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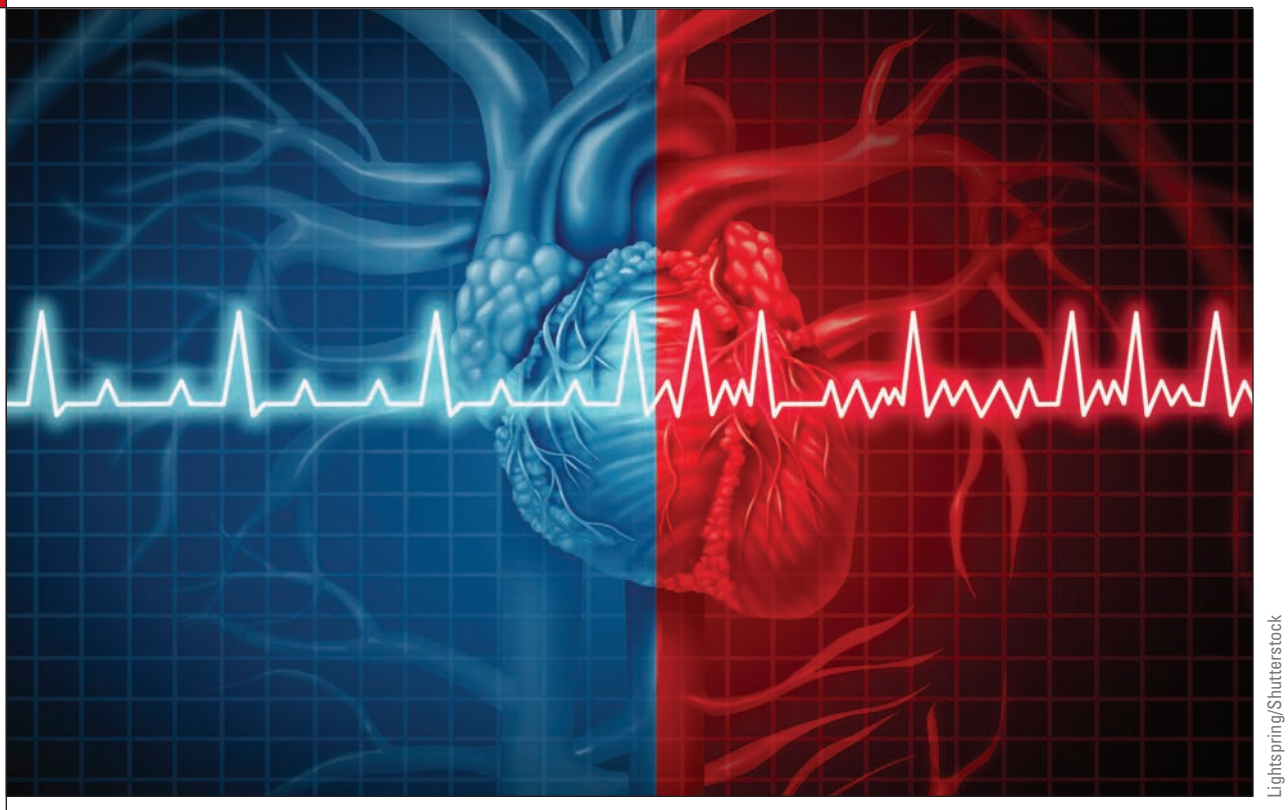

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The ABCs of atrial fibrillation

Abstract: This article provides an outline of the diagnosis and pathogenesis of atrial fibrillation. It introduces a mnemonic for atrial fibrillation treatment guidelines based on recent evidence. Mnemonics use has been associated with improved learning and task organization.[1] Articles using mnemonics to inform clinical practice have been well-received and implemented.[2,3]

By Mohamed Toufic El Hussein, PhD, NP and Lauren Kilfoil

Atrial fibrillation (AF) is an alteration of normal sinus rhythm characterized by quivering of the atria, resulting in ineffective contractility. At a cellular level, the atrioventricular (AV) node is overloaded with depolarizing impulses of up to 300 to 600 per minute.[4] Due to the pooling and static movement of blood within the atria, there is an increased risk of thromboembolism development in the left atrial appendage. Prompt treatment and long-term management of AF is imperative in order to reduce mortality related to ischemic stroke, coronary artery occlusion, or pulmonary embolism.

Diagnosis

An acute clinical diagnosis of AF is made through ECG monitoring. The ECG for those with AF is characterized by the absence of a P wave, as well as irregularity in the R-R interval.[4] AF may occur episodically (paroxysmal AF), continuously (persistent AF), or permanently due to unresponsiveness to treatment.[5] AF is further assessed through chest radiography and echocardiography.[6] Additionally, AF is categorized as valvular and nonvalvular through diagnostic imaging.[7] A clinical diagnosis of valvular AF is characterized by moderate-to-severe mitral stenosis or the presence of a

Keywords: anticoagulation, atrial fibrillation, cardioversion, pacemaker, rate control

mechanical heart valve, while nonvalvular AF requires the absence of rheumatic mitral stenosis, a mechanical heart valve, or mitral valve repair.[7,8]

Patient education

Due to the narrow therapeutic index of many prescribed medications, patients should be advised of the importance of medication adherence and symptoms of medication overdose. Prevention education for cardiovascular disease should include the implementation of healthy lifestyle practices, such as individualized diet and exercise planning based on age and comorbidities, smoking cessation, and frequent monitoring of cardiovascular risk factors.[9]

The mnemonic

The recommended treatment options for the management of newly diagnosed and permanent AF include anticoagulation, beta-blockers, calcium channel blockers, cardioversion, diuretics, other drugs, electrolytes, and electricity. The NP should aim to achieve a heart rate of less than 110 beats/minute and provide treatment that prioritizes the control of rate over rhythm, as these approaches are associated with optimal outcomes in seminal articles in the field of cardiology.[5,10,11] The NP should acknowledge individual patient circumstance and comorbidities and complete a baseline assessment of renal and hepatic functions before initiating or titrating pharmacotherapy.

Anticoagulation

Anticoagulation therapy reduces the risk of thromboembolism in AF.[5,12] Patients with a lower stroke risk and those with contraindications are considered unsuitable candidates for anticoagulant therapy.[5] AF treatment with anticoagulation requires careful consideration of existing comorbidities and potential adverse reactions. Though the use of anticoagulants has been associated with a reduced risk of ischemic stroke, the NP should screen for abnormalities in bloodwork, with special consideration to international normalized ratio (INR), electrolyte balance, and liver and kidney function. Non-vitamin K antagonist oral anticoagulants (NOACs) are recommended over vitamin K antagonists (VKAs) in nonvalvular AF, though this is clinically dependent on the patient's stroke or bleeding risk.[12]

The choice of anticoagulant therapy in AF requires a thorough understanding of the patient's risk factors and medical history, with special consideration for stroke risk. The recommended method of assessing the patient's risk of stroke in nonvalvular AF is commonly known as the CHA_2DS_2-VASc scoring system.[5,12,13] (See *CHA_2DS_2-VASc scoring for anticoagulation therapy eligibility*.) The patient's risk score for an ischemic event is calculated with consideration for presence of congestive heart failure, hypertension, diabetes, vascular disease, and a history of stroke, transient ischemic attack, or thromboembolism, as well as age and gender. In the context of nonvalvular AF, low-scoring patients should not be offered anticoagulant treatment, while treatment should be considered for those with higher scores indicating increased risk of stroke.[13]

NOAC therapy. The Canadian Cardiovascular Society recommends the use of NOAC therapy rather

CHA_2DS_2-VASc scoring for anticoagulation therapy eligibility[5,12,13]

Congestive heart failure	1
Hypertension	1
Age (≥75)	2
Diabetes mellitus	1
Stroke, transient ischemic attack, or thromboembolism (history)	2
Vascular disease	1
Age (65-74)	1
Sex **c**ategory (female)	1

For male and female patients with scores ≥ 1 and ≥ 2, respectively, and nonvalvular atrial fibrillation, prophylactic use of anticoagulants should be considered.

HAS-BLED scoring system for bleeding risk assessment[5,14]

Hypertension	1
Abnormal renal and/or liver function (1 point each)	1 or 2
Stroke	1
Bleeding history or predisposition	1
Labile INR	1
Elderly (>65 years)	1
Drugs (antiplatelet agents, NSAIDs)/alcohol use (1 point each)	1 or 2
Scores ≥3 indicate a higher risk of bleeding	

Key: INR, international normalized ratio; NSAIDs, nonsteroidal anti-inflammatory drugs

than warfarin (a VKA) in nonvalvular AF.[15] In determining a patient's suitability for a particular NOAC treatment regimen, the NP should be mindful of the contraindications for use (see *Contraindications for NOAC therapy*), as well as elevated risk of bleeding based on HAS-BLED score (see *HAS-BLED scoring system for bleeding risk assessment*).[5] Recent guidelines recommend NOAC treatment over the use of warfarin in patients with a history of intracranial bleeds, variable diet, and poor access to lab testing.[8] Due to the distinct pharmacologic profiles of each NOAC, the NP should consider the patient's medical history, diagnostic results, and individual preference when initiating treatment.[5]

VKA anticoagulation. The use of warfarin is recommended in patients with moderate-to-severe mitral stenosis or the presence of a mechanical valve.[7,12] The NP should consider the use of warfarin in patients with contraindications for NOAC therapy, except for in pregnancy, when low-molecular-weight heparin is recommended.[8] Warfarin dosing should be titrated based on the patient's INR in order to maintain a consistent therapeutic level of 2.0 to 3.0, and 2.5 to 3.5 in patients with mechanical valves, though INR ranges remain patient-specific.[16]

Combination therapy. On rare occasions, such as after the surgical insertion of a coronary artery stent, the short-term use of antiplatelet medications paired with warfarin or a NOAC is indicated. However, recent recommendations from the American Academy of Family Physicians advises against the long-term use of a combination of anticoagulants and antiplatelet therapies due to the significantly increased risk of major bleeding.[5] In fact, antiplatelet therapy including aspirin and/or clopidogrel used in combination with warfarin or dabigatran has been associated with adverse reactions and a low safety profile.[5]

Contraindications for NOAC therapy[8]

- Mechanical heart valve
- Valvular atrial fibrillation
- Renal impairment with creatinine clearance <30 mL/min
- Severe liver dysfunction
- Pregnancy
- Breastfeeding
- Use of potent P-gp or CYP3A4 inhibitors or inducers

Source: Adapted from Guidelines and Protocols Advisory Committee. Use of Non-Vitamin K Antagonist Oral Anticoagulants (NOAC) in Non-Valvular Atrial Fibrillation. BCGuidelines.ca. 2015.
Key: NOAC, non-vitamin K antagonist oral anticoagulant; P-gp, P-glycoprotein

Alternate interventions. For patients for whom long-term anticoagulation therapy is contraindicated, the NP should consider a percutaneous implant that occludes the left atrial appendage, a site where many clots originate, as an intervention for anticoagulation.[12] The device lowers the risk of thromboembolism in AF.

Beta-adrenergic blockers

In the acute and long-term management of AF, the NP should implement treatment with beta-adrenergic blockers in order to effectively control heart rate. The use of beta-blockers is preferred over calcium channel blockers in patients diagnosed with coronary artery disease or systolic dysfunction.[12] For patients experiencing acute exacerbations of AF, the use of an I.V. beta-blocker may be indicated in order to control ventricular rate.[12] For long-term management of heart rate, the NP should implement treatment with an oral beta-blocker.[12] Beta-blockers should be administered and titrated in order to achieve a heart rate below 110 beats/minute.[5]

Calcium channel blockers

The use of nondihydropyridine calcium channel blockers is recommended for managing heart rate in AF.[5] There is no conclusive evidence to favor calcium channel blockers over beta-blockers for rate management, though calcium channel blockers are preferred in patients with asthma.[5] This treatment option should be avoided in patients with decompensated heart failure. Doses and route of administration should be titrated to achieve a controlled rate below 110 beats/minute.[8]

Cardioversion

A critical step in the process of managing acute AF is determining the stability of the presenting patient. Hemodynamic instability refers to a cluster of symptoms secondary to the dysrhythmia that indicate decompensation, and include a systolic reading less than 90 mm Hg, altered level of consciousness, cardiac ischemia, and/or worsening heart failure.[7] The use of electrical cardioversion is recommended in symptomatic unstable AF.[12] Cardioversion to a stable sinus rhythm can be achieved through pharmacologic or electrical means, depending on the patient's existing comorbidities and acute presentation. For pharmacologic cardioversion, flecainide or propafenone can be used in patients without structural heart conditions. Amiodarone may be indicated for acute management of AF, as discussed later.[12]

For patients with acute AF requiring immediate conversion to sinus rhythm, synchronized electrical cardioversion is indicated. Prior to the procedure, the NP should order anticoagulation, as well as sedation.[17] If the duration of AF exceeds 48 hours, or the duration is unknown, a transesophageal echo may be indicated to assess for the presence of a thrombus within the atrium or atrial appendage prior to cardioversion.[13] In the case of elective cardioversion, treatment with a NOAC or VKA 3 weeks prior to the procedure is recommended. After the procedure, the patient should undergo anticoagulant therapy for a minimum of 4 weeks, regardless of the procedure's elective or emergent nature.[13]

Diuretics

AF management requires careful consideration for fluid balance, especially for patients with concomitant heart failure.[17] The stretching and remodeling of the atria in heart failure can trigger the onset of AF, while chronic dysfunction associated with AF may cause heart failure.[18] With an understanding of the pathophysiology of AF and concomitant heart failure, the NP might consider the administration of a diuretic in order to achieve a therapeutic effect of reduced cardiac congestion.[18] A recent systematic review and meta-analysis identified mineralocorticoid receptor antagonists, including spironolactone or eplerenone, as a therapeutic treatment option for AF, associating their use with significant reductions in new-onset and recurrent AF.[19]

Other drugs

The use of amiodarone as an antiarrhythmic and rate control agent may be indicated in order to maintain normal sinus rhythm, most effectively in cases of uncontrolled ventricular rate.[12] In fact, the American Association of Family Physicians recommends the use of amiodarone as a rhythm-controlling agent when rate-controlling agents are ineffective, though its use should be limited to unresponsiveness or contraindications to other therapies.[5,20] While amiodarone may be effective for the management of AF in acute settings, the many systemic adverse reactions, including thyroid, genitourinary, skin, gastrointestinal, and CNS changes, may limit its long-term use.[21] A thorough medical history and ongoing follow-up is required for the patient receiving treatment with amiodarone.

Digoxin may also be used as a rate-controlling agent in the context of AF as a second-line treatment option, though its use requires caution due to the drug's potential adverse reactions.[4] In fact, a recent systematic review and meta-analysis evaluating the adverse reactions of digoxin use in patients with AF found an association between the drug's use and all-cause mortality.[22] Additionally, the pharmacokinetics of digoxin involves a slow peak effect of rate control, which is not optimal in cases of severe acute AF.[23] However, the update to the AF guideline from the American Heart Association/American College of Cardiology/Heart Rhythm Society maintains a weak recommendation of digoxin administration in patients with AF and acute coronary syndrome with associated left ventricular dysfunction and heart failure, or hemodynamic instability.[7] Due to its rate-controlling action with no associated effect on BP, digoxin may be indicated in patients experiencing clinically low BP.[23] Due to digoxin's narrow therapeutic range, the NP should titrate doses to maintain serum levels below 1.2 ng/mL in order to avoid toxicity.[24]

Electrolytes

Electrolyte balance, particularly of potassium and magnesium, is essential for the maintenance of normal sinus rhythm and the prevention of adverse cardiac events. In patients with chronic cardiovascular comorbidities being treated with diuretics and/or angiotensin-converting enzyme inhibitors, monitoring of electrolyte balance is especially important.

Potassium. Due to the essential role of potassium in maintaining an electrical gradient at a cellular level, any extreme alterations in its balance can precipitate or worsen the existence of cardiac dysrhythmias. In fact, recent evidence has associated low serum potassium (<3.5 mmol/L) or high serum potassium (>5.0 mmol/L) with an increased risk of mortality in patients with AF.[25]

Magnesium. In a recent randomized controlled trial conducted by Bouida and colleagues in Tunisia, the synergistic effect of high and low dosing of magnesium sulfate ($MgSO_4$) combined with traditional rate-controlling agents in controlling heart rate in rapid AF was compared against a placebo.[26] The three treatment arms, specifically low-dose $MgSO_4$ (4.5 g in 100 mL 0.9% sodium chloride I.V.; n = 148), high-dose (9.0 g in 100 mL 0.9% sodium chloride I.V.; n = 153), and placebo (100 mL 0.9% sodium chloride I.V.; n = 149) were evaluated for their efficacy in achieving and maintaining at least a 20% reduction in ventricular rate from baseline or a reduction to 90 beats/minute after 4 hours and 24 hours in combination with standard rate-controlling

agents (digoxin, diltiazem, and beta-blockers). A statistically significant number of participants in treatment groups receiving $MgSO_4$ achieved rate control by 4 hours compared with the placebo. After 24 hours, more of the low-dose $MgSO_4$ group had achieved and maintained sinus rhythm—a statistically significant difference from the placebo group ($P = .005$) and the high-dose group ($P = .03$). Bouida and colleagues recommend the use of 4.5g $MgSO_4$ in 100 mL 0.9% sodium chloride I.V. in conjunction with standard rate-controlling agents to achieve and maintain rate control in rapid AF.[26]

Electricity

Catheter ablation, a procedure used to cause remodeling of cardiac tissue, is recommended for patients with symptomatic AF and heart failure with a reduced left ventricular ejection fraction.[7] In fact, in a recent randomized controlled trial comparing long-term success of catheter ablation versus amiodarone in managing AF in patients with heart failure, catheter ablation was associated with more optimal outcomes.[27] The benefits of catheter ablation for patients with persistent AF may include symptom reduction and decreased likelihood of hospitalization.[28] Given the number of existing ablation procedures that isolate particular regions within the atria, the NP should be aware of each patient's indications for treatment. Due to the influence of pulmonary veins on AF occurrence, pulmonary vein isolation may be required. This method removes triggers that may potentiate fibrillation, and therefore may be effective in paroxysmal AF.[29] Alternatively, a maze procedure creates scar tissue within the atria to ablate AF while maintaining electrical conduction via the sinoatrial (SA) node.[29] AV nodal ablation is indicated in patients who remain unresponsive to pharmacologic rate-controlling interventions or other ablation procedures.[17,20,30] Due to the destruction of pacing cells, patients receiving AV nodal ablation will likely require permanent right ventricular pacing.[31]

The implantation of a cardiac pacemaker may be indicated as a third-line treatment in patients with AF who remain unresponsive to rate- and rhythm-controlling therapies.[32] However, a permanent pacemaker is required in cases of AV nodal ablation. Right ventricular pacing has historically been indicated in patients after AV nodal ablation, though biventricular pacing may be preferable in cases of left ventricular dysfunction.[31] A randomized controlled trial comparing biventricular pacing to traditional right ventricular pacing determined that biventricular pacing yielded improved outcomes in patients with AV block, heart failure, and left ventricular systolic dysfunction.[33]

Conclusion

In managing AF in patients, the NP should be diligent and efficient in providing necessary care to return to normal sinus rhythm. For the patient experiencing persistent AF, pharmacotherapy should be administered and titrated in order to prevent adverse cardiac events and maintain hemodynamic stability. In the acute setting, the use of this mnemonic device in AF management will provide ease of recall of best evidence. Collaboration with the patient, as well as ample education, is necessary in the provision of adequate and sustainable management of AF. NP

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The authors have disclosed no potential conflicts of interests, financial or otherwise.

DOI-10.1097/01.NPR.0000681780.47800.a3