# Quantification of the Magnetic Susceptibility of the Heart Using MRI: Demonstration on Normal Subjects

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*Introduction*

Cardiac complication due to iron overload is the leading cause of death in thalassemia patients treated with long-term transfusion therapy. Cardiac iron overload can be treated with chelation therapy, and the availability of a cardiac iron evaluation technique is potentially life saving for these patients. Currently there is no established modality for clinicians to measure the iron level in the heart [1]. The tissue iron concentration and magnetic susceptibility has a well defined, $B_0$ independent linear relationship [2,3]. This work proposes the possibility of measuring the magnetic susceptibility of the heart using MRI.

*Theory and Methods*

In MRI, the susceptibility is usually measured against a reference material. We note that there is a lipid layer of a few millimeters thick that is present on the surface of the left ventricle. Fatty issue in the body is not a site for iron accumulation under iron overload. We propose to use this lipid layer as reference for measuring the magnetic susceptibility of the cardiac tissue [4]. With this approach, the susceptibility difference $\Delta\chi$ between two macroscopic compartments in close contact can be obtained from the phase jump $\Delta\phi$ on gradient echo images across the interface and the angle $\theta_n$ between $B_0$ and the normal direction of the interface. For this particular case, we also need to consider the fact that the spins at two sides of the interface have different chemical shifts, giving rise to the second term on the right hand side:

$$\Delta\phi = 2\pi \cdot \mathbf{TE} \cdot \mathbf{f}_0 \cdot \frac{\Delta\chi}{3}(1 - 3\cos^2\theta_{\mathbf{n}} + \mathbf{S_{hf}}) + \mathbf{TE} \cdot 2\pi \cdot \Delta\mathbf{f}$$

Here TE is the echo time of gradient echo imaging, $f_0$ is the RF transmitter frequency, $S_{hf}$ is a contact shift term, and $\Delta f$ is the difference in resonance frequency between water and the fat. Here we assumed that the signal from the heart mainly comes from water and signal from the fatty tissue mainly comes from the lipids. In reality both sides contain water and lipid contributions. By choosing $TE \cdot \Delta f \sim$ integer, this assumption is not necessary and the effect of this term can be minimized.

Image data were acquired on a 1.5 T Philips Gyroscan Intera whole body scanner using a 3D T1-weighted (RF-spoiled) TFE sequence with cardiac triggering and respiratory navigator gating. Dual gradient echo acquisition was used with TR = 9.8 ms, TE = 4.7 and 7.1 ms, when the water and fat signal are approximately in phase and out of phase, respectively. Other imaging parameters were flip = 20°, TFE factor = 16 for FFE readout, number of signal average = 1, FOV = 300 mm, 10 to 15 slices with 1.6 to 2 mm thickness, in plane resolution = 0.586x0.586 $mm^2$. Data were acquired using a cardiac array coil in approximately 10 minutes. The data from one coil element was analyzed. Internally developed software allowed the selection of any spatial point on the muscle-lipid interface using a cursor. Using the amplitude image from the second echo, a curve fit routine calculated the normal direction of the interface, allowing us to calculate the angle between the $B_0$ field and the interface normal vector. The phase jump was measured on images with TE = 4.7 ms. At this TE, there remained a small phase difference between the tissue water and fat. This residual phase difference was corrected using the signals from the intercostal muscle and fat attached to it. Two control subjects have been studied.

*Results and Discussion*

The result from one control subject is shown in the Figure. The white box in the left panel indicates the region shown in the middle panel. A lipid layer on the surface of the left ventricular wall is clearly visible. This lipid layer serves as a reference for measuring the susceptibility of the cardiac tissue. The middle panel shows the phase map at TE = 4.7 ms (top) and amplitude map at TE = 7.1 ms (bottom). The right panel shows the phase profile along the dark line in the middle panel. Each data point on the phase profile is derived from an average of 9 pixels from 3 slices. There is a phase jump from the myocardium to the lipid layer, which can be quantified using a least squares procedure. The quantitative results from the 2 subjects are summarized in the Table.

This technique measures the susceptibility of epi-myocardium, which is the main site for iron accumulation. Testing on patients with cardiac iron overload is needed to demonstrate the applicability for clinical examinations. There could be difficulties in quantification related to heterogeneity in the iron distribution because cardiac tissue fibrosis is common when the iron level is high. In addition, the lipid layer might be too thin in certain patients to allow a reliable measurement. Much work is still needed to develop software tools that maximize the utilization of data from a large surface area. Further investigations will be conducted to understand the requirement on the signal to noise and spatial resolution for reliable quantification. The susceptibility measurement could be combined with the T2* approach [5] in patient examinations. Presently, calibration between T2* and cardiac iron concentration is not available. On the other hand, an animal model study demonstrated that the magnetic susceptibility of the iron in the heart is the same as that in the liver [6], and a $\Delta\chi$ of $1.0 \times 10^{-6}$ corresponds to an iron level of 0.63 mg Fe/g wet tissue. Therefore, susceptibility measurement has the advantage that data can be directly converted to a tissue iron concentration.

Fig.

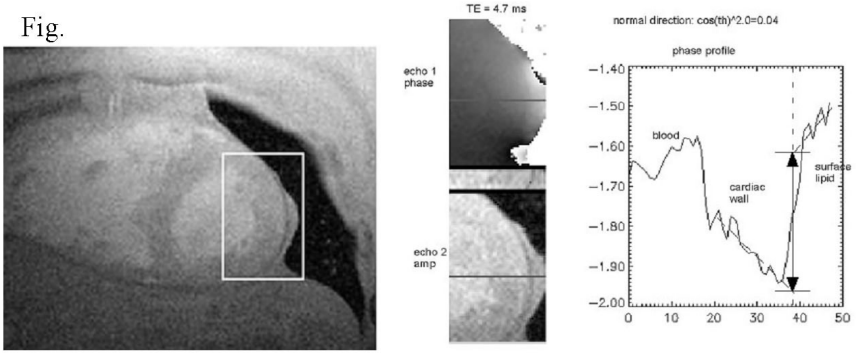


Table. Susceptibility in two control subjects.

| Sub. | $\cos(\theta_n)^2$ | $\Delta\phi$ | $\Delta\chi$ (S.I.) |
|---|---|---|---|
| 1 | 0.04 | 0.30 | $0.54 \times 10^{-6}$ |
| 2 | 0.94 | -0.03 | $0.03 \times 10^{-6}$ |

*References:*

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