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5 August 1957

Multiple Dipole Representation of the Human Heart for Vectorcardiography

Vectorcardiography has progressed to the point where designers of lead systems claim to take into account a multiple dipole representation of the heart. This is stated to be an improvement over considering the heart as a single dipole source, since the heart's volume is indeed appreciable compared with its distances from measuring electrodes, and the electrical activity is spread throughout the heart's wall. The spatial vector loop is then interpreted as a summation of individual dipole contributions, where the perfect lead system is defined as one which gives equal weight to dipoles regardless of their location within the heart volume.

This interpretation leads to pitfalls which bear investigation. Actually, the resulting loop does not contain all the available information concerning a complex heart generator but, indeed, it responds to only the dipole component of such a generator. Only if the heart can be accurately represented by a single dipole of fixed location can a vector loop by itself completely describe its electrical behavior. It is a common misconception among vectorcardiographers that separately located dipole sources can accurately be accounted for by the locus of a single vector loop; for if this were true the next logical (and legitimate) step would be to attribute the loop to a single equivalent dipole.

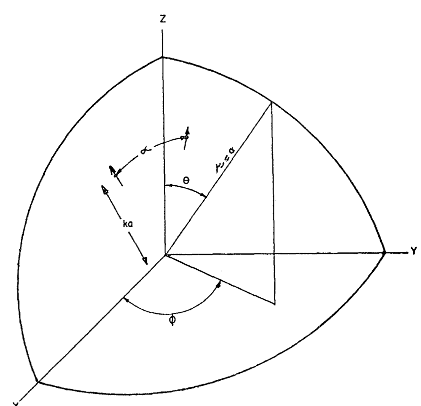

Fig. 1. Sketch showing location of a two-dipole source in a sphere of radius *a*. The dipoles are located and oriented in the *x-z* plane. The eccentricity is given by *k*, which is the percentage of the radius *a*, and the angular separation is given by α. The positive sense of the dipoles is given by the direction of the arrows, which in this sketch represent radially oriented dipoles; θ is measured from the *z* axis, and φ is measured from the *x* axis in the *xy* plane.

The purpose of this study is to show that separately located dipole sources in volume conductors can produce markedly different boundary potentials compared with those produced by a single equivalent dipole source. This single equivalent dipole is assumed to be the dipole component of the complex generator; the difference in boundary potentials represents available information to which the previously defined perfect lead system fails to respond.

Accordingly, boundary potentials produced by two dipole sources in a conducting sphere were calculated and compared with potentials produced by a single equivalent dipole. The equivalence was determined after the Gabor and Nelson (*1*) formulation of equal surface integrals of potential had been applied to a two dimensional disk conductor. The published formulas for two dimensions were found to be in error concerning signs; they should read (*2*):

$$XM_\gamma + YM_x = \gamma\oint V(y\,dy - x\,dx)$$

$$M_x = \gamma\oint V dy\,;\ M_y = -\gamma\oint V dx$$

$$XMy + YM_x = \gamma\oint V(y\,dy - x\,dx)$$

where X and Y are coordinates of equivalent dipole location; M_x and M_y are the component strength of the equivalent dipole; V is boundary potential; and γ is the conductivity.

When these corrected formulas were applied to two dipoles in a circular disk, the equivalent dipole had a strength equal to the vector sum (taken at the same origin) of the individual dipoles and a location based on a "center of gravity" consideration. When these results were extended to the sphere, the strength of the equivalent dipole was still the vector sum of the two dipole sources, and its location was taken as the "center of gravity" of the two sources (Fig. 1). There is, of course, no assurance that this equivalent dipole or one found by actually performing the indicated integrations for location gives the best match as far as actual surface potentials over the entire sphere are concerned. However, the results show that no single dipole can accurately match the two dipole potentials.

The results are shown in Fig. 2 for radial components and in Fig. 3 for tangential components. Two equal strength dipoles located at 40 percent of the sphere's radius and separated by 60 degrees were used since this represents extreme conditions. It is obvious that large errors are inherent in recording only the single dipole component of a two dipole generator. The errors are larger for radially oriented dipoles than for tangentially oriented ones. The comparisons are, of course, worse for increasing eccentricity and angular separation and are perfect for either zero eccentricity or zero angular separation. It should be noted that, if four radial dipoles are symmetrically placed on a cone of revolution, the comparison is vastly improved. Four dipoles, located and oriented thus, simulate a uniform double-layer source, which Frank (*3*) has shown to be very similar to a single dipole.

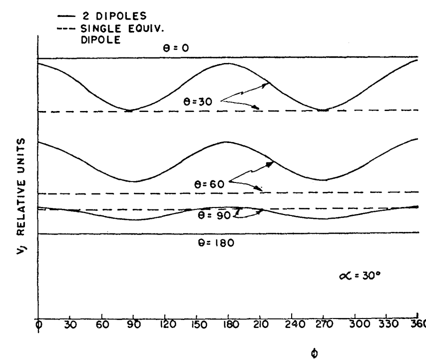

Fig. 2. Potentials over a hemisphere due to two radially oriented dipoles compared with a single equivalent dipole; φ and θ are spherical coordinates illustrated in Fig. 1. The dipoles are located and oriented in the planes of φ = 0 degrees and φ = 180 degrees, with an eccentricity of 40 percent of the radius and an angular separation of 60 degrees. The positive sense is away from the origin for both dipoles. Note that θ = 0 and 180 degrees are single points on the sphere surface.

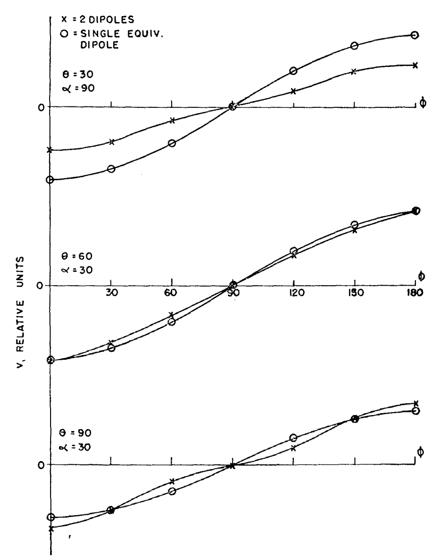

Fig. 3. Same as Fig. 2, except that the dipoles are tangentially oriented with an additive sense.

This study has shown that, *if* the electrical activity of the human heart *cannot* be represented by the action of a single equivalent dipole, a single spatial vector loop by itself cannot possibly contain all the available information. Indeed, if the evidence that diseased hearts are non-dipolar is verified, there would be a strong argument for the necessity of some type of precordial scalar leads.

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2. This can be shown by deriving the two-dimensional case from the same hypotheses and retaining individual unit vectors. Thus in place of dy we obtain $(\vec{a}_x \cdot \vec{n})dl$ and for dx we ob- $(\vec{a}_y \cdot \vec{n})dl$ where $\vec{a}_x$ and $\vec{a}_y$ are unit vectors in the x and y direction, respectively, and $\vec{n}$ is a unit vector normal to the path of integration. For a counterclockwise direction of integration, $(\vec{a}_x \cdot \vec{n})$ is positive for positive dy and negative for negative dy. For the same direction, however, $(\vec{a}_y \cdot \vec{n})$ is positive for negative dx and negative for positive dx. Thus, for the published equation to be correct (assuming a counterclockwise direction of integration), every dx should be replaced by a minus dx.
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4. This study was supported in part by research grant H-339-C from the U.S. Public Health Service.

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Equipotentiality Versus Cortical Localization

In an otherwise excellent paper, Weinstein and Teuber recently presented (*1*), without taking specific notice of it, clear-cut evidence that the crux of a possible scientific *synthesis* of the opposite sides of the "equipotentiality-versus-cortical-localization" controversy resides in the different kinds of intelligence for which one tests.

These authors correctly infer that Lashley's (*2*) position tends to be substantiated by their findings that "injury to any lobe of the brain, in either hemisphere, can interfere with performance on certain nonlanguage tasks." Furthermore, they state that Lashley's view of equipotentiality is opposed, as is Rylander's [that is, impairment is maximal following injury to the frontal regions (*3*)], by their findings that "performance on a standardized test of 'general intelligence,' such as the AGCT [Army General Classification Test], . . . shows little or no change 10 years after penetrating brain wounds unless the entrance wound included the left parietotemporal region."

They fail, however, to note that this latter statement strongly supports Nielsen's (*4*) views about the validity of the cortical localization concept—that is, that the cortical areas on the *major* side of the brain may be differentiated in terms of their language functions. Moreover, their analysis, as shown in Fig. 1, tends to confound the issue by failing to make specific comparison of the aphasics with the nonaphasics in their experimental sample. Had this been done, the picture of what happens when the left parietal lobe is alone injured would have stood out more clearly.

It appears to me that the results obtained by Weinstein and Teuber could have been predicted from Nielsen's position (*4, 5*), yet no reference is made to him in their article. This is an oversight, because the AGCT seems to be for the most part a test of verbal intelligence (*6*) and, therefore, well suited to testing hypotheses in this area. Their results, which I believe are a solid contribution to the field, happily seem to square nicely with my recent statement (*7*) "that, while there are exceptions on both sides, *animal* experimentation continues to support Lashley's theory of mass action and equipotentiality, but the literature dealing with aphasia in *humans* tends more and more to substantiate Nielsen's confirmation of the classic teaching of cerebral localization. The crux of the disagreement, which by the way is so often overlooked by critics of cerebral localization, is just this: cerebral localization in aphasia deals with language, and language is the most important difference between animals and man"

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4 October 1957

In his comment on our paper (*1*), Holmes emphasizes that our results support a fairly definite localization of language skills in the left hemisphere of the brain. His interpretation is based on a point that we have stressed ourselves, namely that the Army General Classification Test is a verbal instrument. Conceivably even slight traces of aphasia, undetected by other means, might be revealed by inferior performance on the test. We fully agree with Holmes' comment with regard to this possible interpretation.

Holmes further regrets that we fail to compare aphasics with nonaphasics. This comparison was deliberately omitted because of the obvious nature of the results: aphasia is an impairment of language, and the AGCT is a verbal test. However, the comparisons desired by Holmes can be reconstructed from the data contained in our Fig. 1 (*1*). The mean and N of each group are given before and after elimination of aphasics. Thus, the mean drop in score of the group of 10 men (including aphasics) with a left parietotemporal lesion was 18.70. The mean drop in score of the same group after elimination of the four aphasics was 11.17. Arithmetic computation reveals the mean loss of these four aphasics to be 25.01. Incidentally, our Fig. 1 was set up specifically to show "what happens when the left parietal (and other) lobes are alone injured"; what happened was a significant drop in AGCT score after left parietotemporal lesions, and the absence of such a drop after lesions in other parts of the brain.

We find it difficult to follow Holmes when he says that our findings confirm Nielsen's views on localization of function in man. In our brief article we could not enter into the complex history of this field, but it seems clear that a preponderant role of the left hemisphere in man (particularly the left parietotemporal region) for language skills has been observed for nearly a century. In this respect, our findings seem to need no emphasis, since they merely reconfirm what is well known; but just for this reason we cannot see how that laterality difference could specifically support Nielsen's views.

Holmes apparently sees the issues of localization in terms of the classical dichotomy of specific and general effects. He considers Lashley's findings in the rat as an instance of general (nonlocalized) change after cortical removal and invokes Nielsen as an exponent of specificity for man—that is, that every lesion which produces any symptoms produces symptoms of a different kind, depending on its location.

The findings from our laboratory have induced us for some time to reject this dichotomy. Our work has shown rather consistently that brain injuries in man tend to produce twofold effects, "specific" (localizable) and "general" (nonfocal) alterations (*2*). Thus, all subgroups of the population we have studied have shown deficits on certain perceptual