

Atrial Fibrillation

A clinical introduction

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Atrial Fibrillation

Atrial fibrillation is the most commonly occurring type of arrhythmia, affecting nearly 1% of the population under the age of 60 years⁽¹⁻⁴⁾. The prevalence increases to more than 6% for people over the age of 80 years^(4, 5). In the USA, an estimated 2.2 million people have paroxysmal or persistent atrial fibrillation^(4, 6).

The rate of atrial fibrillation after cardiac bypass surgery is 20–35% and 50% after valve surgery. It may be associated with underlying heart disease such as congestive heart failure or rheumatic heart disease. It may also occur as a result of an acute myocardial infarction, hypoxia, hypertension, myocardial ischemia, digitalis toxicity or hyperthyroidism. Other causes include pneumonia, lung carcinoma, effusion and pulmonary embolism^(5, 6).

Definition

A normal heartbeat consists of one P wave (atrial activity) and one QRS complex (ventricular activity). In atrial fibrillation, abnormal electrical activity results in an *irregularly irregular* rhythm (statistics of R-R interval sequences are different from those with irregular rhythm of other origin) with the absence of P waves. The irregular atrial activity causes rapid contractions of the atrium, incomplete emptying of the atrium and improper filling of the ventricles⁽⁷⁾.

The erratic electrical impulses cause the atria to quiver, creating a fibrillating waveform with a loss of P waves. A consequence of the quivering atrium is a loss of atrial kick, causing blood to pool in the atria and ventricles increasing the risk for thromboembolism and stroke. In non-rheumatic atrial fibrillation, the rate of ischemic stroke is 5%, which is 2–7 times greater than for a person without atrial fibrillation⁽⁴⁻⁶⁾. Sequelae from a rapid atrial fibrillation include a decrease in cardiac output and hemodynamic instability requiring immediate cardioversion. The risk for stroke related to atrial fibrillation increases to 36% for patients aged 80–89 years⁽⁴⁾. Atrial fibrillation increases the mortality rate 50–90% in both men and women⁽⁶⁾.

Types

Often patients are unaware of any rhythm disturbances. This is true of atrial fibrillation; it may be symptomatic or asymptomatic^(5, 7). Classification of atrial fibrillation starts with clinical presentation, either acute onset or chronic. Acute atrial fibrillation may be related to a transient and reversible cause. Chronic atrial fibrillation is viewed as recurrent or prolonged episodes or an unknown history or duration.

Chronic atrial fibrillation is subdivided into paroxysmal or sustained, and sustained is further classified as persistent or permanent. Paroxysmal episodes are self-terminating but may be recurrent. Sustained atrial fibrillation refers to episodes greater than 30 seconds in duration and which are not self-limiting. Persistent atrial fibrillation may require urgent medical, pharmacological or electrical intervention⁽⁴⁾. Permanent atrial fibrillation is a sustained condition that is either not responsive or not appropriate for cardioversion.

There is no general agreement on terminology. A suggested timeframe for paroxysmal chronic atrial fibrillation is an episode of less than seven days, while chronic episodes have a duration of greater than seven days. Also, a paroxysmal episode lasting longer than 48 hours may be termed persistent⁽⁷⁾. It is not uncommon for patients to switch subgroups or evolve to chronic atrial fibrillation. The choice of therapy is reflective of a patient's condition at a given point in time⁽⁵⁾.

Therapies

The clinical objective of each classification is to dictate the therapeutic approach. In acute atrial fibrillation, immediate cardioversion is indicated if there is hemodynamic compromise. Once the goal of heart rate control is achieved, anticoagulation therapy should be initiated to decrease the risk of thromboembolism. Treatment of the causative or precipitating factor is essential to achieve and maintain a normal sinus rhythm. For example, treatment of hyperthyroidism may be the only intervention necessary to resolve an associated acute onset of atrial fibrillation.



Atrial Fibrillation: "irregularly irregular" ventricular rhythm with an absence

In a joint publication released by the American College of Cardiology (ACC), the American Heart Association (AHA) and the European Society of Cardiology (ESC), recent research suggests that multiple re-entrant pathways may be present in atrial fibrillation, unlike other arrhythmias which have a single re-entrant circuit^(4, 7). The Practice Guidelines suggest a higher success rate with pharmacological or electrical cardioversion when atrial fibrillation has been present for less than 24 hours. A hypothesis that atrial electrical remodeling occurs during persistent atrial fibrillation is supported by higher reported success rates with early intervention^(4, 7).

In paroxysmal atrial fibrillation, the clinical objective is to introduce antithrombotic therapy and to reduce the frequency of occurrences. Infrequent or mildly symptomatic patients may not require antiarrhythmic medications. Anticoagulation therapy is indicated prior to cardioversion for persistent atrial fibrillation or as long-term prophylaxis against stroke and thromboembolism.

According to the ACC/AHA/ESC Guidelines for the Management of Patients with Atrial Fibrillation, anticoagulation therapy is recommended for 3–4 weeks before and after cardioversion in atrial fibrillation of unknown duration or atrial fibrillation lasting more than 48 hours. Medication management is an ongoing task to coordinate the current state of atrial fibrillation with any underlying heart conditions⁽⁴⁾.

Persistent atrial fibrillation can in most cases be successfully treated with electrical cardioversion. Rates of cardioversion to a sinus rhythm vary from 70 to 90%⁽⁷⁾. However, patients undergoing cardioversion are at significant risk of thromboembolism and thus require anticoagulation therapy prior to an elective cardioversion⁽⁵⁾. Permanent atrial fibrillation is the category designated for patients who do not maintain a sinus rhythm after cardioversion or are not candidates for the procedure. Therapeutic goals for these patients include heart rate control and prevention of thromboembolism⁽⁷⁾.

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