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RETINAL DAMAGE SECONDARY TO CHRONIC LIGHT EXPOSURE, Thresholds and Mechanisms*

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ABSTRACT

The effect upon the retina of exposure to large fields of bright visible light has been evaluated. The thresholds for permanent retinal damage for four hour exposures in rhesus monkeys have been established for white light, and laser lines of 514.5 nm, 488 nm, 457.9 nm, and 590 nm. The damage has been evaluated by ophthalmoscopy, electroretinography and light and electron microscopy. The shortest wavelength light (457.9 nm) is more effective in causing damage, particularly histological damage, which is spread throughout the fundus and throughout the retinal layers. Functional damage shown by the electroretinogram follows a different action spectrum without the increased effect in the blue. There appears to be more than one mechanism for retinal damage in chronic light exposure, and at least one mechanism is not dependent solely upon the visual pigment and the pigment epithelium. Thresholds of permanent damage appear to be within one or two log units of light levels encountered in the normal visual environment. Newer data suggest that this damage is additive. Daily one hour exposures for four days produce damage equivalent to a single four hour exposure at the same retinal irradiance.

INTRODUCTION

Our work in chronic light damage began in 1967, shortly after Noell's (1966) report on retinal damage in rats. We have established the permanent damage threshold for white light and several lines of the argon laser in rabbits (Lawwill, 1973) and monkeys (Lawwill, 1972, 1973, 1976). The threshold retinal irradiance for damage in these two diurnal animals for four hour exposures to white light is at least three log units higher than that in the rat (Lawwill, 1972, 1973). Noell (1971) has reported that in the rat the damage shows a spectral sensitivity similar to visual sensitivity. The spectral sensitivity in the rat is different from what we have found (Lawwill, 1972, 1973) and from that which Ham (1975) has reported in monkeys.

In our experimental model, we are exposing a large area of the posterior pole to a relatively even illumination of moderately bright light for a period of hours. We are not dealing with the thermal damage caused by small spot laser burns and photocoagulation. The level of light we use is unlikely to

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raise the temperature of the retina even one degree centigrade (Clarke & Geeraets, 1969).

We find not only that the threshold is lower with blue light, but that the morphological changes may be slightly different from those previously described for white light and 514.5 nm laser light. Primary damage occurs in retinal layers which we think of as relatively transparent to the damaging wavelength. We find that no retinal layer has a threshold different from other layers and that the variability throughout a single fundus is large, both as to which layer is affected and as to whether there is damage. The location and degree of damage may be very different in physically adjacent areas of the fundus.

METHODS

The experimental animals are female rhesus monkeys weighing between 3 and 4 kg. Prior to exposure, the animal is given 0.12 mg of atropine sulfate and 8 mg of phencyclidine HCl intramuscularly. When the monkey is sedate, it is placed in a primate chair. Initiation of anesthesia is by means of a 20 mg/kg intravenous dose of sodium pentobarbital. A constant intravenous infusion of sodium pentobarbital (0.22 mg/min., 2.84 mg/ml) is begun and is continued throughout the four hour exposure. The eye to be exposed is dilated with one drop each of atropine 1%, tropicamide 1%, and phenylephrine 10%. The light is presented to the eye in Maxwellian view as diagrammed for the white light souce in Figure 1. The eye lid is held open by a blepharostat or adhesive tape and saline irrigation keeps the cornea moist. The alignment and position of the eye in the exposure beam are constantly observed on closed-circuit television. The intensity of exposure is constantly monitored via a beam splitter in the light path reflecting on an Eppley thermopile. All exposures are for a period of four hours with a constant intensity light source covering either 50.1 degrees solid angle (1.5 cm^2) or 102 degrees solid angle (2.5 cm^2 of retinal area). The intensity of the beam is measured with a Gamma Scientific Model 2020a spectroradiometer calibrated with a standard spectroradiometric source traceable to the National Bureau of Standards. The homogeneity of the field is checked with a small (2.5 mm diameter) cosine receptor by measuring the intensity at the center and edge of the field. The uniformity is maintained within 30%.

The retinal area exposed is calculated both from the angle of convergence of the incident beam and from direct measurement of the chord of the exposed section and the diameter of the eye using a freshly enucleated monkey eye. The area value used in calculating the irradiance is taken from the direct measurement. There is about a 20% difference between the two results.

We evaluate retinal damage induced by light by four measures: ERG, ophthalmoscopy, light and electron microscopy. Our methods of evaluation have been reported (Lawwill, 1973, 1972, and 1976).

The flash ERG is recorded prior to exposure at least twice a week until a stable amplitude is achieved. At 24, 72, and 144 hours after exposure, and two or three times per week thereafter, the ERG is recorded binocularly by the procedure established in this laboratory (Lawwill, 1972). In brief, the animal is tranquilized with 8 mg phencyclidine HCl, I.M., and the pupils are dilated with tropicamide 1% and phenylephrine 10%. Modified Burian-Allen type monkey contact lens electrodes are inserted and the animal is pre-adapted in a 1370 cd/m^2 white ganzfeld hemisphere. The stimulus is provided by a Grass PS2 photostimulator with the intensity set at sixteen. The xenon flash lamp of the Grass instrument is placed inside the hemisphere to provide a ganzfeld type stimulus.

ERG's are recorded, beginning three minutes after completion of two

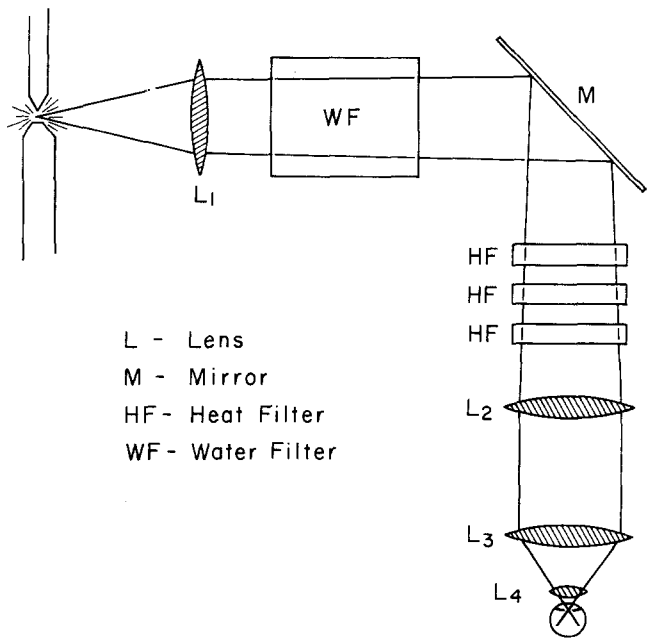

Fig. 1. This is a schematic drawing of the light exposure optics for white light. The collimated beam is passed through water (W.F.) and glass (HF) heat filters and through the final lens (L3, L4) into the eye. The beam is focused in the pupil and spreads out to cover a very large area of the fundus. For laser exposures, an expanded collimated beam is passed through the final lens only and produces an even pattern of illumination over the same large area of the fundus.

minutes light adaptation, and then every three minutes for twenty-seven minutes. A typical ERG daily record for one monkey is shown in Figure 2. For presentation, the a- and b-wave amplitudes for each eye for the seventh flash, i.e. following twenty one minutes of dark adaptation, for each day are charted. Evaluation of the functional damage after exposure is made on the basis of the decrease in amplitude and the persistence of this decrease after exposure. The response of the opposite eye serves as a control during this period. The damage is graded on a scale from 0 to 4+. In all cases. the quantification of damage is carried out in a double blind manner.

Ophthalmoscopy is performed regularly before and after exposure with the indirect ophthalmoscope; and any change is graded on a scale of 0 to 4+, without the grader having knowledge of the exposure level. The slightest question of edema or pigmentary change is graded as +. A definite change in appearance of the fundus no matter how transient, is graded 1+. A 4+ grading is assigned when there is extensive damage to the retina and pigment epithelium which persists indefinitely, showing apparently stable findings two to eight months after exposure.

The second eye is used as an experimental eye well after the first eye has stabilized, usually after sixty days. The monkeys are terminated at various intervals after the second eye is exposed, usually sixty days following ex-

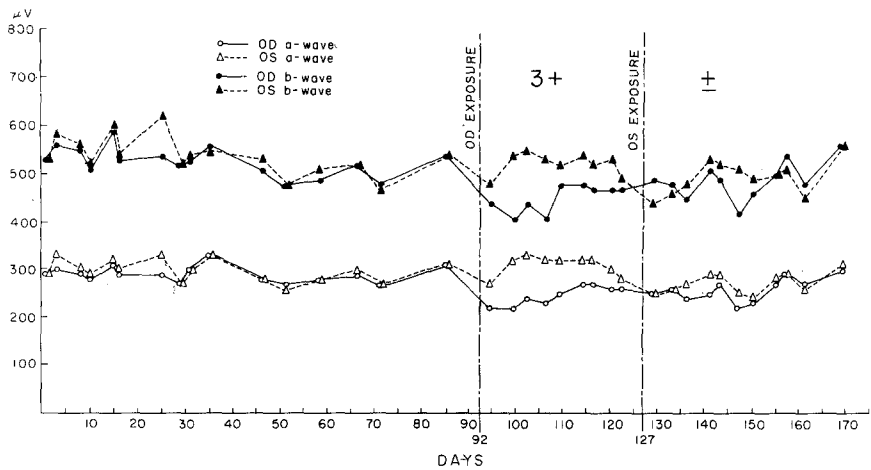

Fig. 2. This is the record of the ERG a- and b-wave amplitudes of both eyes of a monkey over a 170 day period. The points are for the seventh flash in a series recorded every three minutes after beginning dark adaptation. The day of exposure is marked for each eye. The grades assigned for damage are recorded above.

posure. The animal is anesthetized. The eyes are immediately enucleated, slit circumferentially, and placed in phosphate buffered glutaraldehyde solution (Sabatini, 1963) at room temperature. After twenty-four hours, the eyes are removed from the fixation and divided in half through the macula and optic disc. One half is processed for paraffin embedded light microscopy. The other half is placed in phosphate buffered wash solution according to the technique of Gordon et al., (1963), and post fixed in phosphate buffered 1% OsO_4 solution (Millonig, 1961) at four degrees C. for one hour. The tissue is rinsed and dehydrated in a graded series of ethanols, then infiltrated with Maraglas 655. At this time the eye is cut into small pieces in a grid system and oriented in flat embedding molds containing Maraglas.

The LKB ultramicrotome is used to further orient the blocks so that a perpendicular plane of the retinal layers can be viewed. Sections 0.5 μm to 1 μm are placed on glass slides and stained with a phosphate buffered 1% toluidine blue, viewed and photographed with a Zeiss photomicroscope.

Thin sections exhibiting silver and gold interference colors are picked up on 300 by 150 mesh grids, triple stained with Reynolds lead citrate (1963) uranyl acetate and lead citrate, and examined with a Siemen's Elmiskop 1A electron microscope.

Histological change in each of six retinal layers is graded and recorded separately, without the examiner having knowledge of the exposure history of the specimen. A slightly different grading system is applied for each of the six layers. In general, the least observable change from normal architecture is graded $\pm$. In the case of pigment epithelium, this means a change in the morphology of the pigment, and an increase in mitochondrial swelling. For the cone outer segments, $\pm$ means mild shrinkage and distortion of the proximal discs; for the rod outer segments, vesicle formation and minor thumbprint patterns; for the inner segments, swelling of mitochondria and distortion of the junction with the outer segment; for the outer nuclear layer, swelling with a decreased density of nuclei; for the inner nuclear layer, cellular swelling and minor nuclear chromatin clumping; and for the ganglion cell layer, minor swelling. For this grading system the variable swelling of the fiber layers is not included, though swelling in the plexiform layers is also apparent evidence of damage. The category of 1+ for each layer begins with the detection of those changes which imply cell death, such as cellular membrane disruption or pycnosis of nuclei, when limited to only an occasional cell in the layer. 2+ and 3+ signify death of more cells and 4+ indicated severe damage or total loss of elements within that layer. The results section will better illustrate the histological grading system.

RESULTS

Figure 3 is an electron micrograph showing the mildest effects of light on the pigment epithelium. There is a transformation of the pigment from the normal cigar-shaped bodies to rounder, larger 'balled-up' forms located more deeply in the cell. There are also associated lipofuscin granules. The earliest swelling of the mitochondria is shown. This section would be graded a ± change of the pigment epithelium. Figure 4 (Plate I) shows 1+ changes of the pigment epithelium using light microscopy of a thick plastic embedded section. In this retinal section near the macula, there is some 'balling-up' of the pigment epithelium. There are also early changes in the nuclei consisting of crenation. Figure 5 (Plate I) shows 3+ changes in the pigment epithelium with loss of pigment from pigment epithelial cells, swelling, and the beginning formation of phagocytes. Pigment is included in the phagocytic cells rather than in the pigment epithelium in some areas. Figure 6 (Plate I) shows almost total loss of pigment epithelial cells with extensive phagocy-

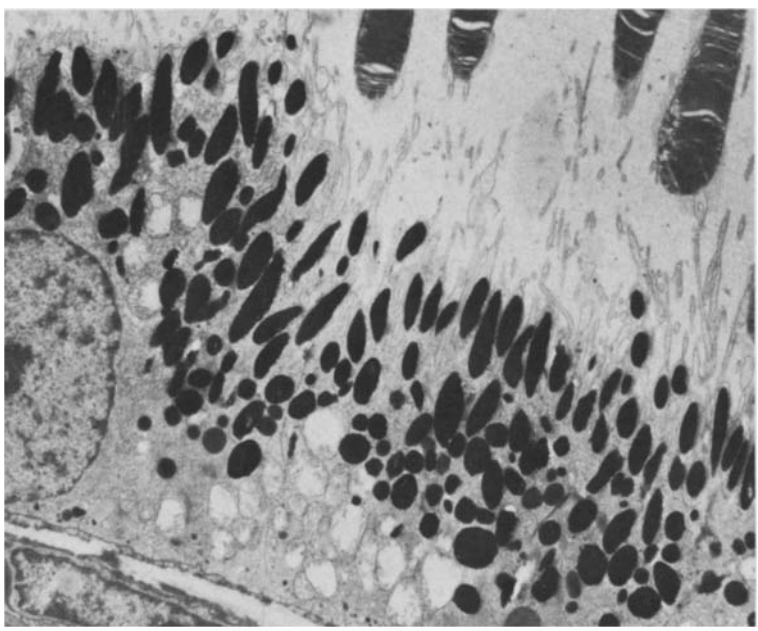

Fig. 3. Electron micrograph (x5,000) of portion of pigment epithelial cell and adjacent outer segments showing 'balling-up' of pigment granules and mitochondrial swelling. These minor changes which are not as evident in normal eyes are graded ±.

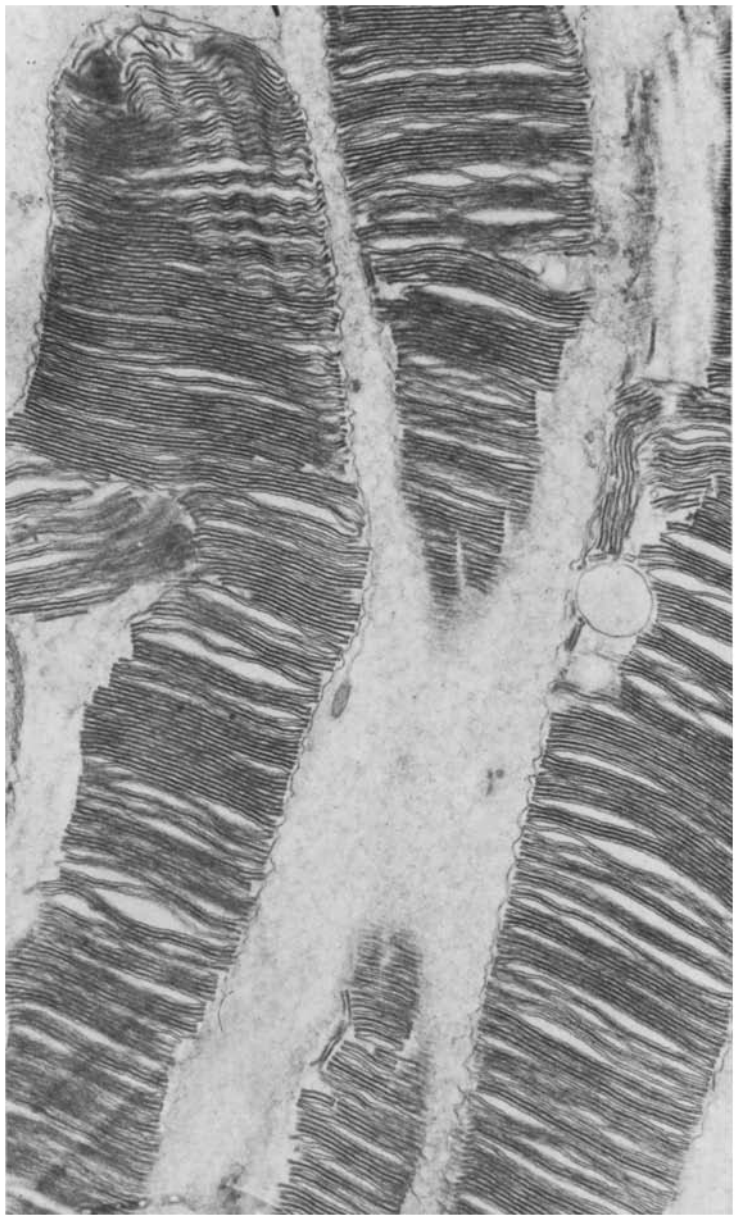

Fig. 7. Electron micrograph (x22,000) of ± changes in the outer segments. There is vesicle formation and disorientation of the lamellae of the outer segments.

tosis in the area of the outer segments and pigment epithelium. This is a 4+ change in the pigment epithelium.

Figure 7 is an electron micrograph showing the earliest distortions of the outer segments. This ± change consists of vesicle formation seen in the lamellae and mild distortion of the outer segments. Figure 8 also shows ± changes in the outer segment area. This swelling of outer segments is quite prominent as an early change and may still be present up to two months after the initial insult. Figure 9 (Plate I) shows 2+ changes in the outer segments. There is severe distortion of the outer segment area and some outer segments are missing. Figure 10 (Plate I) shows 4+ changes with complete loss of outer segments.

Figure 11 is an electron micrograph showing a swollen cone inner segment

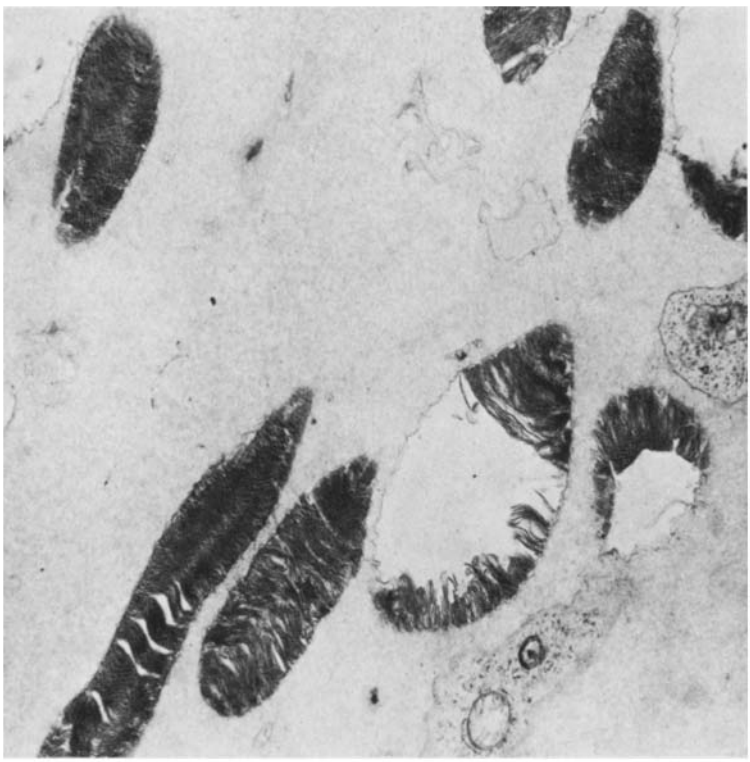

Fig. 8. Electron micrograph (x9,000) of ± damaged outer segments. This swelling and separation of the cellular membrane can persist for several months after the exposure.

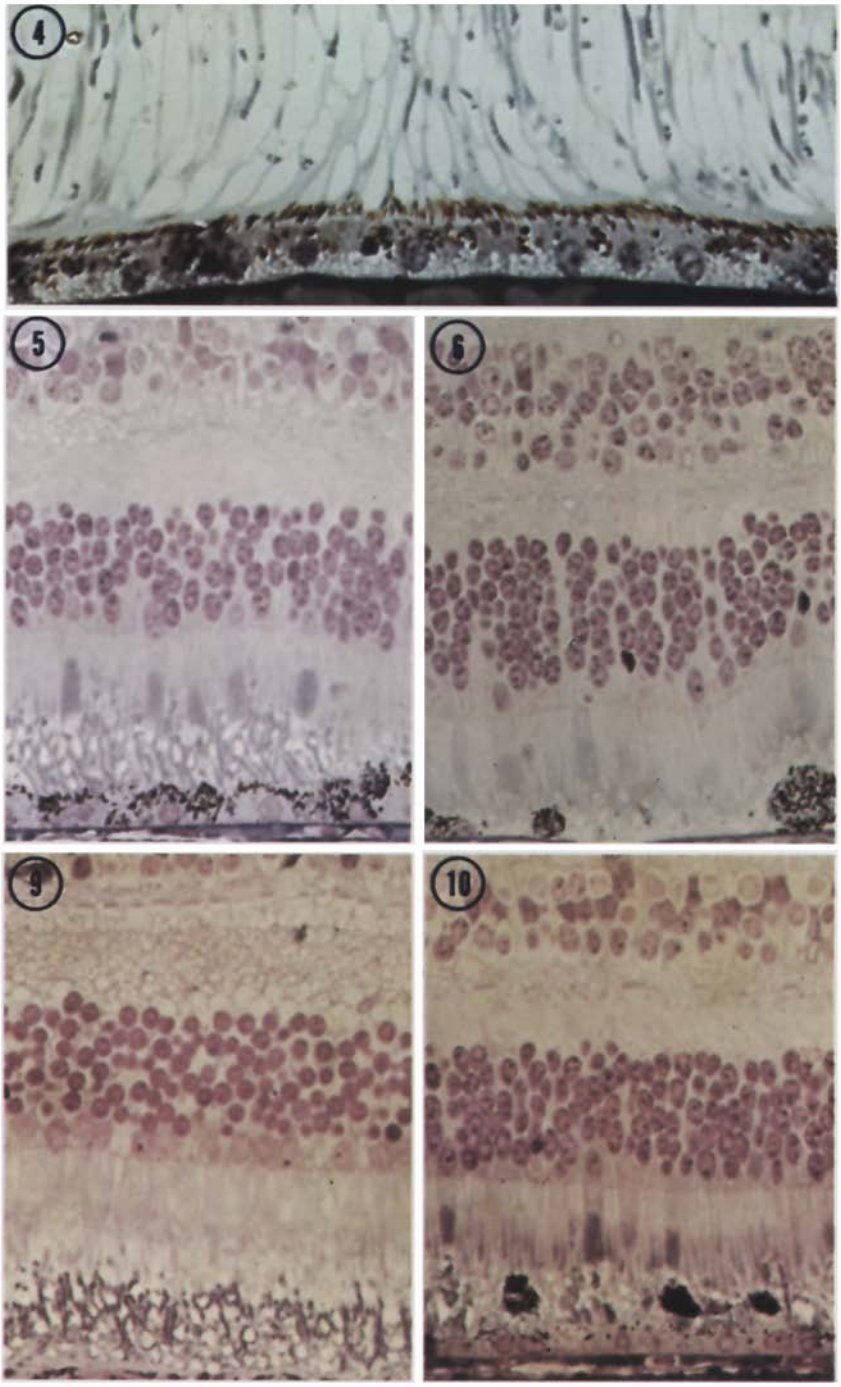

Fig. 4. (Plate I) Light photomicrograph (x600) of the pigment epithelium showing extensive mitochondrial swelling and mild crenation of pigment epithelial nuclei. These changes are graded ±.
Fig. 5. (Plate I) Light photomicrograph (x400) showing 3+ changes in the pigment epithelium with cellular swelling, loss of pigment from the cells and the beginning of phagocyte formation with engulfed pigment.
Fig. 6. (Plate I) Light photomicrograph (x400) showing 4+ pigment epithelial changes with total loss of the pigment epithelium and extensive phagocytosis.
Fig. 9. (Plate I) Light micrograph (x400) showing 2+ changes in the outer segments with severe distortion of the outer segments and with some segments totally disrupted.
Fig. 10. (Plate I) Light micrograph (x400) showing complete destruction (4+) of outer segments. The entire receptor cell is affected in this area.

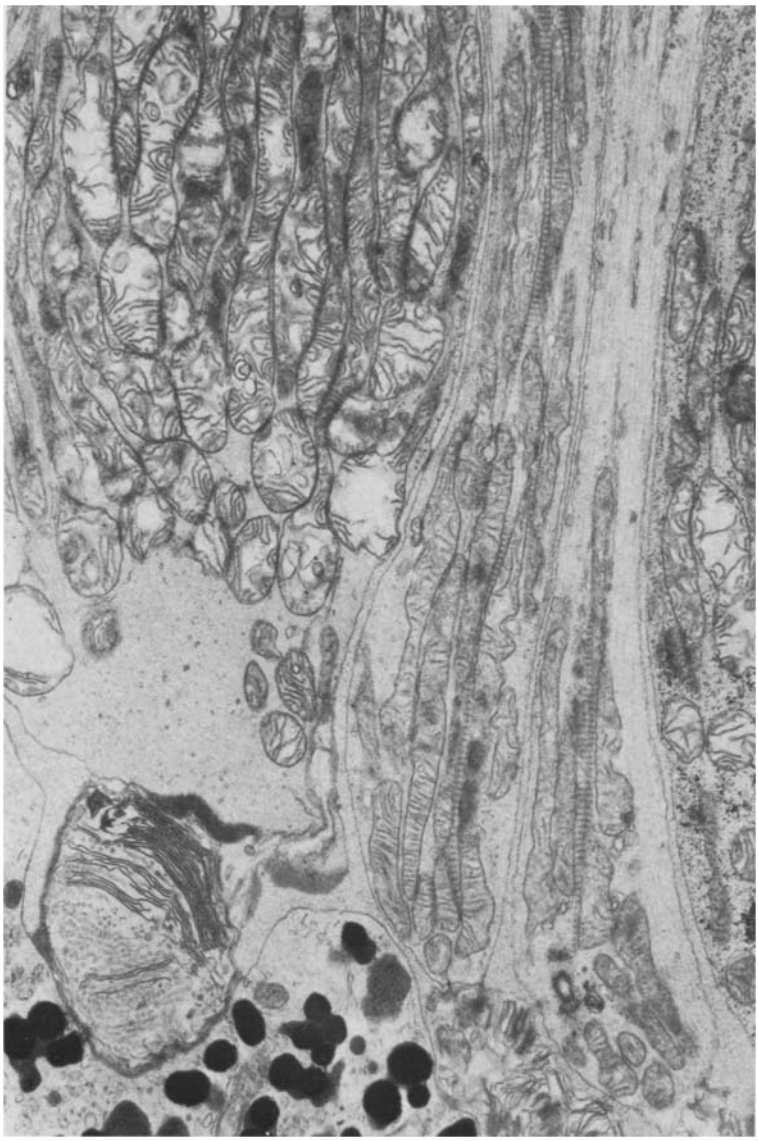

Fig. 11. Electron micrograph (x10,500) showing a cone inner segment, distorted mitochondria, and distorted connection to its outer segment surrounded by more normal rod inner segments. This is graded a ± change of the inner segments.

surrounded by normal rod inner segments. The mitochondria of the cone are particularly swollen and the connection to the outer segment is distorted, but the segment appears viable. This is graded as a + change of the inner segments. Figure 12 (Plate II) shows 1+ changes of the inner segments. The inner segments are more severely swollen, and there is a necrotic segment present. Figure 13 (Plate II) shows 4+ changes with total necrosis of the inner segments.

Figure 14 (Plate II) shows 1+ changes in the outer nuclear layer. There is scattered pycnosis. Figure 15 (Plate III) shows 4+ changes in the outer nuclear layer, but only ± changes in the inner nuclear layer. There is extensive pycnosis in the outer nuclear layer while there is only swelling and occasional distortion of the nuclei in the inner nuclear layer. Figure 16 (Plate III) shows 1+ changes in the inner nuclear layer with more numerous pycnotic nuclei.

Figure 17 (Plate III) shows extensive pycnosis in the inner nuclear layer and is graded 2+ for this layer.

Figure 18 (Plate IV) shows 2+ changes in the ganglion cell layer. The cells are swollen and a few are necrotic. Figure 19 (Plate IV) shows 4+ changes in the ganglion cell layer as well as the outer nuclear layer with extensive changes and many pycnotic nuclei.

We have now exposed eighty-seven monkey eyes to six different wavelengths of light at retinal irradiances between five microwatts and 100 milliwatts per square centimeter of retina. Our evaluation has included over 3,000 ERG's and histologic sections of eighty one eyes. Two hundred thirty-one plastic embedded blocks on twenty-six eyes have been evaluated and analyzed, so far. We have recorded damage grades on each block for six retinal layers, separately (pigment epithelium, outer segments, inner segments, outer nuclear layer, inner nuclear layer, and ganglion cell layer). These damage scores have been made into a histogram for each block, each representing an area of a fundus. Reproductions of these histograms have then been placed on drawings which depict the location in the fundus from which the blocks were taken. The resulting pattern of damage has been evaluated subjectively. In Figure 20, block A1 includes the area around the disc and block A3 includes the central macula. The central macula is spared, while the blocks on either side show more extensive changes. The area under the fiber layer next to the optic disc also shows less damage, as do the more peripheral areas.

The histological findings which we have described have not been found in unexposed normal eyes fixed by our methods. The degree and area of retina affected by these changes has paralleled the exposure level even though the exposure level was not known at the time the grading was performed. We,

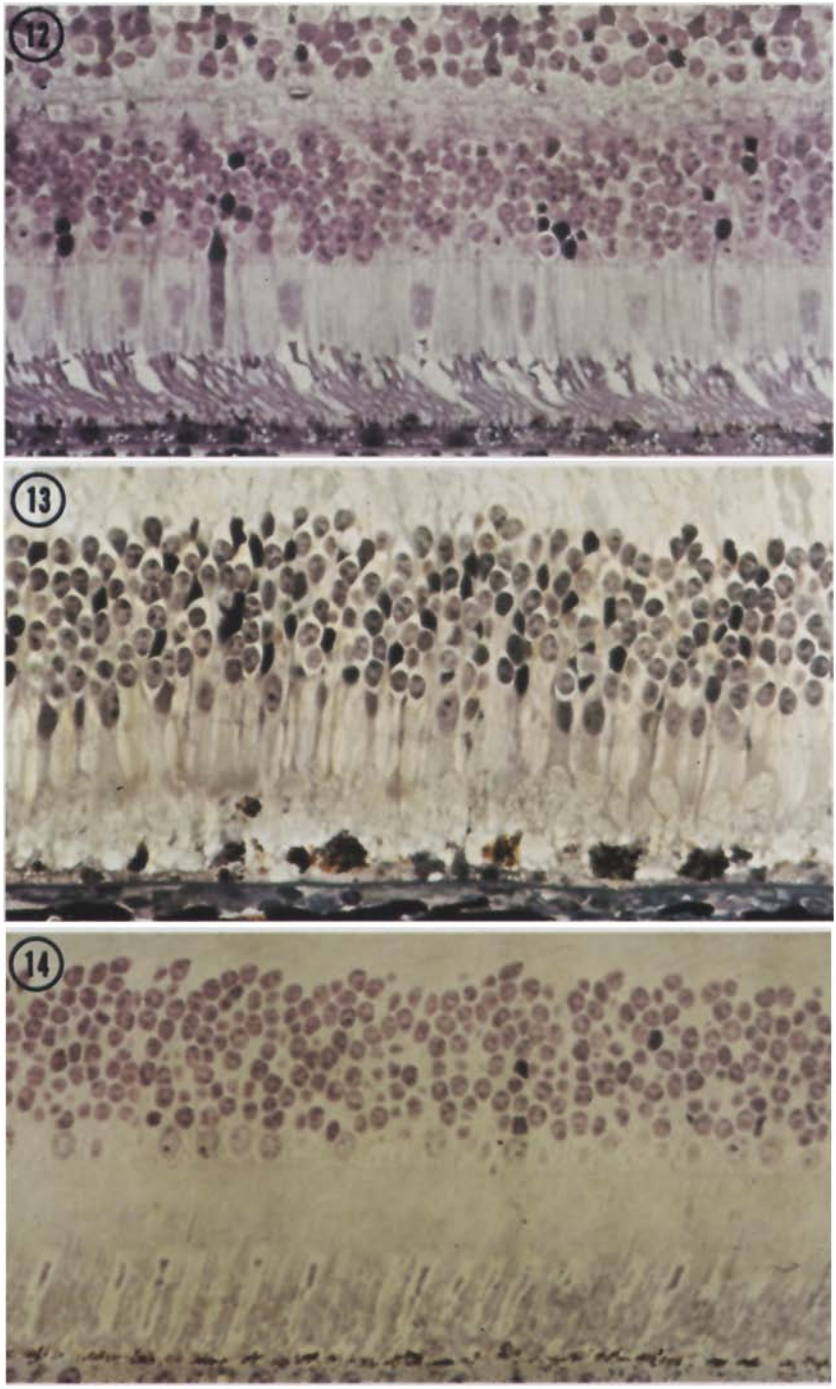

Fig. 12. (Plate II) Light micrograph (x400) showing 1+ inner segment changes. Most segments are severely swollen and at least one is obviously necrotic.
Fig. 13. (Plate II) Light micrograph (x400) showing total (4+) necrosis of the inner segments. The outer segment and pigment epithelium are also destroyed.
Fig. 14. (Plate II) Light micrograph (x400) showing 1+ changes in the outer nuclear layer where there is scattered pycnosis of nuclei.

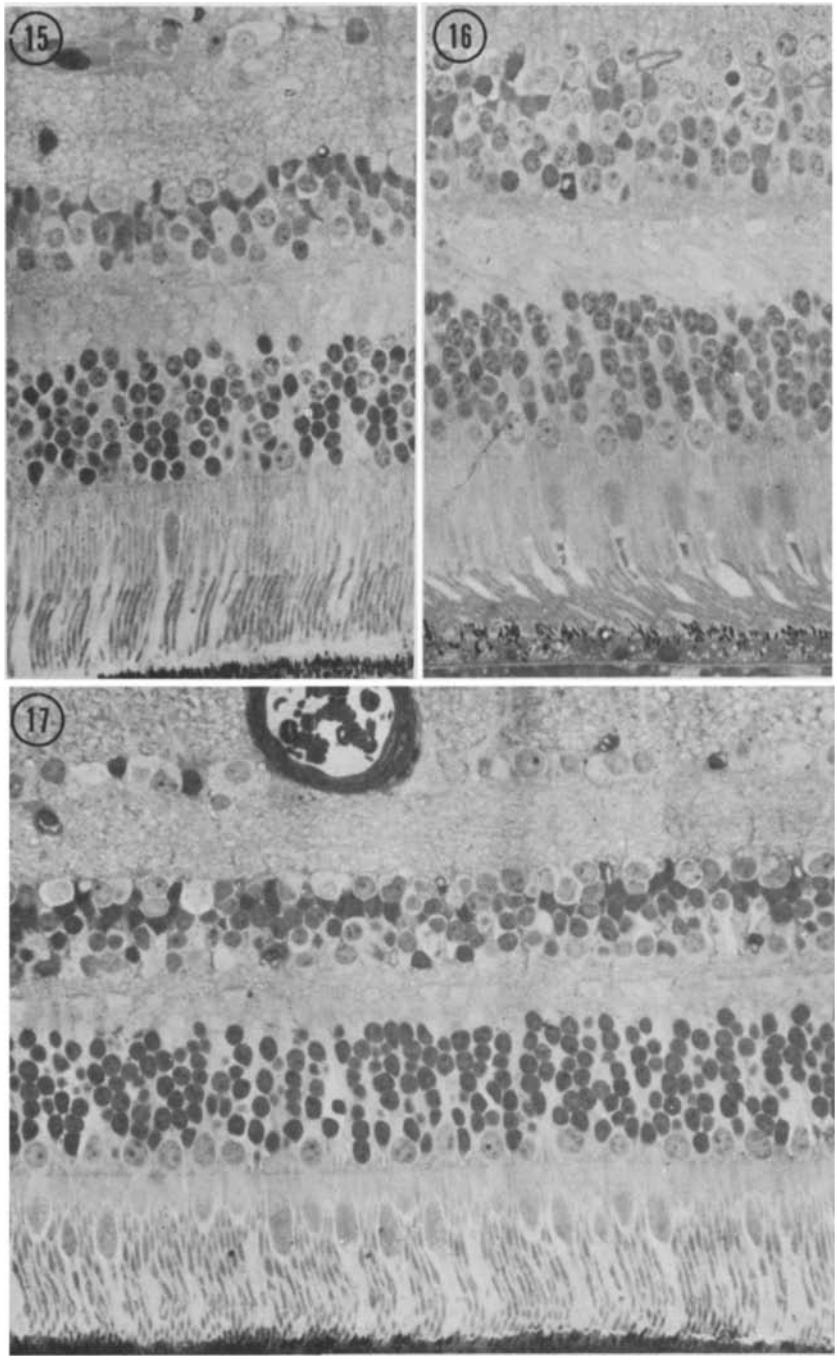

Fig. 15. (Plate III) Light micrograph (x400) showing 4+ changes in the outer nuclear layer where there is extensive pycnosis of the nuclei. There is also swelling and some distortion of the cells of the inner nuclear layer.
Fig. 16. (Plate III) Light micrograph (x400) showing 1+ changes of the outer nuclear layer with scattered pycnotic nuclei.
Fig. 17. (Plate III) Light micrograph (x400) showing more extensive pycnosis of the inner nuclear layer, graded 2+.

therefore, believe that these changes which we have described are a direct result of the exposure.

The findings of the histological evaluation of twenty-six eyes are that:

(1) The damage is very patchy and varies randomly as to the retinal layer and fundus area most affected, except for specific changes in and around the macula and the optic disc. (The central macular area consistently shows less damage than the area just surrounding the macula. The parafoveal and paramacular areas show the greatest amount of damage in all layers. The area underlying the thick nerve fiber layer adjacent to the disc is less affected.)

(2) As threshold is neared, the damage to the pigment epithelial and ganglion cell layers decreases most rapidly; and, at the lowest levels, the cone and the inner and outer segments are the only structures affected.

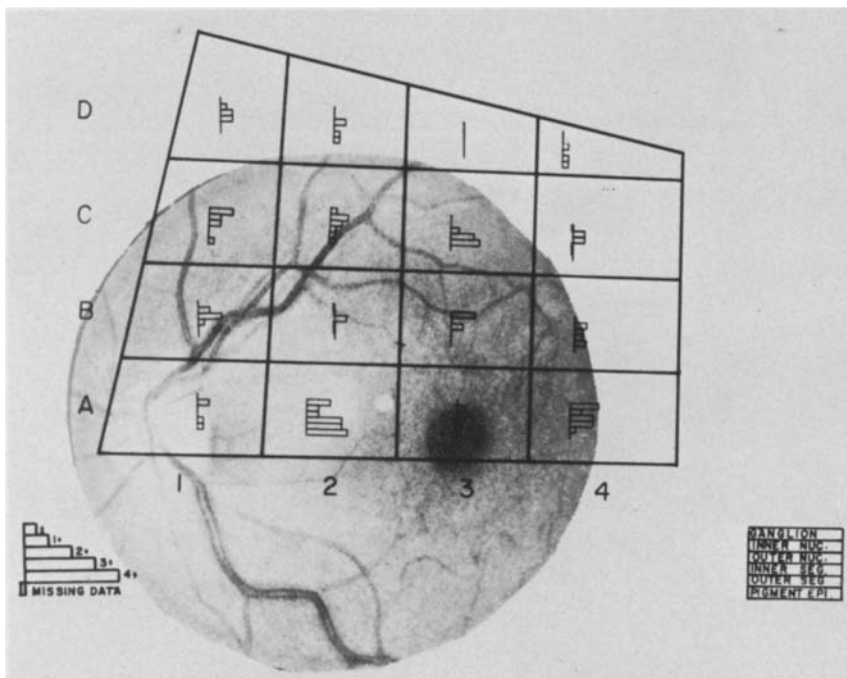

Fig. 20. Diagram showing the areas of one eye from which blocks were taken for histology. The histograms denote the grade of damage seen in that block for each of six retinal layers. The top of the histogram represents the inner layers while the bottom is the pigment epithelium. This is a sample of the procedure used in twenty-six eyes to describe the distribution of damage by areas and layers.

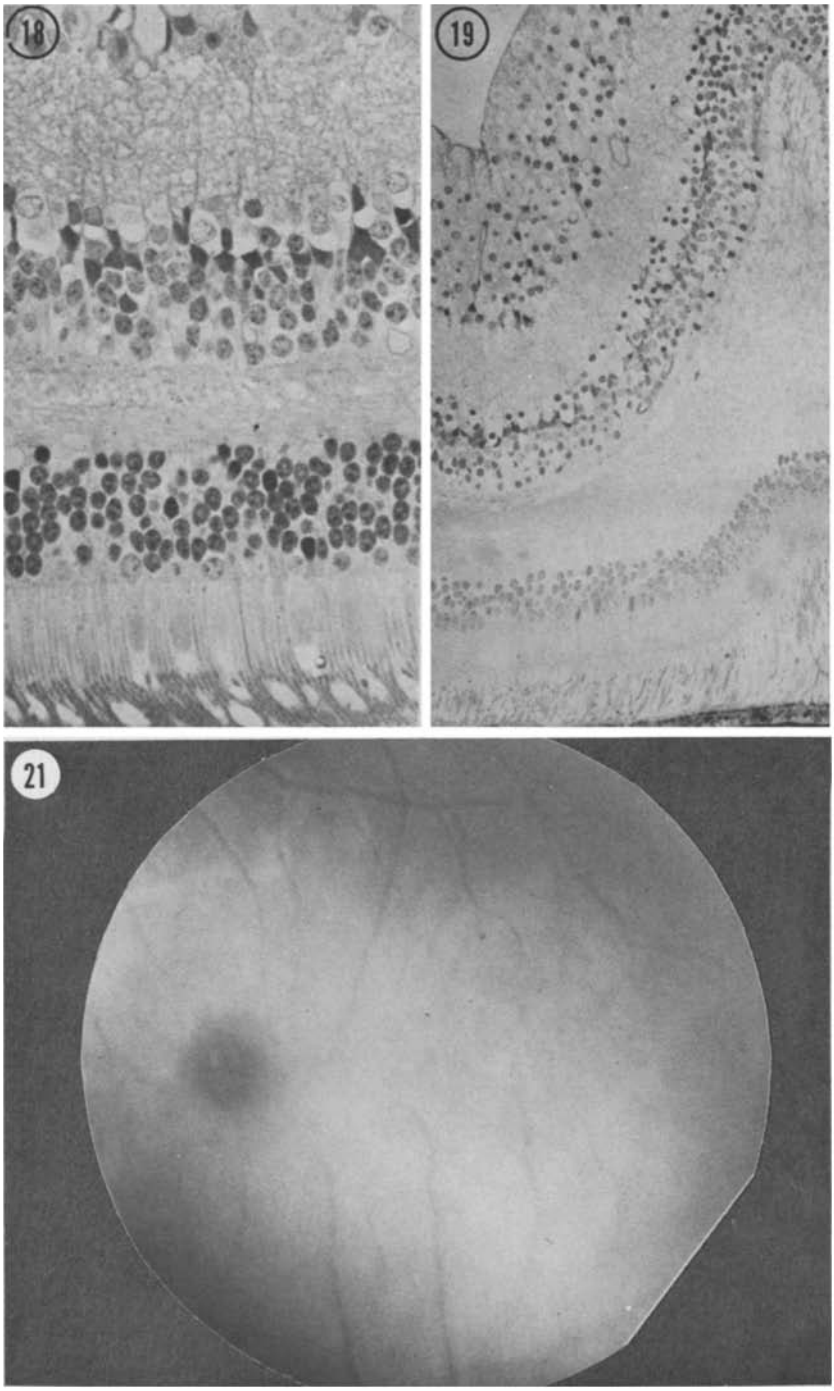

Fig. 18. (Plate III) Light micrograph (x400) showing 2+ changes in the ganglion cell layer with swollen cells and a few necrotic cells.

Fig. 19. (Plate IV) Light micrograph (x180) showing extensive, 4+ changes in the ganglion cell layer as well as the outer nuclear layer. There are many pycnotic nuclei in each layer.

Fig. 21. (Plate IV) Fundus photograph of an eye with 4+ damage showing extensive loss of pigment epithelium. The central macula is better preserved. The demarcation line between severely damaged pigment epithelium and less damaged is very distinct. There was not a difference in light exposure level coincident with this demarcation line

(3) In the macula, the thick internal layers may be severely damaged when the pigment epithelium and outer retinal layers show no changes.

The second area of interest is the relationship of functional change to histological change. As shown in figure 2, the flash ERG returns to near normal after two to four weeks in spite of widespread severe ophthalmoscopically and histologically observed damage. The ERG amplitudes continue to be depressed only in the most extensively damaged eyes.

Figure 21 (Plate IV) shows 4+ damage on a fundus photograph. There are widespread pigment epithelial defects present. Fluorescein angiography of the same fundus highlights the pigment epithelial defects in this severely damaged eye.

Histological sections are slightly more sensitive than the ERG in detecting damage, but in the case of blue light there is an extreme discrepancy. Figures 22, 23, 24, and 25 show the damage evaluation results for all four techniques for all eyes in each exposure group, plotted against exposure level. When a particular measure is repeated for different techniques in the same eye or in another eye exposed at the same level, each is plotted. Each dot on the graph represents a level of damage for one test occurring in one eye at

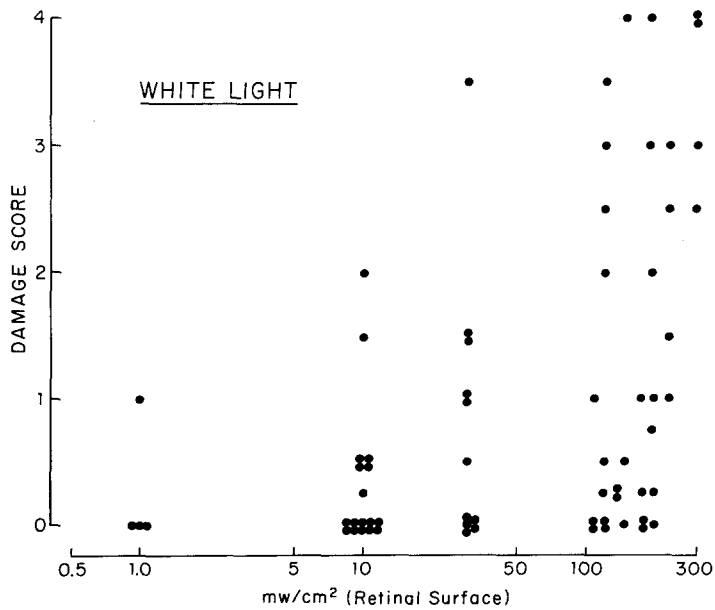

Fig. 22. Graph of damage scores for white light exposures plotted against retinal irradiance not corrected for ocular transmission. The threshold for minor damage is around 10 mw/cm^2.

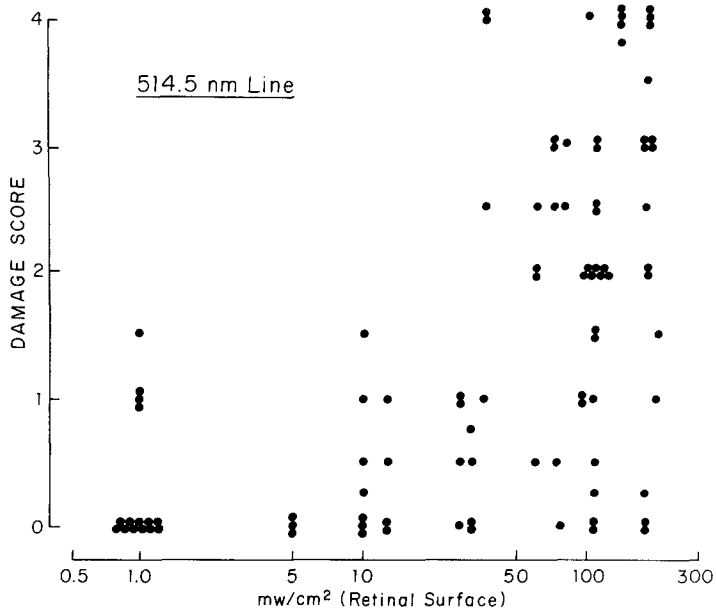

Fig. 23. Graph of damage scores for 514.5 nm exposures plotted against retinal irradiance not corrected for ocular transmission. The threshold for damage is around 10 mw/cm^2.

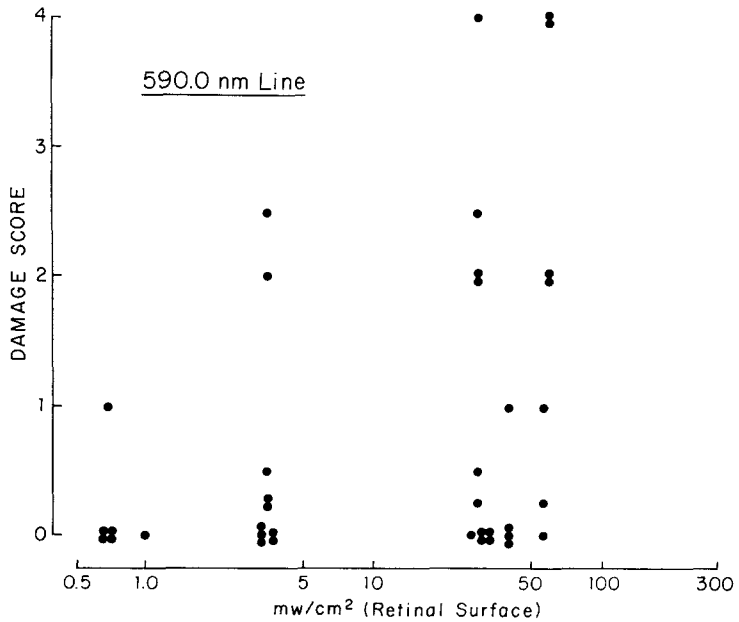

Fig. 24. Graph of damage scores for 590 nm exposures plotted against retinal irradiance not corrected for ocular transmission. The threshold for damage appears to be around 10 mw/cm^2.

that exposure level. In an attempt to detect the lowest level of damage, we have selected very sensitive grading criteria. This causes a higher proportion of false positive results. Therefore, we take 1+ to be the lowest level of definite light related damage. There are also a few 1+ scores which we suspect are artifactual, but the thresholds are determined from the overall trend of the data. In Figure 22, our threshold data for white light shows 1+ damage at approximately 10 mw. In Figure 23, the data for the 514.5 nm green line is not much different except that a greater chance of severe damage is present slightly above threshold. In Figure 24, the threshold for damage at 590 nm is not greatly different from that for white light or the green 514.5 nm line. In Figure 23, the threshold for the blue 457.9 nm line is significantly different showing damage at 2 mw or less. The histopathological comparisons in twenty-six eyes show little difference in damage between the several wavelengths as to which retinal layers sustain more damage. Figure 26 shows that the histological damage scores are the same or slightly lower than the ERG damage scores for the 514.5 nm line; but for the 457.9 nm line (Figure 27), the histological damage is proportionately higher than the functional, ERG change.

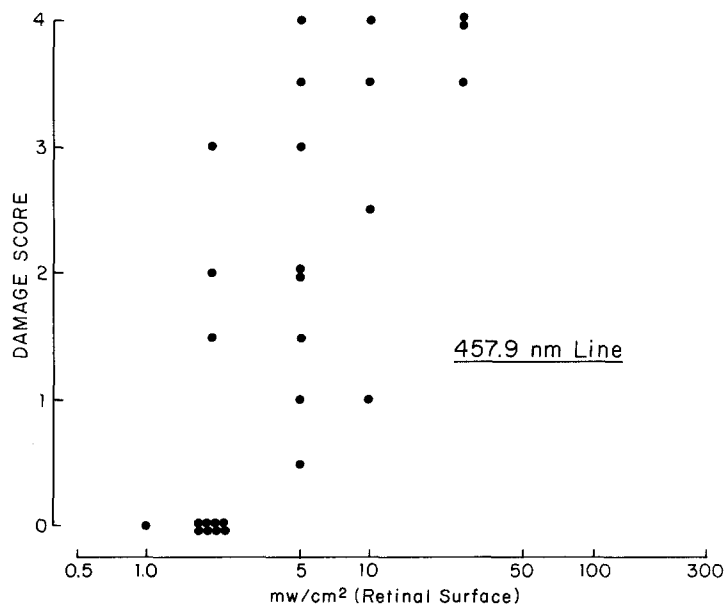

Fig. 25. Graph of damage scores for 457.9 nm exposures plotted against retinal irradiance not corrected for ocular transmission. The threshold for damage is at least as low as 2 mw/cm^2 and may be slightly lower.

Figure 28 shows the damage threshold data for the 488 nm line plotted on the same axis with the ERG a-wave threshold for the same line. For this purpose, the ERG was produced with flashes from the exposure system with the monkey in the normal position for exposure. The monkey was dark adapted for twenty minutes. Neutral density filters were used to adjust the intensity of the beam, and a mechanical shutter was used to deliver 20 ms stimuli from the system. The ERG's were recorded and measured by the same method as for exposures.

Figure 29 gives relative luminances under several common conditions with four hour damage thresholds on the same scale. Human psychophysical matches were made between the exposing beam of the experimental apparatus in Maxwellian view and the diffuse reflection from a white card illuminated by a portion of the same beam. The photopic luminance of the card was measured directly with a photometer. The exposure beam was measured, and the irradiance on the human retina was calculated. These ratios between retinal irradiance and photopic luminance were used to plot the damage thresholds on the common luminance scale.

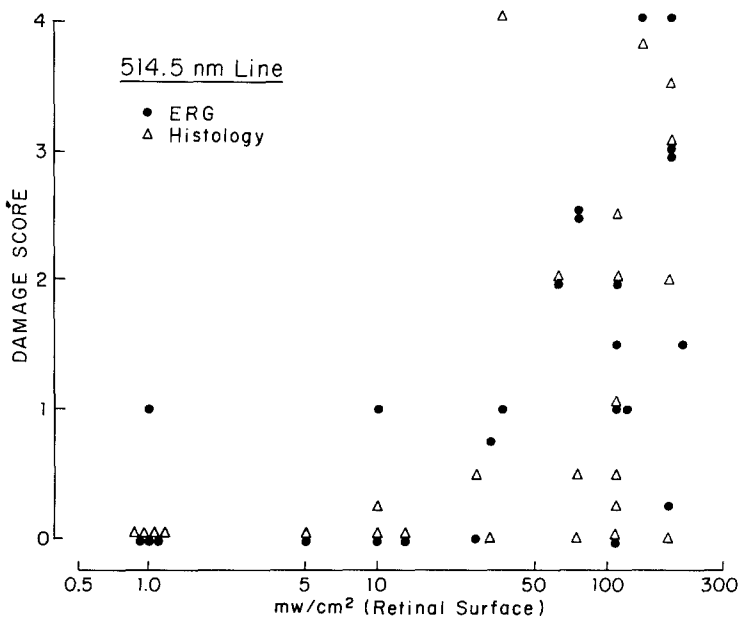

Fig. 26. Graph of damage for 514.5 nm exposures measured by ERG scores (○) and by histology scores (△) plotted against retinal irradiance not corrected for ocular transmission. The ERG is of slightly less sensitivity in detecting damage.

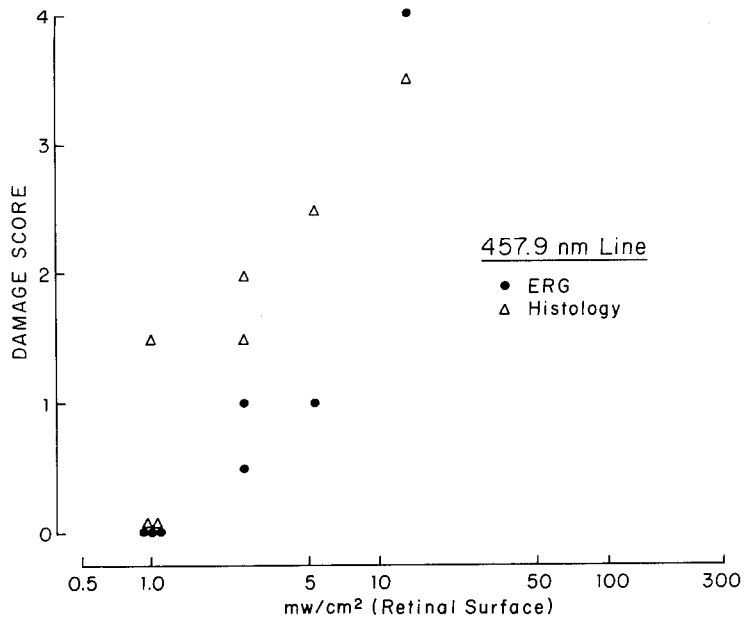

Fig. 27. Graph of damage for 457.9 nm exposures by ERG scores (○) and histology scores (△) plotted against retinal irradiance not corrected for ocular transmission. The ERG is much less affected than the histology would suggest at this wavelength.

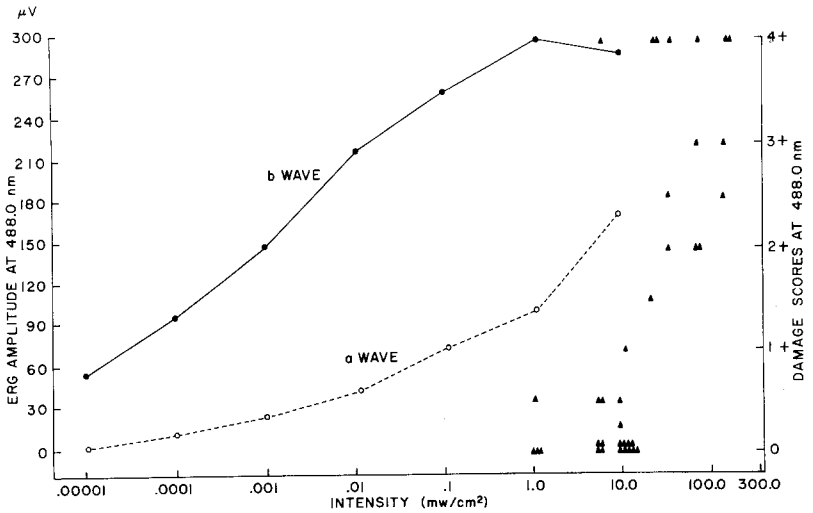

Fig. 28. Graph of damage scores for 488.0 nm exposures plotted against retinal irradiance not corrected for ocular transmission. The a-wave response for a 20 ms exposure to the same measured light source is shown on the same irradiance scale. The 40 μv a-wave threshold is about three log units less than the damage threshold.

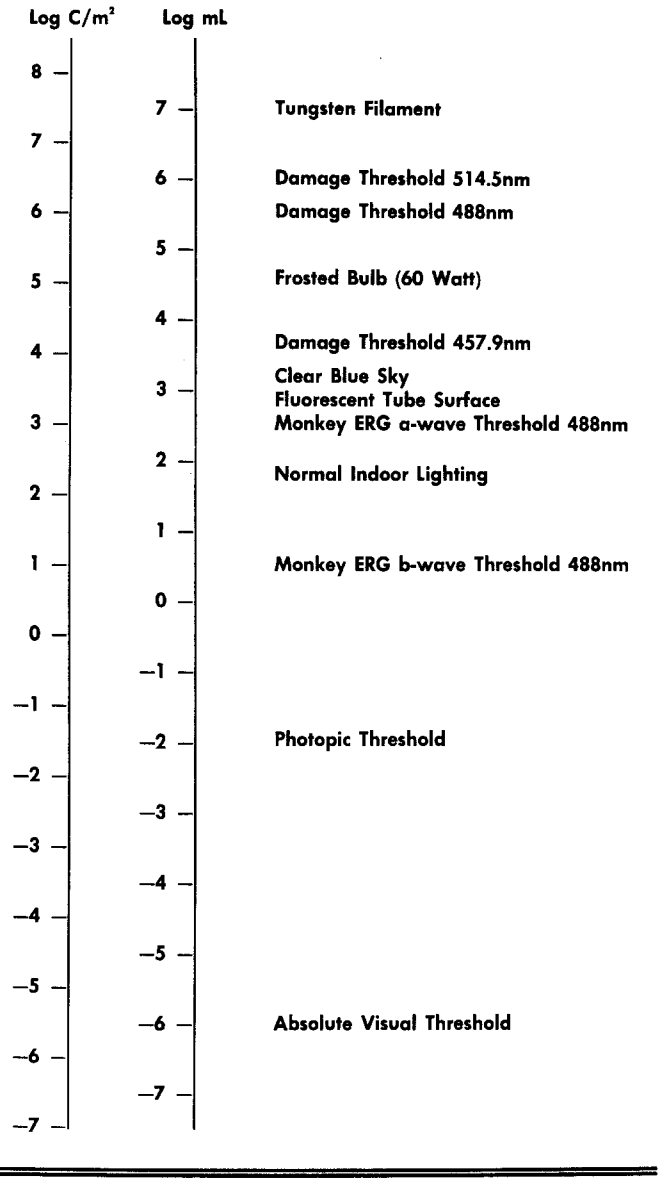

Fig. 29. This figure shows the relative brightness of common objects compared with damage thresholds for three laser lines converted to luminances by human psychophysical matching in the experimental apparatus. These matched ratios may differ from the calculated irradiance/luminance ratio by as much as 0.8 log unit.

The hypothesis that the receptors are primarily affected is not borne out by either the ERG or the histological data. The blue-light threshold is lower than the green-light threshold, and the receptors are not necessarily affected before the inner layers of the retina.

In the range within one log unit of threshold, all cellular elements of the retina are susceptible to direct light damage. Therefore, it appears unlikely that the visual pigment or the visual pigment cycle is uniquely related to the damage mechanism. Whatever the absorber is, it is more effective in the blue and is spread evenly throughout the retinal layers and throughout the fundus; except that the central macula appears protected while the peripheral macula, and just beyond, appears particularly susceptible. There is a discrepancy between the histologically observed damage and the functional damage shown by the ERG. This discrepancy is greater at shorter wavelengths. This might imply two mechanisms with different spectral sensitivities operating simultaneously – one effecting histologically observable damage and one producing more transient functional changes.

The distribution of damage within the fundus which we have found here is different from that which we described in rabbits (Lawwill, 1973). In rabbits the damage was concentrated along the visual streak where there is the highest concentration of receptor cells. Since the damaged area was oblong in shape and any inhomogenieties of the round exposure field were concentric, the pattern was not an artifact of the exposure field. In the monkey the damage is slightly greater in an area seven to fifteen degrees from the fovea, but the difference is not as marked. This area in the monkey also contains the highest concentration of receptor cells, but no relationship between these factors is proved.

In the pigment epithelium, early damage is manifested by the disorientation of the pigment granules. They are no longer lined up parallel along the inner border of the pigment epithelial cells, but are distributed randomly in the inner two-thirds of the cell. This is associated with the configuration of the villae of the pigment epithelial cells, which appear shortened or absent. This change may reflect a retraction of the processes of the pigment epithelium in which some of the granules are located. Lipofuscin granules appear more numerous and the basal infolding is less prominent.

The threshold for measurable permanent retinal damage is surprisingly close to some everyday viewing conditions. The most intense conditions in everyday experience may be within one log unit of damaging levels of light, even though the retinal irradiance is reduced by a constricted pupil. As the light approaches the blue end of the spectrum, less brightness is required to

cause damage because the damage threshold in irradiance is less and the same irradiance produces less brightness. It appears that there is a slight overlap between potential exposure and damage.

Sperling & Harwerth (1971) have found permanent psychophysical colored light threshold shifts in rhesus monkeys after exposures at even lower retinal irradiances. Marshall, Mellerio & Palmer (1972) have seen specific cone outer segment changes in pigeons after twelve hour exposures at a retinal irradiance of about 1 mw/cm^2 of white light. These findings are in reasonable agreement with our thresholds.

Though, it is likely that discomfort would preclude our enduring light levels which would be damaging, it is still possible that prolonged light exposure could potentiate aging of the retina in susceptible individuals. Further studies of additivity are necessary. We suspect additivity because one hour exposures on successive days at 20 mw/cm^2 at 514.5 nm have produced the same damage expected for a single, four hour exposure. These initial findings must be substantiated by further investigation, but these results could suggest danger of retinal damage in humans under what are thought to be normal environmental conditions. If environmental conditions are so close to tolerance, it would not be unreasonable to postulate an increased aging effect on the retina by long term exposure to high level light. And, individual susceptibility would be expected to vary. One might ask if senile macular degeneration might be potentiated in susceptible individuals by a life time of high light levels.

SUMMARY

Threshold data for retina damage is reported for several lines of the argon and dye lasers for four hour exposures in the rhesus monkey eye. The eye is found to be more sensitive to damage from the 457.9 nm line than from those of longer wavelengths. Histological evaluation of 231 areas in twenty-six eyes shows that there is no specific retinal layer more susceptible to light damage at any particular wavelength. Susceptibility varies throughout the retina, with higher thresholds in the center of the macula and around the optic disc. These findings suggest that visual pigment is not the sole mediator of retinal damage in chronic light exposure. The damage threshold for four hour exposures is three to four log units above the ERG a-wave threshold for 488 nm light, and within one log unit of the luminance provided by a bright snowy landscape. The damage threshold expressed in luminance for 457.9 nm light approaches the highest lighting conditions found in everyday

life. Because of the differences between individuals, there may be people who will develop retinal damage at environmental levels of light. Some of the people who develop macular degeneration may be in this group.

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