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LEARNING OBJECTIVES

Upon successful completion of this lesson, you should be able to:

1. define atrial fibrillation, its diagnosis, signs and symptoms
2. customize the goals of therapy for a patient with atrial fibrillation
3. describe the difference between rate and rhythm control
4. risk stratify each patient in terms of risk of cardioembolic stroke, and in doing so, select the appropriate antithrombotic agent
5. explain the importance of adequately anticoagulating patients with AF

INSTRUCTIONS

1. After carefully reading this lesson, study each question in the post-test and select the one option you believe is the best answer. Although more than one option may be considered acceptable, only one option is the best answer.
2. To pass this lesson, a grade of at least 70% (14 out of 20) is required. If you pass, your CEU(s) will be recorded with the relevant provincial authority(ies). (Note: some provinces require individual pharmacists to notify them.)

ANSWERING OPTIONS

- A. For immediate results, answer online at www.pharmacygateway.ca.
- B. Mail or fax the printed answer card to (416) 764-3937. Your reply card will be marked and you will be advised of your results within six to eight weeks in a letter from *Pharmacy Practice*.

Atrial Fibrillation: An Overview for the Community Pharmacist

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First described in 1909, atrial fibrillation (AF) is the most common sustained cardiac arrhythmia in the general population.¹ Although AF is less common in patients under 60 years of age, prevalence in adults doubles with each decade, and is estimated to occur in 10% of those > 80 years of age.² Relative to patients in sinus rhythm, the mortality rate of patients with AF doubles and is related to the severity of heart disease.² Once patients are diagnosed with AF, they are often poorly informed of the condition. The most feared complication of AF is stroke. Data show that appropriate prophylaxis for stroke is prescribed only for a minority of patients³ and, of those receiving it, less than half are treated to an appropriate target.⁴ Pharmacists are optimally positioned within the healthcare system to positively impact the care of patients with AF.

The purpose of this lesson is to provide an overview of AF, reviewing pharmaco-

logic options for the management of patients seen in a community setting.

**Atrial fibrillation—
an irregularly, irregular
rhythm**

Atrial fibrillation is classically described as an “irregularly, irregular” rhythm.⁵ To appreciate this, a basic understanding of normal conduction through the heart is necessary. Impulse generation begins in

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the sinoatrial node, located in the upper right atrium, and is transferred to the left atrium to allow contraction of both atria. The impulse is then gathered at the atrioventricular (AV) node which, akin to a “gatekeeper,” regulates impulse conduction from the atria to the ventricles. The conducted impulse then travels through the ventricles, with resultant ventricular contraction. Assessment of one’s pulse is reflective of ventricular contraction.

USEFUL FACT

Ejection fraction (EF) is defined as the proportion of blood ejected from the left ventricle with each contraction. This measure is done from the left ventricle as this is reflective of oxygenated blood reaching the body. A “normal” EF is approximately 60%. Left ventricular dysfunction (commonly referred to as heart failure) is defined as an EF < 35-40%.⁶

For patients in sinus rhythm, this is a very coordinated process with atrial contraction followed by ventricular contraction. With AF, however, this process is not synchronous. The atrial:ventricular rate of contraction is not 1:1 (as with sinus rhythm), but typically 2–4:1.⁵ The atrial rate of contraction for patients with AF exceeds 350 beats per minute, while ventricular contraction occurs at a rate less than 180 beats per minute (any faster would not be compatible with life). Atrial contraction at a rate this fast is simply ineffectual, resulting in sluggish blood flow in the atria (placing patients at risk for clot formation that could lead to stroke) and a reduction in the proportion of blood flow into the ventricles occurring as a result of atrial contraction.

While it is common for patients with AF to have a rapid ventricular response, some patients will not have tachycardia.⁵ Due to its gatekeeper role, the AV node typically lets every third or fourth atrial impulse pass

through to the ventricles because it is in a “refractory period.” The refractory period is a time interval during which the AV node is either unable to accept any impulse or will only partially accept an impulse. AF is an irregularly, irregular rhythm because 1) the rate of impulse conduction is irregular and 2) the strength or amplitude of the impulse is irregular (with partial impulse conduction during the refractory period).

table 1
Predominant patterns of AF¹⁰

Pattern	Description
paroxysmal	typically lasts < 24 hours and is self-terminating within 7 days of recognized onset
persistent	not self-terminating within 7 days or requires termination by pharmacologic or electric means
permanent	cardioversion has failed or clinical judgment deems not to pursue cardioversion
recurrent	2 or more episodes of paroxysmal or persistent AF

DIAGNOSIS/ETIOLOGY OF AF

AF is underdiagnosed and undertreated in the community.⁷ Evidence shows that simply assessing a patient’s pulse is useful to rule out AF,⁸ and routine screening of the patient’s pulse has been the only strategy to improve detection.⁹ Once an irregular pulse is identified, patients should have an objective assessment of their rhythm⁷⁻⁹ typically with a 12-lead electrocardiogram (ECG), although a Holter monitor (ambulatory recording of electrical activity of the heart) may be used.

The cause of AF is often debated, with multiple triggers and substrates contributing. However, it generally can be broken down into cardiovascular and non-cardiovascular causes.¹⁰ Cardiovascular causes

include electrical anomalies (believed to originate in the pulmonary veins), congenital heart disease, and factors that increase pressure in the left atrium (such as hypertension, heart failure or valvular disease). Non-cardiac causes that may be reversible include autonomically-mediated (vagal) AF, hyperthyroidism, pulmonary disease (e.g., sleep apnea, chronic obstructive pulmonary disease, pneumonia), and toxin-related (e.g., caffeine, alcohol) etiologies. Some patients may have AF post-operatively in cases of major vascular, abdominal or thoracic surgery.¹¹

Upon identifying AF, determination of the predominant pattern (paroxysmal vs. persistent) should be performed (Table 1).¹⁰ It may not be possible to determine the pattern with the initial presentation. Furthermore, the pattern may change over time. While paroxysmal AF accounts for < 40% of all AF cases, it is notable that as many as one-third will progress to persistent AF one year after onset.¹²

SIGNS AND SYMPTOMS

The signs and symptoms of AF range from asymptomatic to truly disruptive.^{13,14} Should a patient have symptoms with some episodes of AF, they are likely not to have symptoms with every occurrence of AF. Patients with persistent AF experience times of being both symptomatic and asymptomatic. Most patients with AF experience vague symptoms of fatigue and a general decline in exercise tolerance.¹⁵ Patients commonly report feeling “butterflies in their chest” and may have symptoms of palpitations, chest pain, lightheadedness, dyspnea or syncope. Assessing the impact that symptoms have on a patient with AF is central in the management of this arrhythmia.

GOALS OF THERAPY

The three therapeutic considerations in each patient with AF include rate control,

rhythm control, and thromboembolic (stroke) prophylaxis.¹³ For rate control, the goal of therapy is to control the ventricular response rate by ensuring the AV node is an effective gatekeeper. The goal of therapy for rhythm control, if pursued, will vary depending on whether the patient has paroxysmal or persistent AF.^{10,16} For paroxysmal AF, the goal is to minimize the frequency and associated symptoms of paroxysms. The goal with persistent AF is to achieve and maintain sinus rhythm. While not all patients will require rate or rhythm control and some will require both, all patients should have their stroke risk assessed with appropriate antithrombotic therapy implemented.¹³ Further, evidence reveals there is similar stroke risk for patients with paroxysmal and persistent AF,^{17,18} simplifying the assessment for antithrombotic therapy.

RATE CONTROL

The goal of rate control is to achieve an appropriate ventricular response rate during rest and exercise, while avoiding extremes of bradycardia and tachycardia. With rate control alone, the irregular or chaotic atrial activity remains, leaving the patient still at risk for clot formation in the atrium. Heart rate targets generally should range between 60–80 beats per minute at rest and 90–115 beats per minute during exercise, with patient symptoms dictating targets.¹⁵

Management of rate control encompasses an assessment of the patient's baseline heart rate at rest and exercise, their symptoms and well-being, and comorbid medical conditions.^{15,19} Some patients with AF will not have an elevated heart rate and, hence, will not require pharmacologic therapy for rate control. For those requiring therapy, options consist of nondihydropyridine calcium channel blockers (NDHP CCB), beta-blockers (BB) and digoxin (Table 2).¹⁹

Matching the patient with the most appropriate agent is key.

Data indicate that more patients receiving BB monotherapy achieve adequate rate control compared to those receiving monotherapy with a NDHP CCB.¹⁹ BBs, however, are more likely to reduce exercise tolerance making them a less attractive option in active patients. While BBs are front-line therapy for patients with heart failure (HF), dosing of these agents requires slow and careful titration to ensure patients are able to tolerate them. Given this, it is often problematic to reach BB doses sufficient to suppress the AV node in patients having HF and AF. Digoxin therapy is less effective as monotherapy and used as an adjunct with BB or NDHP CCB therapy.¹⁹ Further, in situations of high sympathetic tone (such as exercise), the AV nodal blocking effects of digoxin can be over-ridden, making it a more attractive option in sedentary patients. Combinations of BBs and NDHP CCBs should be avoided due to the potential for bradycardia, hypotension and conduction defects (e.g., complete AV block).¹⁹

RATE VERSUS RHYTHM CONTROL

With the emergence of antiarrhythmic agents 50 years ago, rhythm control for AF became preferred over rate control based on the belief that patients would be less symptomatic, have a better quality of life and not require thromboembolic prophylaxis.²⁰ This, however, came into question 20 years ago and clinical trials began to test this theory. The publication of the AFFIRM trial in 2002²¹ dramatically altered practice philosophies for the management of AF. This trial randomized patients to receive rhythm control (with stroke prophylaxis as per the investigator's direction) or rate control (with long-term warfarin therapy). The primary endpoint was all-cause mortality, and the results revealed a trend toward increased mortality in those receiving rhythm control; contrary to results anticipated by many. Further, more patients receiving rhythm control were hospitalized, likely due to adverse drug reactions. The results of this trial made clinicians critically evaluate the use of antiarrhythmic drugs in the management of AF.

To apply the findings of AFFIRM to your patients, it is essential to consider the

table 2

Rate-controlling agents¹⁹

Drug	Dose	Comments
nondihydropyridine calcium channel blockers • diltiazem • verapamil	• 120–360 mg* daily • 180–480 mg† daily	• consider if young or active, have normal systolic function or contraindication to beta-blockers • less long-term efficacy data with verapamil relative to diltiazem
beta-blockers • atenolol • bisoprolol • metoprolol	• 25–200 mg daily • 2.5–20 mg daily • 25–200 mg BID	• consider if coronary artery disease with myocardial infarction, left ventricular dysfunction
digoxin	• 0.125–0.5 mg daily	• consider in sedentary patients or those with symptomatic heart failure as monotherapy or combination therapy • enhances the efficacy of beta-blockers and nondihydropyridine calcium channel blockers

*controlled dose formulation, †sustained release formulation

table 3

Rhythm-controlling agents ¹⁶			
Drug	Dose	Patient population	Side effects*
amiodarone** (Class III)	100–200 mg daily	consider if: • left ventricular dysfunction • hypertension with LVH	<ul style="list-style-type: none"> • photosensitivity • thyroid dysfunction • hepatic toxicity • neuropathy • pulmonary fibrosis • corneal microdeposits • GI intolerance
sotalol (Class III)	40–160 mg BID	consider if: • hypertension with LVH • CAD contraindicated in renal failure	<ul style="list-style-type: none"> • bradycardia • torsades de pointes • dyspnea • fatigue • dizziness • headache
propafenone (Class 1C)	150–300 mg TID	consider if: • structurally normal heart • CAD without left ventricular dysfunction • hypertension with LVH	<ul style="list-style-type: none"> • dizziness • nausea/vomiting • unusual taste • constipation • proarrhythmia (including atrial) <ul style="list-style-type: none"> • HF • VT • bradycardia
flecainide (Class 1C)	50–200 mg BID	indicated for paroxysmal AF consider if: • structurally normal heart • hypertension with LVH contraindicated with left ventricular dysfunction	<ul style="list-style-type: none"> • worsening of HF • new or worsening of ventricular or supraventricular arrhythmias • vision disturbance • headaches • nausea • dyspnea • fatigue

*not inclusive; patients may experience other side effects or adverse events with these drugs
**AF is an off-label indication
CAD=coronary artery disease; HF=heart failure; GGastrointestinal; LVH=left ventricular hypertrophy; VT=ventricular tachycardia

variation in practice exists with rhythm control for AF; there is not one practice algorithm that is followed. There are three options for cardioversion—antiarrhythmic drugs, electrical cardioversion and ablation.¹⁴ This lesson will focus on antiarrhythmic drugs and only define the latter two options. Further, only drugs commonly used on a long-term basis to maintain sinus rhythm will be highlighted.

Ablation is a relatively new, emerging strategy showing promising results.¹⁴ This invasive, catheter-based procedure delivers energy to aberrant conduction pathways believed to be causing AF.

Selection of antiarrhythmic drug therapy must be tailored to the patient (Table 3).¹⁶ The more heart disease (HF, coronary heart disease or hypertension) a patient has, the fewer options exist for maintenance of sinus rhythm with pharmacologic therapy. Amiodarone is commonly used in those having left ventricular dysfunction, and is frequently reserved as a second choice (to sotalol) for those having normal ventricular function with either coronary artery disease or hypertension.^{16,24} Where appropriate, some clinicians will often try to use sotalol before amiodarone, given the side effect profile of amiodarone and its exceptionally long half-life. Use of sotalol is cautioned in elderly patients with renal failure owing to the concern of accumulation and risk of arrhythmia formation.

Direct current (electrical) transthoracic cardioversion is a procedure wherein energy is delivered through electrodes placed on the patient's chest.¹³ This 'resets' the rhythm allowing the sinoatrial node to take over the pace of the heart.

characteristics of patients in this trial. To be eligible for the AFFIRM trial patients had to be able to have either rate or rhythm control implemented, therefore, were not symptomatic with AF requiring cardioversion. These patients tended to be elderly without HF and had either recurrent or persistent AF.

So who should have rhythm control attempted at this time? Simply stated, if a patient has symptoms severe enough to impact their quality of life or if this is the first time a patient has been found to be in AF (and it is unlikely they have had AF for a long period of time), it is reasonable to proceed with a rhythm control strat-

egy.^{16,22,23} Patients more likely to respond tend to be younger, have less heart disease (no hypertension or left ventricular hypertrophy, no HF, no mitral valve disease and have a small left atrium that is less than 55 mm in diameter), have not failed prior attempts at cardioversion, have AF for a short period of time (less than 6–12 months) or to have paroxysmal AF.^{16,22,23}

RHYTHM CONTROL

Rhythm control has two elements: one is to convert or 'reset' the chaotic atrial activity to sinus rhythm (referred to as cardioversion), the other is to maintain sinus rhythm long-term.¹⁶ Tremendous

Although sotalol is a BB, it should be thought of as an antiarrhythmic and never used for rate control alone due to risk of arrhythmias. Amiodarone, too, has rate-controlling properties, but should not be used for this sole purpose. In general, Class I agents (flecainide and propafenone) should not be used in patients with left ventricular dysfunction due to concerns of exacerbating this condition.

Overall, amiodarone is more efficacious than other agents in maintaining sinus rhythm long-term, but its use is often limited, secondary to the non-cardiac side effect profile.¹⁶ Agents other than amiodarone have similar efficacy with, at best, approximately 50% of patients maintaining sinus rhythm after one year of therapy.¹⁶

Thromboembolic (stroke) prophylaxis

The most feared complication of AF is cardioembolic stroke.⁵ With AF, the atria are beating so fast (> 350 beats per minute) that the ineffective atrial contraction creates a milieu of stasis, posing the risk of clot formation. The vast majority (> 90%) of emboli form against the left atrial appendage wall—this appendage offers further stasis given its structure as an outpouching of the left atrium.²⁵ Patients with AF are at risk of stroke (and not pulmonary embolism) because the embolism dislodges from the left atrium and is pushed out to the circulation through the left ventricle. Stroke occurring as a result of AF is referred to as cardioembolic because the embolism originates from the heart. Every patient with AF should be assessed for antithrombotic therapy to avoid cardioembolic stroke. Informed antithrombotic choices must balance both the individual patient's risk of stroke against their risk of bleeding.

RISK STRATIFICATION FOR CARDIOEMBOLIC STROKE

The risk of cardioembolic stroke in patients with AF varies depending upon the presence of valvular heart disease, whether the patient's had a prior stroke, transient ischemic attack (TIA) or systemic embolism and any other risk factors for heart disease.²⁶ Upon risk stratifying an individual patient, it is important to determine what the patient's risk of stroke is without warfarin therapy and balance this with their overall risk of bleeding while taking warfarin. Clearly, the reduction in stroke risk must exceed the risk of bleeding in order for warfarin to be appropriate.

Patients with AF due to valvular heart disease have a very high baseline risk of stroke,^{27,28} as do patients requiring secondary prophylaxis (i.e., those with a history of stroke, TIA or systemic embolism).²⁹ These patient populations should be prescribed warfarin therapy, even if they also have substantial bleeding risk.

If the patient has not had a stroke, TIA or systemic embolism and has non-valvular AF, they may be candidates for either warfarin or acetylsalicylic acid (ASA) prophylaxis, depending upon their baseline risk of stroke.^{14,26} If the patient has two or more of the following, they are high risk and warfarin is strongly recommended: age > 75 years, hypertension, diabetes or moderate to severe left ventricular dysfunction.²⁶ Patients having only one of the aforementioned risk factors are at intermediate risk for stroke, and warfarin is recommended, but ASA is an option in this group.²⁶ For patients that have no risk factors for stroke that are < 75 years of age, ASA 75–325 mg daily is recommended.²⁶ Should there be no clinical history of heart disease or objective determination of structural heart disease, and the patient is < 60 years of age, they are classified as having "lone" AF.¹⁴ With lone AF, the risk of stroke without warfarin therapy is very small (~1.3%/year)³⁰ and ASA therapy is suggested.

Unlike for rate and rhythm control, the predominant pattern of AF is not relevant in evaluating the most appropriate anti-thrombotic agent for stroke prophylaxis.¹⁸ Further, data reveal that patients with paroxysmal and persistent AF have a similar risk for stroke.^{18,31}

CARDIOVERSION – RISK OF THROMBOEMBOLIC STROKE

Regardless of the strategy used for cardioversion (drug, electrical, ablation), appropriate peri-procedural anticoagulation therapy must be implemented. All patients having scheduled cardioversion done, regardless of their risk stratification for stroke, should be anticoagulated.²⁶ Patients must have at least three weeks of therapeutic International Normalized Ratios (INRs) prior to, and four weeks following, a successful procedure wherein sinus rhythm is maintained. The former is thought to provide enough time to prevent new clots from forming and allow existing clots to firmly adhere against the atrial wall. For the latter, it takes at least four weeks for atrial contractility to resume (minimizing stasis of blood flow). It is common, however, for these patients to be anticoagulated for at least 3–6 months or even lifelong, owing to the risk of recurrence of asymptomatic AF.^{13,21} Should warfarin be discontinued following successful cardioversion, clinicians ought to ensure patients are still in sinus rhythm via an objective assessment (e.g., ECG).

The pharmacist's role in managing AF

Pharmacists are well-positioned within the healthcare system to have an impact on the management of patients with AF. Depending upon their setting and scope of practice, the pharmacist's role may range from delivering education to proactively managing the patient's care. Data shows that AF is underdiagnosed,⁷ and screening for AF is best done by assessing a patient's pulse,^{8,9}

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clearly a role a pharmacist could assume. Upon detection of an irregular pulse, collaboration with other healthcare providers (i.e., physician colleagues) should be done to render a diagnosis—done through objective assessment (typically an ECG). All patients with AF should have appropriate antithrombotic therapy initiated—this is a role many pharmacists either can, or already actively, fulfill.

To risk stratify patients with AF for stroke risk, simply ask about their medical history (ascertain any issues such as heart valves, history of stroke, TIA, hypertension, diabetes, HF and patient age) to assist with this. Upon identifying a patient that may benefit from warfarin therapy, pharmacists are well-positioned to educate patients and healthcare providers regarding the need for warfarin. These patients can be referred to an anticoagulation clinic—services dedicated to using systematic processes to optimize the care of these patients. These clinics are becoming increasingly common in Canada. To locate a clinic near you, refer to the Anticoagulation Forum website (www.acforum.org).

Once warfarin is prescribed for a patient with AF, their INR should be targeted to 2.5 (range 2.0–3.0).^{14,26} Case-controlled data reveal that compared to having an INR of 2.0, the risk of having a stroke doubles with an INR of 1.7, and triples with an INR of 1.5, highlighting the importance of avoiding sub-therapeutic anticoagulation.³² Clinicians should target the mid-point of the range to minimize both sub-therapeutic and supra-therapeutic anticoagulation.

Upon filling a prescription for warfarin, the pharmacist has an opportunity to inquire about the indication for therapy and what drug information was provided by their prescriber. In doing so, this would facilitate the ability to provide supplemental information. Pharmacists can provide education encompassing the benefits of warfarin therapy, the rationale and need for

regular INR testing and the multitude of factors impacting warfarin therapy such as concomitant medical conditions, lifestyle factors (e.g., alcohol, caffeine), and changes to concomitant drug therapies.


A comprehensive list of drug interactions is beyond the scope of this article, however, general principles should be addressed. Warfarin is extensively metabolized by the liver. The S-isomer of warfarin is more potent, having an activity 4–8 times greater than the R-isomer.³³ The S-isomer is metabolized by the cytochrome P450 2C9 isoenzyme, making it most relevant for drug interactions attributable to induction or inhibition of hepatic isoenzymes. These interactions tend to be delayed, so reassessing warfarin therapy in 3–5 days is strongly recommended; especially for those known to interfere with INR. To manage drug interactions, one must also assess the potential for protein displacement, impact on metabolism/catabolism of clotting factors, alteration in vitamin K status within the gastrointestinal tract (e.g., antibiotics), and any potential contribution toward increasing a patient's bleed risk (e.g., antiplatelets).

Different anticoagulation clinics might use different approaches but the following describes how one Canadian clinic encompasses several elements in the management of warfarin drug interactions.³⁴ The first step is to assess the patient's baseline risk of clotting (e.g., stroke if the indication is AF) and overall risk of bleeding. This provides information related to the potential aggressiveness of anticoagulation. By way of example, if a patient has AF due to a valvular cause, the risk of stroke in the setting of sub-therapeutic anticoagulation is high. If this patient does not have risks for bleeding, any potential interaction would be managed conservatively to ensure therapeutic anticoagulation is sustained. Second, the current anticoagulation status of the patient is reviewed to determine the

degree of alteration in dosing that may be necessary. Next, the timing of the interaction and extent of the interaction published in the literature or experienced clinically is used to guide warfarin dosage alteration and timing for reassessment of the INR. The clinic has only a handful of medications that mandate an immediate reduction in warfarin therapy, such as trimethoprim/sulfamethoxazole, metronidazole, fluconazole (list not all inclusive). For others, like amiodarone, the practice is to determine if loading doses were prescribed (if so, the interaction will be seen sooner) and monitor these patients more consistently (i.e., weekly) with gradual warfarin dosage reductions, as the effects of amiodarone ensue.

For a general overview of drug interactions with warfarin, the reader is referred to David Juurlink's review.³⁵ The Thrombosis Interest Group of Canada website also has information on warfarin for health professionals,³⁶ as well as downloadable warfarin information for patients.³⁷ Clearly, the role of the pharmacist in managing AF varies, but could impact the life of this population.

Summary

Atrial fibrillation is a very common irregularly, irregular rhythm. Therapeutic considerations for AF include rate control, rhythm control and stroke prophylaxis. Should pharmacologic therapy be necessary for rate control, it is imperative to match the agent with the patient's concomitant conditions. If the patient is symptomatic or newly diagnosed with AF it may be appropriate to pursue rhythm-controlling strategies. Lastly, all patients should be risk stratified to ensure appropriate antithrombotic therapy is prescribed. Pharmacists are well-positioned within our healthcare system to assist in the screening, risk stratification for stroke and delivery of care for patients with AF. 

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Questions

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1 Which of the following statements about AF is incorrect?

- a) AF is the most common sustained arrhythmia in the general population.
- b) The mortality of patients with AF is about the same as those in sinus rhythm.
- c) Stroke is the most feared complication of AF.
- d) The incidence of AF increases with age.

2 Atrial fibrillation is classically described as an irregularly, irregular rhythm. Which of the following statements is correct?

- a) All patients with AF will have an atrial rate > 350 bpm as well as a rapid ventricular response rate > 90 bpm.
- b) In AF, there are consistently two or three atrial contractions for every ventricular contraction.
- c) In AF, both the rate and amplitude or strength of ventricular contractions are irregular.
- d) Paroxysmal AF is more likely than persistent AF to be irregularly irregular.
- e) None of the above.

3 Assessing a patient's pulse in the pharmacy is a good way to screen for AF.

- a) true
- b) false

CASE: applies to questions 4-7

4 S.D. is a 78-year-old woman with paroxysmal AF. You have her full medical history, which includes AF, HF, hypertension, osteoar-

thritis, a myocardial infarction two years ago, and anxiety. She tells you she has "fluttering in her chest" when her heart goes "out of rhythm." Which of the following statements is incorrect?

- a) S.D. is likely to have symptoms with each AF occurrence.
- b) S.D.'s risk factors for AF include her age and her history of HF and hypertension.
- c) In addition to "fluttering," AF symptoms that S.D. might report are fatigue and lightheadedness.
- d) Chest pain experienced by S.D. could be from coronary artery disease or from AF.

5 If rate control were chosen to control S.D.'s AF, which of the following would be most appropriate?

- a) diltiazem with the addition of digoxin, should diltiazem be inadequate
- b) metoprolol with the addition of digoxin, should metoprolol be inadequate
- c) verapamil with the addition of digoxin, should verapamil be inadequate
- d) sotalol with the addition of digoxin, should sotalol be inadequate
- e) none of the above

6 S.D. tells you she also takes "a lot of water pills because of fluid" and that the doctor put her on a pill to get rid of the "bad heart rhythm." Checking into her lab tests, you find

she has renal dysfunction. Given what you know about S.D., which antiarrhythmic is the best choice for her?

- a) propafenone
- b) sotalol
- c) flecainide
- d) amiodarone

7 You proceed to risk stratify S.D. to determine her thromboembolic risk. Which of the following best fits this patient?

- a) S.D. is at high risk because her history is consistent with valvular AF.
- b) S.D. is at high risk because she has non-valvular AF with a history of myocardial infarction.
- c) S.D. is at high risk because she has non-valvular AF with at least three additional risk factors.
- d) S.D. is at intermediate risk simply due to her age.
- e) S.D. is at low risk because she has paroxysmal AF.

8 Which of the following AF patients is/are good candidates for rhythm control?

- a) a 73-year-old male with AF initially documented three years ago and a long-standing history of hypertension and diabetes
- b) a 69-year-old male with an initial presentation of AF and a history of gout and osteoarthritis
- c) a 55-year-old otherwise healthy female whose recurrent AF symptoms significantly affect her quality of life
- d) both a) and c)
- e) both b) and c)

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