

Atrial Fibrillation

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Atrial fibrillation (AF) is an irregular, disorganized rhythm characterized by a lack of organized mechanical atrial activity. The atrial rate is commonly >300 bpm. It is the most common sustained arrhythmia.

Consequences:

- Irregularity results in variable ventricular filling and stroke volume.
- Often associated with rapid heartrate. When prolonged, this can result in tachycardia induced cardiomyopathy.
- Inadequate atrial emptying (loss of “atrial kick”)
- In some patients it can impair ventricular filling
- These effects can decrease cardiac output and cause syncope, dyspnea, and fatigue
- Stasis of blood, especially in the appendage, favors the formation of thrombi and creates a risk of stroke
- 70% of patients will experience systems such as palpitations, hypertension, chest pain and dyspnea.

AF is a potent independent risk factor for stroke.

The prevalence of AF increases with age as does the stroke rate from atrial fibrillation.

10-15% of patients over 80 will develop atrial fibrillation

The risk of a thromboembolic event varies with the cause of the atrial fibrillation.

Atrial fibrillation of any cause accounts for about 45% of cardiogenic thromboembolic events.

In the setting of Rheumatic Heart Disease the risk of stroke is increased 17-18 fold.

In the setting of non-rheumatic valvular disease it is 5-6 fold increased.

Most strokes are:

Clustered around the onset of atrial fibrillation (25%)

Have an increased incidence in the first year after the onset (10-15%)

Occur after the first year at a rate of about 5% per year

Is similar with chronic and paroxysmal atrial fibrillation.

When there is no organic heart disease, hypertension, and the patient is younger than 60 the risk of stroke is small with a rate of about 1.3% over 15 years. These patients will represent only about 3% of all patients with atrial fibrillation. This is called **Lone Atrial Fibrillation**.

Assess for concomitant disease associated with atrial fibrillation:

Thyroid

Pulmonary

Pericardial
Valvular Disease
Ischemic Heart Disease
Hypertensive Heart Disease

Precipitation factors for single or paroxysmal AF

Acute MI
Acute alcohol intoxication
Pulmonary infection
Surgery
Chest Trauma
Pericarditis
Pulmonary embolism

Chronic Atrial Fibrillation

35% Ischemic Heart disease
45% Hypertensive Heart Disease
20% Rheumatic Valvular Heart disease in past, now less common

Treatment

Unstable

When a rapid ventricular response does not respond promptly to pharmacological measures for patients with AF with ongoing myocardial ischemia, symptomatic hypotension, angina, or HF, immediate R-wave synchronized direct-current cardioversion is recommended.

-Safe/Emergent electrical cardioversion with airway protection and sedation if possible

Stable

Goals

1. **Rate control**
2. **Evaluate need for anticoagulation**
3. **Rhythm control:**
 - A. **Planning for electrical or pharmacologic conversion to sinus rhythm**
 - B. **Antiarrhythmic therapy for maintenance of sinus rhythm**

1. Rate Control

- A. Digoxin: 1.0mg IV over 24 hour in .25 mg increments q 6 hr.
Slow Onset/and not effective for high catecholamine states
- B. Beta Blockers:
 - i. Short Acting IV Esmolol - 9 minute half life
Load 500 ug/kg then drip at 50-200ug/kg/min
 - ii. Long acting IV and Oral (Propranolol, Metoprolol, & Atenolol)
- C. Calcium Channel Blockers: Diltiazem and Verapamil have negative chronotropic
To be preferably avoided in combination with B-Blockers

Points on Rate control

1. Poorly controlled AF occasionally can result in ventricular dysfunction..
2. Optimal resting heart rate in chronic AF is 60-90
3. Digoxin helpful at rest but has little effect on controlling peak heart rate during vigorous exertion.
4. Beta Blockers are effective in slowing ventricular response to exercise but may have a negative exercise effect.
5. Calcium Channel Blockers blunt exercise heart rate response and may improve exercise tolerance
6. Digoxin and either calcium or beta blockers may be synergistic.
7. Amiodarone can be used for rate control when the above therapies are inadequate.
8. When pharmacologic tx is inadequate chronically, insertion of a pacemaker and ablation of the AV node is a reasonable option

2. Evaluate need for anticoagulation

Atrial Fibrillation of unknown duration should be anticoagulated to an INR 2 – 3 for a minimum of 3 – 4 weeks prior to elective cardioversion by either pharmacologic or electrical means.

A precardioversion TEE can substitute for the 3 week anticoagulation period but post cardioversion anticoagulation should be given as atrial stunning can cause thrombus post conversion and in case atrial fibrillation recurs.

POINTS

1. Aspirin has been shown to be better than placebo for AF
2. Coumadin has been shown to be better than placebo for AF
3. Coumadin has been shown to be better than aspirin for AF when risk factors present
4. Lone atrial fibrillation has such a low embolic rate that anticoagulation has not been recommended. Because of the safety of antiplatelet therapy with aspirin most authorities recommend aspirin for lone AF.

At least five landmark trials showed coumadin to be more effective than placebo

	Acronym	Reduction	INR Range
NEJM 327: 1406-1412, 1992	SPINAF	79%	1.4-2.8
JACC 18:355, 1991	CAFA	42%	2.0-3.0
Circulation 84:527, 1991	SPAF	67%	2.0-4.5
Lancet 1:L175-178, 1989	AFASAK	58%	2.8-4.2
NEJM 323: 1505, 1990	BAATAF	86%	1.5-2.7

ASA:

AFASAK 75 MG/DAY DID NOT SHOW A STATISTICALLY SIGNIFICANT BENEFIT
 SPAF 325 MG/DAY DID SHOW A REDUCTION IN PATIENTS <75 YEARS OF AGE BUT NOT IN PATIENTS > 75

TWO ADDITIONAL TRIALS STUDIED THE EFFICACY AND SAFETY OF ASPIRIN

SPAF II Lancet 343: 687-691, 1994
EAFT Lancet 342: 1255-1262, 1993

Summary:

Aspirin, regardless of dose, results in a 20 – 25% stroke risk reduction over placebo

Coumadin results in a 60 – 70% stroke reduction

Major Bleeding from coumadin is < 1% per year

Intracranial hemorrhage rate is <1% / year but may be higher in patients >75 y/o

Current Guidelines:

1. **Antithrombotic therapy to prevent thromboembolism is recommended for all patients with AF, except those with lone AF or contraindications.**
2. **The selection of the antithrombotic agent should be based upon the absolute risks of stroke and bleeding and the relative risk and benefit for a given patient.**
3. **For patients without mechanical heart valves at high risk of stroke, chronic oral anticoagulant therapy with a vitamin K antagonist is recommended in a dose adjusted to achieve the target intensity international normalized ratio (INR) of 2.0 to 3.0, unless contraindicated. Factors associated with highest risk for stroke in patients with AF are prior thromboembolism (stroke, transient ischemic attack [TIA], or systemic embolism) and rheumatic mitral stenosis.**
4. **Anticoagulation with a vitamin K antagonist is recommended for patients with more than 1 moderate risk factor. Such factors include age 75 y or greater, hypertension, HF, impaired LV systolic function (ejection fraction 35% or less or fractional shortening less than 25%), and diabetes mellitus.**
5. **Aspirin, 81–325 mg daily, is recommended as an alternative to vitamin K antagonists in low-risk patients or in those with contraindications to oral anticoagulation.**
6. **Antithrombotic therapy is recommended for patients with atrial flutter as for those with AF.**

CHADS2 RISK FACTORS: Congestive heart failure, Hypertension, Age>75, Diabetes (all 1 point), Previous TIA or Stroke (2 points).

Treatment:

CHADS2	TX
0	ASA or none (lone AF)
1	Warfarin or ASA
2+	Warfarin

3. Rhythm Control

A. Planning for cardioversion

Cardioversion to sinus rhythm either by pharmacologic or electrical means results in restoration of

systolic function of the atrium. Most thrombi embolize when conversion occurs, so there is a risk of cardioversion. The risk varies with the cause and setting of the cardioversion.

AF of unknown duration Not anticoagulated	5-7%
AF of unknown duration Anticoagulated 3 weeks (INR 2-3)	.8-1.5%
AF of unknown duration On heparin with a cardioversion without a thrombus seen on TEE	0-1%

Electrical DC synchronized cardio version is >97% successful at the restoration of sinus rhythm. It may only last a few minutes, days, months, or years!

1. **Direct-current cardioversion can be useful to restore sinus rhythm as part of a long-term management strategy for patients with AF.**
2. **Patient preference is a reasonable consideration in the selection of infrequently repeated cardioversions for the management of symptomatic or recurrent AF.**
3. **Frequent repetition of direct-current cardioversion is not recommended for patients who have relatively short periods of sinus rhythm between relapses of AF after multiple cardioversion procedures despite prophylactic antiarrhythmic drug therapy.**
4. **Electrical cardioversion is contraindicated in patients with digitalis toxicity or hypokalemia.**

Pharmacological Enhancement of Direct-Current Cardioversion

5. **Pretreatment with amiodarone, flecainide, ibutilide, propafenone, or sotalol can be useful to enhance the success of direct-current cardioversion and prevent recurrent AF.**
6. **In patients who relapse to AF after successful cardioversion, it can be useful to repeat the procedure following prophylactic administration of antiarrhythmic medication.**

Ibutilide (Corvert) is approved for the conversion of atrial fibrillation.

Action: Prolongs action potential duration in myocytes and increases both atrial and ventricular refractoriness. (Class III electrophysiologic effects).

Dose: .01 mg/kg Up to 1mg total does IV over 10 minutes

Warning 1.7% of patients sustain polymorphic ventricular tachycardia. Ibutilide should not be used in patients receiving concomitant antiarrhythmic drugs which prolong the QT interval

Serum Potassium and magnesium should be checked and normalized prior to Ibutilide

B. Antiarrhythmic Therapy

Many antiarrhythmics have been used to maintain sinus rhythm after conversion to sinus; No one is superior to the other in all cases. It is probably true that the worse the organic heart condition the less

likely any drug will be effective. Amiodarone is probably the most efficacious but also has numerous potential toxicities that must be monitored. Predictors of the maintenance of sinus include:

Duration of the atrial fibrillation

Size of the left atrium

LV systolic function

Agents

Class I

A. Quinidine, Procainamide, Disopyramide (rarely used nowadays).

C Flecainide, Propafenone (Rythmol) (used in patients without CAD or significant structural heart disease)

Class III

Amiodarone

Sotalol

Dofetilide (Tikosyn)

Points

1. In post MI patients, Class I antiarrhythmics have increased mortality. They are usually avoided in all patients with LV dysfunction.
2. Class IA and III agents prolong the QT interval and can cause torsades, less so with amiodarone.
3. Dofetilide and amiodarone are effective and safe even in patients with LV dysfunction; other agents should usually be avoided. Sotalol can sometimes be used with caution.
4. Post-operative atrial fibrillation following coronary artery by-pass surgery is common (30-70%) and generally resolves spontaneously if the rate is controlled. Beta blockers or amiodarone are the drugs of choice.

Ablation of atrial fibrillation has become increasingly common since the discovery in the late 1990s that isolation of the pulmonary veins often eradicates paroxysmal AF. It is less effective in chronic AF and additional ablative techniques have evolved and are continuing to evolve. Currently AF ablation is effective in 60-90%, depending on substrate.

Rate vs Rhythm Control: Five trials done between 2000-2004 compared these two strategies. In general, in patients who are asymptomatic, a rhythm control strategy is not clearly superior to rate control. However, many patients are symptomatic or functionally compromised (often unaware they are compromised until converted to SR) and this does not apply to those patient groups.