Relationship Between the ECG, Ventricular Activation,
and the Ventricular Conduction System
in Ostium Primum ASD

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SUMMARY
A combined electrophysiologic–anatomic study was performed in a dog with a congenital atrial septal defect (ASD) primum. Comparisons were made between the dog and electrophysiologic data obtained in four patients with ASD primum. The ECGs and VCGs in both the dog and patients revealed the characteristic features of superior orientation, counterclockwise rotation, and slow-rightward terminal forces in the frontal plane QRS. In both the dog and the patients, epicardial ventricular activation revealed early posterior LV wall excitation and later anterior left ventricular (LV) depolarization. Also, there was a marked delay in the activation of the RV in both dog and patients. In the dog, Purkinje (Pj) potentials were recorded from the endocardium of the anterior and posterior LV wall and the anterior RV wall. The endocardial-Pj activation times of these three regions were correlated with the measured lengths of the three major divisions of the conduction system supplying them. Premature activation of the posterior LV Pj and delayed activation of the anterior LV and RV Pj was observed. The marked asynchrony of Pj activation was due to a developmental asymmetry of the ventricular conduction system, characterized by markedly different lengths of the three major divisions. Purkinje conduction velocity was 1.5 m/sec, and there were no areas of block or slow conduction. The agreement between the dog and human data indicated an identical mechanism for patients with ASD primum. The terms left anterior-superior hemiblock and right bundle branch block are inappropriate descriptions of this lesion.

Additional Indexing Words:
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Although the typical ECG and VCG in ostium primum atrial septal defects (ASD) are not totally unique to this condition, they nevertheless are so characteristic that they have become a diagnostic hallmark of this form of congenital heart disease.1

The ECG pattern of superior orientation of the predominant QRS forces with right and anterior displacement of the terminal QRS and a counterclockwise frontal VCG loop, associated with most A-V cushion defects, has also been noted in adult patients with acquired heart disease2 and in certain apparently normal subjects. An interesting and somewhat perplexing question is how the same ECG characteristics can occur under such diverse circumstances.

Experimental studies by Watt et al.3,4 have established that large septal lacerations, isolating the anterior-superior left ventricular wall from the main (central) Purkinje network, result in left axis deviation in dogs and baboons. From this data it can be accepted that this ECG pattern in patients may result from disruptive lesions of the conduction...
system; however, other data suggest a different mechanism in the case of ostium primum defects. The studies of Durrer, Roos, and van Dam indicated an unusual sequence of ventricular activation characterized by earlier depolarization of the posterior (inferior) epicardial surface. Spach et al. suggested that this earlier posterior wall excitation might be explained by an abnormal distribution of the left ventricular Purkinje network. The detailed anatomic study of Feldt et al., in which measurements of the various components of the conduction system were made, was also consistent with earlier activation of the posterior left ventricular wall. In spite of these latter studies, it is still not absolutely clear how the altered anatomy of the conduction system affects the spread of activation in the ventricles. Whether the posterior (inferior) LV wall is actually activated prematurely or the anterior (superior) LV wall activation is delayed cannot be determined without direct measurements of the activation times of the anterior and posterior Purkinje system and adjacent endocardium. Additionally, the mechanism of the terminal QRS force abnormality in this defect, variously inferred to be a result of complete or incomplete right bundle branch block, is not actually known. An ECG-electrophysiologic-anatomic correlation study is required to adequately evaluate these relationships.

The opportunity to study a dog with a congenital ostium primum ASD permitted us to make these direct correlations between the surface ECG, epicardial and endocardial (Purkinje) activation sequence, and the macro- and microscopic anatomy of the heart. The data in the dog has been compared with the ECG and epicardial activation data in human subjects with ostium primum defects as a method for validating a similar mechanism of the characteristic ECG in patients with ASD primum.

**Methods**

**Ventricular Activation**

The sequence of ventricular activation was studied in four patients and one dog with ostium primum defect. In the patients, only epicardial activation data was obtained. In the dog, epicardial, intramural, endocardial, and Purkinje activation data were obtained. The endocardial activation data was obtained in the dog from the tip recording points (adjacent to the ventricular cavities) of intramural needle electrodes, and also by direct endocardial mapping during cardiopulmonary bypass. At the end of the study, the locations of the endocardial recording points on the electrodes were identified in relation to anterior and posterior papillary muscles and Purkinje network. Both bipolar and unipolar potentials were recorded. The times of the peaks of the bipolar potentials were determined in relationship to a fixed location (and time) reference electrode. Details of the methods used in the present study for correlating ventricular activation with the VCG have been published previously.

Only the data from one of the four human subjects is shown. All subjects demonstrated a similar sequence of ventricular activation with earlier posterior (inferior) wall activation and later anterior (superior) left ventricular wall activation and latest activation on the right ventricular epicardium. The patient chosen for comparison with the dog was selected because he demonstrated a similar total ventricular activation time and QRS duration. In the patients, the onset of early endocardial activation was not obtained since needle electrodes were not placed across the ventricular walls. Time zero for the epicardial maps of the patient shown in fig. 2. was arbitrarily selected to correspond to that of the dog. Epicardial activation sequence maps were constructed by identifying the location of each epicardial point on colored polaroid photographs. Purkinje system conduction velocity was calculated for spread down the right bundle branch using a special electrode grid applied to the right septal surface as described previously.

Correlation between ventricular activation and the VCG was performed by recording McFee XYZ leads preoperatively, simultaneously with selected limb leads (I, II, III or aV F). During the ventricular activation studies either lead II or aV F was recorded simultaneously with the cardiac potentials and was used to correlate the activation sequence data with previously recorded vector loops (XYZ scalar potentials).

Anatomic studies in the dog were performed as follows: Immediately at the end of the activation studies, the heart was removed; both the right and left septal bundle branch–Purkinje system were identified by iodine staining. Photographs were immediately obtained. The lengths between the common origin and the distal ramifications of the posterior and anterior radiations of the left Purkinje network and of the right bundle branch were measured. The locations at which the endocardial–Purkinje potentials were recorded were identified. Subsequently, the heart was perfused via the coronary arteries and then immersed in a buffered formalin solution. After fixation, a block including the entire specialized atrioventricular (A-V) conduction system (A-V node, bundle of His and both bundle branches) was isolated for histologic study. Nine micron serial sections were obtained on continuous film strip according to the method of Pickett and Sommer. Additional measurements of the lengths of the anterior and posterior radiations of the left Purkinje network, as well as the right bundle branch, were obtained from these histologic sections.
Results

Comparison of ECG and VCG of Patient and Dog with Ostium Primum

The six limb leads and McFee vectorcardiogram for both subjects are shown in fig. 1. Note the similar superior orientation of QRS forces as manifested in both the ECG and VCG. Also note the counterclockwise rotation of the vectorcardiographic loops and the slow terminal inscription. There were noticeable differences between the patient and dog ECG-VCG data. These were in part explained by some differences in the activation sequence as shown in fig. 2. However, even with the differences in torso-heart geometry in the dog as compared to the human, there was still similarity of superior orientation, rightward and slower terminal forces, and counterclockwise rotation of QRS.

Comparison of the Epicardial Activation Sequence in the Patient and Dog with Ostium Primum

In figure 2 (above) a correlation between the frontal VCG and the epicardial activation sequence is displayed for the patient. Three views of the heart are shown. A similar correlation for the dog with the ostium primum is illustrated below. Correlations are presented for several time points in the vectorcardiogram and the corresponding events of ventricular activation in both subjects. Additionally, comparisons can be made between the human and the dog. The time-course for the spread of ventricular activation in fig. 2 is demonstrated by the zones with different zipitone patterns; the inserted numbers indicate the activation times of local ventricular regions. The boundaries between regions of different zipitone patterns represent the location of the epicardial wavefront, at the designated instants in time. The corresponding times during ventricular activation are indicated on the accompanying frontal VCG loops. The earliest region of epicardial excitation was recorded on the posterior (inferior) surface of the left ventricle (white area) in both subjects. The earliest epicardial activation in the dog actually occurred at 17 msec, near the center of the white zone. Note that with the onset of left ventricular breakthrough at 20 msec, (i.e., the termination of wavefronts in the inferior LV wall) the loops proceeded superiorly. This was due to the presence of unopposed wavefronts in the superior aspect of the ventricles. Also, note that with breakthrough of the wavefront on the lateral aspect of the left ventricle in both subjects (at 30 msec in the patient and 35 msec in the dog) the loops were displaced laterally to the right, away from the region of breakthrough. Note that the slow segment (40 to 70 msec) of the

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**Figure 1**

ECG-VCG in Patient and Dog with ASD Primum. The standard and unipolar limb leads and frontal VCG loop are shown. Note the predominant $S$ waves in leads II, III, and $aV_R$, and predominant $R$ waves in $aV_L$ and $aV_R$ in the two subjects. Also note the counterclockwise rotation, predominant superior orientation, figure-of-S and slow terminal inscription of QRS loop.
vectorcardiographic loops in both subjects was related to isolated right ventricular activation.

An obvious difference in the pattern of epicardial activation between the patient and the dog was the presence of right ventricular breakthrough at 30 msec in the patient and later (40 msec) in the dog. There was a marked phase shift in the epicardial activation time of the right ventricle in both subjects. Normally, epicardial right ventricular activation occurs before that of left ventricular epicardial activation and frequently, depolarization of the outflow tract of the right ventricle actually precedes depolarization of the posterior basal region of the left ventricle.

Figure 2
Correlation between Epicardial Excitation Sequence and VCG in Patient and Dog with ASD Primum. Three views of the heart are shown and the frontal VCG is presented. The lines between zones of different ziptone patterns indicate the position of the wavefront at each 10 msec instant after onset of epicardial excitation. The corresponding times during ventricular activation are indicated on the accompanying VCG loops. Note that in both the patient and dog early epicardial activity (breakthrough) occurs on the posterior (inferior) LV wall, (white area), later on the anterior (superior) LV wall and finally on the RV epicardium. Posterior LV excitation was not only relatively earlier than anterior LV excitation, but occurred absolutely earlier in QRS in the dog and all of the patients. Also note that the VCG loop shifts away from sites of earlier epicardial breakthrough toward areas containing the intramural wavefront which has not yet reached the epicardial surface.

Anatomic Findings in the Dog with ASD Primum

Figure 3 illustrates a right posterior view of the heart with the right atrial wall removed. The septal defect measured 18 mm at its largest diameter. In fig. 4B, another view of the canine heart with ASD primum is presented with the left ventricular septal surface exposed by a lateral incision extending from left atrium to left ventricular apex. The left ventricle has been opened like a book and the anterior (superior) endocardial surface and anterior papillary muscle are to the left and the posterior endocardial wall and posterior papillary muscle are on the right. An iodine stain of the left ventricular endocardial-Purkinje system is illustrated. Note
the asymmetry of the conduction system in the dog with ASD primum (fig. 4B) as compared with the normal dog heart presented in figure 4A. Measurements of the various lengths of the anterior and posterior left Purkinje fascicles and the right bundle branch revealed a left posterior (inferior) division length of 49 mm, a left anterior (superior) division length of 70 mm and a right bundle branch length of 93 mm. In fig. 4B, note the posterior (inferior) displacement of the common left bundle due to the septal defect. The adherence of mitral valve tissue to the rim of the ventricular portion of the defect is characteristic of the ostium primum form of the A-V cushion lesion. The position of the anterior branches of the Purkinje network defined the junction between posterior ventricular septum and adherent mitral valve.

The Purkinje activation times for the endocardial-parietal Purkinje branches of the anterior and posterior left ventricular walls and the left bundle are different in the normal dog and the dog with the ASD primum. The earliest Purkinje activation time, that of the common left bundle, is indicated by zero msec. Note that in contrast to the almost synchronous onset of Purkinje activation in the normal dog (14 and 15 msec) there was a marked asynchrony in the activation of the endocardial surfaces of the anterior (21 msec) and posterior (8 msec) walls in the dog with the ASD primum. In comparison to this normal animal, the anterior endocardial Purkinje time was 6 msec later and the posterior wall Purkinje was 6 msec earlier in the dog with the ostium primum defect. Thus, in comparison to the normal dog, there was true delay of activation of the anterior wall and actual premature activation of the posterior wall. Because the degree of delay of the anterior wall (6 msec) so closely paralleled the degree of premature activation of the posterior wall (also 6 msec), it suggested an asynchrony of excitation due principally to the anatomic Purkinje asymmetry resulting from the posterior-inferior displacement of the conduction system. Further evidence of asynchrony of activation of the three major divisions of the Purkinje system due to asymmetry in the length of the three components is illustrated in figure 5.

Figure 5 demonstrates a correlation between the measured lengths of the three major components of the ventricular Purkinje system and the associated activation times of the endocardial surfaces supplied by these three divisions. Lead aVF is displayed with three ventricular electrograms simultaneously recorded from bipolar terminals at endocardial locations supplied by the left posterior inferior division of the Purkinje system (LPID), the left anterior superior division (LASD), and the right bundle branch (RBB). The three bipolar electrograms were recorded from points near the tips of three intramural electrodes. These electrodes were inserted across the ventricular wall in the posterior and anterior left ventricle and anterior right ventricle. By scanning each of the 14 bipolar pairs on each intramural electrode, it was possible to define the endocardial surface of the ventricle at each position and record Purkinje potentials from each of these three locations. Activation of the endocardial Purkinje branches of the left anterior-superior division (Pj in LASD) occurred 13 msec after that of the left posterior-inferior endocardial Purkinje (LPID). The earliest right ventricular endocardial Purkinje activation time adjacent to the distal part of the right bundle branch (RBB) occurred 17 msec after anterior and 30 msec after posterior endocardial Purkinje activation. Normally, the RV endocardium is activated 5-10 msec after the onset of left ventricular endocardial Purkinje activation.

The velocity of propagation of activation down the right bundle branch was 1.5 m/sec. Assuming a uniform velocity of 1.5 m/sec for the entire Purkinje
system, this value was used to calculate expected activation times for the distal ends of each of the three major branches of the Purkinje system. The lengths of each division are indicated to the left in this figure. At the bottom of this figure, the numbers represent the duration in msec between activation of the endocardial surfaces supplied by the three major divisions of the Purkinje system. The measured differences are indicated by the vertical dotted lines and can be compared with those calculated assuming a uniform Purkinje propagation velocity of 1.5 m/sec over the different lengths of the three major divisions. The closeness of this correlation indicates that the marked asynchrony of Purkinje activation must have been due to the different lengths of the three major Purkinje trunks and was a consequence of developmental displacement of the Purkinje system rather than Purkinje block or slow conduction.

**Discussion**

The data obtained in this study is relevant to the questions of whether the posterior wall activates abnormally early in ASD primum due to a short posterior Purkinje branch or whether the anterior wall activates abnormally late due to some form of left anterior superior hemiblock. The posterior left ventricular wall in this dog with a spontaneously occurring ASD primum did activate earlier than normal due to an abnormally short distance between the origin of the left posterior Purkinje division and the posterior wall. In normal dogs, posterior LV epicardial excitation occurs between 30 to 40 msec. In normal human subjects with a QRS duration of 70 msec, the posterior wall midway between base and apex is usually activated between 40 to 50 msec instead of 20 msec as in the patient shown in fig. 2. The endocardial activation of the anterior (superior) wall was also delayed,
not as a consequence of block or slow Purkinje conduction, but as a result of an increase in the length of the Purkinje conduction pathway. Also, the delay in right ventricular activation was not due to slow or blocked Purkinje conduction as the term RBBB implies, but resulted from the marked increase in the length of the right bundle branch.

Correlation between directly measured Purkinje activation times; this indicates that the asynchrony of Pj activation is determined solely by the asymmetry of the conduction system.
activation times and the measured lengths of the three different Purkinje branches demonstrated an asynchrony of endocardial activation resulting from an anatomic (developmental) asymmetry of the conduction pathways. In addition to the altered program of endocardial activation, there is a spectrum of hemodynamic alteration, resulting in different degrees and combinations of right and left ventricular hypertrophy, which probably accounts for some of the observed variations in the ECG and VCG of different patients.

Although no endocardial activation data was obtained in the patients, the similarity of their electrocardiograms, vectorcardiograms, epicardial activation sequences, as well as the gross and histologic anatomy previously reported in patients with this disease, strongly suggests a similar mechanism for the electrocardiogram in human subjects with the ostium primum defect. Thus the terms left anterior-superior hemiblock, fascicular block, complete and incomplete right bundle branch block are inappropriate terms in describing the electrocardiogram and vectorcardiogram in patients with this form of congenital abnormality.

From the epicardial, endocardial, and intramural activation data obtained in the dog, as well as from the epicardial activation data obtained in human subjects, the unique characteristics of the frontal plane vectorcardiogram can be explained. Figure 6 is a representation of the major events of ventricular activation which most profoundly influence the electro-vectorcardiographic recordings. On the right of this figure, a cross-section through the heart parallel with the frontal plane is shown. Intramural electrograms obtained in the present studies were used to construct activation fronts at three instants during ventricular depolarization (10, 30, and 40 msec). A vector equivalent of each wavefront is indicated by the arrows on the cross-section of the heart. The vector is constructed by drawing a perpendicular to the opening in the wavefront according to the method of Abildskov and Klein. This vector represents the resultant electric field due to the absence of cancelling forces. The length of each vector is proportional to the length of the opening in the activation wavefront. The frontal vectorcardiogram is shown on the left and its displacements may be correlated with the activation vectors at 10, 30, and 40 msec. Assuming uniform dipole moment for each wavefront, no force (vector) would be generated by a closed wavefront. A force (vector) equal to the area of the opening (length of dotted line in this two-dimensional case) would result due to loss of cancelling forces when the wavefront intersected a boundary (endocardium or epicardium). Note that the onset of posterior (inferior) endocardial activation resulted in inferior displacement in the vectorcardiographic loop. Because activity began posteriorly, it reached the posterior epicardial surface first. The posterior opening in the wavefront resulted in an absence of posterior (inferior) forces. The anterior (superior) forces were uncanceled at this point in time (30 msec) and resulted in a superiorly oriented vector. The effect of earlier termination of wavefronts in the left ventricle resulted in unopposed wavefronts in the RV wall at 40 msec.

Since the last phase of activation was confined to the right ventricle and there were no simultaneously enlarging or terminating wavefronts in other regions of the ventricles, there were minimal fluctuations in the spatial orientation of the cardiac field and therefore the QRS loop was characterized by a slow terminal inscription. Also, this slow terminal QRS velocity was associated with a normal rate of propagation of the localized RV activation.

In this figure, the relative lengths and angles of the vectors derived from the activation sequence are not exactly comparable to those determined for the VCG. This is because only wavefronts in one cross-section of activation are depicted and also

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**Figure 6**

Effect of Activation on the VCG. A correlation between the frontal VCG and intramural ventricular activation (charged wavefronts) for three instants in QRS is presented. Vectors representing the cardiac field for these three instants were constructed perpendicular to the opening in the activation fronts. The dotted lines indicate the length of segments necessary to close the wavefronts and the magnitudes of the vectors are equal to the openings (equal to the length of dotted lines). Note the effect of activation on the VCG.

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because the irregularities of geometry and conductive nonhomogeneity of the thoracic volume conductor alter the projected cardiac field in a complex manner. However, considering these factors, there is good correspondence in the two sets of vectors indicating the causal relationship between the VCG loop and the events of ventricular activation. The unique asynchrony of endocardial activation and the characteristic ECG-VCG features of the ASD primum are a direct consequence of posterior-inferior displacement and resultant superior-inferior and left-right asymmetry of the Purkinje network.

The previous studies of Watt and Pruitt\textsuperscript{3,4} and Rosenbaum\textsuperscript{2} have emphasized the importance of lesions of the Purkinje system in producing regional delays in ventricular activation. The present data emphasizes another mechanism of superior axis, right-anterior terminal forces, and QRS prolongation. It is possible that still other mechanisms (diseases) produce ECG characteristics similar to the present ones. Thus, the adoption of labels which implicate a specific mechanism, such as “left anterior, superior hemiblock” etc., should be replaced by a vocabulary based on a greater perspective of the multiple factors involved. Electrocardiographic terminology should be anatomically precise when possible, and also should recognize the limitations of the ECG (in any form) to define the underlying pathophysiology.

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References

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