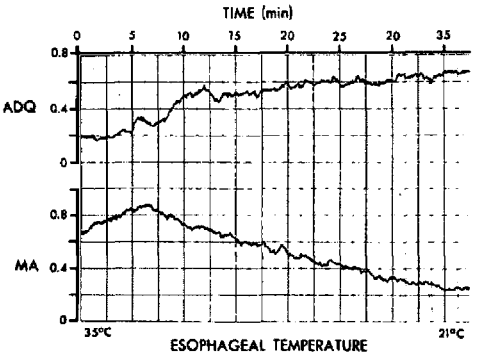

FIGURE 7. The effects of hypothermia during cardiopulmonary bypass in a child.

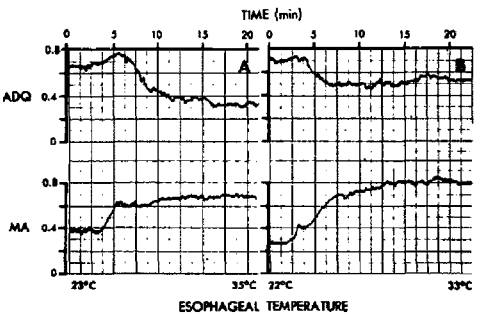

FIGURE 8. The effects of recovery from hypothermia on the ADQ and MA in two children. A recovery of ADQ is almost complete; B. the ADQ has not returned to normal.

(apparently due to disappearance of the EEG signal), the MA rose slightly.

During cardiopulmonary bypass with hypothermia, in a child, the ADQ increased steadily in response to cooling. The MA increased initially and then decreased almost linearly (Figure 7). During rewarming, the MA quickly returned to normal levels or higher, but the ADQ responded more slowly and in some cases remained high for a considerable time (Figure 8).

DISCUSSION

The EEG provides information generated simultaneously by many continuous biochemical processes within the brain, so that specific interpretation requires identification of the individual signals. The problem of selecting specific features of the EEG with which to follow the effects of anaesthesia is difficult.

The Fourier power spectrum provides a histogram of the distribution of relative signal strength

of the frequency bands of the EEG spectrum.[1,4,5] The display clearly reveals shifts in relative strength of the bands, information not available from visual analysis of the raw EEG signal, but the results are still in complex statistical form. Another method uses an analogue device (Cerebral Function Monitor; Devices Ltd., Welwyn Garden City, England),[6] which provides a filtered, compressed tracing of the EEG. This reveals only major trends in brain metabolism[7,8] and is relatively insensitive to the effects of inhalational anaesthetics.[9]

The ADQ monitor evaluates and compares the time course and efficacy of anaesthetic agents. As shown in Figures 3 to 6, the four agents tested in dogs produced similar shifts in the ADQ even though they had seemingly dissimilar effects on the EEG. Particularly noticeable is the effect of ketamine, which stimulates the central nervous system (CNS):[10] its effect on the ADQ is similar to that of halothane, which depresses the CNS. These uniform responses to such different agents indicate that the ADQ is sensitive to the depth of unconsciousness produced by anaesthetic or other agents. The observed rise in ADQ during hypothermia suggests that the ADQ also responds to changes in brain metabolism.

Summary

This simple on-line monitor provides simultaneous noninvasive quantification of continuous processes within the brain, by measuring the augmented delta quotient (ADQ; the proportion of low frequency components in the electroencephalogram). The response of the ADQ, both during experiments in animals and in children undergoing surgery, during cardiopulmonary bypass with and without hypothermia, demonstrates that this concept may prove valuable both to monitor anaesthetic depth and to warn of impending cerebral impairment.

Résumé

L'auteur décrit un moniteur simple qui permet l'évaluation continue quantitative et non invasive de la fonction cérébrale par la mesure du quotient delta augmenté (QDA ou la proportion des ondes de basse fréquence de l'électroencéphalogramme). La présente étude a pour but de montrer l'utilité du QDA enregistré chez l'animal de laboratoire et l'enfant pendant la chirurgie sous circulation extra-corporelle, avec et sans hypothermie, comme moniteur de la profondeur anesthésique et de l'ischémie cérébrale.

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