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REVIEW

# Management of sustained arrhythmias for patients with cardiogenic shock in intensive cardiac care units

## *Prise en charge des arythmies chez les patients en choc cardiogénique en unité de soins cardiaques intensifs*

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**Summary** Cardiac arrhythmias that occur in patients referred to intensive care units worsen symptoms and outcomes and need urgent correction, especially in patients admitted for refractory heart failure. Electrical storm is a frequent reason for referral to an intensive care unit. Specific, efficient and rapid management of patients presenting with various arrhythmias is therefore mandatory and procedures should be known by any physician involved in an intensive care unit. This article reviews the current knowledge on the management of supraventricular

*Abbreviations:* AF, atrial fibrillation; AVNRT, atrioventricular node reentrant tachycardia; AVRT, atrioventricular reciprocating tachycardia; $CHA_2DS_2$-VASc, Cardiac failure, Hypertension, Age $\geq$ 75 [Doubled], Diabetes, Stroke [Doubled]—Vascular disease, Age 65—74 and Sex category [Female]; CRT, cardiac resynchronization therapy; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; PVC, premature ventricular contraction; VF, ventricular fibrillation; VT, ventricular tachycardia.

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and ventricular arrhythmias in this setting, from medications and sedation to ablation and more exceptional therapy. It also covers the occasional indications of resynchronization in refractory heart failure and the interest for haemodynamic assistance when specific therapy fails.


**MOTS CLÉS**
Choc cardiogénique ;
Fibrillation atriale ;
Orage rythmique ;
Ablation par cathéter

**Résumé** Les arythmies cardiaques survenant chez les patients en Unité de Soins Intensifs aggravent les symptômes et le pronostic et nécessitent une correction urgente. L'orage rythmique est un motif fréquent d'admission aux Soins Intensifs. Une prise en charge rapide, efficace et spécifique de ces patients présentant des arythmies variées est donc indispensable et doit être connue de tout médecin impliqué dans les Unités de Soins Intensifs. Cet article résume les connaissances actuelles dans la prise en charge des arythmies supraventriculaires et ventriculaires qui peuvent nécessiter un traitement par antiarythmique, sédation et/ou ablation, ainsi que les indications occasionnelles de resynchronisation pour les patients en insuffisance cardiaque réfractaire et l'intérêt des assistances hémodynamiques quand ces thérapies sont insuffisantes.


## Background

The interactions between arrhythmias and chronic heart failure have been well established. In contrast, the mechanisms and management of sustained arrhythmia for patients with cardiogenic shock in intensive cardiac care units are less clear. The treatment of these arrhythmias in the setting of severe acute heart failure is often urgent, with the aim to limit rapid heart rates that may exacerbate heart failure. This manuscript addresses some issues related to the acute management of sustained atrial or ventricular arrhythmias and the need for resynchronization therapy for patients hospitalized in intensive care units, with a more specific emphasis on therapeutic options for patients with electrical storm.

## Atrial fibrillation, atrial flutter and other supraventricular tachycardias

Atrial fibrillation (AF) is the most common arrhythmia that can occur in patients with heart disease in general and, specifically, in the intensive care unit [1]. It may precipitate heart failure by worsening left ventricular filling and lowering left ventricular ejection fraction (LVEF). The risk of thromboembolism should be managed, in all cases, according to the most recent guidelines [2] before cardioversion and according to $CHA_2DS_2$-VASc (Cardiac failure, Hypertension, Age $\geq$ 75 [Doubled], Diabetes, Stroke [Doubled]—Vascular disease, Age 65—74 and Sex category [Female]) score evaluation for long-term anticoagulation. However, in patients with acute cardiogenic shock, cardioversion may be used urgently, without performing a transoesophageal echocardiogram to rule out the presence of left atrial thrombus, if urgent transoesophageal echocardiography is unavailable or felt to carry some risk in very sick or unstable patients. Furthermore, unless the patient has a typical presentation, it can be difficult to confirm whether AF is the cause (tachycardiomyopathy in a patient with no history of heart failure or any underlying heart disease) or the consequence of an underlying cardiomyopathy. Patients with tachycardiomyopathy may have a much better prognosis once converted into sinus rhythm [3,4]. In this setting, a precise history of the arrhythmia and timing of events is needed in order to decide on the best treatment.

Current guidelines [2] classify AF into paroxysmal, persistent, long-lasting persistent or permanent AF. This classification may help to make a decision regarding AF ablation according to the likely success of the procedure. For paroxysmal and persistent AF, the success rate is generally high or acceptable, but success is less likely in patients with long-lasting persistent or permanent AF, so other treatment strategies may be chosen. Ablation is best performed when the haemodynamic status of the patient has been stabilized. Therefore, three situations should be distinguished:

- is AF recently occurring in a patient with established heart disease?
- is AF causing tachycardiomyopathy?
- is it a permanent AF leading to heart failure by the persistence of an accelerated/irregular ventricular rhythm?

In any situation, the management strategy will first depend of the identification of underlying causes (thyrotoxicosis, electrolyte disorders, uncontrolled hypertension, valvular disease) or precipitating factors that may be corrected (pulmonary infection, post-surgery, myocardial ischaemia or alcohol).

For new or recent-onset AF, treatment should be to terminate AF and prevent future recurrences using antiarrhythmic drugs, followed by AF ablation if needed. Urgent electrical cardioversion is recommended if AF is thought to be contributing to a worsening of haemodynamic status in order to improve the clinical condition (Class I A) [2]. Concomitantly,

an intravenous bolus of amiodarone (see below) may be recommended in preparation for direct current cardioversion to improve the success rate. However, it should be remembered that, in patients with severe haemodynamic conditions, amiodarone could be deleterious due to its additional calcium channel blocking properties, beta-blocker and class 1 effects and/or the vasodilator effect of the solvent. Oral loading may be preferred in these cases, according to the degree of urgency. Anterior—posterior electrode placement and biphasic waveforms provide almost complete acute success of electrical cardioversion. AF recurrences are, however, frequent [5]. Immediate and early recurrences of AF should be distinguished from cardioversion failure [6]. These phenomena have been suggested to be related to repetitive ectopic atrial activity or changes in cellular electrophysiology. Using intravenous amiodarone, if not contraindicated, may help to maintain sinus rhythm. Thyrotoxicosis and hypokalaemia should be investigated when AF recurs within minutes or seconds. In case of tachycardiomyopathy, early recurrence of AF despite anti-arrhythmic drugs (mainly amiodarone) may lead to an indication for urgent pulmonary vein isolation with or without additional ablation targets in selected cases, with the possibility of curing the patient in the longer term.

For permanent AF with a high ventricular rate, the ventricular rate may be controllable with specific pharmacological therapy. Intravenous amiodarone (150 mg over 10 minutes, then 1 mg/minute for 6 hours followed by 0.5 mg/minute for 18 hours) and digoxin (0.25 mg intravenous bolus and 0.75—1.5 mg over 24 hours) may be used in this critical situation to control ventricular ventricular rate. Beta-blockers may be used once the haemodynamic status of the patient has stabilized or when heart failure is mainly driven by diastolic dysfunction. Calcium channel blockers should definitely be avoided in patients with low LVEF. Atrioventricular junction ablation [7] and cardiac resynchronization therapy (CRT) implantation (when LVEF is reduced) are only used after pharmacological treatment has failed or is contraindicated. Due to the critical situation, a CRT implantation should only be performed, if possible, once the haemodynamic status has stabilized. Atrioventricular junction ablation should be performed very soon after CRT implantation in these cases. In the absence of distal conduction disturbances, His bundle pacing is another option. While the optimal resting heart rate has been suggested to be around 110 bpm in patients with permanent AF in the absence of heart failure, a lower rate is probably better in patients with heart failure, in whom a heart rate of 70—100 bpm may be targeted. In acute heart failure, the optimal rate will depend on the haemodynamic response of the patient.

Atrial flutter should be managed as AF in case of cardiogenic chock: anticoagulation and cardioversion/ablation when the ventricular rhythm is not controlled. Ablation of cavotricuspid isthmus is much easier than AF ablation and is feasible even when a patient is in a relatively unstable haemodynamic condition.

Other supraventricular tachycardias can also occur [8], such as sinus node reentry or atrial tachycardia and, more rarely, junctional ectopic tachycardia or incessant atrioventricular node reentrant tachycardia (AVNRT), incessant atrioventricular reciprocating tachycardia (AVRT) or permanent junctional reciprocating tachycardia. These arrhythmias are not always previously known about. Atrial tachycardia is more frequent in patients with comorbidities such as chronic obstructive pulmonary disease, coronary artery disease or chronic heart failure, and may be induced by an acute infection, particularly in elderly patients [9]. Vagal manoeuvres may help to differentiate AVNRT and AVRT from atrial tachycardia by stopping AVNRT and AVRT. Adenosine may sometimes be needed and can be proposed even in patients with severe heart failure due to its short half-life. Amiodarone may be necessary in patients with cardiogenic shock, as well as digoxin (provided risk of rapid preexcitated tachycardia is excluded), as other antiarrhythmic agents are contraindicated [10], with the exception of flecainide in case of rapid AF in patients with Wolff-Parkinson-White syndrome. Ablation should be considered quickly as an option when tachycardia is resistant to pharmacological treatment.

## Electrical storm

### Definition and epidemiology

Electrical storm is currently defined by: three or more distinct episodes of sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) over 24 hours; incessant VT for > 12 hours per day; or three or more appropriate implantable cardioverter-defibrillator (ICD) therapies in 24 hours (separated by 5 minutes from each other) in patients with an ICD [11]. Electrical storm happens in 10—20% of patients implanted for secondary prevention and in around 5% of those implanted for primary prevention [12—15] during a follow-up of a few years. Therefore, the prevalence in the longer term is probably higher, making electrical storm a daily clinical situation.

The prognosis of electrical storm has repeatedly been found to be poor, mainly caused by a high short-term mortality (10—20% at 48 hours) (especially when no therapeutic solution can be found), reaching 20—35% at longer-term follow-up [13,14,16,17]. Even though no difference in prognosis has sometimes been reported compared to controls [12,14], electrical storm is considered as an independent risk factor by some other authors [13]. Electrical storm was a strong mortality risk factor with a risk ratio > 3 and was associated with an increased combined risk of death, heart transplantation and hospitalization for heart failure in a meta-analysis [18]. This is probably due to both the specific population (end-stage heart disease) and the repetitive ICD shocks, which are highly suspected to increase mortality [19,20].

Populations prone to electrical storm are mainly represented by elderly men with ischaemic cardiomyopathy and low LVEF [12—14,21,22]. Recurring monomorphic VT is the most common arrhythmia [21]. Chronic renal failure, ischaemic heart disease, lack of beta-blocker use and wide QRS complexes have also been associated with electrical storm [23—25]. Many factors that can act as triggers have been described, such as acute ischaemia, electrolyte imbalance, congestive heart failure, sepsis or fever, thyrotoxicosis, proarrhythmic drug effects, digitalis use and recent surgery, and these should be eliminated when possible [11]. Unfortunately, precipitating factors remain

unidentified in 30% of cases [14] and are reversible in < 10% of cases [11].

## Management of electrical storm

Acute management of electrical storm includes several therapies or actions that should be immediately and concomitantly administrated. Search for a treatable trigger, sedation, standard reanimation and intravenous or oral antiarrhythmic drugs (according to the emergency level) are the first-step therapies. Management of electrical storm should be tailored to the patient's characteristics and conditions and the clinical setting (Table 1).

### Preadmission management

Trigger elimination using anti-ischaemic medications and, eventually, thrombolysis, potassium and magnesium infusion or isoproterenol infusion should be done according to the clinical setting. Coronary artery revascularization is essential in the setting of acute ischaemia or acute coronary syndrome, especially for polymorphic VT or VF. Other preadmission treatments include:

- haemodynamic management (vasopressive amines), sedation (see below).
- antiarrhythmic drugs (beta-blocker, amiodarone, lidocaine) or isoproterenol for special conditions (see below).
- stopping ICD therapy by putting a magnet on the device can (only once the patient is carefully monitored by appropriate medical or paramedic staff) and using external direct current shocks when strictly needed.

### Hospitalization in intensive care units

- A 12-lead electrocardiogram should be repeated in order to document and characterize premature ventricular contractions (PVCs) or VT.
- Elimination of the trigger when possible.

**Table 1** Management of VT/VF storm (adapted from Pedersen et al. [62]).

| |
|---|
| Admission to the intensive care unit |
| ICD reprogramming |
| Management of underlying causes or precipitating factors (ischaemia, electrolyte disturbances, proarrhythmic drugs) |
| Beta-blockade |
| Antiarrhythmic therapy (amiodarone as first-line therapy) |
| Sedation, intubation/deep sedation |
| Catheter ablation favouring substrate ablation |
| Consider risk/benefit ratio of mechanical haemodynamic support |
| Consider neuraxial modulation (thoracic epidural anaesthesia, cardiac sympathetic denervation) and, finally, heart transplantation when previous steps are unsuccessful |

ICD: implantable cardioverter-defibrillator; VF: ventricular fibrillation; VT: ventricular tachycardia.

- ICD interrogation (for ruling out inappropriate therapy), reprogramming for avoiding ICD shocks (which may deplete the battery) and avoiding/delaying unnecessary interventions or deprogramming left ventricular pacing (which may induce recurring VT in some cases).
- Using ICD for allowing higher rates of pacing (which may have a preventive action).
- In patients without an ICD or pacemaker, transient rapid pacing using a percutaneous electrode catheter may allow painless repetitive terminations of VT and prevention of recurrences in some cases.

Many of the above-mentioned techniques are used to interrupt the vicious circle of increased adrenergic tone caused by ICD shocks, which may reinduce arrhythmias. In this context, sedation is an essential step during electrical storm. It allows disruption of increasing adrenergic tone (due to stress and pain caused by repetitive ICD shocks) and thus has an efficient preventive role for recurring arrhythmias. Anxiolytic drugs (benzodiazepines such as midazolam) or remifentanyl are usually considered [11]. Propofol is used when deeper sedation is sought [11]. Dexmedetomidine, which possesses additional alpha 2 agonist properties, can also be used in this setting [11]. General anaesthesia and assisted ventilation should be rapidly proposed in case of recurrent arrhythmias despite sedation [11].

### Antiarrhythmic drugs

Antiarrhythmic drug therapy is the cornerstone and first-line specific therapy in electrical storm and should be proposed for every patient with electrical storm or recurring malignant ventricular arrhythmias. The choice of the drug depends on the clinical setting, type of cardiomyopathy and type of arrhythmia. Simultaneous prescription of different drugs should be carefully considered in case of severe cardiomyopathy, where sedation/general anaesthesia may be a preferred first option (Table 2). Currently, three main drugs are used in most situations (beta-blockers, amiodarone and lidocaine).

Beta-blockers are the cornerstone of antiarrhythmic agents in electrical storm, decreasing the harmful consequences of exaggerated adrenergic tone that is present in electrical storm. Oral or intravenous beta-blockers should urgently be prescribed according to the emergency level and state of consciousness. Intravenous infusions of propranolol (0.15 mg/kg over 10 minutes), metoprolol (2—5 mg every 5 minutes up to three doses in 15 minutes) or esmolol (300—500 mg/kg over 1 minute) may be used [11]. The only contraindications are severe asthma, relevant bradycardia without pacing capabilities and cardiogenic shock in patients with altered ventricular systolic function.

Amiodarone has been shown to decrease recurrences of arrhythmias from 53 to 12% in electrical storm in retrospective studies [14]. This is the only class 3 antiarrhythmic drug that has been shown to cause a significant decrease in VT recurrence rates in ICD-implanted patients in meta-analyses, although it does not have a significant effect on long-term mortality [26]. In acute myocardial infarction complicated by out-of-hospital cardiac arrest due to VT or VF, amiodarone decreases mortality at admission compared to placebo and is more efficient that lidocaine [27]. An intravenous infusion of amiodarone may be preferred in

**Table 2** Haemodynamic effects of antiarrhythmic drugs in heart failure (adapted from Santangeli et al. [47]).

| Haemodynamic effects of antiarrhythmic drugs | | | | | | |
|---|---|---|---|---|---|---|
| Vaughan Williams Class Drug | Arterial blood pressure | PCWP | Cardiac output | Systemic vascular resistance | Predominant effect | |
| | | | | | Vasodilatation | Myocardial depression |
| Class IA | | | | | + | |
| Quinidine | ↘ | ≈ | ↗ | | | |
| Disopyramide[a] | ≈ | ↗ | ↘ | ≈ | | ++ |
| Class IB | | | | | | |
| Lidocaine | ≈ | ≈ | ≈ | ≈ | | |
| Mexiletine | ↗ | ↗ | ↘ | ↗ | | + |
| Class IC | | | | | | |
| | ≈/↘ | ↗ | ↘ | ≈ | | ++ |
| Flecaine/propafenone[a] | | | | | | |
| Class III | | | | | | |
| Amiodarone | ≈/↘ | ↗ | ≈/↘ | ≈/↘ | + | ≈/+ |
| Sotalol | ↘ | ↗ | ↘ | ≈ | ≈/+ | + |
| Dronedarone[a] | ↘ | ↗ | ↘ | ↘ | | ++ |

PCWP: pulmonary capillary wedge pressure.
[a] Contraindicated in heart failure patients.

acute and urgent situations (150 mg over 10 minutes, then 1 mg/minute for 6 hours, then 0.5 mg/minute for 18 hours; maximum 2 g over 24 hours), followed by oral administration as soon as possible (800—1200 mg/day in divided doses until a total of 10 g has been given; then 200—400 mg/day) [11,28].

Class 1 drugs, especially intravenous lidocaine (1 mg/kg then 30 mg/kg over 24 hours) and oral mexiletine (200—400 mg three times a day, taking into consideration some specific restrictions in France) [11] may be efficient, especially in the setting of myocardial ischaemia, because sodium channel blockade is increased by tissular acidosis/hyperkalaemia. They may be used after failure of beta-blockers and amiodarone. Caution should, however, be observed for patients with very altered EF. The use of other class 1 drugs is more exceptional due to their negative inotropic and proarrhythmic properties, but may be proposed in healthy hearts or structural heart diseases with preserved EF. In a meta-analysis, lidocaine was found to decrease mortality at admission after out-of-hospital cardiac arrest [29]. Very recently, quinidine has also been demonstrated to be effective for recurring polymorphic VT in the setting of acute myocardial infarction or after coronary revascularization procedures [30].

Magnesium infusion has no role in electrical storm, except for long QT-related arrhythmias or in case of potassium/magnesium depletion [29].

Finally, in very special situations (Brugada syndrome, malignant early repolarization, idiopathic VF or short QT syndrome), isoproterenol infusion (0.1 to 1—3 μg/minute to achieve sufficient heart rate increase) and oral hydroquinidine (300—600 mg, 2—3 times a day, with plasma dosing monitoring) may be the only effective therapies [31—35]. Beta-blockers, magnesium infusion and fast pacing should be used in case of electrical storm occurring in congenital long QT, while recurring torsades de pointes in acquired long QT may be treated according to the triggering event (isoproterenol then fast pacing in bradycardia-induced long QT, potassium and magnesium in drug-induced long QT) [35].

In selected cases, patients with unstable VT or repetitive VF may also benefit from mechanical haemodynamic support, such as extracorporeal membrane oxygenation, catheter-based microaxial flow pump (e.g. Impella™) or intra-aortic balloon pump [11]. Haemodynamic support can reduce the arrhythmic burden by increasing coronary perfusion and reducing afterload, myocardial wall stress and myocardial oxygen demand. This also prevents multiple organ failure [11]. However, because of inherent complications, except for refractory cases not allowing any delay, mechanical support should be better proposed after failure of ablation, if possible. Alternatively, ablation in very high-risk patients may be performed with mechanical support (see the *Haemodynamic support and VT ablation* section).

Finally, more exceptional therapy may be proposed in refractory cases, such as neuraxial sympathetic modulation [11] (thoracic epidural anaesthesia or left or bilateral cardiac sympathetic denervation by surgical or thoracoscopy access), renal denervation or even heart transplantation, although this should remain exceptional for arrhythmic grounds only and proposed only after failure of every other therapy (Table 1).

## Radiofrequency ablation

### *Indications*

Even if a mortality benefit has never been demonstrated in randomized controlled trials, catheter ablation has

repeatedly shown its superiority to medical therapy in reducing arrhythmic burden [26,36]. Moreover, freedom from recurrent VT after ablation has been associated with improved survival [37—39]. For these reasons, catheter ablation should not be considered a last resort therapy, but a valuable option in patients presenting with electrical storm related to structural heart disease in the absence of a reversible cause. Radiofrequency catheter ablation is effective, not only in the acute management of electrical storm, leading to a control of ventricular arrhythmias in up to 80—90% of patients, but also over longer-term follow-up, improving VT- and electrical storm-free survival [22]. However, performing VT ablation at an earlier stage (after a first episode of sustained VT) may be more beneficial and is a class IIa indication in patients with ischaemic cardiomyopathy to reduce VT recurrence and electrical storm [40].

In the recent VANISH trial, a trend towards a 34% relative risk reduction of electrical storm recurrences was observed in patients treated by ablation compared to escalation of antiarrhythmic drugs [36]. In a pooled meta-analysis, which included 471 patients with electrical storm treated invasively by different ablation strategies, acute elimination of all inducible ventricular arrhythmias was reached in 72% of cases [21]. The clinical arrhythmia was effectively suppressed in 91% of patients, with a complication rate of 2% and a procedure-related death rate < 1%. After a median follow-up of 1.2 years, 94% of patients were free from electrical storm and 72% were free from any VT. Overall mortality was 17% at 1.2-year follow-up, with most of the deaths related to progressive heart failure (62%) [21].

Electrical storm is the reason for VT ablation in 20—35% of cases [22,39,41]. The latest guidelines from the European

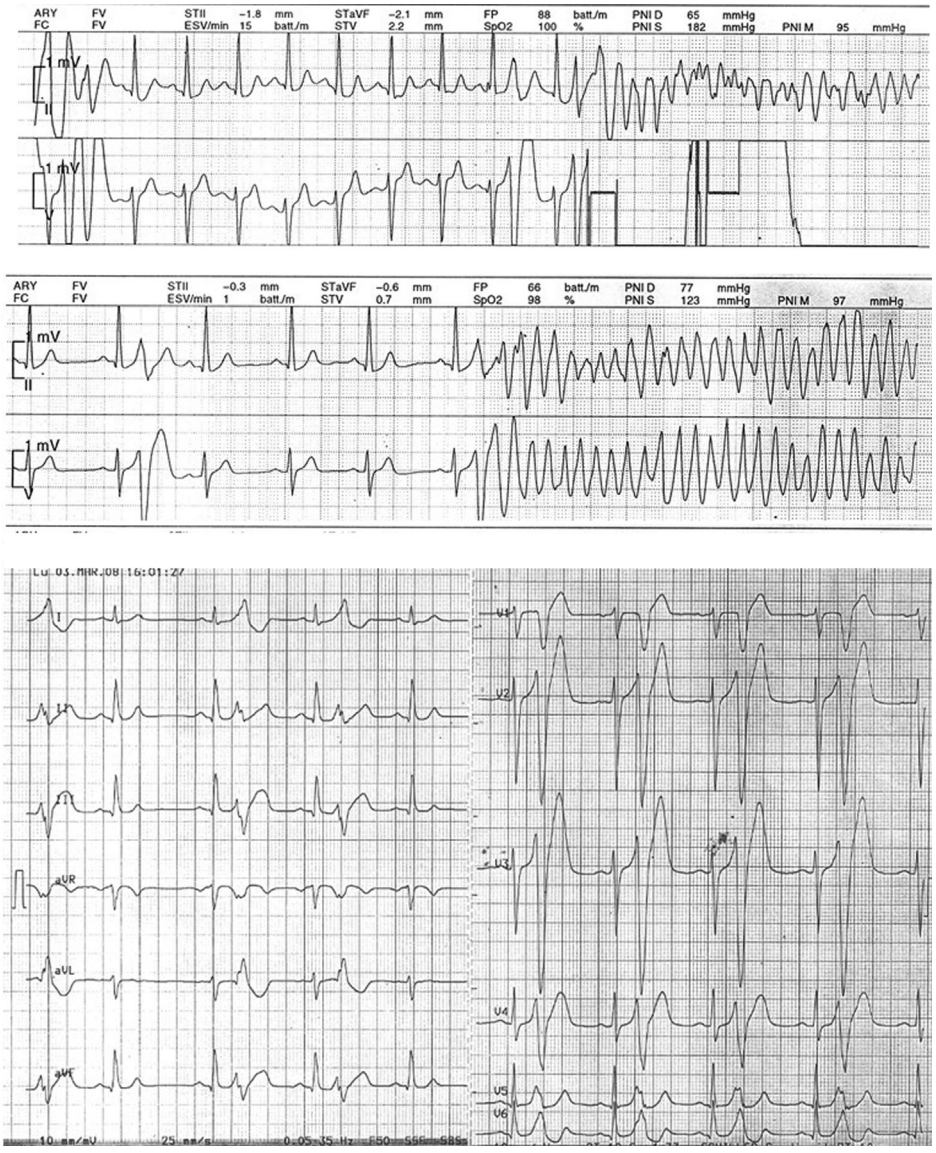


**Figure 1.** Example of a 42-year-old patient without structural heart disease who suffered 20 shocks within 24 hours. The two upper panels show initiation of VF triggered by a monomorphic PVC. The lower panel shows a 12-lead electrocardiogram identifying the initiating PVC as coming from the right Purkinje network, which is an important piece of information in case of ablation. PVC: premature ventricular contractions.

Society of Cardiology [40] mention a class 1 indication with a level of evidence B in the absence of transient reversible cause for urgent catheter ablation in patients with scar-related heart disease presenting with incessant VT or electrical storm to stop electrical storm, reduce VT recurrence and improve outcomes. Concerning polymorphic VT/VF in coronary artery disease, radiofrequency catheter ablation at a specialized ablation centre followed by the implantation of an ICD should be considered in patients with recurrent VT, VF or electrical storm despite complete revascularization and optimal medical treatment (Class IIa, level of evidence C). In patients with polymorphic VT/VF without structural heart disease, catheter ablation for long-term suppression/prevention of an electrical storm or recurrent ICD discharges should be considered. Most of the time, it will target the initiating PVCs (Class 1 indication, level of evidence B), which often come from the Purkinje system. Continuous 12-lead electrocardiograms should therefore be recorded to identify PVC origin (Fig. 1). Cardiac imaging may also help to identify and locate a specific substrate for the arrhythmia (Fig. 2). In the setting of Brugada syndrome, substrate ablation targeting the epicardial right ventricular outflow tract/right ventricle free wall is the main target. Plymorphic VT/VF ablation should be optimally performed in experienced centres.

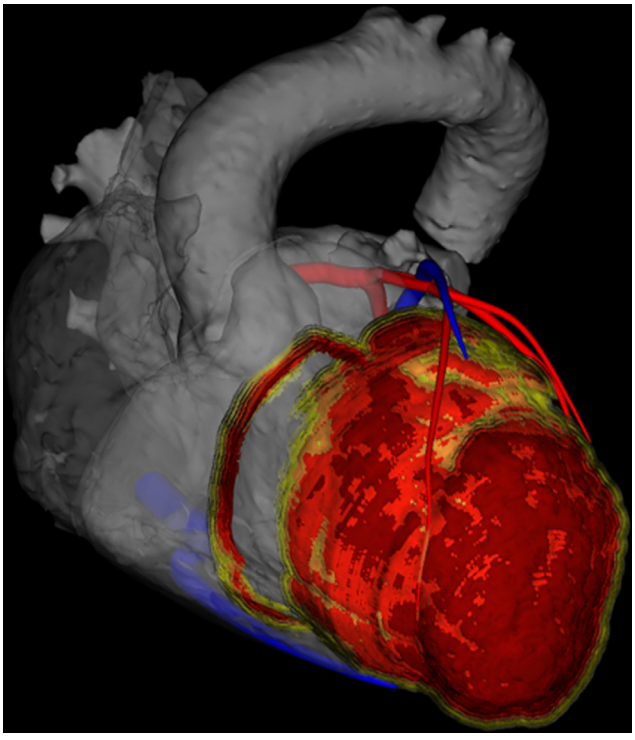

**Figure 2.** Computed tomography scan of a patient with electrical storm in the setting of an old large anterior myocardial infarct analysed using the music software (Inheart®). Colours from red to yellow represent the scar through the different thickness of the myocardium (red being 1 mm and yellow 4 mm). The heterogeneity of thickness is the anatomical substrate for potential isthmii, this shows the complexity of the scar, may explain electrical storm and offers a potential target for ablation. These images can be integrated into three-dimensional mapping systems.

### *Techniques*

Patients in the electrophysiology laboratory need to be in a relatively stable condition (as stable as possible). Ablation will require the patient to lay flat for 2—4 hours without moving. If the patient keeps having shocks or cannot lay flat, sedation and/or haemodynamic support before ablation should be discussed. The PAINESD score has been shown to predict acute haemodynamic decompensation during VT ablation procedures (Table 3) [42]. In our experience, mechanical haemodynamic support is rarely needed for catheter ablation even in the context of electrical storm, especially when performing substrate ablation and not VT activation mapping. In practice, mechanical haemodynamic support should be anticipated in case of cardiogenic shock and frequent recurrent ICD shocks just prior to ablation.

Left ventricular thrombus needs to be ruled out before starting ablation, as thrombus may be found in 4—5% of patients referred for VT ablation, particularly in case of ventricular aneurysm/low ejection fraction. In case of AF and/or transseptal access, checking for left atrial appendage thrombus is appropriate. Transthoracic echocardiography with or without contrast is the minimal examination for left ventricular thrombus evaluation and, if needed, transoesophageal echocardiography can rule out left atrial appendage thrombus. If feasible, contrast cardiac computed tomography may eliminate all intracardiac thrombi. In the presence of intraventricular thrombus and recurrent electrical storm, epicardial access may be discussed [43]. In case of left atrial appendage thrombus, the procedure should be postponed. In case of untractable storm, a retrograde aortic approach without induction and a strategy based solely on substrate ablation should be favoured.

Three-dimensional mapping systems are almost systematically used for VT ablation. Although there is some debate concerning the best strategy for VT ablation, there is increasing evidence favouring substrate ablation versus clinical VT ablation alone [44]. In the setting of electrical storm,

**Table 3** PAINESD risk score [42]: proposed scoring system to identify patients at high risk of haemodynamic decompensation undergoing catheter ablation that may benefit from prophylactic mechanical circulatory support.

| Variable | Score (maximum = 31) | |
|---|---|---|
| Pulmonary disease (chronic obstructive) | 5 | Low risk: ≤ 8 |
| Age > 60 years | 3 | |
| Ischaemic cardiomyopathy | 6 | Intermediate risk: 9—14 |
| NYHA class III or IV | 6 | |
| Left ventricular Ejection fraction < 25% | 3 | High risk: ≥ 15 |
| Electrical Storm | 5 | |
| Diabetes mellitus | 3 | |

NYHA: New York Heart Association.

data are lacking. On one hand, all effort should be made to eliminate the clinical VT responsible for the storm, but on the other hand, performing the procedure in sinus rhythm makes it safer with less risk of haemodynamic impairment. Typically, a substrate map is performed in sinus/paced rhythm. During map acquisition, pacemapping can be performed to identify VT exit. de Chillou et al. [45] proposed a pacemap to identify the VT isthmus. Then, ablation is performed starting in the scar area of VT exit and extended to the entire substrate if the patient is stable enough. In case of incessant slow VT, the procedure starts with activation mapping and VT ablation. Although there are no robust data, utilization of a catheter with limited irrigation may be better in patients with low ejection fraction with less fluid infusion. Non-inducibility of clinical VT is the minimal endpoint, otherwise the outcome is extremely poor [22]. However, the procedure should optimally aim at substrate elimination, if the patient is stable enough.

A specific entity concerns patients presenting with electrical storm a few days after poorly/late/non-revascularized myocardial infarction. They may present with recurrent polymorphic VT/VF initiated by PVC induced by surviving Purkinje fibres within the scar. There is sometimes pause-dependent initiation and, therefore, pacing at a fast and stable rate (90—100 bpm) may prevent VT initiation. However, if polymorphic VTs continue, ablation of the surviving Purkinje fibres in the scar may effectively stop the storm [40,46].

### Haemodynamic support and VT ablation

Since VT and heart failure are linked, one should be mindful of both electrophysiological and haemodynamic status before VT ablation when managing electrical storm [47]. Acute haemodynamic decompensation occurred in 11% of patients during VT ablation in a single-centre study of 193 patients treated for drug-refractory scar-related VT [42]. Acute haemodynamic decompensation was defined as sustained hypotension (systolic blood pressure < 80—90 mmHg) despite increasing doses of vasopressors and requiring mechanical haemodynamic support and/or procedure discontinuation. When acute haemodynamic decompensation occurred during catheter ablation, the risk of death was six-fold higher than in patients without acute haemodynamic decompensation.

In a multicentre study of VT ablation and heart failure, mechanical haemodynamic support was needed in 7% of patient with New York Heart Association (NYHA) class II—III and in 22% of those with NYHA class IV [48]. While the level of evidence for intra-aortic balloon pumps in this setting seems marginal, several other mechanical haemodynamic support systems have been evaluated for VT ablation. These include: extra-corporeal life support (including extracorporeal membrane oxygenation), the Impella™ percutaneous ventricular assist device (Abiomed, Danvers, Massachusetts) and the TandemHeart™ device (CardiacAssist, Pittsburgh, Pennsylvania). There is no direct comparison between these devices in the clinical setting, but extracorporeal life support demonstrated a better haemodynamic effect on mean arterial pressure during fast VT (300 bpm) and VF over the Impella™ and TandemHeart™ devices in a swine model. [49]. Impella™ can be relatively easily inserted and managed by an electrophysiologist, but it is only able to deliver 2.5 L/minute output with Impella 2.5. If a higher flow rate is needed, use of extracorporeal life support, TandemHeart™ or Impella CP/5™ must be chosen, but this requires surgical insertion. Of note, Impella™ cannot be used together with retrograde aortic access for ablation, while transseptal access is not possible using TandemHeart™.

No randomized study has specifically evaluated the benefit of mechanical haemodynamic support in VT ablation. Using the PAINESD score (Table 3 [42]) to stratify the risk of acute haemodynamic decompensation, Santangeli et al. suggested that extracorporeal life support may be implanted prior to ablation for patients with a score > 15 [42]. However, activation/entrainment mapping was used in this study, and this strategy may enhance acute haemodynamic decompensation. In this high-risk population, ablation substrate modification in sinus rhythm should be preferred, obviating the need for VT induction and mapping, and survival free from VT being better compared to conventional entrainment/activation mapping [44].

When electrical storm is triggered by recurrent VF or poorly tolerated VT, and when drugs and sedation have failed, extracorporeal life support may be a prudent option. However, mechanical haemodynamic support carries an additional risk and significant morbidity. Mechanical haemodynamic support implantation as a rescue procedure during VT ablation for drug-refractory cardiogenic shock confers a very poor outcome, with 16 of 21 patients dying in the first 10 days in one study [50].

General anaesthesia and drugs such as propofol induce vasoplegia and hypotension. When compared to conscious sedation, general anaesthesia is associated with more use of vasopressors (87% vs 12%) [51]. Monitoring usually only relies on invasive blood pressure, while non-invasive cerebral oximetry offers additional monitoring value [52].

Studies of mechanical haemodynamic support have not demonstrated any impact on VT-free survival [53], but this can allow a longer time for activation and entrainment mapping and more VT terminations during ablation. Thus, systematic prophylactic mechanical haemodynamic support implantation before ablation still needs more evidence to be widely adopted. To prevent acute haemodynamic decompensation, careful evaluation of haemodynamic status is necessary, general anaesthesia should be avoided (when possible) and substrate ablation should be preferred.

## Cardiac resynchronization therapy

Regardless to the type of device (CRT-pacemaker or CRT-defibrillator), there are only a few conflicting reports about the use of CRT in patients with left bundle branch block, reduced LVEF and end-stage class IV heart failure requiring continuous intravenous inotropic therapy or in cardiogenic shock. Guidelines have not yet defined the role of CRT in such patients [54].

Previous experience in patients on inotropic therapy have suggested that CRT would not be indicated in such cases due to very high mortality [55,56]. However, in these reports, most patients had ischaemic cardiomyopathy. In other series [57—59], favourable responses were obtained

**Table 4** Key messages for the management of arrhythmias in the context of cardiogenic shock.

| |
|---|
| Atrial fibrillation |
| New or recent onset or suspicion of tachycardiomyopathy: cardioversion after amiodarone loading or urgent ablation when unsuccessful |
| Permanent: rate control and atrioventricular node ablation and pacing if unsuccessful |
| Supraventricular tachycardia (flutter or other types) |
| Rate control, urgent cardioversion or ablation |
| Recurrent ventricular tachycardia/fibrillation |
| ICD reprogramming |
| Management of precipitating factors |
| Beta-blockade and antiarrhythmic therapy |
| Sedation, general anaesthesia |
| Ablation |
| Mechanical haemodynamic support when needed |
| Consider neuraxial modulation and/or heart transplantation when previous steps are unsuccessful |

ICD: implantable cardioverter-defibrillator.

with CRT. In these series, dilated cardiomyopathy was more often present, possibly explaining these divergent results, as patients with dilated cardiomyopathy are likely to be better responders to CRT than those with ischaemic cardiomyopathy [11].

Recent case reports have shown clinical improvement after CRT implantation in patients with left bundle branch block who are concomitantly on intra-aortic balloon pump support [60] or left ventricular assist device [61]. The rapid haemodynamic improvement on CRT seems to have an additive effect on the unloading of the left ventricle and improved organ perfusion achieved by the left ventricular assist device or intra-aortic balloon pump. Therefore, this combined treatment should be discussed when the risk of complications using mechanical devices is high in these unstable patients.

## Conclusion

The development of sustained atrial or ventricular arrhythmias in patients with cardiogenic shock who are hospitalized in intensive cardiac care units may require individualized evaluation and proactive management. Key messages for the management of arrhythmias in the context of cardiogenic shock are included in Table 4. A systematic and holistic approach is needed to improve the management of patients with these arrhythmias and to reduce the risk of lethal worsening of haemodynamic status.

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## Disclosure of interest

The authors declare that they have no competing interest.

## References

[1] Moss TJ, Calland JF, Enfield KB, Gomez-Manjarres DC, Ruminski C, DiMarco JP, et al. New-onset atrial fibrillation in the critically ill. Crit Care Med 2017;45:790—7.
[2] Kirchhof P, Benussi S, Kotecha D, Ahlsson A, Atar D, Casadei B, et al. 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. Eur Heart J 2016;37:2893—962.
[3] Nerheim P, Birger-Botkin S, Piracha L, Olshansky B. Heart failure and sudden death in patients with tachycardia-induced cardiomyopathy and recurrent tachycardia. Circulation 2004;110:247—52.
[4] Donghua Z, Jian P, Zhongbo X, Feifei Z, Xinhui P, Hao Y, et al. Reversal of cardiomyopathy in patients with congestive heart failure secondary to tachycardia. J Interv Card Electrophysiol 2013;36:27—32.
[5] Mayr A, Ritsch N, Knotzer H, Dunser M, Schobersberger W, Ulmer H, et al. Effectiveness of direct-current cardioversion for treatment of supraventricular tachyarrhythmias, in particular atrial fibrillation, in surgical intensive care patients. Crit Care Med 2003;31:401—5.
[6] Chugh A, Ozaydin M, Scharf C, Lai SW, Hall B, Cheung P, et al. Mechanism of immediate recurrences of atrial fibrillation after restoration of sinus rhythm. Pacing Clin Electrophysiol 2004;27:77—82.
[7] Hoffmayer KS, Scheinman M. Current role of atrioventricular junction (AVJ) ablation. Pacing Clin Electrophysiol 2013;36:257—65.
[8] Boriani G, Fauchier L, Aguinaga L, Beattie JM, Blomstrom Lundqvist C, Cohen A, et al. European Heart Rhythm Association (EHRA) consensus document on management of arrhythmias and cardiac electronic devices in the critically ill and post-surgery patient, endorsed by Heart Rhythm Society (HRS), Asia Pacific Heart Rhythm Society (APHRS), Cardiac Arrhythmia Society of Southern Africa (CASSA), and Latin American Heart Rhythm Society (LAHRS). Europace 2019;21:7—8.
[9] Lowenstein SR, Halperin BD, Reiter MJ. Paroxysmal supraventricular tachycardias. J Emerg Med 1996;14:39—51.
[10] Holt P, Crick JC, Davies DW, Curry P. Intravenous amiodarone in the acute termination of supraventricular arrhythmias. Int J Cardiol 1985;8:67—79.
[11] Muser D, Santangeli P, Liang JJ. Management of ventricular tachycardia storm in patients with structural heart disease. World J Cardiol 2017;9:521—30.
[12] Credner SC, Klingenheben T, Mauss O, Sticherling C, Hohnloser SH. Electrical storm in patients with transvenous implantable cardioverter-defibrillators: incidence, management and prognostic implications. J Am Coll Cardiol 1998;32:1909—15.
[13] Exner DV, Pinski SL, Wyse DG, Renfroe EG, Follmann D, Gold M, et al. Electrical storm presages nonsudden death: the antiarrhythmics versus implantable defibrillators (AVID) trial. Circulation 2001;103:2066—71.
[14] Greene M, Newman D, Geist M, Paquette M, Heng D, Dorian P. Is electrical storm in ICD patients the sign of a dying heart? Outcome of patients with clusters of ventricular tachyarrhythmias. Europace 2000;2:263—9.
[15] Sesselberg HW, Moss AJ, McNitt S, Zareba W, Daubert JP, Andrews ML, et al. Ventricular arrhythmia storms in postinfarction patients with implantable defibrillators for primary prevention indications: a MADIT-II substudy. Heart Rhythm 2007;4:1395—402.
[16] Izquierdo M, Ruiz-Granell R, Ferrero A, Martinez A, Sanchez-Gomez J, Bonanad C, et al. Ablation or conservative management of electrical storm due to monomorphic ventricular tachycardia: differences in outcome. Europace 2012;14:1734—2179.

[17] Bansch D, Oyang F, Antz M, Arentz T, Weber R, Val-Mejias JE, et al. Successful catheter ablation of electrical storm after myocardial infarction. Circulation 2003;108:3011—6.
[18] Guerra F, Shkoza M, Scappini L, Flori M, Capucci A. Role of electrical storm as a mortality and morbidity risk factor and its clinical predictors: a meta-analysis. Europace 2014;16:347—53.
[19] Poole JE, Johnson GW, Hellkamp AS, Anderson J, Callans DJ, Raitt MH, et al. Prognostic importance of defibrillator shocks in patients with heart failure. N Engl J Med 2008;359:1009—17.
[20] Moss AJ, Schuger C, Beck CA, Brown MW, Cannom DS, Daubert JP, et al. Reduction in inappropriate therapy and mortality through ICD programming. N Engl J Med 2012;367:2275—83.
[21] Nayyar S, Ganesan AN, Brooks AG, Sullivan T, Roberts-Thomson KC, Sanders P. Venturing into ventricular arrhythmia storm: a systematic review and meta-analysis. Eur Heart J 2013;34:560—71.
[22] Carbucicchio C, Santamaria M, Trevisi N, Maccabelli G, Giraldi F, Fassini G, et al. Catheter ablation for the treatment of electrical storm in patients with implantable cardioverter-defibrillators: short- and long-term outcomes in a prospective single-center study. Circulation 2008;117:462—9.
[23] Brigadeau F, Kouakam C, Klug D, Marquie C, Duhamel A, Mizon-Gerard F, et al. Clinical predictors and prognostic significance of electrical storm in patients with implantable cardioverter defibrillators. Eur Heart J 2006;27:700—7.
[24] Arya A, Haghjoo M, Dehghani MR, Fazelifar AF, Nikoo MH, Bagherzadeh A, et al. Prevalence and predictors of electrical storm in patients with implantable cardioverter-defibrillator. Am J Cardiol 2006;97:389—92.
[25] Verma A, Kilicaslan F, Marrouche NF, Minor S, Khan M, Wazni O, et al. Prevalence, predictors, and mortality significance of the causative arrhythmia in patients with electrical storm. J Cardiovasc Electrophysiol 2004;15:1265—70.
[26] Santangeli P, Muser D, Maeda S, Filtz A, Zado ES, Frankel DS, et al. Comparative effectiveness of antiarrhythmic drugs and catheter ablation for the prevention of recurrent ventricular tachycardia in patients with implantable cardioverter-defibrillators: a systematic review and meta-analysis of randomized controlled trials. Heart Rhythm 2016;13:1552—9.
[27] Kudenchuk PJ, Brown SP, Daya M, Rea T, Nichol G, Morrison LJ, et al. Amiodarone, lidocaine, or placebo in out-of-hospital cardiac arrest. N Engl J Med 2016;374:1711—22.
[28] Siddoway LA. Amiodarone: guidelines for use and monitoring. Am Fam Physician 2003;68:2189—96.
[29] Huang Y, He Q, Yang M, Zhan L. Antiarrhythmia drugs for cardiac arrest: a systemic review and meta-analysis. Crit Care 2013;17:R173.
[30] Viskin S, Chorin E, Viskin D, Hochstadt A, Halkin A, Tovia-Brodie O, et al. Quinidine-responsive polymorphic ventricular tachycardia in patients with coronary heart disease. Circulation 2019;139:2304—14.
[31] Maury P, Hocini M, Haissaguerre M. Electrical storms in Brugada syndrome: review of pharmacologic and ablative therapeutic options. Indian Pacing Electrophysiol J 2005;5:25—34.
[32] Belhassen B, Glick A, Viskin S. Excellent long-term reproducibility of the electrophysiologic efficacy of quinidine in patients with idiopathic ventricular fibrillation and Brugada syndrome. Pacing Clin Electrophysiol 2009;32:294—301.
[33] Haissaguerre M, Sacher F, Nogami A, Komiya N, Bernard A, Probst V, et al. Characteristics of recurrent ventricular fibrillation associated with inferolateral early repolarization role of drug therapy. J Am Coll Cardiol 2009;53:612—9.
[34] Bun SS, Maury P, Giustetto C, Deharo JC. Electrical storm in short-QT syndrome successfully treated with Isoproterenol. J Cardiovasc Electrophysiol 2012;23:1028—30.
[35] Al-Khatib SM, Stevenson WG, Ackerman MJ, Bryant WJ, Callans DJ, Curtis AB, et al. 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: executive summary. Circulation 2018;138:e210—71.
[36] Sapp JL, Wells GA, Parkash R, Stevenson WG, Blier L, Sarrazin JF, et al. Ventricular tachycardia ablation versus escalation of antiarrhythmic drugs. N Engl J Med 2016;375:111—21.
[37] Tung R, Vaseghi M, Frankel DS, Vergara P, Di Biase L, Nagashima K, et al. Freedom from recurrent ventricular tachycardia after catheter ablation is associated with improved survival in patients with structural heart disease: an International VT Ablation Center Collaborative Group study. Heart Rhythm 2015;12:1997—2007.
[38] Komatsu Y, Maury P, Sacher F, Khairy P, Daly M, Lim HS, et al. Impact of substrate-based ablation of ventricular tachycardia on cardiac mortality in patients with implantable cardioverter-defibrillators. J Cardiovasc Electrophysiol 2015;26:1230—8.
[39] Vergara P, Tung R, Vaseghi M, Brombin C, Frankel DS, Di Biase L, et al. Successful ventricular tachycardia ablation in patients with electrical storm reduces recurrences and improves survival. Heart Rhythm 2018;15:48—55.
[40] Priori SG, Blomstrom-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, Camm J, et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: the Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J 2015;36:2793—867.
[41] Sacher F, Tedrow UB, Field ME, Raymond JM, Koplan BA, Epstein LM, et al. Ventricular tachycardia ablation: evolution of patients and procedures over 8 years. Circ Arrhythm Electrophysiol 2008;1:153—61.
[42] Santangeli P, Muser D, Zado ES, Magnani S, Khetpal S, Hutchinson MD, et al. Acute hemodynamic decompensation during catheter ablation of scar-related ventricular tachycardia: incidence, predictors, and impact on mortality. Circ Arrhythm Electrophysiol 2015;8:68—75.
[43] Berte B, Yamashita S, Sacher F, Cochet H, Hooks D, Aljefairi N, et al. Epicardial only mapping and ablation of ventricular tachycardia: a case series. Europace 2016;18:267—73.
[44] Di Biase L, Burkhardt JD, Lakkireddy D, Carbucicchio C, Mohanty S, Mohanty P, et al. Ablation of stable VTs versus substrate ablation in ischemic cardiomyopathy: the VISTA randomized multicenter trial. J Am Coll Cardiol 2015;66:2872—82.
[45] de Chillou C, Groben L, Magnin-Poull I, Andronache M, Magdi Abbas M, Zhang N, et al. Localizing the critical isthmus of postinfarct ventricular tachycardia: the value of pace-mapping during sinus rhythm. Heart Rhythm 2014;11:175—81.
[46] Komatsu Y, Hocini M, Nogami A, Maury P, Peichl P, Iwasaki YK, et al. Catheter ablation of refractory ventricular fibrillation storm after myocardial infarction. J Am Coll Cardiol 2019;139(20):2315—25.
[47] Santangeli P, Rame JE, Birati EY, Marchlinski FE. Management of ventricular arrhythmias in patients with advanced heart failure. J Am Coll Cardiol 2017;69:1842—60.
[48] Tzou WS, Tung R, Frankel DS, Vaseghi M, Bunch TJ, Di Biase L, et al. Ventricular tachycardia ablation in severe heart failure: an International Ventricular Tachycardia Ablation Center Collaboration Analysis. Circ Arrhythm Electrophysiol 2017;2(1) [pii: e004494].
[49] Ostadal P, Mlcek M, Holy F, Horakova S, Kralovec S, Skoda J, et al. Direct comparison of percutaneous circulatory support systems in specific hemodynamic conditions in a porcine model. Circ Arrhythm Electrophysiol 2012;5:1202—6.

[50] Enriquez A, Liang J, Gentile J, Schaller RD, Supple GE, Frankel DS, et al. Outcomes of rescue cardiopulmonary support for periprocedural acute hemodynamic decompensation in patients undergoing catheter ablation of electrical storm. Heart Rhythm 2018;15:75—80.
[51] Nof E, Reichlin T, Enriquez AD, Ng J, Nagashima K, Tokuda M, et al. Impact of general anesthesia on initiation and stability of VT during catheter ablation. Heart Rhythm 2015;12:2213—20.
[52] Mittnacht AJ, Dukkipati S, Mahajan A. Ventricular tachycardia ablation: a comprehensive review for anesthesiologists. Anesth Analg 2015;120:737—48.
[53] Muser D, Liang JJ, Castro SA, Hayashi T, Enriquez A, Troutman GS, et al. Outcomes with prophylactic use of percutaneous left ventricular assist devices in high-risk patients undergoing catheter ablation of scar-related ventricular tachycardia: a propensity-score matched analysis. Heart Rhythm 2018;15:1500—6.
[54] Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. Eur Heart J 2016;37:2129—200.
[55] Auricchio A, Abraham WT. Cardiac resynchronization therapy: current state of the art: cost versus benefit. Circulation 2004;109:300—7.
[56] Konstantino Y, Iakobishvili Z, Arad O, Ben-Gal T, Kusniec J, Mazur A, et al. Urgent cardiac resynchronization therapy in patients with decompensated chronic heart failure receiving inotropic therapy. A case series. Cardiology 2006;106:59—62.
[57] Cowburn PJ, Patel H, Jolliffe RE, Wald RW, Parker JD. Cardiac resynchronization therapy: an option for inotrope-supported patients with end-stage heart failure? Eur J Heart Fail 2005;7:215—7.
[58] Herweg B, Ilercil A, Cutro R, Dewhurst R, Krishnan S, Weston M, et al. Cardiac resynchronization therapy in patients with end-stage inotrope-dependent class IV heart failure. Am J Cardiol 2007;100:90—3.
[59] Rao BH, Kalavakolanu S, Chandrasekar K, Sastry BK, Narasimhan C. Cardiac resynchronization therapy in hemodynamically unstable heart failure patients. Indian Heart J 2007;59:185—7.
[60] Bonanno C, Ometto R, La Vecchia L, Fontanelli A. Acute haemodynamic effects of intra-aortic balloon pump and cardiac resynchronization therapy. J Cardiovasc Med (Hagerstown) 2008;9:719—24.
[61] Keilegavlen H, Nordrehaug JE, Faerestrand S, Fanebust R, Pettersen R, Haaverstad R, et al. Treatment of cardiogenic shock with left ventricular assist device combined with cardiac resynchronization therapy: a case report. J Cardiothorac Surg 2010;5:54.
[62] Pedersen CT, Kay GN, Kalman J, Borggrefe M, Della-Bella P, Dickfeld T, et al. EHRA/HRS/APHRS expert consensus on ventricular arrhythmias. Europace 2014;16:1257—83.