Effects of Pericardial Effusates of Various Conductivities on Body-Surface Potentials in Dogs

Documentation of the Eccentric Spheres Model

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SUMMARY. The purpose of this study was to discover the cause and magnitude of changes in the body-surface potentials occurring when: (1) fluids of various conductivity were added to the pericardial sac, or (2) the volume of the blood within chambers of the heart was either increased or decreased. Fluids added to the pericardium were physiological saline, whole-blood, and mineral oil. Magnitudes of body-surface potentials were compared to the predictions based on a mathematical eccentric spheres model of the heart and torso developed previously by Rudy and Plonsey. Data demonstrated conclusively that there is a nonlinear relationship between the body-surface potentials and the conductivity of the pericardial layer. This relationship is one in which the body-surface potentials of the anterior chest were found to decrease when conductivity of the pericardial layer was either increased or decreased. These changes in body-surface potentials were caused solely by alterations in the conductivity and volume of the fluid effusate. It was demonstrated that these changes were not caused by any "stretching" or "compression" of the cardiac tissue caused by the altered fluid volumes in and around the heart. Findings were accurately predicted by the eccentric spheres model, thereby confirming the model's usefulness as a predictive instrument. The model provides an explanation for the nonlinear relationship that was exhibited by the data. (Circ Res 55: 788–793, 1984)

RUDY and Plonsey (1979a, 1979b, 1980) proposed an eccentric spheres model of the heart and torso capable of predicting the effect of pericardial effusions of varying conductivity on the amplitude of body-surface potentials generated during ventricular activation. Based on the results obtained from this mathematical model, they proposed that the surface potentials recorded from the chest would decrease due to the development of a pericardial effusion having an effusate conductivity either above or below that of myocardium (normal), whereas effusates similar in conductivity to myocardium would produce only slight changes in amplitude. In addition, these predicted results were due solely to changes in the conductivity of the medium and were not due to effusate "compression" of the heart. This study was conducted to ascertain the possible mechanism(s) for alterations in voltages of the QRS seen on the body-surface that might have been produced by: (1) compression of the heart, (2) alterations in the ventricular activation sequence, or alterations in fluid conductivity and/or volume, either within the heart or within the pericardial sac.

Methods

Ten healthy mature dogs were used in this study. Their weights varied between 35 and 45 pounds. They were anesthetized and maintained in surgical anesthesia with fentanyl-droperidol-pentobarbital and were ventilated with a Harvard respirator. Four dogs were used to evaluate the effects of acute reduction or increase in ventricular lumenal volume on the ventricular activation sequence. In this portion of the study, five unipolar electrodes were sutured to the epicardium (Fig. 1). The five epicardial leads were designated as RVb, RVa, LVb, LVa, and LVs. Lead RVb was placed over the right ventricular base. Lead RVa was placed over the right ventricular apex. Lead LVb was placed over the area of the left ventricular base at the free-wall. Lead LVa was placed over the left ventricular apex. Balloon-tipped cardiac catheters were placed in both vena cavae and in the ascending aorta. While electrograms from the epicardial electrodes were recorded on an E for M photographic oscillograph (low frequency cutoff = 120 Hz), the balloons in the vena cavae were inflated, thereby obstructing nearly all venous return. The heart could be observed to decrease in size (Fig. 2). After a 5-minute recovery period, the balloon in the ascending aorta was inflated, thereby obstructing the left ventricular outflow. The heart could be observed to increase in size (Fig. 2). Thus, epicardial electrograms were recorded during acute changes in heart size. If configuration and timing of these electrograms remained unchanged from normal during the balloon inflations, we presumed that the pathways of ventricular activation remained unchanged. To ascertain whether known changes in ventricular activation could be detected by the evaluation of only 5 unipolar epicardial electrograms, we induced premature ventricular beats by pricking the epicardium at numerous positions on the right and left ventricles.
Six dogs were used to evaluate the effects of various conductivity pericardial effusions on the body-surface potentials. An 8F NIH-tip cardiac catheter was placed into the pericardial sac via a median sternotomy. The thorax was closed and the pneumothorax reduced via a thoracic drain, which was kept under negative pressure as the dog was permitted to breathe normally. Circumferential thoracic unipolar electrocardiograms, along with lead Vr, were recorded from six positions placed symmetrically with respect to the median sagittal plane, and at the 5th to 6th intercostal space (Fig. 3). While monitoring the circumferential thoracic ECGs, a 60-ml pericardial effusion was produced (by way of the pericardial catheter), first, with saline (conductivity = 15 × 10⁻³ mho/cm), next with blood drawn from the animal prior to the experiment (conductivity = 6 × 10⁻³ mho/cm), and last with mineral oil (conductivity = 50 × 10⁻⁶ mho/cm). Each of the fluids was withdrawn before the next fluid was injected. All of these effusates were maintained at 39°C prior to their injection into the pericardial sac. The electrocardiograms were compared for changes in QRS amplitude before and after the various effusions.

The measurements taken from the pericardial effusion ECG recordings were QRS peak amplitude, translated to percentage of the control QRS peak amplitude. The resulting tables were treated by an analysis of variance and covariance with repeated measures using the Greenhouse-Guysyar alpha correction factor (BMDP Statistical Software, Inc.-Program 2V). A one-sided t-test was used due to the a priori design of the experiment (Sokal and Rohlf, 1969).

**FIGURE 1.** Schematic diagram of the left lateral view of the heart with locations of epicardial electrodes. R stands for right, L stands for left, a stands for apex, b stands for base, and FW stands for free-wall.

**FIGURE 2.** Left lateral radiographs of the thorax showing changes in the ventricular size produced by inflation of intravascular balloons. Upper left is with no balloon inflation. Upper right is with inflation of balloons in both vena cavae, therefore obstructing return of flow to the heart which produces reduction in ventricular volumes. Lower left is with inflation of balloon in the pulmonary trunk, obstruction of right ventricular outflow, and increase in right ventricular volume. Lower right is with inflation of balloon in ascending aorta obstructing left ventricular outflow and producing left ventricular dilation.
FIGURE 3. Schematic drawing of dog on its back, showing location of circumferential thoracic electrodes.

Results

Effects of Ectopic Ventricular Beats and of Varying Ventricular Volume on the Epicardial Activation Sequence

Configuration of epicardial electrograms was consistent from beat to beat; however, when premature beats were induced from any focus, configurations, but mainly timing of the depolarization spikes, changed noticeably (Fig. 4). When ventricular volumes were changed by inflation of the balloons, neither timing nor configurations of the electrograms were altered significantly.

Effects of 60-ml Pericardial Effusions, with Media of Various Conductivities, on the Configuration of the QRS of Circumferential Thoracic Unipolar Electrocardiograms

Configurations of the electrocardiograms were relatively consistent from beat to beat; however, variations were observed during ventilation, so only those beats occurring during the respiratory pause were compared. In general, QRS amplitudes in all leads from all animals decreased with an effusion of saline or mineral oil (Figs. 5 and 6). Changes in voltages for each effusate, recorded at each lead, are shown quantitatively in Tables 1 and 2 and in Figure 7. It can be observed that changes of voltages in V1 and V3 were generally greater than for any other lead. It may also be noted that, although the mineral oil to blood comparison for lead V7 is significant, it is so if the mineral oil mean is greater than the blood mean. Thus, a linear relationship is described by the surface potentials of lead V7 for the three effusates.

Discussion

Voltages of QRS decreased significantly and greatly with effusions of saline (high conductivity) or mineral oil (low conductivity), and varied insignificantly from normal with effusions of autologous blood. Since all of the effusions were of identical volume (60 ml), and since there was no change in the epicardial activation sequence when the heart was decreased in size by obstructing the venous return (i.e., simulation of cardiac compression), we could not incriminate cardiac compression and either a Brody effect (1956) or altered ventricular activation as the causes of the observed reduction in body-surface potentials. Therefore, the reduction in voltages produced by effusates of conductivity different than blood, relative to the reduction in voltages produced by blood, and the reduction in voltages produced by the blood itself were all attributable to the electrical field theory effect proposed by Rudy and Plonsey (1979a).

A 60-ml pericardial effusion was selected because it provides for a visceral-parietal pericardial dimension of approximately 5 mm, which is analogous to the dimension used by Rudy and Plonsey (1979a) in their mathematical calculations.

We recorded circumferential thoracic electrocardiograms; whereas the model of Rudy and Plonsey (1979a, 1979b, 1980) predicted potential changes for only a single point comparable to our lead V3. Changes in lead V3 appeared to be greater than for any other lead; in fact, voltages in the leads on the right hemithorax and over the cardiac base (V10) changed very little. The model of Rudy and Plonsey (1979a) predicted such variations in voltage with variations in the orientation between the electrodes and the mean QRS vector.

No explanation is available for the linear relationship found for lead Vf, other than the fact that Rudy and Plonsey’s model was not designed to predict voltages for distal leads.

Our conclusions differed from those of Manoach et al. (1972), who stated that cardiac compression, and not a field effect, was responsible for the diminution of body-surface potentials with effusion. His control studies were conducted on open-chested cats in which there is an obvious violation of the noninvasive continuous-current flow restrictions imposed by the eccentric spheres model of Rudy and Plonsey (1979a). For the future, we believe that any studies attempting to reconcile ventricular activation with the distribution of body-surface potentials must be conducted on animals with a closed chest (i.e., a noninvasively designed experiment).
FIGURE 4. Bipolar epicardial electrograms recorded from locations outlined in Figure 1 during interventions outlined in Figure 2. Notice nearly identical timing and configuration for electrodes independent of heart size. With left ventricular premature depolarization and obvious changing in sequence of ventricular activation, notice the profound changes in timing and in configuration of epicardial electrograms.

FIGURES 5 and 6. Circumferential unipolar electrocardiograms obtained after instillation of medium of varying conductivity into the pericardial sac. Notice that QRS voltages remained nearly constant with controls when effusate was autologous blood, but that effusate with either mineral oil (low conductivity) or saline (high conductivity) produced reductions in voltages.
This study supports the mathematical calculations of Rudy and Plonsey (1979a) relating the amplitude of body-surface potential to the conductivities of, and boundary relations between, concentric rings of tissue surrounding the heart—which is eccentrically located within a torso. It demonstrates that there is no simple relationship between the volume of pericardial effusate and changes in the QRS voltage without consideration of the conductile properties of the effusate.

Results from this study may be applied to the conclusion reached by Unverferth et al. (1979), which is that the only practical way to detect the clinical condition of pericardial effusion with medium of conductivity different than that of blood (i.e., saline type effusion) is to be able to compare one of the patient's pre-effusion ECGs with a post-effusion ECG. These results indicate that, for a saline-type effusion, one should observe a decrease in the potential of the chest leads (predominantly $V_1$ and $V_3$) from the pre-effusion ECG to the post-effusion ECG. If an echocardiogram indicates that

![Figure 7](http://circres.ahajournals.org/DownloadedFrom)

**Figure 7.** Percentage of normal R-wave amplitudes remaining after instillation of various pericardial effusates. S is saline, B is blood, and MO is mineral oil. Values are means and 95% confidence intervals. Means for saline and mineral oil are significantly different from blood in all leads.
an effusion is present, but no noticeable decrease in chest lead potential is evident, then there is a greater likelihood that the effusate is blood.

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