

Cardiac arrhythmias in the emergency settings of acute coronary syndrome and revascularization: an European Heart Rhythm Association (EHRA) consensus document, endorsed by the European Association of Percutaneous Cardiovascular Interventions (EAPCI), and European Acute Cardiovascular Care Association (ACCA)

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Despite major therapeutic advances over the last decades, complex supraventricular and ventricular arrhythmias (VAs), particularly in the emergency setting or during revascularization for acute myocardial infarction (AMI), remain an important clinical problem. Although the incidence of VAs has declined in the hospital phase of acute coronary syndromes (ACS), mainly due to prompt revascularization and optimal medical therapy, still up to 6% patients with ACS develop ventricular tachycardia and/or ventricular fibrillation within the first hours of ACS symptoms. Despite sustained VAs being perceived predictors of worse in-hospital outcomes, specific associations between the type of VAs, arrhythmia timing, applied treatment strategies and long-term prognosis in AMI are vague. Atrial fibrillation (AF) is the most common supraventricular tachyarrhythmia that may be asymptomatic and/or may be associated with rapid haemodynamic deterioration requiring immediate treatment. It is estimated that over 20% AMI patients may have a history of AF, whereas the new-onset arrhythmia may occur in 5% patients with ST elevation myocardial infarction. Importantly, patients who were treated with primary percutaneous coronary intervention for AMI and developed AF have higher rates of adverse events and mortality compared with subjects free of arrhythmia. The scope of this position document is to cover the clinical implications and pharmacological/non-pharmacological management of arrhythmias in emergency presentations and during revascularization. Current evidence for clinical relevance of specific types of VAs complicating AMI in relation to arrhythmia timing has been discussed.

Keywords

Ventricular tachycardia • Ventricular fibrillation • Atrial fibrillation • Acute myocardial infarction • Reperfusion

Table of Contents

Introduction	1604	Relation between location of myocardial infarction, ventricular arrhythmias and outcomes	1604h
Preamble	1604a	Other arrhythmias in emergency presentations and acute revascularization	1604i
Prehospital arrhythmia	1604a	Acute atrial fibrillation	1604i
Stable vs. unstable, non-sustained vs. sustained and monomorphic vs. polymorphic ventricular arrhythmias ...	1604a	Atrial fibrillation and heart failure	1604i
Prehospital ventricular premature beats	1604a	Atrial fibrillation and acute coronary syndromes	1604i
Prehospital monomorphic non-sustained ventricular tachycardia	1604a	Pre-excitation syndromes and other supraventricular arrhythmias	1604j
Prehospital sustained monomorphic ventricular tachycardia	1604b	Summary, recommendations, and areas for future research ..	1604k
Prehospital polymorphic ventricular tachycardia	1604b		
Prehospital ventricular fibrillation	1604b		
Out of hospital cardiac arrest	1604b		
In-hospital arrhythmias	1604c		
Pre-reperfusion ventricular arrhythmias	1604c		
Reperfusion-induced ventricular arrhythmias	1604c		
Early post-reperfusion ventricular arrhythmias (within 48 h)	1604d		
Late post-reperfusion ventricular arrhythmias (>48 h) ...	1604e		
Post-discharge arrhythmia	1604f		
Chronic phase of ischaemic cardiomyopathy	1604f		
Recurrent ischaemia-induced vs. scar-induced ventricular arrhythmias	1604f		
Ventricular arrhythmias in relation to complete revascularisation vs. incomplete revascularisation, i.e. staged procedures (PCI, CABG) or failed/impossible complete revascularization	1604g		
Ventricular arrhythmias during myocardial infarction scar formation—current evidence for optimal timing and therapy choices (pharmacotherapy ± wearable cardioverter-defibrillators ± implantable cardioverter-defibrillators)	1604g		
Treatment of ventricular arrhythmias in the first 48 h of acute myocardial infarction	1604h		
Treatment of ventricular arrhythmias in the 48-h to 40-day period post-myocardial infarction	1604h		

Introduction

Despite major therapy advances over the last decades, management of complex supraventricular and ventricular arrhythmias (VAs), particularly in the emergency setting or during acute revascularization for myocardial infarction (MI), remains a challenge. There are also implications for management, particularly in the setting of out of hospital cardiac arrest (OHCA), and whether aggressive revascularization attempts are justified.

Ventricular arrhythmias, such as ventricular tachycardia (VT) and/or ventricular fibrillation (VF) may occur at any time of MI, beginning from the early minutes of acute infarction till the remote post-MI period. Although the incidence of VAs has declined in the hospital phase of acute coronary syndromes (ACS) mainly due to prompt revascularization and early introduction of optimal medical therapy, the risk of cardiac arrest and sudden cardiac death (SCD) remains increased after MI and it is perceived to be highest in the first 30 days (1.2–2.3%).^{1–5} Published data suggest that sustained VAs are predictors of worse in-hospital outcome in the setting of ACS.^{6–10} However, specific associations between type and timing of VAs and applied treatment strategies, especially coronary revascularization (whether it is very early, late or delayed, complete or incomplete/impossible/not indicated procedure) and long-term prognosis in acute myocardial infarction (AMI) are vague.

Atrial fibrillation (AF) is the most common supraventricular tachyarrhythmia that accounts for 0.5% all emergency visits.¹¹ It may occur at any time of ACS complicating its course. It is assumed that over 20% AMI patients may have a history of AF, whereas the new-onset arrhythmia may occur in 5% patients with ST elevation myocardial infarction (STEMI). Importantly, patients who were treated with primary percutaneous coronary intervention (PCI) for AMI and developed AF have higher rates of adverse events and mortality compared with subjects free of arrhythmia.^{12–14} In addition, recent reports suggest that time of AF occurrence (very early vs. late) in relation to AMI location (anterior vs. non-anterior) may also affect clinical outcomes.^{15–18}

The scope of this position document is to cover the management of arrhythmias in emergency presentations and acute revascularization. Current evidence for clinical relevance of specific types of VAs complicating MI in relation to timing of the occurrence of MI will be discussed. In recognizing these issues with arrhythmias in emergency presentations and acute revascularization, the European Heart Rhythm Association (EHRA) in collaboration with the European Association of Percutaneous Cardiovascular Interventions (EAPCI) and the Acute Cardiovascular Care Association (ACCA), convened a Task Force to review the clinical implications of such arrhythmias, and to emphasize evidence-based approaches for risk stratification and appropriate pharmacological or non-pharmacological treatments, where evidence exists. However, ultimately the decision on management must be made between the healthcare provider and the patient in light of individual factors presented and potential risks and benefits involved.

Preamble

Members of the Task Force were asked to perform a detailed literature review, weigh the strength of evidence for or against a particular treatment or procedure, and include estimates of expected health outcomes where data exist. Patient-specific modifiers, co-morbidities, and issues of patient preference that might influence the choice of particular tests or therapies are considered, as are frequency of follow-up and cost effectiveness. In controversial areas, or with regards to issues without evidence other than usual clinical practice, a consensus was achieved by agreement of the expert panel after thorough discussions. This document was prepared by the Task Force with representation from EHRA, EAPCI, and ACCA. The document was peer-reviewed by official external reviewers representing EHRA, EAPCI, and ACCA.

Consensus statements are evidence-based when possible and derived primarily from published data or determined through consensus opinion where data are not available. However, the current systems of ranking level of evidence have become complicated such that their practical utility can be compromised. We opted for an easier and user-friendly system of ranking using 'coloured hearts' that should allow physicians to easily assess the current status of the evidence and consequent guidance. This EHRA grading of consensus statements does not have separate definitions of the level of evidence. This categorization, used for consensus statements, must not be considered as directly similar to that used for official society guideline recommendations, which apply a classification (Class I–III) and level of evidence (A, B, and C) to recommendations used in official guidelines.

Thus, a green heart indicates a 'should do this' consensus statement or indicated treatment or procedure that is based on at least one randomized trial, or is supported by strong observational

evidence that it is beneficial and effective. A yellow heart indicates general agreement and/or scientific evidence favouring a 'may do this' statement or the usefulness/efficacy of a treatment or procedure. A 'yellow heart' symbol may be supported by randomized trials based on a small number of patients or results which are perhaps not widely applicable. Treatment strategies for which there is scientific evidence of potential harm and should not be used ('do not do this') are indicated by a red heart (Table 1).

Finally, this is a consensus document that includes evidence and expert opinions from several countries. The pharmacologic and non-pharmacologic anti-arrhythmic approaches discussed may, therefore, include drugs that do not have the approval of governmental regulatory agencies in all countries.

Prehospital arrhythmia

Stable vs. unstable, non-sustained vs. sustained, and monomorphic vs. polymorphic ventricular arrhythmias

Up to 6% of patients with ACS still develop VT or VF within the first hours after the onset of symptoms, most often before arriving hospital.¹ There is limited data regarding the impact of pre-reperfusion VA on remote outcomes in patients with STEMI, as the majority of studies excluded subjects with prehospital or pre-procedural arrhythmias.¹⁹

Prehospital ventricular premature beats

Ventricular premature beats (VPBs), which are typically asymptomatic, are common during AMI with a reported incidence as high as 93%.²⁰ The early occurrence of VPBs does not predict short- or long-term mortality, but frequent and/or multiform VPBs that persist more than 48–72 h after a MI may be associated with an increased long-term arrhythmic risk.²¹




On the other hand, it is not clear which VPBs are benign, and which are not. There are a number of reports found repetitive VPBs, but not frequent VPBs alone, were associated with an increased risk among MI patients.^{22–25} However, some studies showed that an increased frequency of VPBs alone may be associated with an increased mortality risk.^{26,27} Because of this uncertainty, and potential drug toxicity of anti-arrhythmic drugs, suppression of VPBs using anti-arrhythmic drugs is usually not recommended in prehospital setting.

Prehospital monomorphic non-sustained ventricular tachycardia

Monomorphic non-sustained VT (NSMVT) is the most common form of prehospital VT, which is easily recognized, and most often managed without difficulty in the prehospital setting. It ranges from 1% to 7%.^{28,29} In the first 24–48 h after an infarction, and it is usually due to abnormal automaticity or triggered activity in the region of ischaemia or infarction.

In patients with asymptomatic NSMVT, suppression with anti-arrhythmic drugs has not been shown to improve outcomes.³⁰ Hence, it is not recommended to treat asymptomatic NSMVT with anti-arrhythmic drugs in prehospital setting. However, in the rare case,

Table 1 Scientific rationale of recommendations

Definitions where related to a treatment or procedure	Consensus statement	Symbol
Scientific evidence that a treatment or procedure is beneficial and effective. Requires at least one randomized trial, or is supported by strong observational evidence and authors' consensus.	Recommended/ indicated	
General agreement and/or scientific evidence favour the usefulness/efficacy of a treatment or procedure. May be supported by randomized trials based on small number of patients or not widely applicable.	May be used or recommended	
Scientific evidence or general agreement not to use or recommend a treatment or procedure.	Should not be used or recommended	

This categorization for our consensus document should not be considered as being directly similar to that used for official society guideline recommendations which apply a classification (I–III) and level of evidence (A, B, and C) to recommendations.

when arrhythmia is frequent and causes haemodynamic compromise, anti-arrhythmic drugs may be useful.

Prehospital sustained monomorphic ventricular tachycardia

Sustained monomorphic VT (SMVT) is less common than NSMVT in prehospital setting. These arrhythmias occur in approximately 2% to 3% of patients with STEMI^{21,31} and less than 1% with a non-ST elevation myocardial infarction (NSTEMI) or unstable angina.³² Sustained monomorphic VT is associated with larger MI size.³¹ It may also be related to previous scar.

Early SMVT is usually associated with higher in-hospital mortality due to cardiac arrest and possibly to exacerbation of ischaemia and extension of the infarct.^{21,31} Whether early SMVT is associated with an increased long-term mortality risk among patients who survive to hospital discharge is unclear.²¹ These patients should be treated based on the recommendations in the current guidelines.¹

Prehospital polymorphic ventricular tachycardia

This is a common rhythm occurring in prehospital cardiopulmonary arrest. It is, however, less well characterized in the out-of-hospital arena. One paper suggests that polymorphic ventricular tachycardia is more common during cardiopulmonary arrest than previously thought, but responds poorly to advanced cardiac life support therapy.³³ These patients also should be treated based on the recommendations in the current guidelines.¹

Prehospital ventricular fibrillation

Ventricular fibrillation is the most frequent mechanism of prehospital SCD. The occurrence of VF among patients with an acute MI, if occurring within the first 48 h, is associated with an increase in early mortality, but little or no increase in mortality at 1–2 years among patients who survive to hospital discharge.^{21,34}

Out of hospital cardiac arrest

The incidence of SCD is estimated at 4.2 per 1000 person-years and declined over time.^{35–38} A quarter of OHCA victims experience cardiac arrest in the setting of STEMI.³⁹ ST elevation in any lead (including aVR), shockable initial rhythm and chest pain before OHCA are

predictors of AMI as cause of cardiac arrest.⁴⁰ These data reinforce the rationale of the integration link between the resuscitation systems and a centre with emergent angiography and primary PCI facilities.

The role of angiography/PCI in those with NSTEMI is more debatable but an important proportion of these patients have acute lesions amenable by PCI,⁴¹ some of them with complete coronary occlusion despite the ECG suggesting NSTEMI. In some metropolitan areas in USA and Europe transportation of the OHCA resuscitated patient to an acute coronary care intervention centre is associated to a better survival.^{42,43} However, because the survival at hospital discharge remains low for OHCA survivors the result of the integrated approach will be an increase in intra-hospital mortality.³⁹ Recent systematic reviews and meta-analysis confirmed that early access to catheterization laboratories is associated with higher functional survival and favourable neurologic outcome.^{44,45}

Prompt recognition and activation of the emergency call, quick bystander-initiated resuscitation, prompt application of automated external defibrillator (AED) and early advanced life support are linked to significant increase in survival after OHCA. Unfortunately, because of the regional discrepancies in resuscitation related care the survival in real life is suboptimal. Bystander AEDs are applied to only 4% of victims; projection studies suggested that a general AED bystander use for OHCA would increase the survival from 9% to 14% (and for witnessed arrest the survival would increase from 16% to 29%).⁴⁶ If there is no trained bystander available the role of the called dispatcher is crucial⁴⁷; the median OHCA recognition is around 74%.⁴⁸ The most AED responsive arrhythmias are pulseless VT and VF. There is a limited place for anti-arrhythmic drug therapy in the setting of OHCA; amiodarone (as first line therapy for adults with refractory VT/VF), lidocaine or nifekalant (as alternative to amiodarone) demonstrated a controversial benefit in increasing hospital admission survival; however, there is no benefit regarding the survival at the hospital discharge.⁴⁹ Long-term survival of OHCA in patients with AMI who underwent PCI in trained centres is better compared with general OHCA population. In patients remained alive after 30 days, there are no differences in further survival compared with MI patients without OHCA.⁵⁰ Out of hospital cardiac arrest seems to be not an independent predictor of mortality in AMI complicated by cardiogenic shock.⁵¹

Assessment of the short-term prognosis for OHCA victims focusing on the neurological outcome is essential in order to identify those who could benefit most from adapted intensive care. Many parameters are to be used in order to increase the accuracy, especially in patients with prolonged coma after resuscitation; these include pre-hospital circumstances, ECG or in-hospital recorded biomarkers and imaging. A resulting clinical score would help the intensive care physician to stratify the patients and to adapt the care, or recognize the cases where aggressive intervention may be futile.⁵²

The traditional intensive care unit used score Apache II has low discriminatory power for OHCA victims.⁵³ The OHCA score⁵⁴ was externally validated, however, it was generated on a small subgroup of patients and has limited specificity.⁵⁵ A recent simple score,⁵⁶ using the cohort of the Target Temperature Management trial, identified 10 independent predictors of poor prognosis from the prehospital circumstances and patients' status at hospital admission; the score with a good discriminatory power [area under curve (AUC) 0.818], comparable with other scores, permits early identification of patients with poor prognosis.

Another earlier study score⁵⁷ with a good accuracy (AUC 0.810), based on a cardiac arrest registry, investigated 21 parameters and showed that age, time to return of spontaneous circulation, amount of adrenaline and shockable rhythm have the predictive value for survival as all 21 variables. The Cardiac Arrest Hospital Prognosis (CAHP)⁵⁵ score was internally (using a registry population) and externally validated and use a simple nomogram identifying three risk groups according to their neurological outcome. The score has specificity close to 100% for the highest risk group (>200 points). This largely used score uses seven objective parameters for stratification: age, arrest setting, shockable rhythm, duration of collapse during basic life support (BLS), time to spontaneous return of circulation (ROSC), pH, and amount of epinephrine used.

A more sophisticated score⁵⁸ used 11 biomarkers in conjunction with clinical variables (Apache II score, arrhythmia history and time to ROSC); three of biomarkers combined with Apache II score and age were determinants of a favourable neurological outcome at hospital discharge (with a ROC AUC of 0.938). However, this score implies a delay in decision and availability of additional tests.

The most recently described clinical score is the NULL-PLEASE score, incorporating multiple adverse resuscitation features (Nonshockable rhythm, Unwitnessed arrest, Long no-flow or Long low-flow period, blood PH <7.2, Lactate >7.0 mmol/L, End-stage chronic kidney disease on dialysis, Age \geq 85 years, Still resuscitation, and Extracardiac cause).⁵⁹ This score has been proposed to help identify patients with OHCA who are unlikely to survive, where unduly aggressive revascularization attempts may be futile. The simple NULL-PLEASE score has been shown to be predictive for early in-hospital outcome of OHCA, with a 3.3-fold greater odds for fatal outcome at the score values of \geq 5, which was present in 88% of non-survivors.⁵⁹

In-hospital arrhythmias

Pre-reperfusion ventricular arrhythmias

Complex VT/VF are relatively common during the early 48 h of AMI and also have prognostic impact. About 6–10% of STEMI patients

develop significant arrhythmias, mainly polymorphic VT, often degenerating into VF during early in-hospital phase, with an incidence higher than NSTEMI.⁶⁰ Pre-reperfusion VAs are more common than reperfusion-induced, early post-reperfusion or late post-reperfusion arrhythmias in STEMI.¹⁶ Haemodynamic instability, cardiogenic shock, left ventricle ejection fraction (LVEF) <40% and the sum of ST-segment deviations (change from isoelectric line expressed in microvolt) in all leads are independent predictors of VT/VF both in STEMI and NSTEMI.⁸

Urgent reperfusion is the most important therapy, as acute ischaemia usually triggers these arrhythmias. Intravenous beta-blockers and/or amiodarone are useful if no contraindications exist. Intravenous amiodarone may cause phlebitis (it is advisable to use a large peripheral vein, avoid administration >24 h and use preferably volumetric pump), arterial hypotension, bradycardia/AV block. Early intravenous beta-blockers must be avoided in case of hypotension, cardiogenic shock, severe bradycardias and may be harmful for inferior infarction especially with right ventricular involvement. Correction of electrolyte imbalances is strongly recommended, while treatment with angiotensin-converting-enzyme inhibitors (ACE-I)/angiotensin II receptor blockers (ARBs) and statins should be started within the first 24 h (ACE-I/ARB in particular with anterior MI, heart failure, LV systolic dysfunction, or diabetes).⁵⁰ Repetitive electrical cardioversion/defibrillation may be necessary. If there is insufficient control, lidocaine may be considered, although no comparative studies are available and adverse effects must be considered; guidelines recommend a careful use of anti-arrhythmic drugs, because of limited evidence for their benefit and a negative effect on early mortality.⁶¹ Transvenous overdrive pacing can be considered (as second choice) for recurrent VAs with haemodynamic intolerance not controlled by amiodarone, beta-blockers or repetitive electrical cardioversion.⁵⁰




The prognostic role of early VT/VF in STEMI is still controversial. Historical data suggested that early VT/VF increase in-hospital and 30-day mortality but not long-term risk.⁶² However, recent studies have called into question this notion. Kosmidou *et al.*⁶ reported that early pre-reperfusion VAs in STEMI patients were indeed associated with increased 3-years rates of all-cause death and stent thrombosis. Moreover, the clinical impact of VAs in STEMI is also dependent on the timing of arrhythmias. Early VT/VF occurring before, during or after reperfusion is burdened with different mortality rates probably because of different arrhythmic mechanisms and different patients' characteristics. Podolecki *et al.*¹⁹ recently demonstrated that long-term mortality after STEMI was predicted by pre-reperfusion VAs [hazard ratio (HR) 2.76] and late-reperfusion VA (HR 3.39), while reperfusion VAs did not affect 5-years outcome.

Non-ST elevation myocardial infarction patients experience early sustained VAs less frequently than STEMI (<2%), but still they present an increase in overall and arrhythmic mortality at 1-year follow-up.⁷ Further studies are required that should clarify which patients are at risk for recurrent VT/VF after discharge and which interventions should be carried out to decrease residual arrhythmic risk (Table 2).

Reperfusion-induced ventricular arrhythmias

Reperfusion induced sustained ventricular tachycardia/fibrillation (VT/VF), defined as an arrhythmia occurring at the time or within the

Table 2 Management of ventricular arrhythmias in the acute phase of MI

Correction of electrolyte imbalances (hypokalaemia and hypomagnesaemia) is recommended in patients with VT and/or VF.		1.63
Intravenous beta-blockers and/or amiodarone treatment is indicated for patients with recurrent polymorphic VT and/or VF unless contraindicated.		64
Electrical cardioversion/defibrillation is the intervention of choice to promptly terminate life-threatening VAs.		65
Prompt and complete (even staged) revascularization is recommended to treat myocardial ischaemia presenting with recurrent VT/VF.		66,67
Intravenous lidocaine can be considered (as second choice) for recurrent VAs with haemodynamic intolerance not controlled by amiodarone, beta-blockers, or repetitive electrical cardioversion.		61
Overdrive pacing should be considered if VT is frequently recurrent despite anti-arrhythmic therapy and cannot be controlled by repetitive electrical cardioversion.		68,69
In hemodynamically unstable patients with refractory VAs a percutaneous LVAD (Impella, TandemHeart, or extracorporeal life support) may be considered.		69,70
In patients with recurrent life-threatening VAs sedation (preferably with benzodiazepines) or general anaesthesia to reduce sympathetic drive should be considered.		60
Early administration of iv beta-blockers at the time of presentation should be considered in haemodynamically stable patients. ^a		69,71
Asymptomatic, non-sustained and hemodynamically well tolerated VAs should not be treated with anti-arrhythmic drugs before reperfusion ('wait and see').		1
Prophylactic treatment with anti-arrhythmic drugs, with the exception of beta-blockers, is not recommended.		72,73

^aIntravenous beta-blockers must be avoided in patients with hypotension, acute heart failure or AV block, or severe bradycardia.

ACE-I, angiotensin-converting-enzyme inhibitors; ARB, angiotensin II receptor blocker; iv, intravenous; LVAD, left ventricular assist device; VA, ventricular arrhythmia; VF, ventricular fibrillation; VT, ventricular tachycardia.

first minutes after restoration of coronary blood flow, is relatively common during primary PCI, as it affects 4–5% of STEMI patients.^{9,74} Actually, accelerated idioventricular rhythm (AIVR) (15–42%) and non-sustained VT (up to 26%) are the most frequent reperfusion arrhythmias, however, due to their benign character no specific anti-arrhythmic therapy is needed.⁷⁵ Several clinical and angiographic parameters have been reported to be associated with increased risk of VAs development. They include: an inferior wall STEMI, especially right coronary artery (RCA)-related infarction, pre-procedural thrombolysis In myocardial infarction (TIMI) flow grade of 0 to 1, Killip class > I at admission, greater total baseline ST deviation, lack of pre-procedural beta-blocker therapy.^{8–10,74}

Acute ischaemia has deleterious impact on the myocardial metabolism causing anaerobic glycolysis leading to acidosis and accelerated potassium efflux from the myocytes.⁷⁶ This cascade of events results in electrolyte imbalance and electrical instability, which facilitate VAs development in the early phase of MI.⁷⁶ Myocardial reperfusion, although beneficial for the ischaemic myocardium, may also lead to abrupt changes in ionic and electrical balance promoting life-threatening VAs.⁷⁷ It is believed, that the Na⁺/Ca²⁺ exchange pump, slowly activating delayed rectifier K⁺ current as well as changes in sarcoplasmic reticulum proteins are the main factors responsible for the proarrhythmic effect of reperfusion.⁷⁷ Reperfusion-induced VAs may arise from abnormalities in impulse initiation (focal/ectopic activity) or impulse propagation (re-entry). Ventricular tachycardia is a predominant type of reperfusion arrhythmias and non-re-entrant mechanisms are responsible for initiation of most VT episodes.⁷⁸ It is also postulated, that some cases of VAs during primary PCI may be provoked by factors associated with the infarct-related artery (IRA), e.g. spasm of the RCA, contrast injection directly into the sinoatrial

node artery, conus branch occlusion or excessive catheter manoeuvres.^{19,79}



The prognostic significance of reperfusion-induced VAs remains still highly questionable. Most authors reported VT/VF occurring during PCI to be associated with significantly, even up to five-fold, increased in-hospital mortality, and 30-day mortality.^{8–10,19} On the contrary, the PAMI trial (The Primary Angioplasty in Myocardial Infarction Trial) did not demonstrate intra-procedural VAs to portend worse in-hospital prognosis.⁷⁴ The impact of reperfusion-induced VAs on long-term outcomes is also controversial, however, according to most reports, this type of arrhythmia seems not to be associated with an increase in long-term mortality.^{10,19,74}

Ventricular arrhythmias occurring during primary PCI should be treated according to general rules of VT/VF management recommended by European Society of Cardiology (ESC) guidelines.^{1,61} In patients with unstable sustained VT or VF electrical cardioversion/defibrillation should be performed.⁶⁵ Early beta-blocker treatment can help prevent recurrent VAs.⁸⁰ Amiodarone administration should be considered to control recurrent haemodynamically relevant VAs.^{81,82} Other anti-arrhythmic drugs (e.g. flecainide, procainamide, and propafenone) are not recommended, as they cause significant slowing of conduction, that in the setting of ACS may result in aggravation of VAs.^{72,83} In haemodynamically unstable patients with refractory VA a percutaneous left ventricular assist device should be considered.⁷⁰

Early post-reperfusion ventricular arrhythmias (within 48 h)

Post-procedural life-threatening VAs occur in 1.6–4.4% of STEMI population within 48 h of symptom onset.^{9,19,84} The incidence of

Table 3 Monitoring and treatment of ventricular arrhythmias in the acute phase of MI

Patients with ACS who present late (e.g. 12 h) from the onset of the symptoms, with incomplete revascularization, or presence of arrhythmogenic substrate (e.g. documented arrhythmias, prior MI, LVEF <40%, known untreated coronary disease) prior to the event, should be considered at increased risk for arrhythmia development during initial evaluation.		61
Close monitoring (continuous ECG) for at least 24 h is recommended in all AMI patients. ^a		61,87
Prompt coronary angiography and complete revascularization when feasible is recommended in patients with recurrent VT and/or VF		66,67
Monitoring for >24 h in patients should be considered for patients with MI at intermediate-to-high risk for cardiac arrhythmias, including those presenting with the following characteristics: haemodynamically unstable, major VAs <24 h, LVEF <40%, failed reperfusion, additional critical coronary stenoses of major vessels or complications related to percutaneous revascularization.		61
Development of late sustained unstable VAs and subsequent cardiac arrest should prompt a re-evaluation of the index revascularization procedure to look for areas of potential incomplete revascularization and residual ischaemia.		1,61,98
Management of refractory VAs and electrical storm include identifying and correcting underlying ischaemia, use of amiodarone, beta-blockers and electrolyte correction as needed, potential device reprogramming in patients who have ICD. Rescue ablation targeting triggers or substrates for VF or (less frequently in this phase) re-entry monomorphic VAs can be needed in refractory cases and performed by experienced electrophysiologists.		1,63,64

^aFurther monitoring is needed in patients with at least one of the following criteria: failed reperfusion, complications related to PCI, haemodynamic instability, presenting major arrhythmias, reduced LVEF (<40%) or additional critical stenosis/es of major coronary artery/ies.

ACS, acute coronary syndrome; AMI, acute myocardial infarction; ICD, implantable cardioverter-defibrillator; LVEF, left ventricle ejection fraction; MI, myocardial infarction; PCI, percutaneous coronary intervention; VA, ventricular arrhythmia; VF, ventricular fibrillation; VT, ventricular tachycardia.

early VAs in STEMI patients is even five-fold increased compared with those with NSTEMI-ACS.⁸⁵ Ventricular fibrillation and/or polymorphic VT are more often triggered by acute ischaemia and therefore may be an indicator of incomplete reperfusion or recurrence of ischaemia after primary PCI (e.g. acute stent thrombosis), whereas monomorphic VT is believed to be most often related to the presence of a pre-existing arrhythmogenic substrate (e.g. myocardial scar).^{31,61,86,87} The incidence of unsuccessful PCI was reported to be four times higher in patients suffering from early VAs compared with VA-free population.⁸⁸ Moreover, early VF is more likely to develop in younger patients (<60 years) presenting with AMI complicated by new-onset AF.⁸⁹ It is also postulated, that some genetic factors might predispose to early VAs in the setting of AMI.^{90,91}

Early VAs are associated with up to six-fold increased in-hospital mortality, whereas long-term prognosis seems not to be significantly affected by VAs occurring within 48 h of AMI.^{19,85,88,89} In a prospective follow-up, cohort study patients with VF in the acute phase of MI had low and very similar incidence of later SCD as compared with VF-free patients during 5-year observation.⁸⁹ In a large non-selected population of STEMI patients treated with PCI no arrhythmia-related death was reported during index hospitalization in patients, who suffered from early VAs and were alive after 48 h.⁸⁸ However, there are also reports suggesting, that prognostic values of VT and VF occurring in the acute phase of MI may be different.^{7,85,92,93} According to a large registry of ACS early VT was independently associated with increased 1-year mortality.⁸⁵ Moreover, early monomorphic VT was reported to be associated with significantly higher incidence of adequate ICD interventions compared with early VF, as well as to be the independent predictor of death during long-term follow-up.⁹²




Pharmacological treatment of early VAs should be the same as for VAs occurring during PCI.^{1,60,61,87} Non-pharmacological management of VAs developing within 48 h of symptom onset is also generally consistent with the management of PCI-related VAs (see below).^{1,60,61,87}

Because of very limited data on the clinical significance of early VT and mostly derived from observational studies, further prospective trials are needed.

Late post-reperfusion ventricular arrhythmias (>48 h until discharge)

The incidence of late VAs, defined as arrhythmias occurring >48 h from hospital admission after MI, has declined in recent decades as the consequence of early revascularization and progress in pharmacological and non-pharmacological therapies.¹ Yet, in a study of 277 patients with invasively managed NSTEMI-ACS—where malignant VAs (e.g. sustained VT, VF) were detected in 7.6% of patients—the median time for their occurrence was 72 h, and 40% of VAs episodes occurred beyond 48 h.⁹⁴ On the other hand, in a study of STEMI patients undergoing primary PCI, the incidence of malignant VAs was similar in magnitude (6.7%), but only 10% of VAs episodes occurred beyond 48 h from the procedure.⁹ Importantly, every type of malignant VAs (e.g. premature ventricular complexes, sustained VT, VF) portends significantly worse in-hospital prognosis, but late VAs are associated with a higher risk of death than early VAs.^{95–97} As such, identifying patients at risk of late VAs is crucially important. ESC guidelines for ACS recommend rhythm monitoring for >24 h in patients with MI at intermediate to high risk for cardiac arrhythmias, including those presenting with the following characteristics: haemodynamically unstable, major VAs (e.g. sustained VF, VT) <24 h, LVEF <40%, failed reperfusion, additional critical or chronically occluded coronary stenoses of major vessels or complications related to percutaneous revascularization.⁸⁷ Patients treated for STEMI or NSTEMI-ACS in the context of multivessel disease should preferentially undergo complete revascularization (CR) during the index hospitalization, particularly if intermediate-to-high risk of cardiac arrhythmias is identified.⁹⁸ The best timing and revascularization modality for patients with myocardial infarction, multivessel disease and

Table 4 Evaluation and management of patients with ischaemia-induced and scar-induced ventricular arrhythmias in chronic phase of ischaemic cardiomyopathy

Coronary angiography and revascularization is recommended in patients with ischaemia-induced VAs i.e. polymorphic VT or VF.		61,87,114
Catheter ablation in specialized and experienced centres is recommended in patients presenting with incessant scar-induced Vas, i.e. monomorphic VT.		115–118
Optimal pharmacological therapy with ACE-I (or, when intolerant, ARBs), beta-blockers and MRAs is recommended in patients with HF with systolic dysfunction (LVEF \leq 35%) and scar-induced VAs.		109–111,118
Oral amiodarone or catheter ablation is recommended in patients with recurrent ICD shocks due to sustained scar-induced VAs.		115–117,119
Oral amiodarone may be considered for relief of symptoms from scar-induced VAs.		30,113,119,120
Amiodarone or catheter ablation should be considered after a first episode of sustained scar-induced VT in patients with an ICD.		113,115–117,119,121,122
Therapy with sodium channel blockers (class IC) is not recommended in patients with scar-induced VAs.		112,123
Prophylactic treatment with anti-arrhythmic drugs other than beta-blockers is not indicated.		72,73,123

ACE-I, angiotensin-converting-enzyme inhibitors; ARB, angiotensin II receptor blocker; HF, heart failure; ICD, implantable cardioverter-defibrillator; LVEF, left ventricle ejection fraction; MRA, mineralocorticoid receptor antagonist; VA, ventricular arrhythmia; VF, ventricular fibrillation; VT, ventricular tachycardia.

intermediate-to-high risk of VAs should be carefully appraised by the local multidisciplinary heart team.⁹⁸

Clinical presentations of late VAs include stable non-sustained VAs, sustained unstable VAs and electrical storm (defined as three or more episodes in any 24-h period), cardiac arrest and SCD. Late recurrent VAs may be indicators of late presentation, incomplete reperfusion, recurrent acute ischaemia, residual ischaemia due to coronary artery disease (CAD) left untreated (e.g. non-culprit lesions) or the presence of pre-existing arrhythmogenic substrates.¹ The role of guideline-directed medical therapy to prevent VAs (e.g. amiodarone, beta-blockers) and electrolyte correction as needed must be emphasized. Some patients may develop electrical storm and/or incessant VAs despite CR and treatment with anti-arrhythmic drugs. These patients are very challenging and can require general anaesthesia or mechanical haemodynamic support and are candidate for urgent catheter ablation of arrhythmic triggers (usually re-entry in monomorphic VAs or focal in polymorphic VAs/VF).¹ In sustained late VAs not triggered by recurrent ischaemia (usually caused by a re-entry mechanism), where functionally CR has been achieved, evaluation for implantable defibrillator is recommended, also in combination with catheter ablation, according to current European guidelines (Table 3).

Post-discharge arrhythmia

Chronic phase of ischaemic cardiomyopathy

Recurrent ischaemia-induced vs. scar-induced ventricular arrhythmias

Very late VAs may occur in the chronic phase after MI when remodelling has taken place.⁹⁹ These VAs may be caused by scar or recurrent ischaemia. In structural heart diseases, re-entry mechanism is responsible for most sustained VAs. Scar with fibrosis after MI creates conduction block and the excitation wavefront might circulate along the border of the scar or through a path within the scar region.^{100,101} Myocardial scar especially the border zone of scar area is

the substrate for re-entrant VAs which appear as monomorphic VT.¹⁰⁰

The mechanisms of recurrent ischaemia-induced VAs are more complex. Transient ischaemia lead to abnormal automaticity in ventricular myocytes and Purkinje fibres by partial depolarization of the membrane potential creating injury currents between ischaemic tissue and healthy myocardium. Re-entry mechanism could also occur during recurrent ischaemia. The excitation wavefront may flow from endocardium with longer action potential durations to epicardium with shorter action potential durations.^{102,103} Ventricular arrhythmias in the setting of ischaemia are more often polymorphic VT or VF than monomorphic VT. Myocardial scar with ischaemia may be stronger predictor of VAs and SCD than each one separately.¹⁰⁴

Left ventricle ejection fraction evaluated in echocardiography is used routinely to assess the risk of post-MI VAs and SCD. Electrophysiological study (EPS) especially in patients with NSMVT due to prior MI and LVEF \leq 40% could demonstrate the presence of a substrate for re-entrant tachyarrhythmia and might be useful to identify patients at risk for VAs and guide therapeutic management.^{1,105,106} The PROTECT ICD study (Programmed Ventricular Stimulation to Risk Stratify for Early Cardioverter-Defibrillator Implantation to Prevent Tachyarrhythmias Following Acute Myocardial Infarction) aims to evaluate the role of EPS-guided ICD implantation, in patients early following MI (first 40 days).¹⁰⁷

The substrate for monomorphic VT could also be identified using signal-averaged electrocardiography (SA-ECG) with late ventricular potentials recording. The use of this technique has been declined over the years but SA-ECG has high negative predictive value (>95%) thus normal signals suggest the lack of a substrate for monomorphic VT. Added to other non-invasive and invasive tests, SA-ECG may be a valuable tool in risk stratification.¹⁰⁸ Cardiac magnetic resonance (CMR) is a promising non-invasive imaging technique. The infarct scar size and surface area measured by CMR have been predictive of inducibility of monomorphic VAs at EPS, and may be a better predictor of the SCD than LVEF⁹⁹ but large-scale trials evaluating ICD implantation guided by CMR beyond LVEF are lacking.

Table 5 Secondary prevention of sudden cardiac death—optimal timing for implantable cardioverter-defibrillator implantation after acute myocardial infarction

<48 h	48 h to 40 days	>40 days
VT/VF related to index MI (acute ischaemia)	VT/VF not related to index MI (if no new/recurrent ischaemia)	VT/VF not related to index MI (if no new/recurrent ischaemia)
ICD not indicated	ICD indicated	ICD indicated

h, hour; ICD, implantable cardioverter-defibrillator; VF, ventricular fibrillation; VT, ventricular tachycardia.

The therapeutic options in scar-induced post-MI VAs, i.e. in monomorphic VT are catheter ablation, anti-tachycardiac surgery on the background of a cardioverter-defibrillator and anti-arrhythmic drug therapy. Guideline-directed optimal medical therapy as a secondary prevention of MI is crucial in preventing cardiac adverse events including VAs and SCD.^{61,98} In patients with HF and reduced LVEF optimal pharmacological therapy with ACE-I (or, when intolerant ARBs), beta-blockers and mineralocorticoid receptor antagonist should be optimized.¹⁰⁹ The role of beta-blockers in reducing mortality in post-MI patients with reduced LVEF have been proven.^{80,110,111} Class IA and IC anti-arrhythmic drugs increased mortality after MI thus should not be used in a pharmacological therapy of scar-induced post-MI VAs.¹¹² Amiodarone may relieve the symptoms of VAs and reduced episodes of arrhythmias but has no favourable effect on survival.¹¹³

Indicating that polymorphic VT/VF should prompt investigation into a potential ischaemia-related mechanism, coronary angiography and revascularization strategies should be implemented in these arrhythmias (Table 4).

Ventricular arrhythmias in relation to complete revascularization vs. incomplete revascularization, i.e. staged procedures (PCI, CABG) or failed/impossible complete revascularization

Complete revascularization is generally defined as revascularization of all coronary lesions not related to the IRA with a stenosis greater than 50% of the vessel in artery diameter of 2 mm or larger. Chronic total occlusion (CTO) is usually defined as the presence of TIMI 0 or 1 flow grade in a coronary artery vessel lasting over 3 months.¹²⁴

Despite advances in the pharmacological treatment, PCI techniques and stent developments, a considerable number of patients experience re-occlusion of IRA or have persistent IRA occlusion after AMI. About 40–50% of patients have multivessel disease (MVD), and approximately 12–13% of patients with acute STEMI have CTO in non-IRA.^{125–128} Multivessel disease and CTO in non-IRA are an independent mortality predictors and are associated with a higher adverse cardiac events rate during long-term follow-up.^{129–131} Recurrent VA may be an indicator for incomplete revascularization (IR) and two non-randomized studies suggested acute coronary angiography in survivors of out-of-hospital cardiac arrest.^{66,67} Nevertheless, the level of evidence according to the guidelines is low (Class I, C).⁶¹ Nonetheless, there is a lack of knowledge in the burden and the impact of malignant VAs on adverse outcome in patients with IR. Although several retrospective studies showed worse outcome in patients with IR vs. CR in the era of drug eluting stents, these

studies did not find any significant difference in the incidence of malignant VAs in the IR group vs CR.^{132,133}

In theory, if ischaemia is a trigger for VAs correction of CAD lesions by revascularization should prevent arrhythmia occurrence. Indeed, studies suggest lower mortality after revascularization in patients with ischaemic moderate to severe left ventricle dysfunction but only in some of them arrhythmia-free survival were observed.^{134–136} In major studies, CR did not prevent VAs and this was observed in patients with depressed as well as with preserved LVEF.^{137–140}

A study in patients with prior MI and mean LVEF 38% ± 9% and VT showed arrhythmia recurrence in more than 50% of the study group during long-term follow-up despite CR.¹³⁸ In another study in patients with CAD, ICD and depressed LVEF there was no reduction of appropriate ICD therapies after CR.¹³⁹ Study performed in patients with prior MI, sustained VAs in the absence of ACS and LVEF > 40% have also showed no influence of coronary revascularization on the recurrence of malignant arrhythmias.¹³⁷ No significant influence of CR vs. IR on the incidence of VAs was observed regardless of the type of revascularization [PCI/coronary artery by-pass grafting (CABG)], comparable occurrence of VAs was observed in patients with VAs before and after surgery revascularization.¹⁴⁰

Thus, the influence of ischaemia in the genesis of sustained VAs in patients in chronic phase of MI is controversial. Ventricular arrhythmias are mainly due to re-entry mechanism around the scar which is not affected by revascularization.¹⁴¹ It seems that the time of reperfusion is crucial for the development of post-infarct VAs. It was shown that delayed reperfusion (>5 h) was associated with a six-fold increase of inducible VT occurrence compared with early reperfusion (≤3 h) independent of LVEF. Delayed reperfusion as well as LVEF became independent predictors of spontaneous VA.¹⁴²

Ventricular arrhythmias during myocardial infarction scar formation—current evidence for optimal timing and therapy choices (use of pharmacotherapy, wearable cardioverter-defibrillators, implantable cardioverter-defibrillators)

Implantable cardioverter-defibrillator has become the mainstay therapy for prevention of SCD in MI survivors who were either resuscitated from cardiac arrest or haemodynamically unstable VAs (secondary prevention), or those who are at increased risk of SCD due to post-MI ventricular dysfunction, heart failure, or both (primary prevention).^{1,61,87,143}

The risk of cardiac arrest and SCD remains increased after MI and it is perceived to be highest in the first 30 days (1.2–2.3%), followed

Table 6 Primary prevention of sudden cardiac death—optimal timing for implantable cardioverter-defibrillator implantation after acute myocardial infarction

<48 h	48 h to 40 days	>40 days
ICD not indicated (regardless of VT/VF presence)	ICD not indicated (if VT/VF occurs despite no new/re-current ischaemia, ICD is indicated as per secondary prevention criteria [Table 5])	ICD indicated for those with: <ul style="list-style-type: none"> • EF \leq30% (NYHA I) • EF \leq35% (NYHA II-III) • expected survival \geq1 year in good functional status
WCD not indicated	WCD not indicated	WCD not indicated

h, hour; ICD, implantable cardioverter-defibrillator; LVEF, left ventricle ejection fraction; NYHA, New York Heart Association class; VF, ventricular fibrillation; VT, ventricular tachycardia; WCD, wearable cardioverter-defibrillator.

by a progressive decline until a plateau after a few months.^{2–5,113,144} Thereafter, the risk of SCD increases again. The MADIT-II (Multicenter Automatic Defibrillator Implantation Trial II) study showed a 31% survival benefit in MI survivors with LVEF $<$ 30%, in whom ICDs were implanted $>$ 1 month after MI (median of 60 months), compared with standard therapy alone.³ The survival benefit from ICD became apparent approximately 9 months after device implantation. Substudies of the MADIT-II trial showed further that the ICD did not confer survival advantage to patients who had experienced AMI in the 18 months before randomization¹⁴⁵ and those which had undergone myocardial revascularization in the 6 months preceding device implantation.¹⁴⁶

Treatment of ventricular arrhythmias in the first 48 h of acute MI

Ventricular arrhythmias that occur within 48 h of acute MI (predominantly VF or polymorphic VT) are perceived to be related to electric instability triggered by acute ischaemia, reperfusion, necrosis, and autonomic changes.⁶² Thus, prompt revascularisation along with optimal medical therapy are key anti-arrhythmic therapies. Despite conflicting data on clinical relevance of early post-MI VAs (see chapters above),^{6,7,19,62} the occurrence of VAs within 48 h of acute MI is presently not considered an indicator for SCD prevention with an ICD or wearable cardioverter-defibrillator (WCD) (Tables 5 and 6).¹

Treatment of ventricular arrhythmias in the 48-h to 40-day period post-myocardial infarction

Despite data showing the highest risk for SCD within the first month of MI, a primary prevention with ICD failed to improve overall survival in two prospective randomized controlled trials (RCTs) as compared with optimal medical therapy alone: the DINAMIT (Defibrillator in Acute Myocardial Infarction Trial)⁴ and the IRIS (Immediate Risk Stratification Improves Survival Trial).⁵ In both RCTs, the reduction of SCDs in the ICD arm was offset by an unexpected increase in non-SCDs, presumably related to heart failure, ischaemia, or both. Other potential reasons for lack of ICD benefit are: only approximately 50% of SCDs post-MI are perceived to be arrhythmic; defibrillation testing (DFT), right ventricular pacing and ICD shocks may have deleterious myocardial effect and thus increase mortality in patients with left ventricle dysfunction, heart failure or both; patient selection bias in RCTs cannot be excluded.^{147,148} Also, the VEST trial (Vest Prevention of Early Sudden Death Trial) failed to

show significant reduction of SCDs in patients who were randomized within 7 days of acute MI to wear a WCD or remain on optimal medical therapy alone.^{149,150} In high-risk post-MI patients with LVEF $<$ 35% (average of 28%) WCDs did not reduce arrhythmic mortality in the 3-month period, yet the SCD occurred in 1.6% of the WCD group vs. 2.4% in the control group ($P = 0.18$).

Thus, in line with current guidelines ICD therapy is not recommended in the first 40 days of acute MI unless secondary prevention criteria are met.^{1,61,87} Following that period patients with initially compromised left ventricle function (LVEF \leq 40%) should be reassessed for primary prevention of SCD, given that initial myocardial stunning releases over time and LVEF improvement is largely complete by 14 days, particularly amongst reperfused patients.¹⁵¹

Relation between location of myocardial infarction, ventricular arrhythmias and outcomes

Clinical outcomes in patients after MI are related to myocardial type and location. In STEMI, the in-hospital mortality is higher than in NSTEMI, whereas long-term prognosis is similar or even worse in NSTEMI.^{152–155} Anterior wall MI has been shown an independent predictor of large infarct size and mortality in patients with STEMI.^{156,157} The extent of peri-infarct and 'border zone' is associated not only with mortality rates but myocardial scar size correlates also with the risk of monomorphic VT.^{158,159} Infarct surface area assessed by CMR is a better predictor of spontaneous VT and inducibility of arrhythmia on EPS than LVEF.^{158,160,161}

The overall occurrence of VAs in patients with anterior and non-anterior infarction is comparable but differs in relation to the timing of arrhythmia. Subjects with non-anterior wall infarction have mainly reperfusion VAs, whereas late post-reperfusion arrhythmias occur more often in patients with anterior wall MI.^{9,19,74} It was also observed that the outcome in patients with VAs is associated with STEMI location.¹⁹ Ventricular arrhythmias in patients after anterior MI are independently associated with increased mortality rates during long-term follow-up.¹⁹ More extensive size of the scar and border area, higher incidence of HF might be the reasons for higher VAs incidence in those groups of patients.

Ventricular tachycardia cycle length is also related to the myocardial scar size and site. Indeed, it is significantly longer (slower VT) in patients after anterior wall infarction than in those after inferior or

Table 7 Intravenous dosing regimen for the drugs indicated for acute rate control in patients with AF with rapid ventricular response in patients with heart failure

Drug	Dosing regimen
Amiodarone	Initial dose: 5 mg/kg in 1 h followed by 50 mg/h
Digoxin	0.25 mg each 2 h up to 1.5 mg

postero-inferior MI (faster VT).^{162,163} Success rates of endocardial VT ablation are higher than in non-ischaemic cardiomyopathy. This is due to the fact that in patients after MI the arrhythmogenic substrate is usually identified within the subendocardial myocardium because infarction commonly proceeds from the endocardial to epicardial stratum. The subepicardial arrhythmogenic substrate is observed mainly in patients with old inferior or postero-inferior scar.^{162,164} Epicardial VT ablation is required in approximately 15% subjects after inferior or postero-inferior MI and rarely in patients after anterior wall infarction.^{162,165} Moreover, in patients requiring epicardial VT ablation the endocardial arrhythmogenic substrate is relatively small.¹⁶²

Other arrhythmias in emergency presentation and acute revascularization

Acute atrial fibrillation

Atrial fibrillation affects 1–2% of the population and 9% of the population older than 80 years.¹⁶⁶ Most AF patients seeking attention in the emergency department are haemodynamically stable. However, rapidly conducted AF can cause haemodynamic instability. Furthermore, AF may occur in the acute setting of another emergency like ACS, acute cardiac decompensation, pneumonia sepsis, and other conditions.

Stroke prevention is a priority in the management of AF patients, and thromboprophylaxis whether oral or intravenously should be a consideration even in the emergency setting.^{167,168} Use of the CHA₂DS₂-VASc and HAS-BLED scores for risk stratification are supported by a recent comprehensive systematic review and evidence appraisal of the published literature, commissioned by the Patient-Centered Outcomes Research Institute (PCORI) to update a 2013 Agency for Healthcare Research and Quality (AHRQ) review.¹⁶⁹



Atrial fibrillation and heart failure

Heart failure and AF often coincide and AF worsens the prognosis in both, heart failure patients with preserved and reduced LVEF.¹⁷⁰

In the acute setting urgent cardioversion is indicated in patients with haemodynamic compromise when AF is thought to be a major contributing factor. This may carry an increased risk of thromboembolism in patients with unknown or inadequate anti-coagulation.

Usually, the first step is to achieve rapid adequate ventricular rate control. In euvo-lae-mic HF patients with mild symptoms initiation of oral beta-blocker therapy may suffice. If the patient is more

Table 8 Management of atrial fibrillation in patients presenting with acute heart failure

Urgent electrical cardioversion is recommended if AF is thought to be contributing to the patient's haemodynamic compromise in order to improve the patient's clinical condition.	 166
For patients in symptomatic heart failure an intravenous bolus of amiodarone should be considered to reduce ventricular rate.	109,166
For patients in symptomatic HF an iv bolus of digoxin may be considered to reduce ventricular rate.	 109,166
In patients presenting with only light HF symptoms beta-blocker, usually orally, are safe and recommended as first line treatment to control ventricular rate.	109,166

AF, atrial fibrillation; HF, heart failure; iv, intravenous.

symptomatic and shows signs of haemodynamic compromise an intravenous bolus of amiodarone or digoxin should be given (Table 7).¹⁰⁹

In some instances of patients with poorly controlled ventricular rate, AF by itself causes a potentially reversible form of heart failure with reduced LVEF, tachycardiomyopathy. The question whether AF is the cause or a sequel of HF can only be answered by restitution of sinus rhythm. In tachycardiomyopathy the LVEF may then recover (Table 8).¹⁷¹

Atrial fibrillation and acute coronary syndromes

In STEMI patients around 9% develop AF during or immediately after PCI. Sinus bradycardia (28%) and sinus tachycardia (22%) are other frequently observed supraventricular arrhythmias.^{60,75}

In a community-based study on 3220 patients with an incident MI AF was present in 9% of the population before the MI.¹⁷² A total of 23% developed AF during follow-up, thereof 7% within 2 days and another 4% within 30 days of infarction. Notably, the development of AF was associated with a significant increase in mortality (HR 3.8). Interestingly, this association was not observed in patients developing AF within 2 days of their MI.

Clinically, most ACS patients tolerate AF well. Most patients developing AF should probably be anti-coagulated, which leads at least temporary to triple therapy with an increased risk of bleeding events.¹⁷³ The topic is controversial since AF may be only a transient arrhythmia in the setting of an acute MI. Patients with a CHA₂DS₂-VASc score of 0 or 1, on the other hand may not need OAC and can be managed with dual anti-platelet therapy. Recent evidence suggests that patients with a first episode of AF during their MI had a risk of 13–24% of developing AF after a median follow-up of 1037 days compares to only 6% of those remaining in sinus rhythm during their MI.¹⁷⁴ The management of such patients has been comprehensively reviewed in the 2018 Joint European consensus document on the management of antithrombotic therapy in AF patients presenting with ACS and/or undergoing percutaneous cardiovascular interventions and in the 2018 EHRA consensus document of on management of arrhythmias in critically ill and post-surgery patients.^{175,176}

Should AF persist, the patients should be anti-coagulated at least 3 weeks prior and 4 weeks after cardioversion¹⁷⁶ with either VKA or NOAC.

Should the rate be poorly controlled beta-blocking agents are the first line medication in this context. In haemodynamically compromised patients intravenous amiodarone or urgent cardioversion should be considered.

Finally, one needs to bear in mind that AF with rapid ventricular response could also lead to a Type II MI. In this case, invasive therapy may not be possible. This decision can only be taken in the clinical context of the individual patient (e.g. known complex CAD).

Pre-excitation syndromes and other supraventricular arrhythmias

Pre-excitation is present when an accessory pathway (AP) conducts in the antegrade direction. Accessory pathways can participate in various arrhythmias. The most common form of AP-associated

tachycardia is orthodromic atrioventricular re-entrant tachycardia (AVRT). Less often the AP acts as the antegrade limb during antidromic AVRT or during reciprocating tachycardia associated with multiple APs. Atrial fibrillation, atrial flutter, and other atrial tachyarrhythmias might also occur in the presence of an AP. Since the AP does not possess decremental conduction properties, rapid conduction via the AP could lead to very fast ventricular response potentially resulting in haemodynamic compromise or degeneration to VF posing patients at risk of SCD.¹⁷⁷ Sometimes this might be the first manifestation of pre-excitation syndromes.

All AP-associated arrhythmias can present as a medical emergency. In cases of antidromic or pre-excited tachycardias or AF

Table 9 Dosing regimens for the drugs indicated for the treatment of patients with pre-excitation syndromes in the emergency setting

Drug	Dosing regimen
Procainamide	Initial dose: 15–18 mg/kg iv over 25–30 min not >50 mg/min (may be repeated to a maximum cumulative dose of 1000 mg) Maintenance dose: 1–4 mg/min iv infusion (until arrhythmia terminates or side effects develop)
Ibutilide	0.875 mg iv administered in 10 min, dose may be repeated after a 10 min waiting period
Propafenone	1.5–2 mg/kg iv administered over 10 min
Flecainide	1.5–2 mg/kg iv administered over 10 min

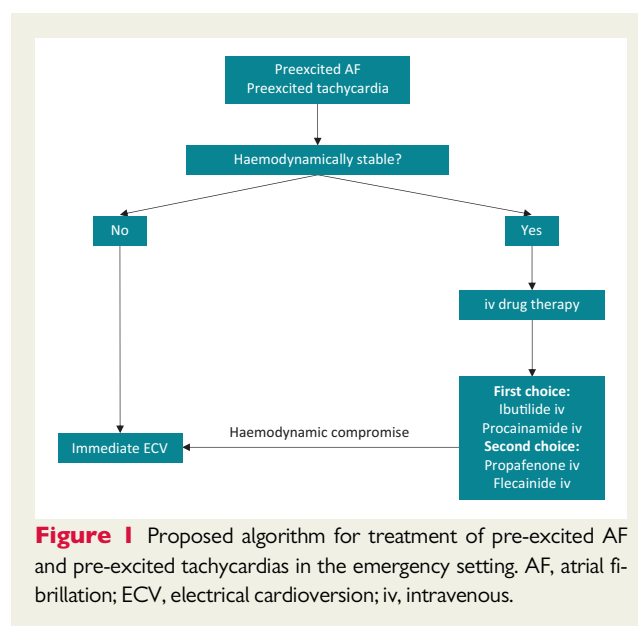


Figure 1 Proposed algorithm for treatment of pre-excited AF and pre-excited tachycardias in the emergency setting. AF, atrial fibrillation; ECV, electrical cardioversion; iv, intravenous.

Table 10 Management of patients with pre-excitation syndromes in the emergency setting

Synchronized electrical cardioversion should be performed in haemodynamically unstable patients with orthodromic AVRT, antidromic AVRT, other AP-associated reciprocating tachycardias, pre-excited tachycardias and pre-excited AF

In stable patients with pre-excited AF ibutilide or iv procainamide administration should be considered.

Both medications act to suppress AP conduction therefore reducing rate of ventricular response. Both medications can also restore sinus rhythm.

Vagal manoeuvre and intravenous adenosine should be used for sinus rhythm restoration in patients with pre-excitation syndromes and narrow QRS tachycardia. The ability to perform immediate electrical cardioversion should be readily available

Intravenous propafenone or flecainide may be administered in haemodynamically stable patients with pre-excited AF to suppress AP conduction and to restore sinus rhythm.

Intravenous beta-blockers, verapamil, and diltiazem may be used in patients with orthodromic AVRT in patients with pre-excitation syndromes to restore sinus rhythm in an emergency setting when other therapeutic options have been ineffective at a facility able to provide immediate electrical cardioversion.

Intravenous amiodarone, digoxin, beta-blockers, verapamil and diltiazem should not be used in an acute setting in patients with pre-excited AF. These medications are potentially harmful in this setting as they can lead to acceleration of ventricular rate. This is most likely due to abolishing competitive concealed AP conduction following AV node conduction slowing, hypotension-induced catecholamine release or shortening of AP refractoriness.

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leading to haemodynamic compromise immediate electrical cardioversion is the only option to restore normal rhythm.^{178,179} Medical therapy is reserved for stable patients with these conditions. It consists of agents which block or suppress AP conduction and/or lead to sinus rhythm restoration. Intravenous ibutilide, procainamide, propafenone and flecainide have been shown to be effective and are most commonly used in this setting^{180–182} (Table 9). Ajmaline may also be considered in these cases.¹⁸³ Intravenous amiodarone has been shown to incur additional risks of increasing ventricular response because of its potential to cause hypotension and subsequent catecholamine release and is therefore considered potentially harmful in pre-excited AF.¹⁸⁴ Atrioventricular node conduction blocking drugs are also considered harmful because of their propensity to enhance conduction via the AP by elimination of competitive concealed AP conduction following AV node conduction slowing or hypotension-induced catecholamine release.^{185,186} Cardiac glycosides have also been shown to shorten AP refractoriness.¹⁸⁷ Recommendations for treatment of pre-excitation syndromes in the emergency setting are shown in Table 10. Figure 1 shows a proposed treatment algorithm.

Summary, recommendations and areas for future research

Both ventricular and supraventricular arrhythmias are common in emergency presentations and in relation to revascularization of ACS. The time of presentation as well as the arrhythmia diagnosis are major determinants of the types of and prognostic impact of arrhythmia. Patients presenting with polymorphic VT or VF should be suspected of having acute myocardial ischaemia whereas monomorphic VT is the typical presentation in patients with older fibrotic infarct areas in the myocardium. Electrical cardioversion is still the most efficient way to convert tachycardia in patients with all types of acute tachycardia and should always be chosen in unstable patients.

Atrial fibrillation is a very common arrhythmia during or immediately after PCI. Up to one in four patients with incident MI will develop AF during follow-up, and the development of AF is associated with significantly increased mortality. The possibility of pre-excited tachycardia should be considered in tachyarrhythmia in emergency situations.

There is a need for large randomized clinical trials to establish new knowledge about optimal treatment and assess the impact of arrhythmia in emergency presentations. Important clinical trials were performed in times where patients were not treated as aggressive as today with respect to revascularization and heart failure. In an ideal world, these RCTs should be repeated on contemporary patient populations with current optimal medical therapy and revascularization strategies.

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In the original version of this article, there were errors in the statement regarding members of the Task Force. This has now been corrected online and in print.

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