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Effects of Laser Radiation on the Mammalian Eye*

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Historical Introduction

The effects of visible electromagnetic radiation on the eye can be traced back, at least, to Galen (130 - 200 A.D.); he seems to have been the first to describe "eclipse blindness". (1) Galileo is known to have injured his eyes by observing the sun through his famous telescope. (2) It was not until 1916, however, that Verhoeff and Bell (3) described scientifically the phenomenon of eclipse blindness in terms of thermal injury to the pigment epithelium (PE) and choroid as a result of the sun's image focussed on the retina. In this classic paper it was pointed out that these retinal lesions were, in fact, "burns" and not caused by some esoteric action of ultraviolet light or by excessive stimulation of the photochemical systems responsible for vision. Eclipse burns were irreversible lesions not to be confused with the reversible phenomenon of flashblindness. In the Mediterranean and Pacific theatres during World War II, observers, using optical instruments to spot planes attacking from the direction of the sun, also experienced retinal burns. (4)

The development of nuclear weapons at the close of the war introduced another retinal hazard. Buettner and Rose, (5) in 1953, were the first to emphasize the vulnerability of the eye to nuclear detonations within the earth's atmosphere, pointing out that the optical properties of the eye compensated for the "inverse square law" of attenuation out to distances where eye resolution failed.

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In 1954 rabbits exposed at 42.5 miles from a nuclear explosion received mild retinal lesions; [6] later, in 1958, rabbits exposed to high altitude nuclear explosions over Johnson Island received retinal lesions out to distances of 300 nautical miles from the explosion. [7] Several cases of retinal damage in humans exposed to nuclear weapons are on record.

Meanwhile, during the early part of 1950, Meyer-Schwickerath [8] began experimenting with light coagulation as a clinical technique for treating retinal detachment and certain types of intraocular tumors; in his early experiments the sun was used as the source of radiation; later, in collaboration with Littmann, [9] a light coagulator was developed in Germany and used successfully on patients. The first clinical use of light coagulation of the retina reported in this country was by Guerry [10] in 1958. Light coagulation as a method of treating certain ocular pathologies is used now extensively in many clinics both in this country and abroad.

The recent advances in research and development of optical masers and lasers have produced a new and novel research tool in biology and medicine as well as another potential hazard for the eye. Gordon, Zeiger, and Townes [11] of Columbia University were the first to obtain a successful optical maser in 1954. Maiman [12] succeeded in developing the ruby solid state laser in 1959. Remarkable technological advances have been made in the past few years; today, the generation of coherent electromagnetic radiation by stimulated emission extends spectrally from the ultraviolet to the far infrared, involves gaseous, solid state, and liquid lasers, covers a power range from milliwatts to gigawatts and provides intense radiation beams with unique properties of directionality, coherency, monochromaticity, and polarization. Lasers may be operated to produce continuous radiation (CW) or pulses ranging from a few nanoseconds (ns) to

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several milliseconds (ms). Their widespread use [13] in industry, in the military, and in medicine involves applications as diverse as communications, range finding, and light coagulation of the retina.

Properties of the Eye

Lasers emit highly directional, spacially confined, beams of radiation; angular divergences range from fractions of a milliradian to about 10 milliradians; the diameter of the beam at the source is generally confined to less than 2 cm. In the near field, the entire cone of laser radiation, depending upon beam diameter relative to pupillary diameter, may enter the eye; here, the diameter of the image focussed on the retina will depend on the product of the angle of divergence and the equivalent focal lenght of the human eye, taken as approximately 17 mm. For angles of divergence exceeding 1 milliradian the image diameter is likely to exceed the diffraction limit for the human eye. There is conflicting opinion as to what constitutes the minimum image diameter under diffraction limited conditions; most authorities estimate it to lie between 10 and 20 microns (μ).[14] At large distances from a laser source (so-called far field) the cone of radiation bathes the entire eye but the beam entering the eye is almost parallel. This means that the image diameter on the retina will be diffraction limited (10 - 20μ). Thus, unless optical instrumentation is employed, laser beams impinging directly on the eye produce small images on the human retina, varying in diameter from about 200μ down to the diffraction limit. [15] It is interesting to note that the diameter of the sun's image on the human retina is approximately 160μ.

The transmission of light through the human ocular media (OM) as a function of wavelength has been measured by Geeraets; [16], [17] it extends from approximately 400 to 1400 mμ, exceeding 0.80 in the range 500 to 950 mμ.

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There are two peaks in the near IR, a large one at 1100 mμ and a much smaller one (0.10) at 1300 mμ. As might be expected the transmission of the OM is similar to that of water.

A negligible portion of the radiation striking the retina is absorbed by the photopigments in the inner layers of the retina as compared to that absorbed in the PE and in the choroid. Absorption in these elements has been measured for 24 human eyes by Geeraets. [17] Curves showing absorption vs wavelength in the most lightly and in the most heavily pigmented human eyes measured in this group have been plotted for both the PE and the choroid for light incident on the cornea. These curves give what Bredemeyer [18] has called the "spectral effectiveness factor"; exposure parameters being equal, it is possible to compare the effectiveness of various laser wavelengths in terms of transmission and absorption within the human eye. For example, maximum and minimum absorption in the PE at the ruby wavelength (694.3 mμ) is 0.40 and 0.15, whereas for neodymium doped glass (1060 mμ) the coefficients are respectively 0.15 and 0.05. Peak absorption in the PE for light incident on the cornea occurs between 500 and 550 mμ. Thus, the first harmonic of neodymium doped glass (530 mμ) and the green lines in the argon CW laser are in the spectral region where maximum absorption by the PE occurs. Actually, for light incident on the cornea, the absorption in the PE and in the choroid was found to be approximately equal in the 24 eyes studied. However, the PE has a thickness of roughly 10μ as compared to a choroidal thickness of about 100μ, so that the energy absorbed per unit volume (J/cm^3) in the PE may exceed that absorbed in the choroid by as much as a factor of 10. It is not surprising, therefore, that for mild lesions of the retina produced by radiation pulses of relatively short time duration (0.2 ms - 2.0 ms) damage is observed histologically to occur in the PE and in the strata of the retina immediately adjacent to the PE rather than in the choroid. [19]

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Contributing factors to this histological observation are that the temperature gradients favor heat flow towards the inner layers of the retina rather than towards the choroid and that the delicate structure of the stratified retina is presumed to be more sensitive to thermal damage than the vascularized choroid.

Experimental Production of Mild Lesions

The production of mild lesions in the rabbit retina by a pulsed ruby laser beam and by beams of white light has been studied as a function of average irradiance and exposure time. [20] Mild lesions are defined as being just visible ophthalmoscopically within 5 minutes after exposure and are based on probit analysis of RD-50 data as previously described. [21] These lesions are not considered to be threshold burns since irreversible damage has been demonstrated at lower levels of irradiance by more refined techniques such as histochemistry, electroretinography, and electron microscopy. [19, 22] Also, it is well known that irreversible lesions appear at a lower irradiance when the observation time post exposure is extended to several days. Some recent data are shown in Figure I where average irradiance or power density in watts/cm^2 vs exposure time in seconds is plotted logarithmically for mild lesions produced by a cone of radiation having a diameter of 800μ on the rabbit retina. For exposure times between 175μs and 1.2 ms the white light source is an electronically pulsed xenon lamp previously described; [23] white light exposures longer than 2 ms are obtained by operating the xenon lamp continuously (CW) with a filter in the beam to remove wavelengths greater than 900 mμ and using a mechanical shutter to control exposure times. Ruby laser exposures, both multiple spiked and Q-switched, are produced by an improved version of the apparatus previously described by the authors. [24] All laser exposures have also a cone of radiation of 800μ diameter on the retina. Laser exposures are indicated by crosses to distinguish them from the white light exposures.

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Mild lesions resulting from exposures to ruby multiple spikes (200 - 300μs) and to the pulsed xenon lamp (175μs) require approximately the same average irradiance (2 - 3 KW/cm^2) and are histologically similar. On the other hand 30 ns pulses of ruby radiation with average irradiances between 2 - 3 MW/cm^2 result in mild lesions which are different in histological appearance [19] and cannot be explained solely in terms of thermal injury. No data on the dotted region between 30 ns and 175 μs are currently available though this region is now under investigation using a Kerr cell as a gating mechanism. For long exposure times the curve tends to become horizontal to the x-axis, thus defining for a given image size a limiting retinal irradiance for mild lesions. This implies that the maintenance of retinal tissue at some temperature above ambient for long periods of time does not produce visible tissue coagulation. The basic problem for CW exposures is to determine what constitutes a safe temperature for retinal tissue.

Thermal Concept of Retinal Injury

The biological effects of laser radiation can be investigated in terms of thermal injury for retinal irradiances below the threshold for non-linear effects and for exposure times of sufficient duration to allow the application of the classical laws of heat conduction. This will include the CW operation of lasers and most multiple spike operations where the peak irradiance of individual spikes is not high enough to introduce non-linear effects.

Any attempt to correlate retinal burn data with a theory of thermal injury based upon chemical kinetics and critical temperatures for protein denaturation must begin with a knowledge of temperature-time history in the retina. Vos [25] [26] and others [27] [28] have treated theoretically the problem of heat flow in the retina. Vos calculates the temperature at the center of the PE assuming that all of the energy is absorbed uniformly throughout the PE and the choroid, taken as 10μ and

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100μ in thickness respectively; he considers a square image of either 10 or 100μ in thickness immersed in an infinite medium of water. His results are shown in Figure II as a log-log plot of retinal temperature rise per unit of incident radiation vs exposure time in seconds for several different image sizes. It is apparent that temperature rise and the time taken to approach a steady state temperature are important functions of the image size. These curves include contributions to the temperature of the PE by heat flow from the choroid; however, the choroidal contribution is minimal for exposure times less than approximately 1 ms. This is because the heat front takes an appreciable time t to propagate a distance Z; as shown by Vos, $t = Z^2/4K$ for the heat front to reach full effectiveness, where K is the thermal diffusivity. Taking $K = 1.5 \times 10^{-3}$ cm^2/sec for water as a first approximation to retinal tissue, Z is approximately 25μ for an exposure time of 1 ms.

Since most laser pulses rarely exceed 1 - 2 ms, it follows that only the PE will be involved for mild lesions. Some appreciation for geometrical proportions can be gained from Figure III which is a schematic diagram illustrating the ratio of image diameter to thickness for exposure times less than 1 ms and greater than 10 ms. Image diameters ranging from 200 - 20μ are chosen as illustrative of predicted laser beam images on the human retina for near and far field conditions. Short exposure times correspond to a thin disc as illustrated in the lower left of Figure III; radial heat flow will be negligible except for very small images. Experimental support for this model comes from histochemical evidence illustrating minimal radial spread beyond the image diameter for exposure times less than 2 ms, whereas the longer exposures (> 20 ms) demonstrate a marked spread of biological damage beyond the confines of the original image. [22] Equally convincing are the negligible

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differences in the irradiance needed to produce mild lesions with image diameters of 1000, 800, and 500μ as shown in a previous study [24] and the histological appearance of the lesions which are restricted to the PE and the inner layers of the retina. [19]

For exposure times greater than approximately 10 ms there is time for heat flow from the choroid to make appreciable contributions to the PE. Histological evidence confirms the involvement of the choroid as well as the PE for mild lesions produced by long exposure times. The model under these circumstances is illustrated on the lower right hand side of Figure III. Image diameter and absorbing thickness (PE + choroid) are now comparable and radial heat flow becomes an important factor in dissipating heat which would otherwise reach the PE. For image diameters approaching the diffraction limit (10 - 20μ), radial dissipation becomes so great that the choroid contributes a negligible amount of heat to the PE. Thus, for small image diameters the PE rapidly approaches steady state temperature equilibrium as illustrated by Vos' curves in Figure II. Since these curves are given as a log-log plot the temperature difference at the center of the PE for steady state equilibrium can differ by a factor of 10 or more for image sizes of 20 and 200μ. The influence of image size on the maximum temperature attained in the PE provides a safety factor for direct exposure of the eye to the sun and to CW lasers with small angles of divergence. Blood flow in the choroid is another factor which protects the retina from excessive temperatures. Geeraets has shown that for long exposure times heat loss from blood flow increases the irradiance necessary to create a mild lesion in the rabbit retina. [29] However, for small image sizes and short exposure times such as those likely to be encountered on exposure to pulsed laser beams, the effect of blood flow is negligible.

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Ridgeway [30] in this laboratory has calculated the maximum temperature in the PE by solving the differential equation for heat flow in the steady state; the solution has been programmed on a computer for cylinders of various diameters for a length of 100 μ, assuming exponential absorption of 40 per cent in a 10μ depth of PE and 60 per cent in a 100μ depth of choroid. Some results from this program are shown in Figure IV, which is a plot of the irradiance in watts/cm^2 vs image radius in microns needed to attain temperature rises of 5, 10, and 20°C. The increase in irradiance with decrease in image radius agrees with Vos' results for long exposure times where the transient solution approaches the equilibrium state. In fact, at the larger image sizes the equilibrium temperature attained is almost exactly proportional to the ratio of image diameters. Taking the minimum irradiance in Figure I as 6 watts/ cm^2 for an exposure of 3 min, and using the plateau temperature from the Vos curve in Figure II for an image 800μ in diameter, the retinal temperature rise will be 17.6°. A similar estimate using the steady state solution taken from Figure IV gives a retinal temperature of 17.7, an agreement to better than 1 per cent.

Retinal Injury at High Power Densities

The thermal concept of retinal injury is inadequate to cope with the biological effects induced in the eye by the high power densities (megawatts/cm^2) and short exposure times (5 - 50 ns) produced by giant laser pulses resulting from Q-switching. Electronic excitation and/or ionization from intense electric field gradients are fast processes ($\cong 10^{-14}$sec) but heat conduction does not extend spacially beyond 0.1μ from the site of absorption during the time

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of a giant pulse. Melanin granules in the PE and choroid are generally larger than this. Thus, the pigment granules or absorbing sites in the PE would reach high temperatures during the early stages of a giant pulse, producing ionization and possibly a plasma which would be opaque to incoming photons. A number of processes including two quanta excitation, Raman and Brillouin scattering, shock waves, frequency doubling, and reradiation by black body emission must be considered as possible modes of energy dissipation which can occur before heat conduction has time to play a prominent role. A further complication involves the classical temperature concept when applied to time intervals of 10^{-8}sec insofar as macromolecules (proteins and nucleic acids) are concerned.

Histological evidence indicates that for mild lesions induced in the rabbit retina by Q-switched ruby pulses having an image diameter of 800μ damage is confined to the PE and the inner layers of the retina (2 - 3 MW/cm^2, delivered in 30 ns); at somewhat higher power densities (5 - 10 MW/cm^2) subretinal hemorrhaging occurs in the region between the PE and the choroid. When power densities are increased still further (20 - 50 MW/cm^2) material is extruded into the inner retina and the vitreous. At these power densities hemorrhaging into the vitreous occurs; this represents catastrophic damage which may result in loss of the eye. These observations suggest shock waves as a possible mode of biological damage to the retina.

Berggvist et al [31] have studied the production of mild lesions in the rabbit retina by both Q-switched and multiple spiked ruby laser pulses. They report that much higher power densities (170 MW/cm^2) are required to produce minimal lesions when the image or spot size is 50μ in diameter. However, when an optical system was used to produce mild lesions having a spot diameter

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of 800 μ, the retinal irradiance was 3.7 MW/cm^2 for a 16 ns pulse, a figure which agrees in order of magnitude with the author's findings for ruby Q-switched pulses (2 - 3 MW/cm^2 for 30 ns exposure time). [32] The authors do not have, at present, any data for Q-switched pulses on the rabbit retina for image sizes less than 500μ in diameter. No measurable difference in the irradiance required to produce mild lesions has been found for image sizes of 1000, 800, and 500 μ. More information is needed on non-linear processes and their biological effects before an increase in irradiance with decrease in image size for Q-switched exposures can be explained.

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FIGURE CAPTIONS

Figure I: A log-log plot of average irradiance in watts/cm^2 vs exposure time in seconds for mild lesions in the rabbit retina. The image diameter on the retina was 800μ. x - laser; • - xenon lamp.

Figure II: A log-log plot of temperature in °C per unit of incident irradiance (4.18 J/cm^2/sec) vs time in seconds for several image diameters. It is assumed that all of the incident radiation is absorbed, 0.4 in PE and 0.6 in choroid. Adapted from Vos [26].

Figure III: A schematic diagram of the mathematical model used to describe thermal injury in the retina.

Figure IV: A plot of irradiance in watts/cm^2 vs image radius in microns for equilibrium temperatures of 5, 10, 20 °C in the retina, assuming exponential absorption, 0.4 in PE and 0.6 in choroid. Adapted from Ridgeway [30] steady state solution.

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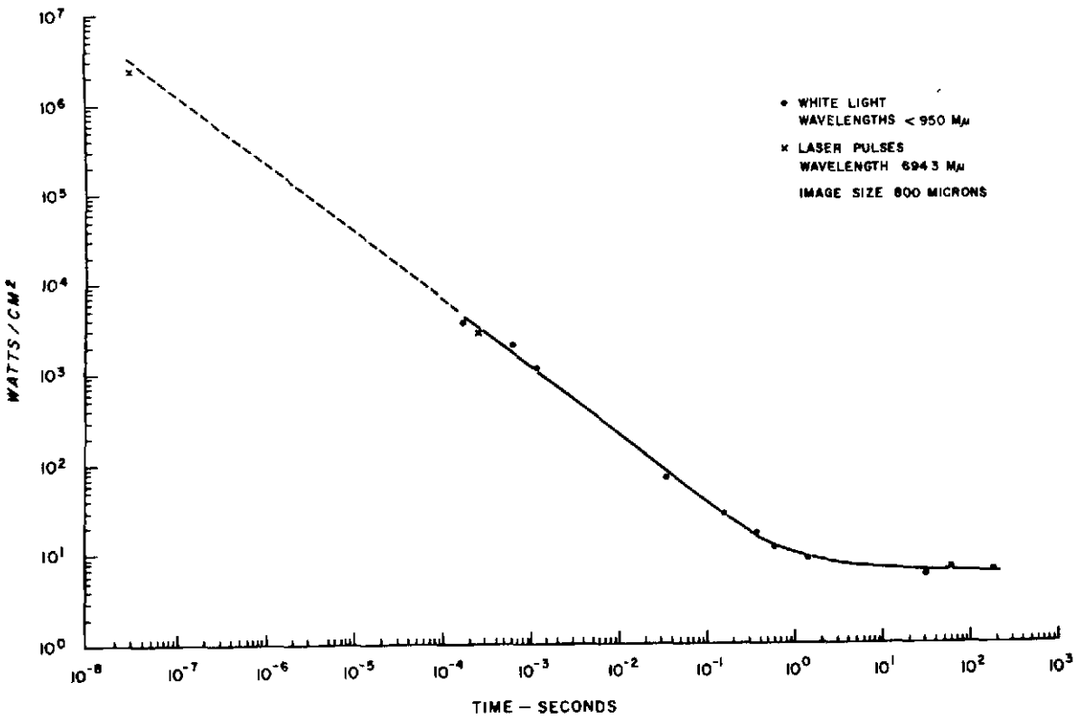

Figure I: A log-log plot of average irradiance in watts/cm^2 vs exposure time in seconds for mild lesions in the rabbit retina. The image diameter on the retina was 800μ. x - laser; • - xenon lamp.

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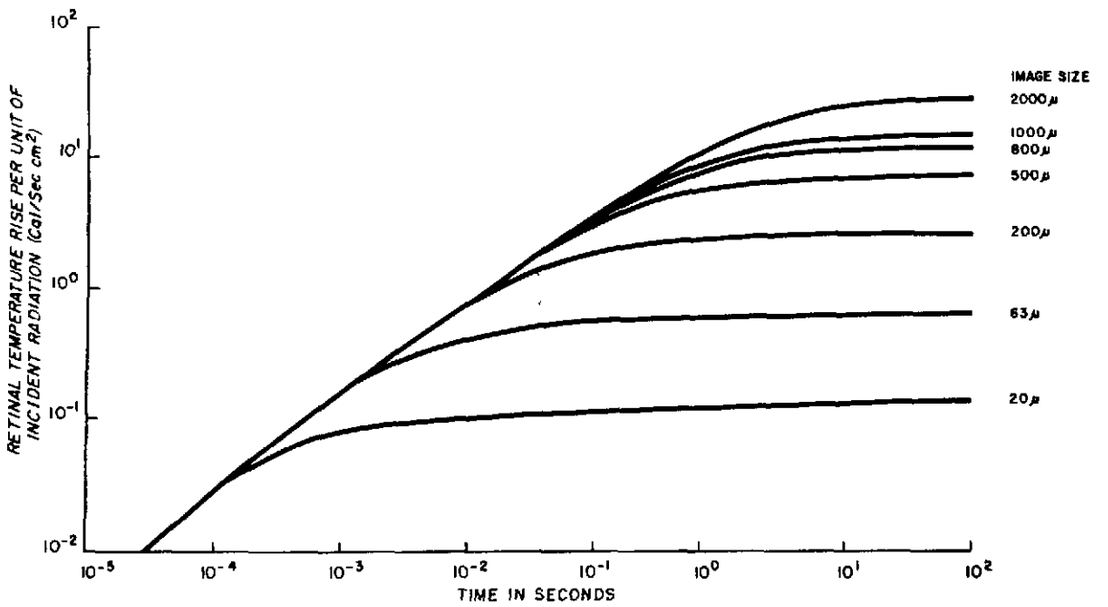

Figure II: A log-log plot of temperature in °C per unit of incident irradiance (4.18 J/cm^2/sec) vs time in seconds for several image diameters. It is assumed that all of the incident radiation is absorbed, 0.4 in PE and 0.6 in choroid. Adapted from Vos [26].

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SCHEMATIC DIAGRAM OF MATHEMATICAL MODEL

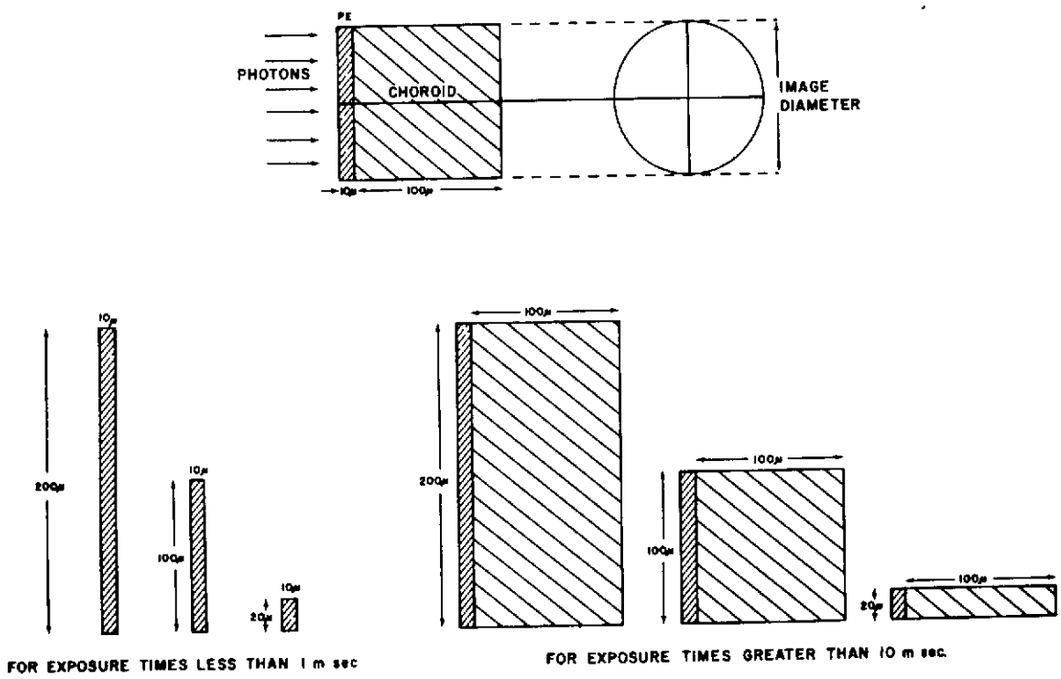

Figure III: A schematic diagram of the mathematical model used to describe thermal injury in the retina.

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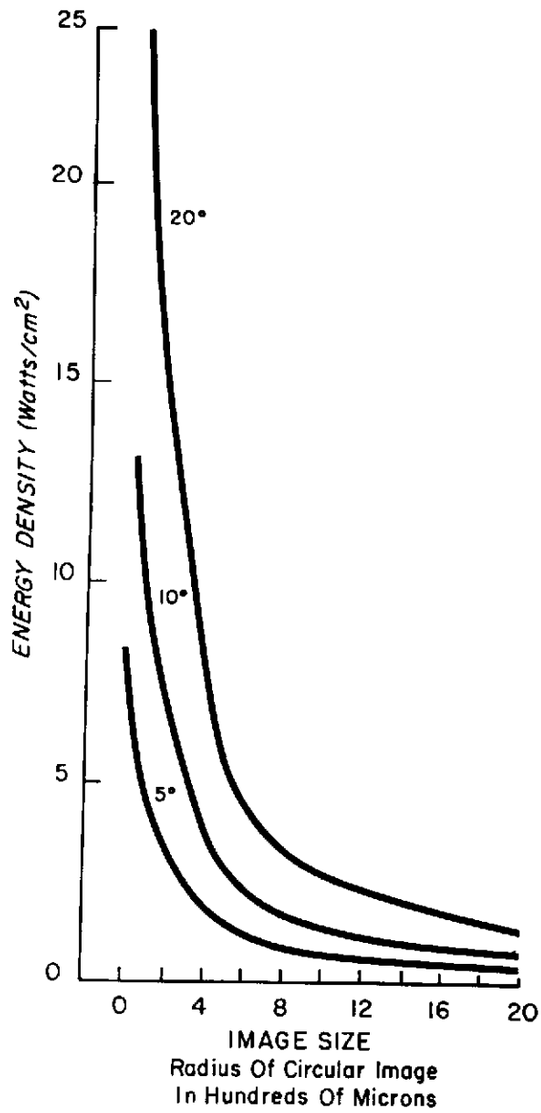

Figure IV: A plot of irradiance in watts/cm^2 vs image radius in microns for equilibrium temperatures of 5, 10, 20 °C in the retina, assuming exponential absorption, 0.4 in PE and 0.6 in choroid. Adapted from Ridgeway [30] steady state solution.

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