The electroretinogram during orbital compression following intraorbital regional block for cataract surgery

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The electroretinogram (ERG) is a transient biopotential that reflects the electrical response of the distal retina to photostimulation. Disturbances in retinal circulation produce characteristic abnormalities in the ERG wave form. The objective of this study was to investigate the changes in the ERG produced by combined retrobulbar and peribulbar injections of a large volume (8 ml) of local anaesthetic, followed by ocular compression. Electroretinogram recordings were obtained from skin electrodes placed on the infero orbital ridge in response to stroboscopic flash stimulation in 34 adult patients undergoing cataract surgery: (a) prior to regional anaesthesia (baseline condition); (b) within one minute after regional anaesthesia of the orbit (block condition); (c) after ten minutes of orbital compression with a Honan's device at 30 mmHg. (compression condition); (d) and five minutes after removal of orbital compression (recovery condition). The ERG implicit times of both a- and b-wave increased ($P < 0.001$) after anaesthetic block. The amplitude of the a- and b-waves also decreased ($P < 0.001$) immediately following anaesthetic block and continued to decrease following application of the compression device ($P < 0.01$). Following removal of ocular compression the amplitude of the b-wave increased ($P < 0.01$). Only the a-wave implicit

time ($P < 0.005$) decreased with release of ocular compression. These findings are compatible with the ERG changes of transient retinal ischaemia produced by ocular compression.

L'électrorétinogramme (ERG) est constitué par un biopotential transitoire qui reflète la réponse de la rétine distale à la photostimulation. Les dérangements de la circulation de la rétine produisent des anomalies caractéristiques de la forme d'onde de l'ERG. Cette étude vise l'examen des changements de l'ERG produits par l'injection rétrobulbaire et péribulbaire de grands volumes (8 ml) d'anesthésiques locaux suivie de compression oculaire. En réponse à des stimulations lumineuses intermittentes, on enregistre l'électrorétinogramme à partir d'électrodes cutanées placées sur le rebord orbital inférieur de 34 adultes soumis à une chirurgie pour cataracte: a) avant l'anesthésie régionale (condition de base); b) dans la minute qui suit la réalisation de l'anesthésie régionale de l'orbite (condition de bloc); (c) après dix minutes de compression avec un appareil de Honan à 30 mmHg (condition de compression); d) et cinq minutes après l'arrêt de la compression (condition de récupération). La période de latence des ondes a et b de l'ERG augmente ($P < 0,001$) après le bloc. L'amplitude des ondes a et b décroît ($P < 0,001$) immédiatement après l'application de l'appareil de compression ($P < 0,01$). Après le retrait de l'appareil de compression oculaire l'amplitude de l'onde b augmente ($P < 0,01$). Seule la période de latence de l'onde a ($P < 0,005$) diminue avec le relâchement de la compression oculaire. Ces constatations sont compatibles avec les changements qui surviennent à l'ERG lors d'une ischémie rétinienne transitoire causée par compression oculaire.

Key words

ANAESTHESIA: ophthalmic;
ANAESTHESIA TECHNIQUES: regional: retrobulbar;
MEASUREMENT TECHNIQUES: electroretinography;
MONITORING: evoked potential.

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Accepted for publication 14th May, 1994.

The electroretinogram (ERG), recorded from the cornea in response to brief flash stimulation, has two primary components, the "a-wave" and "b-wave" (Figure 1) which appear to reflect synaptic and post-synaptic activity in the inner nuclear layer, distal to the ganglion cell body layer. Disturbances in retinal circulation produce revers-

FIGURE 1 Representative flash ERG waveform. This illustrates the a- and b-wave components recorded to brief stroboscopic flash stimulation. Stimulus flash is indicated by the downward arrow. The implicit time periods from stimulus onset to the peak of the a-wave and b-wave components is illustrated.

ible abnormalities in the a- and b-waves of the flash ERG during acute retinal ischaemia in rabbits produced by increased intraocular pressure (IOP).^{1,2} Similar ERG abnormalities have been documented in humans with chronic retinal ischaemia of diabetic retinopathy.³ Kothe concluded from study of the retinal vascular perfusion pressure using ERGs that "the normal operation of the generators of the ERG is dependent upon the retinal vascular perfusion pressure (RVPP) being maintained within some ideal and narrow range."⁴

Intraorbital regional anaesthesia is associated with decreased ocular perfusion in humans.⁵ Retrobulbar anaesthesia has been implicated in ischaemic complications such as central retinal vascular occlusion, optic atrophy and ischaemic optic neuropathy following cataract surgery.^{6,7} An oculocompressive device or digital massage is usually applied after administration of intraorbital regional block⁸ for variable periods to improve the surgical conditions by reducing IOP and intraorbital tissue pressure. Transient increases in IOP and compression of the extraocular circulation, in particular the ciliary arteries,⁹ may cause a decrease in ocular perfusion. In addition, amide local anaesthetics impair relaxation of porcine ciliary arteries caused by bradykinin.¹⁰ Bradykinin, which is formed in the blood by activation of the kinin system, is one of the most potent agonists for endothelium-dependent relaxation of human ocular arteries.¹¹ Interference with this local vascular control mechanism, which is nitric oxide-dependent, may contribute to the decrease in ocular perfusion seen during intraorbital regional anaesthesia. If microvascular alterations result in venous stasis or thrombosis of retinal veins, then permanent impairment of vision can occur. Fluorescein angiography demonstrates small branch vein occlusions in

some patients with unexpected vision loss following cataract surgery.* In this study we describe the effects of local intraorbital anaesthesia and orbital compression on the ERG, the neurophysiological correlate of electroretinal function.

Methods

This study was approved by The University of Calgary Conjoint Medical Ethics Committee. Written informed consent was obtained from 34 adult patients (7 men, 27 women) scheduled for elective intraocular surgery and requiring regional anaesthesia of the orbit.

Regional block technique

Anaesthetic solution was injected in two locations, one inferotemporal to the globe (retrobulbar and within the rectus muscle cone) and the other medial to the globe (peribulbar). The anaesthetic solution consisted of bupivacaine 0.375% and lidocaine 2% with hyaluronidase 2.5 U · ml⁻¹. Four ml of anaesthetic solution were injected into each location.

The retrobulbar injection was made with a 27-gauge 31-mm disposable needle. The tip of the needle was introduced from the inferotemporal fornix of the conjunctiva with the needle aligned tangentially to the globe with its entry point midway between the lateral canthus and the lateral limbus. The needle was advanced to a depth of about 25 mm.

The medial pericone injection¹² was made with a 30-gauge 25-mm disposable needle. The needle tip was introduced on the medial side of the caruncle with the needle aligned perpendicular to the frontal plane of the face and five degree toward the medial wall of the orbit. The needle was advanced to a depth of 18 mm.

ERG recording

Flash ERGs were recorded to brief (20 microsec duration) stroboscopic full field white flash stimulation (Grass Instruments PS22 stimulator) through open eyes and dilated pupils. The flash luminance was 300 candela · m⁻² and the stimulus was presented at a rate of 4.1 flash · sec⁻¹.

Resultant averaged ERGs were analyzed in the time domain. The time period from stimulus onset to the trough of the a-wave (peak implicit time of a-wave) and b-wave (b-wave implicit time) was determined from visual inspection of the ERG tracings. In addition, the amplitude of the a-wave was measured from stimulus onset to the trough of the a-wave in microvolts. The b-wave amplitude was determined from the trough of the a-wave in microvolts (Figure 1).

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Electroretinograms

Electroretinograms were recorded with platinum wire-lid-hook electrodes (Henley Medical, U.K.) placed in the lower fornix. The ipsilateral earlobe was used as a reference site and the patient was grounded through the forehead. Electroretinograms were differentially amplified and bandpass filtered between 0.1 and 100 Hz on a Cadwell Excel (Caldwell Laboratories, Kennewick, Washington) signal averaging system and 50 artifact-free sweeps of 150 msec duration were sampled to produce each average ERG. Data acquisition was usually completed within 90 sec. Resultant averaged ERGs were analyzed in the time domain. The peak implicit times (time from stimulus to peak of the wave) of the a-wave and b-wave were determined from inspection of the ERG tracings.

Electroretinogram measurements

Flash ERG recordings were obtained prior to (baseline condition) and immediately following regional anaesthesia (block condition). Recordings were repeated after ten minutes of orbital compression with a Honan's device at 30 mmHg. (compression condition), and five minutes after cessation of orbital compression (recovery condition).

Statistical analysis

Multivariate analysis of the variance (ANOVA) using repeated measures was performed with subsequent post-hoc paired comparison significance testing using Scheffé procedure.

Results

Typical ERG changes in waveform morphology during regional block and compression are shown in Figure 2. Figure 3 illustrates changes in ERG mean implicit time and amplitude as a function of regional anaesthesia and ocular compression. The Table displays peak implicit times and amplitudes of both a- and b-waves of the ERGs across the experimental conditions. Both the implicit times and amplitudes are normalized to the baseline condition values and expressed as a percentage of baseline. Post-hoc paired comparison using Scheffé procedure demonstrated decreased amplitude of both a- and b-waves from baseline immediately following injection in the peribulbar and retro-bulbar space ($P < 0.0001$). Following application of the Honan's compressive device the amplitude of both the a- ($P < 0.001$) and b-waves ($P < 0.0001$) decreased. Five minutes after removal of ocular compression the amplitudes of the a-wave showed partial recovery while the b-wave showed an increase in amplitude ($P < 0.01$). In a similar manner, the implicit times of both a- and b-waves increased ($P < 0.0001$) after intraorbital injection. Only the implicit time of the b-wave continued to increase with application of compression ($P < 0.05$).

FIGURE 2 Typical ERG changes in waveform morphology that occur during regional block and mechanical oculocompression. The a- and b-waves are indicated by upward and downward arrows respectively.

FIGURE 3 Changes in normalized ERG mean implicit time and amplitude values of the a- and b-wave components during regional block and oculocompression. Error bars represent one standard deviation unit from the group mean.

TABLE Peak implicit time (it) and amplitude (amp) of a- and b-wave components of the flash ERG during regional anaesthesia of the orbit (% baseline)

	Block Mean + sd	Compression Mean + sd	Recovery Mean + sd
a-wave it	118 ± 13*	120 ± 22	108 ± 10§
a-wave amp	65 ± 29*	42 ± 18†	56 ± 22‡
b-wave it	116 ± 12*	126 ± 18†	117 ± 12‡
b-wave amp	61 ± 23*	40 ± 20†	56 ± 19§

*Difference between block condition and baseline ($P < 0.0001$).

†Difference between compression condition and block ($P < 0.05$).

‡Difference between recovery condition and compression ($P = NS$).

§Difference between recovery condition and compression ($P < 0.01$).

$n = 34$ for all measurements.

However, the a- ($P < 0.005$) and b-wave ($P = NS$) implicit times showed varying degrees of recovery with release of compression.

Discussion

The current study demonstrates that reductions in the amplitudes of a- and b-waves of the flash ERG occurred following administration of intraorbital regional anaesthesia and the application of an orbital compression device. Peak implicit times of both the a- and b-waves also increased. Similar ERG abnormalities are seen in rabbits when the retinal circulation is altered to produce acute retinal ischaemia.^{1,9}

The ERG pattern seen during orbital regional anaesthesia may be produced by several different mechanisms that may contribute individually or in combination to decrease retinal perfusion. First, mechanical compression of the retrobulbar circulation may occur following the injection of anaesthetic solution into the retrobulbar compartments if the local tissue compartment pressure is greater than the critical closing pressure of the vessels within that space. When a vessel network passes through a pressure chamber, a Starling resistor effect may occur.¹² The outflow of aqueous from the eye is mainly determined by the pressure difference between the IOP and the episcleral venous pressure. As the aqueous moves through the trabecular mesh into Schlemm's canal through the sclera to episcleral veins the flow through the system is determined by the facility, C_{trab} , of the system. If variables other than the pressure head between the IOP and the episcleral venous pressure, P_e , are constant then the rate of flow F_{trab} through the system is determined by:

$$F_{\text{trab}} = (IOP - P_e)C_{\text{trab}}$$

Mechanical compression of the retrobulbar tissue space may therefore initiate the decrease in retinal perfusion that occurs with retrobulbar anaesthesia.

Second, increased intraocular pressure is known to occur when intraorbital regional anaesthesia is used. We have previously reported that the effect of combined peribulbar and retrobulbar injections of 10.5 ml resulted in mean IOP increases of 3–4 mmHg.⁸ The importance of IOP determinations in the post-block condition is that IOP is a major determinant of retinal perfusion. Retinal vascular perfusion pressure (RVPP) is the difference between the central retinal mean pressure (CRA) and the IOP

$$RVPP = CRA_{(\text{mean})} - IOP$$

Experimental increases in IOP, and consequently the RVPP can be caused by ophthalmodynamometry and body inversion. Both of these non-invasive procedures cause an increase in IOP but the effect on RVPP differs.⁴ Body inversion induces an increase in the IOP because of the increase in ocular blood volume. However, since the arterial blood pressure in the ocular vessels also increases,

FIGURE 4 Anatomical schematic of retinal layers and their vascular supply. The outer third of the retina is supplied by choroidal circulation, whereas the central two-thirds of the proximal retina is supplied by the central retinal artery.

the perfusion pressure does not decrease during inversion. On the other hand, ophthalmodynamometric compression of the globe causes an increase in the IOP that restricts blood flow in the central retinal artery and, thereby, decreases the RVPP. While IOP can easily be doubled by both body inversion and compression dynamometry the effect on RVPP is very different. After retrobulbar anaesthesia the IOP is temporarily increased; however, the RVPP continues to be decreased after the IOP has returned to baseline.⁵ Changes in IOP alone would not account for the observed changes seen in the ERG. It is known that autoregulation of the retinal circulation exists.

Third, autoregulation of the retinal circulation may be affected. Interference by bupivacaine with local vascular control mechanisms were postulated to explain the sustained decrease in retinal perfusion and decreased ocular pulse volume observed after injection into the retrobulbar space.⁴ Using oculo-oscillo-dynamography after the injection of 5 ml bupivacaine, Hessemer demonstrated reduction of perfusion pressures by an average of 5.6 mmHg ($P < 0.001$) and reduction of the ocular pulse volume by 39%.⁴ Fifteen minutes after the injection the perfusion pressures and ocular pulse volumes were further reduced. These findings observed during retrobulbar anaesthesia may reflect an inhibitory effect on ocular hemodynamics by bupivacaine since IOP has returned to baseline by this time. The ERG changes observed in this study persisted after the IOP and orbital tissue pressure had returned to baseline values. The demonstration by Meyer *et al.*⁹ that porcine ciliary arteries are prevented from relaxation by bradykinin in the presence of amide-type anaesthetic agents may be a possible explanation for the sustained decrease in ocular blood flow.

The retina can be divided into three major anatomical levels that correspond to specific levels of electrical activity (Figure 4). The outer third depends on the choroidal

circulation for its metabolism, whereas the middle and inner thirds depend on the central retinal artery. Experiments in cats show that the electrical activity of the inner (proximal) retina is unaffected during systemic hypoxia as long as the arterial oxygen tension (PaO_2) is >40 mmHg. This is due to effective regulation of the inner retinal tissue by PO_2 by retinal autoregulation. In contrast, electrical signals generated in the outer (distal) retina begin to change when PaO_2 decreases below 70–80 mmHg.

If the anaesthetic solution reaches these tissues in sufficient concentration, then the electrical signal would be affected. After retrobulbar injection of anaesthetic, the local anaesthetic agent can be detected in the peripheral arterial blood.¹³ This demonstrates that there is diffusion of the local anaesthetic into retrobulbar vessels. It is known that retrobulbar injection of anaesthetic will decrease ocular perfusion, but it is not known whether the local anaesthetic is carried into the retina in sufficient quantity to affect autoregulation of the retinal circulation or the electrical signal from the retina. The optic nerve is usually affected by peribulbar anaesthesia, and almost always by intraconal injection.¹⁴ However, the electrical activity of the retina is thought to be independent of optic nerve function.

Electroretinogram changes have been shown to reflect retinal tissue hypoxia.¹⁵ It is unlikely that the gradient of concentration of local anaesthetic to the retinal tissues could change rapidly enough to account for the ERG changes observed immediately following intraconal injection. The ERG changes may reflect transient retinal ischaemia produced by raised tissue and IOP and sustained by interference of local vascular control of the retinal circulation. We are investigating the interaction of anaesthetic effect and oculocompression on the ERG to determine the clinical effect of each.

Acknowledgments

This study was supported by an operating grant from the Foothills Hospital Research Foundation. The investigators wish to thank Dr. C.J. Eagle, Dr. G.E.M. Kirker and Dr. J.R. Maltby for helpful editorial comments on the manuscript.

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