Useful addition to acute myocardial infarction diagnosis in patients with left bundle branch block: an algorithm using electrocardiographic and biomarker analysis

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Patients presenting with acute coronary occlusion, manifested by ST segment elevation myocardial infarction (STEMI), benefit from prompt reperfusion therapy, including either fibrinolysis or primary percutaneous coronary intervention (PCI).[1] When considering reperfusion therapy with either PCI or fibrinolysis, delays are associated with a markedly higher rate of short-term and long-term mortality[2]; furthermore, increased time to initiation of reperfusion therapy from symptom onset is associated with increased risk of poor cardiac function with heart failure and other cardiovascular comorbidities.[3] In patients with left bundle branch block (LBBB), the diagnosis of acute myocardial infarction (AMI) resulting from acute coronary occlusion is much more difficult; in fact, the LBBB ECG pattern is considered a confounder to the diagnosis of AMI. This diagnostic difficulty results from the altered depolarisation pattern encountered in LBBB…of course, altered depolarisation results in altered repolarisation, producing ST segment/T wave configurations which obscure the ECG findings of myocardial ischaemia and infarction.

The diagnostic challenge does not infrequently result in delays in the initiation of both PCI and fibrinolysis with associated less optimal outcomes in terms of both mortality and subsequent development of heart failure.[4] While the LBBB pattern frequently confounds the ECG diagnosis of AMI, these patients are usually very ill on presentation, experiencing cardiogenic shock, acute heart failure and malignant dysrhythmias; thus, the clinician must consider AMI as a potential explanation for the severity of presentation in these ill patients with LBBB. Improved early recognition of AMI in this challenging population may improve outcomes.

The analysis in this study by Nestelberger *et al*[5] is based on three large, prospective, multicentre studies with a central adjudication of AMI applying the universal definition of AMI. Nestelberger *et al*[5] prospectively evaluated the use of ECG criteria combined with high-sensitivity troponin determinations for the diagnosis of AMI in patients presenting to the emergency department (ED) with LBBB; specifically, they used the previously validated Sgarbossa criteria, both the original[6] and modified versions,[7] combined with a high-sensitivity cardiac troponin assay (hs-cTn). In this study, the authors evaluated 8830 patients presenting to the ED with chest pain, of whom 247 (2.8%) demonstrated an LBBB pattern on the ECG. Of these individuals, 75 (30%) received a final diagnosis of AMI. Receiver operating characteristic curves were constructed for values of hs-cTn and cut-off values selected for a desired positive predictive value of ≥80% (hs-cTnI ≥45 ng/L or hs-cTnI ≥52 ng/L). They similarly selected values for Δhs-cTn levels with a PPV of ≥80% (Δhs-cTnT >3 ng/L at 0 or 2 hours, or Δhs-cTnI >4 ng/L at 1 hour).

The authors[5] noted no difference in AMI occurrence rates between patients with new (or presumably new) LBBB as compared with pre-existing LBBB. They found that 30% of study patients had AMI as the final hospital diagnosis; AMI occurrence was noted as follows considering the chronicity of the LBBB: pre-existing LBBB with 29% AMI versus new (or presumably new) LBBB with 35% AMI. In all patients with LBBB, the authors report that the diagnostic ECG criteria (Sgarbossa and modified Sgarbossa) had a relatively low sensitivity (3% and 12%, respectively) but high specificity (99% and 97%, respectively) for AMI. Using a combination of ECG diagnostic criteria (ie, modified Sgarbossa criteria) and troponin analysis (the described hs-cTn cut-off ranges), the authors report that a subsequent diagnostic algorithm was highly accurate in identification of AMI. In their algorithm, the authors also propose a time frame in initial troponin draw of >3 hours from symptom onset as an alternative to obtaining 1-hour or 2-hour delta troponin levels. This is congruent with previous expert recommendations that patients presenting within 2 hours of symptom onset may benefit from evaluation of serial troponin levels.[8] The authors concluded that most patients presenting with suspected AMI and LBBB will be found to have alternative diagnoses. In addition, the combination of ECG criteria with high-sensitivity troponin testing at 0 and 1 hour or at 0 and 2 hours allowed for both an early and an accurate diagnosis of AMI in these diagnostically complex, potentially very ill patients with LBBB.

The *Heart* paper by Nestelberger *et al*[5] has considered several important areas of diagnostic information in patients with chest pain with LBBB suspected of AMI. First, they have reconfirmed that AMI is not the most frequent diagnosis in patients with chest pain with LBBB. Up to 2010, new or presumably new LBBB pattern in a patient clinically suspected for AMI was considered an STEMI-equivalent ECG presentation.[9] Prudent clinical practice as well as numerous investigations in this area, however, found that AMI was not the most common diagnosis in these patients with chest pain with new LBBB. In fact, Kontos *et al*[10] and Jain *et al*[11] noted AMI occurred in 29% and 33%, respectively, of these patients. Thus, in 2013, the American College of Cardiology and the American Heart Association jointly noted that '*…new or presumably new LBBB at presentation occurs infrequently…and should not be considered diagnostic of acute myocardial infarction in isolation.*'[1] Nestelberger *et al*[5] report that 30% of patients with LBBB had AMI as the final hospital diagnosis, a rate very comparable with the existing literature on the topic.

Second and importantly, they have demonstrated yet again the accurate nature of the original and modified Sgarbossa criteria in the ECG diagnosis of AMI in the patient with LBBB. Smith *et al*,[7] in the development report of the modified Sgarbossa criteria, noted both high sensitivity (91%) and specificity (90%) for this ECG decision rule in the diagnosis of AMI in LBBB. In Nestelberger *et al*,[5] a very high specificity (97%) of the modified Sgarbossa criteria was noted, somewhat balanced by a very low sensitivity (12%). Yet, in this instance, a high specificity

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allows the clinician to rule in AMI with confidence in the setting of a clinically suspected AMI.

Lastly and of most significance, the authors have added high-sensitivity troponin determinations to the above ECG criteria, enabling an early and accurate diagnosis of AMI in these diagnostically challenging patients. This combination of diagnostic tools is perhaps the most significant and valuable finding of this study. With the removal of LBBB as an STEMI-equivalent pattern, the rate of false-positive cardiac catheterisation laboratory activation dropped considerably. Yet clinicians remained concerned that certain patients with LBBB did, in fact, present with AMI and could experience either a delayed or entirely missed diagnosis of AMI as a result of this altered initial approach. The missed diagnosis of AMI in these patients with LBBB would then result in an increased rate of adverse outcome, including death, postmyocardial infarction compromised left ventricular function, significant long-term heart failure and arrhythmic complications.

In response to this concern, Cai *et al*[12] published an algorithm considering cardiovascular stability, the original and modified Sgarbossa criteria, serial observations with troponin and ECGs, and echocardiography. In this approach when evaluating the patient with LBBB suspected of AMI, the clinician is first queried regarding haemodynamic instability or acute heart failure; if either is present, reperfusion therapy with fibrinolysis or PCI is recommended. If the patient is stable from haemodynamic and respiratory perspectives, the original and modified Sgarbossa criteria are then considered; a Sgarbossa score ≥ 3 or modified Sgarbossa ST:S ratio ≤ -0.25 prompts the clinician to consider acute reperfusion therapy. Lacking instability and positive Sgarbossa ECG criteria, the clinician is then urged to consider serial troponin testing with repeat ECGs with echocardiography; if positive, then coronary angiography is recommended. The Cai *et al* algorithm is a very rational approach to this challenging clinical situation.[12] Its major limitation, however, is the need for a prolonged period of serial troponin testing in haemodynamically stable patients with Sgarbossa-negative ECGs; such testing could require as long as 4–8 additional hours of observation prior to establishing an AMI diagnosis. Such delays in diagnosis and initiation of reperfusion therapy in the patient with LBBB AMI can result in a higher risk of death and an increased rate of cardiovascular adverse events.[4]

The study by Nestelberger *et al*[5] builds on the Cai *et al* approach,[12] allowing for a potentially earlier and perhaps a more accurate diagnosis of AMI. The Nestelberger *et al* algorithm[5] is a three-step process, using initial ECG Sgarbossa criteria (step 1) and two subsequent high-sensitivity troponin determinations (steps 2 and 3). At initial presentation, with either positive original and/or modified Sgarbossa ECG criteria in a patient suspected of AMI, these highly specific ECG findings support the diagnosis of AMI and the need for immediate coronary angiography, presumably with consideration of reperfusion therapy. If the initial ECG is 'Sgarbossa negative', then the use of high-sensitivity troponin testing at presentation provides the second opportunity for AMI diagnosis and immediate or early coronary angiography, again presumably with consideration of reperfusion therapy. If the initial high-sensitivity troponin is negative for AMI, a repeat troponin determination at either a 1-hour or 2-hour interval is made; this second high-sensitivity troponin determination, if positive in either an absolute or delta troponin manner, supports a diagnosis of AMI and immediate or early coronary angiography, once again presumably with consideration of reperfusion therapy. The diagnostic accuracy of this approach is certainly impressive. The ability to arrive at the diagnosis of AMI rapidly, quite early in the clinical course, is also impressive.

The results reported by Nestelberger *et al*[5] are encouraging and further our diagnostic abilities in these complex, potentially ill patients. It should be stressed, however, that these data are limited to patients with suspected AMI due to acute coronary occlusion; at the present time, the algorithm cannot be extrapolated to patients presenting with other possible aetiologies for hs-cTn elevation (eg, troponin elevations due to demand ischaemia, sepsis or renal insufficiency)—this issue is the major limitation of this algorithm. Additionally, in patients with LBBB but without ECG changes diagnostic of AMI, negative hs-cTn determinations should not be construed to preclude the need for further evaluation or treatment of alternative, non-AMI causes of chest pain (eg, unstable angina, pulmonary embolism and so on). Despite these shortcomings, the authors have reaffirmed the high specificity of the original and modified Sgarbossa criteria for AMI; they have also proposed a reasonable, stepwise algorithm using both ECG and serum marker data for the evaluation of ED patients with LBBB and suspected AMI. This approach allows for both an early and an accurate diagnosis of AMI in these diagnostically challenging patients and increases the opportunity for benefit from intervention and reduces the considerable risk of adverse outcome from delays in appropriate management.

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REFERENCES

1 O'Gara PT, Kushner FG, Ascheim DD, *et al*. ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology* 2013;2013:e78–140.
2 Foo CY, Bonsu KO, Nallamothu BK, *et al*. Coronary intervention door-to-balloon time and outcomes in ST-elevation myocardial infarction: a meta-analysis. *Heart* 2018;104:1362–9.
3 Shiomi H, Nakagawa Y, Morimoto T, *et al*. Association of onset to balloon and door to balloon time with long term clinical outcome in patients with ST elevation acute myocardial infarction having primary percutaneous coronary intervention: observational study. *BMJ* 2012;344:e3257.
4 Al Rajoub B, Noureddine S, El Chami S, *et al*. The prognostic value of a new left bundle branch block in patients with acute myocardial infarction: A systematic review and meta-analysis. *Heart Lung* 2017;46:85–91.
5 Nestelberger T, Cullen L, Lindahl B, *et al*. APACE, ADAPT and TRAPID-AMI Investigators. Diagnosis of acute myocardial infarction in the presence of left bundle branch block. *Heart* 2019:heartjnl-2018-314673.
6 Sgarbossa EB, Pinski SL, Barbagelata A, *et al*. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. *N Engl J Med Overseas Ed* 1996;334:481–7.
7 Smith SW, Dodd KW, Henry TD, *et al*. Diagnosis of ST-elevation myocardial infarction in the presence of left bundle branch block with the ST-elevation to S-wave

ratio in a modified Sgarbossa rule. *Ann Emerg Med* 2012;60:766–76.
8 Roffi M, Patrono C, Collet J-P, *et al*. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent st-segment elevationtask force for the management of acute coronary syndromes in patients presenting without persistent ST-Segment Elevation of the European Society of Cardiology (ESC). *Eur Heart J* 2015;2016:267–315.
9 O'Connor RE, Brady W, Brooks SC, *et al*. Part 10: acute coronary syndromes: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation* 2010;122:S787–S817.
10 Kontos MC, Aziz HA, Chau VQ, *et al*. Outcomes in patients with chronicity of left bundle-branch block with possible acute myocardial infarction. *Am Heart J* 2011;161:698–704.
11 Jain S, Ting HT, Bell M, *et al*. Utility of left bundle branch block as a diagnostic criterion for acute myocardial infarction. *Am J Cardiol* 2011;107:1111–6.
12 Cai Q, Mehta N, Sgarbossa EB, *et al*. The left bundle-branch block puzzle in the 2013 ST-elevation myocardial infarction guideline: from falsely declaring emergency to denying reperfusion in a high-risk population. Are the Sgarbossa Criteria ready for prime time? *Am Heart J* 2013;166:409–13.