International Journal of Clinical Monitoring and Computing 1: 201–210

Computerized monitoring of the EMG and EEG during anesthesia

An evaluation of the anesthesia and brain activity monitor (ABM®)

Harvey L. Edmonds, Jr.[1] and Markku Paloheimo[2]

[1] *Department of Anesthesiology, University of Louisville, Louisville, KY 40292, USA;* [2] *Department of Anesthesia, Surgical Hospital, Kasarmink 11–13, SF-00130 Helsinki 13, Finland*

Keywords: amplitude analysis, electroencephalography (EEG), electromyography (EMG), frequency analysis

Summary

An intraoperative evaluation was made of the electroencephalographic (EEG) and electromyographic (EMG) functions of the Anesthesia and Brain activity Monitor (ABM®). This device derives both these measures from a single electrode pair that is typically placed on the mid-forehead and mastoid process. The evaluation consisted of 1) quantifying the zero-crossing frequency (ZXF) of the EEG and mean integrated voltage of both measures (MIV_{EEG} and MIV_{EMG}) that occurred during induction and emergence from general anesthesia in 17 patients and 2) case reports sampled from an additional 41 patients.

Alone or combined, variations in these parameters did not consistently accompany changes in the depth or adequacy of anesthesia as determined by standard clinical signs (e.g. heart rate, blood pressure, movement). Interpatient variability in the EEG measures during recovery from anesthesia was so large that neither the absolute value of ZXF nor that of MIV_{EEG} could discriminate between moderate (i.e., maintenance) and light (i.e., emergence) anesthesia. Although MIV_{EMG} uniformly decreased in anesthetized, unparalyzed patients (compared to the pre-operative awake state), noticeable increases during recovery often did not occur until limb movement was observed. Additionally, the common use of neuromuscular blockers made interpretation of low MIV_{EMG} values quite difficult during anesthesia maintenance.

However, selected individual case reports illustrated the potential benefit of routine intraoperative, microprocessor-based EEG/EMG monitoring. The single channel EEG/EMG display of the ABM seems sufficient to warn the anesthesiologist of pathologic decreases in cerebral electrical activity. Marked depression of cerebral function is associated with accidental anesthetic overdose, hypoxia or global ischemia. Additionally, the device should be useful for monitoring burst-suppression or isoelectric EEG patterns intentionally produced during barbiturate or isoflurane coma for cerebral protection.

Introduction

Until the advent of the microprocessor, intraoperative monitoring of the electroencephalogram (EEG) required cumbersome, expensive equipment, a dedicated operator and a skilled interpreter. These disadvantages and the large volume of information generated by paper EEG tracings limited this type of monitoring to a few, specialized centers (14). Now, however, a wide range of microcomputer-based devices are commercially available that can record, analyze and display EEG information in highly condensed forms. These devices vary widely in complexity and therefore cost.

The simplest EEG analyzers provide single measures of the amplitude and frequency content of one channel of EEG. Most commonly, the frequency characteristics (period) of the signal are measured by the mean zero-crossing frequency (ZXF). Amplitude of the signal is indicated by mean integrated voltage (MIV) or its squared value (power). Examples of devices using this period-amplitude analysis are the Cerebral Function Analyzing Monitor® (1) and the Anesthesia and Brain activity Monitor® (ABM) (3). At the other end of the scale lie instruments that simultaneously indicate EEG amplitude (power) and frequency content of multiple channels (11). The purpose of the present study was to determine if the simplest form of analysis (single channel period-amplitude) was sufficient to reliably monitor cerebral electrical activity and the depth of anesthesia during routine surgical cases.

The ABM was chosen for this determination because it additionally measures both spontaneous and electrically evoked electromyographic (EMG) activities. Fink (4) and Harmel et al. (5) have shown that, in the absence of neuromuscular blocking agents, the resting tone of skeletal muscles is related to the magnitude of anesthetic effect. Furthermore, some spontaneous activity persists with the use of neuromuscular blockers if the amplitude of the electrically evoked EMG response is not depressed more than 60–70% (Paloheimo, unpublished observation).

Methods

Subjects

ABM recordings were made on 58 subjects, aged 2 months to 78 years, who were to undergo general anesthesia for a surgical procedure. Illustrative case reports were obtained from this population. Additionally, a subgroup of 17 subjects was selected (Table 1) who had received general anesthesia under a set of very similar circumstances (Table 2). Quantitative statistical comparisons were restricted to this subgroup.

Table 1. Subject profile for quantitative EEG evaluation during standardized anesthetic protocol.

Halothane (0.5–1%)		Enflurane (1–2%)		Isoflurane (0.5–1%)	
Age	Sex	Age	Sex	Age	Sex
18	M	24	F	27	F
20	F	36	F	30	F
22	F	53	M	31	M
46	M	60	M	38	F
60	F	62	M	38	F
				38	F
				46	F

Table 2. Standardized anesthetic protocol.

A. Pre-medication
   1. morphine sulfate 0.1 mg/kg or fentanyl 0.6 mcg/kg
   2. glycopyrrolate 3 mcg/kg
   3. d-tubocurarine 40 mcg/kg

B. Induction
   thiopental sodium 2.5 mg/kg

C. Endotracheal intubation
   succinylcholine 2 mg/kg

D. Maintenance of anesthesia with gaseous anesthetics
   1. nitrous oxide: oxygen (60:40)
   2. halothane (0.5–1%) or enflurane (1–2%) or isoflurane (0.5–1%) for 1–2 h

E. Neuromuscular blockade
   1. pancuronium bromide 0.1 mg/kg
   2. prostigmine 0.2 mg/kg, if needed for reversal of blockade

Characteristics of the ABM

Combined EEG/EMG monitoring was achieved with an Anesthesia and Brain activity Monitor (ABM), DATEX/Instrumentarium Oy, Helsinki, Finland, (Puritan-Bennett Corp., Boston, Mass. in the USA). Additionally, the ABM provided parallel displays of end-tidal carbon dioxide, systemic blood pressure and the electrically evoked neuromuscular response (NMT) to train-of-four stimulation. These latter variables are of considerable importance in the accurate interpretation of changes in EMG and EEG signals during anesthesia.

The ABM analyzes a biopential obtained from adhesive pediatric ECG electrodes placed typically on the mid-forehead and mastoid process. Portions of the signal are selectively filtered to obtain spontaneous frontalis muscle EMG (65–300 Hz) and EEG (1.5–25 Hz). This filtering eliminates low frequency movement and 50–60 Hz powerline contamination. Additional filtering removes the DC component of the AC signal which prevents inaccuracies in zero-crossing frequency determination associated with fluctuations in electrode impedance.

Amplitude of the EMG and EEG signals is quantified and displayed in an analogus manner. The filtered signals are full-wave rectified which eliminates the negative components of the signal by producing a pulsating DC current at twice the frequency of the AC input. After integration, the signal is passed through a log amplifier whose output is proportional to the logarithm of the input. Values are computed as mean integrated voltage (MIV)/sec. These numerical values are displayed on a video monitor and updated each second. Trending is accomplished by displaying fixed 10 sec averages of each parameter as a series of vertical bars (histograms) on 'logarithmic' scales (Fig. 1). A video display of the most recent 30 min period and a graphics printout of the entire operation are provided. Presence of an EMG signal equal to or greater than 40 scale units, whether real or artifactual, is indicated by a slight (one-dot) elevation on the base of the video and graphic displays and a striped pattern on the EEG video display. The letters OVLD (for overload) are presented below the digital EMG value on the video display when it exceeds 70% of value necessary to saturate the EMG amplifier. A full scale signal on the EMG trace represents an amplitude of 15 microvolts whereas the maximal EEG amplitude value displayed is 60 microvolts. Frequency content of the EEG signal is estimated by the mean zero-crossing frequency (ZXF). Similarly, the digital value for this parameter is displayed each second and 10 sec averages are shown in histogram fashion. Detailed descriptions have been made of the theory (10), clinical applications (6, 7) and limitations (8) of ZXF/MIV (period-amplitude) analysis.

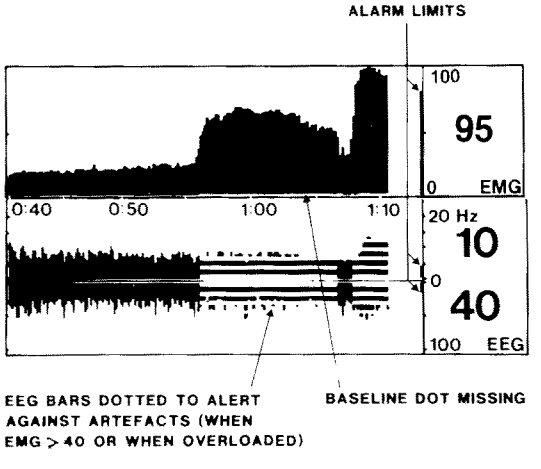

Figure 1. The appearance of the trended EMG (upper trace) and EEG (lower traces) histogram displays are shown. Note in particular the indications of possible artifactual contamination and the alarm limits. See Methods for a detailed description.

The thick vertical bars between the EMG/EEG histograms and their current digital values represent the thresholds for audio alarms. EMG values above threshold and EEG values below threshold trigger the alarm. Activation of the alarm and adjustment of the thresholds are under user control.

The EMG and EEG traces of the ABM are deceptively simple in appearance. Some of the actual complexity derives from the method of display. Although scaling for the EMG and EEG traces is described by the manufacturer as 'semi-logarithmic', it is more precisely characterized as non-linear. The display algorithm modifies the output of the log amplifiers to further accentuate low amplitude changes. Calibration curves of sine wave signals were used to depict the relationships between actual and displayed amplitude measures (Fig. 2). In practice, this non-linearity may be of value in detecting small changes in low amplitude signals. However, complexity or difficulty in quantification may arise if the user does not understand the scaling method.

The NMT portion of the monitor produces a train-of-four (TOF) stimulation of a motor nerve every 20 sec. The integrated amplitude recorded from corresponding muscles is compared to a non-relaxed reference response (obtained during an automatic calibration procedure), scaled accordingly, and displayed linearly as four bars, respec-

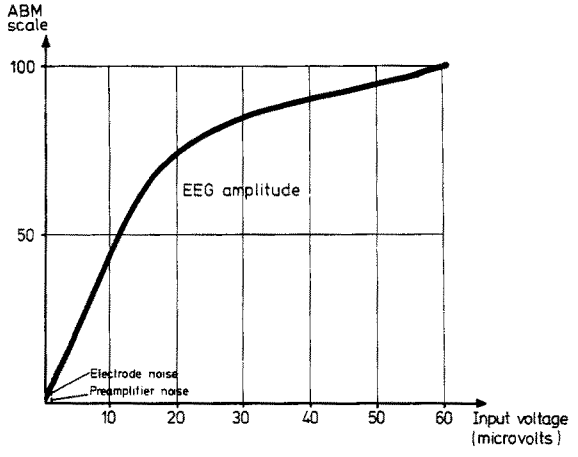

Figure 2. The non-linearity of the EEG amplitude scale is depicted in this figure. An absolute change in the amplitude of the input results in larger display changes for low amplitude than high amplitude signals.

tively (Fig. 3). Means of three successive TOF responses form a minute-to-minute trend on the video screen. Percentage of the first TOF response (T1) of the reference level (T1%) and the ratio of the fourth to the first (T4/T1 × 100 = TR%) are also displayed digitally on the video screen. A decrease in the amplitude of the fourth TOF response to the first ('fade') is indicated by a series of larger amplitude thin vertical bars (first response) superimposed on the lower amplitude solid background (fourth response) (Fig. 3).

The automatic NMT calibration procedure began immediately after thiopental induction but before administration of succinylcholine for endotracheal intubation. The ABM searches for a supramaximal stimulation current by stepwise increases from 5 mA. When two successive responses differ less than 10% a 'maximal stimulation current' is defined. A supramaximal current (maximal + 10 mA) is then used to excite all responsive neuromuscular units and establish a 100% reference response. The reference level is retained in the processor memory, even in the event of power failure, until a reset button is depressed for more than 5 sec.

Every 10 sec a RS-232C serial I/O port on the rear of the ABM provides the digitized values (in ASCII code) used to produce all histogram displays. The filtered, but otherwise unprocessed, analog EEG and end-tidal CO_2 waveforms are also available at a rear panel connector for direct observation on an oscilloscope or pen recorder and for parallel processing by other devices.

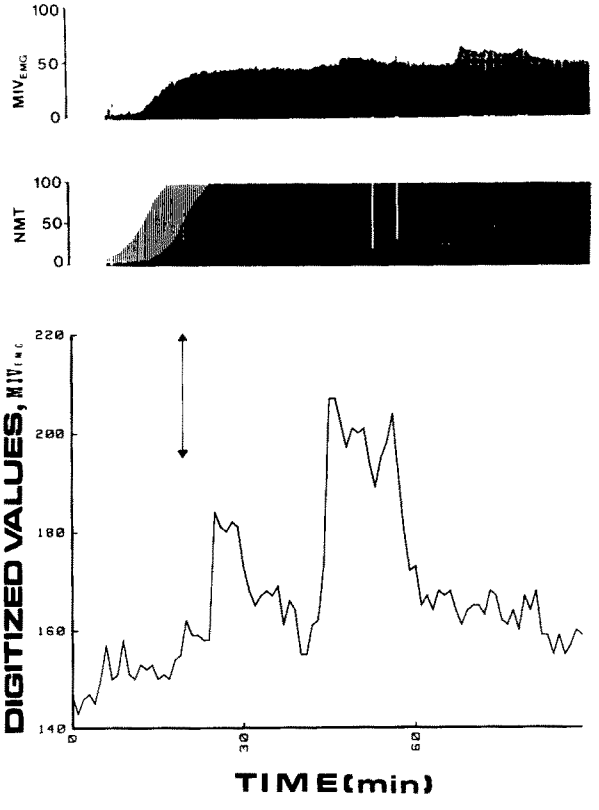

Figure 3. This figure illustrates recovery of spontaneous (EMG) and electrically evoked (NMT) responses from a non-depolarizing neuromuscular blockade produced by vecuronium. 'Fade' in the train-of-four response is indicated on the NMT by the thin vertical bars (first response) superimposed on the solid background (fourth response). The bottom trace plots the digitized values (from the serial I/O port) used to generate the EMG trace on an expanded linear scale. The infusion rate of the anesthetic alfentanil was halved at the arrow.

Intraoperative evaluation

Average one-minute values of ZXF, MIV_{EEG} and MIV_{EMG}, obtained from the graphics printout of the ABM display, were determined 1–2 and 10 min after induction of anesthesia with thiopental. The precise time point chosen for the first measurement was the lowest observed ZXF (e.g., maximal anes-

thetic effect). The time of appearance of this minimum varied slightly among subjects because of the difference in circulation times. The 10 min point was chosen because prominent thiopental effects had subsided and it was unlikely that alveolar concentrations of gaseous anesthetic were yet adequate for surgical anesthesia (9).

In addition, these variables were quantified during emergence. Average 1-min values were obtained immediately before and 10–12 min after discontinuation of inhalational anesthetics. EEG data were not used if indications of artifactual contamination existed (e.g. overload message or elevated baseline on EMG display). EMG data were not used if the amplitude of the first of the electrically evoked TOF responses was less than 30% of the baseline obtained during induction. Values recorded during early vs. late induction and maintenance vs. emergence were compared by Student's *t*-test for paired observation (two-tailed). Since mean values produced by the 3 gaseous anesthetics at each time point did not differ, they were combined prior to statistical comparison.

Data compression techniques used in the video and graphics displays considerably decreased the sensitivity of the period-amplitude analysis. This is because the 0–255 fullscale resolution of the analog-digital converters was reduced to a 0–20 linear scale for ZXF and 0–100 logarithmic scales for MIVs. Therefore, in some cases the original digitized values used to generate these displays, available at the RS-232C I/O port, were stored by a Hewlett-Packard 9816 computer for further analysis.

Experimental evaluation

Due to the infrequency of intraoperative situations that severely disrupted cerebral electrical activity (e.g., cerebral ischemia), it became necessary to produce these changes experimentally to complete the evaluation of the ABM. These studies were performed on adult mongrel dogs anesthetized with an alfentanil infusion and immobilized with pancuronium bromide. Incomplete global ischemia was generated by ligation of all intercostal arteries in addition to the subclavian and brachiocephalic arteries. The blood pressure rise typically seen following the onset of cerebral ischemia was blunted by splenectomy. In some animals the global ischemia was made complete by the rapid withdrawal of sufficient blood to keep mean carotid artery pressure below 30 mm Hg (i.e., distal to the ligation) while maintaining mean systemic pressure above 70 mm Hg. The brain-thorax temperature difference was recorded to provide a continuous, qualitative estimate of the degree of cerebral hypoperfusion. The EEG signal was obtained from stainless steel screw electrodes fixed in the skull over central and occipital cortex.

Results

Depth of anesthesia

Using period-amplitude analysis, it has been observed that, except for very deep levels of anesthesia (i.e., flat EEG), mean EEG amplitude is directly related to the depth of anesthesia (7); the inverse is true for frequency. Such a relationship between these simple EEG descriptors and anesthetic depth is shown in Fig. 4. In this case, the

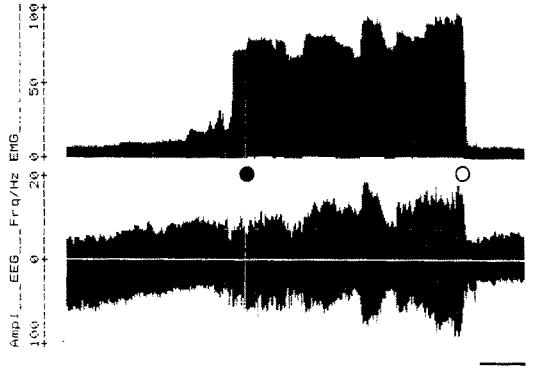

Figure 4. Depletion of an isoflurane vaporizer (approximately at the beginning of the trace) resulted in a marked increase in EMG amplitude (●) several minutes before the first observed movement (○). EMG contamination of EEG frequency (ZXF) and amplitude (MIV_{EEG}) histograms (between ● and ○) is also apparent. Pancuronium bromide (0.1 mg/kg i.v.) abolished skeletal movement and the source of contamination (○). Note decreases in MIV_{EEG} and increases in ZXF as the depth of anesthesia decreases prior to, and after, the period of EEG contamination. The horizontal bar at the bottom right is a 10 min time marker.

accidental depletion of an isoflurane vaporizer was initially unnoticed. Prior to any signs of increased autonomic activity or movement in this unparalyzed patient a marked increase in ZXF and decrease in MIV_{EEG} was seen, suggestive of cerebral activation.

Reults of the statistical analysis of EEG changes during induction are in partial agreement with this observation. During the period of maximal thiopental effect (1–2 min) the group mean (± s.d) ZXF was 3 ± 1 Hz. By 10 min postinjection it has increased markedly to 8 ± 4 Hz ($t = 6.22$, 16 df, $P < .001$). Similar, but less striking, changes occurred with MIV_{EEG}. The 1–2 min value of 73 ± 22 ABM amplitude scale units (ASU) decreased to 64 ± 21 ASU ($t = 2.31$, 16 df, $P < .05$). However, during emergence from anesthesia there were no significant changes in either EEG measure. The ZXF and MIV_{EEG} during surgical anesthesia, 8 ± 4 Hz and 54 ± 20 ASU, respectively, changed to 9 ± 3 Hz and 47 ± 18 ASU in the emergence period. Thus, large anesthetic-related increases in patient responsiveness seen during emergence, as judged by the clinical signs of anesthetic depth, were not regularly accompanied by predictable changes in the EEG.

Induction of general anesthesia was uniformly associated with a marked decrease in MIV_{EMG} from the conscious level. Unfortunately the magnitude of this change could not be quantified precisely, because the high level of spontaneous frontalis muscle activity in conscious subjects saturates the EMG preamplifier. Nevertheless, the waking MIV_{EMG} was very high (e.g., >70 ASU) in all patients preoperatively. With adequate anesthesia and in the absence of obvious movement artifact or electrical interference, a marked decrease in the EMG amplitude occurred and persisted until discontinuation of anesthesia. If spontaneous EMG activity was not totally obliterated by high doses of neuromuscular blocking agents, apparently nonartifactual increases in MIV_{EMG} sometimes (8/58) occurred with decreasing anesthetic depth. The phenomenon is illustrated in the case described above (Fig. 4). Depletion of the anesthetic vaporizer led to increased EMG activity prior to any observable movement.

Generally, however, MIV_{EMG} was not related to depth of anesthesia in a linear fashion. During emergence from anesthesia EMG activity was most often (50/58) seen to increase abruptly (i.e., with 10–20 sec) either preceeding or coincident with movement of skeletal muscles. The mean value recorded during anesthesia maintenance (8 ± 2 ASU) was not significantly changed during emergence (27 ± 26 ASU) because of enormous intersubject variability.

Cerebral hypoxia

A global decline in brain electrical activity is a well-established correlate of severe hypotension or hypoxia. Such events are detected by a reduction of ZXF and MIV_{EEG} to values normally encountered only during hypothermia, very deep anesthesia or post-ictal depression. During our evaluation only one case of probably hypoxia occurred. In the course of a mask induction of anesthesia with

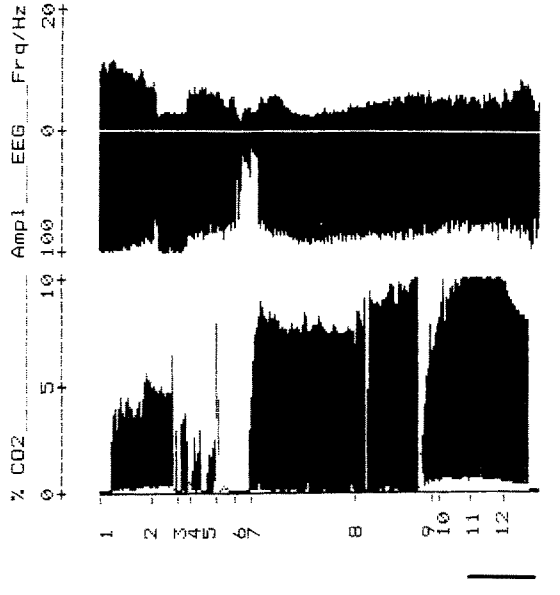

Figure 5. Induction of halothane by face mask began shortly before event marker 2. Laryngospasm and apparent hypoxia began to devolop at marker 3. Both EEG frequency and amplitude were greatly diminished at marker 5. Establishment of a patent airway following intubation at marker 7 promptly resulted in increased EEG activity. Subsequent ventilator malfunction (before marker 9) lead to an elevated end-tidal PCO_2 with only slight changes in EEG amplitude and frequency. The horizontal bar at the bottom right is a 10 min time marker.

halothane, a 6-year-old girl developed laryngospasm and became cyanotic. End-tidal PCO_2 values indicated hypercarbia although arterial PO_2 samples were not obtained (Fig. 5). This apparent cerebral hypoxia was associated with marked decreases in ZXF and MIV_{EEG}. Had the laryngospasm developed only after enclosure of the patient in surgical drapes, the EEG and capnographic functions of the ABM would have provided the only immediate indications of possible hypoxia.

Cerebral ischemia

Figure 6 depicts the ABM EEG changes associated with experimentally induced complete (left panel) and incomplete (right panel) cerebral ischemia. Complete ischemia resulted in a total abolition of cerebral electrical activity within 15–20 seconds of vascular occlusion (clamp). In this situation the difference in brain-thorax temperature (i.e., brain temperature) fell rapidly.

If the systemic blood pressure was not controlled, the hypertension produced by the vascular clamping seemed to prevent the immediate production of complete cerebral ischemia. The slower decline in brain-thorax temperature suggests that cerebral blood flow was not totally disrupted by clamping. EEG changes were consistent with this interpretation. The preischemic unprocessed EEG, containing much low voltage high frequency activity (tracing A), changed to an early ischemic pattern consisting almost entirely of high amplitude very low frequency waves (tracing B). Even-

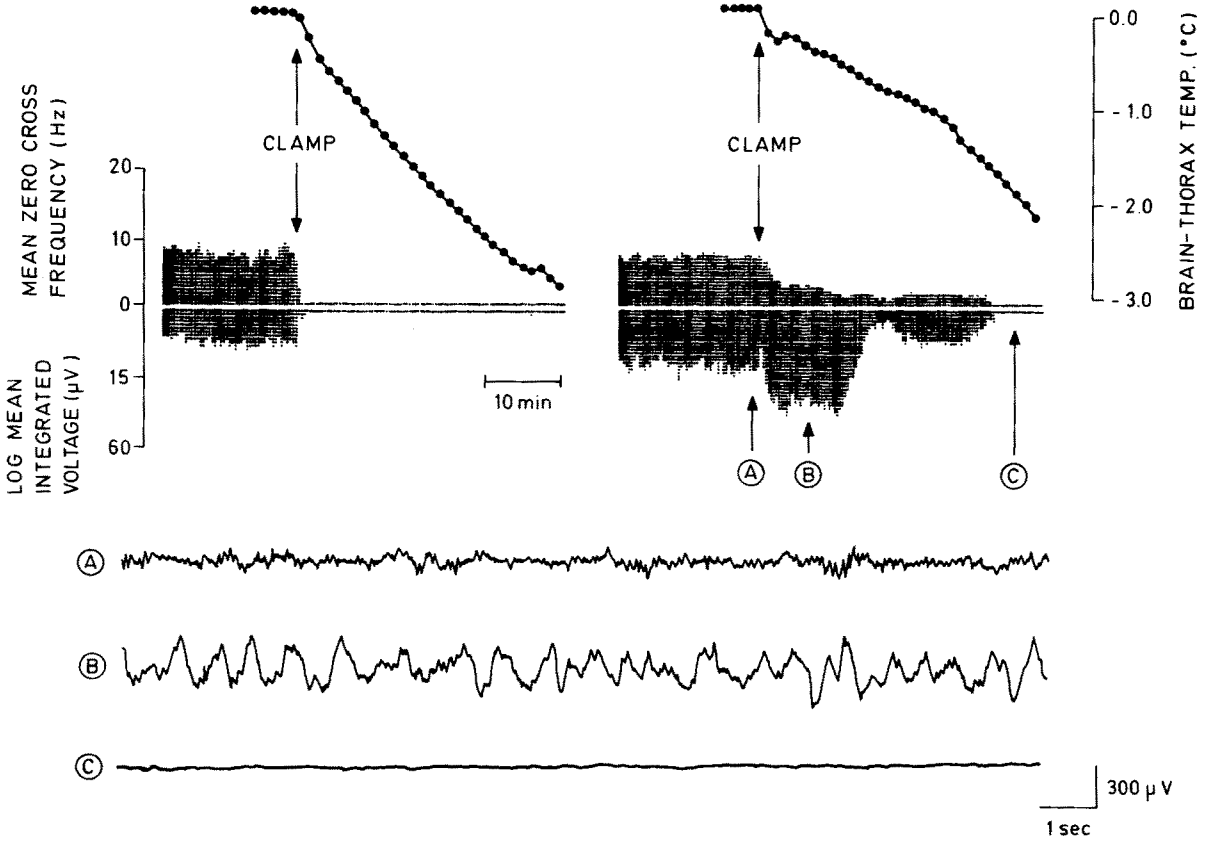

Figure 6. The upper left panel illustrates the rapid fall in brain temperature (e.g. brain-thorax temp.) and immediate cessation of EEG activity following complete global cerebral ischemia produced by vascular clamping and controlled hypotension. In contrast, incomplete ischemia due to clamping with uncontrolled reflex hypertension led to more gradual reductions in canine brain temperature and EEG activity (upper right panel). Segments of unprocessed EEG activity associated with these changes are shown in the bottom traces A, B, and C.

tual complete cessation of cerebral electrical activity some 30 min after onset of vascular clamping (tracing C) was preceded by late ischemic changes (low amplitude low frequency). These characteristic ABM EEG patterns were consistently observed in 15 dogs with complete ischemia and 13 dogs with incomplete ischemia.

Rescaling of the ABM digital output

During the study on experimentally induced cerebral ischemia we compared the ZXF, MIV_{EEG} and MIV_{EMG} data from the graphic display with digitized values of these parameters obtained from the RS-232C serial I/O port. This comparison indicated that marked changes observed in digitized parameters were sometimes undetectable from the low resolution graphic display. Therefore, in an additional surgical case we examined the relationship between the digitized and graphically displayed values of these parameters during a controlled change in the level of anesthesia. Digitized values were stored and replotted on an expanded scale (see Methods).

An adult male ASA class I patient, following premedication with diazepam 10 mg, was anesthetized with an alfentanil (4 mcg/min) infusion. Immobilization was achieved with vecuronium (50 mcg/kg). After the most stressful portion of the surgery the amplitude of the electrically evoked neuromuscular response (NMT) had returned to 90% of the pre-immobilization baseline (Fig. 3, left portion, 2nd trace). At this time the alfentanil infusion rate was halved (Figs. 3 and 7 at arrow). Thus, subsequent changes in MIV_{EMG} could not be attributed to the effects of the neuromuscular blocker. During the following hour a marked increase in ZXF was readily apparent on the expanded scale (Fig. 7 bottom panel) while no such change could be seen on the ABM graphic display (Fig. 7, upper panel). ZXF values 45–60 min after change to the lower 2 mcg/min infusion rate did not overlap with those seen at the end of the 4 mcg/min infusion period. In contrast, the MIV_{EEG} was too variable to be of use in indicating a decreasing level of anesthesia. The baseline MIV_{EMG} (Fig. 3 upper trace) had not increased 1 h after the alfentanil

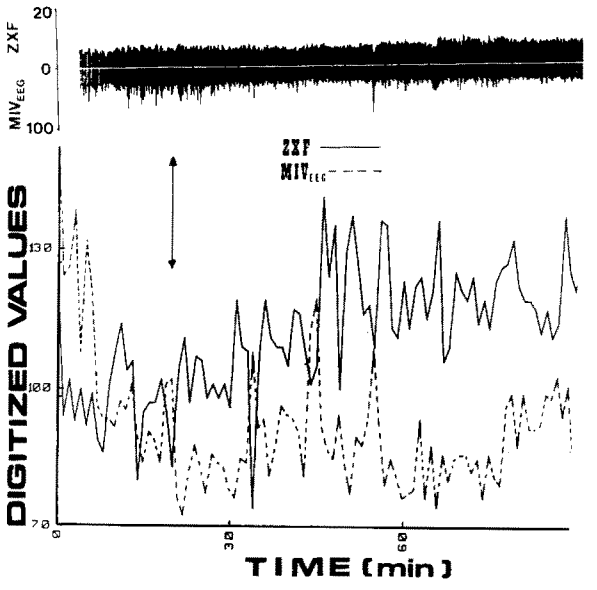

Figure 7. This ABM recording was obtained from an anesthetized patient in the absence of marked surgical stress. The rate of alfentanil infusion was halved at the arrow. No changes in the ABM graphic EEG display (upper trace) were apparent up to one hr after the decreased infusion, although clinical signs indicated enhanced patient responsiveness. A clear increase in ZXF is seen on the expanded scale (bottom panel) following the decreased administration rate of this short-acting anesthetic. However, the fluctuations in MIV_{EEG} were too large and varied to observe a consistent trend.

infusion rate was halved. Instead, paroxysms of increased amplitude occurred during this period. These paroxysms were readily detectable only on the expanded digitized scale (Fig. 7, bottom panel). The EMG changes on the logarithmic ABM graphic display were almost indiscernable.

Discussion

An important consideration in the evaluation of clinical monitors is the distinction of fact from artifact. There are at least five causes for artifactual contamination of the cranial biopotential employed by the ABM for EEG and EMG analysis. The first is improper electrode contact with the skin. Second, contamination may occur by monopolar electrocautery or a similar device. These two sources of contamination are indicated on the ABM by maximal increases in EMG and EEG

amplitude, an overload message, elevated baseline on the video and graphic EMG displays and a striped pattern on the video EEG display.

The third and fourth causes are more difficult to detect, however. Bipolar electrocautery artifact may appear as brief transients of moderate amplitude superimposed on a low amplitude baseline. The varying frequency of this signal may result in contamination of either EMG or EEG. In the present evaluation, this source was avoided in the quantitative comparison by using data obtained only at the initiation and completion of the surgical procedure. Increased slow and rapid movements of the extraocular muscles appearing during emergence from anesthesia may contaminate both EMG and EEG displays. Unfortunately, the magnitude of such contamination can be assessed only by polygraphic recording of both vertical and horizontal eye movements. Therefore, the influence of eye movements on EEG/EMG signals obtained during emergence from anesthesia in this study could not be determined.

Fifth, the EEG may be contaminated by large amplitude EMG signals. Much muscle activity lies outside the ABM EEG frequency range (1–25 Hz) and is therefore selectively filtered out. However, significant muscle activity in the EEG frequency range can occur and thus contaminate both frequency and amplitude measurements. Unfortunately, it is not currently possible to distinguish between these low frequency signal sources in unparalyzed subjects. These comments on artifactual contamination should be borne in mind during the following discussion on the limitations of intraoperative applications of the ABM.

The major limitation of zero-crossing frequency analysis is that widely different EEG patterns may have the same ZXF (8). Additionally, its mathematical appropriateness is limited to narrow bandwidth signals whose frequency spectrum is Gaussian (8). Sulg (13) has verified such appropriateness in drug-free, awake, relaxed human subjects with eyes closed (i.e., alpha rhythm dominance). However, bandwidths and frequency spectra may vary widely in other behavioral states such as anesthesia, sleep, and coma.

Despite these seemingly formidable limitations, ZXF seems to be useful in the detection of pathologic or anesthetic-induced marked depression of cerebral function. For example, Sotaniemi et al. (12) have shown that the mean EEG frequency is the clinically most useful and statistically most accurate descriptor of frequency changes associated with cerebral ischemia. Our evaluation illustrated the usefulness of ZXF in describing the changes in cerebral activity associated with the administration of an induction dose of thiopental. Also, the single incident of probable hypoxia observed in our series of cases was associated with a large decrease in ZXF.

The inability of the ABM ZXF display to discriminate moderate from light anesthesia during emergence may be due partly to its highly compressed form. Expansion of the frequency scale to fully utilize frequency information provided by the analog/digital converter may facilitate detection of changes in patient responsiveness. For example, in experimental studies, we compared digitized ASCII values of ZXF with a series of spectral EEG parameters accompanying incomplete global ischemia in the dog (2). ZXF was significantly correlated with the decreased theta, alpha and beta and increased delta activity. However, Tolonen and Sulg (15) found significant correlation between ZXF and cerebral blood flow during infarct only on the affected hemisphere. Therefore, a single channel EEG analysis may not be adequate to reliably detect focal decreases in cerebral activity if the common fronto-occipital electrode derivation is employed.

Moderate changes in MIV_{EEG} often occur during a surgical procedure but the causes are many and varied. In our experience it could not be used to determine the depth or even adequacy of anesthesia. Instead, its reliable application was limited to the early detection of a situation of potentially great clinical significance – cerebral hypoactivity. A global decline in brain electrical activity is a well-established result of severe hypotension or hypoxia. Such events are detected by a marked reduction in MIV_{EEG} to values normally encountered only during global ischemia, hypothermia, very deep anesthesia or post-ictal depression. During our evaluation of the ABM, EEG amplitude changes occurring during the single case of proba-

ble hypoxia and the results of the experiments on global ischemia were consistent with this concept.

Based on these considerations we make the following conclusions about the usefulness of the ABM for intra-operative monitoring. First, alone or in combination, changes in the EEG and EMG displays do not necessarily accompany changes in the common clinical signs of anesthetic depth or adequacy. However, marked depression of cerebral activity (e.g., induction of anesthesia with thiopental) is reliably associated with a very low mean zero-crossing frequency. Thus, the ABM should be useful in providing audible and visible warnings of anesthetic overdose. This capability would also be of value when adjusting anesthetic dose to intentionally produce burst suppression or flat EEG patterns during cerebral protection. Second, in the absence of deep anesthesia or hypothermia, marked hypoxia or global ischemia lead to a large decrease in mean zero-crossing frequency of the EEG. Immediate warning of these conditions by the ABM may give operating room personnel additional valuable time to correct the causes of the depressed cerebral function. The trended EEG display can assist in the evaluation of the patient's response to resuscitative measures. Third, the ABM can determine relatively small changes in the electrical activity of the frontalis muscle in anesthetized, unparalyzed patients. However, the clinical significance of the absence or presence of such changes remains unclear because of large intersubject variability. Viewed from the perspective of patient safety, some form of EEG recording seems justified for routine intraoperative monitoring of cerebral function. The ABM appears to be one of the new generation of microprocessor-based devices suitable for this purpose.

Acknowledgements

HLE thanks Dr P.A.J. Janssen for support and use of his laboratory facilities, Professor A. Wauquier for his valuable guidance and W. Melis, W. Vanden Broeck and J. Van Loon for their expert technical assistance during the canine experiments conducted at Janssen Pharmaceutica, Beerse, Belgium. The helpful cooperation of DATEX and Puritan-Bennet Corp. is also gratefully acknowledged.

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Accepted 9 January 1985