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Atrial Fibrillation: Integrating New Guidelines into Practice

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The Canadian Cardiovascular Society (CCS), the European Society of Cardiology, and the American College of Cardiology Foundation/American Heart Association/Heart Rhythm Society (ACCF/AHA/HRS) have recently provided guidelines or focussed updates on the investigation and management of atrial fibrillation (AF). With an emphasis on the 2010 Canadian Guidelines, this issue of *Cardiology Rounds* will review highlights of the guideline-recommended strategies and treatments and putting these guidelines into practice using a hypothetical patient with AF.

"In theory, there is no difference between theory and practice. In practice, there is." – *Yogi Berra*

Case Report

The patient is a 73-year-old woman with no prior major illnesses. She presents to your outpatient office with a 3-month history of intermittent "fatigue, shortness of breath, and lightheadedness," which would begin any time of day, last 12-24 hours, and feel very unpleasant, as if she "had the flu." These episodes occur once every 1-2 weeks, and she feels well in between the episodes. After a few months, she consults you on a day when she is feeling particularly unwell. During the physical examination, you find that she has an irregularly irregular pulse. What do you do now?

The *sine qua non* of AF diagnosis is an electrocardiogram (ECG), preferably a 12-lead ECG. The pulse can be irregularly irregular because of frequent atrial or ventricular premature beats, or AF/atrial flutter. Subsequent treatment will in large part be directed at improving this patient's symptoms, and it is therefore imperative that the practitioner establish "symptom – rhythm correlation" so that both patient and practitioner can have accurate expectations regarding which symptoms are explicitly related to the atrial arrhythmia.

When AF is diagnosed, the practitioner should establish the potential cause of AF, and coexisting cardiovascular diseases, which may influence treatment strategies. Most importantly, blood pressure (BP) needs to be measured very carefully[1] since ≥60% of all patients with AF have hypertension as the primary cause or a co-factor.[2] It is reasonable to consider AF as a "complication" of hypertension in these patients, since the risk of major vascular events in patients with hypertension and AF is substantially higher than in patients with hypertension but no AF.[3,4] The history and physical examination should focus on the search for underlying structural heart disease, especially valvular disease, coronary artery disease (CAD) with prior myocardial infarction, peripheral vascular or cerebrovascular disease, and cardiomyopathy, as well as acute illnesses which may cause AF. Sleep apnea is a common and often overlooked etiological factor in AF, and can be present in 30%-50% of patients with AF, often but not always accompanied by snoring, and subjectively poor quality sleep. Alcohol is often considered an important cause of AF, but is rarely a sole cause except with clear-cut alcohol excess. An echocardiogram is an essential part of the initial investigation.

An important next step is the classification of AF into a pattern: paroxysmal, persistent, or permanent ("accepted"). This terminological labelling is useful, since paroxysmal AF is that type which will spontaneously self-terminate, whereas persistent AF episodes require cardioversion or pharmacological therapy to terminate the episodes. Permanent AF is a pattern in which the practitioners have decided that attempts to restore and maintain sinus rhythm are futile or unnecessary, and thus antiarrhythmic drug therapy or ablation for sinus rhythm restoration need no longer be considered.

The most important consideration in deciding on a long-term strategy for management in a patient with AF is the effect the rhythm disorder has on the patient's quality of life and well-being,

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Table 1: The Canadian Cardiovascular Society Severity of Atrial Fibrillation (SAF) Scale[7]

SAF Class	Effect on the patient's general quality of life (QoL)
0	**Asymptomatic** with respect to AF
1	Symptoms attributable to AF have **minimal** effect on patient's general QoL • Minimal and/or infrequent symptoms, or • Single episode of AF without syncope or heart failure
2	Symptoms attributable to AF have a **minor** effect on patient's general QoL • Mild awareness of symptoms in patients with persistent/permanent AF, or • Rare episodes (eg, less than a few per year) in patients with paroxysmal or intermittent AF
3	Symptoms attributable to AF have a **moderate** effect on patient's general QoL • Moderate awareness of symptoms on most days in patients with persistent/permanent AF, or • More common episodes (eg, more than every few months) or more severe symptoms, or both, in patients with paroxysmal or intermittent AF
4	Symptoms attributable to AF have a **severe** effect on patient's general QoL • Very unpleasant symptoms in patients with persistent/paroxysmal AF and/or • Frequent and highly symptomatic episodes in patients with paroxysmal or intermittent AF and/or • Syncope thought to be due to AF and/or • Congestive heart failure secondary to AF

a focus that is perhaps obvious but nonetheless crucial to emphasize. In this respect, AF is different from, for example, hypertension or diabetes where the disease being treated may be asymptomatic and has severe but potentially preventable long-term consequences. In AF, there is no strong scientific evidence that restoring and maintaining sinus rhythm delays or prevents complications of AF.[5,6] As a result, a primary goal of treatment is to assist patients in living with their disease to obtain the best possible quality of life. This includes both freedom from symptoms, minimizing adverse drug effects, and helping patients better understand their disease to minimize the impact of illness on well-being.

Since quality of life is complex and multifaceted, it is often thought to be difficult to measure. The CCS Severity in Atrial Fibrillation (SAF) Scale[7] is a simple and easily applied semi-quantitative measure of the effect of AF on an individual's overall well-being that is recommended to be used in the 2010 CCS AF guidelines (Table 1).[2] Similar in concept to the New York Heart Association functional class for heart failure symptoms, this scale can serve as an efficient "shorthand" for patient assessment, communication, and to facilitate treatment decisions. Simple ways to establish the SAF scale include questions such as, "What do you do during symptomatic episodes?" "Have you cancelled any planned activities because of the symptoms?" and "How much has the disease (and its treatment) affected your quality of life?" It is important to recall that quality of life can be very differently altered for the same degree of symptom frequency, severity, and duration in different subjects. For example, some patients have infrequent and mildly symptomatic episodes of AF, but may be so concerned about the consequences of an event that they refrain from leisure activities such as travel or previously pleasurable physical exercise.

In this context, patients are often advised to refrain from consuming caffeine or alcohol, and to avoid stressful activities/situations and exercise for fear of precipitating AF. Apart from alcohol excess in certain predisposed individuals, there is no evidence that any of these lifestyle factors promote AF, and patients often get substantial benefit from being reassured that it is not helpful or necessary to restrict these activities if they have AF.

Assessing Stroke Risk

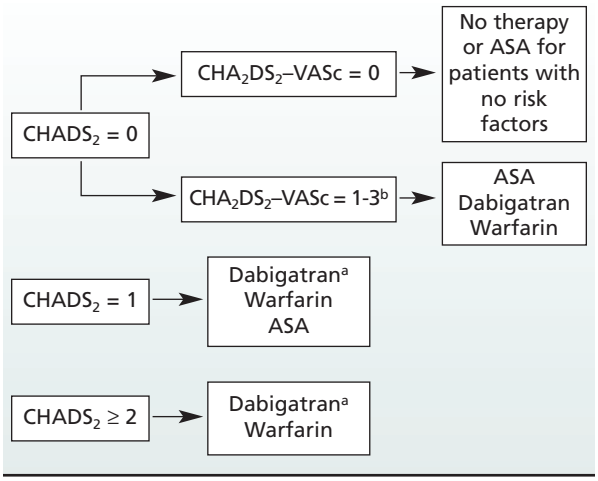
A necessary part of the initial evaluation is the assessment of the risk of stroke. In general, patients with AF are at a substantially increased risk of stroke compared to control populations without the arrhythmia. However, the risk of stroke in AF patients can be highly variable, from as low as ≤1% per year in the lowest-risk subgroups, to ≥15% per year in those at high risk.[8] Patients with AF and severe valvular disease, particularly mitral stenosis, are at very high risk of stroke and require systemic oral anticoagulation. For the largest group, patients with "non-valvular" AF, stroke risk will vary depending on the presence of well understood risk factors for stroke, which include advancing age, hypertension, diabetes, or heart failure, and very importantly a prior history of stroke or transient ischemic attack (TIA). This last risk factor is the most important, since a prior stroke or TIA identifies a patient at very high future risk of its recurrence, thus mandating systemic anticoagulation.

Risk factors that have not always been considered in risk-stratification schemes include female sex, and the presence of vascular disease such as cerebrovascular, coronary artery, or peripheral vascular diseases. The CCS guidelines recommend the well-established $CHADS_2$ classification[9] – Congestive heart failure, Hypertension, Age, Diabetes, and prior Stroke/TIA (2 points) – and the administration of oral anticoagulants to all patients with any of the risk factors; ie, a $CHADS_2$ score ≥1.[8] The European Society of Cardiology guidelines[10] amplify this recommendation slightly, in adding factors such as vascular disease, age between 65-75 years, and female sex to the risk factors (CHA_2DS_2-VASc).[11] For example, the patient illustrated above would receive 1 point each for female sex and

Table 2: Comparison of $CHADS_2$[9] and CHA_2DS_2-VASc[11]

Risk Factor	Score	Risk Factor	Score
$CHADS_2$		***CHA_2DS_2-VASc***	
Congestive heart failure	1	Congestive heart failure	1
Hypertension	1	Hypertension	1
Age ≥75 years	1	Age ≥75 years	2
Diabetes mellitus	1	Diabetes mellitus	1
Stroke/transient ischemic attack	2	Stroke/transient ischemic attack	2
Maximum score	**6**	Vascular disease	1
		Age 65-74 years	1
		Sex category (ie, female sex)	1
		Maximum score	**9**

Figure 1: Anticoagulant Use Based on $CHADS_2$ and CHA_2DS_2–VASc Scores



[a]Dabigatran is the preferred oral anticoagulant over warfarin in most patients; [b]Includes non-traditional risk factors of age 65-74 years, female sex, and vascular disease. European guidelines[9] recommend anticoagulation for ≥2 of these risk factors.

ASA = acetylsalicylic acid

age >65 years, independently of any of the "traditional" $CHADS_2$ risk factors (Table 2).

How Does One Manage Stroke Prevention in Practice?

New guidelines have simplified previous recommendations by recommending oral anticoagulation in most patients except for the small minority who have no risk factors for stroke; eg, male patients <65 years with no other risk factors (Figure 1). The higher the stroke risk, the greater the absolute benefit derived from oral anticoagulation, both compared to no treatment or compared to acetylsalicylic acid (ASA). Although ASA is probably superior to placebo, it is less effective than oral anticoagulation, and carries a nontrivial and underappreciated risk of serious bleeding.

If oral anticoagulation is contemplated, most patients should be treated with the newly available thrombin inhibitor, dabigatran, in preference over warfarin.[8] Evidence for this recommendation is derived largely from the Randomized Evaluation of Long-Term Anticoagulation Therapy (RE-LY) trial,[12] in which 150 mg bid of dabigatran resulted in lower stroke risk than warfarin, as well as a similar overall bleeding risk but a significantly lower risk of life-threatening or intracranial bleeding. Important caveats regarding dabigatran include a contraindication in the face of severe renal dysfunction, the absence of reversibility – ie, an "antidote" – and the inability to monitor compliance or routinely measure the extent of anticoagulation precisely; the latter is possible with warfarin therapy. Advantages associated with dabigatran include the absence of a need to monitor the international normalized ratio (INR), and a consistent anticoagulant effect not altered by the many factors that affect the intensity of anticoagulation under warfarin.

Case (cont.)

Investigations in our patient reveal that she has a BP of 145/95 mm Hg on repeated measurements, normal renal and hepatic function, and no symptoms of sleep apnea. She has no risk factors or symptoms suggesting coronary artery or peripheral vascular disease, and her exercise tolerance when not in AF is good. She is thus in $CHADS_2$ stroke risk category of 1 (CHA_2DS_2-VASc of 3), and, because the symptoms are affecting her activities, in SAF class 2. An echocardiogram shows normal left-ventricular size and function, and slight left-atrial enlargement (LA diameter of 4.2 cm). How do you treat this patient?

The initial care of patients with AF centres around improving quality of life and decreasing stroke risk. For the latter, therapy with dabigatran 150 mg bid is the most effective strategy, and recommended in the CCS guidelines.[8] (Note: Dabigatran is not currently covered by provincial drug formularies.) For the treatment of AF itself, an initial reasonable approach is to attempt rate control, to minimize the severity of episodes of AF without necessarily reducing their frequency. Most clinicians use beta-blockers for this purpose, although calcium channel blockers are also reasonable and may be less likely to be associated with adverse effects that limit exercise tolerance.[13] In addition, calcium blocker therapy is often preferred to beta-blockers as the initial treatment of hypertensive patients.

Case (cont.)

The patient is classified as having paroxysmal AF and the initial strategy chosen is rate control. The patient receives antihypertensive therapy (ramipril 5 mg daily), a beta-blocker (bisoprolol 5 mg daily), and dabigatran (150 mg bid). The causes and consequences of AF are carefully explained to her, and she is reassured that the disorder is not life-threatening and will not "cause a heart attack." She is also told that the primary object of treatment is to improve her general well-being, and not necessarily to stop altogether the episodes of AF. A specific "therapeutic contract" is made, depending on patient needs and desires. A follow-up plan is made, which will explicitly consist of reassessing patient well-

being in a few months, with a plan to change therapy if symptom improvement is inadequate for the patient's expectations.

The patient continues to have episodes of weakness, dyspnea, and fatigue, albeit less severe than before treatment. However, she reads on the Internet that "AF can cause stroke and represents an emergency." One month later, she develops another episode of symptomatic AF, and although she is only mildly symptomatic, she presents to a local emergency room. On ECG, she has AF with a ventricular rate of 100 beats/minute, and a BP of 130/85 mm Hg; the ECG is otherwise normal. She receives intravenous procainamide, but does not convert to sinus rhythm. She is admitted to hospital and plans are made for a cardioversion the following morning. The arrangements are made, but one hour before the planned cardioversion she spontaneously reverts to sinus rhythm and is discharged with no change in medication.

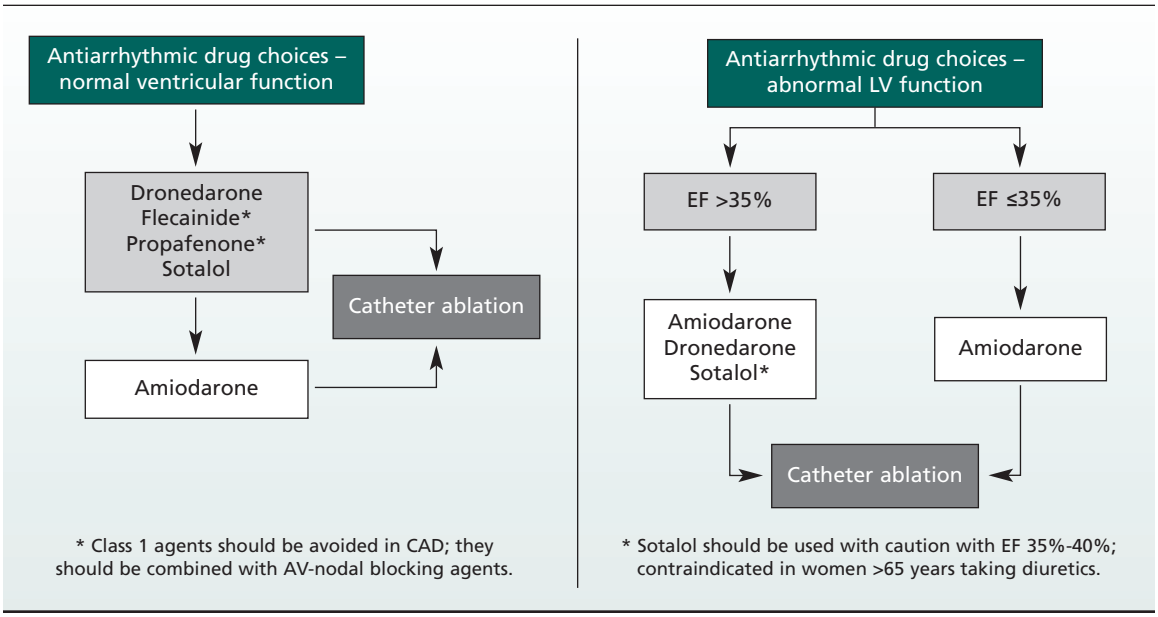
The above scenario is common, and is likely avoidable. There are approximately 20 000 emergency-room visits for the primary diagnosis of AF in Ontario per year, and approximately 40% of these patients are admitted to hospital.[14] The recent CCS guidelines suggest that hospital admission be restricted to patients who have substantial hemodynamic compromise, or symptoms consistent with heart failure or acute myocardial ischemia.[15] None of these criteria are present in our patient. She is protected from stroke by receiving continuous systemic anticoagulation. The pattern of AF has previously been observed to be paroxysmal, and therefore it is expected that the current episode will terminate spontaneously without specific therapy. Even if it does not, outpatient cardioversion can be arranged following discharge from the emergency room. If patients are distressed and very symptomatic, additional rate control can be administered given the empiric observation that slowing ventricular response is usually associated with symptomatic improvement. However, rate control does not necessarily have to be intensified in patients with no or acceptable symptoms and a faster than normal heart rate. The new CCS guidelines, in patients with persistent or permanent AF, recommend ventricular rate control therapies to achieve a resting heart <100 beats/minute, or as guided by symptoms.[16] "Pushing" rate control therapies to achieve the resting heart rate targets recommended in prior guidelines (<80 beats/minute) is unnecessary and no longer recommended. Similar to the Canadian guidelines, the updated ACCF/AHA/HRS 2010 guidelines[17] recommend: "Treatment to achieve strict rate control of heart rate (<80 bpm at rest or <110 bpm during a 6-minute walk) is not beneficial compared to achieving a resting heart rate <110 bpm in patients with persistent AF who have stable ventricular function (left ventricular ejection fraction 0.40) and no or acceptable symptoms related to the arrhythmia, though uncontrolled tachycardia may over time be associated with a reversible decline in ventricular performance. (Level of Evidence: B – New recommendation)."

Case (cont.)

The patient returns to your office, and is dissatisfied with the outcome of therapy thus far. She finds the frequency and severity of symptomatic episodes bothersome and very unpleasant, and has restricted her activities as a result. What do you do now?

Among patients who have inadequate response to the rate-control strategy, the CCS guidelines recommend therapy to maintain sinus rhythm, using antiarrhythmic drugs initially, and if necessary radiofrequency ablation in selected cases.[16] Importantly, the target outcome for the

Figure 2: Therapy Choices for Rhythm Control, Stratified by LV Function



CAD = coronary artery disease; AV = atrioventricular; LV = left ventricular; EF = ejection fraction

rhythm-control strategy is a reduction in, but not necessarily the elimination of, all episodes of symptomatic AF. Antiarrhythmic drugs are employed to reduce the frequency and duration of episodes of AF, and the choice of available antiarrhythmic drugs as outlined in the CCS guidelines is illustrated in Figure 2.

In this particular case, one cannot be certain that the patient does not have CAD, given her age and the risk factor of hypertension, although some would argue that the absence of symptomatic myocardial ischemia during episodes of AF represents a "negative stress test." Class 1c drugs (propafenone and flecainide) should thus be used with caution, since they are contraindicated in patients with CAD. Drugs that in the past were the most commonly used for rhythm control in Canada included sotalol and amiodarone.[18] Many practitioners have concerns about the use of sotalol as initial therapy in older women, since female sex and older age (often associated with renal dysfunction) are important risk factors for proarrhythmia with sotalol. In addition, doses of sotalol that are likely to be effective in the prevention of AF recurrence are higher than those typically used in practice, and the 240-360 mg per day required relatively frequently causes symptomatic bradycardia, fatigue, or proarrhythmia.[19] Amiodarone is a highly effective antiarrhythmic agent, but carries a substantial long-term burden of toxicity and is therefore no longer recommended as first-line antiarrhythmic therapy in Canadian or international guidelines.

Case (cont.)

The patient undergoes an exercise stress test with radionuclide scintigraphy, because of the presence of hypertension and the possibility of a false-positive result. This test shows a small area of reversible perfusion defect in the inferior wall at a relatively high workload. The patient does not have chest pain during the stress test.

The presence of coronary disease indicates that Class Ic antiarrhythmic drugs such as propafenone and flecainide are contraindicated. Attention to CAD modifiable risk factors is important, including measurement and treatment of low-density lipoprotein cholesterol, achieving a normal body weight, an exercise regimen as tolerated, absence of smoking, and identification of diabetes, if present. Although beyond the purview of this document, coronary angiography is likely not indicated in an asymptomatic patient with mild myocardial ischemia at a high workload.

The patient receives sotalol 80 mg tid, and her bisoprolol is stopped. At a follow-up examination 3 months later, she reports fewer episodes of dyspnea and dizziness; only about 1 per month lasting 24 hours. However, on close questioning states she is "tired all the time" and does not have sufficient energy to engage in everyday activities including shopping, cooking, and cleaning. Her ECG shows sinus rhythm at 47 beats/minute. What do you do now?

The symptoms are likely due to an adverse effect of the sotalol. Although the drug has been effective in reducing the number of episodes of presumed AF, antiarrhythmic effectiveness is not synonymous with patient-related benefit from a drug, since her overall quality of life is if anything worsened. Sotalol is discontinued and she is placed on dronedarone 400 mg bid.

Case (cont.)

The patient returns again to clinic 3 months later, saying that in general she feels much better, although she reports that she continues to have infrequent episodes (once every ~6 weeks) of mild fatigue, weakness, and dizziness that last about 12 hours. By chance, she is having one of these episodes during the current visit. An ECG shows AF with a ventricular rate of 85 beats/minute and no other abnormalities. Her BP is 130/85 mm Hg. She has read more Internet reports regarding the association of AF with risk of stroke, and that ablation can "cure AF." What do you do now?

Appropriate management of patients with AF requires a carefully detailed explanation of the risks and benefits of all available therapies. The primary goals of AF treatment are improving patient well-being, reducing morbidity and hospitalizations, and reducing the risk of stroke. The only therapy that has been documented, in a blinded randomized study, to reduce hospitalizations and cardiovascular death in AF is dronedarone, as shown in the ATHENA (A placebo-controlled, double-blind, parallel arm trial to assess the efficacy of dronedarone 400 mg bid for the prevention of cardiovascular hospitalization or death from any cause in patients with AF/atrial flutter) study.[20] The updated ACCF/AHA/HRS 2010 AF guidelines[17] recommend that "Dronedarone is reasonable to decrease the need for hospitalization for cardiovascular events in patients with paroxysmal AF or after conversion of persistent AF. Dronedarone can be initiated during outpatient therapy. (Level of Evidence: B – New recommendation)." Appropriate management consists of a careful balancing of treatment risks and benefits with patient wishes and desires, and a thorough understanding of the balance between treatment expectations and the likely outcomes. The patient is informed that radiofrequency ablation ("pulmonary vein isolation") is a useful procedure, but is generally reserved for situations where drug treatment is clinically ineffective. This is because one-half of patients require 2 procedures, each of which is associated with a 2%–4% of serious morbidity, and because 20%–25% of patients do not receive therapeutic benefit from the ablation procedure.[21] In addition, there is no good evidence that ablation, even if completely successful, obviates the need for lifetime stroke prevention with anticoagulation.

Upon reflection, the patient decides to defer radiofrequency ablation and decides to attempt to resume her previously normal lifestyle.

Case (cont.)

The patient is seen in follow-up 1 year later. She continues to have infrequent (once every 2–3 months) episodes of mild fatigue and lightheadedness lasting about 12 hours, but is carrying on a normal life otherwise and is reassured that her risk of serious morbidity related to the AF is very small. She understands that it is unnecessary to present to an emergency department if symptoms recur. The

need for adherence to her medical regimen, close follow-up of her BP, and general measures to reduce the risk of complications of CAD are again emphasized.

Conclusion

AF has many facets and each patient is unique. Care of AF patients can be simplified and made more consistent if one remembers the cardinal features of AF management:

- Establish the causes and underlying diseases associated with this patient's AF, and the pattern
- Assess the consequences of AF to the patient's quality of life; all subsequent therapies will be evaluated against this benchmark
- Assess and quantify stroke risk, and treat accordingly

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