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**Information about AF**

***ATRIAL FIBRILLATION***

- I. Definition: Disorganized supraventricular tachycardia, ineffective atrial contraction, and irregular, chaotic and often rapid ventricular rate.
- II. Pathogenesis
  1. Atrial fibrillation (AF) is frequently triggered by a premature atrial contraction which by re-entry emits impulses at a rate of 300-700 per minute; these impulses enter the atrioventricular (AV) node randomly and because of the AV node's slower rate of conduction not all the impulses are conducted resulting in a ventricular rate that is slower than the atrial rate, as well as irregular; less commonly, atrial impulses result in a adequate or insensitive ventricular response which can lead to severe bradycardia or sudden death.
  2. Ineffective emptying of atrial due to electrical stimulation from multiple atrial foci leads to hemodynamic instability, ineffective cardiac output, and stasis of blood, which may results in thrombus formation and complications such as peripheral embolization and stroke.
  3. Factor which predispose patients to develop AF are the following:

- Organic heart disease that cause atrial distention such as ischemia or infarction, hypertension, valvular disorders, obstructive cardiomyopathy, rheumatic heart disease, or Woff-Parkinson-White syndrome.
  - Metabolic disease such as hyperthyroidism and hypothyroidism.
  - Right atrial stretch due to pulmonary embolus and chronic lung disease.
  - High adrenergic tone secondary to alcohol withdrawal, sepsis, or excessive physical exertion.
4. The most common predisposing factors are the coronary artery disease, acute Respiratory illness, cardiothoracic surgery, and hyperthyroidism.

### III. Clinical Presentation

1. AF is the most common, sustained, cardiac arrhythmia and cause more hospital admissions than any other arrhythmia.
2. Many patients are asymptomatic whereas a few have life-threatening Symptoms.
3. Patients with rapid ventricular responses after complain of chest pain, Fullness in neck, palpitations, fatigue, dizziness, or syncope secondary to decreased blood pressure.
4. In patients with organic heart disease, AF can lead to hemodynamic decompensation, hypotension, pulmonary edema, cardiac standstill or death.
5. In the past, patients with preexisting left ventricular failure or coronary obstruction had a poor prognosis; today, prognosis is fair if patients are closely followed and participate in cardiac rehabilitation.

### IV. Diagnosis Test

1. Order a 12-lead ECG; AF is characterized by absence of P-waves and Baseline fibrillatory activity; usually the ventricular rate is irregular (This finding is very important).
2. Order thyroid function test.
3. Order an echocardiogram.
  - Detects valvular abnormalities, pericardial effusion, enlarged chambers, Ventricular contraction abnormalities or other wall motion abnormalities.
  - Is helpful in predicting the success of cardioversion (a dilated left atrium or severe left ventricular failure has a low success rate).
  - Transesophageal echocardiograms (TEE) are the most sensitive for identifying left atrial thrombi, but are expensive.
  - Consider ordering electrolytes, blood urea nitrogen (BUN), and creatinine.
  - Ambulatory ECG readings may be necessary (i.e, 24-48 hour holter).
  - Consider chest x-ray to evaluate cardiomegaly.

V. Plan/Management:

Goals of therapy are threefold: control ventricular rate, prevent recurrences (maintain sinus rhythm), and prevent thromboembolism and stroke.

- Hospitalization is usually required for treatment of acute AF, particularly if ventricular rate is  $>170$  per minute or  $<50$  and patient has underlying cardiac disease (consult cardiologist); patients often revert to sinus rhythm when predisposing factors are removed; otherwise, direct-current-

cardioversion (DCC) or intravenous drug therapy is indicated to stabilized patient until oral drug trials are initiated.

- For patients who have mild stable symptoms with no cardiovascular compromise, oral therapy to control ventricular rate with an AV nodal blocking agent may be the only therapy that is needed; remember, however, these drugs do not convert an acute episode back to sinus rhythm and they do not prevent recurrences of AF; these drugs may also result in an inappropriately low ventricular response:
  - a. Digoxin slows the ventricular rate by blocking conduction through the AV node.
  - b. Beta-blockers also control ventricular response especially during exercise; these agents may be particularly beneficial when AF is associated with hyperthyroidism or other hyperadrenergic states such as MI or sepsis.
  - c. Calcium channel blockers reduce both resting and exercise heart rate.
  - d. Clonidine may also be effective in controlling ventricular response, but further studies are needed.
- The AV nodal blocking drugs do not have any specific effect in promoting sinus rhythm; however, they do facilitate hemodynamics which may results in rate control; to restore sinus rhythm direct current cardioversion or pharmacologic cardioversion with an antiarrhythmic drug is often necessary.

1. Elective DCC is sometimes the treatment of choice when the duration of the AF is less than 12 months and when the atria are of normal size (not enlarged and boggy).
  - a. Usually, patients are prescribed anticoagulant therapy (i.e., warfarin) for 3-4 weeks prior to cardioversion.
  - b. Unfortunately, the majority of patients with long-standing AF revert back to AF after cardioversion.
  - c. Cardioversion is the most effective means of restoring sinus rhythm but can result in embolisms, hypotension, pulmonary edema, and major arrhythmias.
  
2. Pharmacological conversion involves an antiarrhythmic drug; Selection of a drug should be based on potential adverse effects in specific patients; because of dangers of proarrhythmia; drugs are usually initiated in the hospital with continuous cardiac monitoring.

TYPE	DRUG	DOSE
IA	Quinidine	324-648 mg q8h
		300-600 mg q8h
	Procainamide (Procan-SR)	50 mg/kg/day divided in 2 doses q12h
	Disopyramide (norpace CR)	200-400 mg q12h
IC	Flecainide (Tambocor)	50-150 q12h; Max. 300 mg/day
	Propafenone (Rythmol)	150-300 mg q8h; Max 300 mg q8h
III	Sotalol HCl (Betapace)	80-240 mg q12h; Max 320 mg/day
	Amiodarone (Cardarone)	Initiate in hospital; 800 – 1200 mg/day

	In divided doses. After control is achieved, give 400-600 mg/day for one month, then gradually lower to maintenance dose of 200-300 mg/day within 6 months.
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- Long-term maintenance of sinus rhythm
  1. Approximately 75-85% of patients not maintained on any antiarrhythmic drugs reverts to AF in 1-2 years; nonetheless it is reasonable to initially try to maintain sinus rhythm without medications.
  2. For patients who have recurrent AF or are likely to have recurrent AF, the following two approaches are acceptable.
    - a. Combine antiarrhythmic drugs with AV nodal blocking drugs (digoxin, betablockers, and calcium channel blockers); continue anticoagulation (this approach is most often recommended).
    - b. Maintain heart rate control with AV nodal blocking drugs and continue anticoagulation.
- Anticoagulation therapy is recommended for the prevention of stroke and other embolic complications.

- Patient Education:
  1. Remind patients to quit smoking, avoid sleep deprivation, and limit Their use of stimulants (i.e., caffeine, sodas, chocolate) and alcohol.
  2. Teach relaxation techniques to reduce stress.
  3. Teach patients and family members to watch for signs of complications such as extremely rapid heart rate, edema and weight gain, increasing dyspnea on exertion, and chest pain.
  4. Teach signs and symptoms of digitalis toxicity (arrhythmias, anorexia, nausea, vomiting, diarrhea, lethargy, confusion, and visual disturbances such as scotomas and color perception changes) and/or adverse effects of other drugs.
  5. Intensive teaching is needed for patients on warfarin.
- Permanent pacers and/or ablation of AV junction are alternatives that can be discussed with patients who have disabling symptoms.
- Follow Up: Close follow up is essential
  1. Patients who have their first episode of AF should return to clinic 24-48 hours for reevaluation.
  2. Patients who were electrically cardioverted should have frequent cardiac monitoring with ECG usually at intervals of less than 1 week, 2 weeks, 1 month, 3 month, and then, every 3 months.

3. For patients taking amiodarone order chest-xray, liver function test, and thyroid test every 3-month during first year, then every 6 months.
4. Patients on antiarrhythmic agents should have liver enzymes measured the first 4-8 weeks of therapy and patients with risk factors for developing cardiac complications to therapy should have electrocardiograms ordered the first weeks of drug therapy and then in 3-6 months may be helpful to reduce risk of toxicity.
5. Patients on digoxin should be carefully monitored for digitalis toxicity (serum drug levels are not routinely ordered); electrolytes, BUN, creatinine and ECG are often ordered 1-2 weeks after therapy is initiated, and then every 1-6 months.
6. Monitor INR in-patients on warfarin every 2-3 days until therapeutic level is attained; after therapeutic level is achieved, monitor INR weekly for several weeks to ensure maintenance of therapeutic level, then every 1-2 months.