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AHA SCIENTIFIC STATEMENT

Atrial Fibrillation Occurring During Acute Hospitalization: A Scientific Statement From the American Heart Association

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ABSTRACT: Acute atrial fibrillation is defined as atrial fibrillation detected in the setting of acute care or acute illness; atrial fibrillation may be detected or managed for the first time during acute hospitalization for another condition. Atrial fibrillation after cardiothoracic surgery is a distinct type of acute atrial fibrillation. Acute atrial fibrillation is associated with high risk of long-term atrial fibrillation recurrence, warranting clinical attention during acute hospitalization and over long-term follow-up. A framework of substrates and triggers can be useful for evaluating and managing acute atrial fibrillation. Acute management requires a multipronged approach with interdisciplinary care collaboration, tailoring treatments to the patient's underlying substrate and acute condition. Key components of acute management include identification and treatment of triggers, selection and implementation of rate/rhythm control, and management of anticoagulation. Acute rate or rhythm control strategy should be individualized with consideration of the patient's capacity to tolerate rapid rates or atrioventricular dyssynchrony, and the patient's ability to tolerate the risk of the therapeutic strategy. Given the high risks of atrial fibrillation recurrence in patients with acute atrial fibrillation, clinical follow-up and heart rhythm monitoring are warranted. Long-term management is guided by patient substrate, with implications for intensity of heart rhythm monitoring, anticoagulation, and considerations for rhythm management strategies. Overall management of acute atrial fibrillation addresses substrates and triggers. The 3As of acute management are acute triggers, atrial fibrillation rate/rhythm management, and anticoagulation. The 2As and 2Ms of long-term management include monitoring of heart rhythm and modification of lifestyle and risk factors, in addition to considerations for atrial fibrillation rate/rhythm management and anticoagulation. Several gaps in knowledge related to acute atrial fibrillation exist and warrant future research.

Key Words: AHA Scientific Statements ■ atrial fibrillation ■ critical illness ■ hospitalization ■ postoperative period

Atrial fibrillation (AF) can manifest in a broad range of acute medical and surgical conditions. Although previously postulated as transient and isolated events, accumulating evidence suggests that AF detected in the setting of acute care or acute illness is associated with a high risk of long-term AF recurrence,[1–15] warranting attention during acute hospitalization and long-term follow-up as well as the need for specific guidance. Serving as a dedicated expansion on this subject beyond management of AF addressed in existing guidelines,[16–18] this scientific statement will specifically address existing knowledge, practical management considerations, and opportunities for future research on AF that acutely manifests in the setting of acute care or acute illness, including during hospitalization for another condition.

The writing group has reviewed data from randomized clinical trials (RCTs), registries, and observational studies.

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Given the broad reach of this topic, we expect a multidisciplinary audience for this scientific statement, including cardiologists, cardiac electrophysiologists, nursing and allied health professionals, cardiac surgeons, anesthesiologists, surgical specialists, intensivist, hospitalists, internists, emergency department (ED) physicians, neurologists, and pharmacists.

Definitions

In this scientific statement, we introduce the term acute AF. Acute AF is defined as AF detected in an acute care setting or during an acute illness. Acute AF has sometimes been referred to as secondary AF in prior literature. The writing group chose to move away from the term secondary AF because it is often unclear whether AF detected in acute care settings is truly secondary to or attributable to the acute issue and would not have otherwise arisen. In other words, AF might have been present in the individual before the acute illness but not previously diagnosed or detected.

Therefore, this characterization of AF as acute AF pertains to the contextual presentation of AF, that is, AF detected or managed for the first time during acute illness such as during acute hospitalization for another condition. The acute AF may be paroxysmal or persistent. The acute AF may be symptomatically felt by the patient or asymptomatically detected on rhythm monitoring or ECG. The further characterization of acute AF (as paroxysmal/persistent, symptomatic/asymptomatic) is consistent with existing clinical documents.[18,19] General definitions and classifications of AF were provided in the 2014 American Heart Association/American College of Cardiology/Heart Rhythm Society guideline on management of AF[16] and in the 2017 Heart Rhythm Society/European Heart Rhythm Association/European Cardiac Arrhythmia Society/Asia Pacific Heart Rhythm Society/Latin American Society of Cardiac Stimulation and Electrophysiology expert consensus statement on catheter and surgical ablation of AF.[19]

Scope of the Issue and Public Health Significance

Acute AF is increasing in incidence[20] and manifests across a range of medical and surgical settings. In medical patients, the incidence ranges from 1% to 46% across different patient cohorts.[1,21–24] In patients with sepsis, the incidence of acute AF varies with the severity of sepsis, with an incidence of 8% to 10% in sepsis, 6% to 22% in severe sepsis, and 23% to 44% in septic shock.[1,21,23,24] Acute AF is associated with longer length of hospitalization,[23–25] greater morbidity[15,21,22,26] and mortality,[21,23,26–28] and high rates of recurrent AF.[1,15]

In the surgical setting, acute AF occurs in the setting of both noncardiac and cardiac surgery. In the context of noncardiac surgery, depending on the type of noncardiac surgery, 3% to 16% of patients develop acute AF,[5] the occurrence of which has been associated with longer hospitalization,[29] greater morbidity[30–33] and mortality,[31–33] higher costs,[29] and subsequent AF recurrence.[5]

AF occurring acutely after cardiac surgery is a specific form of acute AF. Postoperative AF in the setting of cardiac surgery is common, affecting ≈32% of patients after coronary artery bypass grafting, 49% of patients after concomitant coronary artery bypass grafting and aortic valve replacement, and 64% of patients after coronary artery bypass grafting and mitral valve replacement,[34] with high rates of recurrence over subsequent years.[9,11,12] Development of postoperative AF after cardiac surgery is associated with longer hospitalization,[35,36] greater short-term[35,37] and long-term morbidity,[38–40] greater mortality,[35,37–39,41] recurrent hospitalizations,[42] and consequently increased cost of care.[43] Thus, regardless of the hospitalization setting, acute AF is not benign.

SUBSTRATES AND TRIGGERS OF ACUTE AF

A conceptual model incorporating considerations of both substrates and triggers can serve as a useful framework for approaching acute AF (Figure 1).

Substrates for acute AF pertain to atrial scars or electrical or structural remodeling.[44] Atrial scars may result from chronic underlying processes[45] such as volume or pressure overload from valvular heart disease, cardiomyopathy, long-standing hypertension, chronic kidney disease, or myopathic states involving atrial myopathy. Substrates for acute AF may also arise from prior cardiac surgeries[44] (as related to scars from atriotomy, cardiopulmonary bypass, prior valvular surgery, maze procedure), thoracic surgeries, or existing pulmonary disease.[45] Additional substrates include pericardial fat pad[46] and pulmonary vein automaticity.[47]

Triggers for acute AF include inflammation, local mechanical stress, oxidative stress, electrolyte imbalance, and shifts in autonomic tone.[44,48] Potential sources of triggers include infection, pericardial effusion and inflammation, long procedural time, hemodynamic shifts, volume loss or overload, intraprocedural and postprocedural pulmonary complications, and medications, including inotropic agents.[45,49,50]

Acute AF can therefore be conceptualized as the provocation of the susceptible substrates by the acute triggers, leading to the manifestation of AF during acute hospitalization. Building on this concept, acute AF may represent previously unrecognized AF or unmasking of an underlying predisposition to AF in the setting of an acute trigger (Figure 2).

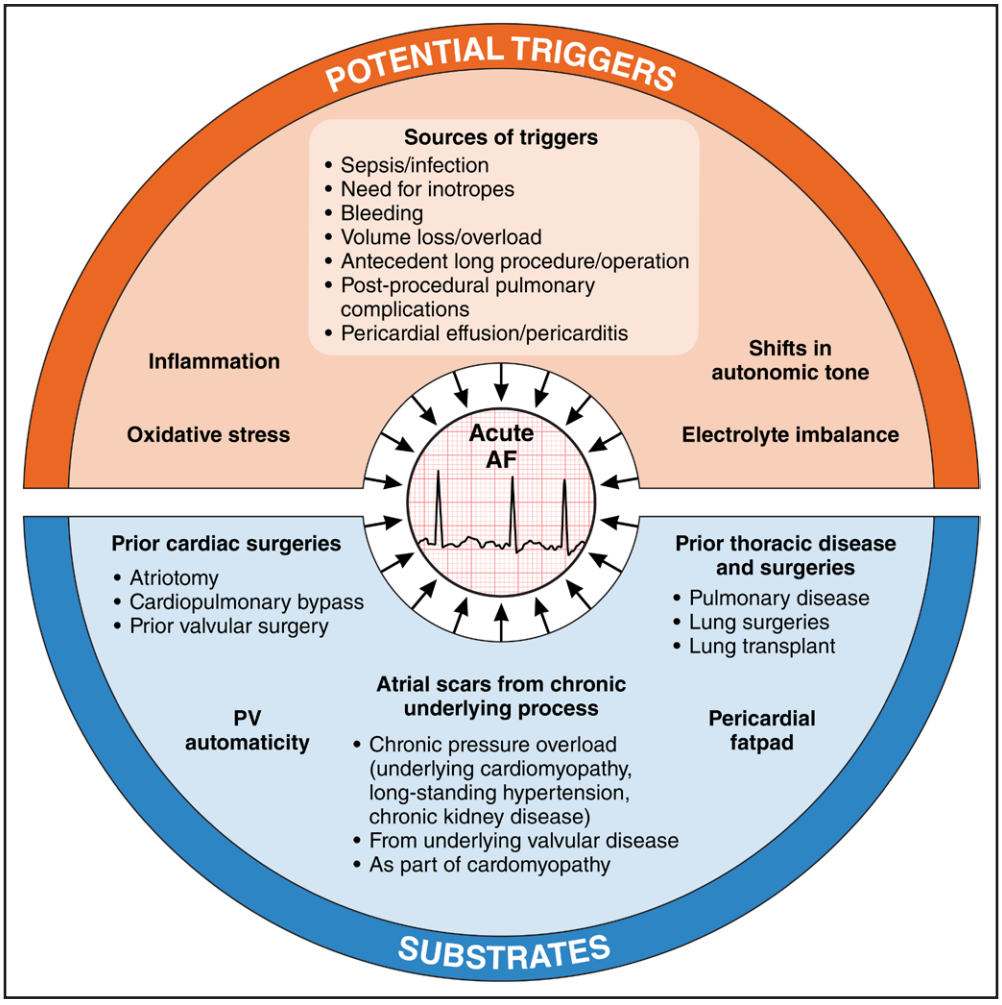

Figure 1. A conceptual model of substrates and triggers of acute AF.
AF indicates atrial fibrillation; and PV, pulmonary vein.

DETECTION OF ACUTE AF

Physical examination may raise suspicion for AF with the identification of an irregularly irregular pulse or auscultated heart rhythm, variable intensity of S_1, or variable pulse amplitude. Electrocardiographic modalities for the detection of AF during acute hospitalization include 12-lead ECG on presentation for acute illness in the ED or during hospitalization or continuous electrocardiographic monitoring with inpatient telemetry. Continuity of electrocardiographic monitoring during the hospital stay influences the detection of acute AF, with continuous monitoring with telemetry being more likely to detect acute AF compared with episodic ECG.[5]

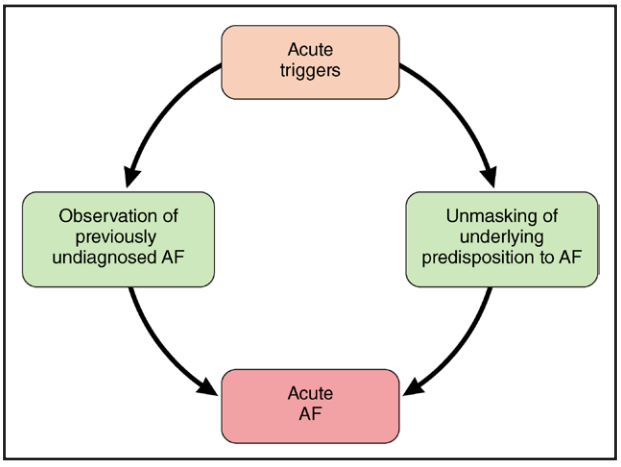

Figure 2. Potential mechanistic pathways of acute AF.
AF indicates atrial fibrillation.

Detection and Consideration for Intensity of Electrocardiographic Monitoring

Patient-based risk scores (such as the CHA_2DS_2-VASc score initially developed for thromboembolism,[51] the ATRIA score initially developed for thromboembolism,[52] the HATCH [hypertension, age, transient ischemic attack or stroke, chronic obstructive pulmonary disease, and heart failure] score initially developed for AF progression,[53] and the POAF score for postoperative AF[54]) have been evaluated as tools to predict acute AF in hospitalized patients.[54–61] Consistent with the

consideration of substrates, the risk scores can be considered a composite marker of vulnerable substrate.

Of these, the CHA_2DS_2-VASc score has been studied most extensively. In medical settings, the CHA_2DS_2-VASc score has correlated with acute AF in patients hospitalized for pneumonia,[57] myocardial infarction,[62] and stroke.[55] In the setting of cardiac surgery,[54,56,58,59,61,63] the CHA_2DS_2-VASc score has outperformed the HATCH or the POAF score.[58]

Additional factors such as type of surgery[5,34] and elevated BNP (brain natriuretic peptide)[64,65] have been associated with acute AF risks. Risk factors for acute AF in the setting of cardiac surgery have also been summarized as an expert consensus table as part of a Society of Cardiovascular Anesthesiologist/European Association of Cardiothoracic Anaesthetists practice advisory.[66]

Patients at higher risk for acute AF may benefit from increased intensity of electrocardiographic monitoring during hospitalization. Refining predictive and risk stratification models for acute AF is an area of active research.[67] Developing a framework to triage the intensity of electrocardiographic monitoring during acute hospitalization is an important area for future research.

ACUTE MANAGEMENT

Overview of Acute Management

Building on the conceptual framework of substrates and triggers, management of acute AF should be tailored to the patient, underlying structural substrates, and contextual triggers. Principal goals of management of AF occurring during acute hospitalization are optimization of hemodynamics, alleviation of patient symptoms, and reduction of short- and long-term risks of thromboembolism.

Acute management of AF occurring during hospitalization requires a multipronged approach. Key dimensions of acute management include identification and treatment of triggers, selection and implementation of rate/rhythm control, and management of anticoagulation.

Acute Triggers: Identification and Treatment

A priority in the management of acute AF is the identification and treatment of potential triggers because rate and rhythm control may be less likely to succeed until the acute illness improves. Potential acute triggers and sources of acute triggers are illustrated in Figure 1. Identification and management of triggers related to a patient's acute AF would benefit from multidisciplinary partnership with the care teams managing the patient's acute hospitalized conditions.

AF Management: Acute Rate and Rhythm Management

Because acute AF may spontaneously convert to sinus rhythm,[68–72] an initial rate control and delayed cardioversion "wait-and-see" approach may be reasonable for hemodynamically stable asymptomatic patients with acute AF while acute triggers are being aggressively treated. However, physiological ramifications of AF can include decreased systemic blood pressure and cardiac output, increased pulmonary vascular pressures, and atrioventricular valve regurgitation.[73] Effects vary among patients and may relate to rapid ventricular rates associated with AF or atrioventricular dyssynchrony.[74] Given the variability in acute conditions and patient comorbidities, acute AF may have variable impact on hemodynamics and patient tolerance.[75,76] In hemodynamically unstable patients, immediate electrical cardioversion is the treatment of choice.

In hemodynamically stable patients, the acute rate or rhythm control strategy should be individualized with consideration of the patient's capacity to tolerate the potential rapid rates or atrioventricular dyssynchrony and the patient's ability to tolerate the risk of rate or rhythm control strategy (Figure 3). Use of a rhythm or rate control strategy depends on the patient and structural substrate, the hemodynamic consequences of AF, and the adequacy of the rate control strategy. Given the risk of acute thromboembolism with acute rhythm control of AF, any decision to proceed with a rhythm control strategy will also need to consider the risk of stroke and the need for adjunctive short- and long-term anticoagulation.

Acute Rate Control

Rate control medications reduce the ventricular rate in AF by increasing the refractoriness of the atrioventricular node.[77] Rate control medications for acute AF management are summarized in Supplemental Table 1. The choice of rate control agent may depend on patient characteristics and comorbidities, with specific contraindications and clinical considerations given in Supplemental Table 1.

Target heart rate for optimal rate control in the setting of acute AF has not been established. Existing data suggest an initial heart rate <110 bpm as a reasonable target for hemodynamically stable outpatients (AFFIRM [Atrial Fibrillation Follow-up Investigation of Rhythm Management])[78] or patients with permanent AF (RACE II [Rate Control Efficacy in Permanent Atrial Fibrillation]).[79] Recent guidelines recommend <110 bpm as a general target for AF rate control, but a stricter target of a resting heart rate <80 bpm for patients with deterioration of left ventricular function, symptoms, concomitant cardiac resynchronization therapy, or diagnosis of tachycardia-mediated cardiomyopathy.[18] In the specific context of AF after cardiac surgery, rate control targeting heart rate <100 bpm is reasonable for asymptomatic patients.[42,80]

Further studies to determine whether a rate control target of <110 bpm may be reasonable for acute AF, with the exception of AF occurring in the setting of cardiac surgery,

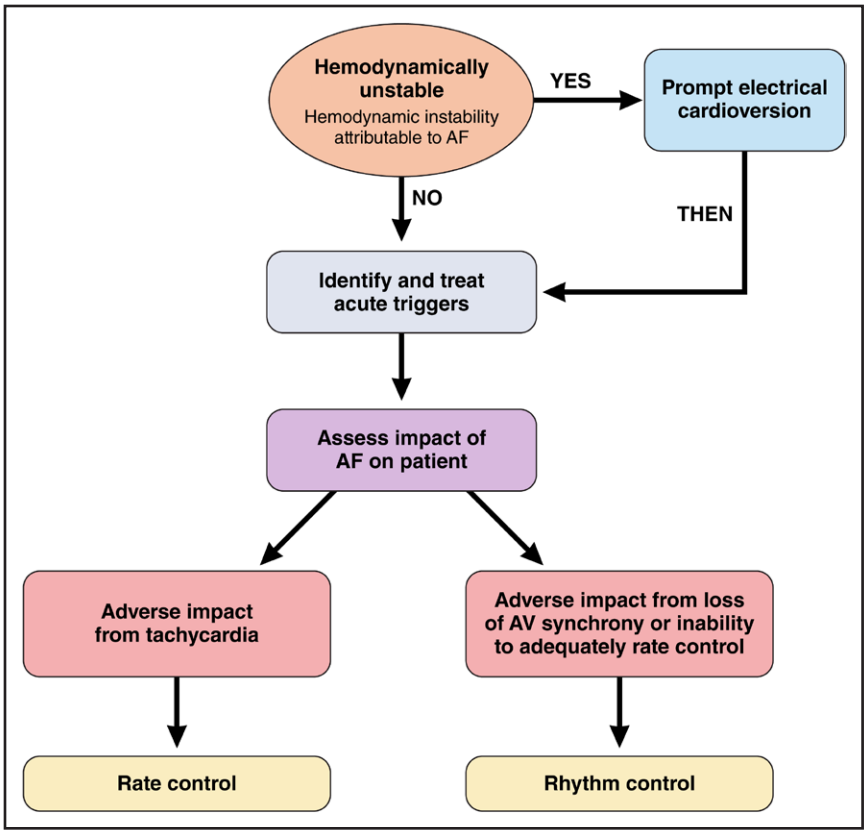

Figure 3. Approach to acute management of triggers and rate vs rhythm control strategy in acute AF. Assessment of the impact of AF on the patient provides a rational approach to pursue rate control or rhythm control or both. Management of acute triggers is important and facilitates the success of rate and rhythm control strategies. AF indicates atrial fibrillation.

ventricular dysfunction, or concomitant cardiac resynchronization therapy, would be helpful. A heart rate <110 bpm may be reasonable to permit some leniency while balancing diastolic filling. However, the specific optimal heart rate for a patient's acute AF may correlate with what may be hemodynamically optimal in the specific setting and may be individualized with dynamic observation of pulse pressure and mean arterial pressure as surrogates for optimal output and perfusion. In the setting of concomitant acute medical or surgical conditions, achievement of optimal heart rate will also include addressing associated acute triggers and ensuring that tachycardia is not a compensatory response to hemodynamic distress.

Acute Rhythm Control

In hemodynamically unstable patients, immediate electrical cardioversion with direct current cardioversion (DCCV) is the treatment of choice.[16,80–82] In hemodynamically stable patients intolerant of atrioventricular dyssynchrony, acute rhythm control can be achieved either with electrical cardioversion or pharmacologically with antiarrhythmic medications.[16,17,80,81,83,84] Rhythm control should also be considered for patients unable to attain clinically adequate rate control despite optimal use of atrioventricular node–blocking agents and identification and management of acute triggers.

Real-world data demonstrate the safety and efficacy for both electrical cardioversion and pharmacological cardioversion in contemporary practice.[83,85,86] In patients with recent onset of AF, the RAFF2 study (Trial of Electrical Versus Pharmacological Cardioversion for RAFF in the ED) demonstrated high success of restoration of sinus rhythm with either an upfront electrical cardioversion strategy or a stepwise strategy with initial pharmacological cardioversion and then DCCV if pharmacological cardioversion was unable to restore sinus rhythm (92% in the DCCV only group versus 96% in the medication/DCCV group; P=0.07).[87]

Antiarrhythmic Medications for Acute Pharmacological Cardioversion or Maintenance of Sinus Rhythm

Antiarrhythmic medications may be used for acute chemical cardioversion or maintenance of sinus rhythm. Choice of antiarrhythmic medication has to be tailored to the individual patient given the unique safety profiles of each agent.[16,17] In the appropriate population, ibutilide,[16,88,89] dofetilide,[16,90] flecainide,[16,91] propafenone,[16,92] amiodarone,[16,93] procainamide,[87] and vernakalant (not approved by the Food and Drug Administration for use in the United States)[18,94] are used for acute pharmacological cardioversion, typically with a more gradual time course for acute conversion to sinus rhythm noted with amiodarone[16,93] or dofetilide.[90] Supplemental Table 2 details the dosing and clinical considerations of antiarrhythmic medications for acute pharmacological cardioversion of AF. In general, the choice of agent depends on the individual situation and underlying clinical substrate such as cardiac and renal function. Ibutilide can be a reasonable choice for patients unable to receive anesthesia in the absence of existing QT prolongation. The most important concern is torsade de pointes, so the

patient must have a normal QTc interval. Ibutilide effects are short lasting (<4 hours); thus, it is not an ideal drug if recurrence of arrhythmia is expected. For patients able to take oral medications and without underlying structural heart disease, either propafenone or flecainide oral bolus is an option, with the advantage that they can be transitioned to ongoing dosing. Amiodarone also has the advantage of transitioning from intravenous to oral form, but cardioversion with amiodarone takes longer than with the aforementioned drugs. Because amiodarone may prolong the QT interval, the use of amiodarone may limit concomitant and subsequent pharmacological options given concerns for potentiation of QT prolongation. Procainamide is also an intravenous option for acute conversion of AF. While using intravenous procainamide, the patient needs to be closely monitored for hypotension, QT prolongation, and proarrhythmia. In the critical ill patient, electrical cardioversion is effective, but relapse is common[95]; similarly, relapse would likely be common after pharmacological cardioversion until the underlying acute illness subsides or adequate drug levels of rhythm control agent have been achieved.

Electrical Cardioversion

Electrical cardioversion with DCCV has a high success rate for restoring sinus rhythm.[96–101] Electrical cardioversion is safe,[85] rapid, and more effective than pharmacological cardioversion alone,[18,98,101] with the tradeoff of the need for sedation with electrical cardioversion.[18,102] A patient's suitability for anesthesia and the ideal anesthesia regimen to support electrical cardioversion benefit from multidisciplinary considerations.

The efficacy of DCCV can be improved by use of biphasic energy[103] and upfront high fixed energy instead of a strategy of energy escalation.[104] Application of electrode pads in anterior-posterior orientation makes physiological sense for optimization of the vector for current delivery. Prior study of patients with persistent AF undergoing DCCV with escalation of energy found anterior-posterior orientation of the electrode vector more successful at restoring sinus rhythm than an anterior-lateral vector.[105] A subsequent study of patients with recently diagnosed AF undergoing DCCV using biphasic upfront 200 J found similar success rates with electrode vectors in either the anterior-posterior or anterior-lateral orientation.[87] These results together suggest that when the energy output is already optimized as biphasic and high energy and the AF is of recent onset, either vector orientation may be suitable.[87] However, when energy output is not high or when the AF is of longer duration, the anterior-posterior orientation may be more effective. In patients with a longer duration of AF, antiarrhythmic medications may also be administered as a pretreatment to facilitate electrical cardioversion.[16,18,85,106] In patients with obesity, the use of paddles, manual pressure augmentation, and further escalation of electrical energy improved the success of electrical cardioversion.[107] In patients with failed initial electrical cardioversion, optimization of vector and energy delivery, manual pressure augmentation, and pretreatment with an antiarrhythmic can facilitate success of repeat electrical cardioversion.

Monitoring During and After Cardioversion

Cardioversion with either an electrical or a pharmacological approach warrants electrocardiographic, hemodynamic, and oximetry monitoring during and after cardioversion.[18,81] Postcardioversion monitoring for pharmacological cardioversion is recommended for a duration of time that is equal to half of the therapeutic half-life of the medication,[81] and for electrical cardioversion with anesthesia, the duration of monitoring after electrical cardioversion would be as per usual postanesthesia monitoring for the extent of anesthesia necessary to support the performed electrical cardioversion. In patients who receive ibutilide, the most important concern is torsade de pointes, which usually occurs within 30 minutes of drug administration; nevertheless, close monitoring is required with a defibrillator readily available for 4 hours or until QT normalizes. Bradycardia is common after cardioversion because of sinus node suppression of automaticity. Bradycardia commonly improves after the patient wakes up and the sinus node recovers while in sinus rhythm; it is uncommon for severe bradycardia to require intervention other than drug dose adjustment in the rare instances that bradycardia fails to resolve. It is important to note that sinus node function can eventually normalize if the patient is able to maintain sinus rhythm.[108]

Anticoagulation During Acute Hospitalization

General Considerations

A decision to initiate anticoagulation needs to balance the risk of thromboembolism against the risk of bleeding and should involve shared decision-making with the patient. General considerations for anticoagulation for patients with AF are based on substrates, with CHA_2DS_2-VASc[51] score of ≥2 for men or ≥3 for women as an accepted indication for anticoagulation[16–18] in the absence of contraindications and significant bleeding risks. In the setting of acute illness, potential prothrombotic and coagulopathic milieu[109–112] and periprocedural hemostasis may also need to be considered. Once a decision for anticoagulation is made, the feasibility and timing for initiation of anticoagulation will likely depend on the context of the acute illness. Considerations in association with specific conditions are discussed further in a subsequent section (Acute Management Considerations in Specific Settings and Populations).

Whether incorporation of markers of prothrombotic potential and coagulopathic states[109,110,112,113] may

further refine the candidacy of patients with acute AF for anticoagulation is an important area for future research. Consistent with newly detected AF in general, there is uncertainty in the optimal threshold of AF burden to initiate anticoagulation.[18,114,115] Future research to better understand the optimal threshold of acute AF burden to initiate anticoagulation would be beneficial and clinically practical.

Thromboembolic Risks of Acute Cardioversion

Thromboembolic risks in the setting of acute cardioversion are attributed to potential existing thrombus, a change in atrial mechanical function with restoration of sinus rhythm, atrial stunning after cardioversion, and a transient prothrombotic state.[116,117] Thromboembolic risks and considerations of anticoagulation apply to both pharmacological cardioversion and electrical cardioversion.[118,119]

Related to anticoagulation management in the setting of acute cardioversion, the prior concept of safe to cardiovert without further assessment or anticoagulation if AF duration has been no more than 48 hours has been challenged.[17,18] Subsequent study found time to cardioversion ≥12 hours to be an independent predictor of thromboembolic complications.[120] More recent data demonstrated that even when the reported duration of AF is no more than 48 hours, the thromboembolic risks were not homogeneously low but rather increased in patients with increasing CHA_2DS_2-VASc score.[121–123] Together, these findings suggest a more nuanced consideration of duration of AF (<12, 12–24, 24-48 hours) and patient-based risk factors (CHA_2DS_2-VASc score) in the pericardioversion management of the patient. When early cardioversion is planned, in the absence of 3 weeks of precardioversion anticoagulation with ascertainment of strict compliance or time in therapeutic window, a transesophageal echocardiogram to exclude existing intracardiac thrombus before cardioversion is recommended by current guidelines.[17,18] Cardiac computed tomography, especially with delayed contrast-enhanced image acquisition protocol, has emerged as an alternative imaging modality to exclude intracardiac thrombus.[124–126] Anticoagulation is recommended to be initiated as soon as possible before AF cardioversion.[17,18] When intracardiac thrombus is excluded, cardioversion may proceed with the patient on therapeutic anticoagulation.[127,128]

After cardioversion, uninterrupted anticoagulation is recommended for 4 weeks,[16–18] the putative period for recovery of mechanical atrial systole. Only in patients with CHA_2DS_2-VASc of 0 in men or 1 in women with very low associated thromboembolic risks[21,122] may omission of such uninterrupted postcardioversion anticoagulation be considered.[17,18] Thus, the decision to cardiovert a hemodynamically stable patient with acute AF should include care team discussions and patient counseling of anticoagulation compliance over the 4 weeks after acute cardioversion without anticipated interruptions, for example, as related to upcoming procedural needs. If interruption of anticoagulation is anticipated in a hemodynamically stable patient with acute AF, a focus on rate control first and deferring cardioversion until no further interruption of anticoagulation is anticipated may be preferable.

Options for Anticoagulation During Acute Hospitalization

There are parenteral and oral options for anticoagulation during acute hospitalization. Dosing and considerations are summarized in Supplemental Tables 3 and 4, respectively. Given rapid time to therapeutic efficacy and ease of continuation, direct oral anticoagulants (DOACs) have been recommended for thromboembolic prophylaxis including in the setting of cardioversion for AF, barring contraindications.[17,18] Post hoc analyses of the pivotal phase III studies comparing DOACs with warfarin for anticoagulation of nonvalvular AF and subsequent dedicated prospective trials specifically related to cardioversion support the efficacy of DOACs for thromboembolic prophylaxis in the setting of cardioversion.[129–136] Strategies to manage potential bleeding and availability and access to reversal or mitigating agents should also be part of the consideration in deciding on the choice of anticoagulant.

Acute Management Considerations in Specific Settings and Populations

In the Setting of the Emergency Department

The ED is frequently the first point of diagnosis and management of acute AF. Subsequent management of acute AF may occur as part of an acute hospitalization or in an observation unit.[137] Emergency presentation of hemodynamically unstable acute AF is managed with immediate DCCV. Assessment of acute AF in hemodynamically stable patients in the ED includes determination of concomitant medical or surgical processes as potential triggers and initiation of multidisciplinary management. The decision for rate or rhythm control mirrors the inpatient setting, and these approaches are not mutually exclusive. Rate control is often initiated early in the ED course; rhythm control may be subsequently undertaken. Initiation of anticoagulation in the ED is safe.[138] Considerations for anticoagulation include not only substrate-based assessment for thromboembolic risks (such as through the use of the CHA_2DS_2-VASc score[51]) but also assessment of active bleeding risks or contraindications.[139] Evaluation of an anticipated need for interruption of anticoagulation, in the near future or as part of necessary procedural management of concomitant processes, may also influence candidacy for acute cardioversion in hemodynamically stable patients with acute AF. Transesophageal echocardiography may be performed to assess for left atrial or appendage

thrombus before cardioversion, as discussed above.[128] This is particularly important for patients without anticoagulant therapy or those uncertain of compliance with anticoagulant or time in target range on warfarin. Antiarrhythmic medications may be administered to facilitate rhythm conversion before DCCV and to maintain sinus rhythm after DCCV.[16,18,85,106]

In Critically Ill Patients

Aggressive management of acute illnesses and prompt treatment of triggers remain the cornerstone of acute AF management in critically ill patients.[2,81] It may be appropriate to wait to directly treat the acute AF until further treatment of the acute illness if the rapid heart rate is a compensatory mechanism for the critical illness.[2] In patient whose acute AF is causing hemodynamic compromise, immediate DCCV is the strategy of choice.[18,82] In the absence of hemodynamic compromise, both rate and rhythm control strategies may be considered.[81] Electrical cardioversion may be successful, but early relapse is common in patients who remain acutely ill.[95] Amiodarone and propafenone have demonstrated efficacy for pharmacological cardioversion in this population.[140] Prior limited studies suggest that for acute AF in the critically ill, metoprolol may provide better rate control compared with diltiazem[141] and that esmolol use may be associated with improved arterial elastance[142] and reduced short-term mortality.[143,144] Although the reduction of in-hospital mortality with β-blockade was no longer evident after multivariable adjustment reflective of more favorable hemodynamic profile before initiation of β-blockade, when feasible, β-blockade remains a reasonable choice for rate control given its demonstrated efficacy in this regard.[145]

Critically ill patients with new-onset AF have a >2-fold higher risk of in-hospital ischemic stroke compared with those without AF.[21] In patients with sepsis, however, CHA_2DS_2-VASc alone poorly predicts the risk for ischemic stroke.[146] Parenteral anticoagulation in patients with acute AF and sepsis did not reduce risks for ischemic stroke[146,147] and was associated with increased clinically significant bleeding in one study.[146] Available evidence does not favor routine acute anticoagulation in patients with sepsis with acute AF. Further research combining risk scores based predominantly on chronic conditions (such as the CHA_2DS_2-VASc risk score) with considerations of the acute coagulopathic[112] and prothrombotic[148] milieu may be useful to better assess the benefits, risks, and optimal selection of critically ill patients with acute AF for acute anticoagulation.

Coronavirus Disease 2019

Similar to critical illness in general, AF in the setting of coronavirus disease 2019 (COVID-19) is related to disease severity.[149,150] The first wave of COVID-19 in particular, however, has been associated with thrombotic events, especially among those with greater illness severity.[151] Preadmission oral anticoagulation in patients with preexisting AF with COVID-19 was associated with a lower likelihood of adverse events during the hospitalization.[152] Broader data on thromboprophylaxis in COVID-19[153–156] may offer additional insights into the consideration of thrombotic states and choices for potential anticoagulation. Whether the propensity for thrombosis or benefits of anticoagulation would remain applicable to the subsequent variants of the COVID-19 pandemic or to individuals who have received vaccines and boosters needs to be further assessed.

Hyperthyroidism

Goals for management of acute AF in the setting of hyperthyroidism include efforts to restore the euthyroid state and, if feasible, β-blockade for rate control.[16] Although hyperthyroidism may induce a hematologically prothrombotic state, with an increase in factors VIII and IX, fibrinogen, von Willebrand factor, and plasminogen activator inhibitor-1,[157] hyperthyroidism is not included in the risk stratification scheme for thromboembolism in patients with AF.[51] Correlation of hyperthyroidism with clinical thromboembolism has been controversial; anticoagulation for patients with thyrotoxicosis and AF is guided by CHA_2DS_2-VASc risk factors in the 2014 American Heart Association/American College of Cardiology/Heart Rhythm Society guideline on the management of AF.[16] Recent data noted increased risks of ischemic stroke and systemic embolism in patients with AF with hyperthyroidism in the first year of AF diagnosis but with a reduction of increased risks with treatment of hyperthyroidism.[158] These nuanced data underscore the importance of treating the hyperthyroidism and may prompt additional consideration for anticoagulation while attempting to restore the euthyroid state during the first year of AF diagnosis.

Stroke

The temporal relationship between AF and stroke is complex.[159–164] Diagnosis of AF during acute hospitalization for stroke may also represent detection of previously unrecognized AF (consistent with one of the outlined pathways of acute AF in Figure 2). Broad consideration of the temporal relationship between AF and stroke is beyond the scope of this scientific statement. However, pertinent to this scientific statement are the observations that a subset of AF can manifest in temporal proximity to stroke events[159,160,164,165] and that sometimes first diagnosis of AF may occur in close temporal proximity to incident stroke,[165] posing implications for clinical management and the timing of anticoagulation. Approximately 6.5% to 15% of strokes occur in hospitalized patients, and stroke is more common in the perioperative setting and in patients with high-risk conditions such as acute coronary syndromes or prothrombotic states.[166] Strokes

associated with AF more commonly involve the middle cerebral artery territories (much less commonly the vertebrobasilar territories) and may be amenable to mechanical thrombectomy.[167] In general, the risk of early re-embolization is low.[168] Urgent anticoagulation with the goal of preventing early recurrent stroke or preventing neurological deterioration is not recommended.[169] Because of the low risk of early recurrent stroke and the risk of worsening hemorrhagic transformation, it is reasonable to delay oral anticoagulation for 4 to 14 days among those with acute ischemic stroke in the setting of AF.[169] Ongoing RCTs (ELAN [Early Versus Late Initiation of Direct Oral Anticoagulants in Post-Ischaemic Stroke Patients With Atrial Fibrillation; NCT03148457], OPTIMAS [Optimal Timing of Anticoagulation After Acute Ischaemic Stroke; NCT03759938], TIMING [Timing of Oral Anticoagulant Therapy in Acute Ischemic Stroke With Atrial Fibrillation; NCT02961348], and START [Optimal Delay Time to Initiate Anticoagulation After Ischemic Stroke in Atrial Fibrillation; NCT03021928]) will specifically assess early versus late initiation of anticoagulation using DOACs in patients with AF-related ischemic stroke. Issues related to the long-term use of oral anticoagulants are addressed in other guidelines.[17,170]

Noncardiac Surgery

The treatment of AF after noncardiac surgery should include the identification and correction of potential triggers and sources of triggers (Figure 1).[171] Of particular importance is the exclusion of bleeding, which would influence the overall management of a patient's acute AF after noncardiac surgery. Adrenergic surge, volume loss, inflammation, and shifts in autonomic tone in the setting of bleeding may contribute as triggers, and tachycardia may be compensatory for acute blood loss. Ongoing bleed would preclude initiation of anticoagulation necessary for rhythm control strategy.

Given that AF after noncardiac surgery frequently spontaneously reverts to sinus rhythm,[172] it may be reasonable to treat AF after noncardiac surgery with a rate control strategy when the tachycardia is not a compensatory response for acute volume loss and underlying triggers have been identified and aggressively treated. A rhythm control strategy may be considered for selected patients who remain symptomatic despite rate control or in whom rapid restoration of sinus rhythm may be preferred given comorbidities such as heart failure or severe ischemia.[173] Anticoagulation indication is balanced by the need for surgical hemostasis. Therefore, safety, feasibility, and the timing of initiation of acute anticoagulation warrant close discussions with the care teams involved in the specific surgery. Multidisciplinary discussion should include individualized considerations of intraprocedural bleeding, adequacy of surgical hemostasis at the end of the surgery, likelihood for rebleed or susceptibility to surgical site bleeding on anticoagulant challenge, and potential ramifications of bleed (particularly debilitating in circumstances such as spinal surgery).

Cardiac Surgery

Prophylaxis for AF After Cardiac Surgery

In recognition of the high prevalence and associated adverse outcomes of postoperative AF in the setting of cardiac surgery,[9,11,12] prophylactic treatments are recommended by society guidelines.[16,18,174] β-Blockade and amiodarone are currently the agents of choice for pharmacological prophylaxis for postoperative AF in the setting of cardiac surgery.[18] Data based on small studies, predominantly demonstrating efficacy of β-blockade in reducing postoperative AF in isolation or in combination with amiodarone,[175–177] have led to preoperative β-blockade as a quality metric.[176] However, preoperative β-blockade within 24 hours of isolated coronary artery bypass surgery as a quality measure has been challenged[176]; a study of 140 000 propensity-matched individuals from the Society of Thoracic Surgeons database found β-blockade to be associated with a small but finite increase in the rate of postoperative AF without a reduction of morbidities or mortality.[178] It is not clear whether evaluation of more gradual administration of β-blockade upstream instead of within the 24-hour perioperative window or the inclusion of patients with concomitant valvular surgery who have even higher risks for postoperative AF may be differentially associated with outcomes. Amiodarone is the most universally accepted antiarrhythmic agent used for the prevention of AF after cardiac surgery. Its efficacy was initially demonstrated in 1997 with a small, double-blind, placebo-controlled, randomized study encompassing 124 patients.[179] Subsequent small studies have consistently shown its efficacy in both oral and intravenous form.[180] Because of its side-effect profile, it is typically reserved for patients at high risk for postoperative AF. Consistent with this, a multinational survey of cardiac anesthesiologists demonstrated limited use of amiodarone as prophylaxis for postoperative AF, citing concerns for risks related to the side-effect profile of amiodarone.[66]

In terms of colchicine, although the AF substudy of the COPPS study (Colchicine for the Prevention of the Postpericardiotomy Syndrome) demonstrated a reduction of postoperative AF and length of stay,[181] leading to inclusion in the 2014 American Heart Association/American College of Cardiology/Heart Rhythm Society guideline as a IIb recommendation,[16] the subsequent COPPS-2 study (Colchicine for Prevention of Postpericardiotomy Syndrome and Postoperative Atrial Fibrillation)[182] and END-AF study (Effect of Colchicine on the Incidence of Atrial Fibrillation in Open Heart Surgery Patients)[183] did not demonstrate statistically significant

reductions of postoperative AF during hospitalization after cardiac surgery, and associated diarrhea led to discontinuation of colchicine in more than half of colchicine recipients.[183] Recent comprehensive guidelines[18] noted data for colchicine as pharmacological prophylaxis for postoperative AF in the setting of cardiac surgery not to be robust. Other agents such as renin-angiotensin system inhibitors, nonsteroidal anti-inflammatory agents, steroids, statins, omega-3 fatty acids in the form of fish oil supplements, calcium channel blockers, digitalis, and angiotensin-converting enzyme inhibitors have been tested in small-scale clinical trials, showing varying efficacy in prophylaxis for AF after cardiac surgery.

Notable updates in this arena include further considerations of pericardiotomy and botulinum toxin for the prophylaxis of AF after cardiac surgery. The recently published prospective, single-center, randomized PALACS study (Posterior Left Pericardiotomy for the Prevention of Atrial Fibrillation After Cardiac Surgery) found posterior left pericardiotomy to be associated with a reduction in the incidence of postoperative AF in selective patients undergoing coronary bypass or aortic valve or aortic surgery without significantly added risks.[184] The phase 2 multicenter, randomized NOVA study (NeurOtoxin [Botulinum Toxin Type A] for the Prevention of Post-Operative Atrial Fibrillation)[185] completed its recruitment in December 2021. Future studies may further assess the clinical utility and implementation feasibility of these potentially promising modalities.

Acute Treatment of AF After Cardiac Surgery

Treating hemodynamically stable, asymptomatic, acute AF after cardiac surgery with rate control as the initial strategy is reasonable[18] given the similar length of hospitalization, morbidities, and mortality with a rate control target of <100 bpm or rhythm maintenance with amiodarone.[42] When acute pharmacological cardioversion is considered, ibutilide[16,89] and vernakalant (not approved by the US Food and Drug Administration for use in the United States)[18,94] have demonstrated specific efficacy in acute pharmacological cardioversion of AF after cardiac surgery.

Special consideration should be given to patients who develop atrial arrhythmias after surgical ablation, which may vary in extent of ablation lesion sets.[186] Management of this specific population would benefit from a heart team approach[187] with close collaboration of the cardiac electrophysiologist and the operating surgeon in further individualizing acute treatment strategies and plans for longer-term[188] follow-up. Acute management strategies will need to take into account potential concomitant sinus node dysfunction and an early period of extensive atrial inflammation.

Acute Anticoagulation Considerations for AF After Cardiac Surgery

Data related to anticoagulation for AF after cardiac surgery are limited. Balancing the risk of stroke in this vulnerable population against the risk of major bleeding in this postsurgical population is key. Retrospective studies have highlighted the association of AF after isolated coronary artery bypass surgery with increased adverse outcomes of ischemic stroke and thromboembolism[35,40] and mortality[35,41] but have shown conflicting results in terms of the potential benefit of early initiation of oral anticoagulation.[40,41] The conflicting results may relate to the nonrandomized nature of oral anticoagulant treatment; recipients of oral anticoagulants may be older, more frequently have a history of heart failure,[40] and have increased risks for both thromboembolic and bleeding complications. Furthermore, although the retrospective studies included populations who had undergone cardioversion,[40,41] the timing of anticoagulation in relation to the cardioversion was unclear.

In the absence of data from dedicated RCTs in this regard, the anticoagulation protocol used in the Cardiothoracic Surgical Trials Network RCT on rate versus rhythm control[42] has frequently been extrapolated for clinical care. The study protocol recommended anticoagulation if patients remained in AF or had recurrent AF 48 hours after study randomization; anticoagulation with warfarin targeting an international normalized ratio of 2 to 3 was recommended, and bridging with low-molecular-weight heparin was allowed.[42] Anticoagulation was recommended to be continued for 60 days unless complications occurred.[42] However, it is important to note that the Cardiothoracic Surgical Trials Network RCT on rate versus rhythm control was focused primarily on rate versus rhythm control and was not powered to detect thromboembolism and stroke. Recognizing this gap in knowledge in clinical practice, the ongoing randomized PACES (Anticoagulation for New-Onset Post-Operative Atrial Fibrillation After CABG study; NCT04045665) will provide specific insights in this regard.

For patients after surgical ablation for AF, the current guideline recommends oral anticoagulation as soon as feasible given endothelial disruption during ablation while balancing the risk of postoperative bleeding.[18] One recent development has been the addition of left atrial appendage ligation at the time of cardiac surgery to reduce the risk of stroke in patients with a history of AF. LAAOS III (Left Atrial Appendage Occlusion Study III) demonstrated that concomitant left atrial appendage occlusion performed during cardiac surgery in patients with prior AF and a high CHA_2DS_2-VASc score (mean, 4.2) continued on long-term oral anticoagulation over follow-up significantly reduced stroke and thromboembolism risk over a follow-up of 3.8 years (4.8% in the occlusion group versus 7.0% in the no-occlusion group; hazard ratio, 0.67 [95% CI, 0.53–0.85; P=0.001]).[189] Because of this study, there has been growing enthusiasm for broader clinical implementation. Although modern techniques

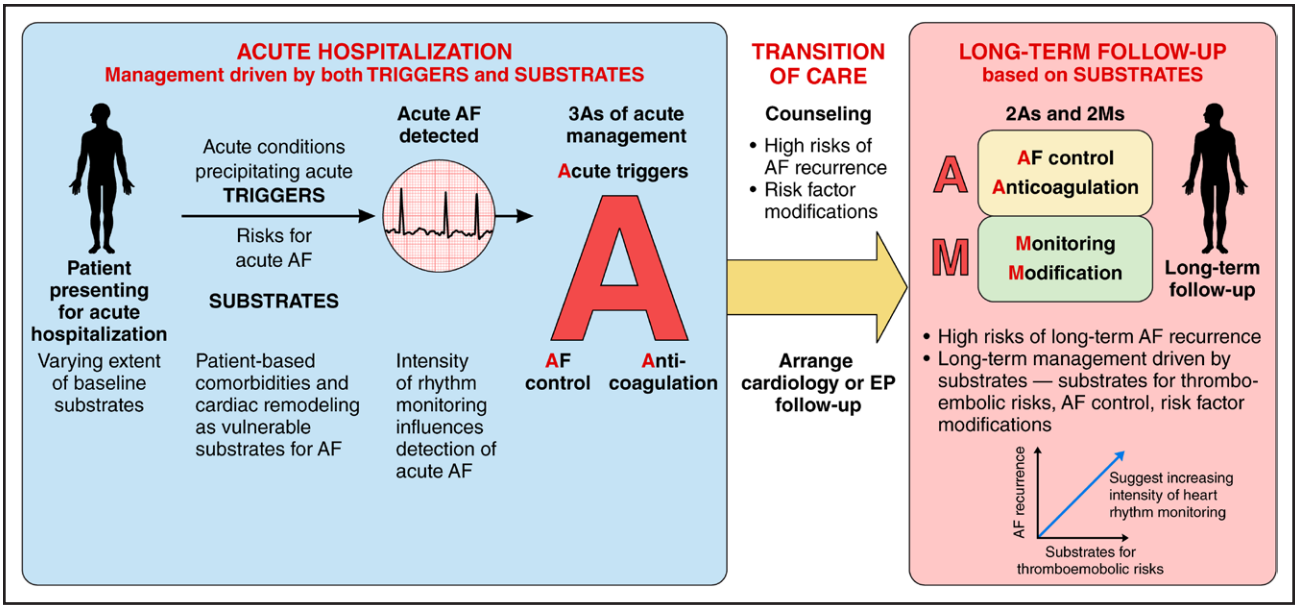

Figure 4. Care pathway for acute AF.
AF indicates atrial fibrillation; and EP, cardiac electrophysiology.

for surgical left atrial appendage have improved the success of closure,[190] given the potential for incomplete ligation,[190–192] management of postoperative AF in patients with reported left atrial appendage closure include formal evaluation of the left atrial appendage with transesophageal echocardiogram or computed tomography to assess for adequacy of closure.

Atrial Flutter

Management of atrial flutter in the acute care setting follows the same principles as AF management. Often, atrial flutter is more difficult than AF to rate control medically, necessitating cardioversion for patients with atrial flutter. Although most studies on electrical cardioversion were performed on patients with AF, the same principles concerning precardioversion and postcardioversion anticoagulation apply (see the Thromboembolic Risks of Acute Cardioversion section). For medical cardioversion of atrial flutter, ibutilide and dofetilide have been shown to be more effective than the class I medications[193–195] or intravenous amiodarone.[196,197] The class I medications can also have the untoward effect of slowing the flutter cycle length, resulting in rapid ventricular rates from 1:1 atrioventricular conduction of atrial flutter if conversion is not achieved.[198,199] Ablation for typical atrial flutter in the acute setting may also be considered given its excellent success rate,[200–202] although patient risk factors and concurrent acute illnesses may necessitate medical optimization before they are considered good ablation candidates. Because up to 50% of patients with atrial flutter can be found to have incident AF after atrial flutter,[203] long-term AF surveillance monitoring is prudent after treatment for atrial flutter.

LONGER-TERM MANAGEMENT

For patients with acute AF, patient counseling about the risk of recurrent AF after the acute illness is resolved and access to follow-up care are important aspects of discharge planning (Figure 4).

Acute AF Recurs Long Term

Patients with acute AF in the setting of acute medical illness[1,2,15] and noncardiac[3–6] and cardiac[8,9,11–14] surgeries have high rates of AF recurrence long term. The 5-year AF recurrence rates were 42% to 68%,[1,4] 39%,[7] and 32% to 76%[4,8–10] in patients with acute AF in the setting of acute medical illness,[1,4] noncardiac surgeries,[7] cardiac surgeries,[8,9,11–14] respectively, with higher detection of recurrence in studies using continuous heart rhythm monitoring.[8–10]

Long-Term Follow-Up Care

Given the notable long-term AF recurrence, patients with acute AF warrant long-term outpatient follow-up evaluation and management. In patients with newly diagnosed AF from the Veterans Health Administration, receipt of cardiology care versus only primary care was associated with a reduction of stroke and death, potentially mediated by early prescription of oral anticoagulant therapy.[204] Clinical follow-up evaluation with the cardiovascular specialist include assessment of family history, associated conditions and risk factors, structural heart disease, thromboembolic risk, and symptoms.[16] Further management of AF, anticoagulation, and considerations for monitoring and guidance on appropriate risk factor modification are key aspects of long-term

management of patients with acute AF (Figure 4). The rise of telehealth has the potential to increase access to cardiovascular specialists for patients in rural and underserved areas. AF diagnosis, evaluation, and management are well suited for telemedicine when paired with mobile technology for the assessment of heart rate and rhythm.[205,206]

Options and Strategies for Heart Rhythm Monitoring

Follow-up heart rhythm monitoring is prudent because recurrent AF may be asymptomatic or may not be reliably identified by symptoms. Heart rhythm monitoring may further inform individualized shared decision-making and guide AF management strategies in conjunction with ongoing cardiovascular follow-up and assessment. Clinical monitoring modalities include 12-lead ECG (seconds), Holter (hours to days), continuous ambulatory monitoring patches (days to weeks), event monitors (multiple weeks), and implantable loop recorders (years).[206] Potential incorporation of direct-to-consumer mobile health technologies into the clinical arena is an area of active research.[206,207]

Different monitoring strategies have been used to identify long-term recurrence of AF in patients who had acute AF during hospitalization. Among observational studies of patients with acute AF after cardiac surgery, higher detection rates of recurrent AF were reported in studies that used continuous longer-term monitoring.[8–10] Although no RCT has specifically compared different monitoring strategies for outpatient follow-up of patients with acute AF during hospitalization, insights may be drawn from trials that compared different monitoring strategies in clinical populations for the purpose of identifying AF. The CRYSTAL-AF RCT (Cryptogenic Stroke and Underlying AF)[207a] and SEARCH-AF RCT (Detection of Atrial Fibrillation After Cardiac Surgery)[208] assessed the comparative detection of an implantable cardiac monitor versus usual outpatient follow-up care in poststroke and post–cardiac surgery patients, respectively, predominantly without in-hospital detection of AF (SEARCH-AF[208] included a small number of patients who had AF lasting <24 hours after cardiac surgery). Both studies demonstrated increased sensitivity for the detection of AF with longer-term continuous monitoring such as with an implantable cardiac monitor compared with usual care.

In the absence of RCTs of monitoring strategies specific to acute AF, we suggest shared decision-making with consideration of patient substrate for AF and thromboembolic risks in the selection of the monitoring strategy. Given the high risk for AF recurrence in patients with acute AF across medical and surgical acute conditions, patients with acute AF with a CHA_2DS_2-VASc score of ≥2 for men or ≥3 for women, an accepted threshold of increased annual risk of stroke, may warrant longer duration of monitoring, and consideration of longer-term continuous monitoring strategy may be reasonable. A conceptual illustration to consider increasing intensity of heart rhythm monitoring in patients with substrates for elevated thromboembolic risks and AF recurrence is shown in Figure 4. The optimal frequency, duration, and modality of long-term monitoring for patients with acute AF remain unclear and need further research to guide care in this population with high long-term AF recurrence risks.

Considerations for Long-Term Anticoagulation

The decision to pursue longer-term anticoagulation is based on patient substrate and thromboembolic risk barring contraindications to anticoagulation. Retrospective registry data suggest similar long-term thromboembolic risks in patients with AF with and without acute triggers precipitating manifestation of AF.[6,209] Given the high rates of long-term AF recurrence after acute AF episodes,[1–13,15] barring significant contraindications, initiation of long-term anticoagulation according to the patient's stroke risk similar to the general approach for long-term anticoagulation in patients with AF may be reasonable. Current guidelines recommend long-term anticoagulation for patients with a CHA_2DS_2-VASc score of ≥2 for men or ≥3 for women, with preference for DOAC over vitamin K antagonist.[17,18] Dosing, pharmacological properties, and additional considerations of the oral anticoagulants are summarized in Supplemental Table 4. For patients who have undergone cardioversion as part of the management of acute AF, anticoagulation should not be interrupted during the 4 weeks after cardioversion due to concerns for thromboembolism and atrial stunning.[17,18] Decisions for further anticoagulation beyond the 4 weeks after cardioversion should be guided by substrate. In patients with high thromboembolic risk (such as those with high CHA_2DS_2-VASc scores or equivalent), continuation of anticoagulation, barring contraindications, may be reasonable because acute AF may be a manifestation of previously undiagnosed and likely recurrent AF.

Currently, for patients with acute AF after cardiac surgery, long-term anticoagulation may be considered according to the anticipated net benefit and informed patient preference. Results from the ongoing prospective, randomized PACES will provide some guidance based on results from randomization to oral anticoagulant plus background antiplatelet therapy (versus antiplatelet only) for the protocol duration of 90 days. For patients who underwent surgical AF ablation, oral anticoagulation as soon as feasible during hospitalization and long-term anticoagulation based on CHA_2DS_2-VASc score are recommended.[18] Follow-up of patients with AF after cardiac surgery with reported left atrial

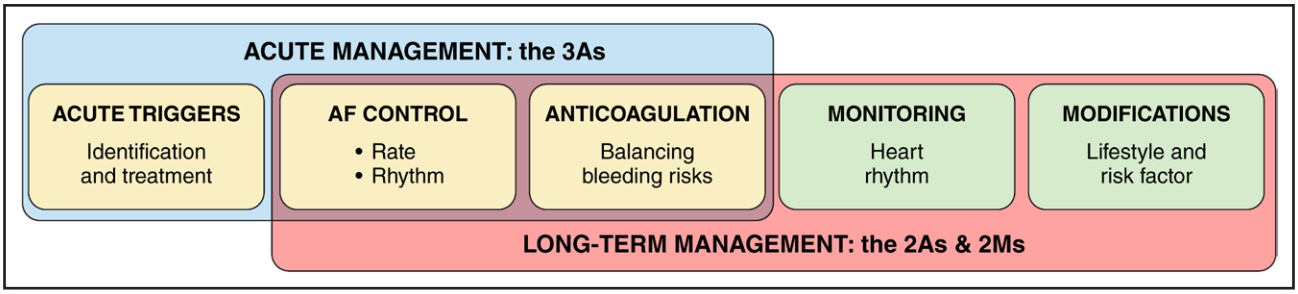

Figure 5. Summary of management of acute AF.
AF indicates atrial fibrillation.

appendage closure warrants evaluation of the left atrial appendage with transesophageal echocardiogram or computed tomography to assess for adequacy of closure and further consideration for anticoagulation based on adequacy of closure as well as additional assessment of the patient's thromboembolic risk.

In patients who developed acute AF after noncardiac surgery, registry data suggest that initiation of oral anticoagulation within 30 days after discharge was associated with a reduced risk of thromboembolic events.[6] The randomized prospective ASPIRE-AF trial (Anticoagulation for Stroke Prevention In Patients With Recent Episodes of Perioperative Atrial Fibrillation After Noncardiac Surgery; NCT03968393) will further inform the optimal long-term oral anticoagulation strategy for patients who develop acute AF after noncardiac surgery.

Longer-Term Rhythm Management

Long-term rhythm management should be individualized as part of a shared decision plan with each patient. In EAST-AFNET 4 (Early Treatment of Atrial Fibrillation for Stoke Prevention Trial), an early rhythm control strategy within 1 year of AF diagnosis was associated with a lower risk of adverse cardiovascular outcomes.[210] Whether this benefit extends to AF initially detected in the acute care setting merits further research.

The choice of rhythm control method is based on the balance of multiple factors, including patient preference, risk factors, and comorbidities. Long-term antiarrhythmic medication options are described in Supplemental Table 5. The choice of antiarrhythmic drug is guided by patient baseline comorbidities and potential drug-drug interactions. Once any antiarrhythmic medication has been initiated, long-term monitoring is required to

Table 1. Acute AF: 10 Key Implications for Clinical Practice

10 Key Takeaways	
1	Acute AF is defined as AF detected in the setting of acute care or acute illness; this includes AF occurring during acute hospitalization. Acute AF may be detected or managed for the first time during acute hospitalization for another condition.
2	Acute AF is associated with high risk of long-term AF recurrence, warranting clinical attention during acute hospitalization, at transition of care, and over long-term follow-up.
3	A framework of substrates and triggers can be useful for the evaluation and management of AF occurring during acute hospitalization. AF after cardiac surgery is a distinct type of acute AF.
4	Acute management of AF occurring during hospitalization requires a multipronged approach. Key components of acute management include identification and treatment of triggers, selection and implementation of rate/rhythm control, and management of anticoagulation.
5	Acute rate or rhythm control strategy should be individualized with consideration of the patient's capacity to tolerate the potential rapid rates or atrioventricular dyssynchrony, as well as the patient's ability to tolerate the risk of either the rate or rhythm control strategy.
6	In hemodynamically unstable patients, immediate electrical cardioversion with DCCV is the treatment of choice. Rhythm control should also be considered for patients unable to attain clinically adequate rate control despite optimal use of atrioventricular nodal blocking agents and management of acute triggers. Electrical cardioversion is the most effective method to achieve acute rhythm control. Hemodynamic monitoring and considerations for thromboembolic prophylaxis are warranted for both electrical and pharmacological cardioversion.
7	Indication for anticoagulation is based on substrate, with feasibility and timing for anticoagulation based on patient's bleeding risk and contextual considerations of the acute conditions.
8	Given the high risks of AF recurrence in patients with acute AF, clinical follow-up and extended heart rhythm monitoring are warranted to tailor longer-term management. Management of AF and modifications targeting the substrate should be instituted. Long-term management will be heavily tied to the substrate, guiding follow-up, long-term heart rhythm monitoring, and considerations for rhythm management strategies.
9	Overall management of acute AF addresses substrates and triggers. The 3As of acute management are acute triggers, AF rate/rhythm management, and anticoagulation. The 2As and 2Ms of long-term management are AF rate/rhythm management, anticoagulation, monitoring of heart rhythm, and modification of lifestyle and risk factors.
10	Patients with acute AF benefit from close interdisciplinary care collaborations, allowing appropriate treatments tailored to patient's underlying substrates and acute conditions.

AF indicates atrial fibrillation; and DCCV, direct current cardioversion.

Table 2. Areas for Future Research in Acute AF

Aspect of acute AF	Area for future research
Detection	Patients at higher risk for acute AF may benefit from increased intensity of electrocardiographic monitoring during hospitalization. Developing a framework to triage the intensity of electrocardiographic monitoring during acute hospitalization is a practical area for future research.
Burden	Consistent with the current uncertainty in the optimal threshold of AF burden to initiate anticoagulation, further research is needed to identify the optimal threshold of the burden of acute AF to initiate anticoagulation in the acute setting.
Substrates and triggers	Using the conceptual framework of substrates and triggers, additional research in better understanding the substrates and triggers of acute AF in different acute conditions will further improve condition-specific considerations and treatments.
Specific risks	Additional dedicated studies of large cohorts are needed to better understand the acute thromboembolic risks of acute AF, in association with specific populations as pertinent, to provide condition-specific insights to further refine the candidacy and threshold for acute anticoagulation.
Acute milieu	Further research combining risk scores based predominantly on chronic conditions (such as the CHA_2DS_2-VASc risk score) with considerations of the acute coagulopathic and prothrombotic milieu may be useful to better assess the benefits, risks, and optimal selection of patients with acute AF for acute anticoagulation.
Long term	Given the high rates of long-term AF recurrence, the optimal frequency and modality of long-term monitoring in patients who have experienced acute AF need further study.

observe for potential toxicities. The potential toxicities and monitoring strategies are described in Supplemental Table 5.

Several randomized trials have shown that AF ablation is more effective at maintaining long-term sinus rhythm compared with medical therapy.[211,212] Mortality benefit with AF ablation has been observed in patients with heart failure.[213,214] Over long-term management, in applicable patients, ablation may be considered as an early management strategy at experienced centers.[17,18]

Longer-Term Adjunct Risk Factor Evaluation and Modifications

Obesity,[215–217] obstructive sleep apnea,[218,219] hypertension,[220] diabetes,[221] physical inactivity,[222,223] and alcohol abuse[224–226] have been shown to be risk factors for AF. Intensive weight loss,[227] comprehensive risk factor modifications (with weight loss, blood pressure management, glycemic control, sleep-disordered breathing management, alcohol reduction, and tobacco cessation counseling),[228,229] and aerobic exercise training[230,231] have been demonstrated to reduce AF burden and to improve quality of life.[227–232] Aggressive risk factor modification and lifestyle interventions[18,233] should be pursued for all patients with acute AF, in tandem with long-term AF management strategies.

IMPLICATIONS FOR CLINICAL PRACTICE AND FURTHER RESEARCH

Table 1 delineates the Writing Committee's consensus for implications for clinical practice. In addition, the Writing Committee has outlined the current gaps in knowledge and areas for future research in acute AF in Table 2.

CONCLUSIONS

Acute AF is defined as AF detected in the setting of acute care or acute illness. Acute AF may be detected or managed for the first time during acute hospitalization for another condition (Figure 2). Acute AF is common and not benign. Acute AF is associated with high risks of long-term AF recurrence, warranting clinical attention during acute hospitalization and over long-term follow-up (Figure 4). A conceptual framework of substrates and triggers can be useful for considering AF occurring during acute hospitalization. Overall management of acute AF addresses substrates and triggers. The 3As of acute management are acute triggers, AF rate/rhythm management, and anticoagulation (Figure 5). The 2As and 2Ms of long-term management include monitoring of heart rhythm and modification of lifestyle and risk factors, in addition to considerations for AF rate/rhythm management and anticoagulation (Figure 5). Several gaps in knowledge related to acute AF exist and warrant future research.

ARTICLE INFORMATION

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

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*Modest.

†Significant.

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Pamela J. McCabe	Mayo Clinic	None	None	None	None	None	None	None
Jonathan P. Piccini	Duke University Medical Center	AHA (grant to my institution for which I serve as PI)†; Abbott (grant to my institution for which I serve as PI)†; Gilead (grant to my institution for which I serve as PI)†; Johnson & Johnson (grant to my institution for which I serve as PI)†; NHLBI (grant to my institution for which I serve as PI)†; Philips (grant to my institution for which I serve as PI)†	None	None	None	None	Abbott†; Allergan*; Biotronik*; ARCA biopharma*; Boston Scientific†; JHJ*; LivaNova*; Philips†; Medtronic†; Sanofi†; Milestone*	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

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