Efficacy of forced-air and inhalation rewarming by using a human model for severe hypothermia

M. S. L. GOHEEN,1 M. B. DUCHARME,2 G. P. KENNY,1 C. E. JOHNSTON,1 JOHN FRIM,2 GERALD K. BRISTOW,3 AND GORDON G. GIESBRECHT1

1Laboratory for Exercise and Environmental Medicine, Health, Leisure, and Human Performance Research Institute, and Department of Anesthesia, Faculty of Medicine, University of Manitoba, Manitoba R3T 2N2; and 2Defense and Civil Institute of Environmental Medicine, North York, Ontario, Canada, M3M 3B9

Goheen, M. S. L., M. B. Ducharme, G. P. Kenny, C. E. Johnston, John Frim, Gerald K. Bristow, and Gordon G. Giesbrecht. Efficacy of forced-air and inhalation rewarming by using a human model for severe hypothermia. J. Appl. Physiol. 83(5): 1635–1640, 1997.—We recently developed a nonshivering human model for severe hypothermia by using meperidine to inhibit shivering in mildly hypothermic subjects. This thermal model was used to evaluate warming techniques. On three occasions, eight subjects were immersed for ∼25 min in 9°C water. Meperidine (1.5 mg/kg) was injected before the subjects exited the water. Subjects were then removed, insulated, and rewarmed in an ambient temperature of −20°C with either 1) spontaneous rewarming (control), 2) inhalation rewarming with saturated air at ∼43°C, or 3) forced-air warming. Additional meperidine (to a maximum cumulative dose of 2.5 mg/kg) was given to maintain shivering inhibition. The core temperature afterdrop was 30–40% less during forced-air warming (0.9°C) than during control (1.4°C) and inhalation rewarming (1.2°C) (P < 0.05). Rewarming rate was 6- to 10-fold greater during forced-air warming (2.40°C/h) than during control (0.41°C/h) and inhalation rewarming (0.23°C/h) (P < 0.05). In nonshivering hypothermic subjects, forced-air warming provided a rewarming advantage, but inhalation rewarming did not.

It is generally agreed that treatment of the hypothermic victim should minimize the postexposure afterdrop in core temperature (Tco) and promote a steady, continuous rate of rewarming to a level where physiological homeostasis can be maintained. To date, laboratory research on field treatment for hypothermia has been limited to mild hypothermic conditions (i.e., Tco > 33.0°C) in which subjects shiver vigorously. Because skin warming inhibits shivering thermogenesis, external application of moderate sources of exogenous heat provides little rewarming advantage if vigorous shivering is present (3, 5). External warming will only provide a benefit to shivering patients if the amount of heat donated exceeds the amount of shivering heat production that is inhibited. Warm-water immersion is one method of donating a high amount of heat and promoting a rapid rate of rewarming (24), but it may also increase the afterdrop in Tco (11). Forced-air warming has also been used to donate a considerable amount of heat (∼200 W). Although the rewarming rate was not increased, Tco afterdrop was decreased by 30% (8). Whereas warm-water immersion is impractical in the field, forced-air warming systems could be adapted for field use.

Inhalation of warm, humidified air or O2 has also been used extensively as a noninvasive internal application of exogenous heat (17). Comparative studies have shown little (12, 23, 24) or no (3, 4) rewarming benefit over shivering thermogenesis alone in mildly hypothermic subjects. This is not surprising given the limited calculated heat transfer capabilities of this method (29), the probable inhibitory effect of inhalation rewarming on shivering, and the small temperature differences between inspired gas and the body core of only 5–10°C at relatively high Tco values (i.e., >33°C).

Despite the equivocal results in shivering subjects, exogenous heat is likely beneficial for victims of severe hypothermia (i.e., Tco < 30°C) in whom shivering is impaired or absent (1). For obvious ethical reasons, it is impossible to lower human Tco values to below 30°C, where the shivering response is naturally suppressed. However, we have recently developed a human model for severe hypothermia in which meperidine (1.5 mg/kg iv) was administered to inhibit shivering in mildly hypothermic subjects (7). During rewarming under these nonshivering conditions, the afterdrop in esophageal temperature (Tco) reached values as great as 2.5°C compared with 0.4°C with shivering intact. Without the endogenous heat production of shivering, Tco remained at or near nadir values for up to 150 min postimmersion.

This new protocol was used to evaluate Tco during recovery in fieldlike conditions [ambient temperature (Ta) = −20°C] during 1) spontaneous rewarming with no exogenous heat donation (control), 2) inhalation rewarming with saturated air heated to −43°C, and 3) forced-air warming with a newly developed system. We hypothesized that, compared with spontaneous nonshivering conditions, forced-air warming would decrease the afterdrop and enhance rewarming, whereas inhalation rewarming would provide a smaller but significant rewarming advantage.

METHODS

With approval from our Faculty Human Ethics Committee, eight healthy subjects (2 women, 6 men) were studied after giving informed consent. The subjects were without allergy history or adverse reactions to or chronic use of narcotics. The eight subjects were 30 ± 5 (SD) yr old, had a mass of 74 ± 7 kg, were 177 ± 8 cm tall, had a sum of 4 skinfolds (biceps, triceps, subscapular, and suprailiac) of 54 ± 10 mm, and had 18 ± 6% body fat.

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Subjects were studied on three occasions. During cold-water immersion they received injections of meperidine to inhibit shivering thermogenesis. They then exited the water and lay in an insulated enclosure (see Warming systems) for potential rewarming by means of endogenous heat production only, inhalation rewarming, or forced-air warming.

Instrumentation. \( T_{aw} \) was measured by an esophageal thermocouple positioned at the level of the heart (20). Aural canal \( T_{a} \) was measured by a flexible cotton-covered thermocouple probe placed adjacent to the tympanic membrane. The probe was then secured in place by occluding the aural canal with cotton and placing tape over the external ear surface. Rectal temperature \( T_{r} \) was monitored with a rectal thermocouple inserted to a depth of 15 cm past the anal sphincter. Single-channel electrocardiogram and heart rate (HR) were monitored continuously, and blood pressure was monitored at 5-min intervals with an automated blood pressure cuff (Dinamap 845 XT, Critikon). An intravenous line was introduced into a right arm or hand vein for drug and/or saline administration.

Oxygen consumption \( (\dot{V}O_2) \) and cutaneous heat flux and temperatures were measured according to methods described previously (8, 9). \( \dot{V}O_2 \) was determined by an open-circuit method from measurements of expired minute ventilation \( (V_{E}) \) and inspired and mixed expired gas concentrations sampled from a 10-liter fluted mixing box. Subjects wore a snugly fitting face mask with a one-way valve that was connected to the appropriate instrumentation by a suitable length of corrugated plastic tubing. The corrugated tubing that was exposed to \( -20^\circ \text{C} \) air was insulated to prevent condensation within the tubing. A thermocouple was attached to the inflow side of the one-way valve on the face mask to measure inspired air temperature. During the inhalation rewarming condition, the entire one-way valve was insulated to maximize temperature of the inspirate.

Cutaneous heat flux and temperature were measured from 12 sites by thermal flux transducers (Concept Engineering, Old Saybrook, CT). Flux was defined as positive when heat traversed the skin toward the environment (6). Body surface area \( (BSA) \) was calculated \[ BSA \ (m^2) = \frac{\text{weight}^{0.425} \times \text{height}^{0.725} \times \text{cm}}{0.007194} \] and the following regional percents were assigned based on those of Layton et al. (15): forehead, 7% upper chest, 8.8% abdomen, 8.7% scalp, 8.8% lower back, 8.7% anterior thigh, 9.5% posterior thigh, 9.5% shin, 6.5% calf, 6.5% dorsum of the foot, 7% dorsum of the hand 5.0% and arm, 14%. Flux values from each transducer \( (W/m^2) \) were then converted into watts per region \[ \text{flux at region (W)} = \text{transducer flux (W/m²) \times BSA (m²) \times regional percentage \times 0.01} \]. Temperature was also measured on the plantar surface of the left great toe and the ventral surface of the third right fingertip to monitor calf-to-toe and forearm-to-finger skin temperature \( (T_{sk}) \) gradients for indications of peripheral vasodilation during rewarming. Rapid rises in finger temperature and decreases in forearm-to-finger temperature gradients below 4°C have been used to signify cutaneous vasodilation (25). At 30-s intervals, thermal, HR, and metabolic data were averaged for the preceding 30-s period, displayed graphically on the computer screen, and recorded in spreadsheet format on a hard disk.

Warming systems. The new forced-air warming system consisted of a mobile insulated wooden warming box (1.6 m long × 0.725 m wide × 0.33 m high) that supported a nylon webbed stretcher (2.14 m long). With the subject lying on the stretcher, a wire frame (curved side to side) was placed over the subject. The rostral portion of the box was hollow and contained two 1,200-W electric heaters and six circulating fans below the webbed stretcher. Two more 1,200-W heaters and three fans were contained in an enclosed section on top of the wire frame just above the subject’s chest. A down sleeping bag (13 clo) was then placed over the wire frame and attached securely to the wooden box via Velcro strips. The head of the subject (which was exterior to the insulated enclosure) was covered with a down hood. The enclosure allowed exposure to air of all anterior and posterior skin surfaces from mid thorax to the shoulders. Thermocouples were used to measure \( T_{aw} \) values above and below the torso. During forced-air warming, the circulating fans were activated and the heaters were thermostatically controlled to maintain \( T_{aw} \) within the enclosure at \(-46–48^\circ \text{C}\) with a maximum local \( T_{aw} \) of \( 43^\circ \text{C} \). For consistency, subjects were placed in the warming enclosure for all trials, with the fans and heaters turned off during the control and inhalation rewarming trials.

In the inhalation rewarming trials, a Res-Q-Air inhalate delivery system (model HT 1000, CF Electronics, Comack, NY) was used. A 12-V marine battery provides power to heat water in the system reservoir. Inspired air is first drawn through the water where it is heated and saturated, thus providing humidified inspirate at the mouth at \(-34^\circ \text{C} \). Protocol. Each subject was cooled on three occasions separated by at least 3 days. Trials were conducted at the same time of day. Subjects were instructed to abstain from alcohol and medications for a period of 24 h before each study. They were also asked to fast for 8 h before coming to the laboratory to minimize any potential nausea caused by meperidine infusion. The subjects, who were dressed in swimsuits, were instrumented and sat quietly, covered by a blanket, at a \( T_{aw} \) of \(-22^\circ \text{C} \) for 10 min of baseline data collection. Before immersion they were dressed in a thin plastic body suit. Removal of this suit after immersion allowed a rapid transition to the rewarming phase and ensured that the subject was completely dry after immersion (minimizing evaporative heat loss during rewarming without the time consuming, and mechanically stimulating, process of towel drying).

Subjects were then immersed to the shoulders in a stirred water bath in which the water temperature was lowered from 20 to 9°C within 10 min by the addition of ice. For comparative purposes, the immersion time and removal \( T_{aw} \) for each subject were kept similar for all trials. Ten minutes before the end of immersion the subjects were given 1.5 mg/kg of meperidine IV (diluted in 10 ml of saline) injected in five 2-ml aliquots in successive 2-min intervals. They were then hoisted out of the water, and the body suit was removed. Subjects were fitted with an insulated hood, and a pulse oximeter was placed on the middle finger to monitor arterial oxygen saturation as an indication of adequate oxygenation. During the forced-air condition, insulated mitts (up to the level of the elbow) and boots (up to the level of the knee) were placed over the distal limbs to minimize peripheral warming and consequent vasodilation, which could exaggerate afterdrop through redistribution of heat from the core to the periphery. For consistency, this procedure was also followed in the control and inhalation rewarming protocols.

Subjects were then placed supine in the rewarming enclosure. An airtight neck seal was accomplished by insulating the subject’s head and neck area. The rewarming enclosure was then rolled into an environmental chamber where \( T_{aw} = -20^\circ \text{C} \). No exogenous heat source was used in the control trials. In the inhalation rewarming trials, the Res-Q-Air inhalate delivery system was placed in the rewarming enclosure and connected to the input valve of the face mask, providing humidified inspirate at \(-43^\circ \text{C} \). During the forced-air warming trials, the heaters and circulating fans were activated just before the rewarming enclosure was transported into the environmental chamber. Treatment continued...
until either $T_{es}$ increased to 36.8°C or 150 min had elapsed. In the latter situation, subjects were then immersed in 40°C water until $T_{es}$ increased to a normothermic level.

In our previous study (7) subjects were cooled for an average of 52 min to an average $T_{es}$ of 35.9°C. This cold stimulus was sufficient to cause a slight disinhibition of shivering during rewarming. To eliminate this effect, we decided to shorten immersion times and provide supplemental injections of meperidine (to a cumulative maximum dose of 2.5 mg/kg) as required to maintain shivering suppression and reverse any increases in $V\dot{O}_2$. Therefore, spontaneous rewarming (i.e., control) trials were performed first in all subjects. In six of the eight subjects, shivering resumed during rewarming, and the immersion time and/or meperidine dose were adjusted for a second control trial. In the other two subjects, the immersion time and meperidine dose in the initial trial were sufficient to completely eliminate shivering throughout rewarming. Inhalation rewarming and forced-air warming trials then followed control trials in a balanced design, with the immersion time and supplemental meperidine dosing schedule for each subject following that established during their control trials.

Data analysis. All data were averaged for the baseline period (10 min) and for subsequent 5-min intervals during immersion and postimmersion. The following variables were calculated for each trial: the afterdrop (difference between $T_{es}$ on exit from cold water and its nadir), the length of the afterdrop period (time between exit from cold water until $T_{es}$ returned to original exit $T_{es}$), the rate of rewarming (calculated by linear regression for $T_{es}$ data during the linear increase after the $T_{es}$ nadir), total net cutaneous heat transfer, and area-weighted mean $T_{sk}$ ($T_{sk,avg}$). $V\dot{O}_2$ was used as an indicator of metabolic heat production, and values greater than baseline were attributed to shivering thermogenesis (2). Data for the three trials were compared by using an analysis of variance for repeated measures with Tukey’s post hoc test used to identify significant differences. Results are reported as means ± SD. $P < 0.05$ identified statistically significant differences.

RESULTS

$T_{co}$ responses. Subjects were immersed for an average of 26 min. After immersion, $T_{es}$ continued to drop in all three conditions during the preparatory phase for rewarming (i.e., suit removal and transfer to rewarming enclosure) (Fig. 1). During forced-air warming, the afterdrop was attenuated and the rewarming rate was greater compared with the other two conditions ($P < 0.05$; Table 1). We have previously calculated the length of the afterdrop period as the time between exit from cold water until $T_{es}$ returned to original exit $T_{es}$ (5). Even after 2.5 h of control and inhalation rewarming, average $T_{es}$ remained at or near nadir levels and in no case returned to the exit $T_{es}$ values. For a comparison between all trials, time to nadir (the time from water exit to the nadir in $T_{es}$) was calculated and was significantly shorter in the forced-air warming condition ($P < 0.05$).

The three $T_{co}$ indexes followed similar patterns and were not significantly different from each other. $T_{re}$ responded to body heat content changes more slowly than did the other two indexes, being higher during cooling and lower during rewarming after the afterdrop period. $T_{es}$ readings were not affected by warm inspired air during rewarming. During this protocol $T_{es}$ actually decreased to a lower nadir value than did $T_{co}$ by 0.4°C.

Metabolic and cardiorespiratory responses. $V\dot{O}_2$ increased during cooling until meperidine infusion (Fig. 2). Postimmersion in both the control and forced-air warming conditions, $V\dot{O}_2$ continued to drop to or below baseline levels and remained at these low levels for the remainder of the trials. During inhalation rewarming, $V\dot{O}_2$ was significantly higher at 30 min postimmersion ($P < 0.05$). $V\dot{E}$ was similar in all conditions, increasing from ~13 l/min during baseline to ~22 l/min before meperidine injection and then decreasing to ~13 l/min just before the subjects exited the cold water and to ~9 l/min postimmersion. HR increased from baseline values of ~74 beats/min to a maximum of ~81 beats/min just before meperidine infusion. Postimmersion HR declined and stabilized below baseline values, being significantly greater during rewarming.

### Table 1. Rewarming parameters

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Inhalation Rewarming</th>
<th>Forced-Air Warming</th>
</tr>
</thead>
<tbody>
<tr>
<td>Afterdrop, °C</td>
<td>1.40 ± 0.2</td>
<td>1.22 ± 0.5</td>
<td>0.85 ± 0.4*</td>
</tr>
<tr>
<td>Time to nadir, min</td>
<td>77.9 ± 32.6</td>
<td>75.8 ± 27.9</td>
<td>13.1 ± 5.8*</td>
</tr>
<tr>
<td>Rate of rewarming, °C/h</td>
<td>0.41 ± 0.4</td>
<td>0.23 ± 0.2</td>
<td>2.40 ± 1.0*</td>
</tr>
<tr>
<td>Enclosure $T_a$, °C</td>
<td>22.6 ± 0.8</td>
<td>24.0 ± 2.2</td>
<td>47.5 ± 1.0*</td>
</tr>
<tr>
<td>Above torso</td>
<td>21.5 ± 1.4</td>
<td>22.1 ± 1.0</td>
<td>47.7 ± 1.4*</td>
</tr>
<tr>
<td>Below torso</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$T_{sk,avg}$, °C</td>
<td>27.0 ± 3.7</td>
<td>28.3 ± 3.9</td>
<td>35.5 ± 1.0*</td>
</tr>
<tr>
<td>5 min postcooling</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$T_{cal-tor}$, °C</td>
<td>8.9 ± 1</td>
<td>9.8 ± 2</td>
<td>8.9 ± 4</td>
</tr>
<tr>
<td>30 min postcooling</td>
<td>14.5 ± 2</td>
<td>15.3 ± 4</td>
<td>9.1 ± 6</td>
</tr>
<tr>
<td>End of rewarming</td>
<td>10.0 ± 2</td>
<td>11.2 ± 1</td>
<td>9.0 ± 6</td>
</tr>
<tr>
<td>$T_{arm-finger}$, °C</td>
<td>6.1 ± 2</td>
<td>7.6 ± 2</td>
<td>4.1 ± 4</td>
</tr>
</tbody>
</table>

Values are means ± SD. $T_a$, ambient temperature; $T_{sk,avg}$, area-weighted mean skin temperature; $T_{cal-tor}$, calf-to-tor skin temperature; $T_{arm-finger}$, arm-to-finger skin temperature. *Significantly different from control and inhalation rewarming, $P < 0.05$. 

Fig. 1. Average esophageal temperature response for control, inhalation rewarming, and forced-air rewarming trials. For clarity, SD bars are plotted for only 2 conditions at a time. In the forced-air condition, all subjects were warmed to an esophageal temperature of 36.8°C. Final value plotted for this condition is lower because this is the last point at which all 8 subjects were still warming. *Significantly greater than control and inhalation rewarming, $P < 0.05$. 


During baseline, all subjects were vasoconstricted in the lower extremities, and six were vasoconstricted in the upper extremities. After 30 min postimmersion, extremities were vasoconstricted in all subjects during the control and inhalation rewarming trials (Table 1). During forced-air warming, peripheral vasodilation occurred after 30 min in the fingers of three subjects and by the end of warming in six of the subjects. Only one subject showed evidence of vasodilation in the toes.

**DISCUSSION**

This study is the first to pharmacologically inhibit shivering and create a human model for severe hypothermia to evaluate relative efficacy of emergency field rewarming techniques. Inhalation rewarming did not significantly lower the magnitude of afterdrop, shorten the rewarming period, or improve the rate of rewarming compared with spontaneous rewarming. Forced-air warming with a high source of heat (up to 270 W) reduced the afterdrop by 30–40% and resulted in a 6- to 10-fold increase in rewarming rate.

During inhalation rewarming, our nonshivering subjects rewarmed at slightly lower rates than those reported by Lloyd (16) (0.5°C/h) in unconscious victims, with Tco values below 30°C, who were probably not shivering. The slightly higher rewarming rates in the report by Lloyd may be due to the higher inspired air temperatures used (50–80°C) vs. 43°C in the present study. In comparison, studies employing inhalation rewarming in shivering subjects report much higher rates of rewarming of between 0.8 and 1.4°C/h (10, 12, 18, 22–24, 28). This is consistent with data demonstrating that, with shivering intact, both metabolic heat production and rewarming rates increase as initial Tco and Tsk decrease (22).

Our lack of difference between rewarming rates for control and inhalation rewarming trials is consistent with other studies in which inhalation rewarming rates were between 63 and 133% of spontaneous (shivering-intact) rewarming rates (3, 10, 18, 24). Although Hayward et al (11) reported an 80% increase in rewarming rate with inhalation rewarming, only one subject was studied, and these results may not be generalizable. In the present study, rewarming was conducted at a Ta of 20°C to maximize the difference in inspired air temperature between control (−20°C) and inhalation rewarming (43°C) trials. Two other studies have conducted rewarming at a Ta of −20°C with shivering subjects at average Tco values of 35.4°C (19) and 33.9°C (4). In both cases, spontaneous and inhalation rewarming rates were similar.

The proposed advantages of inhalation rewarming are a decrease in respiratory heat loss and an increase in heat donation through the respiratory tract. Some authors have calculated that inhalation rewarming can improve heat balance by only 23 kcal/h (26) or 17.1 kcal/h (29), of which only 6.0 kcal/h are actually due to heat donation itself with the rest due to prevention of heat loss (29). These are relatively small values considering they are only 16–22% of resting metabolic heat production observed during our inhalation rewarming.
trials and that humans can increase heat production by a factor of 5 during vigorous shivering. Also, for every 1 kJ of respiratory heat added via inhalation rewarming, shivering metabolic heat production has been shown to decrease by 1.4 kJ (21) and 1.95 kJ (24). In contrast, when shivering was suppressed in our hypothermic subjects, metabolic heat production was slightly increased during inhalation rewarming. This increase may be at least partly explained by added work of breathing introduced by the apparatus itself.

Hayward and Steinman (12) have suggested other benefits of inhalation rewarming, including rehydration, stimulation of respiratory mucociliary activity, and direct heat transfer from the upper airways to the hypothalamus, brain stem, and other brain structures. Warming of the respiratory and cardiovascular centers could help stabilize cardiorespiratory parameters even if total body heat content was not increased significantly. Also, central thermoregulatory control may be reinstated in a severely hypothermic victim. There are numerous anecdotal reports where application of inhalation rewarming significantly improved the hypothermic patient’s pulse rate and mental state within 20–40 min (R. Douwens, personal communication). This is consistent with inhalation therapy warming the brain stem and/or other brain structures without significantly increasing core body heat content.

It may be difficult to demonstrate a thermal advantage with inhalation rewarming in the present laboratory study because of inherent limitations. Clinically, a moderately to severely hypothermic patient would have depressed ventilation, thus minimizing respiratory heat transfer. Also, the airway warming system used in this study may not represent all inhalation therapy systems because other devices may deliver hotter air with greater saturation (i.e., slightly cooled steam).

Forced-air warming systems have generally been used for prevention or reversal of hypothermia in surgical patients (14), but the potential value for prehospital stabilization for accidental hypothermia has also been suggested (8, 27). We have previously demonstrated that a conventional forced-air warming system (Bair Hugger, Augustine Medical) transfers a moderate amount of heat (≈100 W) to the body surface in normothermic subjects (6) and a greater amount of heat transfer in hypothermic subjects who had much lower Tsk values (237 W initially, falling to 163 W after 30 min) (8).

We expected that the newly designed forced-air warming system would transfer even more heat to our subjects. Although total heat transfer to the body was only moderately greater, the new system redistributed heat away from the extremities toward the torso, including the back. As expected, afterdrop was decreased and rewarming rate was increased significantly. The decreased afterdrop was likely a result of decreased thermal gradients for conductive heat loss through direct warming of peripheral tissue. This protective effect may prevent a victim’s Tco from dropping below the threshold for cold-induced ventricular fibrillation (1). The high amount of heat transfer during forced-air warming (average ≥200 W) facilitated more rapid rewarming than did spontaneous rewarming. In severely hypothermic nonshivering victims, endogenous heat production is likely below 50 W at a Tco of 27°C (1). Therefore, forced-air warming could be beneficial for severe hypothermia during transport.

The main safety concern with forced-air warming is burn prevention. At the end of 30 min of forced-air warming Tskavg was only 35.5°C, and local Tsk did not exceed 43°C in any area. Under these conditions the risk of burns is likely minimal (13).

To maintain shivering suppression throughout the rewarming periods, the decrease in Tco was necessarily small. Despite the relatively high Tes, peripheral tissues were cooled sufficiently to produce temperature gradients resulting in larger afterdrops (0.9–1.4°C) than reported in other studies with shivering subjects at lower Tco values (3, 5). The comparative results in this study are qualitatively valid, although the absolute changes would likely be even greater at lower Tco values.

In summary, Tco did not increase significantly in nonshivering, mildly hypothermic subjects even after 150 min of spontaneous or inhalation rewarming. However, external heat delivery with forced-air warming decreased Tco afterdrop by 30–40% and increased the rewarming rate by 6- to 10-fold. These data indicate that exogenous heat is likely beneficial to a severely hypothermic nonshivering patient. A portable forced-air warming system holds considerable promise for field use because of the rapid reversal of core cooling, the high rate of rewarming, and overall comfort provided to the victim. Finally, our new human model for severe hypothermia will allow the study of other rewarming methods, without the complicating factor of shivering thermogenesis.

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Address for reprint requests: G. G. Giesbrecht, Univ. of Manitoba, Faculty of Physical Education and Recreation Studies, 102 Frank Kennedy Bldg., Winnipeg, MB, Canada R3T 2N2 (E-mail: Giesbre@cc.umanitoba.ca).

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