exercise training (AE+SC session C). The groups were conducted using a randomized crossover design, consisting of 60% of heart rate reserve, 60 min/day during 3-day as exercise training in healthy male smokers (23 ± 7 years, duration of smoking: 58.7 ± 10.0 months; number of cigarettes per day: 10.71 ± 0.47; mean ± SEM).

After overnight fasting, with measurement of heart rate and blood pressure, the ANS activities was assessed using power spectral analysis of HRV at resting condition and post period. Exhaled carbon monoxide(CO) to confirm smoking status and forced vital capacity(FVC) to test pulmonary function were measured before and after the session. Results: Although 3-sessions showed no affect to the FVC, exhaled CO was significantly reduced in sessions Bp (0.004) and Cp (0.006), respectively. Mean HR significantly decreased in session Ap (0.01) and session Cp (0.027), but HRV parameters did not change in session A and session B. During session C, HRV parameters showed decreased total frequency (p = 0.005) and high frequency (p = 0.12) in frequency domains and significantly increased SDNn (p = 0.035), pNN50(p = 0.03), and RMSSD (p = 0.043) in time domains, respectively. Conclusions: Short-term aerobic exercise with smoking cessation led to a significant increase in the parasympathetic nerve system over a 3-day period among smokers. Thus, in terms of HRV aerobic exercise and smoking cessation will be an effective intervention for young smokers.

1282 Board #22 May 29, 8:00 AM - 9:30 AM
TRPV1 Channels in Human Skeletal Muscle Feed Arteries: Evidence of a Potential Role ln Heat-Induced Synchronizedness
Stephen J. Ives1, Jayson R. Gifford2, Song Y. Park1, Robert H.I. Andthbacha3, Russell S. Richardson2, Skidmore College, Saratoga Springs, NY. 1University of Utah, Salt Lake City, UT. (No relationships reported)

It remains unknown, if transient receptor vanilloid type 1 (TRPV1) channels exist in skeletal muscle feed arteries of humans, what role they may play in modulating vascular function, and if activation of this heat sensitive receptor may mimic heat-induced synchronizedness. PURPOSE: To determine the existence and potential role of TRPV1 channels in the vascular function of human skeletal muscle feed arteries. METHODS: Skeletal muscle feed arteries were obtained from 10 humans (60±5 yrs) and studied using wire myography with and without TRPV1 agonism. Potassium chloride (KCl), phenylephrine (PE), acetylcholine (ACh), and sodium nitroprusside (SNP) Concentration response curves (CRCs) were performed to characterize non-receptor and receptor-mediated vasoconstriction as well as endothelium-dependent and independent vasorelaxation, respectively. To determine if the effects of TRPV1 activation were specific to adrenergic subtype, dexmedetomidine (DEX, α2-receptor agonist) CRCs were also assessed. All CRCs were performed under control and capsaicin (TRPV1 agonist) conditions. RESULTS: Capsaicin significantly attenuated vasoconstriction in response to PE (49±10 vs. 24±6 %KClmax), DEX (29±12 vs. 22±6 %KClmax), and KCl (102±14 vs. 26±8 %KClmax), but significantly enhanced vasorelaxation with ACh (76±10 vs. 112±15 %Achrelaxation) and tended to effect SNP in the same manner (86±15 vs. 103±10 %SAprxrelaxation) (control vs. capsaicin conditions). Capsaicin suppressed α2-receptor function (DEX) to a greater extent than the α1-receptor (PE). CONCLUSION: Human skeletal muscle feed arteries express functional TRPV1 channels, capable of attenuating vasoconstriction and the sympatholytic effects of exercise-induced heat, possibly in an endothelium-dependent mechanism. Supported by NIH PO1 HL-091830.

1283 Board #23 May 29, 8:00 AM - 9:30 AM
The Role of TRPV Ion Channels in Heat-induced Synchronizedness of Human Skeletal Muscle Feed Arteries
Jayson R. Gifford1, Stephen J. Ives2,Song Y. Park1, Robert H.I. Andthbacha3, Joel D. Trinity1, Russell S. Richardson2,1University of Utah, Salt Lake City, UT. 2Skidmore College, Saratoga Springs, NY. Salt Lake VAMC, Salt Lake City, UT. (No relationships reported)

Heating isolated human skeletal muscle feed arteries (SMFA) to temperatures observed during exercise results in attenuation of α1-adrenergic vasoconstriction. How this sympathetic response to heat is initiated and whether this affects α1-adrenergic vasoconstriction in human SMFA is unknown. PURPOSE: To determine if heat inhibits α1-adrenergic vasoconstriction, similarly to α2-adrenergic contraction, in SMFA and determine if the temperature-sensitive vanilloid type transient receptor potential (TRPV) ion channels mediate this effect. METHODS: SMFA from 26 subjects (53 ± 3 yrs) were studied using wire myography. α1 (phenylephrine, PE) and α2 (dexmedetomidine, DEX)-induced concentration response curves (CRC, 10^-10^/M) were assessed at 37˚C and 39˚C with and without 30μM of the TRPV inhibitor ruthenium red (RR). Endothelium function (acetylcholine, ACh) and smooth muscle function (sodium nitroprusside, SNP and potassium chloride, KCL) were also assessed with CRC’s under these conditions. Comparisons were made using repeated measures analysis of variance (p<0.05) followed by a Tukey’s post hoc test. Data represent mean maximal response ± SEM. RESULTS: At 37˚C DEX elicited a small contractile response compared to PE (3.1 ± 5 ± 12 %KClmax, respectively). Heating to 39˚C completely prevented α1-induced contractions (0.4 ± 0.5 %KClmax, p<0.05) and significantly attenuated α2-induced contractions (25 ± 6 %KClmax, p<0.05). TRPV inhibition restored α1 and α2-induced contraction at 39˚C (45 ± 8 and 4 ± 1 %KClmax, respectively). Inhibition of TRPV channels blunted ACH relaxation from 57±9 to 37±9 % relaxation (p<0.05) without affecting smooth muscle function as assessed by (KCN, SNP, p<0.05). CONCLUSION: The α1 adrenergicceptors, which were unresponsive to DEX in the heat, are more susceptible to elevated temperature than their α2 counterparts. Inhibition of the TRPV ion channels restored adrenergic contraction in the heat while blunting ACH-induced dilation. Thus, it appears that the temperature-sensitive TRPV ion channels sense the increase in temperature to initiate the sympatholytic effect of heat, possibly in an endothelium-dependent mechanism. Supported by NIH PO1 HI-091830.

1284 Board #24 May 29, 8:00 AM - 9:30 AM
3d Strain For The Left Ventricular Function Evaluation In Athletes With Bicuspid Aortic Valve.
Gianni Pedrizzetti1, Laura Stefani2, Alessio De Luca1, Loira Toncelli1, Giorgio Galanti1. 1Department of Engineering and Architecture, Florence, Italy. 2Sports Medicine Center, Florence, Italy. (No relationships reported)

Purpose : Bicuspid Aortic Valve ( BAV) is the most common congenital cardiac disease in general population as well as athletes. Left Ventricle ( LV) performance of BAV athletes, has been recently studied by the 2D echocardiographic exam and also by deformation parameters analysis. An initial impairment of the LV contractility of rotation, and torsion has been demonstrated. The study aims to verify the role of the 3D investigation approach to add more information on this aspect. Methods: A group of 20 young male asymptomatic BAV athletes with mild aortic regurgitation was matched with a tricuspid aortic valve (TAV) ones and studied by 2D and 3D echocardiographic exam to obtain the principal 2D and 3D strain parameters. Results: All the 2D echo parameters were within the normal range for both with slight differences for the diastolic function . In BAV group only the Global Secondary Strain (GSS) was significantly higher than TAV ones ( TAV : -6.6±3.0, BAV : -12.4±6.3, p=0.0029). The increase of CS and SS noticed in terms of global values, are significant at the apical level (CS: p<0.001; SS: p=0.0008) and at the median levels (CS: p=0.008; SS: p<0.001) Conclusions: Asymptomatic and trained BAV athletes, show some differences of mechanical and contraction functions with an major participation of the medio- apical segment of the LV if compared to TAV ones. Considering the normality of the LV morphology and function, an eventual involvement of the myocardial ultrasound structure can be, at present, only hypothesized. The possible protective role of exercise in preserving the normal LV function will need a longer follow up in future.

1285 Board #25 May 29, 8:00 AM - 9:30 AM
Glenn Stewart1, Akira Yamada1, Luke Haseler1, Justin Kavanagh1, Jonathan Chan2, Surendran Sabapathy1. 1Griffith University, Gold Coast, Australia. 2The Prince Charles Hospital, Brisbane, Australia. (No relationships reported)

Transient reductions in myocardial strain - assessed in a supine recovery state - have been reported following prolonged (>180 min) high-intensity exercise; however, it is unknown if shorter duration exercise can impair cardiac function. Furthermore, it is unclear if reductions in myocardial strain following exercise are masked by persistent cardiac sympathovasoreactivity and altered loading conditions during supine recovery.

Purpose: This study compared strain before and after 60 min of high-intensity cycling under resting and low-intensity exercise (normalised loading) conditions.
Methods: Left (LV) and right (RV) ventricular global longitudinal strain (GLS), and LV segmental longitudinal, circumferential and radial strain were assessed in 13 competitive cyclists (age: 28 ± 5 yrs, VO2max: 4.8 ± 0.8 L/min). Each participant performed 60 (45) min on a cycle ergometer at 75% (85%) HR Max preceded by a 60-min high-intensity cycling intervention (CRIT60). Echocardiography was performed to measure strain 1) at rest in a semi-recumbent position (45° supine, 45° left lateral) and 2) during low-intensity cycling exercise in a semi-recumbent position (power output: 91 ± 8 W; heart rate: 100 ± 3 beats/min). RESULTS: Average heart rate increase in response to the CRIT60 was 169 ± 4 beats/min (185% of maximum heart rate). Mean arterial pressure was reduced after the CRIT60 at rest (96 ± 1 mmHg vs. 86 ± 2 mmHg, p < 0.001) but not during low-intensity exercise (98 ± 2 mmHg vs. 97 ± 2 mmHg, ns). At rest there were no significant changes in strain. During low-intensity exercise, LV GLS (-20.0 ± 0.5 % vs. -18.2 ± 0.4 %, p < 0.01) and RV GLS (-29.6 ± 1.6 % vs. -27.7 ± 1.8 %, p = 0.1) were significantly reduced after the CRIT60 intervention.
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Aim of this study was the detailed assessment of the LV function with 3DST after exercise, these limitations can be eliminated.

Conclusions: Changes in ventricular strain following exercise were more evident when assessed during exercise echocardiography and may be indicative of functional cardiac limitations following 60 min of high-intensity exercise. Reductions in LV strain were unique to the septal wall and may reflect bi-ventricular interactions.

Methods: We examined LV function of 60 marathon runners (♂, age 42±9y, BMI 23.0±2.0 kg/m²) with the new software “TomTec 4D LV-Function” one week before (V1), and (V2), 24h, and 72h after the race. Besides ejection fraction (EF), we also assessed maximum rotation (twist=∑ of the rotation of the basal and apical LV plane), time to peak rotation (TIP), and LV dysynchrony (systolic dysynchrony index SDI I & II).

Results: EF was similar immediately post- compared to pre-race (V1: 56.2±6.9 % vs. V2: 55.9±6.5 %, p=0.48), increased within 24h significantly (58.6±6.1 %, p=0.02), and returned to baseline values within 3days (56.4±5.9 %, p=0.87) post-race. Parameters of 3DST showed a significant increase of twist both immediately (V1: 15.4±4.5° vs. V2: 17.9±5.3°, p=0.001) and 24h post-race (17.8±4.1°, p=0.004) and to baseline values within 72hrs (16.7±4.7°, p=0.06). This increase within the first 24hrs post-race resulted primarily in the increase of the rotation of the basal LV-plane (V1: -6.1±2.1° vs. V2: -7.8±2.8°, p=0.001). However, there was a significant and clinically relevant increase of LV dysynchrony immediately post-race (median [IQR]: SDI1 4.41 [3.48-6.40] vs. 7.61 [6.09-9.26], p=0.001; SDI2 3.49 [2.50-7.74%] vs. 7.42 [2.70-11.33%], p=0.015) with normalization within 72hrs (p=n.s.).

Conclusions: We demonstrated increased LV-twist immediately post-marathon race which was mainly due to an increased rotation of the basal plane. Additionally, we observed an increased intraventricular LV dysynchrony. However, this impairment had no influence on LV-EF and might be compensated by the increased twist maintaining cardiac output adequately.

Declines in Left Ventricular (LV) diastolic filling performance, which occurs with aging, can lead to heart failure. Exercise has been known to help reduce changes in LV diastolic performance and attenuate LV dysfunction. PURPOSE: To assess the impact of a common musculoskeletal injury on the age-related changes in LV diastolic filling parameters in active mice. METHODS: Thirty male mice (CBA/J) were randomly placed into one of three groups: the transected CFL group, the transected ATFL/CFL group, and a sham group. Three days after surgery, all mice were individually housed in a cage containing a solid surface running wheel and daily running wheel measurements were recorded (distance, duration, speed). Before surgery and every 6 weeks after surgery LV diastolic filling parameters and heart rate were measured (E and A waves) under 2.5% isoflurane inhalation training at the first week for 15 minutes per day, and the daily 180-minute interventional high-intensity swimming training from the fourth to seventh week. Before the coronary artery occlusion, the ischemic preconditioning group received a couple cycles of coronary artery left anterior descending occlusion for 10 minutes, followed by reperfusion for 10 minutes. RESULTS: The results of morphological staining showed that the number of infant cells of the control group is higher than those of the swimming group and ischemic preconditioning group. The blood examination results showed that the enzyme concentrations in heart, liver and kidney of the control group are higher than those of the swimming group and ischemic preconditioning group. Conclusions: This study found that the daily 180-minutes high-intensity swimming training intervention has the effect of enhancing myocardial protection.

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