Localization of Ventricular Arrhythmias for Catheter Ablation

The Role of Surface Electrocardiogram

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KEYWORDS

• Ventricular arrhythmias • Site of origin • Electrocardiography • Catheter ablation
• Ventricular tachycardia • Idiopathic ventricular arrhythmias • Structural heart disease

KEY POINTS

- Preprocedural analysis of 12-lead Electrocardiogram (ECG) of a clinical arrhythmia is of pivotal importance to predict the site of origin either in case of idiopathic ventricular arrhythmias (VAs) or in patients with structural heart disease (SHD).
- The main mechanism underlying idiopathic VAs is generally an abnormal and rapid activation of a focal area of normal myocardium.
- VAs in SHD generally result from a scar-related reentry mechanism.
- ECG analysis might help tailoring the ablation strategy to optimize procedural duration, increase the probability of success, and recognize and prevent risks and complications.

INTRODUCTION

Over the past few decades, significant advances have been made in the diagnosis and management of ventricular tachycardia (VT). Antiarrhythmic drug therapy (eg, amiodarone and β-blockers) may significantly reduce the risk of ventricular arrhythmias (VAs) but carries a significant risk of side effects, which frequently lead to drug discontinuation.[1] Implantable cardioverter defibrillators (ICDs) are the mainstay of treatment of primary and secondary prevention of sudden cardiac death (SCD).[2,3] ICD therapies,

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Abbreviations

AMC	Aortomitral continuity
ASOV	Aortic sinuses of Valsalva
AV	Atrioventricular
BBR	Bundle branch reentry
ECG	electrocardiogram
ICD	Implantable cardioverter defibrillator
LBBB	Left bundle branch block
LCC	Left coronary cusp
LV	Left ventricular
MA	Mitral annulus
MDI	Maximum deflection index
MI	Myocardial infarction
NCC	Noncoronary cusp
OT	Outflow tract
PA	Pulmonary artery
PPM	Papillary muscle
RBBB	Right bundle branch block
RCC	Right coronary cusp
RV	Right ventricular
SCD	Sudden cardiac death
SHD	Structural heart disease
SOO	Site of origin
TA	Tricuspid annulus
VA	Ventricular arrhythmia
VT	Ventricular tachycardia

either antitachycardia pacing and/or shock, can effectively terminate potentially life-threatening VAs.[2,3] In selected patients, a combined approach based on antiarrhythmic drug therapy and ICD implantation may reduce the incidence of VAs and improve survival. In addition to antiarrhythmic drug side effects, however, recurrent ICD therapies may correlate with worsening quality-of-life scores and increase mortality rates.[4] Catheter ablation has emerged as an effective treatment of recurrent VTs, either idiopathic or secondary to structural heart disease (SHD). Idiopathic VTs account for approximately 10% of patients with documented VTs and occur in the absence of cardiac structural abnormalities or ion channellopathies.[5] In this case, specific anatomic structures (eg, ventricular outflow tracts [OTs], atrioventricular [AV] annuli, and papillary muscles [PPMs]) are involved in the development of arrhythmias. Catheter ablation of idiopathic VTs is an effective procedure, whose success rate ranges between 80% and 100%[6–8] and is relatively safe; nevertheless, anatomic obstacles (eg, coronary arteries, AV conduction system, and epicardial fat pads) might make the procedure more challenging in some cases.

In a majority of patients with recurrent episodes of sustained VT, it is possible to identify a structural arrhythmogenic substrate, which can be the result of cardiomyocyte replacement and scar formation. VT ablation has been demonstrated to be superior to antiarrhythmic drugs in patients suffering from VAs due to SHD; additionally, substrate modification seems superior to standard ablation in reducing the risk of VA recurrences and all-cause mortality.[9,10]

Preprocedural analysis of 12-lead ECG of the clinical VA is of pivotal importance to predict the arrhythmia site of origin (SOO) either in cases of idiopathic VAs or in patients with SHD. Although ECG interpretation might be complicated by factors like chest wall deformity, metabolic and drug effects, and presence and distribution of scar, a detailed analysis of 12-lead ECG can be a valuable mapping tool and provide useful information in localizing the VA SOO. This might help tailor the ablation strategy to optimize procedural duration, increase the probability of success, and recognize and prevent risks and complications. The aim of this article is to review the ECG features of both idiopathic and scar-related VAs and discuss their potential implications for optimizing the ablation strategy.

LOCALIZATION OF IDIOPATHIC VENTRICULAR ARRHYTHMIAS: GENERAL CONSIDERATIONS

Anatomic Considerations

OT VAs are the most common form of idiopathic VAs, accounting for approximately 10% of all VAs. The OT has a complex anatomy, and its understanding is crucial to be able to interpret correctly the 12-lead ECG. The OT centers on the 2 semilunar valves, pulmonary valve and aortic valve, and includes the right ventricular OT (RVOT), the pulmonary artery (PA), the para-Hisian region, the left ventricular OT (LVOT), the mitral annulus (MA), aortic root/sinuses of Valsalva (ASOV), and the left ventricular (LV) epicardium above (LV summit). The RVOT is a conical structure bordered superiorly by the pulmonic valve and inferiorly by the right ventricular (RV) inflow tract and the top of the tricuspid valve. The RVOT can be divided into 3 segments: proximal, mid, and distal. The proximal segment lies to the right of the LVOT, beginning at the superior margin of the tricuspid annulus (TA). The mid and distal segments lie anterior and leftwards to the aortic root, wrapping around the LVOT. The posteroseptal aspect of the RVOT is adjacent to the right coronary cusp (RCC); the anteroseptal aspect is adjacent to the LV epicardium and the left anterior descending coronary and anterior to the RCC and a portion of the left coronary cusp (LCC). The lateral aspect of the RVOT is the RV free wall. The pulmonary valve lies above the ventricular septum. In young patients, the valve is parallel to the aortic valve, which