Body surface mapping improves early diagnosis of acute myocardial infarction in patients with chest pain and left bundle branch block


Objective: To test prospectively depolarisation and repolarisation body surface maps (BSM's) for mirror image reversal, which is less susceptible to artefact, in patients with acute ischaemic-type chest pain, and to compare these BSM criteria with previously published 12 lead ECG criteria.

Methods: An 80 lead portable BSM system was used to map patients presenting with acute ischaemic-type chest pain and a 12 lead ECG with left bundle branch block (LBBB). Acute myocardial infarction (AMI) was defined by serial cardiac enzymes. Each 12 lead ECG was assessed by the criteria of Sgarbossa et al and Hands et al for diagnosis of AMI. Depolarisation and repolarisation BSM's were assessed for loss of mirror image reversal of QRS with ST-T isointegral map patterns and a change in vector angle from QRS to ST-T outside 180° — findings typically seen in LBBB with AMI.

Results: Of 56 patients with chest pain and LBBB, 10 had enzymatically confirmed AMI. Patients with loss of BSM image reversal were significantly more likely to have AMI (odds ratio 4.9, 95% confidence interval 1.5 to 16.4, p = 0.007). Loss of BSM image reversal was significantly more sensitive (67%) for AMI than either 12 lead ECG method (17%, 33%) albeit with some loss in specificity (BSM 71%, 12 lead ECG 87%, 97%). Patients with AMI compared with those without AMI had a greater mean change in vector angle outside the normal range (180° ± 15°), particularly between QRS isointegral and ST60 isopotential (the potential 60 ms after the J point at each electrode site) BSM's (19° ± 9°, p = 0.038). Loss of image reversal and QRS-ST60 vector change outside 180° ± 15° had 61% sensitivity and 82% specificity for AMI (odds ratio 7.0, 95% confidence interval 2.0 to 24.4, p = 0.001).

Conclusions: BSM compared with the 12 lead ECG improved the early diagnosis of AMI in the presence of LBBB.

METHODS

Patients

Consecutive patients admitted to an acute medical cardiology unit in a tertiary hospital between September 1995 and November 1999 were mapped. Patients were included if they had chest pain suggestive of AMI with LBBB on the initial ECG and a BSM obtained within 12 hours of the onset of pain, before or within 15 minutes of starting fibrinolytic treatment (if given) and within 15 minutes of the index 12 lead ECG. Baseline characteristics were defined as: hypercholesterolaemia (fasting total cholesterol > 5.2 mmol/l (> 202.8 mg/dl) or current treatment with lipid lowering treatment), hypertension (serial blood pressure readings during admission > 160/90 mm Hg or current with antihypertensive treatment), diabetes mellitus (fasting plasma glucose > 7.8 mmol/l or a previous diagnosis requiring dietary modification, oral medication or insulin), and familial history of ischaemic heart disease (first degree relative with age of onset < 55 years for men or < 65 years for women). All patients had serum creatine kinase measured on admission and on the following day. AMI was defined as creatine kinase > 2 times the upper limit of normal (upper limit 140 IU/l) and/or increased creatine kinase MB fraction (> 25% or > 7% of the total creatine kinase).

Abbreviations: AMI, acute myocardial infarction; BSM, body surface map; GUSTO-1, global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries; LBBB, left bundle branch block; MI, myocardial infarction
12 lead ECG analysis
LBBB was considered to be present if all of the following criteria were met: QRS duration > 120 ms; small or absent initial R waves in V1 and V2 followed by deep S waves; broad, notched R waves in leads V5 and V6 and usually I and aVL; and absent septal Q waves in left sided leads. 

The 12 lead ECGs were analysed by two methods. The first method (Sgarbossa et al) allocated scores for three 12 lead ECG features: ST elevation > 1 mm in any lead concordant with the QRS complex; ST depression > 1 mm in leads V1, V2, or V3; and ST elevation > 5 mm in any lead, discordant with the QRS complex. The second method (Hands et al) required any one of the following for diagnosis of acute or prior MI: Q waves in strips, referenced to anatomical markings:

- R wave regression from V1 to V4; or V5; or primary ST -T wave changes in leads I, aVL, V5, or V6; R wave regression from V1 to V4; or V5; or primary ST-T wave changes in > 2 adjacent leads.

BSM recording
The BSM system used has previously been described. Briefly, the system consists of an anterior and posterior electrode harness, a recording unit, and a personal computer. The flexible plastic harness contains 64 anterior electrodes (including proximal limb electrodes) and 16 posterior electrodes. Silver–silver chloride electrodes are screen printed onto the harness, with self adhesive hydrogel pads allowing good skin contact. The electrodes are arranged in 11 cardiac vectors, referenced to anatomical markings: anterior (from right to left)—right mid clavicular line, right parasternal line (V1 line), left parasternal line (V2 line), left medial-clavicular line (V3 line), left mid clavicular line (V4 line), left anterior axillary line (V5 line) and left mid axillary line (V6 line); and posterior (from right to left)—right mid axillary line, right posterior axillary line, left paraspinal line, and left posterior axillary line. BSMs are recorded over five seconds at a sampling rate of 1 kHz simultaneously and bandwidth of 0.05–100 Hz. The digital data are stored on a 512 kb memory card and downloaded on to a personal computer.

Data processing and display
Data were processed using custom software. Noise was removed using subtraction filtering. Leads of poor quality were manually identified and subtracted with values obtained by linear grid interpolation. To avoid excessive reliance on interpolation, a recording was deemed unusable if there were more than six poor quality channels, orthogonal adjacent poor quality channels (that is, horizontally or vertically but not diagonally adjacent), or any posterior poor quality channels. The QRS complex providing the best overall signal quality was selected. Markers were placed at QRS onset, J point, and T wave offset (but before any U wave). Maps were displayed in colour contour format with green equal to zero, positive values were given linear allocation from green towards red and negative values were given linear allocation from green towards blue. The most positive value was termed the "maximum" and the area surrounding it (> 50% of the value of the maximum) was termed the "maxima". The most negative value was termed the "minimum" and the area surrounding it (> 50% of the value of the minimum) was termed the "minima". The cardiac vector was drawn from minimum to maximum. Colours were displayed on a flat representation of the thorax, depicted as if "unwrapped" from a vertical split at the right axilla (fig 1).

BSM analysis
QRS and ST-T isointegral maps (that is, the area under the QRS and ST-T curves, respectively, at each of the electrode sites) were displayed in addition to ST0 and ST60 isopotential maps (the potential 0 ms and 60 ms, respectively, after the J point at each electrode site). The normal QRS isointegral map of a patient with LBBB but without AMI (fig 1A) typically shows maximum potential located over the left anterior chest, left axilla, or left posterior chest; with minimum potential located on the upper midanterior chest. The normal ST -T iso- integral map from a patient with LBBB is typically a mirror image of the QRS map—that is, maximum potential on the upper or midanterior chest and minimum potential located over the left anterior chest, left axilla, or left posterior chest. Thus, the cardiac vector (drawn from minimum to maximum) typically changes direction by approximately 180°

For this study, mirror image reversal was defined to be present if the central point of the maxima on the QRS isointegral map concurred (>1 electrode position horizontally, vertically, or diagonally) with the central point of the minima on a repolarisation map (for example, ST-T isointegral map) and the central point of the minima on the QRS isointegral map concurred (>1 electrode position) with the central point of the maxima on the ST-T isointegral map. Loss of image reversal was considered to be suggestive of AMI (fig 1B). A normal change in vector angle (for LBBB without AMI) on comparing depolarisation (QRS isointegral) with repolarisation maps (ST-T isointegral, ST0 isopotential, or ST60 isopotential) was defined as 180±15°. A change in direction outside these limits was considered to be suggestive of AMI.

Statistical analysis
Baseline categorical variables were analysed by chi-squared and continuous clinical variables by analysis of variance or Mann-Whitney U test as appropriate. A probability value of p < 0.05 was taken as significant. All patients were required to give informed consent, using a local ethics committee approved patient information sheet and consent form.

RESULTS
Patient demographics
Fifty six patients with acute chest pain were identified who met enrolment criteria, 18 with AMI as diagnosed by cardiac enzymes (of whom seven received a fibrinolytic agent) and 38 without significant enzyme rise (of whom three received a fibrinolytic agent based on initial clinical suspicion of AMI). Table 1 shows baseline demographics.

12 lead ECG analysis
Sgarbossa et al criteria correctly identified 6 of 18 patients with enzymatically confirmed AMI and excluded AMI in 37 of 38 patients (sensitivity 33%, specificity 97%, positive predictive value 86%, and negative predictive value 76% for AMI). Hands et al criteria correctly identified only 3 of 18 patients with enzymatically confirmed AMI and excluded AMI in 33 of 38 patients (sensitivity 17%, specificity 87%, positive predictive value 38%, and negative predictive value 69% for AMI).

BSM analysis
Image reversal analysis (whereby loss of mirror image reversal comparing QRS and ST-T isointegral maps was suggestive of AMI) correctly identified AMI in 12 of 18 patients and excluded AMI in 27 of 38 patients (67% sensitivity, 71% specificity, 52% positive predictive value, and 82% negative predictive value for AMI) (table 2). Patients with loss of image reversal were significantly more likely to have AMI (odds ratio (OR) 4.9, 95% confidence interval (CI) 1.5 to 16.4; p = 0.007). BSM image reversal analysis was significantly more sensitive for AMI than either lead ECG method (p < 0.05 v Sgarbossa et al; p < 0.001 v Hands et al) with some loss in specificity (Sgarbossa et al p < 0.003; Hands et al p < 0.001).

Comparing the QRS isointegral with each of the three repolarisation BSMs (ST-T isointegral, ST0 isopotential or ST60 isopotential) and taking loss of mirror image reversal in at least two of three maps as suggestive of AMI, sensitivity was 56% and specificity 74%.
Change in vector direction from QRS isointegral to each of the three repolarisation BSMs (ST-T isointegral, ST0 or ST60 isopotential) showed that those with AMI compared with those without AMI had a greater mean change in vector angle outside the normal range (180° ± 15°), particularly for the QRS-ST60 comparison (19° vs 9°, p = 0.038).

Loss of image reversal and QRS-ST60 vector change outside 180°±15° correctly identified AMI in 11 of 18 patients and excluded AMI in 31 of 38 patients (61% sensitivity, 82% specificity, 61% positive predictive value, and 82% negative predictive value for AMI). Patients with loss of image reversal and QRS-ST60 vector change outside 180°±15° were

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Baseline characteristics of patients presenting with chest pain and left bundle branch block</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All patients (n=56)</td>
</tr>
<tr>
<td>Mean age (years) (range)</td>
<td>68 (38–88)</td>
</tr>
<tr>
<td>Male sex</td>
<td>42 (75%)</td>
</tr>
<tr>
<td>Smoking current/former</td>
<td>15 (27%)/20 (36%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>24 (43%)</td>
</tr>
<tr>
<td>Diabetes NIDDM/IDDM</td>
<td>4 (7%)/2 (4%)</td>
</tr>
<tr>
<td>Family history of IHD</td>
<td>28 (50%)</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>27 (48%)</td>
</tr>
<tr>
<td>Previous angina pectoris</td>
<td>41 (73%)</td>
</tr>
<tr>
<td>Previous MI</td>
<td>26 (46%)</td>
</tr>
<tr>
<td>Previous PCI</td>
<td>13 (23%)</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>6 (11%)</td>
</tr>
</tbody>
</table>

* Diagnosis based on serum cardiac enzymes.
CABG, coronary artery bypass grafting; IDDM, insulin dependent diabetes mellitus; IHD, ischaemic heart disease; MI, myocardial infarction; NIDDM, non-insulin dependent diabetes mellitus; PCI, percutaneous coronary intervention.
significant more likely to have AMI (OR 7.0, 95% CI 2.0 to 24.4; p = 0.001).

DISCUSSION

The diagnosis of AMI in the presence of LBBB is of key importance, as the high mortality associated with this condition can be reduced by fibrinolytic treatment. Blind administration of a fibrinolytic agent, however, to all patients with chest pain suggestive of AMI and LBBB on the 12 lead ECG is not an optimal practice because of the risk of severe haemorrhage, especially intracranial bleeding, estimated to occur in LBBB without AMI.

In the past, to diagnose AMI in the setting of LBBB, several approaches to 12 lead ECG analysis have been proposed but the results have been disappointing (table 3). Sgarbossa et al.11 analysed 131 patients with LBBB and AMI enrolled as part of the GUSTO-1 (global utilization of streptokinase and tissue plasminogen activator for occluded coronary arteries) trial and reported criteria with 36% sensitivity, 96% specificity, 88% positive predictive, and 61% negative predictive values for AMI. However, one of the components, ST elevation > 5 mm in any lead discordant with the QRS complex of that lead, has been found by Madias et al.12 to occur in LBBB without AMI. Hands et al. tested 11 ECG criteria for diagnosis of acute and/or prior MI in 35 patients with LBBB (24 having AMI or prior MI). Four of these criteria were found to be highly specific (91–100%) and predictive (86–100%) but to have poor sensitivity 21–29%. Eriksson et al.1 and Shlipak et al.6 also reported poor sensitivity. Wackers et al.17 tested 11 ECG criteria for diagnosis of acute and/or prior MI in 35 patients with LBBB (24 having AMI or prior MI). Of these criteria, ST elevation had a sensitivity of 54%, abnormal Q waves 31%, and notching of the upstroke of the S wave in V3 or V4 27%. He also found serial ECG changes to be most sensitive for diagnosis of AMI in the presence of LBBB (sensitivity 67%), but time to treatment is crucial in managing AMI.

Almost all work performed on BSMs in patients with ischaemic heart disease with LBBB has been recognising prior MI.18–21 Musso et al.22 were not able to identify prior MI in patients with LBBB by visually inspecting BSMs but differences in potential magnitudes were observed, whereby lower values were seen in patients with prior MI than in those with uncomplicated LBBB. Suzuki et al.23 recorded QRST isointegral maps during sinus rhythm and right ventricular pacing in 62 patients with previous MI and 26 patients without MI. Abnormalities over the anterior left chest with old MI.

Table 2  Image reversal and change in vector angle comparing depolarisation and repolarisation maps

<table>
<thead>
<tr>
<th>Reference</th>
<th>ECG criteria</th>
<th>Acute myocardial infarction (%) (n=18)</th>
<th>Not acute myocardial infarction (%) (n=38)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sgarbossa et al.8</td>
<td>Q wave in &gt;2 of leads I, aVL, V5, V6</td>
<td>29 (91) 88%</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Hands et al.9</td>
<td>R wave regression V1-4</td>
<td>21 (100) 100%</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Sgarbossa et al.8</td>
<td>S wave notching &gt; 2 of leads V3-5</td>
<td>29 (91) 88%</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Eriksson et al.7</td>
<td>Primary STT changes in &gt; 2 adjacent leads</td>
<td>25 (91) 86%</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Eriksson et al.7</td>
<td>Concordant ST elevation &gt; 1 mm</td>
<td>21 (74)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Wackers8</td>
<td>ST depression &gt; 1 mm V1-3</td>
<td>7 (100)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Sgarbossa et al.8</td>
<td>R wave regression V1-4</td>
<td>7 (100)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Wackers8</td>
<td>S wave notching V3-5</td>
<td>29 (84)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Wackers8</td>
<td>ST/ T wave concordant QRS</td>
<td>27 (NA)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Hands et al.9</td>
<td>Cabrera’s sign</td>
<td>21 (NA)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Hands et al.9</td>
<td>Chapman’s sign</td>
<td>21 (NA)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Hands et al.9</td>
<td>Abnormal Q waves</td>
<td>31 (NA)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>ST elevation &gt; 1 mm in concordant leads</td>
<td>7 (100)</td>
<td>100%</td>
<td>71</td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>ST depression &gt; 1 mm in lead V1, V2, or V3</td>
<td>3 (100)</td>
<td>100%</td>
<td>71</td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>ST elevation &gt; 5 mm in discordant leads</td>
<td>19 (82)</td>
<td>32%</td>
<td>70</td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>QRS notching</td>
<td>39 (57)</td>
<td>28%</td>
<td>68</td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>RS V6</td>
<td>26 (79)</td>
<td>35%</td>
<td>71</td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>Cabrera’s sign</td>
<td>7 (86)</td>
<td>17%</td>
<td>68</td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>Discordant ST elevation &gt; 7 mm or discordant ST depression &gt; 2 mm</td>
<td>3 (99)</td>
<td>50%</td>
<td>70</td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>Positive T waves in lead with upright QRS</td>
<td>3 (93)</td>
<td>17%</td>
<td>69</td>
</tr>
<tr>
<td>Shlipak et al.6</td>
<td>Chapman’s sign</td>
<td>3 (92)</td>
<td>14%</td>
<td>68</td>
</tr>
</tbody>
</table>

Cabrera’s sign is notching of the upstroke of the S wave in lead V3 or V4. Chapman’s sign is notching of the upstroke of the R wave in lead 1, aVL, or V6. NA, Not available.
were seen. For diagnosing prior MI they found 84% sensitivity and 81% specificity if the sum of the QRST isointegrals below the normal range exceeded 100 mVms. Nishiyama et al. compared QRST isointegral BSMs, 12 lead ECGs, and vectorcardiograms in paced rhythm in controls and in patients with prior inferior MI and found that, of the three methods, BSM had superior correlation with the severity of left ventricular wall motion abnormalities.

During mapping of patients with AMI with LBBB, it has been found that the injury currents generated by acutely infarcting tissue disturb the patterns of repolarisation typically seen in patients with LBBB uncomplicated by AMI. Thus, AMI may result in loss of the mirror image reversal of the positions of maxima and minima normally seen between the depolarisation (QRS) and repolarisation (ST-T) maps. Similarly, AMI may result in a change in the cardiac vector (running from minimum to maximum) outside the normal 180°–15° typically seen between QRS and ST-T maps. However, measurement of the vector angle may be susceptible to even minor degrees of artefact, particularly in posterior electrodes, leading to an erroneous change in vector angle. Furthermore, eccentric loci of maximum or minimum positions within maxima and minima areas, respectively, may lead to apparently abnormal vector changes despite mirror image reversal between QRS and ST-T isointegral maps. Thus, the measurement of the vector angle may be misleading such a result.

The results obtained using BSM are encouraging. Sensitivity for AMI is significantly superior to that of 12 lead ECG algorithms. Although specificity is reduced, it is within acceptable limits to allow this method to be clinically useful. Analysis of patient subgroups and other cardiac conditions such as those with prior MI, left axis deviation, and left ventricular hypertrophy was not possible, as numbers would have been too small to draw meaningful conclusions.

The importance of sensitivity over specificity in patients with LBBB and AMI should be interpreted with caution, nevertheless, our results with BSM image reversal compared with Sgarbossa et al. 12 lead ECG criteria would enable correct identification of an additional 102 patients, but would not necessarily identify the number of false positives identified by BSM.


REFERENCES


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Heart 2003 89: 998-1002
doi: 10.1136/heart.89.9.998

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