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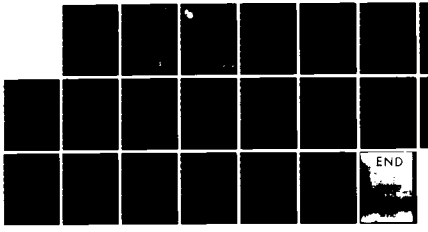

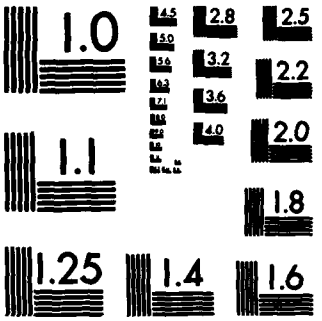

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HARRY ZWICK, PhD

DIVISION OF OCULAR HAZARDS

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Visual Function Changes after Laser Exposure - Zwick

Article One. Visual Function Changes after Chronic or Low-light Exposures
Aritcle Two. Experimental Assessments of Vision Function Changes in the Non-human Primate following Acute Laser Exposures

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VISUAL FUNCTION CHANGES AFTER LASER EXPOSURE

by
HARRY ZWICK, *Ph.D.*

Contents

PREFACE

The two articles in this publication appear as Chapters 2 and 3 in the *Handbook of Laser Bioeffects Assessment,* Volume 1. *Bioeffects Data.* E. S. Beatrice, M.D. and D.M. Penetar, Ph.D., editors. To offer wider dissemination of this information, two articles are presented in this Laboratory Note No. 84-48, Letterman Army Institute of Research, Presidio of San Francisco, California, where the author, Harry Zwick, Ph.D. is a Research Psychologist in the Division of Ocular Hazards. Dr. Zwick has published extensively on visual function and visual function changes—results of laboratory studies with a number of non-human species.

VISUAL FUNCTION CHANGES AFTER CHRONIC OR LOW-LIGHT EXPOSURES

Harry Zwick, PhD

Wolfe* established a grading scheme for classifying acute laser exposure and its effect on human visual acuity (page 15). Emphasis was placed on acute suprathreshold retinal exposure and the gross tissue change produced by such exposure in the retina. Light effects research on both animal and human subjects has indicated that light can produce more subtle functional and retinal tissue change in the retinal photoreceptors and related structures. Levels for such change need not be sufficient to produce thermal events in the retina (i.e. exposure levels less than Wolfe grade 0) but can be produced by photochemical damage mechanisms that are presently in the early stages of comprehensive explanation.

In this article, the effects of low-level light exposure on various measures of visual function are reviewed. Simple absolute threshold measurements of vision to more complex measures of pattern vision and color discrimination will be discussed with respect to low-level light damage and the possible morphological changes that may underlie such visual function loss. Studies that reflect the capacity of the visual system to recover from such exposure are also reviewed. The objective is to provide the reader with the fullest possible basis of the effects of light on visual function and retinal morphology correlates, and thereby a fuller understanding of the complexities of the dynamics involved in visual-induced dysfunction.

VISUAL ADAPTATION AND ABSOLUTE THRESHOLD STUDIES

Investigators (1, 2) suggesting the involvement of a photoreceptor system reported prolonged recovery in dark adaptation following bright light environmental exposure in humans. These investigations showed that recovery to full dark-adapted levels required in excess of 24 hr. While such findings did not reveal permanent change, the recovery time

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*John A. Wolfe, M.D., CAPT, US Public Health Service, Laser exposure of the human foveomacular retina and its effect on vision. *In:* Beatrice ES, Penetar DM, eds. *Handbook of Laser Bioeffects Assessment.* Volume 1. *Bioeffects Data.* Presidio of San Francisco, California: Letterman Army Institute of Research, 1984. Chapter 1. (Doctor Wolfe's present address is Phoenix Indian Medical Center, 4212 N. 16th Street, Phoeniz, Arizona 85016.)

was in excess of normal physiological recovery expected for rod vision. Similar findings were obtained in conditioned rodents (3, 4). Unusually long-term dark adaptation functions in either the albino or pigmented rat were found after 30 min of moderate white light exposure. While no pathologic evaluations were performed in these investigations, a correlation with optic neural atrophy was suggested to explain long recovery times as well as pupillary response mechanisms to prolonged exposure. The possibility of initial rod photoreceptor dysfunction was not suggested but not ruled out, as later work with comparable light levels and exposure durations have produced direct rod photoreceptor alteration.

The work of Noell and associates (5) demonstrated a definite correlation between a functional measure, the electroretinogram (ERG), and gross retinal degeneration induced by prolonged exposure to flourescent light. Using the ERG as a criterion response Noell et al (5) showed that the damage action spectrum was superimposable with the absorption spectrum of rhodopsin (the photopigment contained in rod outersegments). Gorn and Kuwabara (6) showed a similar action spectrum with ERG criteria. Kuwabara and Gorn (7) presented electron micrographic data that suggested a greater involvement of the rod outersegment than was suggested by the light microscopic data (5).

Clear evidence suggesting a close correlation between photoreceptor alteration and visual absolute threshold was demonstrated by Henton and Sykes (8) in the albino rat. These investigators showed a close correlation between photoreceptor alteration and absolute visual threshold in animals exposed cumulatively to 1000 lux illumination from flourescent light. Photoreceptor alteration progressed from outersegment vesiculation at 12 hr to innersegment involvement at 36 hr. Pyknosis of photoreceptor nuclei also progressed being less than 1% at 12-hr exposure and increasing to 5% at the end of 36 hr of exposure. Changes in absolute threshold were linearly related to light exposure with increasing loss in absolute threshold for 12, 24, and 36 hr of exposure. At 12 hr, loss in threshold was 1 log unit whereas at 36 hr of exposure, the loss was 2 log units in absolute visual threshold. While photoreceptor alteration and absolute threshold were correlated, no corresponding change in the pigment epithelium or the bipolar or ganglion cells of the inner retina was detectable.

While these investigations (6, 7, 8) suggest a good correlation with function and photoreceptor alteration, the obvious discontinuities between structure and function even in these studies cannot be overlooked. Even though early dark adaptation studies in human and animal subjects suggest that photoreceptor adaptation processes are affected by low-level exposure, no specific photoreceptor dissociation of effect can be gleaned from these studies. The works of Noell and others (7, 8), however, clearly suggest involvement of the rod photopigment, although light damage in

these experiments usually involved retinal structures other than photoreceptors. In recent work (8, 9), the correlation between structure and threshold measure is clearer, although even in these studies alternatives are not mutually exclusive. Henton and Sykes (8) reported dramatic correlation between function and structure for white light exposure but less so for near ultraviolet (UV) exposure (9).

CONE FUNCTION

Selective functional loss correlated with specific cone classes in the non-human primate retina has been demonstrated by Sperling and his associates (10, 11). In these experiments, rhesus monkeys were trained on an increment threshold spectral sensitivity test. A monochromatic test target is presented to the subject on an intense white light background and the threshold energy for just detecting this test target against this background is determined at test wavelengths every 20 nm across the visible spectrum (400-700 nm). The reciprocal of threshold energy-sensitivity-plotted as a function of wavelength defines the increment spectral sensitivity for the normal unexposed eye. For measurements made at background intensities sufficient to suppress rod function, a three-peaked spectral sensitivity function is obtained with prominent peaks at 460, 550, and 620 nm. Harwerth and Sperling (10) found that repetitive exposure to multiple bleaches of intense blue light at 5 log trolands (-2.75 log mW/cm^2) produced prolonged depression in spectral sensitivity in the blue region of the increment spectral sensitivity curve, corresponding with loss of the portion of the function fitted by the blue cone nomogram. Less permanent alteration in increment spectral sensitivity and qualitatively different effects on spectral sensitivity was obtained for exposure in the intermediate spectral region at 520 nm. In this experiment, recovery occurred within 4 weeks after exposure and spectral sensitivity curves returned to their before-exposure shape by exhibiting three peaks in the visible region. In more recent work (11), rhesus retina examined after short wavelength light exposure that produced the deficit in "blue" light sensitivity revealed swollen photoreceptors when examined under light microscopy.

While the work of Sperling and associates (10, 11) clearly provides evidence for photoreceptor alteration when measured functionally, evidence that light could disrupt normal neural channels between photoreceptor classes via photoreceptor input is suggested also by their intermediate light effects at 520 nm. Such exposure produced a dichromacy in the spectral sensitivity function resulting from the depression of the green cone process (520 nm) and the temporary loss of its neural modulation function on the long wavelength cone system. Both this observation as well as the inability to suppress the long wavelength cone system with long

wavelength light suggest that breakdown of neural retinal channels is one factor in functional spectral sensitivity loss.

Evidence that long wavelength cone systems can be suppressed with neural connection involvement by intense light exposure was obtained by Zwick and Holst (12) in measurements of turtle spectral sensitivity for low voltage ERG criteria. In these experiments on-line sensitivity was measured with a vector voltmeter technique (13). At levels of coherent light exposure just below those producing permanent loss in spectral sensitivity, strong evidence was obtained that the short (blue-sensitive) and intermediate photoreceptor system was neurally tied to the long wavelength cone system. At levels that produced permanent change in spectral sensitivity, a loss in long wavelength sensitivity was always accompanied by a smaller but significant loss in the short wavelength region of the spectrum. These results suggest that one class of photoreceptors normally modulating the output of another may indeed be damaged through the neural feedback from the system it modulates (12).

SUPRATHRESHOLD STUDIES - PATTERN VISION AND HUE DISCRIMINATION

In a well-documented series of investigations, Anderson and O'Steen (14) and Bennett et al (15) have shown that black-white pattern vision tasks in rodents are insensitive to nearly total destruction of rod photoreceptors. The evidence from recent investigations (16-18) strongly suggests that pattern vision in rats can be disrupted with either increased cumulative exposure or higher exposure levels. The presence of residual cones capable of mediating photic functions in the absence of rod domination has been suggested (19, 20). This explanation at present, however, has little morphological support (18).

Recent work from Wright and Sperling (21) has shown that rhesus demonstrating long-term loss in blue cone increment spectral sensitivity, also demonstrate reduced hue discrimination in the blue region of the spectrum. Color discrimination function loss for animals showing transient intermediate increment spectral sensitivity loss has also been demonstrated. Thus, suprathreshold measures of color vision can be correlated with other functions of receptor dysfunction, but may reflect receptor and post-receptor dysfunction.

VISUAL ACUITY DYSFUNCTION STUDIES

An early investigation of visual acuity dysfunction was described

by Smith (22), who found that American servicemen stationed in the Pacific during World War II developed deficits in visual acuity associated with funduscopically observable macular change. While quantitative dosimetry of the outdoor exposure conditions could not be made, it was apparent that such effects on visual function could not be produced by any significant temperature rise in the retina. Such changes while not attributable to thermal mechanisms of retinal damage probably involve both photoreceptor and other retinal structures. The lesion described by Ham and associates (23) for blue light exposure is not unlike that described by Smith (22).

Since Smith's observation (22), an extensive series of studies has been devoted to analyzing the effects of high as well as low intensity light exposure on visual acuity in non-human primates. The earliest of these investigations showed a fair correlation with the extent of foveal damage and visual acuity loss, after accounting for foveal edema present initially after intense levels of light exposure (24-26). Long-term followup of visual acuity (6 mo) after solar-induced foveal damage (27) revealed that visual acuity could recover to normal pre-exposure criteria, although spectral sensitivity measured with a Landolt ring acuity test still revealed foveal alteration (28). Morphological investigations of Tso (29) suggest that such recovery is consistent with foveal repair processes that take place over time following light-induced macular edema. According to Tso (29), photoreceptors adjacent to the damaged or missing photoreceptors eventually fill in the space originally occupied by damaged photoreceptors so that the retinal mosaic necessary for visual resolution is maintained even if the spectral organization in this area is altered. Merigan et al (30) also investigated retinal spatial and temporal processes following foveal macular insult and reported loss of fine spatial frequencies as well as loss in low temporal frequencies many months after exposure. While exposure conditions were not identical in these two studies (29, 30), the authors suggested that low temporal frequency loss might modulate the high spatial frequency loss from a distal peripheral retinal site.

In more recent work in our laboratory (31), we demonstrated that cumulative punctate foveal lesions, no larger than 20 to 50 microns in foveal retinal diameter, provided little evidence of permanent acuity loss. Such exposure in an awake task-oriented rhesus monkey is capable of producing transient loss in acuity as well as contrast sensitivity but little change in permanent visual spatial function. One explanation of these data is that foveal receptive fields are indeed larger than that which is normally attributed to them. Another theory is that alteration in normal neural interactions within the fovea produced by local damage may be capable of altering foveal receptive fields to compensate for critical tissue loss. Some evidence for neural reorganization from these experiments was provided by preliminary observations of contrast sensitivity slope changes

which appeared to be based primarily on increases in sensitivity for larger spatial frequencies.

Most of the investigations (24-26, 28-31) were performed with suprathreshold exposures, thus, eliminating the possibility of examining the dynamics of functional alteration at the transition from temporary to permanent functional loss in the absence of retinal edema. In experiments designed to measure the immediate effects of acuity loss from foveal exposure in awake task-oriented rhesus (32-34) thresholds for permanent change were far more consistent with histological criteria than for exposures made under anesthesia where several days elapsed before acuity could be remeasured in exposed eyes. At transition levels of exposure from temporary to permanent visual acuity loss, more than one exposure was required to produce a permanent loss in visual acuity. Such was not the case at levels of exposure below the transition energy levels. At these subtransition levels, each exposure pulse produced the same temporary deficit on acuity. In addition, when these measurements were made for spectral acuity targets, the transition from temporary to permanent acuity loss shifted downward to levels approximating the maximum permissible exposure (MPE) for extended source visible laser viewing. MPE levels evoked permanent loss in spectral acuity or spectral sensitivity for foveal acuity criteria regardless of whether the exposure wavelength was in the blue-green (515 nm) or in the red (647 nm) regions of the spectrum. While such exposure represented a single brief flash, no previous work has reported permanent and low-level effects in the long wavelength region of the spectrum for primate subjects (10, 23).

The effects of these exposures mimicked to a degree those obtained by Harwerth and Sperling (10) for 520 nm incoherent light exposure in that the spectral sensitivity function showed a notch at 520 nm which lasted at least 12 mo before recovery was complete. In the long end of the spectrum, evidence of a selective spectral effect was indicated, although followup of animal subjects was limited to only several weeks of postexposure threshold measurement in the wavelength region. In similar experiments, where spectral sensitivity was investigated for diffuse 514 nm laser exposure many times below the MPE level (35, 36), repetitive 2 hr daily exposure eventually produced prolonged loss in spectral sensitivity similar to that obtained by Harwerth and Sperling (10) for intermediate spectral exposure (520 nm). In these experiments, exposure was over the entire retina, as animals were exposed to diffuse argon laser light through hemispheric irradiation. Measures of spectral sensitivity for various acuity gap sizes were obtained both on days where animals were exposed to diffuse light for 2 hr and on days where no exposure was given. Changes in spectral sensitivity were not immediate. The initial changes in spectral sensitivity occurred for the finest gap-size criterion and only after 12 to 14 hr of total cumulative exposure. Two months after 40 hr of exposure, spectral sensitivity for the highest gap size showed a dip at 520 nm and, similar to Harwerth and Sperling (10), the

spectral sensitivity function could be fitted by a short and a long wavelength cone monogram. Unlike their study, these deficits were tracked for more than 3 yr in two animals with little change. In fact, evidence that progressive loss in spectral sensitivity for larger acuity criteria with the passage of time but without additional exposure was clearly obtained in two of the animals (36).

While presently these experiments are limited with respect to the number of animals exposed, the consistency both within and across two subjects led to several additional investigations in simpler animal species to determine the relevancy of coherence and speckle pattern produced from diffuse laser sources. In brief, we determined that the speckle produced by a laser incident on a diffusing surface played a role in producing a prolonged change in visual spectral sensitivity measured by electrical criteria in turtle cone functions (37, 38). The possibility that the unique aspects of diffuse laser light might have contributed, in part, to primate visual sensitivity loss at these low-light levels needs further consideration.

Morphologically, examinations of photoreceptor outersegments from retinas of monkeys exposed similarly but not trained indicate little differentiation in the degree of photoreceptor vesiculation, although an unusual amount of this type of morphology was readily obvious in both exposed and patched rhesus retinas (39, 40). When the foveas of these animals were examined, however, differences between the patched and exposed eyes were evident (41). Exposed eyes demonstrated a greater number of striated rootlets and basal bodies contained in the retinal pigment epithelium. While the significance of this increase is not clear, striated rootlets in the photoreceptor (42), as recently described, course all the way to the ganglion cell. The possibility that this alteration is involved with a complex and subtle retinal photoreceptor alignment function is only speculation at this point, but reasonable speculation in view of other recent psychophysical demonstration of the malleability of the Stiles-Crawford type 1 relationship (43, 44).

DAMAGE MECHANISMS

In considering the comparison of functional and morphological criteria of photoreceptor damage, it must be realized that the matrix of factors contributing to these criteria is not identical. Where functional measures depend on photoreceptor input, they have correlated well with photoreceptor morphological change (9, 11). In other studies functional and morphological correlation has not been as good. In rodent pattern perception experiments, changes in photoreceptor morphology occurs well in advance of loss in pattern perception (14-20). Pattern perception is lost long after

photoreceptor damage as exposure is cumulated to well over 100 total hours. In primate studies, correlation with apparent cone loss as well as the consequence of neural connections between classes of cone systems is reflected in spectral sensitivity measurement (10, 11, 35-38). In some studies (35, 16, 20), delayed change in function was observed upon cumulative exposure. Furthermore, in the investigation of coherent light, Zwick and associates (35-39, 41) found evidence to suggest both functional and morphological factors that have yet to be identified in studies where either white light or incoherent monochromatic light exposure is utilized. Foveal damage studies suggest a complicated ability of the fovea to alter its neural receptive fields under damage conditions that may involve extrafoveal retina through neural interaction with the fovea.

The possibility that more than one non-thermal damage mechanism may be operative simultaneously is an important consideration when attempting to explain the results of functional studies. At least two types of non-thermal damage mechanisms have been identified by various studies. Ham et al (23), and Lawwill et al (45) have identified by funduscopic and retinal histological criteria a non-thermal retinal damage mechanism that seems to have limited photoreceptor involvement. The work of Ham et al (23) suggests that melanin photochemical mechanisms are responsible for non-thermal lesions effective with blue light exposure, while Lawwill et al (45) suggested more nonspecific factors in the retina in explaining apparently the same type of action spectrum. However, whereas one explanation (23) involves a photochemical response of melanin in the short wavelength region, the other (45) explanation is nonspecific in its site of non-thermal damage. Non-thermal mechanisms based on photoreceptor dysfunction have been proposed by Sperling and associates (10, 11, 21). These studies suggest that low level light can alter photoreceptor structures prior to alteration in other retinal tissue. Regardless of which of these explanations is correct, recent data in the near UV have indicated that the peak of the damage action spectrum developed by Ham et al (23) continues to rise by more than one half a log unit for funduscopic criteria. Histological examination of these exposures indicates greater photoreceptor damage than RPE damage.

The complexity in non-thermal retinal damage mechanisms suggests that functional criteria may reflect several different morphological damage processes that are operative simultaneously. Therefore, clear cut identification at the functional level of photoreceptor retinal damage characteristics may be confounded by the simultaneous operation of other complex retinal damage processes.

FUNCTIONAL REVERSIBILITY, ACUTE EXPOSURE

Reversibility of functional loss has been observed often in visual function studies of light "damage." While data at present are dependent upon the particular scheme and purpose of specific experiments, it appears plausible that if active damage processes are involved in light exposure, then comparable repair processes may also be at work especially at threshold damage levels. Evidence of their presence should be reflected in functional measures.

Perhaps the most familiar evidence of repair, as measured by recovery for visual acuity, was that observed in the solar retinitis reports of Penner and McNair (27). These investigators reported that 50% of these patients initially having foveal macular damage and visual acuity loss as a result of solar eclipse exposure demonstrated full recovery within 6 months after exposure. Similar findings were reported in animal studies where gross foveal damage was produced by intense photic exposure (24-26). However, while fine acuity did return to near normal levels in these animals, altered foveal spectral sensitivity remained (29), reflective of residual damage to the fovea. Tso (29) has explained acuity recovery from such injury as a filling-in process of adjacent photoreceptors into the damaged foveal region.

After a period of months, the foveal mosaic necessary for fine acuity has been reconstructed so that fine resolution is again possible; parafoveal cones have largely replaced foveal cones. Alteration in foveal spectral sensitivity (28, 29) is, thus, not inconsistent with the morphological changes induced by such retinal injury.

In recent experiments (31) loss in visual acuity as well as contrast sensitivity were measured immediately after exposure to laser light producing small (20-50 microns) punctate lesions in the foveal area. No permanent loss from such lesions was obtained at first. With increased exposure over several months, evidence of altered contrast sensitivity slopes and increased sensitivity of the spatial frequency spectrum was observed. With additional exposure, loss in the finest spatial frequency was obtained eventually and coincident with this loss was a return to original baseline slope. These data suggest that some type of neural compensatory mechanism (alteration in foveal receptive fields or initially larger foveal receptive fields capable of dealing with fine spatial input) may be operative in maintaining normal acuity within limits of cumulative punctate foveal damage. Alteration in foveal receptive fields could result from foveal damage which would not be suspected by anatomical examination of foveal neural connections.

At levels of acute exposure, where gross retinal damage is not the overriding factor, recovery mechanisms are still inferrable from functional data. In a series of experiments on awake task-oriented

rhesus (31, 32, 33), the transition exposure level for a brief foveal exposure was determined. At that level, where temporary acuity loss transitioned to more permanent visual acuity loss, successive exposure had a cumulative effect on acuity, lengthening recovery time until recovery completely failed. This phenomenon was only observed at the intensity level that eventually produced permanent change in acuity. Below this level, no such additivity was ever observed. Full-term recovery from these exposures required at least 12 mo in three animals tested.

Exposures made in either the intermediate (514 nm) or the long wavelength region (647 nm) were similar in energy required for permanent change and as well as in the time required for full recovery.

FUNCTIONAL REVERSIBILITY, CHRONIC EXPOSURE

In the chronic light exposure experiments of Sperling and his collaborators (10, 11, 21), a differential effect was noted between exposures made in the blue vs exposures made in the green region of the spectrum for about the same energy levels. Sperling's group always found that blue light (460 nm) was more effective than green light (520 nm). Blue light exposure produced permanent suppression of the peak in the blue region of the increment spectral sensitivity function, whereas only a temporary dichotomy in the increment spectral sensitivity function was produced by green light exposure.

One explanation of this finding is that blue cones are metabolically different than other cones and more vulnerable to light exposure. Recent procein yellow experiments in primate retina have indeed suggested a differential metabolism between blue cones and other types of primate cone (46).

Similar visual function experiments performed with diffuse coherent green light (514 nm) exposure suggest that the interplay of neural factors operative in this region of the spectrum between photoreceptor mechanisms cannot be ignored. Zwick and his associates (35, 36) found that cumulative low-level exposure in this spectral region produced a delayed change in spectral sensitivity that was similar to the dichromacy reported by Sperling and his group (10, 11, 21), but it was permanent rather than transitory. In related studies (37, 38) on cone processes in turtle, it was demonstrated that characteristics of laser light incident on a surface, e.g. the speckle pattern, could alter neural interactions present between cone types in the turtle retina. It is, therefore, not implausible that neural interactions between classes of cone photoreceptors in some way maintain a normal balance, offsetting deleterious light exposure

effects, and are rendered somewhat inoperative when diffuse coherent light exposure in employed. Additional evidence has been obtained (41) suggesting that coherent light may make critical changes in the photoreceptor/RPE interface that may function to change critically the normal alignment of photoreceptors to incident light. How such a mechanism would be coordinated with neural mechanisms or photoreceptor metabolism is unknown but does suggest that the retina may have multiple pathways to protect itself from deleterious light exposure (43, 44).

COMMENTS

Low-level laser exposure can indeed alter visual function. Such exposure may produce an increase in ocular casualties and could alter the performance baseline of visual function required for critical visual performance. While low-level chronic exposure may not directly alter visual performance, it might well alter the threshold level for visual performance disruption; therefore its potential influence needs to be factored into laser safety. While protection from low-level exposure might eventually be developed, optimal protection will be a long-term developmental goal. An understanding of the basic resident recovery mechanisms associated with damage from low-level sources may lead to effective protection regimens, perhaps involving biological enhancement of natural protection already present in the human ocular system.

REFERENCES

1. Clark FA, Johnson ML, Dreher RE. The effect of sunlight on dark adaptation. Am J Ophthalmol 1946;29:823-836

2. Hecht S, Hendley CD, Ross S, Richmond PV. The effect of exposure to sunlight on night vision. Am J Ophthalmol 1948;31:1573-1580

3. Blystad W. Dark adaptation in pigmented rats: the effect of exposure to sunlight on night vision. Acta Ophthalmol 1951a;29:49-67

4. Blystad W. Visual adaptation in albino rats. Acta Ophthalmol 1951b;29:69-83

5. Noell WK, Walker VS, Kang BS, Berman S. Retinal damage by light in rats. Invest Ophthalmol 1966;5:450

6. Gorn RA, Kuwabara T. Retinal damage by visible light a physiological study. Arch Ophthalmol 1967;77:115-118

7. Kuwabara T, Gorn RA. Retinal damage by visible light. Arch Ophthalmol 1958;79:69-78

8. Henton WW, Sykes SM. Changes in absolute threshold with light induced retinal damage. Physiol Behav 1983;31:179-185

9. Henton WW, Sykes SM. Recovery of absolute thresholds with UVA-induced retinal damage. Submitted for publication, 1983

10. Harwerth RS, Sperling HG. Prolonged color blindness induced by intense spectral light in rhesus monkeys. Science 1971;174:520-523

11. Sperling HG, Johnson C. Histological findings in the receptor layer of primate retina associated with light-induced dichromacy. Mod Prob Ophthalmol 1973;13:291

12. Zwick H, Holst GC. Experimental alteration of the red cone photoreceptor process. Mod Prob Ophthalmol 1976;17:258-263

13. Padmos P, Norren D. The vector voltmeter as a tool to measure electroretinogram spectral sensitivity and dark adaptation. Invest Ophthalmol 1971;11:105-114

14. Anderson KV, O'Steen WK. Black-white and pattern discrimination in rats without photoreceptors. Exp Neurol 1972;34:446-454

15. Bennett MH, Dyer RF, Dunn JD. Light-induced retinal degeneration: effect upon light-dark discrimination. Exp Neurol 1972;34:434-445

16. Bennett MH, Dyer RF, Dunn JD. Visual dysfunction after long term continuous light exposure. Exp Neurol 1973;40:652-660

17. Weizenbaum F, Colavita F. Behavioral and electrophysiological changes in visual sensitivity following prolonged exposure to constant light. Exp Neurol 1975;48:440-446

18. Cicerone CM. Cones survive rods in the light damaged eye of the albino rat. Science 1976;194:1183-1185

19. Lavail MM. Survival of some photoreceptor cells in albino rats following long-term exposure to continuous light. Invest Ophthalmol 1976;15:64-70

20. Lemmon V, Anderson KV. Behavioral effects of retinal degeneration. Exp Neurol 1979;63:35-49

21. Wright A, Sperling HG. Psychophysical biochemical and histological studies of spectrally selective photic damage to primate retina. Presidio of San Francisco, CA: Letterman Army Institute of Research, 1983. Contract No 08114-83

22. Smith HE. Aetinic macular retinal pigment degeneration. US Naval Bulletin 1944;675-680

23. Ham WT, Mueller HA, Sliney DH. Retinal sensitivity to damage from short wavelength light. Nature 1976;260:153-154

24. Yarckzower M, Wolbarsht ML, Galloway WD, Fligsten KE, Malcolm R. Visual acuity in a stumptail monkey. Science 1966;152:1392-1393

25. Weiskrantz L, Cowey A. Comparison of the effects of striated cortex and retinal lesions on visual acuity in the monkey. Science 1967;155:104-106

26. Graham ES, Farrer DN, Young FA, Klopfer DF. Retinal lesions and visual acuity in the rhesus monkey. Holloman Air Force Base, NM: 6571st Aeromedical Research Laboratory, Technical Report, 1969;ARL-TR-69-3

27. Penner R, McNair JN. Eclipse blindness. Am J Ophthalmol 1966;61:1452-1457

28. Zwick H, Bedell RB, Bloom K. Spectral and visual deficits associated with laser irradiation. Mod Prob Ophthalmol 1974;13:299-306

29. Tso M. Photic maculopathy in rhesus monkey. Invest Ophthalmol 1973;12:17-34

30. Merigan WH, Pasternak T, Zehl D. Spatial and temporal vision of macaques after central retinal lesions. Invest Ophthalmol 1981;21:17-26

31. Zwick H, Bloom KR, Lund DJ, Beatrice ES. Effects of minimal or punctate foveal lesions on visual acuity and contrast sensitivity in the rhesus (Abstract). ARVO 1983:Invest Ophthalmol Suppl, Spring 1983:293

32. Robbins DO, Zwick H, Holst GC. A method for producing foveal retinal exposures in an awake, task-oriented, rhesus monkey. Behav Res Meth Instrum 1973;5:457-461

33. Robbins DO, Zwick H. Changes in spectral acuity following brief laser (647 nm) exposure. Colour Vision Deficiencies V, 1980:106-110

34. Robbins DO, Zwick H, Haenlein M. Changes in spectral acuity following laser irradiation. In: Proceedings of the Human Factors Society V, 1980;24:152-156

35. Zwick H, Beatrice ES. Long-term changes in spectral sensitivity after low-level laser (514 nm) exposure. Mod Prob Ophthalmol 1978;19:319-325

36. Zwick H, Stuck BE, Beatrice ES. Low-level effects on visual processing. In: Proceedings of Society of Photo-Optical Instrum Engrs 1980:55-62

37. Zwick H, Jenkins DJ. Coherency effects on retinal neural processes (ERG) of Pseudemys. Colour Vision Deficiencies V, 1980:146-150

38. Zwick H, Robbins DO, Knepp A. Changes in tectal spectral sensitivity and receptive field organization following coherent light exposure. Colour Vision Deficiencies V, 1980:151-156

39. Stuck BE, DeVillez G, Beatrice ES, Zwick H. Microscopic evaluation of rhesus retina after repeated low-level exposure to diffuse argon laser radiation (Abstract). ARVO 1980:Invest Ophthalmol Suppl, Spring 1980:189

40. Pitts DG, Chu LWF, Bergmanson JPG. Induced retinal changes from flourescent light exposure (Abstract). Invest Ophthalmol Suppl 1983;24:255

41. Schuschereba ST, Zwick H, Stuck BE, Beatrice ES. Basal body and striated rootlet changes in primate macular retinal pigmented epithelium after low-level diffuse argon laser radiation. Presidio of San Francisco, CA: Letterman Army Institute of Research. 1982;Technical Note 82-35TN

42. Schuschereba ST, Zwick H. Ciliary rootlets in primate rods and cones. Presidio of San Francisco, CA: Letterman Army Institute of Research. 1982;Technical Note 82-34TN

43. Enoch JM, Birch DG, Birch EE. Monocular light exclusion for a period of days reduces directional sensitivity of the human retina. Science 1979;206:705-707

44. Applegate RA, Bonds AB. Induced movement of receptor alignment toward a new pupillary aperture. Invest Ophthalmol 1981;21:869-873

45. Lawwill T, Crockett TS, Courrier G. Retinal damage secondary to chronic light exposure, thresholds, and mechanisms. Doc Ophthalmol 1977;44:379-402

46. De Martino FM, Schein SJ, McCrane EP. Staining of blue sensitive cones of the macaque retina by a flourescent dye. Science 1981;213:1278-1281

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Wolfe's Grades of Injury and Visual Acuity following Laser Retinal Injury*

Grade	*Ophthalmoscopic Findings*	*Range of Visual Acuity in Early Phase after Injury*					
		Subgrade A			*Subgrade B*		
I	Retinal edema	20/15	to	20/25	20/30	to	20/200
II	Retinal necrosis	20/15	to	20/40	20/40	to	20/400
III	Subretinal and/or intraretinal hemorrhage	20/15	to	20/50	20/100	to	20/400
IV	Vitreous hemorrhage and/or full-thickness retinal hole	20/15	to	Fc or worse	20/100	to	Fc or worse

A = Extrafoveal lesion; B = Foveal lesion; Fc = Finger counting

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EXPERIMENTAL ASSESSMENTS OF VISION FUNCTION CHANGES IN THE NON-HUMAN PRIMATE EYE FOLLOWING ACUTE LASER EXPOSURES

Harry Zwick, PhD

The general effects of light on visual function are discussed in another paper.* This discussion will continue to explore visual function effects but will be restricted to those effects that have produced profound acute changes.

Morphological effects are reported in other sections of this document. At sufficient power levels, lasers can burn and even cause hemorrhage in retinal tissue. Human accident reports, though incomplete by their nature, suggest that where damage has occurred, visual function and visual performance are decreased, as measured clinically.

However, the questions underlying loss of visual function and visual performance must be answered in more detail. Changes in retinal morphology suggest functional losses, but do not provide measurement of such losses. Similarly, post hoc human accident reports suggest but cannot substitute for experiments where exact dosages and effects on vision can be measured.

The macaque monkey is frequently the model (laboratory animal). Its acuity and contrast visual functions are similar to those functioning in humans. Where species differences exist, they can generally be resolved by differences in optical properties of the eye rather than differences in the organization of the sensory retina (1), although not without some exception (2).

This report will review past and present assessments on how the effects of foveal loss on visual function affect the fovea of non-human primates. The fovea has received major attention because of its critical importance to spatial and color vision and its critical location within the eye. The fovea in both the human and macaque eye is located on the visual axis of the eye. Thus, the attention it has received is warranted.

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*Zwick H. Visual function changes after chronic or low-light exposures. Presidio of San Francisco, California: Letterman Army Institute of Research, 1984. Laboratory Note No. 84-48:1-15.

MEASUREMENTS OF VISUAL ACUITY DEFICITS PRODUCED BY GROSS FOVEAL DAMAGE

In the animal studies (3-15), the principal ocular measurement was high contrast spatial visual acuity. This measuure was adopted by convention because it was the visual test most familiar and one with maximum validity. However, other measures of visual function were used in more recent studies which supplement this measure and expand on the overall effect of acute laser exposure on visual function (3). Spectral sensitivity of the eye to various "colors" across the spectrum has proven a sensitive measure. More recently, contrast sensitivity functions have allowed us to assess the effects of foveal exposure on the entire spatial frequency spectrum. Effects of exposure on threshold discrimination of small as well as large targets is possible. This information is important to the tactical question of how much visual function remains after exposure.

Since maximal acute vision is based on an intact fovea, the destruction of this region of sensory retina should result in a significant acuity loss. The initial investigation was done by Yarckzower et al (4). These investigators used a single stumptail macaque monkey and exposed the foveas of both eyes with a ruby laser photocoagulator. The spot size in this experiment for a single pulse was 500 microns (assuming optics were the same as those in conventional ruby laser photocoagulators). Pulse duration or number of pulses to achieve a foveal lesion was not given. Acuity changed from 20/30 (114 min of arc) before exposure to 20/180 (9.0 min of arc) after exposure.

Acuity measurements were made after anesthesia had abated; a period of at least 24 hr after exposure is estimated. Since both eyes were exposed, it is difficult to say whether acuity was totally free of anesthetic effect at the time of after-exposure measurement. If it was not, then absolute acuity changes to 20/180 might be somewhat inflated. Funduscopy and histology confirmed that the fovea was destroyed leaving a 1000 micron lesion centered on the fovea.

In a comparable investigation, Weiskrantz and Cowey (5) made lesions of varying sizes within the fovea with a xenon arc photocoagulator. These lesions were never as severe as those of the previous study. The maximal effect on acuity never exceeded a 60% deficit relative to pre-exposure baseline. They found that acuity loss was proportional to the size of lesion in the central fovea. The larger the central lesion, the greater is the loss in acuity. These losses are similar to human acuity off-axis measurements, where acuity drops off rapidly for measurements made outside the fovea.

In another investigation, Graham et al (6) examined monocular acuity losses produced by a ruby laser. They used a large spot exposure, about 950 microns centered on the fovea. They exposed the fovea at a level that was approximately 2.5 times the threshold dose

required for a lesion to be produced in the periphery of the same eye. The pulse exposure was 200 usec. Measurements of acuity 6 days after exposure showed about 60% deficit in acuity and, more importantly, only a slight decrease in acuity of the unexposed eye. At the end of 24 days, acuity in the normal eye was normal and a 60% deficit was still present in the exposed eye. Funduscopic examination at the end of 36 days revealed foveal lesions, as well as areas of the macula that appeared morphologically intact. These lesions were more similar to those reported by Weiskrantz and Cowey (5) than to those observed by Yarckzower et al (4).

SPATIAL RESOLUTION AND RESIDUAL VISION DEFICITS

It is evident from the above discussion that foveal damage will cause significant loss in visual acuity and that such loss may be directly related to the amount of foveal-macular tissue affected. However, when such lesions are produced they are not static entities. Often edema is present, especially at levels above threshold. Under such conditions, it is entirely possible that during the first several days or weeks after exposure, acuity measurements will vary as fluid accumulates and then dissipates in the retina.

To test this notion the investigators (3) found that losses in acuity during the first 2 wk after exposure were considerably more severe than losses measured in subsequent weeks. Measurements of acuity for photopic as well as scotopic illuminance background levels revealed initial effects across both scotopic and photopic retinal processes. Resolution of scotopic losses occurred within 2 wk after exposure, a period consistent with resolution of edema. Long-term losses in photopic acuity and spectral sensitivity were evident from 6 mo to more than 2 yr after exposure.

The most recent investigation in this type of foveal function was performed by Merigan et al (7). This investigation involved measurement of spatial resolution loss in rhesus following foveal destruction. Unlike previous investigations, spatial vision was investigated for the entire spatial frequency domain. (The measurement of visual acuity reflects only the finest resolution point where target contrast requirements are maximum. One can also measure the detection of large targets where target threshold contrast is much lower. This measurement is important to understanding how vision for low contrast targets is affected after foveal destruction.) The exposure parameters for this investigation were different from those in the study by Merigan et al (7). A single pulse from an argon laser (514 nm) was funduscopically centered on the fovea. Spot size was 1000 microns for a power level of 2 W. Measurements of visual function were not reported until 8 mo after exposure. At this time,

spatial contrast sensitivity functions showed maximal loss for the finest acuity targets with little loss for large acuity targets. This later result is important for comparisons with investigations to be reviewed in the following section of this report.

IMMEDIATE EFFECTS OF LASER EXPOSURES

While the studies described previously indicate that foveal damage affects fine spatial vision, immediate measurements were not always made, as many of these studies required anesthetized animal subjects. Anesthesia generally requires about 24 hr to abate. A second group of investigations (8-12) have been performed to assess the immediate effects of laser exposure, especially to determine the effects of those levels where anesthetic effects lasting 24 hr or more would normally mask out shorter term effects lasting only minutes or even seconds. In order for these measurements to be made, however, a behavioral technique for rapidly measuring visual acuity must be developed. Animal behavioral techniques for rapid psychophysical threshold measurement were introduced by Blough (13). Graham et al (9) adopted this technique for measurement of acuity thresholds in rhesus and used it to measure the immediate effects of single-pulsed (1.06 micron) laser exposure on rhesus (D.N. Farrer, PhD, USAFSAM, personal communication, 1982). An exposure level of 1.1 mJ for an unfocused beam was used. Funduscopically a retinal hemorrhage was produced. Immediate loss in acuity was obtained. Followup measurements were not made on subsequent days, so the long-term effects of this lesion were not assessed.

A similar procedure was developed by Robbins et al (10). In this procedure foveal exposure was not directly assessed by funduscopy on subsequent sessions. The immediate effects of increasing corneal exposure power levels were determined up to a corneal power level where permanent loss in visual acuity occurred. Thus, this experimental paradigm sought to find the transition between temporary and permanent functional loss. In this sense an important bridge was made between permanent effects, already known to occur at higher exposure levels and possible shorter term effects, that could not be measured in the presence of anesthetic masking effects.

While the transition from temporary to permanent visual acuity loss was reduced with test target characteristics (spectral vs white light acuity targets), temporary visual loss lasting several minutes could always be obtained as much as a factor of 10 below the level where losses in acuity became permanent. These effects were originally obtained with spot sizes of 150 to 350 microns for 100 msec exposure durations. Recent investigations (12) showed comparable effects for spot sizes as small as 50 microns. Effects measured with visible light in the red region of the spectrum (633 and 647 nm) were

no different than effects measured in the green region of the spectrum (514 nm). Such effects are probably not the result of a gross tissue change, as was the case in the other studies (3, 4, 9). The transient changes are the neural/photochemical variety, although comparisons with these studies are difficult because measures of acuity are not identical to measures of visual sensitivity (13).

The two previous experiments [(10) Farrer, personal communication, 1982] have raised the issue that relatively small spot laser exposure can produce significant transient effects on visual acuity. Such effects would have been missed in studies where anesthesia prevented measurements of visual function within the first 30 min after exposure. Robbins et al (11) investigated immediate transient effects below threshold burn, and Farrer (personal communication, 1982) investigated similar effects at retinal hemorrhagic levels. We have little information of retinal morphological disturbance at intermediate levels.

INTERMEDIATE LEVELS OF RETINAL DISTURBANCE (MINIMAL SPOT EXPOSURE)

To bridge the gap between the long-term and immediate effects of laser exposures to the retina, a third series of experiments was conducted in which we investigated pulsed exposure from a frequency doubled neodymium laser source (532 nm) on visual acuity and contrast sensitivity (12). A correlation was obtained in these experiments between transient changes in acuity or contrast sensitivity and retinal morphology. Unlike previous work, the lesions were small (20-50 u), "punctate" in appearance, and only observed in the foveal-macular area. Maximal immediate deficits in acuity ranged from deficits of 40% to 90% across measurements in two animals. In three additional animals, immediate changes in contrast sensitivity functions measured from 0.78 to 13.39 min of arc) varied from deficits of 40% to deficits of 70%. Most exposure sessions produced deficits of at least 40% averaged over the initial 2 min after exposure. (Note: In this experiment thresholds were digitized in 2 min running average blocks both for acuity and contrast sensitivity.)

Full recovery of spatial function required 10 min or more in some cases. Measurements of contrast sensitivity made across the spatial frequency spectrum were nearly uniform in both initial deficits and overall recovery time. This observation contrasts with that of Merigan et al (7) who had reported effects on spatial contrast sensitivity only in the high spatial frequency range. As the damage produced and the after-exposure measurement times are vastly different between these two experiments, comparison is difficult. However, it is entirely possible that we are actually dealing with different degrees of damage. Merigan et al (7) were not able to obtain stable

measurements until 8 mo after exposure. In this time, gross damage may well have resolved leaving only local foveal/macular injury, which resolved to some degree over such long after exposure periods (3, 14).

The non-selectivity of exposure on the spatial frequency spectrum measured in the Zwick et al (12) experiment may be due to small localized edematous sites that clear in the time course measured for recovery. Or, alternatively, such effects may result from the disruption of complex neural communication channels existing both within the fovea and between foveal and extrafoveal retinal sites. Passive "blanking" of the fovea can reduce the contrast sensitivity function across a much broader range of spatial frequencies than that typically ascribed to the fovea (15). This notion suggests that foveal disruption may have consequences both for fine acute vision as well as more "rugged" low contrast vision. The latter is extremely important in tactical scenarios where contrast conditions are poor and targets are extremely small and difficult to detect.

POTENTIAL PERFORMANCE LOSS IN COMBAT WITH VISUAL FUNCTION LOSS

However, we do not wish to broach the combat scenario question without some investigations that have gone beyond the immediate visual function loss and examined performance loss in an idealized military scenario. Callin et al (8) investigated the effects of single-pulse (200 nsec) small spot laser exposure on rhesus monkeys trained in a compensatory tracking task. They used pulsed laser light from a dye laser for either single 515 nm exposure or "white light" laser exposure comprised of 480, 508, and 595 nm laser lines. They found that this single pulse exposure from 0.55 to 3.75 uJ at the retina could produce distraction from this task for periods ranging from 1 to 3 sec. Often no effect at all was produced.

One potential explanation of these differences involves the geometry of the test and exposure conditions. In the Callin at el (8) experiment, a small spot "flashed" a small retinal region but the target subtended considerably more retinal area than the exposure. Furthermore, no attempt to restrict the exposure to the fovea was made in this experiment. In the experiments of Robbins et al (11) and Zwick et al (12), the exposure was always coaxial with the gap in a Landolt ring that subtended less than 1 min of arc. If an animal was performing at a 20/20 or better acuity criterion, then the flash occurred and presumably the fovea was exposed, as it would have been in line with the gap in the Landolt ring. Furthermore, Zwick et al (12) obtained evidence of foveal damage. Robbins et al (11) reported initial deficits in keeping with prediction from off-axis acuity expectations, as discussed by Weiskrantz and Cowey (5).

Thus, where military tasks require foveal capability, i.e. fine visual acuity, it may be reasonable to assume that losses in tracking behavior greater than reported by Callin et al (8) will result from threshold or near threshold foveal retinal burns.

The effects of exposures off the fovea have not been systematically investigated in the manner that has been applied to foveal exposures. Yet, the question of the effects of peripheral retinal exposure or damage is not irrelevant. The potential for many exposures made outside the fovea may be as large as that for foveal exposure, although much information suggests that primates, especially human primates, foveate during a great percentage of their visual experience. It may be relatively safe to say that most peripheral exposures would not affect high contrast acuity. It is more difficult to say that such exposure or damage, if extensive enough, would not affect peripheral retinal functions such as movement perception, dark adaptation, and more complex visual functions involved in night vision. Adequate data to answer this question are unavailable at this time.

Conclusions

This review of available animal investigations suggests that foveal exposure from intense laser sources alters visual function, as measured in visual acuity, spectral sensitivity, and contrast sensitivity experiments. Little evidence is available for intense flash effects on specific visual tasks. A task that required 20/20 acuity under minimal contrast conditions would have a high visual function load; a task having low acuity and high contrast conditions would have a low visual load or requirement. In applying the performance criteria of Wolfe (16) to the experiments described in this paper (Table), we have attempted to bridge the gap between visual function loss and performance. One should be cautioned, however that with limited animal data, such extrapolation is extremely difficult and should be used only as a general first approximation to prediction.

TABLE

Changes in Rhesus Spatial Vision and Visual Performance Induced by Intense Laser Exposure

Authors Wavelength Pulse Width	Foveal Exposure Level	Foveal Spot Size (u)	Foveal Damage	Recovery Time	Visual Decrement Wolfe Grade(16)*
Yarkczower et al (4) 694, 20 nsec	Ruby Photo-coagulator	> 500	Total 1000 micron lesion fovea	None obtained in post period 24 hr	IB-IIB †
Weiskrantz, Cowey (5) Xenon, long pulse	Xenon Photo-coagulator	> 500	0/5 to 3 optic disc diameters	None given	IB-IIB
Graham et al (6) 694 nm 200 nsec	2.34 J/cm^2	950	Partial 300 microns	None over 36 days post	IB-IIB
Farrer ‡ 1.06 20 nsec	1.0 mJ	<100	Lesioned area partial 1000	Not available	IIIB
Zwick et al (3) 694 nm 20 nsec	1.1 mJ	1000	Central 300	Scotopic 2 wk photopic 6 mo to 3 yr	IIB
Merigan et al (7) 514 nm 2W (laser head) 50 msec	Argon laser Photo-coagulator 2W (laser head)	1000	300 foveal center	Not given	IIB

Table (concluded)

Changes in Rhesus Spatial Vision and Visual Performance Induced by Intense Laser Exposure

Authors Wavelength Pulse Width	Foveal Exposure Level	Foveal Spot Size (u)	Foveal Damage	Recovery Time	Visual Decrement Wolfe Grade(16)*
Robbins et al (10,11) 633, 647, 514 nm 100 msec	1-10 mW (corneal power)	50-350	Not observed time of exposure	Temporary in 20 min	0-1 Transient
	11 mW				ITB Permanent§
Zwick et al (12) 532 nm 20 nsec	0.5 J-3.5 J TIE	20-50	Punctate lesions	Full recovery 12-16 min	Pre IB Transient
Callin et al (8) 513 nm white (480.508,595 nm)	0.55 J-3.5J TIE	20-50	...	1-3 sec	0 (No effects to transient effects reported)

Wolfe's Grades of Injury and Visual Acuity following Laser Retinal Injury

Grade	Ophthalmoscopic Findings	Range of Visual Acuity in Early Phase after Injury	
		Subgrade A	Subgrade B
I	Retinal edema	20/15 to 20/25	20/30 to 20/200
II	Retinal necrosis	20/15 to 20/40	20/40 to 20/400
III	Subretinal and/or intraretinal hemorrhage	20/15 to 20/50	20/100 to 20/400
IV	Vitreous hemorrhage and/or full-thickness retinal hole	20/15 to Fc or worse	20/100 to Fc or worse

A = Extrafoveal lesion; B = Foveal lesion; Fc = Finger counting

† Measurements not made beyond 24 hr; full recovery time unknown

‡ Personal communication, 1982, DN Farrer, PhD, USAFSAM

§ Grade determined at 60 minutes or up to 9 months after exposure

. . .No evidence that foveal exposures were made

REFERENCES

1. Cavonious CR, Robbins DO. Relationships between luminance and visual acuity in the rhesus monkey. J Physiol 1973;32:239-246

2. Zwick H, Robbins DO. Is the rhesus protanomalous? Mod Prob Ophthalmol 1978;19:238-242

*3. Zwick H, Bedell RB, Bloom KR. Spectral and visual deficits associated with laser irradiation. Mod Prob Ophthalmol 1974;13:299-306

*4. Yarckzower M, Wolbarsht ML, Galloway WD, Fligsten KE, Malcolm R. Visual acuity in a stumptail monkey. Science 1966;152:1392-1393

*5. Weiskrantz L, Cowey A. Comparison of the effects of striated cortex and retinal lesions on visual acuity in the monkey. Science 1967;155:104-106

*6. Graham ES, Farrer DN, Young FA, Klopfer DF. Retinal lesions and visual acuity in the rhesus monkey (Macaca mulatta). Holloman Air Force Base, NM: 6571st Aeromedicine Research Laboratory 1969;Technical Report ARL-TR-69-3

*7. Merigan WH, Pastermak T, Zehl D. Spatial and temporal vision of macaques after central retinal lesions. Invest Ophthalmol 1981;21:17-26

*8. Callin GD, Devine JV, Garcia P. Visual compensatory tracking performance after exposure to flashblinding pulses. II Sub-damage threshold laser irradiation of rhesus monkey subjects. Brooks AFB, TX: USAF School of Aerospace Medicine, 1981;Technical Report: SAM-TR-81-7

9. Graham ES, Farrer DN, Crook GH II, Garcia P. A self-adjustment procedure for measuring the visual acuity of rhesus monkeys. Behav Res Meth Instr 1971;2:301-305

*10. Robbins DO, Zwick H, Holst GC. A method for producing foveal retinal lesions. Behav Res Meth Instr 1973;5:457-461

*11. Robbins DO, Zwick H, Hanelin M. Changes in spectral acuity following laser irradiation. In: Proceedings of Human Factors Society 1980;24:162-166

*12. Zwick H, Bloom KR, Lund DJ, Beatrice ES. Laser ocular flash effects. West Point, NY: In: Proceedings of 1982 Army Science Conference, 1982:

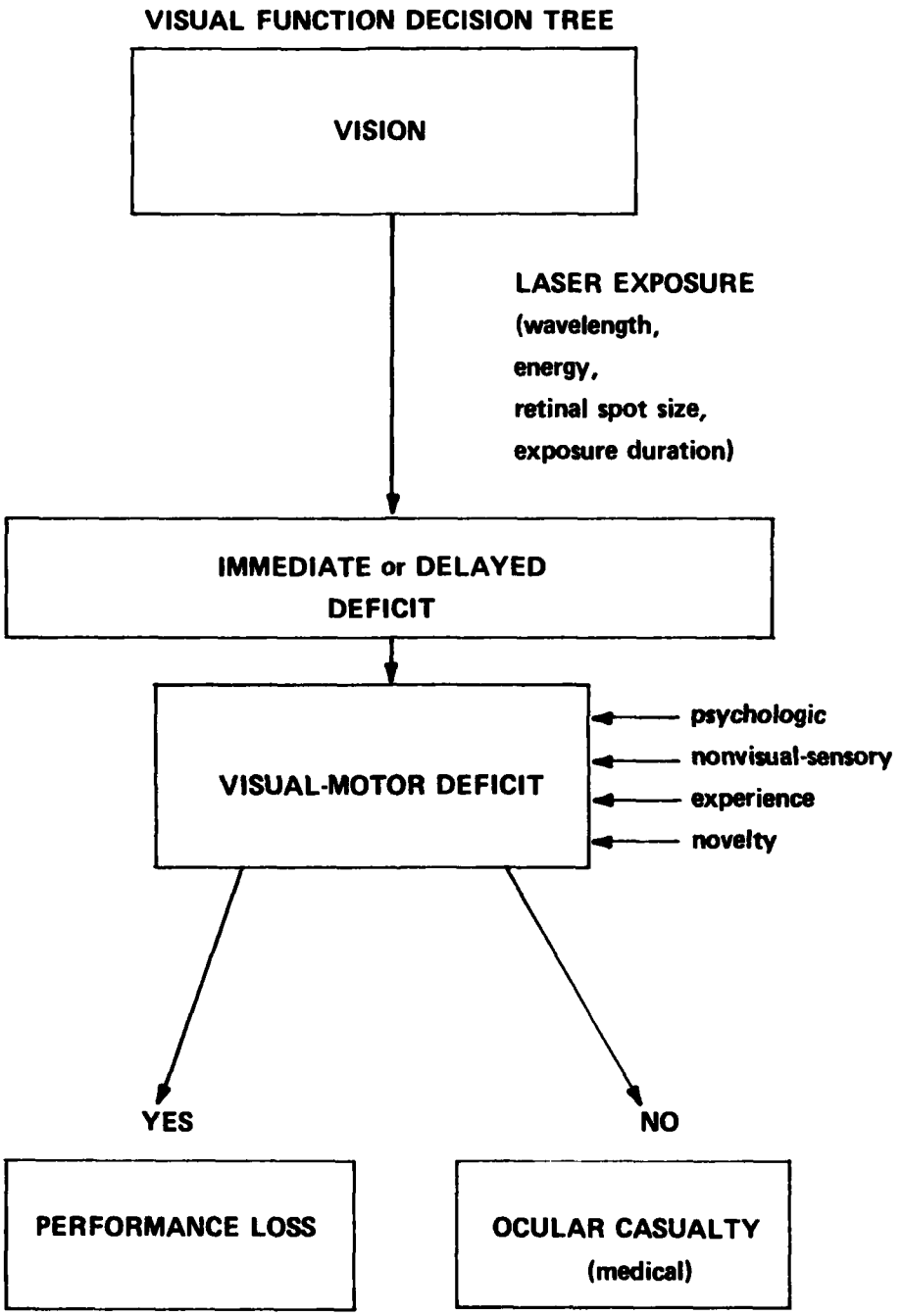
*Denotes references used in Table.

13. Blough DO. Dark adaptation in the pigeon. J Comp Physiol Psychol 1956;49:425-430

14. Tso M. Photic maculopathy in rhesus monkey. Invest Ophthalmol 1973;12:17-34

15. Kelly DO. Photopic contrast sensitivity without foveal vision. Optics Letters 1978;2;79-81

16. Wolfe JA. Laser exposure of the human foveomacular retina and its effect on vision. In: Beatrice ES, Penetar DM, eds. Handbook of Laser Bioeffects Assessment. Volume 1. Bioeffects Data. Presidio of San Francisco, CA: Letterman Army Institute of Research, 1984:Chapter 1


VISUAL FUNCTION DECISION TREE
VISION
LASER EXPOSURE
(wavelength,
energy,
retinal spot size,
exposure duration)
IMMEDIATE or DELAYED
DEFICIT
VISUAL-MOTOR DEFICIT
psychologic
nonvisual-sensory
experience
novelty
YES
NO
PERFORMANCE LOSS
OCULAR CASUALTY
(medical)

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