ATRIAL FIBRILLATION

Dx by ECG: irregularly irregular QRS complexes without p waves; QRS is usually narrow, unless there is aberrant conduction in His-Purkinje system (e.g. pre-existing BBB)

Definitions:
paroxysmal (vs. persistent) – starts & stops spontaneously (tx as chronic)
chronic – persists despite tx or based on a decision not to convert
lone – occurring in pt <65 yo & not assoc w/ any other cardiovascular dz

Mechanism: multiple migrating reentrant microwavelets, sometimes arising from repetitive activation of ectopic foci in pulm veins; chronic arrhythmia leads to atrial electrical remodeling ("afib begets afib")

Secondary Causes (acute vs. chronic*)
atrial stretch: htnsive crisis, HF, acute pulmonary dz (hypoxia, PE, COPD, pna), htn*, ASD*, valve dz* (esp mitral), cardiomyopathy*
atrial irritatability: ischemia*/MI, myocarditis/pericarditis, hyperthyroid, post-cardiac sx, drugs/ingestions (theophylline, caffeine, EtOH, amphetamines, cocaine), high catecholamine states (post-op, infxn, dehydration, stress, sleep deprivation)

Effects: dyssynchrony & loss of atrial "kick" to diastole; rapid ventricular response ("RVR"), disorganized atrial activity & stasis (→ clot); chronic tachycardia can induce cardiomyopathy

Symptoms: aSx, palps, hypotsn, HF Sx; most common is *fatigue & non-specific complaints*

Overview of Management
1) decide if & when rhythm control is needed *(*benefits: Sx relief, stroke prevention, avoidance of cardiomyopathy)*
2) establish rate control
3) long-term stroke prevention

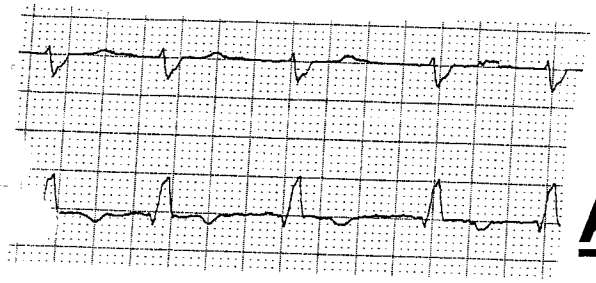
Initial Approach
1) Is the pt unstable? (hypotsn, bad HF, ACS, demand infarct, WCT w/ pre-excitation) → DC cardioversion
(start biphasic @100J, 150J, then 200J; start monophasic @200J)
2) If pt stable, is there RVR? If yes..
→ start rate control tx...
if nl LV fxn & no active HF: use B-blocker or CCB
if LV systolic dysfxn (EF<40%) or active HF: consider dig, IV esmolol, IV amiodarone
*treat any reversible underlying cause (e.g. infxn, dehydration)
→ If pt spontaneously converts to sinus, focus on treating any reversible underlying causes
3) If pt rate-controlled & still in afib, start anticoagulation (IV hep gtt or LMWH)...
→ if definite onset <48 hr (rarely the case) AND no structural cardiopulm dz
(e.g. LV dysfxn, htn, h/o PE, MV dz, etc.) → acute cardioversion (chemical or DC)
→ if duration >48 hr / unknown OR known structural cardiopulm dz...
- consider TEE to rule out LA/LAA clot → cardioversion
- consider full anti-coag x 3 -4wks → cardioversion +/- TEE → 4-12wks anti-coag *(ACUTE, NEJM 2001;344:1411)*
- consider starting long-term anticoag if deemed likely to recur

Dosing of meds you might use ...	
diltiazem	IV push: 20 mg (or 0.25 mg/kg) IV push; if inadequate after 15 min, may repeat 25 mg (or 0.35 mg/kg) IV push drip: 5 mg/hr titrated up to 15 mg/hr po: 15-30 mg q6h short-acting, then switch to long-acting after titration
metoprolol	5 mg IV push q5min up to 3 times prn
esmolol	drip: 0.5 mg/kg push, then 0.05 mg/kg/min (short t1/2)
digoxin	0.5 mg IV push, then 0.25 mg IV q6h x 2
amiodarone	150 mg IV push, then 1 mg/min x 6 hr, then 0.5 mg/min x 6hr, then 600 mg po bid x 2wk, then 200-400 mg po qd

Stroke Prevention
- Low risk pts (no RFs*) have annual stroke risk rate of 2-3% per yr; any RFs have risk 5-7% per yr
- Aspirin or warfarin better than placebo *(SPAF, Circ 1991;84:527. BAATAF, NEJM 1990;323:1505.)*
- Warfarin better than aspirin in elderly *(AFASAK, Lancet 1989;28:175.)* but ↑bleed risk in <75 yo, though risk is more acceptable given benefits in >75 yo *(SPAF-II, Lancet 1994;343:687.)*
- Aspirin 325 mg qd is adequate for low-risk pts w/o *RFs (consider weighing risk of stroke vs. bleed in age 65-75); warfarin recommended for everybody else *(SPAF-III, JAMA 1998;279:1273.)*

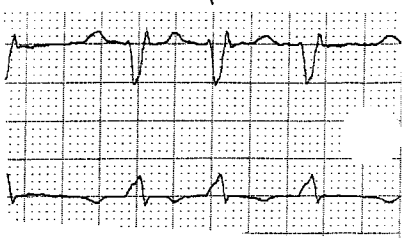
***Risk factors**: htn, previous TIA/CVA, HF/LV dysfxn, MV dz, age>75*

Rhythm vs Rate Control
- No mortality benefit of rhythm vs. rate control w/ minimal Sx *(AFFIRM, NEJM 202:347:1825. RACE, NEJM 2002;347:1834.)*
- However, consider rhythm control for symptomatic afib & young pts w/ minimal Sx



Atrial Fibrillation

— Sap Hakdar
July 2002



Definition: rapid, irregular fibrillatory waves that vary in size, shape and timing.

Mechanisms

- Trigger (autonomic tone, Δwall tension)
  - arising reentrant circuit
  - extinction
  - Collisim
- Ectopy at pulmonary veins

⟹ if chronic ⟹ atrial electrical remodeling

⇓

"the longer you stay in afib, the harder it is to get out!"

Hemodynamic effects:

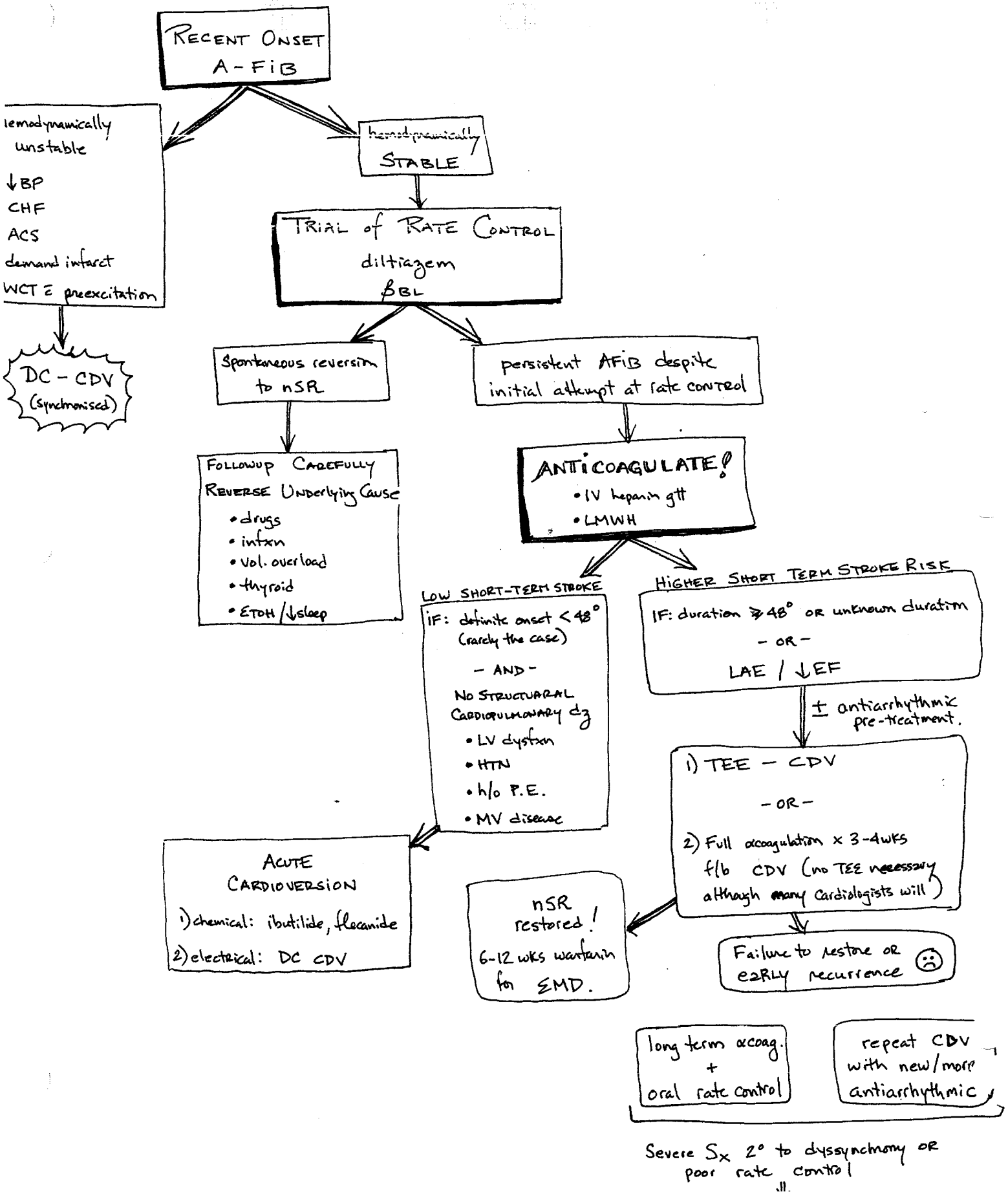
- Loss of atrial component ("kick") to diastole (dys-synchrony)
- Rapid ventricular response
- Disorganized atrial activity and stasis ⇒ clot!

⟹ if chronic, tachycardia-induced CM

Symptoms: asymptomatic, palpitations, hypotension, CHF/pulmonary oedema.
Pearl: *fatigue and nonspecific complaints are most common presentation*
Atrial fibrillation impairs quality of life in many!

3 KEY QUESTIONS IN THE APPROACH TO ATRIAL FIBRILLATION

1) **When and how should I ultimately restore sinus rhythm?**
2) **How should I achieve appropriate rate control?**
3) **What do I do for stroke prevention?**


RECENT ONSET A-FIB
Hemodynamically unstable
↓BP
CHF
ACS
demand infarct
WCT ± preexcitation
DC-CDV (synchronised)
hemodynamically STABLE
TRIAL of RATE CONTROL
diltiazem
βBL
Spontaneous reversion to nSR
FOLLOWUP CAREFULLY
REVERSE UNDERLYING CAUSE
• drugs
• infxn
• vol. overload
• thyroid
• ETOH / ↓sleep
persistent AFIB despite initial attempt at rate control
ANTICOAGULATE!
• IV heparin gtt
• LMWH
LOW SHORT-TERM STROKE
IF: definite onset < 48° (rarely the case)
- AND -
NO STRUCTUARAL CARDIOPULMONARY dz
• LV dysfxn
• HTN
• h/o P.E.
• MV disease
HIGHER SHORT TERM STROKE RISK
IF: duration ≥ 48° OR unknown duration
- OR -
LAE / ↓EF
± antiarrhythmic pre-treatment.
1) TEE - CDV
- OR -
2) Full acoagulation x 3-4wks f/b CDV (no TEE necessary although many cardiologists will)
ACUTE CARDIOVERSION
1) chemical: ibutilide, flecanide
2) electrical: DC CDV
nSR restored!
6-12 wks warfarin for EMD.
Failure to restore OR EARLY recurrence ☹
long term acoag. + oral rate control
repeat CDV with new/more antiarrhythmic
Severe Sx 2° to dyssynchrony OR poor rate control

Drugs for Rate control in Atrial Filbrillarion

Drug	Dose for acute control	Sustained therapy	Comments
Diltiazem	20mg IV bolus → 20mg 15 minutes later → maintenance infusion at 5-20 mg / hr.	Diltiazem controlled release 180-300 mg PO QD.	Can cause hypotension. Works synergistically with Digoxin.
Verapamil	5-10 mg IV push over 3 min → can repeat 30 min later'; No reliable maintenance infusion rate	Verapamil SR 120 – 240 mg QD or BID	Negatively inotropic. Can elevate digoxin level.
Metoprolol	5 mg IV push. Can repeat Q5 min for total of three doses. No IV continuous infusion rate.	50 to 400 mg daily divided in BID doses (e.g. 100 mg po BID)	Ischemic protection in CAD May cause AV block Negative inotropy
Esmolol	0.5 mg/kg IV push, repeat Q 1-2 minutes prn and then start infusion @ 0.05 mg/kg/min titrating up to 0.2 mg/kg/min	No PO therapy. IV gtt 0.05 – 0.2 mg/kg/min	Causes hypotension. Fast on – fast off.
Digoxin	1.0-1.5 mg IV or orally over 24 hr in doses of 0.25 to 0.5 mg.	0.125 to 0.5 mg qd po or IV.	Renally cleared! Minimal utility in adrenergic states. Poor efficacy in exertional HR control

Pearl: *Use one rate controlling agent at a time (with the exception of perhaps dig). Sosnay committed no less than three AV nodal assassination as an intern! Watch your SBP and CHF symptoms!*

Drugs for restoration and maintainance of sinus rhythym

(kids, don't try this at home without a monitor!)

Drug	Dose conversion	Dose for maintenance	Comments
Flecanide	300 mg PO	50 –150 mg PO BID	FDA approved ONLY for paroxysm structurally normal heart! (but works great!) hepatically cleared.
Propafenone	600 mg PO	150 – 300 mg PO BID	Same as flecanide
Dofetalide	0.5 mg PO BID	0.5 mg PO BID	Must be hospitalized to start drug
Procainamide	100 mg IV Q 5min to max dose of 1000 mg	Procainamide slow release 1000 – 2000 mg PO BID	Drug induced SLE Not FDA approved for AF Useful in preexciteation + afib
Ibutilide	1 mg IV over 10 min (if > 60 kg) 0.01 mg/kg over 10 min (if < 60kg) may repeat once if afib persists in 10 min	No PO maintenance therapy Available	Can cause torsade (esp if low EF) Useful in pre-treatment for DC CDV of chronic afib to promote maintenance.
Amiodarone	1200 mg IV infusion over 24 hours (the so called "amio- load"	600mg/day for 2 weeks, then 200-400mg / day thereafter	Slow onset moderate conversion efficacy causes bradycardia long-term toxicity

In broad strokes...

- Paroxysmal afib with structurally normal heart → flecanide, propafenone.
- CAD (but no CHF) → sotalol, amidarone
- CHF (EF < 35%) → amiodarone (and sometomes dofetalide)

Pearl: *amiodarone and dofetalide are the only drugs that are proven to NOT kill people with heart failure! (sorry for the double negative)*

Pearl: *Failure to terminate afib with a specific antiarrhythmic agent does NOT mean that the same drug will be ineffective in maintaining sinus rhythm after electrical cardioversion.*

Long Term Anticoagulation

It's all about **RISK STRATIFICATION** to determine the absolute stroke risk vs. bleeding risk on anticoagulation!

Risk factors for stroke

- Hypertension
- Previous TIA / stroke
- CHF or LV dysfunction
- Mitrai vaive disease
- Age > 75

If no risk factors (i.e. low risk), annual stroke rate is 2-3% / year → may be able to use ASA.
If any risk factors, annual stroke risk is 5-7% / year → warfarin please!

Anti-thrombotic therpy (*SPAF-III data – Blackshear et al, Lancet 1996;348:633-8*)

Age	Risk Factors for Stroke	Rcommended Therapy
< 65	None	Aspirin (no proof that ASA Better than placebo in this group)
> 65-75 (those with higher bleeding rate in SPAF)	None	Risk stratify for hemhorrhage c/w Small stroke risk
Any age	One or more	Warfarin strongly advised unless clear Contraindications are present!

General principles gleaned from clinical trials:

- All patients with atrial fibrillation should receive long-term anticoagulant therapy with warfarin unless they are young (<65) and have NO risk factors (not even HTN) – therefore the patients who can get away with no anticoagulation are rare!
- In the absence of risk factors aspirin alone may be adequate (no clinical trial has compared ASA vs. placebo)
- Advanced age (>65-75) is a risk factor for both stroke and bleeding.
- In patients who are at high risk for stroke, the relative risk of stroke exceeds that of bleeding. Warfarin should be used if at all possible.

Pearl: *Both the risk of stroke and the efficacy of warfarin among patients with persistent arrhythmia were EQUIVALENT to those with paroxsysmal arrhythmia. (SPAF-II data)*