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ELECTROPHYSIOLOGY AND THE ORTHOPTIST

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Abstract
Electrophysiological examination of the visual system traces an image from the retina to the visual cortex. By using a combination of tests we are able objectively to establish the integrity of the total visual pathways or to isolate a lesion to a particular area.

Some of these tests are described, i.e. the electroretinogram, electro-oculogram, visually evoked potential and auditory brain stem response and their clinical importance is discussed.

Key Words
Electrophysiology, orthoptist, electroretinogram, electro-oculogram, visually evoked potential, auditory brain stem response.

INTRODUCTION

Any living cell is accompanied by several physico-chemical phenomena of which the electrical is most easily demonstrated. The visual tissues are no exemption to this rule.

The production of electrical activity in the retina obeys the general rules of cell electrophysiology. All living cells including rods and cones possess an electrical charge. This charge is different on each side of the cell membrane. This difference is known as the potential difference. It is due to an imbalance of ions across the membrane, i.e. between the intra-cellular contents (higher K^+ concentration) and extra-cellular fluid (higher Na^+ concentration).

The biophysical characteristics of the cell membrane are changed suddenly by an appropriate stimulus (e.g. light for the photo receptors). This alters the characteristics of the cell membrane separating intra and extra cellular media resulting in an influx of Na^+ and efflux of K^+ with an abrupt alteration of the potential difference – an action potential. This is a very basic explanation for the foundation of electrophysiological responses of vision.

History

Reymond Dubois in 1849 first discovered the resting potential of the eye i.e. the potential difference between the cornea and retina. In 1865 Holmgren noted that a change in this potential was produced by light. In Edinburgh in 1873 Dewar and McKendrick measured this alteration and attempted a human electroretinogram. The method was considered too exhausting and uncertain to permit qualitative or clinical investigation. Further developments occurred as a result of the work by Einthoven and Jolly, 1910, Sach, 1929, and Granits in 1933. But until Rigg in 1941 and Karfe in 1944 produced a contrast lens electrode this investigation remained difficult. Modern electronic and computer development has resulted in a marked increase in activity in this field.

Ocular electrophysiological tests of vision include the following examinations:–

1. Electroretinogram E.R.G.
2. Electro-oculogram E.O.G.
3. Visually evoked cortical potential V.E.P.

Electrophysiological examinations of ocular motility include:–

1. Ocular electromyography
2. Electronystagmography
3. Auditory brain stem response

The E.R.G. (Electroretinogram)

This represents the action potential of the retina. It is produced by a brief stimulus, e.g. a flash of light and is recorded by electrodes placed on or near the eye.

The form of the E.R.G. (Fig. I). The initial response is the A wave – a negative (in our laboratory downward deflection) wave of relatively minor amplitude. This is followed by a positive peak of much larger amplitudes. The final response is the C wave a less well defined positive wave. The responses are altered by the state of light or dark adaptation.

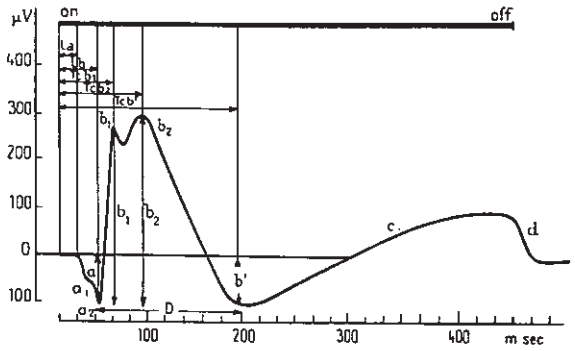

Figure I. Normal E.R.G.

The small oscillations on the surface of the B wave, oscillatory potentials, are a more recently described phenomenon of considerable significance.

The cellular origin of the E.R.G. components is as follows:–

A wave – inner segment of receptor cells
B wave – Muller cells
Oscillatory potentials – Feed back amacrine to bipolar cells
C wave – pigmentary epithelium

Thus the E.R.G. is a function of the outer layers of the retina – pigment epithelial layer of the bipolar cells.

The E.O.G. (Electro-oculogram)

This measures the change in the resting potential of the eye as it passes from dark-adapted to light-adapted phase.

It is a measure of the integrity of the pigment epithelium. The amplitude of this response is normally far greater in the light adapted state than it is in the dark adapted state. The ratio between these two resting potentials expressed as a percentage is known as the Arden Index.

Clinically the value of these two tests is inseparable and in investigation of retinal function usually both tests are required.

Definitive diagnostic assistance is available in:

A. Disorders of choroid
B. Disorders of pigment epithelium
C. Hereditary central retinal dystrophies and degeneration
D. Receptor cell disease
E. Retinal degenerations
F. Retinal intoxications
G. Trauma

The Visually Evoked Potential

This test pursues the electrical activity from the inner layers of the retina (ganglion cell layer) to the cortex i.e. via the optic nerves, chiasm and radiation. Disease or disorder at any area in this pathway can result in disruption of this response. This response can be used as a measure of retino-cortical conduction and because of the large cortical area devoted to fibres from the maculae it represents principally the reception of the central retinal message.

Technique of Measurement of the V.E.P.

Electrodes are placed over the occipital region (positive) and a reference electrode is applied to the vertex. The patient is then placed before a suitable visual stimulator. One of the most commonly used stimuli is a checkerboard which is so arranged that the black and white squares can alternate in position i.e. produce a "pattern-reversal stimulus".

To effectively extract the biological signal from the electrical noise of muscles and other brain activity each stage of signal processing must be as efficient as possible.

The Pre-Amplifier

The electrodes are connected initially through short cables to a pre-amplifier. This is required because biological signals are so minute and easily distorted by ever present electrical interference. Biological signals need to be amplified before interference (noise) can dilute the required signal. The output from the pre-amplifier is connected then to a further device.

The Biological Amplifier

This is an electronic signal processor capable of increasing the amplitude of tiny biological electrical impulses by many thousand times.

Biological signals as low as a micro volt (i.e. one millionth of a volt) can be increased to one volt so that they can be displayed on oscilloscopes, drive XY plotters, or be converted to digital code for computer processing.

The Averager

The signals from the amplifier are fed to an averager (which is usually computer controlled) when they are so small that they cannot be distinguished from the noise. This will "average out" the noise while the signal (being always present) will be additional and will grow, e.g. one thousand samples of a nana volt (10^{-9} volt) will produce a microvolt signal (10^{-6}) with noise virtually absent.

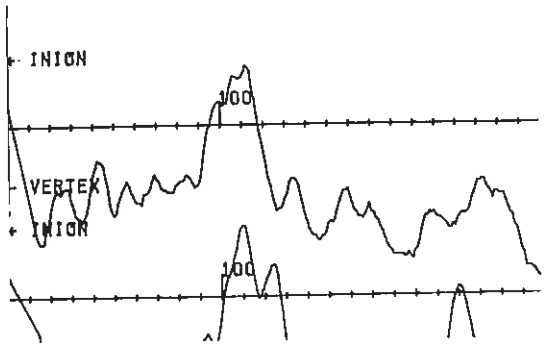

Figure II. The normal V.E.P.

The signal having been collected can then be displayed, measured and printed.

The Normal Visual Evoked Potential

The normal V.E.P. (Figure II) has several wavelets. The fifth or principal wave occurs at 110± 5 milliseconds from the time of the stimulus. It is regarded as normal if it has an amplitude of greater than 7.5 microvolts, i.e. has a magnitude of 7 millionths of a volt and occurs about one tenth of a second after the stimulus.

Disease or disorder of the visual pathway will result in an abnormality of the V.E.P. This can be represented by any of the following:–

a) Decreased amplitude
b) Altered waveform
c) Prolonged latent period
d) Abolished response

The clinical use of the V.E.P. has been the subject of a previous paper[1]. The more common uses are summarised in Table I.

This paper will discuss the use of the V.E.P. in objectively determining vision.

TABLE I
COMMON CLINICAL USES OF THE V.E.P.

A. LESIONS OF THE OPTIC NERVE AND ANTERIOR VISUAL PATHWAYS FREQUENTLY DISPLAY ABNORMAL V.E.P.

1) Demyelinating lesions
2) Compressive lesion of the anterior visual pathways
3) Toxic lesions
4) Ischaemic lesions
5) Optic atrophy

B. ASSESSMENT OF VISUAL ACUITY

1) In infancy or incapacitated patients
2) In hysterical amblyopia
3) Amblyopia

C. VISUAL FIELD ASSESSMENT

Two techniques are available
1) Using a multiple electrode technique
2) Using half or quadrantic field stimulation

D. SCOPE FOR FUTURE DEVELOPMENTS

1) Refraction
2) Disturbance in colour vision
3) Detection of learning disabilities

a) The V.E.P. in Assessment of Visual Acuity

The V.E.P. obtained with a pattern reversal stimulus increases in amplitude with increasing element size until it reaches the modal size of receptive field centres (i.e. subtends an angle of 10′ – 20′). Subsequent increase in stimulating element size results in a decrease in amplitude (Fig. III). With diminished visual acuity maximum amplitude will be seen with a larger stimulus (e.g. 40′).

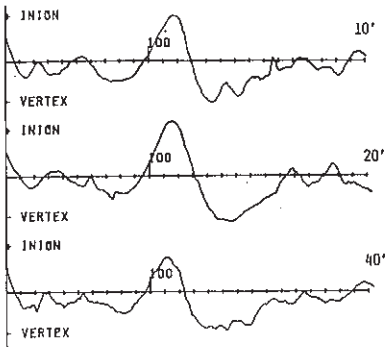

Figure III. V.E.P. with variable size checkerboard stimulus. Largest response occurs with 20′ checkerboard.

Methods of estimation of visual acuity using the V.E.P. has been suggested by a number of authors Sokol[2], Dobson[3], Atkinson[4]. A variety of techniques are described, e.g. graphs of amplitude of response to varying size of stimuli are extrapolated to zero.

b) Refractive Error

The amplitude of response of the V.E.P. will be greatest when the image is in clearest focus on the retina. An objective refraction is therefore possible by varying the corrective lens to obtain the maximal response. If a linear grating is used and this is rotated the axes of astigmatism can be discovered.

c) Amblyopia (Fig. IV)

More simply the amplitude of response can be compared between the two eyes. The amplitude of one subject's eyes will be equal with equal visual acuity. Reduction of one response means reduction of vision in one eye. A definitive diagnosis of reduced vision and therefore amblyopia is thus possible at any age in the absence of pathological or refractive changes. Latent period of the principal peak may be prolonged in the amblyopic eye.

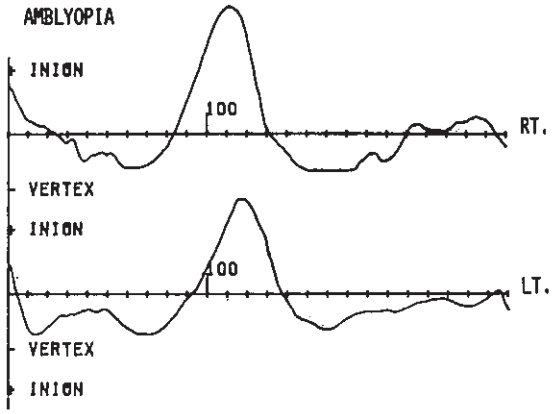

Figure IV. The left V.E.P. is reduced and delayed due to amblyopia.

d) Binocular Vision

Single binocular perception of images falling on the retina may be obtained by neural summation of images or by neural suppression. This can be confirmed electrophysiologically. Under normal viewing conditions virtually the same images fall on corresponding retinal areas i.e. dioptic viewing. This results in neural summation. In disorders of binocular vision the image on each retina differs in terms of sharpness of focus, size, orientation, intensity or correspondence i.e. dichoptic viewing. This results in neural suppression.

Summation of V.E.P. (Fig. V)

In dioptic viewing the binocular V.E.P. is greater than the monocular V.E.P. Summation is greater for sharply focussed patterns than for diffuse lights. If identical patterns are presented dichoptically,binocularly summation occurs, but as presentation of pattern to one eye is varied binocular suppression occurs, increasing with the degree of dissimilarity (Fig. VI).

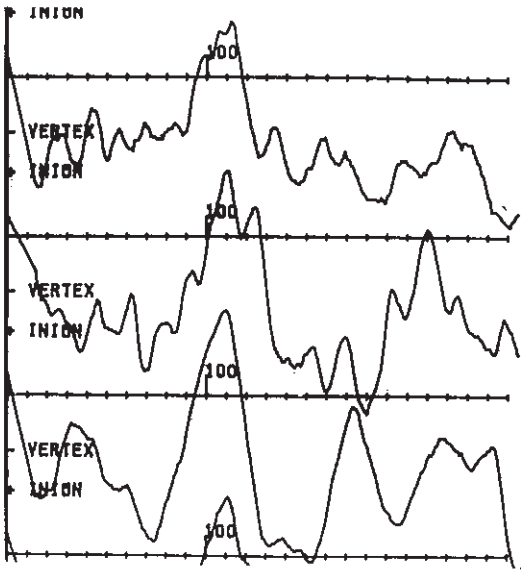

Figure V. Summation of V.E.P. with binocular viewing. The lowest tracing shows the increase in response with binocular vision.

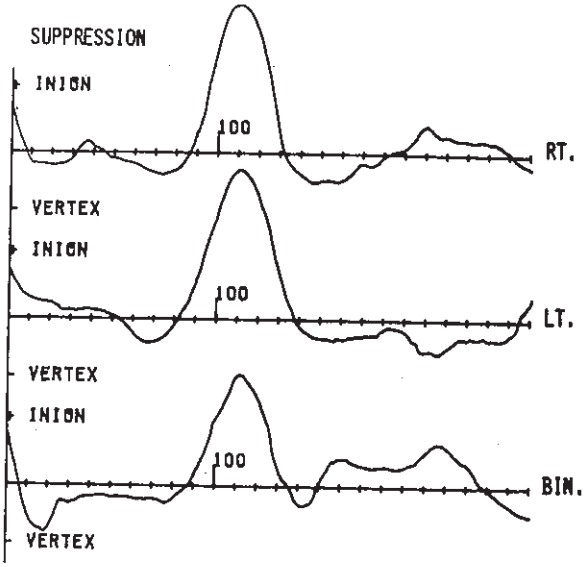

Figure VI. Suppression of V.E.P. with dichoptic stimulation. The lowest response shows a reduction in response with dichoptic stimulation.

The V.E.P. thus enables objective diagnosis of binocularity and even the clinical ability to objectively determine stereo acuity becomes possible with a special stereoscopic stimulating pattern[5].

Having determined the presence or absence of binocularity a further evoked response is becoming increasingly important in investigation of eye movement.

The Auditory Brain Stem Response

This response is one of the most exciting electrophysiological tests for the ophthalmologist and particularly the orthoptist.

The auditory evoked response uses an auditory response to elicit an electrical response from the brain stem. This may be followed to the cortex but unlike the visually evoked potential the anatomocal generator of each of the early waves of this response is known. (Fig. VII)

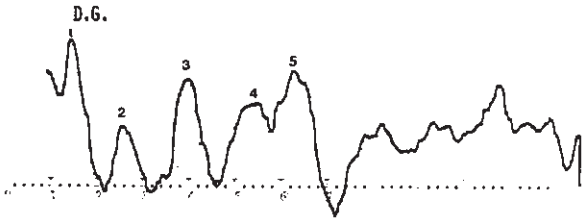

Figure VII. The normal auditory evoked potential origin of Wave I – cochlea
Wave II – cochlear nucleus
Wave III – superior olivary complex
Wave IV – lateral lemniscus
Wave V – inferior colliculus
Wave VI – medial geniculate body

The pathway of the auditory impulse through the brain stem to the inferior colliculus is therefore in close association of the nuclei of the III, IV and VI nerves. Abnormalities of the A.B.R. have been noted in.

1. Duane's syndrome
2. Congenital esotropia
3. Gaze palsies

1. Duane's Syndrome

Hoyt[6] noted an abnormality in the A.B.R. in a moderate number of patients with Duane's Syndrome. This is demonstrated in Fig. VIII.

2. Congenital Esotropia

The neurological aetiology of congential esotropia is usually suspected . Fig. IX displays the A.B.R. from an infant with congenital esotropia. The only evidence of neurological damage in this

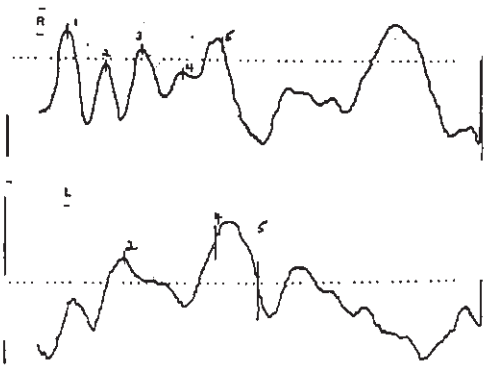

Figure VIII. The A.B.R. in Duane's syndrome shows an abnormal wave III and delayed waves IV & V.

child was a body tremor. The A.B.R. gives definite evidence of brain stem damage as an aetiological factor.

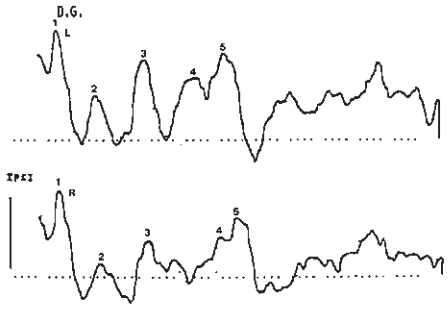

Figure IX. The A.B.R. with a brain stem lesion shows a delayed wave IV & V with decreased amplitude in the right tracing.

3. Gaze Palsies

Review of 20 multiple sclerosis patients who had a history of a gaze palsy showed that 90% had an abnormal A.B.R. This was a much greater percentage than the 60% with abnormalities of the A.B.R. in those multiple sclerosis patient's without a history of a gaze palsy.

Review of patients with a history of gaze palsies of other aetiologies is currently being undertaken.

The auditory evoked response may well become more an ophthalmic than an aural investigation.

CONCLUSION

Electrophysiological investigation is an important clinical part of ophthalmology. In particular visual and auditory evoked responses offer potential for investigation of neuro-ophthalmic disorders and research into function of the visual pathways which were previously unavailable.

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