Retinal Damage by Light

Practical Implications Related to Ophthalmic Instruments

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Retinal Light Damage

Light may cause damage to living tissue in a range from barely detectable, temporary changes to gross destruction. The eye and its retina, though designed to gather light necessary for vision, are also susceptible to such deleterious effects.

Although the visual system responds to wavelengths between 400 and 750nm, light in the range of 400-1400nm is transmitted by the ocular media and focused onto the retina. Within this spectral range, three types of retinal damage may occur: "(1)non-linear effects from picosecond and nanosecond pulses; (2) thermal effects for pulses in the range microseconds to seconds and for cw exposure to wavelengths above approximately 500nm; and(3) photochemical effects for long term exposure to wavelengths below approximately 500nm." [11] We will be concerned here with only the thermal and photochemical effects.

Thermal lesions, or heat burns of the retina, by ophthalmoscopic examination, appear as circumscribed lesions with well marked borders which contain a central area of yellow fluid. Photochemical lesions, in comparison, tend to have a very subtle yellow tinge throughout, and exhibit no distinct borders.[16]

It has been shown [1, 11] that the type of lesion which results is a factor of the spectral distribution of the incident radiation. Ham, et. al.[11] have shown that "threshold lesions from 1064nm exposures are purely thermal in type, while lesions from 441.6 nm exposures are purely photochemical in type." For intermediate wavelengths, the lesions produced represent a

combination of photochemical and thermal effects, thus implicating both mechanisms. Researchers [11] have proposed a thermally enhanced photochemical effect.

The thermal environment, as a single variable, has been shown to affect retinal damage. Increasing the body temperature of an animal during light exposure increases the rate and extent of retinal damage. Ward and Bruce[31] have shown that "irradiance thresholds for chorioretinal damage in rhesus monkeys vary linearly with core temperatures between 34° and 39°C." Noell, et. al.[22] found that the capacity of light to produce damage is extremely dependent upon the tissue temperature within an amazingly narrow range (1.5°C).

Roulier[28] lists the threshold for retinal coagulation as 15°C. above the ambient temperature, though it is noted by Kuwabara and Gorn[17] that a temperature rise of 4° to 5°C. above body temperature can generate irreversible damage in the sensory retina if of long duration. Vos and Clarke, et. al.,[30] found that an 8°-15°C. rise in temperature is required for thermal injury when photocoagulation is the mechanism of damage. Hansen and Fine[30] conclude that a 2°C. rise in temperature (as evidenced from fever) can be considered safe over an extended period of time.

Wavelength of the source, as another factor, is also found to cause variable effects. Anderson, et. al.[1] concluded that the high-energy wavelengths of the visible spectrum are primarily responsible for light induced retinal degeneration. Albino

rats were exposed for extended periods of time to 6 ft-c. (9.6×10^{-6} w/cm^2) of white (380-750nm), blue (437-538nm), green (545-580nm), or red (609-633nm) light; damage was determined histologically. Continuous blue light was the most effective in paralleling the effects of white light. Constant green light was approximately one-half as effective as blue light, and red was the least effective in producing retinal degeneration.

Solar retinitis, which results from prolonged or purposeful exposure to the sun, has traditionally been explained by a thermal mechanism. It was thought that the retinal pigment epithelium absorbs energy and converts it to heat with resultant destruction of the pigment epithelium and photoreceptors. Researchers,[11, 30] however, have more recently shown that such a lesion may not be explained solely in terms of thermal damage; the maximum temperature on the retina may be significantly less than the threshold for coagulation (from 1.5° to less than 3°C. above ambient).

Ham, et. al.[30] find it "plausible...to explain solar retinitis in terms of photochemical effects with some thermal enhancement from the infrared component in the sun's spectrum." They have demonstrated that "a simulated solar spectrum (400-1400nm) with a major portion of the energy in the short visible wavelengths was more damaging to the retina than a long wavelength spectrum (700-1400nm) by a factor of five." They also infer that for a continuous solar spectrum, as the retinal irradiance is reduced and exposure duration increased, thermal

insult becomes less pronounced while photochemical effects become more prominent. Theoretical considerations are cited to support this reasoning:

> "Thermal damage to the retina is to be expected for near infrared radiation since the photopigments are not involved and energy absorption takes place predominantly in the melanin granules of the pigment epithelium and the choroid. Photochemical effects are <u>not</u> to be expected at near infrared wavelengths because energy absorption involves vibrational quantum states which can readily dissipate their energy as heat to the aqueous environment without adversely affecting molecular structure. However, for shorter wavelengths in the visible spectrum electronic excitation becomes the dominant mode of energy absorption."

Lanum[19] also concludes that "in view of...experimental evidence that the thermal model does not adequately explain light damage, it is interesting to pursue the photochemical model as an alternative." Pointing to the fact that chemical reactions are generally facilitated by temperature increases, she adds, "...accelerating light damage by raising the body temperature of the animal need not imply that the damage is a direct result of heat transfer," and that "...it is impossible to eliminate the possibility that light and thermal damage are closely related [as there are] certainly some continuities between them."

Gladstone and Tasman[10] recognize this "nonthermal, possibly photochemical, lesion that occurs from a longer, lower intensity exposure." They present three cases in which patients were seen with a clinical diagnosis of solar retinitis, but without an initial compatible history. Evidence for a long-term low-intensity exposure to the sun was substantiated in two of the

cases, and the above theoretical considerations were used to help explain the mechanisms by which the patients were injured.

Many examples of retinal damage from extremely intense light sources as the sun and lasers can be cited. Damage, however, may result from longer exposure to a moderately intense source.

In 1965, Noell discovered that lights of moderate intensity could cause retinal damage in rats. Since that time, photoreceptor damage from moderate and even low levels of illumination has been well documented.(Noell, et. al.,1966; Gorn and Kuwabara, 1967; Kuwabara and Gorn, 1968; Friedman and Kuwabara, 1968; Grignolo, et. al., 1969; Marshall et. al., 1971; Marshall, et.al., 1972; Kuwabara and Okisaka, 1976)

Most studies of retinal light damage have employed a fluorescent illuminant because of the intensity of visible light which can be generated with very little heat output. Also, fluorescent sources, as compared to incandescent sources, are preferred because their spectrum more closely approximates daylight and the "natural" environment of an animal.

Many investigators have previously used, as the criterion for threshold retinal damage, the production of a retinal lesion visible by ophthalmoscopic examination. Investigations by more sensitive techniques have, however, demonstrated changes at levels far below these "thresholds." Two such techniques for retinal damage analysis are (1) histological studies utilizing

both light and electron microscopes, and (2) analysis of biological electrical phenomenon such as the electroretinogram.

The most detailed picture of light damage is presented by studies employing the electron microscope; it provides the greatest resolution and allows detection of damage from lower intensity lights. Lanum[19] considers four of the numerous studies completed in outlining a general picture of the changes noted.

Kuwabara and Gorn[17] exposed albino rats to relatively cold light of approximately 750 ft-c (1.2×10^{-3} w/cm^2) at room temperature for one hour to several weeks. O'Steen, Sheer, and Anderson[23] exposed albino rats to fluorescent illumination of 18 ft-c (2.9×10^{-5} w/cm^2) at room temperature (25°C.) for 4-6 months. Hansson[12] exposed male albino rats to "intense" visible light at an elevated body temperature (39°C) for two 30 minute periods separated by two hours of darkness. Lanum also cites the work of Grignolo, Orzalesi, Castellazzo, and Vittone, who "exposed albino rats to 1500-2000 lux ($2.3\text{-}3.0 \times 10^{-4}$ w/cm^2) with the temperature elevated about 10° above room temperature (39°C) for a period ranging from 8 hours to 100 hours."

Lanum's summary of research findings states:

> "Hansson noted that an increase occurs first in the space between Bruch's membrane and the pigment epithelium concommitant with structural changes in the mitochondria and endoplasmic reticulum of the epithelial cells. Noell, et. al. also noted histological damage to the pigment epithelium but found it to be simultaneous with receptor cell damage."

"The other studies found the first alterations occurring in the outer segments of the photoreceptor cells. The outer segments appeared swollen, the lamellar structure was disrupted with the disc membranes becoming thinner, showing separations, winding about, and forming tubules and vescicles. Pyknosis and swelling of mitochondria were then observed in the inner segment. Kuwabara and Gorn commented that with extremely bright light or high temperature, changes may occur in the cell body prior to the disruption of the outer segment. Hansson apparently had these conditions and noted inner segment changes in the microtubules and cross-striated filaments. However, even in his study, a few inner segments remained after the outer segments were broken and phagocytized."

"The rod outer segments continue to change, becoming large, round, or pear-shaped bodies. The microvilli of the pigment epithelium increase in length and number, and invade further into the outer segments. The outer segments break into smaller pieces and seem to be ingested by an increasing number of phagocytizing cells. Glial cells around the blood vessels have a number of lysosomes and contain cell debris. As the outer segments lose connections with the inner segments and begin to disappear, the inner segments become shrunken with pyknotic nuclei and interruptions of the limiting membrane. Finally, all the damaged photoreceptors disappear. The damage is usually more rapid and complete in the center of the retina. If the damage is not extensive, Muller cells may grow and close off the damaged from the undamaged portions of the retina."

"In the Hansson study and in the 80-100 hour exposed animals of Grignolo, et. al., all of the receptor cells and the pigment epithelium disappeared. The basement membrane remained intact and glial cells filled the space left by the missing cells. The bipolar layer was still intact."

"Kuwabara and Gorn, and O'Steen, et. al., showed an intact pigment epithelium with the receptor cells completely gone. When this happens the Muller cell and pigment epithelial cell microvilli elongate and interdigitate into a permanent adhesion. Cells of the inner nuclear layer, ganglion cells, and glia remain normal in appearance."

Messner, et. al,[21] and Cicerone[5] report that following continuous exposure to fluorescent light for extended periods of time, damage to rod photoreceptors far exceeds that to cones.

Cicerone [5] found that following exposure to 100 ft-c (1100lu/m^2) for a 12 to 24 hour period, the animal's dark adaptation curve was pushed to higher thresholds. The two branches, however, were not shifted equally; the rhodopsin rod branch was elevated by 1.31 log units, while the cone branch was elevated by only 0.2 log unit. Also, following this exposure, "the spectral sensitivities of the surviving mechanisms are consistent with single pigment Dartnall nomograms with peak sensitivities at wavelengths of 450, 520, and 560 nm." Cicerone also states that "LaVail (1976) provided anatomical evidence that cones survive rods after long exposure to constant light in the Fischer albino rat. The percentage of cones detected by light and electron microscopy increased from 1.5 percent of all photoreceptors in the rat to about 60 percent with very long exposure."[5]

Analysis of electroretinograms following exposure to light has also been utilized in assessing retinal damage. Reuter and Hobbelen[26] found that "continuous exposure to high levels of light results in a reduction of the a- and b-wave amplitudes, and an increase of the time to peak of the waves." Rapp and Williams[25], Gorn and Kuwabara (1967), Lawwill (1973), and Noell, et. al.[22], have noted the relationship between rhodopsin loss and the degree of ERG loss.

Rapp and Williams[25] note that "the log ERG (b-wave) sensitivity varied directly with the rhodopsin content of exposed retinas even as progressive deterioration was apparent." Gorn and Kuwabara (1967) "used broad spectrum blue, green and red lights of equal energy. The damage from the different

sources was not equal but instead related positively to the efficiency of each source in bleaching rhodopsin."[19] Lawwill (1973), however, found that a monochromatic source of 514 nm, which is only 30% more effective in bleaching rhodopsin than a control source, is five times more effective in reducing the rabbit ERG." Noell, et. al.,[22] found "the most effective energy in producing damage is the most effective at bleaching rhodopsin (about 500 nm)."

Noell, (1965) found that control rats,

> "...show reduced ERG's after a one hour exposure to a light of intensity great enough to reduce the retinal rhodopsin concentration 10 to 30% of the total present in the dark adapted state. The ERG reduction seems irreversible and histology reveals that the receptor cells disappear completely when the ERG is absent. Receptors show partial damage when the ERG is only reduced."[19]

Noell, et. al.,

> "...Followed with a comprehensive study in which the ERG was measured after placing normal albino rats in approximately 185 ft-c (3.3×10^{-3} w/cm^2) of green (490-580 nm) illumination for varying times at varying temperatures. When the animals were placed in the dark for ERG assessment a slowly increasing amplitude was seen for 3 hours followed by a decline for 24 hours. The authors interpreted the initial increase as possible recovery from light adaptation and the subsequent decrease as progressive deterioration in cell function. With few exceptions the ERG shows no recovery over 3 to 4 weeks from the level shown 24 hours after exposure. The reduction of the a-wave is a more consistent index than the b-wave. If the ERG is found to be normal at 24 hours, further reduction (to 10%) is sometimes seen in the next 7 days. If the reduction is less than 50% at 24 hours some recovery occurs during the second week. Histologically, the retinas appear normal if the ERG reduction is not greater than 50%."[19]

Gorn and Kuwabara (1967),

> "...placed animals at normal ambient temperature in 2000 ft-c (3.2×10^{-3} w/cm^2) or continuous fluorescent illumination. The first effects of a reduced ERG were seen after 2 to 4 hours of exposure. After two days the flashes eliciting the ERG had to be increased by 3 log units to reach the pre-exposure level threshold ERG's. After one week no ERG could be elicited. After two days, not only was the amplitude reduced, but the waveform was changed. The a-wave was gone while the b-wave was reduced and the latency of the b-wave was increased. The animals exposed to two days of illumination showed recovery if placed in the dark. After 15 days in the dark, there was greater recovery for threshold ERG's than for ERG's of maximum amplitude. Although the waveform returned to normal along with the b-wave latency, the maximum amplitude did not."[19]

Noell, et. al.[22] pronounced light damage "irreversible." This statement is disputed by studies using lower light levels, lower temperatures, or a longer follow-up period. Recovery from light damage has been shown histologically as well as with ERG's. It "seems to begin in the pigment epithelium and to proceed very slowly with the formation of new lamellar membranes in the outer segments. Though new discs are formed, their arrangement is often quite irregular. This irregularity does not seem to prevent a return to apparently normal electroretinogram response."[19] Rapp and Williams[25] found that "even seriously distorted rod outer segments are capable of initiating retinal signals as long as some rhodopsin is present within them.

Lanum[19] concludes that,

> ... "There is a point beyond which recovery seems impossible. Just how much damage must be sustained before it is irreversible is not clear. The receptor cell bodies must not be destroyed, and the less damage sustained by the pigment epithelium, the easier the recovery seems to be. The receptor cell is a neuronal cell and apparently it can recover from injury as long as the nucleus and cell body are viable."

Although retinal damage by light has been described in detail, we lack knowledge of the mechanism by which it is produced. Of the suggestions proposed by investigators, most were made originally by Noell, et. al.[22]:

> "First, the light could activate destructive oxidizing reactions. The vertebrate photoreceptor disc membranes are largely composed of highly unsaturated fatty acids which are subject to peroxidation. The absorption of light by the photopigment begins a series of reactions during which some free radicals are produced. These free radicals can initiate chain reactions, which, if unchecked by the presence of antioxidants such as vitamin E, could proceed to the peroxidation of the lipid membranes. Presumably in normal environmental conditions new disc membranes are produced as fast as peroxidation and the shedding of old discs."[19]

Delmelle[6] supports this theory, stating that bright illumination of the retina could lead to singlet oxygen production. This metastable species could lead to membrane damage involving oxidation of either the proteins or lipids, or both. He notes that, "inasmuch as lipids are concerned, singlet oxygen can induce their peroxidation."

Secondly, "prolonged light adaptation might lead to metabolic damage detrimental to the maintenance of cell life. Noell, et. al. think the time course of cell destruction is too rapid to give plausibility to this hypothesis over a metabolic poison."[19] As a result of his work, however, Hansson (1970, 1971) believes that "photoreceptor damage is a secondary destruction stemming from the failure of the pigment epithelium in its supportive, nutritive function."[19]

Noell, et.al.[22] also proposed a third possibility, which is that bleaching produces a potentially toxic photoproduct

against which the retina has little protection. In normal situations this product may be eliminated too rapidly to cause damage, but when continuous exposure results in accumulation, retinal integrity is compromised.

Gorn and Kuwabara (1967) have also proposed that "the reaction might be a direct one without necessary intermediate photoproducts. Simply the chronic bleaching of photopigments might subsequently impair the membranes of the outer segments.

Noell (1974) has more recently proposed a mechanism which incorporates his initial suggestion. "Adverse chemical reactions, such as lipid peroxidation, are initiated when the photopigments absorb light. Maintaining rhodopsin in a bleached state enhances these reactions which...may spread to the nuclear region and produce cell death."[19]

Research involving retinal light damage has also been related to the functional status acquired by the animals involved. Farrer, et. al.,[8] found that, at levels of energy density on the retina which are 80 to 50% below the threshold thermal burn level, no loss in visual acuity can be detected in the Rhesus monkey by Landolt ring testing.

Anderson and O'Steen (1972) exposed albino rats to 18 ft-c of continuous illumination for 30 days, the same conditions that they had used previously to produce receptor free eyes.

> "These animals, with no apparent outer segments correctly chose at a 90% criterion level the lighted end of a T-maze. They were also able to discriminate horizontal stripes versus vertical stripes subtending either 4^{o} or 2^{o} of visual angle. The latter is particularly amazing, for the limit of the normal rat's acuity is 1^{o} of visual angle. There were no differences either in performance or time to criterion whether the animals were trained before or after the continuous light exposure."[19]

Lanum, in reviewing these findings, comments:

> "Given the fairly uncontrolled situation of a T-maze where an animal can move about freely, it is possible that the rat was not actually resolving the lines but responding to some other cue instead. For instance, a rat moving his head up and down would get an alteration of brightness ("flicker") when facing horizontal stripes that would not occur with vertical stripes. It would certainly not be the first time an animal arrived at a correct response by a method different from that expected by the experimentor."[19]

Lai, et. al.,[18] studied age-related and light-associated retinal changes in rats. The number of photoreceptors gradually decreased in control rats as the animals aged. This reduction occurred uniformly from the ora serrata to the posterior pole except in two cases in which loss was more severe in the posterior pole. The inner and outer segments of remaining photoreceptors in affected retinas remained histologically normal. Also, a selective degeneration of the peripheral retina was discovered in aged rats. The incidence and severity of this peripheral degeneration increased with age and prolonged exposure to comparatively high-intensity light. Such results indicated that the peripheral degeneration was a age-related change which appeared to be exaggerated by ambient light. It is noted that "peripheral retinal degeneration is a common disorder of man," and that "...degeneration in aged Fischer rats mimics the human disease histologically and is therefore a potentially important animal model for peripheral retinal degeneration in man."[18]

Newborn infants suffering from hyperbilirubinemia (neonatal jaundice) are placed in fluorescent blue light for continuous periods of from one to six days. Photo-oxidation of bilirubin in the skin and subcutaneous tissue results in the formation of chemicals which are not toxic to brain tissue. Due to possible ocular damage, it has become common practice to protect the infant's eyes during treatment.

Sisson, et. al. (1970) used newborn piglets to investigate the potential retino-toxic effect of such phototherapy. They observed that "exposure to the blue light for as short a time as twelve hours produced irreversible changes in the outer retinal layers."[24]

Lanum.[19] notes two studies involving children exposed to phototherapy as infants:

> "The first study was done on a group of 4-year olds who had been exposed to six days of 90 ft-c illumination and the second on 4-year-olds exposed to 42 hours of 500 ft-c illumination while the eyes were covered with gauze pads. No decrement in scotopic functioning was found in either group of children when they were tested with computer-averaged electrograms. No noticeable acuity problems were present so cone function was also apparently in normal ranges."
>
> "The researchers were not, however, satisfied that they had eliminated the possibility of cone damage or of damage to small areas of the retina. They note that possibly there could have been mild damage but that recovery could have occurred between the therapy as infants and the time of testing four years later."

Messner, et. al.[21] cite the work of Kuwabara and Okisaka, who suggested that ,

> "...the excess of rod and cone elements compared to optic nerve fibers in primates provides a safety margin for the anticipated senescent changes in the outer nuclear layer of the retina. Thus, although a considerable number of rods and cones degenerate, enough intact cells remain to allow for apparently normal visual function. Without histologic examination, a significant loss of outer cellular elements might remain undetected. Therefore visual acuity and ophthalmic examination in a photic damaged human infant may be normal in childhood although considerable tissue has been lost."

They suggested that because both aging and photic trauma produce loss of rods and cones, the effects of phototoxicity may be additive to the normal aging of the retina. This effect may become clinically evident many years later, when the cellular attrition due to aging is combined with the early photic cell loss, to result in visual compromise.

Retinal pathologies may be affected by the light environment. Dowling (1962,1964) has shown that "in rats with inherited retinal dystrophy, some retinal deterioration continues even with complete light exclusion." He has demonstrated, however, that "functioning can be preserved longer by keeping the animals in the dark, and the damage is correspondingly accelerated by light."[19] Charles and Machemer (1974) found that "darkness has a protective effect on detached photoreceptors and that light exposure plays a significant role in the detachment-induced atrophy of photoreceptors."[19]

The ultimate question we must ask is, "how much light causes damage?" As one considers the numerous factors involved, it becomes evident that this question has no simple answer.

Investigation of thresholds for purely thermal lesions, or "actinic retinitis", appear to be straight-forward, and indeed, given certain circumstances and damage criteria, a minimum value can be calculated. Using a carbon arc U.S. Army searchlight, DeMott, et. al.[7] concluded that "at irradiance levels greater than 2 cal/cm^2/sec, the radiant exposure necessary to produce an ophthalmoscopically visible lesion... lies between 0.5 and 1.5 cal/cm^2. Farrer, et. al.[8] found the threshold as determined by lesions in the paramacular and equatorial regions of the fundus was 6.67 $\pm$ 1.06 J/cm^2 (1.60 $\pm$ 0.25 cal/cm^2). The generally accepted value for a threshold thermal lesion of this type is 1.0 cal/cm^2 incident on the retina, or 0.01 cal/cm^2 incident on the cornea.[16]

Bartleson[2] points out that the threshold exposure which will produce a chorioretinal thermal lesion is a function of six factors: (1) the rate of energy delivery to the eye, (2) the duration of exposure, (3) the size of the retinal image, (4) the shape of the retinal image, (5) the transmittance of the ocular structures, and (6) the degree of fundal pigmentation. One must consider these variables in any discussion of threshold determinations for thermal lesions; because of these variables, "there is ... no single value of radiant exposure that can be named as a threshold."

For lesions of other than purely thermal nature, many more considerations must be recognized. Sliney[29] points out that at retinal irradiance below approximately 0.1 W/cm^2 thermal injury should not take place since the calculated temperature elevation

in the retina is less than $1^{o}C$. He notes, though, that light induced retinal injury has been reported in experimental animals at exposure levels of a factor of 10 to 1000 times below any thermal levels of injury. Harwerth and Sperling (1975), Ham and colleagues (1976) and Lawwill (1973) have shown that continuous exposure to blue light at levels as low as 10^{-4} W/cm^2 can cause permanent retinal changes.

As discussed previously, exposure to light may cause retinal changes in the absence of visible lesions. Factors such as duration, spectral distribution, and intensity of the source must be considered in addition to numerous physiological variables of the animal. We are uncertain as to what kind of retinal changes indicate "deficits in visual function sufficient to produce a degradation in visually guided behavior."[11] In addition, as we are unsure of the mechanisms by which such retinal damage is produced, we cannot determine what variables are critical in its production. In short, we have not determined what significant retinal changes are, much less the factors we must specify to avoid their occurrance.

At this point in time, we must rely on the appearance of visible lesions, and/or obvious decrements in visual function such as decreased visual acuity, color vision loss, or visual field loss, to specify retinal damage. In taking this approach we should be cognizant of the fact that, in many circumstances, these thresholds may be specified as higher than they should be; very little light may be needed to affect such changes as those degenerations which occur with age.

Clinical Application

As clinicians, we must be concerned with the tolerance of the human retina to light exposure, particularly as it relates to the use of ophthalmic instruments.

Lanum[19] notes that the indirect ophthalmoscope is "a special case of an incandescent illuminant." Dawson and Herron (1970) found that this instrument "has 90% of its power in the infrared region beyond 775 nm."[19]

Friedman and Kuwabara (1968), Ts'o, et. al. (1972), and Ts'o (1973) have produced damage to monkey eyes with the indirect ophthalmoscope. Damage,

> "...was produced after 15 minutes of exposure calculated to be 0.27 w/cm^2...The eye was also paralyzed so the light was constantly on the same point of the retinal surface. It is interesting to note that the lesions produced...were not ophthalmoscopically visible for 24 hours to several days. The severity of the lesion depended on the body temperature of the animal, but it was not possible to lower the body temperature of the animal to the point that no damage occurred with a 1-hour exposure."
>
> "The typical changes observed in the receptor layers with light damage were present and the pigment epithelium was also involved...Ts'o and associates allowed some animals to live for several months and were able to demonstrate recovery."[19]

Calkins, et. al.[3] calculated the retinal irradiance likely to be received by a patient from some commonly employed operation microscopes. This irradiance varies from 0.10 to 0.97 w/cm^2 for emmetropic and myopic patients, and from 0.085 to 0.59 w/cm^2 for aphakic patients. (For high hyperopic and aphakic patients, the pupil does not act as the limiting stop in the system.) A calculated "safe time" indicates how long exposure at this level of irradiance must be maintained before the ANSI laser safety

guidelines are exceeded. The retinal maximal permissible exposure (MPE) of 2.92 joules/cm^2 is reached from 1.8 to 29 sec. for emmetropes and myopes, and from 3.7 to 49 s for aphakic patients.

Also, they calculated that ophthalmoscopically visible changes might occur in 50% of subjects exposed to such a source after a range of five to 49 minutes of continuous exposure. For aphakic patients in the same conditions, one may expect to see changes after 8.3 to 82 minutes of exposure.

Calkins and associates find these calculations disturbing when "one considers that ophthalmoscopically visible retinal lesions have been produced in monkey eyes by white light sources with roughly the same light exposures that the patient may, under certain conditions, receive from an operation microscope." They also acknowledge the fact that "...there is good reason to suspect that light sources (coherent or noncoherent) that emit more heavily toward the blue end may have a higher probability of producing retinal changes than even laser safety standards would suggest."[3]

The question has been raised as to why retinal damage is not seen clinically if such light sources are actually unsafe. Calkins, et. al.[3] suggest that it may be seen as cystoid maculopathy (Irvine-Gass syndrome) and accelerated macular degeneration following surgery. With knowledge of the amount of light that a patient may be exposed to during surgery, they conclude that if light exposure does not result in cystoid maculopathy, it must be causing some other frequently encountered retinal condition.

> "Since 40% to 50% of routine cataract extractions are followed by cystoid maculopathy, which does not seem to correlate consistently with vitreous traction, age, sex, complications, medications, or hypotony, a correlation with light exposure appears to be a fruitful area for further investigation."[3]

Revision of the illumination systems of operating microscopes is urged. With knowledge of research findings no one would purposely illuminate a normal retina continuously with an indirect ophthalmoscope for 15 minutes, yet it produces more than five times less retinal irradiance than the average microscope. "The same level reached by an average indirect ophthalmoscope on a medium voltage setting in 15 minutes (61 joules/cm^2) is reached by [an] operation microscope... after only one minute (worst case conditions are assumed for both instruments)."[3]

Henry, et. al.[14] have also suggested that light exposure may be related to chronic cystic macular edema. After noticing this condition as a more frequent complication of eye surgery, these physicians reviewed changes in their surgical procedure during the associated time period. They noted an increase in the operating time due to a procedural change, and the installation of two new operating lights which delivered 2000 ft-c of light each, and were focused on the patient's eye from above, to be almost in line with the patient's visual axis. In view of the fact that photic maculopathy has been well documented in animals from amounts far less than used in surgery, they conclude that there is "every reason to assume light focused on the human eye during eye surgery while the natural defense mechanisms are

in abeyance causes stress on the delicate retina and creates or hastens macular disease processes."[14] They recommended using light directed on the eye at an angle, with only as much illumination as is necessary, in addition to covering the cornea with an opaque shield, except during the lens extraction.

Robertson and Erickson[27] studied the effect of prolonged indirect ophthalmoscopy on human eyes which were either totally blind, or contained a tumor requiring enucleation. The corneas were anesthetized. Using a setting of 7.5 v., light was focused on the retina through a condensing lens for a total duration of 45 minutes, with rest periods of 30 seconds every five minutes. Radiometric and photometric measurements were 0.42 W and 11,000 mL, respectively, of light incident on the cornea.

They found that,

> "Concentrated prolonged light exposure...caused grossly visible progressive corneal edema along with significant discomfort. Even with light exposures sufficient to cause these complications of the anterior segment, retinal injury was recognizable only with electron microscopy and was characterized by irregular bending and twisting of the outer segments of the photoreceptors, changes that are considered reversible in nonhuman primate studies."[27]

Measurement of Light Output from Ophthalmic Instruments

Measurements were made to determine the light output of various ophthalmic instruments. Inquestioning whether ocular damage may result from routine examination procedures, the output of individual instruments and the retinal illumination produced by them should be compared to levels previously shown to cause retinal damage.

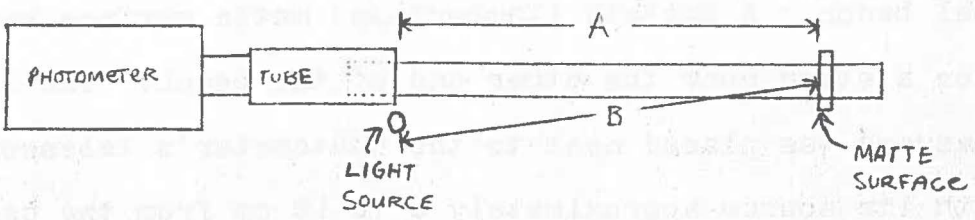
The following method was utilized to ascertain the output of direct, monocular indirect, and binocular indirect ophthalmoscopes, and of retinoscopes. The instrument used was the Gamma-Scientific Auto Photometer. (See Fig. 1)

The photometer unit was placed on a counter at the end of an optical bench. A MacBeth (Lambertian) matte surface was secured on a stand near the other end of the bench. The light to be measured was placed next to the photometer's telescopic tube, with its source approximately 8 to 10 cm from the center of the tube. The binocular indirect source and end of the tube were 85 cm from the matte surface; all other light sources were 75 cm from the surface (as was the tube entrance in these cases).

In measuring the luminance of biomicroscope sources, a method was employed by which the luminance of the source was acquired directly. A thin sheet of white paper was mounted in a cardboard frame and placed in the face plane of the biomicroscope. A narrow slit of light was focused on the paper; it was also visible from the opposite side. The photometer was then

focused on this plane, the paper was removed, and the beam extended to a full diameter circle. Because of the source intensity measured in this way, a 3.0 and a 4.0 Neutral Density Kodak Wratten filter were required at the end of the tube to obtain readings.

Fig. 1



A and B are specified distances

Measurements

Direct Ophthalmoscopes
(Welch Allyn)
@75 cm.

1. 1.85 fL
2. 1.58
3. 1.73
4. 2.02
5. 1.72
6. 1.70
7. 1.05
8. 1.80
9. 1.52
10. 1.48
11. 2.12

High: 2.12 fL
Avg.: 1.857 fL

Monocular Indirect Ophthalmoscopes
(American Optical)
@75 cm.

1. 1.56 fL

2. 2.5v 0.08
   6.0H 0.62
   6.5v 0.91
   7.5v 1.46

3. 1.71

Binocular Indirect Ophthalmoscopes (@85 cm.)

1. B & L / Frigitronics

#1	Low	0.102 fL	#2	L	1.49	#3	L 15.2
	High	1.15		H	12.8		H 127.0

2. Keeler

   Max. 102.1 fL

3. Medical Instrument Research Assoc. (Surgical)

   Max. 20.5

4. B & L

#1	Low	0.34 fL	#2	L	5.5	#3	L 92.3
	High	2.23		H	16.8		H 196

5. B & L

#1	Low	0.36 fL	#2	L	4.0	#3	L 35.0
	High	3.3		H	29.8		H 194

Retinoscopes (@ 75 cm.)

Welch Allyn

1. 0.75 fL
2. 1.39
3. 1.12
4. 0.98
5. 1.53
6. 1.24
7. 1.31

High: 1.53 fL
Avg.: 1.19 fL

Copeland

1. 0.94 fL
2. 0.52
3. 0.46

Biomicroscopes

1. Haag-Streit
   a. 5v 0.33 fL
   b. 6v 0.53 fL
   c. 7.5v 0.91 fL
2. Marco IIB
   Max. 0.1 fL
3. Marco IIB
   Max. 0.31 fL
4. American Optical
   Max. 0.07 fL
5. Nikkon Photo-Zoom
   Max. 0.08 fL

In order to determine the luminance of the source, one must work backwards from the photometric measurement which is of the luminance of the matte surface. As an example, consider the average measurement for the direct ophthalmoscope:

The average luminance of the MacBeth surface is 1.857 fL.

$$fL = \frac{1}{\pi}\ ^{cd}/_{ft^2} \quad \therefore \pi fL = 1\ ^{cd}/_{ft^2}$$

$$1.857\pi = 5.83\ ^{cd}/_{ft^2}$$

Because $10.8\ ft^2 = 1\ m^2$

$$L_{(matte)} = \frac{5.83\ cd}{ft^2} \times \frac{10.8\ ft^2}{m^2} = 63.01\ ^{cd}/_{m^2}$$

Luminous Emittance (M) $= \pi L$

$$63.01\pi = 197.94\ ^{lm}/_{m^2} = M$$

Luminous Emittance (M) equals Illumination (E) onto the surface, but the reflection factor for the test plate is 0.73 if viewed at an angle of 25° or less. (This procedure utilized an angle of Approximately 7°)

$$\frac{197.94}{0.73} = 271.15\ ^{lm}/_{m^2} = E$$

Luminous Intensity, $I = Ed^2$

$$d = 0.75m$$

$$\therefore I = (271.15\ ^{lm}/_{m^2})(0.75m)^2 = 152.52\ lm$$

THE FILAMENT AREA OF THIS SOURCE IS 0.0027 cm^2

$$\therefore L = \frac{152.52}{0.0027} = 56,489.96 \text{ cd/cm}^2 \cong 56,490 \text{ cd/cm}^2$$

RETINAL ILLUMINATION (E_R)

$$E_R = 0.36\, L \cdot A_{p(cm)} \cdot T_\lambda$$

FOR AN 8mm pupil, $r = 0.4$ cm

$\pi r^2 = 0.50265 \cong .503 cm^2$

$$E_R = (0.36)(56,490)(0.503)(0.5)$$

$$E_R = 5114.6 \text{ lm/cm}^2$$

TO SIMPLIFY THE ABOVE INTO SINGLE EQUATIONS:

$$\frac{([x]fL)(\pi^2)(10.8)(0.75)^2}{0.73} = I$$

$$I = ([x]fL)(82.13403) \cong ([x]fL)(82)$$

$$L = I / \text{FILAMENT AREA}$$

$$E_R = (L)(0.090477) \cong (L)(0.09)$$

Direct Ophthalmoscopes

1. Average: 1.857 fL

   $L = 56{,}490 \text{ cd/cm}^2$

   $E_R = 5114.6 \text{ lm/cm}^2$

2. Maximum: 2.12 fL

   $L = 64{,}385 \text{ cd/cm}^2$

   $E_R = 5795 \text{ lm/cm}^2$

Monocular Indirect Ophthalmoscopes

(Filament area = 0.0102 cm^2)

1. Maximum: 1.71 fL

   $L = 13{,}747 \text{ cd/cm}^2$

   $E_R = 1237 \text{ lm/cm}^2$

Binocular Indirect Ophthalmoscopes

(d = 85 cm. $\therefore$ [x] fL)(105.5) = I

1. B & L/Frigitronics (filament area = 0.02 cm^2)

   Average high measurement: 172.3 fL

   $L = 908{,}882.5 \text{ cd/cm}^2$

   $E_R = 81{,}799 \text{ lm/cm}^2$

2. B & L/Frigitronics

   Maximum: 196 fL

   $L = 1{,}033{,}900 \text{ cd/cm}^2$

   $E_R = 93{,}051 \text{ lm/cm}^2$

3. Keeler

   Measurement: 102.1 fL

   $L = 538{,}577.5 \text{ cd/cm}^2$

   $E_R = 48{,}472 \text{ lm/cm}^2$

Retinoscopes

(d = 75 cm.; filament area = 0.01 cm^2)

1. Average Welch Allyn: 1.19 fL

   $L = 9758\ cd/cm^2$

   $E_R = 878.2\ lm/cm^2$

2. Maximum: 1.53 fL

   $L = 12,546\ cd/cm^2$

   $E_R = 1129\ lm/cm^2$

An attempt was made to determine the actual density of the 3.0 + 4.0 neutral density filters. Due to experimental error, this data was not acquired. Calculations must then address a range of variability in the effective density of the filters. Thus, computations are performed for 6.5 and 7.5 neutral density filters in addition to a "pure" 7.0 filter.

Biomicroscopes

1. 0.33 fL

   a. (7.0 ND) $L = 1.1 \times 10^4\ cd/cm^2$

      $E_R = 1013.7\ lm/cm^2$

   b. (6.5 ND) $L = 3.5 \times 10^3\ cd/cm^2$

      $E_R = 320\ lm/cm^2$

   c. (7.5 ND) $L = 3.5 \times 10^4\ cd/cm^2$

      $E_R = 3206\ lm/cm^2$

2. 0.53 fL

   a. (7.0 ND) $L = 1.8 \times 10^4\ cd/cm^2$

      $E_R = 1628\ lm/cm^2$

   b. (6.5 ND) $L = 5687\ cd/cm^2$

      $E_R = 515\ lm/cm^2$

   c. (7.5 ND) $L = 5.7 \times 10^4\ cd/cm^2$

      $E_R = 5149\ lm/cm^2$

3. 0.91 fL

   a. (7.0 ND) $L = 3.1 \times 10^4$ cd/cm^2
   $E_R = 2795$ lm/cm^2

   b. (6.5 ND) $L = 9763$ cd/cm^2
   $E_R = 884$ lm/cm^2

   c. (7.5 ND) $L = 9.8 \times 10^4$ cd/cm^2 (Maximum found)
   $E_R = 8840$ lm/cm^2

4. 0.1 fL

   a. $L = 3393$ cd/cm^2
   $E_R = 307.2$ lm/cm^2

   b. $L = 1073$ cd/cm^2
   $E_R = 97$ lm/cm^2

   c. $L = 10{,}729$ cd/cm^2
   $E_R = 971$ lm/cm^2

5. 0.07 fL

   a. $L = 2375$ cd/cm^2
   $E_R = 215$ lm/cm^2

   b. $L = 751$ cd/cm^2
   $E_R = 68$ lm/cm^2

   c. $L = 7511$ cd/cm^2
   $E_R = 680$ lm/cm^2

6. 0.31 fL

   a. $L = 1.1 \times 10^4$ cd/cm^2
   $E_R = 952$ lm/cm^2

   b. $L = 3326$ cd/cm^2
   $E_R = 301$ lm/cm^2

   c. $L = 3.3 \times 10^4$ cd/cm^2
   $E_R = 3011$ lm/cm^2

Conclusions

Robertson and Erickson[27] measured the luminance of a binocular indirect ophthalmoscope to be 11,000 mL. The maximum obtained for such an instrument in this experiment was 1.0×10 cd/cm^2, or 328 mL. (1 mL = 3.18×10^{-4} cd/cm^2) This finding is approximately two log units less than that obtained by researchers whose source was found to produce reversible retinal changes following its prolonged use.

The maximum luminance found for a biomicroscope source, which was the most intense of all instruments measured in this experiment, was 9.8×10^4 cd/cm^2, or 31 ml. This measurement is approximately 3 log units below that found by Robertson and Erickson.

The maximum luminance found for a direct ophthalmoscope was 64,385 cd/cm^2, or 20 mL, which is again three log units below the above finding.

Data acquired from various manufacturers shows findings that are generally less than those arrived at in this experiment, sometimes by as much as almost two log units. These findings, however, were derived through different experimental procedures. It should also be noted that for the same instrument, a particular direct ophthalmoscope, the research departments of two different manufacturers acquired measurements varying by one log unit, apparently utilizing the same procedure and conditions.

Certainly, comparison of experimental findings to a limited number of sources, and to measurement acquired by different

means is inadequate. However, in order to investigate the most relevant aspects of a light source, measurements should not be made with only a photometer; a radiometer should be utilized to ascertain the total energy output of the source. As has been discussed, retinal damage is not a function of how bright the source appears to be, but of the total energy output, with consideration of its spectral distribution.

Ideally, the relative contribution of all wavelengths should be evaluated with consideration of their additive effects. The amount of short wavelength and long wavelength irradiation have been shown to result in different types of changes.

As a result of this experiment, "ball-park" measurements of source luminance and resulting retinal illumination for "worst case" conditions have been calculated for each instrument. Based on these findings, and comparison of clinical examination conditions to those utilized by researchers who have produced retinal damage, we can fairly safely conclude that no significant retinal damage is expected to result from routine examination procedures. Prolonged use of the direct ophthalmoscope, even for the extended periods of time often encountered by students when first learning the technique, is probably not damaging to a healthy subject. The source is generally not focused on the same retinal area for a long enough time to create cumulative effects.

Because questionable findings have resulted from prolonged use of such intense light sources, and because their effect on other than healthy retinas has not been clearly established,

it may be wise to limit their use in certain instances. Undoubtedly, the potential benefits obtained by the use of these examination procedures far outweigh the possible dangers. However, extremely prolonged use of such intense sources should be evaluated for use on certain patients with pathologies such as retinal detachments, retinitis pigmentosa, and possibly some other degenerative conditions. Such guidelines may rarely be pertinent in private or small group practices, but their application should be given some consideration in educational institutions, where the potential for unreasonably prolonged internal examinations is much more frequent.

In summary, surgical microscopes utilize the most intense light sources of all ophthalmic instruments. The literature presents convincing studies which implicate the use of intense light during surgical procedures in the production of cystoid macular edema. Unless proven otherwise, suggested procedures designed to reduce such light exposure should be adopted, as they are apparently well founded. Other less intense sources have not been implicated in the production of irreversible retinal damage, however, their use for extremely prolonged periods of time may be discouraged in certain cases of retinal pathologies.

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