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EHRA POSITION PAPER

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)

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Preamble

Critically ill patients are the patients requiring critical care. According to the *American Medical Association (AMA) Current Procedural Terminology document*, delivered in 2017,[1] critical care is the direct delivery by a physician(s) or other qualified health care professional of medical care for a critically ill or critically injured patient. A critical illness or injury acutely impairs one or more vital organ systems such that there is a high probability of imminent or life-threatening deterioration in the patient's condition. Critical care involves high complexity decision-making to assess, manipulate, and support vital system function(s) to treat single or multiple vital organ system failure and/or to prevent further life-threatening deterioration of the patient's condition. In patients who are critically ill, management of arrhythmias implies a series of considerations that are strictly linked to this setting. Decisions on arrhythmia management have to be taken with limited support of evidence and in a clinical context where the risk–benefit ratio is usually much more problematic than in elective conditions.

Consensus guidelines and position papers on management of arrhythmias and devices do not usually include a specific focus on the critically ill patient, a setting where many comorbidities (kidney or hepatic dysfunction, respiratory insufficiency, infections, toxic states, electrolytes derangements, acidosis, etc.) are frequently present, with mutual interactions that may make clinical decision-making very challenging. Similarly, the post-surgery phase corresponds to another setting where transient factors may facilitate the occurrence of arrhythmias, whose management may be complicated by the concurrent treatments typical of the post-operative phase (inotropes, catecholamines, etc.), as well as by the associated high sympathetic tone and the difficulties in managing anticoagulants.

For many arrhythmias occurring for the first time in these acute phases, it may be difficult to assess what can be the risk of recurrences at long-term and whether antithrombotic treatment is needed to counteract arrhythmia-associated thrombo-embolic risk [as in the case of atrial fibrillation (AF)].

Both care of critically ill patients and care of patients in the post-surgery phases involve also management of previously implanted devices, such as pacemakers or implantable cardioverter-defibrillators (ICDs), with frequent need for device reprogramming, according to new arrhythmias onset, need to support the haemodynamics or need to avoid electromagnetic interference due to medical instruments. It is also possible that temporary pacing can be necessary in these settings, for transient bradyarrhythmias. These aspects of devices require to be focused, since no recent documents released by experts in arrhythmia and devices management are available.

Table 1 Scientific rationale of consensus statements

Definitions where related to a treatment or procedure	Consensus statement instruction	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 (as indicated by an asterisk).	'Should do this'	
General agreement and/or scientific evidence favour the usefulness/efficacy of a randomized trials based on a small number of patients or which is not widely applicable.	'May do this'	
Scientific evidence or general agreement not to use or recommend a treatment or procedure.	'Do not do this'	

This categorization for our consensus document should not be considered as being directly similar to that used for official society guideline recommendations.

The aim of this document is to involve experts in the field of arrhythmias and devices, as well as experts in acute critical care in order to focus all the aspects that are specific to these acute settings, highlighting the main drivers of clinical decision-making and providing some advices to be considered for improved patient management.

To address this topic, a Task Force was convened by the European Heart Rhythm Association (EHRA), with representation from the Heart Rhythm Society (HRS), Asia Pacific Heart Rhythm Society (APHRS), Cardiac Arrhythmia Society of Southern Africa (CASSA), and Latin American Heart Rhythm Society (LAHRS) with the remit to comprehensively review the published evidence, and to publish a joint consensus document on the management of arrhythmias and cardiac electrical devices in the critically ill and post-surgery patient.

Evidence review

This document was prepared by the Task Force with representation from EHRA, HRS, APHRS, CASSA, and LAHRS. The document was peer-reviewed by official external reviewers representing EHRA, HRS, APHRS, CASSA, and LAHRS. Consensus statements are evidence-based and derived primarily from published data. In controversial areas, or with respect to issues without evidence other than usual clinical practice, a consensus was achieved by agreement of the expert panel after thorough deliberation.

Differently to guidelines, 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 (*Table 1*).[2] 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.

Thus, a green heart indicates a 'should do this' consensus statement or indicated treatment or procedure that is based on at least one randomized controlled 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 RCTs based on a small number of patients or which is 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.

Diagnosis of arrhythmias in the setting of intensive care units

In intensive care units (ICUs), vital sign parameters including electrocardiogram (ECG), respiration, invasive pressures, and peripheral oxygen saturation are routinely displayed at the bedside and central stations. Physiologic monitor devices also contain arrhythmia computer algorithms that trigger an alarm when a change in cardiac rhythm is detected. An initial critical step is determining if an arrhythmia is truly present. Many ICUs have inpatient telemetry monitoring systems that allow continuous recordings of heart rhythms which allow printing of snapshots for a detailed analysis.

Medical device alarms are sometimes triggered by artefacts resulting from electrical interference created by devices in the patient environment or by motion. False alarms may induce stress in both patients and medical staff.[3] Individual algorithms to accurately classify different life-threatening arrhythmias with the goal of suppressing false alarm generation in ICUs have been developed.[4,5] Careful analysis of all telemetry recordings must be made to distinguish artefact from life-threatening cardiac arrhythmias, specifically polymorphic ventricular tachyarrhythmias. Physicians should correlate the abnormal rhythm with the clinical situation of the patient and carefully look for QRS complexes in the artefact.

Documentation of arrhythmia is advocated in guidelines, for example, the diagnosis of AF requires rhythm documentation using an ECG showing the typical pattern of AF.[6,7] The documented arrhythmia should last sufficiently long for a 12-lead ECG to be recorded, or at least 30 s on a rhythm strip. New-onset AF in ICU patients should be considered of great significance if associated with haemodynamic impairment; anyway it requires specific management and may prolong the duration of hospitalization. The 2014 American Association for Thoracic Surgery Guidelines recommend monitoring with continuous ECG telemetry in patients who are undergoing procedures that pose high (>15% expected incidence of AF) or intermediate (5–15%) risk for AF or who have significant additional risk factors (CHA_2DS_2-VASc ≥ 2) for stroke or have a history of pre-existing or paroxysmal AF before their surgery.[8]

The accurate diagnosis for a wide QRS tachycardia most often need the availability of a 12-lead ECG, searching for atrioventricular (AV) dissociation, capture and fusion beats, and analysing QRS morphology and axis in order to confirm ventricular tachycardia (VT), or alternatively supraventricular tachycardia (SVT) with bundle branch block.[9] Onset and termination of the arrhythmias should possibly be documented by ECG recordings. Anyway, in

Definitions where related to a treatment or procedure	Consensus statement instruction	Symbol	References
If a 12-lead ECG is not available, an ECG rhythm strip should be recorded for at least 30 s in order to define an atrial or ventricular arrhythmias as 'sustained', which may have clinical implications for prognosis and management.	'Should do this'		6,7
A 12-lead ECG is most often needed to establish the definitive diagnosis for a wide QRS tachycardia and should be done unless the haemodynamic instability dictates immediate arrhythmia termination.	'Should do this'		9

case of wide QRS complex tachycardia clinical elements such as history of myocardial infarction and/or heart failure are associated with a very high pre-test probability that the arrhythmia corresponds to a VT.

A sustained VT, by commonly accepted definition, lasts for more than 30 s or requires termination (e.g. cardioversion) in less than 30 s because of haemodynamic compromise, and this definition carries important implications for both management and prognosis, in every specific clinical context [underlying heart disease, left ventricular ejection fraction (LVEF), comorbidities, etc.].

Analysis for a severe bradyarrhythmia should identify its mechanism and distinguish sinus bradycardia/pause or AV block. QRS width may help to identify supra- or infra-Hisian location in case of AV block. Circumstances in case of transient bradyarrhythmia may help to identify an extrinsic vagal origin or facilitation of the episode.

Arrhythmias in the critically ill and post-surgery patient: incidence and risk factors

Acute medical conditions

Sepsis

Arrhythmias are common in the ICU and are more likely to occur in older patients and in patients with severe sepsis or septic shock.[10] There is a clear association between sepsis and arrhythmias.[11] Goldman[12] noted that a major infection was present in 31% and was felt to be the major precipitant in 26% of supraventricular arrhythmias in patients. In another series, the onset of a supraventricular arrhythmias was associated with myocardial infarction in 4% of patients, bacterial pneumonia in 7.5%, and wound infection in 8.5%.[11] Nearly 33% of critically ill patients with sepsis have AF, and 10% have new-onset AF. Sepsis is thus at least as likely as an acute cardiac event or an electrolyte abnormality to be the underlying precipitant of a supraventricular arrhythmia. Arrhythmia patients had a relative risk (RR) of bacterial pneumonia of 7.4 [95% confidence interval (CI) 5.5–9.9] and an increased risk of bacteraemia (RR 6.2; 95% CI 4.0–9.7).[11] Indeed, 20–30% of arrhythmia patients may have underlying sepsis, most often in the lower respiratory tract.[13,14]

In sepsis, a review of the literature found higher rates of prevalence of supraventricular tachyarrhythmias (8–13.6%) rather than ventricular tachyarrhythmias (prevalence around 2%)[15] Management should be directed towards treatment of the underlying cause of sepsis and rate control under expert medical guidance while treating the underlying cause. The arrhythmia often resolves if the underlying cause is promptly identified and treated (medically and/or surgically, according to its specific nature). In case of important haemodynamic decompensation, urgent cardioversion is needed. It is essential for physicians and surgeons to consider, investigate, and diagnose any underlying surgical problem that may have triggered the arrhythmia. Increased awareness and education of junior surgical staff, and early involvement of senior surgical staff in the care of these patients, may avoid detrimental delay.[16]

Acute respiratory insufficiency

Post-operative pulmonary complications (atelectasis, pneumonia, pulmonary oedema, acute respiratory failure, and pulmonary embolism) are common, particularly after abdominal and thoracic surgery, pneumonia and atelectasis being the most common.[17]

Arrhythmias are likely to occur in patients with acute respiratory failure. Ventricular arrhythmias may have poorer prognosis, since they may deteriorate into ventricular fibrillation (VF) or cardiac arrest. The exact causes of these arrhythmias are often uncertain, but metabolic abnormalities associated with respiratory failure are highly suspect. These disturbances can alter the transmembrane action potential of cardiac conducting tissue, causing electrophysiologic phenomena known to trigger arrhythmias.

Until a specific aetiology is confirmed, treatment should focus on identifying and correcting possible causes, such as metabolic abnormalities, congestive heart failure, etc. Cardioversion and antiarrhythmic drugs should be used in life-threatening situations. On the other hand, tachycardia and arrhythmias may also be the cause of cardiogenic pulmonary oedema and acute respiratory insufficiency in post-surgical patients, with the arrhythmia treatment being the principal goal.

In patients with chronic obstructive pulmonary disease (COPD), supraventricular arrhythmias were slightly more common than ventricular arrhythmias, the most frequent being atrial tachycardia and multifocal atrial tachycardia (MAT). Supraventricular arrhythmias tend to recur. Ventricular arrhythmias are often preceded by premature ventricular contractions, supraventricular arrhythmias, or other ventricular arrhythmias. In reports from the 70s, ventricular arrhythmias were found to be associated with a very poor prognosis, with an in-hospital mortality up to 70% for patients with ventricular arrhythmias.[18] In a more recent study, COPD was a significant predictor of the occurrence of VT and death at long-term, also independently of systolic function, and in patients with COPD and VT mortality was around 50% at 8 years.[19] According to these data from literature,

continuous ECG monitoring of patients with chronic airway obstruction who present with acute respiratory failure would be of value in predicting prognosis and identifying patients likely to develop serious arrhythmias possibly worsening prognosis.

Acute kidney insufficiency

The onset of acute kidney injury (AKI) can complicate the clinical course of patients admitted in the ICU and promote the onset of arrhythmias, especially AF. The presence of pre-existing renal dysfunction may also be a risk factor for the occurrence of arrhythmic events in patients who experienced an acute event requiring hospitalization.

The onset of AKI in ICU patients is not rare and is favoured by the presence of pre-existing chronic kidney disease (CKD).[20,21] The incidence of AKI varies depending on the definition used.[22] In critically ill patients, AKI is associated with an increased risk of both early and long-term mortality, about double compared to patients with preserved renal function.[23–25] A large multinational study reported an incidence of AKI of 5.7%, in a population of patients in a setting of critical illness.[20] More recently, a study based on a cohort consisted of 97 ICUs, originating from 33 different countries showed that AKI occurred in 57.3% of patients.[26] Acute kidney injury is particularly common in patients with cardiac surgery. A meta-analysis performed considering 47 cohorts of patients who underwent an aorto-coronary bypass showed a pooled rate of 18.2% of AKI.[24]

The arrhythmia most frequently associated with AKI in the ICU is AF. Acute kidney injury requiring dialysis may constitute a serious complication in patients hospitalized for AF and AF hospitalizations complicated by AKI have quintupled over the last decade in the USA, causing an associated increased risk of mortality.[27] The onset of AF in the surgical ICU and after myocardial revascularization is an independent predictor of AKI.[28,29] In turn, the incidence of AF is almost double in patients who suffered cardiac surgery complicated by AKI (27.7%), compared to those with normal renal function (16.8%).[30] In addition, in patients with acute coronary syndrome treated with primary angioplasty, the onset of contrast-induced nephropathy is an independent predictor of new-onset AF, with a two-fold increase in risk.[31] The presence of pre-existing CKD is itself a risk factor for the onset of new-AF in patients with acute sepsis[32] or after cardiac surgery.[33–35]

Limited data are available on kidney failure and ventricular arrhythmias in acute patients. In a large population of patients hospitalized in the coronary care unit, a close relationship between renal dysfunction and bradyarrhythmia (complete heart block and asystole) has been demonstrated. There were also graded increases in the risk of sustained VT and ventricular fibrillation to worsening renal function.[36] In a population of patients with severe hyperkalaemia requiring hospitalization, 74% had AKI (new-onset AKI or superimposed on CKD), 43% had cardiac arrest, and 35% showed ventricular arrhythmias.[37]

The degree of renal function impairment is of great importance for the dosing of antiarrhythmic agents. As reported in the Supplementary material online, Appendix, the dosage of antiarrhythmic drugs in critically ill patients has to be defined according to renal and hepatic function, according to the specific pharmacokinetic properties of the agent to be administered.

Definitions where related to a treatment or procedure	Consensus statement instruction	Symbol	References
12-lead ECG should be performed in all patients after an acute brain injury.	'Should do this'		38,39
24-h Holter monitoring may be considered in patients with acute brain injury in order to better diagnose transient, paroxysmal arrhythmic events.	'May do this'		38
If any ECG abnormality is recorded on the 12-lead ECG during admission, serial ECGs and telemetry monitoring should be done.	'Should do this'		38,39
In case of prolongation of QTc interval (female QTc > 460 ms; male QTc > 450 ms) in 12-lead ECG during admission, ECG should be monitored during hospital stay.	'Should do this'		46
Use of beta-blockers may be considered among patients after acute brain injury.	'May do this'		38,42
Great caution and appropriate monitoring are required when using antiarrhythmic agents in the presence of drugs that may prolong the QT interval, and discontinuation is recommended if the QT exceeds 500 ms.	'Should do this'		46
Therapy with antiarrhythmic agents is not recommended in patients with a prolonged QT interval before treatment >500 ms or in patients with significant sinoatrial or atrioventricular node dysfunction who do not have a functioning pacemaker as a back up in case of marked bradycardia.	'Do not do this'		46

Arrhythmias among patients with acute brain injury

Changes in ECGs among patients with acute brain damage in the absence of heart disease are common and have been noticed in up to 90% of cases.[38,39] The most frequent ECG abnormalities were prolongation of QT interval, ST elevation or depression and T wave inversion.[40] Cardiac arrhythmias (such as AF, AV block, ventricular ectopy, and ventricular tachyarrhythmia) are less common and have been reported in up to 25% patients with acute brain injury in the absence of heart disease. Among patients with acute stroke and subarachnoid haemorrhage (SAH), the range may be higher up to 28% and 37.5%, respectively.[38]

A combination of catecholamine secretion and Cushing reflex, which is vagal bradycardia reflex due to increased intracranial pressure (ICP), can cause life-threatening arrhythmias.[38,39] The degeneration of the hypothalamus, insular, or brainstem cerebral region during acute brain damage is the most arrhythmogenic.[38,41] Antiadrenergic drugs might be helpful to decrease incidence of cardiac lesion after undue sympathetic activation.[38,42]

Among patients with acute stroke and SAH, AF is the most common arrhythmia.[38,43,44] Prolongation of QT interval (QTc > 470 ms) and decreased heart rate in SAH patients have been found as the independent risk factors for ventricular arrhythmias. In this group of patients the previous therapy with angiotensin receptor blockers or angiotensin-converting enzyme inhibitors significantly decreased the incidence of ventricular arrhythmias.[38,45] In patients after intracranial haemorrhage (ICH), the incidence of cardiac arrhythmias seems to be non-significant.[38]

Arrhythmias among patients with cancer

Cancer is the second leading cause of death in the world. There is a strong correlation between cancer and cardiac arrhythmias, which may be related to previous heart disease or be the result of the tumour or of its complications or of chemotherapies. Many agents used for chemotherapy are known to cause cardiotoxicity, such as anthracyclines or monoclonal antibodies. The incidence of arrhythmia can increase among oncological patients with electrolytic abnormalities, cachexia, and systemic or local inflammation.[47–50] A ESC position document on cardio-oncology has recently been published.[49]

Exposure of the heart during radiation therapy (RT) can potentially lead to heart failure or cause arrhythmias, including supraventricular, ventricular arrhythmias, and AV node dysfunction. New RT technology for breast cancer with reduced doses of radiation to the heart has been associated with lower incidence of arrhythmias.[47,51–55] Patients with cardiac implantable electronic devices (CIEDs) undergoing RT are also at increased risk of CIED dysfunction[56] and a substantial heterogeneity in the professional figures involved in the management of these patients exist, as highlighted by a survey from EHRA.[57] Therefore, it is recommended to follow the specific protocols elaborated by consensus of experts, targeted to a tailored risk assessment for ensuring a safe management of patients with a CIED before, during and after radiotherapy.[58–60] According to these protocols, CIEDs should be periodically interrogated to rule out the presence of malfunctions or spontaneous arrhythmias during the course of radiotherapy (once every week in high risk patients or only once during and after the course of radiotherapy in low and moderate risk patients). If

Definitions where related to a treatment or procedure	Consensus statement instruction	Symbol	References
A 12-lead ECG should be performed in all oncological patients.	'Should do this'		49
The echocardiography should be performed in all oncological patients undergoing potentially cardiotoxic oncotherapy before, during, and after therapy.	'Should do this'		49
New techniques of radiotherapy with limiting area of the irradiation should be preferred to classic radiotherapy.	'Should do this'		58–60
For oncological patients with CIEDs who undergo radiotherapy it is recommended to follow the specific protocols elaborated by consensus of experts, targeted to a tailored risk assessment for ensuring a safe management before, during, and after radiotherapy.	'Should do this'		58–60
In patients with CIED undergoing radiotherapy, the devices should be periodically interrogated (once every week in high risk patients or only once during and after the course of radiotherapy in low and moderate risk patients) to rule out the presence of malfunctions or spontaneous arrhythmias. If remote monitoring of CIEDs is available more frequent interrogations can be programmed and performed manually or automatically.	'Should do this'		60
In case of use of drugs at risk of QTc prolongation, monitoring with frequent 12-lead ECGs should be performed.	'Should do this'		46
24-h Holter monitoring may be considered in all oncological patients in case of symptoms suggestive of arrhythmia.	'May do this'		47–50
In case of prolongation of QTc interval (female QTc > 460 ms; man QTc > 450 ms) drugs which may cause prolongation of QTc are contraindicated.	'Do not do this'		46

remote monitoring of CIEDs is available, more frequent interrogations can be programmed and performed manually or automatically.[60]

Several chemotherapeutic agents and supportive drugs (opioids, antidepressants, antiemetics, antibiotics) can cause the prolongation of QT interval leading to torsade de pointes.[47,49,61] Atrial fibrillation is the most common supraventricular arrhythmia in these patients, for example post-operative AF after lung resection.[62,63] Anthracyclines, melphalan, and Interleukin-2 use may be associated with the development of AF. It may be useful to monitor the patients receiving these drugs, especially in those who had documented ECG abnormalities or arrhythmias during past exposure to a given chemotherapeutic regimen.[64]

Acute surgical conditions (trauma and burns, post-general surgery, post-cardiac surgery)

Arrhythmias in trauma and burn patients

Both trauma and burns can lead to the requirement of fluid resuscitation, catecholaminergic infusions and can be complicated by sepsis and renal failure. The incidence of AF in patients hospitalized with severe trauma or burns is high, reported at 4.3–14.8%[65–67] for patients with trauma admitted to an ICU, and 3.2% of burn patients.[68] These injuries lead to the systemic inflammatory response syndrome,[69] which has been shown to predict development of AF in these patients.[65]

Severity of disease, age, and use of exogenous catecholamine infusions also predicted development of AF, not surprisingly given known arrhythmogenic effects of these drugs. Hypokalaemia is common in trauma patients,[70] felt possibly due to catecholamines. Positive fluid balance also is associated with development of AF.[66] 'Trauma-induced secondary cardiac injury', as defined by elevations in biomarkers, has also been described,[71] which could also contribute to development of AF. Indeed, mortality and length of stay are increased in trauma and burn patients who develop AF.[65–68] While AF is an independent predictor of mortality,[67] they cannot exclude the possibility that AF is a marker for severity of multi-organ failure.[65,66,68]

Treatment of AF specifically in this setting has not been well described. In one study, over 80% of those with new-onset AF resolved spontaneously, most within 1 day.[66] One study showed mortality was lower in trauma patients with AF on beta-blockers.[67] For patients with pre-existing AF who suffer traumatic injury, management of anticoagulation can be challenging. For stable patients, careful monitoring without reversal may be appropriate.[72] If bleeding is life-threatening, warfarin can be reversed with vitamin K and prothrombin complex concentrates containing prothrombin, Factors VII, IX, and X (prothrombin complex concentrates) have been shown to be effective.[73] Of the novel oral anticoagulants (NOACs), only dabigatran, a thrombin inhibitor, has a specific monoclonal antibody antidote.[74] Reversal agents for Factor Xa inhibitors are under development, but, until their availability, administration of prothrombin complex concentrates may be considered.[72]

The incidence of ventricular arrhythmias following general trauma or burns has not been described. The mechanisms mentioned above might also increase vulnerability to ventricular arrhythmias, particularly in those with underlying heart disease. Ventricular arrhythmias in the setting of a blunt chest deceleration trauma, such as a motor vehicle accident, should prompt investigation for myocardial contusion. Contusion, characterized by patchy necrosis and intramural haemorrhage, is caused by a combination of direct pressure on the myocardium and indirect effects due to shear stress from increased intrathoracic pressure.[75] Blunt thoracic trauma carries a 20% risk of blunt cardiac injury, including cardiac rupture, valvular or other injury in addition to contusion, and severe thoracic trauma a 76% risk.[76] Diagnosis can be challenging as abnormal ECG and elevated troponin and echocardiography lack high sensitivity and specificity, magnetic resonance imaging or computed tomography scan can be considered in individual cases.[76] It is recommended that patients with abnormal ECG or positive troponin after blunt chest trauma be monitored for arrhythmia and heart block.[77,78]

Although electrical injuries are commonly benign, they could be life-threatening in case of extensive burns or internal organ damage, with risk of malignant ventricular tachyarrhythmias.[79] A 12-lead ECG is recommended and cardiac monitoring in ICU is recommended for at least 24 h in case of documented arrhythmias, ECG abnormalities or increase in cardiac troponin.

The length of monitoring in case of blunt trauma or electrical injuries is not well established but the trend of cardiac troponin may be a reasonable guide.

Post-general (non-cardiac) surgery

While many arrhythmias are transient and short lasting without altering the recovery phase after non-cardiac surgery, they do have the potential to pose a threat to patient's health, prolong hospital stay, and in a minority of patients are associated with a risk of death.[80]

The reported incidence of arrhythmias following major non-cardiac surgery ranges from 4% to 20%, depending on the type of surgery performed (*Table 2*), the degree of cardiac monitoring undertaken, and the type of arrhythmia studied.[81–86]

The incidence of post-operative AF after non-cardiac surgery varies widely, ranging from 0.3% to 26%[86–91] (*Table 3*). Atrial arrhythmias occur most frequently 2–3 days post-surgery and are considered related to multiple factors, also including sympathetic stimulation associated with an inflammatory response. The incidence of post-operative AF is usually reported to be higher in thoracic surgery vs. other types of surgery, as shown in the *Table 2*. Particular attention has been traditionally dedicated to post-operative AF which is associated with patient age, preoperative heart rate, and male sex.[103–105] The pathophysiology of post-operative AF has to be considered as multifactorial, as shown in *Figure 1*. Apart AF, about 3% of patients develop paroxysmal SVTs while small numbers develop other atrial arrhythmias such as paroxysmal or MATs.[106]

Asymptomatic premature ventricular contractions generally do not require perioperative therapy or further evaluation. Very frequent ventricular ectopy or runs of non-sustained VT may require antiarrhythmic therapy if they are symptomatic or result in haemodynamic compromise[107] Patients with new-onset post-operative complex ventricular arrhythmias, particularly polymorphic VT, should be evaluated for myocardial ischaemia, electrolyte alterations, or drug effects (see 'Haemodynamics and arrhythmias' and 'Acute and mid-term management of arrhythmias' sections).

Bradyarrhythmias that occur in the post-operative period are usually sinus bradycardia secondary to some other cause, such as

Table 2 Factors facilitating the occurrence of post-operative atrial fibrillation

General patient-related factors:
Older age
Male gender
Caucasian ethnicity
Comorbidities: hypertension, obstructive sleep apnoea, pulmonary hypertension, chronic obstructive pulmonary disease, congestive heart failure, ischaemic heart disease, structural or valvular heart disease, and diabetes mellitus
History of arrhythmias with prior surgeries
Prior documented AF, wash out from beta-blockers or antiarrhythmic agents
Withdrawal from alcohol, benzodiazepines, and cocaine
Hyperthyroidism
Uncontrolled pain
Electrolytes imbalance (hypokalaemia and hypomagnesaemia)
Hypoxia
Hypotension
Hypovolemia and dehydration
Hypothermia
Severe anaemia
Acute heart failure/cardiac ischaemia
Infection (sepsis and pneumonia)
Inflammatory response (local and systemic)
Surgery related risk factors:
Type of surgery (incidence in cardiac or thoracic surgery > abdominal surgery or orthopaedic surgery)
Emergency surgery
Surgery for advanced malignant lung cancer
Prolonged duration of surgical intervention
Surgical complications
Post-operative blood transfusion required

AF, atrial fibrillation.

medication, electrolyte or acid–base disturbance, hypoxaemia, or ischaemia. Pain can also heighten vagal tone, leading to sinus bradycardia and even heart block, despite baseline normal conduction. Transient bradycardias and asystole are frequent in the ICU setting and may occur during patient turning or trachea suctioning and is probably due to transiently increased vagal tone. Acutely, bradycardia usually responds to atropine. Persistent symptomatic bradyarrhythmias due to sinus node dysfunction (SND) and AV block will respond to temporary transvenous pacing.[108]

Post-cardiac surgery

Atrial fibrillation is by far the most commonly reported arrhythmia after cardiac surgery followed by ventricular and supraventricular arrhythmias.[109] The estimated incidence of new-onset post-operative AF (POAF) varies between 30% and 50% after cardiac surgery and between 10% and 30% following thoracic surgery[8] which has remained unchanged over the past decades despite improvements in surgical procedures, post-operative care and anaesthesiology. Time of onset is typically 1–5 days after surgery peaking at Day 2.[110]

Although risk factors for POAF overlap with those for AF in general, the most consistently reported risk factor is advanced age[110] and AF incidence increases exponentially past the age of 55 with a five times higher risk in patients aged 72 or older than in patients younger than 55.[110] Other risk factors for POAF relate to the type and complexity of surgical procedures and patient characteristics, being more common after combined coronary artery bypass graft (CABG) and valve surgery (35–60%) than isolated valve (35–40%) or CABG (20–30%) surgery,[110,111] as well as longer aortic cross-clamp time, greater weight, and mitral valve surgery.[111,112] Off-pump CABG and transcatheter valve procedures have lower incidence of POAF in most large studies but are still of great concern.[113] Independent predictors of POAF after isolated CABG include CHA_2DS_2-VASc score, severe obesity, preoperative beta-blocker use, preoperative antiplatelet therapy, and renal failure.[114] The association between POAF and preoperative beta-blockers reported by several studies may be related to the rebound phenomenon occurring after temporary discontinuation of the drug.

The prevailing assumption that AF may occur as an isolated event without recurrence when a secondary precipitant can be associated with the episode and the notion that treatment of such underlying reversible precipitant may 'terminate the arrhythmia without recurrence,' is contradicted by the observed 42%, 56%, and 62% AF recurrence rates 5-, 10-, and 15-years after secondary precipitants such as cardiothoracic surgery, and with similar stroke and mortality risk as in those without secondary precipitants.[115] Post-operative AF adversely affect patient outcomes in terms of morbidity, hospital stay, long-term outcomes, thrombo-embolic stroke, and mortality[110,116–120] (*Table 4*). Post-operative complications including renal failure, wound infection, stroke, and myocardial infarction were significantly higher in patients with POAF than in those without.[122] Inotropic support, use of intra-aortic balloon pump, and ventilation time were also considerably higher in patients with POAF, as were the in-hospital, 30-day, mid-term and long-term mortality rates,[122] which all translate into prolonged ICU time, additional days in the hospital and expanded hospital treatment costs.[110–112] A retrospective propensity matched, multivariable regression analysis comparing 1-year outcomes showed that POAF patients had longer post-operative length of stay (+3.9 days) and higher discharge costs (+$13 993) than no POAF patients, while there was no difference in 1-year quality of life scores.[128]

In a recent meta-analysis of studies on new-onset atrial fibrillation (NOAF) after CABG including 16 studies, with overall 108 711 participants and a median follow-up period of 2.05 years an increased long-term risk of stroke in the presence of NOAF was found (unadjusted studies effect-sizes = 1.36, 95% CI 1.12–1.65; $P = 0.001$, adjusted studies effect-sizes = 1.25, 95% CI 1.09–1.42; $P = 0.001$).[129] The results of this meta-analysis further highlight that the presence of NOAF in patients post-CABG is associated with increased long-term risk of stroke compared with patients without NOAF and should be interpreted also in light of the evidence that patients with NOAF after CABG develop chronic AF at long-term (average 8.5 years) in around 28% of cases, with a five-fold increased risk of chronic AF as compared with patients in sinus rhythm post-CABG operation.[130]

Table 3 Incidence of post-operative AF in non-cardiac surgery

Authors	Type of study	Number of patients	Age	Type of surgery	Incidence of new-onset post-operative AF
Thoracic surgery					
Dyszkiewicz and Skrzypczak[92]	Retrospective	298	NA	Pulmonary resection	8.4% (in pneumonectomy 24%)
Roselli *et al.*[93]	Retrospective	604	64 ± 11	Pulmonary resection	19%
Salvatici *et al.*[94]	Prospective	400	NA	Pulmonary resection	18%
Nojiri *et al.*[95]	Prospective	126	66 ± 9	Pulmonary resection	23%
Imperatori *et al.*[96]	Prospective	454	65.4 ± 8.8	Pulmonary resection	9.9%
Murthy *et al.*[97]	Retrospective	921	67 ± 8	Oesophagostomy	22%
Onaitis *et al.*[98]	Retrospective	13 906	67 median (59–74)	Pulmonary resection	12.6%
Wu *et al.*[99]	Retrospective	10 563	57 ± 12 in non-AF vs. 60 ± 8 in AF ($P < 0.001$)	Lung surgery	3.3% intraoperatively (41% during lymph node dissection)
Non-cardiac non-thoracic surgery					
Goldman[12]	Prospective	916	NA	Non-cardiac surgery	4.0%
Kahn *et al.*[100]	Retrospective	1210	NA	Orthopaedic surgery	4.8%
Brathwaite[101]	Prospective	462	67 ± 18	Non-cardiac surgery	10.2%
Polanczyk *et al.*[11]	Prospective	4181	66 ± 9	Non-cardiac surgery	6.1%
Christians *et al.*[89]	Retrospective	13 696	74	Non-cardiothoracic surgery	0.3%
Batra *et al.*[13]	Prospective	226	74	Colorectal surgery	13.0%
Siu *et al.*[102]	Retrospective	563	67 ± 13	Colectomy	4.4%
Walsh *et al.*[14]	Prospective	51	66.3	Colorectal surgery	26.0%
Bhave *et al.*[90]	Retrospective	370 447	NA	Non-cardiac surgery	3.0%

AF, atrial fibrillation; NA, not available.

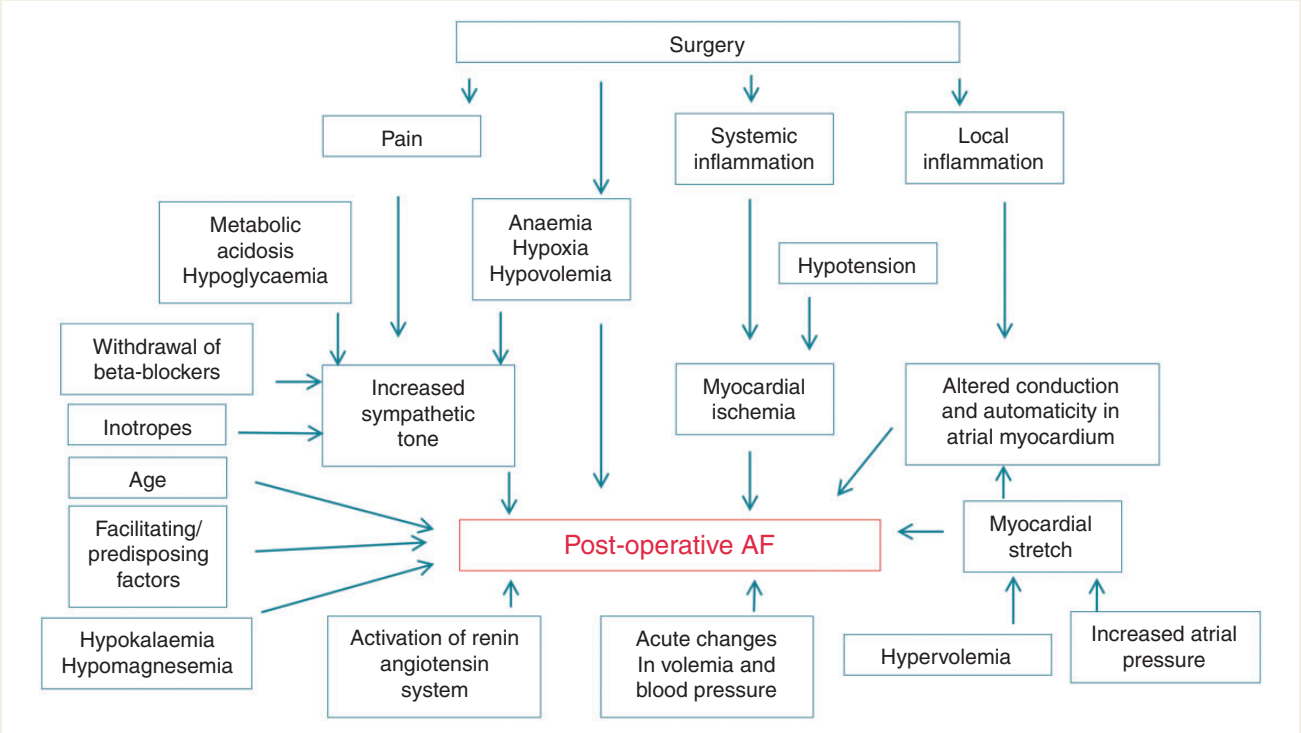

Figure 1 Factors involved in the complex pathophysiology of post-operative AF. AF, atrial fibrillation.

Table 4 Summary of studies including at least 500 patients reporting long-term effects of post-operative atrial fibrillation after various types of cardiac surgeries

Authors	No. of patients	Study type	Cardiac surgery	AF recurs HR/RR	Stroke HR/OR	Mortality HR/OR	FU (years)
Ahlsson *et al.*[121]	5571	Cohort	CABG	/8.31	NA	1.57	6.9
Attaran *et al.*[122]	17 379	Retro	CABG	NA	↑	↑	10
Biviano *et al.*[113]	1879	RCT	TAVR	↑	NS	2.14	1
Bramer *et al.*[116]	856	Cohort	MVS	NA	NA	2.09	3.1
El-Chami *et al.*[123]	16 169	Cohort	CABG	NA	↑	1.21	6
Filardo *et al.*[124]	9268	Cohort	CABG	NA	NA	/2.08	30 days
Girerd *et al.*[125]	2986	Retro	AVS/MVS	NA	NA	1.22/0.87	5.3
Horwich *et al.*[119]	8058	Retro	CABG	NA	1.26	1.2	5.7
Kaw *et al.*[117]	40 112	Meta	CABG	NA	NA	2.19	4
Lee *et al.*[115]	1171	Cohort	CABG	4.99	NA	↑	41 months
Mariscalco *et al.*[118]	9495	Cohort	CABG/VS	NA	NA	1.22/NS	7.9
Melduni *et al.*[120]	603	Cohort	CABG/VS	3.52	NA	3.25[b]	8.3
Swinkels *et al.*[126]	569	Retro	AVS[a]	NS	NS	NS	17.8
Villareal *et al.*[127]	994	Retro	CABG	NA	NA	/3.4	4–5

AF, atrial fibrillation; AVS, aortic valve surgery; CABG, coronary artery bypass graft; FU, follow-up; HR, hazard ratio; MVS, mitral valve surgery; Meta, meta-analysis; NA, not available; No, number; NS, no significant differences between patient groups; OR, odds ratio; RCT, randomized clinical trial; Retro, retrospective study; RR, relative risk; TAVR, transcatheter aortic valve replacement; ↑, increased for patients with post-operative AF.
[a]All patients cardioverted to sinus rhythm before discharge.
[b]Only if late AF.

In the long-term, CABG related POAF has around an eight-fold risk of AF recurrence and a two-fold risk of mortality.[119,121,127,131] Patients with POAF discharged on warfarin after CABG had a 22% relative reduction in long-term mortality compared with those not receiving warfarin.[123] The risk for AF recurrence and stroke is increased both early[122,123,127,132] and on long-term after CABG[119,121,133] in POAF patients. The stroke rates were 12.1% in POAF patients vs. 8.4% in others after mean 5.7 years with a hazard ratio (HR) of 1.26 ($P < 0.0034$).[119] Increased long-term mortality were also observed in POAF patients after both aortic and mitral valve surgery,[116,125] and treatment aimed to restore sinus rhythm before discharge did not affect long-term survival after aortic valve surgery.[126]

In a meta-analysis of randomized trials prophylactic beta-blocking agents reduced the incidence of POAF after CABG from 32.8% to 20%.[134] A similar study reported a 59% reduction of the incidence of POAF after cardiac surgery by Landiolol, a short-acting beta-blocker.[135] Carvedilol was more effective than metoprolol in reducing the POAF incidence in patients undergoing CABG according to other meta-analysis.[136] In a large meta-analysis, amiodarone, beta-blockers, sotalol, magnesium, atrial pacing, and posterior pericardiotomy, all significantly reduced the rate of POAF after cardiac surgery compared with controls with decreased hospital length of stay and treatment cost, and with similar efficacies among beta-blockers while magnesium's efficacy was slightly less.[137] Beta-blockers, sotalol, amiodarone, magnesium, and atrial pacing all reduced the incidence of POAF after cardiac surgery in a similar meta-analysis, although only amiodarone and pacing significantly reduced length of stay and amiodarone alone significantly reduced the stroke rate.[138] Perioperative statin therapy has been found to decrease the risk of POAF and the length of hospital stay in patients undergoing cardiac surgery[139] but a more recent study with rosuvastatin failed to confirm these results.[140] Inhibitors of the renin–angiotensin–aldosterone system have also demonstrated reduced incidence of POAF after cardiac surgery[141] while the positive effects of perioperative polyunsaturated omega-3 fatty acids were weakened by considerable clinical and statistical heterogeneity.[142] Although steroids reduced the risk of POAF with shortened length of ICU stay and hospitalization, they also increased the risk of hypotension in patients undergoing cardiac surgery.[143] Magnesium administration[109,143,144] and vitamin C[145,146] both prevented POAF after CABG according to meta-analysis. Conflicting evidence still exists for preventive effects of colchicine on POAF despite a lower incidence of post-pericardiotomy syndrome and the higher adverse event rates reduce its potential benefits.[147,148] Posterior pericardiotomy effectively prevented POAF in patients undergoing CABG,[149] while smaller studies of thoracic epidural anaesthesia[150] and pulmonary vein isolation[151] failed to demonstrate such efficacy. Based on these data, perioperative beta-blocker therapy is currently recommended and amiodarone may be considered for the prevention of POAF after cardiac surgery.[7]

The use of antiarrhythmic drugs after discharge should be guided by the patients symptoms and AF recurrence rates on long-term.[7] After a recent comparison of rate control and rhythm control for manifest POAF, neither treatment strategy showed a net clinical advantage over the other based on equal numbers of hospitalization days and similar results regarding complication rates of persistent AF at 60 days.[152] The conclusion is however questionable since follow-up was short, and the study failed to evaluate symptoms and quality of life, the most important criteria for treatment. Given the positive effects of amiodarone, a rhythm control strategy is

recommended provided symptoms and AF recurrences are observed.

Some data on POAF are partly contradictory. Future studies may determine whether increased arrhythmia surveillance after cardiac surgery or adherence to general AF management principles in patients with POAF will reduce morbidity and mortality. The lower recommendation class for long-term anticoagulation therapy in patients experiencing POAF when compared to the general AF patients,[7] and the recommendation of cardiology follow-up in other guidelines[8] is questionable. Although it is well recognized that POAF independently predicts complications such as stroke and increased mortality, the lack of consensus regarding best practices for anticoagulation therapy has led to a continuing major variation in practice patterns.[8] It therefore seems logical to apply the same recommendations regarding long-term anticoagulation for POAF patients based on their individual stroke and bleeding risk factors.

Fluid and electrolyte disturbances: risk of arrhythmias and management

This section is given in Supplementary material online.

Haemodynamics and arrhythmias

This section is given in Supplementary material online.

Acute and mid-term management of arrhythmias

Cardiac arrhythmias occurring in critically ill or post-surgery patients may represent a recurrence of previously diagnosed arrhythmia (e.g. paroxysmal AF) or a contingency in the setting of critical illness. Sustained cardiac tachyarrhythmias or significant bradyarrhythmias occur in up to 40% of patients hospitalized in ICU and are associated with significantly increased length of hospitalization and in-hospital mortality rates compared to ICU patients without arrhythmias.[10,11,153,154]

Clinical manifestation may range from asymptomatic arrhythmia to cardiorespiratory arrest. The variety of possible rhythm disorders and multifactorial underlying causes/precipitating factors mandate a systematic and methodical approach in the management of cardiac arrhythmias in critically ill patients, including supportive, diagnostic, therapeutic, and (as needed) resuscitative measures encompassing not only the cause of the arrhythmia but also systemic effects such as impaired end-organ perfusion and function.[155,156] After an arrhythmia is confirmed (i.e. the true existence of cardiac rhythm disorder has been distinguished from an artefact), the urgency of treatment depends on the haemodynamic status. Patients with life-threatening haemodynamic instability (i.e. symptomatic hypotension and/or signs of vital organs hypoperfusion) require immediate electrical cardioversion or defibrillation (*Figure 2*).[155] Once the patient is sufficiently stabilized, identification of correctable underlying causes and treatment of underlying conditions should be undertaken.

The treatment of specific cardiac arrhythmias not requiring immediate advance life support measures, or following successful return of spontaneous circulation, is discussed below.

Atrial fibrillation and atrial flutter

There are limited data on optimal management of AF in critically ill or post-surgery patients,[152,157,158] since the data extrapolated from studies involving non-critically ill AF patients may not apply to these particular situations. High ventricular rates and loss of atrial systole resulting from AF may lead to significant haemodynamic deterioration and symptom worsening. If rapid ventricular response in unstable AF patient is a compensatory response to underlying condition, synchronized direct current (DC) cardioversion is unlikely to provide sustained benefit without concomitant intensified treatment of underlying condition and potentially reversible triggers (*Figure 3*).

In preparation to DC cardioversion, conscious patients should receive sedation, and endotracheal intubation may be needed to prevent aspiration. Anterior–posterior electrode placement and biphasic waveforms provide better success than lateral electrode positioning and monophasic DC shock.[7] In post-thoracic surgery patients a shock of 200 J should be first attempted, because of high impedance and suboptimal electrode placement due to chest tubes and wound dressing.[8] Pre-treatment with amiodarone,[159,160] sotalol,[159] ibutilide,[161] vernakalant,[162] flecainide,[163] or propafenone[164] may facilitate DC cardioversion and reduce immediate recurrences but the experience with the use of these drugs is limited in critically ill, excluding amiodarone (see 'Antiarrhythmic drugs in the critically ill and post-surgery patient: indications, dosages, interactions, adverse effects, proarrhythmia, and risk–benefit ratio' section). Overall, DC cardioversion of AF during critical illness is often unsuccessful, which may be an additional marker of poor prognosis.[165] Reported success rates are as low as 30–37% despite multiple attempts, and recurrences are frequent.[166]

New-onset AF is often a reversible manifestation of critical illness; generally, >50% of AF episodes will spontaneously convert to sinus rhythm within 72 h without specific antiarrhythmic intervention[167] whilst <15% of patients with new-onset AF will leave the ICU in AF.[10,32,168] In patients not requiring emergent DC cardioversion, adequate ventricular rate control and management of the underlying conditions may be a prudent initial approach, whilst pharmacological cardioversion could be attempted if symptoms persist despite adequate rate control, or satisfactory rate control is not achieved.[7,8]

Antiarrhythmic and rate control drugs are discussed in detail in 'Antiarrhythmic drugs in the critically ill and post-surgery patient: indications, dosages, interactions, adverse effects, proarrhythmia, and risk–benefit ratio' section. In general, beta-blockers (if tolerated) are particularly effective for rate control in the increased sympathetic tone setting (the short-acting esmolol is often preferred in the critically ill patient). Amiodarone is effective in rhythm control (and may provide a good rate control until cardioversion) and is a useful agent when beta-blockers or calcium blockers are contraindicated, whilst intravenous magnesium may increase the success of cardioversion in patients in whom amiodarone was ineffective.[158,169–171] Calcium channel blockers (diltiazem or verapamil) may be considered for rate control in patients intolerant of beta-blockers, whilst digoxin may be used as a third-line therapy.

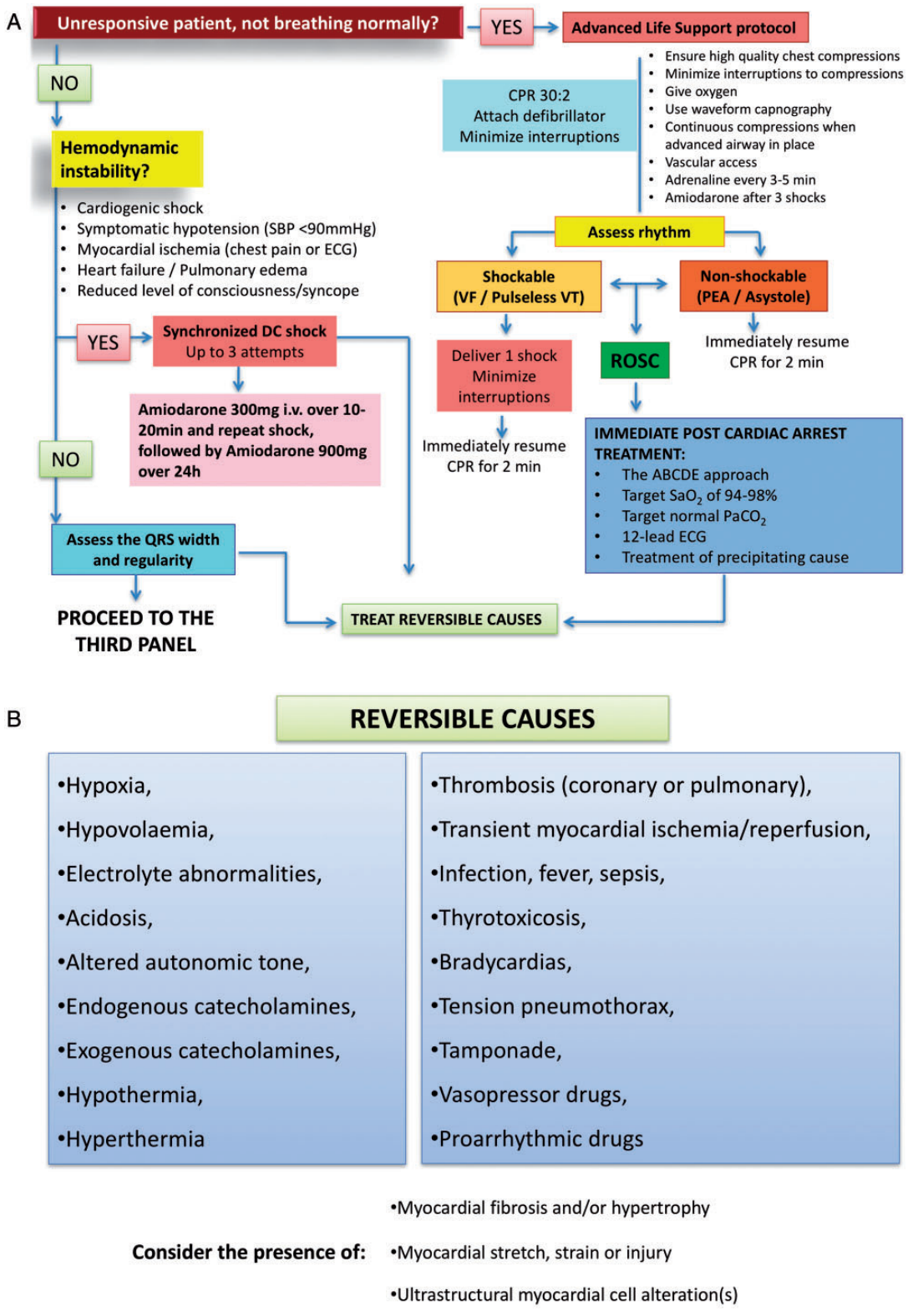

Figure 2 Management of cardiac tachyarrhythmias in critically ill and post-surgery patients (from Monsieurs *et al.*[144]) AF, atrial fibrillation; AFl, atrial flutter; AT, atrial tachycardia; AVNRT, atrioventricular nodal re-entrant tachycardia; AVRT, atrioventricular re-entrant tachycardia; BBB, bundle branch block; CPR, cardio-pulmonary resuscitation; DC, direct current; ECG, electrocardiogram; ICU, intensive care unit; LBBB, left bundle branch block; MAT, multifocal atrial tachycardia; PEA, pulseless electrical activity; RBBB, right bundle branch block; ROSC, return of spontaneous circulation; SBP, systolic blood pressure; ST, sinus tachycardia; VF, ventricular fibrillation; the ABCDE approach: A, airway; B, breathing; C, circulation; D, disability; E, exposure.

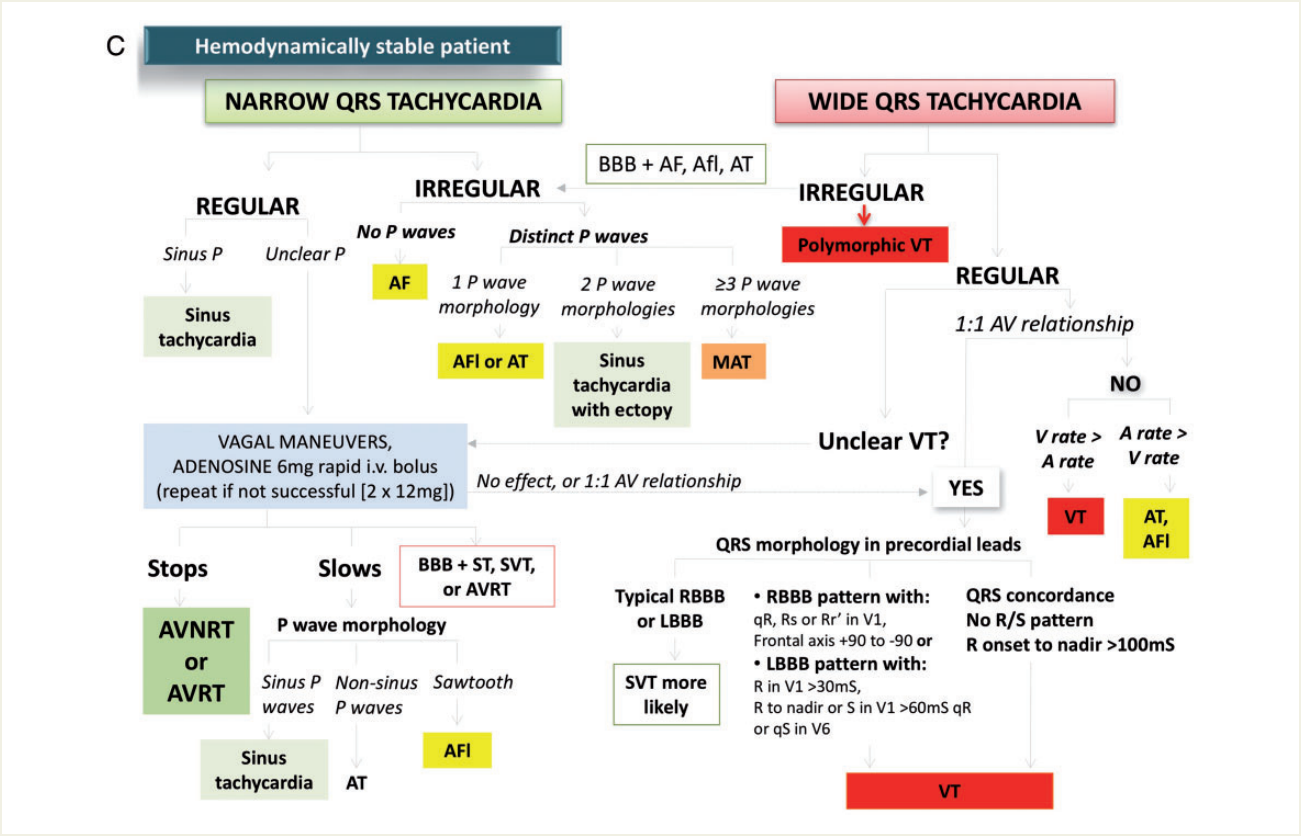

Figure 2 Continued.

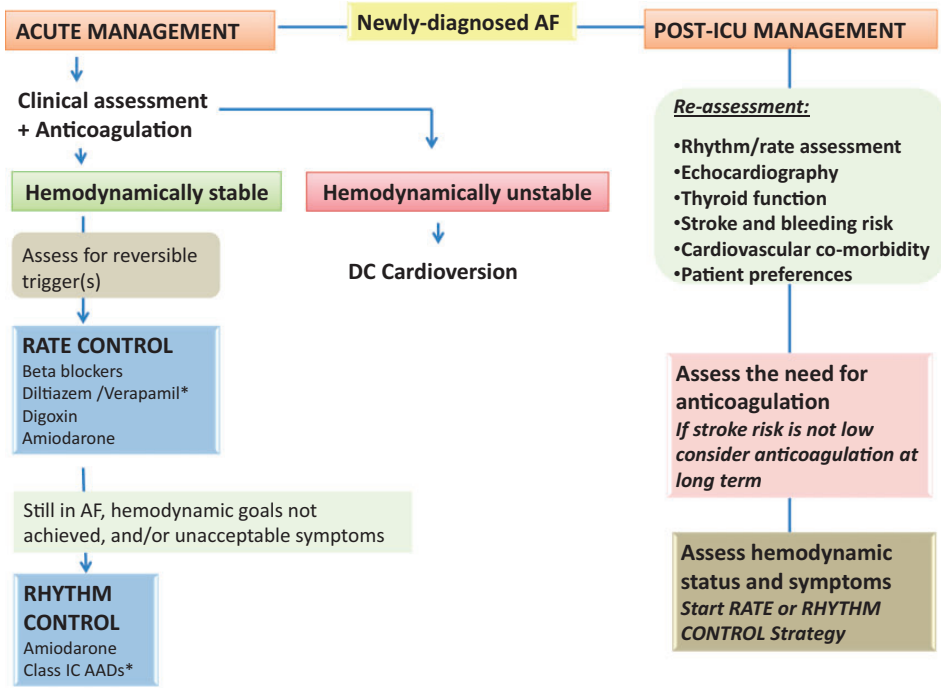

Figure 3 Management of atrial fibrillation in critically ill and post-surgery patients. AADs, antiarrhythmic drugs; AF, atrial fibrillation; ICU, intensive care unit. *Use with caution, not as a first-line therapy.

In post-cardiac surgery patients, post-operatively beta-blockers reduced the incidence of post-operative AF,[134,137] but amiodarone (used either pre- or post-operatively) was superior to beta-blockers and was associated with shorter hospital stay.[138,172,173] Despite positive findings in several meta-analyses,[174,175] a prospective controlled trial of preoperative statin use for the prevention of post-operative AF was negative,[140] and prophylactic effect of post-operative overdrive biatrial pacing has not gained widespread use.[8,137,176] Similarly, amiodarone alone or in combination with magnesium decreased new-onset AF and ICU stay post-non-cardiac surgery.[177–181] However, in a randomized trial of new-onset AF in patients post-myocardial revascularization and/or valve surgery, which compared rate control with target resting ventricular rate of <100 b.p.m. vs. rhythm control using amiodarone (and DC cardioversion, as needed), there was no significant difference in the length of hospitalization or sinus rhythm at discharge (89.9% vs. 93.5%), or at 60 days (84.2% vs. 86.9%).[152]

Whether these findings can be generalized to other critically ill patients is not known and requires further research. Patients with new-onset AF during acute illness often experience recurrent AF and have increased long-term risk of stroke, heart failure, and death.[103,115,182] As long as the patient is haemodynamically stable, the choice between (lenient or strict) rate and rhythm control should be symptom driven (lenient and strict rate control strategies were non-inferior in unselected AF population[183] and those with heart failure[184]).

Once AF is documented (either paroxysmal or non-paroxysmal, symptomatic or 'silent'), thromboprophylaxis must be considered irrespective of whether rate or rhythm control strategy is planned. Critical illness itself may be associated with increased thrombo-embolic and/or bleeding risk, and strokes in medically ill patients are associated with higher in-hospital mortality and less favourable outcome compared to community-onset strokes.[185,186] The prevention of AF-related stroke in critically ill or post-surgery patients may be challenging owing to concomitant thrombocytopenia, frequent multi-organ malfunction, co-medication interacting with anticoagulants, invasive devices, and multiple unscheduled invasive procedures that may substantially increase the risk of severe bleeding. Anticoagulation issues in critically ill and post-surgery patients are discussed in 'Anticoagulation issues in the critically ill and post-surgery patient with cardiac arrhythmias' section.

Acute and mid-term management of atrial flutter (AFl) is broadly similar to the management of AF. However, optimal ventricular rate control may be more difficult, and DC cardioversion may require lower energies compared with AF.[187]

Supraventricular tachycardia

Apart from AF or AFl, SVT is usually a regular narrow-QRS tachycardia (less than 120 ms) in the absence of pre-existing bundle branch block or, more rarely, antidromic reciprocating tachycardia (wide complex tachycardia).[188,189] There are two mechanisms for SVT: (i) increased sinus node automaticity (sinus tachycardia) or atrial tachycardia (AT) and more rarely automatic junctional tachycardia or (ii) re-entry in AV nodal re-entrant tachycardia (AVNRT), permanent junctional reciprocating tachycardia (PJRT) and AV reciprocating tachycardia (AVRT). These arrhythmias may be previously known and sometimes already treated by antiarrhythmic drugs (AADs). Multifocal atrial tachycardia occurs more frequently in patients with chronic obstructive pulmonary disease, coronary artery disease, or chronic heart failure, and may be induced by an acute infection, particularly in elderly patients.[190] It can be mistaken for AF as it is characterized by irregular rates and by a possible lack of P wave activity on ECG (*Figure 2*).[191,192]

Vagal manoeuvres may help differentiate AVNRT and AVRT from AT by stopping AVNRT and AVRT.[193] If vagal manoeuvres fail, intravenous AADs should be administered for arrhythmia termination in haemodynamically stable patients. In narrow-QRS tachycardia adenosine, calcium channel blockers (verapamil), or beta-blocking agents are the drugs of first choice, obviously taking into account the potential for an hypotensive effect. Adenosine has the advantage of its rapidity of onset and short half-life. Longer acting agents (intravenous calcium channel blockers or beta-blocking agents) will be more efficient in patients with recurrences.[194–196] In wide QRS tachycardia (greater than 120 ms), it is important to differentiate SVT from VT (*Figure 2*). If the diagnosis is not possible the tachycardia should be treated as a VT.[188] Flecainide may be used intravenously in AVRT or AT but is contra-indicated in case of low LVEF or hypertrophic cardiomyopathy.[197] Amiodarone may be necessary in case of impaired LVEF or heart failure.[198]

Correction of the precipitating causes should be part of the treatment, as conversion to sinus rhythm may occur after these measures are taken. Direct current cardioversion is indicated in unstable patients. In case of recurrences, ablation will be the solution particularly when tachycardia is not well tolerated and may be sometimes necessary in acute situations.

Ventricular tachyarrhythmias

Ventricular arrhythmias may have devastating consequences, especially in patients with an already stunned myocardium due to extracorporeal circulation during cardiac surgery. Transient impairment of cardiac perfusion, particularly in patients with severe coronary artery disease or cardiomyopathy, may worsen cardiac function and precipitate various tachyarrhythmias. In these patients, correction of precipitating factors, such as ischaemia and electrolyte abnormalities is indicated[199] (*Figure 4*).

Premature ventricular complexes

Premature ventricular complexes (PVC) must be distinguished from atrial ectopy with aberrant ventricular conduction.[200] Isolated post-operative PVCs usually do not exhibit increased risk of malignant ventricular arrhythmias, but frequent PVCs (>30/h) may influence the short-term outcome by reducing ventricular function.[201,202] However, long-term prognosis after surgery is more closely related to the left ventricular (LV) function than to post-operative ventricular arrhythmias.

Asymptomatic and haemodynamically stable PVCs or non-sustained ventricular tachycardia (NSVT) usually do not require specific acute or long-term treatment other than the correction of any precipitating cause. Lidocaine or beta-blockade has been used successfully in reducing haemodynamically significant or symptomatic PVCs, although without detectable effect in mortality.[200,203,204] Patients with preserved LVEF and asymptomatic NSVT after surgery

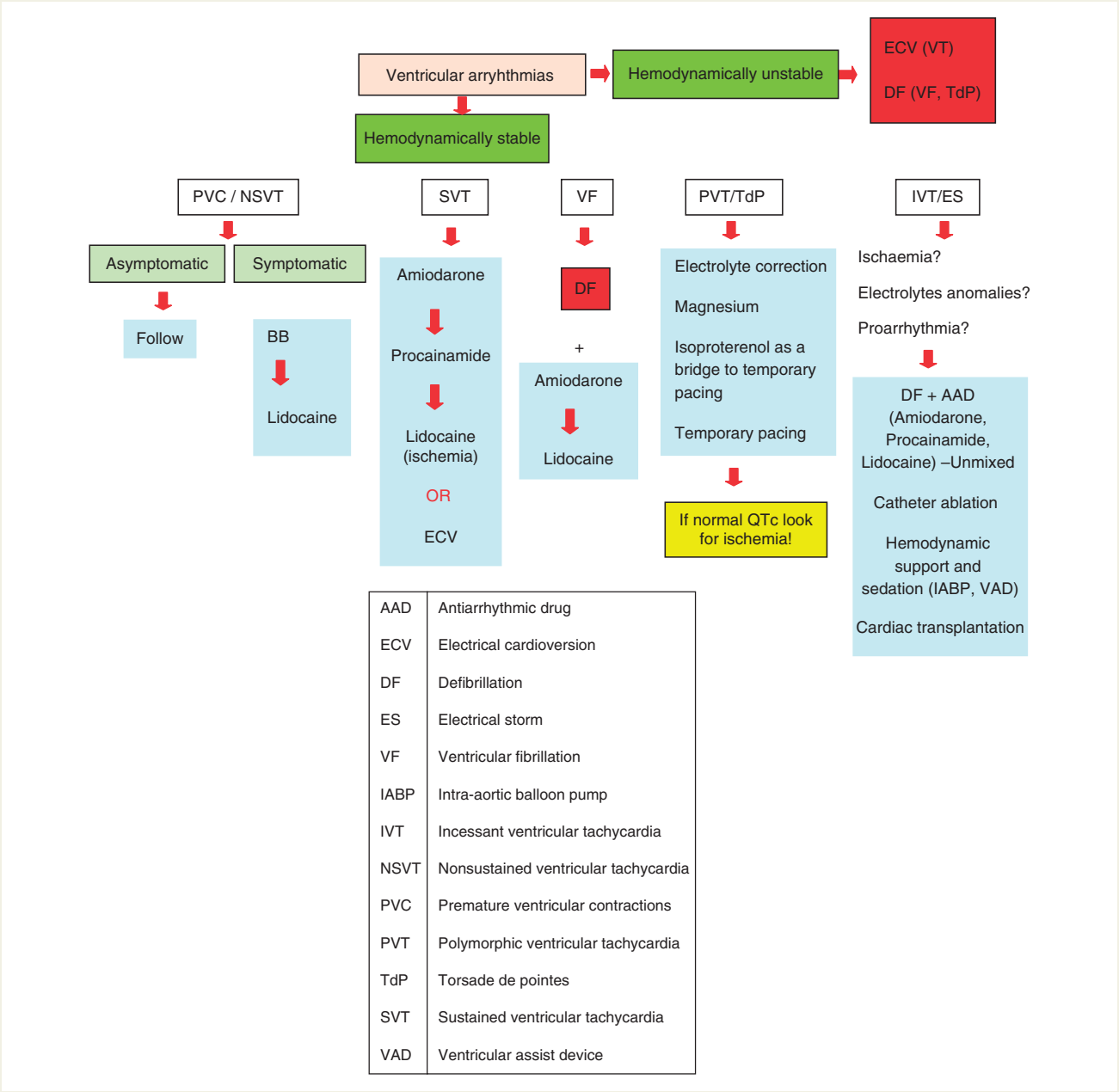

Figure 4 Management of ventricular arrhythmias in critically ill and post-surgery patients. AAD, antiarrhythmic drug; DF, defibrillation; ECV, electrical cardioversion; ES, electrical storm; IABP, intra-aortic balloon pump; IVT, incessant ventricular tachycardia; NSVT, non-sustained ventricular tachycardia; PVC, premature ventricular contractions; PVT, polymorphic ventricular tachycardia; SVT, sustained ventricular tachycardia; TdP, torsade de pointes; VAD, ventricular assist device; VF, ventricular fibrillation.

generally have a favourable long-term prognosis and do not require electrophysiological study. If symptomatic, suppression with beta-blockers can be an option.[205]

Sustained monomorphic ventricular tachycardia

Patients with sustained ventricular arrhythmias have poorer short-and long-term prognosis. The mortality is high, around 50% in hospital and then additionally 10% within 2 years.[205,206] The haemodynamic state of patients with ventricular arrhythmias depends upon the rate of the tachyarrhythmia and LV function. Wide QRS complex tachycardias may be either ventricular or SVT (due to aberrancy, pre-existing bundle branch block, or anterograde pre-excitation). However, in patients with prior infarction, the most frequent diagnosis is VT. If feasible, a 12-lead ECG and atrial electrograms through temporary epicardial wires placed at the time of cardiac surgery may help in the diagnosis looking for ventriculo-atrial dissociation (V > A).

Immediate cardioversion should be performed for haemodynamically unstable VT without pulse. Electrical cardioversion for stable sustained VT can be used either as the first choice or for those who do not respond to antiarrhythmic medications, with 150–200 J in biphasic defibrillators. Sedation with short-acting agents should precede energy delivery in awake patients. Risk stratification before

hospital discharge, including electrophysiological study, may be considered to define the presence of a substrate conditioning an arrhythmic risk independently on reversible causes, in order to make the most appropriate decisions on an ICD implantation[207] or VT ablation in patients already implanted with an ICD.

Haemodynamically stable sustained VTs may be initially treated with intravenous amiodarone (a 300 mg bolus is given over 1 h, followed by infusion at 900 mg/24 h). Amiodarone is often better tolerated in patients with systolic dysfunction than the other antiarrhythmic drugs (see 'Antiarrhythmic drugs in the critically ill and post-surgery patient: indications, dosages, interactions, adverse effects, proarrhythmia, and risk–benefit ratio' section). Lidocaine is less effective if there is no acute ischaemia.[84] In patients with slower VT who are still retaining ventricular epicardial wires, overdrive pacing may be performed and may be useful in preventing VT by suppressing ventricular ectopy. Electrical cardioversion/defibrillation should be easily available, because acceleration of VT and degeneration to VF are possible.[208]

Ventricular fibrillation

Although immediate defibrillation is needed, The Arrest and Alive trials[209,210] support the notion that amiodarone should be considered as the first-line AAD, and it should be given to patients with VF/pulseless VT refractory after 2–3 shocks plus adequate cardio-pulmonary resuscitation and use of vasopressor. Lidocaine may be used if amiodarone is not available.[211] Amiodarone has non-competitive alpha- and beta-blocking effects, so that rapid i.v. loading may exacerbate haemodynamic instability during the initial (rapid) loading phase in patients with severe LV dysfunction.[84,212,213] In these cases, additional vasopressor drugs may be necessary, and occasionally intra-aortic balloon counterpulsation, extracorporeal membrane oxygenation (ECMO) or left ventricular assist device (LVAD).[212,214]

Long-term management of patients presenting with VF requires a full cardiological evaluation in order to assess if transient factors favouring VF onset are really the cause of the arrhythmia and if they can be corrected and prevented, in order to take an appropriate decision on the need for ICD implantation,[46] as detailed in 'Patient follow-up, risk of arrhythmia recurrences, and clinical decision-making at long-term' section. The severity of patient conditions and expected survival should obviously be considered in such decision-making, which may be particularly challenging.

Polymorphic ventricular tachycardia-torsades de pointes

Torsade de pointes (TdP) is a syndrome consisting of polymorphic VT associated with QT prolongation. Acquired QT prolongation can result from drugs (most notably Type IA or Type III antiarrhythmic agents, tricyclic antidepressants, non-sedating antihistamines, erythromycin, and antifungal agents), electrolyte abnormalities (hypomagnesaemia and hypokalaemia), and other conditions as hypothyroidism and cerebrovascular accident. The management of TdP differs markedly from other forms of VT. Stopping of all QT prolonging drugs and correcting of electrolytes are crucial. These patients may also benefit from use of isoproterenol. Temporary pacing to increase the ventricular rate is also frequently needed.[207] Polymorphic VT with normal QT interval usually occurs in the setting of acute ischaemia and/or severe left ventricular dysfunction.[10,153] The rhythm easily degenerates into VF.

Incessant ventricular tachycardia and electrical storm

Ventricular tachycardia that repeatedly recurs and persists more than 12 h despite repeated attempts to terminate the arrhythmia is designed 'incessant'. Recurrence for >3 times/24 h of sustained VT requiring intervention is referred to as electrical storm.[215]

It is important to exclude ongoing myocardial ischaemia and to correct reversible causes. Proarrhythmia has to be considered if the VT became slower and incessant after antiarrhythmic drugs. Its treatment is maintaining haemodynamic support until drug is excreted and avoiding combination of antiarrhythmic drugs. Intra-aortic balloon counter pulsation can be helpful for haemodynamic support and sedation or general anaesthesia quiets episodes and restores stability in some cases. ICD is not indicated for acute management of patients with electrical storms. Catheter ablation is a relevant option in incessant monomorphic VT and can be life-saving. The location of some circuits deep to the endocardium or in the epicardium are main causes for failure of endocardial ablation. For patients with incessant VT, remaining options then include arrhythmia surgery, placement of a ventricular assist device or cardiac transplantation.

Sinus node dysfunction

Sinus node dysfunction (SND) most commonly occurs secondary to senescence of the sinoatrial node and surrounding myocardium. Medications such as beta-blockers, non-dihydropyridine Ca-channel blockers (i.e. verapamil or diltiazem), digoxin, antiarrhythmic drugs, ivabradine, acetylcholinesterase inhibitors, parasympathomimetic agents, sympatholytic drugs, etc. may unmask subclinical SND. Other conditions that may cause SND include hypothermia, hypoxia, hypothyroidism, muscular dystrophy, inflammation, cardiac surgery for congenital or acquired heart disease causing surgical trauma to the sinus node and/or sinus node artery, central nervous system disease associated with increased intracranial pressure and increased parasympathetic tone, electrolyte imbalance (hypokalaemia, hypocalcaemia), etc.[216,217] Correction of extrinsic causes is essential to the management of critically ill patients with SND. Medications affecting sinus node function should be stopped if possible. If needed, SND can be acutely managed using atropine (0.04 mg/kg intravenously every 2–4 h) and/or isoproterenol (0.05–0.5 mcg/kg/min intravenously), but sometimes a transvenous temporary pacemaker may be required despite medical therapy.[216,217]

In patients with SND manifesting as tachy-bradyarrhythmias, the tachyarrhythmias can be treated with digoxin, beta-blockers, or other antiarrhythmic drugs, but such patients should be closely monitored for the exacerbation of bradyarrhythmias and occurrence of symptoms such as dizziness, syncope, congestive heart failure, etc.. In case of symptom or arrhythmias aggravation, permanent pacemaker therapy may be required.[218]

Atrioventricular block

Atrioventricular block may occur in different critical situations with possible temporary issues for pacemaker (PM) implantation (*Figure 5*). These may be an active infection, a critical haemodynamic status, bleeding, or anticoagulation concerns or a difficult choice between a

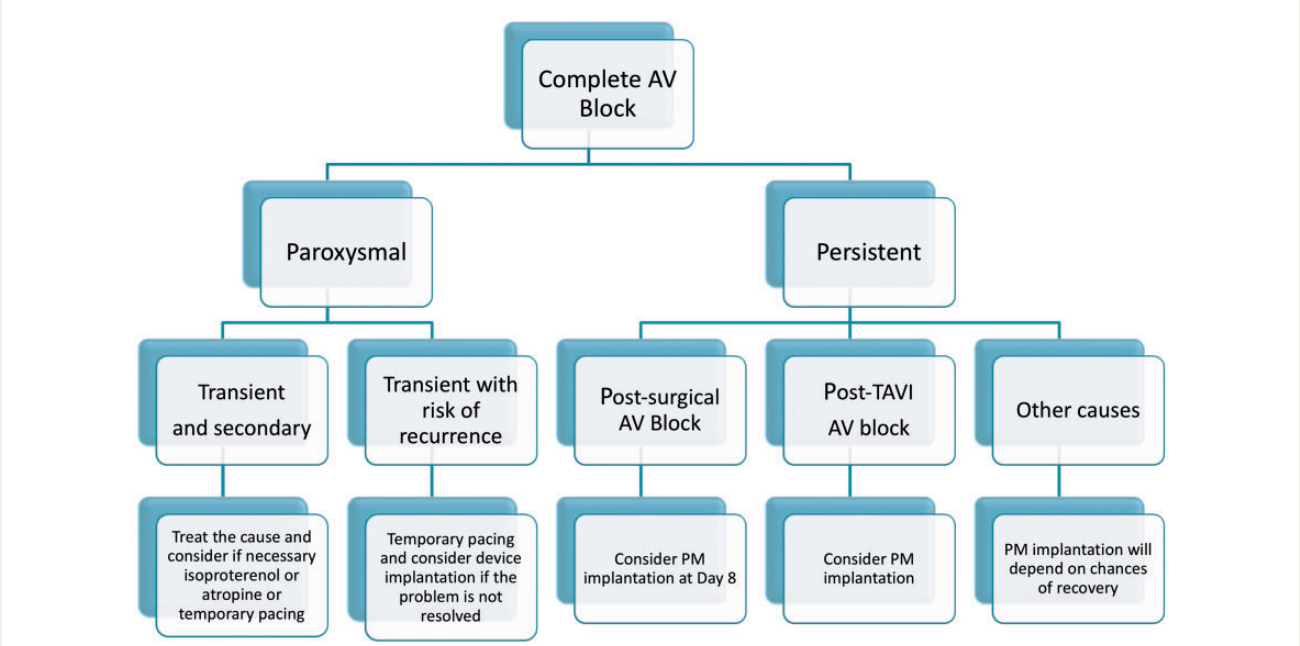

Figure 5 Management of AV block in critically ill and post-surgery patients. AV, atrioventricular; PM, pacemaker; TAVI, transcatheter aortic valve implantation.

VVI(R), DDD(R) or cardiac resynchronisation therapy (CRT) devices (PM or ICD) according to a possible underlying heart disease.[219] In these cases, a precise medical history, and an echocardiographic evaluation are required before taking the optimal decision. Some situations result in a reversible AV block where permanent cardiac pacing is not indicated,[206,220–222] such as acute inferior myocardial infarction, an adverse event of AADs including β-blockers, calcium channel blockers or lithium, or a consequence of the patient medical status (e.g. hypervagotonic status related to emesis, endotracheal suctioning, or endoscopy).

While waiting for the decision of implanting the device, some measures should be taken to prevent prolonged asystole, TdP, or heart failure related to bradycardia. Atropine administration (in case of supra-Hisian block) or isoproterenol infusion may improve AV conduction. Temporary transcutaneous or transvenous pacing may be indicated in haemodynamically unstable patients or when asystole is symptomatic or life-threatening. When maintaining AV synchrony is necessary in patients with impaired LV function, temporary dual chamber pacing may be preferred. The use of jugular or subclavian veins should be preserved for device implantation, and therefore other routes should be preferred. When the use of jugular or subclavian veins is impossible or contraindicated (occlusion or infection), intracavitary leadless PM implantation from a femoral vein may be an alternative.

Two situations are particularly recurrent in the ICU: (i) AV block following cardiac surgery (valve surgery and CABG) as a consequence of direct injury of septal conduction tissue and oedema.[222–227] Permanent pacing may be indicated in 2–4% of patients and more frequently (7.7%) after repeated valve surgery. Placement of an epicardial LV lead should be considered at the time of surgery in order to facilitate resynchronization therapy if needed in patients with high risk of AV block and low LVEF in order to use it when a permanent PM is indicated; and (ii) more recently, transcatheter aortic valve replacement (TAVR) has emerged as a new aetiology of AV block that is frequently transient, leading to the difficulty of determining when to implant PM and in which patient.[228,229] Most of patients are octogenarians with comorbidities, and AV block may necessitate CRT implantation in case of low LVEF.

Antiarrhythmic drugs in the critically ill and post-surgery patient: indications, dosages, interactions, adverse effects, proarrhythmia, and risk–benefit ratio

This section is given in Supplementary material online.

Anticoagulation issues in the critically ill and post-surgery patient with cardiac arrhythmias

Patients who are critically ill and post-operatively are predisposed to cardiac arrhythmias, the most common of which is AF.[230] The development of AF in patients who are critically ill, with (e.g.) sepsis, or in the ICU patients indicates a poor prognosis, with an associated increase in

adverse outcomes.[182,231] For example, Walkey et al.[182] showed that most sepsis survivors with new-onset AF during sepsis have AF occurring after discharge from the sepsis hospitalization and have increased long-term risks of heart failure, ischaemic stroke, and death.

The appropriate management in such patients is complex as such patients have not been studied in large randomized trials and decision-making would need to factor the following:

- Disease severity
- Life expectancy
- Comorbidities, including bleeding tendency or predisposition, renal function, etc.
- Intrinsic prothrombotic state, including immobility, underlying pathology such as cancer, post-surgery state, etc.
- Additional interventions, such as central lines for infusions (often multiple), intravascular haemodynamic monitors, temporary pacing, arterial lines for blood pressure monitors and blood gas sampling, etc.

Limited data from observational cohorts (see *Table 5*) have reported how stroke risk scores ($CHADS_2$, CHA_2DS_2-VASc) are modestly predictive of thromboembolism in critically ill patients with atrial arrhythmias.[234] There have also been various cohorts that have focused on AF in relation to sepsis or in the ICU setting. Various strategies for the assessment and long-term management of patients with new-onset AF during critical illness has recently been reviewed, with the prevention of thromboembolism only being part of the holistic approach.[157]

The serious implications of AF in the critically ill can be inferred from various observational cohort studies (*Table 5*). In a retrospective hospitalized cohort study, Walkey et al.[32] showed new-onset AF was common (5.9%) in patients hospitalized with sepsis (compared to 0.65% of those without sepsis, OR 6.82, 95% CI 6.54–7.11) and was related to an increased risk of in-hospital stroke (2.7-fold) and mortality. The mere development of AF during sepsis in the critical ill (or any other acute illness) indicates that such patients would have a propensity to develop incident AF.

Amongst cancer patients (*Table 5*), oral anticoagulation (OAC) significantly reduced the risk of thromboembolism, although not all cohorts were focused on AF *per se* (where data were limited), but on venous thromboembolism. An European Society of Cardiology position paper on cancer treatments and cardiovascular toxicity has recently been published, addressing some of the management aspects.[49]

Anticoagulation management

The approach to such patients for anticoagulation follows the general approach to thromboprophylaxis for AF (*Figure 3*). The initial step is to assess stroke/thrombo-embolic and bleeding risks, using the CHA_2DS_2VASc and HAS-BLED scores. In general, low risk patients (CHA_2DS_2VASc 0 in males, 1 in females) do not need long-term antithrombotic therapy for stroke prevention—but such patients would be rare in the critical care arena, and the immobility (or other comorbidities) leading to a prothrombotic state may necessitate the administration of parenteral anticoagulation (e.g. subcutaneous heparin). However, heparin may not impact on ischaemic stroke in the short-term, but could potentially result in bleeding depending on associated comorbidities.[233]

After the initial exclusion of low risk patients, long-term stroke prevention should be considered in AF patients with ≥1

Definitions where related to a treatment or procedure	Consensus statement instruction	Symbol	References
For stable patients with POAF of 48-h duration or longer, anticoagulation [with warfarin aimed at an INR 2.0 to 3.0, or a novel oral anticoagulant (NOAC)] is recommended for at least 3 weeks prior to and 4 weeks after cardioversion, regardless of the method (electrical or pharmacological) used to restore sinus rhythm.	'Should do this'		7,241
Transoesophageal echocardiography (TOE) guided cardioversion may be considered as an alternative to 3 weeks preprocedural anticoagulation and in this early cardioversion strategy the role of TOE is to exclude cardiac thrombus. Such an approach has to be followed by 4 weeks of anticoagulation or anticoagulation at long-term in patients with conventional indications to long-term anticoagulation.	'May do this'		7,241
For any patient with POAF, the indication for long-term anticoagulation has to follow the same approach as patients presenting with AF outside of a post-surgical context.	'May do this'		7,241
During the first 48 h after the onset of POAF, anticoagulation before and after cardioversion has to take into consideration the patient's risk of thromboembolism (CHA_2DS_2-VASc score) and the need for long-term anticoagulation, in association with the risk of post-operative bleeding.	'May do this'		7,241
When initiating anticoagulation attention to modifiable bleeding risk factors should be prioritized, with use of the HAS-BLED score to identify those patients at high bleeding risk for more regular review and follow-up.	'May do this'		7

Table 5 Examples of observational studies about thrombo-embolic risk in critically ill, sepsis, and ICU patients

Authors	No. of patients[a]	Patient population	Setting	Anticoagulation (no. of patients)	Summary
Critically ill					
Darwish *et al.*[232]	115	AF + sepsis	Retrospective, single centre	OAT ($n = 35$)	No strokes in this small cohort. Anticoagulation-related complications occurred more often in the group who received anticoagulation [8.6% (3/35) vs. 0%, $P = 0.008$].
Walkey *et al.*[233]	113 511	AF + sepsis	Retrospective, propensity score matched	Parenteral anticoagulation ($n = 13\,611$)	CHA_2DS_2-VASc scores poorly discriminated the risk of ischaemic stroke during sepsis (C statistic 0.526). Parenteral anticoagulation for AF did not change the rates of in-hospital ischaemic stroke events (RR 1.08; 95% CI 0.62–1.90) or bleeding (RR 1.23; 95% CI 0.88–1.72).
Walkey *et al.*[32]	49 082 (2896)	Sepsis (new-onset AF + sepsis)	Retrospective population-based cohort	Unknown	Compared with severe sepsis without new-onset AF, patients with new-onset AF during severe sepsis had greater risks of in-hospital stroke (adjusted OR 2.70; 95% CI 2.05–3.57; $P < 0.001$) and in-hospital mortality (adjusted OR 1.07; 95% CI 1.04–1.11; $P < 0.001$).
Walkey *et al.*[182]	138 722 (9540)	Sepsis (new-onset AF + sepsis)	Medicare 5% sample	Unknown	Compared with patients with no AF during sepsis, those with new-onset AF during sepsis had greater 5-year risks of hospitalization for heart failure (adjusted HR 1.25; 95% CI 1.16–1.34), ischaemic stroke (HR 1.22; 95% CI 1.10–1.36), and death (HR 1.04; 95% CI 1.01–1.07).
Champion *et al.*[234]	846 (108)	ICU patients (AF)	Prospective observational study	Unknown	During this 15-month study period, there were 12 (11%) AF-related arterial thromboembolism occurring 6 days after AF onset. Both $CHADS_2$ [OR 1.6 (1.1; 2.4); $P = 0.01$] and CHA_2DS_2-VASc scores [OR 1.4 (1.04; 1.8); $P = 0.03$] were significantly associated with systemic thrombo-embolic events.
Duarte *et al.*[230]	430	ICU patients (AF)	Cohort in seven general ICUs	Unknown	Incidence of acute new-onset AF was 11.2%. Patients with AF had higher ICU and hospital mortality.
Cancer					
Lee *et al.*[235]	2168	AF + cancer	Retrospective, single centre, propensity score matched	OAT ($n = 1380$)	After 1 year after cancer diagnosis, anticoagulation significantly reduced the composite endpoint.
Laube *et al.*[236]	163	AF + cancer + rivaroxaban	Retrospective, single centre	OAT ($n = 163$)	After adjusting for competing risks, the estimated 1-year cumulative incidence of ischaemic stroke was 1.4% (95% CI 0–3.4%) and major bleeding was 1.2% (95% CI 0–2.9%).
Ording *et al.*[237]	11 855 (56 264)	AF + cancer (AF only)	A population-based cohort study	Vitamin K antagonists ($n = 10\,046$), NOAC ($n = 1809$)	One-year risks of thrombo-embolic complications on VKA were similar in those with (6.5%) and without (5.8%) cancer (HR 1.0 95% CI 0.93, 1.1). This also was found for bleeding complications [5.4% vs. 4.3%, HR 1.1 (95% CI 1.0, 1.2)]. For AF patients with cancer on NOAC, risks were also similar for thrombo-embolic complications [4.9% of cancer patients vs. 5.1% of non-cancer patients, HR 0.80 (95% CI 0.61, 1.1), and for bleeding complications (4.4% vs. 3.1%, HR 1.2 (95% CI 0.92, 1.7)].
ICU					
Moss *et al.*[231]	8356 (1610)	ICU (AF + ICU)	Retrospective, single centre	Unknown	In propensity-adjusted regression analyses, clinical new-onset atrial fibrillation was associated with increased hospital mortality (OR 1.63; 95% CI 1.01–2.63) and longer length of stay (2.25 days; 95% CI 0.58–3.92).

AF, atrial fibrillation; CI, confidence interval; HR, hazard ratio; ICU, intensive care unit; NOAC, novel oral anticoagulant; OAT, oral anticoagulant therapy; OR, odds ratio; RR, relative risk; VKA, vitamin K antagonist.

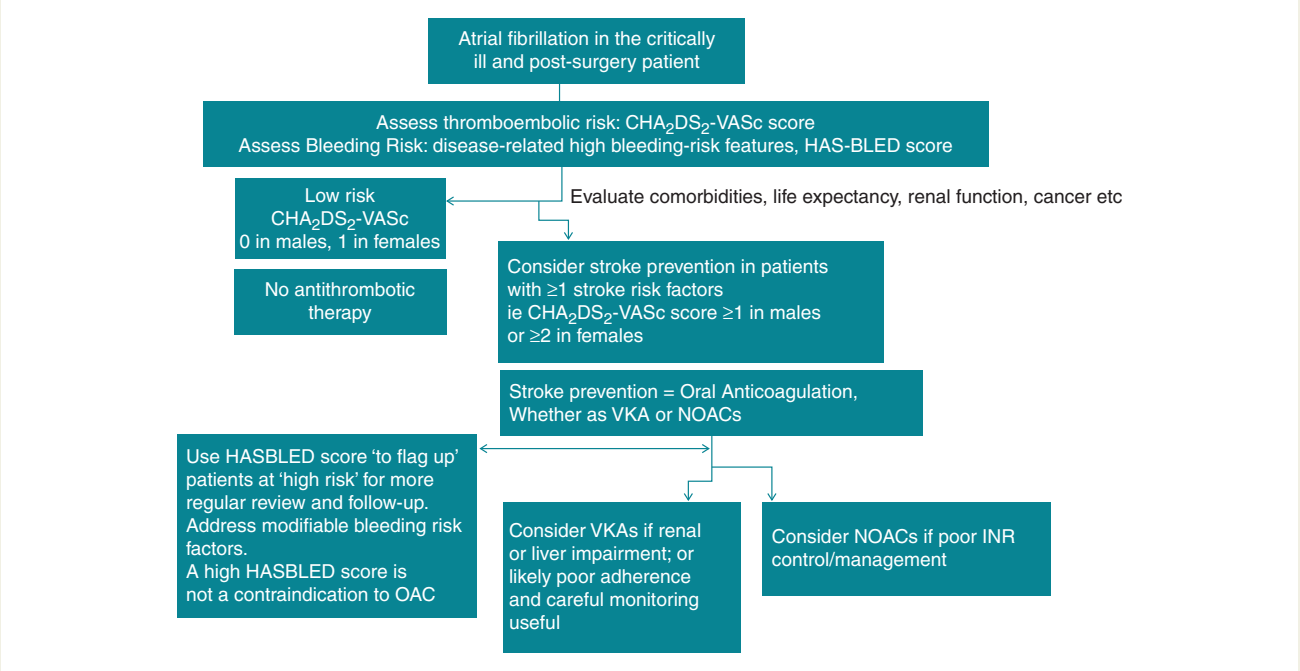

Figure 6 Suggested approach to thromboprophylaxis in patients with AF occurring post-surgery or in the context of a critical illness. NOAC, non-vitamin K antagonist oral anticoagulant; OAC, oral anticoagulant; VKA, vitamin K antagonist.

stroke risk factors. Stroke prevention means OAC whether as vitamin K antagonists (VKA) with a TTR >65–70% (and various clinical features can help identify patients likely to do well on VKA[238]), or with a NOAC. In all patients, attention to modifiable bleeding risk factors should be prioritized, although use of a formal bleeding risk score like HAS-BLED is a superior strategy compared to simply relying on modifiable bleeding risk factors, for clinicians to identify those patients at high bleeding risk for more regular review and follow-up[239,240] (*Figure 6*).

After a bleeding the reinitiation of oral anticoagulation has to be considered at a time which may vary according to type and severity of bleeding, removal of facilitating factors, and type of anticoagulant (warfarin or specific type of NOAC).[7] In case of ICH, reinitiation of NOACs after 6–8 weeks can be considered if the cause of haemorrhage as well as risk factors can be treated.[241] In case of no possibility to treat risk factors for haemorrhage, left atrial appendage occlusion can be considered even if this treatment requires dual antiplatelet treatment for some weeks after the intervention and is not devoid of complications.[7,241]

In case of cardioversion of AF or AFl the same recommendations of patients presenting out of the intensive care area have to be followed.[7]

Temporary pacing: indications and management

Temporary cardiac pacing (TCP) is a long-established, life-saving technique designed for the short-term treatment of patients suffering from rhythm disturbances and associated haemodynamic compromise both in the acute setting [e.g. complete AV block due to acute coronary syndrome (ACS)] as well as during scheduled interventions [e.g. before CIED re-implantation in pacemaker-dependent patients after previous device/lead(s) extraction due infective endocarditis or dysfunction].[46,219,242] Therefore, TCP may be used until a transient or reversible cause of bradyarrhythmia has resolved or has been treated (e.g. electrolyte or metabolic alterations, drug intoxications, injury to conduction system during cardiac surgery, myocardial infarction, myocarditis/pancarditis, spinal cord injury) and as a bridge to permanent pacing depending on the clinical scenario.[46,219,242,243] However, TCP pacing lacks strong unambiguous scientific evidence supporting its use in particular circumstances and indications for TCP are rather clinically driven. Due to potential risk of complications (generally 2–18%) temporary pacing should be avoided whenever possible.[219,244] It should be restricted only to symptomatic patients with unquestionable indications such as high-degree/complete AV block without stable escape rhythm or life-threatening bradyarrhythmias and used for a very limited period of time only if positive chronotropic drugs (e.g. atropine, isoproterenol, and epinephrine) have failed to stabilize the heart rhythm.[219] Temporary pacing wires may sometimes also be useful for intermittent overdrive pacing in specific tachyarrhythmias (e.g. in patients with TdP or polymorphic VT associated with bradycardia or QT interval prolongation).[46] Formally acknowledged indications for TCP are listed in the consensus statements below. There are several approaches to TCP: transcutaneous, transvenous, epicardial, transoesophageal. However, the first two methods are most commonly used. The use of a specific approach is dictated by the urgency for pacing, indications/contraindications, technical aspects, and potential complications of either technique.

Definitions where related to a treatment or procedure	Consensus statement instruction	Symbol	References
Temporary cardiac pacing is recommended in patients with symptomatic high-degree/complete AV block without stable escape rhythm despite pharmacotherapy in patients with ACS (block usually resolves within 2–7 days) or acute phase of myocarditis/pancarditis.	'Should do this'		46,219,242
Temporary cardiac pacing is recommended in patients with symptomatic bradycardia despite pharmacotherapy in patients with ACS or acute phase of myocarditis/pancarditis.	'Should do this'		46,219,242
Temporary cardiac pacing is recommended in patients with bradycardia dependent ventricular arrhythmias during the acute phase of myocarditis/pancarditis.	'Should do this'		46,219,242
Symptomatic high-degree/complete AV block without stable escape rhythm after cardiac surgery or transcatheter aortic valve implantation is an indication for temporary pacing (may resolve up to 7 days).	'Should do this'		219
Symptomatic SND after cardiac surgery and heart transplantation is an indication for temporary pacing (may resolve from 5 days up to some weeks after surgery).	'Should do this'		219
Temporary implantation of an active fixation electrode connected to an external device may be considered in pacing-dependent patients requiring prolonged antibiotic therapy before permanent CIED re-implantation.	'May do this'		247–253
Temporary cardiac pacing is not routinely recommended in asymptomatic patients with bradycardia, high-degree AV block with stable escape rhythm or bifascicular block (with or without first-degree atrioventricular block).	'Do not do this'		219

Transcutaneous external pacing provided by patches and an external defibrillator does not warrant reliable ventricular capture and should only be instituted in an emergency setting as a bridge to other means of temporary pacing when administration of chronotropic drugs is insufficient.[219]

Transvenous pacing is more reliable, painless, and enables both atrial and ventricular pacing. It however requires central venous access (thus cannot be initiated at once) and has some risk of complications (2–18%, particularly if performed in extreme emergencies), which cannot be neglected.[244] In general, internal jugular vein is preferable over femoral access due to increased incidence of deep vein thrombosis and infections in the latter. Complications of conventional transvenous pacing include not only local bleeding/haematoma, pneumo/haemothorax, lead malfunction/dislocation, proarrhythmia, cardiac perforation/tamponade but also an increased risk of further CIED infection.[245,246]

Thus if prolonged TCP is expected in a pacemaker-dependent patient, temporary use of an active fixation lead connected to an external re-usable pacemaker has been proposed in order to reduce potential adverse events.[247–253] Temporary epicardial pacing may be necessary in the perioperative period after cardiac surgery, whereas prophylactic cardiac pacing during non-cardiac surgery is not recommended.[81,219] The indications for perioperative TCP are the same as previously mentioned, although perioperative bradyarrhythmias usually respond well to pharmacological treatment and TCP is rarely required.[81]

Management of implantable devices in the critically ill and post-surgery patient

This section is given in Supplementary material online.

Arrhythmias, devices, and end-of-life

Implantable cardioverter-defibrillator (ICD) shocks improve survival in those at risk for sudden death. However, all patients will eventually die, whether through chronic terminal illness such as cancer or heart failure which, while unpredictable, usually exhibits a trajectory of inexorable functional decline[254] or through the development of acute overwhelming critical illness. In one recent study, 80% of a group of 130 consecutive deaths in ICD recipients occurred in hospital.[255] ICD shocks are painful, characterized by those experiencing them as, 'a punch in the chest', 'being kicked by a mule', 'putting a finger in a light socket'.[256] As recounted by family members, 20% of ICD patients are subjected to shocks when dying.[257] Post-mortem ICD interrogation shows this number even higher: 35% of patients had shockable ventricular arrhythmias in the last hour of life, with 24% having electrical storm.[255] Some people living and dying with progressively severe disease, or critically ill patients for whom further

treatment is futile, might choose to avoid life prolonging treatment, favouring possible sudden death by lethal arrhythmia.[258,259]

In order to decrease shocks and improve quality of life in dying patients, EHRA and HRS convened a multidisciplinary group of doctors, nurses, patients, lawyers, and ethicists, whose recommendations were published in 2010, 'EHRA/HRS Expert Consensus Statements on the management of cardiovascular implantable electronic devices in patients nearing end-of-life or requesting withdrawal of therapy'.[260,261] These documents described the ethical and legal underpinnings of deactivation of ICDs and highlighted the importance of proactive communication around ICD management by clinicians. Execution of an Advance Directive (AD) for ICD patients was strongly encouraged, in order to avoid ethical dilemmas for surrogate decision-makers should a patient nearing the end-of-life no longer be able to communicate his wishes.

However, advanced care planning specifically linked to cardiovascular disease has been poorly espoused by cardiac patients, and many clinicians find difficulty in discerning transition points when established therapy might become inconsistent with patients' shifting preferences for care or rendered futile.[261–265] While use of ADs is increasing for ICD patients, these are still enacted in just 30%.[266]

One barrier to improving palliation and timely deactivation of ICD shocking therapies for those with both acute and chronic terminal illness is the identification of when medical treatment becomes futile. The World Medical Association has defined medically futile treatment as that which offers 'no reasonable hope of recovery or improvement' or from which 'the patient is permanently unable to experience any benefit'.[267] Acknowledging futility is also relevant to defining ceilings of care, particularly in elucidating realistic outcomes of intensive care utilization, or those from cardio-pulmonary resuscitation, often subject to over-optimistic expectation.[268–272]

Communication is critical to avoid ICD shocks and other futile intervention at end-of-life. Accepting that people exhibit a broad spectrum of health literacy,[273] and the frequent requirement for patients and families to deal with highly technical issues in an unfamiliar environment, imparted information needs to be made comprehensible, with adequate time for assimilation and deliberation in a suitable setting.[274] This discourse should be offered as a balanced personalized approach, tailored to their needs, thereby allowing them to participate fully in a valid shared decision-making process as proposed in the Salzburg Statement and bioethical guidelines.[275,276] While some are under evaluation, currently there is a paucity of specific decision aids addressing ICD deactivation to facilitate this process.[277–279] Several ongoing studies are evaluating interventions to improve communication around end-of-life goals of care.[280,281]

Patient follow-up, risk of arrhythmia recurrences, and clinical decision-making at long-term

Arrhythmias either of supraventricular or ventricular origin have prognostic implications for critically ill patients during the phase of hospitalization in an ICU. Patients who develop arrhythmias are more likely to be sicker than those not presenting arrhythmias and often present very complex clinical scenarios.[10,158,282]

An important clinical question is if an arrhythmia occurring in the acute phase of a critical illness, in a milieu characterized by many concurrent factors facilitating arrhythmogenesis, has to be considered as a simple marker of transient conditions or, rather, carries some implications also for the outcome post-discharge from the ICU, with potential impact on long-term prognosis, thus requiring a specific post-ICU decisions-making.

In order to systematically approach this complex clinical issue, supraventricular arrrhythmias, specifically AF need to be distinguished from ventricular arrhythmias.

Supraventricular arrhythmias and atrial fibrillation

In 1998, a position paper from the European Society of Cardiology reported that 'AF may be related to acute causes and may not recur should the cause disappear or be cured' and indicated that the term 'transient' AF was often applied to these situations, corresponding to cardiac or thoracic surgery, as well as acute alcohol intake ('holiday heart syndrome'), electrocution, acute myocardial infarction, acute pericarditis, acute myocarditis, pulmonary embolism, hyperthyroidism, and acute pulmonary disease.[283] Twenty years later, data from a series of studies indicate that these statements, which have important implications for daily practice, need to be revised.

When a supraventricular arrhythmia (SVT) develops during the acute phase of a severe illness, the short-term (i.e. at 30 days) prognosis is worse than for patients without detected arrhythmias. This has been shown for AF developing in the days that follow thoracic surgery[284,285] and need to be assessed also for any patient developing new-onset AF in the context of an acute illness.

Other organized SVTs (except for AFl) are less common than AF in critically ill patients. They often lead to haemodynamic imbalance and may require DC cardioversion.[286] The pathophysiology of SVT is well established and evidence-based treatments are commonly available.[188] As well as AF and AFl, they have a significant impact on the prognosis of patients.[282]

New-onset AF maintains its prognostic significance after discharge from an ICU. Patients with AF-precipitating factors have a consistent risk of recurrences in the long-term, even if transient precipitating factors resolve.[103,115,182,287] The risk of recurrences does not differ between patients with and without precipitating factors. Moreover, the risk of stroke and heart failure is also elevated. The association between new-onset AF developing in the setting of an acute illness and increased risk of mortality has been object of investigation, and the mortality at 30–60 days may vary from 15.8% (in the setting of liver transplant) to 72% (in post-surgical sepsis).[288–290] In a retrospective study analysing administrative databases, POAF was seen in 3% of over 370 000 patients in a setting of non-cardiac-surgery[90] and POAF appeared to increase the likelihood of death.

Patients undergoing isolated coronary artery bypass graft, showed an increased mortality at 6 years when new-onset AF developed, without a previous history of AF (adjusted HR 1.21, 95% CI 1.21–1.32; $P < 0.0001$).[123] In this study, the survival curves showed an early divergence and the distance between the two curves increased over time. Moreover the same study showed a significantly increased risk of post-operative stroke in patients with POAF (3.2% vs. 1.3% no

Table 6 Short-term (28–60 days) prognosis of atrial fibrillation in critically ill patients in the literature

Authors	Design	Size	Setting	Summary of finding	Comment
Moss *et al.*[231]	Retrospective	8356	Surgical/trauma/ burn ICU	NOAF not associated with survival after hospital discharge (HR 0.99; 95% CI 0.76–1.28 and HR 1.11; 95% CI 0.67–1.83, respectively, for SCAF and clinical NOAF) Prior AF HR 1.55; 95% CI 1.29–1.88	AF detected in 1610 admissions (19%), with median burden less than 2%. NOAF was subclinical or went undocumented in 626, or 8% of all ICU admissions.
Kotova *et al.*[298]	Prospective	933	Post-lobectomy	30-Day survival better in no POAF (97.1%) than in POAF patients (90.3%) $P = 0.0003$	POAF developed in 12%
Gillinov *et al.*[299]	RCT	523	Cardiac surgery	Rate vs. rhythm control in POAF after cardiac surgery no differences in 60-days readmission for all cause and cardiac causes reasons	POAF in 33% of patients
Guenancia *et al.*[297]	Prospective	66	Septic shock	NOAF did not predict 28 day mortality.	NOAF in 44% of patients (1/3 SCAF detected by 7-days-Holter monitoring). No differences in severity of illness and treatment
Chen *et al.*[290]	Retrospective	741	Medical ICU	60-Day mortality (NOAF 51% vs. no NOAF 23%; adjusted OR 1.99, 95% CI 1.01–3.91; $P = 0.047$).	NOAF 7.2%. NOAF identified through ICD-9 codes charge and confirmed by reviewing medical records
LaPar *et al.*[301]	Retrospective	49 264	Cardiac surgery	POAF patients had a higher unadjusted incidence of 30-days hospital readmission (9.4% POAF vs. 7.5%; $P < 0.05$)	Data from administrative databases from 2001 to 2012
Vannucci *et al.*[289]	Retrospective	727	Liver transplantation	RR of 1-month mortality in the pre-transplantation atrial fibrillation group was 5.29 at 1 month ($P = 0.0034$; 95% CI 1.73–16.18)	Pre-transplantation prevalence of AF 2.5%
Saxena *et al.*[296]	Retrospective	19 497	CABG surgery	Patients with POAF demonstrated a greater 30-day mortality on univariate analysis but not on multivariate analysis ($P = 0.376$). POAF independently associated with shorter long-term survival ($P = 0.002$)	28.5% of the patients developed POAF
Walkey *et al.*[32]	Retrospective	3 144 787	Severe sepsis	Rehospitalization with a new ischaemic stroke occurred in 2.0% with NOAF during severe sepsis, 1.5% with pre-existing AF during sepsis, and 1.3% without AF during severe sepsis.	Non-significantly increased risk of rehospitalization with incident ischaemic stroke (multivariable-adjusted HR 1.51; 95% CI 0.98–2.33; $P = 0.06$).
Salman *et al.*[288]	Retrospective	81	Sepsis in medical, obstetric-gynaecology, and surgical critically ill patients	Paroxysmal AF independently associated with 28-day mortality (OR 3.284; 95% CI 1.126–9.574).	Paroxysmal AF developed in 31% of patients
Devereaux *et al.*[302]	Prospective	8531	8351 patients with, or at risk of, atherosclerotic disease who were undergoing non-cardiac surgery randomized to receive extended-release metoprolol succinate or placebo	New clinically significant developed in 2.2% patients on metoprolol vs. 2.9% on placebo (HR 0.76, 95% CI 0.58–0.99; $P = 0.0435$)	At 30-day follow-up new clinically significant AF was associated with an increased risk of stroke at 30 days (OR 3.51; 95% CI 1.45–8.52)
Amar *et al.*[285]	Prospective	100	Oesophagectomy	Patients in whom SVT developed had a higher rate of 30-day mortality rate (SVT 15% vs. 3% no SVT; $P = 0.013$)	Incidence of SVT 13%. SVT was not the direct cause of death
Amar *et al.*[284]	Prospective	100	Pulmonary surgery	Patients who developed SVT had a higher 30-day mortality rate (SVT 3/18 vs. no SVT 1/82 $P < 0.02$)	Incidence of SVT 18%.

AF, atrial fibrillation; CABG, coronary artery bypass graft; CI, confidence interval; HR, hazard ratio; ICD-9, International Classification of Diseases revision 9; ICU, intensive care unit; NOAF, new-onset atrial fibrillation; OR, odds ratio; POAF, post-operative atrial fibrillation; RCT, randomized clinical trial; RR, relative risk; SCAF, subclinical atrial fibrillation; SVT, supraventricular tachyarrhythmias.

Table 7 Long-term prognosis of atrial fibrillation in critically ill patients

Authors	Study design	Size	Setting	Summary of findings	Comments
Carrera *et al.*[291]	Retrospective	10 836	Medical ICU	One-year survival after ICU discharge was similar for NOAF and previous AF groups and worst when compared with non-AF (54%, 52%, 75%; $P < 0.001$, log-rank)	Non-AF patients as reference. One-year-survival rate better for non-AF patients
Ambrus *et al.*[292]	Prospective	282	ARDS	NOAF during ARDS associated with increased 90-day mortality (NOAF 43% vs. no NOAF 19%, APACHE-adjusted OR 3.09, 95% CI 1.24–7.72; $P = 0.02$).	Patients with NOAF 10%
Tonorezos *et al.*[293]	Retrospective	1177	Haemopoietic cells transplant ≥40 y.o.	Patients with AF/AFl and SVT post-transplant have greater probability of death within 1 year of transplant (41% vs. 15%; OR 3.5; 95% CI 2.1–5.9; $P < 0.001$)	
Guenancia *et al.*[297]	Prospective	66	Septic shock	NOAF did not predict 90-day mortality of NOAF vs. sinus rhythm patients (41% vs. 43% respectively, $P = 0.88$).	NOAF in 44% of patients (1/3 SCAF detected by 7-days-Holter monitoring). No differences in severity of illness and treatment
Lubitz *et al.*[115]	Retrospective	1409	Surgical, medical, cardiac ICU	Mortality for NOAF (HR 1.00 95% CI 0.87–1.15) was similar between those with and without secondary precipitants	Median follow-up 5.4 years
Gizurarson *et al.*[303]	Prospective	35 232	CCU	AF increased 1-year mortality (HR 1.32, 95% CI 1.16–1.50; $P < 0.001$)	
Walkey *et al.*[182]	Retrospective	138 722	Sepsis	When compared with patients without AF, patients with NOAF had a greater risk of post-sepsis mortality (5-year unadjusted risk, 74.8% vs. 72.1%; multivariable-adjusted HR 1.04 95% CI 1.01–1.07),	relative heart failure and mortality risks associated with NOAF (vs. no AF) declined over time
Thorén *et al.*[294]	Retrospective	6821	CABG surgery	POAF related to increased cardiac mortality (HR 1.4, 95% CI 1.3–1.5); death related to arrhythmia (HR 1.8, 95% CI 1.6–2.0); cerebrovascular disease (HR 1.4, 95% CI 1.2–1.6); and heart failure (HR 1.4, 95% CI 1.3–1.6)	32% developed POAF. Median follow-up was 9.8 years
Vannucci *et al.*[289]	Retrospective	727	Liver transplantation	Compared with patients without atrial fibrillation, the relative risk of death in the atrial fibrillation group was 3.28 at 1 year ($P = 0.0008$; 95% CI 1.63–6.59)	Pre-transplantation AF 2.5%
Al-Shaar *et al.*[295]	Retrospective	1196	CABG surgery	POAF significantly worse, yet time-varying, 0- to 18-year survival vs. no POAF	
Gialdini *et al.*[103]	Retrospective	1 729 360	Cardiac and non-cardiac surgery	One-year risk of stroke for POAF (vs. no POAF) HR (95% CI) for non-cardiac surgery 2.0 (1.7–2.3) and HR for cardiac surgery 1.3 (1.1–1.6). The association with stroke was significantly stronger for POAF after non-cardiac vs. cardiac surgery ($P < 0.001$ for interaction)	24 711 (1.43%; 95% CI 1.41%–1.45%) had POAF; 13 952 (0.81%; 95% CI 0.79%–0.82%) experienced a stroke after discharge
Lomivorotov *et al.*[304]	Randomized	39	Cardiac surgery	2-years presence of AF 27, 8% in controls and 35% in fish oil group ($P = 0.64$).	Evidence from loop recorder monitoring. Fish oil in preventing post-CCH AF. Two stroke/TiA in the placebo group.
Imperatori *et al.*[96]	Prospective	454	Lobectomy	Post-operative AF independently predicted poorer 5-years survival (HR 3.75; 95% CI 1.44–9.08)	POAF occurred in 45 (9.9%). Median follow-up 36 months (maximum: 179 months).

Continued

Table 7 *Continued*

Authors	Study design	Size	Setting	Summary of findings	Comments
El-Chami *et al.*[123]	Retrospective	16 169	CABG	New-onset AF occurred in 2985 (18.5%) patients. POAF independently predicted long-term mortality (HR 1.21; 95% CI 1.12–1.32). Adjusted effect of warfarin on mortality in POAF patients seems to be protective (HR 0.78, 95% CI 0.66–0.92).	Mean follow-up of 6 years (range 0–12.5 years). Patients with POAF on warfarin 20.5%
Meierhenrich *et al.*[165]	Prospective	687	Septic shock	Two-year follow-up: trend towards an increased mortality in septic shock patients with new-onset AF (vs. no AF), but the difference did not reach statistical significance ($P = 0.075$).	Failure to restore SR was associated with increased ICU mortality (71.4% vs. 21.4%, $P = 0.015$)
Bramer *et al.*[305]	Prospective	5098	CABG surgery	POAF was an independent predictor of overall and late mortality HR 1.35 ($P = 0.012$ and $P = 0.039$, respectively)	Median follow-up 2.5 years
Ahlsson *et al.*[121]	Retrospective	571	CABG surgery	POAF patients, 25.4% had AF at follow-up vs. 3.6% of patients with no POAF ($P < 0.001$). Mortality was 29.7% in POAF and 14.8% in non-POAF ($P < 0.001$). Death due to cerebral ischaemia more common in POAF group (4.2% vs. 0.2%, $P < 0.001$)	Median folllow-up 6 years. Post-operative AF occurred in 165/571 patients (28.9%)
Filardo *et al.*[306]	Retrospective	6899	CABG surgery	Ten-year unadjusted survival was 52.3% for POAF and 69.4% for no POAF. After adjustment, POAF significantly associated (HR 1.29; 95% CI 1.16, 1.45) with increased risk of death	
Berton *et al.*[307]	Prospective	505	CCU for AMI	After 7 years of follow-up, AF/FL was found to be associated with all-cause mortality (adjusted OR 1.6; 95% CI 1.2–2.3) and sudden death (adjusted OR 2.7; 95% CI 1.2–6.4) log-rank $P < 0.0001$	ACE-inhibitors and digitalis protective against all-cause and sudden death
Mariscalco *et al.*[118]	Retrospective	9495	Cardiac surgery	POAF independently affected long-term survival in CABG patients (HR 1.22; 95% CI 1.08–1.37). Non-significant effect on valve or combined surgery	The overall AF incidence was 26.7%, subdivided into 22.9%, 39.8%, and 45.2% for CABG, valve surgery, and combined procedures. Median follow-up 7.9 years
Frontera *et al.*[43]	Prospective	580	Spontaneous SAH neurological ICU	After adjusting for several variables arrhythmias were independent predictors of mortality (adjusted OR 8.0, 95% CI 1.9–34.0; $P = 0.005$) and severe disability or death (mRS 4–6; adjusted OR 6.9, 95% CI 1.5–32.0; $P = 0.014$) at 3 months	Arrhythmias (mainly AF/Afl 76%) developed in 4.3%. Sixteen of 25 (64%) with any arrhythmia died.
Villareal *et al.*[127]	Retrospective	6475	CABG surgery	At 4–5 years, survival worse in patients with POAF (74% vs. 87%, $P < 0.0001$). On multivariate analysis, POAF independent predictor of long-term mortality (adjusted OR 1.5, $P < 0.001$)	AF diagnosed in 16% of the population.
Almassi *et al.*[308]	Retrospective	3855	Cardiac surgery	6-month mortality significantly higher in AF 9.36% vs. 4.17% no AF ($P < 0.001$)	The incidence of post-operative AF was 29.6%.

ACE, Angiotensin Converting Enzyme; AF, atrial fibrillation; Afl, atrial flutter; AMI, acute myocardial infarction; APACHE, acute physiologic assessment and chronic health evaluation; ARDS, acute respiratory distress syndrome; CABG, coronary artery by-pass graft; CCU, coronary care unit; CI, confidence interval; HR, hazard ratio; ICU, intensive care unit; mRS, modified Rankin scale; NOAF, new-onset atrial fibrillation; OR, odds ratio; POAF post-operative atrial fibrillation; SAH, subarachnoid haemorrhage; SCAF, subclinical atrial fibrillation; SVT, supraventricular tachycardia; y.o., years old.

Table 8 Recurrences of atrial fibrillation after discharge from a critical illness

Authors	Study design	Size	Setting	Summary of findings	Comment
El-Chami *et al.*[309]	Prospective	23	CABG surgery	60.9% of patients with POAF develop recurrent AF	Recurrences diagnosed with loop recorder
Lowres *et al.*[310]	Prospective	42	Cardiac surgery	Within 3 weeks recurrence in 10/42 participants (24%)	Recurrences diagnosed with iphone and iECG sent 4-times a day. Symptoms were not a reliable indicator of AF recurrence (only 30% symptomatic)
Lubitz *et al.*[115]	Retrospective	1409	Medical, surgical, cardiac ICU	AF recurred in 544 of 846 without permanent AF (5-, 10-, and 15-year recurrences of 42%, 56%, and 62% with vs. 59%, 69%, and 71% without secondary precipitants; multivariable-adjusted HR 0.65, 95% CI 0.54–0.78)	Median follow-up 5.4 years. Stroke risk (n = 209/1262 at risk; HR 1.13 95% CI 0.82–1.57) was similar between those with and without secondary precipitants
Guenancia *et al.*[300]	Prospective	100	CABG surgery	At 1-year follow-up, 30% of SCAF patients had developed symptomatic AF vs. 7% in the SR group (P = 0.03) and 11% in the clinical AF group (P = 0.21).	No hx of AF. New SCAF 13%, clinical AF 21%. One stroke in SCAF group and 1 in SR group
Walkey *et al.*[182]	Retrospective	138 722	Sepsis	Within 1 year, 44.2% of patients with NOAF during sepsis were given another AF diagnosis, as compared with 57.2% of patients with prior AF and 7.7% of patients with no AF.	Ischaemic stroke hospitalization associated with new-onset AF (vs. no AF) (5.3% vs. 4.7%; HR 1.22, 95% CI 1.15–1.47)

AF, atrial fibrillation; CABG, coronary artery bypass graft; CI, confidence interval; HR, hazard ratio; ICU, intensive care unit; NOAF, new-onset atrial fibrillation; OR, odds ratio; POAF, post-operative atrial fibrillation; SCAF, subclinical atrial fibrillation.

POAF; $P < 0.0001$); in patients with POAF, warfarin was administered in approximately 20% of the patients, with a positive impact on mortality (adjusted HR 0.78, 95% CI 0.66 to 0.92). The negative prognostic value of AF was confirmed in patients discharged from medical ICU,[291] post-acute respiratory distress syndrome,[292] treated with haemopoietic cells transplant,[293] cardiac, and non-cardiac sugery.[103,107,294–296] However, other studies do not confirm the negative prognostic implications of AF.[231,297] Studies summarizing short-term and long-term mortality are listed in*Tables 6* and *7* and it appears that the association of AF with a negative outcome is more clear in the long-term rather than in the short-term.

With regard to arrhythmia recurrences, in a retrospective study[182] considering patients admitted for sepsis, patients with new-onset AF had a greater rate of recurrent AF at 5 years (63.5% AF in previous AF, 54.9% new-onset AF without previous AF, 15.5% in no AF; $P < 0.0001$) independent of a history of previous AF. Another retrospective survey,[115] from the Framingham study, was conducted in 1409 patients, admitted for various reasons and presenting with new-onset AF with or without precipitant factors (surgery, infection, acute myocardial infarction, thyrotoxicosis, acute alcohol consumption, acute pericardial disease, pulmonary embolism, or other acute pulmonary disease). The results showed that the long-term risk of AF recurrences was substantial even in patients with secondary precipitants although the risk of recurrences was statistically lower. The stroke risk was similar between those with and without secondary precipitants of AF (*Table 8*).

In a randomized placebo-controlled trial (the POISE trial)[302] evaluating the effect of metoprolol succinate in preventing the composite endpoint of cardiovascular death, non-fatal myocardial infarction, and non-fatal cardiac arrest after non-cardiac surgery, development of new clinically significant AF was associated with an increased risk of stroke at 30 days (OR 3.51; 95% CI 1.45–8.52). The population attributable risk (i.e. the proportion of the outcome attributable to this risk factor with causality proven) was 6.9% (2.1–20.4).

In critically ill patients after cardiac and non-cardiac surgery, the risk of developing POAF is different between the two scenarios but the actual risk and the increase in risk of stroke are substantial in both populations. In detail, the rates of stroke at 1 year for POAF after cardiac surgery were 0.99% and for non-cardiac surgery 1.47%, while the increase in risk of stroke for POAF (vs. no POAF) corresponded to a HR (95% CI) for cardiac surgery of 1.3 (1.1–1.6) and for non-cardiac surgery of 2.0 (1.7–2.3).[103]

New-onset AF is subclinical in 8% of the patients,[231] but there is a substantial risk of underdiagnosis. The latter issue depends on intermittent electrocardiographic monitoring, since symptoms are unreliable indicators of AF.[310] When the search for AF was conducted with continuous monitoring systems (loop recorder), 60.9% of patients with POAF were shown to develop recurrent AF.[309]

In summary, new-onset AF in critically ill patients could increase mortality but it certainly has a high risk of recurrences and stroke in this type of patients, as well as in those without secondary precipitating factors. The lack of randomized controlled trials in this field

Table 9 Long-term outcome and prognosis of patients suffered a VA/VT/cardiac arrest for secondary reasons

Authors	Study design	Size	Setting	Summary of findings	Comments
El-Battrawy *et al.*[321]	Retrospective	114	Stress cardiomyopathy	Patients with LTA have a worse 30-day prognosis than patients without LTA (23% vs. 5.9% $P < 0.05$) but similar 1- and 3-year outcome	Administrative databases. Three years of follow-up
Mosorin *et al.*[322]	Retrospective	48	OHCA patients treated with CABG	30-Day post-operative mortality was higher but not significant among OHCA patients than non-OHCA (6.3 vs. 0%, $P = 0.24$). At 5-year, the overall survival rate was 80.7% in OHCA and 84.5% in non-OHCA ($P = 0.98$)	ICD in two patients alive at 3.8 and 4.4 years after CABG.
Røsjø *et al.*[323]	Prospective	155	OHCA	Admission hs-TnT levels were higher in 1-year non-survivors compared to survivors. hs-TnT did not add prognostic information to established risk variables in multivariate analyses	
El-Chami *et al.*[199]	Retrospective	14 720	Cardiac surgery	Sustained VT/VF occurred post-operatively in 248 patients (1.7%). VT/VF associated with increased adjusted long-term mortality (HR 2.53, $P < 0.001$)	3.5 years of follow-up
Cacciotti *et al.*[324]	Prospective	75	Stress cardiomyopathy	One patient with VF cardiac arrest. Two patients died at follow-up, one for sudden death	Mean follow-up 2.2 years
Annane *et al.*[10]	Prospective	1341 (2% VA)	ICU (CCU and post-operative)	Neurological sequel rates were in survivors with VA vs. non-VA (OR 7.53; 95% CI 1.60–35.50). After adjusting for prognosis factors and propensity scores, VA increased mortality vs. non-VA (OR 3.53; 95% CI 1.19–10.42)	
Wyse *et al.*[318]	Prospective	4450 (278 transient/correctable cause)	AVID registry	Adjusted outcome worse ($P = 0.008$) in the group with VT/VF due to transient or correctable causes vs. primary VT/VF	Main cause of transient/correctable VA were ischaemic events, electrolyte imbalance
Anderson *et al.*[317]	Prospective	4451	Survivors from VA in the AVID registry	Crude mortality rates (mean follow-up 16.9 months) were: stable (asymptomatic) VT, 19.7% (497, 98 deaths); VT/VF with transient/correctable cause, 17.8% (270, 48 deaths); and unexplained syncope, 12.3% (390, 48 deaths).	Patients with seemingly lower-risk or unknown-risk VA's (asymptomatic VT, and VT/VF associated with a transient factor) have a high mortality similar to that of higher risk, AVID-eligible VA's patients

AVID, antiarrhythmics vs. implantable defibrillators; CABG, coronary artery bypass graft; CCU, coronary care unit; CI, confidence interval; hs-TnT, high-sensitivity troponin T; HR, hazard ratio; ICD, implantable cardioverter-defibrillator; ICU, intensive care unit; LTA, life-threatening arrhythmias; OHCA, out-of-hospital cardiac arrest; OR, odds ratio; VA, ventricular arrhythmias; VF, ventricular fibrillation; VT, ventricular tachycardia.

prompts us to suggest the application of recent AF guidelines.[7] Nonetheless, the American College of Chest Physicians Guidelines specifically state that their recommendations 'do not apply to patients with a single, transient, self-limited episode of AF associated with acute illness'. [311] In instituting oral anticoagulation special attention must be dedicated to haemorrhagic risk in frail patients and correction of modifiable risk factors for bleeding. Subclinical episodes of AF, as revealed by implanted devices, are also associated with an increased risk of stroke, and should be object of specific consideration.[312–316]

Ventricular tachyarrhythmias

This section will consider ventricular tachyarrhythmias due to transient/correctable causes. Outside of this setting, the strategies for primary or secondary prevention of VT/VF are well established, with a well-defined role for implantable defibrillators with or without resynchronization features.[46]

The terms 'transient' or 'correctable' factors usually apply to patients admitted to ICU for VT/VF due to acute coronary syndromes, electrolyte imbalances, and QT prolongation during pharmacotherapy that affect repolarization. Whether these facilitating and/or precipitating factors are really removable is the key issue in order to guarantee avoidance of arrhythmia recurrences. This may be complicated by the apparent absence of alternatives to some drugs (i.e. psychiatric drugs).

In antiarrhythmics vs. implantable defibrillators, patients who have experienced out-of-hospital VT or VF thought to be of transient or correctable cause (e.g. in conjunction with myocardial infarction, acute ischaemia, drug overdose, or severe electrolyte imbalance) were included in a registry to evaluate whether their outcome differed from that of patients without transient or correctable causes.[317] The outcome of these patients was poor and patients identified with a transient or correctable cause for their VT/VF remained at high risk for death, similar to that of patients with primary arrhythmias.[317,318] The issue is that the 'transient and correctable' factors may not be correctable or, even, that so-called 'transient factors' are not really the cause of the arrhythmia.[319] Indeed, patients with hypo- or hyperkalaemia have the same rate of ICD intervention as patients with normal serum potassium level.[320] A complete picture of the patient, considering age, comorbidities, and cardiac conditions is thus needed before excluding a patient from ICD treatment, even if this is matter of controversy (*Table 9*). It is noteworthy that it may be difficult to prevent electrolyte disorders in renal failure or in dialysis patients.

The ESC guidelines on management of ventricular arrhythmias[46] recommend that in case of ventricular tachyarrhythmias developing in association with 'transient causes' (e.g. drugs, electrolyte imbalances, chest trauma) it is indicated to do a full cardiovascular evaluation and evaluate for presence and extent of cardiovascular diseases (with history, ECG, echocardiogram/CMR, and other diagnostic tests, if indicated) and then evaluate for complete reversal of causes. In addition, patients who experienced life-threatening arrhythmias during the course of Takotsubo syndrome have a worse 30-day prognosis than patients who did not.[321] Interestingly, 1-year outcome is similar between patients with and without ventricular arrhythmias in the acute phase of the disease.

In critically ill patients with transient impaired LVEF, a wearable cardioverted defibrillator may be used for a limited period of time, until LV function has recovered sufficiently, following insults such as myocardial infarction, post-partum cardiomyopathy, myocarditis, or interventions such as revascularization associated with transient LV dysfunction. Similarly, patients with a history or at risk of life-threatening ventricular tachyarrhythmias who are scheduled for cardiac transplantation may be temporarily protected with a wearable defibrillator, as a bridge to transplant.[325]

Areas for future research

In the setting of critically ill and post-surgical patients there are some unmet needs with regard to the risk of arrhythmias and arrhythmia-related complications that deserve further research. These areas can be summarized, as follows:

Definitions where related to a treatment or procedure	Consensus statement instruction	Symbol	References
In critically ill patients with AF-precipitating factors, it is indicated to consider that these patients have a consistent risk of recurrences in the long-term, even if transient precipitating factors resolve, and that the risk of stroke, heart failure, and mortality are also elevated as compared with patients without AF.	'Should do this'		92,115,182,287
In the case of life-threatening ventricular tachyarrhythmias developing in critically ill patients in association with 'transient and correctable factors' (e.g. drugs, electrolyte imbalances, transient ischaemia), it is indicated to do a full cardiological evaluation and assess if these factors are really the cause of the arrhythmia and if they can be corrected and prevented, thus leading to a complete reversal of causes, or not.	'Should do this'		46
In critically ill patients with transient impaired LVEF, a wearable cardioverted defibrillator may be used for a limited period of time, until LV function has recovered, or, similarly, it may be used as a bridge in patients waiting for a cardiac transplant.	'May do this'		325

- Improved risk stratification for the risk of developing a sustained arrhythmia and particularly AF during exposure to transient facilitating factors (surgical interventions, sepsis states, electrolytes imbalances, etc.) in order to improve patient monitoring and promptly detect and manage the arrhythmia in different settings and according to different types of surgery.
- Better evaluation of the efficacy of preventive medical treatments targeted to reduce the risk of incident AF in the post-operative phase of different types of surgical interventions.
- Collection of data from real world practice on the epidemiology and management of AF occurring in the post-operative phase, with specific focus on prescription of oral anticoagulants. Protocols for managing these patients and collection of data on complications and outcomes are needed.
- Improved clinical risk stratification with regard to the haemorrhagic risks of oral anticoagulants in the specific setting of the post-surgical phases or in the setting of critically ill patients in whom transient factors may interact with various comorbidities and increase the risk of bleeding, as well as assessing the impact on outcomes of preventing and correcting specific transient risk factors for bleeding.
- Improved integrated care of critically ill patients, by enhancing the use of multidisciplinary teams at a local level, given the increased complexity of patients currently requiring intensive care.

Conclusion

The development of arrhythmias in the ICU patient may significantly worsen prognosis and requires thorough evaluation and proactive management in the ICU and post-ICU settings. Correction of the numerous precipitating causes should be part of the treatment, as arrhythmias may decrease or disappear after these measures are taken. Despite of a relative lack of randomized trials and robust data evaluating therapeutic strategies, a systematic and holistic approach is needed to improve the management of these common issues and to reduce their significant health care burden.

Supplementary material

Supplementary material is available at *Europace* online.

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