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High-Temperature Triggering of Soft-Tissue Regeneration by Er:YAG Laser

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SUMMARY

a) Introduction

In Er:YAG laser (λ = 2940 nm) procedures, it is the tissue's water content, not its pigment, that plays the role of an absorbing chromophore. Therefore, the temperature elevation ΔT is not laser-induced at locations of particular pigments, such as melanin or hemoglobin, but is limited to the superficially irradiated tissue layer, with the irradiation thickness determined by the laser's extremely short optical penetration depth (δ) [1-3].

During pulsed laser procedures, the resulting temperature pulse consists of the temperature ramp-up heating phase, during which the temperature reaches its maximal value (T_{max}), and of the cooling phase, during which the temperature returns back to its initial temperature T_0. The heating phase lasts for the duration of the laser intensity pulse (t_p), while the cooling phase is determined predominantly by the rate of the heat flow away from the heated superficial tissue. Here, Er:YAG is at a significant advantage since, due to its highest absorption in tissue water, the temperature decay time $t_d \approx \delta^2/D$ is extremely short (D is the tissue's thermal diffusivity) [3].

In this paper, we report on a measurement of the thermal pulse as induced by a short Er:YAG laser pulse (t_p = 0.3 ms) on the patient's skin. A special high-speed thermal camera (FLIR A6750 SLS, manufactured by FLIR Systems, USA) at a fast frame rate of 4000 Hz was used to detect the extremely fast temperature evolution during and following a single Er:YAG laser pulse generated by a Dynamis SP laser system equipped with a 5-mm full-beam R11 handpiece (both manufactured by Fotona d.o.o., Slovenia). The laser pulse fluence of F_p = 0.8 J/cm^2 was set to be just below the ablation threshold $F_{abl} \approx$ 0.9 J/cm^2.

b) Results

Figure 1 shows the measured temporal temperature profile at the skin surface, together with the simulated temperature profile during and following a pulsed Er:YAG laser irradiation. For the temperature simulation, a numerical model was applied of the physical process of soft-tissue resurfacing, as originally developed to study thermo-mechanical ablation with mid-IR lasers [3].

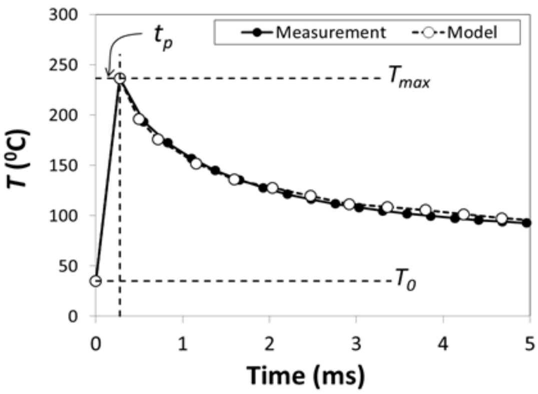

Fig 1: Simulated and measured thermal pulse as generated by a single Er:YAG laser pulse of t_p = 0.3 ms (Fotona Dynamis SP) with a pulse fluence of F_p = 0.8 J/cm^2.

The best fit of the numerical model to the measured temperature profile was obtained by using an optical penetration depth of $\delta \approx$ 4 μm. We attribute this slightly longer modeled penetration depth compared to what is considered to be the optical penetration depth of Er:YAG in skin, $\delta \approx$ 1-3 μm, to the fact that the software of the used thermal camera assumes a uniform body temperature, and therefore the measured temperature represents a weighted average of the skin temperature within the longer penetration depth of the detected thermal radiation.

c) Discussion

As can be seen from Fig. 1, for fluences slightly below the skin ablation threshold, the measured temperature reaches its maximal value at $T_{max} \approx$ 250 ^{0}C. This is in good agreement with our numerical model predicting T_{abl} = 256 ^{0}C, and as well with other reports [3-6].

Even though the measured maximal temperature is much higher than the critical temperature for irreversible damage of around 55-65 ^{0}C, as given by the standard Arrhenius model of skin damage [7,8], no blistering or other appreciable thermal damage to the skin is observed during "cold" Er:YAG laser resurfacing. This can be explained by the extremely short duration of the skin's exposure to the elevated temperatures, on the order of only 1 millisecond (See Fig. 1). Namely, for extremely short exposure times, the critical temperatures have been found to be significantly higher (above 260 ^{0}C) than what would be expected from the standard single-process Arrhenius model [4, 9-11].

The generated extremely short "heat shock" pulses are a unique and valuable characteristic of Er:YAG laser treatments. Namely, when using any laser to create a laser pulse, the duration and the shape of the resulting thermal exposure pulse within the tissue typically do not follow the duration and the shape of the delivered laser pulse. Therefore, even if the laser pulse duration is short, the volume of the heated tissue is typically relatively large, and the major mechanism by which this large volume cools down is the relatively slow diffusion of the deposited heat into the surrounding unheated tissues. It is therefore the thermal diffusion rather than the temporal pulse width of the delivered laser pulse which typically sets the lower limit for the achievable exposure time. Typical cooling times of the laser-irradiated tissues are therefore in the order of seconds or longer. This limits the treatment temperatures for the regeneration of the tissue to about 45 to 70 ^{0}C.

Extremely short-duration Er:YAG laser heat shock pulsing is therefore made possible by this laser's uniquely short absorption length ($\delta \approx 1$ μs), resulting in a very thin heated tissue volume, leading to extremely short temperature decay times $t_d \approx \delta^2/D$. It is to be noted that for a CO_2 laser, with δ being already approximately 10 times longer, short duration (≈1 ms) heat shocks are not possible, and the safe maximal treatment temperatures are limited to below about 120 ^{0}C, which is significantly below the skin ablation temperature of T_{abl} = 256 ^{0}C encountered during ablative laser resurfacing. This may explain the reported higher occurrence of complications following CO_2 laser resurfacing [12], as compared to Er:YAG laser resurfacing.

d) Conclusions

The intense heat shocks with maximal temperatures up to ≈250 ^{0}C, that can be safely delivered to the epithelium tissue using Er:YAG laser, may represent an additional mechanism of action for the reported excellent clinical effects of Er:YAG laser resurfacing [4, 13, 14]. This indirect tissue-regeneration triggering mechanism is complementary to the direct slow thermal stimulation of deeper lying fibroblasts and other cells, and is based on stimulating signal transduction processes for transcription factor activation, gene expression and fibroblast growth, thus leading to new collagen and extracellular matrix formation [4, 15-21].

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