Lasers in Medicine

Presented by

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1. Appropriate radiation dose and temperature ranges

Visual perception is a complex phenomenon that is initiated when electromagnetic radiation from the sun reaches the retina and the visible spectrum is converted from radiant energy into sensation by the phototransduction in the retinal photoreceptors. The ability to translate electromagnetic radiation into usable visual information relies on a complex interaction between the different structural and functional components of the eye and the brain. Electromagnetic radiation has a dual wave-particle nature, but when it is absorbed by a photoreceptor at the retina, its particle nature is dominant. The portions of the electromagnetic spectrum that interact with the eye are shown in Figure **1**. They are referred to as optical radiation and include wavelengths from ultraviolet (100-400 nm, UVR), visible light (400-760 nm, VIS) to infrared (above 760 nm, IR) . The International Commission on Non-Ionizing Radiation Protection (ICNIRP) defines several subgroups of ultraviolet or invisible radiation classified into UVA (315-400 nm), UVB (280-315 nm) and UVC (100-280 nm).

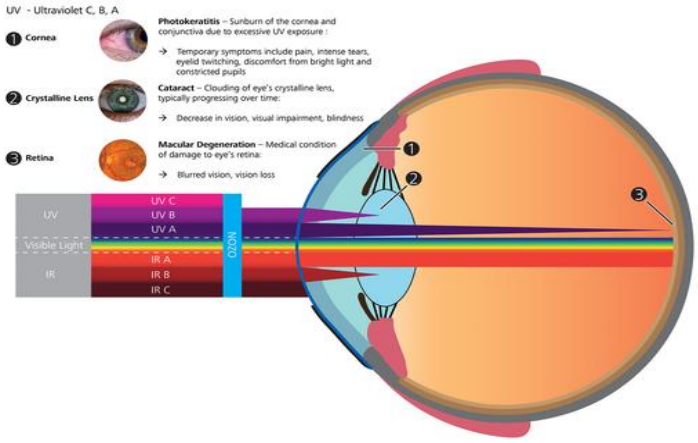


Figure (1)

Infrared radiation has also been subdivided into 3 groups depending on the wavelength: IRA (700-1400 nm), IRB (1400-3000 nm) and IRC (3000-10 000 nm) 1. We follow these subdivisions as they are useful for safety and hazard evaluations. They separate the wavelengths into bands of roughly similar photon energy, tissue penetration and general classes of bio-effects. Visible light is referred to as short ($\lambda = 420$ nm), medium ($\lambda = 530$ nm) and long wavelength ($\lambda = 560$ nm) corresponding to the peak absorption spectra of the cone visual pigments. Light is transmitted through the eye and then after absorption, scattering and transduction, the signals are compressed and send via the optic nerve to the brain directing both visual perception and circadian rhythm. Because of its function and structure, the eye is most susceptible to light damage; it is designed to focus incoming light rays to form images on the neural retina

2. Laser radiation penetration depth in tissue

This has the effect of concentrating the light or increasing the power density of light on the retina. Thus, light delivering a radiant exposure insufficient to produce skin damage may indeed cause injury when focused on the retina. The absorption spectrum of each ocular tissue 3, 4 must be taken into account to understand its differential sensitivity with respect to wavelength (Figure 1). The primary factors that determine whether ambient radiation will injure the human eye outside of defined laboratory conditions are: (1) the intensity of the light, (2) the wavelength received by ocular tissues and (3) the age of the recipient 4. In the eye, UVR is not known to contribute to visual perception and there are strong evidences that acute high dose exposure to UVR causes photokeratitis and photoconjunctivitis, while even low dose chronic exposure to UVR is a risk factor for cataract, pterygium and squamous cell carcinoma of the cornea and conjunctiva 5-7. There is weaker evidence in relation to other, noncancer eye conditions, related with the oxidative stress (OS) induced by UVR exposure.

3. TYPES OF RADIATION -INDUCED DAMAGE TO THE OCULAR TISSUES

Three main types of tissue light damage are distinguished 8: thermal (inflammatory response), photomechanical (stress confinement) and photochemical (photo-oxidation). Important differences among these photic damages are related not only to their damaging mechanism but also to their primary absorbers, thresholds and light inducing spectrum 8. One of the main differences between the photochemical and the other damages is their light-inducing sources 8. While thermal and photomechanical damages occur only due to coherent light sources, for example, lasers, photochemical damage may also be produced by exposure to solar radiation. Solar light reaching the eye is essential for visual perception but despite that the eye has several UVR protective mechanisms, discussed in detail in section 3.1, it is easy to expose it to levels that exceed these natural defense limits. We continue with a brief description of the damaging mechanisms in the thermal and photomechanical types and discuss in detail the role of UVR in the photochemical damage, which is thought to be the most common mechanism by which light exposure causes cellular damage. An overview of the different types of damage is given in Table 1.

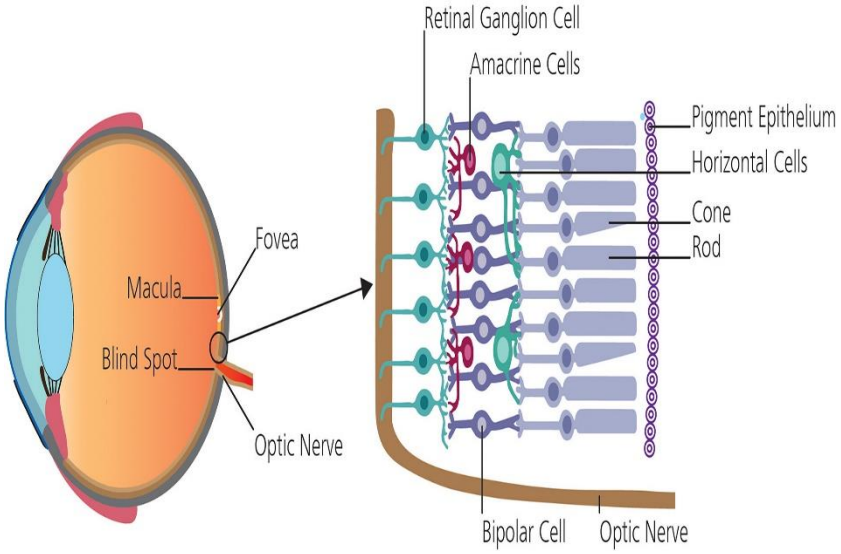
Table 1. Mechanisms of light damage to the eye

Mechanism	Spectra	Exposure time (short pulses)	Temperature increase
Photothermal	upper end VIS; near IR	~100 ms	$10°C < \Delta T < 100°C$
Photomechanical	near IR	~10 ns	ΔT ~10 000°C
Photochemical		linear	
Ablative	UVC	~1 µs	$\Delta T < 10°C$
Oxidative	UVA and VIS	long ~s, min	$\Delta T < 10°C$
Photosensitized			
Type I and type II	UVA	~months, years	$\Delta T < 10°C$
Delayed effects	UVA and UVB	~months, years	$\Delta T < 10°C$

4. Temperature Effect

Photodisruption occurs when the laser exposure deposits energy into the optical zone in a pulse that is shorter than the relaxation time required to relieve the mechanical stress produced in the tissue by thermoelastic expansion 8. The local temperature in the optical zone during the laser exposure may reach up to 10 000°C. Tissue damage results from actual mechanical compressive or tensile forces. Tensile forces resulting in the formation of

microcavitation bubbles, in particular, are lethal for retinal pigment epithelium (RPE) and other cells of the retina (Figur 2)



Figur (2)