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Karl Schulmeister and Mathieu Jean

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MANIFESTATION OF THE STRONG NON - LINEARITY OF THERMAL INJURY

Paper #901

Karl Schulmeister and Mathieu Jean

Seibersdorf Laboratories; Laser, LED and Lamp Safety Test House, 2444 Seibersdorf, Austria

Abstract

It is well known that whether or not thermal injury of tissue occurs depends critically on both tissue temperature and the duration of elevated temperature. Thermal injury can be well modelled by the Arrhenius integral. The Arrhenius parameters used to model thermal injury of the skin, cornea and retina, result in a highly non-linear (i.e. very strong) dependence on temperature and a relatively weak dependence on the duration of elevated temperature (or pulse duration for laser exposure). We present a number of examples of computer model and experimental threshold data where the trends can be understood on the basis of the strong non-linearity of thermal injury with temperature.

Introduction

We know from experience that when contact with a hot surface occurs, it depends on the temperature of that surface whether or not we burn ourselves. We also know from experience that a short exposure to a hot surface, such as just briefly tipping on to it, does not lead to injury, while prolonged contact with the same hot surface would lead to injury. It is thus clear that whether or not thermal injury occurs depends both on the temperature of the tissue as well as on the duration of the elevated temperature (frequently also referred to as “temperature-time-history”). We will show that the dependence on temperature is highly nonlinear, leading to only a very weak dependence, for a given temperature rise on the ‘exposure’ duration. This understanding is helpful when it comes to interpret the pulse duration dependence of thermal injury, the treatment of irregular pulse trains or irregular retinal irradiance profiles.

We were prompted to search out for the dependency of thermal injury on temperature and exposure duration by the work of Brian Lund on the influence of eye movement on retinal injury [1]. Lund showed that the injury threshold is reduced only by a relatively small degree compared to a stable eye, since the local tissue temperature of a moving spot is equivalent to the temperature of a stationary spot, and it is only the

exposure duration of a given spot that is reduced by eye movements.

Modelling Thermal Injury

Thermal injury to tissue is well described by the Arrhenius equation, Eq. 1

$$\Omega = C_1 \cdot \int e^{-\frac{C_2}{T(t)}} dt \quad (1)$$

where $T(t)$ is the absolute temperature as function of time t , C_1 is a frequency factor (with the simplifying assumption that it is temperature independent) and C_2 is an activation energy expressed as temperature. Ω is a measure of the degree of thermal injury and the constants are usually chosen so that $\Omega \geq 1$ expresses that injury occurred (i.e. $\Omega = 1$ would be the threshold scenario). For a given laser exposure, $T(t)$ can be calculated by solving the heat flow equation, usually numerically such as with finite element or finite difference methods, but with simplifying assumption also analytically.

This concept has been used since the 1970’s to model injury thresholds for laser exposure to the skin or eye [2,3,4], and was recently used to provide the basis for an understanding of the spot-size dependence of retinal thermal injury as function of pulse duration [5] which made it possible to significantly increase exposure limits for pulsed extended sources [6]. For the calculations presented here, we use the constants $C_1 = 10^{10}$ 1/s and $C_2 = 83\,600$ K, which are similar to those proposed by Welch and Polhamus [4].

For the case of a constant temperature T , the integral of Equation 1 simplifies to a multiplication of a constant value of the exponential function with C_1 and the exposure time t

$$\Omega = C_1 \cdot e^{-\frac{C_2}{T}} \cdot t \quad (2)$$

Basic temperature and time dependence

Two features of the Arrhenius integral are important for the discussion in this paper: first, the value of the integrand is highly non-linear with temperature since

the temperature is in the exponent, second, the linear dependence on time t .

Jacques [7] points out that Equation (2) can be used to calculate a critical exposure duration t for a given tissue temperature T by setting $\Omega = 1$

$$t = 1 / C_1 \cdot e^{-\frac{C_2}{T}} \quad (3)$$

which is plotted in Figure 1 for the constants C_1 and C_2 given above.

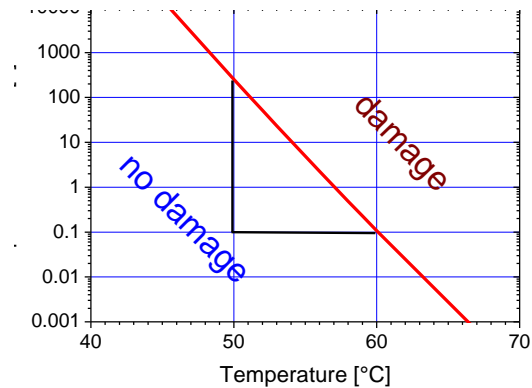


Figure 1. Critical exposure duration for a given constant tissue temperature.

The parameter C_2 characterizes the steepness of the curve: the higher C_2 , the steeper the curve. For the parameter usually applicable to retinal thermal injury, the curve can be considered as very steep as compared to other tissues and types of thermal denaturation [7]. As indicated in Figure 1, for a change in tissue temperature from 50 °C to 60 °C, the exposure time that leads to injury decreases by more than three orders of magnitude, from 300 seconds to 100 ms, respectively. The argument can also be turned around: if the exposure duration is extended by a certain factor, say, from 1 s to 10 s, then the tissue temperature needs to be reduced only by 3 °C in order to compensate for the increase in exposure duration of a factor 10.

Thus, for thermal injury of tissue such as the skin, cornea or retina, the temperature is much more critical than the exposure/pulse duration; the importance of the ‘time-temperature history’ was therefore somewhat overemphasised when it was sometimes referred to as ‘critical’.

Manifestations of Non-linear Dependence on Temperature

In the following, we give examples where the strong non-linear dependence of thermal injury on temperature manifests itself in experimental trends.

Weak dependence on exposure duration

One example is the dependence of injury threshold as function of pulse duration, as shown in Figure 2 (injury threshold data from [8,9,10]).

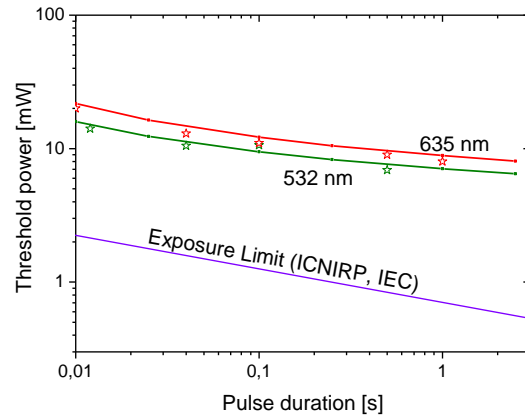


Figure 2. Experimental injury thresholds for retinal injury from laser radiation with two different wavelengths as function of pulse duration (stars) for minimal spot size condition. The lines are results from the computer model also used to calculate other damage data in this paper.

In terms of risk analysis and understanding of laser safety classes, the data shows that a power limit, such as 1 mW, is much more important than the associated exposure duration. In other words, the risk for injury can be drastically increased by only a relatively minor power increase, but the extent of the exposure duration (for instance somewhat longer than 0.25 s at 1 mW) is far less critical [11].

Short pulses, extended sources

For the case of a given pulse duration the retinal spot size is larger than the radial diffusion length, the centre of the retinal spot is not cooled before the pulse is over, and the temporal temperature profile does not reach the steady-state level. The temperature as well as the damage integral is shown for such an example in Figure 3a. The exposure shown in the plot comprises two pulses with 100 ms each, separated by 300 ms. When the second pulse commences, the temperature in the tissue has not yet returned to its initial value, thus the second pulse reaches a peak temperature that is a few degrees higher than the first pulse. The temperature profile for this case is presented for the threshold exposure, i.e. exposure to these two pulses leads (just) to injury. The development of the injury integral, Equation (1), is shown in Figure 3a. The strong non-linearity of thermal injury with temperature manifests itself most clearly in the discrepancy between the peak temperatures and the partial damage

values: although the peak temperature of the first pulse is only 1.6 degrees lower than the peak temperature of the second pulse, the first pulse only contributes about 23 % to the damage, i.e. at the end of the first pulse, the damage integral equals 0.23. The other 77 % are contributed by the second pulse.

Another manifestation of the strong non-linearity of thermal injury is that the steep increase of the damage integral only occurs shortly before the peak temperature is reached (best seen for the second pulse), and the damage integral also remains constant right after the pulse has finished, even though the temporal temperature profile is not that steep, neither in terms of temperature rise nor in terms of temperature decrease after the pulse. Thus it is mainly the peak temperature at the end of the pulse that is the main contributor to the damage integral, and the temperature needs to be only a little bit smaller (either before or after the pulse) so that it does not contribute to the damage.

A similar behaviour was noted for irregular pulses in our paper at ILSC 2009 [12], showing examples where one pulse with 30 % higher peak irradiances as the other smaller pulses dominated the damage process inasmuch as the other smaller pulses were irrelevant.

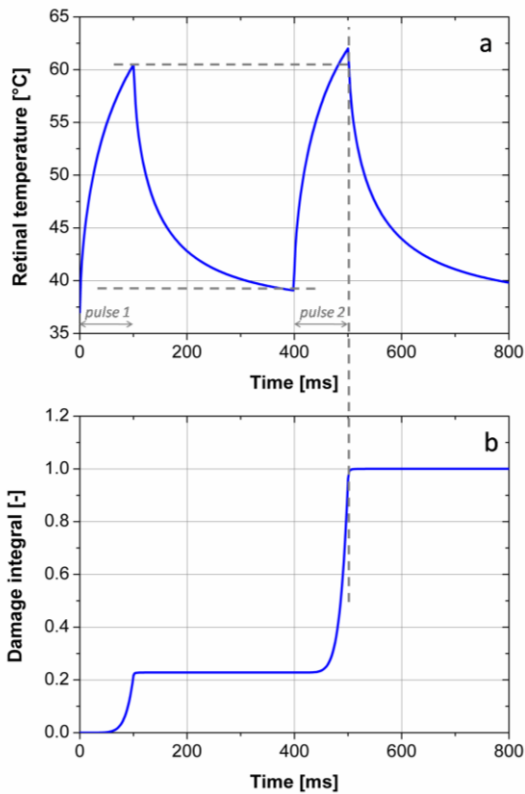


Figure 3. a) Temperature profile for two 100 ms where the steady state temperature is not reached (spot diameter 500 μm). b) temporal development of the damage integral.

Critical Temperature as Function of Pulse Duration

We have seen in the previous example that the cooling phase after the end of the pulse does not noticeably contribute to the damage integral, i.e. is not relevant for retinal injury. This was not always appreciated before, and it was somewhat erroneously believed that the longer cooling phase associated to larger spot sizes affected the injury threshold, and that the time-temperature history is critical. It was thought that larger spots are more hazardous because they take longer to cool off, extending the duration of elevated temperatures, as compared to small spots. However, the peak temperature necessary for producing a minimum injury is even higher for extended sources, as shown in Figure 4.

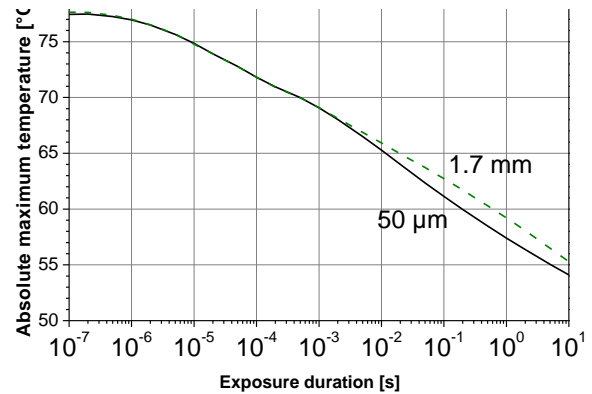


Figure 4. Temperature at the minimal visible lesion rim at the end of the pulse for threshold injury exposure to 532 nm radiation for two different retinal spot sizes.

This is because for small spots, a constant steady state temperature is reached during the pulse for pulse durations longer than about 10 ms, with a rectangular temporal profile. For extended spots, the temporal profile is rather triangular in shape as shown in Figure 3, so that relevant temperatures only occur for a short duration. Therefore, for extended sources, to result in the same Arrhenius integral value, a somewhat higher temperature (but only 2 °C higher) is necessary to compensate for the shorter duration of elevated temperature. (However, the irradiance and therefore the injury threshold to reach that temperature depends more strongly on the spot size, due to radial cooling affecting the centre of smaller spots earlier than larger spots, see for instance discussion in [5,6].)

Summary

We have shown in several examples how strongly thermal injury depends on temperature and that the dependence on exposure duration/pulse duration is often secondary, as is the cooling phase after the pulse. An understanding of this dependence helps to correctly interpret the exposure limits for difficult exposure scenarios such as for irregular pulses or retinal profiles.

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Meet the Authors

Karl Schulmeister, PhD, is a consultant on laser and broadband radiation safety at the Seibersdorf Laboratories, where also a specialized accredited test house is operated. Karl is a member of ICNIRP, the commission responsible for developing exposure limits for laser and broadband radiation on an international level. He is also the secretary of IEC TC 76 WG1, the working group responsible for IEC 60825-1. The research in his group over the last six years concentrated on thermally induced injury, providing the base for improving the spot size dependence of the retinal thermal exposure limits.

Mathieu Jean, MSc, is a PhD student registered at the Univ. Techn. Vienna, conducting his work at Seibersdorf Laboratories. He has optimized a retinal injury computer model and validated it against all available experimental injury thresholds, so that the model can be used for quantitative hazard and risk analysis of laser products (for instance scanned emission or irregular pulses). Mathieu also developed a computer model for laser induced injury of the cornea and the skin.