The Relationship Between Intramuscular Temperature, Skin Temperature, and Adipose Thickness During Cryotherapy and Rewarming

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ABSTRACT. Jutte LS, Merrick MA, Ingersoll CD, Edwards JE. The relationship between intramuscular temperature, skin temperature, and adipose thickness during cryotherapy and rewarming. Arch Phys Med Rehabil 2001;82:845-50.

Objective: To describe the relationships among muscle temperature, skin temperature, room temperature, body core temperature, time, and subcutaneous adipose thickness during cryotherapy and rewarming.

Design: A multiple linear regression with 5 independent variables (skin temperature, body core temperature, subcutaneous adipose thickness, room temperature, time) predicting intramuscular (IM) temperature.

Setting: A sports injury research laboratory.

Participants: Fifteen volunteers with thigh skinfold measurements smaller than 40mm.

Interventions: Thirty-minute cryotherapy treatment (ice bag) followed by a 120-minute rewarming period.

Main Outcome Measures: The relationship between skin and IM temperature was described, and an equation predicting IM temperature by using room temperature, skin temperature, body core temperature, time, and adipose thickness was developed.

Results: Pearson's correlations between each predictor variable of IM temperature during cryotherapy were skin temperature, $r = .46$; skinfold, $r = .37$; time, $r = -.59$; core temperature, $r = .21$; and room temperature, $r = -.47$. During rewarming, the correlations were skin temperature, $r = .71$; skinfold, $r = .27$; time, $r = .76$; core temperature, $r = -.05$; and room temperature, $r = -.21$. A multiple regression equation ($R^2 = .76$) was developed to predict IM temperature during cryotherapy. A separate equation ($R^2 = .81$) was developed to predict muscle temperatures during rewarming.

Conclusions: During and after ice application, no single predictor adequately explained the change in IM temperature. Skin surface temperature was a weak predictor of IM temperature during cryotherapy and should not be used as the sole dependent measure in cryotherapy efficacy studies.

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CRYOTHERAPY IS COMMONLY used to treat musculoskeletal injuries.[1] Cold, applied directly to the skin, reduces both skin and deep tissue temperatures and is commonly used to reduce pain[2-4] and spasm,[5] and to retard edema formation.[4,6] Furthermore, cold may reduce secondary injury[7,8] through the suppression of cellular metabolism[1,9-12] and cellular oxygen consumption.[13] Decreased tissue temperatures also reduce blood flow and edema formation.[6,14] Because cryotherapy treatments reduce tissue temperatures and suppress cellular metabolism, they are desirable for acute injury management. Cryotherapy is also commonly used as a postacute adjunct to rehabilitation, particularly because of its analgesic, antispastic, and anti-inflammatory properties.[4,5]

In lieu of more direct measures, the clinical efficacy of cryotherapy has often been assessed through skin surface temperature measurements.[2,3,7,8,10,11,15-18] Skin temperature measurements have been used presumably because of the ease with which they can be obtained. One of the basic assumptions in the skin temperature–based cryotherapy literature is that changes in intramuscular (IM) temperature are strongly related to changes in skin temperature. This assumption may not be correct. In fact, there is cursory evidence[8,19] that skin temperature and IM temperature may not be strongly related.

Researchers[11,15,20,21] have shown an immediate increase in skin temperature with removal of cold. Others[8,19] reported a continued decrease in deep tissue temperature after removal of ice treatments. Still others[8,11,22,23] have reported that IM tissue temperatures do not return to preapplication temperatures during the 4 hours after ice application. However, in no study has the relationship between skin temperature and deep tissue temperature been reported because much cryotherapy research is based solely on skin temperature data.

In addition, there is some controversy over the effect of subcutaneous adipose layer thickness on intratissue temperature. Lowden and Moore[24] showed that temperature change in deep tissue is inversely related to skinfold ($r = .69$) and limb circumference ($r = .80$). Likewise, Johnson et al[23] observed a positive relationship between percentage of body fat and IM temperature during cryotherapy. More recently, Zemke et al[25] reported a weak but positive correlation between subcutaneous layer thickness and change in temperature during ice bag or ice massage application, suggesting that the thicker the fat layers, the greater the decrease in temperature. This relationship is directly opposite that reported by other researchers.[23,24]

The strength of the relationship between skin temperature and IM temperature is unknown. Past researchers have shown trends for both skin and IM temperatures, but no correlation or regression analyses have been reported. Therefore, the 2-fold purpose of this study was (1) to examine the specific relation-

ship between skin temperature and IM temperature (at 2cm below subcutaneous adipose layer) and (2) to describe the relationships between muscle temperature and the combination of skin temperature, room temperature, body core temperature, and subcutaneous adipose thickness.

METHODS

In this study, a multiple linear regression equation predicting IM temperature was developed. The variables used to predict IM temperature were subcutaneous adipose thickness, average skin interface temperature, body core temperature, room temperature, and time.

Subjects

Fifteen healthy volunteers (mean age ± standard deviation [SD] 22.9 ± 1.5yr; height, 169.2 ± 8.4cm; weight, 69.8 ± 13.1kg; anterior thigh skinfold, 21.2 ± 8.6mm) were recruited from a university student population. Individuals with vascular or neurologic pathology, or anterior thigh skinfold measurements greater than 40mm were excluded. The maximum skinfold criterion was chosen because subjects with greater skinfold thickness would require insertion of an IM thermocouple to depths deeper than is possible by using 21 gauge × 3.8cm (1.5in) hypodermic needles. Before participation, volunteers completed a health status questionnaire and gave informed consent. The school's human subjects committee approved the use of human subjects in this study. None of the subjects in this study reported negative reactions to the cold treatments or from the implantation of IM thermocouples.

Instruments

All data measurements were made by a sole investigator to reduce measurement variability. Skin and intratissue temperature data were collected by using type-T (copper-constantan) thermocouples[a] interfaced with a thermocouple thermometer (Iso-Thermex-16).[a] This equipment is accurate to within 1%.[26] Surface and ambient temperatures (controlled environment) were measured by using exposed-junction thermocouples with insulated leads (TX-31).[a] Intratissue temperatures were measured by using a single implantable, ungrounded-junction thermocouple (diameter = .4mm) with an insulated lead (TX-23-21)[a] implanted by using a 21 gauge × 3.8cm hypodermic needle. Implantable thermocouples were disinfected by immersion in CidexPlus™ 3.4% glutaraldehyde solution.[b] Core body temperature was measured by using a ThermoScan model HM3 infrared thermometer.[c] Skinfold measurements were made by using Lange skinfold calipers.[d]

Experimental Procedures

While supine on a treatment table and wearing shorts, subjects' right, anterior midthigh skinfold (vertical) was measured and used to determine the depth of the intratissue thermocouple placement, as described by Merrick et al.[8] For this study, IM thermocouples were inserted to a depth 2cm to the adipose layer (adipose thickness = one half of the skinfold thickness).[8]

After skinfold measurement, a small area on the middle of the right anterior thigh was shaved and cleaned. Thermocouples were placed on the prepared area in a 2 × 2 array. Three of the 4 thermocouples were placed on the skin surface and held in place with Dermaclear™ tape.[b] The fourth thermocouple (implantable) was inserted to a depth 2cm to the adipose layer by using a 21-gauge hypodermic needle, as previously described.[8] Proper insertion depth was calculated as one half of the skinfold thickness + 2cm. Measuring the distance from the skin surface to a known point on the thermocouple lead ensured that the correct insertion depth was achieved. After the thermocouple was implanted, the hypodermic needle was removed and the fine-wire thermocouple was secured in place with additional Dermaclear tape. One additional thermocouple (TX-31), located away from contact with subjects, was used to measure ambient temperature.

Body core temperature was determined by taking right tympanic membrane temperature. Body core, skin interface, and IM temperatures were recorded at 30-second intervals for the duration of the study. Subjects were supine for a minimum of 15 minutes before starting temperature measurements in an effort to allow body temperatures to stabilize and to control for temperature fluctuations resulting from any preexperiment physical activity.

After the thermocouple set-up, each subject received an experimental treatment divided into pretreatment (3min), treatment (30min), and posttreatment (120min) periods. During the pretreatment, subjects laid supine on the treatment table for 3 minutes to ensure that the temperature of the tissue around the intratissue lead was stable. Then, a 500-gram bag of cubed ice[e] was applied over the thermocouples on the thigh and was secured with a standard 15.2-cm (6-in) wide elastic wrap.[b] Air was evacuated from the icebag in an attempt to improve conforming of the bag to the thigh. The ice treatment lasted 30 minutes, after which the icebag and elastic wrap were removed. Posttreatment temperature measurements were made for 120 minutes after removing the icebag and wrap.

At the end of the posttreatment period, the thermocouples were removed. Antibiotic ointment and adhesive bandages were applied to the insertion point. Subjects received instructions regarding wound care and were also instructed to seek care at the student health center and to inform the investigator if any signs of infection were noted.

Statistical Procedures

A multiple regression model for predicting IM temperature during both the treatment (cooling) and posttreatment (rewarming) periods was developed. Predictor variables in these models included subcutaneous adipose thickness, mean skin interface temperature, body core temperature, ambient room temperature, and time. To develop the models, Pearson's product-moment correlations between each predictor variable and IM temperature were computed for both the cooling and rewarming periods by using SPSS, version 8.0 for Windows.[f] Based on their correlations to IM temperature, several curve estimations were developed for each predictor. Of these, the curve estimation equation providing the best prediction of IM temperature (ie, equation producing the highest R^2 value) was used in a forced multiple linear regression model to predict IM temperature.

All possible combinations of predictors were examined in an effort to explain the greatest amount of the variance in IM temperature. Two linear regression equations were developed to predict IM temperature, one during ice application and, the other, after the icebag was removed (rewarming).

RESULTS

Figure 1 represents the temperature values across time during cooling and rewarming. Table 1 contains Pearson's correlations between IM temperature and the best estimating models of the predictor variables and their type of best-fit model used. These best-fit models were used in a multiple linear regression equation predicting IM temperature for both cooling (during ice application) and rewarming (after ice application). Note that the complexity of the individual relationships makes these equations somewhat difficult to interpret.

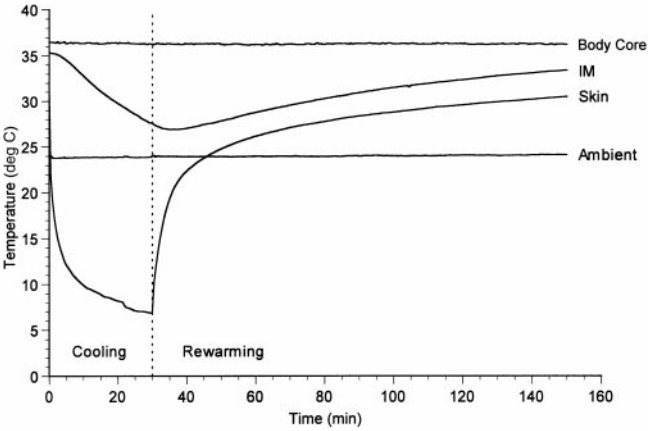

Fig 1. Time course for temperature variables (mean ± SD).

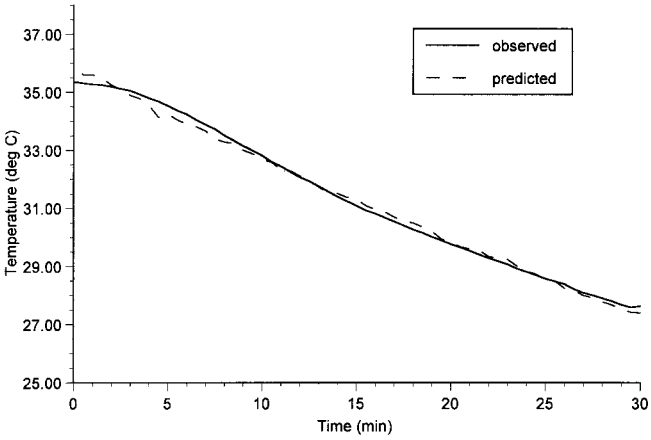

Fig 2. Observed and predicted 2-cm subadipose temperature during cryotherapy.

The IM cooling predictive equation ($R^2 = .76$) is as follows:

$$\text{Equation 1: IM temperature} = (-8.195) - (.00424 \times t) + \{.167[36.4485 - (39.908/T_S)]\} + \{.597[-6.577 + (4.9345 \times SF) - (.1971 \times SF^2) + (.0025 \times SF^3)]\} - \{.696[-1150.2 + (101.431 \times T_R) - (2.1720 \times T_R^2)]\} - \{.762[4883.14 - 201.48 \times T_C + .514 \times T_C^3]\}$$

where t = time, T_S = skin temperature, SF = skinfold, T_R = room temperature, T_C = body core temperature.

The IM rewarming predictive equation ($R^2 = .81$) is as follows:

$$\text{Equation 2: IM temperature} = (-44.152) - (.433 \times T_C) + \{.312[-9.5690 - 4.7115(\text{Int})]\} + \{.813[32.4771 - .8830 \times T_S + .0292 \times T_S^2]\} + \{1.176[50.5011(.9791^{T_R})]\} + \{.653[18.7262 + 1.3138 \times SF - .418 \times SF^2 + .0004 \times SF^3]\}$$

with the same variables as described for equation 1.

Figure 2 depicts the IM temperatures predicted from equation 1 and their relationship to the actual IM temperatures during the cooling period; figure 3 does the same for equation 2 during the rewarming period.

DISCUSSION

Temperatures During Cooling

IM temperatures (fig 1) declined in a pattern that is typical with this mode of cryotherapy.[8] During the course of the 30-minute cold treatment, IM temperature declined just over 8°C, whereas skin temperature declined approximately 27°C; nearly 3 times as much. Core temperature also declined slightly (2°C).

During cooling, no single predictor adequately explained IM temperature, but when combined in a least-squares multiple regression, these predictors were able to explain 76% of the IM temperature. Because multiple factors contribute to IM temperature during cryotherapy, it becomes rather complex to predict these temperatures and such prediction is probably not practical during most clinical treatments.

Of the predictor variables examined during cooling, time was the strongest single predictor of IM temperature ($R^2 = .35$). This finding was contrary to that of Lowden and Moore,[24] who reported that the $\log_{10}$ of time was a weaker predictor of IM temperature than either skinfold or arm circumference.

Neither body core temperature nor room temperature appears to have much influence on local IM temperature. Body core temperature explained only 4% of the IM temperature variance, whereas room temperature explained 23%. The small size of these relationships is probably related to the high degree of stability of both body core temperature (mean, 36.4° ± 0.4°C) and room temperature (mean, 23.8° ± 1.0°C). These relationships have not been reported in previous studies.

Skin interface temperature was also a weak predictor of IM temperature during cooling, explaining only 21% of the variance in muscle temperature. Additionally, it should be noted that skin temperature begins to warm immediately after removing the cold, while IM temperature continues to fall for several minutes. In fact, skin temperature explained less variance than room temperature. Clearly, use of skin surface temperature as a single predictor of the efficacy of cryotherapy on reducing IM

Table 1: Predictor Variable Relationships to IM Temperature

	During Cooling			During Rewarming		
Predictor	Pearson's *r*	R^2	Relationship to IM Temperature	Pearson's *r*	R^2	Relationship to IM Temperature
Skinfold (mm)	–.37	.14	Cubic	.27	.07	Cubic
Skin temp (°C)	.46	.21	Inverse	.71	.50	Quadratic
Core temp (°C)	.21	.04	Cubic	–.05	.00	Linear
Room temp (°C)	–.47	.23	Quadratic	–.21	.04	Compound
Time (min)	–.59	.35	Linear	.76	.58	Logarithmic

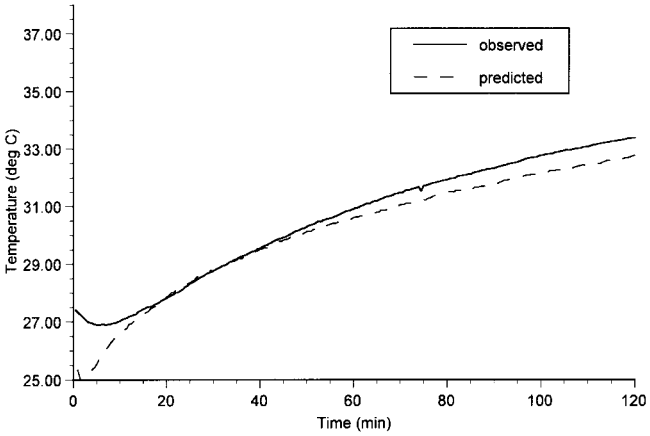

Fig 3. Observed and predicted 2-cm subadipose temperature during rewarming.

temperatures is inadequate. Instead, cryotherapy researchers should actually measure IM temperature, or, at the very least, they should collect enough additional data to allow its estimation by using a predictive equation such as that presented here.

Skinfold thickness was weakly related (table 1) to IM temperature, accounting for only 14% of the IM temperature variance, making it a weaker predictor than either room temperature or skin temperature. This suggests that subcutaneous adipose thickness plays a considerably smaller role during cryotherapy than has been previously suggested in the literature.[23,24] Lowden and Moore[24] showed a relatively strong inverse relationship (R^2 = .64–.47) between skinfold thickness and rate of muscle temperature change during 5 minutes of ice massage. Similarly, Johnson et al[23] reported a R^2 of .66, quite close to that of Lowden and Moore, for the relationship between the lowest IM temperature reached and percentage of body fat during lower leg immersion in 10°C water for 30 minutes.

The strength of the relationship between skin temperature and IM temperature in our study is similar in magnitude to that reported in Zemke et al.[25] In their study, they observed a weak relationship between subcutaneous thickness and time to change in IM temperature and lowest temperature. However, though our findings are similar in magnitude, they are opposite in direction. Zemke[25] reported greater change in temperatures with thicker skinfold measurements. Such findings contradict those of every other study examining this relationship.

All 3 studies[23-25] mentioned have significant methodologic differences that may explain the different findings. In fact, these differences are so great that it may not be appropriate to compare findings. One major difference is the depth at which temperatures were measured. In the studies by Lowden and Moore[24] and Johnson,[23] IM temperature was measured at absolute depths of 2 and 2.53cm, respectively; in our study and that of Zemke,[25] IM temperatures were measured at depths 2cm deep to the adipose layer.

Other differences among the studies are the modes of cryotherapy used. Ice massage was used by Zemke[25] and Lowden and Moore[24]; Johnson[23] used cold bath immersion; we used ice bags. The ice cups used in ice massage have less direct contact surface than ice bags or cold-water immersion. Cold-water immersion surrounds the entire limb, substantially increasing the surface area in direct contact with the cold. The body segments used in each study also varied. Zemke[25] and Johnson[23] examined temperature changes in the calf; Lowdon and Moore[24] examined the biceps brachii; we examined the anterior thigh. The quantity of subcutaneous adipose at each of these sites may differ and explain some of the variations. Last, different methods of reporting were used, making comparisons difficult. In 2 of the studies,[24,25] change in IM temperature was reported, rather than actual tissue temperatures.

Temperature During Rewarming

IM temperature did not return to baseline values during the 120-minute passive rewarming period observed in this study. As was the case during cooling, no single predictor adequately explained IM temperature during rewarming. However, when these variables were combined in a least-squares multiple regression, they explained 81% of the variance in IM temperature.

As was observed during the cooling period, time was the best single predictor of IM temperature (R^2 = .58) during rewarming. As a single predictor, skin temperature explained the second largest amount of IM variance (50%) during rewarming. Although much better than during cooling, skin temperature is still not a good predictor of IM temperature. This also suggests the need for direct measurement of muscle temperature during the rewarming portion of cryotherapy studies.

Skinfold thickness explained only 7% of the IM temperature variance during rewarming. Likewise, room temperature is also a poor single predictor, accounting for only 4% of variance. Core temperature was the poorest predictor, clarifying less than 1% of the variance.

Predicting Intramuscular Temperature

Two multiple regression equations (equations 1, 2) were developed to predict IM temperature because of the low predictive ability of any single variable during cooling or rewarming. Many of the individual variables in these equations appear as nonlinear functions, that includes cubic and logarithmic equations. These nonlinear functions were used because they maximized the R^2 of each individual variable in singly predicting IM temperature. Therefore, by combining these functions in the multiple regression equations, we accounted for the greatest amount of the variability in IM temperature.

The cooling equation (equation 1) explains 76% of the IM temperature variance. When comparing the predicted IM temperatures to those observed during cooling (fig 2), the difference between the predicted and observed is less than 1°C at each time point over the entire treatment. Previous investigators[24] also developed a regression model to determine the "end ice temperature." Although their equation is considerably less complex, it also accounts for less variance (72%) and was developed on the arm rather than on the leg.[24] In addition, skinfold represents a larger portion of the variance than our findings suggests.

The rewarming equation (equation 2) explains 81% of the muscle temperature variance. This equation appears to be less accurate during the first 6 minutes of rewarming (fig 3), the period during which IM temperature continues to decline after removing the ice. After this 6-minute period, the predicted IM temperature differs from the observed temperature by less than 1°C at each time point. The use of equations 1 and 2 should be limited to normal subjects under thermoneutral environmental conditions.

Methodologic Limitations

There are several methodologic restrictions that limit the generalizability of our findings. The first is the population

studied. This was a controlled laboratory study using readily available, college-aged subjects who were uninjured. The use of this select population hinders generalizing to other populations, such as geriatric or pediatric populations, because of differences (eg, in thermoregulation, adipose thickness, fat-free mass). Likewise, injured subjects may not respond in the same fashion. Further study of temperature changes in injured subjects is needed.

The second limitation is adipose thickness. Because our subjects were reasonably young, they tended to have relatively less adipose than would typically be seen in an older population. Likewise, we limited this study to subjects with anterior thigh skinfolds of less than 40mm. This was because of a procedural limitation. Our thermocouples are inserted by using hypodermic needles, and the length of the needle limits how deeply we may insert the thermocouple. In subjects with very thick adipose, we could not insert the thermocouple deeply enough to meet our measurement standard depth relative to adipose thickness.

The third limitation is ambient temperature. This study was conducted in a controlled-temperature environment in which there was very little variation in ambient temperature. The use of a limited range of values of a predictor variable, as is the case with both adipose thickness and ambient temperature in this study, limits the correlation between these variables and the dependent variable (IM temperature). For this reason, one should use caution when attempting to use our regression equations to predict IM temperature in subjects with very thick adipose thickness or in environments other than a temperature-controlled room.

Implications for Researchers

As previously mentioned, skin temperature is a poor single indicator of IM temperature during cryotherapy. Although it is a much better predictor during rewarming, it is still not adequate as a single measure of the IM cooling efficacy of cryotherapy treatments. Cryotherapy researchers should either collect enough additional data to estimate IM temperatures by using predictive equations such as those presented here, or they should directly measure IM temperatures directly. Likewise, care should be exercised when drawing conclusions from previous research in which skin temperature was used to assess the efficacy of the cryotherapy. For example, Mlynarczyk[18] suggested that 30 minutes was the most effective duration for cryotherapy treatments based on alteration of skin temperature and the length of time necessary for postcryotherapy rewarming. Inferences about IM temperature based on this study may not be correct. Similarly, care should be taken when comparing the results of studies in which dissimilar modes of cryotherapy, body parts, or reporting methods are used.

CONCLUSION

Our findings are important in the clinical use of cryotherapy. Neither skin temperature nor adipose thickness alone are good predictors of IM temperature over the duration of a cryotherapy treatment. Instead, time was the strongest single predictor of muscle temperature during cooling and rewarming. Composite temperature estimates are better still. It should also be noted that IM cooling did not plateau during the 30 minutes of cryotherapy application. Presently, the cryotherapy duration needed to reach a plateau in cooling IM temperatures is unknown and certainly is not reached during typical cryotherapy treatments. Therefore, clinicians should be most concerned about the duration of and the time between cryotherapy treatments. Unfortunately, these factors are not yet well described and additional research is needed.

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