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Thomas Deneke · Karin Nentwich · Elena Ene · Artur Berkovitz · Kai Sonne · Philipp Halbfaß

Klinik für Interventionelle Elektrophysiologie, Rhön-Klinikum Campus Bad Neustadt, Bad Neustadt a. d. Saale, Germany

Acute management of ventricular tachycardia

Introduction

The acute management of patients with sustained ventricular tachycardia (VT) often involves the whole armamentarium of evaluation and supporting hemodynamics, diagnosis of underlying cardiac pathology and treatment using electrical, medical or/and interventional termination of VT.

The basic concept of acute management of any non-tolerated (hemodynamically compromising) tachycardia is immediate termination using defibrillation or r-wave-synchronous cardioversion. Beyond this concept, treatment of VT patients may become comparatively complex and depends upon electrocardiogram (ECG) morphology of ventricular arrhythmia (VA) (polymorphic versus monomorphic VT), including timing of episodes and underlying cardiac pathology and history (idiopathic versus cardiomyopathy/structural heart disease versus channelopathy). Patients presenting with recurrent ongoing VA episodes ("shock-refractory VA") should be differentiated from patients with a single ongoing VT event, since the former patients require effective rhythm stabilization therapy versus acute termination of VT only. In some cases, VA can be mediated by ischemia or left ventricular (LV) stretch (e.g. as in acute cardiac decompensation), but in most cases VT is due to an underlying cardiac substrate including electrically inert scar areas and slowly conducting VT channels (mostly a reentrant mechanism).

This article focuses on the most commonly seen VT patients in clinical routine and discusses potential diagnostics and treatment options for rhythm stabilization based on current published data and guidelines/recommendations [1–7] as well as personal experience. Acute in-hospital management of VT patients may be needed in any emergency department, intensive care units or regular low-care wards and their specificities undermine the clinical importance of arrhythmia treatment expertise (experienced electrophysiologist [EP]) in emergency decision-making. Identifying patients at risk of future arrhythmic events is important for acute management as well as for long-term implantable defibrillator therapy [1, 3, 4].

Acute treatment of sustained ventricular arrhythmia

Immediate termination of non-tolerated VA is crucial and early cardioversion/defibrillation is recommended in all currently available guidelines. In patients with tolerated ongoing VA (monomorphic VT) 12-lead ECG documentation of QRS morphology during VT is essential for planning future treatment, especially for VT ablation. ECG documentation of wide complex QRS tachycardia may help to differentiate between VT versus supraventricular tachycardia (SVT) with functional or preexisting QRS widening (**Fig. 1 and 2**). In total, 85% of regular wide-complex tachycardia are VT, especially if there is a history of underlying structural heart disease. VA-ECG may help to differentiate the mechanism and uncover underlying cardiac abnormalities, identify potential treatment options and establish a differential diagnosis if the mechanism is unclear [1, 3–6, 8].

As VA may acutely impair LV function and lead to hemodynamic compromise beyond termination of the arrhythmia appropriate support using vasopressors, percutaneous or surgical hemodynamic support and mechanical ventilation may be required in some cases [8–13].

There is a clear difference in management between monomorphic VT and polymorphic VT/VF. Whereas monomorphic VT is usually substrate-based reentry or idiopathic, polymorphic VT/VF may be drug-induced and should usually warrant acute coronary evaluation and further diagnostics to identify initiating triggers. Indications for ischemia-induced VA events can be seen on ECG during phases between VA episodes and ST-segment elevation, or depression/T-wave negativity may direct attention towards acute coronary syndromes and sufficient treatment of ischemia that may suppress VA [1–4].

In general, the management of VA patients should be directed toward four important aspects in a stage-like approach:

1. Acute termination of ventricular arrhythmia
2. Evaluation of arrhythmia mechanism—diagnostics
3. Prevention of recurrent VA episodes (acute and chronic)
4. Treatment of underlying heart disease, heart failure management if needed

Acute termination of ventricular arrhythmia

Acute termination of VA should be considered immediately if the arrhythmia causes hemodynamic compromise. This

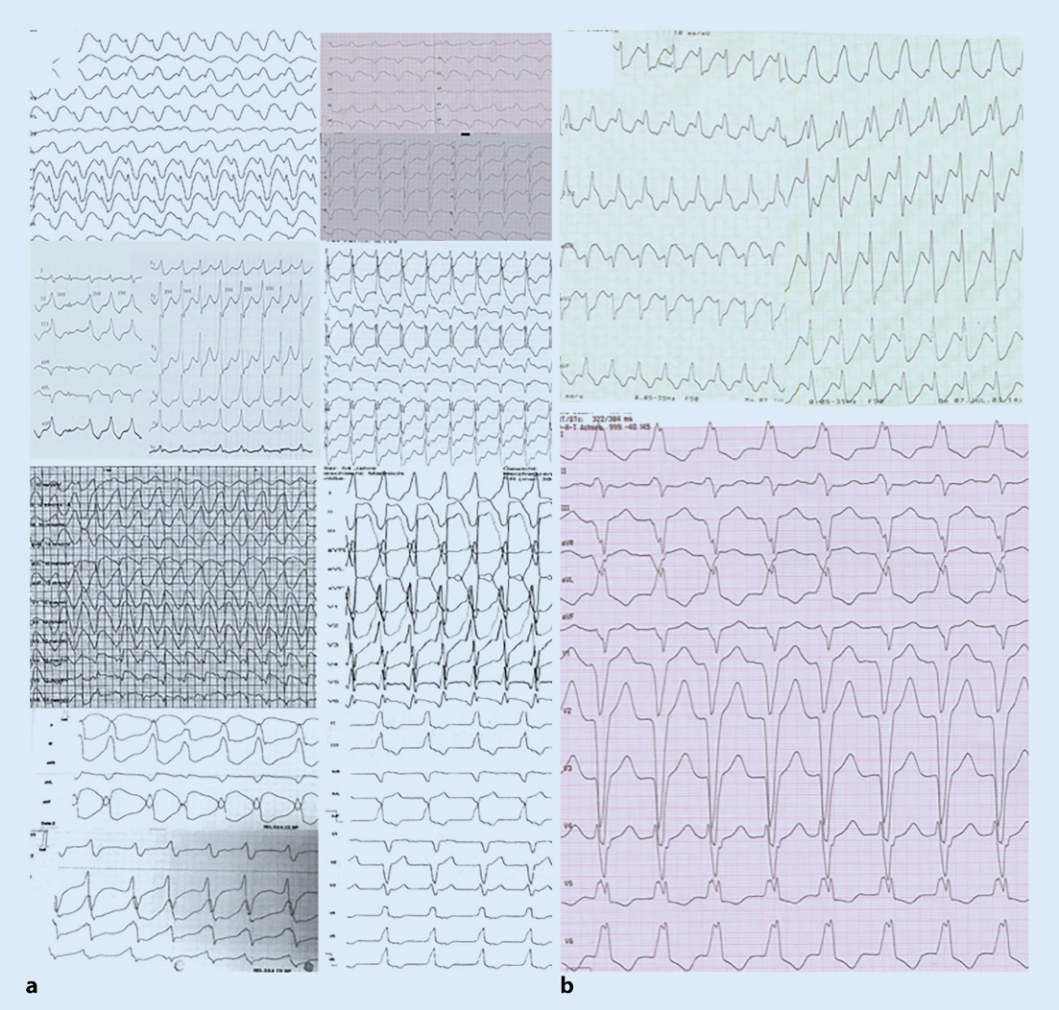

Fig. 1 ◀ Examples of wide-complex tachycardias: **a** ventricular tachycardia and **b** supraventricular tachycardia with functional right-bundle-branch block and pre-existing left-bundle-branch block

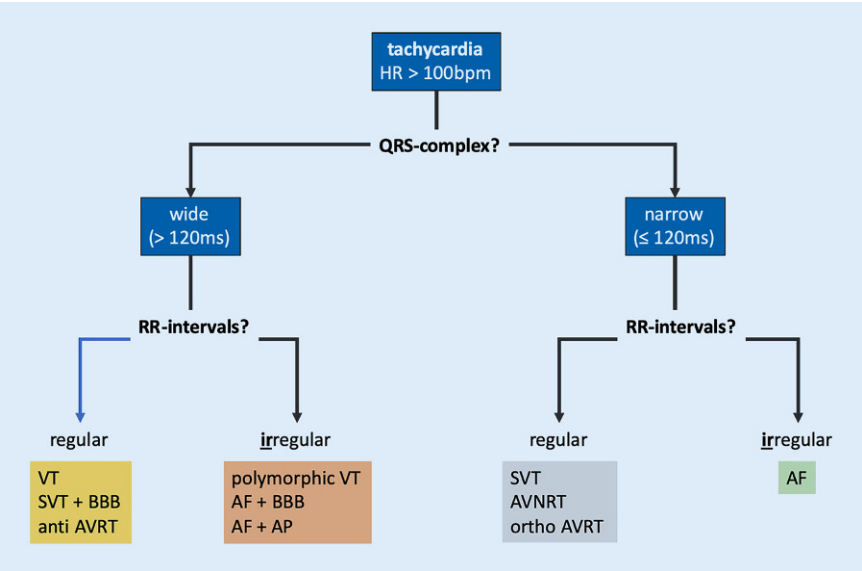

Fig. 2 ▲ Differential diagnosis of tachycardia mechanisms based on electrocardiographic criteria. *bpm* Beats-per-minute, *SVT* supraventricular tachycardia, *AVRT* atrioventricular reentry tachycardia, *AVNRT* av-nodal reentrant tachycardia, *AF* atrial fibrillation, *BBB* function or preexisting bundle-branch-block-like QRS morphology, *VT* ventricular tachycardia

is usually measured by blood-pressure measurement and systolic blood pressure <70 mm Hg is an indicator for compromise. Although tachycardia rate and origin (supraventricular versus ventricular) may play a role, hemodynamic compromise is usually seen only with heart rates >150 bpm—depending on LV function. In cases with tachycardia leading to cardiogenic shock immediate conversion of VA is indicated using either defibrillation or R-wave synchronous cardioversion. In general, polymorphic VT or VF leads to hemodynamic collapse and immediate termination is needed, whereas monomorphic VTs may be well tolerated depending on arrhythmia heart rate, duration of arrhythmia and LV function/cardiac underlying abnormalities. Whatever the case may be, if possible, a 12-lead ECG of the arrhythmia should be performed to scrutinize for arrhythmia ori-

Abstract · Zusammenfassung

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Abstract

Acute management of patients with ventricular arrhythmia (VA) is aimed at immediate VA termination if the patient is hemodynamically instable and early termination after initial diagnostic work-up if tolerated. Prolonged episodes of VA may lead to hemodynamic and metabolic decompensation and early resumption of normal ventricular activation is warranted. Termination is best performed by electrical cardioversion, anti-tachycardia pacing (if available, in cases with an implanted defibrillator [ICD]) or defibrillation. Antiarrhythmic drug treatment may lead to rhythm stabilization in cases of VA recurrence. Scrutinizing the electrocardiogram (ECG) of VA is extremely helpful to differentiate potential mechanisms, underlying cardiac pathologies and identify treatment options, as well as a differential diagnosis if a ventricular origin is unclear. In general, structural VA should be differentiated from idiopathic and non-structural (idiopathic) VA. On the other hand, based on ECG morphology VA should be classified into monomorphic versus polymorphic ventricular tacyhcardia (VT)/ventricular fibrillation (VF). Polymorphic VT/VF may be related to reversible causes as well as genetically determined arrhythmia syndromes and a specialized treatment pathway may be chosen: (1) VA termination, (2) evaluation and treatment of potential VA causes, (3) acute (medical treatment) and chronic (interventional treatment using catheter ablation) prevention of recurrence and (4) treatment of underlying heart disease, if identified, are crucial pillars of VA management. These patients can be managed in dedicated VT units and by multispecialty teams integrating all potential aspects of rhythm stabilization and treating underlying cardiac abnormalities. Heart failure management in patients with reduced left ventricular function may be crucial for the long-term prognosis.

Keywords

Arrhythmias, cardiac · Catheter ablation · Defibrillator · Structural ventricular tachycardia · Heart failure

Akutversorgung bei ventrikulärer Tachykardie

Zusammenfassung

Die Akutversorgung bei ventrikulären Arrhythmien (VA) richtet sich nach der hämodynamischen Toleranz. Bei hämodynamischer Instabilität ist eine sofortige Terminierung indiziert, ansonsten eine frühzeitige Terminierung nach initialer diagnostischer Aufarbeitung inklusive 12-Kanal-Elektrokardiogramm (EKG). Längere Episoden von VA können zur hämodynamischen und metabolischen Entgleisung bis zum kardiogenen Schock führen; eine schnelle Rückkehr zur normalen ventrikulären Stimulation wird angestrebt. Die Terminierung von VA erfolgt am besten mit einer elektrischen Kardioversion, Überstimulation (soweit verfügbar, in Fällen mit implantiertem Defibrillator) oder Defibrillation. Eine antiarrhythmische medikamentöse Therapie kann zur Rhythmusstabilisierung bei rezidivierenden VA-Episoden führen. Die genaue Analyse des EKGs der VA ist bei der Differenzierung des Mechanismus von großem Nutzen. Gleiches gilt in Bezug auf die Detektion einer möglichen zugrunde liegenden kardialen Erkrankung, die Abwägung möglicher Therapieoptionen und die Differenzialdiagnostik, wenn ein ventrikulärer Ursprung unsicher ist. Es sollten strukturelle von idiopathischen oder nichtstrukturellen VA differenziert werden. Auf Grundlage der EKG-Morphologie können monomorphe von polymorphen ventrikulären Tachykardien (VT) oder Kammerflimmern unterschieden werden. Generell können polymorphe VT oder Kammerflimmern potenziell reversible Ursachen haben oder mit genetisch determinierten Arrhythmiesyndromen zusammenhängen, die durch spezielle Interventionen therapierbar sind. 1. VA-Terminierung, 2. Evaluation und Behandlung potenzieller Ursachen, 3. akute (medikamentös) und langfristige (interventionell, z. B. mittels Katheterablation) Prävention von VA-Rezidiven und 4. Behandlung der zugrunde liegenden Herzerkrankung (falls vorhanden) sind kritische Bestandteile des VA-Managements. Die Patienten können in ausgewiesenen VT-Unit und von multidisziplinären Teams behandelt werden, die sämtliche potenziellen Aspekte der Rhythmusstabilisierung sowie der Behandlung zugrunde liegender Herzerkrankungen einbeziehen. Für die Langzeitprognose von VA-Patienten mit einer eingeschränkten linksventrikulären Funktion kann eine optimierte Herzinsuffizienzbehandlung essenziell sein.

Schlüsselwörter

Herzrythmusstörung · Katheterablation · Defibrillator · Strukturelle ventrikuläre Tachykardie · Herzinsuffizienz

gin, mechanism (including supraventricular versus ventricular) and as a potential target arrhythmia for future cardiac interventions (like catheter ablation).

In cases of well-tolerated VA termination may be postponed in order to record an ECG and perform initial basic diagnostics/history. Termination of VA should be performed early, since prolonged episodes of tolerated VT may cause hemodynamic compromise and heart failure. In patients with implanted defibrillators (ICD) anti-tachycardia pacing (ATP) as well as diagnostic differential pacing may be feasible to rule out SVT versus VT and also has the potential to terminate VA. The use of cardioversion over antiarrhythmic drug (AAD) administration is recommended to terminate any VA. Routine administration of AADs to prevent recurrences of episodic VA is not recommended. Amiodarone or lidocaine have been recommended in current guidelines as the only effective AAD for VF/polymorphic VT unresponsive to defibrillation (class of recommendation IIb) and there is insufficient data to support routine use of ß-blocker administration or adminis-

Table 1 Examples of antiarrhythmic drugs that may be used in specific indications to medically treat ventricular arrhythmia (VA) or prevent VA recurrences

Antiarrhythmic drug	Potential indication
Amiodarone	Structural VT, VF
Ajmaline	Structural VT, VF
ß-blocker	Structural VT, idiopathic VT, long QT syndrome, catecholaminergic polymorphic VT
Mexiletine	Long QT syndrome 3, structural VT
Chinidine	Short QT syndrome, Brugada syndrome
Verapamil	Fascicular tachycadia, idiopathic VT
Isoprenaline	Short-couples PVC-induced VF, Brugada syndrome

VT ventricular tachycardia, *VF* ventricular fibrillation, *PVC* premature ventricular contraction

Table 2 Stepwise approach to rhythm stabilization in patients with recurrent structural ventricular arrhythmia

Phase/category	Interventions
Phase/category 1	Mild sedation ß-blockade Initiate optimized heart failure medication/cardiac recompensation/decrease fluid overload
Phase/category 2	Specific antiarrhythmic medication: amiodarone or ajmaline (combination with mexiletine)
Phase/category 3	Deep sedation using propofol General anaestesia
Phase/category 4	Percutaneous hemodynamic support
Phase/category 5	Acute catheter ablation
Phase/category 6	Inteventional ganglionated stallatum blockade

tration of any AAD early after reversion of VA [1–7].

Depending on the exact diagnosis of the underlying heart disease different AADs may be effective for arrhythmia termination or prevention of recurrences ([3, 4, 10, 14]; ◘ **Table 1**).

There is a paucity of data on combining AADs (except for ß-blockage or verapamil), and combining AADs should be restricted to patients in whom a trial of multiple single AADs is not effective. Side effects may be pronounced and unpredictable when combining specific AADs.

Evaluation of arrhythmia mechanism

Diagnosing the arrhythmia mechanism is critical for risk stratification for sudden cardiac death, future treatment options and possible prevention of recurrences of VA. Taking appropriate medical and medication history is essential, since some VAs may be due to medication side effects. In addition, broad complex regular tachycardia is VT in over 95% of cases if structural heart disease is diagnosed. Blood sampling (including electrolytes, cardiac markers including troponin, etc.) may identify potential reversible causes of polymorphic VA. Scrutinizing the ECG during tachycardia (if available) and after termination of VA in intrinsic rhythm is crucial in detecting, for example, myocardial ischemia as the cause of VA and in identifying the origin of the arrhythmia. In this scenario immediate coronary diagnostics and treatment may help to stabilize rhythm. Monomorphic VT is usually not due to myocardial ischemia (but rather underlying substrate formation) and even if coronary stenoses are diagnosed angioplasty or surgical repair will not decrease monomorphic VT recurrences [15–17]. Polymorphic VT or VF is more often related to myocardial ischemia and therefore special attention in this regard is crucial ([15]; see ◘ **Figs. 3 and 4**).

Prevention of recurrent ventricular arrhythmia episodes

Preventing VA recurrences is an important pillar of management of VA patients. Whereas patients with a single episode of VA usually do not require specific treatment, patients with recurrent VA episodes mandate acute rhythm stabilization including treatment of reversible causes (see Sect. *Evaluation of arrhythmia mechanism*), medical and/or interventional electrophysiological treatment. For chronic rhythm stabilization catheter ablation of VT has been proven to be beneficial in idiopathic VTs of right ventricular origin and patients with ischemic cardiomyopathy [1–3]. Specifically, in ischemic scar-related VT, catheter ablation has been documented to be superior to AAD treatment, leading to significantly lower VT recurrences [1–4, 10, 18].

Prevention of VA recurrences depends on the underlying arrhythmia mechanism (see sects. *Acute termination of VA* and *Evaluation of arrhythmia mechanism*) and the hemodynamic situation of the patient. Patients with recurrent VA usually have underlying heart disease and have been evaluated in our center based on standardized workflows and treatment in a specifically dedicated VT unit [8, 9, 11–13].

A standardized workflow with a stepwise approach for patients with recurrent VA episodes has been effective in early rhythm stabilization in the authors' clinical setting. These categories include basic supportive treatments and successive increments in invasiveness and aggressiveness. Basic principles include relieving stress, antiarrhythmic medication, treatment of heart failure, hemodynamic support and potential interventional arrhythmia suppression (◘ **Table 2**).

Phase 1 interventions imply sedation (usually midazolam), ß-blockade (propranolol or metoprolol) and initiation of optimized heart failure medication and cardiac recompensation if needed. Phase 2 includes intensified and specific antiarrhythmic treatment including amiodarone (also in the case of prior oral amiodarone treatment) or ajmaline as an alternative, mexiletine in the

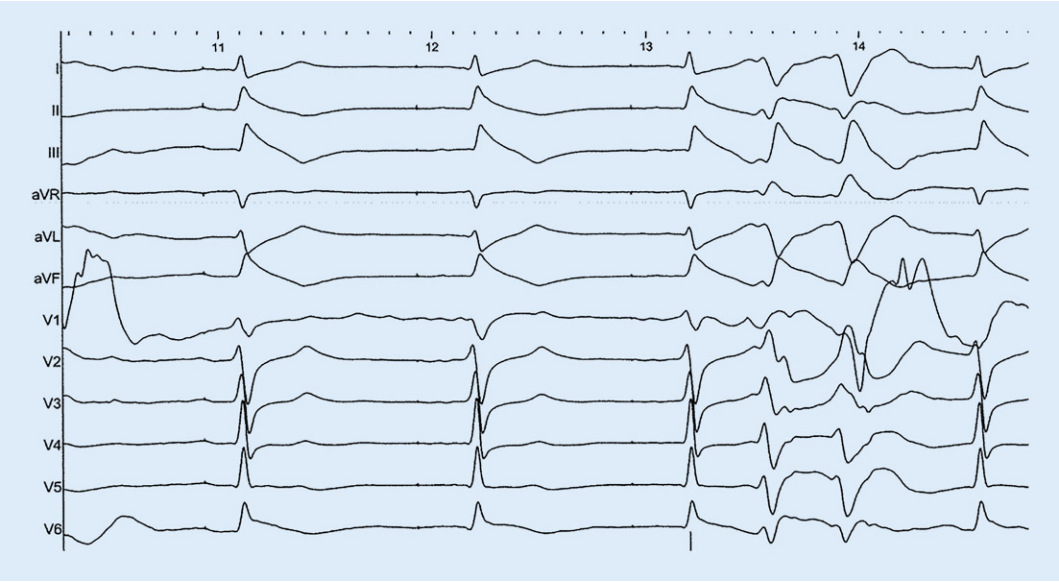

Fig. 3 ◀ Onset of polymorphic ventricular tachycardia in a patient with coronary spasm. See ST-segment elevation in leads II, III and aVF before onset of ventricular arrhythmia

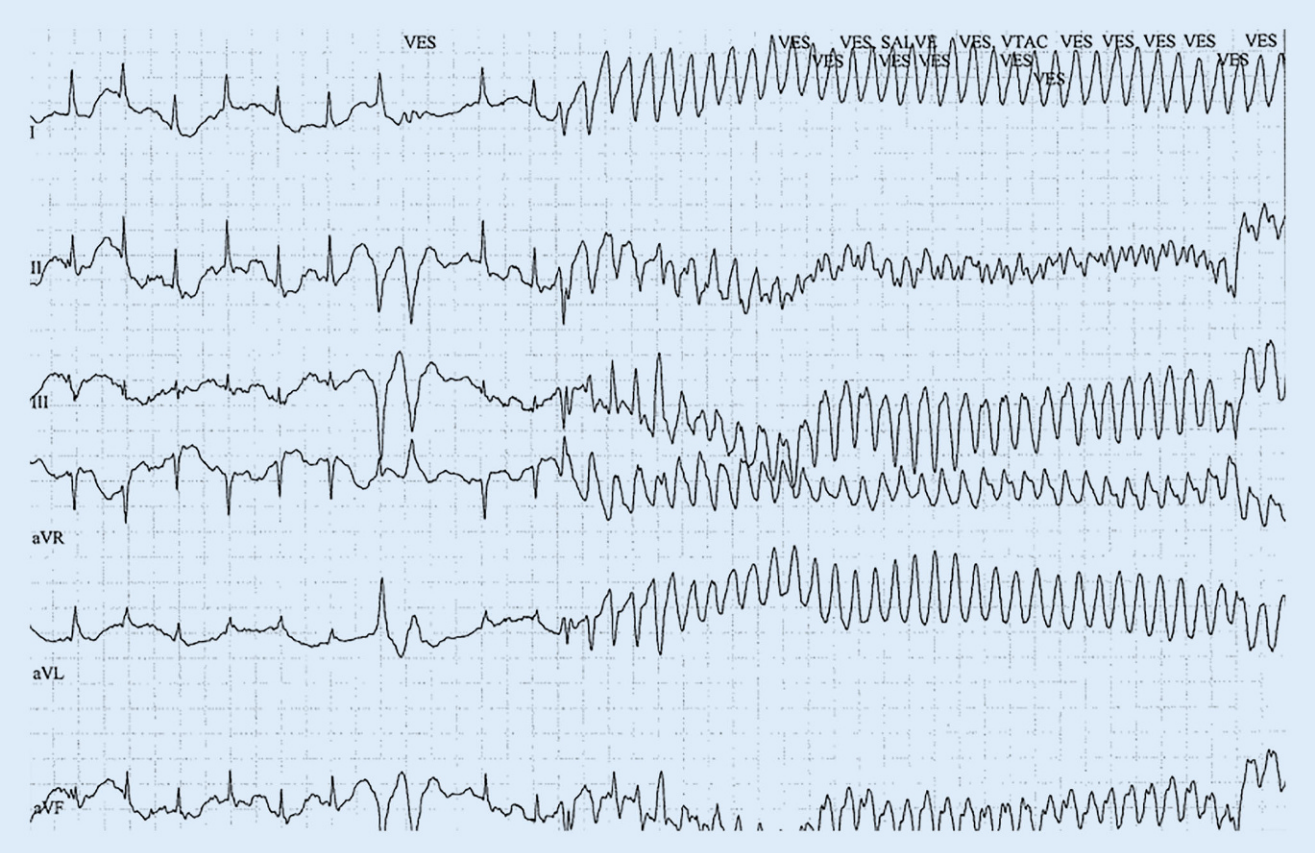

Fig. 4 ▲ Ischemia-induced polymorphic ventricular tachycardia (VT) in a patient with coronary artery disease undergoing physical stress testing. See ST-segment depression in leads II, III and aVF as an indicator for coronary insufficiency and onset of polymorphic VT

case of high-dose oral amiodarone pretreatment. Phase 3 strategies consist of measures for further stress reduction like deep sedation using propofol or other narcotics. The phase 4 intervention involves percutaneous hemodynamic support and phase 5 as a next step includes catheter ablation of either triggers for focally induced VAs or monomorphic VT and is available on a 24-h 7-days/week basis in some dedicated centers. The last resort in phase 6 is interventional ganglionated stellate blockage. In a recent analysis, over 90% of cases were effectively rhythm stabilized using phase 1 and 2 interventions. Phases 4–6 may only be offered in specialized heart centers and networks centered around these have been recommended.

In patients with ICD and recurrent discharges special caution is needed to clarify whether recurrent ICD treatment is appropriate for terminating VA, is ineffective in ongoing VA or may be inappropriate for atrial arrhythmia or ICD malfunction/lead dysfunction. Cardiopulmonary resuscitation and advanced life support in patients with ICDs is performed in the same way as in other patients.

Appropriate programming using long detection intervals and anti-tachycardia pacing is recommended in ICD patients to prevent recurrent unnecessary ICD shocks.

Chronic arrhythmia and heart failure management

Whereas preventing long-term recurrent VA episodes is relevant and most effectively achieved using catheter ablation, no study to date has documented the prognostic benefits of catheter ablation on mortality. On the other hand, optimized heart failure management including chronic optimum medical therapy as well as interventional strategies (e.g. left ventricular assist devices, cardiac resynchronization [CRT] therapy, interventional valve treatment) are an important pillar and may decrease long-term mortality. In an interesting study on patients admitted to intensive care units due to decompensated heart failure or sustained VA, cause of death was comparable and most commonly worsening of heart failure in both groups [19]. Therefore, the management of patients with recurrent VA should integrate multiple specialties of medicine and organization of a "VT team" as a multimodal approach may be helpful. A dedicated VT unit with experienced personnel and multiple specialties including cardiology, electrophysiology, heart failure specialists, imaging, cardiac surgery and intensive care has been shown to be an ideal setting for diagnosing and treating complex VA patients.

In comparative trials on monomorphic ischemic VT patient catheter ablation has been documented to be more effective than current drug regimens in suppressing recurrent VA episodes. No randomized trials on the effect of catheter ablation in non-ischemic cardiac substrates are available and efficacy of catheter ablation is lower in these cardiac pathologies, mostly due to the more complex substrate and complex ablation strategies usually involving endocardial and epicardial targets [20]. Catheter ablation is an important cornerstone of chronic rhythm stabilization. For acute arrhythmia termination acute catheter ablation is only needed in a minority of cases if appropriate and predefined standardized protocols [1–4, 8, 9, 11–13] are used for conventional rhythm stabilization.

Chronic heart failure management is probably the most important pillar of treatment in patients with recurrent structural VA and needs to be acutely initiated, but continuously adopted and optimized [19]. This may imply not only optimal medical therapy (OMT), but also device-based treatment options as part of a heart failure team decision. In contrast, most patients with idiopathic VA or no underlying structural abnormalities (e.g. channelopathies) must be appropriately diagnosed and treated in a specialized electrophysiology unit. Identifying patients that will need management beyond VA termination is part of the initial triage process and potentially transferring these patients for further specialized diagnostics and treatment in a network of cooperating centers may be helpful.

The decision regarding ICD therapy should be made based on current guideline recommendations. Patients without structural heart disease and with no genetically determined arrhythmia syndromes (like Brugada syndrome, LQTS syndrome) are generally at no increased risk for arrhythmic death and effective arrhythmia suppression may effectively alleviate VA and the risk for sudden cardiac death [1, 3, 4].

Practical conclusions

- **The basic concept of acute management of any non-tolerated (hemodynamically compromising) tachycardia is immediate termination using defibrillation or r-wave-synchronous cardioversion.**
- **Diagnosing the arrhythmia mechanism is critical for risk stratification for sudden cardiac death, future treatment options and possible prevention of recurrences of VA.**
- **Preventing VA recurrences is an important pillar of management of VA patients.**
- **Treating underlying structural heart disease and heart failure are also important cornerstones of chronic management of patients with structural VT.**

Corresponding address

Prof. Dr. Thomas Deneke
Klinik für Interventionelle Elektrophysiologie,
Rhön-Klinikum Campus Bad Neustadt
Von-Guttenberg-Str. 11, 97616 Bad Neustadt
a. d. Saale, Germany
thomas.deneke@campus-nes.de

Compliance with ethical guidelines

Conflict of interest. T. Deneke, K. Nentwich, E. Ene, A. Berkovitz, K. Sonne and P. Halbfaß declare that they have no competing interests.

For this article no studies with human participants or animals were performed by any of the authors. All studies performed were in accordance with the ethical standards indicated in each case.

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