Diagnosis of Ventricular Aneurysm and Other Severe Segmental Left Ventricular Dysfunction Consequent to a Myocardial Infarction in the Presence of Right Bundle Branch Block: ECG Correlates of a Positive Diagnosis Made via Echocardiography and/or Contrast Ventriculography

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Background: A diagnostic ECG sign of a ventricular aneurysm (VA) consequent to a myocardial infarction (MI) in the presence of complete left bundle branch block was recently described, and consists of the presence of ST-segment elevation (+ST), instead of the expected ST-segment depression (–ST), in leads V_{4-6} . Generally, complete right bundle branch block (RBBB) is associated with –ST in ECG leads V_{1-3} . We hypothesized that stable +ST, instead of the expected –ST in leads V_{1-3} in patients with RBBB could be also diagnostic of a VA and other severe segmental left ventricular dysfunction (VA/SSD). Thus, this study was performed to explore the feasibility of using the ECG to diagnose a VA/SSD in the presence of RBBB, and to evaluate the determinants of such diagnosis.

Methods: The frequency of +ST ≥ 1 mm in leads V_{1-3} was assessed in patients with RBBB, prior MI, and a VA/SSD diagnosed by echocardiography and/or contrast left cine-ventriculography. The ECG correlates for a positive or negative diagnosis of a VA/SSD were explored.

Results: Out of 4197 files of our cohort of the Cardiology Clinic, RBBB was detected in 175 patients. Of these, 28 had an old MI, and had a VA/SSD diagnosed by ≥ 1 of noninvasive and/or invasive non-ECG tests. Twenty-one of these 28 patients had stable +ST in ≥ 1 of leads V_{1-3} (Group 1), and 7 did not (Group 2). Thus, the sensitivity of this ECG criterion for the diagnosis of VA/SSD was 75%, and the specificity was 100% in this highly selective group. VA/SSD in the septal and anterior myocardial regions was more frequent in the patients of Group 1, than in the patients of Group 2 ($P = 0.03$ and 0.02 , correspondingly). The number of myocardial territories involved with the VA/SSD, or the ejection fraction were not different in the two groups ($P = 0.65$ and 0.55 , correspondingly).

Conclusion: VA/SSD can be diagnosed in the presence of RBBB by the concordant to the QRS repolarization changes (+ST) in leads V_{1-3} . Positivity of this ECG marker for VA/SSD correlates with involvement of the septal or anterior myocardial regions, and represents mechanistically a superimposition of primary repolarization alterations, overcoming the secondary such changes.

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electrocardiography; ventricular aneurysm; severe segmental left ventricular dysfunction; right bundle branch block; echocardiography; contrast ventriculography

Bundle branch blocks are characterized by prolonged QRS complexes associated with secondary repolarization changes whose polarity is opposite to that of the QRS complexes.^{1–8} The hallmarks

of complete right bundle branch block (RBBB) are QRS complexes ≥ 120 ms in duration with late forces pointing toward V_1 – V_3 leads associated with secondary (discordant to the QRS complexes)

repolarization changes (ST-segment depression [−ST] and T-wave inversion).^{1–8} Repolarization deviations concordant to the QRS complexes are considered primary, i.e., owing to other causes than the conduction abnormality (often ischemia, or myocardial infarction [MI]). Thus, ST-segment elevation (+ST) recorded in leads V₁–V₃ should be considered primary in nature, and a cause for its presence should be sought.

Recently, diagnostic ECG characteristics of ventricular aneurysm (VA) in the presence of left bundle branch block (LBBB) were described and consist of stable +STs in left precordial leads with predominantly upright QRS complexes (particularly in V₅).^{9–11} We wondered whether VA and other severe segmental left ventricular dysfunction (VA/SSD) in conjunction with RBBB could lead to similar persisting +ST in leads V₁–V₃. Indeed, in the past few years, we had encountered a few such patients with an old MI, RBBB, and +ST in V₁–V₃, with a VA subsequently confirmed by non-ECG diagnostic methods, and thus we wanted to explore this issue systematically. A Medline literature search on the diagnosis of VA in the presence of RBBB did not lead to any relevant work. Thus, we contacted a cross-sectional survey to define criteria of VA/SSD in the presence of RBBB, their sensitivity, specificity, and other attributes.

METHODS

Patients

All 4197 files of our entire Cardiology Clinic cohort were reviewed, searching for patients with RBBB, old MI, and VA/SSD, diagnosed via non-ECG testing (echocardiography [ECHO] and/or contrast left cine-ventriculography [LV-gram]). The ECG attributes and determinants of a positive or negative VA/SSD diagnosis were then explored. Since the main focus of our investigation was the ECG expression of VA/SSD, other clinical and laboratory attributes, risk factors, management, and outcome during the acute phase of MI were not considered.

Research Variables

All available ECHOs and LV-grams (all biplanes) of the patients with RBBB were reviewed by the authors, and this included both the images and the official hospital reports attached to the patients records; however, the analysis was based on the specific wording used in the official hospital re-

ports. A VA/SSD consequent to an old MI was considered to be present when a diagnosis of a VA, dyskinesis, or akinesis with a dilated myocardial segment at systole and diastole was mentioned in the report of ECHO and/or LV-gram,¹² and through such characterization, a VA/SSD+ and VA/SSD- subgroups of patients were formed. Our definition for a VA was liberal, and included VA, dyskinesis, or akinesis, the latter two included as SSD. Both a dilated segment with dyskinesis or akinesis in the LV-gram, or a dilated segment with hyperechoic features, decreased thickness, displaying absence of systolic thickening, and revealing akinesis, or dyskinesis in the ECHO were considered reasons for a positive non-ECG diagnosis of a VA/SSD. We tabulated a positive or negative diagnosis of VA/SSD, referred to any of six myocardial territories (apex, anterior, posterior, septal, lateral, and inferior) by any of the two non-ECG tests. If a particular territory was interpreted as showing VA/SSD by only one of the two tests, the positive diagnosis was considered in the analysis; also the same approach was used when 1 of ≥ 2 of ECHOs, or LV-grams per patient were available. When composite terms (i.e., anterolateral or inferoposterior) were used, information was considered for both constituents of such terms (i.e., anterior and lateral or inferior and posterior, correspondingly). In addition, the total number of the myocardial territories involved with VA/SSD in each patient were tabulated and used as a variable. Ejection fraction (EF) measured by LV-gram was also used as a variable.

Q-waves and history consistent with MI were not a prerequisite for inclusion in the study, since often Q-waves disappear following an MI (although such characterization is unlikely in the setting of a VA owing to a large MI), multiple MIs lead to a "pseudonormalization" of the ECG owing to cancellation, many patients could have suffered silent MIs, or had their MIs at another hospital or even in another country, and thus we could not document with certainty the occurrence of such pathology. Other variables included the presence of +ST ≥ 1.0 mm, measured at 60 ms from the "J" point, and taking the T-P as the isoelectric line, and Q-waves in ≥ 1 of V₁–V₃, anterolateral (I, aVL, and V₄–V₆), or inferior (II, III, and aVF) leads. Isolated Q-waves in leads III and V₁ were ignored. Although the main focus of this communication was to examine the ST-segments in leads V₁–V₃ (where a modulating effect on the ST-segments would be expected by the VA/SSD in patients with RBBB), we have

evaluated our material for ST-segments changes in all other ECG leads; also Q-waves in all ECG leads were evaluated for their contribution to the diagnosis of VA/SSD.

The sensitivity and specificity of the proposed ECG criterion of VA/SSD was calculated. In addition, an explanation for a true positive or a false negative ECG diagnosis of a VA/SSD, depending on its location, was sought. The records of the patients with the combination of RBBB, MI, and VA/SSD were reviewed regarding history of ventricular arrhythmias, sudden death, and implantation of a cardioverter/defibrillator (ICD). The patients and/or their relatives were also contacted, as to patients' current status; they were classified as "noncontacted," if such contact failed.

Statistical Analysis

We used Fisher's exact test for the assessment of the frequency of involvement of the six differ-

ent territories with VA/SSD, or the presence of the various ECG variables in relationship to the proposed ECG diagnostic criterion of VA/SSD, and unpaired *t*-test to evaluate the number of territories involved with VA/SSD, the age, and EF, in the two groups of patients; the Levene test was applied to determine whether the pooled versus the separate variance estimates should be used.¹³ P values were 2-tailed, and were considered statistically significant when P was <0.05. The SPSS/PC + 4.0 statistical package was used in the analysis.¹³

RESULTS

A total of 4197 files were reviewed and 175 patients with RBBB were identified. The diagnosis of VA/SSD had been made in 28 patients aged 66.4 ± 10.1 (mean $\pm$ SD; range 45–86) years, including 26 males on the basis of a non-ECG test (Fig. 1); 118 patients had an evaluation by such tests,

Figure 1. Four-chamber view of an echocardiogram revealing the presence of a VA involving the septal and apical myocardial regions. Abbreviations: ANEU, aneurysm; LV, left ventricle; RV, right ventricle; LA, left atrium; RA, right atrium.

Figure 2. Twelve-lead ECG revealing RBBB and Q-waves in leads III, and V₁–V₄ diagnostic of an old MI; the unexpected in the presence of RBBB, +STs (arrows) indicate an underlying VA. Abbreviations, as explained in the text.

whereas 57 patients did not. Twenty-one patients had a VA/SSD by ECG (+ST in V₁–V₃ leads) (Fig. 2), whereas seven patients did not. Since the diagnosis of VA or dyskinesia for at least one myocardial territory was made by ECHO and/or LV-gram for all 28 patients, the differentiation by the ECG in Groups 1 and 2 did not reflect a difference in the makeup of left ventricular abnormality present (VA, dyskinesia, or akinesis). No patients were identified with a positive diagnosis of VA/SSD by the ECG, and negative such diagnosis by the non-ECG tests. Thus, the specificity of the proposed ECG criterion for the diagnosis of VA was found to be 100%, whereas

the sensitivity was 75%, when referring to the subgroup of 28 patients with a VA/SSD. However, in reference to the 118 patients who underwent a non-ECG evaluation the sensitivity was 75% and the specificity was 93%. As per definition, +ST in leads V₁–V₃ was not seen in any of the patients of Group 2, whereas it was detected in 15, 21, and 19 patients of Group 1, correspondingly, with the respective sensitivities being 53.6, 75, and 67.9%. In contrast, +ST in leads V₄–V₆, and in leads II, III, and aVF was found in eight and four patients of Group 1; and one and three patients of Group 2, correspondingly ($P = 0.37$ and 0.31).

Q-waves in leads V₁–V₃ were not found in patients of Group 2, whereas it was detected in 17, 20, and 18 patients of Group 1, correspondingly ($P = 0.0003$, 0.00001 , and 0.0001). From the 28 patients, 21 (75%) had a Q-wave in leads V₂ and/or V₃. Thus, the sensitivity and specificity of Q-waves in leads V₂ and/or V₃ for detection of VA/SSD was 75% for the cohort of 28 patients, whereas for the cohort of the 118 patients who were evaluated for the presence of a VA/SSD the sensitivity was 75%, and the specificity was 73%, indicating that 27 patients without a VA/SSD had a Q-wave particularly in V₂. Q-Waves in leads V₄–V₆ were detected in eight patients of Group 1, and one patient of Group 2 ($P = 0.37$). In contrast, Q-waves in leads II, III, and aVF were found in three patients of Group 1, and five patients of Group 2 ($P = 0.009$).

The number of patients with VA/D/A in the six different myocardial territories, the number of myocardial territories involved with VA/SSD, and the EF in the two groups are shown on Table 1. Only the differences in the involvement of the septal and anterior territories were statistically significant.

One patient of Group 1 suffered ventricular tachycardia and received an ICD and two died (1 of an unknown cause and 1 with asystole); four patients could not be contacted. In the Group 2, one died of an unknown cause and three could not be contacted.

DISCUSSION

It is widely accepted that QRS changes can be interpreted in the setting of RBBB, and that RBBB leads to less distortion of the QRS complexes than LBBB. Thus, it may even be debatable whether diagnosis of MI on the basis of the assessment of QRS complexes is problematic in the presence of RBBB. However, there is not much literature regarding the

Table 1. Number of Patients with RBBB and Diagnosis of VA/SSD by Non-ECG Modalities, According to ECG Diagnosis of VA/SSD, and to the Different Myocardial Territories

	Apex	Anterior	Posterior	Septal	Lateral	Inferior	#MyoTer	EF ^a
+ECG for VA/SSD (n = 21)	17	17	6	14	3	12	3.2 ± 1.2	38.7 ± 17.3
–ECG for VA/SSD (n = 7)	6	2	4	1	2	6	3.0 ± 1.2	43.8 ± 12.8
P	0.78	0.02	0.21	0.03	0.57	0.17	0.65	0.55

VA, ventricular aneurysm; SSD, severe segmental left ventricular dysfunction; +, positive; –, negative; #MyoTer, number of myocardial territories; EF, ejection fraction.

^aEF was measured in 16 of 21 patients with a +ECG, and 5 of the patients with a –ECG.

behavior of ST-segments in patients with MI and RBBB, and no information at all regarding the ST-segment changes in patients with RBBB and VA. On theoretical grounds, the possibility should be considered that the interplay of primary and secondary ST-segment deviations in patients with RBBB may render the diagnosis of MI or VA difficult. Accordingly, a modulating effect of RBBB on the +STs, consequent to an acute MI, was clearly shown in our previous work, where RBBB occurring intermittently in patients with acute anterior MI, produced intermittent attenuation of the +STs in V₁ and V₂ leads.¹⁴ Such reduction of the amplitude of +ST from an acute anterior MI or VA, as a result of RBBB, could be occasionally extreme to the point of elimination, rendering the ST-segment isoelectric. Experience shows that patients with acute MI and RBBB may show transient +ST in any ECG lead, including V₁–V₃. Such interplay of ST-segment changes can be conceptually extended to the patients with old MIs (where persistent +STs could suggest the presence of a VA),^{15,16} and this formed the basis for our hypothesis that VA/SSD can be diagnosed in the presence of RBBB. Discrimination of acute MI from VA in a patient with RBBB is based on the clinical context, and the time course of the repolarization changes (declining amplitude of +STs suggests an MI, whereas stable +STs denote a VA). The prevalence of VA/SSD in conjunction with RBBB in our cohort was similar to the one of VA with LBBB (28 and 27 cases, correspondingly).¹¹ The proposed criterion for the diagnosis of VA/SSD in the presence of RBBB was found to be 100% specific and quite sensitive, in contrast to the ECG criterion of VA in the presence of LBBB (75% vs 18.5%).¹¹ However, the sensitivity of the ECG criterion for the diagnosis of VA in patients with LBBB in that study rose to 41.7%, when patients with apical VA were considered. This may be explained by a favorable “matching” of lead V₅ (used

for diagnosis of VA in patients with LBBB), which is the lead optimally posed for “scanning” of the apical region.^{17–19} Similarly, leads V₁–V₃ (used for the diagnosis of VA in conjunction with RBBB) were ideally suited for recording the +STs owing to the VA/SSD in this study, where aneurismal or other severe regional involvement of septal/anterior territories was found to be very prevalent. In fact, this was the only determinant of a positive ECG diagnosis of VA/SSD, with the extent of VA/SSD or the EF being of no consequence (Table 1). However, it should be noted that the EF was not measured in all patients (Table 1). In addition, these last two findings do not imply that the number of involved territories or the EF are not associated with the presence of a VA/SSD, but that patients with the proposed ECG criterion for VA/SSD do not have more extensive involvement, or lower EF, as per our findings. Moreover, as these two parameters were suggesting that the VA/SSD in Group 1 was larger (Table 1), the lack of statistical significance of the difference between the size of the VA/SSD in Groups 1 and 2 may be due to the small number of patients in Group 2 (type 1 error).¹³ Involvement of septal/anterior regions with VA/SSD was “matched” by the exclusive presence of Q-waves in Group 1. In contrast, a VA/SSD involving the inferior myocardial region in Group 2 patients was associated with significantly more frequent presence of Q-waves in leads II, III, and aVF, than in Group 1 patients. ECG is insensitive to posterior and apical VA in patients with RBBB, and thus it should not be used as a screening test at all for this purpose. It is more important to focus on the feasibility of diagnosing a VA/SSD in the presence of RBBB on the basis of the described herein ECG criterion (Fig. 1), than to emphasize the degree of sensitivity or specificity found in our research cohort, taking into consideration the limitations of our study, being retrospective, and employing nonstrict and predefined

characterization of VA/SSD. Perhaps, evaluation of VA/SSD by other diagnostic modalities, presumably more accurate (e.g., magnetic resonance imaging) may lead to different results, or to better correlations of imaging and the ECG. The present study is also limited by the fact that 25% of the patients were not accounted for in the follow-up evaluation.

Our patients had segmental left ventricular areas indicative of VA or SSD, thus the following comments may not strictly apply to our cohort, since the 28 patients we are describing had not purely just a VA. Nevertheless, these patients had large areas with VA/SDD, and low EF, both of which are associated with increased mortality. Patients with VA are prone to sudden death, and are known to have a sixfold increase in mortality, when compared with patients without a VA and a similar left ventricular EF.^{20,21} In our patients with VA and LBBB, sudden death was documented in seven patients, and three patients had an ICD implanted,¹¹ whereas only three patients died in the present study (2 of unknown cause and 1 with asystole), and one had an ICD implanted. Moreover, follow-up was comparable in the two studies, with 9 of 27 patients in the LBBB cohort, and 7 of 28 in the one with RBBB not accounted for at the time of data analysis. Whether there is a difference in the rate of sudden death or need for ICD implantation in patients with VA and LBBB, and VA and RBBB cannot be resolved definitively by our studies, although the former in our experience appears to be a group with a higher risk. MADIT II²² established the role of ICD in preventing sudden death in patients with a history of MI, low EF, and no evidence of ventricular arrhythmias. In that study, 101 patients among 1232 had a nonspecific intraventricular conduction delay (IVCD), LBBB, or RBBB; only 16 had RBBB, with 9 allocated to the group who received an ICD, and 7 to the group who had conventional therapy. As analysis in that study does not refer to patients with VA (with or without IVCD, LBBB, or RBBB), one cannot ascertain whether such patients derived greater benefit from the ICD than patients without these characteristics. It would be of interest to have an ad hoc analysis of MADIT II with attention on the above, although the number of patients involved was small (total of 37 with LBBB and 16 with RBBB), with the corresponding proportions of patients with associated VA being unknown. In addition, COMPANION,²³ another ongoing study, evaluating patients with IVCD and congestive heart failure, and implementing biventricular pac-

ing and/or ICDs may supply relevant answers to the above questions. It is in the context of these two trials,^{22,23} and similar works that the present communication and our previous work,¹¹ may realize their clinical implications. Before our data can be quoted in the clinical setting, particularly in reference to the sensitivity and specificity of the proposed ECG criterion for VA/SDD, a prospective study is needed. In such a study, the non-ECG evaluation needs to be strictly well defined by design, and calculation of the specificity and sensitivity of the proposed herein ECG criterion for different left ventricular abnormalities needs to be made. Finally, the resemblance of the ECG features that we have used to make the diagnosis of VA/SSD in our patients with RBBB, with what one encounters in Brugada syndrome,²⁴ where specifically the leads V₁₋₃ are also singled out for diagnostic purposes as showing +ST, deserves some qualification. In patients with RBBB and a VA/SDD, Q-waves from the previous MI will also be present in V₁₋₃ and other leads, and imaging modalities would be diagnostic for segmental left ventricular contraction abnormalities.

REFERENCES

1. Wagner GS, ed. *Marriott's Practical Electrocardiography*, 9th Edition. Baltimore, Williams & Wilkins, 1994, pp. 79, 83, 96.
2. Mirvis DM, ed. *Electrocardiography. A Physiologic Approach*. St. Louis, Mosby, 1993, p. 304.
3. Beckwith R ed. *Grant's Clinical Electrocardiography*, 2nd Edition. (Revised) New York, McGraw-Hill Book Company, 1970, p. 121.
4. Macfarlane PW, Vetch Lawrie TD. *Comprehensive Electrocardiography*. New York, Pergamon Press, 1989, p. 476.
5. Dunn Mi, Lipman BS, eds. *Lipman-Massie Clinical Electrocardiography*, 7th Edition. Chicago, Year Book Medical Publishers, Inc., 1989, p. 146-190.
6. Goldberger AL, Goldberger E, eds. *Clinical Electrocardiography. A Simplified Approach*, 5th Edition. St. Louis, The C.V. Mosby Co., 1994, p. 73.
7. Goldberger AL, ed. *Myocardial Infarction. Electrocardiographic Differential Diagnosis*. St. Louis, The C.V. Mosby Co., 1975, p. 70.
8. Sodi-Pallares D, ed. *New Bases of Electrocardiography*. St. Louis, The C.V. Mosby Co., 1956, pp. 405, 419.
9. Madias JE, Kaminetsky A, Solanki N. Myocardial infarction-induced ventricular aneurysm in the presence of complete left bundle branch block: A case report suggesting a new electrocardiographic diagnostic criterion. *J Electrocardiol* 1999;32:179-183.
10. Madias JE. Myocardial infarction-induced ventricular aneurysm in the presence of complete left bundle branch block [Letter to the Editor]. *J Electrocardiol* 1999;32:373-375.
11. Madias JE, Ashtiani R, Agarawal H, et al. Diagnosis of myocardial infarction-induced ventricular aneurysm in the presence of complete left bundle branch block. *J Electrocardiol* 2001;34:147-154.

12. Feigenbaum H. Echocardiography, 5th Edition. Baltimore, Williams & Wilkins, A Woverly Co., 1994, pp. 468, 469, 470-472.
13. Norusis MJ. The SPSS Guide to Data Analysis for SPSS/PC+, 2nd Edition. Chicago, IL, SPSS, Inc., 1991, pp. 118, 231, 264.
14. Madias JE, Venkataraman K, Hood WB, Jr. Precordial ST-segment mapping. Clinical studies in the Coronary Care Unit. *Circulation* 1975;52:799-809.
15. Ford RV, Levine HD. The electrocardiographic clue to ventricular aneurysm. *Ann Intern Med* 1951;34:998.
16. Madias JE. Electrocardiography in myocardial infarction [Review]. *J Electrocardiol* 1979;12:411-423.
17. Blackburn H, Taylor HL, Vasquez CL, et al. The exercise electrocardiogram during exercise: Findings in bipolar chest leads of 1449 middle-aged men, at moderate work levels. *Circulation* 1966;34:1034.
18. Ellestad MH. Stress Testing: Principles and Practice, 4th Edition. Philadelphia, F.A. Davis Co., 1996, p. 104.
19. Madias JE, Tziros C, Knez P, et al. Transient ischemia of the apex (among other territories) as the only independent determinant of positivity of exercise ECG. (Abstract) *Circulation* 1998;98:(Suppl. I):I-293.
20. Hassapoyannes CA, Stuck LM, Hornung CA, et al. Effect of left ventricular aneurysm on risk of sudden and nonsudden cardiac death. *Am J Cardiol* 1991;67:454-459.
21. Hochman JS, Brooks MM, Morris M, et al. Prognostic significance of left ventricular aneurysm in the Cardiac arrhythmia Suppression Trial (CAST) population. *Am Heart J* 1994;127:824-832.
22. Moss AJ, Zareba W, Hall WJ, et al. For the Multicenter Automatic Defibrillator Implantation Trial II Investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med* 2002;346:877-883.
23. Bristow MR, Feldman AM, Saxon LA. Heart failure management using implantable devices for ventricular resynchronization: Comparison of medical therapy, pacing, and defibrillation in chronic heart failure (COMPANION) trial. COMPANION steering committee and COMPANION clinical investigators. *J Card Fail* 2000;6:276-285.
24. Brugada J, Brugada R, Brugada P. Right bundle branch block and ST-segment elevation in leads V₁ through V₃; a marker for sudden death in patients without demonstrable structural heart disease. *Circulation* 1998;97:457-460.