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## Managing Atrial Fibrillation

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### INTRODUCTION

Although most emergency physicians will have an established routine for managing the emergency department (ED) patient with atrial fibrillation, in the last 4 years 9 new updates and guidelines for the management of these patients have been published by European, Canadian, and US professional groups,<sup>1–8</sup> rendering many of those practices out of date. We discuss our approach to the ED patient with atrial fibrillation (or atrial flutter, for which the recommendations are the same) according to the most recent guidelines<sup>1–9</sup> and our expertise in the area.<sup>10–17</sup>

### THE UNSTABLE PATIENT

First, it is important to carefully consider why the patient is unstable and whether the atrial fibrillation is the cause. Many patients are hypotensive as a result of sepsis, gastrointestinal hemorrhage, or other causes, and have a long history of atrial fibrillation, with an abnormally high pulse rate because of their acute illness. These patients will likely not convert with the immediate cardioversion that is recommended in atrial fibrillation guidelines<sup>3,5,7</sup> because the atrial fibrillation is long-standing. Their hypotension is usually caused by another source that needs to be addressed. For these patients, it may be helpful to slow the pulse rate slightly to reduce myocardial demand, but recall that many of them will require a relatively fast pulse rate to compensate for their decreased stroke volume (otherwise, their cardiac output will decrease).

Second, we contend that the definition of stability represents a continuum, rather than a dichotomous state. Although a patient who is losing consciousness is clearly unstable (and requires immediate cardioversion despite the risk of stroke if the duration of atrial fibrillation is >48 hours), the tachypneic patient with early signs of heart failure may have time for pharmacologic intervention. We outline an approach to one of the most challenging unstable atrial fibrillation patients, the hypotensive, conscious patient with atrial fibrillation of unknown duration (Figure 1). Challenging because sedation may worsen the hypotension, but cardioversion without sedation should be avoided. There are relatively few data to

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support any of the outlined approaches (or one over another); they represent both guideline recommendations and approaches we routinely use.

### **Amiodarone**

Amiodarone is used for rate control in this setting, not for cardioversion (which usually takes 4 to 6 hours with intravenous amiodarone).<sup>5,7</sup> However, anticoagulation (with heparin) is advisable, given that cardioversion to normal sinus rhythm may occur with this drug.

### **Digoxin**

Another guideline-endorsed option is intravenous digoxin. Although slow in onset, anecdotally it often improves the blood pressure within 30 minutes.<sup>18</sup>

### **Diltiazem**

In all guidelines, it is recommended that nondihydropyridine calcium-channel blockers (eg, diltiazem) be avoided in the setting of hypotension or heart failure, although the quality of evidence for the recommendation is poor.<sup>2,5,7</sup> Many emergency physicians have found that by cautiously slowing the pulse rate with intravenous diltiazem, the blood pressure actually increases, presumably because of increased ventricular filling time. If this option is selected, doses should be administered in small amounts, followed by assessment of the response.

In the clearly unstable patient, immediate electrical cardioversion is required (Figure 1). If possible, administer heparin first.<sup>1</sup> Given the time of onset for subcutaneous heparin, we use intravenous heparin in this situation.

## **THE STABLE PATIENT**

Most ED atrial fibrillation patients will be alert, with a well-perfusing blood pressure. Approximately 20% of these patients will experience chest pain with the fast pulse,<sup>19</sup> but in the majority this is demand-related and not due to a rupture of an atherosclerotic plaque. One study found that ED atrial fibrillation patients without evidence of significant ST-segment changes were at very low risk for acute myocardial infarction.<sup>20</sup> Another found that atrial fibrillation did not change the relative risk of an acute coronary syndrome in ED patients who had chest pain syndromes.<sup>21</sup> We recommend using clinical judgment to rule out an acute coronary syndrome, including determining whether the chest pain started before or after the palpitations. The ECG findings before and subsequent to rate or rhythm control should be incorporated into the assessment.

### **Rate or Rhythm Control**

Both options are available in the stable patient who has been in atrial fibrillation (clear onset of palpitations) for fewer than 48 hours. After 48 hours, rate control is generally the only option because of the increased risk of stroke.<sup>1,5,9</sup>

For patients aged 65 years and older, several landmark trials showed that there was no difference in outcomes with rate versus rhythm control after 5 years.<sup>22,23</sup> Thus, for these patients, unless they are very symptomatic, we generally use a rate-control strategy.

Conversion to sinus rhythm at a later date remains an option in this scenario, after a minimum of 3 weeks of anticoagulation.

Although 5-year outcomes were no different in these trials for patients aged 65 years or older, the longer-term outcomes may be quite different for a 50-year-old patient who has another 30 to 40 years to live in atrial fibrillation. Both the European and American guidelines imply that these patients should have restoration of normal sinus rhythm.<sup>2,5</sup> Whether that is conducted by the emergency physician on the day of presentation (if duration is <48 hours) or several weeks later by a cardiologist depends on emergency physician comfort with cardioversion, hospital policies, and other factors.

### Approach to Rate Control

If the rate-control option is selected, the most common medications used are  $\beta$ -blockers and nondihydropyridine calcium-channel blockers (Table 1). Digoxin does not provide adequate rate control, with the exception of patients who are entirely sedentary (it does not control pulse rate during any exertion).<sup>24</sup> Currently, the only indication for digoxin is when rate control with a  $\beta$ -blocker or calcium-channel blocker has failed, in which case digoxin is added to the regimen.<sup>2,5,7,25</sup> Anecdotally, cardiologists tend to use  $\beta$ -blockers, whereas emergency physicians prefer diltiazem. Given the potential for hypotension when combining  $\beta$ -blockers with calcium-channel blockers, we recommend using one or the other, not both.

The evidence for  $\beta$ -blockers versus calcium-channel blockers is weak: several studies found no difference in outcomes,<sup>26</sup> other than a possible worsening of exercise tolerance with  $\beta$ -blockers.<sup>27</sup> Another found that in patients older than 65 years, calcium-channel blockers were associated with higher 90-day mortality in ED patients with atrial fibrillation<sup>12</sup>; however, indication bias is a possibility in that retrospective study. In our practice, we tend to use  $\beta$ -blockers for patients with coronary artery disease<sup>6</sup> (given that several hypertension guidelines recommend  $\beta$ -blockers for those with a history of angina or myocardial infarction)<sup>28,29</sup> and diltiazem for other patients.

The goal of rate control varies by guidelines: less than 110 beats/min for European<sup>2</sup> and US guidelines (but the latter stipulates that patients have a normal left ventricular ejection fraction),<sup>5</sup> and less than 100 beats/min for Canadian guidelines.<sup>30</sup> All are based on the Rate Control Efficacy in Permanent Atrial Fibrillation: a Comparison between Lenient versus Strict Rate Control II (RACE II) trial, which found no difference in patient outcomes with tight control (resting pulse rate <80 beats/min) versus looser control (resting pulse rate <110 beats/min)<sup>31</sup>; however, there were relatively few patients with a pulse rate in the 100 to 110 beats/min range in that study. Therefore, we use a goal of a resting pulse rate of less than 100 beats/min in the ED.<sup>14</sup>

### Approach to Rhythm Control

Contraindications to ED rhythm control are listed in Figure 2.<sup>7</sup> Cardioversion can be achieved by electrical cardioversion with procedural sedation or by pharmacologic cardioversion (most commonly with procainamide<sup>19</sup>). ED studies have consistently shown

electrical cardioversion to be approximately 90% effective,<sup>5,32,33</sup> whereas procainamide is approximately 60% effective.<sup>34</sup>

If electrical cardioversion is chosen, one study suggests that these patients should not receive rate control first because this practice was associated with lower rates of cardioversion success.<sup>35</sup> In general, the data suggest that an anterior-posterior pad placement (the “sandwich”) is best,<sup>36</sup> although there is conflicting evidence.<sup>37</sup> Whatever the orientation of the pads, the goal is to avoid placement over bone (eg, sternum) or fatty tissue (eg, large breasts) because the former will decrease the conduction of the electricity (and therefore require more energy to cardiovert the patient) and the latter will increase the distance from the heart. A biphasic machine is better than monophasic because it requires less electricity to achieve cardioversion. In general, we prefer higher initial voltages (minimum 150 J for biphasic and 100 J for monophasic [may start higher]) because fewer high-voltage shocks are less harmful than more lower-voltage ones.<sup>2,5,7</sup> If the patient is extremely obese, consider using the paddles and applying as much weight or force as possible (~20 lbs/paddle) to improve conduction and the chances of success.<sup>5</sup>

Occasionally, the patient converts with electrical cardioversion and then reverts to atrial fibrillation within a very short time frame (eg, less than a minute). Another shock should be administered if the patient is still sedated,<sup>5</sup> but consider administering intravenous rate control immediately afterward, which we have found sometimes “holds” the patient in normal sinus rhythm. This would be later followed by the oral form of the rate-control agent.

Anticoagulation for cardioversion (electrical or pharmacologic) is controversial. Some guidelines advise that no oral anticoagulation is required if the onset was within 48 hours and the patient is discharged from the ED in sinus rhythm.<sup>7</sup> Others contend that oral anticoagulation should be offered according to usual anticoagulation guidelines (eg, CHA<sub>2</sub>DS<sub>2</sub>-VASc score<sup>38</sup>), regardless of whether the patient is in sinus rhythm.<sup>2,5</sup> We take the latter approach, and a recent study on ED cardioversion without oral anticoagulation supports this choice.<sup>39,40</sup> Until further studies are published that refute the findings of this recent observational study, it is prudent to initiate oral anticoagulation according to usual anticoagulation guidelines (see below).<sup>3,5</sup>

### Oral Anticoagulation

The recommendations for who should receive ongoing oral anticoagulation are similar across professional guidelines, with slight variations (Figure 3). What is key to note is that all groups now recommend oral anticoagulation for many more patients, including all patients aged 65 years or older (Canadian and European guidelines).<sup>3,5,6</sup> Both the US and European guidelines use CHA<sub>2</sub>DS<sub>2</sub>-VASc to determine oral anticoagulation eligibility. The American Heart Association recommends oral anticoagulation for CHA<sub>2</sub>DS<sub>2</sub>-VASc score greater than or equal to 2, and the choice of oral anticoagulation or aspirin or nothing for a score of 1. In comparison, the European Society of Cardiology recommends oral anticoagulation for CHA<sub>2</sub>DS<sub>2</sub>-VASc score greater than or equal to only 1.<sup>3,5</sup> There remain few indications for aspirin therapy (Figure 3). To assess bleeding risk, all guidelines endorse HAS-BLED (Figure 4), in which a score greater than or equal to 3 indicates high risk of bleeding.<sup>41</sup>

Our recent study found that discharged ED atrial fibrillation patients who were eligible for oral anticoagulation were much more likely to be receiving it a year later (75%) if they were provided with a prescription in the ED compared to those for whom oral anticoagulation initiation was left to the primary care provider (34%).<sup>42</sup> The sample size was small; however, a larger study with long-term outcomes is not likely to be soon forthcoming. The study suggests that the emergency physician has an opportunity to decrease the patient's long-term risk of stroke. Given the overwhelming evidence that oral anticoagulation prevents strokes,<sup>43</sup> we initiate eligible patients on oral anticoagulation in the ED (Table 2).

Whether a novel oral anticoagulant is preferable to warfarin depends on the guidelines consulted, and, more importantly, the patient's ability to stay within therapeutic range (International Normalized Ratio [INR] 2–3) while receiving warfarin. If patients can stay in range most of the time (>60–65% of the time), their protection against stroke is likely better than with the novel oral anticoagulants.<sup>44–46</sup> However, many patients cannot achieve this; therefore, novel oral anticoagulants may be better for most. Currently, dabigatran, rivaroxaban, and apixaban are approved for use in nonvalvular atrial fibrillation by the Food and Drug Administration, Health Canada, and the European Medicines Agency.

Dabigatran is cleared exclusively through the kidneys. Although the drug can be used with certain levels of renal failure, as emergency physicians (who do not follow patients), we would not routinely offer dabigatran, or any novel oral anticoagulants, to anyone with any suggestion of renal failure. In the Randomized Evaluation of Long-Term Anticoagulation Therapy (RE-LY) trial, the Rivaroxaban Once daily oral direct factor Xa inhibition Compared with vitamin K antagonism for prevention of stroke and Embolism Trial in AF (ROCKET-AF) trial, and the Apixaban for Reduction in Stroke and Other Thromboembolic Events in Atrial Fibrillation (ARISTOTLE) trial, dabigatran, rivaroxaban, and apixaban had a lower rate of life-threatening bleeding than warfarin.<sup>44–47</sup> Reversal agents are not yet available for novel oral anticoagulation (compared with 4-factor prothrombin complex concentrate for warfarin) but are likely to come to market in the next 2 years. Patients with mechanical heart valves or hemodynamically significant mitral stenosis should be treated only with warfarin (they were excluded from the novel oral anticoagulant trials).<sup>44–46</sup>

### Disposition From the ED

In recent collaborative work, we found that 69% of ED visits with a primary diagnosis of atrial fibrillation resulted in hospitalization in the United States compared with 37% in Canada's most populous province.<sup>48</sup> The largest intercountry variation was for patients younger than 65 years: 64% of visits made by these patients resulted in hospitalization in the United States versus 25% in the Canadian cohort. Several Canadian studies have suggested that discharge home is safe.<sup>11,33</sup> Presumably the younger US patients are being admitted for further testing; however, this can be performed on an outpatient basis. We recommend that hospitalization be reserved for patients with the following: another ED diagnosis (eg, pneumonia), presence of acute coronary syndrome or heart failure, or failure of rate control (unable to achieve <100 beats/min) or rhythm control.<sup>7</sup> Otherwise, these patients are likely safest in their own homes with close outpatient follow-up care.<sup>12</sup>

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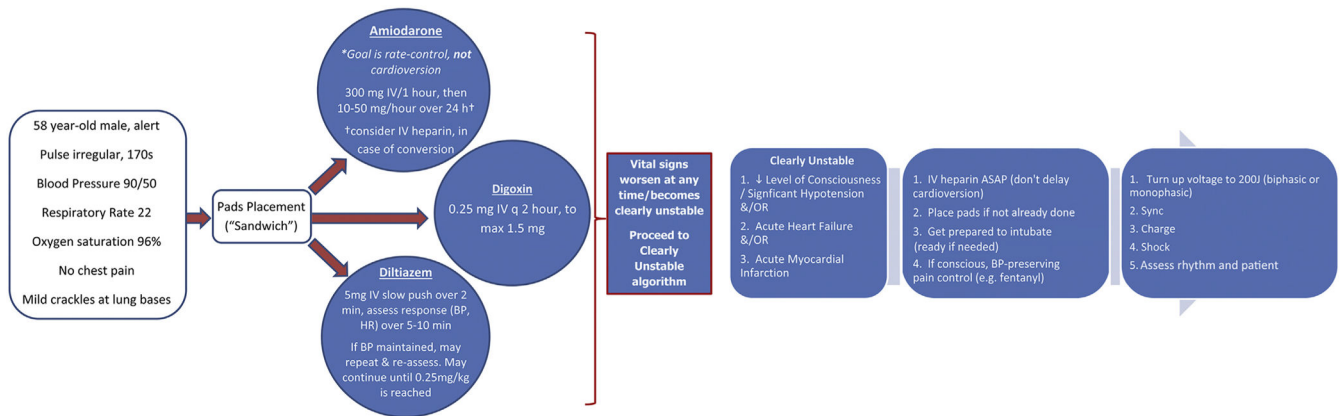
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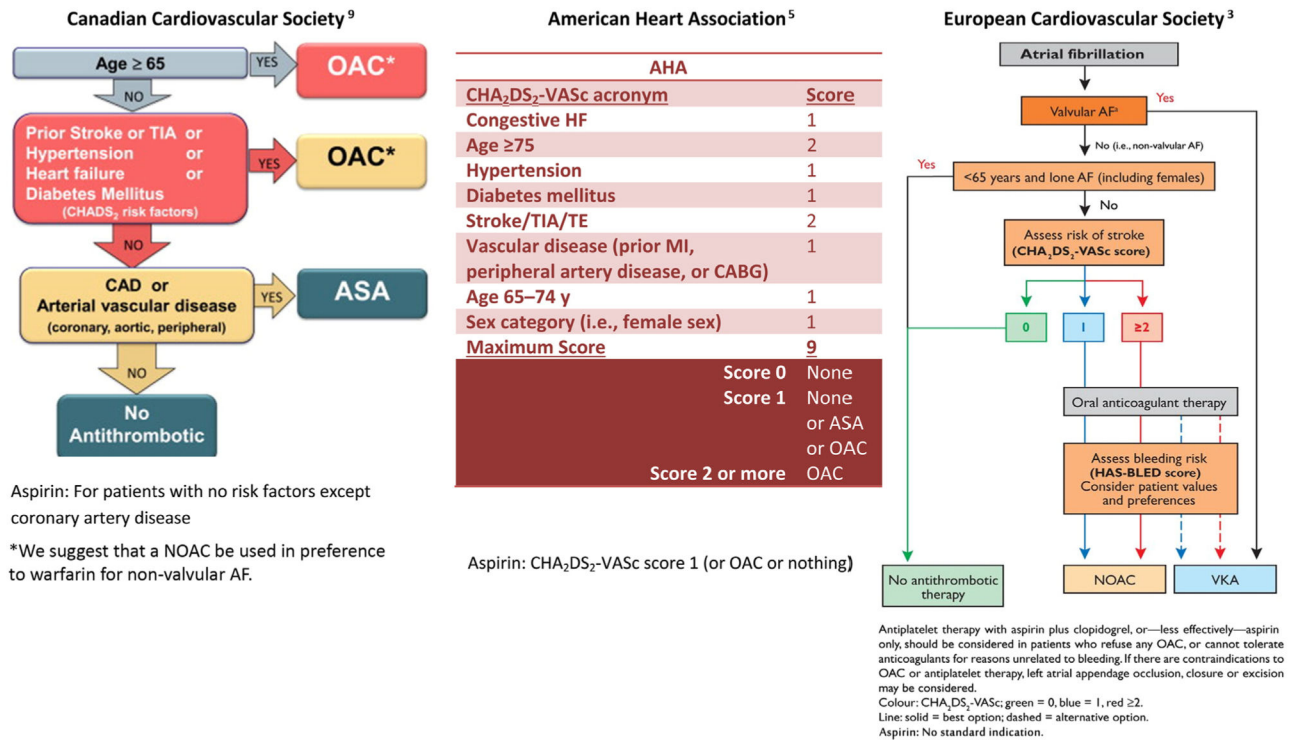




**Figure 1.** Management options for the hypotensive, conscious patient, which may be attempted to avoid immediate cardioversion. We recommend selecting only 1 of the 3 options; if not effective (pulse rate decreased and blood pressure increased or maintained), obtain expert consultation. If vital signs or level of consciousness worsens, proceed to immediate cardioversion.

1. Duration of atrial fibrillation >48 hour
2. High risk for stroke
  - a. Mechanical heart valve
  - b. Rheumatic heart disease
  - c. Recent stroke or transient ischemic attack
3. High risk for ventricular tachycardia/fibrillation
  - a. Digoxin toxicity
  - b. Severe hypokalemia

**Figure 2.** Contraindications to rhythm control (using either electrical or pharmacologic cardioversion).<sup>2,5,9</sup>



**Figure 3.** Recommendations for who should receive oral anticoagulation or aspirin across professional groups. Figures reproduced with permission from Oxford University Press on behalf of the European Cardiovascular Society and Elsevier, Inc on behalf of the Canadian Cardiovascular Society.

Letter	Acronym definition	Points
H	Hypertension*	1
A	Abnormal renal & liver function (1 point each)	1 or 2
S	Stroke	1
B	Bleeding (history or predisposition)	1
L	Labile INRs <sup>†</sup>	1
E	Elderly (age 65+)	1
D	Drugs or alcohol (1 point each) <sup>‡</sup>	1 or 2
Total score		/9

**Figure 4.**

HAS-BLED tool for assessing risk of bleeding (score of  $\geq 3$  indicating high risk of bleeding).

\*Hypertension defined as systolic pressure  $\geq 160$  mm Hg. <sup>†</sup>Labile INR defined as within therapeutic range  $< 60\%$  of the time. <sup>‡</sup>Drugs defined as antiplatelet drugs (eg aspirin, clopidogrel), alcohol  $\geq 8$  drinks per week.

**Table 1**

Common rate-control options in the ED: generally starting with an intravenous medication, followed by an oral medication in the same class (administered once the pulse rate is controlled [ $<100$  beats/min] with intravenous formulation, or 15 minutes after final intravenous administration).

Rate-Control Medication	Form	Standard Initial Dose*	Notes
<b><math>\beta</math>-Blockers</b>			
Metoprolol	IV	5-mg slow push/2 min; repeat every 5 min to max 15 mg	Maximal pulse rate reduction occurs at $\approx 5$ min
Metoprolol	PO	25 mg (twice a day)	May give 37.5 mg, 50 mg, or up to a total of 100 mg, depending on pulse rate response (peak response is at $\approx 1.5$ h)
Bisoprolol	PO	2.5–5 mg (once a day)	Good choice for patients with reactive airways
Carvedilol	PO	3.125 mg (twice a day)	Good choice for patients with a history of heart failure
<b>Nondihydropyridine calcium-channel blockers</b>			
Diltiazem	IV	$\sim 20$ -mg slow push/2 min (0.25 mg/kg); may give another 25 mg (0.35 mg/kg)	Maximal pulse rate reduction occurs at 2–7 min May switch to infusion 5–15 mg/h after second dose
Diltiazem	PO	120–240 mg (twice a day or once a day) <sup>†</sup>	
Verapamil	PO	40–80 mg (3 times daily) <sup>‡</sup>	More potential to cause hypotension than diltiazem

IV, Intravenous; PO, per os (by mouth).

\* The dose of oral medications may need to be titrated up in follow-up outpatient care, to obtain consistent resting (ventricular) pulse rate less than 100 beats/min.

<sup>†</sup> Higher doses are usually required for effective rate control with oral diltiazem. Because the dose may need to be altered, we recommend twice-daily dosing to start, which may be changed to once-daily dosing once the effective rate-control dose is determined.

<sup>‡</sup> We have limited experience using this medication and prefer diltiazem because of less hypotension and drug interactions. Available intravenously as well.

**Table 2**

Typical dose selection for oral anticoagulants initiated in the ED for patients without evidence of renal failure (eg, creatinine clearance >60 mL/minute), with instructions to follow up with the primary care provider.<sup>2,5,9</sup>

Oral Anticoagulant	Patient	Initiating Dose/Prescription
Warfarin	70 kg	5 mg once daily×3–5 days; obtain INR
	60 kg or age ≥ 80 y	2.5 mg once daily×3–5 days; obtain INR
<b>Novel oral anticoagulants</b>		
Dabigatran	70 kg	150 mg twice daily×1–2 wk
	60 kg or age ≥ 80 y	110 mg twice daily×1–2 wk US: 75 mg twice daily×1–2 wk*
Rivaroxaban	70 kg	20 mg once daily×1–2 wk
	60 kg or age ≥ 80 y	15 mg once daily×1–2 wk
Apixaban	70 kg	5.0 mg twice daily×1–2 wk
	60 kg or age ≥ 80 y	2.5 mg twice daily×1–2 wk

\* This is the Food and Drug Administration recommended dose, based on modeling studies; however, it has not been prospectively validated. Other countries use 110 mg twice a day as the lower dose.