A Case of Sudden Death
Questions of Management
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(CHEST 1991; 99:1511-14)

CASE PRESENTATION
A 65-year-old woman sustained cardiac arrest at home in August, 1989, was successfully resuscitated out of hospital, and subsequently hospitalized elsewhere. She showed acute hypoxic encephalopathy, but recovered from this. Subsequent study showed electrocardiographic and enzymatic evidence of acute anterior myocardial infarction. She was asymptomatic at the time of discharge. Two months later, on October 21, 1989, a second cardiac arrest occurred at home. She was electrically cardioverted at home by paramedical personnel and was then referred to this hospital. When she arrived, she was asymptomatic, alert and cooperative. Physical findings disclosed normal vital signs with a fourth heart sound and a soft third heart sound. Otherwise, the cardiopulmonary examination was negative. A signal-averaged electrocardiogram showed normal findings with no evidence of late depolarization. An electrocardiogram (Fig I) showed evidence of prior anterior myocardial infarction with left axis deviation, a pattern which remained unchanged during the hospitalization. There were occasional ventricular ectopic beats sometimes sufficiently early to interrupt the T waves of preceding cycles. Cardiac enzymes showed no evidence of acute infarction. Resting nuclear blood pool scan disclosed a resting left ventricular ejection fraction of 20 percent with hypokinesis of the anterior wall, and dyskinesia of the apex. Cardiac catheterization performed on the following day disclosed 90 percent occlusion of the left anterior descending coronary artery just distal to the first septal perforator artery and a 90 percent stenosis of the proximal circumflex artery. Minimal nonobstructive disease was found in the right coronary artery. The left ventricle was normal in size with severe hypokinesis in the anterolateral wall. The apex was dyskinetic and the remaining portions of the ventricle contracted moderately well.

QUESTIONS FOR CONSULTANTS:
1. How would you manage this patient?
2. Is a signal averaged ECG of any use in management of this case?
3. Would an electrophysiologic study help in clinical decision making?
4. Would coronary bypass surgery or angioplasty be of any value in controlling this type of arrhythmia?
5. Under what circumstances would you recommend use of an automatic implantable cardiac defibrillator or other similar device?

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FIGURE 1

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Comments by Hein J. J. Wellens, M. D.

Management of this patient should be based upon risk estimation of the chance of sudden or nonsudden cardiac death in the near future, the type of arrhythmia causing cardiac arrest, and careful evaluation of pump function and myocardial perfusion.

The Past

The history tells us that the first episode of cardiac arrest occurred in the setting of an acute anterior wall myocardial infarction. No information is available as to the findings on exercise testing prior to discharge which could give us an idea about presence or absence of residual ischemia outside the infarcted region at that time.

The Present

A second episode of cardiac arrest occurred two months later. A new myocardial infarction was ruled out. As shown by the Seattle experience,1 cardiac arrest in the absence of acute myocardial infarction in the patient with coronary artery disease has a high incidence of an early recurrence.

As we discussed elsewhere2 the answers to four clinical questions allow risk estimation in the patient suffering from VF or sustained VT late after myocardial infarction. The risk for cardiac death rises progressively with the total number of positive answers. The four questions are: 1) Did the patient have a cardiac arrest (loss of consciousness) during the arrhythmia? 2) Did the arrhythmia occur within day 3 to day 60 after an acute myocardial infarction? 3) Is the patient (during regular sinus rhythm) in New York Heart Association class 3 or 4 for dyspnea? 4) Did the patient suffer more than one myocardial infarction in the past? In our patient, the answers are: 1) yes; 2) yes (day 60); 3) no; 4) no. This profile gives the patient a 15 percent chance of dying suddenly and a 25 percent overall chance of a cardiac death within two years.2

Work-up in hospital showed a resting LVEF of 20 percent with extensive wall motion abnormalities and severe two-vessel disease in the LAD and circumflex coronary artery. No mitral incompetence was found. Interestingly, the signal-averaged ECG showed no late potentials, which is in agreement with the finding of anterograde flow through the stenosed coronary arteries as recently reported by Lange et al3 and also suggests that an electrophysiologic substrate for a reentrant sustained ventricular tachycardia is unlikely, and that in all probability ventricular fibrillation has been the arrhythmia responsible for the cardiac arrest.

The absence of late potentials on the signal-averaged electrocardiogram makes it unlikely that sustained ventricular tachycardia can be initiated during an electrophysiologic study. I would not perform an electrophysiologic study, therefore, because it would not provide me with information that could help me in selecting antiarrhythmic drug therapy or in identifying the area of abnormal impulse formation which is required for successful surgical or electrical ablative therapy of the arrhythmia.

Although no anginal complaints were present after the second episode of cardiac arrest, I would order an exercise test combined with a nuclear myocardial perfusion study to determine residual ischemia outside the infarcted area. When residual ischemia is present, I would advise PTCA or bypass surgery, depending upon the coronary anatomy,4 and not recommend an automatic implantable defibrillator.

If no residual ischemia can be demonstrated, implantation of a defibrillator is indicated in view of the estimated risk of dying suddenly. Because of the left ventricular wall motion abnormalities and the coronary angiographic findings I would ask the surgeon to perform bypass surgery as well, realizing, however, that such a request is not firmly supported by scientific data.

One last aspect is the use of an ACE inhibitor in this patient. Evidence is increasing that, especially in anterior wall infarction, wall motion abnormalities and cardiac dilatation can be reduced by the administration of these drugs.5 Such treatment should preferably be started shortly after myocardial infarction. There are reports, however, that such therapy might still be useful when started later.6 Information about blood pressure and kidney function is essential in making the decision to implement this therapy in the long-term management of the patient.

References

Academic Hospital, Maastricht, The Netherlands.
Comments by Mark E. Josephson, M.D.*

Is a signal-averaged ECG of use in the management of this case?

The signal-averaged ECG, as developed by Simon,
1,2 is a tool used to detect low amplitude, high frequency signals and to terminal parts of the QRS which might be associated with the arrhythmogenic substrate responsible for ventricular tachyarrhythmias. The presence of these low amplitude signals is dependent upon the number and duration of abnormal, fragmented electrograms in the heart.3,4 The timing of these electrograms also is important. In the presence of an anterior wall infarction, fragmented electrograms may be present but have onset early in the QRS and therefore may end prior to the end of the QRS. Thus, they may be masked within the total QRS deflection, thereby failing to give a late potential. Moreover, patients with sustained ventricular tachycardia have longer signal-averaged ECGs and a higher incidence of late potentials than those presenting with cardiac arrest, thus suggesting a smaller substrate associated with the more rapid tachycardia associated with sudden death. Therefore, the negative signal electrocardiogram in no way would help me in the management of this case.

Would an electrophysiologic study be useful?

Assessment of this patient would absolutely require an electrophysiologic study since a known substrate (prior anterior septal infarction) is present. In patients with cardiac arrest, induction of a rapid sustained uniform ventricular tachycardia is a useful finding which is likely to have a positive predictive value for recurrence of the event if it is not dealt with. The induction of polymorphic tachycardia is less specific, although it may be observed in 30 percent or so of patients with cardiac arrest.5,6 While the induction of a polymorphic ventricular tachycardia and/or ventricular fibrillation in a normal patient without prior heart disease carries no clinical significance, therapy directed at such arrhythmias has been empirically found useful in the population of patients with cardiac arrest. This is simple Bayesian analysis in which a test to predict a finding which has a very low incidence of being present in the population to be tested is likely to be associated with a higher false positivity in that population. Conversely, if such a test is utilized in a population at high risk for the event, it may carry a higher predictive weight. A good example is the case of exercise testing in which positive exercise tests are frequently meaningless (false positive) when they occur in patients with no likelihood of coronary artery disease, but are meaningful when performed in a patient who has a high likelihood of having coronary disease. In an analogous fashion, the induction of a sustained uniform ventricular tachycardia in this patient and probably the induction of a sustained polymorphic tachycardia would be useful in helping me direct clinical therapy. One could use this finding to test the efficacy of antiarrhythmic agents using the ability to induce these arrhythmias as an end-point.5,9 The ability to induce a uniform ventricular tachycardia would also allow for the possibility of map-guided surgery. Failure to induce a tachycardia, however, does not mean the patient is without risk. Studies are now available which demonstrate between 20 and 30 percent of patients with cardiac arrest who have no inducible arrhythmia will have a recurrence.9 A preliminary study in our patient population suggests that patients at risk are those who have a substrate defined by either a positive signal-averaged ECG or endocardial catheter mapping demonstrating a significant portion of the ventricle with abnormal electrograms. Thus, sinus rhythm mapping of the left ventricle in a patient in whom no ventricular tachycardia is induced might be useful.

Would coronary bypass surgery or angioplasty be of value in controlling this type of arrhythmia?

If it could be demonstrated that a fixed arrhythmogenic substrate was present, as manifested by the ability to reproducibly initiate a sustained uniform ventricular tachycardia or the demonstration by sinus rhythm mapping of a large area of abnormal electrograms, the performance of a revascularization procedure in and of itself would not be sufficient to prevent recurrences. Studies in which a sustained monomorphic tachycardia has been induced have repeatedly shown that one cannot reliably treat this effectively with revascularization.11 Moreover, in the absence of an inducible arrhythmia, the presence of a fixed substrate, as demonstrated by either a positive signal-averaged ECG or marked evidence of abnormal endocardial electrograms by sinus rhythm mapping, would suggest that therapy in addition to revascularization was necessary to deal with a fixed substrate.

Under what circumstances would you recommend use of an automatic implantable defibrillator or other device?

The indications for an implantable device in general are: (1) drug refractory sustained ventricular arrhythmias, (2) life-threatening ventricular arrhythmias in whom surgery cannot be safely performed, (3) the presence of a cardiac arrest in the patient without a known definable arrhythmogenic substrate (those with normal hearts or those with cardiomyopathy). In all instances, ischemia should be addressed in addition to any specific antiarrhythmic therapy. This patient should have an exercise thallium test to evaluate whether or not reversible hypoperfusion was present. If so, revascularization of the patient should be per-

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formed. At that time, if a uniform monomorphic tachycardia was induced, I believe the procedure of choice would be to map it and perform a subendocardial resection or another surgical ablative technique to cure the patient. This will provide the patient with the best hope for long-term cure and no antiarrhythmic or anti-ischemic therapy and reasonable chance of a normal lifestyle.12 If surgery was impossible, an implantable defibrillator should be used. While this certainly has been shown to reduce sudden death subsequent to the procedure, it carries with it limitations on activity (driving should not be allowed), and the recognition that the arrhythmia must occur without attendant symptoms (syncope) prior to the drive firing.14 The device, while effective, is currently the most expensive form of therapy and, therefore, should be used only in selected patients.

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EDITORIAL COMMENT

After the coronary arterial lesions were demonstrated by coronary cineangiography, cardiac surgery was performed in which the left internal mammary artery was implanted into the left anterior descending coronary artery and a saphenous vein was utilized for implantation into the distal circumflex artery. At the same time, patches were placed surgically for later use with an implantable antiarrhythmic device. One week later, electrophysiologic study was undertaken and programmed ventricular stimulation failed to produce either ventricular tachycardia or fibrillation. Therefore, without evidence to suggest benefit from a more complex antiarrhythmic device or pharmacologic treatment, the decision was made to use an implantable cardiac defibrillator, which was then employed. Since that time (approximately eight months), she has had a satisfactory clinical course, although she has noted on two occasions subjective sensations compatible with intracardiac electrical shock, confirmed by interrogation of the implantable device.

The differing opinions expressed by Drs. Wellsen and Josephson, both deviating from the management described, point out the considerable latitude in which we find contemporary management of serious arrhythmias. Signal-averaged electrocardiography, a new technique, promises better selection of individuals to undergo more complex evaluation and treatment, but further data are required before its final place is established. As our knowledge increases, together with improvement in various antiarrhythmic devices and pharmacologic methods, we hope future management of such perplexing problems will become more efficient and standardized.

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