Effect of Intracardiac Blood on the Spatial Vectorcardiogram

I. RESULTS IN THE DOG

By Clifford V. Nelson, Peter W. Rand, Evangelos T. Angelakos, and Paul G. Hugenholtz

ABSTRACT

Studies were done on dogs in vivo to determine the effect of intracardiac blood on the electrocardiogram. By exchange transfusions of packed cells or of Rheomacrodex, hematocrit was raised to over 70% or lowered to 16%. The electrical resistivity of the blood changed accordingly from control values of 150 ohm-cm to over 500 ohm-cm or to as low as 67 ohm-cm. The x-, y-, and z-vector leads were processed by analog computer. Computer output signals were: spatial dipole moment, M, vertical angle, V°, and horizontal angle, H°. Three peaks of M (M1, M2, and M8) were found at mean times of 29%, 42%, and 64% of QRS duration. Increases in blood resistivity to values near that of the myocardium caused M1 to decrease to 33% and M2 to 40% of control values, but M8 rose to 125% of control values. These results are in accord with radial excitation for M1 and M2 and tangential excitation for M8. Decreases in hematocrit below control values caused opposite changes. Normal intracardiac blood causes an apparent increase of M1 by a factor of 3 and of M2 by 2.5 and a decrease in M8 of about 25%.

KEY WORDS 
- dipole moment
- blood resistivity
- inhomogeneity
- thorax resistance
- end-diastolic volume
- electrocardiogram
- hemocoencentration
- hemodilution

The relationship between the current dipole moment of the heart and the surface electrocardiogram (ECG) is influenced by many factors. An increase in magnitude of the ECG may be caused by a greater size or number of dipoles or by decreased cancellation within the current generator itself. This is the conventional explanation for changes in spatial magnitude in ventricular hypertrophy or infarction. Of equal importance, however, are factors which affect transfer impedance, such as the position of the heart in the thorax, the resistivity of body tissues, the amount of air in the lungs, and the presence of intracardiac blood.

Previous studies by our group (1–3) have added experimental evidence to the model studies of Brody (4) and Bayley and Berry (5), which indicated a strong augmentative effect of the lower resistance of intracardiac blood (about 140 ohm-cm instead of 480 ohm-cm for muscle) on radial dipoles and an opposite attenuating influence on tangential dipoles. Since heart excitation studies by Scher and Young (6) and Durrer and co-workers (7) have shown that radially directed dipoles predominate during initial phases of ventricular depolarization and that tangential forces...
are greater in later phases, one might expect the influence of intracardiac blood on the QRS wave to be complex. In addition, since the QRS wave is generated at the time when diastolic ventricular volume is largest, it is possible that variations in end-diastolic volume will affect the ECG by altering the total quantity of low-resistance blood. Finally, if the electrical resistivity of the blood could be made equal to that of the myocardium, a more nearly homogeneous electrical situation would result and more accurate values of the "real" dipole moment would be obtained. The present study was conducted to test these postulates in intact animals.

**Methods**

Seventy-four sets of measurements were made in 17 dogs (9.1–21.1 kg), anesthetized with sodium pentobarbital (30 mg/kg, iv). All dogs were heparinized with a mixture of 1% heparin in saline. Five were ventilated mechanically; the remainder, after endotracheal intubation, breathed spontaneously. The right femoral vein and artery were cannulated for withdrawal of blood samples and infusion of pharmacological agents. The arterial catheter tip was positioned fluoroscopically at a point midway between the base of the aortic valve and the apex of the left ventricle. Left ventricular pressure was measured intermittently. In several dogs, femoral arterial blood pressure was monitored as well.

In two experiments (one hemodilution, one hemoconcentration) dye-dilution curves were obtained after injection of Cardio-Green (indocyanine green) into the left ventricle. The curves were obtained in the root of the aorta with a fiberoptic hemoreflection system (American Optical Co.). The output—the ratio of reflected light intensity at 910μ to that at 805μ—was also fed into an integrator so that cardiac output could be read directly during the procedure. Details of the calibration procedure and of calculation of cardiac output, ejection fraction, and end-diastolic volume have been given previously.

The lead system developed by Nelson et al. (10) was used for measurement of vectorcardiograms. Prior to placement of the catheters, the dog's chest was shaved and measurements were made of the distance between the top of the shoulders and the junction of the hind limbs with the body (inguinal region). (The distance between the top of the shoulders and a point midway between the xiphoid and inguinal region is now used.) This distance was divided into fourths, and 8 electrodes were put on each of the 8 fourths of this distance.

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Note: V1, M2, M3, and M4 = leads 1, 2, and 3 of the dipole moment, respectively. See text for discussion.

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### Table 2

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VCG = vectorcardiogram. Other abbreviations are the same as in Table 1.

*For experiments 1 and 2, P = mean arterial blood pressure; for experiment 3, P = left ventricular systolic pressure.

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Three levels. Voltages from these 24 electrodes were applied to a resistance network for determination of the x and z voltages. A foot-head lead was used for the vertical component.

The x, y, and z voltages from the resistance network were amplified and, along with a respiration signal, applied to a seven-channel magnetic tape recorder. Records were taken at 60 inches/sec, replayed at 15 inches/sec, giving a time expansion of 32, and fed into a direct-writing recorder. The paper speed and amplification were such that high resolution was obtained. For each record, five expiratory QRS complexes were measured and averaged at 2.5-msec intervals. In most experiments the x, y, and z voltages were also applied to an EAI model TR-20 analog computer programmed to give spatial magnitude, M, and two angles, V° and H°, defining the direction of M. The coordinate system and angles conformed with the recommendations of the American Heart Association Committee on Electrocardiography (11). Thus +x, +y, and +z voltages correspond with vectors to the left, downwards, and posterior. $V^°$ is the angle of $M$ with the horizontal plane, positive values downwards (caudally), range $= \pm 90^°$. $H^°$ is the angle between the projection of $M$ on the horizontal plane and the +x axis, positive values counterclockwise, range $= \pm 180^°$. In some experiments, thorax resistivity was measured by the method proposed by Schmitt (12). The resistance-box potentiometers were set to maximum, so that each electrode was connected to a 500-kohm resistor. Current was introduced between head and legs, and the potential difference between pairs of electrode rows was measured (10).

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**Figure 1**

Changes in dipole moment (M) during hemoconcentration (expt. 2, Table 2). As hematocrit is increased from 39% to 84% and resistivity from 143 ohm-cm to 671 ohm-cm, the first peak ($M_x$) decreases, and the second peak ($M_z$) increases.
The experiments were divided into three series. In the first series of seven experiments, observations were made on the effect of withdrawal and immediate reinfusion of the dog's own blood. In addition, studies were done on the effect of spontaneous vs. mechanical breathing of the dog and on the effect of altering pH. In the second series of three experiments, whole blood was withdrawn. After hemoconcentration, using additional cells from other animals, equal amounts of blood were then infused. In the third series of five experiments, hematocrit was reduced by rapid withdrawal of varying amounts of blood from the left ventricle and by infusion of equal amounts of plasma or 10% Rheomacrodex (Pharmacia, Upsala) in saline, molecular weight 40,000.

The level of hematocrit that was thus established depended on the general state of the dog and on the experiment duration. After each manipulation, the dog was allowed to equilibrate for 30 minutes, and then a new set of measurements was made. In two dogs, sudden withdrawal of left ventricular blood volume was attempted in an effort to study the effect of changes in intracardiac blood volume at a constant hematocrit. After each manipulation, three groups of observations were made. First, electrocardiographic data were recorded for 30 seconds. Then pH, hematocrit, and blood resistivity were determined on a 3-ml sample of left ventricular blood, and finally a determination of cardiac output was made. Blood resistivity was measured by a conductivity bridge. Depending on hemodynamic state and the dog's size, between 4 and 10 sets of measurements were made.

FIGURE 2  
Effect of hemoconcentration on angle H° (expt. 2, Table 2). As hematocrit and resistivity increase the angle H° turns posteriorly earlier in the QRS wave.

Results

Normal Values.—Fifteen of the 17 dogs had two peaks in their spatial magnitude curve. Of these, the first peak was larger in 11 dogs, and the second peak was larger in 4 dogs. Two dogs had three peaks in their M curve. The peaks were designated M₁, M₂, and M₃, depending on their time of occurrence. Values of V° and H° at the times of these peaks were given corresponding subscripts. Table 1 gives the means, ranges, and s's for normal values in all dogs.

Hemoconcentration Experiments.—Results for three experiments in which packed cells...
were exchanged for normal blood are shown in Table 2. Hematocrits were raised from normal values of 39–46% to 67–80%. Blood resistivity increased to as high as 671 ohm-cm. The table shows that these increases in hematocrit were accompanied by decreases in $M_1$ and $M_2$ and by increases in $M_s$.

In the experiment shown in Figure 1, the variations in $M$ were plotted at 12, 20, and 35 minutes after the fifth exchange (vectors 6–8 in Table 2) to see if a time-dependent change occurred. The three curves were essentially identical.

Variations in $H°$ during the QRS wave for different hematocrits are shown in Figure 2 (same experiment as shown in Fig. 1). Increases in hematocrit or resistivity had a much smaller effect on $V°$. A time record of an entire experiment (expt. 3, Table 2) including the measurements of stroke volume and end-diastolic volume is shown in Figure 3.

Hemodilution Experiments.—Table 3 shows results for four experiments in which plasma or Rheomacrodex was exchanged for whole blood. Hematocrit and resistivity ($p$) were brought down to very low values, i.e., 6–16% for hematocrit and 67–81 ohm-cm for resistivity. In all cases, $M_1$, or $M_2$, or both, increased. A typical experiment is illustrated in Figure 4. Hematocrit was reduced from 33% to 14% and resistivity from 127 ohm-cm to 78 ohm-cm. It is evident that the peaks of $M_1$ and $M_2$

### Table 3

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<tr>
<th>Expt.</th>
<th>Duration (min)</th>
<th>VCG no.</th>
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<th>$p$ (ohm-cm)</th>
<th>$M_1$ (k nut-cm)</th>
<th>$M_2$ (k nut-cm)</th>
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<th>Heart rate (beats/min)</th>
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VCG = vectorcardiogram. Other abbreviations are the same as in Table 1.

* $P^*$ = left ventricular systolic pressure for expt. 3; $P = $mean arterial blood pressure for other experiments.

† This vector was recorded after blood was removed but before plasma was infused.

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increased, but $M_3$ fell. In the one instance where end-diastolic volume was measured, a gradual increase from 48 ml to 50 ml was observed as hematocrit was lowered from 33% to 14%.

No significant changes were seen in the vertical angles, although lowering the hematocrit sometimes caused small decreases in the values of $V_1^\circ$ and $V_2^\circ$. Changes in $H_1^\circ$ were minimal. Reducing hematocrit caused more negative (anterior) values of $H_2^\circ$ by about 20°.

The results of both hemoconcentration and hemodilution experiments are summarized in Figure 5. As $\rho_1/\rho_{tc}$ increased in value, $M_1/M_{tc}$ decreased. The correlation coefficient was $-0.93$ when $\rho_1/\rho_{tc} < 1$ and $-0.98$ when $\rho_1/\rho_{tc} > 1$ (Fig. 5A).

In Figure 5B, $M_2/M_{tc}$ shows a similar decrease with increasing resistivity ratio. Coefficients of correlation for the data of Figure 5B and C were calculated only up to $\rho_2/\rho_{tc} = 2.90$, since beyond this point the relationship was nonlinear. For $\rho_2/\rho_{tc} < 1$ $r = -0.48$, and for $\rho_2/\rho_{tc} > 1$ $r = -0.94$. For $\rho_3/\rho_{tc} < 1$ $r = 0.17$, but for $\rho_3/\rho_{tc} > 1$ $r = 0.88$. In general, there was a very good

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**FIGURE 4**

Changes in dipole moment ($M$) during hemodilution (expt. 3, Table 3). As resistivity was decreased from a control value of 127 ohm-cm to values of 87 ohm-cm and 78 ohm-cm by exchange transfusions of plasma, $M_1$ and $M_2$ increased and $M_3$ decreased. $M_1$ is observed as a definite peak in the control curve only.

**FIGURE 5**

Data points for all hemoconcentration and hemodilution experiments. Top: $\rho_1$ is the blood resistivity after transfusions and $\rho_{tc}$ is the resistivity of control intracardiac blood for a given experiment. $M_1/M_{tc}$ is the dipole moment of the first peak of $M$ divided by the corresponding control value of dipole moment. The straight lines and equations are the best least-square fits to the data. Middle: Same as top except that the subscript 2 denotes observations made and measurements taken at the time of the second peak in the spatial magnitude curve. Bottom: Same as top except that the subscript 3 denotes observations made at the time of the third peak in the spatial magnitude curve. Note the difference in scaling in the three graphs.
correlation between $M/M_e$ and $p/p_e$. The regression equations are given in the illustrations.

Control Experiments.—There were no significant changes in the experiment in which vectorcardiograms (VCG) were simply repeated at intervals. In five measurements over a period of 2 hours, the ranges of $M_1$ and $M_2$ as percents of the means were 10.6% and 17.7%, respectively.

Of the three experiments with whole blood exchanges, the best result was one in which homologous blood was used. In five exchanges, hematocrit was 36–37% and the range of blood resistivity was 113 ohm-cm to 123 ohm-cm. Ranges of $M_2$ and $M_8$ were 8.9% and 19.3% of the mean values, respectively.

In one of the early experiments, there appeared to be a change in dipole moment magnitude when the dog was hyperventilated. Consequently, three experiments were done in which the dog was deliberately hyperventilated. In one of these, the respiration rate was varied from 2400 ml/min to 875 ml/min. This produced a change in blood pH from 7.71 to 7.41 and a rise in $P_{co_2}$ from 16 to 34. $M_1$ and $M_8$ both increased during this period. There was also an increase in mean arterial blood pressure from 114 mm Hg to 138 mm Hg. The reverse could also be shown. Changes in $M_1$ and $M_8$ therefore followed changes in $P_{co_2}$ and mean arterial blood pressure.

$P$ and $T$ Waves.—In 13 normal dogs, the mean P-wave duration of 68 msec (range 44 to 90 msec) agreed closely with a published value of 70 msec (13). All dogs had an $M_1$ peak, and three also had an $M_2$ peak. Mean values and ranges were: $M_1$ magnitude = 98 k ma-cm (conductivity milliamperes-centimeters) (65 to 179 k ma-cm), $V_1^0 = 48^\circ$ (22 to 71$^\circ$), $H_1^0 = -7^\circ$ (−55 to 19$^\circ$), $M_1$ time =49% duration of the P wave (33 to 64%), $M_2$ magnitude = 55 k ma-cm (19 to 78 k ma-cm), $V_2^0 = 17^\circ$ (−34 to 49$^\circ$), $H_2^0 = -50^\circ$ (−137 to 4$^\circ$), $M_2$ time = 68% duration (56 to 83%).

A plot of $M/M_e$ vs. $p/p_e$ for the P waves is similar to that shown in Figure 5C. For $p/p_e > 1$, the correlation coefficient was $r = 0.81$ and the regression equation was $M/M_e = 0.86 + 0.14p/p_e$. When $p/p_e < 1$, $r = 0.66$ and $M/M_e = 0.47 + 0.53p/p_e$.

The angles were not greatly changed by the altered hematocrit. In the control experiments, there was no correlation between P-wave amplitude and pH. The amplitude was lower in some records for which mean arterial blood pressure was low. For the vectors recorded after blood was removed but before fluid was added, P-wave amplitudes were increased.

For the T waves, six animals had an $M_1$ peak only, one had an $M_2$ peak only, and six had both $M_1$ and $M_2$ peaks. Mean values and ranges were: $M_1$ magnitude = 111 k ma-cm (58 to 165 k ma-cm) $V_1^0 = 19^\circ$ (−31 to 61$^\circ$), $H_1^0 = -78^\circ$ (−130 to −15$^\circ$), $M_1$ time = 53% duration (40 to 69%), $M_2$ magnitude = 131 k ma-cm (29 to 339 k ma-cm), $V_2^0 = 19^\circ$ (−19 to 37$^\circ$), $H_2^0 = -80^\circ$ (−29 to −112$^\circ$), $M_2$ time = 78% duration (73 to 84%). The duration was 160 msec (113 to 200 msec). For $p/p_e > 1$, all values of $M/M_e$ were greater than 1.0, although there was a fairly wide scatter of points. The regression equation was $M/M_e = 0.25 + 0.69p/p_e$, $r = 0.64$. For $p/p_e < 1$, $r = 0.15$.

Thorax Resistivity Measurements.—The mean thorax resistivity for five dogs was 369 ohm-cm, with a range of 345 ohm-cm to 392 ohm-cm. Thorax resistivity was measured during one of the dilution experiments, in which blood resistivity was decreased from 176 ohm-cm to 79 ohm-cm. The thorax resistivity first dropped from 384 ohm-cm to 372 ohm-cm but then increased to a final value of 406 ohm-cm. There appeared to be no relationship between blood resistivity and thorax resistivity measured in this manner.

Discussion

Of various hemodynamic factors which might affect the QRS complex, the end-diastolic blood volume may be the most important, since it constitutes a low resistance mass (normally 70 ± 20 ml/m$^2$ body surface area of resistivity 140 ohm-cm in humans) which occupies the ventricular cavity at the time ventricular activation takes place. Furthermore, since the "Brody effect" (4) postulates that the influence of this blood mass will
be to enhance radial dipoles as its resistivity is lowered or its quantity increased, marked alterations in early peaks in the dipole moment could be erroneously ascribed to changes in left ventricular muscle weight (normally 96 ± 16 g/m² body surface area of resistivity 480 ohm-cm). There is little experimental proof of this hypothesis, however. Although Angelakos and Gokhan (14) found reversible voltage changes in venous inflow, specific measurements of dipole moment during alterations in intracavitary blood mass have not been made.

In general, the changes in $M_1$ and $M_2$ observed in this study were in accord with the concept that excitation occurs mainly in a radial direction during the first half of total QRS excitation time, whereas changes in $M_3$ correspond to a predominantly tangential spread during the second half of QRS excitation time. For a further analysis of this difference in cardiac excitation as depolarization progresses, the original studies of Scher and Young were examined (6, 15). Their studies showed an average QRS duration of 35 msec, whereas the present study shows a mean QRS duration of 46 msec. The difference may be explained by the high gain and fast recording speeds which permitted higher resolution and accuracy than Scher and Young could obtain when they timed the QRS duration with lead II at normal amplification. This conclusion is further justified by their statement that some very early and late activation could not be recorded. To make both studies comparable, a total duration of 37.5 msec for the data of Scher and Young was assumed, so that 5 msec on their graphs became 7.5 msec or 20% of total duration of QRS. Since the data in the present study are all expressed as a percent of total QRS time, the results of both studies can be compared.

In the present study, the mean $M_3$ vector occurred at 29% and pointed anteriorly, slightly to left of center, and somewhat downward ($V_3^o = 22°$, $H_3^o = -80°$). At this time, the data of Scher and Young showed that active tissue surrounded both ventricular cavities, although endocardial breakthrough occurred on the right. Activity around each cavity joined in the septum. It is likely, therefore, that the peak of $M_3$ occurred at a time when inside-outside excitation in both walls was taking place and that excitation was mainly radial. The slightly posteriorly directed excitation in the septum is offset by vectors pointing forward in the anterior section of the heart.

For $M_2$, the mean vector occurred at 42% of QRS and pointed to the left, anterior, and downwards ($V_2^o = 34°$, $H_2^o = -40°$). This time corresponded to the 15-msec period of the data of Scher and Young when there was further spread towards the epicardium and more endocardial breakthrough on the right. The diagrams show wave fronts spreading outwards on the anterior, and also around the left side, and extending to right posterior sections of the heart. These wave fronts are now farther away from the cavities and also include some tangential spread.

The mean $M_4$ vector, at 64% duration in this study, pointed to the left, posterior, and slightly downwards ($V_4^o = 10°$, $H_4^o = 69°$). The excitation data showed that the left posterior sections of the heart were activated after 60% of the QRS wave had been inscribed. There was also late septal activation in the basal region. These wave fronts now are near the epicardium and relatively far from the cavities so that the intracardiac blood should have less effect. Activity during the last few milliseconds of ventricular activity is predominantly in the upper septum, which is excited by a wave proceeding from apex to base (15). It is clear that during and after the $M_4$ peak, there was a major change from radial to tangential spread.

The results of the present study were also compared with the surface potential data of Boineau et al. (16), with 37.5 msec taken as 100% duration of the QRS wave for their data. At their 10-msec time corresponding to our $M_4$, a complex multipolar potential distribution was found. There were three potential maximums, one slightly to the right of the ventricular midline and slightly above center, a second at the same vertical level and to the
left, and a third on the left side but considerably lower down. The negative peaks are high and posterior. Values of $V_r^\circ = 22^\circ$ and $H_r^\circ = -80^\circ$ for $M_1$ are reasonable for a resultant for this distribution.

$M_2$ at 42% with $V_2^\circ = 34^\circ$ and $H_2^\circ = -40^\circ$ would correspond to a time between Figures 2B-3 and 2B-4 of Boineau et al. There were now two positive maximums to the left of the midline, one slightly above center and the other fairly low. At the time of $M_3$, at 64% of the QRS wave, with $V_3^\circ = 10^\circ$ and $H_3^\circ = 69^\circ$, there was a larger negative peak to the left of the midline and a smaller one higher up. Positive maximums were located at the lower midline and higher up to the left of the negative peaks. Again, the directions of $M_1$ and $M_3$ are in reasonable agreement with the surface potential data. Since the lead system used had 24 electrodes around the thorax, a good sampling of the potential field was obtained.

Durrer and co-workers (7) stated that the human activation sequence is similar to that in the dog heart. Their diagrams also show radial spread during early portions of the QRS wave and tangential spread later. The conclusions drawn from this study may, therefore, be applicable to the human being.

In fact, Rosenthal and co-workers (17) found changes in spatial vectors when hematocrit was reduced during plasmapheresis in patients with polycythemia. These authors noted an earlier occurrence of the left maximum spatial vector, in other words, an augmentation of early QRS vectors as the hematocrit was brought down. They further indicated that there was relatively little change in end-diastolic volume, a fact also observed in this study (Fig. 3) in the one dog where this was measured.

The results after acute alterations in hematocrit indicate that changes in resistivity were the principal causes for the marked alterations in dipole moment (Fig. 5). With $\rho/\rho_e = 2.5$, the blood resistance was about equal to that of the myocardium. Dipole moments measured under these conditions should be more indicative of the true excitation forces of cardiac muscle than those measured when normal blood is present inside the heart. Comparing these $M$ values with those for $\rho/\rho_e = 1$, the conclusion can be drawn that the effect of normal intracardiac blood is to increase the apparent values of $M_1$ by a factor of 3 and of $M_2$ by 2.5, but to decrease $M_3$ by about 25%. In other words, to correct for the presence of intracardiac blood in the normal intact dog, $M_1$ should be divided by 3 and $M_2$ by 2.5, but $M_3$ should be multiplied by 1.25. This gives a completely different picture of the real dipole strength during ventricular depolarization, as evident in figure 1.

The observed changes in P vectors were consistent with those seen in QRS activation. Since atrial depolarization is largely tangential, increases in hematocrit should lead to an increase in magnitude. This was indeed observed ($r = 0.81$) when $\rho/\rho_e > 1$. The effects of altered hematocrit on the T wave were inconclusive, although there was a general increase in T-wave magnitude as resistivity increased.

An unknown factor was the effect of altered hematocrit on overall body resistivity. This would depend on the distribution of blood in the various organs. Although the value of blood in the lungs is fairly high, about 10% (18), the proximity of the intracardiac blood to the myocardium would appear to have the dominant effect. The lung normally has a much higher resistivity than does the heart, and an increased hematocrit would tend to keep this relationship constant. In the present study, there was little relationship between hematocrit and overall thorax resistivity and little change in resistivity with respiration. It is possible that a large percent of the current flows from head to legs through the low-resistance surface layer (19).

In conclusion, the results indicate that the magnitude of body surface potentials are profoundly influenced by the presence of intracardiac blood. Voltages due to radial excitation during early portions of the QRS wave are enhanced, but later occurring, predominantly tangential, forces are attenuated. It appears that corrections should be made...
for the effects of intracardiac blood if more accurate relationship between the measured dipole moment and the actual strength of heart excitation forces are desired. Alterations in blood resistivity may explain, in part, the electrocardiographic changes observed by others during anemia and polycythemia.

References
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