Body Surface Potential Maps in Patients with Pulmonic Valvular and Aortic Valvular Stenosis of Mild to Moderate Severity

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SUMMARY Multiple-lead surface potential maps were compared throughout ventricular activation in nine patients with mild-to-moderate pulmonic stenosis and in 12 patients with mild-to-moderate aortic stenosis. Abnormal patterns of potential distribution were found in aortic stenosis, including three patients without electrocardiographic evidence of left ventricular enlargement. When related to the onset of depolarization, abnormal departures started later, peaked later, lasted longer, and were much more intense, more uniform and discrete in aortic stenosis. In pulmonic stenosis, abnormal departures started earlier, but were more dispersed in timing and location than in aortic stenosis. The left ventricle appears to be the more remote, though more powerful and compact, generator. The right ventricular shell, however, is nearer to the surface, and is more anatomically extended in surface area, permitting much wider shifts in wavefront location and orientation as a result of small differences in pressure, or volume, or location of the heart in the thorax.

RECORDING AND ANALYSIS of surface potential maps in myocardial infarction, left ventricular outflow obstruction, fascicular block and angiographically proven coronary artery disease without clinical evidence of myocardial infarction have enhanced understanding of the electrical phenomena in these conditions. Often, we can appreciate definitive findings on surface maps which are inapparent on the ECG and the vectorcardiogram. Manifestations in the body surface map of alterations in the depolarization process produced by severe right ventricular hypertrophy due to various entities have been reported. In this report, we compare the body surface potential maps in uncomplicated, mild-to-moderate outflow obstruction of the right ventricle with mild-to-moderate outflow obstruction of the left ventricle. To avoid confounding with various degrees of gross conduction defect, patients with severe, long-standing obstruction were not included.

Methods

The patients in this study were evaluated for their cardiovascular problems at the affiliated hospitals of the University of Louisville School of Medicine, Louisville, Kentucky. The cardiovascular evaluation, including histories and physical examinations, ECGs, vectorcardiograms, phonocardiograms and chest x-rays constituted the basic background data in all patients. These data are shown in table 1.

The pulmonic stenosis group included nine patients, eight of whom were catheterized. The aortic stenosis group included 12 patients, 10 of whom were catheterized and had coronary arteriograms. Patients with coronary occlusive disease, aortic or pulmonic regurgitation, other valvular heart disease, conduction defects, pulmonary disease and systemic hypertension were not included in this study.

Body surface potential recordings were made from 142 sites on analog magnetic tape. The data were originally recorded at frequencies between 0.01–3 kHz, and stored at sampling rates of 1000 bits/sec after analog-to-digital conversion. Final processing occurred either on-line or off-line using a PDP-9 computer programmed to produce symmetric projection maps of surface potential at 5-msec intervals, representing the entire sequence of ventricular activation, and to produce abnormal departure maps. The mode of obtaining the original information and processing it into departure maps is illustrated in figure 1.

To establish a 95% confidence limit, the normal range ± 2 SD of surface potentials at each location, and at each millisecond throughout ventricular activation, were acquired by recording body surface maps from a normal population of males among informed volunteers from the hospital and medical school personnel. Subjects were considered normal only if they had a complete cardiovascular history and physical examination that revealed no remarkable family history of hypertension or myocardial infarction before the age of 65 years. We excluded patients with a personal history of hypertension, smoking more than a half pack daily, obesity as determined by optimum weight for height using Metropolitan Life Insurance data, or those with abnormal lipid profiles. Additionally, the ECG, vectorcardiogram including both loops and scalar x, y, z leads, and postero-anterior and lateral x-rays of the chest, in each instance had to be within normal limits. The departure map was produced by subtracting from each patient’s
map the normal range established from this population. Fiducial point is determined by a rapid computer search of each of the 142 sites and the earliest evidence of ventricular activation was determined. Because early ventricular activation occurs at three or more relatively distant separated sites, we believe the method is relatively reliable in sensing such early activation in both the normal and abnormal states. So far, the envelope specifying the 95th percentile of potential variation at each millisecond, from 142 sites, has been a reliable method to distinguish between the normal and the abnormal person.\textsuperscript{5, 6}

To identify map abnormalities against a background of normal, the normal range at a given instant after the onset of the map and at a given location was subtracted from the corresponding instant at the same location from each subject's map, leaving only the residue of abnormal voltage distribution. A map of such residual abnormal potentials has been called a departure map.\textsuperscript{1, 2} An example is illustrated in figure 2. Instantaneous flat departure maps would indicate that no departure from the normal range occurred at any electrode site at any instant throughout ventricular activation. Instances in which potential was abnormal in a positive direction manifest as peaks in a departure map, while residual abnormal departures in a negative direction manifest as sinks or valleys.

**Results**

Directly recorded maps at 10, 20, 30, 40 and 50 msec instants from a normal subject, a patient with a gradient across the pulmonic valve of 28 mm Hg, and a patient with aortic stenosis with a gradient across the aortic valve of 64 mm Hg are shown in figure 3. Compared with the maps of a normal subject, the maps from the pulmonic stenosis patient show earlier (related to onset of activation) and more prominent positive potentials beneath and to the right of the sternum and a broad band of abnormal positivity to the left of the sternum. However, the maps of other patients with pulmonic stenosis were sometimes indistinguishable from normal when the gradient was minimal. Compared with the normal subject and the patient with pulmonic stenosis, differences are apparent in the aortic stenosis patient. The peak of positivity in the left anterolateral and upper chest is of a higher amplitude, is delayed in onset, and persists longer. The 40-msec map shows the onset of right anterior negativity in the patient with aortic stenosis; this is delayed compared with the normal subject, where it is in the 30-msec map. However, in the patient with pulmonic stenosis, this phenomenon is also seen in the 30-msec map and therefore is indistinguishable from the normal subject. As a group, however, the onset of this negativity was delayed both in aortic stenosis and pulmonic stenosis patients.

Departure maps from the patient with pulmonic stenosis and the patient with aortic stenosis discussed above were compared (fig. 4). The departure from the flat grid represents the location, the intensity, and the instant during ventricular activation where the

**Figure 1.** Method used to obtain a surface map. On the right side of the figure is shown a directly recorded map at an instant. The map has been unfolded from around the chest and tilted to give pseudo-three dimensional view of the heart’s potentials. Twenty columns and seven rows represent 140 points from where potentials are recorded. The upper boundary of the map is at the level of the sternoclavicular junction, and the lower boundary is just above the umbilicus. Columns marked VL represent the vertebral line, R and L the right and left midaxillary lines, respectively, and MSL the midsternal line. For time reference, the first surface manifestation of the ventricular depolarization is used as the zero point for the directly recorded as well as the departure maps.
patients' voltage patterns deviate from normal, ± 2 SD. Both patients show abnormal upper left chest positivity. However, in the patient with pulmonic stenosis, this is earlier, more centrally located and broader and appears as double peaks, but is of less amplitude than the pattern from the patient with aortic stenosis.

Group comparisons were possible using the mean maps derived from a sample of the normal population, the mean maps of the pulmonic stenosis patients and the mean maps of the aortic stenosis patients (fig. 5). The upper left positivity, as described above, is more prominent in aortic stenosis patients compared to that in normal subjects and in patients with pulmonic stenosis. The peak of this positivity is at 50 msec in the patients with aortic stenosis and is, therefore, delayed compared with the normal group. In the pulmonic stenosis patients the maximum peaking occurs at about 40 msec. Similarly, this positivity is sustained longer in the aortic stenosis group than in the normal subjects, or the pulmonic stenosis group, and recession, although not seen in this figure, occurred well after 50 msec. Normal upper left positivity representing left ventricular free wall excitation and indistinguishable from that seen in the normal subject is also detected in the average maps from the pulmonic stenosis patients. Finally, the average time of onset of surface negativity (possibly related to epicardial breakthrough) in both pulmonic stenosis and aortic stenosis is delayed until after 30 msec, while it occurred before 30 msec in the normals.

In an effort to focus further on significant points of comparison, we constructed maps of the mean of the abnormal departures for each group at 15, 25, 35, 45 and 55 msec instants (fig. 6). We find that the left upper abnormal positivity in the aortic stenosis group still persists, especially in the 35, 45 and 55 msec maps, and is quite discrete. However, when the abnormalities in the pulmonic stenosis group are averaged, they virtually cancel out and disappear.

Figure 7 shows abnormal departure maps from each patient in the pulmonic stenosis group at 20 msec. Abnormal positivity is present, but at greatly divergent locations. Further, the onset, peak and duration of ab-
Figure 4. Departure maps from the pulmonic stenosis and aortic stenosis patients shown in figure 3. Abnormal left positivity is seen in both patients. However, in the pulmonic stenosis patient the timing is earlier, and it is located more centrally and appears as double peaks. Its amplitude is less, however, than that of the aortic stenosis patient.

Figure 5. Group means of directly recorded maps from all the pulmonic stenosis and aortic stenosis patients at 10, 20, 30, 40 and 50 msec instants are compared with temporally comparable mean maps from a group of normal patients. Note again the more prominent, delayed and longer-lasting positivity in the aortic stenosis group. The delay in onset of right anterior negativity is evident both in pulmonic stenosis and aortic stenosis.

Figure 6. Mean maps of the abnormal departures. The dominant upper left abnormal positivity is still present in the mean maps of the aortic stenosis group, especially prominent at 35, 45 and 55 msec. The abnormal departures in the maps of individual pulmonic stenosis patients essentially disappeared with averaging.
normal positivity in patients 1 and 4 occurred before 20 msec, while in patients 6, 7 and 9, peak positive abnormality occurred well after 20 msec. Thus, individual-to-individual temporal and spatial dysynchrony is common in the pulmonic stenosis group. When all the maps representing abnormal surface voltage were pooled these diffuse and dysynchronous abnormalities canceled out (fig. 6).

Figure 8 provides a further basis for contrast of the electrical expression of the two ventricles. The abnormal departure maps at 40 msec from each patient in the aortic stenosis group are illustrated. Some variation in chest location of its expression is noted, but there was far greater consistency in the timing and location of significant abnormal positivity compared with the pulmonic stenosis group, especially in the maps from patients 1, 2, 4, 5, 6, 8, 9 and 11 (fig. 8). Thus, the dominant theme is preserved in spite of

**Figure 7.** Abnormal departure maps at 20 msec instants from all the nine pulmonic stenosis patients. Abnormal positivity is present at this instant in patients 3 and 8, but it occurred before this instant in patients 1 and 4, and after 20 msec in 6, 7 and 9, indicating temporal dysynchrony. The peak systolic gradients (mm Hg) across the pulmonic valve in these patients were as follows: #1 = 10, #2 = 22, #3 = 14, #4 = 10, #5 = 46, #6 = 16, #8 = 28. Patient 7 was not catheterized, but the presence of a grade III/VI ejection systolic murmur located in the pulmonic area, peaking at mid-to-late systole, associated with an early systolic click, decreased P2, no radiological evidence of chamber enlargement and normal ECG suggested the diagnosis of moderate pulmonic stenosis.

**Figure 8.** In contrast to the pulmonic stenosis patients (fig. 7), the abnormal departure maps at 40 msec in aortic stenosis show greater consistency indicated by the presence of abnormal positive potentials in patients 1, 2, 4, 5, 8, 9 and 11. Patients 2, 5 and 8 show map abnormalities in spite of the absence of electrocardiographic evidence of left ventricular enlargement. Peak systolic gradients (mm Hg) across the aortic valve in these patients were as follows: #1 = 50, #3 = 69, #4 = 36, #6 = 55, #7 = 48, #8 = 50, #9 = 64, #10 = 65, #11 = 60, #12 = 66. Patients 2 and 5 were not catheterized, but both were thought to have moderate aortic stenosis on the basis of III-IV/VI ejection systolic murmur located in the aortic area and peaking at mid-to-late systole, moderate decrease in the carotid upstroke and moderately prolonged ejection time, normal heart size on chest x-ray and normal electrocardiograms.
averaging, since there is less cancellation effect. In patients 1, 4, 6, 7, 9, 10, 11 and 12 the standard ECG showed changes consistent with left ventricular hypertrophy and in patients 2, 3, 5 and 8 it did not. However, in patients 2, 5 and 8 we found map abnormalities suggesting an altered sequence of excitation, in spite of the lack of routine electrocardiographic evidence of left ventricular hypertrophy. We noted no map abnormalities in patient 12, though his ECG was consistent with left ventricular enlargement and he had a 66 mm Hg peak systolic gradient across the aortic valve.

The comparison summary of the mean abnormal departures is shown in table 2. This demonstrates that the abnormal positivity in the maps of patients with aortic stenosis started later, peaked later and lasted longer compared with those from the patients with pulmonic stenosis. Similarly, the right lower abnormal negativity also started later, peaked later and lasted longer in the aortic stenosis group.

**Discussion**

Various aspects of the electrocardiographic and vectorcardiographic manifestations of uncomplicated right ventricular outflow obstruction and left ventricular outflow obstruction are well-known.\(^8\)\(^-\)\(^10\)

Details of the spread of activation recorded by body surface potential mapping in moderate-to-severe right ventricular hypertrophy in dogs\(^11\) and in humans have\(^12\)\(^-\)\(^13\) also been described. A common finding in these studies was a generalized delay in the right ventricular electrical events compared with normal subjects. This report describes the comparative aspects of the surface electrical manifestations produced by uncomplicated right ventricular and left ventricular outflow tract obstruction. To get representative samples without gross conduction defect, we studied patients with mild-to-moderate obstruction in both categories. We did not include patients with intracardiac shunts, coronary artery disease, hypertension, other valvular diseases, or those with significant pulmonary disease; nor did we include patients with pulmonic or aortic stenosis who had associated valvular insufficiency. The departure map concept was used to focus on abnormalities that exceeded the range of normal.

The complexities and limitations of translating the epicardial events to the electrical phenomenon recorded by the body surface is recognized.\(^14\) However, within limits, a predictable relationship does exist between the events on the surface of the heart and the events on the body surface.\(^12\)\(^-\)\(^16\)

Therefore, we assume that the positive potentials on the surface of the heart appear as peaks on the surface map and negative potentials as dips or valleys. Right front chest negativity has been related to right ventricular epicardial breakthrough, though no attempt is made at exact correlation of these events in terms of orientation, time and strength.\(^12\)\(^-\)\(^14\)\(^-\)\(^16\)

We do not know why, in our patients, the initial negativity, which is usually thought of in the normal as related to epicardial breakthrough, is preserved, but delayed. This is difficult to understand, when it is well-known that epicardial mapping, even in significant forms of right ventricular enlargement in the presence of atrial septal defects with R' in the ECG, has not been absolutely delayed, and the R' reflects not necessarily some degree of right bundle branch block morphology, but rather is a reflection of the enlargement of the outflow tract. We suspect that in the patients with pulmonic stenosis the delayed evidence of the early negativity usually related to right ventricular epicardial breakthrough is a reflection of some degree of temporal alteration of the usual sequence of events. We’re not certain whether this causes the surface map to be less of an analog to epicardial excitation than it is in the normal, but we are certain that patients with the disease do have these abnormal departures of their surface potential pattern. Likewise, in the group with aortic stenosis the meaning of the delay in the early anterior negativity is uncertain. We had considered that in these patients early anterior negativity represented the combined effect of right ventricular epicardial breakthrough and breakthrough on the left side of the septum and that early cancellation with later dominance of the left sided contribution prevailed. This would parallel our earlier experience with relatively severe hypertrophy of the left ventricular outflow tract with markedly delayed evidence of the first anterior negativity.\(^1\) These considerations should be considered speculative until enough simultaneous epicardial and surface maps are available to support or refute these considerations.

Compared with the pattern in normal subjects, abnormal positivity located in the upper chest anteriorly was detected both in aortic stenosis and pulmonic stenosis patients. However, this abnormality in aortic stenosis started later in the depolarization sequence and lasted longer than it did in pulmonic stenosis. The peak of this positivity also occurred later in aortic stenosis, and was more discrete and consistent in timing. The left ventricular delayed and longer-lasting positivity is understandable, and almost expected, in aortic stenosis. This dominant cardiac chamber gains even more mass, and left ventricular electrical events become predictably delayed. The advancing wavefront has a further distance to travel, and in more severe instances may even encounter areas of slowed conduction velocities, or altered pathways, as minor degrees of conduction defect develop. In contrast, this abnormality in pulmonic stenosis patients was diffuse in location and dysynchronous in time. The effect of this was further emphasized when the departure maps

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<th>Table 2. Comparison of the Departure Map</th>
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Abbreviations: PS = pulmonic stenosis; AS = aortic stenosis.
from the pulmonic stenosis patients were pooled. The abnormal potentials canceled out, leaving almost flat departure maps. In the aortic stenosis group, findings were similar enough from individual to individual to be preserved when the patients’ abnormal maps were averaged.

The effect of right and left ventricular outflow obstruction on the onset of surface negativity was also studied and compared with the similar phenomenon in normals. The aortic stenosis patients showed a delay in this phenomenon. In pulmonic stenosis patients it was also delayed, but this delay was less marked compared with the aortic stenosis group. This latter finding may have resulted from two factors. The first is the inherent discrepancy between the thinner right ventricular wall compared with the left. The second factor is our choice of patients with mild-to-moderate pulmonic stenosis as opposed to those with severe pulmonic stenosis and massive right ventricular hypertrophy.

In studies of moderate-to-severe right ventricular hypertrophy the right ventricular electrical events were delayed, and unlike normals, the depolarization process in some instances continued in the right ventricle later than in the left ventricle. In the present study of mild-to-moderate right ventricular outflow tract obstruction we detected abnormalities early in the depolarization sequence.

The disparities in the manifestations of the depolarization process between the two ventricles described above may be the result not only of fundamental anatomic, but also physiologic differences between the two ventricles. These differences are further enhanced in the course of the ventricular responses to pressure loading. For example, the left ventricle is of considerably greater mass, lies to the left posteriorly and quite deep in the chest compared with the right, and is more richly endowed with an endocardial Purkinje network. In contrast, the right ventricle has a much thinner wall, is located anteriorly and much nearer the chest wall, is somewhat less endowed with Purkinje fibers and behaves as a less powerful, though more distributed source of electrical potentials. Wider shifts, then, in the chest wall reflection of the activation process appear to result from relatively small alterations in pressure, volume, or anatomic lie in the chest in the case of right ventricular obstructive lesion compared with the left. The left ventricle, though more remote from the recording electrodes, is a significantly more powerful, discrete, compact and focused generator of electrical current, resulting in more uniformity in timing, location and magnitude of voltage expression of abnormalities related to hypertrophy secondary to a pressure load.

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