The importance of right bundle branch block in myocardial infarction

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Bundle branch blocks in patients presenting with myocardial infarction have been shown to indicate an increased risk of both short- and long-term mortality after myocardial infarction. While left bundle branch block is considered to be an equivalent of ST-segment elevation in patients presenting with chest pain, right bundle branch block is thought not to interfere with ECG interpretation in myocardial infarction. The article summarizes evidence for right bundle branch block as a risk factor for a poorer outcome after myocardial infarction and discusses the interpretation of ECG changes in myocardial infarction patients with right bundle branch block.
Key words: Myocardial infarction – ECG – Bundle branch block – Reperfusion – Risk factor

Lorenzová A, Widimský P. **Význam blokády pravého Tawarova raménka u infarktu myokardu.** *Cor Vasa 2009;51(9):578–583.*

Bylo prokázáno, že blokády Tawarových ramének u pacientů s infarktem myokardu signalizují vyšší riziko jak krátkodobé, tak dlouhodobé mortality po infarktu myokardu. Zatímco blokáda levého Tawarova raménka je považována za ekvivalent elevací úseků ST u pacientů s bolestí na hrudi, u pravého Tawarova raménka se předpokládá, že neinterferuje s hodnocením EKG u infarktu myokardu. Článek shrnuje údaje o blokádě pravého Tawarova raménka jako rizikového faktoru pro horší prognózu po infarktu myokardu a interpretaci změn EKG u pacientů s infarktem myokardu a blokádou pravého Tawarova raménka.
Klíčová slova: Infarkt myokardu – EKG – Blok Tawarova raménka – Reperfuze – Rizikový faktor

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Abbreviations

AMI – acute myocardial infarction
BBB – bundle branch block
ESC – European Society of Cardiology
LAH – left anterior hemiblock
LBBB – left bundle branch block
LMCA – left main coronary artery
LPH – left posterior hemiblock
RBBB – right bundle branch block
STEMI – ST-elevation myocardial infarction

Introduction

Both left and right bundle branch blocks (BBB) in patients presenting with myocardial infarction indicate an increased risk of in-hospital mortality, as has been shown in a recent clinical trial.[1] Yet, according to the myocardial infarction management guidelines of the European Society of Cardiology,[2] only left bundle branch block (LBBB) is considered to be an equivalent of ST-segment elevation with an indication for urgent reperfusion. Attitudes towards the issue of BBBs in acute myocardial infarction have evolved since the pre-reperfusion era; thrombolysis and later percutaneous coronary interventions brought us not only better and effective care for patients, but also many new and challenging questions.

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Guidelines

Myocardial infarction treatment guidelines of the American Heart Association published in 1996 pointed out that "symptoms consistent with acute myocardial infarction and LBBB should be managed like ST-segment elevation", without further reasoning or references.[3] In comparison, the guidelines of the European Society of Cardiology issued in the same year stated that in patients presenting with ST-segment elevation or any BBB, early reperfusion should be attempted.[4] These guidelines did not distinguish between the occurrence of left or right BBBs in the presentation of acute myocardial infarction (AMI) and the recommendation for subsequent treatment was the same for both of the conditions. In most of these documents, only a few sentences were dedicated to the description of ECG changes, and no exact definitions were provided.

The European guidelines released in the year 2000 were the first that exactly defined which ECG changes are substantial for diagnosing myocardial infarction.[5] The magnitude of ST-segment changes to be considered significant was clearly described. The issue of BBBs was mentioned only marginally, stating that LBBB makes recognition of AMI difficult or impossible and that further research is needed to define the criteria for diagnosing AMI in the presence of BBB. Concerning RBBB, the document only stated that it does not interfere with the ability to diagnose Q waves.[5]

Interestingly, the guidelines of the American College of Emergency Physicians for management of patients presenting with suspected AMI or unstable angina, published in 2000,[6] concluded that reperfusion should be considered in all patients with a clinical presentation suggestive of myocardial infarction in the presence of any type of BBB. This statement was based on the results of the GISSI[7] and ISIS-2[8] trials where patients with any type of BBB without distinction were included. In these trials, thrombolysis was employed (at that time the widely prevalent method) as the primary method of reperfusion, with primary angioplasty being an option, and both with the same indication criteria.

As a basic document for consequent guidelines concerning management of acute myocardial infarction, European and American cardiologists prepared jointly in 2007 a consensus document "Universal definition of myocardial infarction".[9] A clear definition of ECG manifestation of acute and prior myocardial infarction by ST-T segment changes and Q waves was presented. With reference to the BBBs, this document carefully stated that diagnosis of myocardial infarction is difficult in the presence of LBBB and, in another sentence, a classification of fatal myocardial infarction in patients with ST-segment elevation or new LBBB was given. Regarding RBBB, the document noted that in this type of BBB with ST-segment elevation or Q waves, myocardial ischemia should be considered. It is clear that RBBB plus ST-segment elevation or RBBB plus Q waves in the clinical context of chest pain is diagnostic of AMI. However, the diagnostic value of new onset RBBB ± left anterior hemiblock (LAH) without ST-segment elevations or Q waves is much less clear *(Figure 1)*.

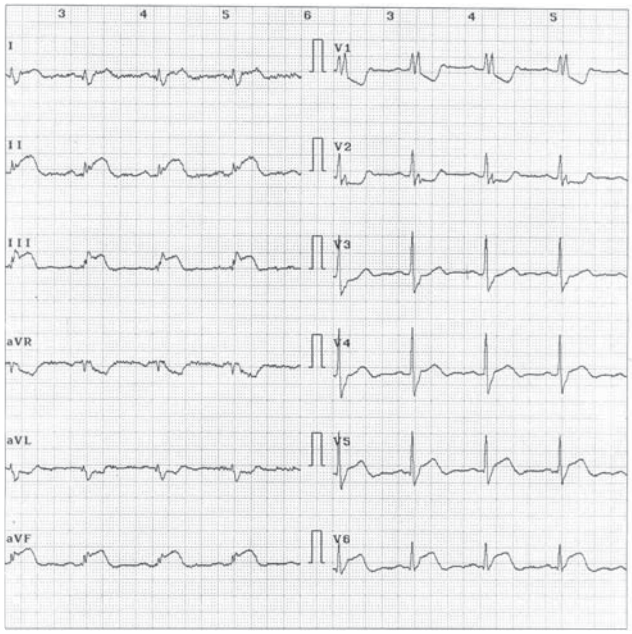

Figure 1 **RBBB with ST-segment elevations in a patient with total occlusion of circumflex artery**

The following ESC guidelines for management of ST-segment elevation myocardial infarction (STEMI) declared that ST-segment elevation and new LBBB were equal indications for urgent reperfusion therapy (similar to the AHA guidelines from 1996),[2] without mentioning how the situation should be assessed in the presence of RBBB.

There is no doubt today that new, or presumably new, LBBB in patients presenting with chest pain is the equivalent of ST-segment elevations requiring acute reperfusion. However, the situation is less clear with patients presenting in the emergency department with new RBBB and chest pain. What evidence do we currently have for RBBB, myocardial infarction and reperfusion? Are we going to equivocate between new LBBB and RBBB one day, or will we define exactly the difference in the diagnostic evaluation of ECG?

Evidence for RBBB

Pre-reperfusion era

The first clinical studies concerning RBBB and myocardial infarction were conducted in the early 1970s. These studies showed that patients with AMI complicated by RBBB had a poorer prognosis.[10] Most of these studies addressed the issue of placing temporal or permanent cardiac pacing in AMI patients,[11] as was the case, for example, in the study of Gould et al., reporting 77% in-hospital mortality in patients with AMI and RBBB.[12] Patients especialy with a bi-fascicular block [RBBB + LAH or left posterior hemiblock (LPH)] were at high risk of developing complete heart block. However, use of cardiac pacing was only partially helpful in improving the prognosis of these patients, who died mostly of heart failure or ventricular fibrillation. At autopsy, extensive necrosis of the left ventricular anterior wall and most of the interventricular septum was observed.

Overall, it was concluded that myocardial damage was so extensive that this was the factor determining the prognosis and not the conduction disorders. In all these articles, AMI with RBBB were diagnosed when the patient had ST-segment elevation or pathological Q waves present and RBBB was accompanying these changes.

Later studies have emphasized the high rate of pump failure as the main cause of death in these patients. In the study of Dubois et al.[13] where 1 013 consecutive AMI patients were included, patients with BBBs (both left and right) had more complications of myocardial infarction (like pericarditis, atrial fibrilation, ventricular fibrilation, atrioventricular block) and had higher Killip class on admission. Both in-hospital mortality (32% vs. 10%, respectively; $p < 0.001$) and 3-year post-hospital mortality (37% vs. 18%, respectively; $p < 0.001$) were much higher among patients with complete BBB. It was concluded that cardiogenic shock and progressive congestive heart failure were the main causes of death in patients with myocardial infarction complicated by BBBs.[13]

Thrombolysis

In another study, it was demonstrated that patients presenting with new RBBB at the time of AMI had a much poorer prognosis than those with documented old RBBB and in-hospital mortality was 38 % vs.14%, respectively; $p < 0.05$.[14] In this study by Ricou et al., only patients with anterior myocardial infarction were included. Myocardial infarction was diagnosed as the presence of Q waves or QS complex and elevation of serum creatine kinase. The highest mortality was observed in patients with RBBB and associated left ventricular failure, and new RBBB was also found to be an independent marker of increased in-hospital cardiac mortality. Overall, it was concluded that these are very high-risk patients and further diagnostic testing such as coronary angiography should be performed.

Lately, more emphasis has focused on ST-segment changes rather than Q waves and reperfusion procedures were urgently performed at the time of hospital admission.

In their analysis performed on the cohorts of the GUSTO 1 and TAMI 9 trials, Newby et al. examined whether thrombolytic therapy alters the incidence of BBBs and clinical outcome in patients with AMI.[15] The overall incidence of BBBs in the combined cohort was 23.6%, and left anterior descending artery infarcts accounted for most of BBBs – 54% (information regarding the occurrence of BBB was correlated to angiographic data). RBBB was the more common type of BBB, occurring in 13% vs. 7% of LBBB. It was concluded that thrombolytic therapy reduced the overall mortality rate associated with persistent BBB. Despite this reduction, persistent BBB remained predictive of higher mortality in patients with myocardial infarction.

Wong et al. studied the prognostic diversity between different types of BBBs in thrombolyzed patients with AMI in the cohort of the HERO-2 trial.[16] The main keynote of this study was the fact that the existing risk score algorithms, like those used in the GUSTO[17] and TIMI[18] study, did not take RBBB into account. In the risk assessing algorithm of the GUSTO study, only the location of the myocardial infarction was included, but not the exact description of ECG changes (e.g., type of BBB).[17] For the TIMI risk score, selection of included variables was based on their relative prognostic contribution according to the full logistic regression model.[18] Ten variables with the most predictive information were included (this model covered 97% of overall prognostic information). However, RBBB could not be included in the TIMI statistical model because it was not among the monitored variables: according to the definition of the study population, only patients with ST-segment elevation or LBBB were eligible. In Wong's study, RBBB accompanying anterior AMI at presentation and new BBB (both LBBB and RBBB) early after fibrinolytic therapy were independent predictors of the high 30-day mortality. For the diagnosis of AMI with RBBB, other ECG changes (ST-segment elevations) had also to be present.

These findings prompted considerations about the role of BBB in diagnosing myocardial infarction. LBBB is regarded as an equivalent of ST-segment elevations in patients presenting with chest pain due to the possibility of complete masking ST-segment elevations or Q waves. Criteria for diagnosing AMI in the presence of LBBB exist (Sgarbossa's criteria, see *Table 1*),[19] but they are not widely used and the approach of LBBB being an equivalent of ST-segment elevation is preferred. However, according to the retrospective study of Shlipak et al.,[20] only 28% of patients presenting with chest pain and LBBB had myocardial infarction confirmed. Patients with chronic LBBB referred to elective coronary angiography had coronary artery disease proved in 54% of cases.[21]

In a prospective study by Melgarejo-Moreno et al.,[22] the incidence and role of RBBB were examined. Complications of myocardial infarction, like heart failure, atrioventricular block and death were significantly higher in patients with RBBB. Early mortality was significantly higher for new RBBB (43%; $p < 0.001$) than for old or indeterminate (15%)

Table 1

Criterion	Sensitivity (%)	Specificity (%)
ST-segment elevation ≥ 1 mm and concordant with QRS complex	73	92
ST-segment depression ≥ 1 mm in lead V1, V2, or V3	25	96
ST-segment elevation ≥ 5 mm and discordant with QRS complex	31	92
Positive T wave in lead V5 or V6	26	92
Left axis deviation	72	48

Adapted from: Sgarbossa EB, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. N Engl J Med 1996;334:481–7.

RBBB. Additionally, multivariate analysis showed an independent prognostic value of RBBB for early and 1-year mortality.

RBBB is thought not to mask the repolarization phase changes or Q waves; therefore, other ECG changes have to be present to establish the diagnosis of AMI. Some authors warn about this "clear-cut" opinion,[23] pointing out that minor ST-segment elevations in the anterior leads (V1–V4) can be missed due to compensation by pseudo-normalization of negative T waves.

Time of percutaneous coronary interventions

Some more recent studies were dedicated to determining the significance of RBBB in the coronary intervention era. Kurisu et al. showed[24] that in patients with AMI undergoing urgent reperfusion by coronary intervention, the presence of RBBB (either on admission or developing during the course of myocardial infarction) was a significant risk factor for early mortality; 30-day mortality was significantly higher in patients with than in those without RBBB (14.0% vs. 1.9%, respectively; $p < 0.01$). Also, left ventricular ejection fraction was significantly worse in patients with than in those without RBBB (45 ± 14% vs. 55 ± 14%, respectively; $p < 0.01$) *(Figure 2)*.

The study of Kleeman et al.,[25] based on a prospective registry of acute coronary syndromes, elucidated the role of RBBB in myocardial infarction without ST elevation; all previous studies were dedicated to RBBB in ST-segment elevation myocardial infarction. As in other studies, patients presenting with RBBB and both STEMI or NSTEMI were older, had a higher prevalence of previous myocardial infarction, diabetes and renal failure, and more often suffered cardiogenic shock. In the STEMI group of patients, patients with RBBB had more than double both in-hospital (26% vs. 11%, $p < 0.001$) and long-term (19% vs. 9.2%, $p < 0.001$) mortality than patients without RBBB. After adjustment for differences in baseline characteristics, RBBB remained an independent predictor of increased mortality in this group of patients. In comparison, RBBB in patients with NSTEMI was not associated with increased in-hospital mortality after adjusting for baseline characteristics and admission findings. Additionally, this study documented underuse of guideline-recommended short- and long-term treatment in patients with STEMI or NSTEMI and RBBB (this includes primary reperfusion therapy and subsequent pharmacotherapy).[25]

Another quite recent study looking into determinants of in-hospital mortality of patients with myocardial infarction found RBBB to be a significant factor. In their retrospective study on a group of 25 cases of left main coronary artery myocardial infarction, Sakakura et al.[26] discovered that in-hospital mortality (60% in this group) was associated with a history of hypertension, higher heart rate, RBBB and low hydrogen carbonate anion on admission. In further logistic regression analysis, RBBB and low hydrogen carbonate anion were found to be independent predictors of mortality.

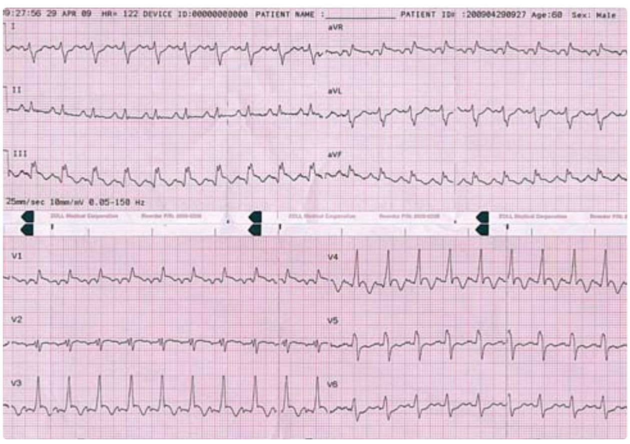

Figure 2 **New RBBB + LPH in a patient with cardiogenic shock. Coronary angiogram showed acute occlusion of LAD and chronic occlusions of left circumflex artery and right coronary artery**

Cardiac surgery

In the field of cardiac surgery, the issue of ventricular conduction disturbances as markers of perioperative myocardial ischemia by coronary bypass surgery was studied. Some studies demonstrated that postoperative occurrence of LBBB predicts an unfavorable long-term prognosis; like the one of Caspi et al.,[27] where LBBB was considered a marker of intraoperative myocardial damage. Other studies such as that of Tuzcu et al.[28] did not show any prognostic significance of LBBB or RBBB after coronary bypass surgery. However, this study only explored the prognostic value of BBBs while not evaluating them as possible markers of myocardial ischemia. In a more recent prospective study of Seitelberger et al.,[29] enzyme analysis, repeated 12-lead ECG, and continuous Holter monitoring were combined for diagnosing perioperative myocardial ischemia. It was concluded that the occurrence of new RBBB following elective CABG was indicative of perioperative myocardial necrosis.

ECG changes and proximal occlusion of coronary arteries

The electrocardiogram is one of the basic tools for diagnosing myocardial infarction, though its specificity is limited by large inter-individual variations in the anatomy of coronary arteries as well as the influence of previous diseases (e.g., previous myocardial infarction). Although topical ECG findings representing individual coronary vessels are well known today, the exact identification of proximal occlusions still remains a widely debated topic.[30]

According to the review article by Nikus,[31] the ECG changes accompanying a left main coronary artery (LMCA) occlusion, one of the most serious acute coronary syndromes, have an ST-segment elevation pattern in most cases. ST-segment elevation in the aVR lead is considered to be particularly typical for a LMCA occlusion.[32] However, conduction disturbances induced by extensive ischemia are also very common in LMCA occlusion.

Hirano et al.[33] retrospectively selected a group of 35 patients with LMCA occlusion from their myocardial infarction registry. Thirteen (37%) patients had RBBB, and only one had LBBB. There were two typical findings significant for LMCA occlusion. The first was RBBB with a left-axis deviation, accompanied by ST-elevation in aVR, as well as ST-segment elevation in leads I, V2–V5 in 70% of cases. The second type of finding was similar to changes in the left anterior descending artery occlusion, e.g., marked ST-elevation in leads V2–V5. Contralateral ST-depressions were common in both cases. Similar results were obtained in another study with 25 patients, with 52% of patients having RBBB.[34]

These findings are not surprising considering the anatomy of coronary arteries and the conduction system. Necrosis of the septum, which is an almost inevitable finding in LMCA occlusion, affects the conduction system below the atrioventricular node, with PR prolongation and wide QRS complex (RBBB pattern) as a consequence.[30] Also, as has been mentioned already, LAH is a typical finding while complete LBBB is not a very common one in LMCA occlusion.[33]

As has been pointed out above, RBBB is generally not believed to pose a major problem in interpreting ECG when diagnosing myocardial infarction, though it has been shown that even interpretation of Q-waves can be tricky.[35] It is believed that RBBB does not cause significant alterations in the spatial orientation of an initial excitation wave front; the depolarization of the interventricular septum runs in an unchanged direction (from left to right). However, this problem has been disputed for decades and some recent works show that the situation is not as easy as we may wish it to be.

Gussak et al.[36] showed that the presence of RBBB after myocardial infarction shortened Q wave duration (most pronounced in aVF, and less pronounced, yet still significant in leads II and III), thus enabling a false-negative diagnosis of inferior myocardial infarction. Similarly, the term "RBBB-dependent Q-wave" was introduced by Rosenbaum,[37] who described the appearance of new Q waves in leads V1–V2 disappearing after the restoration of normal conduction. Thus false-positive and false-negative diagnoses of myocardial infarction can be established when describing ECG with RBBB in a patient presenting with chest pain.

Conclusion

Indications for reperfusion in AMI have evolved over time, with ECG criteria becoming more precisely specified in the management guidelines for this disease. We have enough evidence for urgent reperfusion in patients presenting with ST-segment elevation or LBBB, but the situation in patients with chest pain and RBBB (± LAH or LPH) seems to be much less clear. While numerous studies show that the presence of RBBB in AMI is associated with higher in-hospital and long term mortality, we also expect other ECG changes than RBBB only to be present to meet ECG criteria for diagnosing myocardial infarction. However, as has been pointed out, both false negative and positive interpretations are possible. Other conditions, like pulmonary embolism, may present with chest pain and RBBB. To elucidate this situation, a study observing patients presenting with chest pain and RBBB is needed to evaluate the situation and fill in the gap in our knowledge.

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