Influence of Acute Variations in Hematocrit on the QRS Complex of the Frank Electrocardiogram

By Amnon Rosenthal, M.D., Norma J. Restiaux, M.D., and Stephen A. Feig, M.D.

SUMMARY
The influence of acute variations in hematocrit on the QRS complex of the Frank scalar electrocardiogram was studied in 31 patients with severe polycythemia secondary to cyanotic congenital heart disease and in eight patients with anemia due to thalassemia. Moderate reduction of the hematocrit in the polycythemic group resulted in a significant increase in the magnitude and a delay in both left maximal spatial voltage (LMSV) and maximal anterior force. Raising the hematocrit in the anemic group decreased the magnitude of LMSV and maximal anterior force. A change in the orientation of the spatial vector frequently accompanied induced variations in the hematocrit. Experimental and theoretical studies suggest that alterations in the hematocrit and, hence, intracardiac electrical resistivity distort the magnitude and orientation of the surface-recorded QRS voltages. It is postulated that the effect of high intracavitary blood hematocrit in polycythemia in reducing early QRS voltages is due to its influence on radial myocardial excitation propagated from endocardium to epicardium. Anemia, on the other hand, enhances the voltages due to radial spread of impulses in the myocardium, resulting in an increased magnitude of the LMSV and usually also of the maximal anterior force.

Additional Indexing Words: Anemia Exchange transfusion Vectorcardiogram

Heart defect, congenital Phlebotomy

Theoretical and experimental work suggest that the intracavitary blood mass exerts a definite influence on the magnitude of the electrical vector of the heart. Blood is a good conductor of approximately 160 ohm-cm resistivity at body temperature, while myocardial resistivity is nearly triple that of normal blood, and surrounding lung tissue resistivity is nearly 10 times as great. This body heterogeneity may exert a strong distorting effect on the surface-recorded heart vector, since it tends to produce augmentation of voltages produced by radially oriented forces and suppression of tangential components ("Brody effect").

An increase in the hematocrit, however, results in increased blood resistivity (approximately 350 ohm-cm at a hematocrit of 70%) and a more homogeneous electrical situation. The presence of polycythemia, or anemia, may therefore be expected to alter significantly the direction and magnitude of the heart electromotive forces measured at the body surface.
Clinically, the magnitude and orientation of the QRS spatial vector have been very useful in the assessment of ventricular hypertrophy and estimation of peak ventricular systolic pressure in both acyanotic (aortic stenosis\textsuperscript{7,8} and pulmonary stenosis\textsuperscript{7}) and cyanotic (transposition of the great arteries\textsuperscript{9}) patients with congenital heart disease (CHD). The purpose of the present study, therefore, was to determine what effect variations of the hematocrit in polycythemic and anemic patients may have on the QRS spatial vector as it is recorded on the body surface. While a number of papers have discussed the theoretical implications\textsuperscript{1-10} of, and presented animal experimental data\textsuperscript{9,11,12} on, the relationship between hematocrit and surface-measured electromotive forces, little effort has been directed toward testing these ideas in clinical situations.\textsuperscript{13}

### Methods

Thirty-one patients with polycythemia secondary to cyanotic CHD and eight patients with anemia due to thalassemia major (Cooley's anemia) were studied. Table 1 lists the pertinent clinical data in the patients with CHD and table 2 the clinical data in the patients with anemia.

A Frank orthogonal scalar electrocardiogram in the CHD patients was recorded prior to and 2–24 hr after the replacement of whole blood with an equal amount of fresh-frozen plasma (erythropheresis\textsuperscript{*}), the technique of which has been previously described.\textsuperscript{14} Erythropheresis was completed in 30–60 min. In the anemic patients the electrocardiogram was recorded prior to and after a slow (6–8 hr) transfusion of packed red cells. The electrodes of the Frank lead system were applied in the usual fashion with patients in the recumbent position. Twenty-two of the 31 CHD patients and one of the eight anemic patients were studied in the cardiac catheterization laboratory and the rest in the outpatient department. In the catheterized patients the position of the thoracic electrodes was checked by fluoroscopy. In 23 patients the leads remained attached to the body during the pheresis and were removed only after the postpheresis recording was obtained. Skin electrode resistance, measured in six of these patients, did not change materially between the first and second electrocardiographic recordings. In seven patients with anemia and eight with polycythemia the lead positions were carefully marked; the leads were reapplied, and a scalar electrocardiogram was obtained after the procedure. Venous hematocrit was determined within one-half hour of each recording.

Whole blood volume measurements were performed prior to, and following completion of, the erythropheresis in nine patients and before and after transfusion of packed cells in one patient. Red cell volume was measured with $^{51}$Cr-labeled autologous red cells, and plasma volume with $^{129}$I-labeled albumin. Biplane angiograms for the measurement of left ventricular end-diastolic volume were obtained before and after red cell pheresis in three patients and before and after transfusion in one patient. Details of the technique and method used in the measurements of the end-diastolic volume in this laboratory have been reported previously.\textsuperscript{15}

Electrocardiographic measurements were confined to the QRS complex. The parameters studied included magnitude and orientation of (1) the left maximal spatial vector (LMSV), defined as the maximal spatial voltage to the left, i.e., with azimuth and elevation within the range of 270° to 90°, (2) the right maximal spatial vector (RMSV), defined as the maximal spatial voltage to the right, i.e., with azimuth and elevation within the range of 90° to 270°, (3) the 10-msec vector, and (4) the maximal anterior voltage in lead Z. Scalar display was used in all cases, and a vector loop (using Sanborn model 1507) was also obtained in 12 patients. In subjects studied in the outpatient department simultaneous X, Y, and Z lead input was first photographed at a sweep speed of 25 mm/sec and a square wave standardization signal introduced. The QRS complex was then photographed at a fast sweep (250 or 500 mm/sec) to expand the time scale. The spatial voltage ($\sqrt{x^2 + y^2 + z^2}$), elevation of the spatial vector as projected on the frontal plane ($\tan^{-1}\frac{y}{x}$), herein referred to as elevation, and azimuth ($\tan^{-1}\frac{z}{x}$) were computed at 2.5-msec intervals throughout the QRS complex, using an x-y digitization table connected to a PDP-9 computer.\textsuperscript{†} In the subjects studied in the catheterization laboratory an initial recording was obtained on magnetic tape at 30 inches/sec. With the tape running at slow speed (3X inches/sec) the tracing was then recorded on.

\textsuperscript{*}Strictly speaking, the procedure involves the removal of the other formed elements besides erythrocytes.


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### Table 1

<table>
<thead>
<tr>
<th>No.</th>
<th>Diagnosis</th>
<th>Age/Sex</th>
<th>Hematocrit (%)</th>
<th>Max Z Anti (mv)</th>
<th>LMSV (mv)</th>
<th>RMSV (mv)</th>
<th>Time (ms)</th>
<th>Max Z Anti (mv)</th>
<th>LMSV (mv)</th>
<th>RMSV (mv)</th>
<th>Time (ms)</th>
<th>Max Z Anti (mv)</th>
<th>LMSV (mv)</th>
<th>RMSV (mv)</th>
<th>Time (ms)</th>
<th>Max Z Anti (mv)</th>
<th>LMSV (mv)</th>
<th>RMSV (mv)</th>
<th>Time (ms)</th>
<th>Max Z Anti (mv)</th>
<th>LMSV (mv)</th>
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<th>Time (ms)</th>
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<td>M/F</td>
<td>75</td>
<td>61</td>
<td>0.82</td>
<td>1.23</td>
<td>12.5</td>
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<td>15.0</td>
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<td>0.55</td>
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<td>TOF</td>
<td>M/F</td>
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<td>61</td>
<td>0.82</td>
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<td>1.90</td>
<td>15.0</td>
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<tr>
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<td>TOF</td>
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<td>0.82</td>
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<td>15.0</td>
<td>0.55</td>
<td>1.90</td>
<td>15.0</td>
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<td>1.90</td>
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<td>15.0</td>
<td>0.55</td>
<td>1.90</td>
<td>15.0</td>
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</tbody>
</table>

**Notes:**
- No leftward electromotive force (see text).
- No anterior electromotive force (see text).
- Abbreviations: LMSV = left maximal spatial vector; mv = null; TOF = atrial septal defect; PS = pulmonary stenosis; PAH = pulmonary hypertension.

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Changes in the magnitude of the left maximal spatial vector (LMSV) associated with (A) erythropoiesis and (B) transfusion of packed erythrocytes in eight patients with Cooley's anemia. **Figure 1.**

Table 2

<table>
<thead>
<tr>
<th>No.</th>
<th>Diagnosis</th>
<th>Age/Sex</th>
<th>Hematocrit (%)</th>
<th>LMSV (mv)</th>
<th>Time (msec)</th>
<th>Max Z ant (mv)</th>
<th>Time (msec)</th>
<th>RMSV (mv)</th>
</tr>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
<td>After</td>
<td>Before</td>
<td>After</td>
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<tr>
<td>32</td>
<td>CA</td>
<td>4/F</td>
<td>19</td>
<td>29</td>
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<td>CA</td>
<td>9/M</td>
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<tr>
<td>34</td>
<td>CA</td>
<td>12/F</td>
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<td>29</td>
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<td>14/F</td>
<td>23</td>
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<td>CA</td>
<td>16/F</td>
<td>30</td>
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<td>2.84</td>
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<td>CA</td>
<td>17/F</td>
<td>24</td>
<td>32</td>
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<td>18/F</td>
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<td>2.39</td>
<td>1.74</td>
<td>42.5</td>
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<tr>
<td>39</td>
<td>CA</td>
<td>19/F</td>
<td>27</td>
<td>43</td>
<td>1.91</td>
<td>1.83</td>
<td>35.0</td>
<td>35.0</td>
</tr>
</tbody>
</table>

Mean 23 34 2.25 1.94 36.9 37.8 0.69 0.62 22.5 20.3 0.65 0.67

Standard deviation 5 7 0.48 0.45 5 5.1 0.21 0.21 6.8 8.1 0.38 0.40

P value by two-sided sign test 0.008 1.0 0.29 0.13 0.73

Abbreviations: LMSV = left maximal spatial vector; mv = millivolt; msec = millisecond; max Z ant = maximal Z anterior; RMSV = right maximal spatial vector; CA = Cooley's anemia.

**Results.**

In the polythemic patients phlebotomy and replacement of blood with fresh-frozen plasma reduced the mean venous hematocrit from 73% (range, 64-88%) to 63% (range, 53-75%) (table 1). The acute reduction in hematocrit was associated with an increase in the magnitude of the LMSV in 26 out of 30 patients (P < 0.001). The mean LMSV in 26 out of 30 patients was 1.46 mv (fig. 1A). In 18 out of 30 patients with Cooley's anemia there was a delay (2.5 msec or more) in the occurrence of the LMSV in the course of the decrease in hematocrit (table 1). In 18 out of 30 patients with Cooley's anemia and in seven patients it remained unchanged, and in

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Changes in the magnitude of the left maximal spatial vector (LMSV) associated with (A) erythropoiesis and (B) transfusion of packed erythrocytes in eight patients with Cooley's anemia. **Figure 1.**

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Calculation of the spatial volatges was then carried out as outlined above. Statistical analyses were performed with the use of the two-sided sign test.
The ratio of LMSV to RMSV increased in 24 patients, remained unchanged in one, and decreased in five ($P = 0.005$).

In the patients with Cooley's anemia mean hematocrit increased from 23% (range, 14–30%) to 34% (range, 28–44%) during slow transfusion of packed red cells. The procedure was followed by a decrease in the magnitude of the LMSV in all eight patients ($P = 0.008$; five it occurred earlier ($P = 0.01$) (table 1). There was a notable change (>15°) in the elevation in six patients and in the azimuth in 13 patients. The change in both the azimuth and elevation of the LMSV, however, was not consistently in a given direction. In a few patients the maximal spatial vector (the spatial vector of greatest magnitude) which was directed to the right prior to pheresis occurred earlier as the LMSV increased, resulting in a shift towards the left after pheresis (figs. 2 and 3).

In addition to the change in LMSV there was a consistent change in the magnitude of the peak anterior force in lead Z following pheresis (fig. 4A). After erythropheresis the maximal magnitude of the anterior force in lead Z increased in 28 out of 29 patients ($P << 0.001$). The mean of the maximal Z anterior force for the entire group increased from 0.36 to 0.57 mv (table 1). In 21 of the patients there was a delay (2.5 msec or more) in the occurrence of the peak anterior force when compared to the time of the peak prior to pheresis; in seven it remained unchanged, and in one it appeared earlier ($P << 0.001$). Acute reduction of the hematocrit was also accompanied by variable changes in the magnitude, direction, and time of occurrence of the RMSV, the 10-msec spatial vector, as well as the ratio of the 10-msec to maximal spatial vector. The change in the orientation of the maximal QRS spatial vector in patient 3. A definite change in the direction of the maximal spatial vector from right to left was observed following erythropheresis. (See also fig. 2.)
Changes in magnitude of the maximal Z anterior force associated with (A) erythropheresis in 29 patients with hypoxic polycythemia and (B) transfusion of packed erythrocytes in eight patients with Cooley’s anemia. A decrease in hematocrit (A) was accompanied by an increase in the anterior force while a rise in hematocrit (B) had the opposite effect. The symbol ○ denotes the mean value before and after the procedure.

Table 3

Red Cell and Whole Blood Volume before and after Erythropheresis and Red Cell Transfusion

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Procedure</th>
<th>Red cell volume (ml/kg)</th>
<th>Whole blood volume (ml/kg)</th>
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<td></td>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>2</td>
<td>Ep</td>
<td>69.9</td>
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<td>Ep</td>
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<td>Ep</td>
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<td>14</td>
<td>Ep</td>
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<td>89.9</td>
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<td>16</td>
<td>Ep</td>
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<td>Ep</td>
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<td>20</td>
<td>Ep</td>
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<tr>
<td>34</td>
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<td>12.7</td>
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<tr>
<td>Standard deviation*</td>
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<td>16.0</td>
<td>13.8</td>
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</table>

*Excludes patient 34.

Abbreviations: Ep = erythropheresis; trans = transfusion of packed erythrocytes.
Table 4

Left Ventricular End-Diastolic Volume before and after Erythropheresis and Transfusion

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Procedure</th>
<th>Hematocrit (%)</th>
<th>LVEDV (ml/m²) Before</th>
<th>LVEDV (ml/m²) After</th>
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<td>Ep</td>
<td>83</td>
<td>77</td>
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<td>34</td>
<td>Trans</td>
<td>23</td>
<td>29</td>
<td>81.3</td>
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Abbreviations: LVEDV = left ventricular end-diastolic volume; Ep = erythropheresis; trans = transfusion of packed erythrocytes.

left ventricular end-diastolic volume prior to and following erythropheresis could be obtained on only a few of the patients. Repeated angiograms for the purpose of this study were felt to represent an unacceptable risk in the majority of patients studied. The end-diastolic volumes obtained are outlined in table 4. There was no significant change in heart rate (<9 beats/min) following pheresis or red cell infusion. Although the sample size is small the changes in left ventricular end-diastolic volume did not appear to be large.

Discussion

An acute reduction of the hematocrit in patients with hypoxic polycythemia secondary to cyanotic CHD resulted in an increase in the magnitude of the left maximal spatial vector and an increase in the maximal anterior force as reflected in the Z lead of the Frank scalar electrocardiogram (figs. 5 and 6). Consonant with the increase in the surface-measured electromotive forces which occurred following erythropheresis, transfusion with packed cells in patients with Cooley's anemia was accompanied by a decrease in the left maximal spatial vector and usually also in the maximal Z anterior force (fig. 7). In addition to changes in the magnitude of these forces, there was a significant delay in the time of occurrence of both the LMSV and maximal anterior force in the patients undergoing erythropheresis. Transfusion and erythropoiesis were frequently followed by a notable change in the orientation of the maximal spatial vector (fig. 3). No consistent relationship was observed between the changes in hematocrit and the magnitude or direction of the right maximal spatial vector in either group.

The observed changes in the leftward spatial voltages and anterior forces may be

![Figure 5](http://circ.ahajournals.org/)

*Figure 5*  
*(Left and Right) The effect of erythropheresis on the QRS complex of the Frank scalar electrocardiogram and vectorcardiogram in patient 6. A decrease in hematocrit resulted in an increase of the left maximal spatial vector (LMSV) and the maximal Z anterior force.*

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ACUTE VARIATIONS IN HEMATOCRIT

ACUTE VARIATIONS IN HEMATOCRIT

The effect of erythropheresis on the QRS complex of the Frank scalar electrocardiogram in patient 24. An increase in both the left maximal spatial vector (LMSV) and maximal Z anterior force (arrows) was observed following a reduction in the hematocrit.

due to induced variations in intracardiac or extracardiac resistivity. The electrical conductivity of blood varies inversely with its packed red cell volume and serum protein concentration and directly with total base. In our experience, erythropheresis does not alter serum electrolyte concentration or lower protein concentration; but the reduction in hematocrit is almost certainly accompanied by an increase in the conductivity of the blood. The decreased resistivity of the intracardiac blood mass may be expected to result in augmentation of voltages due to radially directed cardiac electromotive forces and suppression of those due to tangential components. Acute reduction of the hematocrit in dogs by erythropheresis has been shown to result in a marked decrease in blood resistivity. Furthermore, acute experiments in both dogs and monkeys have demonstrated that lowering the hematocrit increases the first or two peaks and reduces the second or third peak of the spatial magnitude voltage. Raising the hematocrit had the opposite effect on these peaks. Early depolarization peaks are associated with septal and free wall excitation. Insofar as these wave fronts are radial, the lower electrical resistance of blood produced by erythropheresis should cause augmentation of those voltages as recorded on the body surface and distortion of the heart vector.

The maximal Z anterior voltage and LMSV represent relatively early peaks in the ventricular excitation sequence. The observed in-
crease in magnitude of these parameters, following a reduction in hematocrit, is therefore consistent with previous theoretical and experimental studies. The late peak corresponds to the tangential spread of excitation in the basal part of the ventricular wall and septum. Since the depolarization wave front in the right ventricle is propagated mainly tangentially one would expect a diminution in the RMSV as a consequence of a reduction in hematocrit. The absence of a consistent response pattern in RMSV in our patients (tables 1 and 2) may possibly be related to the fact that activity of the right ventricular dipoles are drawn out in time. In contrast to erythropoiesis, transfusion of packed red cells in subjects with Cooley’s anemia can be expected to increase the resistivity of intracardiac blood and result in suppression of voltages due to the radially oriented excitation front and, therefore, a decrease in the maximal Z anterior force and LMSV (table 2). The frequent change in orientation of the spatial vector, induced by acute variations in the hematocrit, may also be related to the changes in intracardiac blood mass conductivity and its effect on the spread of impulses in the myocardium relative to the heart cavity (fig. 3). The reason for the observed delay in the occurrence of the LMSV in the majority of patients with erythropoiesis remains unclear.

An increase in intracardiac volume may augment, or possibly induce, changes in the spatial magnitude curves which are similar to those resulting from alterations in blood resistivity. Total blood volume measured in our patients certainly did not increase and in fact had decreased slightly in all but one patient (table 3) and therefore could not account for the changes in recorded QRS potentials. Erythropoiesis results in decreased blood viscosity and peripheral resistance and increased stroke volume and systemic blood flow. Whether the increased flow and stroke volume are associated with a greater end-diastolic volume, improved myocardial contractility, or both, is unknown. There is some evidence from animal experiments that erythropoiesis is accompanied by an increase in left ventricular end-diastolic volume (Nelson, C. V., Hugenholtz, P. G.: Unpublished data). The few end-diastolic volume measurements obtained in this study do not suggest a consistent increase in intracardiac volume (table 4), and the findings do not permit adequate assessment of the independent influence of intracardiac volume versus packed red cell concentration on the recorded spatial vector. The decrease in resistivity of extracardiac blood produced by a decrease in the hematocrit may also have enhanced the voltages recorded at the surface but it is hard to explain the selective augmentation of the leftward and anterior forces on this basis.

The reported observations may be pertinent in the clinical management and assessment of patients with congenital heart disease as well as those with anemia. The estimation of peak systolic pressure or ventricular muscle weight from the electrocardiogram in patients with aortic or pulmonary stenosis may be influenced by the patient’s hematocrit at the time the tracing is recorded. A low hematocrit in a patient with aortic stenosis may thus result in a high LMSV and, consequently, overestimation of left ventricular systolic pressure or left ventricular muscle mass. In the patients with transposition of the great arteries the vectorcardiogram has been a useful tool in the assessment of left ventricular systolic pressure. When left ventricular pressure is low, the Frank electrocardiogram shows dominant right ventricular hypertrophy and a clockwise loop in the horizontal plane. On the other hand, when left ventricular pressure becomes high the left ventricular forces are more prominent and a counterclockwise loop is recorded.

In children with a high hematocrit, however, the horizontal loop remains clockwise despite a high left ventricular pressure. The effect of a high hematocrit on attenuation of the left ventricular forces in this group is therefore of diagnostic importance. The increased left ventricular forces frequently noted in patients with anemia or leukemia...
may be secondary to low hematocrit, an increased intracardiac volume, or both, rather than to left ventricular hypertrophy or an increased muscle mass. A transfusion of red cells in these patients may be expected to diminish the magnitude of voltages due to radially oriented components of the depolarization wave front and alter the interpretation of the Frank electrocardiograms.

In summary, the demonstration of significant changes in anteriorly directed and in maximal spatial forces secondary to variations in hematocrit adds still another component to the large number of factors influencing the surface electrocardiogram.

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References

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