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Effects of Thiopentone Halothane-Nitrous Oxide Anaesthesia Compared to Ketamine-Xylazine Anaesthesia on the DC Recorded Dog Electroretinogram

By *B. Kommonen, U. Karhunen* and *C. Raitta*

Kommonen B., U. Karhunen and C. Raitta: Effects of thiopentone halothane-nitrous oxide anaesthesia compared to ketamine-xylazine anaesthesia on the DC recorded dog electroretinogram. Acta vet. scand. 1988, 29, 23-33. – Eleven ophthalmoscopically healthy dark adapted dogs were examined by DC ERG technique with single flash full field illumination starting with near b-wave threshold blue (tests 1-3) and white (tests 4-6) stimuli of different intensity and ending with 30 Hz photopic flicker smuli (test 7) after light adaptation. All animals were anaesthetized using 2 different anaesthetic methods: Anaesthesia I (A I): Induction with thiopentone sodium, continued with halothane and nitrous oxide in oxygen. Anaesthesia II (A II): Praemedication with xylazine hydrochloride followed by anaesthesia with ketamine hydrochloride. A minimum interval of 1 week was kept between all anaesthesias.

The a- and b-wave amplitudes and latencies were determined. Statistical analysis of results indicated that the a- and b-waves were elicited by weaker intensities in A II. In Tests 3-6 the a-wave was highly significantly ($P < 0.001$), higher in amplitude in A II than in A I. Differencies in b-wave amplitudes were not statistically significant (except Test 1). The b-wave latencies were longer in A I in Test 2 (using low intensity blue light). The a-wave latencies were slightly shorter in A II in Test 6 (using high intensity white light).

In additional experiments the selective action of the different agents (except N_2O) used in A I and A II was studied. Thiopentone alone given to 3 dogs seemed to depress the a-wave selectively.

Halothane given separately to 3 dogs lowered both the a- and b-wave amplitudes.

Ketamine given with a neuromuscular blocking agent to three dogs resulted in responses almost identical to those in A II.

Xylazine with vecuronium given to 4 dogs resulted in responses with slighly depressed a- and b-waves in comparison to ketamine with vecuronium.

The results indicate that when developing an animal model for the electrophysiologic study of human retinal dystropies, the actions of different anaesthetics upon the ERG components are of great importante.

rod and cone responses: canine.

Introduction

Anaesthetic and hypnotic agents usually have a diminishing action on the evoked potentials of the central nervous system. In the retina the selectively depressant effect of rising concentrations of aether on the electroretinogram components has been used in detec-

ting 3 different reproducible steps, identified as a-, b- and c-waves (*Granit* 1933). The ERG a- and b-wave amplitudes in humans were lowered using halothane (*Raitta et al.* 1979) but not by enflurane (*Raitta et al.* 1982) anaesthesia.

Stable reliable single flash retinal responses near threshold values can be obtained from phylogenetically high standing animals such as the dog only under anaesthesia with or without neuromuscular blockade. Therefore, when developing an animal model for the study of human retinal dystrophies the actions of different anaesthetics upon the ERG components are of great importance. The results can be drug and dosage dependant. Rising end-tidal concentrations of halothane lowered the ERG c-wave in Beagles and English setters (*Dawson et al.* 1983). Similar effects of halothane, trichloretylene and methylchloroform have been reported in the DC recorded c-wave of the cynomolgus monkey (*Jarkman et al.* 1985). The a-wave amplitude in rabbits anaesthetized with methoxyflurane, halothane and enflurane decreased in a dose dependant manner (*Tashiro et al.* 1986).

The present investigation was undertaken in order to find out which of the two clinical anaesthetic methods: Thiopentone/halothane/nitrous oxide in oxygen or ketamine/xylazine with oxygen is better suited for the recording of the canine ERG rod and cone response, and to find out if the pharmacologically different drugs might act selectively when given separately.

Material and methods

Eleven healthy ophthalmoscopically normal dogs were examined. Characteristics of the dogs are presented in Table 1. All dogs were premedicated with glycopyrrolate 0.01 mg/kg (Gastrodyn® 0.2 mg/ml, Medica, Finland) intramuscularly 15-20 min before induction of the anaesthesia which was performed at minimum intervals of 1 week.

Table 1. Characteristics of the dogs used in the study.

Number of dogs	Breed	Age (years)	Weight (kgs)	Eye
7	Beagle	1¼-1¾	9-12.5	7 Left
2	Beagle	8	8.5-12	2 Left
2	Beagle/ Finnish harrier	3	12-14	2 Right

Anaesthesia I (Thiopentone/halothane-/nitrous oxide)

Anaesthesia was induced with a mean dose of 16.0 mg/kg thiopentone sodium (range 14-19 mg/kg) (Pentothal® Natrium, Abbott, Italy) in the cephalic vein. After tracheal intubation the dogs breathed spontaneously or ventilation was manually assisted by using 1.5% halothane (Trothane®, ICI Chemicals, England) (7 dogs) - 0.5% halothane (4 dogs) in 3% oxygen with nitrous oxide. This method is referred to as Anaesthesia I (A I). Arterial blood gas analyses were performed immediately after ERG recordings on 8 of the dogs.

Additionally 3 of the dogs were anaesthetized by thiopentone sodium 23 mg/kg intravenously. The ventilation of the tracheally intubated dogs was manually assisted or they breathed spontaneously. Arterial blood gas analyses were made immediately after the ERG recordings of all 3 dogs.

Anaesthesia of 3 other dogs was induced by using 4% halothane in oxygen using a conical face mask, followed by tracheal intubation. Anaesthesia was maintained by 0.5% halothane in oxygen during ERG recordings.

Anaesthesia II of the same 11 dogs (Xylazine/ketamine)

Xylazine hydrochloride 1 mg/kg (Rompun® Vet. 20 mg/ml, Bayer, Germany) was administered intramuscularly in the neck of the dogs. Ten min. later ketamine hydrochloride 10 mg/kg (Ketalar® 50 mg/ml, Parke Davis, UK) was injected intramuscularly in the neck. The dogs breathed spontaneously and were given oxygen at a rate of 3 liters/min through a loosely fitted conical face mask. This method is referred to as Anaesthesia II (A II). Blood gas analyses were performed on 3 of the dogs.

In addition 4 of the dogs were given xylazine hydrochloride 2-3.8 mg/kg intravenously followed by vecuronium bromide 0.17 mg/kg (Norcuron® 4 mg/ml, Organon, Holland).

The dogs were preoxygenated through a conical face mask, tracheally intubated, and their ventilation was manually controlled. After EFG recording the neuromuscular blockade was antagonized by neostigmine with atropine.

Three of the dogs were anaesthetized by using ketamine hydrochloride 8 mg/kg intravenously. Tracheal intubation was facilitated by using vecuronium bromide 0.17 mg/kg and the dogs were manually ventilated with oxygen. Again the neuromuscular blockade was reversed after ERG recordings.

Stimulating apparatus

A photostimulator (Grass PS 22, Grass Instruments, Quincy, Mass., USA) with a xenon flashlamp, flash duration 10 μs, with intensity settings of 1, 2, 4, 8 and 16 was used. During all experiments only the strongest intensity was used and the intensity was lowered by neutral density filters and/or blue filters (Kodak Wratten neutral density filters and Kodak Wratten 47B filter, Eastman Kodak Co, Rochester, NY. USA). According to the manufacturer, the Xenon flashlamps intensity, when measured from its pebbled plexiglas face plate on the axis of its parabola at intensity setting 16 was 0.024 lumen-seconds per square centimeter.

At a flash rate of 30 Hz the manufacturer states that the relative intensity value is lowered from 16 to approximately 10.

In order to obtain near uniform full-field illumination of the retina under standardized stimulus conditions the xenon flashlamp was attached via a filter holder to a globe (LKC Systems inc., Gaithersburg, Maryland, USA) with a diameter of 406 mm. The globe was coated inside with white reflective paint (Eastman Model 6080).

Electrodes and recording apparatus

The ocular potentials were measured by a cotton wick AG-AgCl electrode attached to a plastic lid retractor. The metallic part of the electrode was completely covered by black plastic. A similar type of electrode has been described (*Sieving et al.* 1979). Ground and reference electrodes were Ag-AgCl wires that were inserted through side holes near the conus of 1 ml disposable syringes. The insertion holes for the wires were sealed using black silicone rubber. Electrical contact was achieved by 0.9% saline in the syringes of which a small amount was injected subcutaneously through a disposable 21 gauge hypodermic needle. The reference electrode needle was places 2 cm temporal from the temporal canthus of the eye to be examined. The ground electrode needle was places in the neck.

The potentials were led via shielded wires to a specially built low noise DC differential preamplifier with a band width from 0-1 KHz. Drift was not significant at the registration times of 200 ms used in this investigation. A feature of the preamplifier was

that the base-line was automatically brought to zero before recordings. The amplification was 1000. The potentials were displayed on a dual beam digital storage oscilloscope (Gould 1425, Gould, Hainault, Essex, England). The sweep time in all recordings was 20 ms/cm. A calibration check indicating 100 μV vertically and 20 ms horizontally was built in to the preamplifier. The oscilloscope was triggered by the stimulator. The oscilloscope could be triggered before the flash. The oscilloscope was set at roll mode which allowed the steadiness of the base-line to be judged before each flash. The amplitudes were measured by cursors from the base-line. The latencies were measured from the stimulus moment to the highest point of the different waves. After the data had been copied (by hand) from the oscilloscope screen the stored waveforms were taken from the oscilloscope memory and printed by a penwriter (Servogor 120, BBC, Goerz Metrawatt, Goerz Electro G.m.b.H. Sonnleithnergasse 5, A-1101 Vienna, Austria) at a speed of 10mm/2s using the same amplitude and time calibration as in the oscilloscope, resulting in identical curves on the paper and on the screen. The selected writing speed was slow in order to eliminate distortion of the curves.

The electrophysiologic testing in detail

The dogs were kept in dimly lighted dog cages for at least 1 h before the examination. The pupils were dilated by tropicamide eyedrops (Oftan Tropicamid® 5 mg/ml, Star, Finland) for A II and additionally by cyclopentolate hydrochloride eyedrops (Oftan Syklo® 5 mg/ml, Star, Finland) for A I. The dark adaptation was started immediately after application of the mydriatics and premedication. The dark adaptation time in A I ranged from 31 to 58 min (mean 44 min) and in A II from 37 to 50 min (mean 42,5 min) Oxybuprocaine hydrochloride eyedrops (Oftan Obucain® 4 mg/ml, Star, Finland) were instilled in the eye. A bridle suture was placed in the ventral rectus muscle or in the episclera in order to prevent ventronasal rotation of the bulb. This was not needed in A II nor when the neuromuscular blocking agent was used. The dogs were placed in a lateral position on a rubber surfaced table with their head on a padded styrofoam stand so that the nose formed an approximately 40° angle with the table surface when the dog's head was viewed dorsally. The aperture of the spherical illuminator was directed downwards and it partly enclosed the raised head of the anaesthetized dog when the illuminator was lowered to a standard position. The eye to be examined was placed in the estimated center of the aperture with the cornea about five centimeters inside the aperture. Recordings under different anaesthesias were always made on the same eye of the individual dog.

In all anaesthesias the stimulus intensities and suquence were identical. The stimulator was set at the highest intensity, which was lowered by filters. Six single flashes of different intensity were delivered at 1½ min intervals. The first stimulus elicited by the stimulator was attenuated by a neutral density, ND 1, a ND 2 and a Kodak Wratten 47B blue filter. This intensity resulted in a small b-wave in A II in seven of the dogs. Test flash No 2 was 2 log units higher in intensity. This was achieved by removing the ND 2 filter from the filter frame. Test flash No 3 was delivered after the ND 1 filter had been replaced by a ND 0.6 filter. Before the fourth test flash the KW 47B filter was replaced by a ND 2 filter and the ND 0.6 filter was removed. The fifth test flash was given using only the ND 1 filter and the sixth flash ws delivered without filters. Thereafter the background light of the spherical illuminator was turned on with the largest possible aperture of ⅓. Ten se-

Table 2. Intensity of the different testflashes.

Test (Flash) No:	Filters used	Log relative units above Test 1
1	ND 1, ND 2, KW 47 B (blue)	0
2	ND 1, KW 47 B	2
3	ND 0.6 KW 47 B	2,4
4	ND 2	1,7
5	ND 1	2,7
6	-	3,7
7	-	2,3

conds later a 30 Hz flicker stimulus without filters was delivered. The relative intensities of the testflashes, referred to as tests 1-7, are shown in Table 2.

Statistical analysis of results was performed only in Anaesthesia I and II.

Results

Anaesthesia I and II (Figs 1 and 2)

The nature of the distributions of 11 observations were in most cases quite irregular and statistics pertaining to the normal distribution could not be applied generally. Non-parametric statistics of central tendency-median and that for dispersion quartile difference halved (d) was used in the description of distribution of observations.

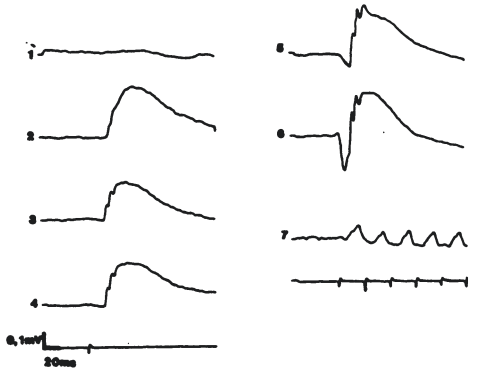

Figure 1. Electroretinograms from one of the dogs in Anaesthesia I. The a-wave is practically absent in tests 2, 3 and 4. A relatively low a-wave is seen in tests 5 and 6. The stimulus moment is indicated under the recordings.

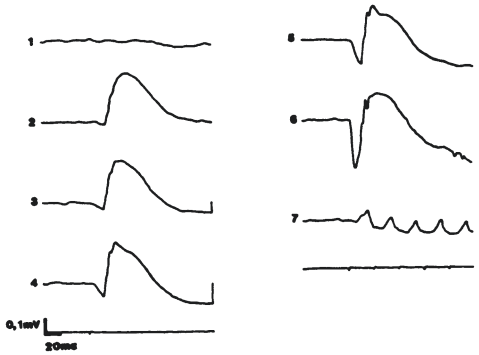

Figure 2. Electroretinograms of the same dog as in Fig. 1 in Anaesthesia II made under identical stimulus and dark adaptation contitions. A well developed a-wave is seen in tests 2, 3 and 4. The a-waves of tests 5 and 6 are also clearly higher in amplitude than in Anaesthesia I. The shorter latency of the b-wave in test 2 when compared to A I is apparent.

Test 1. In A I only 2 dogs responded with weak b-waves. In A II seven dogs, including the same 2 dogs in A I responded with b-waves which in all cases were greater in amplitude than in A I.

According to the sign test the direction of differences had become statistically verified with a low level of significance ($P < 0.05$).

Test 2. In A I only 4 dogs had an a-wave, but in A II all dogs responded with an a-wave. One of the dogs had a higher amplitude a-wave in A I. The Wilcoxon test for pair differences produced a statistically significant P-value ($P < 0.01$).

The median for amplitudes of a-waves was 14 μV in A II and the above mentioned measure of dispersion d was 9.25 uV.

The b-wave amplitudes in both anaesthesias were similar in their main features.

A I Range 52 - 325 μV,
Median 147 μV, d = 88 μV
A II Range 112 - 315 μV,
Median 227 μV, d = 68 μV

A II gave larger amplitudes with smaller dispersion.
Latencies were shorter in A II. The statistics were:
A I Range 47 - 64 ms,
Median 56 ms, d = 3.5 ms
A II Range 42 - 60 ms,
Median 47 ms, d = 6.0 ms
When comparing latencies by dogs in ten comparisons latencies were shorter in A II and in one case they were the same. According to the Wilcoxon Test for pair differencies, the tendency of shorter latencies in A I were statistically significant ($P < 0.01$).

Test 3. All the a-wave amplitudes in A II were larger than the highest in A I. In the latter, three dogs did not display a visible a-wave. According to the Wilcoxon Test for two independent sets of observations the difference in levels of these sets was statistically significantly high ($P < 0.001$).
Statistics for a-wave amplitudes were:
A I Range 0 - 24 μV,
Median 3.5 μV, d = 10.5 μV
A II Range 25 - 112 μV,
Median 45 μV, d = 10.5 μV
Latencies in A I were between 13 and 21 ms and in A II between 15 and 19 ms.
Statistics for amplitudes of b-waves were:
A I Range 73 - 318 μV,
Median 140 μV, d = 58 μV
A II Range 66 - 352 μV,
Median 175 μV, d = 61 μV
The distributions of amplitudes above were fairly similar.
The statistics for the latencies were:
A I Range 38 - 56 ms,
Median 46 ms, d = 6.5 ms
A II Range 33 - 57 ms,
Median 40 ms, d = 4.0 ms
No essential differencies could be detected between sets.

Test 4. The distributions of a-waves in A I and II had the same typical differencies as in test 3 both in amplitudes - and latencies.
Statistics for amplitudes:
A I Range 0 - 38 μV,
Median 10 μV, d = 5 μV
A II Range 35 - 87 μV,
Median 49 μV, d = 9 μV
Statistics for amplitudes of b-waves were:
A I Range 52 - 287 μV,
Median 136 μV, d = 65 μV
A II Range 77 - 374 μV,
Median 192 μV, d = 75 μV
The level of amplitudes in A II seemed to be somewhat higher, but this difference might be random.
Results for latencies were:
A I Range 35 - 63 ms,
Median 40, d = 5.0 ms
A II Range 31 - 50 ms,
Median 39, d = 4.8 ms
With the exception of one extreme value of 63 in the latter set - the next was 49 - the distributions were similar.

Test 5. Again all amplitudes of a-waves in A II were higher than the highest in A I. Statistics:
A I Range 17 - 77 ms,
Median 59 ms, d = 21 ms
A II Range 80 - 196 ms,
Median 147 ms, d = 18 ms
Statistics for latencies of the a-waves:
A I Range 12 - 20 ms,
Median 15 ms, d = 1.5 ms
A II Range 13 - 16 ms,
Median 14 ms, d = 1.5 ms
Statistics for amplitudes of b-waves:
A I Range 63 - 297 μV,
Median 133 μV, d = 68 μV
A II Range 56 - 318 μV,
Median 143 μV, d = 49 μV
Latencies
A I Range 26 - 41 ms,

Median 33 ms, d = 5 ms
A II Range 29 - 57 ms,
Median 32 ms, d = 2.5 ms
Except for a high latency value with a gap in A II, there were no essential statistical differencies between sets.

Test 6. In this series of observations all of the amplitudes in A II were not higher for a-waves than the highest for A I. However every dog had a higher amplitude in A II than in A I. This regular feature on the basis of sign-test was statistically highly significant ($P < 0.001$).
The statistics were:
A I Range 87 - 203 μV,
Median 129 μV, d = 32 μV
A II Range 164 - 343 μV,
Median 276 μV, d = 48 μV
The latencies varied in A I within the range 7-8 ms and in A II the range was 7-10 ms. For 4 dogs the latencies were the same, but in A II they were shorter for 7 dogs. The shortening of latencies in A II had thus been verified by low statistical significance.
Statistics for b-waves were:
A I Range 49 - 297 μV,
Median 112 μV, d = 39 μV
A II Range -3.5 - +311 μV,
Median 140 μV, d = 28 μV
Characteristics for distributions of latencies:
A I Range 24 - 39 ms,
Median 32 ms, d = 5 ms
A II Range 26 - 47 ms,
Median 34 ms, d = 5 ms
Differencies by dogs varied in directions both for amplitudes and latencies of b-waves.

Test 7. The 30 Hz photopic flicker responses under the two anaesthesias were as follow:
A I amplitude Range 20 - 80 μV,
Median 40 μV, d = 10 μV
A II amplitude Range 50 - 110 μV,
Median 70 μV, d = 5 μV
The difference was statistically significantly verified ($P < 0.01$) according to the Wilcoxon Test for pair differences.
A I latencies Range 22 - 28 ms,
Median 24 ms, d = 1.0 ms
A II latencies Range 22 - 24 ms,
Median 22 ms, d = 0.5 ms

Selective action of the anaesthetics
Effects on a- and b-wave amplitudes when thiopentone, halothane, ketamine with vecuronium and xylazine with vecuronium when given separately are shown in Figs. 7 and 8.
Examples of typical recording from dogs under thiopentone (Fig. 3), halothane (Fig. 4), ketamine with vecuronium (Fig. 5) and xylazine with vecuronium (Fig. 6) show the responses elicited by the testflashes.

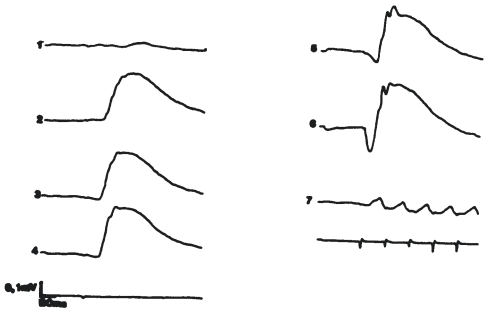

Figure 3. Electroretinograms of a dog under thiopentone solely. Low amplitude a-waves are seen in tests 2-6. The b-waves in tests 1-6 are relatively high, whereas the flicker responses are low (test)).

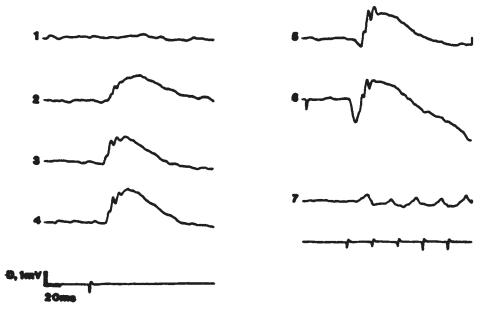

Figure 4. Electroretinograms from the same dog as in Fig. 1 and 2 under halothane solely. Both the a- and b-waves amplitudes are low. Flicker responses have low amplitudes.

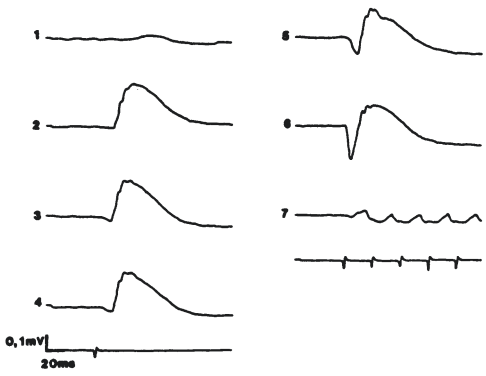

Figure 6. Electroretinograms from the same dog as in Figs. 1, 2 and 4 under xylazine with vecuronium. The a-wave is slightly lower than in Anaesthesia II or in ketamine with vecuronium, but somewhat higher than under thiopentone alone. The b-wave is slightly lower than in Anaesthesia II.

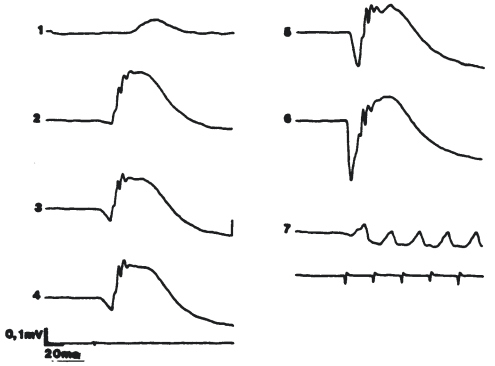

Figure 5. Electroretinograms from the same dog as in Fig. 3 under ketamine with vecuronium. High a-wave amplitudes in tests 3-6 in relation to other anaesthesias. The b-wave amplitudes are also relatively high in test 1-6. Flicker responses show high amplitudes.

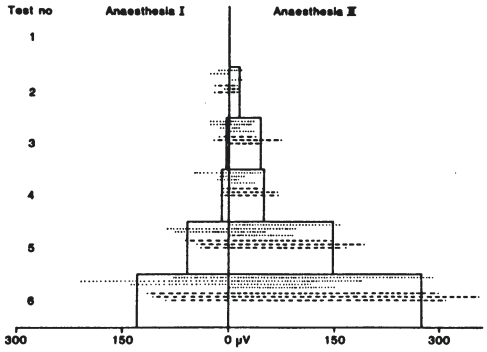

Figure 7. Medians of a-wave amplitudes from recordings of 11 dogs in Anaesthesia I (thiopentone-/halothane/nitrous oxide) and Anaesthesia II (ketamine/xylazine) (horizontal columns) at different stimulus intensities (Test 1-6). Amplitudes from 3 dogs anaesthetized with thiopentone solely (dotted lines) and halothane solely (broken lines) are shown to the left. Amplitudes from 4 dogs anaesthetized by xylazine with vecuronium (dotted lines) and from 3 dogs anaesthetized by ketamine with vecuronium (broken lines) and shown to the right.

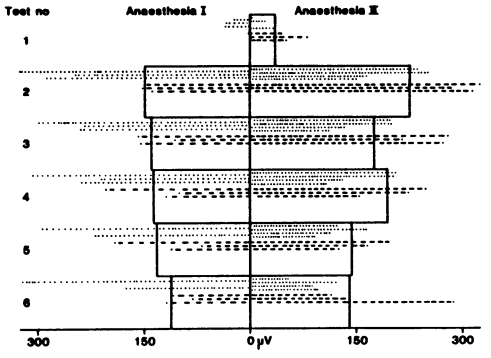

Figure 8. Medians of b-wave amplitudes from recordings of 11 dogs in Anaesthesia I (thiopentone-/halothane/nitrous oxide) and Anaesthesia II (ketamine/xylazine) (horizontal columns) at different stimulus intensities (Test 1-6). Amplitudes from 3 dogs anaesthetized with thiopentone solely (dotted lines) and halothane solely (broken lines) are shown to the left. Amplitudes from 4 dogs anaesthetized by xylazine with vecuronium (dotted lines) and from 3 dogs anaesthetized by ketamine with vecuronium (broken lines) are shown to the right.

The reactions to Test 3 in thiopentone alone tested on 3 dogs were as follows:

a-wave		b-wave	
amplitude (μV)	latency (ms)	amplitude (μV)	latency (ms)
14	16	301	47
28	18	238	58
28	18	241	38

The reactions to Test 3 in halothane alone tested on three dogs were as follows:

a-wave		b-wave	
amplitude	latency	amplitude	latency
14	15	157	33
21	19	108	44
-	-	154	44

The corresponding responses from three dogs under ketamine with vecuronium were:

a-wave		b-wave	
amplitude	latency	amplitude	latency
38.5	15	280	28
73	15	217	29
45.5	17	273	31

The responses for xylazine with vecuronium were as follows:

a-wave		b-wave	
amplitude	latency	amplitude	latency
35	16	199	40
31	15	199	30
14	17	133	35
35	16	112	28

Arterial blood gas analyses in A I showed that the dogs tended to develop slight hypercapnia. Four dogs had PCO_2 values of 39, 40, 43 and 45 mmHg, three dogs had 52, 56 and 58 mmHg. One dog had a PCO_2 of 85 mmHg. This individual did not recover until 2 h after anaesthesia. The cause of this remained unknown. This dog's retinal responses did not differ from the responses of others.

The blood gas analyses from three dogs in A II showed PCO_2 values of 38, 44 and 47 mmHg. During thiopentone anaesthesia the PCO_2 values of three dogs were 39, 53 and 54 mmHg. The PO_2 values in all samples from 14 anaesthesias were high. No obvious correlation between these randomly taken PCO_2 and PO_2 values and the results of the recordings could be detected on the basis of this study.

Discussion

Analysis of the effects of anaesthetics on the light induced action potentials of the retina have not been well described in the literature. It can be assumed that anaesthetics affect the retinal cells in different ways. Knowledge of the action of drugs on the retinal responses in humans is important for electroretinograms in children and incooperative patients. In the present study two different anaesthetic methods have been investigated.

The results showed a highly significant decrease of only the awave amplitude in thiopentone/halothane/nitrous oxide anaesthesia (A I) when compared to ketamine/xylazine anaesthesia (A II). The different action on a- and b-waves was particularly obvious at relatively low intensities using blue light (Test 3). The receptor potential registered in such conditions mainly represents a rod response (*Aguirre* 1975).

On the basis of the results of this investigation it can be assumed that the rods are influenced by halothane. In rabbits toxic effects on the pigment epithelium have been shown by *Johnson et al.* (1973) in long-term halothane anaesthesia. The c-wave was not recorded in the present study.

Halothane, thiopentone and xylazine combined with oxygen produced depressing effects on the a-wave amplitude when given separately to three or four dogs. The b-waves in thiopentone and ketamine anaesthesia were high at low stimulus intensities compared to halothane, which seemed to lower both the a- and b-wave. An additive influence of nitrous oxide on the responses in A I can not be excluded in the present study.

The photochemical reaction in the retinal receptors or the way it generates the a-wave is depressed by halothane and thiopentone, whereas the b-wave, which reflects neurotransmission in the bipolar layer, under thiopentone anaesthesia showed high amplitudes similar to those under ketamine anaesthesia.

These results indicate that halothane and thiopentone had a similar effect on the a-wave but were different in respect to the b-wave.

These findings show that thiopentone seems to have a selectively depressing effect on the a-wave in dogs. However, until now the experimental series were to small to allow definite conclusions.

Theoretically ketamine as the sole agent would be a good choice for ERG recordings (*Sasovets* 1978). Ketamine, however, has side effects (*Haskins et al.* 1985, 1986, *Benson et al.* 1986). Additionally involuntary eye movements and other involuntary muscular activity reduce its suitability for ERG recording. The influence of the neuromuscular blocking agent on the amplitudes and latencies when using ketamine and xylazine as sole anaesthetics could not be excluded. One additional factor that could have influenced the results was the clinical character of anaesthesia. The doses of anaesthetics needed to facilitate the recordings or to guarantee unconciousness during neuromuscular blockade varied. Hence anaesthesia can be considered more qualitative than quantitative. The dosage dependent effect of the drugs on the responses was not studied.

The significantly lowered photopic 30 Hz flicker responses in A I compared to A I indicated that the cone receptors reacted in a similar manner to the anaesthetics as did the rods.

Work is in progress to develope a combinated anaesthesia to meet the requirements for the recording of both fast and slow ERG components.

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Sammandrag

Verkan av tiopental-halotan kväveoxid anestesi jämfört med ketamin-xylazin anestesi på likströmsförstärkt elektroretinogram hos hund.

Elva oftalmoskopiskt friska mörkeradapterade hunder undersöktes med hjälp av likströms ERG med enskilda blixtar som reflekterades över hela synfältet började med blått ljus nära b-vågens retningströskel (test 1-3), följda av vitt ljus (test 4-6) och 30 Hz fotopiskt flicker (test 7). Alla hundar sövdes ner med två olika anestesimetoder: Anestesi I (A I) Induktion med tiopental-natrium, följt av halotan och kväveoxid med syre. Anestesi II (A II): Premedikation med xylazin-hydroklorid följd av anestesi med ketaminhydroklorid. Tidsintervallen mellan experimenten var minst en vecka.

A- och b-vågornas amplituder och latenser mättes. Statistisk analys av resultaten indikerade att a- och b-vågorna framkallades av lägre stimulus intensiteter i A II. I test 3-6 hade a-vågen en statistiskt högt signifikant ($P < 0.001$) större amplitud i A II än i A I. Skillnaderna i b-vågornas amplituder var inte statistiskt signifikanta (utom i test 1). B-vågens latenser var längre i A I i test 2 (blått ljus av låg intensitet). A-vågens latenser var lite kortare i A II i test 6 (vitt ljus av hög intensitet). I tillägsexperiment undersöktes de olika preparatens (utom N_2O) Selektiva verkan på amplituderna och latenserna. Då enbart tiopental gavs åt tre hundar sänktes a-vågen selektivt varemot enbart halotan givet åt tre hundar sänkte både a- och b-vågornas amplituder. Ketamin applicerat tillsammans med ett muskelrelaxans åt tre hundar resulterade i nästan likadana responser som i A II. Xylazin med muskelrelaxans givet åt fyra hundar resulterade i responser med lite lägre a- och b-vågor än ketamin med muskelrelaxans.

Resultaten indikerar att verkan av olika anestetika på ERG komponenter är av stor betydelse vid utvecklandet av en djurmodell för undersökning av humana näthinnedystrofier.

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