

Etiology and initial investigation

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AC Skanes, P Dorian. Etiology and initial investigation. *Can J Cardiol* 2005;21(Suppl B):11B-14B.

The initial evaluation of a patient with atrial fibrillation should include a comprehensive review of historical factors, a physical examination and initial investigations. This evaluation has many important purposes, including the development of a therapeutic strategy for symptom relief, the assessment and management of thromboembolic risks, and the identification of underlying etiology. This evaluation should also review management of risk factors for overall cardiovascular morbidity and the treatment of atrial fibrillation. Baseline history, appropriate laboratory tests, and 12-lead electrocardiogram and echocardiography results should be obtained in all patients to identify the potential etiology and other comorbidities, and to stratify for risk of stroke.

Key Words: Ablation; Arrhythmia; Atrial fibrillation; Cardioversion; Electrophysiology

L'étiologie et l'exploration initiale de la fibrillation auriculaire

L'évaluation initiale de la fibrillation auriculaire (FA) devrait comprendre une anamnèse complète des antécédents, un examen physique et une première série d'examen. L'évaluation vise trois objectifs importants : élaborer une stratégie de traitement pour atténuer les symptômes, évaluer et traiter le risque de thrombo-embolie et rechercher la cause sous-jacente. L'évaluation devrait également tenir compte des facteurs de risque de l'ensemble des maladies cardiovasculaires et de leur traitement. Enfin, il faudrait procéder à une anamnèse de départ, à des examens de laboratoire appropriés ainsi qu'à une électrocardiographie à 12 dérivations et à une échocardiographie chez tous les patients atteints de FA pour rechercher la cause possible et l'existence d'autres maladies concomitantes et pour évaluer le risque d'accident vasculaire cérébral.

RECOMMENDATIONS

Class I

- 1) Baseline history, appropriate laboratory tests, 12-lead electrocardiogram (ECG) and echocardiography should be obtained in all patients to identify potential etiology and other comorbidities, and to stratify for risk of stroke. Details are highlighted in Tables 1 and 2 (level of evidence C).
- 2) Underlying causes or precipitating factors including underlying hypertension should be identified, eliminated or treated (level of evidence C).

Class IIa

- 1) Other ancillary tests should be considered under specific circumstances. Details are highlighted in Table 1 (level of evidence C).

INITIAL EVALUATION

The initial evaluation of a patient with atrial fibrillation (AF) should include a comprehensive review of historical factors, a physical examination and initial investigations. This evaluation has many important purposes including developing a therapeutic strategy for symptom relief, assessing and managing thromboembolic risks, and identifying underlying etiology.

This evaluation should also review management of risk factors for overall cardiovascular morbidity and its treatment.

It is incumbent upon the physician to document AF in at least one ECG lead. A perception of 'rapid irregular palpitations' may be reported during a multitude of rhythms including atrial tachycardia or atrial flutter with variable ventricular response, and occasionally during sinus tachycardia with or without ectopic beats. The approach to treatment and the thromboembolic risks differ significantly for these alternate rhythms.

The predominant pattern of AF should be determined:

The following points are based on references 1 and 2.

- First detected AF;
- Paroxysmal: AF is self-terminating within seven days of recognized onset;
- Persistent: AF is not self-terminating within seven days or is terminated electrically or pharmacologically; or
- Permanent: AF in which cardioversion has failed or in which clinical judgment has led to a decision not to pursue cardioversion.

One may not be able to identify the pattern of AF at the time of initial presentation and the pattern may change over time. An assessment of the severity of symptoms and impact on

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TABLE 1
Initial investigation of atrial fibrillation (AF)

Routine investigation
History and physical examination
Establish pattern (first detected, paroxysmal, persistent, permanent)
Establish severity including impact on quality of life
Identify potential etiology
Consider hypertension, alcohol abuse, thyroid disease and sleep apnea
Determine underlying thromboembolic risk
Develop a treatment strategy based on clinical risk factors
Evaluate likelihood of other arrhythmia – PSVT/atrial flutter
Document prior pharmacological therapies aimed at rhythm and rate control, including effectiveness and adverse effects
Twelve-lead electrocardiogram
Document presence of AF
Assess for left atrial abnormality/left ventricular hypertrophy/conduction disease/pre-excitation/sinus node disease
Assess for myocardial infarction
Measure baseline intervals (eg, QT interval) that may be affected by pharmacological therapy
Transthoracic echocardiography
Assess for chamber size and ventricular function
Assess valvular function
Assess for hypertrophy
Complete blood count, electrolytes, renal function
Thyroid function
Additional investigations of potential value
Chest radiography
When two-dimensional echocardiography is unavailable or difficult to obtain
If specific pulmonary abnormalities are anticipated
Ambulatory electrocardiogram monitoring
This includes 24 h Holter monitor, event recorder or loop monitor
Document arrhythmia and establish symptom-rhythm correlation – AF or alternative contributing arrhythmia (PSVT/flutter)
Assess rate control with activity during AF
Assess for bradycardia that may limit the use of specific rate- or rhythm-controlling agents
Help determine pattern if unclear from history
Treadmill exercise test
Only in those who have an intermediate or high risk for coronary disease
To evaluate rate control
Transesophageal echocardiography
Assess left atrial size
Rule out left atrial thrombus
Facilitate direct current cardioversion (with respect to stroke risk)
Investigate specific underlying etiological factors, especially left ventricular hypertrophy associated with hypertension
Electrophysiological study
Documented or suspected underlying PSVT
Consider atrial flutter ablation in those where this forms a substantial part of the symptom burden

PSVT Paroxysmal supraventricular tachycardia

quality of life should be performed. Symptoms associated with AF are highly variable, with some patients being truly asymptomatic and others having highly disruptive symptoms. The impact of these symptoms on lifestyle as well as a record of emergency room visits, hospital admissions and cardioversions should be made.

TABLE 2
Etiology of atrial fibrillation (AF)

Cardiovascular causes
Hypertension
Valvular disease
Coronary artery disease with prior myocardial infarction
Cardiomyopathy
Dilated
Hypertrophic
Restrictive
Pericardial disease
Electrical/senescence
Bradycardia-tachycardia (sick sinus) syndrome
Frequent/prolonged episodes of AF may cause electrical and structural remodelling of the atria, promoting further AF
Genetic/familial
Postoperative
Congenital heart disease
Noncardiovascular causes
Autonomically mediated (vagal)
Toxin – eg, alcohol
Endocrine – eg, thyroid disease
Pulmonary disease – chronic obstructive pulmonary disease, pneumonia, sleep apnea
Neurological – usually associated with myopathic muscle diseases
Idiopathic
Occult hypertension
Occult genetic causes

Symptoms at the termination of paroxysms should be sought and, if present, a symptom-rhythm correlation should be made using an ambulatory ECG (Holter, event recorder or loop recorder) if possible. Patients with tachycardia-bradycardia (sick sinus) syndrome often have sinus pauses, especially at the termination of AF. Symptomatic pauses may require pacing. Asymptomatic pauses may limit the use of rate- or rhythm-controlling agents in these patients.

Both paroxysmal supraventricular tachycardia (PSVT) and atrial flutter can cause a tachycardia-induced tachycardia and degenerate into AF. Successful ablation of the underlying PSVT can eliminate both the supraventricular tachycardia and the associated AF (3-6). Therefore, it is important to elicit and investigate any history of regular palpitation. This can be further explored by ambulatory ECG recording during symptoms, especially at the onset.

The underlying etiology and associated factors should also be determined. Specifically, efforts should be made to determine 'potentially reversible' causes such as excessive alcohol consumption that can trigger AF. Likewise alcohol withdrawal – the 'holiday heart syndrome' is also recognized as a potential trigger. Thyroid disease can be a potentially reversible and important cause of underlying AF. This may be particularly difficult to diagnose in the elderly. Hypertension is likely the most common cause of AF. Careful blood pressure assessment and investigation for underlying hypertension should be undertaken. It is incumbent upon the physician to make careful blood pressure determinations as outlined by the Canadian Hypertension Society for the diagnosis of underlying hypertension (7,8). AF may be the first presentation of an otherwise untreated

hypertensive patient. It is important to note that home blood pressure and 24 h blood pressure monitoring devices are greatly influenced by the variable rate during AF and, therefore, are not useful during this condition. Nonetheless, during sinus rhythm, these devices may add to the diagnostic yield of hypertension (see Canadian Hypertension Society Guidelines [7,8]). Other clues to an underlying etiology of hypertension include the presence of left ventricular hypertrophy detected using ECG or echocardiography.

Coronary artery disease with prior myocardial infarction and valvular disease are obvious etiologies of AF. Moreover, the left ventricular function will influence choices of therapy for both rate control and rhythm control. Some patients have a strong family history of AF, which may have a genetic basis in these cases (9).

Screening evaluation for obstructive and nonobstructive sleep apnea should be performed and investigation considered if suspected. Obstructive sleep apnea may be associated with obesity and hypertension, and may lead to AF. Moreover, management of the obstructive sleep apnea may facilitate control of hypertension and underlying AF. While not well investigated, it is likely that wide swings in autonomic tone associated with sleep apnea may facilitate development of AF in these patients. Holter monitoring for nocturnal bradycardia may be a useful screening test in these patients. In some patients, a formal sleep study may be required. Other forms of pulmonary disease, (eg, chronic obstructive pulmonary disease) are associated with AF. Investigations such as chest x-ray and pulmonary function tests should be performed as appropriate.

It is also important at the initial evaluation to determine thromboembolic risk in each patient. Large clinical trials have determined clinical risk factors for stroke associated with AF (10-14). Recommendations for anticoagulation therapy use these clinical risk factors to determine the use of acetylsalicylic acid versus warfarin therapy (see the American College of Chest Physicians recommendations for anticoagulation therapy in AF [Connolly and Gillis, pages 71B-73B]). It is important to highlight that these recommendations do not distinguish between patterns of AF. Paroxysmal AF in large clinical trials results in a similar risk for stroke as persistent or permanent AF (15).

It is important to document which rhythm- and rate-controlling agents have been used and discontinued in the past and the reason for discontinuation, such as perceived inefficacy or adverse effects.

The physical findings suggestive of AF include an irregular pulse (that may or may not be rapid in rate), an irregular jugular venous pulse and variation in the loudness of the first heart sound. Confirmation using ECG should be made if possible. The ventricular response during AF and the associated blood pressure should be noted (see above). The physical examination may also uncover associated valvular disease, myocardial disease or congestive heart failure.

A number of routine investigations are warranted in all patients presenting with a history of AF (Table 1). An ECG is useful both in sinus rhythm and AF. Evidence of left atrial enlargement, left ventricular hypertrophy, pre-excitation, underlying conduction disease or clues as to the underlying etiology of AF should be sought. A transthoracic echocardiogram should be performed in all patients. This will evaluate left ventricular function, which is useful for determination of specific therapies for rate control and antiarrhythmic drugs. Left atrial size should be noted, as well as any evidence of left atrial or left

atrial appendage thrombus; however, this is rarely observed with the transthoracic echocardiogram. Depending on practice patterns, prompt echocardiography may not be easily attained. Under these circumstances, a posteroanterior and lateral chest x-ray may replace the echocardiography to screen for cardiomegaly or left atrial enlargement. Otherwise, chest x-ray should not be routinely performed unless a specific underlying diagnosis is sought.

Routine bloodwork should be performed. Specifically, a complete blood count should be performed at least once. Urea and creatinine should be performed as a screen for renal function. This will influence choices and dose of drugs as well as potentially highlight end-organ damage of hypertension or adverse effects of other cardiovascular drugs. In the case of a history of excessive ethanol use, liver enzymes should be determined. A lipid profile is recommended in most patients as part of an overall assessment of cardiovascular risk. Thyroid function is not routinely measured due to cost. However, in elderly patients or those with clinical suspicion of hyperthyroidism, thyroid function should be measured. The yield of routine screening is likely to be low. Nonetheless, the impact of untreated hyperthyroidism can be significant and hyperthyroidism is frequently occult and difficult to diagnose from clinical presentation in the elderly population (16).

Ambulatory ECG monitoring is not routinely performed but has a number of important purposes. Holter monitoring, transient event recordings or loop recordings may document the underlying AF. It is also useful in an attempt to uncover PSVT or atrial flutter contributing to the AF (see above). Holter monitoring is very useful for assessment of rate control during activities of daily living and exercise. It may be also uncover associated bradycardia either caused by pharmacological agents or associated tachycardia-bradycardia (sick sinus) syndrome. It may be useful to determine the pattern of AF in minimally symptomatic patients who are unable to determine whether intervening sinus rhythm occurs. Such determination may have influence on plans for antiarrhythmic agents and direct current cardioversion.

The routine use of treadmill exercise testing is not recommended. It is best reserved for assessment of underlying functional capacity in patients during persistent or permanent AF as well as determination of the adequacy of rate control with exercise. Because myocardial ischemia is a rare cause of AF, treadmill exercise testing is not routinely recommended for patients without a history of ischemic symptoms. Patients who present with AF and associated chest pain may require assessment for underlying ischemic disease. Likewise, routine cardiac troponin evaluations should not be performed in patients with acute presentation of AF in whom there is a low likelihood of underlying ischemic disease.

Transesophageal echocardiography is not routinely required. It has become part of a useful strategy to rule out the presence of left atrial appendage thrombus and to facilitate direct current cardioversion in selected clinical scenarios (17-19).

Electrophysiological studies should be considered in patients with idiopathic AF at a young age, especially in those with documented regular supraventricular tachycardias or a history suggesting an underlying supraventricular tachycardia (ie, a history of regular palpitations preceding irregular palpitations). Underlying PSVT, due to accessory pathway-mediated tachycardia, AV node re-entry tachycardia or focal atrial

tachycardia, both inside and outside of the pulmonary veins, may cause AF as a tachycardia-induced tachycardia. Successful ablation of such PSVTs may also eliminate AF in young patients with these substrates (3-6). In addition, electrophysiological study and catheter ablation of underlying atrial flutter should be considered when atrial flutter forms a substantial part of the symptom burden. The best results occur when atrial flutter is the predominant or sole rhythm disturbance. A more thorough discussion of this topic is presented in Guerra and Skanes, pages 31B-34B, and Simpson et al, pages 67B-70B.

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