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**Increased post-immersion afterdrop following B-adrenergic blockade**

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Abstract
It is well established that, during the initial rewarming from mild hypothermia, core temperature continues to decrease before returning towards its pre-immersion state (1). This phenomenon is known as the afterdrop, and has been ascribed to circulatory changes at the periphery, as well as to continued core-to-periphery thermal conduction, both of which may account for continued central-body heat loss after removal from the cold (2,3,4). In a recent series of experiments, in which we studied interactions between cold-water immersion, B-adrenergic blockade, plasma leptin concentration, rewarming and skin blood flow control, we also investigated the afterdrop. Our observations have revealed that altered post-immersion metabolism can also contribute to the afterdrop phenomenon.

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INCREASED POST-IMMERSION AFTERRDROP FOLLOWING β-ADRENERGIC BLOCKADE


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INTRODUCTION

It is well established that, during the initial rewarming from mild hypothermia, core temperature continues to decrease before returning towards its pre-immersion state (1). This phenomenon is known as the afterdrop, and has been ascribed to circulatory changes at the periphery, as well as to continued core-to-periphery thermal conduction, both of which may account for continued central-body heat loss after removal from the cold (2, 3, 4).

In a recent series of experiments, in which we studied interactions between cold-water immersion, β-adrenergic blockade, plasma leptin concentration, rewarming and skin blood flow control, we also investigated the afterdrop. Our observations have revealed that altered post-immersion metabolism can also contribute to the afterdrop phenomenon.

METHODS

Ten healthy (5 female and 5 male) subjects were immersed in 18°C water (60 min), and subsequently rewarmed in air at 40°C (60 min) with, and without, β-adrenergic blockade (propranolol). Subjects presented at 0800 h, after an overnight fast, for blood sampling and anthropometric measurements, then consumed a standard breakfast (38 kJ·kg⁻¹ plus 5 ml·kg⁻¹ fluid). Subjects were instrumented at 1100 h, rested for 30 min, and were immersed to the fourth intercostal space. Each immersion test commenced between 1200-1230 h. Propranolol (Deralin 40: Alphapharm Pty. Ltd, Australia) was administered orally 60 min prior to taking baseline measurements.

Rectal temperature was recorded using a thermistor, inserted 12 cm beyond the anal sphincter (YSI type-401, Yellow Springs Instrument Co. Inc., Yellow Springs, OH, U.S.A.). Skin surface temperatures were measured at eight sites (EU type, Yellow Springs Instruments Co. Inc., Yellow Springs, OH, U.S.A.): forehead, right scapula, left upper chest, right arm, left forearm, left hand, right anterior thigh and left calf. Mean skin temperatures were derived as an area-weighted summation. Temperatures were sampled at 15-s intervals (1206 Series Squirrel, Grant Instruments Ltd., Cambridge, U.K.). Cardiac frequency was recorded from ventricular depolarisation (Model PE3000, Polar Electro Sport Tester, Kempele, Finland), and also sampled at 15-s intervals.

Skin blood flow was determined using laser-Doppler velocimetry (thigh: Vasamedics Inc., TSI Laserflo BPM, U.S.A.: 780 nm wavelength), and forearm blood flow was measured using venous-occlusion plethysmography (forearm: EC4 Plethysmograph, D.E. Hokanson Inc., U.S.A.). Both sets of data were simultaneously collected under neutral-warm conditions prior to immersion, and again during the rewarmed period.

Blood samples (antecubital vein) were taken upon arrival, just prior to immersion, during immersion at 25 and 60 min, and every 10 min during rewarmed. Samples were kept on ice and centrifuged within 20 min (2000 g), with separated plasma stored at 80°C for subsequent analysis. Circulating glucose and non-esterified free fatty acid concentrations were determined.
using enzymatic, colorimetric methods (NEFA C, Wako Pure Chemical Industries Ltd., Osaka, Japan; Glucose HK, Roche Diagnostics Pty Ltd., Castle Hill, Australia) using a COBAS Mira Plus system (Roche Diagnostics Pty Ltd., Castle Hill, Australia).

RESULTS

Whilst the water temperature was not profoundly cold, the rapid immersion did induce a mild cold-shock response. Propranolol acted to suppress both the absolute and proportional cold-shock tachycardia \((P<0.05)\), as well as the pre-immersion cardiac frequency \((-10 \text{ b/min}, P<0.05)\) and cardiac frequency during immersion \((P<0.05)\). This latter change represented a parallel displacement of the cardiac response \((P>0.05)\).

Propranolol did not affect body temperatures, or their dynamic responses during immersion \((P>0.05)\). There was no effect of propranolol on serum glucose concentration \((P>0.05)\). However, circulating non-esterified fatty acid concentration was reduced \((-70\%, P<0.05)\).

Skin and forearm blood flows increased during rewarming \((P<0.05)\), but were equivalently suppressed in the propranolol condition \((-18\% \text{ and } -21\%)\), with the latter being significant \((P<0.05)\). Despite blocking the vasodilatory \(\beta_2\) receptors, the afterdrop was larger following propranolol administration \((0.68^\circ\text{C} \pm 0.09 \text{ versus } 0.46^\circ\text{C} \pm 0.06, P<0.05)\).

DISCUSSION

While the contributions of the convective (circulatory) and conductive components of the afterdrop phenomenon cannot be excluded during these trials, they do not explain the difference in afterdrop observed between the two conditions. Indeed, the significant suppression of rewarming vasodilation, implies that the rapid return of a bolus of cooler peripheral blood, while significant under some circumstances \((5)\), played a minimal role in the current experiments.

Not only was the convective component minimised following \(\beta\)-adrenergic blockade, but the afterdrop was enhanced. It must be assumed that conductive cooling between the two conditions was not markedly affected by propranolol administration, and may perhaps even have been suppressed. Therefore, we suggest that the increased afterdrop may have resulted from a reduced thermogenic response, as supported by a powerful suppression of circulating non-esterified fatty acids, during both the immersion and rewarming phases of the experiment.

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REFERENCES