Effects of Various Environmental Temperatures on Effort Angina

Claes Lassvik and Nils-Holger Areskog

From the Department of Clinical Physiology, University of Linköping, Region Hospital, Linköping, Sweden

ABSTRACT

Eleven patients with effort angina and a history of cold intolerance performed short-term bicycle exercise tests at various room temperatures, 20, 10, 0 and -10° C, and a few patients also at -30° C. A significant reduction of maximal working capability (expressed as maximal work load, MWL), limited by moderately severe angina, was found at -10° C (7% ⁺ 1, SEM, P<0.05) compared with normal room temperature. At 0 and 10° C changes of MWL were small and not significant, and at -30° C no further decrease of MWL was seen. About half of the patients, however, showed a tendency toward a decrease in MWL with decreasing environmental temperature, and the decrease in MWL correlated significantly with an increase in rate pressure product (RPP) during exercise at both 0 and -10° C. Thus, the decrease in working capability on exposure to cold could be explained by an increase in heart work. Warming up effects of exercise, counteracting the cold-induced increase in peripheral vascular resistance, were indicated by a diminishing difference in systolic blood pressure between a cold and normal environment with increasing work time.

INTRODUCTION

As early as in 1786, Heberden (4), in his classical description of angina pectoris, noted that cold weather could cause aggravation of symptoms. In a pilot study by the present authors a majority (96%) of a material of 50 anginal patients gave a history of cold intolerance. The main physiological reason for the unfavourable effect of exposure to cold is an increase in the peripheral vascular resistance, causing an increase in the systemic blood pressure (BP) and in the myocardial oxygen consumption (8). The BP increase might thus decrease the myocardial oxygen reserve and the maximal working capability in anginal patients. Exercise, on the other hand, causes muscular vasodilation and a decrease in the total vascular resistance, which might reduce the effects of exposure to cold. This study was carried out to investigate

- whether exposure to various environmental temperatures causes a decrease in the working capability in patients with effort angina and a history of cold intolerance,
- whether the changes of working capability correlate to changes of simple non-invasive hemodynamic variables and
- whether the physiological effects of cooling might be overcome by warming-up effects during short-term exercise.

MATERIAL

Eleven male patients (all non-smokers, mean age 55 years, range 45 - 61) with a history of cold intolerance were invited and gave their informed consent to participate in the study, which was approved by the Ethical Committee of the Medical Faculty. All of the patients had a history of typical and stable effort angina for at least 12 months, and 3 patients had suffered one or more myocardial infarctions. All had exercise-induced anginal pain and ECG changes, with 0.2 - 0.6 mV horizontal or down-sloping ST depression at the end of exercise. At a standardized stepwise ergometer test their working capability, expressed as W_{max} 6 min (11) was on an average 95 W (range 70 - 121), in all cases limited by angina. None had hypertension. Six patients were taking beta-adrenergic blocking agents in stable doses. No other medication except short-acting nitroglycerine was allowed.

METHODS

The exercise tests were performed in a climate chamber at room temperatures of $10^{\circ}C \stackrel{+}{-} 1$ (SD), $0^{\circ}C \stackrel{+}{-} 1$, $-10^{\circ}C \stackrel{+}{-} 1$ and $-30^{\circ}C \stackrel{+}{-} 1$, with a wind velocity of 2.2 m/s $\stackrel{+}{-} 0.1$, and outside the chamber at $20^{\circ}C \stackrel{+}{-} 2$ and no wind. The relative humidity was $40\% \stackrel{+}{-} 8$ at 20 and $10^{\circ}C$ and was calculated to be 40 - 75% at 0, -10 and $-30^{\circ}C$. Temperatures were measured with Cu/Cn thermocouples, wind velocity with a warm-wire anemometer and humidity with a hair hygrometer.

At normal room temperature the patients were dressed in normal indoor clothing and at the lower temperatures standardized clothing was added. The mean skin temperature was found to decrease by 5.2° C during exercise in a cold environment (-10°C, 2.2 m/s) with this clothing.

Ten of the patients performed exercise tests at room temperatures of 20, 10, 0 and -10° C with 30-min rest intervals between the tests and with randomization of the test order. An initial trial test at normal room temperature was always carried out. Thus the patients performed 5 serial exercise tests each time. Three of the patients performed exercise tests at normal room temperature and

at -30° C. One patient exercised only at 20, -10 and -30° C, and his results are included only in the study at a room temperature of -30° C.

Sitting exercise tests were performed on a bicycle ergometer (Siemens-Elema 380) with an initial load of 50 W and with a continuous increase of the load by 10 - 30 W per min. Load increments were chosen to give a total exercise time of 4 - 8 min, with moderately severe angina as the end-point of exercise. A precordial ECG (CH_{1-6} , with the forehead as reference) was recorded continuously at a paper speed of 10 mm/s, and ST depression at the appearance of angina and at maximal work was measured at a paper speed of 50 mm/s. The heart rate (HR) was calculated from the recordings and the systolic blood pressure (BP) was measured by the cuff method. For further details, see Lassvik & Areskog (7).

HR and BP were measured after 5 min of sitting on the bicycle before the exercise, every min during exercise, just before termination of the test and after exercise every min for 4 min. The rate pressure product (RPP = HR \cdot BP) was calculated. During exercise, the work load at the appearance of pain (APWL) and the maximal work load (MWL) were noted. Another two points during exercise were selected, describing events at individually equal work loads, namely after 3 min, when most of the patients did not experience pain, and after approximately 4.5 min, when all patients had anginal pain. After exercise, the time for disappearance of pain (DPT) was noted. The tests were performed at the same time of day by each patient. Eating, intake of nitroglycerine and muscular effort were not allowed for 2 hours before the tests.

The results were evaluated by Student's t-test for paired observations and regression analyses (1).

RESULTS

The working capability, expressed as APWL and MWL, did not change significantly at room temperatures of 10 and 0°C but decreased significantly at -10° C compared with normal room temperature (both by 7 \pm 3 %, mean \pm SEM, P<0.05, Table 1). The individual reactions varied markedly (Fig 1). The decrease in MWL exceeded 5% in only 1/10 patients at 10°C, in 3/10 at 0°C and in 6/10 at -10° C, compared with normal room temperature. The ST depression at APWL and MWL was somewhat greater at 0 and -10° C than at normal room temperature, but the differences were not significant. DPT did not differ significantly between any of the environments (Table 1).

The heart rate did not change significantly between any of the environments (Table 2). At rest and after exercise BP was higher the lower the environmental temperature, indicating progressively increasing vasoconstriction. On the other hand, <u>during exercise</u> the absolute BP increase compared with the BP at normal room temperature was relatively smaller and significant only at 0 and -10° C.

	Room temperatures				
	20 ⁰ C (control)	10 ⁰ С (а	0 ⁰ C Ibsolute char	-10 [°] C nges)	
Work load at appearance of pain, APWL (W)	128 - 6	1 + 1	-3 ± 2	-9 * 2 **	
Maximal work load, MWL (W)	140 - 10	4 - 1	- 2 ⁺ 1	-10 - 3 *	
Time of disappearance of pain, DPT (s)	142 ± 52 -	-31 ± 35	-43 ⁺ 42	-34 [±] 39	
ST depression at APWL (mV x 10)	1.5 ⁺ 0.1	0 ± 0	0.3 - 0.2	0.2 + 0.2	
ST depression at MWL (mV x 10)	2.5 - 0.2	0 ± 0	0.2 - 0.2	0.2 ± 0.2	

Table 1. Angina and ST depression. Control values at normal room temperature and absolute changes in cold environments.

Mean values ⁺ SEM. * P<0.05, **P<0.01



Fig 1. Work performance, expressed as MWL, at various room temperatures. Individual reactions.

This indicates a decrease in peripheral vascular resistance during exercise, probably a warming-up effect. After exercise the BP increase on exposure to cold, compared with normal room temperature, was even more marked than before exercise (Table 2).

Compared with normal room temperature, a significantly higher RPP was found <u>at rest</u> at all cold temperatures. <u>During exercise</u> the differences between the cold environments and normal room temperature progressively diminished. At 3

min of exercise the difference was significant at 0 and -10° C and at 4 min of exercise only at -10° C. At the end of exercise (MWL) the difference in RPP was not significant at any cold temperature. This implies that the initial increase in myocardial oxygen demand in a cold environment gradually decreases during exercise. After exercise RPP was again significantly higher after 2 min at 0 and -10° C, and after 4.5 min at all cold temperatures (Table 2).

		Room temperatures			
		20 ⁰ C	10 ⁰ C	0°C	-10 ⁰ C
		(control)		(absolute changes	s)
Heart rate Rest Exercise, MWL Recovery,	e, beats/min 3 min 4.5 min 2 min 4 min	$\begin{array}{c} 62 & \frac{+}{+} & 9 \\ 93 & \frac{+}{+} & 9 \\ 111 & \frac{+}{+} & 18 \\ 116 & \frac{+}{+} & 20 \\ 67 & \frac{+}{+} & 16 \\ 64 & - & 15 \end{array}$	7 + 42 + 11 + 11 + 11 + 12 + 12 - 1	5 + 3 2 + 1 2 + 1 2 + 1 0 + 1 -2 - 2	$\begin{array}{c}2 & + & 1\\3 & + & 2\\3 & + & 1\\0 & + & 1\\-5 & + & 3\\-5 & + & 3\end{array}$
Blood pres Rest Exercise, MWL Recovery,	ssure, mm Hg 3 min 4.5 min 2 min 4 min	$\begin{array}{c} 109 & \stackrel{+}{=} & 7 \\ 151 & \stackrel{+}{=} & 17 \\ 164 & \stackrel{+}{=} & 25 \\ 168 & \stackrel{+}{=} & 29 \\ 137 & \stackrel{+}{=} & 12 \\ 121 & \stackrel{-}{=} & 13 \end{array}$	$\begin{array}{c} 14 & \stackrel{+}{-} & 4 & ** \\ 5 & \stackrel{+}{-} & 4 \\ 0 & \stackrel{+}{-} & 1 \\ 0 & \stackrel{+}{-} & 1 \\ 9 & \stackrel{+}{-} & 3 & * \\ 10 & \stackrel{-}{-} & 3 & ** \end{array}$	$\begin{array}{cccccccccccccccccccccccccccccccccccc$	$\begin{array}{cccccccccccccccccccccccccccccccccccc$
Rate press units • 10 Rest Exercise, MWL Recovery,	sure product, 3 min 4.5 min 2 min 4 min	68 ⁺ 12 141 ⁺ 27 182 ⁺ 49 195 ⁺ 57 91 ⁺ 19 77 ⁺ 18	17 + 5 * 7 + 5 2 + 2 2 + 2 8 + 5 10 - 5 *	$\begin{array}{c} 20 & \stackrel{+}{-} & 4 & *** \\ 13 & \stackrel{+}{-} & 5 & * \\ 7 & \stackrel{+}{-} & 5 \\ 6 & \stackrel{+}{-} & 4 \\ 14 & \stackrel{+}{-} & 4 & ** \\ 21 & \stackrel{-}{-} & 4 & *** \end{array}$	$ \begin{array}{r} 18 \\ + \\ 14 \\ + \\ + \\ 5 \\ + \\ 10 \\ + \\ 5 \\ + \\ 6 \\ + \\ 4 \\ 16 \\ + \\ 18 \\ + \\ 6 \\ + \\ 4 \\ + \\ 8 \\ + \\ 6 \\ + \\ \end{array} $

Table 2. Heart rate, systolic blood pressure and rate pressure product (HR \cdot BP) at various room temperatures. MWL = maximal work load.

Mean values [±] SEM. * P<0.05, ** P<0.01, ***P<0.001.

At 10° C the decrease in MWL correlated significantly with the increase in HR at 4.5 min exercise (P<0.05, Table 3). At 0 and -10° C the decrease in MWL correlated significantly with the increase in HR at 3 min, in BP at 4.5 min and in RPP at both 3 and 4.5 min of exercise (P<0.05). No correlation was found between the decrease in MWL and work time, performed work or medical therapy (beta-blockers).

The 3 patients who exercised at -30° C did not show any further decrease in working capability (Fig. 1) or increase in HR, BP and RPP compared with -10° C.

However, at rest a marked increase in BP was found, on an average $16 \stackrel{+}{-} 4 \text{ mm Hg}$, indicating a further increase in peripheral resistance.

Table 3. Coefficients of correlation for regression between relative (%) changes of MWL and relative (%) changes of heart rate (HR), systolic blood pressure (BP) and rate pressure product (RPP) at various environmental temperatures.

	10 [°] C	0 [°] C	-10 [°] C
HR 3 min	-0.29	-0.64 *	-0.68 *
HR 4.5 min	-0.71 *	-0.42	-0.53
BP 3 min	-0.35	-0.40	-0.47
BP 4.5 min	-0.08	-0.70 *	-0.62 *
RPP 3 min	-0.39	-0.69 **	-0.65 *
RPP 4.5 min	-0.58	-0.73 *	-0.62 *

* P<0.05, ** P<0.01

DISCUSSION

Few studies appear to have been made concerning the effects of cold on effort angina under controlled conditions. Riseman & Stern (9) and Freedberg et al. (3) found that almost 1/3 of their patients performed a smaller number of steps (Master's step-test) after a few minutes of exposure to an environmental temperature of about 10° C, with no wind and hospital clothing. In a pilot study, Koch (5) found a significant reduction of the maximal working capability in a moderately cold room (0 - 4° C) with no wind. The heart rate (HR) and systolic blood pressure (BP) increased significantly during exercise. In the study by Epstein et al. (2), with light exercise and mild cooling (15° C), an increase in peripheral vascular resistance and mean blood pressure was found, with no difference in reactions between normal subjects and patients with angina pectoris.

In this investigation serial exercises were used and in an earlier study the reproducibility of effort angina was found to be good, with no successional effects. The coefficient of variation of maximal work load (MWL) was on an average 4% (6). In another study no successional effects of exposure to cold were found (7). Thus, the exercise procedure was considered to be suitable.

This study has shown that although a low environmental temperature may cause a marked decrease in the working capability in patients with effort angina and a history of cold intolerance, some patients are little affected. Mild cooling is well tolerated by most patients, at least in the laboratory situation used here.

The values for rate pressure product (RPP) at the end of exercise did not differ at the various environmental temperatures. This indicates that the relationship between myocardial oxygen demand and RPP (10) is valid also in a cold environment. Thus, as the cold-induced decrease in MWL correlated significantly with the increase in RPP during exercise, both at 0° C and at -10° C, the observed decrease in working capability was probably caused by an increase in heart work. This is in accordance with the findings in our earlier study at a room temperature of -10° C (7).

During exercise the difference in BP between the cold environments and normal room temperature diminished. Thus, the increase in peripheral resistance was probably counterbalanced by exercise-induced muscular vasodilation. One reason for the observed difference in cold susceptibility might be a difference in warming-up during exercise, due to differences in work time and load. This was not supported by the results, however. Therefore, differences in autonomic reactivity during exercise in a cold environment (sympathetic stimulation and vagal withdrawal, respectively) are likely to be present. However, patients with a very limited coronary reserve might suffer more, because of an insufficient time for warming-up effects to be exerted.

In contrast to the findings of Epstein et al. (2), the increase in resting BP caused by mild cooling $(10^{\circ}C)$ was completely abolished during exercise, and the changes in MWL were negligible. Their loads were, however, relatively lower than ours, and the work time shorter. Further, we found only minor adverse effects of exposure to a very cold environment $(-30^{\circ}C)$ compared with $-10^{\circ}C$, which seems remarkable. During exercise, however, the marked increase in BP noted at rest rapidly diminished, and RPP during exercise did not differ between the two environments.

In conclusion, patients with effort angina and a history of cold intolerance showed a significant, though variable decrease in working capability on exposure to a room temperature of -10° C, while only a tendency towards a decrease was observed at 0° C. The increase in HR and BP during exercise could explain the deterioration of work performance at both -10° C and 0° C. Warming-up effects of exercise seem to be important, and explain why a temperature of 10° C caused no deterioration of work performance compared with a normal room temperature.

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Address for reprints:

Dr Claes Lassvik Department of Clinical Physiology University Hospital S-581 85 Linköping Sweden