Body Surface Potential Mapping and Left Circumflex Occlusion: Unmasking the Hidden Acute Myocardial Infarction

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Treatment of patients with ST-segment–elevation myocardial infarction (STEMI) represents a significant success story. Over the past 20 years, mortality has decreased substantially, with most in-hospital deaths limited to patients who have either cardiogenic shock or cardiac arrest. However, this reduction in mortality is critically dependent on prompt treatment, which in turn is dependent on early diagnosis. In contrast, treatment of patients with non-STEMI is less time dependent. Unfortunately, a significant minority of patients who have non-STEMI actually have acute vessel occlusion without diagnostic ST-segment elevation on their ECG (25% of non-STEMI patients in a meta-analysis[1]), leading to both delayed diagnosis and treatment,[1–4] with resultant worse outcomes.[1–3]

The left circumflex artery (LCX) frequently supplies the posterior and lateral wall and, at times, parts of the inferior wall. Although the majority of patients who have LCX occlusion undergoing primary revascularization have diagnostic ST-segment elevation, a significant minority do not.[4,5] Recognition of other ECG findings may provide a clue to diagnosis. When the posterior wall is involved, anterior ST-segment depression can occur, with confirmation obtained by the presence of posterior ST-segment elevation seen after application of ECG leads to the back.[6,7] In other cases, although the ECG does not meet strict criteria for STEMI, an experienced clinician may identify subtle ST-segment elevation suggestive of acute myocardial infarction (AMI).[8,9]

Occluded culprit vessels in the absence of ST-segment elevation in AMI patients are not uncommon.[1–3] Despite the lack ST-segment elevation, outcomes are not benign. In a meta-analysis that included almost 41 000 patients, the complication rate was increased by 41%, with 67% higher mortality.[1] Earlier diagnosis and revascularization would likely significantly reduce this event rate.

It is with this background the authors in this issue of the *Journal of the American Heart Association (JAHA)* report on the ability of a multilead extended ECG technique, body surface potential mapping (BSPM), for identifying patients who have unsuspected LCX infarction.[10] This ECG technique uses multiple leads applied to the patient's anterior and posterior chest (in the current study, 80 leads: 64 anterior, 16 posterior), enabling more complete visualization of cardiac electrical activity. The development of BSPM is an extension of the rationale of supplemental right-sided and posterior leads.[6,7] The output from BSPM is displayed in multiple formats, including a 12-lead ECG, an 80-lead ECG, and on-color contour maps that represent various deflections of the ECG. Since its initial development, modifications have improved its utility and ease of use, including modifications for body habitus, improved display algorithms with the body map applied to a torso image,[11] and leads embedded on a vest that are applied to a patient's chest and back, avoiding the necessity of individually placing each electrode. Currently used devices, when used routinely, take only a few extra minutes compared with the routine ECG; in the author's experience, it is ≈3 to 4 minutes. Over the past 10 to 15 years, BSPM has been demonstrated to increase the sensitivity for identifying patients with AMI in both single and multicenter studies.[12–16]

In the current study,[10] the authors assessed the use of 80-lead BSPM for the difficult-to-identify myocardial infarction (MI) population, those with LCX occlusion. Over a 10-year period, 314 patients were included (1) who underwent coronary angiography and had a culprit LCX stenosis, (2) who had an initial ECG that did not demonstrate diagnostic ST-segment elevation, (3) who were diagnosed with AMI, and (4) who underwent 80-lead BSPM. Appropriate exclusions

were applied: patients who had bundle-branch block or paced rhythm (which would preclude ECG interpretation), prior bypass surgery (difficult to determine the culprit artery), or >15-minute delay between the standard 12-lead ECG and BSPM (ensuring the 2 ECGs were temporally related). Likely a reflection of the author's extensive experience with this device, no patients were excluded for having a poor-quality BSPM.

Consistent with prior studies,[12–16] the sensitivity of BSPM was significantly higher than the standard 12-lead ECG for diagnosing MI. As one would suspect, ST-segment elevation by BSPM not apparent on the 12-lead ECG was detected most commonly posteriorly, in 48% of patients. Sensitivity and positive predictive value were very high, at 91% and 93%, respectively, reflecting in part the enriched population in which 80% of patients were diagnosed with MI. Importantly, predictive accuracy was unchanged after excluding the subgroup of patients who had significant anterior ST-segment depression or posterior ST-segment elevation.

As with any study, there were some limitations. It is unclear why patients with left ventricular hypertrophy were excluded, whereas those who had prior MI were not. Specificity was lower than one might expect at 72%, given the high proportion of patients who had MI. The authors did not comment on potential reasons for the causes for false-positive 80-lead BSPMs; more information on this would have been helpful. Although standard criteria were used for identifying STEMI on the initial 12-lead ECG, it is unclear how many patients could have been identified who had high suspicion based on ST-segment changes that did not quite meet criteria for STEMI, although results were unchanged after excluding patients who had significant anterior ST-segment depression or posterior ST-segment elevation.

Perhaps the largest question is whether performing BSPM in all patients with potential acute coronary syndrome is worth the added cost and time. Given the large number of patients who present with possible acute coronary syndrome ($\approx$10% of emergency department patients) and the relatively low frequency of those who have AMI (usually <10% of possible ACS patients, compared with the 80% rate of MI seen in the current study), application to all patients would be logistically difficult and would increase cost. In the largest study performed to date (n=1830), BSPM identified only an additional 1.3% AMI patients.[16] Although the additional time it takes to perform the BSPM is not dramatic (in one study it could be performed within 10 minutes),[17] the time required has greater implications when applied to the thousands of emergency department patients who have possible acute coronary syndrome.

Despite these limitations, the current study addresses a blind spot in the evaluation of patients with AMI. Although other techniques, such as emergent ultrasound, now ubiquitous in emergency departments, may provide similar information, interpretation of wall motion is one of the most difficult things to perform. In my experience, identifying wall motion abnormalities requires extensive skill and can be difficult, particularly in those with suboptimal windows (not an uncommon situation in the emergency department setting), even in those patients with STEMI.

A potential solution is targeting BSPM to higher risk patients, such as those who have anterior ST-segment depression, the presence of minor ECG changes consistent with STEMI,[8,9] posterior or inferior wall motion abnormalities on ultrasound, or positive troponin. Although the last strategy could result in delayed diagnosis in patients with initially negative troponin who present early after infarction onset,[18] contemporary troponin-sampling strategies can reduce the time to diagnosis within 1 to 3 hours of presentation, a strategy that would still be significantly faster compared with those seen in prior studies.[1–4,16]

In conclusion, the current study demonstrates the utility of BSPM to identify AMI in an AMI population that is frequently difficult to diagnose: those with LCX occlusions. This study and prior ones[12–16] have clearly demonstrated that this technique significantly increases the diagnosis of AMI. Future studies should target end points other than AMI diagnosis, such as time to diagnosis and treatment, and outcomes, as well as overall cost.

Disclosures

None.

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