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Review

Idiopathic ventricular arrhythmias
Relevance to the anatomy, diagnosis and treatment

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ABSTRACT

Idiopathic ventricular arrhythmias (VAs) are ventricular tachycardias (VTs) or premature ventricular contractions (PVCs) whose mechanisms are not related to myocardial scar. Idiopathic VAs occur most commonly without structural heart disease, but can occur with structural heart disease. Imaging tests, such as echocardiography, nuclear test, and cardiac magnetic resonance imaging, are helpful for excluding any association of an idiopathic VA occurrence with myocardial scar. Since catheter ablation emerged, the sites of idiopathic VA origins, commonly endocardial but sometimes epicardial, have been increasingly recognized. Idiopathic VAs usually originate from specific anatomical structures, and exhibit characteristic electrocardiograms based on their anatomical background. Idiopathic VAs are basically benign, but they require medical treatment or catheter ablation when idiopathic VAs are symptomatic, incessant, or produce left ventricular dysfunction. This review describes the up-to-date information on the prevalence of idiopathic VA origins relevant to the anatomy, and diagnosis, and treatment of idiopathic VAs.

Contents

Introduction

Idiopathic ventricular arrhythmias (IVAs) are ventricular tachycardias (VTs) or premature ventricular contractions (PVCs) whose mechanisms are not related to myocardial scar. IVAs occur commonly without structural heart disease (SHD), but can occur with SHD [1,2]. Classically, VTs originating from the right ventricular outflow tract (RVOT) and left posterior fascicle are well known as IVAs. However, since catheter ablation emerged, IVAs originating from other endocardial and also epicardial sites have been increasingly recognized (Fig. 1). IVAs usually originate from the specific anatomical structures, and exhibit characteristic electrocardiograms based on their anatomical background. Basically, IVAs are not life threatening, but are often symptomatic and also can cause tachycardia-induced cardiomyopathy [3,4]. Therefore, it is important for cardiologists to update their knowledge about IVAs. This review describes the up-to-date information on the prevalence of IVA origins relevant to the anatomy, and diagnosis, and treatment of IVAs.

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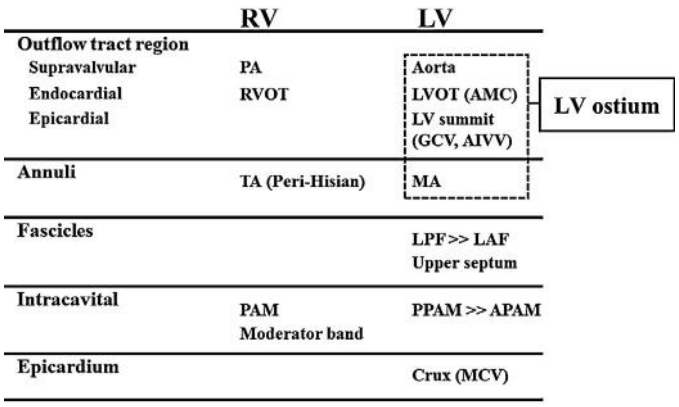

Fig. 1. Idiopathic ventricular arrhythmia origins. AIVV, anterior inter-ventricular vein; AMC, aorto-mitral continuity; APAM, antero-lateral papillary muscle; GCV, great cardiac vein; LAF, left anterior fascicle; LPF, left posterior fascicle; LV, left ventricle; LVOT, LV outflow tract; MA, mitral annulus; MCV, middle cardiac vein; PA, pulmonary artery; PAM, papillary muscle; PPAM, postero-medial papillary muscle; RV, right ventricle; RVOT, RV outflow tract; TA, tricuspid annulus.
Source: This figure was modified from Yamada [9], with permission.

Prevalence of IVA origins relevant to the anatomy

The sites of IVA origins have been identified by electrophysiological mapping and confirmed by successful catheter ablation. The most common site of IVA origins is the ventricular outflow tract [1,5]. IVAs originate more often from the RVOT than the left ventricular outflow tract (LVOT). In the RVOT, the septum is a more common site of IVA origins than the free wall. The most common site of IVA origins in the LVOT is the aortic root followed by the sites underneath the aortic coronary cusps (ACCs) (Fig. 2A) [6,7]. In particular, the site underneath the left coronary cusp (LCC) is termed the aorto-mital continuity (AMC). The mitral annulus (MA) is also one of the major sites of IVA origins [8,9]. The antero-medial aspect of the MA may overlap with the AMC. Anatomically, the aortic and mitral valves are in direct apposition and attach to the elliptical opening at the base of the left ventricle (LV) known as the LV ostium [10,11] (Fig. 2A). Because there is no myocardium between the aortic and mitral valves (fibrous trigone), most idiopathic LV VAs can originate from along the LV ostium. The LV myocardium comes in direct contact with the aorta at the base of the ACCs (Fig. 2A). When IVAs arise from the most superior portion of the LV ostium (the aortic sinus of Valsalva), they can be ablated within the base of the ACCs. It has been reported that some IVAs can be ablated from the junction (commissure) between the left and right coronary cusps (L-RCC) [12]. In these VAs, catheter ablation from underneath the ACCs is often required for their elimination. Anatomically, the superior end of the LV myocardium makes a semicircular attachment to the aortic root at the bottom of the right and left coronary cusps. However, because of the semilunar nature of the attachments of the aortic valvular cusps, the superior end of the LV myocardium is located underneath the aortic valves at the L-RCC (Fig. 2A). Therefore, IVAs that can be ablated at the L-RCC should be classified into the same group as the VAs that can be ablated within the ACCs. In this setting, these IVAs may be defined as IVAs arising from the aortic root [7]. It has been reported that IVAs can rarely be ablated from within the non-coronary cusp of the aorta (NCC) [7,13,14]. Spatially, the aortic root occupies a central location within the heart, with the NCC anterior and superior to the paraseptal region of the left and right atria close to the superior atrioventricular junctions (Fig. 2B) [11]. In healthy human hearts, the NCC is adjacent to the atrial myocardium on the epicardial aspect and the NCC does not directly come in contact with the ventricular myocardium (Fig. 2B). Indeed, atrial tachycardias can be ablated from within the NCC. However, the clinical observation that a non-coronary sinus of Valsalva aneurysm can rupture into the right ventricle (RV) as well as the right atrium supports the assumption that the NCC may be attached to the ventricular myocardium where IVAs can arise from [13]. IVAs can arise from the pulmonary artery with a ventricular myocardial extension from the RVOT [15]. It should be noted that ventricular myocardial extensions never occur in the aorta [11].

IVAs can originate from the atrioventricular annuli including the MA [8,9] and tricuspid annulus (TA) [16]. IVAs originating from the MA and TA account for 5% and 8% of all IVAs, respectively. MA VAs can originate from any of the regions along the MA, but the antero-lateral and postero-septal aspects of the MA are the most common and second most common sites of MA VA origins, respectively [8,9]. TA VAs can originate from any regions along the TA, but more often originate from the septal aspect, especially in the antero-septal or para-Hisian region than the free wall [16].

IVAs can arise from the intra-cavital structures including the papillary muscles (PAMs) [17–21] and moderator band (MB) [22]. PAM VAs account for approximately 7% of patients with IVAs [17–21]. LV PAM VAs are known to arise more commonly from the postero-medial PAM than the antero-lateral PAM [19]. The sites of the PAM VA origins are limited to the base of the PAMs. IVAs can rarely originate from the PMs in the RV [21]. IVAs can arise from all 3 RV PAMs, but half of them arise from the septal PAM [21]. It has been recently reported that the MB, although rarely, can be a source of IVAs including PVCs, VTs, and ventricular fibrillation [22]. Anatomically, the MB is considered to be a part of the septomarginal trabeculation, crossing from the septum to the RV free wall and supporting the anterior PAM of the tricuspid valve (Fig. 3A) [22].

IVAs can arise from the Purkinje network, most commonly from the left posterior fascicle followed by the anterior and septal fascicles [20,23,24]. The left anterior fascicle runs along the MA. The peripheral Purkinje network extends to the surface of the PAMs and MB. Therefore, these VAs have to be differentiated from IVAs originating from the PAMs, MB, and atrioventricular annuli.

IVAs arise commonly from the endocardial side, but can arise from the epicardial side [25] and rarely from the intramural site [26]. There are two major sites of origin of idiopathic epicardial VAs, such as the crux of the heart [27] and LV summit [28]. Anatomically, the crux of the heart is formed by the junction of the atrioventricular groove and the posterior inter-ventricular groove and corresponds roughly to the junction of the middle cardiac vein and coronary sinus, near the origin of the posterior descending coronary artery (Fig. 2C) [27]. A region of the LV epicardial surface that occupies the most superior portion of the LV has been termed the LV summit by McAlpine (Fig. 2D) [10,28]. The LV summit is bounded by the left anterior descending coronary artery and left circumflex coronary artery. This region near where the great cardiac vein (GCV) ends and the anterior inter-ventricular cardiac vein begins is one of the major sources of epicardial IVAs. The LV summit is bisected by the GCV into an area lateral to this structure that is accessible to epicardial catheter ablation (the *accessible area*) and a superior region that is inaccessible to catheter ablation due to the close proximity of the coronary arteries and thick layer of epicardial fat that overlies the proximal portion of these vessels (the *inaccessible area*) [28]. The prevalence of LV summit VAs has been reported to account for 12% of idiopathic LV VAs. Among these VA origins, 70%, 15%, and 15% of them have been identified within the GCV, accessible area, and inaccessible area, respectively.

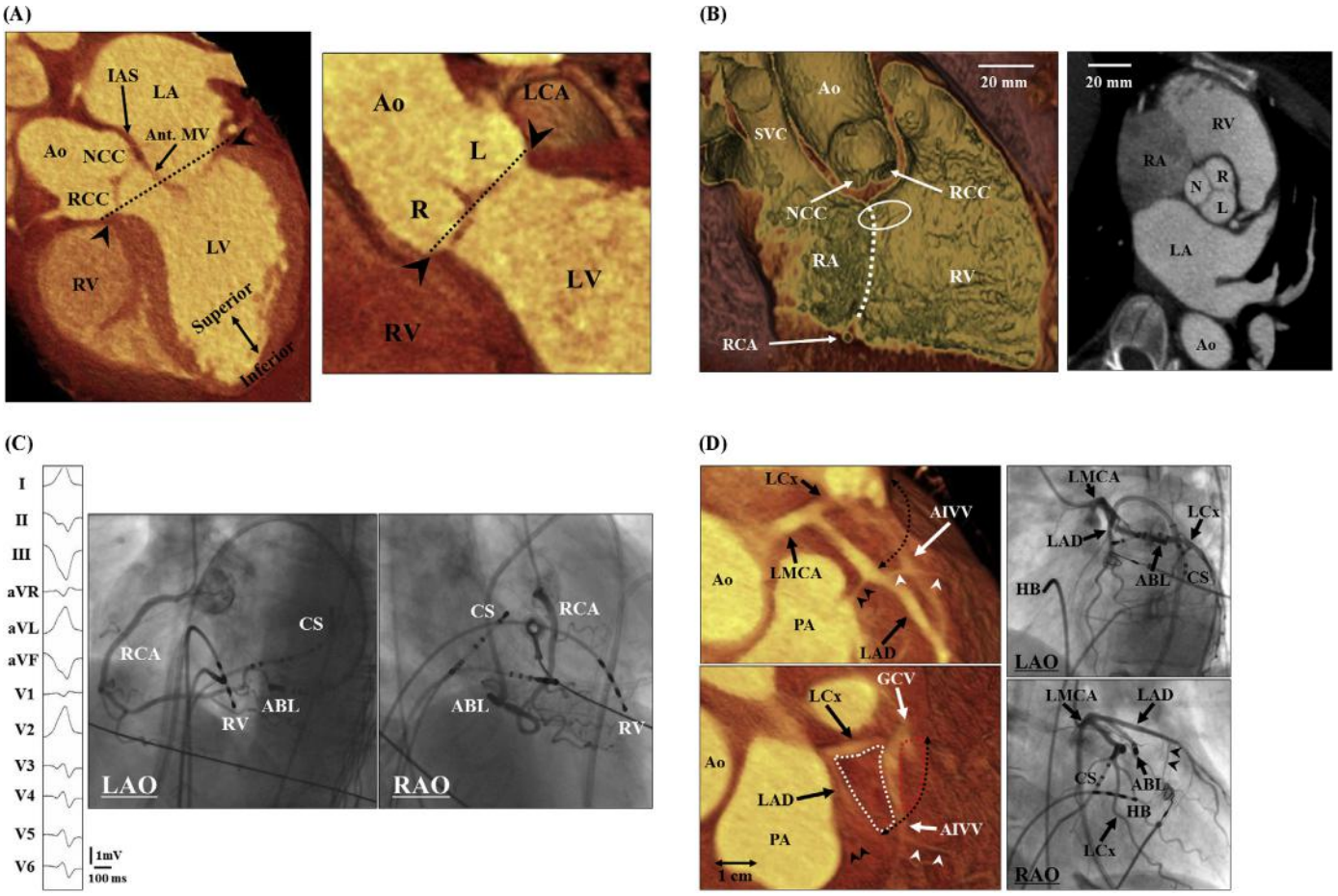

Fig. 2. (A) Two-dimensional computed tomography (CT) images showing the relationships between the ventricular myocardium and aortic sinus cusps. The arrowheads indicate the superior edge of the ventricular myocardium connecting with the left coronary cusp and right coronary cusp (RCC), and the dotted line the ventriculo-arterial junction (the ostium of the left ventricle). Ant., anterior; Ao, aorta; IAS, inter-atrial septum; L, left coronary cusp; LA, left atrium; LCA, left coronary artery; MV, mitral valve; NCC, non-coronary cusp; R, right coronary cusp. This figure was reproduced from Yamada et al. [7] with permission. (B) Two-dimensional (right panel) and three-dimensional (left panel) CT images. The dotted line indicates the tricuspid annulus and solid circle the right ventricular His bundle (HB) region. L, left coronary cusp; N, non-coronary cusp; RA, right atrium; RCA, right coronary artery; SVC, superior vena cava. The other abbreviations are as in the previous figures. This figure was reproduced from Yamada et al. [14] with permission. (C) Twelve-lead electrocardiograms exhibiting the ventricular arrhythmia originating from the crux of the heart (left panel) and fluoroscopic images exhibiting its successful ablation site. ABL, ablation catheter; CS, coronary sinus; LAO, left anterior oblique; RAO, right anterior oblique. The other abbreviations are as in the previous figures. This figure was adapted from Doppalapudi et al. [27], with permission. (D) CT (left panels) and fluoroscopic (right panels) images exhibiting the LV summit. The LV summit was defined based on the fluoroscopy and coronary angiography as the region on the epicardial surface of the LV near the bifurcation of the left main coronary artery that is bounded by an arc (black dotted line) from the left anterior descending coronary artery (LAD) superior to the first septal perforating branch (black arrowheads) and anteriorly to the left circumflex coronary artery (LCx) laterally. The great cardiac vein (GCV) bisects the LV summit into a superior portion surrounded by the white dotted line (the *inaccessible area*) and an inferior portion surrounded by red dotted line (the *accessible area*). The white arrowheads indicate the first diagonal branch of the LAD. LMCA, left main coronary artery. The other abbreviations are as in the previous figures. This figure was reproduced from Yamada et al. [28] with permission.

Diagnosis of IVAs

Imaging

IVAs are defined as VAs originating from healthy ventricular myocardium. Therefore, any association of myocardial scar with an occurrence of VAs has to be excluded for a diagnosis of IVAs. Echocardiography and exercise stress testing are basic exams to demonstrate no evidence of SHD. However, IVAs can occur with SHD. If VAs originate away from the myocardial scar, they are considered idiopathic. Therefore, an imaging study, such as echocardiography, nuclear test, or cardiac magnetic resonance imaging (cMRI), should be performed to locate the site of the scar. Frequent IVAs can cause tachycardia-induced cardiomyopathy. When evidence of myocardial scar is excluded by a nuclear test or cMRI despite a reduced LV function, tachycardia-induced cardiomyopathy is likely to be present.

Electrocardiogram

IVAs usually originate from specific anatomical structures, and exhibit characteristic electrocardiograms (ECGs) based on their anatomical background. In general, the first clue in 12-lead surface ECGs for predicting a site of an IVA origin is a bundle branch block pattern in lead V1. A right bundle branch block (RBBB) pattern clearly suggests an origin in the LV, whereas a left bundle branch block (LBBB) pattern suggests an origin in the RV or interventricular septum. Second, an inferior axis (dominant R waves in leads II, III, and aVF) suggests an origin in the superior aspect of the ventricle, whereas a superior axis suggests an origin in the inferior aspect. A negative QRS polarity in lead I suggests an origin in the LV free wall [2,8], and a QS pattern in lead V6 suggests an origin near the apex (Fig. 3) [2,20]. An R/S wave amplitude ratio >1 in lead V6 suggests an origin in the base (ventricular outflow tract or annuli), whereas an R/S wave amplitude ratio <1 suggests an origin in the

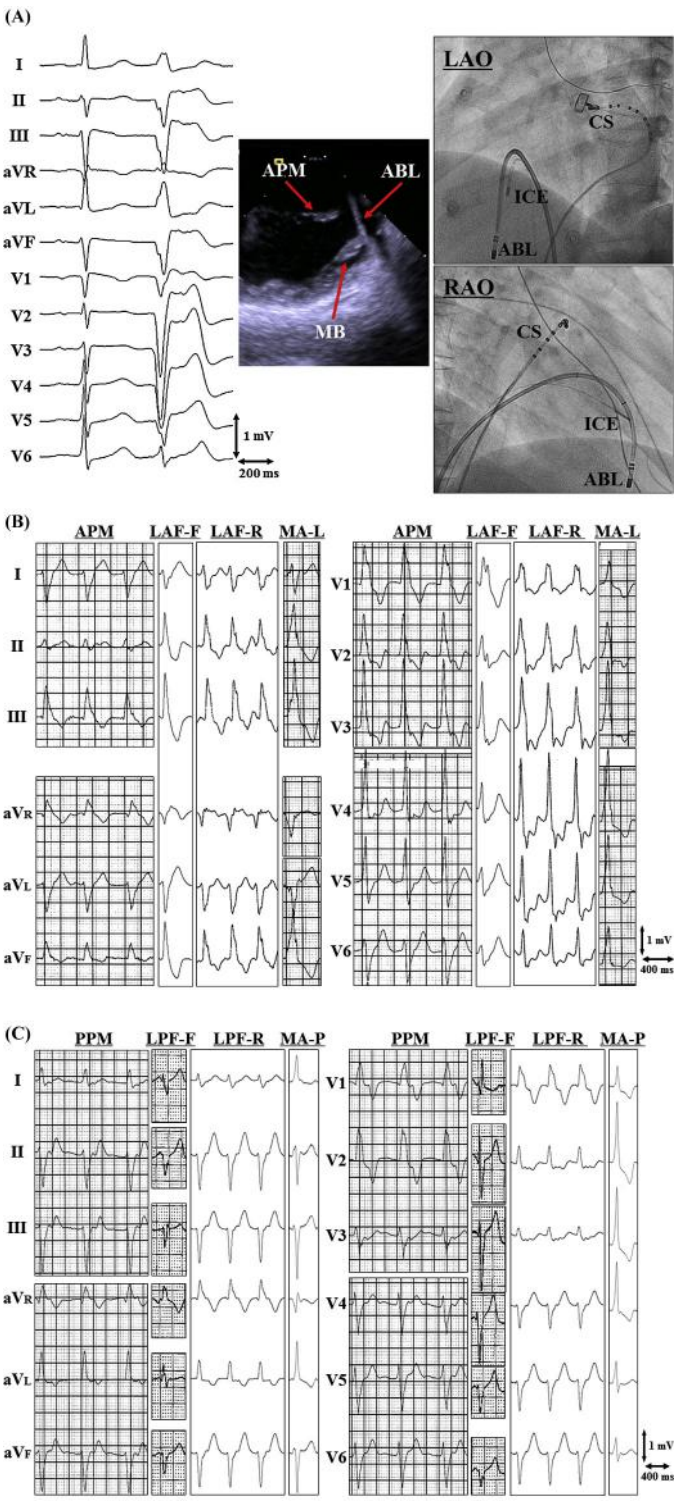

Fig. 3. (A) Twelve-lead electrocardiograms exhibiting a premature ventricular contraction originating from the moderator band (MB) (left panel), and an intra-cardiac echocardiographic image (middle panel), and fluoroscopic images (right panels) exhibiting the successful ablation site of the premature ventricular contraction originating from the MB. ICE, intra-cardiac echocardiography catheter. The other abbreviations are as in the previous figures. This figure was modified from Yamada [9], with permission. (B) and (C) Representative 12-lead electrocardiograms of the QRS complexes during ventricular arrhythmias (VAs) originating from the antero-lateral (B) and postero-septal (C) regions in the LV. APM, antero-lateral papillary muscle; L, lateral portion; P, posterior portion; PPM, postero-medial papillary muscle; X-F, R, VAs with a focal or macroreentrant mechanism. This figure was reproduced from Yamada et al. [20], with permission.

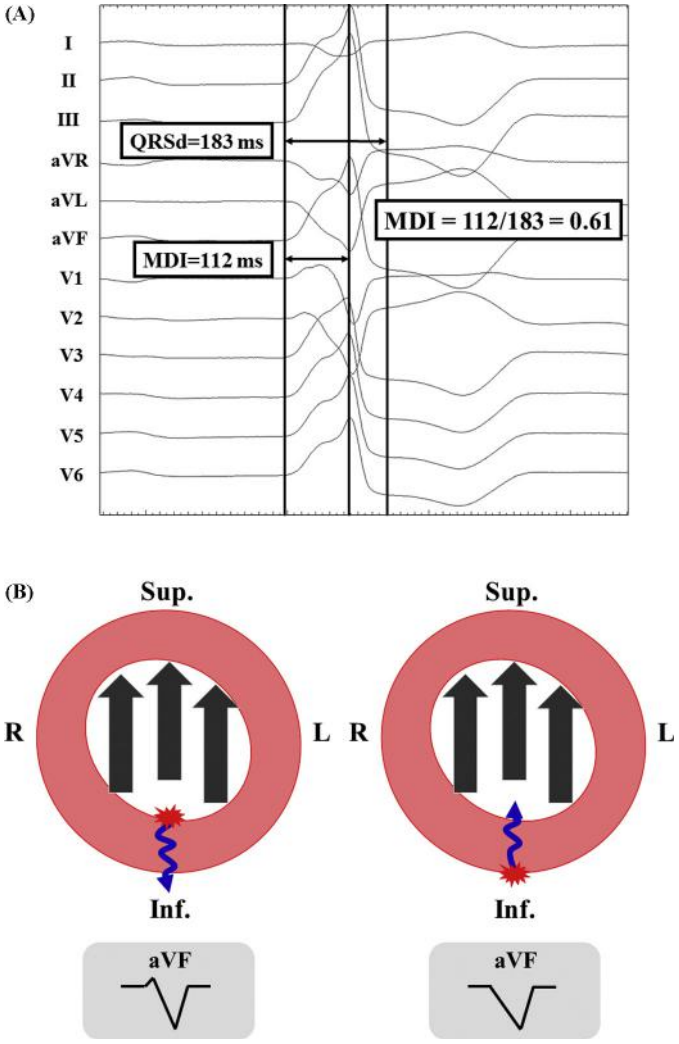

Fig. 4. (A) Twelve-lead electrocardiograms exhibiting a ventricular arrhythmia originating from the LV summit and the measurement of the maximal deflection index (MDI). (B) Schema showing the mechanism to explain the difference in the QRS morphology in lead aVF during ventricular tachycardias with endocardial (left) and epicardial (right) foci. Inf., inferior; L, left; R, right; Sup., superior.
This figure was reproduced from Yamada [25], with permission.

middle of the ventricle (papillary muscles or left fascicles) (Fig. 3) [2,20]. Twelve-lead ECGs are helpful for determining the likely epicardial VT origins (Fig. 4). Because in human hearts the Purkinje network is located only in the subendocardium, ventricular activation from the epicardial origin requires more time to reach the Purkinje network, resulting in a slow onset of the QRS during epicardial VTs. Based on this mechanism, several parameters predicting epicardial VT origins have been proposed: a "pseudo-delta" wave duration >34 ms, QRS duration >200 ms, delayed intrinsicoid deflection of >85 ms, RS complex duration >121 ms, and maximum deflection index (MDI) (calculated by dividing the shortest time from the QRS onset to the maximum deflection in any of the precordial leads by the total QRS duration) of >0.54 (Fig. 4A) [29,30]. When ventricular activation propagates from an epicardial origin at the LV free wall or ventricular posterior wall, the total activation vector should go from a lateral toward medial or from an inferior toward superior direction, resulting in a QS pattern in lead I or lead aVF (Fig. 4B) [25]. On the other hand, when ventricular activation propagates from an endocardial origin on the LV free wall or ventricular posterior wall, a part of the activation vector should go toward the lateral or inferior direction,

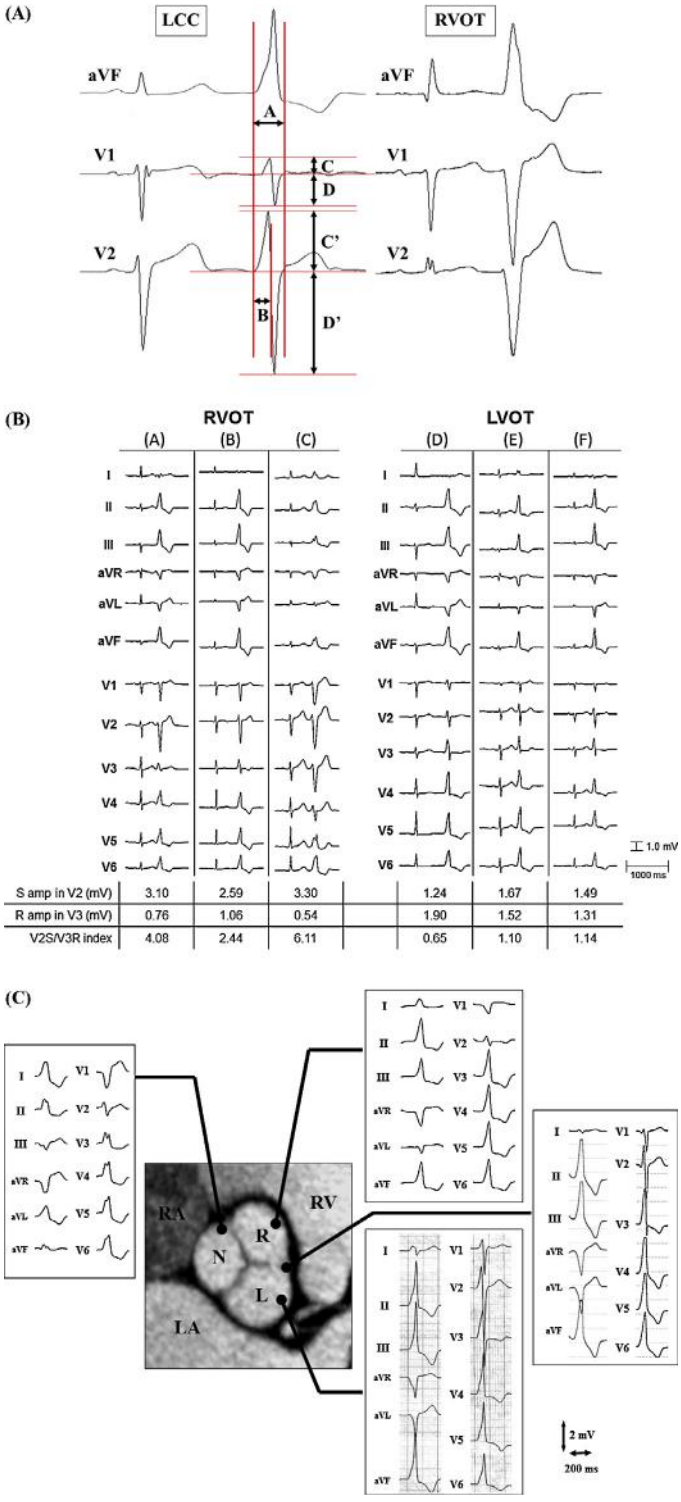

Fig. 5. (A) Examples of an electrocardiographic analysis of ventricular arrhythmias (VAs). The first beats are sinus and the second beats are VAs originating from the LCC and RVOT. A indicates the total QRS duration, B the longer R-wave duration in lead V1 or V2, determined in lead V2 from the QRS onset to the R-wave intersection point where the R wave crosses the isoelectric line, C the R-wave amplitude, measured from the peak to the isoelectric line, and D the S-wave amplitude measured from the QRS nadir to the isoelectric line. The R/S-wave amplitude ratio in lead V2 (C′/D′) is greater than that in lead V1 (C/D) and C′/D′ is determined as the R/S-wave amplitude index. The R/S amplitude index is less than 0.3 and the R-wave duration index less than 0.5 during RVOT VAs, whereas they are not during LCC VAs. This figure was reproduced from Yamada et al. [5], with permission. (B) Representative 12-lead electrocardiograms of VAs originating from the ventricular outflow tract. The first beat is a sinus beat and the second is a premature ventricular contraction (PVC) in each panel (A–F). The S-wave amplitude in lead V2, R-wave amplitude in lead V3, and V2S/V3R index are listed below each panel. All RVOT PVCs exhibited a V2S/V3R index of >1.5, while all LVOT PVCs exhibited a V2S/V3R index of ≤1.5. The PVCs were successfully ablated in the RVOT septum (A and B), RVOT free wall (C), left coronary cusp (D), right coronary cusp (E), and aorto-mitral continuity (F). The other abbreviation is as in the previous figure. This figure was reproduced from Yoshida et al. [31] with permission. (C) Two-dimensional CT images and representative 12-lead electrocardiograms of VAs originating from the aortic root. L, left coronary cusp; N, non-coronary cusp; R, right coronary cusp. The other abbreviations are as in the previous figures. This figure was reproduced from Yamada et al. [7], with permission.

which reflects the activation conducting through the wall of ventricular muscle toward the epicardium, resulting in the presence of an initial R wave in lead I or lead aVF (Fig. 4B). Therefore, a QS pattern in lead I or aVF suggests an epicardial origin in the LV free wall [9] or the ventricular posterior wall, respectively (Fig. 4B). All these ECG features are more accurate without SHD than with it because without SHD the ventricular activation propagates away from VA origins through healthy ventricular myocardium in a predictable manner.

The ECG characteristics of IVAs originating from the RVOT and LVOT are similar, because anatomically, the RVOT and LVOT are located close to each other (Fig. 2B). The ECGs of idiopathic outflow tract VAs are characterized by positive R waves in all inferior leads and deep S waves in both leads aVR and aVL (almost QS pattern) (Fig. 5). RBBB QRS morphology clearly suggests a VA origin on the left side. However, when LBBB QRS morphology is observed, it is often difficult to differentiate RVOT VAs from LVOT VAs. Because anatomically the LVOT is located posterior to the RVOT (Fig. 2B), LVOT VAs exhibit taller and wider R waves in leads V1 and V2 than RVOT VAs. Therefore, the precordial transition is helpful for differentiating RVOT VAs from LVOT VAs. When the precordial transition is later than lead V4, the VAs are likely to originate from the RVOT, and when the precordial transition is earlier than lead V2, the VAs are likely to originate from the LVOT. However, when the precordial transition is in lead V3, it is most difficult to differentiate RVOT VAs from LVOT VAs. Among multiple ECG algorithms to differentiate RVOT VAs from LVOT VAs, two ECG algorithms may be recommended, the magnitude and width of the R wave or QRS complex in leads V1 and V2 (R/S-wave amplitude and duration indexes) [6] (Fig. 5A) and V2S/V3R amplitude ratio [31] (Fig. 5B), because they can simply and accurately make a diagnosis by an ECG of VA only. The R/S-wave amplitude in leads V1 and V2 is measured as the amplitude of the QRS complex peak or nadir to the isoelectric line. The R/S-wave amplitude index, calculated from the percentage of the R/S-wave amplitude ratio in lead V1 or V2 (whichever is greater), is considered more useful than the R/S-wave amplitude ratio alone in lead V1 or V2. The R-wave duration index is calculated by dividing the longer R-wave duration in lead V1 or V2 by the QRS complex duration. An R/S amplitude index of <0.3 and R-wave duration index of <0.5 may strongly suggest a VA origin on the right side (Fig. 5A) [6]. The V2S/V3R amplitude ratio is calculated by dividing the amplitude of the S wave in lead V2 by that of R wave in lead V3. A V2S/V3R amplitude ratio of ≤1.5 can predict LVOT VA origins and that of >1.5 RVOT VA origins (Fig. 5B) [31]. This ECG algorithm is useful even when the precordial transition is in lead V3, and has been proven to be the most accurate among the previous ECG algorithms to differentiate RVOT VA origins from LVOT VA origins.

Although the 3 ACCs are located next to each other, IVAs that can be ablated within each ACC may be differentiated by ECGs (Fig. 5C) [7]. IVAs that can be ablated within the right coronary cusp (RCC) and at the L-RCC rarely exhibit an RBBB pattern, and IVAs that can be ablated within the NCC always exhibit an LBBB pattern. The R-wave amplitude ratio in leads III to II (III/II ratio) is useful for differentiating LCC VAs from RCC VAs. When the III/II ratio is >0.9, VAs are more likely to be ablated within the LCC. A qrS

pattern in the right precordial leads may be highly specific for an L-RCC VA origin (Fig. 5C) [12]. The ECG characteristics of NCC VAs are similar to those of RCC VAs (Fig. 5C) [13]. However, an S wave in lead III is present during NCC VAs although it is not during RCC VAs. When the III/II ratio is <0.65, VAs are more likely to be ablated from within the NCC.

All MA VAs exhibit an RBBB pattern and monophasic R or Rs in leads V2 to V6 (Fig. 6A) [8,9]. Because the origins of all MA VAs are located in the posterior portion of the LV, distant from the precordial electrodes, the activation from the MA VA origins propagates toward these electrodes, resulting in an early precordial transition and concordant positive QRS pattern in leads V2 to V4 during MA VAs. The ECG characteristics are helpful for predicting sites of MA VA origins [8,9]. The polarity of the QRS complex in the inferior and lateral leads (I and aVL) is positive and negative in antero-lateral MA VAs while it is negative and positive in posterior and postero-lateral MA VAs, respectively. MA VAs originating from the free wall of the MA are characterized by a longer QRS duration sometimes with pseudo-delta waves and notching in the late phase of the R or Q wave in the inferior leads, which may result from phased excitation from the LV free wall to the RV (Fig. 6A). Posterior MA VAs exhibit a dominant R wave in lead V1, whereas postero-septal MA VAs exhibit a negative QRS component in lead V1 (qR, qr, rs, rS, or QS).

All TA VAs exhibit an LBBB QRS morphology and positive QRS polarity in leads I, V5, and V6 (Fig. 6B) [16] because the TA VA origins are located on the right anterior side of the heart, and the activation propagating from TA VA origins toward the apex generates a positive QRS polarity in leads V5 and V6. The R wave in lead I is usually taller during TA VAs than during RVOT VAs because the TA is located more rightward and inferior to the RVOT. For the same reason, a positive QRS polarity in all of the inferior leads is rare in TA VAs but common in all RVOT VAs. During TA VAs, a QS or rS pattern in lead aVL is rare, and the QRS polarity in lead aVL is positive in almost all TA VAs, which is not the case for RVOT VAs. Among all TA VAs, the QRS duration and Q-wave amplitude in each of the leads V1 to V3 are greater in TA VAs originating from the free wall of the TA than in those from the septal wall of the TA

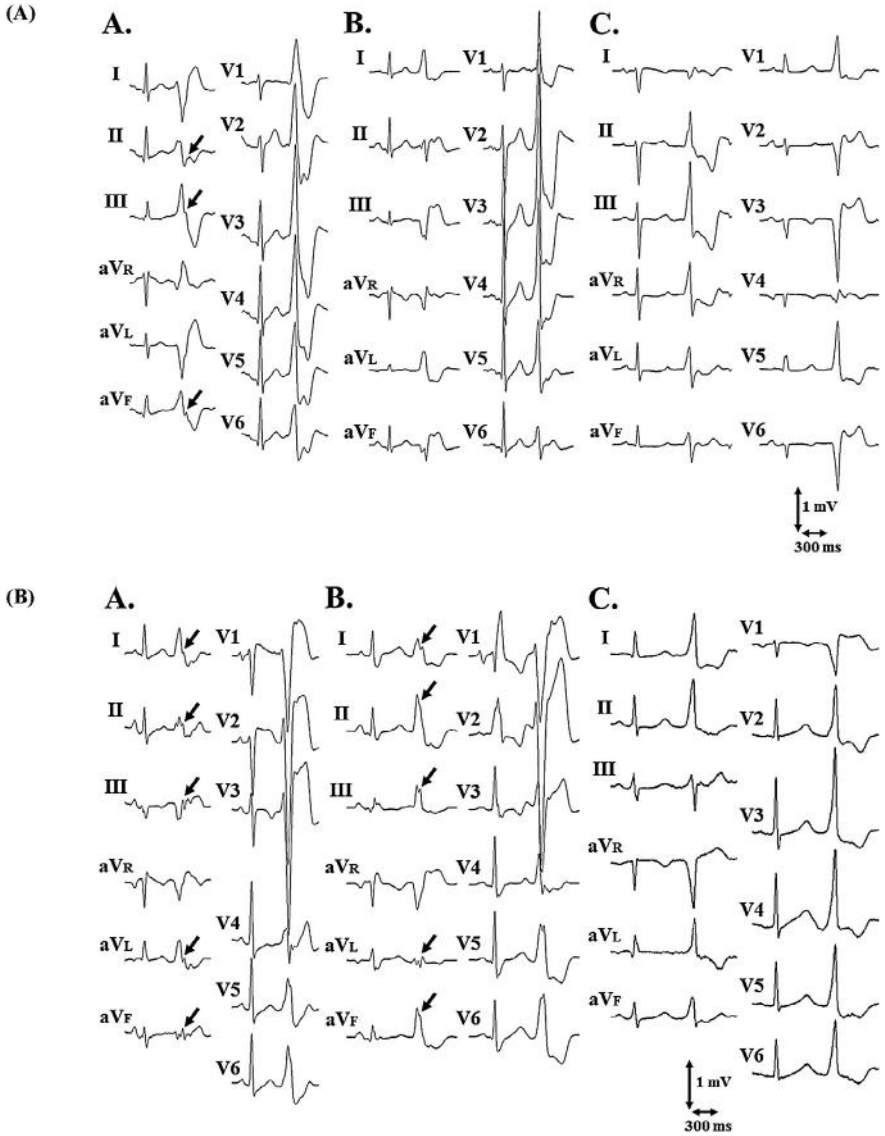

Fig. 6. (A) Representative 12-lead electrocardiograms of the premature ventricular contractions originating from the antero-lateral (a), posterior (b), and postero-septal (c) aspects of the mitral annulus. The arrows indicate "notching" of the late phase of the QRS complex in the inferior leads. This figure was reproduced from Yamada [9], with permission. (B) Representative 12-lead electrocardiograms of the premature ventricular contractions originating from the postero-lateral (a), anterior (b), and antero-septal (c) aspects of the tricuspid annulus. The arrows indicate "notching" of the late phase of the QRS complex in the limb leads. This figure was reproduced from Yamada [9] with permission.

[16]. Septal TA VAs exhibit an early precordial transition (lead V3), narrower QRS duration, and QS in lead V1 with the absence of notching in the inferior leads while the free wall TAVAs are associated with a late precordial transition (>lead V3), wider QRS duration, the absence of Q waves in lead V1, and the presence of notching in the inferior leads (the timing of the second peak of the notched QRS complex in the inferior leads corresponds precisely with the LV free wall activation) (Fig. 6B). A negative QRS polarity in the inferior leads predicts VA origins in the posterior aspect of the TA, and otherwise, VA origins in the mid to anterior aspects of the TA are suggested.

IVAs originating from the antero-lateral and postero-septal PAMs in the LV exhibit RBBB and right inferior and left or right superior axis QRS morphologies, respectively (Fig. 3B and C) [17–20]. IVAs originating from the posterior or anterior RV PAMs more often exhibit a superior axis with a late precordial transition (>lead V4) as compared with septal RV PAM VAs, which more often exhibit an inferior axis with an earlier precordial transition (≤lead V4) [21].

Because of the close anatomical relationship, it is important to distinguish PAM VAs from MA VAs and LV fascicular VAs by ECGs (Fig. 3 B and C) [20]. The ECG features, such as an rS in lead I, rS in lead aVR (for only the LV antero-lateral region), qR in lead aVL, Q in lead V1, S wave amplitude ratio in leads III to II <1.5, and R/S ratio ≤1 in lead V6 (the last 2 parameters are for only the LV postero-septal region) can accurately distinguish MA VAs from PAM and LV fascicular VAs [20]. However, the ECG features are similar for PAM and LV fascicular VAs, and an R/S ratio ≤1 in lead V6 in the LV antero-lateral region and a QRS duration >160 ms, and qR or R waves in lead V1 (as compared with an rsR′ for fascicular VTs) in the LV postero-septal region may be the only reliable predictors for differentiating PAM VAs from LV fascicular VAs [20].

IVAs arising from the MB exhibit a distinctive ECG morphology, LBBB and left superior axis QRS morphology, sharp downstroke of the QRS in the precordial leads, and relatively narrow QRS duration (Fig. 3A) [22]. MB VAs not only have a late precordial transition pattern, typically after lead V4, but also the transition is always later than that of the sinus QRS. Among the idiopathic RV VAs, a late precordial transition and superiorly directed nature are helpful for distinguishing MB VAs from VAs originating from the RV base or septum [22].

IVAs arising from the crux of the heart exhibit a left superior axis QRS morphology with deeply negative deltoid waves (QS pattern) in the inferior leads and an early precordial transition (a prominent R wave in lead V2), which may be associated with a polarity reversal between leads V1 and V2 (Fig. 2C) [27]. It is noted that a QS or large S wave is possible in lead V6 although crux VAs arise from the LV base. The common ECG characteristics of LV summit VAs are a right inferior axis QRS morphology, wider QRS, and larger MDI than the other idiopathic LVOT VAs [28]. The MDI [30] of these epicardial IVAs is usually >0.55.

Treatment of IVAs

Treatment of IVAs should be tailored according to the type of VAs, PVCs or VTs, and the absence or presence of SHD (Fig. 7) [3,4]. In the absence of SHD, the most common indication for treating PVCs remains the presence of symptoms that are not improved by an explanation of their benign nature and reassurance from the physician. Exercise stress testing should be considered to determine whether PVCs are potentiated or suppressed by exercise, to assess whether longer duration VAs are provoked especially when symptoms are associated with exercise. PVCs that worsen with exercise should prompt further investigation as these patients are more likely to require treatment. Frequent asymptomatic PVCs may have to be treated if PVC-induced cardiomyopathy is present. For patients with a PVC burden of 10% (approximately 10,000 PVCs/24 h), follow-up with repeat echocardiography and Holter monitoring should be considered. In patients with fewer PVCs, further investigation is only necessary should the symptoms increase. For patients without SHD and mild symptoms, the first step in the treatment of patients with PVCs is education of the benign nature of this arrhythmia and reassurance. For patients whose symptoms are not effectively managed in this manner, beta-blockers or non-dihydropyridine calcium antagonists may be attempted although the efficacy of these agents is limited with only 10–15% of patients achieving a 90% PVC suppression, similar to placebo [3,4]. It should also be recognized

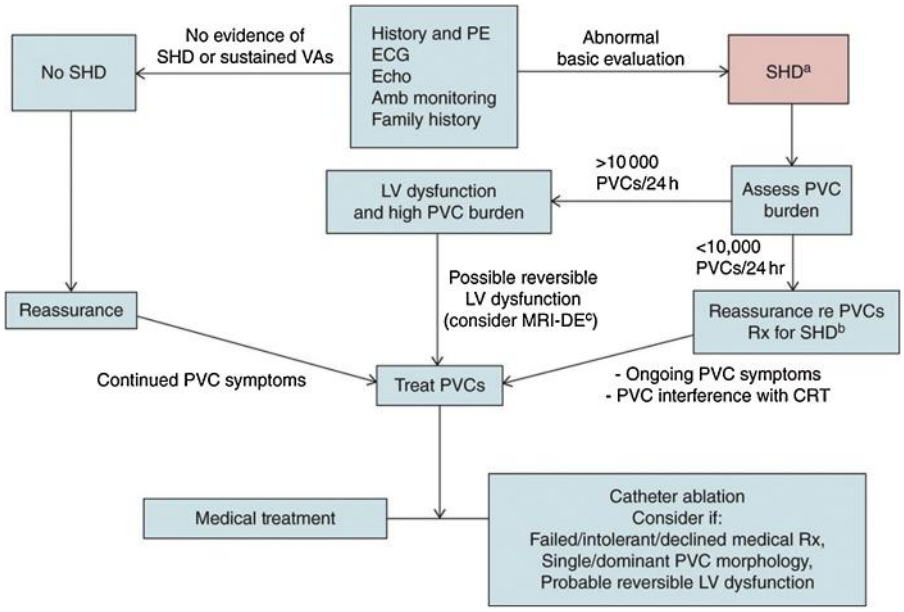

Fig. 7. Schema exhibiting the management of PVCs. (a) Absence of high scar burden suggests reversibility; (b) medical therapy + implantable cardioverter-defibrillator. CRT, cardiac resynchronization therapy; LV, left ventricular; MRI-DE, magnetic resonance imaging with delayed enhancement; PE, physical examination; Rx, therapy; SHD, structural heart disease; VAs, ventricular arrhythmias.
Source: This figure was reproduced from Pedersen et al. [3,4] with permission.

that these agents may themselves produce significant side effects rather than relieve the PVC symptoms. Membrane-active antiarrhythmic drugs (AADs) are more effective for suppressing PVCs. Because these agents may increase the risk of mortality in patients with significant SHD, perhaps with the exception of amiodarone, caution is advised before using them for PVC suppression.

Randomized trials of PVC suppression with catheter ablation have not been performed. However, multiple studies have indicated a high efficacy of ablation with PVC elimination in 74–100% of highly symptomatic patients with a high PVC burden [3,4]. Procedural success may be dependent on the site of origin with a lower efficacy reported for epicardial foci than for other sites [1–4]. Although complete PVC elimination is the goal of ablation, partial success may still be associated with significant improvement in the LV systolic function. The efficacy of catheter ablation may be reduced for patients with multiple morphologies of PVCs or those for whom the clinical PVC morphology cannot be induced at the time of the procedure [1–4]. The published complication rates of catheter ablation for PVC suppression are generally low (<1%) [1–4]. Catheter ablation of PVCs is recommended for highly selected patients who remain symptomatic despite conservative treatment or for those with high PVC burdens associated with a decline in the LV systolic function [3,4].

Idiopathic VTs are basically monomorphic and hemodynamically stable. In the absence of SHD, sustained idiopathic VTs are generally associated with an excellent prognosis [1,3,4]. Rarely, idiopathic VTs can have a malignant clinical course, usually with a rapid rate or a short initiating coupling interval [1,3,4]. Most idiopathic non-sustained VTs (NSVTs) originate from the RVOT or LVOT. These arrhythmias only require treatment if they are symptomatic, incessant, or produce LV dysfunction. The treatment of these VTs is either medical with beta-blockers, non-hydropyridine calcium blockers, or class IC drugs, or catheter ablation [1,3,4]. NSVTs with a focal mechanism may also occur from the papillary muscles and respond to beta-blockers or catheter ablation [3,4,17–20]. Reentrant LV fascicular VTs usually present as a sustained form, and can be treated with verapamil or mexiletine, though with a relatively high recurrence risk on oral therapy [3,4,23,24]. Catheter ablation can be recommended when idiopathic VTs are highly symptomatic and drug-refractory, especially if they are exercise-induced [1,3,4].

Conclusions

The sites of IVA origins have been increasingly recognized. IVAs usually originate from specific anatomical structures, commonly endocardial but sometimes epicardial, and exhibit characteristic ECGs based on their anatomical background. IVAs are basically benign, but they require medical treatment or catheter ablation when IVAs are symptomatic, incessant, or produce LV dysfunction.

Conflict of interest

The author declares no conflict of interest related to this article.

References

[1] Stevenson WG, Soejima K. Catheter ablation for ventricular tachycardia. Circulation 2007;115:2750–60.
[2] Yamada T, Kay GN. Optimal ablation strategies for different types of ventricular tachycardias. Nat Rev Cardiol 2012;9:512–25.
[3] Pedersen CT, Kay GN, Kalman J, Borggrefe M, Della-Bella P, Dickfeld T, Dorian P, Huikuri H, Kim YH, Knight B, Marchlinski F, Ross D, Sacher F, Sapp J, Shivkumar K, et al. EHRA/HRS/APHRS expert consensus on ventricular arrhythmias. Europace 2014;16:1257–83.
[4] Pedersen CT, Kay GN, Kalman J, Borggrefe M, Della-Bella P, Dickfeld T, Dorian P, Huikuri H, Kim YH, Knight B, Marchlinski F, Ross D, Sacher F, Sapp J, Shivkumar K, et al. EHRA/HRS/APHRS expert consensus on ventricular arrhythmias. Heart Rhythm 2014;11:e166–96.
[5] Yamada T, Kay GN. How to diagnose and ablate ventricular tachycardia from the outflow tract and aortic cusps. In: Al-Ahmad A, Callans DJ, Hsia HH, Natale A, Oseroff O, Wang PJ, editors. Cardiac electrophysiology clinics; hands-on ablation. Minneapolis: Cardiotext Publishing; 2013. p. 292–301.
[6] Ouyang F, Fotuhi P, Ho SY, Hebe J, Volkmer M, Goya M, Burns M, Antz M, Ernst S, Cappato R, Kuck KH. Repetitive monomorphic ventricular tachycardia originating from the aortic sinus cusp: electrocardiographic characterization for guiding catheter ablation. J Am Coll Cardiol 2002;39:500–8.
[7] Yamada T, McElderry HT, Doppalapudi H, Murakami Y, Yoshida Y, Yoshida N, Okada T, Tsuboi N, Inden Y, Murohara T, Epstein AE, Plumb VJ, Singh SP, Kay GN. Idiopathic ventricular arrhythmias originating from the aortic root: prevalence, electrocardiographic and electrophysiological characteristics, and results of the radiofrequency catheter ablation. J Am Coll Cardiol 2008;52:139–47.
[8] Tada H, Ito S, Naito S, Kurosaki K, Kubota S, Sugiyasu A, Tsuchiya T, Miyaji K, Yamada M, Kutsumi Y, Oshima S, Nogami A, Taniguchi K. Idiopathic ventricular arrhythmia arising from the mitral annulus: a distinct subgroup of idiopathic ventricular arrhythmias. J Am Coll Cardiol 2005;45:877–86.
[9] Yamada T. Idiopathic ventricular tachycardia from mitral annulus, papillary muscles and other sites. In: Bhargava K, Asirvatham SJ, editors. Practical cardiac electrophysiology. New Delhi: Jaypee Brothers Medical Publishers Pvt. Ltd.; 2016. p. 543–66.
[10] McAlpine WA. Heart and coronary arteries. New York: Springer-Verlag; 1975.
[11] Yamada T, Litovsky SH, Kay GN. The left ventricular ostium: an anatomic concept relevant to idiopathic ventricular arrhythmias. Circ Arrhythmia Electrophysiol 2008;1:396–404.
[12] Yamada T, Yoshida N, Murakami Y, Okada T, Muto M, Murohara T, McElderry HT, Kay GN. Electrocardiographic characteristics of ventricular arrhythmias originating from the junction of the left and right coronary sinuses of Valsalva in the aorta: the activation pattern as a rationale for the electrocardiographic characteristics. Heart Rhythm 2008;5:184–92.
[13] Yamada T, Lau YR, Litovsky SH, Thomas McElderry H, Doppalapudi H, Osorio J, Plumb VJ, Neal Kay G. Prevalence and clinical, electrocardiographic, and electrophysiologic characteristics of ventricular arrhythmias originating from the noncoronary sinus of Valsalva. Heart Rhythm 2013;10:1605–12.
[14] Yamada T, McElderry HT, Doppalapudi H, Kay GN. Catheter ablation of ventricular arrhythmias originating in the vicinity of the His bundle: significance of mapping the aortic sinus cusp. Heart Rhythm 2008;5:37–42.
[15] Sekiguchi Y, Aonuma K, Takahashi A, Yamauchi Y, Hachiya H, Yokoyama Y, Iesaka Y, Isobe M. Electrocardiographic and electrophysiologic characteristics of ventricular tachycardia originating within the pulmonary artery. J Am Coll Cardiol 2005;45:887–95.
[16] Tada H, Tadokoro K, Ito S, Naito S, Hashimoto T, Kaseno K, Miyaji K, Sugiyasu A, Tsuchiya T, Kutsumi Y, Nogami A, Oshima S, Taniguchi K. Idiopathic ventricular arrhythmias originating from the tricuspid annulus: prevalence, electrocardiographic characteristics, and results of radiofrequency catheter ablation. Heart Rhythm 2007;4:7–16.
[17] Doppalapudi H, Yamada T, McElderry HT, Plumb VJ, Epstein AE, Kay GN. Ventricular tachycardia originating from the posterior papillary muscle in the left ventricle: a distinct clinical syndrome. Circ Arrhythm Electrophysiol 2008;1:23–9.
[18] Yamada T, McElderry HT, Okada T, Murakami Y, Doppalapudi H, Yoshida N, Allred JD, Murohara T, Kay GN. Idiopathic focal ventricular arrhythmias originating from the anterior papillary muscle in the left ventricle. J Cardiovasc Electrophysiol 2009;20:866–72.
[19] Yamada T, Doppalapudi H, McElderry HT, Okada T, Murakami Y, Inden Y, Yoshida Y, Yoshida N, Murohara T, Epstein AE, Plumb VJ, Litovsky SH, Kay GN. Electrocardiographic and electrophysiological characteristics in idiopathic ventricular arrhythmias originating from the papillary muscles in the left ventricle: relevance for catheter ablation. Circ Arrhythm Electrophysiol 2010;3:324–31.
[20] Yamada T, Doppalapudi H, McElderry HT, Okada T, Murakami Y, Inden Y, Yoshida Y, Kaneko S, Yoshida N, Murohara T, Epstein AE, Plumb VJ, Kay GN. Idiopathic ventricular arrhythmias originating from the papillary muscles in the left ventricle: prevalence, electrocardiographic and electrophysiological characteristics, and results of the radiofrequency catheter ablation. J Cardiovasc Electrophysiol 2010;21:62–9.
[21] Crawford T, Mueller G, Good E, Jongnarangsin K, Chugh A, Pelosi Jr F, Ebinger M, Oral H, Morady F, Bogun F. Ventricular arrhythmias originating from papillary muscles in the right ventricle. Heart Rhythm 2010;7:725–30.
[22] Sadek MM, Benhayon D, Sureddi R, Chik W, Santangeli P, Supple GE, Hutchinson MD, Bala R, Carballeira L, Zado ES, Patel VV, Callans DJ, Marchlinski FE, Garcia FC. Idiopathic ventricular arrhythmias originating from the moderator band: electrocardiographic characteristics and treatment by catheter ablation. Heart Rhythm 2015;12:67–75.
[23] Tsuchiya T, Okumura K, Honda T, Honda T, Iwasa A, Yasue H, Tabuchi T. Significance of late diastolic potential preceding Purkinje potential in verapamil-sensitive idiopathic left ventricular tachycardia. Circulation 1999;99:2408–13.
[24] Nogami A, Naito S, Tada H, Taniguchi K, Okamoto Y, Nishimura S, Yamauchi Y, Aonuma K, Goya M, Iesaka Y, Hiroe M. Demonstration of diastolic and presystolic Purkinje potentials as critical potentials in a macroreentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia. J Am Coll Cardiol 2000;36:811–23.
[25] Yamada T. Transthoracic epicardial catheter ablation: indications, techniques, and complications. Circ J 2013;77:1672–80.

[26] Yamada T, Maddox WR, McElderry HT, Doppalapudi H, Plumb VJ, Kay GN. Radiofrequency catheter ablation of idiopathic ventricular arrhythmias originating from intramural foci in the left ventricular outflow tract: efficacy of sequential versus simultaneous unipolar catheter ablation. Circ Arrhythm Electrophysiol 2015;8:344–52.
[27] Doppalapudi H, Yamada T, Ramaswamy K, Ahn J, Kay GN. Idiopathic focal epicardial ventricular tachycardia originating from the crux of the heart. Heart Rhythm 2009;6:44–50.
[28] Yamada T, McElderry HT, Doppalapudi H, Okada T, Murakami Y, Yoshida Y, Yoshida N, Inden Y, Murohara T, Plumb VJ, Kay GN. Idiopathic ventricular arrhythmias originating from the left ventricular summit: anatomic concepts relevant to ablation. Circ Arrhythm Electrophysiol 2010;3:616–23.
[29] Berruezo A, Mont L, Nava S, Chueca E, Bartholomay E, Brugada J. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. Circulation 2004;109:1842–7.
[30] Daniels DV, Lu YY, Morton JB, Santucci PA, Akar JG, Green A, Wilber DJ. Idiopathic epicardial left ventricular tachycardia originating remote from the sinus of Valsalva: electrophysiological characteristics, catheter ablation, and identification from the 12-lead electrocardiogram. Circulation 2006;113:1659–66.
[31] Yoshida N, Yamada T, McElderry HT, Inden Y, Shimano M, Murohara T, Kumar V, Doppalapudi H, Plumb VJ, Kay GN. A novel electrocardiographic criterion for differentiating a left from right ventricular outflow tract tachycardia origin: the V2S/V3R index. J Cardiovasc Electrophysiol 2014;25:747–53.