Idiopathic VPC: Distribution Of FOCI And Tips Of Ablation

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Abstract

Idiopathic Ventricular Premature Contraction (VPC) is currently more routinely referred for electrophysiology evaluation. Usually it carries a good prognosis but, when symptomatic or suspected to produce ventricular dysfunction, will require treatment. Nowadays, RF ablation has great advantages over antiarrhythmic drugs. Classically the outflow tract (right or left), with the typical inferior axis with left (eventually right) bundle branch block like ECG morphology, is considered the most frequent site of origin for idiopathic VPC, but with the widespread of EP procedures and advancement of technology making possible to map and ablate difficult locations, it is possible to see a growing and changing population referred for idiopathic VPC ablation, displaying that, almost any region of the heart may be source of this kind of arrhythmia that can be successfully treated. A well-planned procedure, with the presumed region of origin settled and employing the current technology and knowledge (tips), will have a high chance of cure.

Introduction

Ventricular premature contraction (VPC) in a apparently healthy young adult with no previous history is not so uncommon, and may cause emotional stress until the accurate diagnosis is done. Described as “idiopathic ventricular arrhythmia”, it is defined as the arrhythmia not related with any detectable structural cardiac disease. It accounts for about 20% of all patients referred for evaluation of ventricular arrhythmias. Most patients are symptomatic and will need treatment; other are asymptomatic but have a high burden of the arrhythmia and may be at risk of developing a reversible form of left ventricular dysfunction (tachycardia-induced dilated cardiomyopathy) and will need treatment as well. Pharmacologic treatment of these arrhythmias, usually with beta-blockers, calcium channel blockers and even amiodarone, has only modest efficacy (around 25 to 50%). Most young patients do not desire long-term medical therapy or are not drug tolerant. Some patients can even feel a worsening of symptoms after medications due to drug induced bradycardia or to an increase in VPC burden. Considering that usually the source of the arrhythmia is unifocal, ablation has undoubtedly some advantages over medical treatment, since it has a high chance of cure. Therefore, broadly speaking, the key decision is if the patient needs or not treatment, if so, the best option is usually the RF ablation.

Distribution of FOCI

Ventricular premature contraction in structural normal heart may arise from any region but the outflow tract VPC / VT is the most common form, known almost as synonymous of idiopathic ventricular arrhythmia; one concern, is to differentiate from initial form of arrhythmogenic right ventricular cardiomyopathy/dysplasia. The reason these arrhythmias come most frequently from outflow is still not clear. The typical electrocardiogram features is the inferior axis and left branch block morphology. For a long time it has been taught that the right ventricular outflow tract (RVOT) accounts for the majority (about 80%) of idiopathic VPCs, however, taken into account the great growing of referred population, the foci location has changed significantly in the last decade. Maybe due to the ability of mapping complex or difficult structures and the widespread of ablation procedures, the kind of patient that is referred for electrophysiology evaluation nowadays has been show a shift to other regions, outside of RVOT. Recently Penela et al. studied 117 patients with outflow tract VPC / VT and found almost the same distribution of foci from left and right side (left: 51%; Right: 49%). A newly and big study from Latchamsetty et al. give a compelling evidence for distribution of foci in current VPC ablations based on 8 international centers, comprising 1,185 patients between 2004 and 2013. They found and described the origin as follow: RVOT: 45%; Aortic Cusps: 15%; Papillary Muscles: 5%; Epicardium: 11%; Other (not specified) origin: 24%. Therefore any area of the heart may be source of idiopathic ectopy.

Key Words:
Idiopathic, Ventricular Premature Contraction, Ablation, Outflow tract.
was the source of origin of 8% of idiopath
ventricular arrhythmias have been described.

The Fascicles of the left bundle branch are classically known as
source of a typical idiopathic VT, but it may arise ectopy as well; it
is ready identified by a narrow QRS and the characteristic ECG mor-
phology of the fascicle of origin: left posterior fascicle with a right
bundle branch block and left axis deviation; left anterior fascicle with
morphology of right bundle branch block and right axis deviation; high
septal fascicle with relatively narrow QRS complex and normal axis.

Epicardial and/or Peri-Vascular are other source of idiopathic
ectopy. Scanavacca et al suggest that these arrhythmias may be accessed
by the venous system or by subxiphoid epicardial mapping, but do not recommend an epicardial approach on
the first procedure for outflow tract arrhythmias; one concern
is the presence of the coronary arteries and phrenic nerves. A de-
lay in the initial time of the QRS complex indicated a epicardial
origin as suggest by the Maximum Deflection Index (MDI).

One important aspect in counseling patients with idiopathic
arrhythmias, essentially outflow tract, is the challenge of pre-
dicting the site of origin, therefore discussion about treatments
options and intervention planning. There are several ECG crite-
ria that try to locate the focus (Figure 1), some of them are based
on complex process analysis or formulas. The inspection of QRS
morphology of the VPC compared with the normal sinus rhythm
(“the V2 transition ratio”) is practical and helpful, Figure 2.

Beyond morphologic criteria, others ECG characteristics and clin-
cal aspects may be valuable in arrhythmia discrimination. Recently
Bradfield et al. demonstrated that arrhythmias that arise from sinus
of Valsalva and great cardiac vein have a highly variable coupling
interval with the preceding normal QRS, probably due to a lack of
electronic coupling with the surrounding myocardial. PVC originating
from RV or LV myocardial have a relative fixed coupling interval
compare with PVC from sinus of Valsalva and great cardiac vein; a
pronounced variability in couple interval (Delta > 60ms) helps dis-
criminate the origin of PVC. Another interesting study from Penela
et al. evaluated clinical characteristics in patients with outflow tract
arrhythmias; they showed that the presence of hypertension, male
gender and age > 50 years were independent predictive of LV out-
flow tract origin and a score with these three variables was proposed.

Tips of Ablation

Different from reentry ventricular tachycardias, which can be usu-
ally reproduced with programmed ventricular stimulation techniques,
ventricular premature beats has the disadvantage of being less easily

Outflow Tract is still the most common site and is classified
as one group apart. This area include the following structures:
right ventricular outflow tract (RVOT), left ventricular outflow
tract (LVOT), the aortic sinuses of Valsalva, the area of aortomi-
tral continuity, the superior basal septum near the His bundle, the
pulmonary artery, and the epicardial surface of the outflow tracts.
Although some clinical differences may exist, these arrhythmias
have similar characteristics and share the same behavior. Actually
the right and left ventricular outflow tracts are in close rela-
tionship and this explains the difficult of differentiating them by
ECG criterion and the phenomenon of shifting, when 2 discrete
morphologies are seeing changing during the catheter ablation.

Tricuspid Anulus was the source of origin of 8% of idiopath-
ic VPC/VT as described by Tada et al.; they show that the ma-
jority (74%) of them originated from the septum portion. Yam-
da et al. presented a case report where they did ablation of
tricuspid annulus VPC using a halo-type catheter to help mapping.

Mitral annulus ventricular arrhythmias have been described
as 5% of idiopathic arrhythmias in a large series; the authors
showed that in all patients an S-wave was present in lead V6.

Papillary Muscles has a rich network of Purkinje fibers, these structures
are accountable for 4.2% of idiopathic VT/PVC in one series;
it may arise in normal hearts but, when compared with fascicular
and mitral annular PVC, it seems that these arrhythmias occur in old-
er patients and are often related with the presence of coronary artery
disease and left ventricular dysfunction; they also showed that pap-
illary ectopy has a wider QRS duration than fascicular arrhythmias.

The Epicardial surface of the posterior left ventricle.

Tips of Ablation

Determined from reentry ventricular tachycardias, which can be usu-
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ventricular premature beats has the disadvantage of being less easily

reproduced. All antiarrhythmic medications should be discontinued for at least five half-lives before the ablation. Not uncommonly, even in patients with very frequent clinical arrhythmia, it may be necessary to perform the procedure in the absence of ectopic beats. It is helpful to see in the Holter if the arrhythmia is more frequent during the sleep or vigil. In the last case a deep sedation must be avoided.

One of the key point of ablation of idiopathic VPC is to have the arrhythmia (spontaneously) in the moment of the procedure or to identify a protocol that induce the arrhythmia; It is really important for mapping and for having an end point during the procedure. There is no unique maneuver or protocol that works satisfactorily for ever case.

Despite being unpredictable, it is usually possible to trigger the ectopies appearance, so we always try to perform the procedure on the day that the patient was scheduled, but many researchers suspend the procedure if there is no spontaneous arrhythmia.

At the beginning, in the case there are no arrhythmias, with the patient fully monitored and awakened, some ventilation maneuvers (depth breath or Valsalva) may provoke VPC allowing to get recordings (that must be saved) for pace mapping. After sedation, ventilation changes induced by the anesthesiologist, like short hypoxia periods, may also reproduce the arrhythmia. After placing the catheters, there is a good chance that some atrial or ventricular stimulation protocols with different cycle length or extra-stimuli, may reproduce the arrhythmia during the stimulation or after stopping, during the pause. Ventricular stimulation must be attempted with programmed stimulation or burst. Some people try isoproterenol (despite epinephrine seems to be better), but in our experience it may be trick; some times with the sinus rate increasing it may eliminate any sporadic ectopy that could be present; also, with the vigorous myocardial contraction due to isoproterenol, the manipulation of the catheter (and pace mapping) is more difficult or even risky. It seems that isoproterenol works better when it is given as small bolus than continuously (progressive), but in our experience it may be trick; some times with the sinus rate increasing it may eliminate any sporadic ectopy that could be present; also, with the vigorous myocardial contraction due to isoproterenol, the manipulation of the catheter (and pace mapping) is more difficult or even risky. It seems that isoproterenol works better when it is given as small bolus than continuously (progressive), because there is a better chance that the arrhythmia is induced after the peak effect of the drug, when the sinus rate is slowing down.

**Vagal Stimulation**

Another very interesting issue is autonomic stimulation to promote cardiac reflex for triggering some arrhythmias.17,18 This can be important to achieve the morphology in 12 lead ECG of a PVC that eventually becomes completely absent at the time of the procedure in order to proceed with the pace mapping. We are studying the potentiality of vagal stimulation performed by placing a catheter inside the internal jugular, up to the superior wisdom tooth level by using even the RF catheter temporarily detached from the RF generator and connected to a neurostimulator, Figure 3.

In this place, it is usually easy to get an intense vagal stimulation that causes transient asystole, Figure 4-A. Soon after the vagal effect, a reflex sympathetic response occurs usually causing the appearance of PVC, Figure 4-B. Depending on the case, that may be better than the isoproterenol infusion because it triggers the sympathetic response by natural paths. Paradoxically, by causing an important heart rate increase, the isoproterenol may prevent the PVC appearance just because of the diastolic extent reduction.

**Pace Mapping**

It is a great aid technique pacing point-to-point areas suspected from the ECG PVC morphology. The main objective is to reproduce the morphology of spontaneous PVC in 12-lead ECG through electrical stimulation of a certain point. This step can be accomplished by direct subjective comparison of the operator or aided with automated computerized systems, Figure 5. A major advantage of this method is that it can be performed in the absence of the arrhythmia since the PVC morphology in a 12-lead ECG technically identical to the ablation ECG, can be compared. An important drawback is that the pace mapping identifies the starting point of the PVC appearance in the myocardium and not necessarily the actual focal origin.19 Despite being not totally necessary, the use of three-dimensional electroanatomical mapping system and, recently, the rotational angiography may be of great value for mapping assistance.20

**Computer Aided Pace Mapping**

Other area of great interest is to improve the performance and the quality of the pace mapping technique, making it faster and less de-
dependent of the interpretation of the operator. Considering that sometimes we do not have the PVC frequency as desired, pace mapping is the main approach being the technique that allows getting close of the site of origin and save time. There are some reports dealing with automatic template for computerized interpretation of pace mapping.

Ablation of Valsalva Sinus PVCs

A very delicate situation is when PVC is located in the sinus of Valsalva because of the risk of coronary lesion. Only the non-coronary sinus does not originate PVCs as it relates only with the fibrous skeleton of the heart. That means, whenever there are PVCs of this location they are surely originated from the left or right coronary sinus, whose ablations should deal with the risk of coronary injury, Figure 6. One fundamental tip for ablation in the right or left Valsalva coronary sinus is to perform the coronariography immediately before the RF release. A simple and practical way we have proposed is to place the contrast dye in the irrigation system of the ablation catheter. After positioning the RF catheter in the most appropriate mapping place, radiographic contrast product is directly injected through the irrigation system. This should be repeated immediately before each new ablation case the catheter position has been changed. If the contrast shows that we are far from coronary ostium we can ablate without risk. Conversely, if it is found that we are very close to the coronary ostium the ablation must be suspended (Figure 7).

Activation Mapping

It can be performed directly with the RF catheter or through the electroanatomic mapping system. It is certainly the key element to the success of ablation. Generally, the potential that indicate the position of the arrhythmogenic focus with great chance of success precede the QRS surface of the PVC onset in 10 to 60ms, Figure 4-B. The morphology of these potentials is also of great value especially the QS pattern in unipolar recording of the distal pole or the reverse potentials comparing two distal dipoles of the ablation catheter.

References

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