Advances in nonpharmacologic therapies for ventricular arrhythmias

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Ventricular tachycardias differ in their clinical course and prognosis, as this group includes arrhythmias of diverse etiologies. Idiopathic ventricular tachycardias have good prognosis and catheter ablation is an alternative for long-term antiarrhythmic drug treatment. In some patients, sophisticated mapping systems and approaches (pericardial and surgical) have to be used. Ventricular tachycardias related to structural heart disease are more difficult to ablate due to the diffuse character of underlying disease and in most cases with ventricular dysfunction, an option to use an implantable cardioverter–defibrillator should be considered. Recent studies have shown that implantable cardioverter–defibrillators are the only effective option for secondary prevention of sudden cardiac death. Primary prevention of sudden cardiac death is most effective when an implantable cardioverter–defibrillator is implanted; however, this approach is costly and needs elaborating criteria for risk stratification. Optimal treatment of underlying heart disease and correction of risk factors will help to decrease the number of patients suffering from VTs and dying suddenly.

Keywords: catheter ablation, implantable cardioverter–defibrillators, ventricular tachycardia

Ventricular tachycardias (VTs) often present as a clinical emergency and are the main mechanism of sudden cardiac death (SCD). A small number of currently available antiarrhythmic drugs and their limited efficacy to treat VT shown by clinical trials [1], led to a rise in numerous nonpharmacologic approaches to VT management. The purpose of this review is to highlight the current status of nonpharmacologic therapies for VT.

Establishing the diagnosis of VT

VT is defined as three or more consecutive premature beats originating in the ventricles. The width of QRS complexes is more than 120 ms due to ventricular origin. VT should be differentiated from other rhythms that may present with wide QRS complexes (supraventricular arrhythmias with bundle branch block aberration and pre-excitation). Certain diagnoses of rhythm origin (ventricular vs supraventricular) are sometimes difficult to establish, and an intracardiac electrophysiologic study may be indicated. In everyday practice, especially in acute settings, it is necessary to rely on the electrocardiographic and clinical signs to make the decision and administer appropriate therapy. It is useful to remember that VT constitutes approximately 80% of wide QRS complex tachycardias and even more (~95%) in emergency cases with wide QRS complex tachycardias.

There are many known algorithms for the differentiation between VT and supraventricular tachycardias with wide QRS complexes. The latest guidelines suggest analyzing the 12-lead electrocardiograph (ECG) and clinical history along with the effects of vagal maneuvers and adenosine [2]. Very important criterion such as atrioventricular (AV) dissociation are quite often missed on 12-lead ECG; however, its presence or absence can be verified using other easily applicable techniques such as transesophageal recording of atrial activity. A diagnostic approach to the wide QRS complex tachycardia is presented in **Figure 1**.

Classification & mechanisms of VT

VTs can be classified using many criteria including etiology, electrophysiologic mechanisms and ECG and clinical features – universal classification including all of the criteria would look cumbersome. Thus classifying VTs into idiopathic and related to structural heart disease is practical from clinical and electrophysiologic points of view.

The substrate of idiopathic VTs is limited to a small area in which a triggered activity or the re-entrant circuit occurs. Ablation of this limited area is often effective to cure the patient and these VTs have a good prognosis even if untreated.

VTs related to structural heart disease more often have multiple substrates due to diffuse myocardial changes (**Figure 2**). The mechanism in this group is usually re-entry that may use large

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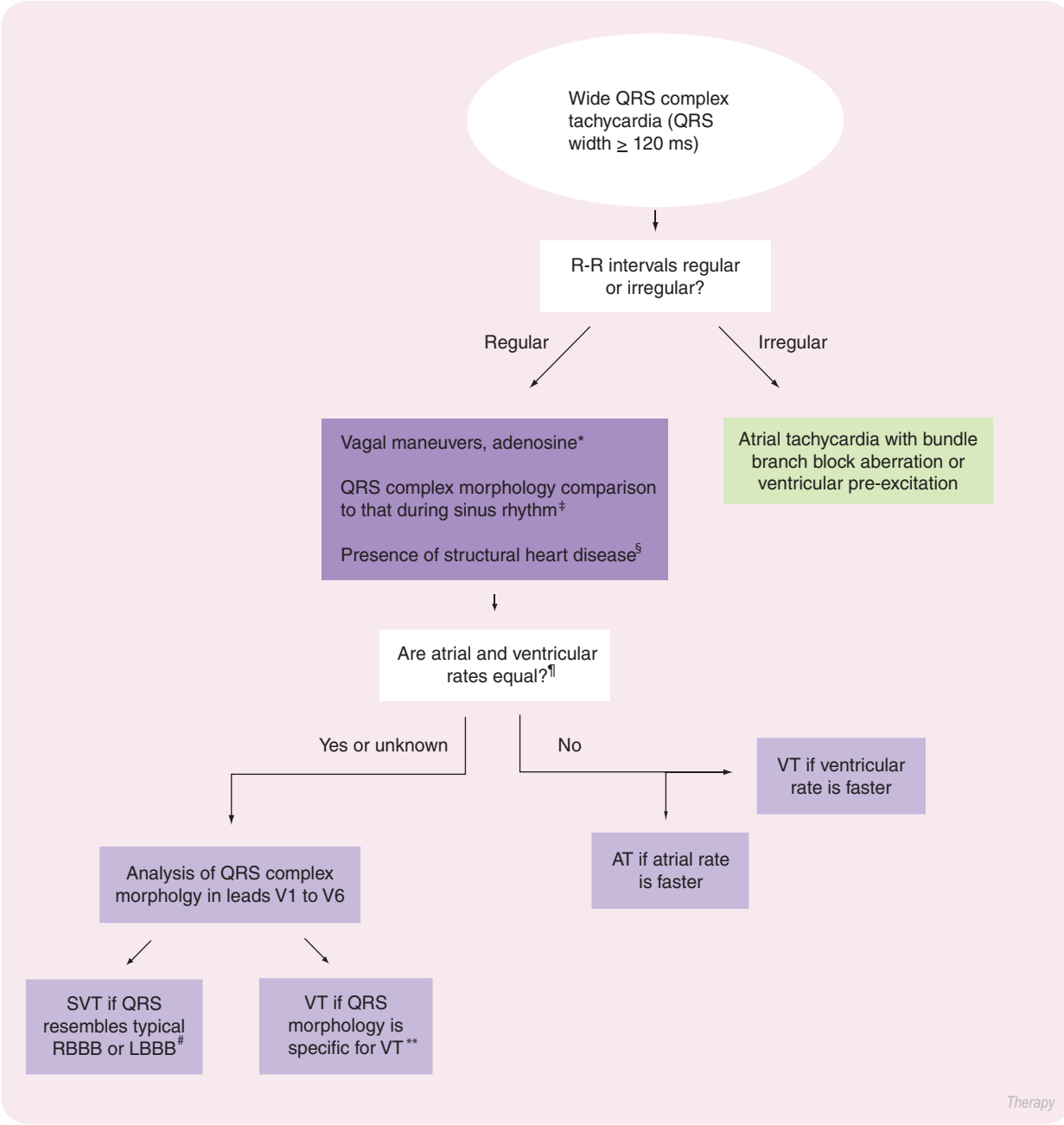
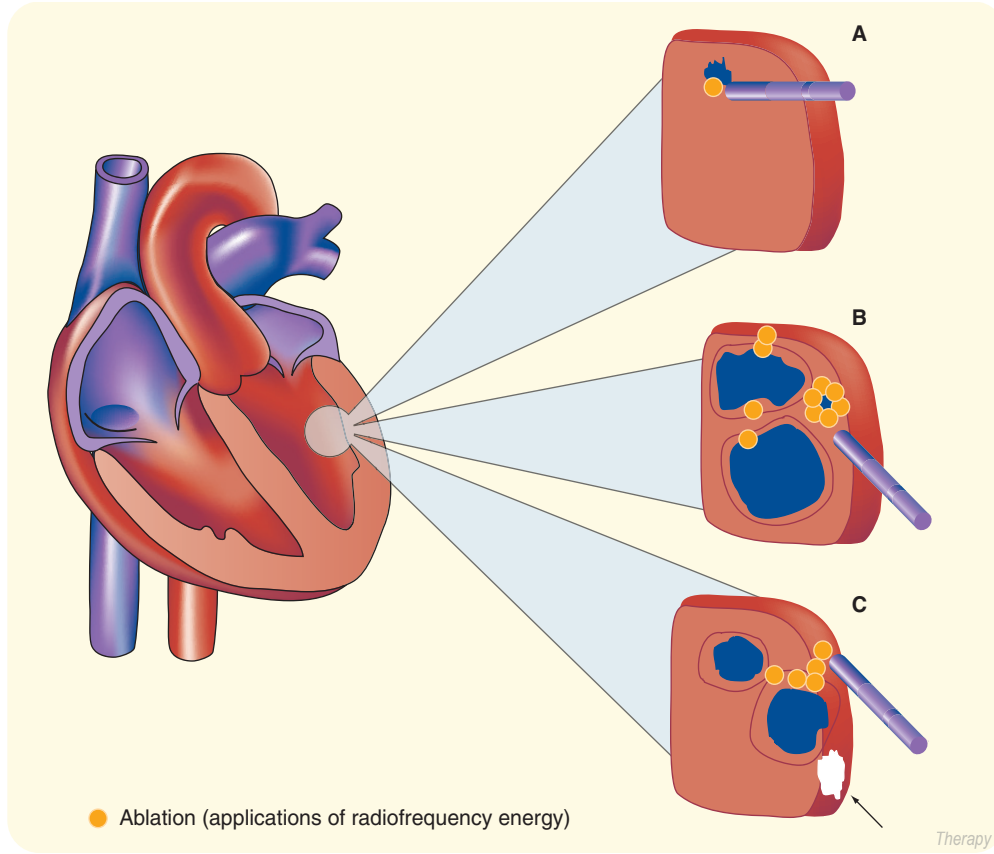
Figure 1. Diagnostic algorithm to differentiate ventricular tachycardia from other wide QRS complex tachycardias.



*Tachycardia termination suggests SVT.
‡Identical QRS morphology suggests SVT, but bundle-branch re-entry may be possible.
§Presence of structural heart disease suggests VT diagnosis.
¶This could also be verified using transesophageal or endocardial recording ot atrial activity, or clinical signs of atrioventricular dissociation.
#Typical LBBB shape is still possible in bundle-branch re-entrant VT.
**This may be concluded if no typical LBBB or RBBB is seen, or there are particular QRS shapes specific for VT.
AT: Atrial tachycardia; SVT: Supraventricular tachycardia; LBBB: Left bundle branch block; RBBB: Right bundle branch block; VT: Ventricular tachycardia.
Adapted from [2].

Figure 2. Ablation procedure for ventricular tachycardias.



(A) Focal ventricular tachycardias are characterized by a single focus of arrhythmia that can be readily ablated. **(B)** Ventricular tachycardias related to structural heart disease may have multiple tachycardia circuits due to the presence of multiple zones of slow conduction. In these cases, substrate mapping and various lines of ablation may be used. **(C)** In some cases tachycardia circuits may be located deep in the myocardium or subepicardially. In addition, functionally determined zones of inhomogenous conduction (arrow) may appear and serve as a substrate for new forms of arhythmias.

area of myocardium or have a 3D path using deeper myocardial layers. For these reasons, linear ablation (rather than focal ablation) is necessary to block the spread of re-entrant activation. The prognosis is more serious, especially if ventricular function is depressed, because VT more quickly causes hemodynamic instability, and degenerates into a faster form or ventricular fibrillation.

Ablation techniques

Nonpharmacologic curative approaches to VT started with surgery for VT related to postinfarction scars [3,4]. Encircling ventriculotomy and subendocardial resection have been accompanied by cryoablation when necessary. Catheter ablation of VT has been used with direct current shocks applied through endocardial electrode catheters [5–7]. However, this technique has limited application due to the necessity of anesthesia and possible risks of barotrauma and cardiac perforation.

Endocardial catheter ablation of VT began to evolve rapidly when radiofrequency energy was introduced into clinical practice. This procedure uses standard-sized (tip of 4 mm length) deflectable catheters; however, as larger amounts of energy have to be delivered to create larger or deeper (transmural) lesions, 8 mm or irrigated-tip (also called cool-tip) catheters may be used. These catheters are introduced into the right or left ventricle under local anesthesia together with several diagnostic catheters (usually positioned in the right atrium and His bundle area). To guide the ablation catheter to the target area, different criteria can be used depending on the mechanism and substrate of the VT, including:

- Activation mapping which aims to find areas with the earliest local activation time compared with the onset of the QRS complex (-25 ms or earlier is preferable). This technique is appropriate to find the foci of idiopathic VT, and is, as an adjunct to map substrates in VTs, related to structural heart disease
- Pace mapping involves matching the shape of paced QRS complexes to that of spontaneous arrhythmia, which may prove useful if the arrhythmia is nonsustained
- Entrainment mapping is a method of pace mapping applied during ongoing tachycardia. It is useful during ablation of VTs related to structural heart disease and evaluates both the shape of QRS complexes and the distance between the pacing spike and the onset of the QRS complex
- Substrate mapping is used to search zones of abnormal electrical activity (scars, low voltage and delayed electrical activity) during the sinus rhythm [8]. This may be necessary because VTs related to structural heart disease may cause hemodynamic instability and cannot therefore be mapped directly [9]

Noncontact mapping in patients with poorly tolerable VTs may also be used [10]. The EnSite™ system uses a multielectrode balloon placed into the ventricle and reconstructs the electroanatomic map from the single beat of tachycardia. A number of possible difficulties may arise during the ablation procedure and these are listed in **Table 1**, together with their possible solutions.

Catheter ablation of particular forms of VT

Idiopathic VTs

Idiopathic VTs occur (by definition) in people without detectable heart disease or genetically determined molecular abnormalities, and if symptomatic, can be treated using catheter ablation. Idiopathic VTs comprise only 10% of all VTs and are not a large clinical problem compared with VTs related to structural heart disease. However, catheter ablation is rewarding as it avoids lifelong medical therapy and often cures the patients, returning them to a normal life. Details pertinent to catheter ablation of idiopathic VTs are listed in **Table 2**.

Table 1. Possible difficulties during ablation of ventricular tachycardias.

Type of arrhythmia	Possible difficulties	Solutions
VTs related to structural heart disease	Complex anatomy of the substrate	3D reconstruction of the electroanatomic map during VT (CARTO™, EnSite™ NavX™ systems)
	Hemodynamic instability during VT	Single-beat electroanatomic reconstruction of the heart chamber during VT (EnSite system)
		Substrate mapping/ablation during the sinus rhythm
		Antiarrhythmic drug administration to slow down the tachycardia
	Deep localization of arrhythmia circuits	8 mm or cool-tip catheters to create deeper lesions
		Epicardial approach
	Multiple substrates/types of VT	Substrate mapping/ablation
	Noninducibility during ablation	Substrate mapping/ablation
Idiopathic VTs	Noninducibility or nonsustained character of VT	Intravenous infusion of isoproterenol to induce/facilitate the arrhythmia
		Pace-mapping if VT is still not inducible during ablation procedure

VT: Ventricular tachycardia.

Table 2. Forms of idiopathic ventricular tachycardias and details pertinent to catheter ablation.

Form of idiopathic VT	Clinical & electrocardiographic features	Remarks on catheter ablation
Right ventricular outflow tract tachycardia	Most often stress induced, adenosine and verapamil sensitive Left bundle branch block morphology with R-wave transition in leads V3–V4, and inferior axis deviation in frontal plane	Foci are located in the septal or free wall of the right ventricular outflow tract, sometimes above the pulmonary valve or subepicardially
Left ventricular outflow tract tachycardia	Adenosine sensitive Right or left bundle branch block morphology with R-wave transition in leads V2–3, and inferior axis deviation in frontal plane	Foci are located in the left or right coronary cusps of the aortic valve, or below the aortic valve, sometimes subepicardially
Left fascicular idiopathic tachycardia	Verapamil (not adenosine) sensitive	Ablation is guided by activation mapping and search for Purkinje potential in the septal part of the left ventricle
	Right bundle branch block with superior axis deviation (common, or posterior fascicular, form) Inferior axis deviation or other frontal QRS plane axis may be seen in other forms	Block in the corresponding fascicle (posterior fascicle in the typical form) is often seen after successful ablation

VT: Ventricular tachycardia.

Right ventricular outflow tract tachycardia

This VT is recognized as the most common idiopathic VT [11]. It can be terminated by vagal maneuvers, adenosine (this favors the probability of triggered cAMP-mediated activity), also β-blockers and verapamil. Catheter ablation of the right ventricular outflow tract (RVOT) VT has a high success rate of approximately 90% [12]. If there are multiple variants of QRS complexes in one patient, suspicion concerning the possible arrhythmogenic right ventricular cardiomyopathy (ARVC) may be raised [13], and other diagnostic tests (genetic analysis, magnetic resonance imaging) should be performed. The focus of this VT can be found using activation mapping or pace mapping, and intravenous infusion of catecholamines is often used to provoke the tachycardia and to test the effectiveness of ablation. Some foci could not be ablated from the right ventricle, and those showing a prominent R-wave in lead V1 were thought to be located subepicardially. It is known now that these foci may be ablated in the left ventricle (this group evolved into the entity of the left ventricular outflow tract tachycardia).

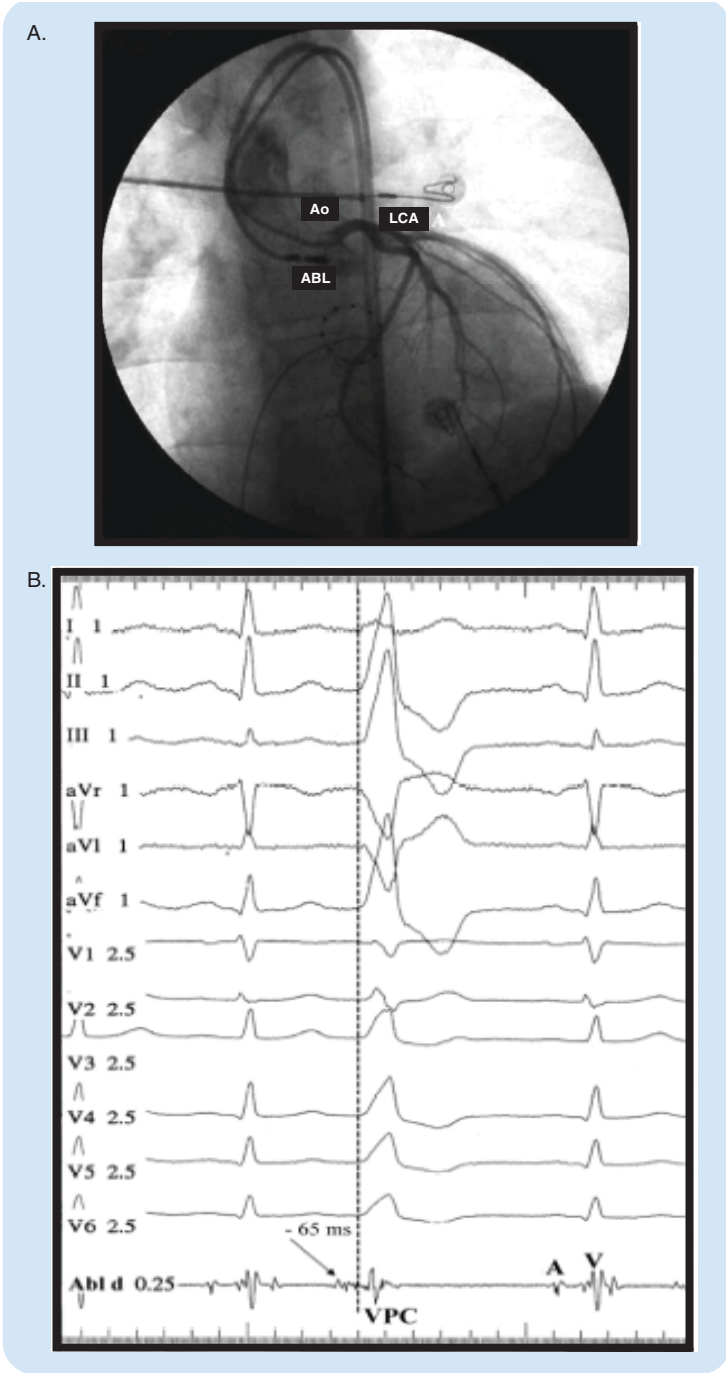
Left ventricular outflow tract tachycardia

Nonpharmacologic treatment of this form of VT developed relatively late compared with the RVOT group. Foci of left ventricular outflow tract tachycardia may be located either below or even above the aortic valve cusps [14]. Therefore, during ablation of this tachycardia in the aortic sinus of valsalva, caution has to be exercised not to damage the left main coronary artery and often two catheters have to be introduced via femoral arteries and positioned in the aortic root – one to cannulate and mark the coronary artery and another for ablation (**Figure 3**). There are still VTs with inferior axis deviation that cannot be ablated using the two previous approaches. A stepwise approach to these 'outflow tract' VTs has recently been described, including mapping in the right ventricular outflow tract, pulmonary artery, left ventricular outflow tract, coronary sinus branches and via pericardial puncture [15].

Left fascicular idiopathic tachycardias

Another common form of idiopathic VT is left fascicular (verapamil-sensitive) tachycardia. The common (interfascicular) form probably arises in the Purkinje network of the left posterior fascicle and is characterized by right bundle branch-type QRS morphology with superior axis deviation. Activation mapping shows the early ventricular activity at the site of successful ablation, and a high-frequency signal, a possible potential of Purkinje system, is often observed (or even two potentials, favoring the fact of reentry rather than triggered activity) [16]. Ablation at these sites is effective in around 90% and the left posterior hemiblock is often seen after successful ablation. Other variants of this VT can

Figure 3. Catheter ablation of left ventricular outflow tract tachycardia.



(A) Ablation catheter is positioned in the left coronary cusp of aortic valve and its distance to the left main coronary artery (~22 mm) is safe to attempt ablation.
(B) Ablation catheter detects both A and V signals. During VPC, early fractionated ventricular activity (-65 ms to the onset of QRS complex) is recorded (arrow).
A: Atrial; Ao: Aortic valve; AbL: Ablation catheter;
LCA: Left coronary artery;
V: Ventricular; VPC: Ventricular premature contraction.

arise in the left anterior fascicle. Interfascicular VT (re-entry using both fascicles of the left branch) is also possible.

Tachycardias associated with structural heart disease

VT related to postinfarction scars

This group is the most common form of VT encountered in clinical practice (80%), due to the spread of coronary heart disease. It remains to be discovered how necrosis in the area supplied by a damaged coronary artery can lead to a transitional area of surviving myocardial cells as well as areas of slow conduction. Mapping of these VTs is more complicated and entrainment pacing in the critical slow zone is the most reliable technique. Many patients tend to have several forms of postinfarction VT, some of them being very fast and poorly tolerated, thus not amenable to catheter ablation. Therefore, substrate mapping during sinus rhythm may be used, and areas of slow conduction may be connected by ablation lines to the mitral valve, or one to another (**Figure 2**) [17,18]. Substrate mapping during right ventricular pacing can also be performed [19]. This can additionally bring out the zones of functional conduction block (a possible substrate for re-entry) due to the changed direction of ventricular activation. Parietal thrombi near the ablation target can be dislodged by an ablation catheter and pose further dangers. The overall success rate of catheter ablation is lower than in idiopathic VTs – approximately 60%. Catheter ablation may be used as a single therapy if there are no risk factors for SCD, or as an adjunct therapy to avoid frequent discharges of an implantable cardioverter–defibrillator (ICD). In the absence of ICD, long-term follow-up of initially successful ablation of postinfarction VTs shows a long-term mortality of more than 10% [20]. Depressed left ventricular function is the main independent predictor of poor outcome.

Tachycardias in idiopathic dilated cardiomyopathy

In this group, VTs are often syncopal due to poor left ventricular function, and the effectiveness of catheter ablation is not high due to the diffuse and progressive character of the disease and multiple forms of VT [21]. Bundle branch re-entry tachycardia (see below) is often induced in these patients. ICD is the main option to prevent SCD in high-risk patients [22]. Ablation of zones of slow conduction is possible to decrease the frequency of ICD shocks [23].

Bundle branch re-entry VT

This form of re-entry is common in patients with depressed ventricular function and defects of interventricular conduction. Most commonly, activation spreads down the right bundle branch and ascends via the left bundle branch [21]. The shape of QRS complexes is indiscernible from the left bundle branch block aberration, but careful analysis of the ECG and clinical signs (i.e., atrioventricular dissociation) leaves few doubts concerning the ventricular origin. These tachycardias are usually fast and cause hemodynamic instability [24]. Ablation of the right bundle branch abolishes this VT; however, it does not eliminate the risk of SCD if other risk factors are present [25].

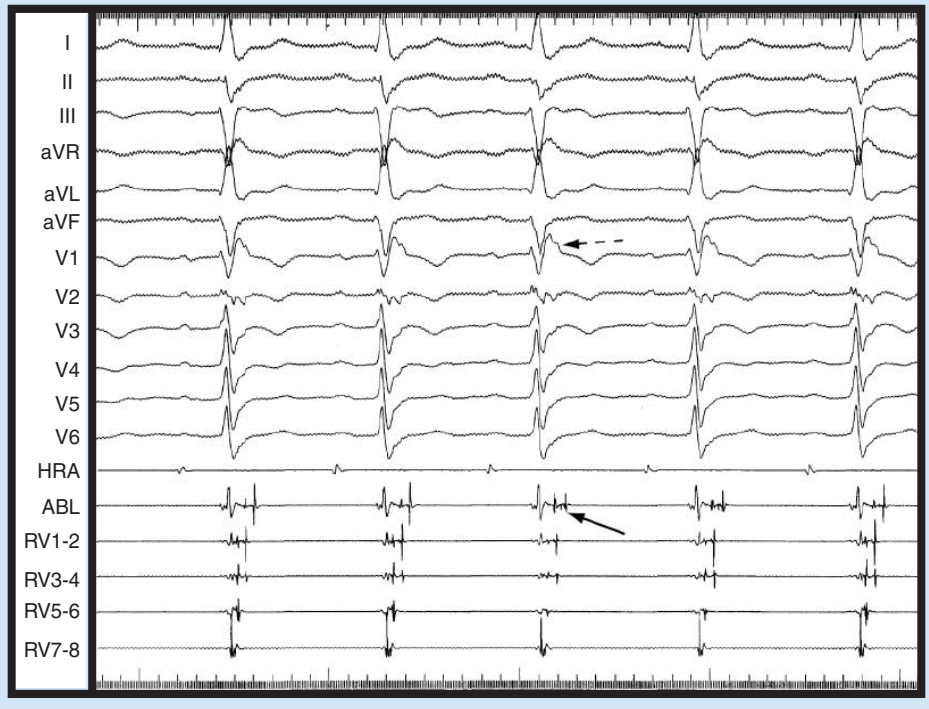
VTs in hypertrophic cardiomyopathy

Patients with hypertrophic cardiomyopathy have an increased risk of SCD caused by hypertrophy, disarraying of myocardium and dispersion of electrophysiologic properties. Polymorphic VTs and fibrillation may occur due to this inhomogeneity, and ICDs are considered a measure of secondary and primary SCD prevention [26]. Catheter ablation is not indicated in this group.

Arrhythmogenic right ventricular cardiomyopathy

A classic form of ARVC described by Fontaine is characterized by a dilated right ventricle and triad of bulges [27]. Usually multiple forms of sustained VT can be induced due to the diffuse character of the disease. Catheter ablation may be performed if VT paroxysms are frequent and substrate mapping usually reveals multiple zones of slow conduction due to fatty degeneration of ventricular myocardium and multiple zones of slow conduction (**Figure 4**). Surgery for VT associated with ARVC is now obsolete. Causes of SCD are described in patients with ARVC, therefore patients with aborted SCD or those with markers of high risk should receive an ICD [28]. There are patients without gross abnormalities of the ventricles who are still at risk [29] and

Figure 4. Substrate mapping in the right ventricular arrhythmogenic dysplasia.



12 standard ECG leads are presented together with the HRA and ABL electrograms, also RV electrograms (1–2 to 7–8). One of slow conduction zones found in the inflow of the right ventricle is characterized by the presence of delayed local activation (solid arrow). This is also seen on the surface ECG as ε-waves (broken arrow).
ABL: Ablation catheter; ECG: Electrocadiogram; HRA: Right atrial; RV: Right ventricular.

the development of molecular genetics should give more diagnostic criteria for the screening of such patients.

VT after surgery for congenital heart disease

The most common anomaly that gives rise to a VT substrate after surgical correction is tetralogy of Fallot [30]. The surgical procedure of complete repair creates scars (anatomic obstacles) in the outflow tract and in the ventricular septum, and VT may rotate around these scars. Catheter ablation is facilitated using various 3D electroanatomic reconstruction systems and ablation line is drawn from the right ventricular infundibulotomy scar to the pulmonary valve [31]. The SCD risk stratification is still unresolved in this group of patients; however, ICD implantation is necessary if the patient has been resuscitated.

VT related to primary electrical diseases

A number of gene defects responsible for cardiac myocyte ion channel dysfunction have been discovered. This group includes the long QT interval syndrome, Brugada syndrome, short QT interval syndrome, catecholaminergic polymorphic VT and others [32]. These anomalies create a dispersion of myocardial refractoriness and conduction, leading to a possibility of polymorphic VT and ventricular fibrillation. To date, the only option to prevent SCD in these patients is ICD implantation. However, some patients can experience multiple VT shocks as a result of frequent VT episodes ('electrical storm'). It has been noted that the trigger of polymorphic VT may be ventricular premature beats originating in one focus, and their ablation could decrease the number of VT episodes. Of note is that electrograms from these sites often show sharp presystolic spikes, probably potentials of the Purkinje system [33,34].

Epicardial approach to ablation

In some cases, the circuit of VT may be located closer to the epicardial surface of the heart and endocardial application of radiofrequency energy is not effective. This is most often observed in patients with VTs related to Chagas' disease; however, it may also be applicable to other forms of VT [35,36]. In these cases, the ablation electrode is introduced into the pericardial space using a pericardial puncture, and navigated to the site of interest. Caution has to be exercised not to damage the coronary arteries that run epicardially. The epicardial approach will probably be used more often since the experience of the epicardial approach to treat other rhythm disturbances (i.e., the pericardial approach to isolate pulmonary veins to treat atrial fibrillation) is widening and gained skills and techniques such as thoracoscopic visualization could be used for ablation in more VTs.

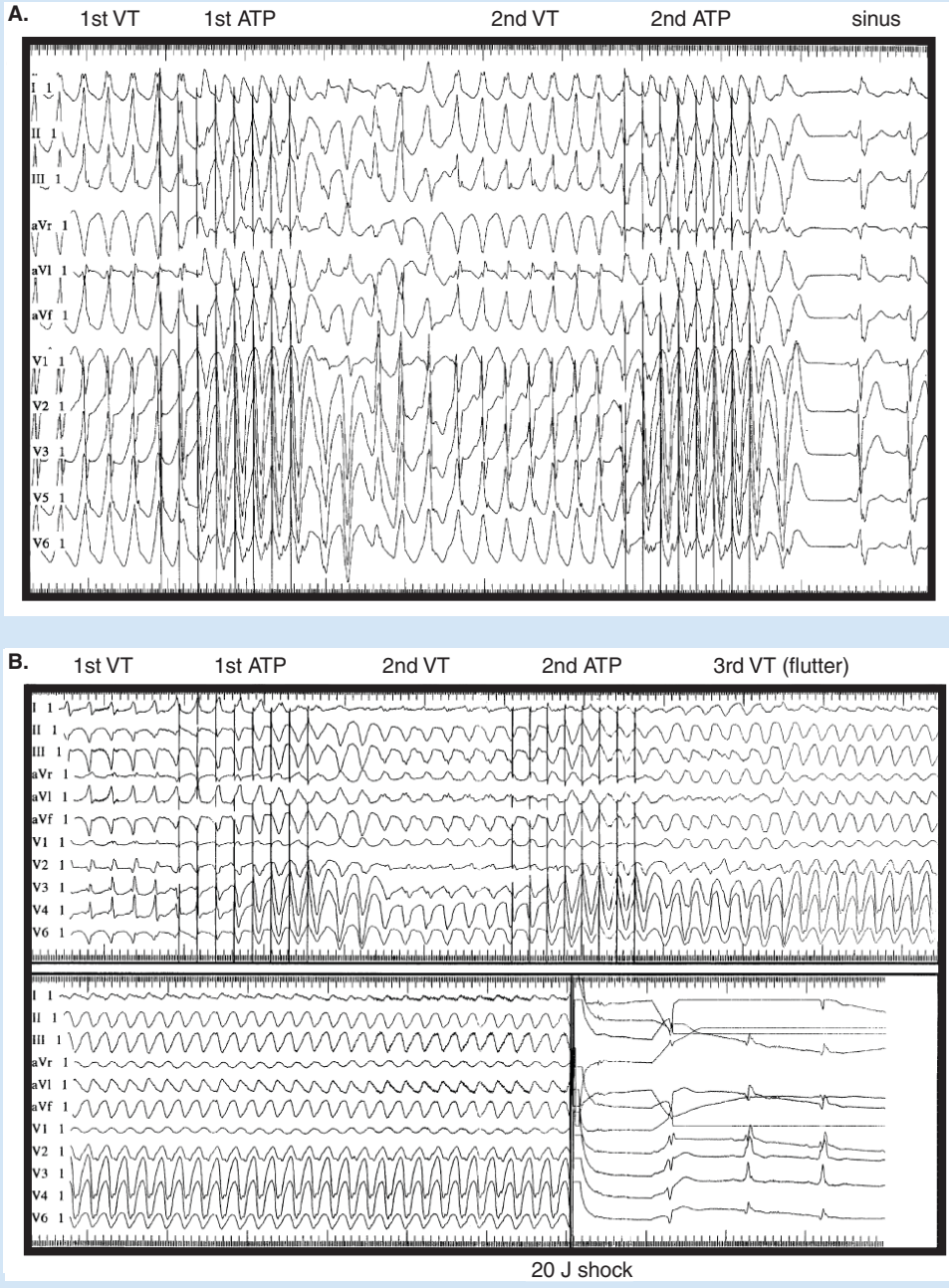
Surgical treatment for VT

The principles of mapping various cardiac arrhythmias were first learned during surgical procedures, and arrhythmia surgery was the predecessor of catheter ablation. Surgical isolation of the lateral wall of the right ventricle has been used for VT associated with ARVC; however, it has now been replaced by ICD, catheter ablation and heart transplantation [37]. Surgical treatment of VT related to ischemic heart disease had success rate (abolition of VT) of approximately 85%, with a mortality rate of 5 to 10%. Today, few centers have experience with mapping-guided VT surgery, and as a stand-alone procedure it has almost been abandoned as less traumatic and almost equally effective catheter ablation can be used, or ICD is indicated. However, surgical ablation of postinfarction VT can be performed concomitantly with other surgical procedures such as coronary revascularization, mitral valve repair, or restoration of the left ventricular geometry [38]. The latter procedure effectively abolishes VT even without mapping [39]. In the absence of other indications for cardiac surgery, patients with postinfarction VT are operated if other treatment modalities fail. VTs related to the diffuse involvement of myocardium (nonischemic cardiomyopathy) are not usually amenable to surgical treatment.

Sudden cardiac death & implantable cardioverters–defibrillators

Contemporary ICDs are capable of detecting multiple forms of VTs and ventricular fibrillation and administering appropriate therapy (**Figure 5**). These therapeutic measures (tiered therapy) range from bursts or ramps of antitachycardia pacing, R-wave synchronized shocks of small energy, to high energy shocks up to 30 to 35 J or 700 to 800 V. Advanced electrogram analysis and storage features allows therapy to be tailored to the patient. Diagnostic algorithms help to discriminate VTs from sinus and other supraventricular tachycardias and decrease the chance of inappropriate shocks. Implantation of contemporary ICD devices with endocardial

Figure 5. Tiered therapy delivered by an ICD.



(A) ICD detects VT and delivers an ATP burst. After this, tachycardia slightly changes (lead V2) but continues. The second ATP burst at slightly faster rate restores sinus rhythm. **(B)** Two continuous parts. Two bursts of ATP are not succesful and degenerate the VT to ventricular flutter (upper part). ICD detects this arrhythmia and delivers a 20 J shock that restores sinus rhythm (bottom part).
ATP: Antitachycardia pacing; ICD: Implantable cardioverter–defibrillator; VT: Ventricular tachycardia.

leads carries lower morbidity and procedure-related mortality, comparable with that which occurs with pacemaker implantation.

Guidelines for ICD implantation were first published in 1998, revised in 2002 and are corrected accordingly to the results of new trials [40–42]. In patients resuscitated from SCD due to VT, ICD is the only reliable option for secondary prevention [43,44]. This is reflected in recent recommendations of SCD management [45]. Trials such as the Multi-center Automatic Defibrillator Implantation

Trial (MADIT) I, Multicenter UnSustained Tachycardia Trial (MUSTT) and others have shown that ICDs are the most effective measure for primary SCD prevention in high-risk patients with VTs [46,47].

Recently, two large trials on primary SCD prevention in patients with depressed left ventricular function and without documented VT (MADIT II in patients with coronary heart disease and SCD-HeFT in nonischemic cardiomyopathy) have shown that prophylactic ICD implantation decreases overall mortality [48]. This shows that depressed left ventricular function is an independent predictor of a higher risk of SCD. However, if the expanded indications suggested by these trials are followed, the number of worldwide ICD implants will increase more than twice. This raises the debate of economic burden of expanded indications for ICD therapy, as indications for ICD implantation for primary prevention vary from country to country. ICDs prevent SCD but do not modify the underlying heart disease (like dilated cardiomyopathy) and in some patients overall life expectancy may not be prolonged significantly. The process of combinating ICDs with biventricular pacing in patients with heart failure is still controversial, and to date only the Comparison of Medical Therapy, Pacing and Defibrillation in Heart Failure (COMPANION) trial has shown an improvement in prognosis and quality of life (QoL) can be achieved [49,50].

The MADIT II and Dual Chamber and VVI Implantable Defibrillator (DAVID) trials have shown a bigger incidence of heart failure in ICD patients with a higher percentage of ventricular pacing. This is due to the fact that the right ventricular lead in these patients is in an apical position, and pacing from this site is less physiologic. Resulting ventricular dyssynchrony and mitral regurgitation worsens heart failure. According to these data, ventricular pacing should be used as little as possible in ICD patients with heart failure. Using the alternate pacing sites, or biventricular pacing, could be considered.

An important problem in view of the expanding number of ICD implants is that QoL is decreased as a result of the adverse psychologic effects of ICD shocks, regardless of whether or not they are appropriate. ICD shocks cause discomfort, anxiety and preoccupation. Recently reported results from the Optimal Pharmacological Therapy in Implantable Cardioverter Defibrillator patients (OPTIC) trial showed that amiodarone (especially in combination with a β-blocker) and to a lesser extent, sotalol or another β-blocker alone, decrease the number of ICD shocks and improve the wellbeing of the patients [51].

Approach to the patient with diagnosed VT

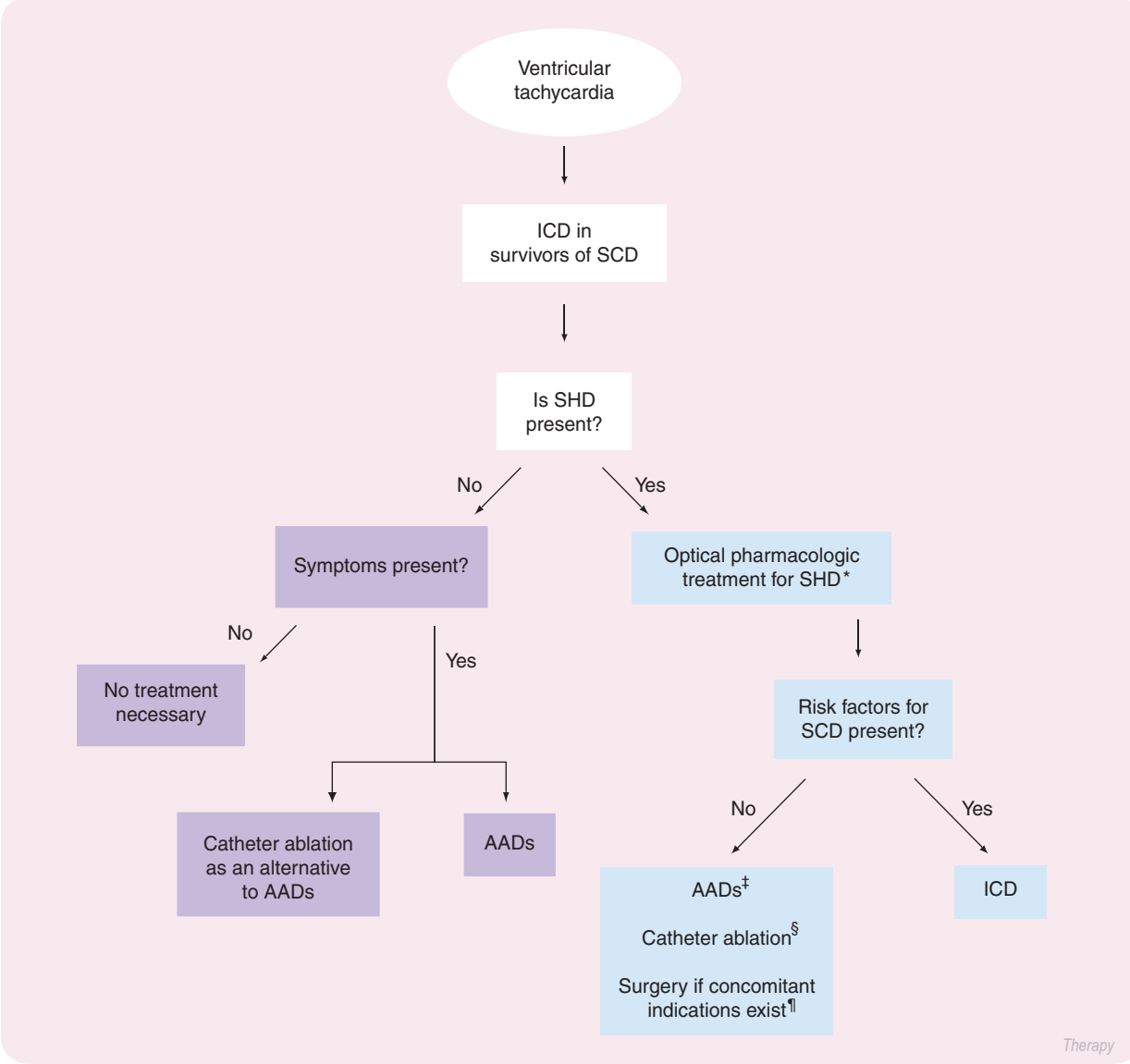
In patients with idiopathic VTs, no treatment may be used. If symptoms are present, antiarrhythmic drugs may be used or catheter ablation may be chosen to avoid long-term drug therapy. Unfortunately, some idiopathic VTs may be related to still poorly identifiable genetic disorders, such as some forms of arrrhythmogenic dysplasia.

Patients with VTs related to structural heart diseases and molecular abnormalities have an increased risk of SCD. Criteria for risk assessment are still yet to be discovered, of which the most important include poor ventricular function, QRS prolongation, and left ventricular hypertrophy [52]. These patients often have multiple forms of VTs, and ablation of one does not reliably decrease the risk of SCD. New areas of slow conduction leading to dispersion of electrophysiologic parameters (the cause of re-entry) may arise due to disease progression. Transient ischemia or catecholamine stress create new zones with different conduction time and refractoriness, leading to the possibility of polymorphic VT and ventricular fibrillation. Other factors that lead to inhomogeneity of ventricular muscle are fibrosis, edema, loss of intercellular coupling (connexins) and nerve sprouting.

Treating only high-risk groups is not effective in the prevention of SCD due to the relatively small number of people saved. Primary prevention in the general population is important for mortality decrease in the future, emphasizing the importance of healthy lifestyle, correcting hypertension, diabetes, smoking, obesity and other risk factors. Optimal pharmacologic treatment of underlying heart disease, for example, the use of β-blockers, angiotensin-converting enzyme inhibitors, statins and aldosterone receptor blockers is known to decrease the chance of arrhythmia [45,53]. Analysis of finished and future trials, as well as stratification of risk factors could provide the answer as to which patients could benefit from particular treatment modalities [54].

The algorithm of VT management is presented in **Figure 6**. Choosing the most appropriate treatment should include assessment of numerous clinical factors, and every patient with this diagnosis requires an individual approach.

Figure 6. Schematic approach to the management of patients with diagnosed ventricular tachycardia.



*Indication for primary prevention of SCD may vary from country to country.
‡Only b-blockers and amiodarone are recommended, other AADs do not influence or increase SCD risk.
§Catheter ablation is indicated as class IIb in sustained, hemodynamically tolerable VT that is refractory to drug treatment [45,53].
¶Concomitant indication includes revascularization, restoration of left ventricular geometry, valvular surgery and aneurysmectomy.
AADs: Antiarrhymic drugs; ICD: Implantable cardioverter–defibrillator; SCD: Sudden cardiac death; SHD: Structural heart disease.

Expert commentary

Ventricular arrhythmias describe the heterogeneous group of rhythm disturbances, varying from benign to lethal. It is known that patients with poor ventricular function have the highest risk of sudden death. Primary and secondary prophylaxis of sudden death by cardioverter–defibrillator implantation and drug therapy is clearly indicated in high-risk patients, but the latter represent only a minor part of total sudden death numbers. Expanded indications for primary sudden death prevention by cardioverter–defibrillator implantation could not only cause considerable economic strain, but also give rise to a higher rate of perioperative complications and problems related to long-term lead performance. Newer techniques like subcutaneous lead placement could solve these problems. Conversely, some patients with supposedly

benign arrhythmias, such as right ventricular outflow tract premature beats, die suddenly after a reassuring consultation with a cardiologist offering no therapy. The search for other factors to clarify the high-risk patients therefore remains important.

Outlook

Until newer pharmacologic and gene therapy approaches are developed for prophylaxis and treatment of heart diseases, nonpharmacologic approaches to VT remain the most effective measure for the prevention of SCD and symptomatic treatment of VT. Catheter ablation remains a very effective method and an excellent alternative to drug treatment of idiopathic VTs. In VTs related to structural heart disease, hybrid approach is needed, including optimal medical therapy for underlying disease, antiarrhythmic drugs and catheter ablation. ICD devices will evolve and will be implanted widely for primary prevention in high-risk patients, yet more criteria of high risk have to be defined.

Highlights

- Ventricular tachycardias (VTs) may be subdivided into idiopathic and those related to structural heart disease. The two groups differ both in prognosis and selection of treatment.
- Mapping and ablation techniques have enabled the creation of electroanatomic maps of both ventricles in an attempt to find and ablate arrhythmogenic foci and zones, both endocardially and epicardially. Irrigated-tip and 8-mm tip catheter radiofrequency ablation is able to create lesions of sufficient depth.
- Idiopathic VTs could be ablated in majority of cases (effectiveness >90%) and improve the quality of life of patients. However, idiopathic VTs comprise only 10% of all VTs.
- VTs related to structural heart disease tend to have multiple forms, and ablation is possible to eliminate approximately 60% of arrhythmias. Patients with depressed ventricular function and other markers of high risk are often candidates for ICD implantation. Catheter ablation could be used as adjunct to avoid frequent ICD shocks.
- Surgery for postmyocardial infarction VTs still has a place, when combined with revascularization procedures, mitral valve repair and restoration of the left ventricular geometry.
- ICDs have been shown as the only reliable option for secondary prophylaxis of sudden cardiac death (in resuscitated patients). The superiority of ICDs for primary prophylaxis of SCD has also been shown, but widespread use of ICDs poses significant financial and organisational issues.
- Ventricular pacing in patients with implanted ICDs often worsens the heart failure. Possible solutions could be programming the ICD to lower rates, or use devices capable of biventricular pacing.
- Psychologic problems in patients with implanted ICDs can be decreased using optimal medical therapy to reduce the number of VT episodes.

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