Does Local Heating Really Help Diabetic Patients

Increase Circulation

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Abstract

Nine control and 8 diabetic subjects participated in a series of experiments to determine what effect hot packs vs. water immersion had on local tissue heating and if local heating produced significant changes in the circulation of diabetic patients. The results of the experiments showed that, to a large extent, hot packs were not effective in heating deep tissues when compared to water bath immersion. Further, for the diabetic patients, even when heated in a water bath, their limb and skin blood flows were not altered to any large extent by heating. The implication is that much longer exposures to heat need to be used in patients with diabetes to have a good clinical outcome.

Key words; circulation, diabetes, hot packs, hydrotherapy
Introduction

Warming or cooling tissue is one of the most common therapeutic modalities used in physical therapy (Petrofsky 1992, Petrofsky and Lind 1981, Hayes 1993, Hecox et al, 1994, Michlovitz 1996). Cooling is usually achieved by cold packs or water immersion while heating can be accomplished by either of these two modalities, diathermy, or ultrasound (Hayes 1993, Hecox et al, 1994, Michlovitz 1996). In studies where heat has been applied, the increases in temperature have been reported to be effective in increasing tendon extensibility (Lehmann, el., al, 1970) and reducing joint stiffness (O’Dell 1975). Increasing skin temperature also increases local blood flow to tissue (Greenberg 1972) and thereby aids the healing process (Abramson et al 1966) by, for example, reducing muscle soreness (Weber et al 1994). Likewise, the use of cold packs has been reported to be effective in reducing muscle spasticity in patients with MS and reducing swelling in tissue after acute injuries. Whereas heating the forearm increases blood flow, cooling is associated with a decrease in blood flow to the forearm (Karunakara et al 1999).

Numerous studies have shown the advantages and disadvantages of one therapeutic modality versus another. For example, Borrell (Borrell el. al. 1980) showed that fluid therapy was more effective than superficial heating to increase the temperature of the skin and joint capsules. He also found it to be better than hot Paraffin wax treatments or hydrotherapy. Some studies have reported better results when using a combination of hot packs and ultrasound (Abramson et al 1967, Lehmann el., al, 1978). Body fat can be a confounding factor insulating deep tissue from temperature changes (Petrofsky and Lind 1975).
But the assumption in all cases are 1) hot packs effectively change tissue temperature and 2) changing skin and tissue temperature has a positive therapeutic effect, i.e. increases circulation to heal tissue. One population of particular concern are diabetic patients who undergo physical therapy for orthopedic injuries and receive hot packs or whirlpool treatments to increase circulation. The concern arises since a common complication found in both Type I and Type II diabetes is damage to the autonomic nervous system which can result in circulation defects. (Accurso et al 2001)

Cardiovascular and autonomic impairment in patients with Type I and Type 2 diabetes have been reported previously (Martine 1953, Ewing and Clarke 1986, Fealey et al 1989). Of particular interest is the relation of autonomic impairment and its effect on the skin. The damage to the skin associated with autonomic dysfunction first results in increased skin circulation. Skin, subcutaneous and muscle blood flows are all elevated in uncontrolled diabetics (Gunderson 1974, Haggendal et al 1970, Christianson 1970, Mathiesen et al 1985). As the sympathectomy proceeds, blood flow first increases to the skin and may be 5 times higher than the normal flows (Archer et al 1984). It is believed that a loss of sympathetic control increases AV shunt activity, increasing overall skin flow but reducing nutritive flow to the skin, thereby causing susceptibility to diabetic ulcers (Rendell and Bamisedum 1992, Edmonds et al 1982). Over time, skin and muscle lose their ability to vasodilate and blood flow is dramatically impaired. (Petrofsky et al 2003). If the limb no longer vasodilates in response to heating, then hot pack therapy may not be effective in this population.

Therefore, the purpose of the present investigation was to determine the effect of hot packs vs. water bath immersion on deep tissue heating. Once the range of
temperatures in the skin and deep tissues were determined during therapeutic treatment, the skin and limb blood flows in diabetic patients were assessed to see if there was a therapeutic increase in circulation for patients with diabetes during heating. There were 3 series of experiments. In the first 2, the relationship between deep tissue temperature and bath and hot pack insulation were developed. In the third series, local heating of the skin, heating of the arm and finally whole body heating was used to assess the reactivity of the circulation to heat in diabetic subjects.
Subjects

Three series of experiments were conducted. The first series involved four male subjects whose ages, heights and weights are listed in table 1. (Control 1) All subjects had body fats between 10 and 20 percent. A group of diabetic and controls (control 2) were involved in cardiovascular studies as described below. All experimental procedures were explained to each subject who then signed a statement of informed consent. The committee and human experimentation approved all protocols and procedures. Three of the subjects with diabetes had impaired sensation in their feet upon examination while 5 were normal. There was no report of any strength deficit in any muscle group in these subjects. Four of the 8 diabetic subjects were engaged in aerobic training such as walking of running on a daily basis. The other subjects were active.

Methods

Measurement of Muscle Temperature

Muscle temperature was measured by a thermocouple probe inserted two centimeters deep below the level of the skin above the appropriate muscle group as described below. A twenty-gauge needle was first inserted into the muscle. A thermocouple was then placed down the shaft and the twenty-gauge needle was withdrawn leaving the thermocouple buried in the muscle. Collodion was then applied around the thermocouple to assure a waterproof junction at the insertion point through the skin. The thermocouple output was then displayed by a digital thermometer where the temperature was read to plus or minus 0.1 degree C.
**Hot Pack Application**

Hot packs were standard Chattanooga hot packs of dimension of 10 by 25 cm. They were kept at 160 degrees Fahrenheit and were applied above the appropriate muscle with various layers of towels separating the hot pack and skin as described below under procedures.

**Blood Flow**

*Skin blood flow* was measured by a laser Doppler flow meter produced by Moor Instruments, Inc. The device sat on a stand approximately 0.66 meter from the patient and, while the patient was laying supine, the device scanned the body and produced a picture of the body in terms of the blood flow to the skin. This device was completely non-invasive and had no physical contact with the body and posed no danger.

*Arm blood flow*- Limb blood flow was calculated by Whitney volume plethysmography. This technique uses a bracelet that contains a strain gauge to measure limb volume. A venous occlusion cuff (40 mmHg) is inflated for 5 sec and deflated for 7 sec on the upper arm. Changes in limb volume during the first 2 sec after inflation are proportional to limb blood flow and are recorded to assess the circulation through the entire arm. The wrist is removed from the circulation by an occlusion cuff inflated to 250 mmHg. This technique has been described previously (Whitney 1952, Lind and McNicol 1967, Petrofsky et al 1981).

*Pulsatle skin Blood Flow*- Continuous measurements of pulse pressure in the skin was measured optically with a Hertzman photoelectric plethysmograph. The plethysmograph
consisted of a small plastic ring that contains a light source and photocell. The light
source focuses low intensity red light into the skin and the photo cell records the beam
that is instantly reflected back from the skin. By amplifying this signal electronically
from the photocell, a Pulsatle output is recorded which is proportional to the flow pulse in
the skin under the light. This technique has been described previously (Petrofsky, 1979)

Procedures
Three series of experiments were conducted as described below. The purpose of
the first series was to examine the deep muscle temperature after the application of hot
packs or after immersion in a water bath. The second series examined the change in flow
in the skin after local application of heat and the third series examined the effect of limb
and whole body heating on blood flow.

Series I – In the first series of experiments, four subjects participated. In different
experiments, the lower body was placed in a water bath or hot packs were applied above
the calf. The thermocouple probe was applied in the belly of the gastrocnemius muscle.
The experiment was repeated for each experimental condition. The period of exposure to
hot packs or the water bath was 20 min since this is the length of time most texts
recommend that hot or cold packs be applied (e.g. Hayes 1993, Hecox et al, 1994,
Michlovivitz 1996). The muscle temperature was measured throughout the twenty-
minute period. Towels separated hot packs from the skin with a thickness of 0.5, 1, 2, 3,
or 4 millimeters in thickness from the skin on different occasions since this represents the

Series 2- In this series of experiments, the reactivity of the arm vascular bed was assessed. Muscle temperature varies in the forearm between 27 and 42 deg C. This is due to the fact that the forearm is a shell tissue and temperature varies to help gain or lose heat from the core of the body (Petrofsky et al 1981). Limb tissue temperature varies with room temperature, clothing, body fat content, and the phase of the menstrual cycle (Petrofsky and Lind 1975). Therefore, a thin person may have resting arm metabolism less than 20% that of a person with a high body fat content due to the high $Q_{10}$ of the tissues (Wang and Kawai 2001). Therefore, to remove some of the variability in previous studies, a water bath was used to elevate all forearm temperatures to that of the core, 37 deg C. Subjects placed their arms in a bath with the arm held dependant and the elbow at an angle of 90 degrees such that their arms were submerged to the belly of the biceps muscle. The bath was well stirred. After 15 minutes, resting arm flows were recorded. A 4 min period of arterial occlusion induced by a cuff under the axilla and inflated to 200 mmHg was then used. After this period, flows were measured for 3 min.

Series 3- Skin and whole body heating

The Subjects were led into an environmental room where room temperature was initially at 22 deg C. The subject was placed in the supine position on a table. A thermode whose temperature was 38 deg C was applied above the medial gastrocnemius muscle on the ventral border of the muscle and skin blood flow was measured by a laser
Doppler flow meter while the skin was warmed in 5 control and 8 subjects with diabetes to test skin reactivity to local heating. Next, as the subjects lied comfortably for another ten-minute rest period, pulsatile flow to the foot and skin blood flow over the same area of the gastrocnemius were measured. Skin temperature was measured just distal to the knee. A mark was placed at this location so that Doppler flows would be measured at the same spot. The room temperature was then elevated to 42 deg C. A light blanket was placed over the subject. After 30 minutes, all measurements were repeated.
Results

The results from these experiments are shown in figures 1 through 6. Figure 1 shows the results of the first series of experiments during which subjects immersed their arms in water baths at temperatures of 42°C, 37°C, and 34°C for periods of 20 minutes. Figure 1 shows the average temperature measurements measured in the middle of the medial gastrocnemius muscle for the four subjects. As can be seen in this figure, the average resting muscle temperature, although temperature was measured approximately 2 cm below the level of the skin, averaged just over 30°C. From this initial resting muscle temperature with water bath immersion of the limb, temperatures rapidly increased to final values of 42°C, 37°C, and 34°C, that is the bath temperature at the end of the 20 minute period. Thus immersion in the water bath for a period of 20 minutes warmed the tissue up to the target bath temperature. For all subjects involved in these studies approximately half of the warming took place in the first eight minutes. At the eight minute point, for example, the tissue was only 39°C in the 42 degree bath. The actual tissue temperature was not raised to 42°C until the 16th minute in this warm bath. However, in general, by the end of 20 minutes, the tissue was warmed to the full temperature of the bath as stated above. In contrast, the results were not quite as good when hot packs were used to increase tissue temperature as shown in figure 2.

Authoritative texts recommend using towel thicknesses ranging from a ½ to 4 cm to protect the skin from the hot packs to prevent burns (Hayes 1993, Hecox et al, 1994, Michlovivitz 1996). Therefore, in this series of experiments towel thicknesses separating hot packs from the skin were at thicknesses of 0.5, 1, 2, 3, and 4 cm. With a towel separation distance of 0.5 cm, muscle temperature increased fairly quickly over the first 8
minutes to an average of 37±2.1°C for these 4 subjects from an initial resting temperature of 29±1.4°C. However, by the time 20 minutes had elapsed peak muscle temperature averaged 43±1.7°C for these 4 subjects. On the opposite extreme, for the 4 cm towel thickness, although the resting muscle temperature was 30±1.8°C, after 8 minutes, muscle temperature had only raised to 32±1.9°C and even after 20 minutes muscle temperature was simply that of the core 37±2.1°C. In general then, the thinner the towel layer the warmer the muscle got. However, comparing these data from figure 2 to 1, much quicker and better heating was accomplished in a water bath that with hot packs.

The results of the second series of experiments are shown in figures 3 through 6. Figure 3 shows the results of the vascular occlusion studies. In these studies, diabetic subjects had their arms immersed in a water bath since this provided the most consistent method of heating the whole limb in the first series of experiments. Once the entire limb was heated, resting flows were measured. Following the measurement of resting flows, a 4 minute occlusion period was applied to the limb and the first post exercise flow was measured to assess vascular reactivity in relation to warming the tissue to 37°C, a temperature consistent with water bath immersion and 20 minutes of exposure to hot packs with thin towel layers. This reactivity would be equivalent to the flow increase with light exercise.

As can be seen in figure 3, blood flow in the control subjects after exposure to 15 minutes in a 38°C water bath averaged 0.99±.22 cc blood flow per 100g tissue/min. In contrast, blood flow in the diabetic subjects averaged 0.49±0.13 cc blood flow per 100g tissue/min. This difference between the flows, that is the higher flows seen in the control subjects due to the effect of temperature, was significant when compared to the diabetic
subjects (p<0.01). There was a remarkable consistency of results in the eight diabetic subjects. Although 4 of the subjects suffered from Type I diabetes and 4 from Type II diabetes, only one patient had flows higher than 0.6cc per 100g tissue/min, even after heating in the hot water bath with the whole limb. This patient was unique in that the patient was just diagnosed, within the last 3 months, to have Type II diabetes. The other 6 subjects had average flows of 0.42±.11 cc per 100g tissue/min. In other words, most subjects had flows less than the mean value shown in the figure but the data was biased high by one patient who was on the high side of the group due to the newly diagnosed diabetes. After the period of occlusion, flows of the control subjects increased significantly higher than that of the diabetic subjects as shown in this figure (p<0.01). Flows in the diabetic subjects only increased to 1.59±0.71 cc per 100g tissue/min, whereas in the control subjects it increased to 7.47±1.15 cc per 100g tissue/min. Most subjects showed an increase in flows in the diabetic group irrespective of whether they had Type I or Type II diabetes, though only approximately 1.1 or 1.2 cc per 100g tissue/min. There were two noteworthy exceptions, one patient who was on the high side of the group, having his flows increase to 1.7 cc per 100g tissue/min. and then the patient who was newly diagnosed as having Type II diabetes increased his flows to 3.1 cc per 100g tissue/min.

Likewise, as shown in figure 4, rather than using a water bath, when direct heat was applied to the skin at a temperature of 38ºC through a small thermode, blood flow increased much more in the control group than the diabetic group. Starting from an average flux (laser Doppler flows) of 99±48.33 in the control group, flow increased over a 2 minute period to 131±53.6 flux at approximately 1 minute and finally to a final value
of 149±61.11 flux at the end of the 2 minute period during which mild heat was applied through the thermode directly to the skin. This increase in flow in controls by just heating the skin a few degrees centigrade amounted to about a 50% increase in skin flow. In contrast, for the diabetic group, resting flows were less, averaging 75±30.18 flux units and at the end of the 2 minute period, flow increased to only 91±35.72 flux. Thus the blood flows of the diabetic group with local skin heating only increased by 21%.

When the subjects were heated in a 42°C room for 30 minutes, blood flow increased to the skin. For the control subjects, flows increased from 123+/- 26 flux units to 220+/- 49.3 flux units as shown in this figure. In contrast flows for the diabetes group increased from only 84+/- 23.4 to 119+/- 34.2 units. This is particularly surprising considering the changes in central body temperature that occurred during this period in time. Core temperature in the control subjects increased from an average of 36.8±.163°C to 36.6±.95°C after 30 minutes exposure. In contrast whereas the core temperatures of the diabetic subjects averaged 36.5±.15°C in the cool room, after heating temperature increased approximately 1°C to 37.5±1.11°C. Temperature increased almost three fold higher during the heat exposure in the diabetic subjects than the control subjects. But in spite of that, flows just barely increased to the skin.

Finally, figure 6 summarizes the data on blood flow to the foot during the same heat exposure. Foot flow shown here is in relative flows, that is percentage of blood flows at rest. Flows increased in the warm environment, after the 30 minute exposure, to 1.44±.38 in the diabetic subjects, but 2.7±.31 in the control subjects. Thus flows barely increased at all during heat exposure in the diabetic subjects in spite of the fact that core temperature sustained a large increase. Even more interesting is that when skin
temperature was measured on the leg, another relationship was established as shown in figure 6. Figure 6 shows the relative flows in relation to the skin temperature. As can be seen in this figure, skin temperature sustained a much larger increase to as high as 34.5±1.3°C in the diabetic subjects after 30 minutes of heat exposure. Skin temperature never even warmed to 34 degrees in the control subjects. As skin temperature increased in the control subjects, flows to the foot increased such that at the end of the 30 minute exposure, flows were almost triple that of the subjects in the cool room for the control subjects. In contrast, skin temperature rose continuously as did central body temperature in the diabetic subjects and yet flows seem to plateau and could not increase in spite of and increase in skin temperature as shown in figure 6.
Discussion

It is a common clinical practice to use hot packs to heat tissue after the initial acute phase of orthopedic injuries (Abramson et al 1966,67, Lehmann et al 1974, Weber et al 1994). The assumption is that by heating the tissue, circulation will increase thereby promoting healing in the target tissue (Weber et al 1994). Studies presented here provide two different aspects of a problem on heating that may be of interest to physicians and physical therapists concerning heating with hot packs and water baths.

First, heating of the tissue with hot packs in either control or subjects with diabetes may not be terribly effective. If multiple towel layers were used, then heating is very minimal and very slow. A standard therapy session is a 15 min modality. The tissue may just begin to warm at the 15 min end of the session as per data collected here. Further, in the present investigation, when 4 cm of towels were used, heating was very minimal to the deep tissue. In contrast if only a ½ cm of towels were used, heating was good but there is always a chance of burning the skin, especially in subjects with diminished sensation. Diabetic subjects have been reported to have diminished sensation and therefore hot packs may not be an effective modality for these patients.

Finally, a confounding variable which pertains to many type 2 diabetics is body fat. Individuals who have high body fat content have a very high insulative capacity and therefore hot or cold pack treatment is even less effective (Petrofsky and Lind 1975, 1980). Thus the use of hot packs may always be in question in terms of how much hot packs really heat the tissues.

Autonomic impairments in diabetic patients have been reported previously and most likely account for the circulation defects which have been observed. (Martine 1953,
Ewing and Clarke 1986, Fealey et al 1989). A number of mechanisms have been suggested for autonomic and peripheral nerve damage in diabetes. These include 1) enhanced flux through polyol pathways with increased aldose reductase activity resulting in glycemic product accumulation (principally sorbitol) and a reduction in Na K Atpase activity (Tomlinson et al 1985, 1998) 2) reduction of nerve blood flow causing damage to the nerves (Low et al 1993, Schmidt 1993) 3) formation of non enzymatic glycosylation end products (Yagihashi et al 1992) 4) deprivation of nerve growth factor (Cameron and Cotter 1993) and 5) immunological processes (Ziegler 1994).

Whatever the mechanism, the end result is that when either skin is warmed or deep tissue is warmed, as in the present investigation, flow in the muscle and skin is markedly attenuated. Therefore, the present investigation strongly suggests that the damage is not simply limited to the skin as some investigators have previously alluded to but also to muscle and other deep tissues. Allegedly the damage could also occur in tendons, joints and other deep tissues which normally have a very low blood flow to begin with. The present investigation shows that the vascular response to heating in both skin and deep tissue with diabetic patients is roughly 20% that of control subjects. Even in a newly diagnosed diabetic patient, blood flows were greatly reduced in both skin and deep tissue. Furthermore, even whole body heating which usually elicits a very strong sympathetic response by dilating skin blood vessels and muscle blood vessels had little effect on the skin and deep tissue flows in diabetic patients.

Therefore we recommend, based on this data, that hot packs not be used in diabetic patients because to achieve good tissue warmth, thin towel layers must be used. With thin towel layers in patients with parasthesia of the skin, the skin may burn. In
contrast, this study suggests that water immersion, such as a water bath, Jacuzzi or whirlpool which appear to heat the deep tissues quickly and thoroughly may be preferable to hot packs for tissue heating after an orthopedic injury. While water baths or Jacuzzis should be considered, we suggest that these patients should be left in the baths for much longer periods of time because of the low blood flow response seen in diabetic patients to local heating. Further investigation with comparison clinical outcome data is warranted.


Martine, M (1953) Involvement of autonomic nerve fibers in diabetes. Lancet 1;560-565


Figure 1  This figure illustrates the muscle temperature in the gastrocnemius muscle during a 20 min immersion in water at a temperature of 42(), 37 () and 34 () degrees C. Each point is the mean of 4 subjects on each of 2 occasions.

Figure 2- The temperature of the medial gastrocnemius muscle after up to a 20 min application of hot packs under towels at 0.5(), 1(), 2(), 3(x) and 4 (*) cm thickness. Each point is the mean of 4 subjects on each of 2 occasions.

Figure 3- The effect of warming and vascular occlusion on whole forearm blood flows in 8 patients with diabetes and 5 control subjects. Each point is the mean of all subjects.

Figure 4- The average skin blood flow in flux determined by a laser Doppler flow meter under a thermode in control and diabetic subjects. Type 1 and Type 2 diabetic subjects have been grouped together as one large group. The thermode was left on for 4 0.5 min flow periods.

Figure 5- The effect of whole body heating on skin blood flows above the medial gastrocnemius muscle. Each point is the mean of 8 diabetic and 5 control subjects before and after heating.
Figure 6- The relative flows in the foot (relative to flows in a cool room) in relation to skin temperature for 8 diabetic and 5 controls subjects. Each point is the mean for the group.
Table 1  General Characteristics of Subjects

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Figure 1
Figure 2
Figure 3
Figure 4-
Figure 5
Skin Temperature and Foot Flows

Relative flows % (x100)

Skin Temp Deg C

- diabetic
- control

Figure 6