Experimental Evidence for Regional Cardiac Influence in Body Surface Isopotential Maps of Dogs

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SUMMARY Isopotential maps based on 192-200 body surface electrocardiograms were obtained for 20 dogs during multiple patterns of ventricular activation. The purposes of the study were to determine whether the cardiac location of events responsible for surface potentials had a recognizable influence on surface potential patterns and to examine the influence of electrical events occurring simultaneously in multiple cardiac regions. Substantially different effects of electrical activity in various cardiac regions on body surface potentials were evidenced by the body surface location of potential maxima and minima and by patterns of isopotential lines during early portions of ventricular excitation initiated at different ventricular sites. Simultaneous stimulation at some sites gave surface potential distributions with multiple extrema. These were demonstrated to be due to effects of the different cardiac regions, because addition of potentials due to stimulation of the individual sites duplicated those associated with simultaneous stimulation of the same sites. It was also shown that body surface locations of maxima and minima are not related in the same manner to the cardiac location of the responsible events when these events are present in single and multiple regions. Slopes of potentials due to events in single cardiac regions were shown to combine with slopes produced by events in other regions to yield maxima or minima at new body surface locations. Results of the study support the possibility of regional cardiac examination by electrocardiography but suggest that this will require quantitative descriptions of the details of potential patterns in addition to the location of potential peaks.

EXAMINATION with large numbers of leads may permit evaluation of individual cardiac regions and extend the range and precision of electrocardiographic (ECG) diagnosis. The acquisition and analysis of large amounts of ECG data have been facilitated by simultaneous recording, using multiplexing circuitry, and by computer processing. These methods, supplemented by future technical improvements, can be expected to make examination with large arrays of electrodes practical for clinical application. Diagnostic use of extensive ECG examinations also requires correlation of findings with various cardiac states including specific disease processes. This is a formidable task, and theoretic and experimental studies of mechanisms responsible for the observed phenomena, as well as clinical correlational studies, are required to define the method's diagnostic utility.

The experimental study reported here was conducted to examine some diagnostically significant relationships between the state of the heart in specified regions and its expression in body surface ECG potential patterns. The study was made on dogs. Some observations were made with the chests intact and others after midline thoracotomy and reclosure of the chest wall. The heart was stimulated at various sites, and these sites were stimulated singly or in various combinations. Stimuli were pulses of 2-msec duration and 1½ to 2 times threshold intensity. In some experiments catheter-mounted stimulating electrodes were placed in the atrium and right and left ventricles without thoracotomy; 192-200 electrodes were placed symmetrically on the thorax. Placement of the body surface electrodes required 1–1½ hours, therefore that interval elapsed between placement of cardiac stimulating electrodes and the ECG recording. In the experiments which required thoracotomy, saline-dampened sponges were packed loosely in the open thorax prior to closure. Similar preparations have been used previously to improve the volume-conductor properties of the thorax after thoracotomy. Data were recorded sequentially from banks of six electrodes and a common reference lead in 15 experiments. In the other five experiments from 192 symmetrically arranged torso electrodes were recorded simultaneously on magnetic tape using multiplexing techniques which have been described

Methods

Experiments were performed on 20 dogs anesthetized with sodium pentobarbital (30 mg/kg, iv). Some observations were made with the chest intact and others after midline thoracotomy and reclosure of the chest wall. The heart was stimulated at various sites to produce a variety of cardiac sources whose locations were known. After thoracotomy, bipolar stimulating electrodes were placed on the atrium and at various ventricular sites, and these sites were stimulated singly or in various combinations. Stimuli were pulses of 2-msec duration and 1½ to 2 times threshold intensity. In some experiments catheter-mounted stimulating electrodes were placed in the atrium and right and left ventricles without thoracotomy; 192–200 electrodes were placed symmetrically on the thorax. Placement of the body surface electrodes required 1–1½ hours, therefore that interval elapsed between placement of cardiac stimulating electrodes and the ECG recording. In the experiments which required thoracotomy, saline-dampened sponges were packed loosely in the open thorax prior to closure. Similar preparations have been used previously to improve the volume-conductor properties of the thorax after thoracotomy. Data were recorded sequentially from banks of six electrodes and a common reference lead in 15 experiments. In the other five experiments data from 192 symmetrically arranged torso electrodes were recorded simultaneously on magnetic tape using multiplexing techniques which have been described.
Sequential records from electrode banks were time-aligned by using detection of the R waves or, when the heart was being driven, stimulus artifacts. In the latter case the time reference of the first QRS isopotential maps includes stimulus duration and latency. Data from 10-20 complexes were averaged to reduce effects of ambient noise in the sequentially recorded data. Multiplexed data from single complexes were analyzed.

Data were computer-processed with a PDP-15 system. Potentials were interpolated using a bicubic polynomial and plotted as isopotential maps at 2-msec intervals during the QRS and at 5-msec intervals during the ST-T deflection.

Body surface isopotential patterns were described in relation to the responsible cardiac events. It was not feasible to map the activation sequence in the experiments reported here because of the time required for the extensive ECG examinations. Studies by others have furnished a detailed description of normal ventricular activation and also that which occurs during bundle branch block and ectopic ventricular stimulation. These data provide a description of cardiac events which is applicable to the present study. We compared the location of potential maxima and minima and the form of isopotential lines of maps associated with ventricular drive at various sites with each other. Cardiac events were described in terms of dipolar "sources" from individual cardiac regions. As employed in this study, "source" denotes an electrical event in a particular cardiac region such as right or left ventricle, apex or base, or anterior or posterior ventricular wall.

Some of the data were analyzed with arithmetic operations described by McLaughlin. Potentials due to single cardiac sources were added and isopotential patterns based on their sum were calculated. Such "addition maps" were compared with maps based on recorded potentials when both sources were actually present in the heart.

**Results**

Our major objective was to determine whether body surface potential patterns were substantially affected by the cardiac location of the responsible events. A further objective was to describe the cardiac location-surface potential relationship in terms likely to assist in the development of diagnostic criteria for evaluation of detailed body surface potential patterns.

The cardiac source-surface potential relationship which is fundamental in electrocardiography is recognized to be complex. Source orientation and strength, volume-conductor characteristics of the body, and source location all are factors in the relationship. This multiplicity of factors makes it difficult to rigidly prove and quantitatively define roles of each. Despite this, findings in the present study strongly suggest that body surface potential patterns are substantially affected by the cardiac location of responsible events. An example of the data on which this conclusion is based is shown in Figure 1. Isopotential maps in that figure illustrate the potential distribution 40 msec after application of stimuli at twice threshold intensity to epicardial ventricular sites through bipolar electrodes. Stimulus sites were sepa-
rated by 3 cm; one was located to the right and the other to the left of the interventricular groove.

Isopotential maps shown in Figures 1A and B are the surface potential patterns associated with stimulation of the right and left ventricular sites, respectively. Each shows a single potential maximum and single minimum. Simultaneous stimulation of the same sites was associated with the map shown in Figure 1C, in which two minima are present. It is reasonably certain that the two minima illustrated are the result of the different cardiac locations of the two sources involved. Activation fronts after stimulation of the individual sites which constitute the cardiac sources cannot be assumed to be identical in strength and orientation but definitely do have different cardiac locations. This is supported by the fact that in maps made at earlier times after stimulation of each site the location of poles was similar to those illustrated. It is supported further by the observation from the same experiment illustrated in Figure 1D. That map was associated with simultaneous stimulation of sites separated by only 1 1/2 cm and shows less widely separated minima than those associated with the more widely separated stimulus sites.

Other examples of probable effects of the location of cardiac events on surface potential patterns are shown in Figure 2. It shows isopotential maps for selected intervals during a QRS which resulted from stimulation of the left ventricular apex through catheter-mounted electrodes in a preparation with an intact chest. The sequential maps show the location of the positive pole on the body surface moving upward and to the right in correspondence with the obligatory changes in the cardiac location of ventricular excitation.

Still other examples of effects likely to be due to the different cardiac locations of responsible events are shown in Figure 3. The maps shown are for a late portion of the QRS complex after stimulation of a site near the interventricular septum in two different experiments. The two negative poles in each map are compatible with excitation at two cardiac locations which, under the conditions of the experiments, were likely to be in right and left ventricles.

A probable relationship of surface potential patterns to cardiac source location was evident in all experiments, but the magnitude of effects of source on pattern was variable. Figure 4 shows maps made after independent and simultaneous stimulation of sites separated by 3 cm and located on the epicardial surface of the posterior ventricular wall; the contour of isopotential lines and body surface locations of maxima and minima differ between these maps. The simultaneous presence of both cardiac sources is evidenced by two low level minima on the posterior thorax. Two sources with the same separation on the anterior cardiac surface resulted in multiple minima of greater intensity, as illustrated in Figure 1. These findings suggest that body surface potential patterns are less sensitive to individual regions of the posterior than the anterior cardiac wall. Cancellation of ECG effects by an opposing direction of activation from the two posterior sites cannot be excluded but seems unlikely in that sites were located on a relatively smooth portion of the ventricular wall.

Addition of maps resulting from single dipolar cardiac sources provided diagnostically important insights concerning the distribution of surface potentials when multiple dipolar sources were present simultaneously. An example is shown in Figure 5. These maps were taken from a dog with an intact chest and catheter electrodes in the apices of right and left ventricles. The figure shows maps due to excitation initiated from each of the two stimulus sites, one due to activation from simultaneous stimulation of the same sites, and an addition map. Even though the cardiac sources had the same locations with simultaneous stimulation two distinct maxima are present, but their locations differ from the locations of the maxima when the sites were stimulated individually. The addition map and the map due to simultaneous stimulation of the two sites are identical within the range of experimental and computing errors. These maps demonstrate the influence multiple sources have on body surface potential patterns.

Another example of the complexity of the surface potential pattern and multiple cardiac source relationship is
shown in Figure 6. This illustration shows maps of potential distribution due to two widely separated sources, one on the epicardial surface of the apex of the left ventricle and the other on the epicardial surface of the base of the free left ventricular wall. When these sites were stimulated individually, each of the two sources produced minima at widely separated body surface locations. When both sources were present simultaneously, potentials combined in such a way that potential slopes associated with the individual sources formed a single minimum at a new body surface location. Since the minimum at the new body surface location was not the result of a new location of the dipolar cardiac sources, the findings again illustrate that the body surface location of maxima and minima cannot be used in any simple way to identify cardiac regions responsible for body surface potentials when multiple dipolar sources are present. The addition map closely resembles the map associated with simultaneous stimulation and provides further demonstration that potential slopes combined to form a single minimum in a new location. Similar findings were obtained in all experiments.

Figure 3. Body surface isopotential maps from two dogs after thoracotomy and reclosure of the chest wall. Maps shown in both A and B occurred during late portions of the QRS complexes after stimulation of sites near the interventricular septum (A = 88 msec and B = 64 msec poststimulation). Both maps show two minima which are compatible with separate excitation fronts in right and left ventricles. Isopotential contours are drawn for increments of 100 μV.

Figure 4. Body surface isopotential maps from one dog after thoracotomy and reclosure of the chest. Maps in A and B show potential patterns 25 msec after stimulation of each of two sites separated by 3 cm on the posterior cardiac wall. The map in C shows potentials 28 msec after simultaneous stimulation of the same sites. The body surface location of maxima and minima differ for the three conditions. There are low level, negative potentials over the lower thorax which form two minima but these are so low in amplitude that it is difficult to draw definite conclusions about them. Isopotential contours are drawn for increments of 100 μV.

in which there were multiple dipolar sources during early portions of ventricular excitation. During later parts of ventricular excitation, wavefronts of excitation initiated by multiple site stimulation collide and the cardiac states differ from the states due to stimulation of the single sites.

Discussion

The possibility of ECG evaluation of individual cardiac regions has major potentialities for improved cardiac diagnosis. ECG examination is noninvasive, virtually free of risk, and of proven utility even when the heart is assumed to be a single electrical source which varies in strength and orientation, as in the case of vectorcardiographic study, or a dipole which provides the basis for interpretation of the 12-lead electrocardiogram. Examination with large arrays of electrodes is a promising approach to regional cardiac examination, although useful application presents many scientific and technical problems. Results of the present study contribute to the solution of some of these. On the technical level, we employed the simultaneous recording of a
FIGURE 5  Isopotential maps from one dog with an intact chest recorded 24 msec after stimulation through catheter-mounted electrodes in the right and left ventricles. The map shown in A followed stimulation of the right, and that in B, stimulation of the left ventricle. The map shown in C followed simultaneous stimulation of the right and left ventricles, and that shown in D is the result of addition of potentials from maps A and B. The addition map and that resulting from simultaneous right and left ventricular stimulation show two maxima but the body surface locations of these maxima differ from that which resulted from stimulation of the individual sites. From the addition map it is evident that potential slopes from maps A and B have combined to form potential peaks at new surface locations as a consequence of stimulation of both sites. Isopotential contours are drawn for increments of 100 µV.

A large number of electrocardiograms using multiplexing circuits, and demonstrated its practicality. The study also provides evidence that body surface potential patterns are substantially influenced by the cardiac location of responsible events. This evidence was obtained from dogs: although it is not necessarily applicable to humans, substantial similarities have been demonstrated between normal isopotential maps for dogs and humans. Although regional cardiac influence on body surface potentials was clearly evident when single cardiac regions were active, the study...
also provided evidence of the complex expressions of multiple cardiac regions which act simultaneously. It was demonstrated specifically that the body surface location of potential maxima and minima cannot always be equated simply with the cardiac location of responsible events when multiple regions are active simultaneously. The body surface location of maxima and minima are, however, only one feature of body surface potential patterns. The contour of isopotential lines, the potential gradients at various locations, and the time sequence of changing potential values, gradients, and isopotential contours also are descriptors whose utility for regional cardiac examination should be examined.

Findings from this study suggest that individual cardiac regions have different effects of body surface potential patterns and support the possibility of regional cardiac examination by electrocardiography. Our findings also demonstrate, however, that simultaneous effects of multiple cardiac regions have complex surface potential manifestations. The use of arithmetic manipulations of maps provided insights concerning the distribution of potentials when more than one dipolar cardiac source was present, and this method is likely to be useful in analysis of maps for clinical diagnosis. Separation of effects due to individual cardiac areas, when multiple areas have simultaneous effects, is the major problem for development of regional electrocardiographic examination of the heart.

Reference


Maintained Stroke Volume but Impaired Arterial Oxygenation in Man at High Altitude with Supplemental CO₂

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SUMMARY Hypobaric hypoxia causes hypocapnia and alkalosis, hemoconcentration and increased hematocrit, and a decreased cardiac stroke volume. To assess the role of the hypoxic alkalosis in causing these other changes, five men were exposed to hypobaric hypoxia at a barometric pressure (Pb) of 440 torr with an alveolar O₂ tension of 55 torr for 5 days with 3.77% CO₂ added to the atmosphere to prevent alkalosis. They did not lose weight, and arterial PO₂, tension of 55 torr. By contrast, three men at high altitude without CO₂ supplementation (Pb = 455 torr; alveolar PO₂ = 56 torr) had weight loss, hypocapnia, alkalosis, and decreased stroke volume. Increased hematocrit suggested decreased plasma volumes. During exercise, arterial PO₂ (48 torr) was higher than in the group receiving CO₂. Maximum oxygen uptakes were decreased to a similar degree in the two groups. Catecholamine excretion doubled in the group with CO₂ but in the group without CO₂ catecholamine excretion was unchanged. A normal pH at high altitude apparently maintained plasma volume, which, with the increased catecholamine excretion, may have prevented a decrease in stroke volume. However, the subjects with CO₂ added did not have enhanced oxygen transport, because their arterial oxygenation was impaired.

A DECREASED cardiac stroke volume of unknown cause is largely responsible for the decreased oxygen trans-
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