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Anaesthesia, Visually Evoked Potentials and EEG - a Review

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INTRODUCTION

The rationale for monitoring Visually Evoked Potentials (VEP's) during anaesthesia can be summarized in three main points:

1) Attempts at correlating VEPs and depth of anaesthesia for the purposes of achieving better and/or automatic control of anaesthesia levels.

2) Monitoring of the function and preserved intactness of the optic nerve or chiasma during surgery.

3) Studies of the basic neuronal effects of anaesthetic agents on cortical cells.

Intraoperative VEPs can be significantly altered by a host of drugs. It is therefore crucial to be aware of the different effects of anaesthetics on VEP if any monitoring of visually evoked potentials is carried out during anaesthesia.

BASIC MONITORING TECHNIQUES

Depending to some extent on the purpose of the VEP monitoring, standard techniques can usually be used in the operating room, with an emphasis on proper grounding and shielding of patient and equipment. Silver-silver-chloride electrodes are recommended; pattern or flash stimuli can be used, although flash stimuli are easier to produce in the confines of the operating room. Flash stimuli can be delivered through closed eyelids using an LED array mounted on the interior surface of small goggles or via fibre optic cables transmitting light through a contact lens. The VEP to flash is more variable and less quantitative than pattern reversal VEPs. The rate of stimulation is usually 1 -2 Hz. A single channel recording using an Oz-Cz montage, low filter of 5 Hz, high filter of 100Hz, 500 ms sweep, and an average of 200 - 300 stimuli is usually adequate /Harper and Daube (1989)/. The VEP can be recorded with the eyes taped shut and the pupils small. The effect of taping the eyes shut seems relatively small. In one study, there was no significant difference between the EP amplitude with either the eyes closed or eyes opened. The eyes-closed VEPs were found to be simpler, larger, and easier to interpret

compared with the VEP with eyes opened. Pupillary dilation caused approximately a 30% increase in the VEP amplitude (Domino, 1967; Domino et al., 1963).

ANAESTHESIA AND EEG

Changes in the intraoperative electroencephalogram (EEG) will to some extent also be reflected on the VEP, hence it necessary to be aware of the basic effects of anaesthetics on the EEG. Approximate generalizations that can be made about changes in the EEG patterns associated with increasing depth of anaesthesia (Figure 1) are:

1: development of fast activity (20 to 30 Hz),
2: that is followed by a rhythmic activity of high voltage,
3: which erupts into irregular delta waves,
4: before passing to the burst suppression stage (characterized by the profound relaxation of abdominal musculature).

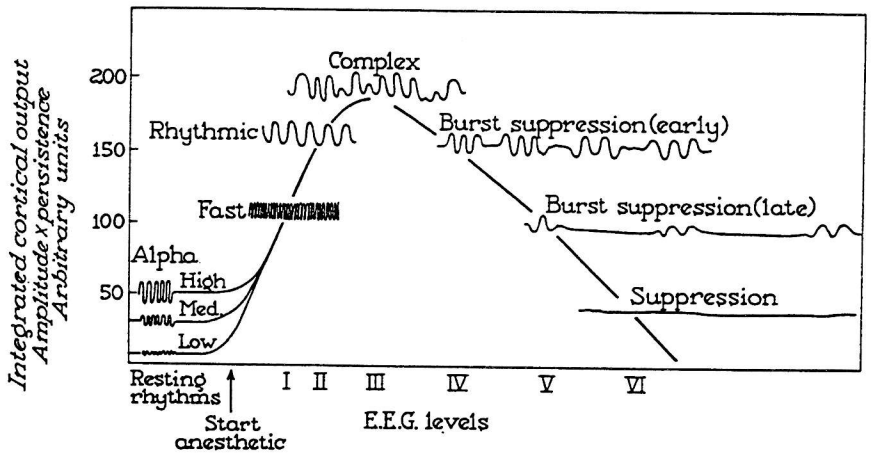

Figure 1. A schematic representation of average EEG pattern changes during anaesthesia. After Martin et al. (1959).

At deeper anaesthesia depths short periods of electrical silence begin to break into the record, signalling a stage at which minor surgery can be performed. At the level for major surgery, the duration of these silent intervals lengthens to 3 or 4 seconds and the record presents the pattern of *burst suppression* , first noticed by Derbyshire et al. (1936).

The emergence of EEG stages depends to a certain extent on the speed of induction of the anaesthesia and on the nature of the anaesthetic. For instance, nitrous oxide when administered as 80% NO_2 and 20% O_2 without additional anaesthetic agents evokes relatively slight effects on the EEG, consisting mostly of a change of amplitude and relatively mild slowing in frequency. There appears to be little relation between such changes and the usually defined level of consciousness /Henrie, 1961; Clutton-Brock, 1961). No stage of burst suppression is reached with nitrous oxide or chloroform /Brechner et al., 1962/.

With the slow administration of barbiturates /Brazier and Finesinger, 1945; Tucci et al., 1949), the initial stage in man is a change from the normal EEG pattern to a fast, high-voltage rhythm, with an initial frequency in the range 25-35 Hz which gradually slows to about 15-20 Hz. This change appears first in the frontal leads and then spreads posteriorly. Similar frequencies appear in the EEG after oral ingestion of barbiturates given in therapeutic doses, representing a subanaesthetic level. The burst suppression stage can be reached by oral ingestion of barbiturates (e.g. pheno-barbital) / Brazier ,1972/.

The EEG changes observed during anaesthesia are recorded at the scalp, and give little clue to the underlying brain mechanism. Implantable electrodes have given additional information, but their use on humans is naturally very restricted. The initial development of fast EEG activity in the prenarcotic stage of nearly all anaesthetics, but most markedly with the barbiturates, is a property of the nerve cells themselves and is found in all regions of neuronal aggregates. Apparently maximal in the superficial layers of the neocortex, the fast activity shows a falling gradient of voltage as one records from deeper points, and in the white matter only the field of the cortical potentials is detectable. If the same recording electrode penetrates as far as the orbital cortex (Figure 2), the deepest point of the needle electrode now enters other cell layers and again shows high voltage fast activity. The important point is that the fast waves of the orbital cortex appear to be independent of those of the cortex of the convexity. It seems reasonable to assume then that the barbiturate at this stage (in Figure 2) is acting directly on cortical cells and that the cortex is not being places into any kind of synchrony from below /Brazier et al., 1956/. A similar fast activity develops in other nerve cell aggregates such as the amygdala and hippocampus in man. These trains of fast waves are independent except in the case of anatomically closely linked structures, such as the posterior hippocampal gyrus and the ipsilateral hippocampus, or the dorsal medial nucleus of the thalamus and the frontal cortex /Brazier , 1968b/.

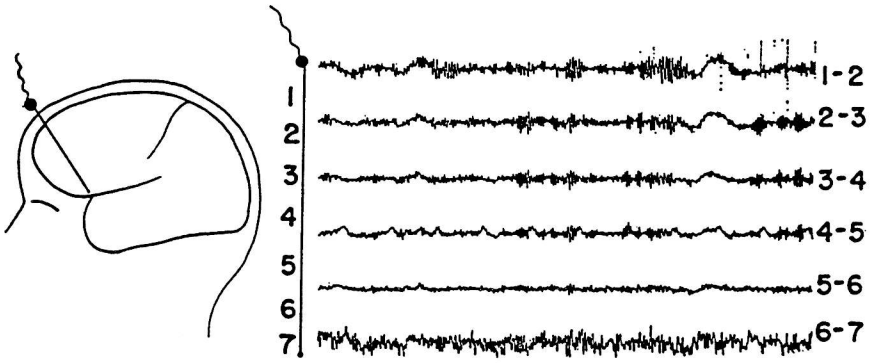

Figure 2. EEG recording from implanted needle electrode during thiopental anaesthesia / Brazier et al., 1956/.

When the anaesthetic level is deepened and the first hyperexitability of the nerve cells has passed, other mechanism come into play and the fast activity is replaced by slow waves. There is little doubt that these slow waves of anaesthesia have a different underlying mechanism from the slow waves of sleep. /Brazier, 1968a; Brazier 1972a; Brazier 1972b/. Mention has been made of the normally close coherence of the EEG activity in the posterior hippocampal gyrus and the ipsilateral hippocampus into which its anatomical projections run. These correlations are lost in normal sleep but are present in anaesthesia /Brazier, 1967a; Brazier 1967b/.

BASIC NEURONAL EFFECTS OF ANAESTHESIA

Anaesthetic agents could act in principle directly on the nerve cells themselves, the axons, the dendrites, or on the transmission of impulses across the synapse. Early exploration of the influence of anaesthetic agents on the nerve axon revealed that in the concentrations used in clinical work it was extremely unlikely that this is where a block takes place /Andersen, 1964/. Nerve fibres are very resistant to interference with transmission of nerve impulses among them. Conduction block does not take place in presynaptic terminals either, as shown by Somjen (1963). The most vulnerable link would then seem to be the synapses in the central nervous system. Nerve cells, when stimulated transsynaptically do not follow the all-or-nothing law, as does propagation in the nerve fibre. They respond to excitatory impulses with a graded depolarization that is proportional to the strength of the stimulus. These responses are known as "excitatory postsynaptic

potentials", or EPSP's. They are long lasting and, if too low amplitude to reach the discharge threshold of the cell can on repeated stimulation sum until the necessary degree of depolarization develops and a spike potential results. In the case of inhibitory action, a graded hyperpolarization of the cell membrane develops, of even longer duration, i.e. an "inhibitory postsynaptic potential", or IPSP, which impairs the ability of the cell to discharge.

The actions of halothane, isofurane and enflurane on spontaneous discharge and evoked action potential activity in mammalian A-delta and C fibre nociceptors from the in vitro rabbit cornea were investigated by MacIver and Tanelian (1990). At 1 MAC , all three significantly increased spontaneous discharge frequency of C fibres. Similar excitatory effects were observed with a potassium channel blocker.The anaesthetics produced burst discharge activity over concentration ranges of 0.25 - 1.5 MAC, and depressed discharge activity at concentrations greater than 3.0 MAC. Variable effects on evoked discharge activity of A-delta fibres were observed: halothane reduced action potential amplitude and increased spike latency, in contrast the ethers decreased both spike latency and action potential amplitude/ MacIver and Tanelian, 1990/.

In a study by Berg-Johnsen and Langamoen (1987), anaesthetic concentrations of isoflurane abolished spontaneous activity of human neocortex neurones (in vitro) and reduced synaptically evoked activity. The individual cells were not rendered inexitable nor was evoked synaptic activity prevented during increased afferent input. No induced epileptiform activity was observed. Isoflurane reversibly hyperpolarized the cell membrane in a dose-dependant manner; the hyperpolarization was accompanied by a reduction in input resistance. The effects remained unchanged after synaptic transmission was blocked / Berg-Johnsen and Langamoen, 1987/.

The effects of halothane, isoflurane, and enflurane on background neuronal activity and reactive capability in the central nervous system have been studied in cats by Agawa et al. (1992). The background activity was assessed by midbrain reticular cell firing, and the EEG in the cortex, amygdala and hippocampus. The reactive capability was assessed by evoked responses in the visual neuronal pathway. All anaesthetics studied suppressed reticular cell firing in a dose dependant manner. Spontaneous EEG spikes developed at 4.8% isoflurane and 3.6% enflurane. Photic stimulation provoked EEG spikes and repetitive stimulation induced seizure activity only at 3.6% enflurane. Halothane and isoflurane suppressed stimulation induced responses in the visual neuronal pathway (amplitude of N1 of visual cortical response 70% ±24.5% of control at 2.4 % halothane and 39.3±27.3% at 4.8%

isoflurane). Enflurane augmented the amplitude of photically evoked responses (398.4±83 at 3.6% enflurane). These results suggest that all agents studied had suppressive actions on background neuronal activity in the order halothane < isoflurane = enflurane /Agawa et al., 1992/.

Near clinical concentrations of halothane, isoflurane and enflurane slightly depress antidromic field potential responses of rat CA1 neurones. These anaesthetics block accomodation of pyramidal cell discharge in the rat hippocampus. Thus the anaesthetics may have a direct effect on membrane K^+ channels such as the Ca^{2+} activated K^+ conductance; the block of accomodation is unlikely to account for the proconvulsant action of enflurane /Southan and Wann, 1989/.

Among the many anaesthetic agents then there may be some which act by suppressing the excitation and others which increase the inhibitory action of the nerve cell. For transynaptic transmission the permeability of the postsynaptic membrane and the character of the ions that pass through it are fundamental processes that must be considered when studying the action of any anaesthetic agent.

EVOKED POTENTIALS AND ANAESTHESIA

The visually evoked potential has been studied in an attempt to define which of the elements of the evoked waveform can be assigned to excitatory postsynaptic potentials of cortical neurons and which can be identified as inhibitory postsynaptic potentials /Creutzfeldt et al., 1966; Purpura , 1955/. In the large pyramidal cells, the first surface-positive wave is associated with a rapid depolarization of the soma membrane caused by the attack on it of afferent impulses reaching it by axiosomatic synapses and causing a burst of EPSP's, i.e. of negativity at the cell-body making the surface above it relatively positive. As depolarization spreads up the apical dendrites, the surface itself loses its relative positivity, as even the most superficial terminals of the dendrites may be invaded resulting in a negative surface wave /Nelson and Frank, 1964/.
Creutzfeldt et al. (1966), after studying the visual system, are of the opinion that IPSP's also contribute to the wave form and duration of this surface negative wave, as they find IPSP's beginning at the soma at about the time that the surface response changes from positivity to negativity and lasting long into the sequence of changes that follow. Hence both excitation and inhibition result from afferent stimulation.
The response at the cortex to sensory stimulation is similar, except in location, whatever the sense modality used. It has a multiple waveform, which can be simplified as consisting of three prominent components:

1) a short latency response coming up the lemnisci and recordable also in the specific thalamic nuclei and reaching the cortex via the thalamocortical radiations.

2) a later component, not recordable in specific thalamic nuclei. This component has presumably travelled through the ascending reticular system and nonspecific thalamus, and thence projecting diffusely to the cortex.

3) a very prominent late component first observed and named by Forbes the "secondary discharge" / Forbes and Morison, 1939/. This late component was shown to have bypassed the specific thalamic nuclei and to be recordable all over the cortex.

These three components react differently to anaesthesia, and not in exactly the same way to all agents. With barbiturates the first component is very stable and requires very deep anaesthesia before it begins to be abolished. The second component is extremely vulnerable, and the third is even enhanced. The suggested explanation is that the second wave is blocked in its ascent through the reticular formation.

The late response of Forbes was first demonstrated in the somatosensory system, but later work showed a similar effect in the visual system. The response's exaggeration is not restricted to barbiturates but also occurs under chloralose, trimethanol, and ether / Brazier, 1963/. It has been suggested that at a stage preceding deep narcosis, the agent might be blocking an inhibitory mechanism whose removal unleashed the late response. The state of anaesthesia clearly does not require that sensory impulses should be prevented from reaching the cortex /Purpura, 1959/.

ANAESTHETIC AGENTS AND VEP

BARBITURATES

Barbiturates in general at relatively low doses cause the VEP amplitude to increase while the overall waveshape becomes relatively simplified (Brazier 1970a). At higher doses, barbiturates can attenuate and eventually abolish the VEP. The overall pattern is
1: amplitude increase at lesser doses, and
2: VEP attenuation and loss at deep levels of anaesthesia.

Serum levels of *pentobarbital* of 2.5 and 3.5 mg % had little effect on the VEP, beyond an amplitude attenuation due to drug-induced hypothermia (Newlon et al 1983).

Thiamyl sodium abolishes the VEP at depths of anaesthesia that also cause isoelectric EEG alternating with burst suppression EEG patterns (Domino et al. 1963). At lighter anaesthetic levels, the VEP was observable, but attenuated in amplitude. With still lighter anaesthesia the early VEP waves become very prominent in amplitude.

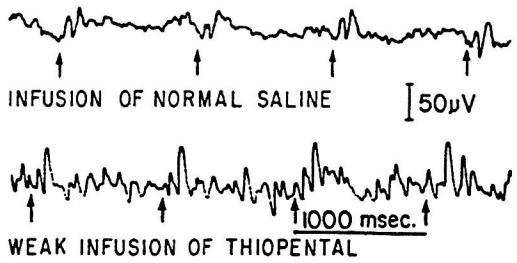

Figure 3. Prenarcotic effect of thiopental on F-VEP. Augmentation of the response is seen, in particular of the second positive-negative complex. Negativity plotted downward. /Brazier, 1961/.

The effect of thiopental on F-VEP has been recorded from implanted electrodes in the posterior hippocampus and the posterior hippocampal gyrus (Figure 4) . The response of the hippocampus is extremely vulnerable to the barbiturate, whereas the hippocampal gyrus is relatively resistant to the same /Brazier 1970b/.

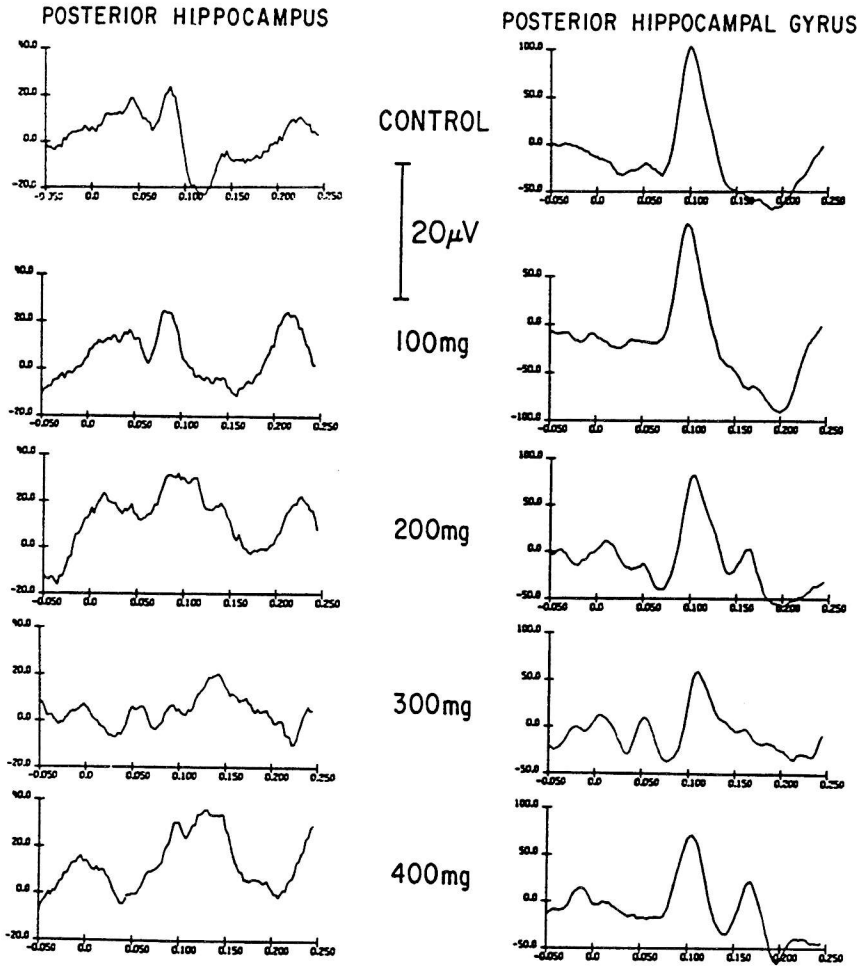

Figure 4. Effect of thiopental on photic response in man. Averaged responses (n=30). Simultaneous recordings from implanted electrodes /Brazier, 1970 b/.

NITROUS OXIDE

Nitrous oxide causes a gradual dose-dependent attenuation of VEP's (Uhl et al. 1980, Sebel et al. 1984). A mixture of 80% nitrous oxide and 20% oxygen results in a VEP approximately 50% of baseline amplitude (Domino et al. 1963). Nitrous oxide concentrations of 10%, 30% and 50% appear to cause VEP attenuation of approximately 10%, 20% and 30% (Sebel et al., 1984). See Figure 5.

THIOPENTONE-FENTANYL-NITROUS OXIDE

In a study by Chi et al. (1989), VEP was recorded before and at 1 min and 2 min after induction of anaesthesia with thiopentone. Anaesthesia was then maintained with fentanyl-nitrous oxide-oxygen combination, and VEP recorded again at 5, 10, 15 and 20 minutes after induction. The 1 and 2 min VEP showed a marked decrease in amplitudes, and increase in latencies. VEP amplitudes returned to control level at 15 min, while latencies remained increased throughout the study period. Therefore **thiopentone should be avoided during the critical period of VEP recording**. Once given, at least 15 min should elapse before VEP recording can be made. Thiopentone-fentanyl-nitrous oxide anaesthesia slightly increases the latencies of the VEP. / Chi et al. ,1989/

FENTANYL, DROPERIDOL

Fentanyl, droperidol, and nitrous oxide increased the latency of the P2 by 10%, with no significant changes in amplitude (at neuroleptanalgesia depths) /Russ et al., 1982/.

HALOTHANE AND FORANE

Halothane anaesthesia can result in moderate VEP amplitude increases similar to that seen with barbiturates (Figure 5). Even at deep halothane doses sufficient to cause profound cardiovascular depression, the VEP may be preserved. Halothane at a concentration above 2% can abolish the VEP (Costa e Silva et al., 1985; Wang et al., 1985). The effect is a direct drug effect, not due to hypotension.

Halothane systematically increases the interpeak latencies. One study indicated that the first positive peak increased in latency from 113 ms in the awake state to 134 msec at 1.13% end tidal halothane concentration (Uhl et al., 1980). Patients under *halothane* and *forane* anaesthesia had significantly larger P1 responses (P-VEP) and significantly shorter latency responses when compare to those under sedation with chloral hydrate. The waveforms obtained under anaesthesia were generally more variable than those obtained from the chloral hydrate sedation group, although they were more similar to the classic awake P-VEPs in overall shape / Fox and Wright,1990/.

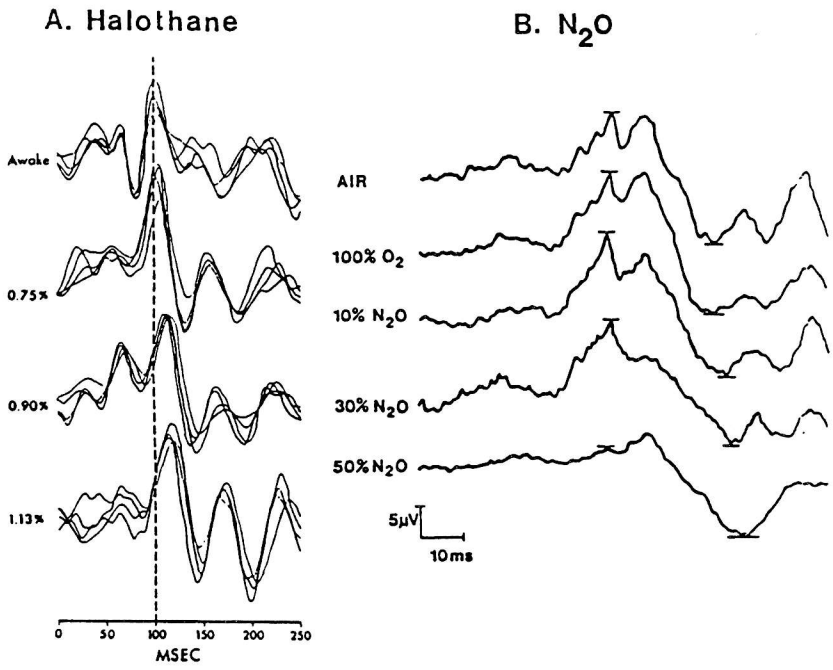

Figure 5. The effects of halothane (A) and nitrous oxide (B) on the VEP. /Uhl et al., 1980 (A); Sebel et al., 1984 (B)/.

ENFLURANE

Chi and Field (1990), studied the F-VEP recorded from Oz-Cz after expired concentrations of 1.2 %, 1.8%, 2.4% and 2.7% of enflurane in 100% oxygen. Amplitude of P100 was decreased and latency of N70 increased significantly with all concentrations of enflurane. No difference was found for different concentrations of enflurane. The latency of P100 increased significantly only at concentrations above MAC and at 2.7%, when it was significantly longer than those at 1.2 and 1.8%. /Chi and Field, 1990 /. In an earlier study by Burchiel et al. (1975), enflurane increased VEP amplitude by three to fivefold at 2.5% - 3.7%. Enflurane can produce seizure activity at a high concentration/ Burchiel et al., 1975/.

One animal report has looked at the effect of enlurane on subcortical and Rolandic cortical VEP's. At lower doses of enflurane (0.5% to 1.5 %), a

gradual dose-dependent amplitude attenuation was seen until the VEP was only 50% of its baseline amplitude. At higher doses of enflurane the VEP grew in amplitude, and at 4% enflurane reached 200% of baseline amplitude. In comparison, the EEG showed hypersynchronous spiking at intermediate anaesthetic doses in these rats, e.g. 1.5% enflurane; and at 4% enflurane the EEG recordings were nearly isoelectric /see Nuwer, 1986/.

ISOFLURANE

Chi and Field (1986) obtained F-VEP responses from 12 ASA I and II patients by using a goggle-mounted LED-array (see tables 1 and 2). Latencies of P1 (i.e. P100) increased with increasing concentrations of isoflurane (Figure 6). Prolongation was statistically significant at levels at or above 0.9 %. Differences of latencies between two consecutive levels of isoflurane were not statistically significant, except for the one between 0.6 % and 0.9%. The amplitude of the P1 decreased significantly at isoflurane levels of 0.6%, at levels above this the reduction was not statistically significant.

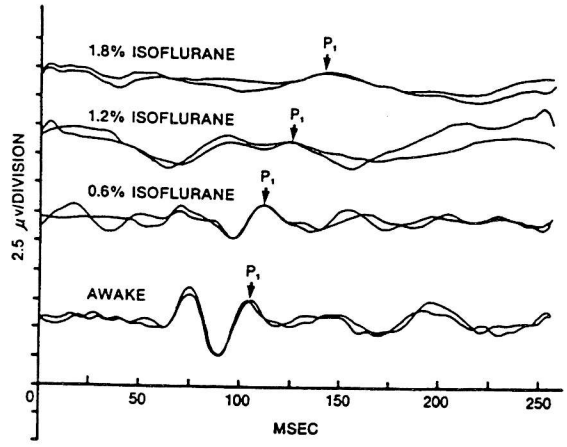

Figure 6. Effect of isoflurane on VEP at different isoflurane concentrations /Chi and Field (1986) /.

60% nitrous oxide to 0.6 % and 1.8% isoflurane increased the P1 latency statistically significantly, but 60% nitrous oxide at 1.2% isoflurane did not significantly increase the latency (compared to isoflurane alone).

60% nitrous oxide did not significantly affect the amplitude of P1, as compared to isoflurane alone (a slight increase in amplitude at 1.2% and 1.8% isoflurane was evident, but this was not statistically significant) /Chi and Field, 1986/.

Table 1. Mean latencies (ms± SD) of P1 of VEP with Isoflurane /Chi and Field, 1986/.

End-tidal isoflurane Concentrations	Latency ms± SD	Significance c.w. Control	Significance c.w. previous concentration
0% (awake)	102.6 ± 8.9		
0.6 %	107.6 ± 10.7	NS	NS
0.9 %	116.4 ± 8.1	p< 0.005	p< 0.05
1.2 %	122.2 ± 8.7	p< 0.001	NS
1.5 %	129.9 ± 8.8	p< 0.001	NS
1.8 %	132.7 ± 15.0	p< 0.001	NS

n= 12
NS= Not Significant
c.w. = compared with

Table 2. Mean Amplitudes (μV± SD) of P1 of VEP with Isoflurane /Chi and Field, 1986/.

End-tidal isoflurane Concentrations	Amplitudes μV ± SD	Significance c.w. Control	Significance c.w. previous concentration
0% (awake)	5.94 ± 2.37		
0.6 %	3.36 ± 2.21	p< 0.001	p< 0.001
1.2 %	2.06 ± 1.38	p< 0.001	NS
1.8 %	1.23 ± 0.79	p< 0.001	NS

n= 12
NS= Not Significant
c.w. = compared with

In a study by Sebel et al. (1986) consisting of 10 patients, the effects of 0.5 %, 1.1% and 1.65% end-tidal isoflurane concentrations were

examined. There were statistically significant increases in the latencies of the SEP N20, VEP and BAEP (waves II and IV) with increasing concentrations of isoflurane. The central conduction time was prolonged. Amplitudes of SEP and VEP potentials were reduced with increasing concentrations of isoflurane / Sebel et al. 1986/.

PROPOFOL

No data are available concerning propofol and VEP. Savoia et al. (1988) tested propofol at infusion rates of 54 and 108 μg/Kg/minute, and found no effects on any components of the Auditory Evoked Potential. Maurette et al. (1988) found that propofol slightly depresses Sensory Evoked Potential responses.

ANAESTHETICS AND VEP: SUMMARY

Table 3 summarizes the approximate effects of various anaesthetics on the VEP at varying depths of anaesthesia on humans. Some of the effects reported have been slightly contradictory, or concerned small patient groups. Briefly the effects of anaesthetics on VEP can be summarized as follows:

-Late component augmented during prenarcotic stage of barbiturate, halothane,ether and forane anaestheia. This stage is transitory preceding the depression of neuronal activity of deep surgical anaesthesia.

-Following prenarcotic stage, barbiturates, ether and halothane cause three clear changes:

a) waveform lengthened
b) the first surface positive component begins to lose amplitude (i.e. the component conveyed by classic afferent system)
c) large surface negative wave abolished (i.e. component arriving by nonspecific afferents). /Bimar and Naquet, 1966/. Similar effects have been reported by Domino with cyclopropane / Domino 1968/, but smaller effects with halothane and nitrous oxide.

- Following prenarcotic stage, chloralose, Viadril, enhance both the first surface positive and the later negative wave without any effect on latency. /Bimar and Naquet, 1966/. (Whereas ether , halothane and barbiturates block evoked responses of neurons at this level chloralose, and Viadril, augment and diffuse the response)

-Enflurane at large concentrations can enhance the amplitude of the late component

Table 3. Summary of the approximate effects of various anaesthetics on the amplitude and latency of the P100 component of the VEP at mild, moderate and deep anaesthesia depths.

	P100				
	amplitude, depth of anaesth. ⇒			latency, depth of anaesth. ⇒	
isoflurane	0	⇓		⇑	⇑
isoflurane and nitrous oxide	0	⇓		⇑	⇑
ether	⇑	0	⇓	0	⇑
forane	⇑	0	⇓	0	⇑
halothane	⇑	0	⇓	0	⇑
enflurane	⇓	⇓	⇑	0	⇑
nitrous oxide	⇓	⇓		0	⇑
chloralose	0	⇑		0	0
Viadril	0	⇑		0	0
cyclopropane	0	⇓		0	⇑
droperidol	0	0		0	⇑
fentanyl	0	0		0	⇑
fentanyl-nitrous oxide	0	0		0	⇑
pentobarbital	0	0		0	0
thiamyl sodium	⇑	⇓	⇓		
thiopentone		⇓			⇑
Valium	⇑	0	⇓	0	⇑

symbol explanation:

0 no effect

⇑ / ⇓ mild increase/decrease of amplitude/latency

⇑ / ⇓ moderate increase/decrease of amplitude/latency

⇑ / ⇓ large increase/decrease of amplitude/latency

OTHER FACTORS INFLUENCING INTRAOPERATIVE VEPs

In addition to the anaesthetic agent used, there are a host of other factors that influence the intraoperative VEPs. Among these are:

Age:

VEP latencies are substantially longer at very young ages, and the waveform somewhat simplified. Intraoperative VEPs have been successfully recorded from infants and children (Albright and Scalabassi, 1985; Reilly et al. 1978). Elderly patients have also been successfully monitored intraoperatively (Russ et al., 1984).

Temperature:

Hypothermia can attenuate VEP amplitudes and increase latencies (Figure 7). Temperatures down to 25 C were tested by Russ et al. (1984); by about 30 C latencies had increased by about 25%. At about 25 C the VEPs became too attenuated to record. On rewarming the VEPs reappeared. Reilly et al. (1978) recorded VEPs among children undergoing cardiac surgery; temperatures down to 20 C were tested. The latencies increased by approximately 5%/C and the amplitudes were attenuated by approximately 8%/C. The results also suggested that the VEP was more sensitive than the EEG to hypoxia and hypothermia.

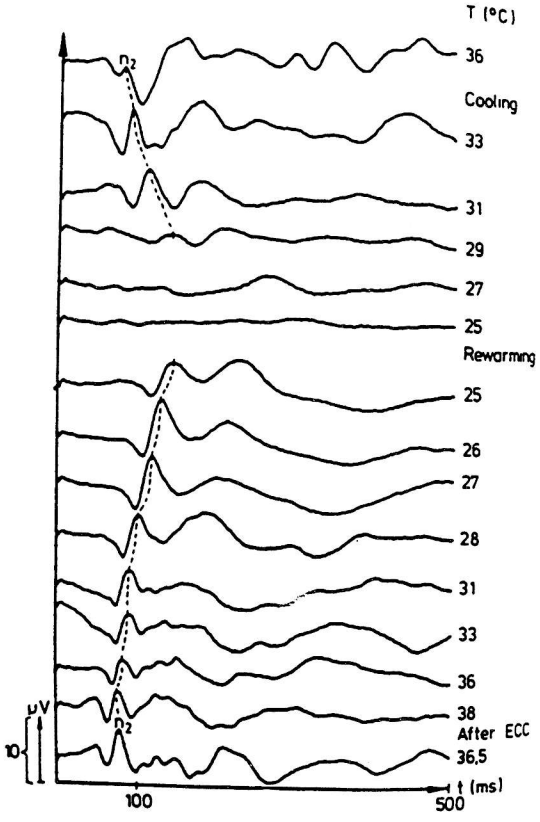

Figure 7. Effect of temperature on VEP. Cooling was relatively rapid (0.6 C/min). The first negative peak is marked with a dotted line / Russ et al., 1984/.

Anemia and Hypoxia:

Nagao et al. (1978) studied the effect of a very low hematocrit in anaesthetized baboons. A gradual amplitude increase in SEPs and VEPs occurred, which was interpreted as to be due to anoxic activation. At hematocrits below 10%, the SEPs and VEPs showed significant increases in all latencies and attenuation in amplitudes. Minowa and Hayashida (1983) studied cats, and as hematocrits fell below 30%, the VEPs and long-latency BAEPs demonstrated a clear accentuation of the amplitudes of the early peaks. With hematocrit values below 15% progressive loss of EP amplitudes was seen. Intraoperatively then, a modest increase in VEP

amplitude could be attributable to anoxic activation rather than to an improving nervous system function.

Blood pressure:

Smith (1975) used deliberated hypotension as part of his surgical approach to certain ophthalmological problems. His results indicate that there is little change in VEP down to blood pressures of 80 mm Hg. Below that level, there is a progressive fall in amplitude. At a brachial blood pressure of 50 mm Hg, the VEP is still present. It could be abolished at that point by firm pressure on the optic nerve.

RELIABILITY OF INTRAOPERATIVE VEPS

The use of intraoperative VEP monitoring is not yet widespread. One reason is undoubtedly the relative unreliability of intraoperative VEPs that some investigators have reported. Several small series of patients have reported reversible changes in intraoperative VEPs which were temporarily related to manipulation of the optic nerve or chiasma / Feinsod et al., 1976; Allen et al., 1981; Costa de Silva et al., 1985/. In one of the largest series of patients published, intraoperative VEP's showed a relatively high rate of positive and false negative changes due to excess amplitude and latency variability /Raudzens, 1982/.

CONCLUDING REMARKS

Most of the studies concerning the effects of anaesthetic agents on VEP have consisted of relatively small groups of patients. It is also unclear as to what extent the effects of burst suppression are taken into account during VEP monitoring. For example some patients are driven to burst suppression at 1.8% end tidal concentrations of isoflurane: if the VEP is summed during the suppression stage as well, it is obvious that the overall VEP amplitude will be diminished without concurrent effects on the latency. Chi and Field (1986) recorded VEPs down to isoflurane concentrations of 1.8% end-tidal concentrations in their study, but no mention is made of how the possible effects of burst suppression were taken into account (if at all).

It is more than likely that improvements in monitoring methods and proper understanding of the effects of anaesthetic agents on the EEG and VEP will in the future increase the reliability and usefulness of intraoperative VEP monitoring.

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