RETINAL DAMAGE FROM THE ILLUMINATION OF THE OPERATING MICROSCOPE: AN EXPERIMENTAL STUDY IN PSEUDOPHAKIC MONKEYS*

BY *A. Rodman Irvine,* MD, *Irmgard Wood* (BY INVITATION), AND (BY INVITATION) *Bruce W. Morris*

INTRODUCTION

AS RECENTLY AS 1974, MOST CATARACT SURGERY IN THE UNITED STATES WAS DONE with spectacle magnifying loops and without implantation of a plastic intraocular lens (pseudophakos).[1] This has changed dramatically in the past few years. Cataract surgery is now performed using the operating microscope and with the implantation of an artificial lens in the majority of cases. Because the operating microscope uses powerful coaxial illumination, the retina is exposed to potentially dangerous levels of light, as pointed out by Hochheimer and co-workers in 1979.[2,3] The use of a pseudophakos increases the risk of retinal damage from this illumination. The calculations of these workers indicated that the focusing power of the lens in a phakic patient significantly increases the retinal irradiance as compared to an aphakic patient. Other investigators have shown that the short wavelengths of light, especially the near UV wavelengths which are absorbed by the normal lens but not by a plastic pseudophakos, are most damaging to the retina.[4-7] The pseudophakic eye is thus at a greater risk of retinal damage from the illumination of the operating microscope than either the normal phakic eye, wherein the lens filters out the most toxic wavelengths of light, or the aphakic eye, wherein the light is not so well focused on the retina. In spite of these experimental and theoretical considerations, clinicians paid little head to these warnings until 1983, when clinically visible lesions produced by the coaxial illumination of the

*From the Department of Ophthalmology, University of California, San Francisco. Sponsored in part by NEI Core grant EY-02162 and That Man May See, Inc and Carl Zeiss, Inc.

operating microscope were first described in patients who had undergone cataract extraction with intraocular lens implantation.[8] The factors which determine why a small percentage of patients develop clinically apparent lesions, whereas the majority do not, have not been fully evaluated. Most worrisome is the possibility that the clinically visible lesion is just the "tip of the iceberg" and that subclinical damage may be present in a larger percentage of patients and over a larger area of the retina than is clinically apparent. One recent study compared the visual acuity 6 months postoperatively in patients undergoing cataract extraction with either of two operating microscopes at a single hospital.[9] One microscope produced three times the illumination of the other, and 6-month acuity results were significantly poorer in the patients who underwent surgery with the brighter microscope, even though no clinically visible retinal lesions were recognized. In addition, Tso and Woodford[10] have raised the possibility that senile macular degeneration may be caused or exacerbated by light exposure. This is an important consideration because many of the patients undergoing cataract extraction with pseudophakos implantation already have a degree of senile macular degeneration. There is now a compelling need for study of retinal damage from the operating microscope in eyes undergoing cataract extraction with pseudophakos implantation. We have undertaken such a study in rhesus monkeys.

MATERIALS AND METHODS

CATARACT SURGERY AND LIGHT EXPOSURE

The rhesus monkeys used in this study were housed in a standard diurnal light cycle. They were anesthetized with a 50:50 mixture of ketamine and xylazine and underwent lens extraction using the Kelman phacoemulsification system, under sterile conditions and with the same instrumentation used in human beings. Only the coaxial illumination of the operating microscope was used. During the surgery, the microscope was tilted maximally (30°), the eye was turned downward with a superior rectus suture, and the coaxial illumination was on the "low" setting. The cornea was covered with a piece of opaque gel-foam at all times that a red reflex was not necessary for the surgery (eg, during suturing but not during the capsulotomy, lens removal, and pseudophakos insertion). The cumulative time during which the cornea was not covered and the retina was thus exposed to the coaxial illumination was measured by the assistant using a stopwatch. The duration of this intraoperative light exposure has been a rather stable 10 to 14 minutes. A superior iridectomy and an inferior sphincterotomy were performed as part of the standard operation to

assure maximal pupillary dilation postoperatively for fundus examination and photography. At the end of the operative procedure, the microscope was returned to an upright, perpendicular position, the eye fixed in a straight ahead position, the coaxial illumination turned to the "high" setting, and the desired additional light exposure was given. It is this additional exposure given immediately following the standard surgical procedure which was varied. By minimizing light exposure during the standard surgical procedure and tilting the eye and microscope so as to deliver this exposure primarily to the inferior retina, we have tried to separate as much as possible the "baseline" exposure from that given immediately at the end of surgery. It did not prove feasible in preliminary experiments to separate the two exposures in time, since at any later time the pupil could not be reliably kept at full dilation and the media were not uniformly clear. This was due to the formation of inflammatory synechiae between the iris and the lens, the deposition of inflammatory precipitates on the pseudophakos, and variable posterior capsular opacification. Although the use of this "additional" light exposure as the variable added to the baseline minimal intraoperative exposure is scientifically displeasing, it seems closest to the clinical situation, where the surgeon is concerned with intraoperative events that prolong the microscopic exposure above the standard minimum. The "additional" light exposure, which is varied during the experiment is aimed so as to produce a clinical lesion in the superior portion of the macula of the rhesus eye. Although it has not proven possible to hit the exact area of the retina desired with the image of the illuminating filament, it has proven possible to consistently place the focal lesion from that image within an area 2° to 6° from the foveola. The variable sensitivity of different parts of the retina to phototoxicity as described by others[5] has thus not seemed to affect significantly the comparison of lesions in different eyes in our study.

Steroid and antibiotic solutions were injected subconjunctivally in each eye at the end of the procedure (2 mg betamethasone and 20 mg gentamicin) and antibiotic and atropine ointment was placed in each eye.

Eleven monkeys were used in this study. One eye from each of two monkeys underwent the following exposures to the coaxial illumination: 30 minutes, 15 minutes, 7.5 minutes, and 4 minutes, respectively. One eye from each of two monkeys served as unoperated controls for the histologic studies. One eye underwent an 8-minute exposure, and one monkey underwent a 4-minute exposure to one eye while the fellow eye underwent a discontinuous exposure consisting of 4 minutes with the light on, followed by 5 minutes with the light off and then another 4 minutes with the light on.

A Zeiss OpMi-6 operating microscope was used in this study. It was equipped with a 30-watt bulb. As measured with a United Detector model 40-x radiometer, our microscope produced 27.7 mW/cm^2 illumination measured at the focal plane 175 mm from the objective lens at the "high" setting and 11.7 mW/cm ± 2 at the "low" setting. This was within the range of illuminations found in several other microscopes which we measured in our operating rooms. Using the calculations of Calkins and Hochheimer[2] at the "high" setting, the "hot spot" formed by the image of the illuminating filament in the microscope would produce a retinal irradiance of 0.49 W/cm^2 in human beings or approximately 0.66 W/cm^2 in the rhesus monkey. The emission spectrum of the Zeiss operating microscope has been reported previously.[11,12]

Keratometry and ultrasonographic axial length measurements performed on the first four rhesus eyes indicated that a 22 diopter posterior chamber intraocular lens produced emmetropia or minimal myopia. This lens was then used for implantation in all of the monkeys.

POSTOPERATIVE CLINICAL EVALUATION

Fundus photographs were taken 48 hours postoperatively. The visible retinal lesion corresponding to the image of the illuminating filament was most readily identifiable 48 to 72 hours postoperatively, as described by Lawwill.[5] It produced a sharply defined oval patch of retinal edema. Because postoperative inflammation with fibrin in the anterior chamber increases during the first few days, photography is easier at 48 hours than at 72 hours. Photography (or occasionally indirect ophthalmoscopy with a drawing of the fundus) at this stage made it easier to find the focal lesion later, both clinically and histologically. By 8 weeks the postoperative inflammation had subsided and photography, including fluorescein angiography was again possible. The latter was most helpful in identifying the lesion at this late stage since the only clinically visible changes were mild changes in the pigment epithelium.

HISTOLOGY

Eyes were enucleated while the animals were under anesthesia with a 50:50 mixture of intramuscular ketamine and xylazine, immediately prior to sacrifice. A 2 to 3 mm incision was made through the sclera into the vitreous near the muscle insertions, and the eyes were placed in a fixative. The primary fixative consisted of 1% paraformaldehyde, 2% glutaraldehyde, with final concentration of 0.068 M sodium cacodylate buffer, and the addition of 0.2% calcium chloride, pH 7.4. The eyes were left in

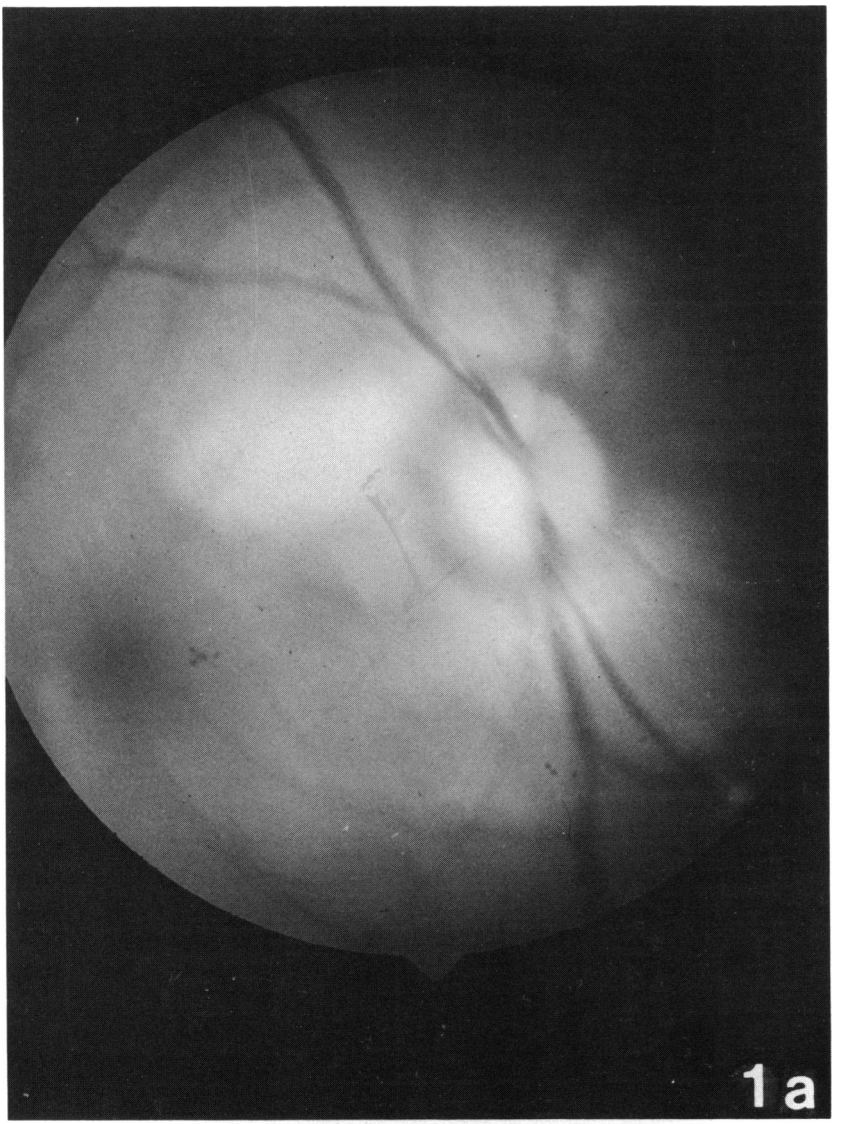
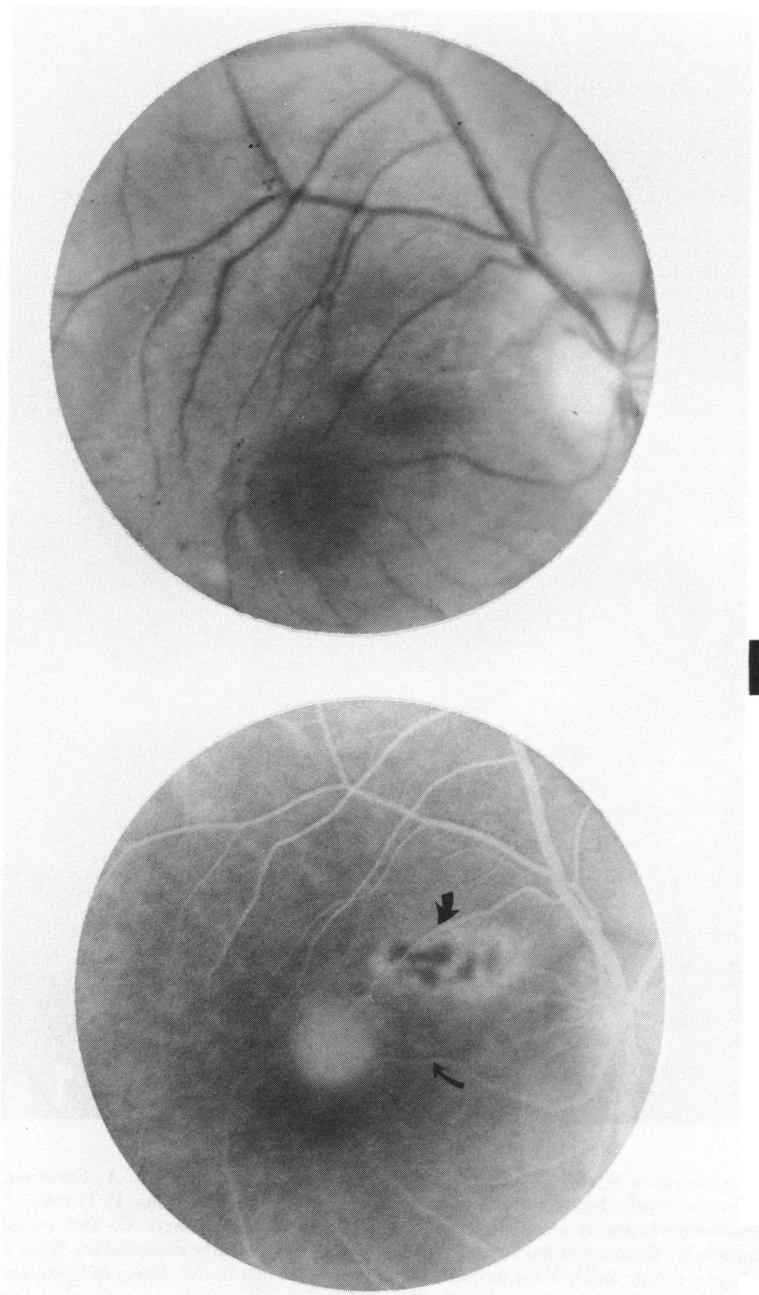


FIGURE 1

Clinical appearance of retinal lesions. Rhesus 5192, 8-minute exposure. A: Forty-eight hours postoperatively, lesion appears as an oval, focal area of retinal edema. B: Two months postoperatively, lesion is seen as an area of mottled pigment change. C: Two months postoperatively, fluorescein angiography makes lesion more clearly discernable. *Wide arrow* indicates venule and *narrow arrow* indicates arteriole that is also shown in histological examination in Fig 2.



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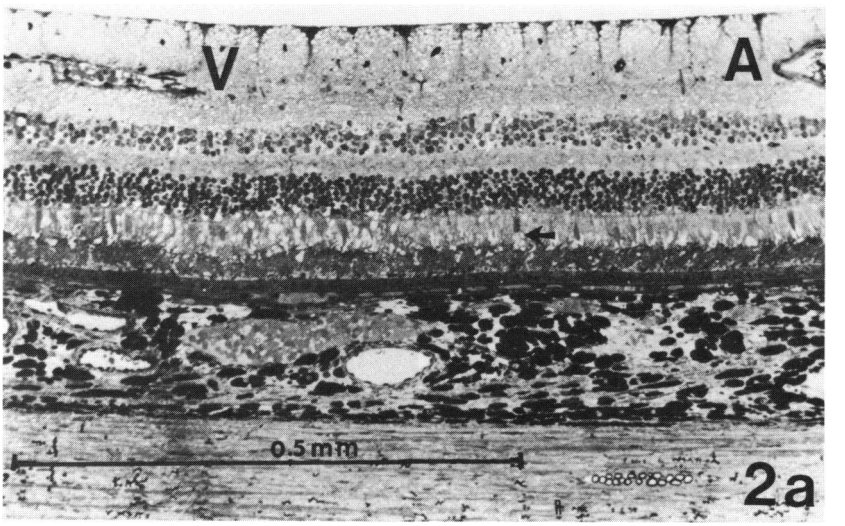
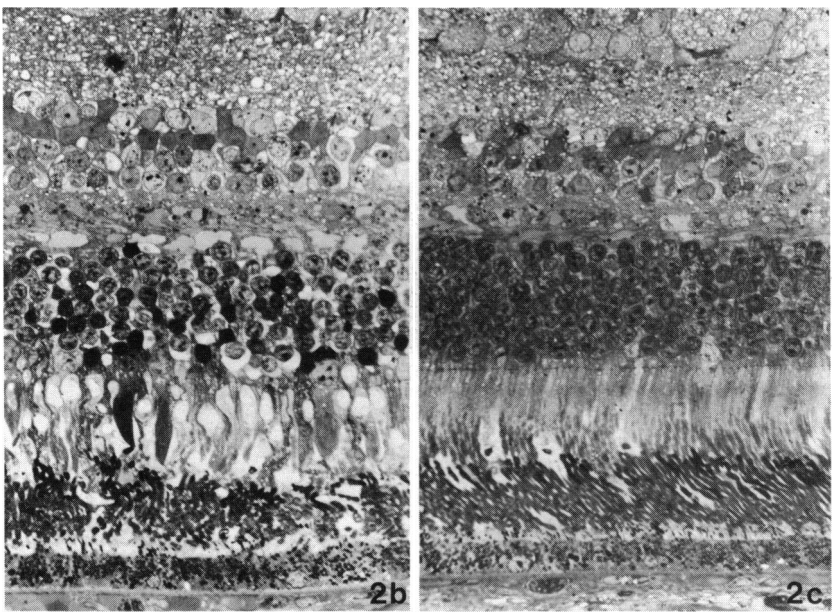


FIGURE 2

Focal nature of this lesion, as seen on different portions of a single histologic slide, plus its correlation with clinically visible lesion demonstrates that changes are not fixation artifact. Rhesus 5192, 8-minute exposure (lesion is that seen by fundus photography in Fig 1 and sectioned vertically; "*A*" indicates arteriole and "*V*" venule shown in Fig 1). A: Low-power micrograph shows arteriole and venule on either side of lesion. Lesion, wherein there are many cytoplasmic vacuoles and pycnotic nuclei, begins at venule and extends toward arteriole. It measures approximately 400 μ (× 166). B: Higher power view through lesion shows vacuolation and damage in all cell layers (× 793). C: High-power from edge of same slide but near arteriole and away from lesion shows a much more normal appearance (× 793). D: Electron microscopy of pigment epithelium and photoreceptor outer segments in center of lesion corresponding with (B) (× 12,210). E: Electron microscopy of pigment epithelium and photoreceptor outer segments away from lesion corresponding with (C) (× 15,355). F: Electron microscopy of photoreceptor inner segments in center of lesion corresponding with (B). Note rods seem more damaged than adjacent cone (× 6105). G: Electron microscope of photoreceptor inner segments away from lesion corresponding with (C) (× 8481).

fixative for 1 hour and then dissected. Eyes were dissected under a Zeiss OpMi-1 operating microscope. The lesion was excised in a tissue piece approximately 4 × 4 mm using fundus photographs and drawings as a guide. In some specimens the lesion was clearly visible under the dissecting microscope. Areas at varying distance from the lesion were excised in a similar manner for comparison. After dissection, the tissue pieces were placed in fresh fixiative, each in their individual labeled specimen vial and fixed for an additional 2 hours. Following with a rinse in 0.068 M sodium cacodylate buffer, pH 7.4, the tissues were postfixed with 1% osmium



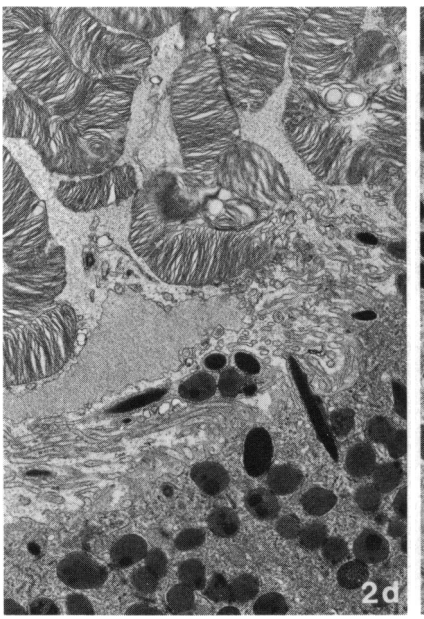
tetroxide in sodium veronal acetate buffer for 2 hours, rinsed in sodium veronal acetate buffer, pH 7.4, and en'block stained with 0.5% uranyl acetate for 1 hour. Following dehydration in graded acetone 50% to 100% and propylene oxide, the specimens were embedded in Araldite 502 and polymerized. One-micron sections of the selected areas were stained with basic fuchsin and methylene blue for light microscopy and photographed, using the Zeiss photomicroscope. Ultrathin sections for electron microscopy of the corresponding areas were primarily stained with 2% uranyl acetate, followed by lead citrate, examined and photographed with a JEOL 100C transmission electron microscope.

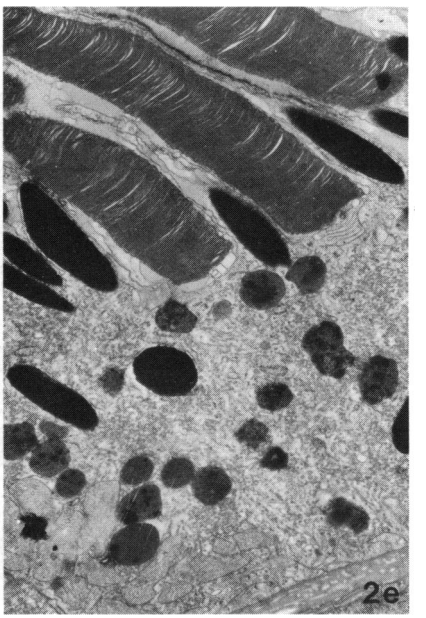
Light microscopic slides were graded according to the schema devised by Currier and co-workers[13] for semiquantitative analysis of retinal damage from visible light.

RESULTS

CLINICAL

Clinically apparent lesions initially appeared as sharply circumscribed oval areas of retinal edema approximately 0.5 × 1.5 mm in size. Lesions were not apparent immediately after surgery but were visible at 24 hours

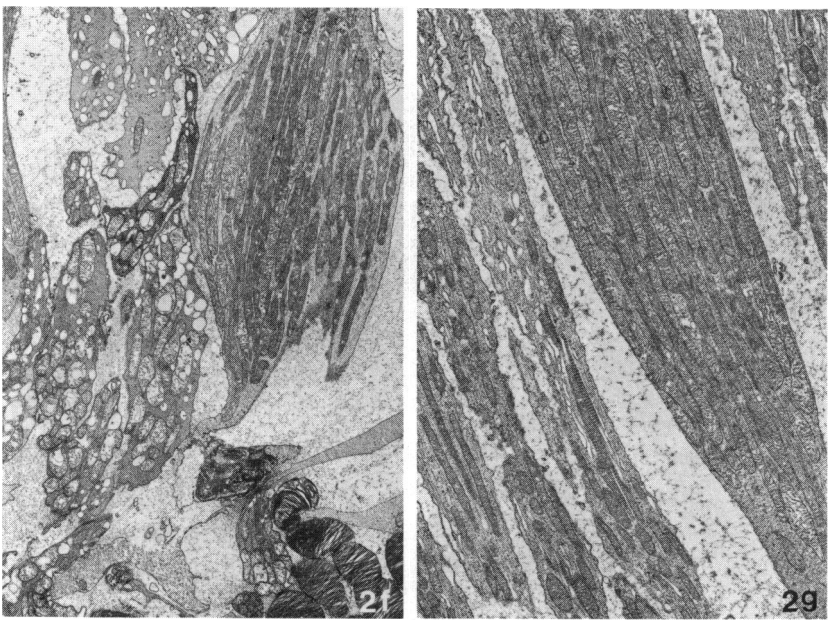




and increased to reach peak intensity at about 72 hours. They then faded and were eventually replaced by an area of mottling of the pigment epithelium which was most easily visualized with fluorescein angiography (Fig 1). Two to 3 months postoperatively, angiography of these lesions showed only mottled transmission of fluorescence with no dye leakage. The lesions thus behaved identically to those produced by Hochheimer and co-workers[3] with the operating microscope and by Lawwill[5] and his well-controlled experimental system using monochromatic light.

No difference was recognized on ophthalmoscopy or fundus photography between the lesions produced at 30-minute, 15-minute, or 7.5-minute exposures, respectively, except for a possible slight decrease in size in the lesions with the shortest exposure times. At 4 minutes' exposure, no clinically visible lesion was recognized. One eye in each of two monkeys was subjected to each of the above exposure times (eight eyes in eight monkeys). All six of the eyes exposed for 7.5 minutes or longer developed clinically visible lesions, whereas neither of the two eyes exposed for 4 minutes did so. The threshold for production of a clinically visible lesion, therefore, lay between 4 and 7.5 minutes.

In one monkey the right eye was given a 4-minute exposure and the left eye was given an exposure of 4 minutes followed by 5 minutes with the

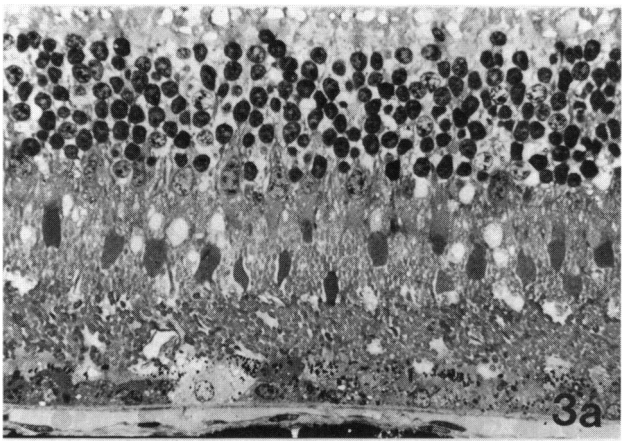


light off and then another 4 minutes' exposure to the same area. No lesion was visible in the right eye, whereas the left eye developed a lesion similar to that produced by 8 minutes of continuous exposure. Thus, it appears that, at least within this relatively short duration, sequential exposures to the same area have an additive effect. Similar findings have been described in other models of light toxicity.[14]

MICROSCOPIC

Although on clinical grounds it was difficult to discern gradations of severity between the 7.5-minute and the 30-minute lesions, histologically, such a differentiation was possible, especially when the grading system of Currier and co-workers[13] was used. There was variability within a given lesion, but by grading the most severe area of each specimen, one could establish some correlation between the severity of histologic damage and the duration of light exposure. Differentiation between mild and severe lesions was most evident in the photoreceptor and pigment epithelial cells, whereas the changes in the inner retinal layers were more similar in the mild and severe lesions, varying in degree rather than in kind.

One difficulty in evaluating retinal lesions produced by light has been the fact that changes similar to those in mild light toxicity lesions can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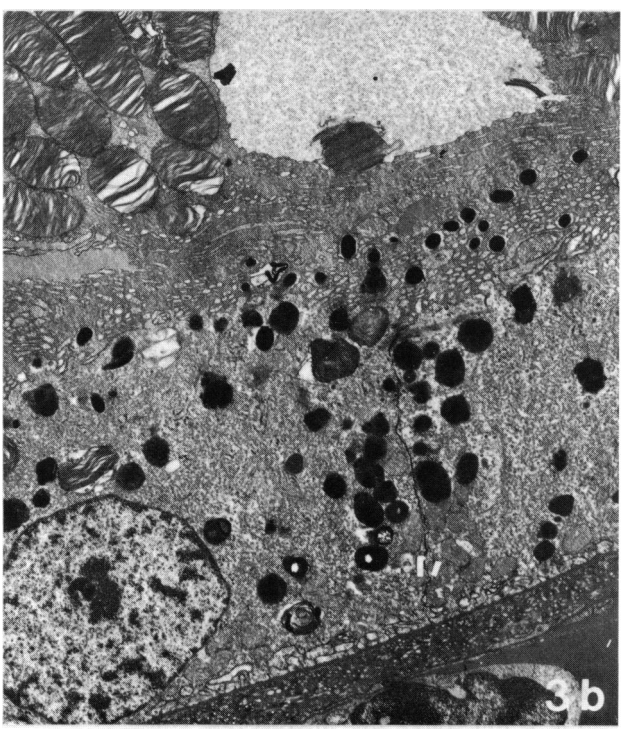
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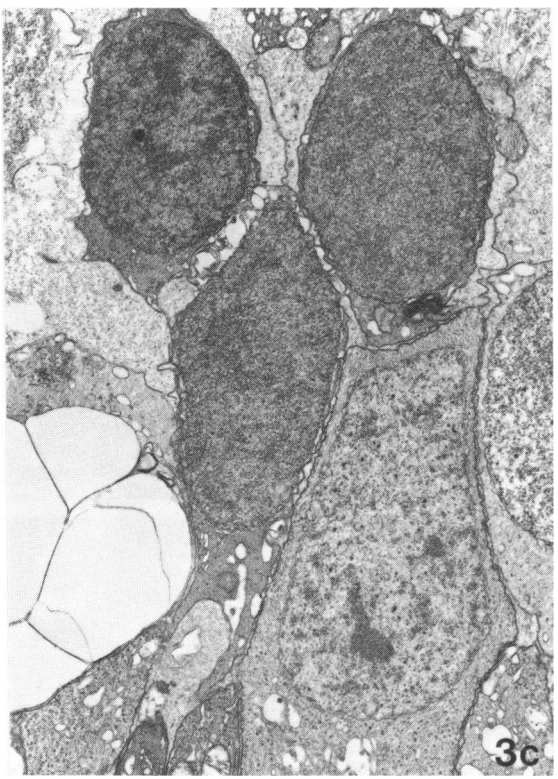
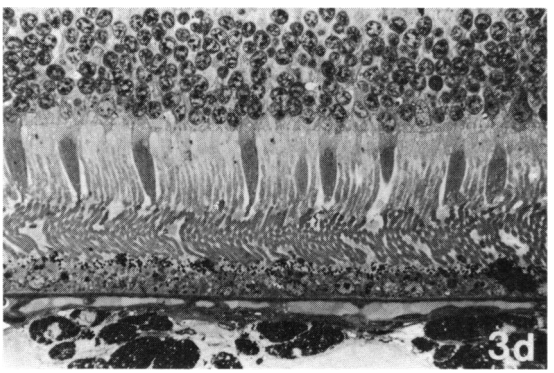


FIGURE 3

Typical "mild" lesion. Rhesus 17492, 7.5-minute exposure, 8 weeks postoperatively. A: Light microscopy of outer retina shows irregular concentration of basophilic organelles in pigment epithelium and one pigment epithelial cell with extraction of its cytoplasm that appears dead, mild derangement of photoreceptor outer segments, vacuolation in inner segments, a few pycnotic nuclei and some "halos" around nuclei in outer nuclear layer (× 544). B: Electron microscopy at lesion shows marked increases in phagosomes and residual bodies in pigment epithelium and marked increase in density of Bruch's membrane (× 6270). C: Electron microscopy at lesion shows vacuolation in outer nuclear layer (× 8481). D: Another section from same eye but approximately 5 mm nasal to lesion shows relatively normal photoreceptors and outer nuclear layer (× 544).

occur as artifacts. Our control eyes did show scattered areas with such changes. The relatively small and focal nature of the lesions in our study, however, allowed us to correlate the clinical and histologic lesions very precisely. Fig 2 illustrates the microscopic study of a vertical section through the center of the lesion seen clinically in Fig 1. It shows the focal nature of the changes and the correlation of these changes with the



location of clinical lesion. This sort of correlation provided convincing evidence that the changes seen in the "mild" lesions were indeed due to light toxicity rather than artifact.

Mild Lesions (Figs 2 and 3). The mildest change in the pigment epithelium was a marked increase in the number of membrane bounded organelles containing osmiophilic, membranous material, which we presume had been ingested from the interphotoreceptor space. These organelles resemble secondary lysosomes and residual bodies. In some lesions, Bruch's membrane seemed filled with basophilic material (Fig 4). As suggested by Tso and Woodford,[10] these changes of light damage are similar to those seen in senile macular degeneration in human beings. The photoreceptors in the mildest lesions showed some disorientation and fragmentation of the outer segments but were not grossly deranged. The inner segments showed cytoplasmic vacuolation. One major difference between our findings and those of Lawwill[5] was that in our system the rods were more severely damaged than the cones, whereas he found the reverse to be true. We found more severe swelling and vacuolation of the rod inner segments than of the cones, and the outer nuclear layer and outer plexiform layer similarly showed more vacuolation in the nuclei and spherules of the rods than in the nuclei and pedicles of the cones. This difference between our findings and those of Lawwill[5] may be due to the fact that we were using the full range of white light emitted by the operating microscope, whereas he was using monochromatic light. Another possible explanation for this difference is the fact that we used higher intensities and shorter exposures than Lawwil used. Some damage to the cells of the inner nuclear layer and ganglion cell layer was seen even in our mild lesions. Changes in the inner retinal layers were seen in

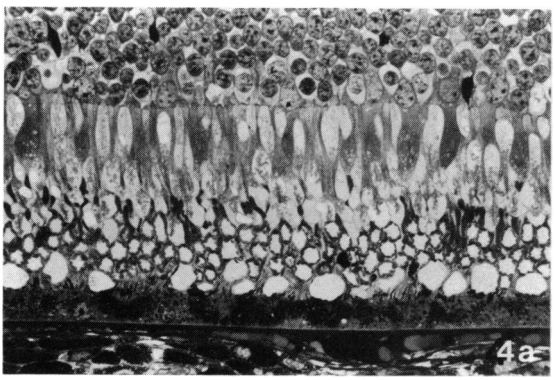


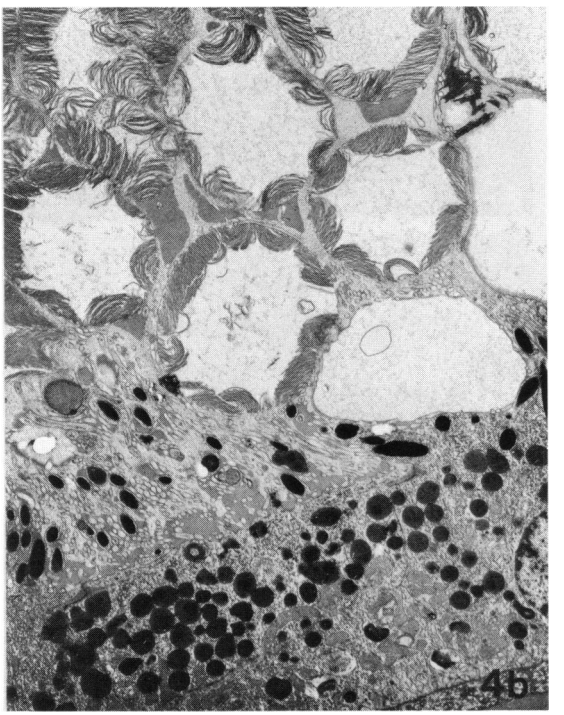
FIGURE 4
Intermediate lesion. Rhesus 7376, 15-minute exposure, 8 weeks postoperatively. A: Pigment epithelium shows irregular accumulation of densely basophilic organelles. Photoreceptor outer segments are markedly deranged, forming "donut" shapes. Inner segments show cytoplasmic vacuolation more marked in rods than in cone cells with pycnotic nuclei. Pycnotic nuclei and cytoplasmic vacuolation are present in outer nuclear layer (× 544). B: Electron microscopy shows pigment epithelium filled with residual bodies and phagosomes. There is marked derangement of photoreceptor outer segments (× 6105). C: Rod spherules in outer plexiform layer show more vacuolation than cone pedicles. Some nuclear "halo" formation is seen in inner nuclear layer (× 544).

light microscopy as cytoplasmic vacuolation, "halo" formation around the nuclei and nuclear chromatin clumping or pycnosis (Fig 2).

A lesion that is somewhat intermediate between a "mild" and a "severe" lesion is seen in Fig 4. Photoreceptor outer segment derangement is clearly greater than in the mild lesions.

Severe Lesion (Fig 5). In the more severe lesions the pigment epithelium lost its normal cuboidal shape and spherical pigment epithelial cells resembling macrophages were piled up in some areas, whereas other areas had just thin cytoplasmic extensions of pigment epithelial cells covering Bruch's membrane. Outer segments were absent in some parts of the most severe lesions and were markedly disoriented and fragmented in others. The remaining retinal layers showed cytoplasmic vacuolation as described in the milder lesions but of a greater degree. Pycnotic cell nuclei were seen with greater frequency in the severe lesions.

In one specimen which had undergone a 15-minute exposure and was examined 11 weeks postoperatively, a marked thinning of the retina dramatically demonstrated the fact that cell loss that had taken place in all layers (Fig 6).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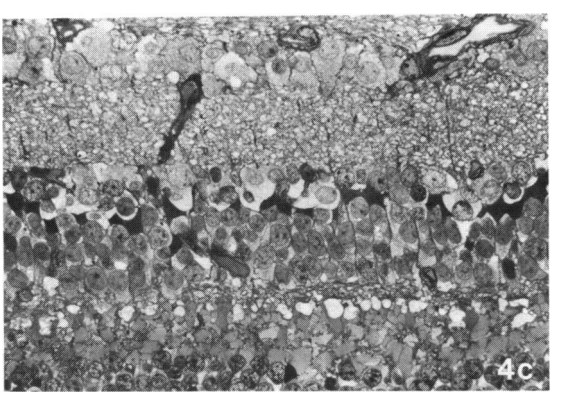
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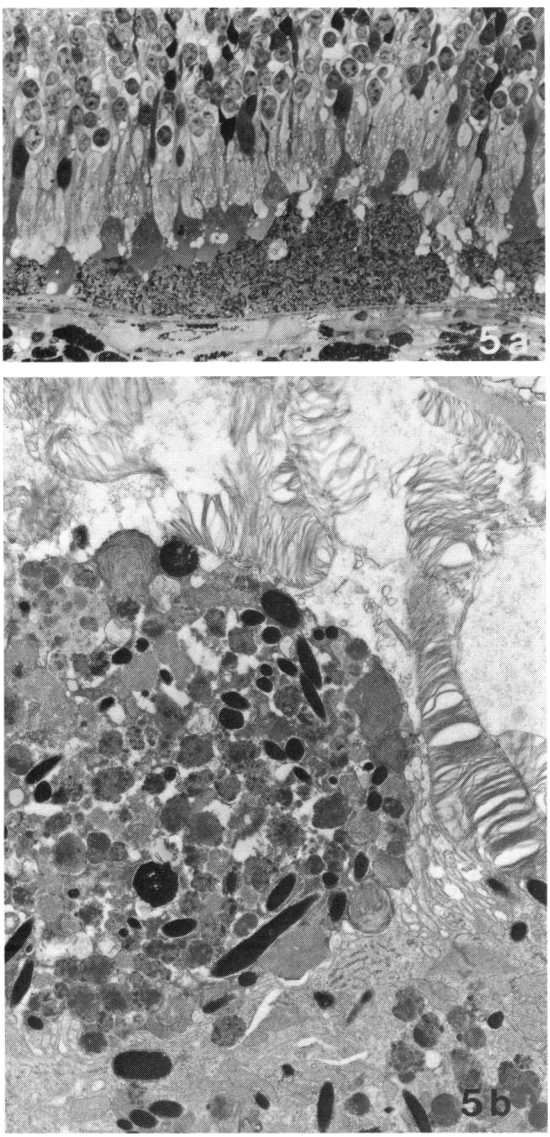
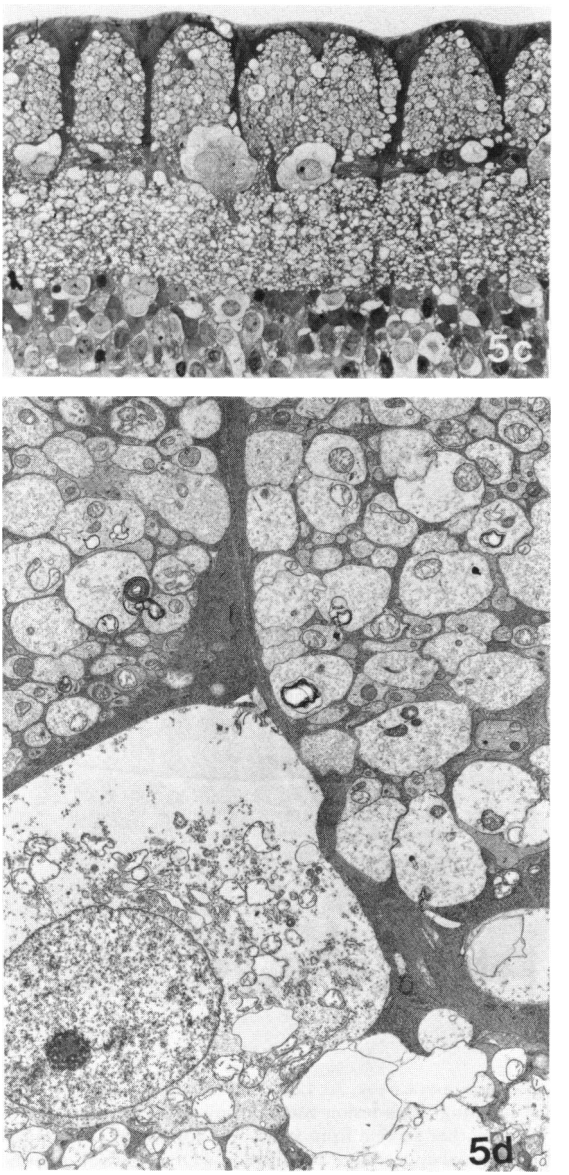


FIGURE 5

"Severe" lesion. Rhesus 16859, 30-minute exposure, 8 weeks postoperatively. A: Rounded pigment epithelial cells can be observed piled up in several layers. Photoreceptor outer segments are completely gone and many more pycnotic nuclei are seen in outer nuclear layer (× 544). B: Electron microscopy shows such a rounded cell full of ingested phagosomes, pigment, and debris from interphotoreceptor space (× 9250). C: Inner retinal layers show more severe vacuolation than in "mild" lesion, including severe ganglion cell damage (× 544). D: Electron microscopy shows severe mitochondrial disruption with cytoplasmic extraction in a ganglion cell (× 6105).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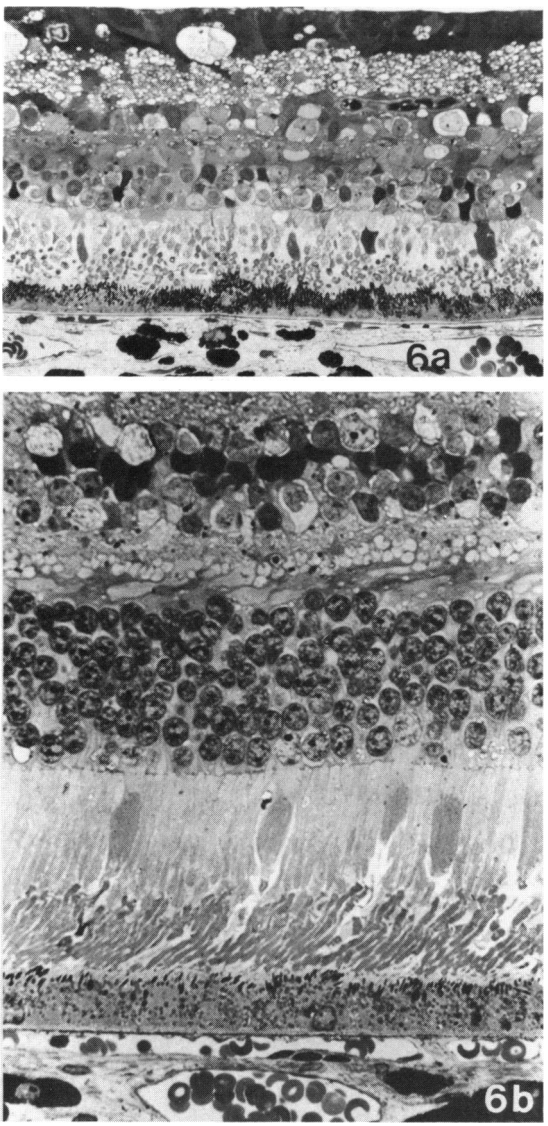
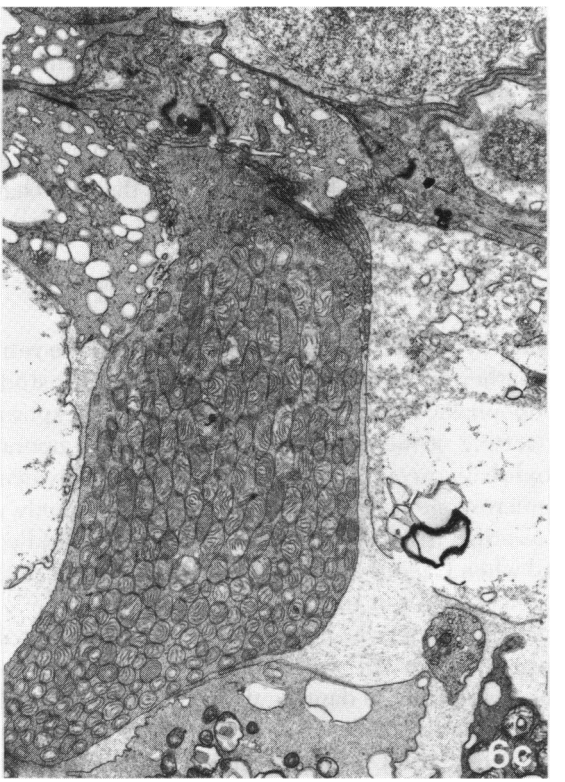


Cell
exposure, 11 weeks postoperatively. A: Light microscopy of lesion shows marked cell loss producing retinal thinning. This lesion was midway between superior portion of disc and foveola (× 544). B: Another section from same eye, approximately 3 mm inferior to lesion, shows more normal retinal appearance and thickness (× 793). C: Electron microscopy from lesion shows a normal appearing cone inner segment full of apparently normal mitochondria adjacent to rod inner segments which show near total loss of their mitochondria and severe cytoplasmic vacuolation (× 9250).



Electron microscopy confirmed the light microscopic finding that the photic damage affected intracellular organelles at all levels of the retina, from the pigment epithelium to the nerve fiber layer (Figs 2 to 6). Axons in the plexiform layers and nerve fiber layers showed swelling and loss of intracellular organelles. Mitochondrial damage was quite striking in all cell layers. Lawwill[5] hypothesized that the shorter wavelengths of light produced damage by interacting with light sensitive molecules in the mitochondria, such as cytochromes, and that if a critical number of mitochondria in a cell were damaged, it might be unable to recover and thus be irreversibly damaged, whereas a neighboring cell with less than the critical number of mitochondria damaged could eventually recover completely. This theory seemed compatible with the findings in our monkeys, which were sacrificed 2 to 3 months following the photic injury. We did sometimes find a normal cell adjacent to severely damaged cells. In addition, the electron microscopy supported the concept that in our

model the rods were more susceptible to damage than the cones. In some instances, cone inner segments seemed quite normal, while adjacent rod inner segments showed severe mitochondrial damage (Figs 2f and 6c). Rod nuclei and spherules were also more severely damaged than cone nuclei and pedicles. This selectively of the cellular damage on the electron microscopic level provides further evidence that the changes attributed to light damage are not fixation artifact.

CONCLUSIONS

The pseudophakic eye is theoretically more susceptible to retinal damage from light than either the phakic or the aphakic eye. Our study indicated that in the pseudophakic rhesus monkey, the threshold exposure with the high intensity setting of the coaxial illumination of the operating microscope for an ophthalmoscopically visible lesion was between 4 and 7.5 minutes. The microscope used in this study was a commonly used model and produced a retinal irradiance somewhere near the middle of the wide spectrum found when Calkins and Hochheimer[2] surveyed all the ophthalmic operating microscopes at the Wilmer Institute.

The histologic changes in our light-exposed monkey eyes were similar to those described by Lawwill[5] with the exception that in our study the rods were more susceptible to damage than the cones. Lawwill[5] believed that in primates with relatively intense and short light exposures, the retinal changes were caused mainly by the mechanism he labeled "blue light damage." Ham and co-workers[15] first described the marked increase in sensitivity of the retina to photic damage from the shorter, blue wavelengths. Both Ham et al[15] and Lawwill[5] believed that this damage was not dependent on either rhodopsin or the specific cone pigments but rather upon light sensitive molecules present in all layers of the retina. Lawwill[5] suggested that mitochondrial cytochromes might be affected. This "blue light" mechanism of damage was felt to be superimposed upon a lesser degree of damage from two mechanisms dependent upon the direct effect of light on the rod and cone pigments, respectively.[5]

In some eyes, the edge of the lesion appeared relatively sharp, as illustrated in Fig 2. In others, it tapered off more gradually and histologic damaged seemed to extend outside the area of obvious clinical damage. Because of the possibility of artifact simulating "mild" lesions, more extensive sectioning and study of our specimens is needed before we can state with assurance whether histologic damage is present in those eyes which received only 4 minutes' exposure and in areas far away from the clinical lesion in those eyes receiving 7.5- and 15-minute exposures.

One could argue that the fundus photography and fluorescein angiography performed upon our monkeys prior to sacrifice might produce light toxicity that would compromise study of the light damage from the operating microscope. These photographic studies were necessary, however, to allow the sort of correlation between the clinical lesion and the microscopic findings that is illustrated in Fig 2. This correlation seems the strongest evidence that the changes described in the "mild" lesion are neither randomly distributed artifact nor damage induced by the photography. The changes in the outer retina of the more severe lesions were distinctive and never simulated by artifact in the controls. Similarly, the full-thickness retinal thinning seen in the lesion illustrated in Fig 6 is a change which could not be produced by fixation artifiact and substantiates Lawwill's claim[5] that the "blue-light" mechanism damages all layers of the retina.

These findings should force every ophthalmic surgeon to reassess his surgical technique so as to minimize the potential for light-induced retinal damage. The microscope illumination should be at the lowest level consistent with adequate visualization, and the cornea should probably be covered during those parts of the surgical procedure where this is possible. Because the shortest wavelengths of light are the most retinotoxic, filters to eliminate those wavelengths, such as the Zeiss UV 430, would seem logical.[16] Finally, some standardization of the illumination level of operating microscopes is needed.

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DISCUSSION

DR THEODORE LAWWILL. Doctor Irvine is to be congratulated for observing, and being the first to publish the short wavelength retinal damage lesion in humans caused by the operating microscope. I should also like to pay respects to the man responsible for our interest in the subject of retinal light damage, Werner Noell. His remarkable discovery in the rat started us all thinking about this potential problem. Of course, it is now known, that the lesion in the rat is different from that in the primate.

In 1977, my group published the figures on light damage thresholds which would predict a retinal lesion in 8 to 12 minutes of illumination by slit lamp and Hruby lens focused on the retina. These figures were appropriate for a standard Tungsten bulb and not the new Tungsten-Halogen bulbs which have a much greater output at the blue end of the spectrum. The lesions we found were histologically similar to those found by Doctor Irvine. In the fundus they spared the fovea, even when severe. Doctor Irvine, in some of his other work has found a similar sparing of the fovea in the human.

Three things might be stated about the short wavelength effect: (1) the threshold is very sharp for time and intensity; (2) brightness is not directly related to the damage because of wavelength dependence; and (3) that this is a true photodynamic effect and is dependent upon the concentration of oxygen.

It is possible that visual acuity is not the most appropriate measure for detecting or evaluating damage from the operating microscope. There are probably significant lesions outside the fovea more often than in, and the fovea itself is somewhat

spared. If time intensity reciprocity continues for exposures shorter than 8 minutes, which we believe it does, it would mean that cutting the light in half would give one twice the operating time before causing a retinal lesion. The new Tungsten-Halogen bulbs have a very high output at the blue end of the spectrum making them much more dangerous for the eye than the old Tungsten bulbs. If one were to filter out the blue light, one would like to get rid of the wavelengths shorter than 500 to 550 nm. This is not well accepted by the surgeons because it causes a yellow cast to the light.

The difference in sensitivity between the rods and cones, which is different between Doctor Irvine's work and mine, probably relates both to the much shorter time period and the wavelength of light he is using. Our findings showing greater sensitivity of the cones was for 1- to 4-hour exposures with green light. The high blue content of the Tungsten-Halogen bulb used by Doctor Irvine would favor the blue light type damage, which is probably not in itself specific for cones or rods.

Again, I should like to congratulate Doctor Irvine on a very excellent scientific presentation and for bringing to the attention of clinicians, an important safety matter.

DR JOHN BULLOCK. I've been concerned about this problem for several years and because of this, and intraocular pressure problems, I do not routinely use Healon. I put a large air bubble in the eye; then I slip the implant in and manipulate it into position. I then leave the air in when I'm closing the eye; I remove the air after the placement of the last corneoscleral suture. I think that this air helps to diffuse the light away from the macula.

DR JOSEPH DIXON. I have a question. Can you calculate or estimate the protection you would get from an intraocular lens which has an ultraviolet filtration factor?

DR ARTHUR JAMPOLSKY. I think the effects of light on the visual system is one of the most fascinating aspects of ophthalmology today. I just want to mention that in assessing these effects, the worst test that one can use is ordinary visual acuity. Half the macular cells can be dead, and one may still have 20/20 vision. The next worst test is angiography. The peak angiographic effect is in about 3 to 4 months, and thus, angiographic manifested leakage is a late sign, long after the "garage door" is closed. The next best test (going upward from the worst) is what has been presented today—histological changes.

The best noninvasive tests are functional tests, and how long it takes clinical ophthalmologists to fully utilize laboratory information. The lag is often long. Low contrast visual acuity is an excellent functional test. Quantified glare-recovery is also a superb test. Blue macular sensitivity, with a yellow surround, is a very sensitive test, one of the earliest for detecting macular change. I wonder how long it will take for all clinical ophthalmologists to strongly advise that their susceptible patients wear ultraviolet absorptive lenses, especially if they are on any of the

many drugs that are retinal-sensitizing, especially to ultraviolet. There is a long list of such common drugs. How long will it take to require that we use filters on our indirect scopes, and on our operating microscopes? Especially since no harm is done by implementing these steps now, and thus incorporating well-established laboratory studies into clinical management that may save vision.

I congratulate the author on his meticulous investigation to highlight the importance of this problem in our everyday clinical management.

DR MAX FORBES. I would like to compliment Doctor Irvine for this outstanding study. I wonder if he could estimate how much benefit would be derived from turning off the coaxial light as soon as the intraocular lens is implanted.

DR ALEXANDER R. IRVINE. I would especially like to thank Doctor Lawwill, who really did all the basic work in this field. We were happy to find that our results were so similar to what he had shown with monochromatic light. Doctor Dixon asked the interesting question about whether the new pigmented intraocular lenses are effective in preventing photic damage from the operating microscope. We had several monkeys in which we put a standard intraocular lens in the right eye and an intraocular lens that was pigmented in the left eye. Unfortunately, the pigment did not seem to have any significant effect in preventing phototoxic damage from the operating microscope. I believe this is because the pigmented intraocular lens we used was designed to filter out only wavelengths less than 400 nm. The tungsten bulb of the operating microscope puts out an emission spectrum that begins at about 400 nm. Thus, a pigmented intraocular lens that might be very helpful for an aphakic patient in preventing retinal damage from the sun at Palm Beach is not helpful under the operating microscope, because the operating microscope has a different emission spectrum. I think that the damage we are seeing from the operating microscope is primarily what Doctor Lawwill calls "blue light" damage and is due to light from about 400 to 500 nm, so unfortunately, and to our surprise, the pigmented intraocular lenses do not protect against this sort of damage. In contrast, preliminary studies indicated that the Zeiss UV 430 filter may offer quite significant protection.

The question of functional testing that Doctor Jampolsky brought up is very critical, and that's where we are really just at a beginning. Now I will say, that to my surprise when we started looking at the histology in the eyes with the heavy exposure, there was some damage outside the area of the apparent clinical lesion. Some of this may have been artifact, but it made us think that it might be worthwhile to bring back the initial six patients in whom we first recognized this lesion clinically and do electroretinogram studies on them. Initially, we had thought that would be a silly study to do because it appeared to be such a focal lesion that it wasn't expected to change the electroretinogram. To our great surprise, when we brought these patients back, Doctor Stone found that if he compared the involved eye with the fellow eye, there was a definite depression of the electroretinogram in the eye with the photic lesion. Two of these patients who

had developed clinically apparent photic macular lesions after their initial cataract extraction and pseudophakos implantation later underwent cataract extraction and pseudophakos implantation in the fellow eye, but in the second eye the surgeon was aware of what had happened in the first eye and, therefore, took special care to decrease light exposure. In those two cases, the second eye had a better electroretinogram than the initial eye. That's the only functional testing that we've done to date, and I must admit I'm surprised at the findings. Doctor Lawwill said he had some concepts, and maybe he would mention them, as to how a relatively focal lesion could cause such a change in the electroretinogram. Perhaps it was because we had moved the eye around so much during surgery that we really produced a good deal of subclinical damage in addition to the one small clinically evident lesion that was recognized.

Finally, Doctor Forbes asked what I think the value would be of turning off the coaxial light and using the side lights as soon as the red reflex is no longer needed. I think that's of real value. I was shocked by the demonstration Dave Copenhagen did for me, however, and which you saw in my initial slides. There you saw that the side lights also produce nice, focal, illuminated images on the retina. They are not quite as intense as the coaxial light, but they are potentially dangerous. You tend to think, since they're coming from the sides, they are not going to strike the posterior pole of the retina. That is true in microscopes where these illuminators are on the right and left sides. However, if you have one of those microscopes where your side illumination has been "improved" by bringing it down below toward the patient's feet and thus out of the way of your hands, then you've got to be careful. That's the situation wherein tilting the eye inferiorly can throw those "side" lights right smack onto the macula.