ORTHOSTATIC INTOLERANCE DURING 63" HEAD-UP TILT FOLLOWING HOT BATH IMMERSION IS LONGER AFTER ETHANOL INGESTION.

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INTRODUCTION: The majority of hyperthermia-related deaths occur in hot tubs, spas and Jacuzzis (1). Alcohol ingestion was associated with most of these deaths (1,2). Exiting a hot bath can produce orthostatic intolerance (O1) and ethanol ingestion exacerbates this condition immediately following hot bath immersion (3). It is not known how long it takes for hyperthermia-induced O1 to subside, and if this time is influenced by ethanol ingestion. Although ethanol is a known vasodilator, its influence on sweating is not clear. As well, the effects of ethanol on esophageal temperature (T_{es}) increase during immersion and T_{es} decrease post-immersion have not been documented. To investigate the effects of ethanol on the duration of O1, sweating and rate of T_{es} change, the present study subjected volunteers to 63" head-up tilt before and after 40°C bath immersion, with or without ethanol ingestion.

METHODS: 4 men and 3 women volunteers were instrumented for T_{es} , mean skin temperature, heart rate and forehead sweat rate (E_{sw}). Blood pressures were determined manually and mean arterial pressure (MAP) calculated. On 2 occasions, subjects were immersed in a warm bath (40° C) until T_{es} increased to a maximum of 39.0°C. In the ethanol condition (ethanol), each subject ingested 1 ml ethanol per kg of body weight in 6 times the volume of orange juice. This resulted in an average blood alcohol concentration (measured by breath analysis) of (mean \pm SE) 75.6 \pm 7.0 mg %; this value was adjusted to correct T_{es} increases (4). In the other condition (control) an equal volume of juice was ingested. O1 was tested by 63" head-up tilt. Prior to immersion, subjects were given one 5 min tilt, bracketed by two 5 min periods of supine rest. Following immersion subjects were subjected to a series of at least 3 tilts (Fig. 1), starting after 5 min of supine rest, at 10 min intervals. Each tilt was scheduled to last 5 min but was terminated with the subjects verbal indications of imminent syncope and a pronounced blood pressure drop. Tilts continued until the subjects tolerated the entire 5 min period without O1; this was after the 3rd tilt in all cases in except 1 in the control condition.

RESULTS: During ethanol ingestion prior to immersion 3 of 7 subjects had a transient onset of sweating. One of these subjects is illustrated in Fig. 1 together with another subject that gave a typical response for other subjects who did not sweat during ethanol ingestion.

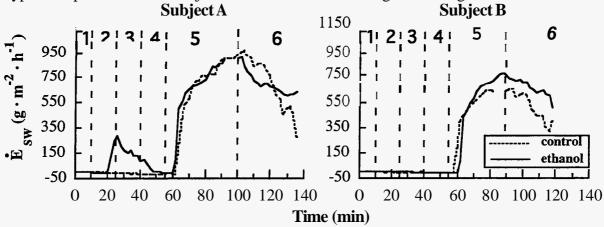


Fig. 1. E_{Sw} for subjects A and B during: rest (1), a drink of juice or juice/ethanol (2), rest after drinking (3), normothermic tilting (4), 40° C bath immersion (5), and post-immersion tilting (6). Subject A sweated during and after ethanol ingestion in the normothermic condition. During and following immersion subject B gave the typical sweating response for the group.

Rates of T_{es} increase of 4.06 ± 0.35 °C·h⁻¹ during control vs 3.61 ± 0.21 °C·h⁻¹ during ethanol conditions were not significantly different. Post-immersion T_{es} rates of decrease of 3.58 ± 0.12 °C·h⁻¹ during control and 3.52 ± 0.12 °C·h⁻¹ during ethanol conditions were also not

significantly different. Prior to immersion no OI was evident in either condition during head-up tilt. After immersion during the 1st tilt 5 of 7 subjects for the control and 4 of 7 for the ethanol conditions showed O1. For both the 2nd and 3rd control tilts 1 of 7 were intolerant, whereas for the ethanol condition 4 of 7 and 3 of 7 were intolerant. In the ethanol condition for the first 3 post immersion tilts females tolerated all tilts while males tolerated only 1 of 12 tilts. The tilt duration of 1st post-immersion tilt tolerated was 3.4 ± 0.5 min for the control and 2.9 ± 0.8 min for the ethanol condition (NS). The tilt duration endured for control vs ethanol conditions were significantly different for the 2nd (4.8± 0.2 min vs 3.4± 0.7 min (P=0.005)), and 3rd $(4.9 \pm 0.1 \text{ min vs } 3.7 \pm 0.7 \text{ min } (P=0.01))$ post-immersion tilts. Mean arterial blood pressure was significantly lower in the ethanol condition for post immersion tilts 1 and 2. Control vs ethanol MAP values were 71.8 \pm 4.5 vs 63.6 \pm 3.4 mm Hg (P=0.01) for tilt 1 and 86.7 \pm 4.4 vs 79.6 ± 2.3 mm Hg (P=0.02) for tilt 2. Tilt 3 MAP values of 90.4 ± 4.9 for the control and 87.9 ± 3.0 mm Hg the ethanol conditions were not significantly different. Fig. 2 gives the MAP of a subject who had a prolonged orthostatic intolerance during post immersion tilting.

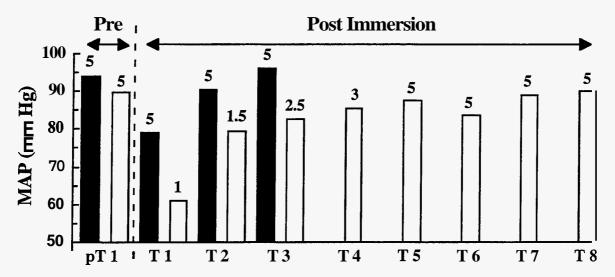


Fig. 2. MAP during pre (pT1) and post **40°C** immersion head-up tilt (TI to T8) in control (black columns) and ethanol (white columns) trials. This subject showed 01for 8 post-immersion tilts in the ethanol condition. The subject tolerated the prescribed 5 minfrom T5 to T8, but was not able stand without signs of syncope until T8 when MAP had returned to the preimmersion level. Above each column are the number of min the subject tolerated the tilt.

During post-immersion tilting, the HR for control vs ethanol conditions for tilt 1 were 116.8 ± 6.3 b·min $^{-1}$ and 116.1 \pm 12.2 b·min $^{-1}$ (NS), for tilt 2 were 91.3 \pm 2.8 and 106.4 \pm 7.4 b·min $^{-1}$ (P=0.002), and for tilt 3 were 86.4 ± 2.9 vs 100.0 ± 4.9 b·min $^{-1}$ (P=0.0007).

CONCLUSIONS: Ingesting ethanol initiated sweating in 3 of 7 normothermic subjects. This suggests that ethanol could be a non-thermal factor influencing sweating. Despite this effect of ethanol, the rates of T_{es} increase during 40°C immersion, and the rate of T_{es} decrease following immersion, were not affected by ethanol ingestion. Following immersion ethanol: 1) increased the incidence of O1 and decreased the time that a 63° head-up tilt was tolerated and 2) increased HR and decreased MAP during the head-up tilt.

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