# A Method with High Spatial and Temporal Resolution for Regional Analysis of Left Ventricular Torsion by MRI Tagging and HARP Tracking 

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## Introduction

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Torsion is the wringing motion induced by contracting obliquely oriented myofibers in the left ventricular (LV) wall, meant to squeeze blood from the ventricle. The following rapid untwisting sucks blood from the left atrium (LA). Since torsion is directly related to myofiber orientation and structure, it is thought to be an important quantifier for the condition of the heart.
In literature torsion was described in several ways $[1,2,3]$. As the difference between apical and basal rotation (twist), or as twist per unit length, which makes it comparable between hearts. Most directly related to fiber structure however, is to describe it as the circumferential-longitudinal shear (CLS) angle. Furthermore, it is not clear how to define the axis of rotation (AoR), which makes circumferential segmental analysis of torsion questionable. Previously, analysis of torsion was mostly limited to systole and only a limited part of the cardiac wall.
In this study, a method is proposed to calculate torsion automatically from tagged MR images over systole and diastole, using displacement data from the entire cardiac wall as seen in short axis slices with high temporal resolution (TR). It is investigated whether it is reasonable to calculate torsion on a segmental basis and the method is used to calculate torsion in five healthy volunteers.

## Methods

Complementary, sinusoidally tagged short-axis cine MR images are made in two orthogonal directions with an SSFP sequence with a TR of 14 ms [4] at apical and basal levels. Prospective triggering was used. With these sets of images, extended harmonic phase (HARP) tissue tracking [5] is performed after contouring the LV in the images. From the obtained displacements, the rotation around the center of mass of the LV is calculated for every correctly tracked point inside the contours in every timeframe. The radius is calculated for every point inside the contours as the distance between the point and the AoR in every timeframe. This means that both the AoR and the radius vary over time. The CLS angle $T$ is then calculated as

$$
\begin{equation*}
T(t)=\frac{\left(\Theta_{\text {apex }}(t)-\Theta_{\text {base }}(t)\right)}{D} \cdot \frac{\left(r_{\text {apex }}(t)+r_{\text {apex }}(1)+r_{\text {base }}(t)+r_{\text {base }}(1)\right)}{4} \tag{1}
\end{equation*}
$$

with $\Theta$ the mean rotation relative to the first timeframe, $r$ the radius, $t$ the cardiac phase number and $D$ the distance between the two slices. To determine the influence of the location of the AoR on the calculated torsion, a test case was used. This was an incompressible deforming cylinder with displacements given by analytical expressions [5]. Global and circumferential segmental (six segments) torsional deviation with a wrongly defined AoR were calculated. This was done for slices at both ends of the cylinder, with AoR moved separately, in the same or in opposite direction in both ends. The \% displacement in AoR from the center of mass relative to the mean radial length is indicated by the parameter AoRdisp. To illustrate the procedure, global, transmural and segmental rotation and torsion were calculated between apex and base in five healthy subjects. Peak rotation and peak torsion were compared over regions using a paired Student's t-test, where p-values below 0.05 were considered significant.

## Results

In the analytical test case, the largest deviations were found in torsion with oppositely displaced AoR between slices. These deviations were $0.05 \%$ per AoRdisp for the global calculation and $0.9 \pm 0.44 \%$ per AoRdisp for the segmental calculation. When segmental calculations were done on human data, torsion was larger in the anterior and anterolateral wall than in the septum and inferior wall ( $p=0.08$ ). Transmural differences in peak rotation and peak torsion were significant ( $\mathrm{p} \leq 0.03$ ): rotation was largest in the endocardium, torsion in the epicardium. Peak torsional values for the healthy subjects are given in table 1. In figure 1, a torsion curve for a healthy subject is presented.

## Discussion

Table 1. Peak CLS angles ( ${ }^{\circ}$ ) for healthy subjects. Values are mean $\pm$ SD.
The global calculation of torsion was shown to be almost independent of AoR location in the test case, while the segmental calculation was not. For a 100\% AoRdisp (AoR in the wall), only $5 \%$

|  | Whole | Infero- | Antero-septal | Anterior | Antero- | Infero-lateral | Inferior |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- | :--- |
|  | circumference | septal |  |  | lateral |  |  |
| Whole wall | $7.0 \pm 1.7$ | $5.9 \pm 2.6$ | $7.1 \pm 2.4$ | $11.1 \pm 5.5$ | $12.3 \pm 4.8$ | $5.9 \pm 1.0$ | $4.3 \pm 3.0$ |
| Endo | $6.7 \pm 1.7$ | $6.0 \pm 2.4$ | $7.6 \pm 2.2$ | $11.0 \pm 5.5$ | $10.9 \pm 3.8$ | $4.8 \pm 1.0$ | $4.0 \pm 2.6$ |
| Mid | $7.2 \pm 1.8$ | $6.3 \pm 2.6$ | $7.9 \pm 3.0$ | $11.9 \pm 6.0$ | $11.9 \pm 4.2$ | $5.4 \pm 1.0$ | $4.4 \pm 2.9$ |
| Epi | $8.1 \pm 2.1$ | $6.8 \pm 3.3$ | $8.3 \pm 3.4$ | $12.8 \pm 6.3$ | $13.1 \pm 4.6$ | $6.1 \pm 1.2$ | $4.5 \pm 2.9$ | deviation in global torsion is expected. In the human subjects, larger anterior and anterolateral torsion were found than septal and inferior. This might be due to physiology, but could also be explained by the definition of AoR. Presumably the mass of the right ventricle should also be included in the analysis. Miscontouring can as well cause differences between segments. This pleads for not using segmental torsion analysis. Calculation of torsion in transmural regions however, can be performed independently of AoR position. Since the fibers in the cardiac wall are known to vary in orientation in the transmural direction,

 this kind of analysis shows great promise for understanding the exact functioning and activation patterns of the myocardium and the cause of the smaller epicardial rotation and larger epicardial torsion in the healthy subjects.
In fig. 1, it is visible that the torsion does not completely restore to zero. This is due to remaining torsion at mid-diastole and the applied prospective triggering (last part of cardiac cycle is missing).

## Conclusion

The proposed method allows for automated calculation of LV torsion with high temporal and spatial resolution over the whole cardiac cycle. It was shown that segmental analysis of torsion is strongly dependent on the choice of the AoR. Transmural analysis however, was not critically dependent on the AoR and thus more suitable for quantifying the torsion values. Also regarding the anatomy, transmural analysis of torsion will be of great interest for further exploration of the mechanics of contraction and relaxation in the human heart.

Figure 1. Transmural differences in torsion (CLS angle) in a healthy volunteer.

## References

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[4] Zwanenburg et al., Magn Res Med 49:722;2003. [5] Tecelao et al., J Magn Res Imag 23:682;2006.

