Effect of the Forearm Tissue Temperature on the Cold Induced Vasodilation

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ABSTRACT

Recent work suggests an influence of the mean body skin (Tsk) and deep body temperatures (Tb) on cold-induced vasodilatation (CIVD). For example, minimum finger temperature (Tfi,min)was lower, and the maximum finger temperature (Tfi,max) was greater during CIVD when Tb was elevated, and the onset time of the CIVD response was reduced at higher Tsk. Question remain, though, about the influence of forearm tissue temperature on CIVD at a given Tsk and Tb. On two different occasions, eleven healthy male subjects pre-conditioned their forearm tissue at two different water temperatures (Tw), 20 and 38°C, until steady state forearm muscle temperature was achieved. After the conditioning period (129 ± 15 and 85 ± 15 min for 20 and 38°C, respectively), the fingers of the conditioned forearm were immersed in a 5°C water bath for 30 min. During finger immersion, Tsk and Tb were similar at each forearm skin temperature (Tsk = 34.3 ± 0.6°C, Tb = 36.8 ± 0.2°C), but temperature 3cm deep into the forearm's flexor digitorum profundus muscle differed significantly, averaging 23.6 ± 1.7°C when Tw was 20°C and 36.7 ± 0.6°C when Tw was 38°C. Arterial blood temperature in the radial artery measured at the wrist averaged 28.2 ± 2.5 and 35.6 ± 0.9°C for the 20 and 38°C conditions, respectively ($p < 0.05$). The two forearm conditions caused significant differences in all the CIVD parameters during the 30 min immersion in 5°C water. During the 38°C condition, the onset time for the CIVD was faster and the average, maximal and minimal Tfi were higher than during the 20°C condition. We concluded that a low forearm tissue temperature impedes the CIVD response despite normal Tsk and Tb, possibly by decreasing the temperature of the arterial blood to the fingers.

1.0 INTRODUCTION

Cold-induced vasodilation (CIVD) is a local cold vascular phenomenon likely caused by a decrease in the release of neurotransmitters from the local sympathetic nerves innerving the arterio-venous anastomoses (AVAs). This cold paralysis of the smooth muscle cells of the AVAs is responsible for the opening of the lumen of the arterioles and the subsequent increase of blood flow and the observed increase in tissue temperature of the digits.

The presence of this physiological reaction has a practical implication for people exposed to cold environments. Wilson and Goldman [1] observed that CIVD prevented freezing in fingers exposed to sub-zero ambient temperatures. Investigators thus believe that CIVD plays a substantial role in reducing the risk of local cold injuries [2], and may be beneficial for improving dexterity and tactile sensitivity during exposure to cold [3].

Many factors have been shown to influence the CIVD responses to cold. Daanen recently provided an extensive review on the topic [3]. One factor that is of particular relevance to this study is the effect of

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skin and core temperatures on the CIVD responses. Daanen and Ducharme [4] and Daanen et al. [5] reported that the CIVD responses are more pronounced when the body core and skin temperatures are warm (hyperthermic state) and suppressed when they are cold (hypothermic state) when compared to normothermia. A suppressed CIVD response will eliminate the risk of increasing the heat loss from the body for people already at risk of hypothermia. On the other hand, a pronounced CIVD response will promote the release of body heat (during local exposure of the extremities to cold water) for people who are hyperthermic.

The influence of the body skin and core temperatures on the CIVD responses raised the hypothesis that the CIVD is not a purely local mechanism. The systemic sympathetic drive may affect the CIVD response and impact on the magnitude of the response and on its onset time [3]. However, it is still possible that the CIVD may be a local phenomenon that is affected locally by the arterial blood flow and temperature perfusing the digits. In other words, a higher core temperature will elevate the blood flow and arterial blood temperature feeding the fingers. This factor alone can locally impact on the CIVD response by facilitating its development independently on the systemic sympathetic drive.

The objective of the present study was to evaluate the effect of changing the arterial blood flow and temperature to the fingers on the CIVD responses by altering forearm tissue temperature while keeping the mean skin and core temperatures neutral. Specifically, we tested the hypothesis that despite a constant mean skin and core temperature, the CIVD responses will be enhanced when the blood flow and arterial blood temperature to the fingers are increased.

2.0 METHODS

2.1 Subjects

Eleven non-smoking healthy male subjects were recruited for the study. The subjects were (mean ± SD) 27 ± 7 years of age, 177 ± 7 cm tall, and weighted on average 77 ± 12 kg. All subjects were informed of the details, discomforts and risks associated with the experimental protocol and had been granted medical approval, before they signed a written consent. The protocol was approved by the Human Ethics Committees of the Defence R&D Canada - Toronto and of the University of California in San Francisco where the study was conducted. Subjects were asked to avoid caffeine, alcohol, and strenuous physical activity for 12 hours before the tests.

2.2 Instrumentation

Mean skin temperature of the body (Tsk) was calculated by averaging the data obtained from 7 thermocouples (Type T; Mallinckrodt Medical Inc, St-Louis, MO) on the skin, according to the weighing coefficients from Hardy and Dubois [6, 7]. Core temperature was measured continuously by using a flexible cotton-covered Mon-a-therm thermocouple probe (Type T; Tyco-Mallinckrodt Medical Inc, St-Louis, MO) placed adjacent to the tympanic membrane (Tcore). The probe was gently inserted in the ear canal until it touched the tympanic membrane and subjects felt a pressure. It was then retracted slightly to relieve the pressure and was secured in place by occluding the aural canal with cotton and placing tape over the external ear surface. The temperature of the volar side of the distal phalanx of each finger (Tfi) of the left hand was continuously monitored with fine 40-gauge thermocouple (type-T) fixed to the skin with surgical tape. The hand was covered by an oversized thin surgical glove. The fingers were immersed to the metacarpophalangeal joints in a 25-L water bath controlled at 5ºC by a temperature controller (YSI, Yellow Springs, OH) and stirred by a Haake E52 jet pump (Haake, Berlin, Germany). The hand of the immersed fingers was at the level of the heart during the immersions.

The muscle temperature of the *flexor digitorum profundus* at a depth of 3 cm was continuously measured on the bulk part of the left forearm using a fine thermocouple probe (Tmusc) [8] according to a method

previously described [9]. The blood temperature of the radial artery at the wrist level of the left hand was continuously monitored using a fine thermocouple probe (external diameter of 0.5 mm) fitting a gauge 21 arterial catheter (Tblood). The size of the probe was small enough to permit an un-restricted blood flow through the radial artery.

2.3 Protocol

The subjects participated in 2 experimental sessions randomly assigned and separated by a week. The room temperature during the experimental session was maintained on average at 22.1 ± 0.7 ºC. The subjects, dressed in shorts and t-shirts, lay semi-supine on a bed while covered by a blanket. The coverage of the body with the blanket was adjusted to maintain thermal neutrality for the duration of the tests. After being instrumented, the subjects pre-conditioned their forearm tissue temperature in two different environments: cold and warm. The cold pre-conditioning consisted of immersing the forearm and hand in a well-stirred water bath maintained at 20ºC until the muscle temperature of the left forearm achieved a new steady-state (approximately 3 hours). The warm pre-conditioning consisted at immersing the left forearm and hand in the water bath maintained at 38ºC until the muscle temperature of the forearm achieved a new steady-state (approximately 1.5 hour). Once a new steady-state tissue temperature was achieved, a temperature controlled water-perfused sleeve (custom made; MedEng Inc, Ottawa, ON) covered the pre-conditioned forearm to maintain the skin temperature of the forearm at either 20 or 38ºC for the duration of the CIVD test. The fingers of the left hand were then immersed to the distal phalanx in a well-stirred water bath maintained at 5ºC for a period of 30 min.

2.4 Calculations and Statistical Analysis

The thermocouples were sampled every 10 sec and averaged over successive one-min periods. The CIVD responses were quantified by the following parameters (see Fig. 1): the minimum finger skin temperature (Tfi, min) before the first CIVD-phase; the maximum finger temperature (Tfi, max) during the first CIVD-phase; the onset time as the time from immersion to Tfi, min; the peak time as the time from Tfi, min to Tfi, max; the amplitude as the difference between Tfi, min and Tfi, max; and the mean finger temperature from the 5^{th} to the 30^{th} minute of immersion (Tfi).

All results are shown as a mean ± standard deviation (SD). Differences were considered significant when $P < 0.05$. The effect of pre-conditioning forearm tissue at each designated temperature on the CIVD parameters, core and mean skin temperatures, muscle temperature and arterial blood temperature were compared with paired t-tests.

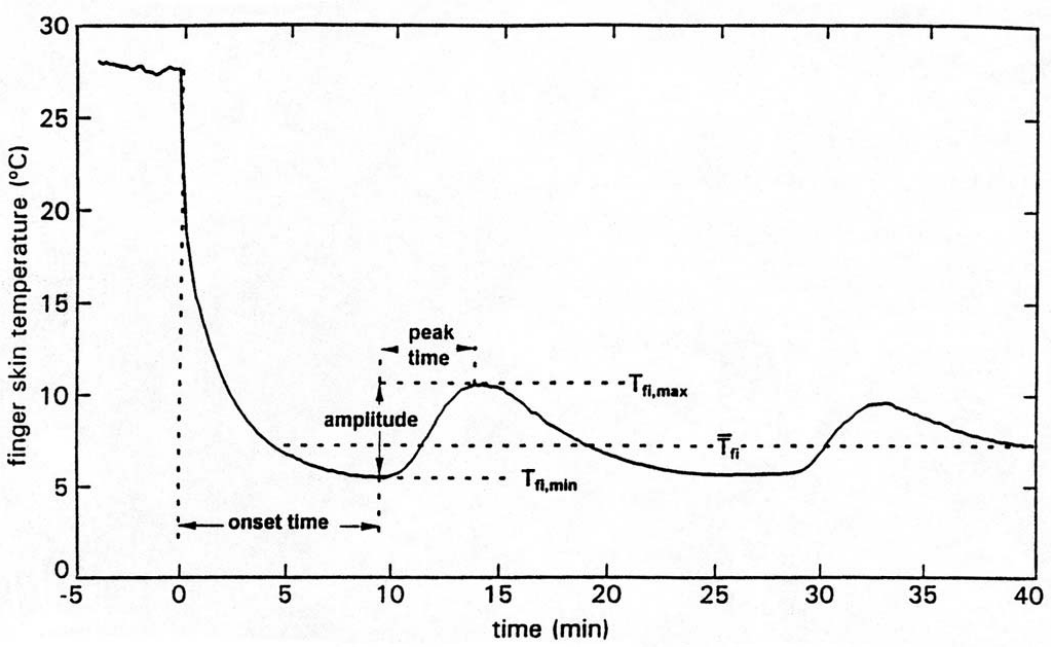

Figure 1: Definition of the parameters of the CIVD responses as measured in this study.

3.0 RESULTS

3.1 New Steady-State Temperatures

Table 1 presents the average Tcore and Tskin for the two thermal conditions of the forearm. No difference was observed between the two conditions: during both conditions the subjects were considered at thermal neutrality. Table 1 presents as well the steady-state temperatures 10 min before the pre-conditioning of the forearm, the steady-state temperatures during the last 10 min of the pre-conditioning period and the last 5 min of the CIVD test for Tmusc and Tblood. Initially, no difference was observed between the two conditions for both Tmusc and Tblood. The pre-conditioning in water at 38ºC increased Tmusc by 2.7ºC while it decreased it by 10.2ºC during the pre-conditioning at 20ºC. Similarly, the arterial blood temperature at the wrist level increases by 2.0ºC during the pre-conditioning in 38ºC water while it decreases by 8.9ºC at the end of the pre-conditioning period in water at 20ºC. By the end of the 30-min CIVD tests, the average temperature of the arterial blood entering the hand was 6.4ºC higher for the 38ºC pre-conditioning as compared to the 20ºC pre-conditioning.

**Table 1: Steady-state temperatures for the two thermal conditions of the forearm.
*: significantly different between the two conditions tested. N = 11.**

Measured parameters		Pre-conditioning of the forearm (mean ± SD)	
		20ºC	38ºC
Tcore (ºC)		36.8 ± 0.2	36.9 ± 0.2
Tsk (ºC)		34.1 ± 0.6	34.4 ± 0.5
Tmusc (ºC)	Initial	34.1 ± 0.6	34.0 ± 1.1
	End pre-conditioning	23.9 ± 0.3	36.7 ± 0.3*
	End CIVD	23.6 ± 0.7	36.6 ± 0.9*
Tblood (ºC)	Initial	34.9 ± 0.3	34.7 ± 0.4
	End pre-conditioning	26.0 ± 0.4	36.7 ± 0.3*
	End CIVD	30.4 ± 0.9	36.8 ± 0.5*

3.2 CIVD Responses

Table 2 presents the CIVD parameters for the two thermal conditions for all subjects. All the CIVD parameters that were calculated were significantly different between the two conditions tested. On average, pre-conditioning the forearm at 38ºC increased the Tfi by 2.1ºC, the Tfi, max by 2.8ºC, the Tfi, min by 0.6ºC, doubled the amplitude from 2.1 to 4,4ºC, and shortened the onset time and the peak time by 2.6 and 5.8 min, respectively.

Table 2: CIVD responses for the two pre-conditioning conditions of the forearm. N = 11.

CIVD parameters	Pre-conditioning of the forearm (mean ± SD)	
	20°C	38°C
Tfi,average (°C)	6.2 ± 0.9	8.3 ± 1.6*
Tfi,max (°C)	7.0 ± 1.1	9.8 ± 1.6*
Tfi,min (°C)	5.0 ± 0.1	5.6 ± 0.3*
Onset Time (min)	7.8 ± 1.4	5.2 ± 0.5*
Peak Time (min)	11.7 ± 5.2	5.9 ± 2.7*
Amplitude (°C)	2.1 ± 1.0	4.4 ± 1.6*

Tfi,average: mean finger temperature from the 5th to 30th minute of immersion
Tfi,max: maximal finger temperature during the first CIVD phase
Tfi,min: minimum finger temperature before the first CIVD phase
Onset Time: time from immersion to Tfi,min
Peak Time: time from Tfi,min to Tfi,max
Amplitude: difference between Tfi,max and Tfi,min
* $p < 0.05$

Figure 2 presents examples of the CIVD responses from two subjects during the pre-conditioning at 38 and 20ºC. The figure on the left is an example for a subject with an absence of CIVD response during the cold water test (non-responder). The figure on the right is an example for a subject presenting a good CIVD response during the cold water test (responder). In both cases, pre-conditioning the forearm in 38ºC water improves the CIVD response and transforms a non-responder into a responder.

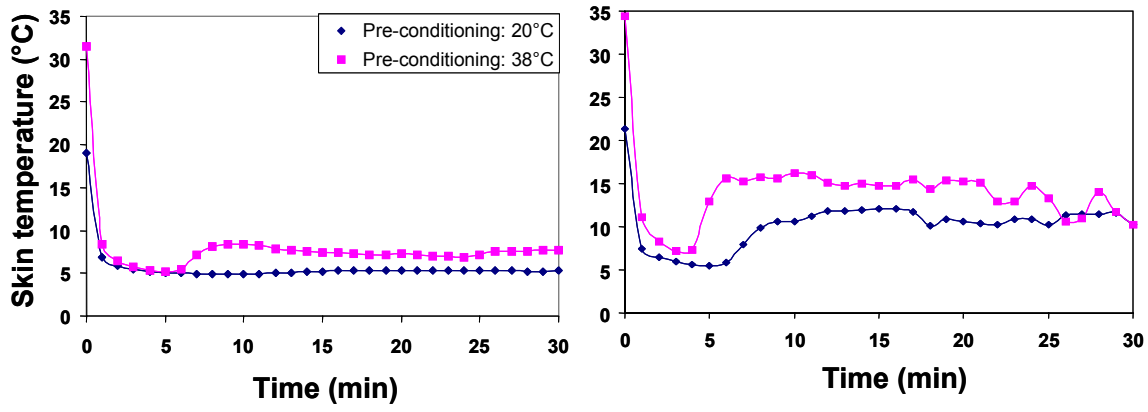

Figure 2: Examples of CIVD responses from two subjects following the pre-conditioning of the forearm in 20ºC and 38ºC water. The left figure shows the responses from a non-responder and the right figure from a responder.

4.0 DISCUSSION AND CONCLUSIONS

The results from the present study show that when the body heat content, as reflected by the skin and body core temperatures, is maintained constant, pre-conditioning the forearm tissue temperature to a new colder or warmer steady-state significantly alters the characteristics of the CIVD responses. This is likely attributed to the effect of the local warming and cooling of the forearm tissues on the arterial blood flow and the temperature of the arterial blood entering the hand and fingers. Indeed, Ducharme and Tikuisis [10] observed that for the same pre-conditioning of the forearm as in this study, the forearm and hand blood flow as measured by plethysmography was decreased to 1.4 ± 0.5 ml/min/100 ml tissue following the immersion in 20ºC water and significantly increased to 6.3 ± 1.1 ml/min/100 ml tissue following the immersion in 38ºC water.

The present study also show that warming the deep forearm tissues by 2.7 degrees increased the arterial blood temperature entering the hands by almost the same magnitude (2.0ºC; Table 1), while cooling the deep forearm tissues by 10.2ºC decreased the arterial blood temperature by a comparable amount (8.9ºC; Table 1). This effect is attributed to the convective heat transfer between the arterial blood and the tissues of the forearm maintained at a new thermal steady-state. Since mean skin and body core temperatures were not affected by the pre-conditioning treatments, we assume that the main effect on the CIVD responses can be attributed to the new steady state of the arterial blood flow and temperature entering the hand and feeding the fingers. If this hypothesis is accepted, then it may be possible that a similar mechanism can explain the effect of a change in core temperature on the CIVD responses. Indeed, an elevated core temperature will likely increase the blood flow to the periphery [11] and increase the arterial blood temperature reaching the fingers, thus changing the local thermal status of the hand and fingers which may facilitate the CIVD responses to a cold water immersion.

Practically, this study shows that local warming of the forearm tissues would facilitate CIVD responses and could potentially transform a non-responder into a CIVD responder. Improving the CIVD responses to cold has the potential of reducing the risk of developing frostbite for people with poor CIVD responses. Actively warming the forearm could be a practical method of enhancing cold protection for people working with their hand in the cold. However, it would be unlikely that this approach will work if the body has a decreased body heat content.

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