Introduction

Atrial fibrillation (AF) has in recent years been the subject of intense investigation in terms of obtaining a better understanding of its mechanism and improving its management. Despite the advances made, AF remains a challenge for the clinician, and it is uncertain whether these theoretical advances have resulted in a significant improvement in the way the vast majority of patients are managed in general practice. Several reasons may account for this situation. One possible reason is incomplete knowledge of the complex mechanism of this common arrhythmia. Another possible reason is the heterogeneous clinical presentation of the arrhythmia. Still, the numerous publications and clinical trials devoted to AF have dealt with this arrhythmia as if it were a single entity. Furthermore, the abundant terminology used to characterize various subsets of AF and the absence of consistency in the definitions have added to the difficulty in communication. Consequently, it has become difficult to compare the results of pharmacologic or nonpharmacologic therapies because different classifications have been used, and the difficulty in characterizing the patient population has not allowed easy or appropriate comparison. The Working Group of Arrhythmia of the European Society of Cardiology (WGA-ESC) and the North American Society of Pacing and Electrophysiology (NASPE) have recognized the need to create a Study Group in an attempt to achieve a consensus on the terminology and classification of AF. This document reports the conclusions reached by this Study Group during a meeting held on June, 13, 2000, and completed by the members soon thereafter. The definitions and classification resulting from this consensus were largely adopted in the ACC/AHA/ESC and NASPE guidelines on AF published in 2001.

The Study Group recognized that there are several ways to approach the classification of AF. The electrocardiographic (ECG) presentation of AF is not very helpful for clinical management of patients, except for the ECG aspect recognized as “focal AF” to designate a rapidly firing focus most commonly arising from the pulmonary veins that may trigger AF. Two-dimensional epicardial mapping has been the basis for a classification of AF proposed by Allessie et al. Other investigators have tried to characterize various aspects of AF based on intracardiac recordings. Noncontact three-dimensional mapping appears to be a useful tool for analyzing atrial activation. Both focal ablation and linear ablation have increased our knowledge of mechanisms, although the road to full understanding is still long. The purpose of the Study Group was to provide the clinician with a simple way to characterize an episode of AF, as well as the presentation of AF in a given patient, being aware that this presentation may change over time. Such classification aims to be helpful for proper management of AF in clinical practice.

Definitions

Atrial fibrillation is an atrial tachyarrhythmia characterized by predominantly uncoordinated atrial activation with consequent deterioration of atrial mechanical function. Atrial fibrillation on ECG is indicated by the absence of consistent P waves; instead there are rapid oscillations or fibrillatory waves that vary in size, shape, and timing and are generally associated with an irregular ventricular response when atrioventricular (AV) conduction is intact. The ventricular response in AF depends on AV nodal properties, the level of vagal and sympathetic tone, and drugs that affect AV nodal conduction, such as beta-blockers, non-dihydropyridine calcium channel blockers, and digitalis glycosides. However, regular RR intervals may occur, for example, in the presence of heart block associated with conduction disease or drug therapy. In patients with permanent ventricular pacing, the
diagnosis may require temporary pacemaker inhibition in order to visualize AF activity.7 A rapid, irregular sustained wide QRS complex tachycardia should suggest AF with conduction over an accessory pathway.

**Arrhythmias Related to Atrial Fibrillation**

Atrial flutter is a more organized arrhythmia than AF. It is characterized by a “sawtooth” pattern of atrial activity called “flutter waves” that are particularly visible on ECG leads II, III, and aVF, with the typical rate in the untreated state ranging from 250 to 350 beats/min. In the typical form, the P waves are negative on ECG leads II, III, and aVF and positive on lead V1. In the reverse typical form, the P waves are positive on leads II, III, and aVF and negative on lead V1. In atrial flutter, there is commonly 2:1 or greater AV block resulting in a ventricular rate of 150 beats/min or less.10

In atrial tachycardia, the P waves are well identified and separated by an isoelectric baseline on all ECG leads. The morphology of the P waves may be helpful in localizing the site of origin of atrial tachycardias. The ventricular rate of atrial tachycardias is quite variable, ranging from 100 to >300 beats/min. A unique type of atrial tachycardia has been identified recently that commonly originates in the pulmonary veins but may occur elsewhere. The tachycardia rate is rapid, typically above 250 beats/min, and often degenerates into AF.11

Atrial fibrillation may occur by itself or in association with other arrhythmias, most commonly atrial flutter or atrial tachycardias. It is important to know that atrial flutter may result from antiarrhythmic agents prescribed in order to prevent recurrences of AF. The ECG may show a pattern that alternates between atrial flutter and AF, primarily due to changing activation patterns in the atria. Atrial flutter may degenerate into AF, and AF may initiate atrial flutter. Atrial fibrillation also may be triggered by other atrial tachycardias, as well as AV reciprocating tachycardia (AVRT) and AV nodal reciprocating tachycardia (AVNRT). Electrophysiologic studies with intracardiac mapping, when indicated, may be helpful in differentiating various types of atrial arrhythmias and their mechanisms.5

**Clinical Classification of Atrial Fibrillation (Table 1)**

As stressed earlier, AF has a heterogeneous clinical presentation. It may occur in the presence or absence of detectable heart disease and in the presence or absence of related symptoms.12 An attack of AF may be self-terminating or require medical intervention for termination. The clinician may have to deal with an attack or episode of AF and to define the pattern over time, which includes the number of episodes, duration of episodes, mode of onset, possible triggers, and response to therapy. Although the pattern of the arrhythmia may change over time, it is of clinical value to characterize the arrhythmia at a given moment. Therefore, it appears necessary to attempt to classify various subsets of patients with AF in order to address properly the management of each patient subset.

Clinical classifications have been proposed, although no single classification can take into account all aspects of patients with AF.13-15 For the clinician, a classification may be helpful if it is based on the clinical presentation and has inherent therapeutic implications.

It has long been recognized time that an episode of AF may be self-terminating or non–self-terminating. The terms chronic and paroxysmal have been used, but the definitions used have been quite variable, resulting in difficulties in comparing studies on AF, effectiveness of treatments, and therapeutic strategies. The Study Group has come to the following consensus on the terminology of AF. This terminology applies to episodes of AF defined as lasting >30 seconds.

It is important for the clinician to ascertain whether an incident of AF is the very first episode, that is, the initial event; whether it is symptomatic or not; and whether it is self-terminating or not. If the patient has had two or more episodes, AF is said to be recurrent. Episodes of paroxysmal AF usually self-terminate within 48 hours and, by definition, in fewer than 7 days. When an episode of AF has lasted longer than 7 days, AF is designated as persistent. In that case, termination using pharmacologic therapy or electrical cardioversion may be required. The time frame of 7 days, although arbitrary, represents the limit beyond which spontaneous cardioversion is unlikely to occur and the success rate of pharmacologic cardioversion is low. Persistent AF may be the first presentation of the arrhythmia or may be preceded by recurrent episodes of paroxysmal AF. When AF is persistent, termination using electrical cardioversion may be required. When AF has been present for some time and fails to terminate using cardioversion or is terminated but relapses within 24 hours, it is said to be established or permanent. Established AF or permanent AF may be the first presentation of a non–self-terminating arrhythmia or be preceded by

<table>
<thead>
<tr>
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<th>Clinical Features</th>
<th>Arrhythmia Pattern</th>
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<tr>
<td>Initial event</td>
<td>Symptomatic</td>
<td>May or may not recur</td>
<td>Antiarrhythmic therapy for prevention is not needed except if symptoms are severe</td>
</tr>
<tr>
<td>(first detected episode)</td>
<td>Asymptomatic (first detected)</td>
<td></td>
<td></td>
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<td></td>
<td>Onset unknown (first detected)</td>
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<tr>
<td>Paroxysmal</td>
<td>Spontaneous termination &lt;7 days</td>
<td>Recurrent</td>
<td>Prevention of recurrences</td>
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<tr>
<td></td>
<td>and most often &lt;48 hours</td>
<td></td>
<td>Rate control and anticoagulation if needed</td>
</tr>
<tr>
<td>Persistent</td>
<td>Not self-terminating</td>
<td>Recurrent</td>
<td>Rate control and anticoagulation if needed</td>
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<tr>
<td></td>
<td>Lasting &gt;7 days or prior cardioversion</td>
<td></td>
<td>if needed or/and cardioversion and prophylactic antiarrhythmic therapy</td>
</tr>
<tr>
<td>Permanent (accepted)</td>
<td>Not terminated</td>
<td>Established</td>
<td>Rate control and anticoagulation as needed</td>
</tr>
<tr>
<td></td>
<td>Terminated but relapsed</td>
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<tr>
<td></td>
<td>No cardioversion attempt</td>
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curable cause. In about 30% of patients, AF may occur in the vast majority of patients, AF is unrelated to an identifiable cause and the absence of recurrence. In contrast, in the presenting episode of AF will result in termination of the atrial rhythm and the absence of recurrence. In these settings, AF rarely represents the major problem. In most cases, treatment of the underlying pathology and/or the presenting episode of AF will result in termination of the arrhythmia and the absence of recurrence. In contrast, in the vast majority of patients, AF is unrelated to an identifiable curable cause. In about 30% of patients, AF may occur in the absence of heart disease (lone AF) or of any disease (idiopathic AF).

Clinical Implications (Table 1)

This clinical classification aims to help the clinician in selecting the correct therapeutic options.2,7 The first detected episode of AF may be symptomatic or asymptomatic. If it is asymptomatic, control of heart rate may be a reasonable therapeutic option.17 If AF is symptomatic, restoration of sinus rhythm with proper anticoagulation if AF lasted more than 48 hours may or may not be indicated. However, prophylactic antiarrhythmic therapy is not indicated for this first episode of AF unless severe symptoms are associated with AF.2 In both situations, long-term oral anticoagulation may be indicated if the patient is at high risk for thromboembolism.

Both paroxysmal and persistent AF have a pronounced tendency to recur. The therapeutic strategy for both subsets may be to maintain sinus rhythm using sodium channel blockers (Class I agents of Vaughan-Williams classification) or potassium channel blockers (Class III agents of Vaughan-Williams classification), or to control heart rate using digitalis glycosides, beta-blockers, or non-dihydropyridine calcium channel blockers.

Long-term anticoagulation is indicated in patients at risk for embolic complications. In persistent AF, restoration of sinus rhythm may be indicated in symptomatic patients with due respect to the anticoagulation rules for cardioversion. In permanent AF, control of heart rate and anticoagulation are recommended.

Long-term oral anticoagulation should be considered in every patient with AF, regardless of the presentation of AF, if risk factors for embolic complications such as age above 65 years, history of embolic complications, valvular heart disease, hypertension (particularly if left ventricular hypertrophy is present), diabetes, recent-onset heart failure, depressed left ventricular function, or increased left atrial size are present.

References