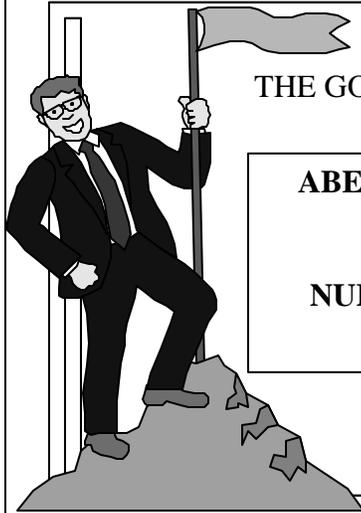


ATRIAL FIBRILLATION

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THE GOLFER

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OBJECTIVES:

- **Identify three factors that impact the ability to achieve or maintain normal sinus rhythm in patients with atrial fibrillation.**
- **Educate health professionals about the benefits and risks of long-term quinidine therapy in patients with atrial fibrillation, both with or without CHF.**
- **Recognize when amiodarone constitutes optimal therapy for selected patients with atrial fibrillation.**
- **Recognize when surgical therapies should be considered for the treatment of AF.**
- **Identify patients with AF who are candidates for aspirin therapy rather than warfarin therapy.**

ATRIAL FIBRILLATION

(BRIEF INFORMATION)

AF affects 2.2 million Americans each year. It's the most common sustained arrhythmia encountered in primary care practice. AF has a prevalence of 4% in the adult population. As the patient population continues to age, the prevalence of this arrhythmia rises as well, from <0.5% in patients 25-35 year of age to >5% in patients >69 years of age.

This disorder is implicated in several disease processes, including CHF, both as cause and result. An association with stroke is well established, with nonrheumatic heart disease and AF.

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AF is characterized by rapid and disorganized atrial electrical activity with a frequency ranging from 350 to 600 beats per minute. The ventricular response to this bombardment of stimuli depends on the nature of the intervening conduction system. The AV node blocks some of these impulses by its effective, albeit short, refractory period. Accordingly the ventricles react irregularly and may contract rapidly rates of 120 to 180 beats per minute, as seen in hearts with healthy conduction system, or slowly at rates of 30 to 60 beats per minute, as seen in complete heart block. With a rapid ventricular response, left ventricular end-diastolic volume is reduced because of the decreased filling time in diastole and the lack of the atrial “kick” to help fill the ventricle. This atrial contribution to ventricular filling increases

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with age, and its absence in AF, couple with comorbid conditions, increases mortality, especially in older patients.

CLINICAL PRESENTATION

Common symptoms of AF include palpitations, dyspnea, syncope, and angina. However the initial presentation in an out patient setting may often be asymptomatic or associated with vague symptoms such as fatigue or weakness. It is, therefore, important for the primary care physician to routinely evaluate cardiac rhythm by auscultation and pulse palpitation, symptoms. Once you establish an irregular rhythm and verify it with an electrocardiogram, take a careful history and perform a thorough physical examination to identify underlying causes.

Patient Presentation

CHIEF COMPLAINT

“I feel fine today but I got a little short of breath yesterday while I was playing golf”

Patient Presentation

HPI

John Lange is a 63 y/o man who presents to the cardiology clinic for a routine follow-up visit with the above complaint. He was diagnosed with AF 5 month ago and was hospitalized at that time for DCC. He was subsequently started on quinidine to prevent recurrence. His most recent serum quinidine level (one month ago) was 3.1 mcg/ml. The patient admits that **he does not always take his quinidine three times a day.**

PMH

MI (anterior wall), In January 1990

AF, as described above

CHF (EF 34%)

HTN, diagnosed in 1975

No hx of CVA, PE, PVD, or DM

Patient Presentation

FH

Father died suddenly at the age of 56; mother died at the age of 72 of unknown causes; only sibling is a brother (age 57) who is alive with hypertension

SH

Retired accountant; non-smoker; social alcohol use (6 to 12 oz. per month). Active; plays golf two three times a week.

MEDS

Quinidine gluconate (sustained-release tablets) 324 mg po Q8H

Warfarin 5 mg po QHS

Patient Presentation

Atenolol 25 mg po QD

Enalapril 15 mg po BID

Digoxin 0.125 mg po QD

Furosemide 20 mg po QD

Allergies - NKDA

ROS

SOB, as noted above. No palpitations, dizziness, lightheadedness, chest pain, or other complaints.

Patient Presentation

PE

GEN -The patient is a pleasant gentleman appearing to be his stated age in NAD.

VS - B/P **135/85**, P **90** (**irregular**), RR 16, T 37.C;

Wt 82 kg, ht 178 cm

HEENT

NCAT:PERRLA;EOMMI;

disc flat; TM intact

COR - **Irregular pulse**; no murmurs, rubs, or gallops

CHEST - **Bibasilar rales**

ABD -NT/ND; no hepatosplenomegaly

EXT - **1+ pitting edema bilaterally**

Neuro - **Intact**

Labs - Na 143 mEq/L

K 4.7 mEq/L

Patient Presentation

Chl. 104 mEq/L	PT/INR - 19.1 seconds/1.64
CO2 content 25 mEq/L	Echocardiogram (4 month ago)
BUN 16 mg/dl	Mild systolic dysfunction,
Serum Creatinine 1.1 mg/dl	hypokinesis of lateral wall, left atrium 47 mm
Hemoglobin 14.2 g/dl	ECG (1 month ago)
Hct 37.5%	NSR, rate 71, anterior infarct, QTc
Plt. 193,000/mm ³	410 ms
WBC 8700/mm ³	CXR (1 month ago) - lung clear.
Digoxin 0.9 ng/ml	Enlarged heart (cardiothoracic ratio 0.4)

Problem Identification

1. a. What etiologies for AF are possible in this patient?
 - MI
 - HTN
 - CHF
 - CAD
 - CARDIOMEGALY
- b. What factors impact on the ability of this patient's heart to maintain NSR?
 - Left atrium 47 mm (enlarged) - cause ineffective atrial contractions and rapid ventricular response. (Normal atrium should measure 3-5 mm).

Problem Identification

- CHF - is a response to cardiac dysfunction in which the heart cannot pump blood at a volume required to meet the body's needs.
 - AF Hx (for around 5 month ago) is a long time. Decrease CO because of ineffective atrial contractions and a rapid ventricular response.
- c. What signs and symptoms indicate worsening heart failure in this patient?
- SOB
 - Bibasilar rales
 - Pitting edema
 - Irregular pulse

Problem Identification

- d. What is the possible cause of the worsening heart failure?
- USE OF ATENOLOL - as negative inotropic agent decreases the velocity of myocardial contraction and the stroke volume. These agents also include procainamide, quinidine and propranolol.
 - The patient's status of having ventricular dysfunction has gotten worse. His impulse of the atrium does not exist.

Desired Outcome

2. What are the treatment goals for Chronic Atrial Fibrillation?

- The goal of treatment is a decrease in ventricular response.
- Restoration of sinus rhythm.
- Decrease the occurrence of strokes or thromboembolic events. (preventing the thromboembolic complication).
- Improve hemodynamics.
- Improve rate control and thereby decrease risk of a tachycardia-induced cardiomyopathy.
- Improve the patients symptoms and thereby their quality of life.

Desired Outcome

- With CHF we should have special precautions, because of the blood stasis in the noncontractile atria, Clots are formed inside of it that can result in systemic and cerebral embolization, especially at the time we are attempting to restore sinus rhythm as the atria start to contract again (risk is about 7%). So it is imperative that anticoagulant therapy be installed in patients with AF, especially those with underlying cardiac disease and those with other risk factor for stroke.

Therapeutic Alternatives

3. The Pharmacist involved in this case was not present during the discussion of potential therapeutic alternatives. Consequently, the following events occurred without pharmacy input.

CLINICAL COURSE

While in clinic, a 12 lead ECG was performed which confirmed AF with a ventricular rate of 95.

Clinical Course

The patient was hospitalized in a telemetry bed, and a serum quinidine level obtained. The medical team continued his outpatient medication regimen. A serum quinidine level on admission was 1.2 mcg/ml. After two days in the hospital, he converted to NSR, and his rales and edema resolved. The patient was subsequently discharged in NSR with no changes to his previous drug therapy.

Optimal Plan

4. What is your assessment of these interventions?

We can use quinidine therapy, to convert to NSR. But it is not recommended to use long time because it can cause other dysrhythmia such as VT and torsades de pointes. Its also toxic. (also medication such as amiodarone and sotalol have minimal proarrhythmic effects).

Assessment Parameters

5. How would you monitor efficacy and adverse effects of quinidine therapy in this patient?

- EKG continuously to determine prolonged PR or QRS segments, QT intervals: discontinue or reduce dose.
- Blood levels (therapeutic levels 2-6 mg/ml).
- B/P continuously for fluctuations.
- Cinchonism: tinnitus, headache, nausea, dizziness, fever, vertigo, tremor; may lead to hearing loss.
- Cardiac status: rate, rhythm, character, continuously.

Assessment Parameters

- Respiratory status: rate, rhythm, lung fields for rales;
- increased respiration, increase pulse; drug should be
- discontinued.

Patient Counseling

6. What important information about quinidine therapy would you provide to the patient ?

Purpose : Its restores you heart rate to NSR.

Patient information/Instruction:

Take exactly as directed, around the clock: do not take additional dose or discontinue without consulting prescriber.

Do not crush, chew, or break sustained release capsules.

Will need regular cardiac checkups and blood test while taking this medication.

Patient Counseling

You may experience dizziness, drowsiness or visual changes (use caution when driving or engaging in tasks requiring alertness until response to drug is known); abnormal taste, nausea or vomiting, or loss of appetite (small frequent mouth care, chewing gum, or sucking lozenges may help); headaches (prescriber may recommend mild analgesic); or diarrhea (exercise yogurt or boiled milk may help- if persistent consult prescriber)

Report chest pain, palpitation, or erratic heartbeat; difficult breathing or wheezing.

CNS changes (confusion, delirium, fever, consistent dizziness)

Patient Counseling

Skin rash; sense of fullness or ringing in ears; or changes in vision.

Follow - up case questions

1. If the patient could not tolerate quinidine therapy, what pharmacotherapeutic alternatives would you recommend ?

Newer type Ic (e.g., flecainide, propafenone) and type III (e.g. amiodarone, sotalol) antiarrhythmic agents may provide alternatives to quinidine.

Allow AF to continue and control Ventricular response with Digoxin, Beta Blockers and Calcium Channel.

Follow - up case questions

2. What non-pharmacological (i.e., surgical) therapies may be considered for treating Chronic Atrial Fibrillation ?

Because of the limitations of pharmacologic treatment of AF and also catheter ablation of the AV junction, electrophysiologists are now focusing increased attention on developing new treatment strategies including catheter ablation of the AF focus.

Some of the non- pharmacology's therapies are:

- **The Maze procedure** - DR. Swart is credited with being the first electrophysiologist to cure AF using a catheter - based procedure designed to replicate the surgical **MAZE** procedure, which is a well established technique to cure AF. The surgical **MAZE**

Follow - up case questions

Procedure involves excision of the right and left atrial appendages and creation of a number of linear lesions that encircle the pulmonary veins.

- **Catheter Ablation** - This procedure has been performed for more than 15 years and is very well established. Ablation of the AV node, with implantation of a pacemaker, is a very useful technique for patients with disabling and drug-refractory AF. It is in ventricular function related to amelioration of tachycardia - induced myopathy. It has been further proposed that subtotal injury to the AV node (modification of the AV node) can result in a slowing of the ventricular rate in AF without the need for pacemaker dependency. This technique is certainly feasible and

Follow - up case questions

Can work, but it is technically difficult to achieve an ideal result and many patients remain troubled by the irregularity of their heartbeat in spite of the fact that it is slower.

3. What is your assessment of this patient's antithrombotic therapy ?

As additional information:

The anticoagulation could be made with either oral warfarin or parenteral heparin, the goal being a 1.5 (INR 2-3) the prothrombin control value and twice the PTT control value, respectively. If restoration of sinus rhythm is desired the therapy should be started 3 weeks prior and maintained for a couple of weeks after procedure (either electrically or pharmacologically). Otherwise anticoagulant therapy should be maintained indefinitely.

Follow - up case questions

The INR can increase due to CHF. The goal is 2-3. Patient had 1.64. It should be monitored weekly and warfarin doses changed to maintain an INR of 2.0-3.0.