THE NATURE OF SOURCES OF BIOELECTRIC AND BIOMAGNETIC FIELDS

ROBERT PLONSEY

Department of Biomedical Engineering, Case Western Reserve University, Cleveland, Ohio 44118

ABSTRACT   The goal of this paper is to examine the origins and relative importance of primary and secondary sources of electric and magnetic fields for excitable tissue. It is shown that for axonal and cardiac tissue a comparison of the relative field strength from both primary and secondary sources shows only the latter to be significant. Even if the divergence and curl of the primary source were independent (and hence were both needed to define the primary source), because the secondary sources all arise from the divergence of the primary source the magnetic field reflects the same source component as the electric field. As a consequence magnetic and electric fields arising from active tissue are strongly linked.

CURRENT FLOW (PRIMARY) FIELDS

The goal of this paper is to examine the origins of the primary and secondary sources of the electric and magnetic fields for excitable tissue and, in particular, to contrast their relative importance. Our greatest interest is devoted to cardiac muscle but most of the paper applies equally well to other excitable tissues. We begin with a consideration of the primary field.

In biophysical studies of excitable tissue the volume conductor medium is characterized as resistive. The electric field, $\overline{E}$, is consequently associated with a current density $\overline{J}$, and these are related by Ohm's Law

$$\overline{J} = \sigma\overline{E} \tag{1}$$

where σ is the conductivity. Eq. 1, in fact, applies throughout all of both intracellular and extracellular space (which are passive), and it is only within excitable membranes that it might not be valid. Associated with the membrane are additional "driving forces" of current, namely diffusion and active transport (1, 2). These are essentially nonelectrical in nature but can enter through the currents they generate, (i.e., currents which arise as a result of the conversion of stored chemical energy and/or metabolic energy [splitting of ATP] into electrical form). While it is possible to describe these contributions mathematically it will be adequate for our purposes to simply introduce into Eq. 1 a nonohmic (nonconservative) current that we designate as an impressed current $\overline{J}^i$. If all field quantities were complex phasors so that σ could represent a membrane impedance (i.e., including membrane capacitance) the impressed current would correspond to the ionic current of the Hodgkin-Huxley formalism (3). Thus, in general,

$$\overline{J} = \sigma\overline{E} + \overline{J}^i \tag{2}$$

and $\overline{J}^i \neq 0$ only within active cellular membranes.

Taking into account the frequency content of electrophysiological signals, as well as the conductivity parameters of physiological media, permits us to establish that all fields of biological origin are quasi static; that is, currents and voltages can be considered as if static at any given instant (4). In particular,

$$\overline{E} = -\nabla\phi \tag{3}$$

where ϕ is the electric potential. Because $\overline{J}$ in Eq. 2 is the total current, then, according to Maxwell's equations, it is solenoidal. Taking the divergence of Eq. 2 and using Eq. 3 leads to Poisson's equation,

$$\nabla^2\phi = \nabla \cdot \overline{J}^i/\sigma \tag{4}$$

and $-\nabla \cdot \overline{J}^i$ is a volume source density function. $\overline{J}^i$ is also a source function for the magnetic field which, in terms of the vector potential $\overline{A}$ (5), satisfies

$$\nabla^2\overline{A} = -\overline{J}^i \tag{5}$$

while the magnetic field $\overline{H}$, is related to the vector potential $\overline{A}$ by

$$\overline{H} = \nabla \times \overline{A}. \tag{6}$$

SECONDARY SOURCES

The solution of Poisson's Eqs. 4 and 5 is trivial for media that are uniform and infinite. Ordinarily in physiological preparations the permittivity and permeability can be

assumed to be uniform but discontinuities in the conductivity are typically seen at both the cellular and the macroscopic levels. The effect of these inhomogeneities can be taken into account through the secondary sources that are generated.

If we make the simplifying assumption that the conductivity is piecewise constant then the secondary sources arise at the interface between media of differing conductivity. For example, at the kth interface that separates two regions with conductivity σ'_k and σ''_k the scalar function $\psi = \sigma\phi$, where ϕ is the electrical potential, satisfies

$$\psi'_k \neq \psi''_k \tag{7}$$

$$\partial\psi'_k = \partial\psi''_k/4jn. \tag{8}$$

Eq. 8 is an expression of continuity of normal component of current ($\sigma'_k\partial\phi'_k/\partial n$) although the inequality of Eq. 7 is true because ϕ_k is continuous and $\sigma'_k \neq \sigma''_k$. The conditions in Eq. 7 and 8 are assured if a current double layer lies at the interface (in an equivalent uniform homogeneous medium with the conductivity that of the field point) whose strength is the discontinuity in the scalar function ψ, namely

$$\overline{K}^i_k = (\psi''_k - \psi'_k)\overline{n} = \phi_k(\sigma''_k - \sigma'_k)\overline{n} \tag{9}$$

where $\overline{n}$ is the surface normal from primed to double-primed region (6). This result was originally noted by Geselowitz (7).

Secondary sources arise not only at the interface between macroscopic regions of different conductivity but also at those which occur on a cellular scale. Thus at the inner and outer membrane surface of a cell we have, applying Eq. 9

$$\overline{K}^i_c = [\phi_e(\sigma_e - \sigma_m) - \phi_i(\sigma_i - \sigma_m)]\overline{n} \tag{10}$$

where σ_e, σ_m, σ_i are the extracellular, membrane, and intracellular conductivities; ϕ_e, ϕ_i are the extracellular and intracellular potentials; and $\overline{n}$ is the outward normal from the cell. While the double layer sources associated with the inner and outer membrane are not congruent their spacing is so small that in Eq. 10 they have been merged. The membrane conductivity σ_m is, strictly, a phasor quantity because it includes a capacitive as well as conductive component. At frequencies associated with action currents the displacement current, at most, equals the conduction current; however, even under these conditions σ_m is very small compared with σ_e or σ_i (8) and Eq. 10 reduces to

$$\overline{K}^i_c = (\sigma_e \phi_e - \sigma_i \phi_i)\overline{n}. \tag{11}$$

ELECTRIC AND MAGNETIC FIELD EXPRESSIONS

The integral solution to Eq. 4 (Poisson's equation) is given (5) by

$$\Phi = -\frac{1}{4\pi\sigma} \int \frac{\nabla \cdot \overline{J}^i}{r} dV \tag{12}$$

where r is the distance from source to field and the del operator is with respect to source coordinates. An alternate form of Eq. 12 follows from the vector identity

$$\nabla \cdot (\overline{J}^i/r) = (1/r)\nabla \cdot \overline{J}^i + \overline{J}^i \cdot \nabla(1/r). \tag{13}$$

If both sides of Eq. 13 are integrated over a volume containing all sources and if the divergence theorem is applied to the left-hand side then this integral must go to zero because $\overline{J}^i$ is zero at the bounding surface. Consequently

$$\int \frac{\nabla \cdot \overline{J}^i}{r} dV = -\int \overline{J}^i \cdot \nabla(1/r) dV \tag{14}$$

and Eq. 12 can also be written

$$\Phi = \frac{1}{4\pi\sigma} \int \overline{J}^i \cdot \nabla(1/r) dV. \tag{15}$$

Eq. 5 can be solved in the same way as Eq. 4 leading to

$$\overline{A} = \frac{1}{4\pi} \int \frac{\overline{J}^i}{r} dV \tag{16}$$

so that, by applying Eq. 6, we get[1]

$$\overline{H} = \frac{1}{4\pi} \int \frac{\nabla \times \overline{J}^i}{r} dV. \tag{17}$$

Using the vector identity $\nabla \times (\overline{J}^i/r) = \nabla(1/r) \times \overline{J}^i + (\nabla \times \overline{J}^i)/r$, integrating over a volume containing all sources and converting the integral on the left-hand side to a surface integral by Stokes' theorem (this integral is zero because $\overline{J}^i$ is zero over the bounding surface) shows that

$$\int (\nabla \times \overline{J}^i)/r \, dV = -\int \nabla(1/r) \times \overline{J}^i \, dV. \tag{18}$$

Consequently, Eq. 17 has the alternate form

$$\overline{H} = (1/4\pi) \int \overline{J}^i \times \nabla(1/r) dV. \tag{19}$$

The total electric and magnetic fields require that both primary and secondary sources be included. Consequently adding $\overline{K}^i_k$(Eq. 9) and $\overline{K}^i_c$(Eq. 11) to $\overline{J}^i$, and using Eq. 15 or 19 leads to

$$\Phi = (1/4\pi\sigma) \int \overline{J}^i \cdot \nabla(1/r) \, dv + (1/4\pi\sigma) \sum_k$$

$$\int (\sigma_k'' - \sigma_k')\overline{n} \cdot \nabla(1/r) \, dS_k +$$

$$\frac{1}{4\pi\sigma} \sum_c \int (\sigma_e\Phi_e - \sigma_i\Phi_i)\overline{n} \cdot \nabla(1/r) \, dS_c \tag{20}$$

$$\overline{H} - \frac{1}{4\pi} \int \overline{J}^i \times \nabla (1/r) \, dV + (1/4\pi) \sum_k$$

$$\int \Phi_k (\sigma'_k - \sigma'_k) \, \overline{n} \times \nabla (1/r) \, dS_k +$$

$$(1/4\pi) \sum_c \int (\sigma_e \Phi_e - \sigma_i \Phi_i) \, \overline{n} \times \nabla (1/r) \, dS_c \quad (21)$$

where the summation is over k macroscopic interface and c excitable cell membranes. The unsubscripted conductivity in Eq. 20 is that of the field point.

RELATIVE CONTRIBUTION OF PRIMARY AND SECONDARY CELLULAR SOURCES

In Eqs. 20 and 21 the primary source, $\overline{J}^i$, lies in the membrane, as explained earlier. Its contribution to the ϕ and $\overline{H}$ field can be compared with that arising from the secondary cellular sources (Eq. 11). Although such a comparison depends on the particular shape of the propagating action potential, the order of magnitude of secondary-cellular-to-primary-cellular source was estimated by Plonsey (8) as 10^7. Swinney and Wikswo (9) estimated this ratio as 10^{11}. So, with respect to computation of $\overline{E}$ and $\overline{H}$ fields, $\overline{J}^i$ can be ignored.

Because the aforementioned referenced papers deal, basically, with a simple single excitable fiber, we consider in some detail here a comparison of the contribution to the electrocardiographic field from primary and secondary sources in the heart. Because the total number of cardiac cells is very large (perhaps 10^{10}) it is convenient to consider a small subset (say 1,000 cells) as an elementary unit. Though very small compared with the total number of cells, it is large enough to be considered through averaged, uniform, properties.

If typical activation patterns are examined (10) it is clear that they involve well-behaved wavefronts that are nearly flat over most regions. Accordingly, in the syncytial cardiac tissue, one can describe the behavior as that of a plane excitation front (i.e., a linear core conductor relationship); for anisotropic tissue the intracellular and extracellular axial resistance per unit length depends on the direction of propagation relative to the fiber axis (11). This forms the basis for a comparison of the first and third terms of Eq. 20 (or 21) which, it will be noted, are integrated over the same membranes. In fact, because each membrane is thin, and $\overline{J}^i$ is uniform, normal to each cell surface, and confined to the membrane, we compare

$$\text{secondary/primary} - \int (\sigma_e \phi_e - \sigma_i \phi_i) \nabla (1/r) \cdot d\overline{S} /$$

$$\gamma t \int \overline{J}^i \nabla (1/r) \cdot d\overline{S}. \quad (22)$$

For the electric field case this expression is the ratio of the respective electric potentials; for the magnetic field case it is an evaluation of the ratio of the respective vector potentials. In Eq. 22 t is the membrane thickness, while γ is the volume to surface ratio for cardiac tissue (considering it composed of cylindrical cells of radius a then $\gamma = $

$\pi a^2/2\pi a = a/2$). Because ϕ_e, ϕ_i, and V_m (the transmembrane potential) are related by linear core conductor theory (11), this provides an expression for transmembrane current as $g_\ell \partial^2 \phi_i / \partial \ell^2$ (ℓ is the direction of propagation and g_ℓ is the effective intracellular conductivity in that direction) and this serves as a good approximation to $\overline{J}^i$ (the displacement current being neglected).

The comparison of the expressions in Eq. 22 can be facilitated by their transformation to axial integrals of the second derivative of the source function divided by r (12). Because the activation wavefronts propagate generally across fiber directions, the transverse resistivities of Roberts et al. (11) show that $\phi_i = 5\phi_e$, a result that implies intramural potentials of ~30 mV, which is a reasonable experimental figure. Consequently, we can take $\phi_i \simeq V_m$, and the comparison of Eq. 22 made on the following expressions which are integrated across the active wavefront (usually of thickness 0.5 mm) per unit area of wavefront

secondary/primary

$$= g_\ell \int \frac{\partial V_m^2 / \partial \ell^2 \, d\ell}{r} \bigg/ g_\ell t \gamma \int \frac{\partial_m^{4V} / \partial \ell^4 \, d\ell}{r} \quad (23)$$

(see also reference 13 for evaluating the cross-fiber source). This can be rewritten

$$\text{secondary/primary} = \frac{1}{t\ell} \frac{\int_0^{0.05} \dfrac{\partial^2 V_m / \partial \ell^2}{r} \, d\ell}{\int_0^{0.05} \dfrac{\partial^4 V_m / \partial \ell^4}{r} \, d\ell} \quad (24)$$

where the wave thickness of 0.05 cm (rising phase of 1 ms times a slow velocity of 50 cm/s) is the basis for the integration limits. Because the field of the secondary source in Eq. 24 is that of a dipole although the primary source field is that of an octapole, a comparison can only be meaningful at specific field locations. Assuming the distance to a field point large compared with 0.05 cm, we can approximate Eq. 24 by

$$\text{secondary/primary} = \frac{1}{t\gamma \Delta Z^2} \cdot \frac{\dfrac{\cos \theta}{R^2}}{\dfrac{15 \cos^3 \theta - 9 \cos \theta}{R^4}} \quad (25)$$

where the field point is at (R, θ) and $\Delta Z = 0.05$ cm is the separation between lower multipoles in generating higher multipoles. We will examine Eq. 25 for a field point on the axis (when $\theta = 0$) and on the anterior chest — say 2.5 cm from an active source assumed on the anterior heart (which will make Eq. 25 as small as possible). Letting $t = 100$ Å, fiber radius $a = 8\mu$, then

$$\text{secondary/primary} = 1 \times 10^{12}, \quad (26)$$

which is roughly the relationship found by Swinney and Wikswo (9) for the nerve axon. This result does depend on

the macroscopic field assumption in which we used averaged uniform properties characteristic of a region of ~1,000 cells. Possibly a careful study of the cellular behavior of the 1,000 cells or so that underlie this macroscopic element would inject a correction factor. However, it is extremely unlikely that it could overturn the essential relationship found above.

DISCUSSION

The introduction of the SQUID (superconducting quantum interference device) (14) with its ability to detect magnetic fields of picotesla strength has given rise to a greatly extended interest in the measurement of magnetic fields of biological origin. The question as to whether such measurements contribute anything basically new relative to their electrical potential counterparts continues as a controversial question since first considered with respect to fields of cardiac origin.

I suggested (15), based essentially on Eq. 12 and 17 that ϕ and $\bar{H}$ contained independent information concerning the source $\bar{J}^i$ because, according to the Helmholtz theorem, (5), $\nabla \cdot \bar{J}^i$ and $\nabla \times \bar{J}^i$ could be assigned independently. Rush (16) subsequently criticized this conclusion based on certain constraints that apply to the $\bar{J}^i$ of bioelectricity that could preclude $\nabla \cdot \bar{J}^i$ and $\nabla \times \bar{J}^i$ being arbitrary. The matter did not seem completely resolved because the geometries considered were simple ones. What I have shown here is that, in fact, the fields measured do not even arise from $\bar{J}^i$, but rather from secondary sources only. These secondary sources, in turn, depend on both the electrical field and the interfaces, and hence are related to $\nabla \cdot \bar{J}^i$ and the geometry. So both electric and magnetic fields have as their sources a common source type. In fact this point has already been made relative to macroscopic secondary sources by Wikswo et al. (17).

We have established here that the sources of both electric and magnetic fields are of the secondary type and that these are themselves established by boundary conditions on the electric field only. Does this mean that measurement of the external bioelectric field completely determines the external biomagnetic field (or vice versa)? Such a conclusion is clearly suggested, but a direct proof is absent. So while this paper almost certainly eliminates what was once thought to be a definite distinction between the properties of bioelectric and biomagnetic fields it does not completely exclude such a possibility.

REFERENCES

1. Plonsey, R. 1969. Bioelectric Phenomena. McGraw-Hill Inc. New York.
2. Chapman, J. B., J. J. Kootsey, and E. A. Johnson. 1979. A kinetic model for determining the consequences of electrogenic active transport in cardiac muscle. *J. Theor. Biol.* 80:405–424.
3. Hodgkin, A. L. 1964. The Conduction of the Nervous Impulse. Charles C. Thomas Publisher, Springfield, IL.
4. Plonsey, R., and D. Heppner. 1967. Considerations of quasi-stationarity in electrophysiological systems. *Bull. Math. Biophys.* 29:657–664.
5. Plonsey, R., and R. E. Collin. 1961. Principles and Applications of Electromagnetic Fields. McGraw-Hill Inc. New York.
6. Plonsey, R. 1974. The formulation of bioelectric source-field relationships in terms of surface discontinuities. *J. Franklin Inst.* 297:317–324.
7. Geselowitz, D. B. 1970. On the magnetic field generated outside an inhomogeneous volume conductor by internal current sources. *IEEE Trans. Magn.* 6:346–347.
8. Plonsey, R. 1981. Magnetic field resulting from action currents on cylindrical fibers. *Med. Biol. Eng. Comput.* 19:311–315.
9. Swinney, K. R., and J. P. Wikswo. 1979. A calculation of the magnetic field of a nerve action potential. *Biophys. J.* 32:719–732.
10. Durrer, D., R. Th. van Dam, G. E. Freud, M. J. Janse, F. L. Meijler, and R. C. Arzbaecher. 1970. Total excitation of the isolated human heart. *Circulation.* 41:899–912.
11. Roberts, D. E. L. T. Hersh, and A. M. Scher. 1979. Influence of cardiac fiber orientation on wavefront voltage, conduction velocity, and tissue resistivity in the dog. *Circ. Res.* 44:701–712.
12. Plonsey, R. 1974. The active fiber in a volume conductor. *EEE Trans. Bio-Med. Eng.* 21:371–381.
13. Plonsey, R., and Y. Rudy. 1980. Electrocardiogram sources in a 2-dimensional anisotropic activation model. *Med. Biol. Eng. Comput.* 18:87–94.
14. Katila, T. 1981. Instrumentation for biomedical applications. *In* Biomagnetism. S. N. Erne, H. D. Hahlbohm, and H. Lubbig, editors. Walter de Gruyter, Berlin. 3–32.
15. Plonsey, R. 1972. Capability and limitations of electrocardiography and magnetocardiography. *IEEE Trans. Bio-Med. Engr.* 19:239–244.
16. Rush, S. 1975. On the independence of magnetic and electric body surface recordings. *IEEE Trans. Bio-Med. Engr.* BME-22:157–167.
17. Wikswo, J. P., Jr., J. A. V. Malmivuo, W. H. Barry, M. C. Leifer, and W. M. Fairbank. 1979. The theory and application of magnetocardiography. *Adv. Cardiovasc. Phys.* 2:1–67.