AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
SCIENTIFIC ASSEMBLY
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Management of Acute Atrial Fibrillation

Atrial fibrillation is one of the most common dysrhythmias encountered in the emergency department. Recent advances in the treatment of atrial fibrillation may improve the way in which emergency physicians treat this condition. Indications for admission will also be discussed in light of these advances.

- Discuss the pathophysiology of atrial fibrillation.
- Describe the efficacy and role of established and new treatments of acute atrial fibrillation.
- Discuss the admission criteria for new-onset atrial fibrillation.

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Wyeth-Ayerst: Speaker

FACULTY

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THE MANAGEMENT OF ACUTE ATRIAL FIBRILLATION

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BRIEF DESCRIPTION OF THE COURSE:

Atrial fibrillation is the most common dysrhythmia seen in the emergency department. Recent advances in the treatment of atrial fibrillation may improve the way emergency physicians treat this condition. This course will discuss the pathophysiology of atrial fibrillation. It will also discuss how patients with atrial fibrillation typically present to the emergency department, as well as the evaluation of patients with this condition. It will detail the pharmacologic management of atrial fibrillation including the efficacy and role of established and newer drugs utilized in the management of atrial fibrillation. In addition, recent studies regarding anticoagulation in atrial fibrillation will be reviewed. Nonpharmacologic methods of preventing and treating atrial fibrillation will be also addressed.

THE MANAGEMENT OF ACUTE ATRIAL FIBRILLATION

Jorge A. Martinez, MD, JD, FACEP, FACP

I. STATISTICS

✶the most common sustained arrhythmia
✶most common arrhythmia presenting to the ED (34.6% of arrhythmias)
✶present in 0.4% of the general population (1 million people)
✶increased incidence in people with DM, rheumatic heart disease, CAD, hypertension, CHF
✶incidence of Afib increases with age
-5% of people > 60 yo
-10% of people > 70 yo
-14% of people > 84 yo
✶2:1 men > women
✶1.3-2.6x increased mortality (especially with increased age, aortic valve disease, CAD, CHF; 12x greater risk in patients with hypertension; 17x greater risk in mitral stenosis)
✶80% with nonvalvular heart disease; 10% with valvular heart disease
✶10% with HOCM; 20% with dilated cardiomyopathy
✶3-20% of MI's complicated by Afib
✶2.5-5x greater risk of stroke in the general population. Factors which increase stroke=age; RHD; CHF; dilated LA; previous MI; hypertension; if TEE demonstrates L atrial thrombus, L atrial "smoke", or reduced blood flow in L atrial appendage
✶causes 45% of embolic strokes
✶nonrheumatic Afib causes 75,000 strokes/year
✶responsible for more physician office visits, ED visits, hospitalizations, and hospital days than any other arrhythmia

II. CAUSES OF ATRIAL FIBRILLATION

A) Electrophysiologic mechanisms:

1) *Reentry*
*** more common in patients with sinoatrial node dysfunction, high atrial pressures (pulmonary hypertension, mitral stenosis), hypoxic/ischemic atrial tissue, atrial distension or enlargement**
*** may see disparity in P wave duration on EKG prior to Afib**
*** higher dispersion of conduction velocities throughout atria as age increases**
*** reentrant mechanism is dependent on atrial mass and size**
***Theory:**

Multiple wavelets spread throughout the atria with different conduction velocities and refractory periods. Atrial fibrillation results from small reentrant circuits activating multiple areas of the atrial mass at the same time. The wavelets are constantly forming, collapsing, and reforming in

the atria as refractory atrial tissue is encountered and bypassed.
Result: multiple wavelets continually re-excite areas of the atrium that had been previously activated by a previous wavelet.

Atrial fibrillation continues until these cycles of wavelets disappear or fail to regenerate. Thus, the shorter the wavelength, the more cycles of wavelengths exist resulting in finer fibrillatory waves are finer. In this case Afib is more likely to persist. However, there are less cycles when the wavelength cycles are longer. These longer wavelength cycles produce coarse atrial fibrillation which is more likely to convert to atrial flutter or NSR.

• Classification of reentry causing Afib:
Type I: single wave front propagates throughout the right atrial free wall
Type II and Type III: the atrium is activated by two or more different activation waves which are separated by multiple lines of conduction block or areas of slow conduction

2) *Vagal mediated*: Increased vagal influence shortens atrial refractory period/increases atrial dispersion/no change in atrial conduction velocity
Clinical presentation=paroxysmal episodes; males 30-50 yo; normal hearts; onset usually at night or while sleeping

3) *Adrenergic mediated*: Increased sympathetic tone (ex. pheochromocytoma)
Clinical presentation=paroxysmal daytime episodes of Afib; usually associated with exercise or stress; evidence of heart disease

4) *Rapid firing single focus in atrium* = Lone atrial fibrillation; WPW Afib

B) Cardiac

- **Ischemic heart disease (CAD, unstable angina, variant angina, MI)**
- **Pericarditis/ Myocarditis/ Endocarditis**
- **CHF/Cardiomyopathy (HOCM, Dilated, Restrictive [Infiltrative], Constrictive)**
- **Sick sinus syndrome**
- **Wolff-Parkinson-White syndrome (Pre-excitation)**
- **Hypertension**
- **Post-Op cardiac surgery**
- **Valvular disease (mitral, tricuspid, aortic, pulmonic)**
- **Rheumatic heart disease (mitral stenosis, mitral regurgitation)**
- **Left ventricular hypertrophy (aortic stenosis)**
- **Congenital heart disease (ASD, VSD, Transposition of great vessels, Ebstein's malformation)**
- **Atrial myxoma, atrial thrombi**

C) Noncardiac

- **Hypoxia**

- **Pulmonary: pulmonary embolism, pneumonia, COPD, pulmonary hypertension**
- **Endocrine: thyrotoxicosis, pheochromocytoma, hypoglycemia**
- **Neurologic: head injury, intracranial hemorrhage, ischemic stroke, intracranial tumors**
- **Alcohol: Holiday heart syndrome**
- **Drugs: atropine, digoxin, theophylline, sympathomimetics, adenosine, antidepressants, anesthetics, caffeine**
- **Physical: lightning, electrical shock, hypothermia, thoracic trauma**
- **Metabolic: hypokalemia, hypomagnesemia, hypercalcemia**
- **Neuromuscular diseases: Friedrich's ataxia, muscular dystrophy**
- **"Lone" atrial fibrillation**
- **Miscellaneous: Push-up palpitations, heroin overdose, nicotine gum, malignancy involving atria, stress**

★Up to 30% of cases of Afib do not have an identifiable cause★

III. CLASSIFICATION OF ATRIAL FIBRILLATION

A) New onset atrial fibrillation: acute onset; ≈50% terminate spontaneously within 24 hours without antiarrhythmic therapy

B) Isolated: associated with an acute self limited disorder (ex. pericarditis, pulmonary embolus)

C) Paroxysmal: self-terminating episodes, usually lasting < 48 hrs

D) Persistent: not self-terminating, must be iatrogenically interrupted

E) Chronic: continuous Afib, difficult to maintain sinus rhythm, must anticipate and prevent complications

IV. PATHOPHYSIOLOGY

Acute onset of Afib→ atrial refractory period shortens→ loss of atrial contraction (loss of 10-40% of ventricular filling)→ increased & irregular ventricular rate → diastasis/diastole becomes shorter & erratic→left/right atrial dilation→ 50% increase in intra-atrial pressures (higher atrial pressure necessary to preserve forward flow from atrium to ventricle because atrial compliance decreases, ventricular volume increases, and ventricular diastole shortened); marked rise in L atrial pressure results in pulmonary edema → shorter diastolic filling period leads to decreased stroke volume & decreased cardiac output ("tachycardia-mediated cardiomyopathy" which is reversible when ventricular rate controlled) → dilation of atrial associated with stagnation of blood → increased incidence of thrombus→With cardioversion (electrical or chemical): ATRIAL STUNNING=atrial mechanical function does not return immediately; may require minutes to 6 months before atria return to normal function

V. CLINICAL PRESENTATION

A) *History*

Patients with Afib present with a variety of symptoms which range from

severe cardiac symptoms related to the rapid ventricular rate, to neurological symptoms related to thromboembolism, to incidental nonspecific complaints which are the result of the atrial fibrillation.

*** Asymptomatic**

*** Nonspecific: fatigue, dizziness/light-headedness, somnolence, tiredness, general ill health, shortness of breath**

*** Rate related: palpitations, cardiac ischemia (chest pain), hemodynamic instability (CHF, pulmonary edema), orthopnea, dyspnea with exertion, decreased exercise tolerance**

*** Loss of atrial contraction/AV synchrony: palpitations, hemodynamic decompensation (dyspnea on exertion, orthopnea, nocturia, paroxysmal nocturnal dyspnea), decreased exercise tolerance**

*** Neurologic: mental impairment (poor concentration, sleep or memory loss, irritability) acute paresis/paralysis, near syncope, syncope**

B) *Clinical Presentation*

Perform detailed physical examination; look for cardiac and noncardiac causes of the Afib.

In patients with healthy hearts: **The onset of Afib usually results in complaints of palpitations, decreased exercise tolerance, and exertional dyspnea (due to rapidly elevated intra-atrial and pulmonary capillary pressure).**

In patients with valvular or myocardial disease: **The disruption of normal ventricular filling due the loss of the atrial contraction (and consequently atrial-ventricular synchrony) leads to rapid onset of heart failure, hypotension, &/or syncope.**

Hemodynamically stable Afib **=ventricular rates < 140 bpm**

✶soft or absent S1 ✶irregularly irregular pulse

✶pulse deficits ✶loss of jugular A waves

✶physical signs of thyrotoxicosis, pulmonary embolus, pericarditis, etc.

Hemodynamically unstable Afib **=ventricular rates > 140 bpm**

✶irregularly irregular pulse ✶distended neck veins

✶pulmonary rales ✶hypotension ✶cardiogenic shock

✶physical signs of thyrotoxicosis, pulmonary embolus, pericarditis, etc.

VI. MEDICAL MANAGEMENT

American Heart Association Guidelines [Circulation 1996; 93:1262]

• Restoration and Maintenance of NSR

• Control of Ventricular Rate

• Prevention of Thromboembolism

"Atrial fibrillation begets atrial fibrillation... [l]ong-standing atrial fibrillation reduces the odds for successful cardioversion and for

maintenance of sinus rhythm. This results from a series of electrophysiologic and anatomic changes that occur [in the atrium] during atrial fibrillation and facilitate reentry within the atria." [Viskin, S, et al, The Treatment of Atrial Fibrillation: Pharmacologic and Nonpharmacologic Strategies, Current Problems in Cardiology, 22(2), February 1997 @ 60.] Simply put, the longer the atria remain in atrial fibrillation, the more prone they are to stay in atrial fibrillation or convert back to atrial fibrillation, even after conversion to sinus rhythm. Thus, the main thrust of the management of atrial fibrillation is to control the ventricular rate and restore sinus rhythm as soon as feasible.

VII.

EVALUATION AND TREATMENT OF ATRIAL FIBRILLATION

A) *Determine and address the underlying cause(s) of atrial fibrillation:*

***Place the patient on cardiac monitor, establish intravenous line, administer oxygen (as needed), and monitor vitals signs closely;**

***Perform detailed history and physical examination. Determine which signs and symptoms are the cause of the Afib vs the consequence of Afib, ex. CHF, cardiac ischemia, neurologic changes**

***Recommended laboratory studies: CBC, routine chemistry (check electrolytes), PT/PTT, Thyroid function tests, CXR, ECG, Echocardiogram, toxicology screen**

- **Electrocardiogram- irregularly irregular rhythm; no visible P waves; isoelectric or fine/coarse fibrillatory baseline; narrow QRS complexes; Ashman's phenomenon**
- **Echocardiogram (TEE best)-determine atrial size; measure left ventricular function and dimensions (chamber & wall); evaluate mitral and aortic valves; search for L atrial "smoke" or thrombus; assess mechanical atrial function after cardioversion**

B) *Control the Ventricular Rate:*

Ventricular rate in Afib typically 130-200 bpm (if conduction system intact) Thus, younger patients have higher ventricular rates compared to elderly, who usually have AV nodal disease

GOAL: Ventricular rate <100/min

❦ *If Hemodynamically unstable*=ELECTRICAL CARDIOVERSION

Limited to unstable patients; effectiveness depends on how long patient has been in Afib; failure rates=20-50%; painful; use sedation- maintain systolic > 90 mmHg; intubate if necessary; paddle placement-anterior/posterior (controversial) vs apex/base; synchronized cardioversion with expiration; Energy=100J converts 50%, 200J converts 85%, additional shocks=360J, wait 3 minutes between shocks to reduce transthoracic impedance; in patients taking digoxin=less energy needed if patient on digoxin; do not cardiovert if patient digoxin toxic. [Hebert M, Atrial fibrillation: Handling the acute emergency, *Emergency Medicine*, 1998; 30(5) @27-28.]

❦ *If Hemodynamically stable*=PHARMACOLOGIC CONTROL OF RATE

a) <u>CALCIUM CHANNEL BLOCKERS</u>: May precipitate CHF, AV block, and bradycardia; hypotension may be due to vasodilation or negative inotropy

♥<u>VERAPAMIL</u>: blocks AV node; (-) inotrope; rapid onset of action
_ Loading dose: 2.5-10 mg over 2 min IV; repeat q 30 min (pretreat with 100mEq calcium to reduce risk of hypotension)
_ Maintenance: IV=0.125 mg/kg; PO=120-360 mg/day in divided doses
***Comments:* May cause hypotension, bradycardia, or CHF; increases digoxin level; acts synergistically with digoxin; ↓ clearance of metoprolol; avoid in WPW Afib**

♥<u>DILTIAZEM</u>: blocks AV node; (-) inotrope (less than Verapamil)
_ Loading dose: IV=0.25-0.35mg/kg over 2 minutes (pretreat with 100mEq calcium)
_ Maintenance: IV=5-15mg/hr; PO=90-360 mg/day in divided doses
***Comments:* May cause hypotension, bradycardia, or CHF; acts synergistically with digoxin or B-blockers**

b) <u>BETA BLOCKERS</u>: Block AV node, negative inotropes, may precipitate bronchospasm (COPD & asthma); may precipitate vasoconstriction in PVD

♥<u>ESMOLOL</u>: probably B-blocker of choice; rapid onset; short duration (T1/2=9 minutes); only in IV form
_ Loading dose: 0.5 mg/kg IV over 1 minute; repeat as necessary
_ Maintenance: 0.05-0.2 mg/kg/min IV (5-20 mg/hr)
***Comments:* Short acting; hypotension common**

♥<u>OTHER B-BLOCKERS</u>

1) <u>Metoprolol</u>: Loading: IV=5mg over 2-4 min IV x 3 doses (max=15mg) ; Maintenance: IV=5-10 mg q 6 hrs; PO=50-100 mg bid

2) <u>Atenolol</u>: Loading: IV=5 mg over 5 min; repeat in 10 minutes Maintenance: PO=25-100 mg once daily

3) <u>Propranolol</u>: Loading: IV=0.5-1.0 mg q 5 min (max=0.15-0.2mg/kg) Maintenance: PO=40-240 mg/day in 3-4 divided doses
***Comments:* use propranolol with extreme caution in patients with CHF and bronchospastic disease**

c) <u>DIGOXIN</u>: Effect mediated primarily through Vagus nerve; positive inotrope; slow onset of action; requires incremental administration; mainly used to potentiate the AV nodal blocking effect of Ca channel and B-blockers; BEST USE=Afib with CHF or sedentary patients with Afib
_ Loading dose: IV=1mg over 24 hrs in 0.25-0.5 mg increments q 6-8 hrs
_ Maintenance: IV or PO=0.125-0.25mg qd
***Comments:* loading may take several hours; may take up to 2 hrs for peak onset; effectiveness attenuated with high catecholamine states (fever, exercise, thyrotoxicosis); excretion impaired in elderly or renal dysfunction**

d) <u>SOTALOL</u>: B-blocker with Class 3 antiarrhythmic properties;

***Best effect when added to digoxin=reduces resting heart rate & heart rate at low levels of exercise; Also lowers peak exercise heart rate;**
NOT 1st line B-blocker;
***BEST USE*: paroxysmal Afib= reduces frequency of paroxysms**

e) <u>AMIODARONE</u>: Intravenous amiodarone more effective in converting Afib to NSR and controlling heart rate than digoxin; central venous access required to administer; intravenous administration may cause hypotension, AV block, ventricular arrhythmias
***BEST USE*: paroxysmal Afib=reduces frequency of episodes and ventricular rate during paroxysms**
_ Loading dose: IV: 300 mg over 30 min;
_ Maintenance: IV: 1-2 gm over 24 hrs

f) <u>MAGNESIUM</u>: Inexpensive; slows ventricular rate; usually given in combination with digoxin; administration of Mg increases K loss; contraindicated in renal failure
_ Loading dose: IV= 2 gm bolus; Maintenance=1gm/hr IV
***Caution*: Watch for respiratory depression or hypotension; maintain Mg level at 2.5 mmol/L; monitor K & keep at no less than 4.0 mmol/L**

✷Suggested Ventricular Rate Control Choices [Blitzer M, et al, Rhythm Management in atrial fibrillation-with a primary emphasis on pharmacological therapy: Part 1, *PACE* 1998; 21 @ 596.]
- **<u>No structural heart disease</u>= Ca blockers > B-blockers > Digoxin**
- **<u>Hypertension</u>= Ca blockers (if LVH) > B-blockers > Digoxin**
- **<u>Ischemic heart disease</u>= B-blockers > Ca blockers/Digoxin > Ablation**
- **<u>Sick sinus syndrome</u> =Pindolol > Digoxin > Pacemaker plus drug**
- **<u>CHF/Dilated cardiomyopathy</u> =Digoxin>B-blocker>Amiodarone vs. ablate & pace**
- **<u>HOCM</u> =B-blocker > Verapamil > Diltiazem/amiodarone vs. ablate & pace**
- **<u>COPD</u> =Verapamil > Diltiazem > Digoxin**
- **<u>PVD</u> =Diltiazem > Verapamil > Digoxin**

C) <u>*Prevent thromboembolism:*</u>
- **patient often not certain of time of onset of Afib**
- **research suggests that thrombi form within hours of onset of Afib**
- **atrial stunning persists after cardioversion = may still develop interatrial clots=coumadin required for 4-12 weeks post-conversion (discontinue after NSR x 2-4 weeks)**
- **risk of stroke escalates when INR < 2.0; Best stroke reduction if INR between 2.0-3.0; Increased bleeding if INR > 3.0. An INR between 2.0-3.0 has been determined to be safe and efficacious because 1) The target INR in the five major trials ranged from 2.0-4.2; 2) Most strokes occurred in patients taking oral anticoagulants when the INR was subtherapeutic; 3) The bleed rate increased substantially in elderly patients when the INR was > 3.0. [See J Crit Ill Jan 1999; 14:11-19]**

♥ **STUDIES:**

① ***The Copenhagen AFASAK Study*** **[Lancet 1989;1:175-178]: Target INR=2.8-4.2; 1,007 patients; Warfarin vs ASA(75mg) vs Placebo**

***End point =thrombotic event*: Warfarin=2.0%; ASA + placebo=5.5%)**

② ***Stroke Prevention in Atrial Fibrillation*** **[Circulation 1991;84:527-539]: Target INR=2.0-4.5; 1,330 patients; Warfarin vs ASA (325mg)**

***End point =thrombotic event*: Warfarin=2.3%; Placebo=7.4%; ASA=3.6%**

③***Boston Area Anticoagulation Trial for Atrial Fibrillation*** **[N Engl J Med 1990;323:1505-1511]: Target INR=1.5-2.7; 420 patients; Warfarin vs No therapy**

***End point=ischemic stroke*: Warfarin=2.14%; Control=2.98%**

④***Canadian Atrial Fibrillation Anticoagulation Study*** **[J Am Coll Cardiol 1991;18:349-355]: Target INR=2.0-3.0; 378 patients; Warfarin vs Placebo**

***Endpoint=Stroke, emboli, intracranial or fatal hemorrhage*: Warfarin=3.5%; Placebo=5.2%**

⑤***Veterans Affairs Stroke Prevention in Nonrheumatic Atrial Fibrillation*** **[N Engl J Med 1992;327:1406-1412]: Target INR=1.4-2.8; 525 patients; Warfarin vs Placebo**

***End point=Clinical cerebral* infarction: Warfarin=0.9%;Placebo=4.3%**

⑥***European Atrial Fibrillation Trial*** **[Stroke 1989;20:1000-1004]: Target INR=2.5-4.0; 1007 patients; Warfarin vs ASA (300mg) vs placebo**

***Endpoint=Stroke, peripheral embolus, MI:* Warfarin reduced stroke risk from 12 to 4%; ASA=15%; Placebo=19%**

⑦***SPAF-II*** **[Lancet 1994;343:687-691]: (Direct comparison between warfarin & ASA) 1100 patients (excluding Lone Afib); Target INR=2.0-4.5; Warfarin vs ASA (325mg/d); Risk of thromboembolism reduced to 3.% with Warfarin; 4.8% with ASA; Higher incidence of bleeding and ICH in elderly taking anticoagulants**

⑧***SPAF-III*** **[Lancet 1996;348:633-638]: (Warfarin & ASA combined) 1044 patients with 1 risk factor for thromboembolism; Randomized to Warfarin @ 1.2-1.5 INR + ASA @ 325mg/d vs Warfarin @ 2.0-3.0 INR (dose adjusted).**

***Results:* Warfarin + ASA group with stroke or embolization in 7.9%/yr vs 1.9%/yr for dose adjusted Warfarin**

For patients with low risk for stroke = Patients on ASA had stroke rate = 1.4%/yr; Patients on warfarin @ INR of 2.0-3.0, stroke rate =1.1%/yr

⑨***Atrial Fibrillation Investigators*** **[Arch Intern Med 1994;154:1449-1457]:**

1) Warfarin significantly lowered annual stroke rate in patients with Afib to 1.4% (68% risk reduction compared to placebo) .

2)Results of trials underestimate the value of warfarin in Afib because many had INR's below current recommended levels.

3) Warfarin's stroke prevention reduced mortality by 33%.

4) Warfarin with NO BENEFIT in patients < 65yo with Lone Afib.

5) The risk of major bleeding was 1.3% with warfarin; 1% with ASA or placebo. Most common finding with bleeding was hypertension.

♥ ***American College of Chest Physicians Guidelines for anticoagulation of***

patients with Afib **[*Chest 1995;108:352s-359s*]; [*See also J Crit Ill; Jan 1999; 14:11-19*]:**

♥ Long-term anticoagulant therapy (INR=2.0-3.0) should strongly be considered for all patient with AF who are > 65 yo

♥ Long-term anticoagulant therapy should be strongly considered for patients < 65yo with any of the following risk factors: previous TIA or stroke, HBP, DM, clinical CAD, mitral stenosis, prosthetic heart valve, thyrotoxicosis

♥ Patients who decline oral anticoagulant therapy or who are poor candidates for anticoagulation therapy should be given ASA, 325 mg/day

♥ Patients < 65y/o with no risk factors for stroke can be appropriately treated with ASA or no antithrombotic therapy

♥ For patients between 65 - 75 y/o without additional risk factors, absolute risk reduction with either form of antithrombotic therapy (oral anticoagulation or ASA), side-effect profile, and inconvenience must be taken into account before selecting long-term treatment

♥ Oral anticoagulation is recommended for patients >75y/o because of high risk of stroke. The preferred target INR is the lower end of therapeutic range of 2.0 to 3.0.

♥ Anticoagulation recommendations for Afib < 48 hrs:

▪ Emergent cardioversion: heparinize prior to cardioversion; then coumadin x 3-4 weeks

▪ Elective cardioversion: (?) heparin (likelihood of emboli <48 hrs is low); TEE searching for inter-atrial thrombus; (?) anticoagulate with coumadin for 4-6 weeks post-cardioversion

♥Anticoagulation recommendations for Afib> 48 hrs:

▪ Prolonged anticoagulation (3 weeks) with coumadin required before attempting cardioversion

▪ TEE to search for inter-atrial thrombus or smoke; if present-continue anticoagulation x 3-4 weeks before cardioversion

▪ anticoagulate with coumadin x 4-12 weeks post-cardioversion

D) *Restore sinus rhythm*:

★ Reasons for restoring sinus rhythm:[Waktare,JEP, Camm AJ, Acute treatment of atrial fibrillation: Why and when to maintain sinus rhythm, *Am J Cardiol* March 1998 @ 4C.]

- **Appropriate/physiologic rate control**
- **Regularization of heart rhythm**
- **Restoration of atrial contribution to cardiac output**
- **Improvement in hemodynamics**
- **Maintenance of normal electrophysiology**
- **Prevention of L atrial dilation**
- **Prevention of L ventricular dysfunction**
- **Relief of symptoms, maintain quality of life, maximize life expectancy**
- **Reduce thromboembolic complications**

METHODS OF RESTORING SINUS RHYTHM

➢**ELECTRICAL CARDIOVERSION: Synchronized electrical cardioversion in unstable Afib**

➢**PHARMACOLOGIC (Class 1A, 1C, and Class 3 antiarrhythmics): These drugs prolong the refractory period of the reentrant wave fronts and impair conduction through the atrial tissue; *RESULT*: reduced number of existing and new wave fronts. They also suppress automaticity.**

a) Class 1A antiarrhythmics:

♥QUINIDINE: rarely used because IV administration causes hypotension; oral loading takes longer to convert Afib; multiple side effects= GI, proarrhythmia, sinus node suppression

♥PROCAINAMIDE: most commonly used drug to convert Afib; NSR restored in 90% of Afib < 1 day but only 9% of Afib > 1 week;
_ Loading dose: IV=15mg/kg given as 100mg q 5 min (max dose=1gm); Stop infusion if fails to respond to 1gm, if QRS or QTc intervals > 130% of baseline, >20% decrease in systolic BP;
_ Maintenance: IV=2-5mg/min infusion; PO=500mg-1gm q 8 hrs
***Comments*: proarrhythmic=torsades de pointe, vagolytic effect may increase ventricular rate therefore give AV blockers first**

b) Class 1C antiarrhythmics

♥FLECANIDE: oral form only in US; superior in preventing recurrences of paroxysmal Afib (68% event free after 4-8 weeks; BEST USE=facilitate conversion of Afib in patients without structural heart disease
_ Loading dose: PO=<400 mg over 3 hrs OR 300 mg single dose resulted in cardioversion in 68% in 3 hrs, 91% in 8 hrs
_ Maintenance: PO=100-200 mg in 2 divided doses
***Comments*: SIDE EFFECTS=paresthesia, visual disturbances, vertigo, fatigue, dyspnea. In addition, adverse cardiac events such as conduction disturbances, worsening of CHF or development of malignant arrhythmias is more common in patients with pre-existing structural heart disease.**

♥PROPAFENONE: oral form only in US; sodium channel, calcium channel, and potassium channel blockade; *BEST USE*=paroxysmal Afib
_ Loading dose: PO=150 mg q 4 hrs OR 600 mg single dose-conversion of 25% of Afib
_ Maintenance: PO=450-900 mg in 3 divided doses
***Comments:* 40-50% of patients remain free of paroxysmal Afib; SIDE EFFECTS=GI distress, regular tachycardia with prolonged QRS and 1:1 AV conduction; sinus node dysfunction**

c) Class 3 antiarrhythmics

♥SOTALOL: less effective in converting Afib; more effective in

maintaining NSR after conversion; less symptoms with recurrent Afib because of B-blocker activity
_ Loading dose: IV=1.0-1.5mg/kg over 10 minutes-reduces ventricular rate in 10 minutes
_ Maintenance: PO=80-320 mg q day
Comments: **QT prolongation with polymorphic ventricular tach (1.4%) and torsades de pointe; use with caution in CHF, renal insufficiency, and prolonged QT interval**

♥AMIODARONE: prolongation of refractoriness of atrial muscle, AV node, and ventricular muscle; B-blocker and Ca channel blocker actions; antagonizes thyroid hormone; IV dose preferable to PO dose because of extended loading period and large side effect profile with PO loading; extremely effective for rate control even in patients with high catecholamine levels (ex. CHF)
_ Loading: IV=5mg/kg diluted in 100 cc NS infused over 10-40 min; may give 2nd loading dose if no response to first dose
_ Maintenance: IV=600-1200 mg over 24 hrs; titrate to ventricular response
***Comments*: may be used in conjunction with digoxin &/or verapamil; more effective than digoxin or verapamil alone; 60% regain NSR within 6 hrs, 92% to NSR within 24 hrs; SIDE EFFECTS=hypotension and flushing within minutes of administration, bradyarrhythmias, thrombophlebitis in smaller veins; works better if LA <4.5 cm**

♥IBUTILIDE: specifically approved for conversion of Afib to NSR; IV form only; rapid onset & short half life (60 min); Action=delayed inactivation of Na slow channel (plateau prolongation) and K channel blockade (prolonged repolarization); converts 30-35% of new onset Afib; 70% of conversions within 60 min
_ Loading dose: IV=0.005-0.025mg/kg (average=1.0mg) over 10 minutes; may give 2nd dose after 10 minutes
_ Maintenance: none
***Comments*: QT prolongation=11%; torsade de pointes=3.6-8.3%-usually occurs before conversion to NSR; more common in LV dysfunction (BE PREPARED)**

★Ca channel and B-blockers=*NO EFFICACY IN CONVERTING ACUTE AFIB TO NORMAL SINUS RHYTHM*

♥♥Algorithm for antiarrhythmic use in atrial fibrillation: [Waldo AL, Prystowsky EN, Drug treatment of atrial fibrillation in the managed care era, *Am J Cardiol* 1998; 81(5A) @ 25C]
1) *Heart Disease*:
• High blood pressure: 1st=propafenone; 2nd=disopyramide, sotalol, amiodarone
• CHF: 1st=amiodarone

• Coronary artery disease: 1st=sotalol, disopyramide; 2nd=amiodarone, quinidine
• Other: 1st=sotalol, propafenone, disopyramide; 2nd=amiodarone, quinidine
2) *Lone Afib*: 1st=flecanide, disopyramide, sotalol; 2nd=propafenone, quinidine, amiodarone

E) *Proper disposition and follow up*:
✶ Traditional wisdom: admit all new onset atrial fibrillation for evaluation of etiology, stabilization, rate control, anticoagulation, and conversion to NSR
✶ Recent recommendations: [*Am J Cardiol* 1998; 81(5A)@31C]
1) Hospitalization necessary: I) clinically hemodynamically unstable or symptomatic; ii) Afib duration unknown with risk of thrombus formation; iii) embolic risk; iv) bleeding risk (with initiation of anticoagulation)
2) Case by case decision: I) asymptomatic, stable with HR<140/min; ii) unknown duration of Afib; iii) established recurrent paroxysmal Afib<12hrs & clinically stable; iv) Afib known to be present < 2 weeks
3) Outpatient evaluation and management allowed: I) rate control easily achieved; ii) chronic Afib > 1 week, clinically stable=anticoagulate as outpatient; iii) paroxysmal Afib by history already on anticoagulants; iv) permanent Afib, clinically stable & already on anticoagulants

VIII. SPECIAL SITUATIONS

1) "Lone" atrial fibrillation*:* Afib that develops without underlying structural heart disease or other precipitating illness.
STUDIES: ♥ *Kopecky et al* [*N Engl J Med 1987;317:669-674*]: Lone Afib defined as Afib in pts < 60 y/o without CAD, hyperthyroidism, valvular disease, CHF, cardiomyopathy, COPD or DM. CVA in only 1.5% of 97 pts over 15 years
♥ Brand et al [*JAMA 1984;254:3449-3453*]: Most patients > 70 y/o; stroke risk = 2.6%/yr
♥ *Atrial Fibrillation Investigators* [*Arch Int Med 1994;154:1449-1457*]: Lone Afib defined as Afib in absence of TIA, HBP, CHF, cardiomyopathy, angina, or MI. Overall stroke risk = 1.5%/yr;
Risk of stroke strongly related to age = a) <60yo=0%; 60-69y0=1.6%; 70-79yo=2.1%; >80yo=3%
♥ *Framingham Study* (average age=70 yo):Lone Afib caused 11% of Afib; stroke risk increased 4x; annual mortality rate=3.8%
♥ *Mayo Clinic* (average age=44 yo): Lone Afib in 2.7% of population over 30 yrs; stroke risk=0.55%; annual mortality rate=0.4%/yr
♥♥General recommendations: Anticoagulants not necessary in uncomplicated cases (<65 yo); Aspirin suggested in all cases

2) Atrial fibrillation in myocardial infarction*:* Occurs in 3-20% of AMI's, usually within the first week; increased short term and long term mortality;

mainly in elderly patients and/or large infarcts
AHA recommendations [J Am Coll Cardiol, 1996;28 @2346]:

1. Electrical cardioversion in patients with severe hemodynamic compromise or intractable ischemia.

2. Rapid digitalization to slow a rapid ventricular response and improve LV function.

3. Intravenous B-blockers to slow rapid ventricular response in patients c/o clinical LV dysfunction, bronchospastic disease, or AV block.

4. Heparin should be given.

3) Atrial fibrillation in Wolff-Parkinson-White syndrome:
Patient with WPW→develops AV nodal reentry with narrow complex tachycardia→AVNR may degenerate into atrial fibrillation→ if AV node bypassed and accessory pathway utilized→accessory pathway's refractory period different than AV node's refractory period→accessory pathway allows 1:1 conduction of atrial impulses to the ventricle→ventricular rate accelerates to atrial rate (may reach 300/minute; rapid, wide and irregular pattern)→ rapid ventricular rate degenerates to Vfib

★Treatment of choice= ELECTRICAL CARDIOVERSION★

★Antiarrhythmic drugs=Class 1A (Procainamide), Class 1C (Propafenone), and Class III (Amiodarone). DO NOT USE drugs that increase AV node blockade (B blockers, Ca channel blockers, digoxin).

4) Atrial fibrillation in thyrotoxicosis:
★Most common arrhythmia in thyrotoxicosis=15% in one study; incidence increases with age
★Increased blood volume and decreased peripheral resistance =pronounced palpitations
★Rapid ventricular rate and circulatory overload =congestive failure
★Embolic complications common (10-40%); incidence of emboli ⇑ with age
★Increased clearance at receptors= higher doses of B-blockers or digoxin
★Increased clearance of clotting factors= smaller dose of coumarin required
★B-blockers=initial drug of choice to control ventricular rate and symptoms
★Afib spontaneously to NSR in 62% of patients treated for thyrotoxicosis (⇑ resistance in patients with heart disease or long standing Afib)
★Elective cardioversion after patient euthyroid x 8-10 weeks (with anticoagulation)
★High recurrence rate with DC cardioversion; add Class 1A antiarrhythmics

IX. NON-PHARMACOLOGIC MODALITIES

1) *Prophylactic atrial pacing*:
Utilized in patients with paroxysmal Afib &/or electrophysiologic evidence of inter-atrial conduction delay

Benefit found in patients whose Afib is initiated by:
★ Increased vagal tone
★ Increased atrial ectopic beats
★ Sick sinus syndrome (23% with VVI developed Afib; 14% with AAI)
★ Without sinus disease but severe sinus bradycardia & high vagal tone prior to onset of Afib

2) *Implantable atrial defibrillator*:
◆ Utilizes R atrial and coronary sinus leads
◆ Atrial defibrillation @ 0.5-5.0 J in paroxysmal Afib; >5.0 J in chronic Afib
◆ *Key*: prevents atria from remaining in Afib=limits electrical remodeling in the atria
◆ Single chamber & dual chamber defibrillators being investigated; >500 implanted
◆ Noted to detect Afib; deliver appropriate shocks with defibrillation; rarely bradyarrhythmias or ventricular arrhythmias initiated
◆ Patients do feel chest pain due to skeletal muscle stimulation; may cause microscopic hemorrhage/necrosis and thrombosis at site of shock

3) *Surgical ablation*:
➢Corridor procedure: creates a corridor from the SA node to the AV node
***Pros:* allows impulse from SA directly to AV node; protects AV node from rapid fibrillatory rate; *Cons*: Atria continue to fibrillate=loss of AV synchrony & continued risk of thrombus**

➢Maze procedure: used to atrial tissue mass necessary to sustain Afib; involves multiple incisions in both atria & bilateral appendectomies=series of dead-ends for wavelets⇒Afib cannot be maintained ⇒ atria reverts to NSR
***Pros:* Afib, thrombus formation; *Cons*: prolonged atrial recovery time; requires open heart surgery⇒reserved for drug refractory Afib**

4) *Catheter ablation*:
Indirect ablation: radiofrequency ablation of AV node &/or His Bundles; controls ventricular rate by producing complete heart block; atria continue to fibrillate⇒ loss of AV synchrony and continued risk of intra-atrial thrombus
Direct ablation: similar to maze procedure; Goal: to compartmentalize the atrium by creating transmural linear lesions in the atrial myocardium which will block the wave fronts; when enough atrial tissue compartmentalized⇒Afib cannot be maintained⇒atria return to NSR
***Problems*: R atrial ablation alone does not eliminate Afib; interatrial tachycardias may develop after ablation of tissue; radiofrequency causes ⇑⇑ heat in atria ⇒ atrial charring & thrombus formation**

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