Atrial Fibrillation

Richard W. Sloan, MD, Nikitas J. Zervanos, MD, Richard L. Snyder, MD and Edward G. Zurad, MD
Lancaster, Pennsylvania

DR. NIKITAS J. ZERVANOS *(Director, Family Practice Residency Program)*: During the time from July 1, 1980, to September 30, 1983, atrial fibrillation was the seventh most common diagnosis seen in patients aged 65 or older in our Walter L. Aument Family Practice Center. Among admissions to Lancaster General Hospital during this period, atrial fibrillation or flutter was the 13th most common admission diagnosis. Since residents in this program encounter patients with atrial fibrillation often, and the evaluation and management of this disorder are rather complex, a review of this topic is appropriate and timely. I have asked Dr. Snyder to present the case of a patient who presented to our Family Practice Center about five years ago with new-onset atrial fibrillation.

DR. RICHARD L. SNYDER *(Third-year Family Practice Resident)*: The patient was a 54-year-old white male farmer who presented to the Family Practice Center in January 1980 complaining of low back pain. The physician examining the patient noted a rapid irregular pulse and obtained an electrocardiogram, which showed atrial fibrillation. The patient had noted some dyspnea on exertion over the last three weeks but was otherwise asymptomatic from a cardiovascular standpoint. This patient has been closely followed in the Family Practice Center since 1980. We will be discussing the sequence of historical and physical findings as well as diagnostic and therapeutic events.

DR. RICHARD W. SLOAN *(Associate Director, Family Practice Residency)*: If you were presented with this patient, what history would you consider pertinent?

DR. SNYDER: Obviously a thorough cardiac history should be obtained. This patient denied a past history of rheumatic heart disease, heart murmur, and myocardial infarction. He was quite active physically and denied any exertional chest discomfort. The duration of atrial fibrillation is important. This patient had no past electrocardiograms. Since he complained of some increased dyspnea on exertion of three weeks' duration, it is possible that the onset of atrial fibrillation was three weeks prior to his visit. Of course, there was no way to know for sure, but it was assumed his dysrhythmia was of recent onset.

The past medical history should also be reviewed for a history of chronic lung disease, diabetes mellitus, pulmonary embolus, or hyperthyroidism. This patient did have some findings of mild chronic obstructive pulmonary disease on his chest roentgenogram. However, he denied any sputum production, and his peak expiratory flow as measured on a Wright peak flow meter was 350 L per minute. There was no evidence of cor pulmonale. It would be important to take a medication history. Sympathomimetics, amphetamines, methylxanthines, and excessive thyroid hormone administration could cause atrial fibrillation in some patients. The review of systems should elucidate key symptoms involving the cardiac, pulmonary, and endocrine systems. This patient denied the following cardiac symptoms: chest pain, palpitations, orthopnea, syncope, and ankle swelling. The pulmonary review was negative for pleuritic chest pain, sputum production, asthma, and hemoptysis. There were no symptoms suggestive of hyperthyroidism. Finally, it would be

From the Family Practice Residency Program, Lancaster General Hospital, Lancaster, Pennsylvania. Requests for reprints should be addressed to Dr. Richard W. Sloan, PO Box 3555, Lancaster, PA 17603.

important to ask about past transient ischemic attacks, stroke, or other symptoms that would suggest an embolic event. The history for these disorders was also negative.

DR. SLOAN: It is important to remember that atrial fibrillation is not a definitive diagnosis, but rather a sign of an underlying pathophysiologic process. The most common causes of atrial fibrillation are left ventricular failure and mitral valve disease with resultant left atrial distension. Hypertension is not a risk factor for atrial fibrillation unless there is associated left ventricular hypertrophy or dilatation with increased left atrial filling pressures. Ischemic heart disease is a cause of atrial fibrillation only when it causes acute or chronic heart failure. Pulmonary embolus is an uncommon cause of atrial fibrillation. When reviewing the medications that the patient is taking, remember to ask about alcohol consumption. Atrial fibrillation has been reported to be a consequence of binge drinking in otherwise healthy people.[1,2] You mentioned that there were no symptoms suggestive of hyperthyroidism. It is important to note that atrial fibrillation may be the only sign of hyperthyroidism, especially in elderly patients.[3] Dr. Snyder, tell us what physical findings you would seek in this patient.

DR. SNYDER: This patient appeared to be in good health. He was in no obvious distress. The blood pressure was 114/68 mmHg and the pulse was 110 beats per minute. The most important part of the physical examination would be the cardiac examination. The cardiac impulse was not palpable. The rhythm was irregularly irregular. S_1 varied in intensity. There was no murmur audible. S_3 and S_4 were absent. I would look for signs of congestive heart failure. The chest was clear. There was no organomegaly or peripheral edema. The thyroid gland was not enlarged, nor were any nodules palpable.

DR. SLOAN: In addition to recording the patient's peripheral pulse rate, the apical rate should also be recorded. Patients with rapid atrial fibrillation often have a pulse deficit; that is, the apical rate is greater than the peripheral pulse rate. When the R-R interval is very short, the time for ventricular diastolic filling is limited, which results in an accentuation of the first heart sound resulting from the premature but rapid closure of the wide-open mitral valve.[4] During these short cycles the stroke volume is quite low and may not result in a palpable peripheral pulse. When the R-R interval is prolonged, allowing adequate time for ventricular filling, the intensity of the first heart sound is reduced and the stroke volume is increased, which explains your observation that S_1 varied in intensity. In most patients with untreated atrial fibrillation, the ventricular rate is usually between 120 and 180 beats per minute. Patients with ventricular rates of less than 120 beats per minute probably have some degree of intrinsic atrioventricular (AV) node dysfunction. You mentioned that no murmurs were audible, an important observation, since mitral valve disease is an important cause of atrial fibrillation. In patients with rapid atrial fibrillation, the murmur of mitral stenosis is extremely difficult to hear. The patient should be placed in the left lateral decubitus position. Using the bell portion of the stethoscope and listening over the apex, a diastolic rumble may be audible. The murmur is easier to hear during long cycles (prolonged R-R interval). The classic murmur of mitral stenosis is modified in patients with atrial fibrillation, because of the absence of the presystolic accentuation, which is caused by atrial contraction. Atrial contraction is also required to produce the fourth heart sound; therefore, the fourth heart sound is never present in a patient with atrial fibrillation.

DR. WILLIAM H. BACHMAN *(Associate Director, Family Practice Residency; Director, Walter L. Aument Family Practice Center)*: An important decision that needed to be made during this patient's first visit was whether to admit him to the hospital for evaluation and treatment. I would think that a patient in this age group with new-onset atrial fibrillation should be admitted to the hospital.

DR. SNYDER: Remember that this patient did not present with complaints referable to the cardiovascular system but rather an unrelated complaint, low back strain. In fact, the patient was surprised to learn that he had a rapid irregular pulse. One factor that may influence the decision to admit is an assessment of causative factors. Conditions that can cause atrial fibrillation are shown in Table 1. If the index of suspicion for a myocardial infarction, pulmonary embolus, or another acute insult is high, then admission to the hospital is certainly indicated. The only causative

factor that could be identified in this patient was mild chronic obstructive pulmonary disease.

DR. SLOAN: In reference to coronary artery disease, the rapid ventricular rate seen with atrial fibrillation provides a provocative test similar to a treadmill stress test. The electrocardiogram should be examined closely for ST segment changes characteristic of myocardial ischemia. If a patient presents with rapid atrial fibrillation and angina pectoris, it should be considered a medical emergency requiring aggressive treatment in a medical constant care unit. Another important indication for admission is significant hemodynamic compromise. Loss of the "atrial kick" in a patient with severely impaired left ventricular function may result in acute decompensation. This factor, coupled with an inadequate time for ventricular filling and reduced stroke volume, may result in congestive heart failure and hypotension. The patient we are presenting appeared to have no significant hemodynamic compromise, In addition, his ventricular rate was relatively slow for untreated atrial fibrillation. For these reasons, we decided to proceed with evaluation and treatment as an outpatient. Dr. Zurad, how would you proceed with the evaluation and treatment of this patient?

DR. EDWARD G. ZURAD *(Third-year Family Practice Resident)*: In addition to a rhythm strip, a 12-lead electrocardiogram should be performed. The typical findings of atrial fibrillation include the absence of P waves and an irregularly irregular R-R interval.[5] The electrocardiogram should be examined closely for ST-T wave changes. A chest roentgenogram should be obtained to determine heart size and to look for signs of congestive heart failure, pulmonary embolus, or chronic lung disease. The echocardiogram is a very useful diagnostic test and should be performed routinely as part of the initial workup. This study allows for the noninvasive measurement of chamber sizes. It is important to know the left atrial size. This measurement helps determine the patient's risk for systemic embolus and predicts the likelihood of successful cardioversion.[5,6] The echocardiogram is also helpful in examining the mitral valve, looking for evidence of mitral stenosis or mitral regurgitation. This patient's echocardiogram demonstrated a normal mitral valve and a slightly enlarged left atrium. Other diagnostic studies should be considered according to the suspected etiology. In the case we are discussing today, pulmonary function testing and an arterial blood gas might be useful in assessing the severity of this patient's lung disease. Thyroid indices should be obtained in all patients with atrial fibrillation. Radioimmunoassay of triiodothyronine (T_3 RIA) is the most sensitive test for diagnosing hyperthyroidism. Routine blood work should also be obtained, including a complete blood count, blood glucose, serum electrolytes, and a serum creatinine. A blood alcohol level would be useful to confirm inebriation in certain patients. If anticoagulation is anticipated, a prothrombin time, partial thromboplastin time, and platelet count should be obtained.

TABLE 1. CONDITIONS THAT CAN CAUSE ATRIAL FIBRILLATION

Etiologic Category	Disease State or Drug
Cardiovascular	Mitral valve disease* Congestive cardiomyopathy Coronary artery disease** Myocardial infarction** Hypertension*** Pericarditis Cardiac surgery
Pulmonary	Pulmonary embolus† Pneumonia† Chronic obstructive pulmonary disease (cor pulmonale)
Endocrine	Hyperthyroidism Pheochromocytoma
Drugs	Alcohol Methylxanthines Sympathomimetics Amphetamines

**Either mitral stenosis or mitral regurgitation*
***Ischemic heart disease is a cause of atrial fibrillation only when it produces increased left ventricular and left atrial filling pressures*
****Hypertension is a risk factor for atrial fibrillation only when it is associated with cardiomegaly, electrocardiographic evidence of left ventricular hypertrophy, or congestive heart failure*
†Pulmonary embolus and pneumonia are uncommon causes of atrial fibrillation

DR. SLOAN: This brings us to a consideration of treatment options. What are the therapeutic

Continued on page 30

Continued from page 27

TABLE 2. DRUG THERAPY IN ATRIAL FIBRILLATION

Rate Control	Anticoagulation	Cardioversion
Digoxin β-Blocker Verapamil	Warfarin Heparin	Quinidine Disopyramide Amiodarone*

*Investigational drug

goals in a patient with atrial fibrillation? The most important and first goal to consider is control of the ventricular rate. Other important therapeutic questions are (1) is the patient a candidate for medical or electrical cardioversion, and (2) should the patient be given anticoagulants? Currently, there are three important drugs that could be considered to control the ventricular rate (Table 2). These include digoxin, verapamil, or a β-adrenergic blocking drug. Dr. Zurad, which drug would you select for this particular patient?

DR. ZURAD: Digoxin (Lanoxin) would be the drug of choice in most clinical situations. Since we have committed ourselves to outpatient treatment in a well-compensated patient with a relatively slow ventricular response, we have time to titrate the dose as required. This patient was given digoxin, 0.50 mg by mouth followed in six hours by a second dose of 0.25 mg; thus, his loading dose was 0.75 mg. He was then told to take a 0.25 mg tablet daily. Alternatively, we could have used digoxin in solution capsules (Lanoxicaps). This preparation is more completely absorbed than digoxin (Lanoxin) tablets, but the onset of action would have been about the same, and Lanoxicaps are considerably more expensive.

DR. ALAN S. PETERSON *(Associate Director, Family Practice Residency Program)*: I agree with your initial choice of digoxin in this patient. Prior to the availability of β-blockers and verapamil, digoxin was used exclusively for rate control in patients with atrial fibrillation. Compared with some β-blockers and verapamil, digoxin is inexpensive and can be taken once daily. Digoxin is certainly the drug of choice in patients with a history of congestive heart failure or congestive cardiomyopathy and atrial fibrillation. The only problem with digoxin is that it may take several days to several weeks to titrate the dose and adequately control the rate. In a hospital setting where rapid control of the ventricular rate is desired, intravenous verapamil may be considered.[7,8]

DR. SLOAN: It is important to remember that digoxin's dose-response curve in atrial fibrillation is considerably different from the dose-response curve for congestive heart failure. In patients with congestive heart failure, the desired therapeutic response is increased contractility with an associated increase in cardiac output. The optimum total body digoxin pool to produce this response is approximately 10 μg/kg (lean body mass). In treating atrial fibrillation, the desired therapeutic response is a reduction in ventricular rate. A higher total body digoxin pool (12 to 15 μg/kg of lean mass) is required to accomplish this objective.[5,9] Digoxin is bound to myocardial receptors at a relatively slow rate. Serum digoxin concentrations obtained 8 to 12 hours after an oral dose correspond the best with drug concentrations in the myocardium. However, serum digoxin concentrations are of little value in adjusting the dose of digoxin in patients with atrial fibrillation. In these patients, the ventricular rate is a much better predictor of therapeutic effect than the serum drug concentration. Some clinicians advocate ignoring the serum digoxin concentration altogether and titrating the digoxin dose upward until the rate is controlled. In some patients, this may require a loading dose of 2 to 4 mg.[4]

When using large loading doses of digoxin to control the ventricular rate, several precautions must be kept in mind. First, if the patient spontaneously converts to normal sinus rhythm, what appeared to be the right amount of digoxin to control the rate may now be excessive. Signs of atrioventricular block may be noted on the electrocardiogram. Second, large doses of digoxin may necessitate postponing electrical cardioversion until the digoxin level is lowered. Electrical cardioversion in a patient with excessive myocardial digoxin concentrations may result in malignant ventricular dysrhythmias. If quinidine therapy is instituted 24 to 48 hours prior to electrical cardioversion, the serum digoxin concentration will be further elevated. It is important to be able to recognize the electrocardiographic signs of digi-

talis excess in patients with atrial fibrillation.[10] The most important finding is regularization of the ventricular rate (Table 3). Any patient with a history of chronic atrial fibrillation who demonstrates a regular pulse needs an electrocardiogram to determine whether the patient has converted to normal sinus rhythm or is digitalis toxic with a junctional escape rhythm.

DR. ZURAD: After receiving a loading dose of digoxin of 0.75 mg followed by a maintenance dose of 0.25 mg for two weeks, the patient still had an apical rate of 120 beats per minute. Since this patient was considered a candidate for cardioversion, it was elected not to increase the digoxin dose but rather to add a second drug to control the ventricular rate. Metoprolol was selected.

DR. ZERVANOS: Why did you choose metoprolol (Lopressor)?

DR. ZURAD: When this patient was initially seen at the Family Practice Center, oral verapamil was not available. Therefore, a β-adrenergic blocking drug was selected as the second drug. The cardioselective β-blocker, metoprolol (Lopressor), was chosen because the patient had mild chronic lung disease. Incidentally, there was no history of asthma, and no wheezing was audible in the chest. The starting dose of metoprolol was 25 mg twice daily, increased to 50 mg twice daily several days later. On this regimen the patient's ventricular rate was controlled at 85 beats per minutes.

DR. SLOAN: In a patient with asthma or significant airway obstruction, the best second drug to control the ventricular rate would be verapamil. It is important to note that verapamil will elevate the serum digoxin concentration significantly, and that the digoxin dose is generally reduced when verapamil is added.

DR. ELLEN M. GEORGE *(First-year Family Practice Resident)*: One of the more difficult issues for me is deciding which patients with atrial fibrillation should be anticoagulated. Why did you elect to anticoagulate this patient?

DR. SNYDER: This patient was a relatively young man who was accustomed to hard physical work on the farm. Although we did not know the exact duration of his atrial fibrillation, we thought that it was of short duration. In addition, his echocardiogram demonstrated that his left atrium was only slightly enlarged at 4.3 cm. For these reasons we felt he was a candidate for cardioversion. Since the risk of systemic emboli is increased during cardioversion, especially in patients with sustained atrial fibrillation and left atrial enlargement, an early decision was made to proceed with anticoagulation. When cardioversion is elective, the patient is generally anticoagulated for two to four weeks before medical or electrical cardioversion is attempted. If cardioversion is successful, anticoagulation should be continued for one or two additional weeks and then discontinued.

TABLE 3. ELECTROCARDIOGRAPHIC FINDINGS OF DIGITALIS TOXICITY IN A PATIENT WITH ATRIAL FIBRILLATION*

1. Ventricular rate less than 75 beats per minute
2. Junctional escape beats that occur at regular intervals at a rate of 35-50 beats per minute
3. Complete entrance block with a slow regular junctional escape rhythm
4. Accelerated regular junctional rhythm

**Listed in order of occurrence with increasing digoxin doses*

DR. GEORGE: What about a patient with chronic atrial fibrillation who is not a candidate for cardioversion? Should these patients be anticoagulated?

DR. SLOAN: This presents a more difficult question. The Framingham data[11,12] revealed a rather poor prognosis in patients with chronic atrial fibrillation, even in the absence of apparent organic heart disease. During a follow-up period of 22 years, the incidence of embolization was 40 percent. However, whether epidemiologic methods adequately excluded patients with organic heart disease from this group is debatable. The incidence of stroke in patients with atrial fibrillation has been estimated to be six times greater than that seen in an age-matched control group without the arrhythmia.[13-15] The mortality in a group with atrial fibrillation is also higher.[16] Some studies suggest that nearly 20 percent of all strokes occur in patients with atrial fibrillation. Nevertheless, some elderly patients have chronic atrial fibrillation without any evidence of organic heart disease. Elderly men with otherwise normal hearts and chronic atrial fibrillation with a relatively slow ventricular response have been called "lone atrial

fibrillators."[4,17] Most of these patients are asymptomatic, have a favorable prognosis, and require minimal treatment. The risk of chronic anticoagulation can be considerable, especially in elderly patients. The decision to anticoagulate must be made individually for each patient after carefully considering the risk of systemic emboli vs the risk of anticoagulation. Patients with mitral stenosis or idiopathic hypertrophic subaortic stenosis and an enlarged left atrium are at the highest risk for systemic emboli and are generally anticoagulated. Patients with a normal mitral valve but a large left atrium or congestive heart failure are at moderate risk for systemic emboli. Those with a low risk of systemic emboli include patients with no detectable organic heart disease, a normal mitral valve, a normal-sized left atrium, and a normal heart size.

DR. ZERVANOS: You mentioned why you felt this patient was a candidate for cardioversion. Since his left atrium was enlarged at 4.3 cm, is it not true that his chances for successful cardioversion are reduced?

DR. SNYDER: Yes. The success rate for either medical or electrical cardioversion decreases with increasing left atrial size. Also, the longer the patient has been in atrial fibrillation, the less the chance of successful cardioversion. Since this patient had only a slightly increased left atrial size and probably short-term atrial fibrillation, we felt cardioversion was worth a try.

DR. ROBERT W. NIEGISCH *(First-year Family Practice Resident)*: Since the patient was asymptomatic from a cardiovascular standpoint, why not just let him remain in atrial fibrillation and control his rate?

DR. SLOAN: There are a number of disadvantages that a patient accrues by remaining in sustained atrial fibrillation. The loss of the "atrial kick" results in a 15 percent reduction in cardiac output. This amount may not be a significant clinical problem in a patient with good left ventricular function; however, in a patient with severe left ventricular dysfunction, the atrial component of the cardiac output may be critical. A second disadvantage of atrial fibrillation is the loss of normal heart rate modulation in relation to exercise. Most patients with atrial fibrillation experience an exag-

Continued on page 34

Continued from page 32

gerated heart rate response to exercise. Moreover, recovery of the resting pulse rate is delayed. Finally, atrial fibrillation increases the patient's risk of systemic emboli with its associated complications.

DR. ZURAD: After consulting with the cardiologists, we elected to institute quinidine sulfate therapy, 200 mg every six hours four days before the patient was to be admitted for elective cardioversion. Remembering the quinidine-digoxin interaction,[18] we decreased the dose of digoxin to 0.25 mg alternating with 0.125 mg daily. The day before admission we checked a serum digoxin concentration, which was 0.94 μg/mL; a serum potassium, which was 4.1 mEq/L; and a prothrombin time, which was 23.1 seconds (control 12.1 seconds). He was admitted to the hospital the following day and underwent successful cardioversion. During the subsequent 24 to 48 hours numerous premature atrial contractions were noted. A serum quinidine concentration was subtherapeutic at 1.6 μg/mL, and his quinidine dose was increased to 300 mg every six hours. He was discharged from the hospital the following day.

DR. PETERSON: Why didn't you check a quinidine level prior to admission? You would have found it to be subtherapeutic and could have optimized the quinidine dose prior to cardioversion. Who knows, perhaps he would have converted medically.

DR. SLOAN: That certainly would have been an acceptable approach. Since this patient required 360 watt-seconds to convert him electrically, I doubt whether he would have converted medically.

DR. ZURAD: Several weeks after discharge from the hospital, the patient remained in normal sinus rhythm, and his warfarin and metoprolol were discontinued. Two years later he remained in normal sinus rhythm and expressed an interest in stopping his digoxin and quinidine. His digoxin was discontinued in April of 1982, and two months later his quinidine was discontinued. In August 1982 he was off all cardiac medications and felt well.

DR. GEORGE: How long should quinidine therapy be continued after successful cardioversion?

DR. SLOAN: Unfortunately, there are no well-established guidelines to answer that question. If the atrial fibrillation was caused by an acute event such as a myocardial infarction, cardiac surgery, pulmonary embolus, or pericarditis, then short-term therapy (three to six months) would be sufficient. However, if the precipitating causes are still present or slowly progressive, then long-term therapy would be required. The only causative factor that we could identify in this patient was chronic lung disease, albeit mild. If his lung disease was the cause of his atrial fibrillation, then we would expect him to be at high risk for recurrence.

DR. ZURAD: The patient again presented to the Family Practice Center in late November of 1982 complaining of dyspnea on exertion and fatigue. His pulse was noted to be 100 beats per minute and frequent irregular beats were noted. A Holter monitor was obtained, which demonstrated frequent episodes of atrial fibrillation and atrial flutter with 2:1 block. Ventricular rates up to 180 beats per minute were noted during sustained episodes of supraventricular tachycardia, which lasted as long as 25 minutes. The patient was started on a maintenance dose of digoxin 0.25 mg/d and quinidine gluconate 325 mg every eight hours.

DR. NIEGISCH: Should a Holter monitor have been obtained one to two months after the quinidine was discontinued? Perhaps we would have discovered earlier that the patient needed continual treatment before he became symptomatic.

DR. SLOAN: In retrospect that would have been a good idea.

DR. GEORGE: Quinidine therapy was started before the patient was fully digitalized. Isn't it true that quinidine can increase the ventricular rate in nondigitalized patients with atrial fibrillation?

DR. SLOAN: Yes. The paradoxical increase in heart rate is related to two pharmacologic properties of quinidine. First, quinidine has a vagolytic effect on the atrioventricular node, which allows the passage of more atrial impulses. Second, quinidine has a stabilizing effect on the atrium and reduces the number of atrial impulses that are bombarding the AV node. Normally, the number of atrial impulses is 300 per minute or greater. The

Continued on page 36

Continued from page 34

AV node cannot conduct 300 impulses per minute and, in fact, is refractory when many impulses enter the nodal tissue. Thus, many more impulses enter the AV node than exit (concealed conduction). Quinidine decreases the number of atrial impulses, which reduces the percentage of time that the AV node is refractory and allows the conduction of an increased proportion of impulses (reduces concealed conduction).

DR. ZURAD: A repeat Holter monitor was obtained one month later, which again showed frequent runs of atrial fibrillation with rates up to 210 beats per minute. The patient was seen by a cardiology consultant, who recommended stopping the quinidine and treating the patient with verapamil, 120 mg every 8 hours, and digoxin 0.125 mg/d. The consultant also recommended rechecking the thyroid indices. Repeat thyroid indices were normal. The patient did well on the new medical regimen and was not seen again until April of 1983. A repeat Holter monitor was obtained at this time, which showed no supraventricular tachycardia but there were frequent unifocal premature ventricular contractions averaging 21 per 1,000 beats. A serum digoxin concentration was obtained, which was 0.35 μg/mL. No additional action was taken. Three months later the patient was reevaluated in the Family Practice Center. He complained of mild shortness of breath but denied chest pain, palpitations, or syncope. A rhythm strip demonstrated premature ventricular contractions every third or fourth beat. The PR interval was slightly prolonged at 0.22 seconds; the QT interval was normal. Quinidine therapy was reinstituted and titrated upward to a dose of 400 mg every six hours. The serum quinidine concentration on this dose was 2.6 μg/mL. The patient's premature ventricular contractions disappeared on this regimen. He did not develop diarrhea and felt well. His rhythm strip demonstrated no significant prolongation of the QT interval. A year later he continues on a medical regimen of digoxin 0.125 mg/d, verapamil 120 mg every 8 hours, and quinidine 400 mg every 6 hours. He continues to work full-time on the farm and feels well. He has refused formal pulmonary function testing, but his peak expiratory flow remains stable at 350 L per minute.

DR. ZERVANOS: This certainly has been an informative and practical discussion. The continuity that we have established with this patient in the Family Health Center over the last five years has contributed greatly to the successful management of a complex cardiovascular problem. The long-term follow-up has also increased my understanding of the evaluation, treatment, and natural history of atrial fibrillation. I would like to thank all of you for an excellent presentation.

References

1. Thornton JR: Atrial fibrillation in healthy non-alcoholic people after an alcoholic binge. Am J Cardiol 1984; 54:755-761
2. Spodick DH: The role of alcohol in new-onset atrial fibrillation. Arch Intern Med 1984; 144:862-863
3. Cobler JL, Williams ME, Greenland P: Thyrotoxicosis in institutionalized elderly patients with atrial fibrillation. Arch Intern Med 1984; 144:1758-1760
4. Morris DC, Hurst JW: Atrial fibrillation. Curr Probl Cardiol 1980; 5:1-51
5. Sloan RW: Atrial fibrillation. Am Fam Physician 1982; 25:165-171
6. Pabst P, Goldschlager N, Selzer A: Left atrial size and atrial fibrillation in mitral stenosis. Circulation 1973; 48:1282-1287
7. Antman EM, Stone PH, Muller JE, Braunwald E: Calcium channel blocking agents in the treatment of cardiovascular disorders (two parts). Ann Intern Med 1980; 93:875-885, 886-904
8. Hwang MH, Danoviz J, Pacold I, et al: Double-blind crossover randomized trial of intravenously administered verapamil. Its use for atrial fibrillation and flutter following open heart surgery. Arch Intern Med 1984; 144:491-494
9. Willoughby ML, Dunlap DB: The treatment of atrial fibrillation and flutter: A review. Heart Lung 1984; 13:578-584
10. Kastor JA, Yurchak PM: Recognition of digitalis intoxication in the presence of atrial fibrillation. Ann Intern Med 1967; 67:1045-1051
11. Kannel WB, Abbott RD, Savage DD, et al: Epidemiologic features of chronic atrial fibrillation. The Framingham study. N Engl J Med 1982; 306:1018-1021
12. Selzer A: Atrial fibrillation revisited. N Engl J Med 1982; 306:1044-1045
13. Sherman DG, Goldman L, Whiting RB, et al: Thromboembolism in patients with atrial fibrillation. Arch Neurol 1984; 41:708-710
14. Moss AJ: Atrial fibrillation and cerebral embolism. Arch Neurol 1984; 41:707
15. Kelley RE, Berger JR, Alter M, et al: Cerebral ischemia and atrial fibrillation: Prospective study. Neurology 1984; 34:1285-1291
16. Gajewski J, Singer RB: Mortality in an insured population with atrial fibrillation. JAMA 1981; 245:1540-1544
17. Benditt DG, Benson DW, Dunnigan A, et al: Atrial flutter, atrial fibrillation, and other primary atrial tachycardias. Med Clin North Am 1984; 68:895-918
18. Hager WD, Fenster P, Mayersohn M, et al: Digoxin-quinidine interaction. Pharmacokinetic evaluation. N Engl J Med 1979; 300:1238-1241