Comments to the Editor

Interpreting Biomagnetic Fields of Planar Wave Fronts in Cardiac Muscle

ABSTRACT The recent results of Holzer and co-workers reveal the existence of net currents that flow along the front of a planar wave propagating through cardiac tissue. This is an important contribution toward the better understanding of the physics of biomagnetic fields. However, although the authors claim their results reveal particular bidomain properties, we show in this short letter that the results allow multiple interpretations. For instance, cardiac anisotropy by itself may also explain the existence of a net current along the wave front. Based on our calculations, we suggest additional experiments that would allow distinguishing between these two explanations and thus provide further evidence on the basic physics behind cardiac biomagnetism.

INTRODUCTION

The magnetic field generated during the electric propagation on cardiac tissue is still a subject of research. The recent article of Holzer et al. (2004) is an important contribution toward the understanding of the basic physics behind such biomagnetic fields. Using a unique combination of transmembrane potential and B_z magnetic component maps, the authors' experiment reveals the existence of net currents that flow along the front of a propagating planar wave (J_y, as illustrated in Fig. 1). These findings are in contrast to the traditional assumption of Frank (1953), where a layer of current dipoles travels parallel to the propagation direction (J_x, as in Fig. 1). The new results of Holzer et al. (2004) show that a more sophisticated mechanism is taking place.

A possible explanation for the experimental findings is based on the theoretical work of Roth and Woods (1999) and on the bidomain model (Henriquez, 1993): the magnetic field would be generated by a net current that accounts for the sum of intra- and extracellular currents of cardiac tissue. Due to the unequal anisotropies in the intra- and extracellular domains, the bidomain net current has a very distinct property—it flows along the front of a propagating planar wave, i.e., there is no net current orthogonal to the wave front ($J_x = 0$; see Fig. 1). This is a very distinct source mechanism from the one accepted to explain cardiac electric fields.

Although Holzer and co-workers claim their recent results reveal such distinct bidomain properties, we show next in this short letter that the results allow multiple interpretations by the fact that J_x does not contribute to B_z. For instance, a current density J that flows mainly along the cardiac fibers (see Fig. 1) generates the same B_z magnetic component as the one generated by the J_y current component. Therefore, the experimental results presented in (Holzer et al., 2004) do not support for bidomain properties.

THE INFORMATION CONTENT OF B_Z IS INSUFFICIENT

The experimental work of Corbin and Scher (1977) has shown the importance of the cardiac tissue anisotropy in the generation of electrocardiograms. We next calculate the magnetic field vector generated by a propagating planar wave, assuming that the net current ($\mathbf{J}$) flows preferentially along the cardiac fibers ($J_x, J_y \neq 0$ for $\theta \neq 0°, 90°$), as a result of the cardiac tissue bulk anisotropy. Our calculations show that the B_z magnetic component depends only on J_y. Therefore, the cardiac tissue anisotropy is also a possible explanation for the experimental results of Holzer et al. (2004).

We consider a planar wave of transmembrane potentials ($\partial V_m/\partial y = 0$) propagating toward the x direction on an infinite plane of small width h at $z = 0$. Cardiac fibers have an inclination θ. The anisotropy of the tissue can be modeled by a conductivity tensor that in some arbitrary direction is given by

$$\sigma = R^{\mathrm{T}} \begin{pmatrix} \sigma_{\mathrm{l}} & 0 \\ 0 & \sigma_{\mathrm{t}} \end{pmatrix} R,$$

where the matrix R reflects the change of coordinate from the cardiac fiber direction to the direction of propagation, and σ_{l} and σ_{t} are the conductivity values along and transversal to the fibers, respectively. Since all cardiac fibers have an inclination of θ, we have

$$\sigma = \begin{pmatrix} \sigma_{\mathrm{l}} \cos^2\theta + \sigma_{\mathrm{t}} \sin^2\theta & (\sigma_{\mathrm{l}} - \sigma_{\mathrm{t}}) \cos\theta \sin\theta \\ (\sigma_{\mathrm{l}} - \sigma_{\mathrm{t}}) \cos\theta \sin\theta & \sigma_{\mathrm{t}} \cos^2\theta + \sigma_{\mathrm{l}} \sin^2\theta \end{pmatrix}.$$

The net current can be expressed as $\mathbf{J} = \boldsymbol{\sigma}\, \nabla V_m$, and thus

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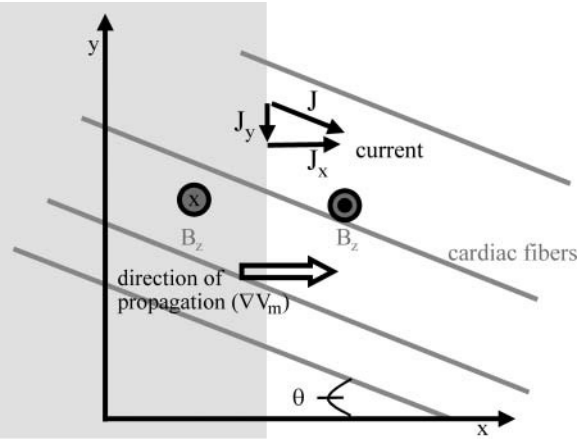

FIGURE 1 A planar wave propagating toward the x direction on an infinite plane. Cardiac fibers have an inclination θ. Current density ($\mathbf{J}$) flows mainly along the cardiac fibers. Only the projection of $\mathbf{J}$ into y (J_y) generates the z component of the magnetic field, B_z. The J_x contribution to B_z cancels out due to the symmetry of the plane wave (see text for details).

$$\begin{aligned}\mathbf{J} &= (J_x, J_y)\\ &= (\sigma_l \cos^2\theta + \sigma_t \sin^2\theta, (\sigma_l - \sigma_t)\cos\theta\sin\theta)^T \partial V_m/\partial x.\end{aligned} \tag{1}$$

From Eq. 1, we see that due to the tissue anisotropy, even with $\partial V_m/\partial y = 0$, we have $J_y \neq 0$.

The magnetic field can be calculated with the Biot-Savart equation

$$B(r') = \frac{\mu_0 h}{4\pi}\int_{-\infty}^{+\infty}\int_{-\infty}^{+\infty}\left(J \times \frac{r-r'}{|r-r'|^3}\,dx\,dy\right),$$

where $\mathbf{r} = (x,y,0)$ and $\mathbf{r}' = (x',y',z')$.

Under the above assumptions, since the current $\mathbf{J}$ varies only with x, we can rewrite the Biot-Savart equation and find

$$B_x(r') = \frac{\mu_0 h z'}{2\pi}\int_{-\infty}^{+\infty}\left(\frac{J_y}{(x-x')^2 + z'^2}\,dx\right)$$

$$B_y(r') = -\frac{\mu_0 h z'}{2\pi}\int_{-\infty}^{+\infty}\left(\frac{J_x}{(x-x')^2 + z'^2}\,dx\right) \tag{2}$$

$$B_z(r') = -\frac{\mu_0 h}{2\pi}\int_{-\infty}^{+\infty}\left(\frac{(x-x')J_y}{(x-x')^2 + z'^2}\,dx\right), \tag{3}$$

where we have used that

$$\int_{-\infty}^{+\infty}\left(\frac{(y-y')}{|r-r'|^3}\,dy\right) = 0,$$

and

$$\int_{-\infty}^{+\infty}\left(\frac{1}{|r-r'|^3}\,dy\right) = \frac{2}{(x-x')^2 + z'^2}.$$

Equation 3 shows that J_x does not contribute to B_z due to the symmetry of the planar wave propagation, i.e., J_x does not vary with y ($\partial J_x/\partial y = 0$). B_z reflects only the y component of the current distribution. Therefore, the experimental results of Holzer et al. (2004), by revealing the existence of currents along the wave front, i.e., $J_y \neq 0$, do not distinguish between the distinct bidomain properties (which predicts $J_x = 0$ and $J_y \neq 0$) and the influence of the tissue anisotropy ($J_x \neq 0$ and $J_y \neq 0$).

PROPOSAL OF ADDITIONAL EXPERIMENTS

One way to distinguish between the two mechanisms, bidomain and tissue anisotropy, is via the θ-dependence of the maximum amplitude of B_z. Bidomain theory predicts the following dependence on θ for J_y (Roth and Woods, 1999):

$$J_y = \frac{(\sigma_{il}\sigma_{et} - \sigma_{el}\sigma_{it})\cos\theta\sin\theta}{(\sigma_{il}+\sigma_{el})\cos^2\theta + (\sigma_{it}+\sigma_{et})\sin^2\theta}\,\frac{\partial V_m}{\partial x}.$$

The dependence on θ for J_y due to the tissue anisotropy is given by Eq. 1. Thus, by initiating planar waves with different inclinations with respect to the cardiac fiber, one could try to verify which model best fits the experimental data. Unfortunately, the large error for estimating the fiber angle (12°) and the B_z amplitude variation in the order of 1.0 nT, as reported in Holzer et al. (2004), could still disturb the experimental data interpretation.

Equation 2 shows another and perhaps easier way how the two described mechanisms could be distinguished. The horizontal component B_y of the magnetic field depends on J_x, and should be thus always equal to zero, as predicted by the bidomain theory. In contrast, if only the anisotropy of the tissue is playing the role, J_x should always be different than zero, as described by Eq. 1. A SQUID microscope design with a vertical pickup coil (Matthews et al., 2003) would be able to measure the horizontal B_y and B_x components.

CONCLUSION

So far, the relation between bidomain theory and cardiac biomagnetism was mainly investigated by theoretical and modeling works and still lack sound experimental evidence. The confirmation of this theory would support a novel tool based on magnetic sensors, extremely promising for basic investigation on cardiac electrophysiology. The combination of electric and magnetic sensors during in vitro experiments would provide one with a better understanding of cardiac intracellular currents, intracellular conductivity, and gap

junction effects during normal and abnormal cardiac electric propagation. We believe the additional experiments described here would be a beneficial supplement to the valuable approach of Holzer et al. (2004) and provide further evidence for the establishment and proof of a theory for cardiac biomagnetism.

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