Periocular CO2 laser resurfacing: severe ocular complications from multiple unintentional laser impacts on the protective metal eye shields

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Periocular CO₂ laser resurfacing: severe ocular complications from multiple unintentional laser impacts on the protective metal eye shields

Short title: Ocular complications of CO₂ laser resurfacing

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Conflict of Interest Disclosures: All authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

Background and Objectives: A 36-year old woman underwent CO_2 laser resurfacing for periocular rhytides using protective stainless steel Cox II ocular shields. Immediately after the treatment, corneal lesions were seen in both eyes. The left ==eye subsequent developed== corneal ulceration and scarring, a deformed iris, cataract and lower eye lashes showing signs of acute burns. The right cornea had a small inferior mid-peripheral superficial lesion and concomitant lower mid-peripheral burned eye lashes. Our objective was to determine the most likely cause of these ocular complications.

Study: We estimated temperature-time combinations that could induce corneal injury and cataract. Heat conduction effects from a heated cornea to the lens and from a heated ring of periocular skin to the cornea were computed. The temperature response of a shield following CO_2 laser irradiation was determined.

Results: We computed that cataract can develop when the corneal temperature reaches e.g. 80 °C for 14 sec. A periocular ring of heated skin contributes little to the corneal temperature. After 7 pulses of consecutive CO_2 laser bursts in 7.5 s, the total shield area already reached a homogeneous temperature of 63 °C.

Conclusion: Despite uncertainties in procedural details and modeling of cataract temperatures, the eye injuries were caused beyond doubt by heating of tear-covered metal eye shields by at least 10 consecutive but unintentional laser impacts.

Key words: CO_2 laser; periocular skin resurfacing; ocular complications; temperature-time predictions of cataract formation; corneal melting; burned eye lashes; metal eye shields

INTRODUCTION

Fractional carbon dioxide (CO_2) laser treatment has become a common procedure to treat various cutaneous conditions, including periocular rhytides. Sporadically, dermal side effects and complications occur [1] whereas very few publications address ocular complications [2-8]. We present a patient who was treated for periocular rhytides with a CO_2 laser. Immediately after the treatment, her vision was decreased and the eyes were irritated. Although both eyes had been protected by stainless steel Cox II shields, corneal clouding was seen in both eyes, with subsequent corneal melting and scarring in the left eye, with a deformed iris and the development of cataract. The lower eye lashes showed signs of acute burns. The right eye was less severely damaged.

Our objective was to determine whether heating of the metal eye shields by absorption of unintentional CO_2 laser impacts and/or heat conduction towards the cornea and the lens from a heated ring of periorbital skin by intentional CO_2 laser impacts could explain the complications. Our 2nd aim was to review the literature on this complication.

To achieve the 1st objective requires calculating: (1) temperature-time combinations that result in (a) irreversible injury of the cornea, and (b) lens injury that leads to cataract; (2) temperature effects of heat conduction from (i) a hot cornea to the lens and (ii) a hot ring of periorbital skin to the cornea; And, measuring the temperature response of a Cox II eye shield to CO_2 laser impacts.

Challenging was that neither sufficient clinical details were available nor metal eye shield temperatures when hit by laser impacts, and that temperature histories of thermal lens cataract were unknown. This complication was handled by combining measured shield temperatures after laser impacts with analysis of existing cataract data and considering scenarios that comply with the clinical procedure.

MATERIALS AND METHOD

Calculating heat diffusion from a hot cornea to the lens and from a hot ring of periorbital skin to the center of the cornea

For the thermal representation of an eye we used a sphere of water with a diameter of 2 cm and a thermal diffusivity of 1.5×10^{-7} m^2/sec, very close to the value of water (1.43×10^{-7} m^2/sec) and the value often used for tissue (1.77×10^{-7} m^2/sec [9]). We assumed that the anterior surface of the lens is 2 mm away from the corneal surface. Because heating of the cornea occurred by a heated metal eye shield and/or by heat conduction from the heated periorbital skin, the temperature of the cornea is unknown. Therefore, we used the corneal

temperature as a parameter that we varied between 60 and 100 °C. The initial lens temperature was 37 °C (Table 3 of [10]). The temperature rise of the anterior lens capsule caused by heat conduction from the hot cornea follows by solving the bio-heat diffusion equation in this cornea-lens geometry using commercial package Comsol®.

The effect of heat diffusion from a series of laser spots on the lower orbital rims was (over)estimated by taking a fully filled circumferential *ring* of periorbital skin that is instantaneously put at temperature levels between 60 and 100 °C, kept constant during 30 s; after that, the eye cooled down (Fig. 1). We used Comsol® again to calculate the temperature of the corneal center (Fig. 1). Because actual laser bursts are given consecutively and placed mainly on the lower rim, this approach obviously leads to overestimated temperatures compared to the original treatment. This approach was used for convenience to show (below) that even these exaggerated corneal and thus also on lens temperatures are small.

Measured temperature response of a Cox II eye shield to CO_2 laser impacts

We measured the temperature response of a Cox II stainless steel eye shield at the concave side in response to CO_2 laser impacts on the convex side. First, freshly-clotted blood was placed on the convex side of the metal shield to mimic the absorption of laser impacts by the tear film that exists on the shield during actual treatment. Tear has a thermal diffusivity close to that of freshly-clotted blood which is for 84% made up of water [11]. Second, we applied a honeycomb CO_2 laser pattern of 9 mm width and length, 1.3 mm spot diameter, 0.225 Joule per individual pulse, a pulse frequency during formation of the honeycomb pattern of 100 Hz, spot density 4 (i.e. 100% surface density of the laser spots), with a close to 1 Hz repetition frequency for consecutive honeycomb patterns. Third, we measured the temperature during and after multiple CO_2 laser impacts on the Cox II eye shield using a thermal camera (Seek Thermal, Comact XR). We included a black matted surface (Thorlabs BFP1) at the concave side of the metal to increase the emissivity of that surface. Finally, calibration was performed by testing at temperatures of 0 and 100 °C.

RESULTS

Clinical case

A 36-year old woman was treated with the UltraPulse CO_2 laser (Lumenis, Dreieich, Dreieichenhain, Germany) for periocular rhytides at a cosmetic clinic according to a standard protocol. The patient was placed horizontally, and an

anesthetic cream (lidocaine 23 mg/ml, tetracaine 3.5 mg/ml) was applied to the periocular skin for 45 minutes. After removal of the cream, oxybuprocaine 0.4% eye drops and oculentum simplex ointment were applied to both eyes. Subsequently, stainless steel ocular shields (Type Cox II, size 25.5 x 22 x 1 mm, Oculo-Plastik Inc, Montréal, Québec) were placed, covering each eye, which caused distress to the patient. She was placed in a more supine position. For our analysis below it is essential that the eye lids under such circumstances never spontaneously close and hence do not cover the total area of the metal shield. The periocular skin was then treated with the laser in the Active FX mode using the CPG-scanning hand piece. The laser beam had a honeycomb pattern with an individual spot size of 1.3 mm, with energy per pulse of 0.1 J, frequency of 100 Hz and pulse duration of <1 ms. For most of the procedure a spot density 3 (82% surface coverage of the individual pulses) was used and a spot density of 4 (100% coverage) for a row of second passes along the lower orbital rims.

During the procedure, which lasted 10 to 12 min, the patient did not indicate any problem. After removal of the eye shields, the patient indicated irritation and blurred vision in both eyes, which were consequently rinsed with sodium chloride 0.9% solution. Examination showed conjunctival hyperemia in both eyes and a white opacity of the left cornea. As her pain increased, she was immediately referred to the Ophthalmology Department (Leiden University Medical Center). At presentation, the best-corrected visual acuity was 0.6 (Snellen) in the right eye and 0.2 in the left eye (normal is about 1.0). The right cornea (Fig. 2A) showed greyish epithelium with a small inferior mid-peripheral superficial grey-white opacity with positive fluorescein staining. The left eye showed a superficial and deep intense white opaque cornea, positive for fluorescein staining, with stromal edema, limbal ischemia and a dilated pupil; the natural lens was not visible (Fig. 2B). Patient underwent superficial corneal debridement in both eyes, which failed in the left eye due to the rigidness of the superficial corneal layer. Both eyes were treated with dexamethasone-tobramycin ophthalmic ointment, cyclopentolate 1% eye drops, serum and cyclosporine eye drops, vitamin C tablets and oral doxycycline. After five weeks, the right eye had recovered with a remaining minor midperipheral corneal opacity; visual acuity was 1.25. The left eye had a visual acuity of 0.02. Slit-lamp examination showed a persistent thickened cornea with a central epithelial defect, dilated pupil, deformed iris with a curled-up pupillary rim. The lens showed cataract with an anterior subcapsular star shape, a well-known ophthalmic characteristic of thermal damage. During the whole period, the eye pressure of both eyes remained within normal limits.

Two years later, she underwent cataract surgery on the left eye (Department of Ophthalmology, Maastricht). The final visual acuity at 35 months was 1.25 in the right eye, with a clear anterior segment, and 2/300 (hand motions at 2 m

distance) in the left eye, with an intense white diffuse stromal scar with corneal vascularization and central stromal contraction and thinning, corneal flattening, a permanently curled-up pupillary rim and dilated pupil. For her left eye she received a cosmetic contact lens with iris coloring. Seven years later (Fig. 2C,D), the only treatment of her left eye consists of tear substitutes (Fig. 2D).

Temperature-time combinations that irreversibly injure the cornea and that produce a thermal cataract

Assuming a normal corneal temperature (35 °C, [12]), Fig. 3 shows the temperature-time behavior for corneal injury as measured in rabbit eyes [13]. Data from 181 humans [14-16] showed a similar corneal temperature of 34.8 °C and, although the size of the eyes and the eye lids are quite different, we have used Fig. 3 as a reasonable approximation for human corneas.

In the literature, we found only two temperature histories that produced a cataract in rabbits (Fig. 2 of [17]). Luckily, this allows deriving a generic temperature history of cataract formation by Arrhenius theory (Appendix A), with the results also shown in Fig. 3.

Heat diffusion computations from the hot cornea to the lens and from a hot ring of periorbital skin to the center of the cornea

A curled-up iris and the typical aspect of the anterior lens implied that a thermal cataract had developed. From Fig. 3, the duration of the corneal temperature that predicts a cataract (open circles in Fig. 4) can be estimated. The computed anterior lens temperatures of cataract development in response to cornea temperatures of 60 to 100 °C are also shown in Fig. 4. The corneal temperature-time combinations found are: 60 °C-30 s; 67 °C-23 s; 70 °C-20 s; 80 °C-15 s; 90 °C-11 s and 100 °C-10 s. The combination 67 °C-23 s will be used in the Discussion (*Study limitations*) to estimate the effects of using 0.1 J per pulse instead of 0.225 J.

The computed temperature increase at the center of the cornea in response to an instantaneous hot ring of periorbital skin, of 3 mm thickness (Fig. 1), with exposure to temperatures between 60 and 100 °C during 30 s and cooling thereafter, varied between 4.4 and 11.5 °C. As stated earlier, these corneal temperature effects overestimate the actual behaviors since laser bursts are given consecutively and mainly on the lower rim. Therefore, periorbital laser impacts along the lower rim cause a few degrees increased corneal temperature only, hardly affecting the anterior lens temperature.

Measured temperature response of a Cox II eye shield to CO_2 laser impacts

The presence of a tear film on the metal shield is an essential aspect of this

laser treatment, because CO_2 laser light significantly absorbs in water. Local irritation and oxybuprocaine eye drops will stimulate tearing and the formation of a tear film. Therefore, experiments have been conducted with blood clotted on the metal shield; blood mimics the water film and is obviously easier to handle. Figure 5 shows the measured temperatures of a Cox II eye shield in response to 19 CO_2 laser honeycomb impacts ("plus" signs on the horizontal axis). First, we used a sterile Cox II eye shield. Second, we added blood drops to the convex part of the shield, waited 1 minute until the drops were clotted, and irradiated the eye shield area with the clotted blood on its surface. As blood contains about 84% of water [11], we estimated the temperature response to 19 laser bursts of the eye shield with water (representing a tear film), by multiplying the data with clotted blood by 1/0.84 = 1.19. After 7 pulses in 7.5 s, the total shield area already reached 63 °C. Subsequently, the shield area retained a uniform temperature, also during cooling (137 sec).

DISCUSSION

Most likely scenario that caused the corneal injuries

We questioned how 10-20 consecutive CO_2 laser pulses could have had such an impact on the left eye. The pulses were part of a normal procedure and we hypothesized that the 2nd pass on the left lower orbital rim had a more upward directed CO_2 laser hand piece, perhaps because the patient's head was directed slightly more upward than normal, and that every CO_2 laser burst impacted on the eye shield. The exceedingly large absorbance of CO_2 laser light by tear-covered stainless steel would have caused a significant temperature rise of the metal shield. The shield's excellent thermal properties would lead to spreading of the heated area over the total metal (and thus corneal) surface within 7.5 s (Fig. 5, dashed line *"% Heated Area"*). This hypothesis implies that the lower eye lashes should be burned. Because examination of the eyelids showed that this was indeed the case (Figs. 2A,B), we believe that placement of multiple unintentional CO_2 laser impacts on the metal eye shields is the most likely scenario that caused the ocular complications in this patient. Heat conduction from the hot cornea also heated up and injured the iris and caused a cataract. We emphasize that intraocular damage following this CO_2 laser procedure has not been described before.

This paper must create awareness for all physicians who perform periocular CO_2 laser resurfacing procedures that unintentional CO_2 laser irradiation of metal eye shields, despite protecting the cornea against direct laser impact, has the capability of significantly heating up these shields (see e.g. [18]). This effect is

even intensified by the tear film (Fig. 5), a mechanism that has not been described before.

Study limitations

Unfortunately, actual treatment details are unknown, e.g. whether the 2nd pass around the left lower orbital rim occurred immediately after the 1st pass and whether the 1st pass of laser bursts also hit the eye shield. If that were the case, the 2nd pass would have started with an already increased shield temperature. For simplicity, we assumed that the first pass laser bursts missed the eye shield.

The way we modeled laser impacts on the periorbital skin led to overestimated responses. Since the resulting temperature rises were found to be negligible, the extent of overestimation is irrelevant. Also, the Arrhenius parameters for thermal cataract formation from only two experimental temperature-time combinations (Appendix A) have limited accuracy. However, the derived temperature-time curve (Fig. 3) is the only one available to date that describes cataract formation from an increased lens temperature. The outcome, that these cataract-causing temperatures are below those of corneal damage temperatures suggest that the prediction is meaningful; the opposite would have contradicted the observed damage events.

Cooling of the metal eye shield held in air (Fig. 5) is (much) faster than in the clinical situation, placed on the eye ball and intermittent layer of oculentum simplex, which has isolating properties (a 2.5 times smaller thermal diffusivity, Appendix B). Thus, it takes quite a long time before a temperature rise of the cornea has diffused away, contributing to corneal thermal damage and cataract formation at sufficiently high temperatures (Figs. 3 and 4).

The CO_2 experimental laser parameters (0.225 J) differed from those used clinically (0.1 J). However, the estimated temperatures of the Cox II shield with added water (Fig. 5) in response to 0.225 J laser pulses, can be easily transformed into temperatures from 0.1 J pulses by dividing temperature increase by 2.25. After 19 pulses, this would give a temperature of about 67 °C instead of 107 °C. Then, 70 °C would have been reached after 24.5 s, thus after 21 pulses. However, a longer time duration also lowers the critical temperature for corneal injury and cataract formation (Fig. 3). From Fig. 4, one can see that development of a cataract occurs when the cornea temperature is about 67 °C during about 23 s.

Incidence of this complication

This thermal complication either seems to be extremely rare or has been underreported in the literature as we found only five papers on this subject. In three, the authors described the thermal cause [2], one written in Korean [6], the 3rd [3] gave a 0.3% incidence of corneal injury after CO_2 laser blepharoplasty,

however, without details. In the other two [5,8], we believe the cause was thermal but the authors proposed other mechanisms.

Other possible causes

The review by Blanco et al. [4] did not refer to Widder et al. [2] and Christian et al. [5] but included many other mechanisms. Chemical damage can occur when the cream to anesthetize periorbital skin reaches the cornea, or when corneal eye shields are cleansed with chlorhexidine prior to insertion [5]. Thermally-burned eye lashes, eyelid skin and eyebrow hair have been described in a patient who was wearing mascara while cautery was applied to her eyelid lesion after excision [7]. Although our patient has tattooed eyeliners (Fig. 2), these lines were not damaged.

CONCLUSION

Despite uncertainties concerning procedural and cataract modeling details, our experiments on the Cox II shield (Fig. 5), the burned lower eye lashes (Fig. 2A,B), and the supporting heat diffusion computations have shown beyond doubt that consecutive CO_2 laser impacts hit both eye shields caused the left total shield area to reach a high temperature, probably over 70 °C, followed by a slow cooling rate. Heat conduction (Fig. 4) caused curling-up of the iris and a thermal cataract. The right shield also received CO_2 laser impacts but less than the left shield and only around the central part of the eye, shown by mid-peripheral corneal damage and burned lower middle eye lashes.

Few treatment options exist for thermal ocular injury. Our case thus emphasizes the importance of knowing the pitfalls of periocular CO_2 laser resurfacing procedures and, especially now that fractional CO_2 laser resurfacing is gaining in popularity, suggests adapting the safety guidelines with respect to the use of metal eye shields. Particularly, the eye shields should not only be positioned properly but the position of the CO_2 laser beam, visible by the pilot beam, should also be cautiously monitored as long as the laser is switched on.

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APPENDIX A. ARRHENIUS THEORY OF CATARACT FORMATION

We combined the two temperature-time $(T - \tau)$ combinations that produced a cataract in rabbits (Fig. 2 of [17]): 43.5 °C during 10 min and 41.5 °C during 20 min with Arrhenius kinetic rate theory ([19], Eq. 13.42), using

$$\ln(\tau) = \frac{E/R}{T} - \ln(A) \tag{1}$$

E is activation energy [J/mole], R the universal gas constant [8.3143 J/mole/K], T the temperature in Kelvin (0 °C is 273.15 K), and A the frequency factor [s^{-1}]. From the two data points the two unknowns in Eq. (1) follow as $E/R = 34{,}530.5$ K and $\ln(A) = 102.65$. The $(T - \tau)$ curve for a cataract is also given in Fig. 3 (lower curve).

APPENDIX B. THERMAL DIFFUSIVITY OF OCULENTUM SIMPLEX

We used the available chemical composition and thermal data of the components, i.e., 40% paraffin (thermal conductivity k = 0.25 W/m/°C, thermal diffusivity α = 0.081x10⁻⁶ m²/s), 51.5% vaseline (α not found, k = 0.18 W/m/°C), 6% lanoline (α = 0.053x10⁻⁶ m²/s), and 2.5% cetostearyl alcohol (here we used glycerol, α = 0.01x10⁻⁶ m²/s). Assuming that α is proportional to k (implying identical product of density and heat capacity) gives α = 0.064x10⁻⁶ m²/s for vaseline. Then, based on the composition, an estimated thermal diffusivity of oculentum simplex is: α ≈ (0.4x0.081 + 0.515x0.064 + 0.06x0.053 + 0.025x0.143)x10⁻⁶ = 0.07x10⁻⁶ m²/s. Tissue has about 0.18x10⁻⁶ m²/s thus oculentum simplex has about a 2.5 times lower thermal diffusivity than tissue.

CAPTION TO FIGURES

Fig. 1. Eye (blue sphere) with a ring-shaped skin layer of 3 mm thickness and 9 mm length around the orbital rim representing the periorbital skin (indicated in green), which was kept during the simulations at 60 to 100 °C during 30 s (indicated by a reddish line), and the metallic shield of 1 mm thickness in the middle area (dark blue layer).

Fig. 2. (A) Right eye, directly after treatment. Light burning of the lower middle lashes and perilimbal redness. (B**)** Left eye, directly after treatment. Severe burning of the lower eye lashes at the side of the nose and slight burning of all other lashes. The central cornea shows epithelial and stromal clouding. **(C)** Right eye seven years after treatment. **(D)** Left eye seven years after treatment. N.B. This patient has tattooed eyelines around both eyes.

Fig. 3. Temperature-time duration curves derived from rabbit experiments that predict (a) irreversible thermal corneal injury, using 35 °C as normal corneal temperature [10], and (b) a thermal cataract, Eq. (1).

Fig. 4. Temperature of the anterior lens, at 2 mm inside the eye, assumed at 37 °C, as a function of the time of heat conduction duration for several corneal temperatures (60 °C to 100 °C). The temperature-time points that will predict the development of a cataract are indicated as circles.

Fig. 5. Measured temperatures during heating and cooling of the Cox II shield in air in response to 19 CO_2 laser bursts (black lozenges) at 0.225 Joule, about 1 Hz per burst, 1.3 mm spot diameter, 9 mm width/length honeycomb pattern, about 100 Hz for individual pulses. Time points of the bursts are indicated by "plus" signs on the horizontal axis. At the right side (black dashed line, "% Heated Area (19 pulses with blood"), we plotted the area of the shield (in %) with an increased temperature. Also shown are measurements without added blood (black circles), and the computed estimated curve of the eye shield with water added (thick black dashed line), assuming whole blood contains 84% of water [11].
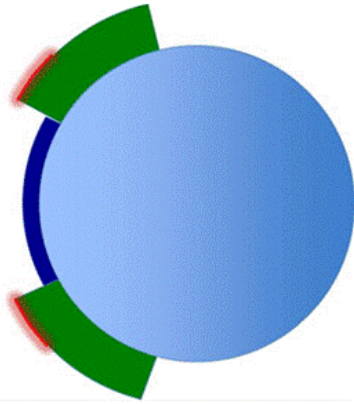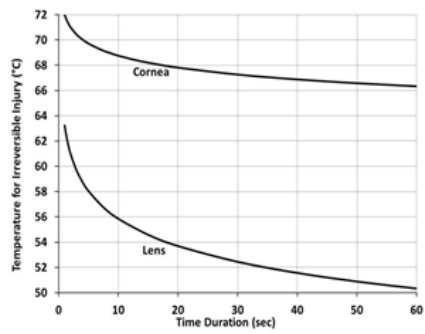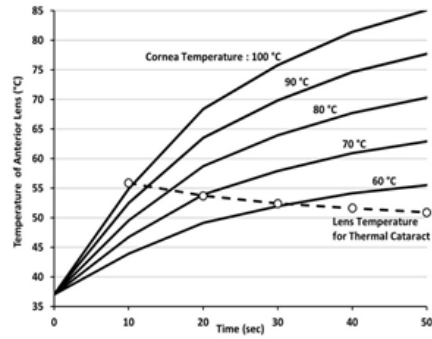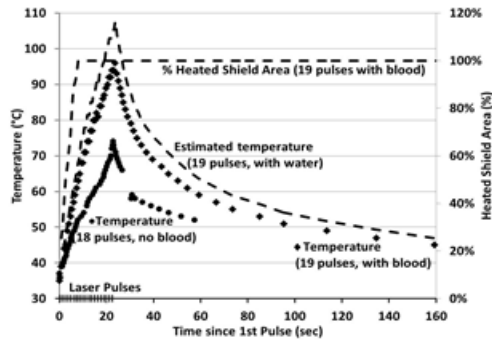
Fig.1

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Fig.3

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Fig.5

REPLY TO THE COMMENTS OF THE 2 REVIEWERS

Reviewer 1:

I read, with distress, the manuscript, Periocular CO2 laser resurfacing: severe ocular complications from multiple unintentional laser impacts on the protective metal eye shields. This manuscript must be published. It is a shame that it has taken seven years to get this information out.

Thank you. We agree.

One can question the assumptions made in the simulations. However, the clinical results are real. The authors are careful to state that they are simulating events long after they happened with a lack of detail. Overall, the work is carefully and conservatively presented.

Thank you.

There are a couple of questions that I have:

1. In the Materials and Methods, the thermal diffusivity of the eye is given as 1.5 x 10-7 m2/sec. The source for this value should be cited. If this is not a measured quantity, how the authors estimated this value should be explained.

Thank you. We changed it as: "--- we used --- a thermal diffusivity of 1.5×10^{-7} m^2/sec, <u>very close to the value of water (1.43×10^{-7} m^2/sec) and the value often used for tissue (1.77×10^{-7} m^2/sec [9])</u>."

The new Ref 9 is: "van Gemert MJC, Welch AJ. Approximate solutions for heat conduction: Time constants. In: Welch AJ, van Gemert MJC (Editors). Optical-Thermal response of laser-irradiated tissue, Chapter 13, page 425, Plenum Press, New York, 1995."

2. In the Results and under "Temperature-time combinations that irreversibly injure the cornea and the produce a thermal cataract" the authors make the conclusion that the time dependence of a temperature variation measured in a rabbit is the same as the time dependence of a temperature variation in a human is the same because the initial temperatures are the same. This conclusion cannot be made. The geometry (size) of the eyes are quite different, as are the sizes of the eye lids. I can accept using the rabbit eye parameters as a reasonable approximation…but it should not be stated that the initial temperatures prove a correlation between the two different eye systems.

Thank you.

We changed it as: "Assuming a normal corneal temperature (35 °C, [11]), Fig. 3 shows the temperature-time behavior for corneal injury <u>as measured</u> in rabbit eyes

[13]. Data from 181 humans [14-16] showed a <u>similar</u> corneal temperature of 34.8 °C <u>and, although the size of the eyes and the eye lids are quite different, we have used</u> Fig. 3 <u>as a reasonable approximation for human corneas.</u>"

Otherwise, this was a very carefully done evaluation and I complement the authors.

Thank you very much indeed.

Reviewer: 2

I have carefully studied this interesting accident report and the authors' attempts at an experimental simulation. However, I think the authors have made some seriously erroneous assumptions that places the validity of their study into serious doubt. Since lids of the patient should have been closed and 10.6 µm laser radiation should not penetrate the lids.

No, the lids of the patient were NOT closed.

We added (RESULTS, Clinical case, page 5): "<u>For our analysis below it is essential that the eye lids under such circumstances never spontaneously close and hence do not cover the total area of the metal shield.</u>"

One of the dermatologic authors (AW) regularly uses eye shields and he states that the eye lids tend to open spontaneously except if the patient purposely pushes them to become closed but even then eye lids often reopen. Even companies that produce eye shields don't state the necessity of keeping eye lids closed just because they assume that eye shields always protect adequately.

The Cox II shields are meant to be under the lids and not directly irradiated.

We agree.

Certainly, multiple (19 bursts) direct aiming of a beam at the shield is simply not plausible by a trained operator.

The operator was actually very experienced and we obviously don't know how this became possible.

As with all too many accident cases, there certainly could be a misrepresentation of the facts the laser user who does not wish to seem to be incompetent or at serious fault.

Incompetent: no. At serious fault: yes.

The authors should wonder why routine sauna exposures for 15 minutes at moist air temperatures of 90 C (good conditions for heat transfer) do not result in cataracts of millions of Scandinavians.

Interestingly, an article published on February 2nd in the NRC (a Dutch major newspaper) with title: "Why don't you get burned in a sauna of 90 degrees" referred to a physician in one of the Dutch burn centers who stated that surviving these air temperatures is possible because of the huge difference that exist between the thermal properties of air and water. Our body can handle high air temperatures due to the very small heat conduction of air combined with cooling of the skin by vaporization of its sweat. Also, inhaling of hot air is possible because of the cooling by passing through the nose and upper airways. However, if some water is thrown on the hot stones you immediately experience this as heating. They ended with the example of a contender of the World Sauna Championship who died following a stay of a few minutes in a sauna of 110 degrees where every 30 sec some water was thrown on the stones.

Because this subject is unrelated to our case we have not tried to include it in the manuscript.

However, I think the authors have made some seriously erroneous assumptions that places the validity of their study into serious doubt.

We would have expected that a reviewer who makes this statement (actually incorrect, see our 1st reply), would have provided an alternative mechanism that also explains the total thermal destruction of the full area of the cornea, the clinical reality here.