CARDIOVASCULAR AND METABOLIC SCIENCE www.ancam.org.mx

REVIEW

Vol. 32 No. 4
October-December 2021

Deductive analysis of the electrocardiogram to determine the site of origin of premature ventricular beats/contractions

Análisis deductivo del electrocardiograma para definir el sitio de origen de las extrasístoles ventriculares

Keywords:
Electrocardiogram, ventricular premature contractions, site of origin, electrocardiogram analysis, ventricular arrhythmia location.

Palabras clave:
Electrocardiograma, extrasístoles ventriculares, sitio de origen, análisis del electrocardiograma, localización de arritmia ventricular.

Enrique Asensio-Lafuente,* Jorge Álvarez-de la Cadena-Sillas,‡ Emanuel Sánchez-Guevara,§ Gustavo Solache-Ortiz,¶ Humberto Rodríguez-Reyes,‖ Susano Lara-Vaca**

* Cardiologist, EP, División de Medicina Interna, Hospital H+ Querétaro, Qro.
‡ Cardiologist Instituto de Corazón de Querétaro, Qro.
§ Cardiologist, EP, Hospital Star Médica Querétaro, Qro.
¶ Cardiologist, Instituto de Cardiología Preventiva de San Juan del Río, Qro.
‖ Cardiologist, EP, Sociedad Cardiovascular y de Arritmias SOCAyA, Aguascalientes, Ags.
** Cardiologist, EP, Hospital Ángeles León, Gto.

Mexico.

Received: 14/07/2021
Accepted: 22/09/2021

ABSTRACT

Premature ventricular contractions (PVC's) have become a therapeutic target, especially when the arrhythmia burden is higher than 10% of the recorded beats. Knowing their point of origin can help to optimize the different treatment strategies, especially the invasive ones. Deductive electrocardiogram (ECG) analysis is a rational methodology that assesses the activation vector and morphologies of the PVC's thus allowing a quite precise definition of the arrhythmia originating focus. Knowledge of the different cardiac structures within the chest and the heart itself as well as between them facilitates the comprehension of the vectors and morphologies on the twelve-lead ECG and gives valuable information to complete the patient's clinical picture. Here we present the main ways to analyze the PVC's ECG from the classical concepts, and we include diagnostic parameters to identify their point of origin.

RESUMEN

Las extrasístoles ventriculares se han convertido en objeto de tratamiento, especialmente si la carga de arritmia es mayor al 10% de los latidos en un día. Conocer el sitio de origen de las mismas permite optimizar la planeación de las estrategias de tratamiento, especialmente las invasivas. El análisis deductivo del electrocardiograma (ECG) es una forma racional de analizar los vectores de activación y las morfologías de las extrasístoles, gracias a la cual se puede definir de manera bastante precisa el foco de origen de la arritmia. Conocer la posición de las estructuras cardiacas en el tórax y en el propio corazón, así como las relaciones entre ellas, facilita la comprensión de los vectores y morfologías de los complejos en el ECG de doce derivadas y es una información valiosa para completar el panorama clínico del enfermo. En este trabajo se presentan las principales formas de analizar el ECG de las extrasístoles ventriculares a partir de los conceptos clásicos y se incluyen parámetros de diagnóstico para definir su origen.

INTRODUCTION

Premature ventricular contractions (PVC's) are common arrhythmias that have to be evaluated in the complete clinical context of the patient. Their presence is related to higher mortality risk in the presence of cardiac disease, and possibly so among subjects with a structurally normal heart. The risk of sudden cardiac death in each of them might be different, but in any case, higher than in the general population. Patients with PVC's also have a high risk to develop a «tachycardiomyopathy» or an arrhythmia-induced cardiomyopathy that will cause heart failure (HF). Development of HF will depend on the arrhythmia burden, the patient's co-morbidities, use of medications, metabolic or electrolyte disorders, presence of channelopathies and family history as main factors.[1-4]

How to cite: Asensio-Lafuente E, Álvarez-de la Cadena-Sillas J, Sánchez-Guevara E, Solache-Ortiz G, Rodríguez-Reyes H, Lara-Vaca S. Deductive analysis of the electrocardiogram to determine the site of origin of premature ventricular beats/contractions. Cardiovasc Metab Sci. 2021; 32 (4): 214-225. https://dx.doi.org/10.35366/102773

The present work is intended to help the clinician determine the approximate anatomical origin of the PVC's since that knowledge might help to define therapeutic plans that currently consider ablation procedures earlier in the treatment process because of its increasing success rates.[5]

It is important for the clinician to be able to determine the site of origin of PVC's for several reasons, even if the treatment is intended to be done by an electrophysiology service. It is known that the right ventricle's PVC's, especially the ones originating from the outflow tract, usually have a benign course and they usually appear in structurally normal hearts, while the PVC's arising from the left ventricle might be related to structural heart disease and thus, usually have a worse prognosis.[6] A higher arrhythmia burden induces tachycardiomyopathy or at least ventricular dysfunction. Nonetheless, that risk is higher when the PVC's come from the tricuspid annulus, even when compared against the ones that originated from the papillary muscles or the left bundle branch fascicles.[7,8] The ventricular function deterioration is independent of the presence or absence of baseline heart disease.[9-13]

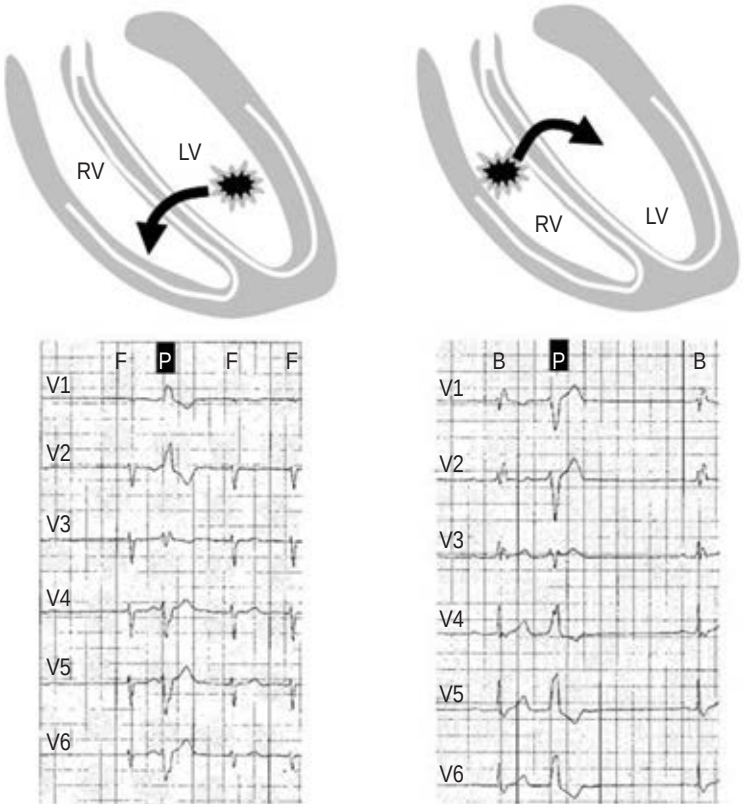


Figure 1: When a PVC is originated in the ventricular myocardium, the action potential induces a «cell-to-cell» depolarization pattern similar to the one seen when there is a bundle branch block. That «jump» from one ventricle to the other induces a left bundle branch block (LBBB) when the PVC comes from the right ventricle, and if generated in the left ventricular, it will show a RBBB pattern as a general rule, but with exceptions mentioned in the text.

Arrhythmia units are still scarce in Mexico, that is why specialized evaluation is usually delayed, and the clinician will be prompted to make appropriate and timely therapeutic decisions. Review all of them is out of reach of the present work, even though it has to be taken into account that the presence of structural heart disease – ischemia scars, hypertrophic cardiomyopathy or dysplasia, among others – restrict the choice of antiarrhythmic drugs, that have frequent side or toxic effects. Some fascicular PVC's will respond to verapamil, that can be used as a «bridging» therapy while waiting for ablation. Propafenone might also be used in subjects without structural heart disease. It must be considered that the PVC might arise from the lesion site - ischemia scar, for instance – or maybe originated from a random independent focus or as a pro-arrhythmic effect of drugs. In these circumstances, the definition of the origin site allows optimizing ischemia treatment, for example, while a more definitive therapy is defined and implemented.

The 12-lead electrocardiogram (ECG) has some limitations to define a precise anatomic location, however it allows locating the PVC's origin to a certain region, frequently in the vicinity of a specific intra-cardiac structure, even though it might also originate from a random point in the myocardium.

With this information, the prognostic significance of the PVC's can be more precise, and the planning for an invasive therapeutic strategy will be more efficient.[14]

The Mexican Electrocardiography School has used the deductive analysis technique since its origins.[15] Putting together the depolarization axis in the frontal and horizontal plane information, along with the morphological analysis of the ECG waves and their duration and other data, allowing to define the PVC's

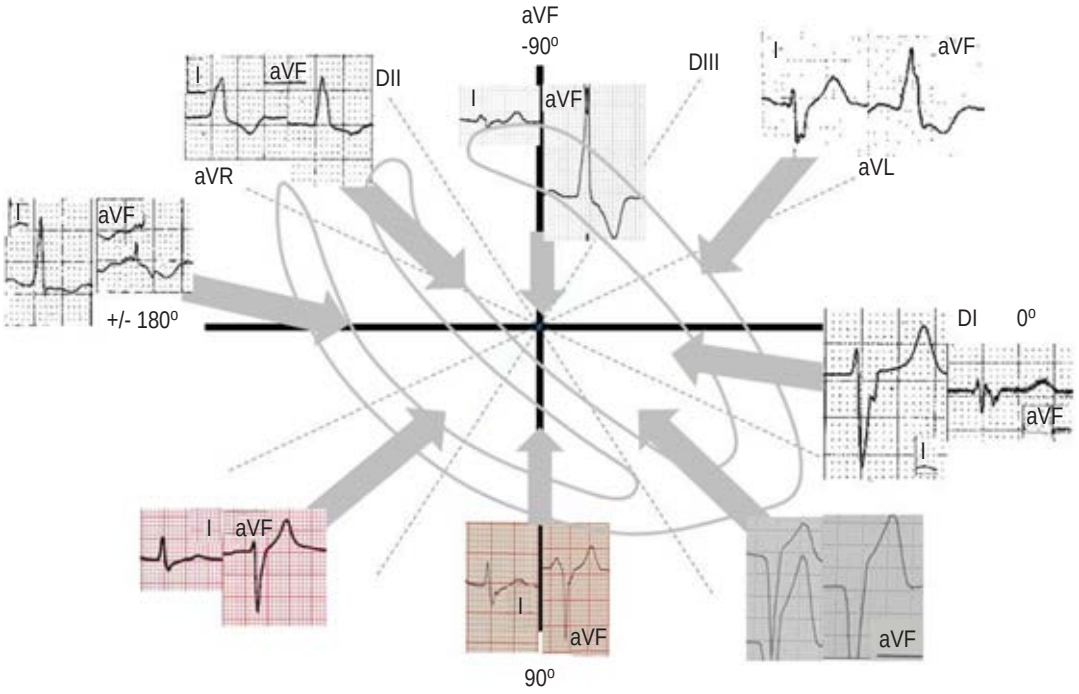


Figure 2: The main ventricular depolarization axis in the frontal plane allows to approximately determine the site of origin of a PVC to a superior or inferior focus and left or right of the originating ventricle. An important issue is that the main depolarization vector is directed away from its originating point. Thus, if a PVC shows a left inferior axis, its origin is an upper right location. In the image, the ECG recording is located in the approximate origin point and the arrow points in the ventricle's main vector's depolarization direction.

origin with enough accuracy and it is a familiar tool for many of us.

The present simplified review is intended to facilitate a better comprehension of the ECG criteria useful to establish the origin point of PVC's.

PREMATURE VENTRICULAR CONTRACTION'S MORPHOLOGY

The PVC's morphology is determined by its site of origin. Usually, the depolarization focus is located within the myocardium, away from the specialized conduction system. That focus generates a stimulus that has to cross the myocardium and induce ventricular contraction. As in the bundle branch blocks, when the PVC is originated in the left ventricle (LV), the PVC adopts a Right bundle branch block (RBBB) morphology and vice versa, when the PVC has a left bundle branch block (LBBB) morphology, its origin is usually in the right ventricle (RV), exception made in some cases of PVC's from the aortic cusps, that might show an LBBB pattern too.[16-18] This phenomenon is explained by the myocardial activation sequence. When the action potential initiated by the automatic focus of the PVC crosses the myocardium, it has to make a «jump» from one ventricle to the other, an action that implies a delay in the depolarization of the ventricular myocardium *(Figure 1)*. When there are polymorphic PVC's, the patient's clinical context must be known and included in the analysis. Some patients with extensive scarred tissue might have a single generation focus within the myocardium, with several «exit» points, or present with different generation foci distributed through the fibrotic area. Cardiac magnetic resonance imaging (MRI) studies in subjects undergoing ventricular tachycardia (VT) ablation have shown that polymorphic PVC's or PVC's with right bundle branch block

morphology are frequently related to a larger amount of fibrotic tissue.[19]

DEPOLARIZATION AXES

The depolarization axes in the frontal and horizontal plane allow a more precise identification of the ventricular ectopic focus.

The depolarization axis can be determined using DI and aVF *(Figure 2)* in the usual way for the frontal plane, which shows four 90º main quadrants (upper left 0 to -90º, upper right -90º to -180º, and so on with the lower left and lower right), and V1 and V6 for the horizontal one *(Figure 3)*. This way, one can locate the quadrant of origin of the PVC from the main depolarization vector in a right-to-left, superior-inferior and anterior-posterior direction.

The main depolarization axis can be determined in the same way that the atrial or ventricular depolarization vector direction is defined, but it is important to remember that the main vector is directed away from its source. Thus, if a PVC depolarization vector has a right-inferior axis, its source is most likely the left superior quadrant. If it is positive in V1, it comes from the posterior segments,[20] but it comes from the anterior region if V1 is negative. If the vector is positive in V6, it comes from the right, and if it is negative in V6, it comes from the left. The work by Asirvatham demonstrates that V1 is useful to differentiate between PVC's arising from the right ventricle outflow tract or, in a more general way, from any of both outflow tracts. If the main QRS deflection is negative in V1 and the transition zone is delayed (V4), the origin is usually anterior –right ventricle (RV)–.[20,21] The presence of positive deflections with earlier transition zones (V2) suggests a posterior origin, possibly related to the aortic cusps.[20]

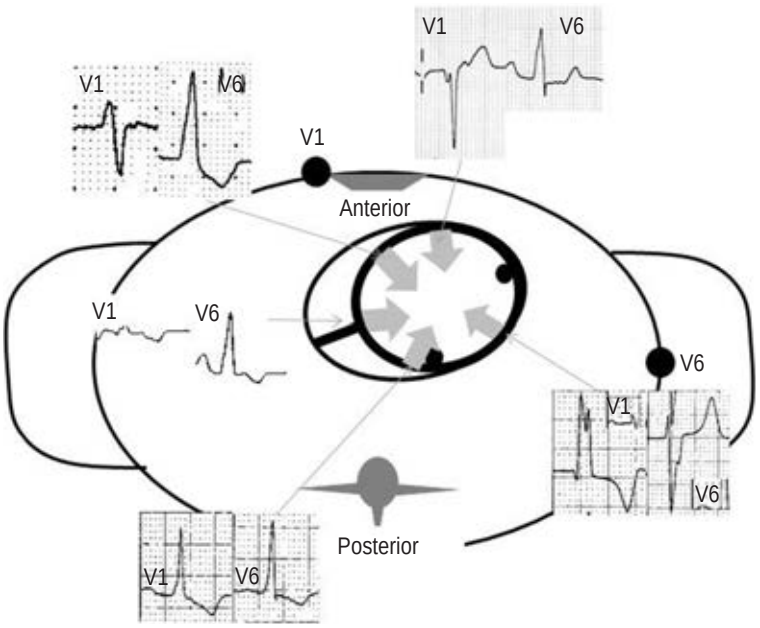


Figure 3: The horizontal plane's depolarization axis allows the definition of an approximate origin point for the PVC in the anterior-posterior and lateral-medial dimensions. Again, the main depolarization vector is directed away from its origin point. If a PVC has a positive vector in V1 and negative in V6, its origin is most likely left postero-lateral or left lateral. In the image, the ECG record is located in the approximate origin site. The volume arrow points in the main depolarization vector's direction. The thin arrow points to its vector.

At this point, morphological information must be added. For instance, if the PVC has an LBBB pattern, the PVC is originated in the right ventricle, although it has to be kept in mind that PVC's from the right aortic cups might also show an LBBB pattern. If the depolarization axis is directed towards the left inferior quadrant, The PVC is most likely originated in the right superior segment of the right ventricle. If the QRS is negative in V1 and positive in V6, its origin is located in the anterior and superior segment of the right ventricle. The structure located in that position is the right ventricular outflow tract. If the main depolarization vector of the PVC's QRS is directed in a right superior direction, with an RBBB morphology, positive in V1 and negative n V6, the PVC is arising from the posterior mid-apical segments of the left ventricle. A structure that might originate that PVC is the postero-medial papillary muscle *(Figures 4 and 5, Table 1)*.[22]

Further on, we will review specific features from specific locations since there are regions where structures are superimposed, and the precise differentiation of a focal origin is more complex.

EPICARDIUM OR ENDOCARDIUM?

Most ventricular arrhythmias arise from the endocardium; nonetheless, ablation

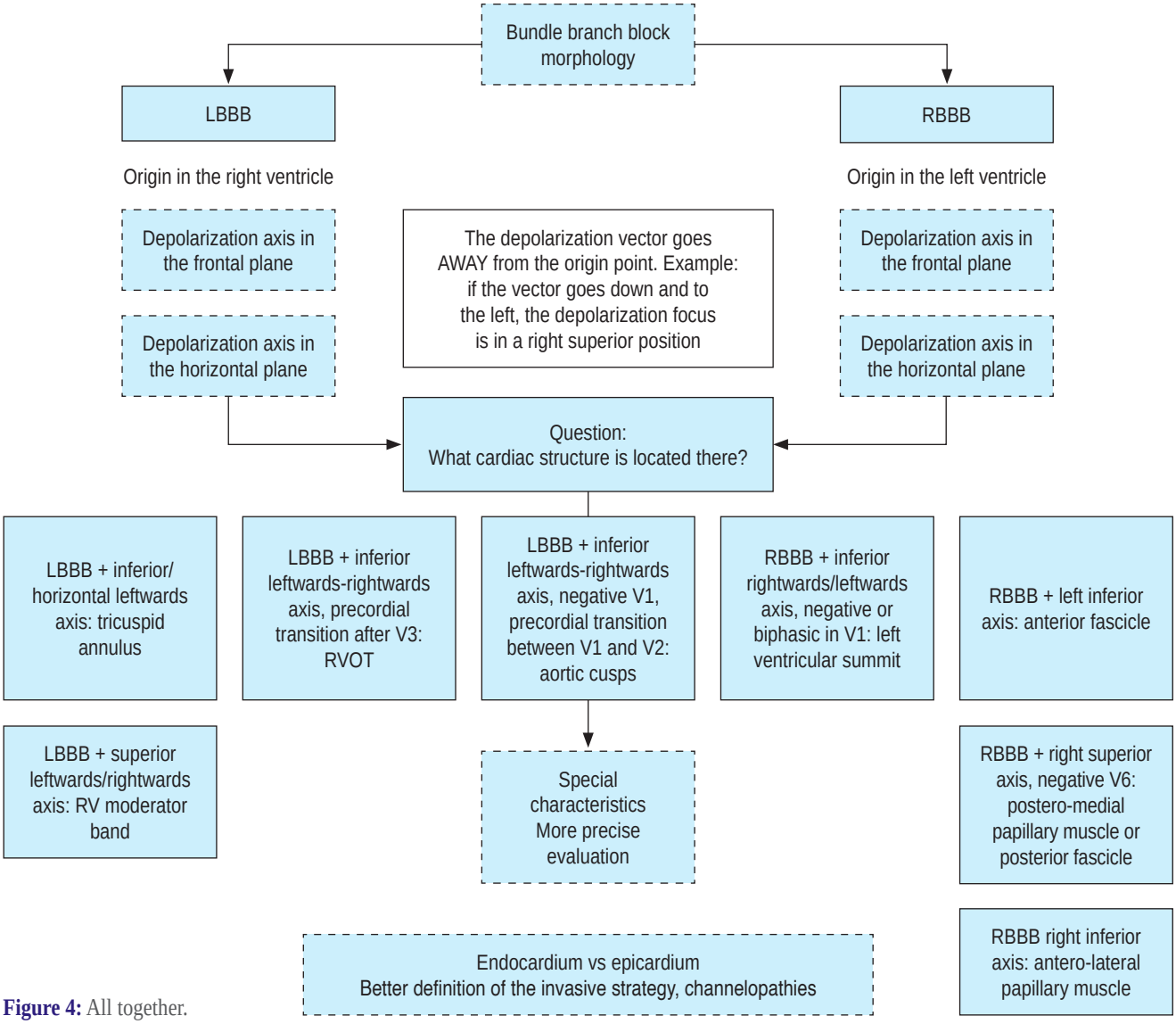


Figure 4: All together.

studies have demonstrated that a certain number of them come from the epicardium.[23,24] The main heart disease has to be considered too; for example, among people with arrhythmogenic right ventricular cardiomyopathy or a dilated cardiomyopathy, an epicardiac origin is more frequent than in patients with idiopathic arrhythmias or ischemic heart disease.[25] Brugada syndrome, a complex channelopathy, can be treated with ablation of the right ventricular epicardium, even in the absence of PVC's from that area.

Another element to consider is the morphology of the scar itself – which can be defined using MRI – in ischemic heart disease subjects. In them, an automatic focus might have different «exit» points through different myocardium channels, thus mandating extensive ablation to isolate the whole scarred tissue. The different exit points might show different ECG patterns.

The ECG of the epicardiac arrhythmias usually shows an initially slow depolarization pattern until it reaches the His-Purkinje system. It manifests as a pseudo-delta wave that lasts

more than 34 ms in the precordial leads, an intrinsecoid deflection in V2 longer than 85 ms (measured from the start of the QRS complex to the R wave peak) as well as other criteria listed in *Table 2* and illustrated in *Figure 6*.[24]

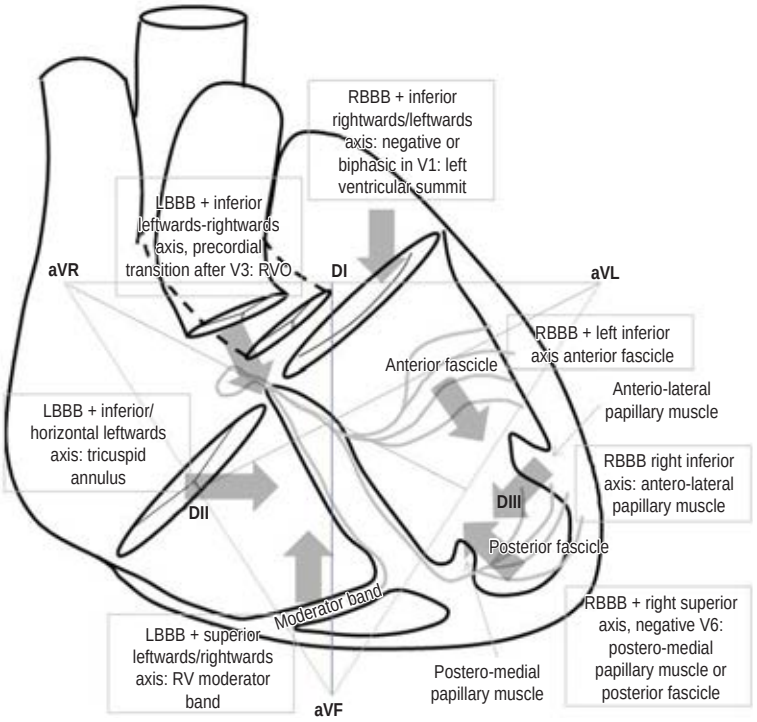


Figure 5: All together: main depolarization vectors from the main intra-ventricular structures.

SPECIFIC ORIGIN FOCI

Outflow tracts or superior ventricular foci

Both ventricles outflow tracts have a complex superposition, in a way that different kinds of ventricular arrhythmias might have similar ECG features *(Figure 7)*.[25] To avoid unnecessary complications, we have decided to suppress anatomic discussions. From an ECG standpoint, it is important to know the location of the anatomical structures adequately, but the

Table 2: Main electrocardiographic features of the epicardiac ventricular arrhythmias.

Epicardiac ventricular arrhythmia features
Pseudo-delta wave > 34 ms
Intrinsecoid deflexion in V2 > 85 ms
Shorter RS complex > 121 ms
Maximum deflexion index in precordial leads > 0.55 (onset to QRS peak/total QRS duration)
Q wave in DI (antero-lateral epicardiac ventricular arrhythmia)
Q wave in inferior leads (inferior epicardiac ventricular arrhythmia)

Modified from: Boas R et al.[14]

Table 1: Getting things together.

Depolarization vector main direction	Bundle branch block	Possible origin
Inferior or horizontal leftward axis, with a postero-anterior direction	Left bundle branch	Tricuspid annulus
Inferior axis with a leftward, center or rightwards precordial leads transition after V3	Left bundle branch	Right ventricular outflow tract
Inferior axis with a leftward, center or rightwards axis, precordial leads transition prior to V3	Left bundle branch	Aortic cusps
Superior vertical, right of leftwards axis	Left bundle branch	Moderator band
Inferior or horizontal axis with a right wards deviation, negative V6	Right bundle branch	Mitral annulus
Inferior left axis, monophasic S pattern V1	Right bundle branch	Left ventricular summit
Superior right axis, negative V6	Right bundle branch	Posterior fascicle or posterior-medial papillary muscle
Inferior right axis	Right bundle branch	Antero-lateral papillary muscle
Inferior left axis	Right bundle branch	Anterior fascicle

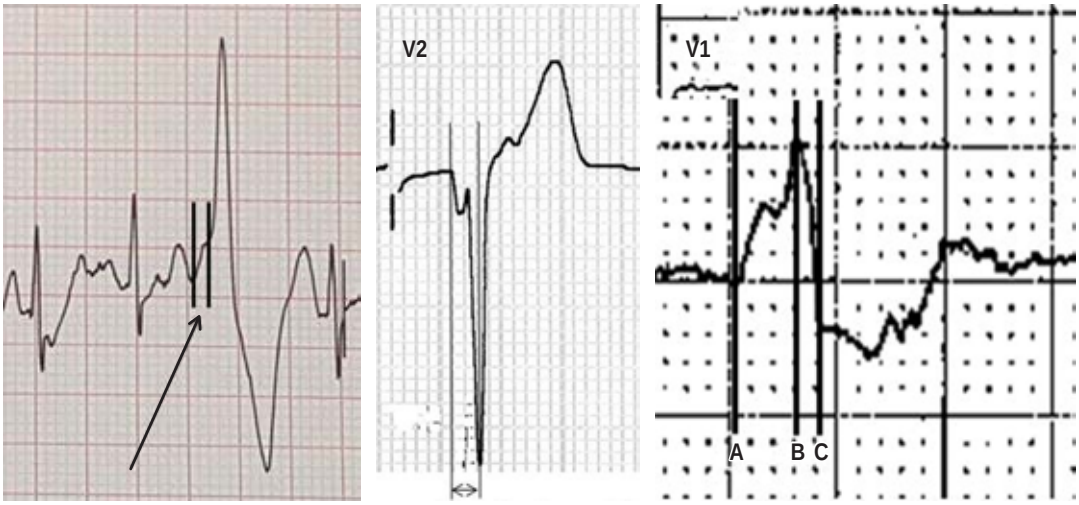


Figure 6:

Ventricular epicardiac arrhythmia features.

Pseudo-delta
> 34 ms

Intrinsecoid deflexion
> 85 ms

Maximum index deflexion
AB/AC = X
120/160 ms = 0.75

anatomic discussion about the existence or not of a left outflow tract might only be confusing in this work.

Right ventricular outflow tract (RVOT)

This structure is located in the superior region of the precordium, in the center of the chest. The RVOT surrounds the left ventricular outflow tract and crosses it in the anterior portion, so that the pulmonary valve is located in front and to the left of the aortic valve.[25] The RVOT is the site of origin of 70 to 80% of the idiopathic arrhythmias of the outflow tracts. The PVC's originating from that point will show an LBBB pattern and an inferior axis in the frontal plane that might slightly deviate right or left. The RVOT can be conceptualized as a semi-circle structure with two opposed crests. The anterior or free wall, and the posterior or septal wall have posterior (right) and anterior (left) extensions. Thus, the RV free wall is located behind the sternum, but both outflow tracts are superimposed. A transition at V3 or later (V4, V5, V6) suggests that the origin is the RVOT. Since the RVOT is an anterior structure inside the thorax, the horizontal axis will be posterior (negative V1, usually with a QS morphology).[25] If the ventricular depolarization axis goes leftwards (positive DI), the origin focus might be in the posterior RVOT, whereas if the axis deviates to the right (negative DI), the focus is usually more anterior *(Figure 7)*.[25,26] Arrhythmias from the septal wall of the RVOT usually show an earlier transition (V3-V4). It is rather unusual that RVOT PVC's have a transition before V2.

Left superior foci

The left superior foci, also known as the «left ventricle outflow tract» (LVOT), can be located in structures such as the aortic cusps (aortic valve sinuses), the mitro-aortic continuity, the superior basal septum or the left ventricle's summit.[26]

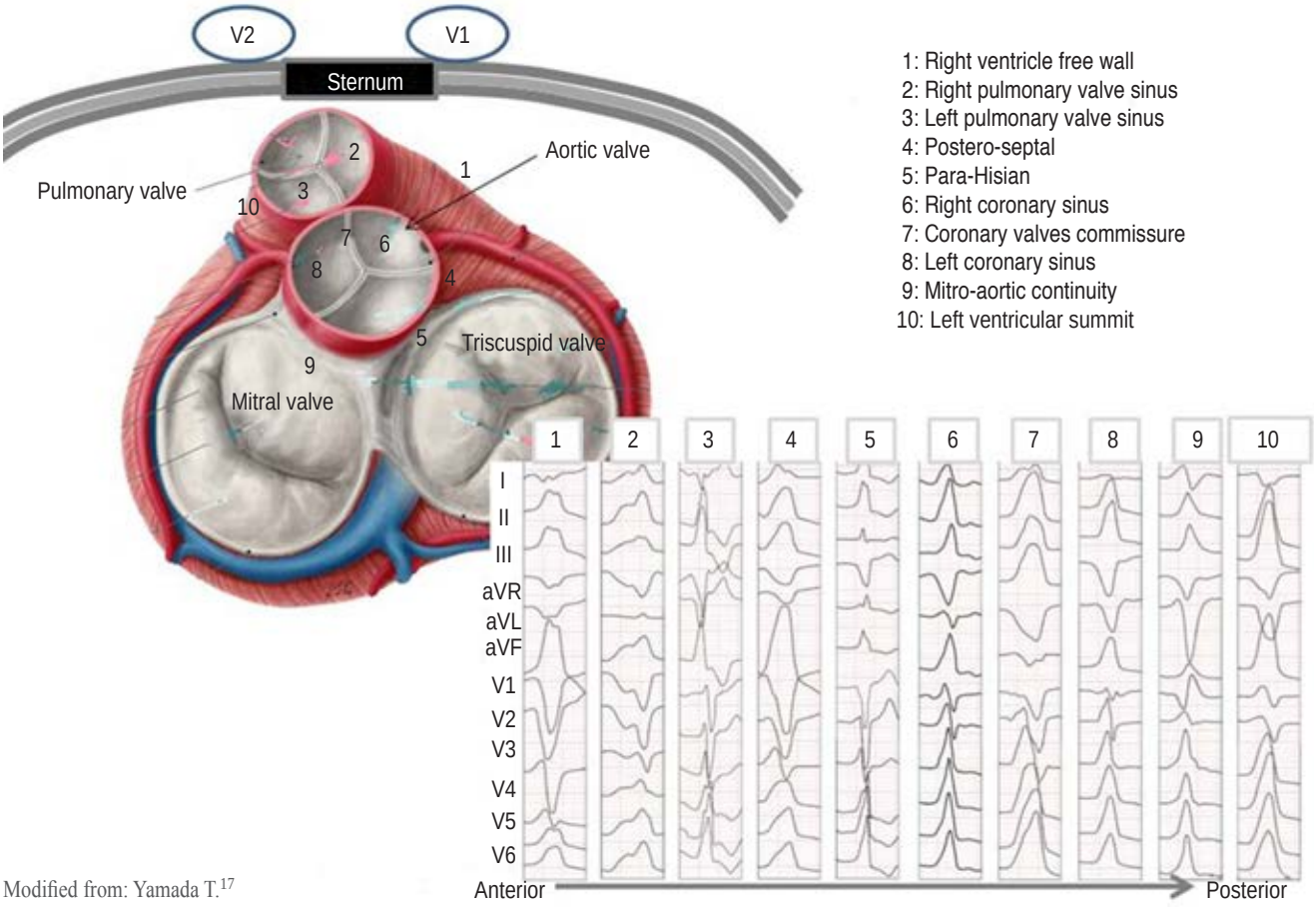
Those structures are responsible for 15 to 25% of the idiopathic ventricular tachycardias.[26,27] The LVOT is an elliptical opening in the left ventricle in which the mitral valve has a posterior and leftwards position when compared to the aortic valve position, which is almost in the centerline of the chest. Both valves share a fibrous band that is the «mitro-aortic continuity» and comprises the anterior leaflet of the mitral valve and the left and non-coronary leaflets of the aortic valve *(Figure 7)*.[25]

Aortic cusps

Premature ventricular contractions might originate from the myocardial tissue within the inter-leaflet commissure and in the base of the aortic sinuses. Despite this possible source of confusion, especially when considering the different approaches for arrhythmia ablation, the term «aortic cusps» is widely accepted as an origin focus for PVC's.

Premature ventricular contractions generated there have an inferior axis that can go right or leftwards and show an LBBB block pattern in 80% of cases. Some ECG characteristics are common to the LVOT and RVOT arrhythmias because of the vicinity of the posterior RVOT to the aorta. The PVC's from the aortic cusps usually have a larger R wave - usually 50% larger than the sinus rhythm QRS –, absence of S wave in V5-V6, and an earlier transition in the precordial leads, when compared with the nearby sites of the RVOT.[24,28] The PVC's from the right coronary sinus might show an rS pattern, with a wide «r» and transition in V3. The QRS complex shows less amplitude in DII and DIII with higher positivity in DII because that sinus is located in a more inferior and rightwards position when compared to the left coronary sinus. The higher the sinus, usually the higher the QRS amplitude.

The left coronary sinus shows an earlier transition, between V1 and V2, with a



Modified from: Yamada T.[17]

Figure 7: Schematic view of the intra-thoracic location of the main structures in the outflow tracts or both ventricles' upper portions. The main depolarization axis will be determined by the anatomic location of the originating structure, its relations with close structures and then, to the position of the surface ECG electrodes (precordial leads) and its relative exit point.

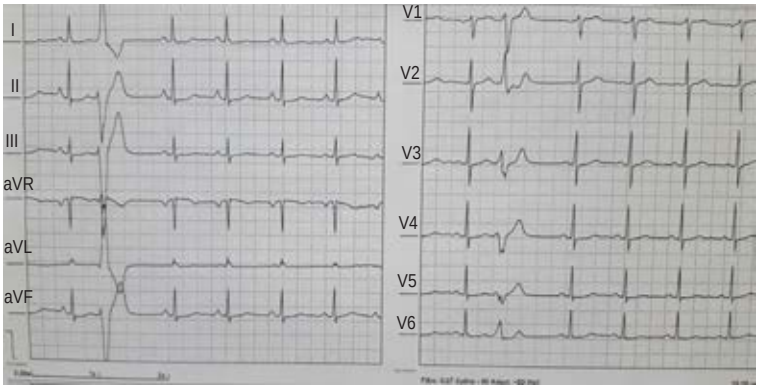


Figure 8: Shows a PVC with LBBB morphology (V1, V6), left superior axis (positive DI and aVL, negative aVF). The PVC's intrinsecoid deflexion is 100 ms. There is a QRS slurring in the precordial leads, with a transition area in V5-V6. The QRS goes from 160 to 180 ms in different leads.

wider R wave (longer than 120 ms in V2) and a higher voltage *(Figure 7)*. This sinus is located in the left lateral and posterior position; thus, the R wave is usually negative in DI and positive in DII, DIII, with a more positive DIII.

Some relatively common idiopathic PVC's originate in the commissure between both coronary sinuses. They show a slurred QS complex in V1, and the transition is in V3.

Mitro-aortic continuity

This region has some Purkinje-like conduction fibers or tissue. The ventricular arrhythmias that arise from here present as monophasic R waves in all the precordial leads, with a RBBB pattern or they might show a qR pattern in V1.[24,29] Others have found that an early R/S wave transition (V2) and an R wave in V3 are frequently seen in PVC's from the anterior mitral-aortic continuity, while the same transition pattern but displaying high R waves in V1 and V3 suggest a middle mitro-aortic continuity origin.[30,31]

Left ventricle summit

This region is the highest portion of the epicardium of the left ventricle and is the origin of up to 12% of the outflow tracts arrhythmias.[32,33] It is the area between the left anterior descending artery, the circumflex artery and an imaginary arch depicted between both vessels at the level of the first septal branch of the anterior descending artery. The PVC's from this point have an RBBB morphology with the left inferior axis, unless the originating focus is displaced towards the anterior interventricular vein, in which case the PVC might have a LBBB morphology owing to the proximity to the interventricular septum. In the precordial leads and the horizontal plane, there is a monophasic R wave pattern similar to the one found in the PVC's from the mitro-aortic continuity. The PVC's that come from the «inaccessible» area of the LV summit show a LBBB with high voltage in DII and DIII and an absence of the s wave in V5 and V6. As in most epicardiac arrhythmias, PVC's usually have a pseudo-delta wave or an initial slurring of the QRS complex *(Figure 7)*.[24,33] Some cases show a «Rupture» pattern, manifested by an LBBB (QS) in the anterior precordial leads, with a less negative V2 and a more negative QS in V3, suggesting that in that specific area, the originating foci might be intra-myocardial rather than epi or endocardiac.[34,35]

Para-Hisian arrhyhmias

These might represent up to 3% of the idiopathic ventricular tachycardias. They show an LBBB morphology with a narrower QRS complex, inferior axis and an early transition, even if V1 frequently displays a QS pattern. The depolarization vector usually follows the normal depolarization direction. Because of its posterior origin, to the right and slightly below the mitral annulus, R waves can be identified in aVL and DI.

Right ventricle's inferior foci

Moderator band

The RV moderator band is a potentially arrhythmogenic structure because it contains a His' right bundle branch fascicle. Owing to its size and insertions, it is a complex element when approaching it for ablation.[36]

Premature Ventricular Contractions arising from it show a LBBB with a superior leftwards

frontal depolarization axis. The PVC's QRS lasts for 135 to 165 ms, somehow narrow, but with a 100 ms intrinsecoid deflection in precordial leads that also show a relatively late transition (usually after V4). Nonetheless, it has to be kept in mind that the transition zone might change according to the exit point within the band itself *(Figure 8)*.[36,37]

Left ventricle's inferior foci

The PVC's that arise from the left ventricle's inferior foci show a RBBB and a superior rightward or leftward axis, according to the relative position of the exit point compared to the interventricular septum (IVS) *(Figure 9)*.

Fascicular arrhythmias

Arrhythmias coming from the anterior fascicle have a RBBB morphology and an inferior rightward axis, while the ones coming from the posterior fascicle will show a RBBB morphology with a leftwards superior axis in the frontal plane.[16] Ventricular tachycardia (VT's) arising from the fascicles usually has a re-entry mechanism. Thus they need a PVC to initiate and they can be verapamil sensitive.[38]

Papillary muscles

Papillary muscles (PM) can be the source of arrhythmias in structurally normal hearts as well as in diseased ones.

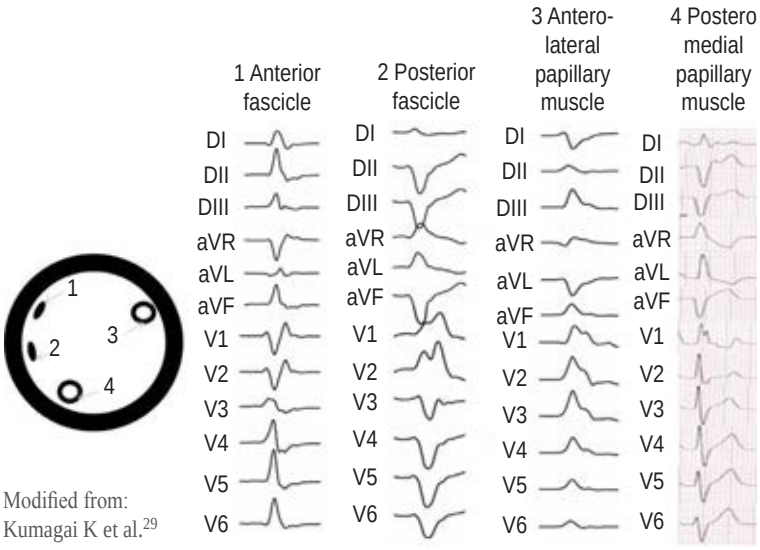
The PVC's originated in the antero-lateral PM usually have a RBBB morphology, a depolarization axis to the right and a transition area between V3 and V5. It is also frequent that they show a depolarization discordance in the inferior leads (positive DII with negative DIII).[39-41]

Arrhythmias from the postero-medial PM will also show a RBBB morphology, a superior axis in the frontal plane and transition between V3 and V5, without the discordance in the inferior leads.

Papillary muscle vs fascicular arrhythmias

Both PVC's show a resembling ECG pattern, but there are important differences.

The papillary muscle PVC's have a wider QRS complex (150 ± 15 ms vs 127 ± 11 ms), and they lack a rsR' activation pattern in V1. They also are monophasic (R or Rs) and they show a q wave in V1.[39,42] Fascicular arrhythmias usually have a q wave in DI or aVL (qR or qRs).



Modified from: Kumagai K et al.[29]

Figure 9: The left circle schematically shows the left ventricle with both fascicles and papillary muscles, numbered according to the ECG tracings. All PVC's have a RBBB morphology (V1), although the ones originating from the anterior fascicle are narrower. Main depolarization vectors are negative in DI in 3 and 4 (The papillary muscles) because those structures are more laterally located when considering the center line. Polarity in aVF is negative in 2, 3 and 4 because they are more inferior structures within the ventricle itself, and thus, they induce an «upwards» depolarization, while the anterior fascicle will do so in a cephalo-caudal direction.

CONCLUSIONS

Premature ventricular contractions might be associated with a higher risk or mortality in subjects with structural heart disease or even without it. They are capable of inducing tachycardiomyopathy, and that is the reason they have become a therapeutic target.

The deductive analysis of the ECG allows to define the approximate origin of the arrhythmia, and thus, to plan optimal treatment strategies in both conditions.

REFERENCES

1. Hastrup J, Goette A, Dobreanu D, Marinskis G, Mabo P, Blomstrom-Lundqvist C. Outpatient evaluation and management of patients with ventricular premature beats and non-sustained ventricular tachycardia. Europace. 2012; 14: 294-296.
2. Bastiaenen R, Batcharov V, Gallagher M. Ventricular automaticity as a predictor of death in ischaemic heart disease. Europace. 2012; 14: 795-803.
3. Torp C, Kay N, Kalman J, Borgreffe M, Della-Bella P, Dickfeld T et al. EHRA/HRS/APHRS Expert consensus on ventricular arrhythmias. Europace. 2014; 16: 1257-1283.
4. Priori S, Blomstrom-Lundqvist C, Mazzanti A, Blom N, Borgreffe M, Camm J et al. 2015 Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Eur Heart J. 2015; 36 (41): 2793-2867. doi: 10.1093/eurheartj/ehv316.
5. Marcus G. Evaluation and management of premature ventricular complexes. Circulation. 2020; 141: 1404-1418.
6. Al-Khatib S, Stevenson W, Ackerman M, Bryant W, Callans D, Curtiss A et al. 2017 AHA/ACC/HRS Guideline for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. Heart Rhythm. 2018; 15 (10): E190-252.
7. Lewis S, Kanakis C, Rosen K, Denes P. Significance of the site of origin of ventricular premature contractions. Am Heart J. 1979; 97 (2): 159-164.
8. Xu W, Li M, Chen M, Yang B, Wang D, Kong W et al. Effect of burden and origin sites of premature ventricular contractions on left ventricular function by 7 day Holter monitor. J Biomed Res. 2015; 29 (6): 465-474.
9. Labadet C. Las extrasístoles ventriculares contraatacan. Rev Argent Cardiol. 2015; 83 (6): 552-554.
10. Luebbert J, Auberson D, Marchlinski F. Premature ventricular complexes in apparently normal hearts. Card Electrophysiol Clin. 2016; 8 (39): 503-514.
11. Ip J, Lerman B. Idiopathic malignant premature ventricular contractions. Trends Cardiovasc Med. 2018; 28 (4): 295-302.
12. Lin C, Chang S, Lin Y, Lo L, Chung F, Chen Y et al. Long term outcome of multiform premature ventricular complexes in structurally normal heart. Int J Cardiol. 2015; 1: 180-185.
13. Lee V, Hemingway H, Harb R, Crake T, Lambiase P. The prognostic significance of premature ventricular complexes in adults without clinically apparent heart disease: a meta-analysis and systematic review. Heart. 2012; 98 (17): 1290-1298.
14. Boas R, Thune J, Pehrson S, Kober L, Nielsen J, Videbaek L. Prevalence and prognostic association of ventricular arrhythmia in non-ischemic heart failure patients: results from the DANISH trial. Europace. 2021; 23 (4): 587-595.
15. Josephson M, Callans D. Using the twelve-lead electrocardiogram to localize the site of origin of ventricular tachycardia. Heart Rhythm. 2005; 2 (4): 443-446.
16. Sodi Pallares D, Medrano G, Bisteni A, Ponce de León J. Electrocardiografía clínica. Análisis deductivo. México: Ediciones del Instituto Nacional de Cardiología de México; 1968.
17. Yamada T. Twelve-lead electrocardiographic localization of idiopathic premature ventricular contraction origin. J Cardiovasc Electrophysiol. 2019; 30 (11): 2603-2617.
18. Tzeis S, ASvestas D, Yen Ho S, Vardas P. Electrocardiographic landmarks of idiopathic ventricular arrhythmia origins. Heart. 2019; 105 (14): 1109-1116.
19. Oebel S, Dinov B, Arya A, Hilbert S, Sommer P, Bollmann A et al. ECG morphology of premature ventricular contractions predicts the presence of myocardial fibrotic substrate on cardiac magnetic resonance imaging in patients undergoing ablation. J Cardiovasc Electrophysiol. 2017; 28 (11): 1316-1323.
20. Asirvatham S. Correlative anatomy for the invasive electrophysiologist: outflow tract and supravalvular arrhythmia. J Cardiovasc Electrophysiol, 2009; 20: 955-968.
21. Prystowsky E, Padanilam B, Joshi S, Fogel R. Ventricular arrhythmias in the absence of structural heart disease. J Am Coll Cardiol. 2012; 20: 1733-1744.
22. Al'Aref S, Ip J, Markowitz S, Liu C, Thomas G, Frenkel D et al. Differentiation of papillary muscle from fascicular and mitral annular ventricular arrhythmias in patients with and without structural heart disease. Circ Arrhythm Electrophysiol. 2015; 8: 616-624.
23. Wissner E, Stevensoon W, Kuck K, Catheter ablation of ventricular tachycardia in ischaemic and non-ischaemic cardiomyopathy, where are we today? A clinical review. Eur Heart J. 2012; 33 (12): 1440-1450.
24. Fernández J, Berruezo A. How to recognize epicardial origin of ventricular tachycardias? Curr Cardiol Reviews. 2014; 10: 246-256.
25. Enríquez A, Baranchuk A, Briceno D, Sáenz LC, García F. How to use the 12-lead ECG to predict the site of origin of idiopathic ventricular arrhythmias. Heart Rhythm. 2019; 16: 1538-1544.
26. Anderson R, Kumar S, Ramathan P, Wong G, Voskoboinik A, Sugumar H et al. Differentiating right and left-sided outflow tract ventricular arrhythmias. Classical ECG signatures and prediction algorithms. Circ Arrhyth, Electrophysiol. 2019; 12: e007392. doi: 10.1161/CIRCEP.119.007392.
27. Ouyang F, Matthew S, Wu S, Kamioka M, Metzner A, Xue Y et al. Ventricular arrhythmias arising from the left ventricular outflow tract below the aortic sinus cusps: Mapping and catheter ablation via transseptal approach and electrocardiographic characteristics. Circ Arrhythm Electrophysiol. 2104; 7: 445-455.
28. Ouyang F, Fotuhi P, Ho S, Hebe J, Volkmer M, Goya M et al. Repetitive monomorphic ventricular tachycardia originating from the aortic cusp: electrocardiographic characterization for guiding catheter ablation. J Am Coll Cardiol. 2002; 39: 500-508.
29. Kumagai K, Fukuda K, Wakayama Y, Sugai Y, Hirose M, Yamagucho N et al. Electrocardiographic characteristics of the variants of idiopathic left ventricular outflow tract ventricular tachyarrhythmias. J Cardiovasc Electrophisiol. 2008; 19: 495-501. doi: 10.1111/j.1540-8167.2007.01085.x.
30. Santos H, Valente B, Cunha P, Portugal G, Ferreira R, Oliveira M. The aortomitral continuity challenge. Ann Clin Case Rep. 2020; 5: 1890.

31. Chen J, Hoff P, Rossvoll O, De Bortoli A, Solheim E, Sun L et al. Ventricular arrhythmias originating from the aortomitral continuity: An uncommon variant of the left ventricular outflow tract ventricular tachycardia. Europace. 2012; 14 (3): 388-395.
32. Yamada T, McElderry H, Doppalapudi H, Okada Y, Murakami Y, Yoshida Y et al. Idiopathic ventricular arrhythmias originating from the left ventricular Summit: Anatomic concepts relevant to ablation. Circ Arrhythm Electrophysiol. 2010; 3: 616-623.
33. Enriquez A, Malavassi F, Sáenz L, Supple G, Santangeli P, Marchlinski F et al. How to map and ablate left ventricular summit arrhythmias. Heart Rhythm. 2017; 14 (1): 141-148.
34. Liao H, Wei W, Tanager K, Miele F, Upadhyay G, Beaser A et al. Left summit arrhythmias with an abrupt V3 transition: anatomy of the aortic interleaflet triangle vantage point. Heart Rhythm. 2021; 18 (1): 10-19.
35. Hayashi T, Santangeli P, Pathak R, Muser D, Liang J, Castro S et al. Outcomes of catheter ablation of idiopathic outflow tract ventricular arrhythmias with an R wave pattern break in lead V2: A distinct clinical entity. J Cardiovasc Electrophysiol. 2017; 28 (5): 504-514.
36. Barber M, Chinitz J, John R. Arrhythmias from the right ventricular moderator band: diagnosis and management. Arrhythm Electrophysiol Rev. 2020; 8 (4): 294-299.
37. Sadek M, Benhayon D, Suredd R, Idiopathic ventricular arrhythmias originating from the moderator band. Electrocardiographic characteristics and treatment by catheter ablation. Heart Rhythm. 2015; 12: 67-75.
38. Komatsu Y, Nogami A, Kurosaki K, Morishima I, Masuda K et al. Fascicular ventricular tachycardia originating from papilary muscles. Purkinje network involvement in the reentrant circuit. Circ Arrhythm Electrophysiol 2016. 2016; 10: e004549. doi: 10.1161/CIRCEP.116.004549.
39. Enríquez A, Supple G, Marchlinski F, García F. How to map and ablate papillary muscle arrhythmias. Heart Rhythm. 2017; 14 (11): 1721-1728.
40. Good E, Desjardins B, Jongnarangsin K, Oral H, Chugh A, Ebinger M, Ventricular arrhythmias originating from a papillary muscle in patients without prior infarction: a comparison with fascicular arrhythmias. Heart Rhythm. 2008; 5: 1530-1537.
41. Kautzner J, Peichl P. Papilary muscle ventricular tachycardia or ectopy: Diagnostic, catheter ablation and the role of intracardiac echocardiography. Arrhythm Electrophysiol Rev. 2019; 8 (1): 65-69.
42. Al'Aref S, Ip J, Markowitz S, Liu C, Thomas G, Frenkel D et al. Differentiation of papillary muscle from fascicular and mitral annular ventricular arrhythmias in patients with and without structural heart disease. Circ Arrhythm Electrophysiol. 2015; 8: 616-624.

Funding: The authors declare that they received No funding for the present work.

Declaration of interests: The authors declare no conflict of interest.

Correspondence:
Dr. Enrique Asensio Lafuente
E-mail: easensiol@gmail.com