

Cardiac Susceptibility Bite Mark Artifact: Resolving the Conflict

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Purpose: Cardiac cine SSFP imaging often suffers from artifactual distortions in the myocardium, particularly at the inferolateral and anterolateral myocardial margin, that have been described as "bite mark" signal voids^{1,2}. These artifacts potentially compromise the diagnostic quality of the cardiac MR images including morphologic and functional evaluation, particularly for R2* mapping³. Two conflicting explanations have been proposed regarding the source of these susceptibility-related artifacts: 1. the effect of deoxygenated blood in the cardiac veins¹ and 2. field inhomogeneities caused by the heart-lung interface². The purpose of this study was to determine whether the geometry of the heart-lung interface alone accounts for "bite mark" artifacts in vivo.

Methods: Experiments were conducted, after obtaining informed consent and IRB approval, using a clinical 3T scanner (MR750, GE Healthcare, Waukesha WI). Axial cardiac datasets were acquired in five asymptomatic volunteers using a 3D free-breathing, navigated, cardiac-gated, four-echo chemical shift encoded (CSE) spoiled gradient echo sequence⁴ using the body coil for transmission and a 32-channel receive-only phased array coil for signal reception. Imaging parameters included: field-of-view (FOV) 40 x 28-32 cm, slice thickness 3 mm, matrix size 256x160x50, trigger window 10%, trigger delay 70%, TE/TR/ α 1.2ms/6.4ms/15°, 2 echoes per TR in 2 shots.

The data were processed using a chemical shift encoded reconstruction⁵, providing high resolution estimates of the B_0 field map as well as separated water and fat images, and an R2* map. An air-tissue mask was created manually, from which an estimate of the susceptibility distribution was generated using the susceptibility values of water, fat, and air^{6,7}. Further, the susceptibility-induced B_0 field map (i.e. predicted field map) was calculated using the forward transform of the dipole response function^{8,9}. The predicted field map was compared with the B_0 field map estimated from the CSE reconstruction (i.e., the estimated field map). Magnitude images and B_0 field maps were reformatted in the short axis (SA) plane offline using Osirix (Pixmeo SARL). Regions of interest (ROI) were placed within areas of local field inhomogeneity in the mid cavity inferolateral myocardial wall. The mean of the predicted and estimated B_0 map within the ROI was normalized to the mean B_0 in the interventricular septum.

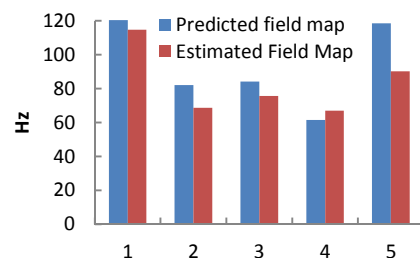


Figure 1: Normalized B_0 field map mean value in the inferolateral myocardium for all five subjects.

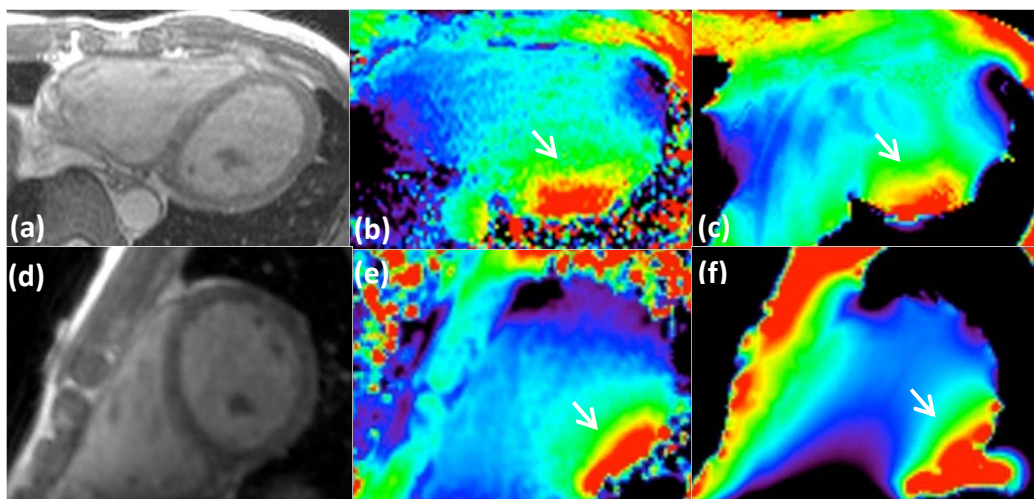


Figure 2: Axial SPGR magnitude image (a), calculated field map (b), and predicted field map (c) are shown. Additionally, SA reformat SPGR magnitude image (d), calculated field map (e), and predicted field map (f) are also shown. The scale for the field maps is shown below. The white and black arrows demonstrate areas of focal inhomogeneity in the inferolateral wall and anteroseptal wall, respectively, with excellent qualitative agreement between the field maps.

Results: All five datasets demonstrated good qualitative and quantitative correlation between the estimated and predicted field maps. A summary of the ROI measurements are shown in Fig 1. Representative axial and reformatted SA images are shown in Fig 2 for subject 3.

Discussion/Conclusion: We have compared a predicted B_0 field map based on the susceptibility distribution of fat, water and air, which qualitatively and quantitatively agreed with the calculated field-map based on CSE reconstruction. The general agreement between the two B_0 field maps indicates that the bite mark artifact can be attributed to the susceptibility interface of the heart-lung boundary. This work provided a unique perspective on the source of the artifacts that are in agreement with the results from the ex vivo experiments by Atalay et al.

References: 1. Reeder et al. MRM 1998; 39:988-998. 2. Atalay et al. MRM 2001;45:341-345. 3. Beache et al. Circ 2001;104:1214-1217. 4. Taviani et al. MRM 2014;72:718-725. 5. Hernando et al. MRM 2010;63:79-90. 6. Schenck et al. Med Phys 1996;23:815-850. 7. Szczepaniak et al. MRM 2002;47:607-610. 8. Salomir et al. Magn Reson Eng 2003;19B:26-34. 9. Sharma et al. MRM 2014;DOI:10.1002/mrm.25163.

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