

Retinal and Choroidal Damage from Long-Term Exposure to a Laser Pointer Beam

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Abstract

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Introduction: Laser pointers are devices that produce a weak laser beam of 630-680 nm wavelength and 1-5 mW power (Class II or III A laser). These devices generally emit a red beam that is used by lecturers and teachers for presentations. Some children use pointers as toys and sometimes direct the beam to their own or others' eyes.

Material and Methods: Following irradiation by a laser pointer beam for 8 seconds the eyes of Chinchilla rabbits were examined by ophthalmoscope, and fluorescein angiography was performed 5, 10 and 15 min after the exposure. The rabbits were killed immediately or 24 h after exposure, the eyes were enucleated, and the histological features of sections from fundus, retina and choroid were observed by transmission electron microscopy.

Results: A fluorescein block was found in the irradiated area immediately after irradiation and it increased in size with increasing time after exposure. The ultrastructural study showed acute oedema shortly after exposure, and thick collagenic bundles after 24h.

Conclusion: Laser pointers with labelled power of less than 1mW are capable of producing visible and ultrastructural lesions in pigmented rabbit eyes.

Key words: Laser Pointers, Retina, Choroid, Ophthalmoscopy, TEM.



Introduction

Teachers and lecturers use laser pointers to highlight key areas on charts and screens during visual presentations. Commonly available laser pointers generally emit red light (between 630 and 680 nm wavelength) although more expensive devices are available which emit green light (532 nm). When used in a responsible manner, laser pointers are not considered to be hazardous (1, 2, 3). However, as the availability of such devices has increased so have reports of their misuse. As a result, the Food and Drug Administration (FDA) issued a warning in December 1997 on the possibility of eye injury to children from handheld laser pointers (4). Of particular concern was the promotion of laser products as children's toys. Unfortunately, children do foolish things, and they are at highest risk of laser injury because of their clear ocular media (5). In the wake of reports of eye injuries involving young children caused by laser pointers, the American Academy of Ophthalmology (October 1998) upgraded an earlier caution to a warning, stating that laser pointers can be hazardous and should be kept away from children (2, 6). In one case a 19-year-old woman had an acute reduction of visual acuity in the right eye after deliberately staring into a commercial class II laser pointer for approximately 10 seconds (3). Also the clinical history and foveal findings of Sell and Bryan in an 11-year-old patient are convincing evidence of pointer injury (7).

The potential for a specific laser to produce eye damage depends on the type of laser, the distance from the laser, the energy of the laser, and total exposure time. The British Standards Institute classification system for lasers and their potential to cause ocular damage (8, 9) includes: Class I, incapable of producing damage; Class II, Low- Power emission ($<1\text{mW}$) in the visible spectrum capable of producing damage after chronic exposure (the human eye's aversion response will limit exposure after 0.25s of exposure to Class II light); Class IIIA, ($<5\text{ mW}$) can cause permanent injury from prolonged viewing or when viewed through optical instruments; Class III B, ($5\text{-}500\text{mW}$ or $<10\text{ J.cm}^{-2}$ for a pulsed system) can cause injury upon direct viewing of the beam and

specular reflections ; and Class IV , high-power emission ($>500\text{mW}$ or $>10\text{ J.cm}^{-2}$) which is injurious to eyes and skin, and poses a fire hazard (10, 11). The FDA classified laser pointers and requires that they have a warning label that cautions users to refrain from staring at the laser beam (1, 5).

The pathophysiological effects of retinal exposure to laser may range from the transient visual effects of glare or flash blindness to more permanent injuries, such as thermal or hemorrhagic lesions. Glare and flashblindness occur in a manner similar to the effects of a camera flashbulb. Photoreceptor cell saturation results in an afterimage, which gradually fades with time. Only wavelengths in the visible spectrum produce glare and flash blindness. The cornea and lens are damaged by ultraviolet and far-infrared wavelengths. The resultant photokeratitis, corneal burns, and cataracts are caused by a photochemical process or thermal denaturation of proteins in the cornea and lens (10, 12, 13).

The retina, itself, is as susceptible as any other part of the body to laser damage. The optical system of the eye intensifies the energy of the laser and renders it more harmful to the retina and choroid. The cornea and lens are capable of concentrating the laser energy 100,000 times before it reaches the retina (14, 15). Lasers can damage the retina and choroid by photochemical, thermal and ablative mechanisms (16, 17, 18, 19).

The human eye is inherently sensitive to some wavelengths. Most laser pointers are red or red orange in colour, with wavelengths ranging from 630 to 680 nm. The 650 nm wavelength pointer is often selected because of its relatively low cost and increased brightness, about two times that of the 670 nm laser pointer (1, 20).

The aim of the current study was to understand the effect of a laser pointer beam on pigmented rabbit retina and choroid.

Material and Methods

Six mature male pigmented Chinchilla rabbits each weighing from 2.3-2.7 kg, were maintained under

standard laboratory conditions (12h light-12h dark). Rabbits 40 to 60 wk of age were used because the power of the cornea and lens undergoes considerable changes in the first 30 wk of life concomitant with the increase in size during growth (21, 22, 23). The rabbits were screened before exposure to ensure that eyes were intact. All procedures were performed during the light cycle. The rabbits were divided into control group, and A and B experimental groups. Experimental rabbits were anesthetized by intramuscular injection of ketamine (10 mg /kg) and the pupils were dilated with a drop of phenylephrine and tropicamid. Eyelids were kept open with adhesive tape. The beam of a laser pointer, with 630-670 nm wavelength and labeled as emitting <1 mW power (class II laser) was directed into the rabbits eyes for 8s (24, 25, 26). Note: power measurement of this pointer showed that its real output was 3.9 mW.

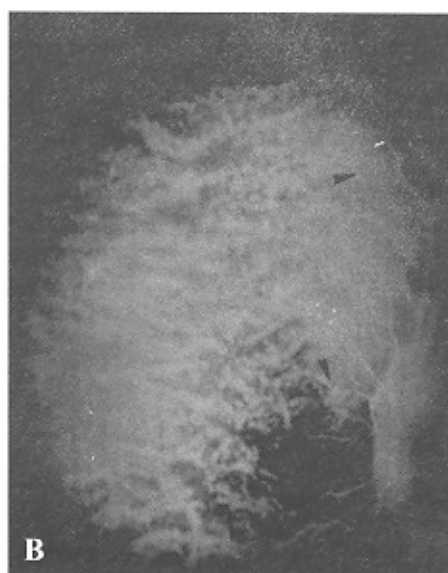
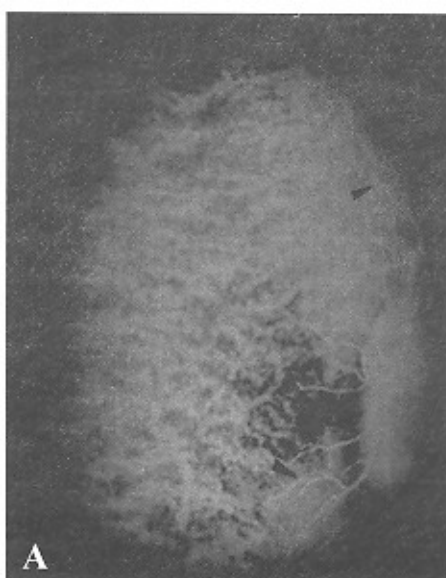
Direct ophthalmoscopic examination was undertaken immediately (group A) and 24 h (group B) after exposure. The eyes were also examined by the use of fluorescein angiography. Two ml of a 10% solution of fluorescein were injected in the marginal ear vein and photographs of the fundus were taken 5, 10 and 15 min later (27, 28).

For ultrastructural studies the animals of control and experimental groups (group A, shortly, and group B 24 h after laser exposure) were sacrificed. The enucleated eyes were fixed by 2% glutaraldehyde and 2% paraformaldehyde in 0.1 M phosphate buffer at pH 7.4. After removal of cornea and lens the eyes were resected and were kept in the fixative at 4°C. In the next days the eyecups were inspected with a stereomicroscope. The irradiated areas were localized (damage spot remained visible as a whitish spot in the resected eye cup); tissue segments containing them and adjacent, unexposed, retina were excised. The tissue segments were rinsed in buffer and post fixed in 1% osmium tetroxide for 1.5h, then dehydrated in acetone, and embedded in Epon. Thin sections (50 nm) were cut and stained with uranyl acetate and lead citrate (27, 28).

Results

* Ophthalmoscopy

Immediately after exposure, retinal damage was detectable in the rabbit eyes by direct ophthalmoscopy. The laser spot was grayish white and, the eye fundus became oedematous without hemorrhage. The lesion expanded 24h after exposure, the oedema remained and scar tissue was formed.



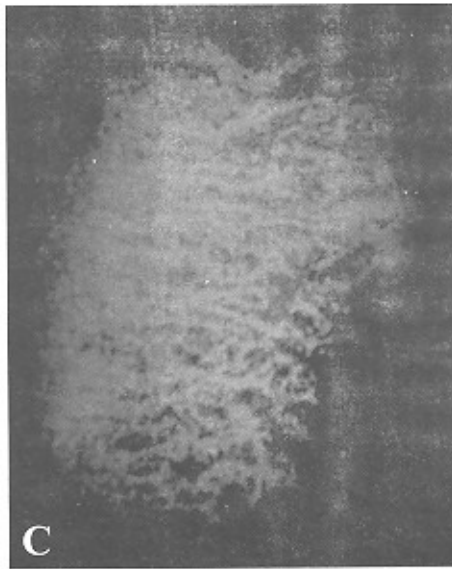


Figure 1: Fluorescein angiographs performed 5(A), 10(B) and 15 min (C) after the exposure. The angiographs show a small fluorescent block (▲) in the irradiated spot (A) that is increasing in size as time increases (B and C respectively). Also there is an increase in the retinal opacity and a decrease in clearance of choroidal vessels (▲) as time increases (A, B and C respectively).

* Angiography

Fluorescein angiography 5 min after exposure of the fundus showed a small fluorescent block in the irradiated spot (Figure 1A) that increased with time (figures 1B and 1C).

149

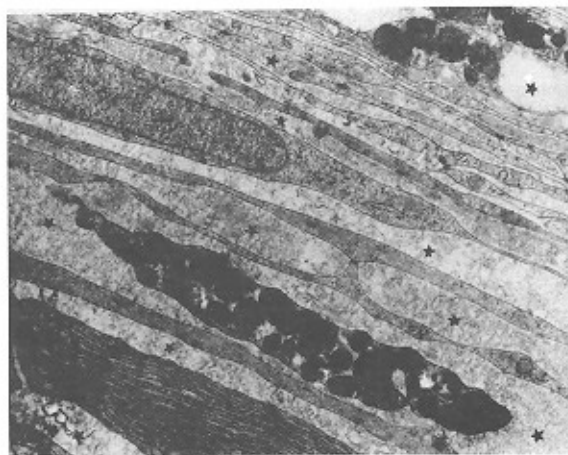


Figure 2. Electron micrograph of choroid, group A. There are wide spaces (*) between choroidal cells that shows existence of oedema in choroid. x 14000, P: plasma cell, F: fibroblast

Also, there was an increase in the retinal opacity and reduction in clearance of choroidal vessels as time after exposure increased to 10 and 15 min.

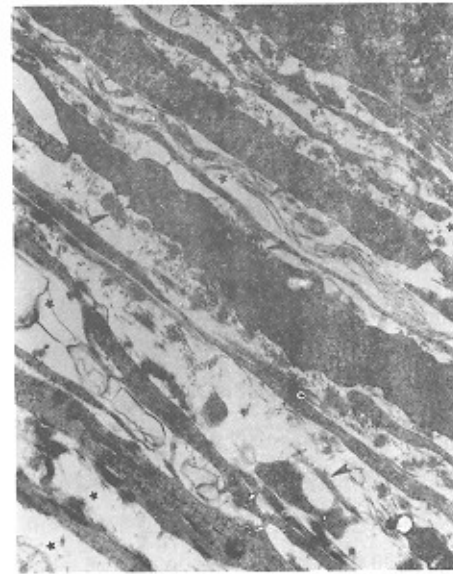


Figure 3. Electron micrograph of choroid, group B. There is oedema (*) in choroid. High density collagen fibers (▲) are observed in intercellular spaces. x 14000

* Electron microscopy

In experimental rabbits (Groups A and B) the retinal pigmented epithelium and their melanin granules appeared intact ultrastructurally. The melanin granules were apically located at their primary site. No damage could be detected in the choriocapillaries, and Bruch's membrane was intact. In group A there were wide spaces between choroidal cells that showed existence of oedema in the choroid (Figure 2).

10 and 15 min.

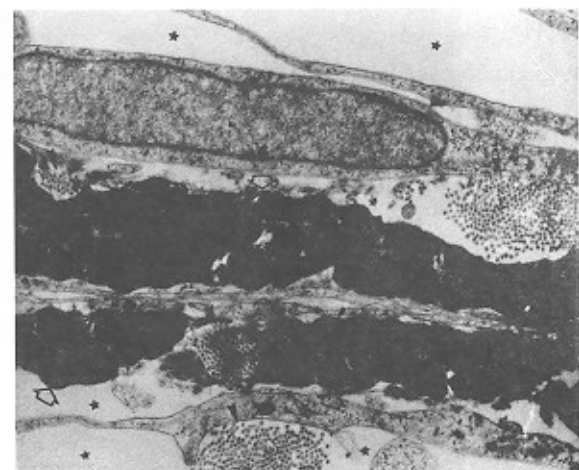


Figure 4: Electron micrograph of choroid, group B. Cross section of thick collagen fibers (▲) is obvious. x 20000 Melanocyte ↑



Figure 5. Electron micrograph of choroid, Control group. The cells are set regularly without any spaces between them. Compare with fig 2, 3, 4. x 14000.

In group B rabbits there were thick collagenic bundles that showed scar formation in the irradiated area of the choroidal layer (Figures 3, 4). Figure 5 is an electron micrograph of the chorion of a rabbit from the control group for comparison with Figures 2,3 and 4.

Discussion

Laser pointers emitting light with a wavelength that is close to the eye's peak response, have the capability of producing an adequate visual stimulus at lower radiant powers. The laser pointer beam is focused by the cornea and lens, and absorbed by the retina and choroid. Injury occurs when the energy level and duration of the laser exposure are sufficient to damage the eye. As the power output of laser pointers is relatively low (<5 mW), the beam causes injury when directed from a close range (10 feet or less) and for several seconds to a few minutes. These injuries can sometimes result in permanent visual impairment(1, 29, 30).

The clinical appearance of retinal lesions after laser exposure is related to the type of laser involved and the power output. Retinal injuries may demonstrate oedema, necrosis, subretinal or intraretinal hemorrhage, vitreous hemorrhage, and macular or retinal holes (10). Retinal and choroidal damage may also be associated with a specific type of laser. Pulsed

lasers in the visible or near infrared spectrum have been associated with significant retinal hemorrhage. Circumscribed retinal and choroidal lesions without significant hemorrhage may indicate that a visible laser in the continuous wave mode, such as a laser pointer, has been used (10). Also pulsed laser irradiation can result in damage to both the inner and outer retinal layers, while the continuous laser radiation produces damage to the outer retinal layer and choroid. A sufficient level of laser light transmitted to the choroid can induce small vessel occlusions and/or oedema (31). Bruch's membrane remains intact, although constituent collagen fibers exhibit signs of thermal damage such as increased density and cross sectional diameter (27), as shown in this study.

Using the theoretical model of Manister et al. the increased temperature produced in the retina after exposure to light can be calculated (32). The thermal response is dependent upon the wavelength of the incident light. A laser pointer with an output energy of 5 mW can produce a temperature increase of 15 to 20°C after 0.1 s exposure and this is sufficient to produce thermal injury (33, 34). An ophthalmoscopically visible damage threshold was detected with 9-15 mW for 150-270 ms exposure in the visible range. Other much more sensitive methods for damage detection, such as fluorescein angiography, microscopy and electron microscopy, lead to a maximum permissible exposure (MPE) value to be defined as 1/10 of the determined visible threshold (33, 34).

Klein et al. measured the power of more than 40 laser pointers from various manufacturers. They found that most fall in laser class III B which means that they have an output power of more than 1mW and were not correctly classified by the manufacturer (34, 35, 36). The power of the pointer used in this study was nearly 4-times greater than stated on the label.

In summary, when used properly, the risk of eye injury from a laser pointer is extremely low. An individual who receives a transient exposure may experience a dazzling effect, resulting in distraction or temporary visual impairment. The duration and severity of these effects varies between individuals and with their state of dark adaptation at the time of exposure.

An eye examination to rule out permanent eye injury from a laser illumination should be performed if after images persist for several hours or if a loss of clarity is apparent (1, 3, 5, 30).

To reduce the risk of eye injury, should they fall into the hands of children or irresponsible individuals, Class II laser pointers (rather than Class III A) are recommended for use by the general public. An increase in the perceived brightness of red laser pointers can be achieved without the need for additional power by selecting those that emit light of wave lengths shorter than 670 nm. While Class III A laser pointers can continue to be used by responsible

adults, they should be replaced by lower powered pointers whenever possible.

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