Genesis of Body Surface Potentials in Varying Types of Right Ventricular Hypertrophy

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SUMMARY
Isopotential body surface maps and the sequence of epicardial excitation were studied in children with three types of pure right ventricular hypertrophy; that is, secundum atrial defects, valvular pulmonic stenosis, and tetralogy of Fallot. Patients with similar types of hypertrophy demonstrated internal consistency as to the anatomy, sequence of epicardial excitation of the right ventricular free wall, and body surface potential distribution; however, these phenomena varied between the three groups for each type of hypertrophy. These studies indicate that diagnostic information is available on the body surface to distinguish: (1) right ventricular hypertrophy due primarily to dilatation (secundum atrial septal defects), (2) right ventricular hypertrophy with symmetrical increase in thickness throughout the right ventricle (valvular pulmonic stenosis with moderate to severe right ventricular hypertrophy), and (3) right ventricular hypertrophy proximal to infundibular narrowing (tetralogy of Fallot) from one another. The body surface events could be accounted for in part by the events measured at the epicardial surface.

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Previous studies in normal adults and in normal children have depicted the time course during QRS of electrical events over the body surface in the form of isopotential surface maps. These studies have indicated that the changing positions of maxima and minima, as well as the presence of multiple maxima and minima, are related to the spread of wavefronts through the ventricles in a manner which is consistent with events depicted for the canine heart. Therefore, the use of isopotential body surface maps has become a valuable approach to gain further insight concerning intracardiac electrical events.

The present investigation approaches the fundamental problem in the human of the relationship of intracardiac electrical events to those produced on the body surface. Primarily because of the proximity of the right ventricle to the anterior chest wall, abnormalities of the right ventricle should be more easily detectable at the surface than those occurring in the more distant left ventricle. This study was designed to obtain partial answers to two questions: (1) How well can one differentiate various types of right ventricular hypertrophy by determining the total body surface potential distribution throughout ventricular excitation? (2) Can the body surface potential distribution be...
related to electrical events within the heart? In an attempt to answer these questions, patients with three types of right ventricular hypertrophy were studied. The types of hypertrophy were: (1) dilatation of the right ventricle secondary to secundum atrial septal defects; (2) hypertrophy of the entire right ventricle, including the outflow tract, secondary to valvular pulmonic stenosis; and (3) infundibular pulmonic stenosis (tetalogy of Fallot) with hypertrophy of the proximal portion of the right ventricle between the tricuspid valve and the infundibular stenosis and with relative normal thickness of the right ventricular free wall of the outflow tract immediately beneath the pulmonary valve (third chamber).

Methods

Patient Population

The underlying diagnosis of all patients who underwent isopotential surface mapping studies was verified by cardiac catheterization and biplane cineangiography. Fifteen patients with secundum atrial septal defects were studied by surface mapping, and at the time of cardiac surgery all 15 underwent right ventricular epicardial excitation studies. Twenty-one children with valvular pulmonic stenosis (right ventricular peak systolic pressure varied from 80 to 150 mm Hg) had surface mapping studies and, of these, 11 had epicardial excitation studies at cardiac surgery. At surgery all patients were observed to have symmetrical hypertrophy of the entire ventricle. The third group consisted of 15 patients with tetralogy of Fallot: right ventricular epicardial studies were performed on all 15 at definitive surgery. All of these patients had infundibular stenosis, marked hypertrophy of the inflow tract, and normal-appearing thickness of the right ventricular free wall in the area immediately beneath the pulmonic valve.

Data Recording and Processing Methods

The methods utilized for recording and processing electrocardiographic data to produce isopotential surface maps have been described previously. In summary, 150 electrocardiograms were recorded from the entire chest after marking a grid over the thorax and upper part of the abdomen. All data were tape recorded, transcribed with an analog-to-digital converter at a sampling rate of 926/sec, and processed by an IBM 360-75 computer. The numerical output from the computer was presented in a format which represented the thoracic potential distribution for instants spaced 1.08 msec apart.

Epicardial Excitation Studies

At the time of cardiac surgery, bipolar tracings were obtained from 20 to 30 points over the ventricular free walls. The procedure required 15 minutes and was carried out during the time of arterial cannulation by the surgeon. The bipolar recording electrode consisted of contacts 1 mm apart. Lead II or III served as a time reference and was recorded simultaneously with each epicardial tracing. All data were recorded on tape and then written out on an oscillograph at a paper speed of 400 mm/sec. For each electrogram, the apex, nadir, or base-line crossing of the intrinsic deflection was used to determine the time of local excitation at each epicardial site. These points were related, in turn, to a sharp deflection point on the reference electrocardiogram. These data were used to construct the sequence of epicardial excitation by drawing isochronous time lines. The body surface maps were time-correlated with the epicardial excitation sequence through the reference electrocardiogram which was recorded during both the epicardial and body surface map studies. By relating the reference electrocardiogram to the previously recorded isopotential surface maps, a method was provided for correlating the changing potential distribution over the body surface with the excitation sequence of the epicardium. The timing error in relating events on the epicardial surface to the isopotential body surface maps was considered to be ± 3 msec. The results of the epicardial activation sequence represent an extension of initial studies by Wallace and associates in patients with congenital heart disease.

Results

Body Surface Potential Distribution

To answer the question as to whether isopotential surface maps provide information for separation of the three types of right ventricular hypertrophy, the time course and position of maxima (highest potential) and minima (lowest potential) were determined for each group and the differences noted. These data are illustrated in figures 1 to 6. In each group the major electrical events formed a similar pattern which was easily discernible from the others. Each condition is represented by a sequence of maps recorded in a typical patient representative of each diagnostic category. The
The characteristic body surface potential distribution in normal children and in those with secundum atrial septal defect.

The upper sequence of maps (A) represents three instants of time during the first half of QRS and the sequence shown beneath (B) illustrates the sequence which was typical of patients with secundum atrial defects. The maps shown above were obtained in individual patients and are typical of the groups they represent. Lead V1 is presented beneath each map (anterior and posterior views) with the arrow indicating the instant represented by the body surface potential distribution. The potential values listed for (+) and (−) represent the values of the maximum (highest potential) and minimum (lowest potential), respectively.

A) Normal sequence in a 7-year-old child: (1) During early QRS (12 msec from onset), a maximum developed over the central chest anteriorly with a minimum over the back. (2) At 22 msec from onset, a complex distribution developed with the maximum positioned over the lower left precordium in the presence of multiple minima. This distribution has been correlated with the onset of right ventricular epicardial breakthrough from epicardial excitation studies. (3) After right ventricular epicardial breakthrough, the central sternal minimum enlarged with the upper chest becoming enveloped by negative potentials (26 msec) and the left precordial maximum began to migrate laterally around the left axillary region (see fig. 2).

B) Eight-year-old child with secundum atrial septal defect: The first half of QRS showed many similarities to the normal events. (1) Initially there was an anterior, centrally placed maximum and left axillary minimum (1 to 8 msec from onset). (2) The maximum enlarged and shifted slightly over the left precordium while the minimum assumed a position in the central back with an additional minimum developing over the upper sternum anteriorly (20 msec from onset). (3) Pseudopods projected downward from the upper part of the sternum with the rapid development of central minimum associated with a maximum over the lower precordium (42 msec).
Figure 2

Body surface potential distribution during the latter half of QRS in normals (A) and in patients with atrial septal defects (B). This figure represents the continuing events exhibited by the patients shown in figure 1.

(A) Normal sequence in a 7-year-old child: (4) During the inscription of the S wave in lead V1, the maximum migrated around the left axilla (38 msec) as negative potentials encompassed an increasingly larger area over the anterior chest. (5) Thereafter, the maximum migrated to the back and negative potentials covered most of the anterior chest (56 msec) with the minimum over the left precordial region. (6) Terminal ventricular excitation was characterized by a decreasing maximum over the back and a decreasing minimum which fragmented over the anterior central chest (66 msec).

(B) Secundum atrial defect: The events during the latter portion of QRS differed markedly from normal. (4) The preceding left precordial maximum migrated in a rightward direction across the lower central chest with pseudopod projections superiorly over the right upper chest (49 msec). Additionally, a second maximum, of smaller value than the one in normals, migrated leftward around the lower left axillary region and disappeared after assuming a position in the posterior left axilla. (5) Thereafter, the lower right chest maximum migrated superiorly along the right parasternal region (61 msec) while the minimum remained stationary over the left precordium. (6) Terminal ventricular excitation was characterized by a centrally located maximum beneath the suprasternal notch and a minimum over the lower central chest (78 msec).

The normal sequence of body surface events is illustrated in figures 1A and 2A which serve as the standard reference for comparison of the abnormal states. Normally, the events during QRS consist of the follow-
Figure 3

Body surface potential distribution in valvular pulmonic stenosis. The body surface isopotential maps illustrated in this figure and in figure 4 were recorded in a 6-year-old child with valvular pulmonic stenosis with a right ventricular pressure of 98 mm Hg. Beneath each map, lead V1 is shown with the arrow indicating the time which coincided with the body surface potential distribution shown.

(A) During early QRS there was the development of an anteriorly located maximum with an associated minimum in the left axillary region (5 msec). (B) Thereafter, the anterior central maximum enlarged and the minimum shifted to the back (14 msec). (C) Subsequently a more complex distribution developed with an enlarged maximum over the lower precordium or central sternum with multiple minima located over the upper right chest anteriorly and posteriorly (23 msec). (D) This distribution developed just prior to the onset of right ventricular breakthrough as determined by direct epicardial excitation studies (25 msec). The maximum was positioned over the lower anterior chest while the minimum assumed a position beneath the left clavicle with a second minimum over the upper part of the back.
Body surface potential distribution in valvular pulmonic stenosis during the latter half of QRS.
The sequence of isopotential surface maps shown above is a continuation for the patient demonstrated in figure 3.

(E) The distribution shown was characterized by pseudopods projecting downward into the left parasternal region from the anteriorly positioned minimum beneath the left clavicle (30 msec). This was associated with a maximum over the lower chest from which pseudopods projected in a rightward direction upward along the right parasternal region. (F) Thereafter, the maximum migrated over the right upper chest anteriorly in the right parasternal region (43 msec) while the minimum assumed the position over the left precordial region. (G) While the minimum remained stationary, superior migration of the maximum continued (60 msec). The body surface potential distribution during terminal QRS (H) was characterized by a maximum over the upper central chest beneath the suprasternal notch with an associated minimum over the lower anterior chest (70 msec).
Body surface potential distribution in tetralogy of Fallot (infundibular pulmonic stenosis). The sequence of isopotential surface maps with accompanying scalar lead $V_1$ is shown for an 8-year-old child with tetralogy of Fallot (infundibular pulmonic stenosis). The major events depicted in this patient were characteristic for all the patients studied in this group.

(A) Initial QRS was associated with an anteriorly located maximum with an accompanying left axillary minimum (7 msec from onset). (B) Thereafter, the anterior maximum increased and assumed a position over the central chest with development of a minimum over the back (14 msec). As the maximum continued to increase (C) negative potentials enveloped the upper portion of the anterior chest and back. (D) This distribution occurred 31 msec from the onset of QRS and correlated with the onset of epicardial breakthrough as determined by direct epicardial excitation studies. Note the two minima, one over the upper back and the other in the left subclavicular region from which pseudopods of negative potential project down over the left precordium. The maximum at this instant was positioned over the lower part of the chest.
Body surface potential distribution during the latter half of QRS in tetralogy of Fallot. These isopotential surface maps are a continuation of those shown in figure 5.

(E and F) The maximum migrated laterally over the right lower chest while the minimum shifted downward over the left precordium (40 and 50 msec). (G) All patients in this group demonstrated the pattern shown above with a slowly moving maximum in a lateral direction over the right lateral chest while the minimum remained over the left precordium (63 msec). Terminal ventricular excitation was characterized by the body surface potential distribution illustrated in (H) with a maximum positioned laterally in the right axillary region and a minimum over the anterior central chest (68 msec).
BODY SURFACE POTENTIALS

The onset of ventricular activation was initiated by a maximum positioned anteriorly and a minimum in the left axilla or back (fig. 1A1). (2) The minimum then migrated rapidly around the left axilla to the back with subsequent movement to the right shoulder region; this occurred concomitantly with the maximum remaining over the anterior precordium. (3) Thereafter, a second minimum appeared over the central sternum and produced the distribution shown in figure 1A2, which has been described by Taccardi as a "saddle distribution" (central anterior minimum separated from a right shoulder minimum by an area of relatively higher potential). This body surface potential distribution in patients with normal right ventricular pressures has been correlated with intracardiac events and related to the arrival of movement of the electromotive surface in the right ventricular free wall on the epicardial surface. (4) The central minimum then enlarged to envelop most of the anterior chest while the maximum shifted inferiorly and to the left (fig. 1A3). (5) The left precordial maximum then migrated around the left axilla to assume a position over the central back while the anterior chest became enveloped by increasingly negative potentials (figs. 2A-6). The movement of the maximum around the left axillary region posteriorly with the concomitant enlargement of the anterior minimum can be accounted for by the completion of right ventricular free wall excitation with the continued movement of the major wavefronts through the left ventricular free wall.

Secundum Atrial Defects (Right Ventricular Hypertrophy Primarily Due to Chamber Dilation)

The surface distribution was rather similar to normal during the first portion of QRS (fig. 1B1).

During the latter half of QRS, the surface distribution differed considerably from normal (fig. 2B). Instead of the precordial maximum migrating leftward, two maxima transiently developed in some patients. The major maximum moved across the lower sternum anteriorly to the right chest (fig. 2B4). At the same time, the left precordium became enveloped by a minimum which was positioned in the area where the conventional precordial leads are positioned for standard electrocardiography. While the minimum maintained its precordial position, the maximum migrated upward along the right sternal border to assume a terminal position beneath the suprasternal notch. The magnitude of the greatest difference in values between the potential maximum and minimum during the latter half of QRS was considerably lower than that found in patients with valvular pulmonic stenosis (those with right ventricular systolic pressures greater than 80 mm Hg) and in tetralogy of Fallot.

Valvular Pulmonic Stenosis (Right Ventricular Hypertrophy Involving the Entire Right Ventricle, Including the Outflow Tract)

The general pattern found in patients with right ventricular peak systolic pressures greater than 80 mm Hg is illustrated in figures 3 and 4. Initially, the potential maximum was located over the anterior chest while the minimum was positioned in the left axilla. Then, as the anterior maximum enlarged, minima developed over the upper chest (fig. 3B-D). Thereafter, the maximum migrated to the right, upward over the right anterior chest while a minimum enveloped the left precordial region (fig. 4E-G).

The difference in potential values between the maximum and minimum was greater than that in patients with secundum atrial defect. The final distribution was characterized by the maximum shifting into the central area beneath the suprasternal notch (fig. 4H). This terminal distribution showed many similarities to that found in the secundum atrial septal defect group.

Tetralogy of Fallot (Hypertrophy Proximal to Site of Infundibular Stenosis)

Although there were variations in the distribution of body surface potentials during the first half of ventricular activation in this group of patients, the latter half of QRS demonstrated remarkable consistency. The
general sequence found is illustrated for a typical patient in figures 5 and 6.

The sequence during the first part of QRS was similar to that found in valvular pulmonic stenosis (fig. 5) with the development of a prominent anterior maximum and upper chest minima. The left precordium then became enveloped by an enlarging minimum, which remained relatively stationary, and the maximum migrated rightward along the lower right chest (fig. 6E-H). In contrast to the sequence in valvular pulmonic stenosis and atrial defects, where the maximum during this interval migrated upward over the right anterior chest, these patients demonstrated slow movement of the maximum in a lateral direction to the right anterior axillary region. Terminally, the maximum was positioned over the right lateral chest, in contrast to the upper sternal position in the other groups of right ventricular hypertrophy.

**Epicardial Events**

Since the various types of right ventricular hypertrophy were associated with different patterns in isopotential surface maps, the next question becomes: Do different types of right ventricular hypertrophy result in varying patterns of ventricular activation? Although extensive intramural exploration of the human heart is not possible, direct epicardial measurements provide valuable information.

**Secundum Atrial Septal Defect**

The sequence of epicardial excitation was consistent in all of the patients in this group, a typical example of which is shown in figure 7. Initial epicardial activity occurred in an area of the right ventricular free wall adjacent to the interventricular septum. The epicardial wavefront then enlarged over the midportion of both ventricles (47 to 49 msec). As the epicardial wavefront enlarged and progressed across the right ventricular free wall, the area adjacent to the pulmonic valve was excited prior to the area positioned laterally at the atrioventricular groove (55 to 61 msec). The latest points of epicardial excitation occurred over the right ventricular wall adjacent to the A-V groove.

![Figure 7](image-url)

Epicardial excitation sequence in secundum atrial septal defect. The sequence shown above was obtained in the 7-year-old child with a secundum atrial defect whose isopotential surface maps are shown in figures 1B and 2B. The bar graph below illustrates the time intervals encompassed for the epicardial areas indicated by the isochronous time lines as related to the onset of QRS. The small darkened squares on the cardiac silhouette represent the recording sites. The isochronous time lines represent the epicardial areas undergoing excitation over a specific interval of time (constructed by connecting points together which were excited at similar times). In the lateral view, the posterior area is indicated with a question mark to indicate that epicardial excitation times were not recorded from the posterolateral area of the left ventricle.

The onset of epicardial excitation (epicardial breakthrough) occurred later than normal in this group of patients. Following breakthrough of activity on the right ventricular free wall in the middle-to-lower third in an area adjacent to the anterior descending coronary artery (ventricular septum), the epicardial wavefront rapidly encroached upon a broad area over the left ventricle (45 to 47 msec). Movement of the epicardial wavefront across the right ventricular free wall was characterized by its lateral and rightward migration such that the wavefront assumed a vertical orientation. The last area of epicardial excitation was located along the atrioventricular groove. Although the latest site of ventricular epicardial excitation occurred along the atrioventricular groove or the right ventricular free wall, body surface events continued for 7 to 10 msec and thereafter with a maximum positioned in an area beneath the sternal notch (cf. fig. 2B). See text.
Epicardial excitation sequence in valvular pulmonic stenosis. The sequence shown was recorded in the patient whose isopotential surface maps are shown in figures 3 and 4. It is representative of the sequential events found in the patients who underwent surgery with epicardial recordings being performed (right ventricular peak systolic pressures varied from 85 to 150 mm Hg). The blank area with the question mark in the posterolateral portion of the left ventricle indicates the area of the left ventricle from which no recordings were made.

Epicardial excitation began in the free wall of the right ventricle in an area adjacent to the ventricular septum. Thereafter, breakthrough occurred rapidly over both ventricular free walls adjacent to the anterior descending coronary artery with the wavefront moving both superiorly and inferiorly (35 msec). Left ventricular epicardial excitation occurred rapidly over a broad area as the wavefront continued to migrate across the right ventricular free wall. Note that the wavefront moved over the inferior portion of the right ventricular free wall more rapidly than in the outflow tract; this resulted in the epicardial wavefront shifting from a vertical orientation in its initial rightward movement to a more horizontal position during terminal excitation as the wavefront progressed superiorly over the outflow tract of the right ventricle (70 msec). The position of the wavefront moving superiorly over the outflow tract correlated with the body surface potential distribution of a terminal maximum positioned beneath the suprasternal notch (cf. fig. 4H).

Valvular Pulmonic Stenosis

The sequence of events in these patients also was consistent and is exemplified by the sequence shown in figure 8. Initial epicardial breakthrough occurred along the lower portion of the right ventricular free wall adjacent to the ventricular septum. The spread of excitation over the epicardium rapidly encompassed both right and left ventricles with left ventricular activation being completed well ahead of that of the right ventricle. As the wavefront progressed across the right ventricular free wall, moving from the initial septal area toward the A-V groove, the orientation of the wavefront shifted from a vertical to a more horizontal position. This resulted from excitation of the lower right ventricular free wall at the atrioventricular groove before excitation of the
Figure 10

Comparison of right ventricular epicardial excitation events and body surface potential distribution during the latter half of QRS. The schematized outline is shown to emphasize the interrelationships of right ventricular events and the resultant body surface potential distribution in three types of right ventricular hypertrophy. The insert above each isopotential surface map indicates the general wavefront orientation in the right ventricular free wall (thickened black line) associated with the accompanying body surface potential distribution. In all three types of right ventricular hypertrophy, the epicardial wavefront migrates in a rightward direction with a maximum projected onto the body surface in an area which
right ventricular outflow tract. Terminal excitation of the epicardial surface occurred in the outflow tract of the right ventricle in the area immediately below the pulmonic valve. Similar results were found by Durrer and associates\(^5\) in patients with valvular pulmonic stenosis and by Hill and Moore in congenital pulmonic stenosis in dogs.\(^6\)

**Tetralogy of Fallot**

All patients with infundibular pulmonic stenosis demonstrated a pattern similar to that shown in figure 9. As in the previous groups, initial epicardial activation occurred in an area along the lower portion of the right ventricular free wall adjacent to the anterior descending coronary artery. This was followed by rapid envelopment of both the right and left ventricles with the wavefront spreading in both an apical and basal direction (msec). The outflow tract activated earlier than in patients with valvular pulmonic stenosis. The wavefront continued its migration to the right and laterally with final depolarization being localized to the free wall adjacent to the atrioventricular groove.

**Relation of Epicardial Events to Body Surface Potential Distribution**

This portion of the study evaluated the causal relationship of right ventricular epicardial activation and the potential distribution produced at the body surface for selected instants of time. A summary of the relationships found for selected instants is presented in figure 10. It should be emphasized that in most patients studied at surgery, the epicardial tracings were more easily obtained from the surface of the right ventricle than from the surface of the left ventricle. Therefore, the correlations were made during the latter half of ventricular activation when the major wavefronts were present in the right ventricle.

**Secundum Atrial Defect (Figure 10A)**

Following the onset of epicardial excitation, which produced an "opening" in the cardiac activation front, a minimum was produced over the left precordium. The activation front which progressed rightward across the right ventricular wall resulted in a maximum over the right lower chest (fig. 10A-left). As the wavefront enlarged and progressed across the right ventricle with a vertical orientation, it shifted the maximum more rightward and superior over the right chest while the minimum remained stationary over the left precordium. The position of the final events on the epicardial surface was along the atrioventricular groove and produced a right chest maximum. However, these epicardial events terminated prior to the completion of body surface QRS events. Terminally there was a maximum located over the upper part of the sternum after completion of excitation of the entire right ventricular free wall (fig. 10A-right; cf. fig. 2B6). The genesis of this terminal maximum probably was related to activation fronts progressing through the upper part of the septum and the crista

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closely approximates the wavefront projections from its "source" side while the minimum on the body surface occurs on the opposite side or "sink" side of the wavefront.

(A) In secundum atrial septal defects, after epicardial excitation was completed over the free wall along the atrioventricular groove, QRS body surface events continued with a terminal maximum over the upper central sternum. This body surface distribution, coupled with the epicardial excitation studies, suggests that terminal ventricular excitation in these patients was associated with wavefronts migrating through ventricular muscle in the area of the crista supraventricularis and upper part of the ventricular septum.

(B) In valvular pulmonic stenosis with symmetrical hypertrophy of the right ventricle, the terminal wavefronts were oriented superiorly in the outflow tract and resulted in the projection of a maximum over the upper central part of the chest.

(C) In tetralogy of Fallot (infundibular stenosis) however, terminal wavefronts migrated laterally through the ventricular free wall in a rightward direction adjacent to the atrioventricular groove, and resulted in the projection of currents on the body surface with a maximum located laterally in the right anterior axillary region.
supraventricularis. This explanation was suggested originally for the human by Burch and DePasquale. Since no recordings were obtained from the inner portion of the heart, direct confirmation is not provided by these studies that late activation of the crista supraventricularis is responsible for the terminal QRS body surface voltage distribution.

Valvular Pulmonic Stenosis (Fig. 10B)

A few milliseconds after the onset of right ventricular breakthrough (initial epicardial excitation), the body surface potential distribution changes rapidly with the development of pseudopods extending from the left upper chest minimum into the left parasternal region in association with a maximum over the lower anterior chest (fig. 10B-left). The rapid development of the pseudopods of negative potentials was related to an enlarging area of activated epicardial surface. As the wavefront moved laterally across the right ventricular free wall, it produced a maximum over the right chest while a minimum was positioned over the left precordium (fig. 10B-middle). Terminal ventricular excitation consisted of movement of the wavefront superiorly over the free wall of the right ventricular outflow tract to produce the upper sternal maximum (fig. 10B-right).

Tetralogy of Fallot (Fig. 10C)

As occurred in the other patients with right ventricular hypertrophy, shortly after right ventricular "breakthrough" occurred over the right ventricular free wall adjacent to the septum, a maximum was projected onto the surface over the right lower midchest while the minimum was located over the left precordial region (fig. 10C-left). As the wavefront moved laterally toward the atrioventricular groove, it produced a maximum over the lower mid-right chest with a minimum in the left precordial region in an area behind the wavefront. Final depolarization of the right ventricle occurred along the lateral-inferior portion of the atrioventricular groove. The orientation of the wavefront terminally was such that it resulted in a maximum in the right axillary region with an accompanying minimum over the central chest.

Discussion

Several points deserve emphasis for this type of investigation. The design of the study was to determine whether the body surface potential distribution could be related to events actually taking place within the heart. Secondly no statistical approach was utilized. Although the patterns found were similar within each individual group, one would expect sufficient biological variation so that these patterns would tend to overlap for a very large patient population. Additionally, no attempt was made to delineate the fine details (voltage differences less than 0.1 mv) available in the total information content of the isopotential surface maps. To wit, the study was designed to provide insight into how the movement of wavefronts through the hypertrophied right ventricle projects current onto the body surface.

It is important to note several of the assumptions and limitations involved. The body surface maps were recorded preoperatively, and the epicardial excitation sequence was demonstrated at thoracotomy. The accuracy with which body surface and epicardial events can be time-aligned by this approach carries inherent error, although the consistency of the results indicates that this error is approximately ± 3 msec. Additionally, it is assumed that the pattern of ventricular excitation remained similar for both surface map and epicardial recordings. Finally, extensive exploration of the heart is severely limited by the short time available at surgery.

The first general question asked in this study was related to the reliability with which one could differentiate various types of right ventricular hypertrophy by inspecting the total body surface potential distribution throughout QRS. The implications of this question are broad and would require the study of a much larger series of patients to delineate the various overlapping patterns.

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that should be present due to biological variation. Inspection of these data does allow one to answer the following question: Can each particular patient in this study be correctly classified as normal, or as having valvular pulmonic stenosis of moderate severity (symmetrical hypertrophy of the right ventricle), tetralogy of Fallot (infundibular pulmonic stenosis), or a secundum atrial defect (symmetrical hypertrophy due to volume overload) on the basis of straightforward inspection of the surface maps?

All of the patients studied with right ventricular hypertrophy differed from normal during the interval immediately following right ventricular breakthrough. Whereas the normals demonstrated the movement of the maximum around the left axilla, patients with right ventricular hypertrophy had a maximum which migrated in a rightward direction over the right chest. A few patients with secundum atrial defects demonstrated two maxima during this interval with the major maximum migrating rightward and the minor maximum moving across the left axilla. We have studied two normal teenage children who also demonstrated two maxima during this interval; in the normals the major maximum was positioned in the left axilla with a minor maximum present for a brief interval over the lower right chest, which then rapidly disappeared. Also, no patients included in this study had mild valvular pulmonic stenosis or secundum atrial defects with small shunts. Patients with mild right ventricular hypertrophy are currently under investigation to determine distinguishing features from normal and from the more severe types of right ventricular hypertrophy.

Within the total population of patients with right ventricular hypertrophy studied, the tetralogy group was consistently different from the others during terminal ventricular activation. A maximum was positioned over the right lateral chest in the tetralogy group, whereas the terminal maximum was located beneath the suprasternal notch in valvular pulmonic stenosis and secundum atrial defects. Although the migration of the maximum over the right chest during the latter half of QRS proceeded in the same general sequence in patients with valvular pulmonary stenosis and in those with atrial defect, these two groups were easily differentiated by the much higher potential differences achieved in pulmonary stenosis than in the atrial defects. Thus, the major features noted allowed each patient to be categorized from the body surface potential maps into the proper category.

The second general question asked concerned whether the body surface potential distribution could be related to electrical events within the heart. The specific question that can be directed to the data acquired at surgery is: Are the surface map events upon which the above classification is based related in an apparent manner to the activation events detectable by epicardial measurements?

In the normals, the migration of the maximum around the left axilla is consistent with the extensive data accumulated in animals. These data indicate that wavefronts are migrating through the left ventricular free wall at a time when there is early completion of excitation of the right ventricular free wall. In all of the patients with right ventricular hypertrophy in this study, the epicardial measurements indicated the presence of wavefronts in the right ventricular free wall later than occurs in the normal. Thus, while the normal left ventricle undergoes excitation after the disappearance of most right ventricular free wall activity, in right ventricular hypertrophy prominent wavefronts continue simultaneously on the right side. Also, many patients in this study demonstrated continued right ventricular free wall wavefronts even after left ventricular excitation was completed.

The epicardial events could account for the distinguishing features between tetralogy patients (infundibular stenosis) versus valvular pulmonic stenosis (symmetrical hypertrophy). In all of the tetralogy patients the terminal wavefronts were positioned at the
lateral inferior portion of the right ventricular free wall in a position to produce the body surface maximum over the right lateral chest; similarly, in valvular pulmonic stenosis the terminal wavefronts high in the outflow tract of the right ventricular free wall correlated with the maximum positioned immediately beneath the suprasternal notch.

However, in the secundum atrial defect group, the latest epicardial events were recorded laterally at the atrioventricular groove, and several milliseconds later the terminal body surface QRS maximum was positioned beneath the suprasternal notch. Thereafter, the epicardial data do not explain the terminal surface events. The epicardial and surface data are not inconsistent, however, since the latest epicardial events occurred before the terminal QRS maximum. Thus these data are consistent with the thesis of Burch and DePasquale7 that terminal ventricular excitation in secundum atrial defects occurs in the crista supraventricularis or the upper part of the ventricular septum.

Finally, the epicardial data did not provide evidence to explain the larger voltages found in the pulmonic stenosis patients compared to the atrial defect group. The differences in magnitude most likely are related to: (1) the considerable increase in thickness of the right ventricular free wall in the patients with pulmonary stenosis studied compared to its thickness in the atrial defect group, and (2) the larger activation wavefront that should develop from the increased thickness of the wall as excitation progressed across the right ventricular free wall.

In summary, the results demonstrate in the human with right ventricular hypertrophy that alterations in the type of hypertrophy result in different patterns of ventricular excitation. Furthermore, a substantial portion of the particular differences in the surface potential distributions clearly result from corresponding particular differences in epicardial (ventricular) excitation. Moreover, the different patterns delineated on the body surface for the various types of right ventricular hypertrophy indicate that the positions of electrodes used in clinical practice assume considerable importance in achieving the ultimate diagnostic potential of electrocardiography and vectorcardiography. Alterations in the pattern of ventricular activation result in the presence of important information in areas on the body surface which currently are not sampled by most lead systems.

References

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SARAH D. BLUMENSCHEN, MADISON S. SPACH, JOHN P. BOINEAU, ROGER C. BARR, THOMAS M. GALLIE, ANDREW G. WALLACE and PAUL A. EBERT

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