# Propagation of time to onset of shortening in patients with non-ischemic dilated cardiomyopathy

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**Purpose:** The purpose of this work was to map the onset time of shortening ($T_{onset}$) in patients with dilated cardiomyopathy, and to characterize the propagation of the onset of shortening by a 3D vector.

**Background:** The amount of mechanical asynchrony is a more appropriate parameter than the amount of electrical asynchrony for adequate selection of patients that are likely to respond to resynchronization therapy [1]. However, a detailed description of the timing of shortening in patients by means of MRI is lacking.

**Methods:** Seventeen patients (56 ± 11 years, 10 male) with non-ischemic dilated cardiomyopathy (DCM) (NYHA class III-IV and EF < 45%) were studied. All patients underwent coronary angiography to exclude the presence of coronary artery disease, and delayed contrast enhancement (DCE) to exclude focal spots of fibrosis. High temporal resolution (14 ms) strain curves were obtained using steady state free precession imaging with tagging and HARP strain analysis [2], for five short axis slices. Imaging parameters for the tagging images were: voxel size 1.2 × 3.8 × 6.0 $mm^3$, flip angle 20º, TR/TE 4.7/2.3 ms, BW 369 Hz/pixel, matrix 256 x 78. For the DCE images, 10 to 15 min. after contrast injection an inversion-recovery gradient echo was applied with: voxel size 1.6 × 1.3 × 5.0 $mm^3$, flip angle 25º, TR/TE 9.6/4.4 ms, BW 130 Hz/pixel, and matrix 256 x 208.

$T_{onset}$ was defined as the beginning of the downslope of the circumferential strain curve and determined by an automated routine [2] for 6 circumferential segments. $T_{onset}$ was regarded as missing when the goodness of fit was less than 85% or when a region was akinetic (peak shortening less than 3%). Seventeen healthy subjects served as control group.

The propagation of the onset of shortening was characterized by the 3D onset of shortening delay vector (OSDV), which was determined by fitting the $T_{onset}$ values to the following equation:

$$\hat{T}_{\text{onset}}(c,l) = T_{const} + OSDV_{LA} \cdot \frac{l}{L-1} + \frac{OSDV_{SA}}{2} \cdot \cos\left(2\pi \frac{c}{C} + \varphi\right) \qquad \text{(Eq. 1)}$$

where $T_{const}$ is a constant, $OSDV_{LA}$ the long axis component, $OSDV_{SA}$ is the length of the short axis component, and $c$ and $l$ indices of the circumferential and longitudinal segments respectively. $C$ and $L$ are the number of circumferential and longitudinal segments. The circumferential segments are numbered counter clockwise, and such that the vector points from septum to lateral wall for $\varphi = 0$. The 3D OS-delay vector was then defined as: OSDV = ($OSDV_{SA}$·cos(φ), $OSDV_{SA}$·sin(φ), $OSDV_{LA}$). The first component of the OSDV gives the delay between the septum and the lateral wall (positive: lateral wall later than septum), the second gives the delay between IN and AN (positive: AN later than IN) and the third gives the delay between apex and base (positive: base later than apex).

**Results:** Although patients were non-ischemic 12% of the segments were akinetic and for that reason yielded no time to onset of shortening. Time to onset of shortening could be determined successfully in 91% that were kinetic. $T_{onset}$ in the DCM patients propagated mainly from the septum to the lateral wall (Fig 1.), and to a less extend from the apex to the base. For 5 patients, $T_{onset}$ was missing in more than 10 segments, and the OSDV was not calculated. The only component of the OSDV that was significantly different from the controls was the first component, pointing from septum to lateral wall. In this direction, propagation is reversed, and markedly delayed with respect to the healthy subjects (Table 1).

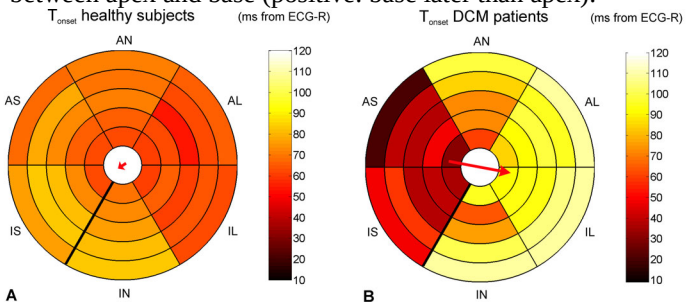


Fig 1. ***A:*** *Maps of the onset time of shortening ($T_{onset}$) averaged per segment over all 17 healthy subjects.* ***B:*** *same map for 17 patients with dilated cardiomyopathy. The arrows are the short axis projections of the 3D onset of shortening delay vectors. Note the marked septal-lateral wall delay in the patients. IS, inferoseptal; AS, anteroseptal; AN, anterior; AL, anterolateral; IL, inferolateral; IN, inferior.*

Table 1. *Onset of shortening delay vectors (OSDV) for both patients (DCM) and healthy subjects. A negative value means that the propagation is opposite to the direction indicated at the top of the column.*

| | Component of the delay vector (ms) | | |
|---|---|---|---|
| | septum → lat. wall | IN → AN | apex → base |
| OSDV DCM | 70 ± 49* | -12 ± 45 | 18 ± 16† |
| OSDV healthy | -12 ± 10 | -9 ± 9 | 9 ± 7 |

Values are means ± SD. *$P$ < 0.0001; †$P$ = 0.068 vs. controls.

**Conclusion**: Left ventricular timing of shortening in human subjects can be mapped non-invasively with MRI tagging. Using a 3D delay vector to denote the propagation of the mechanical asynchrony, we have shown that the main direction of asynchrony in DCM patients is from the septum to the lateral wall. The asynchrony in the longitudinal direction is much smaller, and only slightly larger than the asynchrony observed in healthy subjects.

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**References:** [1] Nelson G.S., et al., Circulation, 101:2703-9, 2000. [2] Zwanenburg J.J.M., et al, Am J Physiol Heart Circ.Physiol, 286:H1872-80, 2004.