Interventricular mechanical asynchrony in pulmonary arterial hypertension: right-to-left delay in peak shortening is due to right ventricular overload and impairs left ventricular filling

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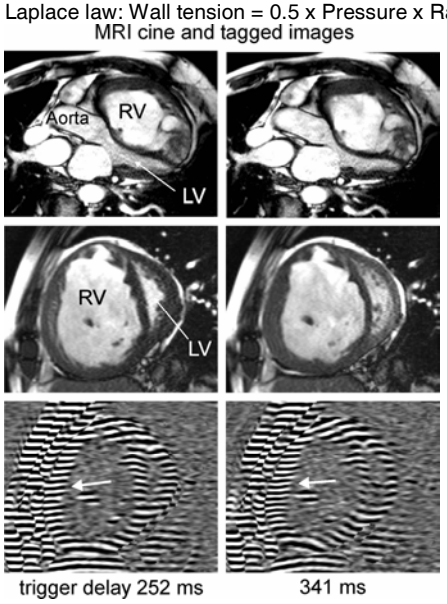
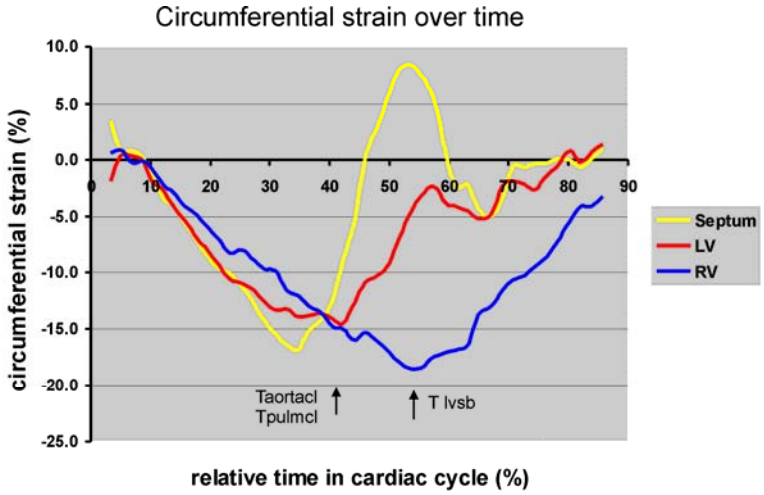
Objective To explore in pulmonary arterial hypertension (PAH) whether the origin of interventricular asynchrony lies in onset of shortening or in duration of shortening.

Methods In 11 PAH patients (mean pulmonary arterial pressure 54±11 mmHg and ECG-QRS width 109±17 ms), MRI myocardial tagging (14 ms temporal resolution) was applied. For the Left Ventricular (LV) free wall, septum and Right Ventricular (RV) free wall, the onset time (Tonset) and peak time (Tpeak) of circum-ferential shortening were calculated. RV wall tension was estimated by the Laplace law: Wall tension = 0.5 x Pressure x Radius

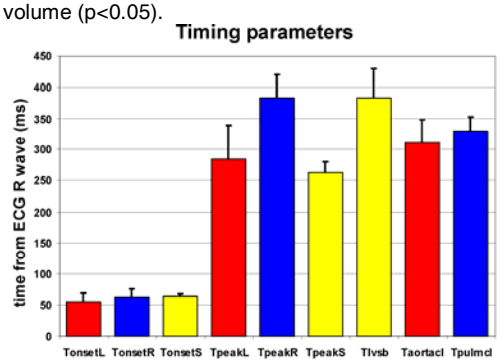


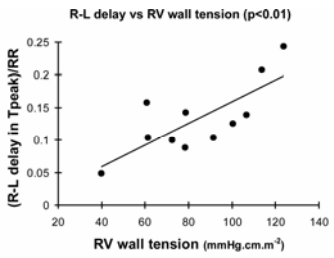
3-Chamber, short-axis and tagged images, at the time of aortic valve closure at trigger delay of 252 ms (left) and the time of peak RV shortening and maximal leftward septal bowing at 341 ms (right). In the tagged image at 341 ms, the distance of the tag lines in the RV wall show further shortening (thick arrows), while the tag lines in the LV wall show relaxation.

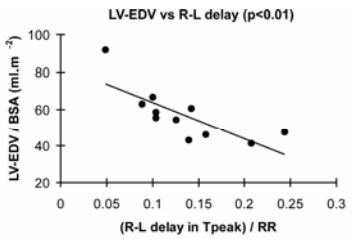


Circumferential shortening starts simultaneously for the LV and RV free walls and the septum, but the RV reaches its peak later. The closure times of aortic and pulmonary valves (Taortacl and Tpulmcl) are coincident with the peak of LV shortening. The time of maximal LVSB is coincident with septal stretching (positive strain), and with the peak of RV shortening.

Results Tonset was 54±11, 65±4 and 65±14 ms for LV, septum and RV respectively. Tpeak was 284±55, 262±18 and 383±37 ms for LV, septum and RV. Maximum leftward ventricular septal bowing (LVSB) was at 384±46 ms, coinciding with septal overstretch and RV Tpeak. The R-L delay in Tonset was 10±11 ms (p=0.13), and the R-L delay in Tpeak 99±25 ms (p<0.0001). The R-L delay in Tpeak was not correlated with the R-L delay in Tonset, nor with the QRS width, but did correlate with RV wall tension (p<0.01). The R-L delay in Tpeak predicted leftward septal curvature (p<0.05), and had a negative effect on LV End-Diastolic Volume (p<0.01) and stroke volume (p<0.05).







Discussion The prolonged RV systole in PAH is probably due to the increased RV wall tension as shown by the correlation between R-L peak delay and RV wall tension. This mechanism is supported by measurements in cardiac trabeculae which provided evidence that an increased load of myocytes leads to a slower shortening velocity.

Conclusion In PAH, the R-L delay in myocardial peak shortening is caused by increased RV wall tension instead of electrical conduction delay. This R-L delay causes LVSB, which makes the last part of RV shortening ineffective and impairs LV filling.