

# Tissue phase mapping reveals profound alterations of segmental left ventricular performance in patients with cardiomyopathy and left bundle branch block

D. Foell<sup>1</sup>, B. Jung<sup>2</sup>, E. Schilli<sup>1</sup>, F. Staehle<sup>2</sup>, C. Bode<sup>1</sup>, J. Hennig<sup>2</sup>, and M. Markl<sup>2</sup>

<sup>1</sup>Cardiology and Angiology, University Hospital Freiburg, Freiburg, Germany, <sup>2</sup>Dept. of Diagnostic Radiology, Medical Physics, University Hospital Freiburg, Freiburg, Germany

**Introduction:** Left bundle branch block (LBBB) is associated with a worse outcome in patients with cardiomyopathy [1,2]. LBBB results in asynchrony of left ventricular contraction and expansion which plays an important role in the course and prognosis of the disease. However, it remains difficult to measure and visualize the exact distribution of LV performance in such patients, as echocardiography despite its excellent temporal resolution is often limited by reduced imaging quality and incomplete coverage of all LV segments. Former MRI techniques had a limited temporal resolution. Recent development in Tissue Phase Mapping (TPM) now allow the quantitative evaluation of regional myocardial velocities with high spatial and temporal resolution [3] in all spatial directions and with full LV coverage. The aim of our study was to evaluate how left bundle branch block alters the segmental left ventricular performance in patients with dilated cardiomyopathy.

**Methods:** 3 short axis slices (basal, midventricular, apical) were acquired with a black blood prepared gradient echo TPM sequence (TR=6.9 ms; temporal resolution 13.8ms; spatial resolution 1.3x2.6mm; venc =15cm/s in-plane, 25cm/s through-plane) with prospective ECG- and advanced navigator gating [4], view sharing and first-order flow compensation (1.5 T, Sonata, Siemens). We examined 19 patients with dilative cardiomyopathy, e.g. with increased LV size and reduced LV function (LVEF < 45%) and 20 age-matched healthy controls. Coronary artery disease was ruled out in all the patients. 7 of these patients had LBBB. Data post-processing (Matlab, The Mathworks, USA) included a transformation of the measured three-directional motion velocities into radial, rotational and long-axis velocities adapted to the LV anatomy. TPM data were analyzed using an extended 16 segment AHA model covering LV base, mid, and apical slices, each subdivided into endo- and epicardial regions (fig. 1). For each subject, segmental velocity-time courses were calculated for each velocity component. Peak and times to peak (TTP) radial and long-axis velocities in systole and diastole were extracted from the velocity-time-curves and averaged for all subjects of a specific group. In addition, the 'wringing motion' of the heart (twist), i.e. the opposing rotational direction of basal versus apical slices, was characterized by calculating the difference between mean basal and apical rotation velocities.

**Results:** Compared to healthy controls, mean peak long-axis velocities and mean radial velocities were reduced and twist was decreased in systole and diastole (table 1, fig. 3) in the patients with cardiomyopathy. In general, the timing of the velocities was more inhomogeneous in the patients. Notably, not only systolic but also diastolic long-axis and radial expansion was altered by LBBB. Segmental analysis revealed several differences in timing and peak velocities between the patients with and without LBBB. Peak systolic and diastolic segmental long axis velocities were more reduced in patients with LBBB whereas systolic radial velocities were more decreased in patients without LBBB (fig. 1). The difference between peak velocities of apical and basal regions was generally reduced in patients compared to healthy controls, but this was even more pronounced in patients with LBBB. Radial systolic peak velocities in the septum and long-axis diastolic peaks in inferior and lateral segments were enhanced in patients with LBBB compared to the patients without LBBB (fig. 2). Moreover, velocity twist demonstrated marked changes and even inverted rotational behavior compared to patients without LBBB and normal controls (fig. 4).

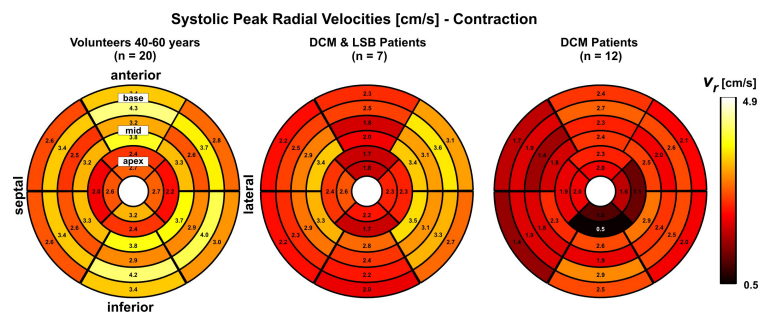
**Discussion:** TPM allows the visualization of the distribution of myocardial velocities and their timing with complete LV coverage. We could demonstrate reduced myocardial velocities and an altered distribution of peak and timing of velocities in patients with dilated cardiomyopathy. In these patients LBBB caused a variety of additional differences in segmental myocardial performance. With the use of new "cardiac resynchronization therapy" (CRT) systems it is now possible to influence the timing of myocardial performance and to improve LV contractility. However the enormous extent of alterations in myocardial performance caused by LBBB has not been elucidated. Interestingly, long-axis motion, rotation and diastolic function, which are difficult to assess by routine diagnostic tools were affected to a large extent. In this context, TPM may help to use new therapeutic options such as CRT more efficiently by providing new insights into how LBBB exactly alters LV performance.

**Acknowledgements:** Deutsche Forschungsgemeinschaft (DFG); Grant # HE 1875/18-1, FO 507/2-1, Bundesministerium für Bildung und Forschung (BMBF), Grant # 01EV0706.

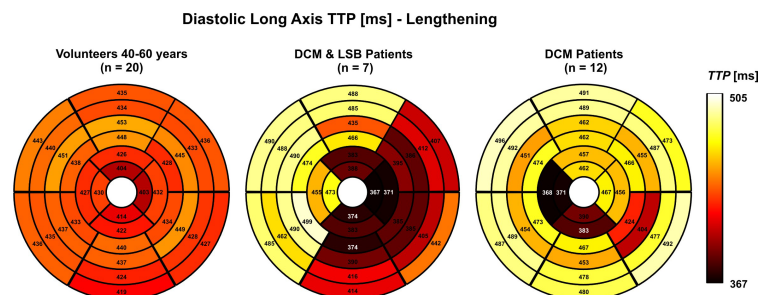
**References:** [1] Baldasseroni S et al., Am Heart J 2002; 143:398-405, [2] Hesse B et al., Am J Med 2001; 110: 253-259, [3] Jung et al. J Magn Reson Imaging 2006; 24:1033-39; [4] Jung et al. Magn Res Med 2006; 55:937-42.

	Age [years]	LV- EF [%]	Vrad sys [cm/s]	Vrad dia [cm/s]	Vlong sys [cm/s]	Vlong dia [cm/s]
Volunteers (n=20)	51.1 ± 3.9	57.4 ± 5.6	3.0 ± 0.6	- 4.3 ± 1.1	4.4 ± 1.5	- 6.2 ± 3.0
DCM + LBBB (n=7)	58.4 ± 10.5	22.6 ± 10.5	2.7 ± 0.9	- 2.3 ± 0.9**	3.3 ± 0.9*	- 3.7 ± 0.8*
DCM (n=12)	49.2 ± 10.1	33.8 ± 10.9	2.4 ± 0.6**	- 2.7 ± 1.1**	4.2 ± 1.0	- 4.8 ± 2.6*

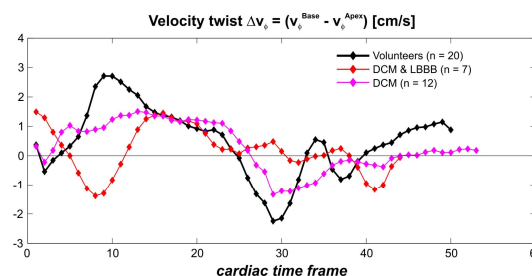
**Table 1:** Averaged values of systolic (sys) and diastolic (dia) peak radial (Vrad) and long-axis (Vlong) velocities in the patients with dilated cardiomyopathy (DCM) with and without left bundle branch block (LBBB) compared to healthy volunteers. LVEF= left ventricular ejection fraction



**Fig.1:** Comparison of systolic peak radial velocities for volunteers (left) with patients with left bundle branch block (DCM & LBB, middle) and without branch block (DCM, right). Peak velocities are colour coded onto an extended AHA16 segment model.



**Fig.2:** Comparison of diastolic times to peak (TTP) long-axis velocities for volunteers (left), patients with branch block (middle, DCM&LBB) and without branch block (DCM, right). TTP velocities are colour coded onto an extended 16 segment model.



**Fig.3:** Temporal evolution of velocity twist (diff. between basal and apical rotation) in volunteers, patients with dilated cardiomyopathy (DCM) and DCM with left bundle branch block (DCM&LBB).