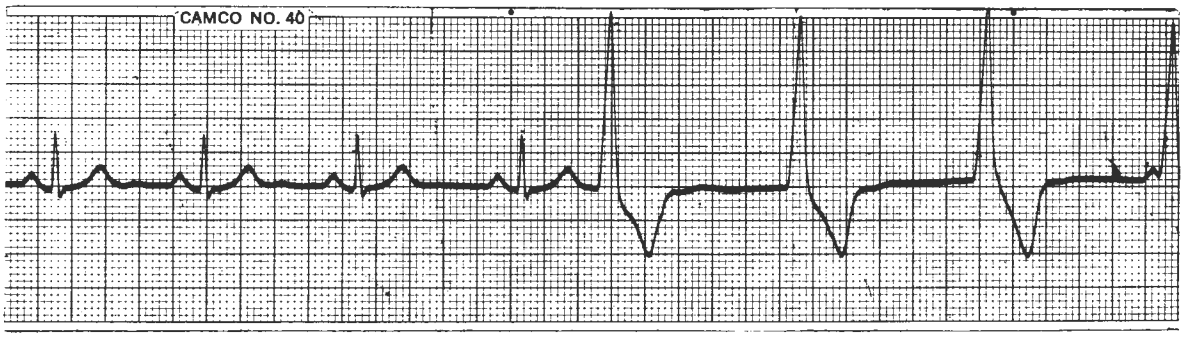
# Case Presentations

CASE I:

A 43-year-old white male construction worker was admitted to the coronary care unit for evaluation of left anterior chest discomfort.

How would you manage the following intermittent arrhythmia, which was noted shortly after admission? Vital signs remained stable.




## CASE I

**Dr. Baird:** Dr. Dreifus, how would you manage this patient's arrhythmia?

**Dr. Dreifus:** You have here a premature ventricular systole which begins a run of slow ectopic beating presumably from the ventricle. It is not occurring on top of the T wave. The victim is in ventricular tachycardia at a rate of about 60 or 62, and it appears to be a slow ventricular idioventricular rate without retrograde P waves. In this case I would try speeding up the sinus rate with a little atropine to eliminate the slow rate.

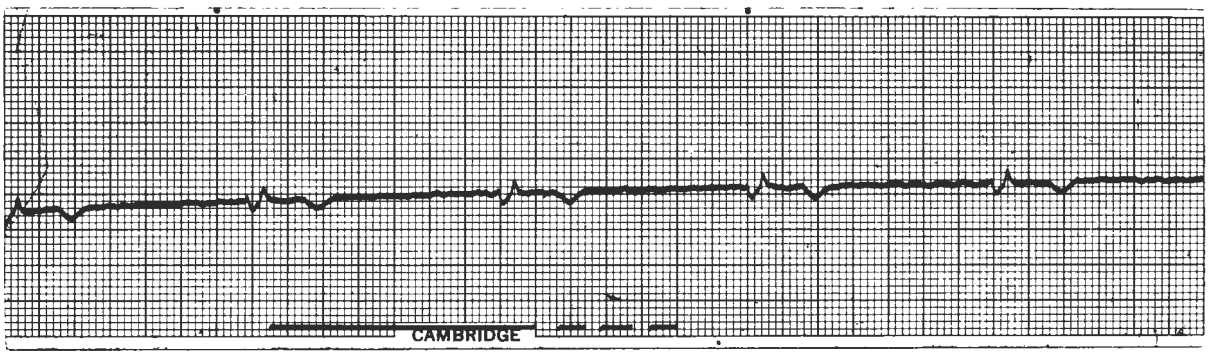
**Dr. Baird:** Would any other panelists like to comment or disagree?

**Dr. Zoll:** I would simply watch the patient and do nothing else.

**Dr. Parsonnet:** In our coronary care unit with continuous monitoring, this is not an infrequent arrhythmia, though Dr. Samet has just whispered in my ear that he sees it rarely in his unit. It is totally benign and has no serious consequences, therefore it is not treated.

CASE II:

This 75-year-old black male represents an arrhythmia related to digitalis toxicity. Is temporary pacing indicated?




## CASE II

**Dr. Baird:** This is a frequent problem in city hospitals. There is no adequate history available on this patient, but apparently he has been taking too much digitalis. The question is, if he has atrial fibrillation with high-degree A-V block, how would you manage him during the interim period?

**Dr. Samet:** First, how symptomatic is this individual?

**Dr. Baird:** He is confused, and he has been nauseated, but there has been no evidence of Stokes-Adams seizures. He has mild congestive failure.

**Dr. Samet:** Well, the rate here is in the low 40's. If he is symptomatic with something that would be improved by increased rate, then I would pace him. If he is not symptomatic with this arrhythmia, and if I felt it was due to digitalis, I would not pace, but stop the digitalis and just watch him. One point I want to stress is that when high-degree A-V block is a manifestation of digitalis toxicity, the administration of potassium is inappropriate and may have untoward consequences. If this is just complete heart block, then the potassium may have no further block-producing tendencies.

**Dr. Baird:** How do the other panelists feel? Dr. Dreifus, are you concerned about pacing a patient who has an arrhythmia due to digitalis?

**Dr. Dreifus:** I think if he is confused, I would do as Dr. Samet stated. The question is whether or not he is symptomatic. Now there is some controversy over potassium. I would tend to agree that if in the subjunctional regions you give enough potassium or give it too fast, you will cause further block. However, there is no doubt that potassium will antagonize digitalis in the N region of the A-V node and increase conduction. If the serum potassium were depressed and if I were worrying about causing further depression, I would then pace the patient and still administer potassium.

**Dr. Baird:** Thank you. What are the hazards with temporary pacing in patients with digitalis-induced arrhythmia? What has been your experience in this situation?

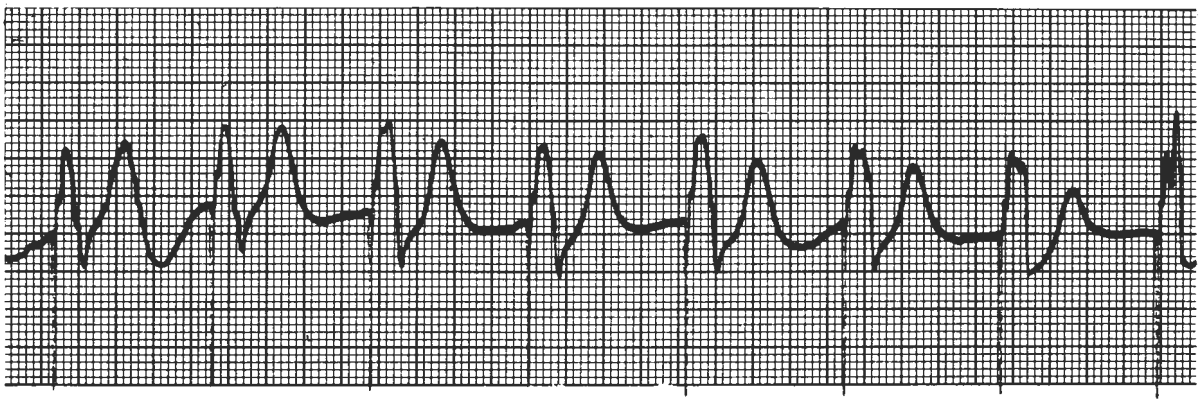
**Dr. Dreifus:** What you are worried about is the mechanical stimulation of that catheter number 1 and 2. If you induce a propagated response from the pacemaker on top of T wave in an over-digitalized patient, is the ventricle more vulnerable? Yes, the ventricle is more vulnerable, and the risk is increased. But with proper placement and prophylactic use of agents while you are passing the catheter and with great skill and few premature ventricular beats, this is really not a problem.

**Questioner:** What is your attitude toward treating this patient with diphenylhydantoin?

**Dr. Dreifus:** I don't think diphenylhydantoin would be of value in this instance. In the concentrations used clinically, we have shown that it increases block in the A-V node. If there were ectopic ventricular beats with block here, I certainly would avoid drugs and go the pacing route.

CASE III:

This patient with a permanent pacemaker developed substernal pain and hypotension. Would sympathomimetic amines be helpful to restore his blood pressure?



## CASE III

**Dr. Baird:** Dr. Zoll, how do you feel about the concomitant use of sympathomimetic amines in patients with pacemakers?

**Dr. Zoll:** With the functioning permanent pacemaker in place, one would treat hypotension with drugs as if the patient were in normal sinus rhythm. As long as the pacemaker rate is reasonably rapid, as in this case, the appropriate vasoconstrictor agents (i.e., neosynephrine or isoproterenol) can be used. Most of these drugs do have a tendency to arouse ectopic activity as well as acceleration of dominant rhythm; however, with a well-functioning pacemaker, a positive inotropic action is not immediately seen.

**Dr. Samet:** My first reaction after seeing this tracing would be to ask for other leads. While this tracing probably represents paced beats followed by a T wave, I am not mathematically certain whether or not these other beats represent premature beats. I would have to see other leads in order to be certain.

**Dr. Baird:** I have used this case to suggest that in certain patients sympathomimetic amines might lower the threshold for ventricular fibrillation, and there might be instances in which untoward administration of these drugs might precipitate ventricular tachyrhythmias. Anybody have any comments or experience with this situation? Dr. Dreifus?

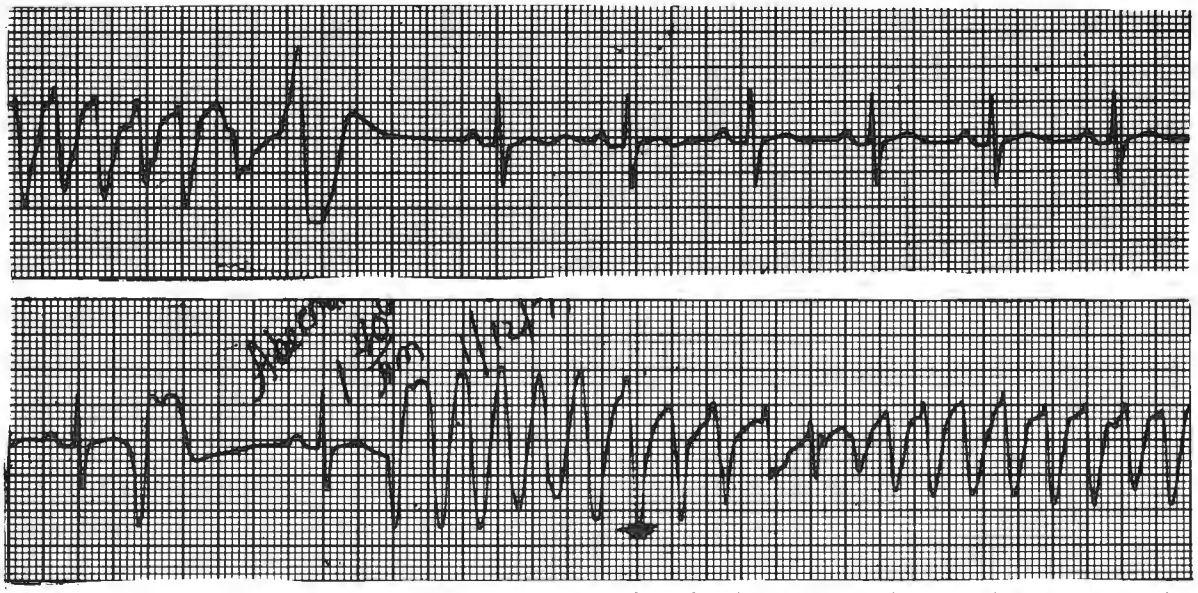
**Dr. Dreifus:** Yes. I learned my lesson on the cardiac surgery table where we used a good deal of sympathomimetic amines, particularly isoproterenol. Beyond a flow of 3 micrograms per minute of isoproterenol, you will get increased ectopic beating that could be fatal to the patient. Therefore, it is a matter of degree how you use these agents. I don't think we can say that they are all good or all bad; we should, however, be aware of the dangers and the limitations of administration.

**Dr. Zoll:** Isoproterenol clearly does arouse and accelerate ventricular pacemakers. There is a definite risk with isoproterenol; but if one gives the drug in a diluted solution, say 1 or 2 micrograms per minute, we can then step it up every several minutes. We usually change the dose every 4 or 5 minutes, watching what is going on all the time. You can keep on going until you get either the desired therapeutic effect of an increased cardiac output or increased blood pressure. If untoward effects appear (i.e., ectopic beats), reduce the dosage or adjust the administration of the dosage until no adverse effects are seen. There is no real limit as to how much you should give a patient. We have given over 30 micrograms per minute of isoproterenol to some patients with complete heart block, in what I consider to be a safe and effective way. On the other hand, some patients might run into trouble with a much smaller dose, in which case you stop and take care of the situation accordingly.

**Dr. Samet:** The arrhythmic effects of the vasoconstrictors really have two aspects. One is the direct aspect which has been alluded to, and the other is an aspect which follows the development of hypertension. That is, when one gets the desired effect by overshooting the mark, reflex arrhythmias secondary to hypertension can develop. Which leads me to my next point: One of the things we have learned to mistrust is the peripheral arterial pressure under these circumstances. We have, in a number of instances, obtained hypotensive readings with the usual blood pressure determination. Because you can readily overshoot the mark, we prefer monitoring the arterial pressure interarterially almost routinely when we feel we have to administer vasopressors. So I warn you not to trust the routinely determined blood pressures in these hypotensive patients.

CASE IV:

This 70-year-old white male was admitted for hematologic evaluation when he suddenly developed chest pain and acute EKG changes suggestive of diaphragmatic myocardial infarction. Several days later, he was placed on oral procainamide to suppress VPC's. However, the following arrhythmia developed.



CASE IV

**Dr. Baird:** Dr. Narula, how would you manage the patient in this situation?

**Dr. Narula:** This looks to be a very interesting arrhythmia which terminated spontaneously. Because of the sudden sinus beat, the possibility of fusion beat is less likely. Therefore, I would say it is probably another ectopic beat which suppresses the ventricular tachyrhythmias and gives the sinus rhythm a chance.

**Dr. Baird:** You would give more suppressant drug therapy?

**Dr. Narula:** Because sinus rhythm is fairly stable in this patient, within the normal physiological range, I think this patient would require suppressant therapy.

**Dr. Baird:** Any other comments?

**Dr. Samet:** In this discussion about the treatment of cardiac arrest and ventricular arrhythmia, I would like to stress that Dr. Zoll and I were just talking about a very simple physical mode which does really work; we have seen a blow on the chest administered by a clinched fist stop ventricular fibrillation; we have also seen it restart the heart that was arrested in just a few seconds.

**Dr. Parsonnet:** I would like to add that as far as therapy in the cardiac care unit is concerned, there is a tendency to worry about the electrocardiogram and forget about the other things happening to the patient. For example, a person who has been in the intensive care unit for a few days, hypoventilating and getting all kinds of drugs, may have serious abnormalities in their blood gases and blood electrolytes. Before you rush off and start using a lot of other things, I think you ought to make sure that you have a normal electrolyte balance. Sometimes, by simply directing the blood gases or blood electrolytes, you can stop the arrhythmias.

**Dr. Dreifus:** I agree with Dr. Parsonnet; you have to look for other problems. But remember, these ventricular premature beats are bisecting the T wave. In other words, the onset is very close to the T wave, and vasoconstrictors will respond to drugs like lidocaine and procainamide. This repeated occurance in the development of fibrillation brings up an interesting hiatus in drug therapy: Do we use a drug like procainamide or lidocaine, which actually decreases fibrillation threshold, or do we switch to an agent like bretylium? We just might end up treating these arrhythmias with bretylium when they are prefibrillatory or fibrillatory. If there are single ventricular premature systoles, you may take the lidocaine approach first just to get rid of the ectopic beating period. However, if they are prefibrillatory, as in this particular record, we may be using bretylium in the future. I think you had better keep a very close eye on the literature to see where we go from here.

**Dr. Baird:** Dr. Samet, do you agree with Dr. Dreifus's enthusiasm in regard to bretylium?

**Dr. Samet:** If we are talking about bretylium as a drug of initial approach, I do not agree. However, if we are talking about bretylium as a drug to be used when others have failed, then I agree emphatically. There are side effects with bretylium even in the horizontal position which does not make it the drug of initial choice. We still rely upon lidocaine, quinidine, procainamide, etc., but have used bretylium with success on a number of occasions. The role of bretylium has to be

kept in proper perspective.

**Dr. Richardson:** What about procainamide as an inciting drug?

**Dr. Baird:** Well, that's a point I was trying to emphasize. Dr. Dreifus, when ventricular tachyrhythmias revert spontaneously, doesn't this give you a clue that quinidine or procainamide was induced?

**Dr. Dreifus:** No.

**Dr. Baird:** Do any of the panelists have experience with recurrent ventricular fibrillation due to quinidine? Our experience is, in a limited number of cases, that they do not require defibrillation, but may be controlled with overdrive pacing.

**Dr. Samet:** Would you tell us a little more about the circumstances developed? I suspect that some of those cases do not represent quinidine sensitivity but inappropriate use of quinidine.

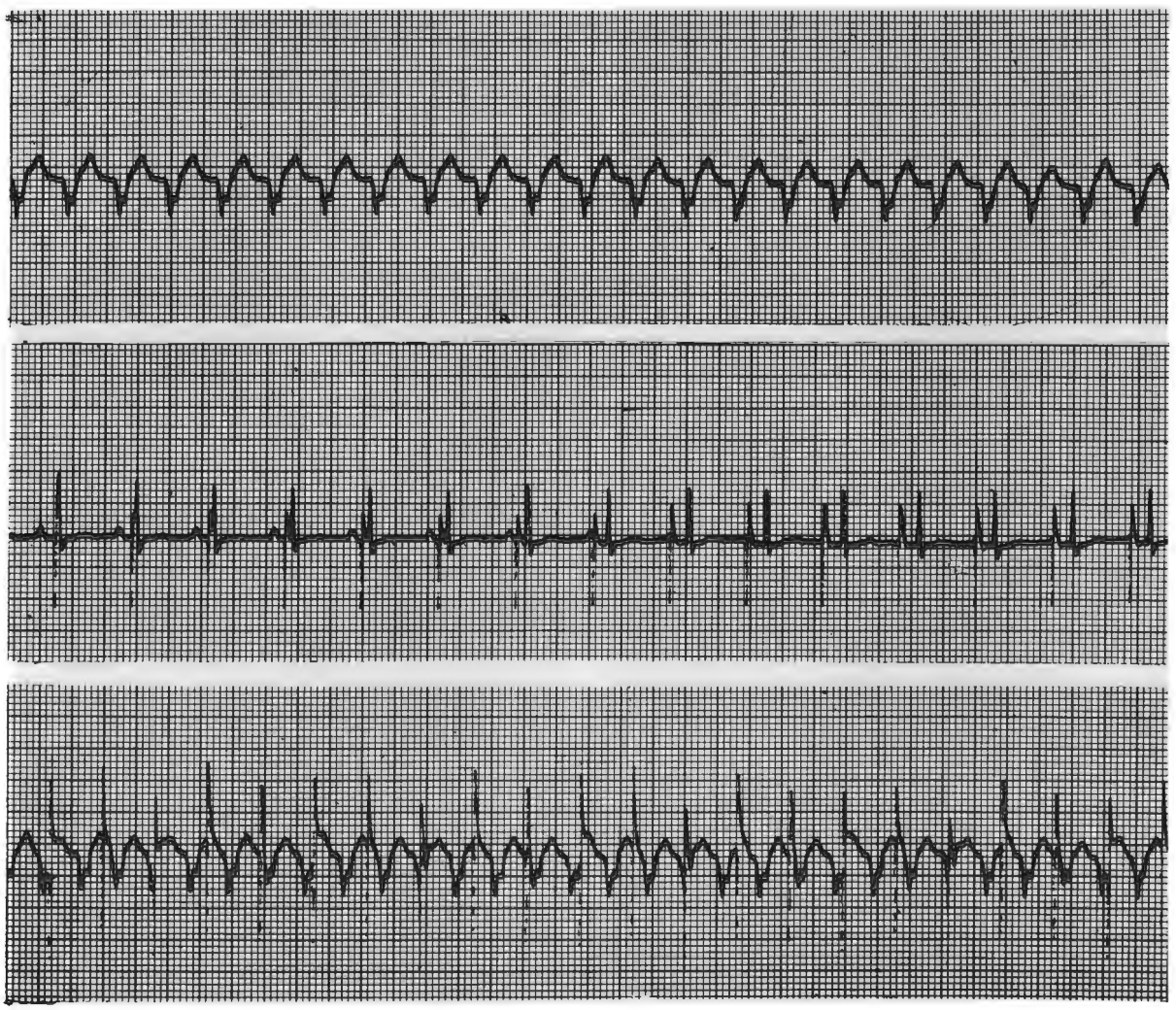
**Dr. Baird:** That is an important point in determining whether it is really a toxic effect, a rate-dependent problem, or an effect related to a true sensitivity as originally described; but this a point of argument. The procainamide was stopped and the patient's ventricular irritability decreased. He did not require any overdrive pacing as a technique suppressing drug-induced recurrent ventricular tachyrhythmias.

**Dr. Narula:** Could it be that the chest pains and the ischemic changes disappeared with time and were responsible for the disappearance of the arrhythmia rather than the discontinuance of the procainamide?

**Dr. Baird:** No.

CASE V:

A 2½-year-old child was in excellent health until he developed a viral illness, which was complicated by rapid heart rate. Initial therapy was digitalization to control "PAT." This failed and recurrent bouts of "VT" required 350 countershocks (100–200 W.S.).



## CASE V

**Dr. Baird:** Here we discuss the advantages and disadvantages of pacing. I have just mentioned the fact that atrial overdrive for ventricular pacing may control a certain arrhythmia. In this case the child, who had been in excellent health, developed a flu-like syndrome associated with recurrent bouts of tachyrhythmia. At first it was thought that these were paraoxysmal atrial tachycardia. However, an intra-atrial lead demonstrated clearly dissociation between the atrial activity and the ventricular rhythm disturbance. The diagnosis was made that this was recurrent ventricular tachycardia in a child who possibly had some type of myocarditis or myocardiopathy. Assuming the diagnosis is correct, what would you recommend in the way of pacing or drugs to control this particular problem? The child had been given 350 countershocks by the pediatric service for recurrent bouts of ventricular tachycardia associated with hypotension. Dr. Dreifus, what are your thoughts on this problem?

**Dr. Dreifus:** Obviously this is a very difficult situation. I have not seen ventricular tachycardia very often in children. The use of digitalis here is contraindicated under the circumstances. I am not at all convinced that this is ventricular tachycardia. It could be junctional with aberrant conduction. Atrial pacing might work in this case if you could get the catheter to stay in place, but that is tough in a kid. Ventricular pacing may also work, but that too is difficult. What I have dealt with in children is the use of propranolol and quinidine. The problem is getting them to take that combination because it tastes terrible. But sometimes that will control the ventricular tachycardia. I have not used bretylium, but I think that if I had my choice after all the electricity that has passed through this kid, I might say this is one of the prefibrillatory or prerepetitive situations and switch to a drug like bretylium rather than push my luck with the other depressant agents.

**Dr. Baird:** Before we comment on bretylium, do you think there is any clinical trial justification of internal defibrillation?

**Dr. Dreifus:** I don't know that we could do it in a child though we do it in dogs. It takes a lot of hardware, and I am wondering if it would really be practical in the end. It works, but maybe Mr. Berkovits will have some ideas on how to develop the electronics to improve it. I would try to solve all problems like this pharmacologically rather than on an internal or external defibrillation basis. Once the patients are put on a prophylactic pharmacological treatment, the arrythmia usually goes away.

**Dr. Baird:** Any other comments? After bretylium was administered intravenously in this child, acceleration of the ventricular rate occurred. Whether continued administration of bretylium would have been effective as an antiarrhythmia, I don't know. After I had caused rate acceleration with the drug, I was no longer asked to follow the patient. Dr. Dreifus?

**Dr. Dreifus:** Small doses of bretylium do have a tendency to aggravate the arrhythmia. There is no doubt about it. There are some electrophysiological reasons why this might occur. The trick is to use enough, but using bretylium in a kid for a rather unknown problem is risky. You may have been right to back off. I might have tried a large dose.

**Dr. Baird:** It has been an enjoyable day and a half. I would like to acknowledge the moderators, Drs. Richardson* and Drew* as well as the panelists for the support and advice required to make this a successful program.

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