Modelling the Effect of MRI Gradient Switches on Electrocardiograms

F. Liu1, H. Zhao1, L. Xia2, S. Crozier3
1University of Queensland, Brisbane, Qld, Australia, 2Zhejiang University, Hangzhou, HangZhou, China, People’s Republic of

Abstract
In MRI, patients are exposed to strong, time-varying gradient magnetic fields, which may be able to induce E-fields/currents in tissues approaching the level of physiological significance. This paper focuses on the theoretical investigations into the induced E-fields in the thorax and evaluates their potential influence on the cardiac electric activity under the assumption that the sites of maximum E-field correspond to the myocardial stimulation threshold. This is only true for conventional MRI technology for patients with existing cardiac pathology. Realistic, whole-body cylindrical and planar gradient coils have been explored as electromagnetic source. The calculations of the induced fields are based on an efficient, quasi-static, finite-difference (FDTD) scheme and an anatomically realistic, full-body, male model. The potential cardiac stimulation is evaluated using an electric model of the heart. 12-lead ECG signals have been simulated and inspected for arrhythmias or other abnormalities caused by the applied fields for both healthy and diseased hearts. The simulations show that the shape of thorax and conductive paths significantly influence the induced E-fields, and these fields are not sufficient to elicit ventricular fibrillation using contemporary gradients devices. Raising the strength and number of switching episodes of gradients beyond this level, however, as is certainly possible in local chest gradient sets, could potentially expose patients to significant risk. For patients with cardiac disease the risk factors are elevated.

Introduction
In vivo studies of the potential cardiac stimulation have been limited by the higher stimulation thresholds for the myocardial muscles and the inherent complexity of this biomedical problem. Clearly cardiac stimulation is potentially more dangerous than peripheral nerve stimulation (PNS). Furthermore, an elicited action potential from only one cardiac cell can enable a spread of excitation over all of the heart (WA Tacker and LA Geddes, Proc. IEEE, 84(3), 355-365, 1996). Hence, although conventional guidelines indicate that contemporary MRI devices operate well below thresholds of cardiac stimulation for normal patients, care must be taken for patients with cardiac pathology. This study is motivated, in part, by reports that a patient with heart disease did experience heart stimulation in a routine MRI scan [F. Lohr et al, J. Magn. Reson. Imag, 9, 624, 1999].

Computational Method
Induced electric fields were computed in the thorax using our recently reported FDTD techniques suitable for gradient calculations [L. Feng et al, Concepts in Magn. Reson.(MRE), 15, 26, 2002]. Whole-body gradient sets, both cylindrical and planar, were simulated with slew rates up to 100 T/m/s. The peak fields were registered in maximum magnitude and location on the heart model and the positions of maximum electric fields considered for the potential to induce transmembrane depolarization in localized cardiac cells. These results were then passed to the electrical heart model which has been previously validated [WX Lu and L Xia, IEEE Trans. Biomed. Eng., 43, 211, 1996]. The stimulation points were considered in the scenarios of continuous (i.e oscillating gradients), repeated or single excitation. These edoxogenous stimulations were then added to the heart’s normal electrical behaviour. The heart model contains about 65000 myocardial cell units with a spatial resolution of 1.5 mm. The excitation conduction system consisting of a sinus node, an atrial-ventricular node, a His-bundle, left and right bundles and Purkinje fibers. Cell type, conduction velocity, and action potential waveforms with variable durations are all considered for each unit. The heart model is mounted in an inhomogeneous human torso model. An excitation propagation algorithm which enables multi-cycle cardiac arrhythmia simulation is used to produce an activation sequence. Electric dipoles, which are proportional to the spatial gradient of the action potential, are generated in all of the cell units. These dipoles give rise to a potential distribution within the torso and various ECG summations can then be made (see Fig. 1).

The potential $\phi$ induced by cardiac field at any field position $r$ inside the torso can be expressed as

$$\phi(r) = \frac{1}{4\pi\sigma_p} \left[ \int_{S_1} J_k \nabla \left( \frac{1}{\rho} \right) dV + \sum_{l=1}^{n} \left( \sigma_{l1}^{(n)} - \sigma_{l2}^{(n)} \right) \int_{S_l} \phi(r) \nabla \left( \frac{1}{\rho} \right) ds \right]$$

Where $J_k$ is the cardiac source; $\sigma_l$ is the local conductivity at the field point; $\Omega$ denotes the source region (i.e. heart); $S_l$ denotes the internal closed interfaces, which separate homogeneous media of isotropic conductivities $\sigma_l$ on the outside and $\sigma^2_l$ on the inside; $S_1$ represents the outer torso surface. This basic formula is subject to the boundary condition that the field components normal to the body surface are zero.

Results
The induced E-fields appear to be largest on the lateral side of the heart, the myocardial surface closest to the coil, the junction of the septum wall and also the most anterior regions of the heart. Example E-field maps are shown in Fig. 2. In these areas, tissue-conductivity may vary abruptly, thereby causing significant charge accumulation. The spatial locations at which the peak E-fields occur were registered by considering different cross-sections of the heart muscle. For the cylindrical X-coils, the peak E-field was about 1.3 V/m at the top of the atria; for the cylindrical and planar Y-coils, the peak field value was about 0.9 V/m at the apex; and the ‘worst’ of the four cases considered was exposure to the Z gradients, which is about 2.3 V/m on the free wall of the right ventricle. Based on these values, and the fact that there is significant disagreement in the literature concerning cardiac thresholds [see, eg, Wang et al, IEEE Eng. Med. Biol conf, p475, 1995], the induced fields have large margins of safety for healthy heart tissue. For a heart with abnormal conduction pathways or other forms of pathology, the simulations indicate that current gradient sets may induce E-fields of sufficient magnitude to cause significant arrhythmias. We have simulated some of these pathologies.

Conclusion
The significant concern for time-varying magnetic fields is to protect patients from life-threatening arrhythmias such as ventricular fibrillation. Based on the simulation results of this report, it would be appear valid to conclude that gradient fields as presently implemented are unlikely to affect the normal functioning of the heart for patients without cardiac pathology. For some cardiac pathologies, such as aberrant accessory pathways, the safety margins are considerably reduced and can be investigated with these models.