In 2000 the intradiscal electrothermal therapy (IDET) procedure for the treatment of discogenic pain was introduced. The technique involves the positioning of an intradiscal catheter with a temperature-controlled thermal resistive heating coil at the inner posterior annulus. The therapeutic mechanism of IDET combines the thermo-coagulation of native nociceptors and in-grown nonmyelinated nerve fibers with collagen shrinkage, stabilizing annular fissures. Thermal nerve root injuries were described with IDET. The temperature in relation to the distance from the catheter tip was investigated. The intradiscal temperature distribution during treatment with IDET was also described.

Objective: To examine the temperature distribution outside the disc near neural structures and the risk of thermal damage to nerve tissue during a correctly performed IDET procedure.

Study Design: Experimental study.

Setting: Biomechanical laboratory of an academic orthopedic surgery department.

Methods: Testing was performed on cadaveric human lumbar spines with 10 intact intervertebral discs in a circulating water bath. Five thermocouples were attached to different locations on the disc. The temperature was recorded for 26 minutes. In addition, surface temperatures were recorded using an infrared camera. For the application of IDET, we used the Electrothermal 205 Spine System by Smith & Nephew and the standard clinical protocol.

Results: The shape of the recorded temperature curves was quite heterogeneous. Inside the spinal canal, temperatures as high as 45.2°C were recorded for a very short time. Temperature monitoring with the infrared camera demonstrated a change in temperature clearly restricted to the nucleus of the disc.

Limitations: The temperature distribution depends on the exact position of the IDET probe, which will never be 100% identical between individual experiments.

Conclusion: This study shows that temperatures generated within the spinal canal during IDET do not appear to be high enough to cause nerve damage.

Key words: IDET, thermal nerve damage, thermal complications, intradiscal electrothermal therapy
In 2000, Saal et al (1) introduced the intradiscal electrothermal therapy (IDET) procedure for the treatment of chronic discogenic low back pain. In the same year, they reported on their first mid-term results with IDET (2). The technique involves the placement of an intradiscal catheter with a temperature-controlled thermal resistive heating coil to a final position at the inner posterior annulus. The established standard heating protocol raises the catheter tip temperature from 65°C to 90°C over 12.5 minutes; the temperature is maintained at 90°C for 4 minutes. The authors aimed to create a temperature of 65°C inside the posterior annulus fibrosus. They proposed the mechanism of action of IDET to be a combination of thermocoagulation of native nociceptors and in-grown nonmyelinated nerve fibers plus collagen shrinkage, stabilizing annular fissures. The procedure has become popular very rapidly with more than 60,000 treatments having been performed until 2005 according to the equipment manufacturer (3). Several studies deal with the indication, the mode of action and the clinical efficacy of IDET (3-5). There are also studies which state that the effect is equivalent to placebo (3,6).

Heary et al described thermal nerve root injuries and similar observations were made by Cohen et al (7,8). However, very few authors have examined this issue experimentally. In the course of an experimental study, Bono et al (9) investigated the temperature as a function of distance from the catheter tip and found temperatures sufficient for collagen denaturation and nociceptor ablation at distances of up to 14 mm from the IDET catheter tip. Kleinstueck et al (10) described the intradiscal temperature distribution during treatment with IDET. In contrast, they showed that less than 2% of points measured inside the disc achieved temperatures sufficient for collagen denaturation. This resulted in an intense discussion about the method’s efficacy (10).

**Objectives**

The objective of our study was to determine whether there is a risk of thermal damage to nerve tissue during a correctly performed IDET procedure. We were interested in determining the temperature distribution at the nerve root level, inside the spinal canal and at the anterior border of the vertebral body in the region of the lumbosacral plexus during the IDET procedure. Our specific questions were whether the critical temperature for nerve injury could be reached during IDET and which anatomic structure absorbs most of the heat generated by IDET.

**Methods**

The testing was performed on fresh frozen cadaveric human lumbar spines. These were obtained from the Institutes of Legal Medicine at the Ludwig-Maximilians-University of Munich and at the University of Rostock. The study was approved by the Institutional Ethics Review Boards of both Institutes of Legal Medicine. In the analysis, 10 intact intervertebral discs of the segment L4/5 were included. All specimens included in the study were retrieved from recently deceased patients (age 29 – 66, mean age 55.2). Specimens with severe degeneration such as fissuring, bridging osteophytes, and other abnormalities were excluded from this study.

The spines were placed into a water bath with a volume of 18 liters, held at a constant temperature of 37°C ± 1°C. Constant heating of the water bath was achieved by using a heating plate; blood and cerebrospinal fluid (CSF) circulation were simulated using an approximately 300 rpm, continuously rotating magnetic stir bar placed into the water bath.

Five thermocouples were attached to specific locations on the vertebra (Fig. 1). Probe 1 was attached to the posterior longitudinal ligament (PLL) from inside the spinal canal at the dorsal border of the annulus. Probe 2 was positioned at the same location, but 2 mm farther inside the spinal canal. Probe 3 was attached on the right side and Probe 4 on the left side of the annulus level with the neural foramina. Probe 5 was fixed...
to the anterior midline of the disc at the anterior longitudinal ligament (ALL). Placement of the probes was controlled by fluoroscopy.

We used the Testo 454 digital thermometer (Testo AG, Lenzkirch, Germany). The connected thermocouples were type K, class 1 with a sensor length of 400 mm and a diameter of 0.25 mm. The temperature was registered with an accuracy of .01°C and a frequency of 0.5 Hz over 800 single measurements per thermocouple. Each test lasted 26 minutes.

In addition, a color-coded infrared camera (Varioscan, Jenotik, Jena, Germany) recorded the temperature changes on the surface of the lumbar specimen in 2-minute intervals. This, however, is not possible when carried out in a water bath. Therefore an additional measurement outside the water bath was performed. For this part of the study, we were not interested in the absolute peak temperature, but in the temperature distribution within the tissue.

For the application of the IDET, we used the Electrothermal 205 Spine System (Smith & Nephew, Marl, Germany). The catheters were placed using the standard introducer needle and by puncturing the disc to be treated via the “safe triangle” at the anterior border of the ascending facet. Catheters were then advanced through the introducer needles under biplanar fluoroscopy control to ascertain that the catheter tips were positioned exactly at the posterior annulus according to the standard treatment protocol. The introducer needles were then carefully withdrawn, the tip positions checked again, and the catheters fixed and connected to the IDET generator. While accelerated heating protocols have been employed, we decided to investigate the established standard protocol only. According to this standard clinical protocol, heating starts at 65°C, increasing the temperature every 30 seconds by another 1°C up to a final peak of 90°C. This temperature is then maintained at a constant level for 4 minutes. The total treatment time is 16.5 minutes.

Results and statistical analysis of the temperature distribution data are shown in a nonparametric presentation of the 50th percentile and the median at each time point. Measurements were performed continuously, but for better presentation, statistical analysis was performed on the values at every 30 seconds, showing a representative diagram (Figs. 2-6).

Fig. 2. The curve shows the temperature profile during IDET on the posterior border of the intervertebral disc inside the spinal canal. Temperature patterns are very smooth and heterogeneous. Peak temperatures are above 45°C for only 2 minutes and in only one curve. The figure shows the nonparametric distribution of temperature with the 50th percentile and the median at each time point.
Fig. 3. Here the temperature profile during IDET 2 mm distant from the posterior border inside the spinal canal is shown. Temperature distribution is similar to Fig. 2, but the median shows lower temperature values.

Fig. 4. The curve demonstrates the temperature profile during IDET at the anterior border of the spinal body. Maximum temperature does not exceed 40°C and is therefore not potentially harmful.
Results

During the majority of measurements, a clear rise in temperature was recorded within approximately 15 minutes of treatment time. The most distinct rise was recorded at the posterior border of the annulus and at the posterior annulus (Figs. 2 and 3). The shape of the recorded temperature curves was quite heterogeneous, depending on the position of the probe (Figs. 2-6). Inside the spinal canal, temperatures up to 45.2°C over...
a period of 2 minutes were recorded. Three measurements showed hardly any difference. During the experiments in which the probes at the dorsal part of the disc (i.e., inside the spinal canal) measured the highest temperatures, the probes placed on the anterior border of the vertebral body measured the smallest changes in temperature. In contrast, measurement cycles recording the highest temperatures on the ventral border showed only slight increases in temperature on the posterior border of the vertebral body.

Monitoring of surface temperature development using the infrared camera demonstrated a change in temperature that was strongly restricted to the nucleus of the disc (Fig. 7). At the center of the vertebral disc a homogeneous rise in temperature occurred. There was a notable decrease in temperature at the annulus fibrosus.

**Discussion**

The heat sensitivity of different tissues depends on the type of tissue, the absolute temperature, and the duration of application. The Arrhenius equation describes this coherence and indicates that the velocity of a chemical reaction as well as the speed of cell destruction increases with rising temperature.

Graphically, the logarithmic rate of inactivation is represented as a function of the inverse absolute temperature. This biphasic curve applies equally to all mammals. The curve progression is represented by the following equation (according to Dewey) (5): $t_2 = t_1 \times R(T_1 - T_2)$. Heat damage of biological tissues is expected to occur below the threshold of 43°C. An increase of 1°C and an application duration to a factor of 6 would be equivalent to an increase of 1°C at a temperature above the threshold of 43°C and an application duration to a factor of 2 (11). This means that between 42°C and 45°C, a temperature rise of 1°C would be sufficient to reduce the tolerated application duration to 50% (12). Sminia (13) observed neurological deficits and death after thermally exposing the brains and spinal cords of rats to temperatures of up to 42.9°C ± 0.4°C for more than 38 minutes. Franken et al (14) showed in rats that an exposure to temperatures above 43°C for one hour leads to motor dysfunction. The higher the chosen temperature, the higher is the potential for damage. Uchiyama (15) showed alterations in the nerve amplitudes in dogs whose spinal cords were exposed to 45°C for over 30 minutes. In a study conducted by Konno (16), the neural damage on 20 pigs was examined after local hyperthermia was applied. The application of 40°C for over 30 minutes resulted in no damage, whereas an application of 60°C for 5 minutes showed histological neural damage and an application of 70°C temperature for 5 minutes resulted in a failure of neural function (16). Fajardo (17) defined the thermal damage threshold on the central nervous system as being 42°C for over 60 minutes. The curve progression is represented by the following equation (according to Dewey) (5): $t_2 = t_1 \times R(T_1 - T_2)$. Heat damage of biological tissues is expected to occur below the threshold of 43°C. An increase of 1°C and an application duration to a factor of 6 would be equivalent to an increase of 1°C at a temperature above the threshold of 43°C and an application duration to a factor of 2 (11). This means that between 42°C and 45°C, a temperature rise of 1°C would be sufficient to reduce the tolerated application duration to 50% (12). Sminia (13) observed neurological deficits and death after thermally exposing the brains and spinal cords of rats to temperatures of up to 42.9°C ± 0.4°C for more than 38 minutes. Franken et al (14) showed in rats that an exposure to temperatures above 43°C for one hour leads to motor dysfunction. The higher the chosen temperature, the higher is the potential for damage. Uchiyama (15) showed alterations in the nerve amplitudes in dogs whose spinal cords were exposed to 45°C for over 30 minutes. In a study conducted by Konno (16), the neural damage on 20 pigs was examined after local hyperthermia was applied. The application of 40°C for over 30 minutes resulted in no damage, whereas an application of 60°C for 5 minutes showed histological neural damage and an application of 70°C temperature for 5 minutes resulted in a failure of neural function (16). Fajardo (17) defined the thermal damage threshold on the central nervous system as being 42°C for over 60

**Fig. 7.** This figure shows an illustration of the temperature profile recorded by the infrared camera every 2 minutes (a-h). After 2 minutes (a) heating of the intervertebral disc starts. After 4 minutes, heat generation can be observed inside the center of the disc (b). After 8-10 minutes, the nucleus of the spinal disc is completely heated (c-f), while 14-16 minutes past the beginning, the peak of the heating is being reached (g-h). The temperature rise is being sharply limited by the annulus fibrosus.
minutes (17). The difference in the thermal conductivity between dry and moist bone as described by Leeson (18) was considered in our experimental setup.

In our experiment, we used the typical catheter placement and the standard clinical protocol for IDET. Differences in temperature distribution merely derive from anatomic variations of each intervertebral disc and the positioning of the IDET catheter. The procedure has to tolerate anatomic variations in order to provide a universally valid application. The exact positioning of the catheter depends to some extent on the surgeon’s judgment, since it is only controlled by fluoroscopy, which has inherent imprecisions and which is subject to interpretation.

The highest temperatures were registered directly behind the posterior border of the intervertebral disc. Since the maximum value of 45.2°C was recorded only about 2 minutes, it can be assumed that – under the premise of correct application – there should not be any heat damage to the spinal cord and the spinal nerves. While Cosman (19) demonstrated that temperatures in this range can lead to tissue damage, the tissue at risk with a correctly performed IDET would potentially be epidural tissues and possibly dura, whereas the cauda equina is protected by CSF flow (19). Kleinstueck et al (10) examined whether IDET produces adequate tissue temperatures to denature annulus collagen or to ablate nerve cells. Their experimental setup was similar to ours. IDET was performed on lumbar spine specimens placed into a 37°C water bath. But a circulation within the water bath simulating blood and CSF flow was not used. This resulted in the recording of temperatures above 50°C. In our experiments, such high temperatures were not measured.

The model of Bono et al (9) is even more similar to our study. In the course of this study, thermocouples were positioned along the annulus fibrosus. Results showed temperatures between 60°C and 65°C at a distance of 2 mm from the catheter. Minimum temperatures of 45°C were recorded at all reading points. Bono et al (9) state that temperatures sufficient for collagen denaturation and nociceptive abolition could be achieved in any intervertebral disc. They recorded slightly higher temperatures at the anterior annulus compared to our readings, ranging from 38°C to 45°C. They did not use a circulating water bath, which would have lowered the actual temperature through convection.

To investigate whether annulus defects can be sealed by means of collagen shrinkage using IDET, Freeman (20) designed an animal study. He positioned thermocouples inside the nucleus and in the posterior annulus, each 2 mm away from the heating catheter. Inside the posterior annulus, an average temperature of 63.6°C and a maximum of 77.1°C were recorded. It must be noted that in this setting the heating coil had been placed into the annulus, unlike the common placement according to the standard clinical IDET protocol.

None of the authors mentioned above discussed whether the recorded temperatures could cause thermal damage in addition to the desired therapeutic effect. Interpreting other authors’ studies along with our own results, we conclude that a temperature of approx. 45°C is reached inside the spinal canal, which can, over a prolonged period of time, cause damage to neurons. The application of this temperature during IDET, assuming correct placement of the heating catheter, appears to be unlikely to cause thermal damage to the nerve structures due to its short application period.

Using a color-coded infrared camera for the first time in the literature to illustrate the temperature distribution during IDET, we could demonstrate the considerable decrease of temperature in the annulus fibrosus. This is best explained by the different anatomic structure of the annulus fibrosus and the nucleus pulposus. The infrared camera depicts surface temperatures. It is not suitable for absolute temperature determination inside a water bath. Hence the anatomic preparation had to be taken out of the circulating water bath and therefore lacks the simulation of a natural environment. For methodological reasons, the temperatures recorded by the infrared camera are shown as relative temperature changes depicted as color changes; they cannot be compared to the absolute temperature values recorded by the probes used in our water bath model. As with any experimental study, there are some limitations to our experimental setup. While the water bath used is capable of maintaining a steady baseline temperature and of providing a certain degree of fluid circulation by using a mechanical stir bar, it cannot realistically simulate the flow of blood and CSF in a living tissue. Neither are the biochemical and physiological conditions identical to living tissue. Nevertheless, it is an established model and in our view adequate for the physical experiments that we performed. A further limitation is the fact that we could not generate exactly identical catheter positions between the different discs used in the experiments. However, this is an inherent feature of the IDET technique and therefore catheter positions are also always a variable in clinical application.
In the present study, we did not perform a pre-study assessment of the individual degrees of annulus degeneration for the experimental discs. Since we found that the annulus represents a relevant heat barrier toward outside structures, further studies will try to correlate this barrier function to the degree of annulus degeneration as assessed by pre-study imaging.

**Conclusion**

In summary, we were able to show that the annulus acts as a protective thermal barrier towards the posteriorly located neural structures as well as towards ventral structures such as vessels, nerve plexus, and the intestine. The temperatures that we recorded at the periphery of the annulus never exceeded 45.2 degrees and hence make a thermal injury as a consequence of IDET very unlikely.

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**References**