right heart catheterization.

Results: TNFα and IL-6 were detected in 6 and 6 patients in CS respectively with absence in PV. IL-6 and TNFαR1, 2 levels in PV was significantly correlated with that of CS (r=0.92, 1.0, 0.1, p<0.001). The level of IL-6 and TNFαR1, 2 in CS have positive correlations with the severity of decompensation (r=0.47, 0.49, 0.52, p<0.001), along with trans-myocardial gradient of IL-6 (r=0.58, p<0.01).

Conclusion: The magnitude of a trans-myocardial gradient of cytokines depends on the severity of heart failure decompensation and therefore, increased plasma level of cytokines in heart failure might be the result of myocardial production.

MeansSEM, *p<0.05 compared to peripheral veins.

### 1110-143

**Limited Diastolic Response to Afterload in Mice**

**Marc D. Feldman, Rebecca Blackwood, Danny Escobedo, Gregory L. Freeman, University of Texas Health Science Center, San Antonio, Texas.**

**Background:** The passive diastolic pressure-volume relationship of the left ventricle (LV) is nonlinear. Therefore, use of preload reserve should move the diastolic (ED) working point to a less compliant portion of the filling curve. Whether this occurs in mice, where exceedingly high heart rates diminish time for diastolic filling, is unknown. Accordingly, we assessed diastolic properties in hearts of open chest mice in response to sustained afterload.

**Methods:** Mice (C57B6 n = 6) were sedated, intubated, and via a sternotomy had a dual frequency pressure sensitive conductance catheter placed into the LV via the apex. A flow probe was placed around the thoracic aorta. Baseline LV pressure-volume relationship were recorded at steady state and during occlusion of the inferior vena cava. The hearts were exposed to sustained aortic occlusion for 7 minutes and recordings were repeated. LVdP/dt and volume, Tau, and chamber compliance (dp/dV) were determined.

**Results:** As shown in the Table, afterload augmentation led to an increase in maximum pressure (Pmax) and LVdP/dt, but Tau, LVEDP and dp/dV did not change.

<table>
<thead>
<tr>
<th>Group</th>
<th>500 ± 30 mmHg</th>
<th>1000 ± 70 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>502 ± 28</td>
<td>91 ± 6</td>
</tr>
<tr>
<td>Afterload</td>
<td>528 ± 31</td>
<td>119 ± 11</td>
</tr>
</tbody>
</table>

**Conclusions:** Although afterload stress caused an increase in LVEDV, there was no additional rise in LVEDP, indicating that the ventricles are operating on the flat portion of the PV curve. At the high operating heart rates of mice, short diastolic filling periods preclude LV filling sufficient to raise LVEDP.

### 1110-144

**Dynamic Expressions of eNOS, iNOS, and TNF-a in Canine Myocardium in Response to Pacing-Induced Dilated Cardiomyopathy**

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**Background:** Nitric oxide (NO) and tumor necrosis factor α (TNF-α) have been implicated in the pathogenesis of dilated cardiomyopathy. Whether and to what extent the failing heart of large mammals expresses these cytokines in the absence of inflammation is unclear.

**Methods:** We investigated the expressions of the constitutive endothelial nitric oxide synthase (eNOS), the inducible NOS (iNOS), and TNF-α in adult dogs under control conditions (conscious but chronically instrumented), following pacing-induced heart failure, and recovery from left ventricular failure using immunohistochemical (IHC) staining technique and Western blotting analysis.

**Results:** In control myocardium, prominent eNOS staining was seen in endothelium of blood vessels of varying sizes within the myocardium, with weak but positive staining in the myocardium. In the non-infarcted myocardium, the inducible NOS (iNOS), and TNF-α in adult dogs under control conditions (conscious but chronically instrumented), following pacing-induced heart failure, and recovery from left ventricular failure using immunohistochemical (IHC) staining technique and Western blotting analysis.

**Conclusions:** Our results suggest that loss of cardiac eNOS expression and emerging cytokine-endothelial influence from increased myocardial TNF-α and iNOS expressions may be pathogenic in the development of dilated cardiomyopathy and congestive heart failure. The influence of these cytokines is reversible following cessation of pacing and correlates with hemodynamic recovery.