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Intraoperative retinal light damage reflected in electrophysiologic data

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Abstract. In a series of 30 unilaterally pseudophakic patients, electroretinograms and electro-oculograms were recorded 6 months postoperatively. The unoperated on fellow eyes served as controls. High intraoperative retinal light exposure (3.4–7.3 mW/cm^2, Zeiss OPMI 6 operating microscope) caused a substantial reduction of electrophysiologic potentials. Light protection prevented deterioration of electroretinogram and electro-oculogram potentials; reducing the bulb voltage, tilting the axis of illumination, filtering short wavelengths and the use of light shields resulted in 4-log-unit lower intensities (0.8–3.7 μW/cm^2).

Abbreviations: ACL, anterior chamber lens; ECCE, extracapsular cataract extraction; ICCE, intracapsular cataract extraction; PCL, posterior chamber lens

Introduction

Ophthalmoscopically detectable retinal light-induced maculopathy induced by the coaxial illumination of the operating microscope has been known since the study by McDonald and Irvine in 1983 [1]. The retina may suffer from intraoperative light exposure without showing obvious morphologic changes. This ophthalmoscopically invisible retinal and pigment epithelial impairment has been documented by means of electrophysiologic examinations [2-5]. The results of these electrophysiologic studies are not apparent to most cataract surgeons. In this study, functional deficits were quantified objectively by means of electrophysiologic ophthalmologic methods.

Table 1. Mean and standard deviation of visual acuity from 30 pseudophakic patients examined in this study.

	Visual acuity	
	Operated on eye	Non-operated on eye
ICCE/ACL (n = 15)	1.00 ± 0.18	0.77 ± 0.25
ECCE/PCL (n = 15)	0.90 ± 0.26	0.85 ± 0.31

Materials and methods

An electrophysiologic study was performed in a series of 30 patients six months after cataract surgery in one eye of each patient. Informed consent was given by all patients. Patients with ophthalmologic diseases other than cataract were excluded from this study. In 15 patients (12 women and 3 men aged 50–83 years: mean ± SD age, 68.2 ± 10.2 years) planned extracapsular extraction (ECCE) and posterior chamber lens (PCL) implantation had been performed; in 15 patients (14 women and 1 man aged 35–86 years; mean ± SD age, 63.7 ± 17.14 years) intracapsular extraction (ICCE) and anterior chamber lens (ACL) implantation had been performed. No retinal or pigment epithelial changes were detectable biomicroscopically. Results of Amsler testing and color vision testing (Farnsworth D-15-hue/saturated/desaturated, Ishihara) were normal. No patient in either the ICCE or the ECCE group stated any complaints relative to possible light damage after operation during the follow-up time. Visual acuities of the patients are shown in Table 1.

Surgery was performed by three different surgeons. The duration of light exposure during surgery was 17 ± 5 minutes for the ICCE procedure and 19 ± 7 minutes for the ECCE procedure.

The light output (radiant energy) from the operating microscope (Zeiss/OPMI 6 S, tungsten bulb, 6 V, 30 W) at different voltage settings used for ICCE and ECCE was measured with a pyroclectric radiometer (Molectron PR200) (Table 2, Fig. 1). In ECCE the light intensities used during surgery were 3.4–7.3 mW/cm^2. In ICCE the light intensities measured at the level of the eye were 3–4 log units lower (0.8–3.7 μW/cm^2) because in ECCE the surgeon used the maximal light setting of the OPMI 6, not being aware of possible light damage, and in ICCE the intensity of the illumination light was reduced to the lowest intensities necessary for surgery. The surgeon performing the ECCE procedure needed higher light intensities, e.g., when using the 'red reflex' during capsule polishing. A corneal light shield

Table 2. Radiant energies for operating microscope Zeiss OPMI 6 S, tungsten bulb, 6 V, 30 W, with a measurement distance of 175 mm*

Bulb voltage (VAC)	Power density	
	With eye shield with filter ($\mu W/cm^2$)	Without eye shield without filter (mW/cm^2)
1.5	0.8	0.1
2.0	1.7	0.3
2.5	3.7	0.7
3.0	6.8	1.4
3.5	12.4	2.6
4.0	19.2	3.4
4.5	27.5	4.9
5.0	39.4	7.3

* Note the 3-log-unit higher power density in the right column. VAC = volts of alternating current.

(Merocel No. 400101) was used in ICCE but not in ECCE. In addition, the illumination light of the OPMI 6 was filtered with a Schott GG475 (yellow-tinted) edge filter in the ICCE procedure, but in ECCE no such filter was used. The spectral distribution of the output was analyzed with a spectrometer and plotted on an x-y recorder (Figure 2).

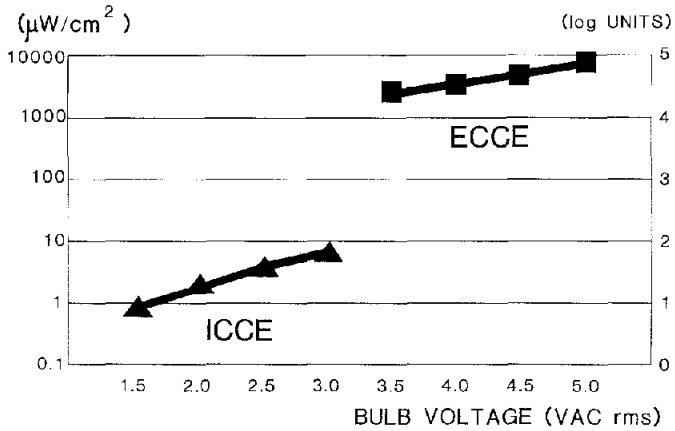

Fig. 1. Radiation output of the operation microscope at different bulb voltage settings, measured at the level of the eye. Left ordinate gives the radiometer reading. VAC rms = volts of alternating current, root mean square. Squares represent light intensities used in ECCE (without light protection); triangles, light intensities used in ICCE (with light protection: eye shield and Schott GG475 filter).

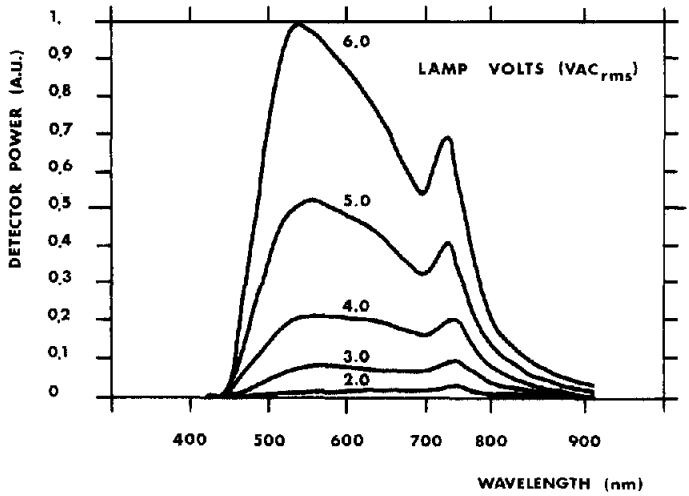

Fig. 2. Spectral distribution of the light output of the operating microscope, measured with a SPEX Monochromator. A.U. = arbitrary units, VAC rms = volts of alternating current, root mean square. The peaks at 750 nm are artifacts due to the extraordinary grating efficiency of the analyzer in this frequency region.

The electrophysiologic tests included the recording of rod- and cone-mediated electroretinograms (ERGs) and the recording of the slow oscillation of the electro-oculogram (EOG). It is assumed that there is no difference in conduction of currents in eyes with ACLs and PCLs.

For ERG, all pupils were dilated with 1 % cyclopentolate. After 20 minutes of preadaptation in a dimly illuminated room (50 lux), Henkes contact lens electrodes (Lovac 1126A) were placed on the cornea with the patient under local anesthesia. Silver–silver chloride skin electrodes (Beckmann, 39170) located on the forehead were used for reference. Ground was connected to the eyelids via the outer contact of the lens electrodes. The face of the patient was placed in the center of a semicircular globe (diameter, 30 cm) to ensure ganzfield stimulation (Ulbricht principle, [5]). Rectangular light stimuli (electromechanical shutter 150-W xenon arc lamp) were projected into the globe. The potentials were amplified, optically coupled and filtered by passive filters (AM502, Tektronix). Cutoff frequencies (3 dB) were set to 0.1 Hz and 10 kHz. The rod-mediated ERGs were recorded after 30 minutes of dark adaptation. Ten rectangular light stimuli (65 cd/m^2, 5-ms duration, 30-second interstimulus interval) were applied. The cone-mediated components were recorded after 10 minutes of light adaptation (33 cd/m^2). One hundred responses were elicited by rectangular stimuli of 3 cd·m^{-2}·s intensity (luminosity × time), 5-ms duration each.

For EOG, the patient was placed in front of a 3/4 sphere, 35 cm in

diameter (ganzfield stimulation, Ulbricht principle [5]). Ten light-emitting diodes serving as a fixation aid were mounted equidistantly in a horizontal row in the sphere and were triggered sequentially. Repetitive horizontal eye movements of 30 degrees and a repetition rate of 2.5 seconds were thus evoked. Silver–silver chloride electrodes (Beckmann, 39170) were fixed at the outer and inner canthi. The floating ground was connected to the midforehead. The pupils were not dilated. After a 35-minute period of dark adaptation, the light-induced increase of the slow oscillation of the standing potential of the eye was elicited by a rectangular light stimulus (fluorescent tubes, 950 cd/m^2) of 15-minutes duration. The potentials were filtered (DC to 10 Hz), amplified and processed by computer (PDP 11 E10 [6]).

Measurements of amplitudes and latencies of rod- and cone-mediated ERG, and EOG components were performed by means of a cursor on the display screen in a masked mode. Wilcoxon's rank-sum test was performed to calculate the significance of the difference of electrophysiologic data between operated on and non-operated on eyes of each surgical procedure (ICCE/ACL and ECCE/PCL) and between the nonoperated on fellow eyes.

Results

Electrophysiologic recordings from eyes exposed to low light intensities during the ICCE and ACL implantation displayed an amplitude increase of the rod- and cone-mediated a- and b-waves and the EOG light peak. After high light exposure of the retina in ECCE and PCL implantation, an amplitude decrease of ERG and EOG components was observed.

Recordings of cone-mediated ERGs and EOGs from pseudophakic eyes after ECCE and implantation of a PCL are shown in Figs. 3 and 4. In Tables 3 and 4 the data of this study as well as the calculated error probabilities are shown.

Differences between the peak latencies of electrophysiologic potentials of operated on and fellow eyes were proven not to be statistically significant ($p > 0.15$). In a comparison of electrophysiologic potentials of the unoperated on fellow eyes of the ICCE and ECCE series, no statistically significant differences of amplitudes ($p > 0.2$) and latencies ($p > 0.1$) were found.

Discussion

In 1954, Littman [7] introduced the use of operating microscopes for ophthalmic surgery. Increasingly higher light intensities were used for better

Table 3. Electrophysiologic data: amplitudes.

	ERG (μV)				EOG slow oscillation (%)
	Rod mediated		Cone mediated		
	a	b	a	b	
ICCE/ACL (n = 15; light output, 0.8 – 3.7 μW/cm^2)					
Operated on eyes	348.5	797.9	126.1	168.0	222.7
(mean ± SD)	±114.9	±198.2	±42.2	±53.9	±34.8
Nonoperated on fellow eyes	321.9	727.4	104.2	142.1	184.5
(mean ± SD)	±96.6	±153.8	±54.1	±70.1	±33.7
Difference (mean ± SEM)	26.5	70.5	21.9	25.9	38.1
	±17.2	±31.2	±10.1	±10.8	±10.2
ECCE/PCL (n = 15; light output, 3.4 – 7.3 mW/cm^2)					
Operated on eyes	254.4	678.4	100.7	139.6	167.5
(mean ± SD)	±113.3	±197.6	±34.2	±66.2	±31.1
Nonoperated on fellow eyes	272.5	692.9	104.7	148.0	195.5
(mean ± SD)	±123.8	±205.5	±36.7	±62.6	±26.4
Difference (mean ± SEM)	−18.1	−14.5	−4.0	−8.4	−28.1
	±19.3	±38.7	±8.6	±6.2	±6.0
Statistics (Wilcoxon's rank-sum test)					
p of difference	0.09	0.09	0.06	0.009	0.0001
p IC/EC	0.2	0.6	0.9	0.8	0.32
Normal (n = 30)*					
Mean + 2SDs	506.4	1006.2	199.8	279.5	256.5
Mean − 2SDs	103.2	397.4	36.1	45.4	138.5
No. of eyes out of normal range					
Operated on eyes (n = 30)	2	4	2	3	6
Nonoperated on fellow eyes (n = 30)	2	2	2	2	2

SD = standard deviation, SEM = standard error of the mean, difference = difference between data of operated on eyes and nonoperated on fellow eyes, *p* = error probability, *p* IC/EC = *p* for comparison of data between nonoperated on fellow eyes for the two surgical procedures (ICCE/ACL and ECCE/PCL).

* Normal range of normal fellow eyes.

Table 4. Electrophysiologic data: latencies.

	ERG (ms)				EOG slow oscillation (min)
	Rod mediated		Cone mediated		
	a	b	a	b	
ICCE/ACL (n = 15; light output, 0.8 – 3.7 μW/cm^2)					
Operated on eyes	31.9	56.1	22.9	47.6	465.4
(mean ± SD)	±1.9	±2.9	±1.6	±1.7	±130.1
Nonoperated on fellow eyes	31.5	56.4	23.1	47.5	481.4
(mean ± SD)	±2.1	±3.0	±2.0	±1.5	±60.2
Difference (mean ± SEM)	0.47	−0.33	0.21	0.07	−16.1
	±0.43	±0.21	±0.46	±0.22	±29.0
ECCE/PCL (n = 15; light output, 3.4 – 7.3 mW/cm^2)					
Operated on eyes	33.3	55.6	23.6	47.7	508.5
(mean ± SD)	±1.9	±2.7	±2.9	±2.6	±68.1
Nonoperated on fellow eyes	32.6	56.6	23.5	48.2	511.3
(mean ± SD)	±2.0	±7.4	±2.2	±2.8	±56.6
Difference (mean ± SEM)	0.73	−1.0	0.07	0.47	−2.7
	±0.44	±1.70	±0.32	±0.27	±9.81
Statistics (Wilcoxon's rank-sum test)					
p of difference	0.7	0.7	0.6	0.1	0.7
p IC/EC	0.1	0.9	0.6	0.4	0.2
Normal (n = 30)*					
Mean + 2 SDs	36.0	72.0	28.5	53.5	638.0
Mean − 2 SDs	28.0	52.5	21.5	44.5	420.0
No. of eyes out of normal range					
Operated on eyes (n = 30)	2	2	5	2	2
Nonoperated on fellow eyes (n = 30)	2	2	2	2	2

SD = standard deviation, SEM = standard error of the mean, difference = difference between data of operated on eyes and nonoperated on fellow eyes, p = error probability, p IC/EC = p for comparison of data between nonoperated on fellow eyes for the two surgical procedures (ICCE/ACL and ECCE/PCL).

* Normal range of normal fellow eyes.

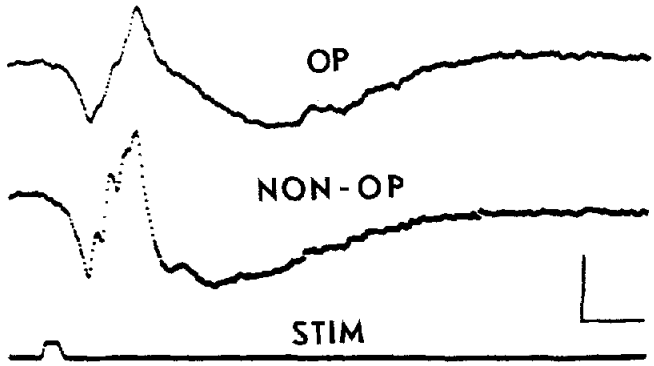

Fig. 3. Recording of the cone-mediated ERG components of a patient after ECCE and PCL implantation. OP = ERG of the operated on eye, NON-OP = ERG of the unoperated on phakic fellow eye, STIM = light stimulus. Calibration: 30 ms, 100 μV.

visualization of the operation field. Internal and semicoaxial illumination systems were constructed. Beam splitters in connection with observer units and/or documenting devices such as photographic, film and television sets attached to the microscope required unproportionally high light intensities. To simulate daylight conditions, the spectral distribution of the light sources was expanded to shorter wavelengths, including the blue-violet colors and even ultraviolet components of the spectrum. Brighter light sources such as halogen lamps replaced the tungsten bulbs. The high output levels of the illuminating systems can cause impairment of the neuroretinal structures of

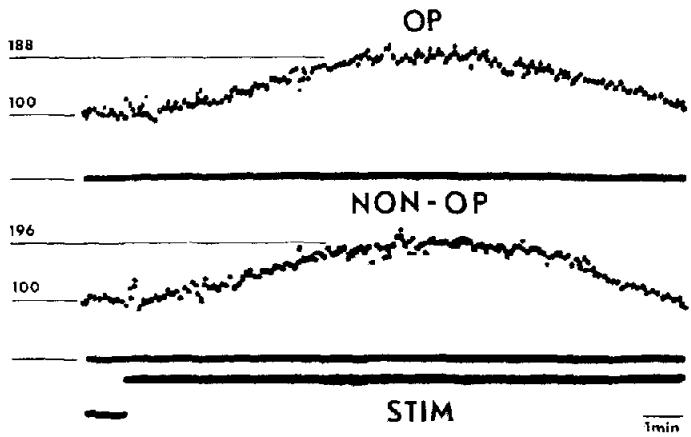

Fig. 4. Recording of an EOG of a patient after ECCE and PCL implantation. OP = EOG of the operated on eye, NON-OP = EOG of the unoperated on phakic fellow eye, STIM = light stimulus. Numbers at the left show the light peak amplitude of the EOG (OP, 188 %; NON-OP, 196 %) and the dark-adapted steady state (100 %).

the eye. As early as 1843, Himly [8] warned ophthalmologists to be careful when using high light intensities in ophthalmologic examinations. McDonald and Irvine [1] reported light-induced maculopathy caused by the operating microscope in extracapsular cataract extraction. Harada and coworkers [9] described three patients with 'light-induced retinopathy' when a video recording device was used in connection with an operating microscope. Robertson and Feldman [10] demonstrated a cause-and-effect relationship between the light of an operating microscope and macular lesions in human eyes. They observed a photic retinopathy in the eyes of two patients after 60 minutes of exposure to the light of an operating microscope.

In 1966, Noell et al. [2] used ERGs to study light-induced damage of the retina in rats. This was the first experimental study on electrophysiology of light damage of the eye. Albino rats were exposed to fluorescent light of different intensities and for different periods. The minimal damaging dose of light in rats was estimated to be about 1 $\mu W/cm^2$ for 1 hour. Skoog and Jarkman [11] exposed albino rats to strong visible light. They studied the photic damage of the retina and the pigment epithelium by recording the a- and b-waves and the c-wave. Their results indicated that the damage of the pigment epithelium was more profound than that of the neuroretina.

In our study, an amplitude increase of the ERG and EOG components could be proven half a year after moderate light exposures in ICCE (0.8–3.7 $\mu W/cm^2$). Obviously, these higher potentials are caused by the higher stimulus intensities passing the clear implant lenses. For the electrophysiologic tests, only patients with the same pupillary size in both eyes and with clear posterior capsules in the ECCE group participated in our study (visual activity; Table 1). Therefore, the reduction of the cone-mediated ERG components appears to be caused by the higher light exposure (3.4–7.3 mW/cm^2) during ECCE. The reduction of the amplitudes of cone-mediated a- and b-waves can be caused by a reduction of the number of cones as well as by a uniform impairment of all cones. The unchanged peak latencies indicate a reduction of the number of cones rather than a uniform impairment. The possibility of shunting or other changes in conduction in the layers of the retina and the pigment epithelium may be considered, thus reducing the electrical potentials without changing its waveform characteristics.

The rod-mediated ERG components were not reduced significantly.

The decrease of the EOG light peak in ECCE was highly significant. This result indicates a deterioration of the pigment epithelium. These findings are in accordance with the results of Skoog and Jarkman's studies [11].

Conclusion

The impairment of electrophysiologic potentials of retina and pigment epithelium appears to be caused by phototoxic effects of the light source of the operating microscope.

The following measures reduce photic damage of retinal structures during eye surgery: reduction of light intensities, filtering short and long wavelengths, including ultraviolet as well as infrared, oblique illumination of the operating field, paracentral noncoaxial light, eye shields, eclipsing the pupil with a 'retina light trap', avoiding beam splitters, photoflashes, television, film and photodocumentation whenever possible, and switching off the illumination whenever the operating microscope is not in use.

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