Expert consensus on acute management of ventricular arrhythmias – VT network Austria ☆

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ABSTRACT

The Arrhythmia Working Group of the Austrian Society of Cardiology (ÖKG) has set the goal of systematically structuring and organizing the acute care of patients with ventricular arrhythmias (VA), i.e. ventricular tachycardia (VT) or ventricular fibrillation (VF) in Austria. Within a consensus paper, national recommendations on the basic diagnostic work-up of VA (12-lead ECG, medical history, family history, laboratory analyses, echocardiography, search for reversible causes, ICD interrogation), as well as further medical treatment and therapeutic measures (indication of coronary angiography, ablation therapy) are established.

Since acute ablation of VT is indicated in the current ESC guidelines as a class IB indication for scar-associated incessant VT or electrical storm (ES; ≥ 3 ICD therapies in 24 h) as well as for ischemic cardiomyopathy (iCMP) with recurrent ICD shocks, organizational measures must be taken to ensure that these guidelines can be implemented. Therefore, a VT network will be established covering all areas in Austria, consisting of primary and secondary VT centers. Organizational aspects of an acute VT network are defined and should subsequently be implemented by the participating hospitals. All electrophysiologic centers in Austria that deal with VT ablation are to be integrated into the network in the medium-term. Centers that co-operate in the network are divided into primary and secondary VT centers according to predefined criteria.

Contents

1. Introduction

The Arrhythmia Working Group of the Austrian Society of Cardiology (ÖKG) has set the goal of systematically structuring and organizing the acute care of patients with ventricular arrhythmias (VA), i.e. ventricular tachycardia (VT) or ventricular fibrillation (VF) in Austria[1]. As a first step, a consensus paper based on the model of ES NET BAVARIA (Electrical Storm Network Bavaria)[2] is presented. Herein national recommendations on the basic diagnostic work-up of VT (12-lead ECG, medical history, family history, laboratory analyses, echocardiography, search for reversible causes, ICD interrogation), as well as further medical treatment and therapeutic measures (indication of coronary angiography, ablation therapy) are established.

In contrast to the rate of implantable cardioverter defibrillator (ICD) implantation, where Austria appears within the highest implantation rate among Western European countries in the recent EHRA White Book, Austria is numerically among the three Western European countries with the lowest rate per million inhabitants in VT ablation[3]. Since acute ablation of VT is indicated in the current ESC guidelines as a class IB indication for scar-associated incessant VT or electrical storm (ES; $\geq$ 3 ICD therapies in 24 h) as well as for ischemic cardiomyopathy (iCMP) with recurrent ICD shocks, organizational measures must be taken to ensure that these guidelines can be implemented[4]. In addition, there is a class IIa indication for ablation after the first sustained VT with iCMP, a class I recommendation for bundle branch reentry tachycardias, a IIa indication

for therapy-refractory arrhythmogenic right ventricular cardiomyopathy (ARVC), IIb for other non-ischemic cardiomyopathies and Brugada syndrome with VT or ES[4].

Classes of recommendations and levels of evidence are given according to the ESC guidelines[4].

2. Initial management in patients with ventricular tachycardia or electrical storm

All recommendations on initial management and acute diagnostics are summarized in Fig. 1 and Table 1. A 12-lead ECG should immediately be recorded in all patients presenting with recurrent ICD shocks or with palpitations due to suspected VT. The initial ECG is essential for the differentiation of ventricular and supraventricular arrhythmias, as well as for the localisation of ventricular tachycardias. Even if VT terminates before the ECG can be recorded, essential information can be drawn from the resting ECG (signs of structural heart disease, acute coronary syndrome, channelopathies, possible triggers, morphology of ventricular premature beats as indicators of source or exit of ventricular tachycardia)[5].

In hemodynamically unstable or unconscious patients with broad complex tachycardia, advanced cardiac life support including electrical cardioversion is indicated[4,6]. In hemodynamically stable patients with monomorphic broad complex tachycardias, vagal manoeuvres can be performed[6]. If this is ineffective in
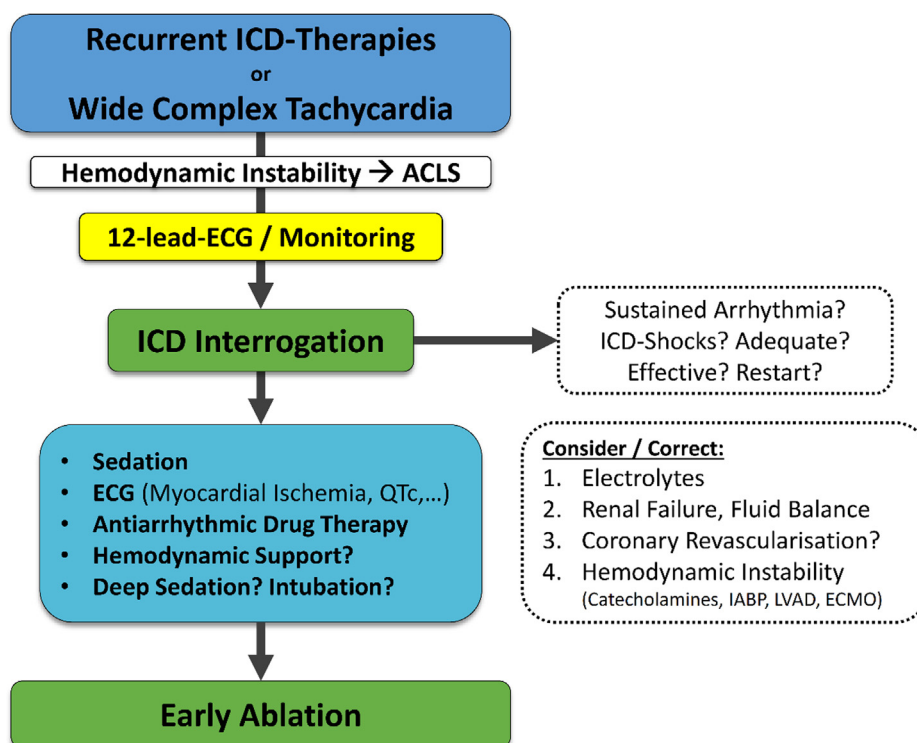

Fig. 1. *Initial Approach and acute diagnostics* in case of ventricular tachycardia and electrical storm. ACLS = advanced cardiac live support, IABP = intra-aortic balloon pump, LVAD = left ventricular assist device, ECMO = extracorporeal membrane oxygenation.

Table 1

Recommendations on initial management and acute diagnostics. VT = ventricular tachycardia; VF = ventricular fibrillation; SCD = Sudden cardiac death.

Recommendations – Initial Approach / Acute Diagnostics	Class	Level	Ref.
12 lead ECG (tachycardia and resting ECG)	I	C	[4]
Antiarrhythmic Drug Therapy (Table 2)			
Electrical cardioversion if drug therapy fails or if patient is hemodynamically unstable.	I	A	[9–12]
Interrogation of the implantable intracardiac pacing / defibrillation device, if present, should be performed as soon as possible, to evaluate the arrhythmia and delivered therapies.	I	C	[4]
A transthoracic echocardiographic study should be conducted in every patient with VTs, after the arrhythmia has been treated accordingly.	I	B	[4,13,14]
In patients with refractory electrical storm despite adequate therapy, a transfer to an intensive care unit with hemodynamic and electrocardiographic monitoring (12 lead ECG monitoring if possible) and deep sedation up to general anaesthesia is indicated.	I	C	[4,15,16]
An acute complete invasive cardiac evaluation should be considered in patients with polymorphic VT / VF or survivors of SCD, as well as in patients with unstable hemodynamics, cardiogenic shock, or persistent angina pectoris symptoms.	I	C	[4,8,17]
An electrophysiology center from the Austrian VT Network should be contacted early in the treatment and the patient transferred if necessary.	I	C	[4,18,19]

terminating the tachycardia, adenosine can be administered. Adenosine terminates AV-node-dependant supraventricular tachycardias with bundle branch block or preexcitation and rare Adenosine-sensitive idiopathic ventricular tachycardias. However, adenosine should not be administered in patients with structural heart disease due to the risk of cardiac arrest requiring resuscitation. The next therapeutic step is the administration of antiarrhythmic drugs (i.e. amiodarone or ajmaline, see chapter Medical Therapy). If antiarrhythmic drug therapy does not terminate VT, electrical cardioversion is indicated[4,6].

Patients who are already equipped with an ICD should be interrogated as soon as possible to assess the arrhythmia load and reveal, whether delivered ICD therapies were adequate and effective or whether VT outside of therapy zones are detectable. If VT persists, it can be terminated using the implanted pulse generator by ATP (antitachycardia pacing) or by forcing an internal shock.

Further diagnostic steps include echocardiography to assess left ventricular function and to exclude structural heart disease as well as laboratory testing to exclude potentially reversible proarrhythmic causes such as electrolyte imbalance (potassium, magnesium).

Polymorphic ventricular tachycardia may result from transient myocardial ischemia, whilst monomorphic ventricular tachycardia is most often caused by scar-related re-entry. Thus, coronary angiography may be indicated in case of polymorphic ventricular tachycardias, ventricular tachycardias in the context of acute coronary syndromes or in case of progression of ischemic cardiomyopathy. However, revascularisation alone is not effective in preventing recurrence of monomorphic ventricular tachycardias[5,7]. Apart from revascularization in acute myocardial infarction, coronary angiography is indicated in persistent symptoms of ischemia or in the following clinical scenarios: Hemodynamic instability, cardiogenic shock, persistent angina refractory to medical therapy, life threatening arrhythmia, cardiac arrest, or acute heart failure[8]. Heart failure drug therapy including betablockers, angiotensin converting enzyme inhibitors, angiotensin receptor blockers, mineralocorticoid inhibitors and the angiotensin receptor neprilysin inhibitor have been shown to reduce sudden cardiac death in heart failure patients, and should therefore be uptitrated after initial stabilizing period.[84,85]

Patients in electrical storm (≥3 VT or VF episodes in 24 h in patients without ICD or ≥ 3 appropriate ICD therapies for VT/VF in 24 h), with incessant VT or high risk of recurrent VTs should be monitored in a specialized unit. If available, they should be monitored with a 12-lead ECG telemetry system. A centre specialized in VT ablation should be contacted early.

The primary goal of the therapy in patients with electrical storm is initial stabilization and early ventricular tachycardia ablation[4]. Initial therapy includes sedation, antiarrhythmic drug therapy, and heart failure therapy including recompensation. In later stages, deep sedation, intubation, or mechanical cardiac support, e.g. with Impella, left ventricular assist devices (LVAD) or extracorporeal membrane oxygenation (ECMO), might be necessary.

3. Antiarrhythmic drug therapy

If a patient in ES cannot be stabilized by electrical cardioversion or defibrillation, a corresponding drug-based antiarrhythmic therapy should be initiated in parallel. When selecting and dosing, it has to be emphasized that antiarrhythmic drugs – particularly in patients with structural heart disease – can also be proarrhythmogenic in up to 7% of patients[20]. Since these side effects can be triggered by electrolyte disturbances, a balanced electrolyte status (calculated for a target serum potassium of 4.5 to 5.5 mmol / L and highly normal serum magnesium) should be targeted before the application of antiarrhythmic drugs.

The occurrence of recurrent symptomatic ventricular arrhythmias with possibly associated defibrillator therapy deliveries inevitably leads to a significant increase in sympathetic tone, which in turn can further provoke short-term recurring arrhythmias.

For this reason, blunting the increased adrenergic tone through the administration of betablockers is certainly the cornerstone of drug-based antiarrhythmic therapy[21]. Since they can better break through the adrenergic tone due to their central effectiveness, unselective betablockers such as propranolol have a certain advantage in the setting of an ES[22]. If a patient is hemodynamically compromised due to a reduced left ventricular ejection function, very short acting betablockers are also suitable due to their better titratability[23].

Further antiarrhythmic therapy depends on the type of VT and underlying disease as shown in Fig. 2.

In addition to betablockers, amiodarone is the second cornerstone of antiarrhythmic drug therapy in the context of ventricular arrhythmias, provided there is no manifest hyperthyroidism or prolonged QT time. The antiarrhythmic effect of amiodarone is heterogeneous with primarily class III effects (prolongation of the ventricular refractory period by delaying the potassium outflow) when taken orally and primarily class I (inhibition of sodium inflow), II (beta-blocking effect) and IV (L-calcium channel Blockers) when given intravenously [24]. The combined administration of beta blockers plus amiodarone is clearly superior to the sole administration of beta blockers [83]. It has been shown recently that repeated loading with amiodarone in patients with ischemic cardiomyopathy and recent ventricular arrhythmias has no benefit in terms of freedom from arrhythmias [25]. However, this study population did not specifically include patients in ES. Thus,
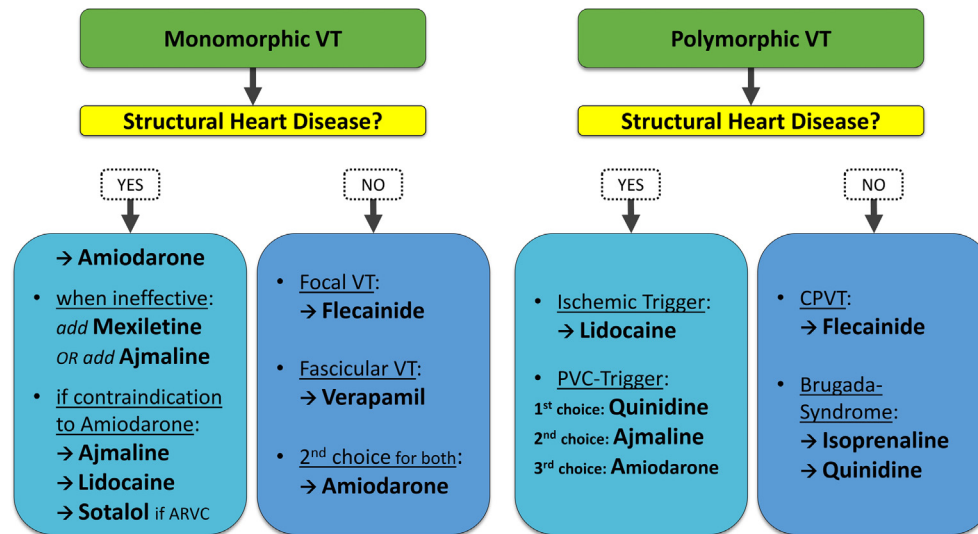
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┌─────────────────────┐          ┌─────────────────────┐
│    Monomorphic VT    │          │    Polymorphic VT    │
└─────────────────────┘          └─────────────────────┘
           │                                 │
           ▼                                 ▼
┌─────────────────────┐          ┌─────────────────────┐
│ Structural Heart     │          │ Structural Heart     │
│ Disease?             │          │ Disease?             │
└─────────────────────┘          └─────────────────────┘
      YES        NO                    YES        NO
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Monomorphic VT – Structural Heart Disease?

YES:
- → **Amiodarone**
- when ineffective: *add* **Mexiletine** *OR add* **Ajmaline**
- if contraindication to Amiodarone:
  - → **Ajmaline**
  - → **Lidocaine**
  - → **Sotalol** if ARVC

NO:
- Focal VT: → **Flecainide**
- Fascicular VT: → **Verapamil**
- 2nd choice for both: → **Amiodarone**

Polymorphic VT – Structural Heart Disease?

YES:
- Ischemic Trigger: → **Lidocaine**
- PVC-Trigger:
  - 1st choice: **Quinidine**
  - 2nd choice: **Ajmaline**
  - 3rd choice: **Amiodarone**

NO:
- CPVT: → **Flecainide**
- Brugada-Syndrome:
  - → **Isoprenaline**
  - → **Quinidine**

Fig. 2. *Specific antiarrhythmic therapy*. Especially in structural heart disease antiarrhythmic therapy should be combined with betablockers as baseline drug therapy. PVC = premature electrical contraction, CPVT = catecholaminergic ventricular tachycardia.

amiodarone can certainly be given in the acute setting of an ES, even if the patient is already on this drug as a long-term medication. The known side effects of long-term administration of amiodarone such as liver dysfunction, thyroid dysfunction, pulmonary fibrosis, and corneal deposits obviously only play a minor role in the acute phase [26].

In contrast to Amiodarone, intravenous Lidocaine (class IB) is an antagonist of the fast sodium channel with lower potency than amiodarone to reduce scar-dependent ventricular tachycardias. However, this drug is recommended as a reserve antiarrhythmic drug when amiodarone is not effective or contraindicated. Especially in the scenario of ischemia-triggered ventricular arrhythmias it has a potent antiarrhythmic effect[4].

Mexiletine is also a class IB antiarrhythmic (oral administration) and is administered as a reserve drug which is used in addition to amiodarone [27]. It has recently been shown that this measure does not contribute to a significant reduction in arrhythmias; however, this analysis was not carried out in a patient population in ES [25]. Apart from that, mexiletine is still used as a reserve drug for long QT syndrome, where it can help reduce the QT time, especially in long QT syndrome 3.

Since the class IC antiarrhythmic drugs flecainide and propafenone have shown increased mortality in patients with structural heart disease, they are contraindicated as long-term therapy in this group of patients[17]. However, they might be used in patients where ES is driven by a stable monomorphic ventricular tachycardia in the absence of structural heart disease (such as idiopathic ventricular tachycardias[4]). Furthermore, they might serve as a bail-out drug strategy in selected patients with structural heart disease, preferentially equipped with an ICD.

The same applies to the calcium antagonist verapamil (class IV antiarrhythmic). This drug is also contraindicated in patients with severely impaired systolic function, but it can be used in the absence of this contraindication (LVEF > 40%). Idiopathic VT, in particular fascicular VT, might be verapamil sensitive and can present as ES [17].

A fulminant form of ES is recurring torsade de pointes tachycardia or ventricular fibrillation triggered by R-on-T phenomenon (very short coupled PVC, superimposed on the T wave), or rapid ventricular tachycardia. The underlying causes might be, for example, Brugada syndrome, idiopathic ventricular fibrillation, or coronary heart disease without an acute ischemic event. For all these entities it could be shown that the class I antiarrhythmic drug quinidine can suppress these premature ventricular contractions (PVC) and thus prevent an ES caused by PVCs [28–30].

Ajmaline is a class IA antiarrhythmic drug, which is used both in the suppression of ventricular and supraventricular arrhythmias. Because of its short half-life it has beneficial pharmacokinetic properties and is therefore easy to use. Unfortunately, available data on its efficacy to reduce VA or ES is only based on older studies, case series or on personal experience, since this drug is rarely used internationally [34,35]. Especially in Anglo-American countries, procainamide is given as an alternative, which has the disadvantage of serious side effects such as drug-induced Lupus and changes in blood counts. Ajmaline should therefore only be used as a second-line therapeutic agent but can certainly be applied in ES to augment a continuous amiodarone infusion [31–33].

Another drug, not classified as an antiarrhythmic agent, but useful in the treatment of electrical storm in specific conditions, is isoproterenol. It is a selective β1 and β2 adrenergic receptor agonist. Commonly used to increase heart rate in 3rd degree atrioventricular block, it also shows efficacy in suppressing ES and ventricular fibrillation in Brugada syndrome, short QT syndrome and early repolarization syndrome[86–89]. This is thought to be enabled partly by accelerating heart rate and thereby suppressing VPBs, and by enhancing the calcium inward current, eliminating the transmural voltage gradient.

4. Device management for electrical storm or incessant ventricular tachycardia

Since most patients with VT or ES already have an ICD implanted due to their underlying heart disease, the following aspects of device management must be considered:

4.1. Reduction of shock delivery

Appropriate ICD shocks are known to be associated with an increased mortality[37,38]. Therefore, the primary aim in ES is to minimize the number of delivered shocks by maximal prolongation of detection time (or detection counter) and maximal increase of heart rate threshold in the VF zone of the ICD[4].

4.2. Preference of ATP for incessant VT

In the case of monomorphic incessant VT, painless therapy with anti-tachycardia pacing (ATP) is preferable[39]. Less aggressive ATP properties ("burst" preferred over "ramp"; coupling intervals ≥ 80%) should be programmed, in order to minimize the risk of VT accelerations, since the latter are mainly terminated by shocks[40]. However, VT accelerations are a potential reason for increased mortality observed in MADIT-RIT after more frequent ATP delivery[38]. On the other hand, no increased mortality was found in patients with only appropriate ATP and no ICD shock delivery[41].

4.3. High rate pacing

High rate pacing is an efficient alternative in some patient groups to reduce VT triggers. Normally heart rates of 90 to 110 beats per minute are considered. If possible (in devices with an atrial lead), atrial overdrive pacing should be preferred. This programming should be sought for special triggers for recurrent VA, such as bradycardia (Brugada Syndrome, LQTS 3) or ventricular premature beats (LQTS, Short-coupled Torsades-de-pointes)[4].

4.4. Forced ATP delivery

If (especially slow) incessant VT cannot be terminated by "automatic" ATP, "manual" or "forced" ATP delivery is one option for acute treatment. In this case, more "aggressive" ATP coupling intervals (e.g. ≤ 75% of VT cycle length) can be applied.

4.5. Switching off anti-tachycardia therapy as last option

Repetitive ICD shocks may lead to an increased catecholaminergic tone[90]. This can in turn increase the risk for recurrent VT, thereby provoking even more ICD shocks and perpetuate ES in a vicious circle. If serial ICD shocks cannot be averted despite reprogramming of detection time and VF-heart rate threshold, the last possible consequence is to switch off the anti-tachycardia therapy. This must be done under continuous deep sedation and intravenous antiarrhythmic medication and hemodynamic support (e.g. in the form of extracorporeal membrane oxygenation (ECMO)) to maintain circulation despite arrhythmia. ICD therapy can be switched off temporarily by using a magnet or permanently by re-programming the device.

5. Ablation therapy and bailout strategies

Monomorphic ventricular tachycardias (VT) are frequently present in the majority of patients with structural heart disease presenting with an electrical storm (ES). Ablation of all clinical and inducible VTs is crucial in the treatment of these patients targeting the abnormal substrate as the basis for the reentrant mechanism (substrate modification).

The recently published VANISH trial (Ventricular Tachycardia Ablation versus Escalated Antiarrhythmic Drug Therapy in Ischemic Heart Disease)[10] showed that VT ablation was superior to the escalation of drug therapy in patients with ischemic cardiomyopathy and an ICD, who had recurrent ventricular tachycardia despite antiarrhythmic drug therapy. There was a significantly lower rate of the composite primary outcome of death, VT storm, or appropriate ICD shocks among patients undergoing catheter ablation than among those receiving an escalation in antiarrhythmic drug therapy (initiation of amiodarone in patients on any other antiarrhythmic drug previously, increase of amiodarone to 300 mg in patients on lower dose, or addition of mexiletine in patients

already on amiodarone 300 mg daily. Therefore, we conclude that VT ablation is an important alternative to antiarrhythmic drug therapy escalation, and, because it may improve outcome, should be favored in selected individuals, especially in patients experiencing VT while on amiodarone. However, due to small patient numbers, the upper confidence interval was 0.98, which suggests that the benefit of ablation might be small. Moreover, the study showed no statistically significant improvement in survival, although it was not powered for this endpoint.

A recent *meta*-analysis of a total of 471 patients reported the efficacy of interventional therapy in ES (including catheter ablation, surgical ablation and transcoronary alcohol ablation)[81]. In this particular study transcoronary alcohol ablation, and surgical ablation have high acute success rates, with a low rate of recurrent storms. The clinical VT was successfully ablated in 92%, and all inducible ventricular arrhythmias were eliminated in 72%. A failed procedure occurred in only 9%, the complication rate was as low as 2% with periprocedural mortality of less than 1%. Over a mean follow-up of 15 months, 94% of patients remained free of ES and 72% free of any VT recurrence. Total mortality was 17%, the majority of patients died due to progression of heart failure (62%).

Another clinical trial investigated the effect of VT ablation of ES in ischemic (ICM) vs non-ischemic cardiomyopathy (NICM)[46]. Catheter ablation of ES was similarly effective in patients with NICM compared with patients with ICM. The acute success rate for VT suppression was 73% and at 60-month-follow-up, 93% of cases remained free of ES. Persistent inducibility of any VT at the end of the procedure was the only independent predictor of VT recurrence. Similar results have been found in another multicenter trial[11].

Catheter ablation is a life-saving procedure in patients with ES related to idiopathic ventricular fibrillation (VF) and/or polymorphic VT. In a small series of 29 patients with ICM and remote myocardial infarction (>6 months ago), ventricular fibrillation (VF) was triggered by monomorphic PVCs originating from the scar border zone with preceding Purkinje-like potentials. Targeting these PVCs prevented recurrence of VF storm in 8/29 patients [12]. In the absence of PVCs, both substrate mapping and catheter ablation appeared to be equally effective.

An interesting approach to VT ablation has recently emerged in patients with Brugada syndrome: although not a structural heart disease, a pathological substrate (in the form of slow conduction, fragmented electrograms and low voltage) can be seen in the area of the right ventricular outflow tract (RVOT), which seems primarily affected by the ion channel disorder. It has been shown that this substrate can be effectively treated through epicardial ablation, thereby preventing arrhythmia recurrence.[49]

PVCs with short coupling intervals inducing polymorphic VTs and VF can also be mapped and ablated. Several locations have been described so far: right and left ventricular Purkinje system, right ventricular moderator band, RVOT and left ventricular outflow tract (LVOT), tricuspid annulus, and left and right ventricular papillary muscles. The response to VT ablation in these cases is very high, however, new triggers were described in the follow-up in around 30% of patients, so that the majority of these patients are treated with an ICD in the long term[50].

In recent years, alternative access routes and new technologies for intervention have been developed in addition to the standard technique of transvenous endocardial catheter-supported ablation. On the one hand, in addition to the clinical target VT, the substrate (scar zone) is now investigated with the aid of pre-interventional imaging (CT and MR) and further characterized with a 3-D mapping system and treated over a wide area (substrate ablation). For most VTs in NICMP, a combined endo / epicardial access is used, for which a dry pericardial puncture via a subxiphoid access is necessary. In addition, bipolar ablations (simultaneous current

delivery via 2 opposing ablation catheters) or current delivery using half-normal saline solutions are applied to cool the electrodes in order to achieve a greater lesion depth with specific substrates (intramyocardial, septal). Finally, a few highly specialized centers use needle ablation, through which a small electrode up to 1 cm is inserted into the myocardium for intramural therapy. Transcoronary ethanol ablation may also be utilized for deeper substrates, and cryoablation has been used surgically for a long time in treatment-resistant cases with NICM[51].

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a genetic heart disease that can often lead to ventricular arrhythmias and sudden cardiac death. A fibrous-fatty remodeling is found in autopsy studies, primarily infiltrating the right ventricular, and occasionally also the left ventricular myocardium, leading to reentry mechanisms. Since the majority of the arrhythmogenic substrate is located on the epicardium, a combined endo- and epicardial ablation procedure is recommended for this form of cardiomyopathy, as this approach can achieve significantly improved freedom from recurrences[52–54].

Increased sympathetic tone is a key factor in the initiation and maintenance of an ES. Therefore, it seems only logical to modulate elements of the cardiac neural axis in order to influence the tendency to develop incessant VT. Sufficient evidence has already been gathered to prove the positive impact of this therapy in patients with long QT syndrome (LQT) or catecholaminergic polymorphic ventricular tachycardia (CPVT). In recent studies, the benefit of sedation has now also been shown in the case of structural heart disease accompanied by an ES[65]. Several procedures have been described so far: thoracic epidural anesthesia (TEA), stellate ganglion (SG) blockage, surgical cardiac sympathetic denervation (CSD) and renal sympathetic denervation (RSD). While the TEA and the SG blockade are temporary measures using a local anesthetic in the epidural space or in the area of the SG, the CSD represents a long-term therapy in which both the afferent and the efferent innervation of the heart are blocked. The lower half of the SG and the thoracic ganglia Th2-Th4 are divided (sympathicotomy) or removed (sympathectomy)[66]. It has been shown that the shock rate in the context of an ES went from 19.6 ± 19 to 2.3 ± 2.9 by CSD and moreover, 90% of the patients showed a reduction in ICD shocks. The bilateral CSD approach has proven to be more effective than the unilateral CSD[67]. This therapy is already offered at several centers in Austria[68]. In RSD, the neural plexus in and around the adventitia of the renal artery is ablated in order to decrease the central sympathetic efflux to the heart by reducing the systemic norepinephrine overflow (spillover) and to inhibit the renin-aldosterone system. Smaller studies have already shown a clinical benefit[82].

Compared to pharmacological therapy, VT ablation in ES reduces the number of recurrences, especially when the procedure is performed early, i.e. within one month[58]. In addition, the benefit is greater when the left ventricular function (LVEF) is still above 25%. Freedom from recurrence after a VT ablation in the ES was associated with an improved survival rate[57]. However, these results could not be reproduced consistently, especially since there is also a tendency towards an increased non-cardiac mortality in ES. In summary patient selection for VT ablation plays an important additional role.

The timing of ablation, the applied strategy and the need for hemodynamic support should be tailored to the specific patient. Patients with advanced heart failure are at high risk of hemodynamic collapse and can benefit from mechanical support during the procedure (LVAD, ECMO). VT ablations with ECMO support have already been carried out successfully in our country in close collaboration with the Austrian VT network[69]. In individual patients, VT ablation should be limited to the critical isthmus of the target tachycardia in order not to overstrain the cardiac reserve[70].

6. VT network

In this consensus paper, the organizational aspects of an acute VT network in Austria are described and should subsequently be implemented[80]. All electrophysiologic centers in Austria that deal with VT ablation are to be integrated into the network in the medium-term. Centers that co-operate in the network are divided into primary and secondary VT centers Fig. 3.

Primary VT centers should usually be the first ones to be contacted from peripheral hospitals for the management of patients with sustained VTs or after ICD shock. They have expertise in treating patients with VT and have the possibility of elective VT ablations. The electrophysiologist of the primary center will accept the patient for further treatment, either in the outpatient clinic or as an inter-hospital transfer. If deemed necessary, the patient will be directed to a secondary VT center. The purpose of primary VT centers is to complement secondary VT centers, extend the net-

Fig. 3. *VT Network Austria.* Collaborating VT centers as of October 2020. Blue arrows: Primary (elective) VT centers. Red arrows: Secondary (acute) VT centers. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 2

Antiarrhythmic drugs. VT = ventricular tachycardia, VPB = ventricular premature beat, LQTS = long QT syndrome, CPVT = catecholaminergic ventricular tachycardia, BB = Betablocker, AVN = atrio-ventricular node, VF = ventricular fibrillation, TdP = Torsade de pointes tachycardia, LVEF = left ventricular ejection fraction, HFrEF = heart failure with reduced ejection fraction.

Antiarrhythmic drug (Vaughan Williams Class) Dose	Special feature	Indication	Important side effects or contraindications
Ajmaline (IA) i.v. single dose 1 mg/kg till max. 100 mg Infusion: speed max. 10 mg/min; continuous infusion: 10–50 mg/h	short half-life, easy titration	VT, VF, malignant arrhythmias triggered by short coupled VPBs, (Ajmaline-test to unmask Brugada-syndrome)	QRS widening, PQ-prolongation, QT-prolongation, pro-arrhythmic effect (stop therapy in case of QRS widening > 25%, PQ-prolongation > 50%, QTc prolongation > 500 ms)
Quinidine (IA) p.o. sulfate 200–600 mg every 6-12hrs gluconate 324–648 mg every 8-12hrs i.v. total 800 mg, rate 50 mg/min	strong blocker of transient K-efflux	VF, Brugada syndrome, SQTS, VF in acute MI, malignant arrhythmias triggered by short coupled VPBs	syncope, TdP, AV-block, nausea, vomiting, QRS widening, QTc prolongation
Lidocaine (IB) i.v. 1–1,5mg/kg bolus, then 0,5–0,75 mg/kg bolus every 5–10 min, max. dose 3 mg/kg Infusion: 1–4 mg/min	advantage of stronger binding and effect in low pH and membrane potential in case of ischemia triggered VTs	VTs caused by acute myocardial ischemia	proarrhythmic, bradycardia, delirium, psychosis
Mexiletine (IB) p.o. 150–300 mg every 8-12hrs	especially as add-on in inefficiency of amiodarone	VT, LQTS3	heart failure, sinus node dysfunction, AV-block
Flecainide (IC) p.o. 50–200 mg every 12hrs **Propafenone (IC)** p.o. 150–300 mg every 8hrs	PQ-prolongation, QRS-prolongation	VPBs, VT, flecainide also for CPVT	bradycardia, med. induced Brugada syndrome, monomorphic VTs due to myocardial scar, eventually acute reduction of LVEF in HFrEF
Beta-Blocker (II) **Propranolol:** i.v. 1–3 mg every 5 min max. 5 mg if necessary repeated after 4hrs **Esmolol:** i.v. 0,5mg/kg bolus, then 0,05 mg/kg/min **Landiolol:** i.v. 0,1–0,3mg/kg bolus, then 0,01–0,04 mg/kg/min	cornerstone of VT therapy advantage of non-selective BB is suppression of adrenergic tonus (e.g. **propranolol**) in case of severely reduced LVEF BB with short half-life (e. g. **esmolol, landiolol**). reduction in sinus rate, increase of AVN refractoriness ultra-short half-life (3–5 min), advantage of less pronounced neg. inotrope effect and reduced risk of hypotension [9,36].	VPB, VT, LQTS, CPVT	hypotension, bradycardia, AV-block, bronchospasm
Sotalol (III) p.o. 160 – 320 mg/day	betablocker and class III antiarrhythmic drug	VT, 2nd line drug in ARVC	QT-prolongation, TdP, bradycardia, AV-block, depression
Amiodarone (III) i.v. 150–300 mg bolus; 1 mg/min for 6hrs, then 0,5mg/min for 18 h p.o. 3x200-400 mg/day, if 10 g total dose 200 mg/day	most important emergency antiarrhythmic drug, even more effective, if combined with BB reduction of sinus rate, QRS prolongation, QTc prolongation, increase of AVN-refractoriness	VT, VF	bradycardia, AV-block, QT-prolongation (proarrhythmic effect, if QTc > 500 ms TdP) contraindication: LQTS, TdP, bradycardia induced VTs
Verapamil (IV) i.v. 2,5–5 mg every 15–30 min p.o.: 240–480 mg/day	reduction of sinus rate and AV-conduction, PQ-prolongation	VT, VPBs, fascicular VT	hypotension, edema, aggravation of HFrEF, AV-block, bradycardia
Isuprenaline (other) i.v. 0.5–20 µg/min	cardiac acceleration to suppress ectopic VPBs, enhancement of the inward calcium current to eliminate the transmural voltage gradient	Electrical Storm in Brugada syndrome, early repolarization syndrome and short QT syndrome	hypotension, tachycardia, hypokalemia

Table 3

Device management. VT = ventricular tachycardia; VF = ventricular fibrillation; ATP = anti-tachycardia pacing; CL = cycle length.

Recommendations – Device Management	Class	Level	Ref.
Detection time should be programmed to the longest possible value and detection cycle length programmed to the shortest possible value, with the aim to reduce unnecessary shocks.	I	A	[38,39,42]
In sustained monomorphic VTs, primary ATP delivery (preferentially burst pacing at ≥ 80% CL) should be considered and the device reprogrammed accordingly.	IIa	A	[38,39,42–45]
To minimize the occurrence of VT / VF triggers (e.g. bradycardia, long QT, short coupled VPBs), pacing at an increased lower rate may be considered.	IIb	C	[4]

Table 4

Catheter ablation therapy. VT = ventricular tachycardia; VF = ventricular fibrillation; RVOT = right ventricular outflow tract; ARVC = arrhythmogenic right ventricular tachycardia.

Recommendations – Ablation Therapy	Class	Level	Ref.
VT ablation is recommended to be performed as soon as possible for monomorphic VT in ischemic cardiomyopathy, refractory to adequate medical treatment.	I	A	[25,46,55–58]
VT ablation should be considered for monomorphic VT in non-ischemic cardiomyopathy, refractory to adequate medical treatment. For the primary procedure, an endocardial or a combined endo-/epicardial approach may be chosen.	IIa	B	[46,47,57,63]
For the treatment of drug refractory VT / VF in ARVC, a combined endo-/epicardial or a primary epicardial ablation should be considered.	IIa	B	[52–54]
For the treatment of drug refractory VT / VF in Brugada syndrome, an epicardial ablation in the area of the RVOT may be considered.	IIb	B	[49,64]
VT ablation may be considered for polymorphic VT / VF, which is idiopathic or occurring after myocardial infarction, and refractory to adequate medical treatment.	IIb	C	[48,50,59–62]

Table 5

Bail-out Strategies. VT = ventricular tachycardia; VF = ventricular fibrillation; ECMO = extracorporeal membrane oxygenation; LVAD = left ventricular assist device.

Recommendations – Bail-out Strategies	Class	Level	Ref.
It is recommended that emergency cardiac surgery is available within a delay of 60 min from all secondary VT ablation centers, for the management of potential complications, and for the possibility of ECMO-implantation.	I	C	this panel of experts
In patients with electrical storm, mechanical circulatory support (e.g. ECMO, LVAD, etc.) should be considered to stabilize the patient before or during an ablation procedure, in particular in patients with a high risk score (e.g. PAINESD, I-VT).	IIb	B	[75–79]
Stellate ganglion blockade may be considered in the treatment of electrical storm, to reduce sympathetic activity.	IIb	C	[65,71,72]
Surgical sympathetic denervation, to reduce permanently sympathetic activity, may be considered in the treatment of refractory electrical storm or in frequent VT recurrence despite medical therapy.	IIb	C	[67,73,74]
High urgent cardiac transplantation may be considered in patients with VT / VF, refractory to all employed therapies, depending on the patient's condition before the event, age and comorbidities.	IIb	C	this panel of experts

work, increase accessibility to patients from peripheral hospitals and diminish waiting periods. They guarantee specialized treatment approaches and help to keep the secondary centers from being overwhelmed with patients manageable in the primary centers.

Secondary VT centers are the second line in treating VT patients. They have at least two electrophysiologists to guarantee specialized care 365 days of the year. These electrophysiologists are experienced in structural and idiopathic VT ablation, including epicardial approaches and VT ablation in unstable patients and under hemodynamic support. Acute coronary diagnostics and intervention must be available on-site. Options for "bail-out" strategies (including ECMO support, acute LVAD implantation, urgent heart transplant listing and cardiac surgery) must either be available on-site or reachable in less than 60 min transfer time. Moreover, close co-operation with a corresponding intensive care unit for transferred patients and the primary cardiological focus of the department needs to be emphasized. It is the purpose of these centers to provide care for all patients which are in VT storm

unresponsive to medical therapy, or which cannot be sustainably stabilized in their primary hospital, or which cannot be managed sufficiently by a nearby primary VT center (need for specialized access routes or equipment for further management, as mentioned above). As they are responsible for often hemodynamically unstable patients, they must have the ability to accept patients within 24 h and perform acute VT ablation procedures, if necessary. Therefore, an electrophysiologist must be available for consultation via the VT hotline any time. (Tables 2–5).

In addition to these requirements, each center is responsible for local concepts for acute cases regarding availability of nursing staff, radiological / technical staff, EP technicians (navigation system) and doctors.

It has to be noted that the Austrian VT network is still expanding to develop more VT centers (especially primary VT centers) to increase accessibility throughout the country and to provide rapid and nearby care to more rural and remote areas. As of today, we have more secondary centers in the network as all Austrian high-volume centers and university clinics are taking part. The goal is to establish at one primary and one secondary center in each part of Austria.

Overview of the criteria for primary and secondary VT ablation centers:

Primary VT Centers (Centers for Elective VT Ablation):

- Expertise in treating VT patients
- Possibility of elective VT ablation
- Acute responsiveness of the respective electrophysiologist via defined paths ("VT Hotline") on most days of the week
- Primary contact persons in the treatment of VT and ES
- Possibility to accept patients for further treatment

Secondary VT Centers (Centers for Acute VT Ablation):

- All criteria of primary VT centers
- 24 h / 7 days availability of an electrophysiologist for consultation
- Acceptance of patients with ES within 24 h
- Experience in epicardial ablation

- Experience in ablation under hemodynamic support (e.g. ECMO)
- At least 2 electrophysiologists experienced in complex VT ablation
- Cardiac surgery on-site or reachable within 60 min
- Close co-operation with an intensive care unit specialized in cardiac care
- Acute coronary diagnostics and intervention must be available on-site.

Declaration of Competing Interest

The authors declared that there is no conflict of interest.

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