EXERCISE-INDUCED HEAT STRESS:
PASSIVE RECOVERY OR SUBSEQUENT TREATMENT

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INTRODUCTION
Continuous exposure to high environmental temperatures makes an individual physiologically and psychologically prone to heat stress injury. [6] Obviously prevention is preferable to treatment. The key to the prevention and treatment of heat stress is to provide individuals with adequate fluids and a periodic exposure to a cool environment. [2,5] Nevertheless, heat stress does occur and treatment is needed. In the past, the cornerstone of treatment has been to rapidly cool the victim (e.g. using ice packs) and to administer fluids. [6] Unfortunately, ice packs or cold water baths are often impractical and time consuming means to treat heat stress in the field or other emergency situations. The present study was performed to gain new insights into the occurrence of exercise-induced heat stress and its subsequent treatment: i.e., passive recovery at room temperature was compared to active cooling by ice packs or convective air.

METHODS
These studies were approved by the University of Minnesota Committee on Human Research and written consent from all volunteers was obtained. Subjects were healthy males not taking any medication and were without any health problems. All subjects had a pre-study medical evaluation, including body weight (nude), skinfold measurements, assessment of physical activity habits, and symptom-limited treadmill exercise testing using the Bruce Protocol. [1] No subject had a rhythm or ST disturbance and all had an above average exercise tolerance (>12 minutes duration; average MET score 16 ± 1.4) (mean ± S.D.). The subjects were directed to arrive postabsorptive (at least 2 hours), but in a fully hydrated state. Subjects were weighed before and after each trial to determine water loss due to sweating. [7] The first exercise period was performed at a dry bulb temperature of 27±3°C and during this session treadmill rates were established (average power was 753 ± 110 Watts). The subsequent exercise trials were at 48±2°C. Each subject exercised for 40 min or until their rectal temperature increased by 2°C. During recovery the subjects randomly either: 1) passively recovered in a cool environment (23±1°C), 2) were actively cooled using ice packs (behind neck, in each axilla, and over the femoral arteries; ~2 kg for each location), or 3) were actively cooled by a thermoelectric device that delivered cool air (14±1°C at 70±8% relative humidity) at a flow rate of 40 cfm (air velocity meter, Omega Engineering, Inc., Stamford, CT) to a full body air-distribution blanket (prototype, Augustine Medical, Inc.). Throughout the experiments (exercise and recovery periods) heart rates (a continuous record using 3 lead surface electrodes; Siemens), blood pressures (every 10 min using an automated blood pressure cuff; Siemens), rectal temperatures (YSI), tympanic temperatures (Mon-a-therm), and skin temperatures (Mon-a-therm) from the cheek, chest, forearm, index finger, abdomen and thigh were monitored (all temperatures were recorded every min).

RESULTS
The exercise at 48°C, relative to 27°C, caused significant increases in rectal temperature (~2°C), heart rates and mean body surface temperatures, whereas mean arterial pressures were significantly lower in the hot environment (p<0.05). The ice pack treatment was more rapid in returning rectal temperatures to baseline values, but caused a persistent elevation in blood pressure. However, there was no difference in the initial rates of cooling (i.e., first 25 min after therapy initiation) between the recovery periods (regression analysis), but a delay in onset of cooling was evident (Figure 1). The shift is exemplified in the time it took for rectal temperature to drop 0.5 and 1.0°C; 10.8±5.1 and 21.6±9.1 min for ice, 13.8±5.6 and 26.2±11.4 min for convective, and 18.3±10.4 and 34.8±18.4 min for the cool environment, respectively. Convective cooling was reported by these subjects to be the therapy with the greatest comfort and caused a significant decrease in the mean surface temperature exceeding that for ice therapy.
CONCLUSION

The seriousness of overexposure to heat while exercising is characterized not only by a decrease in work performance, but also by a predisposition to heat illness. Such disorders can be categorized in ascending order of severity as: heat cramps, heat syncope, heat exhaustion and heat stroke. Normally persons will involuntarily stop working well before they reach heat exhaustion, but competitive athletes or military personnel may push themselves beyond this limit. The level of exercise-induced heat stress observed in the present study were well below those to induce any of these disorders. Nevertheless, the exercise in the hot environment relative to that at a moderate temperature not only induced a larger increase in core temperature, but significantly increased the cardiovascular workload and mean body surface temperature. The cardiovascular responses to the added stress of exercise in a hot versus moderate environment was significant and thus, needs consideration in individuals suffering from heat stress.

The body responds to hyperthermia via effector mechanisms that alter heat loss and gain from the environment that alter heat loss or gain from the environment. The major modes of heat loss are via radiation, conduction, convection or evaporation. The primary mode of heat loss was likely different in each recovery period: i.e., evaporative heat loss was predominant during the passive cooling; conduction (transfer of thermal energy by direct contact) was more important with the ice therapy; and convection both at the surface (air next to the body is heated, moves away and is replaced by cool air) and in-body circulatory (during this therapy vasodilatation was present and mean surface temperature was lowest) was the primary mode with the air-cooling. The utility of convective-air cooling to treat exercise-induced heat stress, relative to ice pack therapy, is promising and warrants further investigation.

REFERENCES


