## The local dynamics of thermal sweat suppression following a systemic cholinergic blockade

C.A. Machado-Moreira,<sup>1</sup> P.L. McLennan,<sup>2</sup> S. Lillioja,<sup>1</sup> W. van Dijk,<sup>1</sup> J.N. Caldwell<sup>1</sup> and N.A.S. Taylor,<sup>1</sup> School of Health Sciences, University of Wollongong, Wollongong, NSW 2522, Australia. and <sup>2</sup>Graduate School of Medicine, University of Wollongong, Wollongong, NSW 2522, Australia.

Human eccrine sweat secretion can be fully inhibited by atropine, systemically administered in the correct dose. However, the dynamics of this suppression have not been thoroughly described. Therefore, using very sensitive methods, local sweat rates were measured simultaneously across several body segments, before and after a cholinergic blockade. Herein are described the temporal characteristics of thermal sweat suppression following atropine infusion. Eight males were passively heated (feet immersion (42-43°C), water-perfusion suit (48°C)). After core temperature increased  $\sim 0.5^{\circ}$ C, and steady-state thermal sweating was established, the core temperature was clamped and atropine sulphate was gradually (over ~1 min) and intravenously infused (dorsal hand: 0.04 mg.kg<sup>-1</sup>). Sweat rates were measured at 1s intervals using ventilated capsules positioned at the forehead, the dorsal surfaces of the forearm and hand, the palm and calf. Three variables were estimated: the phase delay between the start of the infusion and the first evidence of sweat reduction; the time required for full suppression of sweating; and the time constant for this decay. The blockade completely inhibited thermal sweating from all regions (p < 0.05), and this occurred, on average, within 5 min (Table: data are means with standard deviations). Indeed, the first evidence of suppression appeared within 60s, and the mean decay time constant was 144s. The rapidity of this action was remarkable, given that the infusion was delivered intravenously at the hand, and had to diffuse from the periglandular capillary and through the interstitial fluid before it could block receptors on the sweat gland. This was facilitated by an almost immediate cardiac acceleration ( $40\pm4$  beats.min<sup>-1</sup>) and presumably by the rapid reduction in peripheral vasoconstrictor tone to support pressure regulation. It took longer for the full suppression to become established at the forehead (p<0.05), and this delay may be explained by its higher initial sweat rate. Indeed, a larger atropine dose is required to produce inhibition when sweating is more profuse (Cummings & Craig, 1967). Furthermore, it was only the total suppression time that was delayed, while neither the phase delays nor the time constants differed significantly among sites (p>0.05).

Skin site	Phase delay (min)	Time constant (min)	Full suppression (min)
Forehead (N=6)	$0.8 \pm 0.4$	2.6±1.3	$7.8 \pm 2.4^{\dagger}$
Dorsal forearm (N=6)	$0.9 \pm 0.4$	2.1 ±0.7	$3.9 \pm 0.2$
Dorsal hand	$1.2 \pm 0.5$	$2.7 \pm 0.5$	4.4 ±0.6
Palm	$0.9 \pm 0.4$	2.1 ±0.4	$3.7 \pm 0.7$
Upper calf	$1.0 \pm 0.2$	$2.4 \pm 0.4$	5.1 ±1.6
All sites (mean)	1.0 ±0.3	2.4 ±0.3	4.9 ±0.5

<sup>†</sup>significantly different from all other sites.

Cummings, E.G. & Craig, F.N. (1967). Influence of the rate of sweating on the inhibitory dose of atropine. *Journal of Applied Physiology*. **22**: 648-654.