Abstracts

Oral Presentations
Friday, 7:00 to 7:55 am and 1:30 - 3:30pm

Poster Presentations
Thursday during the Lunch Break
Impella 2.5 and CP Use at a Rural Community Hospital; Experience and Outcomes with 97 Implants 2011-2106
Presenter: Charles Wilkins, MD, FACC, FSCAI
Presenter Institution: San Juan Regional Medical Center, Farmington NM

Charles Wilkins MD FACC FSCAI, Faraz Sandhu MD FACC FSCAI, Luther Weathers MD, Sudhakar Girotra MD, Tammie Herrera RTR, RCIS, Monica Nagahiro RN RCIS, Rebekah French RN BSN RCIS, Barbara Charles RN BSN CCCC

San Juan Regional Medical Center, Farmington NM

San Juan Regional Medical Center is a 194-bed community hospital in northwest New Mexico 175 miles from the nearest tertiary medical facility. A coronary interventional program without cardiovascular surgery on site was established in 2007. From November 2011 through September 2016, 97 Impella devices (2.5 or CP) were implanted for mechanical circulatory support (MCS) in patients presenting with an acute myocardial infarction (AMI) and cardiogenic shock (CGS), ventricular tachycardia/ventricular fibrillation (VT/VF) arrest patients with ROSC (return of spontaneous circulation), high-risk PCI patients, and transfer of cardiogenic shock patients requiring a higher level of support.

30 and 180 day survival of patients with AMI/CGS and successful PPCI (primary percutaneous coronary intervention) with Impella MCS was 90%. Initiation of Impella support prior to PPCI in patients with CGS did not significantly delay door to reperfusion times (40 minutes institutional median time vs. 42 minutes Impella pre PPCI) and did not alter survival.

VT/VF arrest patients with successful PPCI and Impella MCS had a 30 and 180-day survival of 75% and 68%.

High-risk PCI patients had a 92% 30-day and 180-day survival. Indications for Impella supported PCI included severe 3-vessel disease (Syntax score > 33), last patent vessel, or unprotected left main coronary intervention in patients who were not surgical candidates or had declined surgery.

Nine patients have been transferred uneventfully with Impella support to a tertiary center for either emergent coronary artery bypass or for consideration of implantation of a left ventricular assist device (LVAD). 30 and 180 day survival in patients transferred for revascularization was 20% but 75% in patients transferred for consideration of an LVAD.

Conclusions: Early Impella use for MCS coupled with a vigorous staff education program provides exceptional outcomes for patients with AMI/CGS, VT/VF arrest, and high risk PCI patients. Impella also allows the safe transfer of critically ill patients from a rural remote setting without cardiovascular surgery support to a tertiary center for either emergent revascularization or for consideration of durable LVAD implantation.
Profiling Plasma Caspase Activity in Heart Failure and During LVAD support as Biomarkers of Cardiac Health and Recovery

Presenter: Frank Jefferson, MD
Presenter Institution: Duke University, Durham, NC

Frank Jefferson, Muath Bishawi, Salvatore V. Pizzo, Chetan B. Patel, Carmelo A. Milano, and Dawn E. Bowles

Background: Many forms of cell death, including apoptosis, have been implicated as a common underlying cellular process involved in human heart disease and failure. Treatments of end stage heart failure through mechanical circulatory assist devices are thought to enable myocardial recovery, partly through reversing or minimizing ongoing cell death. Cysteine-aspartic proteases, or caspases, the downstream effector molecules activated in apoptosis, are elevated in heart failure and are important therapeutic targets as well as potential biomarkers. We hypothesized that plasma caspase activity levels in patients should diminish following LVAD support and these levels may later be predictive of ventricular recovery.

Methods: Longitudinal plasma samples derived from 49 LVAD patients (246 patient samples) and eight healthy participants (20 samples) were profiled for both pan-caspase and caspase 3/7 activity using commercially available bioluminescent assays from Promega (Madison WI). A clinical chart review was also performed to identify patient outcomes post LVAD implantation, and the correlation between caspase activity and adverse events was examined.

Results: Pan-caspase levels were significantly elevated in heart failure patients compared to healthy controls (figure 1a). Unexpectedly, in post LVAD implantation plasma, the activity of all caspases tested increased on average compared to pre-implant levels. These levels did not decrease, in fact, caspase activity over time continued to be elevated overall on LVAD support. Additionally, for patients who eventually experienced a device thrombosis, caspase 3/7 activity was significantly higher 3-5 days after surgery compared to LVAD patients with no thrombosis (p<0.05; figure 1b).

Conclusion: The global apoptotic status of an individual may be informative of his/her overall systemic health status, especially in heart failure. Caspase activity represents a promising biomarker that may help predict thrombosis events in patients undergoing LVAD implantation. It is unclear why systemic caspase activity is elevated post LVAD implantation. Further study is needed to determine if caspase activity relates to ventricular recovery given its elevation in heart failure.

Figure 1
Peripheral Vascular Pulsatility in Heart Failure Patients with Continuous Flow Centrifuge and Axial Left Ventricular Assist Devices: The Effect of Pump Speed.

Presenter: Jay Hydren, MS
Presenter Institution: University of Utah

Jay R. Hydren¹, Andrew C. Kithas², Soung Hun Park¹, Omar Wever-Pinzon², Craig H. Selzman³, William Perry², Camila A.S. Vargas³, Stavros G. Drakos² and Russell S. Richardson¹,²,⁴

¹Department of Nutrition and Integrative Physiology, the ²Department of Internal Medicine, and the ³Department of Surgery, University of Utah School of Medicine, Salt Lake City, UT. ⁴Geriatric Research, Education, and Clinical Center, Salt Lake City VA Medical Center, UT.

Current continuous flow left ventricular assist devices (LVAD) decrease peripheral vascular pulsatility, which may contribute to side effects such as bleeding, thrombotic events and orthostatic intolerance. PURPOSE: To investigate the impact of manipulating LVAD pump speed, documented as revolutions per minute (RPM), on peripheral (brachial artery) pulsatility index (PI) in 20 heart failure patients implanted with a HeartWare (HVAD, n = 10) or HeartMateII (HMII, n = 10) LVAD.

METHODS: Doppler ultrasound blood velocity in the brachial artery was recorded at baseline and 3 minutes after altering RPM, at three different RPM settings above and below baseline (60 RPM increments for HVAD and 200 for HMII). Brachial PI was calculated for each cardiac cycle by dividing the difference between minimum and maximum blood velocity by the time averaged mean blood velocity. LVAD device pulsatility indices that are used clinically were also recorded: maximal blood velocity (HVADVmax) and minimum blood velocity (HVADVmin) (HVAD) and HMIIPI (HMII). Relationships were evaluated using multilevel linear modeling with random intercepts and data are reported as mean±SE. RESULTS: Baseline RPMs were 2509±44 (HVAD) and 9220±75 (HMII). Brachial PI changed significantly across the range of LVAD RPM speeds tested (HVAD: 360; HMII: 1200), from 2.3±0.6 to 4.1±0.9 with the HVAD and from 1.8±0.6 to 3.6±1.0 with the HMII, with no differences in brachial PI between device across relative pump speed stages. Specifically, a 180 RPM decrease of the HVAD resulted in a 0.9±0.1 (37±4%) increase in brachial PI and a 600 RPM decrease in the HMII resulted in a 0.8±0.1 (38±3%) increase. These reductions in pump speed resulted in an ~20.0% fall in LVAD power consumption and a reduction in device reported blood flow of ~9%. Brachial PI correlated with HVAD HWVmax and HWVmin (r = 0.45 and r = -0.31, respectively), and HMII device HMIIPI (r = 0.73), suggesting device derived indices of PI provide a fair to good linear prediction of peripheral vascular pulsatility.

CONCLUSION: Reducing HVAD or HMII LVAD pump speed within a clinically acceptable outpatient range yields a measurable and potentially clinically and physiologically meaningful change in peripheral vascular pulsatility, accompanied by substantial power savings.
Cerebral Autoregulation and Brain-Tissue Oxygenation are Preserved in Patients with Continuous-Flow Left Ventricular Assist Devices

Presenter: William K. Cornwell III, MD  
Presenter Institution: University of Colorado, Aurora, CO

Background: Continuous-flow (CF) left ventricular assist devices (LVADs) are associated with greater survival than previous-generation pumps, which provided pulsatile circulatory support. Nevertheless, there are many risks associated with chronic exposure to minimally/entirely nonpulsatile flow, such as elevated sympathetic nerve activity and hypertension. In addition, stroke impacts greater than 10% of patients within the first year of mechanical support alone, raising concern that the reduction in pulsatility may increase the risk of a cerebrovascular accident (CVA). Therefore, the purpose of this study was to investigate whether changes in cerebral blood flow (CBF) pulsatility influence cerebral autoregulation and CBF-brain tissue oxygenation coupling in this population.

Methods: Six male subjects (67±11 years) with Heartmate II CF-LVADs, who were stable and fully recovered from surgery (18±12 months), underwent hemodynamic assessment. LVAD pump speed was increased in a stepwise fashion from 8367±234 to 10400±219 RPM to generate large oscillations in pulsatility. After each pump speed adjustment, five minutes were allotted for stabilization/re-equilibration, followed by five minutes of data collection. Heart rate and blood pressure were continuously recorded via finger plethysmography (Nexfin). Middle cerebral arterial velocities (MCAV) and cerebral tissue oxygenation index (TOI) were continuously recorded via transcranial Doppler and near-infrared spectroscopy (NIRS), respectively, to assess changes in cerebral perfusion and neuronal activity. Fourier transformation was used to assess cerebral autoregulation (by comparing changes in MAP and MCAV) and CBF-TOI coupling (by comparing changes in MCAV and TOI) at the highest and lowest pump speeds achieved. Data were analyzed in the very low (0.02-0.07Hz) and low (0.07-0.20Hz) frequencies, since the autonomic nervous system exerts its greatest influence over vascular tone in these frequency ranges.

Results: A representative figure of the changes observed in hemodynamic parameters during pump speed adjustments is displayed in Figure 1. Increases in pump speed led to an increase in LVAD flow (4.9±0.7 v. 6.3±1.2LPM at low v. high speed, P<0.05) and mean arterial pressure (MAP, 80±16 v. 97±24mmHg, P<0.05), whereas pulse pressure was reduced (36±18 v. 25±18mmHg, P<0.05). Pump speed adjustments did not affect MCAV (47±11 v. 45±13cm/sec, P=NS) or cerebral TOI (65±10 v. 63±10%, P=NS). Transfer function analysis (Figure 2) demonstrated that the gain for autoregulation (Figure 2A, comparing MAP v. MCAV), as well as the gain for CBF-TOI coupling (Figure 2B, comparing MCAV v. TOI), were similar at low and high pump speeds.

Conclusion: Cerebral autoregulation and CBF-TOI are remarkably preserved among individuals with CF-LVADs, despite large oscillations in pulsatility and arterial pressure. These findings suggest that autoregulatory processes and brain tissue oxygenation are tightly regulated and maintained in the setting of chronic exposure to diminished pulsatility.
Figure 1: Representative figure of hemodynamic oscillations observed at high and low pump speeds in a patient with a Heartmate II CF-LVAD.

BP: blood pressure; MAP: mean arterial pressure; PP: pulse pressure; MCAV: middle cerebral arterial velocity; TOI: tissue oxygenation index.
Figure 2: Transfer function analysis of induced oscillations in mean arterial pressure, cerebral perfusion and oxygenation.

Figure 2A shows transfer function gain, phase and coherence of MAP and MCAV at low and high pump speeds. Gain in the very low (0.02-0.07Hz) and low (0.07-0.20Hz) frequency ranges was similar between low and high pump speeds (P=NS), suggesting preservation of cerebral autoregulatory processes. Figure 2B shows transfer function gain, phase and coherence of MCAV and TOI at low and high speeds. Gain in the very low and low frequency ranges was similar between low and high pump speeds (P=NS), indicating that CBF-TOI coupling is maintained across wide oscillations in arterial pressure and pulsatility.
Scientific Considerations: Accuracy, Physiology, Details (the devil lives here).
Presenter: Alan Morris, MD
Presenter Institution: University of Utah, Intermountain Healthcare, Salt Lake City, UT
Friday: 1:30 - 3:30pm
Session 7: Oral Presentations of Best Abstracts
Chairpersons: Alejandro Bertolotti, MD, Fundacion Favaloro, Buenos Aires, Argentina and Josef Stehlik, MD, MPH, University of Utah, Salt Lake City

Gene expression in Pediatric Myocardium: Pulsatile- vs. Continuous-Flow VAD Support
Presenter: Iki Adachi, MD
Presenter Institution: Baylor College of Medicine, Houston, Texas

Iki Adachi, MD1,2, Min Zhang, PhD3, Sarah Burki, MD, David Morales, MD, Aamir Jeewa, MD, Debra Kearney, MD, Lalita Wadhwa, PhD, Charles Fraser, MD, James F. Martin, MD, PhD3,4

1Division of Congenital Heart Surgery, Texas Children’s Hospital; 2Michael E. DeBakey Department of Surgery, Baylor College of Medicine; 3Department of Molecular Physiology and Biophysics and 4Cardiovascular Research Institute, Baylor College of Medicine, Houston, Texas

Background: In the previous era where pulsatile VADs were predominantly used, cardiac recovery was more common than in the current era of continuous-flow support. While the impact of ‘pulsatility’ on cardiac recovery remains unclear, it cannot be evaluated in the adult population since pulsatile VADs are no longer used. The pediatric population stands in a unique position where both pulsatile- and continuous-flow VADs are utilized, making comparable study feasible.

Methods: RNA sequencing was conducted on ventricular myocardium sampled at durable VAD implant (pre-) and explant (post-) in 23 children that had undergone bridge-to-transplant: pulsatile (n=13, the oldest 7.0 year old) and continuous-flow (n=10, the youngest 9.2 year old). Median (range) age and weight at implant were 6.5 (0.2 – 18) years and 16 (4 - 94) kg. Support duration was 2.5 (0.6 - 9.2) months. Tukey’s test was used for statistical comparison between multiple groups.

Results: Significantly upregulated genes (n=457) and downregulated genes (n=693) were identified using a false discovery rate of ≤0.05 and fold change (post-/pre-) of ≥1.2 (Fig A). Among the upregulated genes, mitochondrial electron transport chain (ETC) genes changed most significantly. Expression level of total 27 ETC component genes from all five ETC complexes were upregulated (B). When comparing the mean mitochondrial gene expressions stratified by device type, the magnitude of upregulation was more substantial with pulsatile VAD than continuous-flow VAD (C). Similar trends were seen in downregulated genes; extracellular matrix and immunity function genes downregulated more substantially with pulsatile VAD (D,E).

Conclusions: This study suggests favorable reverse remodeling process may occur at gene level in the pediatric myocardium during durable VAD support. Though the magnitude of such changes seems more substantial with pulsatile VAD support, careful interpretation would be necessary given the difference in patients’ demographics particularly age distribution.
Novel trends in speckle tracking echocardiography in non-ischemic cardiomyopathy patients with heart failure with recovered ejection fraction (HFrecEF).

Presenter: Stanley Swat, MD
Presenter Institution: Northwestern University Feinberg School of Medicine, Chicago, IL

Stanley Swat1, David Cohen3, Abi S. Baldridge4, Sanjiv J. Shah1, Benjamin H. Freed2, Stuart Prenner2, Jane E Wilcox2

1Northwestern University Feinberg School of Medicine-Department of Medicine, 2Northwestern University Feinberg School of Medicine-Division of Cardiology, 3University of Michigan Health System 4Northwestern University Feinberg School of Medicine-Department of Preventive Medicine

Background:
Heart failure (HF) guidelines recognize a subset of patients with left ventricular (LV) ejection fraction (EF) > 40% who previously had HF with reduced LVEF < 40%. Patients with improvement or recovery in LVEF may be clinically distinct from other groups of HF. Data on predictors of myocardial recovery are limited. We sought to examine the myocardial structural characteristics of patients with non-ischemic cardiomyopathy (NICM) with reduced LVEF < 40% after incident hospitalization for HF using speckle tracking echocardiography and examine trends associated with HF with recovered EF (HFrecEF).

Methods:
All patients, age ≥ 18 and ≤ 85 years, admitted to Northwestern Memorial Hospital between January 1st, 2000 and January 1st, 2013 with first diagnosis of HF with reduced LVEF (HFrEF) not due to flow-limiting coronary artery disease or prior myocardial infarction were identified using the Northwestern University Medicine Enterprise Warehouse. Reduced LV function was defined as LVEF < 40% on baseline echocardiogram within 90 days of index hospitalization. Myocardial recovery status was defined as: absolute LVEF improvement ≥ 10% and final LVEF ≥ 40% within 18 months post incident hospitalization for HF. Echocardiograms were imported and subsequently analyzed using speckle tracking to obtain strain parameters. We performed statistical tests using Chi-Squared, Anova or Kruskal-Wallis where appropriate.

Results:
We identified 282 participants with complete baseline information, of which 275 had follow up LVEF available. For the entire cohort, mean age was 56 years, 47% were female (128/275), 55% were African American (152/275), and 82% (225/275) had hypertension. Baseline demographic data for each group is displayed in Table 1. Myocardial recovery was achieved in 33% (93/275) of patients. Baseline LVEF was similar between the persistently ‘low-LVEF’ and ‘HFrecEF’ groups (24.2 ±7.94% vs 26.4±7.10%, p=0.08), as was global longitudinal strain (GLS) (-7.39±3.38 vs. -8.09±2.67 respectively, p = 0.22). Baseline radial strain was abnormal, but relatively preserved in the ‘HFrecEF’ group compared with the ‘low-EF’ group [4.00 (2.00-7.00) vs. 6.00 (4.00-9.00), p=0.02]. Patients who recovered had smaller baseline LV end diastolic dimension [4.79cm (4.41-5.31) vs 5.8cm (5.16-6.60), p = <0.0001] and shorter QRS duration [88.00ms (82.00-100) vs. 96.00 (86.00-120.00), p = 0.0076] compared with the low-EF group. Despite achieving a ‘normal’ LVEF among the ‘HFrecEF’ group (51.43 ±6.91%), GLS was overall improved, but still remained markedly abnormal (-13.19 ±4.09 vs. -9.09 ±3.94, p<0.001), while global circumferential strain (GCS) values normalized compared with the low-EF group (-22.73±6.14 vs. -15.02 ±6.19, p<0.001). HFrecEF status was associated with improved survival (14% vs. 33%, p=0.0022).

Conclusions:
In a racially diverse cohort of NICM patients with incident hospitalization for HF, patients who experience
myocardial recovery display a unique signature in cardiac structure and mechanics. Despite similar demographics, including baseline LVEF, patients with HFrecEF were more likely to have smaller cardiac dimensions, relatively preserved radial strain, and shorter QRS duration compared to patients with persistently low LVEF suggesting an underlying myocardial substrate that is less myopathic. HFrecEF status was associated with significantly improved survival over patients with persistently low LVEF. However, despite achieving a normal LVEF at follow up, the myocardial substrate is not normal among HFrecEF patients. GLS did improve, but values did not normalize, whereas absolute GCS normalized among HFrecEF patients.

Table 1:

<table>
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<tr>
<th>Baseline Demographics</th>
<th>N</th>
<th>Missing Follow-up Echo (n=7)</th>
<th>PERSISTENT 'LOW-LVEF' (n=182)</th>
<th>'HFrecEF' (n=93)</th>
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### Echo Characteristics

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<td>FOLLOW UP GCS</td>
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<td>QRS DURATION</td>
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<td>88.00 (82.00-100.00)</td>
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Coronary vascular function is improved in ischemic patients after continuous-flow left ventricular assist device (LVAD) implantation

Presenter: J David Symons, PhD
Presenter Institution: University of Utah, Salt Lake City, UT

Trevor Bonn¹, Peter Ferrin², Lance Deeter¹, Nikos Diakos², Josef Stehik², Craig Selzman², Abdallah Kfoury³, Bruce Reid³, Abdulfatah Saidi³, Omar Wever-Pinson², Divya R Verma³, Chi-Gang Yen³, Shawn Guo², Dean Li², Stavros Drakos², J David Symons¹,²

¹College of Health, University of Utah, Salt Lake City, UT; ²Internal Medicine, University of Utah, Salt Lake City, UT; ³UTAH Cardiac Transplant Program, University of Utah, Salt Lake City, UT

Background. Continuous-flow left ventricular assist devices (LVADs) are used in heart failure (HF) patients either as a bridge to transplantation or as a permanent (destination) therapy. HF is precipitated by myocardial infarction (MI) in most (ischemic; I) but not all (non-ischemic; NI) patients. We hypothesized that coronary vascular dysfunction is greater in I (63±2 y; n=12) vs. NI (53±5 y; n=17) male HF patients, and that dysfunction is attenuated by LVAD support. Methods and Results. Arteries from a transmural biopsy of the left-ventricle were obtained, and concentration-response curves to bradykinin (BK, 10⁻⁶ to 10⁻¹⁰ M) and sodium nitroprusside (SNP, 10⁻⁴ to 10⁻⁹ M) were completed after vessels were precontracted to ~65% of maximal tension development. Maximal BK-induced vasorelaxation was less (p<0.05) in arteries from I (68±8%; n=21 arteries, 195±20 μm i.d.) vs. NI (87±3%; n=33 arteries, 213±17 μm i.d.) patients, while responses to SNP (~90%) were similar between groups. These findings indicate endothelial dysfunction is greater in I vs. NI patients at the time of LVAD implant. Next we assessed vascular reactivity in 6 I (58±6 y) and 4 NI (49±11 y) patients wherein samples were obtained at implant and 239±51 days later at explant. Maximal BK-induced vasorelaxation was greater (p<0.05) in coronary arteries from I patients at explant (87±6%, n=14 arteries, 305±30 μm i.d.) vs. implant (53±11%, n=12 arteries, 204±33 μm i.d.). Maximal BK-induced vasorelaxation was similar in coronary arteries from NI patients obtained at explant (79±8%, n=7 arteries, 232±21 μm i.d.) and implant (72±17%, n=5 arteries, 135±13 μm i.d.). Responses to SNP were similar (~93%) at implant and explant for I and NI patients. Total collagen content (“total fibrosis;” TF) was assessed in a subset of patients from both paired groups, by quantifying whole-field stained tissue without excluding any areas. TF (%) was 12±2 and 9±2 at implant and explant, respectively, in 3 NI patients. TF (%) was 23±5 and 13±3 at implant and explant, respectively, in 5 I patients. Conclusion. These preliminary findings indicate that endothelial dysfunction and tissue fibrosis in patients with advanced ischemic cardiomyopathy is improved through LVAD support.
Evidence that Mesenchymal to Endothelial Transition (MET) occurs Secondary to Cardiac Unloading through LVAD support.
Presenter: Ana S. Cruz-Solbes, MD
Presenter Institution: Houston Methodist Hospital, Houston, TX

Ana S. Cruz-Solbes MD, Cesar Uribe MD, Keith A Youker PhD, Javier Amione-Guerra MD, Bashar Hannawi Jerry D Estep MD, Barry H Trachtenberg MD, Guha Ashrith MD, Guillermo Torre-Amione MD, PhD, Erick Suarez MD, Arvind Bhimaraj MD.

Houston Methodist Hospital

Background: Endothelial to mesenchymal transition (EndMT) contributes to fibrosis in heart failure. The role of the reverse phenomenon of MET in recovery of organ pathology is increasingly being recognized. Chronic unloading with LVADs provides an opportunities to study the mechanisms facilitated by unloading. We have demonstrated reduction in TGF-B (a known inducer of EndMT) post LVAD. The current study assesses changes in EndMT/MET after unloading of the LV.

Hypothesis: MET contributes to decreased fibrosis after LVAD implantation secondary to cardiac unloading.

Methods: We performed rtPCR on 13 paired samples of human myocardial LV obtained at LVAD implant and at time of heart transplant. SNAI1 was studied as surrogate marker of EndMT/MET. Fibrosis was quantified using Massons-Trichrome staining while fibroblast were counted with immunohistochemistry (FSP-1). Spearman's correlation was performed. Echocardiographic data was compared to assess effective unloading.

Results: Mean age was 54.8, 78% male, 50% African-American, 42% ICM, 25% had DM, 64% had HTN and all LVADs were HeartMateII. Median days of support was 314 days (100 - 1426 days). Echo data suggest significant chronic unloading (LVEF improved p=0.03, LA volume decreased ~40% p<0.0001, LVIDd decreased p=0.03). Significant decrease in fibrrosis post-LVAD occurred in 86% of the patients (p=0.008) with a decrease in absolute number of fibroblasts (p=0.03). SNAI1 increased significantly (p=0.03) after LVAD support, compared to pre-LVAD. Extent of fibrosis pre-LVAD had a significant correlation (R=0.75, p=0.003) to SNAI1 gene expression post-LVAD.

Conclusion: EndMT/MET play a role in fibrotic/anti-fibrotic mechanisms in human myocardium. SNAI1 expression had been proposed to play a role in the initial transition of MET. Our data supports this hypothesis: higher SNAI1 signal was associated with decreased fibrosis and fibroblast count post-LVAD. Correlation between post-LVAD SNAI1 with increased fibrosis pre-LVAD suggests more MET occurring in patients with a higher number of fibroblasts at baseline. Further studies of these pathways during chronic unloading might be relevant.
Effects of increased myocardial tissue concentration of myristic, palmitic and palmitoleic acids on the course of cardiac atrophy of the failing heart unloaded by heterotopic transplantation.
Presenter: Martin Pokorný, MD
Presenter Institution: Institute for Clinical and Experimental Medicine, Prague


Institute for Clinical and Experimental Medicine, Prague

The present experiments were performed to evaluate if increased heart tissue concentration of fatty acids, specifically myristic, palmitic and palmitoleic acids that are believed to promote physiological heart growth, can attenuate the progression of unloading-induced cardiac atrophy in rats with healthy and failing hearts. Heterotopic abdominal heart transplantation (HTx) was used as a model for heart unloading. Cardiac atrophy was assessed from the ratio of the native- to-transplanted heart weight (HW). The degree of cardiac atrophy after HTx was determined on days 7, 14, 21 and 28 after HTx in recipients of either healthy or failing hearts. HTx of healthy hearts resulted in 23 ± 3, 46 ± 3, 48 ± 4 and 46 ± 4% HW loss at the four time-points. HTx of the failing heart resulted in even greater HW losses, of 46 ± 4, 58 ± 3, 66 ± 2 and 68 ± 4%, respectively (p<0.05 for each time point). Exposure to isocaloric high sucrose diet caused significant increases in fatty acid concentrations in healthy and in failing hearts. However, these increases were not associated with any change in the course of cardiac atrophy, similarly in healthy and post-HTx failing hearts. We conclude that increased heart tissue concentrations of the fatty acids allegedly involved in heart growth does not attenuate the unloading-induced cardiac atrophy.
The Effect of Reverse Remodeling on Intraventricular Flow in the LVAD-Supported Heart Studied in a Mock Circulatory Loop

Presenter: Karen May-Newman, PhD
Presenter Institution: San Diego State University, San Diego, CA

Background

Heart failure etiology varies widely, but common features have been identified to classify patients for appropriate treatment. Among these features are cardiac geometry, as measured by changes in the size and shape of the left ventricle (LV), and myocardial function, indexed by ejection fraction (EF). The interaction of myocardial geometry and function influence remodeling of the heart in response to altered biomechanics, which change the state of stress in cardiac muscle cells and induce a feedback response. The changes observed with concentric and eccentric cardiac hypertrophy are examples of this interaction.

Implantation of mechanical circulatory support immediately boosts systemic blood flow, and is often followed by reverse remodeling (RR) of the heart, which includes a reduction in LV volume (LVV) and an increase in EF. The time course of RR likely reflects a biological response to the altered biomechanics produced by LVAD support, which is reflected in the LV flow dynamics.

Previous studies have measured a rapid reduction of LV volume shortly after LVAD implantation, accompanied by a mild improvement in EF. A better understanding of the observed response may yield new insight into the adaptive treatment needed to coax the heart to recover. Towards this end, the goal of this study was to measure the effect of EF and LVV on the LV flow dynamics of the LVAD-supported heart.

Methods

Reverse remodeling was modeled as a decrease in LVV while matching EF in the absence of LVAD support. Three silicone LV models were created with volumes of 180, 150, and 100 ml, and studied separately in a mock circulatory loop. Each LV model was connected to a Thoratec Heartmate II LVAD at the apex, using a transparent cannula. The circulating fluid was a viscosity-matched blood analog of 40% glycerol and saline.

Measurements of 2-D velocity in the midplane of a model left ventricle (LV) were made for several cardiac conditions using a cardiac simulator which generates LV flow patterns that closely resemble those obtained from patients. The two-dimensional (2D) velocity field of the LV model midplane was measured with Particle Image Velocimetry (PIV), an engineering method in which small fluorescent particles are added to the flow and visualized using a synchronized laser and camera system. The particle movement is used to measure the velocity field within a transparent model. Neutrally buoyant fluorescent particles (20 μm, PMMA-RHB) were added to the blood analog solution through the left atrial chamber. A LaVision PIV system captured images of the LV triggered from the hemodynamic signals, which produced a 40 Hz ensemble-averaged sequence of the 2-D velocity field for the cardiac cycle.

Vortex structures were analyzed by calculating the vorticity from the measured velocity, and using the Q criterion to identify the vortex boundaries. Circulation and kinetic energy (KE) were computed, as well as vortex size, shape and position during the cardiac cycle. A computational analysis of Lagrangian particle transport and LV washout was performed. Particle positions through several cardiac cycles were calculated, and the number of particles remaining at the end of the first, second and third cardiac cycle tabulated and normalized by the initial particle number to obtain the flow fraction. The reduction of flow fraction at the end of the first cardiac cycle constitutes the direct flow (DF), subsequent reduction during the second cardiac cycle is the delayed ejection fraction (DEF), and the remainder is the Retained Flow (RF). These fractions are then
scaled by the total aortic flow in order to compare absolute flow volumes.

**Results**

The baseline condition corresponds to a mean aortic pressure of 65 mmHg and a cardiac output of 3.5 L/min, combined with an LV volume of 180 mL to produce an ejection fraction of ~25%, representative of a dilated cardiomyopathy (DCM) patient with a heart failure severity of NYHA IV. The addition of LVAD support at 11 krpm produced an increase in systemic flow and pressure to approximately 5.7 L/min and 100 mmHg, respectively. Reduction of LV volume (LVV) for matched EF and LVAD support level did not produce a significant change in the overall hemodynamics. Intraventricular flow dynamics and transport were significantly affected by both LVAD support and LVV. Upon the introduction of LVAD support to the baseline LV model, Lagrangian transport was greatly improved, as reflected in a doubling of both DF and DEF, accompanied by a 35% reduction in RF. Vortex KE increased slightly, as observed in our previous studies. The changes in LV flow that accompanied LVV reduction during LVAD support at 11krpm indicated that some indices of transport are worsened, such as DF, which decreased by 70% over the range of LVV examined. In addition, RF increased by 35% with LVV reduction and matched EF. Vortex KE also decreased by 67% when LVV was reduced from 180 to 100 ml.

**Conclusions**

The results of this study indicate that vortex KE is reduced towards the pattern observed for normal hearts as LVV decreases. However, a reduction of LVV without an increase in EF does not provide an advantage for Lagrangian transport in the LVAD supported heart.

Additional studies investigating the effect of EF will further clarify the relative contributions of these two features of RR and perhaps improve insight into myocardial recovery.
Cardiac spinal sensory endings mediate remodeling in the post MI state
Presenter: Irving H. Zucker, PhD, Editor in Chief
Presenter Institution: The American Journal of Physiology-Heart and Circulatory Physiology, University of Nebraska, Omaha, NE

Irving H. Zucker, George J. Rozanski, Paras K. Mishra, Hanjun Wang

The mechanisms responsible for myocardial remodeling in the post myocardial infarction (MI) state has been studied in a variety of animals over the past several decades. Several molecular and signaling pathways have been identified that participate in both the fibrotic process and in the hypertrophic process. The role of cardiac nerves, especially sensory nerves has not been comprehensively investigated with regard to the remodeling process. Sensory endings in the heart transduce both mechanical and chemical stimuli in both normal and diseased states. Chemical activation of sensory nerves is often related to byproducts of myocardial ischemia released from myocytes into the interstitial space. These substances include hydrogen ion, potassium, arachidonic acid metabolites, bradykinin and other peptides. Activation of chemically sensitive afferent nerve endings evoke powerful reflex effects that are either sympatho-excitatory or sympatho-inhibitory depending on the dominance of spinal afferents or vagal afferents. Cardiac afferents that enter the thoracic spinal cord are known to be responsible for initiating ischemic pain as occurs in angina and are sensitized in cardiovascular diseases such as chronic heart failure. Activation of these afferents on the surface of the ventricular myocardium results in release of neuropeptides (Substance P and CGRP), intense local vasodilation and increases in permeability that can be completely blocked by substances that block the neurokinin 1 (NK1) receptor. This initiates an inflammatory state that may contribute to the cardiac remodeling process. Based on this preliminary work we hypothesized that selective removal of cardiac spinal afferent sensory endings would reduce fibrosis and inflammation in the myocardium in both the peri-infarct area and the remote myocardium. Studies were carried out in sham and coronary artery ligated rats approximately 12 weeks after myocardial infarction. The ultra-potent neurotoxin resiniferatoxin (RTX) was used to permanently ablate sensory nerves that lay near the surface of the myocardium. RTX activates the Transient Receptor Potential Vanilloid (TRPV) 1 receptor, increases calcium influx to the extent that it results in neuronal cell death. Sham and MI rats were treated with epicardial RTX or vehicle at the time of MI or sham surgery. After 12 weeks we confirmed complete inhibition of the response to epicardial bradykinin or capsaicin. Resting sympathetic nerve activity was normalized in post MI rats treated with RTX. Left ventricular end diastolic pressure (LVEDP) was significantly elevated in vehicle treated MI rats compared to sham (21.3±1 mm Hg compared to 5.0±0.4 mm Hg; p<0.05). However, there was a significant reduction in RTX MI rats (8.3±0.7 mm Hg). There were no differences in infarct size or ejection fraction. RTX significantly reduced indices of fibrosis including collagen, fibronectin and the TGFβ1 receptor. Scanning EMs of peri-infarct tissue from vehicle treated animals showed thickened and disrupted collagen bundles that were normalized in RTX treated rats. Importantly, MMP2 and MMP9 activities were markedly reduced (zymography) in RTX treated MI rats. Pressure-volume analysis indicated that diastolic function was improved in RTX treated MI rats. Inflammatory mediators such as TNFα and IL1β in remote myocardial areas were reduced in RTX treated animals. CD68 positive macrophages were also reduced in the RTX treated animals. Finally, RTX treated rats exhibited a marked increase in survival over a six-month period (40% in vehicle treated rats compared to 85% in RTX treated rats). Overall, these data provide strong support for a role of cardiac sensory nerves in myocardial remodeling in the post MI and chronic heart failure states. Further work is needed to ascertain the therapeutic window and optimal route of administration.
Combination Bioelectric Stimulator, Micro Infusion Pump and Multi Protein Composition for Heart Recovery in LVAD patients.
Presenter: Leslie Miller, MD
Presenter Institution: Leonhardt Ventures, Salt Lake City, UT

Howard Leonhardt, PhD, Leslie Miller, M.D., Jorge Genovese, Ph.D., Amit Patel, M.D., Doris Taylor, Ph.D., Mark Cunningham, M.D., Ph.D., Craig Saltzman, MD.

Background: LVADs provide near total LV decompression and are the ideal platform to study myocardial recovery, but alone their benefit has been insufficient to induce sufficient and sustained recovery of cardiac function to allow LVAD removal in a significant percentage of patients. Previous stem cell based approaches have been limited by the use of only on a single cell type or gene and a single delivery. We have developed a very novel approach which utilizes bioelectric stimulation followed by continuous infusion of a composite of pro-regenerative proteins to achieve enhanced tissue regeneration and recovery.

Aims: We will examine the comparative benefit of programmable and validated bioelectric stimulation plus continuous infusion of a composition of 10 pro-regenerative proteins directly into an area of infarct-induced scar in patients supported with an LVAD due to previous acute MI. The aim is to demonstrate the enhanced regenerative capacity of this strategy.

Methods: Patients that have undergone LVAD implant for previous AMI, and are stable 2 months post LVAD, will have both a programmable and refillable micro infusion pump implanted subcutaneously in the abdomen and a chronic pacing lead inserted into an area of infarct-related myocardial scar. Previous LVAD patients followed at the UTAH Recovery program will serve as controls. The patients will then be randomized to either bioelectric stimulations alone, continuous infusion of a Pro-Regenerative composition of 10 proteins including SDF, EGF, VEGF, follistatin, and tropoelastin A,B) alone, or the combination. Patients will be followed with the weaning protocol now in place at U Utah to assess myocardial recovery including turn down studies of decreasing duration of LVAD support, and functional and cardiac function by echocardiography.

Conclusion: This study will determine if bioelectric stimulation coupled with continuous, direct infusion of a pro-regenerative composition into myocardial infarct induced scar in chronically infarcted swine is feasible, safe, and induces a significant improvement in cardiac function, reduction in scar tissue, and enhanced vasculogenesis.
Abstract Poster Presentations
Thursday: Lunch Break

Characteristics and management of patients presenting with chest pain to hospital emergency departments in the United States
Presenter: Ragheed Al-Dulaimi, MD, MSc, MPH
Presenter Institution: University of Utah, Salt Lake City, Utah

Describe the demographics and clinical characteristics of patients presenting with chest pain to Emergency departments in the United States as well as the management they received.

Introduction
Acute chest pain is a common presentation in Emergency room and outpatient setting. It can be due to wide spectrum of medical conditions ranging from benign self-limiting illness to life threatening conditions as Myocardial infarction. Patient history and physical examination are helpful, but further evaluation of patients with chest pain with EKG, Cardiac enzymes, or other diagnostic tests might be needed to help reach the final diagnosis.

Methods
Using data from the National Hospital Ambulatory medical Care Survey (NHAMCS) 2010. We perform retrospective cross sectional analysis to describe the prevalence of chest pain, patients’ characteristics as well as the Investigations and management they received in the Emergency departments (ED) across the United States. Visits were identified based on the reason of patient visit to the ED. We also examined the differences in the outcomes (admitted, discharged, died in the ED) by demographics and insurance coverage. We adjust for the complex study design of the survey data in our analysis. The analysis was performed using SAS 9.4 (Carey, NC).

Results
5.3% of visits to the EDs are due to chest pain [Total of 2369412 visits]. The prevalence varied by age, being the highest in patient aged 45-64 (9.3%). Prevalence was higher in males (5.8%) as compared to female (4.8%). While 7.8% had CHF, 13.3% had diabetes. 27.9% of patients with chest pain arrived by ambulance, 57% had cardiac enzymes, and 82.5% had EKG. While 33% admitted to the hospital, 4.1% of them had coronary catheterization. Less than 1% died in the ED. The final diagnosis at hospital or ED discharge were cardiac (26.4%) [70% of were CAD]. However the majority of chest pain were coded as unspecified or other chest pain (42.1%). 3% of patients presented with chest pain were seen in the ED in the last 72 hours. 56.3% reported visiting the ED at least once in the past 12 month (Mean 3 visit/12 month).

Conclusion
Chest pain is a frequent cause of ED visit in the US. It is more common among male, aged 45-64 and those with other chronic illness. Large percent of chest pain went undiagnosed. In addition, significant percent of patients with chest pain had multiple visits to the ED.
New Insights into Symptom Burdens of Advanced Heart Failure Patients with HIV Infection
Presenter: Mahesh Chandrasekhar, MD
Presenter Institution: Georgetown University Hospital/Medstar Washington Hospital Center

Chandrasekhar MA, Ruiz G, Walker K, Groninger H.

Background:
Patients with the human immunodeficiency virus (HIV) carry a high risk of cardiovascular disease, and symptom burden. In an era where patients with HIV who require advanced heart failure (AHF) therapies may be considered candidates, we seek to characterize symptom burden of AHF patients with HIV via a case series of 5 HIV patients being evaluated by an AHF team and an embedded palliative care (PC) service in comparison to a 221 patient consult evaluation.

Methods:
A retrospective chart review was performed on all advanced heart failure patients, receiving PC consultation from January-December 2015. Data collected from PC assessments included relevant clinical issues, reason for PC consultation, prognosis, disposition, and symptom burden at baseline and 48 hours (using Edmonton Symptom Assessment Scale). Descriptive statistics were used to analyze the data including types of PC interventions, length of stay, and time to consultation both alone, and in comparison with non HIV-matched cohorts.

Results:
221 PC consults were performed for AHF inpatients, of which 5 patients were infected with HIV. The most frequent reason in HIV patients was for goals of care discussion (100%), followed by pain control (60%), whereas in no HIV AHF patients, consult questions were for pain (38%), non-pain symptoms (23%), and establishing goals of care (56%). In one case, consultation was because a patient was not a candidate for AHF therapies. Sixty percent of patients had pain, and 80% of patients had dyspnea on presentation. Interventions from the PC team included symptom management recommendations (100%), psychological (80%) and spiritual (60%) counseling. Patients without HIV went home (52%) or to rehab (6%) on discharge with only 11% dying in the hospital and 11% being discharged with hospice services. By comparison, 40% of patients went home, with 20% of patients going to long term care, compared to the 58% of patients without HIV who returned home. One patient in the case series was discharged with hospice (20%), and one patient died while in-patient (20%).

Conclusions:
Both advanced heart failure and HIV are diseases known for poor quality of life, yet little literature exists in this unique population. While there are limitations in this study secondary to population, through the needs for symptom management, psychosocial intervention, and disposition to hospice/inpatient death this case series illuminates burdens in HIV patients with AHF.
Effects of Mindfulness Based Stress Reduction on Functional Mobility and Quality of Life in Patients Post Left Ventricular Assist Device Implantation (MindVAD)
Presenter: Jennifer Chung-Peck, DPT, ATC
Presenter Institution: University of Utah, Salt Lake City, UT

Background & Purpose: The purpose of this study was to evaluate the feasibility of implementing a complementary therapeutic intervention such as Mindfulness Based Stress Reduction (MBSR) and evaluate the effects of intervention on functional outcomes and quality of life in patients with destination left ventricular assist device implantation (LVAD). Mindfulness-Based Stress Reduction (MBSR) is a structured complementary medicine program that uses mindfulness in an approach that focuses on alleviating pain and on improving physical and emotional well-being for individuals suffering from a variety of diseases and disorders.

Case Description: A single subject design was used in the care of a 54 year old male who was hospitalized for heart failure and required LVAD implantation. The standardized questionnaire used was the Minnesota Living with Heart Failure Questionnaire (MLHFQ) which was used to measure the effects of heart failure and treatments for heart failure on an individual’s quality of life. The Functional Independence Measure (FIM) scale was used assess physical and cognitive disability. Due to the acute nature of illness and high acuity, modifications from MBSR included: briefer meditation periods daily (15 to 20 minutes), emphasis on seated meditations rather than mindful movements or walking meditation (due to physical limitations); and no daylong retreat. The participant was evaluated using MLHFQ and FIM scales at weeks 2, 4, 8 and 12 post-operative LVAD implantation.

Outcomes: MLHFQ and FIM scores were positive correlated throughout the study with 50% improvement in MLHFQ and 75% improvement in FIM scores at the end of the 12 weeks.

Discussion: Since this was not a controlled trial, results are not known as to the comparison of patients receiving MBSR intervention and physical therapy and those with standard physical therapy intervention alone. The results warrant further research in the use of alternative therapies such as MBSR in adjunct with physical therapy intervention in a randomized controlled study to further study the effects on quality of life and functional outcomes in patients post LVAD implantation.

References: Limit to only those materials that ensure that the content is evidence-based; minimum 5 references, no more than 10 years old.

Exercise Pressor and Muscle Metaboreflex Responses in Heart Failure Patients with Preserved Ejection Fraction

Presenter: Heather L. Clifton, PhD
Presenter Institution: University of Utah, VA Medical Center, Salt Lake City, UT

Heather L. Clifton1,2, Stephen M. Ratchford1,2, Jennifer R. Vranish5, Joel D. Trinity1,2,3, John J. Ryan2,3, Russell S. Richardson1,2,3,4, Paul J. Fadel5, Satyam Sarma6,7, Mark J. Haykowsky8, and D. Walter Wray1,2,3,4

Author(s) Institution:
1Geriatric Research, Education, and Clinical Center, SLC VAMC, UT; 2Department of Internal Medicine, University of Utah, SLC, UT; 3University of Utah Center on Aging, SLC, UT; 4Department of Nutrition and Integrative Physiology, University of Utah, SLC, UT; 5Department of Kinesiology, University of Texas at Arlington, Arlington, TX; 6Department of Internal Medicine, University of Texas Southwestern Medical Branch, Dallas, TX; 7Institute for Exercise and Environmental Medicine, Texas Health Presbyterian Hospital, Dallas, TX; 8College of Nursing and Health Innovation, University of Texas at Arlington, TX

Background: Exercise intolerance is the primary chronic symptom in heart failure patients with preserved ejection fraction (HFpEF), and often persists even with optimized treatment. Though there are many mechanisms that may contribute to this deficit, disease-related changes in the muscle metaboreflex may play a role. Indeed, it has been previously shown that in heart failure with reduced ejection fraction (HFrEF), input from group III/IV muscle afferents contributes to the development of fatigue and exercise intolerance. Therefore, the purpose of this study was to examine metaboreflex modulation of heart rate (HR) and mean arterial pressure (MAP) in HFpEF patients.

Methods: Eight HFpEF patients (6M, 2F; age 67 ± 4 yrs; BMI 35 ± 3 kg/m2; ejection fraction 63 ± 3%) and seven healthy controls (4M, 3F; age 59 ± 3 yrs; BMI 30 ± 1 kg/m2) participated in this study. Patients were studied under their normal pharmacotherapy. After 30 min of supine rest, participants performed 2 min of isometric handgrip exercise at 30% and 40% of maximal voluntary contraction (MVC), followed by 2 min of post-exercise circulatory occlusion (PECO) to isolate the muscle metaboreflex response. HR (3-lead ECG) and arterial blood pressure (Finapres Medical Systems BV) were measured continuously, and the last 30 sec of exercise and PECO was averaged to determine peak changes in HR and MAP.

Results: Baseline HR and MAP were not different between HFpEF and controls (70 ± 7 vs. 68 ± 5 bpm; 97 ± 4 vs. 103 ± 5 mmHg, respectively). At 30% MVC, comparable ∆HR were observed between groups during handgrip (6 ± 1 vs. 4 ± 2 bpm, HFpEF vs. control) and PECO (2 ± 1 vs. 2 ± 1 bpm, HFpEF vs. control). While ∆MAP was similar between groups during handgrip (15 ± 2 vs. 14 ± 3 mmHg, HFpEF vs. control), HFpEF patients exhibited a greater ∆MAP during PECO (16 ± 2 vs. 8 ± 3 mmHg, HFpEF vs. control, p<0.05). At 40% MVC, ∆HR was not significantly different between HFpEF and controls during handgrip (7 ± 3 vs. 12 ± 2 bpm, respectively) or PECO (1 ± 1 vs. 1 ± 2 bpm, respectively). Similarly, ∆MAP at 40% MVC was comparable between the groups during handgrip (24 ± 5 vs. 24 ± 2 mmHg, HFpEF vs. control), whereas there was a trend towards a greater ∆MAP during PECO in the HFpEF patients (24 ± 5 vs. 16 ± 3 mmHg, p=0.16).

Conclusions: While the overall pressor response during handgrip was comparable between groups, a notable elevation in blood pressure was observed during PECO in the HFpEF patients. It is important to note that these differences occurred with the patients maintaining their normal pharmacotherapy, which often includes antihypertensive agents. These preliminary findings suggest that the muscle metaboreflex is exaggerated in patients with HFpEF, and this may be involved in the exercise intolerance observed in this patient population.
Early weight loss independent effects of sleeve gastrectomy on diet-induced cardiac dysfunction in obese, Wistar rats.
Presenter: Tammy L. Kindel, MD
Presenter Institution: Medical College of Wisconsin

Tammy L. Kindel, MD, PhD*1, Tom Foster, BS1, Paul Goldspink, PhD2, Steven J. Kindel, MD3, John Corbett, PhD4, Michael Widlanksy, MD5, and Jennifer Strande, MD, PhD5.

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Introduction. Several reports suggest that bariatric surgery significantly improves both cardiac function and symptoms of heart failure in 50% of patients with cardiomyopathy. Improvements in cardiac geometry and function occur post-surgically yet the mechanism is unknown but may be due to both weight-loss dependent and independent factors. We predict that the changes in gastrointestinal anatomy after a sleeve gastrectomy (SG) will have weight-loss independent effects on cardiac dysfunction in the setting of obesity. To test this hypothesis, we performed SG in rats with diet-induced obesity and cardiac dysfunction and assessed for weight-loss independent improvements in cardiac function compared to pair-fed (PF) sham rats.

Methodology. Cardiac dysfunction was induced by feeding a 60% kcal from fat diet to male Wistar rats for 10 weeks. Rats were matched by cardiac function and randomized to undergo either a SG (n=12) or PF (n=8) sham surgery. Trans-thoracic echocardiograms (TTE) were performed pre-operatively, and post-operatively at 6 and 13 weeks. Fasting serum samples were obtained at 10 weeks post-operatively for the measurement of fasting glucose, insulin, the quantitative insulin-sensitivity check index (QUICKI), as well as B-type natriuretic peptide (BNP).

Results. Ten weeks of high-fat feeding induced a significant increase in body weight with a significant reduction in EF (85.0 ± 4.3%) by TTE compared to a low-fat diet in age-matched controls (89.4 ± 3.6%, p=0.03). At 10 weeks post-operatively, plasma glucose and BNP levels were significantly lower in SG rats compared to PF rats (BNP: 915 ± 467 pg/mL vs. 1694 ± 959 pg/mL, p=0.05) without significant differences in fasting insulin or QUICKI between the two groups. There was no significant difference in body weight or any functional parameter by TTE between SG or PF rats at 13 weeks post-op. However, a significantly greater percent of SG rats (44%, 4 of 9) had a normal EF at 13 weeks compared to 0% of the PF rats (n=8, p=0.03). Recovered SG rats had a smaller left ventricular internal diameter in systole and end systolic volume with significantly improved systolic function by measurement of EF compared to non-recovered SG rats (EF: 90.7 ± 1.7% vs. 75.4 ± 3.6%, p<0.001).

Conclusions. A SG in Wistar rats with diet-induced obesity and cardiac dysfunction significantly improved systolic function in 44% of rats independent of weight loss at 13 weeks after surgery. This improvement is directly related to weight-loss independent effects of the surgery on the entero-cardiac axis. These results offer a novel metabolic role for bariatric surgery as a treatment modality for obesity cardiomyopathy.
Cardiac remodeling in patients with heart failure undergoing endovascular aortic aneurysm repair
Presenter: Dimitrios Koudoumas, MD, PhD
Presenter Institution: SUNY at Buffalo, Buffalo, NY

Dimitrios Koudoumas MD PhD¹, Mariel Rivero MD², Linda Harris MD¹, Maciej Dryjski MD¹, Hasan H. Dosluoglu MD²

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Objectives: The incidence of abdominal aortic aneurysm pathology with concomitant cardiac dysfunction has been reported as high as 24%. We sought to identify the effect of endovascular aortic aneurysm repair (EVAR) on cardiac systolic and diastolic function in patients with pre-existing heart failure.

Methods: A retrospective chart review was performed in all patients undergoing EVAR with concomitant heart failure between 2004 and 2015. Patient demographics, preoperative and postoperative – before and after 6 months following EVAR - echocardiograms were reviewed.

Results: A total of 29 consecutive patients were included in the study. All patients were male 29/29 (100%). Ischemic cardiomyopathy as the cause of heart failure was present in 25/29 of patients (86.2%) and non-ischemic/idiopathic in 4/29 (13.8%). Mean age was 71.4 ± 9 years, BSA 2.04 ± 0.18 m2, BMI 30 ± 3.9 and main graft body diameter was 30 ± 3.9mm. Mean NYHA class postoperatively was 1.92 ± 0.81 vs. 1.84 ± 0.68 (p=ns) preoperatively. Left ventricular ejection fraction (LVEF) was 30 ± 8.34% vs. 37 ± 8.41% (p=.015), left ventricular end diastolic diameter (LVEDD) was 59.5 ± 9.32mm vs. 54.3 ± 7.8 mm (p=ns), left ventricular end systolic diameter (LVESD) was 47.4 ± 9.7mm vs. 40.9 ± 7.8mm (p=ns), posterior wall (PW) was 11.6 ± 2.1mm vs. 10.2 ± 2.42mm (p=ns), interventricular septum (IVS) was 13.4 ± 2.1mm vs. 12.3 ± 2.5mm (p=ns) and left atrial diameter (LA) was 45.3 ± 2mm vs. 45.5 ± 8.9mm (p=ns) before and after the 6-month time frame following EVAR. Diastolic function measured via E/A was 1.84 ± 1.1 vs. 1.35 ± 0.75 (p= .013) before and after 6 months from endograft implantation. There was a positive correlation between postoperative EF and main graft body diameter with a correlation coefficient of 0.39 (p= .03) and a negative correlation between postoperative LVESD and main graft body diameter with a correlation coefficient of -0.44 (p= .04)

Conclusions: In this retrospective study EVAR was associated with an initial myocardial remodeling which appeared to be sustained up to 6 months following the endograft implantation. Due to retrospective nature of the study along with the small number of patients further studies are necessary.
Rapid histochemical diagnosis cardiovascular tissue: can it impact clinical course of mechanical support and assess myocardial recovery?

Presenter: Jai Raman, MD, FRACS, PhD
Presenters Institution: Oregon Health & Science University

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Objective:
Dignoses in Cardiovascular medicine are limited by the need for excisional biopsies, careful preparation of tissue and multiple stains, which are time-consuming and subject to interpretation. This has limited the use of histopathological evaluation in the heart. We report on the results of a novel rapid stainless technique using Computational Chemical Imaging (CCI) with spectroscopy to provide automated histopathologic diagnoses.

Methods:
Over 50 samples of endomyocardial and open cardiac biopsy specimens were studied using computational chemical imaging (CCI) and microscopy using a combination of Fourier Transform Infra-Red (FTIR) and Discrete Frequency Infra-Red (DFIR) spectroscopy. Samples were deparaffinized and washed off to remove formalin. An early group of specimens was used as a training set, which utilized spectral characteristics and computation to help build a complex chemical map that replicated histological characteristics of each cell type. Over 2000 frequencies were screened to identify the appropriate spectra for cardiac tissue. Proof of concept was developed using cardiac transplant rejection and infarction in myocardial tissue biopsies. The existing reported histopathological diagnoses were used as the standard to compare outcomes with CCI. Scan times for tissue characterization using CCI were also recorded.

Results:
Scan times went from 90 minutes per sample to less than 5 minutes over the 3 years of the study. At a sensitivity of over 90 %, CCI demonstrated a specificity of over 95% for all cell types in the myocardium including small lymphocytes. The most consistent findings with CCI related to collagen deposition as a marker of fibrosis in the interstitium and the presence of intra-cellular glycogen in ischemic cells.

Scan times for tissue assessment are approaching less than a minute and are on-track to make the transformation into live tissue imaging.

Conclusions:
Computational Chemical Imaging using Computation along with Discrete Frequency and Fourier-Transform Infra-red spectroscopy methods can reliably provide automated diagnostic histopathology of cardiac tissue rapidly at the point of care. The evolution of this technology is on track to make live cardiac tissue assessment a reality. This may facilitate quick assessment of myocardial function and/or recovery during mechanical support.
Validation of a Novel Experimental Blood Pressure Monitor for Heart Failure Patients Supported with Continuous-flow LVADs (Left Ventricular Assist Device)

Presenter: Pavol Sajgalik, MD
Presenter Institution: Mayo Clinic, Rochester, MN

Pavol Sajgalik1, Vaclav Kremen2, Vratislav Fabian3, Sudhir S Kushwaha1, Simon Maltais4, John M Stulak4, David L Joyce4, Lyle D Joyce4, Bruce D Johnson1 and John A Schirger1

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Introduction: Continuous-flow (CF) LVAD therapy, an established treatment modality for advanced heart failure (HF), is experiencing exponential growth due to increased durability and progressive engineering of the pumps.1–3 While in a non-LVAD population the arterial blood pressure (BP) is easily obtained by auscultation or oscillometric method, in the CF LVAD patients accurate BP assessment remains challenging due to a reduced pulse pressure. Despite advancements in LVAD technology, a specific LVAD BP monitor is not currently available. Traditional automated oscillometric BP monitors are capable of successfully measuring BP in approximately 55-60% of cases, while manual auscultation allows BP assessment in less than 20% of LVAD measurements.4 Currently clinical management of LVAD patients relies on a Doppler BP method, which significantly overestimates mean arterial pressure.5 Given that one of the major adverse events of LVAD therapy – intracranial bleeding – is associated with poor BP control,6,7 the measurement error and limited availability of Doppler-derived BP could delay proper therapeutic strategy and negatively influence clinical outcomes. Accordingly, we studied the validity and measurement “success rate” of a novel, non-invasive experimental blood pressure (expBP) monitor compared with intra-arterial (I-A) BP. Secondly, we compared the precision of the non-invasive “gold standard” Doppler technique to the I-A BP on the same population.

Methods: Patients on CF LVAD therapy supported either with HeartMate (HM) II, HeartMate III or HeartWare (HW) devices were prospectively studied. The BP was assessed in a stable, supine position shortly after the surgery in the intensive care unit. Invasive (I-A) BP was obtained via radial artery, followed by BP assessed with our brachial single-cuff LVAD monitor and the Doppler technique. Prior to recording the I-A BP, the arterial line was flushed and leveled to the pressure transducer (IntelliVue, Phillips, the Netherlands). An adapted oscillometric principle was used in our expBP monitor to more accurately detect the unique BP characteristics of CF LVAD patients. Doppler ultrasound measures were obtained using a calibrated sphygmomanometer as previously described.8 For each separate technique, measures were done in triplicate and the average was used for subsequent analyses. Variables were summarized as mean (standard deviation) and frequency (percent) for continuous and categorical measurements, respectively. Bland-Altman (BA) plots were constructed for comparisons of measurement agreement between experimental BP monitor, Doppler and I-A BP values, and the Bias + 95 % confidence intervals were derived. Pearson correlation coefficients were also analyzed. Data was analyzed using JMP Pro 10 statistical software package.

Results: A total of 34 patients (7 females; Age 63±10 years; BMI 27.7±7.4 kg.m2) were tested 3.7±8.4 days post LVAD implantation (18 HM II pumps, 3 HM III and 13 HW pumps). Although BP was successfully assessed in all tested patients by the expBP monitor (characterized by at least one successful BP reading per subject), the overall success rate reached 94% (6 failed reading from a total of 102 measurements). The Doppler achieved a 100% success rate. Compared to I-A measurements, the expBP monitor overestimated mean arterial pressure (MAP) by 0.9 [-7.9+9.7] mmHg, while Doppler method overestimated MAP by 5.5 [-7.8+18.9]
mmHg. Correlation between MAP obtained by the expBP monitor and I-A MAP was r=0.89 (p<0.001), while correlation between Doppler MAP and the I-A MAP was r=0.74 (p<0.001). BA plots revealed compared to the I-A systolic pressure that the expBP monitor overestimated BP by 1.0 [-12.1+10.0] mmHg and compared to the I-A diastolic BP, the expBP monitor underestimated BP by 1.4 [-12.2+15.0] mmHg. Summary of findings is depicted in Figure 1.

**Conclusion:** Our results demonstrate closer agreement of mean arterial BP assessed by a novel brachial cuff LVAD expBP monitor prototype than a “gold standard” Doppler technique as compared to the invasively obtained BP. With a satisfactory “success rate” of measurement attempts in challenging hemodynamic conditions of LVAD patients shortly after the surgery, the novel expBP monitor also provides systolic and diastolic BP compared to a single BP value delivered by the Doppler technique. Translation of the LVAD expBP monitor prototype into clinical practice could potentially lead to simplification of BP monitoring, allowing for potentially improved BP control in the LVAD population, and in turn this could potentially positively impact adverse events rates associated with a poor BP control. Further evaluation of these concepts with this technology is warranted.

**Figure 1. Means of BP Values Assessed Invasively, via a novel LVAD BP Monitor and by the Doppler Technique**

![BP Values Chart]

**Literature:**


**Altered mitochondrial calcium signaling in a mitochondrial cardiomyopathy model**

Presenter: Salah Sommakia, PhD  
Presenter Institution: University of Utah, Salt Lake City, UT

Salah Sommakia, Dipayan Chaudhuri  
University of Utah CVRTI

Mitochondrial diseases often feature the early onset of dilated cardiomyopathy, due to the large energetic demand placed by the heart. We sought to investigate whether these failing hearts alter mitochondrial calcium levels to compensate for reduced energy production, since calcium is a potent stimulator of ATP synthesis. Discovery of such mechanisms offers not just further insight into the pathophysiology of congenital mitochondrial diseases, but may reveal targets for heart failure caused by other insults, since mitochondrial dysfunction is a signal event marking reduced viability in most cardiomyopathies. To this purpose, we use a mouse model of heart failure generated through the cardiac-specific knockout (KO) of TFAM (mitochondrial transcription factor A), a gene essential for transcription of mitochondrial DNA. Such TFAM KO mice have deficient ATP synthesis and early-onset dilated cardiomyopathy. We show that, as heart failure develops during the first two postnatal weeks, these mice feature 30-60% reductions in complex I, III, and IV activity, as these complexes have critical subunits encoded by mitochondrial DNA. Such dysfunction inhibits the inner mitochondrial membrane voltage gradient driving ATP synthesis by 25% by 3 weeks of life. In these failing hearts, we find that mitochondria do upregulate their calcium uptake. Using a calcium-sensitive fluorophore (Oregon Green), we determined that KO mitochondria take up calcium at twice the rate of WT mitochondria via enhanced activity of the mitochondrial calcium uniporter (MCU), the main pathway for calcium entry. Additionally, KO mitochondria release calcium at half the rate of WT mitochondria, indicating a reduced activity of the sodium-calcium exchanger, the main pathway for calcium export. Oxygen consumption assays revealed a 50% reduction in respiration of KO mitochondria compared to WT in the absence of calcium. However, respiration in KO mitochondria can be boosted substantially by incubating with calcium, almost to wild-type levels. Our results suggest that TFAM KO mitochondria upregulate calcium signaling pathway as a compensatory mechanism against respiratory deficiencies, and identifying the mechanism may reveal attractive new targets for treating heart failure.
Utilization and outcomes of temporary mechanical circulatory support following heart transplantation
Presenter: Vakhtang Tchantchaleishvili,
Presenter Institution: Mayo Clinic and Foundation, Rochester, MN

Vakhtang Tchantchaleishvili, Kevin Phan, Jessica G.Y. Luc, Joshua Xu, Vakhtang Tchantchaleishvili, Simon Maltais, Tristan D. Yan

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BACKGROUND: Graft dysfunction is the main cause of early mortality following heart transplantation. In cases of severe graft dysfunction, temporary mechanical circulatory support (TMCS) may be necessary. The aim of this systematic review was to examine the utilization and outcomes of TMCS in patients with graft dysfunction after heart transplantation.

METHODS: Electronic search was performed to identify all studies in English literature assessing the use of TMCS for graft dysfunction. All identified articles were systematically assessed for inclusion and exclusion criteria.

RESULTS: Of the 5462 studies identified, 41 studies were included. Among the 11555 patients undergoing heart transplantation, 832 (7.3%) required TMCS with patients most often supported using VA-ECMO (79.4%) followed by RVAD (11.1%), BiVAD (7.5%) and LVAD (2.0%). Patients supported by LVADs were more likely to be supported longer (P=0.003) with a higher death by cardiac event (P=0.013) and transplantation rate (P=0.015), while patients supported with BiVADs were more likely to be weaned (P=0.020).

CONCLUSIONS: TMCS is an effective modality to support patients with graft dysfunction after heart transplantation. Further studies are needed to establish the optimal threshold and strategy for TMCS and to augment cardiac recovery and long-term survival.
Cardio-oncology outpatient units may improve outcome in patients with cardiac amyloidosis

Presenter: Helen Tseliou
Presenter Institution: University of Athens, Greece


Department of Therapeutics, University of Athens, Greece

Aim and background: Cardiac amyloidosis (CA) is associated with poor outcome and prognosis. The aim of the current study was to evaluate the impact of an organized cardio-oncology outpatient unit (C-OOU) on one year mortality and morbidity rates in patients with CA.

Methods: Sixteen consecutive patients with CA referred from the oncologists to our outpatient C-OOU were prospectively included and followed for 1-year. This special infrastructure has been established to deliver better quality of healthcare in oncology patients and comprises of 2 heart failure (HF) specialists, one interventional cardiologist, one electrophysiologist, two heart echo specialists and 7 cardiology fellows. 97.5% of the studied patients were diagnosed with AL amyloidosis. Clinical, echocardiographic and laboratory data were monitored and follow-up visits were scheduled accordingly. All patients received chemotherapeutic agents according to current guidelines and recommendations. One year survival, hospitalization rates and major cardiovascular events were recorded for all the study population.

Results: At baseline, 31% of the patients were at NYHA III and 18% were at NYHA II. Mean Ejection Fraction (EF) was 49.5±9%, intraventricular septum thickness 14.7±3mm, posterior wall thickness was 13.8±4mm and Nt-proBNP was 4611±750pg/ml. Surprisingly high one year survival rates were observed for our study population. Only two patients deceased at the end of the study period. One due to sudden death and another one due to viral lower respiratory tract infection.

Patients with reduced EF (<50%, n=7) were more frequently seen in the outpatient clinic as compared to patients with preserved EF (7 vs 4 visits per year). Two hospitalizations occurred in the group of patients with reduced EF. One for transient ischemic attack and one for HF decompensation. One patient was admitted to the hospital due to HF decompensation in group with the preserved EF. Two patients in each group required high doses of diuretics to control better their HF symptoms.

Conclusions: Close monitoring, evaluation and treatment of patients with cardiac amyloidosis by a special cardio-oncology unit improves significantly the outcome and prognosis of this malignant disease.
Exercise to Enhance Myocardial Recovery via Ambulatory Micro-Axial Flow Pump in a Case of Post-Cardiotomy Shock
Presenter: Gregory Valania, DO
Presenter Institution: Baystate Health, Springfield, MA

Gregory Valania, DO, Mara Slawsky, MD, Josephine Chou, MD, Sotiris Stamou, MD, Dan Engelman, MD, Ali Haider, MD, Evan Lau, MD, Jaime A. Hernandez-Montfort, MD, MPH

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Introduction: Transitions in care towards myocardial recovery in patient supported with temporary left ventricular micro-axial flow pumps are poorly described in the literature. We present a methodology for assessing early myocardial recovery (defined as neurohormonal tolerance off support with no dyspnea at rest or minimal exertion) while on temporary MCS via Impella 5.0 with a case presentation in which clinical, hemodynamic and echocardiography in addition to a component of activity with a stationary bicycle are considered into daily bedside clinical decision making.

Methods: Case Presentation.

Case Presentation: 52 year old previously healthy gentleman admitted with de novo ACS/NSTEMI related acute heart failure syndrome c/w cardiogenic shock and large LV thrombus in the setting of significant multivessel disease underwent emergent CABG x 3 and L apical ventriculectomy with thrombus removal under 40 cc IABP. Postoperative course was complicated with severe refractory cardiogenic/hemocirculatory shock (3 vasoactives, IABP, renal/liver injury and lactatemia) in POD 4 which was followed by Impella CP via femoral approach with partial improvement however hemodynamic evidence of left ventricular load (PCWP > 22, CI 1.9-2.1 on inotropes/CP P8) prompting transition to R Axillary Impella 5.0 as bridge to decision that after 2 weeks exhibited evidence of partial myocardial recovery which led to explantation after a weaning strategy was performed. Ensuring adequate anticoagulation a bedside ECHO/hemodynamic weaning trial was performed at rest with the following baseline hemodynamics on Impella 5.0 P4 to P2: HR SR 74, BP 82/57 (63), O₂ 100% , RA 2 PA 37/10 (21) MV02 54% which did not change with exercise utilizing a stationary bicycle. The device was explanted successfully and patient was discharged home with LVOT VTI 16 cm/sec and adequate neurohormonal therapy: spironolactone, digoxin and valsartan.

Conclusions: Clinical strategies that aid in transitioning patients in shock supported with temporary MCS towards myocardial recovery is a potentially evolving field in which further research is warranted.
Impacts of HFpEF on the Development of Neuromuscular Fatigue during Exercise
Presenter: Joshua Weavil, MS
Presenter Institution: University of Utah, Salt Lake City, UT

JC Weavil, TS Thurston, TJ Hureau, JR Gifford, AD Bledsoe, JN Nativi, RS Richardson, M Amann
University of Utah

BACKGROUND: Patients with heart failure with a preserved (≥50%) ejection fraction (HFpEF) suffer from severe exercise intolerance. Although the exact mechanisms accounting for this impairment are not fully understood, an exaggerated development of neuromuscular fatigue during physical activity may play a critical role.

PURPOSE: To examine the influence of HFpEF on the development of neuromuscular fatigue during exercise characterized by a small muscle mass and, subsequently, minimal central hemodynamic (i.e. cardiac output) constraints.

METHODS: Seven HFpEF (64 ± 1 yr) and 7 age-matched controls (CON) performed dynamic single-leg knee extensor exercise (KE) to exhaustion at a given relative intensity (80% Wpeak; DYN), and intermittent maximal isometric knee extensions (8×15 s, 20 s rest, ISO). CON repeated DYN at the same absolute work rate and same duration as HFpEF. Quadriceps fatigue was quantified as pre- to post-exercise decrease in maximum voluntary contraction torque (MVC). Peripheral and central fatigue were quantified via pre- to post-exercise changes in quadriceps twitch torque (Qtw; electrical femoral nerve stimulation) and voluntary quadriceps activation (VA). Maximal rate of torque development (MRTD) and peak relaxation rate (PRR) were derived from the Qtw kinetics.

RESULTS: DYN. Wpeak was higher in CON compared to HFpEF (53 ± 2 W vs 31 ± 3 W; P < 0.01). When the exercise was matched in terms of absolute work rate (24 ± 2 W) and duration (10 ± 1 min), patients with HFpEF demonstrated a greater exercise-induced reduction in MVC (-30 ± 4% vs -6 ± 4%) and QTW (-54 ± 6% vs -11 ± 4%) compared to CON (all P < 0.05); VA was not affected in either group. At the same relative intensity (80% Wpeak, corresponding to 24 ± 2 W and 43 ± 2 W in HFpEF and CON, respectively), task failure occurred at a comparable time (~10 min, P = 0.53) and both groups demonstrated a similar exercise-induced reduction in MVC (~27%) and QTW (~50%) (P > 0.4). However, compared to CON, HFpEF exhibited a significantly greater exercise-induced reduction in MRTD (-67 ± 5% vs -47 ± 6%) and PRR (-61 ± 5% vs -37 ± 4%). VA fell significantly in CON (-6 ± 2%), but remained unaltered in HFpEF (P = 0.23).
ISO. Quadriceps MVC was similar between groups at baseline (~160 Nm; P = 0.68). Although the exercise did not affect VA in either group (P > 0.12), HFpEF demonstrated a greater exercise-induced reduction in MVC (-26 ± 4% vs -13 ± 4%), QTW (-49 ± 4% vs -23 ± 7%), MRTD (-53 ± 6% vs -20 ± 9%), and PRR (-51 ± 7% vs -26 ± 7%) (all P < 0.05) compared to CON.

CONCLUSION: During physical activity characterized by minimal central hemodynamic constraint, HFpEF patients exhibit a compromised functional capacity compared to CON and an exacerbated development of neuromuscular fatigue during exercise at a given absolute workload. Furthermore, although the development of fatigue is similar between HFpEF and CON during dynamic small muscle mass exercise performed at the same relative intensity, it is critical to emphasize that the absolute workload at a given relative intensity is substantially lower in the patients. This finding further highlights the greater susceptibility to fatigue in this population. Consequently, targeting abnormalities within the skeletal muscle may be a priority in improving exercise tolerance in HFpEF.
EARLY MOBILITY FOR CRITICALLY ILL PATIENTS IN THE CARDIOVASCULAR INTENSIVE CARE UNIT DECREASES ACUTE LENGTH OF STAY

Presenter: Haley Yolken, PT and Bryan Lohse, PT
Presenter Institution: University of Utah, Salt Lake City, UT

Lohse, Bryan D¹; Johnson, Joshua K³; Yolken, Haley A¹; Noren, Christopher S¹; Marcus, Robin ³; Tonna, Joseph E²

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Purpose/Hypothesis: Early mobility in the intensive care unit (ICU) has been shown to be feasible, but challenges to implementation exist for critically ill patients where medical stability has been the priority and safety is a concern. Additionally, resources to provide physical therapy (PT) treatment are often limited. Recognizing these challenges, we instituted a quality improvement (QI) project on the cardiovascular intensive care unit (CVICU) to increase therapy staff in order to increase therapy dosing for appropriate patients. The purpose of this report is to describe the process and effect of increasing early mobility interventions for critically ill patients in the CVICU. We hypothesized that increasing therapy staff would lead to increased therapy time per treatment session with a subsequent improvement in patient mobility and decrease in length of stay.

Number of Subjects: The QI period (September 2015-March 2016) included 29 patients with 26 patients in the season-matched baseline period one year prior.

Materials/Methods: With multidisciplinary support, PT staff developed and implemented patient-specific treatment approaches and increased its number of dedicated PTs on the CVICU from 2 to 4. For the purpose of this research, critically ill patients were defined as those with a CVICU length of stay (LOS) ≥ 10 days. Treatment time was reviewed retrospectively. Additionally, LOS and mobility outcomes using the Activity Measure for Post-Acute Care “6-Clicks” (AM-PAC) were analyzed. All variables were analyzed for a six-month period and compared to the baseline period using one-sided t-tests.

Results: Mean PT treatment time per session increased from 44.3 minutes in the baseline period to 50.4 minutes in the QI period (p=0.04). Mean CVICU LOS did not change during the QI period (20.3 days) compared to baseline (19.6 days; p=0.58), but mean post-ICU LOS was reduced from 6.02 days to 3.90 days in the QI period (p=0.02). Patient mobility improvement (raw AM-PAC score) in the ICU was greater in the QI period (1.4 points) compared to baseline (0.7), but did not achieve statistical significance (p=0.29).

Conclusions: This data demonstrates feasibility of increased therapy dosing in the critically ill population and suggests a relationship with functional outcomes and length of stay. Furthermore, a non-significant improvement in mobility for patients in this QI process was also observed. Considering the complex medical status of the patients studied (CVICU LOS > 10 days), this finding is not surprising as mobility changes in this population are expected to be highly variable. These results warrant further investigation.

Clinical Relevance: In a healthcare system focused on value, this data shows more PT treatment time, rather than less, leads to improved patient outcomes. This data shows that the critically ill population can tolerate increased therapy dosing and suggests a relationship with functional outcomes. Future research should determine the financial impact of this care model and further examine the relationship between increased therapy and functional outcomes in this population.