Principles and Practice of Clinical Electrophysiology of Vision

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Electro-oculography

Hansjoerg E. Kolder

CLINICAL ELECTRO-OCULOGRAPHY

Clinical electro-oculography (EOG) records the voltage difference between the posterior pole of the human eye and the cornea. The evidence from clinical observations, animal experiments, and cellular physiology is convincing that the retinal pigment epithelium is the major if not the sole origin of the EOG. DuBois-Reymond[28] first demonstrated this potential in an animal eye over 140 years ago. The phenomenon has variously been called Bestandpotential, standing potential, resting potential, corneoretinal potential, and corneofundal potential. Marg[103] reviewed the early development of the technique of recording eye movements by utilizing the standing potential. Several books and reviews contribute to an understanding of the history of EOG and its clinical application.[35, 44, 45, 79, 88, 119, 142] No attempt has been made in this chapter to provide a comprehensive list of references, but a sufficiently representative sample of the recent literature is included.

PRINCIPLE OF INDIRECTLY RECORDING THE ELECTRO-OCULOGRAM

The electrical potential generated within the eye is distributed in the globe like in a volume conductor.[120] The eye is surrounded by an electric field, the strength of which is dependent on the electric impedance of the periocular tissue. The potential measured anywhere in the electric field is proportional to the dipole potential. EOG measures a potential in the electric field surrounding the eye that is proportional to the sum of all potential sources (generators) and potential sinks within the eye. The EOG is a mass response and provides only indirect and nonspecific evidence about the anatomical and physiological substrate generating it.

Figure 39–1 schematically shows the principle of recording the EOG. Two electrodes are placed on the skin close to the medial and lateral canthi. A potential difference is thus measured.

The potential will be made up of several components, only one of which is due to the ocular dipole. For example, slowly changing voltages at the electrode-skin interface are to be expected.[83] When the eye moves, these other voltage/current sources should be constant for the short time that is occupied by the saccade. Therefore the eye movement potential is determined by the amplitude of the eye movement and the strength of the intraocular current generator.

Since eye movements are completed in less than 500 ms, the polarization current is unlikely to change significantly during that short time. The EOG potential is then measured before and after an eye movement. The difference between the potentials in two eye positions is measured, recorded, and stored.[15, 16, 19, 54, 83, 95, 127, 137, 151]

Nilsson and Andersson[112] and Skoog[134] have developed a method to record the EOG directly from the eye. A nonpolarizing electrode is placed on the cornea; no eye movements are required. The more widely used indirect method of recording an EOG with eye movements has the advantage of ease of

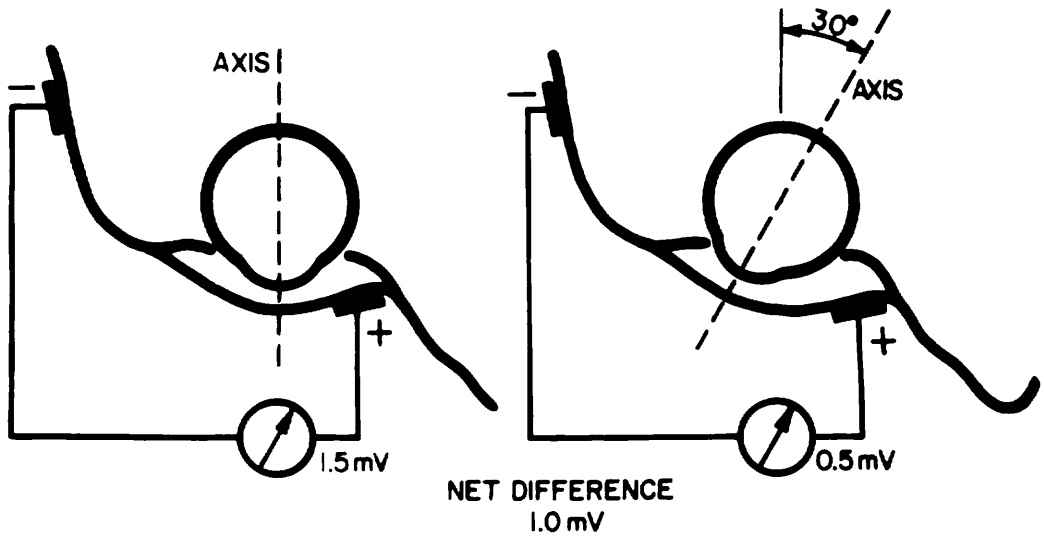

FIG 39–1.
Principle of indirectly recording the human EOG. Two electrodes are placed on the skin next to the canthi. The potential difference is measured between the two eye positions.

application of electrodes to the skin and less discomfort for the patient. The results of the directly and indirectly recorded EOG are comparable.[129]

The reliable and reproducible measurement of the EOG depends on stable amplifiers to measure the potential which is in the order of 1 mV. A differential input amplifier with 1 MOhm or higher input impedance picks up the potential from skin electrodes. A resistance between skin and electrode of less than 5 kOhm is desirable. The frequency band of the amplifier should extend from dc to 10 Hz, although an ac amplifier with a long time constant (10 sec) is acceptable.

The following strategy ensures that the potential difference is recorded when the eye position is stable. The electronic gate is opened 500 ms after a fixation light has been activated to permit sufficient time for the eye to fixate. A measurement of voltage is made, and the amplifiers shorted. Thereafter, another fixation light is activated and an eye movement performed; 500 ms later the gate is opened again, and the potential in this new position is measured and stored. The difference between the two aforementioned potentials is recorded as the EOG. Eye movements are repeated, e.g., every 10 seconds, and the EOG can be plotted as a function of time. Each potential difference from an eye movement can be displayed on a strip chart recorder advancing 2 mm/min; this permits the identification of incomplete eye movements or blink artifacts. Present-day electronic equipment can be used to reject extraneous potentials if they exceed a predetermined value, e.g., ±20% over the preceding potential. Instead of a continuous display these recording systems store all potentials in memory or on disk; a hard copy of the original record with electronic purging of artifacts can be produced or further mathematical analysis initiated.

The frequency with which eye movements are replicated is determined by the resolution required and by the cooperation of the patient, e.g., if the fast oscillation of the EOG (see below) is investigated, eye movements should be repeated every 5 or 10 seconds; one period of the fast oscillation lasts about 120 seconds. If the light peak-to-dark trough ratio is of interest, eye movements may be repeated every minute; the dark trough is normally separated by about 15 minutes from the light peak. Frequent eye movements fatigue patients, probably from insufficient time for blinking. Trains of eye movements, e.g., ten eye movements rapidly repeated every minute, may condition anticipation of the position of a fixation light; the gating of the amplifier then becomes crucial. Inappropriate gating causes artifacts that can be recognized by monitoring the signal before electronic manipulation.

EYE MOVEMENTS

Several methods have been employed for clinical EOG tests.[8, 25, 66, 84, 118, 143, 150] The angle for the eye movements prescribed by fixation lights is often 30 degrees. The EOG potential is then large enough to

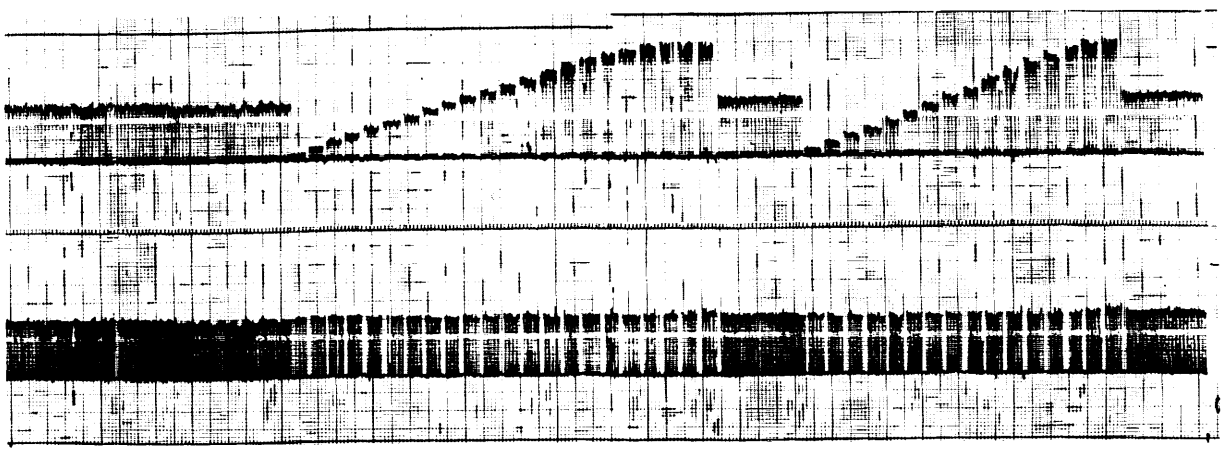

FIG 39–2.
EOG used to monitor eye positions. The *upper* tracing on the left shows eye movements increasing in steps of 5 degrees; on the right the eye movements increase proportionally to the sine of the angle. The *lower* tracing verifies the stability of the EOG; each eye movement over a varying angle is followed by an eye movement over a constant angle. The abscissa is divided into 30-second intervals, the ordinate into 50-μV increments.

be measured reliably, and the peripheral stimulation of the fixation light is easily recognized because it initiates a prompt saccade. The replication of eye movements for EOG recordings is important. For as long as eye movements are made over the same angle, the measured potential difference between the two eye positions is almost exclusively dependent on the potential source for the EOG, disregarding fast changes of the polarization current at the electrodes. Precautions have to be taken in special situations, e.g., proptosis increases the absolute EOG voltage[5]; myopia, when associated with peripheral chorioretinal degeneration, decreases the EOG response to light.[18]

If eye position or movement and derived parameters like globe velocity and acceleration are to be recorded, the EOG should be frequently calibrated by making standard eye movements because of slow changes (over minutes) of the intraocular generators.

Figure 39–2 shows on the upper left a "staircase" of eye movements over linearly increasing angles. The right upper staircase depicts eye movements increasing proportional to the sine of the angle. The lower tracing graphs eye movements over a constant angle of 30 degrees that were alternated with eye movements over varying angles. This method provides a semicontinuous calibration for tests when the EOG potential is critically used to monitor eye position.

The relationship between the angle of eye movement and EOG potential is not strictly linear. If an accuracy in excess of 1 degree of arc is required for recording eye movements, careful and repeated calibrations must be inserted.[23, 41, 115]

LIGHT PEAK

The EOG was long thought to be invariant ("standing potential"), although there were dissenting voices.[105] Over 30 years ago Kris[86] convincingly demonstrated a slow increase of the EOG with adaptation to light and a decrease in dark. This observation was confirmed and expanded extensively.

Figure 39–3 depicts the EOG as a function of time, first in dark, then in light. In the dark the EOG reaches a steady state. The onset of light (400 lux at the eye level) causes the EOG to increase; this is followed by a return to its steady state with two periods of a slow oscillation. The period of the slow oscillation lasted 27 minutes in this test.

The amplitude of the slow oscillation of the EOG, in particular, the first peak, is dependent on light intensity.[9, 10, 38, 39, 62, 86, 94, 105, 146, 152] Since the work of Arden and Kelsey[9] it has been known that a quasilinear relationship exists between the EOG and log luminance over approximately 3.5 log units. This dependence of the EOG on light intensity includes contributions from the scotopic system and the photopic system, at least in their indirect effect on the retinal pigment epithelium. When analyzing the slow oscillation in detail by simulating its waveform with a biological feedback model having at least three components, the linear EOG parameters are dependent on light intensity over 7 log units.[62]

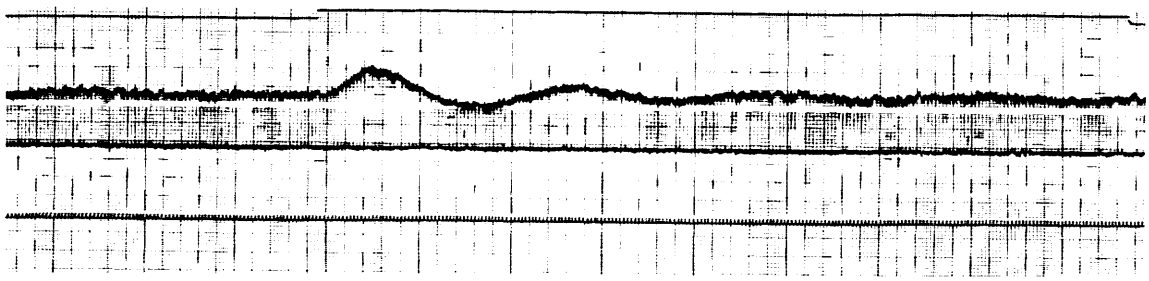

FIG 39–3.
EOG recorded with eye movements repeated every 10 seconds to demonstrate the slow oscillation. Dim red fixation lights were 30 degrees apart. Following adaptation to dark, an incandescent light was turned on as marked on the *top* tracing. The intensity of the illumination received at the eye level measured about 400 lux. The light source subtended 30 degrees. The marks on the *bottom* tracing indicate 30-second intervals; 20 squares on the ordinate represent 1 mV.

The EOG is also dependent on the wavelength of light falling in an eye,[2, 11, 12, 32] and thus contributions from both the scotopic and photopic systems can be demonstrated. In patients with congenital achromatopsia the scotopic response of the EOG is reduced and the light peak delayed, while the fast oscillation remains intact.[159] The response to red light is subnormal, that to blue light normal.[31] Scotopic saturation was tested in order to isolate the contribution of cones to the EOG.[50, 57]

The EOG is a summed response originating in the pigment epithelium. Both the peripheral and macular receptors are involved.[26, 93, 136] Therefore, stimulation of the total retina ("Ganzfeld") is assumed to optimize the EOG response.[33]

Numerous authors have reported normal data for EOGs obtained under specified conditions.[1, 7, 23, 60, 70, 74, 89, 92, 108, 110, 123, 169, 170, 176] The interindividual variation exceeds the intraindividual variation.[63]

The difficulty with recording the EOG is the slow development of a response and the considerable time needed to await a steady state.

Figure 39–4 is representative for an EOG recorded for 90 minutes. Eye movements were repeated every 10 seconds, and no signal processing was performed except zeroing the potential prior to the eye movement. Twenty divisions on the ordinate equal 1 mV, each division on the abscissa denotes 30 seconds, the event marker on the top indicates when the incandescent light was turned on, and the illumination received at eye level corresponded to about 1,400 lux. The EOG stabilized in dark except for a possible shallow minimum about 15 minutes prior to the light stimulus. Within the first 2 minutes of light adaptation a decrease in the EOG is observed that is followed by a "light peak" 8.5 minutes later. The EOG decreases thereafter and reaches a trough in 13.5 minutes; a second light peak occurs 27 minutes after the first light peak, i.e., the period of this slow oscillation is 27 minutes, which gives a frequency of the slow oscillation of 2.2

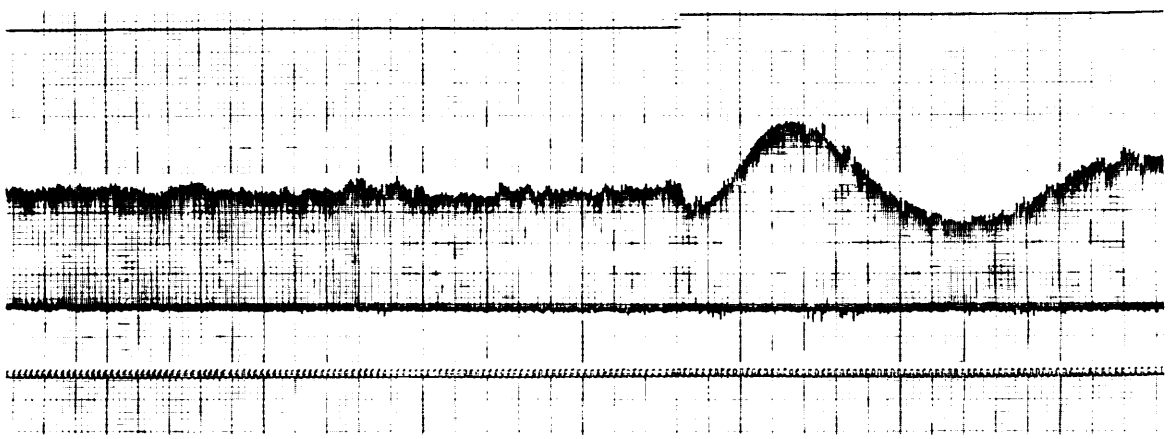

FIG 39–4.
An EOG in response to light of approximately 1,400 lux at eye level illustrates an initial decrease followed by a "light peak."

cycles per hour. The frequency of the fast oscillation (part of it is recognizable as initial decrease in the EOG with light stimulation) is 28.5 cycles per hour. The amplitude of subsequent peaks decreased, and this suggests a damped oscillatory mechanism responsible for the EOG response to light.

RESPONSE PARAMETERS

Several parameters can be measured directly from an EOG: the steady-state potential to which the EOG returns following an oscillation, sometimes referred to as base potential; light peak potential, dark trough potential; the ratio of light peak to dark trough; the ratio of light peak to steady-state potential; the ratio of the first light peak to the second light peak; the time to the first light peak, time to the dark trough; duration of the period; and frequency of the slow and fast oscillations. Parameters derived from mathematical modeling[61] are more complex and have not been tested sufficiently in retinal diseases.

Figure 39–5 exemplifies, with an original EOG tracing and a schematic drawing, the parts of the light response that are customarily measured and clinically used. For this particular recording the EOG response was forced by repeated light and dark phases. Of all the parameters listed, the ratio of light peak to dark trough has found widest acceptance since Arden et al.[8] suggested it. The reason may be quite pragmatic: irrespective of the level of preceding light, the EOG will oscillate to a minimum during a following adaptation to dark ("dark trough"). Timed appropriately, e.g., not more than 15 minutes after the beginning of dark adaptation, a light stimulus will trigger the development of a light peak of maximal or near maximal amplitude. This strategy elicits an oscillation but utilizes only a fraction of the time needed to await a steady state. The light peak-to-dark trough ratio (Arden's ratio) maximizes the response, at least in most cases. It is possible that more and differential diagnostically important information from the EOG remains undetected by using Arden's abbreviated protocol.[156]

MATHEMATICAL MODELS

To refine the evaluation of the EOG response to light, physiological models have been proposed.[61, 63, 145, 149] One model[61] assumes that the in-

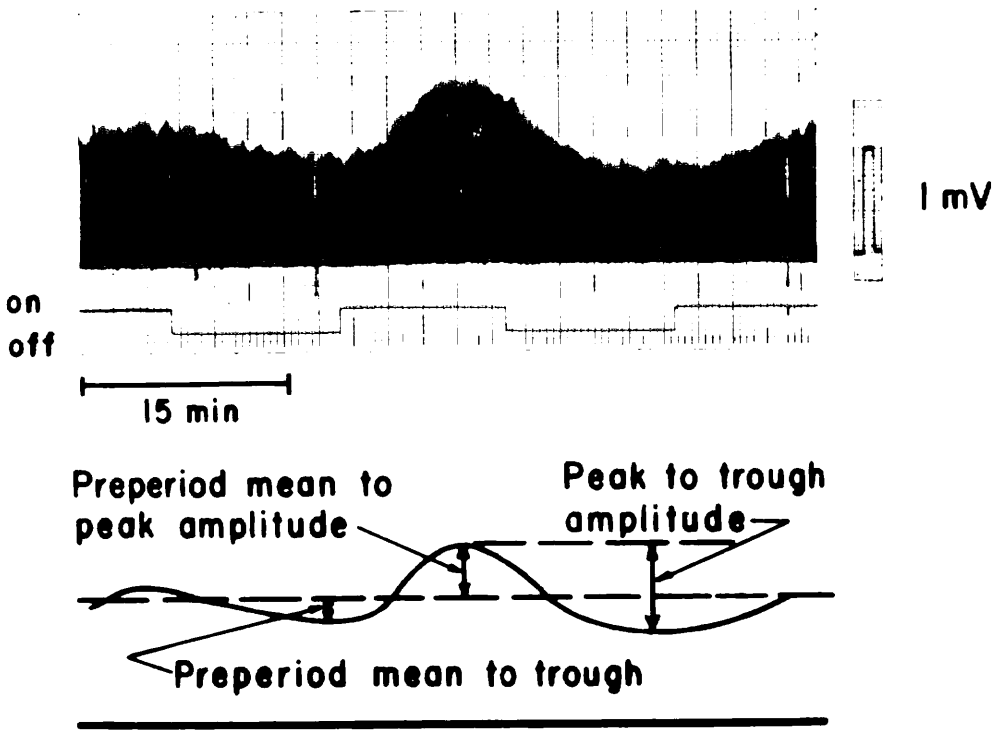

FIG 39–5.
Slow oscillation of an EOG that is forced by light and dark phases each lasting 12.5 minutes. Parameters are indicated that can be used to quantitate the EOG response.

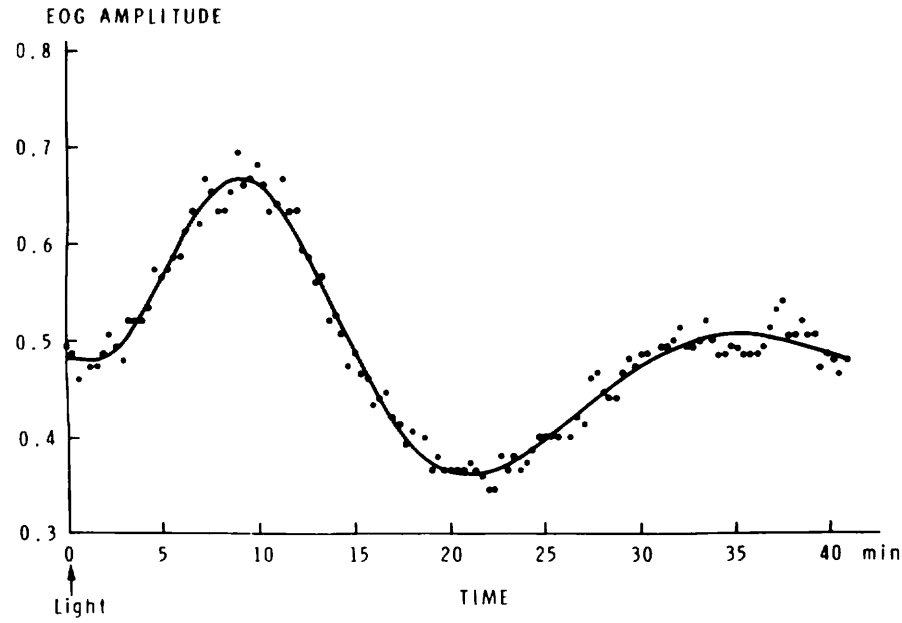

FIG 39–6.
Computer simulation of the human EOG in response to light. The *dots* indicate eye movements repeated every 10 seconds. The *solid curve* was calculated by least-squares fit of a nine-parameter model, assuming the interaction of four mechanisms.

directly recorded response of the EOG is a composite of three or more mechanisms with feedback between them. A set of differential equations, one for each mechanism, describes these interactions. The solution is nonlinear and has nine parameters for four mechanisms (Fig 39–6). Three of the parameters are linear and have been correlated with the intensity of the light over a range of 7 log units. The model has been tested and predicts intraindividual and interindividual variations well.[63] Because of the iterative process needed to solve the equation, considerable computer time was originally required, and no recent attempts using fast computers for the desirable, on-line analysis of the slow oscillation of the EOG have been published.

A multivariant analysis of the factors age, sex, refractive error, and visual acuity was applied to a group of persons without known eye diseases.[81] No factor significantly correlated with the light peak-to-dark trough ratio. Seventy percent of the variance observed could be accounted for by combining the four factors mentioned.

SPECIAL RECORDING CONDITIONS

The clinical recording of the EOG is not strictly an objective test of the function of the eye. The cooperation of the patient is necessary to follow the fixation lights. Direct recording of the EOG by means of a nonpolarizing corneal contact lens electrode circumvents this disadvantage but still requires the cooperation of the patient to tolerate the electrode on the cornea. Patients with poor visual acuity can be asked to make "maximal" horizontal eye movements; even though this technique will not yield an EOG as a function of a constant angular movement, it may be useful as a relative measure of pigment epithelium function. Infants have been tested by means of passive eye movements induced by the vestibular reflex.[51, 167] Children from 5 years of age on can often be motivated to cooperate with an EOG protocol. In animal experiments the globe can be moved passively over the same angle, thus simulating an indirectly recorded EOG. An analogous method has been suggested for patients under general anesthesia. As a rule patients with a visual acuity of 6/60 or better, with best correction, are able to adequately follow the fixation lights. Malingerers can easily suppress eye movements prescribed by fixation lights, thereby rendering impossible the interpretation of EOG test results. But the EOG can be recorded by using an "optokinetic" (striped, rotating) drum as a stimulus as described for small children[24]; the optokinetically induced refixation movement is difficult to suppress. Media opacifications dense enough to reduce the visual acuity to less than 6/60 cause the fixation to be uncertain; EOG test results are then questionable. If one eye has adequate visual acuity and no strabismus or only concomitant strabismus

exists, a useful EOG tracing can be expected from the contralateral eye with opaque media. Light stimulating one eye produces a low-amplitude EOG of inverse polarity in the contralateral, occluded, or diseased eye.[27, 43, 128, 164, 165] This propagated potential may even simulate an attenuated EOG from an anophthalmic socket.[117]

The circadian stability of the EOG is of importance when repeating tests at various times.[6] Long-term continuous recording of the EOG is tiring; therefore, few experiments have been reported that cover 24 hours. In one such set of experiments, the EOG, in its steady state, proved to be stable and independent of the nocturnal decrease in body temperature (i.e., having no circadian rhythm).[79] Other experiments, conducted over shorter periods of time, support the assumption of diurnal variations of the light peak-to-dark trough ratio.[13, 58, 67, 87]

NON-PHOTIC STIMULI

Numerous nonphotic stimuli have been tested for their effect on the EOG.[4, 14, 140] The response to subcutaneously injected adrenalin increases the EOG in a dose-dependent manner; adrenalin does not elicit an oscillatory response.[83] The influence of alcohol[135, 177] and hypoxia[78, 104] have been documented. The effect of acetazolamine on the EOG has been researched specifically with the aim to develop a test of the function of the pigment epithelium, which is independent of light stimuli. An injection of acetazolamide causes a dose-dependent decrease in the EOG[20, 71, 101]; subsequent light stimulation evokes a modified "light rise." It is likely that acetazolamide acts on the pigment epithelium of the ciliary body and on the retinal pigment epithelium. Timolol, a β-adrenergic antagonist, causes a similar decrease in the EOG potential in the steady state.[106] Hypertonic solutions (e.g., 20% mannitol) injected intravenously reduce the EOG in a dose dependent manner without triggering a slow oscillation. The variance of the EOG is less in response to hyperosmosis than to variance of the light peak following stimulation with light. This observation suggests that nonphotic stimulation may provide a more sensitive test of the function of the pigment epithelium than photic stimulation does.[72, 102, 174]

SLOW OSCILLATION

The slow oscillation of the EOG[62, 78, 86, 88, 97, 143, 147, 155] must be initiated by a mechanism that differs from the interaction of photons with receptors in the outer segment. The latter interaction triggers the components of the electroretinogram (ERG). A mediator ("light peak substance") for the transmembrane potential changes in the pigment epithelium has recently been reported.[42] Since the receptors are surrounded by pigment epithelium and metabolically dependent on it, an interdependence is not surprising, although the temporal relationship suggests a "loose" coupling. The time during which the slow oscillation returns to a steady state is too long to be attributable to photopigment regeneration. Oscillatory, damped metabolic changes could be slow but have not been reported from the eye to the extent suggested by the period of the slow oscillation of the EOG. A slow oscillation develops also in response to repeated short light and dark phases. Indeed, an abbreviated slow oscillation (i.e., shortened period) can even be found in response to a high-intensity photoflash.[30, 148] Experiments on rabbits, with indirect recording of the EOG, revealed an inverse correlation between systemic blood pressure and the steady-state part of the EOG.[82] Similar observations were reported from the perfused eye preparation.[111] Metabolic or hemodynamic changes are epiphenomena and need to be correlated with potential changes across the pigment epithelium.

The adduction of the site of origin of the EOG required animal experiments.[75, 82, 166] Numerous species, from amphibians to mammals, show a slow increase in the EOG in light and a decrease in dark. Notable are experiments with rabbits,[36] since their ocular vascular system depends mainly on the choroid, and sheep,[76] whose circulation closely resembles that of humans. Transection of the optic nerve proximal to the entrance of the central retinal artery does not diminish the EOG response to light.[65, 79]

Another significant step in elucidating the origin of the EOG was provided from observations on isolated, perfused eyes.[21, 85, 111] Oxygen saturation, pH, and flow have been identified as factors influencing the EOG.

Intracellular placement of electrodes enabled the recording of transmembrane potentials of pigment epithelial cells as well as active and passive ionic changes during excitation.[49, 73, 98, 100, 109, 116, 138, 139, 168, 172]

The photoceptors are the primary site of the transduction of the energy of photons into the physiological response initiating the EOG. The pigment epithelium has been proved to be the generator source. Conduction of receptor excitation to the pigment epithelium requires a humoral transmitter and may be affected by several neurotropic intermediates and/or pharmaceuticals.[22, 59, 68, 69, 76, 130, 132, 133, 153]

A gradual loss of the light peak of the EOG has been observed in Irish setters affected by the hereditary canine ceroid lipofuscinosis, which is similar to Batten's disease.[113]

FAST OSCILLATION

The fast oscillation of the EOG[56, 80, 126, 144, 162] is discernible by the initial decrease[53, 88] in potential following stimulation with light. This decrease and return to baseline happens over a 1-minute period and blends into the light rise of the slow oscillation of the EOG. Similar but with opposite polarity is the initial response of the fast oscillation of the EOG to dark; this contrasts with an increase in potential during the first minute followed by the dark trough of the slow oscillation.

The *fast* oscillation is critically damped so that only the first half-period is recognizable when testing the EOG by means of a step increase in light intensity. By varying the repetition rates of on-off phases of the stimulating light, the fast oscillation can be brought into resonance (Fig 39–7). The optimal duration of repeated light stimuli occurs between 1 and 1.25 minutes. The amplitude of the fast oscillation is dependent on light intensity.[162] Superposition of two stimuli, one timed to evoke a slow oscillation and another to maximize the fast oscillation, reveals that the fast oscillation is suppressed during the light phase that evokes the slow oscillation,[79] an observation that has been disputed.[96] The amplitude of the fast oscillation is not affected by the amplitude of the slow concomitantly elicited oscillation when the latter is triggered by on-off phases of light of about 1 minute each, which optimizes the fast oscillation. Results from experiments on rabbits, with indirectly recording the EOG, suggest that the fast oscillation is more resistant to hypoxia than is the slow oscillation.[82] The origin of the fast oscillation has been attributed to ionic changes across the pigment epithelium.[48, 99, 171] The c-wave of the ERG correlates well with the fast oscillation of the EOG.[114, 125, 129]

CLINICAL APPLICATIONS

The use of the EOG to diagnose ocular disorders[3, 8, 16, 37, 39, 119] has gained less attention than has testing the ERG. Several causes have been suggested, some are avoidable. The EOG is a mass response, and the cellular mechanism(s) has only recently been thoroughly researched. The EOG develops slowly, and its testing is tedious. The EOG is phenomenologically simple, and the response parameters are seemingly obvious: amplitude and time to peak. As mentioned previously, several more parameters could be used for a more detailed analysis.[90, 91] Additionally, subliminal encroachment of light changes on the EOG has been shown to avoid triggering an oscillation[143]; this procedure permits shortening the time needed to attain a steady state. The EOG response is nonlinear when tested by means of a sinusoidal light stimulus.[17] The correlation between eye movement, for indirectly recording the EOG, and the EOG potential is also nonlinear.[115]

The clinically recorded EOG is important as a test for abnormalities of the pigment epithelium. The EOG is paramount for the diagnosis of Best's disease (see Chapter 91). The EOG might be useful as a test for all conditions manifested as "retinitis pigmentosa." Controversy surrounds the validity of EOG tests adjunct to the diagnosis of choroidal melanoma.[161] Earlier reports on the predictive value of the EOG test in patients using chloroquine for the treatment of rheumatoid arthritis or lupus erythematosus[29, 34, 47, 55, 77] have lost some of their impor-

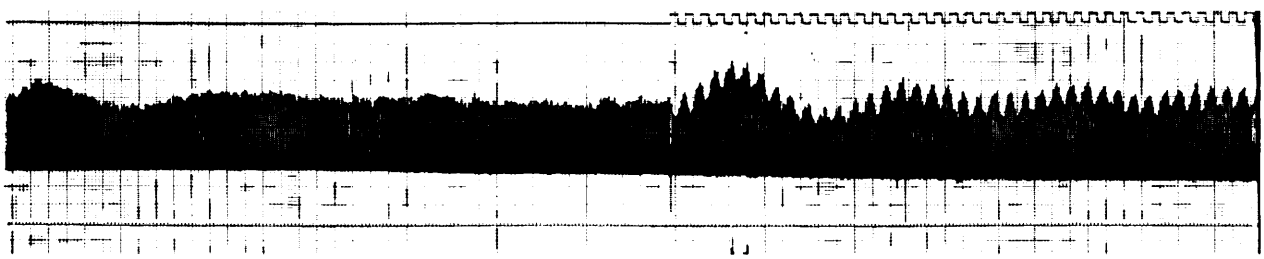

FIG 39–7.
Fast oscillation of the EOG superimposed on a slow oscillation. During 90 minutes of dark adaptation, the EOG oscillates slowly and reaches a quasi–steady state. Thereafter, an incandescent light of about 1,000 lux at eye level is turned on and off every 70 seconds. The abscissa is divided in 30-second intervals, and the lines on the ordinate indicate 50 μV. The *lower* tracing marks the light stimulus.

tance since chloroquine has been replaced by hydroxychloroquine, a drug significantly less toxic.[64] Additional applications of the EOG for the diagnosis of retinal dystrophies appeared in recent publications.[33, 52, 122, 124, 131, 141, 160, 173, 175] The metabolism of the inner retina is supported by the central retinal artery, while the outer retina and the pigment epithelium are dependent on the choroidal circulation. The central retinal artery has been interrupted in animals, and the EOG response was found to be markedly reduced.[46, 107, 154, 163] A similarly diminished response of the slow oscillation of the human EOG has been observed in ischemia from central retinal artery occlusion[121, 157]; the fast oscillation, on the other hand, remained unaffected.[158]

REFERENCES

1. Adams A: The normal electro-oculogram (EOG). *Acta Ophthalmol (Copenh)* 1973; 51:551–561.
2. Afanador AJ, Andrews CE: Rod and cone contribution to the EOG ratio. *Am J Opt Physiol Optics* 1978; 55:101–107.
3. Alanko HI: Clinical electro-oculography. *Acta Ophthalmol Suppl (Copenh)* 1984; 161:139–148.
4. Alexandridis E: Extraretinal influences on the EOG potential. *Bibl Ophthalmol* 1976; 85:86–93.
5. Alexandridis E, Ariely E, Gronau G: Einfluss der Bulbuslage und der Bulbuslänge auf das EOG. *Graefes Arch Ophthalmol* 1975; 194:237–241.
6. Anderson ML, Purple RL: Circadian rhythms and variability of the clinical electro-oculogram. *Invest Ophthalmol Vis Sci* 1980; 19:278–88.
7. Arden GB, Barrada A: Analysis of the electro-oculograms of a series of normal subjects. Role of the lens in the development of the standing potential. *Br J Ophthalmol* 1962; 46:468–481.
8. Arden GB, Barrada A, Kelsey JH: New clinical test of retinal function based upon the standing potential of the eye. *Br J Ophthalmol* 1962; 46:449–467.
9. Arden GB, Kelsey JH: Changes produced by light in the standing potential of the human eye. *J Physiol (Lond)* 1962; 161:189–204.
10. Arden GB, Kelsey JH: Some observations on the relationship between the standing potential of the human eye and the bleaching and regeneration of visual purple. *J Physiol (Lond)* 1962; 161:205–226.
11. Aschoff U: Skotopische und photopische Anteile der Hell- und Dunkelschwingung im Elektrookulogramm. *Dev Ophthalmol* 1981; 4:149–166.
12. Aschoff U: Skotopische und photopische Anteile des Hellanstiegs im Elektrookulogramm (EOG). *Ber Dtsch Ophthalmol Ges* 1979; 76:429–435.
13. Aserinsky E: Effects of illumination and sleep upon amplitude of electro-oculogram. *Arch Ophthalmol* 1955; 53:542–546.
14. Balik J, van Lith GHM: The influence of glucose loading on the electro-oculographic ratio (EOG) in normal subjects and diabetics. *Acta Ophthalmol (Copenh)* 1970; 48:1097–1101.
15. Barber C, Galloway NR: Automated electro-oculography. *Trans Ophthalmol Soc UK* 1973; 93:269–275.
16. Barber C, Galloway NR: The automated electro-oculogram and its clinical uses. *Doc Ophthalmol Proc Ser* 1974; 4:507–511.
17. Benson DW Jr, Kolder H, Homer LD: Non-linear response of the human corneoretinal potential to sinusoidal changes in light intensity. *Pflügers Arch* 1967; 295:361–368.
18. Blach RK, Barrie J, Kolb H: Electrical activity of the eye in high myopia. *Br J Ophthalmol* 1966; 50:629–641.
19. Braakhuis WJM, Thijssen JM: Standardized electro-ophthalmology by using self-calibrating equipment. *Doc Ophthalmol Proc Ser* 1978; 15:317–318.
20. Cox SN, Weinstein G, Arden GB, Bird AC: The effect of acetazolamide on electro-oculogram potential (abstract 87). *Invest Ophthalmol Vis Sci* 1988; 29(suppl):146.
21. Dawis S, Hofmann H, Niemeyer G: The electroretinogram, standing potential, and light peak of the perfused cat eye during acid-base changes. *Vision Res* 1985; 25:1163–1177.
22. Dawis SM, Niemeyer G: Dopamine influences the light peak in the perfused mammalian eye. *Invest Ophthalmol Vis Sci* 1986; 27:330–335.
23. Dawson WM, Maida TM: Reduced variability in the electro-oculogram. *Am J Ophthalmol* 1984; 97:395–396.
24. Dayton GO, Jones MH, Aiu P, Rawson RA, Stella B, Rose M: Developmental study of coordinated eye movements in the human infant. I. Visual acuity in the newborn human: A study based on induced optokinetic nystagmus recorded by electro-oculography. *Arch Ophthalmol* 1964; 71:865–870.
25. De Rouck A, Kayembe D: A clinical procedure for the simultaneous recording of fast and slow EOG oscillations. *Int Ophthalmol* 1981; 3:179–189.
26. Dodt E, Baier M: Area-luminance relationship of a constant light peak of the standing potential in the human eye. *Ophthalmologica* 1984; 188:232–238.
27. Doft BH, Burns SA, Elsner A: The inverse electro-oculogram. *Br J Ophthalmol* 1982; 66:379–381.
28. du Bois-Reymond E: *Untersuchungen über die thierische Elektrizität*, vol 2. Berlin, Reimer, 1849, pp 256–257.
29. Ehrenfeld M, Nesher R, Merin S: Delayed-onset chloroquine retinopathy. *Br J Ophthalmol* 1986; 70:281–283.
30. Elenius V, Aantaa E: Effects of duration and intensity of illumination on the light-induced change in the amplitude of the human electro-oculogram. *Doc Ophthalmol Proc Ser* 1974; 4:205–209.
31. Elenius V, Aantaa E: Light-induced increase in amplitude of electro-oculogram evoked with blue and red lights in totally color-blind and normal humans. *Arch Ophthalmol* 1973; 90:60–63.
32. Elenius V, Lehtonen J: Spectral sensitivity of the standing potential of the human eye. *Acta Ophthalmol (Copenh)* 1962; 40:559–566.
33. Fishman GA, Young RSL, Schall SP, Vasquez VA: Electro-oculogram testing in fundus flavimaculatus. *Arch Ophthalmol* 1979; 97:1896–1898.
34. Franck H, Muller JC, Bronner A: Some consider-

ations regarding the effect of antimalarial drugs on the EOG and the 'macular EOG.' *Doc Ophthalmol Proc Ser* 1978; 15:159–163.
35. François J, De Rouck A, Cambie E, Zanen A: *L'Electro-diagnostic des Affections Retiniennes*, part 3. Le Potential de Repos (Electro-oculographie, EOG). Paris, Masson & Cie, 1974, pp 151–188.
36. François J, De Rouck A, Jönsas C, Fernandez-Sasso D: The rabbit's electro-oculogram. *Acta Ophthalmol (Copenh)* 1969; 47:984–990.
37. François J, De Rouck A, Verriest G, Szmigielski M: An extended clinical test of the ocular standing potential and its results in some cases of retinal degeneration. *Jpn J Ophthalmol* 1966; 10(suppl):257–268.
38. François J, Szmigielski M, Verriest G, DeRouck A: The influence of changes in illumination on the standing potential of the human eye. *Ophthalmologica* 1965; 150:83–91.
39. François J, Verriest G, De Rouck A: Electro-oculography as a functional test in pathological conditions of the fundus: II. Base value and drop during dark adaptation. *Br J Ophthalmol* 1956; 40:305–312.
40. François, J, Verriest G, De Rouck A: Modification of the amplitude of the human electro-oculogram by light and dark adaptation. *Br J Ophthalmol* 1955; 39:398–408.
41. Frey RG: Elektrooculographische Registrierung von disjunktiven Augenbewegungen und von Blickbewegungen unter Prismenbelastung. *Graefes Arch Ophthalmol* 1963; 166:345–358.
42. Gallemore RP, Griff ER, Steinberg RH: Evidence in support of a photoreceptoral origin for the "light-peak substance". *Invest Ophthalmol Vis Sci* 1988; 29:566–571.
43. Garcia-Franco A, Dalmau RM: Contribution to the study of ocular interdependence by electro-oculography. *Doc Ophthalmol Proc Ser* 1976; 10:193–199.
44. Gliem H: *Das Elektrookulogramm. Ein Erfahrungsbericht*. Leipzig, Germany, Georg Thieme Verlag, 1971.
45. Gouras P: Clinical electro-oculography, in Straatsma BR, Hall MO, Allen RA, Crescitelli F (eds): *The Retina, Morphology, Fundus and Clinical Characteristics*. Los Angeles, University of California Press, 1969, pp 565–581.
46. Gouras P, Carr RE: Light-induced DC response of monkey retina before and after central retinal artery interruption. *Invest Ophthalmol* 1965; 4:310–317.
47. Gouras P, Gunkel RD: The EOG in chloroquine and other retinopathies. *Arch Ophthalmol* 1963; 70:629–639.
48. Griff ER, Linsenmeier RA, Steinberg RH: The cellular origin of the fast oscillation. *Doc Ophthalmol Proc Ser* 1983; 37:13–20.
49. Griff ER, Steinberg RH: Origin of the light peak: In vitro study of *Gekko gekko*. *J Physiol (Lond)* 1982; 331:637–652.
50. Hache JC, François P, Goeminne P: Macular electro-oculography. *Doc Ophthalmol Proc Ser* 1976; 10:37–41.
51. Hansen RM, Fulton AB: Corneofundal potentials in human infants. *Doc Ophthalmol Proc Ser* 1983; 37:81–86.
52. Heckenlively JR: The electro-oculogram, in Heckenlively JR (ed): *Retinitis Pigmentosa*. Philadelphia, JB Lippincott, 1988, pp 10–11.
53. Heilig P, Thaler A, Scheiber V: The initial phase of the EOG-oscillation. *Doc Ophthalmol Proc Ser* 1978; 15:149–150.
54. Henkes HE, Denier van der Gon JJ, van Marle GW, Schreinemachers HP: Electro-oculography. A semi-automatic recording procedure. *Br J Ophthalmol* 1968; 52:122–126.
55. Henkind P, Carr RE, Siegel IM: Early chloroquine retinopathy: Clinical and functional findings. *Arch Ophthalmol* 1964; 71:157–165.
56. Hennig J, Täumer R, Pernice D: The fast ocular dipole oscillation. *Doc Ophthalmol Proc Ser* 1974; 4:449–455.
57. Hochgesand P, Hackenberg K: The foveal electro-oculogram. *Doc Ophthalmol Proc Ser* 1974; 4:217–228.
58. Hochgesand P, Schicketanz KH: Wieweit lässt sich durch die Faktoren Person, Tag und Tageszeit die Variabilität des EOG erklären? *Klin Monatsbl-Augenheilkl* 1974; 164:389–392.
59. Hofmann H, Niemeyer G: Calcium blocks selectively the EOG–light peak. *Doc Ophthalmol* 1985; 60:361–368.
60. Höhne W: Zur Auswertung des Elektrookulogramms. *Graefes Arch Ophthalmol* 1974; 192:36–47.
61. Homer LD, Kolder H: Mathematical model of oscillations in the human corneoretinal potential. *Pflügers Arch Gesamte Physiol* 1966; 287:197–202.
62. Homer LD, Kolder HEJW: The oscillation of the human corneoretinal potential at different light intensities. *Pflügers Arch Gesamte Physiol* 1967; 296:133–142.
63. Homer LD, Kolder H, Benson DW Jr: Parameter variations of a model of the oscillation of the human corneoretinal potential. *Pflügers Arch Gesamte Physiol* 1967; 294:103–112.
64. Infante R, Martin DA, Heckenlively JR: Hydroxychloroquine and retinal toxicity. *Doc Ophthalmol Proc Ser* 1983; 37:121–126.
65. Jackson J, Kolder HE, Homer LD, Tigges J: EOG and experimental optic nerve transection. *Doc Ophthalmol Proc Ser* 1983; 37:35–40.
66. Jackson SA: The optimum illuminance level for clinical electro-oculography. *Acta Ophthalmol (Copenh)* 1979; 57:665–668.
67. Jacobs L, Feldman M, Rabinowitz M, Bender MB: Alterations of the corneo-fundal potential of the eye during sleep. *EEG Clin Neurophysiol* 1973; 34:579–586.
68. Jarkman S: Effects of low doses of forskolin on the c-wave of the direct current electroretinogram and on the standing potential of the eye. *Doc Ophthalmol* 1988; 67:305–314.
69. Jarkman S, Skoog K-O, Nilsson SEG: The c-wave of the electroretinogram and the standing potential of the eye as highly sensitive measures of effects of low doses of trichloroethylene, methylchloroform, and halothane. *Doc Ophthalmol* 1985; 60:375–382.
70. Jones RM, Stevens TS, Gould S: Normal EOG values of young subjects. *Doc Ophthalmol Proc Ser* 1977; 13:93–97.
71. Kawasaki K, Mukoh S, Yonemura D, Fujii S,

Segawa Y: Acetazolamide-induced changes of the membrane potentials of the retinal pigment epithelial cell. *Doc Ophthalmol* 1986; 63:375–381.
72. Kawasaki K, Yonemura D, Madachi-Yamamoto S: Hyperosmolarity response of ocular standing potential as a clinical test for retinal pigment epithelium activity: Diabetic retinopathy. *Doc Ophthalmol* 1984; 58:375–384.
73. Kawasaki K, Yonemura D, Mukoh S, Tanabe J: Hyperosmolarity-induced changes in the transepithelial potential of the human and frog retinae. *Doc Ophthalmol Proc Ser* 1983; 37:29–33.
74. Kelsey JH: Variations in the normal electro-oculogram. *Br J Ophthalmol* 1967; 51:44–49.
75. Kikawada N: Variations in the corneo-retinal standing potential of the vertebrate eye during light and dark adaptations. *Jpn J Physiol* 1968; 18:687–702.
76. Knave B, Persson HE, Nilsson SEG: A comparative study on the effects of barbiturate and ethyl alcohol on retinal function with special reference to the c-wave of the electroretinogram and the standing potential of the sheep eye. *Acta Ophthalmol (Copenh)* 1974; 52:254–259.
77. Kolb H: Electro-oculogram findings in patients treated with antimalarial drugs. *Br J Ophthalmol* 1965; 49:573–590.
78. Kolder H: Spontane und experimentelle Änderungen des Bestandpotentials des menschlichen Auges. *Pflugers Arch Gesamte Physiol* 1959; 268:258–272.
79. Kolder HE: Electro-oculography. *Ophthalmologica* 1974; 169:127–140.
80. Kolder H, Brecher GA: Fast oscillations of the corneoretinal potential in man. *Arch Ophthalmol* 1966; 75:232–237.
81. Kolder HE, Hochgesand P: Empirical model of electro-oculogram. *Doc Ophthalmol* 1973; 34:229–241.
82. Kolder H, North AW: Oscillations of the cornea-retinal potential in animals. *Ophthalmologica* 1966; 152:149–160.
83. Kolder H, Scarpatetti R: Registrierung von Änderungen des Bestandpotentials des menschlichen Auges. *Pflugers Arch Gesamte Physiol* 1958; 267:295–298.
84. Kooijman AC, van Norren D, De Sera P, Thijssen GL, van der Heijde GL, Reits D, van der Wildt GJ, Bijl GK: Minimum procedures for visual electrodiagnostic testing. *Doc Ophthalmol* 1986; 62:13–18.
85. Kreienbühl B, Niemeyer G: Standing potential and c-wave during changes in pO_2 and flow in the perfused cat eye. *Doc Ophthalmol* 1985; 60:353–360.
86. Kris C: Corneo-fundal potential variations during light and dark adaptation. *Nature* 1958; 182:1027–1028.
87. Kris C: Diurnal variation in periorbitally measured eye potential level. *Clin Neurophysiol* 1957; 9:382.
88. Kris C: Electro-oculography, in Glasser O (ed): *Medical Physics*, vol 3. Chicago, Year Book Medical Publishers, Inc, 1960, pp 692–700.
89. Krogh E: Inter- and intraindividual variation of the EOG. An assessment and some possible contributing factors. *Bibl Ophthalmol* 1976; 85:115–118.
90. Krogh E: Normal values in clinical electrooculography. III. Numerical evaluation of two dimensionless EOG parameters. *Acta Ophthalmol (Copenh)* 1977; 55:227–236.
91. Krogh E: Normal values in clinical electrooculography. IV. Analysis of two dimensionless EOG parameters and their relation to other variables. *Acta Ophthalmol (Copenh)* 1977; 55:739–749.
92. Krogh E: Normal values in clinical electrooculography. V. Variability within and between eyes. *Acta Ophthalmol (Copenh)* 1977; 55:986–993.
93. Krüger CJ: Der Anteil zentraler und peripherer Netzhautbezirke an der langsamen Hellschwingung im Elektrookulogramm (EOG). *Ber Dtsch Ophthalmol Ges* 1981; 78:741–749.
94. Krüger CJ, Baier M: Increment threshold function of the light-induced slow oscillation of the EOG. *Doc Ophthalmol Proc Ser* 1983; 37:75–80.
95. Kurihara H, Nikara T: Changes of the fast oscillations in diabetic retinopathy and use of a new computerized EOG device. *Doc Ophthalmol Proc Ser* 1983; 37:143–150.
96. Lessel MR, Thaler ARG, Heilig P: The fast oscillation of the electro-oculogram. Amplitudes and latencies in the course of the slow oscillation. *Doc Ophthalmol Proc Ser* 1983; 37:131–135.
97. Lessel MR, Thaler A, Heilig P: The peak latency of the slow oscillation in EOG. *Doc Ophthalmol* 1989; 71:409–412.
98. Linsenmeier RA: Effects of light and darkness on oxygen distribution and consumption in the cat retina. *J Gen Physiol* 1986; 88:521–542.
99. Linsenmeier RA, Steinberg RH: A light-evoked interaction of apical and basal membranes of retinal pigment epithelium: c-Wave and light peak. *J Neurophysiol* 1983; 50:136–147.
100. Linsenmeier RA, Steinberg RH: Origin and sensitivity of the light peak in intact cat eye. *J Physiol (Lond)* 1982; 331:653–673.
101. Madachi-Yamamoto S, Yonemura D, Kawasaki K: Diamox response of ocular standing potential as a clinical test for retinal pigment epithelium activity: Normative data. *Acta Soc Ophthalmol Jpn* 1984; 88:1267–1272.
102. Madachi-Yamamoto S, Yonemura D, Kawasaki K: Hyperosmolarity response of ocular standing potential as a clinical test for retinal pigment epithelial activity. Normative Data. *Doc Ophthalmol* 1984; 57:153–162.
103. Marg E: Development of electro-oculography: Standing potential of the eye in registration of eye movements. *Arch Ophthalmol* 1951; 45:169–185.
104. Marmor MF, Donovan WJ, Gaba DM: Effects of hypoxia and hyperoxia on the human standing potential. *Doc Ophthalmol* 1985; 60:347–352.
105. Miles WR: Modification of the human-eye potential by dark and light adaptation. *Science* 1940; 91:456.
106. Missotten L, Goethals M: Timolol reduces the standing potential of the eye. *Ophthalmic Res* 1977; 9:321–323.
107. Miyake Y: Electrooculographic change in retinal arterial occlusion and its analysis. *Acta Soc Ophthalmol Jpn* 1974; 78:311–323.
108. Momirov D, van Lith GHM, van der Torren K, Vijfvinkel-Bruinenga S: Normal values of the basic

level of the standing potential and its light rise. *Ophthalmologica* 1982; 184:225–231.
109. Mukoh S, Kawasaki K, Yonemura D, et al: Hyperosmolarity-induced hyperpolarization of the membrane potential of the retinal pigment epithelium. *Doc Ophthalmol* 1985; 60:369–374.
110. Müller W, Haase E: Inter- und intraindividuelle Streuung im EOG. *Graefes Arch Ophthalmol* 1970; 181:71–78.
111. Niemeyer G, Steinberg RH: Differential effects of pCO_2 and pH on the ERG and light peak of the perfused cat eye. *Vision Res* 1984; 24:275–280.
112. Nilsson SEG, Andersson BE: Corneal D.C. recordings of slow ocular potential changes such as the ERG c-wave and the light peak in clinical work. Equipment and examples of results. *Doc Ophthalmol* 1988; 68:313–325.
113. Nilsson SEG, Armstrong D, Koppang N, Persson P, Milde K: Studies on the retina and the pigment epithelium in hereditary canine ceroid lipofuscinosis. IV. Changes in the electroretinogram and the standing potential of the eye. *Invest Ophthalmol Vis Sci* 1983; 24:77–84.
114. Nilsson SEG, Skoog K-O: Covariation of the simultaneously recorded c-wave and standing potential of the human eye. *Acta Ophthalmol (Copenh)* 1975; 53:721–730.
115. North AW: Accuracy and precision of electro-oculographic recording. *Invest Ophthalmol* 1965; 4:343–348.
116. Oakley B II: Potassium and the photoreceptor-dependent pigment epithelium hyperpolarization. *J Gen Physiol* 1977; 70:405–425.
117. Pasik P, Pasik T, Bender MB: Recovery of the electro-oculogram after total ablation of the retina in monkeys. *EEG Clin Neurophysiol* 1965; 19:291–297.
118. Pernice D, Täumer R, Rohde N, Hennig J: Several clinical EOG-tests—an overview. Proposal of a varied EOG test. *Bibl Ophthalmol* 1976; 85:25–34.
119. Pinckers A: Clinical electro-oculography. *Acta Ophthalmol (Copenh)* 1979; 57:623–632.
120. Plonsey R: Quantitative formulations of electrophysiological sources of potential fields in volume conductors. *IEEE Trans Biomed Eng* 1984; 31:868–872.
121. Ponte F, Lodato G: Electro-oculographic investigations in central retinal vessel occlusions. *Doc Ophthalmol Proc Ser* 1978; 15:155–157.
122. Priem HA, De Rouck A, De Laey J-J, Bird AC: Electrophysiologic studies in birdshot chorioretinopathy. *Am J Ophthalmol* 1988; 106:430–436.
123. Reeser F, Weinstein GW, Feiock KB, Oser R: Electro-oculography as a test of retinal function. The normal and supernormal EOG. *Am J Ophthalmol* 1970; 70:505–514.
124. Ripps H, Noble KG, Greenstein VC, Siegel IM, Carr RE: Progressive cone dystrophy. *Ophthalmology* 1987; 94:1401–1409.
125. Rohde N, Täumer R: Correlation between the c-wave and the corneoretinal potential in man. *Ophthalmic Res* 1982; 14:442–449.
126. Rohde N, Täumer R, Bleckmann H: Examination of the fast oscillation of the corneoretinal potential under clinical conditions. *Graefes Arch Ophthalmol* 1981; 217:79–90.
127. Rohde N, Täumer R, Braas F: An EOG computer and stimulator for the investigation of the slow retinal potential. Automation of the clinical EOG-test. *Bibl Ophthalmol* 1976; 85:75–83.
128. Ronchi L, Serra A: EOG driven by the contralateral reading eye. *Doc Ophthalmol Proc Ser* 1983; 37:87–91.
129. Röver J, Bach M: C-wave versus electrooculogram in diseases of the retinal pigment epithelium. *Doc Ophthalmol* 1987; 65:385–391.
130. Rudolf G, Wioland N, Kempf E, Bonaventure N: Electrooculographic study in the chicken after treatment with neurotoxin 6-hydroxydopamine. *Doc Ophthalmol* 1989; 72:83–91.
131. Sabates R, Pruett RC, Hirose T: The electrooculogram in 'vitelliform' macular lesions. *Doc Ophthalmol Proc Ser* 1983; 37:93–103.
132. Sato T, Yoneyama T, Kim HK, Suzuki TA: Effect of dopamine and haloperidol on the c-wave and light peak of light-induced retinal responses in chick eye. *Doc Ophthalmol* 1987; 65:87–95.
133. Sato T, Yoneyama T, Kim HK, Matsumoto N, Suzuki TA: Gamma-aminobutyric acid– and picrotoxin-induced changes in c-wave and light peak of retinal potentials in the chick. *Doc Ophthalmol* 1987; 65:333–342.
134. Skoog K-O: The directly recorded standing potential of the human eye. *Acta Ophthalmol (Copenh)* 1975; 53:120–132.
135. Skoog K-O, Textorius O, Nilsson SEG: Effects of ethyl alcohol on the directly recorded standing potential of the human eye. *Acta Ophthalmol (Copenh)* 1975; 53:710–720.
136. Skrandies W, Baier M: The standing potential of the human eye reflects differences between upper and lower retinal areas. *Vision Res* 1986; 26:577–581.
137. Stanziano G, Kaplan H, Koblasz A, Davey K: Microcomputer analysis of clinical ERG, EOG, and other tests of retinal function. *Med Instrum* 1988; 22:12–19.
138. Steinberg RH: Interactions between the retinal pigment epithelium and the neural retina. *Doc Ophthalmol* 1985; 60:327–346.
139. Steinberg RH, Griff ER, Linsenmeier RA: The cellular origin of the light peak. *Doc Ophthalmol Proc Ser* 1983; 37:1–11.
140. Stepanik J: Das Bestandpotential des Auges in der experimentellen Steigerung des intraocularen Druckes beim Menschen. *Graefes Arch Ophthalmol* 1958; 160:226–235.
141. Takahashi Y, Onoe S, Asamizu N, Mori T, Yoshimura Y, Tazawa Y: Incomplete congenital stationary night blindness: Electroretinogram c-wave and electrooculogram light rise. *Doc Ophthalmol* 1988; 70:67–75.
142. Täumer R (ed): Electro-oculography—its clinical importance. *Bibl Ophthalmol* 1976; 85:134.
143. Täumer R, Hennig J, Pernice D: The ocular dipole—a damped oscillator stimulated by the speed of change in illumination. *Vision Res* 1974; 14:637–645.
144. Täumer R, Hennig J, Wolff L: Further investigations

concerning the fast oscillation of the retinal potential. *Bibl Ophthalmol* 1976; 85:57–67.
145. Täumer R, Kapp H, Hennig J, Rohde N: A first electrical analog model of the ODM-oscillations. *Doc Ophthalmol Proc Ser* 1974; 4:457–463.
146. Täumer R, Mackensen G, Hartmann H, Moser U, Stehle R, Werner W, Wolf D: Verhalten des "Bestandpotentials" des menschlichen Auges nach Belichtungsänderungen. *Graefes Arch Ophthalmol* 1974; 189:81–97.
147. Täumer R, Rohde N, Haubach R: The slow dark oscillation of the ocular dipole moment (ODM)—dependence upon the amplitude of the step to darkness. *Doc Ophthalmol Proc Ser* 1976; 10:363–369.
148. Täumer R, Rohde N, Hennig J, Wolff L: Ocular dipole moment (ODM)—variation excited by photoflash. *Doc Ophthalmol Proc Ser* 1976; 10:357–362.
149. Täumer R, Rohde N, Pernice D: The slow oscillation of the retinal potential: A biochemical feedback stimulated by the activity of the rods and cones. *Bibl Ophthalmol* 1976; 85:40–56.
150. Täumer R, Rohde N, Pernice D, Kohler U: EOG: Light test and dark test. *Graefes Arch Ophthalmol* 1976; 199:207–213.
151. Tazawa Y, Mera H, Kondo T: Development of a new semi-automatic recorder for visual electric responses—EOG, ERG, ERP and ENG. *Jpn J Ophthalmol* 1979; suppl:287–292.
152. Ten Doesschate G, Ten Doesschate J: The influence of the state of adaptation on the resting potential of the human eye. *Ophthalmologica* 1956; 132:308–320.
153. Textorius O, Nilsson SEG: Effects of intraocular irrigation with melatonin on the c-wave of the direct current electroretinogram and on the standing potential of the eye in albino rabbits. *Doc Ophthalmol* 1987; 65:97–111.
154. Thaler A, Heilig P: EOG and ERG components in ischemic retinopathy. *Ophthalmic Res* 1977; 9:38–46.
155. Thaler A, Heilig P: The steady state in EOG. *Doc Ophthalmol Proc Ser* 1974; 4:211–216.
156. Thaler A, Heilig P, Gordesch J: Light peak to dark trough ratio in clinical electro-oculography. Influence of dark oscillations on the following light peak. *Bibl Ophthalmol* 1976; 85:110–114.
157. Thaler A, Heilig P, Lessel MR: Ischemic retinopathy: Reduced light peak and dark trough amplitudes in electrooculography. *Doc Ophthalmol Proc Ser* 1977; 13:119–121.
158. Thaler A, Heilig P, Scheiber V: The initial phase of the EOG-oscillation in ischemic retinopathy. *Doc Ophthalmol Proc Ser* 1978; 15:151–153.
159. Thaler ARG, Lessel MR, Heilig P: Light induced oscillations of the standing potential in achromatopsia. *Doc Ophthalmol* 1986; 63:333–336.
160. Thaler ARG, Lessel MR, Heilig P: The fast oscillation of the electro-oculogram in sectorial retinitis pigmentosa. *Doc Ophthalmol Proc Ser* 1983; 37:137–141.
161. Thaler A, Lessel MR, Heilig P: The slow electrooculogram oscillation in malignant choroidal melanoma. *Doc Ophthalmol* 1989; 71:403–407.
162. Thaler ARG, Lessel MR, Heilig P, Schreiber V: The fast oscillation of the electro-oculogram. Influence of stimulus intensity and adaptation time on amplitude and peak latency. *Ophthalmic Res* 1982; 14:210–214.
163. Thaler, Snyder JE, Kolder HE, Hayreh SS: Oscillations of the electroretinogram and electrooculogram in experimental ischemic retinopathy. *Ophthalmic Res* 1978; 10:283–289.
164. Thijssen JM, Pinckers A: Contralateral effects in the electro-oculogram. *Acta Ophthalmol (Copenh)* 1974; 52:441–454.
165. Thijssen JM, Pinckers A: Contralateral effect in the electro-oculogram: Experimental verification and clinical implications. *Doc Ophthalmol Proc Ser* 1976; 10:201–211.
166. Toyama K, Watanabe I: The relationship between the EOG and the ERG recorded with DC-amplifier in the experimental animal. *Jpn J Ophthalmol* 1979; suppl:135–140.
167. Trimble JL, Ernest JT, Newell FW: Electro-oculography in infants. *Invest Ophthalmol Vis Sci* 1977; 16:668–670.
168. Valeton JM, van Norren D: Intraretinal recordings of slow electrical responses to steady illumination in monkey: Isolation of receptor responses and the origin of the light peak. *Vision Res* 1982; 22:393–399.
169. Van Lith GHM, Balik J: The variability of the EOG in the same person. *Ophthalmologica* 1971; 163:63–64.
170. Van Lith GHM, Balik J: Variability of the electro-oculogram (EOG). *Acta Ophthalmol (Copenh)* 1970; 48:1091–1096.
171. Van Norren D, Heynen HGM: Origin of the fast oscillation in the macaque. *Doc Ophthalmol Proc Ser* 1983; 37:73.
172. Van Norren D, Valeton JM: Relation between the EOG, corneal-, intraretinal slow potentials. *Doc Ophthalmol Proc Ser* 1982; 31:104.
173. Weleber RG: Fast and slow oscillations of the electro-oculogram in Best's macular dystrophy and retinitis pigmentosa. *Arch Ophthalmol* 1989; 107:530–537.
174. Yonemura D, Kawasaki K, Madachi-Yamamoto S: Hyperosmolarity response of ocular standing potential as a clinical test for retinal pigment epithelium activity. Chorioretinal dystrophies. *Doc Ophthalmol* 1984; 57:163–173.
175. Yonemura D, Kawasaki K, Wakabayashi K, Tanabe J: EOG application for Stargardt's disease and X-linked juvenile retinoschisis. *Doc Ophthalmol Proc Ser* 1983; 37:115–120.
176. Zonneveldt A, van Lith G: The electrooculogram and its interindividual and intraindividual variability. *Ophthalmologica* 1980; 181:165–169.
177. Zrenner E, Riedel KG, Adamczyk R, Gilg T, Liebhardt E: Effects of ethyl alcohol on the electrooculogram and color vision. *Doc Ophthalmol* 1986; 63:305–312.