



Strål  
säkerhets  
myndigheten

Swedish Radiation Safety Authority

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Research

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Laser pointers and Eye injuries

An analysis of reported cases



## **SSM perspective**

### **Background**

The safety limits that exist for human exposure of laser radiation are essential to reduce the risk of injuries. These values, however, give very little information on what tissue damages that may be expected at various elevated exposure levels. Similarly, the Swedish Radiation Protection Authority (SSM) has very little information on how such tissue damage is related to the impairment of the vision. This type of relationship between an imaginary exposure and a subsequent disability is very useful in the risk assessments that are made in the authority's supervision activities. Also, the damage's evolution over time is information that the authority can make use of in risk assessments.

### **Objectives**

The purpose of this study was to investigate what dose of laser radiation, in terms of intensity and exposure time, may be associated with eye damages. The study has been limited to unwanted exposures of laser radiation from commercially available laser pointers. Of particular interest has been to search for data that clarify the dose-response relationships for functional disabilities that persist more than 6 months.

### **Results**

The study shows that long-term vision loss can occur as a result of involuntary exposure from commercially available (strong) laser pointers at close range. The injury may occur before a normal person is able to respond by closing the eyelid, although there are only a few cases reported. A minor such damage is transient within a few days. It is also likely that such a visible injury to the retina becomes functional, i.e. prevents reading skills. What dosage is required for the disability to become permanent is not clear in the literature. Also, the dynamics of evolution and repair of tissue damages and disabilities are hardly described at all.

### **Need for further research**

The importance and need of a database on laser incidents has been evident during this work. There is also a need for further research on dynamics, treatment of laser damage, long-term permanent laser damage, and on the effect of visual aids and refractive errors in laser pointer retinal damage. Finally, an essential area of research is the development of methods to identify functional visual deficit in the presence of structural retinal damage.

### **Project information**

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This report concerns a study which has been conducted for the Swedish Radiation Safety Authority, SSM. The conclusions and viewpoints presented in the report are those of the author/authors and do not necessarily coincide with those of the SSM.

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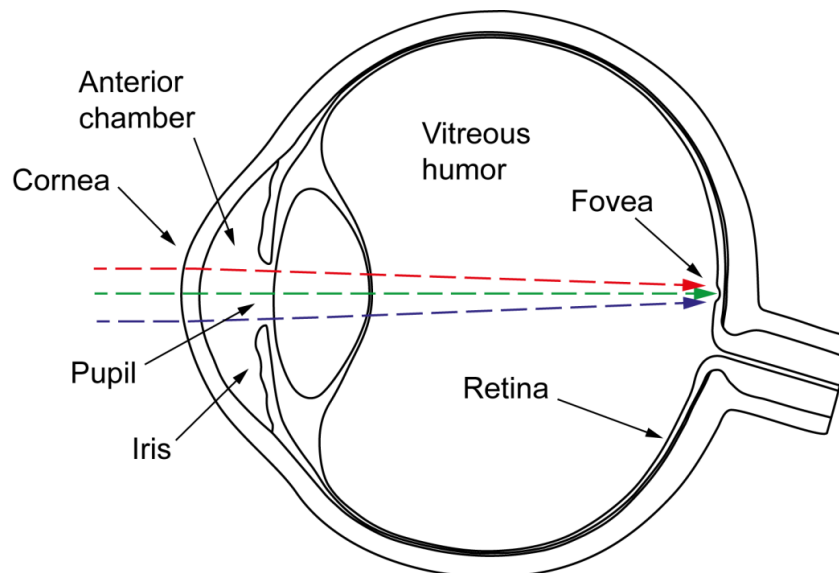


# 1. Introduction

The use and misuse of handheld lasers, also called laser pointers, has increased dramatically in recent years. The laser pointers have become strong enough to dazzle eyes from distances of more than 10 km and are, at shorter distances, capable of damaging the retina [Sloney & Wolbarsht 1980]. The temporary blinding effect of laser pointer exposure can lead to deleterious secondary effects for pilots and drivers. Consequently there is an increasing need to educate the general population about the potentially dangerous illumination from laser pointers. In this study we analyze published intentional and unintentional non-military exposures to laser pointers.

The eye is the most vulnerable part of the body to intense light. The interaction of radiation within the eye is dependent on the wavelength of the radiation:

- Very short-wave and very long-wave radiation is not a specific eye health problem for most people.
- Ultraviolet radiation and most of the longer infrared radiation wavelengths (infrared radiation-B and above) are absorbed in the anterior parts of the eye.
- Visible wavelengths and wavelengths in the infrared radiation-A, are focused by the optics of the eye (the cornea, the aqueous humor, the lens and the vitreous humor) to a small spot on the retina.



**Figure 1.1** The human eye. Only visible and parts of infrared radiation are transmitted and focused on the retina and can damage the retina. Illustration by J Thaug.

The focusing by the eye optics to a spot size of 25  $\mu\text{m}$  on the retina concentrates the energy by a factor of approximately 100 000 [Sloney & Wolbarsht 1980]. This is the major reason why the retina can be harmed by even relatively low laser energies. Besides laser pointers,

other light sources such as sunlight or intense artificial visible light can also cause permanent retinal damage [Cole 2005, Rothkoff *et al.* 1978].

The eye pupil regulates the amount of light that enters the eye. The pupil size changes depending on the ambient light levels. In low light the size of the pupil can be up to 8 mm in diameter compared to 1.5-2 mm in daylight (bright light) [Henderson & Schulmeister 2004]. Thus, pupil size is important for the vulnerability of the retina. An important protective part of the eye is the eyelid. The eyelid functions as an optical shutter and can close in approximately 0.16 seconds after exposure to strong light. In the safety standards this time is set to 0.25 seconds [ANSI Z136.1].

The limits of this study are continuous wave laser pointer exposures in the waveband 400-1400 nm, with exposure duration from 0.05 to 1 second. Initially, a major inclusion criterion was permanent structural or functional retinal damage but due to scant number of such documented cases we have included cases with temporary retinal damage. Secondary effects, that is, other damages than retinal (caused by laser illumination), are not included in the study.

The purpose of this study is to increase the knowledge about retinal damage from commercial non-military laser pointers. This information is used to highlight areas where there is a need for further research. The study can aid the regulating bodies in establishing a better basis for protective measures against laser pointer exposures, and to provide a background for risk assessments of permanent functional visual impairment.

Lasers are classified according to their hazardous potential to the eye or skin, roughly corresponding to intensity output and laser beam characteristics [IEC 60825-1].

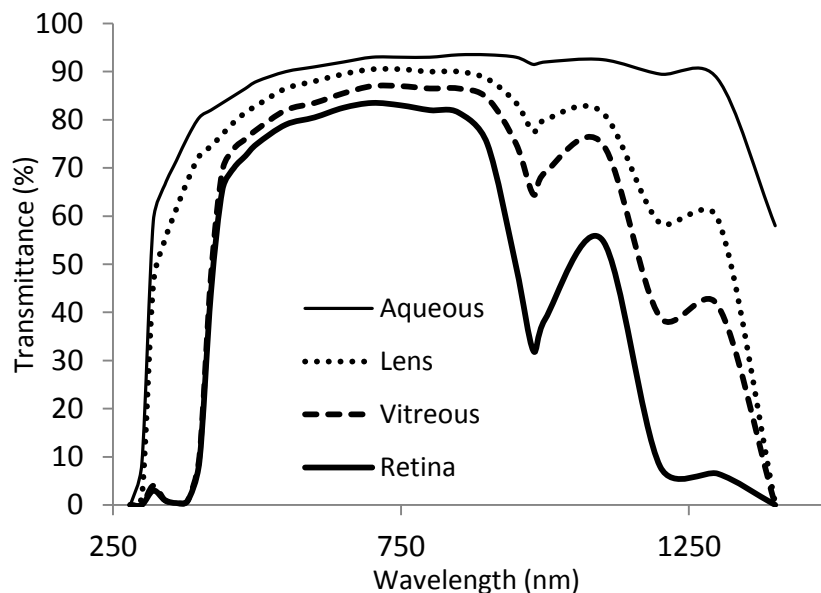
- A Class 1 laser is considered safe under all conditions of normal use.
- A Class 1M laser is safe for all conditions of normal use except when viewed by magnifying optics such as microscopes and telescopes.
- A Class 2 laser emits visible light and eye protection is normally afforded by the blink reflex and other aversion responses.
- A Class 2M laser is considered safe because of the blink reflex unless viewed through optical instruments.
- A Class 3R laser system is potentially hazardous under some direct and specular reflection viewing conditions if the eye is appropriately focused and stable, but the probability of an actual injury is small.
- A Class 3B laser system may be hazardous under direct and specular reflection viewing conditions, but diffuse reflections such as those from paper or textiles are not harmful.
- A Class 4 laser can burn the skin, or cause devastating and permanent eye damage as a result of direct, diffuse or indirect beam viewing. Besides eye damage, a Class 4 laser can ignite combustible solids and liquids.

## 2. Human eye structure and function

The eye has two transparent refractive components, the cornea and the lens, that collect and focus image information onto the retina (see figure 1.1), the light sensitive membrane in the back of the eye. The retinal nerve cells are in turn sending the image information back to the brain. The eye aperture, the pupil, regulates the influx of light onto the lens and retina. The visual sensory organ within the eye is the retina with its several layers of neuronal cells, including the photoreceptor cells cones and rods.

The part of the electromagnetic spectrum relevant for visual function is light, the waveband in between ultraviolet and infrared radiation. All these three wavebands taken together are sometimes called optical radiation. Light is defined as the radiation waveband that is visible for humans, specified as 400-780 nm in the ISO standard, although children might see some ultraviolet radiation. In contrast, the laser safety standards are using the CIE (The International Commission on Lighting) definition of light because the light waveband that elicits an aversion reflex at bright exposure is 400-700 nm, and not the ISO defined 400-780 nm.

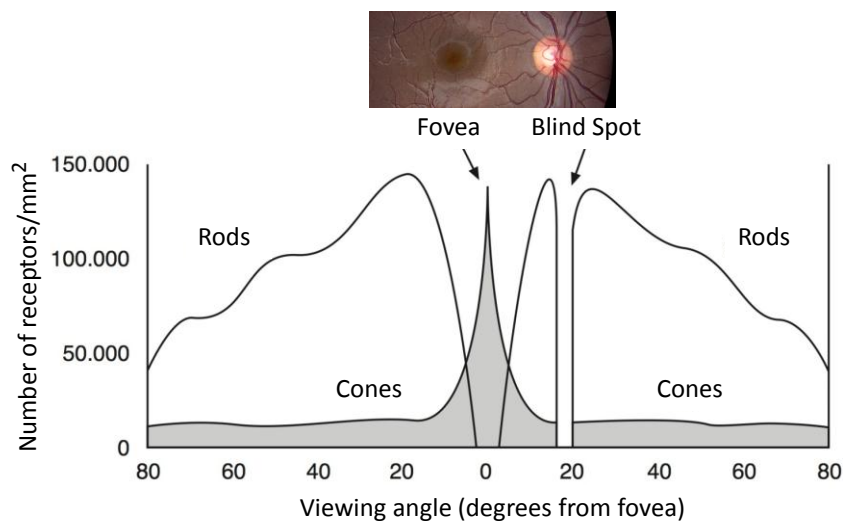
Ultraviolet radiation is mainly absorbed by the cornea, with the lens essentially filtering out the rest. The transmittance of the visible wavelengths is high in the eye optical system, to allow for a good visual function.



**Figure 2.1.** Percentage radiation incident at each tissue level, with 100% at corneal level [Adapted from Boettner & Wolter 1962].

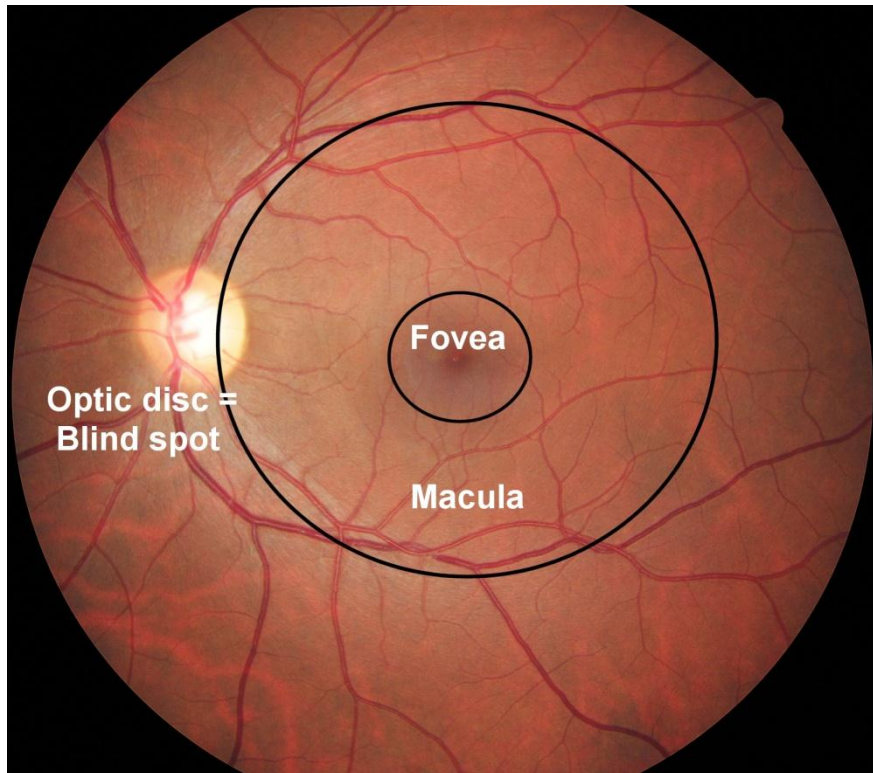
Figure 2.1 displays an example of so called total transmission, both direct and scattered, of optical radiation at various depths of the human ocular media. The invisible infrared radiation in the range 700 to 1400 nm (CIE infrared A) is partially transmitted to the retina while longer infrared wavelengths are mainly blocked by the water content in the ocular media, predominantly the cornea. The 400-1400 nm transmitted part of the optical radiation is called the retinal hazard region. From figure 2.1 it is clear that the ocular media protect the retina from the more harmful parts of solar optical radiation.

The optics of the human eye serves two main functions, to focus image information onto the retina, and to protect the inner parts of the eye from dangerous parts of optical radiation. The optics determines how optical radiation propagates within the eye, either transmitted or dissipated by scatter and absorption. There are two types of photoreceptor cells in the retina, cones and rods. Cones are responsible for high resolution vision and color perception. Main functions of the rods are night vision and movement perception in the peripheral visual field. The distribution of rods and cones is shown in figure 2.2.



**Figure 2.2.** Density distribution of retinal rods and cones. A retinal image is shown above the density plot. The scale and positions of the fovea and blind spot are aligned to match the density plot. Illustration by J Thaug.

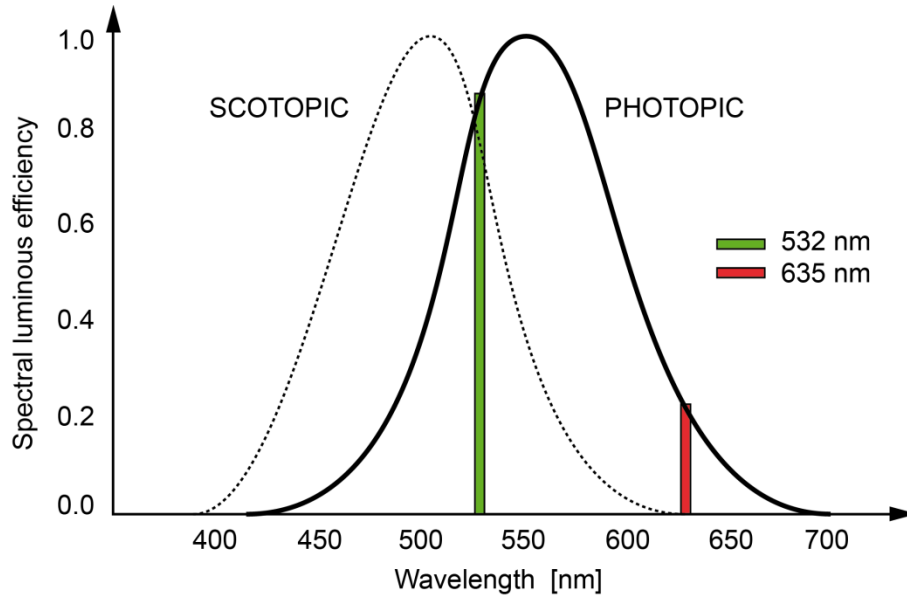
The highly resolved visual acuity in the centermost part of the visual field is explained structurally by the high density of cones within the fovea, the 1.5 mm central part of the macula, see figures 2.2 and 2.3. The rod distribution in the central retina is almost reciprocal to that of the cones. The visual angle corresponding to the fovea is approximately the size of the moon, or the size of a thumbnail at arm's length. Ophthalmologists often define the macula as the area surrounded by the temporal retinal arcade blood vessels and the optic disc, see figure 2.3.



**Figure 2.3.** The central parts of the retina. Photo by Eva Tov, St. Erik's Eye Hospital.

The optics of the eye permits sunlight to enter the eye without imminent risk of damage. This situation will change if a focusing device is used when viewing a light source, if the unaided eye is exposed to high intensity light, or if the eye is exposed to invisible radiation with sufficient intensity. In such cases there is a risk that the retina might be injured.

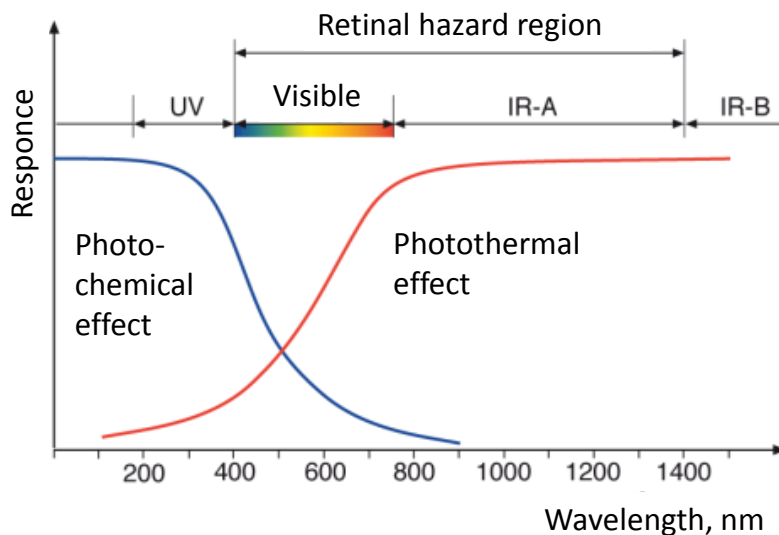
The eye adapts to various degrees of lighting and the two extremes are light and dark adaptation, also called photopic and scotopic vision (see figure 2.4), with an intermediate state called mesopic vision. The visual response in the retina peaks in the green, at around 555 nm. The apparent brightness of light wavelengths below and above the green region is lower, as shown in figure 2.4. This is highly relevant in laser exposures because a blue or red laser emitting the same intensity as a green laser will not be seen as bright as the green laser. This reduces the relative efficiency of the aversion responses for blue and red lasers. Figure 2.4 exemplifies this with the two peaks in red and green from lasers *with the same radiometric intensity*, but the green laser (532 nm) would be perceived as four times stronger than the red (635 nm).



**Figure 2.4.** Brightness action spectrum at scotopic (dark adapted) and photopic (light adapted) conditions. The green and red peaks exemplify two common laser pointer wavelengths, from two lasers emitting the same energy. Illustration by J Thaug.

### 3. Laser pointer damage mechanisms

There are three main mechanisms for laser damage to the retina: photochemical, photochemical and photomechanical. The latter is not covered in detail in this report because continuous wave laser pointers do not induce photomechanical effects in the duration 0.05 to 1 second. In principal, photochemical effects are induced by ultraviolet radiation and visible radiation in the low to mid-range waveband, while photo-thermal effects gradually increase throughout the visible range and become the sole mechanism in the infrared region, see figure 3.1. This is a simplification but gives an idea of the wavelength-dependency for the damage mechanisms. Short exposure duration or pulse length is important for photomechanical effects to occur.



**Figure 3.1.** Proportion of photochemical versus photothermal effects in the retinal hazard region (visible and infrared-A). Illustration by J Thuang.

The waveband region where both mechanisms occur is covered in the safety standards by dual exposure limit formulas. The safety standards view only the photothermal mechanism as important for continuous wave laser exposures less than 1 s in the 400-1400 nm region, while photochemical effects are becoming more prominent at longer exposure durations. This does not exclude photochemical effects at shorter exposure, but there is insufficient data to state that photochemical effects contribute significantly at shorter exposures. Photochemical effects at exposures shorter than 10 s are, in the standards, viewed as self-healing or non-damaging, although there is a lack of data to prove this to certainty.

Laser radiation energy that is transmitted through the ocular media will fall onto the retina and induce various types of energy trans-

fer. Photochemical damaging effects occur even when the rate of energy transfer is less than what is required for a temperature increase. The complexity of the photochemical damage mechanisms is not fully understood, nor are the repair mechanisms. Photochemical damage is mediated by the generation of photooxidative molecules such as free radicals and other reactive oxygen and non-oxygen species. These disturb or disrupt the chemical properties of adjacent molecules and structures, leading to cell death if the antioxidative biochemical protective systems become overloaded. If the energy transfer rate is even higher, the energy dissipating systems fail and a harmful thermal buildup develops. Finally, if the energy deposition rate is extremely high such as with ultrashort laser pulses, photomechanical, or photodisruptive effects results. The rate of energy transfer depends on the intensity and exposure duration of the laser, and size of irradiated area on the retina.

Laser pointers do commonly not emit ultraviolet radiation. Although ultraviolet radiation can damage the anterior eye tissues it is not relevant for this report. In contrast, invisible infrared radiation is often present together with visible light output from laser pointers [Galang *et al.* 2010], thus increasing the potential harmful effects to the eye. The bulk of the light and infrared radiation that is transmitted through the ocular media to the retina is ultimately absorbed in the various subtissues of the retina.

The photomechanical, photothermal and photochemical effects from lasers are applied every day in eye clinics, for treatment of eye diseases ranging from blood vessel related as in retinopathy of prematurity and diabetic retinopathy (see figure 3.2), to tumors and retinal tears. The photothermal mechanism dominates but in special cases the photochemical properties of laser treatment can be utilized to target laser energy deposition to a specific structure, such as a tumor. In photodynamic treatment a pigment molecule, a photosensitizer, is injected into the blood stream and subsequently concentrated in the targeted blood vessel rich tumor, which in turn is exposed to a laser wavelength with high absorption by the pigment. The absorbed laser energy induces a cascade of photooxidative effects, ultimately leading to tumor cell death. Photomechanical effects are used when disruptive properties are needed, for example when treating secondary cataracts, or high intraocular pressure due to pupil blockage of the aqueous humor.





**Figure 3.2.** Laser treatment in diabetes retinopathy, multiple spots in the periphery, sparing the macula. Photo by NEI, USA.

The retina contains various types of radiation-absorbing molecules and structures. Three major pigments for photothermal energy absorption are melanin in the retinal pigment epithelium and the choroid; hemoglobin in the blood vessels; and xanthophyll in the neuroretina. Early signs of photothermal damage in the retina are seen histologically in the pigment epithelium and in the photoreceptors [Green & Robertson 1991, Marshall *et al.* 1975]. Noell *et al.* found during the sixties that ambient light could damage retinas in rats, with a mechanism that was neither photothermal nor photomechanical. This photochemical damage mechanism is dependent on transfer of photic energy to cellular and subcellular structures and pigments [Noell *et al.* 1966]. Common pigments and structures for photochemical energy transfer are lipofuscin, hemoproteins, melanosomes and flavoproteins [Foote 1968, Glickman 2002, Solley & Sternberg Jr 1999]. Photochemical reactions are difficult to study because there is a cascade of short-lived and long-lived reactions where almost all molecules can participate in photochemical energy transfer.

The acute stage in retinal damage is edema in the retinal layers. This is visible as a grayish-white spot that, depending on the laser energy, can appear immediately or develop over several days. The repair stage follows coming weeks, with resolution of the edema. Unrepaired cell damage ultimately leads to cell death, which is seen as hyper- and hypopigmented spots, or scars [Lavyel 1963].

Beneath the neuroretina is a layer of dark pigmented cells, the retinal pigment epithelium, which besides the biochemical supportive function for the photoreceptors also functions as a light sink, removing surplus light within the eye. If the pigment epithelium and underlying Bruch's membrane is compromised, there is a risk of vision-threatening neovascularization below or within the retinal layers. If the laser is

strong enough and hits a blood vessel, a hemorrhage can occur. The vessel will heal and the blood will be resorbed with time but scar formation might follow from larger hemorrhages. Scars in the retinal layers can lead to a detachment of the retina, a dangerous situation for the eye, requiring surgery. Today it is well known that supra-threshold laser treatment can lead creeping, a confluence of individual laser spots into larger scar formations. An example of this is seen in figure 3.3.



**Figure 3.3.** Several confluent laser atrophies, after creeping of laser scarring after supra-threshold laser therapy in the 1980s. Photo by E Tov, St. Erik's Eye Hospital.

If a laser hits the fovea, the central most part of the retina responsible for detailed vision, unrepaired cell damage will lead to cell death, scar formation and permanent reduction of the central visual function. The other critical area in the retina is the optic disc, which is comprised by nerve fibers from the retinal nerve cells transmitting sensory signals to the brain. Damage to the optic disc can have deleterious effects on the visual function. On the other hand, a laser spot damage in any other part of the retina than the fovea and the optic disc will likely not cause any residual observable visual disturbance for the exposed person, as long as no secondary effects occur.

Laser exposure often scares the exposed person who might have read in the newspapers about the dangers of lasers. Non-retinal symptoms and signs are common, such as corneal erosions, red eyes and swollen eyelids. These are caused by rubbing of the eyes, a common phenomenon when someone experiences acute eye discomfort. Other common symptoms are headache, scotoma, burning sensation in

the face, pain within the eye, and floaters. Many of these can hardly be explained by the laser itself, but a few might be caused by post-exposure anxiety.

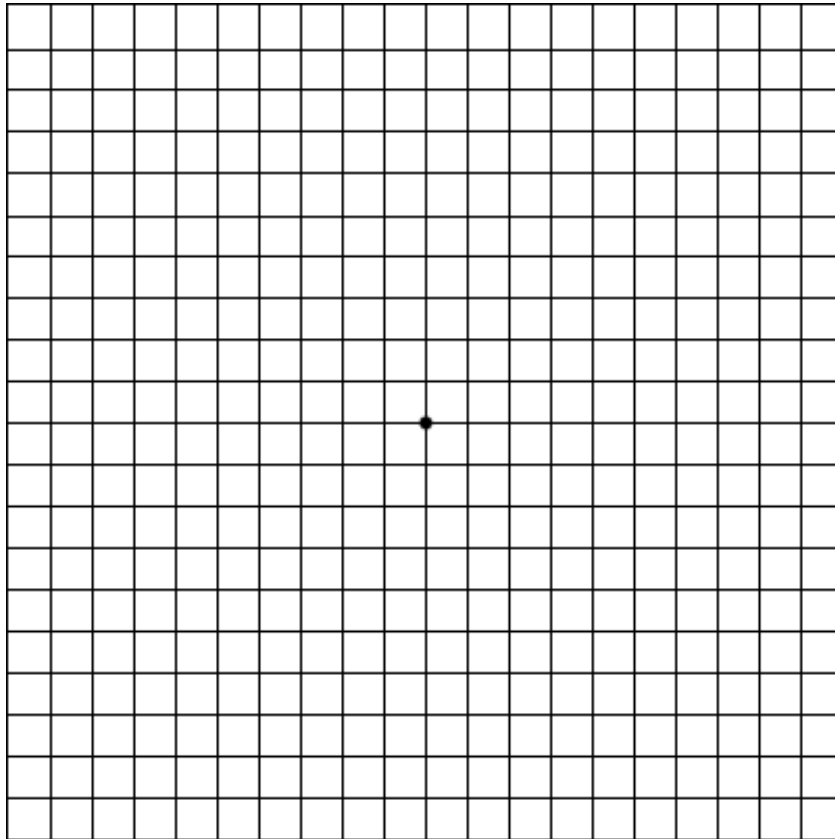
Edema is a general tissue response to trauma and disease in the retina. The various types of non-laser retinal edema presenting in the ophthalmic clinic are treated with laser or pharmaceuticals. To the best of our knowledge there have been no controlled treatment trials in cases of laser-induced retinal edema. There are cases where anti-inflammatory drugs have been tried, but without sufficient sample size or untreated controls the treatment effects cannot be fully validated.

### **3.1 Diagnostic tools for identification of retinal laser damage**

The most common and easiest method for identification of laser retinal damage is by visual examination, so called ophthalmoscopy. This is performed with a microscope or a handheld device. Documentation can be made by photography. High dose laser effects induce whitish-gray spots on the retina, clearly visible to the examiner. However, laser exposures of less intensity do not induce visible spots but there can still be significant damage, requiring other methods for identification. Examples of other commonly available methods to identify structural retinal damage are optical coherence tomography (OCT), which is similar to a CT scan although the image acquisition is made with light and not x-rays; angiography (identifies blood vessel damage); autofluorescence (photographic method to visualize certain chemical signals); scanning laser ophthalmoscopy (SLO); and eye movement recordings. Histology (sectioning of eye tissue) and cellular/subcellular analyzes can identify a wide range of cell and tissue damage but requires removal of the eye. In recent years a new technique called adaptive optics (AO) is now enhancing microscopes, OCTs and other devices for retinal imaging. With the help of AO much smaller structures can be observed.

Unfortunately there is a rather poor correlation between visible retinal damage and functional deficit, although the advances in SLO microperimetry have improved this correlation. Furthermore, even healthy retinas in asymptomatic people, never exposed to laser radiation, exhibit small numbers of visible abnormalities. Structural tests are a necessary first step in the process of verifying retinal injury after laser exposure. Any abnormality thus identified has to be followed over time to verify the damage and repair dynamics. Secondly, complementary functional tests, or psychophysical tests, are required for proper determination of functional deficit. Examples of these tests are visual acuity, reading acuity, computed perimetry (identification of small visual defects in the visual field), contrast acuity, color function, and metamorphopsia grids such as the Amsler card (a simple self-assessment method) (see figure 3.1.1). The difficulties lie in the fact that psychophysical

tests require cooperative responses from the subject, in reality restricting the use of these tests to humans.



**Figure 3.1.1.** Amsler grid card [Amsler 1953], primarily used by patients with macular degeneration, but also used as a self-assessment tool after unintentional exposure to laser radiation [Alesa card].

It is very important to remember that one single retinal injury can have a huge impact on visual function if the injury is located in the fovea or the optic disc. If the injury is permanent, the visual deficit will also be permanent. In contrast, if a similar permanent retinal injury occurs elsewhere in the retina, the person would probably not have any problems at all, providing no secondary effects occur.

## 4. Reported retinal injuries from laser pointers

The easy access to high powered laser pointers in recent years has led to many attacks amongst the civilian population. Only a small fraction of the attacks has so far led to injuries, but with increasing energy of commercial laser pointers the severity of eye injuries is expected to increase.

In this chapter we review medical case histories of retinal injuries caused by commercial laser pointers. The commercial laser pointers can emit continuous wave energies from 1 mW to hundreds (or thousands) mW. The output wavelengths can be in the range 400 to 1400 nm. The wavelengths in the infrared-A are fundamentals that are not properly shielded and therefore emitted together with the visible wavelengths [Galang *et al.* 2010]. Since these invisible wavelengths also are focused on the retina, they can cause an injury. The medical cases range from children that gazed a prolonged time into a laser beam to pranks and illumination of aircraft cockpits.

USA has been the most progressive country regarding laser incident reports [Harrington & Wigle 2004]. But the reported civilian cases involving handheld lasers lack full documentation or in early days were dismissed [Mensah *et al.* 1998]. This was due to many unknown factors related to the cases, such as:

- Reliable information from the victim
- Identification of the laser
- The distance to the laser
- The divergence of the laser beam
- A medical evaluation of the injury by ophthalmic professionals familiar with retinal laser injuries.

The reason for this is because many of the cases involve outdoor laser illumination in which there are apparent difficulties in collecting all relevant information. The most reliable source of information is the military database “The U.S. Army’s Laser Accident and Incident Registry” [Ness *et al.* 1997, Johnson *et al.* 2003]. Most of the cases, however, involve pulsed lasers and are therefore not relevant for this report. The database is primarily available to US military researchers.

The cases included in this report are all found in the open literature. The reported case list is unfortunately incomplete because we have not been able to search within non-English literature and we could not get access to US Federal Aviation Authority data. There are more than 16 000 reported incidents solely against air traffic since 2004. Of these 16 000 cases, 1.5% (approximately 240 cases) led to adverse eye effects [Murphy 2013]. Further, some cases found in the literature are inadequately described [Case 2000, Elias 2005].

## 4.1 Case histories

*Case 1 – A 34 year old man deliberately gazed into a red laser beam*  
[Luttrull & Hallisey 1999]

Laser power	Laser wavelength	Distance	Exposure duration
5 mW	670 nm	20-25 cm	30-60 s

Year	Examination time frame	Symptom/injury	Remarks
1999	First: After 2 days Last: Unknown	Red central scotoma in the left eye and headache	The effects disappeared the day after the exposure

The injury was self-resolved and the visual acuity (VA) remained normal at 20/20 (US), 6/6 (UK), 1.0 (metric).

Normal VA is in the region of 1.0-1.3, depending on age [Frisén & Frisén 1981]. This notwithstanding, many people judge their vision as normal even with VA as low as below 0.5, which is the required VA for a driver's license in Sweden. Self-reported vision is thus not a suitable parameter.

*Case 2 – An 11 year old girl deliberately gazed into a red laser beam*  
[Sell & Bryan 1999]

Laser power	Laser wavelength	Distance	Exposure duration
5 mW	670 nm	5-25 cm (unknown)	several seconds

Year	Examination time frame	Symptom/injury	Remarks
1999	First: After 3 weeks Last: After 11 months	Decreased vision and central scotoma in the right eye	After 6 months the vision was 0.8 in the right eye.

Three weeks later the best corrected visual acuity (BCVA) was 0.33 in the right eye and 0.8 in the left. Three months after the injury, the BCVA improved to 0.8 in the right eye, with a small relative central scotoma. 11 months after the injury, the visual BCVA in the right eye remained at 0.8. The patient reported normal vision with no relative scotoma.

**Case 3 – A 19 year old woman deliberately gazed into a red laser beam** [Zamir & Chowers 1999]

Laser power	Laser wavelength	Distance	Exposure duration
1 mW	670 nm	10 cm	10 s

Year	Examination time frame	Type of injury	Remarks
1999	First: Two weeks after exposure. Last: Three months later.	Two small pericentral scotoma and hypopigmented ring-shaped lesion in the fovea of the right eye.	After 8 weeks vision returned to normal.

After three months the woman still experienced a relative decrease in brightness of objects (the right eye) and the abnormality of retinal pigment epithelium persisted.

**Case 4 – A young male exposed to a laser at a rock concert** [Peterson 2011]

Laser power	Laser wavelength	Distance	Exposure duration
Class 3A (3R), <5 mW	Unknown	Unknown	Unknown

Year	Date of first examination	Symptom/injury	Remarks
2011	First: Same day Last: 2 weeks	Discrete foveal abnormality with subretinal edema	Foveal anatomy improved within 2 weeks

The patient had difficulties in reading two weeks after the exposure. The symptoms improved over time. No VA data was given.

**Case 5 – An 11 year old male deliberately gazed into a green laser beam** [Fujinami *et al.* 2010]

Laser power	Laser wavelength	Distance	Exposure duration
<5mW	532 nm	30 cm	Every day for approximately 10 seconds over a period of approximately 9 years

Date of exposure	Examination time frame	Symptom/injury	Remarks
2001	First: 9 years later Last: 12 years later	Choroidal neovascularization	The injury was caused by repeated laser exposure

A yellow exudate-like or fibrous tissue was found in his right eye, surrounded by subretinal hemorrhage in the macula. Two years later there was a yellow fibrous lesion in the macula, which was leaking at angiography. The pre-exposure VA at 7 years of age 1.0 in both eyes. At 11 years of age his BCVA was 0.2 in the right eye and 1.0 in the left. At 14 years of age neither his BCVA nor the fibrous tissue had improved.

**Cases 6-19 – A survey of 14 people** [Sethi *et al.* 1999]

In 1999 a survey was done at the Eye Hospital in Bristol on 14 people who were exposed to presumably class I, II or 3A green laser radiation. The table below gives the known data for the 14 patients.

Laser power	Laser wavelength	Distance	Exposure duration
1 - 5mW	Unknown	Unknown	Unknown

Year	Examination time frame	Symptom/injury	Remarks
1999	First: 3 h to 30 days Last: Mean 10.5 months	Corneal epitheliopathy, abnormal foveal reflex, perifoveal drusen	No persisting afterimage, scotoma or visual field effects.

In two cases both eyes were hit by the laser beam. The patients were contacted, by phone, again after 10.5 months. Three patients still had some ocular discomfort; two had to wear new refractive corrections and one had persisting symptoms of intermittent ocular discomfort. VA changes for four of the patients were reported. Neither of these symptoms was proven to be linked to the laser exposure.

**Case 20 – A 16 year old male was exposed by friends to a red laser beam** [Israeli *et al.* 2000]

Laser power	Laser wavelength	Distance	Exposure duration
<5mW	670 nm	~1 m	~20 seconds

Year	Examination time frame	Symptom/injury	Remarks
2000	First: After 3 days Last: After 8 months	Blurred vision bilaterally with a red central scotoma.	Both eyes were exposed. The symptoms were resolved spontaneously after two days.

Even though the symptoms disappeared after two days and his vision returned to normal, after 8 months he still had permanent retinal pigment epithelial disturbances in both eyes near the fovea.



**Case 21-23 – Experimental laser pointer exposure to three patients**

[Robertson *et al.* 2000]

The three patients had uveal melanomas and enucleation (=eye removal) were planned.

Laser power	Laser wavelength	Distance	Exposure duration
~1 mW (patient 1, 62 year old male)	673 nm (patient 1)	15 cm	1 minute (fovea)
~2 mW (patient 2, 36 year old woman)	673 nm (patient 2)		5 minutes (5° below fixation)
~6 mW (patient 3, 59 year old woman)	659 nm (patient 3)		15 minutes (5° above fixation)

Year	Examination time frame	Symptom/injury	Remarks
2000	Patient 1. First: Immediately Last: After 11 days Patient 2. First: Immediately. Last: After 86 h. Patient 3. First: Immediately. Last: After 15 days	No injury	Patient 1. Several small drusen. VA 0.8 right eye and 1.0 left eye. Patients 2 and 3. Abnormalities in the outer retina and the pigment epithelium. VA remained at 1.0 both eyes after the exposure.

Patient 1 had no visual defects. Patient 2 recognized that her vision was somewhat pink for a few minutes after the exposure but otherwise no visual defects. In patient 3 the pink vision was, after 2 minutes, concentrated into a pink circular afterimage. After 5 minutes the vision was normal again. Even though this study was well conducted the conclusions drawn by the authors, “the potential for laser pointers to cause eye damage has been exaggerated” and “is negligible”, were rejected by others as “premature and potentially misleading” [Zamir & Chowers 2001, McGee *et al.* 2000]. There is thus a disagreement between researchers [Ajudua & Mello 2007].

**Case 24 – Experimental laser pointer exposure to a patient** [Robertson *et al.* 2005]

A similar study as in cases 21-23 was conducted with a green laser pointer instead of a red laser pointer, on a 55 year old woman.

Laser power	Laser wavelength	Distance	Exposure duration
~3-7 mW	532 nm	15 cm	1 min (fovea) 5 min (5° below fixation) 15 min (5° above fixation)

Year	Examination time frame	Symptom/injury	Remarks
2003	First: Immediately Last: After 20 days	Retinopathy, yellowish discoloration of RPE, 24 h after exposure.	After 5 days granular changes was seen in the retina. VA was 1.0 before exposure and the same after 20 days.

There is thus a difference between a red laser pointer and a green laser pointer regarding the vulnerability of the retina. In cases 21-23 the red laser, under the same conditions, did not cause any injury. In this case however, with a green laser pointer, an injury occurred within 60 seconds.

**Case 25 – Laser illumination of aircrafts** [Nakagawara *et al.* 2008]

Between January 1, 2004 and January 31, 2005 there were a total of 90 (53 involved commercial aircrafts) laser illuminations against aircrafts in USA. In 41 cases the cockpit was illuminated which resulted in 13 cases of “distraction or visual impairment to a pilot”. One case resulted in an injury to the retina.

Laser power	Laser wavelength	Distance	Exposure duration
<5mW	532 nm	Unknown	~5 seconds

Year	Examination time frame	Symptom/injury	Remarks
2004	First: After one day Last: Unknown	Retinal burn.	Sensitivity to bright lights for a period of time. Unable to pilot aircrafts for 3 weeks.

**Case 26 – A 22 year old female was accidentally exposed to a green laser** [Sun *et al.* 2006]

The female was setting up a stage when she accidentally looked into the beam and her right eye was hit.

Laser power	Laser wavelength	Distance	Exposure duration
> 20 mW	532 nm	~1 m (probably)	1 second

Year	Examination time frame	Symptom/injury	Remarks
2005	First: After 5 days Last: After 8 months	Lesion + exudation after 5 days. Enlarged lesion and hemorrhage after 8 months.	Classic choroidal neovascularization in the subfovea.

After 12 months the lesion was unchanged but the hemorrhage had diminished. Her BCVA was 0.08 in the right eye and 1.2 in the left 5 days after the exposure. Her BCVA before the accident was 1.2 in both eyes. The BCVA in the right eye continued to worsen, but improved slightly to 0.01 in the right eye at 8 months.

***Case 27 – A 38 year old male was accidentally exposed to infrared radiation-A, 825-880 nm, laser in his right eye [Wong et al. 2007]***

Laser power	Laser wavelength	Distance	Exposure duration
< 5 mW	825-880 nm	~1-3 m (unknown)	1-2 seconds (twice)

Year	Examination time frame	Symptom/injury	Remarks
2005	First: Unknown Last: After 2 months	Raised macula and pale lesion nasal to the fovea. Retinal detachment	Retinal detachment was unresolved 2 months later (follow-up).

His BCVA after the exposure were 0.5 in the right eye and 1.0 in the left eye. No data is given on BCVA at the two month follow-up. This is probably the first human case regarding infrared radiation-A laser. It is surprising that retinal detachment occurred at such low laser energy. One might speculate that the reported exposure duration was underestimated.

***Case 28-30 – A male (26 years old) and a female (21 years old) were accidentally exposed to a green laser during a laser show + An experimental laser pointer exposure to a patient [Boosten et al. 2011]***

The male was hit in his left eye and the female in her right eye.

Laser power	Laser wavelength	Distance	Exposure duration
Unknown	532 nm	Unknown	Unknown

Year	Examination time frame	Symptom/injury	Remarks
2009	Both: First: After one week Last (male): After 7 months Last (female): After 9 months	Foveal hemorrhage (male). Extrafoveal chorioretinal hemorrhage and a small vitreous hemorrhage (female)	Regression of the hemorrhage after 6 weeks and normalization of the foveal contour (male). Regression of the hemorrhages at 12 weeks (female).

Only the male complained about decrease of visual acuity on the first examination. The OCT identified a hyperreflective area in the internal

retinal layer, without improvement with time. There was a shadow defect on the underlying retinal layers parafoveally at the side of the original laser injury. Even though he had a parafoveal scar, a residual fovea disturbance, and foveal hyperreflective band, his vision returned to normal within 7 months. The female had a BCVA of 1.0 in the affected eye at time of examination. This did not change at next follow up two weeks later. The final follow up was nine months later. The male had a more severe injury in his left eye, and his BCVA at first examination was counting fingers. On follow up after six weeks his BCVA was 0.625. His BCVA at the final follow up seven months later was 1.0 in the effected eye. The irradiance was said to be below  $50 \text{ mW/cm}^2$  at 30 meters and  $10 \text{ mW/cm}^2$  at 50 meters from the laser.

The examiners of these two cases decided to investigate if class 3B (output energies  $<500 \text{ mW}$ ) handheld laser pointers can cause eye injuries. A 44 year old man scheduled for eye removal accepted to be a test subject.

Laser power	Laser wavelength	Distance	Exposure duration
$< 500 \text{ mW}$	532 nm	1 m	0.5 to 64 seconds

Year	Examination time frame	Symptom/injury	Remarks
2009	Immediately	Retinal detachment.	No retinal coagulation was observed. No abnormalities.

The VA before the illumination was 1.0. The VA after exposure was not performed. The eye was immediately enucleated after the experiments. Although the illumination was 0.5 – 64 seconds with a doubling of the exposure duration after each shot, only a local retinal detachment was observed at the laser spots. This is unlikely the only injury an eye would suffer under such circumstances. One explanation to this unlikely event is the laser output, which in this case was not measured. It is well known that there is a large variation in energy output from commercial laser pointers. The energy output from a Class 3B laser is defined as higher than 5 mW and lower than 500 mW.

**Case 31 – A 13 year old male was accidentally exposed to a green or red laser beam during Halloween [Turaka et al. 2012]**

Laser power	Laser wavelength	Distance	Exposure duration
5 mW	532 or 650 ±10 nm	1 m	1 minute

Year	Examination time frame	Symptom/injury	Remarks
2009	First: ≥ 1day Last: After 2 days	Lesion in the fovea region with disruption of retinal pigment epithelial layer and choroidal infarction.	Initial blurry vision and a central scotoma. The day after examination VA improved without any other changes.

His VA at first examination was 0.2, which improved to 0.33 the day thereafter. The authors are unclear on whether it was a green or red laser. They report a green laser with a wavelength of 650 nm, although this wavelength is within the red spectrum.

**Case 32 – A 15 year old male playing with green laser in front of a mirror was exposed several times in both eyes [Wyrsh et al. 2010]**

Laser power	Laser wavelength	Distance	Exposure duration
150 mW	532 nm	~1-2 m (unknown)	Unknown

Year	Examination time frame	Symptom/injury	Remarks
2010	First: After 2 weeks Last: After 4.5 months	Central sub-retinal hemorrhage and retinal edema (left eye). Several hyperpigmented areas in the foveolar region (right eye)	Both eyes were exposed with blurred vision in both eyes

At the first examination his VA in the left eye was only counting fingers at a distance of 1 m (~0.025) and 0.4 in his right eye. At the follow up, 4 months later, his VA had improved to 0.625 (right eye) and 0.8 for his left eye. A parafoveal hyperpigmentation was evident in the left eye after four months. His visual function did improve during this time.

**Case 33 – A teenager playing with a green laser pointer was exposed several times in both eyes [Ziahosseini *et al.* 2010]**

Laser power	Laser wavelength	Distance	Exposure duration
Unknown	532 nm	~0.5 m (unknown)	Unknown

Year	Examination time frame	Symptom/injury	Remarks
2010	First: Unknown Last: 2 months after initial examination	Foveal lesions with pigment epithelial defects. Subfoveal disturbances. Hyperpigmentation.	Visual acuity improved from 0.5 to 1.0 within two months

His VA at first examination was 0.5 in both eyes, which improved to 1.0 in both eyes two months later.

**Case 34 – A classmate shone a green laser beam into a 13 year old boy’s left eye [Ueda *et al.* 2011]**

Laser power	Laser wavelength	Distance	Exposure duration
20 mW	532 nm	Unknown	~1 s

Year	Examination time frame	Symptom/injury	Remarks
2010	First: After one day Last: After 6 months	Hypopigmented spot in the fovea and scotoma 2 inches from the center (6 days after). Split in the retinal layers (after 2 weeks)	Symptoms developed over time.

Visual disturbance was the first symptom observed after seven hours. The symptoms continued to develop over time. Six months later the VA had improved somewhat. His BCVA were 1.2 in both eyes at first examination. An Amsler grid test, microperimetry and OCT were performed six months after the injury, showing that the width of the lesion had decreased from 120 to 50  $\mu\text{m}$ . No BCVA was performed at this follow up.

There are also other interesting reports where laser pointers are mis-used. In one such case 6 small lasers were joined together and used to illuminate drivers [Case 2000]. In another case a grocery laser scanner (633 nm, 10 W) was made portable by using a car battery and used against a police helicopter. No follow up on these laser attacks was found in the open literature.

# 5. Laser safety standards

## 5.1 Maximum Permissible Exposures (MPE) for eye safety

The existing laser safety standards pertinent to exposure to laser pointer radiation are based on animal experiments. The need for controlled exposure parameters and the risk for permanent eye damage in general preclude experiments on humans. Still, there are a small number of publications on experimental laser pointer exposure to humans, where the exposed eyes were scheduled for removal due to pre-existing serious disease [Robertson *et al.* 2000, Robertson *et al.* 2005].

There is an abundance of damage end-points in the literature but the most common is an ophthalmoscopically identifiable injury in the retina. This is also the major weakness in the determination of correlation between laser exposure and visual complaints.

Several national and international organizations publish safety standards in their name, such as ANSI, the US American National Standards Institute; ACGIH, the US American Conference of Industrial Hygienists; IEC, the International Electrotechnical Commission; and ICNIRP, the International Commission on non-ionizing radiation protection. All these organizations are basing their recommendations on existing scientific literature, although the interpretations can vary somewhat among the organizations. The Swedish standard 60825 is based on the European 60825 standard, which in turn is based on the IEC 825 standard. The maximum permissible exposures in the IEC standard are adopted from ICNIRP. The US ANSI guidelines are consistent with those of ACGIH and are used by many countries throughout the world.

A common time limit for MPE calculations after continuous wave laser exposures in the visible waveband is 0.25 s, which is the average aversion reflex time plus a safety factor. Blinking, pupil constriction, eye turn and head turn all influence the retinal exposure and 0.25 s is a reasonable exposure limit for these factors.

The laser safety standards usually use a standard pupil diameter of 7 mm, because red and infra-red radiation elicits little or none pupil reaction. This pupil size is probably larger than the average person exposed to lasers but the use of a 7 mm standard pupil will add a margin to the safety limits. For short visible wavelengths and exposures longer than 10 s, a 3 mm pupil size is used in the derivation of exposure limits for photochemical damage, because these wavelengths elicit an aversion response including pupil constriction. Nevertheless 7 mm pupil aperture averaging is still appropriate in the exposure assessment because of the physiologic eye movements over several seconds of fix-

ation [ICNIRP 2000]. Other “standard” pupils are also used in the different safety standards. At close distance a typical laser pointer beam is narrower than 7 mm, while at far distance the divergence of the laser beam produces a diameter larger than the 4 to 8 mm pupil of a dark-adapted eye. The dark-adapted pupil diameter decreases with increasing age, leading to an increased safety margin in the elderly.

There have been legal ramifications with laser safety standards, and lawsuits can influence how the standards are used. The exposure limits of the US regulatory body OSHA, Occupational Safety and Health Administration, are in some cases less restrictive than the AC-GIH/ANSI limits. The industry sometimes protests against stringent safety limits because the protective procedures required to adhere to the standards are costly. The standard organizations are basing their recommendations strictly on toxicology data, but let the regulatory bodies decide which protection level is acceptable. The IEC/ICNIRP standards are viewed in a similar fashion. Although these four organizations publish laser safety standards in their own names, their MPE values do not differ for a 0.25 s exposure to continuous wave laser pointer radiation in the 400 to 1400 nm range.

Even for an emmetropic eye only one wavelength at a time can be in focus. The reason is that the human eye has a relatively large chromatic aberration. Between 400 and 700 nm the total focus shift is approximately 2 diopters [Thibos *et al.* 1992], an error that is quite large. Despite the large focus shift our vision is normally not disturbed by this aberration and one explanation is that the wavelengths that are most blurred are the ones that the visual system have lowest sensitivity to. But when estimating the retinal exposure intensities the use of simultaneous laser exposures with two different laser lines results in retinal spot sizes that will probably not have the same spread. The chromatic shift is not linear and the eye gets more and more myopic as the wavelength decreases. This blurriness can easily be perceived when looking at bright blue back-illuminated signs or when looking at blue LEDs.

This phenomenon can be of importance in case of a blue laser, since the crystalline lens of the eye usually cannot focus blue light, it can only increase its refractive power from the normal position – not decrease its refractive power. Laser pointers that are emitting green 543 nm radiation are often also emitting the fundamental wavelength of 860 nm, or 1064 nm. These wavelengths are in the infrared-A and the eye, if hit by a green laser, will probably try to focus at the visible green wavelength, or other wavelengths/objects in the surrounding. Even if the laser standards are adding the two laser pointer exposures as point sources (for intrabeam viewing) one of them is obvious out of focus to some degree. Between 543 nm and 860 nm, or 1024 nm, the focus shift is approximately 0.5 diopters or more [Thibos *et al.* 1992]. Since the safety standards do not include factors relating to the wavelength difference or wavelength region of the simultaneous exposure the effect of chromatic aberration is not included. This can give an extra margin in the safety calculations.



## 5.2 Dose-response functions

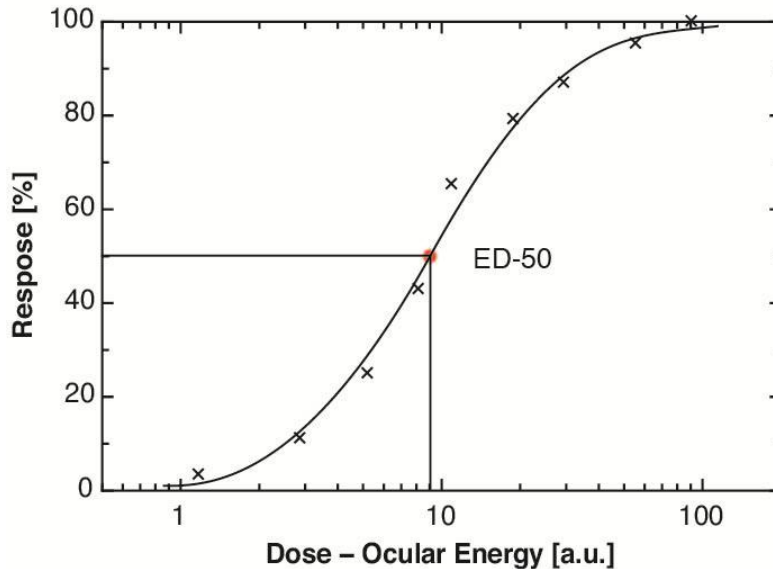
The MPE limits are set well below the exposure thresholds that are known to cause damage to skin and eye. Since the biological variation in damage threshold is strongly individual, the concept of ED-50 is used, i.e. an Exposure Dose resulting in a 50% probability for an observed effect. The ED-50 is a median of data based on experimental studies. The criterion for the ED-50 data is to find the exposure level required to produce a minimal lesion. For the case of the eye, it is the smallest ophthalmoscopically visible change in the retina. This change is a small visible edematous spot, which can occur within 24 to 48 hours of the time of exposure.

When using thresholds like ED-50 data there are always some degrees of uncertainty. Since some damages actually exist below the statistical ED-50 level the exposure limits includes a safety factor. For retinal exposures (retinal hazard region 400-1400 nm) this factor is about one order of magnitude below the ED-50 value [Sloney *et al.* 2002]. Sometimes the size of the safety factors has led to criticism that some standards have safety limits that are impractically stringent. With more and better experiments the safety factors can become smaller, without sacrificing eye health. In some cases with new experimental data having sharp dose-response curves the factor could be as small as 2.5 to 3 [Schulmeister *et al.* 2011].

The ED-50 concept is based on a binary event, effect or no effect. Since there is no such proven true binary threshold in laser retinal damage, a method for determining a Maximal Tolerable Dose in models where a continuous dose-response function exists can be used. The MTD method was originally developed for cataract development after exposure to UV radiation [Söderberg *et al.* 2002]. With this method, a small sample experiment can indicate where a “threshold” is located.

The laser safety experts in the committees have reviewed available threshold data and derived the exposure limits based on current understanding of damage mechanisms. The exposure limits should be viewed as recommendations and not strict thresholds determining safe and unsafe exposures.

It is important to note that for some individuals the safety factor can be much lower, for example if an eye has a higher susceptibility for retinal damage compared to the median value (ED-50) of the dose-response curve. Conversely, injuries may require higher exposure levels than ED-50 for some individuals. One can say that the ED-50 limit equals to a laser exposure that gives a 50/50 risk of causing a minimal detectable lesion. An illustration of a dose-response function is shown in figure 5.1.



**Figure 5.1.** Illustration of ED-50 data in retinal exposure experiments. Ocular energy is given in arbitrary units. Illustration by J Thaug.

When composing the datasets from different experimental data the uncertainties for each laboratory experiment must also be carefully reviewed. In most cases the data comes from animal models that have to be converted regarding the differences in optical performance, pigmentation and retinal structures of the human eye. One concern has been the large variation in slope of the dose-response curve for earlier experimental studies that can be caused by de-focus [Sliney *et al.* 2001]. It is generally accepted that laboratory settings have a common minimal refractive de-focus of 0.25 diopters resulting in larger retinal spot-sizes. Such small deviations are modeled to have large impact on the ED-50 value. Sliney *et al.* [2001] concluded that the safety factor for example when applied to infrared radiation-B wavelengths “can be two-fold and not ten-fold”.

MPE datasets for retinal exposures in the standards are available for a set of different retinal exposure scenarios. Firstly, the exposures are divided into two source categories: “point source” and “extended source”. For the case of a laser pointer exposure, the two categories can usually be thought of as a direct hit (intrabeam viewing, including reflected beams by mirror like surfaces), and a diffuse reflection in some exterior surface. The minimal retinal image from a point source is about 10  $\mu\text{m}$  in diameter due to diffraction and optical aberrations but the corresponding thermal image is effectively about 25  $\mu\text{m}$  in diameter (the observed smallest lesion is also of that order, [Delori *et al.* 2007]). Converted into visual angle it is obvious that many light sources can have an angular extent that yields the same retinal image size as the “minimal” retinal image from a point source. Because of this minimal spot size, even small extended light sources may fall into the “point source” category. To separate between point source and extended source in the standards, the angular extent (retinal area) is compared to a minimal visual angle,  $\alpha_{\text{min}}$ , that has a fixed value of 1.5 mrad. One

reason for defining this angle is to make the standards easier to use since there is no need for more complex calculations of the intensity distribution at the retina. Instead the standards are presented as exposure limits for a corneal plane just in front of the eye and all uncertainties about optical performance is built into the MPE data in the standards. The relation between the two categories are denoted  $C_E$  in ANSI, and is a function of visual angle,  $\alpha$ , and  $\alpha_{\min}$ . There is also a maximal angle,  $\alpha_{\max}$ , from where the injury threshold does not change with increasing spot size.

Even if a point source is imaged as a small point at the retina, the normal eye movements tend to distribute the absorbed heat over larger retinal areas during long exposure durations. The angular size of these movements depends mostly on the exposure time. The exposed retinal area during normal eye movements can be distributed over tens or hundreds of  $\mu\text{m}$  in diameter. For exposures of 100 s or more, the standard uses a fix spot diameter of about 190  $\mu\text{m}$  [Delori *et al.* 2007].

The standards describe MPE limits for continuous wave or single pulse exposures and in the case of multiple or repetitive pulses the safety limits are calculated by using three rules. First, each single pulse must meet the exposure limits; second, a continuous wave equivalent exposure with the same average power must meet the limits; and the third rule is that any single pulse must not exceed the single pulse MPE multiplied by  $n^{-0.25}$ , where  $n$  is the number of pulses that the eye can be exposed to.

For lasers with multiple wavelengths, the quotient of the radiant power divided by the MPE is calculated for each wavelength. For the exposure to be safe, the sum of all quotients must be 1 or less. The need of performing MPE calculations including multiple wavelengths has increased by recent years since the growth of commercially available products with multiple wavelengths [Roach *et al.* 2006]. Handheld laser emitting green wavelengths often use frequency-doubling techniques with the risk of also emitting the fundamental wavelength. There is, unfortunately, very little data for estimating exposure hazards in these cases [Roach *et al.* 2006].

## 5.3 Comparison of case histories and MPE

After identification of the 34 reported cases listed in chapter 4.1 only 12 hold for further analysis without too many assumptions regarding the exposure parameters. Actually, only one case (case 26) falls within the scope of this report; exposure duration between 0.05 and 1 s. Due to the low number of cases all 12 will be analyzed and discussed.

In most of the cases the laser output was known to some extent, but the information about exposure duration and distance was vague. In some cases also the information about the laser wavelength

was a bit confusing. Additionally, in all cases the data for laser beam divergence was not specified and a divergence angle of 1 mrad was therefore presumed for the exposure analysis. Since all distances were relatively short, it is quite safe to state that the beam diameter at the exposed eyes were smaller than the defined 7 mm pupil in the standards. The assumption is based on beam cross-sections due to the angular divergence. The beam diameter, by divergence, was estimated to be 3 mm at the pupil, or smaller, for all cases. Further, an output diameter of more than 4 mm at the laser beam aperture is unlikely. In some of the cases the actual eye pupil size may have been smaller, reducing the risk of retinal injury.

By identifying the exposure data in the selected group of cases, most of them fall into the photothermal damage category, since exposure durations were shorter than 10 seconds. For these cases the calculated corneal irradiance was only compared with the exposure limit for thermal damage according to the standards. In the cases with exposure durations longer than 10 seconds it was also necessary to calculate the exposure limit for photochemical damages.

The data inserted in table 5.1 was, when necessary, calculated by range or combinations of specified output power and/or exposure durations. Both irradiance ( $E$ ) and integrated irradiance ( $H$ ) was calculated in order to be comparable with the exposure limits for both thermal and photochemical damage. For cases with short exposure durations ( $t < 10$  s) the photochemical damage limits was not applicable and limits were not given in the table. The following equations from the ICNIRP guidelines have been used depending on wavelength region; equations 5.1 to 5.3 (400-700 nm) and equation 5.4 (700-1050 nm):

$$\begin{array}{llll}
 H_{limit} = 18 * t^{0,75} * C_E & [\text{J}\backslash\text{m}^2] & t < 10\text{s} & (Eq\ 5.1) \\
 H_{limit} = 100 * C_B & [\text{J}\backslash\text{m}^2] & 10 \leq t < 100\text{s} & (Eq\ 5.2) \\
 E_{limit} = 10 & [\text{W}\backslash\text{m}^2] & 10 \leq t < 100\text{s} & (Eq\ 5.3) \\
 H_{limit} = 18 * t^{0,75} * C_E * C_A & [\text{J}\backslash\text{m}^2] & t < 10\text{s} & (Eq\ 5.4)
 \end{array}$$

where  $t$  is the exposure duration in seconds and  $C_E$  is a factor used for managing extended source scenarios from point source exposures. The  $C_E$  factor for all listed cases are equal to 1 since the exposures are assumed to be normal intrabeam viewing, which for laser pointers is the same as a point source exposure. The  $C_A$  factor in equation 5.4 is a wavelength dependent function:  $10^{0,002(\lambda-700)}$ , where  $\lambda$  is the wavelength of the laser.

**Table 5.1.** Exposure data, min and max respectively, are calculated by extreme combinations of specified output power and/or exposure durations when data are available in spans. Both irradiance ( $E$ ) and integrated irradiance ( $H$ ) data are calculated in order to be comparable with exposure limits for photothermal and photochemical damage. In cases where photochemical damage limits is not applicable the limits are omitted in the table (case 26 and 27). Limiting exposure data regarding to the standards are underlined. A risk ratio (RR) is also calculated as the quotient between exposure data and MPE limits.

Case	Exposure data		MPE		RR
	$E$ [ $W/m^2$ ]	$H$ [ $J/m^2$ ]	$E_{limit}$ [ $W/m^2$ ]	$H_{limit}$ [ $J/m^2$ ]	
1	<u>130</u>	3900	<u>10</u>	2511886	13
3	<u>26</u>	260	<u>10</u>	2511886	2.6
5	<u><math>\geq 130</math></u>	$>1300$	<u>10</u>	4365	$>13$
20	<u><math>&lt;130</math></u>	$<2600$	<u>10</u>	2511886	$<13$
21a	<u>26</u>	1560	<u>10</u>	2884032	2.6
21b	<u>26</u>	7799	<u>10</u>	2884032	2.6
22a	<u>52</u>	3120	<u>10</u>	2884032	5.2
22b	<u>52</u>	15599	<u>10</u>	2884032	5.2
23a	<u>156</u>	9359	<u>10</u>	1513561	15.6
23b	<u>156</u>	46796	<u>10</u>	1513561	15.6
24a	<u>78</u>	4680	<u>10</u>	4365	7.8
24b	<u>78</u>	<u>54595</u>	10	<u>4365</u>	12.5
26	$>520$	$\geq 520$	-	<u>18</u>	$>28.8$
27*	$<130$	$<130-260$	-	2749; <u>4623</u>	$<0.056$
30	<u><math>&lt;12999</math></u>	6499-831925	<u>10</u>	11; <u>4365</u>	$<1300$
31	<u>130</u>	7799	10	<u>4365</u> ; $1 \cdot 10^6$	13

\* Note. In case 27 the exposure falls in the 700-1050 nm wavelength region, all other in 400-700 nm. Exposure values and limits for case 27 are given for one pulse with a span of 1-2 seconds ( $130-260 J/m^2$ , and corresponding dual exposure limits), It was also reported that the exposure occurred two times with an unknown interval, this have not been included in the calculations.

Identification of the exposure data shows that all except one case had exposure levels exceeding the exposure limits. The exception was in case number 27 where the exposure from an infrared radiation laser was found to be below the MPE limit. Some explanations have to be made regarding a few of the calculations. In case number 1 the exposure duration of 30 or 60 seconds does not affect the calculation of MPE or RR. In case number 27 the laser was indicated to be in the range of 825 to 880 nm, without any further specification. Given the wavelength range we have assumed that it is a wide spectrum laser diode, such as a SLD (super luminescent diode), which may have similar spectrum. We have therefore made a rough calculation by replacing the light source with a laser beam at a center wavelength of 852 nm. The difference between 1 and 2 seconds in the calculations gives marginal difference on RR ( $<0.047$  or  $<0.056$ ). In case number 30 the dual sets of MPE limits is a result of the large range of indicated exposure duration, which requires that all three equations (5.1, 5.2 and 5.3) must be calculated. In case number 31 there was an uncertainty about the laser

wavelength (green laser with 650nm?) and therefore exposure limits for both 543 and 650 nm have been calculated. The only difference in the two calculations is the value of the  $C_B$  coefficient. The 543 nm exposure is found to be the worst case with  $H_{limit} = 4365 \text{ J/m}^2$ .

Presented in the table 5.1 are also calculations of a RR number (Risk ratio), a relation between the estimated exposure and the MPE data. The maximal RR was identified for the cases where exposure data had to be compared to both thermal and photochemical damage. Identified limiting exposure data, after comparison of the MPE, are underlined in the exposure data columns. The RR numbers give an indication of by how many times the exposure levels exceeded the safety limits (MPE), or if the exposure levels were on the safe side (i.e.  $RR \leq 1$ ).

The RR numbers are related to the reported injuries described in chapter 4.1. Attempts are also made to grade the reported injuries into “Severity of injury” according to the grading scale presented in the official Journal of the European Union [2010]. For the case of eye injuries the four levels are defined as:

- Grade 1. Temporary pain in eye without need for treatment
- Grade 2. Temporary loss of sight
- Grade 3. Partial loss of sight. Permanent loss of sight (one eye)
- Grade 4. Permanent loss of sight (both eyes)

In table 5.2 the cases and corresponding RR numbers are repeated and compared with a rough estimate of the level of injury, here the grading scale is denoted SI (Severity of Injury). The grading of SI was performed by using identification of the information given in chapter 4.1 Case histories and is not built on statistical evaluations.

**Table 5.2.** Comparison of risk ratio (RR) and severity of injury (SI) of the 12 cases presented in table 5.1. Exposure data and medical history is listed in chapter 4.1.

Case	RR	SI	Short description
1	13	Grade 1 or 2	No permanent damage.
3	2.6	Grade 2-3	Normal VA after 8 weeks, but decreased perception of brightness. Residual abnormality of the RPE.
5	>13	Grade 2-3	Residual change in fundus after 2 years.
20	<13	Grade 2-3	RPE disturbances still after 8 months. Visual symptoms restored after 2 days.
21a	2.6	-	No damages or visual defects.
21b	2.6	-	No damages or visual defects.
22a	5.2	-	Afterimages during a few

22b	2.6		minutes and no visual defects.
23a	15.6	-	Afterimages during 5 minutes
23b	15.6		and no visual defects.
24a	7.8	Grade 2	Retinal changes after 5 days.
24b	12.5		
26	>28.8	Grade 2-3	Lesion and hemorrhage after 8 months.
27	<0.056	Grade 2-3	Retinal detachment.
30	<1300	Grade 2-3	Retinal detachment.
31	13	Grade 2-3	Lesion in the fovea.

Hemorrhage and choroidal neovascularization were found in case 5 (RR>13) and in case 26 (RR>28.8). In both cases the exposures came from green lasers (>5 mW and >20 mW, respectively). Retinal detachment was found in case 27 (RR<0.06) and 30 (RR<1300). It is most surprising that retinal detachments can be a result of the low exposure levels described in case 27 in relation to the MPE limits. Also, in case 23 no injuries were reported despite the relatively high exposure data and RR number. One conclusion found when identifying the presented group of cases (case 27 excluded) is that exposure scenarios with RR equal to 5.6 or lower did not result in any damages. Many of the cases with minor retinal damages had RR numbers of 7.8 (case 24a) or higher. A damage, which begins to appear at exposure levels with RR about 10, indicates that a safety factor of approximately 10 times may have been used in the MPE. Therefore, it is also not inconceivable that the ED-50 limits have been reached in exposure situations when RR values are approaching 10. Since the exposure data in the cited references have many uncertainties the analyses in this report must be viewed with caution.

All calculations are performed by using the exposure durations indicated in the case reports, but the real exposure duration can of course be much smaller if the aversion reflex was reducing the exposure to one or several repeated “pulses”. In such cases we may have underestimated the RR numbers, or in other words, the injuries may have occurred at lower exposure levels.

It can also be of interest of calculating RR data for some fictive cases. In table 5.3 we have listed RR data for ten “cases” with exposures from visible lasers (range: 400-700 nm) and with the assumption of exposure duration limited by the aversion reflex (0.25 s). The (time) integrated irradiance (*H*) was estimated by calculating the area of a homogenous laser beam cross-section using the assumption of a 1 mrad beam divergence. Examples with five different laser output values (50, 100, 200, 500 and 1000 mW) together with 2 exposure distances (10 and 100 m) were included in the calculations. Note that all cases include high power lasers that are detrimental at short distances. Also note that caution must be taken since real beam intensities are not evenly distributed over the cross-section. The calculations can only be used as an indication of the possible risks for eye injuries.

Despite the relatively high output power, cases B, D, and F have exposure levels that are relatively safe, that is, a RR value of  $\leq 1$ . As described above, cases with RR equal to 5.6 or lower did not result in any damages except in case 27. If we compare this estimate with the ten fictive cases we find that even the 1000 mW laser case receives a RR value that are lower than 5.6 for distances longer than 100 meters. But of course, if the beam divergence is smaller than 1 mrad the RR value will increase. On the other hand, using a 1000 mW laser pointer at 10 meters results in a RR value of 500!

**Table 5.3.** Calculations of risk ratio (RR) for 10 fictive cases with the following assumptions: divergence=1mrad, exposure duration=0.25 and a wavelength region between 400 and 700 nm. Exposure limits ( $H_{limit}$ ) is  $<6.36 \text{ J/m}^2$ .

Case	Power [mW]	Distance [m]	$H$ [J/m <sup>2</sup> ]	RR
A	50	10	159.2	25.0
B	50	100	1.6	0.3
C	100	10	318.3	50.0
D	100	100	3.2	0.5
E	200	10	636.6	100.0
F	200	100	6.4	1.0
G	500	10	1591.6	250.1
H	500	100	15.9	2.5
I	1000	10	3183.2	500.2
J	1000	100	31.8	5.0

Most lasers in eye clinics use visible wavelengths, such as with ruby and argon lasers, but infrared radiation-A lasers are also used. In recent years the technique of using frequency-doubled Nd:YAG lasers emitting 543 nm has become more common. Since most strong green laser pointers are emitting the same wavelength there are some interesting comparisons that can be made.

In lasers for therapeutic treatment the output data are usually not higher than the examples presented in table 5.3. One major difference in the exposure situation is the retinal spot size that is much larger than a point source caused by a laser pointer. The retinal spot size is dependent on the equipment used and the operator settings, but for conventional photocoagulation systems the spot is in the order of 100  $\mu\text{m}$ .

Clinical laser exposures can have pulse durations of 10 ms with a mean power of 235 mW (SD 57.2) [Sanghvi *et al.* 2008]. The new PASCAL system uses a laser with 577 nm and projects a flat-top profile onto the retina. The system provides 10 to 100 ms pulses with up to 2000 mW and spot sizes from 100 to 400  $\mu\text{m}$  [Lavinsky *et al.* 2013]. In the study by Sanghvi *et al.* [2008] the mean power used for



the PASCAL-system was 396 mW (SD 100.2) and the spot size was approximately 350-400  $\mu\text{m}$ . The selected exposure data for the two systems are presented in table 5.4 together with (time) integrated irradiance ( $H$ ).

Since the projected spot on the retina is relatively large in diameter the angular extent has to be derived in order to calculate the  $C_E$  factor in equation 5.1. Usually the visual angle is computed by taking the ratio of the source diameter and the distance to the source. But since the spot size is already given in our case, and using the conversion that 290  $\mu\text{m}$  on the retina corresponds to 1 degree, the visual angle is found to be 6 mrad for the 100  $\mu\text{m}$  spot and 24 mrad for the 400  $\mu\text{m}$  spot. For exposure durations of 10 and 100 ms  $\alpha_{\text{max}}$  is calculated to be 20 and 63 mrad, respectively ( $\alpha_{\text{max}}=0.2*t^{0.5}$ ). Identification of the visual angles gives that the  $C_E$  factors must be calculated by using the  $\alpha/\alpha_{\text{min}}$  quotient. The  $C_E$  factors are 6/1.5 and 24/1.5 for the two spot sizes.

**Table 5.4.** Calculations of risk ratio (RR) for two types of photocoagulation systems. Exposure data are taken from [Sanghvi *et al.* 2008].

<b>Power</b>	<b>Exposure duration</b>	<b>Spot size</b>	<b>H</b>	<b>RR</b>
[mW]	[s]	[ $\mu\text{m}$ ]	[ $\text{J}/\text{m}^2$ ]	
235	0.01	0.1	61.1	26.8
396	0.01	0.4	103.0	11.3
396	0.1	0.4	1029.5	20.1

The estimated RR numbers in table 5.4 give an indication of the exposure levels that are used in clinical laser systems. RR numbers are higher than for most of the 12 cases in table 5.1 but during retinal treatment the purpose is to cause a well-defined retinal lesion.



# 6. Proposals for future research

## 6.1 A national database

During this project we realized the importance of a database on laser incidents. In USA laser exposures from pranks, alleged terrorist attacks, military accidents and so on are collected in several databases [Ness *et al.* 1997, Johnson *et al.* 2003, Harrington & Wigle 2004]. The well-established database US Army LAIR (The laser accident and incident registry) has six categories; incident, exam, treatment, laser system, subject, and source information [Ness *et al.* 1997, Hoxie *et al.* 1999]. The databases have been useful in establishing laser safety standards, in research for understanding bioeffects of laser radiation and in determining future research and even methods to treat patients. These databases are unfortunately in several cases restricted to organizations within USA.

The databases require a suitable questionnaire to be filed with the complaint. One such questionnaire has been developed by the US Federal Aviation Authority for the aerospace environment. The questions include information that the exposed aircrew contributes, but parts of the crucial data can only be obtained if the laser is captured, and the eye is examined by a trained ophthalmic professional.

## 6.2 Photochemical effects

Due to methodological difficulties in discriminating between photochemical and photothermal damage, there seems to be a lack of data on photochemical effects after continuous wave laser exposure in the sub-second range, especially with interacting wavelengths or interacting damage mechanisms such as combined photothermal and photochemical effects. Visible light laser pointers often emit unfiltered infrared radiation-A. This type of simultaneous exposure to potentially interacting wavelengths has not been studied in detail.

## 6.3 Effects of visual aids

There is a need to investigate the effect visual aids such as eyeglasses and contact lenses have on the eyes of a laser-exposed person. This type of information is not available today. The diopter strength of the glasses or the lenses plays a major role in risk assessment. New MPE values based on the strength of the glasses or lenses could be calculated and clinically verified.

## 6.4 Treatment of laser pointer retinal damage

Although anti-inflammatory drugs have been used in sporadic cases to treat laser retinal damage, there is a lack of data on which treatment is effective and safe. This situation is at least partly linked to the problem of identifying the exact damage mechanisms.

## 6.5 Secondary injuries

Luckily there are few cases of permanent functional visual disturbance after laser pointer exposures, although we foresee more cases in the future when the laser pointers are getting stronger and cheaper. The major risk situation today is the secondary effects of laser pointer exposure. A worst case scenario is blinding of an airplane pilot or a bus driver that lead to multiple casualties. It is important to further elucidate how the effects of glare from laser pointers can be minimized, either by behavioural responses or by protective filters. Vehicle and plane simulators are helpful tools towards this goal.

## 6.6 Long-term or permanent functional deficit

For risk assessment and for medicolegal purposes, there is a need for studies on long-term or permanent functional deficit after exposure to laser pointers. Structural damage can be assessed by any model system, from *in vitro* to *in vivo*, from animals to humans, and the advances in the OCT technique have made this method one of the most suitable for identifying sub-clinical retinal structural damage. The resolution enhancement achieved by adaptive optics can further help in pinpointing subtle changes that common ophthalmic instruments cannot detect [Morgan 2008].

However, the major challenge is probably long term assessment of functional loss after laser pointer exposure. There is a need to optimize and develop psychophysical tests to confirm or reject a functional deficit in the presence of structural abnormality. It is especially important to develop such tests for use with animals. For humans, reading performance testing is a potentially useful method to psychophysically test visual function deficit [Thaung 2013] after retinal laser damage. Well planned experimental laser exposure on human volunteers with eyes scheduled for removal is likely an efficient way to collect necessary data, although it might be difficult to achieve long-term data. This type of study can be performed in a tertiary ophthalmic oncology clinic as a single center or multicenter study.

## 7. Conclusions

The easy access to commercial laser pointers has led to an increasing trend of misuse of lasers towards main targets such as pilots, drivers and law enforcement personnel. Today's strong laser pointers can flash blind a pilot at a distance of more than 10 km, and at shorter distances cause permanent visual dysfunction or even blindness.

We have analyzed 34 reported cases of laser pointer exposure and related the eye damages to existing MPE values. Among the many reports on laser pointer exposure, only a small proportion has confirmed retinal injuries. Retinal damage was described for red, green and infrared laser pointers and all occurred at very close distance to the laser, in most cases less than a meter:

- Red lasers with  $\leq 5$  mW output power can cause a temporary and relative loss of the central vision.
- Green lasers with  $\leq 5$  mW output power can disrupt the retinal pigment epithelial layer and lead to choroidal infarction. With repeated exposures over years choroidal neovascularization can occur with associated risk of vision loss.
- Green laser with  $\leq 7$  mW output power can cause visible damage to the retinal pigment epithelium.
- Green laser with  $>20$  mW output power can cause enlarging lesion, retinal edema and hemorrhage, if viewed for more than one second.
- Infrared radiation-A laser with wavelength between 825- 880 nm and an output energy of  $<5$ mW can induce retinal edema and focal retinal detachment.

We have identified areas where research could benefit the society. With a national database on laser pointer exposures there is a better chance to find patterns and trends on the use of laser pointers. There is a need for further research on damage mechanisms, treatment of laser damage, long-term permanent laser damage, and on the effect of visual aids and refractive errors in laser pointer retinal damage. Further, the risk of injuries or deaths secondary to laser pointer blinding requires more attention, especially from the aviation authorities. Finally, an essential area of research is the development of methods to identify functional visual deficit in the presence of structural retinal damage.

The laser safety limits of today are both important and meaningful in the industrial or workplace setting where the dangers are well known, as well as the methods of protection. In contrast, intentional use of laser pointers to harm others, or inappropriate use of laser pointers due to lack of knowledge about the potential harmful effects are difficult to intervene with. Two important on-going objectives for the legislative and governing bodies are information campaigns and legislation to minimize the availability of harmful laser pointers.



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