Geerts, Greta A.V.M. (2010). Temperature changes along a dental implant. THE INTERNATIONAL JOURNAL OF PROSTHODONTICS, 24 (1): 58-68

Temperature changes along a dental implant

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Abstract

Aims: The aims of this *in vitro* study were to analyse temperature changes along the surface of a dental implant and to establish the abutment temperature that could cause the critical 47ºC/1min threshold at implant level. **Methods**: Eight thermocouples were attached at 1 mm intervals to an abutment/implant configuration. The model consisted of 2 compartments in a thermostatically controlled environment. The upper compartment represented the oral cavity with the abutment, which was exposed to 20ml of hot water. The temperature at each thermocouple was logged over a period of 10 minutes. A Spearmans Rank correlation test and logistic regression model were used for the statistical analysis of the time/temperature databases and the estimation of the 'effective dose 50' for the abutment (95% confidence interval). **Results**: For 53 test series, the abutment temperature ranged from 52.80ºC to 71.72ºC. There was a positive correlation between the maximum temperature at implant level and the temperature of the abutment. The 47ºC/1min threshold was reached 31 times at the most cervical implant level and decreased in frequency further away from the heat source (14, 6, 3, 1 and 1 times resp.) The ED50 was estimated at 62.3ºC. This means that for an abutment temperature of 62.3ºC there was a 50% chance that 47ºC for 1 minute at implant level would be reached. **Conclusion**: This *in vitro* study supports the hypothesis that abutment temperature is transmitted to an implant. Although results of *in vitro* studies should be interpreted with caution, clinicians should be aware of temperature changes along implants and the potential risk associated with it.

Keywords:Dental implant, Heat transmission, Bone necrosis

Introduction

Successful osseointegration depends on the correct surgical technique and appropriate prosthodontic management. Overheating during implant site preparation is a well recognized cause of implant failure due to lack of osseointegration.¹ The threshold for "irreversible enzymatic disturbance to cortical bone" is reported to be 50ºC for 30 seconds. ² An *in vivo* animal study demonstrated that thermal bone injury occurs at a lower temperature: 47ºC for 1 minute. ³ Results from an *in vitro* model using rat osteoblasts were comparable to these results: transient changes in osteoblasts were noticed at 42ºC and the critical temperature inducing cell death was 45-48ºC. ⁴ The temperature-time ratio of 47ºC/1min as reported by Eriksson and Albrektsson in 1983 is routinely used as a threshold in research studies.

Single-stage implant surgery developed from the traditional 2-stage surgery in search for less intervention and faster implant treatment. This results in the freshly placed implant being exposed to the oral cavity during osseointegration. Implants and their superstructures, often metal, could be considered good heat conductors. Few publications deal with transmission of temperature from a heat source in the mouth to more apical levels along a dental implant. The majority of the studies are *in vitro* models. However, all of them confirm that temperature is transmitted from abutment to fixture, but not necessarily reaching the critical time-temperature threshold to cause thermal bone injury at fixture level.

Direct application of autopolymerizing acrylic resin to an implant abutment caused a maximum increase of 6ºC in temperature, sufficient to cause cervical bone damage. ⁵ A computer-simulated model by Wong et al. (2001) showed that a 60ºC heat source caused a "heat front of over 47ºC to advance 3mm along an implant within 1 second". ⁶ Kreisler et al. (2002) investigated temperature increases at implant-bone level during simulated surface contamination of a cervical peri-implant bone defect using laser.⁷ They reported that power output and time need to be controlled carefully to prevent rapid heat generation that could reach the 47ºC threshold even at an apically located bone-implant interface site. A cervical temperature increase of 10º-13.8ºC for 50 seconds was reported when impression plaster was applied on implants. ⁸ This approaches the threshold value of 47ºC for 1 minute, potentially compromising adjacent bone. Using a bovine *ex vivo* model, Feuerstein et al (2008) measured temperatures above 57º at implant abutment level and inside the implant.⁹ At lower levels of the implant, they recorded temperatures reaching the 42ºC threshold for transient changes. A consecutive *in vivo* pilot study by Ormianer et al. (2009) confirmed a linear correlation of 1) abutment and abutment/implant interface temperatures and 2) abutment and implant cavity temperatures.¹⁰

Intraoral temperatures vary during routine daily activities such as the intake of food and fluids. Drinking hot water may raise the intraoral temperature to $67^{\circ}C^{11}$ and even to $77^{\circ}C^{12}$. Moore et al. (1999) recorded temperatures ranging from 5.6º to 58.8ºC at the upper incisor site and from 7.9º to 54ºC at the upper premolar site over a 24-hour period.¹³ They also reported that changes in oral temperature occured rapidly, while the return to baseline temperature occured more slowly. Feuerstein et al. (2008) reported a maximum intraoral temperature of 76.3ºC for hot beverage consumption and 53.6ºC for hot food.⁹ The aims of this *in vitro* study were to analyse temperature changes along the surface of a dental implant by exposing the abutment to hot water simulating the temperature of hot beverages and to establish the abutment temperature that could cause the critical 47ºC/1min threshold at implant level. The nullhypotheses were: 1) the surface temperature of an implant is not affected by the

temperature of its abutment, 2) the critical 47ºC/1min threshold is not reached at any implant level regardless of the abutment temperature.

Materials and methods

A 3.75mm implant with a 5mm abutment (IBS15 and TB3N, Southern Implants, Irene, South Africa) was mounted in an *in vitro* model consisting of 2 compartments separated by a teflon membrane attached to the neck of the implant (Figure 1). The upper compartment received the 20ml of warm water, the lower compartment was thermostatically controlled to maintain the temperature of the implant at 37 ºC. Seven K-type thermocouples were attached to the implant model by means of epoxy adhesive (Pratley Steel, Pratley, Kenmare, South Africa) at the following sites: the implant abutment above the teflon membrane (channel 2), the implant collar below the teflon membrane (channel 3), and the other 5 thermocouples at 1mm increments apically (channels 4 to 8). The apical part of the implant was isolated with silicone putty and placed in a petri dish filled with water thermostatically controlled to maintain the temperature of the implant at 37ºC. This was achieved by means of a mini-heater element regulated by a proportional-integral-derivative controller (PID) (Rex-C100, RKC Instrument Inc., Ohta-ku, Japan). The PID controller had an independent sensor and regulated the temperature consistently. The entire model was housed in a custom built environmentally controlled chamber that maintained a temperature of 32ºC. Before each test, the test model was calibrated in an effort to simulate body temperature along the entire implant model. The thermocouples were connected to a data logger (Picolog Data Logger, Pico Technology, Cambridgeshire, United Kingdom). The data logger was connected to a computer via a USB cable. The computer was a Pentium 4, core 2 duo, 2 gig ram, 1.8 GHz processor. Data were captured using dedicated software (Picolog Recorder for Windows XP Professional version 5.13.9). One temperature recording at least every 3 seconds for 10 minutes was performed for each test. The data were copied into SASv9 for logistic regression analysis, and MSExcel for Spearmans Rank Correlation analysis.

Figure 2 identifies temperatures and time intervals strategic to the analysis of the results for channels 2 and 3. The same temperatures and time intervals were identified for each subsequent channel.

Results

Fifty three successful tests were performed. The average temperature recorded immediately before zero time (baseline temperature) for all channels was $36.9^{\circ}C$ (\pm 0.7 °C). The maximum abutment temperatures (max2) ranged from 52.80 to 71.72^oC, with an average of 63.30 ^oC and a median of 63.57 ^oC.

Table 1 represents the temperature ranges measured at each level; the shortest observed time to reach 47ºC (a-c) on each level for the tests that reached the threshold of 47ºC/1min and the number of tests for each channel that reached

the threshold of 47ºC/1min. The shortest time recorded to reach 47ºC was 62 seconds on channel 3 (thermocouple closest to the abutment), the longest time to reach 47ºC was 180 seconds on channel 8 (most apically placed thermocouple).

Using the Spearman Rank Correlation test, the following positive correlations were found: between max2 and maximum temperature at the first implant level (max3) (Figure 3); between max2 and reaching the threshold of 47° C/1min (hot2long); between max3 and the last time 47° C was recorded by channel 3 (end time); between max3 and the duration that the temperature was $\geq 47^{\circ}C$ (total_time47); between max3 and hot2long; between max3time and difference max2time and max3 time. The correlation between max2 and max3 associated with being hot 2 long is shown in Figure 4.

The following negative correlations were found: max2 and the time needed to reach 47^oC at implant level (time2_47); max₃ and time2_47; max₃ and the time that 47^oC was measured for the first time at implant level (start time); start time and total time47; end time and time2 47 ; and total time47 and time2_47.

Using logistic regression, the temperature of max2 at which there is a 50% chance that the temperature will exceed 47ºC/1min at the first implant level (ED50), with a 95% confidence interval, was estimated to be 62.3ºC (Figure 5). There was an estimation problem of increasing magnitude the further away the thermocouples were from the heat source. For this reason, a similar analysis was not repeated for channels 4 to 8.

Discussion

This study analysed the temperature changes along the surface of a dental implant following the application of warm water to its abutment. The first nullhypothesis cannot be accepted because the surface temperature of an implant *is* indeed affected by the temperature of its abutment. The second null-hypothesis can be partially rejected because the critical 47ºC/1min threshold *can* be reached at any implant level but it is dependent on the temperature of the abutment and there is an increasing time delay the further away from the heat source.

The positive correlations may be explained as follows: 1) higher abutment temperatures cause higher temperatures at implant level, 2) higher abutment temperatures cause a higher frequency in reaching the threshold of 47ºC/1min, 3) the higher temperature at implant level, the longer it takes to cool off below 47ºC, the longer it remains at 47ºC and the higher the chance to reach the 47° C/1min threshold, 4) the longer it takes to reach max3, the larger the time interval between max2 and max3. The negative correlations may be explained

as follows: 1) higher abutment temperatures create a shorter time to reach 47ºC at implant level, 2) a higher implant temperature is associated with a shorter time to reach 47ºC at implant level and, 3) the faster 47ºC is reached at implant level, the longer the temperature remains at 47ºC.

Table 2 illustrates that for more apically located levels, the number of occasions that the threshold of 47ºC/1min (hot2long) was reached decreased by a factor of about ½ for each 1mm increment along the implant. Within the limitations of this study, it may be concluded that the cervical part is most at risk in terms of temperature changes.

From the time/temperature graphs, it was noticed that the loss of temperature and the time delay between abutment and implant was larger than the time delay and loss of temperature among the different implant levels. This was not statistically analysed. However, this phenomenon may be explained by the slightly longer distance between thermocouples 2 and 3 than between the rest of the thermocouples and by the type of abutment-implant interface. The abutment-implant interface in this model consisted of an external hex connection. Different types of implant-abutment connections, as well as the abutment dimensions and materials may have an influence on heat transmission. This could be investigated further.

With the introduction of warm liquid in the upper compartment, the temperature of the abutment rapidly increases to reach a maximum and slowly returns to the baseline temperature (Figure 2). The temperatures at implant level also rise, but at a slower rate and they never reach the same level as the abutment temperature. Figure 4 shows that for a higher abutment temperature (max2), the temperature recorded at implant level was also higher and the chance to reach the threshold of 47ºC/1min at implant level increases. This is shown by the red dots concentrated on the right side of the scatter plot. This feature was present at all implant levels. However, for more apically located implant levels, less threshold values (hot2long) were reached (Table1).

The ED50 for the abutment temperature and channel 3 was estimated at 62.3ºC (Figure 5). This means that for an abutment temperature of 62.3ºC there is a 50% chance that the implant temperature will exceed the 47ºC/1min threshold. For a maximum abutment temperature of 61° C or lower, the 47° C/1min threshold was never reached at the first implant level. For a maximum abutment temperature of 64ºC or higher the 47ºC/1min threshold was always reached at the first implant level. These abutment temperatures are comparable with temperatures that have been recorded intra-orally.^{9, 11-13}

With time, the temperature values of abutment and implant tend to move towards the same value, suggesting a continuous exchange of energy among

abutment, implant and environment. The immediate environment of the abutment consisted of the upper compartment receiving the 20ml of warm water. Due to model constraints, the water could not be removed from the compartment as would happen *in vivo* during swallowing. Instead, the water was allowed to cool down in situ. Since the abutment temperature was always higher than the temperatures at implant level, this may have resulted in higher implant temperatures than if when the warm water was removed from the upper compartment. On the other hand, only a single dose of warm water was applied to the upper compartment. Drinking a hot beverage exposes the oral tissue to consecutive 'doses' of high temperatures. Although not as fast as previously reported in a computer model ⁶ , this study showed that there was a sudden temperature peak at abutment and implant levels at the time of exposure of the heat source. However, return of the temperature towards the baseline temperature took much longer. This confirms the findings of an *in vivo* study of Moore et al. $(1999)^{13}$ Because of this phenomenon, it would be interesting to study the cumulative effect of consecutive short applications of warm water to resemble the consumption of a hot beverage.

The immediate environment of the fixture was a) thermostatically controlled air at the level of the thermocouples, and b) silicon and thermostatically controlled water apically to the lowest thermocouple. This model differs from the intraoral situation where the fixture is in close proximity with soft tissue and bone. *In vivo* heat transmission might differ compared to this *in vitro* model. This is a study limitation and could be investigated further.

Within the limitations of this *in vitro* study, it is concluded that abutment temperature is transmitted to an implant and that the threshold value of 47 ºC /60sec can be reached at implant level. Although results of *in vitro* studies should be interpreted with caution, clinicians should be aware of temperature changes along implants and the potential risk associated with it.

Acknowledgements

Prof R Madsen (University of Missouri) for his assistance with the statistical analysis.

Derek Abrahams, for his assistance with building the model.

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Figure 1 Schematic representation of the study model.

Figure 2

Example of time temperature graph for channels 2 and 3. *max2* = highest temperature recorded on abutment (channel 2); *max3* = highest temperature recorded on implant (channel 3); *total_time47* = time period that temperature was $\geq 47^{\circ}C$ *(c-d); time2_47* = time interval between zero time and the first time 47ºC was measured by thermocouple 3 (*a-c*)*; hot2long* = this variable received value "1" if the threshold of $47^{\circ}C/\text{min}$ was reached, and "0" if it didn't; $a =$ introduction of warm water in upper chamber (*zero_time =* first time that a temperature $\geq 38^{\circ}$ C at abutment level was recorded); *b* = time when max2 was reached; c = time when 47° C was reached for the first time on implant for channel 3 (*start-time*); $d =$ time when 47^oC was registered for the last time on implant for channel 3 (*end-time*).

Figure 3

Scatter plot of max2 versus the difference of max2 and max3.

Figure 4

Correlation of the temperature of the abutment with the temperatures recorded at first implant level (channel 3). The blue dots represent test series that did not reach the threshold of 47ºC/1 min, the red dots represent series that reached the threshold.

Figure 5 Estimated ED50 and 95% confidence interval.

Table 1

Results for the 8 channels. The shortest *time2_47* is given only for the series that reached the 47° C/1min threshold. Channel 2 = abutment, channel 3 = most cervical position on implant, channels 4 to $8 = at$ 1mm increments more apically on implant. n.a. = not applicable.

