Distinguishing Features of Left Anterior Fascicular Block and Inferior Myocardial Infarction as Presented by Body Surface Potential Mapping

Gurbachan S. Sohi, M.D., and Nancy C. Flowers, M.D.

SUMMARY Total body surface maps obtained from 19 patients with previous inferior myocardial infarction (IMI) were compared with maps obtained from 19 patients with left anterior fascicular block (LAFB) and six more patients in whom electrocardiographic changes were indistinguishable between IMI and LAFB. Three distinguishing features were detected: 1) abnormal high anterior positivity developed both in IMI and LAFB, but its onset was earlier in LAFB; 2) a broad rim of abnormal right lower negativity was seen in both groups, but in IMI it was within the first 40 msec, whereas in LAFB it was found in the middle and later parts of depolarization; 3) abnormal left lower negativity was seen in all the patients with LAFB, but was absent in IMI. Thus, despite similarities in the abnormalities detected, we found definite temporal and topographical differences that should aid in differentiating between IMI and LAFB in ambiguous cases.

FREQUENTLY, it is difficult to distinguish electrocardiographic changes produced by left anterior fascicular block (LAFB) from those produced by remote inferior myocardial infarction (IMI). The reasons for this include the following: 1) The small initial r wave in leads II, III and aV_F characteristic of LAFB may be embryonic and difficult to visualize; 2) with the passage of time an initial r wave may reappear in leads II, III and aV_F after IMI; and 3) coexistence of these two entities, in which one develops in the presence of the other, may produce a masking effect of either.

Some electrocardiographic and vectorcardiographic criteria that contribute singly or in combination to distinguish one from the other have been reported. However, there appears to be no definitive means of distinction, based on the conventional means of recording the electrical activity of the heart. Previous work has shown the advantage of the surface map in more completely characterizing electrical forces in various forms of heart disease, and in IMI in particular.

In this study of 44 patients, we compared and contrasted the changes produced in the body surface map in IMI and in LAFB.

Methods

Inferior Myocardial Infarction Group

This group consisted of 19 patients in whom the diagnosis of inferoposterior myocardial infarction was established by a history of typical chest pain, diagnostic acute ST-segment elevations followed by the appearance of Q waves exceeding 30 msec in duration in the inferior leads, or an increase in the amplitude and width of r waves with acute ST-segment depression and an upright T wave in the right precordial leads. Typical rise and fall in the serum levels of glutamic oxaloacetic transaminase, hydroxybutyric dehydrogenase and lactic dehydrogenase were required to corroborate the diagnosis of acute infarction. In some of these patients, the previously present Q waves had disappeared by the time of study. Any patient with a total QRS duration > 100 msec was excluded from the study. Patients with IMI in whom coexistence of LAFB could not be eliminated with confidence because of the presence of an axis of -45° or over were not included in this study. Body surface potential recordings were made within 2–4 weeks of an acute inferoposterior myocardial infarction.

Left Anterior Fascicular Block Group

Body surface map data were also collected in 19 patients in whom a presumptive diagnosis of LAFB was made on the basis of an rs pattern in leads II, III and aV_F, qR in leads I and aV_L, and a frontal plane axis of −45° or above without other QRS abnormalities. Further, myocardial infarction was excluded on the basis of absence of any history of chest pain in all but three patients, and absence of previous electrocardiographic or serum enzyme evidence of myocardial infarction in all.

Diagnostic cardiac catheterization was performed in the three patients who had atypical chest pain. One patient had normal coronary arteries, while the other two had very minimal, hemodynamically insignificant disease in the anterior descending branch of the left coronary artery. Mild hypertension was present in three patients. Patients with lung disease, valvular heart disease, and evidence of significant coronary artery disease on coronary angiography were excluded from this study.

The patients included in this study are, as nearly as possible, representative of each group, not confounded by the other, because the purpose of the study was to define and describe distinguishing features in each group.
These maps were also compared with the data obtained from a group of 30 normal young males (ages 20–30 years) without any cardiovascular-related risk factors. These data were analyzed to obtain a mean ± 2 SD for each millisecond at each recording site to establish a normal range. This normal range was subtracted at each 5 msec from the groups under study, creating abnormal departure maps as described previously.13,14 (fig. 1). Thus, we compared not only the directly recorded maps in each group, but also the abnormal departure maps. The results of the latter comparison are reported.

**Results**

**Comparison of the LAFB and IMI Groups**

Eighteen of the 19 patients with LAFB were males. The age range of the group was 36–78 years (mean 56 years). The IMI group consisted of 18 males and one female, ages 41–76 years (mean 56 years).

Figure 2 (top) is an ECG from a patient with an old IMI showing residual Q waves in leads II, III and aVf and an axis of about −15°. Figure 2 (bottom) is an ECG from a patient with LAFB and no evidence of IMI that shows rS in leads II, III and aVf, qR in lead aVL and possibly in lead I, and an axis of about −50°. Figure 3 shows the abnormal departure maps of these two patients side by side at 30-, 45-, 60- and 90-msec instants.

Three abnormal findings were noted. The first was a zone of positivity outside the normal range located below the left clavicle that was shown in both subjects (fig. 3). However, when all the patients in both groups were analyzed and compared, significant differences in the timing of this abnormal positivity were found. The abnormal positivity in the LAFB group started at 20–35 msec and lasted until 45–60 msec after the onset of ventricular activation. In IMI, the abnormal voltage expression started later, always after 30 msec, and its location was usually more lateral, though not in the instance shown in figure 3. Such abnormal positivity, though differing in the time of occurrence, was found in 10 of the 19 patients with IMI and 15 of the 19 with LAFB.

The second abnormality was a broad rim of negativity outside the normal range in the right lower chest. It was present in 12 of 19 patients with IMI and 11 of 19 patients with LAFB. Again, the timing of this abnormal negativity was different in each group. In IMI, it was within the first 40 msec, while in LAFB it was found in the middle and later parts of the activation sequence. In three patients with IMI, the negative band exceeding normal limits extended across the midline to the left lower chest.

The third finding was a zone of abnormal negativity in LAFB in the left lower chest in 19 of 19 patients. It usually started after 25 msec (on the average at 35 msec) and lasted throughout much of ventricular activation. In some patients it was simply an extension in time of the right lower negativity described above. No such abnormality was seen in IMI except in three

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**ECG Findings Indistinguishable Between IMI and LAFB**

An additional group of six patients showed a QS pattern in leads II, III and aVf, qR in leads I and aVL, and an axis of −45° or above. On the basis of the electrocardiographic findings, distinction between IMI and LAFB could not be made with confidence. However, no evidence of myocardial infarction based on the other criteria outlined above could be found.

The maps obtained from the subjects under study represent potentials obtained from 140 points on the torso arranged in seven rows and 20 columns, 18° apart, as well as from the head and thigh. These recordings were made on analog magnetic tape and later digitized and converted into isometric projection maps of surface potentials at 5-msec intervals, using the methods described previously.16,17 The body surface map may be considered to be divided posteriorly along the spine, unrolled and tilted so that a pseudo-three-dimensional map is created (fig. 1). Maps thus obtained in each group were examined and compared at 5-msec intervals.
FIGURE 2. (top) An ECG from a patient with inferior myocardial infarction (IMI) showing Q waves greater than 30 msec in leads II, III and aV_{f}. This finding, with some slight residual ST-segment elevation and T-wave inversion, is characteristic of recent transmural myocardial infarction. (bottom) An ECG from a subject with left anterior fascicular block (LAFB) showing rS in leads II, III and aV_{f}, qR in lead aV_{l} and possibly in lead I, and a frontal plane axis of about −50°.

FIGURE 3. Departure maps of a patient with inferior myocardial infarction (IMI) whose ECG is shown in figure 2 (top) and the departure maps of a subject with left anterior fascicular block (LAFB) whose ECG is also shown in figure 2 (bottom). Abnormal upper positivity is present in both subjects, but its timing is earlier in LAFB. Abnormal right lower negativity is also present in both subjects, but lasts longer in LAFB. The abnormal left lower negativity seen in LAFB is not present in IMI (see text for details). VL = vertebral line; R = right mid-axillary line; L = left mid-axillary line; S = mid-sternal line.

TABLE 1. Comparison of Surface Map Abnormalities in Inferior Myocardial Infarction (IMI) and Left Anterior Fascicular Block (LAFB)

<table>
<thead>
<tr>
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<th>IMI</th>
<th>LAFB</th>
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<tr>
<td>Total number of subjects</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>Left upper abnormal positivity</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>Right lower abnormal negativity</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Left lower abnormal negativity</td>
<td>—</td>
<td>19</td>
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patients, in whom it was an extension of the right lower negativity mentioned above. Table 1 summarizes these findings.

The Group with ECG Findings
Indistinguishable Between IMI and LAFB

All the patients in this group were males, ages 44–66 years (mean 53 years). A typical ECG from this group is shown in figure 4, and the departure maps at 25, 30, 35, 40, 45 and 50 msec from the same patient are shown in figure 5. In this group, five out of six patients had the upper anterior abnormal positivity early in depolarization, as described above for LAFB. The left lower abnormality described above in the LAFB group was also seen in each of the six patients. None of these six patients had the right lower negativity. Though the standard electrocardiographic findings in this group did not distinguish between LAFB and IMI, the group behaved like LAFB, suggesting probable distinction between these groups.

Since the surface map abnormalities in this group of six patients without history of infarction were basically similar to the ones seen in LAFB and quite different from the ones seen in IMI, we feel that the subjects in this group represent LAFB. Therefore, data from these six patients were pooled with the data from 19 patients from the LAFB group and averaged.

For group comparison purposes, therefore, the averaged departure maps from subjects without infarction and believed to represent LAFB were compared with the averaged departure maps from the IMI patients. Departure maps at 20, 30, 35, 40, 55, 65 and 70 msec are shown in figure 6. Anterior abnormal positivity is again seen in both groups, becoming evident at 20 msec in LAFB but not until 35 msec in IMI. Anterior abnormal positivity lasted until the 55-msec instant in both groups. A broad rim of left lower abnormal negativity persists in the averaged departure maps of LAFB. It is visible in the 35-msec map and lasts throughout much of the depolarization process. No such abnormality is seen in IMI. The right lower negativity is seen in IMI starting at 30 msec and lasting up to 55 msec. When pooled, the LAFB group failed to show this abnormality. This occurred because this phenomenon was less prominent in this group with wide patient-to-patient temporal and topographical variation; pooling and averaging thus ironed out the individual, small, scattered abnormalities.

Discussion

Because of the additional temporal detail available in the body surface map, and its more extensive sampling, it occurred to us that the surface map could conceivably be helpful in distinguishing LAFB from IMI in ambiguous cases. With this in mind, body surface potential maps in two forms (directly recorded and departure) were analyzed in a group of patients who had clear-cut changes of LAFB and compared with similarly recorded maps from patients with IMI. Three distinguishing features were noted. Abnormal positivity below the left clavicle was found in both groups. However, the timing of this in
the depolarization sequence was earlier in LAFB than in IMI, and in IMI the location was somewhat lateral. The explanation for this in IMI is probably contained in the classic concept of unopposed and enhanced anterior and superior forces due to the loss of muscle posteroinferiorly. Because the early transseptal forces may be maintained intact, the departure from normal does not begin in this group until well within or after the normal Q time zone.

Experimentally produced LAFB in animals has been found to shift the mean QRS axis superiorly and to delay the activation time of the epicardium of the lateral basal surface of the left ventricle. The normal net direction of spread of depolarization, therefore, is presumably altered in LAFB, permitting abnormal spread of depolarization anteriorly and superiorly from the region already activated through the left posterior fascicle. Thus, this abnormal, superiorly directed positivity in LAFB arises from slight initial delay, sustained dysynchrony, and abnormality of direction of activation of the territory supplied by the left anterior fascicle. The left lower negativity seen in LAFB may be due to abnormalities of the timing of epicardial breakthrough as a result of the altered sequence of depolarization. The same is thought to be the likely explanation for the right lower abnormal negativity in this group. The early right lower negativity seen in IMI, however, appears to be due to the loss of inferior forces in IMI.

The group that showed indistinguishable standard electrocardiographic changes between IMI and LAFB behaved like LAFB, in that the abnormal left anterior
positivity was displayed, and all had left lower negativity not seen in IMI. Therefore, this group probably consisted of patients with LAFB and not IMI.

This analysis of body surface maps in IMI and LAFB, therefore, showed that similarities occur in the body surface potential maps in both of these entities, but differences can be seen when maps are analyzed from temporal and topographical standpoints. Thus, the body surface potential maps not only amplify electrophysiologic understanding but also should aid in differentiation in instances of confusion.

References

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