**Interventricular mechanical asynchrony due to right ventricular pressure overload in pulmonary hypertension:**

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**Introduction**

In pulmonary hypertension (PH), leftward ventricular septal bowing is most markedly observed during early left ventricular (LV) diastole, and impairs LV filling [1]. We hypothesize that a prolonged right ventricular (RV) systolic contraction contributes to leftward septal bowing. We tested this hypothesis by measuring the myocardial strain in RV and LV with high temporal resolution in PH patients.

**Methods**

*Imaging* PH patients (n=7) without primary cardiac disease, had pulmonary artery pressure values of 82 ± 29, 32 ± 10, and 52 ± 16 mmHg (systolic, diastolic and mean respectively), as measured by earlier catheterization. For MR imaging, a 1.5 T Siemens Sonata system with a 6-element phased array receiver coil was used. Steady state free precession imaging with complementary myocardial tagging [2] was applied with 14 ms temporal resolution in the mid-ventricular short-axis plane.

*Processing* Harmonic Phase strain analysis [3] yielded functional images of the maximum shortening, also referred to as second principal strain or lambda2, in each temporal frame of the cardiac cycle. The curves of lambda2 over time were calculated in 3 regions of the RV free wall (RV inferior, RV lateral, RV anterior) and the LV free wall (LV lateral). For each region, the time from ECG R-wave trigger to peak value (Tpeak) was calculated as a parameter for systolic duration. Tpeak of each RV region was tested versus Tpeak in the LV lateral region by paired samples t-testing.

**Results**

Figure 1 shows example images in a PH patient. At triggerdelay of 242 ms (left frames) the septum is flattened as explained by the simultaneous shortening in both RV and LV free wall. At 382 ms however, the septum bows to the left because now shortening occurs only in the RV free wall. Figure 2 shows the lambda2 curves for each region in the same patient. The RV lateral region reaches its maximal shortening at 382 ms, at a moment when the LV lateral region has already relaxed.

For all patients, the mean Tpeak values for the RV inferior, lateral and anterior regions were 322 ± 68, 366 ± 57, and 364 ± 53 ms respectively. For LV lateral, the mean Tpeak was 266 ± 54 ms. The Tpeak for RV lateral was 100 ± 43 ms later than for LV lateral ($p < 0.001$). The Tpeak for RV anterior was 98 ± 38 ms later than for LV lateral ($p < 0.001$).

**Discussion**

In PH, the prolongation of RV systole is probably caused by the high prestretch of the RV myocardial fibers, and the large force these fibers must generate to shorten.

**Conclusion**

In PH, the RV pressure overload causes a prolonged RV systole, which extends into the period of LVdiastole. This explains the maximal leftward septal bowing at the end of RV systole, and the subsequent impaired LV diastolic filling.

**References**

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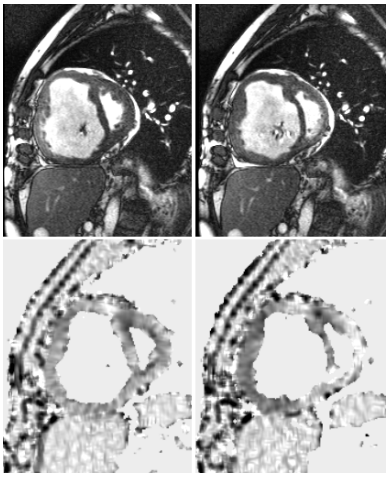
[2] Zwanenburg JJM et al. Magn Reson Med 2003; 49:722-730.

[3] Osman NF et al. Magn Res Med 1999; 42:1048-1060.



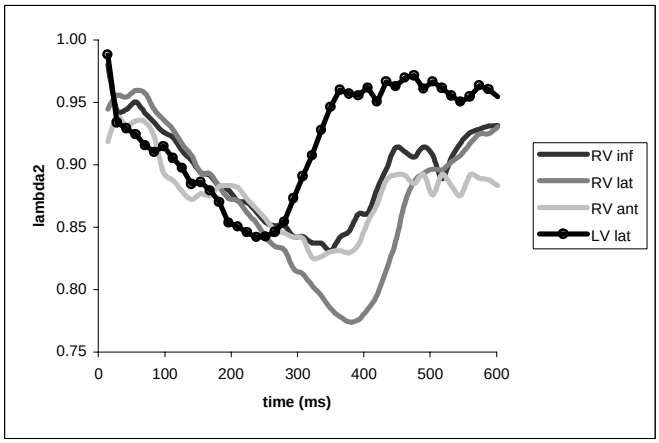



*Fig. 1: Short-axis anatomical images (above) and functional lambda2 images (below), at trigger delays of 242 ms (left) and 382 ms (right). Increased darkness in the lambda2 images corresponds to increased shortening.*

*Fig. 2: Time curves of lambda2 for each RV and LV region*