

ORIGINAL ARTICLE

Vectocardiographic analysis of right ventricular electrical conduction delay

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Abstract

Introduction: The electrocardiographic interpretation of end conduction delay (ECD) in the right ventricular branch of the heart has already generated some hypotheses that this right branch is not single, as anatomically demonstrated, and can be divided into distinct terminal branches when we analyze tracings through the vectorcardiogram.

Methods: There were 227 electrocardiograms selected, with typical characteristics defined as ECD of patients from the electrocardiography service of the Centro Universitário Saúde ABC, of both sexes, in the age range of 18 to 87 years, with varied ethnicities, weight and height, with cardiovascular risk factors or without them. We performed vectorcardiograms in these patients to observe the behavior of the final portion of electrical conduction.

Results: Analyzing the vectorcardiographic tracings of patients who presented ECD in electrocardiogram, we confirmed in the recording by the frontal plane, the presence of ECD but recorded in three distinct regions; 103 patients in the right upper quadrant between -120° and -150° , 45 patients in the right lower quadrant between $+170^\circ$ and -170° , and medial, and 79 patients in the right lower quadrant between $+110^\circ$ and $+140^\circ$.

Conclusion: Electrical depolarization of the heart in the right ventricle in electrocardiographic tracings apparently records typical alterations that we can diagnose as depolarization of a single bundle; but when we performed vectorcardiograms, we recorded three distinct zones of right ventricular depolarization with delay; i.e., three distinct sectors of right ventricle free wall delay, such as type I (upper), type II (lower) and type III (medial).

Keywords: Electrical stimulation, Branch block.

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Authors summary

Why was this study done?

The right branch of the Bundle of His has always been believed to be unique, but several authors have hypothesized that it actually subdivides into more terminal branches, which has not been proven anatomically visible, but we think they are preferential pathways of depolarization. In this review, we used the vectorcardiogram to record it, as it is a more accurate methodology than the electrocardiogram.

What did the researchers do and find?

We investigated with electrocardiographic differences in the final delays of conduction of the right branch and even anatomically, trying to identify anatomical divisions in the Right Branch. Some authors even used vectorcardiograms, but without classifying the types compared to the electrocardiographic tracing, as in this study

What do these findings mean?

With these findings we can prove that there really is a sectorial right ventricular depolarization, which can help us in the differential diagnoses from other pathologies.

INTRODUCTION

The electrocardiographic (ECG) interpretation of electrical end conduction delay (ECD) in the right ventricular (RV) branch has originated several hypotheses; among them, that the right bundle branch (RBB) is not single as anatomically demonstrated, and could be divided into different terminal branches or regions when analyzed through a vectorcardiogram (VCG). The electrical branches of the ventricles are originated in the atrioventricular node (AV node). The His bundle originates the RBB and the left bundle branch, composed by special cells capable of conducting electricity, called cardiomyocytes, with conduction velocity greater than the myocardial cells. Anatomically, the RBB ends in the RV free wall, which leads to cell-to-cell depolarization after that. This change in electrical conduction initiated by the RBB has always intrigued researchers, as they question the possibility of this depolarization in the RV free wall, as already suggested, occurring by the intraventricular conduction system of the RV free wall or by preferential pathways in the myocardium.

The electrocardiographic diagnosis of ECD in the RBB is well established worldwide¹. In this study, we proposed to perform the analysis of the VCG, which is more accurate device in recording the electrocardiographic path, to monitor the behavior of this delay in the RV free wall in order to identify the divisions of electrical conduction by electrical beams or myocardial sections.

AIM

This study aimed to describe the vectorcardiographic tracings of electrical RV ECDs.

METHODS

Design of the study

Study performed during the period from June 1 to September 30, 2017. The ECG of 227 patients from the department of graphic methods of the Centro Universitário FMABC were analyzed. Only ECGs with ECD were included according to the described criteria.

Inclusion criteria

In the study, patients older than 18 years of age were included, who met the criteria for incomplete right bundle branch block (IRBBB) or ECD with 12-lead ECG, according to the 3rd Guidelines of Electro-Vectorcardiography Interpretation of the Sociedade Brasileira de Cardiologia¹.

The electro-vectorcardiographic inclusion criteria were:

- QRS duration between 90 and 100 ms (<120 ms);
- Triphasic morphology in V1 or V1-V2 (rsr', rSr', rsR', RsR'), or morphology in M followed by negative T wave;
- Biphasic morphology in V1 (rS), with notch in the ascending ramp of S wave (atypical IRBBB);
- Presence of S wave of slow or prolonged (slurred) inscription in the left leads: I, aVL, V5 and V6;
- rSr' pattern with final r or R wave of slow inscription in aVR lead;
- Presence of criterion of SI, SII, SIII syndrome;
- Presence of extreme SÂQRS shift in the superior quadrants (SÂQRS to the left of -30°) associated with SII ≥ SIII;
- In patients who had undergone VCG, only those who had 30 or more dashes with slow inscription at the end of the QRS and located to the right in the frontal and horizontal planes (HPs) were included, following the criteria proposed by Pastore *et al*²;
- QRS loop in the HP, of predominantly clockwise rotation, and sometimes eight-shaped;
- Maximum voltage vector usually smaller than normal.

Exclusion criteria

Patients older than 18 years with QRS interval <90 ms and/or ≥120 ms.

VCG test

After the ECG inclusion criteria were met, all 227 patients were invited to undergo a VCG test.

The VCG is a noninvasive exam, which was performed in the cardiology department of Centro Universitário FMABC by a trained cardiologist following the Frank method³. The exam is performed with the patient in the supine position, lasting approximately 5 minutes. The seven electrodes are positioned on the body surface, trunk, leg and neck as shown in figure 1. This exam does not pose any additional risk to patients. Both the ECGs and VCGs were performed using the DMS Cardioscan 12 equipment.

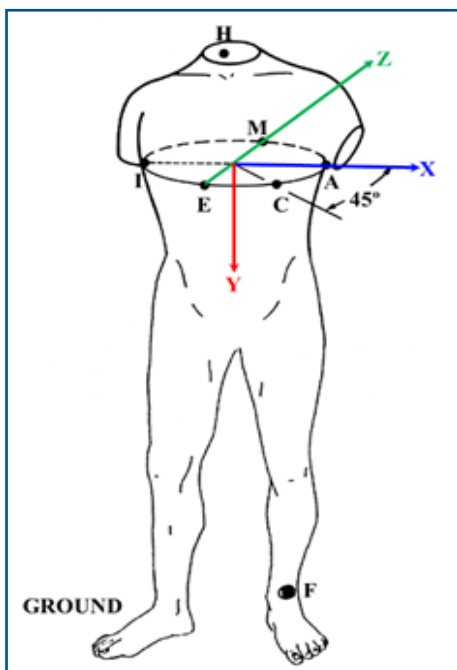


Figure 1: Frank's method – electrodes positioning. Location of electrodes to perform vectorcardiogram using the Frank's method. Seven electrodes are placed on the body surface. H: Posterior region of the neck; F: Left leg; I: Right mid axillary line (4th or 5th right intercostal space); A: Left mid axillary line (4th or 5th left intercostal space); C: Mid clavicular line (4th or 5th left intercostal space); E: Center of sternum (4th or 5th left intercostal space); M: Spinal apophysis (4th or 5th left intercostal space)

The VCG tracings were analyzed (figure 2) to confirm the presence of ECD and its behavior in the recording. For this analysis, the frontal plane (FP) was selected because they have the leads where the electrical axes of the heart are calculated, since in this plane the heart and its electrical bundles can be almost entirely observed.

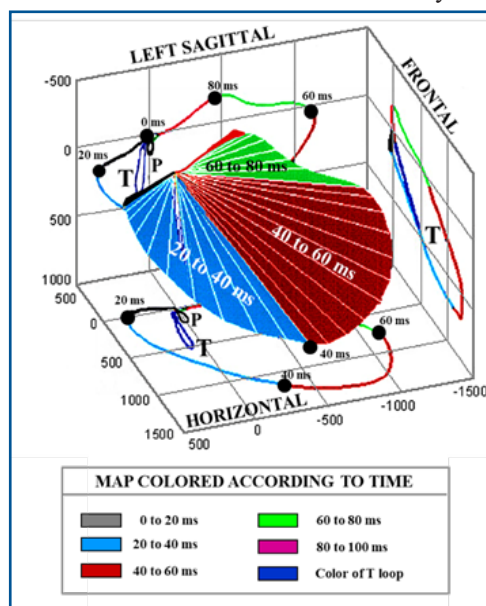


Figure 2: Representation of QRS loops in the three planes of space (frontal, horizontal and left sagittal) represented in colors according to the time elapsed

RESULTS

All 227 patients selected for evaluation of VCG attended the examination. There were 118 men and 109 women, aged between 18 and 82 years (average of 50 years), weight between 65 and 121 kg, and height between 1.58 to 1.89 m. No risk factors, with three asymptomatic cardiovascular risk factors, and baseline patient characteristics are described in table 1.

Table 1: Cardiovascular and electrocardiographic characteristics of patients. ECG: electrocardiogram; DVRA: diffuse disturbance of ventricular repolarization; LAFB: Left anterior fascicular block; LPFB: Left posterior fascicular block

	n	(%)
Women	109	48.1
Men	118	51.9
Cardiovascular risk factors		
3 factors	53	23.34
2 factors	72	31.71
1 factor	69	30.39
No risk factors	34	14.97
Electrocardiogram		
With no other ECG alterations	137	60.35
With DDVR	40	17.62
With LAFB	41	18.06
With LPFB	4	1.76
ST segment alteration	5	2.20

Analyzing the VCG tracings in the FP of patients who presented characteristic of ECD without apparent structural heart disease. The recording of ECD in different sectors of the right quadrant was found; According to the location of ECD in the FP, we made a classification into 3 types (figure 3): Type I, when ECD is located in the right superior quadrant in the FP between -120° and -150° ; Type II, when ECD is located in the right inferior quadrant in the FP between $+110^\circ$ and $+140^\circ$; Type III, when ECD is located between $+170^\circ$ and -170° in the FP.

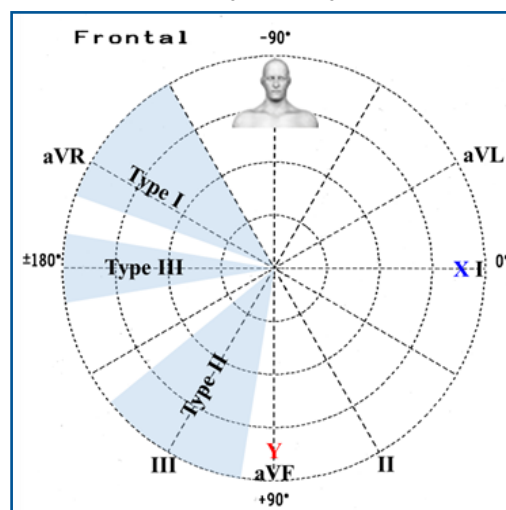


Figure 3: Types I, II and III of ECD. The figure shows the location of ECD at the right of the final/terminal portion of the QRS loop in the FP. The criterion used was ≥ 15 dashes (≥ 30 ms) closer to each other at the end of the QRS loop in at least two dashes

According to the rotation of the QRS loop in the FP, we divided type I into three subtypes: IA, when the QRS loop turns in a counterclockwise rotation and the QRS axis presents extreme leftward shift. This subtype could be difficult to differentiate from left anterior fascicular block; IB, when the QRS loop rotation is clockwise. This subtype may shift the electrical QRS axis to the right, and at times, it

suggests differential diagnosis with left posterior fascicular block (LPFB); IC, when the rotation of the QRS loop is eight-shaped, resembling a monoplane propeller (figure 4).

Table 2 contains information previously published by this research group^{4,5}. Here, in this study, we present these data with the addition of differential diagnosis between type IA of ECD and left anterior fascicular block.

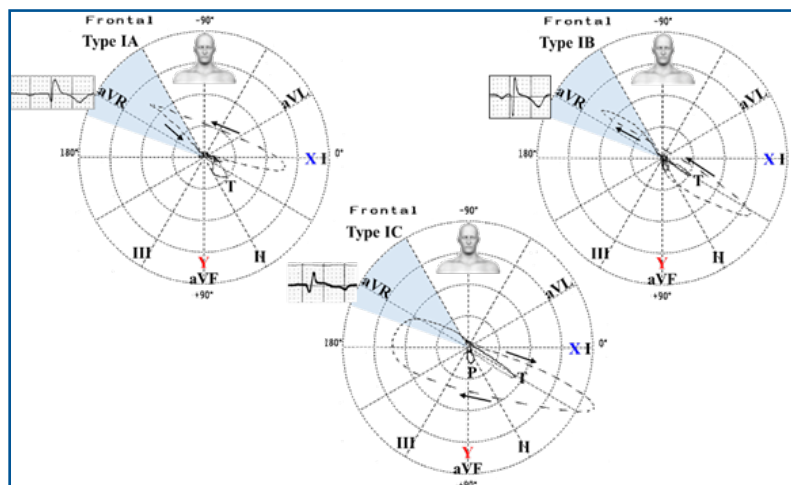


Figure 4: Types I, II and III of end conduction delay with ECG

Table 2: Electro-vectorcardiographic characteristics of type IA

10 ms initial vector of QRS loop	With downward and rightward direction	With downward and leftward direction
qrs morphology in I and aVL	qR pattern	Rs
SII/SIII ratio	SIII	SII>SIII (low sensitivity)
Location of ECD	In the left upper quadrant when present	In the right upper quadrant ²
Prominent R wave in aVR (R ≥ 0.3 mV)	Absent	It could be present and is called aVR sign in BrS ⁶
Morphology of QRS circuit of vectorial cardiogram in the horizontal plane	Similar to normal	Similar to type-C right slurring pattern: initial vector at the front and the left, counterclockwise rotation and ≥20% of the loop area located in the right posterior quadrant in the horizontal plane ⁷

Electro-vectorcardiographic characteristics of type IA

SÂQRS with extreme shift in the left superior quadrant between -30° and -90°; QRS loop of counterclockwise rotation in the FP; rapid passage from left to right of the QRS loop; predominantly negative QRS complexes in inferior leads; prominent S wave in these leads; SII>SIII: useful for differential diagnosis with left anterior fascicular block; prominent and/or wide R wave of aVR; aVR of qR or QR type, with R wave frequently wide.

Electro-vectorcardiographic characteristics of type IB

SÂQRS difficult to be determined: indeterminate or perpendicular to the FP, suggesting the presence in all FP axes, with isoelectric complexes that make this determination difficult. Pointed, eight-shaped QRS loop in the FP, with the initial portions located in the left inferior

quadrant, and the final portions in the right superior quadrant, where the ECD can be located. The aspect of QRS loop is similar to the propellers of a plane.

Electro-vectorcardiographic characteristics of type IC

SÂQRS not shifted or shifted to the right; spikey aspect of the QRS loop in the FP with clockwise rotation; ECD in the right superior quadrant in the FP; frequent SI-SII-SIII; wide R wave in aVR; morphology of IRBBB; rSR' in V1; sagittal plane clearly shows QRS loop perpendicular to Y line; discrete ECD of 30 ms (15 dashes) located in the right superior quadrant between -100° and -160°, or greater.

When the basic cause is QrS, the presence of prominent final R wave in lead aVR: R wave ≥3 mm or R/q ≥0.75 in lead aVR (aVR sign).

Electro-vectorcardiographic characteristics of type II

Characterized by ECD located in the right inferior quadrant. It corresponds to the right inferior bundle territory (LPFB), which makes the differential diagnosis (table 3).

ECG: $\hat{A}QRS$ between $+70^\circ$ and $+100^\circ$; normal QRS duration; S1-R2-R3 pattern with R2 and R3 non-increased pressure (generally ≤ 10 mm), never reaching 15 mm (essential element for the differential diagnosis with LPFB); $R2 \geq R3$ (in LPFB $R3 > R2$); aVR of QS type; possible notch in the descending ramp in inferior leads;

deeper s wave in V2 and/or V3; persistent S wave up to V5 and/or V6; V1: rS, RS or rSR' with S of V1 and V2 possibly enlarged.

VCG FP: initial vectors always on the left, above and below; clockwise rotation; predominant location in inferior quadrants; rapid change from left to right between 30 ms and 50 ms; ECD at the right and below between $+120^\circ$ and $+150^\circ$. HP: QRS loop of counterclockwise rotation; rapid change from left to right, between 40 and 50 ms; ECD backward and rightward. Initial vectors upward and downward; clockwise rotation; posteroinferior rotation; ECD downward and backward.

Table 3: Differential diagnosis between type II ECD and LPFB

	Type II ECD	LPFB
	Normal	Frequent prolongation
Association with inferior infarction	No	Frequent
Pressure of RII and RIII	≤ 10 mm	≥ 15 mm
RII/RIII pressure ratio	RII > RIII	RIII > RII
Notch in R wave descending slope in inferior leads	Absent	Constant notch at the middle-end
Intrinsic shift in aVF, V5 and V6	Normal	Increase: up to 30 ms
Intrinsic deflection in AVL	Normal	Decrease: up to 15 ms
QRS loop appearance in frontal plane	Clockwise and with characteristic rapid passage from left to right, between 30 to 50 ms	Clockwise, appearance of "fat" loop and maximum vector close to $+120^\circ$
Clinical factors that should be excluded	Not stated	Vertical heart, left ventricular remodeling and lateral infarction

Electro-vectorcardiographic characteristics of type III

Type III is characterized by ECD located in the middle territory or anterior fascicle of the right branch, very close or around $\pm 180^\circ$.

DISCUSSION

The electrocardiographic tracings with ECD and concomitant alterations such as LAFB and LPFB, myocardial infarction of inferior wall, electro-vectorcardiographic pattern of Brugada syndrome, or a subpopulation of arrhythmogenic right ventricular dysplasia/cardiomyopathy may, according to the electro-vectorcardiographic recording, have an unspecific diagnosis of ECD. This identifies the right bundle branch as a single branch, which if it occurred as proposed, would depolarize the whole right ventricular free wall simultaneously as a block; however, we identified that in the final portion of the right ventricular free wall there may be depolarization in different portions or sectors, which characterize the right branch or anatomical subdivision of the electrical bundle, or by cell-to-cell trajectory⁵.

The right bundle branch does not present anatomically distinguishable pathways and is considered a single fascicle. The tetrafascicular system (three dependent fascicles that are part of the left bundle branch: left anterior fascicle, left posterior fascicle and left septal fascicle, and continuation of the right bundle branch) activates the Purkinje network, a subendocardial plexiform layer of

dense intramural branches capable of finally activating the small segments corresponding to the myocardium. For this reason, in normal conditions, the myocardium is activated from the endocardium toward the epicardium⁸.

Nagao *et al.*⁹ studied the conduction system in endocardial excitation spreading through the right ventricle (RV). The role of the Purkinje network in an excitation sequence in the RV endocardial surface was studied using an isolated perfused dog preparation. This was electrically stimulated in the proximal right bundle, and the activation time was mapped using contiguous bipolar electrodes, or a microelectrode, or both. The first muscle activation was observed at the junction between the ventricular septum and the free wall at the front of the anterior papillary muscle. After the initial activation, the propagation of ventricular muscular excitation has a radial propagation form and a velocity of 1.67 ± 0.20 m/sec. Muscle activation in this area was almost always preceded by activation in Purkinje fibers by 2 to 6 ms. Thus the Purkinje system is indispensable for widespread excitation in this area. In the lower third of the septum, the excitation sequence was essentially similar to the RV free wall, indicating a contribution of rapid conduction by the Purkinje system. In contrast, in the upper two-thirds of the RV free wall activation of ventricular muscle propagation from apex to base with a significantly delayed conduction and velocity of 0.41 ± 0.88 m/s, and was not preceded by activation of Purkinje fibers, thus indicating a lack or a drastic decrease of involvement of the Purkinje system in the basal areas of

the RV, justifying the physiological ECD of the QRS loop.

The anterior, posterior and septal branches of the right bundle in the RV free wall is a functional distribution. The propagation of excitation of the ventricular endocardial muscle was confirmed by selective transection of one of those special fibers, suggesting the etiological significance of the lesion in the right bundle branch as a cause for several electrocardiographic patterns of IRBBB. In spite of having true right bundle branches in the RV free wall, these proven three preferential pathways justify a trifascicular free wall: it is at least functional.

The functional distribution of the three main right bundle branches through the incisional interruption of the free wall was investigated, and the data obtained revealed that the anterior branch was responsible for the activation of the anterior septum, anterior paraseptal free wall and right ventricular outflow tract. The lateral and posterior branches contributed to the free wall and posterior septum activation. The most severe distortion of the wavefronts was induced by lateral branch interruption. This finding supports the important role of the Purkinje system in the dissemination of RV activation to the free wall muscle.

Conduction delay in the upper septum after anterior or posterior branch incision may reflect the distribution of these branches toward the inferior septum. The maximum delay of ventricular muscular activation induced by incision ranged from 6 to 12 ms. However, when the anterior or posterior branches were interrupted, the maximum delay in activation was, at times, observed in the area proximal to the incision lines. This could be attributed to artificial damage to the septal muscle during the incision procedure. Because of the anatomy of these branches, it was quite difficult to only cut the conducting tissue. Thus, the interruption of the branches may have caused an undesirable lesion in the underlying septal muscle. The current findings are in general agreement with previous *in vivo* studies, where the effects of RV distal interruption were investigated. Moore *et al.*¹⁰, by cutting false tendons, found a similar delayed activation area in the epicardial surface of the RV. Smith *et al.*¹¹ verified a partial delay in the epicardium after making a large laceration on the septal surface of the RV; this delayed area was almost consistent with that obtained in our study, splitting the posterior branch. These two groups recorded an IRBBB pattern in ECG simultaneously. Uhley *et al.*¹² produced two types of IRBBB in the ECG by cutting peripheral RBBB bundles. All these findings tend to suggest a possible etiological significance of lesions on each main RBBB branch as a cause for several types of IRBBB with pattern I. However, more extensive clinical studies are necessary to confirm such possibility, since there are differences between species in the anatomical characteristics of the His-Purkinje system¹³, and between the effects of activation delay within the right ventricular endocardium in the QRS configuration.

The mammalian heart must function as an efficient pump while simultaneously carrying out electrical impulses to conduct the contraction process. In the ventricles, electrical activation begins at the points of insertion of the Purkinje system into the endocardium. As the diffusion component of the subsequent excitation wave propagates from the endocardium in the normal cardiac wall of the free wall without creating directional biases⁹.

The chronology of electrical events that mechanically activate the myocardium has been described as beginning at the level of the septum, spreading to the apex, then to the bodies of both ventricles, and eventually to the base of the heart (apex-base activation). Francisco Torrent-Guasp^{14,15} suggested that the myocardium is a single muscle band that forms a double-helical loop. The contraction of the myocardium would follow the trajectory of muscle fibers that originate in the pulmonary artery towards the body of the ventricles and the aorta (base-to-apex contraction). This would explain the movements in the base of the heart and the twisted movement of the ventricles seen in magnetic resonance studies.

Slow conduction in the RVOT may contribute to a greater risk of arrhythmia⁶. Many of the cases described in literature as LPFB are, the way we see them, type II ECD, and as their electro-vectorcardiographic differences are very subtle, the diagnosis must always be clinical electro-vectorcardiographic.

CONCLUSION

The electro-vectorcardiographic recording in patients with identified ECDs demonstrates that the RV free wall depolarizes in three distinct sectors that occurs either by special conduction cells such as right branch fascicles or sectoral depolarization independent of the final portion of the right bundle branch, but also we not only detected the identification of three distinct sectors in the RV free wall, but it also allows us to identify aberrant tracings that eventually suggest serious adjacent structural heart disease, such as inferior and/or anterior infarctions, chronic obstructive pulmonary disease, fascicular blocks of the right bundle branch similar as left septal fascicular block and LPFB. In addition, these dromotropic disorders can be found in heart diseases that potentially lead to sudden cardiac death, such as arrhythmogenic right ventricular cardiomyopathy/dysplasia. The scarce dissemination of these concepts is surprising, taking into account the relevance of their differential diagnoses.

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Conflicts of interest

The authors declare no conflicts of interest regarding this manuscript.

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Resumo

Introdução: A interpretação eletrocardiográfica do atraso final da condução no ramo do ventrículo direito do coração já gerou algumas hipóteses de que esse ramo direito não é único como demonstrado anatomicamente e que pode ser dividido em ramos terminais distintos quando analisamos o traçado através do vetocardiograma.

Método: Separados 227 eletrocardiogramas com características típicas definidas como atraso final de condução dos pacientes do serviço de eletrocardiografia do Centro Universitário FMABC, de ambos os sexos na faixa de idade de 18 a 87 anos, etnias, peso e estatura variadas com fatores de risco cardiovascular ou sem fator de risco, realizamos vetocardiograma nesses pacientes para observar o comportamento da porção final da condução elétrica.

Resultado: Analisando os traçados vetocardiográficos dos pacientes que apresentavam o atraso final de condução no eletrocardiograma, confirmamos no registro pelo plano frontal, a presença do atraso final de condução, porém registravam em três regiões distintas; 103 pacientes no quadrante superior direito entre -120° e -150° , 45 pacientes no quadrante inferior direito entre $+170^\circ$ e -170° e medial e 79 pacientes no quadrante inferior direito entre $+110^\circ$ e $+140^\circ$.

Conclusão: A despolarização elétrica do coração no ventrículo direito no traçado eletrocardiográfico aparentemente registra alterações típicas que podemos diagnosticar como uma despolarização de um feixe único, porém ao realizarmos vetocardiograma, registramos três zonas distintas de despolarização ventricular direita com atraso, ou seja, três setores distintos da parede livre do ventrículo direito como atraso Tipo I (superior), Tipo II (inferior), e Tipo III (medial).

Palavras-chave: Estimulação elétrica, bloqueio de ramo.

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