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Premature ventricular contraction with short coupling interval: Its significance

Extrasístoles ventriculares con intervalo corto de acoplamiento: Su trascendencia

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The premature ventricular contractions (PVC) are very common, both in healthy and sick people, with a range between benignity and malignancy [triggering malignant ventricular arrhythmias (MVA)], and it has been associated with increased sudden and total mortality.

Within the many premonitory electrical signs of MVA and sudden death (SD), the PVCs are the most important, frequent and argued ones. Harmless or harmful? To treat it or not? The importance of ventricular ectopia and its causal relationship with the induction of MVA has been recognized for many years, but there is a broad spectrum in terms of symptoms and presence or absence of structural cardiac diseases (which can cause, aggravate or be the result of these processes).

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As many PVCs trigger these arrhythmias, it was considered that by decreasing their number, before with antiarrhythmic drugs (AAD), the risk of SD would also decrease[1,2]. In 1986, Josephson[3] predicted that the removal of PVC would fail to increase survival. The CAST (Cardiac Arrhythmia Suppression Trial)[4] confirmed it three years later, the previous ideas were revolutionized and the worst of times for the AAD came, for their own arrhythmogenicity, and the emergence of the implantable cardioverter-defibrillator (ICD), as well as a very successful healing process, such as the ablation of arrhythmogenic substrates[5-7]. Lindsay[8] exposed that the frequency and complexity of ventricular ectopia relates to the risk of MVA, but they are not good predictors for individual patients.

In the past it was thought that the PVCs should be eliminated, then they lost "importance" and today it is known that many are harmless, but some can trigger MVA; hence, there may not be a strong response: they are not all treated but in some cases it is necessary to do so, and with what?

We can ask ourselves: why patients with ventricular ectopia do not have ventricular fibrillation (VF)? Why the different susceptibilities? Why a patient with PVC has a FV, or not, at different times? Are there not recognized autonomic, metabolic, ionic and electrolytic factors? Some fibrillate and others at risk do not, is there an antifibrillatory reserve?

PVCs are between harmful and harmless, between benign or being potentially proarrhytmogenic with a broad spectrum in terms of how to address them, from abstention to drug treatment, ablation and ICD in cases of MVA[9]. It is necessary an integrated vision at the crossroad of treating them or not, not only because of their relationship with arrhythmic mortality but to prevent a heart disease, that could be reversible and behave as a modifiable risk factor[10].

For several reasons it is difficult to record VF episodes and to see how they initiate: failing to save a patient from a cardiac arrest, not witnessed out-of-hospital events, self-limiting episodes, such an urgency that there is no time for taking the electric graphic and proceed to the patient's recovery, wasting the opportunity to record what is happening.

PVCs with short coupling interval (PVCSCI) are electrical predictors and may trigger MVA and episodes of SD in patients with and without structural heart disease. To a lesser degree of coupling of these contractions, worse is the prognosis; therefore, it is one important factor for the risk stratification, but by no means is the only one.

The conflict is presented from the very beginning: what is a short coupling interval? Nogami[11] points out the figure 245±28 ms; Chinushi[12], of 280-300 ms; Callans[1], of 300 ms. Viskin[13] defines a variant with ultrashort coupling interval (ascending branch and peak in T) in the idiopathic VF, in the Brugada syndrome and myocardial infarction; relatively short, if located in the descending branch of the T. The place of the PVC is considered regarding the ventricular repolarization and thus avoiding the variability of the measures when there are changes in the heart rate and other factors.

In some cases, there may be more than one location for the PVC at different times. Nevertheless, the smaller the coupling interval, the worse the prognosis.

Let us see the PVC as trigger for MVA: how many possibilities? a) Many contractions originate MVA or not, b) isolated PVC produce or not MVA c) there are no MVA without triggering contractions.

When evaluating the PVC, there are considered other factors, besides the coupling interval: the person, age, hemodynamic repercussion, structural heart disease or not, left ventricle ejection fraction, transient causes by metabolic or electrolytic disorders, disease of other organs and systems, previous heart rate, site of origin of PVC, density in 24 hours (how many are too many?), uniform morphology or not, width, trigger episodes [ventricular repetitive responses, burst, RR interval, ventricular tachycardia (VT) sustained or not], duration of extrasystolic QRS, QT interval, amplitude of the T, evolution of the disease, type of ion channel if necessary, and use of AAD. There can exist temporal variability of some of these factors[8,14,15].

How to differentiate the benign from the arrhythmogenic in the PVC? In order to decide whether treat them or not, there should be an integrated vision, not to rely just on the aspect. Many remain untreated but if they are, with what? AAD or their deletion, pacemakers, ablation, ICD if it is a MVA, or associated treatments.

The truth is that PVC can trigger or not a VF (isolated episodes or electric storm), in which cases it is important to know what happens immediately before the episode, including hours earlier.

The role of the His-Purkinje system has been recognized in the genesis of MVA, where PVCSCI tend to origin, involved in reentrant phenomena (Purkinje-muscle, Purkinje antidromic-circuit or muscle-Purkije, orthodromic circuit), and ectopic episodes, as triggers of MVA; its ablation would eliminate the trigger element. They participate not only at the origin of these arrhythmias but also in their permanence (VF or polymorphic VT) with some are as more susceptible than others[11,16-18].

The PVCs of Purkinje of the right or left side are characterized by lower duration of the QRS and short coupling intervals. In 2002, Haïssaguerre[5,19] removed them by ablation in patients with idiopathic VF, then in the syndromes of long QT and Brugada (VF, poly-

morphic VT, electric storm). This procedure has also been used in the ischemic heart disease, cardiomyopathies, catecholaminergic polymorphic VT, early repolarization syndrome and others. It is an important therapeutic option although the treatment will be medical if there is no predominant morphology. It is interesting that even without PVCs, mapping the substrate and their removal may be effective in some circumstances, as in the Brugada syndrome[20-23].

ICD prevents death but not the event, that is why the ablation of triggers is added, that does not replace the device in the secondary or primary prevention of SD, but decreases the number of discharges[11,15-18,24,25].

With the ablation of trigger PVC of MVA, a similar reasoning to its removal with AAD in the past should be follow (if they do not trigger MVA), because the method is not exempt of risks nor applicable to all VF, there can also be recurrence or new outbreaks[14].

PVCs are not transcendental concerning the arrhythmic mortality. If frequent, they can lead to a reversible cardiomyopathy and left ventricular dysfunction (it would a modifiable risk factor). Several factors influence this last idea: underlying disease, left ventricular function, comorbidity, alterations of the ion channels, the degree of dyssynchrony, increasing the dispersion and the duration of the action potential, and other dependent of the PVC (density, QRS duration, pleomorphism, origin, coupling, interpolated character, number of focal points). The elimination of PVC by ablation with radiofrequency can improve the ventricular function, though in some occasions there is no knowledge about what was the first, if the PVC led to dysfunction or vice versa. Anyway, it results a modifiable risk factor for AAD or radio frequency[14,26-31]. It is about to identify and modify the reversible triggers that, when eliminated, can restore the cardiac function to normal.

PVC can result from cardiac or systemic diseases (ischemic or non-ischemic cardiomyopathies, inflammatory diseases, valvular, ventricular hypertrophy, congenital heart disease), in which case, the correspondent measures must be apply.

It is necessary to establish the value of the PVC as predictor and trigger of MVA, according to the precocity of their coupling interval (electrocardiographic parameter). The PVCSCI may have a role in the onset of torsade de pointes (helicoidal tachycardia) and VF; they are observed in several entities and episodes of SD, in patients with or without structural heart disease (idiopathic VF; J wave syndromes, Brugada, short and long QT, catecholaminergic polymorphic VT; variant torsion with short coupling)[1,32,33]. It has been observed that the same PVCSCI that triggers the MVA, do not do it in other moments, or only gives place to ventricular repetitive responses, which gives an idea of the variable importance of this predictor, although the degree of precocity of the PVC would have a role in prognosis of these patients.

It is concluded that: the PVCSCI is a premonitory electrical signal trigger of MVA, SD and syncope in patients with and without structural heart disease. The most frequent arrhythmias are the VF and torsade de pointes. A lesser degree of coupling (ultrashort, ascending branch and peak of the T wave and top), greater danger of MVA. The short coupling interval of the PVC is one of the important factors for risk stratification, but not the only one. The therapeutic measures are variable.

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