CONCLUSIONS: Preliminary findings from our ongoing study in overweight and obese adults suggest that acute AE or RE performed the evening prior to an OGTT does not attenuate postprandial decreases in FMD.

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2229 Board #65 June 1 11:00 AM - 12:30 PM

Effects of Obese Skeletal Muscle Cells on Endothelial Cell Angiogenesis

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(No relevant relationships reported)

Vascular disease is a leading cause of morbidity and mortality in obesity. Obesity is associated with impaired endothelial cell (EC) angiogenesis. Skeletal myocytes are important regulators of angiogenesis - EC proliferation, migration, and tube formation.

PURPOSE: Determine the effects of obesity on skeletal muscle regulation of EC angiogenesis.

METHODS: Primary human skeletal muscle satellite cells were isolated from the vastus lateralis from lean (LN) and obese (OB) subjects and differentiated into myotubes (HSkMC). Conditioned medium (CM) from HSkMC was collected after a two-day incubation period and used to treat Human Umbilical Vascular Endothelial Cells (HUVECs). HUVEC proliferation was assessed via cell counting, cell viability was determined using an MTT assay, and EC tube formation (tube length and branches) was measured in matrigel after a 4-hour incubation.

RESULTS: After 24-hour treatment, there was no difference in HUVEC proliferation (LN: 23,333 vs. OB: 22,750, cells) or viability (LN: 181.62 vs. OB: 183.52, AU) between LN and OB HSkMC CM. Also, there was no difference in HUVEC tube length (LN: 23,726 vs. OB: 24,046, AU) or branches (LN: 452 vs. OB: 465, AU) between LN and OB HSkMC CM.

CONCLUSION: In cell culture, there is no apparent effect of obesity on skeletal muscle regulation of endothelial cell angiogenesis. However, incubating cells (SkM and EC) with high glucose or high fatty acid, metabolic challenges that are present in vivo, may reveal insights into obesity impaired angiogenesis.

2230 Board #66 June 1 11:00 AM - 12:30 PM

Perfusive and Diffusive Microvascular Oxygen Delivery During Simulated Hypovolemia and Dynamic Forearm Exercise

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(No relevant relationships reported)

The maintenance of brachial artery blood flow during dynamic forearm exercise in the face of simulated hypovolemia (via lower-body negative pressure (LBNP)), has been previously demonstrated. The distinct facets of microvascular oxygen delivery (i.e. perfusive and diffusive) during such an event, however, have not been described.

PURPOSE: We tested the hypothesis that, during dynamic handgrip exercise, the initiation of LBNP would result in no significant changes in the indices of microvascular perfusive or diffusive oxygen delivery (deoxy-[heme] and total-[heme], respectively) in the exercising muscle.

METHODS: Six men (26.2 ± 1.7 yrs, 85.5 ± 6.2 kg, 177 ± 1 cm) participated in this study. To determine the effects of LBNP in the absence of exercise, LBNP (~30 mmHg) was applied for two minutes following a resting baseline. After recovery to a second resting baseline, subjects performed seven minutes of dynamic handgrip exercise at 20% MVC. During the final two minutes of exercise, LBNP was initiated. Mean arterial pressure (MAP) was measured continuously via calibrated finger photoplethysmography (Finometer Pro, FMS). Absolute concentrations of deoxy-[heme] and total-[heme] of the flexor digitorum superficialis muscle were measured continuously via frequency-domain multi-distance near-infrared spectroscopy (OxiplexTS, ISS).

RESULTS: MAP (92.4 ± 12.8 mmHg), deoxy-[heme] (83.7 ± 14.5 μM) and total-[heme] (343 ± 48 μM) were not different between resting baselines ($p > 0.05$). While all subjects demonstrated an increase in deoxy-[heme] (99.1 ± 8.6 μM) following the application of LBNP at rest, intersubject variability precluded statistical significance ($p > 0.05$). No significant changes were detected in MAP or total-[heme] ($p > 0.05$). Dynamic handgrip exercise resulted in significant increases in MAP (104 ± 14 mmHg), deoxy-[heme] (121 ± 29 μM) and total-[heme] (367 ± 52 μM) ($p < 0.05$); however, the initiation of LBNP during exercise resulted in no significant further changes in MAP, deoxy-[heme] or total-[heme] ($p > 0.05$).

CONCLUSION: The absence of any significant changes in deoxy-[heme] or total-[heme] during simulated hypovolemia (i.e. LBNP) suggests that perfusive and diffusive microvascular oxygen delivery to skeletal muscle was preserved at rest and during dynamic handgrip exercise.

2231 Board #67 June 1 11:00 AM - 12:30 PM

Influence of Short, Disrupted Sleep and High-Intensity Interval Exercise on Brachial Artery Vascular Responses

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(No relevant relationships reported)

Brachial artery flow-mediated dilation (FMD) is a nitric oxide-dependent measure of conduit artery endothelial function that is transiently potentiated by exercise; yet, it is unclear how short, disrupted sleep (SDS) modifies post-exercise FMD responses to a single episode of high-intensity interval exercise (HIIE).

PURPOSE: To determine the influence of a single night of SDS on brachial artery FMD responses after HIIE.

METHODS: Fifteen male participants (age 31.1 ± 5.3 yr; weight 83.5 ± 11.4 kg; BMI 25.8 ± 2.7 kg/m^2; VO_2max 49.1 ± 8.5 ml/kg/min) completed a non-exercise control trial after 9 to 9.5 hrs of reference sleep (REF), HIIE by treadmill running (90% and 40% of VO_2reserve in 3:2 min ratio) to expend 500 kcals after reference sleep (REF+EX) and HIIE after 3 to 3.5 hrs of short and disrupted sleep (SDS+EX) in a randomized crossover design. Ultrasound measurements of brachial artery FMD were obtained by the same technician under standardized conditions just before, 1 hr and 4 hrs after exercise. FMD responses were analyzed using 3 (condition) by 3 (sample point) repeated measures ANOVAs.

RESULTS: FMD was augmented 1 hr after exercise in REF+EX (pre-exercise = 12.5 ± 0.9; 1 hr = 17.2* ± 1.5; 4 hr = 12.5 ± 0.9%) and SDS+EX (pre-exercise = 14.9 ± 1.7; 1 hr = 19.3* ± 2.2; 4 hr = 16.2 ± 2.4%) versus no change in REF (pre-exercise = 12.6 ± 1.4; 1 hr = 11.3 ± 1.0; 4 hr = 13.5 ± 2.1%) ($p < 0.0494$ condition by time interaction). **SUMMARY:** HIIE transiently augments brachial artery FMD and this response is not modified by a single night of short, disrupted sleep.

2232 Board #68 June 1 11:00 AM - 12:30 PM

Obesity Associated Hypertension in Admitted Patients: Treating Isolated Systolic Hypertension May Be Short Sighted

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(No relevant relationships reported)

The close association of excess adiposity and elevated blood pressure is well documented. The role of obesity as a contributing factor for resistant hypertension, cardiovascular disease, and cerebrovascular disease is recognized. Given that exercise training could be used to treat both obesity and elevated blood pressure when patients are admitted for trauma care, identifying obesity-associated systolic hypertension may help with long-term control of the root causes for these illnesses.

PURPOSE: To examine the relationship between body composition, blood pressure, and other measures collected on patient admittance.

METHODS: Data were collected from 2,306 consecutive patients admitted to a Level 1 trauma center between July, 2012 and June 2015. Patients with head trauma or traumatic brain injury were not analyzed. Patients were considered obese if their BMI was ≥ 30. Multiple linear regression was used to examine the effect of obesity on blood pressure. Other significant variables were examined from the database that contributed to the prediction model.

RESULTS: Significant predictors of systolic hypertension included blood lactate, age, obesity, pulse pressure, pH, %O2 saturation and hemoglobin levels (R=0.464; p<0.001). Holding all other variables constant, obesity was associated with a 9.7 mmHg increase in systolic blood pressure (p=0.009). A mild (3 mmHg) increase in diastolic blood pressure was noted, but was not found to be statistically significant (p=0.172).
CONCLUSIONS: The demonstrated relationship between obesity and systolic blood pressure illustrates the need for integrated blood pressure and obesity treatment with exercise training. Therapeutic exercise focused on weight loss goals will likely ameliorate elevated systolic blood pressure. Weight management discussions are challenging, and often avoided by health professionals, but these data show that concomitant antihypertensive medication with therapeutic exercise training may be warranted for prevention of subsequent cardiovascular and cerebrovascular diseases.

2233 Board #69 June 1 11:00 AM - 12:30 PM

Changes in Scattering, Absorption, and Resulting Differential Pathlength Factor During Arterial Occlusion and Reperfusion

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(No relevant relationships reported)

Continuous wave near-infrared spectroscopy (CW-NIRS) has been used to assess microvascular function and the balance between muscle oxygen delivery and oxygen consumption via post-occlusion reactive hyperemia (PORH) tests. However, CW-NIRS relies on the assumption that the scattering and absorption characteristics of the investigated tissue remain unchanged via a constant differential pathlength factor (DPF).
PURPOSE: We tested the hypothesis that the DFP of forearm tissue would be significantly different among the phases of a PORH test (i.e. baseline, arterial occlusion, and arterial reperfusion).
METHODS: 5 subjects (22.6 ± 1.8 yrs, 170 ± 5 cm, 66.0 ± 10.8 kg) completed three PORH tests consisting of 1 min of baseline, 5 min of brachial arterial cuff occlusion, and 3 min of recovery following arterial reperfusion. Reduced scattering (μ_s') and absorption (μ_a) coefficients were continuously measured, and later used to calculate a DPF, at wavelengths of 692 and 834 nm (DPF_{692} and DPF_{834}, respectively) via frequency domain near-infrared spectroscopy (FD-NIRS) during the entire duration of the PORH tests. The minute averaged DPF response was averaged among the three PORH tests.
RESULTS: DPF_{692} was significantly greater that DPF_{834} during each minute of the PORH tests ($p < 0.05$). DPF_{834} did not significantly change during any phase of the PORH test from baseline (3.83 ± 0.79; $p > 0.05$). DPF_{692} was significantly less during the final minute of arterial occlusion (4.07 ± 0.69) when compared to baseline (4.67 ± 0.78; $p < 0.001$). Further, following arterial reperfusion, DPF_{692} was significantly greater (4.91 ± 0.78) when compared to the final minute of arterial occlusion ($p < 0.001$), but not different when compared to baseline.
CONCLUSION: These data demonstrate that the DPF_{692} of forearm tissue does not remain constant across the phases of a PORH test. The assumption of a constant DPF may alter interpretations of data related to microvascular function and the balance between muscle oxygen delivery and oxygen consumption obtained via PORH tests.

2234 Board #70 June 1 11:00 AM - 12:30 PM

Exercise-levels Of Laminar Shear Stress In Combination Of Aspirin And Celecoxib Normalize An Atherognic Environment

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(No relevant relationships reported)

Optimal vascular function is a hallmark of cardiovascular health. Specifically, the balance of vasoconstricting and vasodilating substances in the vascular bed is recognized as a surrogate measure of the health of resistance vessels. Endothelial Nitrix Oxide Synthase (eNOS) is considered to be one of the best indicators of vasokine balance in these vessels, with high levels of expression being considered to be favorable. Further, the balance of the vasodilating/anti-thrombotic substance prostacyclin and vasoconstricing/ pro-thrombotic substance thromboxane in the endothelial cell layer is a further indicator of the overall health of the cardiovascular system. One of the greatest challenges to vascular health and vasodilatory balance is TNFα-mediated inflammation. Uncovering effective strategies that maintain a vascular environment that is more vasodilatory and anti-thrombotic in the face of an inflammatory challenge is favorable.
PURPOSE: To test the ability of various anti-thrombotic and pro-vasodilatory treatments, as well as combinations thereof, to prevent disruptions of vascular health of endothelial cells when faced with an inflammatory challenge in the form of TNFα.
METHODS: Human Umbilical Vein Endothelial Cells HUVECs were pre-treated exercise-like levels of laminar shear stress (LSS), aspirin, celecoxib, and their combination prior to a TNFα challenge. Western blot analysis, as well as calorimetric assays were used to determine levels of eNOS and prostacyclin (6-keto $PGF_{1\alpha}$)/thromboxane (TXB_2) metabolite ratio, respectively.
RESULTS: Neither aspirin, nor celecoxib was effective in preventing TNFα-induced reduction in eNOS. Further, aspirin was unable to maintain baseline levels of prostacyclin/thromboxane ratio in the face of the inflammatory challenge. LSS, aspirin/LSS combination, and celecoxib/LSS combination were all able to prevent TNFα- induced reductions in eNOS levels and prostacyclin/thromboxane ratio.
CONCLUSION: Effective strategies to maintain a healthy endothelium and therefore resistance vessel health, need to include exercise-levels of shear stress to be effective.

2235 Board #71 June 1 11:00 AM - 12:30 PM

Abdominal Aorta Compliance and Distensibility Among Youth Ranging from Normal Weight to Severe Obesity

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(No relevant relationships reported)

PURPOSE: This study evaluated abdominal aorta stiffness and diameter among youth throughout a range of body mass index (BMI) values.
METHODS: Non-invasive ultrasonographic measurements of the abdominal aorta were obtained from 190 youth (92 males; mean±SE: age=12.9±0.2 years). Body composition was assessed by dual- energy X-ray absorptiometry. Obesity status was defined using age- and sex-based BMI percentiles: normal-weight (NW) (≥5th to <85th percentile); overweight (OW) and obese (OB) (≥85th to <1.2 X 95th percentile); and severe obese (SO) (≥1.2 X 95th percentile). Analysis of covariance compared differences by obesity status with adjustments made for race, sex, and Tanner stage. Multiple linear regression evaluated the association of sex, age, and percent body fat (%BF) on abdominal aorta elasticity.
RESULTS: Prior to adjustment, abdominal aorta diameter (aBD) was significantly larger among SO (mean±SE: 11.1±0.4 mm) compared to both OW/OB (9.6±0.3 mm, p=0.006) and NW (8.9±0.3 mm, p=0.001). Abdominal aorta incremental elastic modulus (aIEM) was higher among SO (1153.0±70.8 mmHg) compared to OW/OB (960.4±48.6 mmHg, p=0.044) and NW (846.0±40.9 mmHg, p<0.0001). Abdominal aorta diameter distensibility (aDD%) was lower among SO (14.2±0.6%) compared to NW (16.6±0.6%, p=0.029). Abdominal aorta cross-sectional distensibility (aCSD) was lower among SO (30.6±1.5%) compared to NW (36.4±1.5%, p=0.03). After adjusting for covariates, aBD remained significantly larger among SO compared to OW/OB (p=0.018) and NW (p=0.001); aIEM was significantly higher among SO compared to NW (p=0.002). Adjusted aDD% and aCSD were not significantly different among groups. Age was associated with higher aBD (β=0.41, p<0.001), higher aIEM (β=42.92, p<0.001), decreased aDD% (β=-0.38, p=0.007), and decreased aCSD (β=-0.96, p=0.004). Percent body fat was associated with both higher aBD (β=0.06, p=0.001) and aIEM (β=7.44, p=0.007), while sex was not associated with measures abdominal aorta elasticity and stiffness.
CONCLUSION: The deleterious effect of obesity on arterial stiffness extends to the abdominal aorta. Higher age and BF%, but not sex, was associated with greater abdominal aorta stiffness.