



Pre-hospital body surface potential mapping improves early diagnosis of acute coronary artery occlusion in patients with ventricular fibrillation and cardiac arrest

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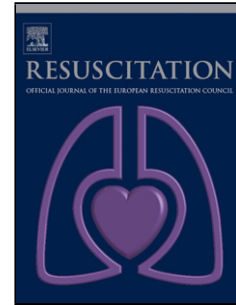
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1 Pre-hospital Body Surface Potential Mapping improves early
2 diagnosis of acute coronary artery occlusion in patients with
3 ventricular fibrillation and cardiac arrest.

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24

25

26 **Abstract**

27 Aims

28 To determine whether 80-lead body surface potential mapping (BSPM) improves detection of
29 acute coronary artery occlusion in patients presenting with out-of-hospital cardiac arrest
30 (OHCA) due to ventricular fibrillation (VF) and who survived to reach hospital.

31

32 Methods and Results

33 Of 645 consecutive patients with OHCA who were attended by the mobile coronary care unit,
34 VF was the initial rhythm in 168 patients. Eighty patients survived initial resuscitation, 59 of
35 these having had BSPM and 12-lead ECG post-return of spontaneous circulation (ROSC) and
36 in 35 patients (age 69 ± 13 yrs; 60% male) coronary angiography performed within 24hrs
37 post-ROSC. Of these, 26 (74%) patients had an acutely occluded coronary artery (TIMI Flow
38 Grade [TFG] 0/1) at angiography. Twelve-lead ECG criteria showed ST-segment elevation
39 (STE) myocardial infarction (STEMI) using Minnesota 9-2 criteria - sensitivity 19%,
40 specificity 100%; ST-segment depression (STD) ≥ 0.05 mV in ≥ 2 contiguous leads -
41 sensitivity 23%, specificity 89%; and, combination of STEMI or STD criteria - sensitivity
42 46%, specificity 100%. BSPM STE occurred in 23 (66%) patients. For the diagnosis of TFG
43 0/1 in a main coronary artery, BSPM STE had sensitivity 88% and specificity 100% (c-
44 statistic 0.94), with STE occurring most commonly in either the posterior, right ventricular or
45 high right anterior territories.

46

47 Conclusion

48 Among OHCA patients presenting with VF and who survived resuscitation to reach hospital,
49 post-resuscitation BSPM STE identifies acute coronary occlusion with sensitivity 88% and
50 specificity 100% (c-statistic 0.94).

51

52

53

54 **Introduction**

55

56 Sudden out-of-hospital cardiac arrest (OHCA) is associated with a poor survival [1]. Of those
57 who survive to reach hospital, Herlitz *et al* [2] have indicated one-month mortality between
58 58% and 86%. Ventricular fibrillation (VF), the most common arrhythmia underlying sudden
59 cardiac death in adults, is triggered mainly by myocardial ischaemia [3, 4]. Acute myocardial
60 infarction (AMI) is one of the main causes of OHCA [1]; coronary occlusions have been
61 documented in 17-48% and significant coronary disease (>50% stenosis) in 25-70% of
62 patients [5].

63 Recent guidelines recommend that patients resuscitated from OHCA who are suspected of
64 having coronary artery occlusion as a precipitant factor should undergo early/immediate
65 coronary angiography with primary percutaneous coronary intervention (PPCI) as indicated
66 [6, 7]. Dumas *et al* [7] showed the prognostic value of ST-segment elevation on 12-lead ECG
67 for coronary artery occlusion in the setting of OHCA to be poor. ECG changes may be
68 difficult to interpret in patients resuscitated from OHCA since acute ischaemia-reperfusion
69 syndrome may also cause myocardial injury, leading to significant ECG changes even in the
70 absence of AMI, i.e. an acutely occluded coronary artery [1].

71 Body surface potential mapping (BSPM) has been shown to improve AMI diagnosis in
72 patients with acute chest pain by detection of ST-segment elevation 'missed' by the 12-lead
73 ECG [8-10]. In this study, we hypothesised that immediate BSPM post-return of spontaneous
74 circulation (ROSC) in patients suffering VF OHCA would improve pre-hospital diagnosis of
75 acute coronary occlusion.

76

77

78 **Methods**

79

80 **Study Population**

81 Retrospective analysis of all patients suffering OHCA, attended by the physician-lead mobile
82 coronary care unit (MCCU) and admitted to our coronary care unit between 01 January 2003
83 and 01 January 2006 was undertaken. The MCCU (physician, specialist cardiology nurse,
84 electrocardiographer and paramedic) provides pre-hospital care for a regional cardiology
85 centre, serves a predominantly caucasian, inner-city population of approximately 300, 000
86 patients and operates 24/7. Patients were included if they had:

87

- 88 1. OHCA;
- 89 2. Initial rhythm VF;
- 90 3. BSPM and 12-lead ECG acquired immediately post-ROSC;
- 91 4. Blood sampled for cardiac troponin T (cTnT) \geq 12hrs post-ROSC; and
- 92 5. Coronary angiography <24hrs post-ROSC

93

94 Demographic data and risk factors for coronary artery disease were documented.

95

96 **BSPM and 12-lead ECG analysis**

97 BSPM and 12-lead ECG analysis was undertaken immediately post-ROSC by the physician
98 leading the MCCU. BSPM was recorded as part of a research protocol using a flexible plastic
99 anterior and posterior electrode harness and a portable recording unit (HeartScape
100 Technologies, Inc.). Application of both the anterior and posterior electrode harnesses takes
101 3-4mins in the post-ROSC patient. The anterior harness contains 64 electrodes, including 3
102 proximally located limb lead electrodes (Mason-Likar position) and a posterior harness with
103 16 electrodes. During the interpretation process electrode locations are categorised to
104 represent anterior, lateral, inferior, high right anterior, right ventricular and posterior
105 epicardial regions [11, 12].

106

107 The BSPMs were uploaded and displayed on a personal computer running PRIME™ analysis
108 software. Printouts were obtained from the processed BSPM of the 80-lead ECG and a
109 colour-contour map displaying ST-segment elevation at the J point (ST0 isopotential map).
110 Using the 80-lead BSPM and colour-contour map, a single cardiologist familiar with BSPM

111 interpretation and blinded to the clinical details and 12-lead ECG coded the BSPM diagnosis
112 as AMI or non-AMI and defined the infarct location. ST-segment elevation measured at the
113 ST0 isopotential point was defined by the following thresholds: anterior $\geq 0.2\text{mV}$ elevation;
114 lateral/inferior/high right anterior/right ventricular $\geq 0.1\text{mV}$ elevation; posterior $\geq 0.05\text{mV}$
115 elevation; with in addition infarct-location described by the ST0 isopotential colour-contour
116 map.

117
118 Twelve-lead ECG abnormalities recorded were ST-segment elevation (STE), ST-segment
119 depression (STD), T-wave inversion (TWI), left (LBBB) and right (RBBB) bundle branch
120 block and non-specific QRS widening. ST segment shifts were measured at the J-point for
121 STE and 80ms after the J-point for STD using the preceding TP segment as a baseline. STE
122 consistent with AMI (STEMI) was defined using the Minnesota 9-2 criteria [13] as $\geq 0.1\text{mV}$
123 STE in one or more of leads I, II, III, aVL, aVF, V₅, V₆ or $\geq 0.2\text{mV}$ STE in one or more of
124 leads V₁ – V₄. STE in lead aVR was defined as $\geq 0.05\text{mV}$. STD was considered significant if
125 $\geq 0.05\text{mV}$ in ≥ 2 contiguous leads. LBBB was defined as QRS duration $\geq 120\text{ms}$ with QS or rS
126 pattern in V₁ and broad R waves in lead I, V₅ and V₆ [1]. RBBB was defined as QRS duration
127 $\geq 120\text{ms}$ with rSR' complex in V₁ and V₂ and S wave in lead I and V₅ or V₆ [1]. Non-specific
128 QRS widening was defined as QRS duration $\geq 120\text{ms}$ without LBBB or RBBB morphology.
129 Twelve-lead ECG analysis was verified on arrival to hospital by a cardiologist blinded to all
130 other clinical details.

131

132 **Coronary Angiography**

133 All patients underwent coronary angiography $< 24\text{hrs}$ post-ROSC. Flow in the culprit artery
134 was graded according to the TIMI flow grade (TFG) criteria. AMI was angiographically
135 defined by the presence of an occlusion in a main coronary artery with TIMI 0/1 flow [14].
136 To avoid misdiagnosing chronic occlusions as AMI, the occlusion had to be easily crossed by
137 an angioplasty guide wire [1] and cTnT concentration was required to increase to $\geq 0.03\mu\text{g/L}$
138 $\geq 12\text{-hours}$ post-ROSC.

139

140 **Statistical Analysis**

141 Data are presented as number (%), mean \pm standard deviation or median (interquartile range).
142 Group comparisons were tested using the unpaired t test and χ^2 test. Continuous clinical
143 variables were tested by analysis of variance. Diagnostic accuracy of the various diagnostic
144 parameters employed were assessed using ROC analysis, with c-statistic (area under ROC

145 curve [AUC]) > 0.75 taken as a satisfactory performance.. Statistical analysis was performed
146 using SPSS version 17.0 for Windows (SPSS Inc, Chicago, Illinois). A p-value < 0.05 was
147 considered statistically significant. Ethical approval for the study was granted by the Local
148 Ethics Committee.

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151 **Results**

152

153 During the study period, 645 patients suffered OHCA and were attended by the MCCU. VF
154 was the initial rhythm in 168 patients. Eighty patients survived initial resuscitation, 59 of
155 whom had BSPM and 12-lead ECG post-ROSC. Of these, 24 patients suffered further OHCA
156 and died pre-hospital prior to coronary angiography. Enrolled were 35 patients (age $69 \pm$
157 13 yrs; 60% male) [Figure 1]. Demographic data are presented in Table 1. Time from OHCA
158 to ROSC was 22 (12, 31) minutes with time from ROSC to coronary angiography 74 (50,
159 126) minutes. At angiography, 26/35 (74%) patients had acute occlusion of a main coronary
160 artery with TIMI 0/1 flow. Of these, 10/26 (38%) patients had triple vessel coronary artery
161 disease. Overall, 29 (83%) patients had cTnT $\geq 0.03 \mu\text{g/L}$ ≥ 12 hrs post-ROSC, i.e. diagnostic
162 sensitivity 92% and specificity 44% for acute coronary occlusion at angiography.

163

164 Diagnostic performances of 12-lead ECG criteria assessed are summarised in Table 2. Of
165 particular note, STEMI by Minnesota 9-2 criteria and STD in ≥ 2 contiguous leads occurred in
166 only 5/35 (14%) and 7/35 (20%) patients respectively. Combination of either STEMI or STD
167 on 12-lead ECG had diagnostic sensitivity 46% and specificity 100% for acute coronary
168 occlusion. In addition, the combination of LBBB, RBBB or non-specific QRS widening
169 occurred in 10/35 (29%) patients and had sensitivity 31% and specificity 78% for acute
170 coronary occlusion diagnosis.

171

172 BSPM performed immediately post-ROSC showed ST-segment elevation detected by
173 Cardiologist in 23/35 (66%) patients and had sensitivity 88% and specificity 100% for
174 diagnosis of acute coronary occlusion (c-statistic 0.94; 95% CI: 0.83 – 0.98). Of these, 16
175 (70%) patients had ST-segment elevation in either the posterior, posterolateral, right
176 ventricular or high right anterior territories (Figure 2). In patients with acute coronary
177 occlusion and ST-segment elevation detected by BSPM only (n=18), culprit coronary
178 occlusions were located in the LCx in 6 (33%), LMS in 5 (28%), LAD in 4 (22%) and RCA
179 in 3 (17%) patients. Of the remaining 8 patients with acute coronary occlusion, 5 patients had
180 ST-segment elevation on the 12-lead ECG and BSPM (LAD occlusion in 3 patients; RCA
181 occlusion in 2 patients).

182

183

184 **Discussion**

185

186 In many patients, sudden cardiac death is the first and only symptom of coronary artery
187 disease [3]. If AMI (acute coronary occlusion) is the cause of cardiac arrest, early reperfusion
188 therapy is of utmost importance [3, 14, 15]. The early out-of-hospital 12-lead ECG can
189 facilitate a fast-track decision on the reperfusion strategy, including immediate pre-hospital
190 thrombolysis [3]. Where PCI is considered, pre-hospital diagnosis of STEMI has the potential
191 to lead to substantial time savings [3].

192

193 The PROCAT registry represents the largest cohort of OHCA patients with coronary
194 angiographic data (n=435) [7]. In this population, 68% patients had initial VT/VF. At least 1
195 significant (>50% reduction in luminal diameter) coronary artery lesion was found in 70% of
196 all patients and in 96% and 58% patients with and without ST-segment elevation on the post-
197 resuscitation ECG respectively [7]. Furthermore, in the total population triple-vessel coronary
198 disease was found in 37% patients and a culprit lesion identified in 202 (46%) patients, most
199 commonly the left anterior descending artery (107/202 [53%]) [7]. In our study, among
200 patients suffering VF and OHCA, 26/35 (74%) had acute occlusion of a main coronary artery
201 at angiography. Of these, no patient had ST-segment elevation detected only by the post-
202 ROSC 12-lead ECG. ST-segment elevation on the standard 12-lead ECG immediately post-
203 ROSC had poor diagnostic sensitivity for the diagnosis of acute coronary occlusion. Of those
204 without ST-segment elevation on 12-lead ECG (n=30), 21/30 (70%) patients had an acute
205 occlusion of a main coronary artery. ST-segment elevation on pre-hospital BSPM improved
206 sensitivity (88%) and maintained specificity for the diagnosis of acute coronary occlusion
207 when compared to the post-ROSC 12-lead ECG. Sideris *et al* [1] have shown that in 418
208 patients angiographically diagnosed with AMI, the sensitivity of ST-segment elevation in
209 ECG was 85% if LAD and RCA were occluded, and 46% for LCx occlusion. In our study,
210 only 13/26 (50%) patients had a culprit occlusion in either the LAD or RCA.

211

212 Given the high incidence of acute coronary syndromes (ACS) in patients with OHCA and the
213 limitations of ECG-based diagnosis, current guidelines recommend considering immediate
214 coronary angiography in all patients post-resuscitation [7]. In clinical practice, ST-segment
215 elevation is still used as a selection criterion for coronary angiography in patients with OHCA
216 [16]. Dumas *et al* [7] showed the predictive value of ST-segment elevation for coronary

217 artery occlusion in the setting of OHCA to be poor with positive and negative predictive
218 values of 96% and 42% respectively. Selection of survivors of OHCA for coronary
219 angiogram based on the presence or absence of ST-segment elevation on ECG is therefore
220 difficult. Such a strategy would lead to neglecting the existence of acute coronary occlusion
221 in patients without ST-segment elevation on 12-lead ECG which should be treated with early
222 reperfusion. Thus, BSPM can facilitate earlier pre-hospital triage to emergent
223 revascularisation and transfer to a PPCI capable facility due to its improved sensitivity for
224 acute coronary occlusion diagnosis in these patients.

225

226 Our observations are limited by the nonrandomised and observational design of our study,
227 which contained no control group. Furthermore, only patients attended pre-hospital by a
228 physician-led MCCU and undergoing BSPM post-ROSC and who survived to coronary
229 angiography were included. Thus, only patients suffering VF and surviving to cardiac
230 catheterisation were analysed. Therefore, future studies are needed to investigate whether
231 pre-hospital BSPM in all OHCA patients has sustained diagnostic value and improves
232 survival in this patient group.

233

234 **Conclusion**

235 In patients successfully resuscitated from OHCA and a presenting rhythm of VF, pre-
236 hospital BSPM post-ROSC identified acute coronary occlusion with better sensitivity than
237 the 12 lead ECG. Additional studies are required to validate our findings.

238

239 **Conflict of interest statement**

240 No conflicts of interest to disclose

241

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290
291

292 Figure legends:

293

294 Figure 1. Overview of methodology to obtain study patients.

295 BSPM = body surface potential map; MCCU = mobile coronary care unit; OHCA = out-of-
296 hospital cardiac arrest; ROSC = return of spontaneous circulation; VF = ventricular
297 fibrillation

298

299 Figure 2. Twelve-lead ECG, BSPM and coronary angiogram in a patient post-ROSC: (A)

300 Twelve lead ECG showing 0.05mV ST-segment depression in leads V3 – V5 and T-wave

301 inversion in lead III and leads V1 – V4; (B) ST0 Isopotential BSPM showing (i) anterior

302 territory minima (blue) [-1.68mm] and (ii) right ventricular and posterior maxima (red)

303 [1.07mm] indicating right ventricular and posterior infarction; and (C) coronary angiogram

304 showing culprit occlusion of the proximal LCx with 60-70% stenoses in both the distal LMS

305 and proximal LAD. Cardiac troponin-T measured 12hrs post-ROSC was 4.35µg/L.

306

307 BSPM = body surface potential map; ECG = electrocardiogram; LAD = left anterior

308 descending artery; LCx = left circumflex artery; LMS = left main stem; ROSC = return of

309 spontaneous circulation

310

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	All Patients (n=35)
Age (yrs)	69 ± 13
Male gender (n [%])	21 (60)
BMI (kg m ⁻²)	27 ± 4
Risk Factors (n [%]):	
Hypertension	30 (86)
Hyperlipidaemia	31 (89)
Current smoker	24 (69)
Diabetes mellitus	20 (57)
Family history of CAD	21 (60)
Past Medical History (n [%]):	
Prior MI	16 (46)
Prior angina	20 (57)
Prior PCI	10 (29)
Prior CABG	0
GFR (ml/min)	48 ± 10
ECG rhythm post-ROSC (n[%]):	
Sinus rhythm	31 (89)
Atrial fibrillation / flutter	4 (11)
Haemodynamics post-ROSC:	
Heart rate (bpm)	83 ± 16
Systolic blood pressure (mmHg)	112 ± 28
Diastolic blood pressure (mmHg)	68 ± 23
Triple vessel coronary artery disease (n[%])	10 (29)
Time from (median [IQR]):	
OHCA to MCCU arrival (mins)	12 (7, 16)
OHCA to ROSC (mins)	22 (12, 31)
ROSC to BSPM / ECG (mins)	4 (2, 7)
ROSC to coronary angiogram (mins)	74 (50, 126)

Table 1. Demographics and risk factors for coronary artery disease in all patients (n=35)

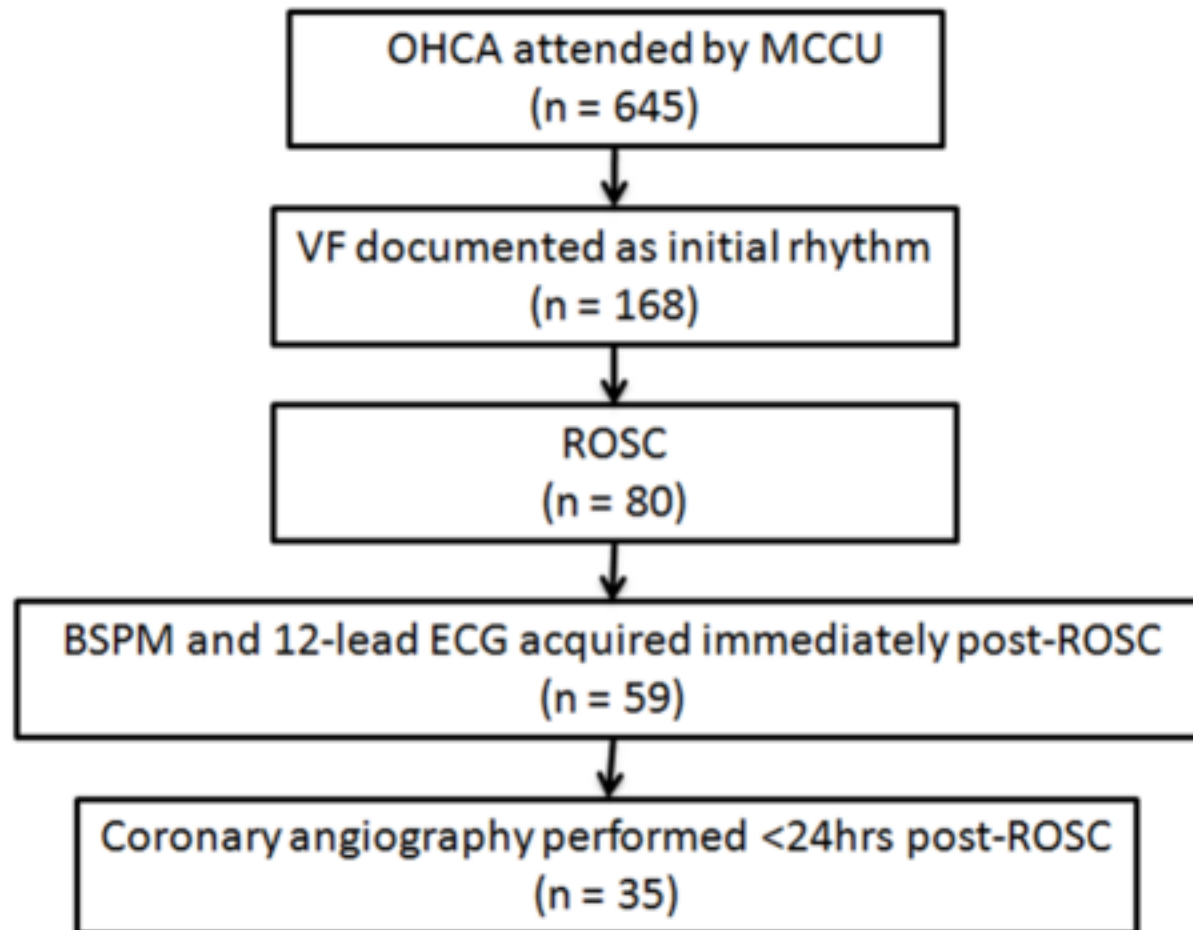
Results are expressed as number (percentage), mean ± standard deviation or median (interquartile range). BSPM = body surface potential map; BMI = body mass index; CABG = coronary artery bypass grafting; CAD = coronary artery disease; MCCU = mobile coronary care unit; OHCA = out-of-hospital cardiac arrest; PCI = percutaneous coronary intervention; ROSC = return of spontaneous circulation.

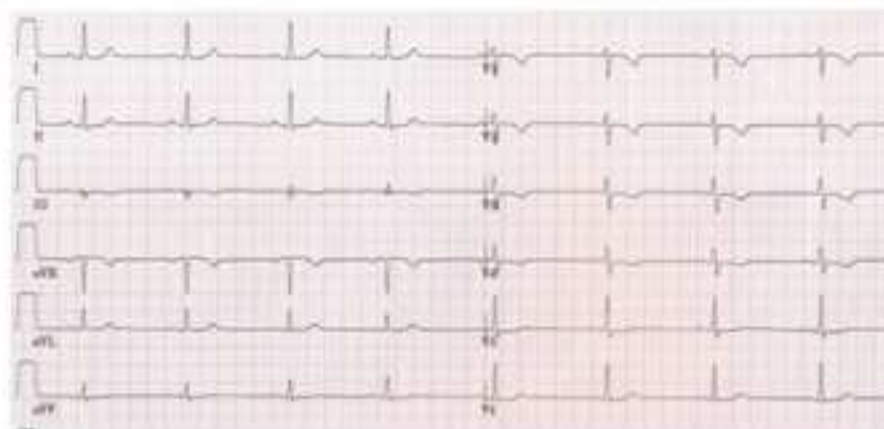
	n (%)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	c-statistic (AUC)
12-lead ECG:						
1. STEMI (<i>Minnesota 9-2 criteria</i>)	5 (14)	19	100	100	30	0.60
2. STD $\geq 0.05\text{mV}$ in ≥ 2 CL	7 (20)	23	89	86	29	0.56
3. TWI $\geq 0.1\text{mV}$ in ≥ 2 CL	4 (11)	12	89	75	26	0.51
4. STEMI (<i>Minnesota 9-2 criteria</i>) <i>or</i> STD $\geq 0.05\text{mV}$ in ≥ 2 CL	12 (34)	46	100	100	39	0.73
5. LBBB	5 (14)	15	89	80	27	0.52
6. RBBB	3 (9)	8	89	67	25	0.49
7. Non-specific QRS widening	2 (6)	8	100	100	27	0.54
8. LBBB <i>or</i> RBBB <i>or</i> non-specific QRS widening	10 (29)	31	78	80	28	0.55
BSPM:						
1. ST0 Isopotential STE (Cardiologist) *	23 (66)	88	100	100	75	0.94

Table 2. Accuracy of post-ROSC 12-lead ECG and BSPM in the diagnosis of acute coronary occlusion at angiography.

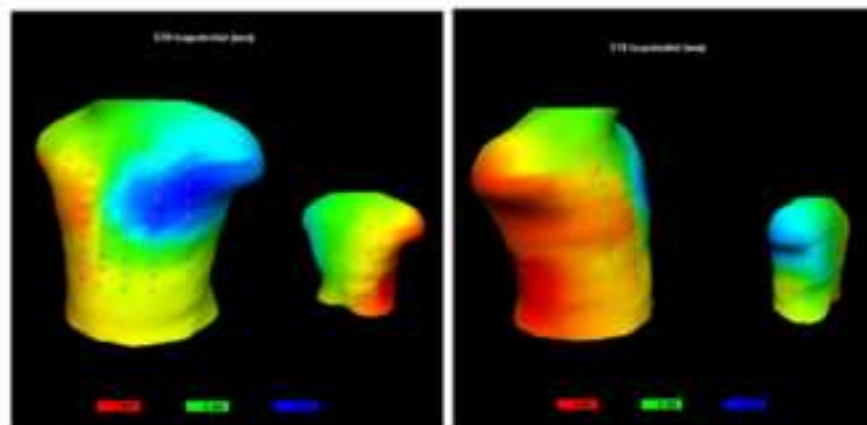
* anterior territory $\geq 0.2\text{mV}$ ST-segment elevation; lateral/inferior/high right anterior/right ventricular territories $\geq 0.1\text{mV}$ ST-segment elevation; posterior territory $\geq 0.05\text{mV}$ ST-segment elevation

AUC = area under curve; CL = contiguous leads; LBBB = left bundle branch block; RBBB = right bundle branch block; STD = ST-segment depression; STE = ST-segment elevation; STEMI = ST-segment elevation myocardial infarction; TWI = T-wave inversion



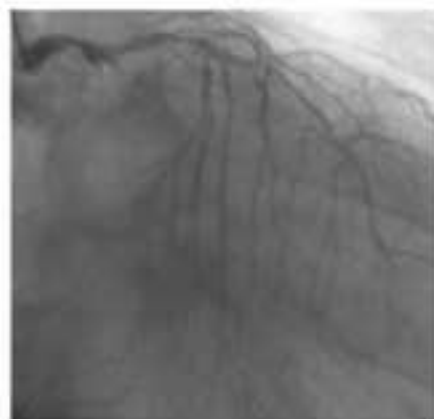


A



B (i)

(ii)



C