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Passive Heat Stress Attenuates the Rise in Blood Pressure During Face Cooling

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(No relevant relationships reported)

The rise in mean arterial pressure (MAP) during the cold pressor test is attenuated by passive heat stress (HS). However, it is not known if HS attenuates the rise in MAP during face cooling (FC).
PURPOSE: Test the hypothesis that HS attenuates the rise in MAP during FC.
METHODS: FC was performed on ten healthy subjects (23±2years, 1 woman) during thermoneutral (TN) conditions and during HS induced by a water perfused suit. Subjects rested supine for 10 min while 34°C water was perfused through the suits. Then a 0°C bag of water was placed on the forehead, eyes, and cheeks for 15 min (FC). Subjects were then given 10 min to recover from FC before 50°C water was circulated through the suit until intestinal temperature (telemetry pill) was 1.0°C greater than TN. Five min of data were collected before FC was repeated. Heart rate (ECG), MAP (photoplethysmography), and forearm skin blood flow (SkBF; laser Doppler) were continuously collected. Forearm cutaneous vascular resistance (CVR) was calculated. Face pain (0 = no pain, 10 = worst pain imaginable) was obtained immediately after FC. Change from TN and HS baseline data were analyzed across 1 min intervals for the first 3 min and every 3 min thereafter during FC.
RESULTS: Baseline HR (71±14 vs. 93±17 bpm) and SkBF (22±14 PU vs. 127 ± 35 PU) were greater and MAP (88±14 vs. 81±6 mmHg) and CVR (4.9±1.8 vs. 0.7±0.3 mmHg/PU) were lower during HS (P<0.05 for all). FC caused a greater decrease in HR during HS (largest difference at 12 min of FC: -7±3 vs. -15±11bpm, P=0.02). FC caused a greater increase in MAP during TN (largest difference at 6 min of FC: 20±10 vs. 1±6 mmHg; P<0.01). FC did not change SkBF across time (P=0.31) nor was there a difference between TN and HS (P=0.61). FC caused a greater increase in CVR during TN (largest difference at 6 min of FC: 1.4±1.2 vs. 0.0 ± 0.0 mmHg/PU; P<0.01). Face pain was not different between trials (TN: 6±3 vs. HS: 4±3; P=0.06).
CONCLUSION: Passive HS attenuates the rise in MAP during FC. The impaired ability to increase CVR appears to contribute to the attenuated rise in MAP during HS with FC.

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Alcohol and Cardiovascular Health: Acute Alterations Versus Chronic Adaptations

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(No relevant relationships reported)

Alcohol abuse is a risk factor for disease but moderate use may be beneficial. Mechanisms for this contrast remain speculative. Differences may be explained by acute alterations rather than chronic adaptations.
PURPOSE: To compare cardiovascular health markers in patients with and without a history of heavy drinking, and patients who are currently intoxicated.
METHODS: Health outcomes of patients treated at a U.S. hospital were analyzed; 2,033 were sober, 273 tested positive for alcohol, and 131 reported a history of alcohol abuse. Dependent variables were systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), hemoglobin, oximetry, and disease incidence. Independent variables were age, sex, anthropometry, and use of alcohol. Independent-samples t tests and chi-square tests evaluated differences between patients with and without a history of alcohol abuse. Linear and logistic regressions tested the effects of alcohol on dependent variables.
RESULTS: Among sober patients, each year of age predicted 0.3 mmHg higher SBP (p<0.001) but no change in DBP (p=0.137). Across the total sample, current intoxication predicted 8.6 mmHg lower SBP (p<0.001), 8.7 bpm higher HR (p<0.001), and 1.0 g/dL higher hemoglobin (p<0.001). Linear regression found patients who tested positive for alcohol to have 4.6 mmHg lower SBP (p=0.002; 95% CI: -7.6 to -1.6) holding confounders constant. Among sober patients, a history of alcohol abuse associated with an elevated HR (p=0.001), lower pulse pressure (p=0.002), lower oximetry (p=0.018), and a trend for reduced SBP (p=0.056) with no difference in DBP (p=0.404). Linear regression found a history of alcohol abuse to lower pulse pressure (p=0.009) and oxygen saturation (p=0.012) and raise HR (p<0.001). Among sober patients, a history of alcohol abuse did not affect the odds of having a myocardial infarction (p=0.805), congestive heart failure (p=0.712), peripheral vascular disease (p=0.997), stroke (p=0.691), diabetes (p=0.107), or dementia (p=0.905); it did associate with a 15-fold increase in the odds of cirrhosis (p<0.001).
CONCLUSIONS: Sober patients with a history of alcohol abuse mimic the cardiovascular profile of intoxicated patients. This suggests that both short and long-term alcohol ingestion may confer modest cardiovascular benefits.

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Cardiac Structure-function And Aerobic Capacity In Individuals With A Competitive Sports History

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(No relevant relationships reported)

Athletes (A) conduct intense physical training to attain peak performance. Intense training is associated with cardiac remodeling and electrical abnormalities. Although certain ECG abnormalities are considered benign, these changes have been linked to sudden cardiac death in A.
PURPOSE: to investigate cardiac structure-function and electrocardiographic changes relative to aerobic capacity (Vo_2max).
METHODS: A cross-section of individuals consented for this university IRB approved study. The population consisted of controls (C; n=21; high school sports (n=8 women, 9 men) or active, no organized sports (n=3 women, 1 man)), college A (CA, completed career <3 years ago; n=9, 5 women, 4 men), and A (presently competing, n=22, 6 women, 16 men). Measurements included anthropometric assessment (DEXA), resting 12-lead ECG, and graded exercise test (GXT) with echocardiography/Doppler ultrasound performed before and following the GXT. An incremental treadmill GXT was conducted (6 mph) to Vo_2max with respiratory gas measurements (open-flow, indirect calorimetry).

RESULTS: CA and A had greater fat-free mass, but bone density was greater in A. While resting heart rate was lower in A and CA, blood pressure, stroke volume (A=87±19 ml; CA=83±23 ml; C=77±18 ml) and cardiac output (A=4.8±1.3 L·min^{-1}, CA=5.2±1.5 L·min^{-1}; C=4.8±1.3 L·min^{-1}) were similar among groups. Left ventricular (LV) end-diastolic dimension and posterior and septal wall thickness provided evidence of cardiac remodeling (eccentric hypertrophy-EH, concentric hypertrophy-CH, concentric remodeling-CR) in C (EH n=3, CR n=4), CA (EH n=1, CR n=2), and A (EH n=2, CH n=1, CR n=4). Overall, ECG analysis showed PVC's (n=3), LV hypertrophy voltage criteria (n=10), peaked T-waves (n=7), J-waves (n=4), U-waves (n=5), wandering pacemaker (n=4), early repolarization (n=5), short PR (n=1), Wenckebach (n=1), small Q waves (n=13). Vo_2max (C=44.7±9.8 ml·kg^{-1}·min^{-1}; CA=44.7±8.2 ml·kg^{-1}·min^{-1}, A=49.9±9.3 ml·kg^{-1}·min^{-1}) and maximal heart rate (C=187±91 b·min^{-1}; CA=194±7 b·min^{-1}, A=187±9 b·min^{-1} were not different.
CONCLUSION: Given similar, moderate levels of Vo_2max and cardiac function, presence of cardiac remodeling and ECG abnormalities among each group raises questions regarding the genesis of these changes relative to training history.

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Free-Weight Resistance Exercise Versus Weight Machines on Pulse Wave Reflection

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(No relevant relationships reported)

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