Cardiac Potentials on Body Surface of Infants with Anomalous Left Coronary Artery (Myocardial Infarction)

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SUMMARY

The body surface potential distribution in two infants with myocardial infarction secondary to anomalous origin of the left coronary from the pulmonary artery demonstrated: (1) the persistence of a minimum in the area overlying the infarct during inscription of the prominent Q wave in V6, and, (2) the presence of a terminal maximum in an adjacent area overlying the infarct. The terminal maximum was considered to represent peri-infarction “block” resulting from persistence of wave fronts in the ischemic muscle surrounding the infarct as suggested by the studies of Durrer and associates. Postoperatively, there was persistence of the initial minima overlying the area of the infarct. Finally, there was disappearance or marked diminution in the terminal maximum overlying the area of the infarct following surgical enhancement of myocardial blood supply.

Additional Indexing Words:
Electrocardiograms Vectorcardiogram Isopotential surface maps
Ventricular excitation Cardiac potentials

The ultimate clinical usefulness of depicting the distribution and time course of cardiac electrical events on the body surface remains to be answered. An initial step must include recording from multiple body areas to define the “total” information content available during varying intervals of cardiac excitation. Several groups\(^1\)\(^-\)\(^3\) recently have demonstrated that isopotential surface mapping provides information not obtainable with conventional electrocardiographic methods and also that the body-surface potential distribution can be characterized as dipolar or nondipolar or both at varying intervals during ventricular excitation.

The purpose of this report is to illustrate the abnormal body-surface distribution of cardiac potentials resulting from myocardial infarction secondary to anomalous origin of the left coronary artery from the pulmonary artery. Based on these results and on information available in the literature, we would like to suggest a possible altered sequence of ventricular excitation in these patients.

Methods

Subject Population

Two infants, aged 7 and 11 months, were admitted to Duke Hospital because of congestive heart failure. Physical examination revealed tachypnea, the absence of murmurs, and a prominent apical first sound. The standard 12-lead

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Electrocardiograms of infants with myocardial infarction secondary to anomalous left coronary artery. The patterns demonstrated in the preoperative tracings in the 11-month-old (A) and in the 7-month-old (B) infant suggest anterolateral myocardial infarction with prominent Q waves in leads I, aVL, V2, and V6. Note the QS deflection in lead V5 in the 11-month-old infant (A).

Electrocardiograms are shown in figure 1 and demonstrate findings suggestive of an anterolateral myocardial infarct which, in an infant, is presumptive evidence for anomalous origin of the left coronary artery from the pulmonary artery. Chest roentgenograms revealed marked cardiac enlargement (fig. 2A). Retrograde aortograms demonstrated that the left coronary artery did not arise from the aorta but arose from the pulmonary artery as indicated by the retrograde filling of this vessel following opacification of the right coronary artery (fig. 2B). Right ventricular pressures were normal. Following injection of contrast medium into the pulmonary artery, the levogram demonstrated a normalized left atrium with marked dilatation of the left ventricular chamber which had poor contractility. Both patients underwent surgery with successful ligation of the left coronary artery at the site of origin on the pulmonary artery. The preoperative and postoperative vectorcardiographic tracings are shown in figures 3 and 4.

Production of Isopotential Maps

Both patients were studied in the supine position after marking a grid on the thorax and the upper part of the abdomen to record from 150 points. The inter-electrode spacing was individually determined for each child and resulted in 2 to 3-cm vertical spacing and 2.5 to 3-cm horizontal spacing over the anterior and lateral surfaces of the chest. The inter-electrode horizontal spacing was 4 to 5 cm over the back. The details of the technique have been previously reported.3 In summary, five tracings were recorded simultaneously from the chest surface using a common left leg reference lead. Each block was recorded in sequence until all points were explored. A

Figure 1

Electrocardiograms of infants with myocardial infarction secondary to anomalous left coronary artery. The patterns demonstrated in the preoperative tracings in the 11-month-old (A) and in the 7-month-old (B) infant suggest anterolateral myocardial infarction with prominent Q waves in leads I, aVL, V2, and V5. Note the QS deflection in lead V5 in the 11-month-old infant (A).
common time reference lead was recorded simultaneously with each block of data. The recording procedure required 45 to 60 minutes. Following the study, each patient was photographed with the grid markings, and the lead placement for vectorcardiographic and electrocardiographic leads was noted.

All data were tape recorded while respiration was monitored with an impedance respirometer. Beats occurring during resting expiration were chosen for analysis and were transcribed by an analog-to-digital converter at a rate of 926 samples per second.

The outputting of instantaneous maps of body surface potential distribution, utilizing data recorded asynchronously with a common time reference, was justified by the assumption that cardiac potentials are projected onto the body surface in a similar manner at the identical phase of resting expiration. The extent to which

Figure 2

Chest roentgenogram (A) and retrograde aortogram (B) in anomalous left coronary artery. These were obtained preoperatively in the 11-month-old infant with myocardial infarction secondary to anomalous left coronary artery. The chest roentgenogram (A) demonstrated marked cardiomegaly. This degree of cardiomegaly also was present in the other infant studied. Retrograde aortogram (B) demonstrated absence of the left coronary artery arising from the aorta, opacification of the prominent right coronary artery, and subsequent filling in a retrograde fashion of the left coronary artery arising from the main pulmonary artery.
Preoperative and postoperative Frank vectorcardiograms of the 11-month-old infant with myocardial infarction secondary to anomalous left coronary artery. The preoperative vectorcardiogram (A) is abnormal with the prominent initial vector to the right with inscription of the horizontal plane QRS loop in a clockwise direction to the right and posteriorly. The postoperative tracings (B) remain abnormal with some reduction in the magnitude of the maximum spatial vector.

Results

Normal Children

The time course of the distribution of cardiac potentials on the body surface of normal children between the ages of 4 and 14 years is shown in figures 5 and 6. The results were similar to those demonstrated by Taccardi1 for normal adults. A review of the normal sequence is shown to provide a reference for comparing the abnormalities to be noted.
in patients with an anomalous left coronary artery.

The normal sequence consisted of the following major events: (1) The initial development consisted of an anteriorly located maximum with a minimum located in the left axilla (fig. 5A). (2) During the following 5 to 10 msec, the minimum migrated to the center of the back while the maximum continued to increase over the sternum (fig. 5B and C). (3) The right upper part of the chest then was gradually invaded by an area of negativity. At 19 to 28 msec from the onset of surface QRS activity, there was the rapid emergence of an area of negativity over the central sternum (this area later became the principal minimum). At this time (fig. 5D), the surface potential distribution was similar to that described by Taccardi with two minima separated by a higher level of potential ("saddle"). (4) As the central sternal minimum increased in value and size, the maximum shifted into the left axillary region (fig. 6E and F). (5) The maximum then migrated to the central back while the minimum covered most to the anterior chest, giving a dipolar potential distribution (fig. 6G). (6) Terminally, the posterior maximum diminished in amplitude while the anterior minimum diminished and fragmented (fig. 6H).
Figure 5

Isopotential surface maps of normal children (ages 4 to 14 years). This figure and figure 6 demonstrate the major events on the body surface of normal children during QRS. The onset of QRS is heralded by the development of an anterior maximum over the sternum with an associated minimum in the left axilla (A). During the continued inscription of the R wave in lead V₁ and the Q wave in lead V₆, the maximum over the sternum increases in magnitude while the minimum shifts posteriorly to the center of the back (B). At a time corresponding approximately to the peak of the R wave in lead V₁ (C), the anterior part of the chest is strongly positive with pseudopod extensions from the central maximum toward the left precordium. At approximately 19 to 28 msec from the onset of QRS, the right upper chest has become invaded by negative potentials which are associated with the rapid emergence of a discrete minimum over the central sternum with another minimum over the right shoulder region (D). Two minima are separated by an area of relatively higher potentials, producing the “saddle” as described by Taccardi in adults. Note also that the maximum has shifted slightly to the left. The central minimum noted in D will later emerge as the principal minimum with disappearance of the second minimum over the right shoulder.

Infants with Anomalous Left Coronary Artery (Anterolateral Myocardial Infarct)

The preoperative isopotential maps for the 11-month-old infant are presented in figures 7 and 8. Although the surface maps showed many small differences in contours and potentials in the two infants, there was marked similarity in the major events, as will be discussed for this patient.

Initially, ventricular activation was manifested by a maximum emerging over the
BODY SURFACE POTENTIAL DISTRIBUTION

Figure 6

Isopotential surface maps of normals (continued.) The central anterior minimum previously described has now enlarged to cover most of the upper anterior chest. This is associated with the persistence of the lower left precordial maximum which begins to move in a leftward direction (E). Subsequently, the maximum migrates rapidly around the left axilla (F) coincident with the intrinsicoid deflections in leads V_1 and V_6. Note that the anterior minimum continues to enlarge. The maximum then reaches a position over the central back (G) while the anterior chest becomes enveloped by negative potentials. Terminally, the posterior maximum and the anterior minimum diminish in magnitude and finally fragment. In the majority of children which we have studied, the terminal pattern is as shown in H with a posterior maximum and anterior minimum. However, in a few older children we have found a terminal distribution associated with a maximum over the back as well as upper sternum anteriorly, as seen in several adults by Taccardi.1

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sternum with a minimum in the left axilla. This initial distribution was similar to that in normal children, although pseudopods extended to the right lower chest from the maximum (fig. 7A). Thereafter the events were abnormal. The minimum persisted on the left lateral chest throughout the first 17 msec (fig. 7B and C). At this time, there was the rapid development of pseudopod projections from the minimum into the central sternal region with the development of a central sternal minimum and left shoulder minimum with an interposed area of more positive potential (fig. 7D). Thereafter, coincident with the intrinsicoid deflection of lead V_1 and rise of R wave in lead V_6, the
Preoperative isopotential surface maps of an infant with anomalous left coronary artery (anterolateral myocardial infarct). The major electrical events to be discussed occurred in both infants, although there was considerable variation with regard to the details of the body-surface potential distribution throughout QRS. The time course and position of events for the 11-month-old infant whose electrocardiogram is shown in figure 1A and the vectorcardiogram in figure 3 are illustrated. The onset of ventricular activation was characterized by a distribution similar to normal with a central anterior maximum and associated minimum in the left axillary region (A). During the following 6 msec (11 ms), the central anterior maximum continued to enlarge and remained in a stable position with persistence of the left axillary minimum (B). At 17 msec from the onset of QRS, the minimum in the left axilla continued to enlarge while there was pseudopod formation toward the central anterior maximum (C). The pseudopod extension from the left axillary minimum rapidly increased with the development of a second discrete minimum over the central lower sternum, incising the area previously occupied by the maximum (D). Model used is a 2-year-old child. (Continued in figure 8.)

previously located central maximum migrated in a rightward direction around the axilla while the centrally located minimum enlarged anteriorly (fig. 8E).

The next interval was characterized by migration of the maximum from the right lower back across the posterior body surface to the left axilla (fig. 8F and G). The remaining 35 msec of ventricular activation were evidenced by a maximum (fig. 8G and H) developing in the left axillary region previously occupied by the minimum during
Isopotential surface maps in anomalous left coronary artery (continued). The previously described central anterior minimum emerged as the principal minimum over the anterior chest (E) while the maximum migrated in a rightward direction to the back. The maximum then migrated rapidly across the back to the left shoulder region (F) with persistence of the anterior central minimum. The remainder of QRS was associated with the maximum shifting into the region of the left axilla (G), which initially had been occupied by the persistent minimum. The final distribution (H) consisted of fragmentation of the anterior minimum with gradual disappearance of the left axillary maximum. Model used is a 2-year-old child.

initial QRS. After the total duration of QRS (98 msec), the left axillary maximum disappeared with negative potentials developing in this area during the S-T segment and T wave.

Isopotential maps were recorded postoperatively to evaluate the effect of surgery on the major surface events. Figure 9 shows four selected instants of time which were chosen to illustrate both the similarities and differences found. The preoperative duration of QRS was measured at 98 msec while postoperatively the duration decreased to 88 msec in the 11-month-old infant shown. The initial events during the first 11 msec of QRS were similar to those of the preoperative state, except that there was a small upward shift of the “zero” (average potential) line over the left hemithorax (fig. 9A). Also, a decrease was noted in the highest potential value reached; that is, the maximum decreased from a preoperative level of 2.2 mv to a postoperative
Postoperative isopotential maps. Isopotential mapping studies following ligation of the left coronary artery at its site of origin from the pulmonary artery were done in both patients. Shown above are four selected instants of time to illustrate both the similarities and differences in the body-surface potential distribution which occurred following enhanced blood supply to the myocardium. Model used is a 2-year-old child.

(A) The initial events were similar to the preoperative state with the development of a maximum anteriorly and a minimum in the left axilla. The highest positive potential value achieved by this increasing maximum was less than half that of its preoperative value.

(B) At 20 msec following the onset of QRS, the maximum remained over the right central chest anteriorly while the minimum persisted in the left axilla with beginning pseudopod formation into the central anterior chest.

(C) At 35 msec the anterior chest remained enveloped by strongly negative potentials with slight pseudopod infiltration over the left axilla from the posterior maximum.

(D) At 78 msec there was absence of positive potentials in the left anterior axillary region which in the preoperative state had been occupied by terminal maximum (compare with figure 8 G and H).

level of 0.96 mv. At 20 msec (fig. 9B), the maximum remained over the right central chest anteriorly while the minimum persisted over the left upper chest laterally. The peak voltage of the maximum at this time showed a decrease from the preoperative level of 3.1 mv to a postoperative level of 1.6 mv while the value of the minimum remained essentially unchanged. Also, this minimum persisted in an abnormal fashion over the
The results of the isopotential surface mapping studies in these two infants with myocardial infarction secondary to anomalous...
left coronary artery are especially interesting since there was marked cardiac enlargement and the position of the infarct was visualized at surgery to be located immediately adjacent to the lateral chest wall. This resulted in close proximity of the area of the infarct and the body surface electrode positions. At surgery in both infants, the position of the infarct in relation to the chest surface indicated that it lay in close proximity to the area where there was persistence of the left lateral chest minimum (fig. 7B and C) during inscription of the prominent Q wave in lead V6.

In most normal children the terminal body-surface potential distribution consists of a posterior maximum with an anterior minimum (fig. 6H). In the two children with myocardial infarction, the terminal distribution preoperatively was characterized by the

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Figure 11

Suggested correlation of intracardiac and body surface events in myocardial infarct secondary to anomalous left coronary artery.

1) During early ventricular excitation, in the presence of anterolateral myocardial infarction, the events should be somewhat similar to normal with the major wave front being oriented to project the maximum intensity of current onto the anterior body surface as indicated by the resultant maximum shown in lower part of figure. Reentry of current would occur in the left axillary region in the area of the minimum depicted.

2) Based on the studies of Durrer, and associates\(^1\) on myocardial infarction in dogs, it is suggested that as the major wave fronts progress through the ventricular free walls, the total dipole layer would be left with a "hole." This would result in a maximum over the anterior central chest with reentry of current through the area of the myocardial infarct which would leave the minimum localized over the area of the infarct in the left axillary region (compare with figure 7).

3) In the presence of peri-infarction block, the terminal portion of ventricular excitation should be characterized by a complex distribution of wave fronts throughout the left ventricle and septum. Although terminal wave fronts normally positioned in the posterior portion of the left ventricle may be present, this area of the left ventricle may have completed its excitation, and there would be continuing wave fronts in the muscle surrounding the infarct (peri-infarction block). These delayed wave fronts in the area near the infarct would result in current being projected onto the surface of the body in the left axillary region which would produce the terminal maximum shown and the area of reentry of current into the body could occur with its maximum intensity over the anterior central chest as indicated by the minimum.
presence of a maximum in the region of the original minimum overlying the infarct. It is of considerable interest to speculate as to the mechanisms responsible for the persistence of the early minimum in the area overlying the infarct and for the development of the terminal maximum in this area.

Durrer and co-workers have conducted extensive studies of the intramural and epicardial excitation sequence in dogs with myocardial infarcts. They found QS complexes in infarcted areas that were completely deprived of muscular tissue. They showed additionally that prolongation of QRS was caused by diminished conduction velocity of the intra-infarction and peri-infarction muscle fibers which had been preserved. They demonstrated that the phenomenon of peri-infarction block was related to decreased conduction velocity in propagation of the excitation wave fronts through the heart muscle surrounding the infarct.

Our group has had the privilege of viewing a motion picture made by Dr. Bruno Taccardi (personal communication) in which he presented a schema of the suggested projection of currents from the heart onto the body surface with the resultant distribution of equipotential lines in the normal subject. Figures 10 and 11, utilizing this approach of Taccardi’s and the information available from Durrer’s group and others, are presented to suggest a likely relationship of events within the heart to the body-surface potential distribution for normal children (fig. 10) and for those with myocardial infarction secondary to anomalous left coronary artery (fig. 11). In the normals, the initial development of the maximum over the central sternum and of the minimum in the left axilla, likely is due to the predominant wave front invading the left septal surface with the equivalent dipole layer oriented to produce current flow through the volume conductor (body) as illustrated in figure 10-1. The current flow lines on the body surface are perpendicular to the isopotential lines which would be oriented as shown. The major current density emerging at the sternum and the major density of current reentry in the left axilla would produce the isopotential line distribution indicated, as found in normal children.

As the sequence of excitation continues with invasion of the ventricular free walls, the major wave front becomes oriented in an anteroposterior direction; this could account for the body-surface voltage distribution demonstrated with an anteriorly located maximum and a posteriorly located minimum (fig. 10-2). Terminally, posteriorly oriented wave fronts in the base of the left ventricle and septum could account for the maximum over the back and minimum over the sternum with the projection of current flow onto the back and the major density of current reentry occurring in the area of the minimum anteriorly (fig. 10-3).

The body-surface potential distribution found in the infants with myocardial infarction might be accounted for by the movement of wave fronts through the ventricles as suggested in figure 11. The initial anterior maximum and left axillary minimum were similar to the normal pattern at this time in ventricular depolarization and probably resulted from the predominant wave front invasion of the left septal surface (fig. 11-1). However, with invasion of the wave fronts in the ventricular free walls, the excitation front was only propagated partially, if at all, into the infarct area. This could account for the persistence of the minimum on the body surface in an area overlying the infarct by current reentering through the “hole” produced by the infarct as originally suggested by Wilson, and associates and illustrated in figure 11-2.

The demonstration by Durrer and associates of continued fragmented wave fronts in the area surrounding the infarct (peri-infarction “block”) could provide an explanation for the persistence of current flow, as suggested in figure 11-3, to produce the terminal maximum in this overlying area. Also, on reviewing these data, Taccardi suggested to us that the terminal maximum probably represented peri-infarction “block” since the subsequent interval of the S-T segment and T
wave was characterized by negative potentials enveloping this area.

References

Dyspepsia, Asthma and Heart Disease

A Century and a Quarter Ago

Hence dyspepsia acquired the reputation of producing certain symptoms, particularly in the head, which are in reality foreign to it, being exclusively the results of a co-existent disease of the heart.

There prevails another error, the converse of the above—that of mistaking anaemic, nervous, dyspeptic, and other varieties of palpitation, for disease of the heart. . . . Having thought this subject of so much importance as to demand a separate article, (see Palpitation,) I shall here only say, that, so far as my own experience enables me to judge, the discrimination may be made with ease and certainty.

An immense proportion of asthmatics—and of the most dangerous and distressing cases, result from diseases of the heart: the same may be said of dropsies, especially those that are universal. If the cause be overlooked, the asthmatic is harassed with a farrago of inappropriate and unavailing, not to say pernicious, remedies; and the hydropic is treated with dangerous activity, or for imaginary affections of the liver, the lungs, or the kidneys. JAMES HOPE: A Treatise on the Diseases of the Heart and Great Vessels, ed. 1 American. Philadelphia, Haswell & Johnson, 1842, p. 23.
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