Effect of Warming of Exercising Legs on Exercise Capacity in Patients with Impaired Exercise Tolerance

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SUMMARY

Objective. The purpose of this study was to determine whether warming of exercising legs improves exercise capacity in patients with cardiac disease and low exercise tolerance. **Background.** Exercising muscle temperature reflects both muscle metabolic rate and muscle blood flow. Increase in muscle temperature in exercising legs is impaired in patients with chronic heart failure. We hypothesized that the mechanisms responsible for impairment of temperature increase in exercising muscle might be related to those responsible for low exercise tolerance. **Methods.** We studied 17 patients with cardiac disease whose anaerobic threshold (AT) ranged from 6.6 to 14.8 ml/kg/min (mean 11.2 ± 1.9 SD). Subjects performed symptom-limited sitting cycle ergometer exercise with or without warming of the thighs. Both thighs were warmed by use of hot packs fixed by supporters. To determine the effect of the supporters themselves on AT and peak $\dot{V}O_2$, the same ergometer exercise was performed by 7 patients with or without supporters. Peak $\dot{V}O_2$ and AT were determined by concomitant respiratory gas monitoring. **Results.** 1) Warming of the thighs increased deep temperature in the thighs ($1.0 \sim 2.8^\circ\text{C}$). 2) AT and peak $\dot{V}O_2$ were significantly improved in the warming exercise compared with the non-warming exercise ($p < 0.01$, $p < 0.01$). 3) There was no significant difference in AT or peak $\dot{V}O_2$ between the exercises with and without supporters. **Conclusion.** The findings of this study indicate that warming of exercising legs improves exercise capacity in patients with cardiac disease and low exercise tolerance. (Jpn Heart J 1996; 37: 855-863)

Key words: Warming Exercise tolerance AT Peak $\dot{V}O_2$ Deep thermometer

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EXERCISING muscle temperature reflects both muscle metabolic rate and muscle blood flow. Regulation of body temperature during exercise in healthy subjects has been studied by numerous investigators.¹⁻⁸⁾ Shellock et al have shown that muscle temperature elevation in exercising legs is impaired in patients with chronic heart failure,^{9,10)} and Bergh and Ekblom have shown that physical performance is impaired when muscle temperature is low in normal healthy subjects.¹¹⁾ We hypothesized that the mechanisms responsible for impairment of temperature increase in exercising muscle might be related to those responsible for low exercise tolerance. This study was designed to determine whether warming of exercising legs improves exercise capacity in patients with cardiac disease and low exercise tolerance.

METHODS

Study subjects: This study included 17 patients with cardiac disease (all males). The mean age was 62.1 years (SD 6.7; range 47–75 years). Thirteen had ischemic heart disease, 1 hypertensive heart disease and 3 had cardiomyopathy. Patients who had symptoms of overt heart failure were excluded.

Study protocol: An electromagnetically controlled cycle ergometer was used in a room with ambient temperature of 24–25°C for exercise studies. Symptom-limited ramp exercise testing was performed with or without warming of the thighs with a 1-hour interval between tests. (Figure 1). The tests were performed in random order. In the warming exercise, both thighs were warmed using hot packs fixed by supporters. Exercise began with a 4-minute warm-up at 0 W,

Figure 1. Diagram of exercise protocol.

followed by 1-W incremental loading every 6 seconds. The electrocardiogram and heart rate were monitored by a 12-lead electrocardiograph. Blood pressure was measured by the cuff method once per minute using an automatic indirect manometer. Expired O_2 and CO_2 and the rate of air flow were measured at rest (sitting on the ergometer) and throughout the exercise period. $\dot{V}O_2$, CO_2 output ($\dot{V}CO_2$) and minute ventilation ($\dot{V}E$) were measured every 30 seconds.

The AT was determined visually by two experienced reviewers and expressed as $\dot{V}O_2$ (ml/kg/min) using the following two criteria: 1) the $\dot{V}E/\dot{V}O_2$ curve, which had been flat or falling, begins to rise as the $\dot{V}E/\dot{V}CO_2$ curve remains constant or falls and 2) the gas exchange ratio, which had been flat or gradually increasing, begins to increase more rapidly.¹²⁾ Peak $\dot{V}O_2$ was determined from data obtained during the final 30 seconds of exercise. The relation of the increase in $\dot{V}O_2$ to the increase in work rate (WR) ($\Delta\dot{V}O_2/\Delta WR$) was determined by Hansen's method.¹³⁾

Deep and skin temperatures were measured at rest and throughout the exercise period using a deep body thermometer with deep and skin probes, respectively. To determine the effect of supporters themselves on AT and peak $\dot{V}O_2$, sitting cycle ergometer exercise was performed by 7 patients with or without the supporters with a 1-hour interval between tests. The tests were performed in random order. The reason for stopping exercise was leg fatigue in all patients. No patient experienced chest pain or oppression during exercise or ischemic changes on ECG. Exercise testing was performed on medication (nitrates, Calcium antagonist) in all patients except one.

Statistical analysis: Data were analyzed statistically using analysis-of-variance techniques and Students t-test; the criterion for statistical significance was $p < 0.05$. Values are given as means $\pm$ SD (standard deviation).

RESULTS

Temperature changes: The resting deep temperature of the thigh was increased from $35.3 \pm 0.4^\circ C$ to $37.3 \pm 0.4^\circ C$ after the warming period ($p < 0.01$) (Figure 2). Deep temperature at peak exercise was $35.7 \pm 0.3^\circ C$ in the non-warming studies, but was $37.9 \pm 0.5^\circ C$ in the warming studies ($p < 0.01$). Thigh skin temperature at rest increased from $32.9 \pm 0.7^\circ C$ to $38.4 \pm 1.8^\circ C$ after the warming period ($p < 0.01$) (Figure 2). At the end of exercise, the skin temperature was $33.4 \pm 0.7^\circ C$ in the non-warming studies, but was $38.2 \pm 0.9^\circ C$ in the warming studies ($p < 0.01$). These findings indicate that the warming effects of hot packs continued throughout the exercise test period.

Figure 3 shows the deep and skin temperatures of the forearm (non-exercising part of the body); there were no significant differences between the warming

Figure 2. Changes in deep and skin temperatures of the thigh. Comparison of the warming studies and the non-warming studies at rest and peak exercise. In the warming studies, deep and skin temperatures of the thigh (exercising portion) were consistently higher than those in the non-warming studies.

Figure 3. Changes in deep and skin temperatures of the forearm. Comparison of the warming studies and the non-warming studies at rest and peak exercise. Bilateral thigh (exercising portion) warming had no effects on deep and skin temperatures of the forearm (non-exercising portion).

studies and the non-warming studies. These findings indicated that bilateral warming of the thighs (exercising muscles) did not affect the deep and skin temperatures in the non-exercising portion of the body.

Hemodynamic changes: Figure 4 shows systolic blood pressures (SBP) at rest

Figure 4. Changes in heart rate and systolic blood pressure. Comparison of the warming studies and the non-warming studies at rest and peak exercise. Bilateral thigh warming had no effects on systolic blood pressure (SBP) and on resting heart rate (HR).

Figure 5. AT and peak $\dot{V}O_2$. Comparison of the warming studies and the non-warming studies. AT in the warming studies was significantly higher than that in the non-warming studies ($p < 0.01$). Peak $\dot{V}O_2$ in the warming studies was also significantly higher than that in the non-warming studies ($p < 0.01$).

and peak exercise. Neither differed significantly between the warming and non-warming studies. Heart rate (HR) at rest did not differ between the two studies, while that at peak exercise increased in the warming studies ($p < 0.025$), probably due to improved exercise tolerance (Figure 4). These findings suggest that bilateral warming of the thighs does not modify systemic hemodynamics at rest and or SBP at peak exercise.

Figure 6. $\Delta\dot{V}O_2/\Delta WR$. Comparison of the warming studies and the non-warming studies. $\Delta\dot{V}O_2/\Delta WR$ in the warming studies was significantly higher than that in the non-warming studies ($p < 0.01$).

Figure 7. AT and peak $\dot{V}O_2$. Comparison of the studies with (+) and without (-) supporters. There was no significant difference in AT or peak $\dot{V}O_2$ between the studies.

Exercise tolerance: Figure 5 shows AT and peak $\dot{V}O_2$ in the warming and non-warming studies. AT (ml/kg/min) in the warming studies was significantly higher than that in the non-warming studies (13.1 ± 2.6 vs 11.1 ± 2.0 , $p < 0.01$). Peak $\dot{V}O_2$ (ml/kg/min) in the warming studies was significantly higher than that in the non-warming studies (17.6 ± 2.4 vs 15.5 ± 2.8 , $p < 0.01$). Peak work rate (W/min) in the warming studies was also significantly higher than that in the non-warming studies (93.0 ± 10.6 vs 86.0 ± 12.6 , $p < 0.01$). Furthermore, the

$\Delta\dot{V}O_2/\Delta WR$ in the warming studies was significantly higher than that in the non-warming studies (9.4 ± 0.9 vs 8.3 ± 0.8 , $p < 0.01$) (Figure 6).

Effects of supporters: Figure 7 shows AT and peak $\dot{V}O_2$ in the 7 patients, with or without supporters. Peak $\dot{V}O_2$ decreased in the studies with supporters compared to those without supporters, however the difference did not attain statistical significance. AT was not significantly different between the two studies. Peak work rate (W/min) decreased in the studies with supporters compared to those without supporters (80.0 ± 11.5 vs 88.6 ± 14.6 , $p < 0.05$). The $\Delta\dot{V}O_2/\Delta WR$ was not significantly different between the two studies (with supporters 8.6 ± 0.7 , without supporters 9.2 ± 1.4).

DISCUSSION

The first thermoregulatory study of body temperature elevation during exercise was reported by Nielsen¹⁾ who showed that increase in the rectal temperature occurred in proportion to work intensity and $\dot{V}O_2$ during exercise. Since then, the body temperature changes associated with exercise in healthy subjects have been investigated in numerous studies,²⁻⁸⁾ although only a few studies have been conducted in patients with cardiac disease.^{8,14)} Shellock et al have reported that muscle temperature elevation in exercising legs is impaired in patients with chronic heart failure.^{9,10)} We hypothesized that the mechanisms responsible for impairment of temperature increase in exercising muscle might be related to those responsible for low exercise tolerance. Therefore, we attempted to determine whether warming of exercising legs improves exercise capacity in patients with cardiac disease and low exercise tolerance. In this study, peak $\dot{V}O_2$ (ml/kg/min) and AT (ml/kg/min) ranged from 11.2 to 19.8 and 6.6 to 14.2, respectively. These values were significantly lower than those in normal healthy Japanese individuals.¹⁵⁾

Mechanisms of warming effect: The findings of this study suggest that warming of exercising muscles with hot packs fixed by supporters improves exercise tolerance (AT, peak $\dot{V}O_2$ and peak work rate) in patients with low exercise tolerance, although supporters themselves had a decreasing effect on peak $\dot{V}O_2$ and peak work rate.

However, the mechanisms by which warming exerts this effect are unclear. Possible mechanisms might include 1) facilitation of enzymatic activities (the metabolic rate in the living body depends on tissue temperature, the higher the tissue temperature, the higher the metabolic rate, as long as enzymes and proteins do not degenerate), 2) increased oxygen delivery to tissues from hemoglobin in the exercising muscle and 3) an increased muscle blood flow due to vasodilation. The present study showed that warming of exercising muscles improved not

only peak $\dot{V}O_2$ (AT) but also the $\Delta\dot{V}O_2/\Delta WR$. Hansen et al have suggested that the $\Delta\dot{V}O_2/\Delta WR$ is an index of oxygen delivery to metabolically active cells.¹³⁾ Therefore, warming of muscles may facilitate oxygen delivery to working muscles. Also, the mechanism by which supporters themselves lower peak $\dot{V}O_2$ is unclear. Output to the blood of lactate produced in the muscles might be delayed by the compression of the supporters.

Measurement techniques: In this study, deep and skin temperatures were measured with a deep body thermometer. The deep body thermometer was designed by Fox¹⁶⁾ and modified by Togawa and Nemoto.¹⁷⁾ Use of the deep body thermometer permitted us to perform this study non-invasively.

Limitations: We used hot packs and supporters for warming. However, we believe our warming method was not ideal, since it was not specific to the muscle, but also warmed the skin. Use of a method which specifically warms muscle is preferable. Exercise testing was performed while the patients were on medications. Although the modification of the result by the drug effect was considered, the warming effect itself was unlikely to be influenced by the drugs. It is possible that excessive muscle warming decreases exercise capacity. Therefore, studies need to be performed to determine which degrees of warming yield the best temperature for exercising muscles.

Conclusions: The findings of this study indicated that the warming of exercising legs with hot packs improves AT and peak $\dot{V}O_2$ in patients with cardiac disease and low exercise tolerance.

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REFERENCES

1. Nielsen M. Die Regulation der Korpertemperatur bei Muskelarbeit. Skand Arch Physiol 1938; 79: 193–230.
2. Wyndham CH, Bouwer WM, Devine MG, et al. Physiological response of African laborers at various saturated air temperatures, wind velocities and rate of energy expenditure. J Appl Physiol 1953; 5: 290–8.
3. Lind AR. A physiological criterion for setting thermal environmental limits for everyday work. J Appl Physiol 1963; 18: 51–6.
4. Saltin B, Hermansen L. Esophageal, rectal and muscle temperature during exercise. J Appl Physiol 1966; 21: 1757–62.
5. Nielsen B. Thermoregulatory responses to arm work, leg work and intermittent leg work. Acta Physiol Scand 1968; 72: 25–32.
6. Saltin B, Gagge AP, Bergh U, et al. Body temperatures and sweating during exhaustive exercise. J Appl Physiol 1972; 32: 635–43.
7. Haslam RA, Parsons KC. Using computer-based models for predicting human thermal response to hot and cold environments. Ergonomics 1994; 37: 399–416.

8. Rowell LB. Cardiovascular aspects of human thermoregulation. *Circ Res* 1983; 52: 367-79.
9. Shellock FG, Rubin SA, Ellrodt AG, et al. Unusual core temperature decrease in exercising heart-failure patients. *J Appl Physiol* 1983; 54: 544-50.
10. Shellock FG, Swan HJC, Rubin SA. Muscle and femoral vein temperatures during short-term maximal exercise in heart failure. *J Appl Physiol* 1985; 58: 400-8.
11. Bergh U, Ekblom B. Physical performance and peak aerobic power at different body temperatures. *J Appl Physiol* 1979; 46: 885-9.
12. Wasserman K. The anaerobic threshold measurements to evaluate exercise performance. *Am Rev Respir Dis* 1984; 129 (S): S35-S40.
13. Hansen JE, Sue DY, Oren A, et al. Relation of oxygen uptake to work rate in normal men and men with circulatory disorders. *Am J Cardiol* 1987; 59: 669-74.
14. Zelis R, Mason DT, Braunwald E. Partition of blood flow to the cutaneous and muscular beds of the forearm at rest and during leg exercise in normal subjects and in heart failure. *Circ Res* 1969; 24: 799-806.
15. Itoh H, Koike A, Taniguchi K, et al. Severity and pathophysiology of heart failure on basis of anaerobic threshold (AT) and related parameters. *Jpn Circ J* 1989; 53: 146-54.
16. Fox RH, Solman AJ. A new technique for the deep body temperature in man from the intact skin surface. *J Physiol (London)* 1971; 212: 8-10.
17. Togawa T, Nemoto T, Yamazaki T. A modified internal temperature measurement device. *Med Biol Engng* 1976; 14: 361-4.