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NARRATIVE REVIEW

Atrial fibrillation in critical illness: state of the art

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Abstract

Atrial fibrillation (AF) is the most common arrhythmia experienced by critically ill patients. It has been associated with adverse short-and long-term outcomes, including an increased risk of thromboembolic events, heart failure, and death. Due to complex and multifactorial pathophysiology, a heterogenous patient population, and a lack of clinical tools for risk stratification validated in this population, AF in critical illness is challenging to predict, prevent, and manage. Personalized management strategies that consider patient factors such as underlying cardiac structure and function, potentially reversible arrhythmogenic triggers, and risk for complications of AF are needed. Furthermore, evaluation of the effects of these interventions on long-term outcomes is warranted. Critical illness survivors who have had AF represent a unique population who require systematic follow-up after discharge. However, the frequency, type, and intensity of follow-up is unknown. This state-of-the-art review aims to summarize the evidence, contextualize the current guidelines within the setting of critical illness, and highlight gaps in knowledge and research opportunities to further our understanding of this arrhythmia and improve patient outcomes.

Keywords: Atrial fibrillation, Critical illness

Introduction

Atrial fibrillation (AF) is common in critically ill patients [1] and its management is a daily challenge for intensive care unit (ICU) clinicians. Given the increasing age of ICU patients, pre-existing AF is common with a prevalence higher than the general population at approximately 10–20% [2, 3]. For these patients, the management of their arrhythmia while acutely ill may differ from their home management, and decisions regarding the resumption of rate and rhythm controlling medications and anticoagulation need to be balanced against the risks of hemodynamic decompensation and bleeding. "New-onset" AF (NOAF) describes the diagnosis of AF in patients without documented history of AF. NOAF is reported to affect between 5 and 15% of all critically unwell hospitalized patients [4, 5] and up to 46% of patients admitted with septic shock [6]. This diverse group of patients also poses a unique challenge, as it is often unclear if they have "*reversible*" AF brought about by exposure to transient triggers that will resolve with recovery from critical illness, or if they have "*provoked*" AF, with underlying abnormal atrial substrate that predisposes them to AF, alongside arrhythmogenic triggers that unmask the arrhythmia [7]. Some patients may experience rapid, potentially reversible cardiac remodeling due to inflammation, lowering the threshold for developing NOAF [8]. This differing pathophysiology will likely lead to differences in clinical response to treatment. While some patients may have resolution with treatment of their underlying condition and correction of modifiable risk factors, others may require additional interventions to control rate and rhythm and have increased risk of AF recurrence after ICU discharge.

Regardless of the type, AF during critical illness is associated with prolonged hospitalization, increased

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mortality [9, 10], and higher thromboembolic risk [4, 10, 11]. Once thought to be an arrhythmia limited to the episode of critical illness, newer data indicates the clinical impact of AF during critical illness persist in ICU survivors. Observational studies have demonstrated increased long term (≥1 year after ICU) mortality among patients with NOAF [12–14], or who have a history of chronic AF [13, 15]. Equally concerning are the increased long-term risks of stroke and heart failure up to six years after hospital discharge [12, 16, 17].

Strong guideline recommendations to inform AF management are challenging due to the heterogeneity of critically ill patients and the multifactorial and complex pathophysiology that drives AF. This is further limited by a lack randomized controlled trials in critically ill populations, forcing extrapolation of retrospective data, small observational studies, and studies in non-critically ill populations. Adoption of guidelines intended for non-critically ill patients to critical care settings may not be practical or appropriate, resulting in significant variability in the management of AF in critically ill patients [18–20].

The objectives of this state-of-the-art review are to summarize current evidence, contextualize the current guidelines within the setting of critical illness, and highlight gaps in knowledge and research opportunities to further our understanding of this arrhythmia and improve patient outcomes. (Fig. 1) A summary of knowledge gaps and research opportunities is presented in Table 1.

Take-home message

Atrial fibrillation is the most common arrhythmia in critically ill patients and is associated with poor short- and long-term outcomes. Management is multifaceted and requires an individualized approach, including consideration of individual patient factors, arrhythmogenic triggers, the risk–benefit ratio of therapies, and the need for follow-up.

Several important questions remain unanswered in critically ill patients and further research is needed to reduce morbidity and mortality.

Who is "at risk" of NOAF?

What is known

NOAF in critically ill patients shares many risk factors with AF in general populations, namely advancing age, male sex, hypertension, obesity, genetic predisposition, diabetes mellitus, cardiovascular or chronic lung disease. More specific to ICU populations are risk factors such as high severity of illness, use of vasoactive medications,

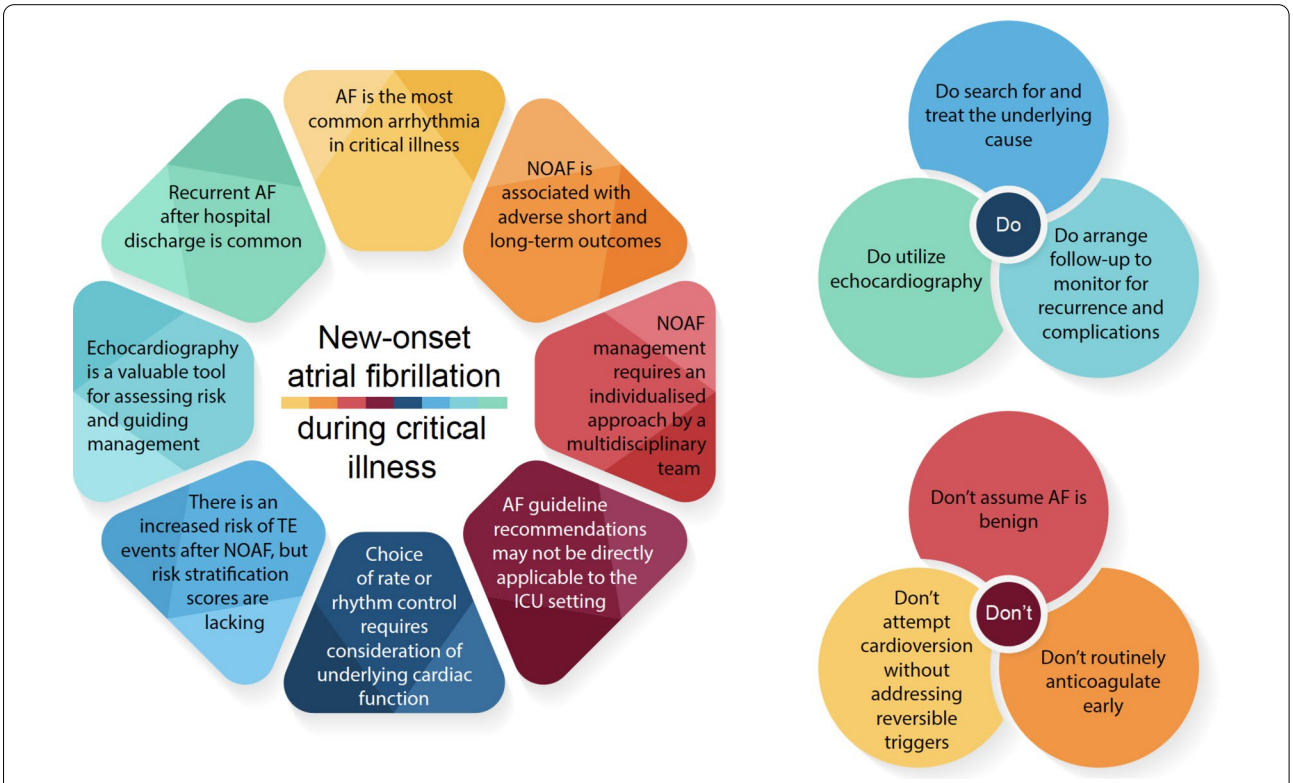

Fig. 1 Key themes (*AF* atrial fibrillation, *NOAF* new onset atrial fibrillation, *TE* thromboembolic, *ICU* intensive care unit)

fluid overload, respiratory failure, sepsis, shock, pulmonary embolism, renal failure, and major surgery [21, 22] (Fig. 2).

Prediction of NOAF before its clinical onset may allow early interventions to prevent occurrence or reduce the duration and severity of the arrhythmia. Several prediction models have been developed specifically for ICU populations that account for combinations of risk factors, clinical findings, and biochemical markers and have demonstrated good discrimination and calibration for the daily prediction of AF. In a study of 1782 patients with sepsis, a score for daily prediction of AF occurrence that included age, obesity, the presence of an immunocompromised state, use of vasopressors, renal failure, serum electrolytes, inspired oxygen, and time since ICU admission yielded good discrimination with a C statistic of 0.81(95% CI 0.79–0.84) and calibration (chi-square 9.38; $p=0.31$) [23]. More recently, models have been developed that account for account for dynamic risk factors of acute or critical conditions such as invasive ventilation, organ dysfunction, and disease progression. A risk tool for predicting AF in critically ill patients integrates a time series of vital signs, blood results, and ventilatory settings. This model demonstrated good performance with an area under the receiver-operating characteristic curve (AUROC) > 0.82 and > 0.91 in ventilated and non-ventilated cohorts respectively [24]. A machine learning model for adult critically ill patients has been derived and validated using the Medical Information Mart for Intensive Care (MIMIC) IV and III databases respectively. It includes dynamic variables such as mechanical ventilation and renal replacement therapy and achieved an AUROC of 0.769 (95% CI 0.756–0.782). Uniquely, high risk was defined as a risk probability of 0.6 and the model was accompanied by a user interface [25]. Similarly, a model with novel features such as a composite score of pre-existing cardiac risk factors achieved an AUROC of 0.820 (95% CI 0.782–0.858) [26].

Knowledge gaps and research opportunities

Over the last decade, there has been an exponential growth of publications using artificial intelligence and machine learning (ML) to predict AF [27]. This likely reflects a general aspiration to use electronic medical records and physiologic waveform data to provide real-time risk assessment that can inform individualized treatment decisions.

Many of the models have been derived and validated in the MIMIC datasets, perhaps limiting the generalizability of these models to other populations. Some have not been validated in an external population. The main limitation of existing models is the lack of prospective use in a clinical setting. While these models provide a probability of developing AF within a certain period of time, the threshold of risk that would prompt a clinician to offer a preventative intervention remains unclear. Further research that considers patient risk, model accuracy, prediction timeframes, and clinician behaviour is needed to facilitate the implementation of clinically useful prediction models.

Can NOAF be prevented?

What is known

Modification of NOAF triggers may prevent development of the arrhythmia. Use of vasoactive medications such as epinephrine and norepinephrine have been associated with the development of NOAF and evidence supports that catecholamine sparing is associated with a reduced incidence of NOAF. In a randomized controlled trial of 776 patients with sepsis treated with catecholamine vasopressors, a higher mean arterial pressure target of 80 to 85 mmHg was associated with higher incidence of newly diagnosed AF compared with a target of 65 to 70 mmHg (6.7% versus 2.8%, $P=0.02$)[28]. Use of vasopressin in combination with catecholamines was associated with lower incidence of NOAF compared with the use of catecholamines alone in a meta-analysis of 3088 patients with shock [Risk Ratio (RR), 0.77 [95% CI, 0.67–0.88] [29].

Knowledge gaps and research opportunities

Besides evidence for catecholamine spearing vasopressors in reducing risk for AF onset, evidence for other preventative pharmacologic interventions is sparse. However, some hypothesis-generating studies and proposed interventions merit further study. A small retrospective study suggested that the administration of low dose hydrocortisone was associated with a 12% reduction in the incidence of NOAF in the acute phase of critical illness [30]. In a propensity score matched retrospective cohort study, the use of dexmedetomidine, a highly selective a2 receptor agonist, was associated with decreased risk of NOAF (hazard ratio (HR) 0.80; 95% CI, 0.71–0.90) [31].

Though both fluid overload and hypovolemia have been identified as a risk factors for NOAF [32], there have been no studies of optimal parenteral fluid management with respect to prevention of NOAF. Restrictive fluid strategies have not been associated with higher mortality in patients with septic shock [33, 34], and evaluation of these strategies on the development of NOAF would be valuable. A recent randomized control trial in a cardiac surgery population found supplementing potassium when serum concentrations fell below 3.5 mEq/L was non-inferior to supplementing when serum concentration fell below 4.5 mEq/L for the prevention of

Table 1 Knowledge gaps, research opportunities, and registered trials for AF in critically ill patients

Knowledge gaps	Research topics	Registered studies in critically ill patients
Risk assessment for the development of NOAF	Use of EMR and physiologic waveform data for development of AI driven risk models	**Intelligent Monitoring to Predict Atrial Fibrillation** (NOTE-AF NCT06600620)
Prevention of NOAF in critical illness	Electrolyte replacements Fluid management Pharmacologic prophylaxis	**Magnesium Prophylaxis for the Prevention of New-Onset Atrial Fibrillation in Critically Ill Patients** (ATOMIC-Mg, NCT05829317)
Optimal pharmacologic agents Rate versus rhythm control Target heart rate Timing of therapeutic interventions	Comparative effectiveness of amiodarone, beta blockers, digoxin, propafenone, etc Use of antiarrhythmics to improve cardioversion success and maintenance	**Magnesium Versus Amiodarone in Atrial Fibrillation in Critical Care** (MAGNAM, NCT05287191**)** **A randomised controlled trial to investigate clinical and cost effectiveness of Amiodarone vs Beta Blockade for new onset atrial fibRillation in icU—a Pragmatic sTudy** (ABBRUPT, ISRCTN59775011) **Rate, Rhythm or Risk Control for New-onset Supraventricular Arrhythmia During Septic Shock: a Randomized Controlled Trial** (CAFS, NCT04844801)
Thromboembolic risk Assessment Timing of anticoagulation Optimal anticoagulant	Development and validation of risk assessment scores Evaluation of DOACs in long-term management of critically ill patients	
Echocardiography Timing of echocardiography and frequency of reassessment Use of TOE to evaluate for LA/LAA thrombus Specific measurements to assess risk for AF recurrence	Use of POCUS for bedside assessment Use of TOE/ cardiac CT/Cardiac MRI for assessment of thromboembolic risk	
Follow-up Frequency and duration of follow-up Best clinical setting for follow-up Prediction of AF recurrence Interventions for prevention of recurrent AF	Risk scoring systems for AF recurrence Wearable technology for follow-up Evaluation of post-ICU clinics Risk factor modification to prevent AF recurrence	**Tracking Atrial Fibrillation After Intensive Care Admission** (TrAFFIC, NCT05229211) **The Short and Long-term Cardiovascular Consequences of Critical Illness: The C3 Study (**C3, NCT04545437) **Post Intensive Care Unit Atrial Fibrillation (PIAF, NCT05860894)**

Abbreviations: *AF* atrial fibrillation, *NOAF* new onset atrial fibrillation, *EMR* electronic medical record, *AI* artificial intelligence, *DOAC* direct oral anticoagulant, *TOE* transesophageal echocardiography, *POCUS* point-of-care ultrasound, *LA/LAA* left atrial/left atrial appendage, *CT* computed tomography, *MRI* magnetic resonance imaging

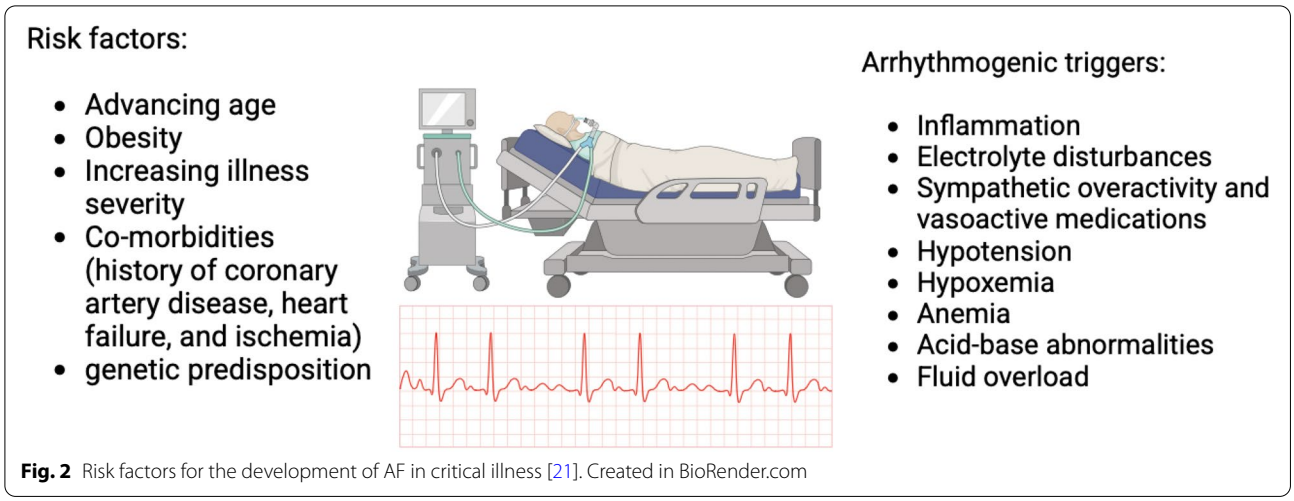

Fig. 2 Risk factors for the development of AF in critical illness [21]. Created in BioRender.com

post-operative AF [35]. Although electrolyte supplementation is also frequently employed by ICU clinicians for the prevention of NOAF [18], there is limited evidence to support supplementation of electrolytes to high-normal serum levels in critically ill patients [36, 37].

Management of AF in the ICU

Both pre-existing AF and NOAF benefit from treatment of the underlying condition and correction of reversible factors such as electrolyte abnormalities, fluid balance, acidosis, and adrenergic overstimulation [38]. In many cases of NOAF, treatment of the underlying cause and correction of reversible factors results in spontaneous cardioversion without the need for other pharmacological intervention [39, 40].

Hemodynamically unstable patients

What is known

Electrical cardioversion is recommended in AF patients with acute or worsening hemodynamic instability due to AF [41]. However, the success of direct current cardioversion in critically ill patients is low, with less than 30% converting to sinus rhythm. For those who initially convert, 40 to 60% will have AF recurrence [38, 42] (Fig. 3). The rate of successful electrical cardioversion is worse in critically ill patients with pre-existing AF, with only 25% converting to sinus rhythm [2].

Current guidelines suggest intravenous amiodarone, digoxin, esmolol, or landiolol for patients with AF who have hemodynamic instability to achieve acute control of heart rate [41]. Of these, amiodarone is the most commonly used medication to manage NOAF in critical illness [18, 19]. Observational data have shown the effectiveness of amiodarone to restore normal sinus rhythm varies greatly from 18 to 94% but may be improved with addition of intravenous magnesium [43]. Amiodarone is also employed as a rate controlling agent in hemodynamically unstable patients, and though it was slower to achieve heart rate control than beta-blockers, calcium channel blockers, and digoxin, no difference in heart rate was found at 6 h in a retrospective cohort study of critically ill patients with sepsis [44].

Several studies in both surgical and non-surgical critically ill patients have examined the use of the highly beta-1 selective, ultra-short acting beta-blocker landiolol, and have demonstrated rapid heart rate control without significant impact on blood pressure [45]. Moreover, treatment with landiolol compared with placebo in patients with septic shock and AF resulted in higher rates of achieving and maintaining a target heart rate (65.4% versus 29.2%, percentage difference 36.2 [95% CI 8.5 to 57.1]) without increased vasopressor requirements [46]. Esmolol has demonstrated superior rate control to amiodarone (64% rate control with esmolol vs. 25% with amiodarone at 40 min) with similar decreases in blood pressure [47].

Digoxin therapy has been shown to be inferior to amiodarone for rate control (adjusted hazard ratio 0.56, [95% CI 0.34–0.92]), it was found to be non-inferior for rhythm control with fewer episodes of bradycardia and hypotension [48, 49], and may be considered in patients with left ventricular (LV) systolic dysfunction or decompensated heart failure [7].

Propafenone was compared to amiodarone in patients with septic shock, NOAF, and normal-to-moderately reduced LV systolic function. The study demonstrated a high cardioversion rate (72.8% versus 67.3%) at 24 h, faster achievement of sinus rhythm (3.7 h vs 7.3 h), and fewer recurrences (52% versus 76%) in patients treated with propafenone compared to amiodarone respectively

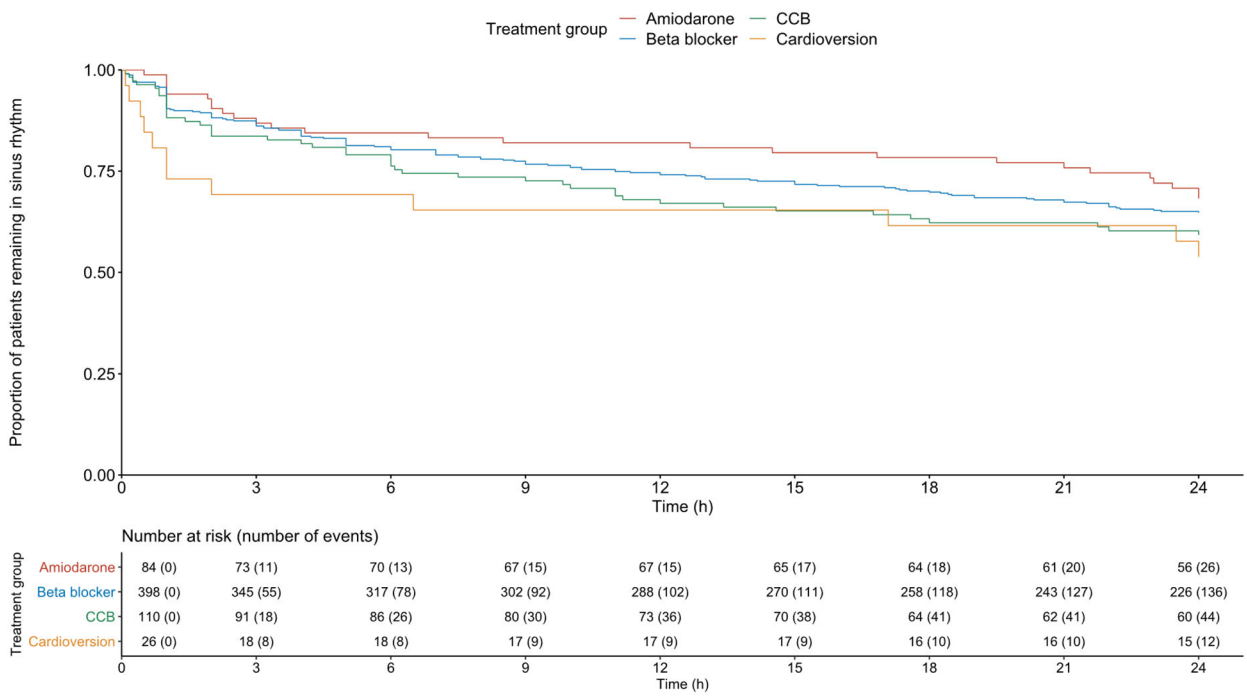

Fig. 3 Kaplan–Meier curves for each treatment of time from achieving rhythm control to reversion to AF: MIMIC-III database (Reproduced with permission from Bedford JP, Johnson A, Redfern O, Gerry S, Doidge J, Harrison D, Rajappan K, Rowan K, Young JD, Mouncey P, Watkinson PJ (2022) Comparative effectiveness of common treatments for new-onset atrial fibrillation within the ICU: Accounting for physiological status. J Crit Care 67:149–156. https://doi.org/10.1016/j.jcrc.2021.11.005

[50]. Patients on propafenone with a non-dilated left atrium (LA) (i.e. LA volume < 40 ml/m^2) presented more frequently with sinus rhythm at 24 h from the start of a supraventricular arrhythmia and had mortality benefit at one year (HR 0.6; 95% CI 0.4–0.9) than those treated with amiodarone. Patients with a dilated LA had earlier rhythm control with amiodarone, and improved mortality at one month (HR 3.6; 95% CI 1.03–12.5). However, there was no difference in long-term mortality at one year [51].

Use of phenylephrine in critically ill patients with septic shock was associated with moderately lower heart rate compared to use of norepinephrine at 1 (−4 beats/min; 95% CI, −6 to −1; $P < 0.001$) and 6 h (−4 beats/min; 95% CI, −6 to −1; $P = 0.004$) in a retrospective cohort study, without differences in secondary outcomes such as conversion to sinus rhythm, vasopressor duration, length of stay, or death [52].

Hemodynamically stable patients

What is known

In the acute setting beta-blockers, diltiazem, verapamil, or digoxin are recommended for patients with AF and a left ventricular ejection fraction (LVEF) > 40% for rate control [41]. Retrospective studies suggest efficacy and mortality benefits with use of beta-blockade for NOAF in critically ill patients. Amiodarone is commonly used for hemodynamically stable patients, however time to ventricular rate control and conversion to sinus rhythm with beta-blockade were comparable with amiodarone [48, 53]. A 38,159-patient analysis of the MIMIC III database indicated an improved 90-day mortality among patients receiving beta-blockers (HR 0.59, 95% CI 0.53–0.65, $p < 0.001$). Amiodarone was associated with higher mortality (HR 1.16, 95% CI 1.05–1.29, $p = 0.004$) [54]. There was no information about the clinical application of each treatment and whether amiodarone was used in patients with higher inotrope and vasopressor use—an important potential bias [53].

A small randomized controlled trial suggested that diltiazem may provide better rate control when a 25 mg bolus followed by an infusion of 20 mg/hr was used compared with amiodarone, prescribed as either a 300 mg bolus, or a 300 mg bolus followed by an infusion of 45 mg/hr of amiodarone ($p = 0.0001$ group 1 vs. group 3, $p = 0.0001$; $p = 0.0001$ group 1 vs. group 2, $p = 0.001$) but was discontinued more frequently due to hypotension [55]. Calcium channel blockers were associated with inferior rhythm control in an analysis of an American database [48].

Knowledge gaps and research opportunities

There is uncertainty regarding the optimal timing of therapy. A wait-and-see approach for spontaneous conversion to sinus rhythm within 48 h of AF onset has been recommended for patients without hemodynamic compromise as an alternative to immediate cardioversion [41]. This delayed treatment strategy has not been evaluated in a critically ill population, and while 95% of ICU clinicians taking part in an international survey responded that observation would be their first line therapy for NOAF in a stable patient, 73% of respondents stated they would provide an intervention within 48 h [19]. In another international survey, 47.8% of participants responded that a heart rate of 120–139 beats per minute would prompt intervention, regardless of duration [18].

It is uncertain whether rhythm or rate control should be the main aim in the ICU management of NOAF in both hemodynamically stable and unstable patients. Regardless of strategy, most patients who survive to ICU discharge will eventually convert to sinus rhythm, with 18% remaining in AF at discharge [38]. The limited evidence outlined above indicates that a one-size-fits-all approach may not be prudent, and individualized treatment based on cardiac function and clinical circumstance is needed. When a rate control strategy is chosen, it is unclear what the optimal target heart rate should be. While lenient heart rate control with a resting heart rate of < 110 beats per minute has been suggested as a target for the non-critically ill population [41] this target may not be appropriate for critically ill patients who rely on heart rate to improve cardiac output.

The causal relationship between AF and poor short- and long-term outcomes in critically ill patients is still unclear. Future study of the impact of treatment choices during critical illness on long-term outcomes will provide important information on this topic.

Risk of thromboembolic events and anticoagulation

What is known

A feared complication of AF during critical illness is the increased short-term risk of thromboembolic events. This increased risk likely results from the mechanical consequences of the fibrillating atria with turbulent blood flow, atrial blood stasis, and activation of the coagulation cascade [56], coupled with multi-comorbidity, immobilization due to critical illness, systemic inflammation, polypharmacy, and invasive procedures [57]. Rates of in-hospital stroke for patients with NOAF have been reported from 2.4% to 7.3%, up to four-fold higher than in patients without NOAF [10, 11]. Similarly, stroke, limb ischemia, intestinal ischemia, hepatic and renal infarcts, and left atrial appendage thrombi have been reported at rates of up to 9.0% at 90-days after ICU admission for patients with NOAF [4, 58, 59].

Knowledge gaps and research opportunities

The question of anticoagulation is one of the most troubling for the ICU clinician. Guidelines now recommend *consideration* of long-term oral anticoagulation in suitable patients with trigger-induced AF at elevated thromboembolic risk [41] but do not offer guidance as to who high risk patients are, when anticoagulation should be initiated, or what medication should be used.

Determining which patients are at risk of a thromboembolic event is a challenge as commonly used thromboembolic risk scoring systems have not shown the ability to discriminate patients who will or will not have a stroke during or after critical illness induced NOAF. A retrospective cohort study of 38,582 patients found the ability of the CHA_2DS_2-VASc (Congestive heart failure, hypertension, age, diabetes, stroke/transient ischaemic attack, vascular disease, age, sex category) score to predict ischaemic stroke during sepsis was poor with a C-statistic of 0.526 [60]. Studies assessing risk scores for AF-associated thromboembolism derived in non-critically ill patients during acute and critical illness have shown poor predictive validity for both short [61] and long-term [62] thromboembolic events, and the CHA_2DS_2-VASc score may overestimate the risk of 1-year stroke for sepsis survivors [61]. In a prospective observational study of 108 patients the $CHADS_2$ score had a C-statistic of 0.7, however this was based on only twelve thromboembolic events [63]. Current guidelines recommend the CHA_2DS_2-VA score for risk assessment in non-critically ill patients; however, this modified score has not been validated in critically ill patients. The poor performance of these scores may be due to their exclusive use of previous diagnoses, comorbidities, and demographics, without consideration of dynamic variables such as rapidly changing cardiac function, adrenergic stimulation, and exposure to vasoactive drugs encountered during critical illness. Development and validation of scoring systems able to predict critically ill patients at risk for thromboembolic events in the short and long-term are vitally needed to guide management in this population.

Even if there were validated scores to identify high risk patients, additional challenges remain. The optimal timing for initiation of treatment is unknown. The benefits of anticoagulation during the acute phase of illness for prevention of thromboembolic events has not been demonstrated. A post-hoc analysis of the AFTER-ICU study did not show any benefit of early

anticoagulation within 48 h of AF onset on a composite outcome of in hospital mortality and ischaemic stroke (HR 0.77; 95% CI 0.47–1.23) compared with anticoagulation after 48 h, without a statistically important difference in bleeding complications [64].

It is unclear if direct oral anticoagulants (DOACs) improve outcomes of critically ill patients compared to warfarin, a medication commonly used in previous anticoagulation studies, but challenging to prescribe due to the need for frequent monitoring, and the time and titration needed to achieve and maintain a therapeutic range. In one small retrospective study of 115 patients with sepsis it was noted that anticoagulated patients treated with unfractionated heparin or warfarin were within therapeutic range less than 50% of their time in the ICU [65], exposing them to bleeding risk without protecting from thromboembolic events. A retrospective cohort study of 82,748 patients discharged after sepsis hospitalization had the unexpected finding that collecting an oral anticoagulation prescription was associated with higher 1-year adjusted risk of ischaemic stroke and transient ischaemic attack (5.69% vs 2.32%) without a significant difference in major bleeding [61]; Warfarin was prescribed in 82% of patients. Similarly, a retrospective cohort of 2304 patients with NOAF and acute coronary syndromes, acute pulmonary disease, or sepsis found no association between anticoagulation and the incidence of ischaemic stroke within three years of follow up (OR 1.22; 95% CI 0.65–2.27); 97% of patients were treated with warfarin. DOACs have an improved safety profile [66] and are recommended over vitamin K agonists such as warfarin in the current guidelines for prevention of ischaemic stroke and thromboembolism in most non-critically ill populations without any specific valvular heart diseases [41, 67]. Studies evaluating DOACs in the long-term management of NOAF in critical illness are needed, with specific focus on risk stratification, monitoring, and timing of implementation.

Use of echocardiography

What is known

Echocardiography has become the imaging modality of choice to evaluate hemodynamically unstable patients in the ICU and guidelines recommend transthoracic echocardiography to guide treatment decisions [41, 68] (Table 2). Echocardiography is a valuable tool for the prediction of both the development of NOAF and the burden of NOAF in critically ill patients. A prospective study of critically ill patients found the development of NOAF was associated with an ejection fraction less than 35% ($P=0.02$), left atrial (LA) dilatation ($P=0.01$), and diastolic dysfunction ($P=0.02$) [32]. Analysis of an Australian ICU cohort found the only independent predictor of AF burden amongst patients with NOAF was LA area, with patients with a high burden of AF having an LA area of 24.4 ± 6.8 cm^2 compared to patients with a low burden of AF having an LA area of 21.3 ± 5.5 cm^2 [69].

The assessment of ventricular contractility can help to avoid administering cardiac depressant medications in patients with a reduced LVEF [41]. Choice of rate or rhythm control strategies may be further guided by echocardiography findings; A LV relaxation disorder and pseudonormal LV filling are more dependent on the atrial kick and would potentially benefit from an attempt at cardioversion rather than rate control [41]. Altered LV end-diastolic pressure may contribute to unsuccessful rhythm control either due to its elevation [70] or due to hypovolemia burdened with excess of endogenous or exogenous catecholamines, warranting correction of reversible causes [32]. Assessment of LA size and function to detect chronic atrial remodeling may guide clinical decision to either restore sinus rhythm or to control heart rate with cardioversion, recognizing that sinus rhythm is less likely to be maintained in patients with large atria or altered atrial function [68, 70].

Echocardiography may be helpful when considering anticoagulation. Transoesophageal echocardiography (TOE) carries advantages in visualisation of the atria and in particular the left atrial appendage (LAA) to rule out clots and to determine atrial flow velocities. The risk of intracardiac thrombus formation can be stratified according to the size of the atria, presence of valve disease, and LV systolic function. For example, a patient in AF with normal echocardiography findings has a 1.5% risk of intracardiac thrombus formation; this rises to 20% in a patient with dilated LA, reduced LV systolic function and an absence of mitral regurgitation [71].

Knowledge gaps and research opportunities

The optimal timing of echocardiographic assessments is unclear as structural changes are dynamic in the acute phase of critical illness and may change due to resuscitation, stabilization, and treatment of the primary illness. The optimal timing of TOE for identification of left atrial appendage thrombus is also uncertain. In non-critically ill populations TOE is recommended prior to cardioversion if AF duration is longer than 24 h [41]. In a pilot study of 94 patients who had at least 6 h of AF or AF that recurred more than twice per day (> 30 s) a TOE was performed within 48 h of NOAF onset and again 48 to 72 h after the first TOE. There was no evidence of LA/LAA thrombus on initial scans. One LAA thrombus was found on the follow-up TOE, and a second was found 17 days later on TOE during workup for an ischaemic stroke [59].

Table 2 Echocardiographic parameters that may assist in prediction and management of AF

Application	Echocardiographic parameters
Prediction of new-onset AF	Increased LA area [69]
	LA dilation [32]
	Enlarged LA end-systolic diameter ≥ 46 mm at arrhythmia onset [68]
	Reduced LVEF < 0.35 [32]
	Diastolic dysfunction [32]
Prediction of AF burden	High burden – LA area > 24.4 ± 6.8 cm^2
	Low burden—LA area of 21.3 ± 5.5 cm^2 [69]
Prediction of recurrent AF	*After ICU cardioversion:*
	Decreased LA emptying fraction < 38.4% at 4 h post cardioversion [68]
	Decreased transmitral A wave velocity–time-integral < 6.8 cm at 4 h post cardioversion [68]
	After discharge:
	Elevated systolic pulmonary artery pressure ≥ 51 mmHg at 4 h post cardioversion [68]
	Increased LA area [73]
	Increased LA volume [72]
Prediction of maintenance of normal sinus rhythm after cardioversion	LA emptying fraction > 44% at 4 h post cardioversion [68]
	A wave velocity–time-interval > 8.65 cm at 4 h post cardioversion [68]
Choice of antiarrhythmic agent	Avoid administration of cardiac depressant medications like vernakalant, calcium channel blockers and 1C class agents in patients with a reduced ejection fraction < 40% [41]
Predicting increased risk of intracardiac thrombus formation	LA dilation [71]
	Reduced LV systolic function [71]
	Absence of mitral regurgitation [71]

Abbreviations: *AF* atrial fibrillation. *LA* left atrial, *LV* left ventricular, *EF* ejection fraction

TOE is a valuable tool for evaluation of LA/LAA thrombus, but optimal timing needs to be assessed.

Echocardiography may provide additional support in the prediction of recurrent AF with implications for further follow-up care. Studies have demonstrated that increased LA size is an important predictor of recurrent AF [72, 73], however, specific cut-offs of the 2D and Doppler parameters for risk prediction are unknown.

Short and long-term follow-up and management

What is known

Recurrent AF after hospital discharge in patients with acute AF during critical illness is common. Studies have demonstrated that patients with NOAF during critical illness have up to 5 times the risk of developing chronic AF compared to those who do not develop AF during their critical illness [12, 17]. Individuals with newly-diagnosed AF during sepsis had recurrence of AF fourfold to sixfold higher than their counterparts with sepsis without AF [17]. Similarly, in patients who developed transient AF during hospitalization for non-cardiac surgery or medical illness, 32% were found to have recurrent AF within one year [61]. The substantial risk of recurrent AF, heart failure, and stroke after acute AF hospitalizations, has prompted recommendations for close post-discharge follow up, ranging from opportunistic screening to cardiology referral [7, 67, 74]. NOAF during ICU admission is often under-recognised and/or under-reported, resulting in loss of opportunity to screen patients and modify risk factors [2, 73].

In a prospective observational cohort of 309 patients with critical illness associated NOAF surviving to hospital discharge, recurrent AF after discharge was identified in 31.9% of patients after median follow up of 413 days. On multivariable analysis, AF burden during ICU stay was associated with recurrent AF after discharge (OR 15.0 [2.8—81.7], p = 0.002), with 63% of patients in the highest AF burden quartile (> 25% of ICU stay) found to have recurrent AF during follow-up [73]. A risk prediction model for recurrent AF after sepsis was improved when intra-sepsis factors such as severity of illness scores, infection type, and extreme laboratory values were added to pre-sepsis factors compared to pre-sepsis cardiovascular risk prediction alone, suggesting a contribution of concurrent events to the increased risk of AF recurrence [75].

Knowledge gaps and research opportunities

There are no clear recommendations for frequency or means of screening discharged patients who experienced NOAF during critical illness.

Lifestyle and risk factor modification has been extensively studied for AF prevention and management in the general population. However, no studies in critical illness survivors focused on weight loss, exercise training, minimization or cessation of alcohol consumption, smoking cessation, and treatment of hypertension, but may provide therapeutic targets to prevent AF recurrence [67]. The relation of social drivers of health to NOAF in critical illness management, particularly after discharge merits further investigation [76].

It is important to recognize that a significant number of patients discharged from the ICU develop post-intensive care syndrome (PICS) which manifests as new or worsening physical, psychological, or cognitive impairments [77]. Studying the role of post-ICU clinics for the follow-up of critical illness survivors who had AF may allow tailored interventions such as physical rehabilitation and psychosocial programs to reduce the risk of AF recurrence, manage AF if it recurs, and improve long-term outcomes.

Conclusions

AF is the most common arrhythmia in critically ill patients and is associated with worse short- and long-term outcomes. A heterogeneous patient population and a complex and multifactorial pathophysiology of illness make management of AF challenging. Significant knowledge gaps prevent optimal management of patients with AF during their critical illness, and important questions remain unanswered, namely if AF is an epiphenomenon of the severity of critical illness or causal in the poor outcomes associated with its development. The duration of AF that is clinically important and what impact interventions within the ICU have on long-term outcomes have yet to be elucidated. Well-designed studies that carefully consider causal relationships, focus on clinically and patient-important outcomes [78], and evaluate the full duration of the patient's journey are needed [79]. Also needed is an individualized approach to management of AF in critically ill patients by a multidisciplinary team, tailored to the patient's baseline cardiac function, type and degree of critical illness, risk for long-term outcomes, and personal preferences. Attentive communication with patients, collaboration between clinicians at all stages of a patient's illness and recovery, and attention to the specific needs of ICU survivors may improve outcomes for critically ill patients with AF.

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Declarations

Conflicts of interest

The other authors have no conflicts of interest to report.

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