Temperature Changes Along a Dental Implant

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Purpose: The aims of this in vitro study were to analyze temperature changes along the surface of a dental implant and to establish the abutment temperature that could cause the critical 47°C/1 min threshold at the implant level. ***Materials and Methods:*** Eight thermocouples were attached at 1-mm intervals to an abutment/implant configuration. The model consisted of two compartments in a thermostatically controlled environment. The upper compartment represented the oral cavity with the abutment, which was exposed to 20 mL of hot water. The temperature at each thermocouple was logged over a period of 10 minutes. The Spearman 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" (ED50) for the abutment (95% confidence interval). ***Results:*** For 53 test series, the abutment temperature ranged from 52.80°C to 71.72°C. There was a positive correlation between the maximum temperature at the implant level and the temperature of the abutment. The 47°C/1 min threshold was reached 31 times at the most cervical implant level and decreased in frequency farther away from the heat source (14, 6, 3, 1, and 1 times, respectively). The ED50 was estimated at 62.3°C. This means that for an abutment temperature of 62.3°C, there was a 50% chance that 47°C would be reached at the implant level for 1 minute. ***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 risks associated with them. *Int J Prosthodont 2011;24:58–63.*

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 resulting from lack of osseointegration.[1] The threshold for irreversible enzymatic disturbance to cortical bone is reported to be 50°C for 30 seconds.[2] An in vivo animal study demonstrated that thermal bone injury occurs at a lower temperature: 47°C 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 42°C, and the critical temperature inducing cell death was 45°C to 48°C.[4] The temperature-time ratio of 47°C/1 min, as reported by Eriksson and Albrektsson,[3] is used routinely as a threshold in research studies.

Single-stage implant surgery developed from the traditional two-stage surgery in a search for less intervention and faster implant treatment. This results in the newly 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 the transmission of temperature from a heat source in the mouth to more apical levels along a dental implant. The majority of these studies are in vitro models. However, all of them confirm that heat is transmitted from the abutment to the implant, but not necessarily reaching the critical time-temperature threshold to cause thermal bone injury at the implant level.

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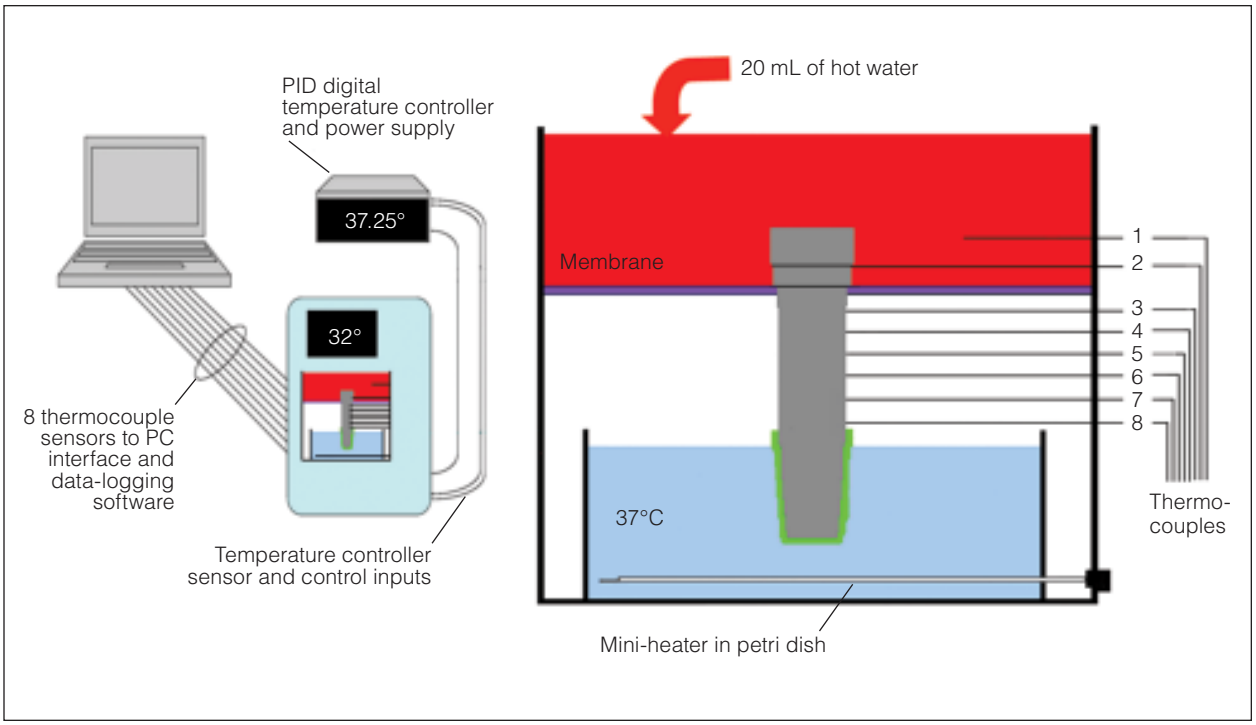

Fig 1 Schematic representation of the study model.

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

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°C[11] and even to 77°C in some instances.[12] Moore et al[13] recorded temperatures ranging from 5.6°C to 58.8°C at the maxillary incisor site and from 7.9°C to 54°C at the maxillary premolar site over a 24-hour period. They also reported that changes in oral temperature occured rapidly, while the return to baseline temperature occured more slowly. Feuerstein et al[9] reported a maximum intraoral temperature of 76.3°C for hot beverage consumption and 53.6°C for hot food.

The aims of this in vitro study were to analyze 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 47°C/1 min threshold at the implant level. The null hypotheses were that the surface temperature of an implant would not be affected by the temperature of its abutment and that the critical 47°C/1 min threshold would not be reached at any implant level, regardless of the abutment temperature.

Materials and Methods

A 3.75-mm implant with a 5-mm abutment (IBS15 and TB3N, Southern Implants) was mounted in an in vitro model consisting of two compartments separated by a teflon membrane attached to the neck of the implant (Fig 1). The upper compartment received 20 mL of hot water; the lower compartment was thermostatically

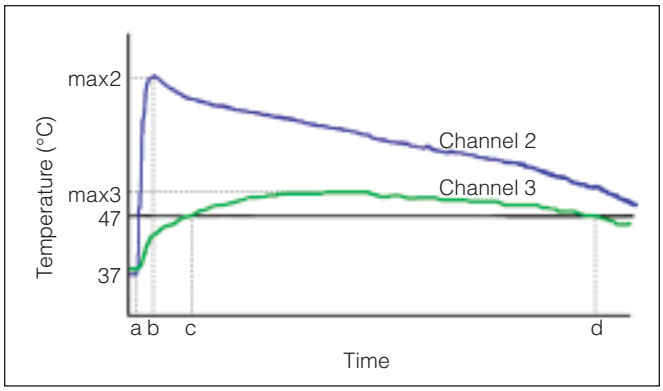

Fig 2 Example of a time-temperature graph for channels 2 and 3. max2 = highest temperature recorded on the abutment (channel 2); max3 = highest temperature recorded on the implant (channel 3); a = introduction of warm water in the upper chamber; b = time when max2 was reached; c = time when 47°C was reached for the first time on the implant for channel 3; d = time when 47°C was registered for the last time on the implant for channel 3.

Table 1 Results for the 8 Channels

	2	3	4	5	6	7	8
No. of tests	53	53	53	53	53	53	53
Lowest max temperature (all tests)	52.80°C	43.03°C	42.45°C	42.01°C	41.65°C	41.17°C	40.95°C
Highest max temperature (all tests)	71.72°C	53.00°C	51.75°C	50.83°C	49.80°C	49.46°C	49.00°C
Average max temperature (all tests)	63.30°C	47.39°C	46.25°C	45.43°C	44.78°C	44.08°C	43.59°C
Shortest time to 47°C (s)*	NA	63	89	115	147	160	180
No. of times threshold reached	NA	31	14	6	3	1	1

NA = not applicable.
*The shortest time to 47°C is given only for the channels that reached the 47°C/1 min threshold.

controlled to maintain the temperature of the implant at 37°C. Seven of eight K-type thermocouples were attached to the implant model by means of epoxy adhesive (Pratley Steel, Pratley) at the following sites: the implant-abutment junction above the teflon membrane (channel 2), the implant collar below the teflon membrane (channel 3), and the other five thermocouples were located subgingivally at 1-mm increments (channels 4 to 8). The apical portion of the implant was isolated with silicone putty and placed in a petri dish filled with water that was thermostatically controlled to maintain the temperature of the implant at 37°C. This was achieved by means of a mini-heater element regulated by a proportional-integral-derivative (PID) controller (Rex-C100, RKC Instrument). 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 32°C. 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), which was connected to a computer (Pentium 4, core 2 duo, 2 GB RAM, 1.8 GHz processor; Intel) via a USB cable. Data were captured using the dedicated software (PicoLog Recorder for Windows XP Professional version 5.13.9, Pico Technology). Temperature recordings were performed at least once every 3 seconds for 10 minutes for each test. The data were copied into SAS v 9 (SAS) for logistic regression analysis and MSExcel (Microsoft) for Spearman 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 at baseline for all channels was 36.9°C (± 0.7°C). The maximum abutment temperatures (max2) ranged from 52.80°C to 71.72°C (mean: 63.30°C, median: 63.57°C).

Table 1 represents the temperature range measured at each level, the shortest observed time to reach 47°C on each level for the tests that reached the threshold of 47°C/1 min, and the number of tests for each channel that reached the threshold of 47°C/1 min. The shortest time recorded to reach 47°C was 63 seconds, registered on channel 3 (thermocouple closest to the abutment), and the longest time to reach 47°C was 180 seconds, registered on channel 8 (most apically placed thermocouple).

Using the Spearman rank correlation test, the following positive correlations were found: between the maximum abutment temperature (max2) and

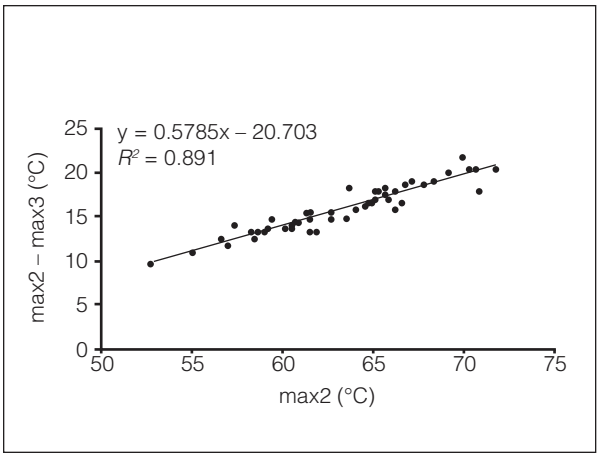

Fig 3 Scatter plot of max2 versus the difference between max2 and max3.

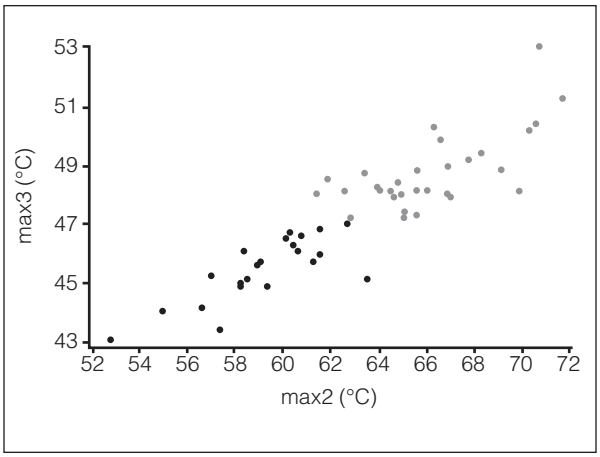

Fig 4 Correlation of the temperature of the abutment with the temperatures recorded at the first implant level (channel 3). Black = test series that did not reach the threshold of 47°C/1 min; gray = series that reached the threshold.

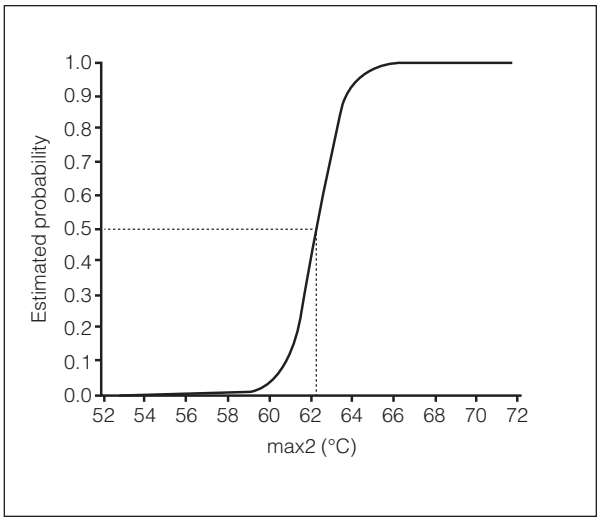

Fig 5 Estimated ED50 and 95% confidence interval.

maximum temperature at the first implant level (max3), between max2 and reaching the threshold of 47°C/1 min, between max3 and the last time 47°C was recorded by channel 3, between max3 and the duration that the temperature was ≥ 47°C, between max3 and reaching the threshold, and between the time spent at max3 and the difference between the time spent at max2 and max3 (Fig 3). The correlation between max2 and max3 associated with reaching the threshold is shown in Fig 4.

The following negative correlations were found: max2 and the time needed to reach 47°C at the implant level, max3 and the time needed to reach 47°C at the implant level, max3 and the time that 47°C was measured for the first time at the implant level, the time 47°C was measured for the first time at the implant level and the duration spent at 47°C, the last time 47°C was recorded and the duration of time it took to reach 47°C, and the duration spent at 47°C and the time it took to register 47°C.

Using logistic regression, the temperature of max2 at which there was a 50% chance that the temperature would exceed 47°C/1 min at the first implant level (ED50), with a 95% confidence interval, was estimated to be 62.3°C (Fig 5). There was an estimation problem of increasing magnitude the farther 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 analyzed the temperature changes along the surface of a dental implant following the application of warm water to the abutment. The first null hypothesis cannot be accepted because the surface temperature of an implant is indeed affected by the temperature of the abutment. The second null hypothesis can be partially rejected because the critical 47°C/1 min 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 farther away from the heat source.

The positive correlations may be explained as follows: *(1)* higher abutment temperatures cause higher temperatures at the implant level; *(2)* higher abutment temperatures cause a higher frequency in reaching the threshold of 47°C/1 min; *(3)* the higher the temperature at the implant level, the longer it takes to cool off below 47°C, the longer it remains at 47°C and the higher the chances to reach the 47°C/1 min threshold; and *(4)* the longer it takes to reach max3, the time interval between max2 and max3 increases. The negative correlations may be explained as follows: *(1)* higher abutment temperatures create a shorter time to reach 47°C at the implant level, *(2)* a higher implant temperature is associated with a shorter time to reach 47°C at the implant level, and *(3)* the faster 47°C is reached at the implant level, the longer the temperature remains at 47°C.

Table 1 illustrates that for more apically located channels, the number of occasions that the threshold of 47°C/1 min was reached decreased by a factor of approximately 0.5 for each 1-mm increment along the implant. Within the limitations of this study, it may be concluded that the cervical portion of the implant is the 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 the abutment and implant was larger than the time delay and loss of temperature among the different implant levels (channels). This was not statistically analyzed. 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 should be investigated further.

With the introduction of warm liquid in the upper compartment, the temperature of the abutment increases rapidly to reach the maximum and slowly returned to the baseline temperature (see Fig 2). The temperatures at the implant level also rose, but at a slower rate, and they never reached the same level as the abutment temperature. Figure 4 shows that for a higher abutment temperature (max2), the temperature recorded at the implant level was also higher and the chance to reach the threshold value of 47°C/1 min at the implant level increased. This is shown by the gray 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, the threshold value was reached a fewer number of times (Table 1).

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

With time, the temperature values of the abutment and implant tend to move toward the same value, suggesting a continuous exchange of energy among the abutment, implant, and environment. The immediate environment of the abutment consisted of the upper compartment receiving the 20 mL of hot water. Due to model constraints, the water could not be removed from the compartment, as would occur in vivo during swallowing. Instead, the water was allowed to cool in situ. Since the abutment temperature was always higher than the temperature at the implant level, this may have resulted in higher implant temperatures than if the water was removed from the upper compartment. On the other hand, only a single dose of hot 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 reported previously in a computer model,[6] this study showed that there was a sudden temperature peak at the abutment and implant levels at the time of exposure to the heat source. However, returning the model to the baseline temperature took much longer. This confirms the findings of an in vivo study by Moore et al.[13] Because of this phenomenon, it would be interesting to study the cumulative effects of consecutive short applications of warm water to resemble the consumption of a hot beverage.

The immediate environment of the implant consisted of thermostatically controlled air at the level of the thermocouples and silicon and thermostatically controlled water apical to the lowest thermocouple

(channel 8). This model differs from the intraoral situation where the implant is in close proximity to soft tissue and bone. In vivo heat transmission might differ compared to this in vitro model. This is a limitation of the current study and should be investigated further.

Conclusion

Within the limitations of this in vitro study, it can be concluded that abutment temperature is transmitted to an implant and that the threshold value of 47°C/1 min can be reached at the implant level. Although the results of in vitro studies should be interpreted with caution, clinicians should be aware of temperature changes along implants and the potential risks associated with them.

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Literature Abstract

Do elderly edentulous patients with a history of periodontitis harbor periodontal pathogens?

This study aimed to determine the prevalence of *Campylobacter rectus, Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Prevotella intermedia, Tannerella forsythia, Treponema denticola, Eikenella corrodens,* and *Parvimonas micra* in a specific elderly population with a history of periodontitis who have never worn dentures. Thirty dentate subjects (mean age: 61.7 ± 7.05 years) previously treated for periodontal disease and 30 edentulous subjects (mean age: 65.8 ± 8.05 years) with a history of periodontal disease were included in this cross-sectional study. Exclusion criteria included uncontrolled systemic diseases, immunocompromised condition, antibiotics within 6 months before the clinical and microbial examination, or the use of prosthetics. Microbiologic samples of cheek mucosa and the dorsum of the tongue were taken from all subjects. In addition, sulcus samples were taken from the dentate group. All samples were analyzed using a bacterial DNA-specific polymerase chain reaction. All pathogens studied were detected in dentate and edentulous subjects. When cheek and tongue samples were combined, *C rectus, A actinomycetemcomitans,* and *E corrodens* presented with a similar prevalence in both groups, whereas the other species were more prevalent, specifically in the dentate group ($P < .05$). In dentate subjects, *P intermedia and T denticola* were present in higher frequencies in the cheek mucosa (26.67% and 66.67%, respectively), whereas *P gingivalis and T forsythia* were more prevalent in the tongue samples (26.67% and 56.67%, respectively). While we previously believed that extraction removed all periodontal pathogens, the authors demonstrated that such pathogens may persist in the oral cavity of edentulous subjects who have had periodontal disease even 1 year after the extraction of all teeth, as well as in the absence of other hard surfaces in the mouth. The presence of these periodontal pathogens in the oral cavity may impact implant survival, possibly accounting for the higher complication rate in patients with a previous history of periodontal disease.

Fernandes CB, Aquino DR, Franco GCN, Cortelli SC, Costa FO, Cortelli JR. *Clin Oral Implants Res* 2010;21:618–623. **References:** 37.
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