Non-Pharmacological Management of Ventricular Tachycardia

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Ventricular tachyarrhythmias (VTA), a major cause of sudden cardiac death, require meticulous management in order to prevent recurrent episodes. Recently, non-pharmacological interventions, including radiofrequency catheter ablation and implantable cardioverter defibrillators (ICD), have become important treatments of VTA. Catheter ablation is curative in a relatively high percentage of patients presenting with idiopathic monomorphic ventricular tachycardia (VT). For VT associated with structural heart disease, however, the efficacy of catheter ablation remains limited, and ICD is the first-line therapy. In a subset of patients presenting with recurrent episodes of ventricular fibrillation (VF), catheter ablation is a therapeutic option when the VF is triggered by specific premature ventricular complexes. In Japan, unlike in the United States and Europe, ICD have not yet been accepted as first-line prevention of sudden cardiac death caused by VTA. The efficacy of ICD is occasionally limited by intolerable complications, such as electrical storm, inappropriate shock delivery and infection. Catheter ablation and ICD therapy might need to be combined for problematic cases. (Circ J 2007; Suppl A: A-97–A-105)

Key Words: Catheter ablation; Implantable cardioverter defibrillators; Ventricular tachyarrhythmia

Catheter Ablation

Radiofrequency (RF) is widely used as the source of energy for catheter ablation, delivered via catheters with a 4 or 8 mm distal tip electrode. The role of RFCA varies according to the VTA that is targeted. It is curative for idiopathic ventricular tachycardia (VT) in patients without structural heart disease, but for VTA in patients with structural heart disease, however, RFCA can be used as adjunctive therapy to lower the likelihood of ICD shock delivery.1

For VT caused by triggered activity or abnormal automaticity, the ablation target is the site of earliest activation during ongoing tachycardia. Pacing from that site (pace-mapping) must produce a QRS morphology identical to that observed during VT. For VT caused by reentry, the most prevalent mechanism in patients with structural heart disease, the target of ablation is the critical slow pathway or isthmus of the reentrant circuit.

Idiopathic VT Originating From the Ventricular Outflow Tract

Idiopathic outflow tract VT is usually observed in young patients without apparent structural heart disease, often occurring in repetitive nonsustained bursts interrupted by sinus rhythm and frequent premature ventricular complexes (PVCs), although sustained VT is not rare. Its onset can be provoked by endogenous or exogenous catecholamines and, in some patients, VT develops during exercise, emotional stress or both. Its most common mechanism is believed to be cyclic AMP-mediated triggered activity, although catecholamine-associated automaticity and intra- or interfascicular reentry have also been proposed. Accordingly, it can be effectively terminated by intravenous administration of adenosine. Although this type of VT is usually not life-threatening, it can be intolerable, causing palpitation, chest discomfort or faintness, and RFCA is attempted to mitigate or eliminate these symptoms, particularly when they are drug refractory. In rare cases, incessant VT can induce a cardiomyopathy, and RFCA is a therapeutic option in such situations.3-5

Origin VT originating from the right ventricular outflow tract (RVOT) is characterized by left bundle branch block (LBBB) morphology of the QRS and a transition usually at or beyond precordial lead V3 of the surface ECG. A free wall origin is suspected when a notched QRS is observed in the inferior leads and an R pattern is recorded in lead I. If the QRS is not notched in the inferior leads, and lead I shows an rs’ pattern, a septal origin is likely. VT originating from above the pulmonary valve is characterized by a higher R wave amplitude in the inferior leads than when it originates from below the valve. Catheter ablation from the left ventricle (LV) or from a cusp of the aortic valve may need to be performed if the precordial...
transition of the QRS occurs before lead V1.6,8 The origin of VT is probably LV epicardial if the precordial QRS transition is at lead V1 and no S wave is present in leads V5–6.

**Mapping** Pace- and activation-mapping is used to determine the site of origin of VT. When the paced QRS morphology is nearly identical to the targeted VT or PVC (pace-mapping score ≥11/12), the site of pacing is very near the VT origin. Pacing from a cusp of the aortic valve or from the pulmonary artery requires higher pulse strength to capture the ventricular myocardium. In activation mapping, the earliest site of local depolarization during ongoing VT is usually recorded 20–30 ms prior to the onset of the QRS complex on the surface ECG. A unipolar electrogram is recorded from the distal tip of the ablation catheter to identify a characteristic QS pattern, corresponding to the earliest site of activation, the target of ablation.9 In some patients, a spike or pre-potential is recorded from the site of successful ablation, though the electrophysiological significance of these potentials has not been clarified.

Nonsustained VT or PVCs, which are both frequently observed at the bedside, often disappear completely during the electrophysiological procedure. The intravenous administration of isoproterenol, atropine or aminophylline helps initiate the VT episodes, whereas in most cases of triggered activity, programmed stimulation fails to induce the tachyarrhythmia.

**Ablation** Because the origin of VT is confined to a small area, we use a 4-mm tip ablation catheter to determine the site of origin and deliver RF energy. In some patients, VT originates from a region instead of a point, in which case variations in the QRS morphology of the VT are usually observed.10 Using the temperature or power control mode, 35–50 W of RF energy is delivered for VT originating from the right ventricle (RV) or LV, while keeping the temperature at ≤55°C. If the VT terminates, or frequent nonsustained VT or PVCs disappear within 15 s of RF application, the ablation is continued for 60–90 s (Fig 1). Otherwise, the application is discontinued after 15 s and the target site is remapped. For ablation from the cusps of the aortic valve or above the pulmonary valve, ablation should start at a much lower energy setting. Coronary angiography is performed before delivering RF energy from an aortic cusp. To avoid serious complications, RF should not be delivered to a site ≤1.0 cm away from a coronary artery. A 90–95% success rate has been reported with RF ablation for this type of VT.10–12

**Verapamil-Sensitive LV Idiopathic VT** This VT was firstly described by Zipes et al in 1979.13 It is effectively suppressed by verapamil, as well as class I antiarrhythmic drugs, and reentry is its most likely mechanism.14

**Origin** Three forms of QRS morphology have been described: (1) right bundle branch block (RBBB) configuration and left axis deviation indicative of a left posterior fascicular origin, (2) RBBB configuration and right axis deviation consistent with a left anterior fascicular origin, and (3) a narrow QRS and normal or right axis deviation, suggesting an upper septal fascicular origin.15,16 In >80% of cases, verapamil-sensitive VT has a left posterior fascicular origin.16

**Mapping** Two diastolic potentials (P1 and P2) are recorded during VT (Fig 2). The mid-diastolic potential (P1) is observed earlier on the proximal than the distal electrode of a multipolar mapping catheter, whereas the pre-systolic Purkinje potential (P2), fused with the local ventricular electrogram, is recorded earlier on the distal than on the proximal electrode of the mapping catheter placed along the septum. Decremental conduction is observed between P1 and P2 during VT.17 Verapamil slows and terminates the

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**Fig 1.** Idiopathic right ventricular outflow tract (RVOT) tachycardia. (Panel A) Site of ventricular tachycardia (VT) origin (earliest activation: EAS) was mapped to the RVOT during ongoing tachycardia. (Panel B) Application of radiofrequency (RF) to that site soon terminated VT. Top tracings: leads I, II and V1 of surface ECG. Map, mapping catheter recording; d, distal tip electrode recording; m, middle tip electrode recording; p, proximal tip electrode recording; u, unipolar electrogram from distal tip electrode.
VT by prolongation of the interval and, finally, conduction block between P1 and P2.

Ablation  In the presence of RBBB morphology and left axis deviation, the site of earliest activation during VT is mapped in the apical mid-septum, while P1 is recorded from the mid-septum. When P1 is recorded, it is the target for RF application because it is considered to be an essential component of the reentry circuit. Nakagawa et al18 and Tsuchiya et al19 have shown that P1 can be recorded from the inferior apical area to the base of the LV septum. To avoid LBBB or atrioventricular block, we start the RF application at the apical site, as identified by P1. An acceptable success rate can also be achieved by delivering RF to P2 only. The earliest activation site during VT with a RBBB QRS morphology and right axis deviation is found on the anterolateral LV wall and diastolic potentials are recorded on the LV mid-septum.20

We apply 30–40 W of RF energy during ongoing VT, keeping the maximum temperature below 55°C, and continue for 60–90 s if the VT is terminated or slowed within 15 s after the onset of RF application. Otherwise, RF application is discontinued after 15 s and the target site is remapped. RF might have to be delivered during sinus rhythm if the catheter position is unstable during VT. Ablation is considered successful when VT is no longer inducible after treatment. The success rate of RF ablation is >95% for VT with left axis deviation, and appears lower in VT with right axis deviation.16,17,20

VT Associated With Underlying Heart Disease
In the United States and Europe, a healed myocardial infarction (MI) is the source of structural heart disease underlying VTA in >90% of patients, whereas nonischemic heart diseases, such as arrhythmogenic RV cardiomyopathy, nonischemic dilated cardiomyopathy (DCM) and repaired congenital heart disease are more common in Japan.22,23 The site of VT origin can be estimated by examination of the QRS recorded during the VT. Briefly, VT with a LBBB pattern points to a LV free wall origin.24

Standard Mapping of the VT  Pace-mapping is performed during spontaneous rhythm. The site where the paced QRS morphology is identical to that of the clinical VT (perfect pace-mapping) is either the exit from a reentry circuit or the earliest activation during VT caused by triggered activity or abnormal automaticity. Pacing sites with a >50 ms stimulus-to-QRS interval might be associated with the arrhythmogenic substrate of the VT.25,26 At such sites, pacing at different cycle lengths, different stimulation intensity, or both, can change the morphology of the paced QRS and enable perfect pace-mapping (Fig 3).27 During sinus rhythm, isolated high-frequency late potentials can be recorded from inside or near the arrhythmogenic substrate. However, separately examining the significance of each isolated high-frequency late potential during the electrophysiological study is a tedious endeavor.

When the VT is morphologically stable and hemodynamically tolerated, detailed activation mapping is performed, with particular attention paid to the surroundings and inside of the diseased myocardial area. When the QRS complex during the VT is driven by a high-frequency, isolated, mid-diastolic potential, it can be targeted for RF application.28 However, a correlation of each mapping site with the reentry circuit should be confirmed by entrainment mapping, using the criteria described by Stevenson et al.29 Briefly, when pacing is performed in the protected zone of slow
Fig 3. Pace-mapping in a patient with healed myocardial infarction. (Panel A) During pacing at 667 ms cycle length, the interval between stimulus artifact and paced QRS (a) =100 ms. (Panel B) During pacing at 600 ms cycle length from the same site, a new morphology of the paced QRS (b) is observed, associated with a longer (120 ms) stimulus artifact-to-QRS interval. Note that the first paced QRS morphology during pacing at 600 ms cycle length is identical to that observed during pacing at 667 ms cycle length. PCL, pacing cycle length. Other abbreviations as in previous figures.

Fig 4. Concealed entrainment in the same patient as in Fig 3. Pacing at a cycle length of 420 ms from the site where the local electrogram was recorded 110 ms before the onset of the QRS complex recorded during VT. The interval from stimulus artifact to onset of QRS (100 ms) is close to the interval between the local electrogram and QRS complex (110 ms) during VT. The post-pacing interval (470 ms) is identical to the cycle length of VT. LV, left ventricle. Other abbreviations as in previous figures.
conduction within the reentry circuit, the paced QRS morphology during the VT is identical to that of the unpaced VT (concealed fusion). Concealed fusion cannot be elicited by pacing from outside the protected isthmus. When pacing from the common slow pathway, the first post-pacing cycle after cessation of entrainment, recorded at the pacing site, should be equal to the VT cycle length (Fig 4). If the post-pacing cycle length is >30 ms longer than the VT cycle length, the pacing site is likely to be located in a bystander region connected to the common slow pathway of the reentry circuit. During pacing at the common slow pathway, the interval measured between the stimulus and the onset of the paced QRS complex should be within 20 ms of the interval measured between the local electrogram and the onset of the QRS complex during spontaneous VT.

Mapping of the VT Substrate. Attempts at standard mapping during VT in patients with structural heart disease might be limited by failure to induce the clinical VT, by hemodynamic instability during tachycardia, or by the induction of different VT morphologies or polymorphic VT. In 1986, Cassidy et al defined the characteristics of bipolar electrograms recorded by mapping of the underlying LV substrate. Pursuing this concept, substrate mapping is now performed clinically using an electro-anatomical CARTO® mapping system (Biosense Webster, a Johnson & Johnson Company). During electro-anatomical mapping with the CARTO® system, the site with bipolar electrogram amplitude ≤0.5 mV is considered to represent the zone of scar tissue, bipolar electrograms between 0.5 and 1.5 mV are in the border zone, and electrogram with amplitudes >1.5 mV indicate normal myocardium. The critical isthmus of the VT reentry circuit can be located in the diseased myocardial zone between 2 areas of scar tissue or between an anatomical obstacle and scar tissue. However, all anatomical isthmuses identified by substrate mapping do not systematically correspond to a critical isthmus of reentry. For macro-reentrant VT, which is often observed after repair of congenital heart disease, electro-anatomical activation mapping enables mapping of the entire activation pathway during VT, and the target isthmus for RF ablation can be clearly identified (Fig 5).

Ablation. We begin standard ablation with a 4-mm tip catheter and 40–55 W of RF energy, applied for 60–120 s, using the temperature or power control mode at a temperature of 50–60°C. An 8-mm tip catheter is required in some patients. A cooled tip catheter can create much larger and deeper lesions within diseased myocardium and might be effective in some patients; however, it is not available in Japan.

We deliver RF energy during VT, if possible, because termination of the VT during RF application suggests that the VT is a critical participant in the reentry circuit. However, termination of the VT might not mean that a sufficient lesion has been created at the VT origin. Therefore, programmed electrical stimulation should be repeated before ending the ablation session. In some patients, RFCA needs to be performed during sinus rhythm. In such instances, the creation of a satisfactory RF lesion is estimated by the observation of a fall in impedance during RF delivery, a decrease in local electrogram amplitude, and the inability to capture by pacing at the site of local electrogram after ablation. VT might not be eliminated, despite the application of RF at the critical site, because of (1) the presence of a wide common slow pathway, (2) multiple slow pathways, (3) an intramyocardial or epicardial origin, (4) poor catheter contact, or (5) insufficient energy to create an effective lesion in the diseased myocardium.
Catheter Ablation of Ventricular Fibrillation (VF)

Catheter ablation has not generally been considered as a therapy for VF. However, some patients suffer from storms of VF episodes and require effective treatment. Haissaguerre et al recently reported episodes of aborted sudden death caused by VF, triggered by PVCs, in 27 patients without structural heart disease.37 Those patients were treated with RFCA at the site of origin of the PVCs, which successfully suppressed the recurrence of VF. The triggered PVC was mapped to the LV or RV Purkinje network in 23, and to the RVOT in 4 patients. A Purkinje potential preceded the QRS complex of the PVC and was sometime observed at the same site during sinus rhythm. Similar observations have been reported by the same and other investigators, in VF associated with coronary artery disease, congenital long QT syndrome or Brugada syndrome.38 These results are preliminary, and RFCA of VF should only be considered in carefully selected patients.

ICD

The ICD is the most reliable preventive therapy for sudden cardiac death in patients with a history of aborted sudden cardiac death, VF or VT, and its efficacy is highest in patients with depressed cardiac function (secondary prevention).39-41 The United States and European countries have recently expanded the use of ICD for primary prevention of sudden cardiac death in high-risk patients, but in Japan, however, the role of the ICD in the primary prevention of sudden cardiac death is not established.

Secondary Prevention of Sudden Cardiac Death

Randomized, secondary prevention, clinical trials conducted in the United States, Canada and Europe have compared ICD with antiarrhythmic drug therapy. The patients included in those trials had been resuscitated from nearly fatal VF or VT. In the AVID trial, the ICD was compared with amiodarone (or sotalol). In the CASH trial, the efficacy of the ICD was compared with that of amiodarone, metoprolol or propafenone, in patients who had survived a VF episode. During a follow-up of 2–3 years, survival was higher among patients treated with an ICD than among patients assigned to antiarrhythmic drugs. It is noteworthy that patients with a LV ejection fraction (LVEF) <26% had the greatest improvement in prognosis conferred by ICD. In contrast, compared with amiodarone, ICD conferred no survival advantage among patients with LVEF >35%. Similar results were achieved in the CIDS trial.41 These observations indicate that patients with moderate to severe LV dysfunction derive the greatest benefits from ICD therapy.

Primary Prevention of Sudden Cardiac Death

Several clinical trials have examined the role of ICD in the primary prevention of sudden cardiac death, and their results generally support the use of ICD in patients with depressed LV function. The MADIT-I44 and MUSTT trials included patients with ischemic heart disease and nonsustained VT. MADIT-I included 196 patients with LVEF ≤35% and sustained VTA induced by programmed electrical stimulation that was not suppressed by intravenous procainamide. Compared with antiarrhythmic drug treatment, the ICD had a beneficial effect on all-cause, long-term mortality. MUSTT included 704 patients with LVEF <40% and sustained VTA inducible by programmed electrical stimulation. In that trial, patients assigned to ICD had lower all-cause mortality than patients who received electrophysiological study-guided antiarrhythmic drug therapy.

The AMIOVART46 and DEFINITE47 trials studied patients presenting with nons ischemic DCM and nonsustained VT or frequent PVC. AMIOVART included 103 patients whose LVEF was ≤35%. That trial showed no survival difference between ICD recipients and patients treated with amiodarone. However, because the mortality was lower than expected, the trial was terminated prematurely. In the DEFINITE trial, which included 458 patients with LVEF ≤35%, the ICD tended to lower mortality compared with other treatments, though the difference did not reach statistical significance.
The more recent MADIT-II and SCD-HeFT trials included patients with depressed LV function, without requiring the presence of ventricular arrhythmia. MADIT-II studied 1,232 patients with ischemic heart disease and LVEF ≤30%. Mortality was lower in the ICD treatment group by 31% compared with the non-ICD treatment group. The SCF-HeFT trial included 2,521 patients with LVEF ≤35% caused by ischemic heart disease (52%) or nonischemic DCM (48%). Mortality was reduced in the ICD treatment group by 23% compared with the group assigned to placebo, and no difference in mortality was observed between the placebo-treated group and the group treated with amiodarone. Furthermore, the beneficial effects conferred by ICD in patients suffering from ischemic heart disease and nonischemic DCM were similar.

Based on the results from those trials, the USA and European countries have expanded the indications for ICD implantation. The American Heart Association/American College of Cardiology practice guidelines have listed the following as class I primary prevention indications for an ICD: patients presenting with ischemic heart disease or nonischemic DCM were similar.

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Based on the results from those trials, the USA and European countries have expanded the indications for ICD implantation. The American Heart Association/American College of Cardiology practice guidelines have listed the following as class I primary prevention indications for an ICD: patients presenting with ischemic heart disease who are ≥40 days after a last MI or with nonischemic cardiomyopathy, with a LVEF ≤50%, in New York Heart Association heart failure functional class II or III while receiving long-term optimal medical therapy, and with a reasonable life expectancy and a stable functional status for 1 year. The European Society of Cardiology guidelines recommend ICD implantation in selected patients with LVEF <30–35% beyond 40 days after a MI, treated with optimal background therapy, including angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, β-adrenergic blockers, and an aldosterone antagonist, when appropriate.

On the other hand, data applicable to Japanese patients are few. Tanno et al reported that the prognosis of survivors of MI appears considerably better than observed in the USA and Europe. The 2005 guidelines for risks and prevention of sudden cardiac death by the Japanese Circulation Society list class I ICD indications for the primary prevention of sudden cardiac death as follows: (1) in survivors of MI, an ICD is recommended if the patient has a history of syncope, inducible sustained VT refractory to medical therapy, clinical nonsustained VT, and a LVEF <40%; (2) in patients with hypertrophic cardiomyopathy, an ICD is recommended in the presence of risk factors for sudden cardiac death (ie, recurrent episodes of syncope, family history of sudden death, marked LV hypertrophy or poor blood pressure response during exercise), clinical nonsustained VT and inducible sustained VT uneventful of pharmacological therapeutic effect; (3) in patients presenting with nonischemic DCM, an ICD is recommended if LVEF ≤50%, in the presence of a history of syncope and inducible sustained VT not suppressed by antiarrhythmic drugs.

Issues and Complications Associated With ICD Therapy

Despite technological progress and developments, and marked improvements in the functions of ICD systems, ICD recipients may encounter difficulties and complications. First, inappropriate ICD therapy is the most prominent issue. Atrial fibrillation might be detected as VF because of the rapid ventricular response rate, and be the cause of multiple high-energy shocks delivered by an ICD. Programming of the ICD is also complex in patients who present with both slow and fast VT, as well as VF. In such cases, sinus tachycardia might be interpreted as slow VT, triggering the delivery of inappropriate shocks. Likewise, an unexpected decrease in the R wave, together with an increase in T wave amplitudes, can cause double counting of the intracardiac electrogram, causing the ICD to discharge inappropriately (Fig 6) which can usually be remedied by implanting an additional sensing lead. Second, VT or VF might recur incessantly (electrical storm), and lower the patient’s quality of life by triggering frequent and consecutive shocks. Because intravenous amiodarone is not available in Japan, we have found, instead, that β-adrenergic blockers, intravenous nifekalant, or both, are effective in suppressing electrical storms. Emergency RFCA might also be considered, particularly for monomorphic VT. Electrical storms are not rare in ICD recipients and tend to recur. Third, ICD therapy is associated with known procedure-related complications, including injury to the heart and vessels, and pulse generator pocket and lead system infection. Finally, the relatively short battery life and the high cost of the treatment are additional complicating issues.

Although ICD terminate VT/VF efficiently, they cannot prevent the development or recurrence of VT/VF, and supplemental treatment might be useful to lower the likelihood of arrhythmic events after implantation of the system. Sotalol might decrease the number of ICD discharges and the role of amiodarone is currently being examined in the Nippon Study. Furthermore, an ICD that includes cardiac resynchronization therapy (CRT-D) is now available in Japan, a device that contributes to lowering the rate of sudden cardiac death, as well as improves the quality of life of patients with depressed LV function.

References

L et al. radiofrequency catheter ablation as a cure for idiopathic tachycardia of both left and right ventricular origin. J Am Coll Cardiol 1994; 23; 1333 – 1341.


Washizuka T, Chinushi M, Watanabe H, Hosaka Y, Komura S, Sugihara H, et al. Nifekalant hydrochloride suppresses severe electr-