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LASER RETINAL INJURY

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DIVISION OF OCULAR HAZARDS

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LASER RETINAL INJURY - WOLFF

(signed)

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Laser retinal injury poses a grave threat to military personnel. Both irreversible damage with potential lifelong visual disability and reversible injury with immediate interference when critical visual demands are need can occur. Laser retinal lesions can be graded ophthalmoscopically: Grade I - retinal edema; Grade II - retinal necrosis (coagulation); Grade III - retinal hemorrhage; Grade IV - vitreous hemorrhage and/or retinal hole formation. All 23 medically reported cases of laser retinal injury show that acute visual effects and permanence of visual disability are directly correlated with increasing

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grade of injury and closeness of lesion to the fovea. Laser protective eyewear gives protection only from specific wavelength(s) of laser radiation.

ABSTRACT

Laser retinal injury poses a grave threat to military personnel. Both irreversible damage with potential lifelong visual disability and reversible injury with immediate interference when critical visual demands are needed can occur. Laser retinal lesions can be graded ophthalmoscopically: GRADE I - retinal edema; GRADE II - retinal necrosis (coagulation); GRADE III - retinal hemorrhage; GRADE IV - vitreous hemorrhage and/or retinal hole formation. All 23 medically reported cases of laser retinal injury show that acute visual effects and permanence of visual disability are directly correlated with increasing grade of injury and closeness of lesion to the fovea. Laser protective eyewear gives protection only from specific wavelength(s) of laser radiation.

PREFACE

This document, Laser Retinal Injury, is published as a separate Institute Report to offer a wider dissemination of the grading scale for retinal laser injuries (i.e. Wolfe's Grades) and a summary of all medically reported cases of all dental laser retinal injuries (23 to date). A version of the report was formerly included in Wolfe JS. Laser exposure of the human foveomacular retina and its effect on vision. In: Beatrice ES, Penetar DM (eds). Handbook of laser bioeffects assessment: bioeffects data (Vol 1). Presidio of San Francisco, CA: Letterman Army Institute of Research. 1984;3-37. Another version of the report has been submitted for publication in the open literature.

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LASER RETINAL INJURY

The risk of ocular injury from laser exposure on the modern battlefield is a realistic possibility. Lasers are currently being used as target rangefinders, designators and illuminators, as simulators of live fire in training (MILES), and in laser-assisted radars and communication devices. Operators of these systems may be inadvertently exposed. Additional threat of exposure exists from the potential utilization of laser energy in offensive weaponry.

Tremendous energy fluxes can be produced by lasers; in fact, power densities many times greater than those on the surface of the sun can be generated. The eye is the organ most susceptible to laser damage since radiant energy in the visible and near infrared spectral region is transmitted through the ocular media onto the retina. The cornea and lens focus this energy upon the retina, concentrating it many times (1).

The visual demands placed on today's soldier are enormous. Although tremendous technological advances have been made in military systems, these systems rely heavily on the operators' hand-eye coordination and fine visual discrimination. For satisfactory task performance and mission completeness these visual demands must be met.

The likelihood of laser eye injuries on the battlefield and their capacity to cause acute reduction in vision compels military medical personnel to become familiar with laser-induced injuries and the visual impairments apt to result from them. Such knowledge is important to assure prompt and accurate diagnosis and treatment and to promote and specify eye protection from lasers of different and multiple wavelengths.

Recently, a grading scale for laser retinal injury was proposed, and all medically reported cases of accidental laser retinal injury were reviewed (2). These injuries were graded and correlations were made between grade of injury and acute visual effects. The purpose of this paper is to present this grading scale and the range of visual impairment associated with these injuries. Cases of accidental laser retinal injury are summarized.

GRADES OF LASER RETINAL INJURY

In 1969, Zweng et al (3) classified retinal lesions in the rhesus monkey based on ophthalmoscopic changes following increasing exposure to ruby laser energy. Their grading scale was intended to serve as a guide to the placement of therapeutic laser photocoagulation burns of optimal intensity. The classification proposed for humans is a modification of that scale (Table).

TABLE

*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

Grade I lesions are characterized by retinal edema (Fig 1A). The involved retina loses its normal transparency, becomes cloudy, and takes on a gray-white color, but the orange-red, underlying choroidal hue is usually still visible. In Grade II lesions, retinal coagulation or necrosis is present, and these lesions appear densely white (Fig 1B), obscuring the underlying choroidal color. In addition, loss of retinal substance, as evidenced by thinning of the tissue, may be seen. Grade III lesions show hemorrhage, either subretinally or within the retina (Fig 1C). The presence of hemorrhage usually indicates a break in Bruch's membrane, between the retina and the choroid, allowing blood from the choroidal vasculature access under or into the retina. In Grade IV lesions, hemorrhage has dissected further into the vitreous and/or there is a full-thickness retinal hole (Fig 1D). Lesions are subclassified, A or B, depending on location within the retinal topography. Subgrade A are extrafoveal and Subgrade B are foveal lesions.

The range of acute reduction in visual acuity during the early phase following laser retinal injury is listed in the Table. In general, the degree of visual acuity impairment is directly proportional to the proximity of the lesion to the fovea. Foveal injury (Subgrade B, Table) can result in pronounced reduction in acuity, even when the injury is not severe. By contrast, extrafoveal injury (Subgrade A, Table) may cause little or no effect on acuity unless there is spread of edema into the fovea or bleeding into the vitreous involving the visual axis. However, extrafoveal lesions may cause a variety of visual field defects which may be disturbing even though they do not reduce visual acuity. In addition, a hemorrhagic extrafoveal lesion, which may go unnoticed at the time of injury because the blood remained contained within the retina or within the vitreous but outside the visual axis, could cause a profound loss of vision if later on the blood dissected its way into the visual axis (4).

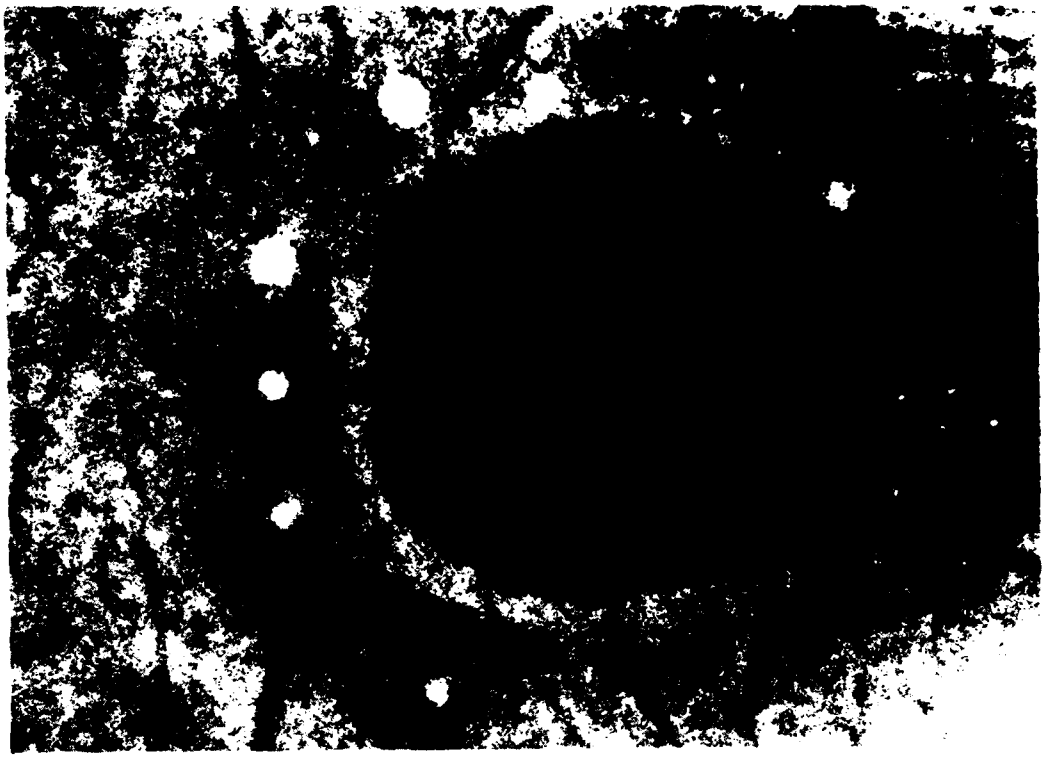

Figure 1A,B.
Laser retinal lesions in rhesus monkey.

Figure 1A (above).
Row of Grade I lesions just outside fovea showing retinal edema.

Figure 1B (left).
Grade II lesion inside fovea with dense center of retinal necrosis and surrounding zone of retinal edema.

-continued-

Figure 1 (concluded) C and D. Laser retinal lesions in rhesus monkey.

Figure 1C (above). Grade III lesion at edge of fovea with subretinal and intraretinal hemorrhage.

Figure 1D (right). Grade IV lesion inside fovea with stream of hemorrhage into vitreous.

ACCIDENTAL LASER RETINAL INJURY

Since the first reported case of accidental laser retinal injury by Rathkey in 1965 (5), an additional 22 cases have appeared in the world's medical literature. The details of these 23 injuries are tabulated in the Appendix. Where information was sufficient, estimated exposure doses were compared to the wavelength appropriate ED_{50}* levels for minimal retinal lesion in the rhesus monkey, and also to the appropriate maximum permissible exposures (MPE). The ratios of the exposure doses (E) estimated for these accident cases to the ED_{50} (i.e. $RED_{50} = E/_{ED50}$) and to the MPE (i.e. $RMPE = E/_{MPE}$) are given in the Appendix.

Nineteen of 23 patients (82.6%) reported immediate visual disturbances ranging from minimal blurring to dense blind spots. One patient reported loss of all vision for 1 to 2 min (6). Five of the 19 patients (26.3%) reported additional immediate symptomatology of a nonvisual nature, such as pain and audible sensations. These nonvisual symptoms occurred in association with severe injury (all five were Grade IV).

Seven of 21 patients (33.3%) were examined the same day as the injury, one was seen "immediately" (7) and another 20 min later (5), and 15 of them (71.4%) were seen by the next day. Seven (33.3%) had visual acuity reduced to 20/200 or worse when first examined; one had bilateral reduction to 20/200 (8). Twelve (57%) had visual acuity no better than 20/100. When first examined, 22 of 23 (95.6%) had a visual field defect, ranging from a central blur on Amsler grid to large absolute central scotomata. Sixteen of the patients (69.5%) had retinal hemorrhages and eight (34.7%) had vitreous hemorrhages (Fig 2). Eight (34.7%) formed retinal holes.

Fifteen out of 22 eyes (68%) with reduced vision when first examined showed improvement several days to several months later. Most of the eyes which did not improve had macular hole formation (Fig 3). One patient developed macular pucker with foveal involvement as a late complication which caused further loss of vision (11).

Nine cases (39%) had either vitreous hemorrhage or retinal hole formation, six of which had both. It is logical that these should occur together. Since the source of the vitreous hemorrhage is usually the choroidal vasculature, a retinal disruption would be needed for blood to get into the vitreous. Of these nine cases, all involved a pulsed mode of laser energy delivery with pulse durations in the nanosecond range. This correlates with the theory of retinal

Figure 2 (above). Day of laser injury. Commotio retinae with preretinal and vitreous hemorrhage. Grade IVB. ***Reproduced with permission from Boldrey et al (17).***

Figure 3 (below). Same case as Fig. 2, three months after laser injury. Foveal hole with edge detachment. Grade IVB. ***Reproduced with permission from Boldrey et al (17).***

damage from acoustic shock waves in the very short (nsec) pulse range causing explosive tissue injury (12, 13). Intermediate (msec to 10 sec) exposures produce mainly thermal effects (13, 14) and long (greater than 10 sec) exposures result in photochemical damage (14).

In the right-hand column of each page (Appendix), the grade or retinal injury is listed. Of the 24 injured eyes, all but two (15) could be graded. Half (11 of 22) of these were in the most severe (Grade IV) category, and nearly three-fourths (16 of 22) were Grade III or worse. Foveal (Subgrade B) injuries outnumbered extrafoveal injuries by more than two to one. All foveal injuries except one (6) received medical attention within one day of the injury. This is understandable owing to the more severe visual symptomatology associated with foveal lesions. All but one injury (8) were unilateral and occurred at close ranges. Frequently histories revealed that the exposures took place during performance of some alignment procedure. This accounts for the high incidence of foveal injuries. Case 19 (8) is particularly noteworthy in that the patient sustained bilateral retinal injury when exposed at a distance of 350 meters from the laser. Initially, the visual acuity was reduced to 20/200 in each eye. The worse eye remained at 20/200 with a foveal retinal hole, while the better eye had improved to 20/20 when examined 3 months after injury.

None of the authors (5-8, 11, 15-23) mentioned that the patient had (or had not) been wearing laser protective safety goggles when the laser injury occurred (Appendix). Goggles are helpful if they protect against the specific laser wavelength; if they do not protect against the wavelength to which one's eye is exposed, an injury will occur. For example, a physicist was operating a Q-switched neodymium laser with a Raman cell. He was wearing laser goggles which gave him specific protection for neodymium (1064 nm) and frequency-doubled neodymium (532 nm) when he was exposed to the laser beam which had been frequency-shifted by the Raman cell to 770 nm. In spite of the laser goggles, he sustained an injury to the parafoveal retina of the left eye. His visual acuity was 20/50 when he was examined 4 days later, and it gradually improved to about 20/25 over a few weeks time (M. Santangelo, MD, personal communication, 1983).

There are several characteristics of these patients which make them particularly interesting from a military perspective. Most of them were engaged in some fine visual task when exposed. In those cases where sex was specified, all except one (6) were male. They were young (mean age, 26.6 years). This compares favorably to the Army where 90% of enlisted personnel are males, and where the average age of enlisted forces is 26 years (letter, U.S. Army Military Personnel Center, 10 June 1983). In the reported cases, 22 of the patients were white, one was Oriental (7). However, nonwhites may be more susceptible to retinal laser damage because of increased energy absorption by melanin in the more heavily pigmented retinal pigment

epithelium and choroid (24). Nonwhites comprise over 40% of the Army's enlisted personnel (letter, USAMPC, 10 June 1983).

Appendix shows that all reported cases of accidental laser exposure had ophthalmoscopic findings correlating with reduction in visual function as measured clinically (i.e. visual acuity and visual field testing). There are no reports in the literature of alleged accidental laser exposures where there is a reduction in vision that is not explained by an ophthalmoscopically detectable lesion. That is not to say, however, that this could not happen. Animal data have shown alterations in visual function, some long-lasting, occurring after foveal exposure to laser radiation below the threshold for minimal detectable ophthalmoscopic lesions (25-27). Furthermore, our current clinical methods of measuring visual function may not be sensitive or specific enough to detect changes in function at low levels of exposure. Recent laboratory evidence in primates indicates that changes in spectral acuity occur before any change is found in achromatic (i.e. black-against white) acuity following foveal exposure to low level laser radiation (25, 26). Contrast sensitivity, static perimetry, and dark adaptometry (especially including retinal profile analysis) may be better methods to assess the function of the peripheral retina, when peripheral retinal laser exposure is suspected. Potential problems may arise for the military on two accounts. First, exposure of personnel performing critical visual tasks to low level laser radiation could interfere with task performance and satisfactory mission accomplishment. Second, medical personnel may have difficulty determining when visual functional impairments unassociated with ophthalmoscopic findings were actually caused by low level laser exposure. Compounding the problem may be the discovery of fundus lesions resembling laser lesions in those personnel at risk to laser exposure who are undergoing routine or unrelated physical examination.

CONCLUSIONS AND RECOMMENDATIONS

The soldier is especially vulnerable to sustaining a laser injury. Military physicians need to increase their awareness of this potential injury and to become familiar with its manifestations. More sensitive and selective clinical tests must be devised to determine the presence of low level laser injuries which may occur before ophthalmoscopically detectable lesions develop. Military ophthalmologists have a responsibility to investigate therapeutic modalities that will offer a better prognosis and a faster recovery from laser injuries. Furthermore, and equally important, we must advocate and support efforts to develop better protective eyewear to prevent these injuries.

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APPENDIX

APPENDIX

Summary of Accidental Laser Exposures

CASE (Ref)	LASER SPECIFICATIONS (RED_{50}/RMPE)	IMMEDIATE SUBJECTIVE EFFECT	TIME FROM INJURY FINDINGS ON FIRST EXAMINATION	TREATMENT	COURSE AND OUTCOME	GRADE OF INJURY
1 (5)	Pulsed Ruby 694.3 nm wavelength 0.8 msec pulse duration	unknown	20 minutes 10/200 dense central scotoma	none mentioned	6 weeks 20/200 pigmented macular scar	IB or IIB
2 (27)	Pulsed Ruby 694.3 nm wavelength 100 nsec pulse duration <100 mJ exposure (<10,000/<500,000)*	positive central scotoma reduced central vision	time unknown 6 degree positive central scotoma foveal edema retinal hemorrhage vitreous hemorrhage	none mentioned	2 months reduced central vision positive central scotoma pigmented macular scar	IVB
3 & 4 (15)	unknown	unknown	time unknown retinal burns in both eyes field defect in one eye	none mentioned	unknown	?
5 (18)	Pulsed Ruby 694.3 nm wavelength	"wind-like force" bright orange flash blurred vision	same day 20/20 3 degree absolute central scotoma macular edema foveal hemorrhage	topical corticosteroids & mydriatics	2 weeks 20/100 absolute scotoma macular hole 12 months no change	IVB
6 (23)	Q-switched Ruby Raman shifted to: 650 nm, 694.3 nm, & 746 nm wavelengths 1-3 mJ exposure (100-300/5,000-15,000)*	paracentral scotoma	19 hours 20/25-2 central field blurred macular lesion into fovea	80 units corticotropin intramuscular	27 days scar nasal to fovea 8 months 23 min paracentral scotoma	IA-IB

APPENDIX

Summary of Accidental Laser Exposures *(continued)*

CASE (Ref)	LASER SPECIFICATIONS (RED_{50}/RMPE)	IMMEDIATE SUBJECTIVE EFFECT	TIME FROM INJURY FINDINGS ON FIRST EXAMINATION	TREATMENT	COURSE AND OUTCOME	GRADE OF INJURY
7 (20)	Q-switched Ruby 20 nsec pulse duration Secondary Beam 694 nm wavelength 1 cm beam diameter 2 mJ per sq cm 377 uJ exposure (assuming 4 mm pupil size) (18.8/6,000)	central scotoma	one day 20/200 5 degree central scotoma foveal hemorrhage vitreous hemorrhage decreased foveal electro-retinogram (FER)	40 mg triamcinolone retrobulbar 400 ml dextran intravenous	3 months 20/50 central scotoma smaller retinal hole	IVB
8 (17)	Pulsed Neodymium YAG 1064 nm wavelength 500 um beam diameter 0.1 sec pulse duration 10 pulses per sec 15 mJ/pulse exposure (150/15,000)	heard a snap bright afterimage dense central scotoma	same day 20/300 absolute central scotoma foveal hemorrhage pre-retinal hemorrhage vitreous hemorrhage	none mentioned	9 days retinal hole	IVB
9 (17)	Q-switched Neodymium YAG 1064 nm wavelength 1.5-3 mm beam diameter 6 nsec pulse duration 10 pulses per sec (60/6000)	felt a "pop" & sudden pain blurred vision floaters photopsias	one day 20/20 Bjerrum scotoma retinal hole retinal hemorrhage vitreous hemorrhage	none mentioned	2 weeks vitreous blood cleared 3 months nerve fiber layer defect	IVA
10 (17)	Pulsed Neodymium YAG 1064 nm wavelength 2.5 mm beam diameter 20 nsec pulse duration 10 pulses per sec 1-2 mJ/pulse exposure (10-20/1000-2000)	decrease in VA central scotoma	one day 20/100 central scotoma foveal subretinal hemorrhage	none mentioned	5 days 20/50 3 weeks pigmented scar 6 weeks 20/25 4 months 20/20-3	IIIB

APPENDIX

Summary of Accidental Laser Exposures *(continued)*

CASE (Ref)	LASER SPECIFICATIONS (RED_{50}/RMPE)	IMMEDIATE SUBJECTIVE EFFECT	TIME FROM INJURY FINDINGS ON FIRST EXAMINATION	TREATMENT	COURSE AND OUTCOME	GRADE OF INJURY
11 (17)	CW Argon 488 nm & 514.5 nm wavelength 1.4 mm beam diameter 8–9 mJ exposure (5.7–6.4/56–62.5)*	paracentral visual blur	one day 20/20 1 degree paracentral scotoma parafoveal edema & necrosis	none mentioned	12 days 20/15–2 macular edema cleared 2 months field defect gone pigmented scar	IIA
12 (17)	CW Argon 488 nm & 514.5 nm wavelength <1 mm beam diameter 3–4 mJ exposure (2.5–6/25–30)*	visual blur scotoma	3 days 20/20–2 central blur on Amsler grid parafoveal retinal necrosis subretinal hemorrhage subretinal fluid macular edema	60 mg prednisone: orally for 5 days (started one week after injury)	one week 20/25 treatment started 11 days 20/15 edema cleared	IIIA
13 (17)	Pulsed Rhodamine Dye 592–594 nm wavelength 6 mm beam diameter 10 nsec pulse duration 10 pulses per sec 0.2 mJ/pulse exposure (37/1000)*	orange flash visual blur scotoma	one day 20/25 small central scotoma parafoveal edema & necrosis	none mentioned	8 days 20/20 edema clearing field defect smaller 2 weeks edema gone field defect same pigmented scar 3 1/2 months field defect noticeable only while testing	IIA
14 (17)	CW Krypton 647.1 nm & 674.2 nm wavelength	visual blur scotoma	one day 20/20 tangentially 1 degree paracentral scotoma parafoveal edema & necrosis	none mentioned	8 days edema cleared field defect 16 months field defect still present	IIA

APPENDIX

Summary of Accidental Laser Exposures *(continued)*

CASE (Ref)	LASER SPECIFICATIONS (RED_{50}/RMPE)	IMMEDIATE SUBJECTIVE EFFECT	TIME FROM INJURY FINDINGS ON FIRST EXAMINATION	TREATMENT	COURSE AND OUTCOME	GRADE OF INJURY
15 (6)	Q-switched Ruby 693 nm wavelength 80 nsec pulse duration 3.0 mJ exposure[22] (200/1500)	bright red flash central scotoma	8 hours 20/100 paracentral scotoma foveal hemorrhage foveal edema	osmolar, vitamin, & tissue therapy pyrogenal injection	3 weeks 20/50 scotoma smaller hemorrhage partially absorbed edema cleared 2 months 20/25 scotoma hardly noticeable atrophic scar	IIIB
16 (6)	Pulsed Ruby 693 nm wavelength 300 usec pulse duration 200 mJ exposure[22] (200/12,500)	sharp flash 1-2 min complete loss of vision return of peripheral vision central scotoma	24 hours 20/100 2-3 degree absolute central scotoma macular hemorrhage	corticosteroids osmolar & stimulation therapy	10 days scotoma gone pigmented scar	IIIB
17 (6)	Q-switched Neodymium 1064 nm wavelength 50 nsec pulse duration 5.0 mJ exposure[22] (50/5000)	very sharp flash central scotoma floaters	48 hours 20/100 3 degree absolute central scotoma macular hemorrhage macular hole vitreous hemorrhage	corticosteroids osmolar and stimulation therapy	16 days 20/70 1 degree absolute central scotoma 6 months vision unchanged pigmentation around hole	IVB
18 (6)	Pulsed Neodymium YAG 1064 nm wavelength 30 nsec pulse duration >3.0 mJ exposure[22] (30/3000)	unknown	8 days 20/50 to 20/70 8-10 degree relative central scotoma macular subretinal hemorrhage preretinal hemorrhage	corticosteroids osmolar & stimulation therapy	15 days 20/40 to 20/50 6-8 degree relative scotoma hemorrhage absorbed	IVA or IVB

APPENDIX

Summary of Accidental Laser Exposures *(continued)*

CASE (Ref)	LASER SPECIFICATIONS (RED_{50}/RMPE)	IMMEDIATE SUBJECTIVE EFFECT	TIME FROM INJURY FINDINGS ON FIRST EXAMINATION	TREATMENT	COURSE AND OUTCOME	GRADE OF INJURY
19 (8)	Pulsed Ruby 693 nm wavelength 25 nsec pulse duration right eye: 3.0 mJ exposure[22] (200/15,000)* left eye: 0.05 mJ exposure[22] (3.3/250)*	sudden severe impact bright pink flash sharp pain central scotoma floaters	1st day right eye: 20/200 left eye: 20/200 2nd day right eye: 20/200 foveal edema foveal hemorrhage left eye: 20/30 parafoveal edema	osmolar, vitamin, & stimulation therapy	3 months right eye: 20/200 8-10 degree absolute central scotoma foveal hole left eye: 20/20 absolute paracentral scotoma pigmented scar	right eye: IVB left eye: IA or IB
20 (21)	Pulsed Neodymium 1064 nm wavelength 30 nsec pulse duration 12 pulses per sec 1-2 MW/pulse 1.5 mm aperature 30-60 mJ/pulse exposure (300/30,000)*	green flash snapping sound central scotoma	3 hours 20/200 10 degree absolute central scotoma macular edema macular hemorrhage vitreous hemorrhage macular tear	local steroids, rest, hemostatic & anti-edema therapy	2 days 20/70 8 degree absolute central scotoma 1 year 20/100 scotoma unchanged	IIB
21 (7)	Pulsed Dye 589 nm wavelength 10 nsec pulse duration 1.2 cm beam diameter 1.66 mJ/Pulse exposure (332/8300)*	central scotoma	immediately CF x 3 feet 5 degree central scotoma foveal hole vitreous hemorrhage	systemic corticosteroids	5 days 20/100 <5 degree central scotoma 12 months vision unchanged	IIB

APPENDIX

Summary of Accidental Laser Exposures *(concluded)*

CASE (Ref)	LASER SPECIFICATIONS (RED_{50}/RMPE)	IMMEDIATE SUBJECTIVE EFFECT	TIME FROM INJURY FINDINGS ON FIRST EXAMINATION	TREATMENT	COURSE AND OUTCOME	GRADE OF INJURY
22 (11)	Q-switched Neodymium YAG 1064 nm wavelength 20 nsec pulse duration 20 mJ/cm² beam energy 4.3 mJ/pulse exposure (assuming a 7 mm pupil) (43/4, 300)	immense flash black circles almost complete loss of vision	8 days 20/200 slowly paracentral arcuate scotoma parafoveal burn vitreous hemorrhage subretinal hemorrhage retinal hemorrhage subhyaloid hemorrhage	none mentioned	3 weeks 20/25 arcuate scotoma parafoveal pucker vitreous hemorrhage absorbed 1 month 20/50 foveal pucker 1 year vision unchanged	IVA
23 (19)	Pulsed Dye 420 nm wavelength rated at 200 KW 6 nsec pulse duration 100 um beam diameter	loss of central vision	time unknown dense central scotoma foveal burn foveal subretinal hemorrhage	prednisone: 60 mg orally/daily tapered over 10 days	3 months 20/40 paracentral scotoma hemorrhage absorbed 5 months 20/25 scotoma gone	IIIB

*Lund DJ, Stuck BE, personal communication, 1993, where RFD_{50} = E/ED50 and RMPE = E/MPE where E is the estimated dose received by the accident victim.

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