Case Studies: Patient Assessment

- Symptom history
  - Frequency consider cardioversion
  - Assess for prophylaxis
  - Anticoagulation
- ECG
  - Ventricular response
  - QRS and QT interval
- Hemodynamic stability
- Echocardiogram
  - Structural heart disease
  - LV ejection fraction
- Thyroid Function
- Drug history
Case I

AL is a 61 year old female with valvular heart disease (secondary to rheumatic fever) who presents to the clinic with chief complaints of shortness of breath on exertion and palpitations. She states that these symptoms presented when walking her dog last evening. Upon rest she notes that she is no longer short of breath but still feels heart palpitations. AL was noted to be in no apparent distress during the physical exam.

Vitals: BP 113/76 mmHg, Pulse 133 beats/min irregular, Resp 16/min and afebrile

PMH: Mild/moderate mitral valve regurgitation
    Dilated cardiomyopathy
    Congestive heart failure (NYHA class II)

ECG: Atrial fibrillation, rate 95-145 beats/min, all intervals within normal limits.

Medications: furosemide 40 mg Bid
            captopril 25 mg BID
Case I

1. **Patient Assessment:** This appears as the patient's first sign and symptoms of AF. The patient has classical signs of AF including shortness of breath on exertion and palpitations. It appears that she has AF due to structural heart disease, that is valvular heart disease.

The patient is clinically stable with normal blood pressure and no signs of myocardial ischemia. The patient has an irregular pulse at a fast rate of 133 BPM. Clinically, without performing an ECG, these signs and symptoms are clearly indicative of AF. The ECG confirms this finding. Of note, all ECG intervals are normal including QRS and QTc. The patient is receiving a diuretic and ACEI likely due to her valvular heart disease. With rheumatic heart disease, mitral valve regurgitation is common. To reduce the regurgitant jet, volume overload and dilated cardiomyopathy, after load reduction therapy is needed. Even with these drugs, the patient has developed a dilated cardiomyopathy with mild CHF symptoms.

2. **Design a therapeutic plan.:**
   - **Explain any additional information needed prior to designing a therapeutic plan.**
     - Echocardiogram to establish an ejection fraction
     - Thyroid function test to rule out other causes
     - Drug history including over the counter sympathomimetic drugs which can precipitate AF
     - Exact time of symptom onset
     - History of bronchospastic disease
     - History of long QTc interval
     - History of recent surgery, peptic ulcer disease or cerebral vascular accidents
   - **What should be done to treat the patient’s symptoms?**
     First order of business is to lower the ventricular rate <100 BPM. Assuming that all of the unknowns listed above are negative except for EF; beta blocker therapy would be the first line of therapy given that hemodynamics are stable. I would recommend either atenolol or metoprolol given orally as 50 mg bid. If more rapid rate control is desired it is acceptable to use IV metoprolol or atenolol.
Case I

AL is asymptomatic and resting comfortably. AL, however remains in AF.

• Explain the pros and cons of rate control management versus cardioversion and maintaining normal sinus rhythm.

Once the patient is committed to rate control and chronic AF it becomes increasingly difficult to maintain normal sinus rhythm. After one year of AF, it becomes nearly impossible. Hence, chronic AF/rate control strategy must be given careful consideration. Is the patient a candidate for life long anticoagulation therapy? Does the patient need normal atrial function to maintain NSR? Can the patient tolerate effective rate control therapy? As the patients ventricular function declines, it becomes increasingly difficult to tolerate these drugs. In such cases, because NSR can not be restore, the patient must have their AV node destroyed (ablated) requiring permanent ventricular pacing. Because of these many unknown issues, most physicians become aggressive in restoring and maintaining NSR. However, maintaining NSR is not without problems. In most cases, antiarrhythmic drugs must be used. The risk of these drugs can be huge especially in patients with significant structural heart disease. Treating each episode with repeated electrical cardioversion is not convenient nor cost effective. There may be a role for implantable defibrillators for this purpose in patients who require NSR but can not use antiarrhythmic drug maintenance. Given the pros and cons of each strategy, the NIH is sponsoring a large clinical study, AFFIRM, which will provide an answer to whether chronic AF/rate control strategy is better, equal or worse than NSR/maintenance strategy regarding morbidity and mortality.
• It was decided to try pharmacologic CV. What would be the most appropriate therapy?

First, you must be certain that the AF is < 48 hours in duration. If not then a trans esophageal echocardiogram must be used to rule out an atrial thrombus. If either criteria is not met, the patient should be put on coumadin for 3 weeks before attempting cardioversion.

If the patients Mg++ and K+ are within normal range as is the QTc interval (<480 ms), then ibutilide would be the recommended drug. Because of the patient’s dilated cardiomyopathy and CHF history it is unwise to recommend a class I antiarrhythmic drug (propafenone or flecainide). These drugs can cause further myocardial depression and worsening heart failure as well as increase the risk for a ventricular arrhythmia. Intravenous procainamide is safer than propafenone and flecainide, although structural heart disease increases the likelihood of negative hemodynamics and proarrhythmia.

• What should be done to maintain NSR?

Since this is the patient’s first event, and the event was well tolerated (normal blood pressure without ischemia), it is reasonable to prescribe no antiarrhythmic drug and wait to determine recurrence frequency. If the patient experiences additional events than maintenance therapy should be used. In this patient’s case there are three antarrhyhtmic choices, sotalol, dofetilide and amiodarone. Sotalol and dofetilide must be started while the patient is in the hospital and should be monitored for three days to rule out long QTc and torsades de pointes proarrhythmia. Amiodarone would be the best choice for this patient given their cardiomyopathy and CHF. Recent studies (CHF STAT and GESICA trials) suggest clinical benefit with amiodarone therapy in patients with CHF and ventricular premature contractions. If the patient is started on amiodarone therapy, there should be baseline liver, pulmonary and thyroid function test. It is recommended to start at a dose of 200 mg/day. Caution should be taken regarding concomitant digoxin and warfarin therapy due to the significant drug interaction potential increasing the effect of each.
• Should anticoagulation be prescribed?

It is recommended to use coumadin in this patient given that they also have valvular heart disease. If the patient can document that they have no additional AF using Event Heart Rhythm Recorders then it is usual clinical practice to discontinue coumadin after 6 months. The rule of thumb is to anticoagulate until proven otherwise. Hence the patient must prove their ability to maintain NSR.
Case I: Follow up

AL is successfully treated and is discharged home. Over the next year AL has had seven hospital admission for symptomatic atrial fibrillation. A echocardiogram two months ago documented an ejection fraction of 32%. She presents today with palpitations, severe shortness of breath and dizziness which is unrelieved by rest. On ECG she is noted to be in atrial fibrillation with a systolic pressure of 85 mmHg. It was decided to electively use DCC, which placed AL into sinus rhythm. On exam after cardioversion, AL was noted to be in less distress and more comfortable.

Medications on Admission:
- digoxin 0.125 mg QD
- metoprolol 50 mg QD
- Warfarin 5 mg QD
- captopril 25 mg TID
- sotalol 120 mg BID, also failed quinidine

What is your assessment of this patient's problems compared to last visit.

The patient has frequent bouts of AF even during pharmacologic maintenance therapy with either sotalol or quinidine. The patient also shows worsening tolerance of AF with poor hemodynamics (very low systolic pressure) with increase symptomatology. The patients EF is low which is consistent with her inability to tolerate AF. This could be exacerbated by her medication profile of beta blocker therapy concomitant with sotalol. This therapy is bound to make AF less tolerable because of the negative ionotropic actions. It is clear that rate control option would be difficult because of the poor tolerance of AF. Therefore, there must be continued effort to maintain NSR. The patient is on warfarin and should continue this therapy because of their multiple bouts of AF increase her risk of embolic stroke.
Case I: Follow up

What will be your therapeutic goal and which therapy will be used to achieve this goal?

The best option at this point is to try amiodarone therapy. The patient has a low EF ruling out the safe use of class I antiarrhythmic drugs. It is possible to try dofetilide, however, since sotalol failed which is mechanistically similar to dofetilide, it is likely that dofetilide will also fail. When starting amiodarone, both digoxin and warfarin doses should be decreased by 50% and monitored very closely.

State expected outcomes from therapy.

The goal of therapy is to maintain NSR. The patient should be monitored closely for recurring AF events. The patient should also be monitored for adverse drug effects and potential drug interactions. The goal is to reduce hospitalization due to AF and this in turn should prevent CHF exacerbations. AF is one of the most common causes of CHF exacerbations. Approximately 30% of CHF patients also have AF. These patients have a higher morbidity and mortality rate most likely due to AF inducing CHF and perhaps AF drugs inducing CHF. To prevent AF drugs from inducing CHF, only beta blockers, digoxin, amiodarone and/or dofetilide should be used. The Diamond trial showed that dofetilide decreases CHF hospitalizations in patients with class III CHF as well as dofetilide decreased AF. Hence, there is an association of decreasing AF and hospitalization. This was pivotal data for dofetilide approval.