

## MP5-11

### **Afterload augmentation is driven by the preload reduction rather than venous congestion in the chronic Fontan circulation**

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#### Background:

Based on the Pressure-Volume framework in the Fontan circulation, enhanced cardiac afterload compromises stroke volume, which exerts beta-adrenergic and RAAS cascades, ultimately resulting in cardiovascular remodeling. Since venous congestion directly accelerates end-organ dysfunction, early establishment of operable circulation with minimal central venous pressure (CVP) by balancing ventricular loading conditions (preload/afterload) would be the key to avoid "Fontan failure". We hypothesized that venous congestion augments cardiac afterload in the Fontan patients.

#### Method:

Transient inferior vena-caval occlusion (IVCO) was applied to induced venous congestion and preload reduction in 110 patients (23 Fontan and 87 non-Fontan), and augmentation of arterial elastance (Ea) was measured under construction of ventricular pressure-volume relationship. In addition to load independent measures of ventricular function, mean circulatory filling pressure (mcfP) was estimated and neurohormonal activations were evaluated.

#### Result:

As compared with non-Fontan patients, lower cardiac output in the Fontan patient was attributed by the augmented afterload with similar contractile function. While induced CVP augmentation at proximal to the IVCO was markedly higher in the Fontan patients as compared to non-Fontan patients ( $p=0.0017$ ), increase of Ea during IVCO was markedly suppressed in the Fontan patients ( $p=0.0090$ ). Interestingly, while Ea augmentation in the non-Fontan patients was affected both by venous congestion ( $p=0.0091$ ) and preload reduction ( $p=0.0011$ ), that in the Fontan patients was solely but markedly augmented by the preload reduction ( $p<0.0001$ , ANCOVA:  $p=0.049$  vs non-Fontan). To further delineate interaction between congestion and afterload, association with mcfP and systemic vascular resistance (SVR) was analyzed. Ea augmentation was negatively correlated with mcfP ( $p=0.025$ ) as well as systemic vascular resistance ( $p=0.047$ ), suggesting importance of decongestion for the preservation of Ea augmentation. Consistent with this, Ea augmentation was negatively correlated with natriuretic peptides (ANP/BNP), while serum level of aldosterone was independent of it.

#### Conclusion:

In the chronic Fontan circulation, insufficient preload, rather than additional venous congestion, predominantly increased afterload, implied vital role of preload preservation. In contrast, Ea augmentation against venous congestion was preserved in the non-Fontan patients implied need for optimizing mcfP by volume and venous manipulations.