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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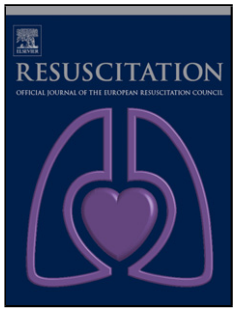

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Pre-hospital Body Surface Potential Mapping improves early diagnosis of acute coronary artery occlusion in patients with ventricular fibrillation and cardiac arrest.

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Abstract

Aims

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

Methods and Results

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

Conclusion

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

Introduction

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

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

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

Methods

Study Population

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

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

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

BSPM and 12-lead ECG analysis

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

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

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

Twelve-lead ECG abnormalities recorded were ST-segment elevation (STE), ST-segment depression (STD), T-wave inversion (TWI), left (LBBB) and right (RBBB) bundle branch block and non-specific QRS widening. ST segment shifts were measured at the J-point for STE and 80ms after the J-point for STD using the preceding TP segment as a baseline. STE consistent with AMI (STEMI) was defined using the Minnesota 9-2 criteria [13] as ≥0.1mV STE in one or more of leads I, II, III, aVL, aVF, V_5, V_6 or ≥0.2mV STE in one or more of leads $V_1 - V_4$. STE in lead aVR was defined as ≥0.05mV. STD was considered significant if ≥0.05mV in ≥2 contiguous leads. LBBB was defined as QRS duration ≥120ms with QS or rS pattern in V_1 and broad R waves in lead I, V_5 and V_6 [1]. RBBB was defined as QRS duration ≥120ms with rSR' complex in V_1 and V_2 and S wave in lead I and V_5 or V_6 [1]. Non-specific QRS widening was defined as QRS duration ≥120ms without LBBB or RBBB morphology. Twelve-lead ECG analysis was verified on arrival to hospital by a cardiologist blinded to all other clinical details.

Coronary Angiography

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

Statistical Analysis

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

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

Results

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

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

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

Discussion

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

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

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

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

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

Conclusion

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

Conflict of interest statement

No conflicts of interest to disclose

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Figure legends:

Figure 1. Overview of methodology to obtain study patients.

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

Figure 2. Twelve-lead ECG, BSPM and coronary angiogram in a patient post-ROSC: (A) Twelve lead ECG showing 0.05mV ST-segment depression in leads V3 – V5 and T-wave inversion in lead III and leads V1 – V4; (B) ST0 Isopotential BSPM showing (i) anterior territory minima (blue) [-1.68mm] and (ii) right ventricular and posterior maxima (red) [1.07mm] indicating right ventricular and posterior infarction; and (C) coronary angiogram showing culprit occlusion of the proximal LCx with 60-70% stenoses in both the distal LMS and proximal LAD. Cardiac troponin-T measured 12hrs post-ROSC was 4.35µg/L.

BSPM = body surface potential map; ECG = electrocardiogram; LAD = left anterior descending artery; LCx = left circumflex artery; LMS = left main stem; ROSC = return of spontaneous circulation

	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 *(Minnesota 9-2 criteria)*	5 (14)	19	100	100	30	0.60
2. STD ≥ 0.05mV in ≥2 CL	7 (20)	23	89	86	29	0.56
3. TWI ≥ 0.1mV in ≥2 CL	4 (11)	12	89	75	26	0.51
4. STEMI *(Minnesota 9-2 criteria)* ***or*** STD ≥ 0.05mV 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 ***or*** RBBB ***or*** 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 ≥0.2mV ST-segment elevation; lateral/inferior/high right anterior/right ventricular territories ≥0.1mV ST-segment elevation; posterior territory ≥0.05mV 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

Figure 1

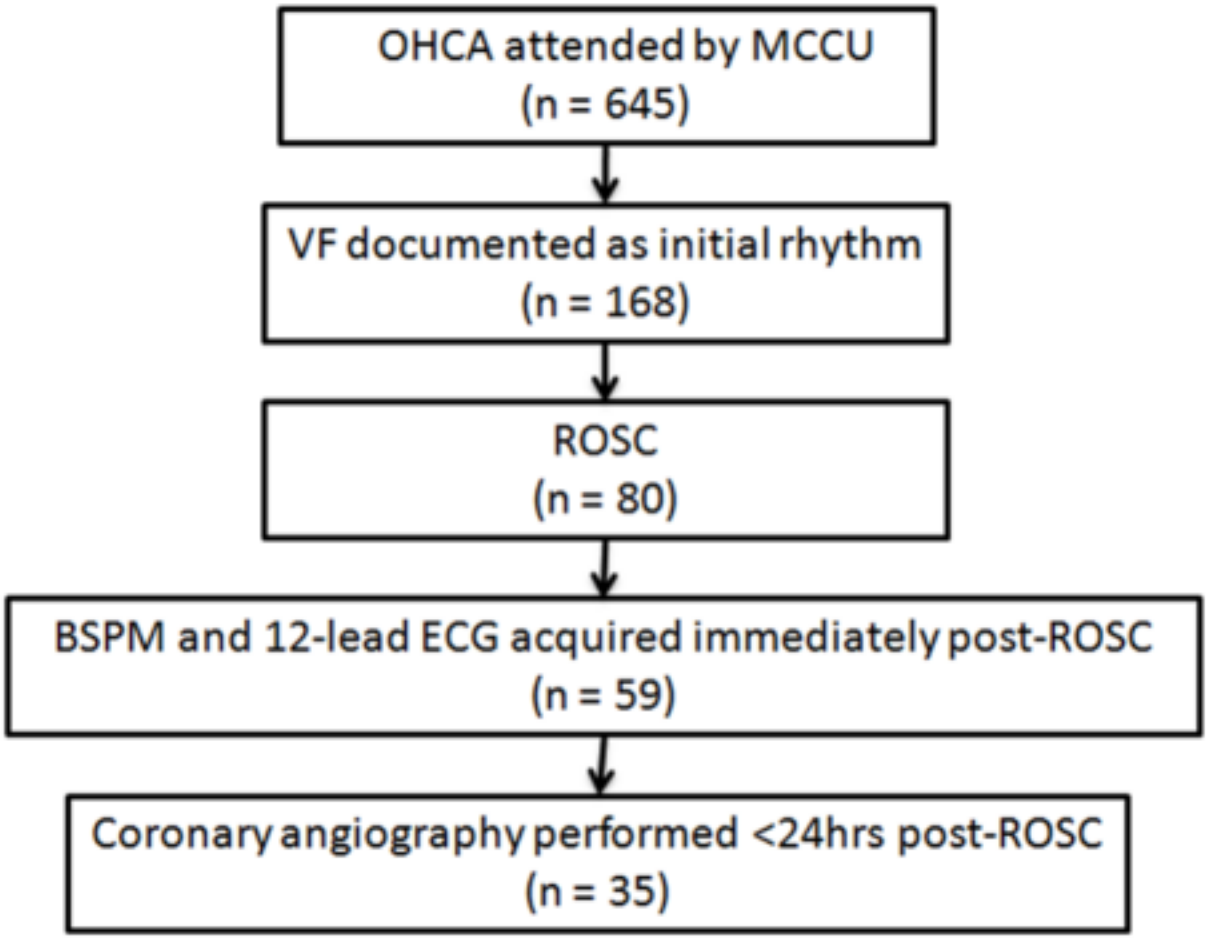
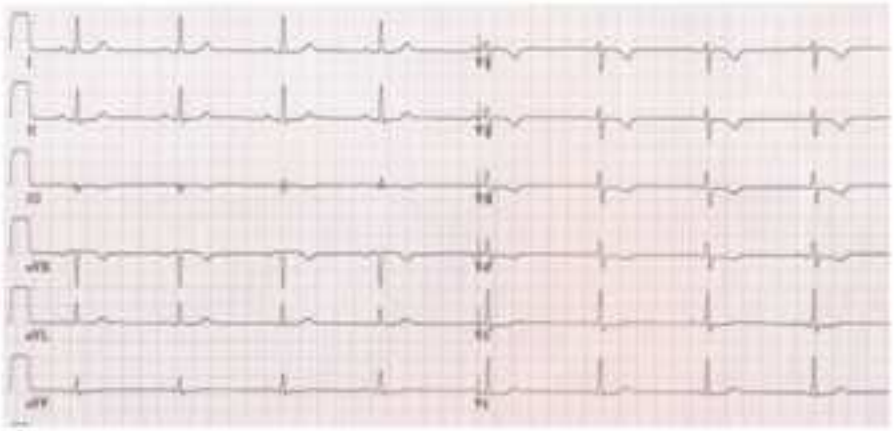

OHCA attended by MCCU
(n = 645)
VF documented as initial rhythm
(n = 168)
ROSC
(n = 80)
BSPM and 12-lead ECG acquired immediately post-ROSC
(n = 59)
Coronary angiography performed <24hrs post-ROSC
(n = 35)

Figure 2



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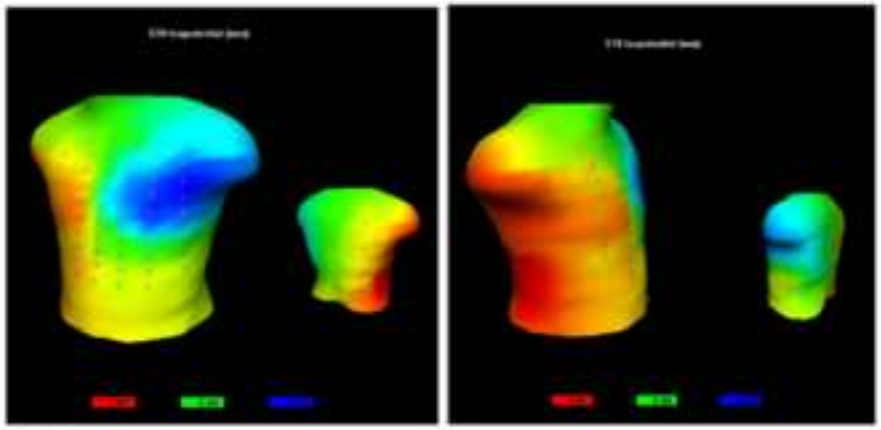

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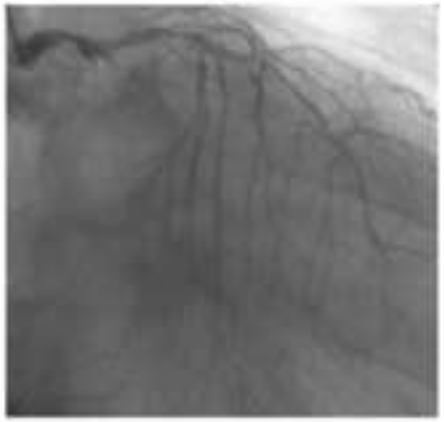

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