

Workshop

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CASE I

Dr. Dickinson: How would you manage this patient?

Dr. Dreifus: I do not believe that there is much to discuss in the first lead of ventricular fibrillation, and I would probably defibrillate the heart. The second one, however, reveals anteroseptal infarction. The exact age is not clear, but it is probably recent. The P-R interval appears to be normal; however, there is left axis deviation with right bundle branch block. Thus, I believe that this is sinus rhythm with bifascicular block and an anteroseptal wall infarction.

Dr. Baird: What are the criteria for the insertion of temporary pacemakers during an acute myocardial infarction?

Dr. Dreifus: The prognosis in patients with acute infarction complicated by bifascicular or trifascicular block is poor. The mortality may approach 60 or 70%, whether or not we utilize pacemakers. There is considerable work to show that patients who have two fascicles blocked with an acute process may eventually involve the third fascicle, and high grade block develops rapidly. I do not know whether I would put one in this patient unless there is associated first degree block. This can be either intranodal or block in the third fascicle. If it behaved like a basic Wenckebach, I would assume that it was in the A-V junction, but if the QRS suddenly dropped out, I would believe that the block is in the distal portion of the third fascicle or the posterior division. I would certainly put in the temporary pacemaker under these circumstances. However, I do not think I would recommend insertion of pacemakers in patients with mono- or bifascicular block alone in the electrocardiogram, even though two fascicles are blocked.

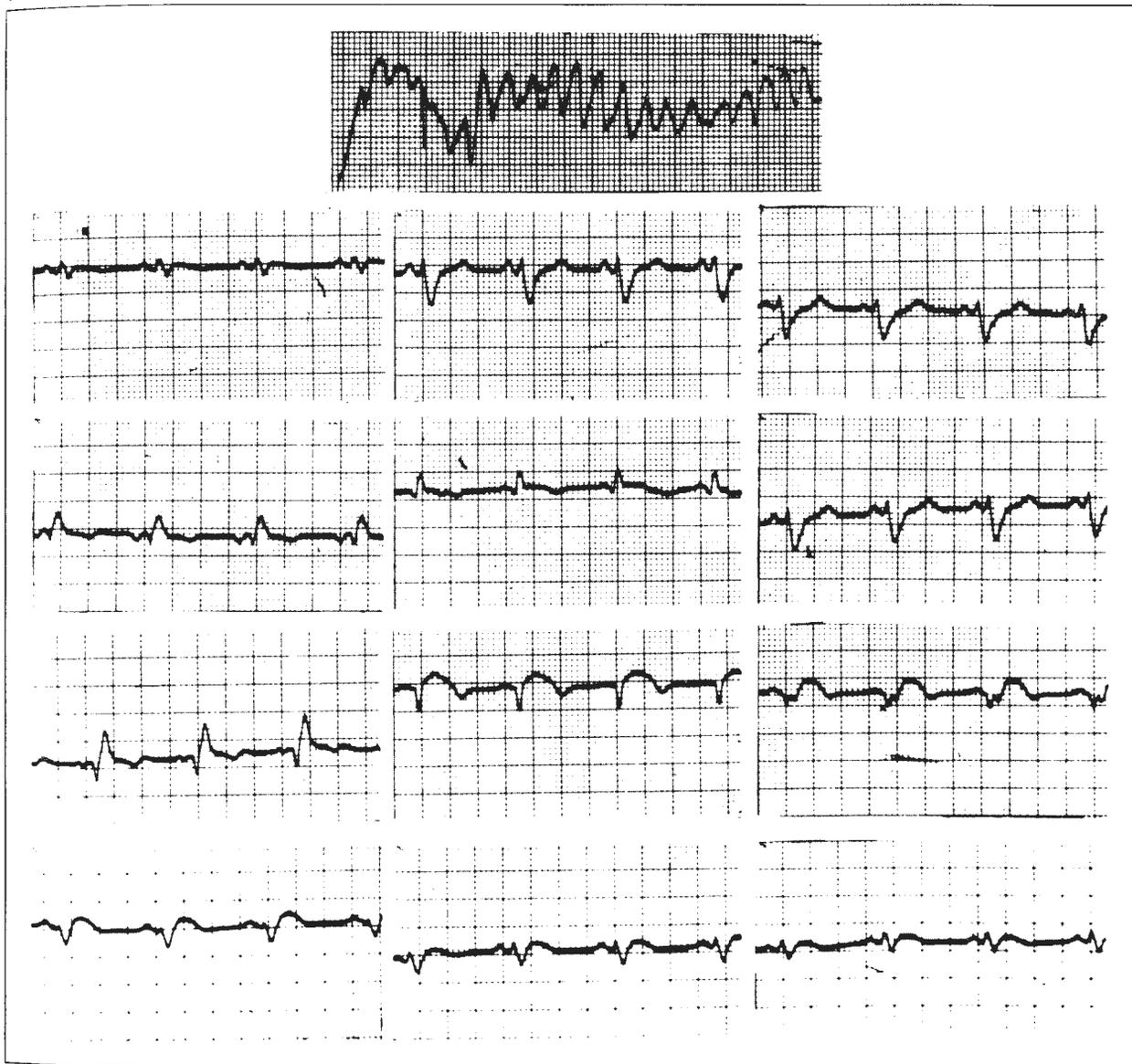
Dr. Dickinson: Are there any other comments regarding this?

Dr. Surawicz: I would like to make a comment. This kind of discussion always breaks down somewhat because we have two groups of people who manage such a problem, the university or teaching hospital with a house staff and the private hospital without house staff, which is quite different. The physician in a small private hospital knows that he has an hour's time and that later he will be busy. Aware of this, he will place in a pacemaker earlier, even though it may not be necessary. That is the situation frequently found in a small private hospital. To open the discussion in this case, I would not recommend temporary pacemaker insertion in a patient with a normal P-R interval, right bundle branch block, and left anterior hemi-block.

Dr. Bigger: At our hospital, Macken and Stock reviewed a series of cases of acute myocardial infarction with anteroseptal infarction and right bundle branch block. Nearly half of these cases developed sudden complete heart block or high degree A-V block without warning or Wenckebach periods.

Dr. Scherlag: I can report on studies that were done in a large series by Dr. Narula and recently published in the *American Journal of Medicine*. The patients with right bundle and left axis deviation, or what people term left anterior hemi-block, showed a 70% incidence of damage in all the "fascicles" of bundle branches, that is, the H-V times were prolonged, indicating bilateral bundle branch block. Twenty-eight to 30% showed that this was truly bifascicular, that is, the posterior "fascicle" was probably intact, and the H-V time was normal. The statistics are identical for both the infarcted as well as the noninfarcted.

Dr. Baird: I think it is interesting to note that the acute development of right bundle branch block during acute myocardial infarction has a mortality

**CASE 1:**

This 56-year-old white male painter was in excellent health prior to the development of substernal pain, diaphoresis two days prior to admission. During the ER evaluation, the patient had ventricular fibrillation and asystole. After defibrillation, a complete electrocardiogram demonstrated the following.

What would you recommend?

of close to 50%; therefore, I am sure Dr. Bigger and I, although we would insert a pacemaker catheter in this situation, realize the prognosis is extremely poor. It is possible that in the future, studies such as selective coronary arteriography with the consideration of emergency aorto-coronary surgery would be a more appropriate approach than the sole management of heart block with the insertion of a standby catheter.

Dr. Scherlag: I would like to make another comment. I agree that in patients with acute infarction and bundle branch block, pacemaker therapy does not appear to aid in terms of survival. However, we have been involved in studies with Dr. Clyde Schoenfeld in the intensive care unit utilizing His bundle recordings. Acute inferior myocardial infarction with Wenckebach phenomena is a situation in which most people do not recommend pace-

maker catheter insertion, but five of our patients showing Wenckebach cycles have developed higher degrees of block. In a given patient I think that Dr. Bigger was quite correct since these patients may show a period of nonconduction, and the pacemaker is the difference between life and death.

Dr. Bigger: In these people with complications such as hypotension, congestive failure, pulmonary edema, or emboli, a period of asystole may be catastrophic. I think that an individual who has borderline compensation of pump failure because one-third of his ventricle is necrotic or nonfunctioning would not survive a minute or two of asystole.

Dr. Dickinson: The audience might be interested in your recommendations concerning types of catheters and the use of portable fluoroscopy, Dr. Bigger.

Dr. Bigger: We often utilize No. 5 Cordis® transvenous bipolar pacemaker catheters or the semi-floating USCI® catheter. We prefer bipolar pacemaker catheters and insert them using portable fluoroscopy in our intensive care unit. Up until two and one-half years ago we utilized a large number of the Davis and Geck floatable platinum probes with a teflon coating made popular by Drs. Kimball and Killip of New York Hospital. We used several hundred of these catheters for various reasons and found them to be satisfactory. At present we use bipolar catheters that have good characteristics for torque manipulation.

Dr. Dickinson: Do you use the brachial or femoral vein?

Dr. Bigger: We have used the external jugular, subclavian, brachial, and more recently, the femoral vein. Probably the most convenient place to insert the catheter, as far as the patient is concerned, is in the external jugular system because both the patient's arms and legs are left free. A cut down on the jugular vein may be difficult for inexperienced personnel because it is friable.

Dr. Dreifus: Dr. Dickinson, may I make one more remark before you leave the pacemaker discussion? We have been using the Swan-Ganz floating balloon catheter and have found it successful in these situations. I think it is an alternative for fluoroscopy, particularly if you want to insert one rapidly. It may be placed through a needle percutaneously. If you need to put in one on a more permanent basis, for example, a week to ten days, you may transfer the patient to a fluoroscopy room and replace it with a stiff catheter. I would recommend that those

of you who cover coronary care units develop some facility in using the Swan-Ganz catheters.

Dr. Baird: Dr. Hoffman, what is the mechanism of ventricular fibrillation observed after temporary occlusion of the coronary artery in the experimental animal?

Dr. Hoffman: I think that I have some idea what the mechanism would be in an experimental animal, and it is very straightforward. If you occlude a branch of the coronary artery and the ventricle does not fibrillate during ischemia—let us say you are fairly far down the anterior descending artery—you leave the artery occluded long enough for cells normally nourished by the vessel to become ischemic and lose a good deal of potassium. Then when you release the occlusion you suddenly reperfuse this bed and move a fairly large amount of potassium out of the ischemic area to the adjacent areas of the myocardium. I think for the most part the so-called “release fibrillation” results from the flushing out from the ischemic area of potassium, lactic acid, and everything else that comes out suddenly from ischemic cells.

Dr. Scherlag: I certainly agree with Dr. Hoffman and would like to ask whether the arrhythmia is on the basis of automaticity or a reentry phenomena. We have performed studies utilizing the Sidney Harris preparation with anterior descending artery tie-offs and reperfusion after several hours. The resultant arrhythmias appear to be due to enhanced automaticity.

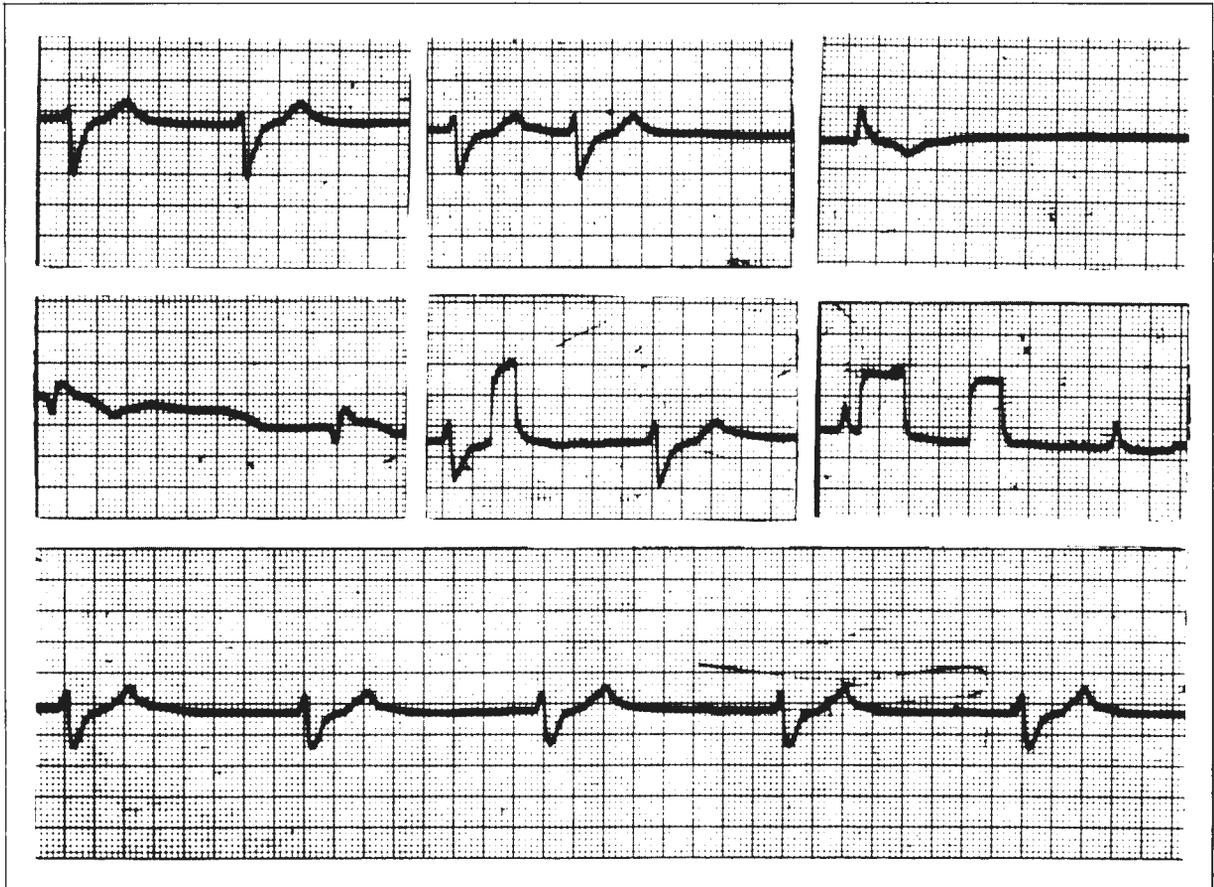
Dr. Bigger: I wonder if you would be more specific about the term automaticity in this situation. Do you mean that the arrhythmias do not stop when you stimulate the vagus? Even a reentrant arrhythmia might continue when you stop the atrium.

Dr. Scherlag: I think it is a good point, and it is difficult to differentiate between reentry and automaticity.

CASE II

Dr. Dickinson: Dr. Surawicz, we would like your interpretation of this and any remarks regarding etiology and treatment.

Dr. Surawicz: I presume that this patient has severe hyperkalemia with the serum potassium above 8 meq/L because of the absence of P waves. The presence of chest pain and syncope raises the possibility of fibrillation and a diaphragmic myocardial infarction with peri-infarction block. I would probably get some clue from the electrocardiogram

**CASE II:**

This 67-year-old white male with arteriosclerotic heart disease, angina pectoris, and mild chronic renal disease developed chest pain and syncope associated with the following electrocardiogram.

How would you manage this patient?

preceding this episode, but if this diagnosis of hyperkalemia is correct, then I would treat it with glucose, insulin, and bicarbonate.

Dr. Dickinson: The potassium level was 9 mg/L. Would you like to be more specific as to how you would use the dextrose and insulin?

Dr. Surawicz: In a case like this, we can give 50% glucose and 1 unit of insulin per 2 g of glucose during a period of electrocardiographic monitoring. I would then give 1–2 ampoules of bicarbonate and see what is happening.

Dr. Hoffman: I agree with Dr. Surawicz. In most mammalian hearts, including the human heart, the atria seem to be more sensitive to hyperkalemia than the ventricles. I do not know why the atria are more sensitive to the potassium than the ventricles and the ventricular conduction system. Perhaps Dr. Surawicz can tell us.

Dr. Surawicz: No, but the question is, why does the rhythm remain regular? Is it an escape pacemaker or is it from the sinus node?

Dr. Hoffman: If I had not read a recent paper by Dr. Fred Pick, I might have said what you wanted. I think that in many instances, if you have a ventricular rhythm of this sort in a patient with hyperkalemia, it may be a sinus rhythm where there is sinoventricular conduction and sinoatrial block. Dr. Pick is a very careful investigator, and he has not been able to reproduce some of our studies. Thus, I have been hedging on it for awhile. To come back to the question of other electrolytes, I imagine that one might wonder about changes in the level of calcium. As the serum calcium is elevated, the only thing this will do to the heart is to antagonize the effects of high potassium. For any given level of potassium, if the serum calcium

is high, the heart is less sensitive to the depolarizing depressant effects of potassium. If the serum calcium is low, the heart will be more sensitive to the same level of serum potassium. Thus, there is an antagonism, but one can demonstrate this experimentally very easily. However, it does not assume much importance in the usual patient.

Dr. Bigger: Why is the patient hyperkalemic?

Dr. Baird: It was not clear to the clinician why this patient who had had chronic renal disease suddenly developed such a problem. It is interesting to note that a temporary pacemaker catheter was inserted as the initial therapy since hyperkalemia was not recognized until later.

Dr. Hoffman: I wonder if I could ask a question reiterating a comment from the audience concerning the general question of the effect of serum potassium level on the uptake of the digitalis by the heart. If the serum potassium is elevated, there is evidence that the uptake of digitalis by the heart is retarded, and therefore, digitalization might be less rapid and less complete. I wonder if Dr. Bigger or someone else could help my vague memory concerning variation among the digitalis compounds.

Dr. Bigger: The first part, I think, has been shown in experimental animals; if the hyperkalemia precedes the administration of digitalis, then its uptake in the myocardium is retarded. Also, I think that it is correct that there is a difference between compounds; however, I am not certain of this.

Dr. Dreifus: If you add magnesium, you can go even farther with digitalis, but this is very impractical from the clinical standpoint.

Dr. Hoffman: It must be remembered that hyperkalemia can result in asystole. If you attempt to overcome the effects of hyperkalemia with calcium, it must be done very carefully since you may develop a completely asystolic heart without atrial activity as well as no ventricular activity. I merely want to express some caution in general; although when interpreting physiologic principles one can reverse some of the effects of high potassium by giving calcium, it is not a completely innocuous procedure.

CASE III

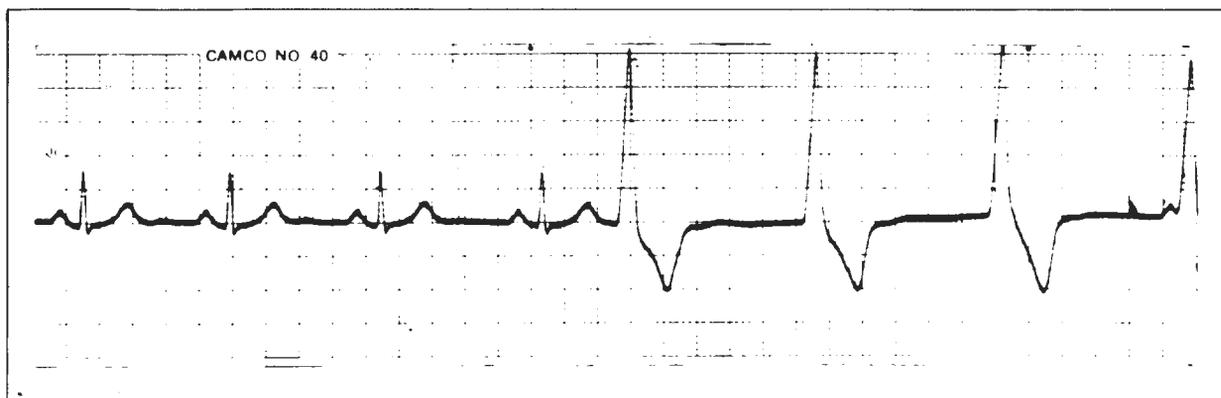
Dr. Dickinson: Dr. Bigger, what is your interpretation of this electrocardiogram?

Dr. Bigger: From left to right, four complexes of normal sinus rhythms are seen. The problem arises with the fifth beat, a tall wide QRS complex with the T wave in the opposite direction. It is premature with a shorter cycle length than that in normal sinus rhythm. Atrial activation is not apparent within the QRS and T complexes of that first wide beat. Atrial activation reappears at the extreme right of the slide. The reason for the depression of sinus node is not clear.

Dr. Dickinson: Would you treat this, and if so, how?

Dr. Bigger: I would first place an atrial lead to determine if atrial activation is hidden in those wide beats. I would not treat until the rhythm was diagnosed and the history, physical, and laboratory examination were satisfactorily completed.

Dr. Moe: In this situation I would like to observe a simultaneous record of the arterial pressure because



CASE III:

This 60-year-old alcoholic was admitted to the Rehabilitation Center and the routine electrocardiogram demonstrated left axis deviation with right bundle branch block. There had been several episodes of questionable syncope probably related to his alcoholism. Is provocative atrial pacing of any value in predicting which patients may be subject to Adams-Stokes seizures?

it is conceivable that this arrhythmia could be vagally induced. The last P-R interval in which the P waves are clearly visible is distinctly longer than the preceding one.

Dr. Dickinson: Dr. Dreifus, could this be ventricular tachycardia with exit block?

Dr. Dreifus: This is a possible explanation, but in my experience it is extremely rare. Inhibition of sinus rhythmicity and vagal influence are probably major factors in this situation. Acceleration of the sinus rate by the administration of atropine, or by raising the foot of the bed, may be an effective approach. I have observed their disappearance by the administration of lidocaine as well. In general, they are benign arrhythmias, and it is rare for them to develop repetitive ventricular tachycardia.

Dr. Hoffman: The fact that lidocaine was administered and that they disappeared does not mean anything. I believe that this type of arrhythmia might be observed off and on again for a few minutes or a half-hour, and then it is questionable whether lidocaine was effective as a therapeutic agent.

Questioner: In one of our patients we have observed that slow ventricular tachycardia was terminated by premature ventricular contraction. I would like to know whether you consider this evidence of a reentry mechanism.

Dr. Moe: This is a very broad question, and therefore, it will receive a very broad answer. Yes, it is possible, but one would have to have more definitive evidence than that to be able to make a diagnosis. I am sorry if I disappoint you, but that is how I feel.

Dr. Hoffman: Dr. Dickinson, I wonder if I might try to confuse the picture a little more. The temptation which I think confronts everybody is to assume that rate and the duration of the QRS complex provide an indication as to the site of the impulse initiation. If you have a normal QRS, it is stated to be above the bifurcation or in the common bundle. If you have a broad, bizarre QRS complex, then you assume that it arises distal to the bifurcation, and the ideal rate would be 40, let us say. I believe this is a

very imprecise set of guidelines because it is quite possible to have an impulse arise in the common bundle, to be conducted aberrantly, that is, to be delayed in one or another of the other fascicles of the conducting systems and to give you a wide QRS complex even though it is originating proximal to the bifurcation. The point is that aberration is usually associated with rapid rate. Yet, when the automaticity of the conducting system is increased, when the diastolic depolarization is fairly marked with long diastolic intervals, the depolarization of the conducting system proceeds farther. Then, even the impulse that originates in the common bundle will be conducted with aberration. In general, it is probably not permissible to use the association of a particular rate or the appearance of the QRS complex to decide that you have ventricular tachycardia or a junctional rhythm. I believe that you can have junctional rhythm with an abnormal QRS, and since the rate is 62, it cannot be stated that it represents accelerated activity in the distal portion of the ventricle. I do not know whether Drs. Bigger, Surawicz, or Dreifus would disagree with me, but I hope that one of them might try.

Dr. Moe: I doubt that all of the various antiarrhythmics act by suppressing automatic activity. All of these examples were from Purkinje fibers, and I think mainly in dogs. One might have the right to ask why, if these drugs all suppressed automatic activity and therefore ectopic automatic activity, do they not suppress all activity totally and thus cause cardiac arrest.

Dr. Hoffman: I cannot answer this in terms of "why." I think that Dr. Moe made a good point and that there are differences in the sensitivity of pacemakers in the various parts of the heart. When you are considering the sinus node, this is a special case, and it is quite insensitive to the slowing effect of any of these agents. In order to appreciably slow a healthy sinus node, you have to use very high concentrations. I do not have the vaguest idea why the sinus node is resistant to this effect of antiarrhythmic drugs, but I am glad that it is resistant.